ADVANCED ELECTROPHYSIOLOGY MODEL USING A WHOLE HEART SIMULATION

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ABSTRACT

Cardiac arrhythmias remain responsible for the majority of sudden cardiac death related coronary artery disease. Identification of new safe and effective drugs to treat this condition remains challenging, despite efforts to understand molecular mechanisms underlying both acquired and congenital forms of cardiac arrhythmias. Mathematical models and simulations have been created to understand the highly nonlinear cardiac electrical rhythm phenomena. Most studies, however, considered either single cell behavior, used simplified cardiac geometries, or have not considered the interactions between cardiac electrical and mechanical function. The current study utilizes the SIMULIA Living Heart Human Model (LHHM) by enhancing the electrophysiological (EP) behavior sufficiently to study drug treatment of cardiac arrhythmias.

The LHHM is a dynamic four-chamber heart model which provides a simulation platform to study phenomena such as safety and efficacy of cardiac medical devices, heat disease progression and EP behaviors. In the baseline model, the EP behavior was modelled using a 2-variable tissue-level phenomenological EP model with the goal of calculating accurate tissue activation times. In order to study more advanced EP behaviors, more advanced theories related to the cellular behaviors are required. In the current study, the electrical cellular behavior of the ventricles is enhanced to utilize the O'Hara-Rudy cellular model, a human ventricular model with 15 currents and 41 internal variables, with the ultimate goal of using the simulation to study pharmaceutical drug

impact on heart function. This mathematical model is highly sophisticated, yet mesh-sensitive; therefore, in the first part of the study we determine the optimal mesh size to obtain a realistic ventricle action potential and activation/repolarization sequence in electrocardiograms. We simulate one heart beat with varying mesh sizes ranging from 0.3mm to 0.7mm, resulting in a total element count up to 100 million. We will determine the maximum mesh size, which achieves a converged conduction velocity of action potential profile, which is the baseline model for the remaining phases of this study. In the next phase, we perform a cost-benefit analysis to determine the ideal High Performance Cluster (HPC) configuration to execute the baseline model. The minimal number of nodes which provides the best performance will be chosen as the cluster configuration for the remaining phase of the study. In the last phase of the study, we selected a few drugs with inhibitory effects on ion currents/channels, such as rapid delayed rectifier potassium current and L-type calcium current to study how drug block changes activation/repolarization sequence from the electrocardiograms.