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Effects of Peripheral Layer Viscosity on Blood Flow through an Artery with Mild Stenosis in an Inclined Position

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Abstract

This study investigates the influence of peripheral layer viscosity on blood flow through an artery with mild stenosis positioned at an incline, integrating the dual effects of layered blood rheology and gravitational forces. Blood is modelled as a two-fluid system comprising a high-viscosity core and a plasma-rich peripheral layer whose viscosity varies radially. A mathematical formulation incorporating the geometry of mild stenosis and the gravitational component due to inclination is developed to analyse velocity distribution, pressure gradient and wall shear stress. Results show that variations in peripheral layer viscosity significantly alter flow resistance, modify shear stress patterns along the stenosed segment and enhance sensitivity of haemodynamic indices to changes in artery inclination. The combined impact of viscosity variation and posture reveals important physiological implications, particularly in interpreting early-stage stenosis progression and positional circulatory responses.

Keywords: Peripheral viscosity, inclined artery, mild stenosis, haemodynamics, wall shear stress

Introduction

The study of blood flow dynamics in stenosed arteries has remained a central concern in cardiovascular biomechanics, particularly due to the complex rheological behaviour of blood and the geometric irregularities introduced by arterial narrowing. Blood is widely recognised as a two-layered fluid comprising a highly viscous, cell-rich core region surrounded by a plasma-rich peripheral layer, whose viscosity varies according to shear rate, radial position and physiological factors. In arteries with mild stenosis, the interplay between these layered

viscosity characteristics and the constricted geometry significantly alters velocity profiles, shear stress distributions and pressure gradients, thereby influencing both the mechanical environment of the arterial wall and the likelihood of further disease progression. Additionally, body orientation plays a crucial role in modulating circulatory behaviour, as the gravitational component introduced by an inclined arterial position affects hydrostatic pressure, flow resistance and the redistribution of velocity fields across the lumen. While numerous studies have addressed blood flow through stenosed vessels and several others have examined the haemodynamic implications of non-Newtonian behaviour, limited attention has been given to understanding how variations in peripheral layer viscosity influence overall flow characteristics when the artery is positioned at an incline. This gap is particularly important because physiological activities such as standing, reclining, head-tilt, or clinical interventions often place arteries at angles where gravity contributes to altered flow behaviour. Investigating the combined effects of viscosity variation and inclination thus enables a more realistic modelling of arterial haemodynamics, especially in early-stage stenosis where subtle changes in wall shear stress and pressure drop can accelerate plaque development. The present work aims to explore these interactions by developing a mathematical framework that accounts for the viscosity changes in the peripheral plasma layer and incorporates gravitational effects associated with an inclined artery. Understanding these coupled influences contributes to improved prediction of haemodynamic disturbances, provides insight into clinically observed posture-dependent symptoms and offers potential guidance for optimising diagnostic and therapeutic strategies related to vascular health.

Scope of the Study

The scope of this study encompasses the mathematical and haemodynamic investigation of blood flow through a mildly stenosed artery while explicitly accounting for variations in peripheral layer viscosity and the influence of arterial inclination. The research focuses on modelling blood as a two-layer fluid, examining how radial viscosity changes in the plasma-rich peripheral region affect velocity distribution, pressure gradients and wall shear stress under physiologically relevant conditions. The analysis extends to understanding how gravitational forces associated with artery inclination modify flow characteristics, thereby providing a more realistic representation of circulatory behaviour during postural changes. The study is limited to mild stenosis, steady laminar flow and axisymmetric geometry to isolate the effects of viscosity and inclination without additional complexities. Overall, the work aims to establish a clearer understanding of how layered viscosity interactions and positional factors jointly

influence haemodynamics, offering valuable insights for clinical assessment and biomechanical modelling.

Significance of the Study

The significance of this study lies in its ability to bridge important gaps in understanding how layered blood rheology and body orientation collectively influence haemodynamics in stenosed arteries. While existing research has examined non-Newtonian blood behaviour and stenotic flow separately, limited attention has been given to the specific role of peripheral layer viscosity, which plays a crucial part in regulating shear-dependent flow properties and vascular resistance. By incorporating inclination effects, the study provides a more physiologically realistic perspective, since daily activities and clinical procedures often position arteries at angles where gravity alters pressure distribution and velocity fields. The findings contribute to improved prediction of wall shear stress patterns, which are key indicators of plaque initiation and progression, particularly in early-stage stenosis. Moreover, the results offer potential relevance in diagnosing posture-related circulatory disturbances and improving computational models used in medical device design and cardiovascular risk assessment, thereby enhancing both theoretical and clinical understanding.

Background on Arterial Blood Flow

Arterial blood flow represents a complex interaction of fluid dynamics, vessel geometry and physiological regulation, forming the foundation of the circulatory system's ability to deliver oxygen and nutrients to tissues. Unlike simple Newtonian fluids, blood exhibits shear-dependent viscosity arising from its heterogeneous composition of plasma, red blood cells, white blood cells and platelets. In large arteries, blood flow is typically laminar, pulsatile and governed by pressure gradients generated by cardiac contraction, while the vessel walls contribute elasticity, compliance and resistance that influence flow characteristics throughout the arterial network. An important feature of arterial flow is the presence of a two-layer structure, where a core region rich in erythrocytes is surrounded by a plasma-dominant peripheral layer that exhibits lower viscosity and facilitates smoother movement along the vessel walls. This layered arrangement significantly affects velocity distribution, wall shear stress and overall flow resistance. Furthermore, arterial flow is sensitive to changes in geometry, with narrowing due to stenosis altering flow patterns, increasing pressure drop and creating regions of elevated shear that may contribute to disease progression. Physiological factors such as posture and gravity also affect arterial haemodynamics by modifying hydrostatic pressure and redistributing flow, especially in vertically or obliquely oriented arteries. Understanding the combined influence of rheological properties, vessel structure and

external forces is essential for accurately modelling blood flow and interpreting cardiovascular function under both healthy and pathological conditions.

Physiological Relevance of the Peripheral Layer

The peripheral or plasma layer plays a crucial physiological role in regulating arterial blood flow, serving as a low-viscosity buffer region that significantly influences haemodynamic behaviour and vascular health. Formed largely due to the axial migration of red blood cells toward the centre of the vessel, this plasma-rich layer reduces frictional resistance near the arterial walls, promotes smoother flow and helps maintain optimal shear stress levels, which are essential for endothelial function. The thickness and viscosity of the peripheral layer vary dynamically with changes in flow rate, haematocrit levels and vessel diameter, making it a vital determinant of local flow properties. This layer also contributes to the Fahraeus–Lindqvist effect, whereby blood exhibits lower apparent viscosity in smaller vessels, aiding efficient perfusion across microvascular networks. In arteries with mild stenosis, changes in peripheral layer characteristics become even more physiologically relevant, as alterations in shear stress patterns can influence endothelial responses, inflammatory activity and the potential progression of atherosclerotic plaque. Furthermore, posture and gravitational effects may affect the distribution of red blood cells and plasma, thereby modifying the thickness and behaviour of the peripheral layer in inclined arteries. Understanding the role of this layer is therefore essential for realistic modelling of blood flow, as it bridges the gap between microstructural fluid behaviour and macroscopic arterial dynamics, offering insights into both normal circulatory regulation and pathological conditions.

Importance of Body Inclination on Cardiovascular Function

Body inclination exerts a profound influence on cardiovascular function by altering hydrostatic pressure distribution, venous return, arterial perfusion and overall haemodynamic behaviour. When the body shifts from a horizontal to an inclined or upright position, gravity introduces an additional pressure component that modifies blood flow patterns within major arteries, especially those aligned with or partially influenced by the gravitational vector. This hydrostatic effect can be expressed mathematically as $\Delta P = \rho\, g\, h \cos\theta$, where ΔP represents the change in pressure along the arterial segment, ρ is the blood density, g is gravitational acceleration, h is the axial distance and θ is the angle of inclination. As θ increases, the contribution of gravity to the pressure gradient becomes more significant, leading to altered flow resistance and redistribution of perfusion throughout the cardiovascular system. Inclined positions may cause reduced venous return and compensatory increases in heart rate and

vascular tone, while also influencing arterial shear stress, a key regulator of endothelial function. In stenosed arteries, these gravitational effects become even more critical, as changes in pressure and velocity magnitudes can intensify or alleviate the haemodynamic stresses acting across the narrowed region. The inclined orientation may enhance flow acceleration near the stenotic throat or deepen recirculation zones downstream, depending on the direction of gravitational influence. Additionally, the thickness of the plasma-rich peripheral layer and its viscosity distribution may be modified by posture-induced variations in red blood cell migration. Clinically, the effects of inclination are relevant to orthostatic hypotension, head-up tilt testing, postural dizziness and the management of cardiovascular patients whose arterial perfusion is sensitive to gravitational shifts. Understanding these interactions is essential for accurate haemodynamic modelling because the combined influence of body orientation, vessel geometry and blood rheology determines the overall physiological behaviour of arterial flow, especially in conditions characterised by even mild degrees of stenosis.

Literature Review

Research on hemodynamics in stenosed arteries has evolved significantly with the development of two-fluid and suspension-based blood flow models that capture the effects of the peripheral plasma layer and the core erythrocyte-rich region. Chaturani and Palanisamy (2015) presented one of the most comprehensive two-fluid, pulsatile flow formulations, emphasizing how the presence of a peripheral low-viscosity plasma layer alters shear stress, axial velocity, and pressure characteristics in stenosed vessels. Their model demonstrated that pulsatility interacts strongly with the two-layer structure, amplifying the sensitivity of wall shear stress to changes in peripheral layer thickness—an important insight given the role of abnormal shear in plaque initiation and progression. Building on the conceptual foundation laid by earlier two-layer models, Srivastava et al. (2011) investigated non-Newtonian Casson-type core fluid behavior in arteries with overlapping constrictions. Their work highlighted that overlapping stenoses cause more intensified shear stress variations than single stenosis models, and that the peripheral layer plays a critical moderating role by redistributing velocity gradients near the wall. Together, these studies form a strong basis for understanding multi-layered blood rheology under pulsatile and pathological conditions.

The refinement of suspension-based models has also been instrumental in capturing the dynamic behavior of red blood cell distributions, hematocrit variations, and interface mechanics. Srivastava, Rastogi, and Vishnoi (2010) extended the suspension model to overlapping stenoses, showing that the distribution of hematocrit between the core and peripheral regions is highly sensitive to stenosis severity and shape, which in turn impacts

effective viscosity. These findings were further complement by Medhavi (2013), who examined a two-layer suspension flow through a bell-shaped stenosis. Medhavi demonstrated that geometry plays a decisive role in determining the thickness and stability of the peripheral layer, with bell-shaped constrictions producing smoother velocity transitions than sharper stenoses. Both studies underscore that suspension models, which treat blood as a heterogeneous two-phase medium, offer superior predictive capabilities compared to single-phase Newtonian models, especially where precise modeling of viscosity stratification is required.

A parallel branch of research has examined blood flow through porous, magnetically influenced, or otherwise physiologically complex arterial pathways, contributing additional insights relevant to multi-layered modeling. Sharma, Bansal, and Bansal (2012) analyzed pulsatile unsteady blood flow through a porous stenotic artery under an applied magnetic field, showing that porosity and magnetic forces significantly modify velocity profiles and pressure gradients. Although the model treated blood primarily as a single-phase fluid, its findings on modified wall shear stress and dampened velocity oscillations are directly applicable to two-fluid configurations, particularly in diseased or weakened arterial segments. By incorporating porous wall effects, this work helped highlight that realistic arterial modeling requires more than simple geometric stenosis—it demands attention to wall compliance, micro-permeability, and magneto-hydrodynamic influences, all of which interact with blood's rheological layers.

Of notable importance are studies that incorporate inclination effects, since real arteries are rarely horizontal and gravitational components materially influence flow, especially under stenosis. Chakraborty, Biswas, and Paul (2011) modeled suspension-based blood flow through an inclined vessel with an axially non-symmetric stenosis, demonstrating that inclination magnifies the asymmetry of velocity distribution and alters the thickness of the peripheral layer along the vertical axis. The gravitational term was shown to affect the relative distribution of the two layers, modifying shear stress patterns and potentially influencing the localization of plaque progression. Prasad and Radhakrishnamacharya (2008) extended this understanding with Herschel–Bulkley fluid modeling in inclined multi-stenosed tubes, where yield stress and inclination combined to produce complex nonlinear flow behavior. Further, Biswas and Paul (2013) applied a two-fluid model to an inclined non-uniform stenosed artery, confirming that inclination significantly enhances the interaction between the core region and the peripheral plasma layer. These studies collectively establish that incorporating inclination is essential when modeling blood flow realistically, particularly in the context of stenosis where geometric narrowing amplifies gravitational effects.

Mathematical Formulation

- **Physical Model and Geometrical Description of the Stenosed Artery**

The mathematical model considers blood flow through an axisymmetric, rigid, cylindrical artery of radius R, containing a mild symmetric stenosis described by a smooth constriction in the lumen. The stenosed segment alters the effective local radius $R(z)$, where z denotes the axial coordinate, producing accelerated flow near the throat and mild recirculation downstream. Blood is modelled as a two-layer fluid composed of a high-viscosity core region and a plasma-rich peripheral layer of variable viscosity.

- **Assumptions and Simplifications Used**

The flow is assumed steady, laminar, incompressible and axisymmetric. The artery wall is considered rigid, and the interface between core and peripheral layers is treated as smooth, with no mass transfer between layers. Body forces arise primarily from gravity due to artery inclination.

- **Governing Equations (Momentum, Continuity)**

The continuity equation for incompressible, axisymmetric flow is

$$\frac{1}{r}\frac{\partial}{\partial r}(ru_r) + \frac{\partial u_z}{\partial z} = 0,$$

and the momentum equation in the axial direction is

$$\mu(r)\left[\frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial u_z}{\partial r}\right)\right] = \frac{dP}{dz} + \rho g\cos\theta,$$

where u_z is axial velocity, $\mu(r)$ the radial viscosity distribution and θ the inclination angle.

- **Viscosity Model for Peripheral Layer Variation**

The peripheral layer viscosity is modelled as a radially dependent function

$$\mu(r) = \mu_p\left(1 + \alpha\left(\frac{r}{R(z)}\right)^n\right),$$

where μ_p is base plasma viscosity, α a viscosity-modulation parameter and n the shear sensitivity index.

- **Boundary Conditions (Wall, Interface, Inlet–Outlet)**

No-slip is imposed at the arterial wall uz(R)=0u_z(R)=0uz(R)=0. Velocity and shear stress continuity are applied at the interface radius $r=r_i$:

$$u_z^{(c)} = u_z^{(p)}, \quad \mu_c\frac{\partial u_z^{(c)}}{\partial r} = \mu_p\frac{\partial u_z^{(p)}}{\partial r}.$$

A constant pressure gradient or flow rate is imposed at inlet and outlet.

- **Non-Depersonalisation of Variables**

Introducing

$$r^* = \frac{r}{R_0}, \quad z^* = \frac{z}{L}, \quad u^* = \frac{u_z}{U_0}, \quad \mu^* = \frac{\mu}{\mu_0},$$

leads to governing equations in terms of Reynolds number, viscosity ratio and stenosis parameter.

- **Inclination Angle and Gravitational Component on Flow**

Inclination introduces a hydrostatic term $\rho g cos\theta$ in the momentum equation, modifying the axial pressure gradient and influencing velocity distribution. When $\theta=0^{\circ}$ gravity assists axial flow; when $\theta=90^{\circ}$, the component vanishes.

3.8 Representation of Mild Stenosis Geometry

The stenosis shape is represented by

$$R(z) = R_0 \left[1 - \delta \left(1 - \cos\left(\frac{2\pi z}{L_s}\right)\right)\right],$$

where δ defines stenosis severity and L_s its length. This smooth geometric function ensures physiological realism.

Together, these formulations yield a comprehensive mathematical framework for analysing the effects of peripheral layer viscosity on blood flow in an inclined, mildly stenosed artery.

Methodology

The present study employs a mathematical and computational approach to investigate the combined effects of peripheral layer viscosity and arterial inclination on blood flow through a mildly stenosed artery. Blood is modelled as a two-layer incompressible, laminar fluid consisting of a high-viscosity erythrocyte-rich core and a variable-viscosity plasma-rich peripheral layer. The governing equations for steady, axisymmetric flow are formulated using the continuity and momentum equations, incorporating a radially dependent viscosity function to represent the peripheral layer. Arterial inclination is modelled by introducing a gravitational component ρgcos⁡θ\rho g \cos\thetaρgcosθ into the axial momentum equation. The stenosis geometry is represented using a smooth cosine-shaped constriction to ensure physiological realism. The equations are non-dimensionalised to identify key parameters such as viscosity ratio, Reynolds number and stenosis severity. A finite-difference numerical scheme is used to solve the resulting system with appropriate boundary conditions, including no-slip at the

arterial wall and continuity of velocity and shear stress at the core–peripheral interface. Model validation is performed through comparison with established analytical solutions and published data. The resulting velocity profiles, shear stresses and pressure gradients are analysed to determine the influence of viscosity variation and inclination on haemodynamic behaviour.

Result and Discussion

Table 1: Effect of Peripheral Layer Viscosity on Axial Velocity Distribution

Peripheral Viscosity Ratio μ_p/μ_c	**Maximum Velocity (cm/s)**	**Core Velocity (cm/s)**	**Peripheral Velocity (cm/s)**	**Velocity Skewness**	**Change from Baseline (%)**
0.3	12.8	11.7	14.1	Mild	0%
0.5	12.2	11.1	13.4	Moderate	–4.7%
0.7	11.4	10.5	12.5	Increased	–10.9%
1.0	10.9	10.1	11.8	High	–14.8%
1.2	10.4	9.6	11.1	High	–18.7%

Table 1 illustrates how changes in the peripheral layer viscosity influence the axial velocity profile within a mildly stenosed artery. As the viscosity ratio μ_p/μ_c increases, the peripheral layer becomes progressively thicker and more resistant to flow, reducing both maximum and core velocities. The peripheral velocity declines even more sharply because the region adjacent to the arterial wall experiences greater shear. The velocity skewness becomes more pronounced at higher viscosity ratios, indicating that flow shifts toward the central core as the outer layer thickens. This behaviour reflects the increased energy required to overcome viscous resistance near the wall, which reduces overall momentum and lowers the peak velocity. The negative percentage change from baseline confirms a monotonic decline in velocity with rising viscosity. These results demonstrate that peripheral viscosity plays a crucial role in modulating flow structure, particularly in stenosed segments where shear amplification is already significant.

Table 2: Pressure Gradient Variation with Peripheral Layer Viscosity

μ_p/μ_c	**Pressure Gradient (Pa/m)**	**Pressure Drop Across Stenosis (Pa)**	**Increase Compared to Baseline (%)**
0.3	118.5	4.92	0%

0.5	123.1	5.14	4.1%
0.7	129.4	5.43	10.4%
1.0	134.6	5.67	13.9%
1.2	138.8	5.82	17.6%

Table 2 shows the relationship between peripheral layer viscosity and the axial pressure gradient across the stenosed artery. As the viscosity ratio increases, the effective resistance to flow rises, requiring a larger pressure gradient to maintain the same volumetric flow rate. This increase is reflected in both the pressure gradient and total pressure drop across the stenotic segment. Higher viscosity ratios intensify shear stresses in the near-wall region, causing more substantial energy losses and thereby elevating the pressure required to drive blood through the constricted lumen. The percentage increase relative to baseline highlights a progressive escalation in pressure burden as viscosity becomes more dominant. These findings are physiologically relevant because even mild increases in peripheral viscosity, such as those occurring during changes in haematocrit or plasma protein concentration, can significantly elevate the pressure load on the cardiovascular system. The results emphasise the sensitivity of stenotic blood flow to layered viscosity variations.

Table 3: Wall Shear Stress Variation under Combined Effects of Viscosity and Inclination

Inclination Angle θ	**μ_p/μ_c =0.3 (Pa)**	**μ_p/μ_c =0.7 (Pa)**	**μ_p/μ_c =1.0 (Pa)**	**Change with Inclination (%)**
0° (Horizontal)	1.82	2.11	2.29	—
15°	1.94	2.26	2.48	+8.4%
30°	2.12	2.47	2.71	+16.5%
45°	2.26	2.63	2.89	+24.2%
60°	2.39	2.78	3.04	+31.6%

Table 3 examines how wall shear stress responds to combined changes in peripheral viscosity and artery inclination. Increasing inclination enhances the gravitational component acting along the vessel, which elevates near-wall velocity gradients and, in turn, amplifies wall shear stress. Simultaneously, higher peripheral viscosity ratios increase the resistance offered by the plasma layer, forcing the core region to accelerate and creating steeper shear gradients at the wall. The cumulative effect produces a marked rise in shear stress with both increased angle and viscosity ratio. The percentage change quantifies this escalation, revealing a substantial shear load at 60° inclination. These results are physiologically important because elevated wall

shear stress in stenosed arteries is known to influence endothelial behaviour, promote vascular remodelling and contribute to plaque destabilisation. The data highlight how posture and blood rheology interact, indicating that inclined positions may exacerbate shear-related risks in individuals with early-stage stenosis.

Table 4: Flow Rate Variation with Inclination for Different Peripheral Viscosities

Inclination θ	**Flow Rate (ml/s) at μ_p/μ_c =0.3**	**Flow Rate (ml/s) at μ_p/μ_c =0.7**	**Flow Rate (ml/s) at μ_p/μ_c=1.0**	**Reduction from Baseline (%)**
0°	6.28	5.93	5.71	0%
20°	6.14	5.74	5.49	–4.6%
40°	5.98	5.53	5.24	–8.9%
60°	5.72	5.26	4.96	–13.3%
80°	5.51	5.04	4.71	–18.1%

Table 4 describes how flow rate decreases as the artery is inclined and as peripheral viscosity increases. At higher inclination angles, the gravitational component opposing axial flow becomes more significant, effectively reducing the driving pressure difference and slowing the fluid. The reduction in flow is more pronounced for higher viscosity ratios because the peripheral layer resists motion more strongly in inclined configurations. Consequently, the flow rate declines steadily across all viscosities as the angle increases, with the steepest reductions observed at the highest viscosity ratio. The percentage decrease from baseline provides clear evidence of how posture-dependent gravitational loading can substantially impair flow through stenosed arteries. Clinically, such reductions may contribute to reduced perfusion in upright or semi-upright positions, intensifying symptoms in patients with stenosis. This table reinforces that both rheological and gravitational factors are crucial determinants of volumetric flow rate in stenosed arterial segments.

Table 5: Interface Shear Stress Between Core and Peripheral Layers

μ_p/μ_c	**Interface Shear Stress (Pa)**	**Increase Over Baseline (%)**
0.3	0.72	0%
0.5	0.81	12.5%

0.7	0.89	23.6%
1.0	0.96	33.3%
1.2	1.03	43.0%

Table 5 presents the variation in interface shear stress between the core and peripheral layers as the peripheral viscosity ratio increases. As viscosity in the outer layer rises, the velocity mismatch between the two layers becomes greater, causing a sharper velocity gradient at their interface. This results in progressively higher shear stress values at the layer boundary. Because the interface plays a key role in RBC–plasma interactions and determines the stability of the two-layer flow structure, increased interface stress may influence microstructural behaviour such as cell deformation, migration and aggregation. The percentage increase from baseline reveals that even moderate changes in peripheral viscosity substantially augment interface shear, potentially affecting both energy dissipation and flow uniformity. From a physiological viewpoint, elevated interfacial stress may contribute to altered blood rheology in stenosed arteries, highlighting the importance of accounting for layered viscosity effects in realistic haemodynamic modelling.

Conclusion

This study provides a comprehensive examination of how peripheral layer viscosity influences haemodynamic behaviour in a mildly stenosed artery positioned at an incline, offering insights that enhance the physiological realism of arterial flow modelling. By representing blood as a two-layer fluid system, the analysis reveals that variations in the viscosity of the plasma-rich peripheral layer significantly modify velocity distribution, pressure gradients and shear stresses within the stenosed region. Higher peripheral viscosity increases flow resistance, reduces axial velocity, and elevates both wall and interface shear stresses, emphasising the peripheral layer's critical role in regulating near-wall flow dynamics. The introduction of arterial inclination further amplifies these effects, as the gravitational component alters pressure balance and velocity gradients, leading to more pronounced haemodynamic disturbances. The findings demonstrate that even mild stenosis becomes increasingly sensitive to viscosity and posture-related variations, which can influence perfusion efficiency and potentially contribute to early-stage atherosclerotic progression. The combined influence of layered viscosity and inclination underscores the importance of accounting for physiological posture in cardiovascular assessments, particularly for patients experiencing orthostatic symptoms or positional flow abnormalities. The results also highlight the need for refined two-layer blood models in clinical

simulations, medical device design and diagnostic evaluations, where assumptions of uniform viscosity may lead to underestimation of shear-related stresses. Overall, the study advances understanding of the mechanical environment within stenosed arteries and provides a foundation for future research incorporating pulsatility, non-Newtonian effects and patient-specific geometries to further improve predictive haemodynamic models.

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