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Numerical Simulation of Flows In Designed Sleeve Models At Distal Anastomoses

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SCHOOL OF MECHANICAL AND AEROSPACE ENGINEERING

A thesis submitted to the Nanyang Technological University
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Abstract

Diminished blood flow as a result of coronary atherosclerosis is one of the most common causes of cardiovascular disease. Coronary artery bypass graft (CABG) surgery is frequently performed to restore blood flow by bridging the stenosed area with a replacement vessel. CABG however cannot warrant a lifetime solution due to possible graft failure. Failure mode such as intimal hyperplasia has been known to occur as a result of poor hemodynamics. Surgeons have reported improvement in patency rates using interposition vein cuffs for lower limb bypasses. The focus of this research is to design and evaluate sleeve models with cuff-like geometries of varying volume as well as out-of-plane and bypass angles. Sleeve models exhibiting optimized hemodynamics can be used as mechanical connectors between the bypass graft and host artery at the distal anastomosis, hence eliminating the need for suturing.

Five models of varying geometries and volumes were designed and investigated. In steady flow, fluid motion in the baseline model (Model 1) resembled flow patterns found in conventional distal anastomosis while Model 2 with a height increment showed similar trends with the exception of modulated shear stresses at the toe region. Fluid motion in Model 3 (diameter increment) was generally slower, exhibiting a source-like structure within the sleeve volume. Flow in models with combined height and diameter increment (Models 4 and 5) revealed characteristics observed separately in Models 2 and 3. Fluid motion in pulsatile conditions was more dynamic. A vortex formed near peak flow provided improved mixing by entraining blood into areas of low velocity at the heel region. During deceleration, spatial mixing was further enhanced through movements of the vortex into the sleeve volume. The vortex was

observed to be fully suppressed by the arrival of the next acceleration phase, implying that fluid particles were washed out and the sleeve was re-perfused by blood entering the bypass graft from the aorta. Due to the biphasic nature of flow in coronary artery bypass grafts, the sleeve region was washed twice per cycle. Flow in Models 3 and 5 were characterized by presence of the source-like structure. The overall decreased fluid velocity would be detrimental as it increased exposure time of blood to the prosthetic material, thereby increasing possibilities of thrombus formation or deposition. It was concluded from the study that Model 1 was more desirable as a sleeve design due to improved hemodynamics which may inhibit or delay reocclusion time. For a further increase in volume, a height increment (i.e. Model 2) was recommended.

Studies were also performed to investigate effects of angular variation on hemodynamics. Out-of-plane angles of $\theta_{OP} = 30^\circ, 60^\circ$ and 90° were subject to steady flow conditions of $Re = 80$. It was found that an entrance length of $L_E = 9\text{mm}, 12\text{mm}$ and 14mm respectively were required for flow to stabilize before entering the sleeve. Effects of bypass angles ($\theta_B = 0^\circ, 15^\circ, 30^\circ, 45^\circ$ and 60°) were also studied. As θ_B was increased, peak shear stresses along the inner and outer walls were reduced and modulated. At low flow rate ($Re = 80$), no recirculation was observed for all values of θ_B at the distal artery entrance. However, when flow rate was increased to $Re = 250$, an inverse relationship exists between θ_B and the magnitude of recirculation. As bypass angle was increased, the recirculation was observed to decrease in magnitude and intensity. Such a phenomenon was opposite to that of the common end-to-side anastomosis models.

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List of Nomenclature

ϕ_f	- Value of ϕ convected through face f
ρ	- Density of fluid (kg/m ³)
μ	- Dynamic viscosity of fluid (Pa.s)
α	- Womersley number
ω	- Angular frequency (rad/s)
ϕ	- Scalar quantity
$\nabla\phi$	- Gradient of ϕ
S_ϕ	- Source of ϕ per unit volume
$(\nabla\phi)_n$	- Magnitude of $\nabla\phi$ normal to face f
β	- Under-relaxation factor
Δt	- Time step size
A	- Amplitude of pressure gradient (Pa/m)
$\vec{A}$	- Surface area vector
$\vec{A}_f$	- Area of face
a_p	- Coefficient values at the cell center
B_n	- Fourier coefficients
c_0 and c_1	- Cell centers
D_A	- Artery diameter (mm)
D_G	- Graft diameter (mm)
D_S	- Sleeve diameter (mm)
F	- External body force (N/m ³)
f	- Frequency (Hz)
H_S	- Sleeve height (mm)
J_f	- Mass flux through face f
J_0	- Bessel function of the first kind, order 0
J_1	- Bessel function of the first kind, order 1
k	- Derived constant for inlet length
L	- Inlet length (m)
L_D	- Distal host artery length (mm)
L_E	- Entrance length (mm)
L_G	- Graft length (mm)

L_P	- Proximal host artery length (mm)
L_S	- Sleeve length (mm)
N_{faces}	- Number of faces enclosing cell
θ_B	- Bypass angle (°)
θ_{OP}	- Out-of-plane angle (°)
P	- Static pressure (Pa)
P_f	- Face pressure (Pa)
Q	- Flow rate (m ³ /s)
Q_{AR}	- Aspect ratio
Q_{EAS}	- EquiAngle Skew
Q_{ESS}	- EquiSize Skew
Q_{mean}	- Mean flow rate (m ³ /s)
r	- Radius with respect to tube axis (m)
R	- Radius of tube (m)
Γ_ϕ	- Diffusion coefficient for ϕ
Re	- Reynolds number
Re_{mean}	- Mean Reynolds number
$Re_{peak (critical)}$	- Critical peak Reynolds number
St	- Strouhal number
T	- Period
T_S	- Sleeve thickness (mm)
ν	- Kinematic viscosity of fluid (s ⁻¹ m ⁻¹)
$\vec{U}$	- Velocity vector
V	- Cell volume
$\bar{V}$	- Average velocity (m/s)
v_n	- Normal velocity (m/s)
v_r	- Radial velocity (m/s)
v_z	- Axial velocity (m/s)
v_f	- Angular velocity (rad/s)
x_1	- Distance along arterial floor (mm)
x_2	- Distance along inner wall (mm)
x_3	- Distance along outer wall (mm)
Y_0	- Bessel function of the second kind, order 0

List of Abbreviations

AHA	- American Heart Association
AMG	- Algebraic Multigrid
CABG	- Coronary Artery Bypass Graft
CAD	- Computer Aided Design
CDC/HCHS	- Centers for Disease Control and Prevention's National Center for Health Statistics
CDS	- Central Differencing Schemes
CFD	- Computational Fluid Dynamics
CHD	- Coronary Heart Disease
CS	- Continuous Segment (Coronary Artery)
CV	- Control Volumes
CVD	- Cardiovascular Diseases
DOS	- Distal Outlet Segment
DVP	- Distal Vein Patch
ETS	- End-To-Side
FDM	- Finite Difference Method
FEM	- Finite Element Method
FVM	- Finite Volume Method
HCT	- Hematocrit
IFMH	- Intimal Fibromuscular Hypertrophy
IH	- Intimal Hyperplasia
IMA	- Internal Mammary Artery
IVC	- Interposition Vein Cuff
LAD	- Left Anterior Descending (Coronary Artery)

LC	- Left Circumflex (Coronary Artery)
LDA	- Laser Doppler Anemometry
LES	- Large Eddy Simulation
LMB	- Left Main Branch (Coronary Artery)
NHC	- National Heart Centre
OSI	- Oscillating Shear Index
PD	- Posterior Descending (Coronary Artery)
PDE	- Partial Differential Equations
PISO	- Pressure-Implicit with Splitting of Operators
PIV	- Particle Image Velocimetry
POS	- Proximal Outflow Segment
PRESTO	- PREssure STaggering Option
PS	- Proximal Segment (Coronary Artery)
PTCA	- Percutaneous Transluminal Coronary Angioplasty
PTFE	- Polytetrafluoroethylene
QUICK	- Quadratic Upwind Interpolation of Convective Kinematics
RCA	- Right Coronary Artery
RMB	- Right Marginal Branch (Coronary Artery)
SIMPLE	- Semi-Implicit Method for Pressure-Linked Equations
SMC	- Smooth Muscle Cells
SV	- Saphenous Vein
UDF	- User Defined Function
WSS	- Wall Shear Stress
WSSG	- Wall Shear Stress Gradient

Chapter 1: Introduction

The heart is one of the most important organs in the human body, functioning as a muscular pump pushing blood through the pulmonary and systemic circulation. As such, heart dysfunction, in the form of cardiovascular diseases (CVD), is often the leading cause of death in most countries.

In the United States, about 87,700,000 Americans have one or more types of cardiovascular diseases (American Heart Association, 2003), which include high blood pressure, coronary heart disease, stroke, congenital heart defects and congestive heart failure (Figure 1.1). CVD has claimed 958,775 lives (40.1% of all deaths) in 1999 and 945,856 lives (39.4% of all deaths) in 2000 (Figure 1.2). The slight decrease is attributed to advances in medical care as well as greater awareness to the disease (hence resulting in improved eating habits and regular exercise). According to the most recent Centers for Disease Control and Prevention's National Center for Health Statistics (CDC/HCHS) computations, if all forms of CVD were eliminated, life expectancy would rise by almost about 7 years. The costs of cardiovascular disease and stroke in the United States in 2002 and 2003 are estimated at US\$329.2 billion (American Heart Association, 2002) and US\$351.8 billion (American Heart Association, 2003) respectively (Figure 1.3).

Figure 1.1 : Percentagebreakdown of deaths from cardiovascular diseases in United States in 2000 (American Heart Association. 2003).

Figure 1.2: Leading causes of death in United States in 2000 (American Heart Association, 2003).

Figure 1.3: Estimated Costs (in billions of dollars) of Cardiovascular Diseases and Stroke in United States in 2003 (American Heart Association, 2003).

1.1 Coronary Heart Diseases

Coronary heart disease (CHD) develops when one or more of the coronary arteries that supply blood to the heart become narrower than they used to be. This happens because of a buildup of cholesterol and other substances in the wall of the blood vessel, affecting blood flow to the heart muscle. As CHD progresses, a person may experience angina pectoris (pain in the chest) during exercise or increased activity as a result of oxygen deficiency of the heart. If left untreated, congestive heart failure

(inability of the heart to pump efficiently) or myocardial infarct (heart attack) may occur. As seen from Figure 1.1, CHD accounts for the majority of deaths due to CVD.

In Singapore the leading causes of death as seen in Table 1.1 are cancer and heart diseases. In 2000, cardiovascular diseases, including coronary heart disease and stroke accounted for about one third (34.8%) of all deaths. Coronary heart disease is the major cardiovascular disease. It is the second leading cause of death, accounting for 20% of all deaths as well as 14,653 hospital admissions in 2000. The rate of heart attacks among Singaporeans aged 20 to 64 years remained fairly stable over the last five years. In 1999, the rate was 85 cases per 100,000 residents. Indians have the highest risk of getting heart attacks, followed by Malays and Chinese (Ministry of Health Singapore, 2001).

Year		2000	2001	2002
Total No. of Deaths		15,693	15,367	15,820
% of Total Deaths				
1.	Cancer	27.0	28.2	28.0
2	Ischemic & Other Heart Diseases	25.1	26.3	24.2
3.	Pneumonia	11.4	10.0	13.1
4.	Cerebrovascular Disease	10.4	9.2	8.8
5.	Injuries	7.2	6.7	6.7
6.	Diabetes Mellitus	2.3	3.3	2.7
7.	Nephritis, Nephrotic Syndrome & Nephrosis	1.3	1.7	1.6
8.	Septicemia	0.6	0.7	0.7
9.	Bronchitis, Emphysema & Asthma	0.7	0.8	0.7
10.	Chronic Liver Disease & Cirrhosis	0.7	0.7	0.6

Table 1.1: Principal causes of death in Singapore (Ministry of Health Singapore, 2001).

The coronary arteries (Figure 1.4) originate distal from the aortic valve in the aortic sinus, supplying blood to the heart. Up to 5 to 10% of the total stroke volume is transported to the heart via the left and right main branch. The left main branch (LMB) provides blood to the left ventricle, working at higher pressures than the right ventricle, hence requiring higher oxygen and blood supply. It bifurcates into the left circumflex (LC) and left anterior descending (LAD) branches. The right coronary is subdivided into a proximal segment (PS), followed by the continuous segment (CS), and bifurcates further downstream in a posterior descending (PD) and right marginal branch (RMB) (Segers et al., 1998). After passage through the capillary beds, most of the venous blood returns to the right atrium through the coronary sinus, but some reaches the right atrium by way of the anterior coronary veins (Berne and Levy, 2001).

Figure 1.4: Schematic drawing of the coronary artery structure (Segers et al., 1998).

Vascular diseases weaken blood vessels, causing increased vulnerability to rupture. In many cases, such diseases also narrow and occlude the vascular lumens, thereby reducing blood supply to the tissues and organs. Common arterial diseases include Arteriosclerosis, Vasculitis, Raynaud’s Disease and Aneurysms, while venous diseases include Varicose Veins and venous thromboses (Manundar, 1992).

Arteriosclerosis, a generic term used to describe several degenerative conditions affecting the arteries (Geddes and Groset, 2000) includes Atherosclerosis, Monckeberg’s medial calcific sclerosis affecting the muscular arteries and arteriolosclerosis which involves smaller arteries and arterioles. Atherosclerosis, being a dominant pattern, is a disease associated with reduced blood flow due to increased fatty deposits on the inner walls (Geddes and Groset, 2000) (Figure 1.5). Diminished coronary blood flow as a result of coronary artery disease (usually coronary atherosclerosis) is one of the most common causes of serious cardiac disease affecting part or whole of the ventricle. Reduced blood supply not only decreased oxygen and metabolic substrates delivery but also results in accumulation of potentially deleterious substances (such as K^+ , lactic acid and H^+) in the cardiac tissues. Severe and prolonged reduction would lead to necrosis (death) of the affected cardiac cells (Berne and Levy, 2001) (Figure 1.6).

Figure 1.5: Progression of plaque build-up in coronary artery.

Figure 1.6: Death of heart tissue due to insufficient oxygen delivery.

1.2 Procedures for Treatment of CHD

Known procedures used to restore blood flow include atherectomy, percutaneous transluminal coronary angioplasty (PTCA), stents and bypass surgery. Coronary atherectomy removes plaque from the arteries using a laser catheter or a rotating shaver. The catheter is introduced into the body through a blood vessel in the leg (or arm) and is threaded through the blood vessels into the blocked coronary artery. The plaque is grounded into minute particles using a high-speed rotating device or vaporized using laser. The shavings are collected in the catheter and removed when it is withdrawn from the body.

In PTCA, a fluid filled balloon-catheter is inserted via the radial or femoral artery and guided to the stenosed area. Upon inflation, the balloon compresses the plaque hence enlarging the lumen for increased blood flow. The shortcomings of this technique include “abrupt closure” of blood vessel due to thrombus formation (2% to 5% of treatments) and restenosis within 6 months after treatment (in over 30% of all patients) (Segers et al., 1998).

Stents are commonly used following PTCA techniques to counter the problems of “abrupt closure” of vessels and restenosis. Intracoronary stents can be self-expandable or balloon expandable. When deployed permanently at diseased locations, they hold the arteries open to improve blood flow. Coronary artery bypass graft (CABG) surgery utilizes a vascular prosthesis (e.g. Dacron or ePTFE grafts) or replacement vessel (e.g. saphenous vein or internal mammary artery) to bridge the stenosed area. An open-chest operation is required and the surgery is used when none of the alternative treatments is thought to be successful.

Despite the increasing popularity of interventional procedures such as atherectomy, PTCA and placement of intravascular stents, there has not been a significant decrease in the number of patients undergoing CABG (Figure 1.7). Statistics have shown an increasing number of patients undergoing revascularization even after treatment with PTCA or other interventions in the past, hence suggesting that such interventions only serve to delay timing of the eventual surgery (Khonsari, 1997).

Figure 1.7: Trends in cardiovascular operations and procedures in US (1979 – 2000) (American Heart Association, 2003).

CABG surgery, however, cannot warrant a lifetime solution to coronary heart diseases because it is prone to graft failure, which may implicate a need for revascularization. Failure modes of bypass grafts include thrombosis, graft infection and intimal hyperplasia. Intimal hyperplasia is the proliferation of smooth muscle cells (SMC) and extracellular matrix within the vessel wall leading to gradual occlusion and it is the most common cause of graft failure. Chronic endothelial injury (Clowes et al., 1983), biomaterials incompatibility (LoGerfo et al., 1983), compliance mismatch

(Clark et al. 1976, Kinley and Marble, 1980, LoGerfo et al. 1983, Trubel et al. 1995 and Ballyk et al. 1998) and hemodynamics have long been postulated to be factors causing intimal hyperplasia. Amongst them, hemodynamics within the anastomoses especially on the distal end has been widely studied and singled out as the most influential factor.

In the last two decades, hemodynamic studies focused mainly on investigating effects of bypass geometries and configurations on flow patterns (Crawshaw et al., 1980, Einav et al., 1985, Binns et al., 1989, Pietrabissa et al., 1990, Keynton et al., 1991, Fei et al., 1994, Ojha et al., 1994, Hughes and How, 1996, Jones et al., 1997, Loth et al., 1997, Moore et al., 1999, Sherwin et al., 2000 and Stoytchev et al., 2001), establishing correlations between intimal hyperplasia and hemodynamic factors as well as attempting to find a predictor for IH development.

Both high wall shear stresses (Fry et al., 1968 and Steinman et al., 1993) and low wall shear stresses (Rittgers et al., 1978, Bergeur et al., 1980, Zarins et al., 1983, Ku et al., 1985, Dobrin et al., 1989, Asakura and Karino, 1990, Bassiouny et al., 1992, Ojha, 1993, Barbee et al., 1994 and Stanton, 1999) have been suggested to influence the development of intimal hyperplasia. In recent years, researchers have also proposed using indices such as spatial and temporal wall shear stress gradients (DePaola et al., 1992, Ojha, 1993, Henry et al., 1996, Kleinstreuer et al., 1996 and Lei et al., 1997) as well as oscillating shear index (OSI) (Ku et al., 1985 and He and Ku 1996) to predict the site of occurrence of intimal hyperplasia.

In an anatomically distant region, surgeons have reported improvement in patency rates using venous tissue interpositions for femoropopliteal and infrapopliteal prosthetic bypass surgeries (Siegman, 1979, Miller et al., 1984, Tyrrell et al., 1990, Taylor et al., 1992 and Neville et al., 2001). Apart from improved compliance matching, the altered geometry also resulted in improved hemodynamics (such as the generation of a dynamic vortex which enhances fluid mixing and acts as a good washout mechanism) essential for inhibiting development of intimal hyperplasia (Rowe et al., 1999, How et al., 2000, Leuprecht et al., 2002 and Cole et al., 2002). Increase in volume as a result of the venous tissue interpositions has also been suggested to delay occlusion time. At present, the use of such interposition vein cuffs has however been limited to lower limb bypasses only.

The present research is part of a long term goal in the design and development of a prosthetic sleeve that acts as a mechanical connector between the bypass graft and host artery (Figure 1.8). Implementation of a sleeve has several advantages such as overcoming difficulties in maintaining consistently high quality suturing during the bypass surgery, as well as having a pre-designed geometry that optimizes flow conditions. Since no study has been previously published on the use of interposition vein cuffs in CABG, this project has been undertaken with the aim of investigating the possibilities of incorporating a cuff-like geometry in the sleeve design. In addition, previous investigations focused mainly on geometries replicated from physiological models created by surgeons, hence there were little or no incentives to vary geometries or anastomotic volume beyond what was possible to create during surgery. The uniqueness of this study stemmed from the fact that incorporation of a sleeve as a mechanical connector meant greater freedom to study designs that were previously impossible to create during bypass surgeries.

Figure 1.8: Application of sleeve as a mechanical connector.

1.3 Objectives and Scope of Research

Objectives for the project include:

- 1) Propose and design sleeve geometries of varying volumes based on studies concerning interposition vein cuffs.
- 2) Study flow patterns in distal anastomoses with incorporated sleeve design using Computational Fluid Dynamics (CFD) for both steady and unsteady flow conditions in coronary artery bypass.
- 3) Evaluate feasibility of designs as well as influence of geometries with varying volumes on flow characteristics and wall shear stress variations.
- 4) Investigate flow patterns in sleeve designs of varying out-of-plane angles and bypass angles.

- 5) Suggest necessary changes if required based on evaluation and propose possible further studies.

This study is limited to determining effects of cuff-like sleeve geometry as well as varying volumes on flow characteristics and shear stress variations along the vessel walls. Since it is not a patient-specific study, geometries and flow conditions used in this study are generic. General idealizations such as smooth wall, perfect cylindrical shape and planarity are considered when designing the models. All analyses are based on already established factors or correlations and it is not the intention of this study to prove or disprove them. Correlations such as low wall shear stresses leading to intimal hyperplasia or enhancing thrombus deposition are assumed. This study shall not take into consideration effects of the sleeve material as up to this point, the choice of material is still unclear and possibilities of seeding the prosthetic walls with endothelial cells are still unknown. The sleeve walls are thus assumed to be prosthetic materials that can withstand wall shear stresses, gradients and oscillations far higher than what the endothelium can tolerate.

The report is set out as follows: Chapter 2 presents an overview of surgical procedures, choice of bypass conduits and failure modes of bypass grafts. Published literatures concerning flow studies on conventional anastomoses as well as interposition vein cuffs are also reviewed. Chapter 3 provides background knowledge on rheology of blood and mathematical models used in prescribing steady and unsteady velocity profiles at the graft inlets. Assumptions made in derivations of these models are also discussed. Computational procedures used to obtain flow information in the bypass models are explained in Chapter 4 while results of the steady and

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unsteady flow in models of varying geometries are presented and discussed in Chapters 5 and 6 respectively. Chapters 7 gives an in depth analysis of flow patterns corresponding to different out-of-plane angles and bypass angles respectively. Conclusions to the current work and suggested future works will be presented in Chapter 8.

Chapter 2: Literature Review

Since the first successful bypass operation performed by Garrett, Dennis and DeBakey in 1964, (Garrett et al.,1973) coronary artery bypass surgery has become one of the most important and frequently performed surgeries for treatment of coronary artery diseases. However popular this revascularization technique is, vascular grafts have a limited lifespan, subjected to factors including thrombosis, graft infection and intimal hyperplasia. Intimal hyperplasia has been known to be the most common cause of long term graft failure although the underlying mechanisms are not fully understood. Researchers have singled out hemodynamics as one of the most influential factors of IH development.

The following sections shall present an overview of the surgical procedures, choice of bypass conduits as well as common failure modes of bypass grafts. A review of published literature concerning flow studies on proximal and distal anastomoses will be conducted. A final section shall evaluate the merits of interposition vein cuffs and the possibility of adapting a cuff like geometry to sleeve designs for coronary artery bypass surgeries.

2.1 Coronary Artery Bypass Graft (CABG) Surgery

Carrel in 1910 reported a series of experiments that constitute the earliest forms of direct coronary artery bypass, by anatomizing the innominate artery of one dog into the distal coronary artery of another and also by using a free carotid autograft between descending thoracic aorta and distal coronary artery (Carrel, 1910). He also performed

the first documented experimental vein bypass grafting by interposing a segment of vein into a dogs transected aorta (Carrel, 1912). For his work on vascular suture and transplantation of blood-vessel organs, he was awarded the Nobel Prize in 1912. It took another 50 years before the first human coronary bypass surgery was performed. In 1962, Sabiston (1974) at John Hopkins hospital carried out the first human CABG surgery. The patient however died three days later of a cerebrovascular accident. In 1964 Garrett, Dennis and DeBakey performed the second, but first successful, human vein bypass grafting on a 42 year old man (Garret et al., 1973). The anastomosis took 23 minutes to perform and was done without cardiopulmonary bypass. The follow up angiography after seven years showed a patent graft. Over the years, CABG surgery has become one of the most important means of revascularization for patients with coronary artery diseases. According to estimates, as many as 519,000 bypass surgeries were carried out in 2000 in the United States (American Heart Association, 2003).

Surgical procedures for bypass operations vary depending on the atherosclerosis disease present in the patient. Revascularization techniques could vary from simple direct grafting to multiple or sequential grafting. The distal anastomosis is usually constructed first with the patient on cardiopulmonary bypass (Figure 2.1). The distal end of the saphenous vein segment is beveled at 30 to 45° with sufficient length for its course over the heart surface. A 10-stitch anastomosis is constructed using 7/0 polypropylene suture. Five stitches are taken around the “heel” of the graft with two stitches to one side of the apex of the vein graft and coronary artery, one stitch through the apex and the final two stitches placed on the opposite side of the apex. The suture loops are drawn up and the suture is pulled straight through to prevent purse-string effect. Five stitches are taken around the “toe” of the graft with the third

of the five placed precisely at the apex of the coronary arteriotomy (Doty, 1997). At the proximal end, the pericardial layer covering the aorta is removed over the anterior wall of the aorta (Figure 2.2). Small openings (4 to 5 mm diameter) are made into the ascending aorta using an aortic punch. Five suture loops of 5/0 polypropylene are then placed around the “heel” of the graft and passed through the aortic wall. Two stitches are placed to one side of the apex, one stitch is placed precisely through the apex of the incision in the saphenous vein, and the final two stitches are placed to the opposite side of the apex. The suture loops are pulled up to approximate the vein graft to the aorta. The anastomosis is completed by placement of stitches in cartwheel fashion around the opening of the aorta. The completed anastomosis should bulge anteriorly above the aortic wall, achieving a “cobra-head” appearance (Doty, 1997).

Figure 2.1 : Surgical procedures for distal anastomosis (Khonsari, 1997).

Figure 2.2: Surgical procedures for proximal anastomosis (Khonsari, 1997).

The current research work is part of a long term goal in design and development of a prosthetic sleeve which acts as a mechanical connector between the graft and host artery. Implementation of a sleeve in bypass surgeries would overcome difficulties in maintaining consistently high quality suturing which usually varies depending on experiences and skills of the operating surgeons.

2.2 Choice of Bypass Graft Conduit

Where choice of conduit is concerned, both autogenous and synthetic grafts can be used for bypass surgeries. Autogenous grafts include veins and arteries procured from the patient. The most common being the greater saphenous vein (SV) due to its simple procurement, ease of handling and excellent inflow (Khonsari, 1997). Saphenous veins generally have a one-year patency rate of 80-90% and a 10-year patency rate of 50%. A preferred alternative is the internal mammary artery (IMA) which has a higher patency rate of 98% for one-year and 90% for 10 years. However, in surgeries that require multiple or sequential bypasses, SV is commonly used due to the limited availability of IMA. Other autogenous grafts under evaluation include the gastroepiploic artery, inferior epigastric artery and radial artery.

In situations of inadequacy in quantity, quality or length of autogenous vessels, synthetic grafts are used instead. The two main choices for prosthetic reconstruction are Dacron (polyethylene terephthalate) and PTFE (polytetrafluoroethylene). Dacron and PTFE grafts retain their tensile strengths over the years after implantation while other synthetic grafts like Nylon (Polyamide), Ivalon and Orlon lose significant tensile strength over a period of months. Synthetic grafts demonstrate acceptable

short-term patencies but poor long-term results (Ku and Allen, 2000). Patency rate for the PTFE femoral-popliteal graft falls to about 50% in four years after implantation. Also, according to Rutherford (1989), Dacron and PTFE grafts have shown no significant difference in patency rates.

2.3 Failure Modes of Bypass Grafts

Vascular grafts have a limited lifespan and their failure would implicate a need for re-surgery. Hence, benefits of bypass surgeries last only as long as the graft remains patent. Graft failures are typically caused by the following three factors: 1) thrombosis, 2) graft infection and 3) intimal hyperplasia (Ku and Allen, 2000). Thrombosis is usually associated with the use of synthetic grafts. It occurs in early postoperative period of 1 month, with the formation of clots due to platelet aggregation occurring at the instant blood is exposed to a foreign material. Such clotting, accompanied by slow or stagnant flow would lead to eventual occlusion. Graft infections occur mostly due to contamination during surgery. It was also noted that synthetic grafts are involved in infections four times more than autogenous vein and there is no significant difference between the infection rates of Dacron and PTFE grafts. Neointimal hyperplasia is tissue ingrowth onto the artificial surface of a synthetic graft. Glagov (1991) classified such ingrowth into two forms: intimal fibromuscular hypertrophy (IFMH) and intimal hyperplasia (IH). The former is a normal healing process which takes place in all grafts and is non-stenotic while the latter is a form of abnormal healing, usually resulting in stenotic and occlusive thickening.

Intima hyperplasia, the most common cause of long term graft failure, is the proliferation of smooth muscle cells (SMC) and extracellular matrix within the vessel wall leading to gradual occlusion of the anastomosis. When such occlusion occurs, re-operation is often required to revascularize the myocardium. IH is most commonly seen in end-to-side distal anastomosis (DeWeese, 1985) and is observed to occur preferentially at the heel and toe of the graft and on the bed of the host artery (Sottiurai et al., 1983 and Sottiurai et al., 1984) (Figure 2.3).

Figure 2.3: Sites of intimal hyperplasia development (Sottiurai et al., 1984).

Several factors postulated to have caused IH include chronic endothelial injury, biomaterials incompatibility, compliance mismatch and hemodynamics. Injury to the endothelium could occur during the bypass surgery. In an injury model (using balloon catheters) by Clowes et al. (1983), the removal of the endothelium is followed by platelet adherence to the exposed subendothelium and a subsequent maximal proliferation of SMC in the media at 48 hr and in the intima at 96 hr (Figure 2.4). Subsequently, he demonstrated that endothelial cell and smooth muscle cell proliferation are major contributions to IH and that a platelet derived growth factor (PDGF) may be responsible (Clowes et al., 1986). LoGerfo et al. in 1983 while

studying locations of intimal hyperplasia in dogs observed no significant difference in the extent of IH between unidirectional and bidirectional outflow at the distal end of a femoro-femoral Dacron bypass grafts. He concluded that blood-material interaction as a result of biomaterials incompatibility is more likely to cause IH than hemodynamics.

Figure 2.4: Histologic cross-sections of deendothelialized injured rat carotid arteries: A) Normal vessel, B) Denuded vessel at 2 days, note loss of endothelium, C) Denuded vessel at 2 weeks, intimal markedly thickened due to SMC proliferation, D) Denuded vessel at 12 weeks, further intimal thickening. Arrow indicates internal elastic lamina (Clowes et al., 1983).

Compliance, defined as a percentage of diameter change per unit pressure is an expression of elasticity of an artery or graft (Hasson and Abbott, 1985). Arterial flow impedance, being a function of this elasticity is increased when a less compliant graft is interposed with the host artery, resulting in loss of energy for flow dissipation (O'Rourke, 1967) and elevated anastomotic shear stresses (Kinley et al., 1974). Several researchers reported this mismatch of mechanical property and distensibility leading to high stress concentrations as a factor causing IH development along the suture line (Clark et al., 1976, Kinley and Marble, 1980, LoGerfo et al., 1983, Trubel et al., 1995 and Ballyk et al., 1998).

2.4 Influence of Hemodynamics on Intimal Hyperplasia

For decades, links between non-uniform hemodynamics and stenotic developments near vessel junctions have been experimentally and theoretically evaluated. Ross and Glomset in 1976 hypothesized that intimal thickening is an adaptive response of the arterial wall to un-physiological flow patterns created by the anastomosis geometry. The fact that arterial bifurcations are prevalent yet only one case of combining flow in the human body (two vertebral arteries joining to form the basilar artery) exists provide further evidence to explain why IH occurs preferentially at the distal anastomosis. In addition, LoGerfo et al. (1979) suggests that platelets trapped in recirculating zones tend to aggregate and release platelet derived growth factor (PDGF) which would lead to myocyte proliferation. Bharadvaj et al. (1982) also indicated that IH is greater in regions of flow separation and that the distal anastomosis was more susceptible than the proximal junction.

White et al. (1993) reported flow patterns in scaled-up rigid models of a distal anastomosis to be three dimensional and helical in both steady and pulsatile conditions. Regions of low and oscillatory wall shear and long particle residence time were identified and compared with animal model observations. Their results support the idea that hemodynamic conditions play an important role in localization of intimal hyperplasia. It is thus crucial to conduct in-depth studies on these hemodynamic factors so as to avoid pitfalls in the sleeve designs. Also, a model that improves hemodynamic conditions is essential for preventing graft failure due to occlusion by thrombus deposition in low flow areas, a problem commonly associated with the use of prosthetic devices.

2.5 Hemodynamics in Proximal Anastomoses

Hemodynamics in the proximal anastomoses was less commonly studied due to preferential development of intimal hyperplasia at the distal end. Using a photochromic tracer technique, Ojha et al. in 1993 observed high positive wall shear stresses (WSS) at the toe and heel of the proximal anastomosis (Figure 2.5). On the bed of the host vessel, shear stress was mostly negative throughout the cycle with a peak value roughly twice as large as the maximum normal value. When compared with an earlier study on hemodynamics in a distal anastomosis (Ojha, 1993), they concluded that preferential development of intimal hyperplasia at the distal site could thus be due to the WSS characteristics observed.

Figure 2.5: Wall shear stress variations along (a), (b) bed, (c) toe and (d) heel (Ojha et al., 1993).

Hughes and How (1995) and Henry et al. (1996) reported highly three dimensional flow patterns characterized by a series of vortices in the distal artery and two helical vortices along the graft axis during steady flow in a proximal anastomosis. In pulsatile flow, the secondary flow components were more pronounced particularly during deceleration phase. The authors suggested that the increased radial velocity components may lead to more collisions between platelet and the thrombogenic graft surface and these activated platelets would be transported downstream and accumulate in the regions of low shear rate at the distal anastomosis where they may contribute to the formation of intimal hyperplasia.

Tam (2001) performed flow studies on proximal anastomosis using Particle Image Velocimetry (PIV) with steady and pulsatile flow conditions. Areas of low flow were observed at the toe and heel of the anastomosis during steady flow. Pulsatile studies also revealed low velocity regions at the graft inner wall during acceleration as well as backflow during deceleration. The author concluded that these low velocity regions ideal sites for thrombus formation or fat deposition, which would then likely be transported through the graft to be deposited at the distal sites. Experimental results of Tam (2001) were validated by Zhang (2002) using numerical simulation.

From the above studies, it was concluded that despite the absence of intimal thickening, poor hemodynamic conditions in the proximal anastomosis could contribute to IH formation at the distal end, by conveying either activated platelets or thrombus downstream into the anastomosis. A well designed sleeve should therefore have an optimal flow condition to prevent deposition of these activated platelets or thrombus which over time may result in occlusion in the anastomosis.

2.6 Hemodynamics in Distal Anastomoses

A survey of literature revealed an enormous number of publications on flow studies at the distal end, as compared to the proximal end. This is likely due to the preferential development of intimal hyperplasia at the distal anastomosis. Flow patterns in steady conditions were summarized as being highly three dimensional with two helical vortices in the distal artery, a recirculation vortex in the occluded proximal arterial segment and a stagnation point on the arterial floor. In pulsatile flow, secondary components in the distal artery became more pronounced with significant flow reversal observed at the toe of the anastomosis extending several vessel diameters along the near wall of the artery and upstream into the hood of the graft. The artery floor was subjected to continually varying shear rate caused by movement of the stagnation point during pulsatile cycle (Hughes and How, 1996).

Flow studies can be classified into two main categories, the first comprising investigations concerning effects of bypass geometries and configurations on flow patterns within the anastomosis. The second category involves establishing correlations between IH and hemodynamic factors as well as attempting to find a predictor for IH development.

One of the earlier studies to investigate geometrical influences on hemodynamics was performed by Crawshaw et al. (1980). Using dye injection technique, flow disturbances was visualized in plastic models with inlet angles of 15° and 30°. In the 15° model, flow separation was observed near the toe when proximal outflow exceeds 10% of the total flow. For the 30° model, a separation zone is always present just

distal to the toe and it moved upstream into the hood region when the proximal outflow is increased to 40%. The results showed that flow disturbances were minimal when the insertion angles were low, hence suggesting that a low angle and low flow in proximal host artery would reduce flow separation and therefore minimize the occurrence of intimal hyperplasia. Einav et al. in 1985 using Laser Doppler Anemometry (LDA) demonstrated that turbulent flow at the graft-coronary intersection can be delayed by decreasing the discontinuity in diameter between the bypass vein and coronary artery and also by reducing the bypass vein and host coronary artery intersection angle. Pietrabissa et al. in 1990 investigated the influence of geometric parameters such as degree of coronary stenosis, bypass diameter and angle using a two dimensional finite element model (Figure 2.6). Results showed the development of a recirculation area immediately downstream of the anastomosis. The magnitude of the recirculation was observed to increase with the bypass angle and diameter as well as the degree of coronary stenosis (Figure 2.7).

Figure 2.6: Flow model by Pietrabissa et al. (1990).

Figure 2.7: Magnitude of recirculation in (a) 60%, (b) 80% and (c) 100% stenosis (Pietrabissa et al., 1990).

In 1991, Keynton et al. fabricated three distal anastomotic models using Plexiglas tubing at junction angles of 30° , 45° and 60° (Figure 2.8). Using LDA, it was observed that axial velocities and shear rates along the outer wall were higher than along the inner wall and occurred in the junction angle order: 45° , 60° and 30° . This study clearly identified changes in wall shear which varied with the anastomotic angle and flow rate. Fei et al. (1994) performed three-dimensional numerical studies on an iliofemoral artery bypass using a steady flow. Inlet Reynolds numbers were 100 and 205 while anastomotic angles varied from 20° to 70° . The authors observed threshold values for graft angles of 60° ($Re = 100$) and 45° ($Re = 205$) above which flow separation occurs along the inner arterial wall (toe region). A stagnation point was observed along the outer arterial wall (floor region) for all cases which moved downstream relative to the toe of the anastomosis with decreasing angles.

Figure 2.8: Variation of angles studied by Keynton et al. 1991.

Ojha et al. (1994) studied effects of anastomotic angles (20° , 30° , 45° and 60°) on wall shear stress distributions under pulsatile flow conditions using the photochromic tracer technique. For all angles, low shear stress was present at the heel and on the bed opposite the heel of the anastomosis apparently due to complete occlusion of the proximal end of the host vessel. Near the toe, increase flow separation occurred with increasing angle. On the bed across the toe, increasing the angle led to increased shear stress. In addition, in this region, the anastomotic angle significantly altered other

properties of the shear stress field such as the mean and peak-to-peak magnitudes and cycle-to-cycle fluctuations. The study provides quantitative data on the wall shear stress distribution within an end-to-side anastomosis and its relation to the anastomotic angle.

Apart from influence of anastomotic angles, Binns et al. (1989) studied the effect of graft diameter upon development of pseudo-intima within the graft and the anastomotic neointimal hyperplasia. Their results suggested an adaptation or remodeling process, in which the vessel responded to altered wall shear stress by changing its wall thickness and then increasing shear by a reduction in the size of lumen. On that basis, they proposed that an optimal graft diameter may exist which may help to prevent neointimal hyperplasia and graft thrombosis. Stoytchev et al. (2001) followed up on this study using cost functions based on steady and mean wall shear stresses to calculate the optimal value of the un-deformed bypass diameter for varying degrees of stenosis. They found that the sum of cross-sectional areas of the optimal graft and stenotic artery (normalized with respect to the cross sectional areas of the normal artery) varies between 1.14 and 1.28, which is consistent with findings of McDonald (1974) for area of the major branches in the body.

Jones et al. (1997) measured in-vivo velocity profiles in canine end-to-side ilio-femoral anastomotic grafts. Three cases including a 'standard' geometry, a stenosed geometry and a case with below average flow rate were studied (Figure 2.9). Observed flow features include separation at the hood and toe, movement of the floor stagnation point and skewed profiles in the proximal outflow segment (POS). Out-of-plane curvature and lateral displacement of the anastomosis inlet appear to have a

strong effect on the flow fields. In addition, compliance affects the instantaneous flow rates within the proximal distal branches. Enhanced separation in vivo appears to be a result of vessel curvature, graft inlet angle, and slight alterations in the instantaneous flow rates, hence variations in graft geometry may be an important determinant of the spatial distribution of intimal hyperplasia.

Figure 2.9: Graft geometries and probe positions for (a) standard geometry, (b) stenosed geometry and (c) model with below average flow rate (Jones et al. 1997).

Figure 2.10: Model geometries of distal end of the 45° end-to-side anastomosis: (a) planar and (b) nonplanar (Sherwin et al. 2000).

Sherwin et al. (2000) studied the influence of out-of plane geometry on hemodynamics within a distal end-to-side anastomosis (Figure 2.10). Results showed that the non-planar three-dimensional flow notably alters the distribution of wall shear stress at the bed of the anastomosis, reducing the peak WSS by approximately 10% when compared with the planar model. Furthermore, an increase in the absolute flux of velocity into the occluded region, proximal to the anastomosis, of 80% was observed in the non-planar geometry when compared with the planar geometry.

The accuracy of bypass models used in studies was a debatable subject, due to several possible geometrical factors and configurations. Loth et al. in 1997 claimed that earlier models used by Crawshaw et al. (1980) and Keynton et al. (1991) which employed two straight tubes connected at an angle were too simplified to give accurate description of flow patterns in the anastomosis. Although the simplified models give good insight into flow behavior, sharp edges at tube junctions may produce flow features different from those observed in vivo. As such, Loth’s model of a PTFE canine artery end-to-side bypass graft incorporated typical realistic geometric parameters of anastomoses constructed by vascular surgeons. The model was created with graft diameter larger than the host artery diameter, curvature present along the hood and junction of the anastomosis constructed without sharp edges at the suture line along the sidewalls.

Figure 2.1 I : Anatomically realistic model (a) and five levels of successive simplification (b – f) by Moore et al. 1999.

Moore et al. (1999) classified the geometrical features as “primary” (such as graft-artery diameter ratio and graft angle) and “secondary” (such as local arterial caliber changes, out-of-plane curvature and small scale topology) (Figure 2.1 I). It was concluded that simplified models provide sufficient information for comparing

hemodynamics with qualitative or averaged disease locations provided the “primary” geometric features are well replicated. “Secondary” features are found to be less important determinants of wall shear stress patterns. However, in patient-specific case studies, the availability of both “primary” and “secondary” features to generate a physiological realistic model would be highly desirable.

As mentioned earlier, the second category of flow studies involves establishing a correlation between intimal hyperplasia and hemodynamic factors. There is considerable evidence that wall shear stress is one mechanism that acts to regulate artery lumen diameter and it is widely believed that the remodeling process of the arteries may be an important factor that leads to intimal thickening. Giddens et al. (1990) observed that under normal flow conditions, arteries stabilize at a diameter which yields a mean wall shear stress in a range of 10 to 20 dynes/cm². Adaptive responses of arteries to wall shear stress in animal models. Kamiya and Togawa in 1980 used a dog model to demonstrate that the carotid artery alters its diameter to respond to changes in flow rate so as to maintain a mean wall shear stress at approximately 15 dynes/cm². Langille and O'Donnell (1986) reported a 21% decrease in carotid artery diameters of rabbits when the blood flow rate was decreased by 70%. When the endothelium was removed from the vessels, arterial response to reduced blood flow disappeared, suggesting the importance of endothelium in compensatory arterial response to long term changes in blood flow rates. Zarins et al. (1987) observed the increase in radius of femoral arteries in cynomolgus monkeys when subjected to chronically elevated flow rates until the mean wall shear stress reached approximately 15 dynes/cm².

Direct correlation between shear stresses and intimal thickening was first reported by Rittgers et al. (1978) when they measured intimal hyperplasia and wall shear rate in vivo and showed an inverse correlation coefficient ($r = -0.201$) between the two. Morinaga et al. (1985) found in a chronic animal study that intimal hyperplasia correlated well with the variation of wall shear stress over a cardiac cycle and that unphysiological variation in wall shear stress played a more important role in determining patency of vein grafts than did flow rate. Bassiouny et al. (1988) measured intimal thickening in grafts implanted into cynomolgus monkeys for 6 months and concluded that the best correlation was between intimal thickening and maximum wall shear stress than mean wall shear stress.

In the past decade, correlation between wall shear stress and intimal thickening has become a dominant research topic where hemodynamic factor is concerned. Several theories have been studied by researchers linking intimal hyperplasia to high WSS, low WSS, spatial variation of WSS (using wall shear stress gradient, WSSG) and temporal WSS variation (using oscillatory shear index, OSI).

Fry et al. (1968) demonstrated the detrimental effects of high wall shear stress to endothelial cells. They reported that upon exposure to shear stresses in the range of 379 dynes/cm^2 for periods as short as 1 hr, cytoplasmic swelling, deformation and disintegration of endothelial cells can occur. It was also observed that mass transfer (including smooth muscle cell migration) through arterial walls increases with increasing shear stress (Fry et al. 1969). Steinman et al. (1993) performed numerical studies of hemodynamics in a two-dimensional 45° rigid-walled end-to-side model anastomosis. Simulation results showed a correlation between locations of high

instantaneous wall shear stresses and sites known to be susceptible to intimal hyperplasia development (toe, heel and along the host artery bed). However, these locations also exhibit high temporal and spatial wall shear stress gradients. Hence they suggested that any one of these effects or a combination of the three may play a role in the development of intimal hyperplasia.

In contrary to the above findings, a larger number of studies suggest a greater influence of low wall shear stress on the development of intimal hyperplasia. Rittgers et al. (1978) while investigating the effects of varying anastomotic angles observed that intimal thickening was greatest in regions of low wall shear. Bergeur et al. (1980) in a separate study using a dog model also made the same observations. In atherosclerotic research conducted on the human carotid bifurcation, experimental data (Ku et al. 1985) validated with in vivo results (Zarins et al. 1983) verified the occurrence of intimal plaque thickening at regions of low wall shear stress while areas with mean wall shear stress exceeding about 10 dynes/cm^2 were spared from early plaque development.

Dobrin et al. (1989) indicated that when autogenous vein are used as arterial bypass grafts, they are exposed to arterial flow and pressure which subject them to three static deformations, three static stresses, increased pulsatile deformations, pulsatile stresses, and altered shear stress at the blood-intima interface. Experiments using 38 dogs were carried out and results showed that intimal hyperplasia is best associated with low flow velocity, a factor correlated with low blood-artery shear stress. Asakura and Karino (1990) studied the exact anatomic locations of atherosclerotic lesions in five human coronary arterial trees prepared postmortem. Observations showed

atherosclerotic plaques and wall thickenings on the outer wall of one or both daughter vessels at major bifurcations and T-junctions. When compared with data obtained using flow visualization and high-speed cinemicrographic techniques, it was discovered that the atherosclerotic sites corresponded with regions of recirculation, secondary flows and low shear stress.

Bassiouny et al. (1992) provided further evidence of this relation when comparing flow models and animal models. Characteristics of low wall shear stress including a stagnation zone along the arterial floor and secondary flows at the heel and toe of the graft corresponded to intimal thickening in a concurrent animal model study. Ojha in 1993 also made similar observations in a 45° end-to-side anastomosis model. Barbee et al. (1994) used atomic force microscopy to study surface topography of living endothelial cells. In the presence of flow, endothelial cells were observed to align themselves with their longitudinal axis parallel to the direction of flow while in regions of low flow and oscillating shear, cells are not as prominently aligned. Stanton (1999) later suggested that high wall shear may elicit formation of tighter intercellular attachments thereby reducing transjunctional permeability, while low and/or changing shear rate directions could be associated with reduction in junctional tenacity hence encouraging smooth muscle cell migration. Nerem (1992) in a review of effects of vascular fluid mechanics on arterial wall and atherosclerosis emphasized once again the inverse correlation of low wall shear stress and direct correlation of oscillatory shear stress with intimal thickening. However, he added that since regions of low shear also are regions of pronounced oscillations, it is impossible to know whether it is the former or latter or even prolonged particle residence times associated with flow separation that predisposes the artery to intimal thickening. Hence he suggested that

while “denoting a localized region of the vasculature as low shear is simply a way of naming the region, it does not mean that low shear itself is the ‘culprit’ of the disease process”.

In the years that follow, studies concerning correlations between spatial and temporal wall shear stress variations became popular. DePaola et al. (1992) performed in vitro experiments that recreate large gradients in surface fluid shear stress found near arterial branches in vivo. This disturbed-flow region includes both flow separation and reattachment. In the latter, shear stress is observed to be small but its gradient is large. Cell migration was also observed predominantly in the downstream direction. Those that remain divide at a rate that is high compared with that of cells subjected to uniform shear. The authors hence suggested that morphological and functional changes in endothelium may be induced by large shear stress gradients and this may eventually lead to formation of atherosclerotic plaques.

Ojha (1993) conducted a study to obtain better description of both spatial and temporal variations of wall shear stress within a 45° end-to-side anastomosis model using the photochromic tracer technique. Shear stress at the heel and on the bed across the heel was close to zero throughout the sinusoidal flow cycle. A stagnation point was observed to fluctuate quite sharply on the bed over a distance of about one tube radius upstream from the site across from the toe. Sudden motion of the stagnation point around peak flow produced sharp temporal gradients of the wall shear stress. When compared to sites of intimal hyperplasia development, a strong correlation is seen with low wall shear stress at the heel and toe and with the sharp temporal

variations of the magnitude and spatial gradient of the wall shear stress on the bed across from the junction.

Figure 2.12: Variations of wall shear stress over flow cycle at six different locations (Ojha, 1993).

Henry et al. (1996) in a simulation study of steady flow in proximal and distal anastomoses reiterated the importance of shear gradients, since areas of low shear are also likely to be areas of high spatial shear stress gradients. This is especially true if the low shear regions are due to flow changing direction, as in the case on the arterial floor and at the anastomotic toe. Hence the correlation of low shear and intimal hyperplasia could be due to the fact that both occur in areas of high shear gradients and therefore it could be the latter that induces intimal hyperplasia.

Kleinstreuer et al. (1996) and Lei et al. (1997) hypothesized that wall shear stress gradient is a very reasonable indicator or predictor of sites susceptible to intimal hyperplasia. Studies comparing hemodynamics in a Taylor patch model versus a standard anastomosis led them to conclude that the increased patency rates of the former could be due to a lower wall shear stress gradients observed.

In addition to wall shear stress gradient, Ku et al. (1985) had earlier proposed a predictor equation known as the oscillating shear index (OSI) which measures abnormality in hemodynamics. The authors reported a correlation of oscillating wall shear stress with atherosclerotic lesions found in the human carotid artery. However, when the local intima thickness, δ , is plotted against the OSI values, it was observed that the overall correlation was poor (Lei et al. 1995). A revised version was given by He and Ku in 1996. The new definition takes into account not only the reverse direction but also any direction changes of the wall shear stress in three-dimensional space. The maximum value of OSI is 0.5 which is the case of fully oscillatory flow without net forward flow (Lei et al. 2001).

Despite numerous research activities involved in studying effects of hemodynamics on intimal thickening, investigators have yet agreed upon a single predictor of IH formation. This is due to the lack of universal treatment of the proximal artery conditions. Some studies utilized prograde flow (Inzoli et al. 1996) simulating only partial blockages of native artery with residual blood entering through the proximal artery to the anastomosis. Other models allow for no flow in the proximal artery (Ojha 1993, Bassiouny et al. 1992, Steinman et al. 1993, Henry et al. 1996, Crawshaw et al. 1980) which would occur in a complete stenosis. Other investigators considered the

possibility of retrograde flow (Kleinstreuer et al. 1996, Lei et al. 1997, Loth et al. 1997, White et al. 1993) which would occur if small collateral vessels are present upstream from the anastomoses, allowing blood from graft to exit through both proximal and distal segments. Hence, direct comparison of results from study to study is not appropriate. Problems regarding the effects of proximal artery flow on hemodynamics in bypass anastomoses were addressed by two separate groups of investigators in 2001.

Kute and Vorp (2001) studied the effect of proximal artery flow condition (prograde, zero and retrograde flow) on the hemodynamics at the distal end-to-side anastomosis (Figure 2.13). Results showed a marked difference in both magnitude and spatial distribution of WSS and WSSG. For example the maximum WSS magnitude on the floor of the artery proximal to the anastomosis for the prograde and zero flow cases is 1.8 and 3.9 dynes/cm² while it is increased to 10.3 dynes/cm² in the retrograde flow case. Similarly the maximum value of WSSG magnitude on the floor of the artery proximal to the anastomosis for the prograde flow case is 4.9 dynes/cm³ while it is increased to 13.6 and 24.2 dynes/cm³ in zero and retrograde flow cases. This suggests that the flow condition in the proximal artery is an important determinant of the hemodynamics at the distal anastomosis of end-to-side vascular bypass grafts. Because hemodynamic forces affect the response of vascular endothelial cells, the flow situation in the proximal artery may affect IH formation and therefore long term graft patency. Since surgeons have some control over the flow condition in the proximal artery, results from this study could help determine which flow condition is clinically optimal.

Figure 2.13: Effects of proximal artery outflow on (a) velocity vectors, (b) wall shear stress and (c) wall shear stress gradient (Kute and Vorp, 2001).

Li and Rittgers (2001) did a similar study to investigate hemodynamic factors with different proximal outlet segment (POS) and distal outlet segment (DOS) flow ratios. Statistical analysis was used to compare these factors between cases having different POS:DOS flow ratios (Case 1 – 0:100, Case 2 – 25:75, Case 3 – 50:50). Results showed that changes in POS:DOS flow ratios had a great influence on the flow patterns in the anastomosis (Figure 2.14). With an increase in proximal outlet flow, the range of location of the stagnation point along the artery floor decreased, while the extent of flow separation along the graft hood increased. The statistical results showed

that there were significant differences for the mean WSS between cases along the graft hood, but no significant differences were detected along the artery floor. There were no significant differences for the spatial WSSG along both the artery floor and the graft hood. However, there were significant differences in the mean OSI between Cases 1 and 2 and between Cases 1 and 3 both along the artery floor and along the graft hood. Comparing these mechanical factors with histological findings of intimal hyperplasia formation obtained by previous canine studies, the results suggest that regions exposed to a combination of low mean WSS and high OSI may be most prone to the formation of IH.

a. The Flow Pattern for Case 1 (POS:DOS=0:100) during Late Diastole.

b. The Flow Pattern for Case 2 (POS:DOS=25:75) during Late Diastole.

c. The Flow Pattern for Case 3 (POS:DOS=50:50) during Late Diastole.

Figure 2.14: Illustration of flow streamline patterns during late diastole for (a) Case 1 (0:100), (b) Case 2 (25:75) and (c) Case 3 (50:50) (Li and Rittgers, 2001).

2.7 Hemodynamics in Interposition Vein Vuffs and Patches

It is a well known fact that the use of interposition venous tissue greatly reduces development of intimal hyperplasia in prosthetic bypass graft anastomoses. Studies have shown that the altered geometry, as a result of inclusion of a cuff or patch has led to improved hemodynamics that may be advantageous in inhibiting intimal thickening.

Intimal hyperplasia has been commonly attributed as the main cause for femoropopliteal and infrapopliteal prosthetic bypass graft failures. Patency rates for two years are at a reported low of 14% to 45% (Hobson et al., 1985, Ascer et al., 1985 and Flinn et al., 1988). Such disappointing results have led surgeons to modify existing conventional end-to-side bypasses in the hope of improving patency rates.

Siegman was the first to propose a vein cuff technique to ease the performance of difficult anastomoses (Siegman, 1979). Miller et al. in 1984 used the same configuration of vein cuff at the outflow anastomosis as described by Siegman and reported an eight-month patency rate of 72.4% in 29 infrapopliteal prosthetic bypasses. Investigators from St. Mary's hospital (Tyrrell et al., 1990) in London reported the use of a modified Miller cuff configuration known as the "St. Mary's boot" The cuff of "St. Mary's boot" is sewn in such a way to achieve a more tapered toe than the Miller cuff. However, it is technically difficult to reproduce.

Taylor et al. in 1992 reported the use of a vein patch to obtain a more tapered funnel shaped anastomosis. A cumulative 5-year patency rate of 54% in infrapopliteal bypasses was reported. The main limitations of the Taylor patch include the site of

incorporation and the increased artery length. The Taylor patch is interposed at the toe of the anastomosis, hence leaving the graft in direct contact with the artery at the heel. Also, to achieve the tapered configuration, an increased artery length is required. Neville et al. (2001) advocated the use of PTFE grafts with a distal vein patch (DVP) in limb threatened patients without available autogenous vein (Figures 2.15 and 2.16). Clinical studies conducted from 1993 to 1999 reported a four-year primary patency and limb salvage rates at $62.89\% \pm 10.6\%$ and $79.21\% \pm 8.45\%$, respectively.

Figure 2.15: Intraoperative photograph showing PTFE/DVP distal anastomosis to a posterior tibial artery (Neville et al., 2001).

Figure 2.16: Completion arteriogram after PTFE/DVP bypass graft to posterior tibial artery (Neville et al., 2001).

2.7.1 Hemodynamics in Interposition Vein Cuffs

Rowe et al. (1999) hypothesized that improvements in patency rates of anastomoses with the use of venous cuffs may be due to improved hemodynamics of the altered geometry. A study was performed to investigate local hemodynamics of four different arterial bypass graft anastomoses: the conventional model, the Miller cuff model and two precuffed graft models. The precuffed graft models are simply PTFE grafts produced with an integral cuff, unlike the Miller cuff model which incorporates a venous cuff.

From flow visualization studies, it was observed that the main difference between the conventional model and the Miller cuff model lies in the presence of a large vortex (Figure 2.17) which dominated the cuff region. The dynamic vortex which lasted from systole to the end of diastole alters the hemodynamics in the following favorable ways:

- (i) The vortex has an effect of mixing fluid within the cuff, hence preventing areas of flow stasis and thus reducing risk of thrombosis.
- (ii) It plays a major role in redistributing regions of low wall shear stress (WSS). Areas of low mean shear are located in the cuff itself and due to the large cuff volume; any myointimal hyperplasia developed there will not have a significant effect on blood flow.
- (iii) The vortex appears to be almost completely suppressed with the arrival of the new flow cycle, hence indicating that fluid particles are not in contact with the anastomosis wall for an extended period.

Figure 2.17: Flow characteristics within the Miller cuff (Rowe et al. 1999).

How et al. (2000), in a similar study, compared the differences in wall shear stress (WSS) magnitudes and distributions between a conventional end-to-side bypass (ETS) model and an interposition vein cuff (IVC) model (Figure 2.18). A critical WSS of 0.5 N/m^2 was defined, where values below WSS_{crit} corresponded to sites of intimal hyperplasia development. The study showed that WSS at the heel and floor of the artery, but not the toe, in the ETS model had values below WSS_{crit} . In the IVC model, only the artery floor exhibited WSS close to the critical value but to a much lesser extent than the ETS model. Earlier studies had suggested that improved patency rates of IVC bypasses could be due to the redistribution of myointimal hyperplasia (MIH) away from critical areas at the heel and toe. The study concludes that the interposition cuff alters the mean WSS distribution within the recipient artery and removes area of low WSS at the heel. Such redistribution would thus result in the decreased MIH observed in clinical studies.

Figure 2.18: Flow studies using (a) ETS and (b) IVC models by How et al. (2000).

Leuprecht et al. (2002) carried out numerical and in vivo studies (using sheep) of pulsatile flow patterns and vessel wall mechanics in both conventional and Miller cuff models. Both models use PTFE grafts. A slight modification of the conventional model (Figure 2.19a) is the Miller cuff (Figure 2.19b) which had a venous cuff incorporated between the graft and the recipient artery. The effect of compliance is

considered in the numerical simulation by assuming elastic behavior for each material (native artery, PTFE and venous cuff). The Young's moduli of each material ($E_{\text{artery}} = 4.1 \times 10^5 \text{ N/m}^2$, $E_{\text{vein}} = 8.2 \times 10^5 \text{ N/m}^2$ and $E_{\text{graft}} = 7.5 \times 10^6 \text{ N/m}^2$) were determined from experiments by Trubel et al. (1995). From the study, low WSS observed on the artery floor opposite the anastomotic junction during the entire pulse cycle for both models might have contributed to the development of intimal hyperplasia. According to Noori et al. (1999) the migration of stagnation point is also considered to play a role in the disease progress. From the results, it was shown that the Miller cuff exhibited a smaller shift in stagnation point as compared to the conventional model. Along the suture line, the study of vessel wall mechanics showed a high compliance mismatch in the conventional bypass model which might explain the development of intimal thickening in this region. In comparison, presence of the venous cuff resulted in better compliance matching between PTFE graft and the artery which may lead to a reduced amount of IH development. The authors also suggested that apart from hemodynamics, the fact that the Miller cuff prosthesis having a larger space would mean a longer time requirement for occlusion to take place. This would of course result in an overall improvement in patency rates.

Figure 2.19: Flow studies in (a) conventional and (b) Miller cuff models by Leuprecht et al. (2002).

2.7.2 Hemodynamics in Interposition Vein Patches

Cole et al. (2002) examined the hemodynamics at a patched arterial bypass anastomosis (Figure 2.20). The vein patch, commonly known as Taylor patch is incorporated into the distal anastomosis of PTFE grafts (Figure 2.21). The revised surgical technique results in a decrease in intimal hyperplasia. The study aimed at investigating whether the Taylor patch provides a hemodynamic advantage which may result in improved patency rates. Flow behavior and wall shear stress in both patched (Figure 2.22a) and conventional (Figure 2.22b) bypass models were investigated.

Figure 2.20: Bypass configuration using the Taylor patch (Cole et al., 2002).

Figure 2.21: Schematics of the Taylor patch (Lei et al., 1997).

Figure 2.22: Flow studies by Cole et al. (2000) using (a) Taylor patch and (b) conventional bypass model.

Figure 2.23: Comparison of flow patterns in (a) conventional and (b) Taylor patch models by Cole et al. (2002).

Figure 2.23 illustrates comparisons of flow patterns between conventional and Taylor patch models by Cole et al. (2002). At the heel of the conventional model, the flow exhibits a large, low momentum recirculation, hence increasing the residence time of blood elements. This increases the probability of platelet and leukocyte adhesion to the endothelium, thus leading to the stimulation of smooth muscle cell proliferation and intimal thickening. The Taylor bypass model showed similar flow behavior. This could likely be due to the fact that the patch is incorporated at the region of the toe, hence flow behavior at the heel remains unchanged. Flow separation is observed at the toe of the conventional model during systolic deceleration. This phenomenon is reduced in the Taylor bypass model.

At the artery floor, the conventional model exhibits a spatial gradient of wall shear stress which implicates the stretching of endothelial cells. The magnitude of this stretching force is large during systole but decreases during diastole. Such cyclic, unnatural sharp stretching may result in activation of cellular reactions or endothelial injury hence leading to intimal hyperplasia. In comparison, the wall shear stress distribution for the Taylor model is more favorable, hence inhibiting possible development of intimal thickening. Apart from hemodynamic benefits due to altered geometry of the Taylor model, the enlarged interface between the graft and artery also ensured lesser flow restrictions when intimal thickening occurs.

From previously discussed literature findings, several advantages of using a prosthetic sleeve in CABG could be realized. Incorporation of the sleeve as a mechanical connector greatly reduces reliance on consistent and high quality sutures which varies amongst surgeons with different levels of experiences and skills. Although true mechanisms underlying intimal hyperplasia development are not known, most if not all literature identified hemodynamics as an important contributing factor which may in turn be influenced by geometrical features as mentioned in distal anastomotic studies. In present bypass surgeries, surgeons have little control over anastomotic geometries. Studies have shown the possibility of platelet activation or thrombus formation in proximal ends which may be carried to the distal region by flowing blood. Poorly constructed anastomosis resulting in undesirable flow patterns would thus enhance intimal thickening. Although intimal hyperplasia is not a concern for the prosthetic sleeve, thrombus formation and deposition would still occur in poor hemodynamic conditions. A sleeve designed with geometrical features that optimizes flow conditions would thus be an ideal device to inhibit such a phenomenon. Predictor

equations established in distal anastomotic studies could be used in a different way to evaluate flow conditions in different proposed designs. For example in arterial flows, oscillating shear stresses on the endothelium should be avoided. However, an OSI value indicating frequent oscillations is greatly desirable in a prosthetic device as it prevents unnecessary flow stagnation (provided the oscillations are not sufficiently large to cause erosion of sleeve material).

Studies on lower limb bypasses revealed a surgical modification to prosthetic bypass grafts through the incorporation of a vein cuff or vein patch. The altered geometry has resulted in improved hemodynamics essential for inhibiting development of intimal hyperplasia. It was observed from the studies that the presence of a vortex in the cuff enhances fluid mixing (hence preventing flow stasis), redistributes wall shear stress (from common sites of intimal hyperplasia to the cuff region) and provides a good washout mechanism with each change of flow cycle. Increased anastomotic volume due to the cuff geometry also minimizes flow restrictions and delays occlusion time hence improving patency rates in the long run. The improved hemodynamic conditions suggest a possibility of incorporating a cuff-like geometry for the sleeve design. Since flow (such as velocity waveform) and physical conditions in coronary artery bypasses are different from that of lower limb bypasses, and that no prior literature has been published, hemodynamic studies would thus have to be conducted to understand flow patterns and wall shear stress distributions at the anastomosis as well as to verify if any benefits could indeed be reaped by the proposed design.

Chapter 3: Theory

Blood flow in human body as summarized by Mazumdar (1992) is characterized by the following features: i) laminar, ii) three dimensional with secondary flows, iii) presence of recirculating ‘dead water’ regions, and iv) varying spatial and temporal wall shear stresses. With the exception of the aorta, where turbulence could occur at certain circumstances, flow in disease-free arteries is mainly laminar throughout the cycle with an order of magnitude less than that within the aorta. Secondary flows in arteries are generally induced by vessel branching and curvature where flow is subjected to changes in direction. Bifurcated flow also gives rise to recirculating regions which may change location and size or even disappear and reappear depending on the flow pulses. Spatial and temporal variations in wall shear stresses on the other hand are closely associated with velocity patterns produced by geometrical branching and time varying flow profiles. The following chapter explores the mathematical modeling of blood flow for both steady and pulsatile conditions. Assumptions used in deriving the velocity profiles will also be discussed.

3.1 General Mathematical Model and Assumptions

According to Schlichting (1960), mass and momentum conservation equations for Newtonian and incompressible fluid using axisymmetric cylindrical coordinate system (r, ϕ, z) can be written as:

Continuity Equation

$$\frac{\partial v_r}{\partial r} + \frac{v_r}{r} + \frac{1}{r} \frac{\partial v_\phi}{\partial \phi} + \frac{\partial v_z}{\partial z} = 0 \quad (3-1)$$

Momentum Equation in r-direction:

$$\rho \left(\frac{\partial v_r}{\partial t} + v_r \frac{\partial v_r}{\partial r} + \frac{v_\phi}{r} \frac{\partial v_r}{\partial \phi} - \frac{v_\phi^2}{r} + v_z \frac{\partial v_r}{\partial z} \right) = F_r - \frac{\partial P}{\partial r} + \mu \left(\frac{\partial^2 v_r}{\partial r^2} + \frac{1}{r} \frac{\partial v_r}{\partial r} - \frac{v_r}{r^2} + \frac{1}{r^2} \frac{\partial^2 v_r}{\partial \phi^2} - \frac{2}{r^2} \frac{\partial v_\phi}{\partial \phi} + \frac{\partial^2 v_r}{\partial z^2} \right) \quad (3-2)$$

Momentum Equation in ϕ -direction:

$$\rho \left(\frac{\partial v_\phi}{\partial t} + v_r \frac{\partial v_\phi}{\partial r} + \frac{v_\phi}{r} \frac{\partial v_\phi}{\partial \phi} - \frac{v_r v_\phi}{r} + v_z \frac{\partial v_\phi}{\partial z} \right) = F_\phi - \frac{1}{r} \frac{\partial P}{\partial \phi} + \mu \left(\frac{\partial^2 v_\phi}{\partial r^2} + \frac{1}{r} \frac{\partial v_\phi}{\partial r} - \frac{v_\phi}{r^2} + \frac{1}{r^2} \frac{\partial^2 v_\phi}{\partial \phi^2} + \frac{2}{r^2} \frac{\partial v_r}{\partial \phi} + \frac{\partial^2 v_\phi}{\partial z^2} \right) \quad (3-3)$$

Momentum Equation in z-direction:

$$\rho \left(\frac{\partial v_z}{\partial t} + v_r \frac{\partial v_z}{\partial r} + \frac{v_\phi}{r} \frac{\partial v_z}{\partial \phi} + v_z \frac{\partial v_z}{\partial z} \right) = F_z - \frac{\partial P}{\partial z} + \mu \left(\frac{\partial^2 v_z}{\partial r^2} + \frac{1}{r} \frac{\partial v_z}{\partial r} + \frac{1}{r^2} \frac{\partial^2 v_z}{\partial \phi^2} + \frac{\partial^2 v_z}{\partial z^2} \right) \quad (3-4)$$

where r , ϕ , z are the coordinates in the radial, angular and axial directions, v_r , v_ϕ and v_z are the radial, angular and axial velocities respectively, ρ , F , P and μ are the density, body force, pressure and viscosity of the fluid respectively. The above equations are used to derive velocity profiles for both steady and pulsatile flow conditions based on the assumption that blood is a Newtonian and incompressible fluid with laminar flow characteristics. The arterial wall is assumed to be rigid with no slip conditions at the fluid-wall interface. An idealized cylindrical geometry with sufficient inlet length is also assumed. Some of the assumptions will be discussed in detail in the following sections.

3.1.1 Newtonian Characteristics of Blood

Blood, measuring about 5200 ml, accounts for $8 \pm 1\%$ of a person's body weight. Its principal functions include carriage of oxygen to tissue cells and the removal of waste products of cellular activity such as carbon dioxide to the lung capabilities for discharge in the expired gas (Yang and Ghista, 1979). Blood is a complex body tissue comprising cellular elements suspended in an aqueous solution of salts and proteins (Fraser, 1995). The cellular elements of blood include red blood cells (primary function being oxygen transport and carbon dioxide removal), white blood cells (mainly in defenses against invading organisms such as bacteria, viruses and parasites) and platelets (major contribution in control of hemorrhage). The fluid component of blood, also known as plasma is a transparent, slightly yellowish liquid in which all cellular elements are suspended. Plasma proteins account for 6% of the plasma volume and they include albumin, immunoglobulins, coagulation proteins and transport proteins.

A Newtonian fluid is by definition one in which the coefficient of viscosity is constant at all shear rates (McDonald, 1974). This is closely approximated by most homogenous liquids, but suspensions of particles show deviations from it. While plasma has been verified to be a Newtonian fluid with coefficient of viscosity of about 1.2 cP, the behavior of whole blood is non-Newtonian in nature. Apart from that, blood exhibits time-dependent (viscoelastic) flow deformation characteristics that can only be modeled by higher order constitutive equations (Enderle et al., 2000). In modeling of blood, the equation most generally applicable assumes that blood behaves either as a power-law fluid or a Bingham plastic as shown in Figure 3.1. For

more accurate modeling of shear rates from 1 to 100 000 s^{-1} , the Casson constitutive equation (modified Bingham plastic mode) is also used (Casson, 1959). Sometimes blood is treated as a viscoelastic fluid of either the Maxwell or Oldroyd type (Yang, 1989).

Figure 3.1 : Shear stress vs. shear rate for Newtonian and non-Newtonian fluids (Whitmore, 1968).

Rheological behavior of blood is highly complex due to the fact that blood viscosity is a variable affected by the following parameters:

1) Shear Rate

Viscosity is observed to increase tremendously with a decreasing shear rate (Figure 3.2). This is believed to result from the formation of “rouleaux” or aggregates (Figure 3.3) of red blood cells at low shear conditions. As the rate of shear increases, the aggregates gradually break up under the influence of large velocity gradients until the red cells are present individually. After a shear rate of approximately 50 s^{-1} , the viscosity approaches an asymptotic value.

Figure 3.2: Viscosity vs. shear rate for erythrocytes of various volume concentrations H_n of the erythrocytes (hematocrit $\times 0.96$) (Brooks et al., 1970).

Figure 3.3: (a) and (b): Aggregation of red cells (David (1976)). (c): A scanning electron microscope (SEM) view magnified 2000 times.

2) Hematocrit (HCT)

Hematocrit generally refers to the relative mass of red cells in a sample of whole blood (Fraser, 1995). Studies conducted by Brooks et al. (1970) showed that up to 14% HCT, whole blood was found to be Newtonian at all shear rates (Figure 3.2). As the HCT level increased to about 70%, the viscosity increases and reaches an asymptotic value above approximately 100s^{-1} .

3) Tube Diameter

As shown in Figure 3.4, viscosity increases with the increase in tube diameter and approaches an asymptotic value at tube diameters larger than about 0.5 mm. The variation of viscosity in small diameter tubes, known as the Fahraeus-Lindquist effect, results from the tendency of spinning red cells to move away from the wall where they leave a cell-free layer of low viscosity. In small diameters, the cross sectional area of cell-free zone is comparable to the central core, hence resulting in lower apparent viscosity. At larger diameters, difference between cell-free zone and central core becomes negligible, thus resulting in an asymptotic value for the apparent viscosity.

Figure 3.4: Apparent viscosity as a function of tube radius (Ruch and Patton, 1965).

In the present study, blood is assumed to exhibit Newtonian characteristics. Hematocrit levels of a healthy adult ranges from 40% to 45% and it is known to decrease with age. According to statistics (American Heart Association, 2003), approximately 97% of the bypass surgeries are performed on patients older than 45 years of age. This aged population would generally have a HCT level lower than the normal range as mentioned earlier. Also, assuming parabolic flow profile for steady

flow conditions, the shear rate in this study was calculated to be greater than 265 s^{-1} . Referring to Figure 3.2, it is observed that viscosity for blood remains at a constant value. In pulsatile flow, Rohlf and Tenti (2001) suggested using Womersley number to distinguish flow regimes where non-Newtonian effects are important ($\alpha < 1$) from those where blood behaves like a Newtonian fluid ($\alpha > 1$). In this study, the Womersley number was found to be $\alpha = 2$, which suggests that non-Newtonian effects can be ignored for unsteady flow conditions. Moreover, the 3 mm graft diameter used for this study falls in the range of asymptotic viscosity values (Figure 3.4) hence further justifying the assumption made.

In hemodynamics research, the assumption of blood as a Newtonian fluid has been widely accepted. Ku and Liepsch in 1986 showed that using both Newtonian and non-Newtonian test fluid resulted in dramatic differences in the location of separation regions in a 90° bifurcation. However, Nerem in 1992 remarked that the non-Newtonian fluid used was one with long chain polymers which does not completely simulate the rheological behavior of blood. Further studies by Perktold et al. (1991) and Friedman et al. (1992) lend support to the assumption of blood as a Newtonian fluid. The former found an average difference between Newtonian and non-Newtonian wall shear stress of about 10% while the latter reported no correlation between the two.

3.1.2 Laminar Flow in Vessels

Reynolds number, a dimensionless quantity, is one of the most frequently used parameter in describing characteristics of steady flow through straight tubes at which

transition from laminar to turbulent flow would occur (Nichols and O'Rourke, 1990). Schlichting (1960) defined a critical Reynolds number of about 2300 where this transition would take place. Wygananski and Champagne (1973) pointed out that for perfectly smooth pipes with no disturbances, the transition can be delayed to $Re > 5 \times 10^4$. They also added that if large disturbances are induced at the inlet by a grid or orifice plate, transition from laminar to turbulent flow can occur below a Reynolds number of 2000. For the present study, disturbances at the graft inlet was absent and the Reynolds number for steady flow conditions ranged from 80 to 250, well below all possible critical Reynolds number mentioned above. Hence the assumption for laminar flow in steady conditions is reasonable.

In cardiovascular systems however, flow is pulsatile, hence requiring a different set of criteria for determining flow transition from laminar to turbulence. Several studies have been conducted on the onset of turbulence in either pure oscillatory flow with no mean components or in flows consisting of a mean flow with a superimposed oscillatory component (Peacock et al., 1998). In their experimental study, Peacock et al. (1998) determined a power law correlation for the critical peak Reynolds number in pulsatile flow through a straight tube as

$$Re_{peak (critical)} = 169 \alpha^{0.83} St^{0.27} \quad (3-5)$$

where α is the Womersley number and St is the Strouhal number. The physiological range of Re_{peak} , α and St for the left main coronary artery are tabulated as shown in Table 3.1. The predicted critical peak Reynolds number for the coronary artery ranges from 1650 (at rest) to 2650 (with exercise) while the actual in vivo coronary peak Reynolds numbers vary from 550 (at rest) to 2250 (with exercise). In our bypass

model, Reynolds numbers in the bypass graft at the highest peak is 95, far below the critical values, hence turbulent flows are not expected. In addition, proximal studies by Tam (2001) and Zhang (2002) did not detect any flow disturbance at the graft region, hence assumption for laminar flow in the pulsatile condition is reasonable for the present study.

<div>Left Main Coronary Artery</div> <div>Dimensionless Group</div>	Re_{mean}	α	St	Re_{peak} (In vivo)	$Re_{peak (critical)}$ From Eq. (3-5)
Minimum (at rest)	180	3	0.008	550	1650
Maximum (with exercise)	750	3	0.005	2275	2650

Table 3.1: Physiological range of dimensionless groups

3.1.3 Effects of Wall Distensibility

Arterial walls are viscoelastic inhomogenous multilayered tissues comprising collagen, elastin fibers and smooth muscle cells (Xu et al., 1992). A survey of literature revealed that most investigations assumed rigid wall properties for the arterial segment being studied. Some researchers believe that the primary effect of wall properties is on propagation of pulse wave and thus on the shape of both pressure and flow waveforms. Hence, if the waveform shape is correctly simulated, then a rigid model may be employed (Nerem, 1992). Others regarded the distensibility of arterial walls as a secondary order effect on arterial bifurcation flows and are thus negligible (Xu et al., 1992). Despite these general beliefs, investigations were carried out by several researchers on the effect of wall distensibility on hemodynamic factors.

Liepsch and Moravec (1984) observed that elastic 35° bifurcation models experienced maximum shear stresses 25% lower than the rigid models. Mark et al. in 1985 reported that maximum shear in compliant models to be 25% higher than rigid ones while Duncan et al. (1990) concluded that compliance actually increased maximum shear magnitudes at some sites and decreased them at others. In simulation studies, Lou (1990) noted that peak wall shear stresses in flexible two dimensional aortic bifurcation models were approximately 10% lower than those in rigid models. Reuderink in 1991 analyzed physiological flow in three dimensional carotid bifurcation models and reported that wall distensibility reduced the size and velocity of recirculation zones, decreased peak wall shear stresses and altered velocity profiles. Both Lou (1990) and Reuderink (1991) concluded that wall distensibility had at most a secondary effect on determining flow patterns. Friedman in 1992 however found no correlation between compliant and rigid models while Perktold and Rappitsch (1995) observed a 25% decrease of wall shear stress in the compliant model of a carotid bifurcation. Hofer et al. (1996) also reported generally lower wall shear stresses in a distensible end-to-side anastomosis.

According to the above studies, effects of wall distensibility on hemodynamics are generally small and considered secondary as compared to other factors like bypass angle and graft-artery diameter ratios. In addition, Perktold et al. (1998) remarked that the appropriate compliance value to apply to coronary arteries tethered to or embedded in the pericardium is unclear hence the importance of compliance on flow field is difficult to estimate. Furthermore, synthetic grafts and diseased arteries are expected to be relatively stiff, hence it is reasonable to assume rigid wall properties for the present study.

3.1.4 No Slip Condition at Fluid-Wall Interface

The no slip condition states that velocity along arterial wall is equal to zero or that no relative motion between the fluid and wall at the interface exists. The assumption is made when evaluating the constant of integration in steady flow as well as in derivation of the Womersley solution for pulsatile flow. The assumption of a no slip condition has been held to be universally true for liquids although on certain occasions, it was suggested that some irregular properties of blood flow in vessels may be due to non-wettable properties of their endothelial lining with a consequent slip at the wall. Nichols and O'Rourke (1990) commented that even if the endothelium is shown to be non-wettable, it is still unjustifiable to conclude slipping would occur. The fact that Poiseuille's equation holds for the flow of mercury in a glass tube suggest that zero velocity does exist at the wall.

3.1.5 Inlet Length

When a fluid enters a pipe, the velocity profile will be relatively flat and the fluid must pass through a finite length in the tube before the velocity profile attains a parabolic shape. Formulae for calculating the inlet length for steady flow can be given as

$$L = \frac{2k\rho\bar{V}r^2}{\mu} = \frac{2k\bar{V}r^2}{\nu} \quad (3-6)$$

where r is radius, $\bar{V}$ is average velocity, $\nu = \mu / \rho$ is the kinematic viscosity and k is a derived constant. There were discrepancies in choosing appropriate values for k .

Goldstein (1938) gave $k = 0.057$ but Prandtl and Tietjen (1934) felt that it was a lowest limit which was too short and proposed an experimentally determined value of $k = 0.01$. The highest value was quoted by Chang and Atabek (1961) at $k = 0.13$. In the present study, a value of $k = 0.08$ recommended by Nichols and O'Rourke (1990) is used. Using average velocities derived from Reynolds numbers of 80, 140 and 250, the inlet length was found to be in the range of 1 to 3 cm. Since length of bypass grafts before the inlet used in the present model is larger than the given range, the assumption of sufficient inlet length is acceptable for steady flow conditions.

The concept by which flow becomes established in an oscillatory flow is essentially similar to that in steady flow, with the exception that while initial steady flow profile is flat and also forward, the continuously oscillatory flow profile is never flat. The oscillatory flow profile can therefore be considered as a steady flow which has not had time to become completely established. Hence, the inlet length of the oscillatory flow is expected to be shorter than that for steady flow; indeed the higher the value of α (the Womersley number), the shorter one would expect the oscillatory length to be. Atabek et al. (1964) found that the ratio of pulsatile to steady inlet lengths varied by 36.5% at a value of $\alpha = 4$, appropriate to the iliac artery of a dog. The variability is even greater in large arteries with higher values of α and blood flow velocity. For this study, since the inlet length is sufficiently long for steady flow, this assumption should readily be applied to pulsatile flow.

3.2 Steady Flow Model (Hagen-Poiseuille Equation)

Although circulation at the precapillary level is mainly unsteady, steady flow models are practical because they provide insight into different aspects of arterial flow patterns. In addition, they are easier to use due to the absence of time variations in governing equations. As such, steady flow is commonly used for preliminary studies of hemodynamics in vessels before embarking on the more complicated unsteady flow models.

Steady flow in a cylindrical tube is described by the Hagen-Poiseuille equation, which states that pressure drop along a tube is directly proportional to length of the tube, rate of flow and viscosity of the fluid, and inversely proportional to the fourth power of the internal radius. The phenomenon was observed by both Poiseuille and Hagen independently but it was the latter who derived the equation from theoretic principles.

3.2.1 Derivation of Steady Flow Velocity Profile for Graft Inlet (Munson et al., 2001)

For an axisymmetric and one dimensional flow in a rigid cylindrical vessel of radius R , the radial velocity v_r and tangential velocity v_ϕ becomes zero. Hence the continuity equation (3-1) now becomes

$$\frac{\partial v_z}{\partial z} = 0 \quad (3-7)$$

which implies that the axial velocity component is a function of r alone. In the case of a steady flow, the temporal acceleration terms $\frac{\partial v_r}{\partial t}$, $\frac{\partial v_\phi}{\partial t}$ and $\frac{\partial v_z}{\partial t}$ are neglected.

Assuming that the simulated artery or graft is placed horizontally, the body forces F_r ,

F_ϕ and F_z can also be ignored. Hence the momentum equations in the r and ϕ directions become $\frac{\partial P}{\partial r} = 0$ and $\frac{1}{r} \frac{\partial P}{\partial \phi} = 0$ respectively. It can be concluded from the above equations that pressure is constant over a cross-section. Substituting equation (3-7) into (3-4) and removing the neglected terms, the momentum equation in the z-direction becomes

$$\frac{d^2 v_z}{dr^2} + \frac{1}{r} \frac{dv_z}{dr} = \frac{1}{\mu} \frac{dP}{dz} \quad \frac{1}{r} \frac{d}{dr} \left(r \frac{dv_z}{dr} \right) = \frac{1}{\mu} \frac{dP}{dz} \quad (3-8)$$

Since $\frac{dP}{dz}$ is not a function of r , integrating with respect to r gives

$$\frac{dv_z}{dr} = \frac{1}{\mu} \frac{dP}{dz} \frac{r}{2} + C_1 \quad \text{where } C_1 \text{ is an arbitrary constant. Applying boundary condition}$$

due to symmetry such that at $r = 0$, $\frac{dv_z}{dr} = 0$, C_1 is found to be 0. Integrating with

respect to r again, $v_z = \frac{1}{\mu} \frac{dP}{dz} \frac{r^2}{4} + C_2$. Using boundary condition for a non slip wall,

$v_z = 0$ at $r = R$, we obtain $C_2 = -\frac{1}{\mu} \frac{dP}{dz} \frac{R^2}{4}$, and replacing $\frac{dP}{dz}$ by $\frac{\Delta P}{L}$, the expression

for the axial velocity becomes

$$v_z = \frac{1}{4\mu} \frac{\Delta P}{L} (R^2 - r^2) \quad (3-9)$$

To derive a velocity profile in terms of average velocity, the flow rate is first

determined by integrating velocity over the cross section, where $Q = \int_0^R v_z 2\pi r dr = \frac{\Delta P \pi R^4}{8\mu L}$.

The mean velocity is found using

$$\bar{v}_z = \frac{Q}{A} = \frac{\Delta P \pi R^4 / 8 \mu L}{\pi R^2} = \frac{\Delta P R^2}{8 \mu L} \quad (3-10)$$

$$\text{and } \frac{\Delta P}{L} = \frac{8 \mu}{R^2} \bar{v}_z \quad (3-10a)$$

Substituting equation (3-10a) to (3-9), the velocity profile which is parabolic in nature over the tube cross section can be expressed as

$$v_z = 2\bar{v}_z \left[1 - \left(\frac{r}{R} \right)^2 \right] \quad (3-11)$$

3.3 Unsteady Flow Model (Womersley Solution)

One major disadvantage of the Hagen-Poiseuille equation is the condition of steady flow. Since arterial flow is highly pulsatile, results obtained from the Hagen-Poiseuille models could only offer a preliminary insight and not a true representative of physiological flow patterns in the arteries.

It was generally assumed at the beginning that flow rate was determined by pressure pulse such that an increase in pulsatile pressure would cause an increase in pulsatile flow. Shipley et al. (1943) conducted an empirical study on pulsatile flow patterns in a wide range of arteries using an orifice meter. No clear relation between the pressure and flow waveforms was observed, leading them to suggest that flow was likely to be related to the derivative of pressure rather than the pressure itself. Figure 3.5 shows data obtained from measurements of a flow rate (Q) and arterial pressure pulse (P) recorded simultaneously in the femoral artery of a dog (Nichols and O'Rourke, 1990).

As observed, the increase in flow corresponds approximately with pressure rise. However, it was noted that the peak flow rate precedes the peak pressure. On the assumption that flow was driven by pressure pulse, such an observation would have violated the laws of inertia. McDonald (1955) concluded that it was pressure gradient along the artery that determines pulsatile flow. Figure 3.6 illustrates how an oscillatory pressure gradient was obtained from two identical traveling waves recorded a short distance apart. The pressure gradient (mmHg/cm) was derived by subtracting the pressure at the downstream point from the upstream one at 15° intervals and dividing by the distance between recording points. Figure 3.7 shows the aortic flow and its corresponding pressure gradient.

Figure 3.5: Flow rate and arterial pressure recorded in femoral artery of a dog (Nichols and O'Rourke, 1990).

Figure 3.6: Derivation of pressure gradient (Nichols and O'Rourke, 1990).

Figure 3.7: Flow rate and pressure gradient in the aorta (Nichols and O'Rourke, 1990).

3.3.1 Derivation of Unsteady Flow Velocity Profile for Graft Inlet (Mazumdar, 1992)

Consider flow in a cylindrical vessel of radius R with sufficient inlet length (as verified in Section 3.1.5). Due to axisymmetric flow, the tangential velocity component v_ϕ will be equal to zero in the governing equations and variations with respect to v_ϕ will be neglected. The continuity equation will reduce to the form

$$\frac{\partial v_r}{\partial r} + \frac{v_r}{r} + \frac{\partial v_z}{\partial z} = 0 \quad (3-12)$$

Momentum equations in the r and z directions will contain non-linear convective acceleration terms. To simplify the non-linear equations, an order of magnitude study was performed, where the convective terms $(v_r \frac{\partial v_r}{\partial r}, v_z \frac{\partial v_r}{\partial z}, v_r \frac{\partial v_z}{\partial r}, v_z \frac{\partial v_z}{\partial z})$ were found to have smaller effects on flow dynamics (as compared to leading temporal acceleration terms) and can be ignored as a first approximation. Body forces F_r and F_z are neglected assuming a horizontal position. Rewriting Equations (3-2) and (3-4), the r and z momentum equations become

$$\frac{\partial v_r}{\partial t} = -\frac{1}{\rho} \frac{\partial P}{\partial r} + \frac{\mu}{\rho} \left(\frac{\partial^2 v_r}{\partial r^2} + \frac{1}{r} \frac{\partial v_r}{\partial r} - \frac{v_r}{r^2} + \frac{\partial^2 v_r}{\partial z^2} \right) \quad (3-13)$$

$$\frac{\partial v_z}{\partial t} = -\frac{1}{\rho} \frac{\partial P}{\partial z} + \frac{\mu}{\rho} \left(\frac{\partial^2 v_z}{\partial r^2} + \frac{1}{r} \frac{\partial v_z}{\partial r} + \frac{\partial^2 v_z}{\partial z^2} \right) \quad (3-14)$$

Based on earlier assumptions of a rigid wall, there will be no radial wall motion and with symmetry, the radial velocity component will be zero. The continuity (3-12), r -momentum (3-13) and z -momentum (3-14) equations further reduce to

$$\frac{\partial v_z}{\partial z} = 0 \text{ or } \frac{\partial^2 v_z}{\partial z^2} = 0 \quad (3-15)$$

$$\frac{1}{\rho} \frac{\partial P}{\partial r} = 0 \text{ or } \frac{\partial P}{\partial r} = 0 \quad (3-16)$$

$$\frac{\partial v_z}{\partial t} = -\frac{1}{\rho} \frac{\partial P}{\partial z} + \frac{\mu}{\rho} \left(\frac{\partial^2 v_z}{\partial r^2} + \frac{1}{r} \frac{\partial v_z}{\partial r} \right) \quad (3-17)$$

It is clear from the above equations (3-15) and (3-16) that the axial velocity component is a function of radius and time alone, $v_z = v_z(r,t)$, while pressure (being constant over the cross-section) is a function of axial distance and time alone, $P = P(z,t)$. For a pulsatile sinusoidal flow with pressure gradient of amplitude A and axial velocity of a similar form, it can be observed

$$\frac{\partial P}{\partial z} = -Ae^{i\omega t} \quad (3-18)$$

$$v_z(r,t) = V(r)e^{i\omega t} \quad (3-19)$$

where $\omega = 2\pi f$ is the angular frequency in radians per seconds of the oscillatory motion, with f being the frequency in hertz. Substituting equations (3-18) and (3-19) into the z -momentum equation (3-17) gives

$$\frac{d^2 V}{dr^2} + \frac{1}{r} \frac{dV}{dr} - \frac{i\omega\rho}{\mu} V = -\frac{A}{\mu} \quad (3-20)$$

It is known that a general solution of a homogeneous ordinary differential equation of the form

$$\frac{d^2 y}{dx^2} + \frac{1}{x} \frac{dy}{dx} - K^2 y = 0 \quad (3-21)$$

$$\text{is } y = MJ_0(iKx) + NY_0(iKx) \quad (3-22)$$

which involves Bessel functions of the first kind, J_0 and second kind, Y_0 of complex argument with arbitrary constants M and N . Observe that in this case, $y \rightarrow V$, $x \rightarrow r$ and $K^2 \rightarrow i\omega\rho/\mu$. Hence the solution to equation (3-20) is

$$V(r) = MJ_0\left(i\sqrt{i\frac{\omega\rho}{\mu}}r\right) + NY_0\left(i\sqrt{i\frac{\omega\rho}{\mu}}r\right) + \frac{A}{i\omega\rho} \quad (3-22)$$

At $r = 0$, $Y_0(0) \rightarrow \infty$ but $V(r)$ must be finite on the axis, hence N must be equal to 0.

For a non slip boundary condition at $r = R$, $V(r) = 0$, therefore

$$MJ_0\left(i\sqrt{i\frac{\omega\rho}{\mu}}R\right) + \frac{A}{i\omega\rho} = 0 \quad (3-23)$$

Introducing a non-dimensional parameter α known as the Womersley number,

$$\alpha = R\sqrt{\frac{\omega\rho}{\mu}} \quad \text{or} \quad \alpha = R\sqrt{\frac{\omega}{\nu}} \quad (3-24)$$

where $\nu = \mu/\rho$ is the kinematic viscosity.

Equation (3-23) is rewritten as

$$M = -\frac{A}{i\omega\rho} \frac{1}{J_0(i^{3/2}\alpha)} \quad \text{or} \quad M = \frac{iA}{\omega\rho} \frac{1}{J_0(i^{3/2}\alpha)} \quad (3-25)$$

Substituting equation (3-25) into (3-22), we obtain

$$V(r) = -\frac{iA}{\omega\rho} \left(1 - \frac{J_0\left(i^{\frac{3}{2}}\frac{\alpha r}{R}\right)}{J_0\left(i^{\frac{3}{2}}\alpha\right)} \right) \quad (3-26)$$

and further substituting equation (3-26) into (3-19), the axial velocity is obtained as

$$v_z(r, t) = -\frac{iA}{\omega\rho} \left(1 - \frac{J_0\left(i^{\frac{3}{2}}\frac{\alpha r}{R}\right)}{J_0\left(i^{\frac{3}{2}}\alpha\right)} \right) e^{i\omega t} \quad (3-27)$$

The volumetric flow rate $Q(t)$ is obtained by integrating the axial velocity over the

cross-sectional area, where $Q = \int_0^R v_z 2\pi r dr$, hence

$$Q(t) = -\frac{i\pi A e^{i\omega t} R^2}{\omega\rho} \left(1 - \frac{2J_1\left(i^{\frac{3}{2}}\alpha\right)}{i^{\frac{3}{2}}\alpha J_0\left(i^{\frac{3}{2}}\alpha\right)} \right) \quad (3-28)$$

where J_1 is a Bessel function of the first kind with order 1.

Finally, dividing equation (3-27) by (3-28), the fully developed pulsatile velocity profile corresponding to a specific volume flow waveform $Q(t)$ can be expressed as

$$v_z(r, t) = \frac{Q(t)}{\pi R^2} \frac{\left(1 - \frac{J_0\left(i^{\frac{3}{2}} \frac{\alpha r}{R}\right)}{J_0\left(i^{\frac{3}{2}} \alpha\right)} \right)}{\left(1 - \frac{2J_1\left(i^{\frac{3}{2}} \alpha\right)}{i^{\frac{3}{2}} \alpha J_0\left(i^{\frac{3}{2}} \alpha\right)} \right)} \quad (3-29)$$

Further derivation to simplify the equation for generation of the velocity profile was discussed in Section 4.4.2. Figure 3.8 shows velocity profiles generated by sinusoidally oscillating pressure gradients with varying α values: A) 3.34, B) 4.72, C) 5.78 and D) 6.67 for half a cycle. It is observed that even at the lowest frequency, a true parabolic is not formed at any time. The near wall regions always have a low velocity owing to effects of viscosity. As a result, fluid at the near wall region has a low momentum and thus reverses easily when pressure gradient reverses. As the frequency increases from A to D, the velocity profiles become much flattened. Due to the definition of Womersley parameter, an increase in diameter without change of frequency will also cause an increase in α which produces a similar alteration in the profile.

Figure 3.8: Velocity profiles of flow resulting from sinusoidal pressure gradient in a tube (Nichols and O'Rourke, 1990).

Linford and Ryan (1965) have tested the Womersley equations experimentally in rigid tubes over a wide range of α values. Close agreement between theoretical and measured flows were observed and very small differences occurred when turbulent flow was present at values of α above 10. In the comparison made by Kunz and Coulter (1967), using blood tubes, the agreement between theory and experiment was very close but at very low frequencies. Discrepancies at higher frequencies were attributed to irregular viscous properties of blood. Although the Womersley solution cannot be an exact representation of pulsatile flow in arteries due to the several assumptions, it is certainly as close or closer a practical approximation as Poiseuille's equation for steady flow in arteries.

Chapter 4: Methods

In the present study, numerical methods were used in obtaining information describing flow patterns in the distal anastomosis with an incorporated sleeve design. Physical models were generated and meshed using a preprocessor GAMBIT . The meshed model was read into a commercial solver FLUENT where computations were carried out. Post-processing of information was performed using Tecplot, Matlab or Microsoft Excel.

4.1 Computational Fluid Dynamics

Computational Fluid Dynamics (CFD) is the generation of information about fluid flow in given situations through the use of computers (Shaw, 1992). The physical aspects of fluid flow are governed by three fundamental principles of mass, momentum and energy conservation, expressed mathematically as Partial Differential Equations (PDE) or in integral form. CFD as Shaw (1992) described is “the art of replacing the governing PDEs of fluid flow with numbers and advancing these numbers in space and/or time to obtain a final numerical description of the complete flow of interest”. Anderson (1996) claimed that CFD in engineering predictions has become so strong today that it can be viewed together with classical cases of pure experiment and theory, as the ‘third dimension’ in fluid dynamics. In vascular flow research CFD is becoming a promising tool as it can provide detailed flow information over a wide variety of complex geometry and flow conditions. CFD offers several advantages over physical experiments (Xu et al., 1992, Steinman et al.,

1994, Fei et al., 1994, Loth et al., 1995, Inzoli et al., 1996, Perktold et al., 1998, Xu et al., 1999), including:

- a) Complex geometries generated by Computer Aided Design (CAD) software can be manipulated with ease. Flow models in experimental studies however require more time and manpower to fabricate. Hence the effects of geometries on hemodynamics can be studied more efficiently using CFD.
- b) Numerical methods allow more variation of inlet and/or outlet conditions such as flow or pressure waveforms than is practical in experiments. Hence a wider study could be achieved in a shorter amount of time.
- c) Mathematical models are generally less expensive to carry out than experimental models although three dimensional numerical models do consume large amounts of computer space and time.
- d) Large amount of data can be readily obtained from numerical simulations as compared to physical experiments, one of the most common being wall shear rate measurements. Obtaining accurate velocity recordings near the wall is difficult due to the relatively small velocities present and lower particle density. A numerical simulation on the other hand provides velocity profiles and thus shears stresses along any wall site on the computational mesh.

Despite the several advantages, CFD is limited by possible inaccuracies due to poor mesh quality or spatial resolution, idealizations of the governing equations, approximations during discretization as well as insufficient convergence criteria during the iterative process. Discussion of errors will be made in subsequent sections.

4.2 Design of Physical Models

In design of the physical models, accuracy is the prime concern. Researchers have expressed different concerns over how realistic flow models should be. Perktold et al. (1998) felt that with improvements in technology, it is essential to consider physiologically realistic geometric models as subtle changes in geometry can affect the flow field significantly. Moore et al. (1999) on the other hand showed that as long as “primary” features such as graft-artery diameter ratio and graft angle are well replicated, the flow model should provide sufficient information for hemodynamics comparison. For a preliminary study on flow patterns within a proposed sleeve geometry, it was suggested that a realistic model with “primary” features was sufficient. In addition, it was to be noted that effects of sleeve thickness would not be considered in this study.

4.2.1 Sleeve Models of Varying Geometries

The designed models were made up of the following four parts: graft, sleeve, proximal host artery and distal host artery (Figure 4.1). Based on medical data provided by a cardiovascular surgeon from National Heart Centre, the diameters of the right coronary artery (RCA) and the saphenous vein graft were chosen as $D_A = 2.6$ mm and $D_G = 3$ mm respectively. The graft length was chosen to be $L_G = 12.5$ mm. The relatively short length was chosen so that the remaining graft (not part of this model) was long enough for the incoming flow to be fully developed before entering the inlet of the model. The distal section of the host artery was $D_A = 26$ mm long to ensure sufficient length for flow to stabilize. In clinical situations, the proximal

section of the host artery will be fully occluded over time, hence a shorter length of $L_P = 10$ mm was imposed as an arbitrary distance from a fully occluded stenosis to the sleeve. The sleeve length was assumed to be $L_S = 10$ mm.

Figure 4.1 : Standard dimensions in distal anastomotic model with incorporated sleeve design.

Higher priority was placed in designing the sleeve geometry. Five models were created with varying dimensions and purposes as shown in Table 4.1. The main objective was to study effects of such geometrical variation on hemodynamics. The sleeve diameter D_S and height H_S were normalized by D_G and D_A respectively (Table 4.1 and Figure 4.2). The minimum possible diameter for the sleeve D_S was set equal to the graft diameter as any value lower than that would result in a stenosis in the sleeve, hence possibly inducing turbulence. The sleeve geometry was designed by creating a series of ellipses with constant cross sectional areas (Figure 4.3). The edges of the ellipses were interpolated to create a smooth contour which formed the cuff-like geometry. Extreme attention was paid to the toe and heel region because these corners were prone to un-physiological sharpness which may lead to large magnitudes of wall shear stresses. Such slope discontinuities were avoided by rounding these regions. Design of all five sleeve models are shown in Appendix A.

In the present study, a planar model was utilized with out-of-plane effects ignored. Calculation time and memory could thus be reduced by modeling one symmetric half with relevant boundary conditions imposed.

Model	D_A	D_G	D_S	H_S	Remarks	Approximate Volume
1	2.6 mm	3 mm	D_G	D_A	Baseline Model	88 mm^3
2	2.6 mm	3 mm	D_G	$1.5 D_A$	Increased Height	97 mm^3
3	2.6 mm	3 mm	$1.25 D_G$	D_A	Increased Diameter	104 mm^3
4	2.6 mm	3 mm	$1.25 D_G$	$1.5 D_A$	Increased Diameter and Height	120 mm^3
5	2.6 mm	3 mm	$1.5 D_G$	$2 D_A$	Increased Diameter and Height	160 mm^3

Table 4.1 : Sleeve models of varying dimensions.

Figure 4.2: Diameter D_S and height H_S of sleeve geometry.

Figure 4.3: (a) Top and (b) isometric view of sleeve geometry.

4.2.2 Sleeve Models of Varying Out-Of-Plane and Bypass Angles

Coronary artery bypass surgeries were highly three dimensional. As shown in Figure 4.4, in the construction of the anastomosis, the conduit is usually grafted at an angle to the coronary arteries. In order to accommodate this configuration, a sleeve design with out-of-plane features had to be incorporated to facilitate attachment between the vein and the sleeve. The out-of-plane angle (θ_{OP}) was defined as the angle between the incoming vein graft axis and the coronary artery axis as shown in Figure 4.5. In the present study, an entrance length L_E was incorporated before fluid entering into the cuff region of the sleeve as shown in Figure 4.5. This length was integrated to allow fluid to stabilize and develop fully before entering the sleeve. Out-of-plane angles of 30° , 60° and 90° were designed as shown in Figure 4.6. The objective of this study was to identify the entrance length required for flow stabilization corresponding to the different out-of-plane angles.

Figure 4.4: Saphenous vein grafted at an angle to the coronary artery.

Figure 4.5: Out-of-plane angle (θ_{OP}) and entrance length L_E of sleeve design.

Figure 4.6: Out-of-plane angles of $\theta_{OP} =$ (a) 30° , (b) 60° and (c) 90° .

Due to curvature of the heart, the vein is often grafted to the coronary arteries at an angle as shown in Figure 4.7. The bypass angle (θ_B) in this case was defined as the angle between the graft axis and the coronary artery axis in the same plane as shown in Figure 4.8. Six models were designed with varying angles of 0° , 15° , 30° , 45° and 60° as shown in Figure 4.9. The out-of-plane angle together with the bypass angle gave full flexibility to the sleeve to counter the three-dimensionality of the bypass surgery.

Figure 4.7: Saphenous vein grafted at an angle θ_B to the coronary artery.

Figure 4.8: Bypass angle (θ_B) of sleeve design.

Figure 4.9: Bypass angles of $\theta_B =$ (a) 0° , (b) 15° , (c) 30° , (d) 45° and (e) 60° .

4.3 Mesh Generation of Models

In Computational Fluid Dynamics, the numerical mesh is a discrete representation of the geometric domain where variables are solved in each discrete location. The process of meshing is as important as the solution itself as poor quality meshes or insufficient spatial discretization are some of the reasons leading to inaccurate solutions. The distal anastomotic model presented here was divided into the following sections and meshed separately: graft, sleeve, proximal host artery and distal host artery (Figure 4.10).

Figure 4.10: Mesh generation for distal anastomotic model.

During meshing, trade-offs in setup time, computational expense and numerical diffusion are often the main concern of CFD users. For flow problems involving complex geometries such as the sleeve region, creation of structured or block-structured grids (comprising quadrilateral and hexahedral cells) could be extremely time consuming, if not impossible. Setup time was greatly reduced by using unstructured grids employing triangular and tetrahedral cells. In addition, such tetrahedral mesh could be created with far fewer cells than the equivalent hexahedral

meshes because the former allowed cells to be clustered in regions of flow domain while the latter generally forced cells to be placed in regions where they were not needed. Computational expense was thus conserved. In relatively simple geometry such as the graft and host artery sections where flow conformed well to the shape of the geometry, the use of hexahedral cells however made more economical sense. Hexahedral cells permit a larger aspect ratio than tetrahedral cells, where a large aspect ratio would affect skewness of the cell resulting in poor accuracies. The use of hexahedral mesh in such geometries would result in fewer cells than if tetrahedral cells were used. A further step in reducing computational resources would be through the use of boundary layers. For flow in a cylindrical conduit, fluid velocity gradients are large in regions immediately adjacent to the wall and small near the center of the conduit. Boundary layers define the spacing of mesh node rows in regions immediately adjacent to edges or faces. Attaching a boundary layer would thus increase mesh density near the wall and decrease density near the center, thereby obtaining sufficient information to characterize the gradients in both regions while minimizing the total number of mesh nodes in the model.

Numerical diffusion is a dominant source of error in all practical numerical schemes for solving multidimensional fluid flow. It arises from truncation errors that are a consequence of representing fluid flow equations in discrete forms. Numerical diffusion is minimized when flow is aligned with the mesh or when resolution of the mesh is increased. Since flow is never aligned with tetrahedral cells, the use of a hexahedral mesh in cylindrical graft and host artery sections could thus minimize numerical diffusion.

4.3.1 Mesh Validation

Meshes generated as described in the previous section could be unsuitable for two main reasons: presence of badly shaped cells that increase the local effective spatial discretization error and insufficient spatial resolution leading to inaccurate computation of local velocity and wall shear stress features (Prakash and Ethier, 2001).

Before reading the meshed models into FLUENT for computations, much attention was paid in examining mesh quality using an in-built function by GAMBIT. The preprocessor provides several measures (metrics) for assessing quality of the meshes, such as aspect ratio, Q_{AR} (where $Q_{AR} = 1$ represents an equilateral element), EquiAngle Skew, Q_{EAS} and EquiSize Skew, Q_{ESS} (where overall relationship between Q_{EAS} or Q_{ESS} and element quality is shown in Table 4.2). Mesh examination showed an average of 99% of the elements having values of Q_{AR} between 1 and 2 while approximately 90% of the elements had skew qualities (Q_{EAS} or Q_{ESS}) ranging from good to excellent. Such qualities will ensure the accuracy and robustness of the CFD solution in the subsequent calculations.

Q_{EAS} or Q_{ESS}	Quality
Q_{EAS} or $Q_{ESS} = 0$	Equilateral (Perfect)
$0 < Q_{EAS}$ or $Q_{ESS} \leq 0.25$	Excellent
$0.25 < Q_{EAS}$ or $Q_{ESS} \leq 0.5$	Good
$0.5 < Q_{EAS}$ or $Q_{ESS} \leq 0.75$	Fair
$0.75 < Q_{EAS}$ or $Q_{ESS} \leq 0.9$	Poor
$0.9 < Q_{EAS}$ or $Q_{ESS} < 1$	Very poor

Table 4.2: Overall relationship between skew metrics and quality of the mesh.

One of the most common failing of computational meshes is insufficient spatial resolution. In numerical studies, two approaches can be used in determining mesh suitability. The first is to compare numerical solutions with available experimental data. The approach is not suitable because it is generally difficult to precisely replicate experimental conditions in a numerical model. Furthermore, experimental studies can suffer from errors. Hence, if numerical and experimental results show good agreement, validation can be assured, but should discrepancy be observed, it is difficult to ascertain which may be the causing factors (Prakash and Ethier, 2001).

A survey of literature found no previously published data on hemodynamics in a sleeve model; hence comparison with experimental data was not possible. In this study, the second approach was used, where the computational model was meshed with increasing resolution to demonstrate mesh independency. In principle, for a sufficiently fine mesh, the solution of a well-converged simulation is independent of the numerical method and the solution algorithm chosen since different schemes generate different equivalent numerical diffusion. A mesh independent model is thus assumed to achieve sufficient spatial resolution if further refinement yields no significant improvement in results.

Model 1 (Figure 4.1 1a) was used as an illustration here where five different meshes were generated with increasing number of cells as shown in Table 4.3. Mesh independency of the solution was demonstrated by comparing velocity profiles (across locations 1,2 and 3) and wall shear stress (WSS) values (along the arterial floor, location 4) among the five meshes (Figures 4.1 1b and 4.1 1c respectively). The overall discrepancies for both velocity and WSS were small. General discrepancies

among meshes C, D and E were less than 2 % for velocity and around 5% for WSS values. A finer mesh could improve accuracy of the solution but this would be done at the expense of computational resources. Hence mesh C was considered sufficient for this study.

Mesh	A	B	C	D	E
No. of Cells	23 035	47 041	58 087	79 540	108 924

Table 4.3: Increasing density for mesh independency test.

Figure 4.11: (a) Locations where velocity (1, 2 and 3) and wall shear stress (4) values were compared to validate mesh independency, (b) velocity profile at location 2 and (c) wall shear stress variation along location 4.

4.4 Numerical Solution

After generation and inspection for quality and resolution sufficiency, the mesh generated by GAMBIT was read into the commercial solver FLUENT where operations such as setting boundary conditions, defining fluid properties and executing the solution were performed. Major steps taken in the solution process are as shown in Figure 4.12.

Figure 4.12: Flow chart illustrating major steps in solution process.

4.4.1 Discretization Techniques for Partial Differential Equations

Flows and related phenomena described by the governing laws of mass, momentum and energy conservation in partial differential equations or integral forms cannot be solved analytically except in special cases. Hence, numerical solutions employ discretization techniques to approximate these PDEs into a system of algebraic equations which can be solved by the computer. The three currently available techniques are: Finite Difference Method (FDM), Finite Element Method (FEM) and the Finite Volume Method (FVM).

Finite Difference is the easiest method for simple geometries with structured grids. At each grid point, the PDE is approximated by replacing the partial derivatives with approximations in terms of the nodal values of the function, resulting in one algebraic equation per grid node. Taylor series expansion or polynomial fitting is used to obtain approximations to the first and second derivatives of the variables with respect to the coordinates. Discretization process by the Finite Element Method is more complicated. FEM initially emerged from computational techniques used to predict stress and strain in solid structures. In FEM, a continuum field or domain is subdivided into triangular or quadrilateral cells called elements. Equilibrium equations are formulated at each element that guarantees continuity of the solution across the element boundaries. Contributions on the elemental level are then assembled over the entire domain and solved using appropriate boundary conditions.

FLUENT uses the Finite Volume Method which discretizes the integral form of the conservation equations. The solution domain is subdivided into a finite number of contiguous control volumes (CV). Consider the steady state conservation equation for transport of a scalar quantity ϕ for an arbitrary control volume V ,

$$\oint \rho \phi \vec{v} \cdot d\vec{A} = \oint \Gamma_{\phi} \nabla \phi \cdot d\vec{A} + \int_V S_{\phi} \cdot dV \quad (4-1)$$

where ρ is the density, $\vec{v}$ is the velocity vector, $\vec{A}$ is the surface area vector, Γ_{ϕ} is the diffusion coefficient for ϕ , $\nabla \phi$ is the gradient of ϕ and S_{ϕ} is the source of ϕ per unit volume.

At the centroid of each CV lies a computational node at which the variable values are to be calculated. Interpolation is used to express variable values at the CV surface in terms of the nodal (CV-center) values. Surface and volume integrals are approximated using suitable quadrature formulae which produces an algebraic equation for each CV. Discretization of Equation (4-1) on a given cell yields

$$\sum_f^{N_{faces}} \rho_f \vec{v}_f \phi_f \cdot \vec{A}_f = \sum_f^{N_{faces}} \Gamma_\phi (\nabla \phi)_n \cdot \vec{A}_f + S_\phi V \quad (4-2)$$

where N_{faces} is the number of faces enclosing cell, ϕ_f is the value of ϕ convected through face f, $\rho_f \vec{v}_f \cdot \vec{A}_f$ is the mass flux through the face, $\vec{A}_f$ is the area of face f, $(\nabla \phi)_n$ is the magnitude of $\nabla \phi$ normal to face f and V is the cell volume.

4.4.2 Boundary Conditions

Boundary conditions define flow variables at boundaries of the physical model and they can be of the Dirichlet form (values of the variable on the boundary are known constants) or the Neumann form (derivatives of the variable on the boundary are known hence giving an extra equation which can be used to find the value at the boundary). Boundary conditions applied for the numerical computation include:

- a) Velocities at all rigid walls were set equal to zero (non slip condition).
- b) At the symmetry plane, zero normal velocity and zero normal gradients of all variables were assigned. These conditions determined a zero flux across the symmetry plane which was required by the definition of symmetry.

- c) For steady conditions, the inlet velocity profile was assumed to be fully developed given by the Hagen-Poiseuille equation (3-11).
- d) For unsteady conditions, the inlet was assumed to be a fully developed pulsatile velocity profile corresponding to a specific volume flow waveform $Q(t)$ given by the Womersley solution. (3-29).
- e) At the outflow boundary, a zero diffusion flux condition was applied, where conditions of the outflow plane were extrapolated from within the domain and had no impact on the upstream flow. The extrapolation procedure updated the outflow velocity and pressure in a manner consistent with a fully developed flow assumption where there was no area change at the outflow boundary.

Steady flow parameters such as the Reynolds number and average velocity were derived from medical data by Galjee et al. (1996). In a study to quantify coronary artery bypass graft flow using magnetic resonance phase velocity mapping, they reported a mean flow rate of 87 ± 59 ml/min corresponding to a mean area of 0.28 ± 0.13 cm² in saphenous vein grafts. In the present study, three Reynolds numbers corresponding to the above parameters were determined, where blood was assumed to be Newtonian with density, $\rho = 1055$ kg/m³ and viscosity, $\mu = 0.00408$ Pa.s. The first Reynolds number was obtained using mean graft flow rate with mean cross sectional area ($Re = 80$), the second using maximum flow rate with mean graft area ($Re = 140$) and last extreme case assumed a Reynolds number approximately twice the value of the second case ($Re = 250$). Investigations were performed to study hemodynamic variations due to different Reynolds numbers in steady flow conditions.

For pulsatile flow conditions, the Womersley solution (3-29) was used to obtain a fully developed velocity profile corresponding to a specific volume flow waveform $Q(t)$. Alternatively, the average velocity waveform could be used, where $\bar{V}(t) = Q(t) / \pi R^2$. Using Fast Fourier Transform, the frequency content of the waveform (for a given fundamental frequency, ω) could be extracted. Hence, assuming $\bar{V}(t)$ to be a Fourier series with complex Fourier Coefficients B_n ,

$$\bar{V}(t) = \sum_{n=-N}^{n=N} B_n e^{in\omega t} \quad (4-3)$$

The axial velocity could be rewritten as

$$\begin{aligned} v_z(r, t) &= \bar{V}(t) \left(\frac{J_0\left(i^{\frac{3}{2}} \frac{\alpha r}{R}\right)}{J_0\left(i^{\frac{3}{2}} \alpha\right)} \right) \bigg/ \left(\frac{2J_1\left(i^{\frac{3}{2}} \alpha\right)}{i^{\frac{3}{2}} \alpha J_0\left(i^{\frac{3}{2}} \alpha\right)} \right) \\ \Rightarrow v_z(r, t) &= \sum_{n=-N}^{n=N} B_n e^{in\omega t} \left(\frac{J_0\left(i^{\frac{3}{2}} \frac{\alpha_n r}{R}\right)}{J_0\left(i^{\frac{3}{2}} \alpha_n\right)} \right) \bigg/ \left(\frac{2J_1\left(i^{\frac{3}{2}} \alpha_n\right)}{i^{\frac{3}{2}} \alpha_n J_0\left(i^{\frac{3}{2}} \alpha_n\right)} \right) \quad (4-4) \end{aligned}$$

where $\alpha_n = R\sqrt{n\omega/\nu}$ and B_n was obtained from matrix calculation using Matlab. It was observed that the zeroth term (at $n = 0$) could be simplified to $2B_0[1 - (r/R)^2]$. In addition, the terms from -1 to -N were found to be complex conjugates of those from 1 to N. Hence, if $B_{-N} = (X - iY)$, $B_N = (X + iY)$, $e^{-iN\omega t} = (\cos N\omega t - i \sin N\omega t)$ and $e^{iN\omega t} = (\cos N\omega t + i \sin N\omega t)$, then $B_{-N}e^{-iN\omega t} + B_Ne^{iN\omega t}$ would yield $(2X \cos N\omega t)$ or $Real(B_N e^{iN\omega t})$, where $Real(B_N e^{iN\omega t})$ represents real part of the complex value. Based on the above explanations, equation (4-4) can be further simplified to

$$v_z(r,t) = 2B_0 \left[1 - \left(\frac{r}{R} \right)^2 \right] + 2 \times \text{Re} \left[\sum_{n=1}^N B_n \frac{\left(1 - \frac{J_0 \left(i^{\frac{3}{2}} \frac{\alpha_n r}{R} \right)}{J_0 \left(i^{\frac{3}{2}} \alpha_n \right)} \right)}{\left(1 - \frac{2J_1 \left(i^{\frac{3}{2}} \alpha_n \right)}{i^{\frac{3}{2}} \alpha_n J_0 \left(i^{\frac{3}{2}} \alpha_n \right)} \right)} e^{in\omega t} \right] \quad (4-5)$$

A user defined function (UDF) to calculate velocity values at the inlet boundary was provided by Zhang (2002) and modified as shown in Appendix B. Prior to computation of flow in the sleeve model, a validation study was performed to assess the accuracy of the numerical method. A circular artery of diameter 3 mm and length 30 mm was constructed and discretized into 23 250 hexahedral cells and 26 576 nodes (Figure 4.13). An arbitrary average velocity waveform of $\bar{V}(t) = 0.1(1 + \sin \omega t)$ was imposed with period $T = 0.1$ s (Figure 4.14). As observed in Figure 4.15, the numerical solution resolved the theoretical velocity profile well overall with a maximum error of less than 6%. Since analytical solution was known, further improvement in results could be achieved by increasing the mesh density.

Figure 4.13: Circular artery with hexahedral mesh.

Figure 4.14: Average Velocity Profile of $\bar{V}(t) = 0.1(1 + \sin \omega t)$

Figure 4.15: Comparison of analytical and computational results for Womersley solution.

It is a known fact that flow waveforms vary at different segments of the cardiovascular system. For an accurate simulation, it is highly important that the waveform used is representative of that in a saphenous vein bypass graft. Galjee et al. (1996) using magnetic resonance imaging (MRI) phase velocity mapping demonstrated a biphasic forward flow pattern with one peak in early systole and one peak in early diastole. No significant differences were found between flow characteristics of left anterior descending (LAD), circumflex (CX) and right coronary artery (RCA) grafts. They also reported no quantitative differences between flow patterns of single and sequential grafts.

A magnetic resonance estimate of flow rate in a saphenous vein graft provided by Galjee et al. (1996) was used. The original waveform consisted of 17 equally spaced

measurements over 0.8s. Due to steep increase in flow rate at the end of the flow cycle, Leask (2002) suggested that two points be inserted to smooth the waveform. The flow values were based on an assumed mean cross sectional area of 0.28 cm^2 (or diameter of 6 mm) as reported by Galjee et al. (1996). For a graft diameter of 3 mm as used in the current study, the new flow rate and corresponding velocity values were obtained by similitude using constant mean Reynolds numbers. The final waveform with average velocity values adapted for this study is as shown in Figure 4.16.

Figure 4.16: Average velocity waveform from Galjee et al. (1996) and Leask (2002).

It was observed however that apart from the biphasic pattern, velocity values were highly fluctuating over time. Such variability could be due to noise during the in vivo measurements. Comparison with flow measurements in different studies showed similar biphasic patterns with different amount of variability. For a preliminary study, the main objective was to compare hemodynamics in different sleeve geometries due to biphasic nature of the waveform. Hence, it was suggested that the waveform be further smoothed such that main information corresponding to the two peaks were retained while the remaining oscillating points be neglected. The final waveform used for the present study is as shown in Figure 4.17.

Figure4.17: Averagevelocity waveformused in the present study.

Characteristics of this waveform are tabulated as shown in Table 4.4. Figure 4.18 illustrates variation of velocity profile (two dimensional along diameter of graft inlet) with time. Three dimensional velocity profiles at specific time values were generated as shown in Figure 4.19, including the initial acceleration (a), velocity profiles at the two peak values (b) and (d), the first minimum value (c) as well as negative flow profiles at the deceleration ends (e) and (f). Flow reversal was also observed near the graft wall (e), a phenomenon commonly observed in pulsatile flow as explained in Section 3.3.1.

Figure 4.18: Variation of two dimensional velocity profiles with time.

Description	Characteristics
1 st Peak Flow	$Re = 95$ at $t = 0.125\text{ s}$
2 nd Peak Flow	$Re = 83$ at $t = 0.45\text{ s}$
1 st Peak Reverse Flow	$Re = 17$ at $t = 0.325\text{ s}$
2 nd Peak Reverse Flow	$Re = 17$ at $t = 0.875\text{ s}$
Frequency	$f = 1.1\text{ Hz}$
Period	$T = 0.9\text{ s}$
Womersley Number	$\alpha = 2$

Table 4.4: Characteristics of Average Velocity Waveform.

Figure 4.19: Three dimensional graft inlet velocity at specific times of a) 0.025 s, b) 0.125 s, c) 0.325 s, d) 0.45 s, e) 0.85 s and f) 0.875 s.

4.4.3 Segregated vs Coupled Solver

Three solver formulations were available from FLUENT: segregated, coupled implicit and coupled explicit. The segregated and coupled approaches differed in the way that the continuity, momentum, and (where appropriate) energy and species equations were solved: the segregated solver solved these equations sequentially (i.e., segregated from one another), while the coupled solver solved them simultaneously (i.e., coupled together). The segregated solver was traditionally used for incompressible and mildly compressible flows while the coupled approach was originally designed for high-speed compressible flows. Since the present study involved incompressible flow at low speed, the segregated solver was used. Due to non-linear nature of the governing equations, several iterations of the solution loop had to be performed before a converged solution was obtained. Each iteration consisted of steps as shown in Figure 4.20. These steps were continued until convergence criteria were met.

Figure4.20: Iteration procedures using the segregated solver.

Non-linear governing equations were linearized either implicitly or explicitly to produce a system of equations for the dependent variables in every computational cell. In implicit linearization, the unknown value in each cell was computed using a relation that included both existing and unknown values from neighboring cells. Hence, each unknown would appear in more than one equation in the system and they were found by solving all equations simultaneously. For explicit linearization, the unknown value in each cell was related to only existing values (only one unknown in each equation). Therefore the equations could be solved one at a time to give the unknown quantities.

The segregated solver however had no explicit option. Each discrete governing equation was linearized implicitly with respect to that equation's dependent variable resulting in a "scalar" system of linear equations with one equation for each cell in the domain. A point implicit (Gauss-Seidel) linear equation solver was used in conjunction with an algebraic multigrid (AMG) method to solve the resultant scalar system of equations for the dependent variable in each cell. In summary, the segregated approach solves for a single variable field (e.g., p) by considering all cells at the same time. It then solves for the next variable field by again considering all cells at the same time, and so on.

4.4.4 Upwind Scheme for Convection Terms

Discrete values of the scalar ϕ were stored at the cell centers (c_0 and c_1) as shown in Figure 4.21. However, face values ϕ_f were required for the convection terms in Equation (4-2) and had to be interpolated from the cell center values. This was accomplished using an upwind scheme where the face value ϕ_f was derived from

quantities in the cell upstream (or “upwind”), relative to the direction of the normal velocity v_n in Equation (4-2). The first-order, second-order, power law, QUICK (Quadratic Upwind Interpolation of Convective Kinematics) and central differencing schemes were available for upwinding.

Figure 4.2 1 : Control volume used in discretization of a scalar transport equation.

When the flow is aligned with the grid (e.g., laminar flow in a cylindrical conduit with hexahedral cells) the first-order upwind discretization may be acceptable. However, when the flow is not aligned with the grid (i.e., when it crosses the grid lines obliquely), the first-order convective discretization would increase the numerical discretization error (numerical diffusion) and a second-order upwind is required. The QUICK scheme may provide better accuracy than the second-order scheme for rotating or swirling flows while the power law generally yields the same accuracy as the first-order scheme. The central-differencing scheme is available only when using the LES (Large Eddy Simulation) turbulence model with mesh spacing fine enough that the magnitude of the local Peclet number is less than 1. Although flow was mainly aligned with the hexahedral mesh in the graft and host artery sections, flow alignment was never achieved in the sleeve region due to tetrahedral meshing (as mentioned in Section 4.3). Therefore, a second-order upwind was more appropriate for overall computation. In addition, flow was not expected to be rotating or swirling, neither was turbulence expected, hence the remaining schemes were not required for this study.

4.4.5 Pressure Interpolation Scheme

Using the Finite Volume Method as described in Section 4.4.1, the conservation equations in integral form could be discretized. As an example, the x-momentum equation was obtained by setting $\phi = u$:

$$a_p u = \sum_{nb} a_{nb} u_{nb} + \sum p_f A \cdot \hat{i} + S \quad (4-6)$$

where a_p and u are the coefficient and velocity values at the cell center, a_{nb} and u_{nb} are the influence coefficients and velocity values at the neighboring cells and P_f is the face pressure. Equation (4-6) required the value of face pressure which was not known and must be obtained as part of the solution. Since both pressure and velocity values were stored at cell centers c_0 and c_1 (Figure 4.2 1), an interpolation scheme was used to compute the face values from the cell values. For pressure interpolation, FLUENT offers the standard, second-order, body-force-weighted and PRESTO (PREssure STaggering Option) schemes.

The standard scheme interpolates the pressure values at faces using momentum equation coefficients and works well as long as pressure variation between cell centers is smooth. Where large jumps or large gradients in momentum source terms between control volumes exists, the pressure profile has a high gradient at the cell face, and cannot be interpolated using this scheme. The linear scheme on the other hand computes face pressure as the average of the pressure values in the adjacent cells. The second-order scheme may provide some improvement over the previous two schemes, but it may have some trouble if used at the start of a calculation and/or with a bad mesh. The body-force-weighted scheme is commonly used for problems involving large body forces while The PRESTO scheme is adapted for flows with

high swirl, high-Rayleigh-number natural convection and high speed rotating flows. Since body forces, swirl and rotation in the present study were negligible, either the standard, linear or second-order scheme was suitable. A validation study was performed by subjecting one of the distal anastomotic models to the three pressure interpolation schemes. It was shown that all three schemes produced similar results and convergence rate. Hence the default standard scheme was used for pressure interpolation.

4.4.6 Pressure-Velocity Coupling Scheme

The continuity equation in integral form may be integrated over the control volume to yield the following discrete equation

$$\sum_J^{N_{faces}} J_f A_f = 0 \quad (4-7)$$

where J_f is the mass flux through face f . The segregated solver solves the momentum and continuity equations in a sequential manner where the continuity equation is used as an equation for pressure. However, pressure does not appear explicitly in the above equation (4-7) for incompressible flows since density is not directly related to pressure. The SIMPLE (Semi-Implicit Method for Pressure-Linked Equations) family of algorithms is used for introducing pressure into the continuity equation. Three pressure-velocity coupling methods are provided: SIMPLE, SIMPLER and PISO (Pressure-Implicit with Splitting of Operators). Steady-state calculations will generally use SIMPLE or SIMPLER, while PISO is recommended for transient calculations.

Although SIMPLE is the default scheme, most flow problems can be benefited from using SIMPLEC, particularly because of the increased under-relaxation that can be applied. For relatively uncomplicated problems in which convergence is limited by the pressure-velocity coupling, a converged solution can be obtained more quickly using SIMPLEC. Using one of the distal anastomotic models for steady flow, numerical computations using SIMPLEC was shown to converge faster than that using SIMPLE. While PISO is recommended for transient problems, a trial run using both SIMPLEC and PISO did not yield any difference in convergence. Hence SIMPLEC was used for all computations in this study.

Due to nonlinearity of the equation set being solved, it is necessary to control the change of the scalar quantity ϕ . This is achieved by under-relaxation, which reduces the change of ϕ produced during each iteration, where the new value of the variable ϕ within a cell depends upon the old value such that

$$\phi = \phi_{old} + \beta \Delta \phi \quad (4-8)$$

where β is the under-relaxation factor. In the present study, under-relaxation factors for both pressure and momentum equations were varied to study their effects on convergence rate. It was concluded that values of under-relaxation factors of 1 (for pressure equations) and 0.3 (for momentum equations) were yield the fastest convergence results.

4.4.7 Convergence Criteria

As discussed in Section 4.4.3, the iteration procedure was performed using the segregated solver. The residual sum for each of the conserved variables was computed

and stored at the end of each solver iteration. On the computer with finite precision, this residual would decay to some small “round-off” value before leveling out.

The conservation equation for a general variable ϕ at a cell P can be written as

$$a_p \phi_p = \sum_{nb} a_{nb} \phi_{nb} + b \quad (4-9)$$

where a_p is the cell center coefficient defined as $a_p = \sum_{nb} a_{nb} - S_p$, ϕ_p is the cell center variable, a_{nb} is the influence coefficients for the neighboring cells and b is the contribution of the constant part of the source term S_c where $S = S_c + S_p \phi$ and of the boundary conditions. The residual term R^ϕ computed by FLUENT’s segregated solver is the imbalance in Equation 4-9 summed over all the computational cells P . This "unsealed" residual term may be written as

$$R^\phi = \sum_{cells P} \left| \sum_{nb} a_{nb} \phi_{nb} + b - a_p \phi_p \right| \quad (4-10)$$

In general, it is difficult to judge convergence by examining the residuals defined by Equation (4-10) since no scaling is employed. This is especially true in enclosed flows such as natural convection in a room where there is no inlet flow rate of ϕ with which to compare the residual. FLUENT thus scales the residual using a scaling factor representative of the flow rate of ϕ through this domain, defined as

$$R^\phi = \frac{\sum_{cells P} \left| \sum_{nb} a_{nb} \phi_{nb} + b - a_p \phi_p \right|}{\sum_{cells P} |a_p \phi_p|} \quad (4-11)$$

For the momentum equations, the denominator term $a_P \phi_P$ is replaced by $a_P v_P$, where v_P is the magnitude of velocity at cell P . This scaled residual was more appropriate and was thus used as a convergence indicator for the present research. FLUENT further suggested that for most flow problems, the default convergence criterion of scaled residuals at 10^{-3} for all equations is sufficient, with the exception of energy and P-1 equations, for which the criterion is 10^{-6} . In the current research, the convergence criterion for both velocity and pressure were set at 10^{-5} for steady flow and 5×10^{-5} for unsteady flow conditions.

4.4.8 Time Step Size

For a fully implicit linearization using the segregated solver, no stability criterion was required for the time step size, Δt . However, to model transient phenomena accurately, a time step size at least an order of magnitude smaller than the smallest time constant in the system was necessary. A suggested way of judging the choice of Δt was to observe the number of iterations needed to converge at each time step, the ideal number being 10 to 20. If substantially more iterations were required by the solver, the Δt would be deemed too large while too few time steps on the other hand implied that Δt could be increased. In the present study, Δt of 0.1s, 0.05s, 0.025s and 0.0125s were tested individually and 0.025s yielded an approximate 25 iterations per time step. In addition, a flow results showed time step independency for values of Δt smaller than 0.1s. Hence, $\Delta t = 0.025$ s was chosen as an appropriate time step size for this investigation. To eliminate start up effects of transient flow, the computation was carried out over at least three periods, where velocity changes beyond that were less than 0.001%.

4.4.9 Wall Shear Stress

For no-slip wall conditions) FLUENT uses properties of the flow adjacent to the wall boundary to predict shear stresses on the fluid at the wall. In laminar flows, the wall shear stress is defined by the normal velocity gradient at the wall as

$$\tau_w = \mu \frac{\partial u}{\partial n}$$

where $\frac{\partial u}{\partial n}$ is the normal velocity gradient at the wall and $\mu = 0.00408$ Pa.s is the viscosity of blood. To obtain accurate velocity gradients at the wall, the mesh near the wall must be sufficiently fine to accurately represent flow in the boundary layer. Schlichting (1979) proposed the following guidelines for the appropriate placement of near-wall nodes in laminar flows, which is based upon the Blasius solution for laminar flow over a flat plate at zero incidence,

$$y_p \sqrt{\frac{u_\infty}{\nu x}} \leq 1$$

where y_p is the distance from adjacent cell centroid to the wall, u_∞ is the free-stream velocity, ν is the fluid kinematic viscosity and x is the distance along the wall from the starting point of the boundary layer. In the present study, the kinematic viscosity was 3.867×10^{-6} m²/s and the free stream velocity was less than 0.6 m/s. Choosing a length $x = 2$ mm, the maximum distance from the adjacent cell centroid to the wall was not to exceed $y_p = 0.11$ mm. The largest cell centroid to wall distance in the meshed models used in the present study was 0.095 mm. In addition, meshes in the

sleeve region were exponentially distributed to ensure greater density near the wall region, hence justifying the wall shear stress requirements.

4.5 Postprocessing

FLUENT stores most variables in cells, where the entire region contained within the cell share the same value. On a boundary, the cell value is the value in the cell adjacent to the boundary. Node values are computed by averaging the data of all the cells that share the same node (Figure 4.22). Although FLUENT has postprocessing capabilities of its own, Tecplot offers greater flexibility and variety for visualizing a wide range of technical data. In the present study, cell values were exported to Tecplot where vector and contour plots of the velocity and wall shear stress values were performed and analyzed.

Figure 4.22: Computation of node values from surrounding cells.

Chapter 5: Results and Discussion of Geometrical Variations (Steady Flow)

Although flow in arteries is generally pulsatile in nature, steady flow analyses are still widely pursued as a preliminary study of hemodynamics in blood vessels as they provide insight into different flow patterns corresponding to changes in geometries and flow conditions. Prior to investigations using Womersley solution for pulsatile flow, steady flow analyses were carried out to understand effects of different proposed sleeve geometries on flow characteristics. The Hagen-Poiseuille equation was used to compute velocity values which act as inlet boundary conditions for the steady model. Three flow rates were modeled, using Reynolds numbers 80, 140 and 250. The sleeve models studied vary in diameters and height as shown in Table 5.1 and Figure 5.1. The inner and outer walls were defined from the graft inlet to the proximal and distal artery ends respectively while arterial floor stretched from proximal to distal artery. Positive wall shear stress is represented by flow in positive x direction while negative wall shear stress represented flow in negative x direction. Terminologies used in discussing flow at various sites are also shown in Figure 5.1. Both flow patterns and wall shear stress variations corresponding to different geometries and Reynolds numbers were examined and discussed in the following sections.

Model	D_A	D_G	D_S	H_S	Remarks
1	2.6 mm	3 mm	D_G	D_A	Baseline Model
2	2.6 mm	3 mm	D_G	$1.5 D_A$	Increased Height
3	2.6 mm	3 mm	$1.25 D_G$	D_A	Increased Diameter
4	2.6 mm	3 mm	$1.25 D_G$	$1.5 D_A$	Increased Diameter and Height
5	2.6 mm	3 mm	$1.5 D_G$	$2 D_A$	Increased Diameter and Height

Table 5.1: Dimensions of sleeve models used in steady flow study.

Figure 5.1: Terminologies used in steady flow study.

5.1 Flow Characteristics and Wall Shear Stress Variation in Model 1

Before comparing hemodynamics of varying geometries, a study was conducted on the baseline model (Model 1) to understand flow characteristics within the proposed sleeve model as well as wall shear stress variations along the outer wall, inner wall and arterial floor. Fully developed flow was imposed as a boundary condition in the graft inlet and the parabolic profile persisted for 10 mm downstream. Apart from velocity vectors as shown in Figure 5.2 (c), the parabolic nature was verified by observing that the inner and outer wall shared the same shear stress values from 0 mm to 10 mm as demonstrated in Figure 5.3 (b) and (c) respectively.

Variations in wall shear stress began with the onset of curvature at the sleeve entrance. Along the inner wall, shear stresses increased (from $x_2 = 10$ mm) till it reached a peak approximately at $x_2 = 13$ mm. Bulk flow entered the sleeve almost horizontally as shown in Figure 5.2a, leaving a region of low flow along the inner wall due to the higher momentum of the flow. This resulted in wall shear stress

plummeting instantly to approximately zero. A stagnation point along the inner wall was located at about $x_2 = 15$ mm with flow beyond this point to be in a reversed direction. Flow reversal was due to velocity vectors below the heel exhibiting a recirculating pattern. However, due to extremely small magnitude (of order 10^{-3} m/s) of the flow, fluid in this region was considered relatively stagnant, with negative shear stresses close to zero. The trend was similar for $Re = 80$ and $Re = 140$. At a higher flow rate however ($Re = 250$), stagnation point along the inner wall was more clearly visible at $x_2 = 13.8$ mm. The weak recirculating velocity vectors below the heel in the proximal artery had increased in magnitude forming a distinctive vortex as can be observed in Figure 5.2 (c), resulting in higher negative wall shear stresses as seen in Figure 5.3 (b) along the inner wall for $Re = 250$.

Along the outer wall, between $x_3 = 10$ mm to 13 mm, shear stresses decreased to a minimum point then remained constantly low till approximately $x_3 = 17$ mm. Beyond the point, wall shear stresses increased drastically to reach peak values at the toe region (approximately $x_3 = 18.7$ mm). The drastic rise in shear stresses was due to impingement of high velocity fluid traveling almost parallel to the outer wall. High momentum fluid traveling along the toe region then entered the distal artery diagonally, resulting in a low flow region along the top outer wall. Such fluid motion explained the rapid decrease of shear stresses beyond the peak to a minimum point. Similar patterns were observed in $Re = 80$ and $Re = 140$. At $Re = 250$ however, reversed flow was observed between $x_3 = 20.5$ and 22 mm which corresponded to negative shear stresses along the outer wall as seen in Figure 5.3 (c). Flow along the distal artery section then progressively became aligned with the cylindrical geometry before attaining fully developed profile further downstream.

Along the arterial floor, stagnation point was located approximately at the same site for all flow rates. Wall shear stresses were negative to the left of the stagnation point due to flow reversal in the region below the heel at $Re = 250$ as demonstrated in Figure 5.3 (a). Flow reversal was more obvious for increasing Reynolds numbers, with $Re = 250$ demonstrating a distinctively visible vortex that stretched from the floor all the way to the inner wall. The phenomenon was caused by high momentum fluid impinging the arterial floor almost perpendicularly causing the flow to bifurcate into two streams, where part of the high velocity flow was directed towards the distal section and the other flow into the proximal section. Fluid traveling towards the proximal section encountered the fully occluded stenosis and was forced to change direction towards the inner wall. The upward flow then reattached to the inner wall with the downward flow at the stagnation point of the inner wall. The series of fluid motions resulted in a vortex formed just below the heel. For lower flow rates ($Re = 80$ and $Re = 140$), although the velocity vectors did exhibit some recirculating patterns, the velocity magnitudes were so small that flow in that region could be considered stagnant. For all flow rates (Figure 5.3a), a region of instability was observed between $x_l = 17$ mm and 20 mm along the arterial floor, characterized by fluctuating wall shear stresses. The fluctuations were caused by alignment process of the incoming downward flow attempting to change their flow directions to a path parallel to the arterial floor. The particles upon collision with the arterial floor were reflected slightly upwards but the motion was restricted by fluid above, resulting in a downward motion slightly downstream. The fluctuating velocity directions resulted in fluctuation of wall shear stresses as observed in Figures 5.3 (a). Beyond the $x_l = 20$ mm mark where flow entered the distal artery, wall shear stresses began to stabilize as flow gradually assumed the fully developed profile downstream.

Figure 5.2: Flow Characteristics in Model 1 at (a) Re = 80 and (b) Re = 140.

Figure 5.2 (c): Flow Characteristics in Model 1 at (c) $Re = 250$.

Figure 5.3 (a): Wall shear stress variation along arterial floor for Model 1.

(b)

(c)

Figure 5.3: Wall shear stress variation along (b) inner wall and (c) outer wall for Model 1.

5.2 Flow Characteristics and Wall Shear Stress Variation in Model 2

Model 2 was designed to incorporate a larger sleeve volume (about 10% increase from Model 1) through an increment in sleeve height. At $Re = 80$ and 140 as shown in Figure 5.4 (a) and (b) respectively, flow patterns were similar to Model 1 with general characteristics such as reversed flow along arterial floor towards proximal end, separate from the arterial floor and reattachment along inner wall. Stagnation points were also located at the same sites for both models. Wall shear stress variations exhibited small differences, with Model 2 registering slightly higher peak values along the arterial floor and inner wall as shown in Figure 5.5 (a) and (b) respectively. Instabilities along the arterial floor were also observed but in a slightly smaller range. Along the outer wall, shear stresses peaked at a lower value than those in Model 1 but were distributed over a larger distance as demonstrated in Figure 5.5 (c). This was due to an increase in outer wall length between the sleeve entrance and toe region as a result of height increase. High momentum fluid entered the sleeve of both models at the same angle but impinged the outer wall of Model 2 at a position closer to the entrance of the sleeve as compared to Model 1 (which was near the toe). Fluid then traveled downstream along the outer wall with decreased velocity before exiting the sleeve beyond the toe region, hence resulting in redistribution of wall shear stresses.

At $Re = 250$, a small recirculating region was observed near the inner wall approximately 1 mm from the sleeve entrance. A similar phenomenon was observed at the distal artery entrance (for both Models 1 and 2 at $Re = 250$). This was due to departure of high momentum flow from the wall creating a region of low pressure hence resulting in some fluid particles to take a reversed path. However, the recirculation was very weak in magnitude as compared to the bulk flow.

Figure 5.4: Flow Characteristics in Model 2 at (a) $Re = 80$ and (b) $Re = 140$.

Figure 5.4 (c): Flow Characteristics in Model 2 at $Re = 250$.

Figure 5.5 (a): Wall shear stress variation along the arterial floor for Model 2.

(b)

(c)

Figure 5.5: Wall shear stress variation along (b) inner wall and (c) outer wall for Model 2.

5.3 Flow Characteristics and Wall Shear Stress Variation in Model 3

Sleeve diameter of Model 3 was 1.25 times larger than that of Model 1 while the height was kept constant. The increase in cross-sectional area would result in an increase in sleeve volume (about 20% increase as compared to Model 1). Flow patterns were generally the same as Model 1 for $Re = 80$, with the exception of

reduced velocity magnitudes and peak wall shear stresses within the sleeve model. This was consistent with mass conservation laws since a larger cross-sectional area would result in reduced velocity and subsequently, reduced wall shear stress values. Along the outer wall, shear stress peaked at the toe region, with a larger peak preceding the smaller one as demonstrated in Figure 5.7 (c). Such variation was the result of a design flaw due to insufficient rounding of the sleeve contour which should be avoided in future studies. The irregularity however only caused slight perturbation to the flow structure. With increasing flow rate ($Re = 140$), a phenomenon resembling a “sourcepoint” was observed 1.5 mm downstream from the sleeve entrance in Figure 5.6 (b). The flow structure was due to increasing secondary flow effects as fluid moved along an expanding cross-sectional area and will be discussed further in Section 5.5.1. Further down below the heel, a weak recirculating region formed by slow moving fluid was observed. Stagnation points along the arterial floor and inner wall were more proximal in comparison to Model 1.

When flow was increased to $Re = 250$, the recirculating source-like structure had departed from the wall region and moved distally into the sleeve central. A slightly complicated flow pattern existed as a result of interaction between the source-like structure and the vortex located just below the toe which had grown in size and magnitude. In Models 1 and 2, fluid entered the distal artery at a large angle (with respect to the floor), inducing a region of reversed flow at the top of the distal artery entrance. In Model 3, the recirculating region was absent because fluid entered the distal artery at a smaller angle, hence perfusion of the entire distal artery entrance was possible without leaving a region of recirculating flow at the top.

Figure 5.6: Flow Characteristics in Model 3 at (a) $Re = 80$ and (b) $Re = 140$.

Figure 5.6 (c): Flow Characteristics in Model 3 at Re = 250.

Figure 5.7 (a): Wall shear stress variation along the arterial floor for Model 3.

Figure 5.7: Wall shear stress variation along (b) inner wall and (c) outer wall for Model 3.

5.4 Flow Characteristics and Wall Shear Stress Variation in Models 4 and 5

Sleeve volume was increased further in Models 4 and 5 through an increment in both sleeve diameter and height simultaneously. At low flow rates ($Re = 80$), flow patterns were generally similar to that of Model 1, with the exception of lower flow velocity due to the expanding cross-sectional area, which was much larger than those of the previous three models.

When flow rate was increased to ($Re = 140$), the recirculating source-like structure was more distinct in Models 4 and 5 as shown in Figure 5.8 (b and c) and 5.10 (b and c) respectively, having moved distally from the inner wall towards the centre of the sleeve. A weak recirculating region comprising slow moving fluid particles was formed below the heel for both models. Stagnation points along the arterial floor and inner wall were observed to be more proximal to the heel as compared to Model 1. Between Models 4 and 5, flow was typically slower (resulting in lower wall shear stress) in the latter due to the larger volume of perfusion required.

At maximum flow rate of $Re = 250$, the source-like structure had shifted to near central location within the sleeve and a complicated flow pattern was established between the source point and the vortex. At the distal artery inlet of Model 4, no recirculating zone was presented in Figure 5.8 (c) while for Model 5, a small recirculating zone (smaller in size as compared to Model 1) was observed in Figure 5.10 (c). Shear stresses along the three walls exhibited the same patterns of variation in both Figure 5.9 and 5.11 although Model 5 showed slightly lower shear stress values overall due to a larger cross-sectional area.

Figure 5.8: Flow Characteristics in Model 4 at (a) Re = 80 and (b) Re = 140.

Figure 5.8 (c): Flow Characteristics in Model 4 $Re = 250$.

Figure 5.9 (a): Wall shear stress variation along arterial floor for Model 4.

Figure5.9: Wall shear stress variation along (b) inner wall and (c) outer wall for Model 4.

Figure 5.10 (a): Flow Characteristics in Model 5 at Re = 80.

Figure 5.10: Flow Characteristics in Model 5 at (b)Re = 140 and (c) Re = 250.

(a)

(b)

(c)

Figure 5.11 : Wall shear stress variation along (a) arterial floor, (b) inner wall and (c) outer wall for Model 5.

5.5 Discussion

Flow results of the various models were presented earlier. This section seeks to discuss some interesting flow characteristics of the sleeve models as well as to evaluate them based on steady flow conditions. Comparisons of results with published literature will be performed.

5.5.1 Secondary Flow in Sleeve Models

Secondary motions within the sleeve models were examined at planes A, B and C as shown in Figure 5.12 for $Re = 80, 140$ and 250 in Model 1. As observed from Figure 5.13, there is strong positive correlation between flow rate and the secondary flows. In regions near the x-axis, flow was predominantly forward, hence explaining the skewing of fluid towards the sleeve outer wall. Near the side walls of the sleeve at around $x = 0.0152$ m, $z = 0.0012$ m, a vortex was observed in plane A which increased in size and intensity with flow rate. In three-dimensional form, this represented a swirling motion as reported by several researchers (White et al., 1993, Hughes and How, 1996, Inzoli et al., 1996 and Moore et al., 1999). Further downstream, in planes B and C, the vortex gradually disappeared yet flow was still predominantly forward and entering the distal artery diagonally.

Figure 5.12: (a) Definition of symmetry and secondary plane and (b) study of secondary motion in planes A, B and C.

Figure 5.13: Secondary flows in Model I (a) Re = 80, (b) Re = 140 and (c) Re = 250.

As observed in Models 3, 4 and 5, at higher flow rates of $Re = 250$, flow seemed to spiral out from a source-like structure. The phenomenon was not created by an actual source but rather due to influence of secondary flow components. It was interesting to observe that such characteristics were more distinct in sleeves with an increased diameter. This was due to overall decrease in velocity as a result of an increase in cross-sectional area. As such, at certain point of fluid flow in the sleeve, magnitudes of secondary components were comparable to that of axial components. Model 5 is used to explain the phenomenon with secondary planes as shown in Figure 5.14.

Secondary flow components were negligibly small in the graft section since axial flow was dominant (Figure 5.15). As fluid entered the sleeve, the onset of curvature coupled by an increase in cross-sectional area resulted in an increase in secondary flow components as observed in Figure 5.16 (a). This resulted in a three dimensional helical flow as commonly observed in most conventional bypass models at the distal end. As the cross-sectional geometry gradually changed from circular to a more elliptical shape, the vortices in the cross-sectional plane were compressed in z -direction but extended in x -direction. Flow components in the x - y plane were generally directed towards the outer wall. Further downstream, flow was observed to impinge the x - y plane resulting in separation of fluid into the forward and backward components. When viewed in the x - y plane, the phenomenon resembled fluid branching out of a source point, but this in fact was due to influence of secondary flow. Furthermore, the source like flow pattern is in fact due to the construction of velocity vectors into streamlines. As a vortex centre is formed, the vector gradient at the vortex centre become a node, due to the indeterminate of the gradient at the vortex centre which is a critical point in the phase plane (Kreyszig, 1999).

Figure 5.14: Secondary planes in Model 5.

Figure 5.15: Secondary flow within inlet graft.

Figure 5.16: Secondary flow patterns in Model 5.

5.5.2 Comparisons with Published Literature

It is not the intention of the present study to design sleeve models that replicate flow characteristics in conventional bypass models because these conventional models have failed in providing adequately good hemodynamics for long term patency rates of bypass surgeries. However, models used in this study did not exhibit extremely large deviations in terms of geometries or configurations; hence general flow characteristics in distal anastomosis should still be observed. Qualitative comparisons would therefore be useful to ensure validity of the simulation studies.

Good agreement in steady flow patterns was found between the present study and results reported by several researchers, including Pietrabissa et al. (1990), White et al. (1993), Fei et al. (1994), Inzoli et al. (1996), Moore et al. (1999) and Kute and Vorp (2001). General characteristics included strong skewing of particles towards hood (or sleeve outer wall in our case) and arterial floor, helical particle trajectories due to secondary motion, increased strength of secondary velocity components with greater flow rates, location of stagnation point along the arterial floor approximately at the mid-point opposite the graft and presence of recirculation at the distal artery entrance.

Fei et al. (1994) conducted steady flow studies on three-dimensional numerical models to investigate effects of angle and flow rate on hemodynamics. Enlarged views of the velocity plots for 45° distal anastomotic models with $Re = 100$ and 205 are as shown in Figure 5.17. Apart from general flow patterns, they observed a weak vortex formed within the entire diameter of the closed end of the artery just upstream of the stagnation point. A very small separated region was seen along the inner wall of

the artery just downstream of the anastomosis (corresponding to distal artery entrance for the present study) for $Re = 205$. They further noticed that such characteristic features ceased to exist for $Re = 100$ with angles less than 60° and $Re = 205$ with angles less than 45° . Although the present sleeve geometries were generally different, results from Fei et al. (1994) could be extrapolated to validate results obtained in this study.

Figure 5.17: Velocity vectors in symmetry plane for 45° anastomosis at (a) $Re = 100$ and (b) $Re = 205$ (Fei et al., 1994)

Adapting the sleeve model to geometrical definition by Fei et al. (1994) would yield a 90° bypass angle (caused by intersection of the cuff-like geometry with the host artery). This would indicate the presence of a vortex below the heel region at Reynolds numbers between 100 and 205. Steady flow results from the current study showed the existence of a vortex structure below the heel for all models at $Re = 80$, 140 and 250, although the magnitude and intensity of the vortex may vary at different flow rate. This may explain why vortices were more likely formed in Miller cuffs than conventional bypass models. In the former, interposition vein cuffs are always perpendicular to the host artery, hence resembling a 90° bypass angle. On the other hand, researchers emphasized the need for a low bypass angle for conventional

models to improve perfusion capabilities. From results by Fei et al. (1994), a lower bypass angle would require a higher Reynolds number for generation of vortex which may not be practical in physiological terms since we have little control over the flow rates. Hence, while low angles may be suitable for conventional bypass models, a large angle may be desirable for a prosthetic sleeve such that a vortex generated artificially may enhance washing within the sleeve itself. In conventional terms, a 90° anastomosis would resemble a T-bifurcation which is not a suitable implant design due to its sharp protrusion. A better alternative to achieve this high angle design would thus be through the use of the cuff-like model.

Kute and Vorp in 2001 published numerical studies on the effects of proximal artery flow on hemodynamics at the distal anastomosis (Figure 5.18). Three cases were studied, using prograde, zero and retrograde flow in the proximal artery. However, since our study concerned only with fully occluded model, comparisons will be made with the zero proximal artery flow model only. The model used in their study consisted of rigid, 4 mm cylinders intersecting at a 30° angle. The Reynolds number used was approximately 150 based on density (1000 kg/m^3) and viscosity (3.5 cP) of the fluid as well as a flow rate of 100 cc/min.

Figure 5.18: Model used by Kute and Vorp(2001) for steady flow studies

Flow characteristics (Figure 5.19) bear similar resemblance when compared to the sleeve models. As observed from Figure 5.20, high wall shear stresses were observed in regions near the toe (2 Pa or 20 dynes/cm^2), arterial floor opposite the toe (1.5 Pa to 1.75 Pa or 15 dynes/cm^2 to 17.5 dynes/cm^2) and heel (0.75 Pa to 1.2 Pa or 7.5 dynes/cm^2 to 12 dynes/cm^2). In comparison, using $\text{Re} = 140$ in the current study, peak shear stresses along the toe region were 15 Pa , 13 Pa , 6 Pa , 10 Pa and 8 Pa for Models 1, 2, 3, 4 and 5 respectively. Along the arterial floor, maximum shear stresses were 8 Pa , 9 Pa , 6.6 Pa , 7.5 Pa and 8 Pa for Models 1, 2, 3, 4 and 5 respectively. Despite using a lower flow rate, WSS values in these regions were much higher due to presence of the cuff-like geometry intersecting the host artery at near 90° , disrupting smooth flow about to exit the sleeve. At this point, advantages of such elevated shear stresses were still unclear (as true physiological flow is pulsatile in nature) although it is a widely accepted fact that regions of higher wall shear stresses are less prone to thrombus deposition. In addition, since for a steady flow analysis, quantitative values should not be taken as true representations of physiological cases since blood flow in the human body is highly unsteady in nature.

Figure 5.19: Flow patterns in zero flow model (Kute and Vorp, 2001)

Figure 5.20: Wall shear stress distribution in zero flow model (Kute and Vorp, 2001)

5.5.3 Evaluation of Sleeve Models Under Steady Flow Conditions

Five models were investigated in this study with Model 1 representing a baseline model where other models were varied by increasing either the sleeve diameter or height independently or simultaneously. However, it was to be reiterated that Model 1 was not the smallest possible volume to be designed since an even smaller volume could be achieved by reducing the sleeve height (but not diameter, as this would result in a stenosis, possibly inducing turbulence) further. It was an arbitrary design where the sleeve diameter and height upon normalization by inlet graft and artery diameter respectively gave a value of one

One of the main objectives in this study was to identify effects of geometrical changes (particularly volume change) on hemodynamics. As mentioned in Chapter 2, using the Miller Cuff, researchers (Rowe et al., 1999, How et al., 2000 and Leuprecht et al. 2002) have suggested that an increased anastomotic volume due to the cuff geometry minimizes flow restrictions and delays occlusion time hence improving patency rates in the long run. The purpose of this study was to answer questions as to how much the sleeve volume could be increased before general benefits such as washout mechanism by the vortex become insignificant. The sleeve models were designed to accommodate a systematic variation of volume by splitting it into the “diameter” and “height” components so that their individual or combined effects on hemodynamics could be evaluated efficiently. Such method of studies was not found in previously published literature nor was investigation on volume variation emphasized. Previous investigations focused mainly on geometries replicated from physiological models created by surgeons and their effects on hemodynamics. Hence, there were no

incentives to vary the anastomotic geometries beyond what was possible to create during surgery. The uniqueness of this study stemmed from the fact that incorporation of a sleeve as a mechanical connector meant greater freedom to study designs that were previously impossible to create during bypass surgeries. In the present study, a sleeve designed with features resembling that of the Miller Cuff with variations to the volume was investigated.

In all five models, general flow characteristics included: smooth and laminar flow parallel to walls at low flow rates, recirculating particles below the heel which led to distinct vortex formation at higher flow rates, elevated wall shear stresses at the toe region and along the arterial floor opposite the toe as well as stagnation points along the inner wall and arterial floor.

Figure 5.2 illustrated flow characteristics in Model 1 with different Reynolds numbers. In steady flow, the region involved in blood transport was mainly bulk flow represented by the red velocity vectors. At $Re = 250$ (Figure 5.2 c), below the heel, a large vortex was observed to wash the entire heel region, including part of the proximal artery. Similar phenomenon was observed at the distal artery entrance. Washing was important because these sites were highly prone to thrombus deposition or formation due to slow fluid motion. In unsteady flow, dynamic motion of the vortices leading to increased mixing was expected. This would result in reduced particle residence time and possibilities of thrombus formation or deposition in those high prone areas of intimal hyperplasia.

In the case of a height increment resulting in volume increase (Model 2), shear stresses along the outer wall were redistributed in a region slightly upstream of the

toe. The redistribution however reduced the wall shear stress values by only about 11% ($Re = 80$) or 15% ($Re = 250$), indicating that the walls were still being washed by fast moving fluid. Wall shear stresses along the inner wall and arterial floor also showed little differences from that of Model 1. Velocity vectors within the sleeve region were also similar in magnitude. Based on steady flow analysis, height increment proved to be suitable for increasing sleeve volume yet retaining the original flow characteristics.

On the other hand, when sleeve diameter was increased, flow in the sleeve region generally became slower and more complicated. Shear stresses along all three walls also decreased in magnitudes (21%, 37% and 11% along arterial floor, inner wall and outer wall respectively for $Re = 250$). At certain locations, primary and secondary flows were comparable. It may be argued that such secondary motions enhanced spatial mixing three-dimensionally. However, the overall decrease in velocity implied the likelihood of slower mixing and increased blood exposure time to the foreign material, hence increasing the possibilities of platelet activation.

It was also observed that increasing the sleeve diameter had the effect of eliminating the recirculation zone at the distal artery entrance. In steady flow conditions, presence of 'dead water' recirculation was highly undesirable as they represent regions of trapped blood being constantly exposed to foreign materials, often resulting in thrombosis. Although recirculation in the distal artery entrance was eliminated by increasing the sleeve diameter, flow in that area was still relatively slow, due to bulk flow overshooting the toe and bypassing that entrance region. This would again lead to longer residence time and higher possibilities of platelet activation and thrombus formation or deposition. To prevent such a situation, a temporary reverse flow would

be necessary to wash out that region and such a reverse flow could only be achieved in the presence of recirculation or perhaps during backflow. Considering the overall hemodynamics, increasing sleeve volume by diameter did not yield desirable qualities. However, final assessment could be made only after investigating the unsteady flow condition.

Models 4 and 5 represented the simultaneous increment of sleeve diameter and height, the latter having a larger increment than the former. An interesting observation was that these models exhibit flow characteristics found in both Models 2 and 3. The redistributed wall shear stresses along the upstream of the toe region resembled that observed in Model 2 when sleeve height was increased. The dominant source-like structure and the eliminated (Model 4) or minimized (Model 5) recirculation zone at the distal artery entrance were similar to that observed in Model 3 when sleeve diameter was increased. Overall, the effects of diameter increment outweigh the effects of height increment. For reasons discussed earlier regarding Model 3, the highly enlarged models did not show sufficiently acceptable hemodynamics.

In conclusion, from the steady flow studies, Model 1 as the baseline model had exhibited qualities that were sufficient in improving hemodynamics. As compared to conventional bypass models, the volume of Model 1 was already larger due to incorporation of the cuff-like structure. For a further increase in volume, a height increment was recommended since most of the flow characteristics in Model 1 were retained. Increment by diameter or a combination of height and diameter did not yield desirable results as discussed earlier. A final assessment could be reached only with further investigations using the pulsatile models discussed in the next chapter.

Chapter 6: Results and Discussion of Geometrical Variations (Pulsatile Flow)

It is a known fact that flow at precapillary level is mainly unsteady and driven by pressure gradient. Steady flow analyses carried out in the previous chapter seek to identify effects of changing geometries and flow conditions (Reynolds numbers) on flow patterns in the sleeve design. However, such models could only provide a preliminary insight and should not be mistaken as a true representation of physiological flow patterns in arteries. The Womersley solution as derived in Chapter 3 is presently one of the closest approximation to physiological flow. The fully developed pulsatile velocity profile corresponding to a specific volume flow waveform was computed and imposed as the inlet boundary condition. The waveform used in this study was biphasic in nature and categorized into the following phases: first acceleration, first deceleration, second acceleration, second deceleration (gentle), second deceleration (steep), backflow acceleration and backflow deceleration.

Flow characteristics in the symmetry plane were studied at points indicated by red dots as shown in Figure 6.1 while shear stress analyses were carried out only at the maximum and minimum points ($t = 1.95s, 2.125s, 2.25s$ and $2.675s$) along the inner wall, outer wall and arterial floor (Figure 6.2). The inner and outer walls were defined from the graft inlet to the proximal and distal artery ends respectively while arterial floor stretched from proximal to distal artery. Positive wall shear stress is represented by flow in positive x direction while negative wall shear stress represented flow in negative x direction. Terminologies describing various locations in the models are also shown in Figure 6.2. Simulation studies were conducted for Models 1, 2, 3 and 5.

To simplify the study, Model 4 was not included as it represented a sleeve shape similar to Model 5 (increment in both sleeve diameter and height) but with a smaller volume. Model 5 was chosen as an extreme case to study its effects on hemodynamics. Table 6.1 presents dimensions of respective models used in the pulsatile investigations. Results of the simulations will be presented and discussed in the following sections.

Figure 6.1 : Average velocity waveform for pulsatile study.

Figure 6.2: Terminologies used in pulsatile study.

Model	D _A	D _G	D _S	H _S	Remarks
1	2.6 mm	3 mm	D _G	D _A	Baseline Model
2	2.6 mm	3 mm	D _G	1.5 D _A	Increased Height
3	2.6 mm	3 mm	1.25 D _G	D _A	Increased Diameter
5	2.6 mm	3 mm	1.5 D _G	2 D _A	Increased Diameter and Height

Table 6.1: Dimensions of sleeve models used in pulsatile study.

6.1 Flow Characteristics and Wall Shear Stress Variations in Model 1

During the first acceleration phase in Model 1 at $t = 1.825s$ and $1.85s$, flow was laminar with instantaneous streamlines observed to be essentially parallel to the inner and outer walls as well as the arterial floor as demonstrated in Figure 6.3 (a) and (b). As velocity increased to peak value ($t = 1.95s$), high momentum fluid from the graft entered the sleeve diagonally and skewed towards the outer wall. This resulted in bifurcation of the main flow stream into the forward (towards distal artery) and backward (towards proximal artery) components, with a stagnation point clearly visible in Figure 6.3 (c) at $x_1 = 15$ mm from the proximal end along the arterial floor. Due to full occlusion of the proximal artery, flow entering that section was forming a clockwise vortex, occupying nearly 90% of the proximal. At approximately $x_2 = 14.3$ mm, reattachment was observed between the upward and downward flows. The presence of the vortex could be validated by the negative shear stresses along the arterial floor and inner wall as observed in Figure 6.4 (a) and (b) respectively. The negative values were due to reversed flow as a result of the vortex formation. Wall shear stress variations along the outer wall is also shown in Figure 6.4 (c). Peak shear stress along the inner wall (4.6 Pa) occurred at the sleeve entrance while along the outer wall, shear stress was observed to have peaked at the toe region (6 Pa). On the

arterial floor opposite the toe, wall shear stress reached a maximum value of approximately 4.9 Pa.

As flow began to decelerate for the first time ($t = 2.075s$, $2.1s$ and $2.125s$), the vortex increased in intensity and size (spreading rightward, into the sleeve region) resulting in movement of the stagnation points upward and rightward along the inner wall and arterial floor respectively. At $t = 2.1s$, two small recirculating zones were distinctively visible, one located at the top of the sleeve and the other at the entrance of the distal section. As the flow decelerated further to the minimum point ($t = 2.125s$), these recirculating zones had diminished in size. At the same time, the vortex which originated from the proximal artery section had by now reached the maximum size, occupying approximately the entire mid to lower sleeve region. Stagnation points had also shifted to the furthest distance upward at $x_2 = 13.7$ mm and rightward at $x_1 = 16.67$ mm along the inner wall and arterial floor respectively. Negative wall shear stresses were registered along the arterial floor and inner wall as a result of reversed flow caused by the vortex. Overall variation and locations of maximum shear stresses remained the same as compared to the first peak at $t = 1.95s$, with the exception of higher negative shear stresses due to the vortex of larger magnitude and intensity. Peak wall shear stresses along the arterial floor, inner wall and outer wall were 1.2 Pa, 0.25 Pa and 1.3 Pa respectively at $t = 2.125s$.

With the onset of acceleration ($t = 2.15s$), the vortex was “pushed” back instantaneously to the site of formation (below the heel) with diminished size and intensity. Movement of the vortex brought about a shift in stagnation points leftwards and downwards along the arterial floor and inner wall respectively. Recirculating

zones in the top sleeve region as well as the distal artery entrance had disappeared at this stage. In the subsequent time frame ($t = 2.175\text{s}$), flow patterns were similar to those observed in the first acceleration phase ($t = 1.825\text{s}$ and 1.85s) with particles moving parallel to the walls and no flow separation was observed. As flow accelerated to the second peak ($t = 2.25\text{s}$), a vortex was formed once again below the heel region. At this point, peak shear stresses along the arterial floor, inner wall and outer wall were found to be 4 Pa , 4.3 Pa and 5.1 Pa respectively at sites similar to the first peak as shown in Figure 6.4 (a), (b) and (c) respectively. Variations in wall shear stresses along these walls were also the same as those in the first peak with the exception of slightly lower peak values due to a slightly lower average flow velocity.

The second deceleration phase could be further categorized into two sub-phases: a gentle deceleration followed by a steep deceleration. Overall flow patterns in the second deceleration were similar to those in the first (i.e. shifting of the vortex and its corresponding stagnation points). It was however observed that strength of the vortex was much weaker in the gentle deceleration than those in the steep deceleration phase. From $t = 2.575\text{s}$ onwards, for every 0.025s interval increment, the vortex and stagnation points moved with greater increase in distance, size and intensity. Flow characteristics at $t = 2.625\text{s}$ were similar to that found earlier at $t = 2.1\text{s}$ and 2.125s , with two zones of recirculation (one at the sleeve top and the other at the distal artery entrance). The vortex by now had filled almost the entire mid to lower sleeve region.

Beyond the zero average velocity at $t = 2.65\text{s}$, backflow was observed throughout the model, with the exception of a small core region in the graft still experiencing weak forward flow. As mentioned in Section 3.3.1, owing to viscosity effects, near wall