Simulation of Left Main Coronary Bifurcation Lesions using 3D Computational Fluid Dynamics Model and its Comparison with 2D

Binu L.S., A. Sukesh Kumar

Abstract— **Coronary artery disease (CAD) is associated with plaque formation in coronary arteries. Left main coronary bifurcation is one of the most affected regions in the human coronary system. Atherosclerotic plaque formation occurs at the sites of branching and bifurcations where low wall shear stress (WSS), oscillating WSS, flow division, particle residence time and stasis are found to occur. Study of blood flow through left main bifurcation in human subjects is difficult due to the lack of adequate techniques The purpose of this work is to simulate and analyse blood flow through left main coronary bifurcation under different stenosed conditions and to predict the possibility of further atherogenesis. Blood flow through left main coronary bifurcation is simulated and analysed using 3D computational fluid dynamics (CFD) model and the results are compared with that of 2D. The seven cases of bifurcation lesions described in literature are modeled and simulated. GAMBIT® and FLUENT® softwares were used for geometry creation and analysis respectively. Most of the 3D results are in agreement with that of 2D. Area weighted Average Wall Shear Stress (AAWSS) and vorticity are found minimum for normal bifurcation and the graphs follow similar but well defined trends with an offset compared to 2D. Average WSS (AWSS) for the regions with less than 1 Pa. is maximum for normal bifurcation and minimum for the lesion type 1.0.0 which infer that normal left main bifurcation is more resistant to atherogenesis compared to atherosclerotic LM bifurcations. The next inference is that among the types of lesions, the type 1.0.0 is severe in terms of greatest risk of plaque proliferation. The observation that the presence of lesions in either distal main branch or side branch or both together along with LM lesion (Type 1.1.0, Type1.1.1 and Type 1.0.1) increases AWSS and hence resist further atherogenesis better, is in confirmation with 2D results. Area Weighted Average Vorticity is established as a feature for lesion type identifcation.**

***Index Terms*— Atherosclerosis, LM bifurcation, Simulation, CFD**

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I. Introduction

Direct studies of blood flow and velocity profiles in normal human subjects are difficult due to the inadequate noninvasive means of measurement. To simulate the blood flow in human vascular system, three different scales of models can be devised. For a precise simulation of a particular region of cardio vascular system, one should use a model that take in to account the fluid structure interaction by using CFD tools such as ANSYS CFX®. An intermediate level of accuracy is provided by a 2D model simulated using packages like FLUENT®. A better level of accuracy can be achieved by a 3D model. Joy P. Ku et al have described the use of computational fluid dynamics in a bypass graft model and compared the blood flow patterns from CFD and MRI [4]. Validation of these simulation results with experimental MRI results demonstrated favorable agreement in blood flow patterns. Dr. Leonid et al experimentally validated the numerical simulations performed by commercial flow solver FLUENT ® [5].

The major flow characteristics directly related to the localization of atherosclerotic plaques are low fluid velocity and resultant low wall shear stress (WSS) [6]. Elevated levels (>1 Pa) of WSS will shield against atherosclerosis. Oscillating WSS, Flow separation, blood flow recirculation and stasis are also common features in the region of atherogenesis/intimal thickening [7, 8, 9, 10, 11]. Byoung et al suggested that a difference in phase between pressure conductance and blood flow can also be a factor of atherogenesis. Their study revealed that the local rheologic influence of the phase difference between the wall pulsation and blood flow on the mean wall shear rate (WSR) and WSS of coronary artery was more than 10 times higher than that of the abdominal aorta. Madanlal et al suggested that plaque formation tends to start from very low flow Reynolds number of 25 with 50% restriction [12]. The entrance regions and segments of the main bifurcation of the left main bifurcation of the left coronary artery are favorable places for atherosclerosis. Studies on WSS distribution with branching angle and branch radii variation are performed by Byong et al [13] and Vahab et al [14, 15]. Richard et al conducted experiments to determine the relation between stenosis and perfusion rate in man [16]. In this study the relation between all anatomic dimensions of coronary artery stenoses and myocardial perfusion reserve (RMPR) in man are established. Coronary flow velocity reserve (CFR) has proven to be an important diagnostic tool that provides

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relevant physiological information regarding the function of LAD.

As per modern vascular biology the endothelial lining of artery is locally responsive to various stimuli originating from the circulating blood and/or neighboring cells and tissues which can lead to atherogenesis. In the present work the 3D geometry of the seven types of LM bifurcation lesions classified by the Medina binary system are created using GAMBIT® software. The analysis is carried out using FLUENT for wall shear stress and vorticity.

II. Methods

A. *Bifurcation Geometry*

Left main coronary artery stenosis is associated with high mortality both in patients receiving treatment and those on drug therapy. It justifies the selection of LM bifurcation for the analysis. Cheemalapati et al conducted a survey on normal coronary artery dimensions in Indians [17]. Wing-Hung et al performed studies on normal human coronary dimensions in post mortem hearts [18]. Gerhard et al experimentally determined layer specific mechanical properties of human coronary arteries [19]. They succeeded in separating the individual layers viz., intima, media and adventitia of human coronary arteries. Dlear et al compared the size of coronary artery in UK south Asian and Caucasian men [20]. Airlee et al suggeated the difficulty of determining left main coronary stenosis with reasonable accuracy [21].

The left coronary artery normally originates left posteriorly from coronary sinus of Valsalva or at the junction of the sinus and tubular portions of aorta (sinotubular junctions) [22,23]. This location allows maximal coronary filling during ventricular diastole. The length of LM, LAD and LCX are taken as 1.5 cm, 1.5cm and 2cm respectively. The diameters are taken as 4mm, 3.6mm and 3mm respectively. LM does not vary in diameter significantly but a tapering of 0.1mm/mm and 0.08mm/mm are observed for LAD and LCX. The angle between LM and LAD is taken as 151^o and the between LM and LCX is taken as 132^o [24]. The exact anatomical model is approximated to a model shown in fig. 1. The curvature of the LCA system due to the curvature of heart surface is taken as 15^o in 3D model and it is neglected in 2D. The walls are assumed smooth, rigid and regular. The coronary wall is made up of a three layer structure with inner intima, then media and outer adventitia with different Young's moduli. But it is assumed as a rigid motionless wall for the lesion study.

According to Medina bifurcation lesion classification system bifurcation lesions with significant narrowing were categorized in to seven types. The Medina classification is a simple binary system whereby significant lumen narrowing is classified as present (1) or absent (0) in the Proximal main branch (MB), Distal MB and Side branch. A similar classification is SYNTAX Score which is a combination of Duke and ICPS systems [25]. Type 1.1.0 means lesions are present in proximal MB, distal MB and is absent in the side branch.

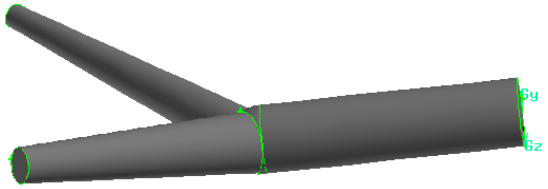

Fig. 1 Normal LCA Bifurcation

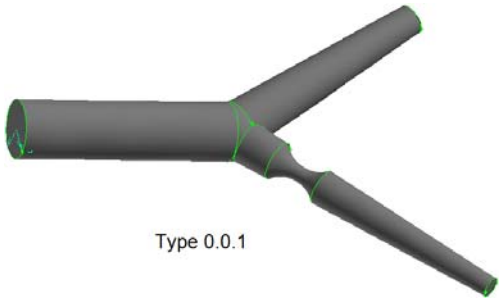

Fig. 2 Lesion at the ostium of SB

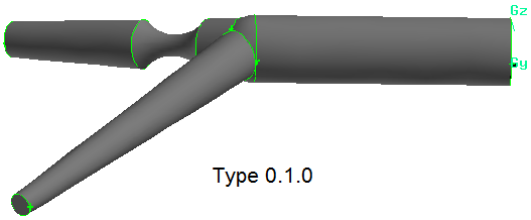

Fig.3 Lesion at the distal MB

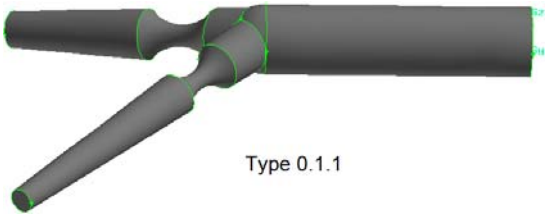

Fig. 4 Lesion at post branching and ostium of the SB

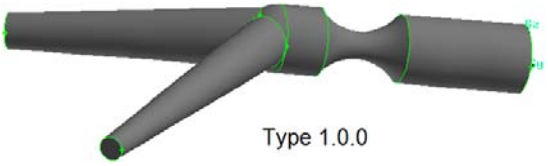

Fig. 5 Lesion at pre-branching

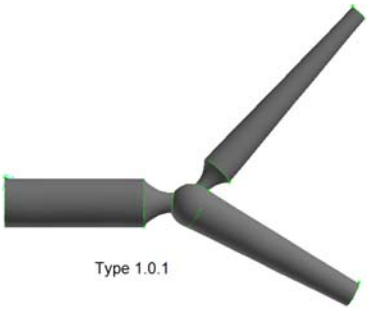

Fig. 6 Lesion at prebranching and ostium of SB

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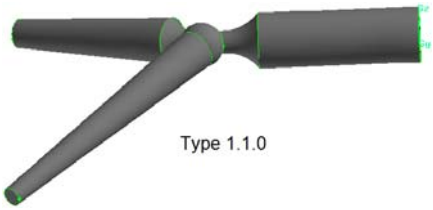

Fig. 7 Lesion in parent vessel only

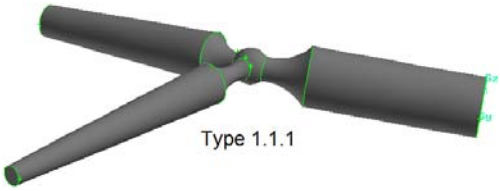

Fig. 8 Lesion at the Bifurcation

Figures from 2 to 8 illustrates the 3D geometries of different possible cases of lesions as per the Medina system. The geometries are created using GAMBIT®. The percentage stenosis introduced in all geometries is 60%.

B. Governinig Equations

Blood is generally a Non-Newtonian fluid, but it is reasonable to regard it as a Newtonian fluid when modeling arteries with a diameter larger than 100μm. The non-Newtonian effect is predominant only in very small blood vessels. So blood is assumed as an incompressible flow with Newtonian behavior governed by the Navier-Stokes equations (1) and (2), where u is the velocity vector, ρ the density, P the pressure, μ the viscosity and t the time. These equations are solved using Computational Fluid Dynamics software FLUENT®. The density of blood, viscosity and the mean inlet velocity are taken as 1050 Kg/m^2, 0.0035 Kg/ms 15 cm/s respectively.

$$\nabla u = 0 \qquad (1)$$

Equation(1) is the continuity equation (mass balance) and the momentum balance equation is

$$\rho \frac{Du}{Dt} = -\nabla P + \mu \nabla^2 u \qquad (2)$$

A grid sensitivity study was carried out for area weighted average WSS and the optimum grid spacing is found as 0.02 with 268779 cells, 551600 faces and 52044 nodes.

C. Boundary Conditions

No slip condition was applied to the walls. A flow division of 59% and 41% were taken through LAD and LCX at the bifurcation. The mean inlet velocity is taken as 15 cm/s. Operating pressure is set as 13.33KPa., the mean blood pressure in human circulatory system.

III. Results

The model of the normal left coronary bifurcation is validated by comparing the results with other experimental and simulation studies. The results are in agreement with the experimental results carried out on patients [16] as well as other simulation studies in literature [6,9,10]. After ensuring the reliability of normal LM bifurcation model, lesions were introduced in the geometry as per Medina binary lesion classification system and analysed using FLUENT®.

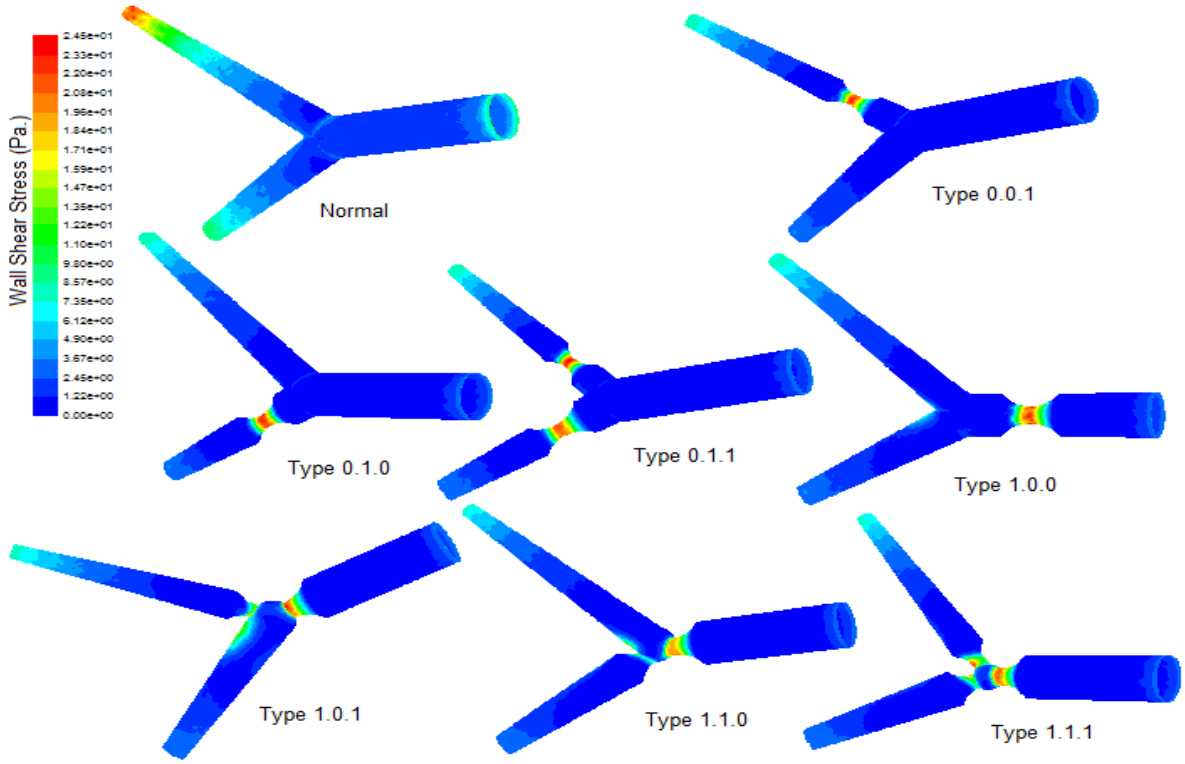

Fig. 9 Wall Shear Stress Distribution on LM bifurcation with different types of lesions

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Fig. 9 gives the wall shear stress distribution in normal left main coronary bifurcation and that with lesions. In the normal case WSS falls below 1 Pa for a very small region only. It occurs at the outer walls at the bifurcation shown by dark blue area. This justifies the development of plaque at the outer walls of the normal bifurcation. In all the affected bifurcations, the dark blue area dominates indicating a low wall shear stress. It shows that normal bifurcation is more resistant to the development of plaque formation than affected cases. The table summerises the Area weighted Average Wall Shear Stress (AAWSS) of LM bifurcation affected by different types of lesions.

Table 1 Area weighted average of wall shear stress

Medina type of Lesion	ID. No.	AAWSS(Pa.)	
		2D Simulation	3D Simulation
Normal	0	2.28	4.05
0.0.1	1	2.03	4.67
0.1.0	2	1.97	4.84
0.1.1	3	1.98	5.48
1.0.0	4	2.37	5.56
1.0.1	5	2.1	6.2
1.1.0	6	2.3	6.22
1.1.1	7	2.6	6.81

Table 2 Average Wall Shear Stress for the region where WSS < 1 Pa.

Medina Type of Lesion	ID. No.	AWSS(Pa.)		No. of nodes where WSS < 1Pa.
		2D	3D	
Normal	0	0.87	0.82	191
0.0.1	1	0.7	0.79	344
0.1.0	2	0.6	0.74	405
0.1.1	3	0.52	0.72	569
1.0.0	4	0.38	0.62	1205
1.0.1	5	0.6	0.71	514
1.1.0	6	0.57	0.67	751
1.1.1	7	0.5	0.67	783

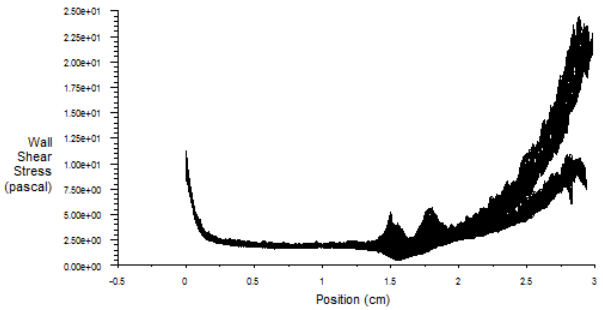

Fig 9a. Wall Shear Stress plot using 3D

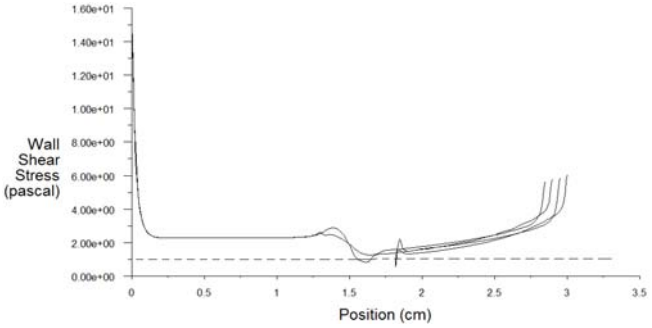

Fig. 9b. . Wall Shear Stress plot using 2D

AAWSS is minimum for normal bifurcation and it increases gradually from type 0.0.1 to type 1.1.1 as in table 1. Wall shear stress distribution along the length of the artery, fig.9a and 9b matches in both 2D and 3D. Fig.10 shows the trend plot of AAWSS for different types of lesions using 2D and 3D simulation geometries.

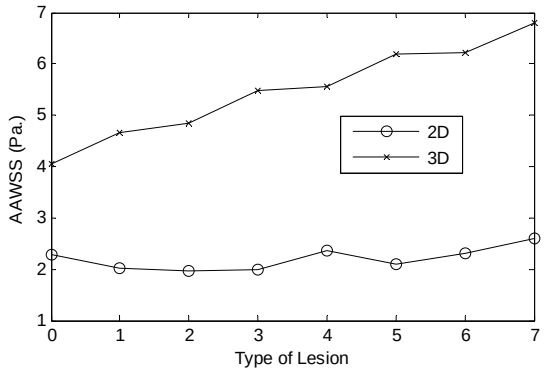

Fig. 10 Area weighted average wall shear stress

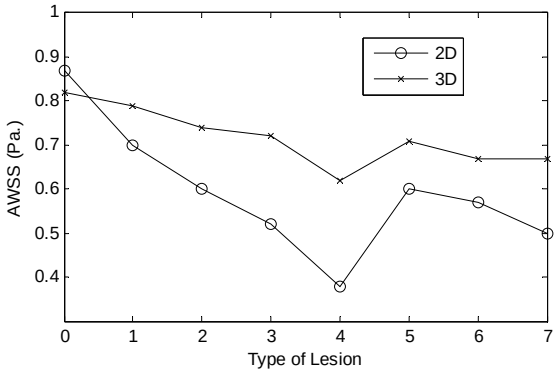

Fig. 11 Average wall shear stress in the region where WSS<1

Table 2 and fig. 11 show the average wall shear stress (AWSS) for the areas where WSS falls below 1 Pa. where there is a greater chance of plaque formation. This region is having the greatest extend in the case of type 1.0.0 lesion indicated by the highest number of nodes (1205 nos.).

AWSS is maximum, 0.82 Pa. (0.87 Pa. in 2D simulation) for normal bifurcation which indicates that normal LM bifurcation is more resistant to atherogenesis compared to bifurcations with lesions. Type 1.0.0 is having the minimum AWSS, 0.62 Pa. (0.38 Pa. in 2D) and hence a greater risk of plaque proliferation. For the remaining cases the AWSS ranges from 0.67 to 0.79 (0.5 Pa to 0.7 Pa in 2D). The possibility of further atherogenesis increases in the order Type 0.0.1 (0.79 Pa), Type 0.1.0 (0.74 Pa), Type 0.1.1 (0.72 Pa), Type 1.0.1 (0.71 Pa), Type 1.1.0 and Type 1.1.1

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(0.67 Pa). Similar trend is observed during 2D simulation also as can be seen from fig. 11.

Fig. 12 and table 3 show the variation of vorticity in normal LM bifurcation and that with different type of lesions. Vorticity is found minimum for normal bifurcation which confirms the statement that normal bifurcation is more resistant to atherogenesis.

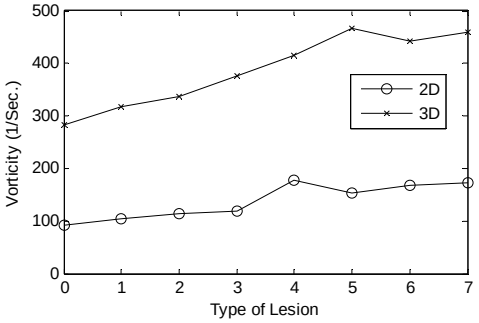

Fig. 12 Area weighted average Vorticity

Table 3: Area weighted average

Medina Type of Lesion	ID. No.	AAV (1/Sec.)	
		2D	3D
Normal	0	92	282
0.0.1	1	105	317
0.1.0	2	115	337
0.1.1	3	119	377
1.0.0	4	177	416
1.0.1	5	154	466
1.1.0	6	168	442
1.1.1	7	173	459

IV. Discussion

AWSS is the average of wall shear stress for the region where the WSS falls below 1 Pa. and hence the atherosclerotic prone area. Of the stenosed cases and normal bifurcation, normal has the maximum value for AWSS 0.82 Pa. (0.87 Pa. in 2D simulation) which confirms that a normal bifurcation is more resistant to intimal thickening and hence atherogenesis. Type 1.0.0 bifurcation has a larger low WSS region and a lesser average value of WSS for that area which will promote more plaque formation

An increase in vorticity indicates that there is more vortex formation. This will cause blood flow recirculation and stasis which is an already established reason for atherogenesis. AAV follows the inverse trend of AWSS. AAV has a minimum value for normal bifurcation for which AWSS is maximum. This is found true in both 3D and 2D simulations.

A lesion in LM alone (type 1.0.0) promotes maximum atherogenesis. The observation during 2D simulation that 'the presence of lesions in either distal main branch and side branch or both along with LM lesion (Type 1.1.0, Type1.1.1 and Type 1.0.1) increases AWSS and hence resist further atherogenesis better' holds true in 3D also.

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