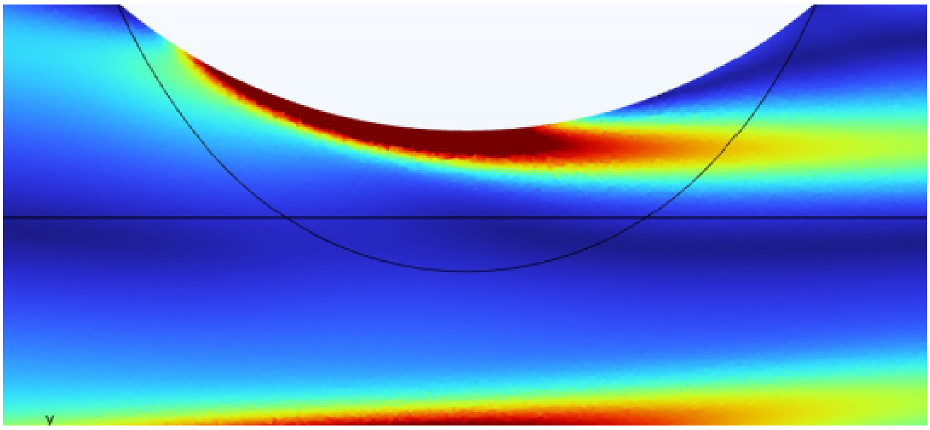
Computational Analysis of Blood Flow Through Asymmetric Stenosis in Arteries

Report Prepared

by

BioCompScience



BioCompScience: Biological Computation Science
Founded 2020
`www.biocompscience.com,www.biocompscience.in, bcseducationtech@gmail.com`

Abstract

The analysis of blood flow is essential for the design and development of medical devices, diagnostic instruments, and therapeutic strategies for vascular diseases such as atherosclerosis, aneurysm, and thrombosis. In this study, blood flow through an asymmetric stenosis in a vessel has been investigated. To achieve this objective, a mathematical model was developed in which the continuity and momentum equations were solved for a non-Newtonian fluid.

The results indicate that velocity increases significantly within the stenosed region, leading to elevated pressure gradients. Additionally, a pronounced variation in velocity along the radial direction was observed, resulting in high shear rates at the surface of the blockage. These shear rates near the blockage are considerably higher than those along the non-obstructed vessel wall.

Contributors

Table 1: List of Contributors for the Project

S.No.	**Name**	**Designation**	**Responsibility**
1	Avinash Kumar	CFD Engineer	Solver Design, Simulation, Pre-processing, post processing
2	Amit Kumar	Thermal Engineer	Simulation
3	Vikash Kumar	Thermal Engineer	Provides strategic guidance and decision-making support
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			

Copyright

Acknowledgments

BioCompScience and Technology sincerely acknowledges and appreciates the efforts of all employees involved in the successful execution and completion of this project. Their dedication, technical expertise, and continuous commitment played a vital role in achieving the desired outcomes of this work.

The company also extends its sincere gratitude to the client for placing their trust and confidence in BioCompScience and Technology, and for providing the opportunity to undertake and successfully deliver this project. Their valuable support and cooperation were instrumental throughout the course of this work.

We also express our appreciation to all individuals and stakeholders who contributed directly or indirectly to the successful completion of this project.

Contents

List of Figures

List of Tables

Chapter 1

Problem Definition and Objectives

1.1 Introduction

The analysis of blood flow is essential for the design and development of medical devices, diagnostic instruments, and therapeutic strategies for vascular diseases such as atherosclerosis, aneurysm, and thrombosis. In this study, blood flow through an asymmetric stenosis in a vessel has been investigated.

Blockage, or plaque formation in arteries, derives from the Greek words atheroma (meaning paste or plaque) and sclerosis (meaning hardening). It has traditionally been associated with aging, typically affecting men over 45 years and women over 55 years. However, with changing lifestyles, atherosclerosis is increasingly observed in individuals as young as their teens and twenties, with its effects often becoming noticeable by the thirties.

Cardiovascular diseases remain one of the leading causes of mortality worldwide, with atherosclerosis playing a central role in many life-threatening conditions such as heart attack and stroke. The development of plaque within arterial walls leads to narrowing of the vessel, known as stenosis, which significantly alters normal blood flow patterns. These alterations can result in elevated pressure gradients, disturbed flow behavior, and increased shear stress, all of which contribute to further progression of the disease and potential rupture of the plaque.

1.2 Objective

The primary objective of this work is to develop a mathematical model and perform a computational fluid flow analysis of blood flow within a stenosed vessel. The study investigates the following parameters to characterize the effects of flow around the blockage:

1. Analysis of blood flow velocity within the vessel.
2. Pressure distribution inside the blood vessel.
3. Evaluation of shear rate along the vessel wall.
4. Other relevant parameters influencing the flow behavior and design considerations.

Chapter 2

Physical Model and Material Properties

2.1 Physical Model

Figure 2.1 illustrates the physical domain, which consists of a blood vessel with a stenosis (blockage). The blood vessel has a diameter of 4 mm and a length of 120 mm. The stenosis has a depth of 1.2 mm and develops from one side of the vessel wall, making it an asymmetric stenosis.

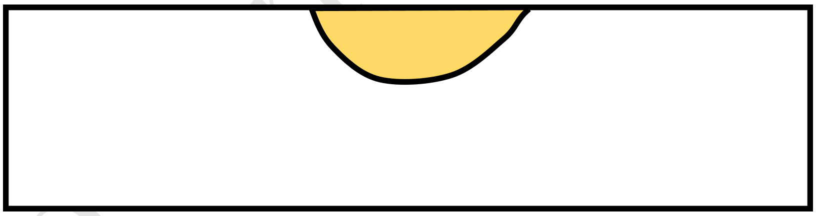


Figure 2.1: Physical domain of energy storage device

2.2 Material Properties

The flowing parameter has been used in this cumputation analysis of blood inside blood vessels.

1. Material: Density of blood 1050 kg/m^3.
2. Kinematic viscosity 3.35 $\times 10^{-6} m^2/s$.
3. Kinematic viscosity 3.35 $\times 10^{-6} m^2/s$.

4. Carreau Model to captured non-newtonian behavior.

Chapter 3

Numerical Model Setup

3.1 Computational Domain

Figure 3.1 illustrates the computational domain, consisting of a blood vessel with a stenosis (blockage). The vessel has a diameter of 4 mm and a length of 120 mm. The stenosis has a depth of 1.2 mm and develops from one side of the vessel wall, making it asymmetric. Figure 3.1 shows the inlet and outlet directions, the location of the blockage, and other relevant parameters.

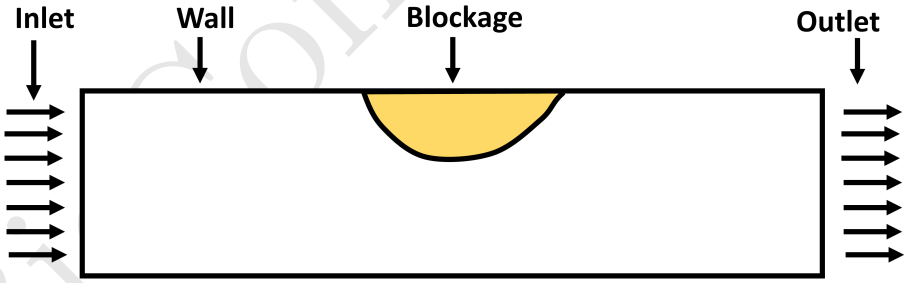


Figure 3.1: Computational domain.

3.2 Mesh Generation

In Figure 3.2, the computational mesh for the domain is illustrated. A highly refined mesh is applied in the atherosclerotic region to capture the velocity and pressure gradients accurately. Additionally, a fine mesh is generated within the boundary layer along the length of the blood vessels.

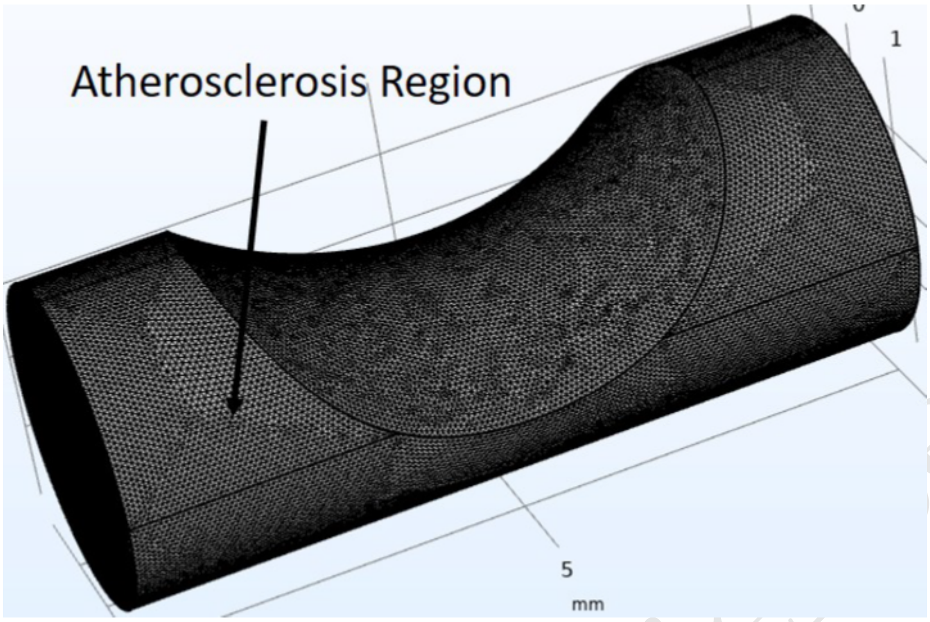


Figure 3.2: Mesh generated for computation.

3.3 Initial Conditions

The initial conditions used in this numerical simulation are listed below:

1. The initial velocity within the domain is 0 m/s.
2. The initial pressure within the domain is 0 Pa.

3.4 Boundary Conditions

1. The inlet velocity is set to 6 cm/s.
2. The velocity gradient at the outlet is zero.
3. The velocity at the vessel wall is zero (no-slip condition).
4. The pressure gradient at the outlet is 0 Pa.
5. The pressure at the outlet is fixed at 0 Pa.

Chapter 4

Mathematical Formulation and Numerical Methodology

4.1 Governing Equation

The governing equations for blood flow consist of the continuity equation and the momentum equation, which together ensure conservation of mass and momentum within the fluid domain. For incompressible flow, the continuity equation enforces that the velocity field remains divergence-free, while the momentum equation describes the balance of inertial, pressure, viscous, and body forces acting on the blood. In addition, the non-Newtonian behavior of blood is incorporated using the Carreau model, which accounts for the shear-thinning nature of blood by allowing the dynamic viscosity to vary with shear rate. This model provides a more realistic representation of hemodynamic behavior, particularly in regions of stenosis where large variations in shear rate occur.

4.1.1 Continuity Equation

$$\nabla \cdot \mathbf{u} = 0 \tag{4.1}$$

The terms in the continuity equation are defined as follows:

1. ∇ is the vector differential operator (nabla), representing spatial derivatives.
2. $\mathbf{u}$ is the velocity vector of the fluid, having components in the coordinate directions.
3. $\nabla \cdot \mathbf{u}$ represents the divergence of the velocity field.

4. The equation equal to zero indicates that the fluid is incompressible, meaning there is no net accumulation or depletion of mass within the control volume.

4.1.2 Momentum Equation

In the context of blood flow, this equation is particularly important because it captures the complex hemodynamic behavior within arteries, especially in regions affected by stenosis. The inertial terms represent the acceleration of blood due to temporal changes and spatial variations in velocity. The pressure gradient term drives the flow through the vascular system, while the viscous stress term accounts for internal resistance due to blood's non-Newtonian nature.

In stenosed arteries, these effects become more pronounced due to abrupt changes in geometry, leading to high velocity gradients, elevated pressure drops, and increased shear stresses along the vessel wall. The momentum equation, therefore, plays a crucial role in predicting flow disturbances and understanding the mechanical environment that contributes to disease progression such as atherosclerosis and thrombosis.

$$\rho\left(\frac{\partial \mathbf{u}}{\partial t} + \mathbf{u}\cdot\nabla\mathbf{u}\right) = -\nabla p + \nabla\cdot\boldsymbol{\tau} + \rho\mathbf{g} \tag{4.2}$$

The terms in the momentum equation are defined as follows:

1. ρ is the density of blood (fluid density).

2. $\mathbf{u}$ is the velocity vector of the fluid.

3. $\frac{\partial \mathbf{u}}{\partial t}$ represents the local (unsteady) acceleration of the fluid.

4. $\mathbf{u}\cdot\nabla\mathbf{u}$ represents the convective acceleration due to fluid motion.

5. p is the static pressure field within the blood vessel.

6. ∇p is the pressure gradient force.

7. $\boldsymbol{\tau}$ is the extra stress tensor, which represents viscous effects (important for non-Newtonian blood flow).

8. $\nabla\cdot\boldsymbol{\tau}$ represents the divergence of the stress tensor (viscous force contribution).

9. $\rho\mathbf{g}$ represents the body force due to gravity acting on the fluid.

4.1.3 Non-newtonian Carreau Model

The Carreau model is widely used to describe the non-Newtonian behavior of blood, particularly its shear-thinning characteristics. In this model, the dynamic viscosity varies with the shear rate, allowing a more realistic representation of blood flow compared to the Newtonian assumption.

At very low shear rates, the viscosity approaches the zero-shear viscosity μ_0, which reflects the higher resistance to flow due to the aggregation of red blood cells. As the shear rate increases, these cell aggregates begin to break down and align with the flow, causing the viscosity to decrease. At sufficiently high shear rates, the viscosity approaches a constant value known as the infinite-shear viscosity μ_∞.

The parameter λ represents a time constant that characterizes the transition between low and high shear rate regions, while n is the power-law index that determines the degree of shear-thinning behavior. This model is particularly suitable for simulating blood flow in arteries with stenosis, where significant variations in shear rate occur, especially near the vessel walls and within the narrowed region.

$$\mu = \mu_\infty + (\mu_0 - \mu_\infty)\left[1 + (\lambda\dot{\gamma})^2\right]^{\frac{n-1}{2}} \tag{4.3}$$

Chapter 5

Results and Discussion

5.1 Pressure Characteristics

In Figure 5.1, the contour plot illustrates the variation of pressure. It can be observed that the pressure magnitude is higher in the direction opposite to the flow, resulting in a favorable pressure gradient.

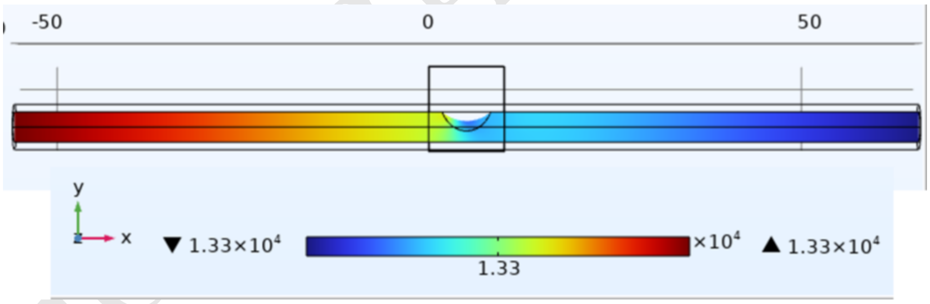


Figure 5.1: Contour plot of pressure variation at time t= 4 s.

In Figure 5.2, the variation of pressure along the longitudinal direction is presented. It can be clearly observed that the pressure gradient within the stenosis region is significantly high, indicating a rapid change in pressure over a short distance. Additionally, it is noticeable that the pressure near the flow exit from the atherosclerotic region initially decreases and then gradually recovers downstream. This behavior reflects the complex flow dynamics associated with the constricted geometry. The maximum pressure recorded in this simulation is 13346 Pa, while the minimum pressure is 13332 Pa.

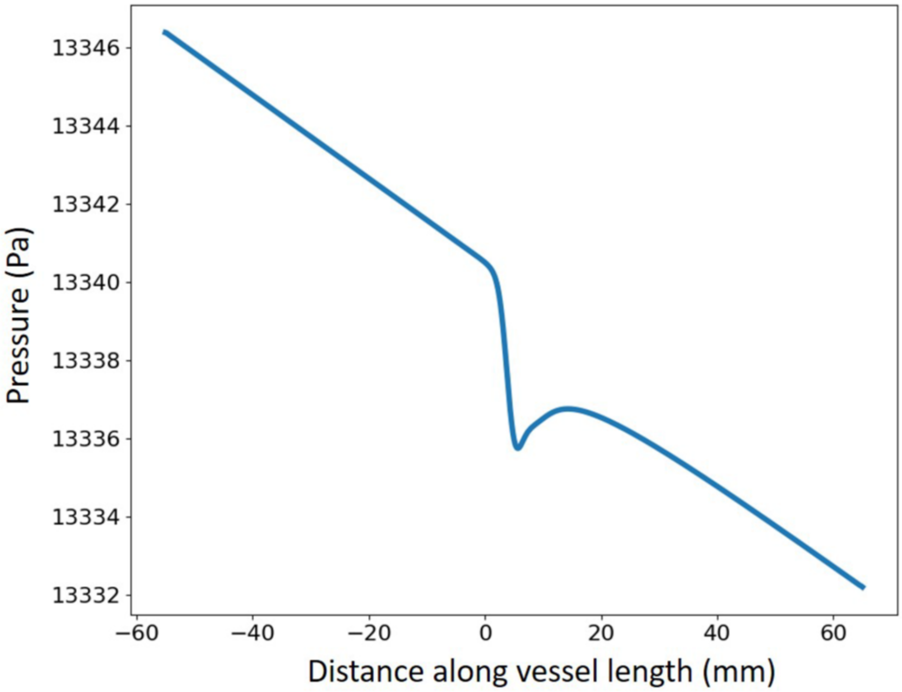


Figure 5.2: Variation of pressure along longitudinal of blood vessels at time t= 4 s.

5.2 Flow Characteristics

In Figure 5.3, the contour plot of velocity is presented to illustrate the flow behavior within the vessel. It can be observed that the velocity magnitude is significantly higher in the core region of the stenosis compared to the surrounding regions. This increase in velocity is consistent with the presence of a strong pressure gradient in the same region, as previously shown in Figure 5.2.

Furthermore, it is noticeable that recirculation zones develop near the vessel walls in the downstream direction of the stenosed region, indicating complex flow patterns. The contour also reveals that the spatial extent of the low-velocity region is relatively smaller within the stenosis compared to the non-stenosed sections of the vessel. This behavior highlights the constricted geometry's effect in accelerating the flow and compressing the low-velocity regions.

In Figure 5.4, the variation of velocity along the radial direction of the blood vessel is plotted at different axial locations. The blue dotted line (x=1x $=$ 1x=1) represents the velocity distribution in the upstream region of the blood flow. The orange solid line (x=5x $=$ 5x=5) corresponds to the velocity variation within the core region of the stenosis, while the green dotted line (x=9x $=$ 9x=9) shows the velocity profile at the downstream location, near the exit of the flow from the stenosed region. It can be observed that the velocity profile in the upstream region exhibits

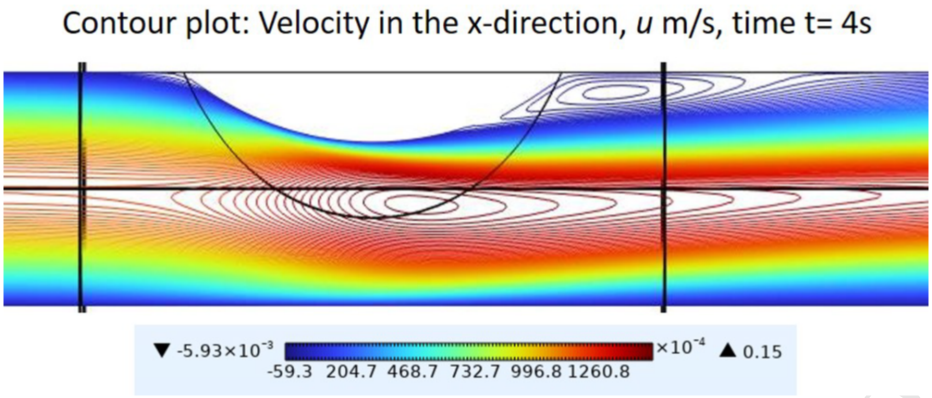


Figure 5.3: Contour plot of velocity variation at time t= 4 s.

a typical parabolic shape, characteristic of fully developed laminar flow. However, within the stenosis, the profile deviates significantly from the parabolic form due to the constricted geometry and increased flow acceleration. At the downstream location, near the exit of the stenosis, the velocity near the vessel wall becomes negative, indicating a local reversal of flow direction. This reversed flow gradually approaches zero at the vessel wall, which suggests the presence of recirculation zones or flow separation near the stenosis wall.

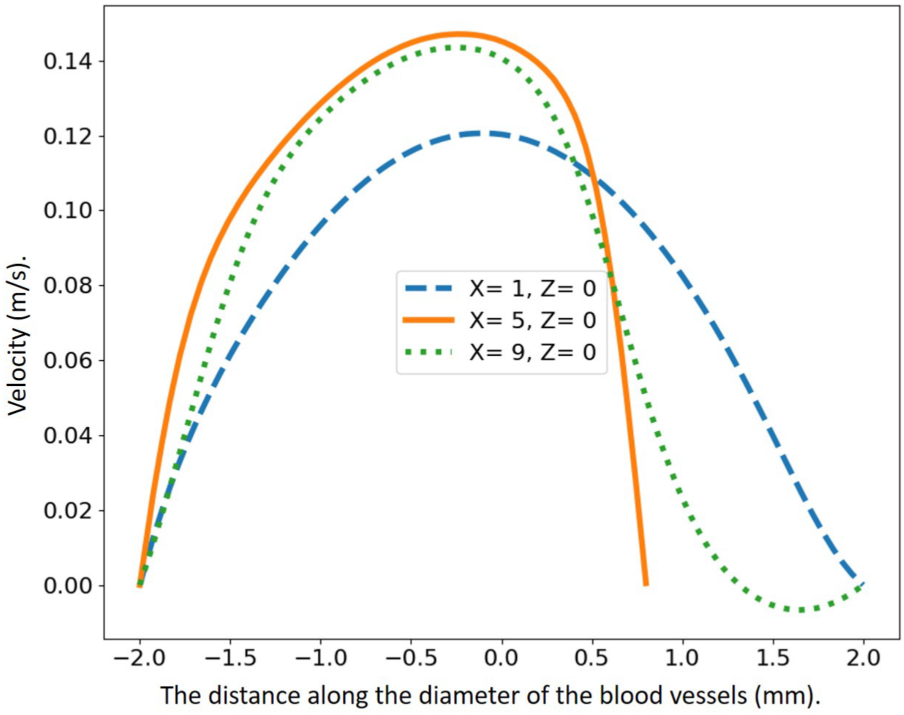


Figure 5.4: Variation of velocity along radial direction at different location at time t= 4 s.

5.3 Shear Rate Characteristics

In Figure 5.5, the shear rate along the vessel wall is presented. It is evident that the shear rate in the stenosis region is higher than in the non-stenotic region. Elevated shear rates may contribute to rupture of the stenosis. This observation is consistent with the increased velocity gradient of blood flow near the vessel wall.

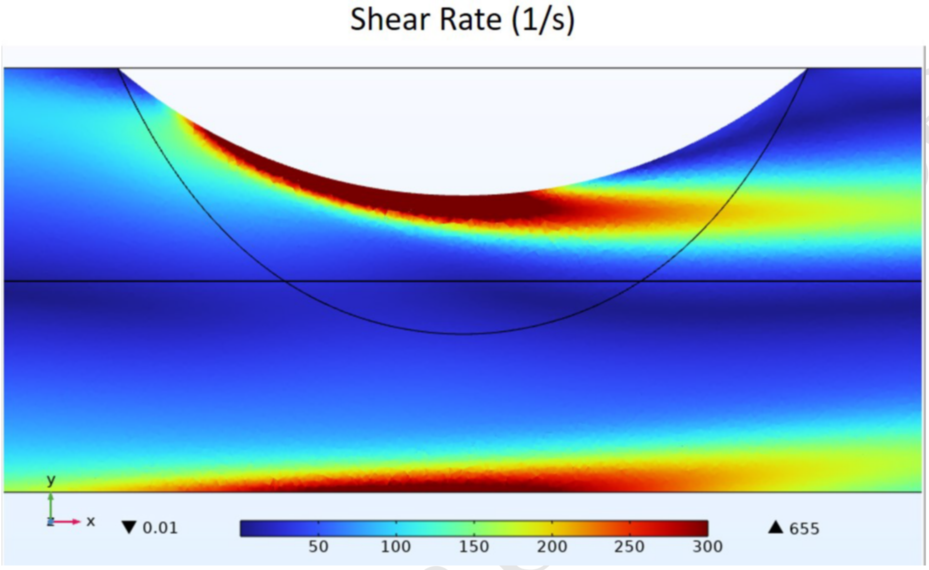


Figure 5.5: Contour plot of shear rate at time t= 4 s.

Figure 5.6 illustrates the variation of shear rate along the stenosis surface and the vessel wall. The solid blue line represents the shear rate on the stenosis surface, while the dotted orange line corresponds to the shear rate on the non-stenotic surface within the same region. It is observed that the maximum shear rate on the stenosis surface reaches 635 1/s , whereas it is 335 1/s on the non-stenotic surface. Such elevated shear rates may contribute to rupture of the stenosis, which can obstruct blood flow and lead to serious conditions such as heart attack.

From a physiological perspective, high shear rates are closely associated with plaque instability. When the shear stress exceeds a critical threshold, it may weaken the fibrous cap of the atherosclerotic plaque, increasing the likelihood of rupture. Plaque rupture can lead to the release of debris into the bloodstream, potentially causing partial or complete blockage of the vessel. This can disrupt normal blood circulation and may result in severe cardiovascular events such as myocardial infarction or stroke.

Furthermore, the contrast in shear rate between stenotic and non-stenotic regions highlights the localized nature of hemodynamic disturbances. These variations play a crucial role in disease progression, as regions exposed to abnormal shear conditions are more susceptible to further plaque development and vascular damage. Therefore, analyzing shear rate patterns is essential for understanding the risks associated with stenosis and for improving diagnostic and therapeutic strategies.

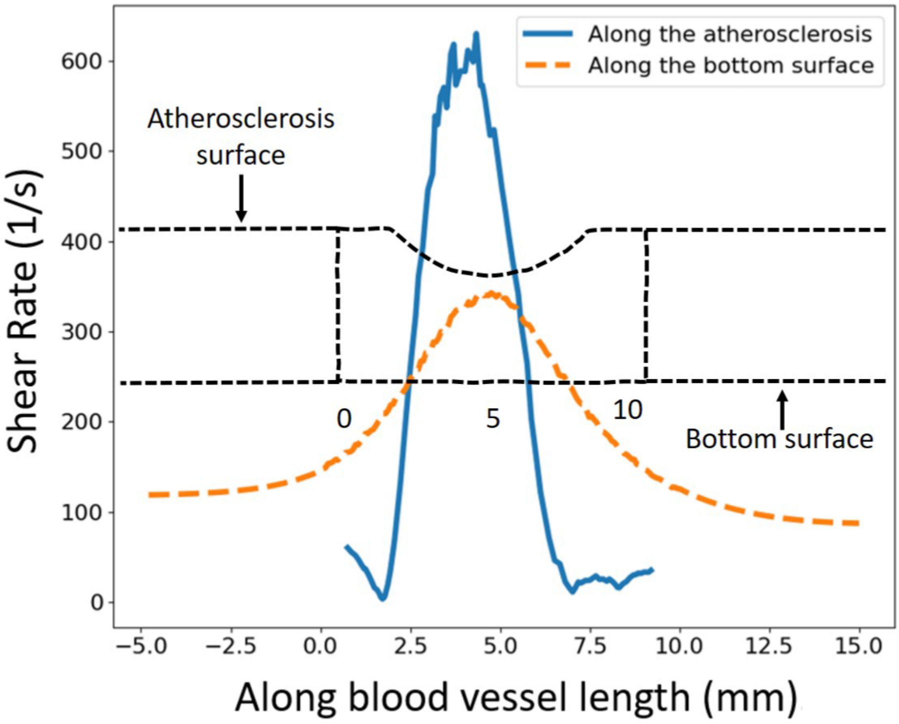


Figure 5.6: Variation of shear rate on the surface of blood vessels at time t= 4 s.

Chapter 6

Conclusions and Future Work

6.1 Conclusion

The present study provides a detailed computational analysis of blood flow through an asymmetric stenosed artery using a non-Newtonian fluid model. The numerical results clearly demonstrate that the presence of stenosis significantly alters the hemodynamic characteristics within the blood vessel.

It is observed that the velocity of blood increases substantially within the narrowed region due to the reduction in cross-sectional area. This acceleration is accompanied by a sharp pressure drop across the stenosis, indicating a strong pressure gradient in the flow direction. Downstream of the stenosis, flow separation and recirculation zones are formed near the vessel wall, reflecting complex and disturbed flow behavior.

The analysis of shear rate reveals that significantly higher shear rates occur at the surface of the stenosis compared to the non-stenotic regions. The maximum shear rate on the stenosed surface is nearly twice that of the normal vessel wall. Such elevated shear conditions are critical, as they can contribute to plaque instability and increase the risk of rupture. Plaque rupture may lead to partial or complete blockage of the artery, potentially resulting in severe cardiovascular events such as heart attack or stroke.

Overall, the study highlights the strong influence of arterial geometry on flow behavior, pressure distribution, and wall shear characteristics. The findings emphasize the importance of hemodynamic analysis in understanding the progression of atherosclerosis and its associated risks. This work can be useful in the design of medical diagnostic tools, treatment planning, and the development of safer and more effective cardiovascular interventions.

6.2 Future work

The present study can be extended in several directions to achieve a more comprehensive understanding of blood flow behavior in stenosed arteries. One important improvement would be the incorporation of pulsatile (time-dependent) flow conditions to better represent realistic physiological blood flow, as actual cardiovascular flow is inherently unsteady.

Disclaimer

The information presented in this report is for research and informational purposes only. BioCompScience and Technology makes no guarantees regarding the accuracy or completeness of the results. The company shall not be held liable for any consequences arising from the use of this information.