

# 3D SIMULATION OF THE BLOOD FLOW IN ARTERIAL STENOSIS

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## REZUMAT

Elementul de bază în analiza curgerii 3D îl constituie evidențierea zonelor de desprindere și reatașare a jetului de sânge de pereții vasului sangvin, respectiv determinarea poziției punctelor de stagnare ce apar la perete. Partea cea mai delicată a structurii curentului de fluid în modelul 3D este generarea și propagarea regiunii de recirculare ce se formează după zona stenozată. Această regiune situată în imediata vecinătate a zonei de minimă secțiune, caracterizată prin indicatori hemodinamici neuniformi, concretizați prin valori mici ale tensiunii pe perete, respectiv gradienti mari de viteză și presiune, coroborată cu acumularea și depozitarea particulelor de sânge în zona de recirculare, constituie principala cauză, atât în producerea aterosclerozei, cât și a formării trombilor. Mai mult, formarea zonei de recirculare, corelată cu disfuncționalitatea pereților arteriali, joacă un rol esențial în generarea trombozei.

**Cuvinte cheie:** hemodinamică, curgere turbulentă, simulare numerică, expansiuni bruște

## ABSTRACT

The basic features scrutinized during the analysis of the 3D flow in blood vessels are the flow separation and reattachment at the vessel wall where two stagnation points appear. An interesting aspect of the flow structure in a 3D stenosis model is the generation and propagation of a vortex, downstream of the constricted region. Specifically, the regions near the expansion wall and the reattachment point are susceptible to both atherosclerotic lesion and thrombi formations, as indicated by non-uniform hemodynamic indicators, such as near-zero wall shear stress and elevated wall shear stress gradients, as well as blood particle accumulation and deposition. Furthermore, disturbed flow regions, correlated with arterial wall dysfunctions, play an important role in the thrombosis generation.

**Key Words:** hemodynamics, turbulent flow, numerical simulation, sudden and smooth expansions

## INTRODUCTION

Stenotic flows are crucial to clinical disease in several other areas. After the plaque cap ruptures, the revealed contents of the atheroma stimulate a blood clotting reaction called thrombosis.<sup>1-4</sup> For the arterial system, thrombosis is initiated by the adherence of platelets at the surface with rapid accumulation of additional platelets. Each fluid mechanics aspect plays

a role in the generation, detection, and treatment of arterial disease.

There is a wide variety of theoretical pipe flow applications in engineering.<sup>5-8</sup> Such analytical studies can also be helpful in the understanding of physiological flows.<sup>3</sup> In either case, the competition of many different factors increases the difficulty of producing a robust mathematical model.

Blood flowing through a vascular segment exerts a tangentially directed shear stress on the luminal surface of endothelial cells, the inner layer of the vascular wall. Wall shear stress is the product of wall shear rate and local blood viscosity. Wall shear stress has been shown to be an important determinant of endothelial cell function.<sup>4,9</sup>

The stenotic vessel geometry is varied and complex, in this situation the stenosis is frequently simplified as a symmetrical or asymmetrical constriction in a cylindrical tube. In general, flow through constricted pipe is characterized by high velocity jet generated in the constricted section and flow separation downstream to the stenosis.

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## MATERIAL AND METHODS

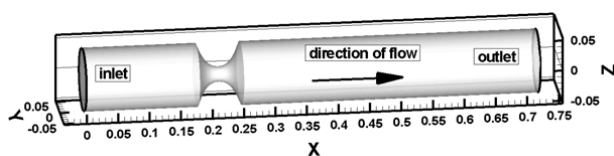
Most studies of artery hemodynamics use experimental measurements, potential flow theory, or computational fluid mechanics. The three dimensional (3D) nature of many of these unsteady flows is a serious obstacle to computational methods because the computational time required is enormous.<sup>10</sup>

However, most computations to date have focused on laminar flow regime since computational modeling of such flows in the transitional and turbulent regimes is a difficult proposition, because both the pulsatile nature of the flow and the relatively low Reynolds number result in a flow that is far from being a fully developed turbulent flow.<sup>1,3,8</sup> Some support for this assertion comes from recent work which indicates the limitations of the predictive capability of existing turbulent models for unsteady/pulsatile flows.<sup>4,7</sup>

The relatively simple geometry shown in Figure 1 produce a very complicated viscous flow field which can be realistically investigated only by using Computational Fluid Mechanics. As pointed in recent publications, the flow in stenotic vessel is not suited to classical mathematical analysis.<sup>5,6,10,11</sup>

The study was performed at the National Center for Engineering with Complex Fluids, from the Polytechnical University, Timisoara, by a multidisciplinary research team of fluid mechanics, engineers and cardiology researchers. The problem of the blood flow in the vessels was simulated using the professional commercial software FLUENT 6.0.<sup>12</sup>

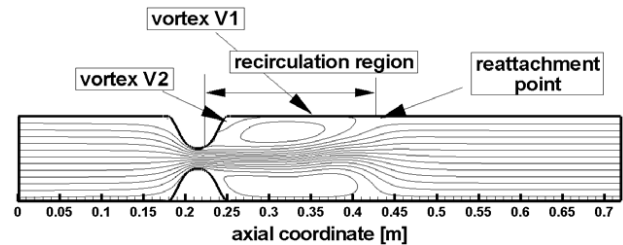
The fluid is assumed incompressible and Newtonian, and the walls were rigid with no slip conditions. At the inlet boundary a fully developed axial velocity profiles is imposed and a constant pressure boundary was imposed at the outlet which was placed sufficiently far from the constricted section. For performing the simulations fixed time step is used. The geometry of the 3D model is given in Figure 1. Structural meshes are employed for the 3D computational domains. In 3D model we used hexahedral cells for computational domain discretization. The Reynolds averaged Navier-Stokes equations, with k- $\epsilon$  turbulence model and pressure-enhanced wall functions were solved.



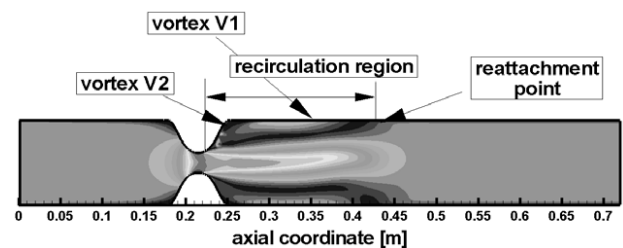
**Figure 1.** Computational domain for realistic 3D stenosis model.

## RESULTS

A typical streamline pattern for liquid flow through the stenotic vessel is presented in Figure 2. Two vortices are developed in the poststenotic region. Vortex V1, rotating counterclockwise and V2 rotating clockwise, respectively, and are increasing between the vessel wall and the jet generated in the constricted region.

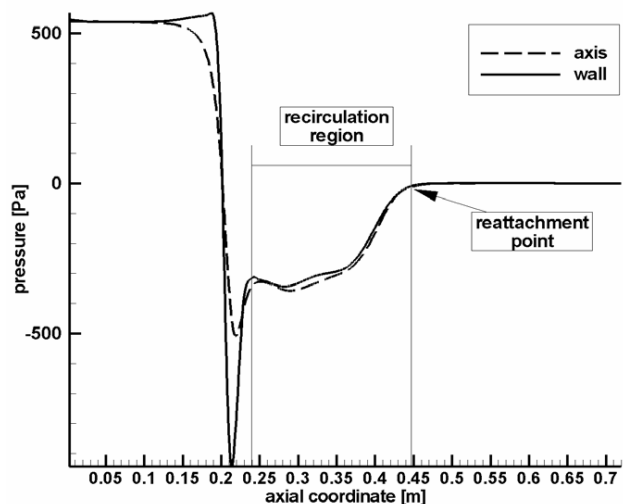


**Figure 2.** Streamlines plot corresponding to the 3D model in longitudinal section plane.



**Figure 3.** Velocity contour plot corresponding to the 3D model in longitudinal section plane.

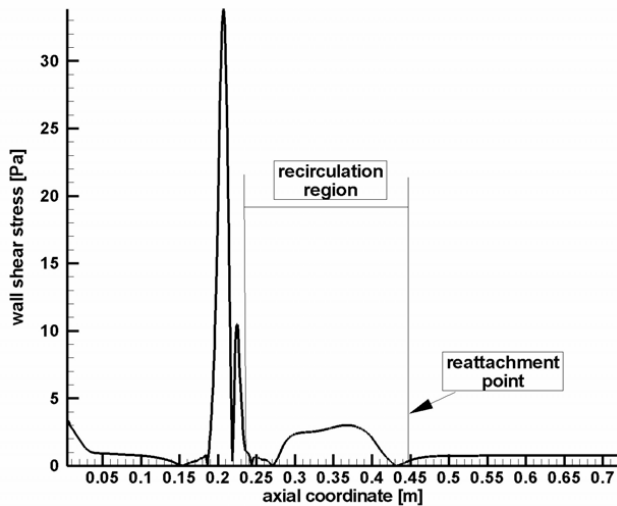
In the contraction section, (Fig. 3) the blood can accelerate to high speeds and produce high gradient velocity field. In this situation, the external pressure may be greater than the internal fluid pressure, and the artery could collapse. A smaller recirculation zone (Vortex V2) is associated with the separation of the



**Figure 4.** Pressure distributions in stenosed vessel (axis of the vessel - dashed line and vessel wall - solid line).

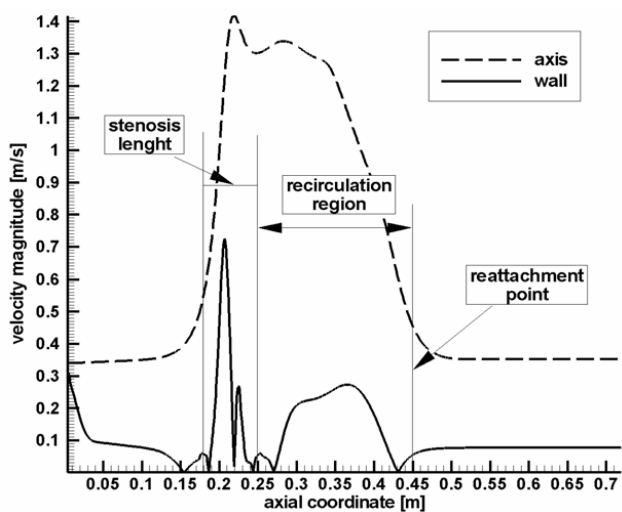
boundary layer from the bottom wall. The formation and propagation of the recirculation zone is one of the most important phenomena in the poststenotic region.<sup>1,4,11</sup>

Figure 4 shows the pressure distribution in stenosed artery. The variation of the pressure distribution can be interpreted directly in the context of the mean streamline pattern shown in Figure 2.



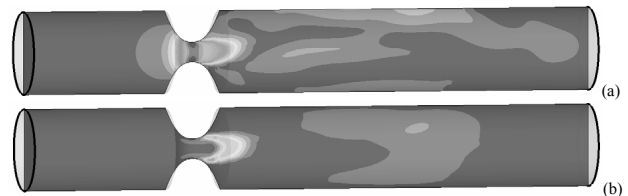
**Figure 5.** Wall shear stress distribution in 3D stenosed artery.

First, the flow enters in the constriction and the increase in velocity (Fig. 5) results in a steep drop in the pressure coefficient which reaches the minimum value at the constricted region. (Fig. 6) Beyond this point, the flow enters the expansion and the pressure coefficient exhibits a step increase. However, because of this strong adverse pressure gradient, the boundary layer on the wall separates at an axial coordinate of about  $x=0.23$  m. Pressure losses generally become significant only for stenoses greater than 50–75% and depend on orifice shape and upstream Reynolds number.<sup>8,13,14</sup>



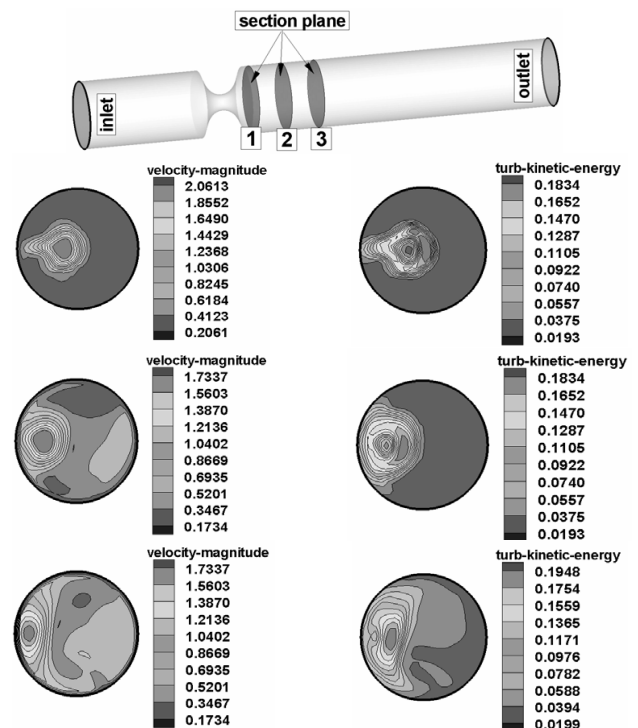
**Figure 6.** Pressure distributions in stenosed vessel (axis of the vessel - dashed line and vessel wall - solid line)

Separation of flow can also contribute to pressure loss and is a major factor at lower percent stenoses. Thus separation will occur in most arterial stenoses. A strong shear layer at the interface of the separation region and the central jet creates additional viscous losses. The throat of the stenosis is a site of high shear rates at the wall, which also contribute to viscous losses.



**Figure 7.** a) Contour plot of the velocity magnitude, b) Contour plot of turbulent kinetic energy (longitudinal section plane in 3D stenosed model).

For low-grade stenoses, the wall and separation shear layers produce most of the losses. For high-grade stenoses, turbulence is the major loss mechanism.<sup>8</sup> This shear layer separates and begins to roll up into a counterclockwise rotating vortex which is observed to attach to the upper wall at about  $x=0.44$  m (Figure 4). Furthermore, the increasing velocity in the stenosis region induces a relatively intense vorticity layer on the bottom wall which is also observed to separate and lift up from the bottom wall. (Fig. 7)



**Figure 8.** Flow characteristics in different transversal section plane. Left - contour plot of velocity magnitude [m/s]; right - contour plot of the turbulent kinetic energy [m<sup>2</sup>/s<sup>2</sup>].

Figure 8 shows contour plots of the velocity magnitude and the turbulent kinetic energy. In all cases, it is observed that there are two distinct regions of increased turbulence activity and these are associated with the two shear layers.<sup>8</sup> Since the turbulent fluctuations are associated with the breakup of the vortices V1, earlier vortex roll-up is accompanied with an upstream advancement of the regions of high turbulence activity.

## **CONCLUSIONS**

The present study is motivated by the quest to understand the dynamics of flows downstream of severe arterial constrictions. In this study, detailed numerical results for time dependent velocity, pressure and wall shear stress distributions have been obtained for 3D axisymmetric stenosis models.

Examination of the vortex dynamics indicates that the dynamics of the flow downstream of the constriction is dominated by two shear layers, one of which separates from the constriction and the other, from the vessel wall.

Examination of the wall pressure fluctuations indicates that the highest intensity occurs at roughly 2–3 channel diameters downstream of the constriction where the separated shear layers impact on the channel walls.

As expected, the mean recirculation zones are associated with high values of the shear stress, which is well known in the haemodynamics field. From a pathological view point, the variation in the shear stress fluctuation level observed here may have several important implications. First, the higher shear stress fluctuation level on the wall indicates that for a symmetric stenosis, greater instance of pathological

behavior in the endothelial cells may occur on the side of the wall on which the stenosis is the thickest.

The computed turbulent kinetic energy indicates that the two shear layers are the primary turbulence-producing mechanisms in the flow downstream of the constriction.

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