Chapter 1

1. Introduction

This chapter contains a brief overview of modeling and simulations of cardiovascular system, cardiovascular system & its physiology, cardiovascular diseases, application of computational fluid dynamics (CFD) in human circulation, literature review on blood flow analysis in cardiovascular system, research motivation, research gaps, objectives of study and thesis structure.

1.1 Modeling and simulations of cardiovascular system

Cardiovascular diseases (CVDs) are among the most severe public health challenges worldwide. The hemodynamic and blood-vascular interaction aspects are widely considered to have a crucial influence on the development of such illnesses (Doost et al., 2016a). Nevertheless, non-invasive blood flow monitoring technologies (such as Doppler ultrasonography and Phase Contrast MRI imaging) cannot give sufficient spatial and temporal resolution to reliably quantify some critical aspects, such as Wear Shear Stress (WSS) (Park et al., 2016). This type of feature is broadly known to perform a crucial role in the evolution of CVD. Without adequate hemodynamic data, it is difficult to properly assess the risk of CVDs and choose the best treatment options. Hemodynamics and heart and blood vessel illnesses are linked in various subtle ways. However, well-documented qualitative comprehension, connections, and detailed knowledge of hemodynamic circumstances are required to estimate risk and evaluate causes (Cao et al., 2021).

Blood velocity, pressure, and shear stress are all essential hemodynamic variables in illness localization and therapy success (Jung and Hassanein, 2008). A thorough understanding of the altered blood flow conditions in both congenital and acquired cardiovascular illnesses can help optimize the medicines used to treat these conditions. The modeling and simulation of the cardiovascular system include developing mathematical models that may be used to replicate the system's behavior under diverse conditions (Kenjereš, 2016). These models can assist researchers in understanding how various physiological processes work and how they interact. Depending on the level of complexity necessary and the precise questions being asked, several techniques for modeling the cardiovascular system exist (Morris et al., 2015). The cardiovascular system can be simplified to the level of a simple hydraulic system, with the heart acting as a pump and the blood arteries as pipes. More comprehensive models can include details such as the heart's electrical activity, the behavior of individual blood cells, and the impact of numerous hormones and other chemicals. One of the primary advantages of modeling and simulation is that it allows researchers to test ideas and forecast how the cardiovascular system will respond under various scenarios without conducting costly and time-consuming experiments (Kamada et al., 2022). Models can, for example, be used to explore the effects of multiple medications on blood pressure or heart rate or to forecast how changes in diet or exercise habits may affect cardiovascular health. Simulation-based methods can be a beneficial framework in this scenario. New numerical tools allow us to see more information about the heart's workings (Williamson et al., 2022).

Furthermore, because the significance of hemodynamics varies so greatly in each illness state, medical imaging and clinical data are frequently employed to create patient-specific numerical models. These simulations can now plan a specific patient's therapy, perform virtual surgery, and enhance the design. Patient-specific simulation is a significant aspect of cardiovascular biomechanics research, diagnosis, surgery planning, medical device manufacturing, and education (Kumar et al., 2015). Modeling the three-dimensional flow of blood in flexible arteries attached to and supported by surrounding tissue and organs is extremely difficult for various reasons, including getting the geometry correct, accurately describing how the tissue behaves, and determining the outflow boundary conditions.

Medical imaging modalities such as ultrasound (US), computed tomography (CT), magnetic resonance imaging (MRI), and electron tomography (ET) are used to generate patient-specific geometries via 3D reconstruction (Sun and Xu, 2014). Limitations occur in spatial resolution confined the segmentation of the wall and their connective components, as well as the estimation of wall thickness, which is an essential parameter to decisive wall stress. Computational models can't predict stable wall thickness because of tissue development and, remodeling. Following the definition of the geometry and preparation of the computational mesh, the physical aspects of the problem must be identified: for the solid mechanics' simulations, and these constitute stress-strain relationships, vessel wall density, blood pressure, as well as constraint conditions; in order to fluid mechanics simulations, these include viscosity and blood density, as well as boundary conditions such as pressure and velocities. Medical imaging modalities are utilized to acquire physiological data that can be further used to generate boundary conditions.

Important hemodynamic variables such as WSS, pressure, and streamlines can only be acquired using three-dimensional formulations in which patient-specific models of the patient's geometry may be constructed, and flow fields and derived quantities can be obtained (Liu et al., 2015). In recent years, researchers have utilized computation methodologies in order to simulate hemodynamics in three-dimensional models of arteries. Overall, the application of modeling and simulation in cardiovascular research has the potential to dramatically increase our understanding of the intricate connections between many physiological systems and our ability to detect and treat cardiovascular disease.

1.2 Cardiovascular system

The Human circulatory system is constituted of the heart, vessels, and blood which flow throughout the human system (Opie, 2004). The heart works as a pump, which regulates blood flow by expanding and contracting cardiac muscles. Blood vessels, which are comprised of arteries, arterioles, capillaries, venules, and veins, are the conduits that conduct blood all over the system. The pulmonary and systemic circulation are two primary circuits in which pulmonary circulation carries oxygen-deprived blood from the right part of the heart to the lungs and oxygen-rich blood back to the left heart (Pandey et al., 2020a). On the contrary, Systemic circulation transports oxygen-rich blood from the left heart to the entire human system and returns back impure blood to the right heart via venae cavae. Apart from mentioned channels, some other channels also exist, such as coronary circulation (heart), cerebral circulation (brain), renal circulation (kidneys), and bronchial circulation (bronchi of lungs).

Microcirculation is the network of tiny blood arteries that carry nutrients to the body's organs (Baskurt and Meiselman, 2003). The lymphatic system is an open-source network consisting of lymph nodes and lymphatic veins that carry lymph from the tissues to the heart via the lymphatic ducts. Additionally, it maintains fluid hemostasis and combat microbial infections. (Figure 1.1) shows the human circulatory system (simplified) and blood flow in the pulmonary and systemic circulations.

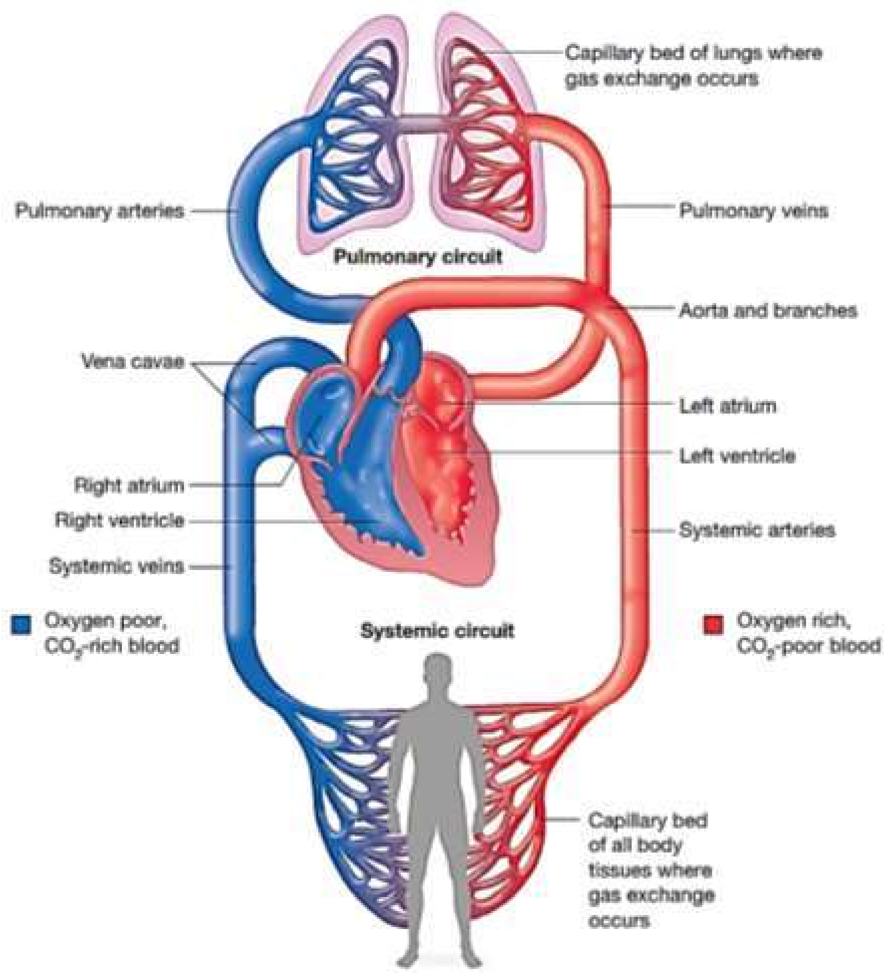

Figure 1.1 : Human cardiovascular sysrtem (“Cardiovascular system,” n.d.)

1.2.1 Heart

The human heart consists of four chambers that pump blood across the body. The pumped blood provides nutrition and oxygen to the organ while eliminating waste (Doost et al., 2015). In the schematic representation of the heart (Figure 1.2), the arrows indicate the direction of blood flow, which is governed by the heart valves. Apparently, the human heart embraced two pumps in a series, i.e., the right ventricle (low-pressure pump) and left ventricle pump (high-pressure pump), that supply blood through pulmonary and systematic circulations, respectively. The circulatory system is further divided into two major circuits- systemic circulation and pulmonary circulation. The systematic circulation takes pure blood from the lungs and delivers it across the body, whereas the pulmonary circulatory system, the right part of the heart, pumps impure blood to the lungs (Krishan B. Chandran, Ajit P. Yoganathan, 2010).

Each systemic and pulmonary circulations have four chambers: left atrium (LA), left ventricle (LV), right atrium (RA), and right ventricle (RV). The upper right-side chamber of the heart is known as the right atrium, which carries oxygen-deprived blood and transfers it into the right ventricles before being pushed onto the lungs via the pulmonary artery for purification purposes. Instead, the left atrium procures oxygen-rich blood obtained via the lungs and pulmonary vein and delivers it into the left ventricle, which further pumps this blood into various parts of the body via the aorta.

The myocardium (cardiac muscle) has three main forms: atrial, ventricular, and specialized excitatory (conducting power). Electrical stimulation causes the atrial and ventricular muscles to contract, raising blood pressure and persuading the blood pump.

The electrical activity is carried throughout the heart by the excitatory and conducting fibers. These specialized conducting fibers contract feebly because of the presence of few contractile fibrils. Actin and myosin filaments are found in the myofibrils that make up the cardiac (atrial and ventricular) muscle. The contraction of the cardiac muscles is caused by the sliding of the actin and myosin filaments over one another.

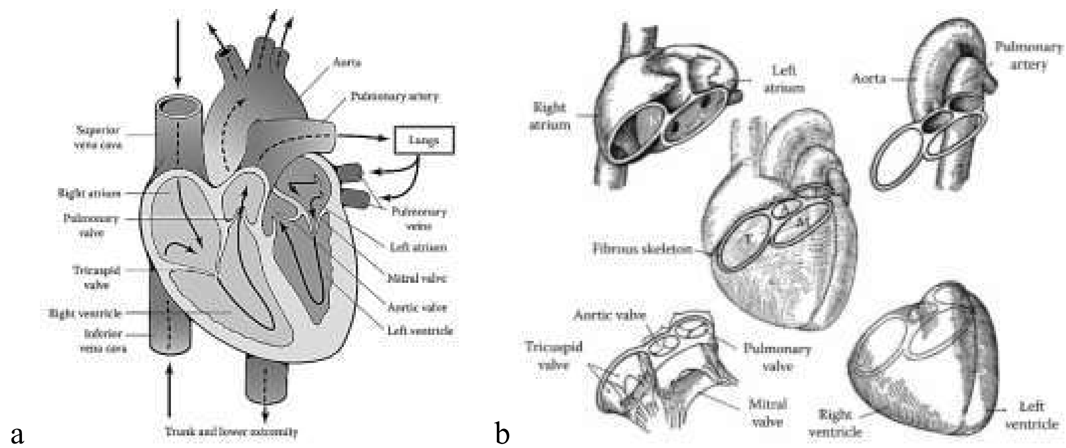

Figure 1.2: Schematic diagram of the four chambers of the heart and the heart valves. The arrows indicate the direction of blood flow (b) The fibrous skeleton and the four chambers of the heart. (Krishan B. Chandran, Ajit P. Yoganathan, 2010)

1.2.2 Cardiac structure

(Figure 1.3) illustrates the geometry of the left and right ventricles schematically. The left ventricle has a cross-section that is more circular, while the right ventricle has a semilunar shape that wraps around a portion of the left ventricle. The anatomical characteristics of the ventricles are tailored to the function of each chamber. For instance, the left ventricle is a high-pressure pump with a modest cavity surface area relative to blood volume due to its cylindrical shape. Left ventricle contraction is characterized by a reduction in the diameter of the cylindrical portion and a longitudinal shortening. Extremely high internal pressure is produced by the contraction of the circumferential fibers in the relatively thick-walled left ventricle. This high pressure provides energy for blood flow in the systemic circulation with high resistance.

Figure 1.3 : A schematic drawing depicting the left and right ventricles. The left ventricle has a more rounded shape whereas the right ventricle has a semilunar shape and wraps around the left ventricle (Krishan B. Chandran, Ajit P. Yoganathan, 2010)

1.2.3 Blood vessels

Substantially three types of blood vessels occur in the human circulatory system, i.e., arteries, veins, and capillaries. The role of blood vessels is to supply nutrients and oxygen to the organs via blood and also carry away metabolic waste from the system. The continuous interaction between blood cells and the arterial wall of the human system results the pulsatile blood flow in the arterial circulation (Charkoudian, 2010). In order to analyze the fluid mechanics of blood flow in human arteries, it is necessary to comprehend the response of the arterial wall and the conduct of blood flow (Conway et al., 2001). Therefore, we will briefly examine blood vessel walls' structural components and mechanics.

1.2.4 Arteries

Arteries are the strongest muscular vessels of the circulatory system, which pass the oxygen-rich blood to the organs by way of the aortic semilunar valve (Ku and Woodruff, 1997). The aorta is the foremost major artery in the human circulatory system. After entering the abdominal cavity at the aortic hole of the diaphragm at the level of the 10^{th} thoracic vertebra, the aorta curves and divides into branches that nourish the upper body (Kumar and Deoghare, 2018). Subsequently, it travels downward and sends off limb-serving branches to the torso, pelvis, perineum, and legs. (Figure 1.4) depicts the arteries, capillary, veins and circulatory system.

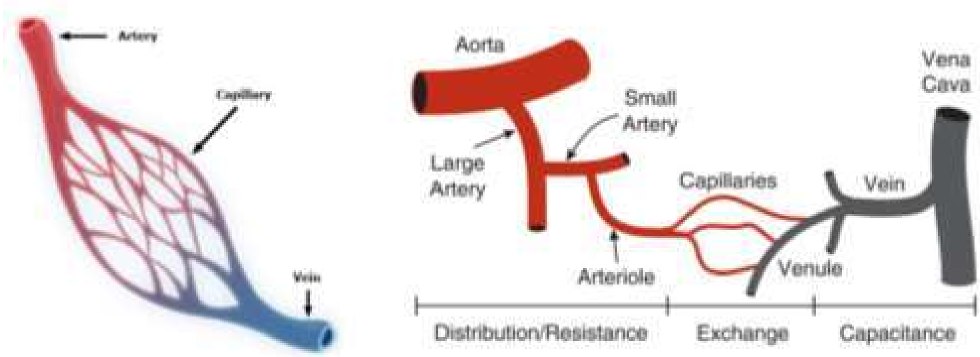

Figure 1.4 : Circulatory system (arteries, capillary, veins and arteriole) ("Capillary system CERT - Capillary - Wikipedia," n.d.)

- **Capillaries**

The capillaries get blood from the arterial system via the arterioles (Akbarzadeh, 2016). Converging capillaries lead to the veins.

- **Veins**

Veins develop from the union of venules, which originate from capillaries (Thomas and Sumam, 2016a). The veins drain the body's upper and lower halves with the assistance of the superior and inferior vena cava, respectively. These two significant veins both lead to the heart's right atrium.

1.2.5 Arteries Morphology

On the basis of diameter, arteries are divided into several categories: arterioles (10-100 mm), muscular arteries (> 0.1 mm), and elastic arteries (> 5 mm), such as the coronary arteries, aorta, carotid arteries, etc. Because atherosclerosis primarily affects arteries, the current study focuses solely on these vessels. In addition, in the present investigation, arteries are considered entirely elastic. However, their mechanical behavior is neither completely elastic nor viscoelastic. Arterial walls are composed of Tunica intima, tunica media, and tunica adventitia, as shown in (Figure 1.5).

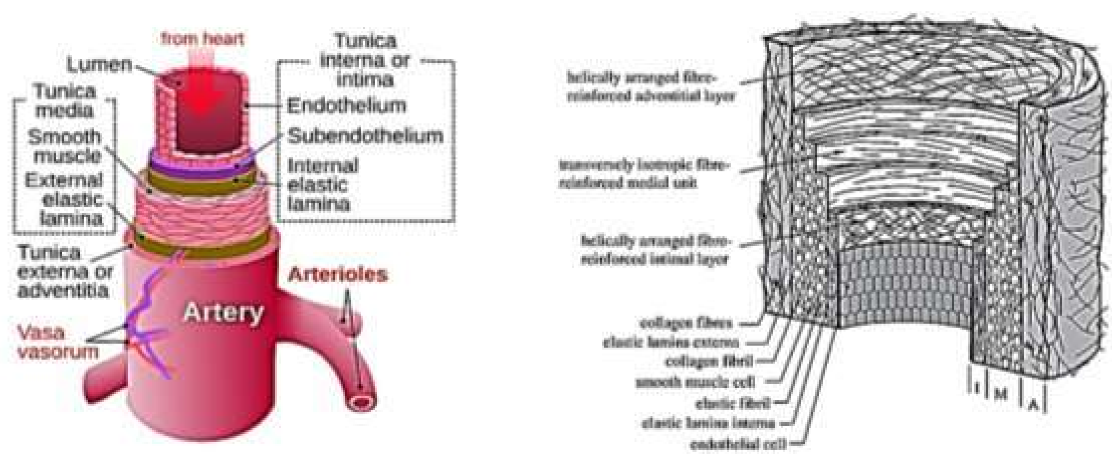

Figure 1.5 : Arteries morphology ("Arteritis - Wikipedia," n.d.)(Gasser et al., 2006)

- **Tunica Intima:**

Tunica intima is the innermost layer of arteries and veins and has direct contact with the blood. A thin basal lamina separates the intima from the sub-endothelium, which consists of a monolayer of endothelial cells. Endothelial cells (0.2 - 0.5 mm) form the interface between the arterial wall and the blood (Bonert et al., 2003). The subendothelial layer of large arteries typically thickens with age or disease (arteriosclerosis).

- **Tunica Media:**

The tunica media is the thickest layer of the vessel wall and have direct contact with both Intima and adventitia media. The tunica media comprises layers of smooth muscle, collagen and is predominantly composed of elastic fibers, most of which are organized in circular sheets form. The smooth muscle cells are spirally arranged across the long axis of the blood vessel. They secrete elastin in the form of perforated lamellae for diffusion. Lamellae increase with age and hypertension (few at birth, 40 to 70 in adults). The most notable histological characteristics of elastic arteries are these lamellae and the extent of the media. Smooth muscle cells are interconnected by a network of elastic fibrils, lending the vessel strength and elasticity (Milutinović et al., 2020). These elongated muscles have multiple cellular extensions that connect to collagen in the media.

- **Tunica Adventitia:**

Tunica adventitia is the outer layer and is made of fibrous connective tissue. This is a relatively thinnest stratum of tissue. Only 10 percent of the adventitia of arteries is made up of elastic fibers, while the majority consists of dense bundles of collagenous fibrils. During systole, the adventitia's collagen impedes elastic arteries from augmenting beyond their physiological limits (Patel et al., 2021).

1.2.6 Blood

Plasma dissolved, red blood cells (RBC), white blood cells (WBC), and platelets make up the human blood. The viscoelasticity of blood is determined by the elastic properties of the red cell membrane and the viscosity of both internal and external fluids. Due to the material properties of the red blood cell membrane and the fluidity of its interior constituents, the cell can readily assume a variety of forms (Beris et al., 2021). However, red blood cell deformation in in vivo and invitro circulation takes place at a largely constant area, which can be designated to the comparatively high dilatational modulus of the cell membrane. After RBC, WBC is the most abundant type of blood cell. However, they account for less than 1% of total blood cell volume in normal human blood and have a negligible effect on the rheological properties of blood's bulk. The viscoelastic interior of WBC makes them more resistant to rapid deformations than RBCs. Hence, RBCs deform considerably less than WBC and require less force to deform compared to WBC. White blood cell deformation requires significantly more force than red blood cell deformation. Consequently, WBC is denser than RBC. Platelets occupy less percentage in total blood volume yet play a crucial function in blood coagulation but have no theological bearing on routine blood simulation. The adhesion of red and white blood cells to the walls of blood vessels increases perceived viscosity. In simulations, blood must be considered as a non-Newtonian fluid due to the different viscosities of red and white blood cells (Yilmaz and Gundogdu, 2008).

1.2.7 Blood Pressure

The blood pressure navigates blood flow through the vessels. Similar to water flowing through conduits from higher to lower pressure, blood also moves from higher to lower osmotic pressure within the body. During the systolic and diastolic state, the contraction and relaxation phases of the heart, blood pressure is measured. In the middle of the normal blood pressure range, a systolic blood pressure of 120 millimeters of mercury and a diastolic blood pressure of 80 millimeters of mercury are considered. This standard measurement is commonly expressed as "120 over 80."Every pulse propels blood throughout the body (Milutinović et al., 2020). Blood pressure is the force exerted by the blood on the arterial walls. Blood pressure decreases from a high level near the heart to a low level distant from the heart. Numerous factors influence blood pressure, including the volume of blood the heart circulates. The diameter of the arteries through which blood circulates is also significant. In general, blood pressure is increased when the heart pumps more blood, and the artery diameter is decreased. Stressful circumstances can cause a transient rise in blood pressure. If a person's blood pressure is measured consistently at 140 over 90, he would be evaluated for hypertension. Untreated hypertension can impair vital organs such as the brain and kidneys and lead to a stroke (Tholl et al., 2004). The detailed visualization of cardiac cycle is shown in (Figure 1.6).

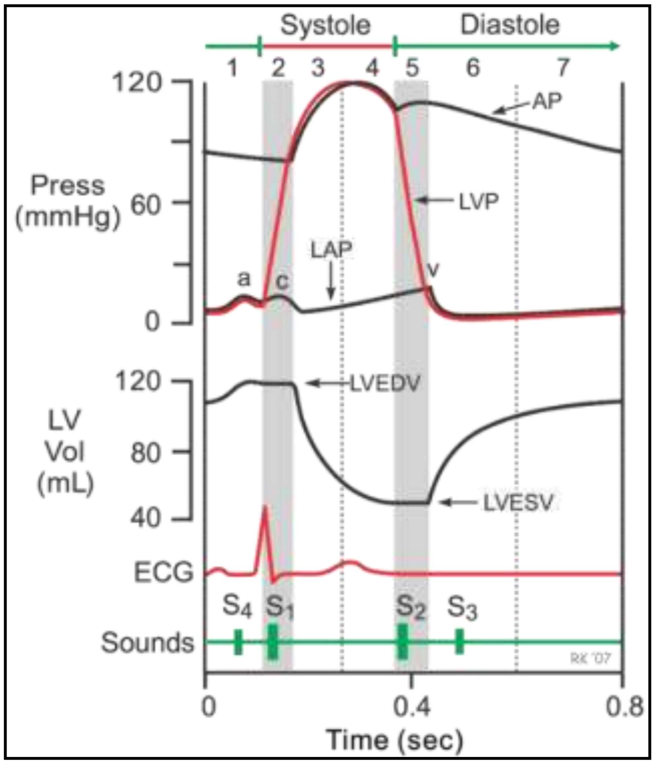

Figure 1.6 : Cardiac cycle ("CV Physiology | Cardiac Cycle," n.d.)

1.3 Cardiovascular disease

Cardiovascular disease (CVD) is a situation that agitates the heart and blood vessels. More than 16 million fatalities per year are attributed to cardiovascular disease; in 2002, more than 5.5 million demises were attributed to strokes (Canchi et al., 2015). In 2001, more than 20 million individuals had suffered from stroke, and 5.5 million of them died ("Cardiovascular diseases (CVDs)," n.d.). Minimum of 68 million Americans suffer from cardiovascular disease, the leading cause of mortality. Approximately 600,000 strokes occur annually ("Cardiovascular diseases (CVDs)," n.d.)) out of 4.5 million stroke victims. In the United States, thousands of cardiac operations are performed daily. In 2004, there were approximately 800,000 coronary bypass or valve repair and replacement procedures. There are several cardiovascular diseases involved with the blood vessels are termed vascular diseases as shown in (Figure 1.7).

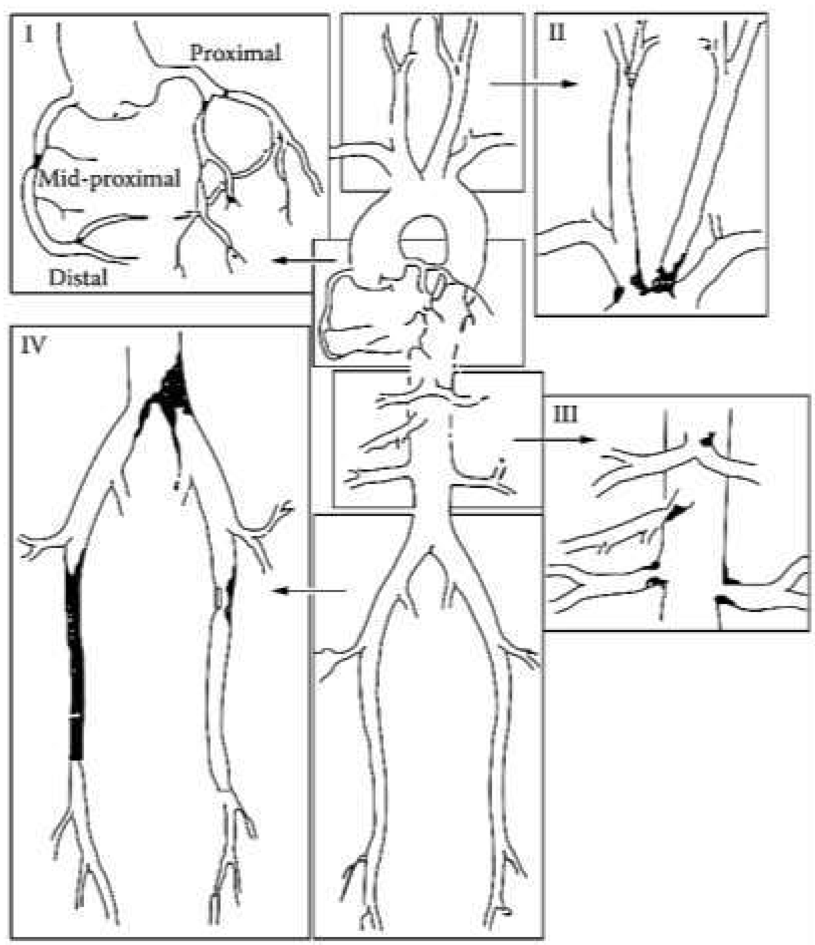

Figure 1.7 : Common sites for the presence of atherosclerotic plaques in the human circulation (DeBakey et al., 1985)

1.3.1 Coronary artery disease

Coronary artery disease is the most prevalent cardiovascular disease linked with the depletion of blood flow in the cardiac muscle due to atherosclerotic plaque formation in the arteries of the heart as shown in (Figure 1.8) ("Global, regional, and national age–sex specific all-cause and cause-specific mortality for 240 causes of death, 1990–

2013: a systematic analysis for the Global Burden of Disease Study 2013," 2015). This condition is responsible for stable and unstable angina, myocardial infarction, and sudden cardiac arrest. (Wong, 2014) A common symptom is chest pain or discomfort that spreads to other areas of the body, such as the shoulder, arm, back, neck, or jaw. Standard imaging criteria derived from cardiac CT or invasive coronary angiography are utilized to establish the presence of coronary artery disease. Angina due to coronary artery disease (CDA) can be caused by a stenosis of >50% or a cross-sectional area reduction of >75%. Thrombus formation following plaque disruption can result in acute coronary syndrome. According to the stenosis severity grading scale, the degree of luminal diameter stenosis is classified as follows: minimal (1-24%), modest (25-49%), moderate (50-69%), severe (70-99%), and occlusion (100%).

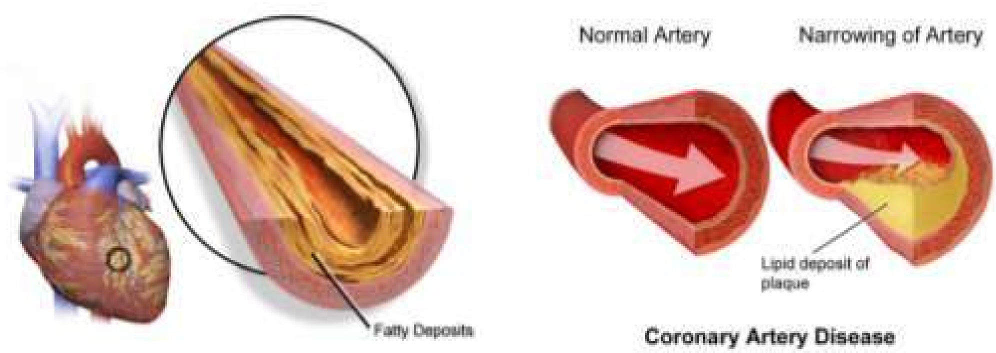

Figure 1.8 : Coronary artery disease ("Blausen 0257 CoronaryArtery Plaque - Coronary artery disease - Wikipedia," n.d.) ("Clogging of arteries treated - Parsi Teb," n.d.)

1.3.2 Peripheral arterial disease

Peripheral arteries associated with the blood supply to lower limbs and their narrowing restrict the blood flow in the lower extremities and may also affect the neck and kidneys as shown in (Figure 1.9) (Creager and Loscalzo, 2018; Nordestgaard et al., 2012).

Diabetes, hypertension, kidney disorders, and elevated blood cholesterol levels are additional risk factors (Lind, 2003; Tsukahara et al., 2015). Atherosclerosis is the prevalent cause of PAD, particularly in the population above the age of 40. In 2015, approximately 155 million worldwide population had PAD (Song et al., 2020). It becomes more prevalent over time. It agitates approximately 5.3% of the 45–50-year-old population and 18.6% of the 85-90-year-old population worldwide (Roth et al., 2020). In the developing country, it adversely affects merely 4.6% of those between the ages of 45 -50 and 15% of those between the ages of 85 - 90.

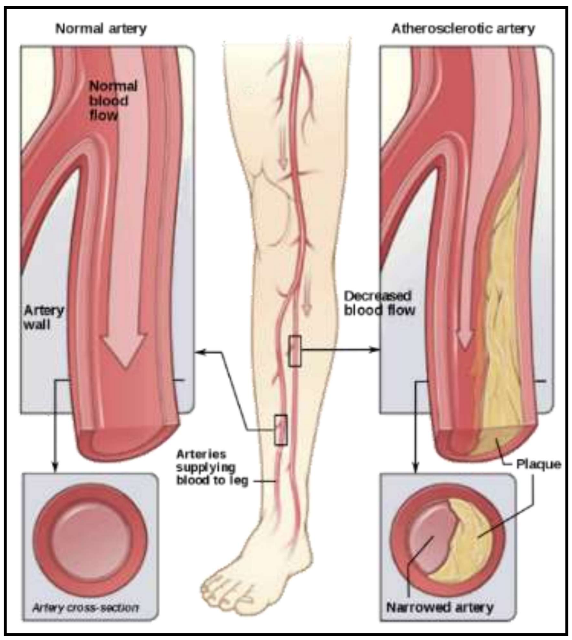

Figure 1.9 : Peripheral arterial disease ("Peripheral Artery Disease – Symptoms & Risks | 50+ World - 50+ World," n.d.)

1.3.3 Cerebrovascular disease

Cerebrovascular disease as shown in (Figure 1.10), comprises a spectrum of conditions that adversely affect the blood vessels of the brain and cerebral circulation. The oxygen and nutrient-supplying arteries are frequently impaired or deformed in this disease (Petersen and Kris-Etherton, 2021). The most typical manifestation of this disease is an ischemic or mini-ischemic stroke and, periodically, a hemorrhagic stroke. High blood pressure is the foremost reason for stroke and cerebrovascular diseases, as it can alter the anatomy of blood vessels and lead to atherosclerosis. Acute stroke is the most prevalent manifestation of cerebrovascular diseases when the blood transport in the brain is compromised (Finegold et al., 2013). Typical stroke signs include one-sided paralysis or numbness, aphasia, changes in vision, and equilibrium problems.

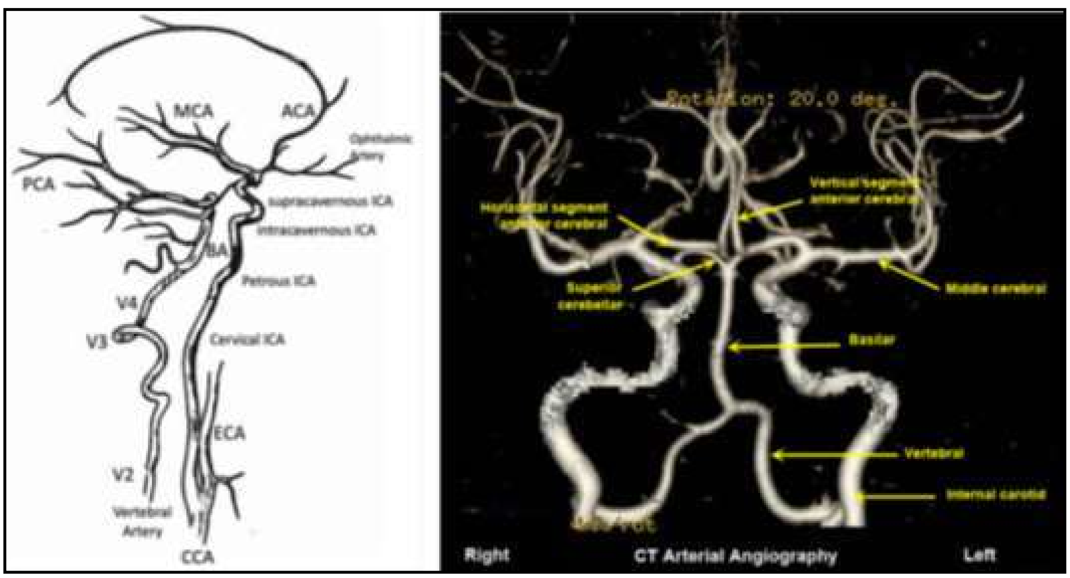

Figure 1.10 : Cerebrovascular disease and CT angiography of human brain ("CT arterial angiography," n.d.) (Barbato et al., 2022)

1.3.4 Renal artery stenosis

Atherosclerosis, or fibromuscular dysplasia, is the most common condition that leads to developing renal artery stenosis (RAS) as shown in (Figure 1.11), which tends to narrow either one or both of the arteries that supply the kidneys (Petersen and Kris-

Etherton, 2021). This tapering of the renal artery leads to reduce the amount of blood flowing to the kidney, leading to high renovascular pressure, a secondary form of high BP. Furthermore, renal artery stenosis (RAS) is the primary cause of chronic kidney disease and coronary artery disease. The vast majority of instances of renal artery stenosis are asymptomatic, and the primary concern is uncontrollable high BP despite the use of medication (Attenberger et al., 2011). The most frequent cause of renal artery stenosis is atherosclerosis, characterized by a narrowing and hardening of the renal arteries due to the accumulation of plaque. This condition accounts for around 90 percent of cases and is known as atherosclerotic renovascular disease.

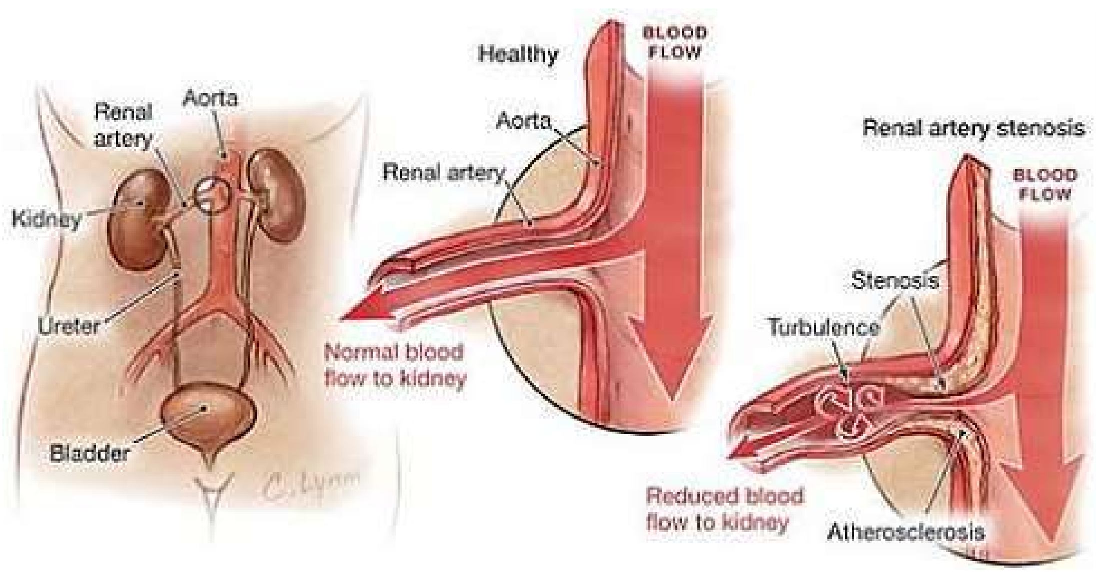

Figure 1.11 : Renal artery stenosis (“Renal artery stenosis - Symptoms and causes - Mayo Clinic,” n.d.)

1.3.5 Aortic aneurysm

Aortic aneurysms as shown in (Figure 1.12) happen when the artery gets bigger than 1.5 times its usual size (Kent, 2014). They usually don't make anyone sick unless they break (Anagnostakos and Lal, 2021; Polzer et al., 2021). Sometimes you might feel pain in your stomach, back, or legs (Stather et al., 2014). The number of people with abdominal aortic aneurysms (or "AAA") varies from 2 to 12%, and about 8% of men over 65 have it (Finol et al., 2003). Most of the time, they are in the aorta in the abdomen, but they can also be in the artery in the chest. Aortic aneurysms happen when the aorta's wall is weak, making it more likely that the artery will burst. When a break happens, there is a lot of internal bleeding, leading to shock and death if not handled immediately (Gillum, 1995). Aortic aneurysms in the abdomen are more common than those in the chest. One reason for this is that there is less elastin in the belly aorta than in the thoracic aorta. Elastin is the main protein in the wall of the aorta that supports weight. Another thing is that the abdominal aorta does not have vasa vasorum, which are blood veins in the wall of the aorta that give nutrients (Vorp, 2007). Most AAA is real aneurysms that affect all three layers (intima, media, and adventitia). AAAs are more common as people get older, with the average age of discovery between 65 and 70.

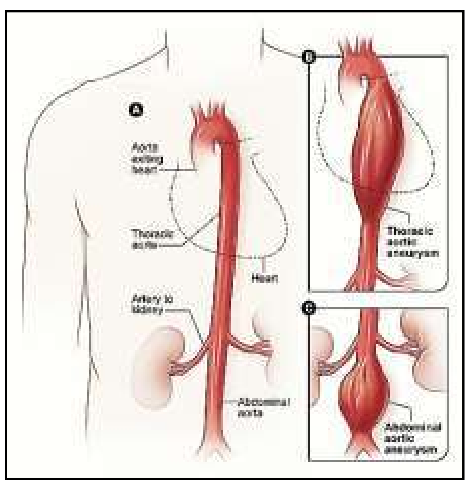

Figure 1.12 : Abdominal aortic aneurysms where, (A) shows a normal aorta. (B) shows a thoracic aortic aneurysm (which is located behind the heart), (C) shows an abdominal aortic aneurysm located below the arteries (Isselbacher, 2005)

1.3.6 Left heart dysfunction

Left ventricular malfunction is a disease in which the heart's left ventricle gets more extensive. It can also cause blood vessels to get smaller. The main purpose of the left ventricle is to pump oxygen-affluent blood to all parts of the body (Uziębło-Życzkowska et al., 2020). So, left ventricular failure can happen if you have a health problem that makes it difficult for your heart to pump blood. Some of these are aortic stenosis, a blood clot in the lungs, heart disease from birth, diabetes, and so on (Abroug et al., 2006). Left ventricular dysfunction can be further sub-categorized into cardiac arrest with preserved ejection fraction (HFpEF) with ejection fraction more than 50%, cardiac arrest with reduced ejection fraction (HFrEF) with ejection fraction less than 40%, and cardiac arrest with mid-range ejection fraction with ejection fraction between 41 and 50%.

1.4 Computational fluid dynamics (CFD)

Computational fluid dynamics (CFD) is a subfield of fluid mechanics that uses computer simulations to investigate and understand fluid phenomena including flow and heat transport. It uses physical, mathematical, and computational techniques to model and foretell fluid flows and their characteristics. Instead of relying simply on costly and time-consuming experimental testing, CFD allows scientific researchers and engineers to get in-depth insights into fluid behavior via the use of advanced computational tools. Simulations in computational fluid dynamics (CFD) are based on the notion of discretizing the equations that govern of fluid flow into a set of algebraic equations. The Naiver-Stokes equations explain the relationship between a fluid's mass, momentum, and energy. To simulate and analyze complicated fluid flow situations, CFD algorithms must numerically solve these equations, which they do by considering variables like velocity, temperature distribution, pressure, turbulence, and many more. Computational fluid dynamics (CFD) has been more relevant in chemical and biomedical engineering in recent years. Heat, momentum, and mass transfer are all fundamental processes in chemistry and biology, and this theory sheds light on how they work.

CFD studies usually involve the following steps:

- **Pre-processing:** This method establishes the overall structure (domain) and then discretizes the field into manageable chunks (control volumes). In addition, we define the actual physical model and the boundary conditions.
- **Solving**: In this step, discretized representations of the equations that govern are iteratively solved to obtain spatial and temporal descriptions of the fluid field in the region of interest.
- **Post-processing:** After fixing the problems, the next step involves analyzing and displaying the outcome digitally.

1.4.1 Governing equations of blood flow

As previously stated, fluid flow laws consist of the conservation of momentum, mass, and energy. Only the energy equation is required when considering flows with substantial variations in density or when heat transfer is significant. Equation 1and illustrate the continuity and momentum equation for Naiver-Stokes formulations characterize blood flow effectively as stated below.

Continuity Equation:

$$\frac{\partial \rho}{\partial t} + \nabla.(\rho \boldsymbol{v}) = 0 \ldots\ldots (1)$$

Momentum Equation:

$$\frac{\partial}{\partial t}(\rho v) + \nabla.(\rho v v) = -\nabla p + \mu \nabla^2 v + \rho \mathbf{F} \ldots\ldots (2)$$

Where **v** is blood velocity, ***t*** is time, ρ is fluid density, p is pressure, μ is fluid viscosity, and **F** denotes the body forces operating on the fluid (per unit mass).

1.4.2 Finite element method (FEM)

The finite element method (FEM) is a computational methodology for solving complex problems in civil, mechanical, biomedical and aeronautical engineering's structural and flow analysis. The FEM assumes that the object of study is divided into finite elements, which may be lined in one-dimensional problems, triangles or rectangles in two-dimensional problems, or hexahedrons, tetrahedrons, or prisms in three-dimensional problems.

The principle of virtual work, also known as the minimum total energy principle, is a popular foundation for structural analysis. This principle states that an elastic body's strain energy (equation.3) equals the work done by the forces acting on it.

$$\int_V \bar{\varepsilon}^T \tau dV = \int_V \overline{\boldsymbol{U}}^T \boldsymbol{f}^B dV + \int_{S_f} \overline{\boldsymbol{U}}^{S_f^T} \boldsymbol{f}^{S_f} dS \ldots\ldots (3)$$

where $\overline{\boldsymbol{U}}$ and are virtual displacements, $\bar{\varepsilon}$ and are virtual strains, and τ are the stresses that cancel out due to the applied loads. The external body weight $\boldsymbol{f}^B$ and surface tractions $\boldsymbol{f}^{S_f}$ are denoted by superscript **S**$_f$, whereas superscript **T** represents the transpose matrix.

Finite element modelling (FEM) works by discretizing a body into elements and connecting them through nodes on their boundary. The continuous motion field is related via interpolation functions (Equation.4). $\overline{\boldsymbol{U}}$ of element *m* to the displacement $\overline{\widehat{\boldsymbol{U}}}$, which is a vector comprised of the three global displacements components U_i, V_i, and W_i at the nodes.

$$\bar{u}^{(m)}(x,y,z) = H^{(m)}(x,y,z)\overline{\widehat{U}}......(4)$$

Where **H** is the displacement interpolation matrix (Equation.5) for moving points, the relationship between strain and displacement tells us which parts have the same strains:

$$\bar{\varepsilon}^{(m)}(x,y,z) = B^{(m)}(x,y,z)\overline{\widehat{U}}...... (5)$$

Here, **B** represents a strain-displacement matrix. The element's stress (Equation.6) is then determined using the specified stress-strain relations, with **D** standing in for element **m** elasticity matrix:

$$\boldsymbol{\tau}^{(m)} = D^{(m)}\varepsilon^{(m)}...... (6)$$

The following is obtained by applying the principle of virtual work (Equation.7) to the assembly of finite elements:

$$\sum_m \int_{V(m)} \bar{\varepsilon}^{(m)T}\tau^{(m)} dV^{(m)} = \sum_m \int_{V(m)} \bar{u}^{(m)T} f^{B(m)} dV^{(m)} +$$

$$\sum_m \int_{S_1{}^{(m)},\ldots S_q{}^m} \bar{u}^{S(m)^T} f^{S(m)} dS^{(m)} ...... (7)$$

Where **q** shows how many sides element m has. The movement, strain, and stress formulas can then be used to figure out an equilibrium formulation for each part. When you add up all of the equilibrium equations (Equation.8) for each element, the following below equation will form:

$$\overline{\widehat{U}}^{T}\left[\sum_m \int_{V^{(m)}} B^{(m)T} D^{(m)} B^{(m)} dV^{(m)}\right]\widehat{U} = \overline{\widehat{U}}^{T}\left[\sum_m \left\{\int_{V^{(m)}} H^{(m)T} f^{B(m)} dV^{(m)}\right\} + \left\{\sum_m \int_{S_1^{(m)},\ldots S_q^{m}} H^{S(m)^T} f^{S(m)} dS^{(m)}\right\}\right] \ldots\ldots (8)$$

Which is the type of global stiffness (Equation.9) formulation controls how far the nodes move and how much force they exert as stated below.

$$KU = R \ldots\ldots (9)$$

Where **K** is the global stiffness matrix and $\mathbf{K}^{(m)}$ is the stiffness matrix for each element (Equation.10):

$$K = \sum K^{(m)} = \sum_m \int_{V^{(m)}} B^{(m)T} D^{(m)} B^{(m)} dV^{(m)} \ldots\ldots (10) \text{ and}$$

$$R = R_B + R_S$$

R is the nodal load vector, which includes the effects of element body forces, R_B (Equation.11), and surface forces R_s (Equation.12). **R** is the nodal force vector, encompassing the effects of $\mathbf{R}_B$ and surface forces. $\mathbf{R}_s$

$$R_B = \sum_m R_B{}^{(m)} = \sum_m \int_{V^{(m)}} H^{(m)T} f^{B(m)} dV^{(m)} \ldots\ldots (11)$$

$$R_S = \sum_m R_S{}^{(m)} = \sum_m \int_{S_1^{(m)},\ldots S_q^{m}} H^{S(m)T} f^{S(m)} dS^{(m)} \ldots\ldots (12)$$

After getting the global stiffness, specified factors (mechanical boundary constraints) and displacements (domain boundary conditions) are added to the equations. Then, stresses and strains, along with the forces and displacements, can be determined by solving the resulting simultaneous equations.

1.4.3 Finite volume method (FVM)

The Finite Volume Method (FVM) uses a mesh to divide the area into small pieces called finite control volumes (CVs). The finite volume comprises a set of surfaces surrounding each node within the mesh. The nodes contain the solution variables and fluid properties. The fundamental process in the (FVM) involves the computation of the conservation equations (Equation.13) over every (CV), preserving the pertinent variable, denoted by $\emptyset$, in a discrete manner for each (CV).

$$\int_V \frac{\partial \emptyset}{\partial t}\, \mathrm{d}V + \int_V \nabla.\boldsymbol{f}(\emptyset) dV = 0 \ldots\ldots (13)$$

The first expression is integrated to obtain the volume average, and the second term is subjected towards the divergence theorem, which transforms the volume integrals into surface integrals:

$$V\, \frac{\mathrm{d}\emptyset}{\mathrm{d}t} + \int_A \boldsymbol{f}(\emptyset)\,.\,\boldsymbol{n}\mathrm{d}A = 0 \ldots\ldots(14)$$

where **n** is the surface normal unit vector and **A** is the total surface area. This could be rearranged so as to produce the following.

$$\frac{\mathrm{d}\emptyset}{\mathrm{d}t} + \frac{1}{V} \int_A \boldsymbol{f}(\emptyset)\,.\,\boldsymbol{n}\mathrm{d}A = 0 \ldots(15)$$

Where the variables are the rates of flow across the surfaces of the various CVs. From the values at the nodes, you can use interpolation or extrapolation methods to find the values at places between the nodes (edge flow).

1.5 Literature review

Hemodynamics analysis plays a crucial job in understanding the hemodynamics and biomechanics of the human cardiovascular system. Computational techniques, such as (CFD) and numerical modeling, have occurred as powerful tools for investigating, blood dynamics, pressure distribution, and wall shear stress. By simulating blood flow through patient-specific anatomical models, researchers have identified flow disturbances, vortex formations, and areas of low or high wall shear stress. These findings contribute to our understanding of disease mechanisms, vascular remodeling, and the impact of hemodynamics on disease progression, guiding the development of targeted interventions. In addition to its diagnostic use, research based on computer simulations of blood flow in a sick circumstance has several potential applications in areas like surgical planning and medical device development. This literature review intends to provide a thorough summary of the existing corpus of knowledge on blood flow analysis in the human cardiovascular system using computational methods, highlighting the advancements, challenges, and future directions in this field. The contributions and recent improvements in hemodynamics modeling and simulation of the cardiovascular system under various pathological challenges in vascular network and ventricle flow are discussed below in the sub-section (1.5.1 -1.5.5).

1.5.1 Recent advancement on cardiovascular disease modelling and simulation

In the late 1990s (Moore et al., 1999; Taylor et al., 1999, 1998), work commenced on the development of image-based modelling methods for simulating blood flow. Since then, several research teams have used these methods to probe the origins of carotid occlusive and aneurysmal illness (Long et al., 2000; Steinman, 2002), coronary arteries (Gijsen et al., 2007), cerebral circulation (Cebral et al., 2005; Shojima, 2004) and aorta (Tang et al., 2006). Analyses of solid mechanics have also utilized patient-specific modeling techniques to forecast aneurysm falling-out risk (Vorp, 2007). Medical device makers have a poor understanding of the anatomical differences, arterial deformation, and biomechanical stresses in the vascular system, which limits the engineering of stents for occlusive disorders and stent implantation to isolate aneurysms. The creation of patient-specific geometric models using imaging data has opened up a new field of application for cardiovascular mechanics, allowing for the accurate prediction of changes in blood flow as a result of future therapeutic treatments. The following subpoints describe recent contributions to cardiovascular disease modeling using patient data.

1.5.2 Aortic aneurysm analysis (AAA)

Blood flow under disease circumstances is very important topic of research as blood flow during normal physiologic conditions. Abnormal blood circulation in the arteries is linked to the prevalence of cardiovascular illnesses, the major cause of death in affluent nations. There have been a lot of simulations of constant-flow blood flow, both computational and experimental. Aneurysms of the abdominal aorta. (Scherer, 1973) They carried out flow visualization studies using in-vitro spherical aneurysm models with continuous flow conditions. His results point to the creation of vortices around aneurysms. (Stehbens, 1974) they utilized a glass model for geometry with various aneurysm configurations. Using flow visualization, he saw that the border layer had begun to split. (Budwig et al., 1993) in the abdominal aortic aneurysm, they examined four distinct Newtonian laminar constant flow diameters. They discovered that the wall shear stress in aneurysm zones is around 10 times lower than at the entry.

(Taylor and Yamaguchi, 1994) they investigated steady circulation in three-dimensional asymmetric computational frameworks based on the assumption that blood is an incompressible Newtonian fluid, whereas (Bluestein et al., 1996) Newtonian laminar and turbulent steady blood flow in an aneurysm model were investigated, and their findings were assessed using the DPIV. (Yu, 2000) performs steady and pulsatile flows for Newtonian blood in unyielding abdominal aortic aneurysms. (Finol and Amon, 2002) performed a computational simulation of a steady-state blood flow in abdominal aortic aneurysms. (Boutsianis et al., 2009a) the researchers investigated the validity of particle tracking velocimetry (PTV) velocity measures in realistic models of abdominal aortic aneurysms.

All previous studies considered blood to be an incompressible Newtonian fluid, despite the fact that its composition does not qualify it as such. Therefore, blood is a non-Newtonian (shear-thinning, viscoelastic, thixotropic) fluid, particularly at low shear rates. (Bessonov et al., 2016). For blood flow in large arteries, non-Newtonian effects can be neglected, and blood viscosity can be assumed to be equal to the high shear rate limit viscosity of blood, 0.0035 Pa s (Marrero et al., 2014). Using the Carreau–Yasuda model, (Marrero et al., 2014) regarded blood as a non-Newtonian (generalized Newtonian) fluid. (Khanafer et al., 2006) Investigated pulsatile blood flow in an abdominal aortic segment with a single aneurysm and regarded blood as a general Newtonian fluid.

Recent research by (Behbahani, 2013) (Philip et al., 2022)(Humphrey and Holzapfel, 2012) examines the application of computational mechanics in the study of intracranial saccular and abdominal aortic aneurysms. In the past few years, image-based modeling methods have been used to conduct patient-specific investigations to determine whether biomechanical factors influence aneurysm initiation, growth, and rupture, as noted in their review (Humphrey and Holzapfel, 2012, 2012; Rissland et al., 2009; Taylor and Figueroa, 2009).

1.5.3 Coronary artery disease analysis (CAD)

The coronary artery level is a particularly challenging vascular location to investigate due to the intricacy of its anatomy and function. Accurate modelling of intracoronary hemodynamics has recently been possible because of advancements in computational fluid dynamics (CFD), giving doctors a novel tool for exploring this vital human system by means of sophisticated mathematical simulations.

(Goubergrits et al., 2008), They looked at how geometric errors affected the study of hemodynamic data and the methods for reconstructing coronary arteries. The research also used the Hausdorff surface distance parameter to objectively evaluate the geometric discrepancies.

(Kim et al., 2010) Explains a method for calculating the flow and pressure of three-dimensional coronary vascular beds by considering the models of the left and right sides of the heart and arterial system and their interactions. This method determines the coronary flow and pressure of three-dimensional epicardial coronary arteries using models of the heart and arterial system and their interactions.

(Sankaran et al., 2012) They create a computational framework for multiscale blood flow modelling and simulation in coronary artery bypass graft (CABG) patients. In this study, the circulatory system is modeled using a patient-specific lumped parameter network (LPN) 0-dimensional (0D) system consisting of resistances, capacitors (compliance), inductors (inertance), elastance, and diodes (valves). By systematically parameterizing the graft geometry, it also evaluates the impact of graft morphology on local hemodynamics and global circulatory dynamics.

(Pinto et al., 2020) They discuss the significance of the blood's viscoelasticity in hemodynamic simulations for diagnosing, preventing, and treating atherosclerotic disease in arteries. In addition, the results demonstrate that the non-linear viscoelastic multi-mode models (Giesekus and sPTT) decrease velocity in regions with greater velocity gradients and a significant difference in peak wall shear stress values compared to the Carreau model solutions. Moreover, the sPTT model should be the preferred option for future applications.

(Pandey and Yadav, 2022) This study examined the importance of various blood viscosity models and other Reynolds numbers in the simulation of blood flow in a multistenosed LCA model. Using angiographic images of a healthy individual, the three-dimensional LCA model was reconstructed. For blood flow simulation, Carreau, Quemada, and Modified Cross viscosity models were considered.

1.5.4 Arterial stenosis

(Nakamura and Sawada, 1988) Investigated the flow of a non-Newtonian fluid (blood) through axisymmetric stenosis using finite element methods. Also discussed, based on the calculated results, is the impact of the non-Newtonian property of blood on the development of vascular lesions, particularly post-stenotic dilatation.

(Linge et al., 2014) They used a three-dimensional k-v turbulence model for flows with Reynolds numbers 500 and 1000 to study the impact of various spiral component magnitudes on vascular stenosis. This study used a k-v turbulence model to simulate pulsatile spiral blood flow through a 75% cross-sectional reduction stenosed conduit.

(Patel et al., 2017) They evaluated physiological flows in rigid pathological arterial flow phantoms simulating an abdominal aortic aneurysm (AAA) at rest with aortoiliac bifurcation and iliac stenosis in vitro using 2D PIV measurements. Also investigated the impact of aortoiliac bifurcation and iliac stenosis on AAA flow dynamics by comparing the reference configuration to the nature of flow patterns, vorticity evolution, vortex core trajectory, and hemodynamic parameters.

(Liu et al., 2021) Using computer simulations and individual patient data, scientists compared the performance of Newtonian and non-Newtonian fluid models for simulating blood flow in people with cerebral artery stenosis. They observed that the time-averaged WSS for most regions of an intracranial aneurysm was comparable using the casson and newtonian rheological assumptions. However, in the dome region where the WSS was poor, the disparity reached a maximum of 55%.

1.5.5 Left ventricle hemodynamics

Combining patient-specific medical imaging data with computational fluid dynamics (CFD) models of left ventricular (LV) flow has demonstrated promising results in getting patient-specific hemodynamics information for functional evaluation of the heart. Dealing the LV manually and using different software for mesh development and registration are common first steps in model-building. In biomedical engineering, image-based modeling of blood flow is an active field of study. It uses computational fluid dynamics (CFD) to calculate patient-specific blood flow information that is not observable in vivo by applying CFD to computer models of the heart, arteries, or veins based on images. Numerous researchers have utilized this framework to investigate the biomechanical basis of illnesses and ways to enhance cardiovascular diagnosis and therapy.

(Khalafvand et al., 2017) They investigate three-dimensional blood flow in the LV using a computer approach in conjunction with magnetic resonance imaging of cardiac motion. Recognize that momentum transmission dominates the vortices during flow acceleration and deceleration via the mitral orifice. (Doost et al., 2016b) They looked at how different non-Newtonian models affected intraventricular hemodynamics, since blood viscosity fluctuates non-linearly with shear rate. Furthermore, it was shown that non-Newtonian models have a considerable effect on intraventricular flow dynamics, while the Newtonian assumption cannot accurately mimic the flow dynamics inside the LV throughout the cardiac cycle.

(Schenkel et al., 2009) They use magnetic resonance imaging (MRI) technology to provide a patient-specific, time-dependent geometry of the ventricle to be simulated. In this work, they identify the high sensitivity of the flow features to the boundary conditions imposed at the inflow.

(Nguyen et al., 2015) Demonstrated a semi-automatic, low-operator-participation approach for LV meshing, smoothing, and reconstruction. It also verifies the model creation using CT and MR benchmark image datasets, as well as demonstrating the practicality of employing the models to conduct CFD simulations of intraventricular hemodynamics.

In summary, this literature review provides a comprehensive overview of recent cardiovascular disease modelling and simulation developments. It highlights the advancements in computational fluid dynamics, multiscale and multi physics models, data-driven approaches, and their clinical applications. The review also addresses the challenges and future directions in this field, underscoring the potential impact of these models in advancing cardiovascular healthcare.

1.6 Purpose of study: Motivation

Atherosclerosis is a chronic illness that causes heart attacks and strokes, impacting millions of people all over the globe. The carotid artery, the aorta, and the coronary arteries are common development sites. Deposition of lipids behind the endothelial layer of the artery, resulting in plaque build-up, is the primary cause of atherosclerosis development. Further supporting the idea that abnormal flow conditions play a significant role in the genesis and progression of atherosclerosis is evidence that plaque formation occurs mainly around bifurcations or curvatures. Research on both cells and whole organisms demonstrates the necessity of studying local hemodynamics in atherosclerotic hotspots. Although non-invasive diagnostic techniques like computed tomography (CT) and magnetic resonance imaging (MRI) provide detailed anatomic information, computational methods like computational fluid dynamics (CFD) and fluid-structure interaction (FSI) allow for the study of local hemodynamics at patient-specific models. To get reliable findings from CFD analysis, it is crucial to recreate anatomical models using CT or MRI scans.

The important cardiovascular diseases linked with substantial morbidity and death include abdominal aortic aneurysm (AAA), stenosis, coronary artery disease (CAD), and irregular blood flow in the left ventricle. Knowledge of their underlying hemodynamics and biomechanics is essential to effectively diagnose, treat, and prevent these disorders. Computational fluid dynamics (CFD) is helpful in studying the intricate flow dynamics associated with these diseases. The complex processes behind cardiovascular disorders provide a formidable challenge, but modeling and simulation offer a unique chance to tackle this problem. These methods allow for the investigation of disease progression, identifying critical factors contributing to disease development, and evaluating treatment efficacy by integrating knowledge from various disciplines such as physiology, biomechanics, fluid dynamics, and mathematical modeling. With this all-encompassing knowledge of disease processes, we can better design specific interventions and tailor treatments to individual patients. This study's impetus emphasizes the importance and potential effect of employing CFD to investigate AAA, stenosis, CAD, and left ventricular blood flow.

In conclusion, CFD research into cardiovascular diseases such as AAA, stenosis, CAD, and left ventricular blood flow can greatly advance our knowledge of these conditions, optimize treatment techniques, and boost patient outcomes. CFD is a helpful tool for cardiovascular research and clinical practice because of its thorough hemodynamic analysis, non-invasive assessment, predictive modelling, and personalized medicine capabilities. Future developments in computational methodologies, validation, and partnerships will boost CFD's translational potential in cardiovascular healthcare. Based on comprehensive literature survey regarding methodology and recent contribution to cardiovascular disease modeling and simulation, five research objectives are chosen for this work described in subsection 1.7.

1.7 Research objectives

In the light of available literature explained above, following are the main objective of the main objectives of the thesis.

- ❖ The pulsatile 3D-Hemodynamics in a doubly afflicted human descending abdominal artery with iliac branching.
- ❖ Effect of rheological models on pulsatile hemodynamics in a multiply afflicted descending human aortic network.
- ❖ Influence of abdominal aortic aneurysm shape on hemodynamics in human aortofemoral arteries: A transient open-loop study.

- ❖ An open loop (0D-3D) modelling of pulsatile hemodynamics for the diagnosis of a suspected coronary arterial disease with patient data.
- ❖ Image based modelling and simulation of hemodynamics in human left ventricle using CT data.

1.8 Contributions of work

The primary contribution of this work is to study hemodynamics. It investigates cardiovascular diseases under pathological conditions like abdominal aortic aneurysms (AAA), Stenosis, multiply afflicted vascular diseases, and coronary artery hemodynamics and applies (0D-3D) modeling approach in left ventricle blood flow analysis using real-time patient data. This study makes several new inferences about how that shape affects blood flow dynamics and relates these findings back to the forecasting of AAA development and rupture. Here we discuss the mechanisms via which specific AAA and RIIAS shapes might lead to a compromised hemodynamic state. We also make some interesting insights into the flow dynamics in aortofemoral arteries from the perspective of a pulsatile boundary condition that is physiologically appropriate to the cardiac cycle. Novel advances in (0D-3D) open-loop system modelling and simulation of the cardiovascular system for the study of coronary artery disease hemodynamics. To give a framework for simulating hemodynamics of the left ventricle under varied pathological situations and to contribute to understanding the difficulty of manual and automated segmentation of the Left ventricle for image-based blood flow dynamics research.

1.9 Thesis structure

This thesis is organized in to seven chapters. Chapter 1 presents overview of cardiovascular system, cardiovascular diseases, modelling and simulation, methodology used for blood flow simulation, literature review providing background of this study, motivations, research gaps and objectives. Chapter 2 deals with patient-specific hemodynamics studies in a doubly afflicted human descending abdominal artery. In this work pulsatile blood flow analysis in doubly afflicted diseased condition (aneurysm & stenosis). Chapter 3 deals with effect of rheological models on hemodynamic in a doubly afflicted human descending abdominal artery network using patient-specific data. In this work suitability of rheological models has been studied according to pathological condition of artery. Chapter 4 deals with influence of abdominal aortic aneurysm shape on hemodynamics in human aortofemoral arteries using open-loop system. In this work effect of morphology and has been studies using real time data of patient from open repository of simvascular. Chapter 5 deals with blood Flow and Pressure Modelling of Suspected Coronary Artery Disease Using (0D-3D) coupling and Open Loop System. In this study we investigated various hemodynamics parameters for coronary artery during cardiac cycle. Chapter 6 deals with image-based modelling and simulation of hemodynamics in LV using CT data. In this study transient blood flow patterns in 3D model of LV generated using the instantaneous CT data of the left ventricle (LV) with clinical pulsatile blood flow rates. Chapter 7 deals with conclusion and future scope of the work is presented.

Chapter 2

The pulsatile 3D-Hemodynamics in a doubly afflicted human descending abdominal artery with iliac branching

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The pulsatile 3D-Hemodynamics in a doubly afflicted human descending abdominal artery with iliac branching

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ABSTRACT

The study of patient-specific human arterial flow dynamics is well known to face challenges like a) apt geometric modelling, b) bifurcation zone meshing, and c) capturing the hemodynamic prone to variations with multiple disease complications. Due to aneurysms and stenosis in the same arterial network, the blood flow dynamics get affected, which needs to be explored. This study develops a new protocol for accurate geometric modelling, bifurcation zone meshing and numerically investigates the arterial network with abdominal aortic aneurysms (AAA) and right internal iliac stenosis (RIIAS). A realistic arterial model is reconstructed from the computed tomography (CT) data of a human subject. To understand the combined effect of the aneurysm and aortoiliac occlusive diseases in a patient, an arterial network with AAA, RIIAS, multiple branches tapering, and curvature has been considered. Clinically significant pulsatile blood flow simulations have been carried out to trace the alteration in the flow dynamics with multiple pathological complications under consideration. The transient blood flow dynamics are investigated via wall shear stress, wall pressure, velocity contour, streamlines, vorticity, and swirling strength. During the systolic deceleration phase, the rhythmic nested rapid secondary oscillatory WSS, adverse pressure gradients, high WSS, and high WP bands are noticed. Also, the above studies will help researchers, clinicians, and doctors understand the influence of morphological changes on hemodynamics in cardiovascular studies.

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