2.1 INTRODUCTION

Blood flow under normal physiological conditions is an important field of study, as is blood flow under disease condition. The majority of deaths in developed countries result from cardiovascular diseases, most of which are associated with some form of abnormal blood flow in arteries. Globally, physicians are of the same opinion that there are three major features of the vascular disease that potentially need treatment (i) vasospasm (spasm of blood vessels), (ii) a proliferative vasculopathy (thickening of blood vessels), and (iii) thrombosis (blood clots) or structural occlusion of the vessel lumen (blockage of blood). Blood clotting is the body’s natural defense against bleeding. A clot, or “thrombus”, develops whenever there is damage to a blood carrying vessel. The platelets and proteins in the blood work together to regulate the clotting process. If the process does not work correctly, a clot can form in the blood vessels.

Several authors reported that thrombus superimposed on ruptured atherosclerotic plaque is commonly found in autopsy studies of heart disease (Davies and Thomas, 1984; 1985; Davies, 1990). Constantinides (1990) discussed the cause of thrombosis

Figure 2.1
(Source:http://upload.wikimedia.org/wikipedia/commons/6/6d/Blausen0259CoronaryArteryDisease02)
in human atherosclerotic artery and reported that thrombosis is also associated with
carotid artery plaque rupture in stroke and transient ischemic attack.

The flow in porous medium spreads a wide application in transport of
macromolecules in aortic media, interstitial fluid flow in axisymmetric soft
connective tissue, thermal therapy, blood flow through contracting muscles, heat
transfer in muscle and skin tissue. Biological tissues contain dispersed cells separately
by voids. Blood enter these tissues through vessels called arteries and perfuse to the
tissue cells via blood capillaries, returned blood from the capillaries is accumulated in
veins where the blood is pumped back to the heart. Porous medium is defined as a
material volume consisting of solid matrix with an interconnected void. It is specially
characterized as ratio of void volume to the total volume of the medium. It is also
specified by its permeability which is a measure of the flow of conductivity in the
porous medium.

Flow through porous medium has been studied by a number of workers. Dash et al.
(1996) showed that in some pathological situations, the distribution of fatty
cholesterol and artery-clogging blood clots in the lumen of the coronary artery can be
considered as equivalent to a fictitious porous medium. David et al. (2001) has
developed a species transport model of platelet accumulation which included
mechanisms of convection, shear-enhanced diffusion, near-wall platelet concentration
and a kinetic model of platelet activation and aggregation for an initial quantitative
estimate of the likelihood of occlusive thrombus in individual patients due to plaque
erosion, artery spasm, incomplete angioplasty or plaque rupture. Xu et al. (2010)
considered the blood clot as a porous medium to account for the transport property of
blood flow in the extension of multiscale model by including a detailed submodel of
surface-mediated control of blood coagulation (Xu et al., 2008; 2009). El-Shahed
(2003) studied pulsatile flow of blood through stenosed porous medium in the
presence of periodic body acceleration. El-Shehawey and EL Sebaei (2000) have
studied peristaltic transport in a cylindrical tube through a porous medium.

The study of flow of an electrically conducting fluid has many applications in plasma
studies, astrophysics, geophysics, irrigation engineering and the boundary layer
control in the field of aerodynamics in the past few years, several simple flow
problems associated with classical hydrodynamics have gained new attraction with in
more general context of Magneto-Hydrodynamics (MHD). It is known from
Magneto-Hydrodynamics that when magnetic field is applied to a moving electrically conducting fluid, electrical currents are induced in the field. The interaction between induced currents and applied magnetic field produces Lorentz force as an external force which retards the blood flow. Magnetic fields of moderate to high intensity can be experienced by human body. The red blood cell (RBC) is a major bio magnetic substance and the blood flow may be influenced by the magnetic field. In general, biological systems are affected by an application external magnetic field on blood flow, through human arterial system. The presence of the stationary magnetic field contributes to an increase in the friction of flowing blood, this is because the anisotropic orientation of RBC in the stationary magnetic field disturbs the rolling of the cells in the flowing blood and thereby the viscosity of blood increases.

Figure 2.1(a)

Kollin (1936) has coined the idea of electromagnetic field in the medical research for the first time in the year 1936. It has been established that the biological systems in general are greatly affected by the application of external magnetic field. As per the investigations reported by Barnothy (1964-1969), the heart rate decreases by exposing biological systems to an external magnetic field. Korchevskii and Marcochnik (1965) have discussed the possibility of regulating the blood movement in human system by applying magnetic field. In the decade of eighties, engineers attracted towards applications of magnetic field in biomedical flow primarily with a view to utilizing
MHD (magnetohydrodynamics) in controlling blood flow velocities in surgical procedures and also establishing the effects of magnetic fields on blood flows in astronauts, citizens living in the vicinity of EM (electromagnetic) towers etc. Keltner et al. (1990) reported an analysis of the pressure changes in vessels of the human vasculature under the action of strong magnetic fields. Their study indicated that 15% Sodium Chloride solutions are retarded by transverse magnetic fields of 2.3 and 4.7 Tesla for fluxes below 0.5 l/min. Several researchers have worked out significant studies on hydromagnetic blood flow in artery (Halder and Ghosh, 1994; McKay et al., 2007; Mekheimer and El Kot, 2008; Rathod and Tanveer, 2009; Tzirtzilakis, 2005). Layek and Mukhopadhyay (2008) and Kumar et al. (2011) worked on a mathematical model to study flow through a variable shape stenosed artery under the influence of magnetic field and demonstrated the effect of stenosis shape and magnetic field on the resistance to the flow.

Several researchers Fung (1984), Mazumdar (1992), McDonald (1960) and Zamir (2005) have given mathematical treatment to the blood flow in arteries subject to various physiological conditions. Young (1968) presented an excellent analysis of flow through an occluded tube under a pulsatile pressure gradient. Mazumdar et al. (1996) studied the time variation of various characteristic of Newtonian flow of blood through a stenosed artery and gave the result on the axial velocity distribution and the pressure gradients of the Newtonian Flow of blood through a constricted circular cylindrical arterial tube for various values of hematocrit and the Womersley parameter and concluded that the pressure gradient attains the maximum at the point of maximum constriction and decreases with increase of hematocrit parameter. Sanyal and Maiti (1998) studied a mathematical model on arterial blood flow in the presence of mild stenosis and obtained the pressure gradient and wall shear stress by series solution. They found that the pressure gradients increases with the increases in hematocrit value which indicates that there is higher value in systolic and lower value in diastolic pressure. Experimental and numerical study carried out by Deplano and Siouffi (1999) for pulsatile flow of blood through stenosis; wall shear analysis and explained the adversity of the stenosis on a healthy artery. Lee and Xu (2002) worked out the results on velocity profiles, Wall Shear Stress intramural strain and stress for the rigid and compliant cases for the mild stenosed tube.
In the present chapter an attempt has been made to deal with the blood flow through an artery filled with porous medium and effects of magnetic field on the hemodynamic in a diseased artery with blood clot and fatty cholesterol which may considered as porous medium.

2.2 FORMULATION OF THE PROBLEM

The present study is to dealt with the flow of blood in diseased artery that is suffer with some deposition in the flow region that we assumed as porous medium. Besides, it is also considered that artery is suffering with stenosis and a static magnetic field is present surrounding the patient.

2.2.1 Blood Rheology

The present mathematical model is modeled with the assumption that blood flow in the tube is a suspension of RBC in plasma. Hematocrit concentration dependent viscosity of the blood is considered that is governed by the Einstein equation

\[ \mu = \mu_0 [1 + \beta h(r)] \]  

where, \( \mu_0 \) the coefficient of viscosity of plasma, \( \beta \) a constant, and \( h(r) \) the hematocrit concentration which vary along the radial direction described by the equation

\[ h(r) = H_m \left[ 1 - \left( \frac{r}{R_0} \right)^n \right] \]  

with \( H_m \) the maximum hematocrit concentration at the axis of tube.

2.2.2 Geometry of the Model

![Figure 2.1(b)](image_url)
The blood vessel geometry is determined by the radius \( R_0 \) of the inlet and outlet unconstricted segment, whereas the radius of the smooth axisymmetric constriction segment is given by

\[
R(z) = \begin{cases} 
R_0 - \frac{L}{2} \left( 1 + \cos \frac{\beta z}{L} \right) & -L \leq z < L \\
R_0 & \text{otherwise}
\end{cases}
\]  

(2.3)

where, \( 2L \) is the length of stenosis and \( \delta \) is maximum thickness of the stenosis. In the cylindrical coordinate system \((r, \beta, z)\) the axis of the vessel coincides with the \( z \)-axis and the origin \( z = 0 \) corresponds to the neck of the stenosis. We are concerned with the static boundary wall, therefore, on the rigid no-slip wall the velocity is vanished. At the inflow section, the radial velocity vanishes and the main flow is assumed to be fully developed. The diameter of the artery is not less than 1mm so that Fahreus-Lindquist effect is not significant. The tube is filled with porous material of constant permeability \( K \).

### 2.2.3 Governing Equations for the Flow Field

In describing the equation of motion, following assumptions were made:

i. There is no cross flow from the vessel’s wall i.e. radial velocity is taken as zero.

ii. The wall of the vessel is taken inelastic in view of the stenosis formation is due to fatty acids, lipoproteins and calcification that hardened the wall surface considerably.

iii. The magnetic field is moderate so that induced magnetic field and electric field are negligible.

iv. The stenosis is axisymmetric.

When an electrically conducting fluid like blood flow in a magnetic field, an electromagnetic force will be produced due to the interaction of current with magnetic field. The electromotive force is proportional to the speed of motion and the magnetic flux intensity \( B \) (Tashtoush and Magableh, (2008)). The Maxwell’s equations are

\[
\text{div} \mathbf{B} = 0
\]  

(2.4)

\[
\text{curl} \mathbf{B} = \nabla \times \mathbf{J}
\]  

(2.5)
\[
curl \mathbf{E} = -\frac{\partial \mathbf{B}}{\partial t} \tag{2.6}
\]

where, \( \mathbf{E} \) is the electric field intensity, \( \mathbf{B} \) is the magnetic flux intensity, \( \varepsilon_m \) is the electric permeability and \( \mathbf{J} \) is the current density. The generalized Ohm’s law

\[
\mathbf{J} = \nabla (\mathbf{E} + \mathbf{V} \times \mathbf{B}) \tag{2.7}
\]

where \( \sigma \) is the electrical conductivity. The induced electromagnetic force \( F^{(em)} \) is defined as

\[
F^{(em)} = \mathbf{J} \times \mathbf{B} = \nabla (\mathbf{E} + \mathbf{V} \times \mathbf{B}) \times \mathbf{B} \tag{2.8}
\]

Following Cowling (1957) and Gupta (1960) that there is no applied or polarization voltage so that \( \mathbf{E} = 0 \). It is assumed that a magnetic field \( \mathbf{B} = (B_0, 0, 0) \) with a constant transverse magnetic flux density \( B_0 \) of moderate strength so that induced magnetic field is negligible. The resultant force, the magnetohydrodynamic force is

\[
F^{(em)} = \mathbf{J} \times \mathbf{B} = -B_0^2 \hat{z} \mathbf{u} \tag{2.9}
\]

Invoking these assumptions, the governing equation of the motion of blood as Newtonian incompressible fluid through a porous medium with axisymmetric condition is given by

\[
\frac{\partial \mathbf{u}}{\partial T} + \frac{\partial \mathbf{u}}{\partial r} \left( \frac{\partial \mathbf{u}}{\partial r} \right) = \mathbf{B}_0^2 \mathbf{u} - \nabla K \mathbf{u} \tag{2.10}
\]

The boundary conditions are

\[
\mathbf{u} = 0 \quad \text{at} \quad r = R(z) \tag{2.11}
\]

\[
\frac{\partial \mathbf{u}}{\partial r} = 0 \quad \text{at} \quad r = 0 \tag{2.12}
\]

Taking unconstructed radius \( R_0 \) and \( t_0 \) as length and time scaling parameter respectively, the governing equation reduced to

\[
\frac{1}{t_0} \frac{\partial \mathbf{u}}{\partial t} = -\frac{1}{y R_0^2} \frac{\partial \mathbf{u}}{\partial y} \left[ \nabla \left( 1 + \mathbf{H}_m (1 - y^n) \right) y R_0 \right] - \frac{\mathbf{B}_0^2 \mathbf{u}}{K} - \frac{\left[ \nabla \left( 1 + \mathbf{H}_m (1 - y^n) \right) \right] \mathbf{u}}{K} \tag{2.13}
\]

\[
= -\frac{1}{y R_0^2} \frac{\partial \mathbf{u}}{\partial y} \left[ (1 + \mathbf{H}_m \mathbf{H}_m y^n) y R_0 \frac{\partial \mathbf{u}}{\partial y} \right] - \frac{\mathbf{B}_0^2 \mathbf{u}}{K} - \frac{\left[ \nabla \left( 1 + \mathbf{H}_m (1 - y^n) \right) \right] \mathbf{u}}{K} \tag{2.13}
\]
\[
\frac{1}{\mu_0 \rho R_0^2} \frac{\partial p}{\partial z} + \frac{\Box}{\Box R_0^2} \frac{\partial}{\partial y} \left[ (1 + k - ky^\prime) y \frac{\partial u}{\partial y} \right] - \frac{\Box^2 u}{\Box} - \frac{\Box(1 + k - ky^\prime)}{K \Box} u
\]

\[
= \frac{1}{\mu_0 \rho R_0^2} \frac{\partial p}{\partial z} + \left( \frac{\Box}{\Box R_0^2} \right) \frac{1}{y} \frac{\partial}{\partial y} \left[ (a - ky^\prime) y \frac{\partial u}{\partial y} \right] - \frac{\Box^2 u}{\Box} - \frac{\Box(a - ky^\prime)}{K \Box} u
\]

dividing by \( \frac{\mu_0}{\rho R_0^2} \)

\[
\frac{R_0^2}{t_0} \frac{\partial u}{\partial t} = - \frac{R_0^2}{\Box} \frac{\partial p}{\partial z} + \frac{1}{y} \frac{\partial}{\partial y} \left[ (a - ky^\prime) y \frac{\partial u}{\partial y} \right] - \frac{R_0^2 \Box^2 u}{\Box} - \frac{(a - ky^\prime)}{Da^2} u
\]

(2.13)

where, \( y = \frac{r}{R_0} \), \( t = \frac{T}{t_0} \), \( \Box \alpha_m = k \) and \( a = l + k \)

The driving force for the motion of blood in the cardiovascular system is a local pressure gradient along the longitudinal direction of the vessel, which in turn is determined by the propagation of the heart pressure pulse. The blood pumped by the heart is of periodic nature, that is pulsating, therefore pressure is periodic, can be expressed in Fourier series (Burton, 1996; Chakravarty and Sen., 2005; Janadzadegan et al., 2009; McDonald, 1960). Therefore, for the sake of simplicity, it is assumed that the pressure gradient is known as a function of time. Taking

\[
- \frac{R_0^2}{\Box} \frac{\partial p}{\partial z} = c e^{i \omega t}
\]

(2.14)

where, \( \omega = 2 \pi f \), \( f \) is the heart pulse frequency and \( c \) is the amplitude of the pulsatile flow.

Also, Taking \( u(y, t) = U(y) e^{i \omega t} \).

(2.15)

Then we have from (2.13)

\[
\frac{1}{y} \frac{d}{dy} \left[ (a - ky^\prime) y \frac{dU}{dy} \right] - (\Box i + H^2 + \frac{a}{Da^2}) U + \frac{ky^\prime}{Da^2} U = -c
\]

(2.16)

where, \( \frac{R_0^2 \Box^2 B_0^2}{\mu_0} = H^2 \); \( \frac{K}{R_0^2} = Da^2 \); \( \alpha^2 = \frac{\rho R_0^2}{t_0 \mu_0} \) are dimensionless parameters,

\( H \) the Hartmann number, \( Da \) the Darcy number and \( \alpha \) the Womersley number.

The corresponding boundary conditions (2.11) and (2.12) are transformed to
\[ U = 0 \quad \text{at} \quad y = \frac{R(z)}{R_0} \quad (2.17) \]

\[ \frac{dU}{dy} = 0 \quad \text{at} \quad y = 0 \quad (2.18) \]

### 2.3 METHOD OF SOLUTION

#### 2.3.1 Calculation for Velocity Profiles

For the solution of differential equation (2.16) we have used the Frobenius method. For implementing, it is required that \( U \) is bounded at \( y = 0 \). The only admissible solution satisfying the boundary condition (2.18) is

\[ U = D \sum_{m=0}^{\infty} A_m y^m - \frac{c}{4a} \sum_{m=0}^{\infty} \lambda_m y^{m+2} \quad (2.19) \]

Here, the second term of the right hand side is the solution corresponding to non-homogenous part of the equation (2.16) and \( A_m \) and \( \lambda_m \) are the series constant, \( D \) is an arbitrary constant to be determined by the boundary condition (2.17).

Firstly, we find the solution of homogenous part of (2.16) with

\[ U = D \sum_{m=0}^{\infty} A_m y^m \quad (2.20) \]

\[ \frac{dU}{dy} = D \sum_{m=1}^{\infty} A_m m y^{m-1} \quad (2.21) \]

\[ \frac{d^2U}{dy^2} = D \sum_{m=2}^{\infty} A_m m(m+1) y^{m-2} \quad (2.22) \]

Clubbing these with the homogenous part of the equation (2.16) we get

\[ \frac{1}{y} \frac{d}{dy} \left[ (a - ky^n) y \frac{dU}{dy} \right] - \left( \frac{\sqrt{2}}{Da} i + H^2 + \frac{a}{Da^2} \right) U + \frac{ky^n}{Da^2} U = 0 \]

\[ \frac{d}{dy} \left[ (a - ky^n) y \frac{dU}{dy} \right] - \left( \frac{\sqrt{2}}{Da} i + H^2 + \frac{a}{Da^2} \right) U y + \frac{ky^{n+1}}{Da^2} U = 0 \]

\[ \left[ (a - ky^n) y \frac{d^2U}{dy^2} \right] + \left[ (a - ky^n) \frac{dU}{dy} \right] + \left[ y(-kny^{n-1}) \frac{dU}{dy} \right] \]

\[- \left( \frac{\sqrt{2}}{Da} i + H^2 + \frac{a}{Da^2} \right) U y + \frac{ky^{n+1}}{Da^2} U = 0 \]
\[
\left[ (a - ky^n) \frac{d^2 U}{dy^2} \right] + \left[ (a - ky^n) \frac{dU}{dy} \right] - \left[ kny^n \frac{dU}{dy} \right] - \left( \square i + H^2 + \frac{a}{Da^2} \right) U = 0
\]

\[
[ay - ky^{n+1}] D \sum_{m=2}^{\infty} A_m m(m+1) y^{m-2} + [(a - ky^n - kny^n)] D \sum_{m=1}^{\infty} A_m my^{m-1} + \\
- \left[ \left( \square i + H^2 + \frac{a}{Da^2} \right) y + \frac{ky^{n+1}}{Da^2} \right] D \sum_{m=0}^{\infty} A_m y^m = 0
\]

Comparing the coefficient of \( y^m \), we have

\[
A_{m+1} = \frac{(\square^2 i + H^2 + \frac{a}{Da^2}) A_{m-1} + k(m+1)(m-n+1) A_{m-n+1} - \frac{k}{Da^2} A_{m-n-1}}{a(m+1)^2}
\]

For the solution of non-homogenous part, let

\[
U = -\frac{c}{4a} \sum_{m=0}^{\infty} \square y^{m+2}
\]

\[
\frac{dU}{dy} = -\frac{c}{4a} \sum_{m=1}^{\infty} \square(m+2) y^{m+1}
\]

\[
\frac{d^2 U}{dy^2} = -\frac{c}{4a} \sum_{m=2}^{\infty} \square (m+2)(m+1) y^m
\]

The equation (2.16) gives,

\[
\Rightarrow \left[ ay - ky^{n+1} \right] D \sum_{m=2}^{\infty} A_m m(m+1) y^{m-2} + \left[ (a - ky^n - kny^n) \right] D \sum_{m=1}^{\infty} A_m my^{m-1} + \\
- \left[ \left( \square i + H^2 + \frac{a}{Da^2} \right) y + \frac{ky^{n+1}}{Da^2} \right] D \sum_{m=0}^{\infty} A_m y^m = 0
\]

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\[
\left[ (a - ky^n) y \right] \sum_{m=2}^{\infty} \Box_m (m+2)(m+1) y^m + \left[ a - k y^n - k n y^n \right] \sum_{m=1}^{\infty} \Box_m (m+2) m y^{m+1} - (\Box^2 i + H^2 + \frac{a}{D a^2}) y \sum_{m=0}^{\infty} \Box_m y^{m+2} + \frac{k y^{n+1}}{D a^2} \sum_{m=2}^{\infty} \Box_m y^{m+2} = 4ay
\]

Comparing the coefficient of \( y^{m+2} \) we have

\[
\lambda_{m+1} = \frac{(\Box^2 i + H^2 + \frac{a}{D a^2}) \Box_{m-1} + k(m+3)(m-n+3) \Box_{-m+1} - \frac{k}{D a^2} \Box_{-m+1}}{a(m+3)^2}
\]

(2.28)

with \( A_0 = \lambda_0 = 1 \) and \( A_m = \lambda_m = 0 \)

The constant \( D \) involved in the solution (2.19) is obtained with the help of boundary condition (2.17) i.e.

\[
U = 0 \text{ at } y = \frac{R(z)}{R_0}
\]

We have, \( 0 = D \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m - \frac{c}{4a} \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+2} \)

and \( D \) is given by

\[
D = \frac{c}{4a} \sum_{m=0}^{\infty} \Box_m \left( \frac{R(z)}{R_0} \right)^{m+2}
\]

(2.29)

then,

\[
U(y) = \frac{c}{4a} \left[ \sum_{m=0}^{\infty} \Box_m \left( \frac{R(z)}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} A_m y^m - \sum_{m=0}^{\infty} A_m \left( \frac{R(z)}{R_0} \right)^m \sum_{m=0}^{\infty} \Box_m y^{m+2} \right]
\]

(2.30)

and \( u(y, t) = U(y) e^{i\omega t} \)
\[ u(y, t) = \frac{c}{4a} e^{i\xi b} \left[ \sum_{m=0}^{\infty} \sum_{n=0}^{\infty} \left( \frac{R(z)}{R_0} \right)^{n+2} \sum_{m=0}^{\infty} A_m y^m - \sum_{m=0}^{\infty} A_m \left( \frac{R(z)}{R_0} \right)^{n+2} \sum_{m=0}^{\infty} \lambda_m y^{m+2} \right] \]  

(2.31)

In particular, in the absence of the hematocrit, the average velocity \( u_0 \) is given by \( u_0 \)

\[ \frac{c_0 e^{i\xi b}}{\left( i + \frac{1}{Da^2} \right)} \left[ 1 - \frac{I_0(\sqrt{i + \frac{1}{Da^2} y})}{I_0(\sqrt{i + \frac{1}{Da^2}})} \right] \]  

(2.32)

The dimensionless form of \( u(y, t) \) with respect to \( u_0 \) is now obtained from equations (2.31) and (2.32) and given by

\[ \bar{u} = \frac{u}{u_0} = \frac{(\alpha^2 i + \frac{1}{Da^2})c}{4ac_0} \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} A_m y^m - \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} \lambda_m y^{m+2} \]  

\[ = \frac{\sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} \lambda_m y^{m+2}}{\sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2}} \]  

(2.33)

### 2.3.2 Calculation for Hemodynamic Indicator Volumetric Flow Rate (Q)

The volumetric flow rate \( Q \) of the fluid in the stenotic region is given by

\[ Q = 2\pi R_0 \int_0^{R/R_0} uy dy \]

\[ = 2\sqrt{B_0} \int_0^{R/R_0} \left[ \frac{c}{4a} \sum_{m=0}^{\infty} \sum_{n=0}^{\infty} \left( \frac{R}{R_0} \right)^{n+2} \sum_{m=0}^{\infty} A_m y^m - \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{n+2} \sum_{m=0}^{\infty} \lambda_m y^{m+2} \right] y dy \]

\[ = \frac{\sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2} - \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} \lambda_m y^{m+2} \sum_{m=0}^{\infty} \lambda_m y^{m+2} - \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} \lambda_m y^{m+2} \sum_{m=0}^{\infty} \lambda_m y^{m+2}}{\sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2}} \]  

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\[ \frac{\pi R_0^3}{2a\mu_0} \frac{\partial p}{\partial z} \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2} - \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+4} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m = \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m \]

(2.34)

Let \( Q_0 \) denotes the flow rate of plasma fluid in unconstricted tube (M=0 and H=0) which is given by

\[ Q_0 = \frac{B_0^2}{8\sqrt{\left( \frac{\partial p}{\partial z} \right)_0}} \]

(2.35)

where, \( \left( \frac{\partial p}{\partial z} \right)_0 \) being the pressure gradient of the fluid in unconstricted uniform tube.

Thus non-dimensional flow rate \( \bar{Q} = \frac{Q}{Q_0} \) is given by

\[ \bar{Q} = \frac{4}{a} \frac{\partial p}{\partial z} \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2} - \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+4} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+4} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m \]

(2.36)

The expression for the relative pressure gradient can be obtained by

\[ P = \frac{\partial p}{\partial z} \left( \frac{\partial p}{\partial z}_0 \right) \]

\[ P = a \frac{Q}{4Q_0} \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+2} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^{m+2} - \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+4} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m \sum_{m=0}^{\infty} \lambda_m \left( \frac{R}{R_0} \right)^{m+4} \sum_{m=0}^{\infty} A_m \left( \frac{R}{R_0} \right)^m \]

(2.37)

2.3.3 Calculation for Hemodynamic Indicator Wall Shear Stress (WSS)

The shear stress at the surface of stenosis is described by

\[ \tau_s = \left[ \frac{\partial u}{\partial r} \right]_{r=R} \]
Which with the help of (23) and value of $\mu(r) = \mu_0[1 + \beta h(r)]$ can be written as

$$\tau_s = \left[ a + \frac{1}{\sqrt{a}} \right] f_{j0} e^{\frac{1}{2} \beta_3} \sum_{m=0}^{\infty} \left( \frac{R}{R_0} \right)^{m+2} \sum A_{m+1} (m+1) y^m$$

$$- \frac{1}{R_0} \left[ \sum A_m \left( \frac{R}{R_0} \right)^m \sum (m+3) y^{m+2} \right]$$

$$= - \mu_0 \left[ a - k \left( \frac{R}{R_0} \right)^n \right] e^{\frac{1}{2} \beta_3} \sum \left( \frac{R}{R_0} \right)^{m+2} \sum (m+1) A_{m+1} \left( \frac{R}{R_0} \right)^m$$

$$- \frac{1}{R_0} \left[ \sum A_m \left( \frac{R}{R_0} \right)^m \sum (m+3) \left( \frac{R}{R_0} \right)^{m+2} \right]$$

$$= - \mu_0 \left[ a - k \left( \frac{R}{R_0} \right)^n \right] e^{\frac{1}{2} \beta_3} \sum \left( \frac{R}{R_0} \right)^{m+2} \sum (m+1) A_{m+1} \left( \frac{R}{R_0} \right)^m$$

$$- \frac{1}{R_0} \left[ \sum A_m \left( \frac{R}{R_0} \right)^m \sum (m+3) \left( \frac{R}{R_0} \right)^{m+2} \right]$$

$$= - \mu_0 \left[ a - k \left( \frac{R}{R_0} \right)^n \right] e^{\frac{1}{2} \beta_3} \sum \left( \frac{R}{R_0} \right)^{m+2} \sum (m+1) A_{m+1} \left( \frac{R}{R_0} \right)^m$$

$$- \frac{1}{R_0} \left[ \sum A_m \left( \frac{R}{R_0} \right)^m \sum (m+3) \left( \frac{R}{R_0} \right)^{m+2} \right]$$

Also, if $\tau_N$ is the shear stress at the wall in the absence of stenosis, then

$$\tau_N = \frac{\sqrt{a} e^{\frac{1}{2} \beta_3}}{R_0} \frac{I_1(\sqrt{i + \frac{1}{Da^2} y})}{I_0(\sqrt{i + \frac{1}{Da^2}})}$$

The non-dimensional form of shear stress is now obtained as

$$\tau = \frac{\tau_s}{\tau_N}$$
2.4 RESULTS AND DISCUSSION

The expression of velocity, wall shear stress, volumetric flow rate, and pressure gradient are obtained and computed data are plotted for different values of Hartmann number $H$, Darcy number $Da$, Womersley number $\alpha$, and Hematocrit $H_m$. The profiles of axial velocity versus radial coordinate for various physical parameters are shown in Figures 2.2 to 2.5. Figure 2.2 depicts that with the increase of Hartmann number ($H$) the flowing fluid is slowed down in axial direction. Besides, deceleration in the flow with increase of Hartmann number the axial velocity profile ceases to remain parabolic and becomes flatten at the centerline region. For $H=4$ the velocity profile is almost flat at the centerline region while on further increasing the value of Hartmann number the profile at centerline region bends in reverse direction. Figure 2.3 shows the velocity profiles at a various level of Hematocrit concentration at the fixed value of Hartmann number $H=5$ (at which in the centerline region profile bend in reverse direction of flow). Besides, slowed down in the axial flow velocity with the increase of Hematocrit concentration, it is plausible that the velocity profile in centerline region for less Hematocrit concentrated blood corresponds more curvature as compared to higher Hematocrit concentrated blood. Figure 2.4 demonstrates that the axial flow velocity increases on increasing Womersley number, i.e., flow velocity will be augmented by raising the oscillation in the flow. Figure 2.5 depicts that if porosity of the medium in the vessel increases there is amplification in the axial velocity. Also, for less porous medium the profiles in centerline region are flatted and converge to parabolic form on increasing porosity of the medium. Figures 2.6 and 2.7 demonstrate the effect of magnetic field on wall shear stress (WSS) in the stenosed region of the vessel. WSS increases with the increase of magnetic field which is in good agreement with studies carried out by (Sud and Sekhon (2003), Halder and Ghosh (1994), Mekheimer and El Kot (2008), Prakash et al. (2004)). In the present
study we found that WSS changes its sign twice in the region of stenosis near by entry and exit of the stenosis as seen in Figure 2.7. The occurrence of these variation suggests that there will be two region of circulation for the value of Hartmann number \( H > 2 \). The magnetic field strength also affecting the location of the circulation region, for weak magnetic field the circulation regions occurs closures to the extremes of the stenosis. While, on increasing strength of the magnetic field the circulation region moves towards the neck of the stenosis that may affect more adversely on the hemodynamic conditions of the flow. Figures 2.8 to 2.10, reveal that the WSS decreases with the increase of Hematocrit concentration, Womersley number and porosity of the medium. But there is no significant occurrence of the circulation region. Figures 2.11 to 2.14 show that volume flow rate of the blood decreases with the increase of Hartmann number, Hematocrit concentration, Womersley number and Darcy number. The effect of magnetic field, porosity of the medium and Hematocrit concentration is quite significant in compare to Womersley number. Figure 2.15 and Figure 2.16 reveals that pressure gradient increases with the increase in Hartmann number and hematocrit concentration respectively.

2.5 CONCLUSIONS

1. The zones of magnetic field in laboratory, EM transmission tower etc., patient suffering with CV disease will have more risk on hemodynamic point of view as the flow in centerline region perturbed from parabolic to zero velocity gradient and reverse flow.

2. Magnetic field may be a cause of circulation and secondary flow in the stenosed region.

3. Both magnetic field and presence of porous medium (the resultant of thrombus in the blood vessel) reduces the amount of blood to be transported to the organs.

In general, the effect of magnetic field in our surrounding due to EM transmission tower, MRI, Magnetic tunnels, bullet magnetic trains etc., are not taken into account but in the present study it is observed that the adverse effect of magnetic field is inversely proportional to the Hematocrit concentration.
Figure 2.2: Variation of axial velocity with Hartmann number at $n=2, Hm=0.45, Da=10, \alpha=1.83, \beta=2.5$

Figure 2.3: Variation of axial velocity $u$ with Hematocrit at $n=2, Da=10, H=5, \alpha=1.83, \beta=2.5$
Figure 2.4: Variation of axial velocity with Womersely number at $n=2, Da=10, H=2, Hm=0.45, \alpha=0.5, \beta=2.5$.

Figure 2.5: Variation of axial velocity with Darcy number at $n=2, Hm=0.45, H=2, \alpha=1.83, \beta=2.5$. 
Figure 2.6: Variation of Wall Shear Stress along the stenosis with Hartmann number at $n=2, Da=10, Hm=0.45, \alpha=1.83, \beta=2.5$

Figure 2.7: Variation of Wall Shear Stress along the stenosis with Hartmann number at $n=2, Da=10, Hm=0.45, \alpha=1.83, \beta=2.5$
Figure 2.8: Variation of Wall Shear Stress along the stenosis with Hematocrit at $n=2, H=5, Da=10, \alpha=1.83, \beta=2.5$

Figure 2.9: Variation of Wall Shear Stress along the stenosis with Womersley number at $H=5, Hm=0.45, Da=10, \beta=2.5$
Figure 2.10: Variation of Wall Shear Stress along the stenosis with Darcy number at $n=2, H=2, H_m=0.45, \alpha=1.83, \beta=2.5$

Figure 2.11: Variation of volumetric flow rate through stenosed region with Hartmann number at $H_m=0.45, \text{Da}=10, \alpha=1.83, \beta=2.5$
Figure 2.12: Variation of Volumetric flow rate through stenosed region with Hematocrit at $H=5, Da=10, P=0.5, \alpha=1.83, \beta=2.5$

Figure 2.13: Variation of Volumetric Flow Rate through stenosed region with Womersely Number at $H=1, Hm=0.45, P=0.5, Da=10, \beta=2.5$
Figure 2.14: Variation of Volumetric flow rate through stenosed region with Womersley number at $n=2, H_m=0.45, P=0.5, Da=10, \beta=2.5$

Figure 2.15: Variation of Pressure Gradient through stenosed region with Hartmann number at $n=2, Da=10, H_m=0.45, \alpha=1.83, \beta=2.5, Q=0.5$
Figure 2.16: Variation of Pressure Gradient through stenosed region with Hematocrit at $n=2, H=1, Da=10, \alpha=1.83, \beta=2.5, Q=0.5$