NUMERICAL ANALYSIS OF BLOOD FLOW VISCOSITY MODELS

Valdirene Morais Brambatti, morais@ita.br
Cláudia Regina de Andrade, claudia@ita.br
Edson Luiz Zaparoli, zaparoli@ita.br
Instituto Tecnológico da Aeronautica, Departamento de Engenharia Aeronáutica e Mecânica, Área de Aerodinâmica, Propulsão e Energia-Praça Marechal Eduardo Gomes, 50, Vila das Acácias, CEP 12228-900, São José dos Campos, SP

Abstract. *Blood is a complex fluid whose flow properties are significantly affected by the arrangement, orientation and deformability of red blood cells. The study of blood flow through is very important because the cause and development of many cardiovascular diseases are related to the nature of blood movement. Blood flow rheology is a complex phenomenon. Presently there is no universally agreed upon model to represent the viscous property of blood. However, under the general classification of non-Newtonian models that simulate blood behavior to different degrees of accuracy, there are many variants. Different viscosity models have been developed to represent the shear rate dependence of blood viscosity: Newtonian, Carreau, Power-law. Hemodynamic simulation studies have been frequently used to gain a better understanding of functional, diagnostic and therapeutic aspects of the blood flow. In the present study, the finite volume method is used to investigate blood flow behavior in a human carotid bifurcation, for the case three dimensional, steady state, incompressible. The momentum equation are solved employing two non-Newtonian viscosity models, as well as the Newtonian model for comparison purposes. Results for the wall shear stress (WSS) distributions and velocity profiles in axial direction of the carotid artery are obtained for both Newtonian and the non-Newtonian viscosity tested models.*

Keywords*:* CFD, carotid, blood flow, non-Newtonian, hemodynamic

1. INTRODUCTION

The blood flow in a carotid bifurcation model has been studied by numerous authors due to the fact that this local hemodynamic analysis is of great clinical interest with respect to both, the genesis and the diagnostics of atherosclerotic diseases. It is well known that the flow separation region of the carotid sinus has the propensity to develop atherosclerotic plaques, and thrombi (Urquiza, et al 2006).

There is considerable indication that complex hemodynamics (blood fluid mechanics) plays an important role in the development of artherosclerosis and other kinds of vascular diseases. The simulation method can be useful for understanding the behavior properties of blood flow and rheological blood properties.

Blood is a suspension of particles in an aqueous solution of constituents. The aqueous solution, called plasma, serves primarily as a transport vehicle for the various cells. The three most important cells in the human blood are the red blood cell, the white blood cells, and the blood platelets (Dinnar,1981).

Because of the major role of the red blood cells in determining mechanical behavior of blood, it is very important to know the volume concentration of red cells. This volume concentration, called hematocrit, ranges between 42 to 45% in the normal human. A change in hematocrit will change the flow behavior of blood from Newtonian to now-Newtonian.

Blood flow rheology is a complex phenomenon. One of the factors in determination of the rheological blood properties is red cell aggregation. This depends to very large extent on interaction of platelets and red blood cells. However, it is the properties of the red blood cells that together with blood plasma are responsible for the mechanical and rheological characteristics of blood.

The resulting shear-thinning behavior caused by rouleaux disaggregations in blood plasma is the principal cause of the non-Newtonian behavior of blood. However, with further increment of the shear rate beyond the low shear rate region, the shear-thinning characteristics disappear and blood demonstrates a Newtonian behavior.

Blood viscosity depends on many factors e.g. hematocrit, temperature, plasma composition, pathological conditions, etc. RBC aggregate face to face if they are brought in contact with each other at low shear rate. These aggregates are known as rouleaux and are formed under the influence of bridging macromolecules especially fibrinogen. Rouleaux formation (coin-like structures) increases the blood viscosity.

At low shear rates (i.e. shear rates $< 1\ s^{-1}$), secondary aggregation of rouleaux occurs leading to the formation of a rouleaux network. At high shear rates ($> 200\ s^{-1}$) these rouleaux segregate and RBC align with the flow and behaves as a Newtonian fluid (Fournier, 2007). Layers of stretched and packed red blood cells sliding on plasma layers will appear in blood composition. Then, the blood viscoelasticity will be dominated by the deformability of the red blood cells.

For a Newtonian fluid, the shear stress-shear rate relationship is linear and the scope is equal to the viscosity. For non-Newtonian fluids, as the blood, the slope of the line at a given value of the shear rate is the apparent viscosity. When the apparent viscosity decreases as the shear rate elevates, blood exhibits a shear-thinning behavior.

Several numerical studies indicate that the influence of shear thinning blood properties is not significant for the flow in large arteries (Perktold et al., 1991; Cho and Kensey, 1991). However, other studies don't neglect this influence (e.g. Rodkiewicz et al., 1990), or apply scaling procedures while comparing Newtonian and shear thinning fluid models (e.g. Baaijens et al., 1993; Ballyk et al., 1994). Usually, the blood non-newtonian behavior is taken account employing different viscosity models. Power-law (Johnston and Corney, 2004), Carreau (Shibeshi and Collins, 2005) and Casson (Siddiqui et al, 2009) are the commonly used models to predict the shear thinning blood behavior but presently there is no agreement upon a universal model to capture the blood hemodynamics.

At the present work, two non-Newtonian viscosity functions (Power-law and Carreau) are used to numerically solve the blood flow in a carotid bifurcation and the results are compared with the Newtonian model. Results show the effects of blood non-Newtonian behavior on both the WSS (Wall Shear Stress) distribution and axial velocity profiles under steady-state conditions.

2. MATHEMATICAL FORMULATION

The blood flow in a carotid bifurcation (see Figure 2a) is modeled applying the three-dimensional, steady, incompressible and considering that the artery walls are rigid. The governing equations can be written in form as

Continuity equation

$$\frac{\partial u_k}{\partial x_k} = 0 \tag{1}$$

Momentum equation

$$\frac{\partial}{\partial x_j}(u_j u_i) = -\frac{\partial p}{\partial x_i} + \frac{\partial \tau_{ij}}{\partial x_j} \tag{2}$$

The shear stress tensor, τ_{ij} is defined as

$$\tau_{ij} = 2\eta(\dot{\gamma})D_{ij}, \text{ with } D_{ij} = \frac{1}{2}\left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_j}\right) \tag{3}$$

where $\eta(\dot{\gamma})$ is the apparent viscosity, D_{ij} is the strain-rate tensor, and $\dot{\gamma}$ is the shear rate.

2.1 The Power-Law Model

When the blood viscosity is modeled following the Power-law model (Cho and Kensey, 1991) it is expressed as a function of the shear rate as

$$\eta(\dot{\gamma}) = k\ (\dot{\gamma})^{n-1} \tag{4}$$

Where k, n, are input parameters. k indicates a measure of the average fluid viscosity (the consistency index); n is a measure of the deviation of the fluid from Newtonian model (the power-law index). The value of n determines the class of the fluid:

$n = 1 \rightarrow$ Newtonian fluid
$n > 1 \rightarrow$ Shear-thickening (dilatant fluid)
$n < 1 \rightarrow$ Shear-thinning (pseudo-plastic).

2.2 The Carreau Model

For the Carreau model, the viscosity is expressed by

$$\eta(\dot{\gamma}) = \eta_\infty + (\eta_0 - \eta_\infty).[1 + (\lambda(\dot{\gamma}))^2]^{(n-1)/2} \tag{5}$$

Where the parameters n, λ, η_0, and η_∞ are dependent upon the fluid. λ is the time constant, n is the power-law index

(as described above for the non-Newtonian power law), and η_0 and η_∞ are, respectively, the zero- and infinite-shear viscosities.

Figure 1 shows Newtonian and two non-Newtonian blood viscosity models as a function of the strain rate. The parameters required in Eq. (6) and Eq. (7) are the same provided in Cho and Kensey (1991). It is also presented the human blood viscosity curve at a temperature of 37°C for comparison purposes.

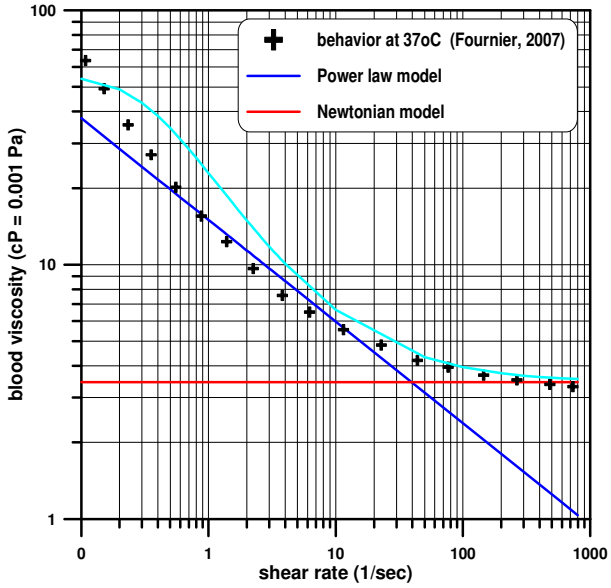
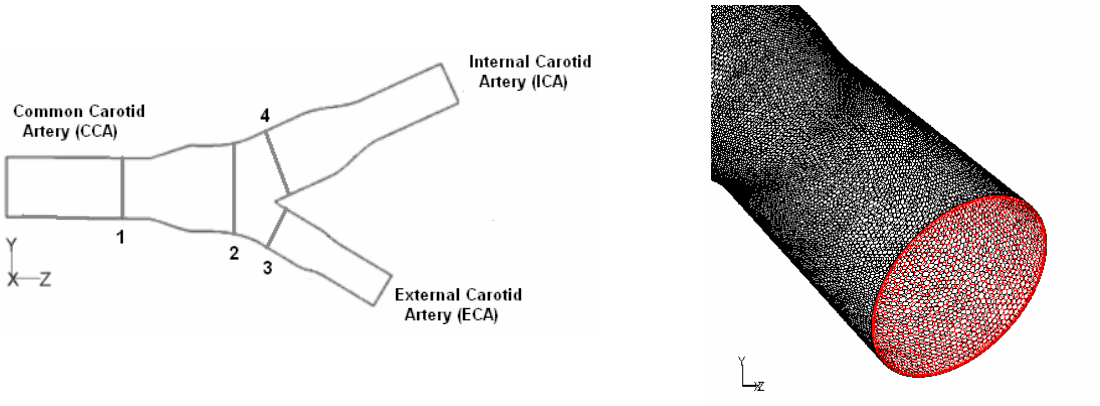


Figure 1. Blood viscosity as a function of shear rate.

It is generally accepted that blood behaves as a Newtonian fluid for shear rate above 200 s^{-1} (Pedley, 1980; Berger and Jou, 2000). It can be seen that the power-law model do not exhibit this behavior as they predict decreasing viscosity at high strain but is a good approximation at lower strain rate values. Note that the Carreau model tends to the constant Newtonian case at high shear rate.

3. SOLUTION METHODOLOGY

The governing equations system was discretized using the finite volume method employing a commercial CFD code (Fluent 6.4). The segregated pressure-based approach (SIMPLE algorithm) is applied for pressure-velocity coupling. An unstructured mesh with polyhedral elements and a prism layer close to the wall regions was generated (Gambit 2.4.6) as shown in Figure 2b. After a mesh refinement study the chosen mesh has a total of 500.000 cells number. Converged numerical solution is obtained when the mass and momentum equations attain a residual inferior to 10^{-6}.



(a) (b)

Figure 2. (a) Sketch of the carotid artery geometry; (b) "Zoom" of the computational mesh in the ECA branch.

The blood axial velocity results will be plotted at the four carotid bifurcation paths over vertical mid-plane, exhibited in Figure 2a. At the main branch (CCA inlet) the carotid artery diameter is $8 \cdot 10^{-3}$ m; at the ICA and ECA outlets, the diameters are 5.6 and 4.6 mm, respectively.

4. RESULTS

At the present work the blood flow inside a carotid bifurcation under steady-state conditions is solved to compare the Newtonian and non-Newtonian viscosity models behavior. No-slip boundary condition was imposed at the rigid artery walls. At the carotid inlet, a uniform velocity profile is imposed with values in the 0.01-0.10 m/s range. At the internal carotid artery (ICA) and external carotid artery (ECA) a mass flow ratio condition is applied: 0.55 and 0.45 at the superior and inferior branches, respectively). Numerical simulations were carried out using the parameters listed in Table 1.

Table 1 - Models of viscosity, η, given in Poise P (1P =0.1 Pas) as a function of strain $\dot{\gamma}$, given in s^{-1}

Models of blood viscosity	**Viscosity apparent η**
Newtonian	$\eta = 0.0345$ P
Carreau	$\eta = \eta_\infty + (\eta_\circ - \eta_\infty)[1+(\lambda\dot{\gamma})]^{(n-1)/2}$ where $\lambda = 3.313s$, $n = 0.3568$, $\eta_\circ = 0.56$ P e $\eta_\infty = 0.0345$ P
Power-Law	$\eta = k(\dot{\gamma})^{n-1}$ where $k = 0.015$ and $n = 0.6$

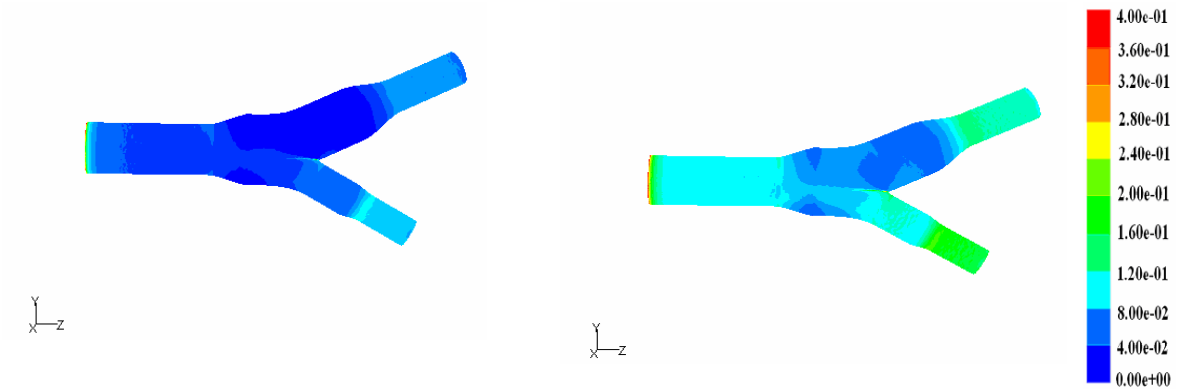
4.1. Wall Shear-Stress results.

In a laminar flow, the wall shear stress (WWS) is defined as a function of the normal velocity gradient at the wall as

$$WSS = \eta \frac{\partial u}{\partial n} \tag{6}$$

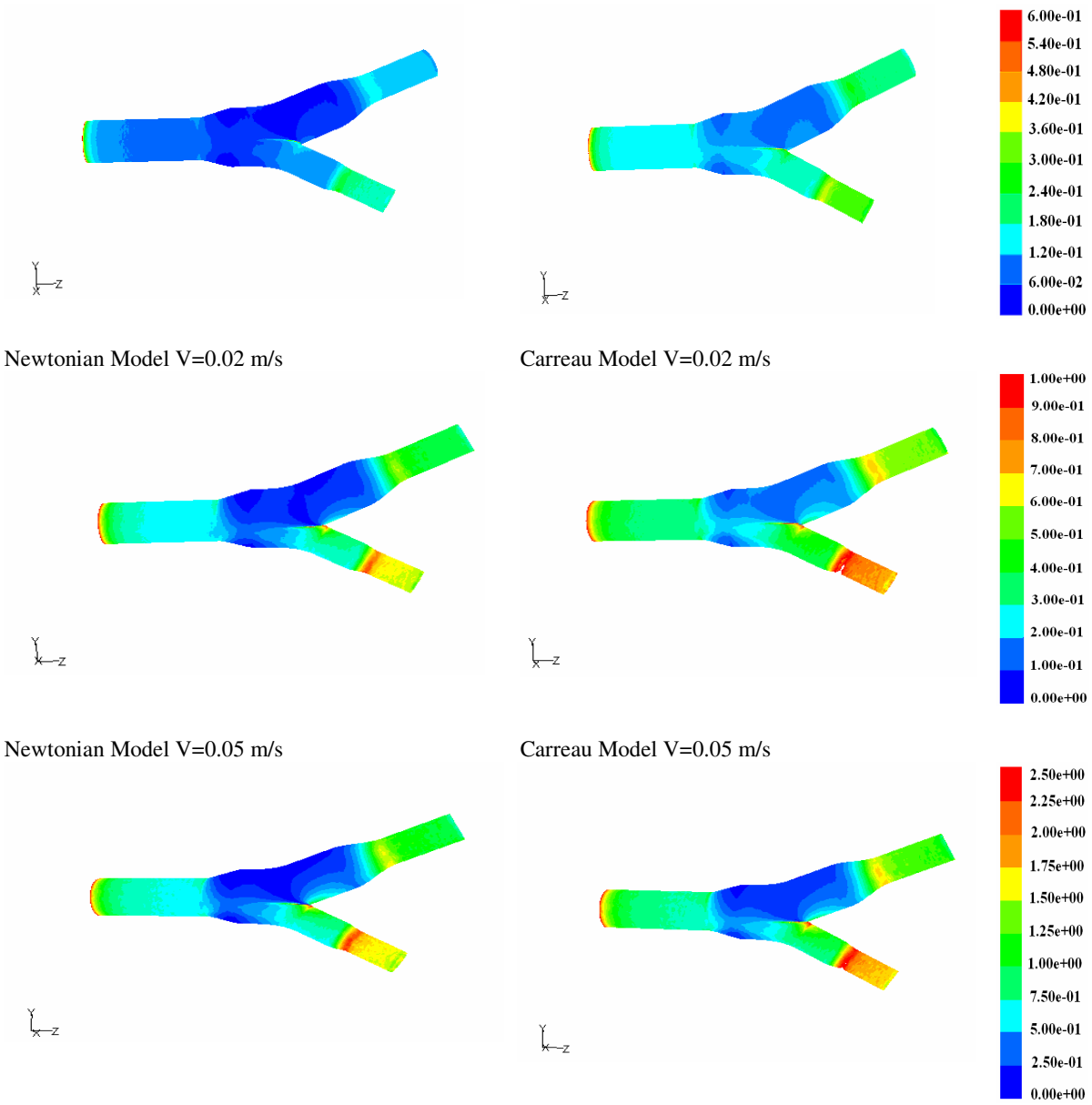
Where n is the direction normal to the wall and η is the viscosity apparent (which depends on the constitutive equation that will be implemented, Eq. 3).

WSS results obtained for Carreau and Newtonian viscosity models are presented in Fig. 6 at different velocities (0.01, 0.02, 0.05 and 0.10 m/s; respectively) at a vertical mid-plane of the carotid bifurcation. As expected, the WSS values elevates as the velocity increases (Eq. 8). A comparison of the WSS between Newtonian and non-Newtonian models show that at lower velocities the shear-thinning effect is more accentuated requiring a non-newtonian viscosity model to suitably capture the blood behavior inside the carotid. As the velocity increases, the two models produce WSS distributions quite similar. Besides, the Newtonian model subestimates the WSS values, mainly at the outlets secondary branches (ICA and ECA at Fig. 2a) when the inlet velocity is low.



Newtonian Model V=0.01m/s

Carreau Model V=0.01 m/s



Newtonian Model V=0.02 m/s | Carreau Model V=0.02 m/s

Newtonian Model V=0.05 m/s | Carreau Model V=0.05 m/s

Newtonian Model V=0.10 m/s | Carreau Model V=0.10 m/s

Figure 3 -WSS (Pa) in artery carotid at different CCA inlet velocities.

It is important to take account the non-Newtonian viscosity models due to the fact that WSS values are extremely low. Even at V = 0.1 m/s, the maximum WSS are 2.5 Pa.

4.2. Velocity field results.

Figure 4a and Figure 4b show the velocity magnitude contours at V = 0.05 m/s (CCA inlet imposed value) for newtonian and Carreau models. As occurred in the WSS distribution, there is little difference between these two viscosity models due to the higher velocity value. Note that the blood flow is accelerated as the carotid diameter (cross-sectional area) decreases at the secondary branches (bifurcation). The maximum values appear close to the artery centerline close to CCA inlet and ECA and ICA outlets. These maximum values are shifted towards the internal walls of the superior and inferior branches after the bifurcation.

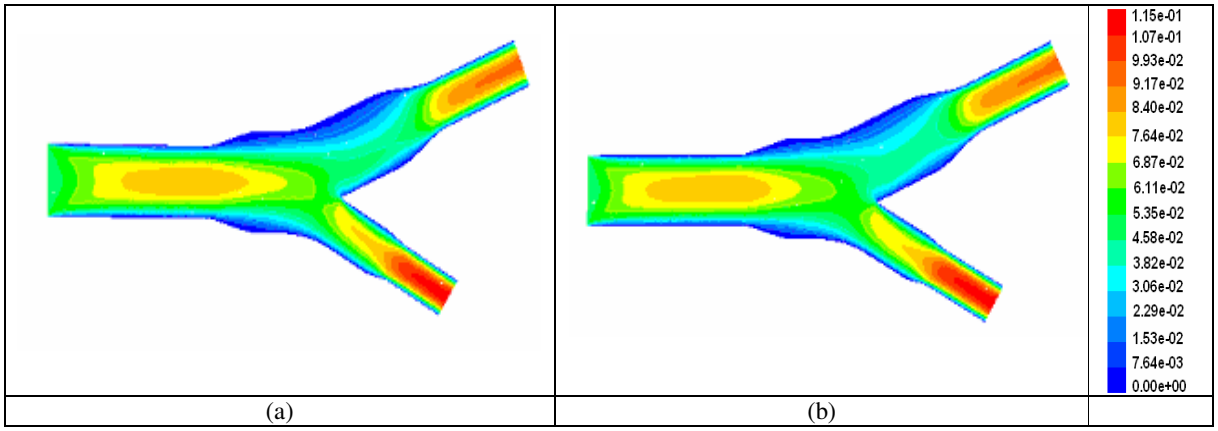


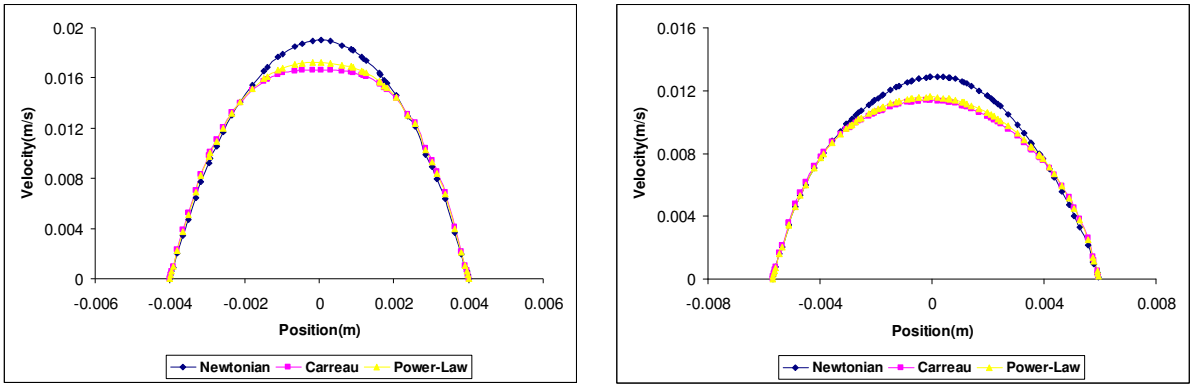
Figure 4. Contours of velocity; (a) Newtonian model (b) Carreau model.

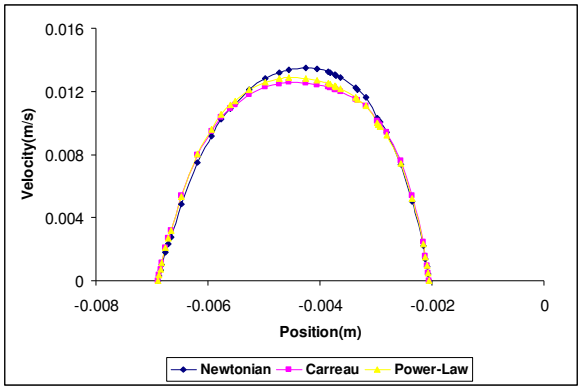
Figure 5 and Figure 6 show the axial velocity profiles for Carreau and power-law non-Newtonian viscosity models as well as Newtonian model for comparison purposes (V = 0.01 m/s and V= 0.05 m/s, respectively imposed at the CCA inlet). The plotted paths are located at the CCA main branch (path 1), before the artery bifurcation point (path 2), at the ECA inferior branch (path 3) and at the ICA superior branch (path 4).

Blood flow field results in the artery carotid indicate that the constitutive equations employed predict different velocity profiles and there is also a variation at different sections of the flow domain. In general, maximum velocity is predicted by Newtonian model exhibiting the known parabolic profile at the artery CCA main branch (Fig.3a). The maximum values obtained for the Newtonian model occur due to its smallest viscosity in the simulated shear rate range.

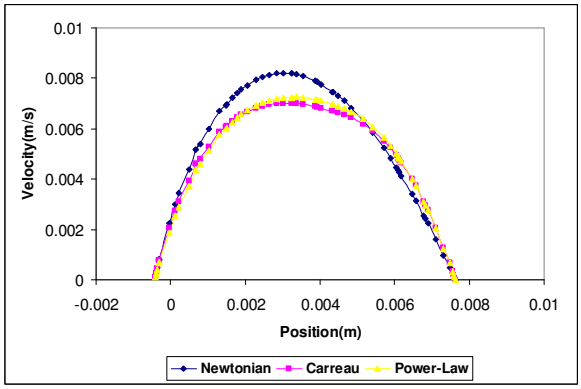
A comparison between the Newtonian and the non-Newtonian results reveals the influence of the blood shear-thinning properties on the velocity distribution. Note that differences in the axial velocity profiles are more pronounced around the carotid centerline and decreases towards the artery walls. For the four plotted paths, both non-Newtonian velocity profiles are flattened in comparison with the Newtonian ones, with higher values occurring in the main branch (path 1, Fig. 3a) in comparison with the others plotted paths.

Results at V = 0.01 m/s also show higher maximum values at the inferior branch (path 3 along the ECA, Fig.3c) in comparison with the superior branch (path 4 along the ECA, Fig.3d) for the three simulated viscosity models. This occurs because the blood flow is decelerated as the diameter (cross-sectional area) increase at the secondary branches (bifurcation).



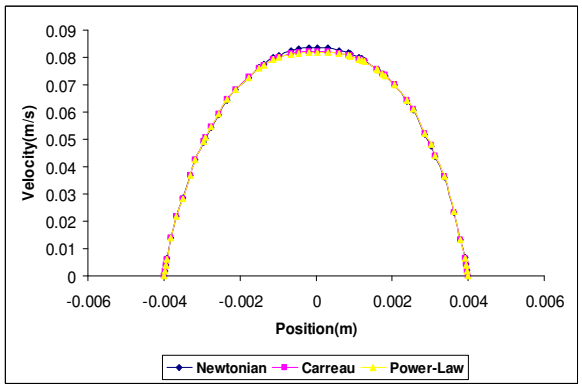


(c)

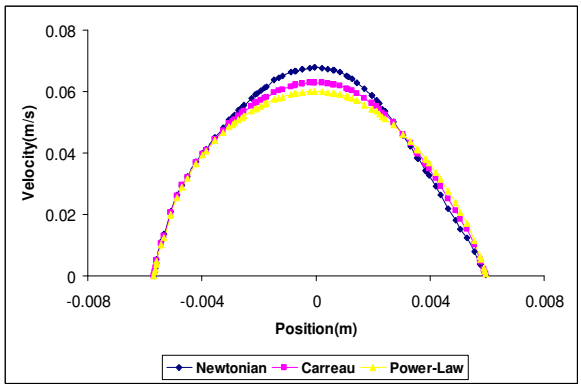


(d)

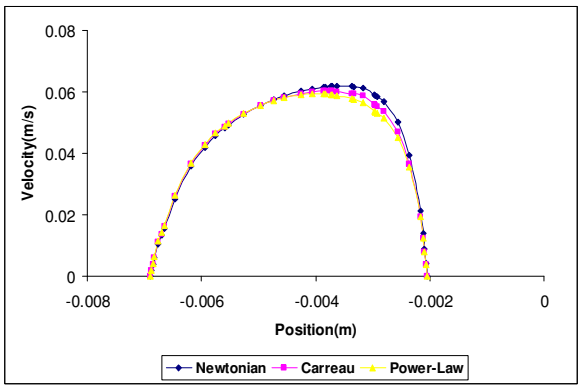
Figure 5 – Blood flow axial velocity profiles at V = 0.01 m/s; (a) Path 1; (b) Path 2; (c) Path 3; (d) Path 4.



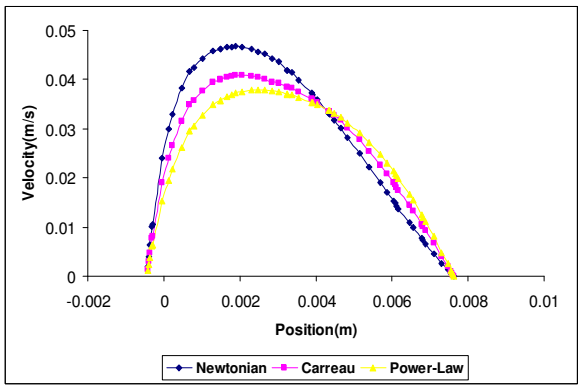
(a)



(b)



(c)



(d)

Figure 6-Blood flow axial velocity profiles at V = 0.05 m/s; (a) Path 1; (b) Path 2; (c) Path 3; (d) Path 4.

At higher inlet velocity (V = 0.05 m/s), the distribution at the CCA main branch (Fig. 4a) exhibits nearly the same profile for the three viscosity models, in contrast with the slower value (V = 0.01 m/s, Fig. 3a). At the ICA (path 3, fig. 4c) and ECA (path 4, Fig. 4d) secondary branches, the axial velocity profiles are flattened and its maximum values are shifted towards the internal side of the carotid bifurcation.

5. FINAL DISCUSSION

At the present work, Newtonian and two non-Newtonian viscosity models were employed to simulate the blood behavior in a carotid bifurcation. It is shown that the non-Newtonian approach is fundamental to capture the blood flow fields at lower velocity values. Under this regime, the blood flow can acquire altered configurations (as stagnant flow regions) and lead to pathological disturbances (such as aneurysms and stenosis).

Numerical simulations confirmed that the carotid bifurcation moves the maximum velocity values towards the internal side of the divider walls (see Fig. 4c and Fig. 4d), changing the symmetric behavior presented in the CCA main branch (see Fig. 3a and Fig. 4a).

Results also shown that the best non-Newtonian model depends on the shear rate (as presented in the Fig. 1). At low velocity values, the Power-law exhibits a better agreement (Fig. 5 and Fig. 6). As the velocity increases, the Carreau model captures better the blood behavior inside the carotid bifurcation.

6. ACKNOWLEDGEMENTS

The first author is grateful to FAPESP (Fundação de Amparo à Pesquisa do Estado de São Paulo) by the financial support to develop this work (grant No. 07/57707-8).

7. REFERENCES

Baaijens, J.P.W., van Steenhoven, A.A., Janssen, J.D., 1993. Numerical analysis of steady generalized Newtonian flow in a 2D model of the carotid artery bifurcation. Biorheology 30, 63-74.

Ballyk, P.D., Steinman, D.A., Ethier, C.R., 1994. Simulation of non-Newtonian blood flow in an end-to-side anastomosis. Biorheology 31, 565-586.

Berger S, Jou L. Flows in Stenotic Vessels. Annual Review of Fluid Mechanics 2000;32:347–382.

Bird, R.B, Stewart, W.E., 1960, "Transport Phenomena", John Wiley & Sons, New York.

Bird, R. B., 1987, "Dynamics of polymeric liquids", John Wiley & Sons, New York.

Brujan E.A., 2000 "Collapse of cavitation bubbles in blood", Europhys. Lett, Vol. 50, pp. 175-181.

Brydson, J.A., (1981), Flow Properties of Polymer Melts, George Godwin LTD, Londres.

Callaghan, S., Walsh, M., McGloughlin, T., 2006, "Numerical modelling of Newtonian and non-Newtonian representation of blood in a distal end-to-side vascular bypass graft anastomosis", Medical Engineering & Physics, vol 28, 70-74.

Cho, Y.I, Kensey, K.R., 1991, "Effects of the non-Newtonian viscosity of blood on flows in a diseased arterial vessel", Part 1. Steady flows, Biorheology.

Delassus, P., Callanan, A., Wallis. F., 2005, "3-D Numerical Simulation of Blood Flow Through Models of the Human Aorta", Journal of Biomechanical Engineering, vol. 127.

Dinnar, URI., (1981), "Cardiovascular Fluid Dynamics", CRC Press, Florida.

Fournier, R. L., 2007, "Basic Transport Phenomena in Biomedical Engineering", Ed.Taylor & Francis, New york, USA, 450 p.

Gijsen, F.J.H, Vosse, F.N, Janssen, J.D, 1999, "The influence of the non-newtonian properties of blood on the flow in large arteries: steady flow in a carotid bifurcation model", Journal of Biomechanics, vol. 32, 601–608.

Johnston, B.M, Corney, S, 2004, "Non-Newtonian blood flow in human right coronary arteries: steady state simulations", Department of Medicine, University of Tasmania, GPO Box 252-34, Hobart, Tasmania 7001, Austrália.

Johnston, B.M, Corney, S, Kilpatrick, D.,2005,"Non-newtonian blood flow in human right coronary arteries: transient simulations", Journal of Biomechanics, vol. 39, 1116-1128.

Kim, C.S., Kiris, C., Kwak, Dochan., 2006, "Numerical Simulation of Local Blood Flow in the Carotid and Cerebral Arteries Under Altered Gravity", Department of Medicine, University of Tasmania, GPO Box 252-34, Hobart, Tasmania 7001, Austrália.

Pedley, T.J., 2002, "Arterial and venous fluid dynamics. In: Computational fluid mechanics", Udine

Perktokl, K., Peter, R.O., Resh, M., Langs, G., 1991, "Pulsatile non-Newtonian flox in three-dimensional carotid bifurcation models: a numerical study of flow phenomena under different bifurcation angles", Journal of Biomedical Engineering 13, 507-515

Rodkiewicz, C.M., Sinha, P., Kennedy, J.S., 1990. On the application of a constitutive equation for whole human blood. Journal of Biomechanical Engineering 112, 198-206.

Sankar, D.S., Hemalatha, K, 2006, "A non-Newtonian fluid flow model for blood flow through a catheterized artery Steady flow", Applied Mathematical Modelling.

Shahcheraghi, N., Dwyer, H.A., Cheer, A.Y., 2002, "Unsteady and Three-Dimensional Simulation of Blood Flow in the Human Aortic Arch", Journal of Biomechanical Engineering, vol. 124.

Shibeshi, S.S, Collins, W.E., 2005, "The Rheology of Blood Flow in a Branched Arterial System", Department of Physics and Astronomy, Howard University, Washington, DC 20059, USA.

Slattery, J. C., 1999, "Advanced transport phenomena", Cambridge University.

Siddiqui,S.U., 2009, "Mathematical Modelling of Pulsatile Flow of Casson's Fluid in Arterial Stenosis", Department of Mathematics, K.N.I.T.,Sultanpur, India.

Tarbell, J.Q.J.M., 2000, "Numerical Simulation of Pulsatile Flow in a Compliant Curved Tube Model of a Coronary Artery", Journal of Biomechanical Engineering, vol. 122.

Urquiza, S.A, Blanco, P.J., Vénere, M.J., Feijóo, R.A., 2006, "Multidimensional modelling for the carotid artery blood flow", Computer Methods Appl. Mech. Eng. 195, 4002-4017.
Versteeg, H.K. e Malalasekera, W., 1995, "An Introduction to Computational Fluid Dynamics", Pearson Education Limited.

8. RESPONSIBILITY NOTICE

The author(s) is (are) the only responsible for the printed material included in this paper.