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A mathematical model for the blood flow in a modeled artery with a stenosis and an aneurysm

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Abstract

Mathematical modeling of blood flows in the stenosed arteries is a crucial and demanding problem. The pulsatile simulations of blood flow through two three-dimensional models of an arterial stenosis and an aneurysm are investigated in this study. Power Law, Casson, Carreau and the Generalized Power Law, these four non-Newtonian blood models as well as the Newtonian model of blood viscosity are used to investigate the flow effects induced by the different blood constitutive equations. There are three main purpose of this study. First purpose is to investigate the variation in wall shear stress in a stenosed artery or aneurysm at different flow rates and degrees of severity. Second purpose is to compare and quantify the difference of the various blood models and judge their significance and lastly, to determine whether the use of the Newtonian blood model is appropriate over a wide range of shear rates.

Keywords: Fluid flows, blood, pulsatile, stenosis, aneurysm, non-Newtonian, wall shear stress, simulations

1. Introduction

The hemodynamics hypotheses of arteriosclerosis were first formulated several decades ago, so flow imaging and computing have played an increasingly important role in advancing our understanding of how blood really flows in large arteries prone to arteriosclerosis, while lumped parameter and linear and non-linear one-dimensional wave propagation methods received much attention in the 1950s through 1980s, Computational fluid dynamics method based on solving the three dimensional equations of blood flow have a dominant influence on recent research efforts to quantify hemodynamic conditions in arteries.

The presence of a stenosis or an aneurysm in an artery may significantly alter the flow field and consequently the flow rate, leading to severe pathological incidences. In the case of a stenosis, the consequences may be cardiac arrest and stroke whereas the development of an aneurysm and its continuous dilation may lead to its rupture causing death or grave disability. Furthermore, the presence of the anomaly itself may produce flow disturbances such as vortex formation, which has been reported as a contributing factor to atherogenesis and thrombogenesis.

Chaturani and Samy^[1] studied a pulsatile flow of Casson fluid through stenosed arteries with application to blood flow. Chakravarty and Chowdhury^[2] observed the response of blood flow through an artery under stenotic conditions. Berger and Jou^[3] discussed the flows in stenotic vessels. Johnston^[4] worked on non-Newtonian blood flow in human right coronary arteries: steady state Simulations.

Neofytou and Tsangaris^[5] discussed on the flow effects of blood constitutive equations in 3D models of vascular anomalies. Sankar and Hemalatha^[6] observed a non-Newtonian fluid flow model for blood flow through a catheterized artery-steady flow. Nanda and Bose^[7] worked on a mathematical model for blood flow through a narrow artery with multiple stenoses.

Kumar *et al.*^[8] discussed on a non-Newtonian arterial blood flow model through multiple stenosis. Shah^[9] studied the effect of clopidogrel on blood flow through stenosed artery under diseased condition. Shah *et al.*^[10] observed a mathematical modeling and numerical simulation of blood flow through tapered artery. Siddiqui *et al.*^[11] investigated a mathematical modelling on blood flow through stenosed artery under the influence of magnetic field.

Shrivastava *et al.* [12] worked on a non-Newtonian fluid flow model for the slip condition on blood flow through a stenosed artery using power-law fluid.

Sriyab [13] studied on the effect of stenotic geometry and non-Newtonian property of blood flow through arterial stenosis. Kumar *et al.* [14] discussed on comparative study of non-Newtonian physiological blood flow through elastic stenotic artery with rigid body stenotic artery.

The purpose of this study are three fold: firstly, to investigate the variation in wall shear stress in an artery with a stenosis or aneurysm at different flow rates and degrees of severity; secondly, to compare the various blood models and quantify the differences between the models and judge their significance and lastly, to determine whether the use of the Newtonian blood model is appropriate over a wide range of shear rates.

2. Mathematical formulation

The flow geometry comprises a tube of diameter D and can be divided into three regions, the inlet, the deformed and the outlet region. In the case of the stenosis, the lengths of these regions are $4D$, $2D$ and $20D$, respectively. For the aneurysm, there are $4D$, $4D$ and $18D$, respectively. The radius of the undeformed inlet and outlet is $R_0 = D/2$. In the case of the stenosis, the radius of the constricted region is given by:

$$R = R_0 \left[1 - \left(\frac{R_0 - R_{min}}{R_0} \right) \left(\frac{1 - \cos\left(\frac{\pi x}{D}\right)}{2} \right)^2 \right] \quad \dots (1)$$

Where R_{min} is the minimum radius at the centre of the stenosis. In this investigation, three different degrees of stenosis were used, 20%, 50% and 80%.

In the case of the aneurysm, the radius of the dilated region is given by

$$R = R_0 + \left(a - R_c + \sqrt{R_c^2 - \left(\frac{b}{2} - x\right)^2} \right), \quad 0 \leq x \leq b \quad \dots (2)$$

Where x is measured from the start of the dilated region, a is the maximum width of the dilated segment and

$$R_c = \frac{a^2 + \left(\frac{b}{2}\right)^2}{2a}$$

Three different values of a is 0.25, 0.4 and 0.55 were used in this investigation.

3. Analytical solution of the problem

The blood flow is assumed to be laminar and incompressible and therefore the Navier-Stokes equations for 3D incompressible flow are given by:

$$\nabla \cdot V = 0 \quad \dots (3)$$

$$\rho \left(\frac{\partial V}{\partial t} + V \cdot (\nabla V) \right) = -\nabla \cdot \tau - \nabla p \quad \dots (4)$$

Where V is the 3D velocity vector, p pressure, ρ density and τ the shear stress.

Five different non-Newtonian blood flow models and the simple Newtonian model are considered in this investigation. The effects of these models on the flow field and the wall shear stress in the vicinity of a stenosis or aneurysm are examined. These blood models are given below.

1. Newtonian model

$$\mu = 0.00345 \quad Pa.s \quad \dots (5)$$

2. Power law model

$$\mu = \mu_0 \left(\dot{\gamma} \right)^{n-1} \quad \dots (6)$$

3. Casson model

$$\mu = \frac{\left[\sqrt{\tau_y} + \sqrt{\eta \dot{\gamma}} \right]^2}{|\dot{\gamma}|} \dots (7)$$

4. Carreau model

$$\mu = \mu_\infty + (\mu_0 - \mu_\infty) \left[1 + (\lambda \dot{\gamma})^2 \right]^{(n-1/2)} \dots (8)$$

5. Generalized power law model

$$\mu = \lambda |\dot{\gamma}|^{n-1} \dots (9)$$

Where

$$\lambda \begin{pmatrix} \dot{\gamma} \\ \dot{\gamma} \end{pmatrix} = \mu_\infty + \Delta\mu \exp \left[- \left(1 + \frac{|\dot{\gamma}|}{a} \right) \exp \left(\frac{-b}{|\dot{\gamma}|} \right) \right]$$

$$\eta \begin{pmatrix} \dot{\gamma} \\ \dot{\gamma} \end{pmatrix} = \eta_\infty + \Delta\eta \exp \left[- \left(1 + \frac{|\dot{\gamma}|}{c} \right) \exp \left(\frac{-d}{|\dot{\gamma}|} \right) \right]$$

4. Boundary conditions

It is assumed that the arterial walls are rigid and no-slip condition is imposed at the walls. At the outlet, stress-free conditions are applied and the pressure is set to zero. Finally, the velocity profile at the inlet is regarded to be that of fully developed flow in a straight tube and can be derived analytically for both the Newtonian and the Power Law fluids. The forms are

$$u = \bar{u} \left[1 - \left(\frac{r}{R_0} \right)^2 \right], \quad 0 \leq r \leq R_0 \dots (10)$$

Where u is the velocity component in the x-direction for the Newtonian flow and

$$u = \bar{u} \left(\frac{3n+1}{n+1} \right) \left[1 - \left(\frac{r}{R_0} \right)^{\frac{n+1}{n}} \right], \quad 0 \leq r \leq R_0 \dots (11)$$

For the non-Newtonian flow, in transient flow, the pulsatile flow at the inlet is given by a time varying forcing function given in (7). This forcing function was scaled to yield a maximum inflow velocity of \bar{u} with a heart rate of approximately 60 beats per minute.

5. Solution methodology

The governing equations are highly non-linear and must be solved numerically using techniques of computational fluid dynamics. In this investigation, these equations are solved using the finite element method as implemented by COMSOL. The flow geometries for the stenosis and aneurysm were first created using Matlab. Then a finite element mesh was placed on these geometries. Briefly, an inlet plane of the artery is meshed in 2D using triangles and this mesh is extruded along the centerline of the artery to create a 3D mesh consisting of hexadrel elements. The mesh used for all computations consisted of 9, 708 elements and 15, 048 nodes for the stenosis and 17, 696 elements and 27, 132 nodes for the aneurysm as shown in Figure 1.

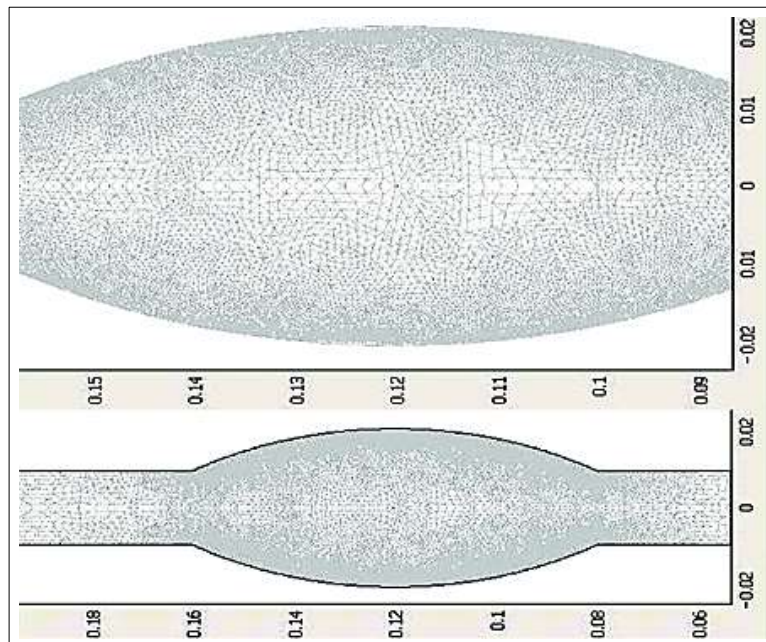


Fig 1: 3D grid for stenosis and aneurysm

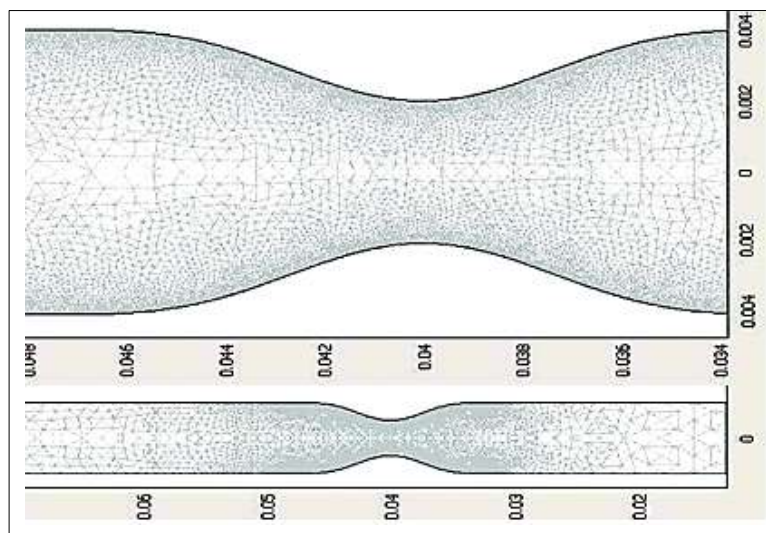


Fig 2: 3D grid for modeled artery with a stenosis and aneurysm

The governing equations were solved completely using the boundary conditions for fully developed flow (10) and (11) at the inlet along with the pulsatile forcing function for the transient case.

6. Results and Discussion

Transient simulations were performed using all five models given above. Three different degrees of stenosis were used namely 20%, 50% and 80% and maximum dilated widths of 25%, 40% and 55% were examined for the aneurysm in Figure 1.

Figure 2 shows that all of the non-Newtonian models considered here except the Power Law model produce a higher pressure difference than the Newtonian model. Specifically, the highest pressure drop is induced by the generalized Power Law model and the lowest by the Power Law model. Similar pattern in pressure differences are obtained at higher flow rates. The distribution of the wall shear stress is one of the most important hemodynamic parameter due to its direct relevance in atherosclerosis formation.

Figure 3 displays the distributions of maximum shear stress for various degrees of severity of the stenosis for all models. It is evident that wall shear stress increases with increasing severity. All models show close agreement with the Newtonian model except for the Power Law model. At 50% stenosis, the wall shear stress predicted by this model is significantly lower than the rest.

Figure 4 explains the distribution of wall shear stress along the geometry at various times. Maximum shear stresses are reached just before the throat of the stenosis. The magnitude of this value increases with higher flow rates. This peak is followed by a negative value indicating the presence of backflow. Further downstream, the wall shear stress steadily regains its undisturbed value. The maximum wall shear stress for various degrees of dilation of an aneurysm is displayed for all models. There is less agreement between the models in this case. Only the Casson and the Carreau models are comparable throughout. The power law model gives a much lower max W_τ value because it exhibits a lower viscosity at the throat of the stenosis where the shear stress is high.

As the flow rate increases, these wall shear stress differences from various models become more prominent indicating significant differences in model behavior. Figure 5 shows that transient simulations were performed using the Generalized power

law model for both the stenosis and aneurysm. Each simulation was from $t = 0$ to 10.0 secs, yielding a heart rate of approximately 60 beats per minute.

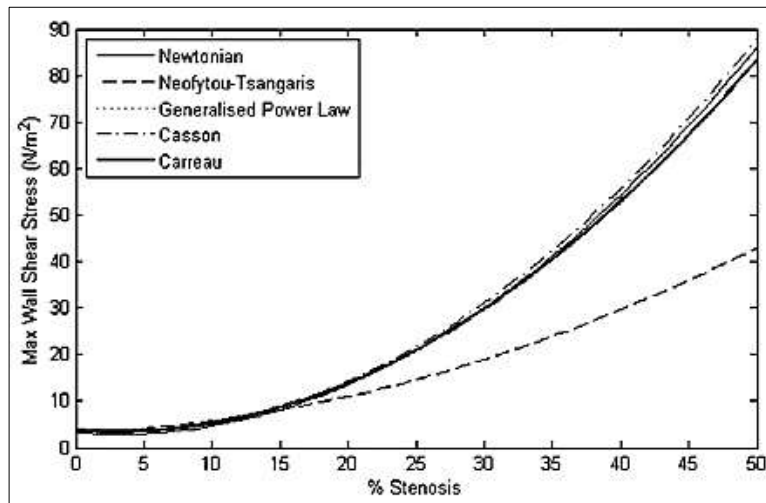


Fig 3: Wall shear stress versus percent stenosis for various models, with 0.11196 m/s inflow rate

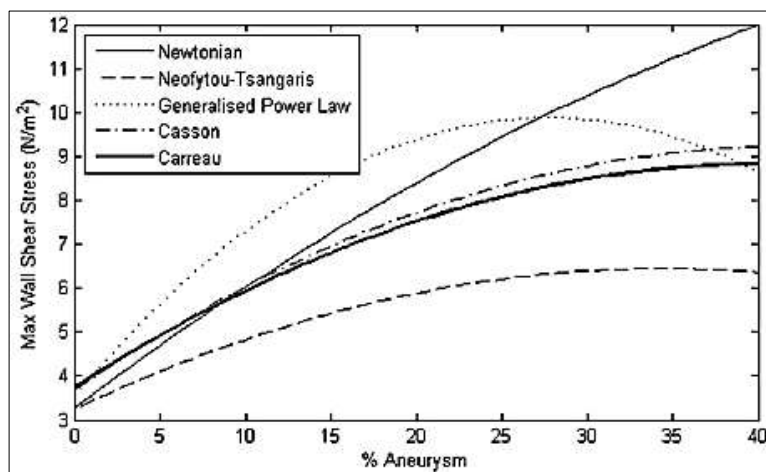


Fig 4: Wall shear stress versus percent dilation for various models, with 0.11196 m/s inflow rate.

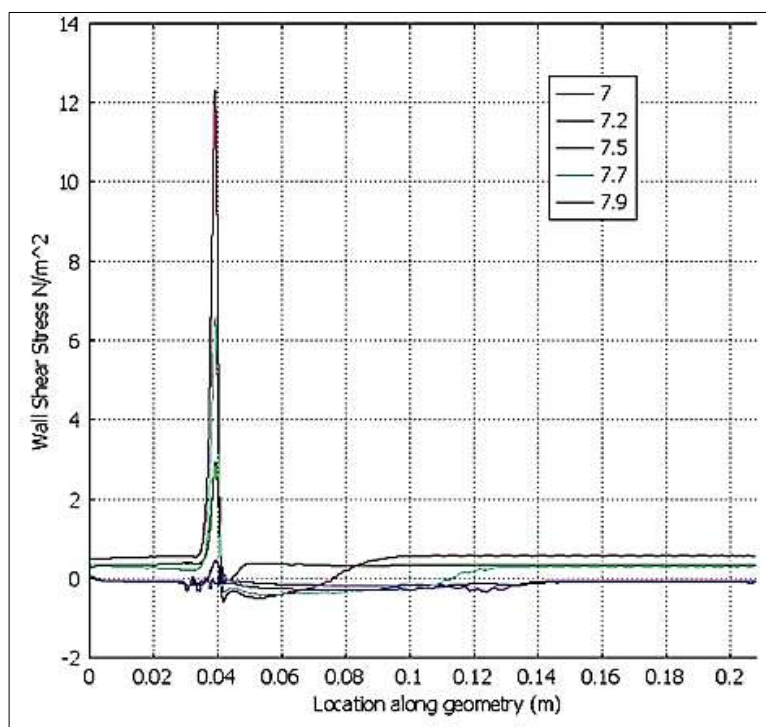


Fig 5: Wall shear stress for 50% stenosis pulsatile generalized power law model at various time intervals, with 0.11196m/s max inflow rate

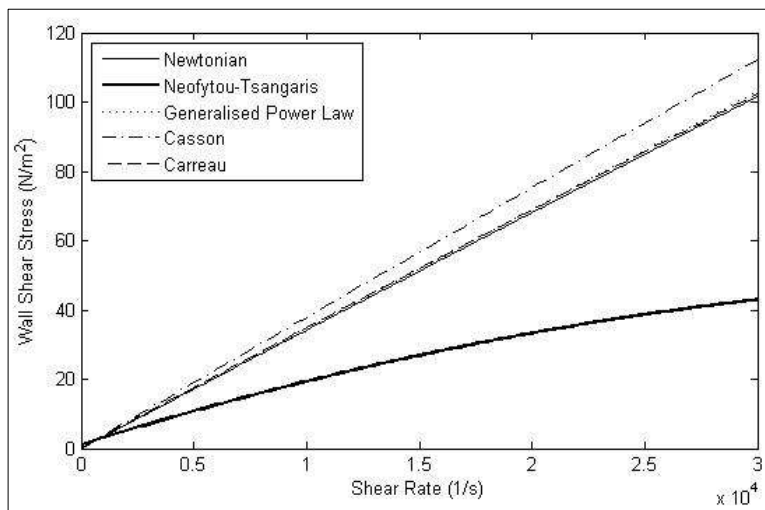


Fig 6: Maximum wall shear stress with shear rate in a stenosis.

Figure 6 expresses that distribution of maximum wall shear stress with shear rate in a stenosis. Again, wall shear stress increases with increasing shear rate with the power law model deviating significantly from the rest.

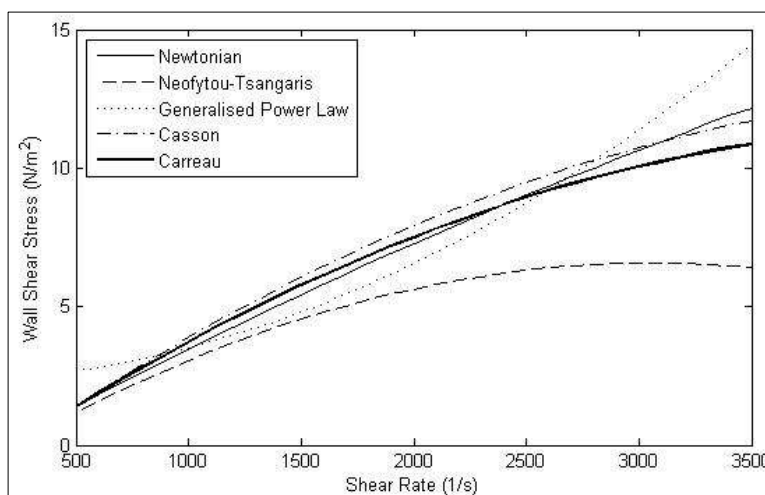


Fig 7: Wall shear stress versus shear rate in a stenosis

Figure 7 displays the maximum wall shear stress for increasing shear rate in an aneurysm. There is less agreement between the models at higher shear rates with the Power law model showing the least agreement.

In the case of pulsatile flow through the aneurysm, the simulation was performed for 10 sec using the Generalized Power Law model. As in the case of the stenosis, results in Table 4 show the maximum Wall shear stress values are produced at mid-cycle corresponding to the peak inflow velocity. However, the maximum pressure is attained just before mid-cycle and the minimum pressure towards the end of the cycle. This maximum pressure is excessively high. It is not clear why this is so and further investigation is planned. The streamline patterns at various points of the cardiac cycle are shown in figure 5. The formation and re-formation of the recirculation regions corresponding to the oscillatory nature of the pulsatile flow is evident. The wall shear stress distribution in pulsatile flow shown in figure 6 display a similar pattern in mid-cycle as in the steady state case. The distribution at other times show some marked differences due to the development of backflow regions inside the aneurysm. In all cases, for both the stenosis and the aneurysm, the flow field and the Wall shear stress changed significantly as the degree of abnormality increased. The recirculation regions become larger progressively and the wall shear stress generally increases, especially for the stenosis.

7. Conclusions

The effects of modeling blood flow through a stenosis and an aneurysm using five different blood rheological models is presented in this investigation. The flow field and wall shear stress distributions produced by each model are investigated for various flow rates and degrees of abnormality. The results show that there are significant differences between simulating blood as a Newtonian or non-Newtonian fluid. It is found that the Newtonian model is a good approximation in regions of mid-range to high shear but the Generalized power law model provides a better approximation of wall shear stress at low shear.

These conclusions are presented under the assumption that the arterial walls are rigid and zero pressure is assumed at the outlet. A more realistic simulation would include elastic walls and incorporate the effects of upstream and downstream parts of the circulatory system into the boundary conditions. This is a long term objective of this study.

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