BIVENTRICULAR HEART MODEL WITH NOVEL PSEUDO-FLUID DOMAINS AND THERMODYNAMICALLY-MOTIVATED GROWTH & REMODELING

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Abstract

A common flaw in existing finite element (FE) models of the heart is the unphysical specification of pressure or volume boundary conditions on the myocardium wall to represent the effect of the ventricle. Changes in the pressure and volume of blood in the ventricle cavity result from active contractility of the myocardium. Therefore, ventricle pressure and volume should be computed model outputs rather than prescribed boundary conditions. To overcome this deficiency without resorting to computationally expensive fluid-structure interaction (FSI) approaches, we propose a novel "pseudo-fluid" framework for highly efficient patient-specific biventricular heart simulations.
We process MRI images of healthy human hearts using a custom workflow to automatically create a 3D patient-specific biventricular mesh with distinct myocardium and "pseudo-fluid" domains representing blood in each ventricular cavity. Our constitutive law for myocardial tissue includes active contractility of cardiac myocytes (1) in addition to passive anisotropic hyperelastic behaviour of collagen and elastin in the ECM (2). Spatial distributions of cell and collagen fibre orientations throughout the myocardium are generated using the Laplace-Dirichlet Rule-Based algorithm (3). In the ventricle cavities our novel "pseudo-fluid" approach is implemented using a custom user-defined material subroutine to determine ventricle volume and pressure changes in response to the actively contracting myocardium. During systole, pressure and volume changes in each ventricle are governed by a bespoke Windkessel-type formulation that incorporates aortic and pulmonary compliance, in addition to the peripheral resistance. This novel approach allows for the simulation of a single cardiac cycle in 30 minutes, 3-4 orders of magnitude faster than traditional FSI heart models (4,5), while accurately capturing key pressure-volume relationships in multi-cycle simulations of inferior vena cava occlusion (6).

We integrate a novel thermodynamically-based growth and remodelling formulation to predict the development of eccentric and concentric hypertrophy. Tissue remodelling is driven by a deviation of the free energy of cardiac myocytes from a homeostatic free energy. Cell free energy is based on active contractility of sarcomeres, in addition to strain energy of the passive components of the cell and the ECM. Eccentric hypertrophy results from in-series addition of sarcomeres as a result of excessive stretching of the ventricle during the filling phase, whereas concentric hypertrophy results from in-parallel addition of sarcomeres

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due to increased resistance to cardiac output (7). This thermodynamically-based approach represents a significant advance on previous phenomenological stress or strain-based growth algorithms.

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