Chapter 7

A Theoretical Study of Blood Flow through a Multi-stenosed Artery with Slip: A Newtonian Fluid Model

7.1 Introduction

Atherosclerosis (stenosis) is a kind of arterial disease that affects the human cardiovascular system (cvs) to a great extent. It is reported that the leading cause in developed countries, may be attributed to the severe attack, emerging from cvs diseases. Literally, stenosis means a narrowing or constriction in the diameter of the bodily passage or orifice. Naturally it is a sort of vascular disease that damages our arteries by a sub-endothelial built-up of fat, cholesterol and other substances. Due to this unbecoming formation (stenosis) in the human arterial wall, the flow of blood is disturbed appreciably and the resistance to flow becomes higher than that in a normal artery region. As such, the fluid mechanical behavior of an arterial stenosis has drawn considerable attention from the researchers. Many researchers have investigated blood flow through stenosed vessels from different objectives (Young, 1968; Young and Tsai, 1973; Lee and Fung, 1970; Rodboard, 1966). The rheology of circulation is intensively discussed by Whitemore 1968. Further, several authors have proposed various mathematical models for flow through stenosed or constricted ducts (Young, 1968; Lee and Fung, 1970; Shukla et al., 1980; Chaturani and Samy, 1986; Radha Krishnamacharya and Rao, 2007). Maruthi Prasad and Krishnamacharya (2008) have considered the steady flow of non-Newtonian fluid flow through an inclined tube of non-uniform cross-section with multi-stenoses. The analysis of blood flow through a symmetrical stenosed artery has been studied by Singh et al. (2009). The flow of blood in a constricted artery with different situations is presented in the papers of Biswas and Chakrabarty (2009, 2010). Recently, steady flow of blood through a stenosed artery in non-Newtonian case, has been considered by Biswas and Laskar (2011).
Many authors (Lee and Fung, 1970; Chow and Soda, 1972; Ehrlich, 1979; MacDonald, 1979; Polard, 1981; Perkkio and Keskinen, 1983; Biswas and Chakraborty, 2009) have studied various aspects of blood flow, by considering blood to behave as a Newtonian fluid. However, human blood is regarded as a suspension of different tiny cells in continuous aqueous substance plasma (Fung, 1981; Guyton, 1970). Although, blood exhibits a non-Newtonian character at low shear rates (Merrill, 1965), at high shear rates, blood behaves like a Newtonian fluid (Tailor, 1959). Also, the existence of velocity slip at the flow boundaries has already reported by many investigators (Vand, 1948; Nubar, 1979; Burn, 1975; Bloch, 1962). Experimental observations (Bugliarello and Hayden, 1963; Bannet, 1967) have also indicated the likely presence of a slip at the blood vessel wall.

In view of the above theoretical models and experimental works, a slip condition at the stenotic wall that seems to be realistic, is employed here. In the present theoretical modeling, steady laminar fully-developed one-dimensional Newtonian fluid (blood) flow through a uniform artery with the presence of two equi-spaced symmetric stenoses at the inner arterial wall, has been investigated.

7.2 Mathematical Formulation

We model the artery as a uniform tube with radius $R_0$ and blood is modeled to behave as an incompressible Newtonian fluid with shear viscosity $\mu$. The flow is considered as steady, laminar and one-dimensional, inside an arterial segment with an axi-symmetric formation of two stenoses at wall.
In the present case, we have considered two successive stenoses separated from each other and developed at the innermost arterial wall and their unfamiliar growths have taken place in an axi-symmetric manner. Thus, the radius of the artery $R(z)$ in the multi-stenotic region (Figure 7.1) can be taken as (Young 1968; Biswas 2000).

$$R(z) = R_0 - \frac{\delta_i}{2} \left[ 1 + \cos \left( \frac{2\pi}{L_0} \left( z - id - \frac{(2i - 1)L_0}{2} \right) \right) \right], \quad \text{if } (i-1) \leq z \leq i(d + L_0),$$

$$= R_0, \quad \text{otherwise},$$

(7.2.1)

where $i=1, 2$ and $R_0$ and $R(z)$ stand for the radii in the normal and stenotic regions, $L_0$, $d$, and $\delta_i$ represent the length, location (i.e., distance between equi-spaced points) and maximum height of the stenosis where as $L$ is the length of this arterial segment.

The governing equation of blood flow through an artery in absence of inertia forces and body acceleration is given by (Biswas 2000, Schlitchting, 1979) as

$$\frac{1}{r} \frac{\partial}{\partial r} \left( -r \tau_{rr} \right) = \frac{dp}{dz},$$

(7.2.2)
where \( r \) and \( z \) denote the radial and axial coordinates, \( p \) the pressure at any point, 
\[
c = \left( -\frac{dp}{dz} \right)
\]
is the pressure gradient in the axial direction and the shear stress \( \tau_z \) of blood 
behaving as a Newtonian fluid is 
\[
\tau_z = -\mu \frac{\partial u}{\partial r}, \tag{7.2.3}
\]
where \( u = u(r) \) is the axial velocity of blood and \( \mu \) is the viscosity of blood. As a result of 
above equations (1-2), the equation governing the fluid flow can be expressed as 
\[
C + \frac{\mu}{r} \frac{\partial}{\partial r} \left( r \frac{\partial u}{\partial r} \right) = 0, 0 \leq r \leq R_0 \tag{7.2.4}
\]

### 7.3 Solutions

In integrating equation (7.2.4), boundary conditions employed are the following:

\[
u = u_s \text{ at } r = R(z), \tag{7.3.1}
\]
\[
\tau \text{ is finite at } r = 0, \tag{7.3.2}
\]

where \( u_s \) is an axial slip velocity (Chaturani and Biswas, 1984) at the constricted wall.

An integral of equation (7.2.4) can be put in the form 
\[
u(r) = B + A \ln r - \frac{Cr^2}{4\mu}, 0 \leq r \leq R_0, \tag{7.3.3}
\]

where \( A \) and \( B \) are the constants of integration.

Using conditions (7.3.1-7.3.2) in equation (7.3.3), the velocity function becomes,
\[
u(r,z) = u_s + \frac{Cr^2}{4\mu} \left( R^2(z) - r^2 \right), 0 \leq r \leq R(z), 0 \leq z \leq L, \tag{7.3.4}
\]

The volumetric flow rate \( Q \), defined by 
\[
Q = 2\pi \int_{r=0}^{R(z)} ru(r,z) dr, \tag{7.3.5}
\]
takes the following form, with the help of equation (7.3.4)
\[
Q = \pi R^2(z) \left[ u_s + \frac{C}{8\mu} R^2(z) \right]. \tag{7.3.6}
\]
From the equation (7.3.6), the pressure gradient can be expressed as
\[
\frac{dp}{dz} = \left( -\frac{8\mu}{R^4(z)} \right)\left( \frac{Q}{\pi} - u_s R^2(z) \right).
\tag{7.3.7}
\]

Integrating equation (7.3.7) between the limits
\[
p = p_i \text{ at } z = L \text{ and } \quad p = p_0 \text{ at } z = 0
\tag{7.3.8}
\]
where \(L\) is length of the tube and \(p_0\) being the pressure at \(z = d\), in the normal artery region, we obtain the pressure drop \((p_0 - p_i)\) in the form
\[
p_0 - p_i = 8\mu \int_0^L R^{-4}(z)\left[ \pi^{-1}Q - u_s R^2(z) \right]dz.
\tag{7.3.9}
\]

The resistance to flow \((\lambda)\), defined as
\[
\lambda = \frac{(p_0 - p_i)}{Q},
\tag{7.3.10}
\]
and, after using equations (7.3.6, 7.3.9), takes the following form
\[
\lambda = 8\mu \left[ \left( \frac{R}{Q} \right)^4 - \frac{Q}{Q_1} R_0^2 u_s \left( L - 2L_0 \right) 
+ \int_{z=d}^{d+L_0} \int_{z=2(d+L_0)} \left( (\pi^{-1} - Q^{-1} R^2(z) u_s) R^4(z) \right)dz \right],
\tag{7.3.11}
\]

where \(Q_1 = Q\) when \(r = R_1\), \(R(z) = R_1(z)\) and \(\delta_1 = \delta_2\).

Expression for wall shear stress, defined by
\[
\tau_{R(z)} = \left[ -\mu \frac{\partial u}{\partial r} \right]_{r=R(z) (z)},
\tag{7.3.12}
\]
becomes,
\[
\tau_{R(z)} = \frac{C}{2} R(z),
\tag{7.3.13}
\]
Apparent Viscosity $\mu_a$ can be computed from the formula

$$\mu_a = \frac{\pi CR^4(z)}{8Q} = \left[ \frac{8u_s}{CR^2(z)} + \frac{1}{\mu} \right]^{-1}, \quad (7.3.14)$$

where $Q$ has been given in equation (7.3.6).

A Second Form

Introducing the following dimensionless quantities

$$\overline{R}(z) = \frac{R(z)}{R_0}, \overline{\delta}_i = \frac{\delta}{R_0} (i = 1, 2), \overline{u}(r, z) = \frac{u(r, z)}{U_0}, \overline{U}_0 = \frac{C_0 R_0^2}{4\mu_1},$$

$$\overline{Q} = \frac{Q}{Q_0}, \overline{Q}_0 = \frac{\pi C_0 R_0^4}{8\mu_1}, \left( \frac{dp}{dz} \right)_0 = -\frac{8\mu_0 Q_0}{\pi R_0^4}, \overline{u} = \frac{u}{U_0}, \overline{C}_0 = \frac{C}{C_0}, \overline{z} = \frac{z}{L},$$

$$\overline{r} = \frac{r}{R_0}, \overline{\tau}(z) = \frac{\tau_R(z)}{\tau_0}, \overline{\mu} = \frac{\mu}{\mu_1}, \overline{\lambda} = \frac{\lambda}{\lambda_0}, \overline{\lambda}_0 = \frac{8\mu_1 L}{\pi R_0^4},$$

the expressions for flow variables in equations (7.3.4-7.3.15) becomes as follows:

**Velocity Function:**

$$\overline{u}(r, z) = \overline{u}_s + \frac{\overline{C}}{\mu} \left( \overline{R}(z)^2 - \overline{r}^2 \right), 0 \leq \overline{r} \leq \overline{R}(z), 0 \leq \overline{z} \leq 1. \quad (7.3.16)$$

**Flow Rate:**

$$\overline{Q} = \overline{R}(z)^2 \left[ 2\overline{u}_s + \frac{\overline{C}}{\mu} \overline{R}(z)^2 \right], 0 \leq \overline{z} \leq 1$$

(7.3.17)
Wall Shear Stress:
\[ \tau_{R(z)} = CR(z). \]  
(7.3.18)

Pressure Gradient:
\[ \left( \frac{dp}{dz} \right) = \left( \frac{\mu}{R(z)} \right) \left( Q - 2u_sR(z)^2 \right). \]  
(7.3.19)

Apparent Viscosity:
\[ \mu_a = \left[ \frac{2u_s}{CR(z)} + 1 \right]^{-1}. \]  
(7.3.20)

Resistance to flow:
\[ \lambda = \mu \left[ \left( 1 - \frac{2u_s}{Q} \right) \left( \frac{L - 2L_0}{L} \right) \right] + \left[ \int_{z=d}^{d+t_o} + \int_{z=2d+t_o}^{2(d+t_o)} \frac{R(z)^4}{Q} \left( 1 - \frac{2u_sR(z)^2}{Q} \right) \right] dz. \]  
(7.3.21)

7.4 Results and Discussions

Analytical expressions for flow variables (in dimensionless form), included in equations (7.3.16-7.3.21) and obtained as functions of different parameters, are computed here graphically, in order to investigate the influence of multi-stenoses in this 1-D flow. The computations have been carried out for noticing their variations with several flow parameters, on these mild constrictions. In the ongoing analysis, we have considered two same sized stenoses \( R(z) = R_1(z) \) and \( \delta_1 = \delta_2 \). In this numerical computation, stenoses with mild form 25%; moderate form 50% and severe form 75%, are used to perform a comparative analysis. The parameter values are taken as \( L_0 = d, L = 3d + 2L_0, C = 0.95, \)
1.10, 1.25; $R_0 = 20 \mu m$, $35 \mu m$, $H = 40\%$, $\bar{\mu} = 1.783$, $2.425$, $\bar{u}_s = 0.05$, $0 \leq \bar{r} \leq 1$, $\bar{d} \leq \bar{z} \leq 3\bar{d} + 2L_0$. The present study corresponds to the flow of a Newtonian fluid with the presence of a single stenosis (when $R(z) = 0 = \delta_2$) and that in a normal artery (when $R(z) = R_0 = R_i(z)$) with the usual condition of zero-slip ($\bar{u}_s = 0.00$) and slip ($\bar{u}_s > 0$) at the vessel wall. The computational results for velocity function, flow rate, wall shear stress, apparent viscosity and pressure gradient, as obtained from the present model, are presented graphically (Figures 7.2-7.11).

Further, fluid flow being unidirectional, in the present case, an employment of axial velocity slip at vessel wall, seems to be appropriate. Many researchers have already reported that the rheological and fluid dynamic properties of blood and blood flow, like velocity profiles, shear stresses, flow rate, apparent viscosity, skin friction etc., could play a significant role in the basic understanding, diagnosis and treatment of many cardiovascular, cerebrovascular, renal and arterial diseases. In the light of their report, analytical expressions of such flow variables and their variations with useful parameters, are obtained numerically and presented in figures (7.2-7.12). In equation (7.20), velocity is found to be a function of several quantities which in general, vary from problem to problem. In case, $\bar{u}_s = 0 = \delta_i (i=1,2)$, $\bar{C} = k$(constant), the present problem reduces to Poiseuille flow model; for $\delta_i = \delta_2$, $\delta_1 > 0$, it leads to one-dimensional (1-D) Newtonian fluid flow (with usual zero-slip or slip) in a constricted artery with the growth of a single stenosis and, for $\delta_i (\delta_1 < = \delta_2)$, it results in the present model under investigation (Figure 7.1), with the presence ($\bar{u}_s > 0$) and absence ($\bar{u}_s = 0$) of a velocity slip. It may be worth recorded that in many existing models, pressure gradient term is taken as constant ($\bar{C} = 1$) where as in our case, $\bar{C}$ is taken otherwise (i.e., $\bar{C} <, > 1$).

### 7.4.1 Velocity Function

Since velocity profiles could provide an elaborate description of flow field, one may be interested to study their flow pattern. A comparison of velocity profiles, obtained from equation (7.3.16) for cases of usual slip or no-slip condition, other parameters etc., is shown in Figures (7.2-7.3). The variations of axial velocity vs. radial distance, in a tube of 20 $\mu m$ semi-diameter at $H=40\%$, $\bar{\mu} = 1.783$ for locations i.e., at the two ends of the first stenosis (Figure 7.2) and at its peak (Figure 7.3), are exhibited.
**Figure 7.2:** Variation of axial velocity with radial distance for different values of pressure gradient and slip velocity for $\delta_1 = \delta_2 = 1/2$.

**Figure 7.3:** Variation of axial velocity with radial distance for different values of shear viscosity and slip velocity for $\delta_1 = \delta_2 = 1/2$. 
It is observed that velocity increases with the slip and that with a rise in pressure gradient term. But as expected, magnitudes of velocity are higher at the two ends of a stenosis than those attained at the site of maximum constriction (i.e., at the minimum cross-sectional area of the tube). The profiles drawn for velocity vs. radial distance for the other stenosis formed in 35 μm tube radius at H=40%, $\mu = 2.425$, exhibit the same trend in profiles i.e., as $\bar{u}_s$ increases, velocity increases and as $\bar{C}$ ($<, > 1$) rises in magnitude, axial velocity increases accordingly. In all these profiles, velocity attains the maximum magnitude at the initiation or termination of stenoses and, the minimum value at their peak (i.e., at maximum constriction). As blood viscosity $\bar{\mu}$ increases in magnitude (with a rise in tube size), velocity decreases in all cases, but velocity increases with slip. It is thus seen that axial velocity increases with the slip and a rise in $\bar{C}$, but decreases with a rise in $\bar{\mu}$ for both uniform and stenosed vessels. However, velocity reaches the maximum magnitude at the tube axis and the minimum value at the vessel wall.

7.4.2 Flow Rate

The rate of flow across every cross-sectional area of an artery, prevailing in the cardiovascular system, seems very important as it is directly linked with the blood circulation in human systems. Figures (7.4-7.5) show the variation of flow rate vs. axial distance for different values of $\bar{C}$, $\bar{u}_s$ and two tube radii ($R_0 = 20, 35 \mu m$).
Figure 7.4: Variation of flow rate with axial distance for different values of slip velocity and shear viscosity for $\delta^1 = \delta^2 = 1/2$.

Figure 7.5: Variation of flow rate with axial distance for different values of pressure gradient $\bar{C}$ for $\delta^1 = \delta^2 = 1/2$.
It is noticed that flow rate $\dot{Q}$ decreases as axial distance increases from the initiation of the first stenosis to its peak and then it increases in the remaining part of this constriction, $\dot{Q}$ is constant in the uniform tube region and in the second stenosis, $\dot{Q}$ exhibits the same kind of behavior, as done in the first constricted region. As usual, $\dot{Q}$ increases with an increase in velocity slip. As $\bar{C}$ increases in magnitude, flow rate increases accordingly. However, as fluid viscosity $\mu$ (at H=40%) increases, flow rate decreases. The behavior of flow rate is found to be alike in both the tubes ($R_0 = 20, 35$ $\mu$m) considered. Thus, flow rate increases with a rise in both $\bar{C}$ and $\bar{u}_s$, but it decreases with a rise in viscosity $\mu$ at 40% H. Its magnitude is the greatest in the uniform artery region (i.e., at the two ends of a stenosis) and the lowest at the middle (peak) of the stenosis. However, these computed values are seen to be greater with an axial slip than those obtained with zero-slip at the constricted wall.

### 7.4.3 Wall Shear Stress

This flow variable is equally important in blood flow modeling as its proper measure can prevent further damage to the vessel wall that was already deteriorated due to the formation of stenoses. In Figure 7.6-7.7, variation of wall shear stress versus the vessel length (axial distance) for different values of stenoses sizes, pressure gradient parameter, is presented.
Figure 7.6: Variation of wall shear stress with axial distance for different values of both stenoses size for $\bar{C} = 0.95$.

Figure 7.7: Variation of wall shear stress with axial distance for different values of $\bar{C}$ for $\delta_1 = \delta_2 = 1/2$. 
It may be referred that with an increase in the magnitude of both the stenoses, shear stress $\tau_{R(z)}$ increases. But wall shear stress decreases with the increasing magnitude of $C$. As axial distance $z$ increases from one end (initiation) of the constriction to its peak (at the middle), shear stress increases and for the remaining half portion of the stenosis, it decreases. However, it is constant in the uniform artery region but exhibits the same kind of behavior in the second stenosis. Thus, wall shear stress distribution in the stenotic region increases with the axial distance in the upstream of the stenosis throat, wherefrom it decreases with the axial distance.

### 7.4.4 Apparent Viscosity

The role of apparent viscosity is quite important as it ($\mu_a$) could be used as a parameter for indicating the different cardiovascular and arterial diseases. Figures (7.8-7.9) exhibit the variation of apparent viscosity vs. axial distance (vessel length) for different values of pressure gradient $C$.

![Figure 7.8: Variation of apparent viscosity with axial distance for different values of stenoses size.](image-url)
As axial length $\bar{z}$ increases along the vessel length, $\bar{\mu}_a$ increases from one end of the stenosis to its peak and there from, it decreases to the downstream of the stenosis. As fluid viscosity $\mu$ increases in magnitude, apparent viscosity increases. Also, in the stenotic regions $\bar{\mu}_a$ increases from one end (i.e., its initiation) at $\bar{z} = d$ to its peak (i.e., at maximum constriction) at $\bar{z} = d + L_0/2$, as radial distance decreases and $\bar{\mu}_a$ decreases in the downstream (i.e., in the portion between its peak at $\bar{z} = d$ and its termination at $\bar{z} = d + L_0$) as radial distance increases, therefore apparent viscosity exhibits both Inverse Fahreus Lindqvist Effect (IFLE) and Fahraeus Lindqvist Effect (FLE) here. Further, as tube radius increases from 20 $\mu$m to 35 $\mu$m, $\bar{\mu}_a$ increases throughout. Thus, $\bar{\mu}_a$ exhibits Fahreus-Lindqvist Effect (FLE) from this point of view. As expected, apparent viscosity decreases with the employment of an axial velocity slip at vessel wall.

7.4.5 Pressure Gradient Profiles

It could play a significant role in the study of blood flow through constricted vessels. The variations of pressure gradient $d\bar{p}/d\bar{z}$ versus axial distance, for different values of slip velocity $\bar{u}_s$, stenosis size, shear viscosity, are exhibited in Figures (7.10-7.11).
Figure 7.10: Variation of pressure gradient with axial distance for different values of shear viscosity for $\delta_1 = \delta_2 = 1/2$.

Figure 7.11: Variation of pressure gradient with axial distance for different values of stenosis size and slip velocity.
As $\bar{u}$ increases, $d\bar{p}/dz$ decreases for both the stenosis considered, but it is not so much prominent for severe stenosis case. As shear viscosity increases from one end (initiation) of a stenosis to its peak, $d\bar{p}/dz$ increases upstream and then decreases in the downstream. In the uniform artery region, $d\bar{p}/dz$ is constant but in the constricted region, it is otherwise. However, its maximum magnitude arises at the peak of the stenosis and the minimum at the two ends (initiation and termination) of the constriction. This behavior is seen to be alike in the second stenosis with an exception that in this case, the highest value attained at the minimum cross-sectional area is less than that obtained in the first stenosis case.

7.5 Conclusion

In the present work, one-dimensional (1-D) flow of blood through a multi-stenosed artery with an axial velocity slip at the constricted wall (Figure 7.1), has been studied. In the analysis, flow is considered steady and laminar and, blood is assumed to behave as an incompressible Newtonian viscous fluid. Although, blood exhibits a non-Newtonian character at low shear rates (Biswas and Laskar, 2011), it is already reported in literature, at high shear rates, generally found in larger arteries, blood acts like a Newtonian fluid (Fung, 1981; Biswas, 2000). Since stenoses are commonly formed and developed in large diameter arteries where blood shows a Newtonian behaviour (Young, 1968), therefore it is rational here, in assuming marvellous body fluid blood to behave as a Newtonian fluid in this constricted artery, with the presence of successive two severe stenoses. An effort is made to study the combined influence of different parameters like, velocity slip, stenosis size and location, pressure gradient, blood viscosity, tube size etc., on the flow variables. Analytic expressions for flow variables viz., velocity function, flow rate, pressure gradient, pressure drop, resistance to flow, wall shear stress and apparent viscosity, in both dimensional and dimensionless forms, are obtained and their variations with flow parameters are presented in Figures (7.2-7.12). It is of interest to record that it includes Poiseuille flow of blood with axial wall slip or no-slip cases and 1-D Newtonian flow of blood in a constricted artery with slip or zero-slip at vessel wall, as its special cases.
Many researchers have already reported that the rheologic and fluid dynamic properties of blood and its flow like velocity profiles, velocity gradients, shear stresses at wall, apparent viscosity of blood, boundary conditions etc., could play a vital role in the fundamental understanding, diagnosis and treatment of many cardiovascular, cerebrovascular, renal and arterial diseases (Biswas, 2000). There is enough evidence that hydrodynamic factors could play a significant role in the formation, development and progression of an arterial stenosis (Young, 1968). Further, the presence of stenosis in one or more of the major blood vessels carrying blood to the heart, brain etc., could lead to various arterial diseases viz., angina pectoris, myocardial infarction, cerebral accident, coronary thrombosis etc. (Biswas, 2000). Keeping in view of above, we have considered blood flow in tubes with the presence of two successive stenoses and investigated the behaviour of such flow variables. From the variation of flow variables (Figures 7.2-7.12), it is easily seen that axial velocity and flow rate increase with an increase of slip velocity but apparent viscosity decreases. Velocity profiles indicate a non-parabolic trend in both the tubes considered. As expected velocity and flow rate attain the highest magnitude at the initiation and termination of this stenosis and the lowest at its throat. However, velocity reaches the maximum at the axis and the minimum at the vessel wall. As fluid viscosity \( \mu \) at 40% H increases in magnitude, both the velocity and flow rate decrease, but with a rise in pressure gradient magnitude, both the flow variables increase in both the tubes considered. Wall shear stress and the apparent viscosity reach the maximum at the throat and the minimum at the two ends of a stenosis. Apparent viscosity exhibits Fahraeus Lindqvist Effect and its Inversion (IFLE) in the stenotic region for both the tube sizes. Pressure gradient is seen to be higher with the slip condition than that with a zero-slip at wall. However, it attains the highest magnitude at the middle of a mild stenosis and the lowest at its two ends. It indicates vanishing of pressure gradient and a negative trend in the profiles drawn for zero-slip case. Thus, the present model could explain two blood flow anomalies viz., plug velocity profiles and, both FLE and IFLE in apparent viscosity for blood flow in a constricted artery.

In this theoretical investigation, it is easily found that with the employment of a velocity slip, speed and flow rate increase but apparent viscosity decreases. Therefore, with slip, flow rate can be accelerated and resistance to flow can be retarded. Thus, it can be
concluded that with the introduction of a slip at the stenotic wall, damage to a diseased or occluded artery could be lowered. In view of above, useful device or appropriate drug could be designed for producing slip at the constricted artery wall. That, in turn may help in reducing the stenosis height on one hand and in restoring the normal blood supply on the other. The existing experiments on blood flow through stenosed arteries provide only one aspect viz., pressure drop, it could be worthwhile in obtaining the other aspects i.e., wall shear stress, slips at the vessel wall, flow rate etc. Such kind of investigations may be useful for determining the growth, development and progression of an arterial stenosis. This in turn, may be important for the better understanding of stenotic diseases (stroke, thrombosis, angina pectoris, arterial disorders, HBSS-Sickle cell diseases etc.). The model proposed may be used in designing of prosthetic blood vessels.