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# PERSONALIZED COMPUTATIONAL MODELING OF LEFT ATRIAL ELECTROMECHANICS

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**Summary:** Atrial fibrillation (AF) is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of mechanical function and associated with increased morbidity and mortality. The complex interactions between electrics and mechanics provide multiple potential pathways through which an altered electromechanical environment adversely affects atrial physiology and function. Personalized computational modeling provides a novel framework for integrating and interpreting the combined role of atrial electrophysiology and biomechanics in AF development and sustenance.

Personalized computational models were generated from high-resolution coronary computed tomography angiography (CTA) data and discretized into high-resolution tetrahedral finite element meshes. The complex left atrial myofiber architecture was estimated using an automated approach informed by anatomical and morphological images and based on local solutions of Laplace's equation. Cellular electrophysiology was represented using a biophysically-based human atrial cell model, while the propagation of the electrical activity was described by the monodomain model. Experimental biaxial mechanical tension test data of human atrial tissue were reinterpreted using a microstructurally-based anisotropic strain-energy function and represented the mechanical response of the left atrial myocardium. The coupling between electrophysiology and biomechanics was achieved using a biophysically-based active contraction model adapted to human atrial cell measurements. The hemodynamic response at the pulmonary veins and the mitral valve was governed by a phase-dependent Windkessel model, while the effect of the ventricles was incorporated using displacement trajectories.

Personalized computational models generated from high-resolution coronary CTA data included the heterogeneous thickness distribution of the left atrial myocardium and qualitatively reflected the complex left atrial myofiber architecture. The specific impact of an altered electromechanical environment, i.e., changes in myocardial stiffness, blood pressure, and ventricular deformation, on left atrial function over the individual phases of the cardiac cycle were quantified. This allows the comparison between healthy controls and patients with different pathological conditions to quantitatively investigate the link between electrophysiology and biomechanics and identify the capacity of the atria to sustain AF.