

Numerical Assessment of Turbulent flow Downstream of Stenosed Aortic Valve with flexible leaflets using Fluid-Solid Interactions Approach

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Abstract—Aortic valve stenosis can trigger regions of turbulent flow downstream of the aortic valve. This disturbed blood flow in this region can cause tissue damages due to increased levels of turbulent shear stress. The aim of the present work was to investigate the effect of aortic stenosis on turbulence parameters of the blood flow over flexible leaflets of aortic valve. In this study the turbulent flow was simulated numerically using $k-\epsilon$ realizable model. The interaction between fluid and structure fields was applied using an implicit iterative method. To characterize the turbulent intensity, turbulent kinetic energy was computed. The results show that the levels of turbulence increases with more severe stenosed valves. The results also revealed that the flexible behavior of the leaflets of the aortic valve can also disturb the flow which can produce regions of turbulence downstream of the aortic valve.

Keywords: *Aortic Valve, Stenosis, Blood flow, Turbulence*

I. INTRODUCTION

Aortic valve (AV) is one of the four valves of the heart which controls the blood flow out of left ventricle to the aorta. The aortic valve stenosis (AS) or progressive narrowing of the aortic valve is the most common valve disease (1). The causes of AS include, congenital heart defect, calcium build up on the valve and rheumatic fever (2). Although the blood flow downstream of the AV is assumed to be laminar with some local disturbances, diseases of AV such as the AS trigger the disturbances and increase the turbulence intensity in this region (3). Increase in turbulence intensity value, disaffects the function of the cardiovascular system in different ways. First of all disturbed blood flow may cause regions of altered shear stress increasing the risk of aortic wall calcification (4). In the other hand dissipation of the turbulent kinetic energy (TKE) into heat, causes irreversible pressure loss. To overcome this loss of mechanical energy the left ventricle has to respond with increased work which causes increased levels of stress on myocardium (5). In addition it has been shown that the shear stress produced by turbulence can cause pathophysiological changes like platelet activation (6) and damages like hemolysis (7). In order to investigate the

level of turbulence downstream of the AV several methods has been developed by researchers. *In vivo* Magnetic Resonance Measurement (MRI) method is commonly used by researchers to capture the dynamics of turbulence. In (5) the turbulence downstream of aortic valve has been investigated using MRI technique and a relation between TKE and irreversible pressure loss has been assessed in the ascending aorta of patients with aortic stenosis. In addition, *in vitro* Particle Image Velocimetry (PIV) has been used to investigate the hemodynamic of the blood flow experimentally by some other researchers. In a recent work (15), PIV measurements were conducted on different aortic configurations and it has been found that pathological aortic valve significantly alters hemodynamic parameters in the aorta.

Additionally, the flow domain can also be solved numerically using Computational Fluid Dynamics (CFD). In (8), (9) a rigid model of aortic coarctation and surrounding wall has been constructed and TKE energy was computed from numerical solution and resulting CFD data. In a similar numerical study (17), three-dimensional flow through a fixed mechanical heart valve has been simulated to investigate the counter rotating vertices downstream of the leaflets.

In the above mentioned numerical studies, vascular system has been modeled as a rigid structure; as a consequence deformation of flexible structure fields was neglected. In general the only approach capable of capturing the interaction between the blood and the surrounding tissues is the Fluid Solid Interactions (FSI) approach (10). From this point of view the FSI models are the most realistic and exhaustive computational tools. In (11) the FSI approach has been employed to a model of the left ventricle with both mitral and aortic valves. Consequently the characteristics of the blood flow predicted successfully, albeit the model's simplicity. In another recent work (12) the results of a numerical analysis of a bi-leaflet aortic valve with FSI approach was validated with *in vitro* experiments. The results revealed a great similarity between simulations and experimental tests. In a similar work (16), effect of existence of asymmetry in leaflets of the AV has been

investigated using a fully coupled FSI approach and it has been shown that the asymmetric geometry can cause a large flow shear stress on the cusps.

In this study the flow over and downstream of the aortic valve is modeled with three different fields, which are fluid (blood), rigid structure (walls) and flexible structure (leaflets) as shown in Fig.1. In this model, the blood flow exerts pressure on the leaflets. This pressure causes structural deformation big enough to change the blood flow itself. The time cycle involving the opening and closing phase of AV, in which the blood ejects from left ventricle to aorta through AV, is considered for analysis. An iterative implicit method has been used to capture the interaction between flexible leaflets and the blood flow.

II. METHODOLOGY

A. Geometrical model and tissue properties

An image involving the aortic valve and the sinotubular junction was obtained using Computerized Tomography Scan (CT-SCAN) technology. The two dimensional geometrical model for numerical analysis was created by using this image in commercial software Design Modeler[®] (ANSYS Inc.). As discussed above the walls were modeled as rigid structure with no deformation. Conversely the flexible leaflets of the AV were assumed to be elastic, incompressible and transversely isotropic. To compare the effect of the stenosis in turbulent parameters 3 different models with different stenosis severities were created. The Young Modulus of elasticity for healthy leaflets was set to 2 MPa, based on the values found in the literature (14). Also for partly Stenosed and sever stenosis models young modulus of elasticity of 10Mpa and 20Mpa were defined. The Poisson ratio also was set to a value of 0.499 (20). The density of the leaflet tissue was set to 1060 kg/m³, equal to blood density to avoid buoyancy effects. The grid for leaflets was generated with 6,842 prism elements using sweep method. The structure field was simulated with the commercial finite element solver ANSYS[®] (ANSYS Inc.).

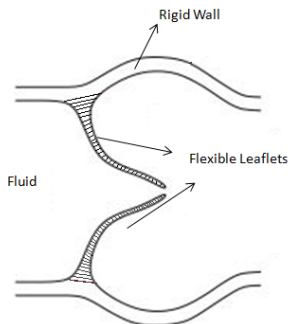


Figure 1 Aortic Valve with fluid, rigid structure and flexible structure zones.

B. Fluid problem and Inlet boundary conditions

The blood was defined as an incompressible fluid with constant density of 1060 kg/m³. Assuming blood as a Newtonian fluid, the dynamic viscosity was set to a constant value of 0.0037 Pa. s. The grid for blood field was generated with 40,856 prism elements using sweep method. The grid for the flow field is shown in Fig.2. Inlet boundary condition (BC) can be obtained using PC-MRI technology. In this paper, the inlet BC was defined as velocity inlet, based on a velocity profile found in literature (13). The velocity profile used as inlet BC is shown in Fig.3. The numerical simulations for the blood field were run through the commercial finite volume solver FLUENT[®] (ANSYS Inc.) release 14.0.

C. Turbulence Model

The Reynolds number for blood flow can be calculated based on the AV inlet diameter, blood velocity at inlet, blood density and blood dynamic viscosity using this equation:

$$Re_D = \frac{\rho U D}{\mu}$$

The peak calculated Reynolds number was 5205. The inlet velocity boundary condition is pulsatile; moreover we have a complex moving domain during the cardiac cycle. As a consequence it was expected to have a turbulence region downstream of the aortic valve. To simulate the turbulent flow a 2 equation RANS model, k- Realizable model was used. An important benefit of applying Realizable k- to Standard k- is that it predicts the spreading rate of the both planar and round jets more accurately and provides better performance for flows involving rotation, separation and recirculation (18). The k- Realizable model introduces an improved transport equation for turbulent dissipation rate, . To set the boundary condition for employing k- realizable model, turbulence intensity value must be defined correctly. This value varies with the Reynolds number and a default value of 1.5 % was defined based on the values found in literature (19). To satisfy the physics of the blood flow in the near wall regions and bridge the viscosity affected region between the vicinity of the endothelium and the turbulent flow region standard wall functions were used.

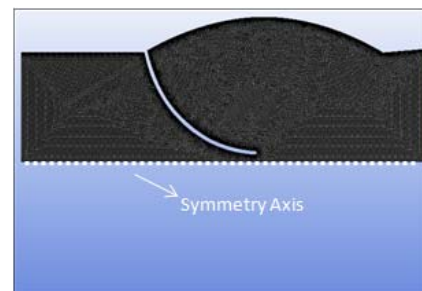


Figure 2. Generated 2-D grid for upper part of the AV with the symmetry axis

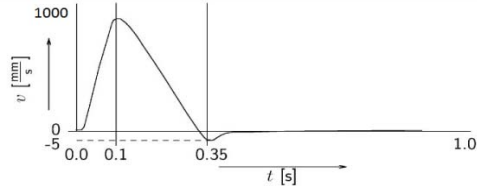


Figure 3. The velocity profile defined as inlet boundary condition for the AV

The standard wall functions give reasonable accuracy for a majority of wall bounded flows. The details about the k-Realizable turbulence model and standard wall functions can be found in (22).

D. FSI problem

As discussed above the blood flow and AV leaflets are strongly physically coupled. To employ the interaction between blood and flexible leaflets, an iterative implicit method was used, in which the blood flow and leaflet zones are solved separately within a time step, and the solution is obtained by iteration within a time step. The general scheme of this method is illustrated in Fig.4. The FSI scheme was generated using WorkBench[®] (ANSYS Inc.). Time step size of 4.5e-05 second was defined for FSI coupling. The simulation was set to start at the beginning of the phase in which the aortic valve starts to open. The iteration stops after a total time of 0.32 s, the duration in which the aortic valve gets closed completely, as defined in AV inlet boundary conditions.

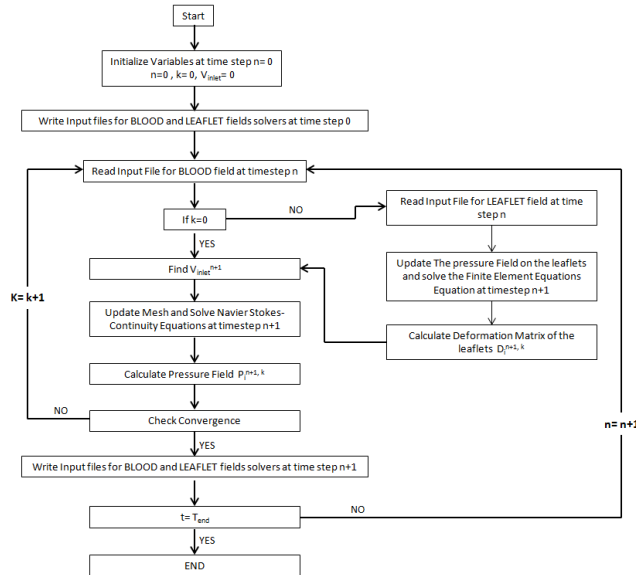


Figure 4. The scheme of the iterative implicit method for employing FSI method in this study

III. RESULTS

A. AV leaflets kinematics and validation of the model

To validate the model, AV leaflet movements of a healthy person was captured using *in vivo* echocardiography method. Model predictions on leaflet movements show good agreement corresponding to experimental data. Comparison of the echocardiography images with the simulation results of healthy model which corresponds to young modulus elasticity constant of 2 MPa is shown in Fig.5 for three different time intervals during the systolic phase. After reaching the max acceleration and peak systolic phase the leaflets oscillate two times according to the echocardiography video, which is also observed in the simulation results. The agreement between the experimental data and simulation results shows the validity and accuracy of the model which was created.

B. Leaflets: strains and wall shear stresses

Forces acting on the Endothelium of leaflets of the AV in laminar and turbulent flow have a same hemodynamic component, shear stress, but in turbulent flow both the duration of the force and its direction is fluctuating (22). Computing the wall shear stress (WSS) near the wall using experimental *in vivo* measurements is a very hard procedure, thus the experimental computation of the WSS is generally neglected by researchers and it is only restricted to numerical simulations and validated by *in vitro* experiments. Time averaged wall shear stress (TAWSS) during systolic phase which is the shear load over time that is subjected to aortic valve was calculated according to simulation results. The results are shown in table 1.

C. Blood flow dynamics

To investigate the dynamics of the blood flow passing the AV, vectors of velocity for four different time intervals during simulation time are plotted in Fig.6, for the model with Young Modulus of elasticity of 10 MPa, in which the color scale is the same for all contours. At max acceleration the peak orifice jet velocity was 1.95 m/s.

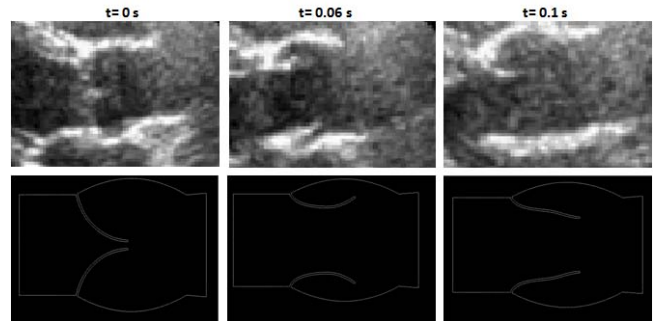


Figure 5. Comparison of the movement of the leaflets between simulation results and experimental *in vivo* data

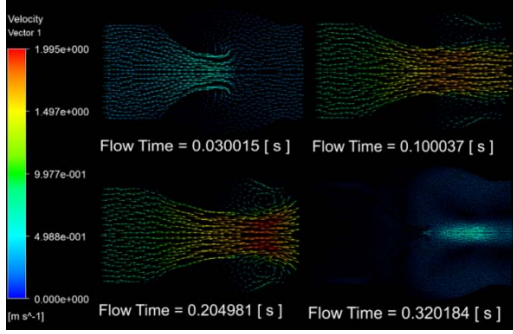


Figure 6. Velocity vectors plotted in four different time intervals according to simulation results for case 2 with Young Modulus of 10 MPa.

As the inlet blood velocity increases a jet flow emerges into the ascending aorta and the oscillation of the leaflets due to flexibility, disturbs the flow creating strong separation zones. It can be noticed that a large scale helical flow which rotates clockwise forms downstream of the AV. During decelerating phase as the velocity decreases, the AV starts to close, causing this helical flow break up to smaller eddies which disturbs the flow.

Kinetic energy of fluctuations can also be calculated in the flow field, to investigate these variations. As the model is 2-D, the fluctuating velocity in z direction is zero. Thus, the turbulent kinetic energy (TKE) can be defined as:

$$E_k = \frac{1}{2} (u_{rms}^2 + v_{rms}^2)$$

Where u_{rms} and v_{rms} are the root mean square values of velocity fluctuations in two directions. The distribution of the TKE has been plotted in Fig.7 for different three time intervals during simulation time for the case 2 with Young Modulus of 10MPa.

The numerical analysis results show how the fluctuations start as the flow passes the AV in the vicinity of the leaflets. It can be observed that fluctuations increase over time thus TKE increases after peak systole and reach maximum values during deceleration phase. The main cause of these fluctuations is the existence of high velocity gradients which leads to small differences in location of shear layer. Considering the turbulent flow characteristics it is expected to have a very low TKE in the centerline of the aortic valve jet which is also observed and is in agreement with CFD results. However due to flexibility and oscillating behavior of the leaflets of the AV, the numerical analysis results indicate some low levels of TKE in some parts of the center line of the jet exists. It is also observed that the TKE increases during deceleration phase of the systolic cycle.

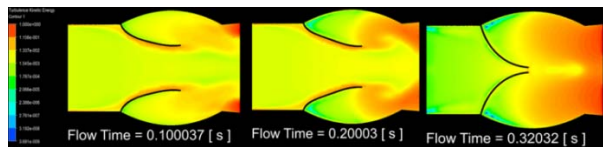


Figure 7. Distribution of total kinetic energy for case 2 for three different time intervals during simulation time

In Fig8. TKE for three different cases at maximum inlet velocity are shown. It can be revealed that the TKE for more stenosed aortic valves is in higher levels in magnitude. This can be related to higher velocity gradients in jet orifice area of the stenosed valves which is the main cause of the disturbance of the flow. In addition the turbulent field is also bigger and more extended in more stenosed aortic valves.

IV. CONCLUSIONS

In this study the flow over aortic valve was investigated using an implicit iterative FSI method. The leaflets of the aortic valve were assumed to be flexible. Because of pulsatile behavior of the flow, moving geometry and relatively high values of Reynolds number, the flow was predicted to be turbulent and a two equation RANS turbulent model, k- realizable was employed to investigate the characteristics of the turbulence. Three different models with different young modulus of elasticity for leaflets of the aortic valves were created and levels of turbulence in three different aortic models were investigated. The results show that the turbulence levels in higher levels of stenosis increases significantly. It has been shown that the oscillation of the leaflets due to flexibility has an important impact in production of regions of turbulence, revealing the fact that neglecting flexibility of the leaflets when simulating the turbulent blood flow can affect the accuracy of the results in an important manner. Although, the accuracy of this work is limited due to 2-D representation of the blood flow model, the results of simulation were in qualitative agreement with experimental results found in literature. Revealing the fact that numerical analysis of the flow field can provide further information which is not available in some *in vivo* methods.

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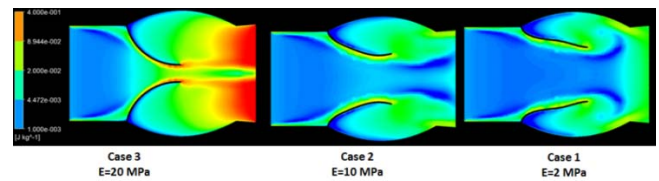


Figure 8. TKE distribution at maximum inlet velocity for three different cases

Table1. Simulation results for three different cases

	Young Modulus of Elasticity (MPa)	Maximum Orifice Jet (m/s)	TAWSS (Pa)	Maximum TKE (j/kg)
Case 1	2	1.4	11.2	6.18 e-2
Case 2	10	1.95	18.4	7.51e-1
Case 3	20	5.08	23.78	9.69e-1

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