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AN EFFICIENT VALVE MODEL BASED ON RESISTIVE IMMERSED SURFACES ENHANCED WITH PHYSIOLOGICAL DATA

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SUMMARY

In order to reduce the complexity of heart hemodynamics simulations, one-way coupling approaches are often considered as an alternative to fluid-structure interaction (FSI) models. A possible shortcoming of these simplified approaches is the difficulty to correctly capture the pressure dynamics during the isovolumetric phases. In this work, we propose an enhanced resistive immersed surface (RIS) model of cardiac valves which overcomes this issue. The benefits of the model are investigated and tested in blood flow simulations of the left heart.

Key words: *Heart hemodynamics, Resistive immersed surfaces, One-way coupling.*

1 INTRODUCTION

Despite the major progress achieved during the last fifteen years (see, e.g., [1, 2]), the full simulation of heart hemodynamics with FSI remains a very challenging problem in scientific computing. Among the main fundamental difficulties, we can mention the large deflections of interfaces, the topology changes induced by contacting leaflets and the subsequent high pressure-drops. In order to mitigate the complexity of the problem, simplified models have recently been proposed (see [3, 4, 5, 6]). Basically, these approaches combine a simplified modeling of valves dynamics with an *one-way* kinematic coupling of the heart hemodynamics with the myocardium mechanics. This means that synthetic or measured displacements are imposed on the boundaries of the fluid cavities.

The reduced complexity of replacing a full FSI model in the heart with a *one-way* coupling approach comes however at a price. Indeed, since the dynamical aspects of the coupling are neglected, pressure within the ventricle is not correctly determined during the isovolumetric phases. In this work, we propose an approach which overcomes this issue. The main idea consists in enhancing a resistive immersed surface (RIS) model for valves dynamics [6, 7] with pressure data coming from measurements or from mechanical simulations. Thus, with this improvement, both kinematic and dynamic aspects of the coupling between fluid and structure are taken into account allowing the correct definition of the pressure when both valves are closed.

2 METHODOLOGY

For the sake of simplicity, we consider only the two fluid cavities – left ventricle and atrium – of the left heart. The corresponding fluid domain is denoted by $\Omega(t)$. We assume that its boundary $\partial\Omega(t)$ is partitioned as $\partial\Omega(t) = \Gamma_{\text{wall}}(t) \cup \Gamma_{\text{ext}}(t)$, where $\Gamma_{\text{wall}}(t)$ corresponds to the part where the motion is prescribed (i.e., the internal walls of the ventricle and the atrium) and $\Gamma_{\text{ext}}(t)$ to the external boundaries (i.e., the aorta and pulmonary veins) where a given pressure p_{ext} is enforced. We denote by $\boldsymbol{n}$ the outward unit normal to $\partial\Omega(t)$. The closed configurations of the aortic and mitral valve are given in terms of an oriented surface denoted $\Sigma(t)$, immersed in $\Omega(t)$, and with an unit normal $\boldsymbol{n}_\Sigma$ (pointing outwards the left ventricle).

The opening and closing dynamics of the aortic and mitral valves are described by a simplified immersed resistive surface model [6, 7] in which the mechanics of the leaflets are neglected. We assume that the pressure within the left ventricle $p_{\rm LV}$ is known (e.g., via measurements or simulations) and that both $p_{\rm ext}$ and $p_{\rm LV}$ are homogeneous in space. We introduce the notation $\delta P = p_{\rm ext} - p_{\rm LV}$. The dynamics of the blood velocity $\boldsymbol{u}$ and pressure p in $\Omega(t)$ are described by the following system involving the Navier-Stokes equations in ALE formalism where $\cdot|_{\mathcal{A}}$ is the ALE time derivative and $\boldsymbol{w}$ is the computational mesh velocity:

$$
\begin{aligned}
\rho\left(\left.\frac{\partial \boldsymbol{u}}{\partial t}\right|_{\mathcal{A}} + (\boldsymbol{u}-\boldsymbol{w})\cdot\nabla\boldsymbol{u}\right) - \nabla\cdot\boldsymbol{\sigma}(\boldsymbol{u},p) + \big(R(t)(\boldsymbol{u}-\boldsymbol{w}) + \delta P\boldsymbol{n}_{\Sigma(t)}\big)\,\delta_{\Sigma(t)} = 0 \quad &\text{in} \quad \Omega(t),\\
\nabla\cdot\boldsymbol{u} = 0 \quad &\text{in} \quad \Omega(t),\\
\boldsymbol{u} = \boldsymbol{w} \quad &\text{on} \quad \Gamma_{\rm wall}(t),\\
\sigma(\boldsymbol{u},p)\boldsymbol{n} = -p_{\rm ext}\boldsymbol{n} \quad &\text{on} \quad \Gamma_{\rm ext}(t).
\end{aligned}
\tag{1}
$$

Here, the symbol $\delta_{\Sigma(t)}$ denotes Dirac's measure on $\Sigma(t)$ and $R(t)$ is a time-dependent function, with values in $[0,R_{\rm max}]$, which describes the opening and closing dynamics of the valves (see [6, 7]). Note that $(1)_1$ enforces the following interface condition across $\Sigma(t)$:

$$
[\![\boldsymbol{\sigma}(\boldsymbol{u},p)\boldsymbol{n}_{\Sigma(t)}]\!] + R\,(\boldsymbol{u}-\boldsymbol{w}) = -\delta P\boldsymbol{n}_{\Sigma(t)} \quad \text{on} \quad \Sigma(t). \tag{2}
$$

where the symbol denotes the $[\![\cdot]\!]$ jump across $\Sigma(t)$. Using the incompressibility condition $(1)_2$, we can show that in the case of both valves are closed (i.e., $R(t) = R_{\rm max}$), the relation (2) yields $R_{\rm max}^{-1}(p - p_{\rm ext}) = -R_{\rm max}^{-1}\delta P$ in the left ventricle, so that p is equal to $p_{\rm LV}$ as wished. The case $\delta P = 0$, i.e., without any interface pressure correction, corresponds to the original immersed resistive surface model [6, 7], which enforces $p = p_{\rm ext}$ during the isovolumetric phases.

3 RESULTS

The motion of the endocardium is prescribed using a displacement field obtained from an electro-mechanical model of the heart [8]. This displacement is extended to the inside of the ventricle using an appropriate non-linear lifting operator [9]. The aortic valve model comes from a computerized tomography (CT) scan and the mitral valve model has been designed with the software 3-matic from physiological *in vivo* data [10]. Finally, the physiological ventricular pressure $p_{\rm LV}$ used for the correction in our tests comes from the above mentioned electro-mechanical simulations [8].

In order to illustrate the difficulties mentioned in the introduction, we first present the results obtained without any correction in the RIS model. In terms of blood velocity and vortices, we computed the velocity field for a typical cardiac cycle lasting 0.8 s. Its typical shape during the filling of the ventricle is depicted in Figure 1 (left). These results are relatively close to the behavior observed in *in vivo* experiments [4, 11]. Blood flow is oriented towards the anterior side of the ventricle surface inducing a local vortex near the apex before the opening of the aortic valve. These results are representative of other 3D simulations we ran. On the contrary, the pressure shows a non-physiological behavior during isovolumetric phases (i.e., when both valves are closed). *In vivo* studies [12] show that the ventricular pressure should decrease towards the atrium pressure immediately after the closing of the aortic valve. In the simulations, it remains equal to the aortic pressure for an additional period of around 0.20 s, as depicted in Figure 1 (right).

To highlight the benefits of the proposed correction, a comparison of the computed pressure with and without the new term is carried on with a simplified geometry of the heart. The setting of this toy model is presented in Figure 2 (left). The pressure is set to $100{,}000$ dyn/cm^2 at the outlet and to 0 dyn/cm^2 at the inlet. An arbitrary periodic displacement has been applied on the ventricle's boundaries Γ_2 to simulate the beating of the heart. Finally, the imposed pressure $p_{\rm LV}$ is a periodic function we chose – depicted in Figure 2 (right) – mimicking the global behavior of the ventricular pressure in real physiological cases. Each change of value of $p_{\rm LV}$ corresponds to the closing or opening of a valve. This is simultaneous with the change of value of the resistance of the valves as shown in Figure 2 (right).

The results obtained with the toy model are presented in Figure 3. In Figure 3 (left), no correction is applied. The computed pressure inside the ventricle is then not well-defined when both valves are closed and get an arbitrary intermediate value between atrium's pressure and aorta's pressure. In Figure 3 (right), the correction is applied on both closed valves. One can observe that the ventricular pressure is now correctly defined when both valves are closed and equal to p_{LV}, the chosen imposed pressure.

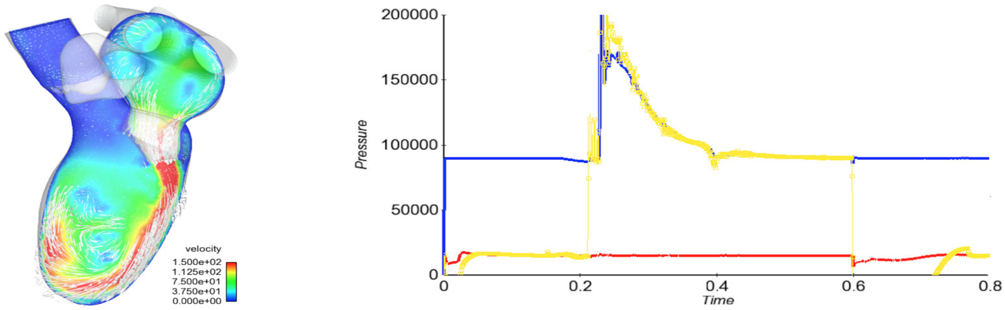

Figure 1: Typical computed results in the realistic full heart model. On the left: 2D cut of the velocity field in cm/s during ventricle's filling. On the right: volume-averaged pressure values in dyn/cm^2 with respect to the time in s during a cardiac cycle. The atrium is colored in red, the ventricle in yellow and the aorta in blue.

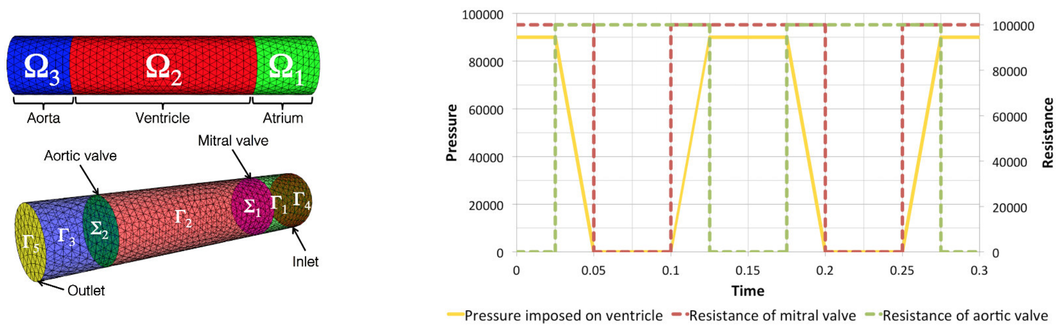

Figure 2: Definition of the toy model. On the left: definition of volumes (Ω_i), boundaries (Γ_i) and RIS (Σ_i). On the right: definition of the pressure imposed on the ventricle p_{LV} in dyn/cm^2 and of the resistance of each valve with respect to the time in s.

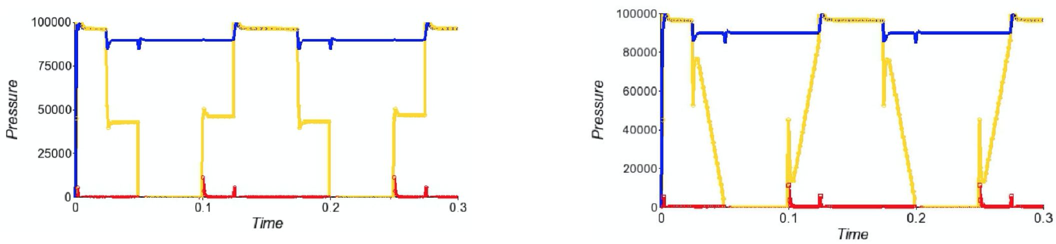

Figure 3: Comparison of volume-averaged pressure values in dyn/cm^2 with respect to the time in s for the toy model. On the left: results computed without corrective term. On the right: results computed with corrective term. The atrium is colored in red, the ventricle in yellow and the aorta in blue.

4 CONCLUSION

A new reduced model for heart valves has been presented to correct the problem of definition of the ventricular pressure during isovolumetric phases. It consists in enhancing the original RIS model with an additional term involving a priori data on the ventricle pressure during the isovolumetric phases. Several tests have been performed to illustrate its benefits: a simulation on a full heart model without any correction to highlight the problem and a comparison of the computed pressure on a simplified geometry with and without the correction.

The proposed approach offers a good compromise between complex fully coupled fluid-structure simulations and physiologically inaccurate one-way coupling simulations. The next step will be the integration of this strategy into the left heart model in order to correctly simulate the opening and closing of the valves as well as the correct ventricular pressure during the whole cardiac cycle.

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REFERENCES

[1] D.M. McQueen and C.S. Peskin. *Heart Simulation by an Immersed Boundary Method with Formal Second-order Accuracy and Reduced Numerical Viscosity*. In Mechanics for a New Millennium, Aref H, Phillips JW (eds). Springer Netherlands: Heidelberg, 2001.

[2] R. van Loon, P.D. Anderson, J. De Hart and F.P.T. Baaijens. A Combined Fictitious Domain/Adaptative Meshing Method for Fluid–Structure Interaction in Heart Valves. *International Journal for Numerical Methods in Fluids*, 46:533–544, 2004.

[3] M. Astorino. Fluid-Structure Interaction in the Cardiovascular System. Numerical Analysis and Simulation. *PhD Thesis*, UPMC, 2010.

[4] C. Chnafa, S. Mendez and F. Nicoud. *Image-Based Simulations show Important Flow Fluctuations in a Normal Left Ventricle: What Could be the Implications?*. Ann. Biomed. Eng., 44(11):3346–3358, 2016.

[5] A. Tagliabue, L. Dedè and A. Quarteroni. Fluid dynamics of an idealized left ventricle: the extended Nitsche's method for the treatment of heart valves as mixed time varying boundary conditions. *M*OX-Report, 61, 2015.

[6] M. Astorino, J. Hamers, S.C. Shadden and J.-F. Gerbeau. A Robust and Efficient Valve Model based on Resistive Immersed Surfaces. *International Journal for Numerical Methods in Biomedical Engineering*, 28:937–959, 2012.

[7] M.A. Fernández, J.-F. Gerbeau and V. Martin. Numerical Simulation on Blood Flows through a Porous Interface. *Mathematical Modelling and Numerical Analysis*, 42:961–990, 2008.

[8] D. Chapelle, P. Le Tallec, P. Moireau and M. Sorine. Energy-Preserving Muscle Tissue Model: Formulation and Compatible Discretizations. *International Journal for Multiscale Computational Engineering*, 10(2):189–211, 2012.

[9] M. Landajuela, M. Vidrascu, D. Chapelle, M.A. Fernández. Coupling schemes for the FSI forward prediction challenge: comparative study and validation. *International Journal for Numerical Methods in Biomedical Engineering*, 2016, DOI: 10.1002/cnm.2813.

[10] N. Ranganathan, J.H.C. Lam, E.D. Wigle and M.D. Silver. Morphology of the Human Mitral Valve: II. The Valve Leaflets. *Circulation*, 41(3):459–467, 1970.

[11] F. Domenichini, G. Querzoli, A. Cenedese and G. Pedrizzetti. Combined Experimental and Numerical Analysis of the Flow Structure into the Left Ventricle. *Journal of Biomechanics*, 40:1988–1994, 2007.

[12] L. Formaggia, A. Quarteroni and A. Veneziani. *Cardiovascular Mathematics: Modeling and Simulation of the Circulatory Cystem*. Springer-Verlag, 2009.