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Functional Analysis of Healthy and Heart Failure Tissue Populations using 3D Cardiac Electromechanical Models

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Computer modelling and simulation of the beating heart must reflect on electrical activation of cells and tissue, mechanical properties of tissue, and their interaction. Electrophysiological properties of the heart have been simulated abundantly and applied to increase mechanistic understanding of disease and improve treatment development. Recent maturity in cardiac mechanical modelling increased quantitative predictive power. However, tight coupling of cardiac mechanics with the underlying electrophysiological properties of the tissue makes modelling clinical mechanical phenomena such as cardiac disease and drug effects difficult. A combination of uncertainty quantification through populations of models and fully coupled electromechanics models provide greater predictive power on cardiac mechanisms and aid treatment development for cardiac diseases.

A fully coupled electromechanics model of ventricular tissue is developed by coupling the O'Hara-Rudy[1] electrophysiology model and the Land[2] mechanics model. A population of models was created by varying 16 electrophysiological and 11 mechanical parameters at the cell level. The population was calibrated from 1000 to 187 models based on biomarkers derived from the action potential shape, calcium transient and active tension. This calibrated population was altered in 11 parameters of the cell model to represent heart failure (based on [3]). A geometry of 20x7x3 mm is simulated with 1.0 or 0.5 mm spatial resolution for mechanics or electrophysiology respectively, with free movement in the fibre direction on one side. Results are extracted at the centre of the tissue, as well as tissue shortening on the free-contracting side.

Relative to the healthy population, heart failure manifests in both electrophysiological and mechanics biomarkers. The action potential takes ~20% longer to recover from activation and peak calcium concentration in the cell is reduced by 50.9%. In a similar trend, mechanical biomarkers show 42.2% reduction in peak active force, slower contraction and more variation in recovery times across the heart failure tissue population. The peak tissue shortening in the fibre direction as a result of free contraction is reduced from 0.68±0.07mm to 0.45±0.07mm (-33.2%) and its peak is delayed by 112ms (38.6%) in heart failure.

These simulations indicate strong effects on electrophysiological and mechanical heart function by population variation and disease such as heart failure. Therefore, strongly coupled 3D models are necessary to assess the impact of biological variation, cardiac vulnerability as well as safe and effective treatment development.

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