EFFECTS OF SMALL CONDUCTANCE CALCIUM ACTIVATED POTASSIUM CHANNELS IN ATRIAL MYOCYTES.

A. Peñaranda, Blas Echebarría, Enrique Alvarez-Lacalle, Inmaculada R. Cantalapiedra

Atrial fibrillation (AF) is one of the most prevalent cardiac diseases. Although it may have diverse causes, genetic screening has shown that a percentage of patients suffering of AF presents a genetic variant related to dysregulation of small conductance Ca2+ activated potassium (SK) channels. SK channels are potassium channels gated by changes in intracellular calcium. The functional role of these channels in cardiac electrophysiology is still under intense debate. While they do not seem to play an important role in healthy hearts – their associated current, $I_{KCa,}$ is smaller than other potassium currents -, there is increasing evidence that they may become relevant under pathological conditions. In fact, both pro- and anti-arrhythmic effects have been assigned to these channels, depending on the clinical situation. In this work, we have incorporated the current through SK channels, I_{KCa}, into an electrophysiological ionic model of human atria myocyte. This allows us to evaluate changes in the action potential under different parameters affecting the kinetics of these channels. We observe a large dependence of the I_{KCa} with the conductance and gate dynamics of the channel. SK channels are sensitive to changes in intracellular calcium dynamics avoiding or decreasing the pro-arrhythmic effect that events as spontaneous calcium release could produce.