CFD ANALYSIS OF STENOSED PULSATILE BLOOD FLOW

Thiago Felipe Calafati, calafati@ita.br[1]
Cláudia Regina de Andrade, claudia@ita.br [1]
Edson Luiz Zaparoli, zaparoli@ita.br[1]

[1]Instituto Tecnológico de Aeronáutica- ITA, Praça Marechal Eduardo Gomes, 50, Vila das Acácias, CEP 12228-900, São José dos Campos, SP

Abstract. *This work focuses on the numerical simulation of the pulsatile blood flow in a human artery, varying the stenosis level and the inlet velocity pulse frequency. The laminar flow is modeled solving the Navier-Stokes and continuity equations using an axisymmetric coordinate system. The finite volume method is employed to perform the domain discretization and a coupled formulation is used to solve velocity and pressure fields. A language-C subroutine is developed to impose the inlet pulsatile velocity and to incorporate the Casson non-Newtonian viscosity model. Three different Womersley number are tested: low (Wo = 4), moderate (Wo = 8) e high (Wo = 12), that induces period decrease and fast acceleration and deceleration as the Womersley number value increases. Besides, the artery stenosis level is changed (25%, 50% e 75%) to compare the Newtonian and Casson models behavior. Results are obtained for (i) stream function inside the artery domain, and (ii) wall shear stress (WSS) distribution. It is shown that the differences between the two viscosity models decreases as the Wo number elevates but the Newtonian model underestimates WSS values, suggesting a non-Newtonian viscosity model to suitably capture the blood flow behavior.*

Keywords*: CFD, stenosed blood flow, hemodynamic , non-Newtonian fluid, Womersley number, Casson´s model*

1. INTRODUCTION

The cardiovascular system primarily functions in nutrient and waste transport throughout the human body. The heart pumps blood through a sophisticated network of branching tubes. Because red blood cells are small semisolid particles, they increase the viscosity of blood and affect the behavior of the fluid. Blood is approximately four times more viscous than water. Moreover, blood does not exhibit a constant viscosity at all flow rates and is especially non-Newtonian in the microcirculatory system. The non-Newtonian behavior is most evident at very low shear rates when the red blood cells clump together into larger particles (Ku, 1997). Under certain circumstances, unusual hemodynamic conditions create an abnormal biological response. Atherosclerotic disease tends to be localized in these sites and results in a narrowing of the artery lumen named stenosis. Detection and quantification of stenosis can serve as the basis for surgical intervention. Thereby, the study of arterial blood flow can lead to the prediction of individual hemodynamic flows and the development of diagnostic tools to quantify the disease. The effects of non-Newtonian nature of blood and pulsatility on flow through a stenosed artery have been investigated by several authors.

Ishikawa et al (1998) studied the effect of non-Newtonian property of blood on flow through a stenosed tube. The flow pattern, separated region and the pressure and shear stress distributions along the wall were obtained. Their results showed that non-Newtonian behavior reduces the strength of the vortex downstream of stenosis.

Buchanan Jr. et al (2000) analyzed the rheological effects on pulsatile hemodynamics under stenosis condition. Transient laminar axisymmetric flow through a tube with a smooth local area reduction of 75% is considered and Newtonian, power law and Quemada viscosity models are investigated. Results showed that with increasing Womersley number, the formation of the primary vortex on the distal side of the stenosis is delayed by the expansion of the jet created by the throat. Besides, as the Womersley number increases the rheological differences among the three models are reduced.

The work of Rohlf and Tenti (2001) focused on the Womersley number role in pulsatile blood flow employing the Casson model. Blood was assumed to be a homogeneous, incompressible, axisymmetric laminar flow considering a rigid walled tube. Those authors recommended the use of the Womersley number as an indicator of whether non-Newtonian effects are important or not once they had confirmed this both analytically and numerically.

The physiological pulsatile flow through stenosed canine femoral artery was investigated by Ortiz et al (2006). normal artery (no diameter reduction) and three different models of axisymmetric stenosis (33%, 50% and 75% of diameter reduction) were tested. Results showed that reversal flow region is more extensive as the stenosis condition intensifies.

Li et al (2007) studied the pulsatile blood flow and vessel wall mechanics in different degrees of stenosis. The disease was modelled as axisymmetric cosine shape stenoses with varying diameter reductions of 30%, 50% and 70%, respectively. Their results suggested that severe stenoses inhibit wall motion, resulting in higher blood velocities and higher peak wall shear stress.

Siddiqui et al (2009) presented a mathematical modelling of pulsatile flow of Casson's fluid in arterial stenosis. Authors concluded that the mean resistance to pulsatile flow is greater than its steady flow value, whereas the mean value of the wall shear for pulsatile blood flow is equal to steady wall shear stress.

A CFD (Computational Fluid Dynamics) study of stenosed blood artery was presented by Calafati et al (2010). A constant degree of stenosis (50% stenosis) was simulated with a rigid vessel wall. Newtonian and Casson viscosity model results for wall shear stress and flow patterns were compared. Authors concluded that Casson non-Newtonian model is more suitable to capture the blood flow pulsatile characteristics.

In the present study, a numerical simulation of the pulsatile blood flow in a human artery is performed varying both stenosis level and the inlet velocity pulse frequency. The objective is to investigate both effects of the Womersley number and stenosis degree (25%, 50% e 75%) on the blood flow behavior.

2. MATHEMATICAL FORMULATION

The blood flow is assumed homogeneous, laminar, and incompressible. The pulsatile effect is also taken account. A straight axisymmetric artery with rigid walls is considered, as presented in Fig. 1.

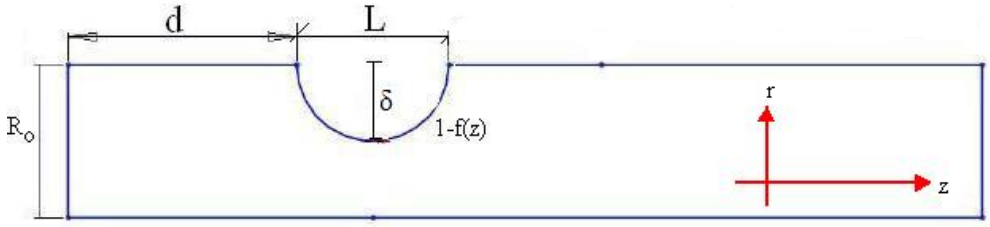


Figure 1. Axisymmetric schematic view of the stenosed artery.

Where:
r = radial coordinate
z = axial coordinate
R_0 = non-constricted artery radius
R = artery radius throughout the reduced (stenosed) region
L = stenosed segment length
d = distance from the start of the arterial segment to the start of the stenosis
δ = height of the stenosis

The stenosed region shown in Figure 1 is obtained as follows:

$$\frac{R}{R_o} = \begin{cases} 1 - f(z) & \text{for } d \le z \le d + L \\ 1 & \text{otherwise} \end{cases} \tag{1}$$

where $f(z) = A[L^{n-1}(z-d) - (z-d)^n]$, with n = 2 and $A = \frac{\delta}{R_o{}^n} \frac{n^{\frac{n}{n-1}}}{n-1}$

At all simulated cases, the tube lenght Z is equal a $6R_o$ and $L = 10\ mm$. For the stenosed artery, the internal surface dimensions are $\delta = 2.5\ mm$, $\delta = 5.0\ mm$ and $\delta = 7.5\ mm$ corresponding to 25%, 50% and 75% obstructions, respectively, (see Fig. 2a to Fig.(2c).

Figure 2. Stenosed artery with (a) 25% stenosis;(b) 50% stenosis and (c) 75% stenosis

Blood is a suspension of particles in an aqueous solution, called plasma. The three most important constituents in the human plasma are the red blood cell, the white blood cells, and the blood platelets. The majority of the formed elements are red blood cells (RBCs) as a result they are important components in determining the flow characteristics of blood. At low shear rates, that is, values less than 100 s^{-1}, the RBCs aggregate and form rouleaux. Rouleaux aggregation disperses as the shear rate increases, reducing the blood viscosity. The resulting shear-thinning behavior caused by rouleaux disaggregations in blood plasma is the principal cause of the non-Newtonian behavior of blood, Dinnar (1981). Thus, two characteristic features emerge from this non-Newtonian approach: (i) the presence of a yield stress and (ii) the dependence of the viscosity with respect to shear rate. To take these effects into account, the governing equations (continuity and momentum) and a Non-Newtonian viscosity model are used are stated as:

$$\nabla.\vec{v} = 0 \tag{2}$$

$$\rho\left[\frac{\partial \vec{v}}{\partial t} + \left(\vec{v}.\nabla\right)\vec{v}\right] = -\nabla p + \nabla.\tau \tag{3}$$

Where:

ρ = blood density; $\vec{v}$ =Velocity vector; p = Pressure field; t = time; ∇( ) = gradient operator; ∇.( ) = divergent operator

τ = stress tensor which is expressed by:

$$\tau = \eta\,\gamma = \eta\left[\nabla\vec{v} + (\nabla\vec{v})^{tr}\right] \tag{4}$$

In Eq. 4 the superscript "tr" indicates the transpose; η is apparent viscosity of blood and γ is the shear rate. When the fluid is Newtonian, the stress tensor is proportional to the shear rate, and the constant of proportionality is called dynamic viscosity (μ). For Non-Newtonian fluids, the viscosity is called apparent viscosity (η). When the Casson model is implemented, the apparent viscosity yields:

$$\eta = \frac{\tau_0}{\gamma} + \frac{\sqrt{C\tau_0}}{\gamma} + C \tag{5}$$

where η is the viscosity of blood, τ_0 yield stress and C is the Casson´s rheological constant. The values of τ_0 and C depends on hematocrit concentration, Fournier (2007). The blood behaves as Newtonian fluids for shear rate above 200 s^{-1}, as shown in Fig. 3. In can be seen that Casson model shows a better agreement with experimental data than Power-Law viscosity model.

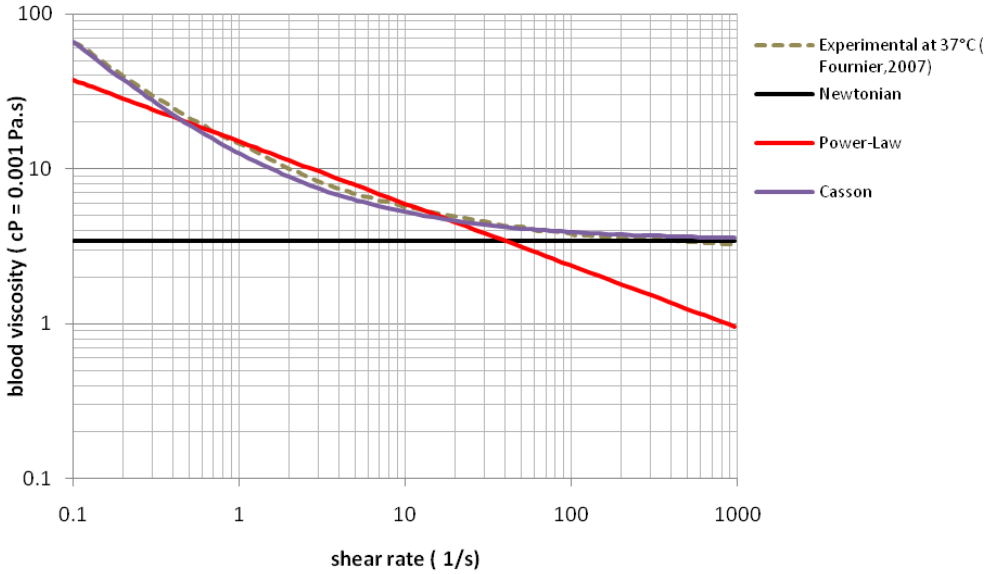


Figure 3. Blood viscosity model behavior and experimental results

The non-linear partial differential equation system represented by Eq. 2 and Eq. 3 requires appropriate boundary conditions. At the present study, no-slip boundary conditions were imposed at rigid walls and the elasticity of the artery wall was dismissed. A null value to the pressure was specified at the artery outlet.

Blood flow and pressure are unsteady in nature. The cyclic nature of the heart pump creates pulsatile conditions in all arteries. The heart ejects and fills with blood in alternating cycles called systole and diastole. Blood is pumped out of the heart during systole. The heart rests during diastole, and no blood is ejected. Thus the blood pressure in arteries is pulsatile, yet does not go to zero during diastole. In contrast, the flow is zero or even reversed during diastole in some arteries such as the external carotid, brachial, and femoral arteries, Ku (1997). This pulsatile behavior is numerically simulated imposing a pulse axial waveform velocity $v_r(t)$ at the artery inlet as:

$$v_r(t) = 0.03 + 0.03\sin(2\pi t/T) \qquad [m/s] \qquad (6)$$

Where:
T = period
t = time

For pulsatile flow, two dimensionless parameters are commonly used to characterize the flow conditions: (i) the Reynolds number $R_e = \frac{\rho \bar{v}(2R)}{\mu}$, where ρ is density of the fluid, $\bar{v}$ is the characteristic velocity determined as the time-averaged mean velocity and μ is dynamic viscosity and (ii) the Womersley number $\alpha = R\left(\frac{\varpi}{v}\right)^{0.5}$ with $\varpi = 2\pi / T$ is the frequency of the cyclic variation, R is the tube radius, and v is the kinematic viscosity. While the Reynolds number is a comparison of the inertial force to viscous force, the number of Womersley can be interpreted as the ratio of the unsteady force to viscous force. It is verified that high Womersley number, Wo > 10, flows correspond to situations involving periods of rapid blood flow acceleration and deceleration (Rohlf, 2001).

3. SOLUTION METHODOLOGY

A CFD package (Fluent 12.1.4 version) is employed to solve the pulsatile blood flow problem. Governing equations system are discretized using the finite volume method with a pressure based approach. As the Casson constitutive relations (Eq. 5) are not included in the Fluent code, it is necessary to develop a C-language subroutine that is coupled to the main software. The main parameters used in the simulations are listed in Tab. 1.

Table 1. Viscosity models and the input parameters used in the simulations

Blood viscosity model	Parameters
Newtonian	ρ= 1050 kg/m³; μ = 0.00345 Pa.s
Casson	τ_0 = 0.005 N and C=0.0035 Pa.s

A mesh sensitivity study (Tab. 2) was carried out by monitoring the mean velocity area weighted value at the artery outlet, as presented in Fig. 4 (red line). As the mesh density increases, the mean velocity tends to stabilize allowing to use the mesh 2 (154,000 cells) for 50% stenosis reduction. A similar analysis was performed for 25% and 75% reduction cases and an intermediate mesh was also chosen to save computational effort.

Table 2. Mesh sensitivity study results for 50% stenosis reduction

Mesh	Density	Mean velocity at the artery outlet
1	69,800 cells	0.02962 m/s
2	154,000 cells	0.02994 m/s
3	242,800 cells	0.02995 m/s

Figure 4. Sketch of the artery showing the computational domain outlet (50% reduction).

All computational meshes were generated employing quadrilateral elements, as can be seen in Fig. 5 for the 50% stenosed artery. During the solver procedure, the solution is converged when the variable residue reaches 10^{-5}.

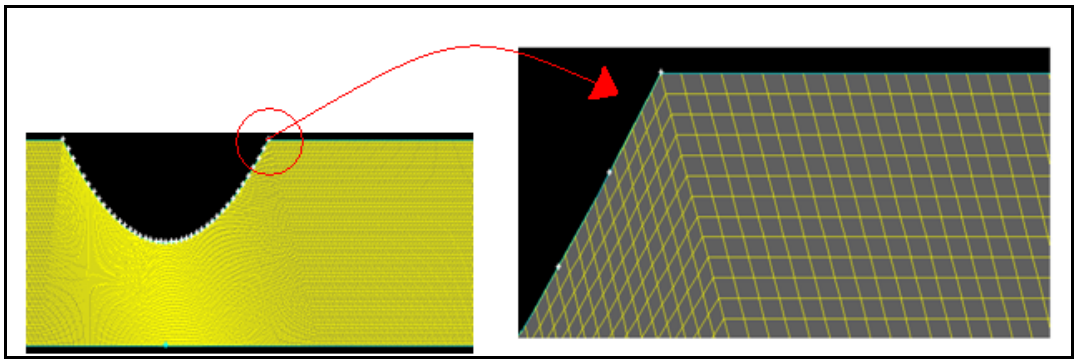


Figure 5. Mesh generated for the 50% stenosed artery with a "zoom" view.

The time-step used varies as the Womersley number increases due to the period reduction, as shown in Fig. 6. The used values are: 0.01 for Wo = 4 (T = 12 s); 0.0001 for Wo = 8 (T = 2.98 s) and 0.00001 for Wo = 12 (T = 1.33 s).

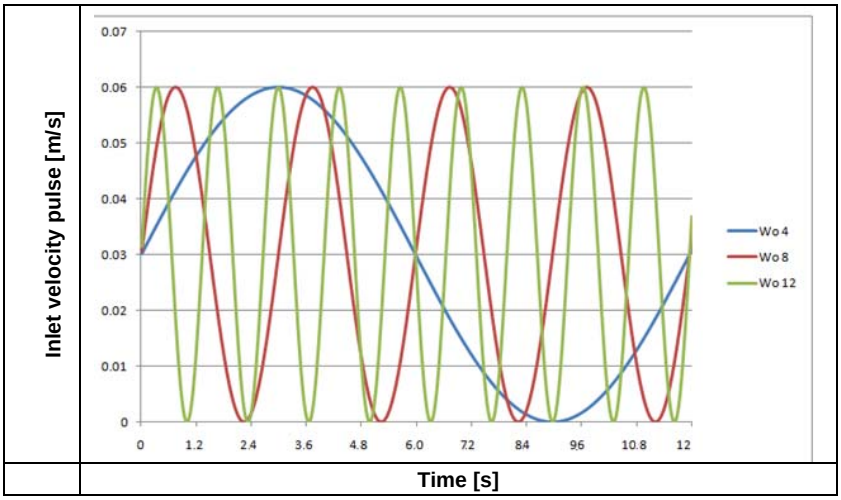


Figure 6. Inlet velocity pulse.

4. RESULTS

The CFD post-processing procedure was performed to obtain stream function contours, wall shear stress (WSS) and velocity profiles for both obstruction levels and Womersley numbers simulated. Figure 6 presents stream function contours results as a function of the stenosis level at t = 0.33 s and Wo = 12 (flow acceleration phase, Fig. 5) obtained employing the Casson viscosity model. It is observed that when the stenosis degree is smooth (Fig.6a, 25% reduction) the flow pattern is little disturbed due to the obstruction and the recirculation zone downstream of the stenosis is almost imperceptible. As the obstruction intensifies, the vortex flow downstream of the stenosis is well defined and the peak in the stream function elevates, Fig.6b, 50% reduction. For the case with 75% reduction, the recirculation zone is axially displaced and the disturbance reaches greater intensity. Note that, at Wo = 12 the inlet pulse impose rapid acceleration and deceleration to the blood flow.

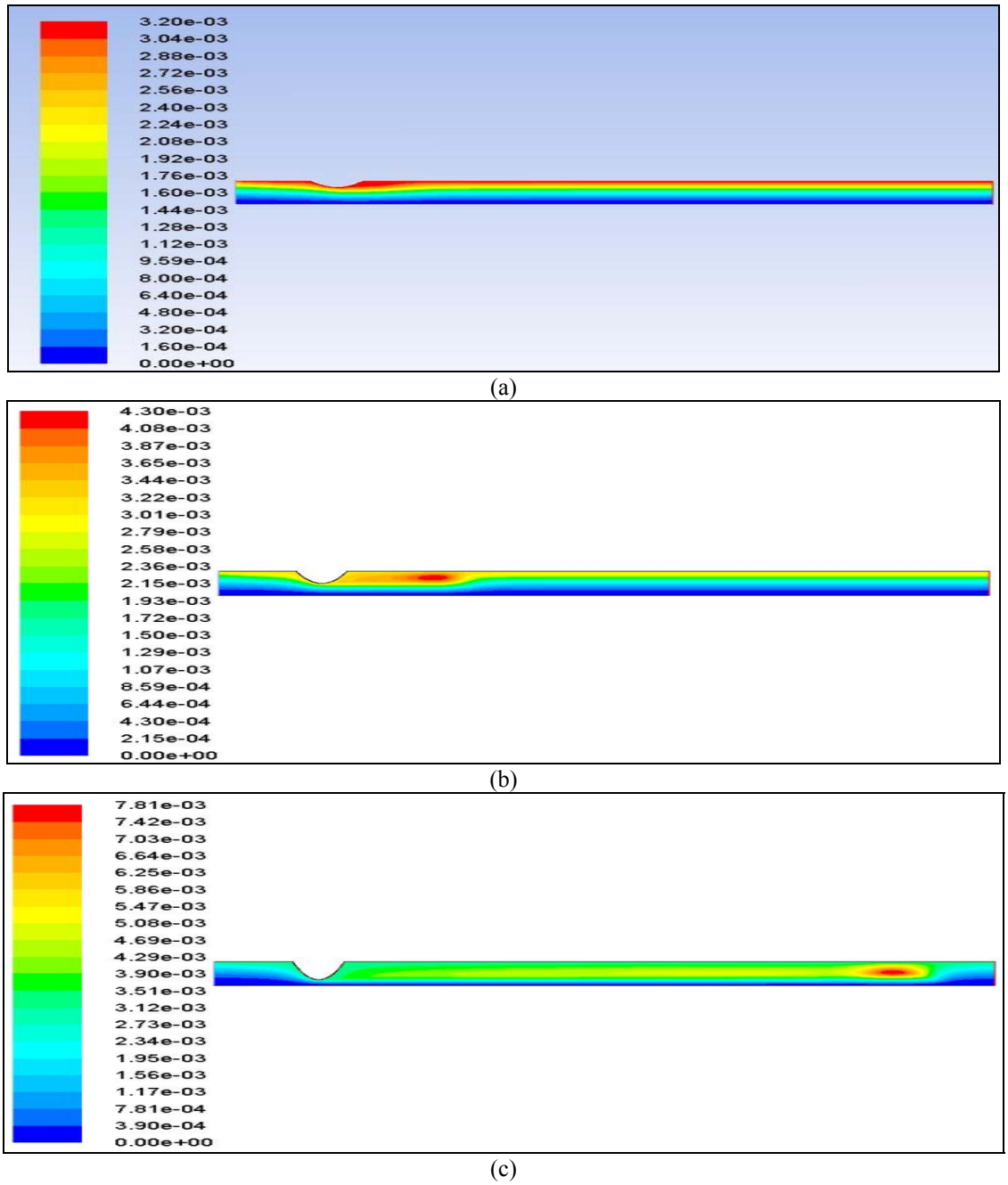
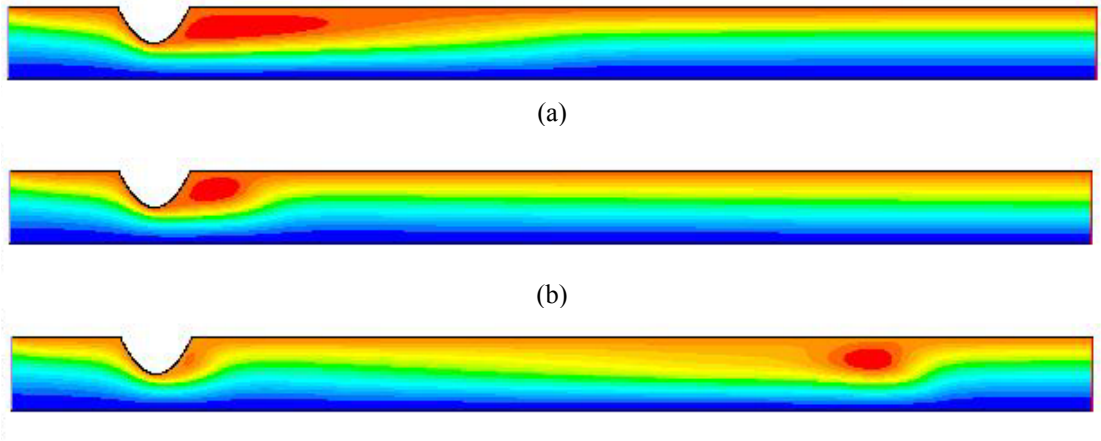


Figure 7. Casson model: stream function contours (kg/s) as a function of the stenosis level at = 0,333 s and Wo = 12. (a) 25%; (b) 50%; (c) 75%

Figure 8 shows stream function contours at 50% stenosis as a function of the Womersley number at t = 12s. It can be seen a displacement of the vortex flow as the Wo number increases but this recirculation zone is contracted at Wo = 12 (Fig. 8c). Besides, as the pulsatile flow has a briefer period (Fig. 6), it is possible to also visualize the initial formation of a secondary vortex immediately downstream of the stenosis region at Wo = 12.



(a)

(b)

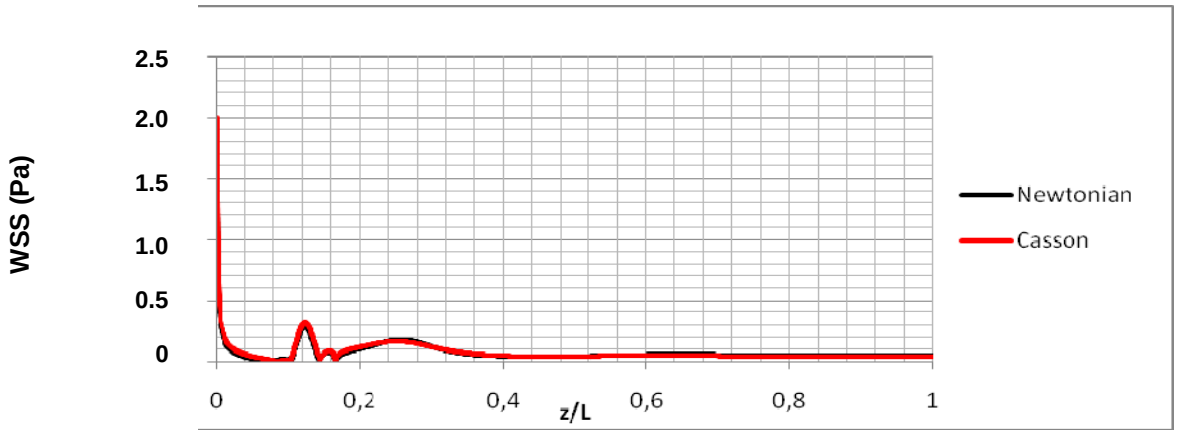
(c)

Figure 8. Casson model: stream function contours as a function of the Womersley number and 50% stenosis. (a) Wo = 4; (b) Wo = 8;and (c) Wo = 12

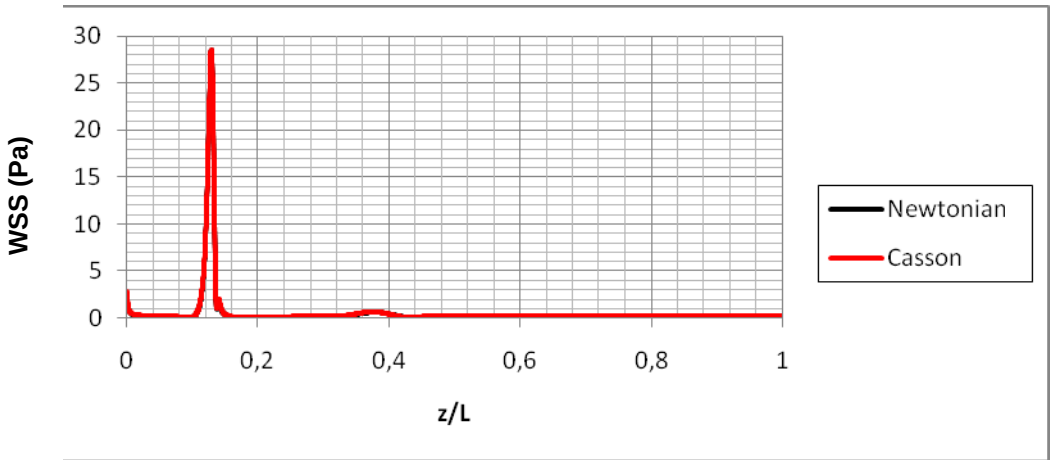
Initiation of atherosclerosis and thrombosis are strongly influenced by hemodynamics variables (Shibeshi, 2006). The goal in characterizing flows in the human vasculature is to quantify the hemodynamic forces that the artery wall experiences due to the system's input pulse, geometry, and blood rheology and see if there is any significant correlation between the characteristics of the flow field and abnormal biological events. In an attempt to reduce the amount of information presented and to more clearly elucidate the effects of the flow field, certain "hemodynamic wall parameters" have been calculated to present this information concisely, Buchanan et al (2000). As blood flow s across the artery, a shear stress (WSS) is generated to retard the flow (Ku, 1997). This wall shear stress (τ_w) is proportional to the velocity gradient at the wall and the apparent fluid viscosity (η) as follows:

$$\tau_w = \eta \frac{\partial V}{\partial n} \quad \text{, where } n \text{ is the unitary vector normal to the wall.} \tag{7}$$

At the present work, the wall shear stress (WSS) results along the stenosis wall are presented both Newtonian and Casson viscosity models at Wo = 12 and for 25% and 75% stenosis (Fig.9a and Fig. 9b, respectively). It is observed a similar distribution employing the Newtonian and non-Newtonian models in concordance with literature results (the two models collapse when Wo > 10, as cited by Buchanan et al, 2000). A comparison between 25% and 75% stenosis levels (Newtonian and Casson models) shows a abrupt increase in WSS values (Fig. 9b) as the obstruction level intensifies due to the elevate velocity gradients at the stenosis throat (flow acceleration).



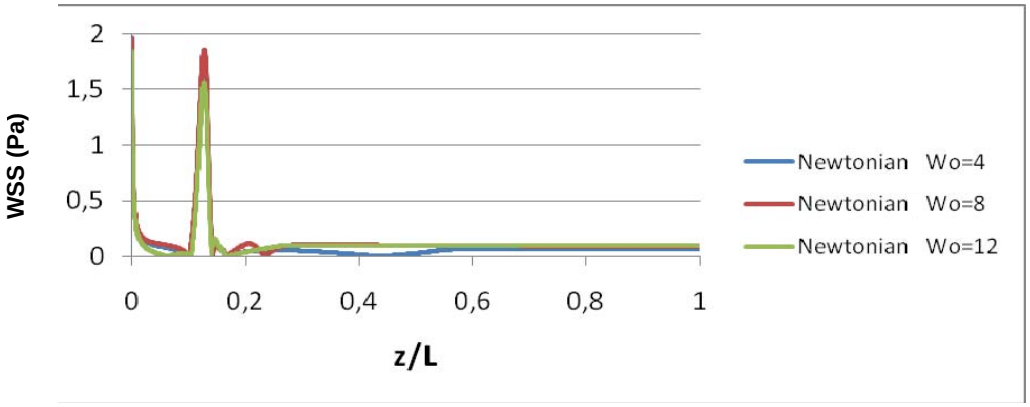
(a)



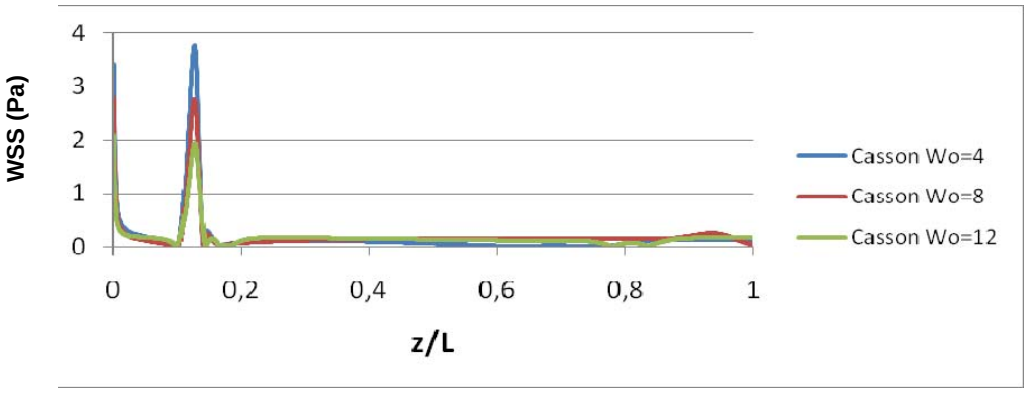
(b)

Figure 9. Wall shear stress distribution: (a) Wo = 12 and 25% stenosis; (b) Wo = 12 and 75% stenosis.

The effect of the Womersley number on the wall shear stress (WSS) is depicted in Fig 10. It is observed that employing both viscosity models, the peak in the WSS values are reduced as the Womersley number increases, although this fact is more significant for the Casson model (4 Pa to 2 Pa, Fig. 10b) in comparison with the Newtonian approach (1.8 Pa to 1.5 Pa, Fig. 10a). These differences confirm the literature results that Newtonian model underestimates WSS values, requiring a non-Newtonian viscosity model to suitably capture the blood flow behavior.



(a)



(b)

Figure 10. Wall shear stress distribution: (a) 50% stenosis and Newtonian model; (b) 50% stenosis and Casson model.

It can be also verified that the two viscosity models present major differences in the WSS peak at low Womersley numbers but as Wo number increases (or inlet pulse velocity period decreases) Newtonian and Casson model results tends to approximate.

5. FINAL REMARKS

At the present work, the blood flow problem was numerically simulated to study stenosis levels and pulsatile inlet velocity period effects on the axisymmetric artery flow patterns. Results showed that as the stenosis degree intensifies, the wall shear stress (WSS) strongly increases due to more intense velocity gradients and leading to the probability of cardiovascular diseases diagnosis. The inlet pulse velocity period also has a significant role in the blood flow behavior. At low Womersley numbers, Newtonian results underestimate the Casson viscosity model WSS values. As the pulsatile flow period decreases (high Womersley number), the WSS peak values are reduced and the Newtonian model can be suitable to capture blood flow rheological properties.

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