

NUMERICAL STUDIES OF FLOW THROUGH
PROSTHETIC HEART VALVES



by

Kym Thalassoudis

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University of Adelaide

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SUMMARY

Computational fluid dynamic techniques are applied to numerically study the characteristics of steady flow through prosthetic heart valves in order to identify separated flow and high fluid stress regions known to cause the pathophysiological problems of thrombosis, haemolysis, sub-haemolytic injuries and endothelial erosion. The valves considered are the Kay-Shiley caged-disk valve and the Starr-Edwards caged-ball valve.

Three numerical models are constructed, each introducing an improvement upon the previous. The first model investigated is for steady laminar flow through the idealized planar geometry of a disk-type valve. Although very simple, this model clearly demonstrates the potential effectiveness and usefulness of more sophisticated numerical and mathematical models. Numerical solutions are provided for several Reynolds numbers.

The second model is an extension of the first model into the turbulent flow regime, examining steady turbulent axisymmetric flow through a Kay-Shiley disk valve. Experimental research indicates that turbulent flow occurs consistently in the ascending aorta of subjects with prosthetic aortic valves. Therefore, the effects of turbulence are incorporated into this numerical model through the $k - \epsilon$ model of turbulence embedded in the TEACH-T computer code. Streamlines, mean axial velocity profiles, turbulent shear stresses and wall shear stress distributions are computed for Reynolds numbers between 600 and 10000. The location, length and maximum reverse-flow velocities of separated flow regions are computed and compared with experimental observations. The numerical solutions are shown to compare favourably with available experimental measurements.

The third model examines steady turbulent axisymmetric flow through a Starr-Edwards ball valve in an aortic-shaped chamber. This prosthesis is the most widely known caged-ball valve and is in use in many centers around the world including Australia. A boundary-fitted coordinate system is generated for the flow domain in the near vicinity of the valve. The coordinate lines follow the left ventricular wall, the sewing ring, an idealized sinus, the aortic wall and the occluder shape. The $k-\epsilon$ turbulence model, successfully used in the previous numerical model, is formulated into a stream function-vorticity approach. The governing partial differential equations are recast into their curvilinear equivalents before being discretised into finite difference equations which are then solved iteratively. Numerical solutions are obtained at physiologically relevant Reynolds numbers and compared to experimental measurements. There is good agreement for the mean velocity fields but turbulent stresses are underpredicted.

Through the application of numerical modelling techniques this research has provided valuable and detailed quantitative data for velocities and stress fields in the vicinity of prosthetic heart valves. The long term application of this research is seen both as an effective design tool and in assisting cardiac surgeons in the proper selection of valve implants for individual patients.

SIGNED STATEMENT

The contents of this thesis have not been submitted to any university for the purpose of obtaining any other degree or diploma, and to the best of my knowledge, it contains no material previously published by any other person, except where due reference is made in the text.

Kym Thalassoudis

PREFACE

I wish to express my sincerest appreciation to Dr. J. Mazumdar for his continued guidance and support during the course of this research. His encouragement and constructive comments made this work possible. Also, I am thankful to Dr. B.J. Noye of the Department of Applied Mathematics for initially encouraging me to pursue this topic and for making available the benefit of his advice on many occasions, and to Dr. I.H. Craig of the Department of Medicine for making available X-ray films of implanted prosthetic valves.

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CHAPTER 1

INTRODUCTION

1.1 General Introduction

Heart valve disease in advanced forms severely affects the performance of a natural heart valve by causing permanent alteration to the valve tissue leading to valve dysfunction. An improper functioning heart valve induces considerable disability and may ultimately lead to death if the problem is not treated. The need for surgical repair or total valvular replacement arises when a patient's quality of life suffers from the ensuing disability. Biological and prosthetic valve substitutes have been employed since the early 1960s when valve replacement was first introduced as a clinical procedure. Today, mechanical prostheses and tissue valves (bioprostheses) are in widespread clinical use in medical centres throughout the world.

Despite over 25 years of development and clinical experience prosthetic heart valves continue to suffer from numerous pathological problems. The ideal valve substitute has as yet to be designed. Although many difficulties can be expected during and immediately following a valve implant, the most debilitating and life-threatening complications are those that begin to develop from the moment of implant and manifest themselves in latter years.

The presence of the prosthesis disturbs the flow of blood producing areas of high fluid stress, high wall stress and separated flow regions. Depending upon their extent, these flow conditions may eventually cause many serious pathological problems including sub-haemolytic injury and haemolysis, endothelial damage and erosion, thrombotic deposition and related thromboembolic events. There is a di-

rect link between the fluid dynamics of heart valves and the pathological problems encountered. Hence, it is extremely important to examine the likelihood of these problems by investigating as fully as possible the fluid dynamics of prosthetic heart valves.

Measurements using *in vitro* experiments provide much valuable information. However, physical measurements are often limited in application due to probe size or are simply not suitable for many regions of the flow domain. This has been the experience of previous researchers in this field (Figliola and Mueller, 1981; Herkes and Lloyd, 1981). Experimental measurements are unable to provide a complete picture of the complex flow patterns.

Computational fluid dynamic techniques provide a powerful noninvasive tool for obtaining good approximations to velocity and stress distributions in a flow field. However, depending upon the nature of the fluid dynamics, the mathematical equations and the discrete modelling, the numerical solutions can range from very accurate to only rough approximations. Current numerical techniques provide acceptably good results for most physical flow problems. Only the more complex flow cases cause difficulties (e.g. turbulent pulsatile flow). The advent of high speed digital computers and ready access to modern computing power has generated strong interest in the development of numerical models to investigate prosthetic valve fluid dynamics. Previous computational research in this field has for the most part concentrated on laminar flow and/or idealized valve geometries. However, in reality, a complex combination of both laminar and turbulent flow and its relaminarization is observed through prosthetic valves during the cardiac cycle. Furthermore, the tissue walls are generally curved.

Therefore, in this thesis the application of computational methods to elucidate

the complex fluid dynamics associated with steady flow through prosthetic heart valves will be examined by including turbulence effects and accurate boundary shape.

1.2 Brief Description of the Heart and its Valves

The human heart is a pulsatile synchronous pump which beats more than 40 million times each year. Its function is to provide the mechanism by which the oxygen requirements of the human body are satisfied by maintaining the circulation of blood.

The human heart has four chambers — two chambers pumping blood and two chambers receiving blood. The right and the left ventricles are the two separate pumping chambers of the heart, pumping blood to the lungs and the body through two large blood vessels. The vessel supplying the lungs is the pulmonary artery, and that supplying the body is the aorta. The two receiving chambers in the heart are the left and right atria. The one receiving oxygenated blood from the lungs is the left atrium, and that receiving deoxygenated blood from the veins of the body is the right atrium.

The human heart has four valves, two on each side. The valves on the right side are the tricuspid valve – an inlet valve, and the pulmonary valve – an outlet valve. The valves on the left side are the mitral valve – an inlet valve, and the aortic valve – an outlet valve. In the natural heart, the valve leaflets open at the centre to allow unobstructed central flow. The action of the four heart valves within the heart acts to restrict blood flow to one direction and prevent substantial backflow.

The aortic and pulmonary valves each consist of three cusps attached to a fi-

brous tissue ring on the inner walls of the aorta and pulmonary artery, respectively. Both outlet valves have similar dimensions. The adult aortic or pulmonary valve is approximately 25 mm in diameter. Behind each cusp is a pouch-like cavity embedded into the artery wall. These regions are known as the sinuses of Valsalva. An important mechanism in aortic valve closure is the production of vortices in the region between each cusp and its sinus. Bellhouse and Talbot (1969) demonstrated this behaviour by constructing a trileaflet valve mounted in a rigid chamber with a model aortic sinus.

The mitral and tricuspid valves are also similar to each other. However, the mitral valve has two major and two minor leaflets, whereas the tricuspid valve has three similarly sized leaflets. Unlike the other valves, the circumference of the mitral valve is not circular but “D-shaped”, measuring approximately 35 mm across the major diameter. A cutaway view of the heart illustrating anatomical relationships is shown in Figure 1.2.1.

After circulating through the body, deoxygenated blood collects in the great veins. It then flows into the right atrium and through the tricuspid valve into the right ventricle. The right ventricle contracts, and together with the closure of the tricuspid valve, the deoxygenated blood is ejected through the pulmonary valve into the pulmonary artery. The blood flows to the lungs where the transfer of carbon dioxide and oxygen occurs. Four pulmonary veins then direct the reoxygenated blood into the left atrium where the blood flows through the mitral valve into the left ventricle. This filling phase of the cardiac cycle is known as diastole.

When the diastolic cycle is complete, the left ventricle contracts abruptly, the mitral valve closes, and blood is ejected through the aortic valve. This phase of the cardiac cycle is known as ventricular systole. Left and right ventricular systole

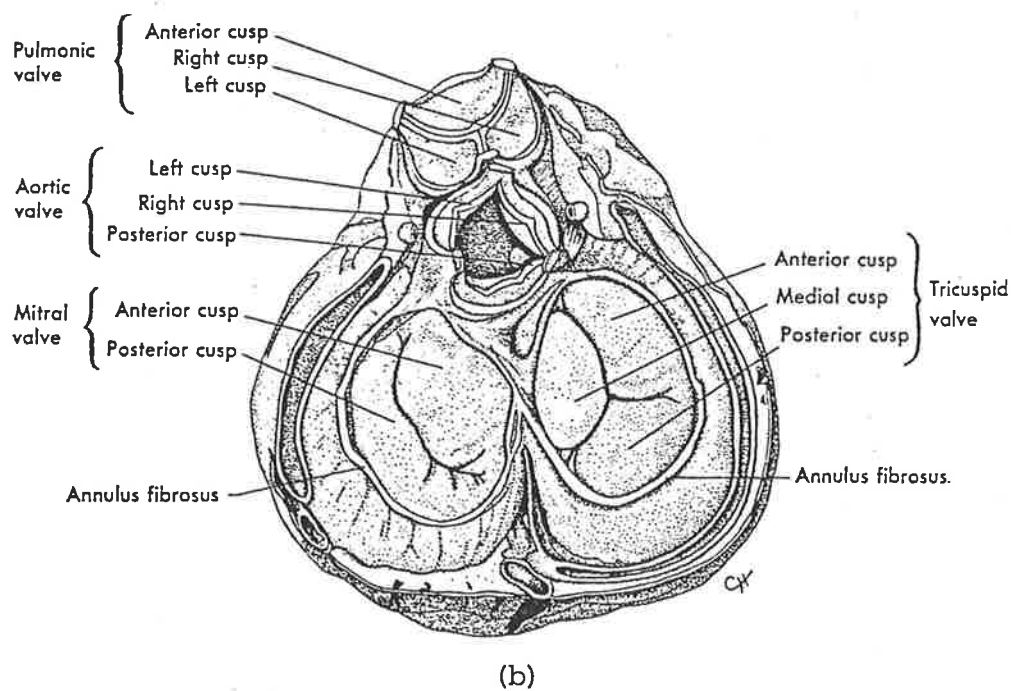
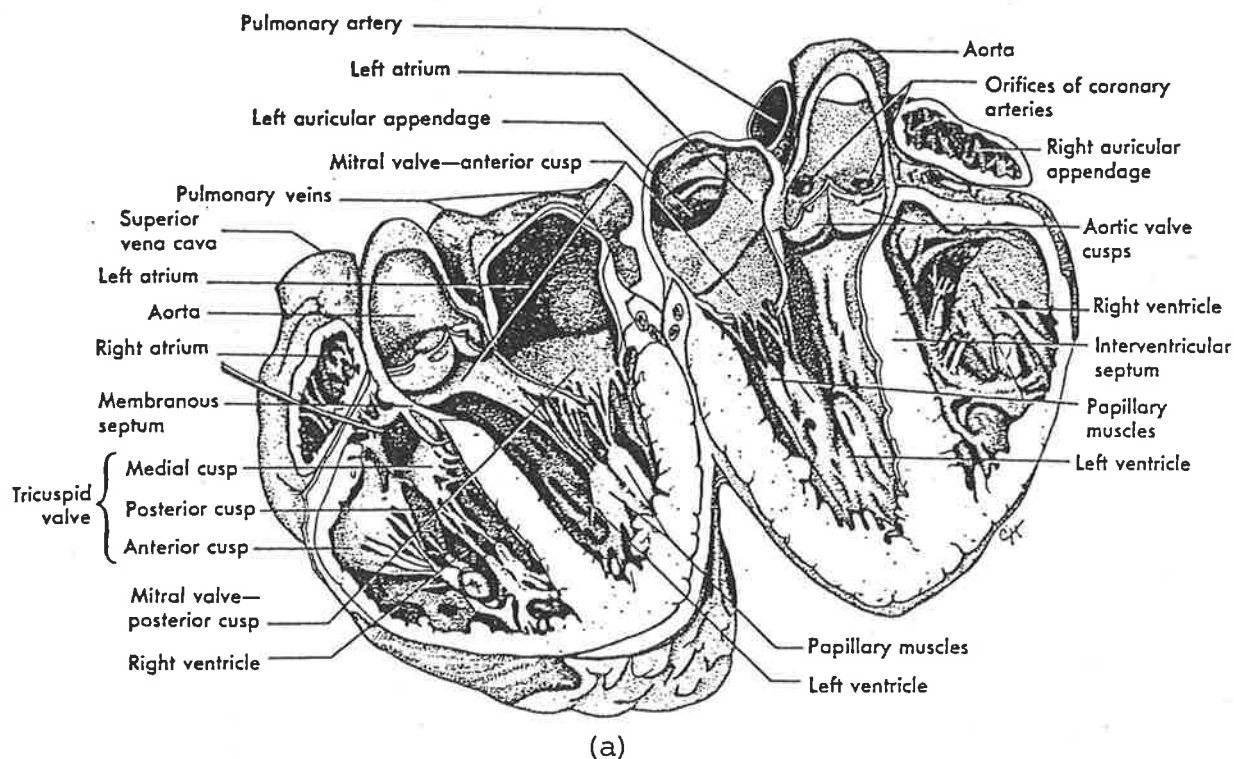


Figure 1.2.1

(a) Drawing of a heart split perpendicular to the interventricular septum to illustrate the anatomical relationships of the leaflets of the A-V and aortic valves.

(b) Drawing of the four cardiac valves as viewed from the base of the heart. Note the manner in which the leaflets overlap in the closed valves.

(Reproduced by permission from Berne and Levy, 1986)

occur simultaneously as do left and right diastole. The muscular contractions are controlled by a group of cells situated in the heart. The blood which is ejected through the aortic valve passes down the aorta into the arteries and then to the veins which reach most of the body. A schematic diagram of blood flow within the heart is shown in Figure 1.2.2.

The left and right coronary arteries supply oxygenated blood to the muscle of the heart known as the myocardium. These arteries originate from two of the three sinuses of Valsalva located at the base of the aorta. The flow of blood through these arteries is crucial for the proper functioning of the heart muscle.

The volume of blood passing down the aorta during each systole equals the left ventricular stroke volume, minus the amount which flows into the coronary arteries, minus the amount of retrograde flow through the aortic and mitral valves. Cardiac output in man varies between about $5 \text{ litres min}^{-1}$ at rest to about $25 \text{ litres min}^{-1}$ during extreme exertion. Normally retrograde flow is negligible except in diseased valves where it may be considerable. The stroke volume may then become several times greater than the flow down the aorta. This is clearly undesirable as the cardiac output of a healthy individual is normally sufficient to fulfill the oxygen requirements of the body.

A valve is called incompetent when it fails to close properly, causing a jet of blood to flow retrogradely through the valve when it should be shut. Both mitral and aortic incompetence are common valvular defects.

Another common valve defect is stenosis. This is a narrowing of the valve which restricts the flow of blood across the valve. Increased pressure drops are consequently induced across a stenosed valve. In mitral stenosis this pressure increase

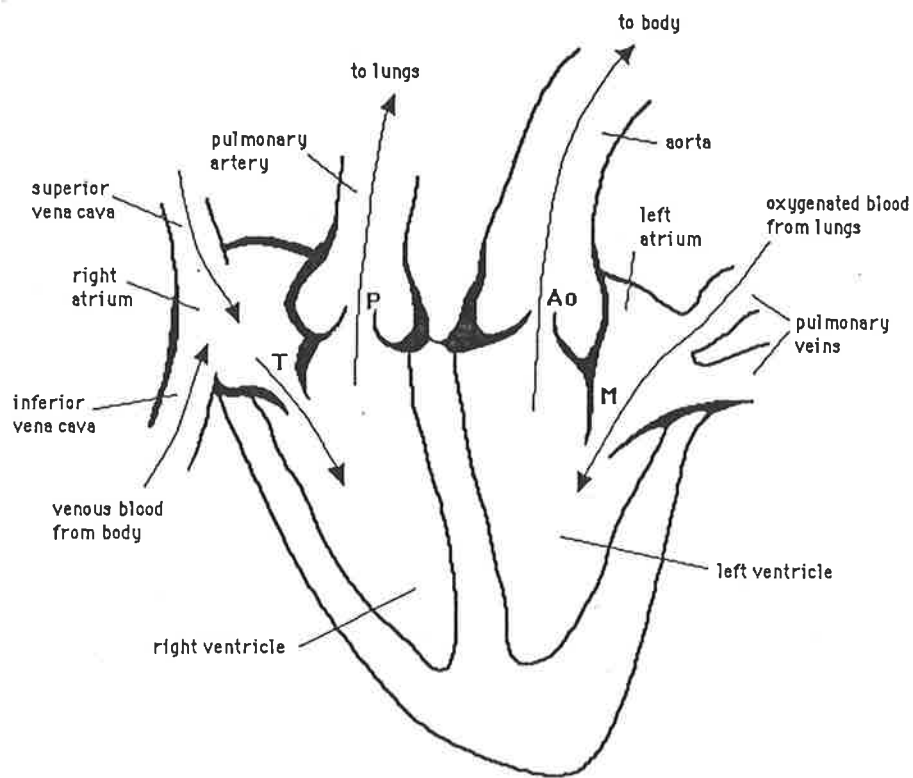


Figure 1.2.2

Schematic diagram of the blood flow through the heart. The arrows show the direction of blood flow. The valves are indicated by symbols: T, tricuspid; P, pulmonary; Ao, aortic; and M, mitral.

is transmitted back through the lungs to the pulmonary artery. The body may then try to compensate for these effects by reducing cardiac output making physical exertion difficult and uncomfortable. Irreversible damage to lung function and right ventricular failure have been known to occur as a result of prolonged severe mitral stenosis. Aortic stenosis also occurs causing a raised left ventricular systolic and a reduced aortic pressure. The effect of blood flowing through a stenosed aortic valve at high pressure is to cause turbulence in the aorta, aortic dilation and deposition of calcium on the aortic wall and on the valve leaflets. Tricuspid stenosis and incompetence often occurs in association with mitral valve stenosis and incompetence, respectively.

The human heart valves may become defective through acquired disease or because of congenital malformation. Congenital defects may initially appear minor, but after many years they can become quite serious. The most common acquired diseases which may cause valvular defects are rheumatic valvular heart disease and bacterial endocarditis. Even though the incidence of rheumatic heart disease is slowly decreasing, it remains present in over 1.5 million adults in the United States, and is responsible for about 15,000 deaths each year in subjects under the 65 years of age. The major damage to the heart from this disease results from gradual scarring of the heart valves which may take many years to become evident. A bacterial invasion of the blood stream by a simple wound could cause gross damage to the heart valve leaflets if circulating bacteria lodge on a small valve lesion. Both of these diseases can result in valvular incompetence, while stenosis can also result from rheumatic valve disease.

Papillary muscle dysfunction can occur due to blockage of coronary artery branches to the papillary muscles of the mitral valve, resulting in sudden mitral

incompetence. Even a myocardial infarct can sometimes injure the attachments of the valves to the wall.

It is clear that if incompetence or stenosis is present in heart valves, then serious consequences can ensue for the heart and circulation. The need for valvular replacement or surgical repair arises when these conditions threaten the life and well-being of the patient.

1.3 Heart Valve Replacement

The preservation of proper circulation is of paramount importance to patient well being, and this often necessitates the replacement of a dysfunctioning valve, or valves, by artificial substitutes. Nearly 75000 prosthetic heart valves are implanted annually throughout the world, with about 30000 in the U.S.A. alone.

Approximately 1400 prosthetic valves have been implanted by the Cardio-Thoracic Unit at the Royal Adelaide Hospital (R.A.H.) over the past 20 years. Annually, approximately 100 valves are now being inserted at the R.A.H. The type of valve most commonly used is the Starr-Edwards caged-ball prosthesis which the Cardio-Thoracic Unit has a 20 year experience. However, approximately 50 % of the valves being inserted in the aortic position are the Björk-Shiley tilting-disk type. The location of the valve implants are roughly divided between the aortic and mitral with about 10 % receiving valves in both positions.

1.4 Objectives of the Numerical Modelling

The governing steady state equations which describe viscous flow are coupled and highly nonlinear. It currently remains impossible to obtain analytic solutions to such complex equation systems. However, the pressing need for solutions to

similarly specified problems in many other fields of research and application has been met through numerical modelling. (Numerical modelling offers the ability to obtain quantitative solutions at a large number of discrete points in a region of interest and hence to observe the behaviour of complex mathematical models under varying conditions.) The advent of high speed digital computers and the subsequent development and improvement of numerical techniques for the solution of complex mathematical relationships has made this a practical reality.

The primary aim of the present work is to obtain detailed quantitative solutions from numerical models of flow through prosthetic heart valves at physiological flow rates. The numerical models are discrete and based upon mathematical models which describe the underlying physics of the flow. The numerical solutions will provide insight on how prosthetic heart valve fluid dynamics can be related to pathological problems.

1.5 Outline of Approach in the Present Study

To a cardiac surgeon unfamiliar with mathematics and computational methods the material contained within this thesis must appear quite daunting. Not wishing to discourage the very people who may benefit most from this research, a non-technical summary follows: The physical laws governing fluid flow can be described using complex mathematical equations. For real-life flow problems these equations may only be approximations to the physics simply because a mathematical formulation remains unknown. Furthermore, the mathematical equations can not be solved using the usual "pencil and paper" approach. Instead, the equations are recast into equivalent forms which enable interpretation and solution by a large-scale computer. Velocity and stress values are obtained at several thousand grid

points distributed throughout the flow field. For the case of flow through prosthetic heart valves, this data is used to construct detailed contour plots in the vicinity of the prosthesis. Thus, much insight can be gained into fluid dynamics of prostheses allowing identification of pathological problem areas in new or existing valves.

In Chapter 2, background information on prosthetic heart valves is presented. It covers history of valve surgery, pathological complications, desirable features and physical experiments.

Chapter 3 examines the way some basic fluid laws apply to flow through blood vessels and prosthetic heart valves. Several turbulent flow models are briefly discussed. Computational methods are also discussed and their application to determining prosthetic heart valve flows.

In the following chapters, three numerical models are constructed each introducing a subsequent improvement upon the previous. In Chapter 4, a model is investigated for steady laminar flow through the idealized planar geometry of a disk-type valve. Although very simple, this model clearly demonstrates the potential effectiveness and usefulness of more sophisticated numerical and mathematical models. The method of finite differences is used to solve the model.

Chapter 5 deals with an extension of the first model into the turbulent regime. It examines steady turbulent axisymmetric flow through a Kay-Shiley disk valve. The effects of turbulence are incorporated into this numerical model by employing the $k - \epsilon$ turbulence model embedded in the TEACH-T computer code.

The most sophisticated of the three models is constructed in Chapter 6. This model examines steady turbulent flow through a Starr-Edwards caged-ball valve in an aortic-shaped chamber. A boundary-fitted coordinate system is generated

for the flow domain in the near vicinity of the valve. The coordinate lines follow faithfully the surrounding physiological and prosthetic shapes. The $k - \epsilon$ turbulence model is formulated using the stream function–vorticity approach. The governing partial differential equations are recast into their curvilinear equivalents before being discretised into finite difference equations which are then solved iteratively.

In the concluding chapter, the clinical significance of this research work is summarised. Avenues for further study are also outlined.

CHAPTER 2

PROSTHETIC HEART VALVES

2.1 Introduction

This chapter examines many important aspects of prosthetic heart valves. It looks at the history of valve replacement, provides a general description of prosthetic valves, discusses the numerous pathological complications that may and very often do arise following a valve implant and presents a set of desirable characteristics that all valve designs should possess for reliable performance and patient safety. Finally, the chapter ends with a brief examination of the numerous *in vitro* measurements performed on cardiac prostheses and on the distinct limitations inherent in these physical techniques.

2.2 History of Valve Surgery and Replacement

Prior to the development of prosthetic valves the surgical correction of valvular defects carried a high mortality rate and any benefits obtained were usually only short-lived. With mitral stenosis surgical treatment was directed towards conversion of the stenosis into incompetence. The first successful treatment was performed by Souttar (1925) who used his fingers to dilate a stenotic mitral valve. Many years later the second successful treatment of this type was performed by Bailey (1949). By the early 1950s, closed valvulotomy, which consisted of blindly inserting the finger through the atrium or ventricle and relieving the stenotic valve in the beating heart, had become a safe and successful procedure. Dilatation of stenotic valves using fingers was almost entirely discontinued in 1959 with the introduction of

the transventricular dilator. This mechanical dilator was appreciated for providing much less general disturbance to the heart.

The surgical treatment of mitral incompetence proved more difficult. In 1957 a method for treating incompetence was developed whereby a circumferential suture was passed around the mitral annulus and drawing it in reduced the effective diameter of the mitral ring (Glover and Davila, 1957). This approach produced some benefit but was eventually replaced by more efficient open-heart surgery.

A new era in cardiac surgery emerged with the development of the heart-lung machine for the extra-corporeal circulation of the blood. It had become possible to operate on the completely arrested open heart while the heart-lung machine served to supply oxygenated blood to the rest of the body. As a direct consequence of this development, a great number of new possibilities became available in valvular repair. The pathway for total valve replacement had been opened and the design of suitable valve replacements constituted a major cardiac research project during the 1960s. The primary consideration of the valve designers involved in the initial development was prevention of any serious flow regurgitation in the closed valve. The pace was set by inventive surgeons driven by their commitment to assist their patients.

In 1953 the first prosthetic heart valve was implanted by Hufnagel without the use of a heart-lung machine (Hufnagel *et al*, 1954). The actual insertion time required only between 3 and 6 minutes. The valve was of the caged-ball type and was placed into the descending aorta of patients for the treatment of severe aortic incompetence. Locating the valve in a more physiologic position was simply too difficult. Unfortunately, the valve was unable to prevent regurgitation from the aortic arch vessels and was also prone to a high incidence of thromboembolic events.

In 1960, Harken performed the first subcoronary insertion of a caged-ball valve in a group of patients with aortic regurgitation (Harken *et al*, 1960). Although the caged-ball prosthesis was at first employed unsuccessfully in the aortic position it was successfully used for mitral valve replacement by Starr and Edwards (1961) in the same year. Following a great number of modifications, the caged-ball prosthesis continues to remain in use today. However, its centrally obstructive design gives it only adequate haemodynamics and is responsible for thrombogenic events and mild haemolysis. The most serious disadvantage of the caged-ball valve is its large spatial requirements which has lead to anatomic complications in some recipients.

The caged-disk valve was introduced in 1965 by several investigators in an attempt to improve haemodynamics, provide a lower profile and lighter weight than the caged-ball valve (Hufnagel and Conrad 1965; Kay *et al*, 1966). Unfortunately, the caged-disk design is the most flow obstructive of all prosthetic valves, particularly in the aortic position where it has been shown to increase pre-existing mitral insufficiency. Other problems include high thrombogenicity, haemolysis, intimal proliferation in the aortic root and disk cocking.

In an attempt to overcome these difficulties the tilting-disk prosthesis was introduced in 1967 by Wada (1967). Lillehei-Kaster and Björk-Shiley tilting-disk valves were introduced soon after (Kaster and Lillehei, 1967; Björk, 1969). Initial problems with disk wear caused by the hinge struts in the Björk-Shiley prosthesis were overcome by incorporating into the design the ability for the disk to turn freely once every 180 to 200 cycles. The potential for disk swelling was removed with the introduction of the pyrolytic carbon disk in 1972. A recent series of the tilting-disk valve use a convex-concave disk which allows greater forward flow by rising further out of its housing when open. The semi-central nature of flow through tilting-disk

valves and excellent haemodynamics have made this valve a popular choice with cardiac surgeons.

St. Jude Medical, Inc. introduced a bileaflet pivoting-disk prosthesis for clinical trial in 1977. The valve consists of two hinged semi-circular leaflets housed in an orifice ring which have an opening angle of 85° and a closing angle of 30° to 35° . The pivoting mechanism eliminates the need for the supporting struts designed into both the Björk-Shiley and Lillehei-Kaster tilting-disk valves. When open the St. Jude valve provides a superior high-flow orifice with nearly complete centralised flow. The St. Jude prosthesis moved from clinical investigation status to clinical use in early 1983 with a complication rate as low or lower than that of any other mechanical valve available.

Several cardiac surgeons have preferred to use biological substitutes for valve replacement. Homograft valves (removed from a cadaver), heterograft valves (removed from a dead animal), or tissue-fabricated valves (constructed from nonvalvular human and animal tissue) have been used extensively for the replacement of diseased aortic and mitral valves. Tissue-fabricated valves are designed to duplicate the flow behaviour of natural valves. Although short-term results are good, the long-term results for tissue bioprosthetic valves are not satisfactory due to calcification, leaflet degeneration and eventual valve stenosis. The main advantage of employing tissue valves over synthetic mechanical valves is their almost complete freedom from thrombus formation. Tissue valve replacements have been more successful in the aortic region than in the mitral area. However, the number of patients reporting long-term success remains small. To this date, no mechanical or biological valve replacement has been developed which is completely satisfactory.

2.3 Description of Prosthetic Heart Valves

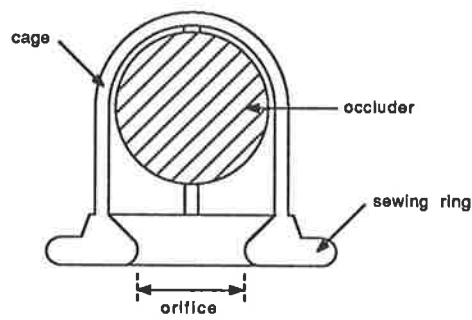
Nearly 50 different prosthetic heart valve designs have been developed since the early 1960s. The vast majority of these valves have been discarded and, of those remaining, several modifications have been made to improve haemodynamic performance, wear characteristics and overall valve function. Only a few types of valve have achieved widespread clinical acceptance. At present there are five major categories of prosthetic valves in use: (1) Caged-ball; (2) caged-disk; (3) tilting-disk; (4) bileaflet pivoting-disk; and (5) tissue bioprosthesis.

A typical mechanical prosthesis consists of four basic components: (1) Occluder; (2) cage, strut or hinge; (3) sewing ring; and (4) orifice ring. The occluder responds passively to pressure and flow changes within the heart to allow forward flow and to prevent substantial backflow. It is free to move under action of blood flow. The cage, strut or hinge retains and guides the motion of the occluder. The prosthesis is attached into the heart by sutures which pass through the sewing ring, into the tissues and back through the sewing ring. Both the cage (or struts) and sewing ring are attached to the orifice ring. Blood flows through the orifice when the valve is open and is sealed by the occluder when the valve closes. Diagrammatic examples of caged-ball, caged-disk, tilting-disk and bileaflet pivoting-disk prosthetic valves are presented in Figure 2.3.1.

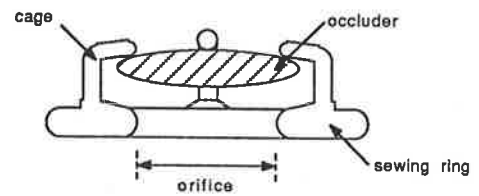
2.4 Pathological Complications

2.4.1 Introduction

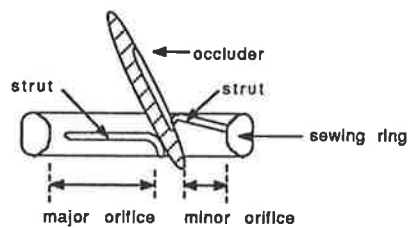
There are numerous prosthesis-related complications that can arise following a valve implant. Fatal and non-fatal problems which are unrelated to valve fluid



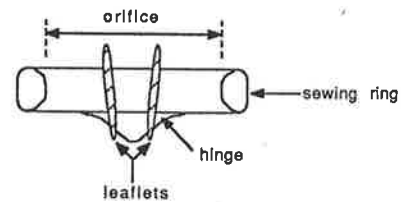
CAGED-BALL



CAGED-DISK



TILTING-DISK



BILEAFLET PIVOTING-DISK

Figure 2.3.1

Diagrammatic representation of major categories of mechanical prosthetic heart valves in use today. The valves are fully open and key components are identified.

dynamics include infection (prosthetic valve endocarditis), haemorrhage during anticoagulation therapy, mechanical malfunction due to material degradation, separation of prosthesis from its attachment site (valve dehiscence) and interference with cardiac function.

Schoen *et al* (1982) reported on complications experienced by a group of 99 patients with prosthetic valves. Of the late deaths (death occurring after postoperative day 30, 46 % overall) infection accounted for 14 %, valve dehiscence for 6 %, haemorrhage for 3 % and valve degeneration for 2 %. The most common causes of prosthesis-related death were directly related to valve fluid dynamics. These were thrombotic problems such as local thrombotic occlusion of the valve (13 %) and thromboembolism from the prosthesis (8 %). The anticoagulation therapy intended to prevent thrombotic complications also contributed to deaths through massive haemorrhage but is in itself unrelated to the valve fluid dynamics. Other non-fatal conditions related to valve fluid dynamics which may cause concern include haemolysis, sub-haemolytic injury, endothelial damage and tissue overgrowth. It is clear, therefore, that the fluid dynamics of prosthetic valves play a crucial role in the aetiology of several important prosthesis-related complications.

The following sections discuss prosthesis-related complications attributable to valve fluid dynamics.

2.4.2 Thrombosis and Thromboembolism

Thrombosis is defined as the formation of a blood coagulum within a vessel or the heart. There are several factors which favour thrombus formation. These include: (1) Stasis, or reduction in velocity of blood flow, which is common in varicose veins and inactivity; (2) derangement in composition of blood as may be

experienced through disease, surgery or childbirth; (3) recirculating, or separated flow caused by disruption to normal flow by the presence of a flow constriction as in a heart valve prosthesis; and (4) alterations in the endothelium which may be due to inflammation, vascular diseases or elevated wall shear stresses.

Wurzinger *et al* (1985a) discuss the complex thrombogenic role of elevated shear forces, recirculating flow zones and flow towards a vessel wall. According to these authors the pathway to coagulation can be summarized as follows: High shear forces act to damage blood components leading to the release of procoagulants. Recirculating flow zones amplify this stimulus and flow perpendicular to a surface mediates the manifestation of a thrombotic deposit on the surface.

Such flow conditions prevail at various locations in the vicinity of heart valve prostheses. During systole, a predilection site for thrombotic deposition occurs near the flow reattachment point of the sinus recirculation zone and along the downstream face of the sewing ring. The large recirculation zone downstream and attached to an occluder-type valve (e.g. caged-disk or caged-ball) is also a region of potential thrombus formation. The presence of a cage or strut further enhances the possibility of thrombogenesis. Varying amounts of thrombus formation have been observed on recovered Starr-Edwards aortic caged-ball valves with a predominance at the base of the cage struts and the apex of the cage (Yoganathan *et al*, 1981). A schematic example of thrombus formation and tissue overgrowth is shown in Figure 2.4.1.

The complications that can arise from thrombosis of a prosthetic valve are: (1) Thrombotic stenosis, when the prosthesis is partially blocked with a thrombus thus inhibiting the valve's performance; and (2) thromboembolic events, when part of the thrombus, known as an embolus, breaks off the main thrombus body and

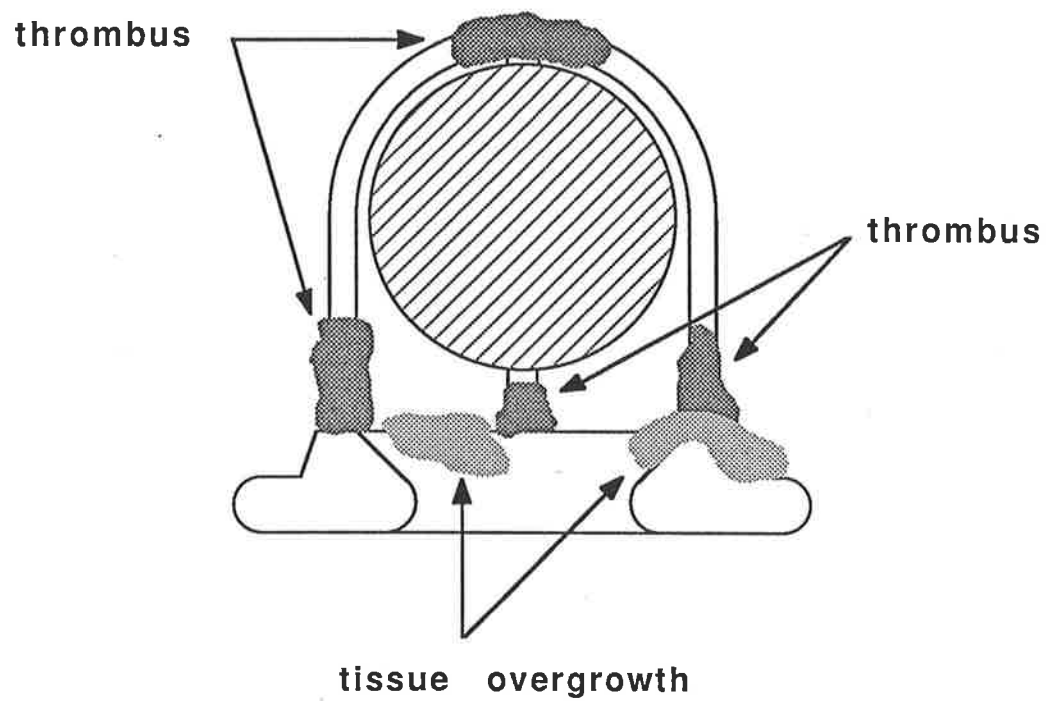


Figure 2.4.1

Diagrammatic example of thrombus formation and tissue overgrowth on a Starr-Edwards caged-ball valve. Redrawn from Yoganathan *et al* (1981).

is carried by the blood to lodge in the small arteries of a vital organ such as the heart, kidney or brain causing temporary or permanent damage. The incidence of thromboembolism from prosthetic valves represents a major threat to patients. Consequently, intense anticoagulation therapy is mandatory with any mechanical valve implant.

2.4.3 Haemolysis

The basic characteristics of shear flow apply not only to pure fluids but also to complex suspensions like blood. Locally accelerated blood flow occurs readily in the vicinity of prosthetic heart valves inducing abnormally high fluid forces, or shear stress. A blood cell which is subjected to a region of shearing action exerted by the surrounding fluid will experience a distribution of shear stress over its entire membrane. Consequently, the cell membrane will be stretched and may suffer irreversible changes detrimental to its essential function. Red blood cells are observed to assume cigar or dumbbell shapes before rupture, or lysis (Sutera, 1977).

Sub-haemolytic injury, or damage to blood cells, is common in prosthetic valve recipients but its effects can usually be compensated by the body's ability to substantially increase its rate of haemoglobin production. If the rate of destruction exceeds the rate of production clinically significant anaemia will be observed. The commonest cause of haemolytic anaemia is the development of a leak between the sewing ring and its attachment site. This leak is physically similar to jet-like flow and induces extremely high shearing forces on the blood. Surgical intervention is generally required to correct such leaks. The prosthesis itself continues to damage blood cells by its mere presence in the flow field, with significant anaemia usually absent. Nevertheless, much experimental and computational work has been un-

dertaken to assess the blood-damaging characteristics of prosthetic valves and the shear levels at which damage can be expected.

Shear Stress, $N m^{-2}$	Experimental Observations
1 – 10	damage to adhered red blood cells (Blackshear, 1972)
≈ 40	damage to endothelial cells (Fry, 1968)
10 – 50	damage to platelets (Hung <i>et al</i> , 1976; Hellums and Brown, 1977)
≈ 95	erosion of endothelial cells (Fry, 1968)
150 – 4000	damage to red blood cells in flow (Blackshear, 1972; Nevaril <i>et al</i> , 1969)

Table 2.4.1

Effects of shear stress on blood cells and endothelium

Estimates for shear induced in-bulk haemolysis thresholds have varied widely, from 150 to over 4000 $N m^{-2}$. Several reasons for these apparent discrepancies have been proposed including the extent to which containing surfaces are responsible for the observed levels of haemolysis. Leverett *et al* (1972) proposed that shear stress and exposure time are the two primary determinants for in-bulk haemolysis. Cells exposed for very short durations to very high stresses do not lyse, but exposure for long durations at much lower stress levels can cause lysis. However, below a certain threshold of fluid stress normal red blood cells will not deform and rupture even if exposed for an indefinite period. Two independent studies, one performed with turbulent shear flow (Sutera and Mehrjardi, 1975) and the other with laminar shear flow (Leverett *et al*, 1972) have shown this low stress threshold is between 150 and 250 $N m^{-2}$. It can also be inferred that the presence of the fluctuating characteristics of turbulent flow are not harmful provided the time-averaged shear stress does not exceed the threshold.

Similar studies have revealed that platelets have a substantially lower tolerance to shear stress than red blood cells during sustained exposure, and that platelet lysis occurs at stresses between 10 and 50 N m^{-2} (Hung *et al*, 1976; Hellums and Brown, 1977). However, it has recently been proposed that high shear forces acting for milliseconds do not induce a significant active release of platelet granule contents directly, but by damaging them, render secondary biochemical activation a matter of course (Wurzinger *et al*, 1985b). A summary of experimental measurements is presented in Table 2.4.1.

2.4.4 Endothelial Damage

The endothelium is a delicate membrane which lines the heart, blood vessels and body cavities. In large arteries such as the aorta, rapidly flowing blood exerts a force, or viscous drag, upon the endothelial lining of the artery which attempts to pull the lining along with the flow. This force, or shear stress, is directly proportional to the velocity gradient near the vessel wall. Normally the levels of wall shear stress are so small that no damage or erosion of endothelial cells occurs. However, during systole, the peripheral flow induced by caged-disk and caged-ball valves, and the semi-central flow through tilting-disk prostheses causes a significant increase in velocity gradients at the proximal portions of the aorta. Experimental measurements to determine the wall shear levels at which damage and erosion of endothelial cells occurs have been undertaken (Fry, 1968) and the results are given in Table 2.4.1. As has already been mentioned, damage to the endothelium also acts to increase the potential for thrombus formation by increasing the "stickiness" of the vessel wall. It is therefore clear that a highly desirable feature of any valve prosthesis should be low levels of wall shear stress.

2.4.5 Tissue Overgrowth

Tissue overgrowth is often seen on valve prostheses recovered during autopsy. Yoganathan *et al* (1981) observed tissue overgrowth on the aortic side of the sewing ring and on the struts of cloth-covered Starr-Edwards caged-ball valves (see Figure 2.4.1). The cloth covering (e.g. polypropylene mesh) on cage struts and valve orifice has reduced the incidence of thromboembolism by allowing infiltration of fibrous tissue which eventually serves as a nonthrombogenic surface. However, excessive tissue growth into the cloth covering will eventually result in valve stenosis and an increase in the pressure drop across the valve, thus diminishing hydraulic performance. The development of less thrombogenic materials (e.g. pyrolytic carbon) for use in valve construction has lead to some progress in the control of tissue overgrowth. Nevertheless, tissue overgrowth is not limited to cloth covered valves where it has been primarily noticed, but appears to depend to some extent on the fluid dynamics of the valve, although this process is not fully understood. High blood flow and the associated effects of fluid scouring may play a role in limiting or preventing tissue overgrowth.

2.5 Characteristics of a Good Replacement Valve

Roberts (1976) published a list of six ideal characteristics that a good prosthetic cardiac valve should possess. These are: (1) It has good haemodynamic characteristics, that is, it is nonobstructive or nearly so, and completely competent, or nearly so; (2) it is nonthrombogenic; (3) it does not degenerate, wear out or vary; (4) it does not significantly alter blood components; (5) it can be inserted with undue difficulty (i.e. exquisite technical skill is not required for its insertion); and (6) it does not disturb the patient through excessive noise. Furthermore, Roberts (1976)

indicated that there was little difference between the four types of prostheses then examined in terms of the ease of surgical implantation, and that none of the valves produced sufficient noise so as to be unduly disturbing. McClung *et al* (1983) added two more desirable valve characteristics to the list above: (7) a low incidence and diminished severity of late prosthetic endocarditis; and (8) anatomic suitability of the valve to implanted location.

Unfortunately, no current prosthesis fully satisfies this set of demanding requirements. *In vitro* testing methods can determine whether several of the requirements are fulfilled, but regrettably, some of the more important characteristics such as the thrombogenicity and the extent of blood trauma a particular valve design may produce have to be established by clinical trials, often after extensive animal tests. The valve characteristics that can be easily assessed through experimental measurements and observations include haemodynamic features, resistance to wear and noise.

Nevertheless, the urgent need to design better valves and to determine problem areas in existing valves has seen many investigators conduct experimental measurements using blood analog fluids and other testing equipment. The following section presents a brief summary of the procedures and results reported by several investigators in this field.

2.6 Experimental Measurements and Observations

The *in vitro* measurement of flow through prosthetic heart valves has been conducted by several researchers. Yoganathan and his co-workers (1979) used laser-Doppler anemometry to obtain measurements of velocity profiles in the vicinity of aortic prostheses in steady flow conditions. By assuming that the flow pattern

between the occluder and the channel walls was jet-like, they obtained expressions for the maximum turbulent shear stresses in this region. Similar expressions were also presented for stresses in the wakes behind spheres and disks. At high flow rates they estimated turbulent shear stresses in the range 10 to 100 N m^{-2} and wall stresses of about 100 N m^{-2} .

Figliola and Mueller (1981) also investigated steady flow through several types of prosthetic valve using a laser-Doppler anemometry system for velocity measurements and a miniature X array fibre film probe for Reynolds stress determination. Their results indicated that the region between the occluder and aortic wall was potentially the most damaging to blood cells and to the endothelial lining of the aortic wall for all the valves tested. Turbulent shear stresses of the order of 10 to 100 N m^{-2} were measured. However, measurements with the probe were not possible in the annular flow region between the occluder and the aortic wall and in the reverse flow regions.

Herkes and Lloyd (1981) utilised an electrochemical technique for a direct investigation of flow-induced wall shear stress in the vicinity of caged-disk and caged-ball prosthetic heart valves. Their measurements were restricted to the aortic wall region where the maximum wall shear stresses found were 48.3 N m^{-2} and 17.4 N m^{-2} for the disk and ball valves, respectively.

Chandran *et al* (1983) conducted an *in vitro* experimental study on several aortic prostheses under pulsatile flow conditions using hot-film velocimetry. Using a three-sensor probe the mean velocity profiles and the turbulent normal stresses were indirectly computed. Comparison of measurements with steady flow at comparable flow rates revealed that the turbulent shear stresses under pulsatile conditions are larger by a factor of two.

Woo and Yoganathan (1985) used a three beam laser Doppler anemometry system to study the velocity and turbulent shear stress fields created by several aortic prostheses under pulsatile flow conditions. All valves studied created very disturbed flow fields, regions of flow separation and/or stagnation, and regions of elevated turbulent shear stress. During peak systole (instantaneous flow rate of $32 \text{ litres min}^{-1}$), peak stresses up to 185 N m^{-2} were obtained downstream of a Starr-Edwards caged-ball valve. The mean shear stresses were up to 110 N m^{-2} at the same location. In the annular flow region poor signal-to-noise ratio meant that no turbulent shear stress measurements were possible. Based on the *rms* axial velocity measurements, turbulent shear stresses of the order of 300 to 500 N m^{-2} were estimated to have occurred in this region. Pulsatile effects, however, are not examined in this thesis.

Despite the vast amount of valuable information that such experimental investigations yield, they are unable to provide a complete and detailed description of the flow characteristics for a particular valve design. Typically, measurements are taken through a series of cross sections of the flow, along a section of the channel wall or with a probe. Several important flow regions such as the area between an occluder and the wall and the sinus zone are simply inaccessible, and it is impossible, for example, to use a probe to measure fluid flow without disturbing the flow one is trying to measure. Furthermore, experimental measurements generally provide little direct information on important flow-related phenomena such as valve thrombogenesis and blood trauma.

In order to improve our knowledge on the potential for valve thrombogenesis and blood trauma a more quantitative approach becomes necessary. In this regard computational fluid dynamics techniques provide a powerful tool. I firmly believe

that these numerical methods will play an increasingly important role in the development of new prosthetic heart valves and in the detection and eventual elimination, or control, of existing flow-related pathological complications. The next chapter discusses the application of numerical methods for the solution of mathematical models.

CHAPTER 3

MATHEMATICAL MODELLING AND NUMERICAL METHODS

3.1 Introduction

Modelling pulsatile blood flow through the cardiovascular system in precise mathematical terms remains an insurmountable problem. The human heart is affected by many physical and chemical factors. Blood vessels are elastic channels with continuously varying dimensions that can have multiple branches. The blood flowing in these vessels is a complex suspension composed of red and white corpuscles, platelets and lipid globules all suspended in a colloidal solution of proteins. In the case of prosthetic heart valves, the situation is further complicated by the presence of an unnatural device in the flow path. In spite of the difficulty created by the multiplicity of interrelated elements, extensive insight into cardiovascular flows can still be obtained by applying numerical solution techniques to simpler physical systems.

The literature contains many examples of mathematical models for a variety of systems (physical, biological, chemical, etc...) with analytic or numerical methods of solution available. Consequently, this chapter is not concerned in detailing the many complex equations which make up fluid mechanics or the finite difference method, but instead restricts the discussion to a relatively qualitative level. The more important aspects are presented as they are needed in the following chapters. This chapter describes some basic fluid laws relating to flow through blood vessels and prosthetic heart valves. Mathematical models of turbulence are discussed. Computational methods are covered for solving nonlinear mathematical flow equations

and generating boundary-fitted coordinate systems. Previous numerical studies of flow through prosthetic heart valves are also discussed.

3.2 Laminar and Turbulent Flow

There are two basic modes of viscous fluid motion — laminar and turbulent flow. If we consider axisymmetric flow in a pipe, then in laminar flow particles follow straight lines parallel to the pipe axis, but in turbulent flow the particles follow a random path through the pipe, and only the average motion is along the pipe axis. Laminar and turbulent flows are essentially states of stability and instability, respectively, and as such, no complete definition of turbulence seems possible.

The Reynolds number Re for flow in a pipe is usually defined as

$$Re = \frac{UD}{\nu} \quad (3.2.1)$$

where U is the velocity of flow at the centre, D is the vessel diameter and ν the kinematic viscosity. The kinematic viscosity is defined as

$$\nu = \frac{\mu}{\rho} \quad (3.2.2)$$

where μ is the molecular, or laminar, viscosity and ρ is the fluid density. Also, the viscosity is defined as the ratio of shear stress σ to the rate of strain du/dy . That is

$$\mu = \frac{\sigma}{\frac{du}{dy}} \quad (3.2.3)$$

Experimental observations indicate that above a critical Reynolds number, transition will occur from laminar to turbulent flow. Thus, laminar flow can become

unstable. Below a certain Reynolds number, small disturbances introduced to the flow are always damped out and the flow remains stable. The random fluctuating motion characteristic of turbulent flow arises from small disturbances which are not damped out, but instead increase with time and eventually interact with each other in a chaotic manner. As a result, differences between laminar and turbulent flow become evident. Firstly, the velocity profiles appear very different (Figure 3.2.1). The fully developed laminar profile is parabolic and the turbulent profile is much flatter near the pipe centre with larger gradients near the vessel walls. Secondly, the “roughness” of turbulent flow acts to increase the effective viscosity of the fluid. In the laminar case, the viscosity is entirely due to molecular effects. However, for turbulent flow the apparent viscosity comes from two sources: (1) molecular viscosity; and (2) convection of turbulent “eddies” which usually form the larger part of the effective viscosity. This additional term is often referred to as the eddy viscosity.

In the human circulatory system, the flow of blood is normally laminar. However, abrupt flow variations due to irregularities and stenoses, as are often encountered with prosthetic heart valves, will produce turbulence. Experimental research indicates that turbulent flow can occur during the period of ejection in the ascending aorta of subjects with elevated cardiac output, and it occurs consistently in the ascending aorta of subjects with abnormal aortic valves or with prosthetic aortic valves (Stein and Sabbah, 1976). Furthermore, the shear stress from equation (3.2.3) is given by

$$\sigma = \mu \frac{du}{dy} \quad (3.2.4)$$

It is apparent from the discussion above and equation (3.2.4) that turbulence will cause a substantial increase in the level of shear stress since both the effective viscos-

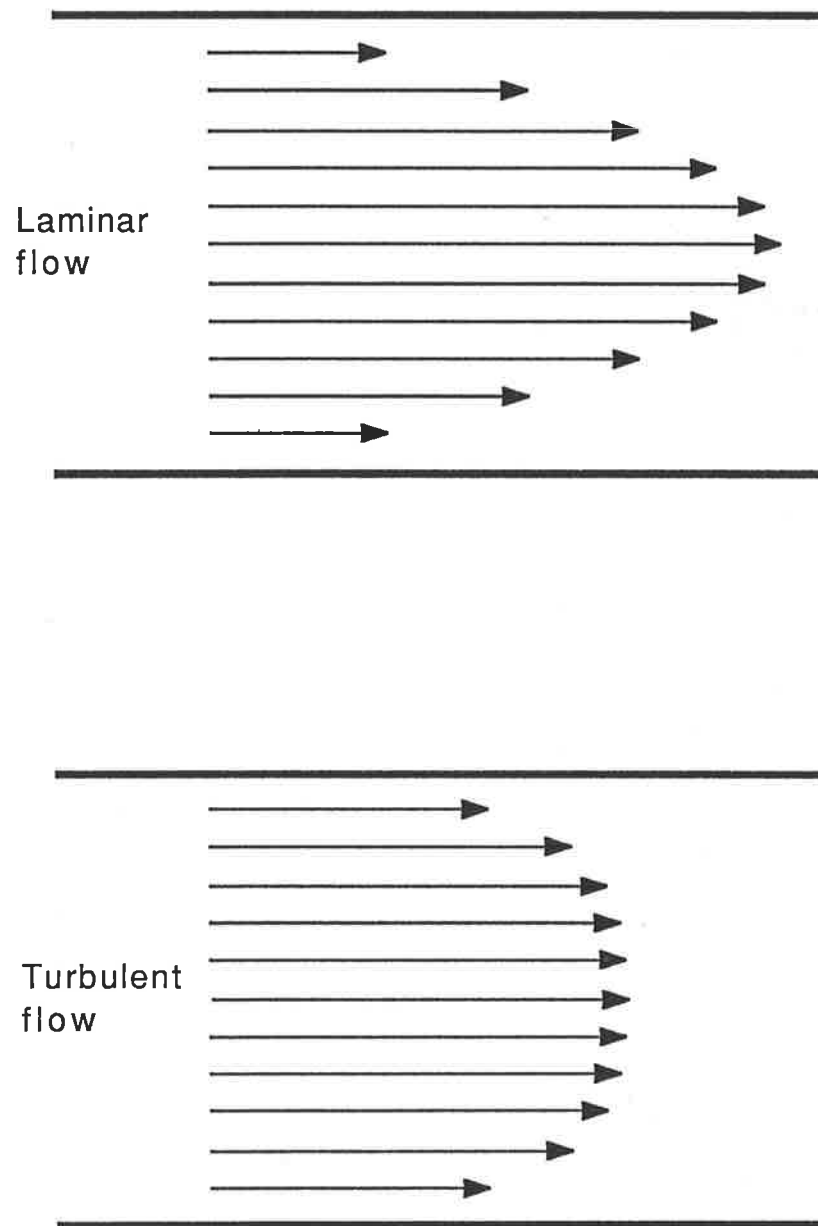


Figure 3.2.1

Diagrammatic representation of fully developed laminar and mean turbulent flow vectors in a pipe at the same flow rate.

ity and the shear strain rate will become greater, particularly near a vessel wall. The pathological consequences of elevated shear forces were discussed in Chapter 2 and are clearly undesirable. In complex systems such as turbulent flow through valve prostheses, equation (3.2.4) provides only limited qualitative descriptions. Given valve geometry, fluid properties and details of the inflow conditions one would prefer to evaluate in totality the velocity and shear stress fields for the entire flow domain.

The connection between the observed complexity of fluid flow and the fundamental physical laws governing such behaviour is usually obscure. Indeed, with what little we know, both the basic laws and experimental observation are required to make the connection. Unfortunately, a mathematical description of fluid flow based on physical laws has not yet developed sufficiently so as to cater for every possible flow situation. Even in cases where the mathematics is known, the formulation is often posed in highly nonlinear terms, thus excluding analytic solution. Similarly, the implementation of experimental measurements is time-consuming and expensive, and the accuracy of observations, as limited as they may be, is often questioned.

Nevertheless, the need to address real-life problems is pressing, and mathematical models of fluid flow, both laminar and turbulent, have been developed and applied successfully to many problems. These models have governing partial differential equations which are based on the well known conservation principles of mass, momentum and energy. The mathematical formulation is usually nonlinear and analytic application to realistic problems is often very difficult or impossible unless simplifications are made. By exploiting the capabilities of modern high-speed computers and numerical techniques, solutions to otherwise unsolvable problems are being found.

The following two sections provide a discussion of turbulence models and the numerical methods that have been applied to fluid flow problems.

3.3 Turbulence Models

Turbulence modelling of complex engineering and physical flows has been a topic of active research for many years. Several mathematical models have been developed, and these are usually classified as either Reynolds stress models or eddy viscosity models. Reynolds stress models are based on the evolutionary equations for the turbulent stresses. They were developed to overcome deficiencies in eddy viscosity models. Although successful in computing free shear flows, they have encountered difficulties in treating wall-bounded separated flows. In addition, their complex structure results in increased computer time and decreased stability. It is questionable whether the benefits outweigh the costs. As a result, Reynolds stress models have been applied to relatively few turbulent flow problems.

Eddy viscosity models, however, are much more popular. These models are based on the Kolmogorov-Prandtl proposal that the eddy viscosity is proportional to the product of a turbulent velocity scale and a turbulent length scale. That is

$$\nu_t \propto q L \quad (3.3.1)$$

where ν_t is the eddy viscosity, q and L are velocity and length scales of the turbulence, respectively. Depending upon the manner in which the eddy viscosity is specified these models can be further grouped into either algebraic or differential models. With algebraic models ν_t is determined by replacing q and L with simple algebraic expressions involving the mean velocity field. Mixing-length models, as they are also known, are excellent for investigating single-zone flows but become

deficient when switching between one type of zone to another within a single flow. This is particularly noticeable when investigating separated flows.

Differential models specify the eddy viscosity in the solution of one or two differential equations. One-equation models prescribe the turbulent length scale but use a partial differential equation to determine the turbulent kinetic energy. The kinetic energy serves to characterise the velocity scale. This approach results in only a marginal improvement over the mixing-length models.

Two-equation models employ the Boussinesq eddy-viscosity concept

$$\overline{u'_i u'_j} = -\nu_t \left(\frac{\partial U_i}{\partial x_j} + \frac{\partial U_j}{\partial x_i} \right) + \frac{2}{3} k \delta_{ij} \quad (3.3.2)$$

where δ_{ij} is the Kronecker delta, and U_i and U_j are the mean components of velocity in the i and j directions, respectively. Differential equations are used to involve both the velocity and length scales. One of the most studied two-equation turbulence models is the $k - \epsilon$ model. This model remains the most widely used approach for the solution of practical turbulent flow problems. In this model, the eddy viscosity is obtained by solving partial differential transport equations for the turbulent kinetic energy k and the energy dissipation rate ϵ . These transport equations are relatively easy to incorporate into existing Navier-Stokes computer codes. The complete equations are presented in the subsequent chapters.

The $k - \epsilon$ model has been successfully applied to a great number of shear flow problems. However, for more complicated flows the model tends to lose its accuracy. The standard $k - \epsilon$ model is based on the assumption that the eddy viscosity is identical for all Reynolds stresses. In two-dimensional flows without swirl this condition has proved adequate for the calculation of thin shear layers.

However, in certain three-dimensional flow situations (e.g. square duct flows) and in planar separated flows the isotropic eddy viscosity assumption proves insufficient by underpredicting velocity fluctuations, and thus giving rise to inaccurate predictions for the Reynolds stresses. This point has been demonstrated by Speziale (1986). The normal Reynolds stresses play an important role in the calculation of the mean velocity field in separated turbulent flows. For example, it is well known that the $k - \epsilon$ model underpredicts the recirculation length downstream of a backward-facing step. Another consequence is the inability to predict secondary flows in non-circular ducts. A refinement which uses an algebraic stress model can be introduced to replace both the Kolmogorov-Prandtl expression (3.3.1) and the Boussinesq eddy-viscosity relation (3.3.2) above (Rodi, 1980).

A limitation on the $k - \epsilon$ turbulence model is the restriction to high Reynolds number flows (i.e. $Re > 20000$). Consequently, it is not directly applicable to the viscous sublayer near boundary walls and to low Reynolds number flows. However, the common approach of using wall functions allows efficient treatment of the near wall regions. The wall-function method is discussed by many authors including Rodi (1980), and is mentioned in the following chapters. In the case of low Reynolds number flows the standard $k - \epsilon$ model and wall-function method are insufficient and alternative approaches have been developed. The most popular of these is the low-Reynolds number turbulence model of Jones and Launder (1973). This model seeks to provide turbulence equations which are valid throughout the laminar, semi-laminar and fully turbulent regions. The costs associated with this improvement are the requirement for many additional computations in the near wall region, increased equation complexity and related numerical instability.

Nevertheless, the $k - \epsilon$ turbulence model and wall-function method is the sim-

plest approach being widely applied to practical turbulent flow problems. Once implemented, it is the “stepping-stone” by which the model refinements mentioned above may eventually be introduced.

3.4 Computational Fluid Dynamics

The development of computational methods to solve nonlinear partial differential equations has been progressing for over 60 years. The advent of high speed digital computers has seen many advances in this field by enabling development, comparison and refinement of the numerical techniques. An historical outline of computational fluid dynamics is available in Roache (1972). Publications such as *Journal of Computational Physics*, *Computers and Fluids*, *Journal of Fluid Mechanics* and *Numerical Methods in Engineering* contain articles describing application of numerical methods to numerous physical problems including physiological flow processes.

One particular method, the finite difference method, remains the predominant technique for obtaining numerical solutions to fluid flow problems. The method can be summarised by the following stages (Noye, 1976):

1. A grid is developed to cover the solution region. At the grid points (i.e. intersection of grid lines) the approximate solution to the problem will be found.
2. All dependent variables are initialised. The initial values are either known exactly, or they may at best be only a rough guess, especially if they require determination through application of the solution procedure.
3. The finite difference equations corresponding to the continuous partial differential equations are constructed. They are used to calculate approximate values

of the dependent variables at all grid points in the solution domain.

4. The computational cycle commences. New values are calculated at adjoining space positions by incrementing some value Δx , and at subsequent time levels by Δt . The process is continued until a predetermined time is reached, or as in the steady state case, the dependent variables at each grid point remain unchanged at successive time levels.

Unfortunately, it is generally impossible to obtain theoretical estimates of the accuracy, convergence and stability of a numerical solution to a problem possessing large nonlinear terms in the governing equations (Noye, 1976). This is especially true for viscous fluid flow problems with large Reynolds numbers. In order to verify the accuracy of finite difference techniques applied to such problems, it is necessary to compare numerical solutions to experimental results and/or employ computational experimentation such as observing solution behaviour when subject to grid refinement. The mathematical theory for convergence and stability of numerical schemes have only been developed for linear systems. Consequently, results from linear theory are used to guide nonlinear problems but many *ad hoc* approaches remain a practical necessity. For example, with fluid flow problems, it is often necessary to anticipate the nature of the solution in advance so that a larger number of grid points can be assigned to regions of interest, particularly those with high velocity gradients. Typically, these regions occur at constrictions and near wall boundaries. Also, specification of iterative relaxation parameters normally requires some testing beforehand to determine optimal values. It may be found that switching to slightly different values after a certain number of global iterations serves to enhance convergence and control stability. Alternating the direction of iterative sweeps also seems to provide additional solution control. Monitoring so-

lution progress is mandatory and its implementation depends upon the nature of the problem, the availability of experimental results for comparison and the degree of interactiveness of the computing environment. In this regard, computer graphics are very useful.

A particularly good introduction to finite difference techniques with emphasis on heat and mass transfer is presented by Noye (1976). In the article he discusses derivative representation of continuous functions at discrete grid points using Taylor's series expansions to approximate forward, backward and central finite differences. He goes on to demonstrate the concepts of consistency, convergence and stability using a mathematical model possessing an analytic solution. The application of both implicit and explicit numerical methods to solve the multi-dimensional transport equation is covered, as are different grid and coordinate systems. Roache (1972) provides greater detail in respect to viscous flow applications involving treatment of nonlinear terms.

3.5 Boundary-Fitted Coordinate Systems

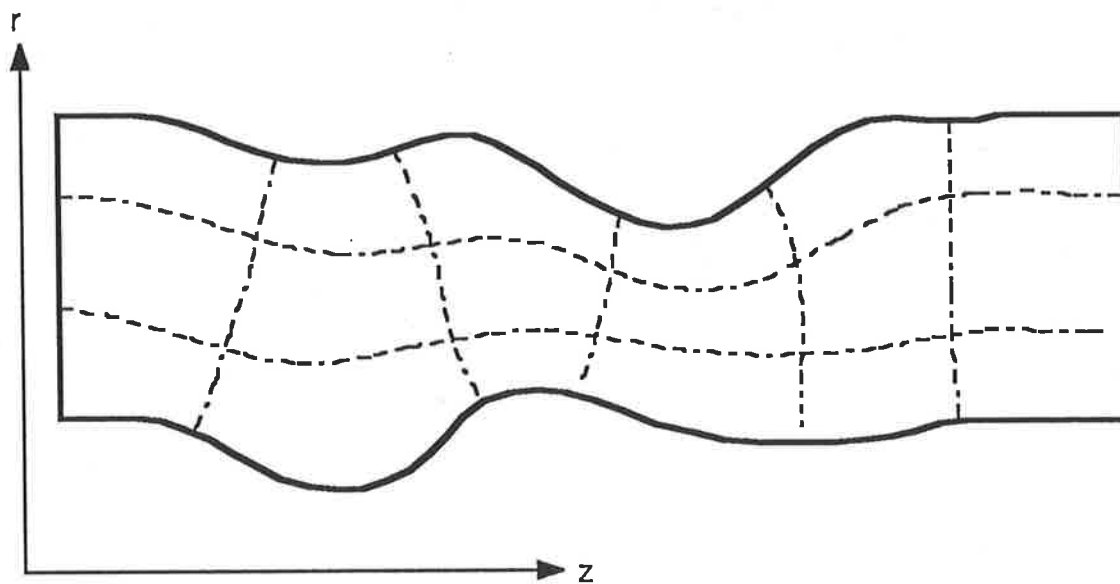
The vast majority of problems associated with computational fluid dynamics are solved using finite difference methods and a regular Cartesian coordinate system. Consequently, flow domain geometry is often simplified by making boundary walls coincide with the rectangular computational mesh. In a sense, the physical domain is fitted to the computing mesh by adjusting the location of grid lines until the flow domain is satisfactorily defined. Clearly, this approach is not suited for application to more general problems, particularly those where curved physical boundaries strongly influence the flow characteristics. Domains with greater geometrical complexity are often solved with the more formidable finite element

method and demand great programming effort when compared to the finite difference approach. A technique that embodies the computational efficiency of finite differences and facilitates application to general body problems is the numerical generation of boundary-fitted coordinates.

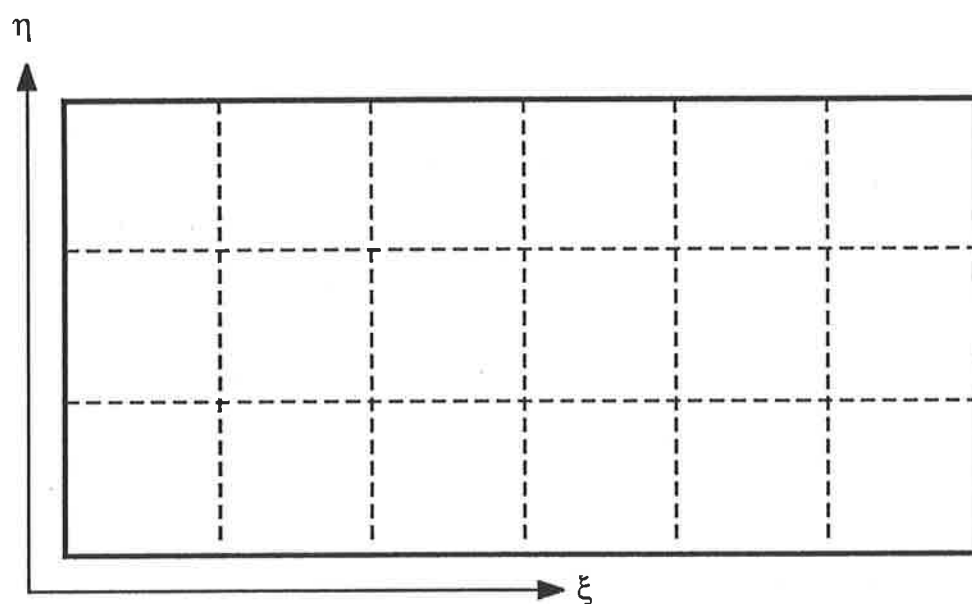
In a boundary-fitted coordinate system, the physical domain (z, r) is numerically transformed, or mapped, onto a calculation domain in coordinates (ξ, η) — a regular rectangular coordinate system (Figure 3.5.1). All numerical calculations involving the governing equations are conveniently performed in the transformed (ξ, η) plane rather than the physical (z, r) plane. Complicated geometries exhibiting much complex structure may best be handled using a composite grid.

The boundary-fitted system approach also offers a substantial advantage over the usual treatment of curved boundaries with finite differences. For example, a rectangular mesh may have been overlayed on any general shape and either a “saw tooth” approximation or interpolation could be employed for the curved regions. It is reasonable to expect that the boundary-fitted coordinate system would provide a better boundary representation in such areas. Other advantages of the boundary-fitted approach include: (1) Simplification of boundary condition specification; (2) accurate implementation of boundary conditions; (3) efficient use of grid points for the calculation area; and (4) flexibility in the type of curved-wall problems that can be addressed.

Once the physical domain is specified, a suitable boundary-fitted computing mesh can be generated. Unfortunately, this is seldom an easy task. The reason for this arises partly from the difficulty in providing a sufficiently good initial estimate for the grid and in the choice of an appropriate numerical procedure. Thompson and Warsi (1982) provide a comprehensive review of numerical methods for generating



Physical Plane



Transformed Plane

Figure 3.5.1

Example of a non-rectangular geometry in the physical plane with schematic grid lines and its corresponding representation in the transformed plane.

curvilinear coordinate systems having coordinate lines coincident with boundary segments. In Chapter 6, one of these methods is applied to generate an orthogonal curvilinear computing mesh for a Starr-Edwards caged-ball prosthesis.

3.6 Numerical Studies of Flow Through Prosthetic Heart Valves

As explained in Chapter 2, the *in vitro* measurement of flow through heart valve prostheses produces useful but limited information, and in some instances, such as the potential for valve thrombosis, very little can be deduced from a finite number of measuring points. As a result, extensive clinical trials are required to establish whether prosthetic valves have acceptably low levels of blood trauma and thrombosis. Computational fluid dynamic techniques, however, provide a powerful tool for determining valve haemodynamic performance as they can yield detailed quantitative data for velocity and stress distributions in the flow field. Thus, conclusions regarding the potential for a valve to induce blood trauma or give rise to thrombotic deposition can be drawn via computational methods. Additional benefits include reliability, efficiency, immediacy and ready adaption to various geometric and flow configurations. Nevertheless, computational techniques and experimental measurements will continue to complement each other as they currently do in many aspects of modelling in other physical sciences.

Several researchers have employed numerical methods to determine blood flow patterns in the vicinity of prosthetic heart valves. Au and Greenfield (1975) applied numerical techniques and principles of fluid dynamics to study stress distribution in blood caused by the axial motion of an occluder in a caged-disk prosthesis. They assumed planar geometry, laminar Newtonian flow and a comparatively low Reynolds number of 50 (based upon distance between the occluder and cage strut, and the

occluder velocity). Time-dependent finite difference equations were formulated and an interactive graphics environment provided continuous monitoring of numerical procedure stability and convergence rates. Changes in maximum stress values as the occluder moved from the fully closed to the almost fully open position were reported. However, the emphasis was on the application of interactive computer graphics to study this and other medical phenomena.

In a subsequent study, Au and Greenfield (1977) again utilised finite difference methods to examine steady laminar flow through a planar model of the Björk-Shiley tilting-disk prosthesis. The accent was again on the totality of the graphics displays which included contour maps and surface plots of computer-formed streamline, velocity, stress and pressure fields. Potential sites for haemodynamically induced thrombogenesis were observed. Importantly, the difficulty associated with computing on irregular boundary segments was noted and it was suggested that the finite element approach could overcome this problem.

Underwood and Mueller (1977) obtained numerical solutions for steady axisymmetric laminar flow through a disk-type valve in a constant diameter chamber for Reynolds numbers ranging from 20 to 1300. The finite difference method was used to compute flow variables and the solution procedure included calculation of a suitable time-step at each interval. Experimentation with both first-order and second-order accurate upwind differences for the advection terms revealed variation in the numerical results — the second method providing better solutions when compared with physical measurements. Regions of separated flow attached to the disk occluder, downstream of the sewing ring and along the aortic wall were identified. Zones of high fluid stress were also noted. The possibility of thrombosis and haemolysis was discussed.

In an extension of the above study, Underwood and Mueller (1979) repeated the calculations, but instead of assuming a constant diameter chamber a more realistic aortic-shaped vessel was considered. A non-uniform grid system was used for both the axial and radial directions enabling close approximation of the aortic wall shape. Differences in solutions between the two chambers were reported. Reynolds numbers examined were between 50 and 600. Regions of separated flow were again present, particularly a recirculating zone on the chamber wall downstream of the sinus of Valsalva. This region was initially observed at a Reynolds number of about 200 and grew with increasing flow rates.

Engelman *et al* (1980) performed comparative studies on the steady laminar flow characteristics of Starr-Edwards caged-ball and Björk-Shiley tilting-disk valves in the mitral and aortic positions. The finite element method was used to model planar non-Newtonian fluid flow through the prostheses. The location of major eddies were identified in velocity vector plots for both valve types. Simulations using a Newtonian model were also carried out with markedly different results to the non-Newtonian model being reported for each configuration case.

Peskin (1977) developed a computational method whereby the mathematical problem is formulated in such a way that the heart muscle and valves appear as special regions of the flow where extra forces apply. These forces give the valves and tissue walls their material and physiological properties. He studied various mitral valves in a planar representation of the heart by assuming laminar Newtonian time-dependent flow. The flow equations were coupled to a nonlinear system of equations describing boundary motion and forces. Unfortunately, the method was restricted to very low Reynolds numbers ($\approx 1/25$ of the appropriate value) due to impractical computational demands. The study illustrated the use of computational methods

to optimise prosthetic heart valve design and encouraged the use of the method to examine valve performance when subjected to changes in the physiological state of the heart (e.g. exercise). Further background and applications of the method are available (Peskin, 1982; McQueen and Peskin, 1983).

Yang and Wang (1980, 1983) applied the $k - \epsilon$ turbulence model to investigate the problem of steady turbulent flow through a disk-type prosthetic heart valve in a constant diameter chamber. They predicted streamline, velocity, stress and pressure fields by solving a set of simultaneous algebraic finite difference equations using a line-iteration technique. Unlike laminar flow studies which can have up to four separated flow regions in a constant diameter chamber (Underwood and Mueller, 1977) and three separated regions in an aortic-shaped chamber (Underwood and Mueller, 1979), the turbulent study uncovered only two such regions. Specifically, attached to the downstream side of the sewing ring and the disk occluder — there was no recirculation zone present along the chamber wall. High levels of fluid stress and wall shear stress were also evident.

Idelsohn *et al* (1985) carried out a comparative study on steady laminar flow through three common prosthetic heart valves. Using the finite element method, velocity, stress and pressure fields were calculated for caged-ball, caged-disk and tilting-disk prostheses in an aortic-shaped chamber. Regions of accelerated and stagnant flow were presented with the aid of computer graphics. Also, the location and magnitude of maximum shear stresses were found. Although these positions were generally in good agreement with experimental measurements, the levels of stress were much lower than those observed in physical simulators. The discrepancy between the numerical and experimental values indicated the important contribution of turbulent shear stresses.

Stevenson and Yoganathan (1985a, 1985b) conducted a numerical investigation for steady turbulent flow through axisymmetric models of trileaflet tissue aortic heart valves. Body conforming grid systems were generated and the turbulent flow equations were solved using finite differences and successive over-relaxation. They computed the flow patterns for five valve geometries ranging from very stenotic to a natural heart valve model. Good agreement was reported between experimental measurements and simulation data for velocity, shear stress and pressure drops. Their numerical model provided much useful information and may become a valuable tool for the development of future tissue prostheses.

In the following chapters three numerical models are developed, each providing substantial improvement in applicability upon the previous.

CHAPTER 4

MODEL I — LAMINAR FLOW THROUGH A CAGED-DISK PROSTHESIS

4.1 Introduction

The first model investigated is for steady laminar flow through the idealized planar geometry of a disk-type valve. Although very simple, this model clearly demonstrates the potential effectiveness and usefulness of more sophisticated numerical and mathematical models. The method of finite differences is used to solve the model. Numerical solutions are provided for several Reynolds numbers. The results of the study presented in this chapter have been published (Appendix C).

4.2 Mathematical Model

The mathematical modelling of blood flow through the complex geometry of a prosthetic heart valve is a difficult task. In such a problem the complex geometries of the valve must be simplified so they can be studied numerically. The simplifications must still contain the essential features of the valve. An approximate solution to the realistic problem may then be deduced from the study performed on the simplified model with the necessary assumptions taken into account. Thus, an insight into the real-life problem can be obtained.

The present analysis is performed on a disk-type prosthetic heart valve which can be used in either the mitral or aortic position. In this study the valve is assumed to be in the aortic position. The sinuses of Valsalva and the structure of the valve cage will undoubtedly influence the flow field near an aortic valve. However, the

occluder and the sewing ring will remain the overwhelming factors influencing the flow characteristics of the valve. Thus, it was decided not to include the sinuses and the valve cage in the present simple model. For the purpose of mathematical modelling, the laminar incompressible two-dimensional steady flow of a homogeneous Newtonian fluid with constant viscosity is assumed. The flow is considered during the greater part of systole when the valve is fully open. Underwood and Mueller (1977) noted this as the worst case in terms of the extent of flow separation and the magnitude of fluid stresses.

A simple geometry is employed for which experimental data is available. The essential features present in this model are the sewing ring, the disk occluder, the aortic wall and a Poiseuille inflow velocity profile. The flow is assumed to be symmetric about the centreline, and hence only one half of the actual geometry is used for the analysis. A definition sketch for the disk valve model is given in Figure 4.2.1. The dimensions of the valve are given in Figure 4.2.2.

Based upon the foregoing assumptions, the governing equations for two-dimensional steady incompressible flow of a Newtonian fluid with no body forces are the well known continuity equation

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0 \quad (4.2.1)$$

and the Navier-Stokes equations

$$u \frac{\partial u}{\partial x} + v \frac{\partial u}{\partial y} + \frac{\partial p}{\partial x} = \frac{1}{Re} \left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} \right) \quad (4.2.2)$$

$$u \frac{\partial v}{\partial x} + v \frac{\partial v}{\partial y} + \frac{\partial p}{\partial y} = \frac{1}{Re} \left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2} \right) \quad (4.2.3)$$

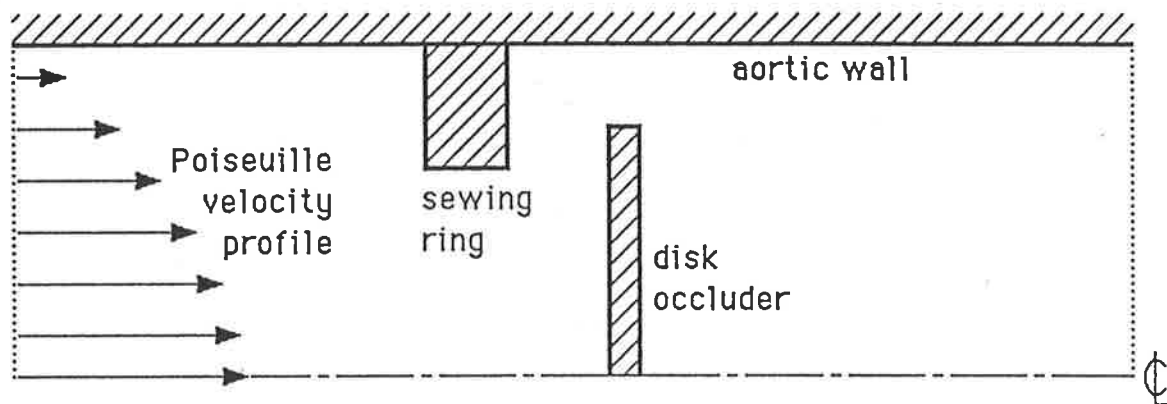


Figure 4.2.1

Geometry and inflow velocity profile for the Model I disk valve.

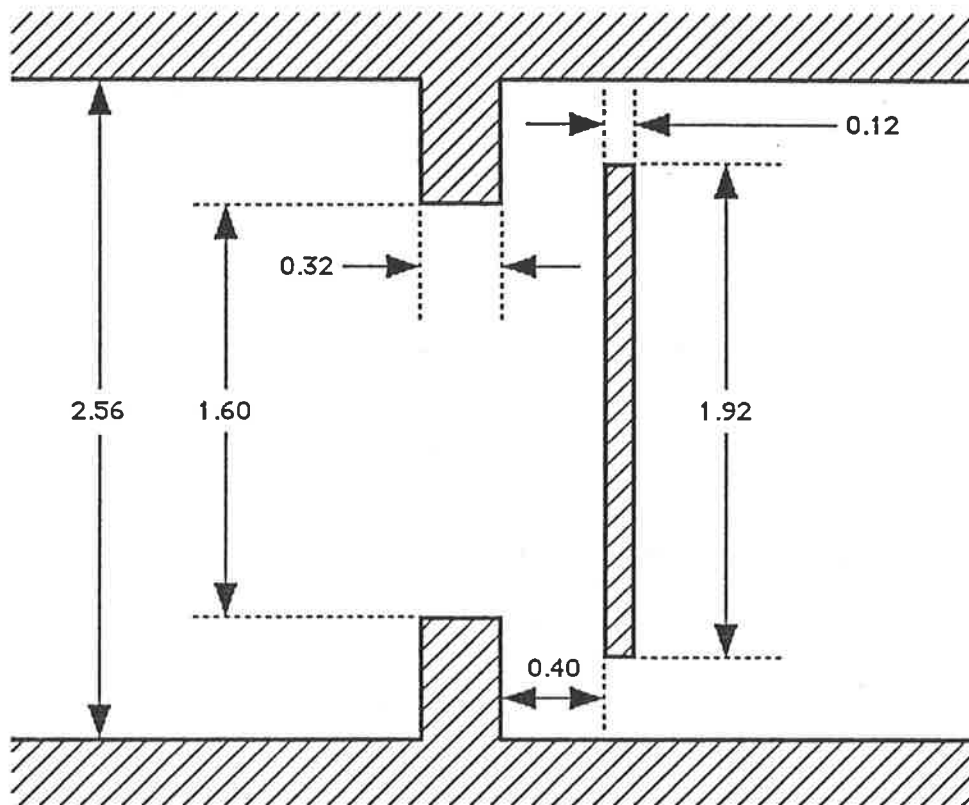


Figure 4.2.2

Dimensions (cm) of the fully open Model I disk valve.

where u and v are the horizontal and vertical velocity components, respectively, and p is the pressure. The Reynolds number Re in Model I is defined as

$$Re = \frac{D\bar{U}}{\nu} \quad (4.2.4)$$

where $\bar{U}$ is the average inflow velocity, D is the channel width and ν is the kinematic viscosity of the fluid.

By introducing the stream function $\psi(x, y)$ and the vorticity function $\omega(x, y)$ defined as follows

$$u = \frac{\partial \psi}{\partial y} \quad \text{and} \quad v = -\frac{\partial \psi}{\partial x} \quad (4.2.5)$$

and

$$\omega = \frac{\partial v}{\partial x} - \frac{\partial u}{\partial y} \quad (4.2.6)$$

the derivatives involving pressure appearing in equations (4.2.2) and (4.2.3) can be eliminated by cross differentiation and subtraction. The vorticity-transport equation and Poisson's equation are then obtained. That is,

$$\frac{\partial \psi}{\partial y} \frac{\partial \omega}{\partial x} - \frac{\partial \psi}{\partial x} \frac{\partial \omega}{\partial y} = \frac{1}{Re} \left(\frac{\partial^2 \omega}{\partial x^2} + \frac{\partial^2 \omega}{\partial y^2} \right) \quad (4.2.7)$$

$$-\omega = \frac{\partial^2 \psi}{\partial x^2} + \frac{\partial^2 \psi}{\partial y^2} \quad (4.2.8)$$

The nonlinearity of the coupled partial differential equations and the disk valve geometry make it impossible to obtain a solution through an analytic approach. Instead, the use of finite difference techniques provides a means by which an approximate numerical solution for the problem may be obtained. Once a numerical

solution becomes available the velocity components can be calculated using a discretisation of equation (4.2.5). The shear stress defined by

$$\sigma_{xy} = -\frac{1}{Re} \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right) \quad (4.2.9)$$

can then be obtained through a further discretisation, as can the horizontal normal stress

$$\sigma_{xx} = -\frac{2}{Re} \left(\frac{\partial u}{\partial x} \right) \quad (4.2.10)$$

The boundary conditions imposed are:

Upstream boundary

A fully developed Poiseuille velocity profile for channel flow is assumed at the inflow boundary. Flow in large arteries is found to assume such profiles. To ensure a horizontal inflow, the vertical velocity component v is set to zero. The inflow stream function and vorticity are subsequently computed from equations (4.2.5) and (4.2.6).

Centreline

The stream function is given an arbitrary value of zero along the centreline. The values of the vorticity and the vertical velocity component are also equated to zero along this axis of symmetry.

Solid walls

The stream function is given a constant (non-zero) value along the entire solid boundary which includes the sewing ring and the aortic wall. Along the occluder boundary the stream function is equated to zero. The no-slip boundary conditions

ensure that both velocity components are zero in these locations. The vorticity on solid walls is calculated from equations (4.2.5) and (4.2.6) by taking a Taylor series expansion of the stream function out from the wall.

Corner points

The value of the stream function at corner points deserves no special treatment and is taken as a constant value. The vorticity at a convex corner point is calculated as an average of two values obtained by considering the x and y directions separately. The vorticity in concave corner points is equated to zero.

Downstream boundary

The outflow boundary conditions employed by Friedman (1972) for the stream function and vorticity are also used here. These conditions are

$$\frac{\partial \psi}{\partial x} = 0 \quad \text{and} \quad \frac{\partial \omega}{\partial x} - Re \frac{\partial \psi}{\partial y} \left(\omega + \frac{\partial^2 \psi}{\partial y^2} \right) = 0 \quad (4.2.11)$$

which satisfy the requirements for horizontal outflow and constant pressure far downstream. The distance of the outflow boundary conditions from the disk valve is considered to be sufficiently far so as not to severely influence the flow upstream. Clearly, a longer outflow region would be desirable but this was not computationally feasible at the time. The vorticity along the downstream boundary is easily computed from these equations.

4.3 Finite Difference Formulation

Before the mathematical model can be formulated into a set of finite difference

equations a computational mesh must be constructed. For simplicity a regularly spaced mesh is chosen and boundary locations are made to coincide with the nearest grid point. The dimensions of the grid spacing in x and y are denoted by Δx and Δy , respectively, and are both equal to 0.04 cm . The computational mesh is a 164×33 grid (5412 grid points). During the initial code development and testing several other grid densities were investigated. Comparison between the numerical solutions indicated some ambiguous results occurring at the lower grid densities. These grids were discarded. A section of the final grid system is shown in Figure 4.3.1.

The governing equations (4.2.7) and (4.2.8) are now written in finite difference form. There are numerous ways in which this discretisation can be achieved. For this simple model the method employed by Friedman (1972) is used as the basis for the finite difference formulation. Defining H and K for convenience as

$$H = \psi_{i+1,j} - \psi_{i-1,j} \quad , \quad K = \psi_{i,j+1} - \psi_{i,j-1} \quad (4.3.1)$$

the advection terms in the vorticity transport equation (4.2.7) are approximated using first order accurate upwind differences if $H \geq 0$ and $K \geq 0$. That is,

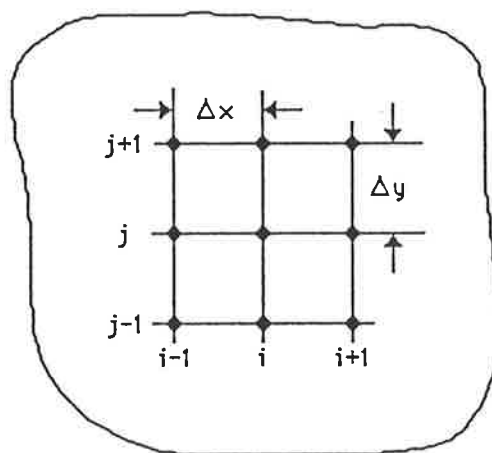
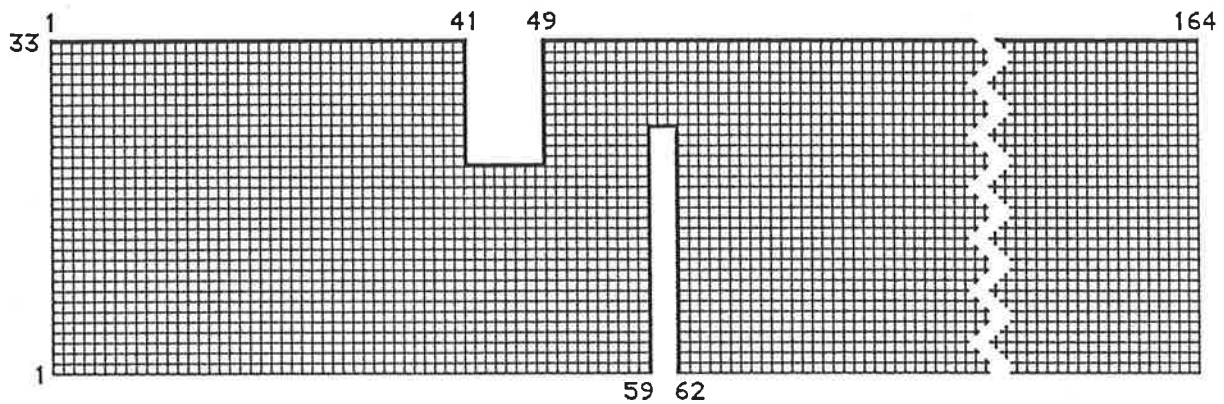
$$\left. \frac{\partial \omega}{\partial x} \right|_{i,j} \approx \frac{\omega_{i,j} - \omega_{i-1,j}}{\Delta x} \quad (4.3.2)$$

and

$$\left. \frac{\partial \omega}{\partial y} \right|_{i,j} \approx \frac{\omega_{i,j+1} - \omega_{i,j}}{\Delta y} \quad (4.3.3)$$

if $H \geq 0$ and $K \geq 0$.

The remaining first derivatives of ψ and second derivatives representing the diffusion terms are approximated by the well known central space approximations.



grid point
nomenclature

Figure 4.3.1

Finite difference grid (164×33) and grid nomenclature for the Model I disk valve.

Substitution into equation (4.2.7) and rearranging yields the following finite difference equation

$$\omega_{i,j} = \frac{\omega_{i+1,j} + \omega_{i-1,j} + \omega_{i,j+1} + \omega_{i,j-1} + \frac{Re}{2}(H\omega_{i,j+1} + K\omega_{i-1,j})}{4 + \frac{Re}{2}(H + K)} \quad (4.3.4)$$

if $H \geq 0$ and $K \geq 0$.

Similar finite difference equations are formulated for the other three possibilities, namely

$$\omega_{i,j} = \frac{\omega_{i+1,j} + \omega_{i-1,j} + \omega_{i,j+1} + \omega_{i,j-1} + \frac{Re}{2}(H\omega_{i,j+1} - K\omega_{i+1,j})}{4 + \frac{Re}{2}(H - K)} \quad (4.3.5)$$

if $H \geq 0$ and $K < 0$,

$$\omega_{i,j} = \frac{\omega_{i+1,j} + \omega_{i-1,j} + \omega_{i,j+1} + \omega_{i,j-1} + \frac{Re}{2}(K\omega_{i-1,j} - H\omega_{i,j-1})}{4 + \frac{Re}{2}(K - H)} \quad (4.3.6)$$

if $H < 0$ and $K \geq 0$, and

$$\omega_{i,j} = \frac{\omega_{i+1,j} + \omega_{i-1,j} + \omega_{i,j+1} + \omega_{i,j-1} - \frac{Re}{2}(K\omega_{i+1,j} + H\omega_{i,j-1})}{4 - \frac{Re}{2}(K + H)} \quad (4.3.7)$$

if $H < 0$ and $K < 0$.

The presence of Re in both the numerator and denominator in the equations above overcomes the cell Reynolds number restriction often associated with solving such fluid flow problems.

Boundary conditions are also discretised. A difficulty of any computational viscous fluid flow problem which employs the stream function–vorticity method is

that the values of vorticity on the no-slip solid boundaries are not known in advance but are required to solve the discretised equations. To overcome this difficulty equation (4.2.6) is used to provide an initial estimate for the wall vorticity once the initial values for the stream function are specified. The wall vorticity at the grid point (i, j) , denoted by $\omega_{i,j}$, is given by the second order accurate expression

$$\omega_{i,j} \approx \frac{3(\psi_{i,j} - \psi_{i+1,j})}{(\Delta x)^2} - \frac{\omega_{i+1,j}}{2} \quad (4.3.8)$$

when the solid wall is facing in the positive x -direction. Similar expression are used for the other possible configurations.

4.4 Numerical Solution

The numerical procedure employed to solve this model is similar to that used by Friedman (1972). It provides a way by which a numerical solution to a steady laminar flow at high Reynolds numbers can be obtained using a relatively small amount of computer time. The procedure is used in a step by step manner to calculate both the stream function and the vorticity as follows:

1. Set initial values for $\psi^{(1)}$ and $\omega^{(1)}$.
2. Use Successive Over Relaxation (SOR) with a relaxation factor of 1.8 only once to calculate the stream function at all the grid points immediately interior to the solid boundaries using the new vorticity values.
3. Calculate the stream function at all remaining interior grid points using the new values determined in Step 2 and the most recent vorticity values.
4. Return to Step 2 and repeat the process until a converged solution is obtained. Denote the converged result by $\bar{\psi}^{(k+1)}$.

5. Set

$$\psi^{(k+1)} = 0.96 \bar{\psi}^{(k+1)} + 0.04 \psi^{(k)} \quad (4.4.1)$$

6. Use the Gauss-Seidel iteration method only once to calculate the boundary vorticity values using the new stream function values.

7. Calculate the vorticity at all interior grid points using the new values determined in Step 6 and the latest stream function values.

8. Return to Step 6 and repeat the process until a converged solution is obtained. Denoted the converged result by $\bar{\omega}^{(k+1)}$.

9. Set

$$\omega^{(k+1)} = 0.3 \bar{\omega}^{(k+1)} + 0.7 \omega^{(k)} \quad (4.4.2)$$

10. Return to Step 2 and repeat the process until both

$$|\psi^{(k+1)} - \psi^{(k)}| < \epsilon_1 \quad (4.4.3)$$

and

$$|\omega^{(k+1)} - \omega^{(k)}| < \epsilon_2 \quad (4.4.4)$$

are satisfied.

The stream function was iterated using the SOR method until the difference between its value at consecutive iterate levels was less than 0.0005. The vorticity function was iterated with the Gauss-Seidel method until the difference between its value at consecutive iterate levels was less than 0.2 % of the maximum vorticity value. A requirement was that the convergence criteria be satisfied at every grid point. The global convergence conditions were set by assigning values to ϵ_1 and

ϵ_2 of 0.0005 and 0.2 % of the maximum vorticity value, respectively. Final global convergence was declared when equations (4.4.3) and (4.4.4) were both satisfied 30 times.

4.5 Discussion of Results

The computational procedure applied in this model has worked well. Converged numerical solutions are obtained for Reynolds numbers $Re = 50, 150, 500, 1000, 3500$ and 6000 . Stream function, vorticity, horizontal velocity, vertical velocity and shear stress solutions are computed at every grid point. The numerical solutions presented are of course subject to artificial viscosity errors introduced by the numerical procedure. This should be kept in mind when comparisons are made with experimental studies, particularly in regions of high velocity diagonal to grid lines.

Stream function contour plots are presented in Figure 4.5.1 for $Re = 50$ to 6000 . The extent of recirculating regions downstream of the disk occluder as well as upstream and downstream of the sewing ring are clearly seen. The location of the separation point on the occluder remained at the downstream corner for all Reynolds numbers tested. This has been the experience of other workers (Underwood and Mueller, 1977, 1979) when first order accurate upwind difference methods have been used for the advection terms. The large recirculating zone behind the occluder contained horizontal reverse flow velocities up to 1.57 times ($i = 80, j = 1$) the average inflow velocity for $Re = 6000$. An interesting feature in Figure 4.5.1 are the relative dimensions of the closed streamlines in the separated flow region immediately downstream of the occluder. As the Reynolds number increases from 500 to 6000, the streamlines, $\psi = -0.05, -0.10$ and -0.15 become markedly smaller, until the innermost one ($\psi = -0.05$) eventually vanishes for $Re > 1000$. Physically, the

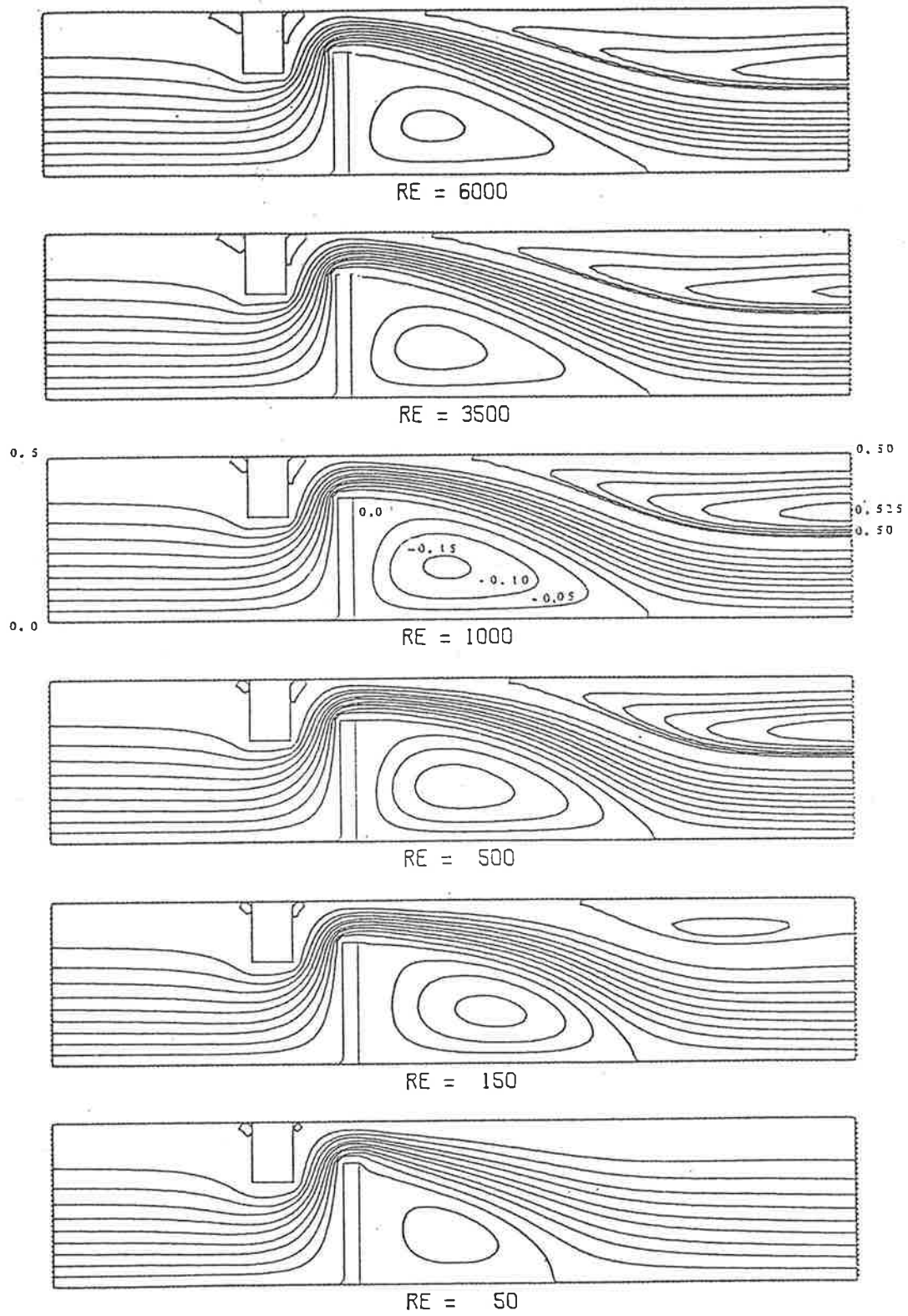


Figure 4.5.1

Computed streamlines for $Re = 50, 150, 500, 1000, 3500$ and 6000 .

flow velocity inside the separated zone decreases relative to the surrounding flow as the Reynolds number increases.

A recirculating region of large proportions also appeared along the aortic wall downstream of the occluder (Figure 4.5.1). Although it was not present at $Re = 50$, this region initially appeared at about $Re = 150$ and grew rapidly with higher Reynolds numbers. The separation point for this region was observed to move upstream as the Reynolds number increased. The largest value of wall shear stress occurring within this zone was -4.1 N m^{-2} ($i = 80, j = 33$) for $Re = 6000$.

Figures 4.5.2 and 4.5.3 show contour plots for the stream function, vorticity, total velocity and shear stress distributions for Reynolds numbers of 1000 and 6000, respectively. A huge region of stagnated flow alongside the aortic wall is evident in the total velocity plot. Changes to the vorticity contours are observed as the Reynolds number increases. The incredibly complex nature of the shear stress distributions in the near vicinity of the disk valve is also evident — information that is simply impossible to obtain in such totality from experimental measurements.

In Figure 4.5.4 plots of the computed horizontal velocity profiles at various cross sections of the flow field are shown. The changing structure of the horizontal velocity profiles at different locations downstream of the valve can be seen as the Reynolds number increases from 50 to 6000. They show the presence of the slow moving reversed flow region lying along the aortic wall. The potential for thrombus formation in this stagnated flow area is clearly enhanced, particularly if the endothelium has been damaged. This separated flow region has also been observed elsewhere — in experimental studies where it was noted as an extremely thin zone (Figliola and Mueller, 1977), and in other laminar numerical studies using a constant chamber diameter (Underwood and Mueller, 1977). It is interesting to

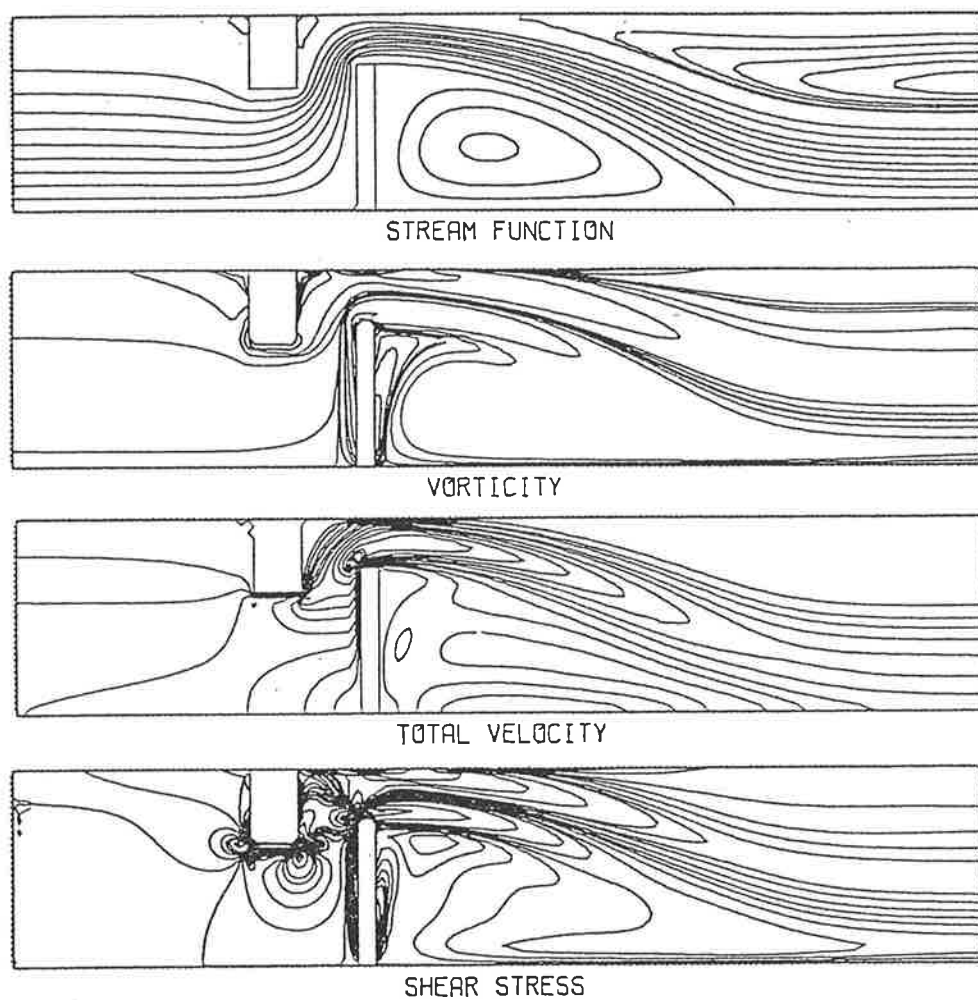


Figure 4.5.2

Computed stream function, vorticity, total velocity and shear stress contours for $Re = 1000$.

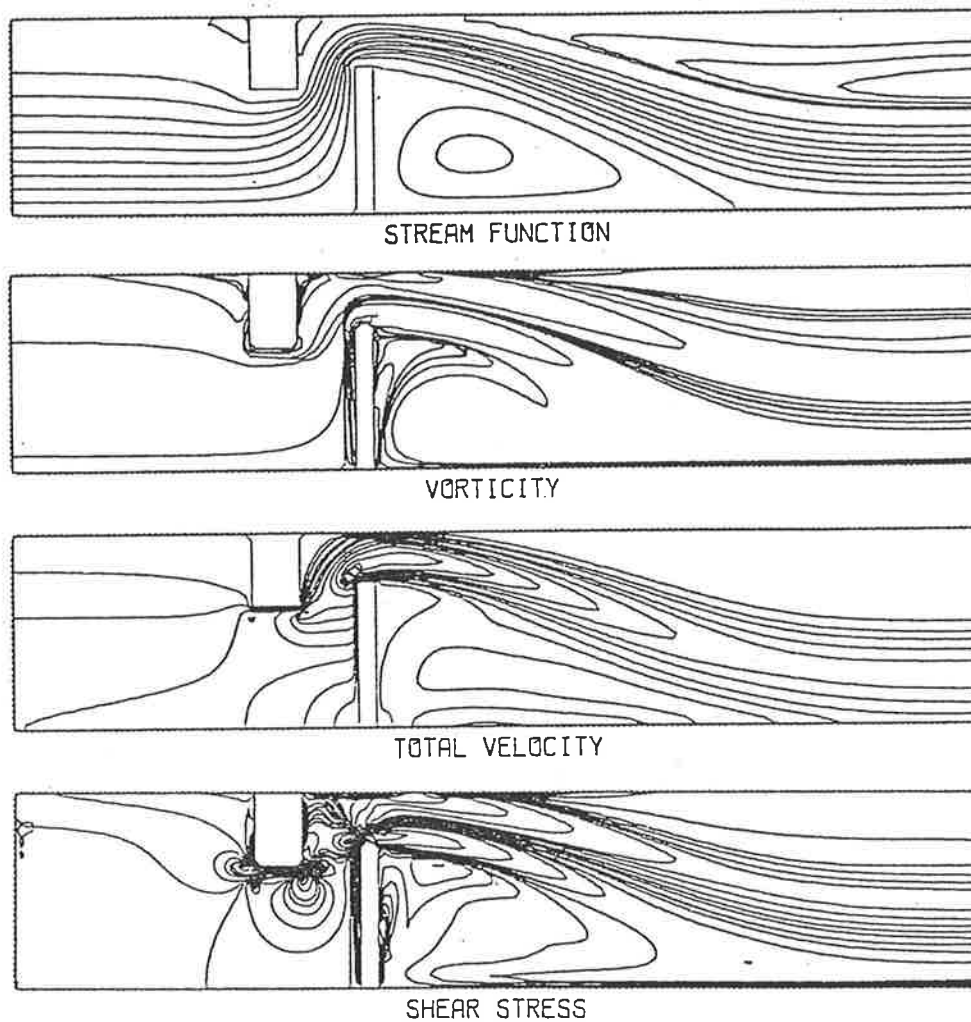


Figure 4.5.3

Computed stream function, vorticity, total velocity and shear stress contours for $Re = 6000$.

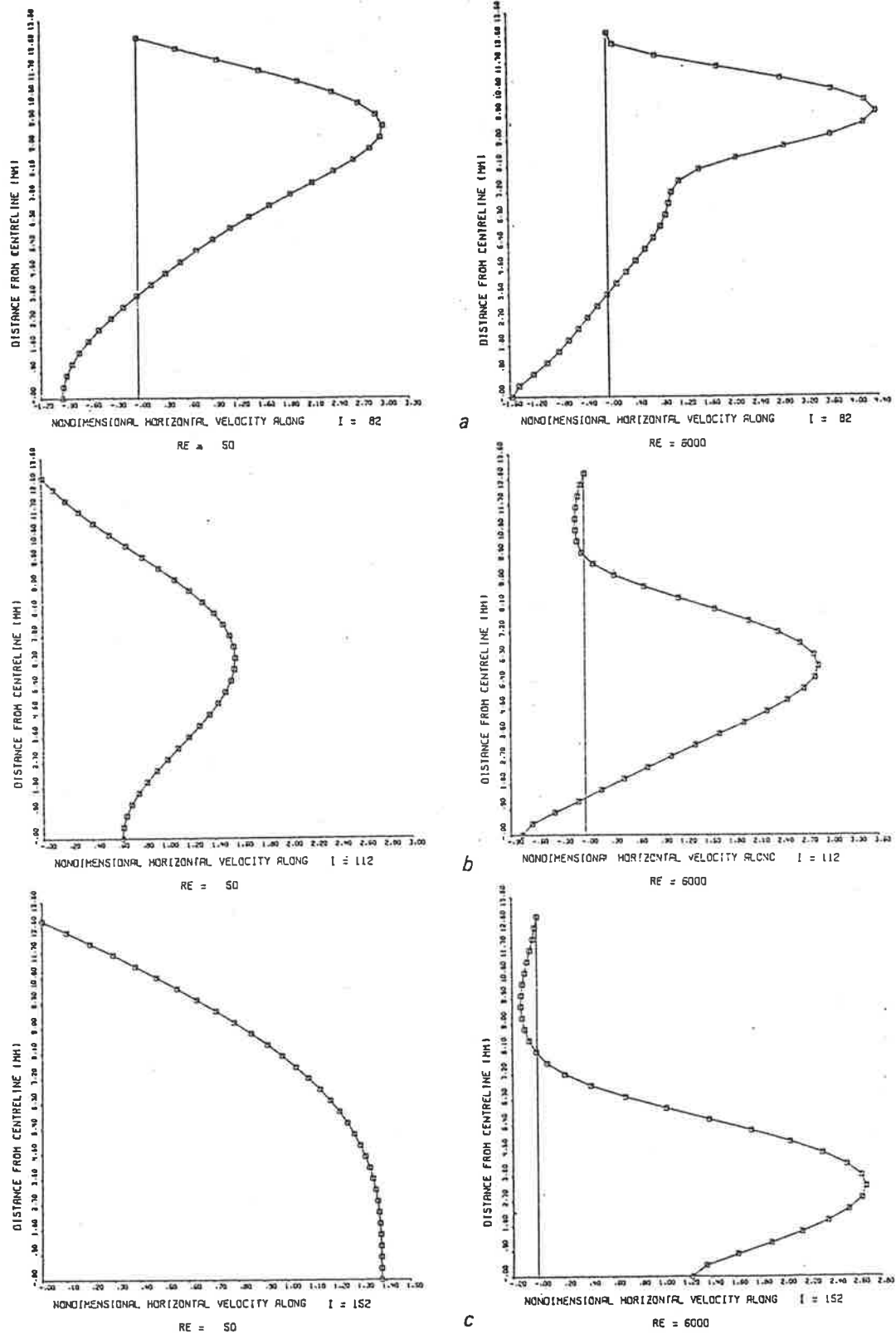


Figure 4.5.4

Computed horizontal velocity profiles for $Re = 50$ and $Re = 6000$ along grid lines (a) $i = 82$, (b) $i = 112$ and (c) $i = 152$.

note that whereas the $Re = 50$ horizontal velocity profile in Figure 4.5.4 (c) almost re-established the parabolic profile it had at inflow, the same can not be said for the $Re = 6000$ case. The great flow disturbance that occurs at this high Reynolds number persists far downstream.

The distribution of wall shear stress along the aortic wall is given in Figure 4.5.5 for $Re = 50$ and 6000. Large variations in the wall shear stress occur as a result of lateral flow (induced by the valve geometry) impinging upon the vessel boundary. The “sharp” corners of the sewing ring structure also influence the wall shear stress levels. Points of flow separation are indicated in Figure 4.5.5 by zero shear stress. The maximum levels of wall shear stress computed upstream of the sewing ring remained below $0.7 N m^{-2}$ for $Re = 6000$. The location of peak wall shear stress remained fixed ($i = 61, j = 33$) for all Reynolds numbers. The shear stress at this point was $61.8 N m^{-2}$ for $Re = 6000$. This is sufficient to damage endothelial cells, adhered red blood cells and platelets. However, the point of peak wall stress coincides with the location of the sinuses of Valsalva which are not considered in this simple model. In reality, the presence of these hollowed-out areas in the aortic root region would reduce the amount of stress, possibly averting extensive damage. The levels of wall shear stress diminished rapidly downstream from this point for all Reynolds numbers. The level of shear stress computed along the sewing ring reached a maximum value of $44.4 N m^{-2}$ ($i = 47, j = 21$) for $Re = 6000$. The maximum level of shear stress in the entire flow domain occurred just below the upstream corner of the occluder reaching $82.8 N m^{-2}$ ($i = 59, j = 23$) for $Re = 6000$. The largest shear stress measured experimentally by Figliola and Mueller (1977) was $105 N m^{-2}$. Table 2.4.1 gives an indication of the type of damage that can be expected to result from exposure to such stress levels. For example, damage to

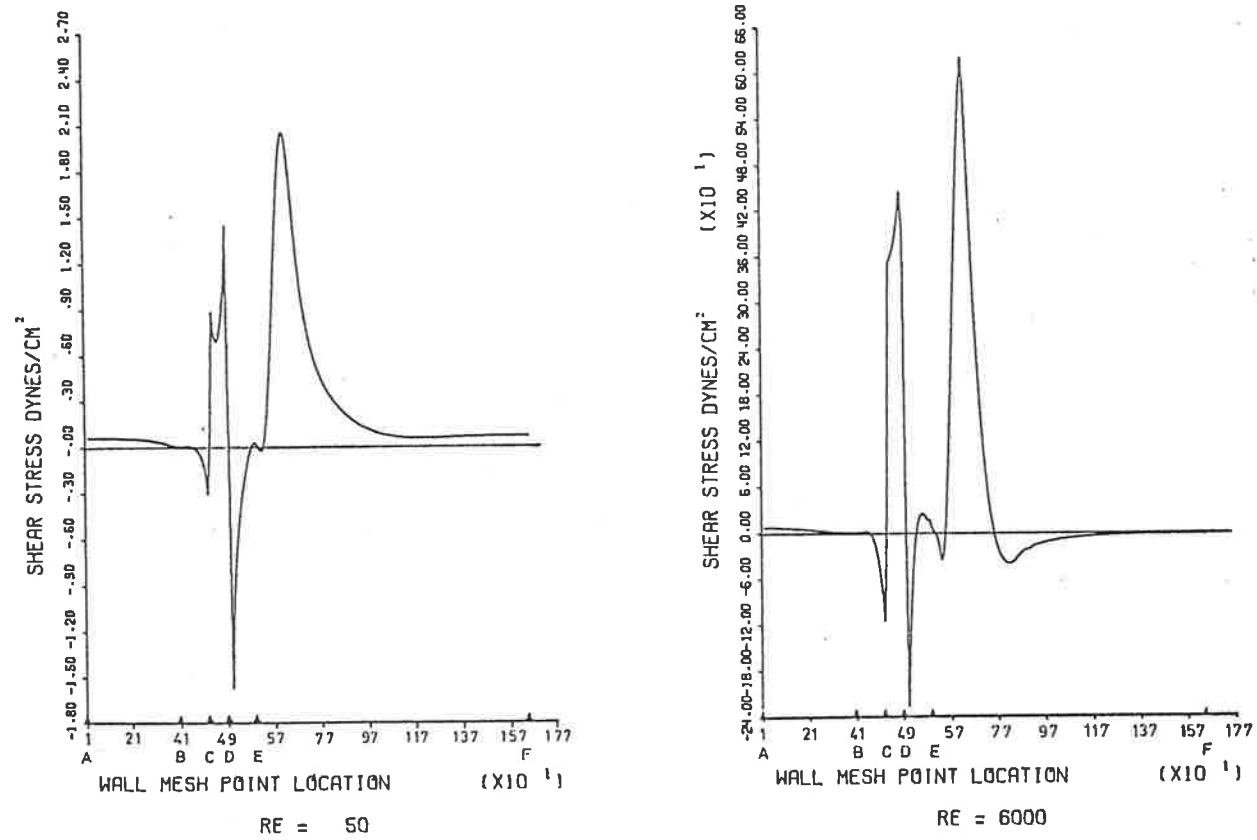


Figure 4.5.5

Computed wall shear stress distributions (dynes cm^{-2}) for $\text{Re} = 50$ and $\text{Re} = 6000$.

adhered red blood cells and platelets is highly probable.

4.6 Conclusions

The laminar shear stresses computed with Model I appear likely to cause damage to adhered red blood cells, endothelial cells and platelets in several regions of the flow domain. Damage to red blood cells in flow is unlikely to occur based on the present results. However, when flow becomes turbulent, the turbulent stresses which are always much larger than viscous stresses could haemolyse blood cells in the vicinity of solid surfaces.

The potential for thrombus formation exists near the regions of separated flow observed at all flow rates. Thrombosis may occur in the recirculating region immediately behind the occluder, attaching itself to the restrictive cage, possibly inhibiting the mechanical performance of the disk valve. Thromboplastin may also be released from blood cells in the separated region lying alongside the aortic wall to enhance clotting. These conditions are clearly undesirable.

Although these results are obtained quite easily from a simple laminar model, they do provide a good illustration of the detailed descriptions of shear stress and velocity that are possible in the near vicinity of prosthetic heart valves. Both the flow patterns and the stress distributions indicate areas of concern and potential danger. However, it should be emphasised that turbulence will occur at the higher Reynolds numbers reported for Model I. The subsequent models incorporate the effects of turbulence.

CHAPTER 5

MODEL II — TURBULENT FLOW THROUGH A CAGED-DISK PROSTHESIS

5.1 Introduction

The second model examined is an extension of Model I as it assumes turbulent flow. It examines steady axisymmetric flow through a Kay-Shiley disk valve. The effects of turbulence are incorporated into this model by employing the $k - \epsilon$ turbulence model embedded within the TEACH-T computer code (Gosman and Ideriah, 1976; Appendix A). This turbulence model has been more widely applied to turbulent flow problems than any other model and is considered the most suitable model available at the present state of the art (Rodi, 1980).

The main aims of this valve model are to extend the work presented in Chapter 4 into the turbulent flow regime, obtain quantitative estimates for velocity, wall shear stress and Reynolds stress distributions in the flow field surrounding the prosthetic valve and to compare the turbulent numerical solutions with experimental observations and previous laminar flow solutions. Experimental data for steady flow through prosthetic heart valves is available in Figliola and Mueller (1977), Yoganathan *et al* (1978, 1979), Figliola and Mueller (1981) and Herkes and Lloyd (1981). The availability of this experimental data will help assess the accuracy of the $k - \epsilon$ model before the procedure is applied to a more realistic boundary-fitted curvilinear coordinate system. The results presented in this chapter have also been published (Appendix D).

5.2 Mathematical Model

The mathematical modelling of turbulent blood flow through the complex geometry of a prosthetic heart valve is a more difficult task than the case of laminar flow discussed in Model I. In such a problem the complex geometries of the valve may be simplified so they can be studied numerically. Even though the practical need for such computations is pressing, an important objective of this model is to examine the accuracy of the $k - \epsilon$ turbulence model when used for prosthetic heart valve flow calculations. Hence, a simple geometry is again employed for which experimental data is available.

The present study is performed on a disk-type prosthetic valve which can be used in either the mitral or aortic position. Although this valve is very infrequently used in practice, it is hoped that studying this valve will help form the basis of future more realistic computations. Here the valve is assumed to be in the aortic position as with previous experimental studies (Figliola and Mueller, 1981; Herkes and Lloyd, 1981; Yoganathan *et al*, 1978, 1979). The blood is assumed to have a density of 1.06 gm cm^{-3} and a laminar viscosity of $3.71 \times 10^{-3} \text{ N sec m}^{-2}$. The assumption of blood behaving like a Newtonian fluid is a reasonable approximation for studying its dynamics in large arteries.

Also, the blood flow is assumed to be turbulent and is only considered during the greater part of systole when the valve is fully open. The essential features of the valve are shown in Figure 5.2.1 and include the sewing ring, the disk occluder and the aortic wall. The boundaries of the flow domain are assumed to be smooth, rigid and impermeable. Hence, a no-slip condition is applied to the mean velocities at the walls. A uniform velocity profile is assumed at inflow and only half the actual valve geometry is considered for analysis due to symmetry. The dimensions of the

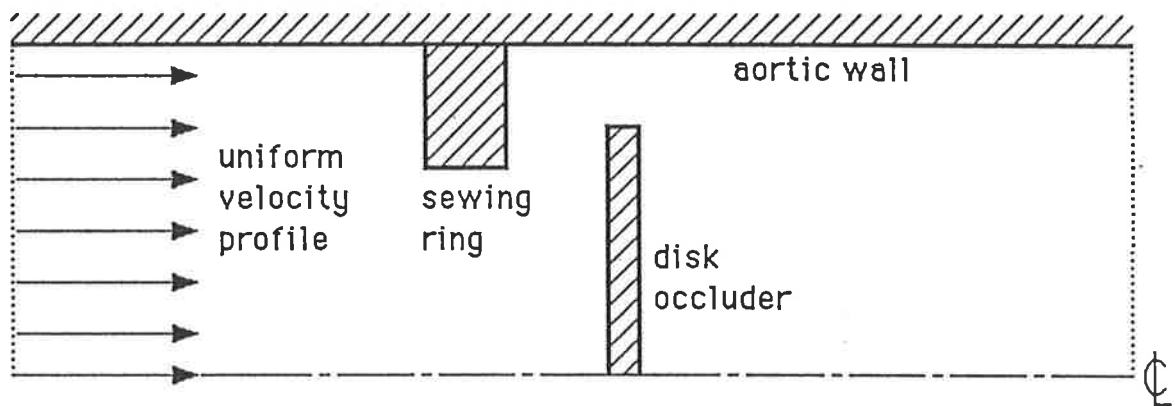


Figure 5.2.1

Geometry and inflow velocity profile for the Model II disk valve.

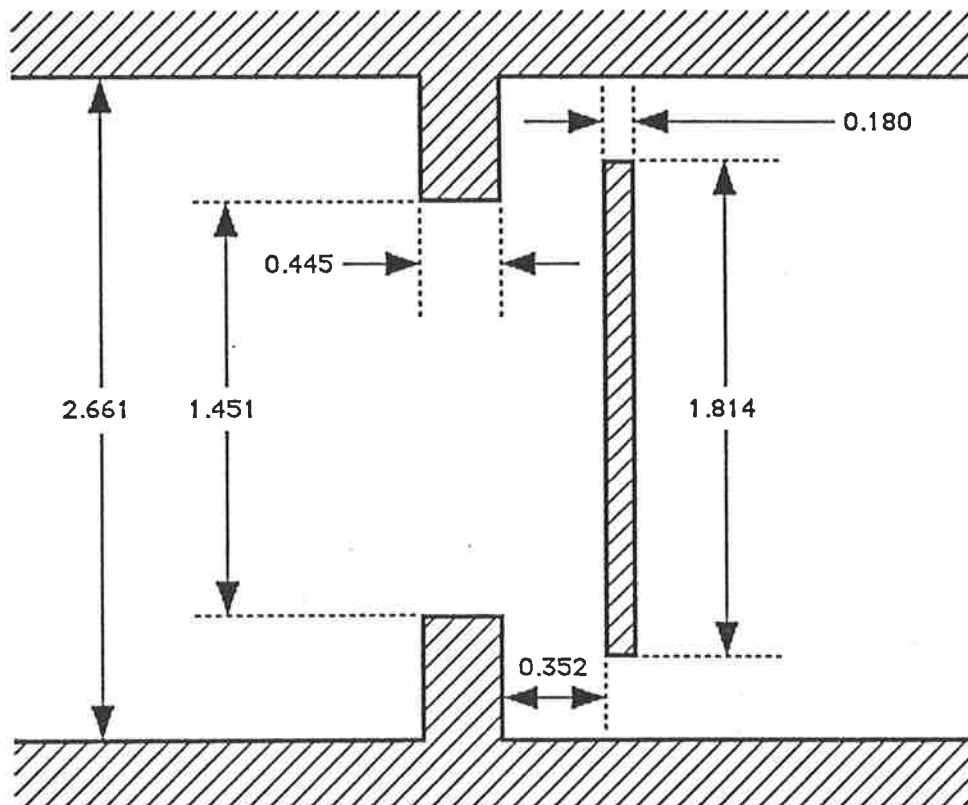


Figure 5.2.2

Dimensions (cm) of the fully open Model II disk valve.

valve are given in Figure 5.2.2. The flow field of the valve is considered for the range $-1 \leq z/D \leq 5$ where the axial distance z is measured from the downstream face of the sewing ring and D is the chamber diameter ($D = 2.661 \text{ cm}$). The velocity components, pressure and turbulence quantities are assigned zero gradients at outflow. The placement of the downstream boundary is considered suitably far from the disk valve so that the prescribed outflow conditions will not influence the flow upstream.

The time-averaged mass and momentum conservation equations for steady, turbulent axisymmetric flows are presented below (Launder and Spalding, 1974).

Continuity :

$$\frac{\partial}{\partial z}(r\rho U) + \frac{\partial}{\partial r}(r\rho V) = 0 \quad (5.2.1)$$

where U and V are the time-averaged velocities in the axial (z) and the radial (r) directions, respectively, and ρ is the fluid density.

Axial momentum :

$$\begin{aligned} \frac{1}{r} \left[\frac{\partial}{\partial z}(r\rho UU) + \frac{\partial}{\partial r}(r\rho VU) \right] = \frac{1}{r} \left[\frac{\partial}{\partial z} \left(r\mu_{eff} \frac{\partial U}{\partial z} \right) + \frac{\partial}{\partial r} \left(r\mu_{eff} \frac{\partial U}{\partial r} \right) \right] \\ - \frac{\partial p}{\partial z} + S_u \end{aligned} \quad (5.2.2)$$

where

$$S_u = \frac{\partial}{\partial z} \left(\mu_{eff} \frac{\partial U}{\partial z} \right) + \frac{1}{r} \frac{\partial}{\partial r} \left(r\mu_{eff} \frac{\partial V}{\partial z} \right) \quad (5.2.3)$$

Radial momentum :

$$\begin{aligned} \frac{1}{r} \left[\frac{\partial}{\partial z}(r\rho UV) + \frac{\partial}{\partial r}(r\rho VV) \right] = \frac{1}{r} \left[\frac{\partial}{\partial z} \left(r\mu_{eff} \frac{\partial V}{\partial z} \right) + \frac{\partial}{\partial r} \left(r\mu_{eff} \frac{\partial V}{\partial r} \right) \right] \\ - \frac{\partial p}{\partial r} - \mu_{eff} \frac{V}{r^2} + S_v \end{aligned} \quad (5.2.4)$$

where

$$S_v = \frac{\partial}{\partial z} \left(\mu_{eff} \frac{\partial U}{\partial r} \right) + \frac{1}{r} \frac{\partial}{\partial r} \left(r \mu_{eff} \frac{\partial V}{\partial r} \right) - \mu_{eff} \frac{V}{r^2} \quad (5.2.5)$$

The effective viscosity μ_{eff} is defined as the sum of the laminar viscosity μ and the turbulent viscosity μ_t . That is,

$$\mu_{eff} = \mu + \mu_t \quad (5.2.6)$$

where the turbulent viscosity is not a property of the fluid but depends on the state of the turbulence (Rodi, 1980) and is given by

$$\mu_t = \frac{C_\mu \rho k^2}{\epsilon} \quad (5.2.7)$$

The additional equations for the turbulent kinetic energy k and its dissipation rate ϵ for steady, axisymmetric flow required to complete the turbulent flow model are presented below.

Turbulent kinetic energy :

$$\begin{aligned} \frac{1}{r} \left[\frac{\partial}{\partial z} (r \rho U k) + \frac{\partial}{\partial r} (r \rho V k) \right] &= \frac{1}{r} \left[\frac{\partial}{\partial z} \left(r \frac{\mu_{eff}}{\sigma_k} \frac{\partial k}{\partial z} \right) + \frac{\partial}{\partial r} \left(r \frac{\mu_{eff}}{\sigma_k} \frac{\partial k}{\partial r} \right) \right] \\ &+ S_k \end{aligned} \quad (5.2.8)$$

where

$$S_k = G - C_D \rho \epsilon \quad (5.2.9)$$

Kinetic energy dissipation rate :

$$\begin{aligned} \frac{1}{r} \left[\frac{\partial}{\partial z} (r \rho U \epsilon) + \frac{\partial}{\partial r} (r \rho V \epsilon) \right] &= \frac{1}{r} \left[\frac{\partial}{\partial z} \left(r \frac{\mu_{eff}}{\sigma_\epsilon} \frac{\partial \epsilon}{\partial z} \right) + \frac{\partial}{\partial r} \left(r \frac{\mu_{eff}}{\sigma_\epsilon} \frac{\partial \epsilon}{\partial r} \right) \right] \\ &+ S_\epsilon \end{aligned} \quad (5.2.10)$$

where

$$S_\epsilon = C_1 \frac{\epsilon}{k} G - C_2 \rho \frac{\epsilon^2}{k} \quad (5.2.11)$$

The generation of kinetic energy from the mean flow by turbulent shear stress is given by

$$G = \mu_t \left\{ 2 \left[\left(\frac{\partial U}{\partial z} \right)^2 + \left(\frac{\partial V}{\partial r} \right)^2 + \left(\frac{V}{r} \right)^2 \right] + \left(\frac{\partial U}{\partial r} + \frac{\partial V}{\partial z} \right)^2 \right\} + S_G \quad (5.2.12)$$

where

$$S_G = -\frac{2}{3} \mu_t \left[\frac{1}{r} \frac{\partial}{\partial r} (rV) + \frac{\partial U}{\partial z} \right]^2 \quad (5.2.13)$$

The coefficients C_1 , C_2 , C_D , C_μ , σ_k and σ_ϵ are constants at high Reynolds numbers (Launder and Spalding, 1974) and their values are taken as:

$$C_1 = 1.44, C_2 = 1.92, C_D = 1.0, C_\mu = 0.09, \sigma_k = 1.0, \sigma_\epsilon = 1.3$$

The Reynolds number Re in Model II is defined as

$$Re = \frac{DU_0}{\nu} \quad (5.2.14)$$

where D is the chamber diameter, U_0 is the mean axial velocity at inflow and ν is the kinematic viscosity.

The Boussinesq eddy-viscosity concept assumes that the turbulent stresses are directly proportional to the mean velocity gradients, and this is expressed mathematically as

$$\overline{\rho u'_i u'_j} = -\mu_t \left(\frac{\partial U_i}{\partial x_j} + \frac{\partial U_j}{\partial x_i} \right) + \frac{2}{3} \rho k \delta_{ij} \quad (5.2.15)$$

where u'_i and u'_j are the fluctuating components of the velocities in the i and j directions, respectively, and δ_{ij} is the Kronecker delta.

5.3 Finite Difference Formulation

An irregular staggered grid system consisting of 1056 grid points (44×24) is used for this model. Several other grid densities were also investigated during code development to ensure a suitable grid density was selected. A section of the final computational mesh is shown in Figure 5.3.1. In such a grid system the velocity components are computed for the points that lie midway along the faces of the control volumes. Hence, the mean axial velocity U is calculated on the faces that are normal to the z -direction. Similarly, the mean radial velocity V is calculated on the faces that are normal to the r -direction. This method of grid specification removes difficulties that would otherwise be encountered (Patankar, 1980). Using this convention the boundaries of the flow domain lie midway between the outermost pairs of grid lines. The horizontal grid lines were uniformly spaced with $\Delta y = 0.06048 \text{ cm}$, but the vertical grid lines were irregularly spaced allowing concentration of grid points in desired locations, in particular the region between the sewing ring and the disk occluder.

The mathematical equations must be discretised into finite difference form for it to be solved numerically on a digital computer. With the TEACH-T computer code (Gosman and Ideriah, 1976) the finite difference equations are derived directly from the conservation and macroscopic physical laws by a control volume analysis, thus ensuring correct representation of the conservation laws. Consider the case of transport of an extensive quantity Φ (e.g. mass, momentum, energy) through the control volume shown in Figure 5.3.2. The rate of increase of Φ in the control

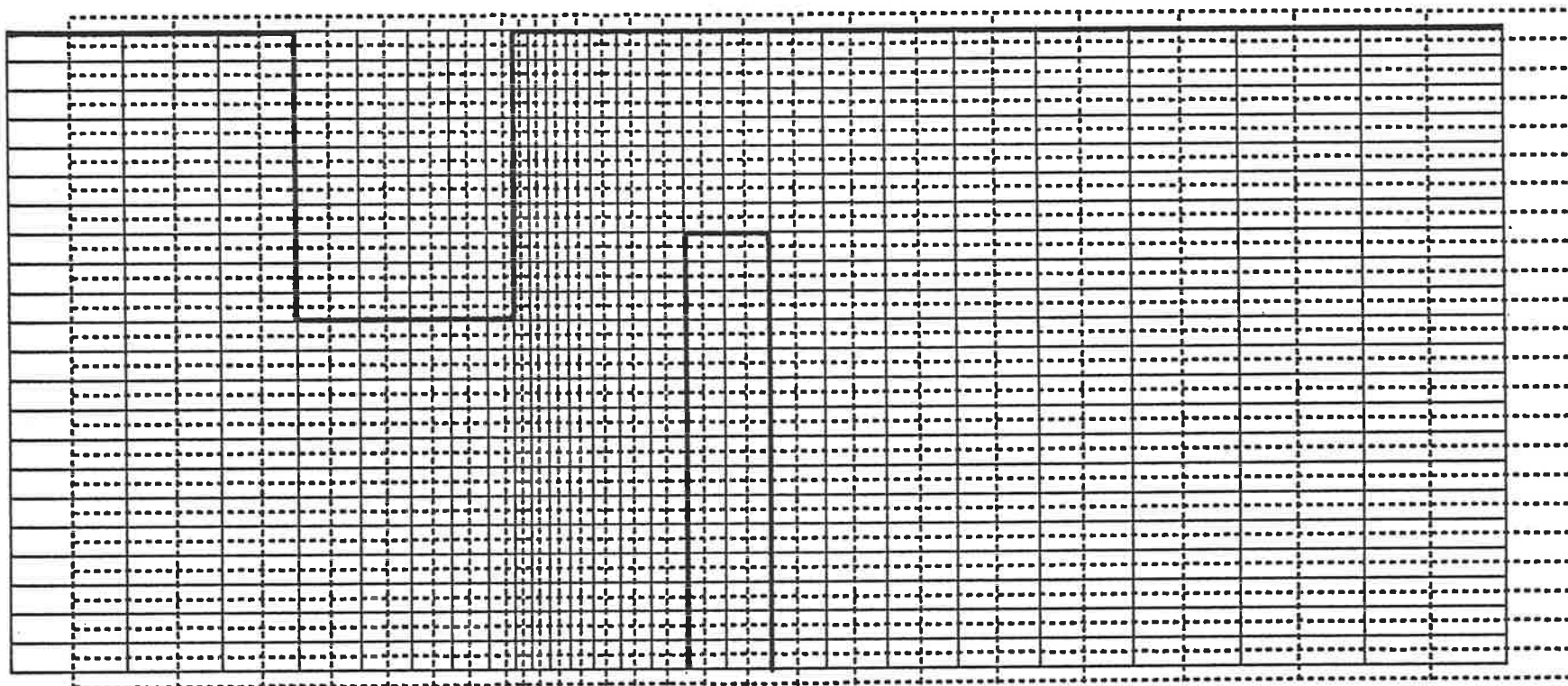


Figure 5.3.1

Section ($i = 6$ to $i = 31$) of the irregular staggered computational grid (44×24) for the Model II disk valve.

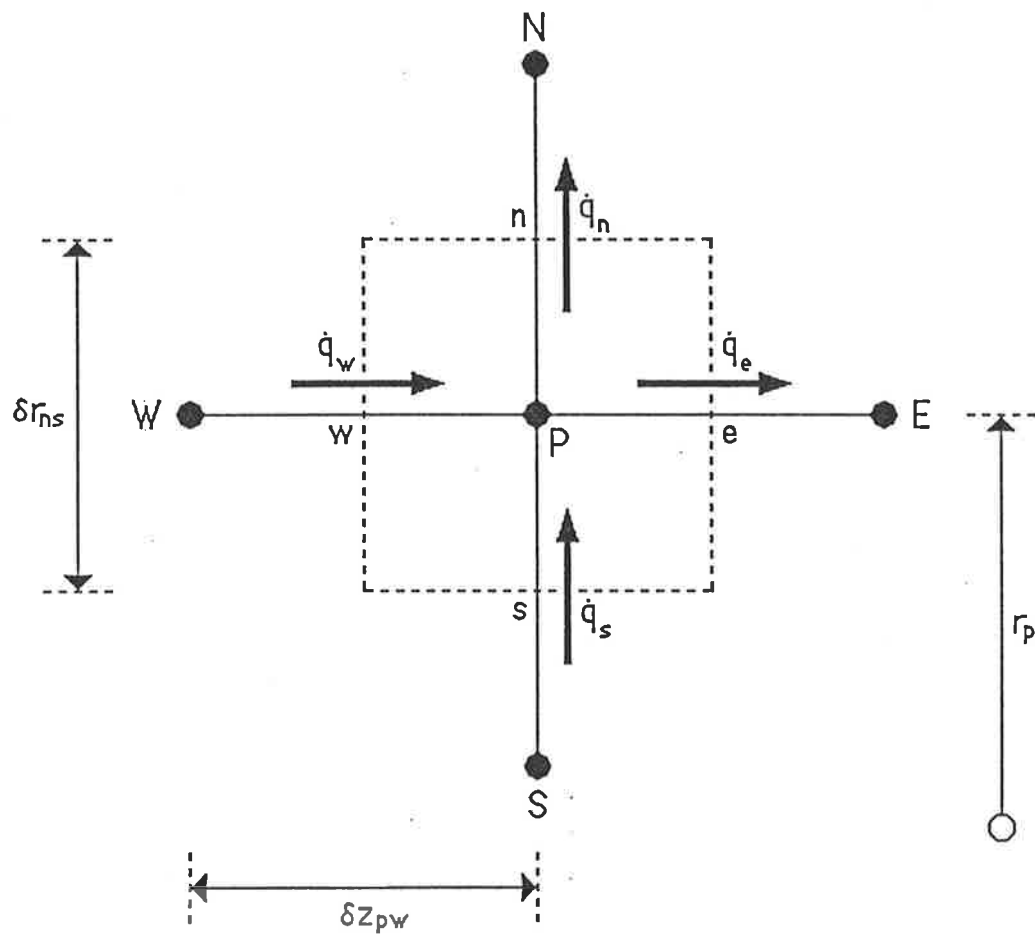


Figure 5.3.2

Control volume and surrounding grid points.

volume equals the net rate of inflow of Φ into the cell by convection and diffusion plus the rate of generation of Φ within the cell. If ϕ is the corresponding intensive property (e.g. U , V , k or ϵ) of Φ then this can be represented by

$$\frac{\Delta(\rho\phi)}{\Delta T} dV + \dot{q}_w - \dot{q}_e + \dot{q}_s - \dot{q}_n + \int_V S_\phi dV = 0 \quad (5.3.1)$$

where T is the time, V is the volume of the computational cell, $\dot{q}$ is the combined convective and diffusive flux of ϕ across the cell faces (n , s , e and w) and S_ϕ is the generation of ϕ per unit volume.

Expressions for the fluxes and the generation term are determined through further application of the macroscopic physical laws. In particular, Spalding (1972) gives an approximation for the total flux of ϕ across the walls of the control volume as a function of ϕ the dependent variable, ρ the fluid density, the mean velocity, A the cell face area and Pe the local Peclet number.

To avoid expensive calculation of exponential terms which arise in the $\dot{q} \sim Pe$ relationship, a further approximation is introduced through the use of a piecewise linear fit (Runchal, 1972). A hybrid differencing scheme is also employed depending upon the value of Pe . A central difference scheme is used for low $|Pe|$, and an upwind difference scheme is used for large $|Pe|$. This has the advantage of numerical stability and also offers a compromise between accuracy and economy.

The generation or source term at the main grid point P is expressed by the linear relationship

$$-\int_V S_\phi dV = b\phi_P + c \quad (5.3.2)$$

where b and c are generally functions of ϕ . The components of the linearised source term are also convenient for the proper insertion of various boundary conditions.

The expressions for the combined convective and diffusive flux and the linearised source term are substituted into equation (5.3.1) and the finite difference equations are conveniently simplified into the following form

$$\left(\sum_{n=N,S,E,W} a_n - b \right) \phi_P = \sum_{n=N,S,E,W} a_n \phi_n + c \quad (5.3.3)$$

where

$$a_W = \rho U_w A_w f_w, \quad a_S = \rho V_s A_s f_s, \quad etc... \quad (5.3.4)$$

and f represents the linear piecewise fit of exponential terms mentioned previously. The subscripts N, S, E and W and n, s, e and w refer to the surrounding grid points and the faces of the control volume, respectively.

5.4 Numerical Solution

The set of simultaneous algebraic equations for the nonhydrodynamic variables (k and ϵ) and the hydrodynamic variables (U, V and p) were solved using the line-by-line method in the Tri-Diagonal Matrix Algorithm (TDMA). However, the SIMPLE algorithm by Patankar and Spalding (1972) must be employed together with the TDMA to solve for the hydrodynamic variables. This is because the pressure p does not appear as the dominant variable in any of the equations. Consequently, a special procedure embodied within the SIMPLE algorithm is used to obtain the pressure. This involves solving the momentum equations from an initial estimate of the pressure distribution, and then correcting the pressure field to enable the newly estimated velocity field to satisfy the continuity equation. The details of this procedure are described by Patankar and Spalding (1972).

Convergence was declared when the sum of the normalized residual sources of each equation were all less than 0.005. That is

$$\sum_{\text{flow domain}} |R_\phi| < 0.005 R_{\phi, \text{ref}} \quad (5.4.1)$$

where the residual source is defined as

$$R_\phi = \left(\sum_{n=N,S,E,W} a_n - b \right) - \sum_{n=N,S,E,W} a_n \phi_n - c \quad (5.4.2)$$

The Cyber 173 computer at the University of Adelaide was used for the numerical computations. For $Re = 10000$, 13.8 minutes CPU time and 346 iterations were required for convergence.

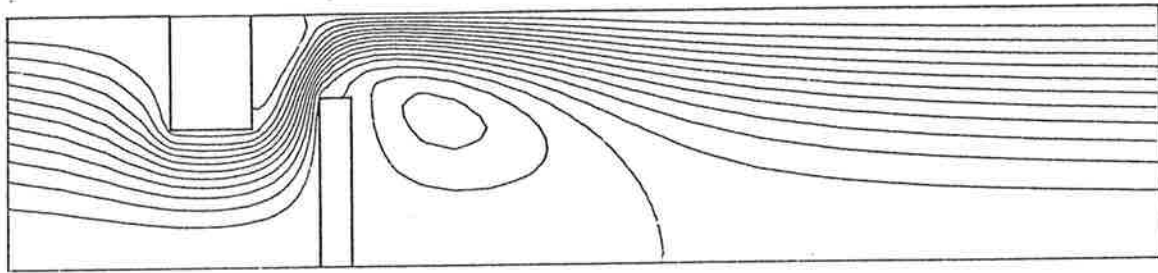
5.5 Discussion of Results

Converged numerical solutions for steady turbulent blood flow through the fully open disk valve were obtained for $Re = 600, 1025, 1500, 2000, 2947, 4200, 6054$ and 10000 (corresponding to steady flow rates of 2.633, 4.499, 6.583, 8.778, 12.934, 18.433, 26.570 and 43.889 *litres min⁻¹*, respectively). Several important aspects of the computed results are summarised in Table 5.5.1.

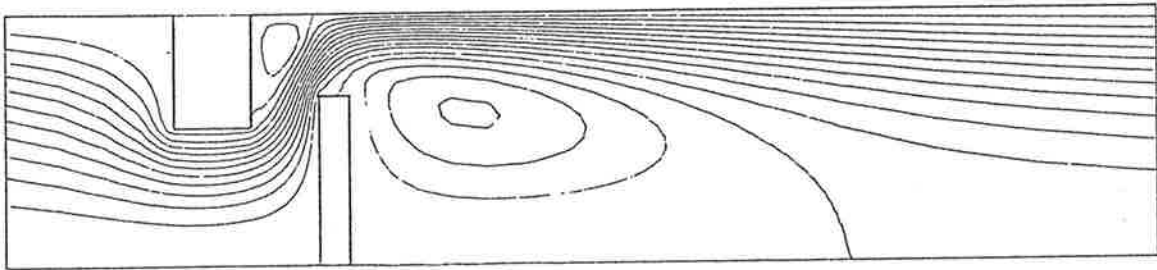
Stream function contours are presented in Figure 5.5.1. Two separated flow regions are located downstream and attached to the sewing ring structure and the disk occluder. Both of these regions have been observed in experiments. (Figliola, 1976; Figliola and Mueller, 1977, 1981). The numerical solutions did not reveal any separated regions upstream of the sewing ring nor along the aortic wall. Experimental measurements of wall shear stress at high Reynolds numbers (Herkes and Lloyd, 1981) have indicated the presence of a very thin recirculation zone lying

Reynolds number	600	1025	1500	2000	2947	4200	6054	10000
Inflow velocity, U_0 (cm sec ⁻¹)	7.892	13.482	19.729	26.306	38.762	55.242	79.628	131.530
Steady flow rate (cm ³ sec ⁻¹)	43.890	74.978	109.720	146.297	215.569	307.220	442.839	731.483
Steady flow rate (lt min ⁻¹)	2.633	4.499	6.583	8.778	12.934	18.433	26.570	43.889
Reattachment length, L/D	0.641	0.809	0.935	1.023	1.141	1.224	1.291	1.351
STREAM FUNCTION, ψ								
Maximum (i, j)	0.0077 (18,22)	0.0137 (18,21)	0.0206 (18,21)	0.0280 (18,21)	0.0421 (18,21)	0.0607 (18,21)	0.0884 (18,21)	0.1474 (18,21)
Minimum (i, j)	-0.0014 (27,14)	-0.0030 (27,15)	-0.0046 (27,15)	-0.0062 (27,15)	-0.0095 (28,15)	-0.0137 (28,15)	-0.0200 (28,15)	-0.0332 (28,15)
AXIAL VELOCITY (U/U ₀)								
Maximum (i, j)	4.42 (13.5,13)	4.61 (13.5,13)	4.69 (13.5,13)	4.74 (13.5,13)	4.77 (13.5,13)	4.80 (13.5,13)	4.81 (13.5,13)	4.80 (13.5,13)
Upstream occluder corner (i, j)	2.66 (20.5,17)	2.76 (20.5,17)	2.83 (20.5,17)	2.88 (20.5,17)	2.94 (20.5,17)	2.95 (20.5,17)	2.96 (20.5,17)	3.00 (20.5,17)
Maximum in annular region (i, j)	2.71 (22.5,22)	2.80 (22.5,22)	2.86 (22.5,23)	2.91 (22.5,23)	2.97 (22.5,23)	2.99 (22.5,23)	3.03 (22.5,23)	3.05 (22.5,23)
Large recirculation zone (i, j)	-1.02 (26.5,02)	-1.20 (27.5,02)	-1.27 (27.5,02)	-1.30 (27.5,02)	-1.33 (28.5,02)	-1.35 (28.5,02)	-1.37 (28.5,02)	-1.38 (28.5,02)
Small recirculation zone (i, j)	-1.02 (17.5,23)	-1.39 (17.5,23)	-1.64 (17.5,23)	-1.79 (17.5,23)	-1.95 (17.5,23)	-2.04 (17.5,23)	-2.11 (17.5,23)	-2.18 (17.5,23)
RADIAL VELOCITY (V/U ₀)								
Maximum (i, j)	5.50 (20,16.5)	5.67 (20,16.5)	5.78 (20,16.5)	5.85 (20,16.5)	5.93 (20,16.5)	5.99 (20,16.5)	6.03 (20,16.5)	6.08 (20,16.5)
Minimum (i, j)	-0.77 (15,20.5)	-1.06 (14,20.5)	-1.38 (14,21.5)	-1.60 (14,21.5)	-1.85 (14,20.5)	-2.03 (14,20.5)	-2.16 (14,20.5)	-2.28 (14,20.5)
TURBULENT SHEAR STRESS, σ_{rz} (N m ⁻²)								
Maximum (i, j)	4.9 (18,18)	13.5 (18,18)	27.6 (18,18)	47.5 (18,18)	95.2 (18,18)	184.7 (18,18)	380.7 (17,17)	1011.3 (17,17)
Minimum (i, j)	-3.9 (21,22)	-11.3 (21,22)	-22.4 (21,22)	-35.9 (21,22)	-68.2 (21,22)	-122.5 (21,22)	-226.2 (21,22)	-532.8 (21,22)
Upstream sewing ring corner (i, j)	-1.0 (09,14)	-1.7 (09,14)	-2.5 (09,14)	-3.3 (09,14)	-5.0 (09,14)	-18.2 (09,14)	-33.8 (09,14)	-79.7 (09,14)
Downstream sewing ring corner (i, j)	4.5 (14,14)	8.0 (14,14)	11.8 (14,14)	15.8 (14,14)	23.4 (14,14)	33.2 (14,14)	47.8 (14,14)	78.5 (14,14)
Upstream occluder corner (i, j)	3.7 (21,17)	6.5 (21,17)	9.6 (21,17)	15.2 (21,17)	28.7 (21,17)	52.7 (21,17)	97.6 (21,17)	229.7 (21,17)
Aortic wall (i, j)	2.9 (25,24)	5.5 (25,24)	8.5 (25,24)	11.8 (26,24)	18.4 (25,24)	33.9 (26,24)	63.9 (26,24)	152.6 (26,24)

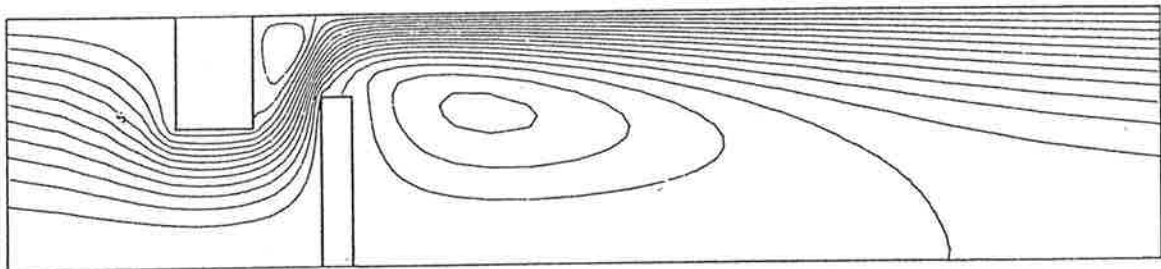
Table 5.5.1
Summary of Model II numerical solutions



$Re = 600$



$Re = 2000$



$Re = 4200$

Figure 5.5.1

Computed mean streamlines for $Re = 600, 2000$ and 4200 from $z/D = -0.503$ ($\lambda = 4.5$) to $z/D = 1.863$ ($\lambda = 36.5$).

along the aortic wall. Figliola and Mueller (1977) have also observed and measured this region using air apparatus in an aortic-shaped chamber for both a model disk and a caged-disk valve. Previous numerical studies based on a mathematical model of laminar fluid flow have also predicted the presence of a recirculation zone immediately upstream of the valve sewing ring and another along the aortic wall (Underwood and Mueller, 1977; Model I in Chapter 4). The failure of Model II to predict this zone is likely due either to the relatively large grid spacing when compared with the observed width of the zone, the use of wall functions which ignore the laminar-turbulent transition zone found near solid boundaries, and, or the numerical diffusion of momentum across the streamlines towards the aortic wall. An improvement to the model could therefore be made by refining the grid size near the boundary and by mathematically modelling the laminar-turbulent transition zone (Jones *et al*, 1973) which is currently neglected for economical reasons. Third order upwind differencing of the convection terms would help to minimise numerical diffusion. It is interesting to note that experimental measurements of velocity profiles in the vicinity of a Kay-Shiley disk prosthesis in an aortic-shaped chamber with a blood analog fluid have revealed only the two larger separated zones (Figliola and Mueller, 1981).

In Figure 5.5.2 a comparison is made between the turbulent numerical solutions and experimental measurements (Figliola, 1976) of L/D , the nondimensional reattachment length of the recirculating zone attached to the occluder. L/D values are given in Table 5.5.1. L/D was observed to increase with Reynolds number for both the turbulent numerical solutions and the experimental measurements. The turbulent flow solutions for L/D remained higher than the experimentally observed values for all Re and agreed best at $Re \approx 1000$. Discrepancies between the numeri-

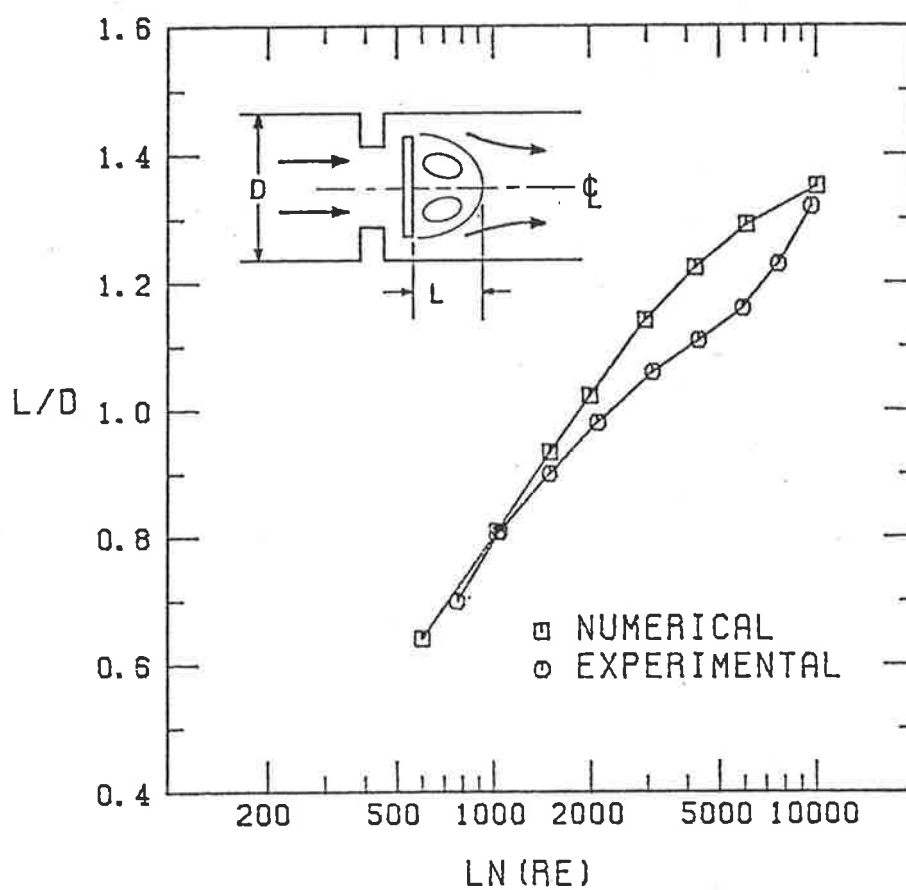


Figure 5.5.2

Comparison of turbulent numerical solutions for L/D with experimental measurements (Figliola, 1976).

cal and observed values of L/D may be due to small geometrical differences between the valves used in the different studies. The separation point for this recirculation zone was attached along the top of the occluder and towards the upstream corner for all Re in agreement with experimental observations (Figliola and Mueller, 1981).

A smaller annular recirculation zone was predicted immediately downstream of the sewing ring. The size and shape of this region remained essentially unchanged for all Re . This zone had a separation point along the downstream face of the valve sewing ring and towards the centreline. The reattachment point along the aortic wall was computed at $z/D = 0.112$ for $Re = 600$, $z/D = 0.116$ for $Re = 1025$ and $z/D = 0.118$ for $Re = 1500$ to 10000 .

High velocities diagonal to the computing mesh are known to introduce a numerical, or artificial diffusion into that region of the flow (Roache, 1976). Such conditions are present in the region between the sewing ring and the disk occluder. Here, an artificial diffusion of momentum will occur across the streamlines and into the small recirculation zone attached to the sewing ring. This results in an under estimate of local velocity profiles tangent to the streamlines which in turn reduces the size of the recirculating zone. The separating streamline is effectively moved towards the concave corner joining the sewing ring and the aortic wall. It is therefore suggested that the actual size of this zone will be larger than that calculated with this model.

Figure 5.5.3 shows the mean axial velocity profiles at various cross sections in the flow field. The largest axial velocities computed in the entire flow domain occurred near the downstream corner of the sewing ring for all Re . Between $Re = 600$ and 10000 the maximum axial velocity increased from 4.4 to 4.8 times the inflow velocity (Table 5.5.1). No experimental measurements of flow velocity in this region

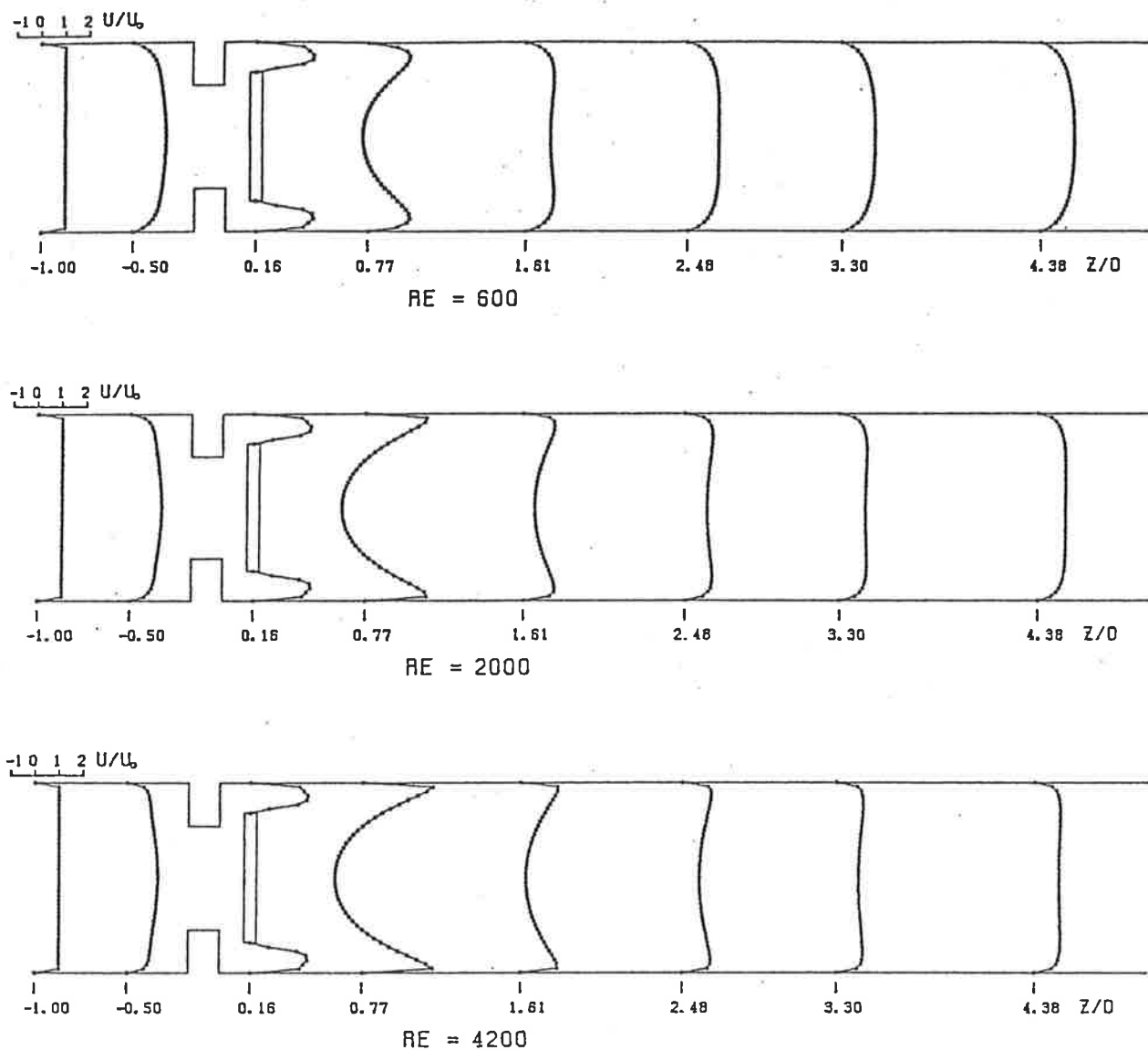


Figure 5.5.3

Computed nondimensional axial velocity profiles for $Re = 600$, 2000 and 4200 from $z/D = -1.0$ to $z/D = 5.0$

are available in the literature for comparison. The maximum axial velocities in the annular region between the occluder and aortic wall were located close to the aortic wall. Between $Re = 600$ and 10000 the peak axial velocities increased from 2.7 to 3.1 times the inflow velocity (Table 5.5.1). For $Re = 6054$ the peak velocity corresponds to 241 cm sec^{-1} . By comparison, axial velocities were 1 to 2% less in magnitude near the upstream corner of the occluder (Table 5.5.1). Figliola and Mueller (1981) measured axial velocities in the vicinity of several prosthetic heart valves in an aortic-shaped chamber. The annular region surrounding the occluder of a Kay-Shiley caged-disk valve contained velocities 2 to 3 times the average mass flow velocity (equivalent to the Model II inflow velocity). At a steady flow rate of $23.4 \text{ litres min}^{-1}$ the peak axial velocity measured was about 3 times the average velocity. Despite the geometrical differences, the numerical velocity predictions of Model II are in good agreement with experimentally measured values.

The presence and intensity of reverse flow downstream of the disk occluder is evident in Figure 5.5.3. Distortion of the axial velocity profiles occurred mainly through the presence of the large separated flow region attached to the disk valve. The velocity profile defects decreased in magnitude downstream of the occluder. These defects had completely vanished by $z/D = 5.0$ for $Re = 600$ to 2947 , but remained present at higher Reynolds numbers. Figliola and Mueller (1981) have observed very similar velocity profile defects and behaviour in the vicinity of a caged-disk valve. The maximum reverse flow velocities in the large separated flow region increased from 1.0 to 1.4 times the inflow velocity over the range of flow rates examined (Table 5.5.1). The peak reverse flow region was located between $z/D = 0.406$ for $Re = 600$ and $z/D = 0.561$ for $Re = 10000$. The maximum computed reverse flow velocities agree very well with experimental measurements

made by Figliola and Mueller (1981). In their study, the negative flow velocities distal to the occluder were 1.0 to 1.5 times the average mass flow velocity. Figliola and Mueller (1981) also suggested that this reverse flow may provide a washing action to help prevent thrombotic deposition. The maximum reverse flow velocities in the annular recirculation zone were located at $z/D = 0.060$ for all Re . They increased in magnitude from 1.0 to 2.2 times the inflow velocity over the range of flow rates examined (Table 5.5.1). These values are larger than those calculated in the main separation zone, and result directly from the close proximity of the smaller zone to the very high velocities found in the vicinity of the valve sewing ring and the occluder edge. Unfortunately, no quantitative experimental data is available for flow velocities in this recirculation zone.

Variation of the wall shear stress profile upstream and downstream of the valve sewing ring is shown in Figure 5.5.4 for $Re = 6054$. Upstream of the sewing ring the wall shear stress remained below 10 N m^{-2} . Downstream of the sewing ring the wall shear stress briefly attained a negative peak of -35.6 N m^{-2} before increasing rapidly to 63.9 N m^{-2} at a point where the flow impinged upon the aortic wall. Downstream of this point, the wall shear stress quickly returned again to a level below 10 N m^{-2} . The negative peak corresponds to a location within the annular recirculation zone. For $Re = 10000$, the maximum aortic wall shear stress computed was 152.6 N m^{-2} at $z/D = 0.344$. At this very high Reynolds number damage and erosion of nearby endothelial cells would occur. The maximum computed wall shear stress values for other Reynolds numbers are presented in Table 5.5.1. At $Re = 6054$, the elevated shear levels would damage the endothelial lining at the point where the flow impinges. Nearby platelets would also be damaged.

For the same valve geometry and Reynolds number, Herkes and Lloyd (1981)

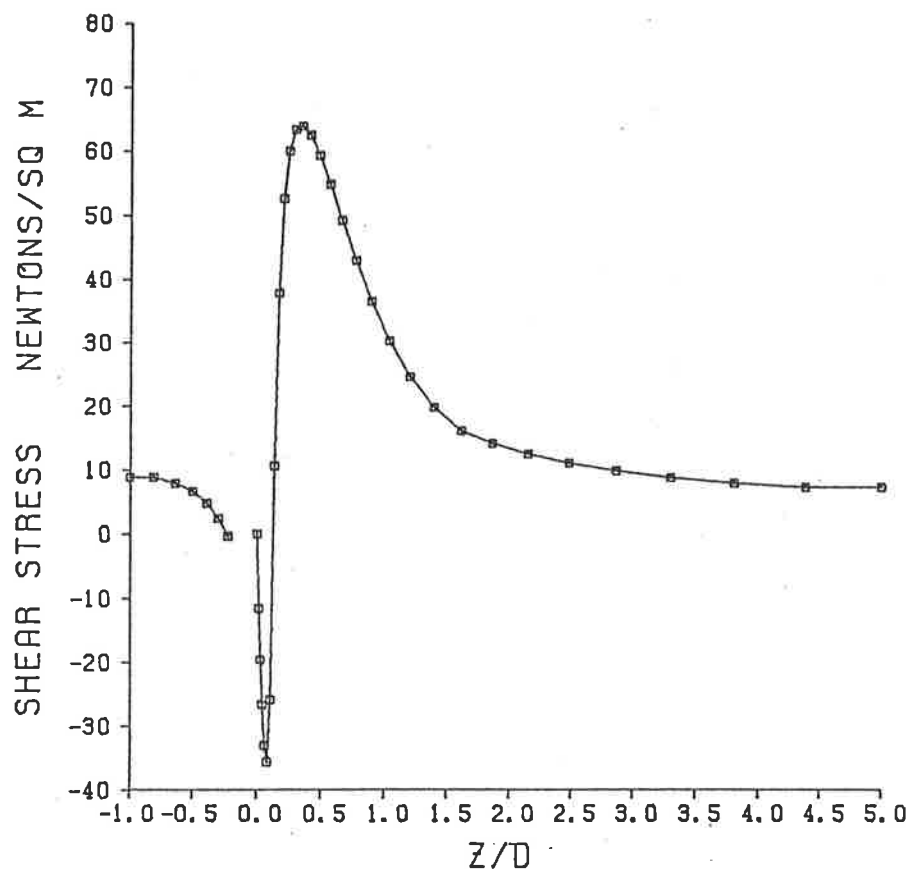


Figure 5.5.4

Computed aortic wall shear stress distribution ($N\ m^{-2}$) for $Re = 6054$
from $z/D = -1.0$ to $z/D = 5.0$

experimentally measured a similar aortic wall shear stress profile using an electrochemical technique. However, they did not report the sharp negative peak computed by Model II immediately downstream of the sewing ring. This may have been due to limited probe resolution. The peak wall shear stress measured was 48.3 N m^{-2} , a value approximately 25 % less than that computed by Model II. For a Kay-Shiley disk valve in an aortic-shaped chamber, Figliola and Mueller (1981) also determined a similar wall shear stress profile. This was achieved by assuming a linear velocity profile between the wall and the mean axial velocity measured nearest to the wall at that axial location. Laser Doppler anemometry was used to perform the velocity measurements. Using this approach, a maximum wall shear stress of 254.8 N m^{-2} was estimated at a flow rate of $23.4 \text{ litres min}^{-1}$. This occurred just downstream of the sinus zone reattachment point. This value is considerably higher than stresses reported here. Furthermore, Yoganathan *et al* (1978) has experimentally measured wall shear stresses of the order of 300 N m^{-2} at comparable flow rates with a similar caged-disk valve (Starr-Edwards 6520-1M). Typically, the Model II numerical solutions for peak wall shear stress are 70 to 80 % lower than the values determined by Figliola and Mueller (1981) and Yoganathan *et al* (1978). In Section 3.3, it was mentioned that the standard $k-\epsilon$ turbulence model tends to underpredict Reynolds stress levels. Refinements are available (Speziale, 1986), but for general turbulent separating flows there is much scope for research work.

Figure 5.5.5 is a contour map of the turbulent, or Reynolds, shear stresses present at $Re = 6054$ for a section of the flow domain. The turbulent shear stresses computed at this physiologically relevant Reynolds number are observed to increase rapidly to a maximum of 380.7 N m^{-2} . This peak shear value occurs in the region between the sewing ring and the occluder just inside the small annular recirculation

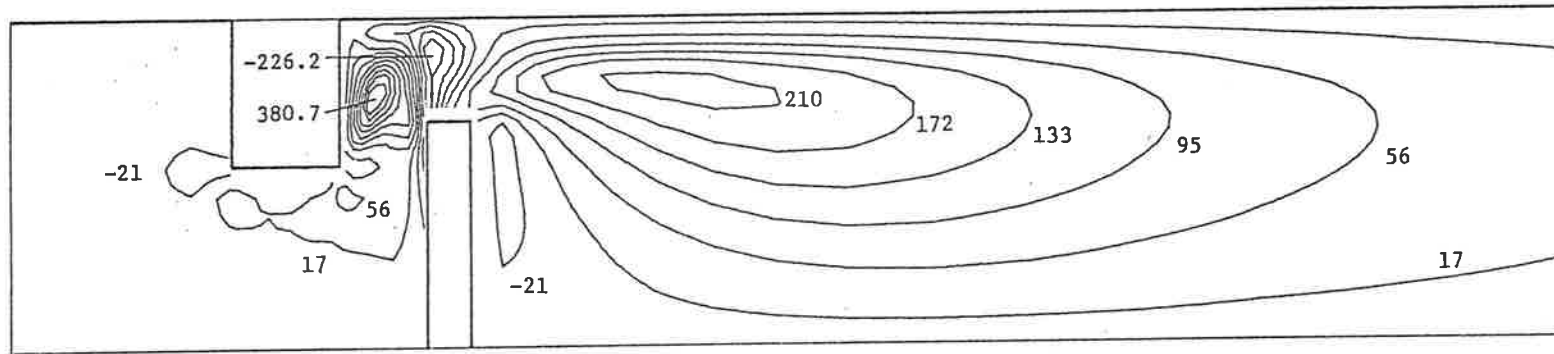


Figure 5.5.5

Computed turbulent shear stress contours ($N\ m^{-2}$) for $Re = 6054$ from $z/D = -0.503$ ($i = 4.5$) to $z/D = 1.863$ ($i = 36.5$).

zone. In the annular flow region between the occluder and the aortic wall a negative peak is observed with a value of -226.2 N m^{-2} . The maximum and minimum values of turbulent shear stress for the other Reynolds numbers are given in Table 5.5.1. Another peak of about 230 N m^{-2} was observed in a large axially elongated zone downstream of the occluder. The location of this zone corresponds closely with the high velocity flow region downstream of the valve. The potential for damage and destruction of red blood cells in flow exists in these three regions.

Turbulent stresses of the same order of magnitude have been determined experimentally in flow. However, experimental studies are currently unable to present such a detailed description of this phenomenon as that given in Figure 5.5.5. Figliola and Mueller (1981) measured a maximum turbulent shear stress of 81.6 N m^{-2} some distance downstream from a Kay-Shiley disk valve in an aortic-shaped chamber. Larger stresses were suspected closer to the valve but could not be measured due to probe blockage. It would be reasonable to expect larger shear values downstream of the Kay-Shiley disk valve if the measurements had been instead performed in a constant diameter chamber with a square-edged disk occluder. The levels of turbulent shear stress downstream of the occluder are particularly sensitive to valve geometry. The lenticular occluder shape and the model sinus in the aortic wall employed by Figliola and Mueller (1981) serve to lessen the levels of turbulent shear stress in flow. Turbulent shear stress values of the order of 200 N m^{-2} computed downstream of the Model II disk occluder are therefore not necessarily excessive given the nature of the Model II valve geometry. Yoganathan *et al* (1979) did not report on any direct measurements of turbulent shear stress in flow. However, he did suggest that the annular region surrounding the occluder could be treated as a type of jet flow. For an unbounded self-preserving jet, where U_{CL} is the mean

centreline velocity of the jet, he estimated the maximum turbulent shear stresses by

$$\sigma_{zr} \approx 0.025 \rho U_{C_L}^2 \quad (5.5.1)$$

For the $Re = 6054$ numerical solutions, the maximum velocity in the annular region was 241 cm sec^{-1} . Hence, the maximum turbulent shear stress that would be predicted by equation (5.5.1) is about 154 N m^{-2} . This value is about 30 % less than the 230 N m^{-2} actually computed in the region downstream of the jet.

Table 5.5.1 also presents computed data for the turbulent shear stress at the upstream and downstream corner of the sewing ring and at the upstream corner of the disk occluder. The complex nature of the shear stress distribution is clearly evident when comparing the relative magnitudes of shear stress at the different locations with flow rates. For example, at $Re = 600$ the shear stress at the downstream sewing ring corner and at the upstream occluder corner are comparable in magnitude, but by $Re = 10000$ the latter is about 3 times larger. A similar comparison can be made for both the upstream and downstream corners of the sewing ring. Damage to platelets is likely near the upstream corner of the occluder for $Re \geq 2000$, near the upstream corner of the sewing ring for $Re \geq 4000$, and near the downstream corner of the sewing ring for $Re \geq 1500$.

Even though the present calculations may not have much direct clinical application, the numerical solutions reported here are within acceptable limits of values observed *in vitro* experimentally, with the exception of the aortic wall shear stresses which are significantly lower than measured levels. This presents a fairly optimistic view for the future numerical analysis of more complex and realistic geometries with

the $k - \epsilon$ turbulence model. It is suggested that only when boundary-fitted grids are used together with the $k - \epsilon$ turbulence model will these computational techniques be employed to advantage in the prosthetic heart valve analysis and design process.

5.6 Conclusions

The $k - \epsilon$ turbulence model is used to obtain quantitative solutions for velocity, wall shear stress and turbulent shear stress distributions near a disk-type prosthetic heart valve at high Reynolds numbers. The maximum mean axial velocities occur near the downstream corner of the sewing ring and are 4.4 to 4.8 times the inflow velocity. Strong reverse flow is identified in the separated regions and the length of the large recirculating zone is numerically computed for high Reynolds numbers. The largest wall shear stresses are predicted in the vicinity of the disk occluder and are of the order of 10 to 10^2 N m^{-2} for $Re \geq 2000$. Damage to the endothelial lining of the aorta is shown to be probable for $Re \geq 6000$. Maximum turbulent shear stresses of 380.7 N m^{-2} are computed for physiological Reynolds numbers. Haemolysis of blood cells at these shearing stress levels is likely. Overall, the numerical solutions of the $k - \epsilon$ turbulence model compare well with experimental observations, with the exception of the computed wall shear stresses which are typically 70 to 80 % lower than measured values. In Chapter 6 the numerical procedure is applied to a realistic boundary-fitted coordinate system of a caged-ball prosthesis in the aortic position.

CHAPTER 6

MODEL III — TURBULENT FLOW THROUGH A CAGED-BALL PROSTHESIS

6.1 Introduction

This model examines steady turbulent axisymmetric flow through a Starr-Edwards caged-ball valve in an aortic-shaped chamber. A boundary-fitted coordinate system is generated for the flow domain in the vicinity of the valve. The coordinate lines follow the left ventricular wall, the sewing ring, an idealized sinus, the aortic wall and the occluder shape. The standard $k - \epsilon$ turbulence model is formulated using a stream function-vorticity approach. The governing partial differential equations are recast into their curvilinear equivalents before being discretised into finite difference equations which are then solved iteratively. The numerical techniques and results reported in this chapter have been published and appear in Appendix E.

6.2 Mathematical Model

The geometry representing a fully open Starr-Edwards 1260-11A caged-ball prosthetic heart valve in an aortic-shaped chamber is shown in Figure 6.2.1. The geometry includes a left ventricular chamber, a sewing ring structure, an idealized axisymmetric sinus, the aortic wall and a ball occluder. The dimensions of the valve are given in Figure 6.2.2. For comparison purposes the dimensions are very close to those employed by Figliola and Mueller (1981) in their experimental measurements. Assumptions made are that blood behaves like a Newtonian fluid in

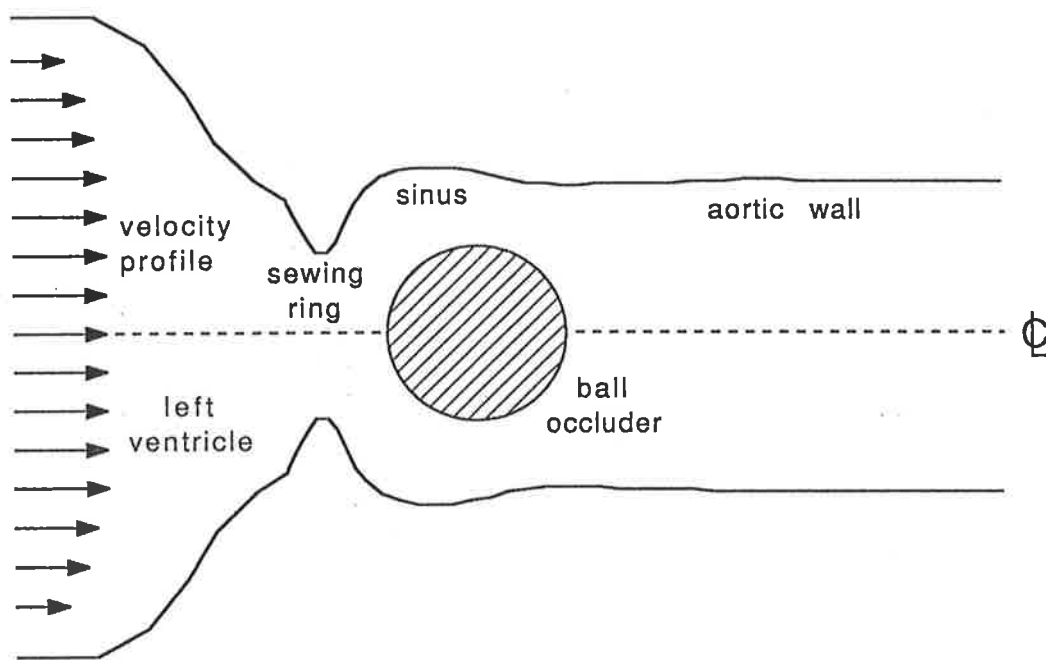


Figure 6.2.1

Geometry and inflow velocity profile for the Model III ball valve.

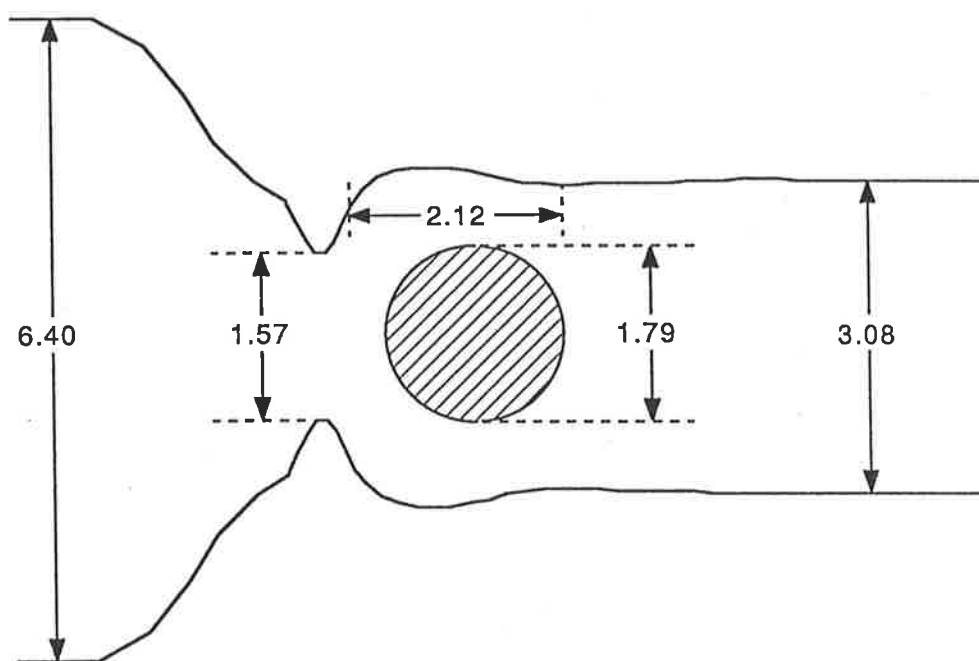


Figure 6.2.2

Dimensions (cm) of the fully open Model III ball valve.

large arteries, the cage containing the occluder has a negligible effect upon the flow, the domain boundaries are smooth and rigid and the flow is axisymmetric. The fluid is given a density of 1.06 gm cm^{-3} and a molecular, or laminar, viscosity of $3.5 \times 10^{-3} \text{ N sec m}^{-2}$ closely matching that of human blood.

The analysis of turbulent flow is difficult and it has become customary to abandon the study of the detailed flow structure and treat the flow as consisting of mean and fluctuating components. Using this approach the governing mass and momentum equations are decomposed through application of time-averaging. This procedure results in additional terms $\overline{\rho u'_i u'_j}$ often referred to as turbulent, or Reynolds, stresses where ρ is the fluid density, u'_i and u'_j are the fluctuating components of velocity in the i and j directions, respectively. Closure of the model equations is achieved through the Boussinesq eddy-viscosity concept which assumes that the turbulent stresses are directly proportional to the mean velocity gradients (Rodi, 1980). In general form this is expressed by

$$\overline{u'_i u'_j} = -\nu_t \left(\frac{\partial U_i}{\partial x_j} + \frac{\partial U_j}{\partial x_i} \right) + \frac{2}{3} k \delta_{ij} \quad (6.2.1)$$

where δ_{ij} is the Kronecker delta, U_i and U_j are the mean components of velocity in the i and j directions, respectively, and ν_t is the turbulent kinematic viscosity defined by

$$\nu_t = C_\mu \frac{k^2}{\epsilon} \quad (6.2.2)$$

where k is the turbulent kinetic energy representing the intensity of the turbulent fluctuations, ϵ is the dissipation rate of turbulent kinetic energy representing the length scale of the energy containing eddies and C_μ is an empirical constant.

The resulting partial differential equations together with the additional terms required for closure are referred to as the $k-\epsilon$ turbulence model. For this model the governing equations are written in a stream function-vorticity ($\psi-\omega$) formulation similar to that employed by Yang and Wang (1983). Thus, we have the following equations,

$$\omega = -\frac{1}{r} \left(\psi_{zz} + \psi_{rr} - \frac{1}{r} \psi_r \right) \quad (6.2.3)$$

$$\frac{1}{r} \left(\psi_r \omega_z - \psi_z \omega_r + \frac{\omega}{r} \psi_z \right) = (\nu_{eff} \omega)_{zz} + (\nu_{eff} \omega)_{rr} + \frac{1}{r} (\nu_{eff} \omega)_r - \frac{1}{r^2} \nu_{eff} \omega \quad (6.2.4)$$

where the effective kinematic viscosity ν_{eff} is defined as

$$\nu_{eff} = \nu + \nu_t \quad (6.2.5)$$

where ν is the molecular kinematic viscosity. The additional equations for the turbulent kinetic energy and its dissipation rate for a steady axisymmetric flow necessary to complete the model are

$$\frac{1}{r} (\psi_r k_z - \psi_z k_r) = \left[\left(\frac{\nu_{eff}}{\sigma_k} k_z \right)_z + \frac{1}{r} \left(r \frac{\nu_{eff}}{\sigma_k} k_r \right)_r \right] + G - C_D \epsilon \quad (6.2.6)$$

$$\frac{1}{r} (\psi_r \epsilon_z - \psi_z \epsilon_r) = \left[\left(\frac{\nu_{eff}}{\sigma_\epsilon} \epsilon_z \right)_z + \frac{1}{r} \left(r \frac{\nu_{eff}}{\sigma_\epsilon} \epsilon_r \right)_r \right] + \frac{\epsilon}{k} (C_1 G - C_2 \epsilon) \quad (6.2.7)$$

The generation of turbulent kinetic energy G from the mean flow is given by

$$G = \nu_t \left\{ 2 \left[U_z^2 + V_r^2 + \left(\frac{V}{r} \right)^2 \right] + (U_r + V_z)^2 \right\} \quad (6.2.8)$$



where U and V , the mean axial and radial velocities, respectively, are defined by

$$U = \frac{1}{r}\psi_r \quad \text{and} \quad V = -\frac{1}{r}\psi_z \quad (6.2.9)$$

C_1 , C_2 , C_D , C_μ , σ_k and σ_ϵ are empirical constants at high Reynolds numbers (Launder and Spalding, 1974) whose values are given by

$$C_1 = 1.44, C_2 = 1.92, C_D = 1.0, C_\mu = 0.09, \sigma_k = 1.0, \sigma_\epsilon = 1.3$$

The governing equations are subject to several boundary conditions. At inflow we assume a fully developed turbulent velocity profile for pipe flow given by

$$U = U_0 \left(1 - \frac{r}{R}\right)^{\frac{1}{7}} \quad (6.2.10)$$

$$V = 0$$

(Schlichting, 1968) where U_0 is the mean axial centreline velocity at inflow, and R the inflow chamber radius. Analytic expressions for the inflow stream function and vorticity are obtained from equation (6.2.10).

The turbulent kinetic energy and its dissipation rate at inflow are approximated by

$$k = 0.03 U^2 \quad (6.2.11)$$

$$\epsilon = \frac{C_D k^{\frac{3}{2}}}{\lambda} \quad (6.2.12)$$

where λ is the macro eddy length scale. The numerical solutions proved insensitive to differing inflow conditions for ψ , ω , k and ϵ with the large blocking effect of the valve dominating the flow conditions in the vicinity of the prosthesis.

At outflow the boundary condition $\Phi_z = 0$ ($\Phi = \psi, \omega, k, \epsilon$) is imposed, and along the centreline the conditions are $\Phi_r = 0$ ($\Phi = k, \epsilon$), $\psi = 0$ and $\omega = 0$. The position of the outflow boundary at a distance of $10 R$ past the ball occluder ensured the outflow conditions had little effect upon the flow upstream. Along solid boundaries the usual no-slip conditions are applied (i.e. $U = 0$ and $V = 0$) and the values of k and ϵ are also set to zero. The usual ‘law of the wall’ conditions are specified on the first grid point away from the wall. That is

$$\frac{U_p}{U^*} = \frac{1}{\kappa} \ln(Ey^+) \quad (6.2.13)$$

$$y^+ = \frac{yU^*}{\nu} \quad (6.2.14)$$

$$k = \frac{(U^*)^2}{\sqrt{C_\mu}} \quad (6.2.15)$$

$$\epsilon = \frac{(U^*)^3}{\kappa y} \quad (6.2.16)$$

$$\omega = \frac{U^*}{\kappa y} \quad (6.2.17)$$

where U_p is the velocity parallel to the wall, U^* is the friction velocity, κ is von Karman’s constant ($\kappa = 0.4187$), E is van Driest’s constant ($E = 9.793$), y^+ is the local Reynolds number and y is the grid point distance from the wall.

The velocity parallel to the wall at the first grid point away from the wall is computed along with the grid point distance from the boundary. An initial estimate for the friction velocity is made by assuming $U^* \approx |U_p|$. Based upon this estimate, y^+ is then evaluated. If $y^+ \leq 0.15$, the values of k and ϵ are equated to zero and the value of ω calculated from equation (6.2.4) is used. This approach proved effective in controlling the stability of the numerical scheme during the early iterations, especially in the vicinity of separation and reattachment points where y^+ would

occasionally assume very low values. For the vast majority of grid points adjacent to the boundary U^* is determined through a simple iterative process. If it is found that $y^+ > 11.63$, that is turbulence effects dominate, then k , ϵ and ω are computed from the equations above. Otherwise, if $y^+ \leq 11.63$ the flow is assumed to be purely laminar and the friction velocity is given by $U^* = y^+$ before k , ϵ and ω are computed. The sign of the vorticity computed above is assigned depending upon the sign of the corresponding U_p .

All the governing partial differential equations are recast into curvilinear coordinates by substituting derivatives in the (z, r) physical plane by their equivalent expressions in the (ξ, η) transformed plane. Such derivative transformations are given in Appendix B.

Equations (6.2.3) and (6.2.4) are transformed into (ξ, η) coordinates to obtain

$$\omega = -\frac{1}{r} \left[\frac{1}{J^2} (\alpha \psi_{\xi\xi} - 2\beta \psi_{\xi\eta} + \gamma \psi_{\eta\eta} + \sigma \psi_{\eta} + \tau \psi_{\xi}) - \frac{1}{Jr} (z_{\xi} \psi_{\eta} - z_{\eta} \psi_{\xi}) \right] \quad (6.2.18)$$

and

$$\begin{aligned} & \frac{1}{Jr} \left[\psi_{\eta} \omega_{\xi} - \psi_{\xi} \omega_{\eta} + \frac{\omega}{r} (r_{\eta} \psi_{\xi} - r_{\xi} \psi_{\eta}) \right] \\ &= \frac{1}{J^2} \left\{ \alpha (\nu_{eff} \omega)_{\xi\xi} - 2\beta (\nu_{eff} \omega)_{\xi\eta} + \gamma (\nu_{eff} \omega)_{\eta\eta} + \sigma (\nu_{eff} \omega)_{\eta} + \tau (\nu_{eff} \omega)_{\xi} \right\} \\ & \quad + \frac{1}{Jr} \left\{ z_{\xi} (\nu_{eff} \omega)_{\eta} - z_{\eta} (\nu_{eff} \omega)_{\xi} \right\} - \frac{\nu_{eff} \omega}{r^2} \end{aligned} \quad (6.2.19)$$

Expressions for α , β , γ , σ , τ and J , the Jacobian, are given in Appendix B.

The k and ϵ equations, (6.2.6) and (6.2.7), respectively, are expanded before they are rewritten using (ξ, η) coordinates. In the transformed plane they become

$$\begin{aligned}
& \frac{1}{Jr}(\psi_\eta k_\xi - \psi_\xi k_\eta) \\
= & \left(\frac{\nu_{eff}}{\sigma_k} \right) \left[\frac{1}{J^2}(\alpha k_{\xi\xi} - 2\beta k_{\xi\eta} + \gamma k_{\eta\eta} + \sigma k_\eta + \tau k_\xi) + \frac{1}{Jr}(z_\xi k_\eta - z_\eta k_\xi) \right] \\
& + \frac{1}{J^2}(r_\eta k_\xi - r_\xi k_\eta) \left\{ r_\eta \left(\frac{\nu_{eff}}{\sigma_k} \right)_\xi - r_\xi \left(\frac{\nu_{eff}}{\sigma_k} \right)_\eta \right\} \\
& + \frac{1}{J^2}(z_\xi k_\eta - z_\eta k_\xi) \left\{ z_\xi \left(\frac{\nu_{eff}}{\sigma_k} \right)_\eta - z_\eta \left(\frac{\nu_{eff}}{\sigma_k} \right)_\xi \right\} \\
& + G - C_D \epsilon
\end{aligned} \tag{6.2.20}$$

and

$$\begin{aligned}
& \frac{1}{Jr}(\psi_\eta \epsilon_\xi - \psi_\xi \epsilon_\eta) \\
= & \left(\frac{\nu_{eff}}{\sigma_\epsilon} \right) \left[\frac{1}{J^2}(\alpha \epsilon_{\xi\xi} - 2\beta \epsilon_{\xi\eta} + \gamma \epsilon_{\eta\eta} + \sigma \epsilon_\eta + \tau \epsilon_\xi) + \frac{1}{Jr}(z_\xi \epsilon_\eta - z_\eta \epsilon_\xi) \right] \\
& + \frac{1}{J^2}(r_\eta \epsilon_\xi - r_\xi \epsilon_\eta) \left\{ r_\eta \left(\frac{\nu_{eff}}{\sigma_\epsilon} \right)_\xi - r_\xi \left(\frac{\nu_{eff}}{\sigma_\epsilon} \right)_\eta \right\} \\
& + \frac{1}{J^2}(z_\xi \epsilon_\eta - z_\eta \epsilon_\xi) \left\{ z_\xi \left(\frac{\nu_{eff}}{\sigma_\epsilon} \right)_\eta - z_\eta \left(\frac{\nu_{eff}}{\sigma_\epsilon} \right)_\xi \right\} \\
& + \frac{\epsilon}{k}(C_1 G - C_2 \epsilon)
\end{aligned} \tag{6.2.21}$$

The generation of turbulent kinetic energy G term given by equation (6.2.8) is also transformed to (ξ, η) coordinates.

Finally, the Reynolds number for Model III is defined by

$$Re = \frac{D U_0}{\nu} \tag{6.2.22}$$

where D is the inflow chamber diameter and U_0 is the mean axial centreline velocity at inflow.

6.3 Coordinate System Generation

In Chapters 4 and 5, the geometric configurations of the caged-disk models were simple and the boundary walls were coincident with the Cartesian coordinate system. In this section, details are presented on the approach used to generate an orthogonal boundary-fitted coordinate system for a fully open Starr-Edwards caged-ball prosthesis.

The method requires that the shape of physical domain be known in advance and not be determined as part of the solution. Initially, the upper surface of the boundary shape shown in Figure 6.2.1 was constructed from a combination of trigonometric functions for the left ventricular chamber, sinus of Valsalva and aortic wall, and cubic splines for the sewing ring. A hemisphere was used for the ball occluder (as only half the valve geometry is necessary due to the symmetry assumption) but this was altered slightly by “smoothing” the fore and aft sections of the hemisphere to reduce singularity effects which became evident during the computation process. The boundary functions for the upper part of the physical domain are:

Inflow chamber, $0.0 \leq z < 4.0$:

$$r(z) = 3.2 \quad (6.3.1)$$

Left ventricular section, $4.0 \leq z < 5.8$:

$$r(z) = 0.95 \cos \left[\frac{\pi}{2}(z - 4) \right] + 2.25 \quad (6.3.2)$$

Sewing ring, $5.8 \leq z < 6.7$:

$$r_i(z) = \sum_{j=1}^4 c_{ij}(z - z_i)^{j-1}, \quad z_i \leq z < z_{i+1}, \quad i = 1, \dots, 7 \quad (6.3.3)$$

i	z_i	c_{i1}	c_{i2}	c_{i3}	c_{i4}
1	5.8000	1.345	-0.41715	0.00000	-43.28503
2	5.9000	1.260	-1.71570	-12.98551	61.42514
3	6.0000	1.020	-2.47005	5.44203	5.41959
4	6.0800	0.860	-1.49527	6.74274	0.39943
5	6.2225	0.785	0.45075	6.91349	0.89021
6	6.3600	0.980	2.40245	7.28071	-42.32405
7	6.4800	1.300	2.32142	-7.95595	12.05447
8	6.7000	1.554	0.57111	0.00000	0.22000

Table 6.3.1

Cubic spline coefficients for the sewing ring structure

where the values of z_i and coefficients c_{ij} are given in Table 6.3.1.

Sinus of Valsalva, $6.7 \leq z < 10.0$:

$$r(z) = 2(10 - z)^{1.5} \sin \left[\frac{3\pi}{4}(z - 6) - \frac{\pi}{2} \right] + 1.54 \quad (6.3.4)$$

Aortic wall, $10.0 \leq z \leq 25.0$:

$$r(z) = 1.54 \quad (6.3.5)$$

The approach adopted for generating a boundary-fitted grid is that described by Mobley and Stewart (1980). The goal is to evaluate grid point values in the physical plane (z, r) subject to the requirement that the grid lines in the transformed plane (ξ, η) form a conjugate harmonic pair, thereby causing the $\xi - \eta$ contours to be orthogonal. That is, they must satisfy the Cauchy-Reimann equations

$$\xi_z = \eta_r \quad (6.3.6a)$$

$$\xi_r = -\eta_z \quad (6.3.6b)$$

In order to control the separation of grid lines in the physical plane, ξ and η are defined as monotonically increasing functions of two variables χ and ζ , respectively. That is,

$$\xi = f(\chi) \quad (6.3.7a)$$

$$\eta = g(\zeta) \quad (6.3.7b)$$

Laplace-type equations are formulated for each of the spatial coordinates in the (z, r) physical plane. In general form, the Laplace-type equations (Mobley and Stewart, 1980) are

$$\mathcal{L}(z) = 0 \quad (6.3.8a)$$

$$\mathcal{L}(r) = 0 \quad (6.3.8b)$$

where the operator $\mathcal{L}$ is defined as

$$\mathcal{L} = \frac{\partial^2}{\partial \chi^2} + \delta^2 \frac{\partial^2}{\partial \zeta^2} - F \frac{\partial}{\partial \chi} - \delta^2 G \frac{\partial}{\partial \zeta} \quad (6.3.9)$$

and

$$\delta(\chi, \zeta) \equiv \frac{f'(\chi)}{g'(\zeta)}, \quad F(\chi) \equiv \frac{f'(\chi)}{f''(\chi)}, \quad G(\zeta) \equiv \frac{g'(\zeta)}{g''(\zeta)} \quad (6.3.10)$$

where $f' \equiv df/d\chi$, $f'' \equiv d^2f/d\chi^2$, etc., and $f' \neq 0$, $g' \neq 0$.

To complete orthogonality the solutions for both z and r are coupled at the domain boundaries by application of the Cauchy-Reimann conditions. A weak constraint is applied so that the final distribution of boundary grid points is not predetermined but is computed through the numerical mapping procedure. For example, in order to solve equation (6.3.8a), z must also satisfy the Neumann condition

$$z_\zeta = -\frac{g'}{f'} r_\chi \quad (6.3.11)$$

along the top and bottom boundaries, whilst Dirichlet conditions are specified on the other domain boundaries. Similar conditions are applied to the domain boundaries when solving equation (6.3.8b), except that Dirichlet conditions are applied to the top boundary using equations (6.3.1) to (6.3.5), to the bottom boundary using the patched hemisphere equation, and a Neumann condition on the remaining walls.

A finite difference method employing central space derivative approximations was developed for solving equations (6.3.8a) and (6.3.8b) but the resultant grid was noticeably non-orthogonal, particularly near boundary segments with high surface gradients (Figure 6.3.1). Conventional error analysis revealed that the coefficients of higher ordered terms in the vicinity of the steep boundary segments were assuming extremely large values. This problem was partially resolved by switching to either backward or forward differencing (depending upon the orientation of the boundary segment) when computing in these regions.

A further improvement was obtained by implementing a simple procedure described by Davies (1981) where orthogonal trajectories are used for the orthogonalisation of a discrete non-orthogonal coordinate system. By using the moderately non-orthogonal coordinate system computed with the Mobley and Stewart (1980) method as an initial value this approach proved successful and easy to invoke.

An iterative scheme is used for the numerical solution of the grid equations. An adequate initial guess is obtained by simple linear interpolation of the boundary values in the radial direction, and equidistant grid lines in the axial direction (Figure 6.3.2). Figures 6.3.3 to 6.3.8 indicate the state of the solution at various iteration steps. The “overlapping” of grid lines predominant during early iterations stems largely from the rather poor initial guess and the accompanying complexity of the geometric configuration. Several other initial estimates for z and r involving

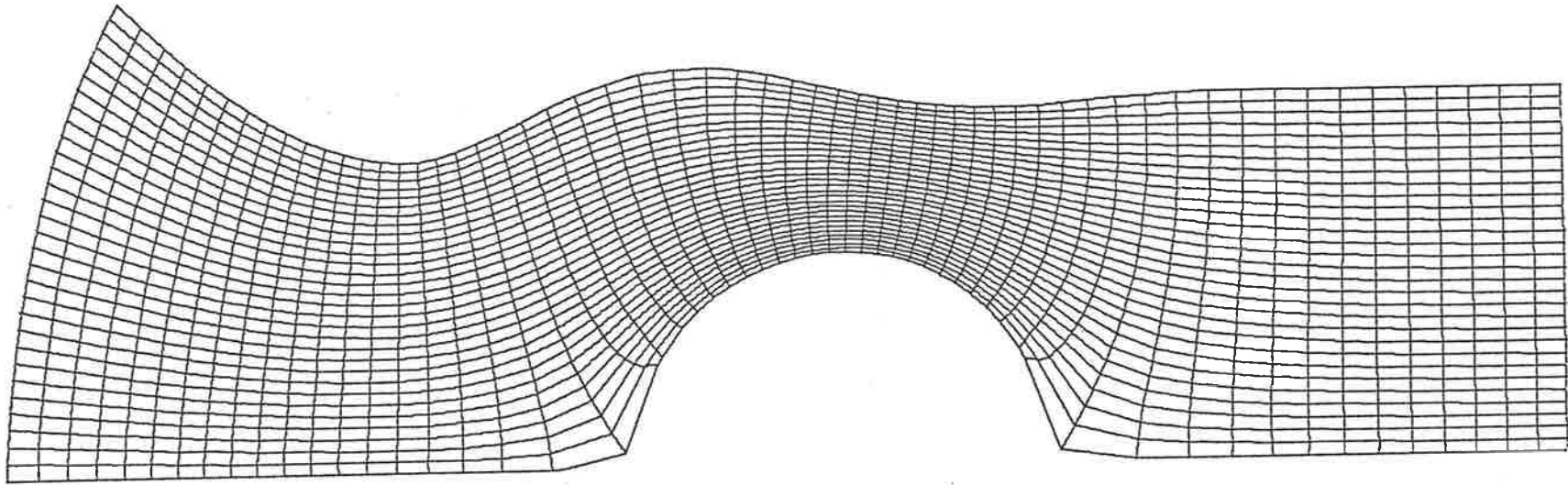


Figure 6.3.1

Section of the computational grid (without sewing ring) illustrating the marked non-orthogonality of grid lines near boundary surfaces when employing finite differences with central space derivative approximations.

compression and interpolation of grid lines were attempted, but with little success. After 300 iterations (Figure 6.3.6) the (ξ, η) coordinate lines were no longer crossing. Convergence, albeit non-orthogonal, was declared after 500 iterations (Figure 6.3.8). These values of z and r were then employed as initial values for orthogonalisation using the method of Davies (1981). The corrected grid system is shown in (Figure 6.3.9). Excess grid lines in the radial direction were removed and some additional lines were eventually inserted in the axial direction (Figure 6.3.10). The final computational grid (120×16) is shown in Figure 6.3.11.

The successful generation of a boundary-fitted grid was prolonged for some time due to difficulty experienced in producing an orthogonal coordinate system. The wisdom of hindsight would dictate that the orthogonality constraint imposed to simplify the transformed governing equations, and hence coding, was far too demanding considering the complex valve geometry. A general coordinate system without this constraint would have proved more practical. In this light, the computer coding necessary to solve the transformed flow equations was written in a general form which did not necessarily require an orthogonal grid.

It was pointed out by Davies (1981) that a difficulty also lies in a lack of definition in what constitutes a discrete orthogonal grid. Davies measured the error in calculating a known invariant which should have been ideally zero in an orthogonal mesh. However, the result differed from zero for two reasons: error associated with the invariant's finite difference approximation in space and error associated with non-orthogonality. The production of orthogonal meshes is by no means an easy task as it depends heavily upon domain geometry. From a practical point of view, it would be more efficient to produce a general coordinate system.

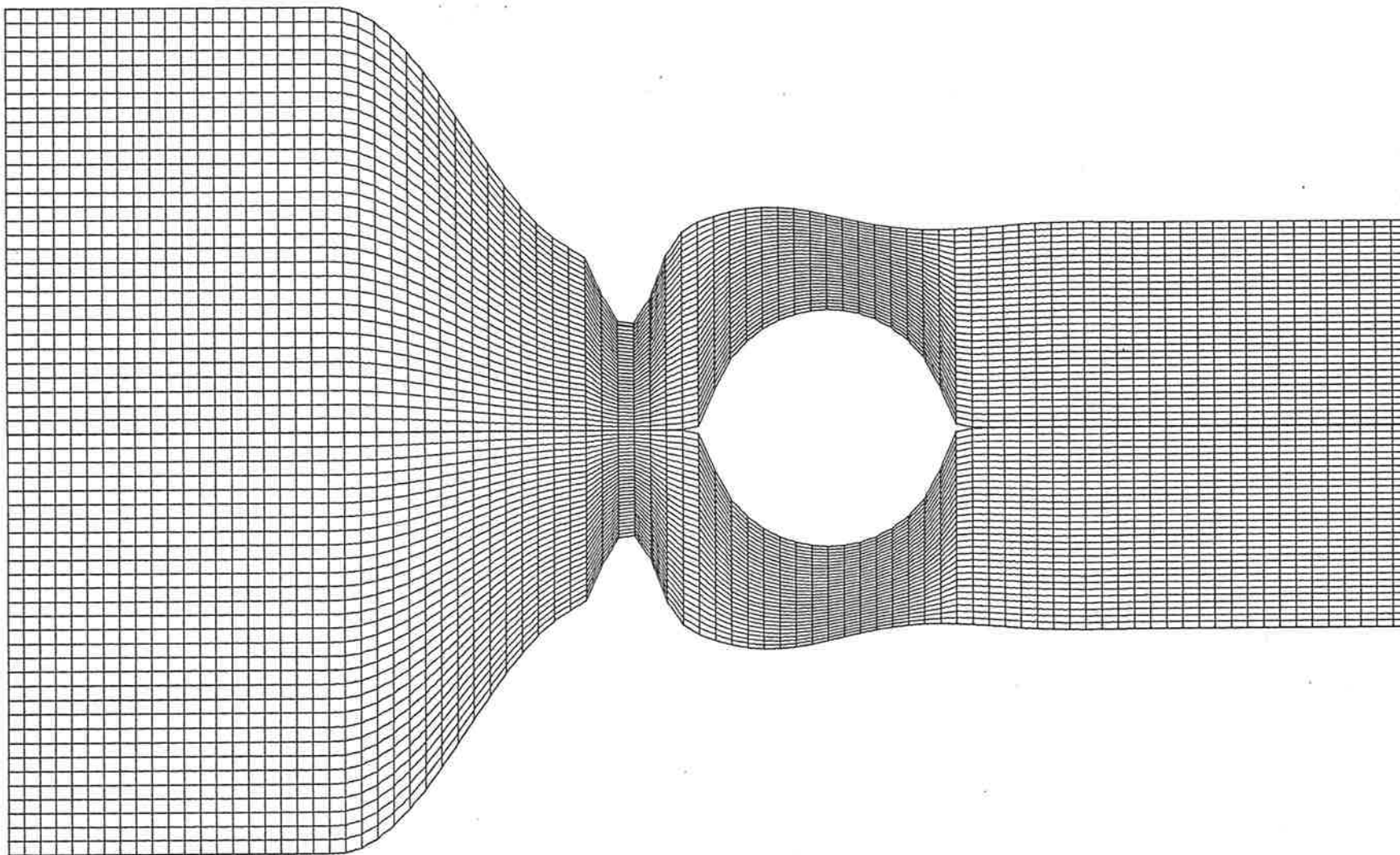


Figure 6.3.2

Section ($i = 14$ to $i = 101$) of the computational grid (101×31) prior to any iteration steps.

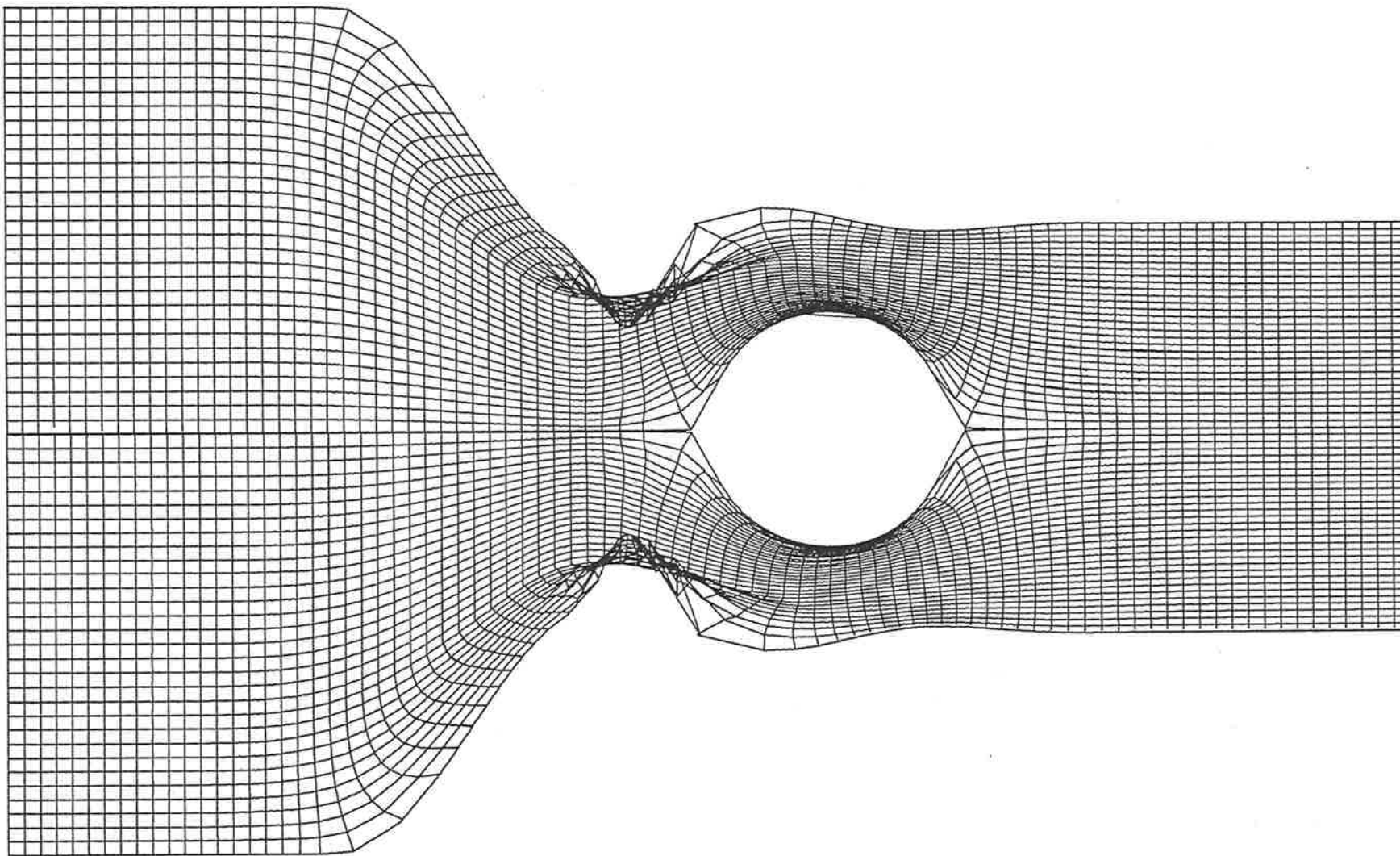


Figure 6.3.3

Section ($i = 14$ to $i = 101$) of the computational grid (101×31) after 10 iteration steps.

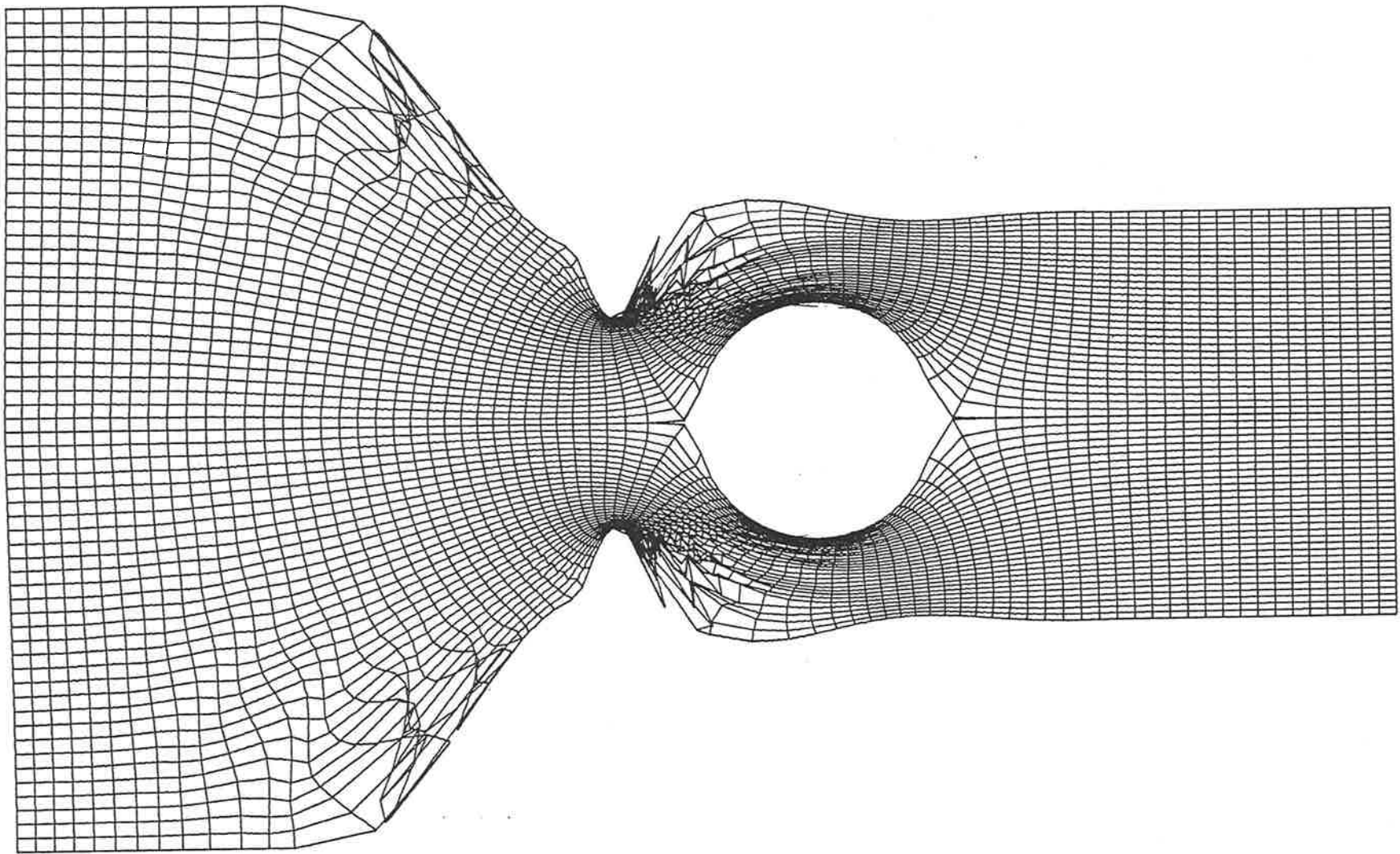


Figure 6.3.4

Section ($i = 14$ to $i = 101$) of the computational grid (101×31) after 100 iteration steps.

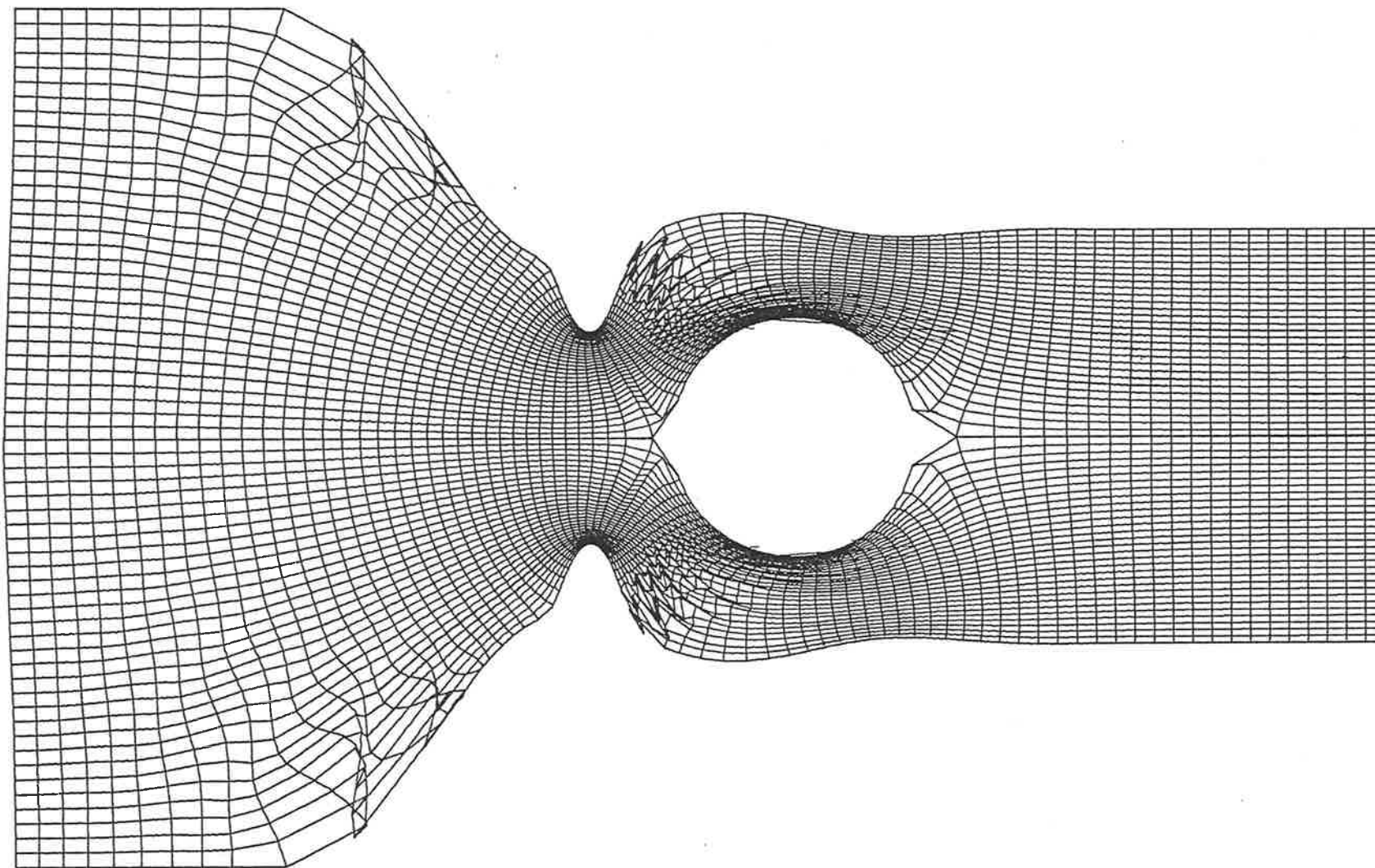


Figure 6.3.5

Section ($i = 14$ to $i = 101$) of the computational grid (101×31) after 200 iteration steps.

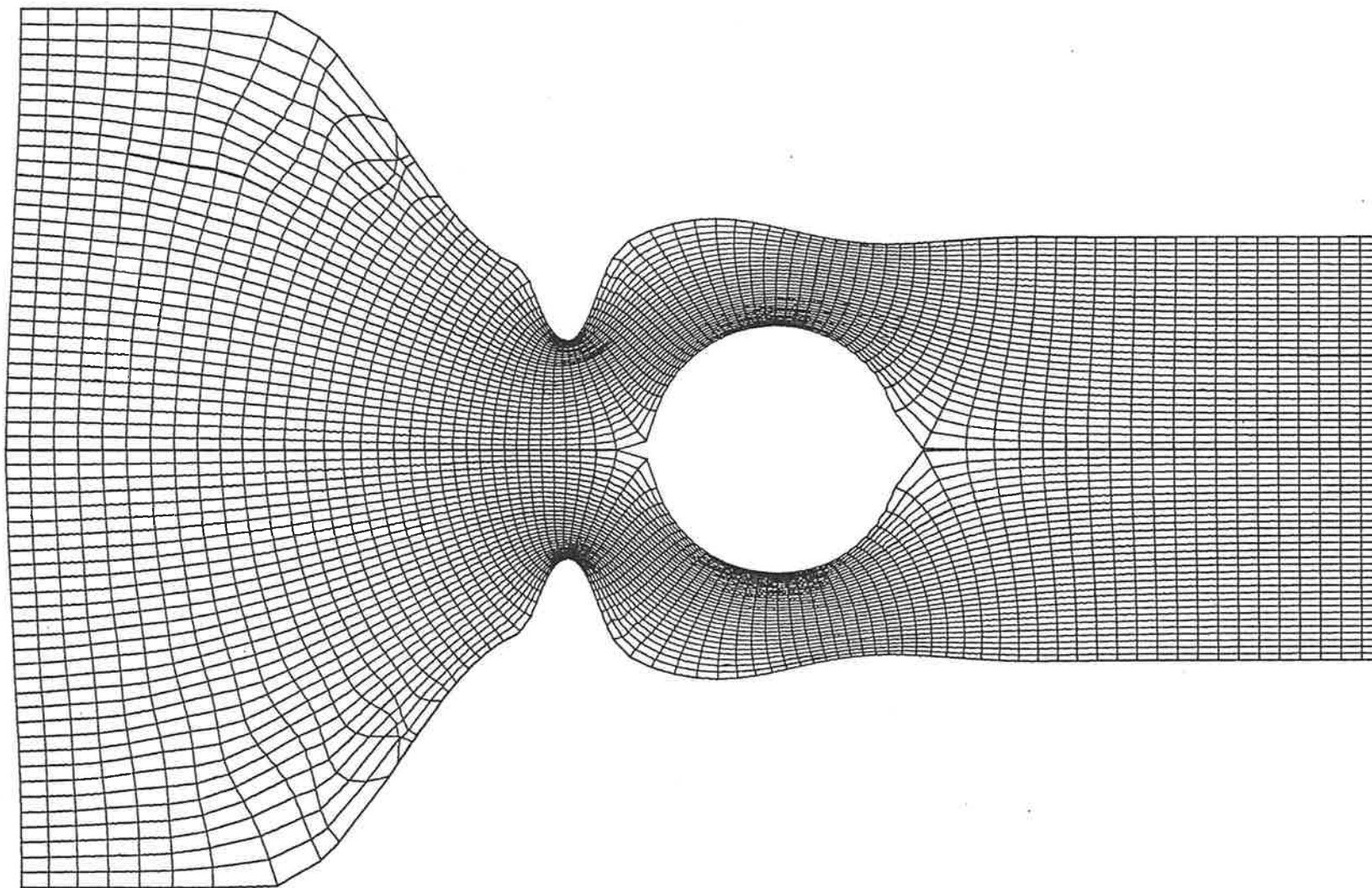


Figure 6.3.6

Section ($i = 14$ to $i = 101$) of the computational grid (101×31) after 300 iteration steps.

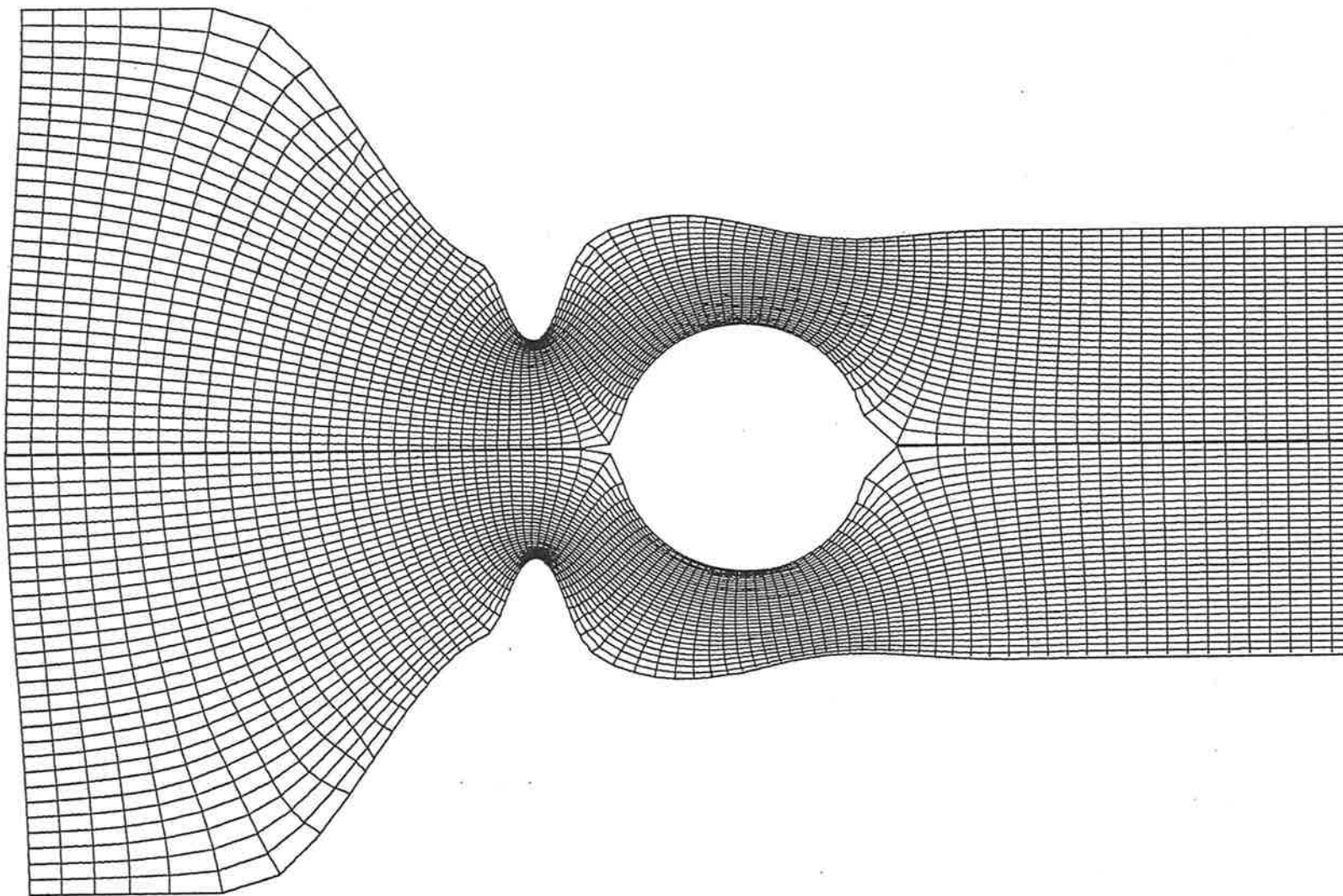


Figure 6.3.7

Section ($i = 14$ to $i = 101$) of the computational grid (101×31) after 400 iteration steps.

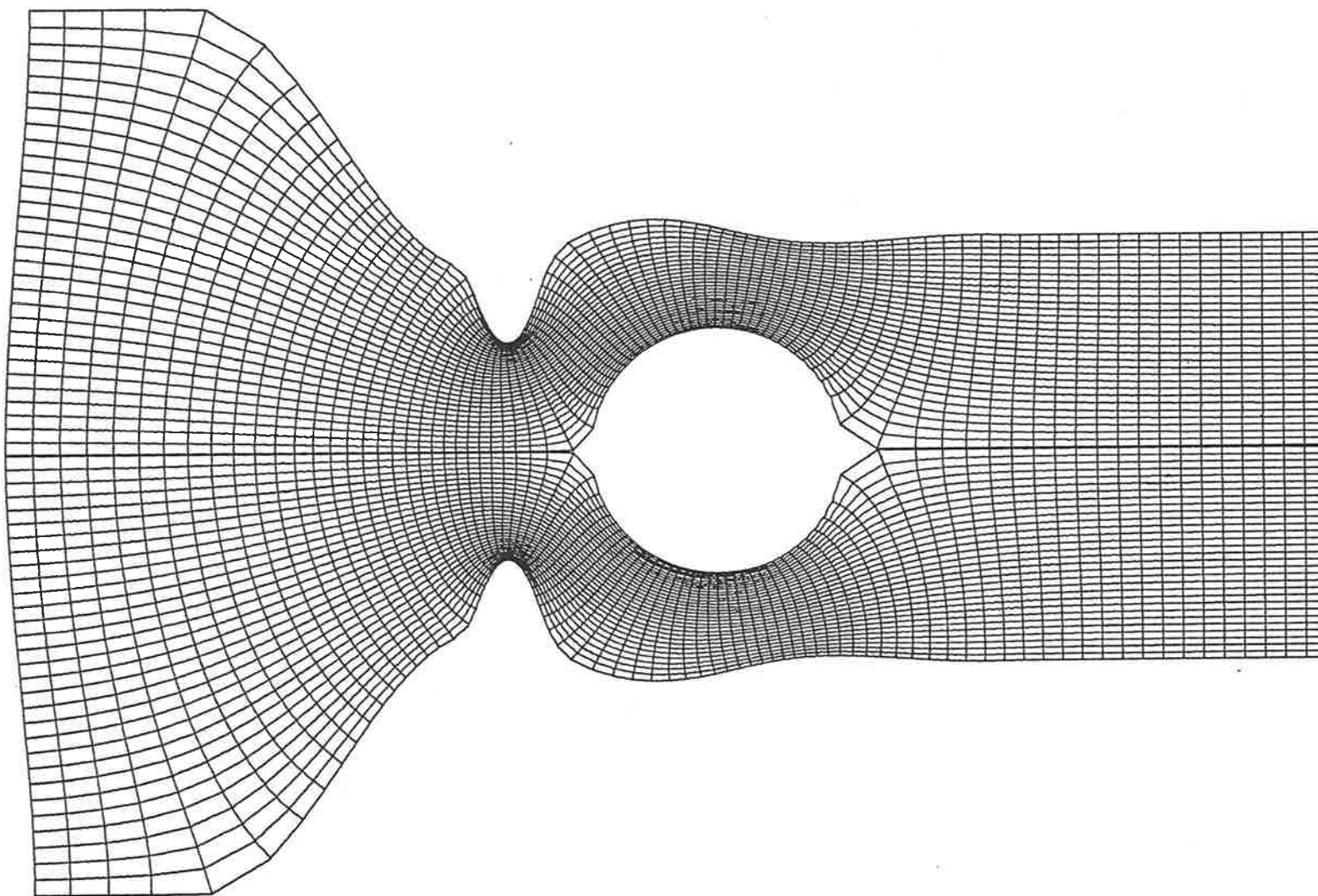


Figure 6.3.8

Section ($i = 14$ to $i = 101$) of the computational grid (101×31) after 500 iteration steps.

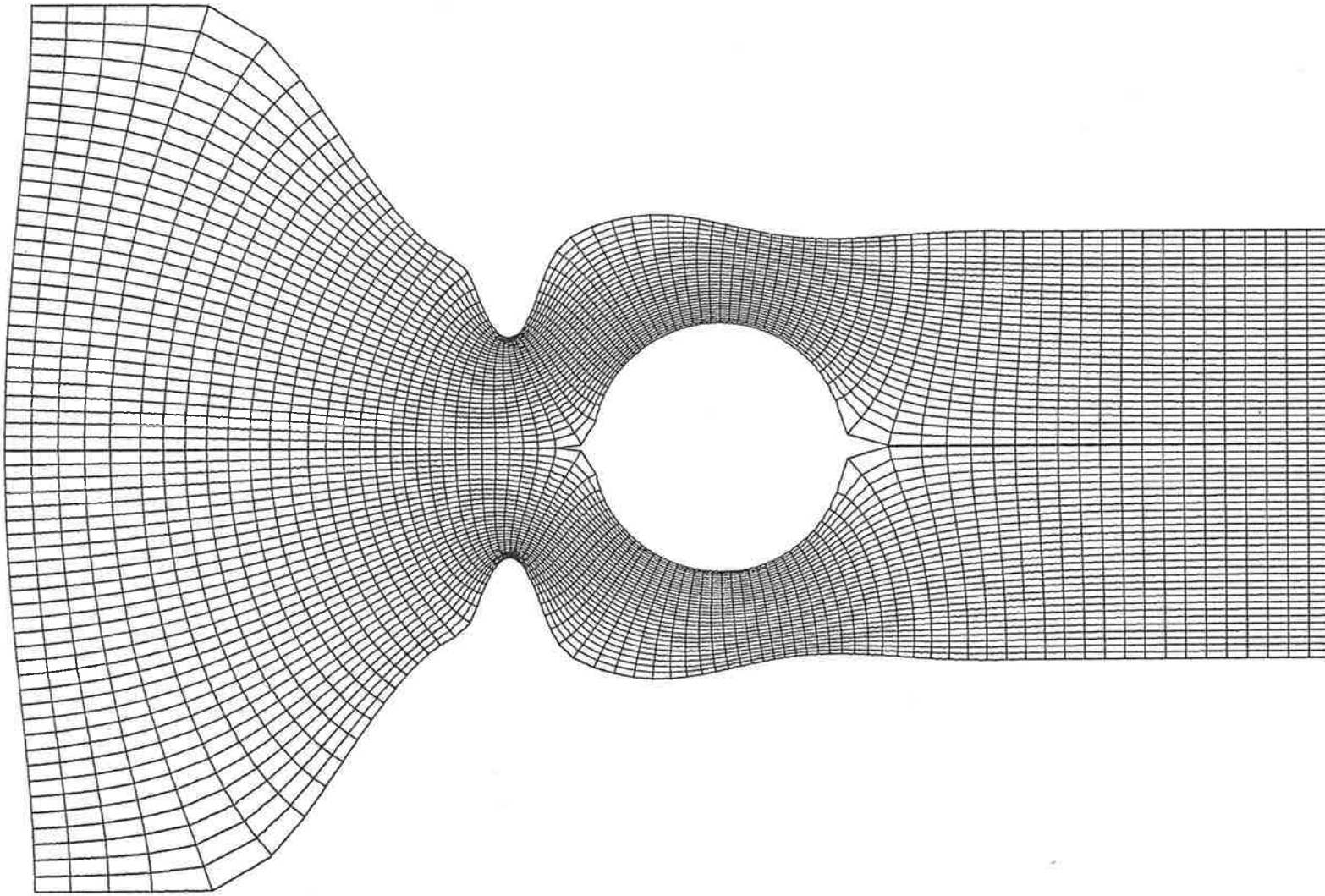


Figure 6.3.9

Section ($i = 14$ to $i = 101$) of the computational grid (101×31) following application of a discrete orthogonalisation procedure (Davies, 1981).

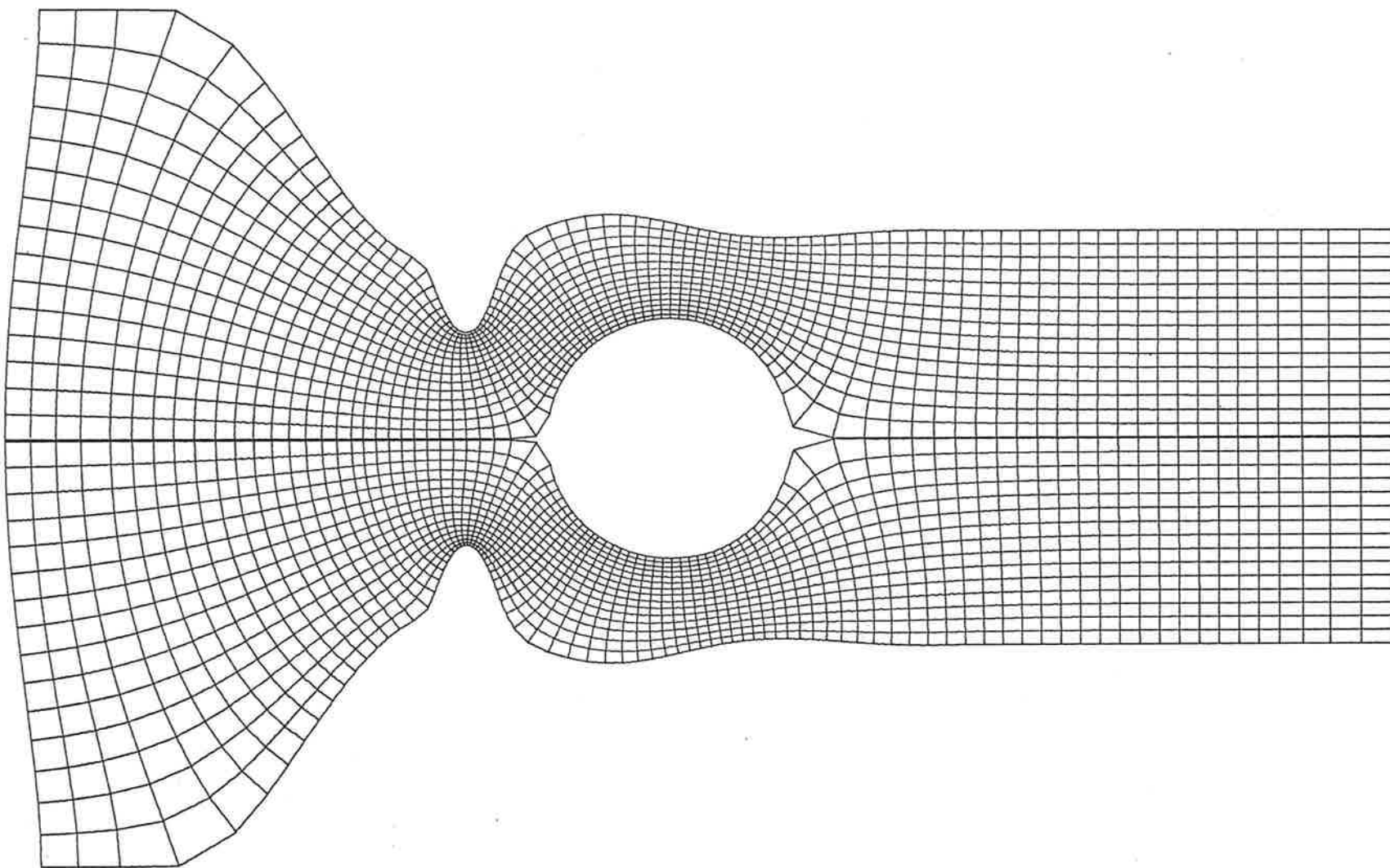


Figure 6.3.10

Section ($i = 15$ to $i = 106$) of the computational grid (120×16) following removal of excess grid lines in the radial direction and insertion of additional axial grid lines.

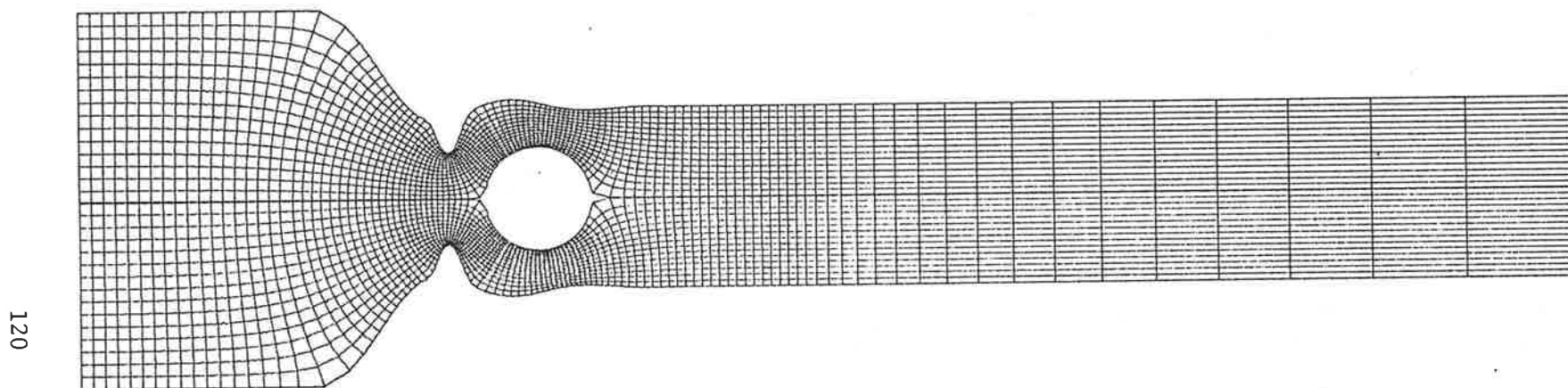


Figure 6.3.11

The final computational grid (120×16).

6.4 Finite Difference Formulation

The transformed governing equations are discretised in the (ξ, η) coordinate system. Central differencing is applied to all derivative terms in equation (6.2.18) and rearranged to yield

$$\begin{aligned} \psi_{i,j} = & \left\{ 2 \left(\frac{\alpha}{\Delta \xi^2} + \frac{\gamma}{\Delta \eta^2} \right) \right\}^{-1} \times \\ & \left[\alpha \frac{(\psi_{i+1,j} + \psi_{i-1,j})}{(\Delta \xi)^2} - 2\beta \frac{(\psi_{i+1,j+1} - \psi_{i+1,j-1} + \psi_{i-1,j-1} - \psi_{i-1,j+1})}{4(\Delta \xi)(\Delta \eta)} \right. \\ & + \gamma \frac{(\psi_{i,j+1} + \psi_{i,j-1})}{(\Delta \eta)^2} + \sigma \frac{(\psi_{i,j+1} - \psi_{i,j-1})}{2(\Delta \eta)} + \tau \frac{(\psi_{i+1,j} - \psi_{i-1,j})}{2(\Delta \xi)} \\ & \left. - \frac{J}{r} \left(\frac{(z_{i+1,j} - z_{i-1,j})}{2(\Delta \xi)} \frac{(\psi_{i,j+1} - \psi_{i,j-1})}{2(\Delta \eta)} - \frac{(z_{i,j+1} - z_{i,j-1})}{2(\Delta \eta)} \frac{(\psi_{i+1,j} - \psi_{i-1,j})}{2(\Delta \xi)} \right) \right. \\ & \left. + J^2 \omega_{i,j} r \right] \end{aligned} \quad (6.4.1)$$

where the indices (i, j) refer to a computational grid point and $(\Delta \xi)$, $(\Delta \eta)$ are the grid spacings in the (ξ, η) system. Note that a staggered grid system was not implemented for Model III.

Equation (6.2.19) is discretised in a similar manner, except for terms such as ω_ξ and ω_η which are treated using upwind differencing for stability purposes. That is, if u and v are the components of velocity in the ξ and η directions, respectively, defined by

$$u = \frac{z_\xi U + r_\xi V}{\sqrt{\gamma}} \quad (6.4.2)$$

and

$$v = -\frac{z_\eta U + r_\eta V}{\sqrt{\alpha}} \quad (6.4.3)$$

then

$$\omega_\xi \approx \begin{cases} \frac{\omega_{i,j} - \omega_{i-1,j}}{(\Delta \xi)}, & \text{if } u \geq 0 \\ \frac{\omega_{i+1,j} - \omega_{i,j}}{(\Delta \xi)}, & \text{if } u < 0 \end{cases} \quad (6.4.4)$$

and

$$\omega_\eta \approx \begin{cases} \frac{\omega_{i,j} - \omega_{i,j-1}}{(\Delta\eta)}, & \text{if } v \geq 0 \\ \frac{\omega_{i,j+1} - \omega_{i,j}}{(\Delta\eta)}, & \text{if } v < 0 \end{cases} \quad (6.4.5)$$

The one-sided approximations for the vorticity together with the central space approximations for the remaining derivatives are substituted into equation (6.2.19) and rearranged to yield a finite difference equation for $\omega_{i,j}$. Upwind differencing is applied in a similar manner to obtain the k and ϵ finite difference forms from equations (6.2.20) and (6.2.21), respectively, but are not reproduced here for brevity.

6.5 Numerical Solution

The finite difference equations are solved iteratively with relaxation parameters to control stability and convergence. Alternating the direction of the iterative sweeps also enhances stability. The inflow boundary conditions for the ψ , k and ϵ were extended throughout the flow domain as initial conditions. Vorticity values along the wall were at first approximated from the initial stream function values with vorticity values being set to zero elsewhere. Once a numerical solution is obtained it is used as the initial condition for a higher Reynolds number flow.

The iterative scheme employed is summarized in steps as follows :

1. Specify initial values for $\psi^{(1)}$, $\omega^{(1)}$, $k^{(1)}$ and $\epsilon^{(1)}$.
2. Update the stream function at grid points immediately away from solid boundaries.
3. Iterate the stream function at interior grid points with a relaxation factor of 1.75 until convergence. Denote the result $\bar{\psi}^{(n+1)}$.

4. Set

$$\psi^{(n+1)} = 0.96 \bar{\psi}^{(n+1)} + 0.04 \psi^{(n)} \quad (6.5.1)$$

5. Evaluate U , V and G .

6. Update the boundary vorticity values.

7. Iterate the vorticity at interior grid points with a relaxation factor of 0.9 until convergence. Denote the result $\bar{\omega}^{(n+1)}$.

8. Set

$$\omega^{(n+1)} = 0.3 \bar{\omega}^{(n+1)} + 0.7 \omega^{(n)} \quad (6.5.2)$$

9. Evaluate ν_{eff} , U , V and G .

10. Apply 'law of the wall' to grid points immediately away from solid boundaries.

11. Evaluate the turbulent kinetic energy at interior points with a relaxation parameter of 0.15. Denote the result $\bar{k}^{(n+1)}$.

12. Set

$$k^{(n+1)} = 0.3 \bar{k}^{(n+1)} + 0.7 k^{(n)} \quad (6.5.3)$$

13. Apply 'law of the wall'.

14. Evaluate the turbulent kinetic energy dissipation rate at interior points with a relaxation parameter of 0.15. Denote the result $\bar{\epsilon}^{(n+1)}$.

15. Set

$$\epsilon^{(n+1)} = 0.3 \bar{\epsilon}^{(n+1)} + 0.7 \epsilon^{(n)} \quad (6.5.4)$$

16. Test for global convergence and return to Step 2 if necessary.

Global convergence is declared when all dependent variables change by less than 1 % of their maximum value between iteration steps for at least 50 times. Testing for global convergence started only after an initial series of 50 global iterations were completed. For $Re = 600$, 123 global iterations were required for convergence resulting in a total of 223 iterations for ψ , 589 for ω , 186 for k and 186 for ϵ . The solution procedure was written in FORTRAN 77 and required approximately 25 minutes CPU to execute on a VAX 11/785 at the University of Adelaide. A similar number of iterations and CPU time were also required for the other flow rates.

The accuracy of the Model III numerical solutions was verified by repeating the calculations for $Re = 1000, 2000$ and 4000 on a 120×31 coordinate system. This was necessary to ensure that a sufficiently dense grid had been chosen for the computations. The denser mesh was generated simply by including some additional axial grid lines to the intermediate grid shown in Figure 6.3.9. The solutions obtained with the 120×31 grid were compared with the solutions based on the coarser 120×16 grid. The converged solutions differed only slightly in ψ , ω , k and ϵ . Typically, the changes were always below 3 % in the immediate vicinity of the valve, but required almost 2.5 times the CPU time to execute. As there were no substantial solution differences between the grid systems, the 120×16 computational mesh was considered dense enough for performing the calculations.

6.6 Discussion of Results

Converged numerical solutions for steady turbulent flow through a fully open Starr-Edwards 1260-11A prosthetic heart valve were obtained for Reynolds numbers of 600, 1000, 1500, 2000, 2800, 4000 and 6000 corresponding to steady flow rates of 4.879, 8.133, 12.199, 16.265, 22.771, 32.530 and 48.796 *litres min⁻¹*, respectively.

Reynolds number	600	1000	1500	2000	2800	4000	6000
Inflow velocity, U_o (cm sec⁻¹)	3.096	5.159	7.739	10.318	14.446	20.637	30.955
Steady flow rate (cm³ sec⁻¹)	81.326	135.543	203.314	271.086	379.520	542.172	813.258
Steady flow rate (lt min⁻¹)	4.879	8.133	12.199	16.265	22.771	32.530	48.796
Reattachment length, L/D	0.327	0.333	0.321	0.322	0.334	0.337	0.354
STREAM FUNCTION, ψ							
Maximum (i, j)	0.1067 _(60,13)	0.1091 _(60,13)	0.1114 _(59,13)	0.1121 _(59,13)	0.1135 _(59,13)	0.1139 _(59,13)	0.1103 _(59,13)
Minimum (i, j)	-0.0104 _(81,06)	-0.0116 _(81,06)	-0.0140 _(81,07)	-0.0146 _(81,07)	-0.0163 _(81,07)	-0.0166 _(81,07)	-0.0164 _(81,07)
AXIAL VELOCITY (U/U_o)							
Maximum (i, j)	15.15 _(44,10)	14.98 _(44,10)	15.15 _(41,14)	15.67 _(41,14)	16.59 _(41,14)	17.01 _(42,14)	18.51 _(42,14)
Large recirculation zone (i, j)	-3.48 _(83,01)	-3.71 _(83,01)	-4.00 _(83,01)	-4.07 _(83,01)	-4.33 _(83,01)	-4.39 _(83,01)	-4.37 _(83,01)
Sinus of Valsalva zone (i, j)	-2.02 _(61,14)	-2.25 _(61,14)	-2.57 _(60,14)	-2.66 _(60,14)	-2.82 _(60,14)	-2.86 _(60,14)	-2.33 _(60,14)
RADIAL VELOCITY (V/U_o)							
Maximum (i, j)	11.33 _(52,03)	10.99 _(52,03)	10.62 _(52,03)	10.33 _(52,03)	9.73 _(52,03)	9.36 _(52,03)	8.16 _(52,03)
Minimum (i, j)	-7.61 _(38,14)	-7.75 _(38,14)	-7.82 _(38,14)	-7.86 _(38,14)	-7.92 _(38,14)	-7.95 _(38,14)	-8.00 _(38,14)
TOTAL VELOCITY (U_{total}/U_o)							
Maximum (i, j)	17.02 _(53,03)	16.87 _(54,03)	16.64 _(54,03)	16.43 _(55,03)	17.10 _(41,14)	17.47 _(41,14)	18.51 _(42,14)
TURBULENT SHEAR STRESS, σ_{rz} (N m⁻²)							
Maximum (i, j)	6.1 _(41,15)	13.7 _(42,15)	26.6 _(42,15)	42.1 _(42,15)	74.4 _(42,15)	138.3 _(42,15)	320.2 _(42,15)
Sewing ring negative peak (i, j)	-0.4 _(41,13)	-1.0 _(41,13)	-2.7 _(41,13)	-4.9 _(41,13)	-16.7 _(41,13)	-38.2 _(41,13)	-208.1 _(41,13)
Sinus of Valsalva zone (i, j)	0.6 _(48,13)	1.3 _(48,13)	3.3 _(48,13)	5.5 _(62,13)	18.0 _(64,13)	35.9 _(63,13)	101.7 _(64,13)
Sinus of Valsalva wall (i, j)	-0.4 _(61,15)	-0.9 _(61,15)	-1.8 _(61,15)	-2.8 _(61,15)	-4.7 _(61,15)	-8.1 _(60,15)	-12.0 _(60,15)
Occluder wall (i, j)	-2.3 _(58,02)	-5.2 _(58,02)	-11.3 _(58,02)	-19.1 _(58,02)	-33.4 _(58,02)	-63.2 _(58,02)	-107.1 _(59,02)
Aortic wall (i, j)	1.9 _(72,15)	3.9 _(73,15)	7.5 _(73,15)	11.2 _(73,15)	17.8 _(74,15)	30.7 _(75,15)	59.0 _(75,15)

Table 6.6.1
Summary of Model III numerical solutions

Many important aspects of the computed results are summarised in Table 6.6.1. Numerical solutions were also obtained for Reynolds numbers of 1232, 2457 and 3077 corresponding to steady flow rates of 10.0, 20.0 and 25.0 *litres min⁻¹* (i.e. 167, 333 and 417 *cm³ sec⁻¹*), respectively. These flow rates were studied to allow comparison between the numerical solutions presented here and the experimental measurements conducted by Yoganathan *et al* (1978, 1979, 1981).

Stream function contours are presented in Figure 6.6.1. The solutions show two regions of separated flow. A large separation zone is attached to the downstream side of the ball occluder and a smaller annular zone is attached to the sewing ring and inside the sinus. Both of these regions have been observed in physical experiments (Figliola and Mueller, 1981; Yoganathan *et al*, 1978, 1979, 1981). No separated regions are predicted upstream of the sewing ring nor along the aortic wall. The solutions obtained with Model II are similar in this respect. The experimental studies conducted by Figliola and Mueller (1981) on flow through a Starr-Edwards 1260-11A caged-ball valve in an aortic-shaped chamber contained separated flow regions attached to the sewing ring (upstream and downstream), the aortic side of the cage struts and distal to the ball occluder. The separation region immediately upstream of the sewing ring was only described qualitatively and appeared to be very small. Very similar observations were also made by Yoganathan *et al* (1979) on experiments conducted using a Starr-Edwards 1260-12A caged-ball prosthesis. With the exception of the regions attached to the cage struts, which were not modelled in Model III, the numerical solutions compare favourably with these experimental observations.

In earlier experimental measurements of air flow through a caged-ball valve in an aortic-shaped chamber, Figliola and Mueller (1977) did indeed observe a separation

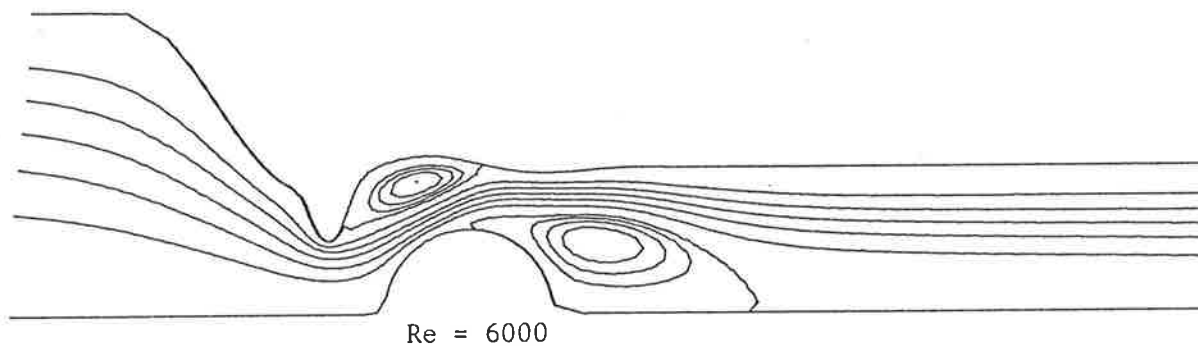
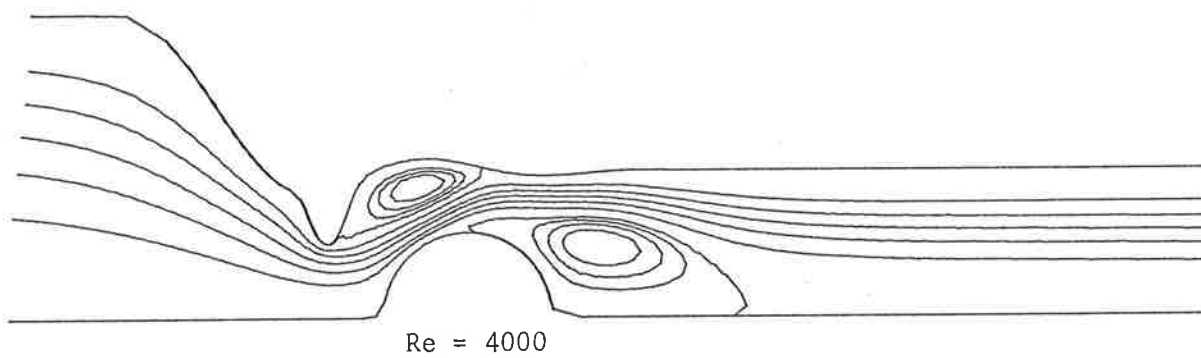
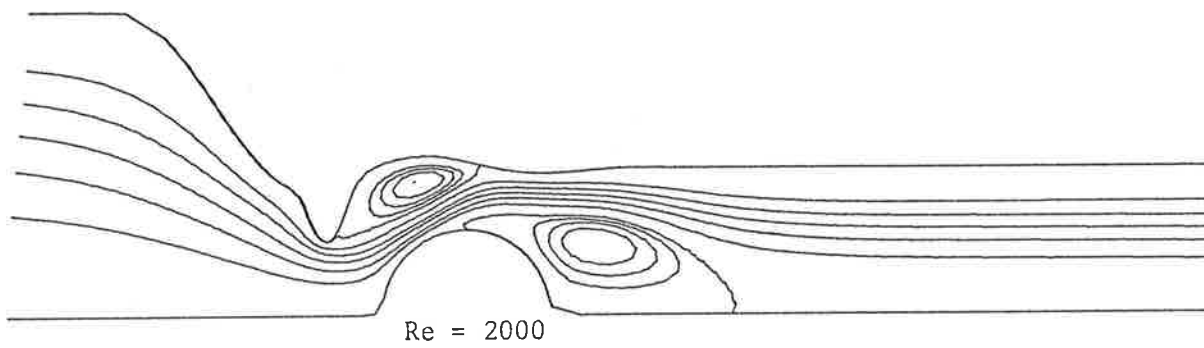
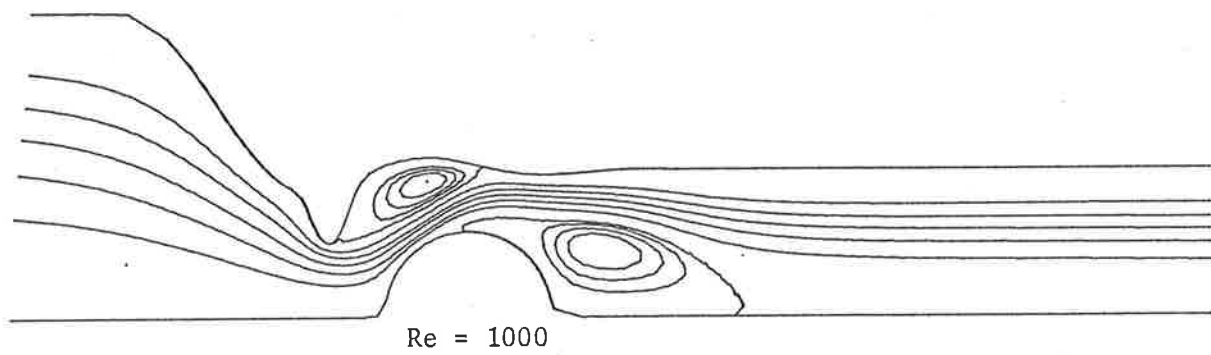


Figure 6.6.1

Computed mean streamlines for $Re = 1000, 2000, 4000$ and 6000 .

zone alongside the aortic wall and another immediately upstream of the sewing ring. However, the aortic wall separation zone was not present in subsequent measurements when using a blood analog fluid (Figliola and Mueller, 1981). Consequently, a direct comparison between the air flow experiments and the Model III numerical solutions is inappropriate.

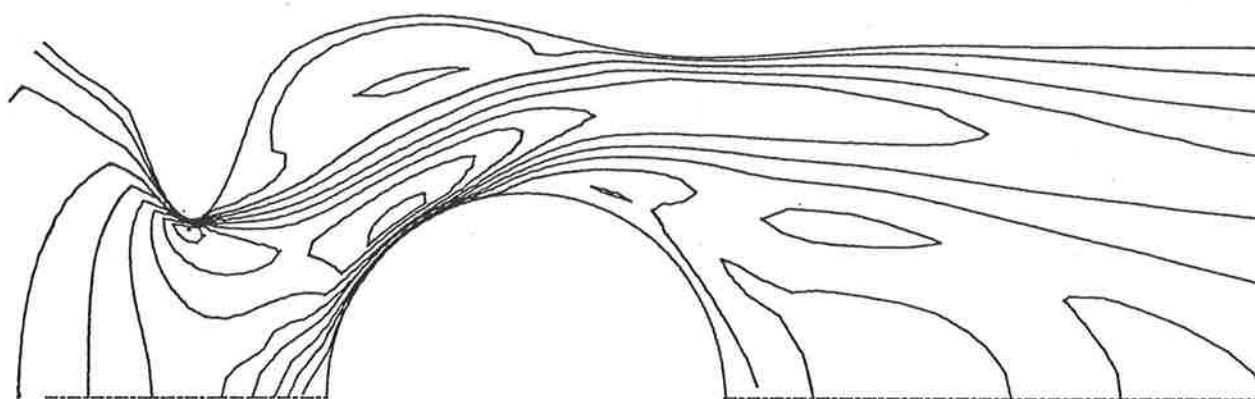
The separated flow regions predicted with Model III exhibited only slight change in size and shape throughout the range of flow rates examined. The nondimensional reattachment length of the main separation zone, L/D , measured from the downstream point of the occluder, had an average value of 0.333 and gradually increased to 0.354 at $Re = 6000$ (Table 6.6.1). These values correspond to 2.1 cm and 2.3 cm, respectively. The maximum diameter of the main zone increased only slightly from 2.0 cm to 2.1 cm between $Re = 1000$ and 6000. However, it did become somewhat broader near its reattachment point at higher flow rates. The reattachment point of the smaller annular zone in the sinus was located 1.7 cm downstream from the sewing ring tip for all flow rates. Yoganathan *et al* (1981) pointed out that although both of these regions have been observed qualitatively by other investigators, little quantitative information was available. They addressed this situation by measuring the width of the large separation zone of the Starr-Edwards 1260-12A prosthesis using both tied and untied ball valves. Their experiments indicated a decrease in the main zone width, measured across the ball apex location, as the flow rate was increased from 167 to 417 $cm^3 \text{ sec}^{-1}$ irrespective of whether the ball occluder was fixed or free. For the fixed occluder, the zone width was approximately 1.5 cm and 1.0 cm at flow rates of 167 and 417 $cm^3 \text{ sec}^{-1}$, respectively. These dimensions are somewhat smaller than the maximum value (2.1 cm) numerically predicted for the 1260-11A valve. No measurements were given for the length of the main separation

zone.

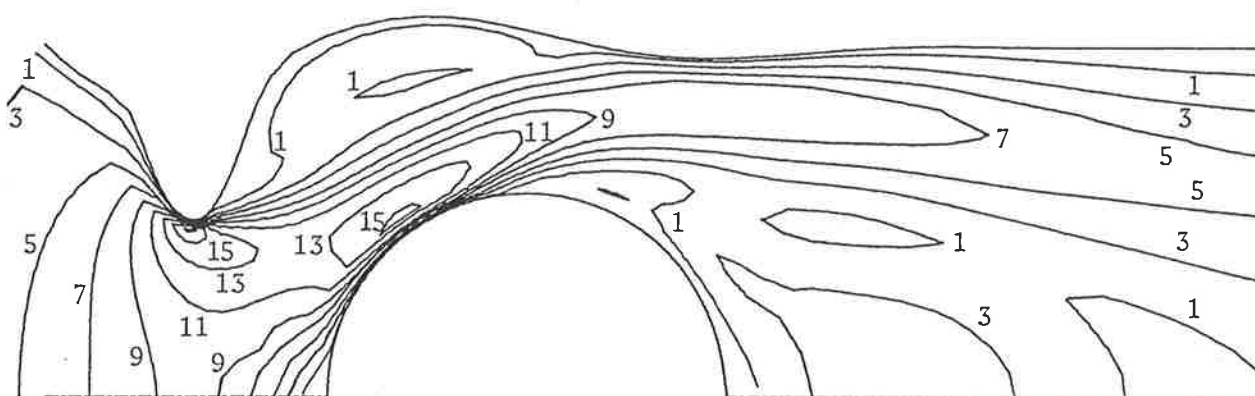
The location of the separation point for both zones remained fixed for all Reynolds numbers investigated. The separation point for the main zone was located along the top of the ball occluder wall at an angle of $\theta = 96^\circ$, where θ is measured clockwise from the upstream point of the occluder ($\theta = 0^\circ$). Figliola and Mueller (1981) reported that this point was not stationary but fluctuated by several degrees around $\theta = 110^\circ$ under steady flow conditions. However, the time-averaged modelling reported can only predict a fixed location. The separation point for the sinus separation zone was located a little downstream of the sewing ring tip in agreement with experimental observations (Figliola and Mueller, 1981; Yoganathan *et al*, 1981).

In another computational study, laminar flow was modelled through a ball valve in an aortic-shaped chamber using a finite element method (Idelsohn *et al*, 1985). In addition to the two separated flow regions predicted with Model III, the laminar flow study also predicted a very large separation zone alongside the aortic wall. It is not completely clear whether this discrepancy with experimental observations mentioned above is due to the method of solution or the choice of an underlying laminar flow model. However, it is almost certainly the latter since previous laminar models for flow through disk valves in aortic-shaped chambers have also predicted a separated region attached to the aortic wall (Underwood and Mueller, 1979).

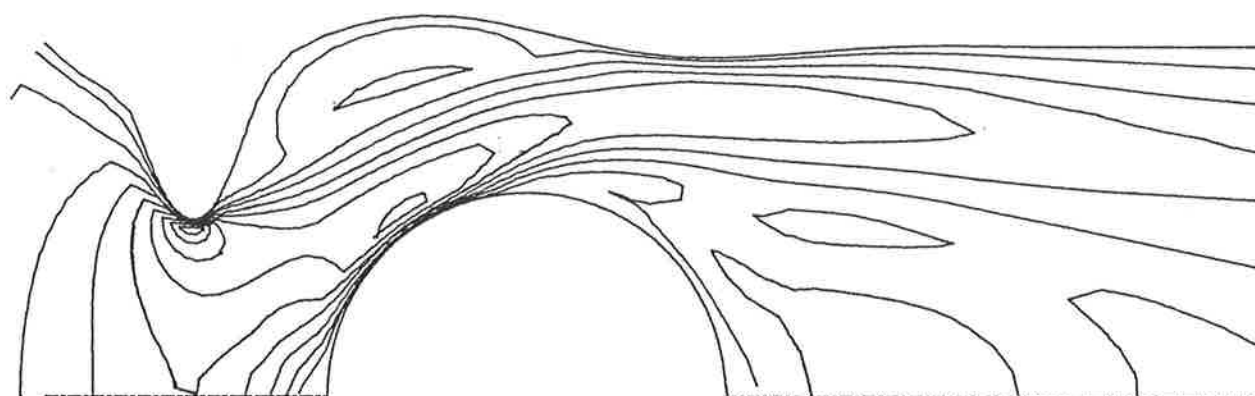
Computed mean total velocity contours are shown in Figure 6.6.2 for several Reynolds numbers. From these figures, the fluid accelerates rapidly as it approaches the primary orifice. The fluid jet encounters the occluder and travels along it briefly before it is deflected to impinge upon the aortic wall. The jet then begins to slowly decelerate and move away from the aortic wall as it continues downstream past the



Re = 2800



Re = 4000



Re = 6000

Figure 6.6.2

Computed total velocity contours (numbered in multiples of U_0) for
 Re = 2800 ($U_0 = 14.45 \text{ cm sec}^{-1}$), Re = 4000 ($U_0 = 20.64 \text{ cm sec}^{-1}$) and
 Re = 6000 ($U_0 = 30.96 \text{ cm sec}^{-1}$).

occluder. Large velocities are computed near the tip of the sewing ring, near the upstream face of the ball surface and in the annular region between the ball and the aortic wall. The contours also reveal the presence of a high velocity region extending between the sewing ring tip and the occluder. However, at physiologically relevant flow rates the largest mean axial velocities occurred consistently near the sewing ring tip and increased from 15.2 to $18.5 U_0$ (Table 6.6.1).

The maximum reverse flow velocities occurring in the main separation zone increased from 3.5 to $4.4 U_0$ for $Re = 600$ to 6000 (Table 6.6.1). In the sinus separation zone these velocities were comparatively smaller, increasing from 2.0 to $2.9 U_0$, but then decreasing to $2.3 U_0$ for $Re = 6000$. Along the downstream face of the sewing ring the computed flow is very slow moving and potentially thrombogenic. For $Re = 4000$, mean total velocities as low as $0.24 U_0$ are present, with values lower than $1.0 U_0$ at nearby grid points. The flow immediately downstream from the ball occluder separation point is also relatively stagnant. For $Re = 4000$ the lowest total velocity is $0.17 U_0$ with many surrounding grid points having values less than $1.0 U_0$.

The potential for thrombus formation near prosthetic surfaces is enhanced by slow moving flow. Figliola and Mueller (1981) concluded that separated flow regions observed experimentally under steady flow conditions correlated very well with the incidence of thrombosis based on reported long term clinical studies. In this study the regions of slow moving flow near a Starr-Edwards caged-ball prosthesis are clearly identified (Figure 6.6.2).

The detail present in Figure 6.6.2 provides a complete description of the flow behaviour in the vicinity of the caged-ball prosthesis. The wealth of information far surpasses the composite picture usually obtained when performing *in vitro* mea-

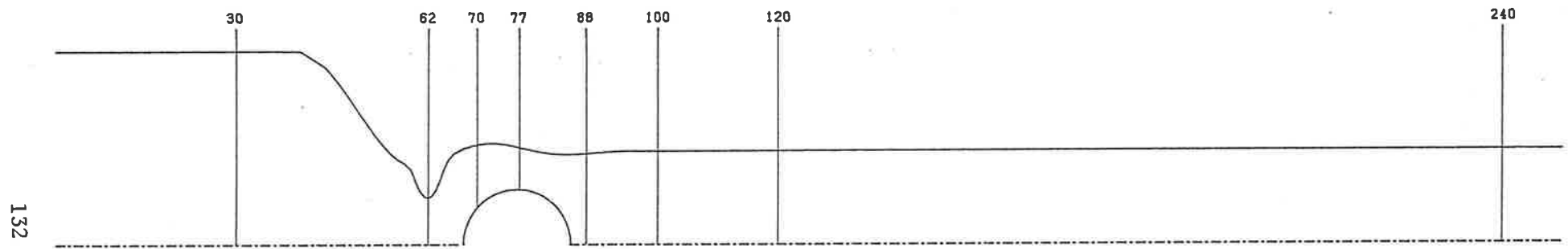


Figure 6.6.3

Cross sections at which the mean axial velocity profiles are computed. Axial distances (mm) are measured from the inflow boundary.

surements at several flow cross sections. Furthermore, the numerical solutions are totally flexible in the manner in which they can be displayed. Hence, to facilitate comparison between the Model III numerical solutions and the experimental measurements performed by Yoganathan *et al* (1979) and Figliola and Mueller (1981), mean axial velocity profiles were computed at several locations in the flow field (Figure 6.6.3). The computations were carried out at steady flow rates of 167, 333 and $417 \text{ cm}^3 \text{ sec}^{-1}$ — the same flow rates used by Yoganathan *et al* (1979).

The computed mean axial velocity profiles are given in Figures 6.6.4 to 6.6.7, with relevant experimental measurements shown in Figures 6.6.8 and 6.6.9. At the $z = 30 \text{ mm}$ location (Figure 6.6.4), the computed velocity profiles appear to be partly influenced by the narrowing chamber dimensions further upstream. They no longer conform to the fully developed turbulent velocity profiles specified at inflow. At the primary orifice location, $z = 62 \text{ mm}$, very high axial velocities (271 cm sec^{-1} at the $417 \text{ cm}^3 \text{ sec}^{-1}$ flow rate) are present near the sewing ring tip. The magnitude of the velocity decreases towards the centreline. No experimental data is available for comparison at this location.

Reverse flow (-38 cm sec^{-1}) present in the sinus separation zone can be clearly seen in the $z = 70 \text{ mm}$ velocity profile (Figure 6.6.5). The maximum axial velocity (187 cm sec^{-1}) occurs very close to the upstream surface of the ball occluder. This profile is very similar in shape to the one experimentally measured at an identical location and shown in Figure 6.6.9. The $z = 77 \text{ mm}$ location corresponds to the region directly above the ball occluder. Here, the magnitude of the axial velocities remain high (156 cm sec^{-1}) but are generally lower than those predicted at the $z = 70 \text{ mm}$ location. Two small areas of reverse flow corresponding to sections of the main and sinus separation zones are evident. Also, the shape of the profiles

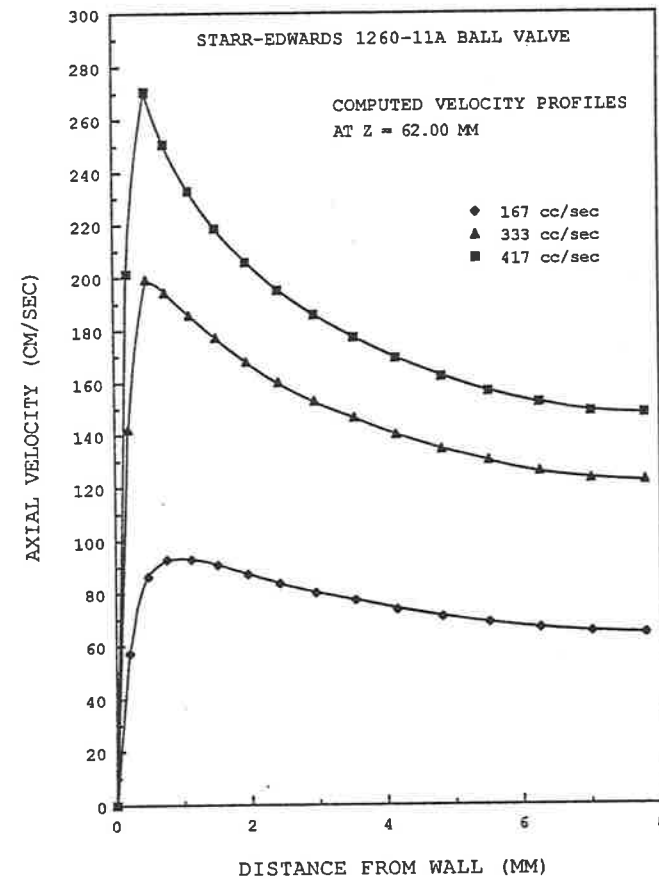
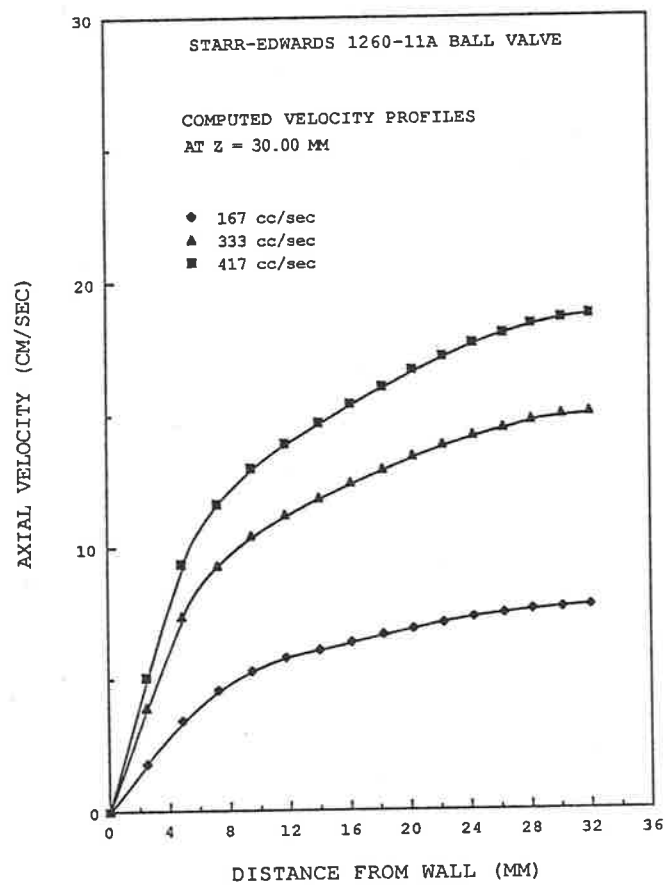


Figure 6.6.4

Computed mean axial velocity profiles at 30mm and 62mm from the inflow boundary (refer to Figure 6.6.3).

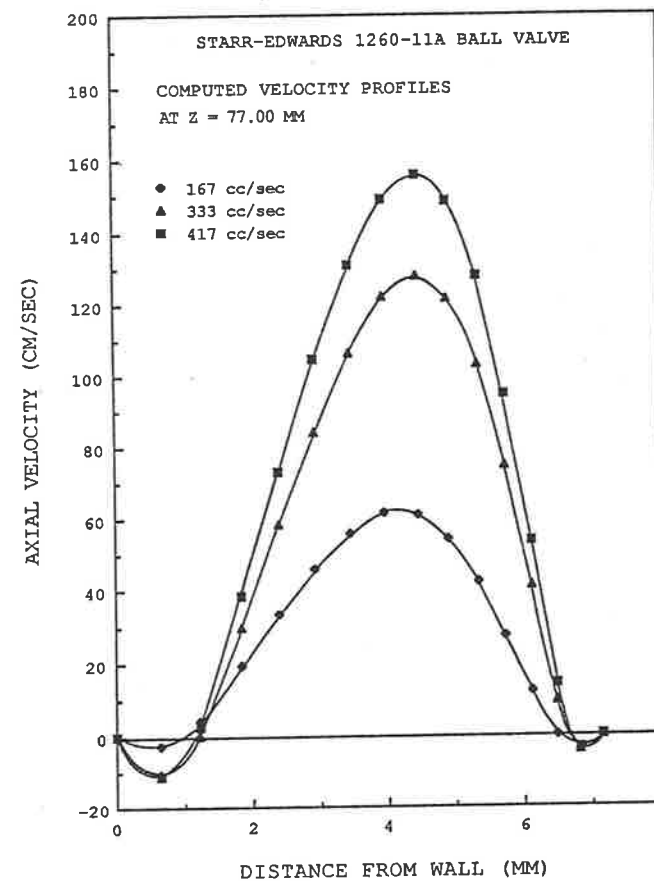
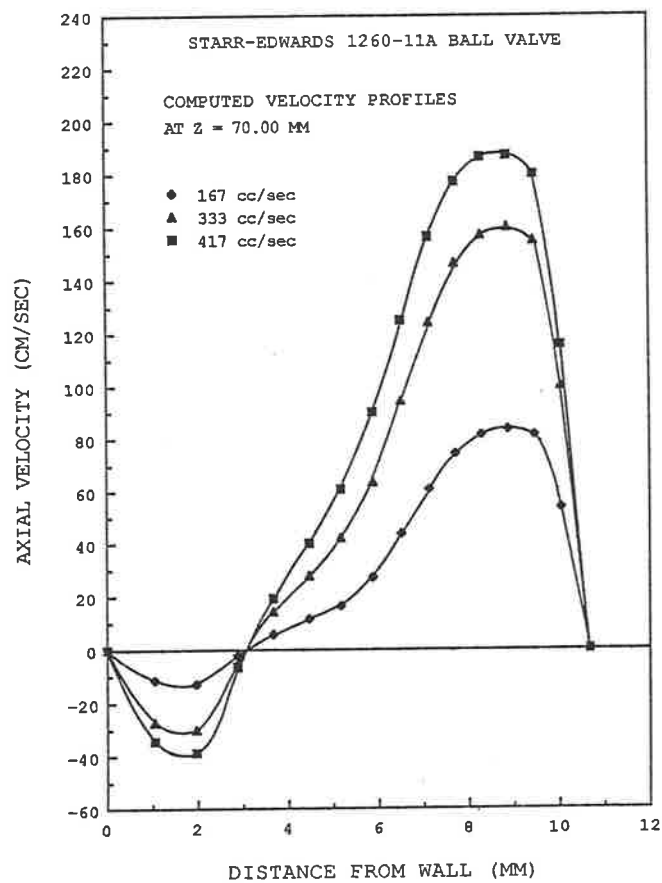


Figure 6.6.5

Computed mean axial velocity profiles at 70mm and 77mm from the inflow boundary (refer to Figure 6.6.3).

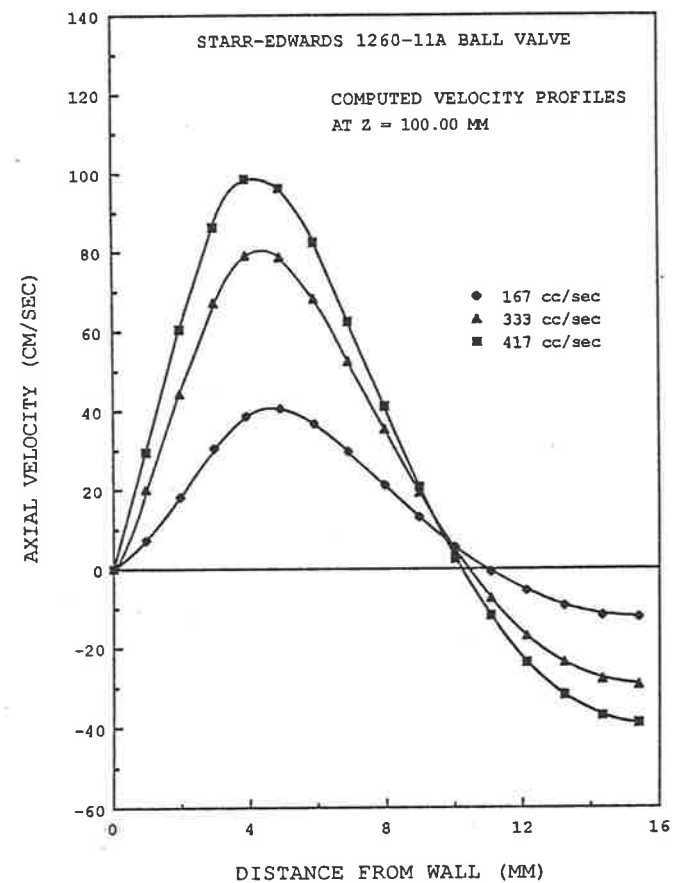
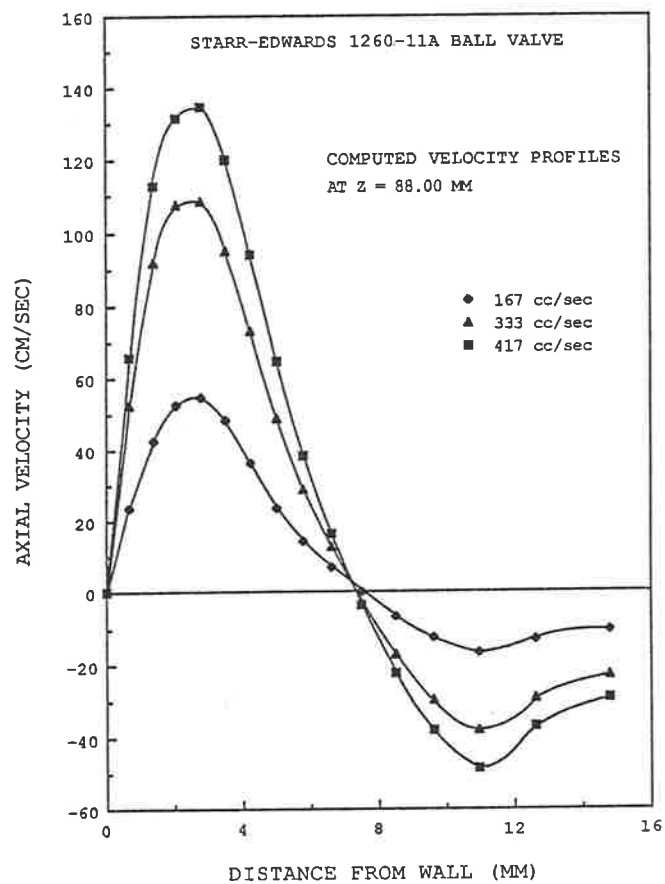


Figure 6.6.6

Computed mean axial velocity profiles at 88mm and 100mm from the inflow boundary (refer to Figure 6.6.3).

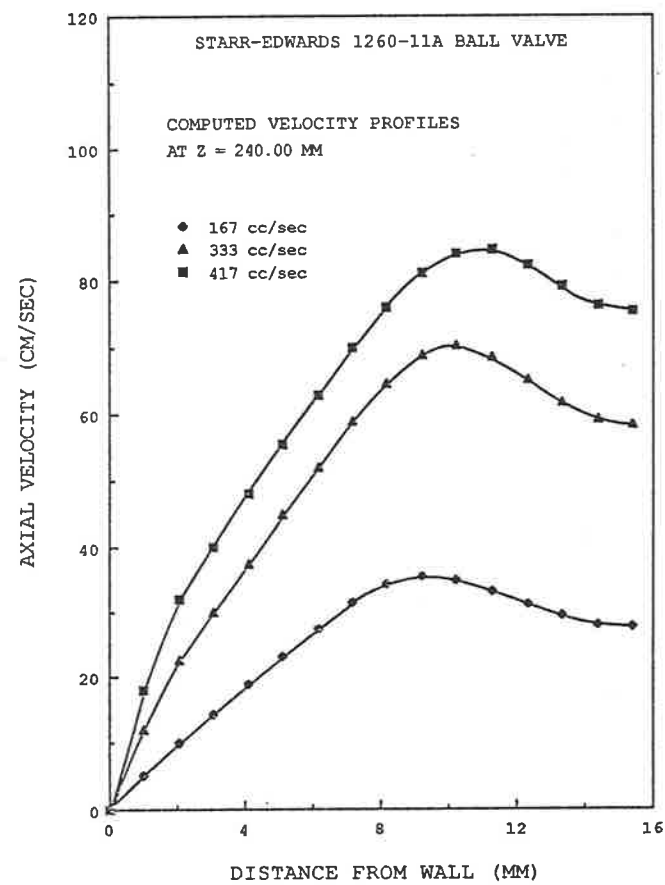
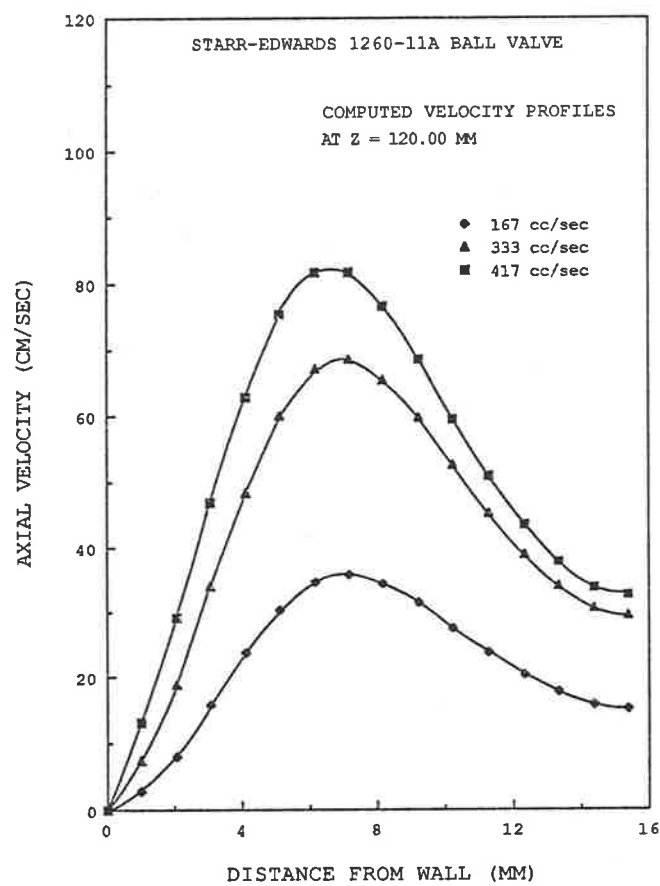


Figure 6.6.7

Computed mean axial velocity profiles at 120mm and 240mm from the inflow boundary (refer to Figure 6.6.3).

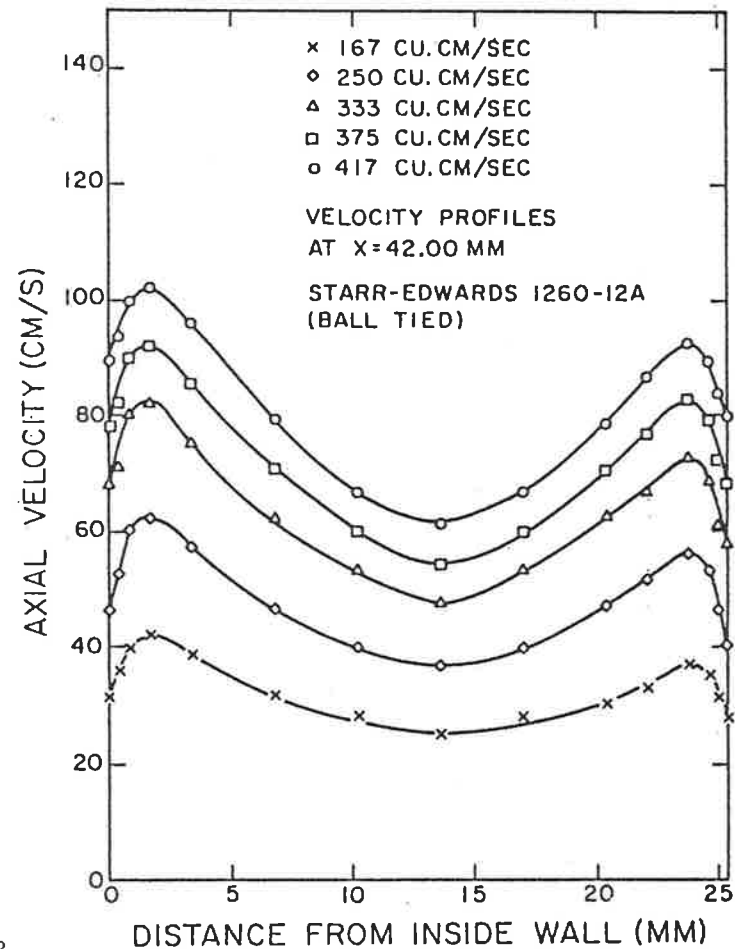
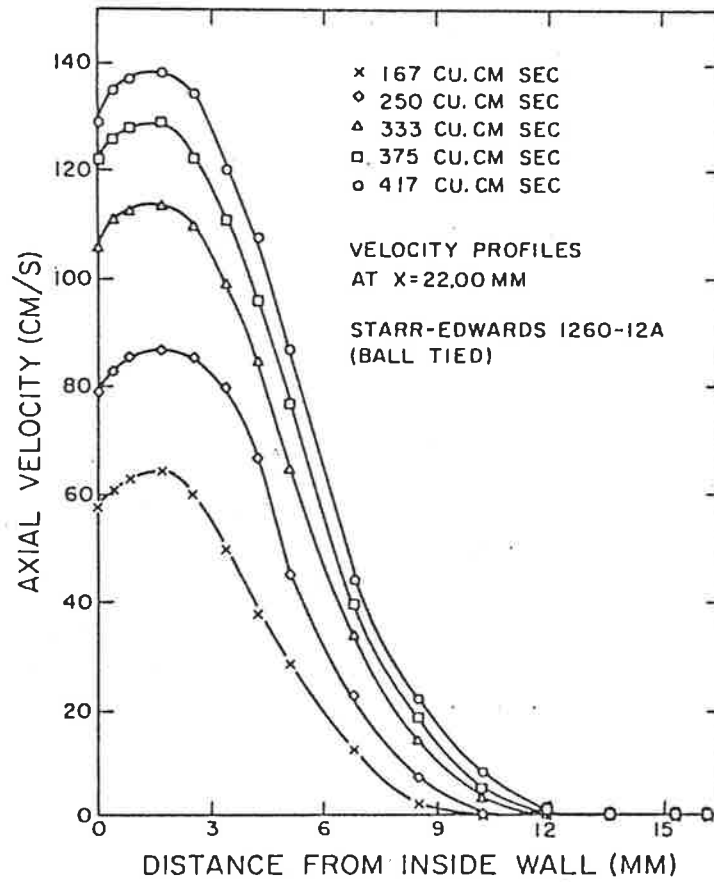


Figure 6.6.8

Experimentally measured axial velocity profiles at 22mm and 42mm downstream of a Starr-Edwards 1260-12A caged-ball prosthesis (Yoganathan *et al*, 1979).

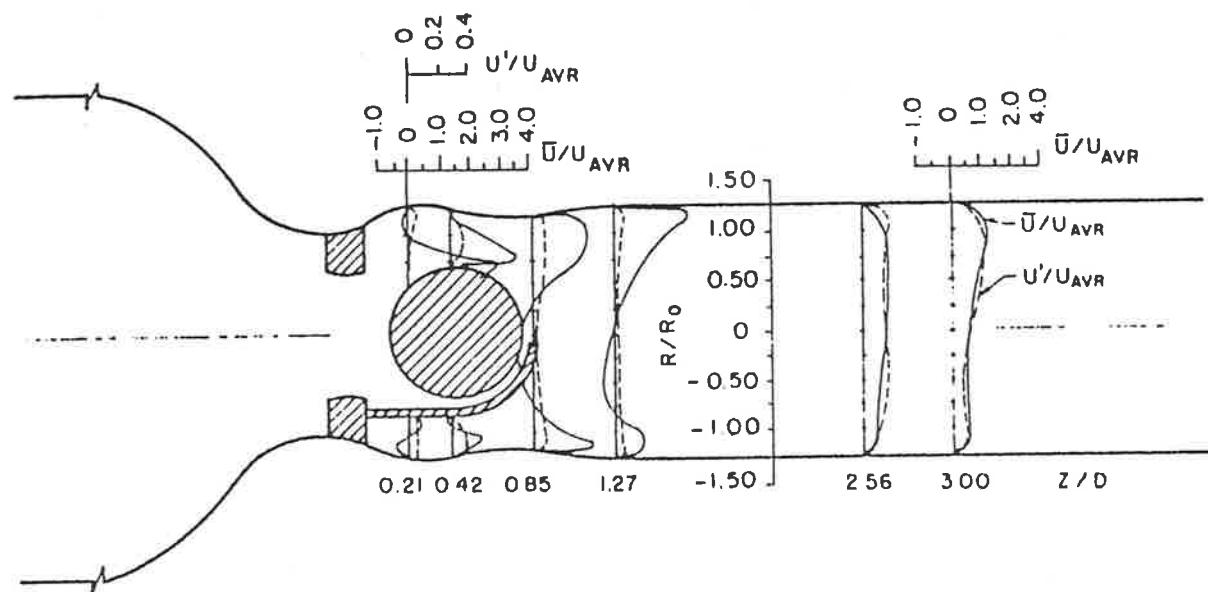


Figure 6.6.9

Experimentally measured mean axial velocity and turbulence profiles in the vicinity of a Starr-Edwards 1260-11A caged-ball prosthesis (Figliola and Mueller, 1981).

differ somewhat to those computed upstream at $z = 70\text{ mm}$. The peak velocities now occur a little further away from the ball surface. This is in good agreement with *in vitro* measurements made at the same location (Figure 6.6.9).

Steady flow rate $\text{cm}^3 \text{ sec}^{-1}$	Computed by Model III cm sec^{-1}	Measured by Yoganathan <i>et al</i> (1979) cm sec^{-1}
167	55	64
333	109	114
417	135	138

Table 6.6.2

Comparison of computed and experimentally measured maximum axial velocities approximately 2 mm downstream of ball occluder.

The $z = 88\text{ mm}$ location (Figure 6.6.6) was chosen to allow a direct comparison between the computed axial velocity profiles and the experimentally determined values at a position approximately 2 mm downstream from the ball occluder. This site corresponds closely to the location of the velocity profile measured by Yoganathan *et al* (1979) at $X = 22\text{ mm}$ (Figure 6.6.8). Even though the valve sizes differ slightly in the two examples (Starr-Edwards 1260 11-A and 1260 12-A) and the cage struts are not included in the numerical model, there is fair agreement at the same flow rates between the maximum axial velocities computed and those measured (Table 6.6.2).

However, the overall shape of the computed profiles at $z = 88\text{ mm}$ differ somewhat to experiments, particularly in the regions near the aortic wall and across the main separation zone. It should be pointed out that Yoganathan *et al* (1979) plotted zero values for the axial velocities in the stagnant zone, but in experiments the velocities actually oscillated between -2 and 2 cm sec^{-1} . Also, in Figure 6.6.8

the data points that appear to lie on the vertical axes correspond to velocity measurements made at distances of 0.034 mm from the flow channel walls.

Nevertheless, it is clear that the shape of the profiles remain very different in the near wall and separation zone regions. Even though the position of the maximum axial velocities predicted through this cross section lie about 3 mm from the aortic wall compared with approximately 2 mm in experiments, the the shape of the profiles between this location and the channel wall are significantly different in the two cases. The Model III velocity profile decreases rapidly towards zero at the wall whereas the measured profile has relatively high values which are only marginally lower at the flow channel boundary (0.034 mm). A similar profile, albeit asymmetric, was also obtained by Figliola and Mueller (1981) for this location (Figure 6.6.9). The computed velocity gradient in this region is clearly smaller than that indicated by measurements. A direct consequence of this result is that much smaller wall shear stresses would be predicted by Model III than those obtained from velocity measurements. The presence of cage struts near the $z = 88\text{ mm}$ location serve to lessen the magnitude of the velocities in this region of the main separation zone. By not being included in the numerical model, a direct comparison to experimental velocity values in this region is pointless.

The computed axial velocity profile at the $z = 100\text{ mm}$ cross section (Figure 6.6.6) shows a reduction in both the maximum and reverse velocities compared to the $z = 88\text{ mm}$ values. The maximum axial velocity computed here was 99 cm sec^{-1} located 4 mm from the aortic wall. The largest reverse flow velocity was -38 cm sec^{-1} occurring at the centreline. No experimental data is available for this cross section.

By $z = 120\text{ mm}$ (Figure 6.6.7) the maximum computed velocity is 82 cm sec^{-1}

at about 7 mm from the aortic wall. Furthermore, reverse velocities are no longer present. Instead a velocity defect (dip in the centre of the velocity profile) is evident near the centreline. Velocity profiles were measured by Yoganathan *et al* (1979) near this region ($X = 42\text{ mm}$ in Figure 6.6.8). They also observed a profile defect but the location and magnitude of the axial velocities differ from the computed values. The measured velocities are about 20 % larger than numerical values with the maximum values occurring about 2 mm from the flow channel wall.

At $z = 240\text{ mm}$ the velocity defect remains present but is now considerably smaller across all flow rates. No attempt was made to numerically force a fully developed turbulent velocity profile for a pipe along the outflow boundary.

Computed wall shear stress profiles are shown in Figure 6.6.10 for $Re = 4000$. Upstream of the sewing ring the wall shear stress remains below 3 N m^{-2} . The shear stress increases rapidly as the flow encounters the primary valve orifice reaching a maximum of 138.3 N m^{-2} at the sewing ring tip. The wall shear decreases rapidly downstream of the sewing ring but elevated levels are again observed along the sinus boundary. Along this boundary the wall shear stress attains a negative peak of -8.1 N m^{-2} which is a direct consequence of the velocities in the sinus separation zone. Further downstream, a broad region of elevated wall stress is seen along the aortic boundary corresponding to where the flow impinges upon the wall (clearly illustrated in Figure 6.6.10). The peak level of computed aortic wall shear stress is 30.7 N m^{-2} . The maximum computed stress along the occluder wall was -63.2 N m^{-2} for $Re = 4000$ at a location corresponding to $\theta = 64^\circ$. The level of stress rises and falls rapidly on either side of this peak (Figure 6.6.10). The maximum computed wall shear stress values for the other Reynolds numbers are presented in Table 6.6.1. Endothelial cells are damaged at stresses of about

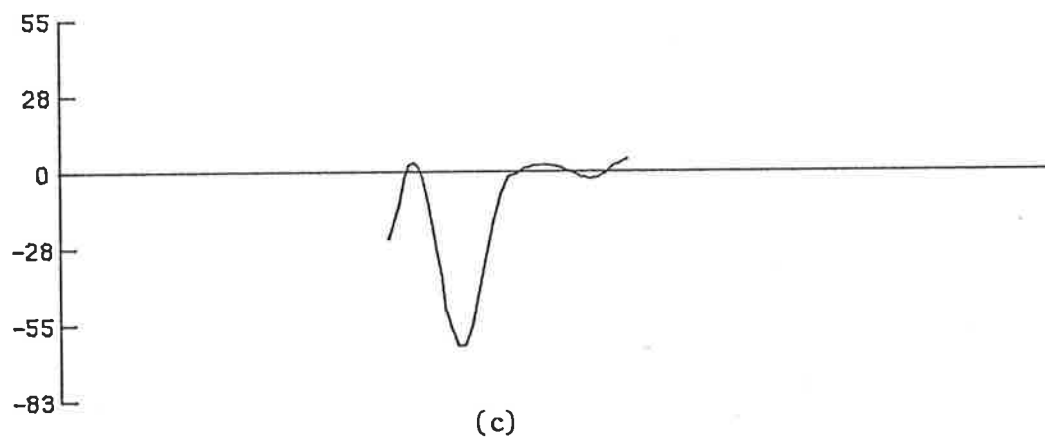
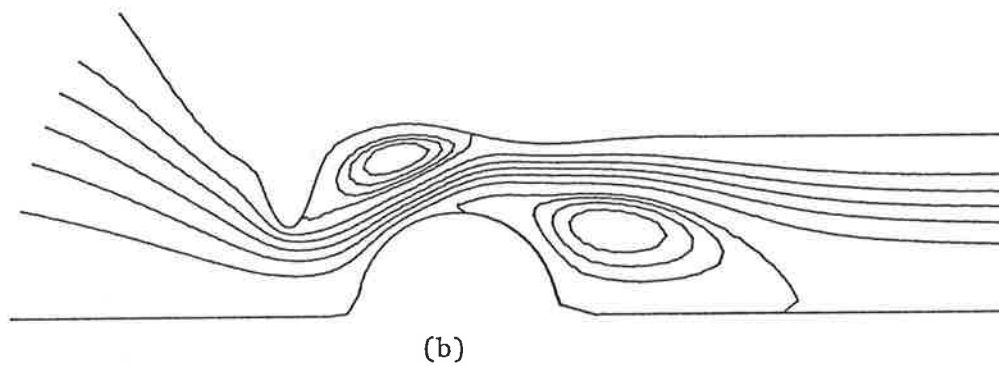
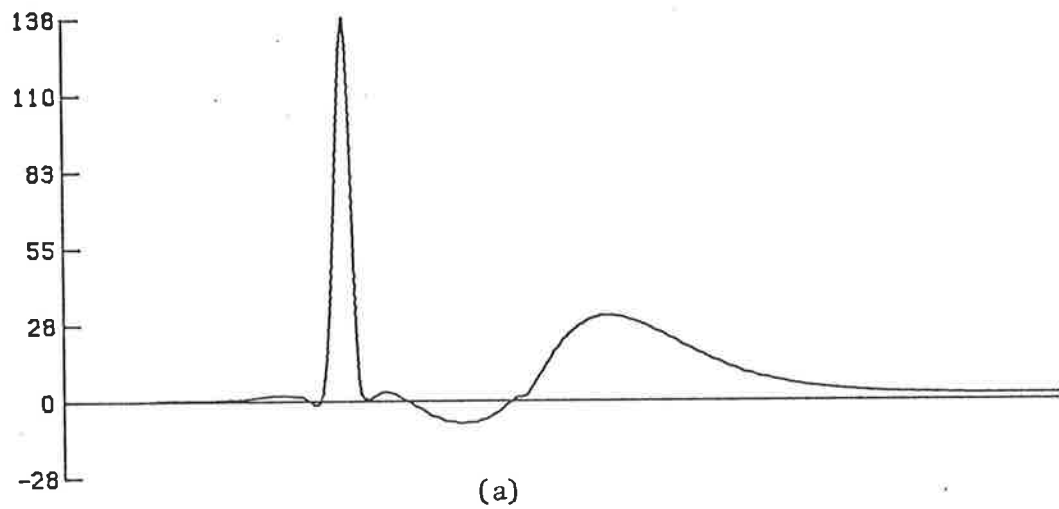


Figure 6.6.10

- (a) Computed aortic wall shear stress ($N\ m^{-2}$);
 (b) corresponding streamlines; and
 (c) occluder wall shear stress ($N\ m^{-2}$) for $Re = 4000$.

40 N m^{-2} and erode away at about 95 N m^{-2} (Table 2.4.1). The present numerical results suggest that endothelial damage will only occur under elevated flow rates.

However, the levels of wall shear stress predicted with Model III are much lower than values reported in experimental measurements. For example, at a steady flow rate of $22.8 \text{ litres min}^{-1}$ ($Re = 2800$) Model III gives a maximum aortic wall shear stress of 17.8 N m^{-2} . Figliola and Mueller (1981) determined wall shear stresses by a method described earlier in Chapter 5. At comparable flow rates ($23.4 \text{ litres min}^{-1}$) they found maximum aortic wall stresses of 82.7 and 59.9 N m^{-2} along the *inside* and *outside* walls, respectively. These values are considerably higher than wall stresses reported here. Also, Yoganathan *et al* (1979) experimentally determined wall shear stresses up to 151.0 N m^{-2} for the Starr-Edwards 1260-12A ball valve at a steady flow rate of $25.0 \text{ litres min}^{-1}$. At the same flow rate ($Re = 3077$), Model III gives a value of only 19.5 N m^{-2} for the Starr-Edwards 1260-11A prosthesis. Clearly, the Model III numerical solutions for peak wall shear stress are about 70 to 80 % lower than the values determined by Figliola and Mueller (1981), and almost 90 % lower than values obtained by Yoganathan *et al* (1979). In Chapter 5, differences of the same order were also observed between the Model II numerical solutions and *in vitro* measurements.

The distribution of turbulent shear stress in flow is shown in Figure 6.6.11 for $Re = 2800$, 4000 and 6000 . Elevated levels of stress are observed in many regions of the flow field but the largest values occur in the vicinity of the sewing ring tip, along the upstream face of the ball occluder, and in a small area of the sinus separation zone near its reattachment to the aortic wall. The maximum turbulent shear stress in the sinus separation zone is 35.9 N m^{-2} for $Re = 4000$. For $Re = 6000$ a maximum shear stress of 101.7 N m^{-2} is computed at this location. A negative

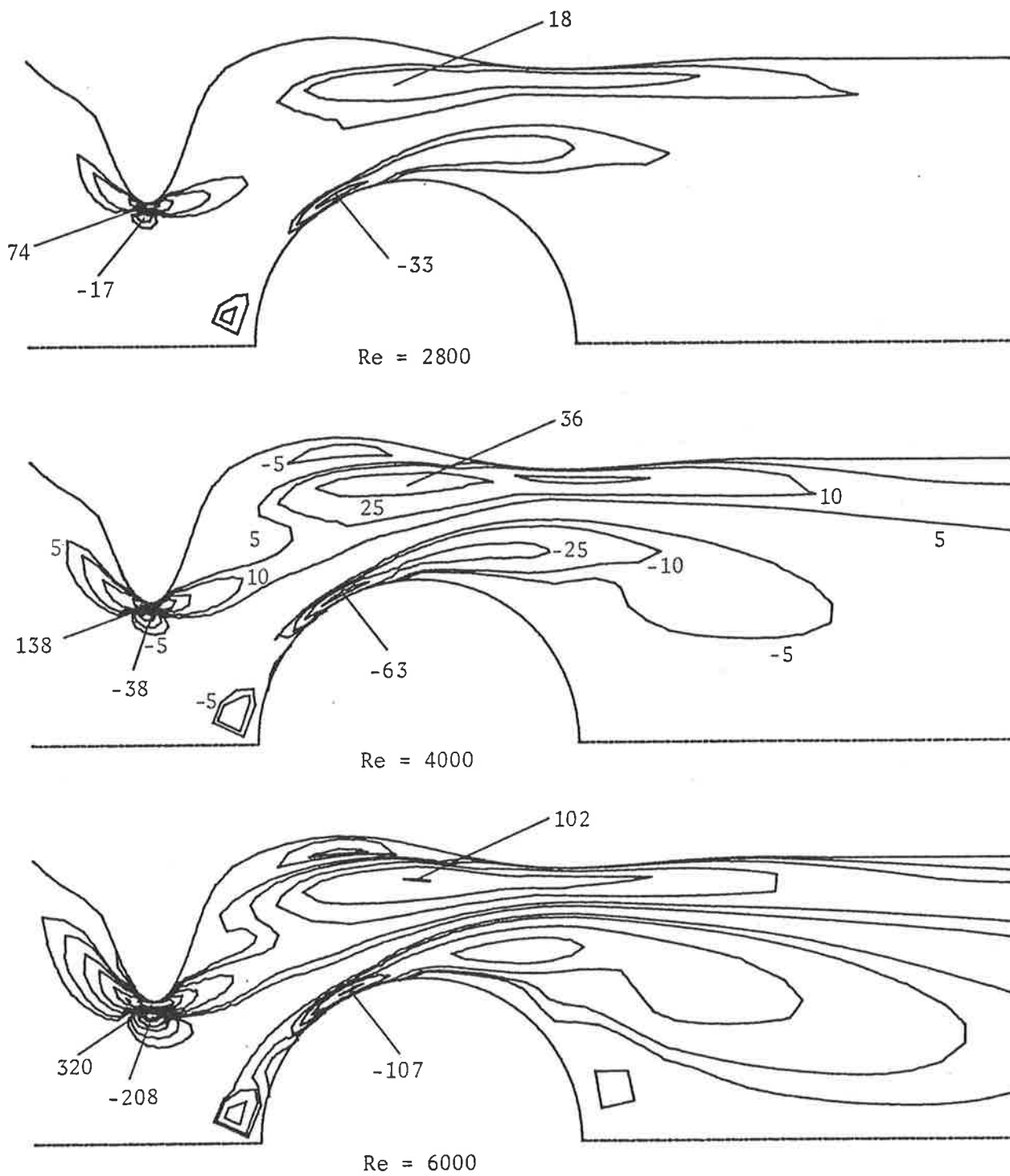


Figure 6.6.11

Computed turbulent shear stress ($N\ m^{-2}$) for $Re = 2800, 4000$ and 6000 .

peak occurs close to the sewing ring tip where the stress is -38.2 N m^{-2} . This peak increases to -208.1 N m^{-2} for $Re = 6000$. The values of turbulent shear stress for the sinus zone and negative peak are given in Table 6.6.1 for the other Reynolds numbers. In the large separation zone elevated stresses were predominant in an area alongside the separating streamline — a region of large velocity gradients. The peak shear stress here was -29.4 N m^{-2} at $Re = 4000$ and occurred just outside the separation zone.

Turbulent stresses of the same order of magnitude have been determined from *in vitro* experiments performed by Figliola and Mueller (1981). They measured bulk flow shear stresses of up to 18.0 N m^{-2} in the annular flow regions of a 1260-11A ball prosthesis. This is somewhat greater than the Model III computed shear levels of around 10 N m^{-2} at $Re = 2800$ indicated in Figure 6.6.11. Yoganathan *et al* (1979) did not report on any direct measurements of turbulent shear stress in flow. Instead, only estimates of shear stresses in the wake behind a sphere were presented so as to understand more about the turbulent shears that are likely to occur in the flow in the near vicinity of an aortic prosthesis. Typically, the estimated magnitude of the shear forces in this region were of the order of 100 N m^{-2} .

During peak systole under pulsatile flow conditions, Woo and Yoganathan (1985) measured values of the mean turbulent shear stress up to 120 N m^{-2} downstream of a Starr-Edwards caged-ball valve. By using axial velocity measurements, the estimated turbulent shear stresses were of the order of 300 to 500 N m^{-2} in the annular flow region. Even if one assumes that turbulent shear stresses under pulsatile flow conditions are larger than steady flow measurements by a factor of two (Chandran *et al*, 1983), the shear stresses computed with Model III are still anywhere up to 80 % lower than experimentally determined values.

A point worth noting is the difference between the turbulent shear stress contour configurations given in Figure 6.6.11 and those presented in a laminar study for a similar valve design (Idelsohn *et al*, 1985). Indeed, when only the molecular viscosity effects were taken into account in the present study the shear stress contours closely resembled the laminar plots. The importance of incorporating a turbulence model in numerical investigations is further emphasized when comparison is made to the levels of stress computed with only a laminar model. Idelsohn *et al* (1985) presented laminar model stress predictions for a caged-ball prosthesis which are about 60 % lower than levels reported by Model III at comparable flow rates.

Physiologically, damage to platelets is likely to occur with stresses of 10 to 50 N m^{-2} as indicated in Table 2.4.1. The present numerical shear stress results, albeit smaller than *in vitro* measurements, indicate that platelet damage will possibly occur in several regions of the flow during systole, especially during high flow rates (e.g. $Re = 6000$). Also, sub-haemolytic injury to adhered red blood cells from elevated stresses is quite likely at systolic flow rates approaching those experienced during rest.

One of the interesting results from the Model III study arises from the analysis of the maximum computed turbulent shear stress at several important flow field locations. For example, in Figure 6.6.12 a logarithmic graph is given for computed turbulent shear stress *versus* flow rate at the sewing ring tip. The computed data closely follows a straight line. Similar relationships were also found for the turbulent stress observed at other locations such as the occluder wall, the aortic wall, the sinus wall and the sinus separation zone. By measuring the slope and *y*-intercept of the line of best fit to the data, coefficients were obtained for a simple exponential

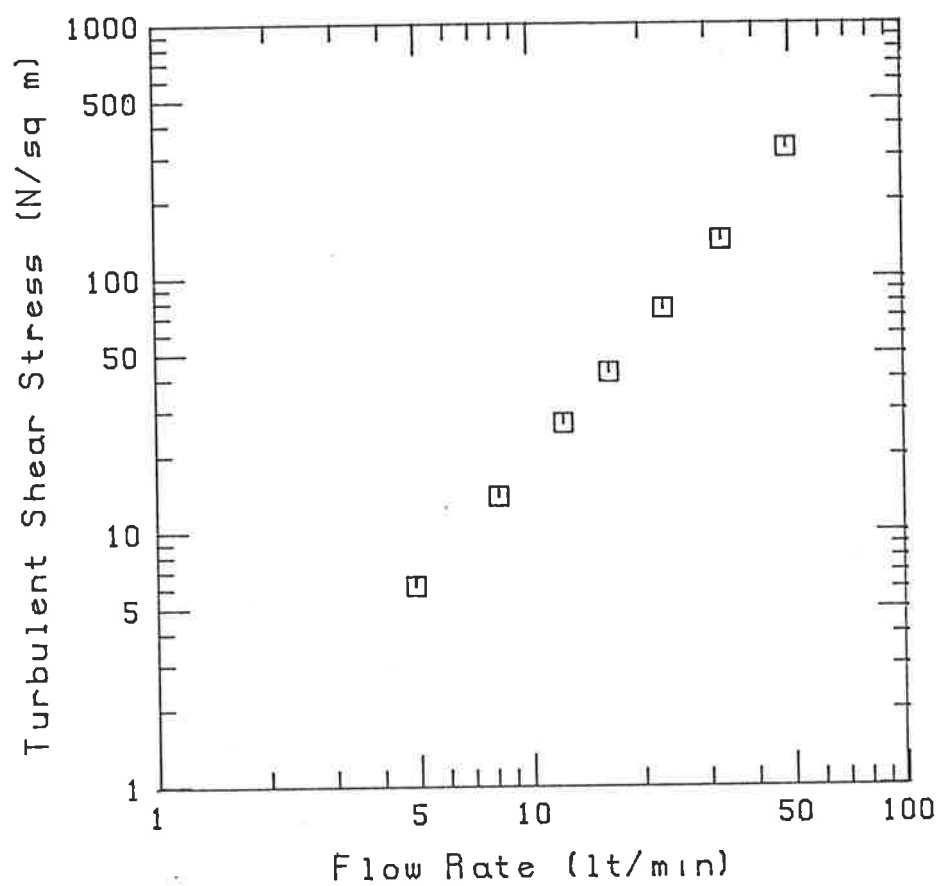


Figure 6.6.12

Computed turbulent shear stress ($N\ m^{-2}$) at the sewing ring tip at various flow rates.

slope =	1.4857	1.5112	2.3072	1.7040	1.6995
y Intercept =	0.5725	-0.9356	-2.1596	0.4498	1.3563

A =	0.1773	0.0392	0.0115	0.1568	0.3882
B =	1.4857	1.5112	2.3072	1.7040	1.6995

steady flow rate (litres/min)	aortic wall	sinus wall	sinus zone	occluder wall	sewing ring wall
10	5.4	1.3	2.3	7.9	19.4
12	7.1	1.7	3.6	10.8	26.5
14	8.9	2.1	5.1	14.1	34.4
16	10.9	2.6	6.9	17.7	43.2
18	13.0	3.1	9.1	21.6	52.8
20	15.2	3.6	11.6	25.8	63.1
22	17.5	4.2	14.4	30.4	74.2
24	19.9	4.8	17.6	35.3	86.0
26	22.4	5.4	21.2	40.4	98.6
28	25.0	6.0	25.2	45.8	111.8
30	27.7	6.7	29.5	51.6	125.7
32	30.5	7.4	34.3	57.6	140.3
34	33.4	8.1	39.4	63.8	155.5
36	36.4	8.8	45.0	70.4	171.4
38	39.4	9.6	50.9	77.1	187.9
40	42.5	10.3	57.3	84.2	205.0
42	45.7	11.1	64.2	91.5	222.7
44	49.0	11.9	71.4	99.0	241.0
46	52.4	12.8	79.1	106.8	260.0
48	55.8	13.6	87.3	114.9	279.4
50	59.3	14.5	95.9	123.1	299.5

Table 6.6.3

Predicted turbulent shear stress ($N\ m^{-2}$) at various flow rates.

relationship, namely

$$\sigma_{rz} = A \exp(B)$$

where σ_{rz} is the computed turbulent shear stress. Values for A and B at different locations are given in Table 6.6.3 together with predicted stress levels at various flow rates. Table 6.6.3 indicates that damage to the aortic wall endothelial lining will occur when peak systolic flow rates exceed $38 \text{ litres min}^{-1}$. Platelets may be damaged in the sinus separation zone at flow rates as low as $20 \text{ litres min}^{-1}$. At higher flow rates platelets will be damaged in many regions of the flow, particularly in the vicinity of the sewing ring and the upstream face of the occluder. Red blood cells may sustain damage in the proximity of the sewing ring tip if the flow rate exceeds $34 \text{ litres min}^{-1}$. These thresholds are lowered further when bearing in mind Model III's tendency to underpredict turbulent shear stresses.

Wurzinger *et al* (1985a) discuss the flow conditions that can lead to thrombogenesis (see Section 2.4.2). In the present study these flow conditions are shown to prevail at a number of locations. In the sinus recirculation zone predilection sites for thrombotic deposition occur near the reattachment point and the downstream face of the sewing ring. In the larger zone, the downstream side of ball occluder is identified as a region of potential thrombus formation.

The numerical streamline and velocity solutions compare fairly well with experimental measurements made by Figliola and Mueller (1981), Yoganathan *et al* (1979), and Woo and Yoganathan (1985). However, the levels of turbulent shear stress reported for Model III remain lower than *in vitro* measurements. Model III is based on the standard $k - \epsilon$ turbulence model. Among several other short comings (see Section 3.3), this model is known to underpredict the effects of velocity fluctua-

tions. A direct consequence is the underprediction of Reynolds stress levels observed here. Speziale (1986) successfully addressed this problem for turbulent channel flow by introducing an additional model term. Correct physical forms of secondary flows in square-ducts were predicted. However, for general turbulent separating flows further investigation is required. Furthermore, the hybrid central/upwind differencing scheme used to numerically solve the model equations is known to introduce artificial viscosities and reduce velocity gradients. The extent to which these effects influence the solutions is unknown.

6.7 Conclusions

Numerical solutions are obtained for steady turbulent flow through a caged-ball valve at several physiologically relevant flow rates. Regions of separated flow, high and low velocity, and elevated turbulent shear stress are clearly identified. The results indicate a high potential for thrombotic deposition on the downstream faces of the sewing ring and the ball occluder (i.e. cage apex). The numerical velocity and streamline solutions compare favourably with experimental measurements and observations. However, the turbulent shear stress levels are typically up to 80 % lower than measured values. A weakness in the $k - \epsilon$ turbulence model appears responsible for the underprediction of Reynolds stresses. Coefficients are given for a simple exponential relationship between the peak computed turbulent shear stress and the steady flow rate at several important locations in the flow field. The numerical solutions indicate possible damage to endothelial cells at high flow rates along a section of the aortic wall corresponding to where the flow impinges. Damage to platelets will occur at high flow rates near the sewing ring tip, the upstream face of the ball occluder and in the sinus separation zone. Damage to red blood cells in flow may occur near the tip of the sewing ring at very high flow rates.

CHAPTER 7

CONCLUSION

7.1 Conclusion

The three numerical models reported in this study provide detailed quantitative descriptions for the steady flow characteristics of caged-disk and caged-ball prosthetic heart valves. The first model, Model I, is by way of comparison to Models II and III very simple. It considers laminar flow through a Kay-Shiley caged-disk valve at several Reynolds numbers. The results indicate the potential effectiveness of such computational studies, but the model is limited in application because of the laminar flow assumption. Model II also considers flow through a Kay-Shiley caged-disk valve except that turbulence is now incorporated into the model. The TEACH-T computer code is employed to solve the system of equations. The numerical solutions differ somewhat from those in Model I, but are more agreeable with experimental measurements and observations. Model III is the most elaborate of the three models. It considers turbulent flow through a Starr-Edwards caged-ball prosthesis in an aortic-shaped chamber. A boundary-fitted coordinate system is generated and the governing flow equations are conveniently solved on a rectangular transformation plane. The behaviour of the model is generally consistent with experimental observations, although the predicted levels of turbulent shear stress are lower than measured values. Regions of slow moving flow, high fluid stress and elevated wall shear are identified. Coefficients are determined from the numerical solutions for a simple power-law relationship between the peak steady flow rate and the level of turbulent shear stress at several important flow locations. In all three models, correlations are made between the computed results and the potential for

pathological problems.

7.2 Clinical Significance of the Present Study

This study presents for the first time solutions to a numerical model for steady turbulent flow through a Starr-Edwards caged-ball prosthesis in an aortic-shaped chamber. This valve has an estimated 122,000 worldwide implants, thus underlining the practical importance of this research.

Using numerical techniques to solve complex mathematical models has provided valuable and detailed quantitative data for velocities and stress fields in the vicinity of prosthetic heart valves — a totality currently unattainable through physical simulators.

Ghista (1980) pointed out the “unlimited potential” of computer design and graphics for the biomechanical design of prostheses and surgical procedures. Accordingly, the long term application of this form of research is seen both as an effective design tool and to assist cardiac surgeons in the selection of a suitable valve implant for individual patients.

7.3 Recommendations for Future Study

The complete understanding of the complex fluid dynamics of prosthetic heart valves and associated pathological complications requires more than a steady flow and rigid boundary analysis. Indeed, the effects of pulsatile flow and elastic wall boundaries will strongly influence the nature of flow through a valve. An investigation of unsteady flow effects has already been commenced by the author.

Furthermore, the $k - \epsilon$ turbulence model can be readily modified to account for

streamline curvature effects. The implementation of third-order accurate upstream differencing methods will also improve the quality of the solutions. Both of these approaches warrant further investigation, and details of such formulations are given by Leschziner and Rodi (1981).

The application of mathematical modelling and computational fluid dynamic techniques for the evaluation of the flow characteristics of prosthetic heart valves has and will continue to provide much valuable information.

APPENDIX A

NOTES ON THE NATURE AND AVAILABILITY OF TEACH-T COMPUTER CODE

1. Description

TEACH-T is a general-purpose computer program which calculates steady laminar or turbulent single-phase, subsonic, two-dimensional flows of all kinds, although it is mainly intended for 'elliptic' or 'separated' flow application.

The code embodies an implicit finite-difference algorithm for solving the velocity-pressure formulation of the conservation equations of mass and axial and radial momentum on a variable-spacing Cartesian or cylindrical-polar computing mesh. For turbulent flow applications, additional equations are solved for the turbulence energy and its dissipation rate, which comprise the dependent variables of the turbulence model employed. The associated wall boundary conditions are applied via 'wall functions' based on equilibrium one-dimensional flow theory.

The code is written in standard FORTRAN IV and has a modular structure which facilitates the inclusion of additional dependent variables, where required. The coding has been performed with ease of understanding, rather than computational efficiency, as the main priority.

2. Availability

Although TEACH-T was originally conceived as an instructional code to accompany courses on Computational Fluid Mechanics taught by the originators and their colleagues, it has been made available at nominal charge to others wishing to acquire it, on the understanding that it is not subject to the guarantees and service that are often attached to commercially-oriented codes.

The code is supplied in card-deck form, together with the following:

- a listing
- a sample output
- a hand-written user manual

Details of charges and postage costs are available by writing to the following address:

Dr. A.D. Gosman
Imperial College of Science and Technology
Department of Mechanical Engineering
Exhibition Road
London SW7 2BX

APPENDIX B

DERIVATIVES AND VECTORS IN THE TRANSFORMED PLANE

This appendix contains a brief set of relations in the transformed (ξ, η) plane. A comprehensive account is provided by Thompson *et al* (1977). The following function definition is applicable throughout this appendix:

$\phi(z, r, t)$ – a twice continuously differentiable scalar function of z , r and t .

$$J = z_{\xi}r_{\eta} - z_{\eta}r_{\xi} \quad (B.1)$$

$$\alpha = z_{\eta}^2 + r_{\eta}^2 \quad (B.2)$$

$$\beta = z_{\xi}z_{\eta} + r_{\xi}r_{\eta} \quad (B.3)$$

$$\gamma = z_{\xi}^2 + r_{\xi}^2 \quad (B.4)$$

$$\sigma = \frac{r_{\xi}(\alpha z_{\xi\xi} - 2\beta z_{\xi\eta} + \gamma z_{\eta\eta}) - z_{\xi}(\alpha r_{\xi\xi} - 2\beta r_{\xi\eta} + \gamma r_{\eta\eta})}{J} \quad (B.5)$$

$$\tau = \frac{z_{\eta}(\alpha r_{\xi\xi} - 2\beta r_{\xi\eta} + \gamma r_{\eta\eta}) - r_{\eta}(\alpha z_{\xi\xi} - 2\beta z_{\xi\eta} + \gamma z_{\eta\eta})}{J} \quad (B.6)$$

For an orthogonal coordinate system $\beta = 0$.

Derivative Transformations

$$\phi_z \equiv \left(\frac{\partial \phi}{\partial z} \right)_{r,t} = (r_\eta \phi_\xi - r_\xi \phi_\eta) / J \quad (B.7)$$

$$\phi_r \equiv \left(\frac{\partial \phi}{\partial r} \right)_{z,t} = (z_\xi \phi_\eta - z_\eta \phi_\xi) / J \quad (B.8)$$

$$\begin{aligned} \phi_{zz} \equiv \left(\frac{\partial^2 \phi}{\partial z^2} \right)_{r,t} &= (r_\eta^2 \phi_{\xi\xi} - 2r_\xi r_\eta \phi_{\xi\eta} + r_\xi^2 \phi_{\eta\eta}) / J^2 \\ &\quad + [(r_\eta^2 r_{\xi\xi} - 2r_\xi r_\eta r_{\xi\eta} + r_\xi^2 r_{\eta\eta})(z_\eta \phi_\xi - z_\xi \phi_\eta) \\ &\quad + (r_\eta^2 z_{\xi\xi} - 2r_\xi r_\eta z_{\xi\eta} + r_\xi^2 z_{\eta\eta})(r_\xi \phi_\eta - r_\eta \phi_\xi)] / J^3 \end{aligned} \quad (B.9)$$

$$\begin{aligned} \phi_{rr} \equiv \left(\frac{\partial^2 \phi}{\partial r^2} \right)_{z,t} &= (z_\eta^2 \phi_{\xi\xi} - 2z_\xi z_\eta \phi_{\xi\eta} + z_\xi^2 \phi_{\eta\eta}) / J^2 \\ &\quad + [(z_\eta^2 r_{\xi\xi} - 2z_\xi z_\eta r_{\xi\eta} + z_\xi^2 r_{\eta\eta})(z_\eta \phi_\xi - z_\xi \phi_\eta) \\ &\quad + (z_\eta^2 z_{\xi\xi} - 2z_\xi z_\eta z_{\xi\eta} + z_\xi^2 z_{\eta\eta})(r_\xi \phi_\eta - r_\eta \phi_\xi)] / J^3 \end{aligned} \quad (B.10)$$

$$\begin{aligned} \phi_{zr} &= [(z_\xi r_\eta + z_\eta r_\xi) \phi_{\xi\eta} - z_\xi r_\xi \phi_{\eta\eta} - z_\eta r_\eta \phi_{\xi\xi}] / J^2 \\ &\quad + [z_\eta r_\eta z_{\xi\xi} - (z_\xi r_\eta + z_\eta r_\xi) z_{\xi\eta} + z_\xi r_\xi z_{\eta\eta}](r_\eta \phi_\xi - r_\xi \phi_\eta) / J^3 \\ &\quad + [z_\eta r_\eta r_{\xi\xi} - (z_\xi r_\eta + z_\eta r_\xi) r_{\xi\eta} + z_\xi r_\xi r_{\eta\eta}](z_\xi \phi_\eta - z_\eta \phi_\xi) / J^3 \end{aligned} \quad (B.11)$$

Vector Derivative Transformations

Laplacian:

$$\nabla^2 \phi = (\alpha \phi_{\xi\xi} - 2\beta \phi_{\xi\eta} + \gamma \phi_{\eta\eta} + \sigma \phi_\eta + \tau \phi_\xi) / J^2 \quad (B.12)$$

Gradient:

$$\vec{\nabla} \phi = [(r_\eta \phi_\xi - r_\xi \phi_\eta) \vec{i} + (z_\xi \phi_\eta - z_\eta \phi_\xi) \vec{j}] / J \quad (B.13)$$

Mazumdar, J., and Thalassoudis, K., (1983) A mathematical model for the study of flow through disc-type prosthetic heart valves.
Medical and Biological Engineering and Computing, v. 21 (4), pp. 400-409.

NOTE:

This publication is included on pages 158-167 in the print copy of the thesis held in the University of Adelaide Library.

It is also available online to authorised users at:

<http://dx.doi.org/10.1007/BF02442626>

Thalassoudis, K., and Mazumdar, J., (1984) Mathematical model for turbulent blood flow through a disk-type prosthetic heart valve.
Medical and Biological Engineering and Computing, v. 22 (6), pp. 529-536.

NOTE:

This publication is included on pages 168-175 in the print copy
of the thesis held in the University of Adelaide Library.

It is also available online to authorised users at:

<http://dx.doi.org/10.1007/BF02443866>

Thalassoudis, K., Mazumdar, J., Noye, B.J., and Craig, I.H., (1987) Numerical study of turbulent blood flow through a caged-ball prosthetic heart valve using a boundary-fitted co-ordinate system.

Medical and Biological Engineering and Computing, v. 25 (2), pp. 173-180.

NOTE:

This publication is included on pages 176-183 in the print copy of the thesis held in the University of Adelaide Library.

It is also available online to authorised users at:

<http://dx.doi.org/10.1007/BF02442847>

BIBLIOGRAPHY

- AU, A.D. and GREENFIELD, H.S. Computer graphics analysis of stresses in blood flow through a prosthetic heart valve. *Computers in Biology and Medicine*, Vol. 4, 279–291, 1975.
- AU, A.D. and GREENFIELD, H.S. Computer prediction of thrombogenic sites for a tilting-disc prosthetic heart valve. *Computers and Biomedical Research*, Vol. 10, 165–182, 1977.
- BAILEY, C.P. Surgical treatment of mitral stenosis. *Diseases of the Chest*, Vol. 15, 377–393, 1949.
- BELLHOUSE, B.J. and TALBOT, L. The fluid mechanics of the aortic valve. *Journal of Fluid Mechanics*, Vol. 35, 721–735, 1969.
- BERNE, R.M. and LEVY, M.N. *Cardiovascular Physiology*, 5th Edition, The C.V. Mosby Co., St. Louis, 1986.
- BJÖRK, V.O. A new tilting disc valve prosthesis. *Scandinavian Journal of Thoracic and Cardiovascular Surgery*, Vol. 3, 1–10, 1969.
- BLACKSHEAR, P.L. Hemolysis at prosthetic surfaces. In *Chemistry of biosurfaces*, Vol. 2, HAIR, M.L. (Ed.), Marcel Dekker, New York, 523–561, 1972.
- CHANDRAN, K.B., CABELL, G.N., KHALIGHI, B. and CHEN, C.-J. Laser anemometry measurements of pulsatile flow past aortic valve prostheses. *Journal of Biomechanics*, Vol. 16, No. 10, 865–873, 1983.
- DAVIES, C.W. An initial value approach to the production of discrete orthogonal coordinates. *Journal of Computational Physics*, Vol. 39, 164–178, 1981.
- ENGELMAN, M.S., MOSKOWITZ, S.E. and BORMAN, J.B. Computer simulation: A diagnostic method in comparative studies of valve prostheses. *Journal of Thoracic and Cardiovascular Surgery*, Vol. 79, 402–412, 1980.

FIGLIOLA, R.S. A study of the hemolytic potential of prosthetic heart valve flows based on local *in vitro* stress measurements. MS thesis, University of Notre Dame, Indiana, (1976).

FIGLIOLA, R.S. and MUELLER, T.J. Fluid stresses in the vicinity of disk, ball, and tilting disk prosthetic heart valves from *in vitro* measurements. *A.S.M.E. Journal of Biomechanical Engineering*, Vol. 99, 173-177, 1977.

FIGLIOLA, R.S. and MUELLER, T.J. On the hemolytic and thrombogenic potential of occluder prosthetic heart valves from *in vitro* measurements. *A.S.M.E. Journal of Biomechanical Engineering*, Vol. 103, 83-90, 1981.

FRIEDMAN, M. Laminar flow in a channel with a step. *J. Eng. Maths.*, Vol. 6, No. 3, 285-290, 1972.

FRY, D.L. Acute vascular endothelial changes associated with increased blood velocity gradients. *Circulation Research*, Vol. 22, 165-197, 1968.

GHISTA, D.N. Biomechanics: Traditions, academic and research set-ups, and some new horizons. *Proc. 2nd Int. Conf. Mechanics in Medicine and Biology*, Osaka, Japan, 5-7 June, 1980.

GLOVER, R.P. and DAVILA, J.C. The treatment of mitral insufficiency by the purse-string technique: Initial Clinical Application. *Journal of Thoracic Surgery*, Vol. 33, 75-101, 1957.

GOSMAN, A.D. and IDERIAH, F.J.K. TEACH-T: a general computer program for two-dimensional turbulent recirculating flows. Department of Mechanical Engineering, Imperial College, London, 1976.

HARKEN, D.E., SOROFF, H.S., TAYLOR, W.J., LEFEMINE, A.A., GUPTA, S.K. and LUNZER, S. Partial and complete prostheses in aortic insufficiency. *Journal of Thoracic and Cardiovascular Surgery*, Vol. 40, 744-762, 1960.

HELLUMS, J.D. and BROWN, C.H. III. Blood cell damage by mechanical forces. In *Cardiovascular fluid dynamics*, HWANG, N.H.C. and NORMANN, N.A. (Eds.), University Park Press, Baltimore, 799-823, 1977.

HERKES, W.H. and LLOYD, J.R. Experimental measurements of the flow-induced shear stress distribution in the vicinity of prosthetic heart valves. *A.S.M.E. Journal of Biomechanical Engineering*, Vol. 103, 267-274, 1981.

HUFNAGEL, C.A., HARVEY, W.P., RABIL, P.J. and McDERMOTT, T.F. Surgical correction of aortic insufficiency. *Surgery*, Vol. 35, 673-683, 1954.

HUFNAGEL, C.A. and CONRAD, P.W. Comparative study of some prosthetic valves for aortic and mitral replacement. *Surgery*, Vol. 57, No. 1, 205-210, 1965.

HUNG, T.C., HOCHMUCH, R.M., JOIST, J.H. and SUTERA, S.P. Shear-induced aggregation and lysis of platelets. *Trans. Am. Soc. Artif. Internal Organs*, Vol. 22, 285-290, 1976.

IDELSOHN, S.R., COSTA, L.E. and PONSO, R. A comparative computational study of blood flow through prosthetic heart valves using the finite element method. *Journal of Biomechanics*, Vol. 18, No. 2, 97-115, 1985.

JONES, W.P. and LAUNDER, B.E. The calculation of low-Reynolds number phenomena with a two-equation model of turbulence. *International Journal of Heat and Mass Transfer*, Vol. 16, 1119-1130, 1973.

KASTER, R.L. and LILLEHEI, C.W. A new cageless free-floating pivoting disc prosthetic heart valve: Design, development and evaluation. *Digest of the 7th International Conference on Medical and Biological Engineering*, Stockholm, 387, 1967.

KAY, E.B., SUZUKI, A., DEMANEY, M. and ZIMMERMAN, H.A. Comparison of ball and disc valves for mitral valve replacement. *American Journal of Cardiology*, Vol. 18, 504-514, 1966.

LAUNDER, B.E. and SPALDING, D.B. The numerical computation of turbulent flows. *Computer Methods in Applied Mechanics and Engineering*, Vol. 3, 269-289, 1974.

LESCHZINER, M.A. and RODI, W. Calculation of annular and twin parallel jets using various discretization schemes and turbulence-model variations. *A.S.M.E. Journal of Fluids Engineering*, Vol. 103, 352-360, 1981.

- LEVERETT, L.B., HELLUMS, J.D., ALFREY, C.P. and LYNCH, E.C. Red blood cell damage by shear stress. *Biophysics Journal*, Vol. 12, 257-273, 1972.
- McCLUNG, J.A., STEIN, J.H., AMBROSE, J.A., HERMAN, M.V. and REED, G.E. Prosthetic heart valves: A review. *Progress in Cardiovascular Diseases*, Vol. 26, No. 3, 237-270, 1983.
- McQUEEN, D.M. and PESKIN, C.S. Computer assisted design of pivoting disc prosthetic mitral valves. *Journal of Thoracic and Cardiovascular Surgery*, Vol. 86, 126-135, 1983.
- MOBLEY, C.D. and STEWART, R.J. On the numerical generation of boundary-fitted orthogonal curvilinear coordinate systems. *Journal of Computational Physics*, Vol. 34, 124-135, 1980.
- NEVARIL, C.G., HELLUMS, J.D., ALFREY, C.P. Jr. and LYNCH, E.C. Physical effects in red blood cell trauma. *American Institute of Chemical Engineers Journal*, Vol. 15, 707-711, 1969.
- NOYE, B.J. An introduction to finite difference techniques. *Numerical simulation of fluid flow. Proc. of an International Conference on the Numerical Simulation of Fluid Dynamical Systems*, Monash University, Melbourne, Australia, 1-112, 1976.
- PATANKAR, S.V. and SPALDING, D.B. A calculation procedure for heat, mass and momentum transfer in three-dimensional parabolic flows. *International Journal of Heat and Mass Transfer*, Vol. 15, 1787-1806, 1972.
- PATANKAR, S.V. Calculation of the flow field. In *Numerical heat transfer and fluid flow*, MINKOWYCZ, W.J. and SPARROW, E.M. (Eds.), Hemisphere, Washington, 113-137, 1980.
- PESKIN, C.S. Numerical analysis of blood flow in the heart. *Journal of Computational Physics*, Vol. 25, 220-252, 1977.
- PESKIN, C.S. The fluid dynamics of heart valves : experimental, theoretical, and computational methods. *Annual Review of Fluid Mechanics*, Vol. 14, 235-259, 1982.
- ROACHE, P.J. *Computational fluid dynamics*, Hermosa, Albuquerque, New Mexico, 1976.

ROBERTS, W.C. Choosing a substitute cardiac valve: type, size, surgeon. *The American Journal of Cardiology*, Vol. 38, 633-644, 1976.

RODI, W. *Turbulence models and their application in hydraulics — A state of the art review*, International Association for Hydraulic Research, Delft, 1980.

RUNCHAL, A.K. Convergence and accuracy of three finite difference schemes for a two-dimensional conduction and convection problem. *International Journal of Numerical Methods in Engineering*, Vol. 4, 541-550, 1972.

SCHLICHTING, H. *Boundary layer theory*, 6th Edition, McGraw-Hill, New York, 1968.

SCHOEN, F.J., TITUS, J.L. and LAWRIE, G.M. Bioengineering aspects of heart valve replacement. *Annals of Biomedical Engineering*, Vol. 10, 97-128, 1982.

SOUTTAR, H.S. The surgical treatment of mitral stenosis. *The British Medical Journal*, No. 2, 603-606, 1925.

SPALDING, D.B. A novel finite difference formulation for differential equations involving first and second derivatives. *International Journal of Numerical Methods in Engineering*, Vol. 4, 551-559, 1972.

SPEZIALE, G.S. On nonlinear $k - \ell$ and $k - \epsilon$ models of turbulence. George W. Woodruff School of Mechanical Engineering, Georgia Institute of Technology, Atlanta, GA 30332, 1986.

STARR, A. and EDWARDS, M.L. Mitral replacement: Clinical experience with a ball-valve prosthesis. *Annals of Thoracic Surgery*, Vol. 154, 726-740, 1961.

STEIN, P.D. and SABBAH, H.N. Turbulent blood flow in the ascending aorta of humans with normal and diseased aortic valves. *Circulation Research*, Vol. 39, No. 1, 58-65, 1976.

STEVENSON, D.M. and YOGANATHAN, A.P. Numerical simulation of steady turbulent flow through trileaflet aortic valve heart valves — I. Computational scheme and methodology. *Journal of Biomechanics*, Vol. 18, No. 12, 899-907, 1985a.

STEVENSON, D.M. and YOGANATHAN, A.P. Numerical simulation of steady turbulent flow through trileaflet aortic valve heart valves — II. Results on five models. *Journal of Biomechanics*, Vol. 18, No. 12, 909–926, 1985b.

SUTERA, S.P. Flow-induced trauma to blood cells. *Circulation Research*, Vol. 41, No. 1, 2–8, 1977.

SUTERA, S.P. and MEHRJARDI, M.H. Deformation and fragmentation of human RBC in turbulent shear flow. *Biophysics Journal*, Vol. 15, 1–10, 1975.

THOMPSON, J.F., THAMES, F.C. and MASTIN, C.W. Boundary-fitted curvilinear coordinate systems for solution of partial differential equations on fields containing any number of arbitrary two-dimensional bodies. NASA CR-2729, 1977.

THOMPSON, J.F. and WARSI, Z.U.A. Boundary-fitted coordinate systems for numerical solution of partial differential equations — A review. *Journal of Computational Physics*, Vol. 47, 1–108, 1982.

UNDERWOOD, F.N. and MUELLER, T.J. Numerical study of the steady axisymmetric flow through a disk-type prosthetic heart valve in a constant diameter chamber. *A.S.M.E. Journal of Biomechanical Engineering*, Vol. 99, 91–97, 1977.

UNDERWOOD, F.N. and MUELLER, T.J. Numerical study of the steady axisymmetric flow through a disk-type prosthetic heart valve in an aortic shaped chamber. *A.S.M.E. Journal of Biomechanical Engineering*, Vol. 101, 198–204, 1979.

WADA, J. Knotless suture method and Wada hingeless valve. *Japan Journal of Thoracic Surgery*, Vol. 15, 88–94, 1967.

WOO, Y.-R. and YOGANATHAN, A.J. *In vitro* pulsatile flow velocity and turbulent shear stress measurements in the vicinity of mechanical aortic heart valve prostheses. *Life Support Systems*, Vol. 3, 283–312, 1985.

WURZINGER, L.J., BLASBERG, P. and SCHMID-SCHÖNBEIN, H. Towards a concept of thrombosis in accelerated flow: Rheology, fluid dynamics, and biochemistry. *Biorheology*, Vol. 22, 437–450, 1985a.

WURZINGER, L.J., OPITZ, R., WOLF, M. and SCHMID-SCHÖNBEIN, H. Shear induced platelet activation — A critical reappraisal. *Biorheology*, Vol. 22, 399–413, 1985b.

YANG, W.J. and WANG, J.H. Numerical analysis of turbulent flows through a disk-type prosthetic heart valve. *Proc. 2nd Int. Conf. Mechanics in Medicine and Biology*, Osaka, Japan, 142-143, 5-7 June, 1980.

YANG, W.J. and WANG, J.H. Turbulent flows through a disk-type prosthetic heart valve. *A.S.M.E. Journal of Biomechanical Engineering*, Vol. 105, 263-267, 1983.

YOGANATHAN, A.P., CORCORAN, W.H. and HARRISON, E.C. Wall shear stress measurements in the near vicinity of aortic heart valves. *Journal of Bioengineering*, Vol. 2, 369-379, 1978.

YOGANATHAN, A.P., CORCORAN, W.H. and HARRISON, E.C. *In vitro* velocity measurements in the vicinity of aortic prostheses. *Journal of Biomechanics*, Vol. 12, No. 2, 135-152, 1979.

YOGANATHAN, A.P., REAMER, H.H., CORCORAN, W.H., HARRISON, E.C., SHULMAN, I.A. and PARNASSUS, W. The Starr-Edwards aortic ball valve: Flow characteristics, thrombus formation and tissue overgrowth. *Artificial Organs*, Vol. 5, No. 1, 6-17, 1981.