

An In-silico Study into the Impact of Electrophysiological Variability at the Cellular Level on the Re-entry Patterns in Atrial Fibrillation.

Authors: Jordan Elliott¹, Olaf Dössel², Axel Loewe², Luca Mainardi¹, Valentina Corino¹, José Felix Rodriguez Matas¹

¹Politecnico Di Milano, Milan, Italy ²Karlsruher Institut für Technologie, Karlsruhe, Germany

Modelling the atria in-silico has become an important method in understanding atrial behaviour. With such large variation in the anatomical characteristics of the heart, significant research has been undertaken to determine the effects of this on the susceptibility to cardiovascular conditions such as atrial fibrillation. Atrial models typically include regional electrophysiological variability, but a typical assumption is that variability on a cellular level has little or no effect on propagation and therefore the susceptibility to and behaviour of an ectopic beat. The aim of the study is to determine the impact of cellular electrophysiological variability on the ectopic beats observed in atrial fibrillation. Using a population of models approach to introduce regional and cellular variability into the atrial model, ectopic beats were initiated in the ostium of the right pulmonary vein (RPV) and that of the left pulmonary vein (LPV). Six ectopic beats were applied in each ostium at a basic cycle length of 130ms, with an aim to create a unidirectional block. The variable model was compared with an equivalent model whereby each region was electrophysiologically homogenous, representing the regional mean action potential of the variable model. For both the RPV and LPV ectopic beats, the sinus rhythm propagation in the variable model reached each ectopic site roughly 10ms later than the average model. This resulted in the window of vulnerability occurring later and consequently resulted in a difference in morphology of the ectopic beat. Unidirectional ectopic beats in the RPV were formed when an ectopic beat occurred at $t=375\text{ms}$ in the variable model. In the average model, the same ectopic beat propagated in all directions. In conclusion, electrophysiological variability at the cellular level has a significant impact on both the total activation time of the atria, and as a result, the initiation and propagation of ectopic beats.

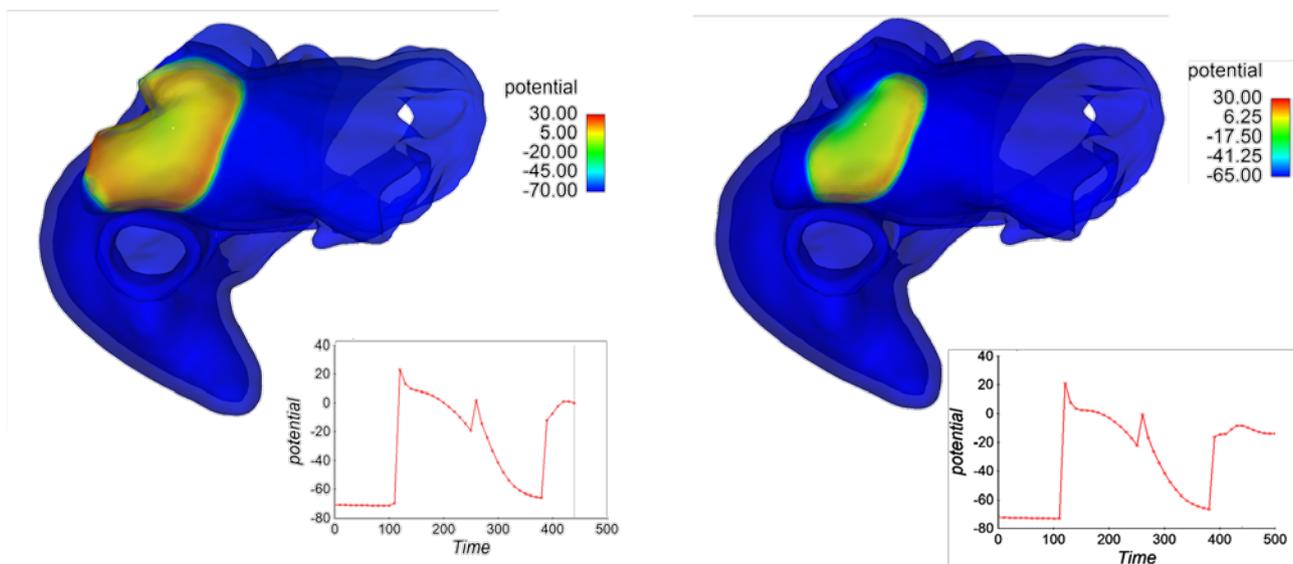


Figure 1 Propagation of the ectopic beat applied to the right pulmonary vein ostium, in the average (left) and variable (right) models. The wave propagation is shown at $t=450\text{ms}$. Ectopic beats were stimulated at a $BCL=130\text{ms}$, initiating at $t=245\text{ms}$.