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Computational Analysis of Blood Flow in Abdominal Aortic Artery Affected by Acute Stenosis

H. Girija Bai[1] , K.B. Naidu[2] and G. Vasanth Kumar[3]

[1,2]Department of Mathematics, [3]Department of Aeronautics,
Sathyabama University, Chennai - 600 119, Tamil Nadu, India.

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In this paper, blood flow in abdominal aortic artery which is affected by acute stenosis is simulated numerically. Computational analysis of variations in the blood flow due to anti-platelet drug are studied by applying different densities. Analysis is done using softwares Gambit and Fluent. The results of this study help us to visualise the consequences of acute stenosis before and after treatment. The simulation results may provide important guidance for treatments for stenosis and surgical interventions that seek to improve blood flow in affected arteries.

Key words: Acute Stenosis; Antiplatelet drug; Computational Analysis; Numerical Simulation; Density Variation.

The majority of deaths are caused by cardiovascular diseases which are connected with some form of irregular ûow in arteries. The arteries are living organs that can adjust to and change with the varying hemodynamic conditions. Atherosclerosis is a condition that develops when a substance called plaque builds up in the walls of the arteries. This buildup narrows the arteries, making it harder for the flow of blood. The obstruction may damage the internal cells of the wall and may lead to further growth of stenosis. This vascular disease is of frequent occurrence, particularly in mammalian arteries. If this disease takes a severe form, it may lead to fatality. If a blood clot forms, it can stop the blood flow. This can cause a heart attack or stroke. If this clot cuts off the blood flow completely, the part of the heart muscle supplied by that artery begins to die. Treatment of atherosclerosis of the carotid artery is dependent on the severity and degree of the disease. The most likely cause is uncontrolled hypertension. Since the arterial tube is constricted, the recirculation of flow occurs, this leads to abnormal flow of blood and leads to stroke. To overcome that abnormality, the heart pumps blood very fast and the flow gets aggravated that leads to HBP.

To prevent or to avoid further occurrence of stroke, medication is vital. If a blood clot forms inside an artery, it blocks the flow of blood to the tissue that the artery supplies, which can damage the tissue. So the main aim is to remove or to prevent the blood to clot.

Medications used to manage atherosclerotic disease include:

- Antiplatelet ('blood thinner') agents (eg, aspirin, ticlopidine, clopidogrel)
- Anticoagulants (eg, warfarin).

Antiplatelet agents are medications that block the formation of blood clots by preventing the clumping of platelets. Antiplatelet therapy reduces the incidence of stroke in patients at high risk for atherosclerosis and in those with known symptomatic cerebro vascular disease. There are three types of antiplatelet agents:

* To whom all correspondence should be addressed.
E-mail: girijanameprakash@gmail.com

1. Aspirin,
2. Tthienopyridines, and
3. Glycoprotein IIb/IIIa inhibitors.

These agents differ in the way they work, their influence, how rapidly they work, and their cost. Aspirin has an important inhibitory effect on platelets in the blood. Aspirin belongs to a class of medications called nonsteroidal anti-inflammatory drugs (NSAIDs). Aspirin is widely used either alone or in combination with other antiplatelet agents to prevent blood clots from forming in arteries. Aspirin prevents blood from clotting by blocking the production by platelets of thrombosane A-2, the chemical that causes platelets to clump.

Heart attack prevention is of two types, primary and secondary. Preventing the first heart attack is called primary prevention. Preventing further heart attacks among patients who already have had a heart attack is called secondary prevention. Long-term, daily aspirin (75-325 mg/d) has been shown to reduce the risk of second heart attacks and improve survival among both men and women. Therefore, survivors of heart attacks usually take daily low dose (75 mg-160 mg/d) aspirin indefinitely to prevent further heart attacks as well as strokes. Aspirin prevents blood clots from forming inside arteries affected by atherosclerosis, but aspirin does not prevent atherosclerosis. Due to its antiplatelet action and fast acting aspirin is easy to use and safe at lower doses. Prolonged use of aspirin at higher doses can cause stomach ulcers, and can also prolong bleeding. For those patients have already liver and kidney disease, this drug can impair function of the kidney and liver. Aspirin, non responders have higher rates of heart attacks, strokes, and death than aspirin responders. The causes of aspirin failure include inadequate dosing, not taking the medication regularly, concurrent intake of other NSAIDs, etc. Aspirin taken long-term is an important part, but NOT the only measure for preventing heart attacks.

Some patients with heart attacks are treated with thrombolytic agents (medications that dissolve clots) to open blocked arteries. It is important to make the distinction that aspirin generally does not open an existing blood clot, but it acts to prevent propagation of the existing clot and the formation of new ones. In all of these instances, there is a risk that blood clots will form again in the arteries, leading to further heart attacks. In all of these cases, aspirin has been shown to be beneficial in preventing new clots, thus reducing the risk of heart attacks and improving both short and long-term survival.

The hemodynamic behavior of the blood flow in arterial stenoses, the impact of different drugs and the antiplatelet reactions bears some important aspects due to engineering interest as well as feasible medical applications. A simple pulsatile flow in an artery with a constriction are analyzed by [1,2]. The antiplatelet drugs was suggested by [3,4]. Flow in a tube with an occlusion by using finite difference scheme for steady and unsteady flow is investigated [5]. The effect of different shaped stenoses suggested by [6,7,8]. Cerebral aneurysms models [9] and shear stress was discussed by [10,11]. The role of drug in vascular disease was suggested by [12,13]. A finite element analysis of simple pulsatile flow in a constricted vessel analyzed by [14]. The blood flow dynamics in a stenosed, subject-specific, carotid bifurcation using the spectral element method numerically analyzed by [15,16]. The pulsatile non-Newtonian flow of blood through stenotic artery [17]. The effects of overlapping blood flow characteristics in a narrow artery [18,19]. The simulation of complex dynamic processes that appear in nature or in industrial applications poses a lot of challenging mathematical problems, opening a long road from the basic problem, to the mathematical modelling, the numerical simulation, and finally to the interpretation of results.

In this paper, the pulsatile flow of a viscous fluid in two dimensional constricted vessel is considered. Due to antiplatelet drug the density of blood gets thinner and leads to the motion of fluid freely inside the arteries. The density variation is given as input under steady and unsteady flow conditions. The study is undergone for acute stenosis in abdominal aortic region with bifurcations at different densities (1060 kg/m^3 – normal, 1030 and 1000 kg/m^3 diluted) and different pressure variations (mild 12000pa = 90.0074mmHg, severe 24000pa = 180.0148mmHg). Using MIMICS, MATLAB and CFD, the analysis of flow patterns was computed and compared which has not been thoroughly investigated so far. Limitations on the amount of the constriction are

ignored. Since arterial wall is gently elastic, we neglect the wall dispensability. Change in diameter in arteries is on the order of 10% [20,21] so the error in fixed diameter is minute.

Governing Differential Equations and Boundary Conditions

Equations of momentum and mass conservation for an incompressible Newtonian fluid can be written as: (absence of body forces)

$$\nabla \cdot \vec{v} = 0 \quad ...(1)$$

$$\rho\left(\frac{\partial \vec{v}}{\partial t} + \vec{v}.\nabla\vec{v}\right) = -\nabla p + \mu\nabla^2\vec{v} \quad ...(2)$$

Where: ρ - density of blood, $\vec{v}$ -Velocity Fields, p - pressure, μ = co-efficient of viscosity.

By giving input pressure 12,000pa, and 24,000pa the velocity is calculated and used as an input, u = u(t), v = 0 on inflow segment, u = v = 0, on stenosed vessel.

Blood density ρ = 1060, 1030, 1000 [kg/m^3] and dynamic viscosity η = 0.003 [kg/ms] (Poiseuille). By giving boundary conditions, the governing differential equations were solved numerically using finite volume method.

A mathematical model has been developed for studying steady and unsteady blood flow through the abdominal aortic stenosed vessel under different density variations due to antiplatelet drug. By considering acute stenosis in abdominal aorta, expressions for the velocity profile, wall shear stress, streamline pattern and pressure gradient have been derived numerically under finite volume method. The above said quantities are computed for a specific set of values for different parameters involved in the model analysis. This serves as an illustration of the validity of the mathematical model developed here. The results estimated on the basis of the computation are presented graphically. The obtained results for different parameters involved in the problem, shows that the flow is appreciably influenced by density variation at different time seconds.

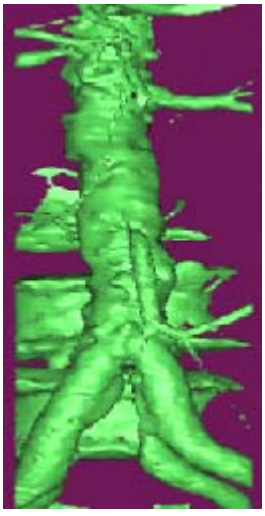

Fig. 1. Abdominal Aortic Artery affected by stenosis with iliac bifurcations. Patient's original artery

RESULTS AND DISCUSSION

Using MIMICS, patient abdominal artery affected by stenosis is generated in Fig 1.

Model geometry was created with single acute stenosis with left and right iliac bifurcations. The domain table. 1 is imported to CFD for analysis and exported to fluent for solving the problem using finite volume method.

Unsteady and steady flow analysis is carried out by considering pave mesh of size 0.01, the nodal values are given in Table 2 and its total

Table 1. Domain of the Geometry

	Min. (mm)	Max(mm)
X	0.0009	0.004
Y	0.001	0.007

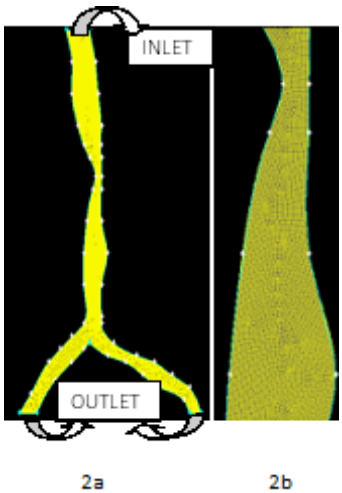

Fig. 2. (2a)Model with bifurcations. (2b)Quadrilateral mesh of size 0.01

volume and area is given in table 3 and 4 respectively.

Velocity Magnitude

The normal blood density in human 1060 kg/m^3 is compared with the diluted densities 1000 kg/m^3 and 1030 kg/m^3. According to different densities and pressures 12,000pa = 90.0074mmHg, 24,000pa = 180.0148mmHg, the inlet velocity vary from 4.758309m/s to 6.72927m/s given in table 5.

Due to constriction of vessel geometry, the motion of fluid varies at each and every node gives the minimum and maximum values within the domain table 6. The maximum velocity occurs mainly in the neck region of the stenosis and the contour of the flow is shown in fig. 3.

The vortices and recirculation region shows the stagnation flow occurs in the downstream region of the constricted stenotic vessel. By considering different positions inside the domain, changes in transverse velocities are shown graphically.

By considering some interior points we visualize how the velocity is disturbed due to plaque. The arrows in Fig. 3h and Fig 3i show that

Table 2. Number of elements

Nodes	24936
Mixed wall faces	1698
Mixed interior faces	47188
Quadrilateral cells	24041

Table 3. Volume statistics

Min. Volume (m3)	Max. Volume (m3)	Total volume (m3)
3.578908e-005	2.268191e-004	2.424768

Table 4. Area statistics

Min. Face area(m2)	Max. Face area(m2)
5.027221e-003	1.778156e-002

Table 5. Inlet velocities for different densities and pressures.

Velocity	1000	1030	1060
12000pa	4.898979	4.827109	4.758309
24000pa	6.928204	6.826563	6.72927

Table 6. Maximum and minimum values of velocities within the domain

Inlet Velocity for different densities and pressures	12000 Unsteady	12000 Steady	24000 Unsteady	24000 Steady
1000,12000= -4.898979,24000= -6.928201	6.622498	6.698445	9.780181	9.926631
1030,12000= -4.827109,24000 = -6.826563	6.636377	6.605142	9.725203	9.808622
1060,12000= -4.75831,24000= -6.729264	6.535642	6.539557	9.692457	9.675941

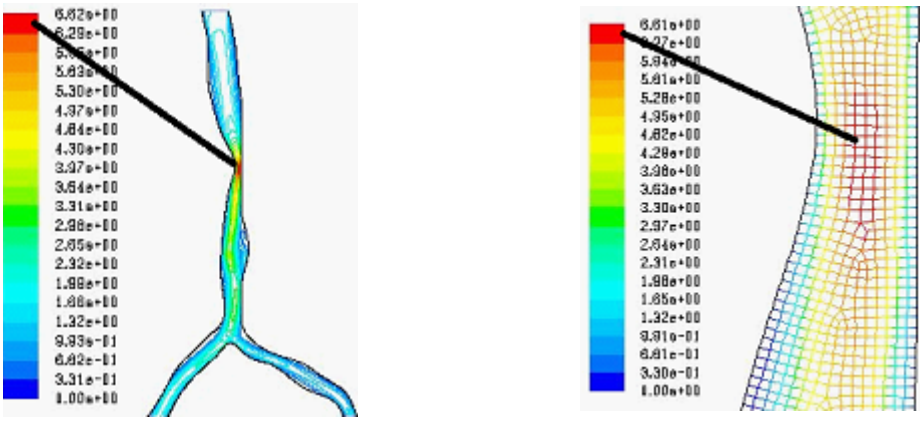

Fig. 3. Contours of Velocity Magnitude

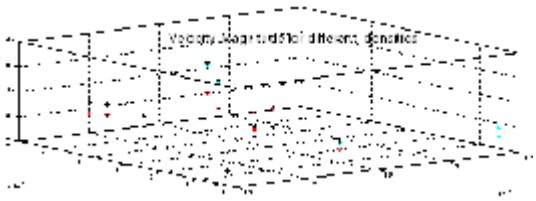

Fig. 3a. Minimum and Maximum Velocity Magnitude within the domain for different densities

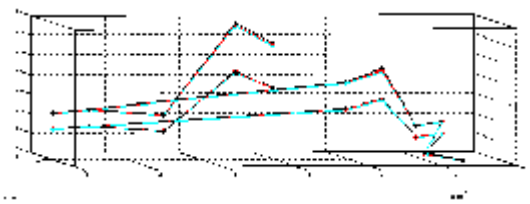

Fig. 3b. Velocity distribution inside the domain at different positions

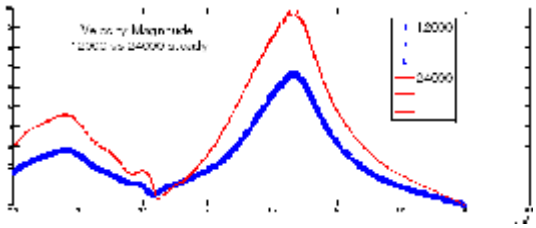

Fig. 3c. Comparision of transverse velocity magnitude under steady state for pressure variations.

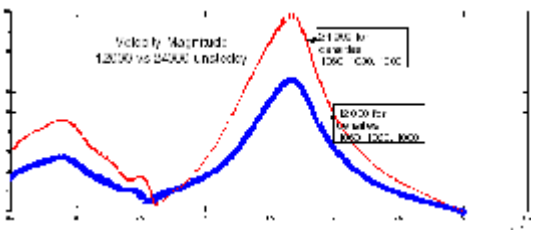

Fig. 3d. Comparision of transverse velocity magnitude under unsteady state for pressure variations.

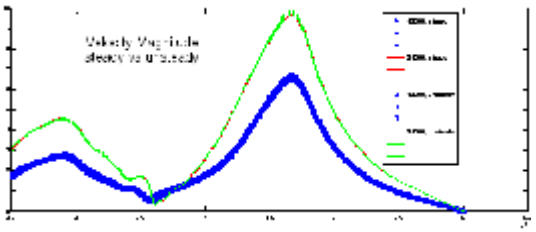

Fig. 3e. Comparision of transverse velocity magnitude for steady and unsteady flow for two pressure variations.

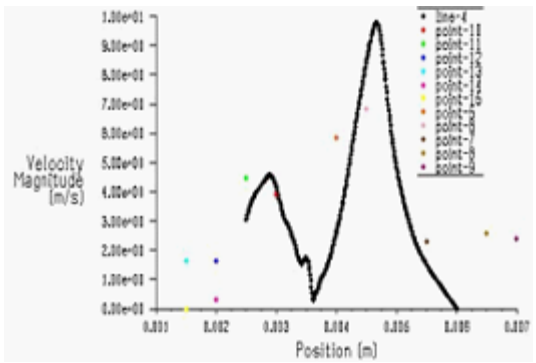

Fig. 3f. Velocity magnitude along y direction

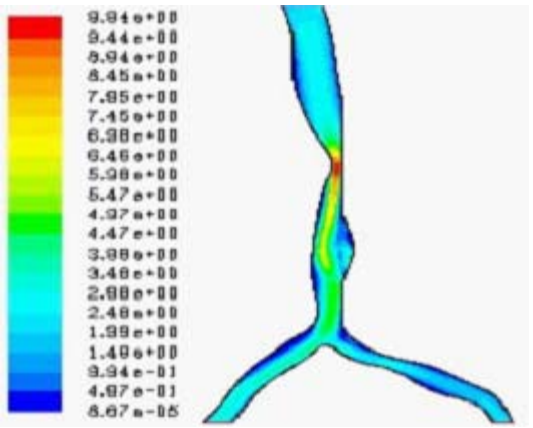

Fig. 3g. Streamlines of velocity magnitude

from the upstream region the velocity increases, and it reaches maximum at the region of stenosis. Figure 3j graphically shows the velocity distribution inside the domain at different position along y direction.

Dynamic Pressure

The force applied to artery walls is measurement as blood pressure. Blood pressure stays high for a long time can cause health problems but normally it rises and falls throughout the day. Normal blood pressure is represented by two numbers 120/80. The systolic pressure indicates the top number and the diastolic pressure indicates bottom number. High blood pressure occurs if systolic e" 140 or diastolic e" 90. The unit of pressure, mmHg defined as 1millimetre of mercury or 1 Torr. 1 mmHg =133. 322368Pascals. For most heart diseases the risky factor is High Blood Pressure (HBP). HBP can quietly damage our body, inner lining cells of our arteries. HBP leads a disease called atherosclerosis or hardening of the arteries, artery walls thick and stiff,. This damage can cause many problems, including heart attack, kidney failure, heart failure, stroke, chest pain (angina), blocked arteries in our legs or arms (peripheral artery disease), eye damage and aneurysms.

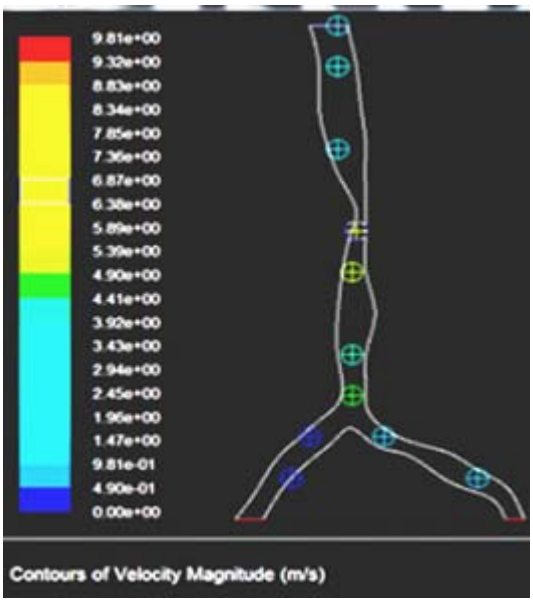

Fig. 3h. Velocity in the region of stenosis

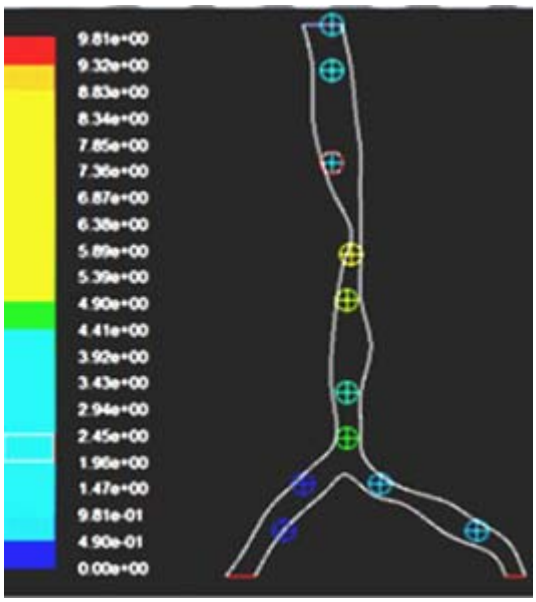

Fig. 3i. Velocity above the region of stenosis

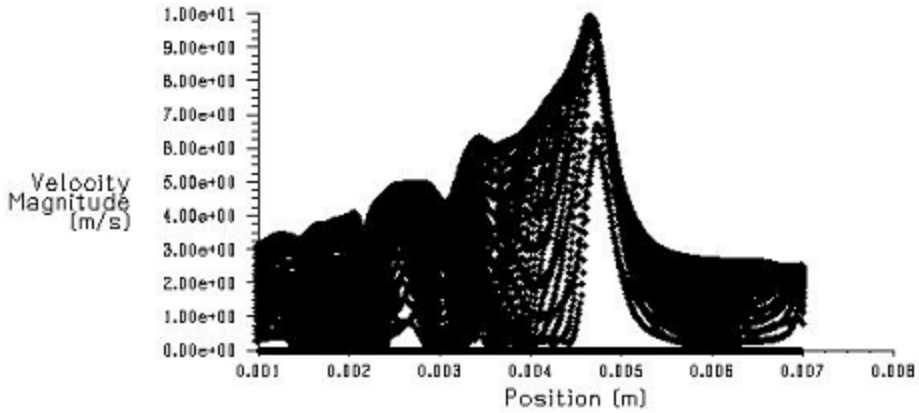

Fig. 3j. Velocity Magnitude of the domain

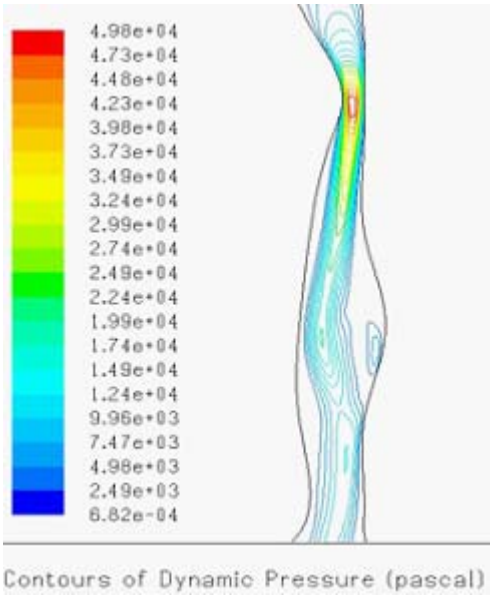

Fig. 4a. Contours of Dynamic Pressure

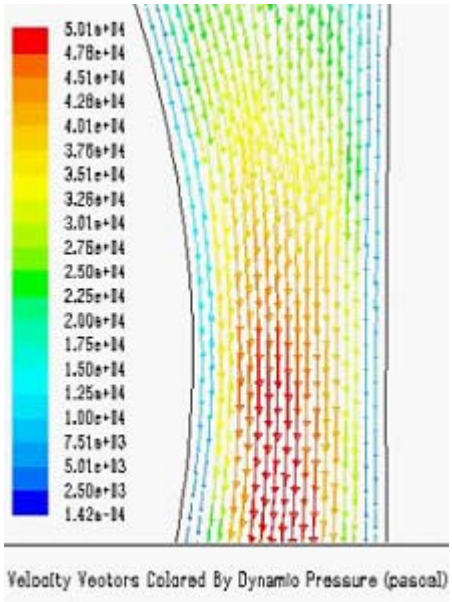

Fig. 4b. Streamlines of Dynamic Pressure

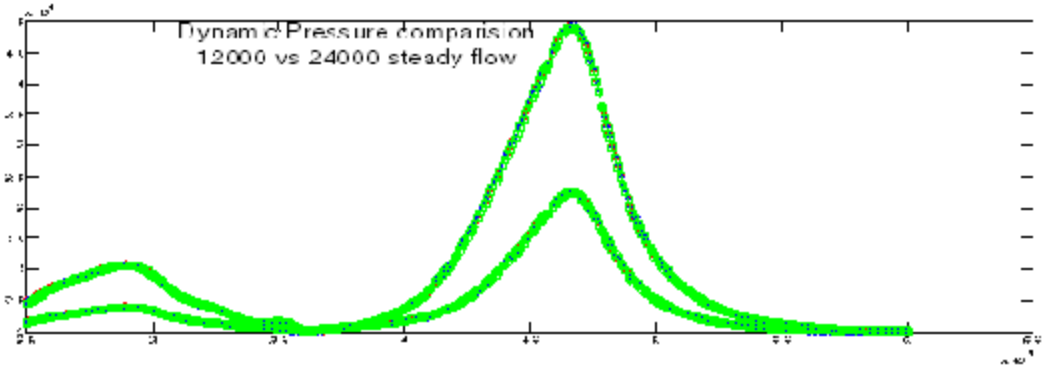

Fig. 4c. Comparision of transverse Dynamic Pressure at steady state for two pressure variations.

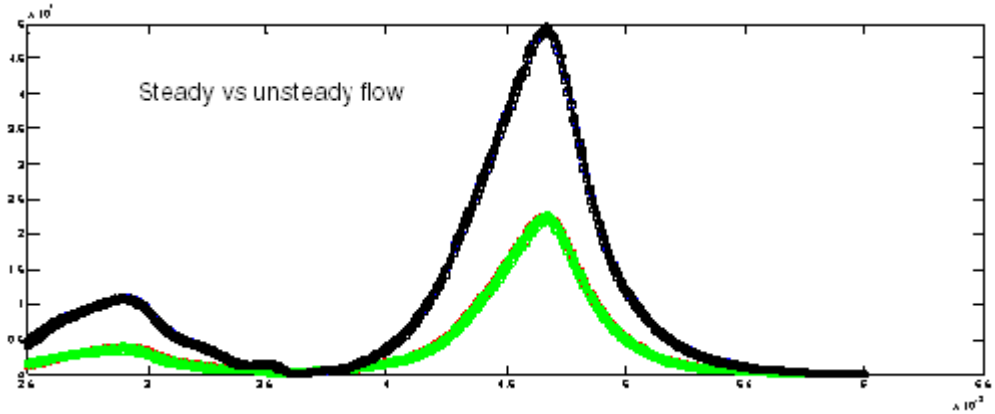

Fig. 4d. Comparision of transverse Dynamic Pressure under Steady vs unsteady flow for pressure variations

By giving input pressures 12,000pa = 90.0074mmHg (normal diastolic), 24,000pa = 180.0148mmHg (high systolic), dynamic pressure variations were calculated and tabulated in table 7.

There is much variation in pressures in the regions of constriction mainly with the increase of δ.

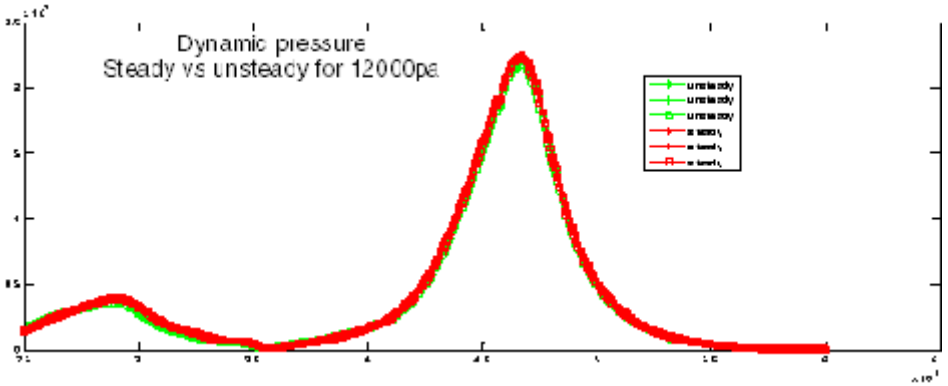

Fig. 4e. Comparision of transverse Dynamic Pressure under steady vs unsteady flow for 12000pa

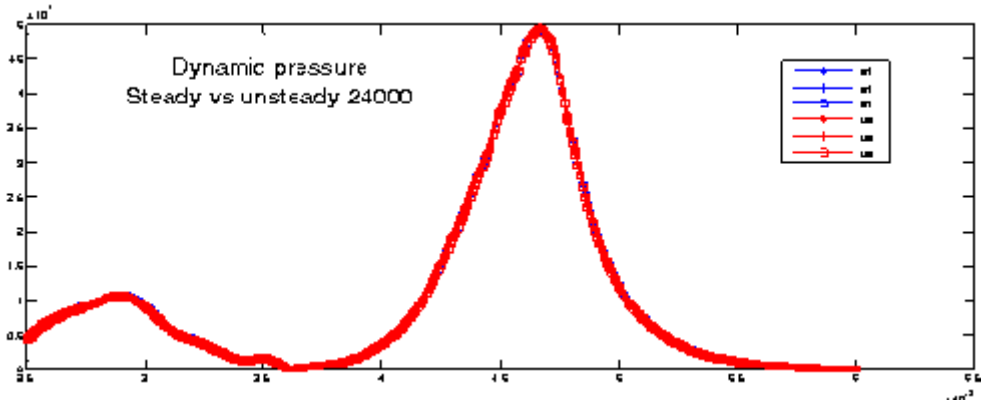

Fig. 4f. Comparision of transverse Dynamic Pressure under steady vs unsteady flow for 24000pa

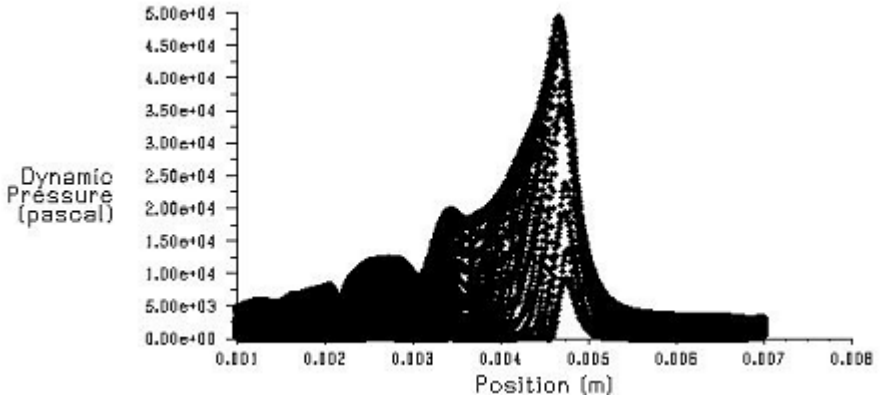

Fig. 4f.a. Dynamic Pressure along y direction

Convergence map

The solution has been converges at different time steps. Equation of continuity, x and y momentum equations are converging correct to three decimal places and the results are shown in figure 7.

Wall Shear and Strain

The contours of Wall shear stress and strain shown in Fig. 8a and 8b. The maximum magnitude of wall shear stress occurs in the region of stenosis and increases with increase in ä. This weaken the wall and increase the possibility of the rupture. High shear and strain occurs near the wall of the constricted regions.

Fig 8c. shows WSS is high for 24,000pa for both steady and unsteady flow and much correlated with data for 12,000pa. Fig. 8d. shows the comparision of strain for 12,000 and 24,000pa.

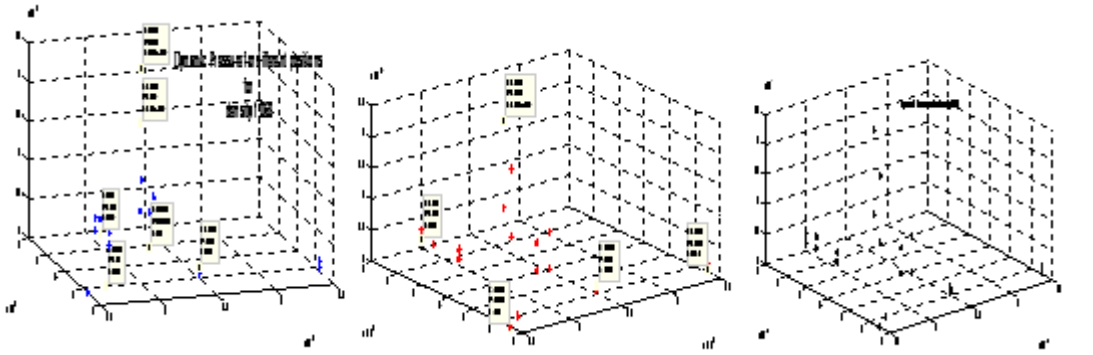

Fig. 4g. Dynamic Pressure at different position for densities 1060, 1030 and 1000

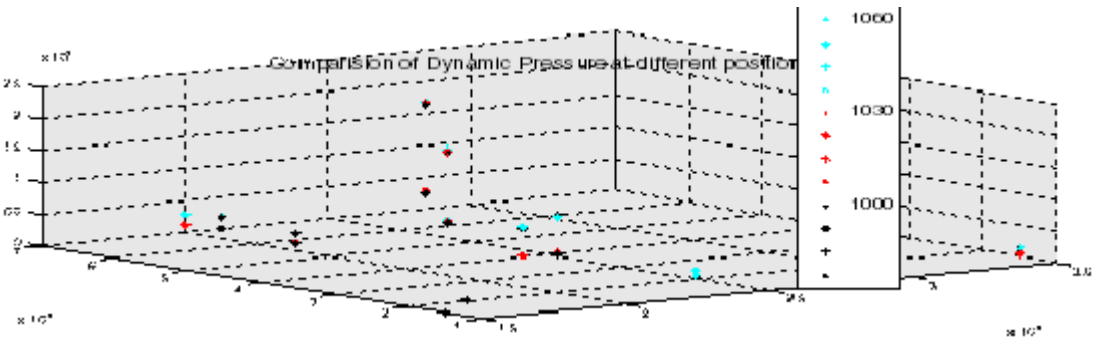

Fig. 4h. Comparision of Dynamic Pressure for different densities.

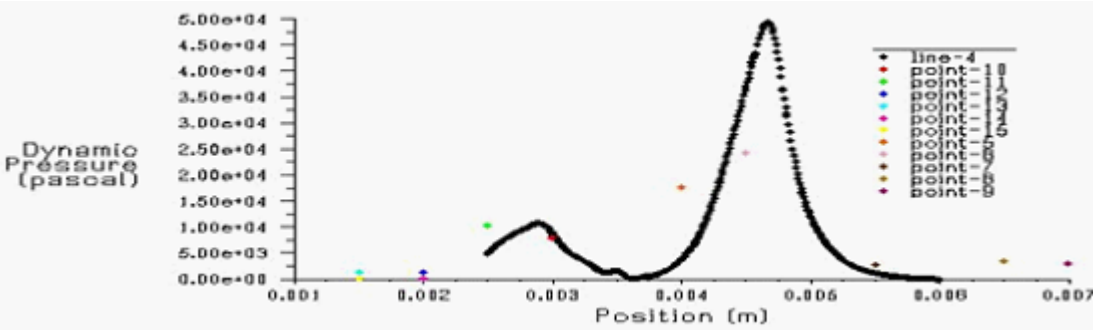

Fig. 4i. Dynamic Pressure at different positions and black line shows along transverse velocity along y direction

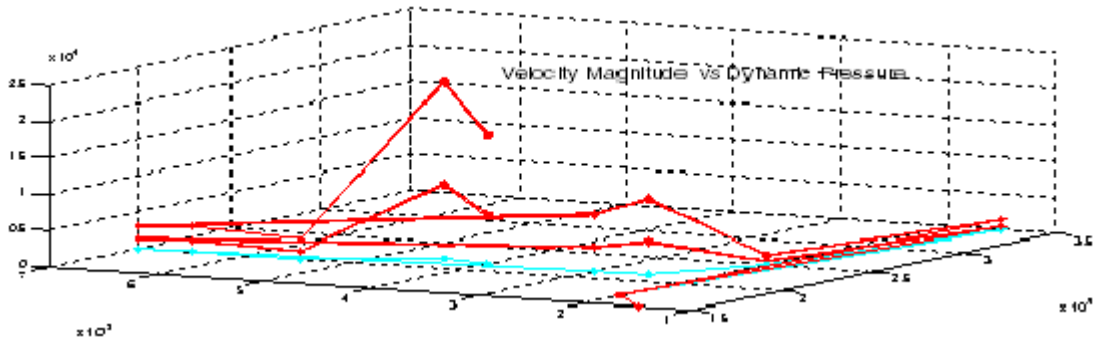

Fig. 5. Comparision of Velocity Magnitude (Cyan) Vs Dynamic Pressure (Red)

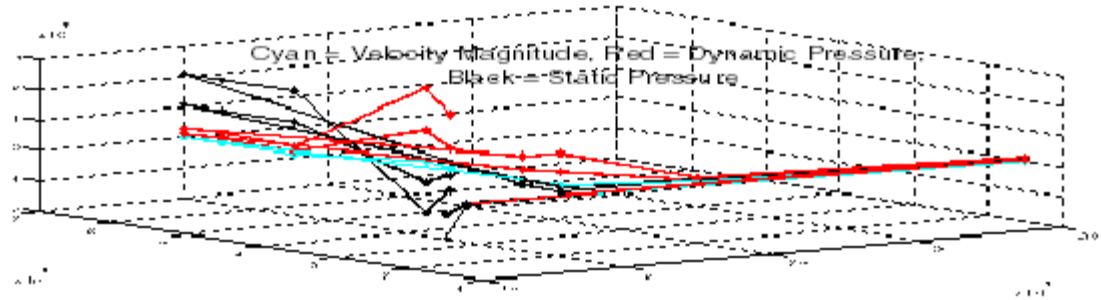

Fig. 6. Comparision of Velocity Magnitude, Dynamic and Static Pressures

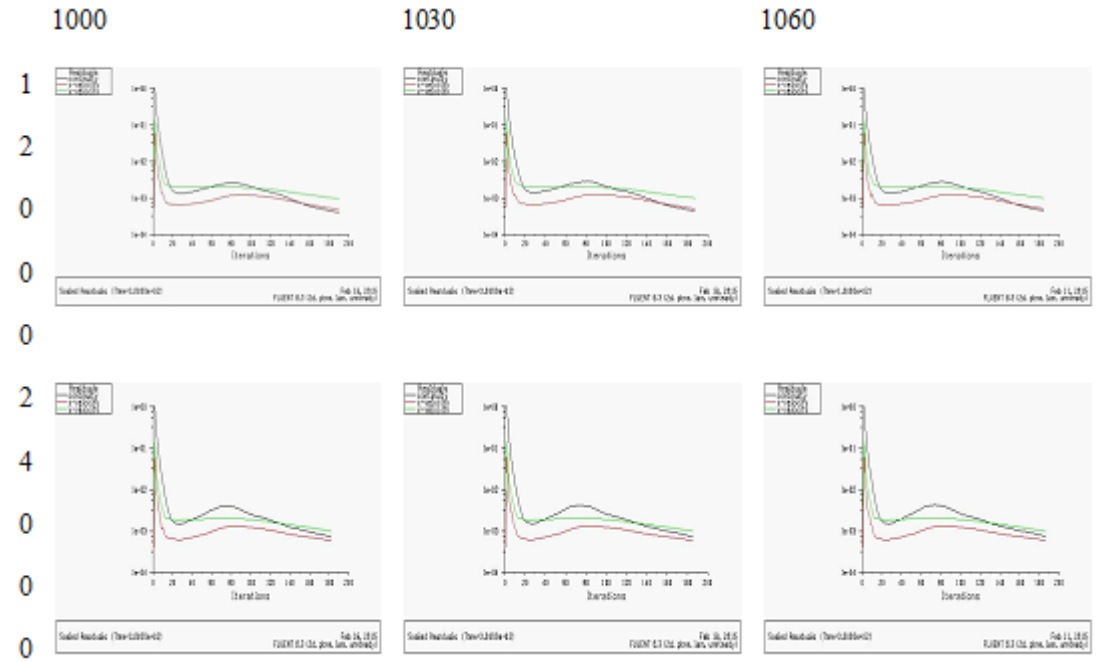

Fig. 7. Convergence of an unsteady flow.

Table 7. Maximum and minimum values of dynamic pressures within the domain.

Dynamic pressure	12000 Min.Steady	12000 Max.Steady	12000 Min. Unsteady	12000 Max. Unsteady	24000 Min. Steady	24000 Max. Steady	24000 Min. Unsteady	24000 Max. Unsteady
1000density	0.0003777761	22434.61	0.0007941552	21928.77	0.0003043454	49269.22	3.240126e-05	47826.19
1030 density	0.0005232768	22468.4	0.001223635	22681.41	0.0001173134	49547.92	0.0002779273	48708.7
1060 density	0.001223584	22665.92	0.001106621	22638.78	0.0005948145	49620.89	0.0006821011	49790.63

Strain profile shows that high pressures have more fluctuations than minimum pressures.

From Fig. 8f. the WSS is very high at y = 0.005 that is the line passing through the stenosis.

Fig. 8g. shows the strain profile along the transverse line within the domain shown in black dots and strain deviations at 11 interior points. Fig 8h. shows the Strain along y direction.

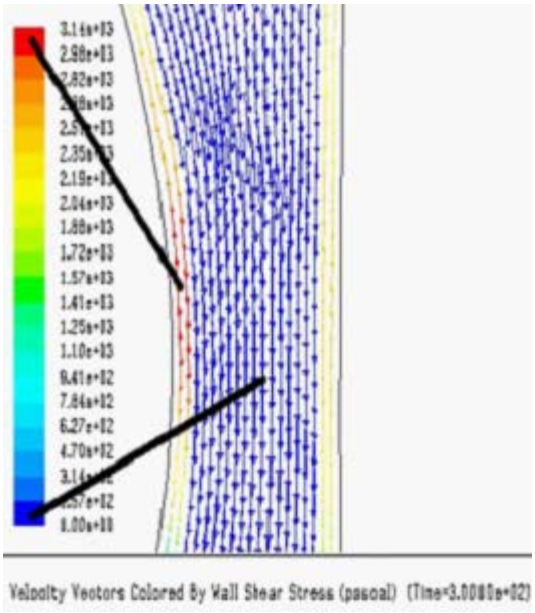

Fig. 8a. Streamlines of Wall shear stress

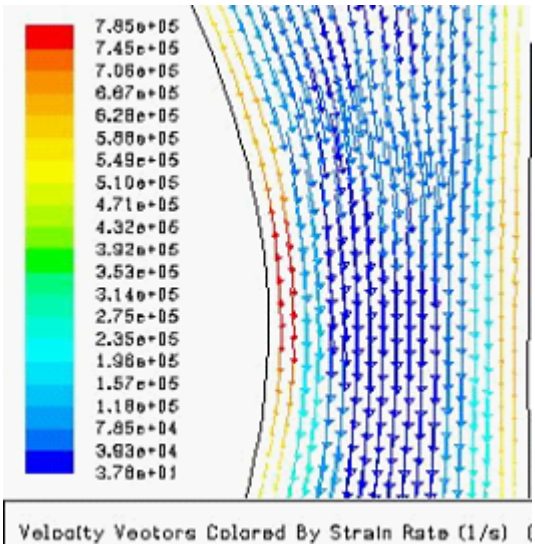

Fig. 8b. Streamlines of Strain

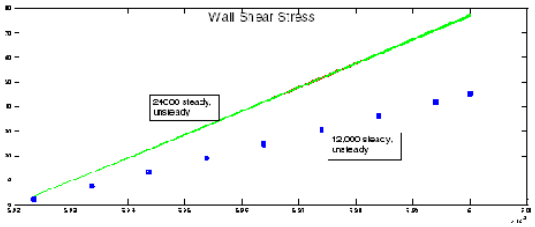

Fig. 8c. Comparision of Wss under steady and unsteady condition

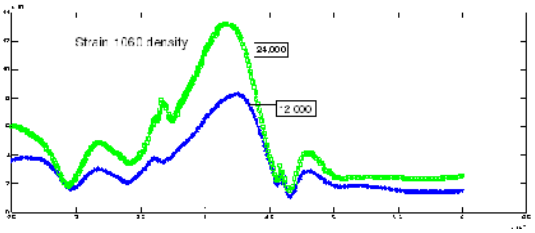

Fig. 8d. Comparision of Strain under various pressures.

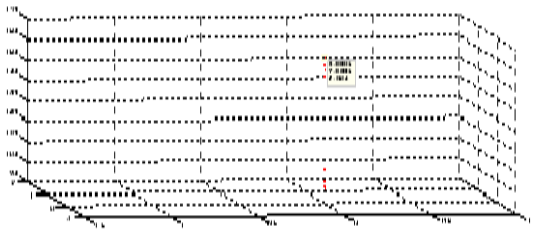

Fig. 8e. WSS at different positions

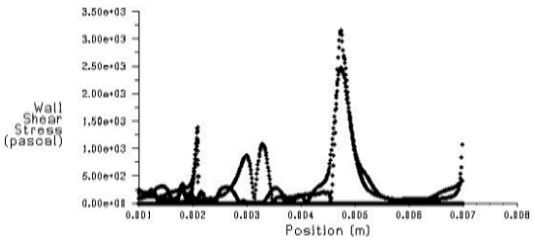

Fig. 8f. WSS along y direction

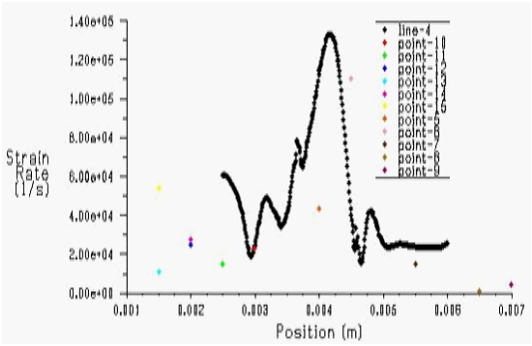

Fig.8g. Strain under different positions

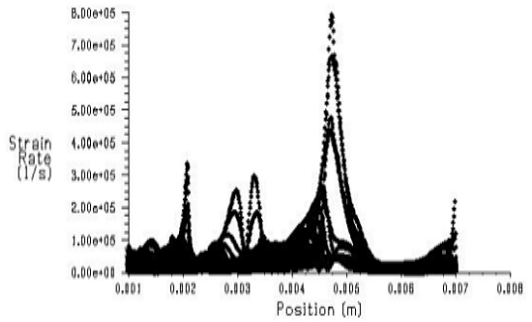

Fig.8h. Strain along y direction

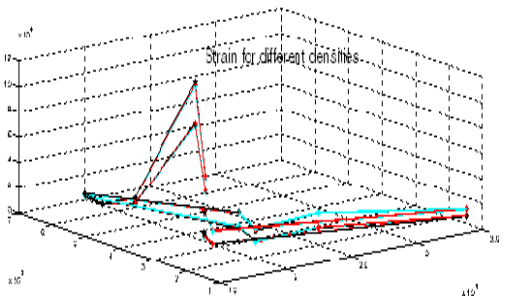

Fig. 8i. Comparision of strain for different densities1000 = cyan, 1030 = red, 1060 = black

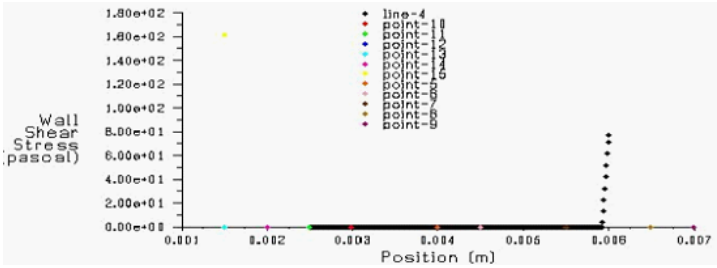

Fig. 8j. WSS at different point inside the domain

CONCLUSION

Flow models are highly dependent on the geometry, pressures, flow densities and different constriction of the vessels and is not similar for all stenoses. Antiplatelet therapy is one of the medications to prevent blood clot. This model analyzed in detailed about pressures and densities. Due to acute stenosis at y = 0.005, the velocity is very high in that region shown in Fig 3, and from fig. 3a to fig. 3j graphs clearly predict that the velocity is gradually increase from the inlet upto the constricted region y = 0.005 and at that point it attaines maximum velocity thereafter it gradually decreases. Pressure reaches high due to acute stenosis and it varies between 21928.77pa to 49790.63pa. Wall Shear Stress attains high at the point y = 0.005 and at remaining points it shows 0 due to viscous flow. Due to antiplatelet drug injected in very low dose we can't able to attain much variation graphically but there is slight changes in values shown in table 6 and 7. Most doctors recommend aspirin in healthy subjects who have one or more risk factors for developing atherosclerosis. Other measures are necessary to prevent atherosclerosis like losing excess weight, controlling HBP, diabetes, lowering LDL cholesterol, increasing HDL cholesterol and stopping cigarette smoking. In the beginning stage we can take the salicylic acid in natural form as it is found in certain vegetables, fruits, nuts, seeds and herbs in various amounts.

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