

Effect of Stenosis and Aneurysm on Atherosclerotic Hemodynamics during a Blood Pulsatile Flow

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Abstract

The danger of rupture for severe cases of stenosis and aneurysms is extensively studied by healthcare professionals and researchers. Numerical researchers have likewise played a role in forecasting this rupture. This study offers a numerical computation of a time-dependent three-dimensional arterial flow of Newtonian fluid, examining the influence of stenosis and aneurysm on atherosclerosis-related hemodynamics. The pulsatile blood flow simulation has been executed with the software code of COMSOL Multiphysics using a finite element approach. Results indicate that the stenotic model creates hemodynamic conditions associated with a higher risk of thrombosis compared to the aneurysm model. A higher viscous stress is found in the stenotic model compared to that of the aneurysm model. Higher magnitudes of velocity and pressure are estimated for the stenotic model. It can be concluded that for the same height of stenosis and aneurysm, the stenosis model poses a severe risk to humans.

Keywords

Stenosis, Aneurysm, Hemodynamics, Atherosclerosis, Pulsatile Flow

1. Introduction

A localized, seditious fibroproliferative reaction to various types of endothelium damage is known as atherosclerotic arterial disorder [1]. During atherosclerotic stage, the buildup and deposition of lipid substances and cholesterol, along with the formation of combinative tissues, result in a fragmental reduction of the artery cross-sectional locality, referred to as stenosis. Conversely, an aneurysm is defined

as a localized expansion of an artery resulting from an acquired or inherited weakness of the wall of vessel. Around the world, cardiovascular illnesses are a major source of morbidity and death. One of the main causes of these disorders is artery stenosis, a disorder in which plaque builds up in the arteries, narrowing them and decreasing blood circulation [2]-[6]. Research on blood circulation within narrowed arteries has garnered considerable attention due to its direct implications for cardiovascular well-being. Numerous factors, including the stenosis's shape, the blood's characteristics, and the existence of disease, affect the extremely complex blood circulation in the aforementioned vessels [7]-[11].

Several studies and computational fluid dynamics (CFD) assessments have been carried out to investigate the flow disruption brought on by human aneurysm formation, leading to cardiovascular disorders [12]-[16]. Narayan *et al.* [17] have analyzed the blood flow in an aneurysm within a magnetic field to evaluate the hemodynamic risk factors. The study has shown that the globular and bilobed forms are more likely to experience sac rupture and lateral neck expansion. Blood pulsation flow has been numerically simulated by Elgar *et al.* [18], who have identified several flow types and examined how they affect hemodynamics. Paramasivam *et al.* [19] have created a program to aid in the diagnosis and management of aneurysms. The current study presents a comparison of stenosis and aneurysm in hemodynamic risk parameters, which is the research gap [20].

This paper aims to examine the hemodynamic conditions within a diseased artery, focusing on a 45% depth related to stenosis and aneurysm models for the analysis. Although a significant amount of research work has been conducted to examine the changes in various parameters in the circulation of blood in various modeled stenoses and aneurysms, no research has been conducted to examine the flow characteristics in terms of hemodynamic risk factors of aneurysms with stenoses of the identical size. The present research offers this numerical investigation.

2. Numerical Methodology

2.1. Model of Simulation

Blood is modeled as a Newtonian fluid because it is a reasonable approximation for flow in large arteries. The two geometric models of stenosis (**Figure 1(a)**) and aneurysm (**Figure 1(b)**) have been taken to examine the mimicked blood flow hemodynamics. The computational domain has features that the lengths of pre-stenosis and post-stenosis areas are 15 mm and 50 mm, respectively. The coordinates X and Y present the direction of horizontal and vertical. The arterial geometry has a radius of 2.5 mm.

2.2. Governing Equations and Boundary Conditions

The equations that govern the continuity and Navier-Stokes are as follows:

The fluid flow's continuity equation is

$$\nabla \cdot \mathbf{u} = 0 \quad (1)$$

The Navier-Stokes equation is

$$\rho \left(\frac{\partial \mathbf{u}}{\partial t} + (\nabla \cdot \mathbf{u}) \mathbf{u} \right) = -\nabla p + \nabla \cdot \boldsymbol{\tau} \quad (2)$$

where \mathbf{u} is the velocity vector, t is the time, ρ indicates the density of the fluid, and p is the fluid pressure. The viscous stress tensor $\boldsymbol{\tau}$ is identified in the following way:

$$\mu (\nabla \mathbf{u} + (\nabla \mathbf{u})^T)$$

where the parameter μ presents the dynamic viscosity of the fluid.

The computational fluid domain's inlet takes the following time-dependent velocity pulse [21], as follows:

$$U_{inlet} = 50955.4 * (0.0000043 + 0.0000026 * \sin(2\pi t/T)).$$

No-slip boundary condition is considered across the wall, and the outlet pressure is static.

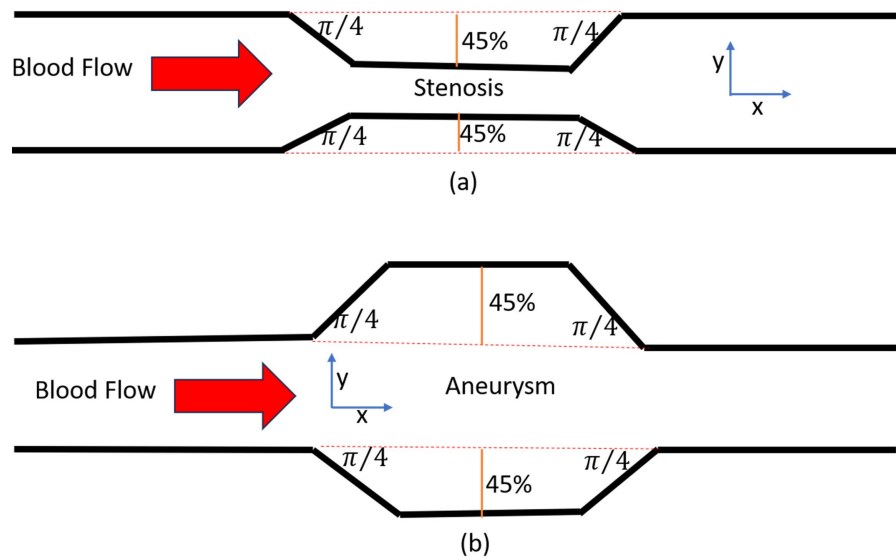


Figure 1. The current simulation model of (a) stenosis and (b) aneurysm.

2.3. Numerical Solution Approach

Computational fluid dynamics is a significant scheme to evaluate the potential outcomes in blood flow investigation. The areas before and after the stenosis are selected to reduce the impact of the boundary conditions of inflow and outflow, and adequately represent the flow characteristics at the downstream region. The finite element scheme-based software code for COMSOL has been employed to capture the simulation outcomes. The selected grid resolution is displayed in **Figure 2(a)**. **Figure 2(b)** exhibits the grid test for various mesh densities. The mesh elements of 204605 are chosen because the simulation outcomes of time-averaged wall pressure are approximately identical for mesh sizes of 204,584 and 306,554. As the flow is pulsatile, the second cycle has been adopted.

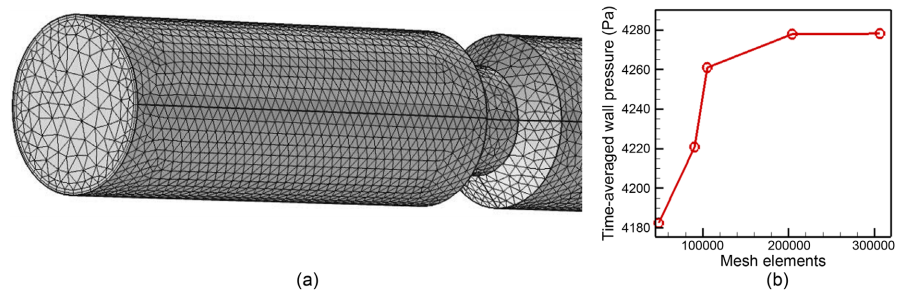


Figure 2. (a) Grid resolution and (b) grid independence test in time-averaged wall pressure.

2.4. Validation

This research validates and confirms the correctness and uniformity of the numerical computational estimation by comparing the results with those of the investigation by Lee *et al.* [22] using axial velocity along the radial coordinate. Furthermore, there is a significant amount of agreement between the present velocity profile and those obtained by prediction Lee *et al.* [22], as displayed in **Figure 3**. Lee *et al.* [22] validate their work with experimental data, so this validation can be claimed as an experimental validation. The axial velocity predicted in a rigid wall at a downstream area ($8.6 \times$ radius) for the time point of 0.08625 sec shows good agreement.

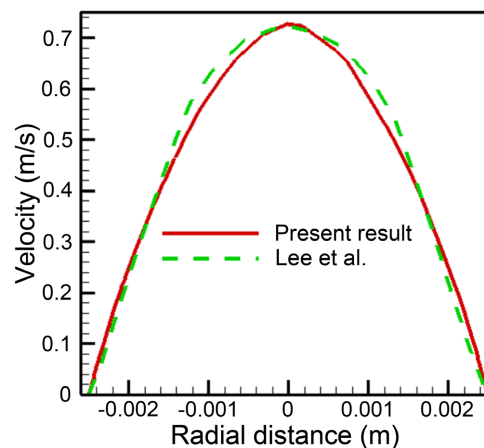


Figure 3. Axial velocity profile is validated with Lee *et al.* [22].

3. Results and Discussion

3.1. Velocity Distribution Due to Stenosis and Aneurysm

In the present research, the pulsating blood flow through stenotic and aneurysm shapes has been modeled by the continuity and Navier-Stokes equations to investigate the influence of variational shapes. **Figure 4** displays the flow disruptions and velocity distributions generated in various sections of the stenotic and aneurysmal shapes with a depth of 45%. The speed is consistently reduced in the area near the wall for both shapes, with this decrease in speed being more significant in stenosis and experiencing a higher velocity than in aneurysm.

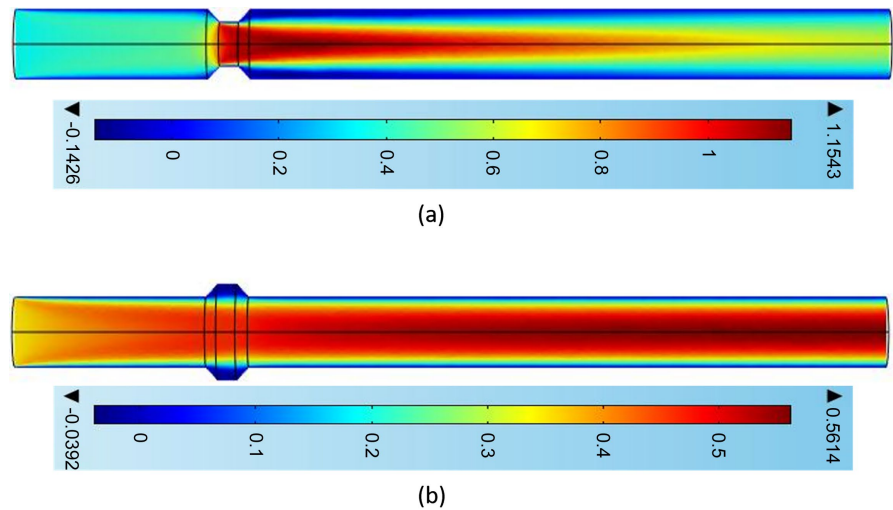
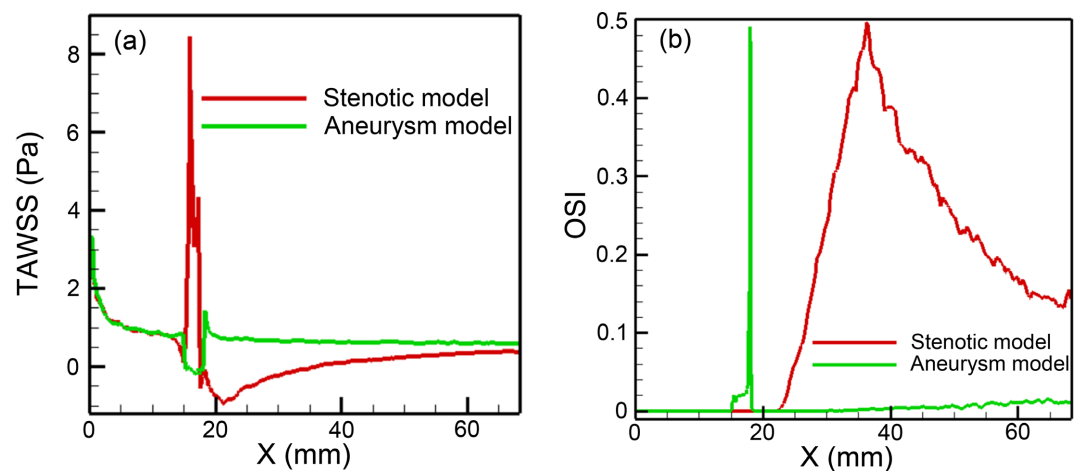


Figure 4. Velocity distribution for (a) stenosis and (b) aneurysm during systolic phase.

3.2. Hemodynamics Bio-Indices' Impact

The time-averaged bio-marker factors, such as time-averaged wall shearing stress (TAWSS), oscillatory shearing index (OSI), and relative residence time (RRT), are displayed axially in **Figures 5(a)-(c)**. It has been noted that, in the case of stenosis, the TAWSS reaches its peak at the stenosis's inlet region since the flow impacts directly on this side, leading to a sudden rise in TAWSS, moderately elevated across the stenosis region, and ultimately declines from the ending sections. However, aneurysm's TAWSS characteristics differ from those observed in stenosis. In this case, the highest TAWSS occurs initially, and after that, it occurs at its highest at the aneurysm's outlet region because the inlet side's flow expands and impacts the ending side, resulting in elevated TAWSS. The highest peaks of OSI and RRT indicate the reattachment points, which correspond to zero for TAWSS. **Figure 5(d)** presents the WSS contours due to the shapes of stenosis and aneurysm, indicating the highest shear stress at the stenosis location, whereas it reflects the opposite magnitudes of wall shear stress due to the shape of the aneurysm.



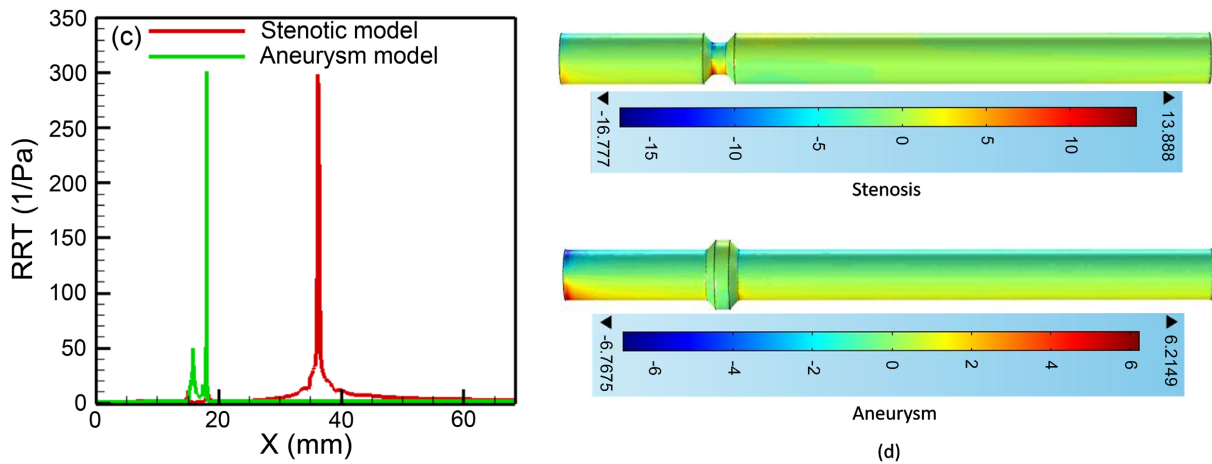


Figure 5. Distributions of (a) TAWSS, (b) OSI, (c) RRT, and (d) contours of WSS, for the models of stenosis and aneurysm.

3.3. Pressure Distribution Contour

The pressure caused by stenosis and aneurysm is a crucial factor in comprehending the critical condition if it is not identified and assessed correctly. In **Figure 6**, pressure contours for stenosis show that the pressure peaks in the areas before the blockage and decreases at the narrowest point of the stenosis model, leading to minimum pressure at the throat of the blockage. Additionally, the pressure rises within the aneurysm but decreases in the remote downstream areas. The pressure contours in **Figure 6** conclusively present that the maximum pressure is attained at its stenosis location for the stenosis model, whereas for the aneurysm model, the lowest pressure is acquired at the post-aneurysmal distal part.

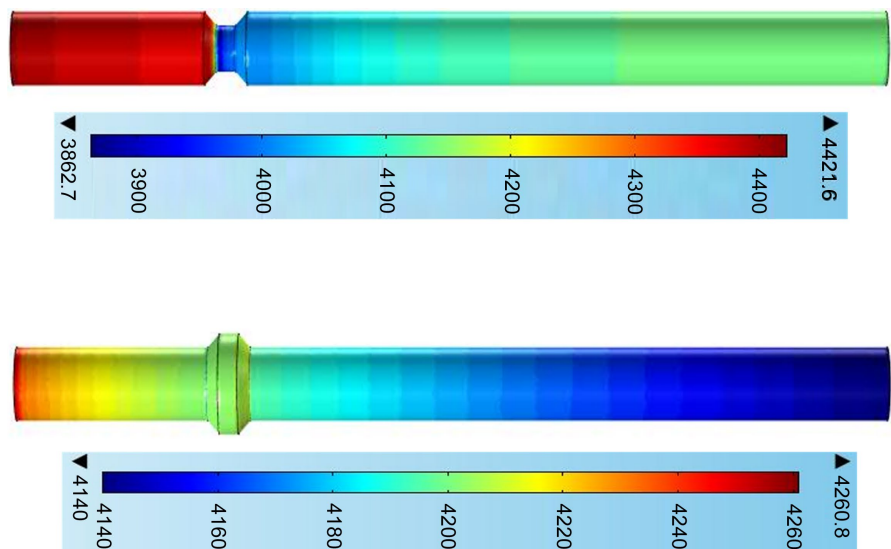


Figure 6. Pressure contours for stenotic model (upper) and aneurysm model (bottom).

4. Conclusions

The current work examines the impact of the laminar flow of pulsation across the arterial stenotic and aneurysmal models using a computational approach of COM-

SOL Multiphysics in three dimensions. The stenosis of the trapezium with the angulation of 45° and depth of 45% has been employed to observe the influence on both models. Time-averaged parameters, including TAWSS, OSI, and RRT, are the leading potential risk factors utilized to capture the atherosclerotic recirculation area. The findings show that, in contrast to the aneurysm model, the stenotic model provides the possible danger of a thrombotic area. The stenotic model exhibits a greater viscous stress than the aneurysm model. Greater values of velocity and pressure are predicted for the stenotic model. It can be inferred that for identical heights of stenosis and aneurysm, the stenosis model presents a significant threat to humans. In conclusion, it can be said that the research outcomes may be used in biomedical applications.

Future extensions of this research may focus on patient-specific non-Newtonian models in fluid-structure interaction methods. The results may be applied in bioengineering areas.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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