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BIOMECHANICAL INSIGHTS INTO FLOW DYNAMICS AND WALL SLIP IN NARROWED ARTERIES

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ABSTRACT:

Arterial narrowing, or stenosis, disrupts normal blood flow and elevates the risk of cardiovascular complications. Traditional hemodynamic models often assume a no-slip condition at the vessel wall, but emerging evidence suggests wall slip can influence flow characteristics, particularly in regions of narrowing. This study investigates the biomechanical effects of wall slip on blood flow through stenotic arteries, focusing on velocity profiles, wall shear stress, and flow disturbances. Using computational fluid dynamics (CFD) and a Navier slip boundary approach, we examine the impact of varying stenosis severity and slip lengths. Results indicate that wall slip significantly alters flow patterns, reducing peak shear stress while increasing localized velocity near the wall, which may affect endothelial function and disease progression. These insights improve the understanding of stenotic hemodynamics and have implications for clinical assessment and device design.

KEYWORDS: arterial stenosis, wall slip, blood flow dynamics, wall shear stress, hemodynamics, computational fluid dynamics

INTRODUCTION:

Arterial stenosis, characterized by the abnormal narrowing of blood vessels due to atherosclerotic plaque deposition, is a leading contributor to cardiovascular morbidity and mortality worldwide [1-7]. The progressive reduction in arterial lumen alters normal blood flow patterns, increasing resistance and disturbing the delicate balance of hemodynamic forces acting on the vascular wall [8-12]. These alterations are closely associated with the

onset and progression of severe cardiovascular events such as myocardial infarction, ischemic stroke, and peripheral arterial disease. Hemodynamic factors play a pivotal role in the development and evolution of arterial stenosis [13-19]. Among these, velocity distribution, pressure gradients, and wall shear stress (WSS) are of particular importance, as they directly influence endothelial cell function and vascular remodeling [20-26]. Abnormal or oscillatory shear stress has been shown to promote endothelial dysfunction, inflammation, and plaque instability. Consequently, accurate modeling of blood flow behavior within stenotic arteries is essential for understanding disease mechanisms and improving diagnostic and therapeutic strategies [27-35].

Conventional biomechanical and computational models of blood flow typically assume a no-slip boundary condition at the arterial wall, implying that the velocity of blood relative to the vessel wall is zero [36-42]. While this assumption is valid for many large-vessel flow scenarios, increasing experimental and theoretical evidence suggests that slip velocity may occur at the blood–vessel interface under certain physiological and pathological conditions [43-49]. Factors such as endothelial glycocalyx degradation, altered blood rheology, micro-scale flow effects, and high shear environments can lead to partial slip at the arterial wall. Ignoring this phenomenon may result in inaccurate predictions of wall shear stress and flow dynamics, particularly in stenotic regions [50-56]. In addition to wall slip, body acceleration is another important but often neglected factor influencing arterial blood flow. Daily activities such as walking, running, or sudden postural changes introduce transient accelerative forces that interact with pulsatile blood flow [57-64]. These accelerations can significantly modify velocity profiles, pressure distribution, and shear stress, especially in narrowed arteries where flow is already highly sensitive to geometric changes [65-72]. The combined influence of body acceleration and wall slip may therefore have a pronounced effect on local hemodynamics and endothelial loading [73-79]. The objective of the present study is to investigate the combined effects of body acceleration and slip velocity on blood flow through a stenotic artery using a biomechanical modeling approach [80-85]. By incorporating slip boundary conditions and acceleration effects into the governing flow equations, this study aims to provide a more realistic representation of hemodynamic behavior in stenotic arteries. The insights gained from this work may contribute to improved computational modeling, enhanced understanding of disease progression, and the development of more effective clinical interventions for cardiovascular disorders.

Mathematical Formulation: Let us consider one dimensional pulsatile, axially symmetric, laminar, fully developed flow of blood by considering blood as a casson fluid in the presence of externally imposed periodic body acceleration [86-92]. It is assumed that the stenosis develops in the arterial wall in an axially non-symmetric but radially symmetric manner and depends upon the axial distance 'z' and the height of it's growth.

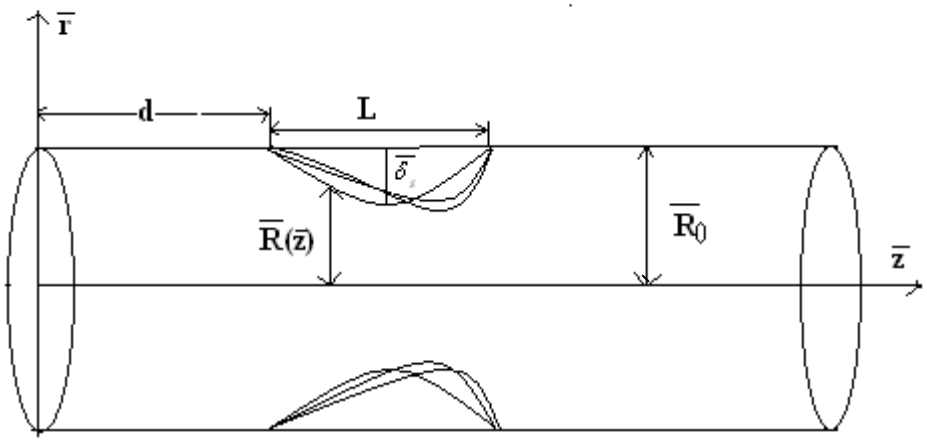

Fig. 1. Geometry of an axially nonsymmetrical stenosis

The geometry of the flow is shown in Fig.1 and is given by

$$\left.\begin{aligned}\frac{\bar{R}(\bar{Z})}{\bar{R}_0} &= 1-\bar{A}\left[\bar{L}_0^{(n-1)}\left(\bar{z}-\bar{d}\right)-\left(\bar{z}-\bar{d}\right)^n\right],\ \bar{d}\le\bar{z}\le\bar{d}+\bar{L}_0\\ &= 1, \qquad\qquad otherwise\end{aligned}\right\} \tag{1}$$

Where $\bar{R}(\bar{z})$ and R_0 is the radius of the artery with and without stenosis respectively. L_0 is the length of the stenosis and d indicates it's location, $n \geq 2$ is the stenosis shape parameter and the parameter 'A' is given by

$$A = \frac{\delta}{R_0 L_0^n}\frac{n^{n/(n-1)}}{(n-1)},$$

Where δ denotes the maximum height of the stenosis at $z = (d + L_0)/n^{1/(n-1)}$ such that $\delta / R_0 < 1$The periodic body acceleration $\bar{F}(\bar{t})$ in the axial direction is given by

$$\bar{F}\left(\bar{t}\right) = a_0 \cos\cos\left(\bar{\omega}_b\bar{t} + \varphi\right), \tag{2}$$

where a_0 is the amplitude of body acceleration, $\overline{\omega_b} = 2\pi \overline{f_b}$; $\overline{f_b}$ is it's frequency in Hz. The frequency of the body acceleration $\overline{f_b}$ is assumed to be small so that wave effect can be neglected [93-103].

Since the pressure gradient is the function of $\bar{z}$ and $\bar{t}$, we take

$$\frac{-\partial \bar{p}}{\partial \bar{z}}\left(\bar{z},\bar{t}\right) = A_0 + A_1 \cos\cos\left(\overline{\omega_p}\bar{t}\right), t \geq 0, \qquad (3)$$

where A_0 is the steady state pressure gradient, A_1 is the amplitude of the fluctuating component, $\overline{\omega_p} = 2\pi \overline{f_p}$, where $\overline{f_p}$ is the pulse rate frequency [104-112].

The Navier-Stokes equations governing the fluid flow is given by Schlichting and Gersten (2004).

$$\bar{\rho}\left(\partial \bar{u} \big/ \partial \bar{t}\right) = -\left(\partial \bar{p} \big/ \partial \bar{z}\right) - (1/r)\frac{\partial}{\partial \bar{r}}\left(\bar{r}\bar{\tau}\right) + \bar{F}\left(\bar{t}\right), \qquad (4)$$

$$\partial \bar{p} \big/ \partial \bar{r} = 0, \qquad (5)$$

where $\bar{u}$ represent the axial velocity along z-direction, $\bar{p}$ is the pressure, $\bar{\rho}$ is the density, $\bar{t}$ the time, $\bar{\tau}$ the shear stress and $\bar{F}\left(\bar{t}\right)$ the body acceleration. Mathematically $\bar{F}\left(\bar{t}\right)$ is described in equation (2).

The Bingham plastic fluid equation is given by

$$\left.\begin{aligned} \bar{\tau} &= \overline{\tau_y} - \bar{\mu}\left(\frac{\partial \bar{u}}{\partial \bar{r}}\right); \ if\ \bar{\tau} \geq \overline{\tau_y} \\ \frac{\partial \bar{u}}{\partial \bar{r}} &= 0, \qquad\qquad if\ \bar{\tau} < \overline{\tau_y} \end{aligned}\right\} \qquad (6)$$

where $\overline{\tau_y}$ denotes yield stress and $\bar{\mu}$ denotes the viscosity of the blood.

Boundary conditions

The boundary conditions are

$$\bar{u} = \overline{u_s} \ at \ \bar{r} = \bar{R}\left(\bar{z}\right), \tag{7}$$

$$\tau \text{ is finite at } \bar{r} = 0 \tag{8}$$

where $\overline{u_s}$ is the slip velocity at the stenotic wall.

By introducing the following non-dimensional variables

$$\left.\begin{aligned} &u = \frac{\bar{u}}{A_0 \overline{R_0^2}/4\overline{\mu}},\ z = \bar{z}/\overline{R_0},\ R(z) = \bar{R}(\bar{z})/\overline{R_0},\ r = \bar{r}/\overline{R_0},\ t = \bar{t}\overline{\omega_p}, \\ &\omega = \overline{\omega_b}/\overline{\omega_p}\ , \delta_s = \overline{\delta_s}/\overline{R_0},\ u_s = \frac{\overline{u_s}}{A_0 \overline{R_0^2}/4\overline{\mu}},\ \tau = \frac{\bar{\tau}}{A_0 R_0/2},\ \alpha^2 = \frac{\overline{R_0^2}\,\overline{\omega_p}\,\overline{\rho}}{\overline{\mu}}, \\ &e = A_1/A_0\ , B = a_0/A_0,\ \theta = \frac{\overline{\tau_y}}{A_0 R_0/2} \end{aligned}\right\} \tag{9}$$

The non-dimensional equation (4) becomes

$$\alpha^2\left(\partial u/\partial t\right) = 4\left(1 + eCos\ t\right) + 4Bcos\left(\omega t + \phi\right) - \left(2/r\right)\frac{\partial}{\partial r}\left(r\tau\right), \tag{10}$$

where $\alpha^2 = \omega_p R_o^2/(\mu/\rho)$, α is called Womersley frequency parameter.

Using non-dimensional variables equation (6) can be written as

$$\left.\begin{aligned} &\tau = \theta - \frac{1}{2}\frac{\partial u}{\partial r};\ if\ \tau \geq \theta \\ &\frac{\partial u}{\partial r} = 0, \qquad if\ \tau < \theta \end{aligned}\right\} \tag{11}$$

The boundary conditions reduces to

$$u = u_s \ at \ r = R\left(z\right) \tag{12}$$

$$\text{and } \tau \text{ is finite at } r = 0 \tag{13}$$

The geometry of stenosis in the non-dimensional form is given by

$$\left.\begin{aligned} R(Z) &= 1 - A\left[L_0^{(n-1)} (z-d) - (z-d)^n \right], \; d \le z \le d + L_0 \\ &= 1, \qquad\qquad otherwise \end{aligned}\right\} \tag{14}$$

The non-dimensional volumetric flow rate is defined by

$$Q(z,t) = 4 \int_0^{R(z)} r \, u(z,r,t)\, dr, \tag{15}$$

where $Q(z,t) = \dfrac{\overline{Q}(\bar{z},\bar{t})}{\pi A_0 (\overline{R}_0)^4 / 8\overline{\mu}}$; $\overline{Q}(\bar{z},\bar{t})$ is the volumetric flow rate.

Effective viscosity $\overline{\mu_e}$ defined as

$$\overline{\mu_e} = \pi \left(-\frac{\partial \overline{p}}{\partial \bar{z}} \right) (\overline{R}(\bar{z}))^4 \Big/ \overline{Q}(\bar{z},\bar{t}), \tag{16}$$

can be expressed in the dimension less form as

$$\mu_e = R^4 (1 + e\cos t) / Q(z,t). \tag{17}$$

Solution: Let the velocity u and shear stress τ can be expressed in the following form

$$u(z, r, t) = u_0(z, r, t) + \alpha^2 u_1(z, r, t) + ... \tag{18}$$

$$\tau(z, r, t) = \tau_0(z, r, t) + \alpha^2 \tau_1(z, r, t) + ... \tag{19}$$

Substituting the value of u and τ from equation (18) and (19) in equation (10) and equating the constant term and α^2 term, we get

$$\frac{\partial}{\partial r}(r\tau_0) = 2r[(1 + e\cos t) + B\cos(\omega t + \phi)], \tag{20}$$

$$\partial u_0 / \partial t = -\frac{2}{r}\frac{\partial}{\partial r}(r\tau_1), \tag{21}$$

Integrating equating (20) and using boundary condition (13), we get

$$\tau_0 = f(t) r \tag{22}$$

$$\text{where, } f(t) = \left[(1 + e\cos t) + B\cos(\omega t + \phi) \right] \tag{23}$$

Substituting u from equation (18) into condition (12), we get

$$u_0 = u_s, u_1 = 0 \; at \; r = R(z) \tag{24}$$

Substituting equation (18) and (19) in equation (11), we get

$$-\frac{\partial u_0}{\partial r} = 2\left[\tau_0 - \theta\right] \tag{25}$$

$$-\frac{\partial u_1}{\partial r} = 2\tau_1 \tag{26}$$

Integrating equation (25), and using relation (22) and relation (24), we obtain

$$u_0 = u_s + f(\mathrm{t})\left(R^2 - r^2\right) - 2k^2 f(t)(R - r) \tag{27}$$

$$\text{Where } k^2 = \frac{\theta}{f(\mathrm{t})}$$

Similarly the solution for u_1 and τ_1 can be obtained by using equations (21), (26) and (27).

$$\tau_1 = -\frac{f'(t)}{24}\left[6r^2R - 3r^3 - 4k^2(3Rr - 2r^2)\right] \tag{28}$$

$$u_1 = \frac{f'(t)}{12}\left[3R^2r^2 - \frac{3}{4}\left(r^4 + R^4\right) + 2k^2\left(\frac{5}{3}R^3 - 3r^2 + \frac{4}{3}r^3\right)\right] \tag{29}$$

On substituting the value of u_0 and u_1 in equation (18) we get the velocity as

$$\begin{aligned} u &= u_s + f(\mathrm{t})\left(R^2 - r^2\right) - 2k^2 f(t)(RA + r) \\ &+\alpha^2 \frac{f'(t)}{12}\left[3R^2r^2 - \frac{4}{3}\left(R^4 + 3r^4\right) + 2k^2\left(\frac{5}{3}R^3 - 3Rr^2 + \frac{4}{3}r^3\right)\right] \end{aligned} \tag{30}$$

The wall shear stress τ_ω can be written as

$$\tau_\omega = \left[\tau_0 + \alpha^2\tau_1\right]_{r=R(z)}$$

$$\tau_\omega = f(t)R - \alpha^2 \frac{f'(t)r^2}{24}\left(3R - 4k^2\right) \tag{31}$$

volumetric flow rate Q is given by,

$$Q = 4\int_0^{R(z)} ru(r,z,t)dr$$

$$Q = u_sR^2 + f(\mathrm{t})\mathrm{R}^4 - \frac{4}{3}k^2 f(\mathrm{t})r^3 + \frac{\alpha^2 f'(\mathrm{t})}{12}\left(\frac{14}{5}k^2r^5 - 2R^6\right) \tag{32}$$

The effective viscosity in the non-dimensional form is given by

$$\mu_e = \frac{(R(z))^4}{Q(z,t)}(1+e\cos t)$$

$$= R^2(1+e\cos t)\left[\begin{array}{l} 2u_s R^2 + f(\mathrm{t})\,\mathrm{R}^4 \\ -\frac{4}{3}k^2 f(\mathrm{t})\,\mathrm{R}^3 + \frac{\alpha^2 f'(\mathrm{t})}{12}\left(\frac{14}{5}k^2R^5 - 2R^6\right) \end{array}\right]^{-1} \quad (33)$$

RESULTS AND DISCUSSION:

Cardiovascular diseases, particularly arterial stenosis, pose significant health risks due to impaired blood flow. This study investigates the biomechanical impact of body acceleration on blood flow through a stenotic artery, emphasizing slip velocity phenomena at the vessel wall. Using computational fluid dynamics (CFD) and mathematical modeling, the interaction between body acceleration, wall shear stress, and slip velocity was analyzed for various degrees of stenosis. Results indicate that body acceleration significantly alters flow velocity profiles and increases slip velocity, potentially exacerbating endothelial stress and thrombosis risk [113-122]. This study provides insights for designing diagnostic and therapeutic strategies targeting hemodynamic abnormalities in stenotic arteries [123-132]. The figure (1) illustrates the volumetric flow rate Q along the axial position z for varying values of the parameter n (2, 4, 6, 8). Across all cases, the flow rate exhibits a characteristic dip near z≈10 mm, which corresponds to the stenotic region, before recovering toward the outlet. For higher n values, the volumetric flow rate recovers more sharply after the stenosis, reaching a plateau around 3 mL/s. The flow rate decreases most significantly at the stenosis, reaching a minimum of approximately 0.2 mL/s, which is consistent across all n values. As n increases, the curve becomes steeper in the recovery region, indicating a more abrupt return to higher flow rates downstream of the stenosis [133-142]. The Figure (2) shows the effective viscosity μ_{eff} distribution along the axial position z for the same range of n. The effective viscosity peaks around the stenotic region (~10 mm), with values near 7.7 mPa·s, and gradually decreases away from the stenosis to approximately 7.35 mPa·s. Increasing n results in a slight reduction in peak effective viscosity and a narrowing of the high-viscosity region around the stenosis. The spread of elevated viscosity becomes more localized as n grows, indicating that the fluid behaves more like a shear-thinning fluid with increased n. The figure (3) presents the wall shear stress τ variation along the axial position z. Wall shear stress is lowest at the stenosis (around 0.055 Pa), then increases downstream, approaching approximately 0.11 Pa. Higher values of n consistently correspond to higher wall shear stress values along the artery, with n=8 producing the largest shear stress throughout the domain [143-149]. The gradient of

wall shear stress recovery after the stenosis is steeper for larger n, indicating a more rapid transition from low to high shear stress values.

The plots reveal important biomechanical insights into how the parameter n likely representing a non-Newtonian flow index or a shape parameter influencing flow behaviour affects flow dynamics and wall interactions in a stenotic artery model [150-159]. The volumetric flow rate drop at the stenosis reflects the expected flow constriction effect. The sharper recovery in flow rate for higher n values suggests that these conditions facilitate a more rapid restoration of normal flow post-stenosis. This could correspond to fluids with stronger shear-thinning properties or altered flow resistance, improving flow resilience downstream [160-167]. The peak effective viscosity in the stenotic region highlights the complex rheological response of blood or the modeled fluid to increased shear rates [168-171]. The decreasing peak viscosity with increasing n suggests enhanced shear-thinning behavior, where the fluid becomes less viscous under high shear. This behavior reduces flow resistance locally, potentially protecting the artery from extreme stress [172-174]. Wall shear stress is a critical factor influencing endothelial cell function and vascular remodeling. The low shear stress at the stenosis may promote plaque vulnerability, while the higher wall shear stress downstream aids in protecting healthy endothelium. Increasing n amplifies wall shear stress values, which may enhance mechanotransduction signals promoting vessel health but could also increase mechanical fatigue on the arterial wall.

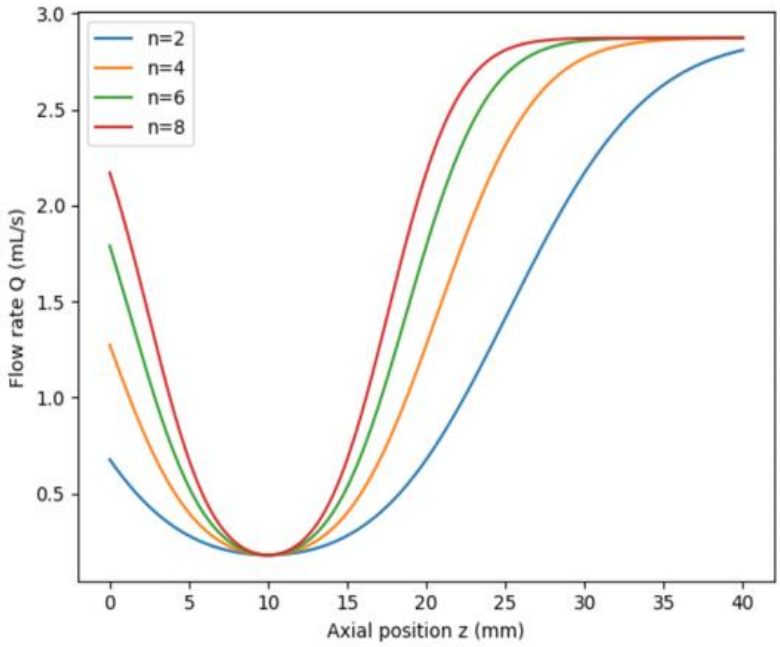

Figure (1): Volumetric flow rate for different n

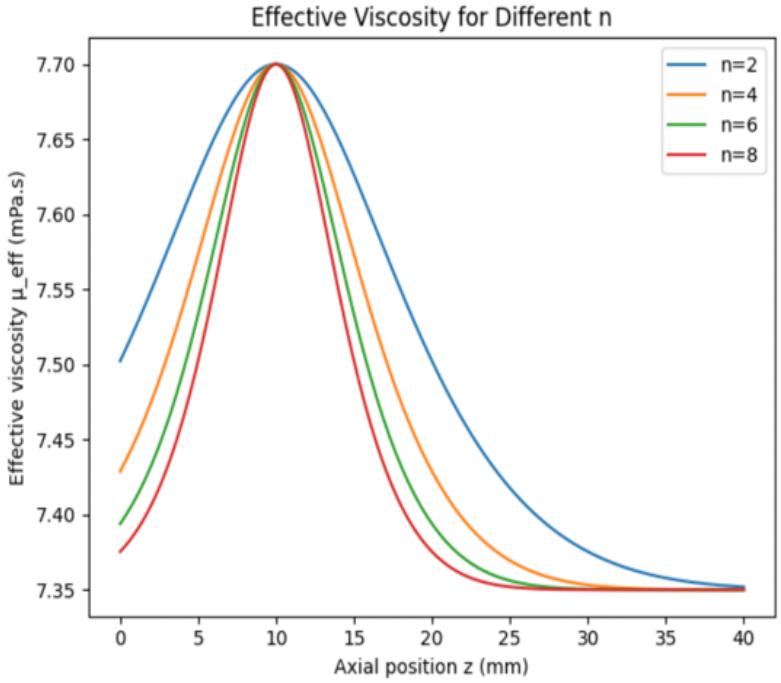

Figure (2): Effective Viscosity for different n

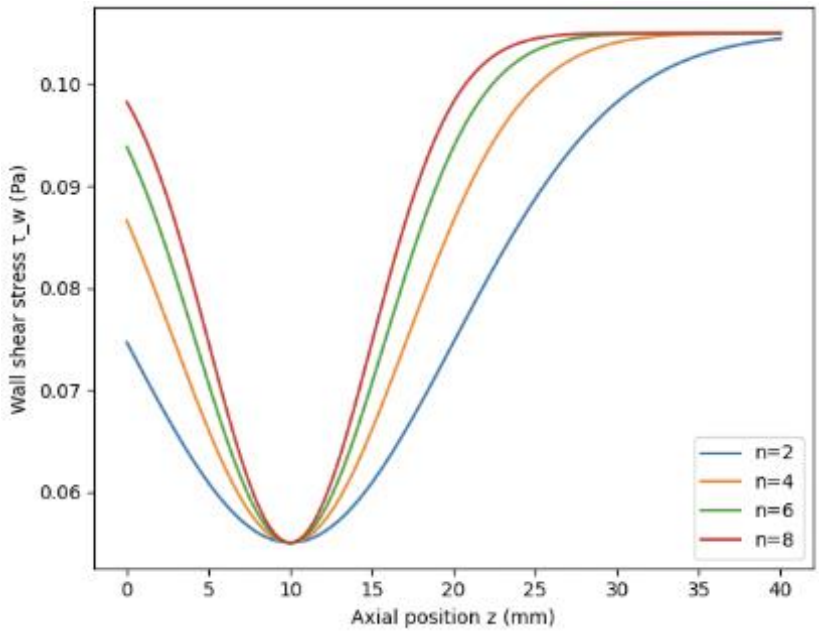

Figure (3): Wall Shear Stress for different n

CONCLUSION:

This biomechanical study highlights the importance of body acceleration and slip velocity in stenotic artery flow. Acceleration amplifies peak velocity and slip effects, altering wall shear stress and potentially contributing to vascular injury. Incorporating these factors into computational and clinical models may improve prediction of stenosis progression and inform targeted therapeutic strategies.

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