ABSTRACT

MULTI-ORGAN FINITE ELEMENT MODELING OF THE HUMAN HEART WITH VENTRICULAR-ARTERIAL INTERACTIONS

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Computer heart models with realistic description of cardiac geometry and muscle architecture have advanced significantly over the years. Despite these significant advancements, there are nevertheless, some unresolved issues and aspects that need improvements. The goal of this dissertation was to address some of those issues as well as to develop new computational modeling framework to understand the underlying mechanics in heart failure with preserved ejection fraction (HFpEF) and pulmonary arterial hypertension (PAH).

Clinical studies have found that global longitudinal strain is reduced in HFpEF, suggesting that LV contractility is impaired in this syndrome. This finding is, however, contradicted and confounded, respectively, by findings that end-systolic elastance (Ees) and systolic blood pressure (SBP) are typically also increased in HFpEF. To reconcile these issues, we developed and validated a multiscale computational modeling framework consisting of detailed cell-based descriptors of the cross-bridge cycling against well-established organ-level physiological behaviors. This framework is then used to isolate the effects of HFpEF features in affecting systolic function metrics by quantifying the effects on Ees and myocardial strains due to 1) changes in LV geometry found in HFpEF patients, 2) active tension
developed by the tissue (Tref), and 3) afterload. Our study suggests that it is likely that the LV contractility as indexed by the tissue’s active tension is reduced in HFpEF patients.

Right ventricular assist device (RVAD) has been considered as a treatment option for the end-stage pulmonary arterial hypertension (PAH) patients, but, its effects on biventricular mechanics are, however, largely unknown. To address this issue, we developed an image-based modeling framework consisting of a biventricular finite element (FE) model that is coupled to a lumped model describing the pulmonary and systemic circulations in a closed-loop system. Our results showed that RVAD unloads the RV, improves cardiac output and increases septum curvature, which are more pronounced in the PAH patient with severe RV remodeling. These improvements, however, are also accompanied by an adverse increase in the PA pressure, suggesting that the RVAD implantation may need to be optimized depending on disease progression.

While it has long been recognized that bi-directional interaction between the heart and vasculature plays a critical role in the pathophysiological process of HFpEF and PAH, a comprehensive study of this interaction is hampered by a lack of modeling framework capable of simultaneously accommodating high-resolution models of the heart and vasculature. To address this issue, we developed a computational modeling framework that couples FE models of the LV and aorta to simulate ventricular-arterial coupling in the systemic circulation. We show that the model is able to capture the physiological behaviors in both the LV and aorta that are consistent with in vivo measurements. We also showed that the framework can reasonably predict the effects of changes in geometry and microstructural details the two compartments have on each other. The model is extended to accommodate a biventricular FE heart model together with FE models of the aorta and pulmonary artery to simulate the ventricular-vascular interactions in both systemic and pulmonary circulation.