A 3D-1D cardiac-vascular computational feedbacked model

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Abstract- *A first version of a cardiovascular coupled model is presented. The modelling tools are Alya, the BSC tool for biomechanical simulations; and ADAN55, the LNCC model of the arterial blood flow. The former solves the heartbeat by Finite Element Method (FEM). The latter is a 1D model of the arterial blood flow in a 55-branched geometry. Previous generations of the involved models had fixed boundary conditions. With this novel tool a feedbacked cardiac-vascular model is obtained, offering more physiological boundary conditions and providing further insight in the cardiac-systemic interactions.*

I. INTRODUCTION

The used models were BSCs Alya, and LNCCs ADAN55. Alya is a 3D, multiscale, multiphysics, HPC code, that allows modelling and solving the heartbeat electromechanic problem using the Finite Element Method (FEM). The ADAN55 is a reduced 55-artery version of the Anatomically Detailed Arterial Network (ADAN) model, which takes into account more than 1500 arteries from an average human body, featuring a physiologically consistent systemic impedance at the aortic root, among other characteristics. Both codes are coupled by a black-box decomposition approach previously proposed in [7]. At each time step the Fluid Structure Interaction (FSI) problem is solved in the ventricle, computing the output flow through the aorta which gives a pressure response back. In previous generations of the models, each one had predefined boundary conditions: for Alya a pressure value was imposed on the endocardium, and an experimentally measured flow contour was imposed in the aortic root of the ADAN55 model. With this novel coupling a more physiological response to the blood ejection is obtained, as the systemic impedance is a complex result of the entire 1D arterial model which changes with the flow generated by the heart model. In the same way, using the heart model output flow connected to the ADAN55 input, offers the possibility to obtain a physiological and feedbacked stimuli that varies with changes in the arterial system.

II. METHODOLOGY

A. Governing equations

The cardiovascular system can be decomposed as a coupled electromechanical system: the propagation of the action potential induces the mechanical deformation of the solid (myocardium), which reduces the inner cavity inducing the pumping action against the fluid (blood) through the arteries. In this way, three problems have to be solved: the propagation of the electric potential, the deformation of the solid due to the electrophysiologic stimuli and outer forces, and the fluid dynamics inside the ventricle and the arteries.

The governing equations for the electrophisology potential model are described in [1]:

$$C_m \frac{\partial \phi}{\partial t} = \frac{\partial}{\partial x_i}\left(\frac{D_{ij}}{S_v}\frac{\partial \phi}{\partial x_j}\right) + I_{ion} \quad (1)$$

where D_{ij} is the diffusion tensor, C_m and S_v are the membrane capacitance, and the surface-to-volume ratio respectively. The expression of I_{ion} is what determines the complexity of the model. In this case we use the Fitzhug-Nagumo model [2] due to its simpleness. The mechanical deformation governing equations are described in [3]. In the expression of the momentum balance of the solids:

$$\rho_o \frac{\partial^2 u_i}{\partial^2 t} = \frac{\partial P_{iJ}}{\partial X_J} + \rho B_i \quad (2)$$

where ρ is the initial density of the body, B_i are the body forces and P_{iJ} the first Piola-Kirchoff stress tensor. A modified version of the Holpzafel and Odgen [4] model is used for the energy function which defines the constitutive model. The coupling between the electrophysiology and the solid model is done through the Hunter and Nash model [5].

Inside the cavity, the fluid is modeled by the incompressible Navier-Stokes, as explained in [6].

The governing equations in the arterial network, and the way they are solved, are described in [7]. The condensed 1D Navier-Stokes equations in compliant vessels comprise momentum and mass conservation as:

$$\frac{\partial Q}{\partial t} + \frac{\partial}{\partial x}\left(\alpha_m \frac{Q^2}{A}\right) + \frac{A}{\rho}\frac{\partial P}{\partial x} + \frac{2\pi R}{\rho}\tau_o = 0 \quad (3)$$

$$\frac{\partial A}{\partial t} + \frac{\partial Q}{\partial x} = 0 \quad (4)$$

$$P = P_0 + \frac{Eh}{R_0}\left(\sqrt{\frac{A}{A_0}} - 1\right) + \frac{Kh}{R_0}\frac{1}{2\sqrt{A_0 A}}\frac{\partial A}{\partial t} \quad (5)$$

where A is the luminal area, R is the radius, Q is the flow rate, P is the mean pressure, ρ is the density, α_m is the momentum correction factor, τ_o accounts for the viscous effects, h is the wall thickness and E and K are the material parameters that characterize the elastic and viscoelastic material responses. (3) and (4) are the momentum and continuity balance, respectively. (5) has the FSI terms for the 1D formulation.

B. Numerical test and results

A ventricle is simulated using an ellipsoidal cavity made of active cardiac tissue, with fibers arranged vertically, with a short non-active tubular part playing the role of the aorta. The

activation potential is started at the bottom. The arterial network is also simplified, including 55 segments, called ADAN55. Figure 3 shows the flowrate and a scaled (1%) pressure in the coupling point. Figure 1 and 2 shows the ventricle-like geometry and part of the ADAN55 mesh.

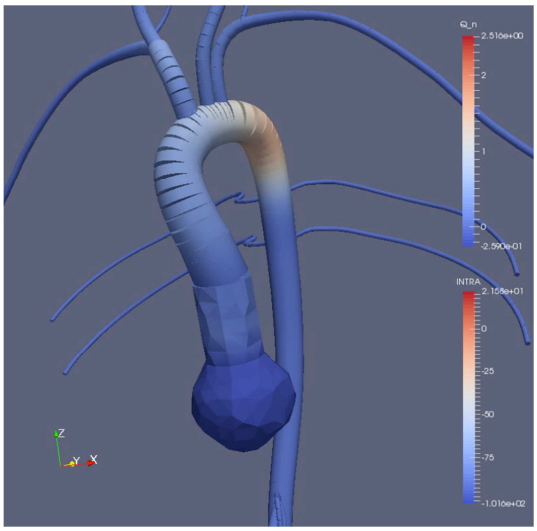

Fig.1 Voltage in the solid and flow in the arterial network.

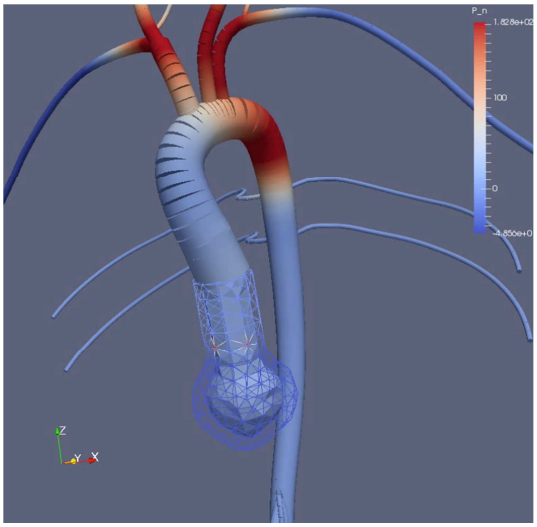

Fig 2.Pressure in the arterial netwrok.

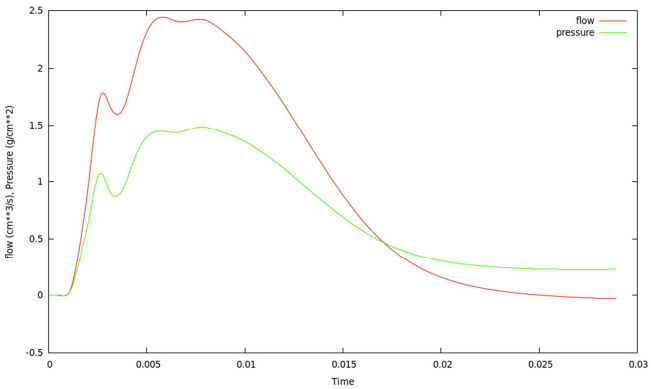

Figure3: Pressure (1% scaling) and flow in the coupling point.

III. CONCLUSIONS

In this work we show preliminary results of a coupled simulation tool that widens the possibilities in cardiovascular modeling. A complete simulator of the cardiovascular system will allow bioengineers and medical researchers to study the response of the full model to changes in any of the components, extending the predictive and descriptive capabilities of the standalone versions of the models, and providing further insight in the cardiac-systemic interactions.

ACKNOWLEDGMENT

The Ph.D. studies of A. S. are partially founded by the EU Brazil Cloud Connect Project, funded by the European Commission under the Cooperation Programme, Framework Programme Seven (FP7).

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