CHAPTER - 1

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Mathematics is the queen of the science.
-Carl Friedrich

1.1 Historical Remarks

Biomechanics is a scientific branch that combines the field of applied mechanics with the field of Biology and Physiology and is concerned with a human body. In biomechanics, the concepts of mechanics are applied to explain or solve biological problems; thus it is a natural science concerned with living systems of mechanics. The modern development of biomechanics started in the fifteenth century, based on the studies of Leonardo da Vinci (1452-1519), though the concepts of biomechanics were probably given in Greece (Aristotle 384-322 BC). Later the contributors to biomechanics were Galileo (1564-1642), Rene Descartes (1596-1650), William Harvey (1578-1657), Robert Boyle (1627-1691), Isaac Newton (1642-1727), Jean Poiseuille (1797-1869) and the others. The development of the biomechanics as a separate branch has improved our understanding the mechanics of human blood flow, mechanics of human joints, the mechanics of airflow in the lungs. Moreover, biomechanics has contributed to the development of medical diagnostic and treatment procedures as well as to development of designing and manufacturing medical instruments and devices for the handicapped. We see that biomechanics represents a strongly interdisciplinary branch.

The great philosopher, Aristotle was the first who spoke on the connection between physics and living things. However, biomechanics in the modern sense began with Galileo Galilei (1564-1642) and William Harvey (1578-1657). Without having
any knowledge of existence blood vessels and having no microscope, only on the basis of certain logics, William Harvey discovered blood circulation. Harvey was dissatisfied with Galen’s theories, and by a clever combination of arguments and experimentation proved that blood must travel in a closed circuit in the cardiovascular system. The circulation of the blood was now established, although it was not understood how blood passed from the pulmonary artery to the pulmonary vein until Malpighi (1628-1694) described the pulmonary capillaries. Richard Lower (1631-1691) noted the difference in colour between arterial and venous blood and said that the blood actually absorbs air in its passage through the lungs. Galileo established that blood can leave the ventricle of the heart in one direction only and measured the capacity of the heart to be two ownces per beat. The hearts beats 72 times per minute under normal health conditions, therefore it pumps 2×72×60 ownces=8640 ownces=540 pounds of blood into the system in one hour.

Besides above mentioned scientists, there are a number of other names in biomechanics, which are equally familiar to engineers. Italian geometer and astronomer, Giovanni Alfonso Borelli (1608-1679), used the principles of levers and other concepts from mechanics to analyze muscle action, was also interested in respiratory mechanics, he was able to show that inspiration is driven by muscles, while expiration is a passive process resulting from tissue elasticity. Robert Boyle (1627-1691) studied about the lung and discussed the function of air in water with respect to fish respiration. The Fick’s law in mass transfer was given by the physiologist Adolf Fick (1829-1901). The hydrodynamicist D. J. Korteweg and Horace Lamb (1849-1934) wrote several beautiful research papers on wave propagations in blood vessels. Otta Frank (1865-1944) worked out the hydrodynamic
theory of the heart. Robert Hooke gave Hooke’s law in mechanics and the word ‘cell’ to biology. L. Euler was the first who wrote a definite paper in 1775 on blood flow in arteries. J. Poiseuille (1799-1869) invented the mercury manometer to measure the blood pressure in the dog’s aorta in 1828 and gave his law of viscous flow in 1840.

However, with such a rich background one may realize that bio-fluid mechanics fell outside the main stream of development of the fluid mechanics for a long time. The serious study of bio-fluid is a fairly recent endeavour. Many of the bio-fluid mechanical problems remain to be unsolved and discussed. The present thesis is therefore devoted to study the bio-fluid mechanical flow problems of stenosis with reference to blood.

1.2 PHYSIOLOGICAL ENVIRONMENT AND ASSOCIATED FLUID MECHANICS

It appears to be necessary to discuss certain aspects of physiology and fluid mechanics before introducing the work done in the thesis.

1.2.1 Blood Physiology

Human whole blood is a very complex fluid that plays a most important role in maintenance, growth, defence, and restoration in body. The function of blood is to feed all the tissues of the body with vital materials and to remove waste. Blood consists of a suspension of cells in an aqueous solution called plasma which is composed of about 90% water and 7% protein. There are about 5×10^9 cells in a milliliter (1cc) of healthy human blood, of which about 95% are red cells or erythrocytes whose main function is to transport oxygen from the lungs to all the cells of the body and the removal of carbon-dioxide formed by metabolic processes.
in the body to the lungs. About 45% of the blood volume in an average man is occupied by red cells. This fraction is known as the hematocrit. Of the remaining, white cells or leucocytes constitute about one-sixth or 1% of the total, and these play a role in the resistance of the body to infection; platelets form 5% of total, and they perform a function related to blood clotting.

In 5 litres of blood in the human body, there are about 25×10^{12} red cells. The mean life of a red cell is about 120 days and the total number of red cells which die per second is

\[
\frac{25 \times 10^{12}}{120 \times 24 \times 60 \times 60} = 2.4 \times 10^6.
\]

The total number of red cells which serves a man in his life time of 60-70 years (about 200 cycles of 120 days each) is about 5×10^{15}. Their total volume is about 2.25×200=450 litres, so that in a life time about 0.5 ton of red blood cells are manufactured in our body. These cells supply oxygen to about 60 million cells of the body.

By considering the fact that 25×10^{12} cells occupy about 0.45×5=2.25 litres of blood, the mean volume of a red cell can be obtained as

\[
\frac{2250}{2.5 \times 10^{10}} \text{cc} = 9 \times 10^{-11} \text{cc} = 9 \times 10^{-17} m^3 = 90 \mu m^3.
\]

The volume of a red cell can also be obtained by considering the shape of a red cell which is biconcave disc with an average diameter of about 8\(\mu\)m and thickness varying from 1\(\mu\)m at the centre to about 2.2\(\mu\)m at the ends. We can also find the surface area of a red blood cell, which is about 140 \(\mu\)m^2. Hence the total surface area of all red cells is about 25×10^{12} × 140 \(\mu\)m^2 = 3500 \(m^2\). This surface area
is important because diffusion of oxygen takes place all along this surface.

**1.2.2 Blood Viscosity**

Viscosity is defined as resistance of a fluid to deformation under shear stress. This describes the fluid’s internal resistance to flow and can be thought of as a form of fluid ‘friction’. Mathematically, viscosity is defined as the ratio of shear stress to velocity gradient.

Blood consists of plasma, blood cells and other materials carried throughout the blood stream. The quantity of particles within the plasma causes blood to have non-Newtonian behavior, which means the viscosity changes with the shear rate of the flow. If shear rate is sufficiently high, blood flow exhibits Newtonian flow behavior. However, under normal conditions it is not viable to ignore the non-Newtonian behavior of the fluid. Plasma in isolation may be considered Newtonian with a viscosity of about 1.2 times that of water. For whole blood, we can measure effective viscosity, and this is found to depend on shear rate. The constitutive equations proposed for whole blood are as follows:

i) \[ \tau = \mu e^n, \text{(Power law equation)} \]

This is found to hold good for strain rates between 5 and 100 sec\(^{-1}\), within having a value between 0.68 and 0.80.

ii) \[ \tau = \mu e^n + \tau_0, (\tau \geq \tau_0) \text{(Herschel-Bulkley equation)} \]

iii) \[ \tau^{1/2} = \mu^{1/2} e^{1/2} + \tau_0^{1/2}, \text{(Casson equation)} \]

This holds for strain rates between 0 and 100,000 sec\(^{-1}\).

iv) \[ \frac{dr}{(\tau + C)^a} = K \frac{de}{(e + B)^{\beta}}, \text{(Oka’s equation)} \]
Besides above, several researchers have suggested that blood can be suitably represented by a suspension model (i.e., a suspension of red cells in plasma). Suspension of cells in plasma up to a volume fraction $\phi$ of 0.05 have a steady shear stress shear strain relationship similar to Newtonian fluid. The suspension viscosity when $\phi \leq 0.05$ is expressed with reasonable accuracy by Einstein’s equation of spheres in suspension.

$$
\mu_s = \frac{\mu_0}{1 - m\phi},
$$

(1.2.1)

where $\mu_0$ is plasma viscosity, $\mu_s$ is suspension viscosity and $m$ is the shape parameter (2.5 for spheres).

One of the assumptions in the above formulation is that the particles do not collapse and behave individually. As $\phi$ increases from 0.05 the suspension viscosity varies from Eq. (1.2.1). Over the years many empirical relations were suggested and used in the literature for viscosity of suspension in the case of blood. Cokelet (1963) derived the relation that for red cell suspensions

$$
\mu_s = \frac{\mu_0}{(1 - \phi)^{2.5}},
$$

(1.2.2)

with a cone and plate viscometer, the apparent viscosity of red cell suspensions is found to be correlated up to $\phi = 0.6$ by Eq. (1.2.2) in which $m$ is expressed by

$$
m = 0.70 \exp [2.49\phi + \frac{1107}{T} \exp (-1.69\phi)],
$$

(1.2.3)

where $T$ is measured in absolute temperature (k).
1.2.3 Human Circulatory System

The human circulatory system pumps five litres of blood through a complex network of passages that passes through the vital organs of the human body, providing nutrients and oxygen that these organs use and carry out the waste products and potentially harmful chemicals away from these organs. The heart is responsible for providing the driving push to move all this blood whereas the lungs allow for the exchange of gases: providing oxygen to be carried to the vital and peripheral organs and taking away the carbon dioxide build-up.

Cardiopulmonary circulation involves the movement of blood from heart to lungs and back again and is important for removing waste gases and saturating the blood with oxygen prior to being pumped from the heart to other portions of the body. The veins bring in blood rich in waste materials, particularly carbon dioxide which results from the combustion processes necessary to generate energy carried out throughout the body. This enters the right atrium of the heart (lower chamber) via two large veins called the vena cavae, which then contracts (systole) and pushes the blood into the right ventricle (upper chamber) via a one-way valve. The right ventricle then contracts to force the fluids out through the pulmonary artery into the lungs, whereby the aforementioned exchange of gases occurs. This blood, now rich in oxygen is further pumped into the left atrium via the pulmonary vein, which is pumped into the left ventricle of the heart and expelled through the aorta, the largest artery in the body (to withstand the high pressures), to the other portions of the body. It is important to note that there are a series of valves within the heart and within the veins around the body that prevent backflow from occurring by sealing off the vessels when the heart is expanding (diastole), causing a lower pressure upstream.
### Table 1.2.1 Geometry and structure of various blood vessels.

<table>
<thead>
<tr>
<th>Vessels</th>
<th>Diameter (cm)</th>
<th>Length (cm)</th>
<th>Wall Thickness (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td>2.5</td>
<td>50</td>
<td>0.2</td>
</tr>
<tr>
<td>Artery</td>
<td>0.4</td>
<td>50</td>
<td>0.1</td>
</tr>
<tr>
<td>Arteriole</td>
<td>0.005</td>
<td>1</td>
<td>0.2</td>
</tr>
<tr>
<td>Precapillary Sphincter</td>
<td>0.0035</td>
<td>0.02</td>
<td>0.003</td>
</tr>
<tr>
<td>Capillary</td>
<td>0.0008</td>
<td>0.1</td>
<td>0.0001</td>
</tr>
<tr>
<td>Venules</td>
<td>0.0020</td>
<td>0.2</td>
<td>0.0002</td>
</tr>
<tr>
<td>Vein</td>
<td>0.5</td>
<td>25</td>
<td>0.05</td>
</tr>
<tr>
<td>Vena Cava</td>
<td>3.0</td>
<td>50</td>
<td>0.15</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vessels</th>
<th>Endothelium</th>
<th>Elastic Tissue</th>
<th>Smooth Muscle</th>
<th>Fibrous Tissue</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td>5</td>
<td>40</td>
<td>25</td>
<td>30</td>
</tr>
<tr>
<td>Artery</td>
<td>5</td>
<td>35</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>Arteriole</td>
<td>5</td>
<td>25</td>
<td>45</td>
<td>25</td>
</tr>
<tr>
<td>Precapillary Sphincter</td>
<td>15</td>
<td>15</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Capillary</td>
<td>100</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Venules</td>
<td>100</td>
<td>0</td>
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<td>0</td>
</tr>
<tr>
<td>Vein</td>
<td>5</td>
<td>40</td>
<td>30</td>
<td>25</td>
</tr>
<tr>
<td>Vena Cava</td>
<td>5</td>
<td>25</td>
<td>35</td>
<td>35</td>
</tr>
</tbody>
</table>
The conduits for blood in man are the aorta, the larger arteries, the main artery branches, the arterioles, the capillaries, the terminal veins, the main venous branches, the larger veins and the vena cava. The size of blood vessels ranges from 1.5 cm in diameter for aorta to about \(8 \times 10^{-4} \text{ cm} (8 \mu \text{m})\) for a capillary. The geometry and the size of different blood vessels are given in Table 1.2.1.

The flow in terminal branches of arteries, arterioles, capillaries, venules, and terminal veins varies intermittently in contrast with the pulsatile flow in aorta and large arteries, however, it is not pulsed. Except capillaries and some venules, the walls of all vessels are composed of collagenous fibers, smooth muscles, elastin and endothelial living cells. Some venules consist of endothelial cells having a small number of fibers whereas capillaries contain only endothelial cells but some venules consists of endothelial cells only. However, larger venules contain smooth muscle. The main pulmonary artery and its major branches have no elastic lamellae unlike the aorta and large arteries. Arteries differ from veins as later have values. The endothelial living provides a smooth interface between flowing blood and the vessel wall which reduces friction associated with blood flowing in vessels. Consequently it influences the contact of the red cells with the vessel wall. Capillaries are not subject to nervous activities and respond passively to the internal pressure within the vessel, whereas other vessels with smooth muscle are subject to nervous activity and are therefore capable of contracting and expanding their diameter.

Poiseuille, a French physician observed that the flow of water is inversely proportional to the length and proportional to the fourth power of the tube radius. His work merits detailed study not only for its historic interest, but also because of bearing on questions that arise subsequently. Probably, Hagen Bach was the first to
give a definition of viscosity, based on the assumptions that (a) flow is laminar, (b) fluid layer next to the wall is stationary, (c) the velocity of each layer is proportional to force acting on it (Newtonian Fluid), the proportionality constant is known as viscosity, (d) the system is thermal; established the following theoretical derivation of Poiseuille law

\[ Q = \frac{\pi P R^4}{8 \mu L}, \]

where \( \mu \) is viscosity, \( Q \) is flow rate, \( P \) is the pressure difference between entrance and exit, \( L \) is tube length and \( R \) being its radius. Poiseuille law was found to be applicable also to blood flow through capillaries.

1.3 Stenosis

The cause and development of many cardiovascular diseases; most common types are ischemia, angina pectoris, myocardial infarction and cerebral strokes; are related to the nature of blood movement and mechanical behaviour of the blood vessel wall, that is why the study of the blood flow through a stenosed artery is very important. Constriction, medically called stenosis or arteriosclerosis, is the narrowing of anybody passage, tube or orifice, comes from Greek words arthero (meaning gruel or paste) and sclerosis (hardness). The common form of arterial narrowing or stenosis is that caused by atheroma, a deposition of fats and fibrous tissue in the arterial lumen. Such constriction of the arterial lumen grows inward and restricts the normal movement of blood where the transport of blood to the region beyond the narrowing is reduced considerably. For instance partial or total occlusion of a coronary artery can reduce the blood supply to the heart, thereby inducing effects such as angina pectoris and enhancing the possibility of a myocardial infarction; likewise stenosis in one or more
major vessels supplying blood to brain can lead to a cerebral strokes, and total or partial occlusion of vessels supplying limbs in common place and cause severe pain and loss of functions in the extremities.

It is well established that the interaction between the blood flow through the artery and the stenosis plays an important role in the wide-ranging spectrum of problems associated with arterial stenosis. There are many faces to this complex problem, ranging from biomechanical aspects at the molecular and cellular level to diagnostic and surgical techniques required for the diagnosis and treatment of advanced cases of arterial stenosis. The fluid mechanics related specific problems can be grouped into three categories as under:

(a) Effects of the stenosis on regional blood flow to peripheral vascular beds

(b) Localization hydrodynamic effects such as velocity distribution, wall shearing stress distribution, pressure distribution along the stenosis, turbulence and separation phenomena

(c) Methods for detection and clinical evaluation of stenoses

For the purpose of theoretical modelling of arterial stenosis, several types of stenosis geometry has been used in the literature, some of these are given as under:

(i) The geometry of the symmetrical stenosis assumed to be manifested in the arterial wall segment is described (Figure: 1.3.1) as:

\[
\frac{R(z)}{R_0} = 1 - \frac{\delta}{2R_0} \left(1 + \cos \frac{2\pi}{L_0} \left(z - d - \frac{L_0}{2}\right)\right); \quad d \leq z \leq d + L_0, \quad (1.3.1)
\]

\[
= 1; \quad \text{otherwise,}
\]
where \((R(z), R_0)\) are the radius of the tube (with, without) stenosis, \(L_0\) is the stenosis length and \(d\) indicates its location, \(\delta\) is the depth of the stenosis at throat.

\(\text{(ii)}\) The geometry of an axially nonsymmetrical but radially symmetric stenosis, assumed to be manifested in the arterial wall segment is described (Figure: 1.3.2) as

\[
R(z) = R_0 + \sum_{n=1}^{6} \delta_n \sin\left(\frac{n \pi z}{L}\right)
\]

Figure: 1.3.2. The geometry of a non-symmetrical stenosis.
\[
\frac{R(z)}{R_0} = 1 - \Delta \left[ L_0^{(n-1)} (z - d) - (z - d)^n \right]; \quad d \leq z \leq d + L_0, \\
= 1; \quad \text{otherwise}
\]  

(1.3.2)

where \((R(z), R_0)\) are the tube radius (with, without) stenosis, \(n \geq 2\) is a parameter (called shape parameter) determining the stenosis shape (the symmetric stenosis occur when \(n = 2\)), \(L\) is tube length, \(L_0\) is stenosis length and \(d\) indicates its location, \(\alpha\) is the ratio of central core radius to tube radius in unobstructed region. The parameter is given by

\[
\Delta = \frac{\delta}{R_0 L_0^{(n-1)}} (n - 1)
\]

where \(\delta\) are the maximum height of stenosis at \(z = d + L_0/\sqrt{n^{(n-1)}}\) such that \(\delta/R_0 << 1\).

(iii) The geometry of the composite stenosis, assumed to be manifested in the arterial wall segment is described (Figure: 1.3.3) as

\[
\frac{R(z)}{R_0} = 1 - \frac{2\delta}{R_0 L_0} (z - d); \quad d \leq z \leq d + L_0/2,
\]

Figure: 1.3.3. Geometry of a composite stenosis in an artery.
\[ L_d(z) = 1 - \frac{\delta}{2R_0} \left( 1 + \cos \left( \frac{2\pi}{L_0} (z - d - L_0/2) \right) \right); \quad d + L_0/2 \leq z \leq d + L_0, \quad (1.3.3) \]

\[ = 1; \quad \text{otherwise}, \]

where \( R \equiv R(z) \) and \( R_0 \) are respectively, the radius of the artery with and without stenosis, \( L_0 \) is the length of the stenosis and \( d \) indicates its location, \( \delta \) is the maximum projection in the lumen located at \( z = d + L_0/2 \).

\textbf{(iv)} The geometry of the bell shaped stenosis, assumed to be manifested in the arterial wall segment, is described (Figure: 1.3.4) as

\[ \frac{R(z)}{R_0} = 1 - \frac{\delta}{R_0} \exp \left( -m^2 \varepsilon^2 z^2 \right), \quad |z| \leq L_0, \quad (1.3.4) \]

\[ = 1; \quad \text{otherwise}, \]

\[ \text{Figure: 1.3.4. The geometry of a bell shaped stenosis in an artery.} \]

where \( R_0 \) is the radius of the arterial segment in the non-stenotic region, \( R(z) \) is the radius of the stenosed portion located at the axial distance \( z \) from the left end of the segment, \( \delta \) is the depth of stenosis at the throat and \( m \) is a parametric constant, \( \varepsilon \) the relative length of the constriction, defined as the ratio of the radius to the half length.
of the stenosis, i.e., \( \varepsilon = \frac{R_0}{L_0} \).

(v) The geometry of the stenosis which is assumed to be manifested in the arterial wall segmented is described (Figure: 1.3.5) as:

\[
\frac{R(z)}{R_0} = 1 - \frac{3}{2} \frac{\delta}{R_0 L_0} \left[ 11(z - d) L_0^3 - 47(z - d)^2 L_0^2 + 72(z - d)^3 L_0 - 36(z - d)^4 \right],
\]

\[
d \leq z \leq d + L_0 = 1; \quad \text{otherwise},
\]

Figure: 1.3.5. The geometry of an overlapping stenosis in an artery.

where \((R(z), R_0)\) are the radius of the tube (with, without) stenosis, \(L_0\) is the stenosis length, \(d\) indicates the stenosis location, \(\delta\) is the maximum height of the stenosis into the lumen, appears the location: \(z = d + L_0 / 6\) and \(z = d + 5L_0 / 6\), \(z\) being the axial coordinate. The height of the stenosis at \(z = d + L_0 / 2\), called critical height, is \(3\delta/4\)
It is now obvious that growth characteristics are arbitrary and many possibilities exist. Although, the stenosis varies with time, the variation is considered to be slow so that flow can usually be considered steady. Thus the fluid mechanics analysis depends only on the instantaneous condition of the stenosis. It is worth mentioning here that majority of abnormalities may be idealized by one of the above discussed stenosis geometries.

### 1.4 Governing Equation

In order to discuss two-phase macroscopic model of blood flow, let us consider an axisymmetric tube of radius $R$, filled with a mixture of small spherical rigid particles in an incompressible Newtonian viscous fluid. Using a continuum approach, the governing equations of motion describing the flow of a particle-fluid system are expressed (Drew, 1974, Srivastava and Srivastava, 1983) as

\[
(1-C) \rho_f \left( \frac{\partial u_f}{\partial t} + u_f \frac{\partial u_f}{\partial r} + v_f \frac{\partial u_f}{\partial z} \right) = - (1-C) \frac{\partial p}{\partial z} + (1-C) \mu_f (C) \]

\[
\left( \frac{\partial^2 u_f}{\partial r^2} + \frac{1}{r} \frac{\partial u_f}{\partial r} + \frac{\partial^2 u_f}{\partial z^2} \right) + CS \left( u_p - u_f \right), \tag{1.4.1} \]

\[
(1-C) \rho_f \left( \frac{\partial v_f}{\partial t} + u_f \frac{\partial v_f}{\partial r} + v_f \frac{\partial v_f}{\partial z} \right) = - (1-C) \frac{\partial p}{\partial r} + (1-C) \mu_f (C) \]

\[
\left( \frac{\partial^2 v_f}{\partial r^2} + \frac{1}{r} \frac{\partial v_f}{\partial r} + \frac{\partial^2 v_f}{\partial z^2} \right) + CS \left( v_p - v_f \right), \tag{1.4.2} \]

\[
\frac{\partial}{\partial r} \left[ (1-C) v_f \right] + (1-C) u_f \frac{\partial}{\partial r} + \frac{\partial}{\partial z} \left[ (1-C) u_f \right] = 0, \tag{1.4.3} \]

\[
C \rho_p \left( \frac{\partial u_p}{\partial t} + u_p \frac{\partial u_p}{\partial r} + v_p \frac{\partial u_p}{\partial z} \right) = -C \frac{\partial p}{\partial z} + CS \left( u_f - u_p \right) \tag{1.4.4} \]
where \( z \) and \( r \) are the cylindrical coordinates with \( z \) measured in the axial direction and \( r \) measured along the radial direction, \( p \) is the pressure, \((u, v)\) denote fluid phase velocity components in the axial and radial directions, respectively, \((u_p, v_p)\) are particulate phase velocity components, \(\rho_f\) and \(\rho_p\) are the fluid and particulate phases density, respectively, \(C\) denotes the volume fraction of the particles, \(\mu_s(C)\) is the mixture viscosity (effective viscosity) and \(S\) is the drag coefficient of interaction for the force exerted by one phase on the other. Since the whole blood is a complex mixture, an attempt to analyse the system in an exact manner is very difficult. We therefore use a number of simplifications based on the properties of blood and the flow situations under consideration. The basic assumptions for present discussions are as under:

(a) Blood is considered as a two phase fluid that is a suspension of red cells in plasma,

(b) The red cell is a rigid neutrally bount spherical particles, the specific gravity of the cell is about 1.1 and that of plasma is about 1.03 such that the effect of gravity on blood flow is very small, i.e. settling tendency of erythrocytes is negligible,

(c) Cell-cell interaction is neglected,

(d) Interaction between two phases is according to \textit{Stoke’s drag law},
The volume fraction occupied by the red cells (concentration of red cells in suspension) is assumed to be a constant,

Since small vessels are similar to a tunnel in gel, the flow may be assumed to be a one-dimensional,

Brownian motion of erythrocytes (red cell) is neglected,

The flow is fully developed and circular tube is considered as infinite,

Flow is steady and is generated by the pressure gradient, which is a function of z only.

As far as rheological properties of blood are concerned, assumption (a) may be reasonable. Blood cells are actually irregularly shaped (generally biconcave discoid type) highly flexible particles. Thus, in the assumption (b) one is limited in not being able to account for the cell shape and the deformation it undergoes during shear flow. However cell deformability is not significant at low shear rates. Effect of cell-cell interaction is felt important at high concentration of cells, which makes present discussion limited to dilute suspension. The results using the constant volume fraction are exactly true in the limit of low concentration. Finally, assumption (g) seems reasonable because of Brownian motion is significant only for very small particles such as protein molecules.

In view of the above assumptions, the appropriate equations (neglecting body forces and body couples) describing the flow of cell-plasma system are simplified to

\[
(1-C) \frac{dp}{dx} = (1-C) \frac{\mu_s(c)}{r} \frac{\partial}{\partial r} \left( r \frac{\partial}{\partial r} \right) u_f + CS (u_p - u_f),
\]

\[
C \frac{dp}{dz} = CS (u_p - u_f),
\]
The expression for the drag coefficient of interaction, $S$ is selected as

$$S = \frac{9}{2} \frac{\mu_o}{a^2} \lambda'(C),$$

$$\lambda'(C) = \frac{4 - 3(8C - 3C^2)^{1/2} + 3C}{(2 - 3C)^2}, \quad (1.4.9)$$

where $\mu_o$ is the fluid (plasma) viscosity and $a$ is the radius of a particle (red cell).

Equation (1.4.9) represents the classical Stoke’s drag for small particle Reynolds number, modified to account for the finite particulate fractional volume through the function $\lambda'(C)$ obtained by Tam (1969).

Many empirical relations have been suggested to express the viscosity of suspension as a function of particle concentration and viscosity of the suspending medium. Einstein was the first to obtain theoretically that the viscosity of suspension $\mu_s$ was related to that of the suspending medium $\mu_o$ for spheres in suspension by $\mu_s = \frac{\mu_o}{1 - 2.5C}$. However, the Einstein’s formula expresses the viscosity of suspension only when $C$ is less than 0.05. As $C$ increases form 0.05, the suspension viscosity varies from the Einstein’s equation. An empirical relation suggested by Charm and Kurland (1965) for the suspension viscosity seems to be reasonable for present discussion and is given as

$$\mu_s = \frac{\mu_o}{1 - mC},$$

$$m = 0.07 \exp \left[ 2.49C + \frac{1107}{T} \exp \left( -1.69C \right) \right], \quad (1.4.10)$$

where $T$ is measured in absolute temperature ($K$). The viscosity of suspension expressed by this formula is found to be reasonably accurate up to $C=0.6$. Charm and
Kurland tested equation (1.4.10) with a cone and plate viscometer and found it to be in agreement within ten percent in the case of blood. The solution of Eqs. (1.4.7) and (1.4.8) are now straightforward and yield, after simple integration subject to the appropriate boundary conditions, the expressions for velocity profiles, the flow rate, effective viscosity using the definitions from fluid mechanics.

Blood flow is generally known to be pulsatile, to include the same one has to consider the terms like \( \frac{\partial u_f}{\partial t} \) and \( \frac{\partial u_p}{\partial t} \) in the flow equations. Certain pulsatile flows have been discussed in the thesis. Other flow characteristics such as pressure drop across a given length of the vessel and shearing stress at the tube wall may be obtained using the necessary definition from fluid mechanics. To study the flow at the entrance region, the inertia terms in the flow equations need to be considered. It is now apparent from the above discussion that blood may be represented by a suspension model that is a suspension of red cells in plasma in small vessels.

1.5 Review of Previous Literature

A large number of theoretical and experimental efforts have been made in the literature to explain the blood flow behaviour when it flows through the vessels of the circulatory system of living beings. To account for the new evidences uncovered through improved experimental theories of blood flow from the numerous relevant and important contributions of Bayliss (1952), Womersley (1955a, 1955b), Taylor (1959), Mc Donald (1960), Whitmore (1963), Copley and Stainsby (1960), Attinger (1964), Fung (1968) and many others, mathematical modelling of blood flow has been subject to constant changes and modifications. Above listed investigators have used single-phase homogeneous Newtonian viscous fluid, a classical approach that
does not account for the presence of red cells (one of the main constituents of blood, which is responsible for many of the blood properties and diseases) to describe blood flow in human circulatory system. Although this approach provides satisfactory tools to describe certain aspects of blood flow in aorta and large arteries, it fails to give an adequate representation of flow field, especially in the vessels of small diameter (2400 – 8μm).

A number of investigators including Haynes (1960), Merrill et al. (1963), Charm and Kurland (1974), Hershey et al. (1964), Cokelet (1963) and Lih (1975) have pointed out that blood, being a suspension of corpuscles, behaves like a non-Newtonian fluid at low shear rates. In particular, Hershey et al. (1966) and Huckaba et al. (1968) have shown that blood flowing through a tube of diameter less than 0.2 mm and at low shear rate less than 20/s, behaves as a power law fluid while Casson (1959), Reiner and Scott-Blair (1959), Charm et al. (1965, 1974) and Merrill et al. (1963) have suggested that blood inhibits yield stress and behaves as a Casson model fluid at a shear rate equal to 0.1/s.

Investigation of Cokelet (1972) and theoretical observations of Haynes (1960) indicate that blood can no longer be treated as a single-phase homogenous viscous fluid in small size vessels (of diameter ≤ 1000μm). It is surprising to note that the individuality of the red cells (of diameter 8μm) is important even in such large vessels (with diameter up to 100 cells diameter). Skalak (1972) concluded that in capillary vessels whose diameter (4-10μm) are equal or smaller than that of a red blood cell, an accurate description of flow requires consideration of red cells as discrete particles. Also, certain observed phenomena in blood flow including Fahraeus- Lindquist effect
(the decrease of apparent viscosity with decreasing diameter of blood vessels), non-Newtonian behaviour, etc. cannot be explained fully by considering blood as a single-phase homogenous fluid. Thus, in dealing with the problem of microcirculation also, the individuality of red blood cells cannot be ignored. It seems to be therefore important and necessary to consider the whole blood as a particle-fluid system while flowing through small vessels. In addition, Bugliarello and Sevilla (1965), and Thurston (1989) have shown experimentally that for blood flowing through small vessels, there is a cell-free plasma (Newtonian fluid) layer and a core region of suspension of all the erythrocytes. With the above discussion in mind, we therefore propose to represent blood in small vessels by a two-fluid model consisting of a core region of suspension of all the erythrocytes and a peripheral layer of plasma (Newtonian fluid).

Constriction, medically called stenosis, is a term that describes narrowing of an artery due to the accumulation of cholesterol, fats and the abnormal growth of tissue. Stenosis most commonly occurs in the large distributing arteries such as the coronary artery, or in the large renal, cerebral, iliac or femoral arteries. A common cause of this constriction is a chronic disease process called atherosclerosis. In the region of narrowing arterial constriction, the flow accelerates and consequently the velocity gradient near the wall region is steeper due to the increased core velocity resulting in relatively large shear stress on the wall even for a mild stenosis (Young 1979; Caro 1973; Fry, 1973; Liu et al. 2004; Poltem et al. 2006). It has been established that development of stenosis in early stage of the disease, is strongly related to the characteristics of the blood flow by Gidden D.P. (1993).
Young (1968) has analyzed the effects of stenosis on flow characteristics of blood treating blood as Newtonian fluid. Although, this approach provides satisfactory tools to describe certain aspects of blood flow in aorta and large arteries, it fails to give an adequate representation of flow field, especially in the vessels of small diameter (2400-8 µm) (Srivastava and Srivastava 1983). With the advent of the discovery that the hemodynamic factors play an important role in the genesis and the proliferation of stenosis has attracted the interest of early investigators to study the response of blood flow through stenotic lesions, since the first investigation of Mann et al. (1938), a large number of researcher have addressed the stenotic development problems under various flow situations including Young (1968), Young and Tsai (1973), Caro et al. (1978), Shukla et al. (1980), Ahmed and Giddens (1983), Ku (1997), Liu et al. (2004), Pralhad and Schultz (2004), Jung et al. (2004), Politis et al. (2007, 2008), Sankar and Lee (2009), Layek et al. (2005, 2009), Siddiqui S. U. (2009), Joshi et al. (2009), Srivastava and coworkers (1995, 2009, 2010, 2012), Mishra et al. (2006), Ponalagusamy (2007), Mekheimer and El-Kot (2008), Layek et al. (2009), Tzirtzilakis (2008), Mandal et al. (2007), Politis et al. (2008), Singh et al. (2010), Biswas and Chakraborty (2010a,b), Nadeem et al. (2011), Mishra and Siddiqui (2011), Bandyopadhyay and Layek (2011, 2012), Medhavi (2011, 2012), and many others. A survey of the literature on arteriosclerotic development reveals that the studies conducted are mainly concerned with the single symmetric and non-symmetric stenoses. However, the stenosis may develop in series (multiple stenoses) or may be of irregular shapes or bell shaped or of composite in nature, etc.

Kanai et al. (1970), has reported that when a catheter is inserted in a stenosed artery, it further increases the impedance to flow and changes the pressure
distribution. Jayaraman and Tewari (1995) have studied blood flow in a catheterized curved artery, by assuming the artery as a curved pipe and the catheter to be co-axial to it. Young and Tsai (1973), Lee (1974), Mc donald (1979), Ahmad and Giddens (1983), Ponalagusami (1986), Back (1994) and Back et al. (1996) studied the mean flow resistance increase during coronary artery catheterization in normal as well as stenosed arteries. Srivastava and Srivastava (2009) have presented a brief review of the literature on artery catheterization with and without stenosis.

Catheter procedure can both diagnose and treat heart and blood vessel conditions. The catheter tool device are used to gather the information such as, assess the amount of blood flow through the heart and blood vessel, to measure blood pressure, to measure the amount of oxygen in the blood, assess the electrical system of heart (ectrophysiology), close the holes in heart, treat abnormal heart rhythms with ablation procedures and intravascular ultrasound diagnosis. A catheter is made of polyester based thermoplastic polyurethane, medical grade polyvinyl chloride etc. For the purpose of flexibility PVC materials containing added plasticizers are used in catheter which enables them to move through the branches or curved paths of the circulatory system. Transducers attached to catheters are of many uses in clinical works and the techniques is used for measuring blood pressure or other mechanical properties in artery (Garbe 1972; Anderson et al, 1986). (Srivastava R., 2011), considered a two-layered model of blood flow through a catheterized artery with axially variable peripheral layer thickness and variable blood viscosity at the wall.

1.6 Thesis Outline

The present thesis deals with the study of flow through constricted tube with reference to blood under various flow situations. It contains the general introduction of the
study of blood flow, the discussion on the various aspects of the blood flow, the methods of solution, the motivation and the future scope of the thesis, etc.

Chapter 2 deals the effects of a composite stenosis on blood flow characteristics in an artery have been investigated. The flowing blood has been represented by a macroscopic two-phase model (i.e., a suspension of red cells in plasma). The expressions for the flow characteristics, namely, the impedance, the wall shear stress and the shear stress at the stenosis throat have been obtained. The effects of hematocrit on these flow characteristics have been discussed thoroughly and briefly.

Chapter 3 deals with the effect of hematocrit and the peripheral layer on blood flow characteristics due to the presence of a composite stenosis in arteries. To estimate the effect of hematocrit and the peripheral layer, the flowing blood is assumed to be represented by a two layered model consisting of a core region of suspension of all the erythrocyte assumed to be a particle-fluid suspension (i.e., a suspension of all erythrocyte in plasma) surrounded by a peripheral layer of plasma (Newtonian fluid). The expression for the flow characteristics, namely, the impedance, the wall shear stress and the shear stress at the stenosis throat, has been derived. To validate the applicability of the present model, quantitative and comparative analysis is performed through graphical representation of numerical results obtained by developing computer programming.

Chapter 4 presents the investigation concerns the fluid mechanical study on the effects of the permeability of the wall through an axisymmetric stenosis in an artery assuming that the flowing blood is represented by a two-fluid model. The expressions for the blood flow characteristics, the impedance, the wall shear stress
distribution in the stenotic region and the shearing stress at the stenosis throat, have been derived. Results for the effects of permeability as well as of the peripheral layer on these blood flow characteristics are shown graphically and discussed briefly.

Chapter 5 deals with the effects of the hematocrit and the permeability of the wall on blood flow characteristics due to the presence of a bell shaped stenosis in an artery. In this analysis, the flowing blood is represented by a macroscopic two-phase model, as a suspension of erythrocytes in plasma. The analytical expressions for the flow characteristics, namely, the flow resistance (impedance), the wall shear stress distribution in the stenotic region and the shearing stress at the stenosis throat have been derived. Results for the effects of permeability as well as of hematocrit on these flow characteristics are shown graphically and discussed briefly.

The flow through the annular space of two concentric circular cylinders is known to have significant applications in engineering as well as in physiology (flow in intestine with an inserted endoscope and the flow of blood in catheterized tubes). Chapter 6 has therefore been devoted to the study of blood flow through the model of a composite stenosed catheterized artery with permeable wall, to investigate the blood flow characteristics. The expressions for the blood flow characteristics—the impedance (resistance to flow), the wall shear stress distribution in stenotic region, the shear stress at the stenosis throat of the stenosis have been derived. Results obtained have been displayed graphically and discussed briefly.

Chapter 7 deals with flow of blood through a narrow catheterized artery with axially non-symmetrical stenosis. Flowing blood has been represented by a two-layered model consisting of a core region of suspension of all the erythrocyte assumed to be a particle-fluid suspension (i.e., a suspension of all erythrocyte in plasma) and a
peripheral layer of plasma (Newtonian fluid). The expression for the flow characteristics, namely the impedance, the wall shear stress, the shear stress at stenosis throat has been derived. The extensive quantitative analysis is performed through numerical computations of the desired quantities having rheological relevance through their graphical representations so as to validate the applicability of the present model.

The relevant references are appended at the end.

1.7 Motivations and Scope of the Thesis

The thesis deals with the bio-fluid mechanical problems of stenosis and the suspension blood flow. Since the advent of the discovery that over seventy five percent of all deaths are caused due to the circulatory disorders, the researches on stenosis (one of the major causes of circulatory disorders) became the subject of interest to the scientists. There had been little work done in the previous literature to observe the effects of hematocrit (red cell concentration) on blood flow characteristics due to the presence of non-symmetrical stenoses in blood vessels. The problem of blood flow using a two-phase fluid (i.e., a mixture of red cells and plasma) through stenotic vessels has therefore been solved. This particular type of work enables one to express the blood flow characteristics as a function of erythrocyte concentration. Very little efforts were made previously to discuss the flow through a non-symmetrical stenosis, attempt has been thus made in the present work to study the blood flow through nonsymmetrical stenoses. As known that for blood flowing in small vessels, there is a cell free plasma layer near the vessel wall, effects of a peripheral layer have also studied in the thesis. It is worth mentioning here that two layered suspension flow seems to be of particular physiological interest. However, it is concluded that
considerable amount of researches are further necessary to discuss the stenotic development problems adequately and in more realistic situations.

Most of the problems on stenosis included in the present thesis deal with a mild stenosis case. The studies are mainly concerned with steady flow in rigid as well as permeable wall. Thus, the consideration of severe stenosis cases, pulsatile flow, inclusion of inertial terms in the flow equations etc., remains the task for future researches.