Personalisation Of Action Potentials Based On Activation Recovery Intervals In Post Infarcted Pigs: A Simulation Study.

Cardiac modeling is a powerful and robust tool in electrophysiology (EP), supporting non-invasive arrhythmia diagnosis and therapy planning. Some studies showed that in silico modelling can be used to predict scar-related arrhythmia risk and ablation targets. However, model personalization is still relying on 'average' EP parameters derived from literature, largely due to a paucity of their identification from EP clinical data. We hypothesize that activation-recovery interval (ARI), a surrogate for action potential duration, APD) can be extracted from intracardiac electrograms (iEGMs) and used to parameterize models for more accurate AP wave simulations per individual case.

In this work we personalised APDs using ARI values extracted from endocardial electro-anatomical maps recorded in sinus rhythm and during pacing in post-infarcted swine (n=8). Specifically, we sought to investigate the differences in model parameters needed to calibrate simulated APDs in healthy tissue and border zone, BZ (i.e., arrhythmia substrate) when using an 'average' ARI computed from all cases versus those calibrated from ARIs extracted per each case.

To simulate AP waves, we used a modified Mitchell Schaeffer model with a FEniCSx implementation. We simulated a 2 cm virtual strand of tissue activated by a stimulus applied 10 times, and then computed an average APD on the strand from the last beat.

Results showed that average ARIs in healthy tissue and BZ for all cases during sinus rhythm were 206.12 $\pm$50.18ms and 213.21$\pm$ 52.1ms, respectively, whereas for pacing cases we obtained 282.5 $\pm$ 74.92ms and 310.43 $\pm$ 98.16ms, respectively. Figure 1 shows exemplary results of APD personalization using 'average' ARIs vs. per case ARIs, demonstrating significant differences. Furthermore, simulated tissue excitability (λ parameter) was reduced in the BZ compared to healthy myocardium.

This work underlines the importance of model personalisation by case, suggesting that is fundamentally needed to accurately reproduce in silico the experimental observations.

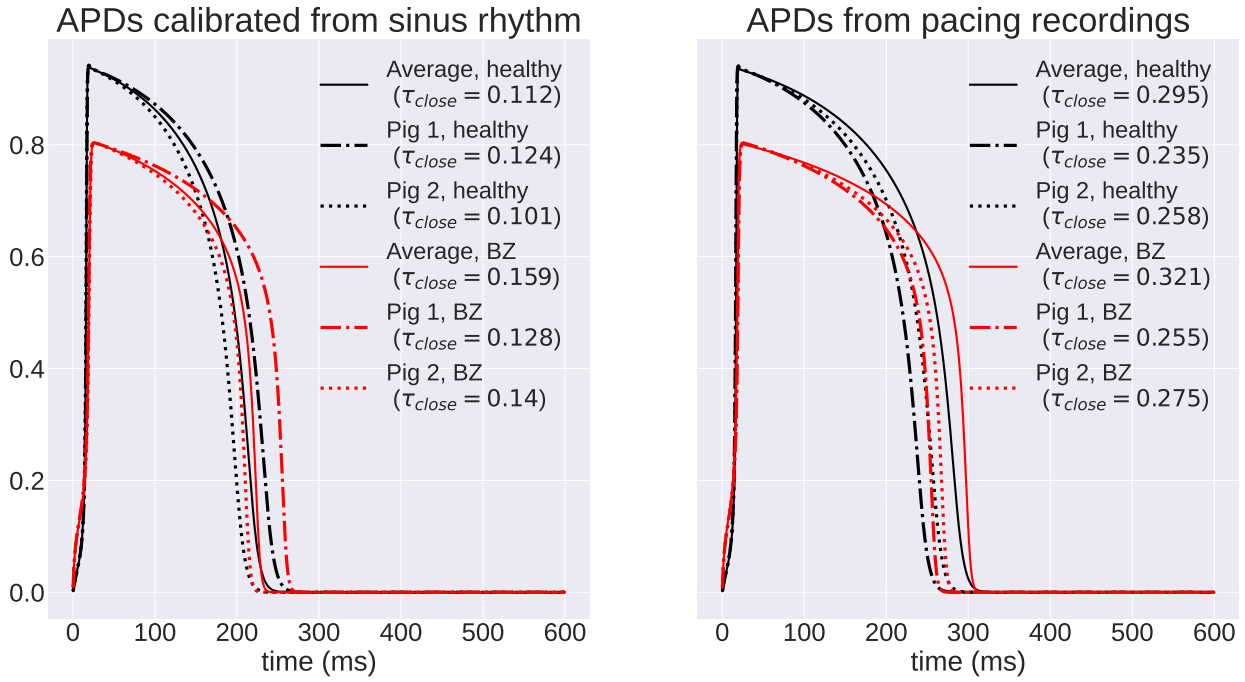

Figure 1: Examples of APD's obtained when calibrating from data in sinus rhythm and pacing recordings (left and right panel), respectively. The following parameters were fixed for all our simulations: $\tau_{in} = 0.3ms, \tau_{out} = 6ms, \tau_{open} = 120ms, u_{gate} = 0.13$. For all healthy cases $\lambda = 0.01$ and $u_{max} = 1$ whereas for the BZ we chose $\lambda = 0.2$ and $u_{max} = 0.9$ to decrease excitability.