

Application of an Efficient Coupled Electromechanical Solver to Investigate End-stage Human Heart Failure

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Computational simulation has become a vital tool in developing our understanding of pathological cardiac conditions such as arrhythmias. However, as the underlying models are enhanced to include more realistic biological features, the construction of efficient and accurate software becomes increasingly challenging. For example, mathematical models which include cardiac tissue mechanics are highly non-linear and dominate the system solve time in coupled electromechanical systems.

We have developed a computational model of coupled electromechanical cardiac activity which uses the finite element method to approximate both electrical and mechanical systems on unstructured, deforming meshes. Performance of this model has been improved by preconditioning the mechanical system and by investigating the resolution required by the mechanical system, relative to the electrical system, to efficiently reproduce biological features to an appropriate level of detail.

We have then utilised this software to model re-entrant cardiac arrhythmias in end-stage failing tissue. Heart failure was simulated in the tissue by introducing changes to: (i) cellular electrophysiological properties that result in an increased action potential duration and altered intracellular calcium handling; (ii) tissue-level properties that simulate the gap junction remodelling, fibrosis and increased tissue stiffness seen in heart failure.

We then evaluated the relative contributions of cell- and tissue-level remodelling to arrhythmogenesis. In particular, the results from these simulations were compared on fixed and moving domains, paying specific attention to the effects of tissue deformation on the generation of spiral wave break-up.