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Pulsatile Flow of Blood in a Constricted Artery with Body Acceleration

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Abstract

Pulsatile flow of blood through a uniform artery in the presence of a mild stenosis has been investigated in this paper. Blood has been represented by a Newtonian fluid. This model has been used to study the influence of body acceleration and a velocity slip at wall, in blood flow through stenosed arteries. By employing a perturbation analysis, analytic expressions for the velocity profile, flow rate, wall shear stress and effective viscosity, are derived. The variations of flow variables with different parameters are shown diagrammatically and discussed. It is noticed that velocity and flow rate increase but effective viscosity decreases, due to a wall slip. Flow rate and speed enhance further due to the influence of body acceleration. Biological implications of this modeling are briefly discussed.

Keywords: Pulsatile flow, Velocity-slip, Body acceleration, Newtonian fluid, Stenosis

MSC 2000 No.: 76Z05, 74G10

1. Introduction

An abnormal growth, formed due to deposits of atherosclerotic plaques in the lumen of an artery, is called stenosis (atherosclerosis) and, its subsequent and severe growth on the artery wall, results in serious circulatory disorders [Young (1979), Biswas (2000), Bali and Awasthi (2007)]. These disorders in circulatory systems may be included as, narrowing in body

passage leading to the reduction and impediment to blood flow in the constricted artery regions, the blockage of the artery in making the flow irregular and causing an abnormality of the blood flow and, the presence of stenosis at one or more of the major blood vessels, carrying blood to the heart or brain etc., could lead to various arterial diseases e.g., myocardial infarction, angina pectoris, cerebral accident, coronary thrombosis, strokes etc. [Young, (1968), Chakraborty et al. (1995), Biswas (2000), Sankar and Lee (2009)]. There is a good amount of evidence that hydrodynamic factors could play an important role in the formation, development and progression of an arterial stenosis [Young and Tsai (1973), Biswas (2000)]. Further, a good many researchers viz., Dintenfass (1977), Chien (1981), Caro (1981), Biswas (2000) have already reported that the rheological and fluid dynamic properties of blood and its flow could play a vital role in the fundamental understanding, diagnosis and treatment of many cardiovascular, cerebrovascular and arterial diseases. As rheological properties and flow behavior of blood are of immense importance in the fundamental study of different diseases in general and arterial stenosis in particular, many authors have proposed theoretical models [Forrester and Young (1970), Morgan and Young (1979), McDonald (1979), Misra and Chakraborty (1986)] and experimental work [Young and Tsai (1973)] for blood flow through stenosed arteries.

In the above theoretical models, different aspects of blood flow through stenosed arteries are studied, by considering blood in behaving like a Newtonian fluid (Schlichting and Gersten (2004)). Young (1968) has analyzed theoretically the effects of stenosis on flow characteristics of blood and, concluded that the resistance to flow (impedance) and the wall shear stress, increase with the increase in stenosis size. Forrester and Young (1970) extended the theory of Young (1968), to include the effects of flow separation on a mild constriction. Results of experimental work on models of an arterial stenosis have been presented by Young and Tsai (1973).

Sankar and Lee (2009) have reported that the study of blood flow through stenosed arteries is very important as because, the cause and development of many arterial diseases leading to the malfunction of cardiovascular system, are to a great extent related to the flow characteristics of blood. Although, blood exhibits a non-Newtonian character at low shear rates [Merrill (1965)], at high shear rates generally found in larger arteries [diameter nearly above 1mm), blood behaves like a Newtonian fluid (Taylor (1959)]. Since, stenosis normally generates and develops in large diameter arteries (in the range of 500 to 2000 μm), where blood shows a Newtonian behavior, it appears to be reasonable in assuming blood to be homogeneous, isotropic, incompressible, Newtonian continuum, having a constant viscosity and density for flow through stenosed arteries (having respective radii 10.0, 5.0, 4.0 and 1.5mm in aorta, femoral, carotid and coronary arteries [Sud and Sekhon (1985)].

The flow of blood in arteries is pulsatile due to the heart pulse pressure gradient and the pulsatile flow (caused by the periodic disturbance) of blood through an artery, has already drawn a considerable attention to the researchers for quite a long time [Womersley (1955), Chandra and Krishna Prasad (1994), Sankar and Lee (2009)], owing to its great importance in medical science. It is also reported that arterial blood flow is highly pulsatile with marked effects on instantaneous velocity distribution and the flow rate varies over a wide range during a flow cycle [Liesch (1986), Sud and Sekhon (1984), Sud and Sekhon (1987)].

Several other studies include, pulsatile blood flow in a rigid elliptic tube with varying cross-section [Mehrotra et al. (1985)], inside an artery with time-dependent stenosis [Misra et al. (2008)] and through a mild stenosed artery [Nagarani and Sarojamma (2008), Sankar and Lee

(2009)]. However, under some exceptional circumstances, e.g., while riding in or driving a heavy vehicle (tractor or tank in particular), while flying in a spacecraft or a helicopter, in particular while landing and taking-off, operating jack hammers, athletes and sportsmen, taking their fast and sudden action etc., human body is subjected to whole-body accelerations (or vibrations), intentional or unintentional. In all such cases, our body is subjected to an external acceleration. Though, human body can adapt to changes but prolonged exposures to such accelerations, may lead to many health problems like, headache, joint pain, vascular disorders, abdominal pain, loss of vision, increased pulse rate etc. [Burton et al. (1974)].

To study the influence of such acceleration, Sud and Sekhon (1987) have presented a mathematical model of laminar and uni-directional blood flow in a stenosed artery, subject to periodic body acceleration. The effect of body acceleration on unsteady flow of blood through a stenosed artery by applying a finite difference scheme is modeled by Mandal et al. (2007). In most of the aforementioned studies, traditional no-slip boundary condition [Schlichting and Gersten (2004)] has been employed. However, a number of studies of suspensions in general and blood flow in particular both theoretical [Vand (1948), Jones (1966), Nubar (1967), Brunn (1975), Chaturani and Biswas (1984), Biswas (2000)] and experimental [Bennet (1967), Bugliarello and Hayden (1962)], have suggested the likely presence of slip (a velocity discontinuity) at the flow boundaries (or in their immediate neighborhood). The apparent (effective) viscosity will be lowered, as a result of wall slip [Brunn (1975), Biswas (2000)].

Recently, Misra and Shit (2007) and Ponalgusamy (2007) have developed mathematical models for blood flow through stenosed arterial segment, by taking a velocity slip condition at the constricted wall. Thus, it seems that consideration of a velocity slip at the stenosed vessel wall will be quite rational, in blood flow modeling. The intent of the present analysis is to study the effects of slip (at the stenotic wall) and the influence of body acceleration, on the flow variables (wall shear stress, velocity profiles, flow rate and effective viscosity) for pulsatile blood (Newtonian fluid) flow through a stenosed vessel.

2. Mathematical Formulation

Consider a pulsatile, axially symmetric, laminar, one-dimensional and fully developed flow of blood through an artery (circular tube) with a mild stenosis (constriction), as shown in Figure 1. The constricted wall of this artery is assumed to be rigid. The geometry of an arterial stenosis (Figure 1) is mathematically modeled as [Nagarani and Sarojamma (2008)]

$$\bar{R}(\bar{z}) = \begin{cases} \bar{R}_0 - \frac{\bar{\delta}_s}{2} \left(1 + \cos \frac{\pi \bar{z}}{\bar{z}_0} \right), & \text{for } |\bar{z}| \leq \bar{z}_0 \\ \bar{R}_0, & \text{for } |\bar{z}| > \bar{z}_0, \end{cases} \quad (1)$$

where $\bar{R}(\bar{z})$ is the radius of the artery in the stenotic region, \bar{R}_0 is the constant radius of the normal artery in the non-stenotic region, \bar{z}_0 is the half-length of the stenosis and $\bar{\delta}_s$ is the maximum height of the stenosis such that $\frac{\bar{\delta}_s}{\bar{R}_0} < 1$ (mild stenosis). It has been reported that the

radial velocity being negligibly small can be neglected for a low Reynolds number in case of a tube embedded with a mild stenosis [Nagarani and Sarojamma (2008), Sankar and Lee

(2009)]. The Navier-Stokes equations of motion in cylindrical coordinate system $(\bar{r}, \bar{\theta}, \bar{z})$ governing the fluid flow are given by [Schlichting and Gersten (2004)]

$$\bar{\rho} \frac{\partial \bar{u}}{\partial \bar{t}} = -\frac{\partial \bar{p}}{\partial \bar{z}} - \frac{1}{\bar{r}} \frac{\partial}{\partial \bar{r}} (\bar{r} \bar{\tau}) + \bar{F}(\bar{t}) \tag{2}$$

$$\frac{\partial \bar{p}}{\partial \bar{r}} = 0, \tag{3}$$

where \bar{u} represents the axial velocity along \bar{z} -direction, \bar{p} the pressure, $\bar{\rho}$ the density, \bar{t} the time, $\bar{\tau}$ the shear stress and $\bar{F}(\bar{t})$ the body acceleration. The constitutive equation of a Newtonian fluid (blood) can be written as

$$\bar{\tau} = -\bar{\mu} \frac{\partial \bar{u}}{\partial \bar{r}}, \tag{4}$$

where $\bar{\mu}$ is the shear viscosity of blood.

The boundary conditions are

$$(i) \bar{u} = \bar{u}_s \text{ at } \bar{r} = \bar{R}(\bar{z}) \tag{5}$$

$$(ii) \frac{\partial \bar{u}}{\partial \bar{r}} = 0 \text{ at } \bar{r} = 0 \tag{6}$$

where \bar{u}_s is the slip velocity at the stenotic wall [Chaturani and Biswas (1984)].

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

$$-\frac{\partial \bar{p}}{\partial \bar{z}}(\bar{z}, \bar{t}) = A_0 + A_1 \cos(\bar{\omega}_p \bar{t}), \quad t \geq 0, \tag{7}$$

where, A_0 is the steady state pressure gradient, A_1 is amplitude of the fluctuating component, $\bar{\omega}_p = 2\pi \bar{f}_p$ and \bar{f}_p is the pulse rate frequency. Both A_0 and A_1 are functions of \bar{z} [Nagarani and Sarojamma (2008)]. For $t > 0$, the flow is subjected to a periodic body acceleration $\bar{F}(\bar{t})$ which has been expressed as [Nagarani and Sarojamma (2008), Sud and Sekhon (1985)]

$$\bar{F}(\bar{t}) = a_0 \cos(\bar{\omega}_b \bar{t} + \phi), \quad t > 0, \tag{8}$$

where $\bar{\omega}_b = 2\pi \bar{f}_b$, \bar{f}_b and a_0 are frequency in Hz and amplitude of body acceleration, ϕ is the lead angle with respect to the heart action. The frequency of body acceleration \bar{f}_b is assumed to be so small that wave effect can be neglected [Nagarani and Sarojamma (2008)].

In order to express, the governing equations of motion and conditions employed, dimensionless and, also to study the behavior of flow variables, we introduce the following

non-dimensional variables (as obtained with the help of characteristic quantities $\bar{R}_0, \bar{A}_0, \bar{A}_1$ etc.) we introduce the following non dimensional variables

$$z = \frac{\bar{z}}{R_0}, R(z) = \frac{\bar{R}(\bar{z})}{R_0}, r = \frac{\bar{r}}{R_0}, t = \bar{t} \bar{\omega}_p, \omega = \frac{\bar{\omega}_b}{\bar{\omega}_p}, \delta_s = \frac{\bar{\delta}_s}{R_0}, u = \frac{\bar{u}}{\frac{A_0 R_0}{4\bar{\mu}}},$$

$$u_s = \frac{\bar{u}_s}{\frac{A_0 R_0}{4\bar{\mu}}}, \tau = \frac{\bar{\tau}}{A_0 R_0}, \alpha^2 = \frac{\bar{R}_0^2 \bar{\omega}_p}{\bar{\mu}}, e = \frac{A_1}{A_0}, B = \frac{a_0}{A_0}, \quad (9)$$

where α is the pulsatile Reynolds number or generalized Womersley frequency parameter. Using non-dimensional variables as enlisted in equation (9), the basic equations (2) and (4) reduce to the following forms

$$\alpha^2 \frac{\partial u}{\partial t} = 4(1 + e \cos t) + 4B \cos(\omega t + \phi) - \frac{2}{r} \frac{\partial}{\partial r}(r\tau), \quad (10)$$

$$\tau = -\frac{1}{2} \frac{\partial u}{\partial r}. \quad (11)$$

Inserting the expression for τ from equation (11), in equation (10), we get

$$\alpha^2 \frac{\partial u}{\partial t} = 4(1 + e \cos t) + 4B \cos(\omega t + \phi) + \frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u}{\partial r} \right). \quad (12)$$

The boundary conditions (5) and (6), in the dimensionless form are

$$u = u_s \text{ at } r = R(z), \quad (13)$$

$$\frac{\partial u}{\partial r} = 0 \text{ at } r = 0. \quad (14)$$

The geometry of an arterial stenosis in dimensionless form is given by

$$R(z) = \begin{cases} 1 - \frac{\delta_s}{2} \left(1 + \cos \frac{\pi z}{z_0} \right), & \text{for } |z| \leq z_0, \\ 1, & \text{for } |z| > z_0. \end{cases} \quad (15)$$

The non-dimensional volumetric flow rate $Q(z, t)$ can be defined as

$$Q(z, t) = 4 \int_0^{R(z)} r u(z, r, t) dr, \quad (16)$$

where $Q(z, t) = \frac{\bar{Q}(\bar{z}, \bar{t})}{\frac{\pi(R_0)^4 A_0}{8\bar{\mu}}}$, $\bar{Q}(\bar{z}, \bar{t})$ is the volumetric flow rate.

The effective viscosity $\bar{\mu}_e$ defined as

$$\bar{\mu}_e = \frac{\pi \left(-\frac{\partial \bar{p}}{\partial \bar{z}} \right) (\bar{R}(\bar{z}))^4}{\bar{Q}(\bar{z}, \bar{t})} \quad [\text{Pennington and Cowin (1970)}] \quad (17)$$

can be expressed in dimensionless form as

$$\mu_e = \frac{(R(z))^4}{Q(z, t)} (1 + e \cos t), \quad (18)$$

where $Q(z, t)$ is defined in equation (16).

3. Method of Solution

Considering the Womersley parameter to be small, the velocity u can be expressed in the following form

$$u(z, r, t) = u_0(z, r, t) + \alpha^2 u_1(z, r, t) + \dots \quad (19)$$

Substituting the expression of u from equation (19) in (12), we have

$$\frac{\partial}{\partial r} \left(r \frac{\partial u_0}{\partial r} \right) = -4r \{ (1 + e \cos t) + B \cos(\omega t + \phi) \}, \quad (20)$$

$$\frac{\partial u_0}{\partial t} = \frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u_1}{\partial r} \right). \quad (21)$$

Substituting u from equation (19) into conditions (13) and (14) we get

$$u_0 = u_s, u_1 = 0 \text{ at } r = R(z), \quad (22)$$

$$\frac{\partial u_0}{\partial r} = 0, \frac{\partial u_1}{\partial r} = 0 \text{ at } r = 0. \quad (23)$$

To determine u_0 and u_1 , we integrate equations (20) and (21) twice with respect to r and use the boundary conditions (22) and (23) (for u_1 , using the expression obtained for u_0), we have

$$u_0 = u_s + f(t) \left((R(z))^2 - r^2 \right), \quad (24)$$

where $f(t) = (1 + e \cos t) + B \cos(\omega t + \phi)$ and

$$u_1 = \frac{1}{16} f'(t) \left(4(R(z))^2 r^2 - 3(R(z))^4 - r^4 \right). \tag{25}$$

Neglecting terms of $o(\alpha^4)$ and other higher powers of α , the expression for axial velocity $u(r, z, t)$ as obtained from equations (19), (24) and (25), is

$$u(z, r, t) = u_s + f(t) \left((R(z))^2 - r^2 \right) + \frac{\alpha^2}{16} f'(t) \left(4(R(z))^2 r^2 - 3(R(z))^4 - r^4 \right),$$

$$|z_0| \leq z, \quad 0 \leq r \leq R(z), \quad t \geq 0. \tag{26}$$

The wall shear stress τ_w (as a result of equations (11) and (19)) becomes

$$\tau_w = -\frac{1}{2} \left(\frac{\partial u_0}{\partial r} + \alpha^2 \frac{\partial u_1}{\partial r} \right)_{r=R(z)}, \tag{27}$$

which is determined, by substituting velocity expressions (24) and (25) into the above equation (27), in the form

$$\tau_w = f(t) R(z) - \frac{\alpha^2}{8} f'(t) (R(z))^3. \tag{28}$$

The expression for volumetric flow rate $Q(z, t)$, as a consequence of equations (16) and (26) becomes

$$Q(z, t) = (R(z))^2 \left\{ 2u_s + f(t) (R(z))^2 - \frac{\alpha^2}{6} f'(t) (R(z))^4 \right\}. \tag{29}$$

Analytic expression for effective viscosity μ_e , as obtained from equations (18) and (29) is given by

$$\mu_e = (R(z))^2 (1 + e \cos t) \left\{ 2u_s + f(t) (R(z))^2 - \frac{\alpha^2}{6} f'(t) (R(z))^4 \right\}^{-1}. \tag{30}$$

4. Results and Discussions

The present model has been developed to study the combined effect of body acceleration, stenosis and slip-velocity on the pulsatile flow of blood through a stenosed tube considering blood as to behave like a Newtonian fluid. The equations governing the abovementioned flow are integrated by using a perturbation analysis with a very small Womersley frequency

parameter ($\alpha = 0.5 < 1$). Analytic expressions are obtained for axial velocity, flow-rate, wall shear stress and effective viscosity.

When slip velocity $u_s = 0$, the present analysis leads to pulsatile flow model of blood in a stenosed artery with body acceleration and usual no-slip at the constricted vessel wall. For $u_s = 0 = \alpha$, $R(z) = 1$ (i.e., $\bar{R}(\bar{z}) = R_0$) and $f(t) = \text{constant}$, it yields to a parabolic velocity profile corresponding to Poiseuille flow.

The existence of slip at the blood vessel wall have been indicated both theoretically (Brunn (1975), Jones (1966)) and experimentally [Bugliarello and Hayden (1962), Bennet (1967)] and the methods to detect (Astarita et al. (1964)) and determine (Cheng (1974)) slip experimentally, have been suggested in literature, the magnitudes of the wall slip are yet to be determined. In order to study the effects and influence of slip on the flow parameters, two values of u_s viz., $u_s = 0$ and $u_s = 0.05$ are taken in this investigation. Further, in many situations, the human body is subjected to body accelerations (or vibrations) intentional or unintentional that may lead to many health problems. Just to study the influence of body acceleration on blood flow through a constricted artery, three values of body acceleration parameter B viz., $B = 0, 1$ and 2 are considered. In the foregoing analysis, we have attempted to show the variations in flow characteristics due to slip (at the stenosed wall), body acceleration and other parameters.

Since velocity profiles provide a detailed description of the flow field, it is of interest to study their pattern. A comparison of velocity profiles, using equation (26) for cases of slip and no slip, body acceleration parameter etc., is shown in Figures 2 and 3. It is observed from the profiles (drawn at $z = 0$) that axial velocity increases with a constant wall slip for fixed magnitudes of e and t . It increases with the radial distance attaining the maximum magnitude at the axis ($r = 0$) and the minimum at the boundary ($r = R(z)$). As the body acceleration parameter B increases, velocity increases. As time $t (\geq 0)$ increases, velocity decreases. However, velocity is seen to be greater in case of a uniform tube than that in a constricted artery. It is clearly indicated that the flow parameters B and u_s bring in significant changes in axial velocity both qualitatively and quantitatively. Velocity profiles indicate a non-parabolic trend (almost plugged profiles for $\delta_s > 0$).

The variation of volumetric flow rate with the pressure gradient parameter e for different parameters is presented in Figure 4. It is observed that flow rate, in all cases, increases with the employment of axial wall slip. As e increases, flow rate increases accordingly. For both slip and no-slip cases, magnitudes of flow rate are seen to be higher in a uniform tube than those in a stenosed artery. As body acceleration parameter B increases, flow rate increases. The lowest and the highest values of Q are noticed at $e = 0$ and $e > 0$ respectively. The profiles obtained with $B = 1, u_s = 0 = \delta_s$ and $B = 0 = u_s, \delta_s = 0.2 (> 0)$ indicate the highest and the lowest trends in the flow rate Q . Thus, considering the variations in B, u_s and δ_s , it is seen that flow rates in uniform arteries are greater than those in constricted arteries. The flow rate is enhanced by the introduction of a body acceleration parameter in the flow regime and it is further enhanced by the introduction of a velocity slip.

The wall shear stress τ_w and its variations with parameters B, e and δ_s , for a full scale of time $t (0 \leq t \leq 360)$ in degrees, have been computed from equation (28) and shown in Figure 5. It could be actually noticed that τ_w is greatly influenced by the body acceleration parameter B in a stenosed artery and as B increases, τ_w decreases. However, τ_w attains its minimum at $t = 180^\circ$ and the maximum at $t = 0^\circ, 360^\circ$.

The variations of effective viscosity μ_e [computed from equation (30)] with axial distance z , axial slip u_s and stenosis height δ_s , are shown in Figure 6. It is observed that the effective viscosity μ_e is less with the velocity slip than that with no-slip at the vessel boundary. As stenosis height δ_s increases, μ_e increases. Also, μ_e varies markedly through the stenosis ($\delta_s > 0$) reaching a maximum value at the section of minimum cross-sectional area (at $z = 0$) and minimum at the two ends ($z = -z_0, z_0$). The corresponding μ_e in a normal artery region ($\delta_s = 0$), is represented by dotted straight lines. However, introduction of velocity slip at the arterial wall reduces the effective viscosity in both uniform ($\delta_s = 0$) and stenosed vessels ($\delta_s > 0$). Also, μ_e increases, as δ_s increases. Thus, μ_e in this case shows Inverse Fahraeus-Linqvist Effect (IFLE).

5. Conclusion

The present analysis deals with pulsatile blood flow through a stenosed artery (Figure 1) subject to periodic body acceleration and an axial velocity slip at the constricted wall. The equations of motion, governing the flow are integrated by using perturbation method. It is of interest that it includes Poiseulli's parabolic profile as its special case. Analytic expressions for flow variables are obtained and their variations with different flow parameters are presented graphically. It is observed that as expected, axial velocity and flow rate increase with the wall slip whereas effective viscosity decreases due to a slip. Also velocity and flow rate increase but wall shear stress shows both increasing and decreasing trends with the rise in body acceleration parameter B . Effective viscosity μ_e increases as δ_s increases. However, μ_e is lowered for both uniform tube ($\delta_s = 0$) and stenosed artery ($\delta_s > 0$), as a result of wall slip.

The observations made in this investigation are in agreement with the theoretical model of Young (1968) that the wall shear stress and resistance to flow increases with an increase in stenosis size. The present analysis shows two anomalous behaviors in blood flow, viz., plugged velocity profiles and Inverse Fahraeus-Lindqvist (IFLE).

In this model, it is noticed that due to the employment of a velocity slip at the constricted wall, axial velocity and flow rate both will increase but effective viscosity will decrease. Also, velocity and flow rate further enhance whereas effective viscosity decreases due to the influence of periodic body acceleration. This clearly indicates that there are great influences of both slip and body acceleration on the flow variables. Further, these show that slip at a diseased artery could play a prominent role in blood flow modeling. It may be worthwhile to notice that employment of slip at wall will accelerate the speed and flow rate on one hand and

retard the resistance to flow on the other. As a result, bore of the vessel will increase, stenosis size will be lowered and rate of flow will be higher than earlier. From the analysis, it may be concluded that with slip, the damages to the vessel wall could be reduced. This kind of reduction in wall shear stress, effective viscosity could be exploited for better functioning of the diseased arterial systems. Hence one may look forward for drugs or devices which would produce slip and use them for treatment of peripheral arterial diseases.

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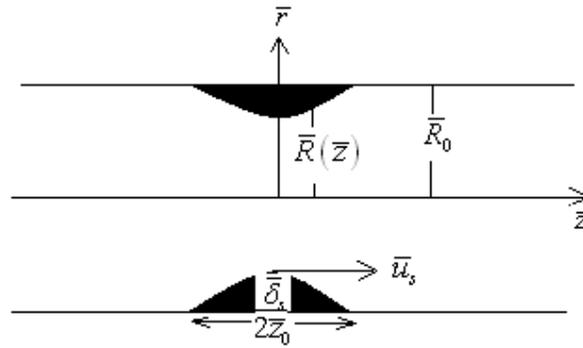


Figure 1. Schematic diagram of an artery with stenosis.

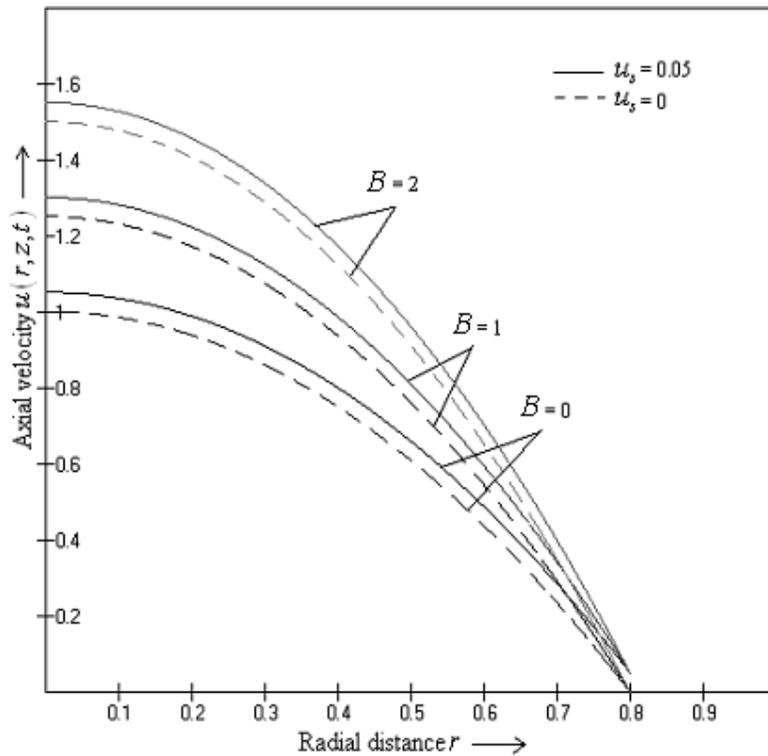


Figure 2. Variation of axial velocity with radial distance at $z = 0$ for different values of u_s and B with $e = 1$ and $t = 1$.

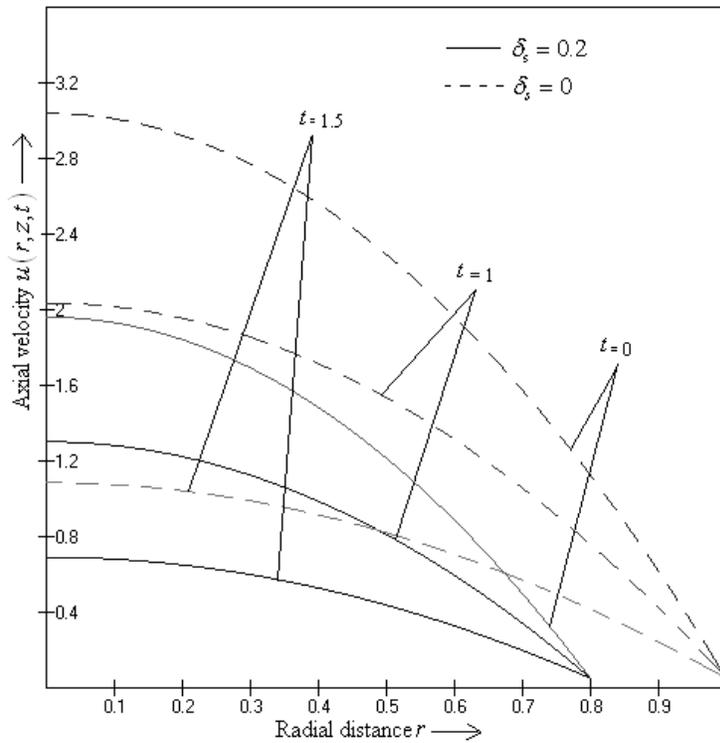


Figure 3. Variation of axial velocity with radial distance at $z = 0$ for different values of time and height of stenosis with $B = 1, e = 1, u_s = 0.05$

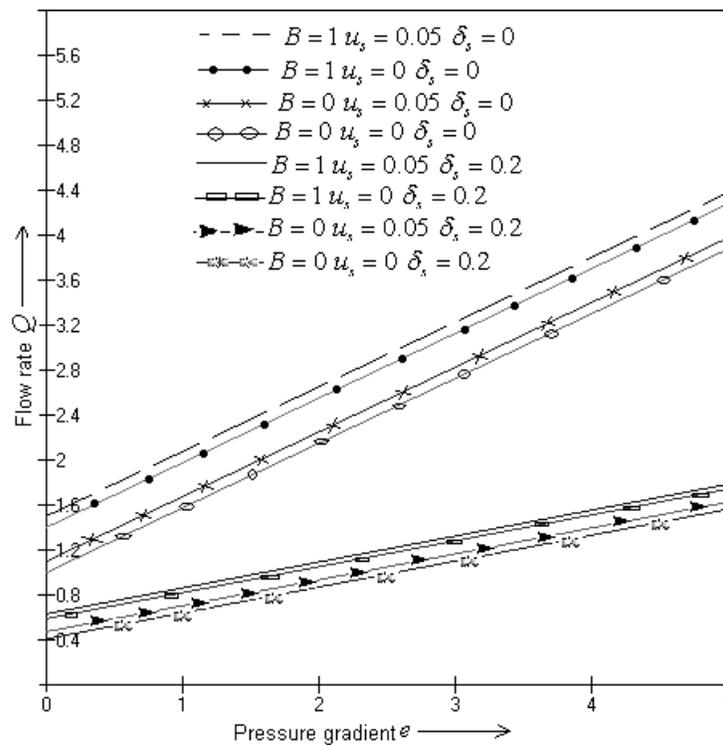


Figure 4. Variation of flow-rate with pressure gradient for different values of B, u_s and δ_s with $t = 1$

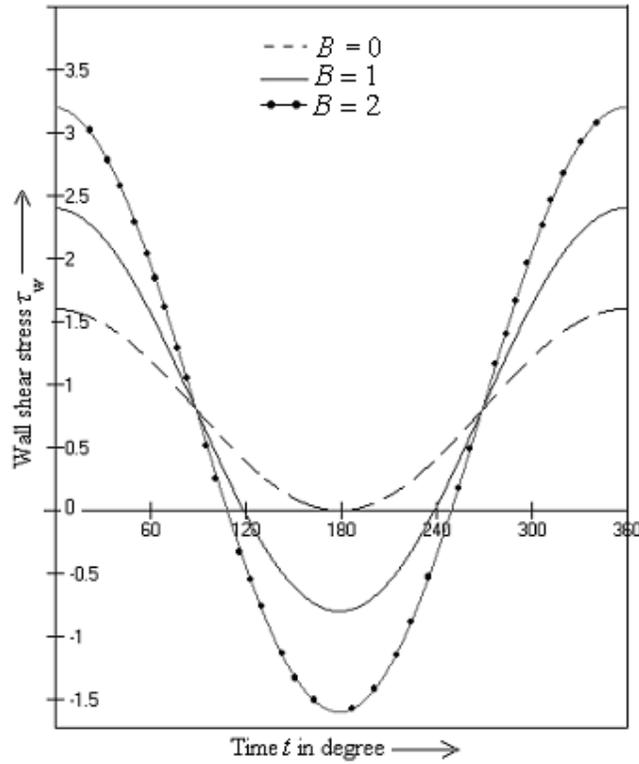


Figure 5. Variation of wall shear stress with time for different values of B with $e = 1$, and $\delta_s = 0.2$

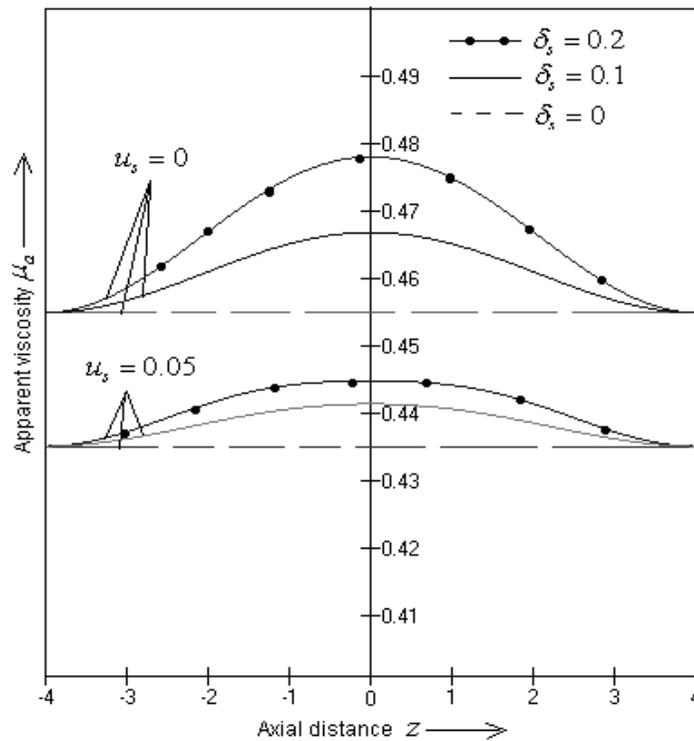


Figure 6. Variation of Apparent viscosity with axial distance for different values of δ_s and u_s with $e = 1$, $B = 1$ and $t = 1$