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Analysis of the effects of plaque deposits on the blood flow through human artery

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ABSTRACT

Arterial disease (atherosclerosis) tends to be localized and results in a narrowing of the artery lumen due to plaque deposits (known as a stenosis). Because of this coronary arteries narrow down to the extent that they are unable to transport sufficient blood to the heart muscle for it to function efficiently. This leads to heart attack and stroke, which is the largest single cause of mortality worldwide. The objective of the present study is to analyze numerically the effects of plaque deposits on the blood flow through human artery. The ability to describe the blood flow through stenosed vessels would provide the possibility of diagnosing the disease in the earlier stages. Numerical simulations of blood flow and tissue interaction (Fluid-Solid Interaction) are performed using commercial codes such as CFX and ANSYS. The blood is considered as incompressible, non-Newtonian fluid and the arterial wall tissue as isotropic, elastic material with uniform mechanical properties. Numerical simulations are performed for different area constrictions and for various sizes, shapes and length of stenosis. The importance of using fluid-structure interaction (FSI) type of simulations is demonstrated by comparing the FSI and non-FSI results. The hemodynamic factors like wall shear stress, pressure and velocity distributions are analyzed. Study of generation of recirculation zones at various locations near the plaque and plaque progression zones is performed.

Keywords— **Atherosclerosis, Fluid-Structure Interaction, Plaque Deposits, Stenosis**

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I. INTRODUCTION

The human blood consists of formed elements that are suspended in plasma. The formed elements of blood are red blood cells, white blood cells and platelets. Almost all vessels (arteries and veins) carrying blood within the body are flexible. Arterial disease (atherosclerosis) tends to be localized and results in a narrowing of the artery lumen due to plaque deposits. Deposition of the plaque in the artery is called stenosis and artery with plaque deposition is called as the stenosed artery. The plaque deposits are nothing but the fatty deposits like cholesterol, unwanted materials present in the blood. Because of such deposition coronary arteries narrow down to the extent that they are unable to transport sufficient blood to the heart muscle for it to function efficiently. This leads to heart attack and stroke, which is the largest single cause of mortality worldwide. The blood flow behavior through flexible blood vessels (artery) could play an important role in the fundamental understanding, diagnosis and treatment of many cardiovascular diseases.

Figure 1 shows the simple geometry of the artery with plaque deposition.

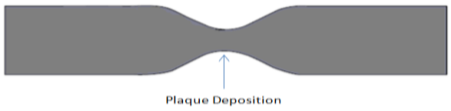


Fig. 1 The geometric model of the stenosis

The stenosis can cause turbulence in blood and reduce the blood flow by means of viscous head losses and flow choking. The best indicator for surgical treatment of atherosclerosis is the degree of stenosis. For diagnosis of a disease numbers of in vivo experimental techniques are available to get information about percent stenosis. But these techniques give little information about hemodynamic factors like blood velocity, wall shear stress, and pressure distribution. The experimental technique are namely X-ray contrast angiography, Doppler ultrasound, Magnetic Resonance Imaging (MRI), MR angiography etc. The application of the computational techniques (CFD) has become an important tool in the investigation of the blood flow in artery. This is because its ability to simulate wall shear stress, velocity and pressure fields in virtual models of the system. The numerical analyses are also helpful in the prediction of outcome of interventions and improve the treatment strategies. And also when compared to experimental investigations, computational methods are less time consuming and less costly.

A. Computational Fluid Dynamics

Computational Fluid Dynamics or CFD is the analysis of the systems involving fluid flow, heat transfer phenomena by means of the computer based simulation. This is very powerful technique and covers wide range of applications like:

1. Aerodynamics of aircrafts and automobiles.
2. Hydrodynamics of ships.
3. Power plant: Combustion in IC engines and gas turbines.
4. Electrical and electronic engineering.
5. Chemical process engineering etc.
6. Biomedical Engineering

Fluid flows are governed by the partial differential equations (PDE) which represents the conservation laws for mass, momentum and energy etc. In CFD analysis procedure these PDE's are converted into set of algebraic equations which can be solved using digital computers. These algebraic equations are solved by iterative method until the desired accuracy is reached.

B. Fluid-Structure Interaction

In Wikipedia the definition of fluid-structure interaction (FSI) is "FSI occurs when a fluid interacts with the solid structure, exerting pressure on it which may cause deformation in the structure and thus alter the flow of fluid itself". Such kind of interaction occurs in many natural phenomena and man- made engineering. Simulations of such type of problem are become more important in the field of the numerical analysis. In order to solve such FSI problem structure and fluid models have to be coupled. Fluid and solid solvers are used to solve respective domains and coupling is used to interchange results between two solvers. Interchange of the results has been considered one of the challenging tasks due to non-linear nature of the fluid solid interface. Much commercial software are developed to simulate FSI problems like ANSYS, ADINA, COMSOL etc. There are mainly two types of the coupling as following.

1) One way coupling: During the first step, converged solution of the fluid calculation provides the forces acting on the structural member. These forces are interpolated to structural mesh and the converged solution is obtained by the structural solver with those fluid forces as the boundary conditions. Because of this structural mesh is deformed. But these displacement values are not sent back to the fluid mesh. This procedure is repeated till both force and displacement values are converged below predetermined values. Here only one way data transfer take place i.e. only force data is transferred from fluid solver to structural solver, no displacement data is send back from the structural solver to fluid solver. Therefore such type of the coupling is called as the one way coupling of the data transfer.

2) Two way coupling: During the first step, converged solution of the fluid calculation provides the forces acting on the structural member. These forces are interpolated to structural mesh and the converged solution is obtained by the structural solver with those fluid forces as the boundary conditions. Because of this structural mesh is deformed. These displacement values are interpolated at the fluid mesh which results in the deformation of the fluid domain. This procedure is repeated till both force and displacement values are converged below predetermined values. . Here two way data transfer take place i.e. force data is transferred from fluid solver to structural solver and displacement data is send back from the structural solver to fluid solver. Therefore such type of the coupling is called as the two way coupling of the data transfer.

C. Literature Review

David N. Ku [1] in his study mentioned fluid mechanics approach for understanding blood flow through human artery. Blood flow in arteries is dominated by unsteady flow phenomena. He mentioned non-dimensional frequency parameter, the Womersley number, governs the relationship between the unsteady and viscous forces. Each fluid mechanics aspect plays a role in the generation, detection and treatment of arterial disease. He also mentioned different in vivo experimental techniques (as mentioned in introduction) which are available for diagnosis of disease. These techniques give information about stenosis, flow rate, nature of plaque with distinct advantages and disadvantages.

Fuat Yilmaz and Mehmet YasarGundogdu [2] studied Critical review on blood flow in large arteries. The purpose of this study is to present of viewpoints on analysis of blood rheology, blood viscosity models, and physiological flow conditions. The blood viscosity models in literature are divided in two main categories namely; Newtonian viscosity models (Lee and Steinman, Einstein) and non-Newtonian viscosity models (power law, carreau, cross). In this study the physical nature of human blood and its viscosity models are well documented.

BahtiyorBuriev et al. [5] studied Fluid-structure interactions of physiological flow in stenosed artery. The geometric models are considered with three different types of

constriction of cross-sectional area of blood vessel (25%, 50%, and 75% of constriction). Analysis is done using commercial software ADINA 8.4 which is developed by finite element method. The comparison is done of the wall shear stress with or without the fluid-structure. In the axial velocity profiles of no-FSI and FSI models were compared, and results of velocity profiles were very much same for both models. The wall shear stress rose sharply and declined quickly reversing its direction at the post-stenosis region is observed.

M. Alishahi et al. [6] studied numerical simulation of blood flow in a flexible stenosed abdominal real aorta. The blood is taken as incompressible, non-Newtonian fluid. And the arterial wall tissue is considered as isotropic, elastic material with uniform mechanical properties. The software ANSYS Multi-physics was used for the FSI simulations between the arterial wall and blood flow in arteries. The results using two models with rigid and flexible walls are presented and compared. Result shows that the blood pressure is lower by 15% for the flexible wall model as compared to the rigid model.

AbsaarUlJabbar et al. [7] have done numerical analysis of pulsatile blood flow around different plaque shapes in human carotid artery.For this purpose three plaque shapes were considered namely trapezoidal, elliptical and triangular. These shapes are taken with same base, height measurements and same area reduction of 30% in artery. Tuning fork model is used for arterial wall. Various CFD simulations were performed to analyze the effect of different plaque shapes on blood flow. Comparison of results showed that trapezoidal shape of the plaque has more effect on blood flow producing highest flow velocities and wall shear stresses.

Sanjeev Kumar and ChandrashekharDiwakar [8] developed a mathematical model of power law fluid with an application of blood flow through an artery with stenosis. In this model they considered artery as axisymmetric circular cylinder and blood flow within this axially symmetric stenosed artery. Power law viscosity model was used for non-Newtonian blood and blood flow is considered as steady and laminar. The governing equation for laminar, incompressible and non-Newtonian fluid (power law fluid) flow subject to the boundary conditions is solved numerically. The analytical expressions for pressure drop, flow rate and wall shear stress have been obtained. Here in this study it is observed that if the height of stenosis is increased then the pressure drop and flow rates are also increased.

Alvaro Valencia and Martin Villanueva [10] studied unsteady flow and mass transfer in models of stenotic arteries considering fluid- structure interaction. Symmetric and non-symmetric stenosed cerebral arteries models were considered for the numerical study. The Carreau non-Newtonian viscosity model is used for non-Newtonian blood. Various hemodynamic factors and effects of inlet boundary conditions (such as pressure inlet or velocity inlet) are studied. It is observed that inlet boundary condition of pressure is useful for obtaining realistic results in stenosed artery.

Donald F. Young and Frank Y. Tsai [13] have studied flow characteristics in models of arterial stenosis (part I-steady flow). In this study a number of steady flow in vitro experiments are described. In thisimportant factors including pressure drop, flow separation, and turbulence arestudied for tubes which are locally constricted. The axisymmetric and non-symmetricplastic models having different area constrictions and lengths of constriction were used in the experiments.The readings are taken for Reynolds numbers varying between 100-5000. Theregion of separated flow was observed. And critical Reynolds numbers for transition from laminar toturbulent flow were measured using hot-film probes. Results of investigations show strong influence ofgeometry, both the size and shape of stenosis on flow characteristics.

Donald F. Young and Frank Y. Tsai [14] have studied flow characteristics in models of arterial stenosis (part II-unsteady flow). In this study, the effect of unsteadiness is determined experimentally using the same models used in the part-I. Oscillating simple harmonic flow of fluid was used and the pressure drop was determined. An approximate equation for calculating the pressure drop across a stenosis was developed and validated experimentally. It was found that the oscillating flow was more stable than the corresponding steady flow for small area reduction. For the severe area reduction in artery the oscillating flow was slightly less stable than for steady flow.

Previous literature gives lots of information on blood flow behavior through stenosed artery. Various numerical studies are mentioned here with different assumption related to blood and arterial wall material. In this study work is focused on fluid- structure interaction simulation of blood flow through stenosed artery.

II. PROBLEM DESCRIPTION

Experimentally it is proved that blood is non- Newtonian fluid and blood flow through artery is unsteady, periodic phenomena. In previous literature various analyses is done with different considerations related to blood, blood flow, arterial wall material and size and shape of stenosis. Each assumption related to any parameter leads the solution away from the practical or real case.

The objective of this study is to analyze numerically (using CFD) the effects of the plaque deposits on blood flow through human arteries. Numerical simulations of fluid flow and tissue interaction (Fluid-Solid Interaction) are done considering the blood as incompressible, non-Newtonian fluid, and the arterial wall tissue as isotropic, linearly elastic material with uniform mechanical properties. The commercial codes such as CFX and ANSYS are used for the analysis.

III. OBJECTIVE AND SCOPE OF THE STUDY

A. Objective of the Study

1) Numerical (CFD) analysis of the effects of the plaque deposits on blood flow through human artery.
2) To develop method for the simulation of blood flow through stenosed artery using two way coupling Fluid-Structure Interaction (FSI) analysis.
3) To investigate and quantify the differences in the results of the FSI and non-FSI simulations.
4) Study of effects on blood pressure, velocity fields and wall shear stress region due to presence of plaque deposits in artery.
5) Study of generation of plaque progression zones in stenosed artery.

B. Scope of the Study

In this work, study will be done about various hemodynamic factors, recirculation zones. This study will helps in determining locations of plaque progression and plaque rupture zones in stenosed artery. Such numerical simulation helps in understanding, diagnosis and treatment of arterial diseases. Such simulations help the people working in the field of physiological fluid dynamics as well as the medical practitioners for the development of new diagnosis tools for arterial diseases.

IV.METHODOLOGY OF THE STUDY

The model problem of blood flow through a stenosed artery is solved using the commercial code ANSYS and CFX. Simulation will be performed for different area constrictions (for different height of stenosis). Analysis is done for wall shear stress, pressure and velocity distributions. Study is done about generation of recirculation zones and plaque progression zones. These results will be compared with results of healthy artery (artery without stenosis). For FSI simulations CFX and Transient Mechanical are coupled in the ANSYS workbench. Two way coupling is used for data transfer (force and displacement) between fluid solver (CFX) and structural solver (Transient Mechanical). Governing equations of fluid flow are solved in CFX while structural motion equations for structural movement are solved in the Transient Mechanical. The data transfer between the two solvers is automated by MFX branch of the ANSYS Multi-Field solver. Non-FSI simulations (i.e. for rigid artery) are carried out in the CFX fluid solver. CFD post is used for the examining and analysing the results.

Two models, (a) a rigid wall model (Non-FSI) and (b) a flexible wall model (FSI) are considered in this study. In the first model wall deformation is not allowed, while in the second model wall deformation is taken into account. Comparison of the results is done between FSI and Non-FSI case. For each wall model three geometries are taken according to different area constriction; 56%, 89% of area constriction and one geometry of healthy artery (Artery without plaque deposition). Results are also compared for arteries with different stenosed rates.

A.Fluid modeling

Naturally blood flow in the artery is unsteady state, periodic phenomena but this analysis is carried out with assumption of steady blood flow. The flow is assumed as the incompressible, laminar and blood as the non-Newtonian fluid. The blood density is taken as the 1060 kg/m^3. In Literature, it is stated that limiting shear rate which represents the transition from non- Newtonian to Newtonian nature of blood is taken as100 s^{-1}. If the shear rate is greater than 100 s^{-1} which is the case in larger arteries, blood is modelled as Newtonian fluid with a constant viscosity of 3.5mPa.s. For small shear rate blood is modelled as non-Newtonian fluid. There is various viscosity models available in literatures which represent the non- Newtonian rheology of blood. In this study, Cross viscosity model is used to represent the non-Newtonian nature of the human blood. Cross model assumes viscosity varies according to law,

$$\frac{\mu - \mu_\infty}{\mu_0 - \mu_\infty} = \frac{1}{(1 + (t * \gamma)^n)}$$

Where, 'μ' is dynamic viscosity, 'γ' is shear rate, 't' is time constant and 'n' is flow behaviour index. And n = 1.028, t = 1.007sec, μ_∞= 0.0345 poise (High shear viscosity) and μ_0= 0.56 poise (Low shear viscosity).

Cross model shows effective viscosity is dependent on shear rate only. In this study, blood flow is considered as single phase flow. This model does not consider aggregation and deformation of Red Blood Cells (RBCs) which affects the blood viscosity. That factor is important in modelling and simulation of Multi-Phase flow including plasma, RBCs, platelets and leukocytes.The governing equations for conservation of mass and momentum of the incompressible blood are described below:

$$\frac{\partial \rho}{\partial t} + \frac{\partial (\rho U_j)}{\partial x_j} = 0$$

$$\frac{\partial (\rho U_i)}{\partial t} + \frac{\partial (\rho U_i U_j)}{\partial x_j} = -\frac{\partial p}{\partial x_i} + \frac{\partial}{\partial x_j}(\mu_{eff}(\frac{\partial U_i}{\partial x_j} + \frac{\partial U_j}{\partial x_i})$$

For i, j =1, 2, 3.

Where, 'μ_{eff}' is the fluid effective viscosity.

B. Solid Modelling

The arterial wall material is considered as the isotropic, linearly elastic material with constant mechanical properties. The Young's Modulus of the arterial wall is taken as 1.08 MPa. And the arterial wall is taken with Poisson ratio of 0.49 and with density of 1200 kg/m^3. The wall thickness is uniform at 2 mm. The movement of the elastic solid is described by mathematical equation as below;

$$\rho_w \frac{\partial^2 u}{\partial t^2} = \rho B + (\lambda + \mu) del.e + \mu\, div(del.u)$$

e = div u

Where, 'u' is the displacements vector, 'ρ_w' is wall density; 'B_i' are the components of the body forces acting on the solid. 'λ' and 'μ' are the Lame's coefficients.

C.Geometry

Three geometries are considered for the analysis with different area constriction. Two geometry with 56%, 89% of area reduction in original lumen area and one geometry of healthy artery (Artery without plaque deposition) is created. These geometries are created in the design modeler tool in ANSYS workbench. For all geometry diameter and total

length is taken as 18.9 mm and 702.4 mm respectively. For FSI simulations thickness of artery is constant at 2 mm throughout the length. The stenosis starts at distance of 263.41 mm from the inlet. The length of the stenosis (plaque deposition) is 75.58 mm. The shape of the axisymmetric stenosis is specified as the cosine curve as follows;

$$\frac{R}{R_0} = 1 - \frac{\delta}{2R_0}(1 + cos\frac{\pi z}{Z_0})$$

For,"$-Z_0 \leq z \leq Z_0$". Here $2Z_0$= 75.58 mm (Length of the stenosis), R_0 is radius of normal artery; R is the radius of artery in stenosed region. 'δ' is the height of the stenosis at the throat('R_0-R' at throat) which is 3.15 mm for 56% stenosis and 6.3 mm for 89% stenosis.

D. Computational mesh

The mesh for all geometry is created in the ICEM CFD software in ANSYS workbench. For structural meshing 'mapped face meshing' method is used with refinement provided at the stenosis section. For fluid domain 'Sweep' method is used with inflation. Fine layers with growth rate are provided in the fluid domain at fluid solid interface. Firstly grid independence test (GIT) for FSI simulation is carried out for 89% stenosed artery for confirming independence of solution on the mesh size.

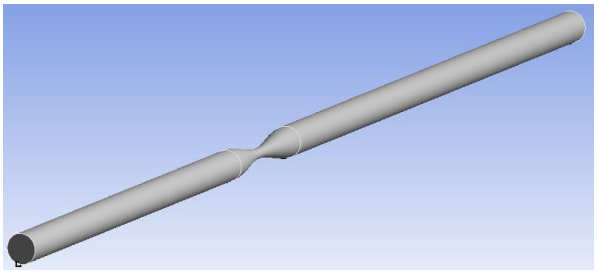


Fig. 2 Geometry of 89% stenosed artery

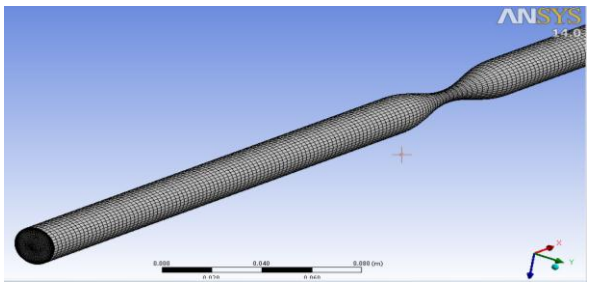
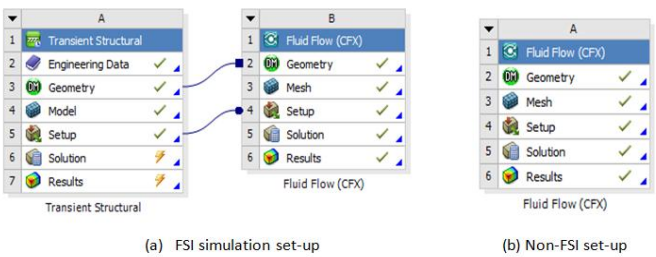


Fig. 3 Computational mesh of Fluid Domain for 89% stenosed artery

E. Simulation Set-up

Analysis set up for the FSI and non-FSI simulations is shown in the Figure 4.



(a) FSI simulation set-up (b) Non-FSI set-up

Fig. 4 Simulation set-up

*1) Transient Mechanical (ANSYS Mechanical) set-up (For only FSI simulations):*Set up in the Mechanical is simple as compared to CFX-Pre. The end edges of the artery are fixed and the calculated fluid forces are applied on the wall surface which is described as a fluid solid interface. Apart from this, it follows the same transient setups like time step size and end time as in CFX-Pre.

2) CFX set-up:

TABLE I
BOUNDARY CONDITIONS

Parts of the Domain	Type of the boundary conditions	
	Type	Condition
Inlet	Inlet	Velocity Inlet(45 mm/s)
Outlet	Opening	Pressure (10KPa)
Artery wall	Wall	No slip Wall

Reynolds number of 500 is applied for all simulations which is suitable for larger arteries. Static gauge pressure of 10 kPa is applied at outlet which is the normal human blood pressure.

TABLE II
SOLVER SET-UP

Tab	Settings		Values
Physics	Fluid		Blood
Definition	Analysis Type		Steady State
	Solver Parameters	Advection Scheme	High Resolution
		Convergence Control	Auto-Timescale
		Convergence Control (Mesh Displacement- FSI)	Timescale Control (coefficient loop)
	External Coupling	Solve ANSYS field (FSI)	Before CFX field

V.RESULTS AND DISCUSSION

A. FSI results comparison of artery with different area constriction

Figure 5 shows the variation of the blood pressure along the axis of the artery for three different percentage of the plaque deposition. Minimum pressure is observed at the throat of the stenosis and maximum pressure drop takes place across length of the stenosis. Maximum pressure drop increases with degree of the stenosis. Maximum pressure drop take place for the 89% stenosed artery. Pressure distribution curve follows approximately similar pattern for healthy and 56% stenosed artery.

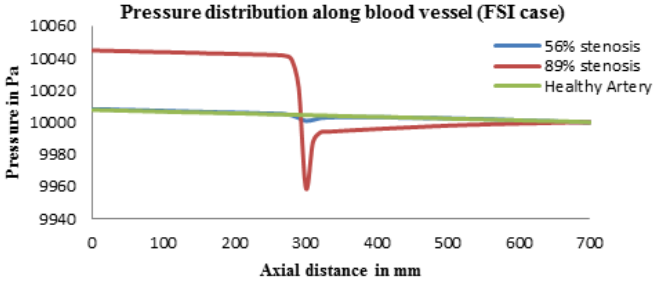


Fig. 5 Pressure Distribution along blood vessel

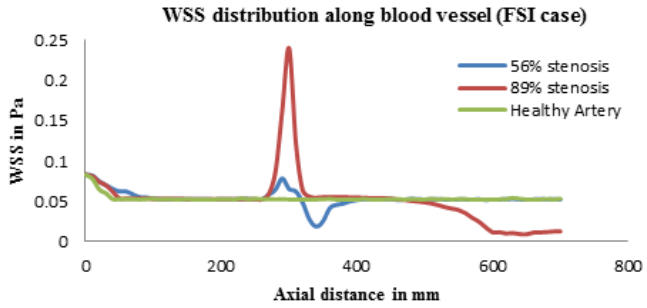


Fig. 6 WSS distribution along the wall of blood vessel

Figure 6 shows the variation of the wall shear stress along the wall of artery for three different percentage of the plaque deposition. Maximum wall shear stress is observed at the throat stenosis and its value increases with degree of stenosis. For 89% and 56% of stenosis 0.24 Pa and 0.0654 Pa of WSS are observed at the throat respectively. For 56% stenosed artery, minimum WSS is observed at downstream section of the stenosis. For 89% stenosis min WSS is observed far away from the stenosis region. For healthy artery WSS is almost constant at the value 0.052 Pa along wall of vessel.

Figure 7 shows the variation of velocity along the axis of artery for three different percentage of the plaque deposition. Maximum velocity is attained at the throat of the stenosis. 89% of stenosis gives the high velocity of the value 0.2429 m/s at throat as compared to 56% stenosis. 56% stenosis gives 0.1 m/s velocity at the throat. For healthy artery velocity is almost constant at 0.04925 m/s along axis.

Figure 8, 9, 10 shows the total mesh displacement at FSI interface for 56%, 89% stenosed, and for healthy artery respectively. For 56% stenosed artery maximum total mesh displacement (about 3.6 mm) is observed at upstream and downstream section of the plaque deposition as shown in figure 8. For 89% stenosed artery maximum total mesh displacement (about 4.4 mm) is observed at downstream section of plaque deposition as shown in figure 9. Healthy artery shows almost constant mesh displacement (of about 0.426 mm) throughout FSI Interface.

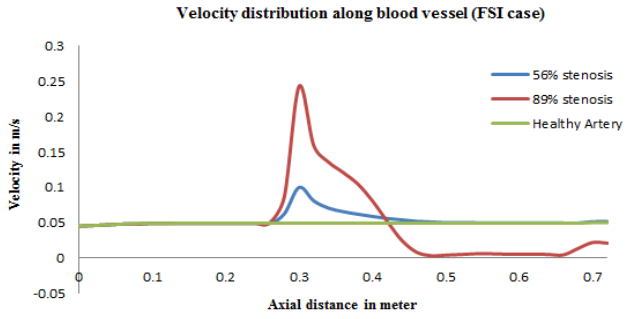


Fig. 7 Velocity distribution along axis of blood vessel

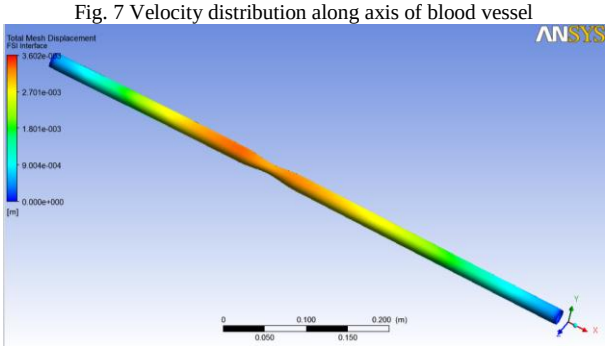


Fig. 8 Total mesh displacement at FSI interface for 56% stenosis

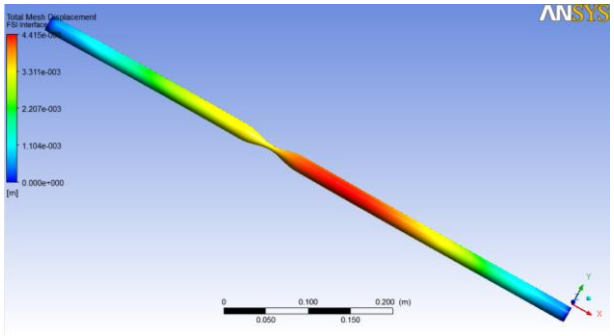


Fig. 9 Total mesh displacement at FSI interface for 89% stenosis

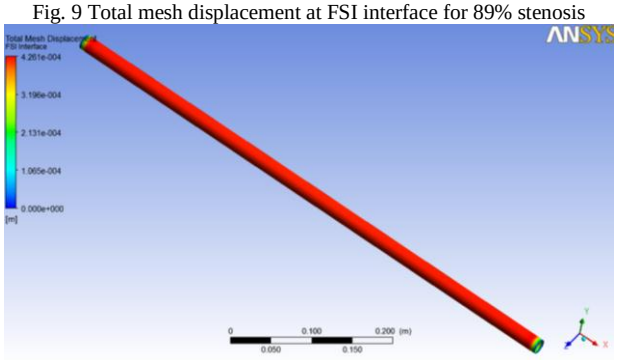


Fig. 10 Total mesh displacement at FSI interface for Healthy artery

B. Comparison of the results between FSI and non-FSI simulations

Figure11, 12, 13 shows pressure distribution along the axis of blood vessel for 56%, 89% stenosed, and for healthy artery respectively. In each figure comparison is given for two cases namely FSI and non-FSI. Axial blood pressure distribution follows normally same pattern with equal values for both FSI and non-FSI simulations for particular area constriction.

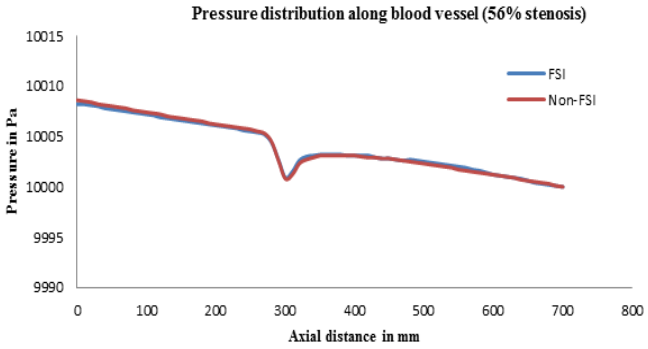


Fig. 11 Pressure Distribution along the blood vessel (56% stenosis)

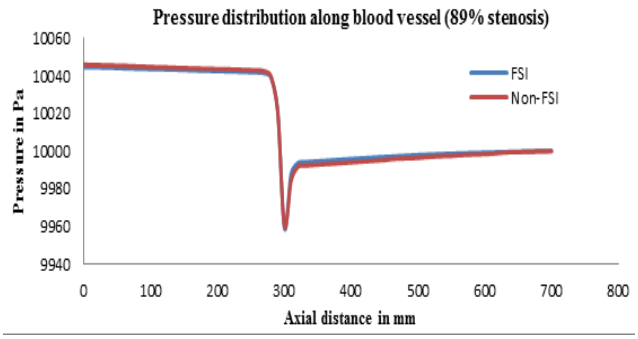


Fig. 12 Pressure Distribution along the blood vessel (89% stenosis)

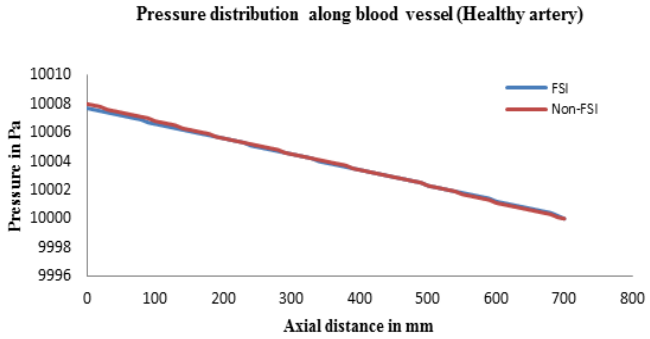


Fig. 13 Pressure Distribution along the blood vessel (Healthy artery)

Figure14, 15, 16 shows wall shear stress distribution along the wall of blood vessel for 56%, 89% stenosed, and for healthy artery respectively. In each figure comparison is given for two cases namely FSI and non-FSI.

In Figure14, both the FSI and non-FSI case shows normally same pattern of WSS distribution but there are slight changes in the peak WSS values and their locations. Maximum WSS is observed as 0.0784 Pa and 0.0823 Pa for FSI and non-FSI cases respectively at the same location. Minimum WSS are 0.01875 Pa and 0.01367 Pa for FSI and non-FSI simulations but their locations are slight different as shown in figure 14.

In Figure 15, both the FSI and non-FSI case follows normally same pattern with equal values of WSS distribution. But the location and value of minimum wall shear stress are different as shown in figure.

In Figure 16, it is shown that both the FSI and non-FSI cases follows approximately same pattern. But the FSI simulation result shows small fluctuations in wall shear stress value along the length of healthy artery.

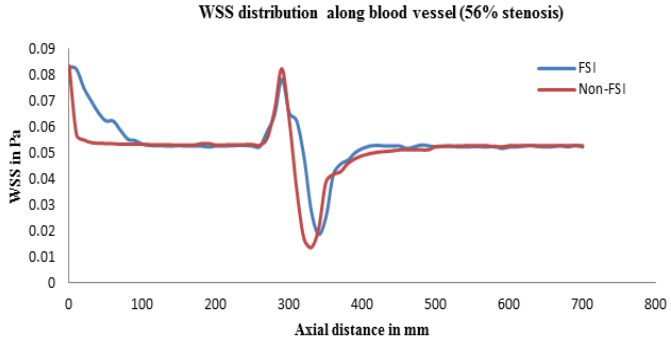


Fig. 14 WSS distribution along wall of blood vessel (56% stenosis)

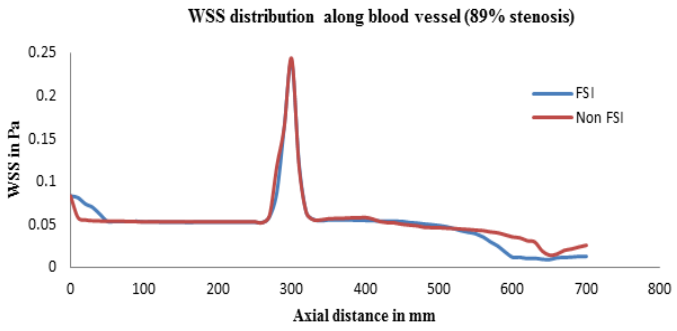


Fig.15 WSS distribution along wall of blood vessel (89% stenosis)

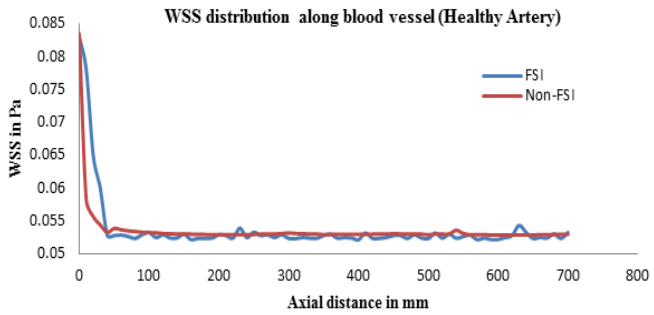


Fig. 16 WSS distribution along wall of blood vessel (89% stenosis)

Figure17, 18, 19 shows velocity distribution along the axis of blood vessel for 56%, 89% stenosed, and for healthy artery respectively. In each figure comparison is given for two cases namely FSI and non-FSI. In figure 17 it shown that both the cases follows the same pattern of velocity distribution but the magnitude of velocity is slightly higher at each location along axis for non-FSI case than FSI case.

Figure 18 shows that there are vast differences in the magnitude of the velocity between FSI and non-FSI simulation for 89% stenosed artery. Maximum velocity is observed at the throat with magnitude of 0.4011 m/s and 0.2429 m/s for non-FSI and FSI simulations respectively.For healthy artery velocity is almost constant along the axis of artery as shown in figure19. But there are difference in the magnitude of the velocity between FSI and non FSI case.

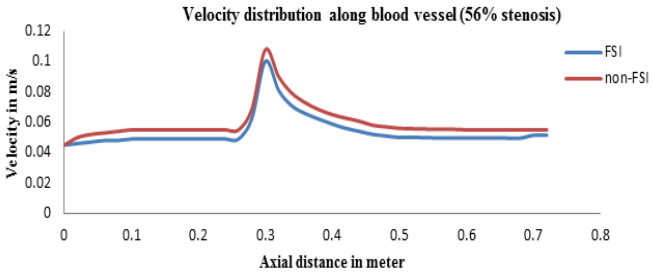


Fig. 17 Velocity distribution along axis of blood vessel (56% stenosis)

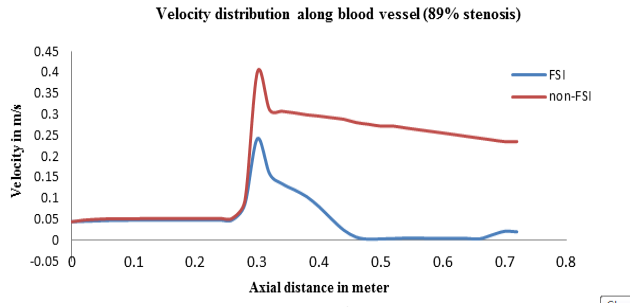


Fig. 18 Velocity distribution along axis of blood vessel (89% stenosis)

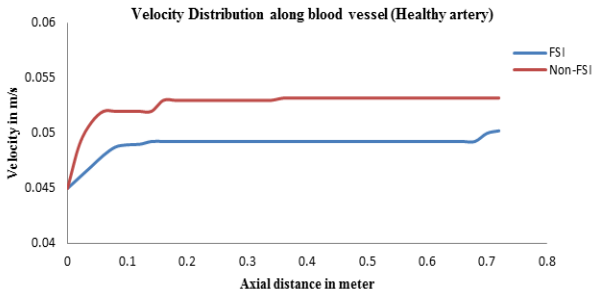


Fig. 19 Velocity distribution along axis of blood vessel (Healthy artery)

From the results we are able to know what changes happens in the hemodynamic factors like WSS, blood flow rate and

pressure distribution due to plaque deposits. Previous studies show that one of the main factors involved in plaque buildup is low wall shear stress. On the other hand plaque rupture can be due to high shear stress as it causes high flow changes in the vessel.

From the results of this study, it is noticed that there is sudden rise in blood velocity, shear stress on arterial wall at the throat of stenosis. Maximum pressure drop is observed across the stenosis. And low wall shear stress is observed at the downstream region of stenosis. Therefore there are possibilities of plaque buildup at downstream region of stenosis and possibilities of plaque rupture at throat of stenosis. Size of stenosis also has a great influence on blood flow parameters. Knowing all this plays an important role in the fundamental understanding, diagnosis and treatment of many cardiovascular diseases.

VI. CONCLUSIONS

In this study, numerical analysis of blood flow and tissue interaction (Fluid-Solid Interaction) is performed. In literature review, previous works related to blood flow through stenosed artery are summarized. Previous studies show that one of the main factors involved in plaque buildup is low wall shear stress. On the other hand plaque rupture can be due to high shear stress as it causes high flow changes in the vessel. From the results of current study, it is possible to identify the locations of low and high wall shear stress in arteries. This helps in the understanding and diagnosis of arterial disease. Now days CFD simulations along with the in vivo experimental techniques is used for diagnosis of arterial disease. CFD simulation of blood flow has become the cutting edge tool to investigate cardiovascular dysfunction and has great scope in future.

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REFERENCES

[1] David N. Ku., Blood Flow in Arteries, Annual Rev. Fluid Mechanics,29, pp.399-434,1997.

[2] Fuat Yilmaz and Mehmet YaarGundogdu, A critical review on blood flow in large arteries; relevance to blood rheology, viscosity models, and physiologic conditions, Korea-Australia Rheology Journal, 20(4), pp.197-211, 2008.

[3] Mir GolamRabby et al., Pulsatile non-Newtonian blood flow through a model of arterial stenosis, 5th BSME International Conference on Thermal Engineering, North South University, Dhaka, Bangladesh, pp.225-231, 2013.

[4] Mohsen Mehrabi and SaeedSetayeshi, Computational Fluid Dynamics Analysis of Pulsatile Blood Flow Behavior in Modeled Stenosed Vessels with Different Severities, Hindawi Publishing Corporation, 2012.

[5] BahtiyorBuriev et al., Fluid-structure interactions of physiological flow in stenosed artery, Korea-Australia Rheology Journal, 21(1), pp.39-46, 2009.

[6] M. Alishahi et al., Numerical simulation of blood flow in a flexible stenosed abdominal real aorta, ScientiaIranica B Journal, 18 (6), pp.1297-1305, 2011.

[7] AbsaarUlJabbar et al., Three-dimensional numerical analysis of pulsatile blood flow around different plaque shapes in human carotid artery, International Journal of Bioscience, Biochemistry and Bioinformatics, 2(5), pp.305-308, 2012.

[8] Sanjeev Kumar and ChandrashekharDiwakar, A mathematical model of power law fluid with an application of blood flow through an artery with stenosis, Advances in Applied Mathematical Biosciences, 4(1), pp. 25-36, 2013.

[9] Jonghwun Jung and Ahmed Hassanein, Three phase CFD analytical modeling of blood flow, Medical engineering and physics, 30, pp. 91-103, 2008.

[10] Alvaro Valencia and Martin Villanueva, Unsteady flow and mass transfer in models of stenotic arteries considering fluid- structure interaction.

