NUMERICAL SIMULATIONS OF BLOOD FLOW IN ARTERIES USING FLUID-STRUCTURE INTERACTIONS

A Dissertation by

Eleyas Shaik

M.S., Computational Mechanics of Materials and Structures,
University of Stuttgart, Germany, 2004

B.E., Mechanical Engineering, Osmania University, India, 2002

Submitted to the Department of Aerospace Engineering
and the faculty of the Graduate School of
Wichita State University
in partial fulfillment of
the requirements for the degree of
Doctor of Philosophy

July 2007
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I have examined the final copy of this dissertation for form and content, and recommend that it be accepted in partial fulfillment of the requirement for the degree of Doctor of Philosophy with a major in Aerospace Engineering.

Klaus A. Hoffmann, Committee Chair

We have read this dissertation and recommend its acceptance:

Kamran Rokhsaz, Committee Member

Walter J. Horn, Committee Member

Roy Y. Myose, Committee Member

Hamid M. Lankarani, Committee Member

Accepted for the College of Engineering

Zulma Toro-Ramos, Dean

Accepted for the Graduate School

Susan K. Kovar, Dean
DEDICATION

To my parents, my sisters, and my wife who offered me unconditional love and support throughout the course of this dissertation
ACKNOWLEDGMENTS

First of all, I would like to thank Dr. Klaus A. Hoffmann, my supporter and advisor throughout my career at Wichita State University, who gave me the opportunity to work on his team and introduced me to the fascinating field of computational fluid dynamics. I could not have imagined having a better advisor and mentor for my Ph.D., and without his common sense, knowledge, perceptiveness, and cracking-of-the-whip, I never would have finished. His time, encouragement, and guidance have been very important to me and to the research reported herein. I am particularly grateful to him for his continual patience and confidence in me. He has been available in every way as a resource, be it emotionally, socially, scholarly, or administratively.

A special thanks goes to Dr. Jean-Francois Dietiker, who is most responsible for helping me complete the writing of this dissertation as well as the challenging research that lies behind it. Dr. Jeff has been a friend and mentor. He taught me how to write academic papers, had confidence in me when I doubted myself, and brought out the good ideas in me. Without his encouragement and constant guidance, I could not have finished this dissertation. He was always there to meet and talk about my ideas, to proofread and mark up my papers and chapters, and to ask me good questions to help me think through my problems.

Besides my advisor, I would like to thank my committee members: Dr. Kamran Rokhsaz, Dr. Walter J. Horn, Dr. Roy Y. Myose, and Dr. Hamid M. Lankarani. I truly appreciate their advice, comments, suggestions, time, and encouragement.

Last, but not least, I thank my family: my parents, for giving me life in the first place, for educating me, for unconditional support and encouragement to pursue my interests, even when the interests went beyond boundaries of language, field, and geography; and my wife, for listening to my complaints and frustrations, and for believing in me.
ABSTRACT

Cardiovascular disease (CVD) is the number one cause of death in the United States and worldwide. Among the various CVDs, coronary artery disease (CAD) is the leading cause of death among both men and women. Of the various forms of CADs, atherosclerosis is the primary cause. To investigate these arterial diseases, numerical simulations of blood flow in the arteries using fluid-structure interactions (FSI) with the finite element method were performed. First, simulations were performed assuming the arterial walls are rigid, and then they were extended to deformable arteries where contraction and expansion of the arteries are considered. Moreover, this study also investigated the outcome of bypass surgeries involving end-to-side and end-to-end bypass anastomosis. To help understand the effect of various flow/material characteristics on these surgeries and related issues, numerical investigations on artery-graft bypass models were conducted.

The primary objectives of this research were as follows: (1) to validate the numerical simulations with existing experimental data, (2) to differentiate the effect of Newtonian and non-Newtonian fluid flow considering three-dimensional rigid models of the artery, (3) to investigate the effect of arterial geometry using both steady and pulsatile flow cases, (4) to provide some indication of the occurrence of atherosclerosis while describing the hemodynamic parameters, (5) to determine the extent of interaction between blood flow and the elastic walls while performing numerical simulations on various arterial geometries with steady and pulsatile flow, (6) to investigate the outcome of bypass surgery (various cases) with natural and synthetic grafts, and (7) to determine the occurrence of intimal hyperplasia following bypass surgery.

In the computations, the non-Newtonian behavior of blood was described using the Carreau-Yasuda model. Generally, good agreement between the numerical and experimental
results was observed in the velocity profiles, whereas some discrepancies were found in wall shear stress (WSS) distributions. The regions of the artery models for both steady and pulsatile flow cases, with low wall shear stresses correspond to regions of the body that are more susceptible to atherosclerosis; or intimal hyperplasia for the case of bypass surgery were identified. It was also found that the geometry of the artery plays an important role in the development of atherosclerosis. The comparison between the simulations considering rigid arteries and deformable arteries showed a substantial increase in wall shear stresses for the rigid artery. In addition, it was observed that the calculated difference in shear stress between the simulations performed using rigid wall assumptions with that of deformable walls was in the range of 30 to 40 percent at the maximum shear stress location. Therefore, it was concluded that the deformation of the arterial wall cannot be neglected while performing blood flow simulations.
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NOMENCLATURE

Roman symbols:

\( a \) Acceleration vector

\( a \) Radius

\( A \) Amplitude

\( C \) Compliance

\( C \) Divergence matrix

\( d \) Displacement

\( \dot{d} \) Velocity

\( \ddot{d} \) Acceleration

\( \hat{d} \) Mesh displacement vector

\( \overline{\hat{d}} \) Prescribed mesh displacement vector

\( D \) Diffusive matrix

\( D \) Elasticity tensor

\( D \) Diameter

\( E \) Strain tensor

\( E \) Young’s modulus

\( f \) Body force per unit mass

\( \tilde{f} \) Prescribed body force

\( F \) Deformation gradient tensor

\( g \) Force

\( i \) Index
NOMENCLATURE (continued)

j  Index

J  Jacobian

k  Index

\( \hat{K} \), K  Stiffness matrix

l  Index

M  Mass matrix

n  Outward pointing normal

N  Convective matrix

p  Pressure

Q  Flow rate

r  Radius

\( \hat{R} \)  Mesh position vector

Re  Reynolds number

s  Strain rate tensor

S  Second Piola-Kirchhoff

t  Time

u  Velocity

\( \bar{u} \)  Prescribed velocity

Greek symbols:

\( \dot{\gamma} \)  Shear rate

\( \Gamma \)  Boundary

\( \delta \)  Kronecker delta
NOMENCLATURE (continued)

Δt  Time step
ΔD  Change in diameter
ΔP  Change in pressure
ε   Strain tensor
λ   Relaxation time
μ   Dynamic viscosity
µ₀  Bulk viscosity
µ₀  Shear rate viscosity at infinite shear rates
µ∞  Shear rate viscosity at infinite shear rates
ν   Poisson ratio
π   Pi
ρ   Density
σ   Stress tensor
τ   Deviatoric stress tensor/traction vector
τ̄  Prescribed traction
Ω   Domain

Subscript:
exp  Experimental
m    Mean
max  Maximum
num  Numerical
ref  Reference
NOMENCLATURE (continued)

**Superscripts:**

- **F**: Fluid
- **I**: Interface
- **k**: Increment
- **S**: Solid
- **U**: Velocity boundary
- **IF**: Interface node related to the fluid
- **II**: Interface node related to the interface
- **IU**: Interface node related to the velocity boundary
- **σ**: Internal stress
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<td>42</td>
</tr>
<tr>
<td>CPU</td>
<td>Central Processing Unit</td>
<td>83</td>
</tr>
<tr>
<td>CSD</td>
<td>Computational Structural Dynamics</td>
<td>42</td>
</tr>
<tr>
<td>CVD</td>
<td>Cardiovascular Disease</td>
<td>vi</td>
</tr>
<tr>
<td>ePTFE</td>
<td>Extended Polytetrafluoroethylene</td>
<td>5</td>
</tr>
<tr>
<td>EC</td>
<td>Endothelial Cell</td>
<td>13</td>
</tr>
<tr>
<td>ECA</td>
<td>External Carotid Artery</td>
<td>29</td>
</tr>
<tr>
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<td>Finite Difference Method</td>
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<td>FEM</td>
<td>Finite Element Method</td>
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<td>FSI</td>
<td>Fluid-Structure Interaction</td>
<td>6</td>
</tr>
<tr>
<td>FVM</td>
<td>Finite Volume Method</td>
<td>33</td>
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<tr>
<td>ICA</td>
<td>Internal Carotid Artery</td>
<td>29</td>
</tr>
<tr>
<td>IH</td>
<td>Intimal Hyperplasia</td>
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<tr>
<td>LDV</td>
<td>Laser Doppler Velocimetry</td>
<td>34</td>
</tr>
<tr>
<td>LIW</td>
<td>Left Inner Wall</td>
<td>65</td>
</tr>
<tr>
<td>LOW</td>
<td>Left Outer Wall</td>
<td>65</td>
</tr>
<tr>
<td>OSS</td>
<td>Oscillating Shear Stress</td>
<td>39</td>
</tr>
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</table>

* Page numbers describing each abbreviation is provided for the sake of reference.
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
<th>Page</th>
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<td>RIW</td>
<td>Right Inner Wall</td>
<td>65</td>
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<tr>
<td>ROW</td>
<td>Right Outer Wall</td>
<td>65</td>
</tr>
<tr>
<td>TL</td>
<td>Total Lagrangian</td>
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<tr>
<td>UL</td>
<td>Updated Lagrangian</td>
<td>49</td>
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<tr>
<td>WSS</td>
<td>Wall Shear Stress</td>
<td>14</td>
</tr>
<tr>
<td>WSSG</td>
<td>Wall Shear Stress Gradient</td>
<td>38</td>
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</table>

* Page numbers describing each abbreviation is provided for the sake of reference.
CHAPTER 1
INTRODUCTION

1.1 Motivation and Challenges

Worldwide, more than 16 million people die each year from cardiovascular disease; more than 5.5 million of these deaths were attributed to strokes in 2002 [1]. In 2001 more than 20 million people suffered strokes, 5.5 million of which were fatal [2]. In the United States, 60.8 million people have at least one type of cardiovascular disease, the leading cause of death. Of these, 4.5 million people have suffered strokes, with approximately 600,000 strokes occurring each year [3]. Thousands of heart surgeries are performed every day in the United States. In fact, in 2004 alone, surgeons performed approximately 800,000 coronary bypass or valve repair and replacement surgeries. And even though there is a shortage of donor organs, about 2,000 people had heart transplants. Myocardial infarction (heart attack) can be a serious result of coronary artery disease (CAD) usually resulting from atherosclerosis, which occurs when a blocked coronary artery causes death to a portion of the myocardium (heart muscle). Cardiac arrest (heart stops beating) may also result from CAD; 90 percent of sudden deaths occur in patients with two or more major arteries narrowed by atherosclerosis.

Statistics show CAD to be the leading cause of death among both men and women in the United States and Europe. For example, approximately 12.8 million Americans suffer from CAD, and nearly 500,000 Americans die from heart attacks caused by CAD. Over 12 million Americans have a history of myocardial infarction or angina (chest pain) or both.

Tables 1 and 2 show the average number of operations and cost per operation in the United States, respectively.
TABLE 1
AVERAGE NUMBER OF OPERATIONS DONE IN UNITED STATES ALONE [3]

<table>
<thead>
<tr>
<th>Name of Operation</th>
<th>Number of Operations per Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Heart valve replacements</td>
<td>95,000</td>
</tr>
<tr>
<td>2) Bypass (cardiac revascularization)</td>
<td>467,000</td>
</tr>
<tr>
<td>3) Heart transplants</td>
<td>2,016</td>
</tr>
<tr>
<td>4) Open-heart procedures</td>
<td>66,000</td>
</tr>
<tr>
<td>5) Coronary artery disease</td>
<td>12,800,000</td>
</tr>
</tbody>
</table>

TABLE 2
AVERAGE COST PER OPERATION IN UNITED STATES ALONE [3]

<table>
<thead>
<tr>
<th>Name of Operation</th>
<th>Average Cost per Operation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Heart valve repair/replacement</td>
<td>$35,000</td>
</tr>
<tr>
<td>2) Heart transplant</td>
<td>$110,000 - $287,000</td>
</tr>
<tr>
<td>3) Bypass surgery</td>
<td>$58,889</td>
</tr>
<tr>
<td>4) Angioplasty</td>
<td>$56,225</td>
</tr>
<tr>
<td>5) Coronary artery bypass graft</td>
<td>$83,919</td>
</tr>
</tbody>
</table>

Cost includes hospital fees, surgeon fees, anesthetist’s fees, percussionist’s charges, etc.
To say that the flow of fluids plays an important role in our lives is an understatement of large proportions. Commonly used examples to support the claim are, the airflow around a car or airplane, the flow of oil through pipelines, and the influence of atmospheric and oceanic flows on the weather.

Although the examples given above do influence our high-tech society in a substantial way, one tends to forget the importance of our basic need: oxygen supply to the human brain and various organs provided by arterial blood flow. It is rather a tragic, if not cynical, fact that the prime cause of death in our advanced civilization is related to cerebrovascular accidents, mainly caused by atherosclerosis, one of the most important causes of death [4].

Atherosclerosis is one of several types of “arterio”-sclerosis, which is characterized by a thickening and stiffening of artery walls. These two terms are often used synonymously. In general, atherosclerosis occurs when one passes the age of thirty, and its existence increases with age. Apart from these purely temporal reasons, lifestyle also seems to play an important role. Both stroke (sudden death of brain cells) and transient ischemic attack (temporary blockage of the blood supply to the brain) can result from thrombolytic and embolitic complications of arteriosclerosis in the common carotid or the coronary arteries. A possible cause for these incidents is the involvement of hemodynamics, the basis for the current research activity. Hemodynamics might be defined to be an important role in the development of atherosclerotic plaques in coronary arteries (and other sites). However, the specific mechanism whereby blood flow patterns influence the development of arterial disease remains a challenging topic in bioengineering. A considerable amount of evidence shows that fluid mechanical forces have a strong influence on the initiation and progression of several cardiovascular diseases, such as atherosclerotic and ischemic diseases [5, 6]. The interaction between the flow and the arterial
Atherosclerosis narrows and stiffens the artery, which ultimately leads to the blockage of the artery and a high risk of heart attack and stroke. Atherosclerotic obstructions can cause severe reduction of the arterial blood flow, leading to organ dysfunction. Fat, cholesterol, and other substances accumulate on the artery walls and form “atheromas” or plaques. Eventually, this fatty tissue can erode the wall of the artery, diminishing its elasticity and interfering with the blood flow. There are various treatments available to cure diseased arteries. Among them, medications such as colestipol, cholestyramine, etc., are usually the first step. Secondly, it is possible to surgically remove the deposits using balloon angioplasty, whereby balloon-tipped catheter is used to flatten the plaque, open the arteries, and increase blood flow past the deposits. Another widely used technique is stenting, which consists of implanting a small metal device inside the artery to keep it open. Finally, an invasive procedure of bypass surgery can be performed where a normal artery or vein from the patient is used to create a bridge that bypasses the blocked section of the artery. The process of taking blood vessels from one part of the body and connecting them to another part is called grafting. More recently, researchers and surgeons have developed a technique by which the affected part of the artery is replaced with synthetic vascular grafts.
When the natural vessel fails, vascular grafts are commonly used to bypass the diseased vessel, enabling an adequate supply of blood to be restored to distal tissues and organs. If possible, the best choice for a replacement vessel is an autograft (autogenous saphenous vein), where sections of the patient’s healthy blood vessels (usually veins) are harvested and implanted at the required location. However, patients with pre-existing vascular disease or patients that already have had autograft procedures do not have blood vessels that are sufficiently healthy to adequately serve as replacements. In such cases, the most common form of treatment has been the use of synthetic polymeric materials, such as ePTFE (extended polytetrafluoroethylene) or Dacron (polyethylene terephthalate) [9], to form permanent replacements for the damaged vessels. The first successful step toward this approach was taken by William J. von Liebig [9]. His patented technique for manufacturing seamless synthetic grafts was used for vascular implants, which could be grafted onto human arteries and blood vessels. Since then, many discoveries have been made in the area of synthetic graft valves, leading to the development of grafts, including treated natural tissues, laboratory-engineered tissues, synthetic polymer fabrics, and synthetic grafts such as Dacron and Teflon (polytetrafluoroethylene) [9].

As the frequency of vascular procedures increases from year to year, researchers are increasingly looking to modify natural materials as a compromise between autografts and purely synthetic grafts that utilize materials such as ePTFE and Dacron [9]. Other uses for vascular grafts include treatments for blood vessel aneurysms and fistulas [10], as well as replacements for diseased arteries in other locations in the body. The success of synthetic grafts depends on a variety of factors, such as mechanical properties and biocompatibility. With regard to the biocompatibility issue, there are two types of mismatch: (1) geometry mismatch, i.e., the diameter of the artery is not equal to the diameter of the graft, and (2) compliance mismatch, i.e.,
the mechanical properties of the graft are not similar to the artery. In order for artery-replacement procedures to be successful, the geometry and compliance of the host artery and graft should match. However, achieving such requirements may not be possible in all situations.

Approximately, 20 to 50 percent of bypass grafts fail after surgery due to formation of intimal hyperplasia (IH) [11, 12]. This disease is characterized by a progressive thickening of the innermost layer of the artery wall in the region where the graft is joined to the artery. As a result, gradual narrowing of the vessel leads to a blockage of the vessel. Also, IH reduces the lumen of the bypass graft, which leads to reduced flow and eventually leads to graft occlusion. Although there are several theories in the literature, the exact cause of IH is uncertain [11, 13-15]. The development of IH in bypass grafts (vein and synthetic grafts) depends on several factors, such as injury, hemodynamics, and graft-artery compliance mismatch. Fluid dynamics within the vascular grafts (hemodynamics) is widely believed to play an important role in the initiation and development of anastomotic IH [16-18]. However, specific mechanisms whereby blood flow patterns influence the development of IH also remain a major unsolved question in bioengineering.

In the past, computations have often been performed using rigid geometries. Computations of unsteady flows in the circulatory system become even more time-consuming when the distensibility of the vessels is taken into account. Fluid-structure interaction (FSI) is of primary interest in blood flow simulations because of the arterial wall modeling process and the subsequent altered flow pattern in pathological states. In the case of pulsatile flows in the human body, the artery expands and contracts its diameter when the heart beats. It is necessary to simulate this dynamic change of the artery geometry caused by the pulsatile flow. Blood flow interacts mechanically and chemically with vessel walls, giving rise to complex FSIs whose
mathematical analysis is still incomplete and which is difficult to simulate efficiently. At a macroscopic level, the arterial wall is a complex multi-layer structure that deforms under pressure forces. Even though constitutive equations have been proposed for the structural behavior of the vessel wall, its elastic characteristics in vivo are still very difficult to determine.

Coronary arteries change shape dramatically over the cardiac cycle. Due to their position on the beating heart, overall coronary arterial geometry (including local curvature) varies significantly, and portions of the coronary arterial tree undergo full or partial collapse during portions of the cardiac cycle. The goal of this research was to use engineering modeling tools to describe complex blood flow patterns, thereby helping to determine the link between blood flow patterns and arterial disease. To describe the highly unsteady, three-dimensional flow that occurs in large arteries, state-of-the-art computational fluid dynamic techniques were used. In addition, compliance mismatch in bypass surgeries was difficult to investigate theoretically and experimentally because it is expensive and limited to easily accessible arteries and specific synthetic grafts. Therefore, computational modeling offers an attractive alternative. With the expansion of computational fluid dynamics (CFD), it has become possible to investigate blood flows in arteries by including most of the physical aspects related to blood flow.

A powerful way to achieve the long-term goal is to correlate patient-specific hemodynamic patterns with patient-specific spatial distributions of artery disease. This, in turn, requires tools for quantifying artery hemodynamics, which was the major focus of this study. As alluded to previously, there are significant engineering challenges in studying arterial blood flow patterns: for example, the coronary arteries are small (and, therefore, hard to image), they move appreciably during the cardiac cycle, and they have an inherently complex, 3-D geometry. Thus, the most fruitful way of overcoming these challenges is via a computational approach.
Modeling of unsteady flow in realistic arterial geometries is computationally intensive, and is becoming progressively more so. Application of such an approach to arterial hemodynamics requires the ability to adapt complex, 3-D meshes in response to time-varying flow fields. Thus, despite the fact that the ultimate area of application of this work is biomedical engineering, the results will be applicable in a variety of areas. For example, FSI will be directly applicable to any incompressible flow in a complex 3-D geometry, such as in an industrial mold-filling process, internal flows with deformable boundaries, aluminum extrusion dies, flow-induced vibrations in external flows over deformable bodies (such as aircraft wings, antennas, etc.), and thermal deformation in a coupled thermal/fluid/structure problem (such as casting, solidification, and crystal growth).

1.2 Common Causes of Arterial Disease

1.2.1 Atherosclerosis

Atherosclerosis is a disease characterized by progressive narrowing and hardening of the arteries over time. Occlusion of the artery lumen can cause a substantial decrease in blood supply to the myocardium, giving rise to chest pains (see Figure 1-1). If the hardened inner layer of the artery ruptures, thrombus may form inside the artery lumen, leading to heart attack. At an early stage of the disease, atherosclerotic plaque formation may induce thrombus formation, i.e., fragments of the thrombus break off and obstruct smaller arteries downstream. Atherosclerosis, which is a leading cause of death, is a condition that primarily affects the aorta, and large- and medium-sized arteries, particularly the coronary and cerebral arteries. The disease is characterized by three phenomena [19]: (1) proliferation of smooth muscle cells, (2) deposition of intracellular and extra cellular lipids, and (3) accumulation of extra cellular matrix components. Due to these phenomena, the vessel wall thickens, and the luminal cross-sectional
area eventually diminishes. The vessel then is prone to occlusion by a thrombus. When this happens, e.g., in the carotid artery bifurcation or coronary arteries, the occlusion may be lethal. It has been shown that locations where low and oscillating wall shear stresses occur correlate well with locations where atherosclerotic lesions form [20-22]. Typical locations are inside bends (coronaries) and non-divider walls of bifurcations (carotid bifurcation). The magnitude and nature of the shear stress depend on the properties of the blood, the properties of the vessel wall, and geometrical factors.

Figure 1-1. A: A normal artery with normal blood flow (the inset image shows a cross-section of the normal artery). B: Artery with plaque buildup, which is partially blocking blood flow (the inset image shows the degree to which the artery is blocked) [23]
Details of flow patterns may affect vessel walls. Flow patterns are greatly influenced by sudden changes in geometry (i.e., branches and bends). Therefore, much attention has been given to the flow in these areas relative to atherosclerosis. It is clear that the locations of initial development of atherosclerosis are regularly associated with the inner curvature of bends and in regions with a tendency toward flow separation at bifurcations of blood vessels. The flow in bends and branches are of interest in a physiological context for several reasons. Additional energy losses due to the local disturbances of the flow are of interest in calculating the airflow in the lungs and in wave-propagation models of the arterial system. Details of the pressure and shear stress distributions on the walls of a bend or bifurcation are of interest in the study of atherogenesis because it appears that the localization of plaque is related to local flow patterns.

A variety of theories have been proposed regarding the cause of atherosclerosis. In some of these theories hemodynamic factors play an important role. A possible role of hemodynamics in the process of atherosclerosis is suggested by its predilection to bends and bifurcations, since flow phenomena exhibit unique characteristics in these areas. Some of the literature indicates that it was thought that endothelial injury caused by high stresses was responsible for atherogenesis [24, 25]. However, later investigations by Caro et al. [26] confirmed that atherosclerotic lesions develop more frequently in regions with low shear stresses and with recirculation rather than in regions with high shear stresses and unidirectional flow [21, 27]. Therefore, detailed insight into the flow phenomena occurring in bends and bifurcations possibly contributes to a better understanding of the role of hemodynamics in the process of atherosclerosis.

Although atherosclerosis is one of the leading causes of death, the pathogenesis has been unclear until lately because of two characteristics:
• Atherosclerosis develops inside the walls of vessels so that the genesis is difficult to observe and test directly in vivo.

• The evolution of atherosclerosis is very slow; disease progression may span decades, which makes experiments to reproduce and study its pathogenesis very difficult.

Because of these characteristics, most information about the disease has come from clinical observations and autopsies that indicated the high frequency localizations of lesions in the vascular system, the categorization of lesions, and the relative importance of various risk factors.

1.2.2 Intimal Hyperplasia (IH)

The majority of vein and synthetic bypass grafts remain patent (the state or quality of being opened) in the first two months after implantation into the arterial circulation. After two months, the patency of these grafts is threatened due to development of intimal hyperplasia [28], defined as the abnormal migration and proliferation of smooth muscle cells with associated deposition of extracellular matrix in the intimal layer of the vein graft or formation of a neointima in the synthetic graft [28]. Intimal hyperplasia reduces the lumen of the bypass graft, which leads to reduced flow and can ultimately lead to graft occlusion. Furthermore, IH is observed after angioplasty, in endarterectomy, and in arteriovenous fistulae for haemodialysis [28]. In the first week after operation, bypass grafts usually fail because of technical factors such as technical errors induced, such as stenosis at the anastomoses and compression or kinking of the bypass graft. Between 2 and 24 months after the operation, vein and synthetic bypass grafts usually fail because of the formation of intimal hyperplasia. There is clinical and experimental evidence that IH forms the basis for vein graft atherosclerosis, an important cause of late vein
graft failure after 24 months after implantation [28]. Furthermore, success of a bypass graft depends on the type of bypass and on the position of bypass graft in the arterial circulation.

Intimal hyperplasia has obviously an important impact on the post-operative health status of patients with vein bypass grafts and synthetic bypass grafts. Occlusion of a bypass graft often results in a hospital readmission and re-operation, and poses a heavy burden for the limited resources of health care providers. Therefore, studies to elucidate the events leading to intimal hyperplasia and potential reduction of intimal hyperplasia have a high priority. Currently, the pathophysiological triggers for intimal hyperplasia in vein and synthetic grafts have been classified as injury, inflammation, and hemodynamic factors [29].

Intimal hyperplasia was first described in 1906. Carrel and Guthrie [30] observed macroscopically that, within a few days after vein graft implantation into the arterial circulation, the anastomotic stitches became covered with a glistening substance similar in appearance to normal endothelium. Since that time IH has been reported in vein grafts implanted into the arterial circulation in several mammals including mice, rats, dogs, sheep, monkeys, and humans.

In 1949, the autologous vein was the first conduit to be used to bypass an obstructed artery in man. Approximately 30 to 50 percent of patients with vein grafts have recurrent ischemic complaints caused by vein graft occlusion after 5 to 10 years. An important cause of vein graft occlusion is the formation of intimal hyperplasia in the vein graft [28]. Other types of bypass grafts have been introduced in the hope to improve bypass graft patency and to lower the incidence of recurrent ischemic complaints. From its introduction in 1968, the autologous internal mammary artery is a successful alternative bypass graft to vein grafts [31]. Patients with internal mammary artery bypass grafts have few recurrent ischemic complaints. Due to their
anatomic location and short length, internal mammary arteries are used only for coronary bypass grafting.

A very successful alternative bypass graft is the synthetic graft. In 1954, the synthetic graft was introduced to bridge arterial defects in man [32]. The synthetic graft has the advantage that it is not limited in length and is always available. The disadvantage of the synthetic bypass graft is the high incidence of occlusion due to the rapid formation of intimal hyperplasia.

The challenge for the future is to improve the patency of autologous vein grafts by controlling the formation of intimal hyperplasia. To improve the performance of vein grafts, the pathophysiology of intimal hyperplasia in these grafts must be studied in detail.

Several types of bypass surgery simulations and aspects of intimal hyperplasia including pathophysiological triggers, and strategies to control intimal hyperplasia in vein/synthetic grafts were also studied in this research.

1.3 Hemodynamics

Hemodynamics or blood dynamics is the study of the properties and flow of blood. Hemodynamic factors that have been suggested to be important in atherogenesis and IH are derived from the velocity field and involve several different forms, such as flow separation and vortex formation, shear stresses, and spatial and temporal shear stress gradients. Between the blood and vessel wall there is a monolayer of cells called endothelial cells (ECs). Any interactions enhanced by hemodynamic factors seem to interplay on the ECs first, and subsequently these actions induce unfavorable cell responses that contribute to atherogenesis and its progression. The injury hypothesis, which suggests that ECs may be damaged or become dysfunctional as a result of hemodynamic factors, is widely accepted as the pathogenesis of atherosclerosis [33]. Local inflammation of the vessel wall is commonly observed [34], which
further strengthens the injury hypothesis. Much attention has been given to fluid dynamic wall shear stress (WSS) which induces a micro environment of interaction (frictional force) between blood and endothelial layer. WSS is a vector whose magnitude is proportional to the blood viscosity and flow velocity gradient normal to the surface, and which acts in a direction parallel to the local velocity at the wall. WSS is difficult to measure directly in vivo or in vitro so that it is generally computed from the local velocity distribution near the wall. Therefore, the investigation of WSS effects requires knowledge of the local velocity field. Correlations with plaque occurrence have been made for low, high and oscillating WSSs. WSS distribution, in turn, is highly influenced by diameter changes, separation zones, recirculation areas, turbulence, and elasticity.

1.4 Computational Fluid Dynamics

The study of hemodynamics with numerical and computational tools can be defined as computational hemodynamics, which forms a subset of CFD. In CFD, numerical simulations of fluid flow are performed. These simulations result from numerical solutions of differential equations derived from physical conservation laws for flows. CFD can provide insight into hemodynamics and possibly could provide some answers to the questions concerning blood flow.

Experimental and computational methods extensively used as research tools in fluid dynamics are also available in hemodynamics research [35]. CFD has been proven to be a reliable technique for investigating time-varying, 3-D flow patterns in a complex geometrical model. In contrast to experimental flow studies, CFD is more convenient for altering model and flow parameters such as inlet velocity, wall conditions, etc. CFD can present results with high resolution if the boundary condition information is correct; therefore, the geometrical data and boundary velocity data that come from in vivo measurements are necessary for realistic
modeling of blood flow in the artery. Medical imaging techniques can supply these data. From CFD solutions, it is straightforward to present velocity distributions for blood flows and to extract WSS and other important hemodynamic factors. These factors suggest that CFD can be a unique and attractive tool for hemodynamics research.

1.5 Fluid-Structure Interactions

Fluid-structure interaction scenarios are those that involve the coupling of fluid mechanics and structural mechanics problems. FSI plays an important role in several different types of applications such as biomedical, material processing, automotive, aeronautical, and civil engineering. Examples of such situations include the following:

- Internal flows with deformable boundaries, arteries, tissue grafts, polymer, or aluminum extrusion dies.
- Flow-induced vibrations in external flows over deformable bodies, such as aircraft wings, antennas, etc.
- Thermal deformation in a coupled thermal/fluid/structure problem, such as casting, solidification, and crystal growth.

The solutions of structural and fluid mechanics problems can be considered a relatively mature technology. Commercial codes have been available from the mid-70s for structural mechanics and from the mid-80s for fluid mechanics. However, the coupling of the two problems brings up new algorithmic issues, such as impulsive starts, velocity coupling, added mass, coupling of Lagrangian and Eulerian approaches, large mesh movements, contact, change in topology, etc.

Fluid-structure interaction uses the solution of coupled equations governing the fluid and solid motions; therefore, inclusion of FSI effects would add significant complications in a
numerical simulation. The complexity of FSI problems is often determined by the displacement size. Also, transient problems tend to be significantly more complex than steady-state problems. The distinction between large and small displacements is a concept often used in order to identify the level of complexity of a structural mechanics problem.

Fluid-structure interaction is of primary interest in modeling blood flow because of the arterial wall remodeling process, as well as understanding many aspects of vascular physiology, clinical therapy such as the mechanism of disease (hypertension, arteriosclerosis, etc.), materials for treatment, matching of artificial vascular grafts to host vessels, assisting physicians in positively identifying individuals who exhibit the development of vascular disease, and accurate modeling of blood flow.

Also, when the relationship provided by Poiseuille (equation 1.1) is considered, it can be seen that for a smooth flow (laminar flow) the volumetric flow rate is provided by the pressure difference divided by the viscous resistance. This resistance depends linearly upon viscosity and length, but the fourth-power dependence upon the radius is dramatically different. Vessel length does not change appreciably in vivo and, therefore, can generally be considered a constant. Blood viscosity normally does not change very much; however, it can be significantly altered by changes in hematocrit, temperature, and low-flow states. The change in radius will alter flow rate to the fourth-power of the change in radius. The relationship between flow and radius for a single vessel is shown in Figure 1-2, where laminar flow conditions are assumed, and pressure, viscosity, and vessel length are held constant. As vessel radius decreases, there is a dramatic fall in flow rate because flow rate is directly related to radius to the fourth-power. For example, when radius is one-half normal (0.5 relative radius), flow is decreased by a factor of 16. The new flow, therefore, is only about 6 percent of the original flow. Moreover, a 19 percent increase
in the radius will double the volumetric flow rate. This illustrates how very small changes in vessel radius can have dramatic effects on flow. Therefore, vessel resistance is extremely sensitive to changes in radius (or diameter). Nevertheless, the relationship clearly shows the dominant influence of vessel radius on resistance and flow and, therefore, serves as an important concept to understand how physiological and pathological changes in vessel radius affect flow and the use of FSI simulations.

\[
\text{Volume flow rate} = \frac{(\text{Pressure difference})}{(\text{Viscous resistance})} = \frac{\pi \text{(Pressure difference)}(\text{Radius})^4}{8(\text{Length})(\text{Viscosity})}
\]

(1.1)

Figure 1-2. Relationship between flow rate and radius

In recent years, the study of FSI effects in biomechanics has intensified, especially in the field of arterial blood flow. Other examples of application of FSI analysis in cardiovascular research is the modeling of blood flow through the left ventricle [36] and the aortic valve [37]. A three-dimensional model of the natural heart with moving valves [38] was simulated using the
immersed boundary method, which was specifically developed for the study of FSI problems in the cardiovascular system.

Arteries differ in size considerably. Large arteries, in general, are very elastic. Due to their large deformations they can store and release elastic energy, making the blood flow more regular than if they were rigid. Smaller arteries are more rigid when compared to larger arteries. Only large- and medium-sized vessels are considered in the current investigation. Moreover, the important physical parameters considered are pressure, velocity and wall displacement; no consideration is given to the aspects of chemical interactions with the wall or the influence of temperature. However, several difficulties for modeling of the blood flow are as follows:

- The flow is transient. This means that steady-state solutions have only limited value. Because of the pulsatility of blood flow, any numerical simulation should include the time dimension; usually periodicity of cardiac cycles is assumed.
- The fluid interacts with the vessel wall. The radius of the large arteries easily varies by 10 percent. This large deformation affects the flow field considerably.
- Boundary conditions must be provided. Because it is impossible to model the entire cardiovascular system, fictitious boundaries are introduced (inflow and outflow). Of course it is possible to prescribe boundary conditions; however, the problem is what they should be. For example, the assumption that the condition should be non-reflecting could be incorrect, if one considers the fact that the flow in each section is influenced by the entire cardiovascular system.
- The geometry is complex. In addition to the fact that most arteries are curved, the essence of the circulatory system is the branching and merging of vessels by bifurcations.
• Time scales vary. In addition to the fast changes in flow in one cardiac cycle (typically one second), the circulatory system also changes on a far larger time scale (measured in years). Consider the evolution between birth and adulthood, and the possible slow growth of plaque and stiffening of arteries, all of which will change the geometry and thus the flow.

1.6 Objectives

Apart from the challenges described above, the development of numerical methods for FSI is a challenging task. Therefore, available software packages for solving the governing fluid and structural equations are employed. Moreover, these packages can model 3-D geometries and offer a variety of parameters for imposing boundary conditions, parallelization of algorithms and data visualization.

The main objectives of the present research are summarized as follows:

• To validate the computational results with experimental data.
  ➢ Using Newtonian fluid.
  ➢ Using a non-Newtonian model.
  ➢ Using an FSI numerical model.

• To perform numerical simulations considering different arterial geometries using both steady and pulsatile flow cases.

• To provide some indication of the occurrence of atherosclerosis while describing hemodynamic parameters.

• To determine the extent of interaction between blood flow and elastic walls while performing numerical simulations on various arterial geometries with steady and pulsatile flows.
• To investigate the outcome of bypass surgery (various cases) with natural and synthetic grafts, and to determine some indication of the occurrence of intimal hyperplasia.
  ➢ In end-to-side anastomosis surgery.
  ➢ In end-to-end anastomosis surgery.
2.1 Cardiovascular System

The cardiovascular system is a major system in the human body, consisting of the heart and blood vessels, whose purpose is to move blood through the body. The heart muscle acts as a pump in the system, expanding and contracting to receive and send blood, respectively. The blood vessels, including arteries, arterioles, capillaries, venules, and veins, are the piping components that allow for distribution of the blood through the body [39].

Blood vessels carry the blood to and from the heart. Blood vessels can be divided into three major classes: arteries, veins, and capillaries. Arteries carry blood from the heart, while veins carry blood back to the heart. Capillaries are small blood vessels that join the arteries and veins. Through the capillaries, oxygen is transferred from blood to the surrounding tissues. Two major differences exist between blood vessel walls and more traditional engineering pipe materials. Unlike pipes, which are generally rigid and solid, blood vessel walls are both elastic and porous. The consequence of this is that, as blood travels through the blood vessels, the walls expand and contract. In addition, nutrients can be transferred to the surrounding tissues through the pores in the blood vessel walls. These pores are selectively permeable, allowing nutrient transfer while preventing red blood cells from leaving the vessels [40].

Two major circulation paths make up the cardiovascular system – pulmonary circulation, which carries blood between the heart and lungs, and systemic circulation, which carries blood from the heart through the body and back again. The purpose of the pulmonary circulation system is to take deoxygenated blood from the heart to the lungs, where carbon dioxide is released and oxygen is added, and return the blood to the heart. The systemic circulatory system
involves the movement of oxygenated blood from the heart through the body. Nutrients and oxygen can then be transferred from the blood to the tissues that surround the blood vessels. The systemic circulatory system begins in the left atrium, which receives oxygenated blood from the pulmonary veins. The blood then moves from the left atrium to the left ventricle where it is pumped into the aorta, the largest artery in the body. From the aorta, the blood travels through the arteries into the capillaries and returns to the right atrium through the veins [39].

The heart is continually expanding or contracting. This motion creates pulsatile blood flow, which moves the blood as pressure waves through the body. One complete cardiac cycle occurs during every heartbeat. Two important terms describe the motion of the heart chambers during the cycle – systole and diastole. Systole is the contraction of a chamber, while diastole is the relaxation of a chamber [39].

2.2 Blood

Blood is a suspension of red blood cells, white blood cells, and platelets in plasma. The viscoelastic fluid behavior of blood is associated with the elastic properties of the red cell membrane and the viscosity of internal and external fluids. Red blood cells constitute more than 99 percent of the particulate matter in blood and 40 to 45 percent of the blood by volume (hematocrit). The material properties of the red blood cell membrane and the fluidity of its internal contents make it easy for the cell to deform into a variety of shapes. However, the deformation of red blood cells in vitro or in vivo in circulation occurs at an essentially constant area, which can be attributed to the relatively high dilatational modulus of the cell membrane.

After red blood cells, white blood cells are the largest in number. However, they constitute less than 1 percent of the total volume of blood cells in normal human blood and exert little influence on the bulk rheological properties of blood. White blood cells are much less
deformable than red blood cells. White blood cells have a viscoelastic interior that makes them several orders of magnitude stiffer than red blood cells under rapid deformations. The stress required to cause the deformation of white blood cells is much greater than for red blood cells. This indicates that white blood cells are more viscous compared to red blood cells. Platelets occupy even less of blood volume than white blood cells. They play an important role in blood clotting, but they are rheologically unimportant to consider for the normal blood simulation. Adhesion of both red blood cells and white blood cells to blood-vessel walls increases the apparent viscosity. Since one must consider different viscosities for both red and white blood cells, blood must be considered a non-Newtonian fluid for simulations.

2.3 Blood Pressure

The red blood cells transport oxygen and waste products by flowing through the blood vessels. Blood flows through the vessels by blood pressure. Just as water flows through pipes from areas of greater pressure to lesser, blood flows through the body from areas of higher pressure to areas of lower pressure. Blood pressure is measured both as the heart contracts, which is called systole, and as it relaxes, which is called diastole. A systolic blood pressure of 120 millimeters of mercury is considered right in the middle of the range of normal blood pressures, as is a diastolic pressure of 80 mm of mercury. In common terms, this normal measurement would be stated as "120 over 80."

Normal blood pressure is important for proper blood flow to the body's organs and tissues. Each heartbeat forces blood to the rest of the body. The force of the blood on the walls of the arteries is called blood pressure. Blood pressure moves from high pressure near the heart to low pressure away from the heart. Blood pressure depends on many factors, including the amount of blood pumped by the heart. The diameter of the arteries through which blood is pumped is also
an important factor. Generally, blood pressure is higher when more blood is pumped by the heart and the diameter of an artery is narrow.

Stressful situations can result in a temporary increase in blood pressure. If an individual were to have a consistent blood pressure reading of 140 over 90, he would be evaluated for having high blood pressure. If left untreated, high blood pressure can damage important organs, such as the brain and kidneys as well as lead to a stroke.

2.4 Morphology of Arteries

Arteries are muscular vessels that carry blood away from the heart. They are contrasted with veins, which carry blood toward the heart. The circulatory system is extremely important in sustaining life. It’s proper functioning is responsible for the delivery of oxygen and nutrients to all cells, as well as the removal of carbon dioxide and waste products, maintenance of optimum pH, and the mobility of elements, proteins, and cells of the immune system. In the Western countries, the two leading causes of death, myocardial infarction and stroke, are each direct results of arterial system deterioration.

Arteries can be subdivided into several groups with ascending diameter: arterioles (10-100 µm), muscular arteries (diameter > 0.1 mm), and elastic arteries (diameter > 5 mm), such as the aorta, the carotid arteries, coronary arteries, etc. Because atherosclerotic disease mainly occurs in arteries, the current study considers these vessels only. Moreover, in the current study, arteries are considered to be purely elastic. However, their mechanical behavior is not purely elastic but slightly viscoelastic.

As shown in Figure 2-1, arterial wall consists of three distinct layers of tissue, which are called tunica intima, tunica media, and tunica adventitia.
Tunica Intima: The innermost layer, which is in direct contact with the blood, is commonly known as intima. The intima generally consists of a monolayer of endothelial cells that is separated from the sub-endothelium by a thin basal lamina. The endothelial cells have a thickness of about 0.2 to 0.5 µm and form the interface between blood and the arterial wall. Large arteries often have a large subendothelial layer, which grows with age or disease conditions (arteriosclerosis).

Tunica Media: The middle layer, which is in direct contact with the intima and adventitia, is commonly known as media. This is the thickest of the three layers. It is made up of smooth muscle cells and elastic tissue or laminae. These elastic laminae have an average thickness of about 3 µm, are concentric and spaced equidistantly, and will disappear altogether in the small arteries. The smooth muscle cells are arranged in a spiral around the long axis of the vessel. They secrete elastin in the form of sheets, or lamellae, which are fenestrated to facilitate
diffusion. The number of lamellae increases with age (few at birth, 40 to 70 in an adult) and with hypertension. These lamellae, along with the large size of the media, are the most striking histological feature of elastic arteries. Smooth muscle cells are interconnected by a network of elastic fibrils, which renders strength and elasticity to the vessel. These muscles have an elongated shape, with many cellular extensions that connect to the collagen in the media.

**Tunica Adventitia:** The outermost layer is known as the tunica adventitia or the adventitia, and is composed of connective tissue. This is a relatively thin tissue layer. The adventitia of arteries is comprised of only 10 percent of elastic fibers and mainly consists of thick bundles of collageneous fibrils. The collagen in the adventitia prevents elastic arteries from stretching beyond their physiological limits during systole.

### 2.5 Mechanical Behavior of Arteries

As pointed out in the previous section, the main constituents of arteries are collagen, elastin, and smooth muscle. The intima mainly consists of elastin; the media consists of collagen smooth muscle, and some elastin; and the adventitia mainly consists of collagen. To understand the mechanical behavior of arteries, it is necessary to know the mechanical properties of these constituents.

**Collagen:** Collagen is the main protein of connective tissue in animals and the most abundant protein in mammals. It is tough and inextensible, having great tensile strength. It is responsible for skin strength and elasticity, and its degradation leads to wrinkles that accompany aging. It strengthens blood vessels and plays a role in tissue development. Its molecular structure is characterized by three helically wound chains of amino acids that are bundled together in micro fibrils. The fibers are normally arranged in a wavy form with wavelength of 200 µm [42].
Due to its waviness, tissue stiffness is low at small strains and the stiffness increases once the fibers are fully stretched [42]. A typical Young’s modulus of collagen is approximately 500 MPa.

**Elastin:** Elastin is a protein in connective tissue, which is elastic and allows many tissues in the body to resume their shape after stretching or contracting. It also helps skin to return to its original position when it is poked or pinched. It has an almost linear stress-strain relationship, with a Young’s modulus of approximately 500 kPa [42].

**Smooth Muscle:** In arteries, about 40 percent of the media consists of smooth muscle cells [42]. Smooth muscle is used to move matter within the body via contraction. It has a Young’s modulus of 100 kPa [43] in deactivated state whereas in activated state it increases to 200 kPa making the vessel stiffer.

Since the focus of current research is not related to mechanical properties or mechanical models of the arteries, only a brief discussion is presented. As mentioned above, the arterial wall has a layered structure making the wall anisotropic in nature [43]. The elastin in the media bears most of the pressure load at small strains, and the collagen fiber network in the adventitia limits the radial deformability at higher blood pressures and causes the steep rise in wall stiffness at higher strains, making the material nonlinear [43].

### 2.6 Pressure Wall Shear Stress and Wall Strain

When the heart contracts, it ejects a substantial amount of blood from the left ventricle into the arterial system. Due to arterial impedance, the pressure rises accordingly. Because of the large arteries compliance, the pressure will propagate as a wave through them. Due to diminishing compliance and reflections, the peak-to-peak value of the pressure rises slightly toward the aortic bifurcation. A typical value of the pressure gradient in a major artery is on the order of 20 Pa/m, whereas the difference between the peak systole and the end diastole is in the
range of 6 kPa. The local velocity of the pressure wave depends on the local properties of the artery and the instantaneous value of the pressure. Apart from geometrical factors such as stenosis (tapering), mechanical properties dominate the way waves propagate through the vessel. For instance, nonlinear properties and geometrical changes of the arteries alter the shape of the pressure wave, whereas the viscoelastic properties of both fluid and the vessel wall tend to reduce the pressure.

Due to viscosity [44], the blood exerts a drag force proportional to the velocity gradient normal to its surface on the endothelial cells, which is termed wall shear stress and is typically in the range of 3 Pa. As already indicated, geometrical factors play an important role with respect to atherosclerosis. Locations where low and oscillating wall shear stresses occur correlate well with locations where atherosclerotic lesions form such as inside bends and non-divider walls of bifurcations.

As the pressure wave propagates through the arterial system, the vessel deforms accordingly. The value and the directions of the wall strain depend on the pressure, the local geometry of the vessel wall, and the local mechanical properties of the arterial wall material and its surrounding tissue. The way in which vessels are suspended within the body is important. For instance, in a straight vessel, the strains will be approximately homogeneous, whereas in bends and bifurcations, the strains will vary quite significantly locally. In addition to wall shear stresses, wall strains at these locations may also be of importance with respect to the process of atherogenesis.
2.7 Carotid Artery Bifurcation

The common carotid arteries (CCA) lie in the neck, along the vertebral column, and branch into the internal carotid and external carotid arteries. There is often a bulbous enlargement at the bifurcation, at the origin of the internal carotid artery, called the carotid sinus. The internal carotid artery (ICA) enters the cranium and helps in supplying blood to the anterior portion of the brain, while the external carotid artery (ECA) branches into several smaller vessels that supply blood to the neck, face, and scalp.

A bifurcation often affected by atherosclerosis (atherosclerotic plaque formation) is the carotid artery bifurcation. Both stroke and transient ischemic attack can result from thrombolytic and embolitic complications of arteriosclerosis in the common carotid artery or the internal carotid arteries. Atherosclerotic plaques, a manifestation of arteriosclerosis, include fatty streaks (lipids and foam cells), gelatinous plaques (collagen fibers around small lipid droplets), and fibrous plaques. Such attacks may result from a reduction in blood flow due to narrowing of the arterial lumen but are generally caused by emboli originating from ulcerating or high-grade lesions. In order to obtain detailed insight into the flow phenomena in the carotid bifurcation, a number of numerical and experimental studies have been undertaken [45-48] to understand the development of atherosclerosis, and these efforts have produced several theories on atherogenesis and atherosclerosis development, including a cellular response to lipids, a thrombogenic response to molecules in blood, an unchecked healing response to endothelial layer injury, a cancer-like proliferation of smooth muscle cells, chronic inflammation, and a hemodynamic effect on arterial cells [49]. By understanding the mechanisms by which plaque will initiate, grow to occlude the lumen, rupture, or remain stable, clinicians will be able to better predict how plaque might develop or react to treatment. Insight will provide a better basis from
which patients and clinicians can select options for treatment of plaque, whether by arterial bypass, angioplasty, stenting, or other therapy. Typically, localized plaque occurs at geometric structures (e.g., branch points, bifurcations, and curves), which produce fluid dynamics patterns (e.g., low-wall shear stress, recirculation regions, and secondary flows) that co-localize with atherosclerosis development [50]. However, these studies were limited to models with rigid walls, while the walls of blood vessels are flexible, causing wall motion and occurrence of wave phenomena.

2.8 Anastomosis

The surgical joining of two ducts or blood vessels using a natural/synthetic graft to allow flow from one to the other is known as anastomosis. It can be divided into two categories:

- **End-to-End Anastomosis**: This method is generally used for large vessel surgeries. It is the simplest, most reliable, and most widely used method. In this type of surgery, the portion of the artery that is blocked (stenosed) is removed, and a synthetic/natural graft is attached end-to-end (Figure 2-2).

- **End-to-Side Anastomosis**: This method can be used for both smaller and larger vessel surgeries. In this type of surgery, the portion of the artery that is blocked is not removed, but instead a bypass route is created using a synthetic/natural graft (Figures 2-3 and 2-4). The angle of union between artery and synthetic/natural graft should be as small as possible in order to minimize turbulence. Angles less than 60 degrees are usually acceptable.
Figure 2-2. End-to-end anastomosis [51]

Figure 2-3. Schematic of a fully stenosed artery with a bypass graft. The upstream graft-host junction is referred to as the proximal side-to-end anastomosis, and the downstream graft-host junction is referred to as the distal end-to-side anastomosis. Arrows indicate blood-flow direction.
Figure 2-4. End-to-side anastomosis [52]
CHAPTER 3
LITERATURE REVIEW

Various aspects of blood circulation are reported in the literature. The particularly large amount of research work and related publications is due to continued widespread incidence of cardiovascular diseases, especially coronary infarctions and arteriosclerosis. Most of the literature on blood flow is concerned about the bifurcation of the arteries, the velocity profile, pressure distribution, and the shear stress induced at the bifurcation and along the walls of the artery. In most investigations, arterial walls are assumed to behave as rigid instead of elastic walls, whereas the concept of using elasticity is needed to obtain practical and more realistic results.

The expansion of computational fluid dynamics has made it possible to investigate blood flow in the arteries by including most of the physical aspects of blood flow. Researchers have used various numerical methods, such as finite element method (FEM), finite difference method (FDM), and finite volume method (FVM) to solve the respective flow equations. CFD methods are particularly suitable for biofluid problems because biological phenomena often involve nonlinear governing equations, moving boundaries, and irregular boundary geometries.

Numerous experiments have been conducted to study flow in bifurcated branches in order to examine regions of recirculation and separated flow, which are low shear stress areas, or areas with high fluctuations in stress, and have been found to cause various arterial diseases, as previously mentioned. Some of the physical experiments to study velocity and pressure distributions in bifurcated arteries have been conducted by Bharadvaj et al. [53], Ku and Giddens [54], and Motomiya and Karino [55]. Results from these experiments have been used to verify numerical solutions by Fernandez et al. [56], Perktold and Hilbert [57], and Rindt et al. [58].
Bharadvaj et al. [53] were the first to focus attention on the geometry of the carotid bifurcation. The average size of the carotid artery bifurcation was measured from the angiograms of 67 subjects. They created a glass model with that geometry. In their steady flow study, streaklines were shown by using hydrogen bubbles and dye. Using the same techniques of Bharadvaj et al. [53], Ku et al. [59, 60] conducted experiments using the pulsatile flow conditions. In the later stage, Van de Vosse et al. [61], studied both stenosed and non-stenosed carotid bifurcations using the laser Doppler velocimetry (LDV) technique. Subsequently, Anayiotos [62] conducted experiments using a compliant bifurcation model and found that mean shear stress at the wall is affected by the cross-sectional changes of the model.

Perktold et al. [63] first simulated Newtonian flow in a two-dimensional carotid artery bifurcation for a Reynolds number of 100. Later the problem was extended to a three-dimensional geometry for a higher Reynolds number. Perktold et al. [64] considered three-dimensional, pulsatile flow using a FEM scheme and compared the effects of Newtonian and non-Newtonian flows. Their investigation showed that the difference between non-Newtonian fluid and Newtonian fluid was small, and no major flow pattern differences were found. Similar results were found by Baaijens et al. [65] and Lou et al. [66].

Nazemi et al. [67] developed a model of the plaque formation process in the carotid bifurcation. In their model, plaque formed where the shear stress values fluctuate from high to low values. According to their two-dimensional study, plaque deposits appear not only in the carotid sinus but also in the external carotid and at the divider wall between the sinus and the internal carotid.

Both Reuderink [68] and Perktold et al. [69] numerically studied the flow through a compliant bifurcation model. However, their results disagreed with the experiments of Anayiotos.
This discrepancy in results was later assumed to be from the arterial cross-section, which remained circular all the time, and also due to treating the dynamics of the walls and fluid dynamics separately.

The physiological range of the Reynolds number in the carotid artery bifurcation is between 100 and 1,000, which is well below the critical Reynolds number (transition Reynolds number). Whether turbulence occurs in the physiological region is still an open question. If turbulence occurs in this physiological range, then it might occur locally or during systole. Under normal physiological conditions, no turbulence was found for steady flow in the carotid artery bifurcation by Ku et al. [59], whereas, Gidden et al. [70] pointed out that turbulence can occur when stenosis reaches a sufficient degree.

Arteries experience nonlinear, large deformations, and early – and late-stage plaques influence stress distributions on the wall. Areas of significant thickening, especially areas with plaque, correlate with a nonlinear increase in carotid artery wall stiffness [71]. Salzar et al. [72] reported that mechanical stress varies over a model of the carotid bifurcation, with the highest stress concentration at the bifurcation and across the sinus bulb.

Endothelial cells, which line the wetted surface of the artery wall, are mechanotransducers, transferring fluid dynamics stresses imposed by the blood into biochemical signals of the vascular cells. One theory of signal transduction is that the stiffness of the membrane itself serves as a sensor for wall shear [73]. The range of mean wall shear stress is 10 to 20 dynes/cm² for many mammalian arteries under normal flow conditions [74]. Arteries respond to transient or long-term deviations from normal flow conditions through vasodilation/vasocostriction or by vessel remodeling, respectively [75-77]. Chronic changes in blood flow trigger a compensatory mechanism in the arteries. Increases in flow result in
remodeling of the arterial wall to increase lumen diameter and thus to maintain wall shear stress levels within the normal wall shear stress range, suggesting a coupling between blood flow and artery wall behavior [76].

The blood flow in an artery with two successive bends and the effect of curvature of the bend was investigated by Hoogstraten et al. [78]. Similar studies were carried out on curved pipes by Berger et al. [79]. In both the studies, they found out that the locations at bends which corresponds to the elevated WSS are susceptible for the development of atherosclerosis.

Various factors affect the arterial blood flow: arterial geometry, flow parameters, pulsatile flow, elasticity of arterial wall, and non-Newtonian behavior of blood. Since the geometry of an artery is of primary importance in blood flow, several authors have introduced various bifurcation geometries [80-84]. Regarding the geometry issue, Perktold et al. [85] investigated various bifurcation angles and presented the effects on blood flow due to changes in geometric bifurcation angles. In a recent study by Younis et al. [86], magnetic resonance phenomena was used to identify the exact geometry of the human artery. Subsequently, that information was used to model the arterial geometry for numerical simulation. In a more recent publication, Gerbeau et al. [87] addressed difficulties in the simulation of blood flow in compliant vessels. Their point of discussion was how to generate fluid and structural meshes of the artery and the solution of the fluid-structure problem with large arteries. They suggested some of the techniques relative to how and which elements should be used to generate the meshes.

Severe arterial stenoses can be diagnosed by measuring pulse wave propagation and reflection in terms of blood pressure. As a result, several researchers have been investigating stenoses using CFD. In one case, Bathe et al. [88] considered a fully coupled problem of
pulsatile blood flow through a compliant axisymmetric stenotic artery using the finite element method. In their investigation, the effect of increasing area reduction on fluid dynamic and structural stresses was considered. Tang et al. [89, 90] considered a 3-D thin – and thick-wall model of the artery with FSI. In their investigation, the software ADINA was used to explore wall deformation and flow properties of blood flow in carotid arteries with symmetric and asymmetric stenosis. Several other efforts using FSI analysis in cardiovascular research include modeling of blood flow through the left ventricle [91] and the aortic valve [92]. A three-dimensional model of the natural heart with moving valves [93] was simulated using the immersed boundary method, which was specifically developed for the study of FSI problems in the cardiovascular system.

Anastomotic intimal hyperplasia, especially at the distal end-to-side/end-to-end anastomosis, is one of the major causes of failure of arterial bypass grafts [11, 12]. This disease is characterized by a progressive thickening of the inner most layer of the artery wall in the region where the graft is joined to the artery. As a result, gradual narrowing of the vessel leads to a blockage of the vessel. IH is more prevalent at the distal anastomosis of an end-to-side graft, where the flow is more disturbed rather, than at the proximal anastomosis [94].

Although there are several theories in the literature, the exact cause of IH is uncertain [11, 13-15]. The development of IH in bypass grafts depends on several factors, such as injury, hemodynamics, and graft-artery compliance mismatch. Fluid dynamics within the vascular grafts (hemodynamics) is widely believed to play an important role in the initiation and development of anastomotic IH [16-18]. However, the specific mechanism(s) whereby blood flow patterns influence the development of IH remains a major unsolved question in bioengineering. In the past, a variety of hemodynamic factors have been proposed in the development of IH, which
includes recirculating zones, turbulence, low and oscillating shear stress (OSS), and temporal and spatial variations of the wall shear stress. However, to determine the exact influence of these factors, quantitative hemodynamic information is required. Several experimental and computational studies have been performed to investigate the correlation between distal anastomotic IH and hemodynamics. Various experimental techniques such as ultrasound Doppler techniques in vitro and in vivo [95, 96], Laser-Doppler anemometry measurements of flow visualizations in vitro [97, 98], and photochromic dye tracer in vivo [99] have been employed by researchers for quantitative flow measurements.

Pietrabissa et al. [100] simulated steady Newtonian blood flow in an aorta-coronary bypass model, while Perktold et al. [101] simulated pulsatile non-Newtonian blood flow. In both of these investigations, a separated region at the toe of the anastomosis was observed. Ojha et al. [102, 103] found regions of low WSS at the heel and toe of the anastomosis along with sharp temporal and spatial variations of WSS along the floor of the artery. Similarly, Bassiouny et al. [104] observed a stagnation zone along the floor of the artery, while separated and secondary flows occurred at the heel and toe of the graft. Bharadwaj et al. [105] indicated that IH is greater in regions of flow separation and that distal anastomosis was more susceptible to hyperplastic lesions than the proximal junction. Fie et al. [106] investigated the effect of artery-graft angle and flow rate upon hemodynamics in distal vascular graft anastomoses. Henry et al. [107] showed that the regions corresponding to IH formation in an end-to-side anastomosis had low WSS with increased spatial wall shear stress gradients (WSSG). Lei et al. [108, 109] and Kleinstreuer et al. [110] performed pulsatile flow simulations in various end-to-side anastomosis models and calculated WSS, WSSG, and OSS and correlated the formation of the IH with
respect to these parameters. Steinmann et al. [99] correlated the areas of extensive IH formation with high instantaneous WSS.

Among the end-to-side anastomosis studies reviewed, the hemodynamic predictor of IH formation has not been agreed upon by various researchers. This is primarily due to the lack of uniform treatment of flow conditions. Furthermore, among these studies the comparison of numerical results with experimental data either has not been conducted or has been conducted in a simplified vitro model using various flow-visualization methods. To verify the validity and versatility of numerical simulations, quantitative comparisons between numerical results and experimental data in real geometries are needed. A quantitative comparison of WSS under the same model geometry would help to delineate the hemodynamic characteristics and identify the important mechanical factor(s) that may be involved in the formation of IH. Therefore, this study was undertaken with the goal of identifying those hemodynamic characteristics that could potentially lead to IH in both end-to-side and end-to-end anastomosis. Also, this investigation was extended beyond the identification of the hemodynamic parameters. Numerical investigation was conducted to assess the effect of compliance mismatch while matching the geometry criterion (artery and graft with the same nominal radius) between the artery and graft considering end-to-end anastomosis surgery.
CHAPTER 4

GOVERNING EQUATIONS AND NUMERICAL APPROACH

For this research, a commercial software package was chosen instead of a code developed in-house. The use of a commercial code allowed significant savings in development time and reduced the need for extensive validation testing. A disadvantage of using a commercial code is the lack of flexibility in controlling certain aspects of the pre- and post-processing as well as the solvers being used. However, considering the time and effort required to develop and test an in-house code, it was determined that the advantages of a commercial code exceed its disadvantages for this research.

A mandatory criterion for choosing a commercial software package for this research was the existence of FSI capabilities. Another essential requirement was the ability to allow various functions as subroutines. Additional non-essential but desirable criteria included the availability of a parallel version to increase the speed at which simulations could be carried out, as well as the availability of both Windows and Linux versions, allowing for computations on different computer systems. Among the various available codes, FIDAP [111] and FLUENT [112] were selected for the current research.

4.1 Fluid-Structure Interaction Theory

Fluid-structure interaction scenarios are those that involve the coupling of fluid mechanics and structural mechanics. In an FSI problem, the stresses and deformations of a given structure are computed simultaneously with the flow parameters that surrounds the structure. That is, the deformations of the solid structure are due to the pressure gradients of the fluid, and likewise the pressure and velocities depend upon the deformation of the structure.
Fluid-structure interaction problems can be simple or complex. For example, the computation of stresses for a given flow field and fixed structural configuration represents a simple problem. Complex problems are those that involve large structural deformations, changes in topology, contact problems, and geometric and/or material nonlinearities. The complexity of any given FSI problem is determined, in large part, by the extent to which the structural component is displaced. Simple FSI problems typically involve structures that do not undergo large displacements. Conversely, problems that involve large structural displacements tend to be complex.

With respect to structural displacements in FSI problems, the terms "small" and "large" are relative and problem-specific. In some problems, displacements on the order of a few millimeters can greatly affect the flow field. As a general rule, however, displacements are considered large only if they are of the same order of magnitude of the smallest length scale that is relevant in the simulation.

4.1.1 Small Displacements

For FSI problems that involve small displacements, the fluid-flow field can be solved assuming that the structural displacement does not affect the flow field. As a result, steady-state FSI problems with small displacements involve only a one-way coupling between fluid and structure. That is, displacements (and stresses) of the structure are affected by the flow field, but the flow field is not affected by the structure. The most appropriate and cost effective way to approach such problems is to set up and solve the fluid flow and structural problems as two completely separate problems.

For transient problems, it is necessary to transfer velocities from the structure to the fluid. For example, in a problem that involves a structure undergoing high-frequency oscillations, the
fluid-flow problem may not be affected by structural deformation but may be affected by the velocity boundary condition at the fluid-structure interface where velocities are transferred from the structure to the fluid.

### 4.1.2 Large Displacements

For FSI problems that involve large displacements, the flow field is affected by structural deformations. As a result, the fluid and structure exhibit a two-way coupling, and it is necessary to resolve the fluid flow problem after each update of the structural configuration. In this sense, the FSI problem can be considered a moving-boundary problem. An additional level of nonlinearity is introduced in such problems, because the fluid and structural problem must be solved iteratively.

Differences in the flow field due to structural deformation are primarily caused by the effect of such deformation on the mesh. Therefore, in problems that involve large displacements, the fluid mesh must be adjusted after each update of the structural configuration. When the structural displacements are of the same order of magnitude as a characteristic length of the flow domain, the re-meshing problem becomes extremely complex and is likely to constitute the most challenging step in the simulation.

### 4.1.3 Coupling Between Fluid and Structure Equations

In its most general form, the mathematical modeling of FSI problems requires the concurrent application of techniques from the fields of computational fluid dynamics (CFD), computational structural dynamics (CSD), and computational mesh dynamics (CMD). Specifically, FSI problems require mathematical coupling of the fundamental equations from each of these three fields.
There are two basic types of coupling algorithms:

- Strong
- Loose

In the strong (or monolithic) approach, CFD equations that describe the behavior of the fluid and CSD equations that describe the behavior of the structure are treated as a single coupled system of equations and are solved simultaneously. In the loose (or staggered) approach, CFD and CSD equations are solved independently of each other.

The strong approach is sometimes suitable for small structural problems but is often slow and requires large amounts of memory. In addition, the full set of algebraic equations associated with the strong approach can be too stiff, because the properties and characteristic scales of the CFD, CSD, and CMD equations often differ significantly from each other. The loose approach, on the other hand, requires relatively small amounts of memory and employs solution techniques uniquely tailored to each field of CFD, CSD, and CMD.

To perform FSI analysis in FIDAP, separate fluid and solid models must be defined. These models are coupled through coincident FSI boundaries during the solution process. Details on the equations governing these models as well as the implementation and solution of this system in FIDAP are presented in the following sections.

4.2 Fluid

The treatment of the fluid domain for FSI problems is identical to that for other CFD problems that involve moving boundaries. The arbitrary Lagrangian-Eulerian (ALE) formulation is employed for such problems. The ALE formulation is the most convenient and natural
approach to describe the fluid motion, in large part because it is based on a mixed Eulerian-Lagrangian viewpoint.

4.2.1 Governing Equations

Continuum fluid flow is governed by Navier-Stokes equations. These equations represent the differential forms of the three basic conservation principles for fluid flows. The first one of the Navier-Stokes equations is the continuity equation, which is a statement of the conservation of mass. The second one is called the momentum equation, which arises from the conservation of momentum. The third equation is obtained by applying the conservation of energy to the fluid flow, and this equation is called the energy equation. This research assumes isothermal conditions for the simulations, and therefore, the energy equation is mentioned here for the sake of completeness.

The principle of mass conservation applied to flowing fluid results in the continuity equation, which can be expressed as

$$\frac{\partial \rho}{\partial t} + \left( \rho u_j \right)_j = 0 \tag{4.1}$$

where \( j = 1, 2 \) for two-dimensional or axisymmetric flows, \( j = 1, 2, 3 \) for three-dimensional flows, \( \rho \) is density, \( u \) is velocity, \( \frac{\partial}{\partial t} \) is the referential time derivative, and \( t \) is time.

For constant density fluids, equation (4.1) reduces to

$$u_{j,j} = 0 \tag{4.2}$$

and is known as the "incompressibility constraint."

The momentum equation is expressed as

$$\rho \left( \frac{\partial u_i}{\partial t} + u_j u_{i,j} \right) = \sigma_{i,j} + f_i \tag{4.3}$$
Where again \( i, j = 1, 2 \) for two-dimensional or axisymmetric flows, \( i, j = 1, 2, 3 \) for three-dimensional flows, and \( \sigma \) is the stress tensor, and \( f \) is the body force per unit mass.

In most cases, \( \rho f \) represents the force due to gravity, in which case \( f \) is gravitational acceleration, \( g \). However, the term \( \rho f \) can also represent a Coriolis or centrifugal force for cases in which the equations are written relative to a rotating frame of reference. It can also represent a Lorenz force for cases in which magneto hydrodynamic effects are present. More generally, this term can be interpreted as any linear combination of such forces.

For a fluid, the stress tensor can be written as

\[
\sigma_{i,j} = -p \delta_{ij} + \tau_{ij}
\]  

(4.4)

where \( p \) is pressure, \( \tau \) is the deviatoric stress tensor, and \( \delta \) is the Kronecker delta. The material properties of the fluid determine the constitutive relation between the deviatoric stress and the strain rate tensor, \( s \), defined by

\[
s_{ij} = \frac{1}{2} (u_{i,j} + u_{j,i})
\]  

(4.5)

For viscous, compressible (variable-density) fluids, the general relation has the form

\[
\tau_{ij} = 2\mu s_{ij} + \left( \mu_b - \frac{2}{3} \mu \right) u_{k,i} \delta_{ij}
\]  

(4.6)

where \( \mu \) is the dynamic viscosity, and \( \mu_b \) is referred to as the bulk viscosity which is proportional to \( \mu \).

For viscous, incompressible fluids, equation (4.6) can be written as

\[
\tau_{ij} = 2\mu s_{ij}
\]  

(4.7)

Combining equations (4.4), (4.5), and (4.7) and substituting the result in equation (4.3) yields
\[
\rho \left( \frac{\partial u_i}{\partial t} + u_j u_{i,j} \right) = -p_j + \left[ \mu (u_{i,j} + u_{j,i}) \right]_j + pf_i
\] (4.8)

which is known as the stress divergence form of the momentum equation for viscous incompressible fluid. If the incompressibility constraint, equation (4.2), is applied and constant viscosity is assumed, then equation (4.8) takes the form

\[
\rho \left( \frac{\partial u_i}{\partial t} + u_j u_{i,j} \right) = -p_j + \left[ \mu (u_{i,j}) \right]_j + pf_i
\] (4.9)

which is known as the Navier-Stokes form of the momentum equation.

Viscosity can be a function of temperature, species concentrations, position, time, and/or strain rate. If it is independent of strain rate, the relationship shown in equation (4.7) is linear, and the fluid is referred to as Newtonian. If the viscosity is a function of strain rate, the relationship in equation (4.7) is nonlinear, and the fluid is referred to as non-Newtonian.

For non-Newtonian fluids, it is customary to define the strain rate tensor, \( \varepsilon \), by

\[
\varepsilon_{ij} = \left( u_{i,j} + u_{j,i} \right) = 2s_{ij}
\] (4.10)

Therefore, equation (4.7) becomes

\[
\tau_{ij} = \mu \varepsilon_{ij}
\] (4.11)

And for viscoelastic fluids, equation (4.11) is generalized to the form

\[
\tau_{ij} = \mu \varepsilon_{ij} + \tau^E_{ij}
\] (4.12)

where \( \tau^E \) is the elastic contribution to the stress tensor.

### 4.2.2 Non-Newtonian Model

For the non-Newtonian case, the shear effect on viscosity must be considered. There are several models in the literature to account for the non-Newtonian behavior of blood, e.g., the Casson model [113] and several models listed in [114]. Yeleswarapu [115] provides a detailed
report on these models. In the current investigation, the complex rheological behavior of blood is approximated using the shear-thinning Carreau-Yasuda model, where the apparent viscosity is expressed as a function of the shear rate, as shown in equation (4.10). Parameters for the Carreau-Yasuda model are taken from Shaik et al. [116] and Gijsen et al. [117], where Gijsen et al. correlated the model parameters with the experiments as

\[
\frac{\mu - \mu_\infty}{\mu_0 - \mu_\infty} = \left[1 + (\lambda \dot{\gamma})^a\right]^{(n-1)/a}
\]  

(4.10)

where \(\dot{\gamma}\) is the shear rate, \(\mu_\infty\) is the shear rate viscosity at infinite shear rates, \(\mu_0\) is the zero shear rate viscosity, \(\lambda\) is the relaxation time in seconds, and parameters \(a\) and \(n\) can be varied to obtain the power law region of the shear thinning behavior of blood. The numerical simulations were conducted with the parameters shown in Table 3:

<table>
<thead>
<tr>
<th>(\mu_\infty) (Pa.s)</th>
<th>(\mu_0) (Pa.s)</th>
<th>(a)</th>
<th>(n)</th>
<th>(\lambda) (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.2 x 10^{-3}</td>
<td>22 x 10^{-3}</td>
<td>0.644</td>
<td>0.392</td>
<td>0.110</td>
</tr>
</tbody>
</table>

### 4.2.3 Arbitrary Lagrangian-Eulerian (ALE) Method

The motion of fluids and solids can be described in two ways, Lagrangian and Eulerian. In the Lagrangian formulation, the motion of particles is tracked in space, whereas in the Eulerian formulation, a fixed volume is observed as particles move through it. What this means...
in finite element analysis is that, in the Lagrangian formulation, material points are fixed to the mesh, while in the Eulerian formulation, material points move through a fixed mesh [118]. For this reason, the Lagrangian formulation is typically used for solids, while the Eulerian formulation is used for fluids. A problem arises, however, in the use of a purely Eulerian formulation for a fluid model containing moving boundaries, since the fixed mesh through which the particles move is also moving. In this case, a hybrid formulation known as the ALE method is appropriate [118]. In this method, the Eulerian formulation is used at fixed boundaries, the Lagrangian formulation is used at moving boundaries, and the ALE formulation is used at the fluid-structure interface. In the governing equations of the ALE formulation, the actual fluid velocity with respect to a fixed mesh is replaced by a relative velocity relating the actual fluid velocity to the mesh velocity. In doing so, the momentum equation is modified as

$$\rho \left( \frac{\partial \hat{u}_i}{\partial t} + u_{i,j} \left( u_j - \hat{u}_j \right) \right) = \sigma_{ij,j} + \rho f_i$$

(4.10)

where $\hat{u}$ is the mesh velocity.

### 4.3 Solid Structure

The structural domain of an FSI problem typically exhibits nonlinear structural behavior. Such behavior can be classified as follows:

- **Material nonlinearity**: The stress-strain relationship is nonlinear, such as for materials that exhibit plasticity.
- **Geometric nonlinearity**: The displacements and rotations are large, and strains are either large or small.

Geometrically nonlinear analyses of structures are usually based on the incremental Lagrangian (material) formulation referring to a known (prescribed or previously calculated)
equilibrium configuration. Within this formulation, either one of two approaches can be employed:

- **Total Lagrangian (TL):** All static and kinematic variables are referred to the initial configuration.
- **Updated Lagrangian (UL):** All static and kinematic variables are referred to the most recently calculated configuration.

The structural domain of the FSI problem can be described by the following elastodynamic equations:

**Momentum Equations:**

\[
\rho \ddot{d}_i = \sigma_{ij,j} + \rho \ddot{f}_i \quad \text{in} \; ^s\Omega(t) \quad (4.11)
\]

**Equilibrium Conditions:**

\[
\sigma_{ij} n_j = ^s\tau_i \quad \text{on} \; ^s\Gamma(t) \quad (4.12)
\]

where \( \rho \) is the material density, \( \sigma \) is the Cauchy stress tensor, \( \vec{f} \) is the externally applied body force vector at time \( t \), \( ^s\tau \) is the externally applied surface traction vector at time \( t \), \( \ddot{d} \) represents the acceleration of a material point, \( d \) is the displacement of a material point, \( ^s\Omega(t) \) is the structural domain/volume at time \( t \), \( ^s\Gamma(t) \) is the boundary surface of the structural domain at time \( t \), and \( n \) is the outward pointing normal vector on \( ^s\Gamma(t) \).

**Constitutive Equation:**

\[
\sigma_{ij} = D_{ijkl} \epsilon_{kl} \quad \text{in} \; ^s\Omega(t) \quad (4.13)
\]

where \( D \) is the material (Lagrangian) elasticity tensor, and \( \epsilon \) is the infinitesimal strain tensor, the components of which are defined by
According to the incremental Lagrangian formulation, the weak form of the momentum equations of the elastic structural body at time $t$ can be expressed by means of the principle of virtual displacement together with the Gauss theorem as

$$
\int_{\Omega(t)} \rho \ddot{\delta} l_i \delta d_i d\Omega + \int_{\Omega(t)} \sigma \ddot{\delta} l_i d\Omega = \int_{\Omega(t)} \rho \ddot{f}_i d\Omega + \int_{\Gamma(t)} \tau_i d\Gamma
$$

where $\ddot{\delta} l_i$ is the virtual displacement vector. This formulation is nonlinear because, at any time step, the current domain, $\hat{\Omega}(t)$, is unknown (i.e., geometry nonlinearity because of large displacements). Thus, at every time step, the solution is obtained in increments, where at each stage, the strains and stresses are measured with respect to a reference configuration.

The total Lagrangian formulation uses $\hat{\Omega}(0)$ as a reference configuration, whereas the updated Lagrangian formulation uses $\hat{\Omega}^k(t)$, that is, the most recent available configuration at increment $k$ and time $t$. In either formulation, the weak form can be written as

$$
\int_{\Omega(t)} \rho \ddot{\delta} l_i \delta d_i d\Omega + \int_{\Omega(t)} S_{ij} \delta \varepsilon_{ij} d\Omega = \int_{\Omega(t)} \rho \ddot{f}_i d\Omega + \int_{\Gamma(t)} \tau_{ij} \delta d_i d\Gamma
$$

where $S$ and $\varepsilon$, respectively, are the second Piola-Kirchhoff stress tensor and the strain tensor. The tensors $S$ and $\sigma$ are related by the matrix equation

$$
S = J F^{-1} \sigma F^T
$$

where $J$ is the Jacobian, and $F$ is the deformation gradient tensor. The tensors $F$ and $E$ are related to the deformation by

$$
F_{ij} = \delta_{ij} + d_{i,j}
$$

$$
\varepsilon_{ij} = \frac{1}{2} \left( d_{i,j} + d_{j,i} + d_{k,i} d_{k,j} \right)
$$
For the case of large deformations involving small strains, $F$ essentially represents a rotation, which leads to the following simplified expression for $S$ as

$$S_{ij} = D_{ijkl} \varepsilon_{kl}$$  \hfill (4.20)

The model represented by equation (4.20) is applicable for those cases in which strains are small, regardless of whether rotations are large or small. Furthermore, in the current research it was assumed that the structural material is homogeneous, isotropic, linearly elastic, and characterized by its Young's modulus, $E$, and Poisson ratio, $\nu$.

For the 2-D plane stress case, the elasticity tensor, $D$, is expressed as

$$D = \begin{bmatrix} 1 & \nu & 0 \\ \nu & 1 & 0 \\ 0 & 0 & \frac{1-\nu}{2} \end{bmatrix} \frac{E}{1-\nu^2}$$  \hfill (4.21)

4.4 Fluid Structure Interface

Once the separate fluid and solid models are defined, they can be solved simultaneously with information transferring between the two models through coincident FSI boundary conditions. Because the solid displacements affect the fluid motion and the applied stress from the fluid affects the solid motion, two-way fluid-structure coupling must be used to solve the governing equations.

For a solution to be obtained along the FSI boundary, dynamic and kinematic compatibility conditions must be satisfied. The kinematic compatibility condition is

$$F_d = S_d$$  \hfill (4.22)

where $F_d$ and $S_d$ are the fluid and solid displacements on the FSI boundary.

With a no-slip wall boundary condition, the above equation leads to the fluid velocity on the FSI boundary being defined as
\[ F_u = S_u \]  \hspace{1cm} (4.23)

The dynamic compatibility condition (the surface force acting on the structure should be opposite to the force acting on the fluid) is

\[ F_g = -S_g \]  \hspace{1cm} (4.24)

where \( F_g \) and \( S_g \) are the fluid and solid forces at the interface on the FSI boundary.

### 4.5 Mesh Domain

For FSI problems that involve large displacements of an elastic structure, the fluid mesh must be regenerated after each update of structural displacement. To generate such meshes, an elasticity-based model, in which the mesh is modeled as a pseudo-elastostatic medium must be employed. This approach is relatively simple and makes it possible to control mesh distortion by means of the proper choice of elastic material properties for the pseudo-elastic medium.

### 4.6 Finite Element Formulation

#### 4.6.1 Computational Fluid Dynamics

The ALE form of the Navier-Stokes equation is used to solve for fluid flow for FSI problems. After spatial discretization by the FEM, the Navier-Stokes equations are expressed in the ALE formulation in matrix notation as

\[
Ma + N(u - \hat{u})u + Dv - Cp = f \ \text{in} \ F\Omega(t) \hspace{1cm} (4.25)
\]

\[
C^T u = 0 \ \text{in} \ F\Omega(t) \hspace{1cm} (4.26)
\]

where \( M \) represents the fluid mass matrix; \( N, D, \) and \( C \) are, respectively, the convective, diffusive, and divergence matrices; \( f \) is an external body force; vectors \( a, u, \) and \( p \) contain the unknown values of acceleration, velocity, and pressure, respectively; \( \hat{u} \) is the mesh velocity; and \( F\Omega(t) \) is the moving spatial domain upon which the fluid is described.
The following compatibility conditions are imposed on the interface between fluid and structure

\[ F \mathbf{u} = S \mathbf{u} \text{ on } \Gamma(t) \]  
(4.27)

\[ F \mathbf{a} = S \mathbf{a} \text{ on } \Gamma(t) \]  
(4.28)

where \((F)\) indicates values related to nodes placed in the fluid; \((S)\) indicates values related to the nodes placed in the structure; and \(\Gamma(t)\) is the interface between fluid and structure at time \(t\). The variables \(S \mathbf{u}\) and \(S \mathbf{a}\) are calculated at the previous iteration loop in CSD.

The fluid velocity and pressure field are solved after initial conditions, boundary conditions, and compatibility conditions are imposed on the fluid domain. For calculating the traction at the interface between fluid and structure vectors, \(\mathbf{a}, \mathbf{u}, \text{ and } \mathbf{f}\) are decomposed into the following:

\[
\begin{align*}
\mathbf{a} &= \begin{bmatrix} F \mathbf{a} \\ U \mathbf{a} \\ \Gamma \mathbf{a} \end{bmatrix}, & \mathbf{u} &= \begin{bmatrix} F \mathbf{u} \\ U \mathbf{u} \\ \Gamma \mathbf{u} \end{bmatrix}, & \mathbf{f} &= \begin{bmatrix} F \mathbf{f} \\ U \mathbf{f} \\ \Gamma \mathbf{f} \end{bmatrix}
\end{align*}
\]

(4.29)

where \((U)\) indicates values related to nodes on the velocity boundary, \((\Gamma)\) indicates values related to the fluid nodes on the interface between fluid and structure, and the symbol \((\Gamma)\) denotes the prescribed values. The load applied by the fluid on the structure along the interface between fluid and structure is obtained according to partitioned equation (4.30) as

\[
\begin{align*}
\begin{bmatrix}
FF & FU & FI \\
UF & UU & UI \\
IF & IU & II
\end{bmatrix}
\begin{bmatrix} F \mathbf{a} \\ U \mathbf{a} \\ \Gamma \mathbf{a} \end{bmatrix}
+ \begin{bmatrix}
FF & FU & FI \\
UF & UU & UI \\
IF & IU & II
\end{bmatrix}
\begin{bmatrix} F \mathbf{u} \\ U \mathbf{u} \\ \Gamma \mathbf{u} \end{bmatrix}
- \begin{bmatrix}
FF & Fu & FI \\
UF & UU & UI \\
IF & IU & II
\end{bmatrix}
\begin{bmatrix} F \mathbf{C} \\ U \mathbf{C} \\ \Gamma \mathbf{C} \end{bmatrix}
= \begin{bmatrix} F \mathbf{f} \\ U \mathbf{f} \\ \Gamma \mathbf{f} \end{bmatrix}
\end{align*}
\]

(4.30)
And the continuity equation (4.26) can be expressed as

\[
\begin{bmatrix}
F C^T & U C^T & I C^T
\end{bmatrix}
\begin{bmatrix}
F u \\
U \bar{u} \\
I u
\end{bmatrix} = 0
\] (4.31)

For the loose coupling approach, the equations (4.30) and (4.31) must be rearranged so that they are explicit in the fluid unknowns. Thus,

\[
 FF^T \begin{bmatrix} F & \dots & F \end{bmatrix} a + FF^T \begin{bmatrix} N & \dots & N \end{bmatrix} u - F \begin{bmatrix} F & \dots & F \end{bmatrix} p = \begin{bmatrix} F & \dots & F \end{bmatrix} \bar{f} - \begin{bmatrix} F U & \dots & F U \\
F I & \dots & F I \\
F T & \dots & F T 
\end{bmatrix} \begin{bmatrix} \bar{u} \\
\bar{u} \\
\bar{u}
\end{bmatrix} - \begin{bmatrix} F U & \dots & F U \\
F I & \dots & F I \\
F T & \dots & F T 
\end{bmatrix} \begin{bmatrix} \bar{u} \\
\bar{u} \\
\bar{u}
\end{bmatrix} (4.32)
\]

\[
 F C^T F u = - U C^T U \bar{u} - I C^T I u
\] (4.33)

In this case, the compatibility conditions at the wetted surface (fluid-structure interface), equations (4.27 and 4.28), can be interpreted as forcing functions applied by the elastic body on the fluid.

The load applied by the fluid on the elastic body along the wetted surface, \( \dot{I}T(t) \), is obtained from

\[
\dot{I} f = \begin{bmatrix} IF & IU & II \end{bmatrix} \begin{bmatrix} F & \dots & F \\
U & \dots & U \\
I & \dots & I 
\end{bmatrix} \begin{bmatrix} a \\
\bar{a} \\
\bar{a}
\end{bmatrix} + \begin{bmatrix} IF & IU & II \end{bmatrix} \begin{bmatrix} F & \dots & F \\
U & \dots & U \\
I & \dots & I 
\end{bmatrix} \begin{bmatrix} u \\
\bar{u} \\
\bar{u}
\end{bmatrix}
\]

\[
+ \begin{bmatrix} IF & IU & II \end{bmatrix} \begin{bmatrix} F & \dots & F \\
U & \dots & U \\
I & \dots & I 
\end{bmatrix} \begin{bmatrix} u \\
\bar{u} \\
\bar{u}
\end{bmatrix} - \begin{bmatrix} I \cdot \nabla \cdot u \end{bmatrix} (4.34)
\]

where \( \dot{I} f \) is the external force vector at the interface applied by the structure on the fluid; \((IF)\) indicates values related to interface nodes related to the fluid; \((IU)\) indicates values related to the interface nodes related to the velocity boundary; \((II)\) indicates values related to the interface nodes; \( \dot{I} \tau \) is the traction applied by the fluid on the structure along the interface; and \( M, N, D, C, \)
\(a, u, \) and \(p\) are known values solved in the previous time step in this equation. The traction \(\tau_I\) thus calculated is imposed on the structure in CSD.

### 4.6.2 Computational Structural Dynamics

The overall structural behavior is solved based on the UL formulation described in Section 4.3. The corresponding matrix equation can be written as

\[
\dot{S}\mathbf{M}\ddot{S}\mathbf{a} + \dot{S}\mathbf{K}\dot{S}\mathbf{d} = \dot{S}\mathbf{f} + \dot{S}\tau - \dot{S}\sigma \quad \text{in } \dot{S}\Omega(t) \tag{4.35}
\]

where \(\dot{S}\mathbf{M}\) and \(\dot{S}\mathbf{K}\) are the structural mass and nonlinear stiffness matrices; \(\dot{S}\mathbf{f}\) is the external body force vector; \(\dot{S}\tau\), calculated using equation (4.34) in CFD, is the traction applied by the fluid on the structure along the interface; \(\dot{S}\sigma\) is the force due to the internal stresses at the most recently calculated configuration; \(\dot{S}\mathbf{d}\) is the vector of increments in the nodal point displacement; \(\dot{S}\mathbf{a}\) is the vector of nodal point acceleration; \(\dot{S}\Omega(t)\) is the structure domain at time \(t\). However, \(\dot{S}\mathbf{f}\) and \(\dot{S}\sigma\) are equal to zero in this study. The equation of motion can be integrated and the displacement, velocity, and acceleration vectors can be calculated.

The displacement at the interface between fluid and structure can be calculated and the surface location is updated as

\[
\dot{I}\mathbf{d} = \dot{S}\mathbf{d} \quad \text{on } \dot{I}\Gamma(t) \tag{4.36}
\]

The mesh displacement can be obtained at the interface between fluid and structure as

\[
\dot{I}\mathbf{d}^I = \dot{I}\mathbf{d} \quad \text{on } \dot{I}\Gamma(t) \tag{4.37}
\]

This mesh displacement \(\dot{I}\mathbf{d}^I\) is imposed at the interface between fluid and structure in CMD for the next time step.

### 4.6.3 Computational Mesh Dynamics

An elasticity-based meshing algorithm was employed to solve the re-meshing problem. The mesh was treated as a pseudo-elastostatic medium, and the same algorithm equation (4.35),
which was used to calculate the displacement of the structural body, was employed. In the algorithm, the mesh was modeled as a pseudo-elastic structure, the deformation of which is based on the boundary condition resulting from equation (4.37) of the structural problem. Such displacements are obtained from

\[ \hat{K}\hat{d} = 0 \quad (4.38) \]

where \( \hat{K} \) is the stiffness matrix, and \( \hat{d} \) is the mesh displacement vector defined by

\[ \hat{d} = \hat{R}(t) - \hat{R}(t_{ref}) \quad (4.39) \]

where \( \hat{R} \) is the mesh position vector, and \( t_{ref} \) is the reference time. The mesh deformation can be referenced to either the initial mesh location \( (t_{ref} = 0) \), in which case \( \hat{d} \) is the total mesh displacement, or to the last calculated mesh location, in which case \( \hat{d} \) is the incremental mesh displacement.

Equation (4.38) can be rewritten in the partitioned form as

\[
\begin{bmatrix}
{\hat{K}}_{FF} & {\hat{K}}_{FI} \\
{\hat{K}}_{IF} & {\hat{K}}_{II}
\end{bmatrix}
\begin{bmatrix}
\hat{d}_F \\
\hat{d}_I
\end{bmatrix}
= \begin{bmatrix}
0 \\
0
\end{bmatrix}
\quad (4.40)
\]

On the interface between fluid and structure, the mesh displacement given by equation (4.37) from CSD is imposed. Therefore,

\[
{\hat{K}}_{FF}' \hat{d}_F = -{\hat{K}}_{FI}' \hat{d}_I \quad (4.41)
\]

which shows that the fluid mesh motion is driven by the elastic body motion. This equation, together with the other boundary conditions on \( ^U\Gamma \) and \( ^F\Gamma \), was solved for the fluid mesh displacement \( ^F\hat{d} \).

The fluid mesh displacement is solved and the mesh geometry is updated. The mesh velocity field can be obtained as
The mesh velocity obtained is imposed in CFD (momentum equation) for the next time step.

4.7 Computational Approach

The general strategy is to perform the following computations at each time step:

1. **CFD:** Use the ALE form of the Navier-Stokes equations to solve for fluid velocity and pressure fields. Calculate the traction at the wetted surface that constitutes the boundary between the fluid and the structure.

2. **CSD:** Apply the surface tractions at the interface calculated in Step 1. Use the UL approach to solve for the displacement of the structure. Update the location of the interface.

3. **CMD:** Use the elastostatic model to determine mesh displacement, and calculate the mesh velocity field. Keep in mind that this step is required only for problems that involve structural displacements that are large enough to significantly affect the mesh in the flow domain.

The overall strategy that FIDAP employs in solving FSI problems can be summarized as follows (Figure 4-1 shows the sequential algorithm).

1. Initialize CFD, CSD, and CMD solvers.

2. Begin time loop.

3. CFD:
   a. Impose initial conditions and boundary conditions ($u_\text{\Gamma}$, $\sigma_\text{\Gamma}$, and $f_\text{\Gamma}$) and transfer-compatibility conditions on $\Gamma (t)$.

\[
\hat{u} = \frac{\hat{d}}{(t - t_{\text{ref}})}
\]  
(4.42)

The mesh velocity obtained is imposed in CFD (momentum equation) for the next time step.
b. Solve for the fluid velocity and pressure fields in $^F\Omega(t)$.

c. Calculate the traction vector at the interface, $^S\mathbf{r} = -\mathbf{r}'$.

4. CSD:

   a. Apply external loads and fluid traction, $^S\mathbf{r}$.
   
   b. Impose initial conditions and boundary conditions on $^S\Gamma(t)$.
   
   c. Integrate the equations of motion to yield the elastic body displacement, velocity, and acceleration vectors $(^sd, ^s\dot{d}, ^s\ddot{d})$.
   
   d. Calculate the displacement field at the wetted surface, $^1\mathbf{d}$, and update the surface location.

5. CMD:

   a. Choose the proper mesh dynamic parameters.
   
   b. Solve for the ALE mesh according to the pseudo-elastic body displacements at the wetted surface $(^1\mathbf{d})$.
   
   c. Update the mesh geometry.
   
   d. Calculate the mesh velocity field.

6. End time loop.
Figure 4-1. Sequential algorithm for the weakly coupled approach
CHAPTER 5
RESULTS and DISCUSSION

The results of computations, comparisons of the results to experimental data, and subsequent discussions are presented in this chapter. The first stage of this investigation was the comparison of the Newtonian viscosity model, which are used in the literature, with that of the non-Newtonian model, which was adopted for the current research according to the experimental data of Gijsen et al. [117]. Before comparing the two models explicitly, their validations were first performed with the available experimental data. Next, the FSI solver was examined and the numerical results were compared to the existing experimental data. Although validation for the FSI case represents, by itself, a challenging application, it addresses the accuracy of the FSI code used. In the later part of this chapter, a series of experimental comparisons with both steady and unsteady flow cases are compared. Moreover, specific point-to-point comparisons are performed with respect to the velocity and shear-stress profiles. Following these validations, various cases with respect to blood flow simulations in different geometries and different surgery cases have been simulated and documented.

5.1 Validations with Experiments

5.1.1 Newtonian Model Validation

In order to validate the Newtonian model, a fully developed steady flow (Reynolds number of 270) in a circular rigid tube was simulated and the results were compared to the experimental data provided by Gijsen et al. [117]. The simulations and data normalization were performed according to the specifications described in [117]. Moreover, the experimental procedure and setup are shown by Gijsen et al. [117]. Figure 5-1 shows the velocity profile
within the tube in comparison to the experimental data. It can be seen that the numerical results are in good agreement with the experimental data.

![Figure 5-1. Comparison of Newtonian model with experiments](image)

### 5.1.2 Non-Newtonian Model Validation

The implementation of the non-Newtonian Carreau-Yasuda model, which was used throughout this research, was validated for a fully developed steady flow in a circular rigid tube. The Carreau-Yasuda parameters, which were used, were adjusted by Gijsen et al. such that the non-Newtonian model matches experimental values of the blood viscosity. Simulations were performed, taking into account the same correlated Carreau-Yasuda parameters and the specifications described by Gijsen et al. [117]. Results were compared to the experimental data. Figure 5-2 shows the velocity profile within the tube in comparisons to the experimental data. It can be seen that the numerical results and the Carreau-Yasuda model agree fairly well with the
experimental data. Normalization of the data is based on the maximum velocity (0.14 m/s) of the Newtonian model shown in the previous section. Moreover, when the velocity profiles of the two models are compared explicitly, it shows that for the same shear rate non-Newtonian model behaved more viscous than the Newtonian model which leads to less velocity.

![Figure 5-2. Comparison of non-Newtonian Carreau-Yasuda model with experiments](image)

5.1.3 FSI Numerical Model Validation

In order to examine the accuracy of the FSI solver for simulating interacting flows, numerical solutions were compared with the available experimental data. Matsuzaki et al. [119] performed experiments on steady flow at moderate Reynolds number in a two-dimensional elastic channel. In the experiments, the fluid pressure acting on the internal wall of an elastic channel caused deflection of the upper wall. These displacements of the upper wall were
measured using optical sensors. The experimental set-up used by Matsuzaki et al. is shown in Figure 5-3.

Employing the same geometry and assuming linearly elastic material properties for the elastic channel, the deflections of the upper wall were compared for the same flow conditions as described by Matsuzaki et al. [119] (Reynolds number of 500). Figure 5-4 presents a comparison between the experimental data and the numerical results. The numerical results are in good agreement with the experimental data.

Figure 5-3. Experimental set-up used by Matsuzaki et al. to measure the deflections

Figure 5-4. Comparison of FSI numerical model with experiments
5.2 Steady and Pulsatile Flow in Carotid Artery Bifurcation

The purpose of this computational study was to investigate the behavior of blood flow in two different geometries of a bifurcated artery and to investigate the dependence of the Newtonian and non-Newtonian properties of blood under both steady and pulsatile flow conditions. The physiological relevance of this study was that it provided an insight to the flow conditions in the arteries, where the flow is pulsatile, and in the capillaries, where the flow is steady.

One aspect of this study was the comparison of the adopted non-Newtonian Carreau-Yasuda model with that of the Newtonian model used in the literature. The comparison was carried out using a Newtonian reference viscosity of 0.0029 Pa-s, resulting in mean Reynolds numbers of 135 and 270 for models 1 and 2, respectively.

5.2.1 Model Geometry

Two different models of arterial bifurcation were examined under both steady and pulsatile flow conditions. The geometry and respective meshes are shown in Figures 5-5 and 5-6, respectively. The basic geometrical data of Model 2 and the pulsatile flow conditions were derived from Ku et al. [60] and Bharadvaj et al. [53]. The geometry of Model 1 represents a capillary bifurcation and is the average of the models described in the literature. The two models differ in daughter branch diameters, as depicted schematically in Figure 5-5. Model 1 has a diameter of 4 mm, which is the same as the daughter branches, while model 2 has different diameters. The models are fully three-dimensional, and the cross-sections of the parent vessel and the branches are circular.

The computational unstructured meshes for the models, generated using GAMBIT [120] with tetrahedral and hexahedral cells, are shown in Figure 5-6. The number of computational
grid cells for Model 1 is 119,046 and for Model 2 is 361,367. The meshes are clustered near the arterial wall to produce a greater accuracy in calculating shear stress.

Figure 5-5. Schematic representation of bifurcation models

LOW:   Left Outer Wall              LIW:   Left Inner Wall
ROW:   Right Outer Wall              RIW:  Right Inner Wall
CCA:    Common Carotid Artery    ICA:   Internal Carotid Artery
ECA:    External Carotid Artery

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5.2.2 Material Characteristics and Boundary Conditions

For the steady-flow simulations, two types of boundary conditions were prescribed at the inlet of the mother vessel:

- Uniform velocity profile: \( u = U_m \) \hfill (5.1)
- Parabolic velocity profile: \( u = U_{\text{max}} \left(1 - \frac{r^2}{R^2}\right) \) \hfill (5.2)

For the unsteady or pulsatile flow simulations, the velocity at the inlet was prescribed with a sinusoidal waveform:

- Pulsatile flow velocity: \( u = U_m (1 + \sin(2\pi \cdot t)) \) \hfill (5.3)

Figure 5-6. Computational meshes used for models

Model 1     Model 2
At the outlet, zero gauge pressure was prescribed (as it is assumed that flow is discharged to zero gauge pressure). For each simulation case, different inlet velocity boundary conditions were imposed. A no-slip boundary condition was imposed at the walls of the arteries.

For the present simulations, blood was considered incompressible. The fluid properties of blood for simulations were specified as density of 1,410 kg/m$^3$ and viscosity of 0.0029 Pa.s for the Newtonian case. For the non-Newtonian case, a user-defined function was developed based on the Carreau-Yasuda model with parameters described in Chapter 4. Figure 5-7 shows the plot of Newtonian and non-Newtonian models used for the computations. At this phase of investigation, to simplify the problem the walls of the vessel were considered rigid. All solutions were verified to be grid-independent. Mesh independence was investigated for different grid densities for both models by comparing the velocity distributions at various locations. For the steady-flow simulations, the convergence criterion for residuals was set to $10^{-5}$.

![Figure 5-7. Newtonian and non-Newtonian models used](image-url)
5.2.3 Steady Flow

A. Model 1

For the steady inlet flow in Model 1, the results, shown in Figure 5-8, show the velocity profiles on an x-z plane cutting through the center (y=0) of the geometry. Since the flow profiles for both Newtonian and non-Newtonian flow appear similar with very minor changes, only one profile for the models is shown. The steady-flow computations were carried out both using uniform velocity and the parabolic velocity at the inlet of the parent artery. Since both cases resulted in the same flow behavior and flow characteristics a short distance downstream of the inlet, only parabolic inlet velocity results are shown. Similar observations also have been experimentally obtained by Walburn et al. [121], and Bharadwaj et al. [53]. In the daughter arteries, the velocity distribution varies with position.

Figure 5-8. Velocity vector profiles for Newtonian model on x-z plane cutting through center of geometry
In Figure 5-9, the comparison of the Newtonian and non-Newtonian velocity profiles is shown at the outlets B and C as in Figure 5-5. The comparison of the Newtonian and non-Newtonian flow in Figure 5-9 illustrates the same behavior as shown by Gijsen et al. [117]. In that study, Gijsen compared the Newtonian and non-Newtonian flow behavior with both experiments and simulation taking a different arterial geometry. In the present study, the comparison revealed that, for Model 1, the effect of Newtonian and non-Newtonian in the velocity profile is not very significant. However, differences in wall shear stress were observed.
Figure 5-10 shows pressure contours on an x-z plane cutting through the center of the geometry. In both cases, the pressure at the apex is higher at a location where the flow splits to the daughter branches. The magnitude of the pressure in the Newtonian case is less than that of the non-Newtonian case. Moreover, the pressure values in the non-Newtonian case are approximately twice that of the Newtonian case.

![Pressure contours](image)

Figure 5-10. Pressure contours on an x-z plane cutting through center of geometry

Figures 5-11 and 5-12 show a comparison of the wall shear stress along the walls of inner and outer arteries for both Newtonian and non-Newtonian cases. It is evident that introduction of the non-Newtonian nature of blood increased the shear stress at the walls.
The shear stress at upstream positions is approximately constant, indicating that flow remains parabolic and has not been disturbed by the downstream bifurcation up to about point \( Z = 0.01 \text{m} \), as shown in Figure 5-11. After location \( Z = 0.01 \text{m} \), the flow splits where the abrupt drop in shear stress takes place immediately downstream of the outer corner.

![Figure 5-11. Comparison of WSS along centerline of outer walls](image-url)
Shear stress distributions along the inner walls of the daughter artery are shown in Figure 5-12. On the inner wall of the daughter arteries, the maximum shear stress is concentrated near the apex region and subsequently drops sharply along the wall and remains nearly constant. Shear stress on the inner and outer walls is shown in Figure 5-13. It can be noted from this figure that the shear stress along both the inner and outer walls are coincident at downstream locations where the flow approaches that of full development.

Figure 5-12. Comparison of WSS along centerline of inner walls
Figure 5-13. Comparison of WSS along inner wall and outer wall

B. Model 2

Figures 5-14 to 5-18 show similar flow behavior as described for Model 1. However, the effect of the non-Newtonian nature of flow illustrates some differences in the velocity profile, as shown in Figure 5-15. The velocity profile for the Newtonian case is parabolic, while for the non-Newtonian case, it is flattened as expected for the shear thinning fluid. The flattening of the velocity profile in the non-Newtonian case for Model 1 is absent, which can be explained by considering the geometry. When compared to Model 1, Model 2 has more curvature in the daughter branches due to ICA and ECA. As a result, high – and low-velocity gradients are
developed at the divider and non-divider walls, making the flow phenomena more complex and, in turn, affecting the axial velocity.

Figure 5-14. Velocity vector profiles for Newtonian model on y-z plane cutting through center of geometry

Figures 5-16 and 5-17 show the same behavior as that of Model 1. Figure 5-18 represents shear stress along the inner walls of the artery. When compared to Figure 5-12, there are high and low peaks of shear stress, which is due to the increase/decrease in the area of the arterial geometry. Due to the arterial curvature of the geometry, gradients in the velocity increased or
decreased as a result of the beginning or ending of the curvature. These gradients increase or decrease the shear stress at the walls of the artery. Therefore, to accurately predict the location of the atherosclerosis, which corresponds to the low WSS regions in the arteries, an exact geometry of the human artery is necessary.

Figure 5-15. Comparison of velocity profiles at outlets of the artery for Newtonian and non-Newtonian fluids
Figure 5-16. Pressure contours on y-z plane cutting through center of geometry

Figure 5-17. Comparison of WSS along centerline of outer walls
5.2.4 Pulsatile Flow

Pulsatile flow simulations were carried out for a pulse cycle of 1 s with $\Delta t = 0.0001$ s. Figure 5-19 shows the sinusoidal waveform of the inlet velocity. The pulse has maximum and minimum velocities of 0.1388 m/s and 0 m/s respectively.

For the pulsatile flow, shown in Figure 5-19, the wall shear stresses in Model 1 at four different points in the pulsatile cycle (marked with arrows) are presented. Figures 5-20 to 5-23
show the comparison of wall shears every 0.25 s and for each side of the wall. Wall shear stresses varied considerably in each case. In the pulsatile case, the comparison of the shear stress is shown to visualize the effect of unsteady nature of the flow in arteries. Since the behavior of the flow is the same for Model 2, the comparison is shown only for Model 1 with Newtonian flow. This comparison illustrates how much variation in the wall shear could be obtained at each time level. From these figures, it is evident that shear stress varies significantly at each time level.

Figures 5-20 through 5-23 show similar behavior of the wall shear stress along the wall for each time level with an increase and decrease in magnitude.

From the above results, it can be observed that to predict the intensity of atherosclerosis in the arteries, an exact pulse cycle of the heart with which the blood pumps out of is necessary. As the pulse cycle for every human is different, so is the intensity of the atherosclerosis.

![Sinusoidal waveform of velocity profile at inlet](image)

**Figure 5-19.** Sinusoidal waveform of velocity profile at inlet
Figure 5-20. Comparison of WSS for Newtonian model along right outer walls

Figure 5-21. Comparison of WSS for Newtonian model along right inner walls
Figure 5-22. Comparison of WSS for Newtonian model along left outer walls

Figure 5-23. Comparison of WSS for Newtonian model along left inner walls
5.3 FSI Modeling of Blood Flow in Arterial Bifurcation

The objective of the current investigation was to perform FSI analysis to compute the WSS in an elastic bifurcated artery. The specific aim was not just to understand the sensitivity of the fluid stresses on the arterial wall but also to locate the susceptible locations by computing WSS to investigate the development of atherosclerosis. Simulations were in direct relevance to the physics of the arterial mechanics. Computations were carried out using FIDAP [111] software. Blood was modeled as an incompressible fluid, and the artery model was based on linear elasticity.

5.3.1 Model Geometry

The governing equations were solved along a two-dimensional bifurcated deformable artery. The model consisted of two separate domains, i.e., the structural domain, consisting of the arterial walls, and the fluid domain, corresponding to the interior of the artery. The inlet and outlet of the artery measured 4 mm in diameter, and the thickness of the arterial wall was 0.2 mm. The total length of the bifurcation was 44 mm where the daughter and parent arteries are 22 mm each. Four-node quad elements were used for both solid and fluid domains. The fluid portion of the mesh consisted of 1,610 elements and 1,742 nodes, while the solid portion consisted of 888 elements and 1,125 nodes. In total, the mesh consisted of 2,498 elements and 2,867 nodes. Figure 5-24 (a) and (b) illustrates the geometry and the computational mesh. The deformable artery region shown in this figure deforms as a result of stresses imposed by the flow.

5.3.2 Material Characteristics and Boundary Conditions

For the simulations, Young’s modulus and Poisson’s ratio were set to $10^7$ dynes/cm$^2$ and 0.3, respectively [124].
The density of the blood and arterial wall was specified as 1.410 g/cm³. Blood viscosity was specified from the non-Newtonian model of Carreau-Yasuda through a user-defined function.

![Diagram of blood flow](image)

**Figure 5-24.** (a) Original geometry (b) Original mesh

At the inlet, velocity corresponding to a Reynolds number of 135 at zero shear rate was prescribed, whereas at the outlet, a zero gauge pressure was prescribed. Moreover, the velocity at the inlet was constrained in the axial direction. No-slip boundary conditions were imposed at the inner walls of the arteries (wall that is in contact with blood). The boundary conditions for the displacements were prescribed as follows: nodes at the inlet and outlet of the artery (solid and fluid sections) were constrained for displacements in the axial and transverse directions. At the
remaining nodes on the external boundaries, zero displacement was prescribed in the direction normal to the boundary, and the nodes were allowed to move freely in the tangential direction. Displacement-free boundary conditions were applied at all other nodes. In the present simulations, linear isotropic elastic material properties were assumed, and for the structure simulations, nonlinear, large deformation schemes were employed. The iterative schemes used proceeded until the fluid, solid, and mesh variables all reached a convergence criterion for the residuals were set equal to $10^{-4}$. The total CPU time for one simulation was about nine hours, using a Pentium dual core processor of 3.2GHz with 3.0 GB Ram.

5.3.3 WSS and Deformation

Figures 5-25 (a) and (b) show the deformed geometry and mesh, respectively. The deformation of the artery is due to the stresses imposed by the blood on the arterial wall. Moreover, the deformation of the artery is large at the end of the parent artery because of the high negative gauge pressure. From the Figure 5-25 (c), which shows the pressure contours (gauge pressure), it is evident that the pressure at the vicinity of the apex is higher where the flow splits to the daughter branches. Moreover, the artery is compressed at locations where the gauge pressure is negative because of the pressure difference which is lower inside the artery and higher outside (atmospheric pressure, zero gauge) the artery. Furthermore, the pressure contours in FSI simulations followed the same qualitative behavior described previously in section 5.2.

Figures 5-26 and 5-27 represent the variation of the WSS along the walls of the artery. In Figure 5-26, the left and right WSSs are practically identical until the end of the parent artery. Subsequently, the change in WSS can be attributed to the different slopes of the daughter artery wall. Moreover, the peak in WSS is due to the increase in velocity, which is due to the decrease in the diameter at the end of the parent artery. Thereafter, the shear stress drops over a narrow
section of the artery where the flow splits and proceeds smoothly along the daughter arteries. However, in Figure 5-27, WSS distributions along the left and right inner walls of the bifurcated region are not identical. This is due to the different slopes in the arterial configurations leading to different wall velocities and stresses. Due to the arterial curvature of the geometry, the gradients in the velocity increased or decreased as a result of beginning or ending of the curvature. These gradients will increase or decrease the shear stress at the walls of the artery.

![Figure 5-25. (a) Deformed geometry (b) Deformed mesh (c) Pressure contour](image)

The magnitude of WSSG in a uniform flow region is by definition zero. If the WSSG value is other than zero, it denotes non-uniform flow behavior. Figures 5-28 and 5-29 represent the variation of the WSSG along walls of the artery. There exists a local high-axial WSSG as well as a local low-axial WSSG on the artery wall. These locations are usually associated with
local deposit of particles, which results in the stiffening of the arterial wall leading to the development of atherosclerosis.

Figure 5-26. WSS along inner walls of artery

Figure 5-27. WSS along inner walls of bifurcated artery

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Figure 5-28. WSSG along inner walls of the artery

Figure 5-29. WSSG along inner walls of the bifurcated artery
5.4 FSI for Pulsatile Flow through Elastic Arteries

In the present study, the internal flow of the blood in an elastic artery was investigated. The effort included analyzing the theory and behavior of the soft tissue walls of an artery, investigating the complex relationship between arterial wall deformations and flow behavior, and analyzing the effect of arterial blood pressure. Moreover, explicit comparisons between rigid-wall and deformable-wall simulations were made in order to investigate the effect of flexible artery simulations on the local WSS patterns. The comparison between simulations of rigid arteries and flexible arteries are necessary in terms of atherosclerosis. Therefore, to achieve specific conclusions between the simulations performed using rigid walls and flexible walls, two different models of arteries were investigated. Computations were carried out by using FIDAP [111] software. The blood was modeled as an incompressible fluid. The artery model was based on linear elasticity.

5.4.1 Model Geometry

Two different models of arteries were examined under pulsatile flow conditions. Model 1 and Model 2 had diameters of 2 cm and the thickness of the arterial walls is 0.1 cm. The total length of the artery models was 8 cm. Model 1 was uniform throughout its length, whereas Model 2 was sinusoidally curved at the mid-length of the artery. The geometry and respective meshes are shown in Figures 5-30 through 5-33. Both models consisted of fluid and solid domains, as shown in Figures 5-30 and 5-31. For the mesh generation, four-node quad elements were used for both solid and fluid domains. The fluid portion of the mesh for Model 1 and Model 2 consisted approximately of 8,000 elements and 8,900 nodes, while the solid portion consisted of 800 elements and 832 nodes. In total, the mesh consisted of approximately 8,800 elements and 9,732 nodes. The meshes were clustered near the interface between the structural and fluid
regions in order to have a smooth transition between the regions and to resolve the boundary layer. The meshes were coarser elsewhere to optimize computational efficiency.

5.4.2 Material Characteristics and Boundary Conditions

The artery wall was assumed to be linearly elastic with a Young’s modulus of 10 MPa. The assumed Young’s modulus corresponded to the mean (intima, media, and adventitia layers) Young’s modulus of the aorta. The Poisson’s ratio was set to 0.3. Blood was assumed incompressible with a density of 1050 kg/m$^3$. Arterial wall density was also specified as 1050 kg/m$^3$. Viscosity of blood was based on the Carreau-Yasuda non-Newtonian model.

**Fluid Boundary Conditions:** At the aortic inlet, a time dependent oscillatory pressure waveform (pulsatile), shown in equation (5.4), with a realistic frequency was used

$$ P(t) = P_0 + A \cdot \sin(\pi \cdot t) $$

(5.4)

Sinusoidal pressure pulse in equation (5.4), which is assumed for simplicity, corresponds to 30 beats/min. The values of $P_0$ and $A$ are both 100 Pa which corresponds to 7.5 mm of mercury. The above values are assumed in order to reduce the convergence difficulties while performing numerical simulations. At the aortic outlet, a zero gauge pressure condition was prescribed. The pressure difference between inlet and outlet drove the fluid (blood) into motion through the aorta causing the wall to deform. Moreover, at the inlet, the velocity was constrained in the axial direction. No-slip boundary conditions were imposed at the inner walls of the arteries (wall which is in contact with blood).

**Structural Boundary Conditions:** The nodes corresponding to the arterial wall at inlet and outlet were constrained in all directions. Displacement-free boundary conditions were applied at all other nodes.
In these simulations, a backward Euler time integration scheme with fixed time step, linear isotropic elastic material, and nonlinear and large deformation schemes were employed. In all simulations, the time step was specified as 0.001s with 2,000 total time steps per cycle. The time marching procedure was carried out until convergence was achieved (criteria equal to $10^{-4}$). In all cases, steady oscillations occurred after a transient regime. The cycle after the transient regime was used as the final periodic solution to post-process the results.

5.4.3 Results

Flow in the arteries with two different geometries was investigated. Both FSI and rigid wall simulations were conducted to compare the effects of a deformable wall. The flow conditions, schematics, and meshes used are shown in Figures 5-30 to 5-33. In all cases the Carreau-Yasuda non-Newtonian model was used. For the rigid wall cases, simulations were carried out with all properties being the same as that of FSI simulations. Figure 5-34 shows the centerline inlet velocity convergence diagram of the curved geometry FSI simulation. It indicates nearly steady-state oscillation after approximately seven cycles. Similar convergence was observed for the straight artery FSI simulation. The converged cycle was used as the final periodic solution and is presented in this investigation.

![Figure 5-30. Schematics of straight artery (Model 1) (not to scale)](image)

![Figure 5-31. Geometry of curved artery (Model 2) (not to scale)](image)
Figure 5-32. Mesh for Model 1

Figure 5-33. Mesh for Model 2

Figure 5-34. Centerline inlet velocity convergence history
A. Waveforms

The velocity waveforms at the inlet for one complete cycle corresponding to the inlet pressure extracted from the results for both geometries are shown in Figures 5-35 and 5-36. Velocity followed the sinusoidal trend similar to pressure pulse for both cases. However, the peaks of the acceleration and deceleration phases were not identical for both geometries. This is due to geometry differences between the two models of the arteries. Since, the flow in both models represented a subsonic flow, disturbances in the domain propagated at both upstream and downstream locations. Therefore, to incorporate the geometry changes, the mass flow rate was adjusted between the two geometries to maintain the same pressure gradient throughout the domain.

Figure 5-35. Velocity and pressure waveforms of straight deformable artery at inlet for one complete cycle
Figure 5-36. Velocity and pressure waveforms of curved deformable artery at inlet for one complete cycle

B. Displacements

Figures 5-37 and 5-38 show wall displacements in the vertical direction at four different time levels in the pulsatile cycle for both straight and curved arteries, respectively. Displacement profile looks similar to Figure 5-4 in which the experiments were performed by Matsuzaki et al. according to Figure 5-3. From the Figures 5-37 and 5-38, it is evident that displacement of the straight artery wall was larger than the curved artery wall. Moreover, maximum displacements of the upper wall were 1.1 and 0.98 mm, respectively. The difference in displacements was 10 percent. This 10 percent reduction in wall displacement for the curved artery suggests that the wall curvature caused the artery to be stiffer, thus leading to less deformation when compared to the straight wall. It follows that the flexural rigidity (given by Young’s modulus x moment of inertia of the cross-section) of the curved artery is larger when compared to the straight artery.
causing it to be stiffer. Furthermore, for each time level, the peak in the displacement was observed at the mid-section of the artery. Also, in the complete cycle, the largest displacement was seen at 0.5 s, which corresponds to highest inlet pressure (as well as inlet velocity). At time level 1.5 s, which corresponds to zero pressure, small or negligible displacement of the arterial wall were observed.

![Figure 5-37. Displacements of upper wall of straight deformable artery](image)

C. Velocity Profiles

Velocity profiles at the outlet section of the straight and curved arteries are shown in Figures 5-39 and 5-40, respectively.
Profiles near the centerline, where the shear rate was small, were flattened, which illustrates the shear thinning behavior of the non-Newtonian fluid. The flatness in the profiles was more intense in the straight artery case, which may be due to the highly disturbed flow due to more deformation of the arterial wall. For both cases, the magnitude of the velocity profiles followed the pressure pulse, i.e., the magnitude of the profile was smaller at 1.5 s time level, which corresponds to the minimum pressure in the waveform, and maximum at 0.5 s which corresponds to the maximum pressure in the waveform.

Figure 5-38. Displacements of upper wall of curved deformable artery
Moreover, the velocity magnitude at each time level was larger in the curved artery when compared to the straight artery. Thus, it required larger mass flow (blood flow) at each time step to maintain the same pressure gradient as that of the straight artery. Furthermore, due to larger deflection (increased area) in the straight artery, the pressure at mid-section was larger than in the curved artery, thus leading to smaller velocity.

![Velocity Profile](image)

**Figure 5-39.** Axial velocity profiles at artery outlet for straight deformable artery
Figure 5-40. Axial velocity profiles at artery outlet for curved deformable artery

D. Contours

Figures 5-41 to 5-43 and 5-44 to 5-46 show the pressure, velocity, and displacement contours for both straight and curved arteries, respectively, at a representative time level of 0.5 s. Pressure was maximum at the mid-section of the arteries, velocity was maximum at the centerline position, and displacements were also maximum at the mid-sections of the artery. A comparison of the three contours for both arterial geometries illustrates that, with a slight change in the geometry of the artery, substantial variations with respect to flow and structural variables were observed. The largest pressure at the mid-section of the arteries was a direct consequence of low velocity and large displacements, and vice versa.
Figure 5-41. Pressure contours for straight deformable artery

Figure 5-42. Velocity contours for straight deformable artery

Figure 5-43. Displacement contours for straight deformable artery
E. Wall Shear Stress

Figures 5-47 to 5-50 illustrates the comparison of wall shear stress along the upper walls of the arteries for both straight and curved cases. In addition, comparisons with the rigid wall cases (simulations performed with the same flow conditions) are shown. A comparison of the
rigid wall case and the deformable wall case is necessary with respect to the atherosclerosis (hardening of artery/loss of elasticity). The wall shear stress profiles in this section could indicate the differences between healthy and diseased arteries. Figure 5-47, which corresponds to the 0.5 s time level, shows a uniform distribution of the shear stress for a straight rigid artery. The uniformity in the shear stress illustrates that there were no flow disturbances along the artery (simple uniform cylinder flow). However, for the same geometry, if the artery wall is changed from rigid to deformable, the shear stress pattern would no longer be uniform, indicating a complex, disturbed flow behavior.

Figure 5-47. Wall shear stress along upper wall of artery at 0.5 s
Moreover, comparing the shear stress magnitudes, it is evident that the rigid wall flow case (diseased artery) had large shear stress values when compared to the deformable wall flow case (healthy artery). This increase in the magnitude of shear stress indicates that the artery lost its elastic property and potentially could be diagnosed with atherosclerosis disease. The same explanation can be extended to the curved rigid artery and curved deformable artery. Furthermore, the calculated difference between shear stresses for rigid walls and that of deformable walls was in the range of 30 to 40 percent at the maximum shear stress location.

![Graph](image)

Figure 5-48. Wall shear stress along upper wall of artery at 1.0 s
The upstream and downstream locations of shear stresses along the artery walls were approximately coincident for the straight deformable artery and the curved deformable artery. Likewise, the same behavior was observed for rigid cases. However, a comparison of the mid-sections of straight and curved arteries showed large fluctuations in shear stress distributions. These high and low peaks of shear stress can be attributed to the increase/decrease in the area of the arterial geometry. Due to the arterial curvature of the geometry, the gradients in the velocity increased or decreased as a result of the beginning or ending of the curvature. These gradients will increase or decrease the shear stress at the walls of the artery.

![Graph showing wall shear stress along upper wall of artery at 1.5 s](image)

Figure 5-49. Wall shear stress along upper wall of artery at 1.5 s
Figures 5-48 and 5-50 follow the same pattern as that of 5-47. In Figure 5-49, the shear stress diagrams for the curved rigid and curved deformable are coincident. That is because there was no displacement of the artery at time level of 1.5 s, as shown in Figure 5-38. However, for the straight rigid and straight deformable cases, the shear stress diagrams were not coincident, which indicates that the displacement of the artery in the deformable case was not zero; instead, there were some relatively small displacements. Thus, in the pulsatile case, the comparison of the wall shear stress revealed that there was a considerable amount of variation in each case considered.

![Graph](image_url)

Figure 5-50. Wall shear stress along upper wall of artery at 2.0 s
5.5 Pulsatile Flow in End-to-Side Anastomosis Model

In this study, the numerical simulation of an end-to-side anastomosis model was performed. First, results for the steady and pulsatile Newtonian fluid flow in a two-dimensional (2-D) rigid-walled end-to-side anastomosis model are compared with the experimental measurements of Steinman et al. in terms of velocity profiles and WSS variations. Subsequently, the simulations were extended to three-dimensions to gain a qualitative understanding of the flow and WSS behavior. The same end-to-side geometry with a mid-femoral waveform as measured by Doppler ultrasonography [99] was used along with the Carreau-Yasuda model to account for the non-Newtonian property of blood.

5.5.1 Model Geometry

Steinman et al. [99], developed an in vitro model of an end-to-side anastomosis and performed photochromic dye tracer measurements of velocity fields under steady and pulsatile flow conditions. The model consisted of a two-dimensional flow channel constructed from ultraviolet-transparent plexiglas. The end-to-side anastomosis model had a channel height of 5 mm, a width of 50 mm, an entrance length of 190 mm, and a graft-vessel angle of 45 degrees. Extensive velocity measurements were performed, and the wall shear stresses were estimated from the slope of the near-wall velocity profiles. Thus, this study provides a well-defined set of experimental data with which numerical results may be compared. The model used in the experimental study of Steinman et al., was purposely designed to have a mathematically described geometry to allow direct translation to a numerical model. The mathematical description of the geometry was used to generate the computational model used in this study. Details of the model geometry, the experimental setup and the measurement technique are found in Steinman et al. [99]. The model geometry and the nomenclature used in this study are shown
in Figure 5-51. GAMBIT [120] was used to develop geometrical models of two – and three-dimensional end-to-side anastomoses, which are shown in Figures 5-52 and 5-53, respectively. For the mesh generation, four-node quad elements were used to mesh the 2-D model, whereas tetrahedral elements were used for the 3-D model. The optimal number of elements was determined from the mesh-independence study.

Figure 5-51. Geometry of end-to-side anastomosis

Figure 5-52. Grid system of 2-D end-to-side anastomosis. Inset shows enlarged view of mesh at the junction of anastomosis
The 2-D meshed model consisted of 10,780 elements and 11,124 nodes, while the 3-D model consisted of 151,378 elements and 62,792 nodes. Meshes were clustered near the arterial wall to produce a greater accuracy in calculating the shear stress.

Figure 5-53. Perspective view of the 3-D end-to-side anastomosis

5.5.2 Material Characteristics and Boundary Conditions

A. 2-D Anastomosis

For steady flow, a Reynolds number of 130 with kinematic and dynamic viscosities of 1.89 cStokes and 1.43 cPoise, respectively, were used at the graft inlet.
For unsteady flow, a flow waveform was specified at the graft inlet. Figure 5-54 illustrates the flow waveform measured at the graft inlet in the in vitro experiment. The data points in Figure 5-54 correspond to the in vitro experiments, which are spline-fitted in order to use them as the inlet condition for the numerical analysis.

No-slip conditions were imposed on all walls, and zero gauge pressure and zero axial velocity gradients were specified at the distal outlet of the artery.

![Figure 5-54. Flow waveform at the graft inlet](image)

In the present simulations, rigid wall conditions were assumed since synthetic grafts and diseased arteries were expected to be relatively stiff (muscular arteries diameter < 5 mm). The assumption of no flow through the proximal end of the host artery was also imposed. This
corresponds to the implantation of the graft in a severely stenosed artery. The lengths of the graft and the artery were made sufficiently long so as to reduce the velocity oscillations in the solutions, and to accurately manage the larger Reynolds numbers and negative flow rates encountered when using the mid-femoral waveform.

For the unsteady cases, a total of 1,000 uniform time steps per cardiac cycle were used. The time-marching procedure was carried out until steady oscillatory motion was achieved. To eliminate the start effect of pulsatile flows, the simulation was carried out over three full cycles. The typical CPU time per cycle was about seven hours for 2-D models and 82 hours for 3-D models, using a Pentium dual core processor of 3.2GHz with 3.0 GB Ram.

**B. 3-D Anastomosis**

The mid-femoral flow waveform shape, as measured by Doppler ultrasonography [99], which is shown in Figure 5-55, was specified at the graft inlet through a user defined function. The physiological flow waveform used, had a peak systolic flow Reynolds number of 1,143 (peak prograde), which is approximately eight times the mean Reynolds number, followed by zero flow and a reverse flow of Reynolds number 495 (peak retrograde), approximately three times the mean Reynolds number. Subsequently, the waveform led to a post-diastolic peak followed by zero flow at the end of the cardiac cycle.

No-slip conditions were imposed on all walls, and zero-gauge pressure and zero-axial velocity gradients were specified at the distal outlet of the artery.

Blood with a density of 1050 kg/m$^3$ was used, while the viscosity was specified from the non-Newtonian model of Carreau-Yasuda through a user-defined function.
5.5.3 Results

A. 2-D End-to-Side Anastomosis Simulations

Velocity profiles under steady-flow conditions obtained from numerical analysis were compared with existing experimental data. The axial component of velocity in the 2-D anastomosis model was compared to the experimental measurements at three different axial locations of 0.3, 2.3, and 3.9 (from the axis shown in Figure 5-51) respectively, as shown in Figure 5-56. The axial locations were non-dimensionalized with respect to the channel height. An excellent agreement between the numerical and experimental results was observed at all locations.
Figure 5-57 shows the axial components of velocity under unsteady flow conditions at the same locations identified previously. The comparison with the experiments was carried out at nine different time levels, as shown in these figures. A good agreement between the numerical and experimental data was observed at all locations and at different time levels with relatively small discrepancies. Discrepancies were found primarily along the lower wall of the host artery and were more prominent at the 0.3 location. Quantitative error analysis shows that the relative error ($|\frac{(u_{\text{exp}} - u_{\text{num}})}{u_{\text{exp}}}|$) between the results was approximately 1 to 7 percent for the entire flow cycle.

Figure 5-58 shows a comparison between the experimental and numerical non-dimensionalized WSS variations at three non-dimensionalized locations of the host artery throughout the flow cycle. WSSs were made dimensionless with respect to $\rho U^2$. 
Figure 5-57. Comparison of axial velocity component at three representative axial locations (2-D, unsteady case): (a) 0.3, (b) 2.3, (c) 3.9
Good to fair agreement between the results was observed, with more discrepancies at the lower wall of the host artery. Qualitatively, a general agreement between the numerical and experimental results could be observed throughout the flow cycle for both lower and upper walls. Large discrepancies appeared at the lower wall of the anastomosis when compared to the upper wall.

Figure 5-58. Comparison of wall shear stress distributions at three representative axial locations: (a) 0.3, (b) 2.3, (c) 3.9 (continued)
Figure 5-58. Comparison of wall shear stress distributions at three representative axial locations:
(a) 0.3, (b) 2.3, (c) 3.9
The upper wall had relative WSS errors $((\tau_{\text{exp}} - \tau_{\text{num}})/ \tau_{\text{exp}})$ in the range of 1 to 12 percent, whereas for the lower wall, errors ranged from 1 to 35 percent throughout the flow cycle. The largest relative error appeared at the location 0.3 (Figure 5-58a) for both lower and upper walls, whereas locations 2.3 and 3.9 had the second and third highest relative error, respectively. This is because at location 0.3, the flow was more disturbed when compared to other locations. However, this error could partly be reduced by increasing the mesh density at the artery-graft intersection. Furthermore, the large relative errors at the lower wall were the consequence of corresponding velocity errors (Figure 5-57a), described previously. The high percentage of error may be attributed to the sensitivity of shear stress toward the variation in velocity profiles. Nevertheless, the overall comparison of shapes of the numerical WSS curves followed the experimental curves well. These results and error levels were comparable to those found in the literature [99]. Also, the accuracy of the experimental results was not provided by Steinman et al. [99].

B. 3-D End-to-Side Anastomosis Simulations

Flow field

The velocity vectors and streamline patterns on the mid-plane for three different time levels corresponding to peak prograde (forward), zero, and peak retrograde (backward) flows are shown in Figures 5-59 and 5-60, respectively.

At peak prograde flow ($t = 0.15$ s) (Fig. 5-59a), fluid accelerated through the graft and entered the recipient artery. There was significant skewing of the velocity toward the floor of the artery-graft junction due to the entrance of the flow from the graft. The skewing effect of the velocity vectors disappeared once the flow progressed downstream of the artery, where the flow became fully developed. Separation and a very small recirculating region were observed at the
toe section of the anastomosis. A large, slowly paced vortex in the host artery was evident just proximal to the graft inlet.

Figure 5-59. Velocity vectors at mid-section of 3-D anastomosis for (a) peak prograde flow, (b) zero flow, and (c) peak retrograde flow (continued)
Figure 5-59. Velocity vectors at mid-section of 3-D anastomosis for (a) peak prograde flow, (b) zero flow, and (c) peak retrograde flow

At zero flow \( t = 0.27 \) s (Fig. 5-59b), the large vortex observed in Figure 5-59 (a), just proximal to the graft, continued to grow in size and speed. Furthermore, the vortex at the toe section grew in size, reaching more than half of the artery diameter. The flow vectors near the walls show that flow was toward the graft, whereas in the body of the graft, flow was toward the artery.

At peak retrograde flow \( t = 0.35 \) s (Fig. 5-59c), the fluid flow accelerated through the artery, entered the graft, and left through the graft. The vortex at the downstream location of the anastomosis in zero flow disappeared, resulting in a simple flow pattern. However, the vortex at the proximal end, which was observed in both prograde and zero flow cases, still existed in this flow. Furthermore, a second vortex (Fig. 5-60c), which was not observed in peak prograde and zero flow, was evident at the hood section of the artery.
Figure 5-60. Streamline patterns at mid-section of 3-D anastomosis for (a) peak prograde flow, (b) zero flow, and (c) peak retrograde flow (continued)
Wall Shear Stress

Wall shear stress distribution at the distal anastomosis is of importance with regard to intimal thickening. Figures 5-61 and 5-62 represent the variation of the WSS over the upper wall (hood section) and lower wall (toe and heel section) of the artery for three different time levels, which correspond to peak prograde (forward), zero, and peak retrograde (backward) flows. The differences in the velocity field at the anastomosis due to three different flows gave rise to distinct WSS patterns.

In Figure 5-61, the region of large increased axial WSS observed in the peak prograde flow of the cycle at graft-artery junction was due to impingement of bypass flow from the graft to the hood of the artery. From that point, shear stress dropped sharply over a narrow section of the artery where the flow split and proceeded smoothly along the artery. The negative shear
stress at the upstream location of the anastomosis indicated the flow reversal and was related to the recirculation in that region. The reduction of flow disturbances downstream of the artery suggest the smooth flow behavior, which is confirmed by the constant value of wall shear stress. Figure 5-62 illustrates the elevated shear stresses at the toe and heel portion of the anastomosis. The negative shear stress at the toe portion suggests a recirculation region. WSS patterns for the zero and retrograde flows followed the same pattern as peak prograde flow with less intensity in magnitude. WSSs were identical at the heel location for prograde and retrograde flows.

![Figure 5-61. Wall shear stress along upper wall of artery for three time levels](image)

Figure 5-61. Wall shear stress along upper wall of artery for three time levels
Figure 5-62. Wall shear stress along lower wall of artery for three time levels

**Wall Shear Stress Gradient**

The magnitude of WSSG in a uniform flow region is by definition zero. If the WSSG value is other than zero, it denotes a non-uniform flow behavior. In order to illustrate a quantitative comparison between the three flow levels, the magnitudes of WSSG along the upper wall of the artery are shown in Figure 5-63. This figure shows WSSG values only in the anastomosis region, since beyond that region the values are zero denoting a uniform flow. A local high axial WSSG as well as a local low axial WSSG existed on the artery wall for all cases. The magnitude of WSSG in the anastomosis region was greatly increased in the prograde flow. This indicates existence of a large variation in the shear stresses along the artery, which is further confirmed from Figure 5-63, in which the variation appears from a high negative value to a high
positive value. However, WSSG magnitude in the other two cases remains almost identical following the general trend of prograde flow with lower magnitude.

Figure 5-63. Wall shear stress gradient along upper wall of artery for three time levels

The pulsatile simulations of 3-D anastomosis clearly showed the elevated and the negative WSSs at the toe, heel, and hood regions of the artery-graft anastomosis. A similar conclusion could be attributed by considering the distribution of WSSG along the artery wall. The same behavior was observed for the other two flow times in the cycle with a slight decrease in the magnitude of the stresses. The region where the WSS is negative corresponds to the recirculation region within the artery. Regions of reverse flow or recirculating flows are usually associated with local deposit of particles, which results in a blockage of the artery, leading to IH [122]. Findings are consistent with the works of several researchers [99, 102, 103, and 104]. The
elevated and negative WSSs, which led to the development of IH at the artery-graft junction, had to be reduced or eliminated by considering design variations in the anastomosis geometry. In summary, the hemodynamics within the artery-graft anastomosis may significantly affect the development of IH. Potential artery-graft anastomosis design improvements that reduce the amount of WSS may have to be performed in order to increase the clinical success of vascular bypass grafts.

5.6 Effect of Proximal Artery Flow at the Distal End-to-Side Anastomosis Model

The purpose of this computational study was to investigate the effect of proximal artery flow condition as well as to gain a qualitative understanding of the flow and wall shear stress behavior in an end-to-side anastomosis model. Two particular stenosed artery cases were considered to identify the effect of proximal artery flow: Case (1) 80 percent stenosed artery (partial stenosis), and Case (2) 100 percent stenosed artery (full stenosis). The results may offer insight about the proximal flow conditions of these stenosed artery cases in order to have a least-disturbed flow behavior at the anastomosis in vascular bypass graft procedures. A physiological relevant flow waveform measured by Doppler ultrasonography [99] was utilized as the graft and artery inlet conditions.

5.6.1 Model Geometry

The schematics of a human bypass graft of a stenosed femoral artery are illustrated in Figure 2-3. Here, femoral artery is completely blocked due to stenosis. To restore blood flow through that artery, the bypass procedure was carried out by attaching both ends of the graft to the sides of the artery. This procedure is called end-to-side anastomosis. To help clinicians understand the outcome of these surgeries, numerical experiments in an end-to-side anastomosis model were performed. For the simulations, a mathematical description of the geometry
proposed by Steinman et al. [99] was used. Moreover, only the right half of the bypass geometry was used to carry out the simulations because of the intense computational time required for the full model. Furthermore, from the clinical point of view, blood entering from the graft to the artery was more of a concern than blood flowing from the artery to the graft. The idealized end-to-side anastomosis geometry (Figure 5-51) consisted of rigid, 5 mm arteries intersecting at an angle of 45 degrees. The model geometry and the nomenclature used in this study are shown in Figure 5-51. GAMBIT [120] was used to develop the CFD model of three-dimensional end-to-side anastomosis, which is shown in Figure 5-53. For the mesh generation, tetrahedral elements were used to mesh the 3-D model. The meshed model consisted of 151,378 elements and 62,792 nodes. Meshes were clustered near the arterial wall to produce greater accuracy in calculating the shear stress.

5.6.2 Material Characteristics and Boundary Conditions

The mid-femoral flow waveform shape, as measured by Doppler ultrasonography [99], which is shown in Figure 5-55, was specified at the graft inlet for Case 2, whereas for Case 1 flow waveform, which is 80 percent of Figure 5-55, was specified. For Case 2, there was no flow from the proximal artery (upstream location), whereas for Case 1, flow waveform was specified at the proximal section of the artery, which is shown in Figure 5-64. The flow waveform in Figure 5-64 represents 20 percent of the flow through the proximal artery, which corresponds to 80 percent stenosis (blockage).

No-slip conditions were imposed on all walls, and zero-gauge pressure and zero-axial velocity gradients were specified at the distal outlet of the artery.

Blood with a density of 1,050 kg/m$^3$ was used, while the viscosity was specified from the non-Newtonian model of Carreau-Yasuda through a user-defined function.
Figure 5-64. Physiological waveform at proximal end of the artery for Case 1

In the present simulations, rigid wall conditions were assumed, since synthetic grafts and diseased arteries are expected to be relatively stiff for arteries (muscular arteries diameter < 5 mm). The lengths of the graft and the artery were made sufficiently long so as to reduce the velocity oscillations in the solutions, and to accurately manage the larger Reynolds numbers and negative flow rates encountered when using the mid-femoral waveform.

For the flow cases, a total of 1,000 uniform time steps per cardiac cycle were used. The time-marching procedure was carried out until steady oscillatory motion was achieved. To eliminate the start effect of pulsatile flows, the simulation was carried out over three full cycles. The typical CPU time per cycle was about 83 hours using a Pentium dual core processor of 3.2GHz with 3.0 GB Ram.

Post-processing of the results included flow field visualization (streamlines and velocity vectors), and the calculation of two parameters, WSS and WSSG, which are usually associated
with IH formation. They were plotted at peak prograde flow (forward flow), zero flow, and peak retrograde flow (reverse flow) for both cases.

5.6.3 Results

A. Flow Field

Streamline patterns and velocity vectors at the mid-plane for two cases (80 percent stenosis and 100 percent stenosis) at different time levels corresponding to peak prograde, zero, and peak retrograde flows are shown in Figures 5-65 to 5-70. A number of features were common for both cases considered. However, there were also distinct differences in the flow field locally for the cases considered based on the selected time levels.

At peak prograde flow (t = 0.15 s), in the 80 percent stenosis case, fluid accelerated from both the graft and the proximal section of the artery and flowed toward the outlet section of the artery (Figure 5-65). However, for the 100 percent stenosis case, fluid accelerated only through the graft and entered the recipient artery. In both the cases, at anastomosis, the velocity vectors toward the lower wall of the artery were significantly skewed due to flow entering from the graft. The skewing effect of the velocity vectors disappeared once the flow progressed downstream of the artery, where the flow became fully developed. Separation and a recirculating region were observed at the toe section of the anastomosis. Apart from the velocity magnitudes, the only difference observed in the case of 100 percent stenosis artery was a large, slowly paced vortex in the host artery just proximal to the anastomosis.

At zero flow level (t = 0.27 s), for both cases, the velocity vectors in the graft region and the artery region downstream of anastomosis were the same (Figure 5-66). Furthermore, a large vortex was observed at the anastomosis in both the cases. However, the vortex in the 100 percent stenosis case was larger and extended towards the proximal artery location with more than half
the size of an artery diameter. Additionally, a second recirculating region was observed in the proximal artery in the 80 percent stenosed artery, along the lower wall.

At peak retrograde flow (t = 0.35 s), the fluid flow accelerated through the distal section of the artery and left through both graft and proximal sections of the artery in the 80 percent stenosis case, whereas for the 100 percent stenosis case, fluid left only through the graft section (Figure 5-67). In both cases, velocity vectors at the distal artery location and in the graft region appeared to be similar. Moreover, the vortex at the hood section was also observed in both cases. However, the vortex at the proximal end of the artery was only evident in the case of the 100 percent stenosed artery.

Figure 5-65. Streamline patterns at mid-section of anastomosis for peak prograde flow

Figure 5-66. Streamline patterns at mid-section of anastomosis for zero flow
Figure 5-67. Streamline patterns at mid-section of anastomosis for peak retrograde flow

Figure 5-68. Velocity vectors at mid-section of anastomosis for peak prograde flow

Figure 5-69. Velocity vectors at mid-section of anastomosis for zero flow
Figure 5-70. Velocity vectors at mid-section of anastomosis for peak retrograde flow

B. Wall Shear Stress

In most physiological situations, blood flows in relatively straight sections of blood vessels. However, when a graft is inserted into an artery, the velocity field becomes disturbed at the anastomosis leading to complicated WSS patterns. Differences in the velocity field at the anastomosis due to the flow in the proximal artery segment give rise to significant differences in resulting WSS patterns. Figures 5-71 and 5-72 illustrate the comparison of the axial WSS patterns at the lower and upper walls of the artery for both 80 and 100 percent stenosed arteries. Comparisons were made at the specific time levels of the pulsatile waveform, thus addressing all possible flow changes within the waveform considered.

In the 80 percent stenosis case, at peak prograde flow, there was an increase in axial WSS (high WSS region) on the upper wall of the artery near the hood region. This was due to converging flows from the graft and proximal artery sections at anastomosis. However, in the 100 percent stenosis case, both an increase (high WSS region) and decrease (low WSS region) in axial WSS patterns existed. The high WSS region extending from distal anastomosis location to hood region was due to the converging flow from graft to artery, whereas the low WSS region, which extends from the proximal anastomosis location to hood region, was due to the
recirculating zone (flow reversal) at the anastomosis. For the lower artery wall, nearly identical WSS patterns were found for both cases considered.

In both cases, at zero flow level, a small region of both high – and low-axial WSSs extending for distal anastomosis location to hood region and hood region to proximal anastomosis location on the upper wall of the artery were observed. Quantitatively, the 100 percent stenosis case had relatively higher magnitudes of WSSs when compared to the 80 percent stenosis case. However, almost identical WSS patterns were observed on the lower wall of the artery for both cases.

At peak retrograde flow level, no elevated WSS patterns were observed at the distal anastomosis region of the upper wall, whereas a low WSS region at the proximal anastomosis region of the upper wall was observed for both cases. On the lower arterial wall, low WSS and high WSS patterns were observed at the heel location for 80 and 100 percent stenosis cases, respectively. However, at the toe region, low WSS were observed for both cases. Moreover, unlike other two flow levels, higher magnitudes of WSS were observed in the 80 percent stenosis case rather than 100 percent stenosis case.

When compared explicitly for the entire waveform, the 100 percent stenosis case had peak low WSS region at peak prograde and zero flow levels, whereas at peak retrograde flow level they were on 80 % stenosis case. Moreover, for the 100 percent stenosis case, a large slowly-paced vortex is observed at all time levels proximal to the anastomosis, whereas for the 80 percent stenosis case recirculating region were only observed during zero and peak retrograde time levels. Regions where the WSS is negative (low) corresponds to the recirculation region within the artery/graft. Regions of reverse flow or recirculating flows are usually associated with local deposit of particles, which results in a blockage of the artery, leading to IH. According to
the comparisons made, it can be inferred that the 100 percent stenosis case was more prone to develop local deposits leading to IH.

Figure 5-71. Wall shear stress along upper wall of artery for three time levels

Figure 5-72. Wall shear stress along lower wall of artery for three time levels
C. Wall Shear Stress Gradient

The magnitude of WSSG in a uniform flow region is by definition zero. In order to compare the flow behavior for both cases considered, at the representative time levels defined previously, the magnitudes of WSSG along the upper wall of the artery are shown in Figure 5-73. Note that the region shown for WSSG corresponds to the anastomosis location only, since the values of WSSG returned to zero beyond this location. In both 80 and 100 percent stenosis cases, the WSSG increased near the hood section of the artery at peak prograde and retrograde flows. However, for the peak prograde, the magnitude of WSSG was large in the 100 percent stenosis case, whereas for peak retrograde flow, it was in the 80 percent stenosis case. Just proximal to the anastomosis, there existed a small peak of WSSG for retrograde flow level in both cases, whereas in the prograde flow, this peak was not observed in the 80 percent stenosis case. Additionally, for peak prograde flow level, WSSG peaks were observed on the distal side of the anastomosis in both cases. Similar peaks were observed for the zero flow level in both cases. However, peaks in the 80 percent stenosis case slightly moved to the right of the anastomosis, compared to the 100 percent stenosis case. This shows that a disturbed flow pattern existed at hood, heel, and toe locations of the graft-artery anastomosis for the waveform considered.

It has been shown that local flow patterns, and hence axial distribution of WSS and WSSG, are dependent on the proximal artery inlet condition. Moreover, it has been shown that hemodynamic parameters, which affect directly the development of arterial diseases, are related to the flow condition at the proximal artery. Therefore, the proximal inlet condition must be considered when investigating end-to-side anastomosis. Furthermore, the percentage of flow rate at proximal artery other than 20 percent may or may not induce the formation of IH, and the graft patency at the anastomosis.
Figure 5-73. Wall shear stress gradient along upper wall of artery for three time levels

5.7 Pulsatile Flow in End-to-End Anastomosis Model

The goal of the current study was to numerically investigate the effect of compliance mismatch while matching the geometry criterion, i.e., the artery and graft having the same nominal radius. Figure 5-74 shows an end-to-end anastomosis where the host artery has been replaced by a geometrically matching synthetic graft [123]. In the current investigation, numerical simulations were performed using FSI. A comparison study was performed for the same artery-graft geometry and different mechanical properties. Results presented here would be useful in selecting a proper graft material for the arterial replacement procedures. Blood was modeled as an incompressible and non-Newtonian fluid, and the artery-graft model was based on isotropic linear elasticity.

5.7.1 Model Geometry

An axisymmetrical FEM model of a cylindrical artery-graft anastomosis designed based on data of Stewart et al. [124] was used to solve pulsatile flow in a deformable artery using FSI.
The model incorporated end-to-end anastomoses of an artery graft at both junctions, as shown in Figure 5-74.

Figure 5-74. Artery replacement with matching graft (printed with permission [123]) and schematic of an end-to-end anastomosis

The geometry of the model was similar to Stewart et al. [124], which was composed of three main segments: (1) artery section, 2.4 cm long; (2) synthetic graft section, 3.2 cm long; and (3) the artery section, 2.4 cm long. All three segments consisted of both fluid and solid domains. The nominal unstressed inner radius and wall thickness were equal to 1 and 0.1 cm, respectively.
The artery-graft region, shown in the figure, deformed as a result of stresses imposed by the flow. In turn, the deformation affected the flow pattern. Therefore, an iterative solution procedure was used, which includes the effects of flow on the deformable boundary, and vice versa.

For the mesh generation, four-node quad elements were used for both solid and fluid domains. The optimal number of elements was determined from the mesh-independence study. The fluid portion of the mesh consisted of 80,000 elements and 80,901 nodes, while the solid portion consisted of 8,000 elements and 8,833 nodes. In total, the mesh consisted of 88,000 elements and 89,734 nodes. The meshes were clustered near the interface between the structural and fluid regions so as to have a smooth transition between the regions and resolve the boundary layer. The meshes were coarser elsewhere to optimize computational efficiency.

5.7.2 Material Characteristics and Boundary Conditions

Compliance \( C \) is the mechanical property of a tube/artery that expresses a change of diameter with respect to change of pressure:

\[
C = \frac{\Delta D}{\Delta P} \cdot \frac{1}{D} \quad (5.5)
\]

where \( D \) is the diameter of the artery, and \( \Delta D/\Delta P \) is the change of diameter with respect to the change of pressure. Compliance is not a fundamental property but is dependent on the mechanical properties of the vessel, the vessel radius, and the vessel thickness. For a fixed deformed state, compliance depends on the current diameter/thickness ratio and on the incremental elastic modulus of the material of the artery. Incorrect use of compliant materials for replacement surgeries may lead to intimal hyperplasia [125-127]. To investigate the biocompatibility of the artery-graft replacement, three simulations were performed: Case (1) compliance match, i.e., Young’s modulus of the artery and synthetic graft was set to \( 10^6 \) dyne/cm\(^2\), which is in the physiological range stated by Stewart et al. [124, 128]; Case (2)
compliance mismatch, where the synthetic graft’s Young’s modulus is reduced by a factor of ten, while keeping the artery’s Young’s modulus the same as Case 1; and Case (3) compliance mismatch, where the synthetic graft’s Young’s modulus is increased by a factor of ten, while keeping the artery’s Young’s modulus the same as Case 1.

For all simulations, Poisson’s ratio was set to 0.3. The density of the blood was set to 1.050 g/cm$^3$. Blood viscosity was specified from the non-Newtonian model of Carreau-Yasuda through a user-defined function.

**Fluid Boundary Conditions:** An oscillatory pressure waveform (pulsatile) with a more realistic frequency, as shown in equation (5.6) was assigned at the inlet:

$$P(t) = P_0 + A \cdot \sin(\pi \cdot t)$$

(5.6)

where the time period of the incoming wave was two seconds. The values of $P_0$ and $A$ were both 100 Pa in agreement with the physiological values. Zero gauge pressure was prescribed at the outlet. The pressure difference between the inlet and outlet drove the fluid (blood) into motion through the aorta causing the wall to deform. Moreover, at the inlet, the velocity was constrained in the axial direction. The radial component of the velocity vector was set to zero along the axis of symmetry. No-slip boundary conditions were imposed at the inner walls of the arteries (wall that is in contact with blood).

**Structural Boundary Conditions:** Boundary conditions for the displacements were prescribed as follows: For the nodes attached to the solid portion of the artery-graft intersection, both displacements in the axial and radial directions were prescribed as zero, since the artery and graft were tied together. At the inlet, outlet, and symmetry boundaries, zero displacement was prescribed in the direction normal to the boundary, whereas the nodes were allowed to move
freely in the tangential direction. Displacement-free boundary conditions were applied at all other nodes.

In the present simulations, a backward Euler time integration scheme with a fixed time step, linear, isotropic elastic material properties were assumed, and for the structure simulations, nonlinear, large deformation schemes were employed. In all simulations, the time step was specified as 0.01s, with 200 total time steps per cycle. The time-marching procedure was carried out until the fluid, solid and mesh variables all reached a convergence criteria set to $10^{-4}$. Once convergence was achieved, time step was incremented, and the procedure continued until the conclusion of the simulation time, which was specified as 40 seconds. In all cases, steady oscillations occurred after a transient regime. The cycle after the transient regime was used as the final periodic solution to post-process the results. The total CPU time for one simulation per cycle, using a Pentium dual core processor of 3.2GHz with 3.0 GB Ram was about 37 hours.

5.7.3 Results

A. Velocity Wave Forms and Profile

Centerline axial velocity waveforms at the graft mid-sections are compared in Figure 5-75. In spite of differences in compliance, the velocities for Cases 1 and 3 were similar. For Case 2, the velocity waveform followed the same characteristics, with an increase in magnitude. This difference in the velocity magnitude was due to the increase in diameter/deformation of the graft section, which is shown in the next section. Large deformation resulted in a large recirculation region, as shown in Figure 5-76. Streamlines for Case 1 and Case 3 remained parallel throughout the domain, whereas a recirculation zone occurred for case 2. The flow in the recirculation zone did not affect the overall mass flow rate. Therefore, Case 2 was similar to the flow in a rigid tube, which is shown in Figure 5-75. A decrease in velocity magnitude in Cases 1 and 3 was due to the
deformation in the graft and artery regions. Since the flow remained attached, an increase in the cross-sectional area led to a decreased axial velocity, which is consistent with the Poiseuille flow.

![Graph showing centerline axial velocity waveforms at graft mid-section](image)

Figure 5-75. Centerline axial velocity waveforms at graft mid-section

Axial velocity profiles (not shown for brevity), corresponding to Cases 1 and 3 were practically identical, whereas for Case 2, an increase in the axial velocity was observed, which is due to the formation of a recirculation zone from large radial deformations. However, for Cases 1 and 3, radial displacements were not sufficiently large to generate a recirculation zone. Velocity profiles were observed to be flattened near the axis of symmetry in all cases, as
expected from the shear thinning fluid property represented by the Carreau-Yasuda non-Newtonian model.

Figure 5-76. Streamline patterns at $t = 1$ s (colored by axial velocity)

**B. Structural Displacements**

Displacements of the inner wall due to pressure forces as a function of axial position at time level of 0.5 s are shown in Figure 5-77. For all cases, local maximum displacement was observed at the middle of each section. Comparing Case 1 and Case 3, displacements of the artery sections were the same at each time level (only one time level shown) because they have the same material properties. However, the peak of displacement for the graft section was larger.
in Case 1. As mentioned previously, the Young’s modulus for the graft section in Case 3 was ten times larger than in Case 1, which caused it to be stiffer, thus less deformable. Comparing Case 1 and Case 2 in Figure 5-77, again displacements of the artery sections were the same for each time cycle. However, the peak of displacement in the graft section in Case 2 was larger than in Case 1, which followed the same logic as above. The graft section Young’s modulus was ten times smaller than the artery section, thus making the graft more elastic, leading to larger displacement of the inner vessel wall.

![Figure 5-77. Displacement of the artery-graft inner wall](image)
C. Wall Shear Stress

Wall shear stress as a function of axial position along the inner wall of the vessel for all three cases at a time level of 0.5 s are shown in Figure 5-78. WSS for the model with the compliant graft was fairly uniform throughout the domain. Similar observations have been previously described [122, 128]. For Case 2, a positive peak in WSS was observed at the artery-graft junction, followed by a negative peak in the graft region. Moreover, in Case 2, except at the end points, an almost constant WSS was observed at the upstream and downstream locations of the artery. The possible reason for the negative peak in Case 2 compared to Case 1 was due to the change in material properties of the graft. However, for Case 3, an almost constant WSS profile was observed. Moreover, the uniformity in WSS in the artery regions of Case 3 was almost identical to that of Case 1. Furthermore, WSS was observed to drop below zero at upstream and downstream locations of anastomosis in the graft region for Case 2. Regions where the WSS is negative corresponds to the recirculation region within the graft. Regions of reverse flow or recirculating flows are usually associated with local deposit of particles, which results in a blockage of the artery, leading to intimal hyperplasia [99, 122]. WSS profiles for all cases in the flow cycle followed the same general trend as discussed above with the reduced levels. WSS for the graft region in Case 2 had a larger peak than that of Case 3, because the graft region for Case 2 was less stiff, inducing more deformations than did the Case 3 graft, which was 100 times stiffer.
Wall shear stress patterns at the upstream and downstream locations of artery-graft intersections as a function of time for all cases are shown in Figure 5-79. It was observed that the profile for Case 1 at both the locations was practically the same. For Cases 2 and 3, the general trend of the WSS pattern was the same, with fluctuations in the magnitude. Case 3 WSS profiles compared well with that of Case 1 at the upstream location, while a slight discrepancy in the magnitude was observed at the downstream location. A similar phenomenon was observed for Case 2 at both locations, but this time, the magnitude of discrepancy was larger. Moreover, from Figure 5-79, it also can be observed that the shear stress at both locations was following the same pattern as that of the axial velocity profile, which is shown in Figure 5-75 for all time levels.
Figure 5-79. WSS pattern at artery-graft intersection for complete cycle
This research was concerned with the study of fluid-structure interaction applied to biomechanical problems related to blood flow in arteries to determine the effect of the artery wall model on hemodynamic parameters. That is, how do simulations performed using a rigid-wall assumption differ from those that utilized a flexible-wall simulations. Moreover, the effects of the Newtonian nature of blood and the non-Newtonian nature of blood were compared for both steady and pulsatile flow cases. In addition, various arterial geometry simulations were performed to provide some indication of the occurrence of atherosclerosis describing hemodynamic parameters. The scope of the study included an investigation of the outcome of various cases of bypass surgery with natural and synthetic grafts in order to determine some indication of the occurrence of intimal hyperplasia.

The aforementioned investigations resulted in the following conclusions:

- The comparison of numerical results with experimental data for steady and pulsatile flows showed excellent agreement in steady flow and minor discrepancies in pulsatile flows. For both steady and pulsatile flows the velocity profile comparisons yielded good agreement whereas some discrepancies were found in the wall shear stress distributions for the pulsatile flows.

- Comparisons of the velocity profiles for the Newtonian and the Carreau-Yasuda non-Newtonian models applied to identical cases showed negligible differences. However, there were significant differences in the level of maximum arterial shear stresses associated with the two fluid models of the blood.
- Apart from the Newtonian and non-Newtonian description of blood, it was found that the geometry of arteries played a significant role in locating the extremes of wall shear stress regions, which are susceptible to the development of atherosclerosis in the arteries. Moreover, it was observed that a small change in geometry had a significant effect on the resulting blood flow and structural response of the artery.

- When compared to steady flow, pulsatile flow results showed a significant difference in the localization of the shear stress at different time levels in the flow.

- The wall shear stresses for rigid arteries were substantially greater than those associated with deformable arteries. This suggests that, as an artery becomes more rigid (less compliant), the maximum shear stress in the wall increases leading to atherosclerosis.

- The maximum shear stresses as computed with the rigid wall assumption were 30 to 40 percent greater than those using the deformable wall assumption. Therefore, the deformation of the arterial wall must be accommodated in blood flow simulations.

- The pulsatile simulations of end-to-side anastomosis clearly showed elevated and negative wall shear stresses at the toe, heel, and hood regions of the artery-graft anastomosis. The region where the wall shear stress was negative corresponded to the recirculation region within the artery. Regions of reverse flow or recirculating flows are usually associated with local deposit of particles, which results in a blockage of the artery, leading to intimal hyperplasia. Therefore, potential artery-graft anastomosis design improvements that reduce the amount of wall shear stress may have to be performed in order to increase the clinical success of vascular bypass grafts.
Moreover, in end-to-side anastomosis, the case with 100 percent stenosis had the maximum elevated low wall shear stress region at peak prograde and zero flow levels, whereas at peak retrograde flow level, the maximum elevated low wall shear stress region was on the 80 percent stenosis case. Therefore, it can be concluded that the 100 percent stenosis case was more prone to develop local deposits leading to intimal hyperplasia than the 80 percent stenosed case.

In the end-to-end anastomosis simulations, wall shear stress patterns for the less-compliant case have fewer variations than that of compliant-match simulations. However, the variations were more substantial for the more-compliant case simulations, for which the expansion of the artery radius was apparent, leading to a lowered wall shear stress in the entire graft region. Thus, intimal hyperplasia is more likely to occur when the graft is less stiff than the artery.
REFERENCES


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[123] Biomaterial & Tissue Engineering Centre (BTEC), University Department of Surgery, University College London, UK & UCL Biomedica.


