

Simulation of blood flow through stenosed artery

B. Ramakrishna

A Dissertation Submitted to
Indian Institute of Technology Hyderabad
In Partial Fulfillment of the Requirements for
The Degree of Master of Technology



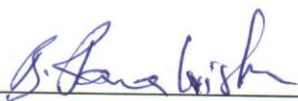
भारतीय प्रौद्योगिकी संस्थान हैदराबाद
Indian Institute of Technology Hyderabad

Department of Chemical Engineering

June, 2013

Declaration

I declare that this written submission represents my ideas in my own words, and where others' ideas or words have been included, I have adequately cited and referenced the original sources. I also declare that I have adhered to all principles of academic honesty and integrity and have not misrepresented or fabricated or falsified any idea/data/fact/source in my submission. I understand that any violation of the above will be a cause for disciplinary action by the Institute and can also evoke penal action from the sources that have thus not been properly cited, or from whom proper permission has not been taken when needed.




(Signature)

(- B. Ramakrishna -)

(CH11M012)

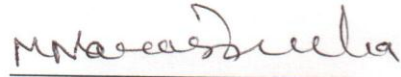
Approval Sheet

This thesis entitled "Simulation of blood flow through stenosed artery" by B. Ramakrishna is approved for the degree of Master of Technology from IIT Hyderabad.



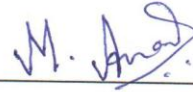
Dr. Parag Pawar, IITH

Examiner



Dr. Narasimha Mangadoddy, IITH

Examiner



Dr. Anand Mohan, IITH

Adviser



Dr. Raja Banerjee, IITH

Chairman

Acknowledgements

I express my gratitude to my supervisor **Dr. Anand Mohan** for suggesting the research problem, and for his guidance and advice throughout the course of my research work. I also thank my committee members **Dr. Raja Banerjee, Dr Parag Pawar, Dr. Narasimha Mangadoddy** for their valuable suggestions. I give special thanks to **Dr. Parag Pawar** and **Dr. Vinod Janardhanan** for encouragement.

It is my pleasure to thank my friends N. Nandakumar and V. Balraju for explaining the use of ANSYS Fluent to me. I also thank my friends, and the Sunshine Team, at IITH for encouraging me.

I express my sincere thanks to my sister, brothers, mother and father for financial and moral support in the last two years.

Dedicated to

My Parents,
who have shaped me and helped me to become a good person

Abstract

Atherosclerosis is a life-threatening disease of the human circulatory system. It can lead to blockage of arteries leading to heart attacks. It is characterized by plaque formation at the walls of the artery lumen. The progression of plaque formation can lead to complete blockage of the artery. The diagnosis of atherosclerosis is very important. So, the study of parameters affecting atherosclerosis is important. We study the effect of percentage blockage and non-Newtonian nature of blood on hydrodynamic factors in an artery that is partially blocked by atherosclerotic plaque. ANSYS Fluent software is used to study these cases. We obtain the velocity profile and wall shear stress profile for 10%, 25%, 50%, and 75% blockage. We also study the effect of various blood models like Newtonian, Power law, Carreau, and Cross models on the fluid dynamic factors. Results show that the percentage of stenosis affects the wall shear stress (WSS) the most. WSS is minimum for 10% stenosis, and maximum for 75% stenosis. The fluctuation of WSS, downstream of the stenosis, is also maximum for the case of 75% stenosis and minimum for the case of 10% stenosis. There is formation of a recirculation zone, downstream of the stenosis, for all models at 50% stenosis.

Nomenclature

η = Viscosity	Pa.sec
η_0 = Viscosity at zero shear rate	Pa.sec
η_∞ = Viscosity at infinite shear rate	Pa.sec
$\dot{\gamma}$ = Shear rate	sec ⁻¹

Contents

Declaration.....	ii
Approval Sheet	iii
Acknowledgements.....	iv
Abstract.....	vi
Nomenclature	vii
1 Motivation and Introduction	1
2 Use of ANSYS FLUENT 13.0	3
2.1 Creation of geometry in ICEM CFD.....	3
2.2 Simulation in FLUENT	5
3 Simulation Results	8
3.1 Effect of % Stenosis	8
3.2 Effect of Non-Newtonian model.....	10
4 Conclusion	14
References.....	15

Chapter 1

Motivation and Introduction

Motivation

Atherosclerosis is a major disease of the human circulatory system. High cholesterol levels, Hypertension and Smoking are risk factors for atherosclerosis. Atherosclerosis is known to follow injury to the endothelium. Subsequently, there is accumulation of lipoproteins, adhesion of monocytes and platelets. This is followed by smooth muscle cell proliferation, and production of extra-cellular matrix followed by lipid accumulation. The end result is the appearance of a lipid-filled plaque that protrudes into the vessel lumen and obstructs the flow of blood (see Kumar et al. 2010)

The study of parameters which affect the progress of atherosclerosis is very important. For instance, it is known that wall shear stress influences the progression of atherosclerosis (see Gibson et al. 1993). To obtain the wall shear stress, we should simulate the flow of blood in artery geometry and calculate the stress using an appropriate material model for blood. The main subject of this project is the simulation of blood flow in stenosis, and the documentation of parameters which affects the progress of atherosclerosis.

Introduction

The circulatory system of the human body consists of two main sub-branches, namely (i) Systemic circulation, and (ii) Pulmonary circulation. We will focus on the systemic circulation.

The Systemic circulation consists of the arteries, arterioles, capillaries, venules, and veins which supply oxygenated blood to the various parts of the human body. The artery diameters range from 20mm to 5mm. At the end of the arteries the pulsatile nature of blood flow is lost, and the flow becomes steady. The diameter of capillaries ranges from 0.5mm to .005mm (van de Vosse & van Dongen 1998). The number of capillaries is approximately a million times that of the number of arteries and veins. This helps in the efficient exchange of solutes with body tissues. The diameter of the veins is in general smaller than that of arteries, but the volume of blood contained is approximately twice that contained in the arteries (see van de Vosse & van Dongen 1998). This is due to the low pressure gradient in the veins.

The rhythmic pumping of the heart induces a pulsatile arterial pressure that varies between 120mm Hg and 80mm Hg. This pulsatile nature is lost when the blood enters the arterioles and the average pressure gradually decreases. The pressure in the capillaries is 35 mm Hg, while it is 10 mm Hg in the venules. Eventually, the pressure becomes near zero in the vena cava.

Blood Rheology

Blood is a Non-Newtonian fluid, and exhibits shear thinning (Charm & Kurland 1965) and viscoelastic behavior (Thurston 1972). The apparent viscosity of blood decreases by 25 to 30 times as the shear rate increases from $.01 \text{ sec}^{-1}$ to 200 sec^{-1} . High viscosity of blood at low shear rate is due to RBC aggregation (Chien et al 1967a), whereas low viscosity at high shear rate is due to RBC disaggregation and deformation (see Chien et al 1967b).

Due to the complexity of its rheological behavior, it has been very difficult to develop a proper three-dimensional model. Non-Newtonian models proposed by earlier scientists include the Casson model, the Cross model and the power law model (see Cho & Kensey 1984). The first viscoelastic model for blood was developed by Phillip & Deutsch (1975). Later, Yeleswarapu et al. (1998) developed a generalized Oldroyd-B model with a shear thinning viscosity function to describe blood flow. Recently, this model was improved by Anand & Rajagopal (2004).

Chapter 2

Use of ANSYS FLUENT 13.0

2.1. Creation of geometry in ICEM CFD

The ICEM CFD 13.0 software is opened by using the ANSYS 13.0 software, Meshing folder. Once ICEM CFD is opened, the procedure given below is used to create the geometry.

Preliminary Steps:

- a) Select create point in the Geometry Toolbar. Select 'Create point' function in the Geometry toolbar, and then 'XYZ'. Create the following point numbers (with corresponding coordinates): 1(0,0,0), 2(120,0,0), 3(120,20,0), 4(0,20,0), 5(55,0,0), 6(60,5,0), 7(65,0,0), 8(65,20,0), 9(60,15,0), 10(55,20,0).
- b) Join the following pairs of points- (1,5), (7,2), (2,3), (3,8), (10,4), (4,1)- using the function 'Create/Modify curves'. Select the desired pair of points in the main window with the mouse to create the lines. Now use the sub-function 'Create Arc', and select the three-point sequences (5, 6, 7) and (10, 9, 8). This results in the semi-circular stenosis arcs being created.
- c) Now, the lines should be named. To do this, go to 'Parts' in the General Window, and Right click 'Parts'. Select the sub-function 'Create part', and now give part name by selecting the required part. Press apply, and repeat the procedure to create the next part. In this manner, the parts 'input', 'output', 'stenosis', and 'wall' are created.

Now that the geometry (of artery with semi-circular stenosis) been created, and the parts have been labeled, we now proceed to “block” the geometry prior to meshing.

Steps for Blocking:

- a) Select the geometry in the Display window. Go to the General Window, and replace the block “Interior” with “Fluid”. Now select the option “2D Planar”, and click “Initialize”. This completes blocking.
- b) Select “Split block” in Toolbar. Select a line that is tangent to the stenosis and parallel to the top and bottom wall. Split the top and bottom walls at the beginning and end of the stenosis.
- c) Use the function ‘Delete Block’ in the Toolbar, and delete the block numbers corresponding to the outer annulus of the pipe. The block numbers are 4,10,23,21,18,16 in ICEM CFD version 13.0.
- d) In the General Window, select the option ‘Block Associate’ and ‘Point Association’. Now, associate the vertices (in the core annular region) with the points.
- e) In the General Window, select the option ‘Edge associate’ and associate the edges of the block with the curves of the geometry (including the stenosis). This completes the creation of the shape of artery with semi-circular stenosis.

Now that the block of the required geometry has been created, we can now generate the mesh inside the block.

Steps for Meshing:

- a) Go to the icon “Mesh” in the Toolbar. Select ‘Curve mesh setup’. In the General window, set maximum element size = 0.1. Now select the curve in the Display Window, and click on “All visible objects”
- b) Go to the option “Blocking” in the General Window, select the option ‘Pre-Mesh Parameters’ and enable the option ‘Update All’.

- c) Go the option “Blocking” in the General Window, select the option ‘Pre-Mesh’, say ‘Yes’ to the question ‘Recompute’.
- d) Go to the option ‘Pre-Mesh’, Right-click, and select ‘Convert to Unstructured Mesh’. This creates the required mesh on the artery inner flow domain. We are now ready to save the geometry with the mesh.

Steps for saving:

- a) Go to ‘Output’ in the Toolbar, and select the options ‘Solver’, ‘FLUENT v6’ and ‘ANSYS’.
- b) Scroll the buttons one by one, and select ‘No Scaling’, and ‘2D’
- c) Save the meshed geometry under an appropriate name using ‘Save’

The above procedure results in the creation of a rigid-walled stenosed artery with length = 120mm length, and diameter = 20mm. The stenosis is at the middle of artery: it has a semi-circular shape, and occupies 50% of the cross sectional area of the artery. The % stenosis is varied when we want to study the effect of % stenosis in our simulation.

2.2. Simulation in Fluent:

We have created the meshed geometry in ICEM CFD using the procedure given above. We are now ready to perform the simulations in FLUENT.

We open ANSYS 13.0, and select FLUENT in the Fluid Dynamics folder. We select the values ‘2D’, ‘Double precision’ and ‘Serial’ for the variables Dimension, Options, Processing Option, respectively.

Steps to input the meshed geometry:

- a) Read the meshed geometry file using ‘File’, ‘Read’, and selecting the already created mesh file (.msh extension)

- b) Scale the mesh to 120 mm x-axis and 20mm y-axis. Select '**mesh was created in mm**' and '**length is viewed mm**' options, and click 'Scale'.

Now that the meshed geometry has been imported, we follow a sequential procedure involving the options in the General window of FLUENT as given below.

Steps to Set-up the simulation:

- a) Go to option '**Models**', and click 'viscous model'; then select 'laminar': a viscous laminar model is enabled.
- b) Go to option '**Materials**' in the General window. The default material parameters are for air: these values are replaced by those of blood, and then renaming 'air' as 'blood'. The properties of blood are density = 1050 kg/m^3 , select model as 'Newtonian' (for example) and viscosity = $0.00345 \text{ kg/(m.sec)}$. Click 'Create /change': this will write the properties of blood into a new material named as 'blood'. Note that the viscosity has to be specified in a different manner for Power-Law, Carreau, and Cross models.
- c) Go to option '**Cell zone conditions**' in the General window. Change 'Cell zone name' to 'fluid', and select the material as 'blood' (the material blood has already been created in the previous step)
- d) Go to option '**Boundary conditions**'. We give 'Input type' as 'velocity-inlet' and 'velocity magnitude' as 0.447 m/s . The 'Output type' is given as 'Pressure-Outlet' and gauge pressure is given as 0.

Steps to Run the simulation:

- a) Go to option '**Solution**'. The 'Solution Methods' is given as 'SIMPLEC' with the default coupling scheme. Now select the values as 'Least square cells based'/'Standard'/'First-order upwind' for the options 'Spatial discretization for gradient'/'Pressure'/'Momentum', respectively.
- b) Go to option '**Solution controls**'. The default under-relaxation factors are given for pressure (= 0.3), density (=1), body forces (=1), and momentum (=0.7). We change the under-relaxation factor for momentum to 0.5 or 0.6 as needed to ensure convergence.

- c) Go to option '**Monitors**'. Select the residuals convergence criteria as 0.001 for x- and y-momentum, and continuity.
- d) Go to option '**Solution Initialization**'. Click 'Initialize from input' after selecting the option 'Calculate from all zones'
- e) Go to option '**Run calculations**'. Enter the 'Number of iterations' as '500'. Click the calculate tab. This initiates the simulations, and the convergence of the solution is shown in the Display window. Typically the solution will converge for all the cases we have examined in less than 500 iterations.

We are now ready to view the results of our calculations.

Steps for Display of results:

- a) Go to option '**Graphics and Animations**'. Go to 'Contours set up' and selecting 'Contours of velocity'. Then select options to 'Fill' the node values, and to use 'Global range' for the display. Now click 'Display' to get the velocity contours for flow of the blood model in the stenosed geometry. Repeat the procedure with appropriate options to display the wall shear stress.

Chapter 3

Simulation Results

The effect of % stenosis, and the choice of non-Newtonian model are studied for flow of blood in an artery with stenosis using ANSYS FLUENT 13.0 software.. The inlet velocity of blood is a steady state value of 0.447 m/s.

3.1 Effect of % Stenosis

We obtain the velocity profile for a power-law fluid model (Non-Newtonian) in an artery with 10%, 25%, 50% and 75% stenosis. The results are given in Figure 1. We observe that, as the size of the stenosis increases, the velocity disturbance increases around the stenosis. We also see that the magnitude of the velocity at the throat of stenosis increases as the size of the stenosis increases: it is the highest for 75% stenosis and least for 10% stenosis. Additionally, the velocity increases as the flow moves through the stenosis, and reaches a maximum at the throat of the stenosis as seen in Figure 2.

The length-wise variation of the wall shear stress (WSS) for the flow of a power law model in an artery with 10%, 25%, 50%, and 75% stenosis is given in Figure 3. We can observe maximum wall shear stress at the throat of stenosis, and this value increases as % stenosis increases. At the length of the wall where there is no stenosis, the shear stress is constant. The WSS increases after the entrance of the stenosis, and reaches a maximum at the throat of stenosis. It then decreases to minimum at the end of the stenosis. After that, it increases again and, after a certain length, reaches its pre-stenosis constant value.

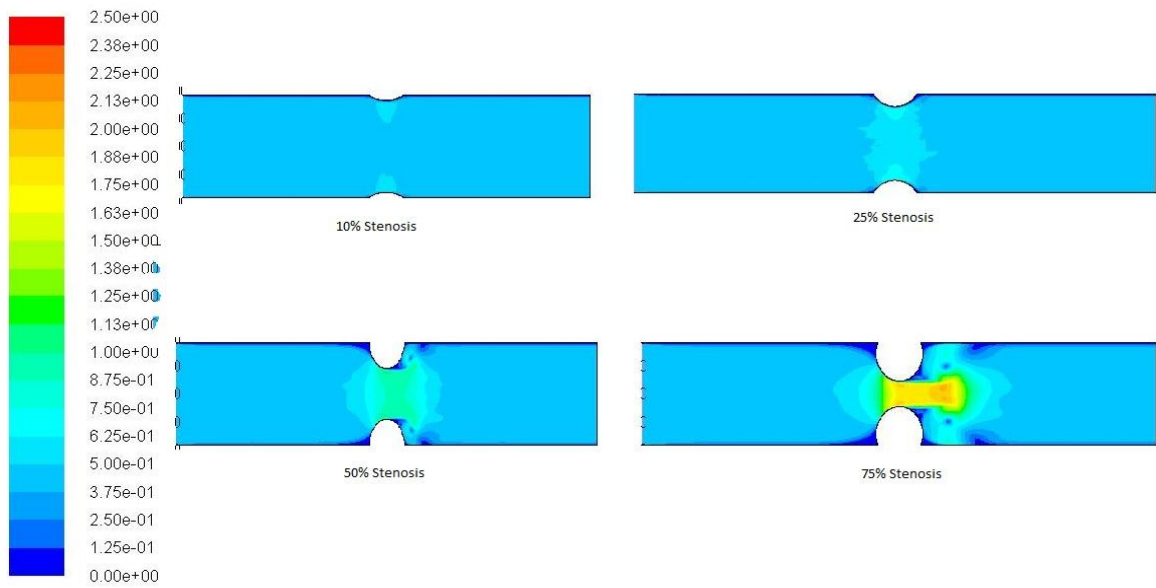
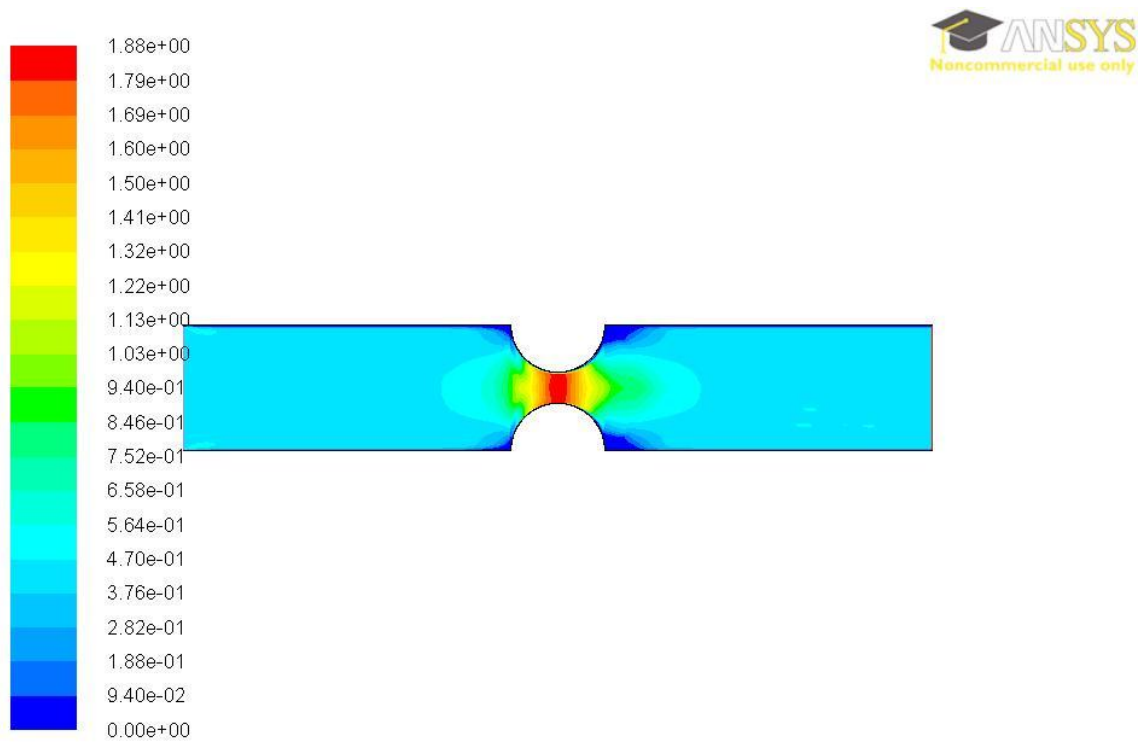


Figure 1. Axial Velocity magnitude for power-law fluid (various % stenosis)



Contours of Velocity Magnitude (m/s) May 22, 2013
ANSYS FLUENT 13.0 (2d, dp, pbns, lam)

Figure 2. Axial Velocity magnitude for power-law fluid (75% stenosis)

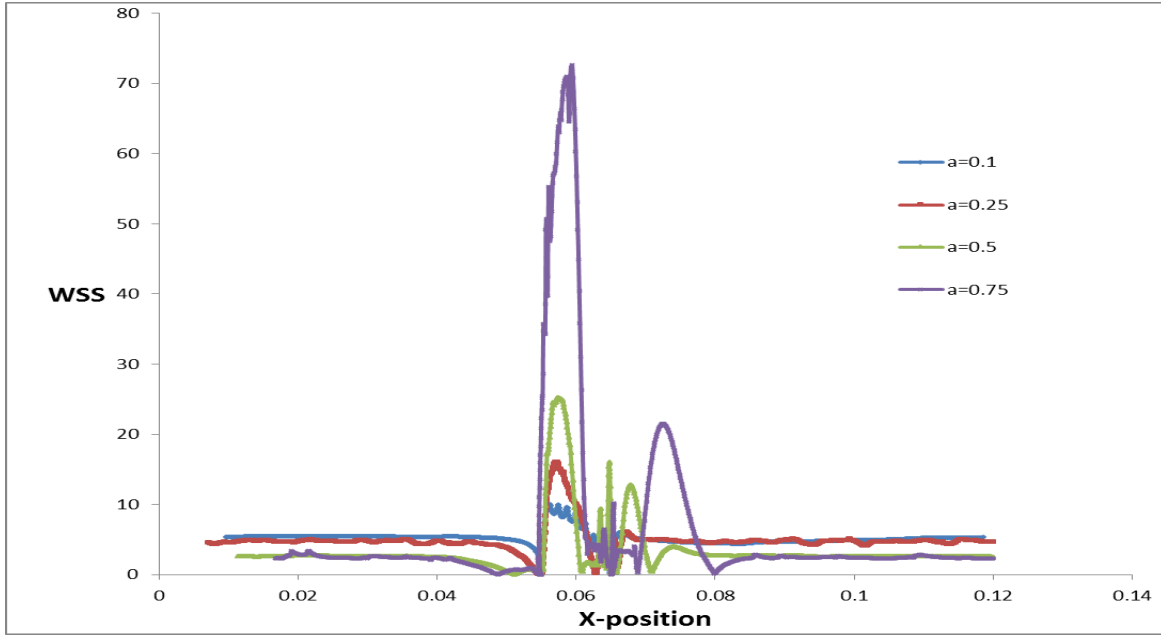


Figure 3. WSS: Length-wise variation for power-law fluid (various % stenosis)

3.2 Effect of Non-Newtonian models

We now study the effect of choice of Non-Newtonian model on the magnitude of the velocity, and the wall shear stress for 50% stenosis with the same inlet velocity. We consider the Newtonian model, power law model, Carreau model, and Cross model as possible choices for blood, and use the material parameters given in Cho & Kensey (1984). The expression for the viscosity (η) in these models is given below.

Newtonian model:

$$\eta = \text{constant}, \quad (1)$$

Here $\eta = 0.00345$ Pa. sec.

Power-Law model:

$$\eta = -m|\dot{\gamma}|^{n-1}, \quad (2)$$

Here $m = 0.042$ Pa.secⁿ, $n = 0.61$.

Carreau model:

$$\frac{\eta - \eta_{\infty}}{\eta_0 - \eta_{\infty}} = \{1 + (\lambda|\dot{\gamma}|)^2\}^{\frac{n-1}{2}}, \quad (3)$$

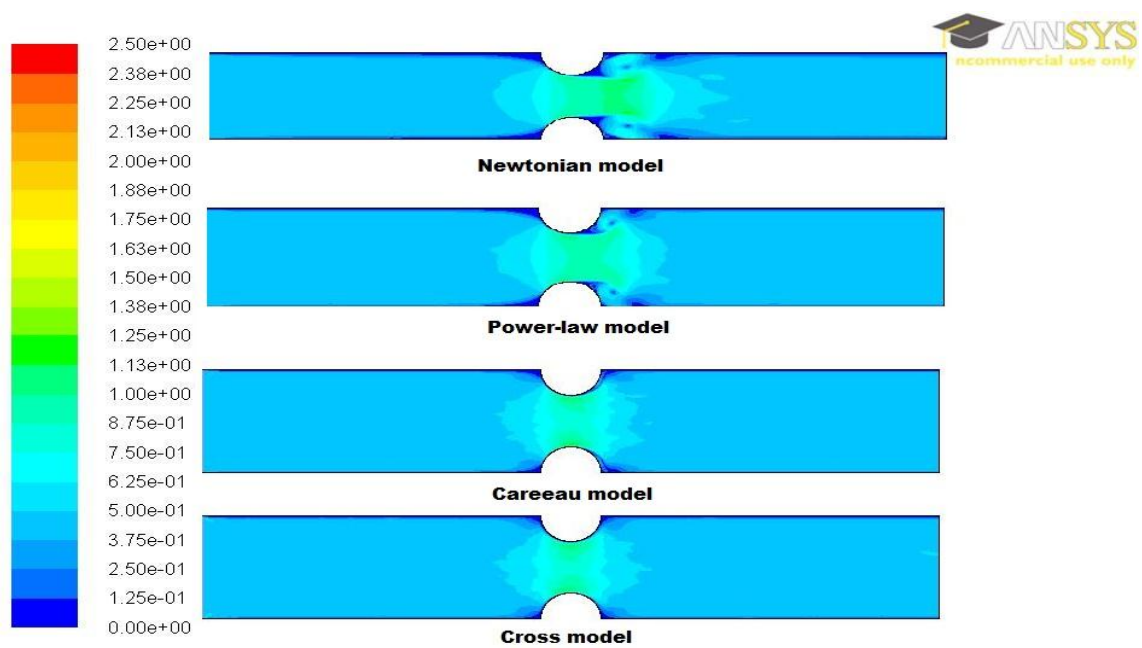
Here, $\eta_0 = 0.056$ Pa.sec, $\eta_{\infty} = 0.00345$ Pa.sec, $\lambda = 3.313$ sec, $n = 0.3568$.

Cross model:

$$\frac{\eta - \eta_{\infty}}{\eta_0 - \eta_{\infty}} = \frac{1}{\{1 + (k|\dot{\gamma}|)^n\}}, \quad (4)$$

Here, $\eta_0 = 0.056$ Pa.sec, $\eta_{\infty} = 0.00345$ Pa.sec, $k = 1.007$ sec, $n = 1.028$.

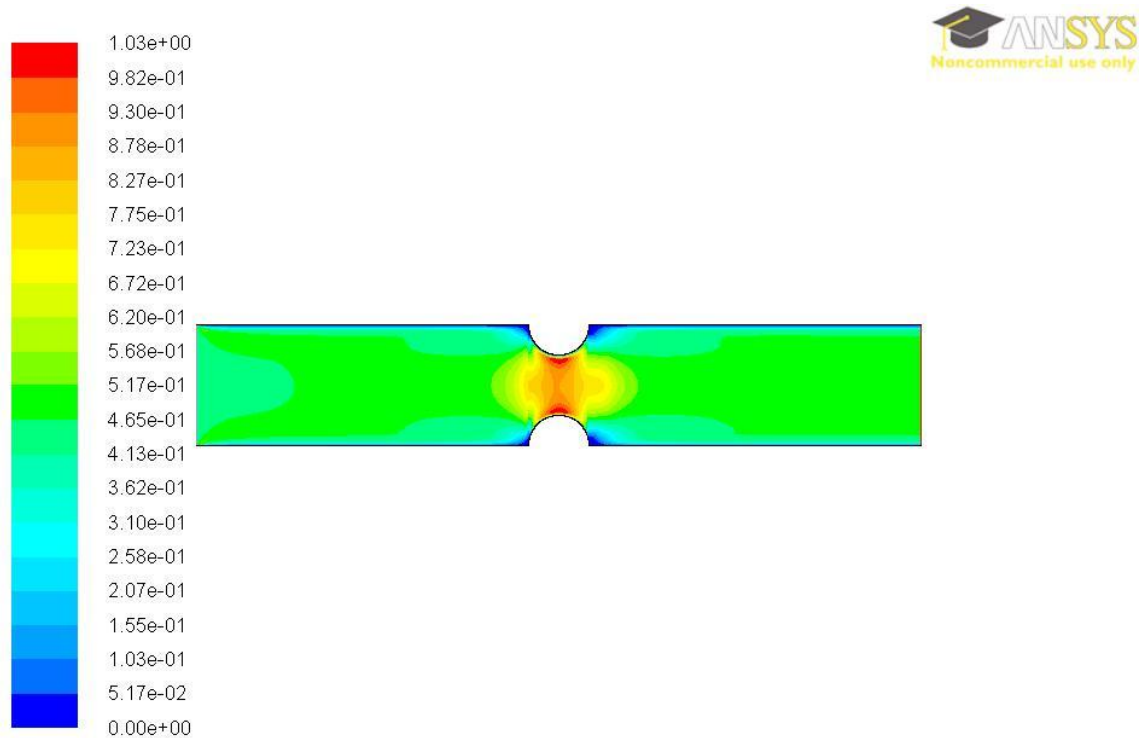
The results for the magnitude of axial velocity are shown in Figure 4 below. The velocity is highest for the Newtonian fluid (due to lowest viscosity = 0.00345 Pa.sec), and lowest for Cross model. The effect of the stenosis (either in disturbing the flow, or recirculation zones) is felt the most when the viscosity is minimum (i.e. for the Newtonian model) and is felt the least for Cross model. Recirculation zones are prominent in the Newtonian model, and they become increasingly less prominent as we consider the power-law, Carreau, and Cross models in that order. However, as we see in Figure 5, a recirculation zone, though small, is still present even for the Cross model.



Contours of Velocity Magnitude (m/s)

May 14, 2013
ANSYS FLUENT 13.0 (2d, dp, pbns, lam)

Figure 4. Axial Velocity magnitude of various blood models (50% stenosis)



Contours of Velocity Magnitude (m/s)

May 23, 2013
ANSYS FLUENT 13.0 (2d, dp, pbns, lam)

Figure 5. Axial Velocity magnitude of Cross model (50% stenosis)

We now consider the length-wise variation of wall shear stress (WSS) for these models: see Figure 6. Peak WSS is highest for the case of Cross model (which has the maximum average viscosity, and a gradual variation of viscosity). However, peak WSS is approximately same for the Newtonian, Power law and Carreau models. After the end of the stenosis region, there is a sudden drop in WSS corresponding to the presence of the recirculation zone. The WSS recovers its original value at a certain distance away from the end of the stenosis: this distance is maximum for the Newtonian model due to its viscosity being the lowest.

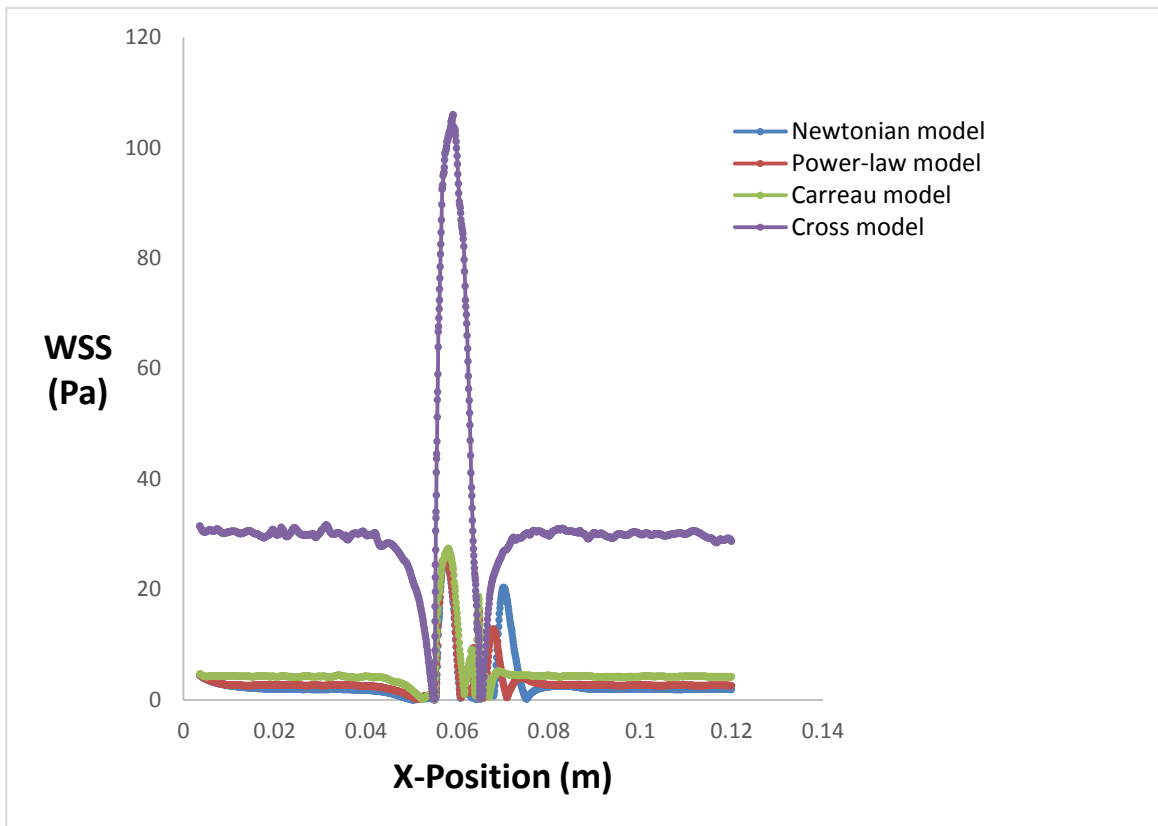


Figure 6. WSS: Length-wise variation of various blood models (50% stenosis)

Chapter 4

Conclusions

The simulation results show that, for a given fluid model for blood, the magnitude of the velocity and the peak WSS increase as % stenosis is increased. Both parameters reach a maximum at the location of peak stenosis. A recirculation zone is formed downstream of the stenosis at all values of % stenosis, and the extent of the recirculation zone increases as the % stenosis increases.

The study using different Non-Newtonian models shows that, for a given % stenosis, the magnitude of axial velocity is maximum for the Newtonian fluid (which has the lowest viscosity) while it is minimum for the Cross model. The recirculation zone is largest for the Newtonian model and smallest for the Cross model. The peak WSS predicted by the Cross model is much higher than those predicted by the other models considered (Carreau, Power-Law, and Newtonian). Hence, using the Cross model in simulations of atherosclerosis progression will lead to prediction of slowest growth rate of stenotic plaque.

The next step in this study should be a simulation of pulsatile flow of Non-Newtonian fluid models in the stenosed artery.

References

- 1) Anand M and Rajagopal K.R., A shear-thinning viscoelastic fluid model for describing the flow of blood. *International Journal of Cardiovascular Medicine and Science*, 4(2): 59-68 (2004).
- 2) Charm S. E. and Kurland G. S., Viscometry of human blood for shear rates of 0–100,000 sec⁻¹. *Nature*, 206:617–618 (1965).
- 3) Chien S., Usami S., Dellenback R. J., Gregersen M. I., Blood viscosity: Influence of erythrocyte aggregation, *Science* 157:829-831 (1967a)
- 4) Chien S., Usami S., Dellenback R. J., Gregersen M. I., Blood viscosity: Influence of erythrocyte deformation, *Science* 157:827-829 (1967b)
- 5) Cho Y. I. and Kensey K. R., Effects of the non-newtonian viscosity of blood on flows in a diseased arterial vessel. (i) Steady flows, *Biorheology* 28:241-262 (1991).
- 6) Elad D. and Einav S., Physical and flow properties of blood, *In: Biomedical Engineering and Design Handbook*, Editor: M. Kutz, McGraw-Hill (2004).
- 7) Gibson C. M., Diaz L., Kandarpa K., Sacks F. M., Pasternak R. C., Sandor T., Feldman C., Stone P. H., Relation of vessel wall shear stress to atherosclerosis progression in human coronary arteries, *Arteriosclerosis, Thrombosis, and Vascular Biology*, 13: 310-315 (1993).
- 8) Kumar V., Abbas A. K., Aster J. C., Fausto N., Robbins and Cotran Pathologic Basis of Disease - 8th Edition, Saunders Elsevier, Philadelphia (2010).
- 9) Marieb E. N., Human Anatomy and Physiology - 6th Edition, Pearson Education (2006).
- 10) Merrill E. W., Rheology of Blood. *Physiological reviews*, 49(4): 863-888 (1969)
- 11) Phillips W.M. and Deutsch S., Towards a Constitutive Equation for Blood, *Biorheology*, 12(6): 383-389 (1975).

- 12) Thurston G. B., Viscoelasticity of human blood. *Biophysical journal*, 12(9): 1205-1217 (1972)
- 13) Van de Vosse F.N. and Van Dongen M.E.H., Cardiovascular Fluid Mechanics - Lecture Notes. www.mate.tue.nl/people/vosse/docs/cardio.pdf, Accessed 14.06.2012 (1998)
- 14) Yeleswarapu K. K., Antaki J. F., Kameneva M. and Rajagopal K. R., The flow of blood in the tubes: theory and experiments. *Mechanics Research Communications* 25: 257–262 (1998).