NUMERICAL STUDY OF BLOOD FLOW IN A VESSEL WITH INCREASING DEGREE OF STENOSIS USING DYNAMIC MESHES

Ana Cristina Ferreira, Filipa Carneiro, Senhorinha Teixeira, José Carlos Teixeira

School of Enginnering, University of Minho, Campus Azurém,

4800-058 Guimarães, Portugal.

Vasco Gama Ribeiro

Centro Hospitalar de Vila Nova de Gaia

Rua Conceição Fernandes – Vilar de Andorinho

4430-502 Vila Nova de Gaia

Abstract

Cardiovascular diseases are, nowadays, the major cause of death in the world. The formation of stenosis in vessels occurs throughout the deposition of arteriosclerotic plaques in the wall vessels, which affects the blood flow profiles. The abdominal aorta and its branches is one of the most expected locations for the development of stenosis. This deposition occurs commonly in larger arteries, but the most probable locations for the development of stenosis correspond to the regions of curvature or branches in the vessels. Therefore, the goal of this study consists in the development of numerical models able to predict the blood flow in the abdominal aorta region. In this study, the simulations were performed through the use of Computational Fluid Dynamic (CFD) tools. This investigation enables the identification of reverse flow area, allowing the analysis of flow patterns, velocity profiles and pressure distribution. The blood was modeled as an incompressible, Newtonian fluid with the viscosity and density values of 0.004 $kg.m^{-1}s^{-}1$ e 1057 $kg.m^{-3}$, respectively. The governing equations were solved through FLUENT software, which requires the domain discretization into control volumes. The simulation was performed using the dynamic meshes to evaluate the effects of the increasing of stenosis degree in the blood flow.

Keywords: Stenosis; CFD; Blood Flow; Dynamic mesh

1. Introduction

Atherosclerosis is a progressive disease initiated through localized fatty streak lesions within the arteries. These lesions can develop into more complex plaques large enough to significantly block blood flow through the arterial tree. This local restriction of the artery is known as an arterial stenosis. Plaque deposition occurs preferentially in a few places in the systemic vasculature, primarily the carotid artery sinus, the coronary arteries, the abdominal aorta and the superficial femoral arteries. As disease advances, so does the severity of stenosis increases. Stenoses are commonly characterized as a percentage reduction in diameter or area of host vessel, which results as an occlusion of flow in the vessel. In clinical terms, stenoses are considered significant when the reduction is near to 70% by area [1, 2].

When arteries become severely diseased, the arterial lumen becomes locally restricted and the flow downstream and upstream can be highly disturbed.

Stenotic flows may feature flow separation, recirculation, as well as strong shear layers that, when combined with flow pulsatility, can result in periodic transition to turbulence in the post-stenotic region. Consequently it is very important to understand how the flow features change with the development of stenoses. Stenotic flows have been characterized in several studies. The initial research about blood flow in stenosed vessels was concentrated on experimental simulations using simple geometries or steady-state axisymmetrical numerical simulations, considering a straight rigid tube with a constriction [2, 3].

A study performed by Young (1979) provided experimental analysis on steady flow through stenosed arteries with varying severity, stenosis length, axisymmetric and asymmetric conditions, and with a range of Reynolds numbers from laminar to turbulent flows. In sum, these studies have shown that, at low fluid Reynolds Number, low degrees of severity tended to have little influence on the flow and a strong shear layer is developed between the central plane and the recirculation region [4]. In addition, blood flow is pulsatile which creates a periodic generation of turbulence.

Many studies included transient conditions in their simulations in order to consider the time varying nature of the flow within arteries and with the goal of evaluate the turbulence effects in the post-stenotic field (Mallinger and Drikakis, 2002; Mittal *et al,* 2003; Sherwin and Blackburn, 2004) [5, 6, 7].Misra *et al* simulated the behavior of flow and the wall response of a stenosed artery incorporating into the simulation fluid harmonic waves and assuming blood as a Newtonian fluid [8]. He and Jackson (2000) observed fundamental aspects of turbulence dynamics: turbulence intensity is attenuated in accelerating flows and increased in decelerating flows mainly associated with the radial propagation of turbulence [9].

Turbulent flow in stenosed pipes has also been numerically studied to analyze flow distal to the stenosis. Varghese and Frankel (2003) simulated pulsatile turbulent flow in a rigid wall stenotic tube. The goal of their study was to predict, through direct numerical simulations, the flow patterns downstream of a stenosis under both steady and pulsatile conditions. The authors concluded that the acceleration of the fluid through the stenosis resulted in wall shear stress (WSS) levels that exceeded those upstream but WSS accompanied the flow separation zones that formed immediately downstream of the stenosis [10].

The application of Computational Fluid Dynamics (CFD) methods has become an important tool in the investigation of blood flow in arteries under normal and pathological conditions. With these methods it is possible to create a computational model of abdominal aorta from which it is possible to predict the flow patterns.

The study reported in this paper focuses upon the application of dynamic meshes to identify the pressure distribution, flow patterns and particularly the reverse flow area in the vicinity of a stenosis.

2. Applying Dynamic Meshes

The dynamic meshes in FLUENT can be used to model flows where the shape of the domain is changing with time due to motion on the domain boundaries and can also be used for steady-state applications. The update of the volume mesh is handled automatically by FLUENT at each time step based on the new positions of the boundaries and input definitions. It is possible to create a starting volume mesh and describe the motion of the boundary using user-defined functions (UDFs) and appropriated boundary conditions.

There are three different types of dynamic mesh topologies, whose parameters and settings can be defined through the definition of moving zones in the model.

The mesh methods available to update the volume mesh in the deforming regions are: Spring-Based Smoothing, Remeshing, and Dynamic Layering. The face Remeshing method

marks the faces that have to be remeshed according to the minimum and maximum length scales and the maximum cell skewness. These faces are mostly close to the boundary conditions that are moving. In the Dynamic Layering, the cells are split or merged using either a constant height or a constant ratio. When using a constant height option the cells are split to create two new layers with different heights.

In the Spring-Based Smoothing method, the edges between two mesh nodes are idealized as a network of interconnected springs. When a movement occurs at the boundaries the nodes that are connected by springs move according to the direction of the force generated. In the FLUENT it is defined a model that contains moving and non-moving regions, which are defined by grouping them into their respective face zones in the starting volume mesh that was previously generated [11].

In order to replicate the increase in stenosis, the method selected was the Spring-Based Smoothing. This method can be used on all types of deforming meshes, triangular or non-triangular elements in the 2D cases. But when using it on non-triangular cells it is recommended that the movement of the cell zones is in one direction and the motion is normal to the boundary. In the spring-based smoothing method the initial spacing of the edges before any boundary motion constitutes the equilibrium state of the and the displacement is defined through a function.

3. Stenosis Model

A schematic representation of the geometry considered in the study is given in the figure 1. The stenosed tube has 100 mm of length and it is composed through different boundary walls: two fixed walls and a moving wall which represents the stenosis perturbation. The fixed wall upstream the stenosis (FIXED WALL1) is 24 mm in length while that downstream the stenosis (FIXED WALL 2) has a length of 44 mm. The moving wall is defined through an arc to represent stenosis. The value of axial distance of the moving wall is 32 mm and, the radial distance from the centre of stenosis, *h*, is 2.5 mm. The radius of the vessel, *R*, is 11 mm.

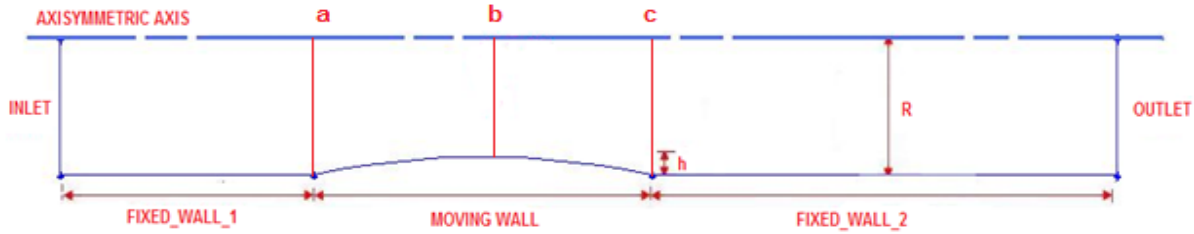

Figure 1. Geometry of the stenosis model showing the location of obstructed (b) cross sectional area and those undisturbed upstream (a) and downstream (c).

The severity of stenosis is determined by the amount of the cross section area that has been obstructed. The vessel section represented is obstructed by a stenosis with a height which can be defined through the relation $\alpha = h/R$, where *h* can be defined as the maximum height of the stenosis. If the unobstructed radius of the vessel, R', is defined to have a unit length, $R' = 1$, the height of stenosis vary between $0 < \alpha < 1$. Consequently, the percentage of the area blocked can be obtained and, therefore the degree of the stenosis can be calculated as:

$$\%\quad Stenosis\ Degree = \frac{\theta - \theta(1-\alpha)^2}{\theta} 100\% \tag{1}$$

where θ represents de vessel's diameter.

At for the initial conditions, the obstruction height, h, considered in the geometry is 2.5 mm, which gives a stenosis severity of 40%.

4. Numerical Procedure

4.1 Grid Generation and Boundary Conditions

The geometry and the computational grid were generated using the GAMBIT software. The mesh scheme generated in the geometry was influenced by the requirements implicit in the dynamic methods during the mesh motion. This includes an additional spacing between the edges before any boundary motion. And, at the same time, it is necessary to reach the equilibrium state mesh.

The fluid was considered incompressible, Newtonian, with a viscosity of 0.004 kg/m.s and a density of 1057kg/m3. The inlet flow was assumed to be fully developed which can be defined by

It was used the following parabolic equation:

$$U = -\frac{U_{\max}}{\delta^2} y^2 + U_{\max} \tag{2}$$

Where *U* is the inlet velocity in the axial direction (m/s), $U_{\max}$ is the maximum axial velocity (approximately 0.234 m/s), δ is the radius (m) and y is the coordinate position in the radial direction.

The maximum value of the velocity was obtained through the curve of pulsatile velocity of during the cardiac cycle at infra renal aorta obtained by Taylor & Draney (2004) [12].

The profile described by equation (2) was then written in the C programming language and this function was then interpreted as an UDF by the FLUENT. The outlet boundary condition was considered as outflow. The motion of the moving wall described in figure 1 was also described by means of a UDF using the dynamic mesh model. The UDF code defines the mesh motion through three dynamic zones, which correspond to the *cg motion* of moving wall and the geometry definition for both fixed walls. The Spring-Based Smoothing was used to update the volume mesh in the deforming zones. The spring constant factor was defined as 0.001.

4.2 Physical formulation

The turbulence generated was simulated with the k-ε model with enhanced wall treatment. The mathematical formulation of the blood flow is imposed by the conservation equations for mass (equation 3) and momentum (equation 4):

$$\nabla \vec{v} = 0 \tag{3}$$

$$\frac{\partial}{\partial t}(\rho\vec{v}) + \nabla.(\rho\vec{v}\vec{v}) = -\nabla p + \nabla(\bar{\bar{\tau}}) \tag{4}$$

where $\vec{v}$ is the fluid velocity vector, ρ is the density, p is the static pressure and $\bar{\bar{\tau}}$ is the stress tensor.

To solve the continuity, momentum and the turbulence equations, the FLUENT software was used. FLUENT uses the finite volume method, which requires a discretization of the domain into control volumes. In the finite volume method, the partial equations are approximated by a set of algebraic equations that are written over the computational domain and then solved.

Solutions are obtained iteratively using the segregated solver with the SIMPLE algorithm and it was used the standard scheme to solve pressure equation. The first order upwind scheme was chosen because dynamic mesh simulations currently work only with first-order time advancement [11]. The convergence is accepted when residuals are below 1e-04.

5. Numerical Results

Figure 2 shows the velocity flow distribution, through the geometry model before the implementation of the mesh motion in the stenosis wall. The initial height of the obstruction before remeshing corresponds to a stenosis severity of 40%.

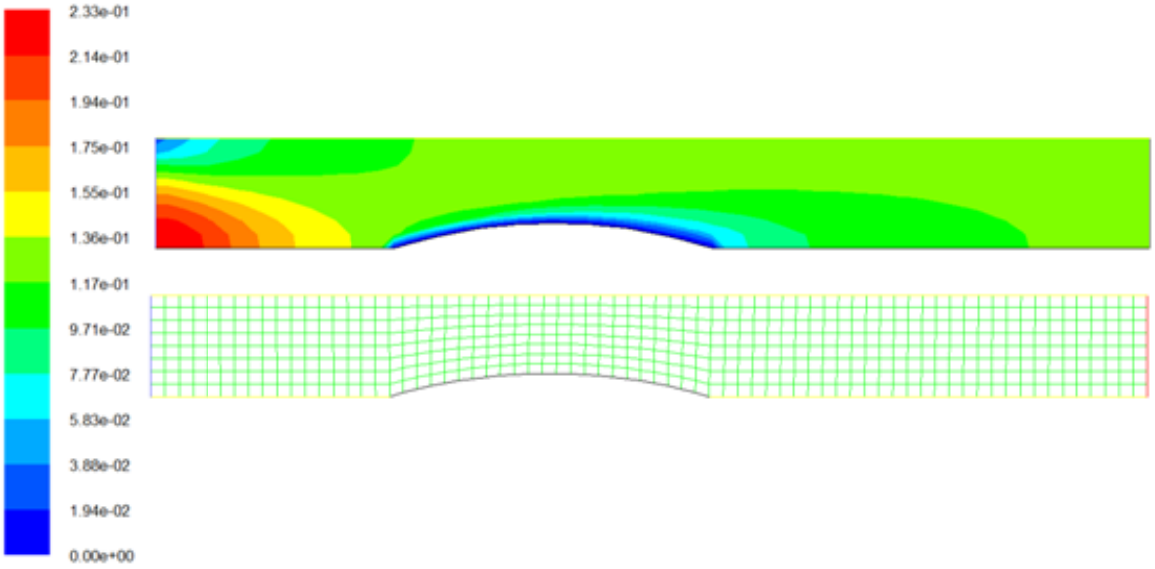

Figure 2. Contours of axial velocity distribution and mesh motion update at model before the changing of the grid, which corresponds to a stenosis severity of 40%.

The velocity inlet parabolic profile was studied and the axial velocity distribution was obtained and compared for various degrees of stenosis. According to Malek *et al,* in clinical, stenoses are considered significant when the reduction (by area) is near to 70% in large arteries [3]. In these cases, the blood flow is drastically affected. So, during the simulations, three different stages in the mesh motion were selected, corresponding to distinct degrees of stenosis: 60%, 70%, and 80%. The application of dynamic mesh compels a displacement at the boundary nodes generating a proportional force along all the springs connected to the node that results in a volume control update for each time step.

Figures 3 through 5 show the distribution of axial velocity and corresponding grid modification resulting from an increasing degree of stenosis.

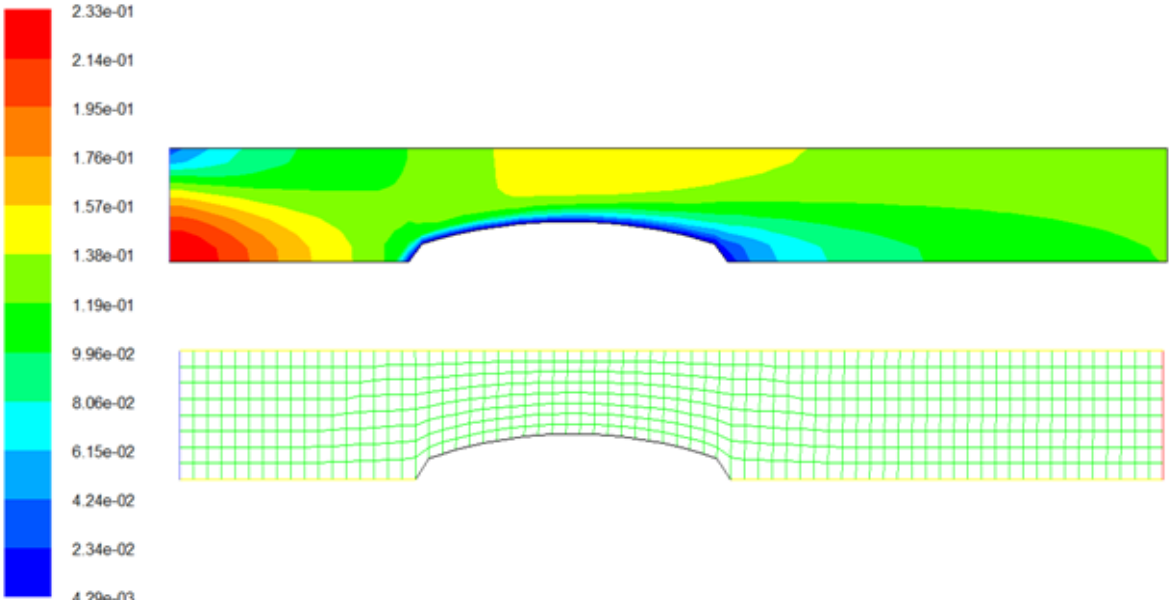

Figure 3. Contours of axial velocity distribution and mesh motion update for a stenosis degree of 60%.

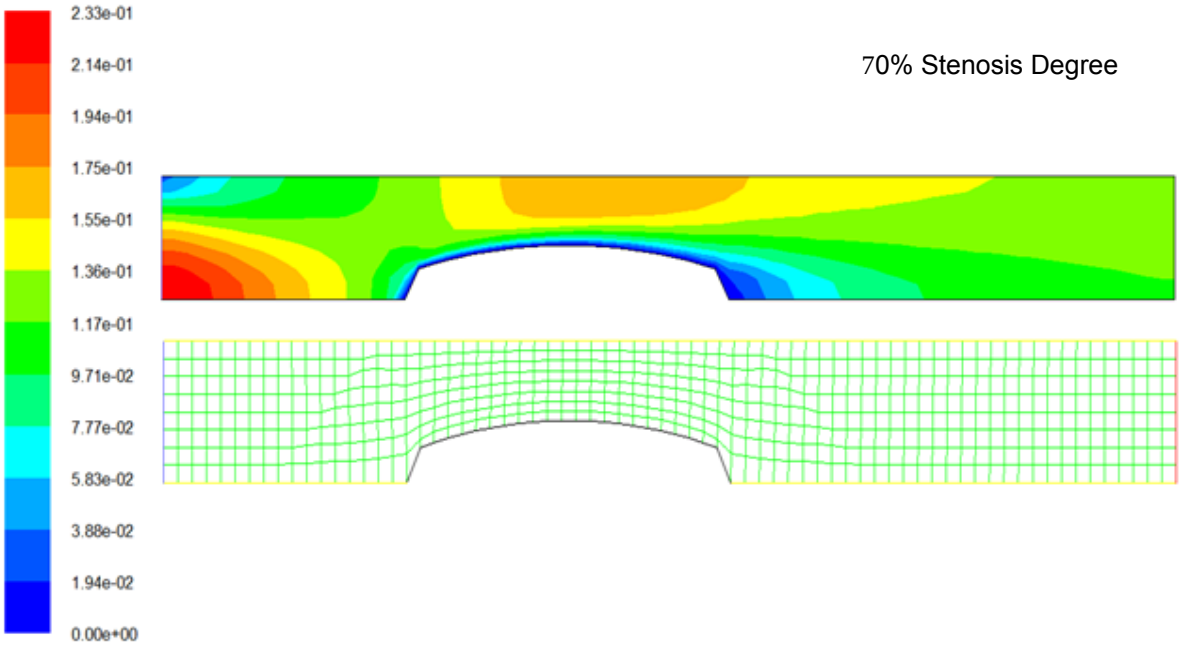

Figure 4. Contours of axial velocity distribution and mesh motion update for a stenosis degree of 70%.

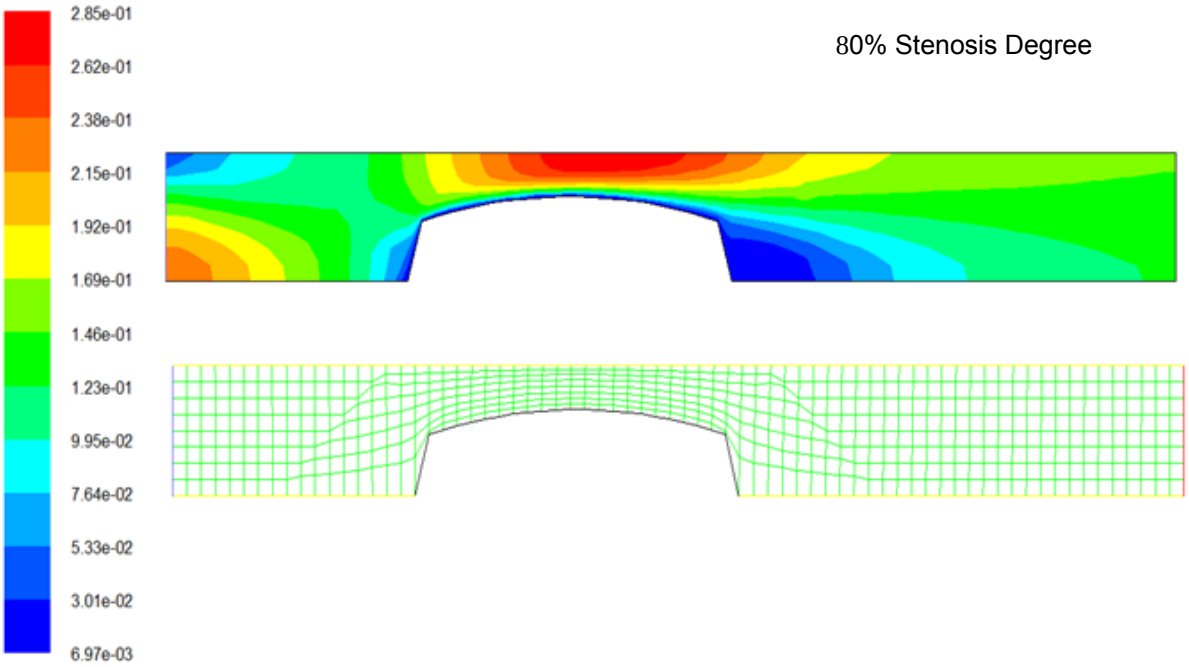

Figure 5. Contours of axial velocity distribution and mesh motion update for a stenosis degree of 80%.

Relatively to the analysis of the velocity distribution, the results from current simulations show that, when applied an inlet parabolic velocity profile, the velocity increases in the central region next to the throat. This increases with the degree of the stenosis. At the centerline of the obstructed vessel zone, the velocity increases from 0.157 m/s when the obstruction is 60% up to 0.285 m/s when the obstruction reaches 80%. The results also show the occurrence of regions of low velocity, mainly on the wall downstream ~~to~~ the obstruction. Such low values are responsible for the formation of recirculation zones in the arterial blood flow, and consequently, may originate the deposition of arteriosclerotic plaques in the wall vessels.

Downstream of the stenosis, flow may become transitional because of the sudden expansion and the possibility of occurrence of recirculation and flow disturbance. Consequently, and besides the lower velocities considered in the inlet velocity profile, it was taken in account the effects of turbulence.

The axial velocity distribution at axial positions a, b and c (see figure 1) for an inlet parabolic profile is shown in figure 6. The axial velocity distribution is compared for the three different degrees of stenosis in order to evaluate the influence of increasing of stenosis degree in the axial velocity distribution at those regions.

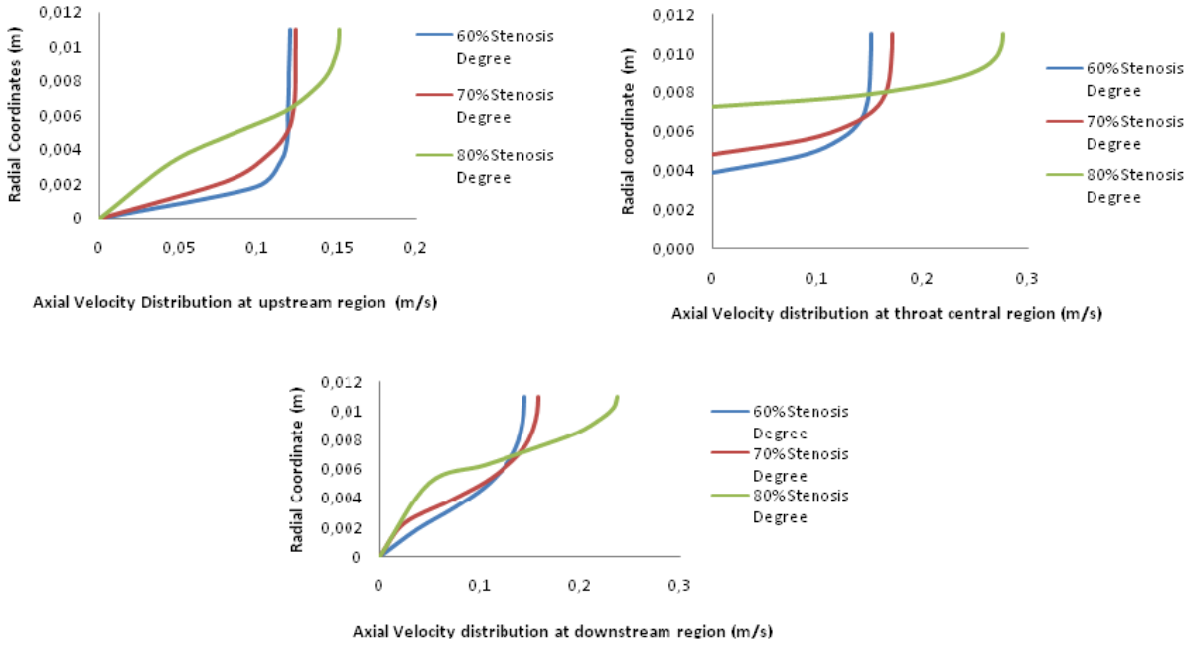

Figure 6. Axial velocity distribution at three axial positions: upstream, stenosis throat and downstream region.

The axial velocity distribution at upstream region of throat is nearly identical for the two lower degrees of stenosis considered in this study, 60% and 70% respectively. At 80% stenosis degree, the magnitude of the velocity increases significantly. The same occurs downstream of the stenosis where the velocities values are higher and the profiles are distinct. At the throat of the stenosis the the maximum velocity reaches 0.028m/s, when the stenosis has an obstruction of 80%.

Figure 7 shows the variation of the pressure along the centerline of the tube. This gives an insight to the influence of the stenosis in the pressure distribution in the vicinity of the obstruction.

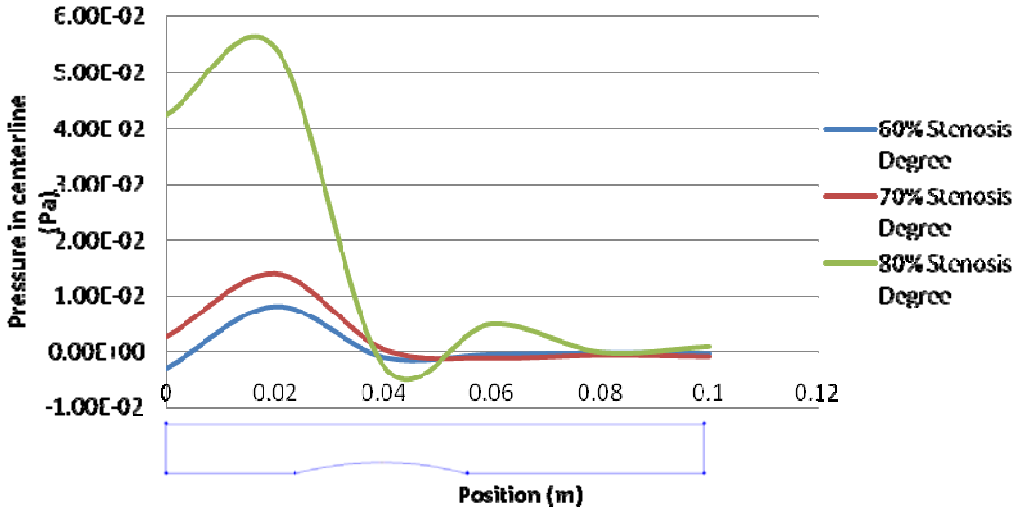

Figure 7. Variation Pressure (Pa) in centerline along the pipe.

For lower levels of obstruction (60% and 70%), the pressure profile behaves in a similar way. The increase of the pressure occurs just upstream of the reduction of the cross section area. Downstream the throat, the pressure recovers. However, further reduction in the available cross section area (stenosis of 80%) induces a steep rise in the pressure upstream of the obstruction and a strong oscillation in the profile, which includes the occurrence of a low pressure. These results suggest that the higher the stenosis degree, the higher is the local pressure drop and a more likely occurrence of cardiovascular stresses.

6. Concluding Remarks

The present study shows that the stenosis affects the distribution of the velocity profiles across the stenosed section. In addition, higher degrees of obstructions prove that severe stenosis causes considerable drop pressure across the obstruction. In addition the occurrence of low velocity regions creates favorable conditions for plaque deposition.

This work proves that an appropriate meshing of the domain enables the modeling of deformable boundaries. This approach can be used to study the flow over deforming walls.

Several assumptions were made in this study. This work considered an idealized straight trunk of the artery and the inlet velocity profile is considered to be fully developed, which in real conditions the anatomic geometry of the artery can be curved. Regarding the fluid side, blood was treated as a Newtonian fluid because this study is concerned in large arteries. However, some researchers have shown that when the shear rate approaches $1200s^{-1}$over the cardiac cycle, which means that blood in some stages of the cardiac cycle may behave as a Non-Newtonian fluid [13].

Moreover, the elastic properties of the wall vessels were not considered yet. As a result, in the future work, the objective is also to comprise the effects of wall elasticity in boundary conditions of the model.

References

[1] Ku, D., “Blood flow in arteries”. Annu. Rev. Fluid Mech. Vol. 29, 1997, pp. 399-434

[2] Berger, S. A. *et al.,* “Flows in Stenotic vessels”. *Annu. Rev. Fluid M*ech., Vol. 32, 2000, pp. 347-382.

[3] Malek, A.M., Alper, S.L.A., Izumo, S. Hemodynamic Shear Stress and its Role in Atherosclerosis. Journal of American Medical Association Vol. 282, 1999, pp.2035-2042.

[4] Young, D. F., “Fluid mechanics of arterial stenosis”, *Journal of Biomechanics*, Vol 101, 1979, pp. 157-173.

[5] Mallinger, F. and Drikakis, D., “Instability in three-dimensional unsteady stenotic flows”, *Int. Journal Heat Fluid Flow,* Vol 23, 2002, pp. 657–663.

[6] Mittal, R. et al., “Numerical study of pulsatile flow in a constricted Channel”, *Journal of Fluid Mechanics* . Vol 485, 2003, pp. 337–378.

[7] Sherwin, S. J. & Blackburn, H. M., “Three-dimensional instabilities of steady and pulsatile axisymmetric stenotic flows”, *Submitted to J. Fluid Mech*, 2004

[8] Misra, J. C. and Chakravaty, “S. Flow in arteries in the presence of stenosis.” *Journal of*

Biomechanics, Vol. 19, 1986, pp. 907-918.

[9] He, S. and Jackson, J., “A study of turbulence under conditions of transient flow in a pipe” *J. Fluid Mech.* 2000, Vol. 408, pp.1–38.

[10] Varghese S. *et al.*, “Numerical Modeling of pulsatile turbulent flow in stenotic vessels”, *Journal Fluid Mechanic,* Vol 125, 2003, pp445-460.

[11] FLUENT, Fluent User’s guide. URL: (www.fluent.users.com) *accessed in January 2009*.

[12] Taylor, C.A., Draney, M.T. “Experimental and computational methods in cardiovascular fluid mechanics”, *Annual Review of Fluid Mechanics*, 2004, Vol. 36, pp.197-231.

[13] O’Callaghan *et al*, “Numerical modelling of Newtonian and non-Newtonian representation of blood in a distal end-to-side vascular bypass graft anastomosis”, *Medical Engineering and Physics*, 2006, Vol. 28, pp.70-74

Corresponding Author

Ana Cristina Magalhães Ferreira

Institution: Universidade do Minho

Phone +351 965856075

E-mail: cristina_ferreira0@hotmail.com