

An integrated multiscale CFD model of the human heart

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Abstract: We introduce a CFD model for the numerical simulation of the heart hemodynamics in both physiological and pathological conditions, by accounting for all the physical processes that influence cardiac flows: moving domain and interaction with electromechanics, transitional-turbulent flows, cardiac valves and coupling with the external circulation. To impose a physiological displacement of the domain boundary, we employ a 3D ventricular electromechanical model coupled to a lumped-parameter (0D) closed-loop model of the circulation and the remaining cardiac chambers. To extend the ventricular motion to the endocardium of the remaining heart, we introduce a novel preprocessing procedure that combines an harmonic extension of the electromechanical displacement with the motion of the atria based on the 0D model. We thus obtain a one-way coupled electromechanics-fluid dynamics model in the ventricle(s). To better match the 3D CFD with blood circulation, we also couple the 3D CFD model to the 0D circulation model. We obtain a multiscale coupled 3D-0D fluid dynamics model that we solve via a segregated numerical scheme. We carry out numerical simulations for a healthy heart and we validate our model by showing that significant hemodynamic indicators are correctly reproduced.

Keywords: Cardiac hemodynamics, cardiac valves, circulation, transitional flows, mitral valve regurgitation, computational fluid dynamics, multiscale models, finite element method.

1. INTRODUCTION

The study of cardiac blood flow aims at enhancing the knowledge of the heart physiology, assessing the pathological conditions and possibly improving the clinical treatment and therapeutics. In the clinical routine, blood flow analysis is conventionally based on imaging techniques. However, their space and time resolution is not accurate enough to capture small-scales features as recirculation regions, possible regions of transition to turbulence and the formation, interaction and dissipation of small coherent structures. Furthermore, imaging-based techniques cannot provide relevant fluid dynamics indicators such as the wall shear stress (WSS) which is correlated with the function and the remodeling of the heart chambers (Ngo et al. (2019)). In this respect, computer based numerical simulations – also known as *in silico* simulations – of the heart and circulation represent a valuable tool to quantitatively assess the cardiac function and to enhance the understanding of cardiovascular diseases.

The numerical simulations of cardiac blood flows should account for several aspects that characterize heart's hemodynamics (Chnafa et al. (2014)). The strong interaction between the fluid and the electromechanical activity of the heart yields a complex multiscale and multiphysics system involving the interaction of several physical pro-

cesses. Furthermore, the cardiac valves affect the blood motion and change the topology of the fluid domain during the heartbeat. In addition, a CFD simulation of the heart should also account for the transitional regime of the blood flow occurring in the heart chambers and, eventually, also for the strict influence between the local dynamics and the hemodynamics of the surrounding circulatory system. In this work, we introduce a CFD model of the heart accounting for all the aforementioned aspects.

2. MODELS AND METHODS

We model the blood flow in the heart chambers via the incompressible Navier-Stokes (NS) equations expressed in an Arbitrary Lagrangian Eulerian (ALE) framework to account for moving domains. To model the presence of valves in the fluid, we use the Resistive Immersed Implicit Surface (RIIS) method, in which the effect of the immersed surface in the blood is enforced via a penalization technique by introducing a resistive term in the momentum balance of the NS equations (Fedele et al. (2017)).

To prescribe a physiological displacement to the domain boundary, we employ the ventricular electromechanics (EM) model developed in Regazzoni et al. (2022); Pier-santi et al. (2022) coupled to the surrounding circulation described by a 0D lumped-parameter hemodynamic model. We introduce a novel procedure that combines an

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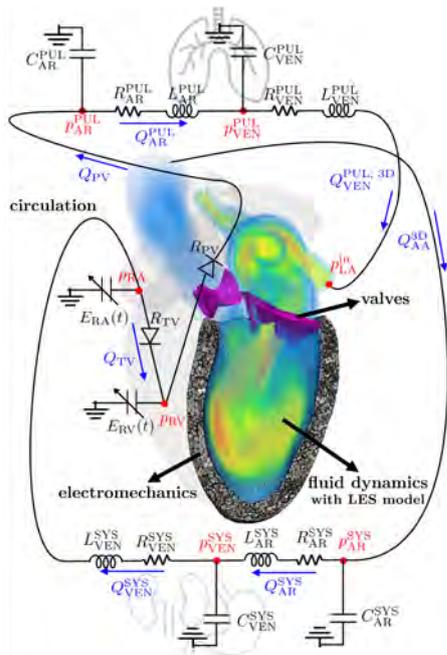


Fig. 1. The integrated multiscale CFD model of the heart. harmonic extension of the the ventricular displacement on the remaining heart chamber(s) based on Laplace-Beltrami equation, and a volume-based model of the atria tuned on the basis of the 0D circulation model (Zingaro et al. (2022)). This yields an integrated and multiphysics problem in which fluid dynamics is one-way coupled to EM in the ventricle(s). The model introduced can directly be applied also to the right heart and to the whole heart geometry.

To address the reciprocal influence between the hemodynamics of the heart chambers and the one of the surrounding cardiocirculatory system, we rely on the geometric multiscale modeling (Quarteroni et al. (2016)). We couple the 3D CFD model of the heart with the 0D lumped parameter model closed-loop model of the circulation introduced in Regazzoni et al. (2022). The interfaces conditions of the 3D-0D CFD model consists of the enforcement of the continuity of pressures and flowrates on the artificially chopped boundaries. In Figure 1 we represent the overall integrated and multiscale computational model.

We discretize the NS-ALE-RIIS equations in space via the finite element (FE) method and in time by means of backward differentiation formulas. We use a Variational Multiscale - Large Eddy Simulation method (Forti and Dede' (2015)) to get a stable formulation of the NS equations discretized by means of FE method; to stabilize the advection-dominated regime and to account for turbulence modeling within the framework of LES (Zingaro et al. (2021)). We discretize the system of ODEs of the 0D circulation model through a 4th order explicit Runge-Kutta scheme. We numerically solve the 3D-0D CFD model by means of a segregated numerical scheme (Zingaro et al. (2022)).

3. NUMERICAL RESULTS

We simulate the heart hemodynamics in physiological conditions and we show that several hemodynamic indi-

cators and flow patterns are correctly reproduced by the computational model when compared with in-vivo data. Furthermore, we simulate pathological scenarios as mitral valve regurgitation and we quantify clinical indicators to grade the severity of the pathology.

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