



ISSN: 2319-5967

ISO 9001:2008 Certified

International Journal of Engineering Science and Innovative Technology (IJESIT)

Volume 2, Issue 3, May 2013

# Computational Fluid Dynamics on Simulation of 2D Left Normal and Narrowing Coronary Artery on Bases of Hemodynamic Factors

Rachna David, A.Paul

PG Student, Mechanical Engineering Department, MNNIT, Allahabad

Assistant Professor, Mechanical Engineering Department, MNNIT, Allahabad

*Abstract— In the present paper, computational fluid dynamics (CFD) simulation on blood flow in human left coronary artery (LCA) predict the plaque in effected region. In arterial system, left coronary arteries are classified into two parts ie .LM (Left main coronary) into LCX (Left circumflex artery) and LAD (Left anterior descending).The arterial wall is affected by atherosclerotic plaques. To purpose of this work is to simulate and analyzed the blood flow in left coronary artery in normal and effected artery to predict the possibility of further atherogenesis. The initial velocity of 15 cm/sec is taken into consideration at inlet. The GAMBIT is used for geometry creation and FLUENT software was used for post processing analysis. The CFD prediction of blood flow physics in coronary artery is useful for diagnosis, prognosis and prevention from coronary diseases. Average Wall Shear Stress (AWSS) is found less, vorticity is found high in abnormal effected artery compared to the normal artery. The trend of vorticity is found inverse to that of AWSS. Normal left main bifurcation is more resistance to atherogenesis compared to atherosclerotic LM bifurcation.*

*Index Terms— Atherogenesis, CFD, LM Bifurcation, AWSS.*

## I.

## INTRODUCTION

Arteries are the blood vessels that carry blood away from the heart. This blood is normally originated exception made from the pulmonary and umbilical arteries. The left coronary artery abbreviated LCA and is known as left coronary artery, is an artery that arises from the aorta above the left cusp of the aortic valve and feeds blood to the left side of the heart [1]. It is the circulation of blood in the blood vessels of the heart muscle. The vessels that deliver blood to the myocardium are known as coronary arteries. The vessels that removes the deoxygenated blood from the heart muscle are known as cardiac veins. The coronary artery that run on the surface of the heart are called epicardial coronary arteries. [2] The coronary artery consist of two main artery, the right and the left arteries. The left coronary artery system branches into the circumflex artery and the left anterior descending artery. Coronary artery deliver blood to the heart muscle, any coronary artery disorder or disease can have serious implication by reducing the flow of oxygen and nutrients to the heart muscle, which may lead to heart attack and possible death. Direct studies of blood flow and velocity profiles in normal human subjects are due to the inadequate non invasive means of measurements. [3] Atherosclerosis (a build up of plaque in the inner lining of an artery causing it to narrow or become blocked) is the most common cause of heart disease. [4] Knowledge of local flow dynamics (hemodynamic) is important for understanding the development and progression of vascular disease such as atherosclerosis and intimal hyperplasia. [5] **Coronary artery disease**-The coronary arteries can become narrowed by "furring" of the artery wall with cholesterol called atherosclerotic plaques. Blood clots can form on these plaques and block the artery, causing chest pain. The heart muscle supplied by the blocked artery starved with oxygen and dies soon afterwards known as heart attack [6].The WSS distribution of the main trunk and the bifurcation of the left main coronary artery of the model in one cardiac cycle are presented, and the result demonstrate that the low and oscillating WSS is correlative with the clinical observation and atherosclerotic prone sites in the left coronary artery.[5] Atherosclerosis, the leading cause of death in the developed world, is associated with systematic risk of factors including hypertension, smoking, diabetes mellitus, among others. Hemodynamic factors such as low wall shear stress (WSS), oscillating WSS, flow division particle residence time stasis facilitate. Hardening of the arteries, also called atherosclerosis, is a common disorder. It occurs when fat, cholesterol and other substance build up in the walls of the arteries and hard structures called plaques. [7].

II.

ARTERY MODEL

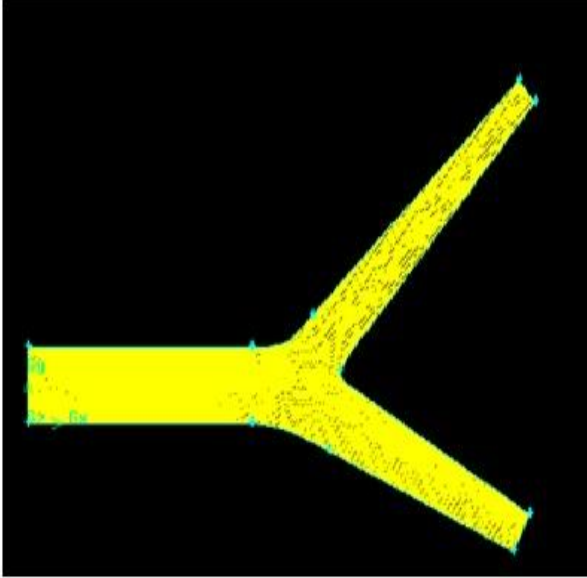
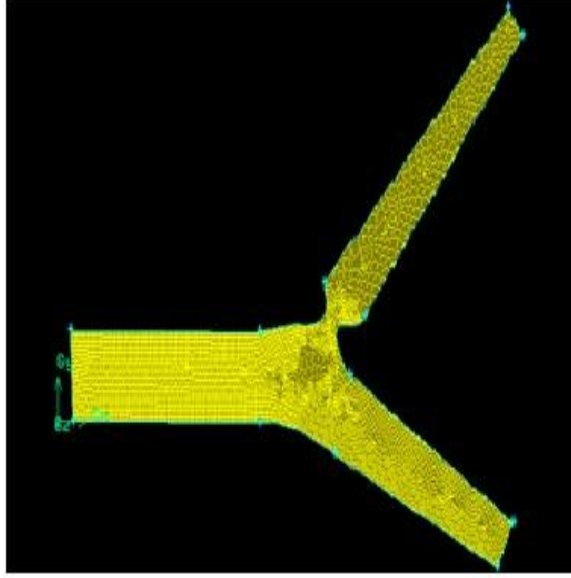
Normal Artery	Abnormal Artery
 <p data-bbox="204 1025 550 1070">Fig 1 Normal LCA Bifurcation</p>	 <p data-bbox="847 1025 1410 1070">Fig 2 Shows Lesion at the ostium of Sub Branch</p>

Fig 1 and Fig 2 Shows The left coronary artery model has been generated in Gambit Software .All dimensions are taken from Binu &Kumar (2012). Cheelampati at al perform studies on normal human coronary dimensions in Indians (8).The length of the LM, LAD, LCX are taken as 1.5cm, 1.5cm and 2cm resp. The diameters are 4mm, 3.6mm and 3mm resp. The angle between LM and LCX are taken as 151 and 132 degree. [3]

III.

COMPUTATIONAL GRID

All lumen geometric data was transferred into a specialized pre-processing program for grid generation (Fluent Inc. Gambit, Lebanon, NH, USA).In total 13372 grid nodes in normal Bifurcation and in abnormal Bifurcation 13161 and element type of Trigonal in both the case. The grid spacing is taken as 0.01.The used mesh is based on the computational results of mesh independence studies.

IV.

GOVERNING EQUATIONS

In this analysis the blood is taken as Newtonian fluid but blood is generally a Non- Newtonian fluid, actually we assume that modeling diameter is larger than 100 micrometer. For this assumption blood is taken as incompressible and it is governed by Navier Stokes equations.

Mass equation:

$$\frac{\partial u_j}{\partial x_j} = 0 \tag{1}$$

Momentum equation

$$\frac{\partial(u_i u_j)}{\partial x_j} = -\frac{1}{\rho} \frac{\partial p}{\partial x_i} + \frac{\mu}{\rho} \frac{\partial^2 u_i}{\partial x_j \partial x_j} \tag{2}$$



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$\mu$  = viscosity coefficient

$u_j$  ( $i, j= 1, 2, 3$ ) is the velocity component in x, y and z direction.

$p$  = pressure

$\rho$  = density of fluid.

#### IV BOUNDARY CONDITIONS

The velocity was assumed to be uniform at the orifice of LMCA. The applied inflow coronary conditions under flow velocity of 0.15 m/sec. A flow division of 59 % and 41% were taken through LAD and LCX at the bifurcation. Operating Pressure is assumed to be 13330 Pa .Dynamic Viscosity is taken as 0.00345 kg/m s. All computational grid data as well as all physical flow data determined from the boundary conditions, were imported into the main CFD solver (Fluent Inc.USA) The numerical code solves the governing momentum equations. In general, these equations solve for mass, momentum conservation. Flow is considered as 2D, steady, Laminar, with no external forces applied on it, while the arterial wall is comprised from non-elastic and impermeable material. [8]

#### VI NUMERICAL SIMULATION

Fluent 6.3 has been applied for the numerical simulation. The applied numerical code utilizes a segregated solver of implicit formulation. Pressure –velocity coupling is based on the SIMPLE scheme. Second order upwind numerical scheme is applied for the mass and momentum equation.

#### VII. COMPUTATIONAL VALIDATION

The computational validation was done with the computational result by Binu &Kumar (2012).Validation was performed WSS and position of the artery. Graph shows the wall shear distribution in different walls of the artery. Different colors represent different walls of the artery. Wall 4 of the artery shows maximum shear stress at bifurcation location.

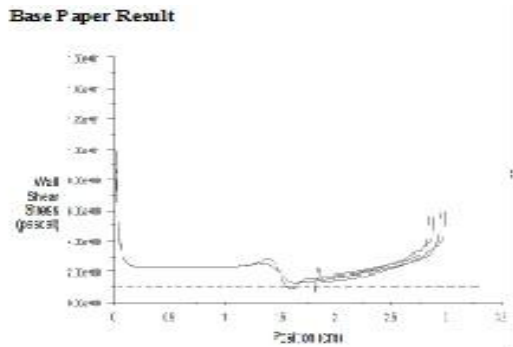


Fig 3

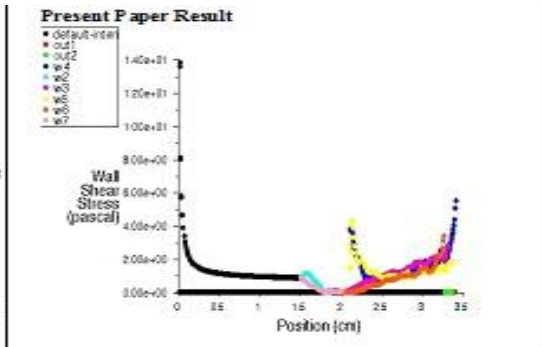


Fig 4

Fig 3 and Fig 4 shows the Normal Bifurcation in the Artery

Fig 3 and Fig 4 shows the Wall Shear distribution validation in Normal Bifurcated Artery. The graph shows that the development of plaque at the outer wall of the bifurcations, The region where the wall shear stress is below the 1 Pa. The max WSS is found to be 14 Pa. Below the 1Pa which the plaque formation can occur.

Table 1 shows the % error found in Wall Shear Stress (WSS) variation in Normal coronary Artery.

Validation(Max WSS) NORMAL CORONARY ARTERY	(WSS)Pa
Present value	14
Paper Value	15
Error (%)	6.66

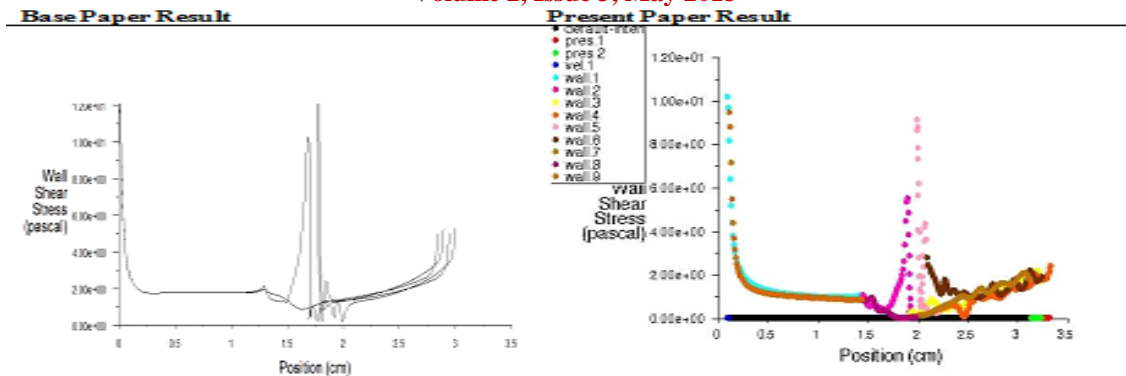


FIG 5

FIG 6

FIG 5 and FIG 6 shows the Abnormal Bifurcation in Artery

Table 2

Validation (Max WSS) ABNORMAL CORONARY ARTERY	(WSS) Pa
Present Value	10.3
Paper Value	11.8
Error	12.7

Table 2 shows the maximum wall shear stress is found to be 10.3 Pa .AWSS is the average wall shear stress of the regions in the artery where the WSS falls below 1 Pa. In fig 5 &fig 6 show WSS plots for Abnormal Artery. Oscillating WSS is observed all the cases and it is below 1 Pa. near the bifurcation. The area of the region where the WSS goes below 1 Pa. Varies between cases as it is observed in the plots.

### V. RESULTS AND DISCUSSION

The model of the left coronary artery bifurcation normal and abnormal case is validated and comparing the results in simulation studies.[3].Fig 7 and Fig 8 gives the strain rate distribution in a normal coronary bifurcation and in abnormal bifurcation case. Graphs observed the development of the plaque at the outer walls of the bifurcation .The plot of pressure distribution along the length of the artery shown in fig 9 and 10. This graph shows the same information as WSS graph gives. Area Averaged strain rate is found to be 446 1/s in normal bifurcation and 312 1/s in abnormal bifurcation artery case.

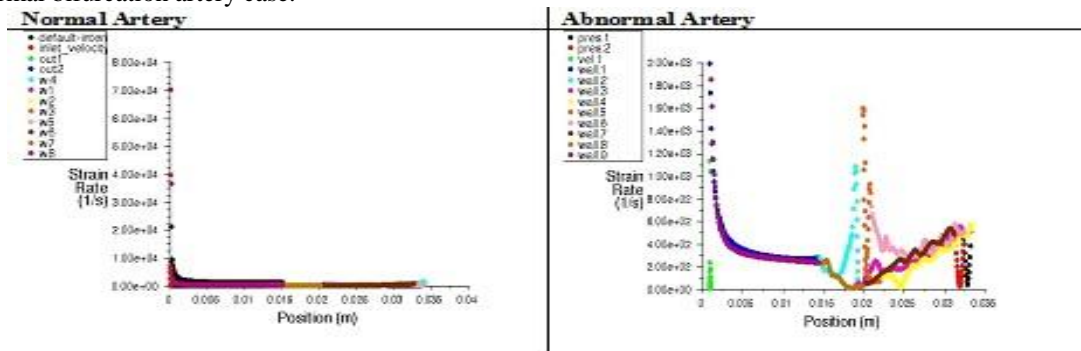


FIG 7

FIG 8

FIG 7 and FIG 8 Shows strain rate varies with position in Normal and Abnormal Coronary Artery

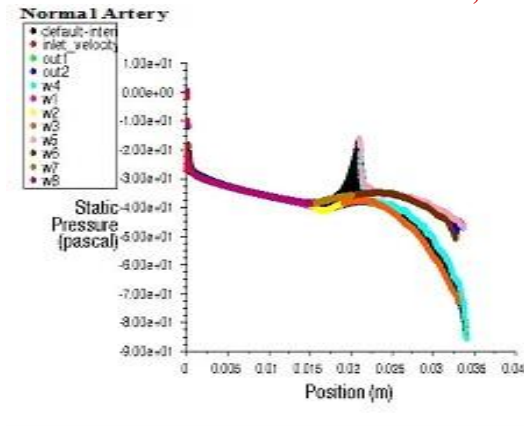


FIG 9

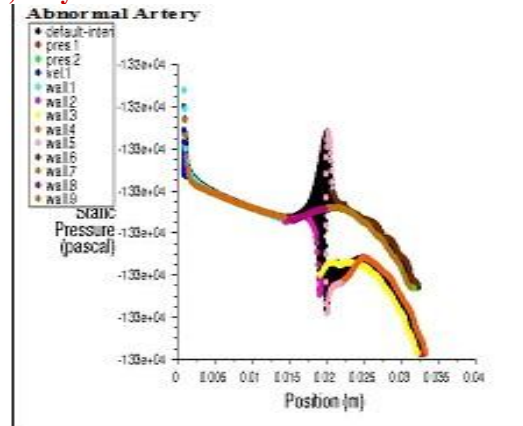


FIG 10

FIG 9 and FIG 10 Shows the static pressure graph with respect to position in Normal and Abnormal Artery.

Fig 11 and Fig 12 Velocity is maximum at the atherosclerotic plaque region where the blood flow area is minimum. when the flow diameter of the lcx is decreased(due to deposition of fats) velocity is increase at this position. Max velocity is found in abnormal artery is 24.3 m/s.

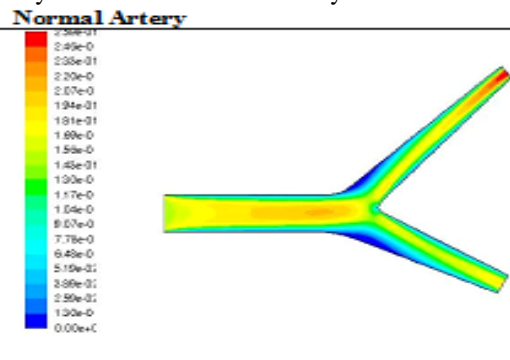


FIG 11

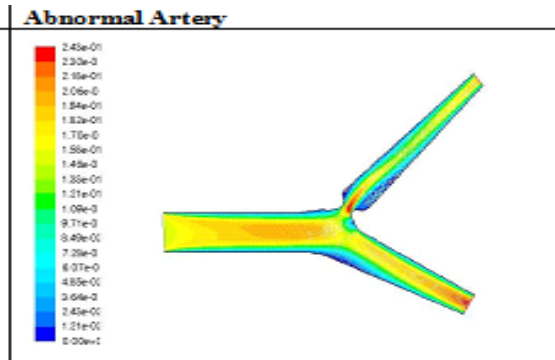


FIG 12

FIG 11 and FIG 12 shows the velocity contours 2D Normal and Abnormal Artery.

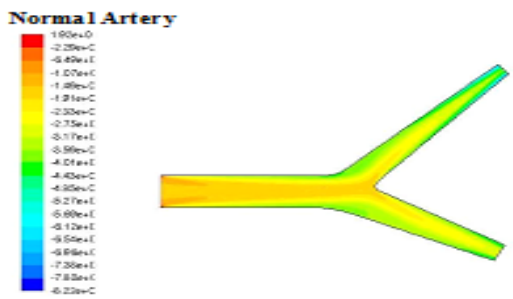


FIG 13

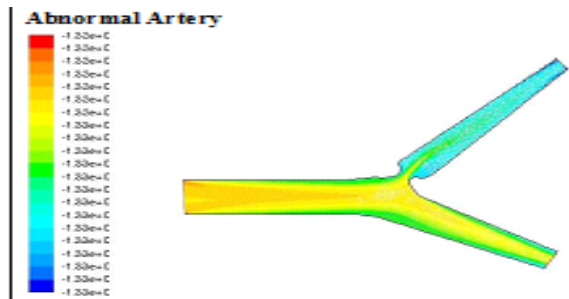


FIG 14

FIG 13 and FIG 14 Shows the pressure contours of Normal and Abnormal Artery.

Fig 13 and Fig 14 Shows the pressure distribution in normal and abnormal artery .High pressure found in middle part of bifurcated normal artery because of velocity in this portion is found to be less. Abnormal narrowing artery observed lesser pressure than normal artery. Fig 15 and Fig 16 gives information about the variation in velocities with respect to position of the normal and abnormal coronary artery. At the bifurcated part in abnormal artery flow area is decreased hence in this portion velocity is found to be increased compare to normal bifurcated artery.

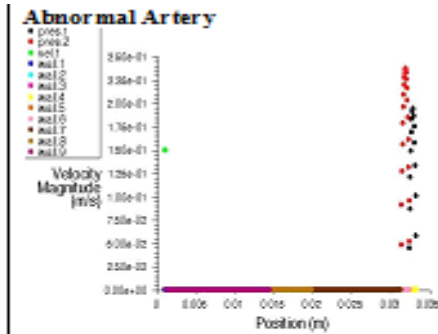
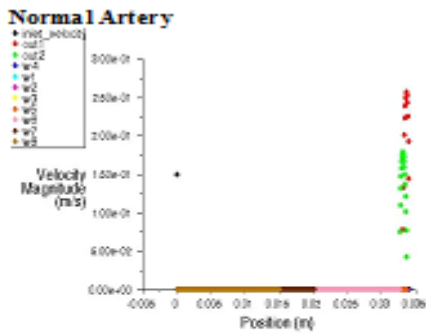


FIG 15

FIG 16

FIG 15 and FIG 16 Shows the velocity magnitude with respect to position of the Artery of Normal and Abnormal Artery

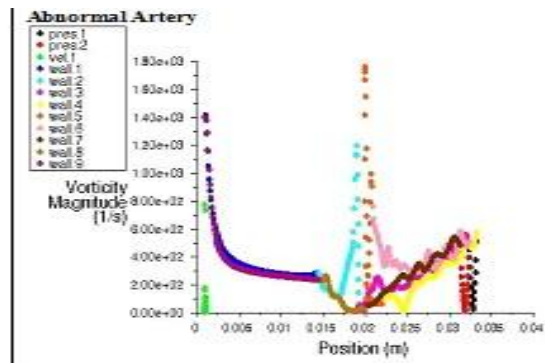
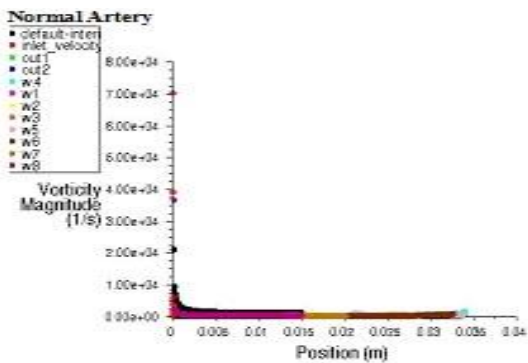


FIG 17

FIG 18

FIG 17 and FIG 18 Shows Vorticity Magnitude with respect to position for Normal and Narrowing Artery.

Vorticity is found higher in abnormal artery compare to normal artery. In normal artery magnitude of vorticity is found in wall 1 and in plaque artery higher vorticity magnitude is found in wall 5.

Table 3

Types of Artery	AAWSS (Pa)	AASR (1/sec)	AWSS (Pa)	AAV (1/sec)
Normal Artery	1.25	446	0.7	121
Abnormal Artery	1.1	312	0.4	132

Table 3 shows that Area Averaged wall shear stress(AAWSS),Area Averaged strain rate(AASR), Averaged wall shear stress(AWSS) is higher in normal artery bifurcation but Area Averaged vorticity(AAV ) is lower in normal compare to abnormal artery.



ISSN: 2319-5967

ISO 9001:2008 Certified

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## VI. CONCLUSION

The AWSS plots shows oscillating response and low values (less than 1 Pa) near bifurcation the amount of oscillation varies between different normal and abnormal cases. In Abnormal artery case are found less WSS compare to normal artery case. The wall of the artery found minimum velocity compare to plaque artery. At the plaque region higher velocity are found because of the reducing the blood flow area. At the bifurcation WSS is minimum less than 1 pa and static pressure is max at the middle of LM at the wall surface it become minimum and vorticity is found max in plaque region, hence become greater possibility of atherosclerosis plaque in the artery.

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## AUTHOR BIOGRAPHY

**Rachna David**, PG student at MNNIT, Allahabad, She had completed Bachelor degree in year 2009 in mechanical engg department from U.P. Technical University, Lucknow and presently pursuing Master degree with specialization in Applied Mechanics Department. Her working area is blood flow analysis in human heart by using CFD. **Email-rachnadavid2k7@gmail.com**

**A. Paul**, an Assistant professor of Mechanical Engineering Department at MNNIT, Allahabad. His area of research Fluid Dynamics, CFD, Turbulence, Bio-Fluids, Human Respiratory System. The author having more than 15 years of teaching, and research experience.