GENERAL

INTRODUCTION
1.1 STENOSIS/ARTERIOSCLEROSIS / ATHEROSCLEROSIS

This is a chronic disease in which thickening, hardening, and loss of elasticity of the arterial walls occurs, leading to impaired blood circulation. The thickening and hardening of the arteries are due to the build-up of calcium deposits in the lumen of the artery causing stenosis. It develops with aging, hypertension, diabetes, hyperlipidemia, and other diseased conditions in the artery. Figure 1.1 shows an artery with cholesterol build up and figure 1.2 shows a normal and a diseased artery.

Atherosclerosis is a specific type of arteriosclerosis, but the terms are often used interchangeably. Both conditions have similar effects on the circulation of the blood throughout the body. Atherosclerosis refers to the buildup of fats in and on your artery walls (plaques), which can restrict blood flow. These plaques can also burst, causing a blood clot to form which may travel to other part of the body and partially or totally block blood flow. Various heart diseases may be the result of arteriosclerosis or atherosclerosis.

The major risk factors that create such situations are the following:

- High blood pressure
- High cholesterol
- Smoking
- Some diseases, such as diabetes, obesity etc.
- A family history of early heart disease

Among all these conditions high blood pressure is more dangerous as the excess strain on the arteries causes them to become weak and calcium and fatty deposits tend to form in these weakened areas causing the blood pressure to become even higher. Figure 1.3
shows the reduced blood flow and clotting of blood due to plaque in an artery and figure 1.4 shows the renal arterial duplex ultrasonography of bilateral renal artery stenosis.

Atherosclerosis symptoms depend on which arteries are affected. For example:

- If atherosclerosis is present in the heart arteries, the person may have symptoms similar to those of a heart attack, such as chest pain (angina).
- If atherosclerosis is present in the arteries which are leading to the brain, the person may have symptoms such as sudden numbness or weakness in the arms or legs, difficulty in speaking or drooping muscles in the face.
- If atherosclerosis is present in the arteries which are related to arms and legs, the person may have symptoms of peripheral arterial disease, such as leg pain when walking etc.

Sometimes atherosclerosis causes erectile dysfunction in men.

(www.emedicinehealth.com/hardening_of_the_arteries/article_em.htm)
Figure 1.1: An artery with cholesterol build up.

Figure 1.2: Normal and diseased artery

Figure 1.3: Reduced blood flow due to plaque in an artery

* www.mayfieldclinic.comPE-CarotidStenosis.htm
Figure 1.4: Renal arterial duplex ultrasonography of bilateral renal artery stenosis, demonstrates a Doppler spectral waveform of the left mid-renal artery, showing elevated peak systolic velocities, consistent with severe stenosis.

1.2 BACKGROUND AND PHYSIOLOGY OF BLOOD FLOW

Blood is a heterogeneous suspension containing several different components. In the typical adult human, blood is composed of approximately forty-five percent plasma and the other fifty-five percent consists of a heterogeneous mixture of cellular components. The percentage of formed elements closely approximates the percentage of RBCs per given volume of blood. This is an important measure in health care as it is an indicator of the oxygen-carrying capacity of blood. Total blood volume in an average adult is about 5 liters or 8% of the total blood volume and blood plasma makes up the remaining 55% (Van et al., 1997). The blood transports many substances (oxygen, carbon dioxide, nutrition, vitamins, etc.), the transport of heat (heating cooling), the transmission of signals (hormones), buffering and the protection of the body. Macroscopically viewed, blood is an incompressible viscous liquid on the base of water with a mean density of 1056 kg/m3 (1.056 g/cm3) and a viscosity of 3.5c Poise (rand et al., 1964 and Chien, 1970). Microscopically blood is a suspension of blood cells in plasma. The major constituents of this formed element group include: red blood cells (RBCs) or erythrocytes, white blood cells (WBCs) or leukocytes, and platelets or thrombocytes. Blood is composed of 4 components:

- Plasma- The liquid part of blood. A dilute solution of salts, glucose, amino acids, vitamins, urea, proteins and fats.
- White blood cells-These are involved in immune system.
- Platelets-These are involved in blood clotting.
- Red blood cells-These are involved in carrying oxygen.
Blood transport in the cardiovascular system depends upon the nature of the blood, its viscosity, its non-Newtonian character, cell concentration and distribution, blood vessel, its geometry, size and shape, curvature, branching, tapering, wall thickness, elasticity, porosity and nature of the flow such as pulsatile, laminar, turbulent, micro-circulation etc. (Fung et al. 1972), (Verma 2008).

1.3 RED BLOOD CELLS/ ERYTHROCYTES

The biconcave flattened discs with a depressed centre, mammalian RBC have a very thin membrane that encapsulates a viscous, liquid hemoglobin solution. It has a regular and mainly constant shape and size. The depressed centre provides increased surface area for the diffusion of gases. It is the hemoglobin which binds to oxygen and allows the RBCs to transport oxygen to cells. Their outer diameter varies between 7.59 µm and 8.5 µm as the thickness is of 2.5 µm (Evans and Fung 1972) and (Linderkamp and Meiselman 1982). They are produced in the bone marrow and have a lifetime of around 120 days inside the body (Cokelet 1986). Their main duty is to provide the cells with oxygen and bring the carbon dioxide away back to the lungs. Within the blood cells the RBCs comprise the largest part. In special flow situations (very slow flow) they clot and form so called rouleaux. This effect changes the properties of the blood (Verma 2008). The proportion of blood volume that is occupied by red blood cells is called hematocrit (Ht or HCT) or packed cell volume (PCV) or erythrocyte volume fraction (EVF). It is normally about 48% for men and 38% for women. It is considered an integral part of a person's complete blood count results, along with hemoglobin concentration, white blood cell count, and platelet count. The density of blood is dependent on the Hematocrit. A high
Hematocrit implies a high density, because all the blood particles are denser than plasma. Similar to the density, blood viscosity is not constant. It is dependent on the Hematocrit as well the shear rate (http://en.wikipedia.org/wiki/Hematocrit). Figure 1.5 shows the accumulation of red blood cells due to stenosis in an artery.
Figure 1.5: Accumulation of red blood cells due to stenosis.

1.4 CLASSIFICATION OF BLOOD VESSELS ACCORDING TO SIZE, LOCATION AND FUNCTION

1.4.1 ARTERIES
Arteries contain more muscle than comparably sized veins. Large arteries stretch when the pressure of the blood rises during systole and recoil during diastole. The elastic recoil of the walls helps to produce a smoother flow of blood in the smaller arteries and arterioles. However, the result is a cardiac cycle-dependent artery diameter. Smaller arteries and arterioles are less elastic than larger artery diameter and contain a proportionally thicker layer of smooth muscle. Thus they maintain a relatively constant diameter. They carry blood from the heart to every tissue in the body. They have thick, elastic walls to withstand the high pressure of blood from the heart. The arteries close to the heart are particularly elastic and expand during systole and recoil again during diastole, helping to even out the pulsating blood flow. The smaller arteries and arterioles are more muscular and can contract (vasoconstriction) to close off the capillary beds to which they lead; or relax (vasodilation) to open up the capillary bed. These changes are happening constantly under the involuntary control of medulla in the brain and are most obvious in the capillary beds of the skin causing the skin to change color from pink (skin arterioles dilated) to blue (skin arterioles constricted). Arterioles are small thick walled vessels that represent the major part of vascular resistance. These resistance vessels serve as “circulatory stopcocks” and control the distribution of blood to various organs (Verma 2008).

1.4.2 VEINS
Veins are large diameter thin walled vessels that bring blood back to heart. They are distensible and (in addition to venules) contain a large fraction of the blood volume. They
carry blood from every tissue in the body to the heart. The blood has lost almost all its pressure in the capillaries so it is at low pressure inside veins and moving slowly. Veins therefore do not need thick walls and they have a larger lumen than artery, to reduce the resistance to flow. They also have semi-lunar valves to stop the blood flowing backwards. Venules are small thin walled vessels that serve to bring blood back to the heart. These vessels are highly distensible and (along with veins) contain a large fraction of the blood volume. Blood is transported back to the heart by venules which empty into progressively larger and larger veins. The pressure in the veins is around 2 mmHg which is insufficient to return the blood to the heart (Van et. al. 1997). However, veins pass between skeletal muscles which contract during motion and naturally provide a massaging action on the veins. The squeezing effect venous pressure and helps to push the blood back up to the heart. One directional flow is maintained by venous valves which close in response to increased pressure (Verma 2008).

1.4.3 CAPILLARIES

Capillaries are the simplest structured vessel. Capillaries are extremely small extremely thin walled vessels (one cell thick) that allow exchange of gases, nutrients, and other small molecules between the blood stream and tissues. They are composed of a single cell layer of endothelium and are about 8 mm in length. They permeate the entire body in a fine mesh to provide the surface area for blood and interstitial fluid transfer. Capillaries only contain about 250 ml of blood at any given time (Van et. al 1997). The amount of blood in a capillary bed is regulated by the precapillary sphincter muscles and by the resistance to blood flow provided by the small arteries and arterioles. The transported substances actually enter and leave the blood in capillary. No exchange of materials takes place in the arteries and veins, whose walls are too thick and impermeable. Capillaries are
very narrow and thin walled, but there are a vast number of them (108 m in one adult), so they have a huge surface area: volume ratio, helping rapid diffusion of substances between blood and cells. Capillaries are arranged in networks called capillary beds feeding a group of cells, and no cell in the body is more than 2 cells away from a capillary. Increases in capillary hydrostatic pressure or capillary permeability can lead to edema (Verma 2008).

1.5 THE STRUCTURE AND FUNCTION OF THE HEART
The heart is made of cardiac muscle, composed of cells called myocytes. When myocytes receive an electrical impulse they contract together, causing a heartbeat. Since myocytes are constantly active, they have a great requirement for oxygen, so are fed by numerous capillaries from two coronary arteries. These arise from the aorta as it leaves the heart. Blood returns via the coronary sinus, which drains directly into the right atrium (Oricile). The heart acts as a double pump to keep blood circulating through blood vessels. It is surrounded by the pericardium whose inner lining produces fluid to lubricate the hearts motion. The heart is composed of three layers: the epicardium forms an outer protective sheath; the myocardium forms the middle layer composed mainly of cardiac muscle tissue; and the endocardium forms the inner layer which is continuous with the endothelium of the blood vessels.

The basic mechanism of the heart is as follows (see figure 1.6):

- The heart is a muscular pump with four chambers and four heart valves.
- The upper chambers, the right atrium (Oricile) and the left atrium (atria-plural for atrium), are thin walled filling chambers.
• Blood flows from the right and left atria across the tricuspid and mitral valves into the lower chambers (right and left ventricles).

• The right and left ventricles have thick muscular walls for pumping blood across the pulmonic and aortic valves into the circulation.

• Heart valves are thin leaflets of tissue which open and close at the proper time during each heart beat cycle.

• The main function of these heart valves is to prevent blood from flowing backwards.

• Blood circulates through the arteries to provide oxygen and other nutrients to the body, and then returns with carbon dioxide waste through the veins to the right atrium; when the ventricles relax, blood from right atrium passes through the tricuspid valve into the right ventricle.

• When the ventricle contract, blood from the right ventricle is pumped through the pulmonic valve into the lungs through pulmonary artery to reload on oxygen and remove carbon dioxide.

• The oxygenated blood then returns to the left atrium by pulmonary vein and passes through the mitral valve into the left ventricle.

• Blood is pumped by the left ventricle across the aortic valve into the aorta and the arteries of the body

(Verma 2008).
Figure 1.6: Structure of the heart

1.6 FLOW RATE OF BLOOD

The flow rate of blood is affected by viscosity and vice versa. At very low flow rate in the microcirculation, the blood viscosity can increase quite significantly. This occurs because at low flow states there are increased cell-to-cell and protein-to-cell adhesive interactions that can cause erythrocytes to adhere to one another and increase the blood viscosity. When the blood viscosity is measured in capillaries of diameters less than 300*10^-6 meter, then this viscosity also depends on the radii of the capillaries. This relation between the viscosity and the capillary radius is known as Fahraeus-Lindquist effect. (McDonald 1960).

1.7 VISCOSITY/VISCOSITY VARIATION

Viscosity of a fluid is that property by virtue of which it is able to offer resistance to flow. It is a property of fluid related to the internal friction of adjacent fluid layers sliding past one another as well as the friction generated between the fluid and the wall of the vessel. Blood viscosity is elevated in hypertensive subjects and it varies in various cases of diseases in the general population. The viscosity of whole blood is strongly influenced by three factors: hematocrit (percentage of volume of packed red blood cells in a given sample of blood), temperature and flow rate. Hematocrit is an important determinant of the viscosity of blood. As hematocrit increases, there is a disproportionate increase in viscosity (Einstein 1906). Higher hematocrit implies higher viscosity (See figure 1.7). The relation between hematocrit and viscosity is complex and many formulas exist. One of the simplest one is the written as follows:

$$\mu = \mu_r(1+2.5Ht)$$
where
\[ \mu = \text{viscosity of whole blood} \]
\[ \mu_p = \text{viscosity of the plasma} \]
\[ Ht = \text{hematocrit (percentage of volume of packed red blood cells in a given sample of blood)} \]

The viscosity of whole blood at a physiological hematocrit of 45 is about 3.2 centipoise (cP), or 3.2 \times 10^{-3} \text{ Pa.s}. At the normal hematocrit of about 40, the viscosity of whole blood (cells plus plasma) is about 3 times that of water. When the hematocrit rises to 60 - 70 which it often does in patients with polycythemia, or abnormally (high red blood cell counts), the blood viscosity can become as high as 10 times that of water. If the hematocrit falls sharply (e.g: Anemia) the blood viscosity decreases to the value of the viscosity of plasma alone. The MKS unit of viscosity is N-s/m^2.
1.8 BYPASS IN ARTERY/BYPASS SURGERY

When the arteries that carry blood to the heart muscle (coronary arteries) become clogged by plaque (a buildup of fat, cholesterol and other substances) which slows or stops blood flow through the heart's blood vessels, leading to chest pain or a heart attack then bypass surgery is needed. Increasing blood flow to the heart muscle can relieve chest pain and reduce the risk of heart attack. In medicine, a bypass generally means an alternate or additional route for blood flow, which is created in bypass surgery, e.g. coronary artery bypass surgery by moving blood vessels or implanting synthetic tubing. It involves creating a detour around a blocked artery, usually a coronary artery that supplies blood to the heart (coronary artery bypass surgery) but may be done in other areas (peripheral bypass). The bypass may be minimally invasive or open heart surgery is required for treatment of this blockage (www.health.com). (See figure 1.8, figure1.9, figure 1.10 and figure 1.11).
Figure 1.8: Double coronary artery bypass surgery, showing the grafting of a section of saphenous vein from the leg to bypass a blockage on the right side of the heart and the diversion of an internal mammary artery to bypass a blockage on the left side of the heart.

Figure 1.9: Femoral Popliteal Bypass

Figure 1.10: Bypass surgery: before and after

Figure 1.11: bypass surgery in right coronary artery

1.9 A RELEVANT SURVEY OF LITERATURE

Stenosis in arteries of humans is a common occurrence and hemodynamic factors play a significant role in the formation and proliferation of this disease. It is well known that at various locations in the arterial system, stenosis may develop due to abnormal intravascular growths. There have been a number of studies for the flow of blood in a stenosed artery (Young 1968), (Shukla et. al.1980), Perkkio and Keskinen (1981), Mishra and Chakravarty (1986) Haldar (1987), (Moshkelani et. al. 2003), etc. using mathematical model.

An analysis on the effect of an axially symmetric, time dependent growth into the lumen of a tube of constant cross section through which a Newtonian fluid is steadily flowing is presented by Young (1968). Shukla (1979) et al. studied the effects of peripheral layer viscosity on physiological characteristics of blood flow through the artery with mild stenosis. It has been shown that the resistance to flow and the wall shear decrease as the peripheral layer viscosity decreases. Influence of blood viscosity on blood flow and the effect of low molecular weight Dextran were studied by Dormandy (1971). He showed that a close inverse correlation exists between changes in viscosity and blood flow, the change in blood flow being three times greater than the change (decrease) in blood viscosity. The effects of viscosity concentration dependence and of the concentration profile of blood flow through a vessel with stenosis have been studied by Perkkio and Keskinen(1983). A two-layered fluid model for blood flow through a stenosed tube has been developed by Shukla (1979), Pralhad and Schultz (1988). The model consists of a core (suspension of RBC’s) and peripheral plasma layer. Resistance to flow and shear stress have been computed for different stenosis height. A steady axisymmetric flow in a
constricted rigid tube is studied by Pontrelli (2001), in which a shear-thinning fluid modeling the deformation dependent viscosity of the blood is proposed and the flow pattern with the distributions of pressure and shear stress at the wall are computed. Most et al. (2003) showed the effect of a reduction in blood viscosity on maximal myocardial oxygen delivery distal to a moderate coronary stenosis. The study tested the hypothesis that a reduction in blood viscosity by means of isovolumetric hemodilution will permit an increase in maximal oxygen delivery to myocardium distal to a moderate coronary arterial stenosis.

A theoretical solution of the unsteady-state momentum equation for the start up flow of a power law fluid in circular tubes is presented by Sestak and Charles (1968). The effect of unsteady flow behavior of blood was studied by Young and Tsai (1973), Clark (1976), Chakravarty et al. (1996), etc. They all presented the unsteady flow mechanism in the stenosed artery subject to a pulsatile pressure gradient arising from the normal functioning of the heart and the body acceleration. Effect of Stenosis on non-newtonian flow of the blood in an artery was studied by Shukla et al (1980). The effects of pulsatility, stenosis and non-Newtonian behavior of blood have been simultaneously studied by Mandal et. al (2007), Sankar and Lee(2009). Results for the rate of flow, the resistive impedance and the wall shear stress have been obtained. Padmanabhan and Devanathan (2006) presented closed-form solutions for equations governing the pulsatile flow of blood through models of mild axisymmetric arterial stenosis, taking into account the effect of arterial distensibility. Results indicate the existence of back-flow regions and the phenomenon of flow-reversal in the cross-sections. Varshney and Katiyar (2008) developed a mathematical model for the pulsatile flow of blood through a
stenosed artery under the influence of externally imposed body acceleration. The governing equations are solved numerically by using a suitable finite difference scheme in order to obtain the velocity, fluid acceleration, wall shear stress and flow rate. Blood behaves as a magnetic fluid (Hayat et. al 2006) due to the complex interaction of the intercellular protein, cell membrane and the hemoglobin. The effect of magnetic field in a stenosed artery was studied by Haldar (1994), Haldar and Ghosh (1993), Ogulu and Abbey (2005), Abdullah and Norsarahaidia (2007), Chandrasekhara and Rudraiah (1980) etc. Unsteady response of non-Newtonian blood flow through a stenosed artery in magnetic field was studied by IKBal (2008) et. al.

Gin et. al. (1999) modeled the diseased carotid artery bifurcation using a physiologically relevant geometric model. They carried out a systematic grid-independence study of the carotid artery bifurcation for the purpose of calculating steady and pulsatile flows. Chakravarty and Sen (2005) presented a mathematical model describing the dynamic response of heat and mass transfer in blood flow through stenosed bifurcated arteries. The geometry of the bifurcated arterial segment possessing constriction in both the parent and daughter arterial lumen frequently appearing in the diseased arteries causing malfunction of the cardiovascular system, is formulated mathematically with the introduction of the suitable curvatures at the lateral junction and flow divider. The respective profiles of the flow field, the temperature and the concentration and their distributions have been obtained. Computer simulations of blood flow with mass transport through the carotid artery bifurcation have been studied by Filipovic and Kojic (2004) by using numerical simulation and finite element method. Some experimental studies have also been
conducted in last few decades with and without bypass, Rodkiewicz et. al. (1988), Sperker et. al. (2002), Poltem et. al. (2006), Chen et. al. (2006), Qiao and Liu (2007) etc. In particular Rodkiewicz et. al. (1988) showed analytically and experimentally that within the scope of a surgery, the effects of variations in the position of the transplant-aorta contact point in the transplant length and in the transplant curvature are relatively insignificant regarding mean flow resistance. They concluded that it is not important how the transplant will be situated and that the space convenience should be surgical determining factor. It has been shown that the rate of blood flow to the kidney may be significantly curtailed if the selected transplant diameter is too small. The patients with atherosclerosis treated medically could be improved considerably by bypass surgery (Sperker et. al. 2002). A numerical study of non-Newtonian blood flow in stenosed coronary artery bypass with grafts was studied by Poltem et al (2006). They investigated the effect of using different bypass angles on the flow pattern. Their result showed that the proper choice of diameter of the graft might improve the balance of inflow and outflow in the coronary artery. Non-Newtonian fluid flow in a stenosed coronary bypass has been investigated numerically by Chen et. al. (2006) using the Carreau–Yasuda model for the shear thinning behavior of the blood. End-to-side coronary bypass anastomosis is considered in simplified model geometry where the host coronary artery has a 75% severity stenosis. Different locations of the bypass graft to the stenosis and different flow rates in the graft and in the host artery has been studied by them. The influence of graft diameter on the hemodynamics of femoral bypass graft, the pulsatile blood flows in three bypass models with different graft diameters were studied by Qiao and Liu (2007).
1.10 AIM OF THE THESIS

In this thesis, an attempt is made to study the various physiological problems and developed mathematical models by considering the behavior of blood to be a Newtonian fluid.

The following effects on blood flow through the artery are considered:

- Effects of accumulation of red cells causing change in the viscosity of blood both in the axial and radial directions
- Simultaneous effects of axial and radial variation of viscosity of blood.
- Effect of peripheral layer viscosity
- Effect of bypass

1.11 WORK DONE IN THE THESIS

In this thesis, we are concerned with the mathematical modeling of some physiological problems. The whole work is divided into seven chapters including the ongoing chapter on general introduction where fundamental concepts along with the review of related literatures are given. Chapters 2-7 are divided into cases dealing with similar type of problems in different frameworks for the clarity of the subject matter presented. Numerical and Analytical results are illustrated through graphs. In the end of the thesis, the relevant references are provided in the alphabetical order for the convenience of the readers. The chapter wise organization of the thesis is as follows:

Chapter 1 describes the general introduction and the survey of the research work done by the other investigators with regard to stenosis, Hematocrit, blood flow, structure and
functions of the heart, flow rate of blood and bypass surgery, so that the present work could be emphasized. It also gives an overview of the physiological properties of blood and blood vessels.

In Chapter 2, effects of radial variation of viscosity on resistance to flow of blood and shear stress in an artery with mild stenosis are analyzed by considering that there exists a radial decrease in blood viscosity from axis to the wall in the whole artery. The analytical results show that resistance to flow and shear stress decrease due to radial decrease in blood viscosity around the stenotic region.

In Chapter 3, a model to study the effects of axial variation of viscosity caused by accumulation of red cells in the stenotic region of an artery with mild stenosis on resistance to flow and shear stress, is proposed and analyzed. This biorheological aspect is taken into account in the usual Newtonian fluid flow model by assuming that the viscosity of blood increases axially in the stenotic region upto the point of maximum height of stenosis after which it decreases. The analysis shows that as viscosity increases, not only resistance to flow increases but shear stress also increases. Similar results are also found when viscosity due to accumulation of red cells increases axially, in the entire inlet region.

In Chapter 4, effects of axial variation of viscosity caused by accumulation of red cells in the entire region of an artery with mild stenosis on flow characteristics are studied. This aspect has been modeled by assuming that accumulation of red cells causes axial variation of viscosity. This effect has the effects of axial variation in an artery with mild stenosis on resistance to flow and shear stress. It is assumed that due to accumulation of
red blood cells in the stenotic region, the blood viscosity increases axially upto the maximum height of stenosis after which it decreases in the entire region of an artery. The analysis shows that resistance to flow or the shear stress at the maximum height of stenosis increases due to change in viscosity around the region of stenosis in the artery.

In Chapter 5, effects of accumulation of red cells causing radial and axial variation of viscosity on resistance to flow and shear stress in an artery with mild stenosis are studied. It is assumed that due to non uniform accumulation of red cells caused by obstruction due to mild stenosis the viscosity varies both axially and radially. The analysis shows that resistance to flow and shear stress at the maximum height of stenosis increase due to increase in viscosity in both radial and axial directions.

In chapter 6, effects of radial variation of viscosity with a peripheral layer on resistance to flow and shear stress in an artery with mild stenosis are modeled and analyzed by considering radial variation of blood viscosity in the entire core region when the blood viscosity varies radially in the central core region. The flow of blood consists of two regions of different viscosity. The blood viscosity is constant in the peripheral region and there exists a radial decrease in blood viscosity in the core region. The analytical results show that resistance to flow and shear stress decrease as the peripheral layer viscosity decreases.

In Chapter 7, a model to study the effect of bypass in a stenosed artery on resistance to flow and shear stress is proposed and analyzed. This biorheological aspect is taken into account in the usual Newtonian fluid flow model. The analysis shows that as height and length of stenosis increases, not only resistance to flow increases but shear stress also
increases for a fixed value of radius of bypass artery. Also as the value of radius of bypass artery increases, the resistance to flow and wall shear decreases and for a certain value of radius of bypass artery these values are similar as in case of no stenosis, hence bypass decreases resistance to flow and wall shear stress in a stenosed artery.

It is hoped that work done in the thesis would be useful to medical scientist working in the area of heart disease particularly related to atherosclerosis.