

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND STUDY

The physiological network of the human body is an embodiment of complex fluid flow systems among which are blood in the circulatory system, airflow in the airways, flow systems for transporting lymph and the urinary circulatory systems [1-3]. Recently, considerable attention has been given to the study of physiological fluid flow in order to understand the development of several arterial lesions such as stenosis, thrombosis, aneurysm [20, 37], etc. Blood is generally regarded as a continuum, especially in the region of large arteries. However, its dynamics is influenced by many factors among which are unusual multiplicity of tube branching, unusual pulsatility, unusual range of Reynolds number due to viscosity variation, unusual distensibility of the containing vessel [3]. The arterial blood flow provides a way for glucose, oxygen and hormones to reach various organs around the body. Blood leaves the heart from the left ventricle into the biggest artery called the aorta. It is important that fresh blood from the aorta goes directly to the brain, because the brain needs oxygen constantly to avoid irreversible damage to it [17, 18]. Another important organ which the blood must pass through is the lungs where waste carbon dioxide is replaced with fresh oxygen. Blood must move from an area of higher pressure on the arterial side to an area of lower pressure on the venous side by means of a pressure gradient. The difference in the arterial and venous pressure facilitates blood flow [1-3] (see Figures 1.1.1).

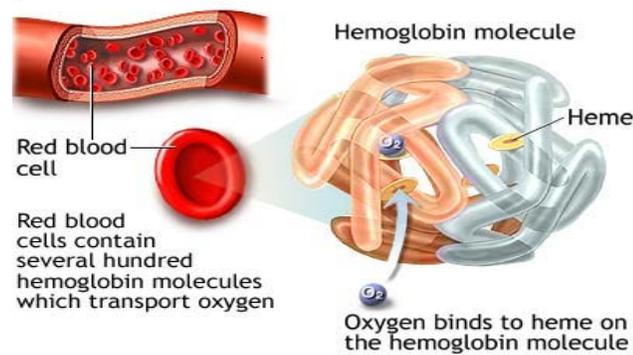


Figure 1.1.1: *Red blood cells in the arterial*

Ideally blood should be studied as liquid containing a suspension of flexible particles. Such a liquid can be characterized by a mechanical behavior which is non-Newtonian. This behavior becomes particularly significant when the particle size is large in comparison to the dimension of the channel in which the fluid is flowing. Red blood cell makes up more than 99% of all blood cell and approximately 40% to 45% of the blood volume (see Figures 1.1.2). In men there is an average of 5.2 millions red blood cells per cubic millimeter and in woman there is an average of 4.6 millions per cubic millimeter. The reason for lower red blood cells in women is that women lose lot of blood each month during menstruation period [22, 30].

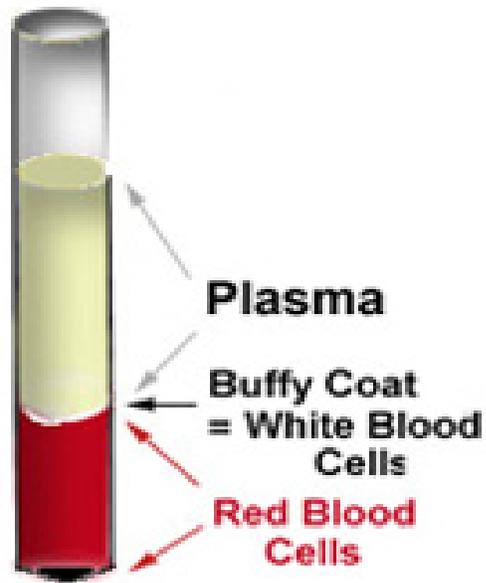


Figure 1.1.2: Composition of the blood

Hematocrit is the ratio of the volume of packed blood cells to the total blood volume and is therefore also known as the packed cell volume [31, 38]. The hematocrit is reported as a percentage or a ratio. In healthy adult individuals the red blood cells constitute approximately 40-48% of blood volume, whereas they may have hematocrits of up to 60%. A low hematocrit reflects a low number of circulating red blood cells and is an indication of a decrease in the oxygen-carrying capacity or of over-hydration. A high hematocrit may reflect an absolute increase in the number of erythrocytes, or a decrease in plasma volume, in conditions such as [37, 41, 42]:

- Severe dehydration – e.g. in case of burns, diarrhea or excessive use of diuretics
- Erythrocytosis – excessive red blood cell production
- Polycythemia vera – abnormal increase of blood cells
- Hemochromatosis – an inherited iron metabolism disorder

High hematocrit is also used as an indicator of the excessive intake of exogenous erythropoitin (EPO), which stimulates the production of red blood cells. Athletes can artificially improve their performance by enhancing the oxygen-carrying capacity with EPO [22, 31]. The concentration of erythrocytes in the blood has a strong influence on blood viscosity. At a hematocrit of 40-45%, blood viscosity is approximately 3 times the value for plasma and approximately 5 times that of water. Blood viscosity shows a curvilinear relation with the hematocrit and it increases sharply when the hematocrit is raised much beyond the normal range [39, 40]. The remarkable deformability of the normal red cells, besides making it possible for them to traverse narrow capillaries, serves to minimize the rise of blood viscosity with increasing cell concentration. The relation between blood viscosity and red cell concentration indicates that, when all other conditions are equal, blood flow would decrease with an increase in hematocrit, especially at high hematocrit levels [38].

Red blood cells are the oxygen carriers [1-3] (see Figures 1.1.3). As they travel away from the heart, they traverse smaller and smaller arteries, finally arriving at the collections of microscopic blood vessels known as capillaries. Here, they exchange nutrients and oxygen for cellular waste products. The waste products are eventually eliminated from the blood stream through the urinary and respiratory systems. The exchange of oxygen and nutrients between the red blood cells and the surrounding tissues occurs through a process called diffusion [33]. During diffusion process, whenever capillaries contain a high concentration of oxygen and nutrients, and the surrounding tissues contain a lower concentration, oxygen and nutrients leave the capillaries and enter the tissues. Conversely, whenever body tissues contain high concentrations of carbon

dioxide and metabolic waste, and the capillaries contain a lower concentration, the waste products diffuse from the tissues into the capillaries and from there are carried by the venous system back toward the heart [3].

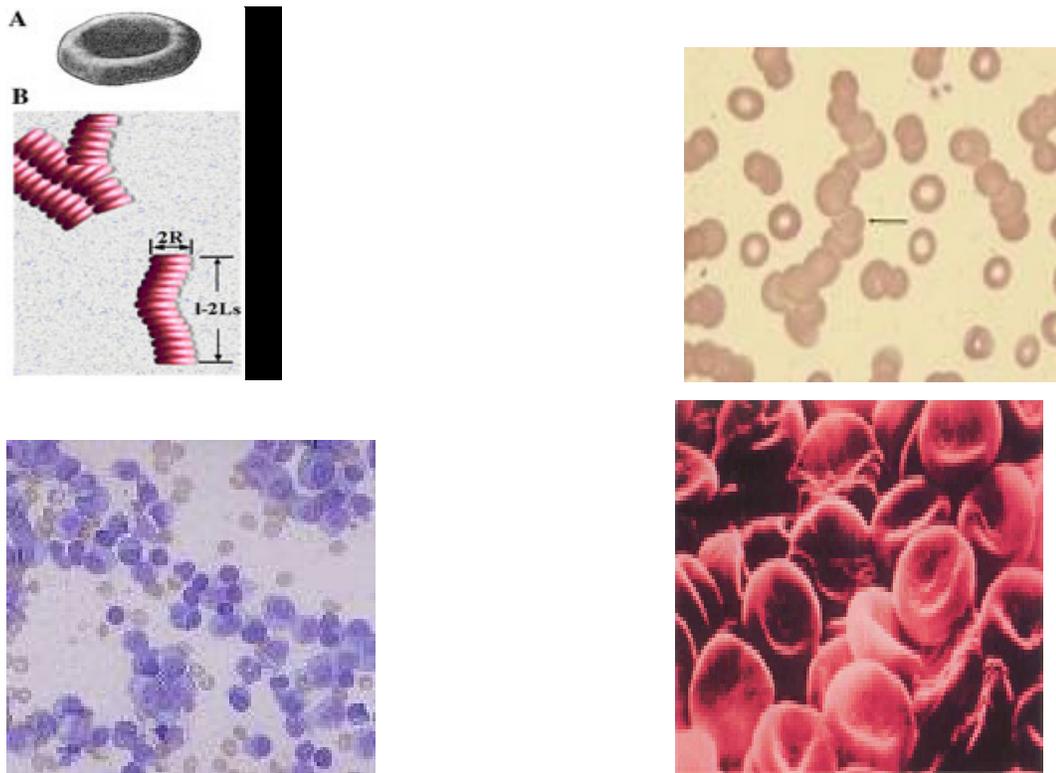


Figure 1.1.3: The red blood cells

In the course of blood flow in a large artery, the red blood cells in the vicinity of the arterial wall move to the central region so that the hematocrit ratio becomes quite low near the arterial wall which results in lower viscosity in this region. Blood viscosity and its major determinants such as plasma viscosity, fibrinogen (a protein involved in

clotting), may be an important risk factor for the early development of atherosclerosis [19, 20]. Atherosclerosis refers to the occlusion of the arterial lumen. The process starts from the build-up of cholesterol, fats and biological debris in the tissue lining the inside of blood vessels [37]. The build-up can obstruct blood to the heart and brain and thereby cause a heart attack or stroke. No single cause for atherosclerosis can be given but it seems to occur more frequently in those who are over weight due to eating, take too little exercise and are under stress and smoke. Therefore cigarette smoking and hypertension were independently associated with higher blood viscosity together with high pulse wave velocity [40]. Hence, smoking and hypertension may change the flow properties of the blood and the behavior of the arterial wall and this may explain the arterial damage observed in cigarette smokers and hypertensive patients [41, 42].

It has been shown that human blood is Newtonian at all rates of shear for hematocrit up to about 12%. At low shear rates blood has a higher viscosity than plasma, the viscosity of the suspension increases and non-Newtonian behaviour is observed as the hematocrit rises, studies of human blood show that viscosity is independent of shear rate when the rate is high with a reduction of 2% shear rate the viscosity increases slowly until a shear rate less than 15% where it rises steeply [17, 18]. At low shear rate the blood viscosity increased since red blood cells can easily form network of aggregated cell structures leading to high hematocrit level. At high shear rate the percentage of red blood cells is too low to produce the aggregate structures and no yield stresses will be found and blood viscosity is low. Blood viscosity in small vessels differs considerably from the viscosity in larger vessels. The progressive variation in blood viscosity with the size of the vessels

is more evident in vessels with a diameter of 0.1mm to 0.2mm. The variation in blood viscosity with vessel size is known as the Fahrawus and Lidquist effect. Moreover due to high shear rate near the arterial wall the viscosity of blood is further reduced in that region [2, 30]. Hence for flow problems in large blood vessels the blood may be treated as an incompressible fluid with variable viscosity, which takes its minimum value near the arterial wall and its maximum value along the central region of the artery.

1.2 HYDRODYNAMIC STABILITY THEORY

The subject of hydrodynamic stability deals mainly with the investigation of the time-space behavior of disturbances in fluid flows. It is a field of active research, because of its various applications in aerodynamics, engineering, geophysics, oceanography and meteorology. The chief stimulus for research in hydrodynamic stability is the need to understand the mechanism of transition to turbulence of laminar flows and the need to find possible ways of controlling the transition process. The mechanism of transition to turbulence of laminar flows is closely connected with the behavior of disturbances of finite amplitudes. Hydrodynamic stability emerged in the nineteenth century as a result of many studies [8, 32, 35]. The contribution by Reynolds [7] should however be emphasized. Through his series of classic experiments on the instability of flow in pipes, Reynolds showed that laminar flow breaks down when the Reynolds number (Re) exceeds a certain critical value - the critical Reynolds number (Re_c). In [4-6, 10, 16] the study of inviscid theory of stability of steady two-dimensional parallel flows was initiated. Rayleigh [6] deduced the celebrated theorem on the role of inflection points in the velocity profile, and this was independently verified by Reynolds in his also

celebrated experiments on transition to turbulence of flow in pipes. The viscous theory began with the work of Orr and Sommerfield as described in [9, 25] who independently derived the famous Orr-Sommerfield equation. The asymptotic methods of analysis of this equation were advanced notably by [12, 15, 26]. The analysis of the Orr-Sommerfield equation is rather involved and in any case well covered in the literature. Other works, notably [11, 26, 34] have pointed out that the flow structure can be described by subdividing the flow field into a multi-layer profile with several asymptotic zones. Meanwhile, the use of spectral methods to investigate the stability of various fluid flow problems has increased in recent years. The popularity of spectral methods comes from the fact that they have been proven to produce more accurate results than the finite difference and finite element in numerical schemes [13, 14]. In this project, we envisage using the spectral method in our analysis.

1.3 MECHANISM OF INSTABILITY

By saying that a system is stable, we actually mean that all the forces governing that system are in equilibrium. In fluid mechanics, these forces are the inertial, viscous, pressure or gravitational forces, say. If the fluid is disturbed in such a way that the forces acting on it are no longer in equilibrium, then instability sets in. Instability can occur for example when a heavier fluid lies on top of a lighter fluid; this will be mainly due to the effects of gravitational forces. From physical arguments, it can be conjectured that viscous forces can damp out small disturbances and hence act as stabilizing influences [8, 9]. However, comparison of the results in the viscous and inviscid studies reveals that viscosity can also serve as a cause for instability [4-6]. According to the studies of

Prandtl [16], viscous forces are capable of inducing Reynolds stresses absent in the non-viscous cases. If such stress converts energy from the basic flow into the disturbance, it could induce instability. Lin [21] later showed that this stress favours the conversion of energy into the disturbance motion.

1.4 SMALL DISTURBANCE CONCEPT

Theoretically speaking, our problem consists of following up in time the behaviour of hydrodynamically possible small wave-like disturbances which are imposed on the basic steady flow inside the blood vessel. The disturbance may originate, for example, at the inlet of a large artery or due to the arterial wall roughness. If the disturbance vanishes with time, the basic flow will be considered stable whereas if the disturbance increases with time, the flow will be considered unstable [8, 9]. We are concerned with small disturbances so that equations governing the behaviour of the disturbances can be linearised [21, 32, 34]. This implies that the quadratic or higher order terms in the disturbance properties and their derivatives will be very small and can be neglected. The linearization process greatly simplifies our task. We consider a linear system of partial differential equations and boundary conditions which have coefficients that vary in space but not in time (t). In such a case solutions containing an exponential factor such as e^{-ict} , for some complex number $c = c_r + ic_i$ (i.e. the phase speed of the disturbance) may be expected. We adopt here the method of normal modes, whereby each disturbance is resolved into dynamically independent wave components (modes). If there is a phase speed whose imaginary part is positive ($c_i > 0$) then e^{-ict} will increase indefinitely with time so that the corresponding disturbance is amplified, and the basic flow is unstable.

The mode is said to be neutrally stable if $c_i = 0$. We say the mode is asymptotically stable or just stable if $c_i < 0$.

1.5 AIM OF THE STUDY

This study aims to investigate theoretically the nature of variable viscosity arterial blood flow and its stability against small disturbances.

1.5.1 Specific objectives

- a) To develop a mathematical model for blood flow with variable viscosity in a large artery.
- b) To investigate the temporal stability of the basic flow against small disturbances.
- c) To investigate the effect of viscosity variation on the flow structure.
- d) To solve high-order linear ordinary differential equations numerically using the Chebyshev collocation spectral technique.
- e) To determine the critical values of flow parameters, for example flow Reynolds number, disturbance wave number and disturbance wave speed.

CHAPTER TWO

VARIABLE VISCOSITY ARTERIAL BLOOD FLOW: A MATHEMATICAL MODEL

2.1 SUMMARY

In this chapter, a mathematical model is developed by treating blood as an incompressible Newtonian fluid with viscosity which is lower near the vessel wall due to the presence of plasma layer in this peripheral region than the viscosity in the central core region which depends on the hematocrit. The governing equations of continuity and momentum are derived and solved. Graphical results are presented and discussed quantitatively.

2.2 INTRODUCTION

Blood together with its composition may be classified as an incompressible non-Newtonian fluid. However, in the course of flow in the large arteries, the red blood cells in the vicinity of the arterial wall move to the central region of the artery so that the hematocrit ratio becomes quite low near the arterial wall, which results in lower viscosity in this region [31, 39, 40]. Moreover, due to high shear rate near the arterial wall, the viscosity of blood is further reduced. Mathematically speaking, blood flow theory includes solving a system of partial differential equations subject to some boundary or initial conditions. The flow in the artery is three dimensional, but the mathematics is extremely difficult to handle. Consequently, some assumptions are made to simplify the

equations to tractable forms and at the same time preserving the characteristics of the flow. The two-dimensional model fairly reduces the problem [1-3, 27-29].

2.3 MATHEMATICAL MODEL

For the development of mathematical model (as illustrated in fig. 2.3.1 below), the following assumptions are made:

- (i) In the large artery, blood is assumed to be an incompressible Newtonian fluid.
- (ii) Due to the presence of plasma layer near the vessel wall, the local viscosity in this peripheral region would be close to that of the plasma and it would be lower than the viscosity in the central core region which depends on the hematocrit.
- (iii) A two-dimensional flow problem is considered.

The governing equations of continuity and momentum for axially symmetric flow of blood through an artery in dimensionless form under the above mentioned assumption are [1-3, 33, 36];

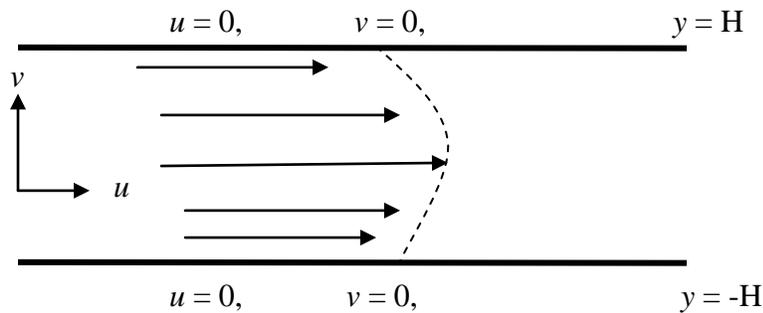


Figure 2.3.1: Geometry of the problem

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0, \quad (2.3.1)$$

$$\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} + v \frac{\partial u}{\partial y} = -\frac{\partial P}{\partial x} + \frac{2}{\text{Re}} \frac{\partial}{\partial x} \left(\mu \frac{\partial u}{\partial x} \right) + \frac{1}{\text{Re}} \frac{\partial}{\partial y} \left[\mu \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right) \right], \quad (2.3.2)$$

$$\frac{\partial v}{\partial t} + u \frac{\partial v}{\partial x} + v \frac{\partial v}{\partial y} = -\frac{\partial P}{\partial y} + \frac{2}{\text{Re}} \frac{\partial}{\partial y} \left(\mu \frac{\partial v}{\partial y} \right) + \frac{1}{\text{Re}} \frac{\partial}{\partial x} \left[\mu \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right) \right], \quad (2.3.3)$$

where x and y are the streamwise and normal coordinates respectively, u and v are the streamwise and normal velocity respectively, t is the time, P is pressure, Re is the Reynolds number. Based on the transverse variation in the hematocrit ratio within the blood vessel (see Figure 2.3.1), the variable viscosity function μ is modeled as;

$$\mu = \sec h(\beta y), \quad (2.3.4)$$

here β is the blood viscosity variation parameter. It is important to emphasize here that an increase in the positive values of β represents a decrease in the blood plasma viscosity in the periphery of the arterial wall. The governing Eqs. (2.3.1)-(2.3.3) have been non-dimensionalised using the following dimensionless variables:

$$\begin{aligned} u &= \frac{\bar{u}}{U}, \quad v = \frac{\bar{v}}{U}, \quad t = \frac{U\bar{t}}{H}, \quad x = \frac{\bar{x}}{H}, \quad y = \frac{\bar{y}}{H}, \quad P = \frac{\bar{P}}{\rho U^2}, \\ \mu &= \frac{\bar{\mu}}{\mu_0}, \quad \nu = \frac{\mu_0}{\rho}, \quad \text{Re} = \frac{UH}{\nu}, \quad G = -\frac{\partial P}{\partial x}, \quad \beta = H\gamma. \end{aligned} \quad (2.3.5)$$

where H is the channel characteristic half width, ρ is the fluid density, U is the velocity scale, ν is the hematocrit viscosity coefficient and G is the constant axial pressure gradient parameter.

BASIC FLOW: The basic steady state of the arterial blood flow system corresponds to a parallel flow with velocities $u = U(y)$ and $v = 0$. The equation and the boundary conditions describing the basic state are

$$\frac{d}{dy} \left(\mu \frac{dU}{dy} \right) = -G, \quad \frac{dU}{dy}(0) = 0, \quad U(1) = 0. \quad (2.3.6)$$

The solution is given by

$$U(y; \beta > 0) = \frac{G}{\beta^2} [\beta(\sinh(\beta) - y \sinh(\beta y)) + \cosh(\beta y) - \cosh(\beta)], \quad (2.3.7)$$

$$U(y; \beta \rightarrow 0) \approx \frac{G}{2}(1 - y^2) + \frac{G}{8}(1 - y^4)\beta^2 + O(\beta^4). \quad (2.3.8)$$

The volume flux is as follows

$$Q = \int_{-1}^1 u(y) dy \quad (2.3.9)$$

$$Q = \frac{-2G(2\beta \cosh(\beta) - \beta^2 \sinh(\beta) - 2\sinh(\beta))}{\beta^3} \quad (2.3.10)$$

The skin friction caused by the blood against the arterial wall is given by

$$\tau = \left. \frac{du}{dy} \right|_{y=1} = -G \cosh(\beta)$$

2.4 RESULTS AND DISCUSSION

The blood viscosity variation model and the flow basic velocity are computed from equations (2.4.1) and (2.4.2) for given fixed values of β and G . Figure (2.4.1) shows the transverse variation of arterial blood viscosity with maximum value in the central region and minimum value at the arterial wall. It is noteworthy that increasing values of viscosity variation parameter β leads to a decrease in the plasma viscosity near the arterial wall and an increase in the viscosity around the central region of the artery due to hematocrit concentration. Figure (2.4.2) illustrates the arterial blood velocity profile. Generally, a parabolic plane-Poiseuille profile is observed with maximum value along the centerline and minimum at the wall. However, an increase in the blood viscosity variation parameter causes a further increase in the blood velocity due to a decrease in plasma viscosity.

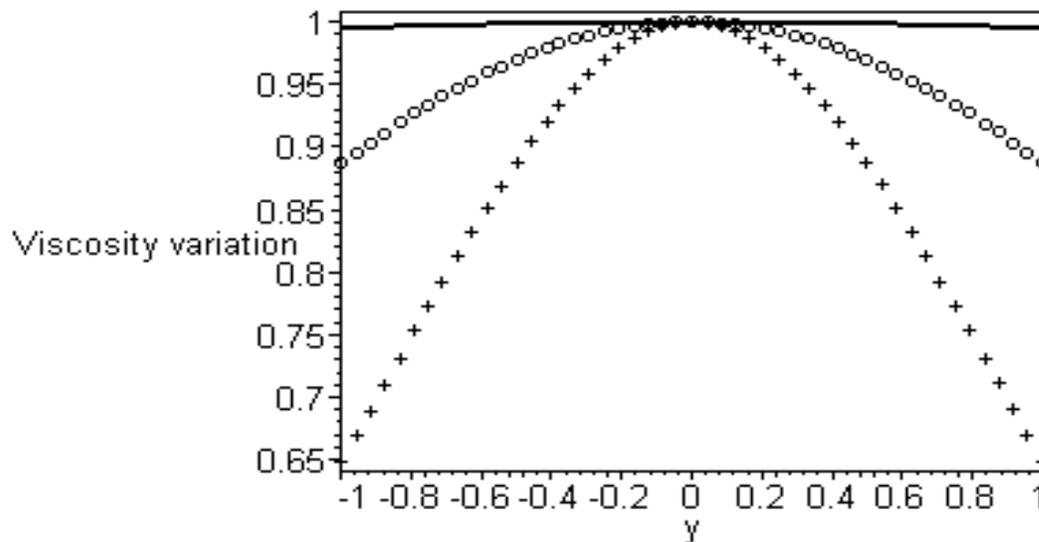


Figure 2.4.1: Arterial blood viscosity variation, _____ $\beta=0.1$, ooooo $\beta=0.5$, +++++ $\beta=1.0$

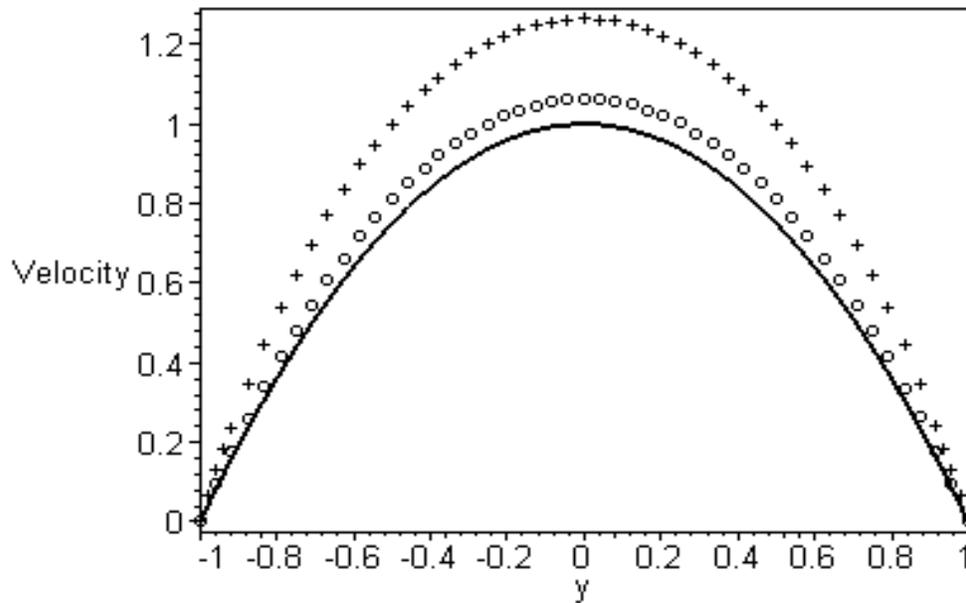


Figure 2.4.2: Velocity profile, $G = 2$; _____ $\beta=0$, ooooo $\beta=0.5$, +++++ $\beta=1.0$

Figure (2.4.3) depicts the volume flux across the arterial cross-section with increasing positive values of β . Interestingly, an increase in the blood volume flux across the artery is observed with a decrease in plasma viscosity near in the wall. Hence, more blood will flow downstream as β increases in values. Figure (2.4.4) illustrates the variation of arterial wall shear stress with increasing values of β . It is noteworthy that a decrease in the plasma viscosity near the arterial wall causes an increase in the skin friction, hence increasing the possibility of any frictional damage to the arterial wall.

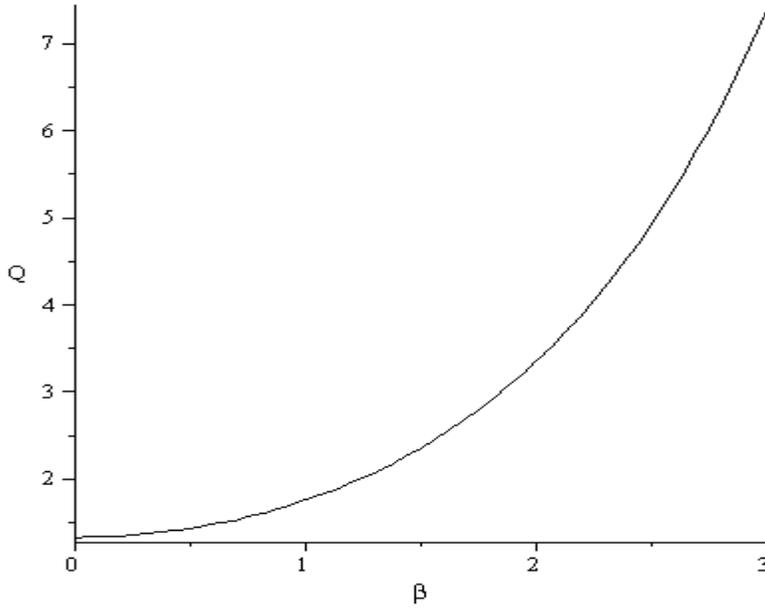


Figure 2.4.3: Variation of volume flux (Q) with blood viscosity parameter (β), $G=2$.

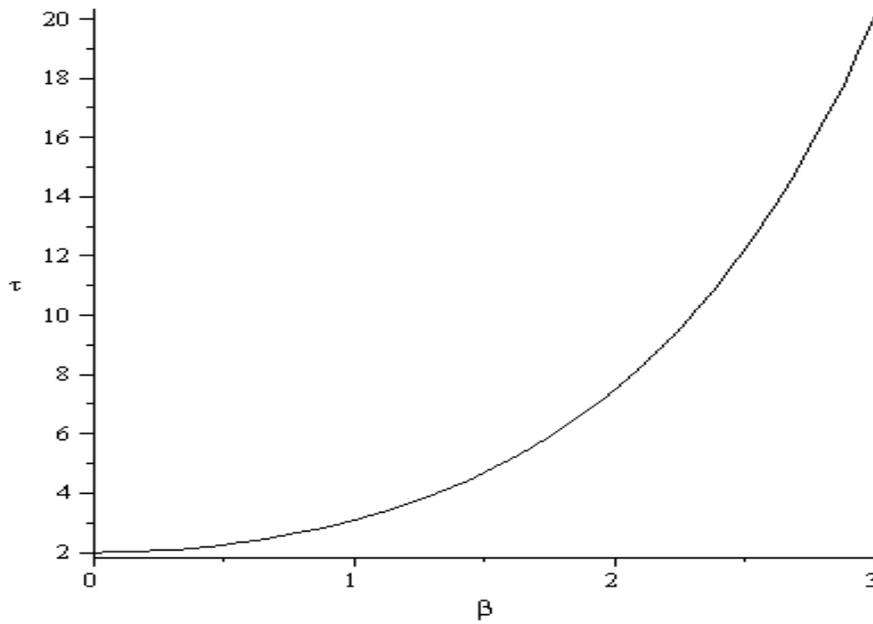


Figure 2.4.4: Variation skin friction (τ) with blood viscosity parameter (β), $G=2$.

CHAPTER THREE

ARTERIAL BLOOD FLOW: LINEAR STABILITY ANALYSIS

3.0 SUMMARY: In this chapter we derived the equation governing the temporal stability of small disturbances in a variable viscosity arterial blood flow. The equation is essentially a generalized eigenvalue problem.

3.1 INTRODUCTION

Here, we examine the temporal stability of the basic steady flow to small two dimensional disturbances in a variable viscosity arterial blood flow. The disturbance may originate in the area of a large artery maybe due to the arterial roughness. If the disturbance vanishes with respect to time then the basic flow will be considered stable and if the disturbance increases with time, the flow will be considered unstable. We are concerned with small disturbances so that equation governing the behavior of the disturbances can be linearised [8, 9, 21, 25, 26, 34]. This implies that the quadratic or higher order terms in the disturbance properties and their derivative will be very small and their derivative will be very small and can be neglected. Squire [35] made an important contribution to linear stability theory when he discovered that two-dimensional waves are the first to become unstable, and that an oblique wave always can be transformed into a two-dimensional wave associated with a lower Reynolds number, using the today well-known “Squire’s theorem.” This threw a smoke-screen over the important role of three-dimensionality, and had the rather counter-productive effect that most of the early work concerned only two dimensional waves.

3.2 DERIVATION OF STABILITY EQUATION

Consider the flow of variable viscosity blood flow in a large artery. In two dimensions, the governing equations as stated in the previous chapter are

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0, \quad (3.2.1)$$

$$\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} + v \frac{\partial u}{\partial y} = -\frac{\partial P}{\partial x} + \frac{2}{\text{Re}} \frac{\partial}{\partial x} \left(\mu \frac{\partial u}{\partial x} \right) + \frac{1}{\text{Re}} \frac{\partial}{\partial y} \left[\mu \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right) \right], \quad (3.2.2)$$

$$\frac{\partial v}{\partial t} + u \frac{\partial v}{\partial x} + v \frac{\partial v}{\partial y} = -\frac{\partial P}{\partial y} + \frac{2}{\text{Re}} \frac{\partial}{\partial y} \left(\mu \frac{\partial v}{\partial y} \right) + \frac{1}{\text{Re}} \frac{\partial}{\partial x} \left[\mu \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right) \right], \quad (3.2.3)$$

In the stability analysis, two-dimensional disturbances will be considered which implies that Squire's transformation [35] is applicable. Introducing small disturbances to the basic flow as follows:

$$u(x, y, t) = U(y) + \hat{u}(x, y, t), \quad v(x, y, t) = \hat{v}(x, y, t), \quad p(x, y, t) = P(x) + \hat{p}(x, y, t), \quad (3.2.4)$$

where \hat{u} , \hat{v} and \hat{p} are very small.

Eq. (3.2.4) is then substituted into Eqs. (3.2.1)-(3.2.3) and the nonlinear terms are neglected. We obtain

$$\frac{\partial \hat{u}}{\partial x} + \frac{\partial \hat{v}}{\partial y} = 0, \quad (3.2.5)$$

$$\frac{\partial \hat{u}}{\partial t} + U \frac{\partial \hat{u}}{\partial x} + \hat{v} \frac{dU}{dy} = -\frac{\partial \hat{P}}{\partial x} + \frac{2}{\text{Re}} \frac{\partial}{\partial x} \left(\mu \frac{\partial \hat{u}}{\partial x} \right) + \frac{1}{\text{Re}} \frac{\partial}{\partial y} \left[\mu \left(\frac{\partial \hat{u}}{\partial y} + \frac{\partial \hat{v}}{\partial x} \right) \right] \quad (3.2.6)$$

$$\frac{\partial \hat{v}}{\partial t} + U \frac{\partial \hat{v}}{\partial x} = -\frac{\partial \hat{P}}{\partial y} + \frac{2}{\text{Re}} \frac{\partial}{\partial y} \left(\mu \frac{\partial \hat{v}}{\partial y} \right) + \frac{1}{\text{Re}} \frac{\partial}{\partial x} \left[\mu \left(\frac{\partial \hat{u}}{\partial y} + \frac{\partial \hat{v}}{\partial x} \right) \right], \quad (3.2.7)$$

Following Orszag [14], we seek a normal mode solution for Eqs.(3.2.5)-(3.2.7) defined in terms of a stream-function as

$$\psi(x, y, t) = \phi(y)e^{i\alpha(x-ct)}, \quad (3.2.8)$$

where $\phi(y)$ is the amplitude function and c, α are the disturbances wave speed and wave number respectively. The disturbance velocity components can be expressed as follows:

$$\hat{u} = \frac{\partial \psi}{\partial y} = \phi'(y)e^{i\alpha(x-ct)}, \quad (3.2.9)$$

$$\hat{v} = -\frac{\partial \psi}{\partial x} = -i\alpha\phi(y)e^{i\alpha(x-ct)}, \quad (3.2.10)$$

where the prime symbol denotes differentiation with respect to y . Substituting Eqs.(3.2.8)-(3.2.10) into Eqs. (3.2.5) –(3.2.7) and eliminating the pressure terms yields

$$\begin{aligned} (U - c)(\phi'' - \alpha^2\phi) - U''\phi &= \frac{\mu}{i\alpha \text{Re}}(\phi^{iv} - 2\alpha^2\phi'' + \alpha^4\phi) \\ &+ \frac{2\mu'}{i\alpha \text{Re}}(\phi''' - \alpha^2\phi') + \frac{\mu''}{i\alpha \text{Re}}(\phi'' + \alpha^2\phi), \end{aligned} \quad (3.2.11)$$

with the boundary conditions

$$\begin{aligned} \phi(-1) &= \phi'(-1) = 0, \\ \phi(1) &= \phi'(1) = 0. \end{aligned} \quad (3.2.12)$$

Equation (3.2.11) is essentially a generalized eigenvalue problem written in the form

$$E\phi = cB\phi, \quad (3.2.13)$$

where the operators E and B are given by

$$\begin{aligned} E &= U(D^2 - \alpha^2) - U'' + \frac{i\mu}{\alpha \text{Re}}(D^4 - 2\alpha^2 D^2 + \alpha^4) \\ &+ \frac{2i\mu'}{\alpha \text{Re}}(D^3 - \alpha^2 D) + \frac{i\mu''}{\alpha \text{Re}}(D^2 + \alpha^2) \end{aligned} \quad (3.2.14a)$$

$B = (D^2 - \alpha^2)$ and

$$D^2 = \frac{d^2}{dy^2} \quad (3.2.14b)$$

It is noteworthy that Eq.(3.2.11) reduces to the classical Orr-Sommerfield equation [9, 21] when $\mu = 1$ which correspond to constant blood viscosity situation with $\beta = 0$. In order to find a non-trivial function ϕ satisfying Eq. (3.2.11) with boundary conditions (3.2.12), the parameters α , Re , β and c must satisfy a certain complex eigenvalue relation, say

$$F(\alpha, c, \beta, \text{Re}) = 0. \quad (3.2.15)$$

For temporal development of the disturbances, α is real and c is complex which can be expressed as

$$c = c_r(\alpha, \beta, \text{Re}) + ic_i(\alpha, \beta, \text{Re}) \quad (3.2.16)$$

The imaginary part of Eq. (3.2.16) determines whether the disturbances grow or decay. When $\alpha c_i > 0$ the disturbances grow; when $\alpha c_i = 0$ they neither grow nor decay, in this case the disturbance modes are said to be neutrally stable.

CHAPTER FOUR

NUMERICAL ANALYSIS, GRAPHICAL RESULTS AND DISCUSSION

4.0 SUMMARY: In this chapter we derived the discrete form of the generalized eigenvalue problem governing the temporal stability of small disturbances in variable viscosity arterial blood flow. Chebyshev collocation spectral method is employed to obtain numerical solutions for the problem. Graphical results and tables showing the computations for the most unstable mode, disturbance growth rate, critical Reynolds number R_{e_c} , the critical wave number α_c , the critical wave speed c_c and the marginal stability curve are obtained for a wide range of the blood viscosity variation parameter β .

4.1 CHEBYSHEV COLLOCATION SPECTRAL METHOD

In this section, we briefly describe the numerical approach employed in solving and analyzing the eigenvalue problem resulting from the stability analysis of flow in a channel saturated with a porous medium. Consider basis functions ϕ_j that are polynomials of degree $N-1$ satisfying $\phi_j(x_k) = \delta_{j,k}$ for the Chebyshev nodes

$$x_k = \cos \frac{(k-1)\pi}{(N-1)}, \quad k = 1, \dots, N, \quad (4.1.1)$$

Where $x_1 = 1$ and $x_N = -1$

(Note that $x_1 = 1$ and $x_N = -1$): The polynomial

$$p(x) = \sum_{j=1}^N \phi_{j+1}(x) u_j, \quad (4.1.2)$$

interpolates the points (x_j, u_j) , that is, $p(x) = u$. The values of the interpolating polynomial's d th derivative at the nodes are

$$p^{(d)}(x) = D^{(d)} u, \quad (4.1.3)$$

where the i, j th element of the differentiation matrix $D^{(d)}$ is $\phi_j^{(d)}(x_i)$. In the spectral collocation method for solving differential equations, the interpolating polynomial is required to satisfy the differential equation at the interior nodes. The values of the interpolating polynomial at the interior nodes are $p(x_{2:N-1}) = u_{2:N-1} = I_{2:N-1} u$ and the derivative values are $p^{(d)}(x_{2:N-1}) = D_{2:N-1}^{(d)} u$. Boundary conditions that involve the derivative can be handled by using the formulas

$$p^{(d)}(1) = D_1^{(d)} u, \quad p^{(d)}(-1) = D_N^{(d)} u. \quad (4.1.4)$$

Now we give a prescription for generating the collocation matrices. Let us denote $D = (d_{i,j})_{i,j=0,\dots,N-1}$ (see Canuto et al. [23]) the $N \times N$ matrix corresponding to the first derivative. It is explicitly given by

$$d_{i,j} = \begin{cases} \frac{c_i(-1)^{i+j}}{c_j(x_i - x_j)}, & i \neq j \\ -\frac{x_j}{2(1-x_j^2)}, & 1 \leq i = j \leq N-2 \\ \frac{2N^2+1}{6}, & i = j = 0 \\ -\frac{2N^2+1}{6}, & i = j = N-1 \end{cases} \quad (4.1.5)$$

and

$$c_i = \begin{cases} 2, & i \in \{0, N-1\} \\ 1, & i = 1, \dots, N-2 \end{cases} \quad (4.1.6)$$

The Differentiation Matrix Suite [24] provides useful Matlab functions for spectral collocation method using Chebyshev polynomials.

4.2 APPLICATION OF SPECTRAL COLLOCATION METHOD TO ARTERIAL BLOOD FLOW STABILITY PROBLEM

The eigenvalue problem derived in Chapter (3) will be solved using the Chebyshev spectral collocation method where the solution of the differential equation and its boundary conditions are expanded as a finite series in Chebyshev polynomials of the form

$$\phi(y) \approx \phi_N(y_j) = \sum_{k=0}^N \tilde{\phi}_k T_k(y_j), \quad j = 0, 1, \dots, N \quad (4.2.1)$$

where T_k is the k^{th} Chebyshev polynomial defined by

$$T_0(y) = 1, \quad T_1(y) = y, \quad T_{k+1}(y) - 2yT_k(y) + T_{k-1}(y) = 0, \quad (-1 \leq y \leq 1), \quad (4.2.2)$$

$\tilde{\phi}_k$ represents the unknown coefficients and y_j are the Gauss-Lobatto collocation points on $[-1, 1]$ defined by

$$y_j = \cos \frac{\pi j}{N}, \quad j = 0, 1, \dots, N. \quad (4.2.3)$$

Substituting equation (4.2.3) into (4.2.1) and requiring that the differential equation be satisfied at the $(N+1)$ collocation points. We obtain $(N+1) \times (N+1)$ algebraic equations which form the eigenvalue problem

$$E\phi = cB\phi, \quad (4.2.4)$$

where

$$\phi^T = (\tilde{\phi}_0, \tilde{\phi}_1, \dots, \tilde{\phi}_N), \quad (4.2.5)$$

is the transpose of the column vector ϕ . The clamped boundary conditions are incorporated explicitly in the first two and last rows of the matrices E and B by setting

the transpose of the column vector ϕ . The clamped boundary conditions are incorporated explicitly in the first two and last rows of the matrices E and B by setting

$$E(m, n) = \begin{cases} 1 & m = n = 0; \\ 0 & m = 0, n = 1, \dots, N; \\ \sum_{n=0}^N D_{0n} & m = 1, n = 0, \dots, N; \\ \tilde{E}(m, n) & m = 1, \dots, N-2, n = 0, \dots, N; \\ \sum_{n=0}^N D_{Nn} & m = N-1, n = 0, \dots, N; \\ 0 & m = N, n = 1, \dots, N-1; \\ 1 & m = N, n = N; \end{cases} \quad (4.2.6)$$

$$B(m, n) = \begin{cases} 0 & m = 0, 1, N-1, N, n = 0, \dots, N; \\ \tilde{B}(m, n) & m = 2, \dots, N-2, n = 0, \dots, N; \end{cases} \quad (4.2.7)$$

where

$$\begin{aligned} \tilde{E} = & U(D^2 - \alpha^2 I) - U'' + \frac{i\mu}{\alpha \operatorname{Re}} (D^4 - 2\alpha^2 D^2 + \alpha^4 I) \\ & + \frac{2i\mu'}{\alpha \operatorname{Re}} (D^3 - \alpha^2 D) + \frac{i\mu''}{\alpha \operatorname{Re}} (D^2 + \alpha^2 I) \end{aligned} \quad (4.2.8)$$

$$\tilde{B} = (D^2 - \alpha^2 I) \quad (4.2.9)$$

$U = \operatorname{diag}[U(y_j)]$, I is the $(N+1) \times (N+1)$ identity matrix and D is the usual differential matrix (Canuto et al. [23]). Here $\operatorname{diag}[\]$ means that the entries placed on the main diagonal of an $(N+1) \times (N+1)$ matrix with the rest of the entries being zero. Using this approach, results in the matrix B being singular. The problem is avoided by employing the idea of Weidmann and Reddy [24] i.e. using Hermite interpolating polynomials that satisfy the boundary conditions, thus, we obtain

$$\tilde{\phi}_0 = 0, \quad \sum_{n=0}^N D_{0n} \tilde{\phi}_n = 0 \quad \text{on } y = 1, \quad (4.2.10)$$

$$\tilde{\phi}_N = 0, \quad \sum_{n=0}^N D_{Nn} \tilde{\phi}_n = 0 \quad \text{on } y = -1. \quad (4.2.11)$$

4.3 RESULTS AND DISCUSSION

Chebyshev spectral collocation method is implemented in MATLAB 5.1 to compute the fastest growing mode although there is no reason to believe that more than one mode of the present problem grows for given fixed values of β , G , α and Re . The convergence rate of the spectral method employed in the numerical experiment is demonstrated in Table 4.3.1. It is interesting to note that at $N=70$ to $N=100$ the imaginary part of the most unstable mode converge up to 8 digits and from $N= 80$ to $N=100$ the convergence rate increased up to imaginary10 digits. Hence, the convergence rate increases with an increase in the size of derivative matrix and the collocation points employed in the computation. Table 4.3.2 shows the numerical results for the eigenvalues of the most unstable mode for increasing values of β at fixed values of G , α and Re . It is interesting to note that a slight increase in the values of β due to a decrease in blood plasma viscosity near the arterial wall has the effect of decreasing the imaginary parts of the wavespeed. This shows that an increase in a decrease in blood plasma viscosity has a stabilising effect on the flow. decrease in blood plasma viscosity near the arterial wall acts like a control parameter that eliminates the growth of small disturbances in the flow field.

Table 4.3.1: Computations showing the convergence of the procedure most unstable mode ($G = 2$, $R_e = 10000$, $\alpha=1, \beta =1$)

N	c (waves speed)
10	0.56720767556780 - 0.00294408098869i
20	0.25611034993082 - 0.00929922067276i
30	0.26848937286877 - 0.00010690738790i
40	0.26846507294410 - 0.00143442422134i
50	0.26837157858502 - 0.00145411481056i
60	0.26836641964802 - 0.00145379743103i
70	0.26836621819964 - 0.00145385265964i
80	0.26836621533910 - 0.00145385712210i
90	0.26836621537906 - 0.00145385722646i
95	0.26836621539255 - 0.00145385720451i
100	0.26836621553378 - 0.00145385703953i

Table 4.3.2: Computations showing the eigenvalue of the most unstable mode ($G=2$, $R_e = 10000$, $R_e=20000$, $\alpha = 1, N=100$)

β (waves number)	c (waves speed)
0.0000000000	0.23752648888092 + 0.00373967084384i
0.1000000000	0.23783691596441 + 0.00364708008511i
0.2000000000	0.23876667964504 + 0.00337568670771i
0.3000000000	0.24031185075161 + 0.00294413963135i
0.4000000000	0.24246778028024 + 0.00238189269759i
0.5000000000	0.24523135666099 + 0.00172677584590i
0.6000000000	0.24860316820788 + 0.00102176838274i
0.7000000000	0.25258895109069 + 0.00031134840173i
0.8000000000	0.25719998734185 - 0.00036202427177i
0.9000000000	0.26245247446899 - 0.00096074513624i
1.0000000000	0.26836621553378 - 0.00145385703953i
5.0000000000	15.99128665570440 - 0.03365818644236i
6.0000000000	38.32239623023935 - 0.01462612066866i

Table 4.3.3: Computations showing the eigenvalue of the most unstable mode ($G=2$, $R_e = 10000$ $R_c=20000$, $\beta = 1$, $N=100$)

α (waves number)	c (waves speed)
0.1000000000	0.54918892627669 - 0.09958491169248i
0.2000000000	0.46793110681176 - 0.07316298645205i
0.3000000000	0.42472646669667 - 0.06278459365044i
0.4000000000	1.20834227248723 - 0.05566598516907i
0.5000000000	0.18822548792957 - 0.04368189196264i
0.6000000000	0.21067169267241 - 0.02782403998107i
0.7000000000	0.22896852233968 - 0.01408636148472i
0.8000000000	0.24428709628862 - 0.00518093718562i
0.9000000000	0.25743106050596 - 0.00116903915401i
1.0000000000	0.26836621553378 - 0.00145385703953i
5.0000000000	1.24828993857017 - 0.01482611451890i

Table 4.3.3 shows that increasing values of the disturbance wave number is destabilizing. The critical Reynolds number Re_c and the critical wave number α_c at the instability threshold for varying values of β are shown in Table 4.3.4. For $\beta=0$, the result obtained is in perfect agreement with the one reported in [14]. We observe that an increase in β leads to an increase in the critical Reynolds number and a slight decrease in the critical wavespeed. This means that the stable region in (Re, α) -plane increases as the plasma viscosity decreases (see Figure 4.3.2). Figure 4.3.1 shows the variation in the growth rate of the most unstable mode against the wavenumber. It is interesting to note that increasing values of β have the effect of damping the disturbances.

Table 4.3.4 Computations showing the critical value at which unstable modes begin to exist ($G=2$)

β	α	R_{ec}
0.0	1.02052	5772.2283
0.1	1.00260	5869.2054
0.2	1.01260	6053.6434
0.3	1.00955	6408.1816
0.4	0.99225	6886.2840
0.5	0.98561	7492.1303
0.6	0.96705	8176.7969
0.7	0.95429	8915.4290
0.8	0.94512	9649.2259
0.9	0.94015	10323.7569
1.0	0.93110	10878.4282
2.0	0.90211	84799.4481

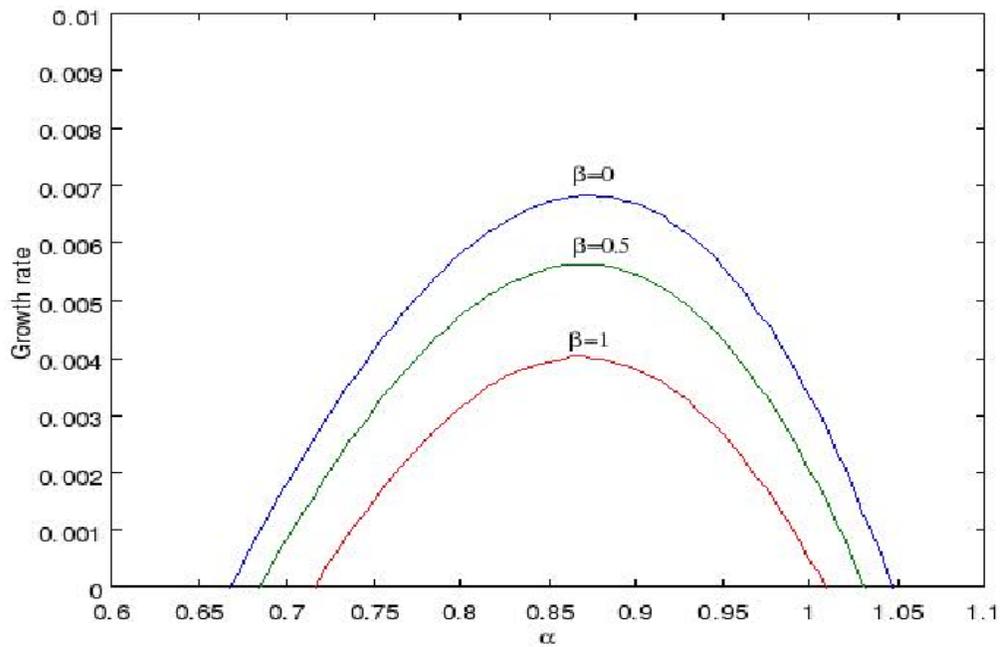


Figure 4.3.1: Growth rate α_i for $Re = 20000$.

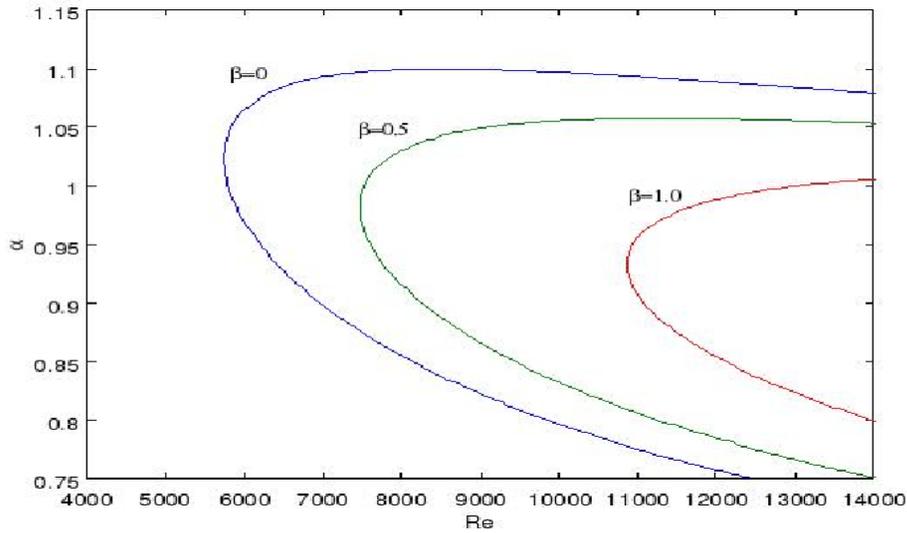


Figure 4.3.2: Marginal stability curve for $G = 2$.

4.4 CONCLUSIONS

The Chebyshev spectral collocation method implemented in MATLAB is employed to investigate the temporal development of small disturbances in a variable viscosity arterial blood flow due to variation in plasma viscosity. We obtained accurately the critical Reynolds number Re_c and the critical wave number α_c for increasing positive values of blood viscosity variation parameter. The velocity increases with increasing values of viscosity parameter (β). Increasing values of viscosity parameter (β) also produce a decrease in the disturbances growth rate. Finally, it is observed that a decrease in plasma viscosity near the arterial wall has a stabilizing effect on the flow.


```

disp(' *****')
disp(' *')
disp(' *          Renold Number = 10000')
disp(' *          Alpha = 1')
disp(' *          N = 100')
disp(' *****')
disp(' ')
beta = input(' YOU CAN GIVE THE VALUE OF BETA= ');
disp(' ')
disp(' ')

if(beta==0)
    U= diag((-1/2*G)*y.^2 + (1/2)*G) + (((-1/8)*G)*y.^4 +
(1/8)*G)*beta^2);
    U2= diag((-G)+(-3/2*G*y.^2)*beta^2);

else
    U = diag((-G*y.*sinh(beta*y)/beta) + (G*cosh(beta*y)/(beta^2))+
((G*sinh(beta))/beta)-(G*cosh(beta))/(beta^2));
    U2 = diag((-G*cosh(beta*y))-((G*beta)*y).*sinh(beta*y));
end

M = diag(sech(beta*y));
M1 = diag(beta*(-sech(beta*y)).*tanh(beta*y));
M2 = diag((beta^2)*(sech(beta*y)).*(tanh(beta*y).^2) - sech(beta*y).*(1-
tanh(beta*y).^2));

A = U*(D2 - alpha^2*I)- U2*I + (i*M/(alpha*R))*( D4 - (2*alpha^2)*D2 +
alpha^4*I) + i*((2*M1)/(alpha*R))*( D3 - (alpha^2)*D) +
(i*M2/(alpha*R))*(D2 + (alpha^2)*I);

B = (D2- alpha^2*I);

A(1,:)=0;
A(N+1,:)=0;
B(1,:)=0;
B(N+1,:)=0;
A(2,:)=D(1,:);
A(N,:)=D(N+1,:);
B(2,:)=0;
B(N,:)=0;
A1=A(2:N,2:N);
B1=B(2:N,2:N);

V=eig(A1,B1);
V=V(5:N-5);
[kk,jj]=max(imag(V));
cc=V(jj);

disp(' ')
disp(' The most Unstable eigenvalue is ');
disp(' ')
disp(cc)
disp(' ')
disp(' GOD BLESS YOU ')

```



```

end

M = diag(sech(beta*y));
M1 = diag(beta*(-sech(beta*y)).*tanh(beta*y));
M2 = diag((beta^2)*(sech(beta*y)).*(tanh(beta*y).^2) - sech(beta*y).*(1-
tanh(beta*y).^2));

A = U*(D2 - alpha^2*I)- U2*I + (i*M/(alpha*R))*( D4 - (2*alpha^2)*D2 +
alpha^4*I) + i*((2*M1)/(alpha*R))*( D3 - (alpha^2)*D) +
(i*M2/(alpha*R))*(D2 + (alpha^2)*I);

B = (D2- alpha^2*I);

A(1,:) = 0;
A(N+1,:) = 0;
B(1,:) = 0;
B(N+1,:) = 0;
A(2,:) = D(1,:);
A(N,:) = D(N+1,:);
B(2,:) = 0;
B(N,:) = 0;

A1 = A(2:N, 2:N);
B1 = B(2:N, 2:N);

V = eig(A1, B1);
V = V(5:N-5);
[kk, jj] = max(imag(V));
cc = V(jj);

disp('
')
disp('    The most Unstable eigenvalue is ');
disp('
')
disp(cc)
disp('
')
disp('                                GOD BLESS YOU                                ')
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

```

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
% The program komaneN.m computes the eigenvalues of the Orr-
Sommerfeld
% of the arterial blood flow, you input in the value of N and it will
% give you the most unstable eigenvalue and alpha is fixed at 1 also
beta=1.
% equation using N+1xN+1 Chebyshev differentiation matrices.
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

disp(' ')

N = input(' YOU CAN GIVE THE VALUE OF N= ');
disp(' ');
[D,y]=cheb(N);
D2=D^2;
D3=D^3;
D4=D^4;
I=eye(N+1);

G=2;
alpha =1;
R= 10000;
beta=1;

disp(' ')
disp(' ')
disp(' This Programs gives us the most unstable eigenvalue at difference
N value ')

disp(' ')
disp(' *****')
disp('*')
disp('* Renold Number = 10000')
disp('* Alpha = 1')
disp('* Beta = 1')
disp(' *****')
disp(' ')

disp(' ')
disp(' ')

if(beta==0)
    U= diag(((1/2)*G)*y.^2 + (1/2)*G) + (((-1/8)*G)*y.^4 +
(1/8)*G)*beta^2);
    U2= diag((-G)+(-3/2*G*y.^2)*beta^2);
else
    U = diag((-G*y.*sinh(beta*y)/beta) + (G*cosh(beta*y)/(beta^2))+
((G*sinh(beta))/beta)-(G*cosh(beta))/(beta^2));
    U2 = diag((-G*cosh(beta*y))-((G*beta)*y).*sinh(beta*y));
end

M = diag(sech(beta*y));
M1 = diag(beta*(-sech(beta*y)).*tanh(beta*y));

```

```

M2 = diag((beta^2)*(sech(beta*y).*(tanh(beta*y).^2) - sech(beta*y).*(1-
tanh(beta*y).^2)));

A = U*(D2 - alpha^2*I)- U2*I + (i*M/(alpha*R))*( D4 - (2*alpha^2)*D2 +
alpha^4*I) + i*((2*M1)/(alpha*R))*( D3 - (alpha^2)*D) +
(i*M2/(alpha*R))*(D2 + (alpha^2)*I);

B = (D2- alpha^2*I);

A(1,:) = 0;
A(N+1,:) = 0;
B(1,:) = 0;
B(N+1,:) = 0;
A(2,:) = D(1,:);
A(N,:) = D(N+1,:);
B(2,:) = 0;
B(N,:) = 0;

A1 = A(2:N, 2:N);
B1 = B(2:N, 2:N);

V = eig(A1, B1);
V = V(5:N-5);
[kk, jj] = max(imag(V));
cc = V(jj);

disp(' ')
disp(' The most Unstable eigenvalue is ');
disp(' ')
disp(cc)
disp(' ')
disp(' GOD BLESS YOU ')
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
% The script file komane.m computes the eigenvalues of the Orr-
Sommerfeld
% and it has been used or called by different file to be runed.
% This program is important to execute other programs.
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

N=100;
[D, y] = cheb(N);
D2 = D^2;
D3 = D^3;
D4 = D^4;
I = eye(N+1);

```

```

G=2;

if(beta==0)
    U= diag(((1/2)*G)*y.^2 + (1/2)*G) + (((-1/8)*G)*y.^4 +
(1/8)*G)*beta^2);
    U2= diag((-G)+(-3/2*G*y.^2)*beta^2);

else
    U = diag((-G*y.*sinh(beta*y)/beta) + (G*cosh(beta*y)/(beta^2))+
((G*sinh(beta))/beta)-(G*cosh(beta))/(beta^2));
    U2 = diag((-G*cosh(beta*y))-((G*beta)*y).*sinh(beta*y));
end

M = diag(sech(beta*y));
M1 = diag(beta*(-sech(beta*y)).*tanh(beta*y));
M2 = diag((beta^2)*(sech(beta*y)).*(tanh(beta*y).^2) - sech(beta*y).*(1-
tanh(beta*y).^2));

A = U*(D2 - alpha^2*I)- U2*I + (i*M/(alpha*R))*( D4 - (2*alpha^2)*D2 +
alpha^4*I) + i*((2*M1)/(alpha*R))*( D3 - (alpha^2)*D) +
(i*M2/(alpha*R))*(D2 + (alpha^2)*I);

B = (D2- alpha^2*I);

A(1,:)=0;
A(N+1,:)=0;
B(1,:)=0;
B(N+1,:)=0;
A(2,:)=D(1,:);
A(N,:)=D(N+1,:);
B(2,:)=0;
B(N,:)=0;

A1=A(2:N,2:N);
B1=B(2:N,2:N);

V=eig(A1,B1);
V=V(5:N-5);
[kk,jj]=max(imag(V));

pp= max(imag(V));

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

```

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%% This program plots the rate of growth curve. This program calls
%% komane.m
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
vstore=[];
aa=[];
vstore=[vstore,0];
aa=[aa,0];
R=20000;
astep=0.005;
beta=0;
for alpha = 0.5:astep:1.1,
    komane;
    vstore=[vstore,alpha*pp];
    aa=[aa,alpha];
end

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

```

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%% This program komaneR.m computes the Reynolds number on the
%% marginal stability curve for a given value of the wavenumber alpha
%% and beta it call komane.m program.
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

```

error = 0;
Rstepdiv=5;
Rstepinitial=100;
Rmax=100100;
Rstepmin=19;
itermax=10000;
itertolerance=0.0000001;
Rstep = Rstepdiv * Rstepinitial;
Rmin=4000;
R = Rmin;
Xa = Rmin;
disp(' ')
beta = input(' YOU CAN GIVE THE VALUE OF BETA= ');
disp(' ')
alpha = input(' YOU CAN GIVE THE VALUE OF ALPHA = ');
disp(' ')
komane;

Ya = max(imag(V));
if Ya > 0
    error = 1;
    error('initial Reynolds to large ...');
end

```

```

Yb = -1;
while (Rstep > Rstepmin & Yb < 0)
    Rstep = Rstep/Rstepdiv;
    R = Rmin;
    while (Yb < 0 & R < Rmax),
        R = R + Rstep;
        komane;
        Yb = max(imag(V));
    end
end
Xb = R;
if Yb < 0,
    error = 1;
    error('localisation fails ...');
end
Y = Yb;
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%                               CONVERGENCE TO THE ROOT
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
iterorr2=0;
arretnewton=0;
while (iterorr2 < itermax & abs(Y) > itertolerance),
    R = Xa - Ya*(Xb-Xa)/(Yb-Ya);
    komane;
    Y = max(imag(V));
    if Y >= 0
        Xb = R;
        Yb = Y;
    else
        Xa = R;
        Ya = Y;
    end
    iterorr2 = iterorr2+1;
end
Rec = R;
disp('    The Reynolds number is ');
disp('                                     ')
disp(R)
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

```

%*****
%*****
% The program name is komane2.m its calls 'renoldnumber.m'
% It compute the critical value at which the unstable modes
% begin to exist.
% As you run the program you input the value of beta first
% and type the word 'alpha' to get the wave number at which
% the most unstable occure andtype the word 'Rec' to find
% the critical value at which the unstable modes begin to exist
%*****

Recerr = 100000;
RR0=100000;
Rmin=5000;
alpha =1.01;

disp(' ***** ')
disp(' * It compute the critical value at which the ')
disp(' * unstable modes begin to exist ')
disp(' * ')
disp(' ')
disp(' Type "Rec" for reynold Number or "alpha" for the wave Number')

disp(' ')
beta = input(' YOU CAN GIVE THE VALUE OF BETA= ');
disp(' ')
disp(' ')
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%% This is the increment value looping.
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
steph=0.0001;

while (Recerr > 0),
    renoldnumber;
    Recerr = RR0-Rec;
    RR0=Rec;
    alpha = alpha + steph;
end

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

```

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%% This program renoldnumber.m computes the corresponding Reynolds
%% number on the marginal stability curve for a given value of the
%% wavenumber alpha and beta it call komane.m program.
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

error = 0;
Rstepdiv=5;
Rstepinitial=100;
Rmax=100100;
Rstepmin=19;
itermax=10000;
itertolerance=0.0000001;
Rstep = Rstepdiv * Rstepinitial;
Rmin=4000;
R = Rmin;
Xa = Rmin;

komane;

Ya = max(imag(V));
if Ya > 0
    error = 1;
    error('initial Reynolds to large ...');
end
Yb = -1;
while (Rstep > Rstepmin & Yb < 0)
    Rstep = Rstep/Rstepdiv;
    R = Rmin;
    while (Yb < 0 & R < Rmax),
        R = R + Rstep;
        komane;
        Yb = max(imag(V));
    end
end
Xb = R;
if Yb < 0,
    error = 1;
    error('localisation fails ...');
end
Y = Yb;

iterorr2=0;
arretnewton=0;
while (iterorr2 < itermax & abs(Y) > itertolerance),
    R = Xa - Ya*(Xb-Xa)/(Yb-Ya);
    komane;
    Y = max(imag(V));
    if Y >= 0
        Xb = R;
        Yb = Y;
    else

```

```

Xa = R;
Ya = Y;
end
    iterorr2 = iterorr2+1;
end
Rec = R;

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%% This program komane4.m plot the graph of the wave number verses
%% the Reynolds number. It shows the region of the stability at
%% difference value of beta, It call komane.m program and renoldnumber.
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

disp('
    beta = input(' YOU CAN GIVE THE VALUE OF BETA= ');
disp('

alpha = 0.75;
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
renoldnumber;
R = Rec;
ainit = alpha;
Rinit = R;
a0 = alpha;
R0 = R;
normgradient = 10;
curvepoints=500;
curvenumber =0;
curvegradient=0.00001;
dR=0.1;
da=0.001;
curvestep=0.005;
for Icurve = 1:curvepoints,
    criticalrey(Icurve) = 0;
end
while (normgradient > curvegradient & curvenumber < curvepoints ),
    curvenumber = curvenumber + 1;
% Estimation of dc / dR
R = R0;
alpha = a0;
komane;
c0 = pp;
R = R0 + dR;
alpha = a0;
komane;

```

```

c = pp;
dcdR = (c - c0)/dR*10000;
R = R0;
alpha = a0 + da;
komane;
c = pp;
dcda = (c - c0)/da;
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
normgradient = sqrt(dcda*dcda + dcdR*dcdR);
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
a0 = a0 + dcdR/normgradient*curvestep;
R0 = R0 - dcda/normgradient*curvestep*10000;
criticalrey(curvenumber) = R0 + i * a0;
end
nn=real(criticalrey);
mm=imag(criticalrey);
plot(nn,mm)

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

4.7 Reference

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