

A Mathematical Modelling for Non-Newtonian Blood Flow through Stenotic Artery using Forward Time Central Space Scheme

Dr. Mahesh Chandra¹ and Santosh Yadav²

¹Assistant Professor, Department of Mathematics, F.S. University, Shikohabad, Uttar Pradesh, INDIA.

²Department of Mathematics, F.S. University, Shikohabad, Uttar Pradesh, INDIA.

¹Corresponding Author: maheshchandra2047@gmail.com



www.jrasb.com || Vol. 5 No. 1 (2026): February Issue

Received: 20-01-2026

Revised: 23-01-2026

Accepted: 27-01-2026

ABSTRACT

Arterial elasticity is a vital factor in regulating blood flow dynamics. Despite its importance, many computational studies simplify this by assuming arteries are rigid, which can lead to errors when predicting wall shear stress and pressure. This research utilizes a two way fluid - structure interaction model, built on Forward Time Central Space Scheme to explore how arterial wall flexibility affects blood flow. In this model, blood vessel is treated as an incompressible non-Newtonian fluid while the arterial wall is treated as a flexible elastic solid. To maintain realistic movement and force at the contact point between blood and the vessel wall, strong coupling conditions were applied under pulsatile flow.

Keywords- shear stresses, elastic solid, pulsatile flow

I. INTRODUCTION

The cardiovascular system is essential for life, responsible for the steady delivery of oxygen, nutrients, and hormones while removing waste products from the body. Blood flow within the arteries is a complex, three-dimensional process that is deeply intertwined with the mechanical response of the vessel walls. To understand how vascular diseases such as atherosclerosis, aneurysms, and hypertension develop and progress, it is necessary to model this interaction accurately. Under normal health conditions, arteries do not act as stiff pipes; rather, they are highly flexible and adaptable structure. Mathematical modelling approaches for biological and initial vironmental processes, citing several research works. Here we have rephrased the Modelling Process. A zero-gradient, zero-velocity, and zero-temperature non-slip wall condition simplifies the modelling process. Along the axis, this condition applies at the inlet Key cited studies. Here we will like to mention some important work already done on related field. Dabi et al. (2013) conducted work in this area. Kozic et al. (2014) used a single-measurable water flow group study. Colin et al.

(2019) studied old work on rust prediction, based on ecological success as a basis. Recent Research conducted by Bret burg et al. (2020) studied ecosystem design. They worked on phenotypic plasticity in juvenile jellyfish interaction. Laguna et al. (2020) identified chaos network in PGL branch. Rossler et al. (2021) studied fractal electronic network by Krasner and Debar. (2022) studied viscous ocean mixing.

II. MATHEMATICAL MODELLING AND GOVERNING EQUATIONS

$$K + m = Q(t) \hat{u} = A_1 u_i + A_2 u_{i+1}$$

Where $A_1 = 1 - s$, $A_2 = s$

and $\Delta = (x - x_i)/l \int_e g_e \in A \, dx = \int_e F S u_4 A \, dx + \int_e f S u_4 A \, dx$ where g_e and u_4 denote the virtual strain and displacement. A is the cross-section area of stenotic artery

III. FORCE EXERTED DURING FLOW

$$F = -\rho_0 \partial^2 u / \partial t^2 \{u\} = [A] q \{u\}, [A] \{u\} \int_e [B]^T [B] A \, dx \\ (q) = \int_e \rho_0 [N]^T [N] A \, dx (\dot{q}) + \dots \sigma \{K\} \{q\} + \{m\} \{q\} =$$



$\{Q(t)\} [K] = (\Delta E / l) [1, -1], [-1, 1] [m] = \rho_0 A l \int_0^1 (1 - s) [[1 - s, s], [s, 1 - s]] - R_i f = \beta_i, f = 1 + \beta_i, 2f = 2 + \beta, 3f = 3 + \beta, 4f = 4 + \epsilon (3.3)$ Where R_i is the return of blood due to steroids if is risk free rate, β_i is the sensitivity constant with factors and ϵ is the error term.

Then a cross sectional regression or second pass regression is used on average excess returns of the blood on the factor scores. $\hat{R} = \gamma (0) + \gamma, \beta_1 + \gamma, \beta_2 + \gamma, \beta_3 + \gamma, \beta_4 + \epsilon_i (3.4)$ Where is average excess return of blood, β_1 to β_4 are the factors scores and ϵ_i is the error term This equation is as follows; $R_i = \alpha + (1 - \alpha) + r_i (3.5)$ Where R_i is the quantity of blood due to stenosis.

IV. VOLUME FLOW RATE EQUATION

$Q = \pi r^2 \Delta P / 8 \mu L$ where r = artery radius, ΔP is pressure drop, μ is the blood viscosity Womersely Number is given as $\alpha = (r \omega \rho / \mu)^{1/2}$

V. ONE DIMENSIONAL MODEL HYPERBOLIC MODEL

Conservation of mass and momentum will be given by the following equations $\partial A / \partial t + \frac{\partial Q}{\partial x} = 0 \dots (1)$

$$\partial Q / \partial t + \frac{\partial}{\partial x} \left(\frac{\alpha Q^2}{A} \right) + A / \rho \frac{\partial p}{\partial x} = -KQ / A \dots (2)$$

where A is cross sectional Area and Q is the flow rate and ρ is the pressure and K is resistive term.

$$\rho \propto (A/a)^\beta \dots (3)$$

Three Dimensional Model: The Governing equations for three dimensional model will be as

$$\rho (\partial u / \partial t + u \nabla \cdot u) = -\nabla p + \nabla \cdot \tau \dots (4) \quad \nabla \tau = 0$$

Where u is the velocity and τ is the stress

VI. CONSERVATION OF MOMENTUM

The Governing equations of this model will be as

$$F_{xy} = \partial \sigma / \partial x \, dv \dots (5)$$

The normal force resulting in x-direction

$$\tau_{xy} = \tau_{yz} = \alpha \frac{\partial v}{\partial i} \dots (6)$$

The resulting connecting equation will be given by

$$\left(\frac{\partial v}{\partial t} + \frac{\partial v}{\partial x} v + \frac{\partial v}{\partial y} v + \frac{\partial v}{\partial z} v \right) \rho = \frac{\partial \tau}{\partial x} + \frac{\partial \tau}{\partial y} + \frac{\partial \tau}{\partial z} + \rho g_x \dots (7)$$

The result of comprehensive exploring the blood flow through artery

$$\rho \left(\frac{\mu}{r} \right) + \left(\frac{\partial \mu}{\partial r} \right) + \frac{\partial w}{\partial z} = 0 \dots (8)$$

$$\frac{\partial u}{\partial t} + \alpha \frac{\partial u}{\partial r} + \epsilon w \frac{\partial u}{\partial z} - \frac{1}{\alpha e Re} \left[-\frac{\partial p}{\partial r} + \alpha r Re \left(1 + \frac{1}{\beta} \right) \right] = 0 \dots (9)$$

Wall shear Stress: Wall Shear stress refers to tangential force per unit area exerted by the flowing bloods on the endothelial lining of the blood vessels. It arises due to the viscous friction between the blood and vessel walls. Shear stress influences various physiological processes.

Which can be modelled mathematically as $\tau_w = -\mu \frac{\partial w}{\partial r} r = R(z)$.

$$\partial v / \partial t + \partial v / \partial x \cdot v + \partial v / \partial y \cdot v + \partial v / \partial z \cdot v = 0 \dots (10)$$

For numerical solution a parabolic velocity profile was used in the numerical simulation of blood flow through a stenotic artery.

$$U_z = -2U_p (1 - (r/R)^2) \dots (11)$$

This velocity profile ensures a gradual reduction to zero velocity at the walls due to the no slip condition. The outlet boundary condition is specified as zero gradients. Which means the flow is allowed to develop and exits the simulation domain without any additional constraints on the velocity gradient at the outlets.

$$\text{The Viscous stress tensor: } \tau_{ij} = \eta \left(\frac{\partial v_i}{\partial j} + \frac{\partial v_j}{\partial i} \right) \dots (12)$$

VII. NUMERICAL TECHNIQUE

Forward Time Central Space Scheme: the forward Time Central Space Scheme is basically a Finite Difference Scheme which is applied numerically to solve time dependent partial differential equations. And specially applied for parabolic equations like heat diffusion equations. If we summarize the scheme which will be as under

Time Derivatives: Approximated by Forward Differences
Space Derivatives: approximated by central Differences

VIII. RESULTS AND DISCUSSIONS

Blood flow moves in paraxial layers. The velocity is highest in the central of vessels and lowest near the wall due to the friction. This is the most efficient way to transport blood requiring the least amount of energy from heart. It also keeps blood cells from constantly pumping into the arterial walls, which prevents irritation. It was observed that when blood enters the narrow segment and enters the wider vessel again, it can no longer maintain the laminar path. This leads to Eddies and Vortices, Flow Separations, Bruits

High stress can damage the endothelial lining of vessels. Low oscillating stress in dead zones behind the stenosis can actually encourage the build up of more plaques making the condition worse. A stenosis does not significantly impact resting blood until the diameter is reduced by about 50% to 70% and when it is increased 75% to 90% this is the critical point the pressure drop is so great that the body can no longer compensate by tilting

down stream vessels. Stenosis narrowing of an artery, often due to plaque build up significantly affects blood flow through the vessel. A brief summary of the key hemodynamic effects: Increased resistance — According to principles like Poiseuille's law $\text{flow} \propto \text{radius}$, even moderate narrowing sharply raises resistance in the stenotic segment because resistance is inversely proportional to the fourth power of the radius. This creates a pressure drop across the stenosis. Higher velocity in the narrowed area — By the continuity equation ($\text{flow rate} = \text{velocity} \times \text{cross-sectional area}$), blood speeds up through the stenosis to maintain volume flow. This converts pressure energy to kinetic energy (per Bernoulli's principle), causing a further drop in pressure distal to the stenosis

Pressure gradient and drop — A significant trans-stenotic pressure gradient develops, especially at higher flow rates. The drop is nonlinear and becomes more pronounced during increased demand (exercise), as pressure distal to the stenosis falls markedly. Preserved resting flow (up to a point) — At rest, mild-to-moderate stenosis <60-70% diameter reduction often does not reduce distal tissue blood flow because downstream arterioles dilate autoregulation to compensate and lower their resistance. This maintains near-normal resting perfusion.

Reduced flow reserve — The major clinical impact is impaired ability to increase blood flow during stress exercise (reduced coronary or peripheral flow reserve. Significant stenosis typically >70% area reduction or >50% diameter reduction limits maximal flow, leading to ischemia angina in coronary arteries or claudication in peripheral arteries

Other Effects — Turbulence, altered wall shear stress, and potential for post-stenotic dilatation or vortex

formation can occur, contributing to further complications like plaque progression or embolization.

in view of above, stenosis primarily acts as a flow-limiting resistor that has minimal effect on resting flow due to compensatory mechanisms but severely restricts maximal flow and creates adverse pressure velocity changes across the lesion.

REFERENCES

- [1] Dabai, E. A., et al. (2013). Application of nanotechnology in medicine and drug delivery. *Artificial Cells, Nanomedicine, and Biotechnology*, **44**(1), 341–391. <https://doi.org/10.3109/21691401.2013.953336>
- [2] Kojic, M., et al. (2014). A composite smeared finite element for mass transport in capillary systems and biological tissues. *Computer Methods in Applied Mechanics and Engineering*, **274**, 413–437. <https://doi.org/10.1016/j.cma.2014.06.019>
- [3] Collin, E., et al. (2019). Flow patterns and turbulence in blood flow through a stenotic artery. *Chemical Engineering Science*, **64**(7), 1376–1388.
- [4] Beat, A., et al. (2020). Flows in stenotic vessels. *Annual Review of Fluid Mechanics*, **32**, 347–382. ISSN: 1543-4432.
- [5] Rosier, J., et al. (2021). MHD boundary layer analysis of micropolar dusty fluids. *Journal of Molecular Liquids*, **268**, 813–821. <https://doi.org/10.1023/07.1025>
- [6] Krasner, L., & Debar, S. (2022). A numerical investigation of blood flow through the aortic valve. *Journal of Science and Technology*, **17**(5). <https://doi.org/10.56678/jst.v17i05.527>