

ANALYSIS OF BLOOD FLOW THROUGH STENOSED ARTERY WITH EINSTEIN VISCOSITY

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Abstract

The presence of arterial stenosis significantly disrupts normal flow, posing heightened risks and exerting a greater impact on the cardiovascular system compared to other geometric abnormalities. The investigation of flow parameters in an artery with mild stenosis necessitates analyzing the varying viscosity of blood from the central core line to the vessel wall. This study employs the Navier-Stokes equation in cylindrical polar form to analyze fluid dynamics in axisymmetric directions, accounting for the effective viscosity of blood at radial distances (Einstein coefficient of blood viscosity). By solving this equation with appropriate boundary conditions, analytical expressions for the velocity profile, volumetric flow rate, pressure drop, wall shear stress, and the ratios of pressure drop and shear stress are obtained for the stenosed artery using the Einstein coefficient of blood viscosity. Additionally, variations in plasma viscosity and hematocrit are examined concerning these flow parameters in the stenosed artery region. These findings underscore the complexity of blood flow dynamics, emphasizing the critical importance of incorporating significant factors such as the Einstein coefficient of blood viscosity to advance our understanding of vascular physiology in the presence of stenosis.

Keywords: Arterial Stenosis, Einstein Viscosity, Hematocrit, Hemodynamic Parameters, Plasma Viscosity.

1 INTRODUCTION

Atherosclerotic plaque is formed by the accumulation of cholesterol, fat, and other foreign particles. Moreover, aberrant tissue development results the stenosis, which restricts blood flow [17, 23]. The buildup of low density lipoproteins, and other particles on the arterial wall is termed atherosclerosis, and it is one of the most prevalent diseases affecting the cardiovascular system. This may arise from low or oscillatory wall shear rates [26]. Stenosis in an artery typically results from the accumulation of cholesterol-rich particles in the interior of an artery, which causes constriction of blood vessel and loss of elasticity that can cause a heart attack or stroke. In the regions where the blood flow is disturbed and at low wall shear stress, narrowing of the lumen may occur. Flow

disturbances are established and enhanced when plaque develops and encroaches the lumen [22, 23]. Heart diseases, such as ischemia, atherosclerosis, and angina pectoris, are among the leading causes of death worldwide. After heart disease, stroke is the second leading cause of mortality. The major cause of stroke is due to the blockage of blood vessel [21]. Rheological behavior of blood in arteries with varying sizes and shear rates. Researchers consider these factors when studying conditions such as mild stenosis in large-diameter arteries and high shear rates, recognizing the transition from non-Newtonian to Newtonian behavior [7, 11, 12]. When analyzing medical imaging results such as angiograms or ultrasound scans. Each type of stenosis shape can have specific hemodynamic implications and may influence the severity and consequences of the blood flow obstruction [24]. Analyzing blood flow through different forms of stenosis is essential to understanding the cardiovascular system and detecting possible health problems [6]. Each of these stenoses obstruct the flow after clotting the blood [34]. When the stenosis worsens flow is significantly altered, finally resulting in the onset of cardiovascular illnesses [5]. Heart diseases are indeed significant consequences of stenosis, particularly when it affects the blood vessels supplying the heart (coronary arteries) or the brain (carotid arteries). Stenosis refers to the narrowing of these arteries, which can restrict blood flow and lead to various cardiovascular diseases. The causes of stenosis can be multifactorial [8]. Theoretical and experimental investigations into the effects of restrictions on blood flow parameters such as speed, viscosity, and resistance are essential for understanding the hemodynamics of blood flow in various physiological and pathological conditions [8, 15, 34].

The hematocrit concentration, the white blood cell, and the platelet count, is a crucial component of a person's complete blood count findings. A blood test determines the number of blood cells per unit volume of blood in our bodies [18, 20]. The hematocrit, which measures red blood cell content, is typically 40% for women and 45% for men [20]. The relationship explained the viscosity, hematocrit, and velocity profile of an ordinary coronary artery. The observed decrease in velocity profiles with increasing hematocrit [4]. According to research, there are several important elements that affect wall shear stress in the blood flow through a tapered artery, including blood velocity, hematocrit percentage, stenosis height, and the slope of the artery [2]. These findings emphasize the interplay between hematocrit, catheter size, and the presence of stenosis in shaping the hemodynamics of blood flow. The observations in catheterized arteries highlight the importance of considering medical interventions, such as catheterization [30]. It has been discovered that the impedance varies with catheter size, hematocrit, and stenosis size (length and height), but it decreases with shape parameters [31]. Wall shear stress is reduced with increasing blood velocity and decreasing arterial slope, increases with increased stenosis height and porous features, and reduces with increasing hematocrit [28]. Investigated how human carotid arteries were affected by pressure gradients, wall shear stress, blood velocity, and volumetric flow rate. As the hematocrit and viscosity rise, the arterial wall shear stress falls, which is a sign that the heart rate is rising [19]. A mathematical analysis of blood flow reveals that higher hematocrit (H) and blood viscosity reduce wall shear stress, implying a faster heartbeat rate. Additionally, decrease in the

arterial wall shear stress caused by a rise in hematocrit (H) damage the veins that surround the arteries. The relationship between hematocrit and blood pressure gradient demonstrates that blood clotting in the human heart can lead to death [18]. Such atherosclerosis destroys the cardiovascular system because blood flow to the heart may be blocked completely, if platelets are activated by extremely high shear stress near the peak of the stenosis [27].

Singh et al. [27] have assumed slightly radially asymmetric stenosis. Misra et al. [16] have discovered analytical expressions for velocity distribution, pressure gradient, total angular velocity, wall shear stress, flow rate, and resistance to fluid motion. Mandal and Chakravarty [14] studied that at low shear rates and in arteries with tiny radii, blood behaves as a non-Newtonian fluid. Harjeet et al. [10] have taken blood as a Newtonian fluid for higher resistance and shear stress encountered during a narrow catheterization. As the catheter size grows, the resistance rises in proportion to the height of the stenosis. Young and Tasi [33] created and empirically validated an approximation formula for predicting pressure drops in stenotic areas. Seeley and Young [25] have taken two blunt plugs to study the pressure drop in the area of multiple stenosis. Biswas and Chakravarty [2] investigated the pulsatile blood flow through a tapering artery with moderate stenosis with slip velocity at the arterial wall. According to Chaturani and Ponalagusamy [7] blood has a nonzero yield stress for low shear rate because erythrocytes deposit, rouleaux are found in the area with low shear stress. Srivastava [29] discovered that the catheter size, hematocrit, and stenosis size all rise the flow resistance and ultimately flow parameter are affected. Onitilo and Usman [18] investigated the hypothesis that increasing hematocrit and viscosity would reduce artery wall shear stress. Haldar et al. [9] have studied other significant factors influencing blood flow in the stenosed artery include velocity, wall shear stress, percentage of hematocrit, and viscosity of the blood. Bali and Awasthi [3] Evaluated blood viscosity as a function of hematocrit and distance from center, and the flow is influenced by the external magnetic field. Verma and Parihar [32] proposed that blood flow in an artery with several minor stenoses would be affected by hematocrit. Their theoretical modeling made the assumption that blood viscosity would follow a linear relationship with hematocrit. Alizadehard et al. [1] has studied red blood cell deformation in microvessels for different vessel diameters, hematocrits, and shear rates. Using a practical-based model with partial computing, blood flow in circular tubes with widths ranging from $9\mu\text{m}$ to $50\mu\text{m}$, 20% to 45% of hematocrit, and shear rate of 20 s^{-1} to 150 s^{-1} .

The literature review mentioned above provides evidence of how stenosis affects blood flow. In this study, we looked at how blood viscosity in plasma, hematocrit, and the position and size of stenosis affected blood flow parameters with mild stenosis. We have used the Navier-Stokes equation in cylindrical polar form in axisymmetric directions. Results for the velocity profile, volumetric flow rate, shear stress ratio, and pressure drop ratio that include the plasma viscosity and hematocrit term can be obtained by analytically solving the equation.

2 METHODS

Considering the continuous blood flow through symmetrical and stenosed artery. Let's say that R_0 and R stand for the artery's radius without and with stenosis respectively for a circular artery. We have considered the flow in axial direction only in an artery with stenosis.

2.1 Model Equation

In this mathematical model, advanced models are built upon the laminar and axisymmetric assumptions, providing a fundamental knowledge of blood flow in stenosed arteries. In a cylindrical polar coordinate system, we applied the Navier-Stokes equations. Three velocity components v^r , v^θ , v^z are considered along the r , θ , and z direction respectively. The mass and momentum balance equation for blood flow through an artery in cylindrical polar forms are [12]

$$\frac{1}{r} \frac{\partial}{\partial r}(rv^r) + \frac{\partial}{\partial z}(v^z) = 0 \quad (1)$$

$$\rho \left(\frac{\partial v^r}{\partial t} + v^r \frac{\partial v^r}{\partial r} + v^z \frac{\partial v^r}{\partial z} \right) = -\frac{\partial p}{\partial r} + \mu \left(\frac{\partial^2 v^r}{\partial r^2} + \frac{\partial^2 v^r}{\partial z^2} + \frac{1}{r} \frac{\partial v^r}{\partial r} - \frac{v^r}{r^2} \right) \quad (2)$$

$$\rho \left(\frac{\partial v^z}{\partial t} + v^r \frac{\partial v^z}{\partial r} + v^z \frac{\partial v^z}{\partial z} \right) = -\frac{\partial p}{\partial z} + \mu \left(\frac{\partial^2 v^z}{\partial r^2} + \frac{\partial^2 v^z}{\partial z^2} + \frac{1}{r} \frac{\partial v^z}{\partial r} \right) \quad (3)$$

where r is the radial coordinate, p is pressure, ρ is blood density and μ is the viscosity of the blood.

We assume that in the axisymmetric flow, $v^\theta = 0$ and that v^r , v^z , and p are independent of θ . For the continuous flow of blood, viscosity μ and density ρ are considered to be constant. The velocity component v is parallel to the z -axis. As only axially symmetric flow along the z -axis has been taken into consideration, then $v^r = 0$, $v^\theta = 0$, and $v^z = v$ after that equations (1) and (3) becomes.

$$\frac{\partial v}{\partial z} = 0, \quad 0 = -\frac{\partial p}{\partial r}, \quad 0 = -\frac{\partial p}{\partial z} + \mu \left(\frac{\partial^2 v}{\partial r^2} + \frac{1}{r} \frac{\partial v}{\partial r} \right) \quad (4)$$

Suppose pressure term as $P(z) = -\partial p / \partial z$, equation (4) reduces to

$$-P(z) \frac{r}{\mu} = \frac{\partial}{\partial r} \left(r \frac{\partial v}{\partial r} \right). \quad (5)$$

The Einstein coefficient of blood viscosity (the effective viscosity of blood at a radial distance r) is given as [13, 20].

$$\mu(r) = \mu_0 [1 + \beta h(r)] \quad (6)$$

where μ_0 is the plasma viscosity, β is a constant that characterizes the dependence of viscosity on hematocrit, $h(r)$ is the hematocrit at radial distance r , and $\beta h(r)$ represents the additional viscosity due to the presence of red blood cells is described by the formula

$$h(r) = H \left[1 - \left(\frac{r}{R_0} \right)^m \right] \quad (7)$$

here m is the number of stenosis, we take $m = 1$, from (6) and (7)

$$\mu(r) = \mu_0 \left[1 - \beta H - \beta H \left(\frac{r}{R_0} \right) \right]$$

put $a_2 = \beta H$, $a_1 = 1 + a_2$

$$\mu(r) = \mu_0 \left[a_1 - a_2 \left(\frac{r}{R_0} \right) \right] \quad (8)$$

2.2 Geometry of Stenosis

Figure 1 illustrates the shape of the stenosis formed by the deposition of layer in the interior of an artery with a cylindrical shape. we have from geometry of stenosis [12]

$$\frac{R}{R_0} = 1 - \frac{\delta}{2R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) \quad (9)$$

where R and R_0 are the radii with and with out stenosis, and δ is the maximum thickness [12]. The boundary condition as stated by [12]

$$v = \begin{cases} 0 & \text{at } r = R, \\ 0 & \text{at } r = R_0, \end{cases}$$

and

$$\frac{\partial v}{\partial r} = 0 \text{ at } r = 0$$

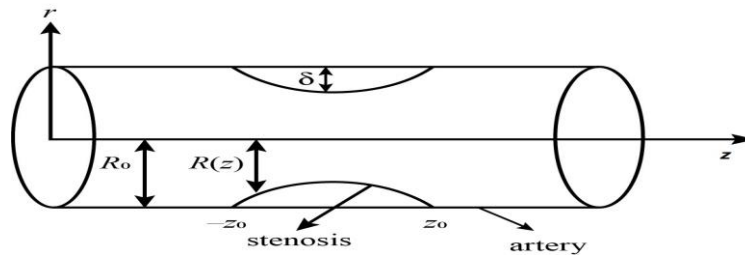


Figure 1: An artery with stenosis shown schematically

2.3 Velocity Profile

From equations (5) and (8)

$$\frac{Pr}{\mu_0 \left(a_1 - a_2 \left(\frac{r}{R_0} \right) \right)} + \frac{\partial}{\partial r} \left(r \frac{\partial v}{\partial r} \right) = 0$$

$$\frac{Pr}{\mu_0 a_1} \left(1 - \frac{a_2 r}{a_1 R_0} \right)^{-1} + \frac{\partial}{\partial r} \left(r \frac{\partial v}{\partial r} \right) = 0.$$

Using binomial expansion and neglect the higher power of r ,

$$\frac{Pr}{\mu_0 a_1} \left(1 + \frac{a_2 r}{a_1 R_0} \right) + \frac{\partial}{\partial r} \left(r \frac{\partial v}{\partial r} \right) = 0.$$

On integration and then apply boundary conditions,

$$\frac{\partial v}{\partial r} = -\frac{P}{\mu_0 a_1} \left(\frac{r}{2} + \frac{a_2 r^2}{3 a_1 R_0} \right).$$

Again integration,

$$v = -\frac{P}{\mu_0 a_1} \left(\frac{r^2}{4} + \frac{a_2 r^3}{9 a_1 R_0} \right) + A(z) \quad (10)$$

using boundary conditions $v = 0$, at $r = R$

$$A(z) = \frac{P}{\mu_0 a_1} \left(\frac{R^2}{4} + \frac{a_2 R^3}{9 a_1 R_0} \right)$$

then equation (10) becomes,

$$v = \frac{P}{\mu_0 a_1} \left[\frac{1}{4} (R^2 - r^2) + \frac{a_2}{9 a_1 R_0} (R^3 - r^3) \right]. \quad (11)$$

This gives the velocity profile of stenosed artery.

2.4 Volumetric Flow Rate

Kapur and Pokharel et al. [12, 22] has mentioned the formula

$$Q = \int_0^R 2\pi r v dr$$

with the help of equation (11) ,

$$Q = 2\pi \int_0^R r \frac{P}{\mu_0 a_1} \left[\frac{1}{4} (R^2 - r^2) + \frac{a_2}{9 a_1 R_0} (R^3 - r^3) \right]$$

after integration,

$$Q = \frac{\pi P R^4}{\mu_0 a_1} \left(\frac{1}{8} + \frac{a_2 R}{15 a_1 R_0} \right) \quad (12)$$

from equation (9)

$$Q = \frac{\pi P R^4}{\mu_0 a_1} \left[\frac{1}{8} + \frac{a_2}{15 a_1} \left(1 - \frac{\delta}{2 R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) \right) \right]. \quad (13)$$

Which is the volumetric flow rate of stenosed artery.

2.5 Pressure Drop Ratio

From equation (12)

$$P = \frac{\mu_0 a_1 Q}{\pi R^4 \left(\frac{1}{8} + \frac{a_2 R}{15 a_1 R_0} \right)}$$

$$= \frac{8 \mu_0 a_1 Q}{\pi R^4} \left(1 + \frac{8 a_2 R}{15 a_1 R_0} \right)^{-1}$$

using binomial expansion and neglect the higher power of $\frac{R}{R_0}$,

$$P = \frac{8 \mu_0 a_1 Q}{\pi R^4} \left(1 - \frac{8 a_2 R}{15 a_1 R_0} \right) \quad (14)$$

from equations (9) and (14)

$$P = \frac{8 \mu_0 a_1 Q}{\pi R_0^4 \left(1 - \frac{\delta}{2 R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) \right)^4} \left[1 - \frac{8 a_2}{15 a_1} \left(1 - \frac{\delta}{2 R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) \right) \right]$$

using binomial expansion, moreover neglect higher power of δ then it becomes,

$$P = \frac{8 \mu_0 a_1 Q}{\pi R_0^4} \left[1 + \frac{2 \delta}{R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) - \frac{8 a_2}{15 a_1} \left(1 + \frac{\delta}{R_0} - \frac{3 \delta^2}{2 R_0^2} + \left(\frac{\delta}{R_0} - \frac{2 \delta^2}{R_0^2} \right) \cos \frac{\pi z}{z_0} - \frac{\delta^2}{2 R_0^2} \cos \frac{2 \pi z}{z_0} \right) \right]. \quad (15)$$

Pressure drop on the stenosed region is

$$\begin{aligned} & Z \ z_0 \\ \Delta P &= \int P dz \\ & - z_0 \end{aligned}$$

from the equation (15) and then integration, we have

$$\Delta P = \frac{16 \mu_0 a_1 Q z_0}{\pi R_0^4} \left(1 + \frac{\delta}{R_0} + \frac{3 \delta^2}{2 R_0^2} - \frac{8 a_2}{15 a_1} \right) \quad (16)$$

if there is no stenosis i.e., $\delta = 0$ then equation (16) becomes

$$(\Delta P)_p = \frac{16 \mu_0 a_1 Q z_0}{\pi R_0^4} \left(1 - \frac{8 a_2}{15 a_1} \right). \quad (17)$$

Ratio of pressure drop is

$$\frac{\Delta P}{(\Delta P)_p} = \frac{\left(1 - \frac{8 a_2}{15 a_1} + \frac{\delta}{R_0} + \frac{3 \delta^2}{2 R_0^2} \right)}{\left(1 - \frac{8 a_2}{15 a_1} \right)} \quad (18)$$

2.6 Ratio of Shear Stress

Kapur and Pokharel et al. [12, 22] have mentioned the formula

$$\tau = \frac{PR}{2}$$

From the equation (14) ,

$$\tau = \frac{4\mu_0 a_1 Q}{\pi R^3} \left(1 - \frac{8a_2 R}{15a_1 R_0} \right)$$

with the help of equation (9), used binomial expansion, moreover neglect the higher power of δ , we get

$$\tau = \frac{4\mu_0 a_1 Q}{\pi R_0^3} \left(1 + \frac{3\delta}{2R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) \right) \left[1 - \frac{8a_2}{15a_1} \left(1 - \frac{\delta}{2R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) \right) \right] \quad (19)$$

if there is no stenosis i.e., $\delta = 0$, then equation (19) becomes,

$$\tau_p = \frac{4\mu_0 a_1 Q}{\pi R_0^3} \left(1 - \frac{8a_2}{15a_1} \right) \quad (20)$$

the ratio of shear stress

$$\frac{\tau}{\tau_p} = \frac{\left(1 + \frac{3\delta}{2R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) \right) \left[1 - \frac{8a_2}{15a_1} \left(1 - \frac{\delta}{2R_0} \left(1 + \cos \frac{\pi z}{z_0} \right) \right) \right]}{\left(1 - \frac{8a_2}{15a_1} \right)} \quad (21)$$

3 RESULTS AND DISCUSSION

The use of computational techniques makes it easier to analyze blood flow parameters in stenosed arteries thoroughly and provides insightful information about the physiological effects and possible treatment options.

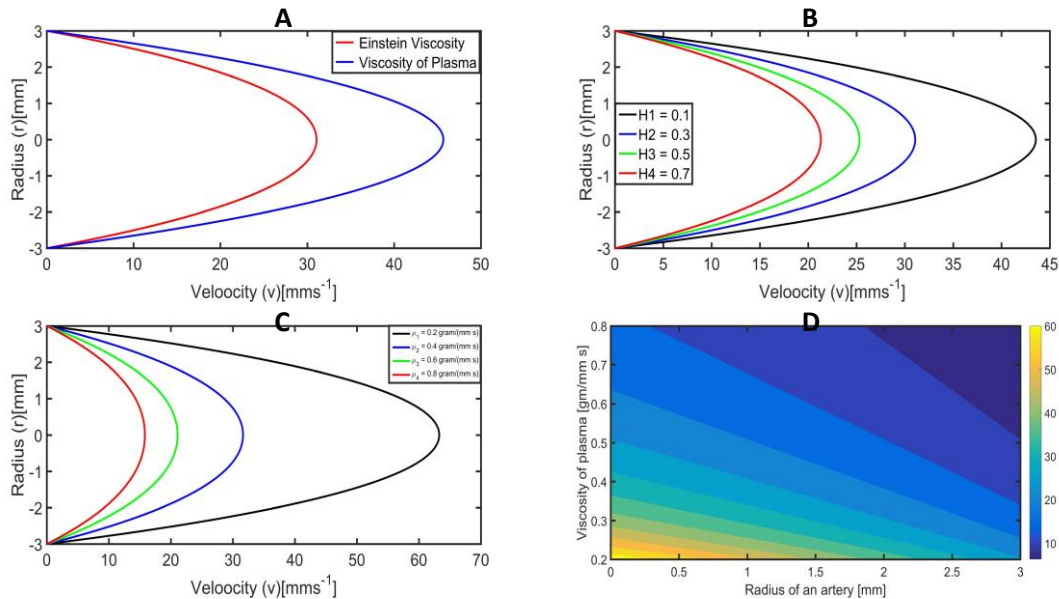


Figure 2: Velocity variations with radial distance for A: comparison viscosity, B: various hematocrit, C: various viscosity, D: various viscosity and radius.

3.1 Blood flow velocity profile across a stenotic artery

Figure 2A, describes the relation between velocity and radius for viscosity of plasma and Einstein viscosity. At the beginning, when $r = 0$ the velocities are 31.04 mm s^{-1} and 45.66 mm s^{-1} for Einstein and plasma viscosity respectively. When $r = 1 \text{ mm}$ the velocities are 27.92 mm s^{-1} and 40.50 mm s^{-1} for Einstein and plasma viscosity respectively. Similarly the radius of an artery is 2 mm , the velocities are 17.43 mm s^{-1} and 25.27 mm s^{-1} for the Einstein and plasma viscosity respectively. It is also observed that velocities are zero at the inner wall of an artery. From the above, for each value of viscosity of plasma, and Einstein viscosity, velocity is maximum at the center and decreases gradually towards the wall. This simulation result shows that the huge effect of Einstein viscosity on velocity is seen in this figure. The conclusion from this analysis is that the Einstein viscosity effects more than the viscosity of plasma on blood flow velocity.

Figure 2B, illustrates the effect of hematocrit on velocity profile while assuming the pressure drop constant. We have assumed pressure 100 mm of Hg and radius of the artery 3 mm . When the hematocrit increases from 0.1 to 0.7 , the velocity decreases from 43.55 mm s^{-1} to 21.31 mm s^{-1} which is about 22.24 mm s^{-1} at $r = 0$. When hematocrit increases from 0.1 to 0.7 , the velocity decreases from 38.91 mm s^{-1} to 19.24 mm s^{-1} which is about 19.67 mm s^{-1} at $r = 1 \text{ mm}$.

Similarly when hematocrit increases from 0.1 to 0.7 , the velocity decreases from 24.67 mm s^{-1} to 12.49 mm s^{-1} which is about 12.18 mm s^{-1} at $r = 2 \text{ mm}$. Finally hematocrit increases by a small quantity then the velocity decreases rapidly at first and then slowly, which the figure makes apparent. The velocity is roughly zero close to the artery's inner

wall and progressively increases as we travel toward the center. When the hematocrit is low, velocity increases with faster rate.

Figure 2C, depicts the effect of viscosity on the velocity profile while assuming the pressure drop constant. We have assumed that radius of an artery is 3 mm. When the viscosity increases from $0.2 \text{ gram mm}^{-1} \text{ s}^{-1}$ to $0.8 \text{ gram mm}^{-1} \text{ s}^{-1}$, the velocity decreases from 63.22 mm s^{-1} to 15.80 mm s^{-1} , which is about 47.42 mm s^{-1} at $r = 0$. When viscosity increases from $0.2 \text{ gram mm}^{-1} \text{ s}^{-1}$ to $0.8 \text{ gram mm}^{-1} \text{ s}^{-1}$, the velocity decreases from 57.05 mm s^{-1} to 14.26 mm s^{-1} which is about 42.79 mm s^{-1} at $r = 1 \text{ mm}$. Similarly, when viscosity increases from 0.2 to 0.8 , the velocity decreases from 36.87 mm s^{-1} to 9.218 mm s^{-1} which is about 27.652 mm s^{-1} at $r = 2 \text{ mm}$. Finally, viscosity increases by a small quantity, then the velocity decreases rapidly at first and then slowly, as shown in the figure. The velocity is zero on the inner wall and progressively increases as we travel toward the center. When the viscosity is low, velocity increases at a faster rate.

Figure 2D, describes the distribution of velocity for different values of viscosity of plasma and radius of an artery. Viscosity μ take values $(0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8) \text{ gram mm}^{-1} \text{ s}^{-1}$. Radius of an artery has values $(0, 1, 2, 3) \text{ mm}$. The velocity v at $r = 0$ and $\mu = 0.2 \text{ gram mm}^{-1} \text{ s}^{-1}$ is

63.28 mm s^{-1} . As the radius increases the velocity decreases for the same viscosity and becomes 25.27 mm s^{-1} at $r = 3 \text{ mm}$. The velocity at $r = 0$ is 42.19 mm s^{-1} for the viscosity of plasma $0.3 \text{ gram mm}^{-1} \text{ s}^{-1}$ and becomes 16.85 mm s^{-1} at $r = 3 \text{ mm}$ for the same viscosity. The velocities at $r = 0$ are $(31.64, 25.31, 21.09, 18.08, 15.82) \text{ mm s}^{-1}$ for the viscosity of plasma $(0.4, 0.5, 0.6, 0.7, 0.8) \text{ gram mm}^{-1} \text{ s}^{-1}$ respectively. The velocities are $(12.64, 10.11, 8.424, 7.22, 6.318) \text{ mm s}^{-1}$ at $r = 3 \text{ mm}$ for the plasma viscosities $(0.4, 0.5, 0.6, 0.7, 0.8) \text{ gram mm}^{-1} \text{ s}^{-1}$ respectively. It is found that the blood velocity gradually diminishes with increasing radius of an artery and viscosity of plasma i.e the flow velocity becomes smaller and smaller as one proceeds away from the center. For equal amount of increases in viscosity of plasma and radius of an artery, the velocity in center has maximum and in the inner wall of an artery has minimum.

From these three figures discussed above, we get that Einstein viscosity is a highly influencing factor to reduce the velocity so it is appropriate to include the Einstein term for better results than viscosity of plasma. We clearly observed that the velocity decreases rapidly with increasing hematocrit percentage and viscosity. The velocity of blood highly affect the viscosity of plasma and radius of an artery.

3.2 Volumetric flow rate through a stenotic artery

Figure 3A, describes the volumetric flow rate at different positions of stenosis, and different lines are drawn to show the increasing values of hematocrit. The positions of stenosis ranges from 0

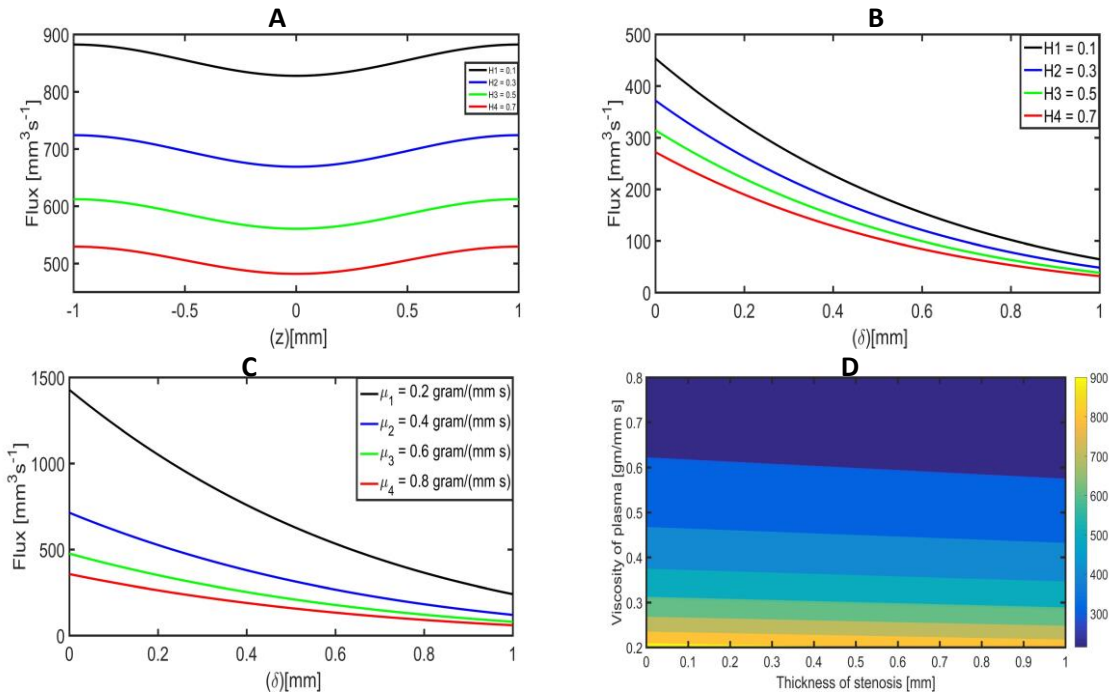


Figure 3: Volumetric flow rate A: at different position of stenosis, B,C: at different height of stenosis, D: at different height of stenosis and viscosity of plasma.

to 1 mm. When the hematocrit is 0.1, the volumetric flow rate increases from $827.50 \text{ mm}^3 \text{ s}^{-1}$ to $882.20 \text{ mm}^3 \text{ s}^{-1}$ approximately, i.e., the difference in this case is $54.70 \text{ mm}^3 \text{ s}^{-1}$. The volumetric flow rate increases from $668.80 \text{ mm}^3 \text{ s}^{-1}$ to $724 \text{ mm}^3 \text{ s}^{-1}$ approximately when hematocrit is 0.3 having difference $55.2 \text{ mm}^3 \text{ s}^{-1}$. Again, the volumetric flow rate increases from $560.40 \text{ mm}^3 \text{ s}^{-1}$ to $612 \text{ mm}^3 \text{ s}^{-1}$ approximately when the hematocrit is 0.5 having difference $51.60 \text{ mm}^3 \text{ s}^{-1}$. In the last case volumetric flow rate increases from $481.80 \text{ mm}^3 \text{ s}^{-1}$ to $529.40 \text{ mm}^3 \text{ s}^{-1}$ approximately when the hematocrit is 0.7 i.e. the difference in this case is $47.60 \text{ mm}^3 \text{ s}^{-1}$. We see that the volumetric flow rate and hematocrit have an inverse relationship. The combined effect of stenosis and hematocrit has a high risk.

Figure 3B, is used to explain the relationship between volumetric flow rate and height of stenosis for varying hematocrit. To show the hematocrit effect separately different lines are drawn for different hematocrit. The maximum height of stenosis is 1 mm and the maximum value of hematocrit is 0.7. The volumetric flow rate decreases from $453.30 \text{ mm}^3 \text{ s}^{-1}$ to $64.56 \text{ mm}^3 \text{ s}^{-1}$ approximately when, the hematocrit is 0.1 and the stenosis increases from 0 to 1 mm. Similarly the volumetric flow rate decreases from $372 \text{ mm}^3 \text{ s}^{-1}$ to $48.29 \text{ mm}^3 \text{ s}^{-1}$ for the hematocrit value is 0.3. In the third line, the volumetric flow rate decreases from 314.50 to $38.51 \text{ mm}^3 \text{ s}^{-1}$ for the hematocrit value 0.5 and $272 \text{ mm}^3 \text{ s}^{-1}$ to $32 \text{ mm}^3 \text{ s}^{-1}$ for the value of hematocrit is 0.7. When we analyze the differences of the amount decreasing in volumetric flow rate, we see that the difference is decreasing with the increase in hematocrit. When the hematocrit are 0.1, 0.3, 0.5, and 0.7, the lines indicating in volumetric flow rate almost parabolic which indicates that change due to the stenosis

is maximum with comparing hematocrit. More effect of hematocrit is seen when the height of stenosis is less than 0.6 mm, when the stenosis is more than 0.9 mm in height, all the lines indicating volumetric flow rate lie in between $64.56 \text{ mm}^3 \text{ s}^{-1}$ and $32 \text{ mm}^3 \text{ s}^{-1}$ for all hematocrit values, indicating a limiting situation. This conclude that more area is necessary to flow more volume and hematocrit is more effective in that case.

Figure 3C, describes the relation between volumetric flow rate and thickness of stenosis (δ) for different values of viscosity of plasma. Different lines are drawn to indicate the effect of different viscosity of plasma. In this case pressure 100 mm of Hg, $R = 3 \text{ mm}$, and hematocrit 0.5 are kept constant. This figure shows that the volumetric flow rate decreases from $1427 \text{ mm}^3 \text{ s}^{-1}$ to $240.90 \text{ mm}^3 \text{ s}^{-1}$ for the viscosity $0.2 \text{ gram mm}^{-1} \text{ s}^{-1}$. For the viscosity value $0.4 \text{ gram mm}^{-1} \text{ s}^{-1}$ the volumetric flow rate decreases from $713.70 \text{ mm}^3 \text{ s}^{-1}$ to $120.50 \text{ mm}^3 \text{ s}^{-1}$, and for the viscosity of plasma value $0.6 \text{ gram mm}^{-1} \text{ s}^{-1}$ the volumetric flow rate decreases from $475.80 \text{ mm}^3 \text{ s}^{-1}$ to $80.30 \text{ mm}^3 \text{ s}^{-1}$ approximately. Finally for the viscosity $0.8 \text{ gram mm}^{-1} \text{ s}^{-1}$ the volumetric flow rate decreases from $356.90 \text{ mm}^3 \text{ s}^{-1}$ to $60.23 \text{ mm}^3 \text{ s}^{-1}$. In this way the volumetric flow rate decreases with increasing viscosity. This figure also tells us that the volumetric flow rate decreases gradually as the viscosity of plasma increases uniformly and other parameters are kept constant.

Figure 3D, describes the distribution of volumetric flow rate for different values of viscosity of plasma and thickness of stenosis. Viscosity μ take values (0.2,0.3,0.4,0.5,0.6,0.7,0.8) $\text{gram mm}^{-1} \text{ s}^{-1}$. Thickness of stenosis has values (0.0,0.1,0.2,0.3,0.4,0.5,0.6,0.7,0.8,0.9,1.0) mm. The volumetric flow rate at $\delta = 0$ and $\mu = 0.2 \text{ gram mm}^{-1} \text{ s}^{-1}$ is $930 \text{ mm}^3 \text{ s}^{-1}$. As the thickness of stenosis increases the volumetric flow rate decreases for the same viscosity and becomes $859.20 \text{ mm}^3 \text{ s}^{-1}$ at $\delta = 1 \text{ mm}$. The volumetric flow rate at $\delta = 0$ is $620 \text{ mm}^3 \text{ s}^{-1}$ for the viscosity of plasma $0.3 \text{ gram mm}^{-1} \text{ s}^{-1}$ and becomes $572.80 \text{ mm}^3 \text{ s}^{-1}$ at $\delta = 1 \text{ mm}$ for the same viscosity. The volumetric flow rate at $\delta = 0$ are (465,372,310,265.5,232.5) $\text{mm}^3 \text{ s}^{-1}$ for the viscosity of plasma (0.4,0.5,0.6,0.7,0.8) $\text{gram mm}^{-1} \text{ s}^{-1}$ respectively. The volumetric flow rate are (429.60,343.70,286.40,245.50,214.80) $\text{mm}^3 \text{ s}^{-1}$ at $\delta = 1 \text{ mm}$ for the plasma viscosities (0.4,0.5,0.6,0.7,0.8) $\text{gram mm}^{-1} \text{ s}^{-1}$ respectively. It is found that the volumetric flow rate gradually diminishes with increasing thickness of stenosis and viscosity of plasma i.e the volumetric flow rate becomes smaller as one proceeds away from the center. For equal amount of increases in viscosity of plasma and height of stenosis, the volumetric flow rate at $\delta = 0$ has maximum and at $\delta = 1 \text{ mm}$ has minimum.

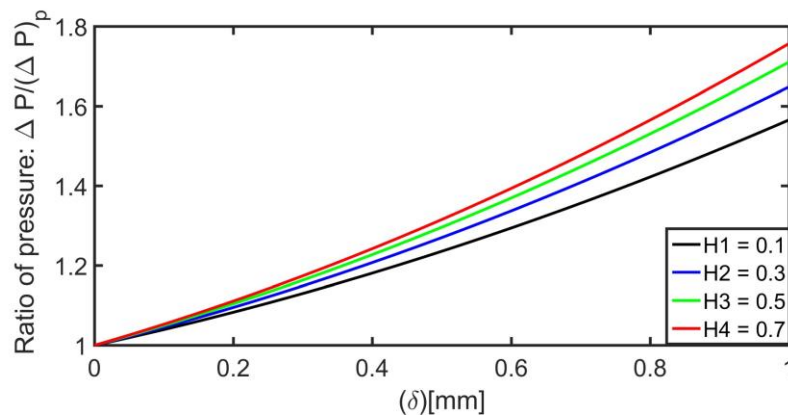


Figure 4: Relation between ratio of Pressure drop for different values of hematocrit with different height of stenosis.

3.3 Pressure gradient across the stenosis of blood flow through a stenotic artery

Figure 4 explains the change in ratio of pressure drop and δ for different values of hematocrit. The lines are looking almost linear because the interval taken here is very small they are parabolic curves. The ratio of pressure drop values is measured when δ lies between 0 to 1 mm. The figure shows that the ratio of pressure drop is maximum which is about 1.757 when the hematocrit is

0.7. The ratio of pressure drop is 1.711 approximately for the hematocrit value 0.5. Similarly it is 1.648 for the hematocrit value 0.3 and nearly 1.566 for the hematocrit is 0.1. The ratio of pressure drop are increasing continuously for increasing hematocrit in the given range. This indicates that the ratio of pressure drop is increasing gradually for increasing hematocrit and height of stenosis. This information may be useful for the measurement of hematocrit.

Table 1: Ratio of pressure drops with hematocrit for different height of stenosis
PP

PP δ P _p (mm _{PP}) P H	0.0	0.2	0.4	0.6	0.8	1.0
0.1	1.0	1.083	1.182	1.296	1.425	1.566
0.3	1.0	1.096	1.209	1.339	1.487	1.648
0.5	1.0	1.105	1.229	1.372	1.534	1.711
0.7	1.0	1.112	1.244	1.396	1.569	1.757

Table 1 radius of an artery is kept constant and hematocrit is increased gradually and it is shown that the ratio of pressure drop is increasing for increasing height of stenosis and hematocrit. The hematocrit are increased by 0.2 in each step. For this equal increment of hematocrit, corresponding ratio of pressure drops is shown in the table. Each row in the table provides informations about

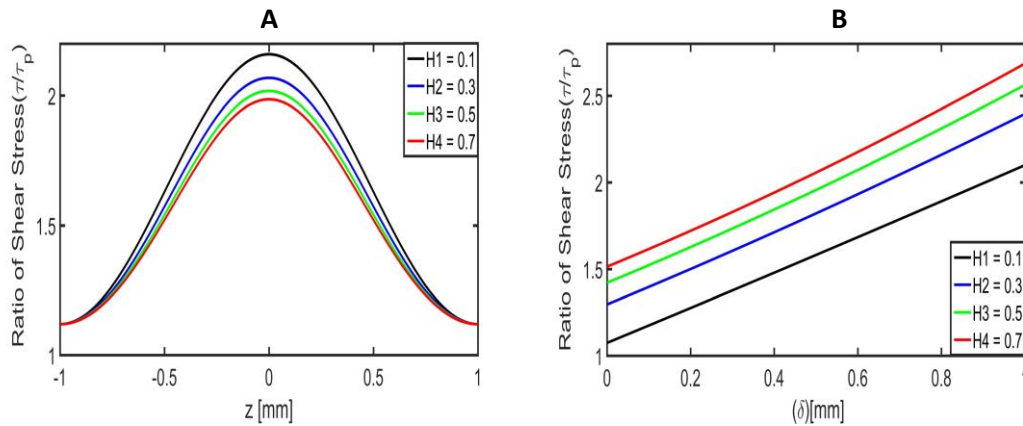


Figure 5: Relation between ratio of the shear stress A: with various position of stenosis for different values of hematocrit, B: at different height of stenosis for different values of viscosity of plasma

how the ratio of pressure drops changes at various stenosis heights concerning a reference pressure drop without stenosis, for different hematocrit levels.

3.4 Shear Stress of blood flow through a stenotic artery

Figure 5A describes the relation between ratio of shear stress at different point of stenosis and different curves are drawn to show the increasing values of hematocrit. Here -1 to 1 is the length of artery having stenosis and the height of stenosis is maximum at $z = 0$. All the lines are symmetrically increasing from -1 to 0 and decreasing after 0 because the stenosis is considered symmetric in shape. It shows that the ratio of shear stress decreases with increasing position of stenosis. Starting from the common point 1.119 the ratio of shear stress increases parabolically and becomes maximum at $z = 0$ which are 2.159 , 2.068 , 2.018 , and 1.985 for the hematocrit 0.1 , 0.3 , 0.5 , and 0.7 respectively. When the hematocrit increases shear stress ratio decreases gradually. The conclusion from this figure is that the shear stress ratio gets increasingly parabolic and declines gradually by less as the hematocrit increases.

Figure 5B depicts the ratio of shear stress at different height of stenosis and different lines are drawn to show the increasing values of hematocrit. The maximum height of stenosis is 1 mm and maximum value of hematocrit is 0.7 . All the lines are increasing from 0 to 1 because the stenosis is considered symmetric in shape. This shows that when the hematocrit is increased uniformly then ratio of shear stress is also increased uniformly. When hematocrit is 0.1 the ratio of shear stress increases and reached 2.10 at $\delta = 1$ mm, when hematocrit is 0.3 , the ratio of shear stress increasing and reached 2.395 at the maximum height $\delta = 1$ mm, as the hematocrit is 0.5 shear stress ratio is increased and attain the value 2.561 as shown in figure. Finally, when the hematocrit is 0.7 the ratio of shear stress is increased linearly and which is about 2.685 at $\delta = 1$ mm. The conclusion from this analysis is that the ratio of shear stress is increasing uniformly with uniform amount for increasing hematocrit. As hematocrit increases, the ratio of shear stress decreases, with the curve adopting a parabolic trend across various stenosis positions.

Conversely, a uniform increase in shear stress ratio accompanies a uniform rise in hematocrit levels, resulting in linear curves for different stenosis heights.

4 CONCLUSION

Atherosclerotic plaque accumulation, alongside aberrant tissue development leading to stenosis, restricts blood flow, contributing to cardiovascular diseases such as ischemia and stroke, highlighting the critical role of stenosis in cardiovascular health and disease progression. The effective viscosity of blood at radial distances, also known as the Einstein coefficient of blood viscosity, quantifies the resistance to flow in blood vessels, crucial for understanding hemodynamics and vascular health. The Navier-Stokes equations in cylindrical polar form have been applied, incorporating Einstein viscosity in an axisymmetric direction. Model expressions for velocity profile, volumetric flow rate, pressure, pressure drop, and shear stress in an artery have been analytically calculated. Furthermore, the ratio of pressure drop and shear stress has been analytically determined for the stenosed part. The velocity of blood is more influenced by the effect of Einstein's viscosity compared to plasma viscosity. As hematocrit and viscosity uniformly increase, the blood velocity initially decreases rapidly and then decreases gradually. A significant reduction in volumetric flow rate is observed at the maximum height of stenosis with the uniform increase in hematocrit, indicating the heightened risk of stenosis effects after incorporating effective viscosity. Volumetric flow rate decreases approximately linearly with uniform increases in plasma viscosity and hematocrit. The ratio of pressure drop and shear stress increases uniformly with the uniform increase of hematocrit, showing a linear relationship with stenosis height. The shear stress ratio exhibits a quadratic relation with stenosis positions. The effective viscosity of blood in the radial direction plays a crucial role in hemodynamics research, significantly influencing blood flow parameters and heightening the risk of total occlusion due to stenosis formation. Blood flow biomechanical modeling holds transformative potential for clinical procedures, surgical outcomes, vascular health understanding, and innovation across bioengineering and medical research, promising enhanced patient outcomes and healthcare advancement.

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