MATHEMATICAL MODELLING OF NON-NEWTONIAN BLOOD FLOW THROUGH A TAPERED STENOTIC ARTERY

ZUHAILA BINTI ISMAIL

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Faculty of Science
Universiti Teknologi Malaysia

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For My Dear Family
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A mathematical model of non-Newtonian blood flow through a tapered stenotic artery is considered. It has been established that the regional blood rheology is altered once a stenosis develops. A stenosis is defined as the partial occlusion of the blood vessels due to the accumulation of cholesterols and fats and the abnormal growth of tissue. The non-Newtonian model chosen is characterized by the generalized Power-Law model and the effect of tapering on the arterial segment is incorporated in the analysis due to the pulsatile nature of blood flow. The flow is assumed to be unsteady, laminar, two-dimensional and axisymmetric. The equations of motion in terms of the viscous shear stress in the cylindrical coordinate system are first derived and then transformed using the radial coordinate transformation before they are solved numerically using a finite difference scheme. Numerical results obtained show that the blood flow characteristics such as the velocity profiles, flow rate, and wall shear stress have lower values while the resistive impedances have higher values compared to the values obtained from the Newtonian model.
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### LIST OF SYMBOLS

- \( R(z,t) \): the radius of the tapered arterial segment in the stenotic region
- \( a \): the constant radius of the non-tapered artery in the non-stenotic region
- \( \phi \): the angle of tapering
- \( l_s \): the length of the stenosis
- \( d \): the location of the stenosis
- \( \tau_m \sec \phi \): the critical height of the stenosis for the tapered artery appearing at \( z = d + \frac{l_s}{2} \).
- \( m \): the slope of the tapered vessel
- \( \omega \): the angular frequency
- \( f_p \): the pulse frequency
- \( b \): constant variable
- \( L \): the finite difference arterial segment
- \( \phi < 0 \): the converging tapering
- \( \phi = 0 \): the non-tapered artery
- \( \phi > 0 \): the diverging tapering
- \( \tau_z \): shear stress
- \( v_z(r,z,t) \): the axial velocity component
- \( v_r(r,z,t) \): the radial velocity component
- \( p \): pressure
- \( \rho \): density of blood
- \( \lambda \): wavelength
\( \frac{\partial p}{\partial z} \) - pressure gradient

\( A_c \) - constant amplitude of the pressure gradient

\( A_i \) - amplitude of the pulsatile component

\( \Delta x \) - increment in the radial directions

\( \Delta z \) - increment in the axial directions

\( \Delta t \) - small time increment

\( Q \) - volumetric flow rate

\( ^\wedge \) - resistance

\( \tau_w \) - wall shear stress

\( V \) - volume
## LIST OF TERMINOLOGY

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<td>Atherosclerosis</td>
<td>arteriosclerosis characterized by irregularly distributed lipid deposits in the intima of large and medium-sized arteries; such deposits provoke fibrosis and calcification. Atherosclerosis is set in motion when cells lining the arteries are damaged as a result of high blood pressure, smoking, toxic substances in the environment, and other agents. Plaques develop when high density lipoproteins accumulate at the site of arterial damage and platelets act to form a fibrous cap over this fatty core. Deposits impede or eventually shut off blood flow.</td>
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<td>Blood</td>
<td>the fluid and its suspended formed elements that are circulated through the heart, arteries, capillaries, and veins; blood is the means by which 1) oxygen and nutritive materials are transported to the tissues, and 2) carbon dioxide and various metabolic products are removed for excretion. The blood consists of a pale yellow or gray-yellow fluid, plasma, in which are suspended red blood cells (erythrocytes), white blood cells (leukocytes), and platelets.</td>
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<td>Cardiac arrest</td>
<td>complete cessation of cardiac activity either electric, mechanical, or both; may be purposely induced for therapeutic reasons.</td>
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Cardiac cycle  - the complete round of cardiac systole and diastole with
the intervals between, commencing with any event in
the heart's action and ending when same event is
repeated.

Cardiovascular  - relating to the heart and the blood vessels or the
circulation.

Cerebrovascular  - relating to the blood supply to the brain, particularly
with reference to pathologic changes.

Diastole  - normal postsystolic dilation of the heart cavities,
during which they fill with blood; diastole of the atria
precedes that of the ventricles; diastole of either
chamber alternates rhythmically with systole or
contraction of that chamber.

Diastolic  - relating to diastole.

Disease  - an interruption, cessation, or disorder of body
functions, systems, or organs.

Elastic  - having the property of returning to the original shape
after being compressed, bent, or otherwise distorted or
a rubber or plastic band used in orthodontics as either a
primary or adjunctive source of force to move teeth.
The term is generally modified by an adjective to
describe the direction of the force or the location of the
terminal connecting points.

Erythrocyte  - a mature red blood cell. Synonym: red blood cell,
haemacyte, red corpuscle, hemacyte.

Hemoglobin  - the red respiratory protein of erythrocytes, consisting
of approximately 3.8% heme and 96.2% globin, with a molecular weight of 64,450, which as oxyhemoglobin (HbO₂) transports oxygen from the lungs to the tissues where the oxygen is readily released and HbO₂ becomes Hb. When Hb is exposed to certain chemicals, its normal respiratory function is blocked; e.g., the oxygen in HbO₂ is easily displaced by carbon monoxide, thereby resulting in the formation of fairly stable carboxyhemoglobin (HbCO), as in asphyxiation resulting from inhalation of exhaust fumes from gasoline engines. When the iron in Hb is oxidized from the ferrous to ferric state, as in poisoning with nitrates and certain other chemicals, a nonrespiratory compound, methemoglobin (MetHb), is formed.

Hypertension - high blood pressure; generally established guidelines are values of more than 140 mmHg systolic, or more than 90 mmHg diastolic blood pressure. Despite many discrete and inherited but rare forms that have been identified, the evidence is that for the most part blood pressure is a multifactorial, perhaps galtonian trait.

Laminar - arranged in plates or laminae.

Laminar flow - the relative motion of elements of a fluid along smooth parallel paths, which occurs at lower values of Reynolds number.

Stenosis - a stricture of any canal; especially, a narrowing of one of the cardiac valves.

Stenotic - narrowed; affected with stenosis.
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<td>Stress</td>
<td>reactions of the body to forces of a deleterious nature, infections, and various abnormal states that tend to disturb its normal physiologic equilibrium (homeostasis) or psychological stimulus such as very high heat, public criticism, or another noxious agent or experience which, when impinging upon an individual, produces psychological strain or disequilibrium.</td>
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<td>Systole</td>
<td>contraction of the heart, especially of the ventricles, by which the blood is driven through the aorta and pulmonary artery to traverse the systemic and pulmonary circulations, respectively; its occurrence is indicated physically by the first sound of the heart heard on auscultation, by the palpable apex beat, and by the arterial pulse.</td>
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<td>Systolic</td>
<td>relating to, or occurring during cardiac systole.</td>
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<td>Vessel</td>
<td>a structure conveying or containing a fluid, especially a liquid.</td>
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<td>Viscosity</td>
<td>in general, the resistance to flow or alteration of shape by any substance as a result of molecular cohesion; most frequently applied to liquids as the resistance of a fluid to flow because of a shearing force.</td>
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<td>Viscous</td>
<td>sticky; marked by high viscosity.</td>
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1.1 Introduction

Heart problem is one of the most common causes of death. Angina pectoris and myocardial infarction are two examples of heart diseases. Angina pectoris is the term used to describe the pain cause when the vessel is not carrying enough blood to the heart muscle. The pain occurs especially when the heart muscle needs more blood. In the case of myocardial infarction or heart attack, part of the heart muscle is destroyed. This causes severe pain in the chest that can lead to death. A healthy person is not aware of having a heart, or the important work it does in making the body works properly. The heart is the strongest organ in the body, and it works like a pump. The heart is like an engine, which can wear out, and breaks down completely, this is called cardiac arrest. Actually how likely someone is to have a heart attack depends on a number of things. The main cause that leads to a heart attack is atherosclerosis (see Figure 1.1.1).
Figure 1.1.1 Atherosclerosis

Chakravarty (1987) mentioned that atherosclerosis occurs when the nature of blood flow changes from its usual state to a disturbed flow condition due to the presence of a stenosis in an artery. Stenosis is defined as a partial occlusion of the vessels caused by abnormal growth of tissues or the deposition of cholesterol as substances on the arterial wall. This research considers the study of blood flow through arteries in the presence of stenosis because it can cause the development of cardiovascular diseases such as stroke and heart attack (see Figure 1.1.2).

Figure 1.1.2 Stenosis that Exists in Coronary Artery
Restricts Blood Flow to the Heart.

The fact that blood exhibits non-Newtonian behaviour was actually first recognised around the turn of the century (Enderle et al. (2000)). From a biofluid mechanics point of view, blood would not be expected to obey the very simple, one parameter, and linearized law of viscosity as developed by Newton. Blood is
nonhomogeneous, anisotropic ionic, composite fluid composed of a suspension of many asymmetric, relatively large, viscoelastic particles carried in a liquid that contains high molecular weight, asymmetric, ionic that behaves in a complicated way under shear – type loading. Therefore, blood exhibits non-Newtonian (nonlinear), time dependent (viscoelastic) deformation (flow) characteristics that can only be modelled by higher order constitutive equations, such as the power-law paradigm (Enderle et al. (2000)).

In physiological flows, there are other important factors that can be accounted for such as the effects of vessel tapering together with the geometry of stenosis. It has been pointed out that most of the vessels could be considered as long and narrow, slowly tapering cones. Besides, the process of systolic and diastolic also affects the vessels segment because it makes the vessel segment converges and diverges. Thus the effects of vessel tapering together with the non-Newtonian behaviour of the streaming blood seem to be equally important, hence deserves special attention.

In the next sections, we present the research background for the project followed by the objectives, scope, significance of research and outline of the dissertation.

1.2 Research Background

A number of researchers have studied the flow of non-Newtonian fluids with various perspectives. Ronald L. Fournier (1998) explained about the field of rheology concerns the deformation and flow behaviour of fluids, the prefix *rheo* is from Greek and refers to something that flows because of the particulate nature of blood. He expected the rheological behaviour of blood to be some what more complex than a simple fluid such as water. He mentioned that in order to understand the flow behaviour of blood, one must first define the relationship between shear stress and the shear rate. Ishikawa et al. (1998) found that the non-Newtonian
pulsatile flow through a stenosed tube is different from Newtonian flow. The non-Newtonian property strengthens the peaks of wall shear stress and wall pressure, weakens the strength of the vortex and reduces the vortex size and separated region. Therefore, he concluded that non-Newtonian flow is more stable than Newtonian flow.

Chakravarty and Mandal (1994) studied the unsteady flow behaviour of blood in an artery under stenotic condition analytically, by considering blood to be a non-Newtonian fluid and by properly accounting for blood viscoelasticity while the geometry of the stenosis was chosen to be overlapping to some extent, depending on time. Chakravarty et al. (1996) investigated the effect of a single cycle of body acceleration on unsteady non-Newtonian blood flow past a time-dependent arterial stenosis. Mandal (2005) pointed out that in some disease conditions, for example, patients with severe myocardial infarction, cerebrovascular diseases and hypertension, blood exhibits non-Newtonian properties. Gijsen et al. (1999) studied the impact of non-Newtonian properties of blood on the velocity distribution. They made a comparison between the non-Newtonian fluid model and a Newtonian fluid at different Reynolds numbers. Comparison reveals that the character of flow of the non-Newtonian fluid is simulated quite well by using the appropriate Reynolds number. Cheng Tu and Michel Deville (1995) noticed that for non-Newtonian flow through 75% stenosis, the influence of the geometrical disturbance affects the flow over a longer axial range.

John Enderle et al. (2000) pointed out those significant attempts to define such non-Newtonian behaviour, however did not appear until the 1960s, when variable-shear rotational viscometers were introduced. Since then, literally dozens of constitutive models have been proposed that attempt to relate shear stress to shear rate in the fluid. They said, the most practical of these is an empirical power law formulation that generalizes Newton’s law of viscosity. R. Manica and A.L. de Bortoli (2003) presented the simulation of incompressible non-Newtonian flow through channels with sudden expansion using the Power Law model. The Power Law model is applied to predict pseudoplastic (shear thinning) and dilatant (shear thickening) behaviour in such expansions. They pointed out that a better understanding of non-Newtonian flow through sudden expansions should lead to
both the design and development of hydrodynamically more efficient process and to an improved quality control of the final products.

The effect of vessel tapering is another important factor that should be considered. Chakravarty and Mandal (2000) formulated the problem on tapered blood vessel segment having overlapping stenosis. The problem is modelled mathematically as a thin elastic tube with a circular section containing an incompressible Newtonian fluid representing blood. Jeffords and Knisley (1956) and Bloch (1962) pointed out that most of the vessels could be considered as long and narrow, slowly tapering cones (Chakravarty and Mandal (2000)).

Inside a normal artery, red and white blood cells and other particles can flow freely to the peripheral organs. The walls of the inner linings of arteries are smooth and uniform in thickness. As an initial study, Formaggia et al. (2003) and Lee and Xu (2002) observed blood flow behaviour in non-stenotic vessel or a normal artery. Over time, however, the stenosis can build up within the artery walls. Quite a good number of theoretical studies related to blood flow through stenosed arteries have been carried out recently, Misra and Chakravarty (1989), Chakravarty (1987) and Chakravarty and Datta (1987). Most of the studies carried out so far have been focused on the presence of mild or single stenosis as discussed by Chakravarty et al. (1995, 1996, 2000), Chakravarty and Mandal (1997, 2000), Taylor et al. (1998), Lee and Xu (2002) and Mandal (2005). Moayeri and Zendehboodi (2003) found that once a mild stenosis is developed, the resulting flow disorder plays an important role in the further development of the disease.

In order to update resemblance to the in vivo situation, some studies have been investigated an overlapping stenosis in blood vessel segment subject to the pulsatile pressure gradient. Chakravarty and Mandal (1996), noted that the problem becomes more acute in the presence of an overlapping stenosis in the artery instead of having a mild stenosis as considered by aforesaid researchers. The study has been extended by Chakravarty and Mandal (2000) to include the time-dependent geometry of an overlapping stenosis present in a tapered artery. However, these studies considered a Newtonian model for blood flow. Beside the mild and overlapping
stenosis, Chakravarty and Sannigrahi (1999) gave special attention to multistenoses which appear in the artery.

There are different methods of solution in solving the problem of blood flow in normal and stenosed artery. Some researchers are solving analytically and some of them use numerical methods. Gerrald and Taylor (1977) used the finite difference method to solve the problem of blood flow in a normal artery. The finite difference method based on the central difference approximation has been employed by Chakravarty and Mandal (1994, 1997) and Mandal (2005). Misra and Pal (1999) observed the blood motion using Crank Nicolson implicit finite difference method. Runge-Kutta formula has been used by Chakravarty and Mandal (1996, 2000), Chakravarty et al. (1995, 1996, 2000) and Chakravarty and Sannigrahi (1999).

Beside the finite difference scheme, the finite element method has also been employed. Sud and Sekhon (1986) used the finite element model of flow in the normal branched arterial system subject to externally imposed periodic body acceleration and the relevance works have been extended by Sud and Sekhon (1987) by considering a stenosed artery. Formaggia et. al. (2003) presented a finite element Taylor-Galerkin scheme combined with operator splitting techniques in order to carry out several test cases.

1.3 Objectives of Research

The main objective of this research is to develop a mathematical model for non-Newtonian blood flow through a tapered stenotic artery.

The specific objectives are:

1. To derive the governing equations of blood flow, comprising the equation of continuity and the equation of motion in terms of the viscous shear stress.
2. To formulate the geometry of mild stenosis.

3. To carry out the radial coordinate transformation on the governing equations.

4. To solve the governing equations numerically using a finite difference scheme.

1.4 Scope of Research

This research takes into consideration the stenotic blood flow through the tapered artery to be incompressible, unsteady, two-dimensional and axisymmetric under laminar flow condition. The flowing blood is treated as a non-Newtonian fluid that is characterized by the generalized Power-law model and is observed through a mild stenosis. The discussion of this problem follows from the work of (Mandal (2005)).

1.5 Significance of Study

The benefits of this study are:

1. The development of a more realistic mathematical model to describe blood flow.

2. The development of a numerical package for the computation and simulation of bio-fluid problems.
1.6 Outline of Dissertation

This dissertation is divided into six chapters including this introductory chapter. Section 1.2 – 1.5 present the research background, objectives, scope and significance of research.

Chapter II presents the derivation of the governing equations. First, we show the derivation of the continuity equation and then the derivation of the equation of motion in terms of the viscous stress tensor, $\tau$. After that, both equations will be converted to cylindrical coordinates. The derivations of these formulae are given in Appendix A. Then, we show the derivation of the mathematical model. The last section in this chapter states the boundary conditions. The next chapter contains a discussion on the geometry of stenosis with their mathematical formulation. Then, we will show how to formulate the geometry of mild stenosis in a non-tapered and tapered artery.

The following chapter presents the transformation of the governing equations using the radial coordinate transformation. Then, the derivations of the radial velocity component, $v_r(x,z,t)$ and the solution of the axial velocity component, $v_z(x,z,t)$ are shown using the finite difference method. In the same section, the volumetric flow rate, the resistance and the wall shear stress will be determined. Next, we will show the numerical procedure to programme the finite difference method using MATLAB programming. The complete program is given in Appendix B. Lastly, in this chapter we state some comments about the numerical results.

Chapter V discusses the numerical results. This chapter will be divided into eight sections including the introduction. In Section 5.2, we discuss the results for the axial and radial velocities at different taper angles under stenotic conditions and also at taper angle with comparison between flow through stenosis, non stenosis and steeper stenosis. Next section, the results and discussion for axial and radial velocity at different times and at different type of fluid in the same times are given. Section
5.4 will be presented the results for axial and radial velocity at different axial positions. The following section will illustrate the results of the variation flow rate, resistance and wall shear stress with time. Last sections we will state some comments about the results obtained. Finally, Chapter VI will conclude the research problem and list out several suggestions for future research.
REFERENCES


