

PHYSIOLOGICAL PULSATILE WAVEFORM THROUGH AXISYMMETRIC STENOSED ARTERIES: NUMERICAL SIMULATION

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Abstract. *The pulsatile flow of blood through the arterial vasculature generates various types of hemodynamic forces such as wall shear stress, hydrostatic pressures, and cyclic strains that can impact vessel wall structure. Hemodynamic regulates blood vessel structure and it also influences the development of several kinds of vascular pathologies. This factor has an important function in the pathogenesis and pathophysiology of atherosclerosis, which is a disease of arteries where localized deposits and accumulation of cholesterol and lipid substances, as well as proliferation of connective tissues, cause a partial reduction in the arterial cross-sectional area (stenosis). This disease is geometrically focal and it tends to happen in localized sites of complex geometry, such as bifurcations, junctions and high curvatures regions. Wall shear stress (WSS) is an important characteristic that should be considered once that atherosclerosis occurs in regions of low shear stress, because of low mass diffusion of lipids, where blood flow is slow and changes direction, according to the cardiac cycle. In addition, the intimal plaque becomes thicker in these regions. The present study is concerned about the analysis of pulsatile blood flow in a model of an idealized normal artery (no diameter reduction) and in three different configurations of axisymmetric stenosed artery (33%, 50% and 75% of diameter reduction). This study assumes the vessel as a rigid wall tube and the blood flow is considered incompressible, homogeneous and axisymmetric. Newtonian behaviour is assumed. For modeling, Dirichlet boundary conditions were applied at the inlet. A physiological waveform of the femoral artery of a dog was considered as a pulsatile velocity input and with no slip boundary conditions applied at artery wall and pressure boundary conditions applied at the outlet. The physiological waveform was analysed into a Fourier Series with five harmonics showing a good accuracy for this number of harmonic. The CFD code used in this work is based on Finite Volume Method (FVM).*

Keywords: *Numerical Simulation, Hemodynamic, Physiological Waveform, Stenosis, Wall Shear Stress.*

1. Introduction

The pulsatile blood flow through artery vessel implicates in several types of hemodynamic forces that can impact in the vessel wall structure. Hemodynamic forces regulate blood vessel structure and also influence the development of vascular pathologies. This factor has an important function in the pathogenesis and pathophysiology of atherosclerosis (Berger and Jou, 2000). Fry (1968) suggested that regions of high shear stress, which implicates in a direct mechanical harm of the vessel wall, are regions where atherosclerosis occurred. Conversely, Caro et al. (1971) stated that atherosclerosis occurs in sites of low shear stress because of low mass diffusion of lipids. Fung (1997) also presented these discussion. Some studies have demonstrated, according with Caro theory, that intimal plaque becomes thicker in regions of low shear stress. Atherosclerosis is a disease of arteries in which localized deposits and accumulation of cholesterol and lipid substances, as well as proliferation of connective tissues, cause a partial reduction in the arterial cross-sectional area (stenosis). This disease is geometrically focal and it tends to happen in localized sites of complex geometry, such as bifurcations, junctions and high curvature regions and it can severally disturb hemodynamics patterns. It is also associated with low and oscillatory wall shear stress (WSS) in sites where blood flow is slow and changes direction with the cardiac cycle. In the other hand, vessel sites with steady blood flow and high shear stress are comparatively disease-free.

The numerical simulation has been used to study hemodynamic behavior inside the artery vessel in order to identify potential regions of atherogenesis, thrombosis, direct endothelial injury or denudation. Even though a stenosed artery is difficult to represent because of its varied and complex geometry, some simplification can be applied in order to facilitate numerical simulation (Marques et al., 2003). A rigid tube has been adopted in this study, considering three

different types of axisymmetric constriction. The hypothesis of a rigid tube for the vessel is not so far from reality, once the atherosclerosis promotes some reduction in the elastic properties of the vessel wall. Several studies have been performed, in a first approximation, in rigid stenosis and most of them have adopted steady flow (Zendehbudi and Moayeri, 1999). Depending on the constriction severity of the atheroma, the flow behavior in the poststenotic region could be disordered and/or in transition to turbulent, although the presence of a laminar flow condition in the prestenotic site. Studies assuming axisymmetric stenosis and steady flow show that the flow characteristics depend on Reynolds number and stenosis geometry. Long et al. (2001) obtained numerical solution assuming a physiological pulsatile flow through different models of stenosis. They also assumed in the modeling rigid tube, laminar flow and Newtonian fluid. They have found that during the systolic deceleration phase there is a formation and development of flow separation zones in the poststenotic region. In addition, they have demonstrated some factors that influence the poststenotic flows, such as the severity and geometry of the stenosis and the pulse waveform of the flow.

Wall shear stress is another important characteristic that should be considered for this study. The WSS distribution is very important information in order to understand the consequences of flow characteristics on endothelial cells, see Fig.1. Endothelial cells of blood vessels are permanently exposed to shear stress and the range of stress in physiological normal pattern is in between 1,0 to 7,0 Pa. When these values grown up to a range higher than 10 Pa, thrombosis can occurs. For values higher than 35 Pa, direct endothelial injury and denudation occur. On the other hand, a low shear stress with values on the order of $\pm 0,4$ Pa is associated with atherogenesis (Malek *et al.*, 1999). Sites of complex geometry, such as bifurcations or stenosis, which are predisposed to stagnation and recirculation of blood flow, can promote the atherogenesis because of the presence of low shear stress in these regions. In addition, blood flow through arteriovenous fistulae has showed regions of low shear stress, which were in the range of atherosclerosis occurrence (Ortiz and Bessa, 2003).

The present study is concerned about pulsatile flow in a model of a normal artery (no diameter reduction) and in three different models of axisymmetric stenosis (33%, 50% and 75% of diameter reduction). A canine femoral artery blood flow waveform was assumed as an inlet condition. This physiological waveform was analysed into a Fourier Series with five harmonics. In this study it is intended to improve our understanding of hemodynamic in pathological and non-pathological canine artery models. Additionally, there was the intention to identify regions of atherogenesis predisposition due to situations of low shear stress and also to identify potential regions of thrombosis, direct endothelial injury or denudation due to high shear stress.

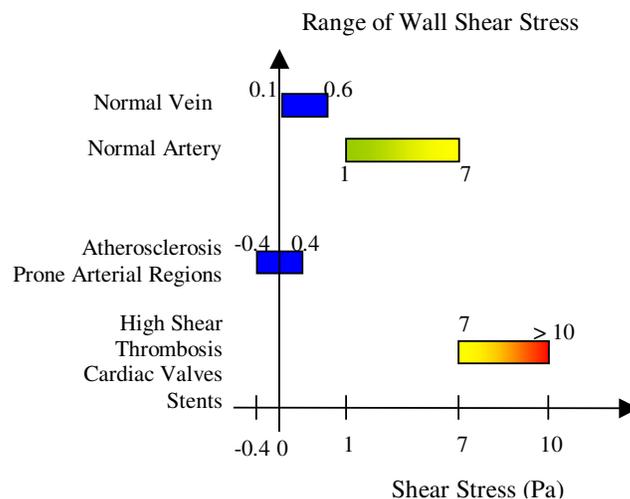


Figure 1. Range of wall shear stress in different vessels associated with different pathologies (Malek *et al.*, 1999).

2. Methods

This study assumes the vessel as being a rigid wall tube and the blood flow is considered incompressible, homogeneous and axisymmetric. Newtonian behaviour is assumed. Four cases have been idealized, one of a normal artery (no diameter reduction) and other three of axisymmetric stenosis (33%, 50% and 75% of diameter reduction). For these models, ideal Dirichlet boundary conditions were applied at the inlet. A physiological waveform of the femoral artery of a dog was applied Fig. 2 (McDonald, 1974). This waveform was considered as a pulsatile velocity input and with no slip boundary conditions and impermeability applied at artery wall. The equation that represents waveform was obtained applying a Fourier Series. By applying the Fourier series on flow waveforms, a trigonometric series can be fitted as an approximation to the actual function. This is achieved by taking a number of readings of equally spaced points that can accurately describe the waveform and apply a numerical Fourier series to the data. A Fourier series was applied to previous data obtained from McDonald (1974):

$$F(t) = \frac{1}{2}a_0 + \sum_{n=1}^{\infty} (a_n \cos(nwt) + b_n \sin(nwt)) \quad (1)$$

where a_0 , a_n e b_n are Fourier coefficients. The first five harmonics were suitable to describe the flow waveform as shown in Fig. 2. The blood flow waveform were represented using 50 data points obtained from their curves. Constant pressure boundary condition was imposed at the outlet, which was placed far enough from entrance (normal artery) or from the constriction site (stenosed artery).

The effects of wall compliance have been neglected in this work. The hypothesis of rigid tube is not so far from reality in this case, considering that atherosclerosis promotes some reduction in the elastic properties of the vessel wall.

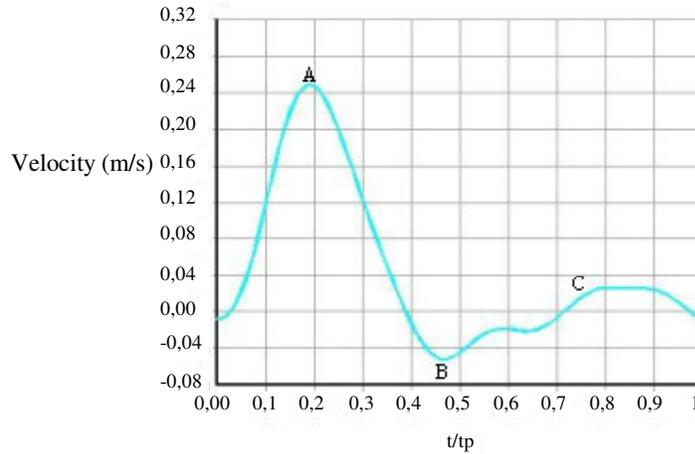


Figure 2. A typical physiological waveform of the femoral artery of a dog

A dimensionless parameter t/tp is introduced to describe a particular point in the cardiac cycle, where t is the specific instant [s] and tp is the period of the cycle [s]. The results were compared considering three different points of the inlet velocity waveform. These three elected points are showed by letters A to C in Fig. 2 and they correspond to values of the dimensionless (t/tp) of 0,19 (A), 0,47 (B) and 0,75 (C).

The governing equations are:

Mass Conservation Equation:

$$\nabla \cdot (\rho \vec{V}) + \frac{\partial \rho}{\partial t} = 0 \quad (2)$$

Momentum Conservation Equation:

$$\rho \frac{D\vec{V}}{Dt} = \rho \vec{g} - \nabla p + \mu \nabla^2 \vec{V} \quad (3)$$

where ρ is the fluid density [kg/m^3], \vec{V} is the velocity vector [m/s], t is the time [s], \vec{g} is the gravity acceleration [m/s^2], p is the pressure [Pa] and μ is the dynamic fluid viscosity [$\text{Pa}\cdot\text{s}$].

The flow rate and pulse were defined according to physiological parameters of Reynolds number and Womersley number. The Reynolds number is a relationship between inertial and viscous forces.

$$\text{Re} = \frac{VL}{\nu} = \frac{4Q}{\pi D \nu} \quad (4)$$

where V is the mean velocity [m/s], L is the characteristic length (in this case is the vessel diameter in m), Q is the volumetric flow [m^3/s] and ν is the kinematic viscosity [m^2/s].

In a pulsatile flow, the pulse rate is determined by Womersley parameter (W), which relates frequency of a pulsatile wave and viscous forces.

$$W = \frac{D}{2} \sqrt{\frac{w}{\nu}} \quad (5)$$

where w is the frequency of a pulsatile wave.

According to the concept of the Stokes layer, the parameter δ_S of Stokes layer thickness or viscous wave penetration is given by:

$$\delta_S = 5 \sqrt{\frac{\nu}{w}} \quad (6)$$

Fluid properties were assumed following literature (Berger *et al.*, 1996). A fluid density of 1050 [kg/m³], a dynamic viscosity of 0.00345 [Pa.s] and a kinematic viscosity of 3.29×10^{-6} [m²/s] were considered. The period of cardiac cycle (t_p) was taken as 0.8 s. The Stokes layer thickness δ_S is equal 0.0032 m, for the simulations presented.

Preliminary simulations were done for all artery models under investigation, to get information in terms of grid and time step independence, which means that all models were independent of the size of the control volume and increment in time (time step). Grid independence was achieved by using 77.000 control volumes and the time step independence was achieved using a time increment of 0,008 s.

A commercially available CFD code (Fluent 6.2 Academic License) was used in this study, based on Finite Volume Method (FVM). The geometry and the discretization grid of each model (stenosed and normal arteries) is given in Fig. 3. The geometrical parameters for all models are as follows: vessel diameter and vessel length equal to 0,006 m and 0,154 m, respectively, for normal artery. Vessel diameter at stenosed site 0,004 m (33% of diameter reduction), 0,003 m (50% of diameter reduction) and 0,0015 m (75% of diameter reduction) and vessel length 0,024 m for all stenosed regions. The stenosis was assumed as axisymmetric. The grid was non-uniformly distributed in the axial direction with fine cells in the stenosis, but uniformly distributed in axial direction in the model of normal artery. In addition, all models were more refined near to the vessel wall Fig. 3.

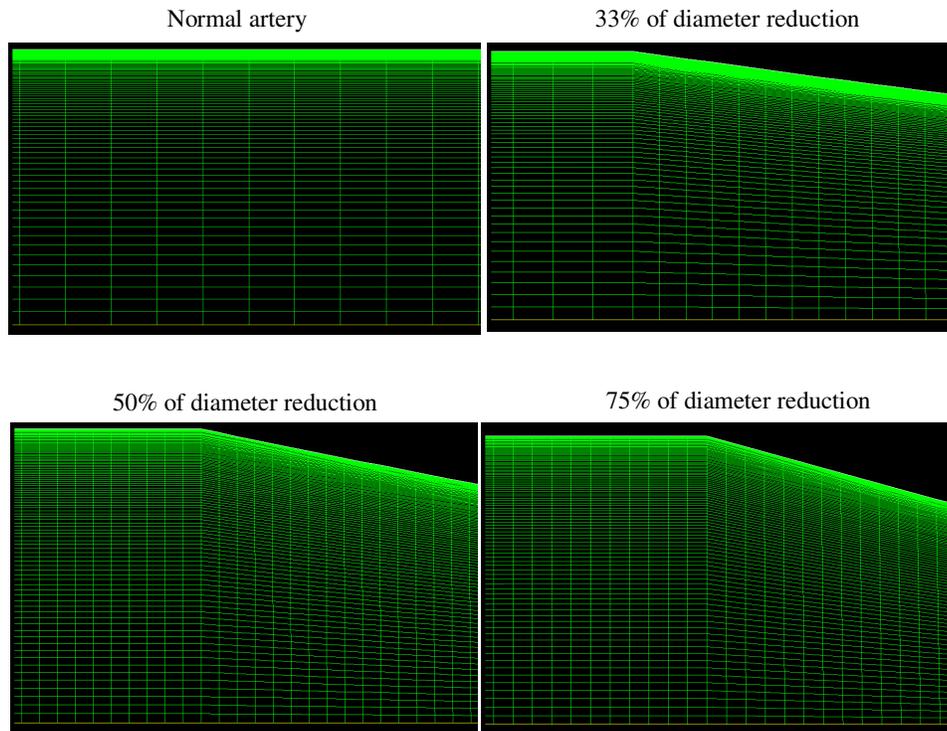


Figure 3. Discretization grid for all normal/stenosed arteries models and its idealized axisymmetric geometries.

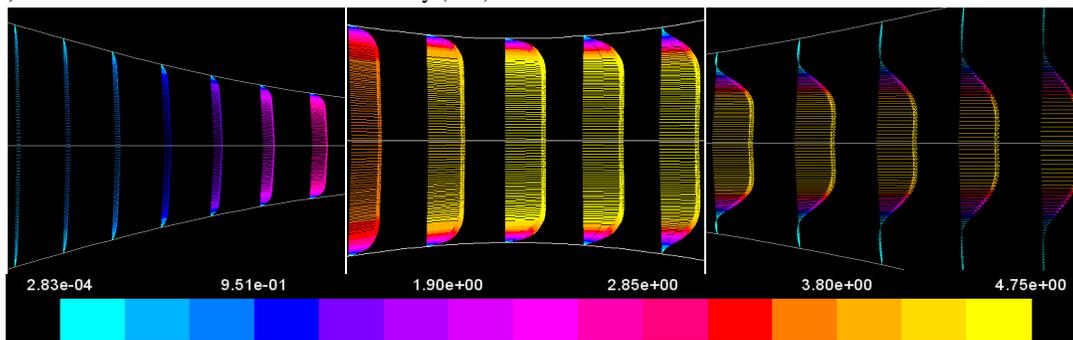
3. Results

The CFD simulations were performed for normal artery (no diameter reduction) and axisymmetric stenosis (33%, 50% and 75% of diameter reduction). Simulations results for flow, including velocity field and WSS distribution obtained for the whole domain, are presented at selected points (A to C) in the cardiac cycle.

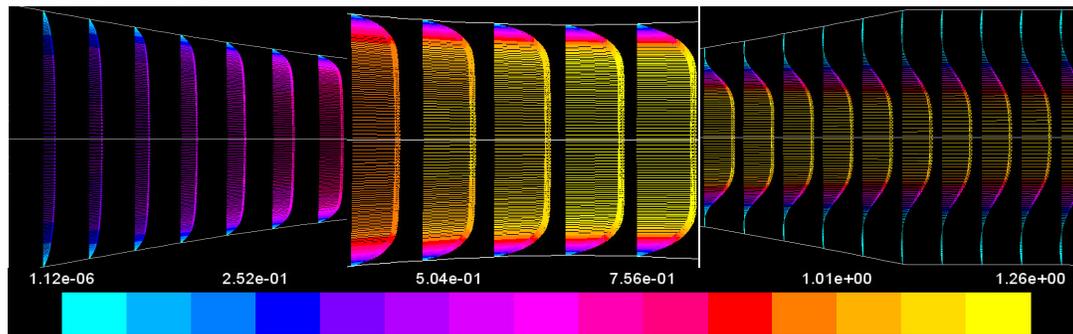
All the stenosed artery models have been divided in two regions in order to compare the velocity field and identify the feature flows between them, and these regions correspond to inlet and outlet of the stenosed site (Fig. 4, 5 and 6).

Figures 4,5 and 6 correspond to a comparison among all stenosed models in terms of velocity field. These models have presented a high velocity field at the stenosed region. A reversal flow occurs for all models at the point B of the cardiac cycle. For the point A of the cardiac cycle, the case of 33% of diameter reduction, it shows that there is a predominance of the main flow compared with the reversal flow. However, the reversal flow region increases according with the increasing of the stenosis. In addition, in the specific case of 75% of diameter reduction, the velocity field has a turbulent profile in the stenosed region, because of the high value of Reynolds number at this site, but it takes some change in the velocity profile at the downstream region. For the point B of the cardiac cycle, the velocity field has almost the same feature for the cases of 33% and 50% of diameter reduction. On the other hand, the case of 75% of diameter reduction has presented a significant change of the velocity field. For the point C of the cardiac cycle, there is a similar velocity field for the cases of 33% and 50% of diameter reduction. Both cases have presented a reversal flow in the prestenotic region. Conversely, the case of 75% of reduction area has showed a reversal flow for almost the whole domain, except for the first half region of the stenosis.

t/tp=0,19 - 75% of diameter reduction – Velocity (m/s)



50% of diameter reduction



33% of diameter reduction

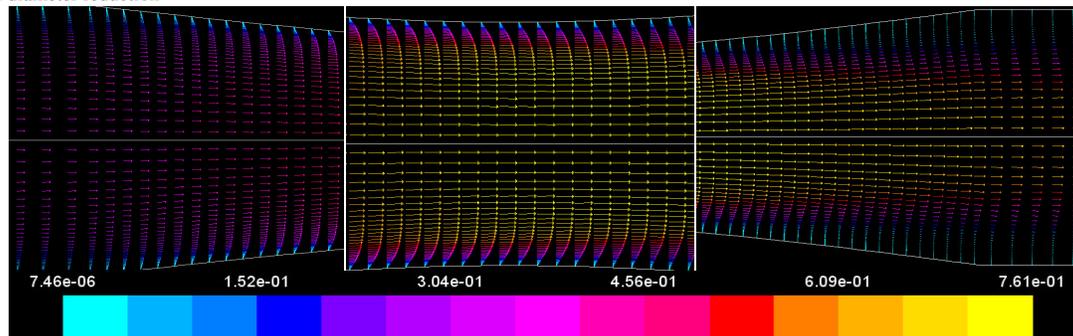
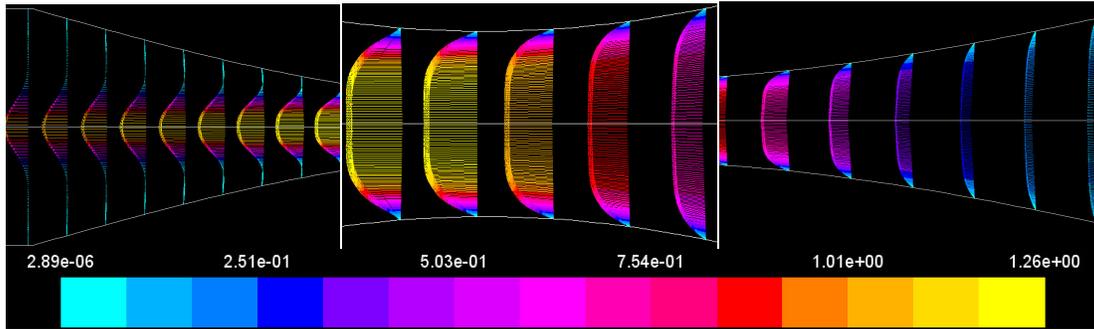
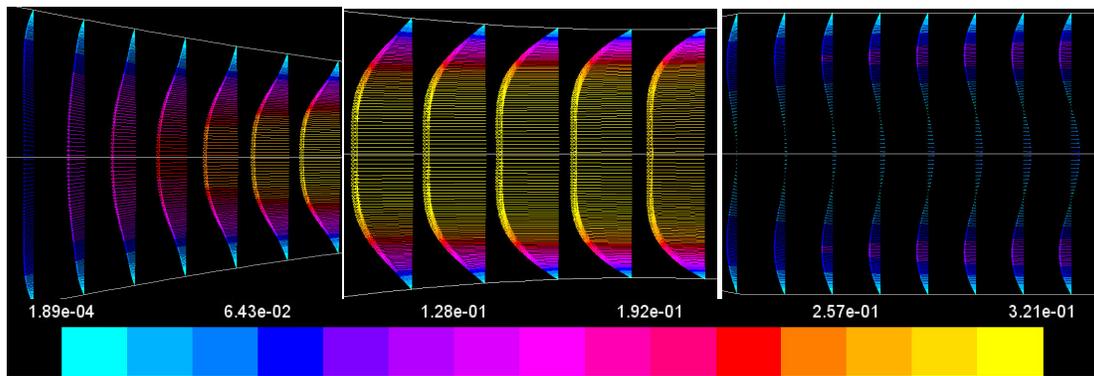


Figure 4. Comparison among velocity field in all stenosed arteries models at point (A) of the cardiac cycle.

$t/t_p = 0,47$
75% of diameter reduction



50% of diameter reduction



33% of diameter reduction

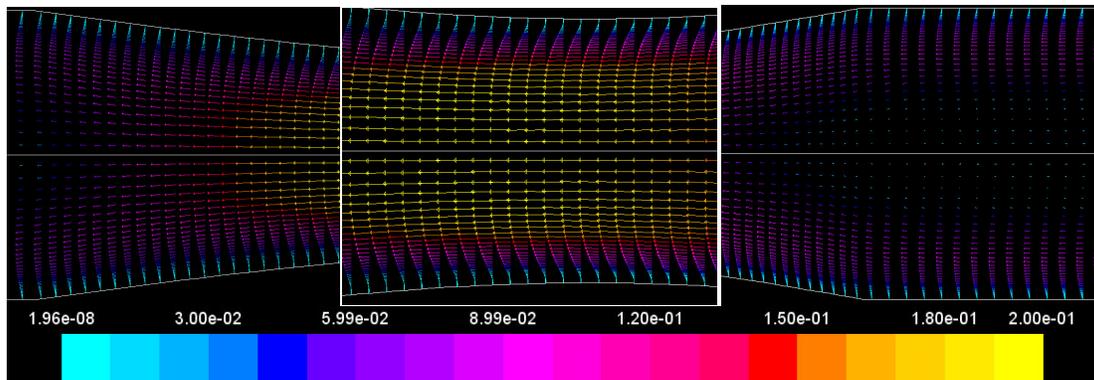
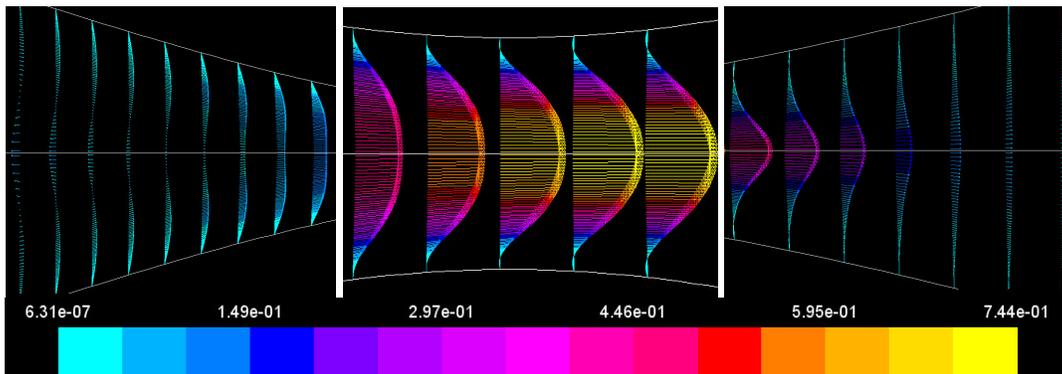
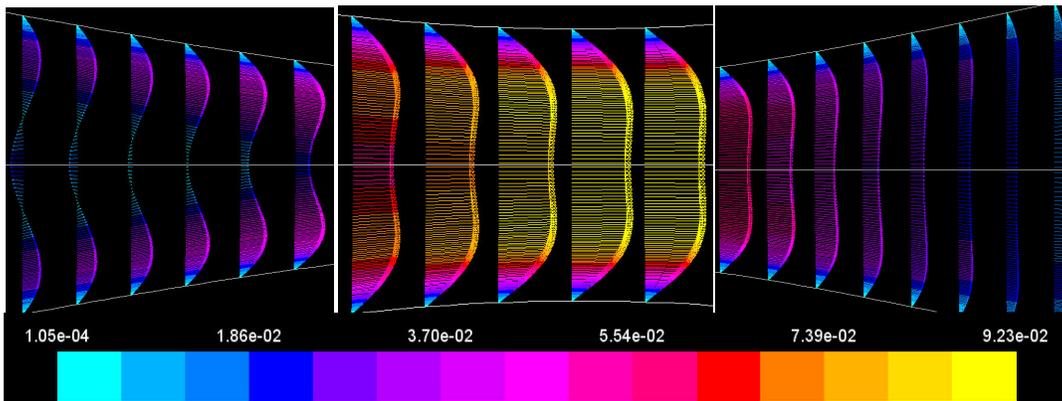


Figure 5. Comparison among velocity field in all stenosed arteries models at point (B) of the cardiac cycle

$t/t_p=0,75$
75% of diameter reduction



50% of diameter reduction



33% of diameter reduction

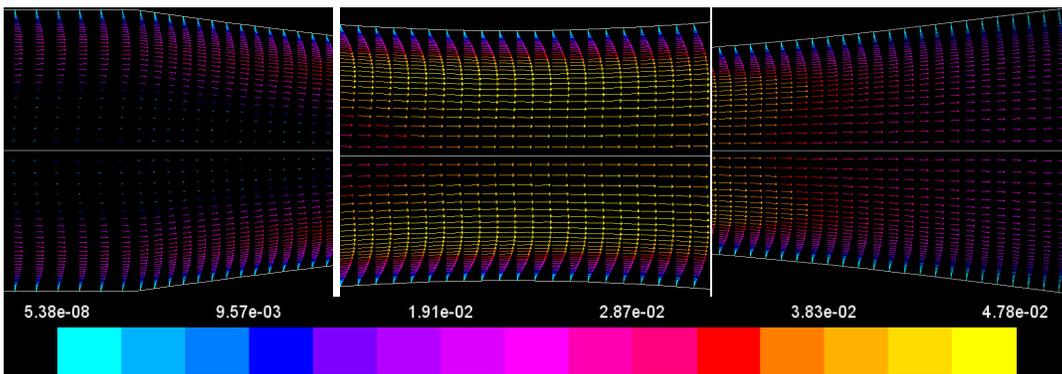


Figure 6. Comparison among velocity field in all stenosed arteries models at point (C) of the cardiac cycle.

In the case of normal lumen (no diameter reduction) some change occurs in the velocity profile, according with the instant of the cardiac cycle (Fig.7).

Normal Lumen (no diameter reduction)

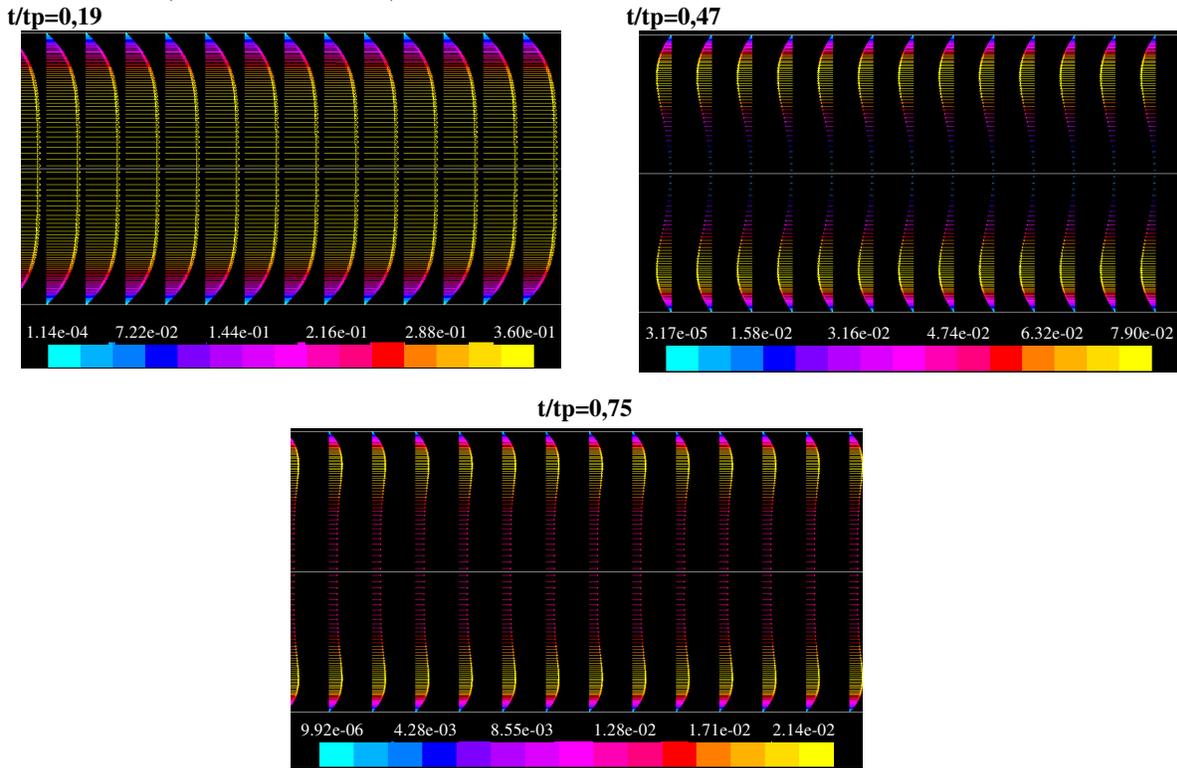


Figure 7. Comparison among velocity field in the normal artery (no diameter reduction) at all points (A to C) of the cardiac cycle.

Figure 8 correspond to a comparison among wall shear stress of all stenosed models at the points A to C of the cardiac cycle, for vessel length equal to 0,154 m. The physiological waveform has highest velocities values at the point A of the cardiac cycle, which means higher values of WSS. In fact, it is noted that the magnitude of the WSS is extremely affected by the artery constriction severity.

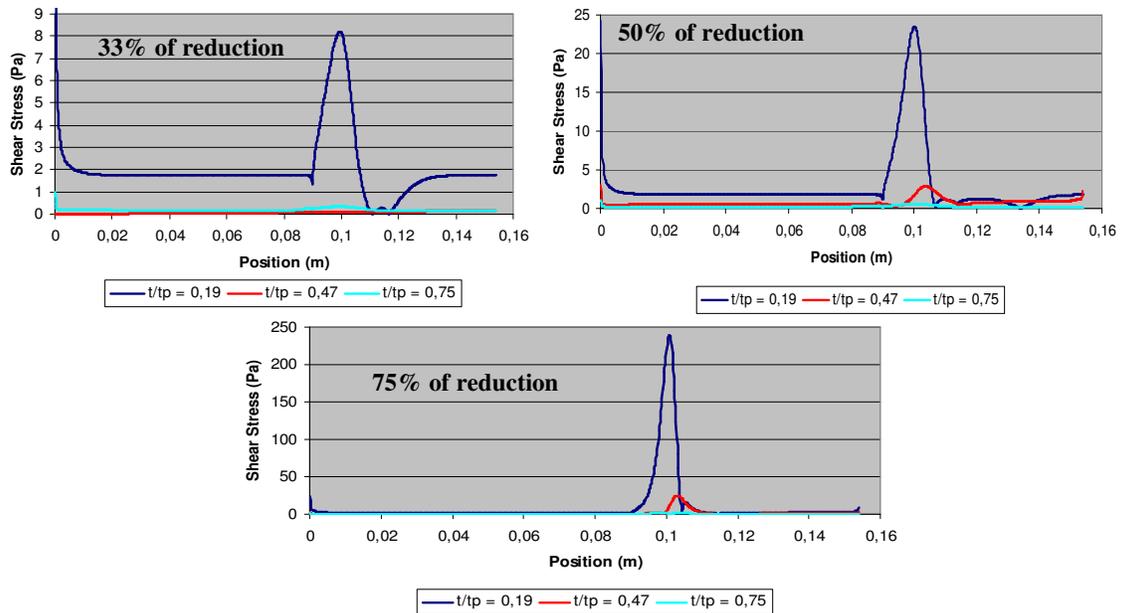


Figure 8. Comparison among distribution of wall shear stress in all stenosed arteries models at all points (A to C) of the cardiac cycle.

4. Conclusions

In this study, numerical results of a pulsatile blood flow through an idealized model of femoral dog artery were presented. The studied cases were performed for a normal artery and axisymmetric stenosed artery with a physiological waveform of the femoral artery of a dog, which matched with a Fourier Series represented with five harmonics. The results include time dependent velocity fields and wall shear stress distributions. In this work some hypotheses were adopted such as rigid vessel wall, incompressible homogeneous Newtonian fluid and axisymmetry.

Since the Stokes layer thickness is of the order of the radius, there is a prevalence of viscous effect in the whole domain. The results show the effect of the unsteadiness in the flow through the artery. Changes of the velocity field happen due to the development of reversal pressure gradient.

It is showed that reversal flow varies according with cardiac cycle phase and also with the constriction severity. Changes the flow feature occur in magnitude and in radial position. The results are strongly time dependent, which means it is affected by the pulsatile flow. The feature of the poststenotic flow will depend on the stenosis geometry and flow characteristic.

The wall shear stress distributions have been observed for the whole domain. These values depend of axial position, cardiac cycle phase, and stenosis geometry. It is a crucial hemodynamic parameter that determines the initiation of atherogenesis. The range of shear stress in physiological normal pattern is between 1,0 to 7,0 Pa, however, the low shear stress observed in this study is of the order of 0,1 Pa, which is associated with atherogenesis. On the other hand, the highest value of WSS found is of the order of 240 Pa, which is in the range of direct endothelial injury and denudation occurrence (higher than 35 Pa).

5. Acknowledgements

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