



Research article

A theoretical model for diffusion through stenosis

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ABSTRACT

Stenosis is caused by an abnormal growth in the artery's lumen. This undesirable growth can change the hemodynamic characteristics of the blood flow which could be injurious to normal health. Theoretical results obtained for specific geometrics are given for the velocity distribution, pressure, wall shearing stress, and other different phenomena. Flow resistance has been shown that the wall shear decreases with decreasing peripheral layer viscosity, but these properties increase with increasing stenosis size. A two-fluid blood model with a core of micro-polar fluid and a periphery of Newtonian blood has been researched in the presence of moderate stenosis. In terms of modified Bessels functions of zero and first order, analytical equations for flow resistance, wall shear stress, and diffusion via stenosis have been found. Therefore, understanding and preventing arterial illnesses need a thorough grasp of the specific flow characteristics of a channel with restriction. The results for wall shearing stress resistance to flow and concentration profiles have been obtained and discussed with the help of graphically.

1. Introduction

Stenosis is caused by an abnormal growth in the artery's lumen which is developed due to the formation of intravascular plaques or the impingement of ligaments or spurs on the vessel wall. As the disease progresses, it affects severely the coronary flow rate and perfusion. The pressure, shear, and other flow parameters, among others, are significant in an arterial system. These are linked to an increase in flow resistance and the presence of a low-pressure area that creates a suction effect, the potential for red and endothelial cell damage as a result of a high shear region, and the potential for a change from laminar to turbulent flow inside the blood vessel that results in high-intensity shear zones that are unfavourable to blood flow and the arterial wall.

Lee and Fung [1] described a numerical result for flows in tubes with local dumbbell-shaped constriction. Kaur [2] solved the problem of heat transfer by one dimensional steady state conditions. Forrester and Young [3] employed approximation techniques to find the flow solution in a tube having a constriction in the shape of a cosine curve. Several works have studied the Viewing the blood as a Newtonian or non-Newtonian fluid, one may determine the blood's flow characteristics in an artery with minor stenosis [Caro et al. [4], Shukla et al. [6], Forrester and Young [5], Rodbard [7], Fox and Hugh [8], May et al. [9]]. Recently, Awasthi and Kaur [10]

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explained method of finite difference is adopted to come up with the model signifying molecular diffusion and facilitated diffusion are responsible for the movement of organisms. Shukla et al. [11,12] examined how peripheral layer affected the blood's ability to flow through an artery with only a mild stenosis. In these investigations, two Newtonian fluids with differing viscosities are used to depict the fluid in the core region and the periphery layer, and or both the fluids are represented by Law fluids of different consistencies m_1 and m_2 completely independent of each other. Awasthi et al. [13,29] solved the problem of displacement components and stress components at the interface of two medium. Awasthi et al. [30] solved the bone crack problem inspired pair of Griffith crack opened by forces at crack faces. Recently, Tandon et al. [14–16] investigated the Micro-structural and blood flow through an artery with mild stenosis is affected by peripheral layer viscosity. This study examines the properties of blood flow through a mildly stenotic artery have been investigated by modelling the blood as a micro-polar fluid in the core enclosed by a peripheral layer having the same viscosity as that of suspension of the whole blood's medium. The effects of percent stenosis, micro-structure, and peripheral layer viscosity have been discussed. It may be observed that the work reported above introduces a constant width of the peripheral layer in almost every paper. The width depending on the stenosis's size and shape under consideration for a more realistic model. Numerous researchers have studied the continuous flow of blood via a stenosed tube and have modelled blood as a Newtonian fluid [Deshpande et al. [17], Macdonald [18], Shukla, et al. [6], Young [5]].

In microcirculation, where the peripheral layer thickness and viscosity effects predominate the flow characteristics, the consequences of stenosis are significantly more significant. In their investigations, assumptions of rigid wall symmetric constriction seem to be the reason because the changes induced by the stenosis predominate in comparison to distensibility and taperness of the walls and the pulsatile nature of the blood flow [Young [19]].

Fung [20], Burn [21] endeavored to recognize the dispersion pathways of water-soluble low molecular weight substances such as ions, sugars, and amino acids. When combined with the information from electron microscopy, it would seem that the endothelial cells are rather impervious to these chemicals and that the inter cellular spaces are the main diffusion channels for these water-soluble compounds. Now till date, the emphasis has been laid down on fluid dynamical aspects and the only discussion of mass transfer was undertaken concerning the transfer between red cells and the surrounding plasma, only peripheral comments have been made regarding the mass transfer from blood to the tissue [Fletcher [22]].

Different substances are exchanged between plasma and the surrounding tissue as blood passes through capillaries; some of these substances, like glucose, package, and albumin, are present under normal physiological circumstances, whereas others are artificially added to the blood or tissue as indicators in an experimental programme. In both healthy and diseased conditions, it is crucial to determine these chemicals' intra- and extra-vascular concentrations [Crone and Lassen [23]]. Analysis of entropy generation shows that the concentration difference parameter maximizes the entropy and minimizes the dimensionless Bejan number [28].

In this article, an attempt has therefore, been made to determine the concentration profiles and related physiological diffusion for healthy and unhealthy systems connected to stenosis brought on by localised lipid deposition. The outcome of the analysis may prove to be more useful in the identification and location of such diseases. An iteration scheme based on picards type iteration method has been developed which yields approximate results but this contributes to many complexities such as possible effect of micro-structural and blood flow is impacted by peripheral layer viscosity, and diffusion through a tube with mild stenosis in pathological states including tapering and inertial effects which are difficult to handle with other techniques. The results for flow are impeded by wall shearing stress and concentration profiles have been obtained and discussed.

2. Formulation of the problem

The shape of stenosis in a cylindrical polar coordinate system has been developed in Fig. 1 and additionally, it was considered that stenosis developed in the artery wall in an axially symmetric way and that its development was influenced by the axial distance Z and thickness of its growth δ_s of the wall. The radius of the wall in the affected region is given in equation (1)

$$\frac{R_2}{R_o} = 1 - \frac{\delta_s}{2R_o} \left[1 + \cos \frac{2\pi}{L_o} \left(Z - d - \frac{L_o}{2} \right) \right], \quad d \leq Z \leq L_o + d \tag{1}$$

= 1 elsewhere

Where δ_s denotes the maximum height of the stenosis, R_o , L_o , d is the radius of the artery with stenosis length of the stenosis and its location respectively.

The governing equation of flow and diffusion for micro-polar fluids (suspension) in the core region $0 \leq r \leq R_1(Z)$ and peripheral layer $R_1(Z) \leq r \leq R_o(Z)$ may be written in this format.

$$(\mu + K) \frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial V_1}{\partial r} \right) + \frac{K}{r} \frac{\partial}{\partial r} (r\omega) = \frac{dp}{dz}, \quad 0 \leq r \leq R_1(Z) \tag{2}$$

$$(\beta + \gamma) \frac{\partial}{\partial r} \left[\frac{1}{r} \frac{\partial}{\partial r} (r\omega) \right] - K \frac{\partial V_1}{\partial r} - 2K\omega = 0 \tag{3}$$

$$\frac{\mu}{r} \frac{\partial}{\partial r} \left(r \frac{\partial V_2}{\partial r} \right) = \frac{dp}{dz}, \quad R_1(Z) \leq r \leq R_2(Z) \tag{4}$$

$$\frac{\partial C_1}{\partial t} + V_1 \frac{\partial C_1}{\partial z} = D_1 \left(\frac{\partial^2 C_1}{\partial r^2} + \frac{1}{r} \frac{\partial C_1}{\partial r} \right) + m_1, \quad 0 \leq r \leq R_1 \tag{5}$$

$$\frac{\partial C_2}{\partial t} + V_2 \frac{\partial C_2}{\partial z} = D_2 \left(\frac{\partial^2 C_2}{\partial r^2} + \frac{1}{r} \frac{\partial C_2}{\partial r} \right) + m_2, \quad R_1 \leq r \leq R_2 \tag{6}$$

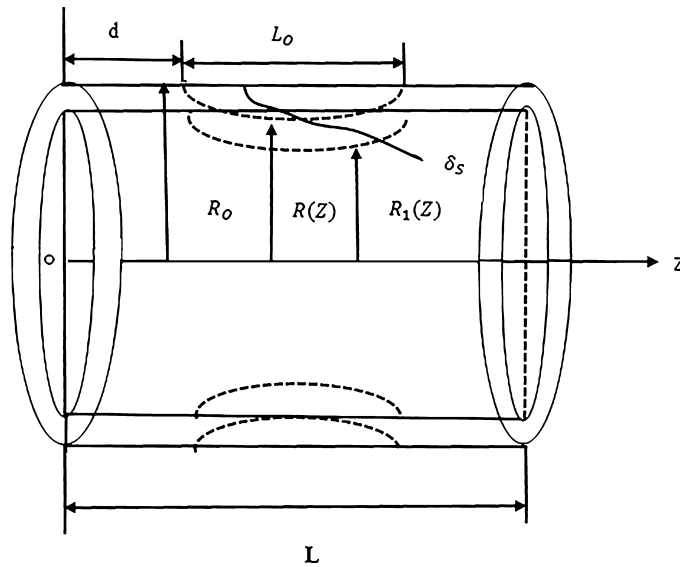


Fig. 1. The geometry of the artery with stenosis.

Where the (V_1, ω) denotes the local particle rotation's suspension speed and velocity $0 \leq r \leq R_1$ and V_2 is the velocity of the fluid in the region $R_1 \leq r \leq R_2$, K is the relative rotational viscosity, μ, β, γ are viscosities and the gradients of particle angular velocity, and $\frac{dp}{dz}$ is constant pressure gradient and r is the radial coordinate and C_1, m_1, D_1, V_1 are the solute concentration, rate of production or degeneration of cells, diffusion co-efficient of under-solved cells and velocity in the region $0 \leq r \leq R_1$ respectively and C_2, m_2, D_2, V_2 are the solute concentration, rate of production or degeneration of cells, diffusion co-efficient of under solved cells and velocity in the region $R_1 \leq r \leq R_2$.

2.1. Boundary condition

$$\begin{aligned}
 &w = 0, \text{ and } V_1 \text{ is finite at } r = 0 \\
 &V_1 = V_2, \quad \tau_1 = \tau_2, \quad \frac{\partial V_1}{\partial r} = -(2+a)w \quad \text{at } r = R_1 \\
 &V_2 = 0, \quad \text{at } r = R_2
 \end{aligned} \tag{7}$$

$$\begin{aligned}
 &C_1 = C_A, \quad \text{at } Z = 0 \\
 &C_2 = C_A, \quad \text{at } Z = 0 \\
 &\frac{\partial C_1}{\partial r} = 0 \quad \text{at } r = 0
 \end{aligned} \tag{8}$$

$$\begin{aligned}
 &-D_1 \left(\frac{\partial C_1}{\partial r} \right) = -D_2 \left(\frac{\partial C_2}{\partial r} \right) \quad \text{at } r = R_1 \\
 &C_1 = C_2, \quad \text{at } r = R_1 \\
 &-D_2 \left(\frac{\partial C_2}{\partial r} \right) = N \left(C_2 - C_0 \right) \quad \text{at } r = R_2
 \end{aligned} \tag{9}$$

Where s is the boundary condition parameter ranging over the interval $0 \leq s \leq \infty$, τ_1 & τ_2 are the shear stress in the two regions described above. N is the retention parameter. C_0 is the same reference concentration of solute.

2.2. Solutions to the problem

The solutions of the equation (2) to (4) with boundary condition (7) are

$$V_1 = \frac{-dp}{2(2\mu + K)} \left[R_2^2 - r^2 + \frac{K}{2\mu} (R_2^2 - R_1^2) \right] + \left[\frac{R_1 \bar{S} K (I_0(r) - I_0(R_1))}{\lambda (\mu + K) I_1(R_1)} \right] \tag{10}$$

$$V_2 = \frac{-dp}{4\mu} (R_2^2 - r^2) \tag{11}$$

Where

$$\lambda^2 = \frac{K(2\mu + K)}{(\beta + \gamma)(\mu + K)}, \quad \bar{S} = \left[\frac{1 + 2\mu}{(S\mu + SK)} \right]^{-1} \tag{12}$$

And I_0 & I_1 consists of modified Bessel functions of order V . Since modified Bessel functions can be approximately calculated for small values as

$$I_0 \cong 1 + \frac{x^2}{4}$$

$$I_1 \cong \frac{x}{2} + \frac{x^3}{16}$$

The solutions of the equation (5) and (6) with boundary condition (8) are

$$C_1(r) = C_1(R_1) + \frac{m_1}{4D_1} (R^2 - r^2) + \frac{\frac{dp}{dz}}{2(2\mu + K)D_1} \left[\frac{R_2^2}{4} (R_1^2 - r^2) - \frac{1}{16} (R_1^4 - r^4) + \frac{K}{8\mu} (R_2^2 - R_1^2) (R_1^2 - r^2) \right] + \frac{\partial C_1}{\partial z} \left[\frac{R_1 \bar{S} K}{D_1 (\mu + K) I_1(R_1)} \left(I_0(R_1) - I_0(r) \right) \right] - \frac{1}{D_1} \frac{(R_1^2 - r^2)}{4(\mu + K)} \frac{\partial C_1}{\partial z} \tag{13}$$

Where

$$C_1(R_1) = C_2(R_2) + \frac{m_2}{D_2} \left[\frac{(R_2^2 - R_1^2)}{2} - R_1^2 \left(\log \frac{R_2}{R_1} \right) + \frac{dp}{dz} \frac{R_2^2 \alpha}{8\mu D_2} \left[\frac{1}{2} \left[\log(R_2 - \frac{1}{2}) - R_1^2 \log \left(R_1 - \frac{1}{2} \right) - \frac{1}{4} (R_2^2 - R_1^2) - R_1^2 \left(\log R_1 - \frac{1}{2} \right) - \frac{1}{4} (R_2^2 - R_1^2) - R_1^2 \left(\log R_1 - \frac{1}{2} \right) \log \left(\frac{R_2}{R_1} \right) \right] \right] - \frac{dp}{dz} \frac{\alpha}{16\mu D_2} \left[\frac{1}{4} \left[R_2^4 \log \left(R_2 - \frac{1}{4} \right) - R_1^4 \left(\log R_1 - \frac{1}{4} \right) \right] - \frac{1}{16} (R_2^2 - r^4) - R_1^4 \left(\log \left(R_1 - \frac{1}{4} \right) \right) \times \log \frac{R_2}{R_1} \right] + \left(\frac{dp}{dz} \right) \beta \left[\frac{R_2^2}{2} \left(\frac{1}{2} (R_2^2 - R_1^2) - R_1^2 \left(\log \frac{R_2}{R_1} \right) - \frac{1}{4} (R_2^4 - R_1^4) - R_1^4 \left(\log \frac{R_2}{R_1} \right) \right) - G \log \frac{R_2}{R_1} \right] \tag{14}$$

$$G = \frac{1}{2} \left(\frac{m_1}{D_1} - \frac{m_2}{D_2} \right) R_1 \frac{dR_1}{dz} + \frac{(m_2 - m_1)}{2D_2} \left[2R_1 \frac{dR_1}{dz} \log \frac{R_1}{R_2} + R_1 \frac{dR_1}{dz} - \frac{R_1^2}{R_2} \frac{dR_2}{dz} \right] + \frac{1}{2N} \left[m_2 \frac{dR_2}{dz} - 2(m_2 - m_1) \frac{R_1}{R_2} \frac{dR_1}{dz} + (m_2 - m_1) \frac{R_1^2}{R_2^2} \frac{dR_2}{dz} \right] + \frac{m_2 R_2}{2D_2} \frac{dR_2}{dz} \tag{15}$$

$$C_2(R_2) = -\frac{m_2}{2N} \frac{(R_2^2 - R_1^2)}{R_2} - \frac{1}{8\mu} \frac{dp}{dz} \frac{R_2^2 \alpha}{N} \left[R_2^2 \left(\log R_2 - \frac{1}{2} \right) - \frac{R_1^2}{R_2} \left(\log R_1 - \frac{1}{2} \right) \right] + \frac{\frac{dp}{dz}}{16\mu N} - \left[R_2^3 \left(\log R_2 - \frac{1}{4} \right) - \frac{R_1^4}{R_2} \left(\log R_1 - \frac{1}{4} \right) \right] - \frac{dp}{dz} \frac{\beta}{4\mu N} \left[\frac{R_2^2}{2} \frac{(R_2^2 - R_1^2)}{R_2} - \frac{1}{4} \frac{(R_2^2 - R_1^4)}{R_2} \right] + \frac{G}{R_2} + C_0 \tag{16}$$

$$\alpha = \frac{(m_2 - m_1)}{2D_2} \left(2R_1 \frac{dR_1}{dz} \right) \tag{17}$$

$$\beta = \frac{(m_2 - m_1)}{2D_2} \left[-2R_1 \frac{dR_1}{dz} \log R_2 + \frac{R_1^2}{R_2} \frac{dR_2}{dz} \right] + \frac{1}{2N} \left[m_2 \frac{dR_2}{dz} - 2(m_2 - m_1) \frac{R_1}{R_2} \frac{dR_1}{dz} + (m_2 - m_1) \frac{R_1^2}{R_2^2} \frac{dR_2}{dz} \right] + \frac{m_2 R_2}{2D_2} \frac{dR_2}{dz} + \frac{1}{D_2} \left[-\frac{m_1}{2} R_1^2 - \frac{\frac{dp}{dz} \left(\frac{\partial C_1}{\partial z} \right)}{2(2\mu + K)} \left(\frac{R_2^2 R_1^2}{2} - \frac{R_1^4}{4} + \frac{K}{4\mu} (R_2^2 - R_1^2) R_1^2 \right) + \frac{1}{D} \frac{\partial C_1}{\partial z} \frac{R_1^2 \bar{S} K}{\mu + K} - \frac{1}{D_1} \frac{\partial C_1}{\partial z} \frac{R_1^2}{2(\mu + K)} \right] \tag{18}$$

After using the above equations (14) to (18), we get

$$C_2(r) = C_2(R_2) + \frac{m_2}{2D_2} \left[\frac{(R_2^2 - r^2)}{2} - R_1^2 (\log R_2 - \log r) \right] + \frac{dp}{dz} \frac{R_2^2 \alpha}{8\mu D_2} \left[\frac{1}{2} \left(R_2^2 \left(\log R_2 - \frac{1}{2} \right) - \frac{R_1^2}{R_2} \left(\log R_1 - \frac{1}{2} \right) \right) \right]$$

Table 1

Variation of resistance to flow (RF) and wall shearing stress (τ_w) with boundary condition parameter (\bar{S}) at 60% stenosis.

\bar{S}	0	0.237	0.727
$RF \times 10 \text{ gm/cm}^4 \text{ sec}$	3.6089	3.6110	3.6161
$\tau_w \text{ gm/cm sec}^{-2}$	3.0096	3.0099	3.0150

Table 2

Variation of wall shearing stress τ_w with hematocrit at 60% stenosis.

%H	40	20	10
$\tau_w \text{ gm/cm sec}^{-2}$	6.9378	6.9357	6.9335

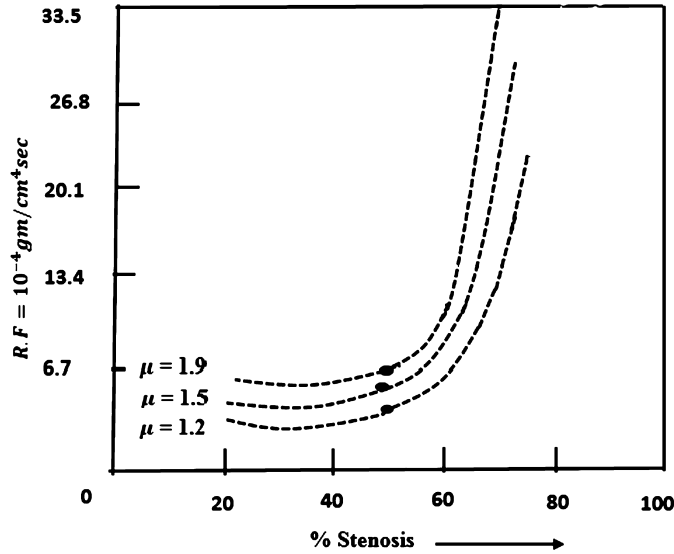


Fig. 2. Variations of resistance to flow with stenosis percentage for different values of peripheral layer viscosity and fixed values of $s = 0.231, H = 40\%, \frac{L_0}{L} = 0.02$.

$$\begin{aligned}
 & r^2 \left(\log r - \frac{1}{2} \right) - \frac{1}{4} \left(R_2^2 - r^2 \right) - R_1^2 \left(\log R_1 - \frac{1}{2} \right) \{ \log R_2 - \log r \} \left[- \frac{\frac{dp}{dz} \alpha}{16 \mu D_2} \left[\frac{1}{4} \left(R_2^4 \left(\log R_2 - \frac{1}{4} \right) \right. \right. \right. \\
 & \left. \left. \left. - r^4 \left(\log r - \frac{1}{4} \right) \right) - \frac{1}{16} \left(R_2^2 - r^4 \right) - R_1^4 \left(\log R_1 - \frac{1}{4} \right) \{ \log R_2 - \log r \} \right] + \frac{\left(\frac{dp}{dz} \right) \beta}{4 \mu D_2} \left[\frac{R_2^2}{2} \left(\frac{1}{2} \left(R_2^2 - r^2 \right) \right. \right. \right. \\
 & \left. \left. \left. - R_1^2 \left(\log R_2 - \log r \right) \right) - \frac{1}{4} \left(\frac{1}{4} \left(R_2^4 - r^4 \right) - R_1^4 \left(\log R_2 - \log r \right) \right) \right] - G \left(\log R_2 - \log r \right) \quad (19)
 \end{aligned}$$

3. Results and discussion

In this article, a two-fluid model of blood with a micro-polar fluid core and a Newtonian fluid peripheral layer has been investigated in the presence of mild stenosis. Analysis of the wall shear stress and flow resistance at the highest stenosis height and diffusion through stenosis based on modified Bessels functions of zero and first order, results have been obtained. It is discussed that the analysis of this article is a generalization of the [Shukla [11], Tandon et al. [14]], two-fluid model, and [Young [19,25]], Newtonian fluid model. Since earlier studies have not considered the diffusion through the stenosis. Therefore, these cases are obtained as certain limiting cases of the present research. Further, it is noted that in the earlier models the blood has either been assumed to be a Newtonian fluid or a two-layered Newtonian fluid model with distinct viscosities in the core and the peripheral region. But experimental results [Gould [26] and Cokelet [24]] imply the presence of a central core of non-Newtonian fluid and a peripheral layer of Newtonian fluid (R.B.C. Suspension). Therefore it is clear that the earlier models are unrealistic. Additionally, it has been found that the velocity profiles in the core rely on the tube's diameter and particle size ratio in addition to hematocrit. Since the two-fluid models with the core of Casson fluid or power-law fluid [Shukla et al. [11,12]] do not think about how particle size affects

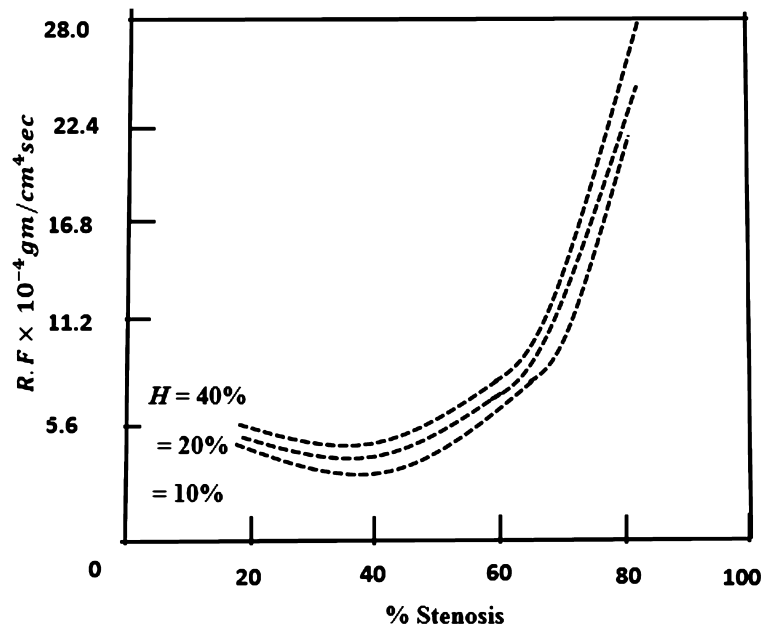


Fig. 3. Variations of resistance to flow with stenosis percentage for different values of Hematocrit and fixed values of $s = 0.231, \mu = 1.5cP, \frac{L_0}{L} = 0.02$.

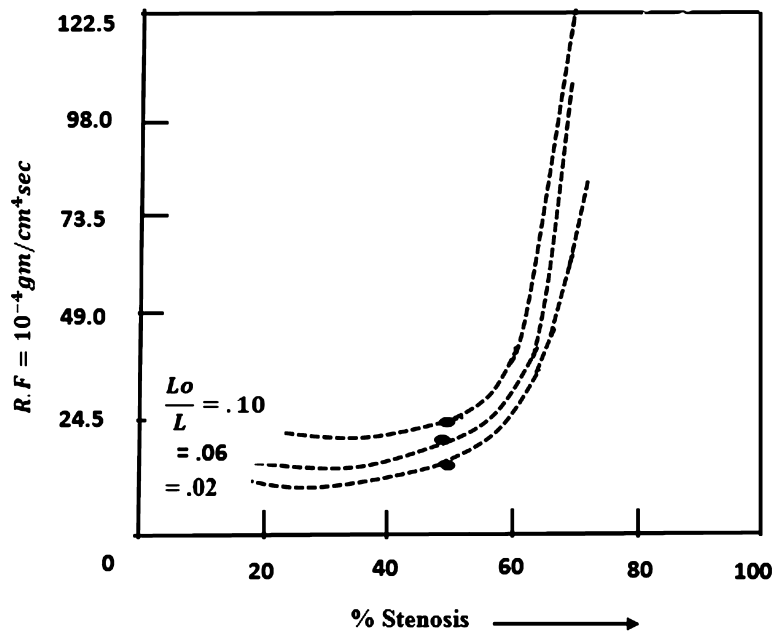


Fig. 4. Variations of resistance to flow with stenosis percentage for different values of length of the stenosis and fixed values of $s = 0.727, H = 40\%, \mu = 1.5cP$.

things. It has a few restrictions. The present model includes all the above effects with diffusion and has improved agreement with the outcomes of the experimental.

Tables 1 and 2 depict the effects of the boundary conditions parameter \bar{S} on resistance to flow and wall shearing stress. Chaturani and Mahagan have determined the values of boundary condition parameter \bar{S} by using the experimental values of the other parameters of the fluid. We have used the same values ($0 \leq \bar{S} \leq 1$). It may be noted that \bar{S} decreases as concentration (i.e. hematocrit) decreases or particle size increases. The wall shearing stress increases with \bar{S} and the resistance to flow also increases. It has been noted that the boundary condition parameter is directly proportional to apparent viscosity and therefore, we conclude that the wall shearing stress as well as resistance to flow increases with apparent viscosity of the blood. These results are also consistent with our result reported above and with the literature.

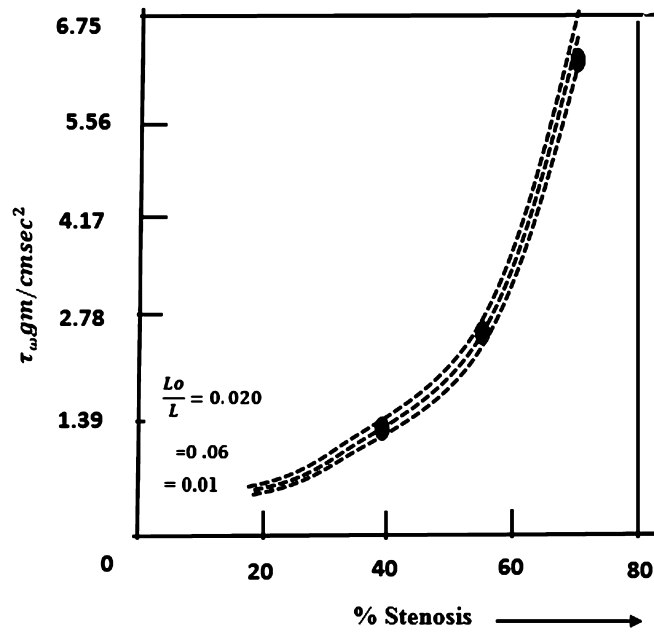


Fig. 5. Variations of wall shearing stress with stenosis and fixed value of $s = 0.727, H = 40\%, \mu = 1.5cP$.

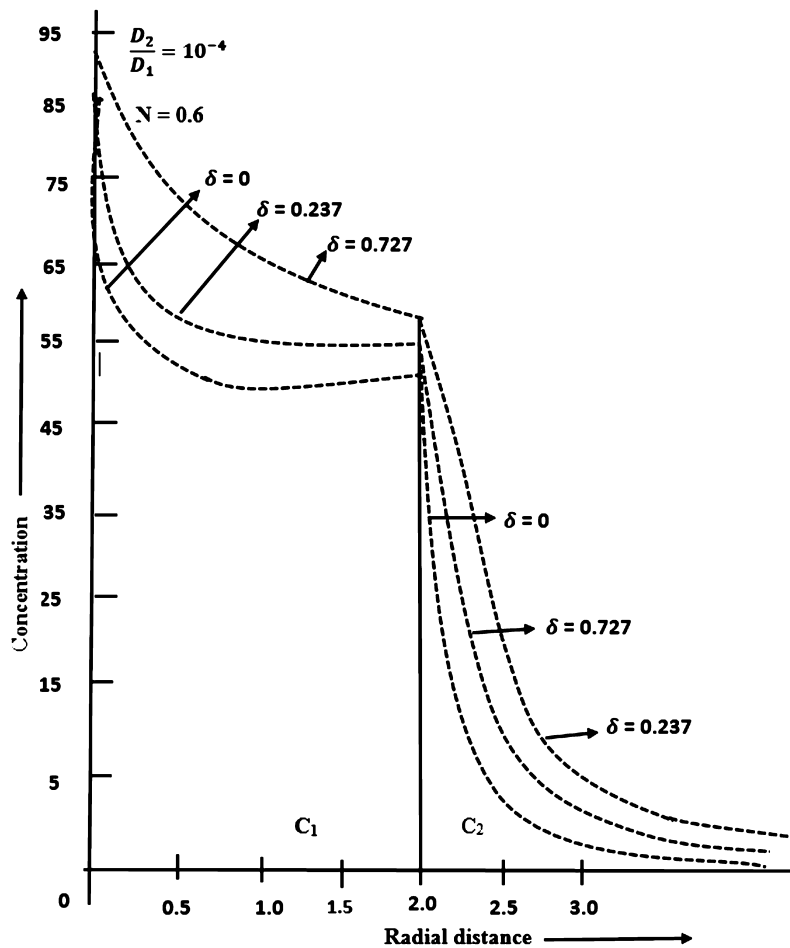


Fig. 6. Concentration profile in the capillary for different values of the parameters s .

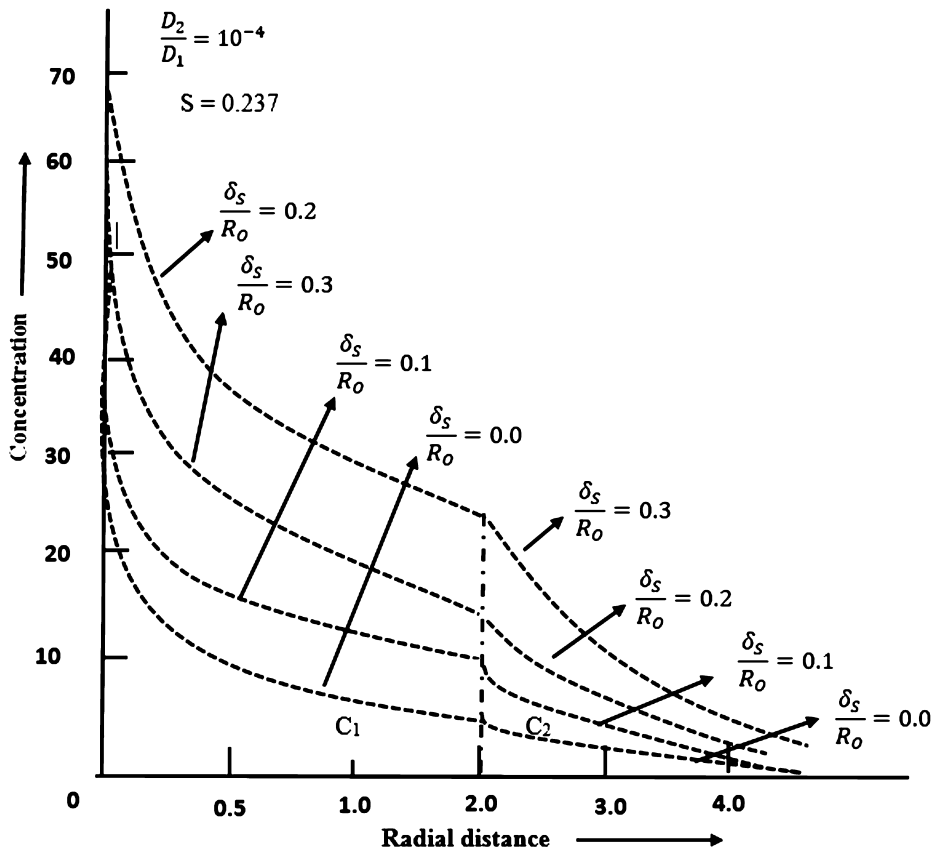


Fig. 7. Concentration profiles in the capillary for different values of $\frac{\delta_s}{R_o}$.

For various levels of peripheral layer viscosity, Fig. 2 shows the fluctuation in flow resistance with percentage stenosis. It is noticeable that the flow resistance rises gradually, reaching a maximum of 40%. Stenosis and thereafter it increases very rapidly. It also rises in proportion to the peripheral layer’s viscosity. Therefore, we may draw the conclusion that the disease affects is more prevalent in people with higher peripheral layer viscosity.

Fig. 3 shows how, at various hematocrit values, the resistance to flow varies with the percentage stenosis. As the hematocrit value rises, it is seen that the flow resistance also rises. It is clear that this is the case since the blood’s apparent viscosity consistently rises as hematocrit does.

According to Fig. 4, the resistance to flow rises as the length of the stenosis increments for a given percentage of stenosis.

Fig. 5 shows that wall shearing stress increases with percentage stenosis and decreases with the length of stenosis similar result has earlier been obtained by Chow and Soda [27]. These results further agree in respect of small constrictions when the effect of stenosis is negligible but the effects are more severe as the percentage of stenosis increases.

Fig. 6 displays the diffusion of dissolved nutrients in the capillary as well as in peripheral layer region. We observe that concentration in the peripheral layer is much less than that in the capillary region. The concentration in the capillary region is maximum near the central line and decreases towards the peripheral layer and the concentration in the peripheral layer also decreases towards the wall. We have also observed from this figure that as S increases concentration increases due to the axial migration of the cells because of narrowing the region of flow.

Fig. 7 describes the diffusion of dissolved nutrients in normal and stenosis capillary the effect of increasing stenosis is to increase the concentration in the peripheral layer as well as in the capillary region. As the stenosis progresses the concentration near the surface increases more rapidly. From this we may conclude once the stenosis is formed, it further increases more rapidly due to the deposition of more cells.

Fig. 8 shows the impacts of the maintenance boundary on fixation as well as in the area of the peripheral layer. Expanding upsides of N depict the expansion in maintenance of the solute inside the capillary region. $N = 1$ infers the total maintenance that is no solute diffuses into the tissue region also, as the maintenance boundaries decline from 1 to 2, more solute diffuses in the tissue region which in term decreases the concentration in the capillary region as well as in the peripheral layer area.

Fig. 9 depicts the variation of concentration in the capillary and peripheral layer region for different ratios of diffusivities. As the proportion diminishes, the focus in the capillary area increments as the ratio falls.

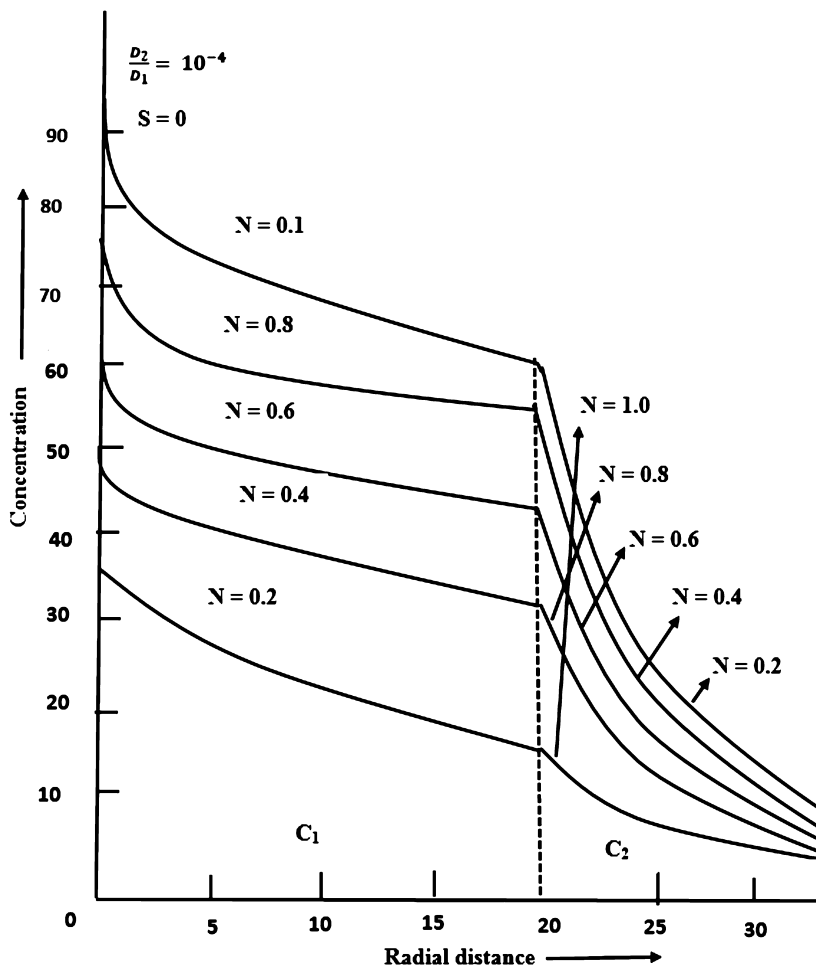


Fig. 8. Concentration profiles for different values of retention parameter.

4. Conclusion

This research focuses on analytical expressions for resistance to flow and wall shear stress at the maximum height of the stenosis and diffusion through stenosis has been obtained in terms of modified Bessel functions of zero and first order along with increased apparent velocity of the blood. These results are consistent with our development reported above and the literature review. As a result, it will undoubtedly assist researchers who are working in this field. The outcomes acquired prove helpful for doctorly uses such as enhancing effectiveness and sensitivity.

5. Significance of this research

The most serious biological reaction is the formation of stenosis, which causes several complications in cardiovascular illnesses. The exciting findings from the studies listed above, as well as the practical applications highlighted, will help medical practitioners predict blood flow behaviour in stenotic arteries. Medical practitioners may use the physical information gathered from individuals diagnosed with diabetes and a variety of other conditions to identify the medicine needed to treat them.

Ethical approval

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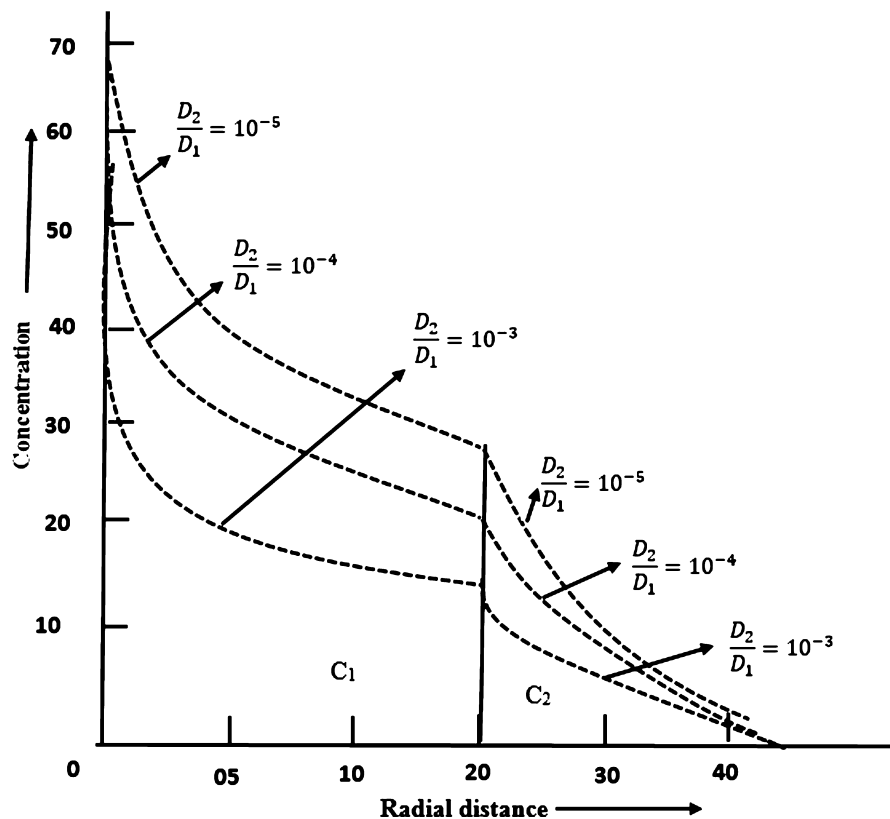


Fig. 9. Concentration profiles in the capillary for different values of $\frac{D_2}{D_1}$.

CRediT authorship contribution statement

Conceived and designed the experiments; A.K. Awasthi. **Performed the experiments;** Harpreet Kaur and Rajendra Kumar Tripathi. **Analyzed and interpreted the data;** A.K. Awasthi, Harpreet Kaur, Rajendra Kumar Tripathi, Masoumeh Khademi and Homan Emadifar. **Contributed reagents, materials, analysis tools, or data;** A.K. Awasthi, Harpreet Kaur, Rajendra Kumar Tripathi, Masoumeh Khademi and Homan Emadifar. **Manuscript is written by;** Harpreet Kaur and Homan Emadifar.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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