



EFFECT OF AXIAL VISCOSITY VARIATION THROUGH AN ATHEROSCLEROTIC ARTERY: A NON-NEWTONIAN FLUID MODEL

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Abstract

A mathematical model has been designed to study the effect of axial viscosity variation on various parameters of blood flow in an overlapping stenosed artery using Herschel-Bulkley fluid . This model has been undertaken to investigate the structure of the flow through an arterial segment with overlapping stenosis. The constitutive equations of the model are solved analytically using the initial conditions and given boundary conditions to get expressions for different parameters such as flow rate ,resistance to flow and wall shear stress . Variations of these flow parameters are analysed graphically.

Keywords- Blood flow, blood viscosity, overlapping stenosis, Herschel-Bulkley fluid, flow rate, resistance to flow, wall shear stress.

studies have turned into a predominated zone of multidisciplinary exploration.

Congenital and acquired cardiovascular diseases represent one of the most important causes of morbidity and mortality in the world. Among the acquired cardiovascular diseases, atherosclerosis is the most common manifestation which is often characterized by arterial narrowing and reductions in blood flow . While the risk factors of this disease such as nicotine, high cholesterol and familiar history are systemic , the manifestations (typically in the form of plaque deposits) are localized in areas of complex flow like the coronary, carotid, abdominal and femoral arteries. Hemodynamic quantities such as blood velocity, pressure and shear stress play a very important role in the localization of disease and in the efficacy of treatments. For both congenital and acquired cardiovascular diseases, a deep understanding of the altered blood flow conditions can enable the optimization of interventions employed to treat these conditions.

Stenosis in the arteries of mammals are a common occurrence and for many years researchers have endeavoured to model the flow of blood through stenosed arteries experimentally and theoretically. The deposition of cholesterol and proliferation of the connective tissues in the arterial wall form plaques which grow inward and restrict the blood flow. In order to have a fuller understanding of the development of these diseases, an accurate knowledge of the mechanical properties of the

I. INTRODUCTION

Mathematical modelling is the application of mathematics to explain and predict real world behaviour. It basically comprises of making an interpretation of certifiable issues into scientific issues, tackling the numerical issues and translating these arrangements in the dialect of real world. A new multidisciplinary science is now arising which bridges the gap and draws upon both the life science and physical science for help and support. This area is Biomechanics. The components in charge of the onset and advancement of vascular diseases have not been completely comprehended, and the related

vascular wall together with the flow characteristics of blood are indispensable. Thus relevant information is deemed to be of great help in the treatment of vascular diseases and also to bioengineers who are engaged in the design and construction of improved artificial organs. Perhaps the actual cause of abnormal growth in an artery is not completely clear to the theoretical investigators but its effect over the cardiovascular system has been determined by studying the flow characteristics of blood in the stenosed area.

The frequently occurring cardiovascular disease, stenosis or arteriosclerosis is the abnormal and unnatural growth in the arterial wall thickness that develops at various locations of the cardiovascular system under diseased conditions which occasionally results into serious consequences Srivastava [12].

To understand the effects of a single mild stenosis present in the arterial lumen, a good number of studies (Young[13] Shukla et al.[11]) on the blood flow through stenosed arteries have been performed. All these studies were made with the assumption that the flowing blood is Newtonian and the geometry of the stenosis is a smooth cosine function. However, it has been observed from experimental investigations that blood, being a suspension of cells, behaves like a non-Newtonian fluid at low shear rates in smaller arteries (Barnett and Han[1]). It is also realized that the Herschel-Bulkley model is a better model than Casson's model (Blair and Spanner[2]). Further, in small diameter tubes blood behaves like a Herschel-Bulkley fluid rather than power law and Bingham fluids (Chaturani and Samy [5]).

Misra et al.[10] developed a mathematical model(Herschel-Bulkley fluid model) for studying the non-Newtonian flow of blood through a stenosed arterial segment. The problem is investigated by a combined use of analytical and numerical techniques and it is noticed that the resistance of flow and the skin-friction increase as the stenosis height increases. Nallapu et al.[9] studied a two fluid model of Herschel-Bulkley fluid flow through tubes of small diameters and assumed that the core region consists of Herschel-Bulkley fluid and Newtonian fluid in the peripheral region and derived the analytical solutions for velocity, flowflux, effective viscosity, core hematocrit and mean hematocrit

and the effects of various relevant parameters on these flow variables have been studied.

In most of the recent literature about stenotic flow in either a rigid tube or a flexible artery, the stenotic geometry has been regarded as time-independent. Such a consideration may suit well for a rigid vessel but for a flexible vessel wall, the stenosis cannot remain static. Moreover, the problem becomes more acute in the presence of an overlapping stenosis in the arterial lumen instead of having a single (mild) stenosis as considered by aforesaid researchers. A consistent mathematical model for the unsteady flow of blood through an overlapping arterial stenosis was put forward first by Chakravarty and Mandal[4] in which the streaming blood was treated as a viscoelastic fluid and the flow was considered only in the axial direction.

Layek et al.[8] investigated the effects of an overlapping stenosis on flow characteristics considering the pressure variation in both the radial and axial directions of the arterial segment and treating blood as Newtonian . Gupta.S et al [6] investigated the effects of stenosis and radial variation of viscosity on the flow characteristics of blood considering laminar,incompressible and non-Newtonian flow of blood using Power-Law fluid model. Jain.N et al. [7] observed various flow characteristics of blood and effect of various parameters of stenosis using Herschel-Bulkley non-Newtonian fluid model considering steady,laminar,one-dimensional flow of blood through an axially non-symmetric but the radially symmetric atherosclerotic artery.

Motivated by these facts , we propose to study the "Effect of axial viscosity variation on blood flow in an overlapping stenosed artery when blood is considered as Herschel-Bulkley fluid ".

II. FORMULATION OF THE MODEL

For the development of mathematical model, following assumptions are made :

- Flow of blood is designed as laminar, steady and non-Newtonian in behaviour and the way of streaming blood is incompressible, homogeneous.
- The flowing blood is not affected by any external force.
- Viscosity of blood varies along the axial direction and there exists a axial decrease in blood

viscosity i.e., it is maximum at the axis and minimum near the wall.

- The stenosis in the artery is axially symmetric and depends upon the axial distance z and the height of its growth.
- Stenosis is taken overlapping.
- Radial velocity in the stenotic region is very small in comparison to the axial velocity Young [16].

The geometry of the stenosis, assumed to be manifested in the arterial segment is described (Chakravarty and Mandal [4]) in Fig. 1 as

$$\frac{R(z)}{R_o} = 1 - \frac{3\delta}{2R_o(l_o)^4} \{ 11(z-d)(l_o)^3 - 47(z-d)^2(l_o)^2 + 72(z-d)^3(l_o) - 36(z-d)^4 \} ; d \leq z \leq d+l$$

$$= 1 \quad \text{otherwise} \quad (1)$$

where R_o is the radius of the artery (assumed to be a rigid circular tube) outside the stenosis, $R(z)$ is the radius of the stenosed portion of the arterial segment, l_o is the length of the stenosis, d indicates its location and δ is the maximum height of the stenosis into the lumen, appears at the two different locations: $z=d+ l_o/6$ and $z=d+5 l_o/6$ The height of the stenosis at $z=d+ l_o/2$, called critical height is $3/4 \delta$.

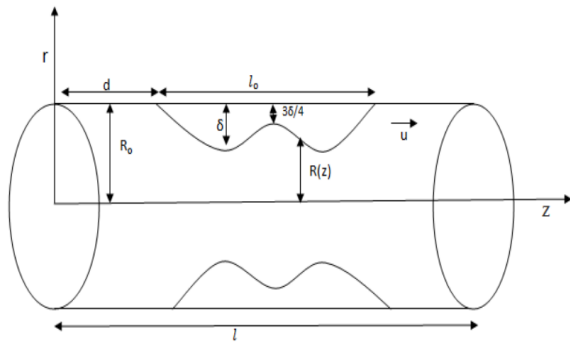


Fig.1 The flow geometry of an arterial overlapping stenosis

The Navier-Stoke equation [14] is

$$-\frac{\partial p}{\partial z} + \frac{1}{r} \frac{\partial}{\partial r} (r\tau) = 0 \quad (2)$$

where r and z be the radial and axial coordinates respectively, p is pressure and τ be shear stress. The flow of blood is assumed to be steady, laminar and incompressible. No external force is being applied on the artery. The viscosity of blood is a function of z and is given by

$$\mu(z) = \mu_o \left(\frac{R(z)}{R_o} \right)^{-\alpha} ; d \leq z \leq d+l_o$$

$$= \mu_o ; \text{ elsewhere} \quad (3)$$

where μ_o is the constant viscosity of the blood and $\alpha = 0, 1, 2, \dots$ is any arbitrary constant parameter, which is the index of viscosity variation in the stenotic region. The form of viscosity variation assumed in equation (3) is very natural because accumulation of blood cells occurs in the stenotic region just before the minimum gap where radius also becomes minimum, but the viscosity becomes maximum.

The constitutive equation for Herschel-Bulkley fluid is given by

$$\tau = \mu \left(-\frac{\partial u}{\partial r} \right)^n + \tau_o ; \tau \geq \tau_o \quad (4)$$

$$\frac{\partial u}{\partial r} = 0 ; \tau < \tau_o \quad (5)$$

where τ_o be the yield stress and μ be the viscosity coefficient of blood. The boundary conditions pertaining to the problem are :

$$\text{At } r=R(z), w=0 \text{ and at } r=0, \frac{\partial w}{\partial r} = 0, \quad (6)$$

Where w is axial velocity and n is the Herschel-Bulkley index number.

The equation of motion describing one-dimensional flow of blood treating as Herschel-Bulkley fluid whose viscosity varies along the z -axis may be expressed as

$$-\frac{\partial p}{\partial z} + \frac{1}{r} \frac{\partial}{\partial r} \left[r \left\{ \mu(z) \left(-\frac{\partial w}{\partial r} \right)^n + \tau_o \right\} \right] = 0 \quad (7)$$

Where τ_o is the yield stress , r and z is the radial and axial coordinate respectively, p is pressure and $\mu(z)$ is the viscosity of blood.

The expression for velocity profiles w ,by integrating equation (7) and using conditions (6), can be obtained as

$$w = A \left\{ \left[\frac{R(z) P}{2} - \tau_o \right]^{1+1/n} - \left[\frac{r P}{2} - \tau_o \right]^{1+1/n} \right\} \quad (8)$$

$$\text{where } A = \frac{2}{P (1+1/n) [\mu(z)]^{1/n}} \quad \text{and } P = \frac{\partial p}{\partial z} .$$

The flow rate Q is given by

$$\begin{aligned}
 Q &= \pi \int_0^{R(z)} r^2 \left(-\frac{dw}{dr} \right) dr \\
 &= C \left[\frac{R(z)^2}{2} \left\{ \frac{R(z) P}{2} - \tau_0 \right\}^{1+1/n} - \right. \\
 &\quad \left. \frac{2 R(z)}{(2+1/n) P} \left\{ \frac{R(z) P}{2} - \tau_0 \right\}^{2+1/n} + \right. \\
 &\quad \left. \frac{4}{(2+1/n)(3+1/n) P^2} \left[\left\{ \frac{R(z) P}{2} - \tau_0 \right\}^{3+1/n} - \right. \right. \\
 &\quad \left. \left. (-\tau_0)^{3+1/n} \right] \right] \quad (9)
 \end{aligned}$$

Where $C = \frac{4 \pi}{(1+1/n) [\mu(z)]^{1/n} P}$

The **pressure gradient** can be obtained as:

$$P = \frac{\partial p}{\partial z} = \frac{2 Q^n \mu(z)}{\pi^n I(r)^n} \quad (10)$$

Where $I(r) = \int_0^{R(z)} r^2 \left[r - \frac{2 \tau_0}{P} \right]^{1/n} dr$

To obtain **pressure drop** (Δp), integrating equation (10) under the conditions:

$$p = p_0 \text{ at } z = 0 \quad \text{and at } z = \ell, \quad p = p_\ell \quad (11)$$

Integrating eq. (10) under boundary conditions (11), we get

$$\Delta p = p_\ell - p_0 = \frac{2 Q^n}{\pi^n} \int_0^\ell \frac{\mu(z)}{I(r)^n} dz$$

Following Burton [3] and Young [13], the **resistance to flow** is:

$$\lambda = \frac{\Delta p}{Q} = \frac{2 Q^n}{\pi^n} \int_0^\ell \frac{\mu(z)}{I(r)^n} dz$$

Now resistance to flow at $r = R(z)$ and at extreme stenosis height is

$$\lambda_R = \frac{2 Q^n}{\pi^n} \int_0^\ell \frac{\mu(z)}{I(R)^n} dz \quad \text{at } z = d + \frac{\ell_0}{2}$$

For normal artery, the resistance to flow is given by

$$\lambda_N = \frac{2 Q^n}{\pi^n} \int_0^\ell \frac{\mu(z)}{I(R_0)^n} dz$$

Where $I(R) = \int_0^{R(z)} r^2 \left[r - \frac{2 \tau_0}{P} \right]^{1/n} dr$ and

$$I(R_0) = \int_0^{R_0} r^2 \left[r - \frac{2 \tau_0}{P} \right]^{1/n} dr.$$

Thus the ratio of resistance to flow is

$$\lambda' = \frac{\lambda_R}{\lambda_N}$$

The **wall shear stress** is given by

$$\begin{aligned}
 \tau_w &= \mu(z) \left(-\frac{\partial w}{\partial r} \right)^n + \tau_0 \quad \text{at } r = R(z) \\
 &= \frac{R(z) \partial p}{2 \partial z}
 \end{aligned}$$

Now using equation (12), we have

$$\tau_w = \frac{R(z) Q^n \mu(z)}{\pi^n I(R(z))^n}$$

From it, wall shear stress at extreme stenosis height is

$$\tau_{ws} = \frac{R(z) Q^n \mu(z)}{\pi^n I(R(z))^n} \quad \text{at } z = d + \frac{\ell_0}{2}$$

For normal artery, the wall shear stress is given by

$$\tau_N = \frac{R_0 Q^n \mu(z)}{\pi^n I(R_0)^n}$$

So the ratio of wall shear stress is

$$\tau' = \frac{\tau_{ws}}{\tau_N} = \frac{R(z)}{R_0} \left(\frac{I(R_0)}{I(R(z))} \right)^n$$

III. RESULTS AND DISCUSSION

The analytical expressions for flow rate, resistance to flow and wall shear stress have been derived and computed numerically. The numerical solutions are presented in graphical form.

Figs.2-7 reveals the variation of flow rate (Q) with stenosis height to radius ratio (δ/R_0) and axial distance to radius ratio (z/R_0) for different values of n , α and τ_0 . It is clear that increment in n , α and τ_0 decreases the flow rate with increment in stenosis height. Also on increasing axial distance, the flow rate first decreases and then increases in the non stenotic region for different values of n , α and τ_0 .

Figs.8-10 shows that resistance to flow increases on increasing the stenosis height. It is clear that resistance to flow decreases on increment of pressure gradient and it increases on increment of n and τ_0 .

Figs.11-13 shows that wall shear stress increases as the stenosis height increases for fixed values of n , P and τ_0 .

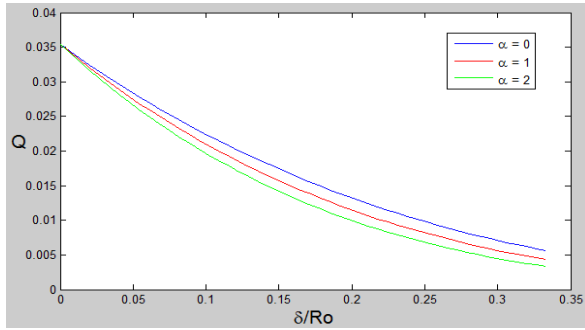


Fig.2 Profiles for Q against δ/Ro for different values of α

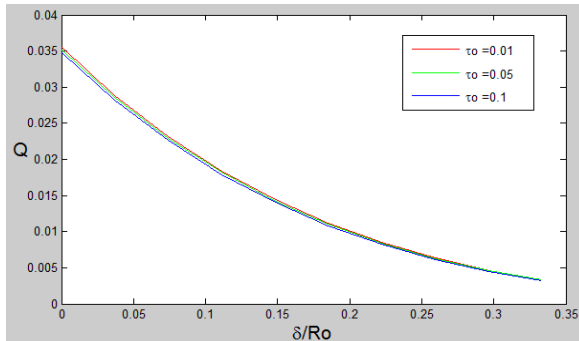


Fig.3 Profiles for Q against δ/Ro for different values of τ_0 .

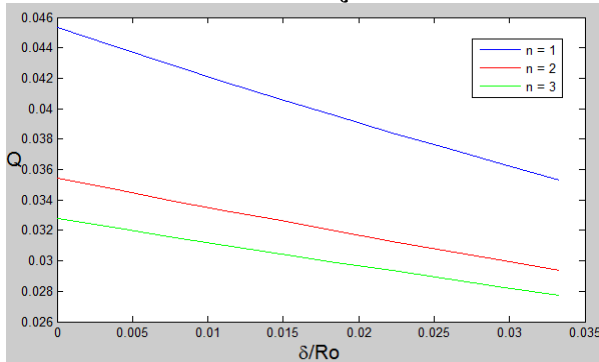


Fig.4 Profiles for Q against δ/Ro for different values of n.

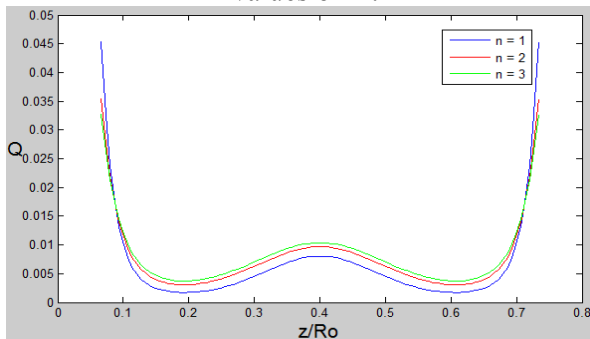


Fig.5 Profiles for Q against z/Ro for different values of n.

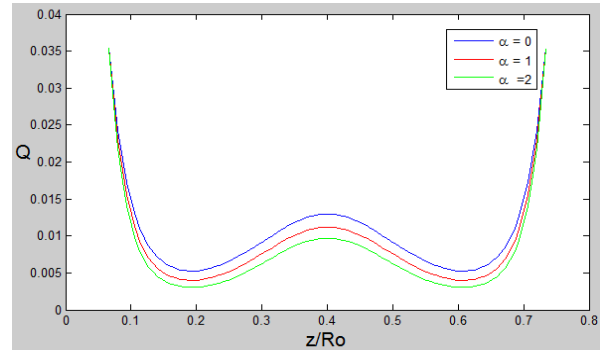


Fig.6 Profiles for Q against z/Ro for different values of α .

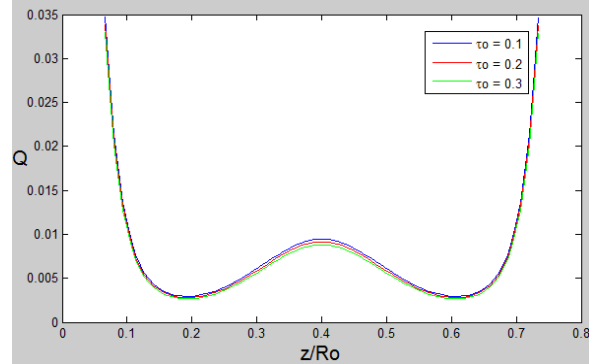


Fig.7 Profiles for Q against z/Ro for different values of τ_0 .

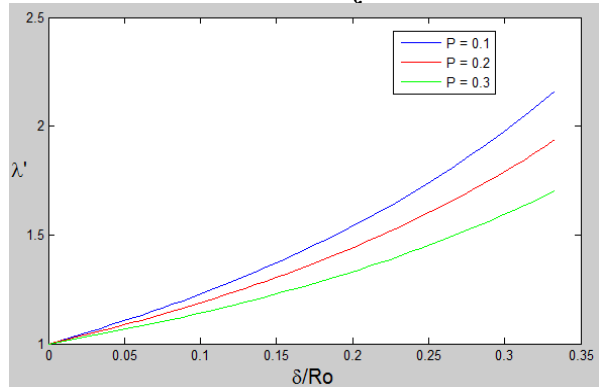


Fig.8 Profiles for λ' against δ/Ro for different values of pressure gradient ($P = \frac{dp}{dz}$).

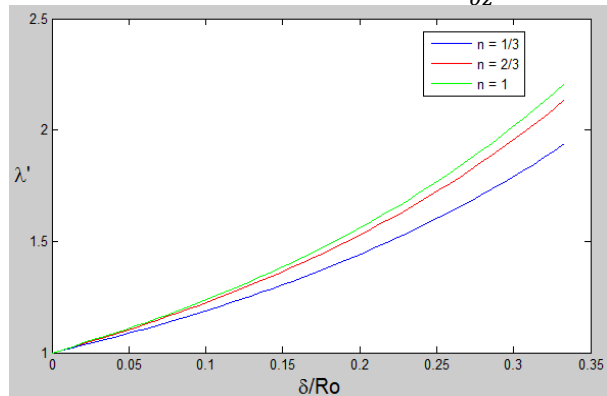


Fig.9 Profiles for λ' against δ/Ro for different values of n.

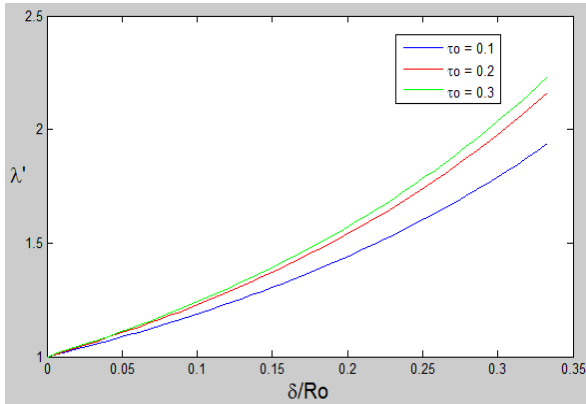


Fig.10 Profiles for λ' against δ/Ro for different values of τ_0 .

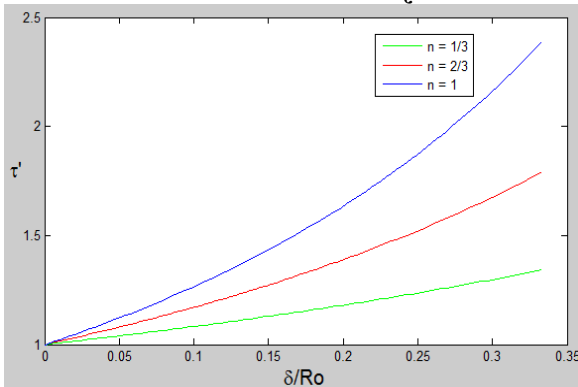


Fig.11 Profiles for τ' against δ/Ro for different values of n .

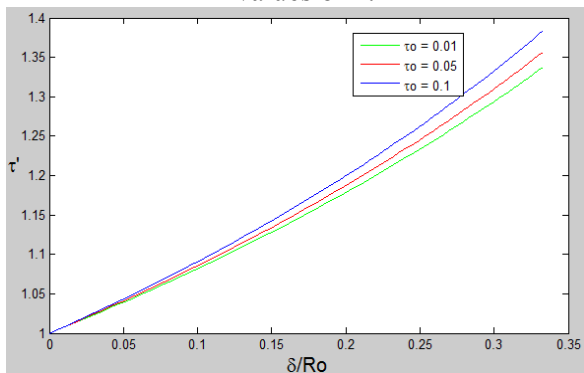


Fig.12 Profiles for τ' against δ/Ro for different values of τ_0 .

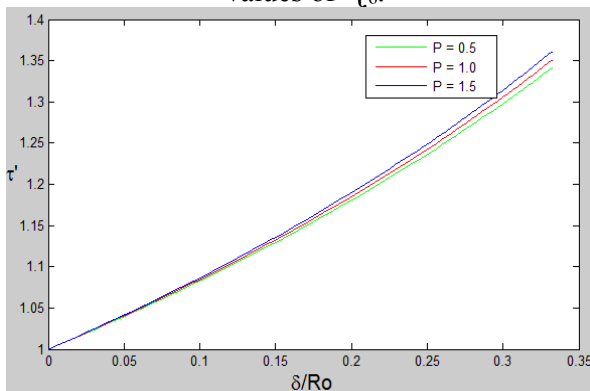


Fig.13 Profiles for τ' against δ/Ro for different values of pressure gradient ($P = \frac{dp}{dz}$).

IV. CONCLUSION

A non-Newtonian fluid (Herschel-Bulkley) model has been applied to investigate the effects on axial viscosity variation of blood flow due to the presence of an overlapping stenosis in arteries. The analytical expression is obtained for the flow rate, resistance to flow and wall shear stress and results are discussed on the basis of graphical presentation. It is found that flow rate decreases, resistance to flow increases and wall shear stress increases as the stenosis height is increased. Flow rate of the fluid first decreases as the axial distance increases and then it increases with the value of axial distance and attains its minimum value when stenosis size is maximum within the stenosis region. This study may be beneficial in the field of medical sciences as the medical scientists do not have precious information about various flow parameters of blood.

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