DIRECT NUMERICAL SIMULATION OF THE INTRA-VENTRICULAR FLOW USING PATIENT-SPECIFIC ANATOMY

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ABSTRACT

A novel computational framework for performing high resolution simulation of intraventricular flow in a patient-specific left heart is developed. The left heart anatomy is reconstructed from magnetic resonance imaging (MRI) data. We develop a physiologic cell activation model for the left ventricle kinematics and prescribe the resulting wall motion as the boundary condition for intraventricular flow simulation. The left ventricle is treated with sharp-interface immersed boundary to handle the large deformation of the wall motion. The simulations reveal the complexity of the flow structure during diastolic filling inside the LV chamber due to the formation and breakups of the vortex ring at the mitral orifice. The current framework demonstrates promising capabilities to evolve into a predictive tool for clinical uses.

INTRODUCTION

Understanding the intra-ventricular flow dynamics is critical for diagnosis and treatment of left heart dysfunction diseases [1, 2]. Recent in vivo measurements have shown the distinct pathological flow patterns from healthy ones [2, 3]. To understand the correlation between the flow pattern and the left heart dysfunction disease, high resolution data of intraventricular hemodynamic in patient-specific anatomy is needed. Although $in-vivo$ measurement techniques have been developed [4] to meet such needs, they currently do not provide sufficient spatial and temporal resolution to examine the complex flow field in region of interest [5]. This is especially true when medical devices have been implanted [6, 7]. The current study is aimed at providing the comprehensive flow structure inside the left ventricle and the descending aorta in both systolic and diastolic phase via high resolution numerical simulation.

The current trend in modeling left heart hemodynamics [1] is to utilize non-invasive imaging techniques such as Magnetic Resonance Imaging to reconstruct the patient-specific kinematics of the LV chamber directly from $in-vivo$ measurements [8]. The reconstruction is usually done via temporal interpolating between successive MRI images [1] or calibrating the tissue properties [9]. The reconstruction of LV kinematics is therefore an important task in order to achieve high accuracy of the computational model.

In this work, we report computational advances that have now made it possible to simulate at physiologic conditions the intraventricular hemodynamics with high numerical resolution in a patient-specific left heart model. We develop a kinematic model for a patient-specific left ventricle. We then prescribe the wall kinematics as boundary conditions for blood flow simulation inside the LV chamber. We resolve for the first time with high resolution, the rich hemodynamic environment inside an intact left ventricle.

METHODS

The left ventricular kinematics

The anatomical left ventricle was reconstructed from MRI scanned images of a healthy subject and was provided to us by the Cardiovascular Fluid Mechanics Laboratory of the Georgia Institute of Technology. The long axis length of the left ventricle is $H = 80mm$ The short axis diameter of the left ventricle is $D_L = 47mm$.

Our kinematics model of the LV chamber is based on the heuristic model of the left ventricular deformation [10]. We use the following assumptions:

1. Only the left ventricle moves. The atrium (base) and aorta are stationary in the cardiac cycle;
2. Only the endocardium surface is tracked. The myocardium and epicardium are not simulated. The wall thus has only one layer;
3. The endocardium surface is triangulated in cells. Each cell possesses its own fiber and response to the electrical excitation;
4. The material points on the endocardium move along spirally oriented fibers during shortening and twisting;
5. The response of the contractile fibers is assumed to be a function of the electrical excitation sent from the SA node.

The time-dependent electrical signal ($S(t)$) propagates on the endocardium surface [11] is modeled by a sine wave function with magnitude E, frequency f , phase lag ϕ in a heart beat cycle T :

$$S(t) = E \cdot \sin(2\pi f \frac{t}{T} - \phi) \tag{1}$$

We assume that each endocardium cell has its own magnitude E. The magnitude E is the function of an electrical time-dependent stimulus $E(p) = E(p(t))$. The response model is of FitzhHugh-Nagumo type:

$$p = p(t)$$
$$E(p) = c_1 \cdot p \cdot (p - c_0) \cdot (p-1) + c_2 \cdot (p-1) \cdot (1 - \exp(-c_3 \cdot p))$$

here c_0,c_1, c_2 and c_3 are parameters of the model.

The velocity magnitude of a material point on the endocardium surface is assumed to be prescribed by a polynomial equation:

$$|V| = \kappa \cdot S \cdot (\frac{2r}{D_L})^2 (\frac{l}{H})^{1.5} \tag{2}$$

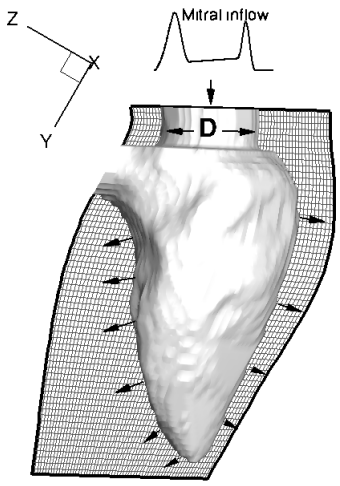
where κ is the parameter of the model, and r and l are the radial and longitudinal distance of the material point to the LV axis, respectively.

We assume that the fiber in each cell follows the analytical approximation in helix form. The fiber direction **b** is therefore the tangent of the helix. To account for the effect of multiple laminar layers of the heart wall [12], we also consider the radial contraction component s. The velocity direction is thus the combination of along fiber contraction **b** and radial contraction **s**. The velocity vector of an endocardium material point is thus:

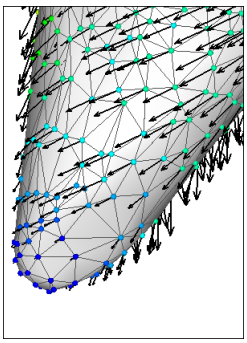
$$\mathbf{V} = |V|(\alpha \cdot \mathbf{b} + \beta \cdot \mathbf{s}) \tag{3}$$

where α and β are parameters of the model.

Herein we use the above model to prescribe the motion of the LV. It should be noted, however, that this model can also serve as a physiologic computational framework for integrating our model with kinematics data obtained from imaging modalities, such as MRI. In this work, we calibrate the controlling parameters (i.e α, β, c_0 etc.) to match the volume rate of change of the LV chamber with reported one in the literature [13].



(a) The computational setup



(b) The physiologic model

Figure 1: a)The computational grid for Computational Fluid Dynamics simulations is a structured grid with the thick outline is shown. At the mitral position, uniform flow is specified as boundary condition as the mitral valve is assumed to be fully open during diastole. b) The blood flow is driven by the LV wall motion resulted from the cell-activation model. The vectors depict the velocity field of the heart wall motion. The colors denote the velocity magnitude values. The aortic valve is fully close during diastole.

Numerical methods and computational setup

We treat blood as incompressible, Newtonian fluid whose motion is governed by the unsteady, three-dimensional Navier-Stokes equations. The numerical method is based on the curvilinear immersed boundary (CURVIB) flow solver we have developed in our group, which is capable of simulating flows with arbitrarily and moving boundaries [14] with non-linear Fluid-Structure Interaction [15]. The left ventricle is treated with immersed boundary method to handle the inherently large wall deformation. The governing equations are solved in a background mesh that contains the complex

Parameters	*Value*
HR	52 bpm
SR	40 %
ESV	65ml
EDV	118 ml
SV	53 ml
EF	45%
E-wave velocity	50 cm/s

Table 1: The global parameters of the left ventricle kinematics resulting from the proposed cell-activation based model. Acronym: HR - Heart Rate, SR - Systolic Rate, ESV - End Systolic Volume, EDV - End Diastolic Volume, SV - Stroke Volume, EF - Ejection Fraction. All quantities are well within the physiologic range.

geometry (see Figure 1) using the *CURVIB* method of [14]. Uniform pulsatile flow is specified at the mitral orifice using the physiologic waveform shown as seen in Figure 1. The aortic valve is assumed to be fully closed during diastole.

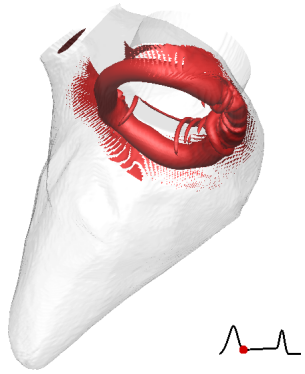
RESULTS

Our left ventricular model results in a physiologic kinematics indicated by several parameters shown in Table 1. The particular flow-rate curve is shown in the inset of Figure 2 over one cardiac cycle. Note that the flow rate at has two distinct positive peaks which are the E-wave and A-wave peak, respectively. The E-wave is the instance when the flow rapidly fills the left ventricle from the passive atrium as the mitral valves opens. The A-wave resulted from the active pumping of atrium to the left ventricle.

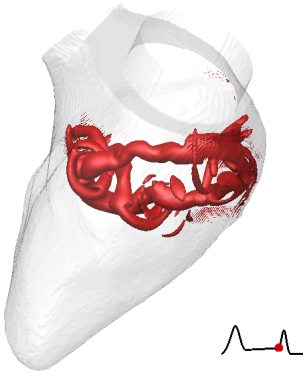
During the diastolic phase the intraventricular flow is dominated by the filling of blood flow from the left atrium into the left ventricular chamber. We observe the existence of a large inclined vortex formed inside the left ventricle chamber after the E-wave as seen in Figure 2a. Although in this case the flow is uniform at the mitral position, the asymmetry of basal ring's geometry creating an inclined ring instead of a perfect circular ring. After the E-wave peak the vortex ring is fully formed and propagates toward the apex. We also find the breakup of this vortex ring as it interacts with the septum wall right after the E-wave filling as shown in Figure 2b. The inclined ring breaks down and assimilates to a large clock-wise vortex at the center of the LV chamber at the end of diastasis as shown in Figure 2.

CONCLUSIONS AND FUTURE WORKS

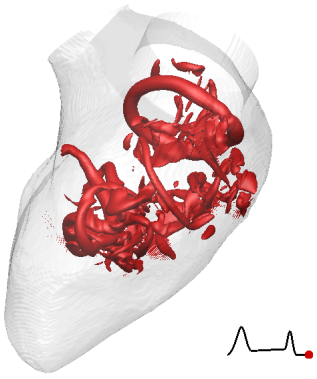
We have developed a novel computational framework capable of performing high resolution simulations intraventricular flow in a patient-specific left heart. The results elucidate the formation and subsequent breakdown of the mitral valve vortex ring during the LV filling process and underscore the



(a) E wave



(b) End of diastasis



(c) End of diastole

Figure 2: The evolution of the mitral vortex ring during diastolic filling is visualized by the iso-surface of vorticity magnitude $\frac{|\omega|D}{U_0} = 6$. a) The formation of the mitral vortex ring during E wave filling. b) The mitral vortex ring is bent and stretched toward the apex. Instabilities develops around its circumference. c) The breakup the E-wave mitral vortex ring and the formation of an additional vortex ring near the mitral orifice at the end of diastole.

complexity of the flow.

Recent studies [2,16] have revealed the complexity of the flow structure during diastolic filling inside the LV chamber. Our results further confirm these observations. Our work also emphasizes the complexity of intra-ventricular flow structure and suggested its dependence on the left ventricular wall kinematics. By changing the parameters of our left ventricle model, we will investigate the dependence of left ventricular flow patterns on the wall kinematics in diastole in future studies.

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