CFD ANALYSIS OF BLOOD FLOW THROUGH STENOSED CAROTID ARTERY USING FLUID-STRUCTURE INTERACTION (FSI)

REPORT UG 2021-2025

BY

Saud Ishtiaque Ahmed	00000392954
Muhammad Rafay Khalid	00000388344
Shaheer Farrukh Abbasi	00000384907
Muhammad Ameer Hamza	00000368358

Project Supervisor:
Dr Khurram Kamal – Professor, NUST-PNEC

Pakistan Navy Engineering College, Karachi

National University of Sciences and Technology, Islamabad, Pakistan

2024

Contents

ABSTRACT

One of the most leading causes of death and disability worldwide is atherosclerosis, a cardiovascular disease. This cardiovascular disease is mainly associated with the buildup of plaque in the arteries carrying oxygenated blood to different organs and tissues of the body. This plaque buildup ultimately leads to blockage or narrowing of the arteries that alter the blood flow patterns causing ischemic events such as stenosis. The carotid artery, mainly responsible for supplying blood to the human brain, is a critical site for the stenosis that makes its hemodynamic analysis more essential for understanding the disease. This Project focuses on the CFD analysis of the blood flow through a stenosed carotid artery with Fluid-structure interaction (FSI), using advanced numerical simulation. The study aims to model the blood flow behavior and its interaction with walls, keeping in view the mechanical deformation of the blood vessel. By simulating the various degrees of stenosis, the study will explore the impact on velocity profiles, pressure distribution, and Wall Shear Stress WSS, with addition to how these factors contribute to development and progression of atherosclerosis. The FSI allows for a more realistic simulation by accounting the interaction between blood flow and arterial walls, which deform under the influence of pulsatile flow and pressure forces. This interaction provides a deeper understanding of the biomechanical environment within the stenosed arteries, especially in regions prone to plaque development. Combining the study of CFD and FSI, this study offers a comprehensive analysis of the hemodynamic changes caused due to stenosis in the carotid artery. The investigation will not only help identify the high-risk areas for the atherosclerosis plaque buildup but help improve the understanding of biomechanical stressors that contribute to plaque progression. This knowledge could be helpful in enhancing the diagnostic methods for detecting stenosis earlier and making strategies to prevent or slow down the formation of atherosclerosis plaques in the carotid artery.

ACKNOWLEDGEMENT:

We would like to extend our gratitude to the faculty who assisted, guided and mentored us throughout the process. We are very thankful they took the time and effort to impart their knowledge and guide us in every step of our project.

We are especially grateful to Dr Khurram Kamal (Project Supervisor) for the continuous recommendations and guidelines he presented to us to ensure our project continues as smoothly as possible. Our project would not have been possible without his extensive knowledge.

We would also like to thank Dr Tahir Ratlamwala for his constructive criticism and guidance which greatly assisted in steering us in the right direction.

We are very grateful to all the staff and faculty for accommodating us at every step of the way.

CHAPTER 1

INTRODUCTION

1.1 Problem Statement

Cardiovascular diseases (CVDs) continue to pose a formidable global health challenge, accounting for a significant proportion of deaths annually. Among these, carotid artery stenosis stands out as a leading contributor to ischemic strokes, a condition that can result in severe and often permanent neurological impairment or death. Carotid artery stenosis is characterized by the progressive narrowing of the carotid arteries due to the buildup of atherosclerotic plaque, a complex process influenced by various factors such as lipid accumulation, inflammation, and endothelial dysfunction. This narrowing restricts blood flow to the brain, leading to reduced oxygen delivery and increasing the risk of ischemic events.

The pathophysiological consequences of carotid artery stenosis are particularly alarming, as they create a vulnerable environment for thrombus formation. When disturbed blood flow occurs in a stenosed artery, it can lead to the formation of blood clots that may dislodge and travel to the brain, resulting in embolic strokes. Alternatively, the reduced blood flow alone can precipitate ischemic strokes due to insufficient perfusion of brain tissue. Despite the serious implications of this condition, current diagnostic methods, including Doppler ultrasound, computed tomography (CT) angiography, and magnetic resonance angiography (MRA), have notable limitations. While these imaging modalities can effectively identify the presence and degree of arterial narrowing, they often fail to provide a comprehensive understanding of the dynamic mechanical behavior of the arterial walls under varying physiological conditions.

Specifically, traditional imaging techniques are limited in their ability to predict how hemodynamic changes, such as velocity, pressure, and flow separation affect the structural integrity of the arterial walls. This lack of insight is particularly critical, as the mechanical stresses exerted on the arterial walls play a pivotal role in the stability of atherosclerotic plaques. Areas of high wall stress are often where plaque rupture is most likely to occur, leading to serious complications such as strokes. Moreover, current diagnostic practices

do not adequately assess the interactions between blood flow and the mechanical properties of the arterial walls, thereby missing critical indicators that could aid in predicting disease progression and stroke risk.

Consequently, there is a substantial knowledge gap in our understanding of the fluid-structure interactions (FSI) present in stenosed carotid arteries. Addressing this gap is essential for developing improved diagnostic tools and therapeutic strategies. Without a more robust understanding of how altered hemodynamics contribute to the progression of carotid artery stenosis and the risk of ischemic strokes, healthcare providers are left with limited means to predict and mitigate these potentially devastating events. This underscores the urgent need for advanced research methodologies that can provide a deeper understanding of the complex interplay between blood flow dynamics and arterial mechanics in the context of carotid artery disease.

1.2 Solution

To deal with the demanding situations posed with the aid of carotid artery stenosis (CAS) and its link to ischemic strokes, a multifaceted approach is crucial. The key to stopping strokes lies in comprehensive information on the complicated fluid-structure interactions (FSI) occurring inside stenosed arteries. By leveraging superior computational strategies like Computational Fluid Dynamics (CFD) and Fluid-Structure Interaction (FSI) models, we are able to simulate the dynamic interplay between blood flow and arterial wall mechanics. These models offer important insights into how disturbed hemodynamics, together with changes in speed, strain, and drift separation, contribute to the danger of thrombus formation and plaque rupture. This approach can perceive areas of high wall stress, that are maximum vulnerable to plaque instability, thus addressing an important gap left by conventional diagnostic equipment inclusive of Doppler ultrasound, CT angiography, and MRA.

Integrating those advanced simulations with actual imaging facts can decorate diagnostic precision. This permits the scientific network to expect not the handiest diploma of arterial narrowing but also the mechanical behavior of the artery underneath physiological conditions. With more special know-how of the way mechanical forces affect arterial integrity, clinicians can highly expect the development of sickness and tailor interventions hence. Moreover, CFD simulations may want to provide customized insights into an

affected person's chance of embolic stroke, enhancing the accuracy of preventive strategies.

Refining diagnostic methods by using combining computational models with modern-day imaging technology will bridge the expertise gap in carotid artery disorder management. This will facilitate the development of extra correct predictive tools for assessing stroke threat and formulating focused therapeutic interventions, together with surgical processes like carotid endarterectomy or stenting. By addressing both the fluid dynamics and structural mechanics of stenosed arteries, this approach aims to mitigate the devastating results of ischemic strokes and enhance patient results

1.3 Aim

This study seeks to conduct a thorough Computational Fluid Dynamics analysis, integrating fluid-structure interaction, to examine the hemodynamic effects of stenosis on blood flow within the carotid artery. The primary focus will be on:

- Design a model for a stenosed carotid artery with varying angles of stenosis.
- Examining the various impacts of varying stenosis severity (0-70%) on blood flow patterns, velocity profiles, and pressure distributions within the carotid artery.
- Evaluating the effect of stenosis on arterial wall deformation, wall shear stress, and other mechanical properties.
- Identifying the areas prone more to plaque formation due to different hemodynamic parameters such as Wall Shear Stress,
- Identifying the critical hemodynamic factors contributing to stenosis progression and plaque formation that led to eventual diseases.
- Investigate the interaction between the blood flow and the arterial wall through FSI coupling, examining how the arterial wall's mechanical properties influence flow dynamics.

CHAPTER 2

LITERATURE REVIEW

2.1 Background Study

2.1.1 Overview of Blood Flow in Arteries

The Blood Flow phenomena through the human artery is a complex phenomenon that is a process governed by both fluid dynamic of the blood and the biological factors of blood altogether. The human Circulatory system is the main role-playing character that is responsible for the transport of oxygenated blood to different parts, organs and tissue of the human body. Maintaining a continuous flow as well as pulsatile flow under pressure is a major role that is played by the Human arteries. Human Blood as a fluid flowing through the arteries is influenced by several factors, that include geometry of the vessel, its elasticity, resistance to flow, and many more. As the fluid (Blood) moves in the arteries, it has to face resistance based on the varying diameter and shape of the blood vessel, it also faces resistance due to any blockage or abnormality possibly present in, such as stenosis. This resistance offered by different things is counterbalanced by the blood pressure to ensure the adequate supply of the oxygenated blood to all the parts of the human body. In short the understanding of the mechanics of the blood flow through arteries is very essential for diagnose and treatment of cardiovascular diseases [1], [2].

2.1.2 Importance of Studying Stenosed Arteries

One of the most common cardiovascular diseases that is caused by the buildup of plaque in the human arteries that carry oxygenated blood form the heart to different organs and tissues is Stenosis. It can cause significant health risks by disturbing or restricting blood flow, that lead to conditions such as ischemia, stroke, or myocardial infarction. Studying Stenosed arteries is crucial because it helps researchers and medical clinicians in understanding how this narrowing and thickening of arteries affects blood flow and how it contributes to disease progression. As the plaque progression starts alteration in

the flow pattern, increase in the Wall shear stress, turbulence in the flow occurs[3]. These alterations in the blood flow parameters help the further development of plaque and potential rupturing of atherosclerotic plaques, which possibly lead to life-threatening consequences for a human being. Modelling of stenosed arteries can help predict the impact of these changes on the vascular system and develop methods to mitigate these effects.

2.1.3 Fluid-Structure Interaction (FSI) in Cardiovascular Systems

Fluid-Structure Interaction (FSI) is an advanced computational method that allows the investigator to simulate both fluid (blood) and solid (arterial walls) behavior. In Human cardiovascular systems, the Fluid (Blood) exerts pressure on the arterial walls and in response arterial walls deform due to the pressure, as blood flow and the arterial walls are coupled in Fluid Structure Interaction (FSI); this interaction makes an impact on the overall Hemodynamics of the blood. This arterial wall deformation specifically in the areas of stenosed can highly influence the blood flow patterns, which in contrast affect the wall shear stress and plaque rupture risk in the arteries. These FSI models help observe more realistic simulation of the cardiovascular behavior between the fluid (blood) and the structure (arteries) by capturing the interaction among them. It also help getting the insights that cannot be achieved by solely studying the fluid or structural behavior[4].

2.2 A Historical Perspective

2.2.1 Early Methods of Blood Flow Simulation

Simulating blood flow heavily relied on analytical methods and physical experiments before the advent of modern computational and simulation tools. Very simplified models of blood flow through arteries were used to estimate the hemodynamic parameters of the blood for both healthy and unhealthy (stenosed) arteries. Being valuable at their time, these early and idealized models do lack in capturing the complexities of real or patient specific arteries and the effect of cardiovascular disease like stenosis. Some approaches but too limited such as in-vitro setup using artificial arteries were also utilized but were unable to accurately mimic the biological

conditions. Their widespread use was limited due to the difficulties and cost effectiveness of these physical models[5].

2.2.2 Development of Computational Fluid Dynamics (CFD) in Biomedical Applications

The study of blood flow in human arteries got revolutionized by the introduction of Computational Fluid Dynamics (CFD) as it provided the means to simulate fluid behavior in complex geometries of the arteries. As in medical imaging technologies got advanced, such as MRI and CT scans, the reconstruction of the patient specific arterial geometries became possible making helpful to simulate blood flow through them and obtaining accurate hemodynamics of the blood flow[6]. This helped biomedical applications achieve a big milestone, as CFD techniques allowed more accurate, precise and detailed analysis of the blood flow dynamics, specifically in the diseased arteries. CFD's ability to model complex flow patterns, such as turbulent flow in stenosed arteries, provided valuable insights into the hemodynamic factors that contribute to disease progression.

2.2.3 Introduction of FSI in Cardiovascular Simulations

Initially the arterial walls were treated as rigid structures while CFD advancement in understanding the blood flow. With the introduction to Fluid Structure Interaction (FSI), it allowed interaction among blood flow and the deformable arterial walls to be modeled. This advancement helped to provide more accurate and complete analysis of cardiovascular function by keeping in view different mechanical properties of arterial walls, such as elasticity, and their response to blood pressure. Study of pulsatile nature of blood flow and dynamic behavior of human arteries during different cardiac cycles became easy as this advancement enabled more realistic and complex physiological conditions[4].

2.2.4 Importance of Modeling Stenosed Arteries for Disease Prevention

For the development of effective preventions and interventions and understanding the progression of cardiovascular diseases such as stenosis, more accurate modelling of the stenosed or diseased arteries is vital. Blood flow as well as the changes in the mechanical stresses on the arterial walls due to the stenosis or simply blockage of the arteries. This can lead to further plaque formation or rupture, resulting in serious

conditions like heart attacks or strokes. By using CFD and FSI to simulate stenosed arteries, researchers can predict which areas of the artery are most vulnerable to these changes, allowing for better prevention and treatment strategies. Such models also provide valuable insights for designing medical devices like stents and for planning surgical interventions.

2.3 Hemodynamics and Blood Flow Properties

2.3.1 Blood as a Newtonian vs Non-Newtonian Fluid

Depending on the flow conditions, Human Blood can exhibit both Newtonian and Non-Newtonian behavior. Blood behaves like Newtonian fluid under high shear rates, such as those found in large arteries, the viscosity of blood remains constant. However, Blood behaves like non-Newtonian in smaller vessels or arteries with slow flow, where its viscosity varies with the shear rate. This change in blood viscosity is primarily due to the presence of cells like red blood cells, which tend to aggregate at lower shear rates. For simulating realistic flow patterns accurately modelling blood's rheological properties is essential, especially in diseased (stenosed) arteries where Non-Newtonian effects become more pronounced [7].Figure [8]

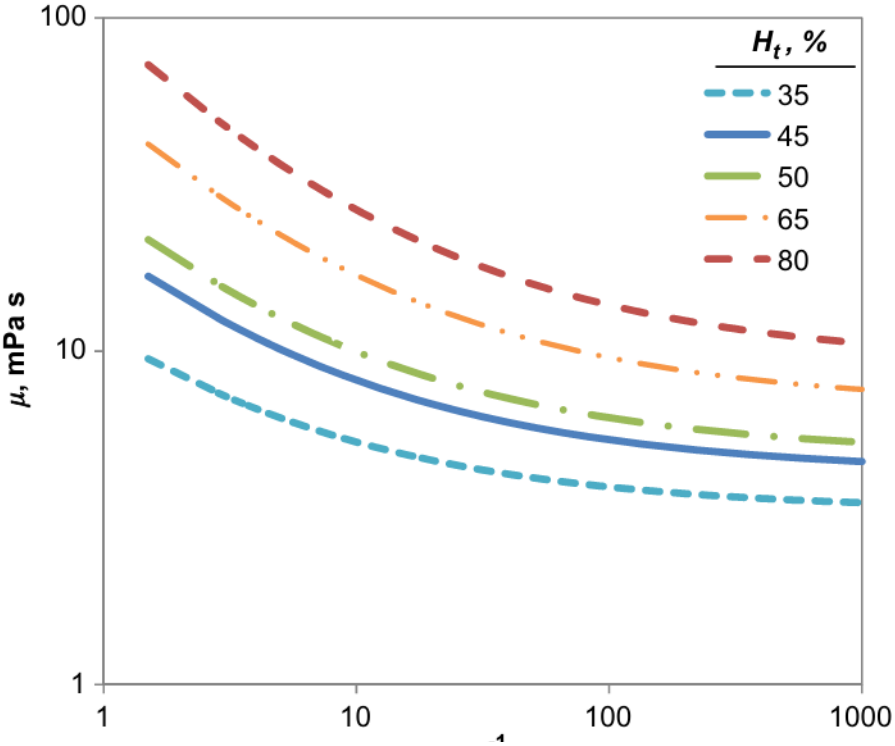

Fig 1. Blood viscosity for various hematocrit values

2.3.2 Role of Wall Shear Stress (WSS) on Arterial Health

The key hemodynamic parameter that influences endothelial function and arterial health is Wall shear stress WSS. In healthy arteries or blood vessel wall shear stress is generally high, as it promotes endothelial cell alignment and inhibits plaque formation. Opposite to it, low wall shear stress, often found in regions of disturbed flow near stenosis or bifurcations, is linked to the development of atherosclerosis. The deposition of lipids and inflammatory cells on the arterial walls is highly promoted by the low wall shear stress values. For predicting the areas prone to development of plaque and developing targeted interventions it is crucial to understand the distribution of Wall shear stress in the human arteries, diseased or healthy[3].

	WSS at Bifurcation (Pa)		WSS at ICA (Pa)		WSS at ECA (Pa)		Velocity and Time
Phase	Carreau	Casson	Carreau	Casson	Carreau	Casson	
Systolic Pressure (SP)	17.1636	8.4883	12.9055	8.0547	15. 3929	9.7997	v = 0.5 m/s at 0.1s
Dystolic Pressure (DP)	1.8575	1.3644	1.8144	1.3553	1.4778	1.0220	v = 0.1 m/s at 0.5s

SP = Systolic Phase, DP = Dystolic Phase, ICA = Internal Carotid Artery, ECA = External Carotid Artery.

2.3.3 Flow Patterns in Stenosed Arteries

Flow separation, recirculation, and turbulence are some of the complex phenomena caused significantly by the alteration of flow patterns due to stenosis. Downstream the fluid, a pressure drop occurs due to increase in velocity of the blood flowing through narrowed regions. This becomes the reason for flow separation where blood moves away from the arterial wall, forming eddies or vortices. Key factor in plaque progression is these disturbed flow patterns that contribute to low WSS, in severe cases, stenosis can cause turbulent flow, further complicating the hemodynamic environment and increasing the risk of adverse cardiovascular events.

2.4 FSI Simulation Techniques for Cardiovascular Flow

2.4.1 A Comprehensive Overview of FSI and CFD Methods

It is important to understand how blood interacts with the walls of arteries, particularly when they are under conditions such as stenosis where the artery narrows. CFD primarily simulates blood flow, while researchers can observe how the walls of arteries respond to that flow by integrating it with Finite Element Analysis (FEA). This integration is particularly important in diseased arteries, where the vessel may enlarge, stretch, or even collapse, and the interaction between blood and the vessel wall is complex. FSI provides a more accurate visualization of the processes occurring within the body.

2.4.2 Benefits of Using FSI along with CFD for Accurate Simulations

Why is it important to couple CFD with FSI? Well, arteries are not rigid pipes as they deform under pressure, especially in the case of stenosis in the carotid artery. When CFD is combined with FSI, we can model how the walls of the artery move and change in real time with the blood flow. This is crucial for conditions like stenosis because the artery is narrowed and reacts differently than healthy ones. By simulation, we can analyze both the blood flow and the vessel's behavior, and we can get a more accurate idea of how diseases progress or how treatments (like stents) might help [9], [10], [11].

2.4.3 Weak vs Strong Coupling Methods

The main difference between strong and weak coupling strategies is how the fluid-structure interaction is handled. Some employ weak coupling, which involves calculating the vessel wall behavior and blood flow independently before connecting them. Although weak coupling can save processing costs, it may not fully reflect the interaction in highly dynamic systems, such as stenosed arteries. However, in critical conditions like stenosis, where blood and wall interaction occur continually, strong coupling solves both problems at the same time, providing a far more precise and detailed image[9], [10].

2.4.4 FSI Techniques Explicit and Implicit

The two main strategies for simulating blood-artery wall interactions are explicit coupling and implicit coupling. The explicit method is faster but sometimes less stable because it analyzes each step separately, especially when the artery has major deformations. In contrast, implicit coupling controls the interaction in a single step, resulting in better accuracy and stability at the cost of greater computational power. It is particularly significant when modeling arteries with excessive formation of plaque in the carotid artery[10], [11].

2.5 Challenges in Simulating Stenosed Arteries

2.5.1 Complexities of Modeling Stenosis

Stenosis is one of the most persistent challenges for simulations. This plaque build-up causes the artery to significantly narrow and become irregular, which alters how blood flows in that particular area. This generates zones of turbulence and recirculation making it hard to fully model. This is because minor variations in the way that this constriction appears on a WSS model could dictate numerous downstream flow patterns [9], [12]. The simulations play a crucial role in predicting the evolution of natural diseases, as recirculation zones harbor further development of especially plaques[13].

2.5.2 Influence of Arterial Wall Deformation on Blood Flow

Blood flowing through a narrowed artery exerts pressure, stretching and deforming the vessel walls. This deformation can dramatically affect blood flow, creating a feedback loop in which the vessel's response further alters the flow dynamics[10], [14]. This interaction is even more pronounced in stenosed arteries, for example, where the structural integrity of the wall is often compromised, resulting in irregular deformations and complicating blood flow [13].

2.5.3 Computational Cost and Time for High-Accuracy FSI Simulations

One of the biggest hurdles in running high-accuracy FSI simulations is the computational power required. To model the fine details of how blood moves through a stenosed artery and how the walls deform, you need highly detailed meshes and small time-steps. This means the simulation can take hours or even days to run[11]. Although

improvements in computing technology have made this easier, it still presents a challenge, particularly when researchers or doctors need results quickly .

2.6 Blood Flow Modeling

2.6.1 Rheological Models of Blood Flow

Rheology, from a medical perspective, is a science dealing with the deformation and flow of matter. Blood is not merely a simple liquid; it flows through various types of blood vessels in complex ways. In the larger arteries such as the aorta and the healthy carotid artery, blood experiences a relatively high shear rate due to rapid flow and wide diameter of arteries. Its viscosity is approximately constant, so blood is considered to be Newtonian fluid. This simplification makes the process of simulating blood flow much easier and faster. The reality is more complex, especially in smaller vessels or when conditions like stenosis occur, causing the artery to narrow. In these situations, blood behaves like a non-Newtonian fluid, which means its viscosity changes depending on the flow rate. This complexity adds to our models but also enhances the accuracy of the results [9], [12], [13].

To account for the variations in blood behavior, scientists use various mathematical models. Some of the most well-known include the **Power Law**, **Carreau-Yasuda**, **Casson**, **Herschel-Bulkley**, and other models. These approaches aim to enhance our understanding of blood flow under various conditions, including normal circulation and situations where plaque causes blockages. One widely recognized model in the field is the Power-Law model, which characterizes shear stress (τ) in the following manner:

$$\boldsymbol{\tau = m\dot{\gamma}^{n}}$$

In this equation “n” and “m” show the fluid's consistency and the degree of non-Newtonian behavior, respectively.

$$\boldsymbol{\eta = m\dot{\gamma}^{n-1}}$$

As the shear rate increases, the apparent viscosity (η) decreases in the case of shear-thinning fluids ($\mathbf{n < 1}$) or increases for shear-thickening fluids ($\mathbf{n > 1}$). However, this

model has limitations, especially in the case of very low or very high shear rates, which leads to inaccuracies in predicting viscosity [10], [15].

Table I. [10]

Viscosity Model	**Parameters**
Newtonian	μ=0.0035Pa. s
Power Law	k=0.0035 n=0.6
Casson	τ_0=0.004Pa μ_0=0.022Pa m=100
Carreau-Yasuda	μ_0=0.022Pa μ_∞=0.002Pa λ=0.11s n=0.392 a=2

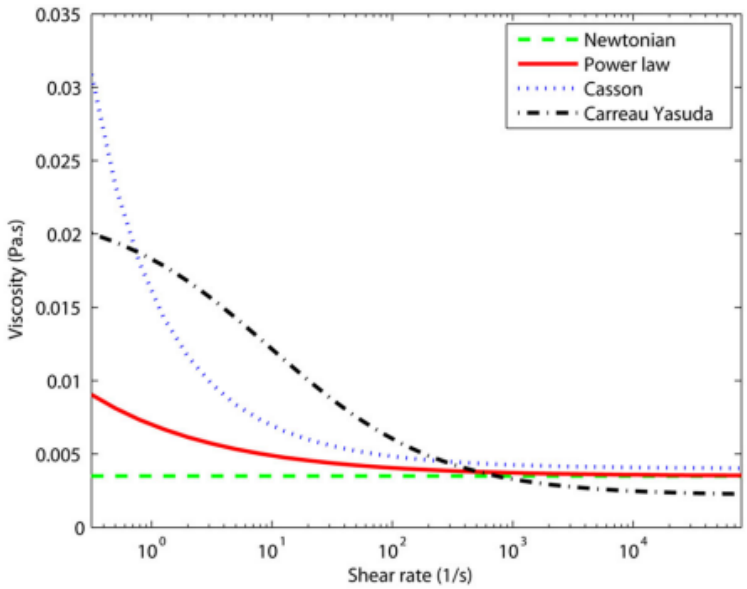

Fig 2. A Graph Representing Relationship between Viscosity and Shear Rate for Different Fluid Models [10]

2.6.2 Role of Navier-Stokes Equations in Blood Flow Simulations

The Navier-Stokes equations (NSEs) play a crucial role in modeling blood flow, particularly due to their ability to describe the motion of viscous fluids. These equations consider factors like velocity, pressure, and viscosity. In stenosed arteries, solving these equations allows us to see how the narrowing of the artery creates disturbed flow patterns, such as vortices or areas of slow-moving blood [10], [11]. These disturbed flows are often where plaque builds up or where the artery is most likely to suffer damage.

Blood flow is mathematically modeled by the Navier-Stokes equations. The conservation of mass and momentum for an incompressible fluid in three dimensions can be expressed as:

Equation 1: Continuity equation for incompressible flow: $\nabla \cdot \mathbf{v} = 0$

Equation 2: Momentum conservation equation:

$$\rho\left(\frac{\partial v}{\partial t} + \mathbf{v} \cdot \nabla \mathbf{v}\right) = \nabla \cdot \boldsymbol{\sigma} + \mathbf{f}$$

Where:

- ρ is the constant density,
- v is the velocity vector,
- σ is the stress tensor,
- f is the external or body force (assumed zero in the simulations).

Equation 3: The stress tensor can be decomposed into hydrostatic and deviatoric stresses:

$$\boldsymbol{\sigma = -pI + \tau}$$

Where:

- p is the pressure,
- I is the identity tensor,
- τ is the deviatoric stress tensor, which is a function of the shear rate tensor D.

Equation 4: The deviatoric stress is given by:

$$\boldsymbol{\tau = \mu\, \dot{\gamma}(D)}$$

Where:

- μ is the dynamic blood viscosity,
- $\dot{\gamma}$ is the shear rate.

In addition, the shear rate tensor D is defined as:

$$\boldsymbol{D = 12(\nabla v + \nabla v^{T})}$$

Finally, the shear rate $\dot{\gamma}$ is defined by a function of D:

$$\dot{\gamma} = \sqrt{2 \sum_{i,j} D_{ij} D_{ij}}$$

2.6.3 Boundary Conditions for Inlet and Outlet in Arterial Models

Accurate boundary conditions are crucial for simulations. The blood that enters the artery from the heart follows a pulsing pattern, which must be accurately represented in the simulation. Likewise, the outlet should reflect how blood exits the artery and flows into smaller blood vessels [9], [14].

The variable flow, as illustrated in Fig. 1, is characterized by changes in flow velocity over time. In this study, the inflow velocity is determined by a time-dependent flow rate 'Q' , which is based on research conducted by Holdsworth et al. The waveform has a duration of T = 1.0 [s], corresponding to a heart rate of 60 beats per minute. During this time, the systolic phase occurs from 0 to 0.47 seconds, while the diastolic phase lasts from 0.47 to 1.0 seconds [11], [16].

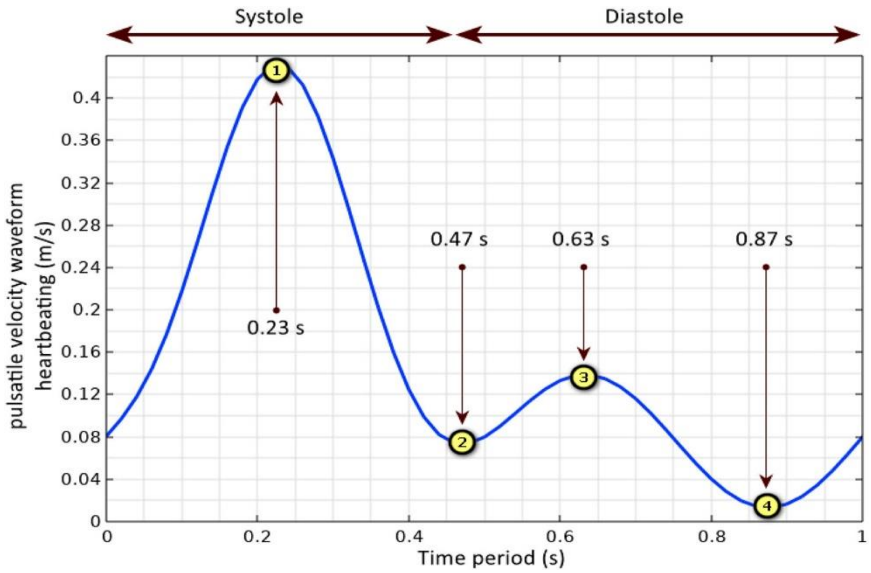

Fig 3. Illustration of Pulsatile Blood Flow

The modeling assumes blood to be homogeneous, incompressible, and possessing a uniform density of ρ = 1060 g/cm^3, along with a viscosity of μ = 0.004 Pa s (refer to Table I). These properties are crucial for accurately simulating blood flow dynamics within the arterial system[11].

Table II. [11]

Parameters	Value	Unit
Blood density	1060	$Kg.m^{-3}$
Blood viscosity	0.004	Pa s
Artery diameter	11	mm
Artery length	200	mm
Max. Inlet velocity	43	$cm.s^{-1}$
Min. Inlet velocity	1.5	$cm.s^{-1}$
Max. Re number	1253	-
Min. Re number	43.7	-
Womersley Number	7.10	-

The Womersley coefficient is a significant factor in this approach, which is expressed as

$$\alpha = r\sqrt{\frac{\omega}{\nu}}$$

The angular frequency of the driving pulse is represented by ω, while ν denotes the kinematic viscosity of blood. For this specific blood circulation, a Womersley index of 7.10 has been recorded, as suggested by Caro et al. [17]. The Womersley index is a dimensionless parameter used to characterize pulsatile flow based on the system's geometry and flow conditions.

Factors of blood as a Newtonian fluid: The blood plasma is mainly composed of water (roughly 91% by volume) proteins, hormones, and glucose and acts as a Newtonian fluid with standard values between 1.1 and 1.3 (megapascal) at the human body temperature of 37°C.

2.7 Structural Modeling of Arterial Walls

2.7.1 Elastic vs Hyper Elastic Modeling of Arteries

Arterial walls contain elastic and collagen fibers that exhibit complex mechanical behavior of arterial walls due to the composition. As blood flows through the arteries under blood pressure, these fibers allow the artery to stretch and recoil as blood pulses through it. When modeling arterial walls, they can be treated as either elastic or hyper elastic materials. A linear stress-strain relationship is assumed for Elastic models which are adequate for small deformations. However, in diseased or highly pressurized arteries deformation is usually large, hyper elastic models are more accurate. Hyper elastic models capture the nonlinear behavior of arterial walls, making them more suitable for simulating realistic cardiovascular conditions, especially in the presence of stenosis[18].

2.7.2 Finite Element Method (FEM) for Structural Simulation

For simulating the mechanical behavior of arterial walls, The Finite Element Method (FEM) is widely used in structural simulations. Finite Element method divides the arterial wall into small, discrete elements, each governed by its own equations of motion. This method allows for detailed analysis of stress and strain distributions within the arterial wall, particularly in regions affected by stenosis. FEM is ideal for modeling the complex geometry and material properties of arteries, making it a critical tool in FSI simulations where both fluid and structural domains interact.

2.7.3 Wall Deformation and Its Effects on Hemodynamics

The deformation of arterial walls has a direct impact on blood flow patterns and overall hemodynamics. As the arterial wall stretches or contracts in response to blood pressure, the diameter of the vessel changes, affecting both velocity and pressure profiles. In stenosed arteries, wall deformation can exacerbate flow disturbances, leading to increased turbulence and changes in WSS. Accurately modeling these deformations is essential for predicting how stenosis progresses and for developing treatments that mitigate its effects. Figure [19]

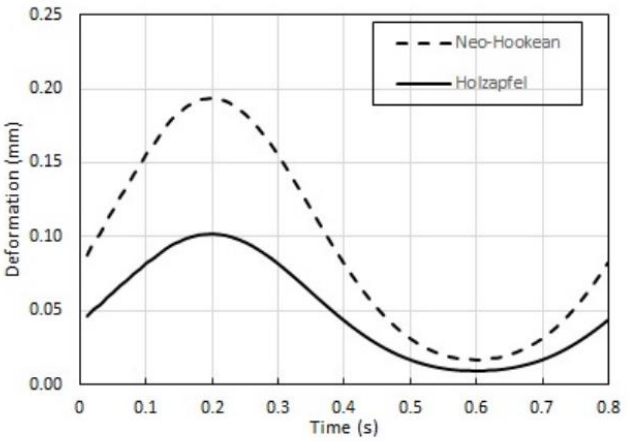

Fig 4. Variation of artery wall deformation at location of maximum stenosis during pulsatile flow

2.8 Performance Parameters in FSI Simulations

2.8.1 Flow Rate and Pressure Drop in Stenosed Arteries

Stenosis directly impacts both the flow rate, and the pressure drop across the narrowed section of the artery. A stenotic arterial lesion that reduces the cross-sectional area of the artery causes an increased velocity and, consequently, a loss in kinetic energy and a pressure drop. Understanding these changes is crucial for diagnosing the severity of stenosis and planning treatments. Accurate predictions of flow and pressure help doctors assess risks and decide on interventions such as surgery or stenting.

2.8.2 Wall Shear Stress and Its Role in Plaque Formation

A key factor in comprehending the progression of plaque is wall shear stress (WSS). Wall shear stress is the drag exerted by flowing blood on the vessel wall. Areas of low WSS are known to encourage plaque buildup, while areas of high WSS can cause plaque to rupture, potentially leading to severe events like stroke[9], [11]. Simulating WSS using FSI can give us detailed overview insights into where plaques are likely to form or rupture, which is invaluable for predicting disease progression and planning preventive strategies [10].

2.8.3 Computational Stability and Convergence in FSI

Simulations must strike a balance between accuracy and computational stability. In arteries with complex shapes, such as those affected by stenosis, it can be challenging to ensure that the simulation converges, meaning it produces consistent results. To accurately capture the rapid changes in blood flow and wall deformation, simulations require carefully refined meshes and time steps. Without these refinements, the results may be unreliable or unstable.

2.9 Clinical Relevance

2.9.1 Application of FSI in Understanding Cardiovascular Diseases

FSI has become a powerful tool in understanding the mechanical factors that contribute to cardiovascular diseases. By coupling blood flow with arterial wall mechanics, FSI provides a more complete picture of how diseases like atherosclerosis develop and progress. FSI models allow researchers to study how changes in blood flow affect arterial walls, and vice versa, helping to identify areas at risk of plaque buildup or rupture. These insights are invaluable for developing new diagnostic tools and treatments, such as stents or drug delivery systems, that target specific hemodynamic conditions.

2.9.2 Insights into the Treatment of Carotid Artery Stenosis

Carotid artery stenosis is a major cause of stroke, and FSI models offer critical insights into how this condition can be treated. By simulating the interaction between blood flow and arterial walls, FSI can predict how different treatment options, such as stenting or angioplasty, will affect the patient. For instance, FSI can be used to evaluate the effectiveness of stents in restoring normal blood flow while minimizing the risk of restenosis. Additionally, FSI simulations can help optimize the design of stents to reduce complications like in-stent restenosis or thrombosis.

2.9.3 Future Prospects of FSI in Medical Research and Surgery Planning

The future of FSI in medical research holds great promise, particularly in the field of personalized medicine. As imaging techniques continue to improve, patient-specific FSI models will become more prevalent, allowing for customized treatment plans based on an individual's unique anatomy and disease state. Surgeons could use FSI simulations to plan procedures in advance, ensuring that the chosen intervention will have the desired effect on blood flow and arterial wall mechanics. The integration of FSI with real-time data from medical devices could also lead to the development of adaptive therapies that respond to changes in the patient's condition during treatment.

CHAPTER 3

DESIGN

3.1 Modelling

3.1.1 Medical Image Acquisition

To identify cardiovascular geometry various medical imaging modalities can be used. In the study of carotid artery imaging, two primary modalities are often considered: CT scans and MRI. CT scans provide high-resolution images, particularly advantageous for visualizing bones and bone-related structures, though their soft tissue contrast is comparatively lower than MRI. Additionally, the use of ionizing radiation in CT poses cumulative risks, limiting its application to cases where the benefits outweigh the hazards. MRI, on the other hand, excels in soft tissue contrast without the radiation risks, making it the preferred choice for this study. Therefore, MRI is chosen over CT to prioritize safety and detailed soft tissue imaging.

MRI scans use magnetic fields, and radio waves to obtain cross-sectional images of the body. During a scan an electric current pass-through coil of wires to generate a magnetic field. Hydrogen protons of water molecules inside the body that normally spin in random directions are then aligned with the magnetic field. A short burst of tuned radio waves is sent through the body which momentarily changes the quantum state of the hydrogen protons (e.g. flips the spin of the proton). When the radio wave stops, the proton returns to its original orientation and in doing so echoes its own radio signal that a scanner detects and deciphers into images. This means that different tissue structures produce different pulse sequences, leading to contrast changes for a number of tissue parameters. In addition, anatomical and physiological variation between subjects requires different pulse sequences to achieve the correct contrast.

The scanned images produced are in a variety of formats, but the most generic and common format is DICOM which contains both image data and patient information. These images are a series of stacked 2D pixels separated by a slice thickness, and when combined provide 3D volumetric data.

3.1.2 Image Segmentation

Image segmentation involves dividing a digital image into segments or regions, enhancing the analysis of specific areas. In medical imaging, it is crucial for differentiating tissues, organs, and abnormalities, aiding in diagnosis and treatment planning.

Segmentation techniques group pixels with similar characteristics, including:

- Thresholding: Segments based on intensity values.
- Edge Detection: Identifies boundaries through intensity changes.
- Region Growing: Expands a region from a seed pixel by adding similar neighboring pixels.
- Machine Learning/Deep Learning: Utilizes trained models to classify pixels with high accuracy.

In research on the carotid artery, segmentation algorithms are actively explored, with various algorithms documented in literature. These can be implemented using programming languages like C, C++, and Java.

Numerous commercial and open-source medical software solutions, such as Slicer, VXL, Fiji/ImageJ, and ITK-SNAP, facilitate image conversion and segmentation through user-friendly interfaces. After segmentation, 3D output files can be saved in CAD formats for computational fluid dynamics (CFD) analysis. Ensuring the output includes necessary geometric and topological relationships is vital for compatibility with CFD software, as additional topological data can enhance the model's usability.

3.1.3 Geometry

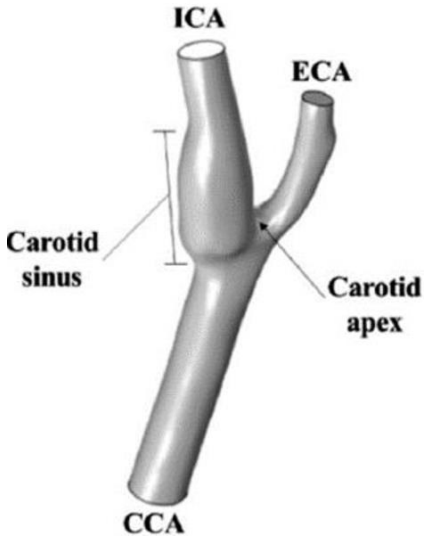

Fig 5. Geometry of the Carotid Artery Bifurcation [20]

The geometry of the carotid artery bifurcation, illustrated in Fig. 5, will be employed for the CFD study [20]. Since it pertains solely to the fluid domain, it will not be suitable for FSI analyses. The CAD model will be developed according to the dimensions provided in Table 1, incorporating a thickness of 0.5 mm for the carotid artery, in accordance with [21].

Table III. Dimensions of the inlet and outlet sections. [14]

Section	Area (mm^2)	Diameter (mm)
CCA	30.898	6.272
ICA	14.670	4.322
ECA	7.184	3.024

3.1.4 Nomenclature

In terms of nomenclature, the common carotid artery (CCA), located upstream of the bifurcation, serves as the inlet. The internal carotid artery (ICA) and external carotid artery (ECA), located downstream of the bifurcation, function as the outlets. The dilation present in the ICA is referred to as the carotid sinus or carotid bulb, while the region separating the two outlet arteries is designated as the carotid apex. Table 1 represents the areas of the sections corresponding to these boundaries.

1. Common Carotid Artery: The CCA originates from the aortic arch on the left and from the brachiocephalic trunk on the right. It serves as the main conduit for blood flow to both the ICA and ECA, ensuring adequate perfusion to the head and neck. Hemodynamic studies have shown that variations in flow velocity in the CCA can impact arterial wall health and plaque formation. This makes the CCA a focal point for assessing cardiovascular risks, especially through imaging modalities like ultrasound and MRI.
2. Internal Carotid Artery: The ICA is responsible for delivering blood to the brain. Its anatomical course is unique as it has no branches in the neck but enters the skull to provide critical blood supply to the brain. Even small stenoses in the ICA can have severe clinical consequences, such as an increased risk of ischemic stroke. Therefore, imaging and monitoring the ICA are critical in stroke prevention and treatment. Computational fluid dynamics studies have also been used to model flow within the ICA to predict potential sites of plaque development.
3. External Carotid Artery: The ECA branches extensively in the neck, supplying blood to the face, scalp, and neck structures. Its anatomical complexity requires careful consideration during surgeries, especially in head and neck procedures. The branches of the ECA make it a significant artery for reconstructive surgeries, and precise anatomical knowledge is crucial to avoid complications during surgical interventions

3.1.5 Material Properties

For Newtonian blood flow, with a density ρf = 1, 060 kg/m 3 and dynamic viscosity, μ= 3.5 × 10^{-3} Pa. s [10]. Regarding the mechanical properties of the vessel, the artery is modelled as a linear elastic isotropic material with a density ρs = 1, 120 kg/m^3, Pois- son's ratio, v = 0.45, and elastic modulus, E = 1.106 ×10^6 Pa[21].

3.2 Meshing Procedure

3.2.1 Meshing Tool

ANSYS Workbench along with ICEM CFD will be used for mesh generation, where both fluid and solid domains will be discretized for FSI analysis.

3.2.2 Meshing Strategy

Fluid Domain: The fluid domain can be meshed with tetrahedral elements, which offer flexibility for complex geometries. Near the arterial walls and stenosis region, prismatic elements will be used to refine the boundary layer. The mesh can be optimized through a Grid Independence Study, starting with different cell sizes (e.g., 0.25, 0.3, 0.4, 0.5, and 0.6 mm). As shown in research conducted by M. Albadawi, cell sizes of 0.3 mm have proven sufficient to capture flow dynamics without impacting the time-averaged wall shear stress (TAWSS) values significantly [22].

Solid Domain (Arterial Walls): The artery walls will be meshed coarsely compared to the fluid domain. This ensures that the structural deformation can be simulated with sufficient accuracy without overwhelming computational demands, especially as structural calculations are more time-intensive than fluid flow [14].

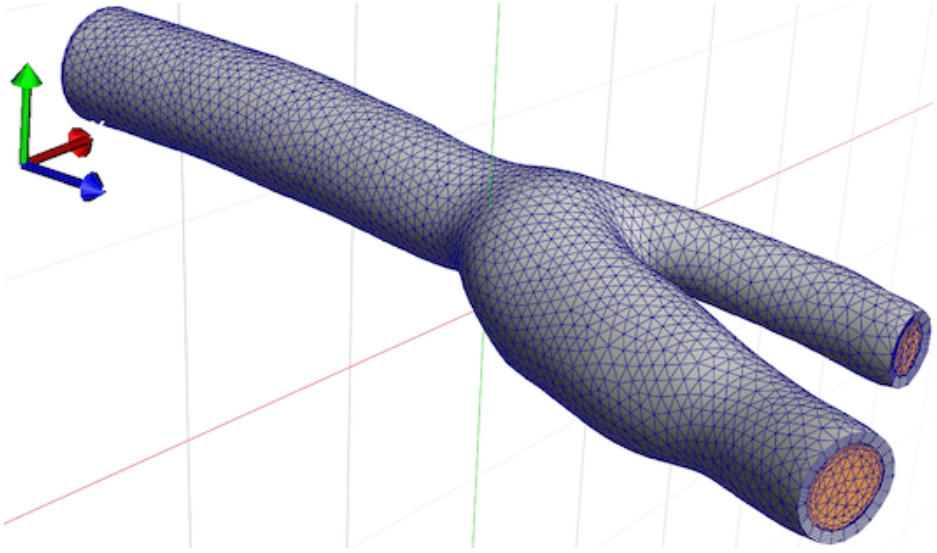

Fig 6. An Example of Meshing done on Carotid Artery

3.2.3 Mesh Refinement and Sensitivity Study

Initial Meshing: A coarse mesh will initially be generated to test the setup and ensure that the model runs smoothly.

Grid Independence Study: A grid independence test can be conducted to verify that the mesh size does not impact key simulation outputs like wall shear stress and velocity distribution. Creating meshes with varying numbers of finite volumes, following a GCI (Grid Convergence Index) method similar to previous studies, to determine the optimal mesh size with minimal error (GCI < 5%) while balancing computational efficiency.[23]

Time-Step Independence Study: Additionally, a time-step independence study can be conducted. Different combinations of time-step sizes (starting from 0.01 s) and iterations per time-step will be tested to ensure that the flow dynamics and wall shear stress remain unaffected by the chosen time-step.[24]

Refinement Strategy: Applying a refinement around critical regions like stenosis and artery bifurcation. The mesh can feature 20 sublayers near the wall to accurately capture flow separation and boundary layer effects, as well as ensure smooth deformation of the arterial walls during FSI coupling.[23]

3.2.4 Meshing Parameters

Element Type and Size: Tetrahedral elements will be employed in the fluid domain for flexibility, and prismatic elements will be added near the artery walls to capture boundary layer phenomena accurately. The element quality metrics such as skewness and orthogonality will be optimized, with an average element quality target of 0.688 and average skewness around 0.226, as seen in past studies.[23]

3.2.5 Boundary Layer Meshing

A well-refined mesh of boundary layer should be generated near the arterial walls. This will include inflation layers to capture the blood flow's viscous effects, particularly around the stenosis. The inflation layer will consist of sublayers, for proper boundary condition treatment.[23]

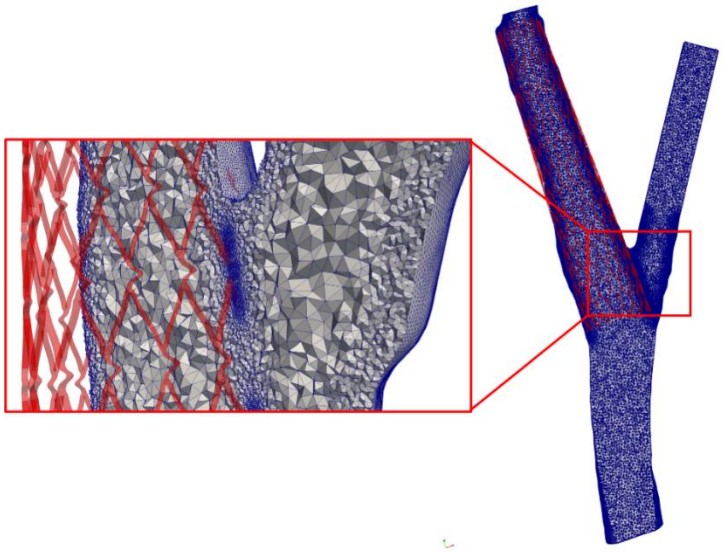

Fig 7. Meshing (Inflation Layers and Tetrahedral Elements) on a Carotid Artery

3.2.5 Future Considerations and Challenges

Meshing of Deformable Walls: Since the project involves FSI coupling, the arterial wall mesh must handle deformations without causing excessive distortion. Adaptive meshing techniques or remeshing strategies can be used to maintain mesh quality as the artery deforms due to blood flow pressure.

Table IV.

Researchers	Blood Model	Viscous Model	Steady/Unsteady	Max Reynold No.	Max Mesh Size
Albadani et al 2022	Non – Newtonian	laminar	unsteady	2000	2,317,041
D.lopes et al 2019	Newtonian	laminar	unsteady	1146	1,012,983
Daniel et al 2022	Non – Newtonian	turbulent	unsteady	Not stated	Not stated
Abhimanyu et al 2022	Non – Newtonian	laminar	unsteady	Not stated	2,061,601

3.3 Expected Results

The expected outcomes for the above study would include the insights of the hemodynamics of the blood flow, the interaction between the blood flow and the arterial walls, and impact of the stenosed artery region on the blood flow. The breakdown for the expected outcomes is as follows.

- Velocity distribution: Understanding the changes in the blood flow velocities near and around the stenosis region. Identifying the areas with accelerated flow within the narrow region of stenosis that causes the development of atherosclerosis. High blood velocities lead to an increase in shear rate that damage the endothelial layer of artery.
- Pressure distribution: Understanding the pressure gradient across the stenosed region of carotid artery. Pressure being high before the stenosed region and drops rapidly in and after the stenosed region of artery according to Bernoulli and continuity equation for pressure drop.
- Disturbed flow patterns: How flow patterns change due to stenosis in the carotid artery, how flow patterns are disturbed, and turbulence is created that ultimately lead to plaque progression.
- Wall Shear Stress: Insights on areas with high and low wall shear stresses that help plaque growth in the carotid artery. High values of wall shear stress within the stenosis and low values of wall shear stress in post stenotic region could provide indicators for the atherosclerosis risk areas.
- Arterial Wall deformation: Understanding how arterial walls respond to blood flow under pulsatile flow and pressure, especially around the stenosed areas.

References:

[1] B. Thomas and K. S. Sumam, “Blood Flow in Human Arterial System-A Review,” *Procedia Technol.*, vol. 24, pp. 339–346, 2016, doi: 10.1016/j.protcy.2016.05.045.

[2] Y. C. Fung, “Blood Flow in Arteries,” in *Biomechanics*, New York, NY: Springer New York, 1997, pp. 108–205. doi: 10.1007/978-1-4757-2696-1_3.

[3] M. Zhou *et al.*, “Wall shear stress and its role in atherosclerosis,” *Front. Cardiovasc. Med.*, vol. 10, p. 1083547, Apr. 2023, doi: 10.3389/fcvm.2023.1083547.

[4] M. Hirschhorn, V. Tchantchaleishvili, R. Stevens, J. Rossano, and A. Throckmorton, “Fluid–structure interaction modeling in cardiovascular medicine – A systematic review 2017–2019,” *Med. Eng. Phys.*, vol. 78, pp. 1–13, Apr. 2020, doi: 10.1016/j.medengphy.2020.01.008.

[5] N. Bessonov, A. Sequeira, S. Simakov, Yu. Vassilevskii, and V. Volpert, “Methods of Blood Flow Modelling,” *Math. Model. Nat. Phenom.*, vol. 11, no. 1, pp. 1–25, 2016, doi: 10.1051/mmnp/201611101.

[6] Y. Mohamed Y. Abdallah and T. Alqahtani, “Research in Medical Imaging Using Image Processing Techniques,” in *Medical Imaging - Principles and Applications [Working Title]*, IntechOpen, 2019. doi: 10.5772/intechopen.84360.

[7] H. Liu *et al.*, “Comparison of Newtonian and Non-newtonian Fluid Models in Blood Flow Simulation in Patients With Intracranial Arterial Stenosis,” *Front. Physiol.*, vol. 12, p. 718540, Sep. 2021, doi: 10.3389/fphys.2021.718540.

[8] A. G. Kanaris, A. D. Anastasiou, and S. V. Paras, “Modeling the effect of blood viscosity on hemodynamic factors in a small bifurcated artery,” *Chem. Eng. Sci.*, vol. 71, pp. 202–211, Mar. 2012, doi: 10.1016/j.ces.2011.12.015.

[9] S. H. Lee, S. Kang, N. Hur, and S.-K. Jeong, “A fluid-structure interaction analysis on hemodynamics in carotid artery based on patient-specific clinical data,” *J. Mech. Sci. Technol.*, vol. 26, no. 12, pp. 3821–3831, Dec. 2012, doi: 10.1007/s12206-012-1008-0.

[10] H. Gharahi, B. A. Zambrano, D. C. Zhu, J. K. DeMarco, and S. Baek, “Computational fluid dynamic simulation of human carotid artery bifurcation based on anatomy and volumetric blood flow rate measured with magnetic resonance imaging,” *Int. J. Adv. Eng. Sci. Appl. Math.*, vol. 8, no. 1, pp. 46–60, Mar. 2016, doi: 10.1007/s12572-016-0161-6.

[11] N. Kaid *et al.*, “Unveiling hemodynamic pulsatile flow dynamics in carotid artery stenosis: Insights from computational fluid dynamics,” *AIP Adv.*, vol. 14, no. 6, p. 065128, Jun. 2024, doi: 10.1063/5.0216867.

[12] E. Earl and H. Mohammadi, “Biomechanics of Human Blood,” in *Biomechanics*, H. Mohammadi, Ed., IntechOpen, 2019. doi: 10.5772/intechopen.78305.

[13] N. Kumar, S. M. A. Khader, R. Pai, S. H. Khan, and P. A. Kyriacou, “Fluid structure interaction study of stenosed carotid artery considering the effects of blood pressure,” *Int. J. Eng. Sci.*, vol. 154, p. 103341, Sep. 2020, doi: 10.1016/j.ijengsci.2020.103341.

[14] D. Lopes, H. Puga, J. C. Teixeira, and S. F. Teixeira, “Influence of arterial mechanical properties on carotid blood flow: Comparison of CFD and FSI studies,” *Int. J. Mech. Sci.*, vol. 160, pp. 209–218, Sep. 2019, doi: 10.1016/j.ijmecsci.2019.06.029.

[15] E. Fatahian, N. Kordani, and H. Fatahian, “The Application of Computational Fluid Dynamics (CFD) Method and Several Rheological Models of Blood Flow: A Review”.

[16] D. W. Holdsworth, C. J. D. Norley, R. Frayne, D. A. Steinman, and B. K. Rutt, “Characterization of common carotid artery blood-flow waveforms in normal human subjects,” *Physiol. Meas.*, vol. 20, no. 3, pp. 219–240, Aug. 1999, doi: 10.1088/0967-3334/20/3/301.

[17] C. G. Caro, T. J. Pedley, R. C. Schroter, W. A. Seed, and K. H. Parker, *The Mechanics of the Circulation*, 2nd ed. Cambridge University Press, 2011. doi: 10.1017/CBO9781139013406.

[18] T. C. Gasser, R. W. Ogden, and G. A. Holzapfel, “Hyperelastic modelling of arterial layers with distributed collagen fibre orientations,” *J. R. Soc. Interface*, vol. 3, no. 6, pp. 15–35, Feb. 2006, doi: 10.1098/rsif.2005.0073.

[19] L. Kallekar, C. Viswanath, and M. Anand, “Effect of Wall Flexibility on the Deformation during Flow in a Stenosed Coronary Artery,” *Fluids*, vol. 2, no. 2, p. 16, Apr. 2017, doi: 10.3390/fluids2020016.

[20] J. Moradicheghamahi, J. Sadeghiseraji, and M. Jahangiri, “Numerical solution of the Pulsatile, non-Newtonian and turbulent blood flow in a patient specific elastic carotid artery,” *Int. J. Mech. Sci.*, vol. 150, pp. 393–403, Jan. 2019, doi: 10.1016/j.ijmecsci.2018.10.046.

[21] M. Jahangiri, M. Saghafian, and M. R. Sadeghi, “Numerical Study of Turbulent Pulsatile Blood Flow through Stenosed Artery Using Fluid-Solid Interaction,” *Comput. Math. Methods Med.*, vol. 2015, pp. 1–10, 2015, doi: 10.1155/2015/515613.

[22] M. Albadawi, Y. Abuouf, S. Elsagheer, H. Sekiguchi, S. Ookawara, and M. Ahmed, “Influence of Rigid–Elastic Artery Wall of Carotid and Coronary Stenosis on Hemodynamics,” *Bioengineering*, vol. 9, no. 11, p. 708, Nov. 2022, doi: 10.3390/bioengineering9110708.

[23] D. Jodko, M. Jeckowski, and Z. Tyfa, “Fluid structure interaction versus rigid-wall approach in the study of the symptomatic stenosed carotid artery: Importance of wall compliance and resilience of loose connective tissue,” *Int. J. Numer. Methods Biomed. Eng.*, vol. 38, no. 8, p. e3630, Aug. 2022, doi: 10.1002/cnm.3630.

[24] Department of Mechanical & Manufacturing Engineering, Manipal Institute of Technology, Manipal, 576104, Karnataka, India. *et al.*, “A Computational Fluid Dynamics Study on Patient-Specific Bifurcated Carotid Artery,” *Eng. Sci.*, 2022, doi: 10.30919/es8d691.