A Multi-physics model of the ventricular valve-valve interaction

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Introduction

The importance of the interaction between the aortic and the mitral valve for the pumping efficiency of the heart has already been proved by Stijnen et al (2009). Valve functionality was found to depend on the flow conditions and the authors have emphasized the importance of future research, based on more geometrically realistic representations of heart valve – ventricle configurations. The purpose of our work was to develop a full 3D CFD-FSI analysis of ventricular valve-valve interaction and to contribute to the understanding of how this interaction is affected by the contractility of the left ventricle and by the systemic arterial tree characteristics (the afterload).

Materials and Methods

The interaction between the aortic (Ao) and mitral (Mi) valve has been studied in the 3D geometry shown in Fig.1. The model consists of a constant volume cylindrical chamber (D_{LV}=80mm, H_{LV}=100mm) with two cylindrical channels holding the two valves. The geometry of each valve was obtained by scanning a 24 mm TAD diameter bileaflet explanted valve (D_c=24mm, H_v=8.5mm). The distance between the two channels is d_v=14mm, and the total length of each channel is $H_C+H_t+H_v$=40mm.
The 3D geometry is connected to a time-varying compliance (1),

$$C_{LV}(t)=\begin{cases}\left\{E_m+\dfrac{E_M-E_m}{2}\left[1-\cos\left(\dfrac{\pi t}{T_S}\right)\right]\right\}^{-1}, & 0\le t\le T_S\\[2ex] \left\{E_m+\dfrac{E_M-E_m}{2}\left[1+\cos\left(\dfrac{\pi}{T_R}(t-T_S)\right)\right]\right\}^{-1}, & T_S\le t\le T_S+T_R\\[2ex] E_m^{-1}, & T_S+T_R\le t<T\end{cases} \tag{1}$$

and to a lumped parameter model representing the arterial system (the afterload). The inlet channel of the model is connected to a constant pressure source (P_{LA}=5mmHg) representing the atrial pressure. The afterload parameters were set to the values given by Westerhof and Noordegraaf (1969), whereas the maximum and minimum ventricular elastance are E_m =67e5Pa/m^3 and E_M=25e7Pa/m^3 respectively. The duration of the simulated cardiac cycle is T_{cc}=0.8s while T_S=T_{cc}/6 and T_R=Tcc/6. The Panel 1 in Fig. 2 shows a plot of the left ventricle elastance defining the values of T_S (Systolic Time) and T_R (Return Time).

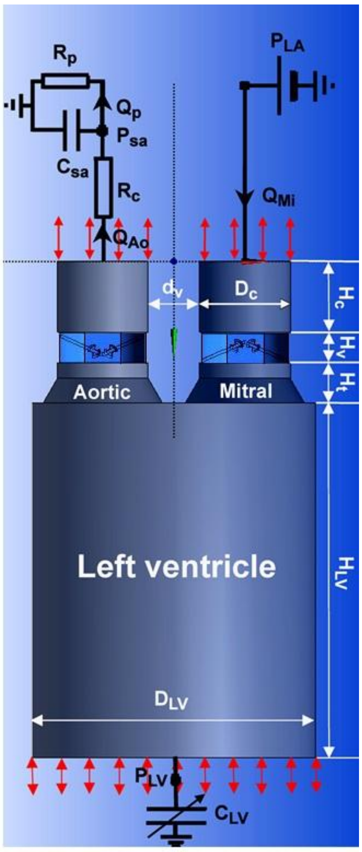


Figure1. 3D representation of the left ventricle and the two valves, coupled to the electrical analogue of the ventricle and the afterload

For the FSI part of the model, the rigid body module available under CFX v13 was used. A rigid body is a solid object that moves through a fluid without itself deforming (one way FSI). Its motion is dictated by the fluid forces and torques acting upon it, plus any external forces such as gravity and buoyancy. A single degree of freedom (rotation around a predefined axis) was considered for each leaflet.

Results and Discussion

The model was initialized at a state corresponding to the beginning of the isovolumetric contraction, with both valves closed and 80 mmHg applied at the outlet (Ao) channel and 5 mmHg at the inlet (Mi) channel. The left ventricle pressure was also initialized to 5 mmHg.

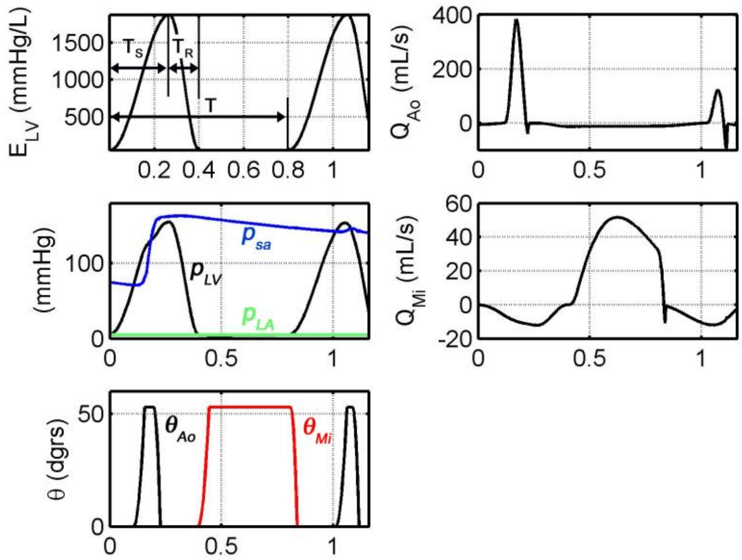
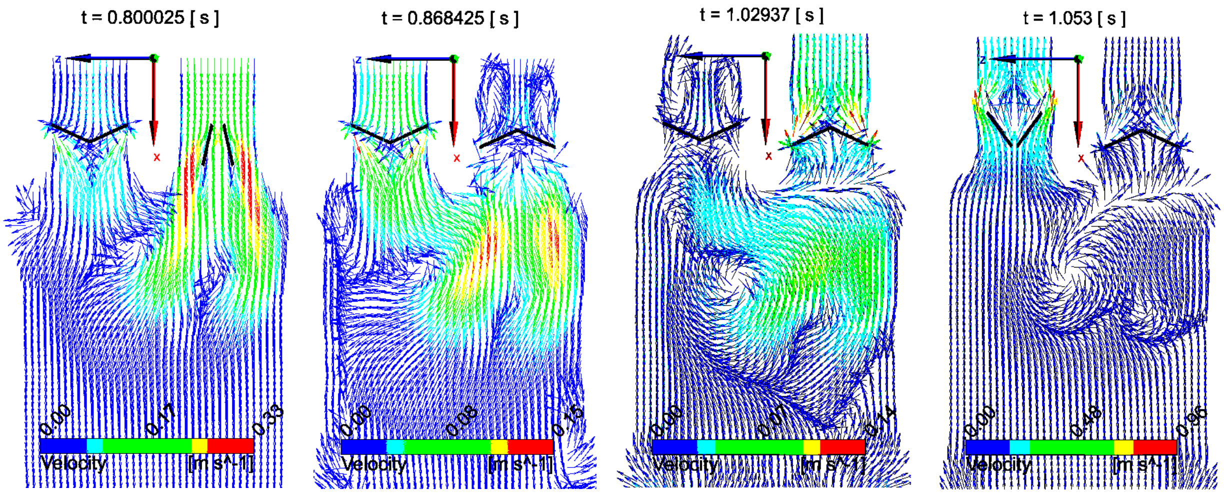


Figure 3. Left ventricle elastance; Ao flow; atrial, ventricular and systemic arteries pressures; Mi flow; position histories of the valves during the first 1.5 cardiac cycles.

After running the model at different values of the afterload parameters, it has been observed that the arterial compliance C_{sa} is decisive for the dynamics of the aortic valve and for the pumping efficiency of the heart. Rigid arteries make the aortic valve to flutter leading to multiple openings and closures, whereas, compliant arteries stabilize the valve. The results plotted in figure 2 correspond to a value of C_{sa}=30e-9 m^3/Pa which is approximately two times greater than the normal value reported by Westerhof [4]. This compliant load allowed the aortic valve to open and close freely.



a) b) c) d)

Figure 3. Fluid velocities at different moments during the second cardiac cycle: a) end diastole, before the Mi valve closure, b) at the end of the Mi valve closure, c) at the end of isovolumetric contraction, d) at peak-systole

Although, this is something that doesn't occur in vivo. We believe that the fixed-volume ventricle used in our model could be a cause of the aortic valve fluttering and the only way to compensate that was to make the arteries more compliant.

After the second Cardiac cycle has been simulated, a different behavior of the coupled model has been noticed compared to the first cardiac cycle. The aortic forward flow is four times lower, whereas, the regurgitant aortic flow is three times greater at the second systole compared to the first one. That can be attributed to the incorrect initialization of the end diastolic volume and/or pressure of the left ventricle at the beginning of the first cardiac cycle.

While waiting for a better initialization of the model or for its steady state solution, we can have a look at the fluid velocity distribution in the symmetry plane of the ventricle. Figure 3 shows a few snapshots of the fluid velocity in the ventricle at different moments, indicating the formation of two jets downstream the mitral valve and two large vortical structures upstream the aortic valve. Smaller vortical structures are present at the other side of each valve, that is, on the atrial side of the mitral valve (fig. 3.b) and in the aorta (fig. 3.c).

Conclusion and perspectives

After simulating the first cardiac cycle completely and the second systole, we realized that a large number of full cardiac cycles will have to be simulated in order to get to the steady state solution. Simulating around 20 cardiac cycles would consume too much time without automating the computation process. That is why; we are currently looking at implementing an automatic remeshing algorithm. Another possibility to improve the efficiency of our model is finding a better integration scheme for the lumped parameter model (currently the Euler method) that would allow increasing the simulation time step (currently 7.5e-5s). Both the automatic remeshing and an adaptive time stepping would be extremely useful for the development of the next phase of our model implementation which will consist of considering a variable volume ventricle. Either a shape parameterization of the left heart geometry (Evans 2001) or an experimental-based approach (Stijnen, 2001) is foreseen. Anyway, before going into any automation of the simulation, we believe that the implementation of a time-varying left ventricle model would be much more effective.

Acknowledgements

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References

1. Stijnen, J.M.A., Bogaerds, A.C.B., de Hart J., Bovendeerd, P.H.M., de Mol, B.A.J.M., & van de Vosse, F.N. 2009 Computational analysis of ventricular valve-valve interaction: Influence of flow conditions. *International Journal of Computational Fluid Dynamics 2009; 23/8*, 609-622. (doi:10.1080/10618560903092035)

2. Stijnen, J.M.A., Influence of prosthetic mitral valve orientation on ventricular flow. Computers in Cardiology 2001; 28:173-175

3. van 't Veera M., van Stratenb B., van de Vossea F., Pijlsa N., Influence of orientation of bi-leaflet valve prostheses on coronary perfusion pressure in humans. *Interactive CardioVascular and Thoracic Surgery 6 (2007) 588–592*

4. Westerhof N., Noordegraaf A., Reduced models of the systemic arteries, *Proc. 8th Intl. Conf. Med. Biol. Eng.: Chicago; 1969*

5. Evans C.J., Bloor M.I.G., Wilson M.J., Shape parameterization of the time-dependent geometry of the heart for steady fluid dynamical analysis. *Journal of Theor. Medicine (2001); 3, 221-230*