A HEMODYNAMIC MODELLING OF THE BLOOD CIRCULATION

A Thesis

by

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This thesis meets the standards for scope and quality of
Texas A&M University-Corpus Christi and is hereby approved.

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December 2016

ABTRACT

A mathematical model of the blood flow through an axisymmetric stenosis vessel was developed using the Navier-Stokes equations. A numerical approach has been used to analyze behavior of the blood flow because an analytical solution of such a problem is impossible.

The method of finite elements was applied to find the velocity and the pressure of the blood flow in stenotic vessels of the cardio-vascular system. The solution of the Navier-Stokes equations was done using Matlab and COSMOL Multiphysics.

The findings of the modeling demonstrated that velocity and wall shear stress significantly increase due to the stenosis in the part of the vessel that is blocking blood flow and that this increase depends on the size of the blockage. The modeling shows that increased velocity, vessel wall shear stress, and significant variations in blood pressure may lead to abnormality of the blood flow, which in turn may be a cause of the heart attacks or/and strokes.

The analysis of this research has been compared with the existing results published in research papers. Future investigations of the effect of a more general boundary for a stenosis are planned.

ACKNOWLEGDEMENTS

First of all, I would like to thank the Texas A&M University-Corpus Christi for giving me a chance to advance my mathematical intellectuality by admitting me to the Master program in Mathematics. I have learned from all faculty members of the department. My foremost appreciation goes to Dr. Alexey Sadovski for being my advisor for this thesis. I would like to acknowledge my other committee member Dr. Devanayagam Palaniappam who provided me his tremendous time to advise me on my thesis. Additionally, I would like to thank, Dr. Scott King for his contribution to this thesis by providing her valuable time.

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CHAPTER I: INTRODUCTION

The major cause of death in developed countries is cardiovascular disease. The medical community is desperately seeking a comprehensive description for the cardiovascular system. That is the reason why Biomedical, Bioengineering, and Computer science communities have interest in finding quantitative details on cardiovascular diseases. According to the Center for Disease Control and Prevention "Overall, 11% of adults aged 18 and over had ever been told by a doctor or other health professional that they had heart disease, 6% had ever been told they coronary heart disease, 24% had been told on two or more visits that they had hypertension, and 3% had been told they had a stroke" [15].

The cardiovascular biomechanics focuses on the cardiovascular system, the heart and blood vessels and studies the mechanics of blood flow. Furthermore, it studies the mechanical factors that are essential for cardiovascular diagnosis, surgery, and intervention.

In developed countries, the mortality rate is more than 40% due to cardiovascular disease such as congenital disease of the heart, its valves or the large arteries. Among those diseases, atherosclerosis leads to the cardiovascular diseases. Atherosclerosis is the progressive state of narrowing of the arteries due to deposition of fat and smooth muscle cells in the artery wall that leads to partially or fully occlusion of smaller distal vessels. Stenosis is another name for artherosclerosis. Unfortunately, the main parts of our body such as the brain (stroke) and the coronary arteries (myocardial infarction) are subjected to damages due to this build up. In our work, we focus on nonlinear mathematical analysis for blood flow in a stenotic area at a small part of the aortic artery.

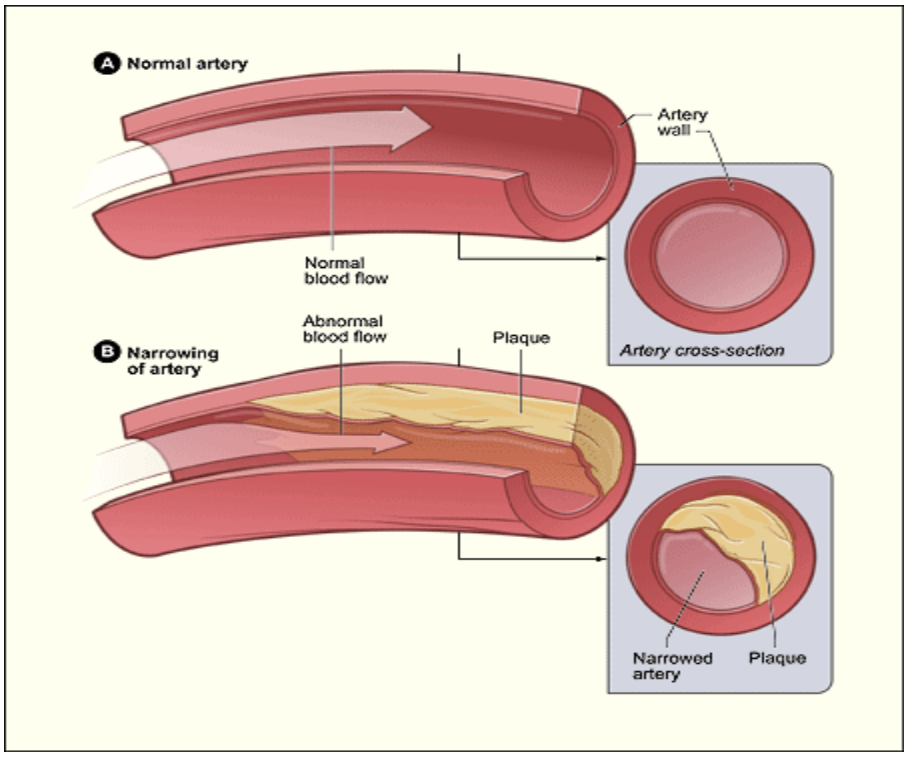

Figure 1. Normal artery and artery with plaque built up from http://www.nhlbi.nih.gov/health/health-topics/topics/atherosclerosis

The effects of blood flow are considered as unsteady; the constitutive behavior of blood is considered as a Newtonian fluid in large arteries. Then we will compare results with the outcomes from other models that we reviewed.

CHAPTER II: CARDIOVASULAR SYSTEM (CVS) MODELLING

CVS model can be solved through two processes Finite Element (FE), and Finite differences method (FDM) approaches [16].

Finite Element Method

The finite element method is used when CVS breaks down in great details. The mathematical model related to CVS could be introduced as ordinary differential equation or the partial differential equation. The partial differential equations models are difficult to solve, due to the highly nonlinearity of the convection term of the Navier-Stokes equations. The analytical solution in the form of Laplace, the Fourier transform method or in form of power series method are practically impossible to solve the partial differential equation in a complex geometry. Therefore, a numerical solution is necessary. The Finite Element method is the best way for solving a non-linear partial differential method [3].

Finite Difference Method (FDM)

The FDM is based on the theory of Taylor expansion to approximate the differential equations. The flow field is dissected into several grids, and the velocity and pressure are approximated by discretizing value of these functions calculated at the grid points [9]. The easy way to define the finite difference method is to convert PDEs equation with spatial and time derivatives to the linear algebraic equations, then it will be solved numerically using Matlab.

The Finite difference method is used to find blood behavior for a healthy vessel, and the finite element method is used to find blood behavior through a stenotic vessel in my thesis.

CHAPTER III: REVIEW OF THE LITERATURE

The CVS blood flow is unsteady in small arteries such as capillaries. The understanding of the behavior of blood flow in the blood vessels provides knowledge about the connection between flow and the development of diseases such as atherosclerosis. Although blood flow is complex, its dynamics can be described through the Poiseuille's model for steady flow and Bernoulli's equation for constricted vessel [4]. Poiseuille's equation is: the volume flow Q = $\int_0^R 2\pi vrdr = \pi R^4 \frac{(P1-P2)}{8\mu L}$, the volume flow increases exponentially with inner radius of vessel increases. The Bernoulli's equation is P+ρgx+0.5ρv^2= constant, where P is pressure, ρ=density.

Verma et al have shown that the viscosity increases as the height of stenosis increases. They concluded that the blood viscosity in a normal blood vessel is lower than the blood viscosity in a stenotic blood vessel. The wall shear stress is high in a stenotic blood vessel with catheterization. However, the wall shear stress decreases sharply with increasing the slip velocity. They have shown velocity profile where the axial velocity increases with the increase of stenotic height [14].

Husain et al have investigated the magnitude of wall shear stress with respect to severity of flow rate. The wall shear stress increases with increasing flow rate. The highest value of wall shear stress reached just before the throat of the stenosis [5].

Sousa et al investigated the qualitative numerical behavior of the blood flow. They have presented graphs of axial flow velocity profile in three different cross-sections around mild stenosis. The axial velocity reached the maximum in the core of the stenosis, but flow stagnation has occurred after the peak of the stenosis core. The low shear stress

has occurred after the peak of the stenosis. The low shear stress could cause a possible formation of atherosclerotic plaques [13].

CHAPTER IV: DISCUSSION OF FINDINGS IN THE LITERATURE REVIEW

The blood flow will be disrupted from normal status with the presence of the arterial stenosis [14]. The study of pulsatile flow is very important to understand the impact of blood flow on atherosclerosis and stroke [14]. Some researchers have found the pulsatile flow of blood through an arterial segment with dependent stenosis was researched. Those researchers used an appropriate nonlinear blood flow model and solved numerically to find blood behavior with influence of periodic body acceleration through a multiple stenosed artery [1]. Mustapha et Al [7] studied the characteristics of blood flow through multi-irregular arterial stenosis. They used MAC method to simulate numerically with using finite element method. The normalized pressure drop is higher for cosine model over irregular model of multiple stenosis. Many researchers investigated the blood flow through the stenosis as a Newtonian fluid, however; experiments have shown a non-Newtonian behavior due to the low shear rate of blood [6]. Recently Chaudhary K. has done researched on blood flow behavior for 20, 50, and 80% blockage of a blood vessel. He has shown wall shear stress and velocity field for 20%, 50% and 80% blockage. According to the simulation results of Chaudhary K.'s work for project work on his Master of Science, the 20% blockage would not harm to our human circulatory system, however 50% blockage would need attention [2].

The computational fluid dynamics is able to describe the flow through stenosed vessels that will be an invasive tool to find earlier diseased state of blood flow system [9]. My thesis uses the Navier-Stokes and continuity equations to find the velocity field, pressure drop and wall shear stress. Treating the blood flow as unsteady flow will be appropriate for my thesis research of the blood flow via stenotic blood vessel.

The unsteady Navier-Stokes equations for two-dimensional blood flow through an axisymmetric stenotic vessel are solved numerically. The process of finding velocity field, pressure, and wall shear stress are divided into three parts to solve numerically.

CHAPTER V: METHODOLOGY/RESULTS

Two-dimensional Flow Solution for a Healthy vessel

Mathematical Model

The finite difference method is used to find velocity along x and y direction with given initial velocities, boundary velocities condition, and constant pressure for rectangular domain [0,1] x [0,1] through a vessel without stenosis as shown in Figure 2. Since the blood initial at rest, the initial condition of velocities at t=0 are zero. With constant pressure, boundary velocities conditions (5), and initial conditions (4), the velocities profiles along x and y direction are found using momentum equations (1), (2), and continuity equation (3).

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x} + v\frac{\partial u}{\partial y} = -\frac{\partial P}{\partial x} + \frac{1}{Re}\left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right) \quad (1)$$

$$\frac{\partial v}{\partial t} + u\frac{\partial v}{\partial x} + v\frac{\partial v}{\partial y} = -\frac{\partial P}{\partial y} + \frac{1}{Re}\left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2}\right) \quad (2)$$

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0 \quad (3)$$

Initial conditions:

u (y, x, t) = 0, where u = x-directional velocity

v(y,x,t) = 0, v= y-directional velocity (4)

P(y,x,t) = 0

Boundary conditions:

u=v=0 no slip condition @ y = 1

u*n=0, v=0 @ r=0 or as slip condition

u = 1, v = 0 @ boundary when x=0 and x=1 (5)

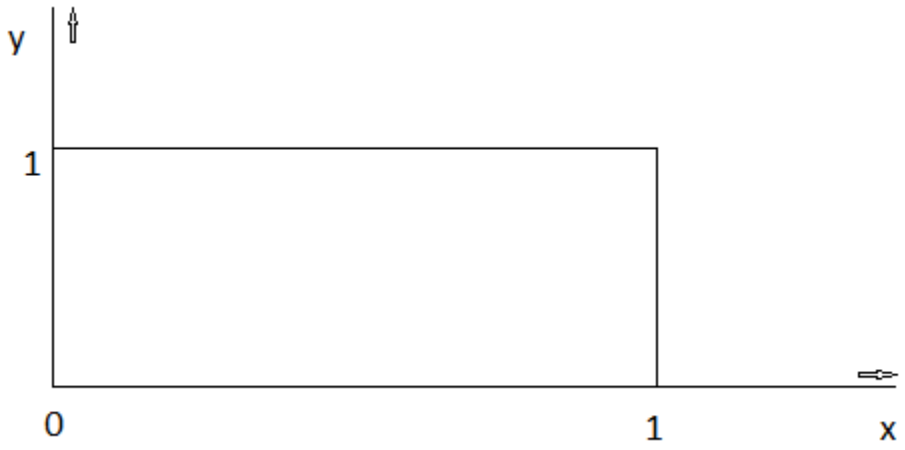

Figure 2: Geometry of blood vessel without stenosis

The flow field is dissected on a staggered grid. The position of the variables such as velocity field and the pressure are calculated as placing those variables at different location as shown in Figure 3. The numerical solution is derived using Matlab code [appendix A], [11].

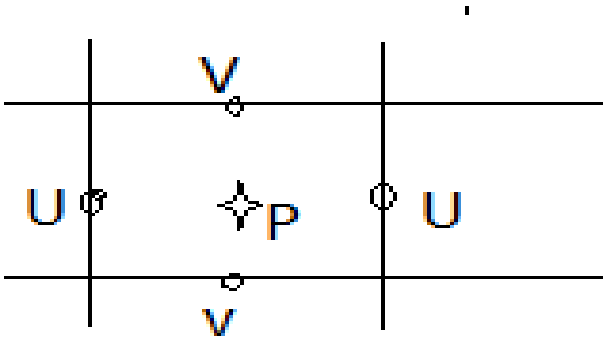

Figure 3: Position of Pressure and Velocities

Numerical solution and analysis

The velocities fields provide a precise explanation of the blood flow, so we are including several axial and y-direction velocity profiles for the healthy vessel. Axial velocity profile has shown in table 1 that represent for Re = 100 and t = 4.

Table 1: Axial velocity profile

		x-axis										
y -axis		0.03	0.1	0.3	0.38	0.5	0.6	0.70	0.76	.83	.93	0.97
	0	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	0.03	1.00	1.00	0.99	0.99	0.99	0.99	0.99	0.99	1.00	1.00	1.00
	.1	0.99	0.98	0.97	0.97	0.97	0.97	0.97	0.97	0.97	0.99	0.99
	.2	0.99	0.97	0.96	0.95	0.95	0.95	0.95	0.96	0.96	0.98	0.99
	.3	0.99	0.95	0.94	0.93	0.93	0.93	0.93	0.94	0.95	0.97	0.99
	.38	0.97	0.91	0.89	0.88	0.87	0.87	0.87	0.88	0.90	0.95	0.97
	.44	0.96	0.88	0.85	0.84	0.83	0.83	0.83	0.84	0.86	0.93	0.96
	0.51	0.92	0.78	0.75	0.73	0.73	0.72	0.73	0.74	0.76	0.86	0.92
	0.51	0.68	0.54	0.53	0.53	0.52	0.52	0.52	0.53	0.53	0.59	0.68
	0.55	0.31	0.44	0.45	0.45	0.45	0.45	0.45	0.45	0.45	0.40	0.31
	0.58	0.17	0.34	0.37	0.38	0.38	0.38	0.38	0.37	0.36	0.27	0.17
	0.62	0.11	0.26	0.30	0.31	0.31	0.31	0.31	0.30	0.28	0.18	0.11
	0.65	0.05	0.15	0.17	0.19	0.20	0.20	0.19	0.18	0.16	0.09	0.05
	.69	0.03	0.10	0.13	0.14	0.15	0.15	0.15	0.14	0.12	0.06	0.03
	.72	0.02	0.07	0.09	0.10	0.11	0.11	0.11	0.10	0.08	0.04	0.02
	.76	0.01	0.05	0.06	0.07	0.07	0.08	0.07	0.07	0.06	0.03	0.01
	.79	0.01	0.03	0.04	0.05	0.05	0.05	0.05	0.04	0.04	0.02	0.01
	.83	0.00	0.02	0.02	0.03	0.03	0.03	0.03	0.03	0.02	0.01	0.00
	.86	0.00	0.01	0.01	0.01	0.02	0.02	0.02	0.01	0.01	0.00	0.00
	.89	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00
	.93	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

	.97	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

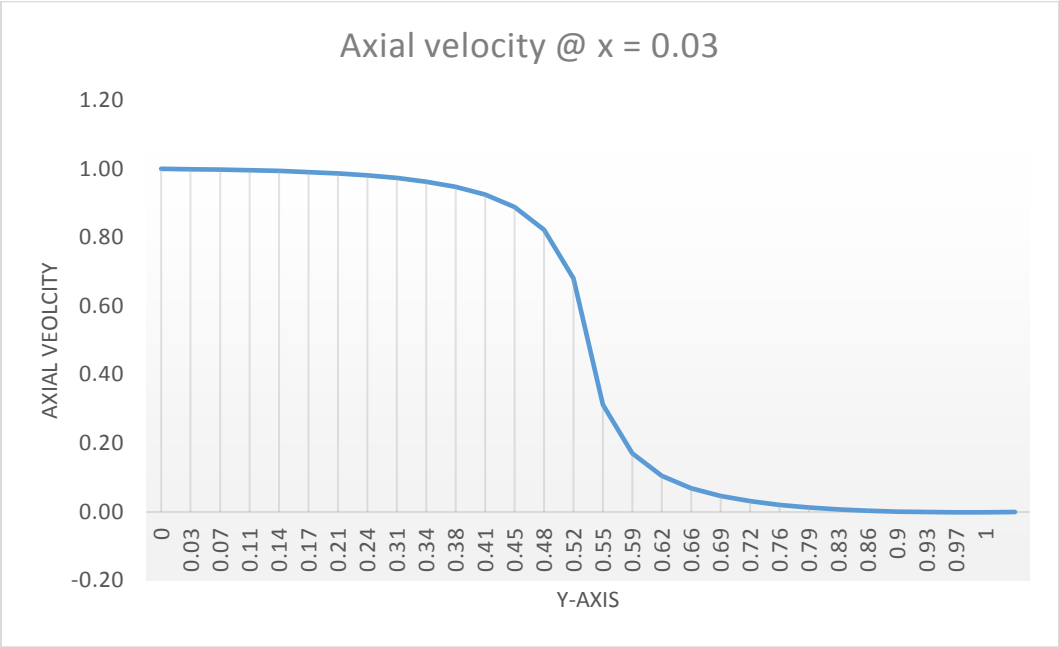

Figure 4: Axial velocity @ x=0.3

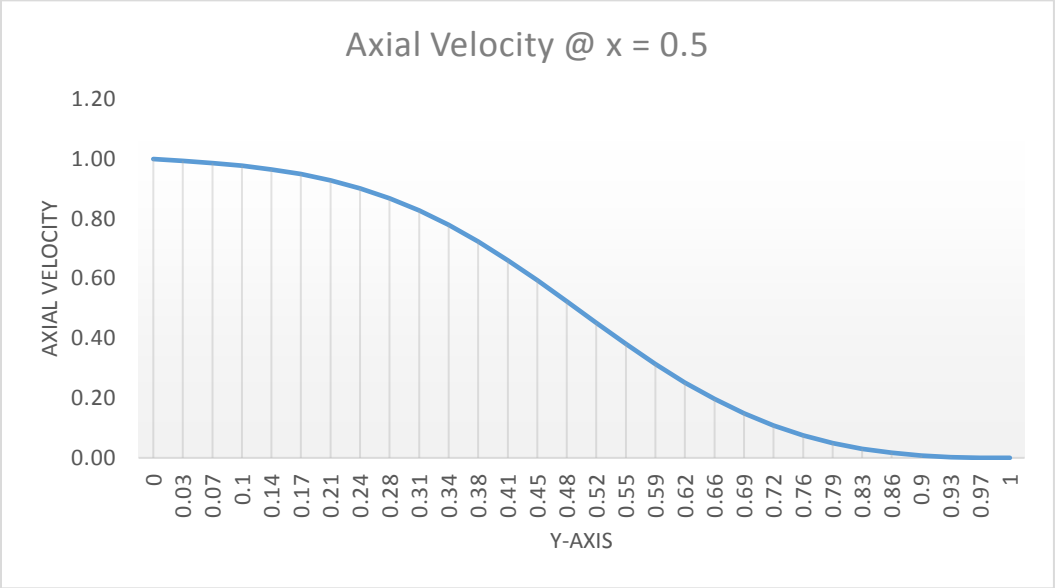

Figure 5: Axial Velocity @ x = 0.5

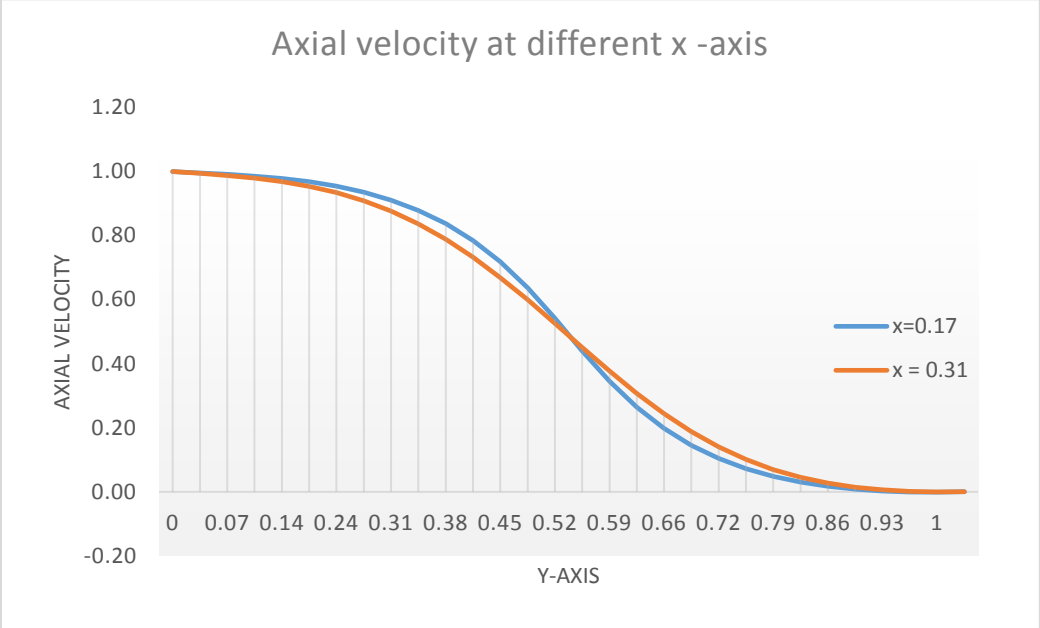

Figure 6: Axial Velocities at different x- axis

Figures 4, 5 and 6 are axial velocity profiles for the healthy blood vessel. The highest velocity reached at 1.0 m/s as we described the velocity for boundary condition @ x=0 and x=1. We can conclude that there is no disruption on blood flow however it reached velocity at 1.0 m/s because there is same pattern of parabolic flow along all the vessel.

Two-dimensional Flow Solution for a Stenotic Vessel in two-dimensional View

Mathematical Model

We used the COSMOL Multiphysics to analyze blood flow through the stenotic blood vessel as geometry given on equation (6) as [10]. This COSMOL Multiphysics is

set up with given the Navier-Stokes (7) and continuity (8) equations. The blood flow is considered as flow through a stenotic circular pipe.

$$f(x)=\begin{cases} 1-\frac{s}{2}\left[1+\cos\left(\frac{\pi}{e}\right)\left(x-\frac{L}{2}\right)\right] & if\ \ \frac{L}{2}-e\ \leq\ \mathrm{x} \leq\ \frac{L}{2}+e \\ 1 & otherwise \end{cases} \qquad (6)$$

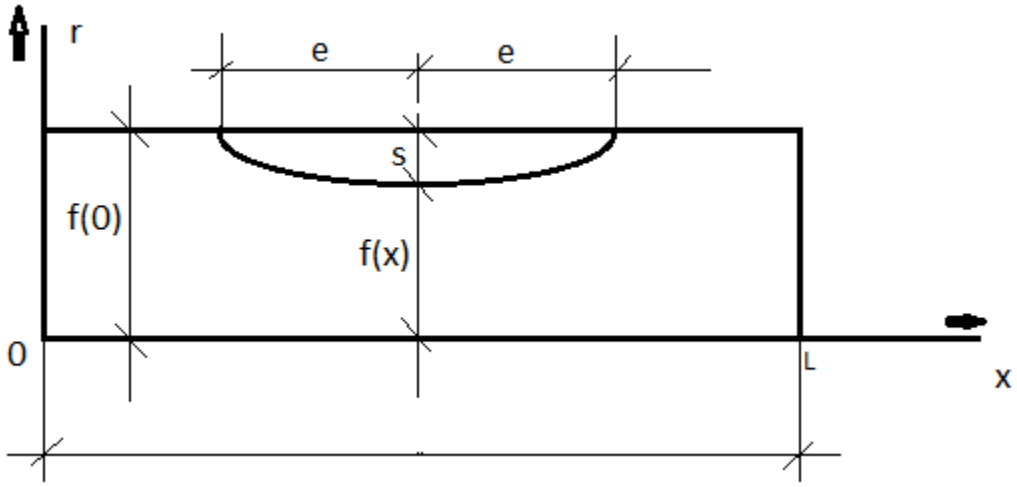

Figure 7. Geometry of an axially symmetrical stenosis

Governing Navier-Stokes equation:

$$\rho\,\frac{\partial U}{\partial t}+\rho\,(\mathrm{U}\,.\,\nabla\,)\mathrm{U}=\nabla\,.\,[\mathrm{PI}+\mu\,(\nabla+(\nabla U)^{T}]+\mathrm{F} \qquad (7)$$

Governing continuity equation:

$$\frac{\partial P}{\partial t}+\rho\nabla\,.\,(\mathrm{U})=0 \qquad (8)$$

Where ρ is density as 1000 kg/m^3
μ is fluid viscosity as 0.1 Pa*s
F is gravitational force as 0
P is as pressure
U is velocity vector of u and v

Initial conditions:

u (r, x, t) = 0, where u = axial velocity

v(r,x,t) = 0, v= radial velocity (9)

P(r,x,t) = 0

Boundary conditions:
u=v=0 no slip condition @ r = f(x)
u*n = 0, v = 0 @ r = 0 or as slip condition (10)
u = 1, v = 0 @ boundary when x=0 and x=4

We set up the good approximation of mesh size to obtain good convergence. The figure 8 has shown mesh with triangular element (14305), and edge elements (595), vertex elements (6). Geometry of a stenotic has been constructed in COSMOL Multiphysics as described in equation (4).

With appropriate boundary conditions, initial condition, Navier-Stokes and continuity equations, we found the result of velocity fields and pressure.

Numerical Solutions and Analysis

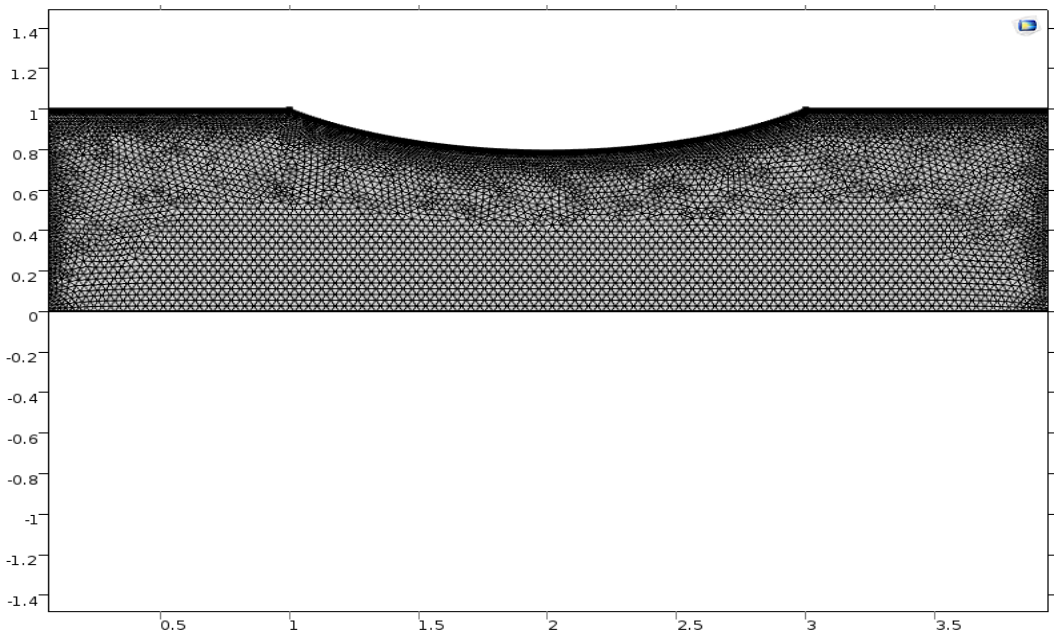

Figure 8: Mesh

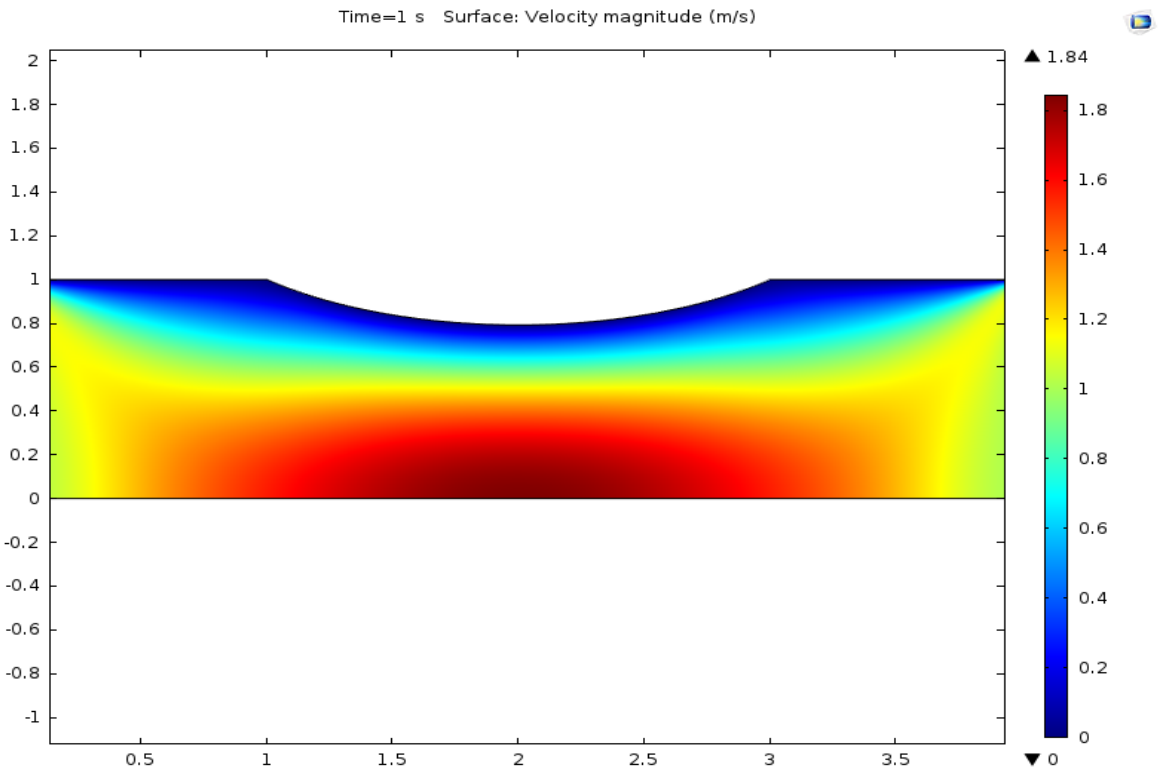

Figure 9: Velocity field at t=0.5 s

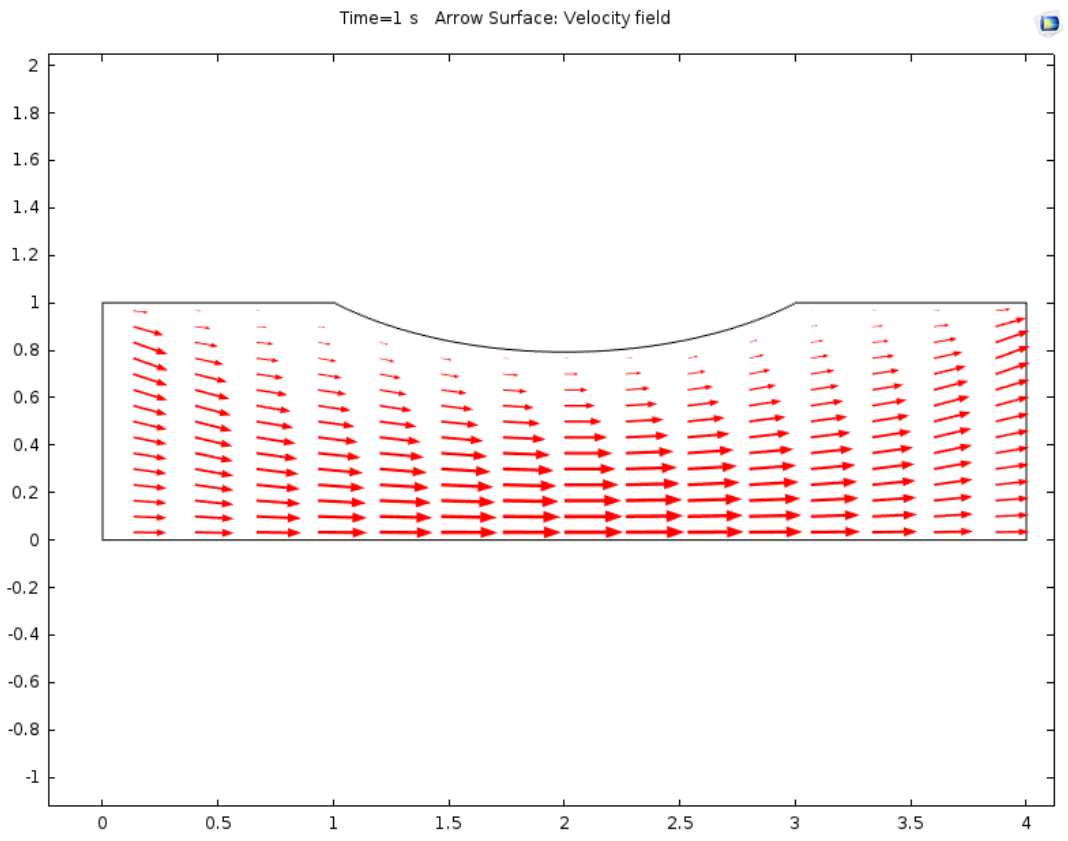

Figure 10: Velocity field at t=1.0 s

It is obvious that the stenosis size has a great influence on the flow field in a stenotic vessel. In my thesis, we are finding the maximum height of stenosis that could harmful to our body system. So we have analyzed for blood flow through the 20% stenotic vessel. The Figures 9 and 10 have shown velocity field for blood flow through a stenotic blood vessel. In Figure 9, the velocity field along the 20% stenotic blood vessel remained as parabolic flow as shown in the Figures 4,5 and 6 before and after the constriction zone. However, the Figure 10 has shown velocity field, and the velocity reached high of 1.8 m/s at the throat of the stenosis vessel which is in good agreement with those results achieved by Pontrelli G., who researched on steady flow through arterial stenosis [9]. For

validation, we took results from [9] as shown in Figure 11 where the dotted line represents for Non-Newtonian, and the continuous line represent for Newtonian.

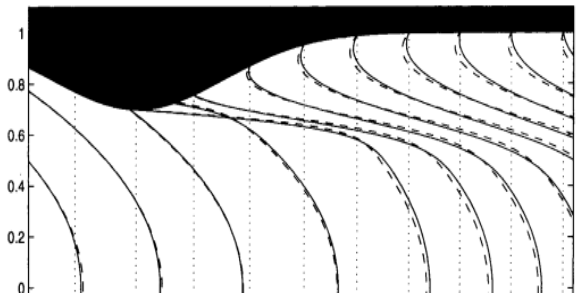

Figure 11: Velocity profile downstream and at stenotic zone.
from (http://www.iac.rm.cnr.it/~pontrell/pub/steno.pdf)

Figure 12 has shown pressure isobar. The same line represents same pressure value. The isobar is equally spaced means it has considerably same pressure. In Figure 12, it has clearly shown that the pressure measures along the constriction zone are lower than pressure upstream the constriction zone. We compare this results to those results done by Willie S. as shown in Figure 13 [12]. He presented the Navier-Stokes equations and analyzed by the finite element method for stenosis with various degrees of constriction. The Figure 13 represents the isobar pressure for 50% constriction vessel. The pressure at the constriction zone is low than the pressure before the constriction. It has clearly explained that the pressure drop will occurred at the constriction zone. Mustapha et al has displayed the result with pressure drop along the constriction zone [7].

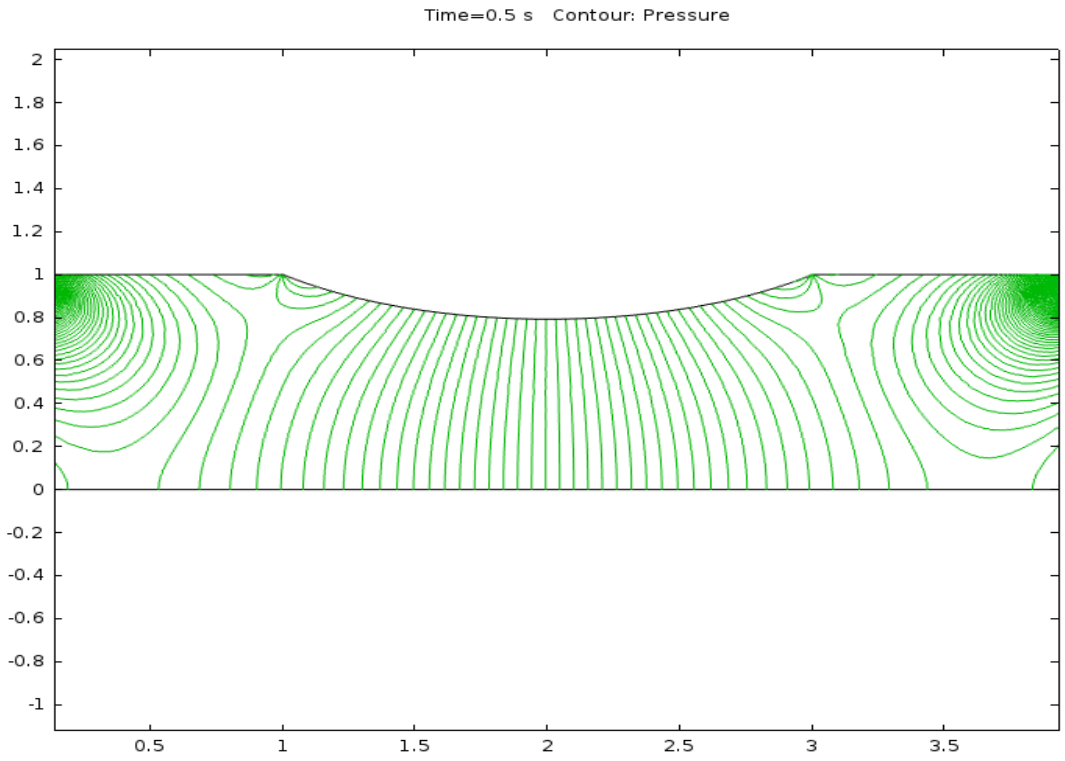

Figure 12: Isobar Pressure along center axis

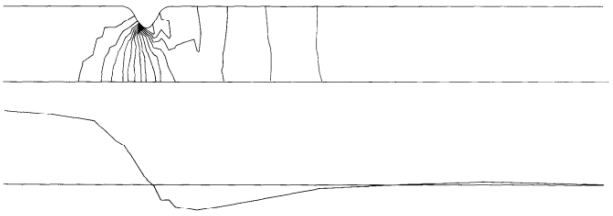

Figure 13: Isobar and Pressure variation along center axis for 50 % constriction from http://www.sciencedirect.com/science/article/pii/0307904X80901845

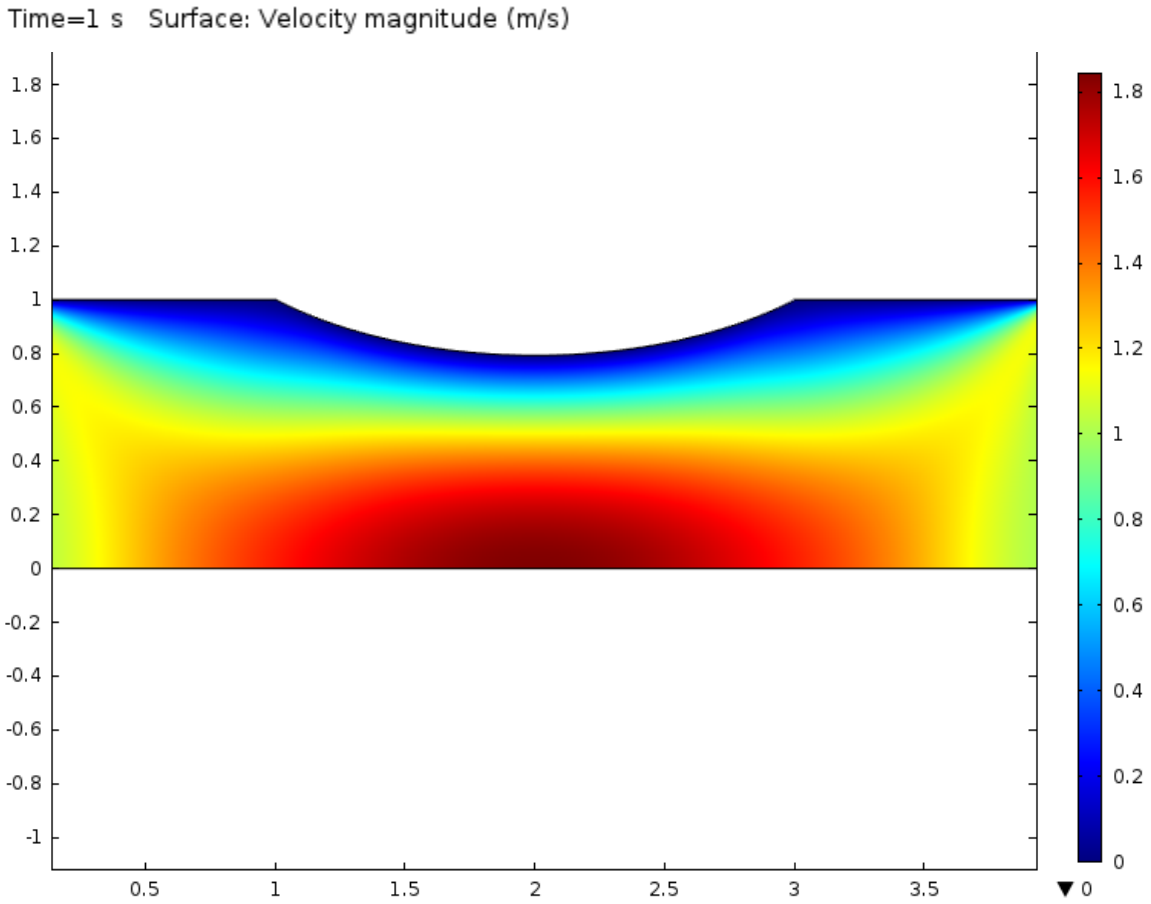

Figure 14: Velocity Field for time steps 0.001

We tooks small time steps as 0.01, and 0.001 to get results for velocity field, the solution is almost same. The Figure 14 has shown results for time steps 0.001. There is threshold for time step because we must check for stability issue. So we could not take too small time steps. We could not take large time step, it is tied to spatial mesh size. The results with time steps 0.001 from Figure 14 is comparatively same velocity results from Figure 9.

Two-dimensional Flow Solution for a Stenotic Vessel in 3-D View

Mathematical Model

Under the assumption that no gravitational forces act on blood flow, blood density is constant, blood flow is unsteady in x and r direction with cylindrical coordinate (r, x), blood flow is Newtonian viscous incompressible through an axially symmetric stenosis, and the artery is a rigid circular tube, the geometry of stenosis will be described as Figure 15 [10].

$$f(x)=\begin{cases} 1-\frac{s}{2}\left[1+\cos\left(\frac{\pi}{e}\right)\left(x-\frac{L}{2}\right)\right] & if\ \ \frac{L}{2}-e \le x \le \frac{L}{2}+e \\ 1 & otherwise \end{cases} \quad (6)$$

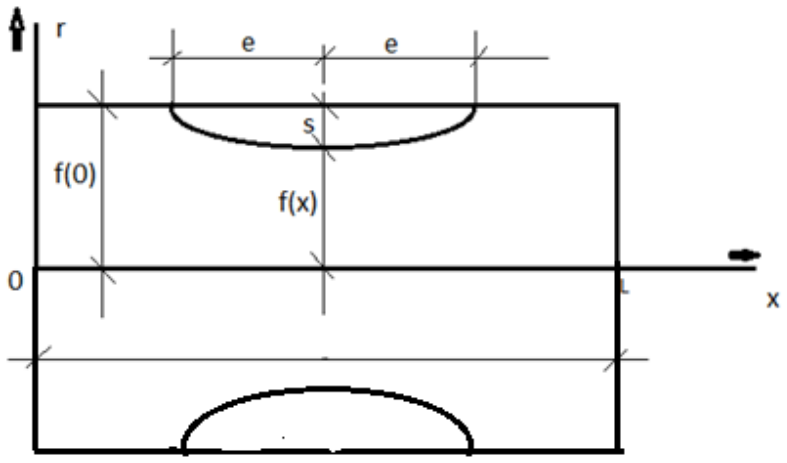

Figure 15. Geometry of an axially symmetrical stenosis

Where f (x)- Tube radius with stenosis

f (0)- Tube radius without stenosis

s - The maximum height of the stenosis

According to the above assumption, the non-dimensional incompressible Navier-Stokes equation can be written as continuity equation (11) and momentum equations (12), (13).

The fluid flow is governed by the Continuity equation

$$\frac{\partial u}{\partial x} + \frac{v}{r} + \frac{\partial v}{\partial r} = 0 \qquad (11)$$

The momentum equation in the radial direction

$$\frac{\partial v}{\partial t} + u\frac{\partial v}{\partial x} + v\frac{\partial v}{\partial r} = -\frac{\partial P}{\partial r} + \frac{1}{Re}\left(\frac{\partial^2 v}{\partial r^2} + \frac{1}{r}\frac{\partial v}{\partial r} + \frac{\partial^2 v}{\partial x^2} - \frac{v}{r^2}\right) \qquad (12)$$

The momentum equation in the axial direction

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x} + v\frac{\partial u}{\partial r} = -\frac{\partial P}{\partial x} + \frac{1}{Re}\left(\frac{\partial^2 u}{\partial r^2} + \frac{1}{r}\frac{\partial u}{\partial r} + \frac{\partial^2 u}{\partial x^2}\right) \qquad (13)$$

Where P = the pressure is described as homogenous Neumann boundary condition, that implies as the pressure is described as a constant.

u (x, r) = the component of velocity in x- direction

v (x, r) = the component of velocity in r- direction

Boundary condition:

u=v=0 no slip condition @ r = f(x)
u*n=0, v=0 @ r=0 or as slip condition (14)
u = c1 @ boundary when x=0 and x=L

The initial velocities:
u(x,r,t) = 0 at t = 0 (15)
v(x,r,t) = 0 at t = 0

Numerical Solutions and Analysis

Wall shear stress and velocity fields are analyzed for two-dimensional blood flow through a stenotic vessel with given Navier-Stokes and continuity equations (11), (12), (13)

in cylindrical coordinate. The geometry of a blood vessel is created with the software (Solidworks) as equation (6). Then, it is exported to computational software (ICEM ANSYS). The numerical simulation is done computer software(Fluent) with considering following physical parameters.

ρ is density as 1000 kg/m^3

μ is fluid viscosity as 0.1 Pa*s

With given boundary and initial condition for velocity described on equations (16), (17), and Pressure as constant, we analyzed velocity field and wall shear force for blood flow through 30% and 80% blockage.

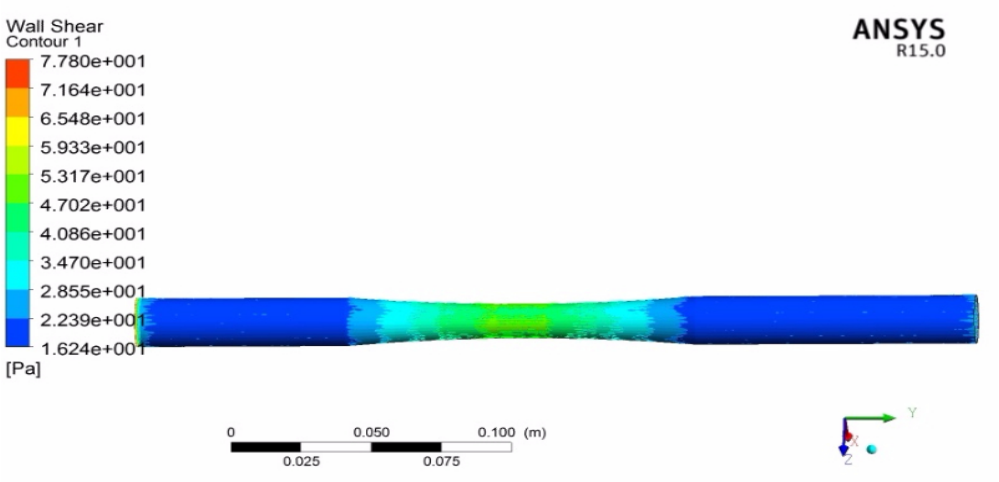

Figure 16: Wall Shear Stress for 30% blockage

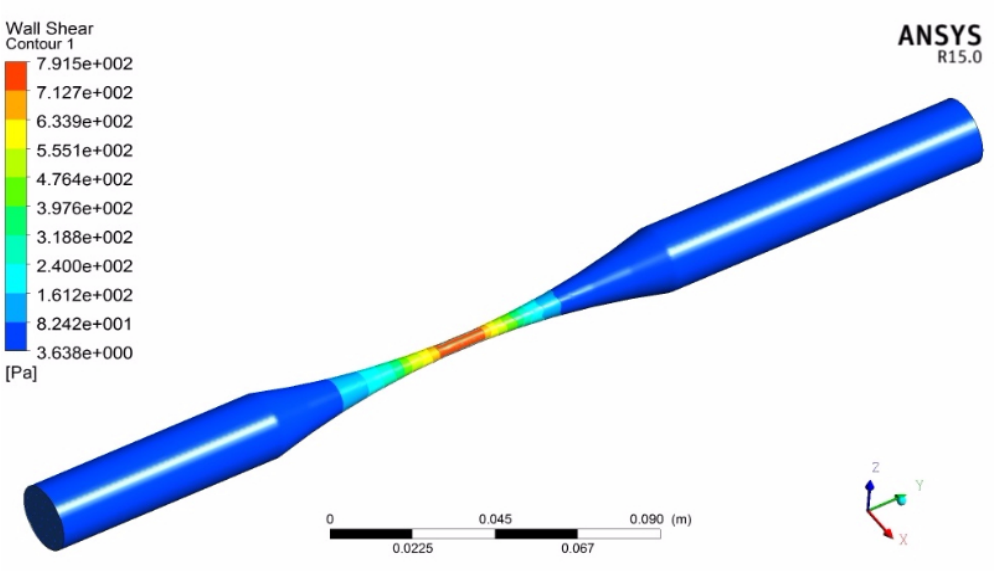

Figure 17: Wall Shear Stress for 80% blockage

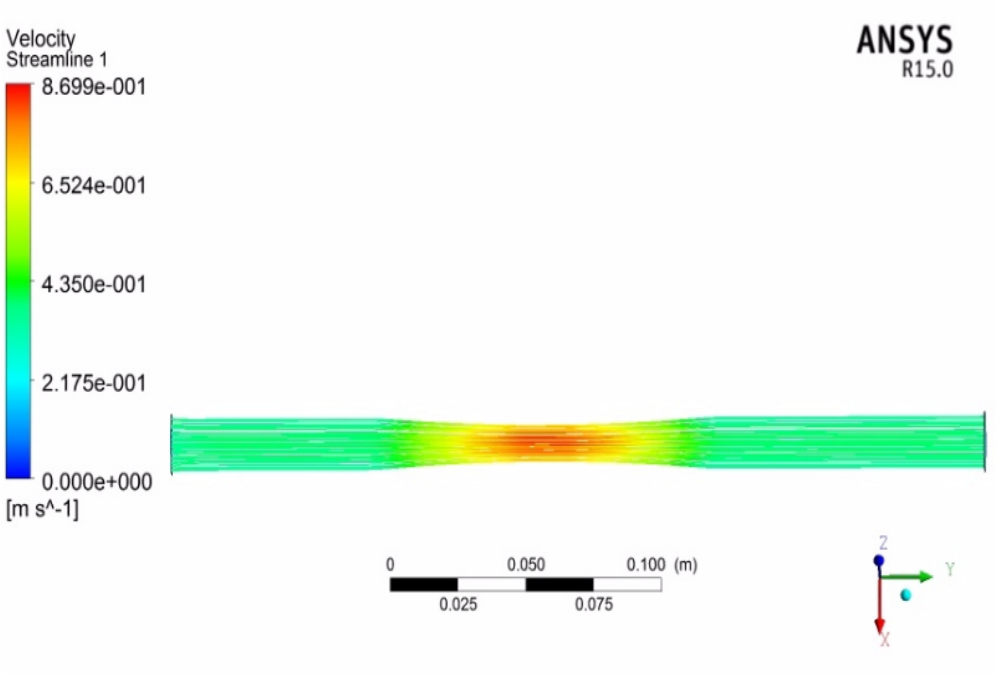

Figure 18: Velocity Field for 30% blockage

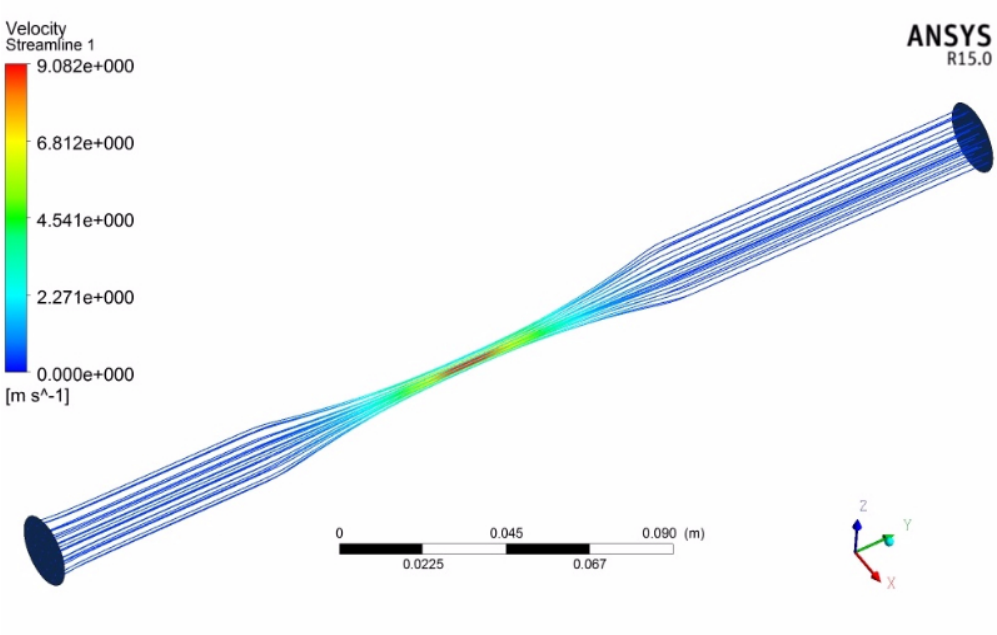

Figure 19: Velocity Field for 80% Blockage

Figure 16 and 17 have shown wall shear stress (WSS) within a range of 16.24 - 77.80 Pa for 30% blockage and 3,64 - 784 Pa for 80% blockage. The highest wall shear stress is 784 Pa which is very critical to our human body. Figures 18 and 19 have shown the maximum velocity at .87 m/s for 30% blockage, and 9.8 m/s for 80% blockage.

CHAPTER VI: CONCLUSION

In my work, a two-dimensional axisymmetric mathematical model is presented to study for blood flow through the healthy and an axisymmetric modeled arterial stenosis. The numerical simulation used the finite difference method for the healthy vessel and the finite element method for a stenotic vessel. The blood flow through a 20% percent stenosis are analyzed with finite element method using COSMOL Multiphysics. The velocity reached at 1.8 m/s at the throat of a stenotic vessel which are risk for aortic blood vessel. The pressure along the constriction zone is lower than pressure before the constriction zone that conclude that there is pressure drop along the constriction zone.

For more accuracy, we used the finite element method with ANSYS-Fluent to analyze blood behavior for 30% and 80% blockage. The wall shear stress reached at 784 Pa for 80% of blockage, and 77.8 Pa for 30% blockage. The velocity reached at 9.8 m/s for 80% blockage and .87 m/s for 30% blockage. The normal velocity range for aortic blood vessel is 0.2 to 0.98m/s. From this work, we can recommend to the medical field that even 20% of stenosis could increase the velocity at the throat of stenosis that is harmful the blood circulation in our body. The decreasing of pressure as the stenosis zone starts could be considered as initiation of compromising body ability. To maintain sufficient blood flow to the organ, the velocity increases to compensate lower body pressure. As the result, we have seen the maximum velocity 1.8 m/s at the throat of stenotic vessel that are critical to our body. As the flow rate increases the wall shear stress increases. In my thesis, we have seen reaching the highest wall shear stress where the maximum velocity reached.

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APPENDICES

```
function Maha_navierstokes
%Maha_NAVIERSTOKES
```

```
%-------------------------------------------------------------------------
-
Re = 1e2;    % Reynolds number
dt = 1e-2;    % time step
tf = 4e-0;    % final time
lex = 1;       % width of box
ley = 1;       % height of box
nx = 90;      % number of x-gridpoints
ny = 90;      % number of y-gridpoints
nsteps = 10;  % number of steps with graphic output
%-------------------------------------------------------------------------
-
nt = ceil(tf/dt); dt = tf/nt;
x = linspace(0,lex,nx+1); hx = lex/nx;
y = linspace(0,ley,ny+1); hy = ley/ny;
[X,Y] = meshgrid(y,x);
%-------------------------------------------------------------------------
-
% initial conditions
U = zeros(nx-1,ny); V = zeros(nx,ny-1);

% boundary conditions
u1 = 0;    v1 = 0;
u2 = 0;      v2 = 0;
u3 = 1; v3 = 0;
u4 = 1; v4 = 0;



%-------------------------------------------------------------------------
-
Ubc = dt/Re*([2*u2(2:end-1)' zeros(nx-1,ny-2) 2*u1(2:end-1)']/hx^2+...
      [u3;zeros(nx-3,ny);u4]/hy^2);
Vbc = dt/Re*([v2' zeros(nx,ny-3) v1']/hx^2+...
      [2*v3(2:end-1);zeros(nx-2,ny-1);2*v4(2:end-1)]/hy^2);

fprintf('initialization')
Lp = kron(speye(ny),K1(nx,hx,1))+kron(K1(ny,hy,1),speye(nx));
Lp(1,1) = 3/2*Lp(1,1);
perp = symamd(Lp); Rp = chol(Lp(perp,perp)); Rpt = Rp';
Lu = speye((nx-1)*ny)+dt/Re*(kron(speye(ny),K1(nx-1,hx,2))+...
     kron(K1(ny,hy,3),speye(nx-1)));
peru = symamd(Lu); Ru = chol(Lu(peru,peru)); Rut = Ru';
Lv = speye(nx*(ny-1))+dt/Re*(kron(speye(ny-1),K1(nx,hx,3))+...
     kron(K1(ny-1,hy,2),speye(nx)));
perv = symamd(Lv); Rv = chol(Lv(perv,perv)); Rvt = Rv';
Lq = kron(speye(ny-1),K1(nx-1,hx,2))+kron(K1(ny-1,hy,2),speye(nx-1));
perq = symamd(Lq); Rq = chol(Lq(perq,perq)); Rqt = Rq';

fprintf(', time loop\n--20%%--40%%--60%%--80%%-100%%\n')
for k = 1:nt
   % treat nonlinear terms
   gamma = min(1.2*dt*max(max(max(abs(U)))/hx,max(max(abs(V)))/hy),1);
   Ue = [u3;U;u4]; Ue = [2*u2'-Ue(:,1) Ue 2*u1'-Ue(:,end)];
   Ve = [v2' V v1']; Ve = [2*v3-Ve(1,:);Ve;2*v4-Ve(end,:)];
```

```
   Ua = avg(Ue')'; Ud = diff(Ue')'/2;
   Va = avg(Ve);   Vd = diff(Ve)/2;
   UVx = diff(Ua.*Va-gamma*abs(Ua).*Vd)/hx;
   UVy = diff((Ua.*Va-gamma*Ud.*abs(Va))')'/hy;
   Ua = avg(Ue(:,2:end-1));   Ud = diff(Ue(:,2:end-1))/2;
   Va = avg(Ve(2:end-1,:)')'; Vd = diff(Ve(2:end-1,:)')'/2;
   U2x = diff(Ua.^2-gamma*abs(Ua).*Ud)/hx;
   V2y = diff((Va.^2-gamma*abs(Va).*Vd)')'/hy;
   U = U-dt*(UVy(2:end-1,:)+U2x);
   V = V-dt*(UVx(:,2:end-1)+V2y);

   % implicit viscosity
   rhs = reshape(U+Ubc,[],1);
   u(peru) = Ru\(Rut\rhs(peru));
   U = reshape(u,nx-1,ny);
   rhs = reshape(V+Vbc,[],1);
   v(perv) = Rv\(Rvt\rhs(perv));
   V = reshape(v,nx,ny-1);

   % pressure correction
   rhs = reshape(diff([u3;U;u4])/hx+diff([v2' V v1']')'/hy,[],1);
   p(perp) = -Rp\(Rpt\rhs(perp));
   P = reshape(p,nx,ny);
   U = U-diff(P)/hx;
   V = V-diff(P')'/hy;




   % visualization
   if floor(25*k/nt)>floor(25*(k-1)/nt), fprintf('.'), end
   if k==1|floor(nsteps*k/nt)>floor(nsteps*(k-1)/nt)
      % stream function
      rhs = reshape(diff(U')'/hy-diff(V)/hx,[],1);
      q(perq) = Rq\(Rqt\rhs(perq));
      Q = zeros(nx+1,ny+1);
      Q(2:end-1,2:end-1) = reshape(q,nx-1,ny-1);
      clf, contourf(avg(x),avg(y),P',20,'w-'), hold on
      contour(x,y,Q',20,'k-');
      Ue = [u2' avg([u3;U;u4]')' u1'];
      Ve = [v3;avg([v2' V v1']);v4];
      Len = sqrt(Ue.^2+Ve.^2+eps);
      quiver(x,y,(Ue./Len)',(Ve./Len)',.4,'k-')
      hold off, axis equal, axis([0 lex 0 ley])
      p = sort(p); caxis(p([8 end-7]))
      title(sprintf('Re = %0.1g   t = %0.2g',Re,k*dt))
      drawnow
   end
end
fprintf('\n')



%=========================================================================
=
```

```
function B = avg(A,k)
if nargin<2, k = 1; end
if size(A,1)==1, A = A'; end
if k<2, B = (A(2:end,:)+A(1:end-1,:))/2; else, B = avg(A,k-1); end
if size(A,2)==1, B = B'; end
end

function A = K1(n,h,a11)
% a11: Neumann=1, Dirichlet=2, Dirichlet mid=3;
A = spdiags([-1 a11 0;ones(n-2,1)*[-1 2 -1];0 a11 -1],-1:1,n,n)'/h^2;
end
save('parameters')
end
```