

Two-layered Blood Flow through a Composite Stenosis in the Presence of a Magnetic Field.

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ABSTRACT

Blood flow through a composite stenosis in the presence of a magnetic field was investigated by examining the effects that an external, uniform, transverse magnetic field has on its velocity, flow rate and three flow characteristics (impedance, wall shear stress and shear stress at the stenosis throat). A two-layered model, consisting of a cell-free peripheral plasma layer and a particle-fluid suspension in the core region, was used to represent blood flowing through a composite stenosis in an artery. The effects of introducing the magnetic field, as well as increasing its intensity were examined. The fluid's velocity and flow rate were reduced when the magnetic field was introduced as well as when its intensity was increased. All three of the flow characteristics increased when the magnetic field was introduced and as its intensity increased. These effects make it more difficult for the blood to flow and may even lead to wall vessel damage and plaque rupture. When hematocrit, stenosis height and tube length were varied the same trends were observed in the presence of the external magnetic field as those observed in the absence of the magnetic field. The two-fluid model flow characteristics were lower than those of the one-fluid model. This knowledge can aid in the improvement of existing diagnostic tools, especially those used in diagnosing cardiovascular diseases.

Keywords: blood flow, stenosis, magnetic field, impedance, shear stress

1. INTRODUCTION

According to the World Health Organisation (WHO), an estimated 17 million deaths that occur annually are due to cardiovascular related complications. The 23.3 million deaths predicted by 2030 ensure that cardiovascular diseases will remain the single leading cause of death worldwide and a popular subject of scientific research [29]. The two main causes of the associated deaths are due to coronary heart disease (disease of the blood vessels supplying the heart muscle) and cerebrovascular disease (disease of the blood vessels supplying the brain). A heart attack or a stroke usually follows when blood flow to the heart or the brain respectively is blocked. Experimental and theoretical blood flow studies which include magnetic effects have been useful in the design of medical devices that are used in diagnosing cardiovascular diseases.

In several situations, it may be necessary to subject the human body to magnetic fields although the full implications of its effects on biological fluid dynamics are still being researched. Magnetic Resonance Imaging (MRI) is a tool that is frequently used in the field of medicine to study blood flow. Low intensity, low frequency magnetic energy has been used to treat chronic pain secondary to tissue ischemia and non healing or slowly healing ulcers. This energy appeared to affect biological processes due to its electrically induced charges. New types of cellular phones, levitated trains and superconducting magnetic energy storage have been designed to utilise electro-magnetic frequencies [26]. Other devices that contain magnetic fields have already been used in cell separation, targeted drug transport carriers, cancer tumour treatment, the reduction of bleeding in surgeries and injuries at accidents and wars, magnetic band aids and tracers [28]. Further studies on the effects of an external magnetic field on blood flow will allow greater flexibility for future designs of such devices.

In medical research, the effect of electro-magnetic fields was first investigated by Kolin [10]. Such studies involving the flow through a channel in the presence of a transverse magnetic field are encountered in a variety of applications such as magnetohydrodynamics (MHD) generators, pumps, accelerators and flow meters. The application of an external magnetic field was then seen as a means to aid in the regulation of the movement of blood in the human circulatory system. Since blood is electrically conducting, MHD principles were easily applied to its flow. By Lenz's law, the Lorentz's force acting on the blood opposes its motion thereby reducing its velocity. This reduction in velocity proves useful in the treatment of the cardiovascular diseases which involve accelerated blood flow, for example in hypertension and hemorrhages. It is also used to provide analgesia, decrease healing time of fractures and to act as a treatment for depression. Myocardial infarctions could even be triggered by man-made magnetic fields since it was found that magnetic fields exceeding 0.05 T can be dangerous to the heart and may result in irreversible changes. Therefore the majority of devices that utilise magnetic fields contain static magnetic fields rather than electro-magnetic ones. Even static magnetic fields can be dangerous since they are known to cause a change in calcium dynamics and alter skin blood flow [1].

Pioneering blood flow models have treated blood flow as a single phase, homogeneous, Newtonian, viscous fluid [11]. Non Newtonian models for blood flow were also developed since blood, being a suspension of corpuscles, does not always exhibit the characteristics of a Newtonian fluid especially in tubes with small diameters [3]. Many blood flow studies also involved the presence of stenosis (an abnormal growth which occurs under diseased conditions in any location of the cardiovascular system) [30]. Stenosis has been modeled as a single stenosis (symmetric, bell shaped, non-symmetric,

composite) or a multiple stenosis (overlapping, irregularly shaped) and its effect on blood flow characteristics has been examined [11], [20]-[23]. Magnetic effects on single fluid (Newtonian, Casson, Micropolar or Couple Stress fluid) models were also examined [5], [14], [27]. One fluid models examining the effects of MHD on blood flow in stenosed arteries were proposed by Ikbal et al., Singh and Rathee, Misra et al. and Sut [7], [18], [16], [24].

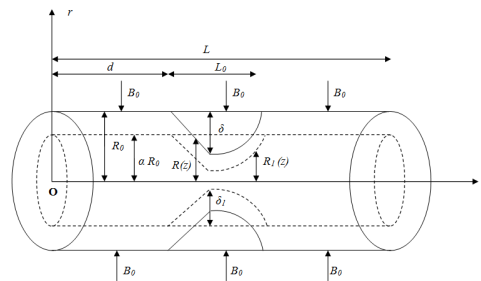
However, based on Haynes' theoretical analysis and Cokelet's experimental observations, a single-phase homogeneous viscous fluid analysis cannot be used to realistically describe blood flow in blood vessels of diameter $\leq 1000 \mu\text{m}$ [4], [6].

Additionally, the discovery that hematocrit (red cell volume fraction) in human blood is approximately 45% and has a strong influence on the blood flow properties of blood has led to the treatment of the red blood cells (erythrocytes) as discrete particles by Skalak [19]. Thus, Srivastava and Srivastava proposed a two-phase macroscopic model (i.e. a suspension of red cells in plasma) for blood in small vessels (of diameter $\leq 2400 \mu\text{m}$) where the individuality of the red cells (of diameter $8 \mu\text{m}$) is significant [20]. Further experimental work provided evidence for the existence of a layer of plasma containing no cells located layer near the walls and a core region containing all the erythrocytes suspended in plasma [2]. Therefore two layered models can be used to describe blood flow in vessels of diameter $\leq 1000 \mu\text{m}$.

To study blood flow in the presence of MHD, two-layered models were proposed [1], [15]. Ponalagusamy and Selvi studied oscillatory blood flow in the presence of a magnetic field through a mild stenosis using a two-layered model where both layers were represented as Newtonian fluids in the presence of a uniform magnetic field using a finite difference method [15]. A more realistic two-layered model for blood flow through small blood vessels consists of the peripheral cell free layer and a core two-phase macroscopic layer. This model has been used to study the effect of stenosis on blood flow characteristics [9], [16], [22], [23]. A magnetic study was conducted by Bali and Awasthi where the red blood cells were treated as magnetic particles to examine the effect this would have on the fluid's velocity, flow rate and effective viscosity but in the absence of stenosis and an external magnetic field [1].

Thus the objective of the present analysis is to provide a model to examine the effect of an external magnetic field on blood flow through a composite stenosis which has not yet been examined in previous literature. This will be done by using a two-layered blood flow model consisting of a cell-free peripheral layer and a core region of erythrocytes in plasma flowing through a composite stenosis in the presence of an external, transverse magnetic field. This two-layered model for blood flow provides a more realistic model for flow in small arteries since the existence of the peripheral layer and the red blood cells in the plasma can no longer be neglected. The study of a composite stenosis is important since the shape of the stenosis which is manifested in the arteries varies. The effect that the external magnetic field has on the fluid's velocity, flow rate and three of its blood flow characteristics, resistance to flow, wall shear stress and shear stress at the stenosis throat will be examined. Knowledge generated in this area can lead to the improvement of existing diagnostic tools for a more effective treatment of patients suffering from cancer, hypertension, myocardial infarction, stroke and paralysis. It can also provide an understanding of the effect that a magnetic field may have on different diseased arterial systems.

2. METHODOLOGY



Application of a uniform, transverse magnetic field in the axial direction

Figure 1 The geometry of the stenosed artery

Consider a two layered model for the flow of blood through a cylindrical vessel which consists of a central core layer of radius R_1 which is a suspension of red blood cells (erythrocytes) in plasma and a peripheral plasma layer of thickness $(R - R_1)$ modeled as a Newtonian, viscous fluid. The blood flow is assumed to be axisymmetrical, fully developed, steady and laminar. The artery is assumed to be uniform, circular and rigid. It is also assumed that a mild, axially nonsymmetric but radially symmetric composite stenosis has manifested in the artery. A uniform, transverse magnetic field is applied in the axial direction. The wall near to the stenosis development is treated as being solid in nature. It is assumed that the length of the artery is much greater than its radius so that entrance, end and special wall effects can be neglected. The stenosis geometry manifested in the arterial segment is described in Figure 1 as [9], [13], [16]

$$\begin{aligned} \frac{(R(z), R_1(z))}{R_0} &= (1, \alpha) - \frac{2}{R_0 L_0} (\delta, \delta_1)(z - d); \quad d \leq z \leq d + \frac{L_0}{2} \\ &= (1, \alpha) - \frac{(\delta, \delta_1)}{2R_0} \left[1 + \cos \frac{2\pi}{L_0} \left(z - d - \frac{L_0}{2} \right) \right]; \quad d + \frac{L_0}{2} \leq z \leq d + L_0 \\ &= (1, \alpha); \quad \text{otherwise.} \end{aligned}$$

where $R \cong R(z)$ is the radius of the tube with constriction and R_0 are the radius of the tube without any constriction. The length of the tube is denoted by L and the length of the stenosis by L_0 . The stenosis starts at $z = d$. In the unobstructed region, the ratio of the radius of the central core to that of the tube is α . The maximum height of the stenosis and bulging of the interface at the location where $z = d + \frac{L_0}{2}$ in the stenotic region is (δ, δ_1) .

In the core region, $(0 \leq r \leq R_1)$, a two-phase macroscopic model is used. The following additional assumptions must be made to simplify the analysis due to the complicated structure of blood [20]:

- (i) the red cell is spherical in shape and rigid,
- (ii) the artery wall is rigid and of infinite length,
- (iii) the flow has rotational symmetry,
- (iv) a modified Stokes drag law governs the interaction between the two phases,
- (v) red cell-cell interaction and Brownian motion is neglected.

Using a continuum approach and these assumptions, the following are the governing equations for the two-phase blood flow in the region $0 \leq r \leq R_1$ in the presence of Lorentz's force.

For the fluid (plasma) phase:

Equation of axial momentum

$$(1 - C)\rho_f \left(\frac{\partial u_f}{\partial t} + u_f \frac{\partial u_f}{\partial z} + v_f \frac{\partial u_f}{\partial r} \right) = -(1 - C) \frac{\partial p}{\partial z} + (1 - C)\mu_s(C)\nabla^2 u_f + CS(u_p - u_f) - \sigma B_0^2 u_f. \quad (1)$$

Equation of radial momentum

$$(1 - C)\rho_f \left(\frac{\partial v_f}{\partial t} + u_f \frac{\partial v_f}{\partial z} + v_f \frac{\partial v_f}{\partial r} \right) = -(1 - C) \frac{\partial p}{\partial r} + (1 - C)\mu_s(C) \left(\nabla^2 - \frac{1}{r^2} \right) v_f + CS(v_p - v_f). \quad (2)$$

Equation of continuity

$$\frac{1}{r} \frac{\partial}{\partial r} \left[r(1 - C)v_f \right] + \frac{\partial}{\partial z} \left[(1 - C)u_f \right] = 0. \quad (3)$$

For the particle (erythrocyte) phase:

Equation of axial momentum

$$C\rho_p \left(\frac{\partial u_p}{\partial t} + u_p \frac{\partial u_p}{\partial z} + v_p \frac{\partial u_p}{\partial r} \right) = -C \frac{\partial p}{\partial z} + CS(u_f - u_p). \quad (4)$$

Equation of radial momentum

$$C\rho_p \left(\frac{\partial v_p}{\partial t} + u_p \frac{\partial v_p}{\partial z} + v_p \frac{\partial v_p}{\partial r} \right) = -C \frac{\partial p}{\partial r} + CS(v_f - v_p). \quad (5)$$

Equation of continuity

$$\frac{1}{r} \frac{\partial}{\partial r} (rCv_p) + \frac{\partial}{\partial z} (Cu_p) = 0. \quad (6)$$

Here (r, z) are two-dimensional cylindrical polar coordinates with z measured along the axis of the tube and r measured normal to the tube axis, $\nabla^2 = \frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial}{\partial r} \right) + \frac{\partial^2}{\partial z^2}$ is a two-dimensional Laplacian operator, (u_f, v_f) and (u_p, v_p) are the (axial, radial) components of the fluid and particle velocities. C denotes the volume fraction density of

the particles, p is the pressure, $\mu_s(C) \cong \mu_s$ is the mixture viscosity (apparent or effective viscosity), S is the drag coefficient of interaction for the force exerted by one phase on the other, ρ_f and ρ_p are the actual densities of the material constituting the fluid (plasma) and the particle (erythrocytes) phases respectively; $(1 - C)\rho_f$ is the fluid phase density and $C\rho_p$ is the particulate phase density, and the subscripts f and p denote the quantities associated with the plasma (fluid) and erythrocyte (particle) phases respectively.

The electrical conductivity of the fluid is σ and B_0 is the component of the constant uniform magnetic field which was applied in the axial direction. The suspension viscosity, μ_s , has been chosen as the following empirical relation [3], [20]

$$\mu_s(C) = \frac{\mu_0}{1 - mC}, \tag{7}$$

where

$$m = (7 \times 10^{-2}) \exp \left[2.49C + \left(\frac{1107}{T} \right) \exp(-1.69C) \right]. \tag{8}$$

Here μ_0 is the fluid viscosity (suspending medium). The temperature of the blood, T , is measured on the absolute scale (K). Charm & Kurland performed experiments to investigate the suspension viscosity using a cone and plate viscometer. A good agreement was found within 10% of the tested cases [3]. It was also found to be reasonably accurate for $C \leq 0.6$ [3], [21]-[23]. The expression for the drag coefficient of interaction, S , from the classical Stokes drag was modified to account for the finite particulate fractional volume is [25]

$$S = 4.5 \left(\frac{\mu_0}{a_0^2} \right) \frac{4 + 3(8C - 3C^2)^{\frac{1}{2}} + 3C}{(2 - 3C)^2} \tag{9}$$

with a_0 as the red blood cell radius.

Using Navier-Stokes equations in the presence of the Lorentz's force, the governing equations in the peripheral region, $R_1 \leq r \leq R$, can be written as

$$\rho_0 \left(\frac{\partial u_0}{\partial t} + u_0 \frac{\partial u_0}{\partial z} + v_0 \frac{\partial u_0}{\partial r} \right) = -\frac{\partial p}{\partial z} + \mu_0 \nabla^2 u_0 - \sigma B_0^2 u_0 \tag{10}$$

$$\rho_0 \left(\frac{\partial v_0}{\partial t} + u_0 \frac{\partial v_0}{\partial z} + v_0 \frac{\partial v_0}{\partial r} \right) = -\frac{\partial p}{\partial r} + \mu_0 \left(\nabla^2 - \frac{1}{r^2} \right) v_0 \tag{11}$$

where (u_0, v_0) are the (axial, radial) components and ρ_0 and the density of the fluid.

The boundary conditions (standard no slip conditions of velocities and the shear stresses at the tube wall and the interface) are given as [13], [16], [21-23]

$$u_0 = 0, \text{ at } r = R,$$

$$u_0 = u_f \text{ and } \tau_p = \tau_f, \text{ at } r = R_1,$$

$$\frac{\partial u_f}{\partial r} = \frac{\partial u_p}{\partial r} = 0 \text{ and } \tau_f = 0, \text{ at } r = 0,$$

where $\tau_p = \mu_0 \frac{\partial u_0}{\partial r}$ and $\tau_f = (1 - C)\mu_s \frac{\partial u_f}{\partial r}$ are the shear stresses of the peripheral and central regions respectively.

It was also assumed that there is no radial flow along the axis of the tube ($v_0 = 0$) and the axial velocity gradient of the

fluid and particle phases of the streaming blood is equal to zero $\left(\text{i.e. } \frac{\partial v_f}{\partial r} = \frac{\partial v_p}{\partial r} = 0 \right)$.

3.RESULT

The following non-dimensional variables are used in this analysis

$$r^* = \frac{r}{R_0}, z^* = \frac{z}{R_0}, d^* = \frac{d}{R_0}, \delta^* = \frac{\delta}{R_0}, L_0^* = \frac{L_0}{R_0}, R_1^* = \frac{R_1}{R_0}, R^* = \frac{R}{R_0}, \mu_s^* = \frac{\mu_s}{\mu_0}, S^* = \frac{S R_0^2}{\mu_0},$$

$$(u_0^*, u_f^*, u_p^*) = \frac{(u_0, u_f, u_p)}{U_0}, (v_0^*, v_f^*, v_p^*) = \frac{(v_0, v_f, v_p)}{U_0}, t^* = \frac{U_0 t}{R_0}, p^* = \frac{p R_0}{U_0 \mu_0}, M^2 = \frac{\sigma B_0^2 R_0^2}{\mu_0}, Re = \frac{\rho U_0 R_0}{\mu_0},$$

where M is the Hartmann number and Re the Reynolds number. After non-dimensionalising all the equations and dropping the “*” notation for simplicity, the following system is obtained

$$\frac{dp}{dz} = \frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial}{\partial r} \right) u_0 - M^2 u_0, \quad R_1 \leq r \leq R, \tag{12}$$

$$(1-C) \frac{dp}{dz} = (1-C) \frac{\mu_s(C)}{r} \frac{\partial}{\partial r} \left(r \frac{\partial}{\partial r} \right) u_f + CS(u_p - u_f) - M^2 u_f, \quad 0 \leq r \leq R_1, \tag{13}$$

$$C \frac{dp}{dz} = CS(u_f - u_p), \quad 0 \leq r \leq R_1, \tag{14}$$

subject to

$$u_0 = 0, \quad \text{at } r = R, \tag{15}$$

$$u_0 = u_f \quad \text{and} \quad \frac{\partial u_0}{\partial r} = (1-C) \mu_s \frac{\partial u_f}{\partial r}, \quad \text{at } r = R_1, \tag{16}$$

$$\frac{\partial u_f}{\partial r} = \frac{\partial u_p}{\partial r} = 0, \quad \text{at } r = 0, \tag{17}$$

in the presence of a composite stenosis given by

$$(R(z), R_1(z)) = (1, \alpha) - \frac{2}{L_0} (\delta, \delta_1)(z - d); \quad d \leq z \leq d + \frac{L_0}{2} \tag{18}$$

$$= (1, \alpha) - \frac{(\delta, \delta_1)}{2} \left[1 + \cos \frac{2\pi}{L_0} \left(z - d - \frac{L_0}{2} \right) \right]; \quad d + \frac{L_0}{2} \leq z \leq d + L_0 \tag{19}$$

$$= (1, \alpha); \quad \text{otherwise.} \tag{20}$$

The expressions for velocities, u_0, u_f and u_p are found to be

$$u_0 = k_1 I_0(Mr) + k_2 K_0(Mr) - \frac{1}{M^2} \left(\frac{dp}{dz} \right), \quad R_1 \leq r \leq R, \tag{21}$$

$$u_f = k_3 J_0(M \gamma r) - \frac{1}{M^2} \left(\frac{dp}{dz} \right), \quad 0 \leq r \leq R_1, \tag{22}$$

$$u_p = k_3 J_0(M \gamma r) - \left(\frac{1}{M^2} + \frac{1}{S} \right) \left(\frac{dp}{dz} \right), \quad 0 \leq r \leq R_1, \tag{23}$$

where

$$k_1 = \frac{\frac{1}{M^2} \left(\frac{dp}{dz} \right) - k_2 K_0(MR)}{I_0(MR)},$$

$$k_2 = \frac{\frac{1}{M^2} \left(\frac{dp}{dz} \right) \left[I_0(MR_1)(1-C)\mu_s \gamma J_1(M\gamma R_1) + I_1(MR_1)J_0(M\gamma R_1) \right]}{\left\{ I_0(MR)[J_0(M\gamma R_1)K_1(MR_1) - K_0(MR_1)(1-C)\mu_s \gamma J_1(M\gamma R_1)] \right. \\ \left. + K_0(MR)[I_0(MR_1)(1-C)\mu_s \gamma J_1(M\gamma R_1) + I_1(MR_1)J_0(M\gamma R_1)] \right\}}$$

$$k_3 = \frac{k_2 K_1(MR_1) - k_1 I_1(MR_1)}{(1-C)\mu_s \gamma J_1(M\gamma R_1)},$$

and

$$\gamma = \sqrt{\frac{1}{(C-1)\mu_s}}$$

where J_n is the Bessel functions of first kind of order n , I_n is the modified Bessel function of the first kind of order n , and K_n is the modified Bessel function of the second kind of order n .

The flow flux, Q , is now calculated as

$$Q = 2\pi \left\{ \int_{R_1}^R ru_0 dr + \int_0^{R_1} r[(1-C)u_f + Cu_p] dr \right\},$$

which simplifies to

$$Q = 2\pi \left\{ \frac{k_1}{M} (RI_1(MR) - R_1 I_1(MR_1)) + \frac{k_2}{M} (R_1 K_1(MR_1) - RK_1(MR)) \right. \\ \left. + \frac{k_3}{M\gamma} (R_1 J_1(M\gamma R_1) - \left(\frac{dp}{dz} \right) \left(\frac{R^2}{2M^2} + \frac{CR_1^2}{2S} \right)) \right\} \tag{24}$$

Since the sum of the fluxes in the two layers is equal to the total flux, it follows that $R_1 = \alpha R$ and $\delta_1 = \alpha \delta$ [23]. Thus the pressure drop $\Delta p (= p$ at $z = 0, -p$ at $z = L)$ across the stenosis is calculated as

$$\Delta p = \int_0^L \left(-\frac{dp}{dz} \right) dz$$

$$\Delta p = \int_0^d \left(-\frac{dp}{dz} \right) R \text{ from Eq.(20)} dz + \int_{\frac{d}{2}}^{\frac{d+L_0}{2}} \left(-\frac{dp}{dz} \right) R \text{ from Eq.(18)} dz$$

$$+ \int_{\frac{d+L_0}{2}}^{d+L_0} \left(-\frac{dp}{dz} \right) R \text{ from Eq.(19)} dz + \int_{d+L_0}^L \left(-\frac{dp}{dz} \right) R \text{ from Eq.(20)} dz \tag{25}$$

where $\left(-\frac{dp}{dz} \right)$ is obtained from Eq. (24).

To obtain the integrals involved in Eq. (25) in closed form poses some difficulty so they will be evaluated numerically.

Non-dimensionless expressions for the impedance (flow resistance), λ , the wall shear stress, τ_w and the shear stress at the stenosis throat, τ_s are obtained by using

$$\lambda = \frac{\bar{\lambda}}{\lambda_0}, \text{ where } \bar{\lambda} = \frac{\Delta p}{Q} \text{ and } \lambda_0 = \frac{8\mu_0 L}{\pi R_0^4},$$

$$(\tau_w, \tau_s) = \frac{(\overline{\tau_w}, \overline{\tau_s})}{\tau_0}, \text{ where } \overline{\tau_w} = -\left(\frac{R}{2} \frac{dp}{dz} \right) \text{ and } \overline{\tau_s} = \left[-\left(\frac{R}{2} \frac{dp}{dz} \right) \right]_{R=1-\delta} \text{ with } \tau_0 = \frac{4\mu_0}{\pi R_0^3}.$$

Here λ_0 and τ_0 are the impedance and shear stress respectively for a Newtonian fluid in the absence of stenosis and magnetic field (i.e., $C = 0, M = 0$), and $(\bar{\lambda}, \bar{\tau}_w, \bar{\tau}_s)$ are (impedance, wall shear stress and shear stress at the stenosis throat) in their dimensional forms.

4.DISCUSSION/ANALYSIS

Plots of the analytical solutions are provided for the following parameter values: $d = 0; L_0 = 1; L = 1, 2, 5; C = 0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6; \delta = 0, 0.05, 0.10, 0.15, T = 25.5 \text{ }^\circ\text{C}$, to observe the effects of the external magnetic field on flow characteristics [11], [16], [17], [30]. Values of the Hartmann number, $M = 0, 2, 4, 6, 8, 10$ are used in this analysis [18], [26]. The parameter α is computed using $\alpha = 1 - \varepsilon / R_0$ in which $\varepsilon \cong \varepsilon(C)$ represents the peripheral layer of thickness as a function of cell concentration. In order for the computation to be done, the temperature was chosen as $T = 25.5 \text{ }^\circ\text{C}$, to be able to use Haynes' analysis where $\varepsilon(\mu m) = 6.18, 4.67, 3.60, 3.12, 2.58, 2.18$ corresponds to hematocrit (%) = 10, 20, 30, 40, 50, 60 respectively. The parameter μ_s is computed with the use of Eqns. (7) and (8). Simpson's rule is used to evaluate the integral involved in Eq. (25) numerically.

The effect of the external magnetic field on the fluid's velocity, flow rate and three flow characteristics (impedance, wall shear stress, shear stress at the stenosis throat) will be analysed. Knowledge of how the magnetic field affects flow in the presence of stenosis can aid in the understanding angina while knowledge of how it affects wall shear stress and the stress at the stenosis throat can help in the theory of plaque rupture. For abnormal hearts, an increase in wall shear stress may result in paralysis or death. The study of the resistance to flow (impedance) is physiologically important as it is a reflection of whether the amount of blood being supplied to the vital organs is sufficient or not [15]. Thus the study of these hydrodynamic factors is valuable to understanding the development and progression of arterial diseases.

From Figure 2, as the Hartmann number (magnetic parameter) increases, the velocity of the fluid decreases in both the layers of the flow. It can also be seen that in the absence of the external magnetic field ($M = 0$), the fluid's velocity is higher than in its presence ($M > 0$). Therefore, the presence of an external magnetic field reduces blood's velocity and as the intensity of the magnetic field is increased it is further reduced which is in agreement with the work of Bali and Awasthi [2]. This occurs due to the opposing Lorentz's force that is introduced when the magnetic field is applied. Hence magnetic fields can be used to control blood flow.

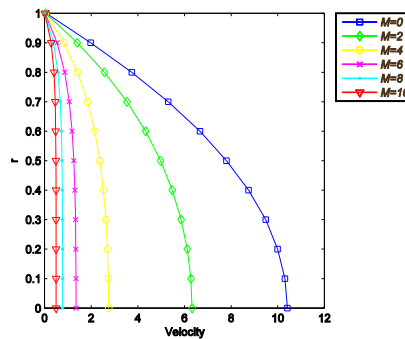


Figure 2 The effect of varying the Hartmann number, M , on the fluid's velocity

The flow rate is higher in the absence of the magnetic field than in its presence (see Figure 3). As the intensity of the magnetic field increases (i.e. M increases) the flow rate decreases. Also generally, as the hematocrit, C , increases, the flow rate decreases both in the absence and presence of the magnetic field [2].

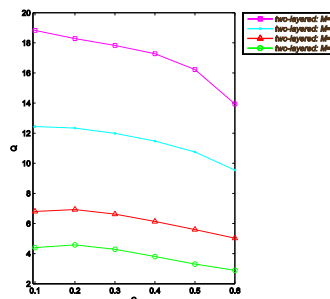


Figure 3 Variation of flow rate with hematocrit, C for various values of the Hartmann number, M

Figure 4 shows that as the Hartmann number, M , increases the impedance to flow, λ , increases and that the presence of the magnetic field causes the impedance to increase. Thus, the presence of the magnetic field increases the resistance to flow and as its intensity is increased, the resistance is further increased. This means it becomes more difficult for the blood to flow through the vessels. Ponalagusamy and Selvi also observed this based on their model [15]. It must also be noted that in the presence of a magnetic field of any intensity, as the hematocrit increases, the impedance increases and as the stenosis height increases, the impedance increases. This means that as the number of red blood cells increases and the larger the stenosis gets, the harder it is for the blood to flow in the vessels which was also observed in the absence of the magnetic field by Sankar et al. [16].

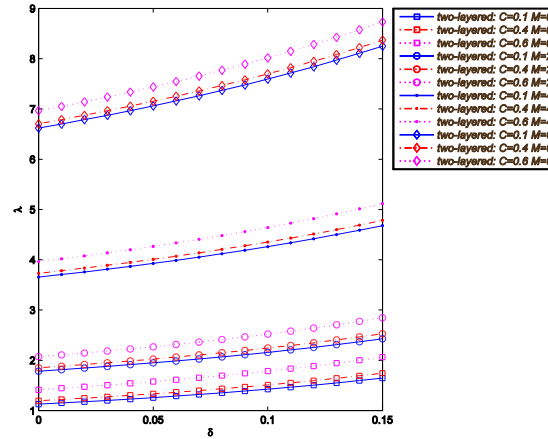


Figure 4 Impedance versus stenosis height for different M and C values

From Figure 5, in the presence of the external magnetic field ($M = 2$), as the artery length increases impedance to flow, λ , decreases. Therefore the longer the artery is without any branching or curving, the easier it is for blood to flow in that region even in the presence of a magnetic field. Also, the two layered model's flow impedance was slightly lower than in the one-fluid model.

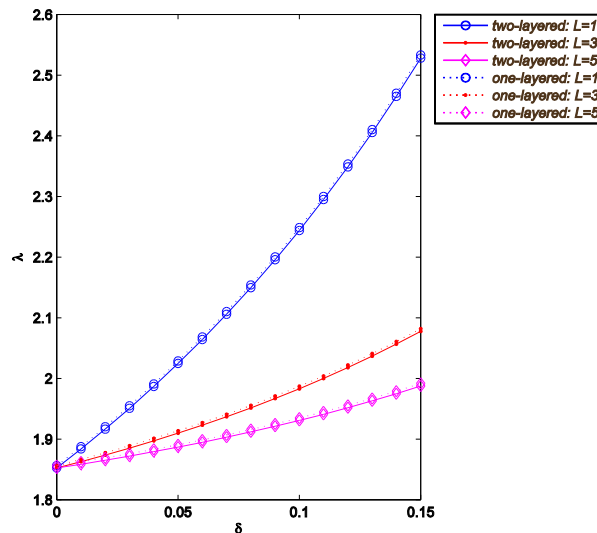


Figure 5 Impedance versus stenosis height for different artery lengths (with $M = 2, C = 0.4$)

The presence of an external magnetic field also affects the wall shear stress. From Figure 6, it is observed that as Hartmann number, M , increases, the wall shear stress increases and that the wall shear stress is higher in the presence of the magnetic field than in its absence. Therefore the presence of the external magnetic field causes a greater force to be exerted on the vessel wall and as the intensity of the field is increased the force also increases which in the long term can damage the walls [5]. Additionally, in the presence of the magnetic field, as the hematocrit, C , increases, the wall shear stress increases which can lead to further damage.

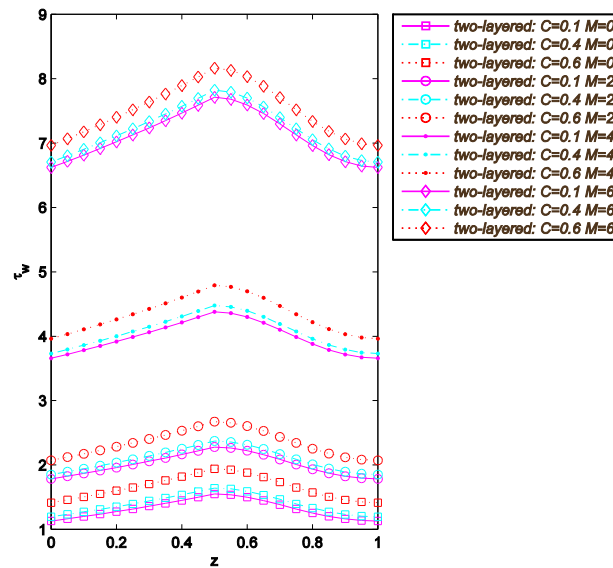


Figure 6 Wall shear stress distribution, τ_w , in the stenotic region for different M and C values

As the stenosis height, δ , increases the shear stress on the wall increases in the presence of the magnetic field (Figure 7). Therefore the larger the stenosis gets, the greater the force is exerted on the wall as it increases rapidly in the upstream of the stenosis and achieves its maximum at the throat of the stenosis ($z = d + \frac{L_0}{2}$) before it begins to decrease downstream to the endpoint of the constriction profile ($z = L_0$). This trend was also observed in the absence of the magnetic field [16]. The two-layered model's wall shear stress was slightly lower than in the one-fluid model.

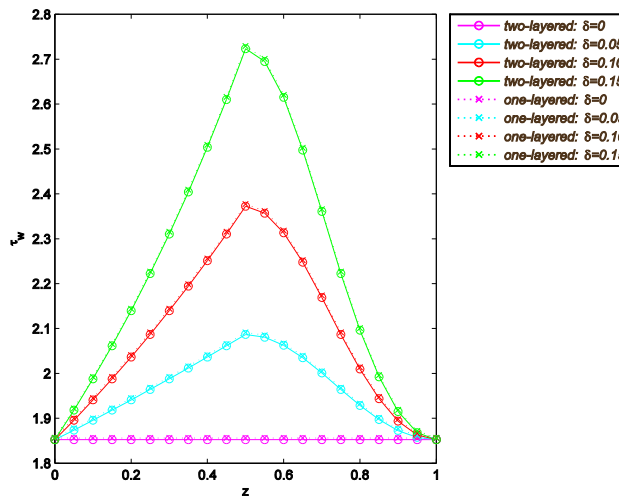


Figure 7 Wall shear stress distribution, τ_w , in the stenotic region for different stenosis heights, δ (with $M = 2, C = 0.4$)

From Figure 8, as the Hartmann number, M , is increased the shear stress at the stenosis throat increases and the shear stress is higher in the presence of the magnetic field than in its absence ($M = 0$). Thus exposure to an external magnetic field increases the shear stress at the stenosis throat and as the intensity increases the shear stress is further increased which may lead to plaque rupture [14]. Also in the presence of the magnetic field as the hematocrit, C , increases the shear stress at the stenosis throat also increases which can further contribute to the possibility of plaque rupture.

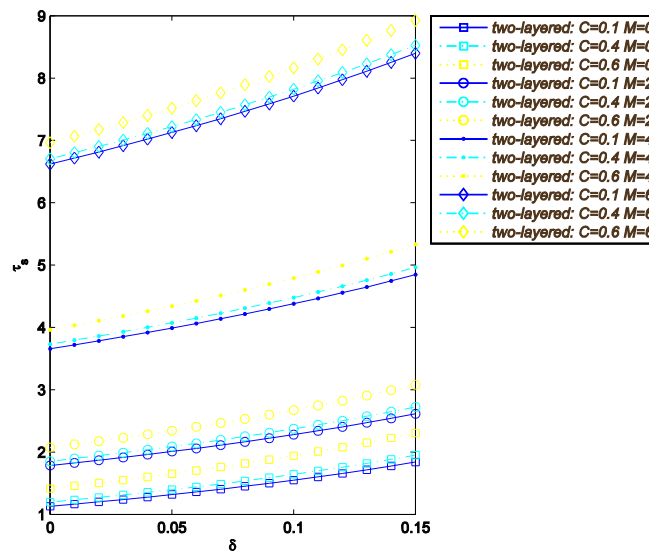


Figure 8 Shear stress at the stenosis throat, τ_s , versus stenosis height, δ for different M and C values

In the presence of the external magnetic field, as the stenosis height increases, the shear stress at the stenosis throat also increases and that the two layered model's shear stress at the stenosis throat was slightly lower than in the one-fluid model (see Figure 9). Thus there is an increased need for the consideration of the peripheral layer (a two-layered flow) when examining blood flow through small blood vessels for different diseases that occur such as plasma cell dyscrasias (hematocrit=28.00%, $\alpha = 0.816$), Hb SS-sickle cell (hematocrit=24.80%, $\alpha = 0.795$), hypertension-controlled (hematocrit=43.13%, $\alpha = 0.928$), hypertension-uncontrolled (hematocrit=43.25%, $\alpha = 0.925$) and polycythemia (hematocrit=63.20%, $\alpha = 0.990$) [2].

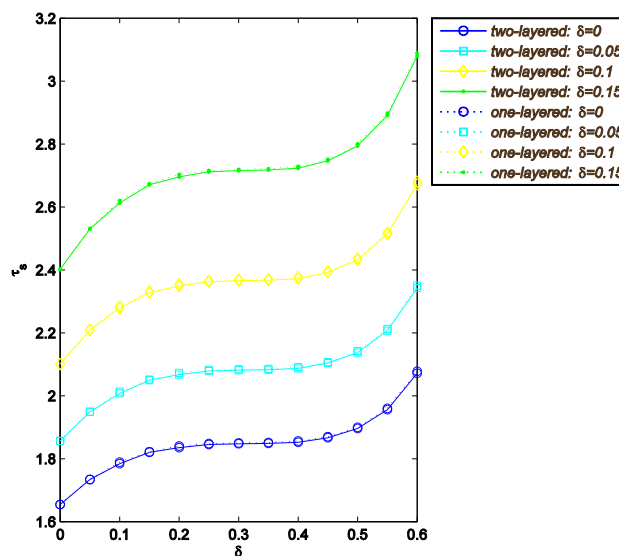


Figure 9 Shear stress at the stenosis throat, τ_s , versus stenosis height, δ (with $M = 2$)

5. CONCLUSION

In this analysis of blood flow in small vessels in the presence of a magnetic field where a composite stenosis is manifested, a two layered model is used to model blood. The two layers are a Newtonian peripheral plasma layer and a core region of erythrocytes suspended in plasma (i.e., particle-fluid mixture). The fluid's velocity, flow rate and three of its flow characteristics are examined.

Both the fluid's velocity and flow rate are reduced as the magnetic field is introduced and as its intensity is increased. This explains why magnetic fields can be used as a control mechanism for blood flow. The impedance to flow is found to increase as the Hartmann number (magnetic parameter), M , is introduced and as it increases. Thus in the presence of the magnetic field it is more difficult for the blood to flow through the vessels. This effect is accentuated as the intensity of the field increases. Both the wall shear stress and shear stress at the stenosis throat increases as Hartmann number, M , is introduced and as the intensity of the field is increased. This has serious implications because it can cause damage to the vessel walls and even lead to plaque rupture.

Flow impedance is found to increase with hematocrit and stenosis height but it decreases with tube length. Therefore in the presence of the external magnetic field, it is harder for the blood to flow if the number of red blood cells increases, if the stenosis increases in size radially or if a very short blood vessel is being considered. The shear stress on the wall increases rapidly in the upstream of the stenosis throat, achieves its maximum at the throat of the stenosis, then decreases downstream. It increases as stenosis height and hematocrit increases, that is, in the presence of a magnetic field, if the number of red blood cells increases or the stenosis increases in size radially, the wall stress gets larger, which can lead to it becoming damaged. In the presence of the magnetic field, variations in the shear stress at the stenosis throat and impedance are similar with respect to all parameters. All three flow characteristics are lower in the two fluid model than those of the one-fluid model. Therefore as a physiological application to the analysis, the peripheral layer becomes particularly important in the study of diseased vessels since the thickness of the peripheral layer (and hematocrit in the blood) varies for different diseases. Some of the popular diseases which affect hematocrit and peripheral plasma layer thickness of the blood when they occur are plasma cell dyscrasias, Hb SS-sickle cell, hypertension and polycythemia.

In conclusion, low intensity magnetic fields can be beneficial because they reduce the blood's velocity. As the intensity increases however, it can lead to fatal effects such as blood vessel wall damage and plaque rupture. The restrictions present in this study (including the rigid wall, steady and fully developed flow and constant thickness of the peripheral layer) do not prevent it from being useful in understanding the flow phenomena. By considering a fully developed flow with $\delta/R_0 \ll 1$, closed form solutions are obtained but this can only be applied to early vessel constriction (mild, composite stenosis). These solutions are used to find the relationships that the flow characteristics have with the variables which are indeed valid even though the parameter δ/R_0 is limited to values up to 0.15 due to the possible flow separation that can occur even at small Reynolds numbers [30]. Studies on two layered flows are not only important to understanding physiological flows (blood flow, protein diffusion, microorganisms movement, particle deposition on the respiratory tract) but also to other areas of research such as powder technology, petroleum transport, aerosol filtration, wastewater treatment, fluidization, mining, power plant piping, corrosive particles in engine oil flow, environmental pollution, lunar ash flows, atmospheric fallout, combustion, agriculture and food technologies [8], [12]. These studies involving the effect of the magnetic field on blood flow in stenosed arteries can lead to the improvement of existing diagnostic tools which contain magnetic fields. The results obtained without considering the presence of a peripheral layer, stenosis and an external magnetic field, can be used to analyse a mixture of particles in a fluid within a circular cylinder in any physical situation.

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