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Modeling of electrophysiology and tension development in the human heart

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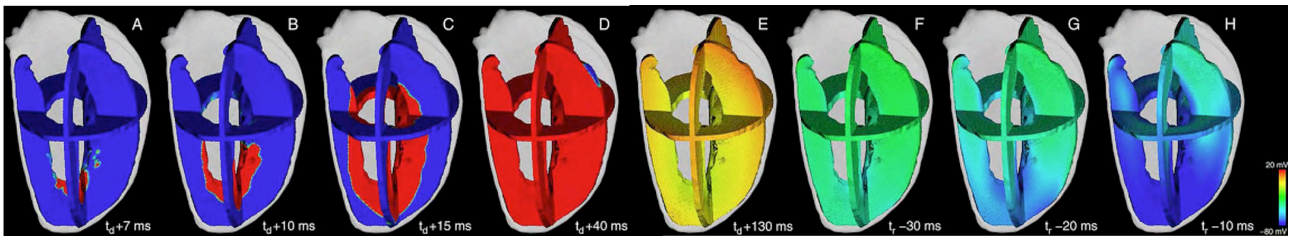
The electrical excitation of cardiac cells causes mechanical contraction. The activity depends on tissue type, heart rate, fiber orientation, distribution of gap junctions and pathologies. Electromechanical properties are not homogeneously distributed due to different ion channel expression. Some pathologies are caused by defects influencing the heterogeneous balance. A large group of pathologies lead to cardiac arrhythmia. Atrial fibrillation (AF) for example is the most common arrhythmia. Mechanisms underlying the interaction of cells and the initiation and perpetuation of arrhythmia are up to now not completely understood. Mathematical models form a basis to understand these mechanisms and interactions in the heart. Application of these models can be used for education and training purposes, for development and tests of drugs, and to develop new medical devices and therapeutic strategies. The models are based on measurement data of protein, cell, tissue, or organ properties and thus help to integrate different scales of measured data.



The modeling of cardiac electromechanics includes several different research topics, i.e. anatomy, electrophysiology, conduction, and tension development. To achieve quantitative simulations, new models were developed and existing models were enhanced. A main objective is to reconstruct human electromechanical heterogeneity in schematic and realistic anatomies. Modeling of heterogeneous electrophysiology is achieved by incorporating measurement data of different regions. The fiber orientations are inserted with rule-based methods. Furthermore, several different pathological states were modeled. A special focus is given on atrial and ventricular arrhythmia, electrophysiological remodeling due to AF and genetic defects like the long QT syndrome. The results help to gain new insights into the heart's function and to support the understanding of pathologies. Furthermore, the effects of drugs can be inserted in the model in order to investigate the effects of these drugs on human electrophysiology even in early development state.

Publications

- G Seemann et al.. Electrophysiological modeling for cardiology: methods and potential applications. *IT – Information Technology* (52), 242-249, 2010
- G Seemann et al.. Modeling of IK1 mutations in human left ventricular myocytes and tissue. *Am J Physiol Heart Circ Physiol* (292), H549-H559, 2007
- G. Seemann et al.. Heterogeneous three-dimensional anatomical and electrophysiological model of human atria. *Phil. Trans. Roy. Soc. A* (364), 1465-1481, 2006