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Mathematical Modeling of Non-Newtonian Blood Flow through Artery in the Presence of Stenosis

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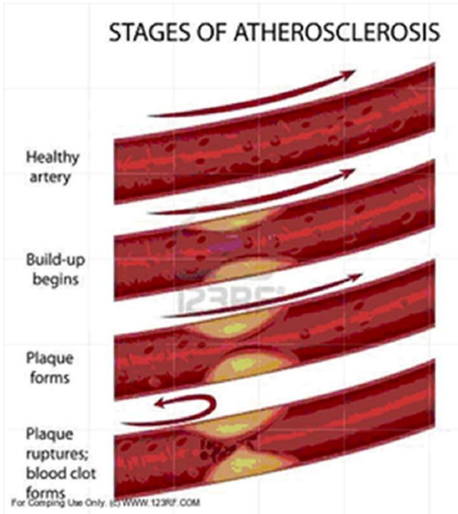
ABSTRACT

The aim of this paper is to develop a mathematical model for studying the non-Newtonian flow of blood through a stenosed arterial segment. Power law fluid represents the non-Newtonian character of blood. The hemodynamic behavior of the blood flow is influenced by the presence of the arterial stenosis. The problem is solved by using analytical techniques with help of boundary conditions and results are displayed graphically for different flow characteristics like pressure drop, shear stress, velocity profile. For the validation of numerical model, the computation results are compared with the results from published literature.

Keyword: Power law fluid model, shear stress, pressure drop, stenosis height.

INTRODUCTION:

Stenosis is a narrowing of any tubular structure in the body, including blood vessels, heart valves, vertebral canal and the GI tract. Blood vessel narrowing is one of the more common usages of this term. High cholesterol could contribute to the stenosis of an artery when it accumulates on the inner wall of the artery. This accumulation, referred to as atherosclerosis, can build up within the artery to the point where it reduces blood flow to organs of the body. When blood flow is reduced, nutrients and oxygen cannot travel to the tissues that need it. Below are a series of illustrations that will help us to understand the process of atherosclerosis (vascular disease) and the kinds of problems that can arise in this condition:



The presence of stenosis can lead to serious circulatory disorders. There is strong evidence that hydrodynamic factors such as resistance to flow, wall shear stress and apparent viscosity may play a vital role in the development and the progression of arterial stenosis. Many researchers [2] feel that the hydrodynamic factors may be helpful in the diagnosis, treatment and fundamental understanding of many disorders. Verma et. al**.** [9] studied the shape of stenosis to blood flow through an artery with mild stenosis and they evaluated, for a given rate of flow, the wall shear stress increases immediately as the stenosis increases in size. Misra & Shit [3] investigated blood flow through arterial segment assuming blood as Hershal-Bulkley fluid. They obtained that the skin-friction and the resistance to flow is maximum at the throat of the stenosis and minimum at the end. Ali et. al. [6] analyzed the effect of an axially symmetric time dependent growth into the lumen of a tube for constant cross section through which a Newtonian fluid is steadily flowing .They investigated the structure of flow through arterial model with one or two sinusoidal stenosis, assuming tha arterial blood flow is quasi-steady. Shah & Siddiqui [8] studied the influence of peripheral layer viscosity on physiological characteristics of blood flow through stenosed artery using Power–law fluid model. It was observed that the resistance to flow increases as stenosis size and peripheral layer viscosity increases. They found that the peripheral layer viscosity of blood in diabetic patients is higher than in non-diabetic patients, resulting higher resistance to blood flow. Thus diabetic patients with higher peripheral layer viscosity are more prone to high blood pressure. Therefore the resistance to blood flow in case of diabetic patients may be reduced by reducing viscosity of the plasma. They also investigated that the wall shear stress decreases as stenosis shape parameter increases but in the case of increasing stenosis size, stenosis length and peripheral layer viscosity wall shear stress is increases. Bali & Awasthi [7] analyzed the effect of external magnetic field on blood flow in stenotic artery. They presented that the resistance to flow increases with stenosis height and red cell, which depends on hematocrit value of blood, on the other side resistance to flow decreases with the increase in the value of Hartmann number. Mathur & Jain [5] studied the pulsatile flow of blood through stenosed arteries, including the effects of body acceleration and a magnetic field.

In present paper we have studied the effects of bell shaped stenosis on resistance to flow, wall shear stress and velocity profile in an artery by assuming the blood as power-law fluid.

THE FORMULATION OF THE PROBLEM AND ANALYSIS

In the present analysis, the following assumptions are made:

- The blood flow is modeled to be steady, one dimensional laminar and the nature of the flowing blood is incompressible, homogenous.
- There is no external force acting on the flowing blood.
- Stenosis developed in the artery in an axially symmetric manner and depends upon the axial distance z and the height of its growth.
- The maximum height of the stenosis is much less as compared to the length and unobstructed radius of the artery i.e., stenosis is mild.
- Radial velocity in the stenotic region is very small in comparison to the axial velocity [11].

Let us consider a bell shaped stenosis geometry given by [3]

$$\mathrm{R(z)} = \mathrm{R}_0\left[1-\frac{\delta}{\mathrm{R}_0}\exp\left(\frac{-m^2\mathrm{b}^2z^2}{\mathrm{R}_0^2}\right)\right] \tag{1}$$

where R_0 stands for the radius of the arterial segment outside the stenosis, R (z) is the radius of the arterial segment under consideration of a longitudinal distance z from the left end of the segment, δ is the depth of the stenosis at the throat and m is a parametric constant, b is characterizes the relative length of the constriction, defined as the ratio of the radius to half length of stenosis, i.e. $\mathrm{b}=\frac{\mathrm{R}_0}{\mathrm{z}_0}$.

Considering the stenosis geometry to be of the form

$$\frac{\mathrm{R(z)}}{\mathrm{R}_0} = \left[1-\mathrm{a}e^{-\varepsilon \mathrm{z}^2}\right] \tag{2}$$

With

$$\mathrm{a} = \frac{\delta}{\mathrm{R}_0} \text{ and } \varepsilon = \frac{m^2\mathrm{b}^2}{\mathrm{R}_0^2}.$$

Let us consider blood as a power law fluid, the constitutive equation for power law fluid is given as

$$\tau = \mu e^{n} \qquad \Rightarrow \left(\frac{\tau}{\mu}\right)^{\frac{1}{n}} = e = \left(\frac{\frac{1}{2}\mathrm{Pr}}{\mu}\right)^{\frac{1}{n}} = -\frac{du}{dr}$$

$$u = \left(\frac{P}{2\mu}\right)^{\frac{1}{n}} \frac{n}{n+1}\, r^{\frac{1}{n}+1} + c \tag{3}$$

Using boundary conditions

τ is finite at r = 0 (regularity condition) (4)

u=0 at r=R(z) (no-slip condition) (5)

$$u = \left(\frac{P}{2\mu}\right)^{\frac{1}{n}} \frac{n}{n+1}\left(R^{\frac{1}{n}+1} - r^{\frac{1}{n}+1}\right) \tag{6}$$

In the absence of the stenosis ($\delta = 0$)

$$u_{p} = \left(\frac{P}{2\mu}\right)^{\frac{1}{n}} \frac{n}{n+1}\left(R_{0}^{\frac{1}{n}+1} - r^{\frac{1}{n}+1}\right) \tag{7}$$

where the subscript p denotes the poiseuille flow

$$\frac{u}{u_{p}} = \frac{\left(R^{\frac{1}{n}+1} - r^{\frac{1}{n}+1}\right)}{\left(R_{0}^{\frac{1}{n}+1} - r^{\frac{1}{n}+1}\right)} \tag{8}$$

Flow rate can be given as [4]

$$Q = \int_{0}^{R} u\, 2\pi r\, dr$$

$$Q = \pi \left(\frac{P}{2\mu} \right)^{\frac{1}{n}} \frac{R^{\frac{1}{n}+3}}{\frac{1}{n}+3} \tag{9}$$

$$P = \frac{2\mu}{R^{1+3n}} \left(\frac{Q}{\pi} \left(\frac{1}{n} + 3 \right) \right)^{n} \tag{10}$$

Pressure drop across the length of the stenosis

$$\Delta P = \frac{2\mu}{R_0^{1+3n}} \left(\frac{Q}{\pi} \left(\frac{1}{n} + 3 \right) \right)^{n} \int_{-z_0}^{z_0} \frac{dz}{\left(1 - a e^{-bz^2}\right)^{1+3n}} \tag{11}$$

In the absence of the stenosis ($\delta = 0$)

$$(\Delta P)_p = \frac{4\mu}{R_0^{1+3n}} \left(\frac{Q}{\pi} \left(\frac{1}{n} + 3 \right) \right)^{n} z_0 \tag{12}$$

$$K = \frac{\Delta P}{(\Delta P)_p} = \frac{1}{2z_0} \int_{-z_0}^{z_0} \frac{dz}{\left(1 - a e^{-bz^2}\right)^{1+3n}} \tag{13}$$

If 2L is the length of the stenosis artery, pressure drop along the length of the artery can be given as

$$\Delta P = \frac{2\mu}{R_0^{1+3n}} \left(\frac{Q}{\pi} \left(\frac{1}{n} + 3 \right) \right)^{n} \left(2L - 2z_0\right) + \frac{2\mu}{R_0^{1+3n}} \left(\frac{Q}{\pi} \left(\frac{1}{n} + 3 \right) \right)^{n} \int_{-z_0}^{z_0} \frac{dz}{\left(1 - a e^{-bz^2}\right)^{1+3n}} \tag{14}$$

When there is no stenosis

$$(\Delta P)_p = \frac{4\mu}{R_0^{1+3n}} \left(\frac{Q}{\pi} \left(\frac{1}{n} + 3 \right) \right)^{n} L \tag{15}$$

$$K_1 = \frac{\Delta P}{(\Delta P)_p} = 1 - \frac{z_0}{L} + \frac{1}{2L} \int_{-z_0}^{z_0} \frac{dz}{\left(1 - a\,e^{-b z^2}\right)^{1+3n}} \tag{16}$$

The shear stress on the surface of the stenosis is given by

$$\tau = \frac{1}{2} P(z)\, R(z)$$

$$\tau = \frac{\mu}{R^{3n}} \left(\frac{Q}{\pi} \left(\frac{1}{n} + 3 \right) \right)^n \tag{17}$$

$$\frac{\tau}{\tau_p} = \left(\frac{R_0}{R} \right)^{3n} \tag{18}$$

RESULTS & CONCLUSION:

The results obtained in this study consist of the expression for velocity profile in Eqs. (8), expression for pressure drop across the length of the stenosis in Eqs. (13), expression for pressure drop along the length of the artery in Eqs. (16) and expression for shear stress in Eqs.(18) and displayed graphically .Figure (1) & (3) shows variation of pressure drop across the length of the stenosis w. r. t. stenosis height and the relative length of the constriction and highlights that the pressure drop increases as $\frac{\delta}{R_0}$ increases and decreases as b increases. Figure (2) depicts that the pressure drop along the length of the artery increases with $\frac{\delta}{R_0}$ and increases as $\frac{z_0}{L}$ increases for fixed n , this is in conformity with the results obtained by Kapur [4].

Figure 1: variation of pressure drop across the length of the stenosis w. r. t. stenosis height at b=0.64 and z=2.5

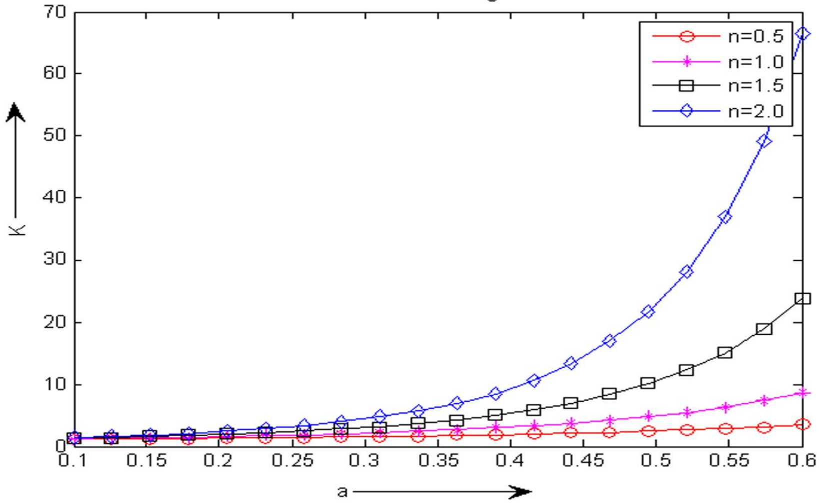


Figure 2: variation of pressure drop across length of the artey using different z0/L

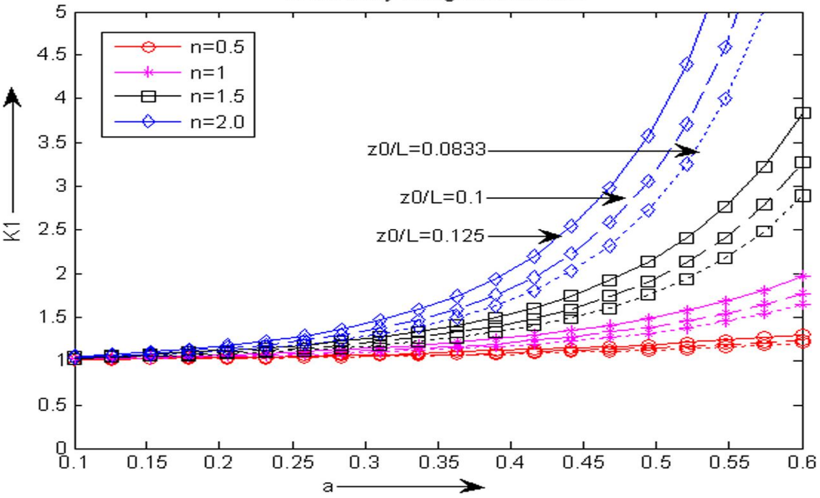
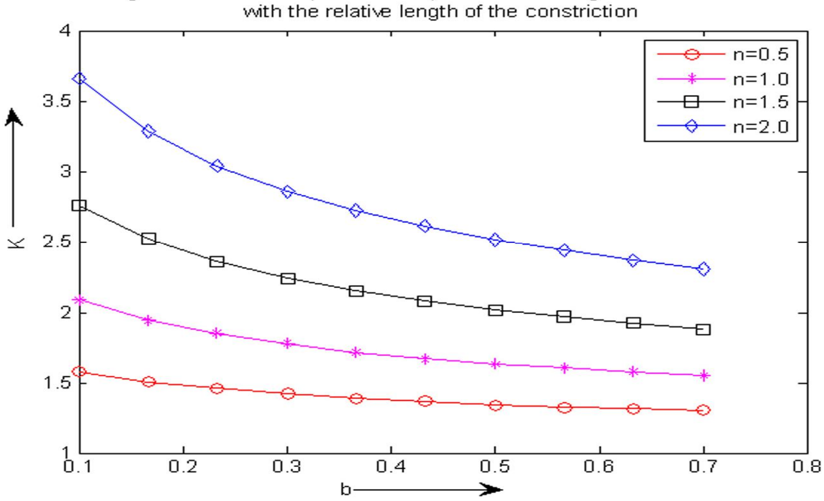


Figure 3: variation of pressure drop across the length of the stenosis with the relative length of the constriction



Variation of Wall shear stress with axial distance z, stenosis height and with b are presented in Fig. 4, Fig. 5 & Fig.6 respectively. Form the figure 4, it can be clearly observed that shear stress increases from its approached magnitude (i.e. at z = -2.5) in the upstream of the throat with the axial distance and achieves its maximal at the throat of the stenosis and then decreases in the downstream and attains a lower magnitude at the end of the constriction profile (i.e. at z =2.5). Magnitude of wall shear stress in uniform tube is lower than its magnitude in stenosed artery, this is in conformity with the results obtained by Biswas and Chakraborty [1]. It can be seen from figure 5 that shear stress increases with the height of stenosis and it is always greater than unity and increases as value of n increases, for fixed $\frac{\delta}{R_0}$. Figure 6 shows shear stress decreases with the relative length of the constriction.

Figure 4: variation of shear stress along the length of the arterial segment

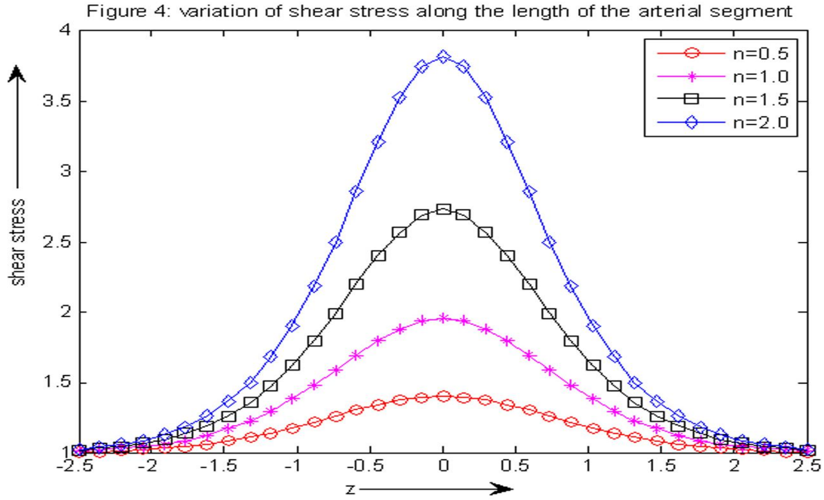
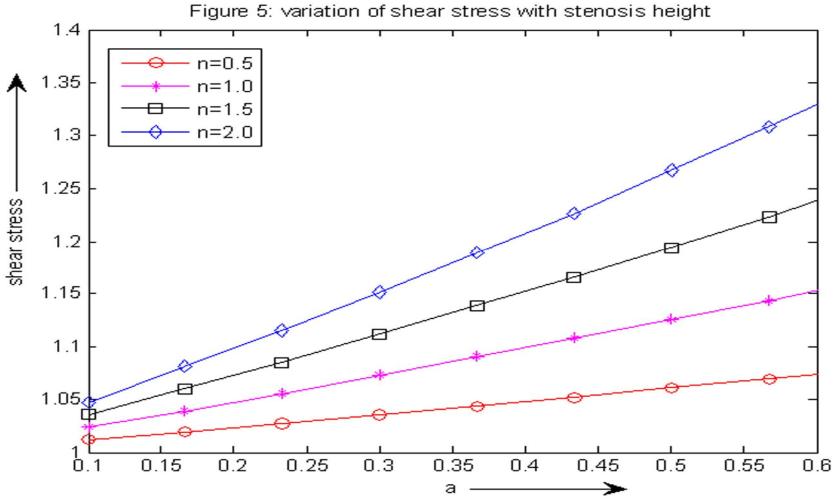


Figure 5: variation of shear stress with stenosis height



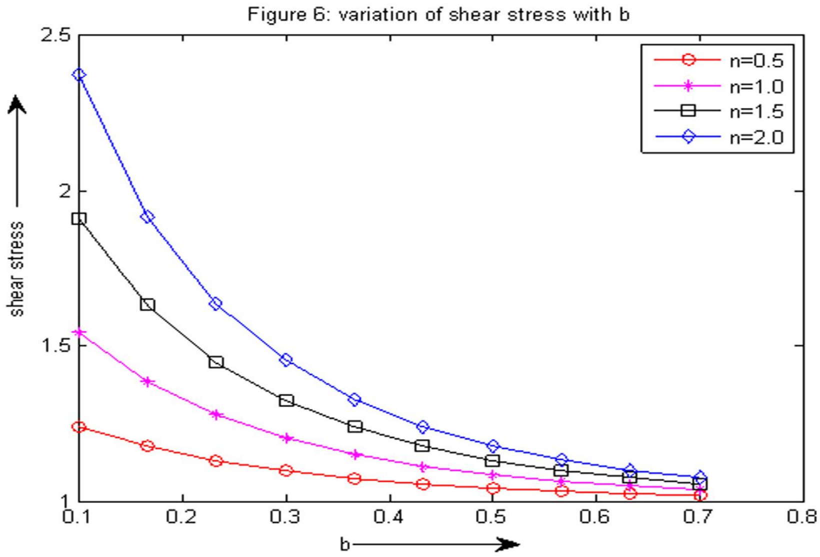
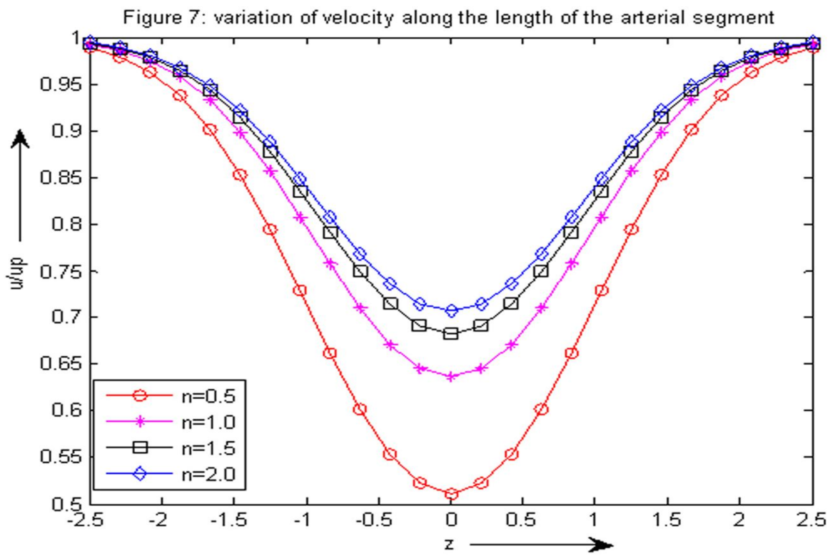


Figure (7), (8), (9), (10) depict the variation of velocity with z, a, radial distance r and b respectively. Figures (7) shows velocity is maximum at the onset of the stenosis and achieves its minimal at the throat of the stenosis. It can be observed from figures that velocity decreases as $\frac{\delta}{R_0}$, r increases and decreases with the relative length of the constriction.



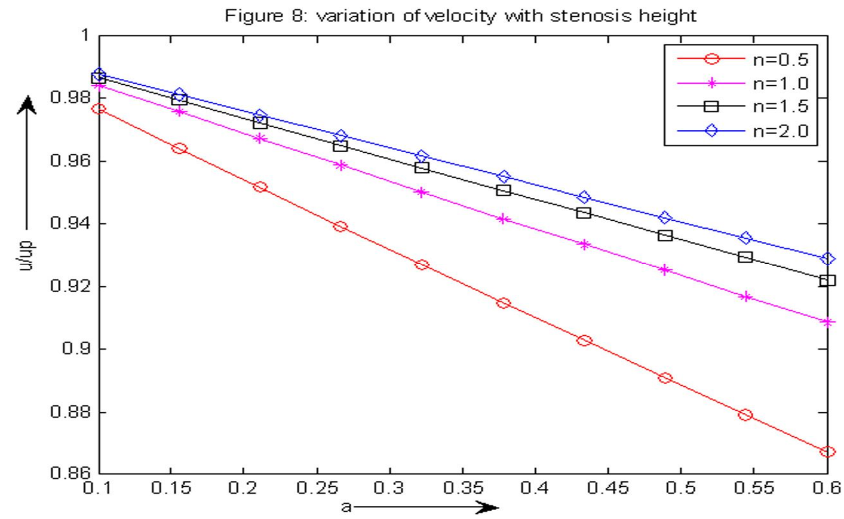


Figure 8: variation of velocity with stenosis height
u/up
a
n=0.5
n=1.0
n=1.5
n=2.0

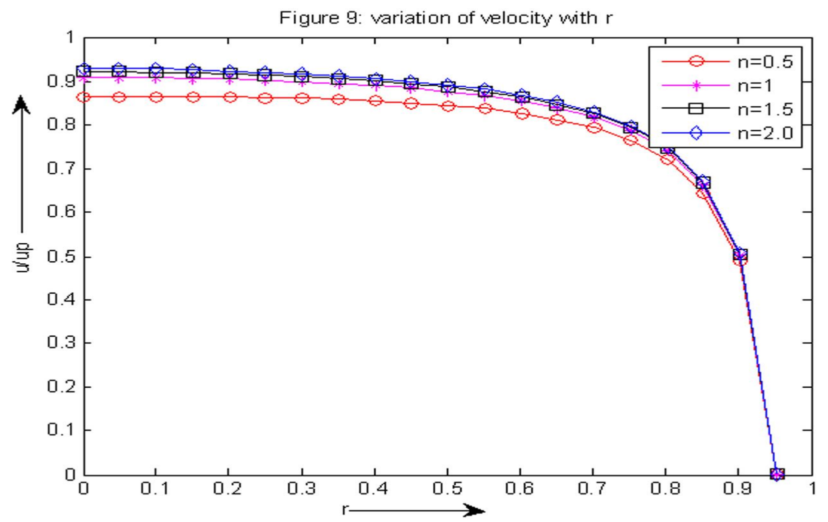


Figure 9: variation of velocity with r
u/up
r
n=0.5
n=1
n=1.5
n=2.0

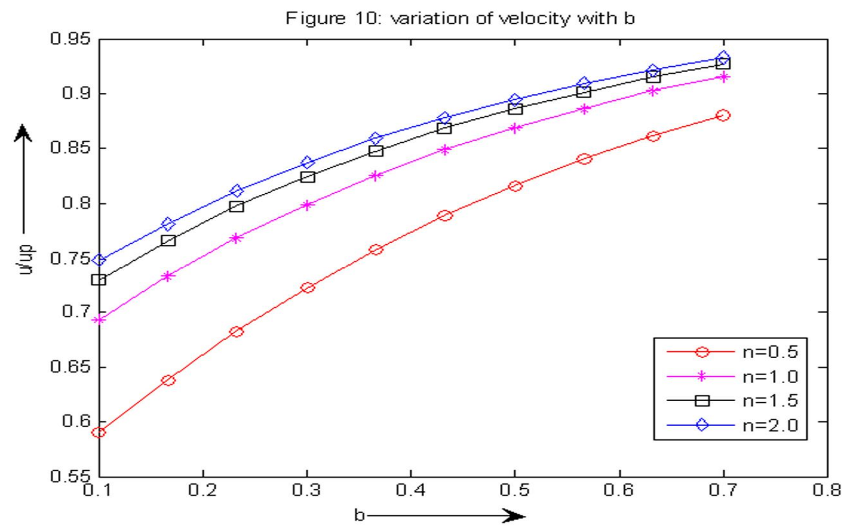


Figure 10: variation of velocity with b
u/up
b
n=0.5
n=1.0
n=1.5
n=2.0

In this paper, we have dealt with the effects of stenosis in an artery by considering the blood as power-law fluid. It has been concluded that the pressure drop and shear stress increases as the size of the stenosis increases for a given non-Newtonian model of the blood.

This analysis bears the potential to examine under the purview of a single study the results for several models, viz. pseudo-plastic power-law fluid model ($n<1$), dilatant power-law fluid model ($n>1$) and the Newtonian viscous model ($n=1$). So this study is more useful for the purpose of simulation and validation of different models in different conditions of atherosclerosis.

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