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The Impact of Left ventricular Outflow Tract Obstruction on the Accuracy of Doppler Hemodynamic Parameters

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ABSTRACT

Doppler echocardiography is the most widely used tool to asses prosthetic valve function using quantitative parameters of prosthetic heart valve function including peak velocity, pressure gradient (*ΔP*), Effective Orifice Area (EOA), and Doppler Velocity Index (DVI). However, in cases of altered Left Ventricular Outflow Tract (LVOT) such as Subaortic Stenosis (SAS), the accuracy of current Doppler hemodynamic parameters proposed by the American society of echocardiography have not been clarified yet. Therefore, it is necessary to evaluate the current Doppler parameters to allow for accurate and early detection of mechanical valve dysfunction which is a key factor for successful treatment.

The goal of this project is to perform a numerical simulation of blood flow through Bileaflet Mechanical Heart Valves (BMHV) under clinically relevant flow conditions. In one scenario we simulated blood flow through normal BMHV with SAS whereas in another scenario we simulated dysfunctional BMHV with concomitant presence of SAS. Finally, we showed that SAS has a significant impact on Doppler parameters, especially in cases of concomitant SAS and dysfunctional BMHV. EOA was selected as the most accurate criterion among other Doppler parameters.

Keywords: LVOT Obstruction, Prosthetic Heart Valve, Echocardiography, CFD, Hemodynamic.

INTRODUCTION

The aortic valve is located between the left ventricle and the ascending aorta to facilitate the flow of blood with minimal impediment during the ejection phase and to close with minimal leakage during the ventricular filling phase [1, 3].

Aortic stenosis (AS) represents a pathological narrowing of the aortic valve. The presence of AS forces the left ventricle to pump extra hard to compensate blood volume through the narrowed region, leading to left ventricle hypertrophy which may create subaortic stenosis (SAS) below the valve due to protrusion of hypertrophied septum in to outflow tract and can be asymmetrical [5] . Blood flow through valves with AS or SAS causes complications that can lead to turbulent flow downstream of the valve, and such turbulence plays a pivotal role in thrombus formation and embolization by activation of platelets [1-4]. When AS is severe, aortic valve replacement is the only treatment to avoid possible congestive heart failure and to improve heart performance [5]. However, complications due to prosthetic valve replacement may arise including, pannus formation which causes an abnormal layer of fibrovascular tissue or granulation tissue. Pannus formation in a prosthetic heart valve occurs in response to healing processes, called tissue overgrowth, which occur in the tissue valve interface and creeps along the suture lines leading to prosthetic valve stenosis or SAS in the left ventricle outflow tract (LVOT). In addition, SAS can be congenital [7, 8]. AS can be corrected by prosthetic valve replacement, but SAS often remains after operation unless septal myectomy is done [5].

Early detection of valve dysfunction is a key factor for successful treatment. Although, Cinefluoroscopy or Computed Tomography can be used to assess mechanical heart valves (MHV) and the severity of AS, these methods are not used with patients for routine follow-up due to X-ray exposure risk. Doppler Echocardiography is the current method of choice for the diagnosis of valve dysfunction. Due to limited visualization capacity, Doppler-derived hemodynamic parameters are mainly used in the evaluation process. By measuring the velocity jet continuously with good alignment of the Doppler beam with the flow axis, one can estimate the pressure gradient (ΔP), effective orifice area (EOA), and Doppler velocity index (DVI). However, the concomitant dysfunctional prosthetic heart BMHV and SAS might affect the accuracy of the assessment of the dysfunctional valve hemodynamic performance with SAS due to:

1. Reverberations caused by movement of leaflets and acoustic wave reflection from leaflets surface.
2. Acoustic shadowing from the sewing ring.
3. Doppler echocardiography may record localized high pressure gradients so it is difficult to determine if the high gradient is due to valve dysfunction of the prosthesis or to localized pressure gradient.

Hence, it is necessary to develop new non-invasive parameters, allowing accurate and early detection of BMHV dysfunction, combined with medical imaging diagnostic techniques by using CFD models. [3, 5, 6]

MODELS AND METHODS

Advanced and reliable CFD software (FLUENT) was used in the current study to investigate the blood flow through a normal and dysfunctional bileaflet mechanical heart valve (BMHV) based on a 27mm St. Jude Medical BMHV with six different 2D models at different flowrate conditions. Flow dynamics were described through BMHV in two scenarios:

1. Healthy Valve in the fully opened position with different cases of SAS severity
2. Concomitant presence of different cases of SAS severity with dysfunctional valve.

In both scenarios, we aim to find the effect of these two cases on Doppler parameters including peak velocity, ΔP, EOA, and DVI.

BMHV model was drawn using AutoCAD software (figure 1). Gambit 2.3.16 was used for constructing MHV geometry and mesh generation (with 188,000 elements) and Fluent 6.3.26 based on finite volume method was used to run the numerical simulations (figure 2). In the current study, the standard $k - w$ model was used with 10% turbulent intensity and 27 mm as inlet/outlet hydraulic diameter and the flow assumed to be steady, turbulent, incompressible, and Newtonian

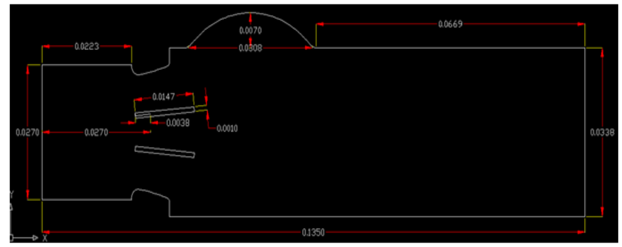

Figure 1: The bileaflet mechanical heart valve model.

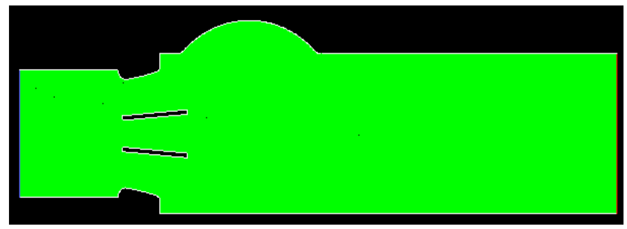
Figure 2: The meshed model with 188,000 elements.

Zero static pressure outlet condition was used as the outlet boundary condition, and no slip condition was used at all walls. Velocity Inlet condition is used as an inlet boundary condition, where all values of velocity inlet specified in table (1) to represent low, normal, and high conditions of flow rate.

$$Q = AV$$

Where: Q is flow rate (m^3/s).
A is sectional area of inlet (m^2).
V is inlet velocity (m/s).

The value blood density was 1060 (kg/m^3), and the value of dynamic viscosity was 0.0035 kg/ms where the Reynolds number ranged from 4088.6 to 17353.7 that represents the turbulent nature of the flow downstream of the valve.

Table (1): Velocity inlet conditions and Reynolds Number

Cases	Inlet velocity (m/s)	Flowrate (m^3/s) $*10^{-4}$
Normal	0.5	2.86
	1	5.723
	1.5	8.584
25 % SAS	0.67	2.86
	1.33	5.723
	2	8.584
50 % SAS	1	2.86
	2	5.723
	3	8.584

RESULTS

Velocity Distribution and Profiles

Figures (3) and (4) show the velocity contours for the entire field for different percentages of SAS at normal flow rate. Due to the difference in SAS severity with concomitant dysfunctional valve, the flow patterns developed upstream and downstream of the valves were different from normal ones. The maximum velocity increased from 1.5 m/s in the normal case (0% SAS) to 1.8 m/s in 25% SAS. When SAS increases from 25% to 50 %, maximum velocity increased to 2.32 m/s. The flow passes through the three orifices (one central and two laterals) in the healthy model where these directions in dysfunctional valve shifted up to mainly the upper lateral orifice when lower leaflet is closed and velocity values are approximately doubled.

In dysfunctional valve (0% SAS), maximum velocity doubled from 1.5 in normal one to 3 m/s where this value increased to 3.6 and 4.45 m/s for 25% and 50% SAS respectively which are approximately doubled from 1.8 and 2.32 m/s in normal valve with 25% and 50% SAS.

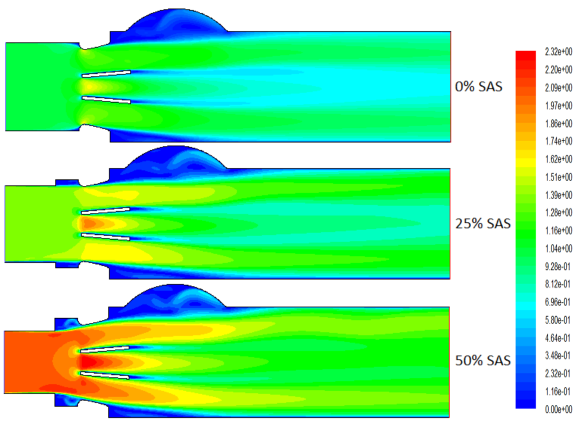

Figure 3: Contours of velocity magnitude (m/s) for normal BMHV with different cases of SAS severity at normal flowrate (Q) =5.723*10-4 (m3/s).

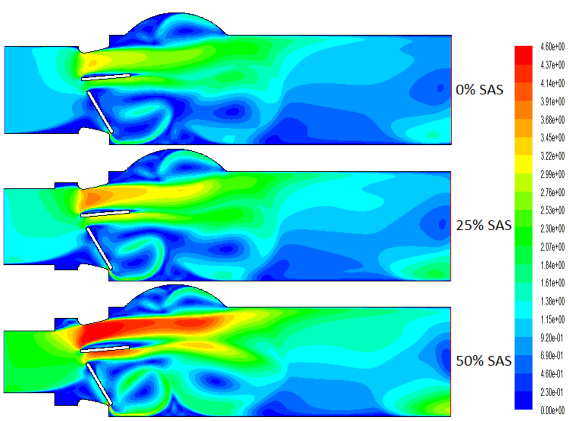

Figure 4: Contours of velocity magnitude (m/s) for Dysfunctional BMHV with different cases of SAS severity at normal flowrate (Q) =5.723*10-4 (m3/s).

Figures (5) and (6) show the velocity profiles at at leaflt trailing edges and 1D, 2D, 3D downstream of the valve for normal and dysfunctional valve with different cases of SAS severity with normal flow rate of $Q = 5.723*10^{-4}$ (m^3/s). In healthy valve with the presence of SAS, the velocity profiles have the same shape (three different jets with maximum velocity in the central orifice). Only the magnitude of the velocity was changed (i.e. the higher the percentage of SAS the higher the velocity magnitude). However, In cases of concomitant SAS and dysfunctional BMHV, the flow is disturbed downstream as well as upstream from the valve. The flow direction in dysfunctional valve shifted up to mainly the upper lateral orifice with maximum velocity especially when lower leaflet is completely closed. Velocity profiles tend to be flattened downstream of the BMHV, as seen in 2D and 3D velocity profiles comparing to 1D.

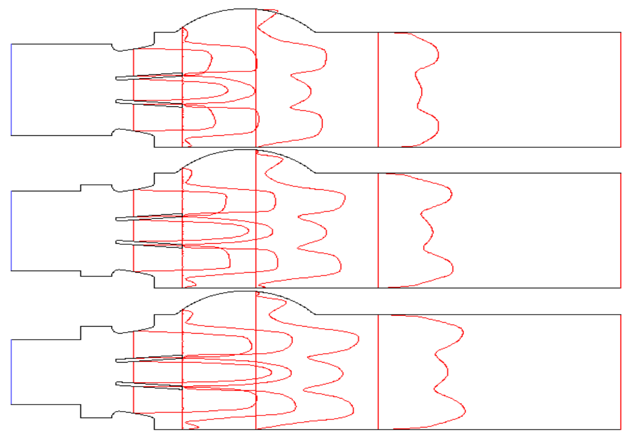
Figure 5: The velocity profiles at 1D, 2D, 3D and leaflets edges downstream of normal BMHV with different cases of SAS severity.

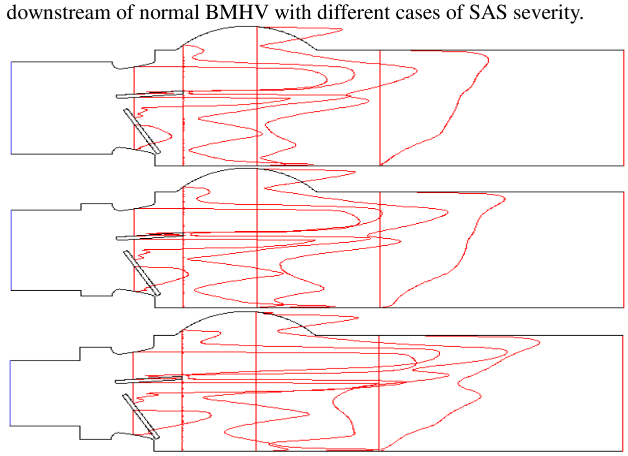
Figure 6: The velocity profiles at 1D, 2D, 3D and leaflets edges downstream of dysfunctional BMHV with different cases of SAS severity.

Clinical Aspects (Doppler Parameters)

1- Doppler Velocity Index (DVI) is the ratio of the peak velocity of the left ventricle outflow tract (LVOT) to the transvalve peak velocity**.**

$$\text{Doppler Velocity Index (DVI)} = \frac{V_{in}}{V_{max}}$$

Where: V_{in}= inlet velocity, V_{max}= maximum velocity within BMHV.

Effective Orifice Area (EOA) is the minimum cross sectional area (called vena contracta) of flow jet when maximum velocity is reached downstream of the valve [5]. EOA gives an indication of obstruction degree to blood flow in prosthetic valve so a large EOA gives small pressure drop and small energy loss. The EOA of (3 to 4 cm^2) considered as normally [5].

2- Effective Orifice Area (EOA) $= \frac{Q_{inlet}}{V_{max}}$ (m^2).

Where: Q_{inlet} = inlet flowrate = V*A (m^3/s), V_{max} = maximum transvavular velocity (m/s).

3- Pressure Gradient (ΔP) was estimated from the simplified Bernoulli equation by using the maximum transvalvualr velocity (V_{max}) where $\Delta P = 4V^2_{max}$ (mm Hg).

Figure 7 shows the relationship between DVI and flowrate for normal and dysfunction BMHV with different cases of SAS severity. The results show that the DVI is flowrate independent parameter. Moreover, DVI is proportional to the severity of SAS and inversely proportional to dysfunction severity. It is worth to mention that the concomitant SAS with dysfunctional BMHV, DVI was reduced to half of its value compared to healthy BMHV with only SAS.

Figure 8 shows the relationship between EOA and flowrate for normal and dysfunction BMHV with different cases of SAS severity. EOA is flowrate independent and inversely proportional to SAS severity as well as valve dysfunction severity, whereas in the concomitant SAS with dysfunctional BMHV, EOA was reduced to half of its value compared to healthy valve with only SAS.

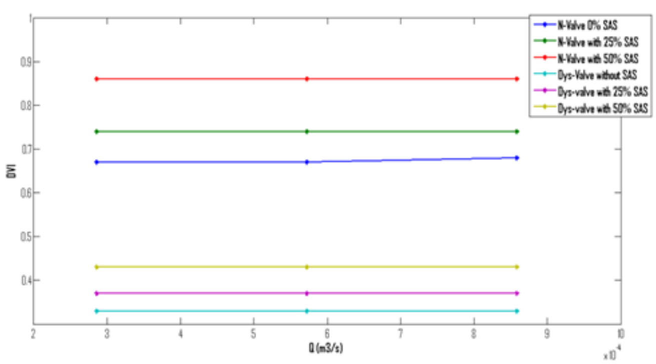

Figure 7: The relationship between DVI and flowrate for normal and dysfunction BMHV with different cases of SAS severity.

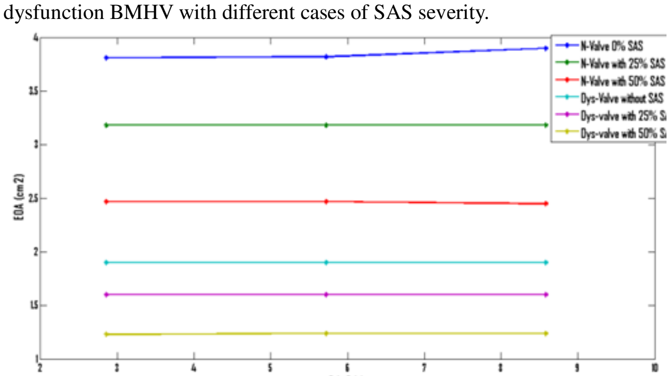

Figure 8: The relationship between EOA and flowrate for normal and dysfunction BMHV with different cases of SAS severity.

DISCUSSION

Doppler Velocity Index an Effective Orifice Area

EOA is flow rate independent and decrease as SAS percentage increase, whereas in the concomitant SAS with dysfunctional leaflet, EOA is reduced to half of its value in healthy valve with only SAS so that EOA does not give the real cross sectional area of the flow jet due to SAS leading to mis-detection of actual valve hemodynamic function. However, DVI is flowrate independent and increase as SAS percentage increases in both healthy and dysfunctional BMHVs valve with SAS. Therefore, the presence of SAS might mask the dysfunction of BMHV by increasing the DVI value.

CONCLUSIONS

The current results show that SAS has a significant impact on Doppler parameters where the Doppler parameters in case of concomitant SAS and dysfunctional leaflet do not reflect actual condition of BMHV and thus the current cutoff values for Doppler parameters should be modified. Also, LVOT size should be considered during BMHV diagnosis. From clinical point of view, with the presences of SAS, EOA was relatively the most accurate parameter. However, BMHV dysfunction is masked due to DVI overestimation and therefore, DVI showed less sensitivity compared to EOA.

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