Computational Hemodynamic Study of Healthy and Pathological Abdominal Aorta

A Dissertation Submitted

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in

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by

Abhishek Kandpal

Registration No.: 801683001

Under the supervision of

Dr. Neeraj Kumar

Assistant Professor, MED

Mechanical Engineering Department
Thapar Institute of Engineering & Technology, Patiala

July 2018

CERTIFICATE

I hereby declare that the thesis entitled, **"Computational Hemodynamic study of Healthy and Pathological Abdominal Aorta"** is an authentic record of my work carried out as per requirements for the award of degree of **Master of Engineering** in **Thermal Engineering** at **Thapar Institute of Engineering & Technology, Patiala** under the supervision of **Dr. Neeraj Kumar**, Assistant Professor, Mechanical Engineering Department, Thapar Institute of Engineering & Technology, Patiala during June 2016 to June 2018. No part of the matter embodied in this report has been submitted to any other university or institute for the award of any other degree.

Date: 16-7-2018

Abhishek Kandpal

It is certified that the above statement made by the student is correct to the best of my knowledge and belief.

Dr. Neeraj Kumar

Assistant Professor

Mechanical Engineering Department

Thapar Institute of Engineering & Technology

Patiala-147004

This work is dedicated to

My mother and Gurus

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Abhishek Kandpal

Abstract

It had been observed from last two decades that the morbidity and mortality rate due to cardiovascular disease increases in the significant manner. Mostly patients around the world are affected by heart disease and systemic disorder such as hypertension & arterial dilation typically called aneurysm. Only in India the mortality rate due to Cardiovascular Disease (CVD) increases up to 28 % until 2017. It has been widely observed that atherosclerosis is the primary cause of cardiovascular disease. In present work abdominal aneurysm (AA) is selected for computational investigation. The abdominal aneurysm is defined as the dilation of the abdominal aorta in lateral direction along the diameter. In this work computational investigation of hemodynamic study of aortic abdominal aorta in healthy and pathological (aneurysed) conditions are done. In computational study, the critical hemodynamics parameters such as wall shear stress about the circumferential location and axial flow velocity are considered. These parameters are taken at the mid plane perpendicular to flow direction of affected region. The another parameter which is flow recirculation is also analyzed for its effect on diseased condition. A comparative study is performed between the healthy and aneurysed (saccular & fusiform) condition of different sizes.

The computation is performed on a three dimensional model created by patient specific DICOM image. The different aneurysm sizes and shape are taken from the cited literature. The results found that the flow velocity of the aneurysed model is oscillating in nature. The average flow velocity varies between 50 cm/sec to 80 cm/s for aneurysed model which is lower than healthy case (80 cm/s). The oscillatory shear stress is least (-0.3 Pa in fusiform 5.5 cm & 6 cm) at 0^o position and maximum (-7.3 Pa in 5.5 cm saccular aneurysm at circumferencial position 270^o) in between 180^o and 270^o. The final conclusion of this work suggested that the aneurysm of 5.5 cm and greater than 5.5 cm is very critical and immediate sugerical intervention is required for preventing it from rupture (hemoharrge).

Keywords: *Aortic abdominal aneurysm (AAA), Saccular and Fusiform, patient specific DICOM.*

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Nomenclature

Parameters	Description
∇	Vector del differential operator
$\boldsymbol{\rho}$	Density of the fluid
$\boldsymbol{\mu}$	Dynamic viscosity
$\boldsymbol{u}$	Velocity space vector
$\boldsymbol{T}$	Transpose matrix
$\boldsymbol{I}$	Identity matrix
$\boldsymbol{p}$	Pressure
$\boldsymbol{F}$	Body force acting on the fluid

Abbreviations

CVD	Cardiovascular Disease
IHD	Ischemic Heart Disease
CHD	Coronary Heart Disease
PVD	Peripheral Vascular Disease
CFD	Computational Fluid Dynamics
WSS	Wall Shear Stress
AAA	Aortic abdominal aneurysm
ECM	Extra Cellular Matrix
CT	Computed Tomography
OR	Open surgery Repair
EVAR	Endovascular aneurysm repair
ICA	Internal Carotid Artery
ECA	External Carotid Artery
MRI	Magnetic Resonance Imaging
STL	STereo Lithography
STEP	STandard for the Exchange of Product model data
DICOM	Digital Imaging and Communication in Medicine
MITK	Medical Imaging Tool Kit
GUI	Graphical User Interface
FEA	Finite Element Analysis
FSI	Fluid Structure Interaction
CCA	Common Carotid Artery
CAD	Computer Aided Design

Chapter 1

Introduction

1.1 Introduction

Cardiovascular system is mainly responsible for transportation of oxygenated blood between the organs of the mammalian body. Its dis-functioning causes severe fatal effect to the body organ functioning and sometimes, it may lead to death. Cardiovascular disease (CVD) is the major cause of death in many countries including India. According to the Registrar General of India CVD is the top killer of Indians, accounting for 23% of all deaths in 2010-13 as compared to 20% in 2004-06 [1]. In a survey, Ischemic heart disease (also known as coronal heart disease) is the top killer across the most states of India, overall CVD affected deaths reaches to 28% [2]. The leading Cardiovascular diseases are CHD (Coronary heart disease) or Ischemic heart disease (IHD), Cardiomyopathy, Aortic disease such as Aneurysm (thoracic, abdominal & intracranial), aortic dissection, Peripheral vascular disease (PVD), Valvulitis (inflammation of the heart valves), Pericarditis (friction between heart layers) and Congenital heart disease. These problems are attracting the attention towards the study of these cardiovascular diseases. The researchers are trying to find the reasons behind the production of the disease either by experiments or by using the computational and numerical simulation techniques. Till seventies, in-vitro analysis (creating artificial models and hemodynamic environment) or in-vivo analysis on animals were the main methods for cardiovascular investigations. But in last few decades, the computational capability has been increased in exponential manner and recent advancement in data-acquisition technology that provide large amounts of quantitative information on vascular geometry, computational investigation plays an important role in analyzing this physics. Thus numerical investigation popularly known as computational fluid dynamics (CFD) found promising field of application in vascular research. Computational investigations of hemodynamic environment of vascular geometry either created by using mathematical modeling or by reconstructed from patient specific vascular morphologies give more insight to this complex phenomenon and having more clinical relevance.

The basics of the study cardiovascular system and diseases related with it, is started with the study of the hemodynamics. Detailed knowledge of hemodynamics as well as response of blood vessel is very much important to understand the healthy and pathological behavior of the human vascular system. Abnormal flow conditions, such as flow separation and flow reversal zones, low and oscillatory shear-stress zones responsible for the development of arterial diseases [3]. Hemodynamics is known as the study of dynamics of blood flow and typically deals with forces required to develop the proper blood flow within the arterial network. It also explains about the physics of blood flow in blood vessels. Competent blood circulation is an important condition for supply of oxygen and other nutrients to all cells, tissues and organs, which leads to a good cardiovascular health and a bonny life. Physicians from medical domain such as a doctors and nurses, the hemodynamic study concentrates themselves on forces acting on blood vessels i.e. blood pressure and through which they analyze the health of heart. For our part of interest, we will concentrate on systemic hemodynamics (systemic concerns with the circulation of oxygenated blood from heart to body). The interest in systemic hemodynamics is obvious: A significant majority of all cardiovascular diseases and disorders are related to systemic hemodynamic dysfunction (abnormality in the operation of a specified bodily organ or system).

With this background the present work is to computationally investigate the hemodynamic environment at normal and pathological blood flow conditions at abdominal aortic region (between renal arteries and abdominal bifurcated zone) using vascular morphologies. Pathological model of abdominal aorta with different sizes are created and simulated under single phase laminar flow condition with actual physiological blood flow rate. This study helps to investigate the hemodynamics under normal and pathological condition and suggests the importance of surgery for fatal diseased condition in accordance with abnormal hemodynamics.

1. 2 Motivation

Atherosclerosis (derived from Greek word ‘athērē which means ‘groats’ and ‘sklērōsis’ which means ‘hardening’) is the most common disease which the artery become narrow and hardened and this happened due to plaque built up on the arterial walls. This disease can happen at any region of the blood vessel in the body [4]. The cholesterol amount present in blood starts building up the plaque and it will have hardened with age and time. On the hemodynamics perspective, the

critical zones of plaque buildup are bifurcated zones. At the bifurcated regions, the flow abruptly changes its direction and the flow gets dethatched (separated) and reattach continuously. This creates void near the walls which plays an important role to plaque built up. This may lead to two main diseases called as Stenosis (arterial blockage) and Aneurysm (arterial dilation). Stenosis mainly causes in small arteries such as coronary arteries (arteries supply blood to the heart) and carotid artery (artery supply blood to brain) which creates blockage in the artery and adequate the blood supply. In the heart when blockage reaches 70 percent, it starts causing inflammation & myocardial dysfunction. The arterial blockage is shown in figure 1. On the other hand, arterial bulging called aneurysm typically occurs in major arteries where plaque deposition takes place which results in hardening of arteries. Other type aneurysm occurs inside the brain (intracranial aneurysm) where arteries are small but their bifurcation angle between the daughter arteries are more. This also happens due to hemodynamic disparity near the bifurcated zone. The Wall shear stress (WSS) is maximum at the bifurcation point.

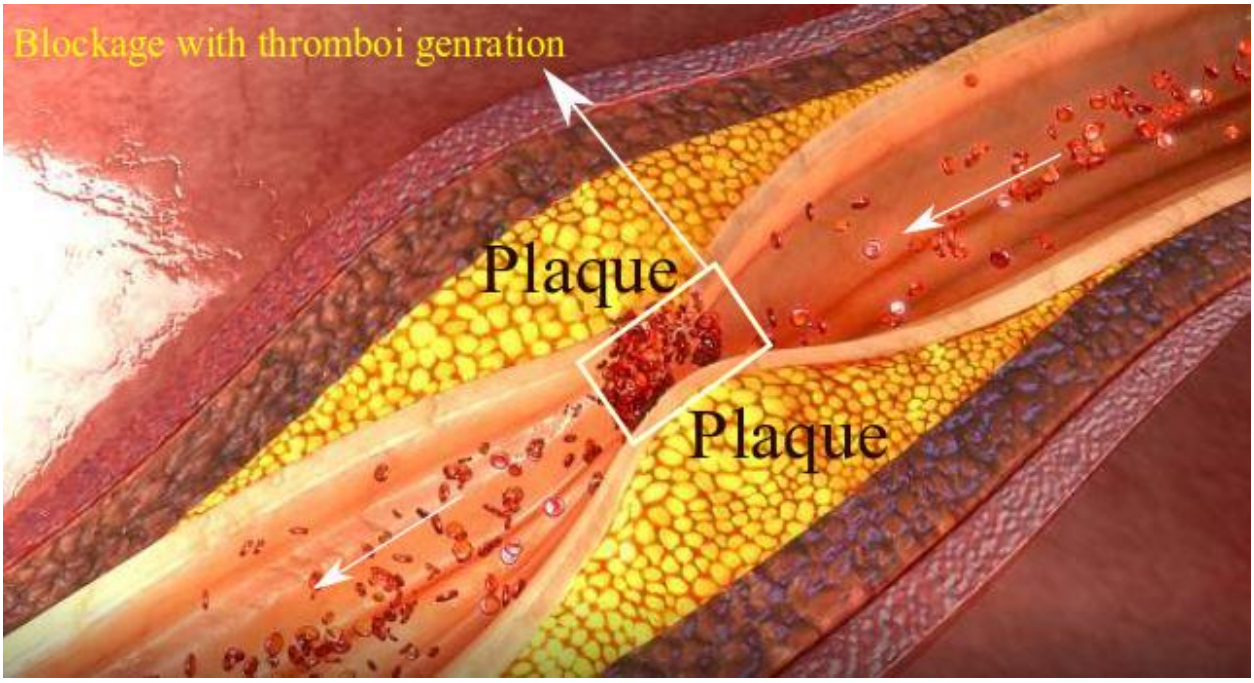


Figure 1: Blockage causing due to atherosclerosis [5]

The cardiovascular disease (CVD) is main cause of the death of Indians. The mortality rate increases to 28 percent in the year 2017 [1]. This disease also affects the developed nation. The mortality from rupture of blood vessels is three times higher in United Kingdom (UK) than United

States (US). During year 2012, 34 in every 100,000 people yearly died from AAA (Aortic abdominal aneurysm) in UK & 9 in every 100,000 died each year in USA [8]. The literature provides the information that the flow abnormality during the blood flow increase the occurrence of disease in the arteries. As mentioned earlier that this effect is more frequent in the bifurcated zone. Our study is to find the reason of the disease and its fatal effects using the engineering computational technique by creating actual physiological environment. For the present study we have selected the abdominal aorta created by patient specific data and for studying the hemodynamics & blood flow fluctuations the saccular and fusiform aneurysms are created for simulation

1.3 Abdominal Aortic Aneurysm (AAA): A brief review

The word Aneurysm is derived from Greek word 'aneurysma' which means 'widening (dilation)' and 'Aneurynein' which means 'to dilate'. According to the Galen the aneurysm is defined as 'an artery having anastomosed (dilation) the affection is called aneurysm' [9]. An aortic aneurysm is defined as the focal dilation of a blood vessel with respect to the actual position [10]. In the human cardiovascular system aorta is the largest blood vessel comprises ascending aorta, aortic arch, thoracic and abdominal aorta. The abdominal aorta is the last & largest end section of the aorta; it supplies blood to the lower region of the body indulging the organs in abdomen & pelvis. The aorta is shown in figure 2. The aorta having thick walls so it can withstand the high blood pressure coming from the heart. Over time the walls of the aorta may weakened due to structural micro molecules integrity degradation of the ECM (Extra Cellular Matrix) such as collagen, elastic and Proteoglycans [11]. This leads to bulging of the aortic wall in outward direction like a balloon. When this condition occurs in the abdominal aorta, this is called as AAA. Figure 3(a) showing the Infrarenal abdominal aortic aneurysm of reconstructed CT (computed tomography) scan and Figure (b) showing different types of aneurysms. In general AAA is defined as increased in dimeter atleast-one and 1.5 times to that of the normal aortic diameter [10,11]. The normal-diameter is approximately 2 cm at the level of renal arteries. 80 % of the AAA occurred between the renal artery and abdominal bifurcation [10], 15 % extend to common iliac arteries. On the hemodynamics perspective the abdominal aortic region is most affected due to the flow distribution and separation at the critical & continuous bifurcation. The blood coming from the thoracic aorta is distributed between the renal and common iliac artery. The two-third of the flow

passes through renal artery (low resistance occur) and one- third of the flow passes through the iliac arteries. Hence, at the rest condition it provides a high resistance and curiously atherosclerotic disease extend along the posterior wall of the relatively straight AA. This phenomenon increases stiffness in the AA and downstream flow applied more pressure on the walls causing dilation of the aortic walls creating aneurysm [13].

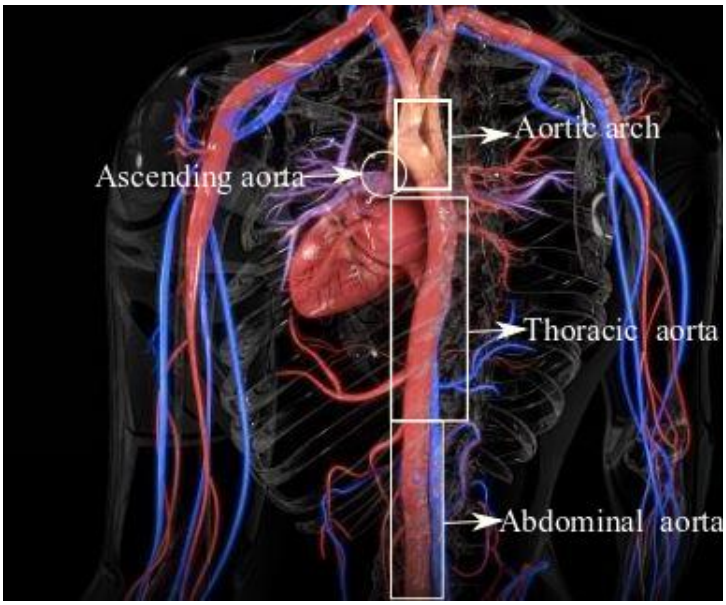


Figure 2: Different parts of Aorta [5]

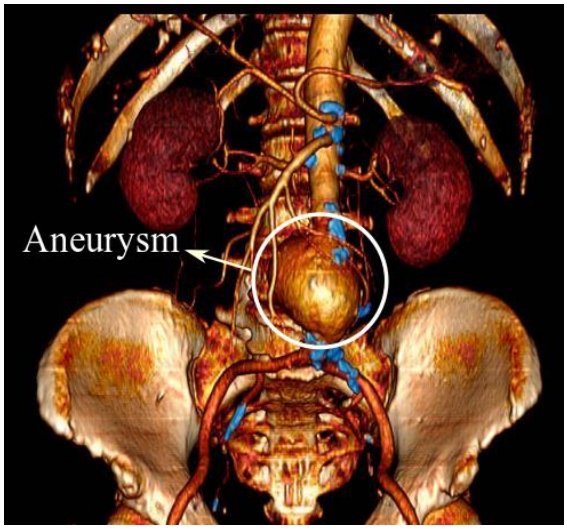


Figure 3:(a) Infrarenal AAA of reconstructed CT scan [5]

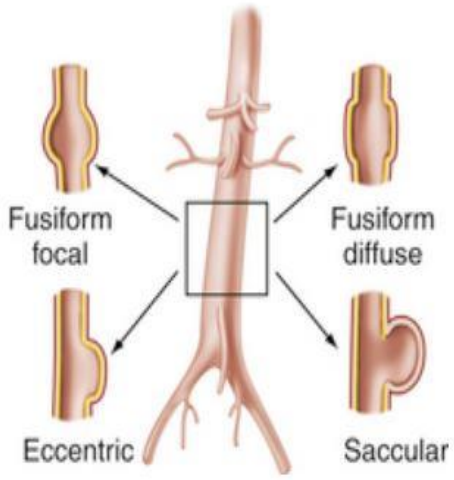


Figure 3:(b) Different types of aneurysm [6]

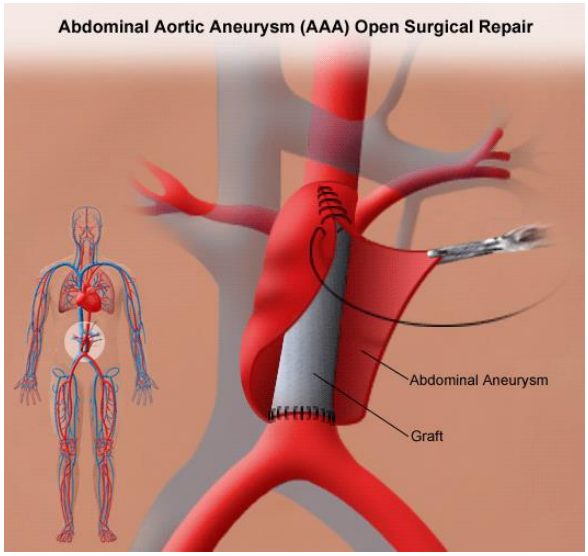
One the historical background, the first aneurysm was observed by ancient Rome in the 2nd century AD. Egyptians (1500 BC) called this 'arterial tumor' whereas Indian ancient surgeon Sushruta

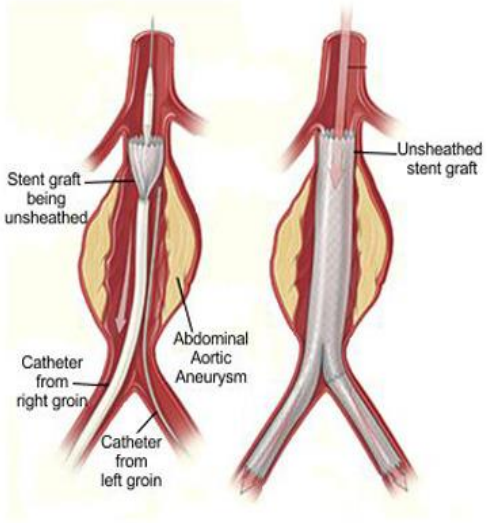
(600-800 BC) called it ‘Granthi’. An ancient Rome surgeon Galen (126-216 AD) first formally described these-tumors as localized swelling which disappears with pressure [12].

Generally abdominal aorta segment diameter greater than 3 cm is considered as aortic aneurysm [10]. This size value of threshold is lower in women than men. For women the lower threshold value is 2.7 cm whereas for men, the value is 3 cm [11]. AAA size greater than 5.5 cm considered as large aneurysm and requires treatment. Usually majority of aneurysm is asymptomatic and it’s an incidental finding. AAA greater than 6 cm needs immediate surgical treatment failing to which it can cause Renal Ischemia, Mesenteric Ischemia, Lower limb Ischemia, hemorrhage and death. In surgical history, first surgical treatment of aneurysm was done by Greek surgeon Antyllus. He tried to treat the disease with proximal and distal ligation (tying) technique. His attempt was unsuccessful but his methods were practiced and improvised with time. The surgery was unsuccessful until 1923. In 1923, Rudolf Matas successfully performed the first aortic ligation on human [12]. Over the time three milestones were achieved in history of aortic surgery:

- Ligation (tying) of the aorta.
- Open surgerical repair (OR) of bulging artery
- Endovascular grafting repair (EVAR) of aorta.

There are two major surgical treatments for aneurysm repair and they are performed according to the condition of patient. First method is open surgery repair (OR) in which the effect region is opened and graft of normal aorta size is fixed inside the aneurysm. The OR method is shown in figure 3 (a). If the patient having heart disease, high risk due to age or other medical complication then second method is used for treatment. This method is called as EVAR (Endovascular aneurysm repair). In this method, the graft is placed inside the aneurysm with the help of guide wire and catheter. The EVAR method is shown by figure 4 (b). In 1952, Voohees inserted the first synthetic graft into rupture AA and in late 1980s Juan Parodi with Julio Palmoz & Hector Barcone-introduced-the-first-catheter-based arterial approach to AAA for medium EVAR technology.





(a) **(b)**

Figure 4: Surgical treatment of Aneurysm [7] (a) Open Surgery Repair (OR) (b) EVAR

1.4 Literature Survey

Major research focusing fluid dynamics in the heart, around valves, and in blood vessels, the hemodynamics origins of vascular disease and blood wall interaction in the microcirculation. There are many researches ongoing in this domain Internationally and nationally in which they generate the morphological data of the models mathematically. There are certain assumptions in the rheology of blood such as Newtonian or Non-Newtonian with taking blood as steady, simple pulsatile in nature or sometimes the real data of the fixed region artery is taken in account and assuming artery walls either rigid (fixed) or linearly dispensable. In this section we are discussing the related works done by the researchers.

S. Aggarwal et al. [10] presented a review paper for understanding the aortic abdominal aneurysm (AAA). This paper is very much basic to understand the medical phenomenon of AAA. The main highlights of discussion are the risk factors (smoking, advanced age more than 60 years, hypertension (high BP) and Caucasian ethnicity), Risk of rupture at different sizes and population

affected with it. Clinical presentation, Diagnosis, Screening, Management and Complications of AAA are presented.

C. Villiard et al. [11] reviews the knowledge in gender difference in AAA and change in the dimension of aneurysm form sex perspective. For the study the reports were collected from e-databases (web of science and PubMed) and the PRISMA group guidelines were followed. On the basis of reports and articles reviewed, it is still inconclusive that gender affects the aneurysm formation and it is sex hormone dependent. Only on the basis of previous case study women are scarce of AAA occurrence and the arterial wall continuous degradation with time are still unknown.

David N. Ku et al. [14] measured the velocity of the fluid by using the LDV (Laser Doppler Velocimetry). He used a Plexiglas model for fluid flow study. The carotid bifurcated model results and the four circumferential distribution are compared at five different axial levels (common carotid aorta, proximal, mid sinus & distal points of ICA and a distal point in ECA). Data of the model were obtained from the 57 patients biplanar angiogram data of age ranging from 34 to 77 years old and from cadavers. He also introduced the concept of OSI (Oscillatory Shear Index) to calculate the wall shear stress. It is very useful to find the flow reversal zones where shear stress changes its direction. From results, he encountered that the OSI is maximum at mid sinus of ICA where plaque generation mostly takes place. The velocity and WSS during the diastolic phase were quite similar to the steady flow condition reported previously.

K. Perkfold et al. [15] done a numerical study of wall shear stress and flow physics (velocity flow profile) in human carotid bifurcated model using 3D geometrical model. He analyzed the parameters by taking the fluid as Non-Newtonian and compared the results with fluid as Newtonian in nature. He used the 3D model of Ku et al. [11]. He applies the similar boundary condition at inlet and flow distribution at ICA and ECA. The Casson's Non-Newtonian fluid flow model had been used under time-dependent condition. The study finally investigated about the complex flow phenomenon at ICA sinus region where the flow separation and flow reversal takes place at outer wall. The comparative study also reveals that there is a minor difference in the flow phenomena and characteristics when fluid is taken rather Newtonian or Non-Newtonian.

K. Perkfold et al. [16] extends their previous study by changing the angle between the bifurcated arteries and checking the dependency of flow parameters with different angles. They used four different models in between three of which only angle of ICA had been changed from 15^o to 40^o with 25^o angle of ECA is fixed and fourth model was taken as equal angle variation of 50^o. The physiological inflow and outflow is similar to the previous work [12]. The fluid again considered as Non-Newtonian using Casson's model. Their results concluded that with increase in angle the flow separation and flow reversal had been enhanced and at the lowest angle difference the flow separation takes place at the terminal end of the CCA whereas in the largest angle difference flow separation develops at the beginning of ICA.

K. Perkfold et al. [17] continues his work on a distensible artery to the investigate the effect of local flow field. A numerical model of blood flow is being developed of human carotid artery to study the mechanical stress on the wall. He takes assumptions for wall displacement and stress analysis increasing linear elastic behavior. The results show that the axial velocity is strongly skewed (sharply parabolic on either side) in the carotid sinus with high velocity gradients at the internal divider wall. The qualitative based comparison between rigid vessel walls and compliant walls had been done. It would be observed that flow separation and recirculation decreases slightly in the carotid sinus region and increase by small in the bifurcation region. In Stress analysis, the value of WSS decreases by 25 percent in the compliant model. They observed that the principal maximum stress at the inner carotid wall shows a complicated field with local high gradients. The value of stress concentration factor is 6.3 in the apex region (max diameter of the sinus). The displacement in the tangential direction is smaller than the normal direction displacement by the order of one magnitude. The maximum displacement is approximately 16 percent of the radius of artery and it is depicted near the bifurcation point.

S. Chakravarty et al. [18] had done his numerical simulation on a two dimensional bifurcated arterial model with symmetrical stenosis on the parent artery. A two dimensional, transient, incompressible, Newtonian, non-linear N-S equations were modified to vorticity-stream function for modelling. The different sizes of stenosis (relevant to actual bio physiology) are analyzed and compared. The comparative results parameter is change in wall shear stress with time. The streamlines and vorticity contours were used to support the comparative results. They concluded

that existence of higher and lower shear stress was due to presence of occlusion. The flow separation may lead to atherosclerosis at region of low shear stress.

S. Les et al. [21] had taken total 43 AAA affected patient specific data and 36 patient data was selected to calculate the mean flow and velocity waveforms for the patient affected with AAA at Supraceliac and Infrarenal region. However, the remaining seven patient model is used to the validate the model. The 36 patient data was interpolated 12 mode Fourier transformation. The final averaged and aligned velocity waveform is used as inflow boundary condition for the present work.

W. E. Merrill et al. [22] had done an experiment to calculate the shear stress of blood with respect to strain rate. The rotational and capillary viscometer was used to solve the purpose. There are two variations of ranges strain rates, according to which it was concluded the blood acts as Newtonian at shear rate was greater than 100 s^{-1} (Newtonian equation is applied) and acts as Non-Newtonian (Casson equation is applied) when shear strain was below 20 s^{-1}. Transition phase is between 20 s^{-1} to 100 s^{-1}. This is used to predict my present model favorable for Newtonian flow.

P M Brown et al. [23] has set an objective to find the risk of rupture with change in AAA size, expansion rate and gender. For this he had selected 476 patients data in between 1976 to 2001 with AAA size greater than 5.0 cm. In this period of time, he had found that 19 patients were on event of surgery, 50 patients were followed with periodic scan until rupture risk become high, 176 patients were free from any rupture risk and 79 patients cause death due to hemoharrage. With the study he found that the risk of rupture is higher in women (3.9 %) than men (1.0 %) in which the size of aneurysm varies from 5 cm to 5.9 cm for both. The rate is four times higher in women than men. He also suggests that the aneurysm size with 6.0 or greater was a fatal condition and requires immediate surgical intervention because the risk of rupture with AAA size 6.0 cm or greater increases to 14.1 % in men and 22.3 % in women.

1.5 Research Gaps

There are many researches ongoing for study on the hemodynamics of the human vascular system. Still there are many unresolved issues in the area of hemodynamic condition in the arteries. Thus there are many unknown facts in the medical research which could be resolved to some extend by computational investigation of this physics. The following are the few research gaps, which this work tries to investigate through computational simulation:

- Most of the computation are done on the artificial models and very few work is done on patient specific model from DICOM image.
- Very few computational investigations have done on the patient specific model of abdominal aortic aneurysm of both types of abnormality.
- Mostly the steady flow is used as inflow boundary conditions.

1.6 Objective of present work

The following are the objectives of this thesis work:

- Creating a patient specific model of AAA from DICOM image.
- Creating the saccular and fusiform case of AAA for comparative study.
- Computational simulation of hemodynamics of the models and suggest the fatal case for the surgery.

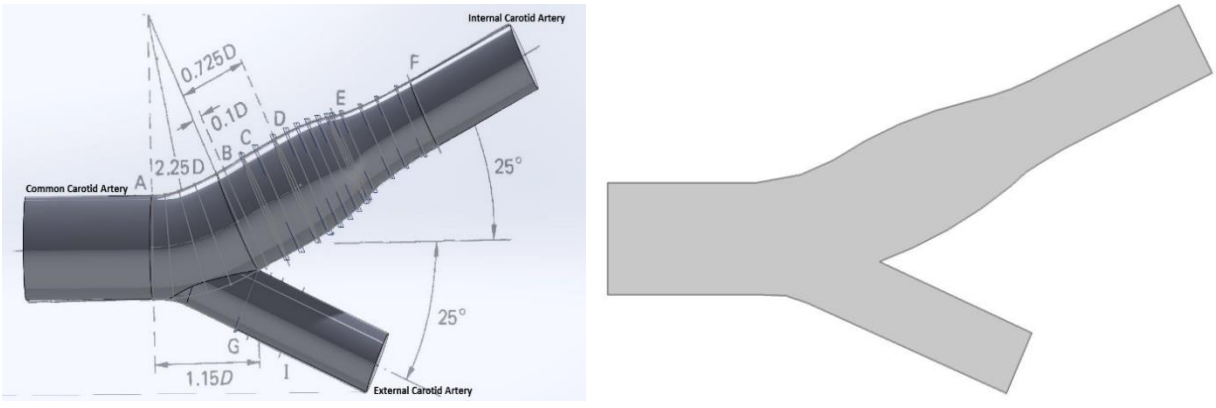
Chapter 2

Modeling Methods

Multiple models are generated by using two modelling softwares they are, SimVascular and SolidWorks. These softwares have their different methods of generating model. In SolidWorks, the model of carotid bifurcated artery taken from Perktold et al. [15], is created by using the tracing method. The carotid bifurcated models are created in 2D & 3D to study the different cases. On the other hand, SimVascular is very extensive software which can create the real patient specific model by using CT/MRI angiogram data.

2.1 Modeling in SolidWorks

As mentioned earlier that 2D and 3D models of Perktold et al. [15] are reconstructed in the SolidWorks. SolidWorks is the modelling software specially designed for generating the solid models. The present model is created by using tracing method. The software has also a feature of importing the models which are generated on other modelling softwares. The most familiar formats for importing the data are STL (STereo Lithography) or STEP (STandard for the Exchange of Product model data) format. The software also helps in live linking the generated model with COMSOL Multiphysics®. The details of linking the software with the software will be discussed. later section. Figure 5 shown explaining the details of the both 2D and 3D model generated in SolidWorks.



(a) **(b)**

Figure 5: Models created in SolidWorks by tracing method (a) 3D model with dimensions (b) 2D model

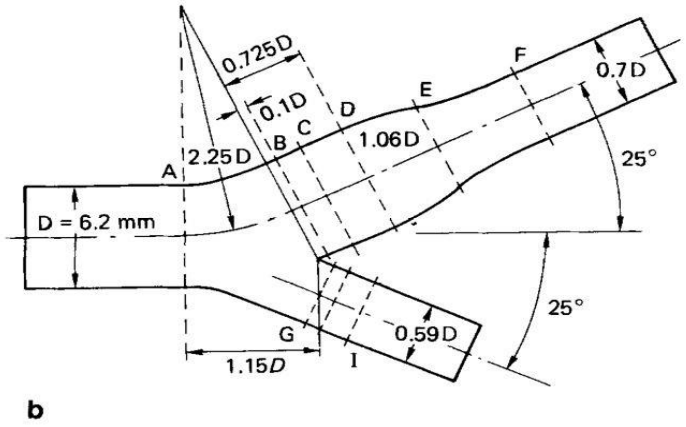


Figure 6: Model taken from Perktold et al. [15] with dimensions

Figure 6 shown above is the reference geometry which is taken from literature by Perktold et al. [15].

2.2 Modelling in SimVascular

SimVascular is an open source software developed by Stanford Medical School. It is a complete packed software which is dedicated for cardiovascular modelling and simulation. In my present work this software is used for generating model using patient specific data from DICOM (Digital Imaging and Communication in Medicine) image. Figure 7 is showing the interface window of the software along with the angiogram. This DICOM image is used for generating the model. The DICOM image is taken from the official website of SimVascular [20].

The processes involved in model generation and reconstruction of vascular geometry are path generation, 2D (contour interpolation) and 3D segmentation and stacking (lofting). The detailed steps involved The detailed modelling process involves the following four steps: (i) Import of DICOM image and path is generated by selecting critical points in the aortic region as shown in Figure 8(a), then (ii) segmentation produces set of points (pixel) for each slice, identifying contour of vascular section. In contour interpolation a suitable curve fitting interpolation is employed on set of points for real approximation of vascular boundary as given in Figure 8(b & c), (iii) The Stacking is done for assembly of 2-D interpolation curve on each slice to get the 3-D reconstruction of vascular geometry as shown in Figure 8(d) and then finally (iv) The model is created and the 3D model was exported to STEP (STandard for the Exchange of Product) format. The complete

procedure from DICOM images to 3-D modelling is done by using an Open-Source package MITK (Medical Imaging Tool Kit)

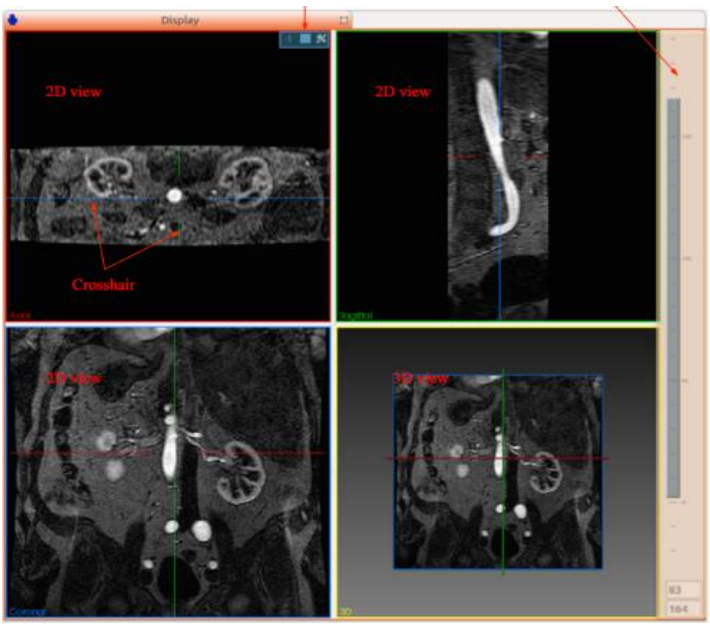
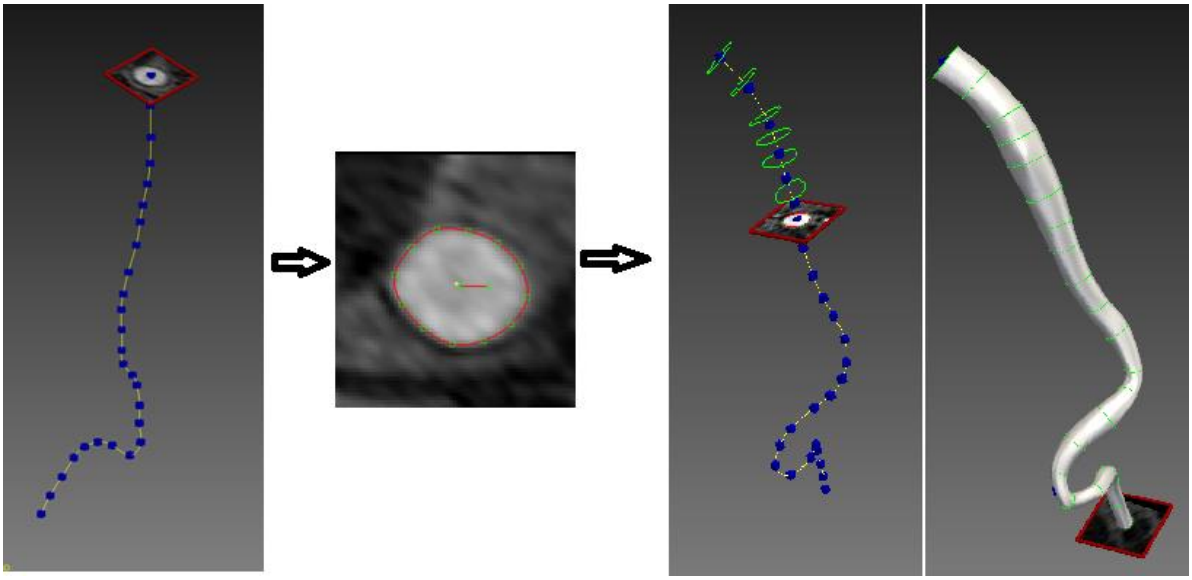


Figure 7: SimVascular GUI Window with patient specific angiogram [20]



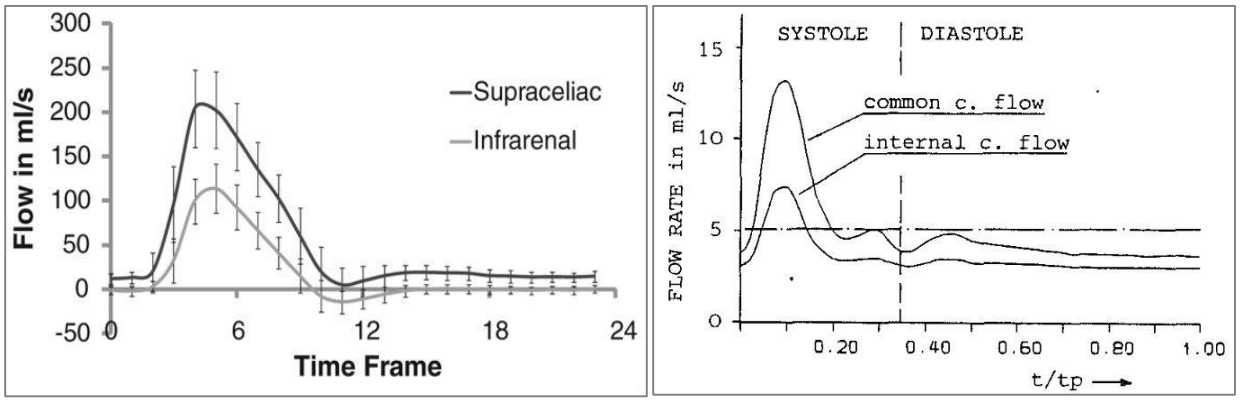
(a) Path Generation (b) Contour Interpolation (c) Segmentation (d) Stacking

Figure 8: Steps to reconstruct vascular from DICOM images [20]

2.3 Mathematical Modelling and Boundary Conditions

The Pre-Processing, Meshing, Simulation and Post-Processing are done on COMSOL Multiphysics. COMSOL Multiphysics is finite element analysis (FEA) based software which can simulate the fluid flow (Laminar, Turbulent, Single phase flow, Multiple phase flow etc.), Typical flow parameters and stresses on critical regions. The flexibility of the software is to couple the fluid flow and structural analysis simultaneously on a single window. This gives a benefit to solve the FSI (Fluid Structure Interaction) problems.

The Simulation is done on 2D & 3D models and patient specific generated model by means of importing model from SolidWorks. It is already explaied initially in this chapter that importing in COMSOL Multiphysics is done by the means of Live link feature in collaboration with SolidWorks. This feature has its own benefits; it enables us to import the geometrical model without converting it into any particular format. After importing the model, the Single phase laminar flow module is selected by adding the physics. The pulsatile inflow data is taken from Perktold et al. [15] for the common carotid artery (CCA) and from Andrea Les et al. [21] for abdominal aorta (AA). The data is imported by using the interpolated function. The pulsatile inflow data for CCA and AA is shown in figure 9.



(a) **(b)**

Figure 9: Pulsatile inflow waveform in ml/sec (a) for abdominal aorta (supraceliac inflow waveform) [21] and (b) for Common carotid artery [15]

In the laminar flow condition, the fluid properties are set according to the blood material having density of 1060 kg/m^3 and dynamic viscosity of 0.004 Pa s. The fluid is taken as Newtonian and Incompressible in nature. The blood rheology is Non-Newtonian (Shear Thinning) fluid but the reason of selecting the blood as a Newtonian fluid is due to the nature of blood in larger arteries. The blood will act as Newtonian fluid in larger arteries whereas it behaves like Non-Newtonian

in arteries with lesser diameters such as arterioles. The shear rate value defines the nature of blood in the artery, if the value of shear rate exceeds 100 s^{-1} then it behaves like Newtonian [14].

The Inflow boundary condition is velocity based and pulsatile (transient) in nature. The value for the inflow is calculated from interpolated function created initially in the definition feature. The outflow boundary conditions are pressure based with given user value of 100 mmHg. The Navier stokes equation (1) and continuity equation (2) defines the laminar fluid flow.

$$\rho \frac{\partial u}{\partial t} + \rho(u \cdot \nabla)u = \nabla \cdot [-pI + \mu(\nabla u + (\nabla u)^T)] + F \quad (1)$$

$$\rho \nabla \cdot u = 0 \quad (2)$$

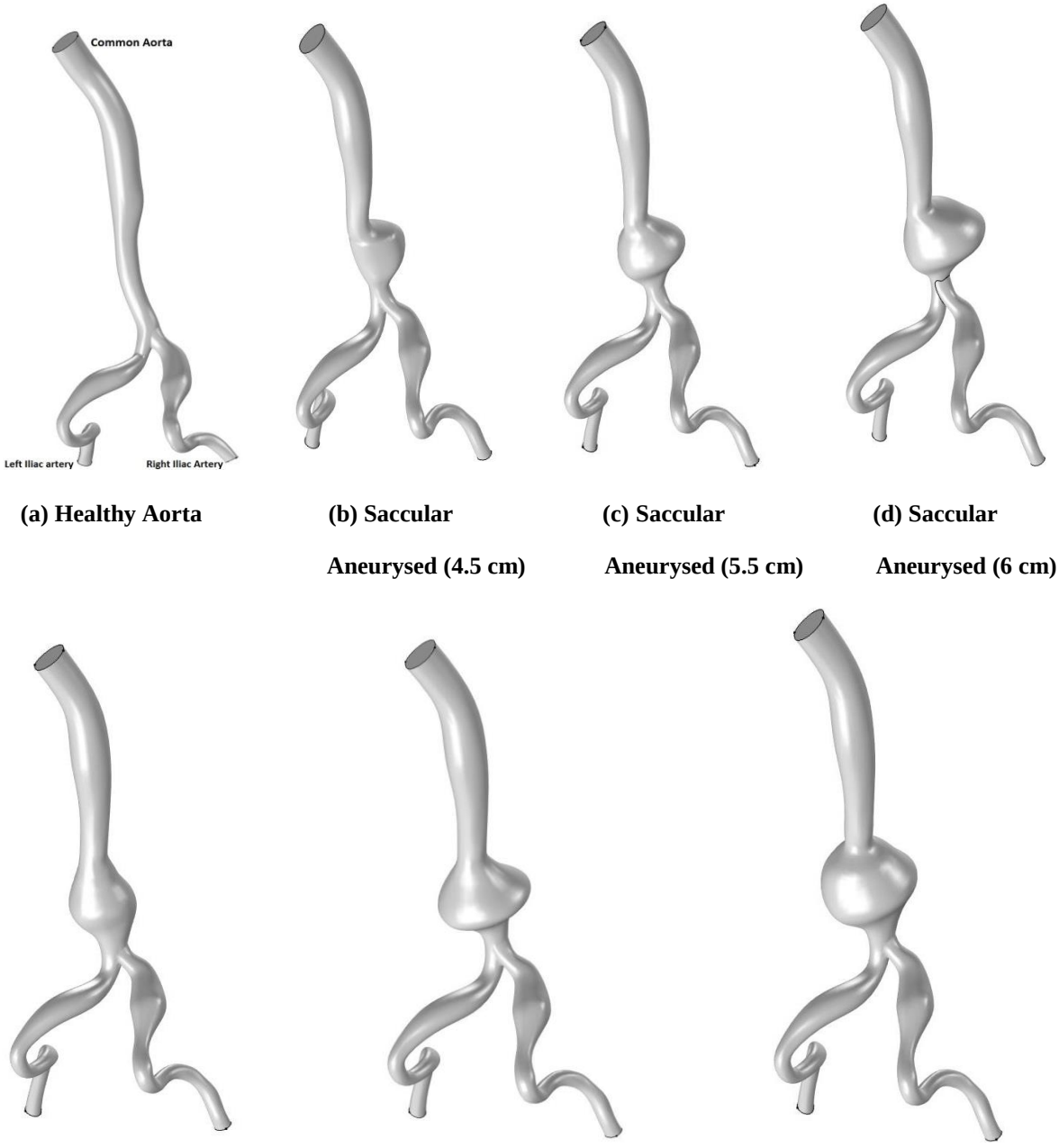
Where ∇ is the vector del differential operator, ρ is the density of the fluid, μ is the dynamic viscosity of the fluid, u is the velocity space vector, T is the transpose matrix, I define the identity matrix, p is the pressure and F is the body force acting on the fluid. The laminar fluid flow is considered because the Reynolds Number (Re) is less than 2000 for the flow.

The walls are being treated as rigid and no slip boundary condition i.e. the walls will not deform during the flow and the velocity of the flow at the wall is zero. Literature study encountered the result that compliance effect of walls doesn't make any significant difference in values of the flow parameters. The initial values are set to zero and reference pressure is set to 1 atm. A time-dependent study is applied differently for the comparative study and the flow through abdominal aorta. The time step 0.0001 s is taken for the comparative study and 0.01 s for AA flow simulation.

2.4 Geometry of the model

As mentioned in earlier sections study, initially the simulation is run on the 2D model for the comparative study with Perktold et al. [15] and after getting favorable results the simulation is replaced by same 3D geometry model. All settings of physics (laminar fluid flow), material, boundary conditions and time dependent study are unchanged. The modeling in the SolidWorks is preferred over the COMSOL because SolidWorks is more flexible in generating the models. The image tracing feature helps to create the model more accurately. Apart from SolidWorks the model of the aorta is created in the SimVascular form the DICOM image. There were different sizes of aneurysms are created in the aorta to check the critical stage of disease. As it is discussed earlier that only one-third of blood flow coming from thoracic aorta passes through the legs and during rest time it offers high resistance across legs blood vessels [13]. The continuous deposition of

plaque decreases the strength of aorta. When these two abnormality occurred simultaneously, it dilates the artery. Also, S Aggarwal et al. [21] encountered that approx. 80 percent of aortic aneurysm occur between the renal artery and aortic bifurcation. The aneurysm affected patient cases were selected from the 36 patient data provided by Andrea Les et al. [21] and then selected cases of saccular and fusiform aneurysm is modelled after that all the models were exported in



(a) Healthy Aorta **(b) Saccular Aneurysed (4.5 cm)** **(c) Saccular Aneurysed (5.5 cm)** **(d) Saccular Aneurysed (6 cm)**

(e) Fusiform Aneurysed (4.5 cm) **(f) Fusiform Aneurysed (5.5 cm)** **(g) Fusiform Aneurysed (6 cm)**

Figure 10: (a) Patient specific healthy aorta (b)-(g) diseased aorta with different sizes aneurysm

STEP format. The sizes of the aneurysms are 4.5 cm, 5.5 cm and 6 cm. The aneurysm size in model is taken by reference of Brown et al. [23]. The model with size 4.5 cm is the under the rupture risk whereas 6 cm having high risk of rupture. The value with 5.5 cm is selected as threshold value [23]. Once again SolidWorks plays an important role in importing the CAD (Computer Aided Design) model in COMSOL. The Live link with SolidWorks feature in the COMSOL help in importing the model for simulation. The normal healthy aorta having approximately 4.5 cm^2 inlet cross-sectional area and two common iliac arteries having cross sectional areas of 1.06 cm^2 (left iliac) and 0.97 cm^2 (right iliac). Due to irregularity in the dimensions of inlet and outlets, they are taken in terms of cross sectional area. The different models of aneurysm are shown in figure 10.

2.5 Meshing

Once all the models were imported in the COMSOL and simulations are performed. The grid independency test is done on the model of Perktold et al. [15]. The model is simulated for one cardiac cycle at the different meshes until the convergence is achieved. The wall shear stress at the peak flow rate and average wall shear stress are calculated at the upper sinus region in the middle. The value of average shear stress is calculated at maximum value of flow rate at 0.1 pulse time.

The mesh size setting used as default predefined by the COMSOL for the fluid dynamics as a physics setting. The mesh size setting for the domain is initially set for normal size and then transformed to extremely fine setting to check the variation in the results. The boundary wall layer is applied on the edges of the domain to generate the finer mesh at the walls help to obtain the precise results of wall shear stress. The final mesh size settings for the domain in figure 11 ranges from 0.281 mm to 0.00324 mm with 1.08 element growth rate in total 18751 elements and boundary mesh size ranges from 0.145 mm to 4.32e-4 mm. The gradient of the velocity is highly variable at the wall and it becomes constant as move towards the center of the domain. All the models are using the combination of tetrahedral and hexahedral elements. Hexahedral elements were used at the boundaries of the domain because they better calculate approximate shear stresses. Hence these elements are used at the walls where the wall shear stress being used to calculate. The remaining domain will use the tetrahedral elements because this element is more flexible in generating accurate mesh in complex 3D geometries. The final mesh size settings for the geometry

of the aortic model shown in the figure 12 ranges from 0.432 mm to 0.0467 mm with 344398 domain elements and 19524 boundary elements.

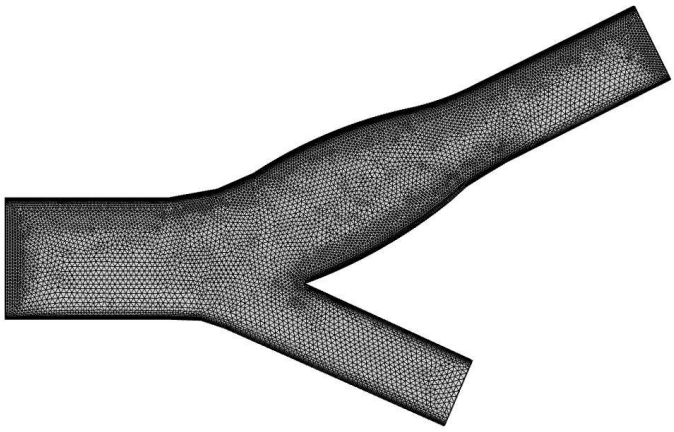


Figure 11: Domain and Boundary mesh for 2D model

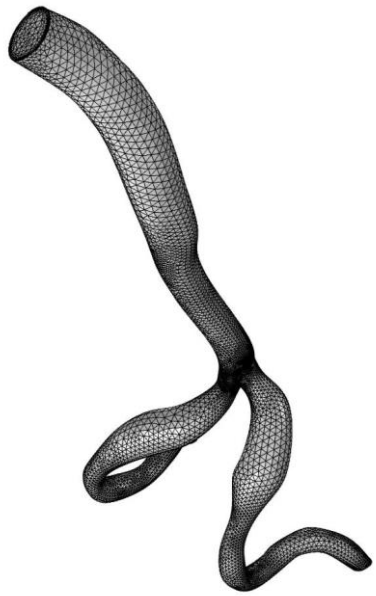


Figure 12: Domain and Boundary mesh for 3D aortic model

Chapter 3

Comparative Study

This section deals with the comparative study of the results of present work and Perktold et al. [13]. The comparative study requires validation of the generated results. The study was initially done on 2D model and then results were validated on the 3D model. The 2D model are selected since they are computationally efficient. Comparative study has been done with wall shear stress on the upper layer of the domain boundary. In present work results are computed for multiple cardiac cycles for 2D and 3D models. This study mainly focuses on the qualitative analysis. The result of the 2D model at typical point D from figure 6 shown in figure 13.

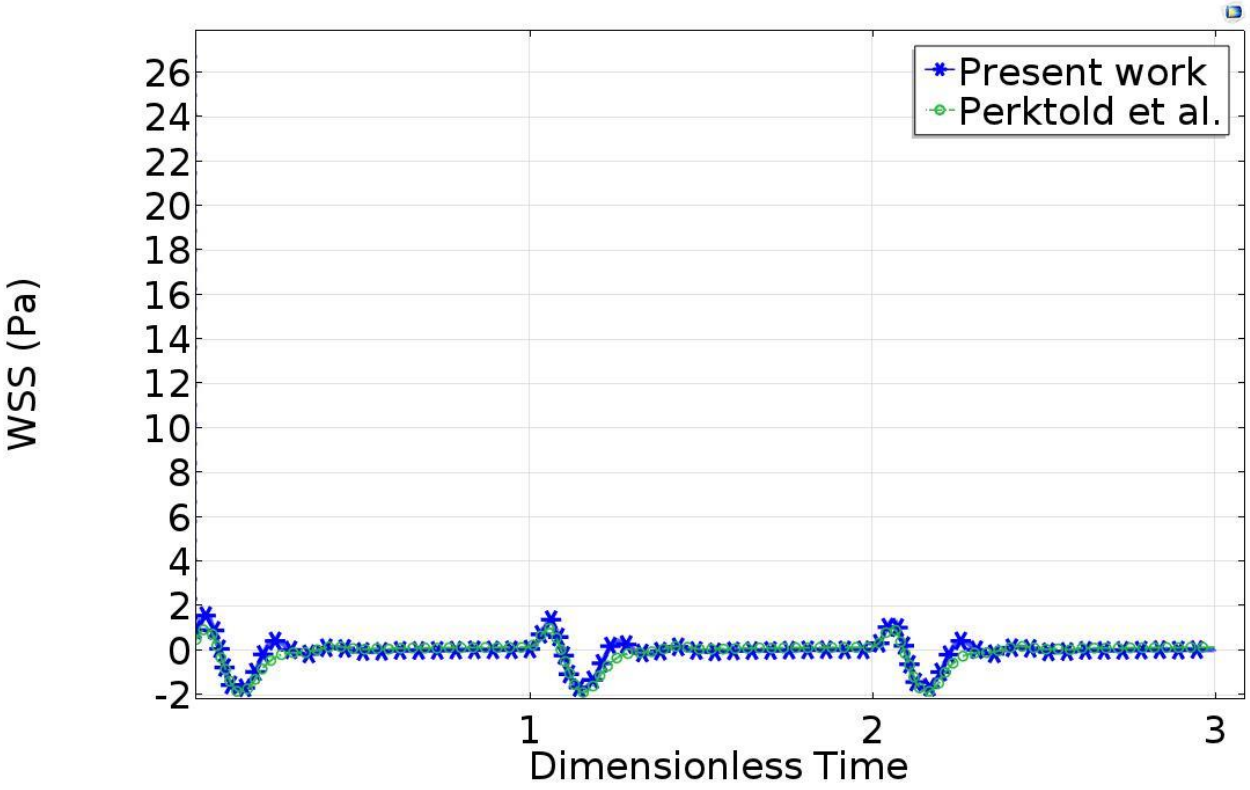


Figure 13: Variation of Wall Shear stress with time at point D

It has been observed from above figure 13, the trend of the graph of the present work is following the same trend of the previous work. For the first cardiac cycle there is difference between the value reported by the Perktold et al. [13] and the present work at $t/t_p = 0.065$. The present value of wall shear stress value is greater than previous reported value but for the second and third cycle they match quite accurately. The reason for this behavior is due to undeveloped flow in the first

cycle. Typically, in the pulsatile flow the intermittent nature creates the arbitrary behavior but after reaching the steady state, when the flow become fully developed, it behaves as a steady in nature. Figure 13 also authenticate my previous comment about fully developing nature of flow. As we can observe the results coming closer with increase in cardiac cycle.

Another point of observation is the value of wall shear stress at $t/t_p = 0.3$. The WSS at that point obtained from our work is higher than the previous results for every cardiac cycle. This may be due to slightly difference in the geometries and meshing technique between our model and Perktold et al [15]. Thus the small error in the geometry orientation can vary the actual results.

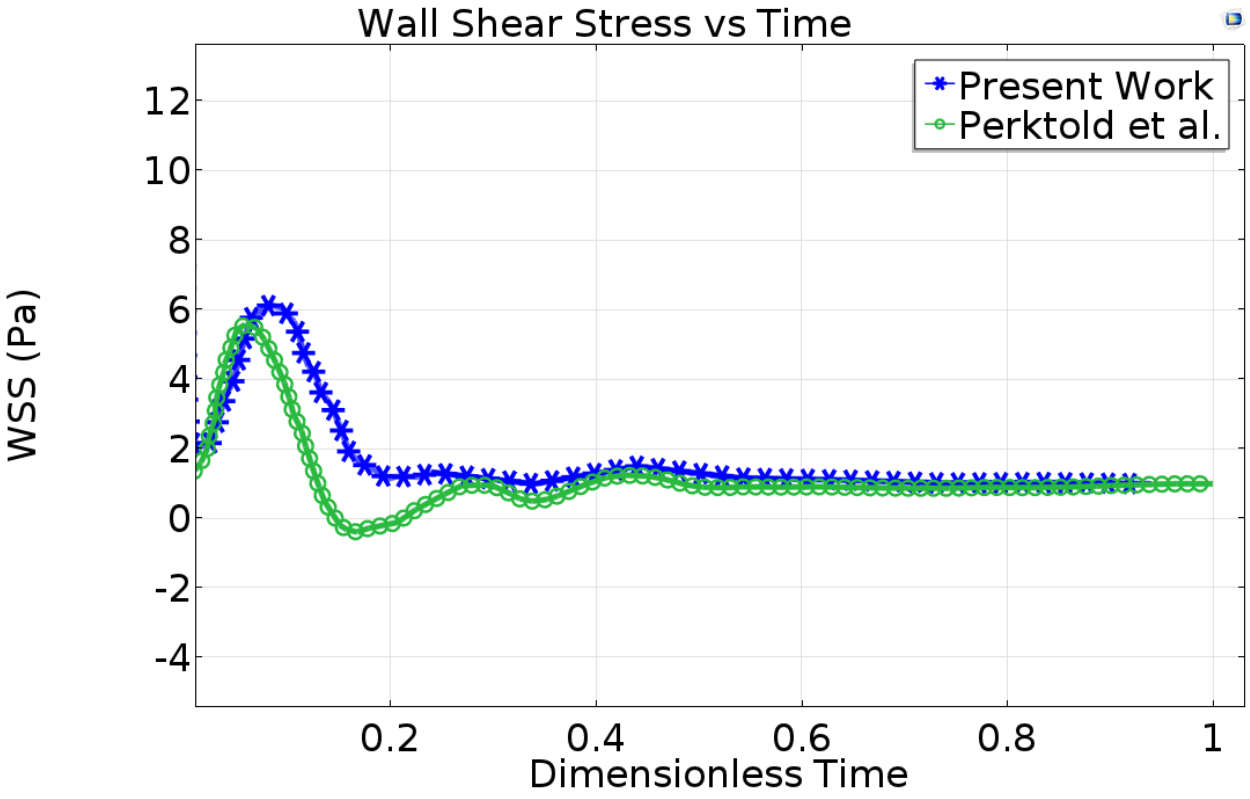


Figure 14: Variation of Wall Shear stress with time flow at position A

Figure 14, 15, 16 & 17 indicates the qualitative comparative study for 3D model. The wall shear stress is calculated at points A, B, D and F as mentioned in figure 6.

Figure 14 indicates the wall shear stress at the position A. This position is on the common aortic region. The generated result shifts laterally during systole of the flow. The peak value of shear stress is somehow being closer to each other. The possible reason for the shift is due to the geometrical error and the mesh variation from actual model. The Perktold et al. [13] model was

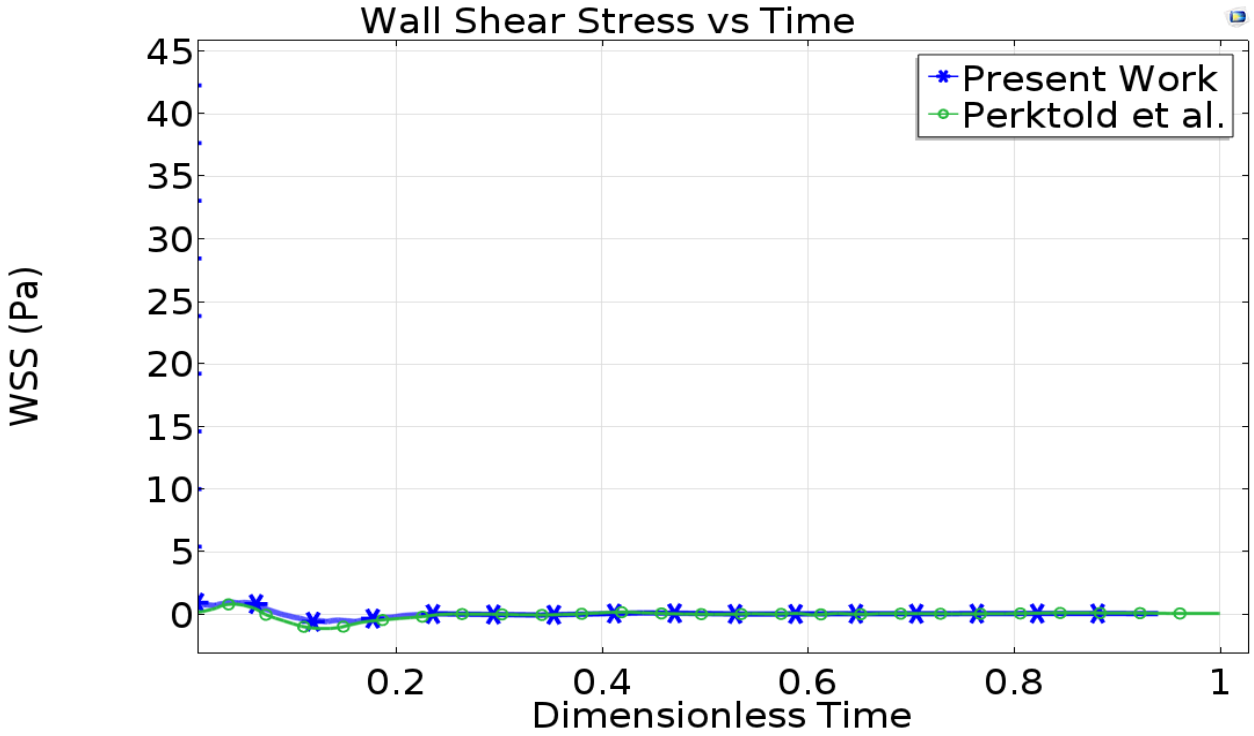


Figure 15: Variation of Wall Shear stress with time flow at position B

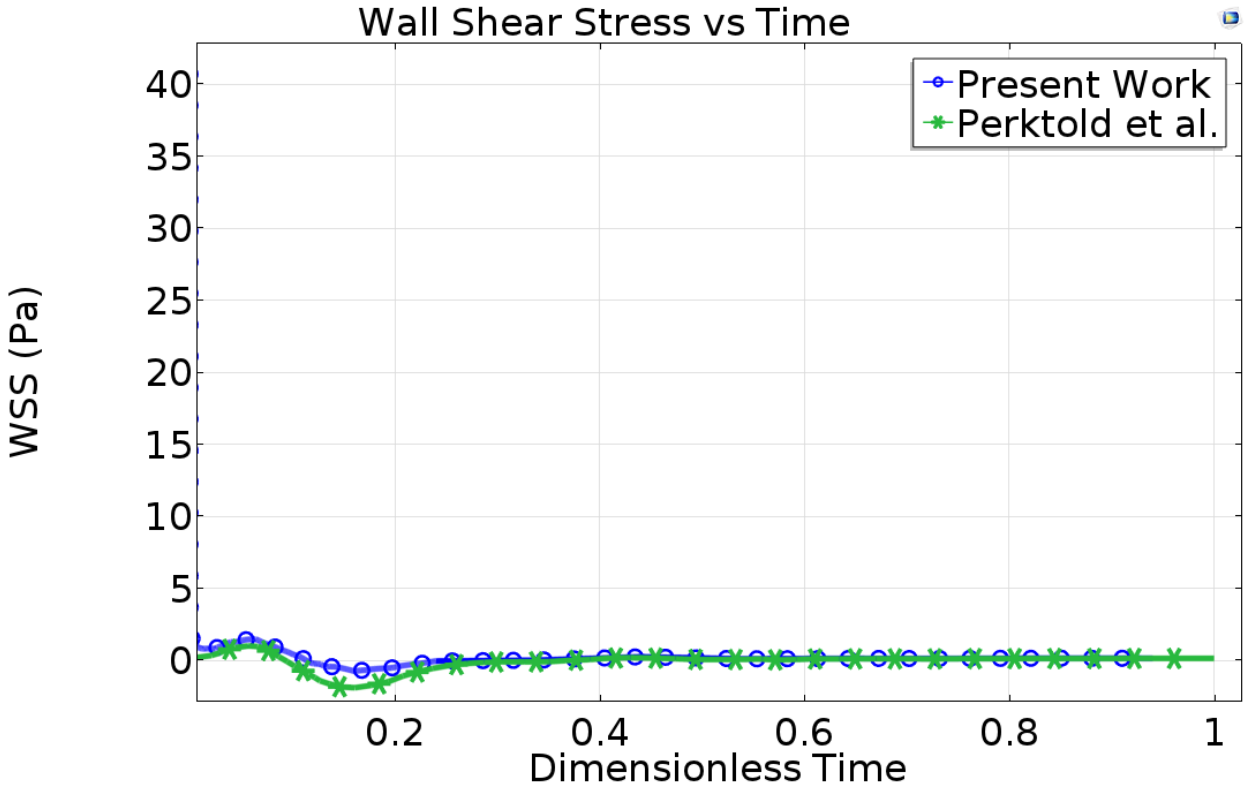


Figure 16: Variation of Wall Shear stress with time flow at position D

having quadrilateral meshing whereas the tetrahedral (at remaining domain) and hexahedral (at walls) meshes are used for the current model.

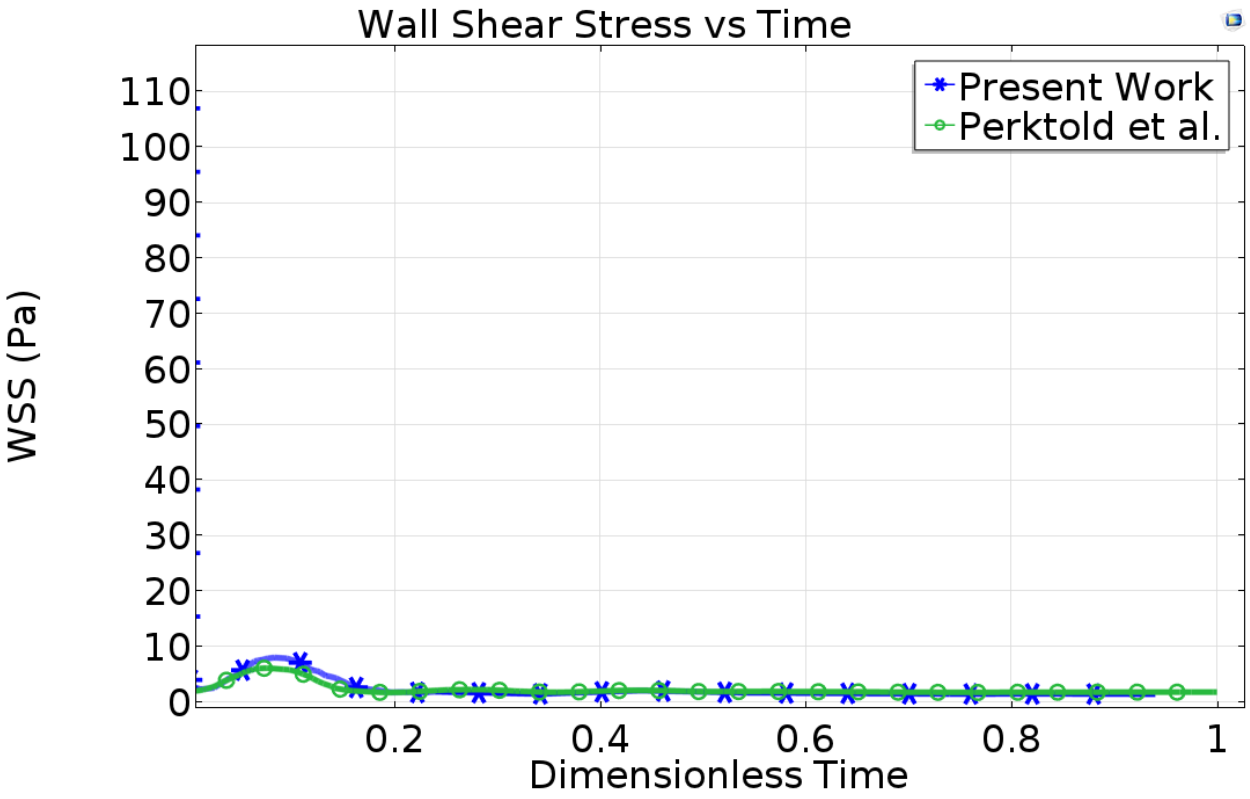


Figure 17: Qualitative Comparative results of Wall Shear stress with flow at position F

Figure 15, 16 & 17 indicating the comparative results of the shear stress at positon B, D and F (Fig. 6) respectively. The position B is proximal to the sinus of the Internal carotid artery (ICA), position D is at the mid sinus of the ICA and position F is the distal point form bifurcated point at ICA (Fig. 6). It has been observed from the respective figures that the value of wall shear stress is lesser than the previous results at peak systolic time. However, the result are overlapping at the diastole phase of the pulsatile flow.

It should be observed from figures 15,16 &17 that the peak value of wall shear stress occurs at different time position. For position B it is at $t/t_p = 0.13$, for position D it is at $t/t_p = 0.16$ and for the position F the time is given by $t/t_p = 0.085$. There is a slight difference in our results and previous reported values. However, in general the data is matching for most part of the cardiac cycle. Hence, by the above discussion we can conclude that the trend followed by the generated results is similar to the literature data.

Chapter 4

Results & Discussions

This section deals with the comparative study of the results of healthy abdominal aorta with pathological condition (aneurysed abdominal aorta). The graphs and contours shown in this chapter are mainly focusing on the fatal disparity at different stages of abdominal aneurysm growth.

4.1 Comparative analysis velocity flow phenomenon

In this section the velocity profile of the flow at the aneurysed section (Fig. 10 b, c, d & e) of the abdominal aorta is plotted at different pulse time. The pulse time at which velocity profiles are plotted are $t/t_p = 0.18$ (peak systole flow), $t/t_p = 0.36$ (mid systole deceleration flow), $t/t_p = 0.46$ (minimum flow), $t/t_p = 0.77$ (mid diastolic flow). The mid line is drawn along the diameter of mid cross section approximately 4.5 cm above the bifurcation point plane.

Figure 18 shows the velocity profiles of the abdominal aneurysm in healthy condition (Fig. 10 a). The maximum flow rate is approximately 1.4 m/sec at $t/t_p = 0.18$ (peak systole) and it is varying with pulse cycle. The average velocity of this flow is approximately around 0.8 m/s or 80 cm/s. These values validate our results with the published data in reference [6]. According to the reference [6], the average velocity of a healthy adult abdominal aorta ranging from 70 cm/s to 100

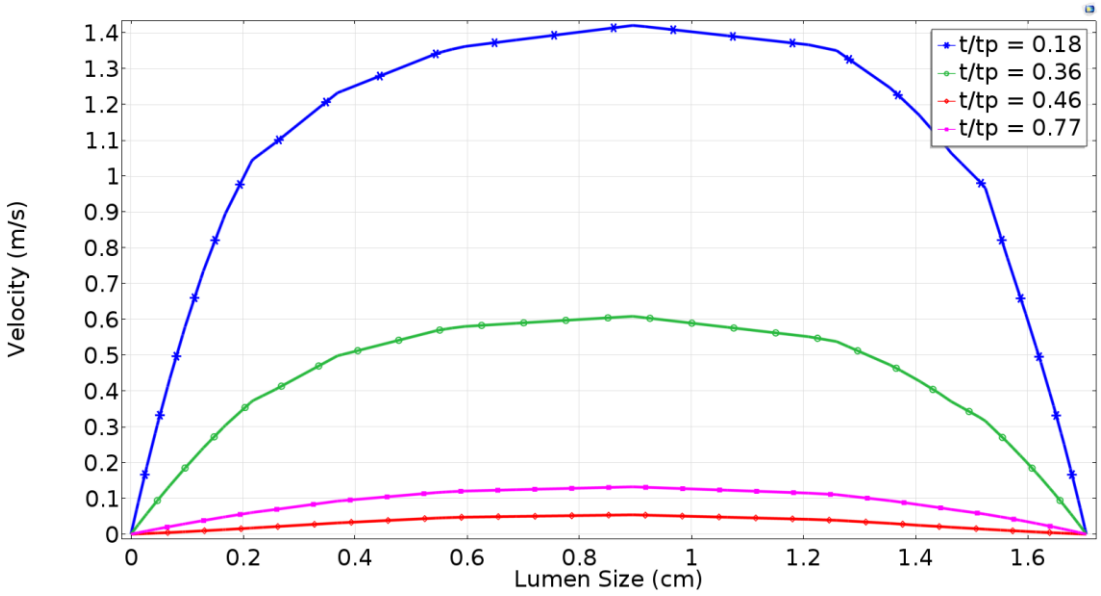


Figure 18: Velocity profile of healthy abdominal aorta

cm/s. Figure 19 indicates the velocity profile of the saccular aneurysm of size 4.5 cm (Fig. 10 b). With the increase in the size of the aneurysm, the value of maximum velocity starts decreasing.

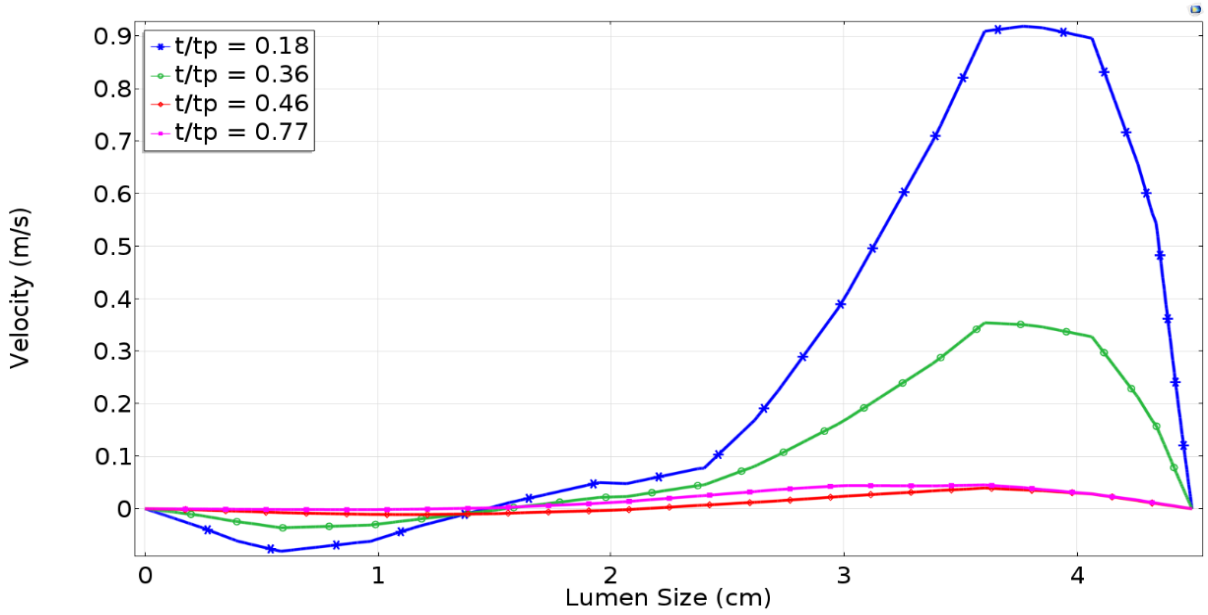


Figure 19: Velocity profile of aneurysm with saccular size 4.5 cm

The value of peak velocity reaches to 0.9 m/s or 90 cm/s approximately for saccular aneurysm size of 4.5 cm. As it is noticed from the figure 19 that the velocity profile for the case is positively skewed towards right. This means the flow recircirulation is formed where the velocity changes its direction from positive to negative and as the flow starts approaching to distolic phase the length of recirculation get increased. For the mid systolic deceleration, the recirculation length increases with 0.5 cm approx at $t/t_p = 0.36$ and it is maximum for the end of distole at $t/t_p = 0.46$. This results are similar with the result published by Perktold et al. [15]. According to their observation the flow starts separating at the beginning the carotid sinus. Recirculation length start developing from proximal location of carotid sinus and its length increased as flow approaches to distolic phase. The velocity of flow is also skewed towards the inner wall of ICA at sinus. The maximum value for the negative velocity is 0.02 m/s at $t/t_p = 0.18$ sec (peak systole) at the saccular region.

The flow phenomenon for remaining two cases with saccular aneurysm of size 5.5 cm (Fig. 10 b) and 6 cm (Fig. 10 b) is also following the similar trends as followed by the previous case. The maximum flow velocity starts decreasing as lumen size of aneurysm is increased.The maximum flow velocity for saccular size of 5.5 cm (80 cm/s approx) and for size 6 cm the value of maximum velocity is again decreased

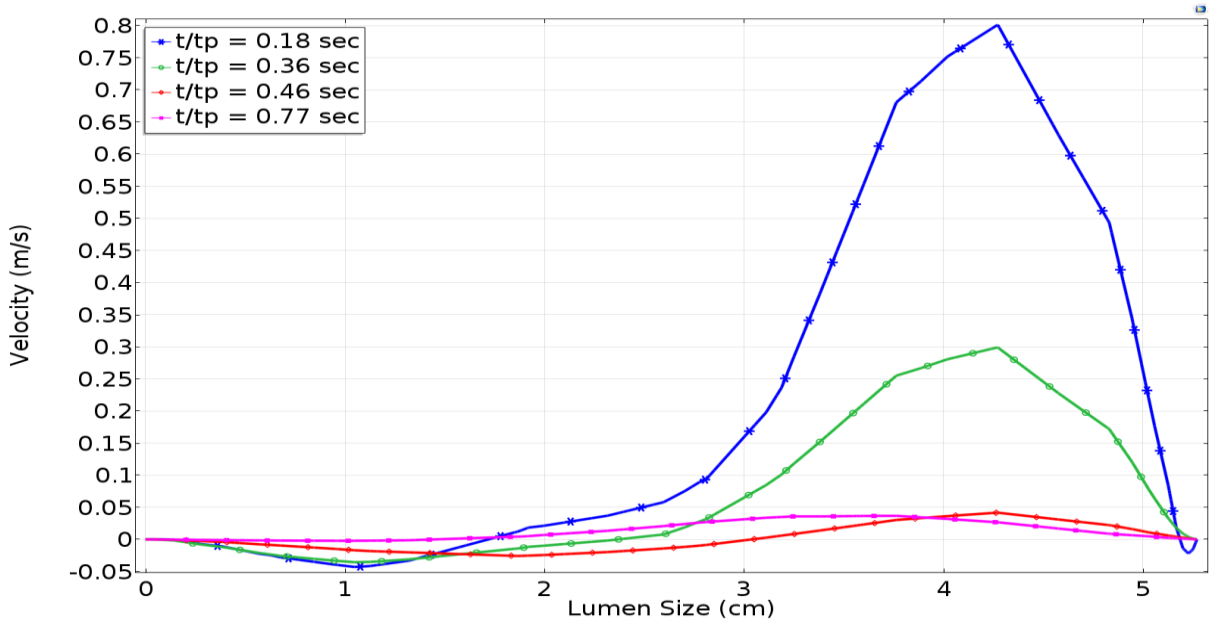


Figure 20: Velocity profile of aneurysm with saccular size 5.5 cm

reached to 70 cm/sec. However, the maxmium negative velocity increases as the size is increased from 4.5 cm to 6cm (figure 19 to 21). The decrease in maximum flow velocity is occuring due to increase in the lumen size and the reason for loss in maximum velocity is filling of blood in the dilated aorta. Correspondingly, the increase in the negative velocity is due to

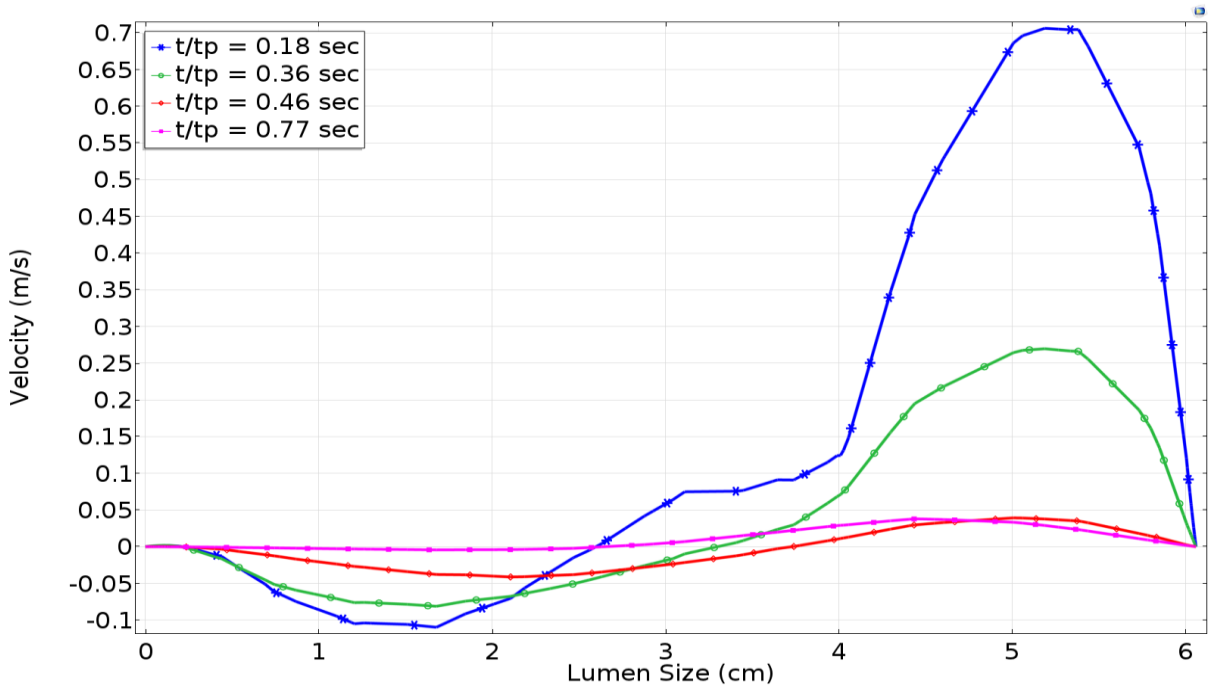


Figure 21: Velocity profile of aneurysm with saccular size 6 cm

the increase in flow recirculation with size in the aneurysed region & it is known that higher recirculation leads higher velocity gradients in opposite direction . This recirculating intensity is maximum for size of 6 cm. The length of recirculation is maximum at the end systolic decelerartion.at $t/t_p = 0.46$. Figure 20 & 21 showing the velocity profile for saccular aneurysm 5.5 cm and 6 cm respectively.

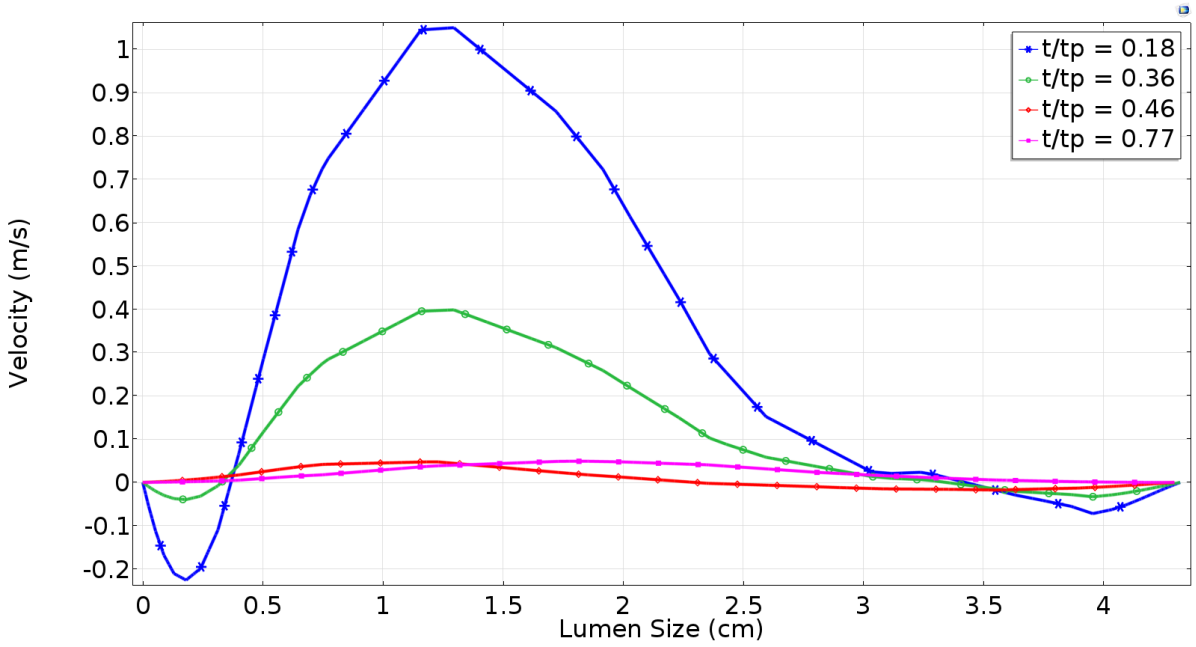


Figure 22: Velocity profile of aneurysm with fusiform size 4.5 cm

Figure 22 indicating the velocity profile for the fusiform case of aneurysm size 4.5 cm (Fig. 10 e). The figure 22 clearly indicating that the recirculation is created on the opposite ends (anterior and posterior) of aneurysm. The velocity profile is more skewed at posterior position which is opposite side to the aneurysm. The maximum velocity of the flow reaches at 1.1 m/s in peak systolic phase. The phemomenon of flow is same as the flow phenomenon in the saccular aneurysm case. Figure 23 and 24 indicate velocity profile for fusiform aneurysm with size 5.5 cm (Fig. 10 e) and 6 cm (Fig. 10 f) respectively. The maximum flow for the fusiform 5.5 cm and 6 cm are 0.82 m/s and 0.7 m/s respectively. The maximum negative velocity is again increased form 0.2 m/s to 0.25 m/s with size of aneurysm form 4.5 cm to 6 cm. Hence, the trends of changing in maximum and minimum flow velocity are similar for both sacccular and fusiform cases.

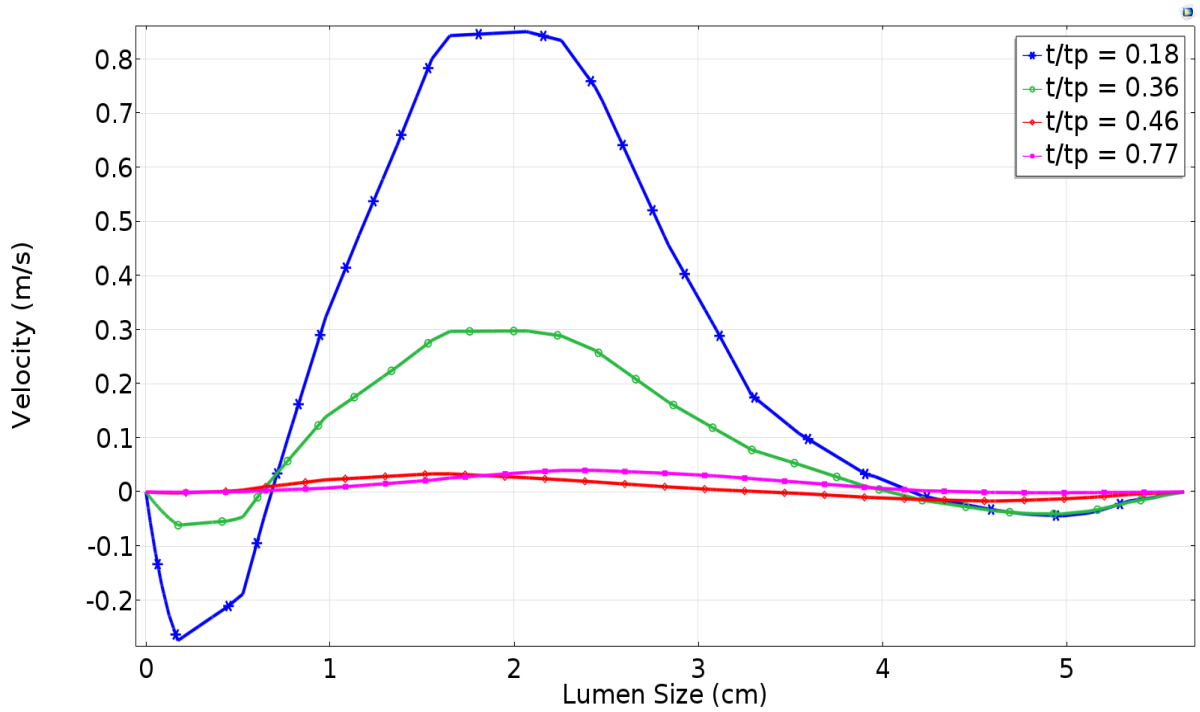


Figure 23: Velocity profile of aneurysm with fusiform size 5.5 cm

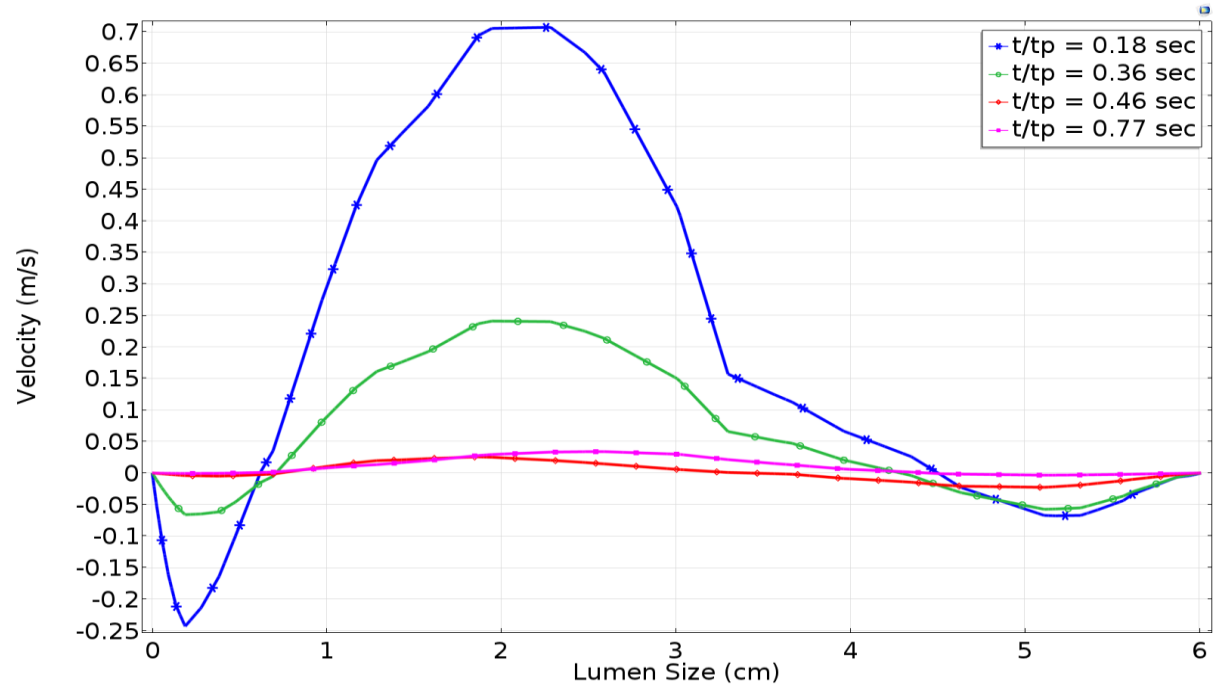


Figure 24: Velocity profile of aneurysm with fusiform size 6 cm

4.2 Comparative analysis of streamline flow

This section discussed about the streamline distribution in the aneurysed section. The comparison is done for maximum (t/t_p = 0.18) and minimum flow (t/t_p = 0.46) condition. The streamlines are indicating the flow in the aneurysed section. It has been clearly observed that the velocity is higher at the peak systolic flow and lower at minimum flow. The recirculation zone length is maximum at the t/t_p = 0.46. As in the previous section, we observed that the velocity profile is single sided

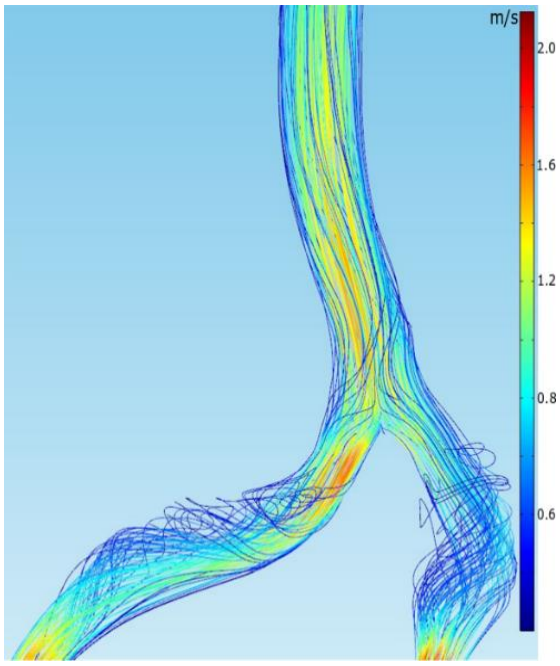
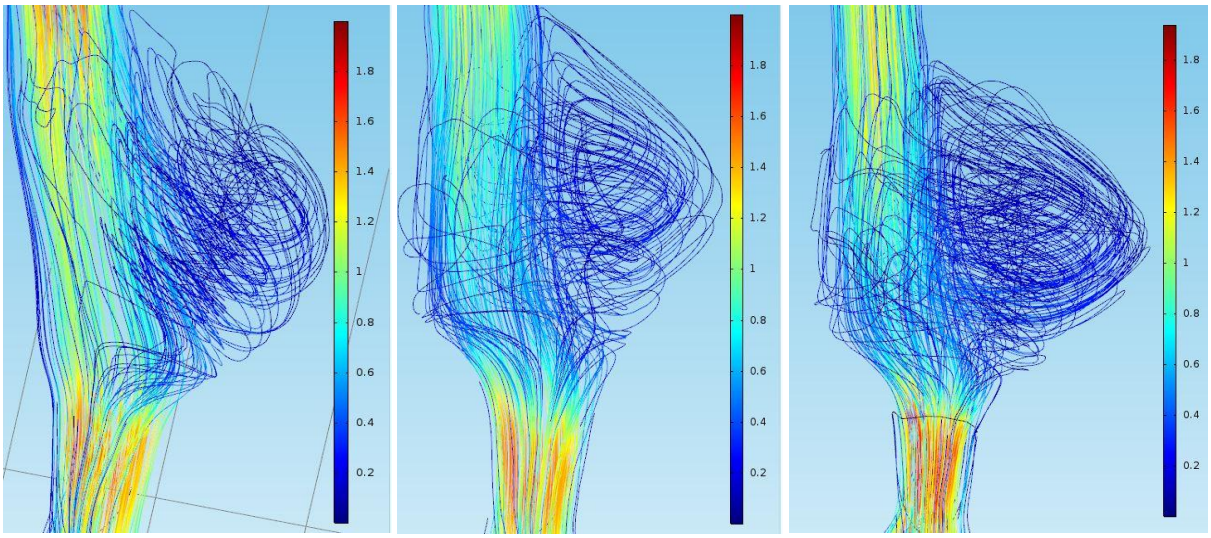
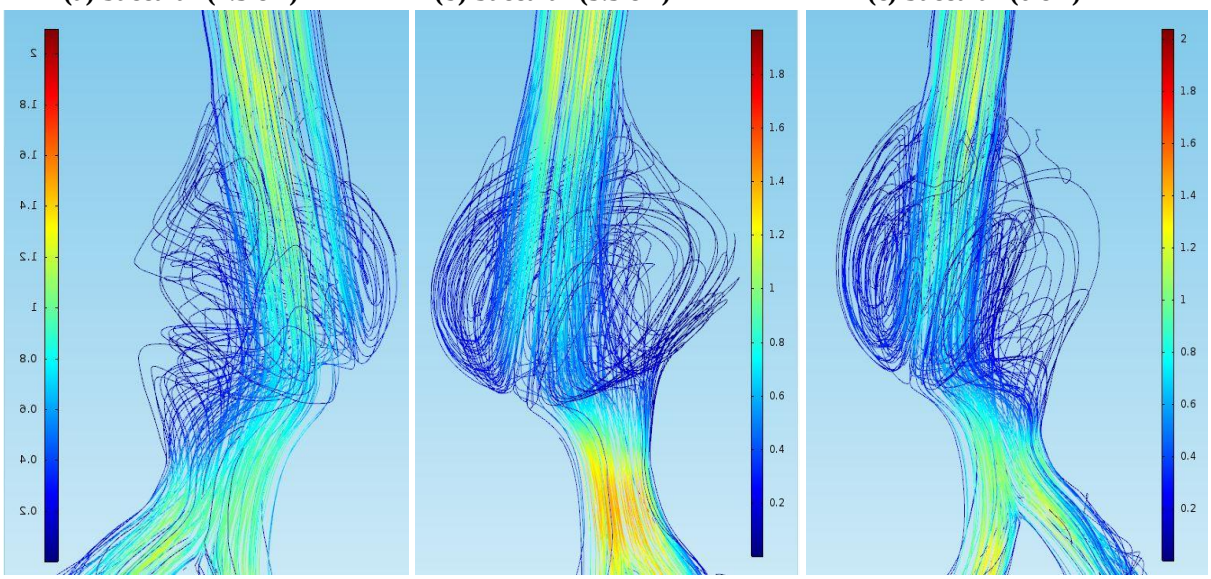


Figure 25: Streamlines flow of healthy abdominal aorta at peak systole pulse cycle t/t_p = 0.18

skewed in saccular case and double sided skewed in fusiform case. However, same phenomenon is also encountered in streamlines. Also, the increase in velocity at the maximum aneurysed is also due to formation of maximum recirculation. Figure 25 shows the streamline flow for the healthy abdominal aorta and figure 26 & 27 are indicating the streamlines flow in the aneurysed aorta in saccular & fusiform respectively.

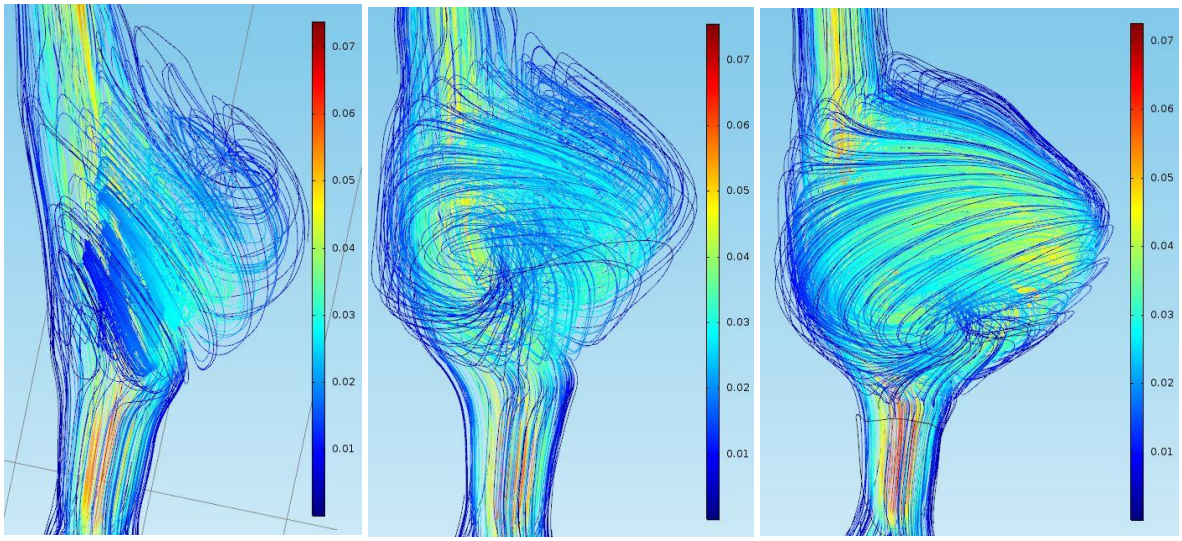


(a) Saccular (4.5 cm) **(b) Saccular (5.5 cm)** **(c) Saccular (6 cm)**

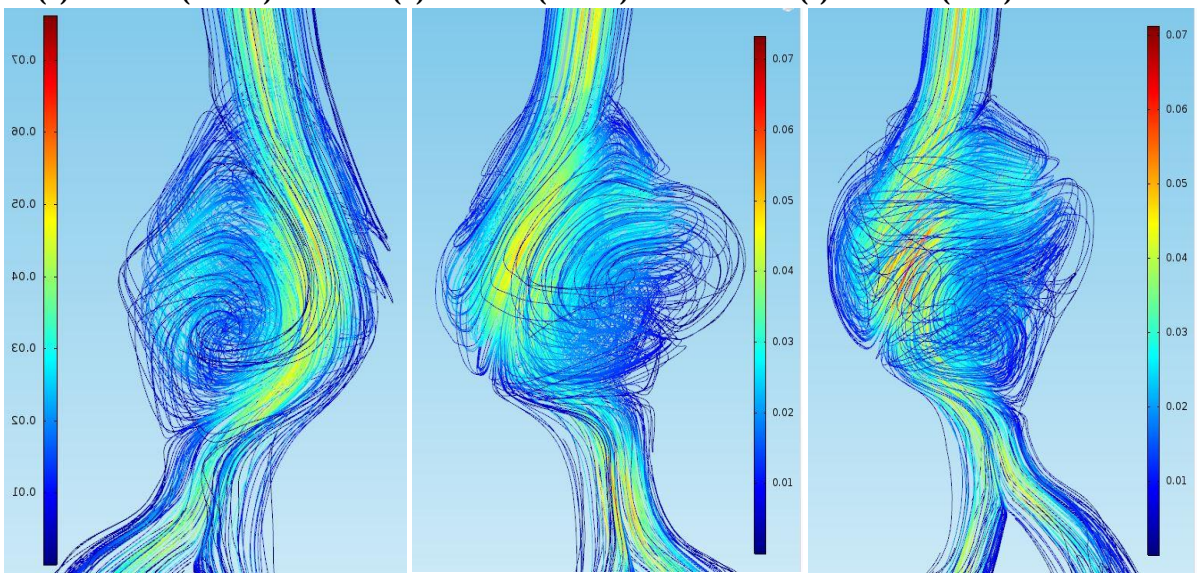


(d) Fusiform (4.5 cm) **(e) Fusiform (5.5 cm)** **(f) Fusiform (6 cm)**

Figure 26 (a)-(f): Streamlines flow of saccular and fusiform AAA at maximum flow @ $t/t_p = 0.18$



(a) Saccular (4.5 cm) **(b) Saccular (5.5 cm)** **(c) Saccular (6 cm)**



(d) Fusiform (4.5 cm) **(e) Fusiform (5.5 cm)** **(f) Fusiform (6 cm)**

Figure 27 (a)-(f): Streamlines flow of saccular and fusiform AAA at minimum flow @ $t/t_p = 0.46$

4.3 Comparative analysis of Wall shear stress

This section compares the wall shear stress at four different circumferencial positions at the pathological region of the aorta. The angular position techniqiue is used to define the points on the circumference of the aneurused plane. These angles are at a differnce of 90 degree with each other. The four positions are taken to select the region of most effected area with oscillatory shear stress.

0^o is the maximum displaced position from the axis of aorta. The regions have commonly two levels of shear stress zones; low oscillatory shear stress zone and high oscillatory shear stress zone.

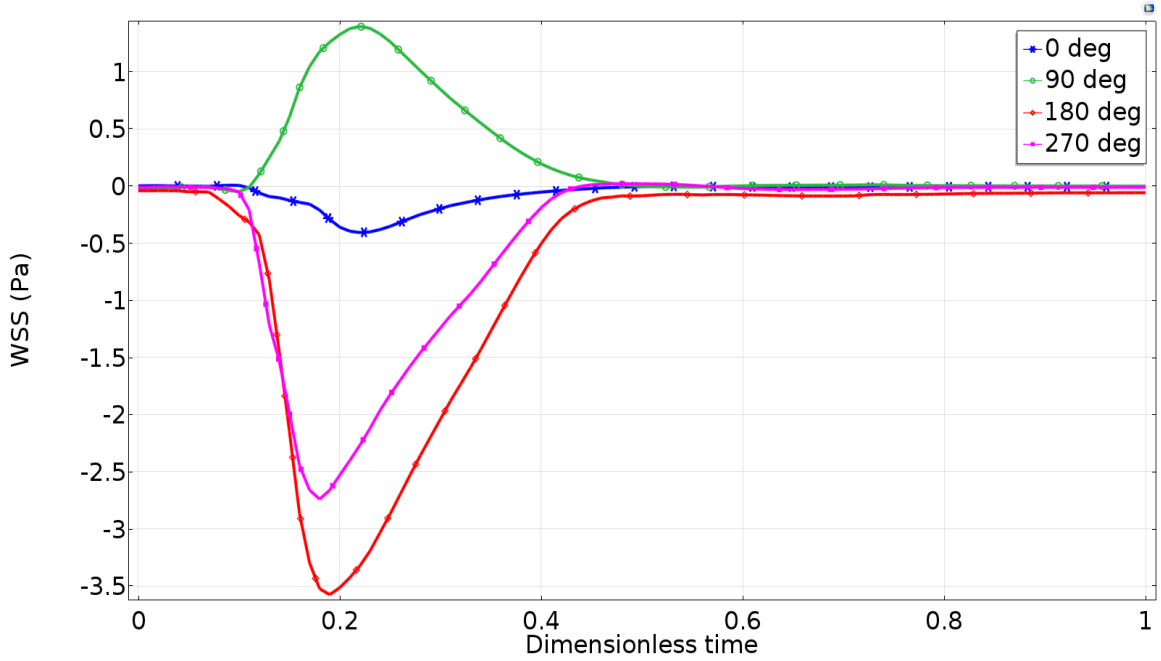


Figure 28: Variation of WSS with time at four circumferential position in saccular 4.5 cm

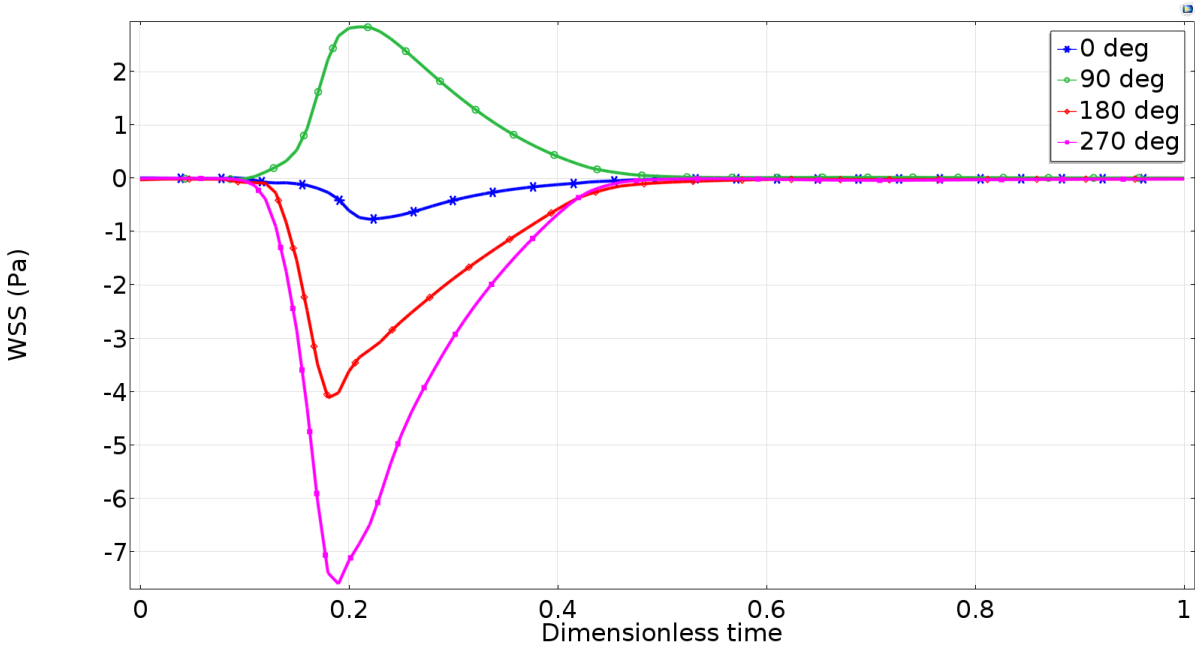


Figure 29: Variation of WSS with time at four circumferential position in saccular 5.5 cm

Figure 28- 33 are showing the variation of wall shear stress with pulse time for saccular and fusiform aneurysm. The wall shear stress is minimum (least value) at 0^o. This may be due to maximum distance of the wall form axis of aorta. The maximum value of wall shear stress is variying for different cases at 180^o and 270^o . For sizes 5.5 cm and 6 cm (both saccular and fusiform) the value of WSS at 270^o.is maximum . Only for size 4.5 cm the both cases having value

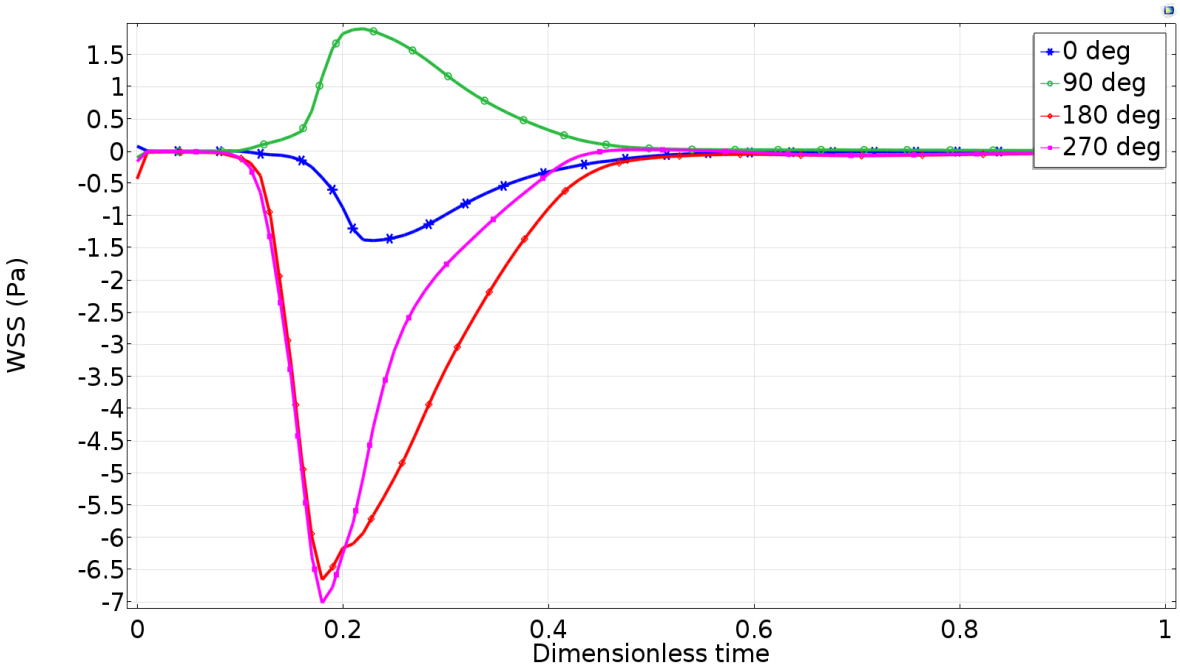


Figure 30: Variation of WSS with time at four circumferential position in saccular 6 cm

of WSS at 180^o is greater than 270^o . It can also be noticed from all the results that WSS varies with pulse time. The figures 28- 33 are also depict that 180^o & 270^o are most effected region of neagtive wall shear stress. In figure 33, the only value of wall shear stress at position 0^o is positive. This is due to the distribution of wall shaer stress at opposite ends (90^o & 270^o) . In this case only the value at 90^o is greater than the values on the negative sides. Hence the maximum value of WSS at 0^o is shifted towards the positive side.

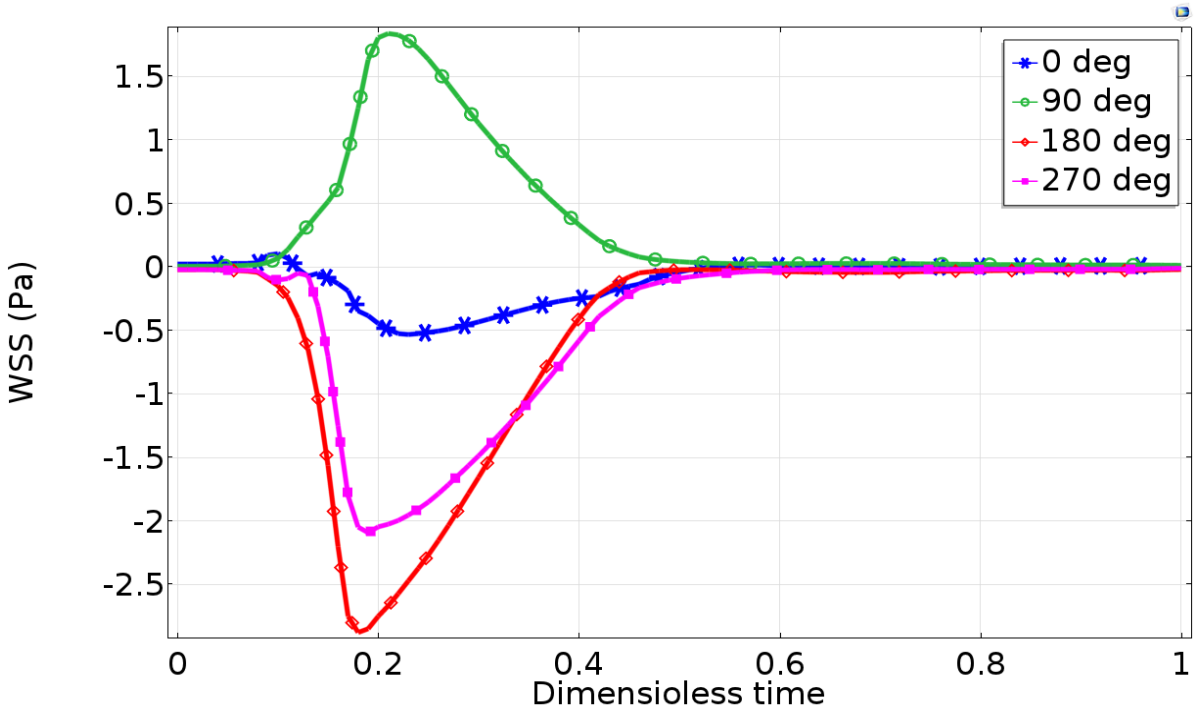


Figure 31: Variation of WSS with time at four circumferential position in Fusiform 4.5 cm

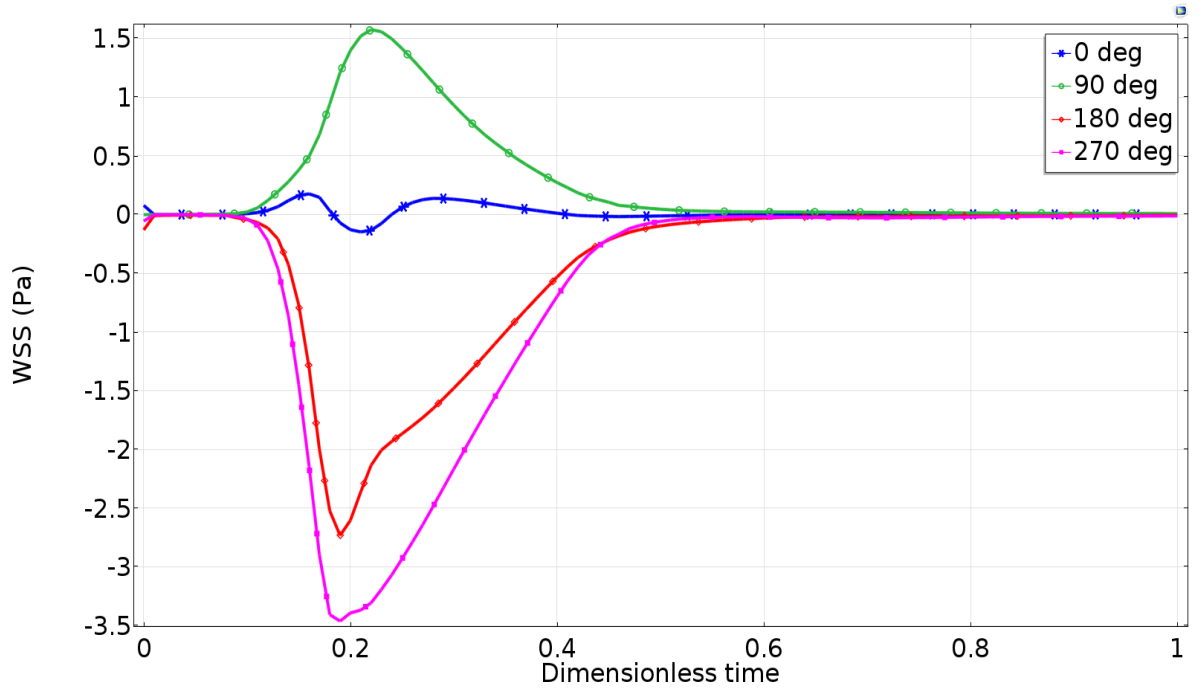


Figure 32: Variation of WSS with time at four circumferential position in Fusiform 5.5 cm

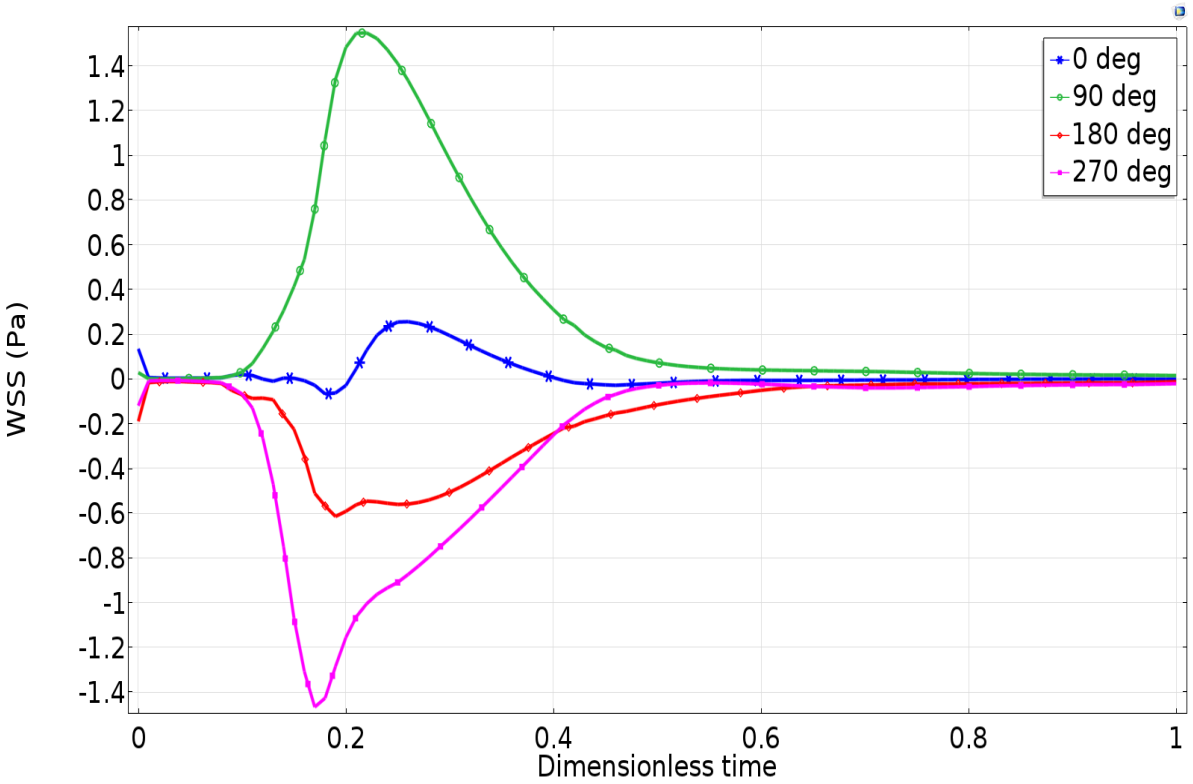


Figure 33: Variation of WSS with time at four circumferential position in Fusiform 6 cm

Table 1 and 2 are showing and comparing the value of wall shear stress at the different circumferencial positions with different aneurysm type and size.

Position **Fusiform Size**	**0º**	**90º**	**180º**	**270º**
4.5 cm	-0.5 Pa	1.7 Pa	-2.1 Pa	-2.7 Pa
5.5 cm	-0.3 Pa	1.6 Pa	-2.8 Pa	-3.5 Pa
6 cm	0.3 Pa	1.5 Pa	-0.6 Pa	-1.4 Pa

Table 1: Values wall shear stress at different position and size of saccular aneurysm

Position Saccular Size	0º	90º	180º	270º
4.5 cm	-0.5 Pa	1.3 Pa	-3.6 Pa	-2.8 Pa
5.5 cm	-0.5 Pa	2.4 Pa	-4 Pa	-7.3 Pa
6 cm	-1.5 Pa	1.7 Pa	-6.7 Pa	-7.2 Pa

Table 2: Values wall shear stress at different position and size of Fusiform aneurysm

The wall shear stress is maximum in the sacuular aneurysm case and the maximum value of wall shear is -7.3 Pa in 5.5 cm saccular aneurysm at circumferencial position 270^{o} and minimum value is at 0^{o} in fusiform (5.5 cm & 6 cm).

Chapter 5

Conclusions

In this study the hemodynamics of the abdominal aorta is analysed by applying actual physiological environment. The three dimensional model of healthy abdominal aorta is created by using real patient specific DICOM image and the aneurysms of sizes 4.5 cm, 5,5 cm & 6 cm are created in the infrarenal part of the abdominal aorta. A computational technique is utilized to study the hemodynamics of different 3D models.

The main objective of the work is to identify the fatal condition of the aneurysm by analyzing the velocity flow phenomenon, streamline flow and the effect of wall shear stress during the pulse cycle. The average flow velocity of the healthy abdominal aorta is approximately 80 cm/sec which is validated with cited literature [6]. The flow velocity of the aneurysed model is oscillating in nature. The average flow velocity varies between 50 cm/sec to 80 cm/s of aneurysed model. This can create deficiency of blood in the lowers limbs (legs) region which causes pain the legs. The WSS is calculated at different circumferencial locations about the plane of aneurysed region (maximum diameter plane). The WSS is used to analyse the critical locations of flow reversal and flow residence. The oscillatory shear stress is least at 0^o position and maximum in between 180^o and 270^o. The low WSS results in the plaque generation at this position and increase the time of blood stanation at that region. This can leads to thromboi and periferal arterial disease (PAD). The value of high stress can cause risk of rupture of aneurysm. Form the results, it is observed that the wall shear stress is maximum in the sacuular aneurysm case and the maximum value of wall shear is -7.3 Pa in 5.5 cm saccular aneurysm at circumferencial position 270^o and minimum value is -0.3 Pa at 0^o in fusiform (5.5 cm & 6 cm). The streamlines of the flow is to visualize the flow reversal zones in a three dimension model. The final conclusion of my work suggested that the aneurysm of 5.5 cm and greater than 5.5 cm is very critical and immediate sugerical intervention is required for preventing it from rupture (hemoharrge).

5.1 Future scope of research

The medical history still is very inconclusive during the reason behind the occurrence of aneurysm. The abdominal aneurysm is an asymptomatic type of disease. The occurrence is 4-6 times more common in men and the risk of rupture is more common in women. The actual reasons of continuous degradation of aortic wall is still remain unknown [11]. This all can be found by creating the actual environment of the human physiological conditions.

Possible future works can be done on the following parameters:

- Blood rheology is taken as Newtonian in the current work but the future study can be done on Non-Newtonian flow by applying Non-Newtonian models such as Casson model, Carreu-Yasuda model, Power law model etc. and results can be compared. Mostly, the blood is either taken as Non-Newtonian for studying the shear thinning nature of blood but in actual blood is a multiphase flow containing RBC, WBC, platelets. So, the actual mathematical modeling of blood can be done and applied to find the more accurate results.
- The live medical imaging technique for the flow analysis could be done for validation of the model.
- The Fluid Structure Interaction simulation of the abdominal aorta model and the blood can be performed to check the effect of compliance on the hemodynamics and the results can be compared with rigid vessel wall.
- The experimental work can be done by creating actual hemodynamic environment. The experiment can be done on the rigid wall model and the compliant model under steady or transient conditions. The results can be compared with the computational simulation.

References

[1] Rukmini S, Cardiovascular disease top killer, Survey by Register General of India, an article published in The Hindu on 16 December 2015.

[2] A Times of India report on "Ischemia heart disease is the top killer across the most states", Surveyed by India state level disease Burden Initiative, November 11, 2017.

[3] Alfio Quarteroni, Massimiliano Tuveri and Alessandro Veneziani, Survey article Computational vascular fluid dynamics: problems, models and methods, Computing and visualization in Science, Vol 2, 2000, pp 163-197.

[4] Frankel Cardiovascular Center (Michigan Medicine), accessed on 15 June 2018, https://www.umcvc.org/conditions-treatments/arteriosclerotic-aortic-disease

[5] https://www.istockphoto.com/in/photos/circulatory-system, accessed on 10 July 2018.

[6] https://radiologykey.com/ultrasound-assessment-of-the-abdominal-aorta, accessed on 2 July 2018.

[7] http://www.edinburghsurgeon.com/aaa.html, accessed on 5 July 2018.

[8] An 'Express' report on 'Abdominal aortic aneurysm: Death rate from burst vessels three times higher in UK than US', published on 24-Nov-2016, accessed on 15 June 2018, https://www.express.co.uk/life-style/health/735893/Abdominal-aortic-aneurysm-symptoms-surgery-treatment-death-rate.

[9] Stebhens W E, History of Aneurysm, Medical History, Vol 2 (4), 1958, pp 274-280.

[10] Aggarwal S, Qamar S, Sharma V, Sharma A, Abdominal aortic aneurysm: A comprehensive review, Experimental & Clinical Cardiology, Vol 16 (1), 2011, pp 11-15.

[11] Villard C and Hultgren R, Abdominal aortic aneurysm: Sex differences, Maturitas, Vol 109, 2018, pp 63-69.

[12] Abdominal Aortic Aneurysm History, accessed on 8th July 2018. https://www.news-medical.net/health/Abdominal-Aortic-Aneurysm-History.aspx

[13] Ku D N, Blood Flow in Arteries, Annual Review Fluid Mechanics, Vol 29 1997, pp 399–434.

[14] Ku D N, Giddens D P, Zarins K C, and Glagov S, Pulsatile Flow and Atherosclerosis in the Human Carotid Bifurcation: Positive Correlation between Plaque Location and Low and

Oscillating Shear Stress, Journal of the American heart association: Arteriosclerosis, Thrombosis and Vascular Biology 1985;5, pp 293-302.

[15] Perktold K, Resch M, Florian H. Pulsatile. Non-Newtonian Flow Characteristics in a Three-Dimensional Human Carotid Bifurcation Model, ASME Journal Biomechanical Engineering 113,1991, pp 463–475.

[16] Perktold K, Resch M, Florian H, Pulsatile Non-Newtonian blood flow in three-dimensional carotid bifurcation models: a numerical study of flow phenomena under different bifurcation angles, Journal of Bio-medical Engineering 1991, Vol. 13, May 1991, pp 507-515.

[17] Perktold K, Rappitsch G, Computer simulation of Local Blood Flow and Vessel Mechanics in a Compliant Carotid Artery Bifurcation Model Journal of Biomechanical 28, 1995, pp 845–856.

[18] Chakravarty S, Sen S, Analysis of pulsatile blood flow in constricted bifurcated arteries with vorticity-stream function approach, Journal of medical engineering & technology, Vol. 32, No. 1, January/February 2008, pp 10 – 22.

[19] Vamadevan A S, Shah B R, Califf RM, Prabhakaran D, Cardiovascular research in India: a perspective, American Heart Journal 2011;161:431-8.

[20] Updegrove A, Wilson, N, Merkow, J, Lan, H., Marsden, A L and Shadden, S C, SimVascular - An open source pipeline for cardiovascular simulation, Annals of Biomedical Engineering (2017) 45: 525. DOI: 10.1007/s10439-016-1762-8 (2017).

[21] Les S A, Yeung Janice J, Geoffrey S M, Herfkens R J, Ronald L D and Taylor C A, Supraceliac and Infrarenal Aortic Flow in Patients with Abdominal Aortic Aneurysms: Mean Flows, Waveforms, and Allometric Scaling Relationships, Cardiovascular Engineering Technology 2010 March; 1(1): DOI:10.1007/s13239-010-0004-8.

[22] W E Merrill and Pelletier G A, Viscosity of human blood: transition from Newtonian to Non-Newtonian, Journal of Applied Physiology, Vol. 23, No. 2, August 1967, pp 178-182.

[23] Brown P M, MD, Zeld T D, Sobolev B, The risk of rupture of in untreated aneurysm: the impact of size, gender and expansion rate, The Society for Vascular Surgery and The American Association for Vascular Surgery, Vol 37 (119), 2003, pp 280-284.