Simulation of ECG under Ischemic Condition in Human Ventricular Tissue

Weigang Lu, Kuanquan Wang* and Henggui Zhang

Harbin Institute of Technology, China, and University of Manchester, United Kingdom

Myocardial ischemia is the foremost precursor of cardiac arrhythmias, which is caused by reduced coronary blood flow to the heart. However, the functional effects of ischemia-induced electrical properties of cardiac cells on ventricular electrical wave conduction have not been fully understood yet. In this study, to simulate ischemic effects, we developed a human ventricular cell and tissue models, which take into account three main pathophysiological consequences of ischemia: elevated extracellular potassium ([K+]o), acidosis, and anoxia. An ATP sensitive K+ current (IKATP) formulated by Ferrero et al. was adapted in the models. Using the model, we first simulated action potentials of endocardial, midmyocardial and epicardial cells with acute ischemic condition and measured respective action potential duration (APD90). Then we quantified the effects of ischemia on the waveform of ECG, particularly on ST segment depression or elevation by using a 2D model of human left ventricular tissue based on the anatomical structure of the human heart. In the ventricular tissue model, different transitional border zones mimicking the heterogeneity created by acute myocardial ischemia were considered. Effects of variation in local ischemic site and size in epicardial, transmural and endocardial regions were modeled respectively. At cell level, simulations showed that under acute ischemic condition, the resting membrane potential was elevated, and the APD was abbreviated by about 15 22% in endocardial cells, 16% 25% in midmyocardial cells and 14% 21% in epicardial cells. At tissue level, ischemia produced a depressed ST segment and change in the profile of ECG. The larger the size of the ischemic region, the more dramatic the changes in the amplitude of ST and T-wave were observed. With transmural ischemia, a double T-wave was formed. These simulation results matched to clinical data. Our model can be successfully implemented to simulate the electrical activity of human ventricular tissue affected by myocardial ischemia.