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Fluid–structure interaction of an aortic heart valve prosthesis driven by an animated anatomic left ventricle

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ABSTRACT

We develop a novel large-scale kinematic model for animating the left ventricle (LV) wall and use this model to drive the fluid–structure interaction (FSI) between the ensuing blood flow and a mechanical heart valve prosthesis implanted in the aortic position of an anatomic LV/aorta configuration. The kinematic model is of lumped type and employs a cell-based, FitzHugh–Nagumo framework to simulate the motion of the LV wall in response to an excitation wavefront propagating along the heart wall. The emerging large-scale LV wall motion exhibits complex contractile mechanisms that include contraction (twist) and expansion (untwist). The kinematic model is shown to yield global LV motion parameters that are well within the physiologic range throughout the cardiac cycle. The FSI between the leaflets of the mechanical heart valve and the blood flow driven by the dynamic LV wall motion and mitral inflow is simulated using the curvilinear immersed boundary (CURVIB) method (Ge and Sotiropoulos, 2007; Borazjani et al., 2008) [1,2] implemented in conjunction with a domain decomposition approach. The computed results show that the simulated flow patterns are in good qualitative agreement with *in vivo* observations. The simulations also reveal complex kinematics of the valve leaflets, thus, underscoring the need for patient-specific simulations of heart valve prosthesis and other cardiac devices.

1. Introduction

In spite of significant recent advances in imaging modalities for studying cardiac hemodynamics [3–5], present-day *in vivo* measurement techniques can only resolve large scale blood flow features [6]. Understanding flow patterns in the heart at physiologic conditions and scales sufficiently fine for establishing quantitatively links between heart disease and patient-specific hemodynamics continues to remain a major research challenge. This challenge becomes even more formidable when prosthetic heart valves are implanted [7]. For instance, *in vitro* experiments with simplified models of the left heart [8–11] and fluid–structure interaction (FSI) computational studies in straight [12–15,1,2,16,17] and anatomic [18] aorta geometries have clearly shown that at physiologic conditions the presence of a prosthetic heart valve gives rise to complex flow patterns characterized by fine scale flow structures and transition to turbulence. This complex and dynamically rich flow environment is widely believed to be the major culprit for the clinical complications that arise following implantation of valve prosthesis [19,20]. Considering the resolution limitations of present-day *in vivo* measurement techniques and the complexity of the underlying flow environment, high-resolution numerical simulation appears to be the only viable option for advancing our understanding of cardiac hemodynamics especially in the presence of prosthetic heart valves. For

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computational algorithms, however, to yield clinically relevant results their degree of realism needs to be drastically enhanced by incorporating into the modeling framework multi-physics elements of cardiac function along with input from modern imaging modalities and *in vivo* measurement techniques. In this paper, we report some progresses in that direction by developing a high-resolution FSI algorithm for simulating a bi-leaflet mechanical heart valve (BMHV) implanted in the aortic position of an anatomic beating left ventricle (LV) whose wall motion is simulated via a lumped-parameter model inspired by cardiac electro-physiology.

Critical prerequisite for simulating the motion of a BMHV in a beating left heart is the development of a model for simulating the LV hemodynamics, which is dominated by the complex interaction of the blood flow with the compliant and continuously deforming heart walls. Patient-specific simulation of the ensuing FSI problem from first principles is a formidable task since total heart function emerges as the result of the coupled interaction of the blood flow with a host of molecular, electrical and mechanical processes that occur across a wide range of scales [21,22]. At the cellular level contractile forces that cause the heart muscle to move are generated as cells are repolarized or depolarized by absorption or release of several ions (such as $[Ca^{+}]$, $[Cl]^{-}$, $[Na]^{+}$, $[K]^{+}$, etc., respectively). The resulting electrical excitation wave propagates throughout the heart via a fast conducting system known as the Purkinje fibers network [23]. Such cable-like conducting system controls the myocardial activation sequence and is thus very important for the emerging LV contraction [24]. At the tissue level, the heart wall is structured into three main layers: the outermost epicardium; the myocardium; and the innermost endocardium, which is in contact with the blood flow. The myocardium is significantly thicker than two other layers and contributes most of the contractile forces. The myocardial muscle fibers bind themselves into "sheet-like" structures [25,21], which are laid on top of each other wrapping around the LV chamber from the base to the apex and vice versa. At the organ level, the activation of cardiac cells also depends on the ions transport through the system of coronary arteries. Therefore, simulation of the whole heart organ continues to remain elusive as it would require a multi-physics simulation framework spanning a variety of scales ranging from metabolism of cardiac cells to the large scale fate transport (oxygen, ions, etc.) in coronary arteries and the aorta [26,4,22].

Available models for simulating blood flow in the heart can be broadly classified based on their spatial dimension and degree of sophistication into four categories [27,28]: (1) lumped and one-dimensional (1D) model; (2) two-dimensional (2D) models; (3) three-dimensional (3D) models with prescribed heart wall motion; and (4) three-dimensional models with coupled FSI simulation of blood flow and tissue mechanics (3D-FSI).

1D models rely on a non-linear relation between the LV pressure and the blood flow via an empirical, black box simulator [29–32,27,33]. Such models are simple to use and can efficiently obtain the pressure and volume curve but they are inherently incapable of providing the flow field inside the LV chamber.

2D models typically simulate idealized LV models [34–36]. Although these models can incorporate more physics than their 1D counter-part, their extension to simulate realistic LV flow in patient-specific geometries is difficult, if not impossible.

3D models employ a three-dimensional LV geometry, which can be idealized [37] or anatomic [38], with the wall motion prescribed either through simple analytical functions [37] or using patient-specific data [39,40,38]. In the latter category of 3D models [39,40,38], the patient-specific LV kinematics is reconstructed directly from *in vivo* MRI measurements. Such models can incorporate a high-degree of patient-specific realism provided that imaging modalities of sufficient resolution are available to accurately reconstruct the wall motion. Since the present day scanning frequency per heart beat (frames/s) is technologically limited, however, temporal interpolation between successive MRI images must be used to reconstruct the LV wall motion over the cardiac cycle [41,39,40,38]. Obviously the accuracy of the resulting kinematics, and consequently the clinical relevance of the 3D hemodynamic model, depends both on the accuracy of the interpolation technique and the initial temporal resolution of the MRI images. The spatial resolution of MRI can also be a potential source of error and uncertainty especially when subtle modes of the LV wall motion, such as twisting due to spiral fiber contraction [28], are to be incorporated in the model. The reconstruction of such feature ensures the fine scale structure of the flow be capture realistically.

From the modeling sophistication standpoint 3D-FSI models [42–47,28,48], are the most advanced as the heart wall is allowed to interact with the blood flow in a fully coupled manner. Critical prerequisite for the success of such models is the development of patient-specific constitutive models for the cardiac tissue that not only account or the interaction of blood flow with the heart wall but also for the interaction of the heart with surrounding organs [22]. These complexities require extensive assumptions on the heart wall structure and electrical activation [28] to enable fully-coupled blood-tissue interaction simulations, which could compromise the physiologic realism of the resulting models [45,44,46,28]. The first attempt to develop such a model was the pioneering work by Peskin [49,50] who assumed that myocardial fibers are discretely distributed. In this model the heart was assumed to be embedded in a periodic domain filled with fluid and the simulations were carried out at conditions that were not physiologic [50]. More recent versions of this model have been able to carry out simulations at higher cardiac volume flow rate [51,52,43,17]. Other 3D-FSI models have attempted to raise the degree of patient-specific modeling realism by incorporating information acquired from non-invasive imaging modalities. Such models have incorporated continuous fiber distribution into the wall model [28] and even attempted to couple the electrical excitation with the tissue response [47,22]. The main challenges confronting this class of models stem from the previously discussed limitations in the resolution of imaging modalities as well as the extensive simplifying assumptions that need to be incorporated in the FSI model.

With only exception the work by Peskin and co-workers [51,52], who simulated native heart valves, all previously discussed computational models have focused on the LV hemodynamics and neglected the presence of heart valves. Furthermore, and to the best of our knowledge, FSI simulations of heart valve prosthesis [see [53], for a recent review] have