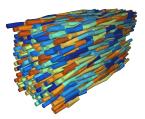
Discontinuity of Mircoscopic Cardiac Conduction in a Computational Cell-by-Cell Model

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Conduction velocity (CV) in cardiac tissue is a crucial electrophysiological parameter for arrhythmia vulnerability. Specifically, pathologically reduced CV facilitates arrhythmogenesis because such CVs decrease the wavelength with which re-entry may occur. Computational studies on CV and how it changes regionally in models at spatial scales multiple times larger than actual cardiac cells exist. However, microscopic conduction within cells and between them have been studied less. In this work, we study the relation of microscopic conduction patterns and clinically observable macroscopic conduction using an extracellular-membrane-intracellular (EMI)



Mesh of a block of cardiac tissue resolved at subcellular resolution with each color representing a different cell. Extracellular space is rendered transparently.

model. This model represents cardiac tissue with its three subdomains at subcellular resolution. By considering cell arrangement and non-uniform gap junction distribution, it yields anisotropic excitation propagation. In a first example, we initiate a macroscopic plane wave propagation in a block comprising 9 myocytes with membrane dynamics resolved with 65535 mesh elements and measure local activation times (LAT) defined as the time in which the transmembrane voltage passes 40% of the action potential amplitude. From the LAT data, we derive CVs and study their local course along the membrane of the cells and at their interfaces. Along the membrane of a cell, we observed a continuously propagating activation wave front due to diffusion of the transmembrane voltage gradient. This analysis will be extended to potential slowdown at the intercalated disc (cell-to-cell coupling via gap junctions). This novel kind of model can for example be used to understand how discontinuous conduction on the microscopic level affects fractionation of electrograms in healthy and fibrotic tissue.