Time-Dependent Study of Blood Flow Through Vertebral Artery Analysing the Effect of Stenosis

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Publication Date: 2025/07/26

Abstract: Vertebral artery stenosis (VAS) is a major contributor to ischemic strokes in the posterior circulation. Computational fluid dynamics (CFD) has emerged as a valuable tool in simulating blood flow dynamics in both healthy and pathologically altered vessels. This study explores the temporal characteristics of blood flow in the vertebral arteries using numerical simulations. We model blood flow under pulsatile conditions in anatomically realistic healthy and stenosed vertebral arteries and evaluate velocity distributions and pressure variations, over a cardiac cycle. The study aims to enhance understanding of hemodynamic alterations caused by stenosis and their implications in vertebrobasilar insufficiency and stroke risk.

Keywords: CFD; Hemodynamics; Vertebral Artery Stenosis; Pressure.

How to Cite: Josephina Harris; Ajit Paul; Bhavna Singh Ghosh (2025) Time-Dependent Study of Blood Flow Through Vertebral Artery Analysing the Effect of Stenosis. *International Journal of Innovative Science and Research Technology*, 10(7), 2050-2055. https://doi.org/10.38124/ijisrt/25jul1204

I. INTRODUCTION

The vertebral arteries are essential components of the cerebrovascular system, supplying oxygenated blood to the posterior part of the brain, including critical regions such as the brainstem, cerebellum, and occipital lobes. Stenosis, defined as a localized narrowing of the arterial lumen, can significantly disrupt normal blood flow, leading to altered hemodynamic conditions that may predispose individuals to ischemic events, transient ischemic attacks, or stroke. Early detection and precise assessment of stenosis are therefore vital for preventing severe neurological deficits and for planning appropriate medical or surgical interventions.

While clinical imaging modalities like Doppler ultrasound, CT angiography, and MR angiography provide valuable anatomical and flow information, they often lack the resolution and detailed insights necessary to fully understand the complex flow patterns associated with arterial stenosis. In this context, numerical modeling and computational fluid dynamics (CFD) have emerged as powerful tools for investigating the hemodynamic consequences of vascular diseases in a controlled and reproducible manner.

Many studies have been carried out to understand the flow dynamics and other hemodynamic parameters, particularly in the case of stenosis[1] and aneurysms [2]. A hemodynamic index was introduced based on blood flow velocity in stenosed and collateral arteries. A case involving multiple stenoses in the vertebral and carotid arteries was examined. The impact of stenosis locations on cerebral blood flow was analyzed for two configurations of the Circle of Willis [3]. A mathematical model for blood flow through stenosed artery was presented. Using a perturbation technique the blood flow was investigated. The importance of roles of many hemodynamic parameters was studied in the development of several diseases related to blood flow [4]. MRI-based CFD was used to evaluate vertebrobasilar hemodynamics in healthy subjects, highlighting geometric influences on flow mixing and shear stress distribution [5]. The blood flow in healthy vertebral arteries were modeled and it was observed that even without pathology, anatomical asymmetries yield varying hemodynamics [6].

The present study focuses on numerically simulating blood flow through the vertebral artery under both healthy and stenosed conditions. Using realistic geometrical models and appropriate rheological properties for blood, we aim to evaluate how stenosis influences velocity of flow, pressure distribution and regions of disturbed or recirculating flow. Such hemodynamic parameters are crucial because they are

https://doi.org/10.38124/ijisrt/25jul1204

linked to the development of atherosclerotic plaques, vessel wall remodeling, and potential thrombus formation.

By comparing healthy and stenosed arterial conditions, this research seeks to enhance our understanding of the pathophysiological mechanisms underlying vertebral artery stenosis. The insights gained from this numerical study could contribute to improving diagnostic criteria, refining risk assessment and guiding clinical decision-making for patients with vertebral artery disease.

II. MATERIALS AND METHODS

For this study, three-dimensional CAD realistic models of both healthy and stenosed vertebral artery were constructed to facilitate detailed study of effect of stenosis on the flow of blood through the vertebral artery.

➤ Geometry and Mesh

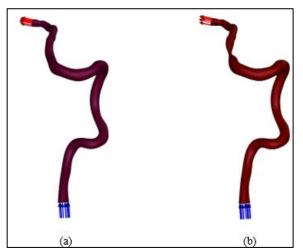


Fig 1: (a) Model A and (b) Model B Indicating the Inlet and Outlet Boundaries.

In this work, a 3D model of a healthy right vertebral artery was constructed from brain MRA images. Model B was constructed with more than 70% stenosis in the V4 segment.

Using ANSYS 2024 the meshing of both the models was done. The surface mesh for model A and model B was generated and five boundary layers were added at the boundary for refining the mesh near the walls, inlet and outlet faces. The volumetric mesh was generated with polyhedral elements with 61087 cells for model A and 62760 cells for model B.

Governing Equations And Boundary Conditions

Blood was modeled as non-Newtonian Casson fluid in both cases and SST $k-\omega$ model was used to consider the turbulence; if any. Blood density was assumed to be 1060 kg/ m^3 .

The equations of conservation of mass (1) and momentum (2) are solved for the flow field along with the two SST $k-\omega$ model given by eq (3) & (4)

$$\nabla \cdot \vec{v} = 0 \tag{1}$$

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

$$\frac{\partial}{\partial t}(\rho k) + \frac{\partial}{\partial x_i}(\rho k u_i) = \frac{\partial}{\partial x_i} \left[\Gamma_k \frac{\partial k}{\partial x_i} \right] + G_k - Y_K + S_k$$
 (2)

(3)

$$\frac{\partial}{\partial t}(\rho\omega) + \frac{\partial}{\partial x_i}(\rho\omega u_j) = \frac{\partial}{\partial x_i}\left[\Gamma_\omega \frac{\partial\omega}{\partial x_j}\right] + G_\omega - Y_\omega + D_\omega + S_\omega$$

(4)

where, \vec{v} is the velocity vector, ρ fluid density, p pressure, τ the stress tensor, Γ the effective diffusivity, G generation term, Y dissipation due to turbulence and D the cross-diffusion term.

At the vertebral artery inlet a pulsatile velocity profile, mathematically as given by Equation (5), is applied by a user-defined function, and at the outlet constant pressure of 0 Pa is applied. The operating condition is set as 13332 Pa [7]. The wall is assumed to be rigid and stationary with no slip condition.

$$v(t) = \begin{cases} 0.5sin[4\pi(t+0.0160236)]\\ if \ 0.5n < x \le 0.5n + 0.218\\ 0.1 \ if \ 0.5n + 0.21 < x \le 0.5(n+1) \end{cases}$$

$$n = 0, 1, 2, 3.....$$
 (5)

➤ Numerical Solution

A comparative computational study was carried out on ANSYS using the finite volume method. **FVM** is a popular numerical technique for solving partial differential equations, especially in fluid dynamics. It works by dividing the computational domain into small control volumes (cells). Conservation laws, like mass, momentum, or energy, are applied to each volume, ensuring fluxes entering and leaving are balanced. This makes FVM inherently conservative and suitable for the geometries used here. It can easily handle complex geometries and unstructured meshes, providing flexibility. The fluid flow equations were resolved by using the segregated solver with the SIMPLE algorithm. The equations were solved sequentially and the solution was obtained iteratively, the process was continued until the desired convergence was achieved. The convergence criteria were set at residuals < 10⁻⁵ for continuity, velocity and turbulence.

III. RESULTS AND DISCUSSION

Temporal study of the blood flow helps in a better understanding of the flow phenomenon due to the pulsatile nature of blood. This study was carried out by taking four time instances as given in Table 1 into consideration for both the geometries.

Table 1: Time Instances Representing the Different Phases of Cardiac Cycle.

Time instance	Phase
T1	Acceleration phase
T2	peak systolic phase
Т3	deceleration phase
T4	minimum diastolic phase

The computational models were developed to represent both a normal vertebral artery and one with a localized stenosis. The simulations successfully captured the essential features of blood flow under physiologically realistic conditions. The pressure distribution on the vessel walls at these four time instances have been presented in Fig. 3. The velocity vectors and velocity streamlines are shown in Fig. 3 and Fig. 4 respectively, for both the model at T1, T2, T3 and T4. In the healthy artery model, flow was observed to be smooth and laminar, with symmetrical velocity profiles and a relatively uniform distribution of pressure along the arterial wall. Velocity streamlines

indicated a streamlined, uninterrupted flow, and the velocity vectors suggested no flow separation.

In contrast, the stenosed artery model revealed significant deviations from the normal flow pattern. The presence of the stenosis resulted in a marked increase in velocity within the narrowed segment, as predicted by the conservation of mass. This was accompanied by a pronounced drop in pressure across the stenotic region, which aligns with Bernoulli's principle. The velocity streamlines in the post-stenotic region exhibited signs of flow disturbance, including recirculation zones and complex secondary flows.

These characteristics are indicative of increased hemodynamic stress and are often associated with endothelial damage, thrombosis, and plaque progression.

The analysis of velocity vectors further highlighted the disturbed flow regime in the stenosed artery. Unlike the healthy model, where vectors remained relatively aligned and parallel to the vessel wall, the stenosed model showed erratic vector orientation in the downstream segment, suggesting high levels of shear stress variability and potential sites of turbulent kinetic energy accumulation. Such regions are clinically significant, as they may correlate with areas prone to further vascular remodeling or aneurysmal dilation.

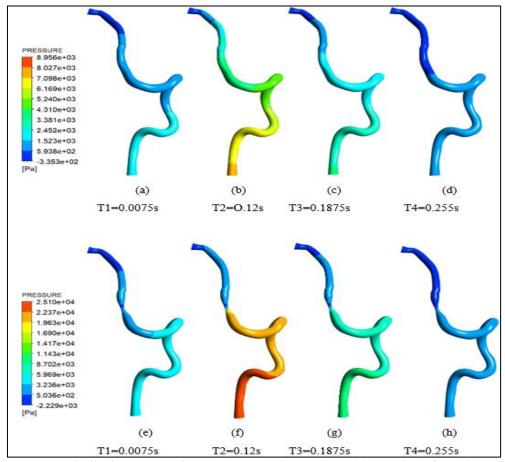


Fig 2: Pressure Distribution at Four Time Instances (a) (b) (c) (d) Model A and (e) (f) (g) (h) Model B

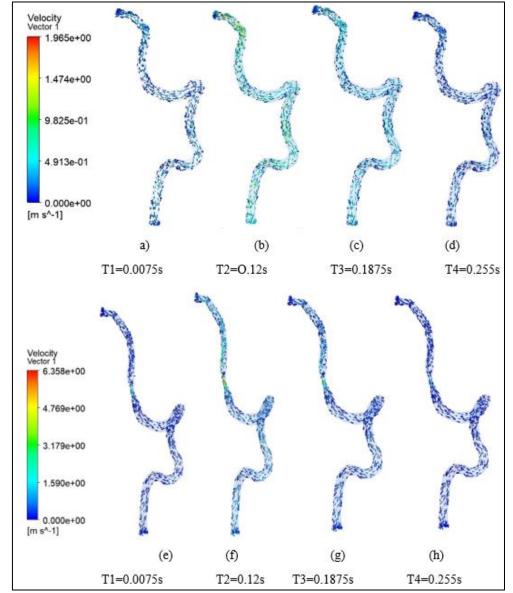


Fig 3: Velocity Vectors at Four Time Instances (a) (b) (c) (d) Model A and (e) (f) (g) (h) Model B

ISSN No:-2456-2165

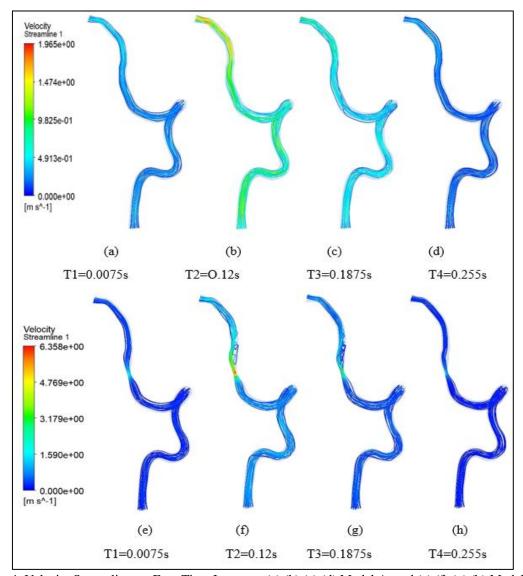


Fig 4: Velocity Streamlines at Four Time Instances (a) (b) (c) (d) Model A and (e) (f) (g) (h) Model B

IV. CONCLUSION

A comprehensive numerical investigation of blood flow dynamics in healthy and stenosed vertebral arteries was carried out using ANSYS. By simulating pulsatile flow conditions and analyzing key hemodynamic parameters such as pressure distribution, velocity streamlines and velocity vectors, this work offers valuable insights into the alterations in flow behavior due to arterial stenosis. Understanding these changes is critical, as abnormal flow patterns are often linked to the progression of vascular diseases and the risk of cerebrovascular events.

Overall, the findings of this numerical study affirm the sensitivity of blood flow patterns to changes in vascular geometry. The use of ANSYS as a simulation platform proved effective in resolving detailed flow structures and quantifying hemodynamic parameters that are difficult to capture through clinical imaging alone. The comparison between healthy and stenosed models underscores the critical role of numerical simulations in assessing vascular

pathology and aiding in the design of medical interventions such as stents or surgical reconstructions.

Future work may involve incorporating patient-specific geometries and inclusion of a larger data set to enhance the physiological relevance of the simulations. Additionally, coupling fluid dynamics with structural analysis could provide insights into vessel wall mechanics under pathological flow conditions. Nonetheless, this study lays a foundational understanding of how stenosis alters vertebral artery hemodynamics, potentially contributing to improved diagnostic and therapeutic strategies in cerebrovascular care.

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