Computational Risk Assessment of Ventricular Tachycardia based on Myocardial Scarring Characterization

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Introduction: Myocardial scarring is typically modelled as non-conductive tissue. We demonstrate employing electrophysiology simulations that detailed micro- and macroscopic modelling of non-ischemic (*Test 1*) and ischemic (*Test 2*) alterations enables patient-specific arrhythmic risk assessment.

Methods: We employed the Ten Tusscher-Panfilov ionic model for cellular behaviour and the monodomain model for macroscopic propagation.
In Test 1, Magnetic Resonance Imaging (MRI) and electro-anatomical mapping (S. M. del Carmine Hospital) were used for ventricular and non-ischemic fibrosis segmentation and for electrical conductivity calibration. In this context, to define suitable ionic models for non-ischemic cardiomyopathies, an optimization framework was developed in collaboration with Inria to calibrate ionic parameters against available optical mapping data.
In Test 2, we exploited MRI (Centro Cardiologico Monzino) for the segmentation of left ventricles and ischemic scars, modelling the latter with conductive channels generated via an open-source Perlin noise algorithm.

Results: Arrhythmogenic substrates were evaluated through ectopic simulations performed using LifeX (Finite Elements library developed at MOX and LaBS, Politecnico di Milano) at fibrosis/scar borders.
In Test 1, the optimization framework successfully fitted the simulated action potential to optical data, enabling the replication of experimental traces (Fig.1, left). Moreover, simulations correctly identified the clinical status of three patients: two were classified as arrhythmic due to high reentry incidence (6/10 arrhythmic ectopic sites), one was non-arrhythmic (3/10) (Fig.1, left).
In Test 2, the inclusion of conductive channels within the scar, rather than treating it as a non-conductive obstacle, promoted the formation of reentry circuits (Fig.1, right), matching clinical follow-up data. Conversely, modelling pathological regions as non-conductive oversimplifies the complexity of the substrate, leading to underestimated risks.

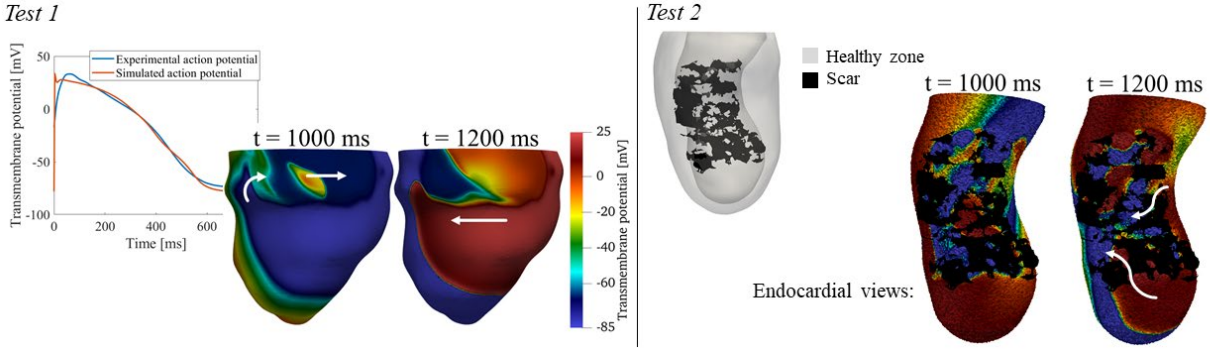

Fig.1. Left: Test 1. Output from the calibration process and evolution of a reentry; the arrows indicate the direction of the wavefront. Right: Test 2. Representation of conductive channels and visualization of the propagation pattern of a reentry.

Conclusion: These findings identify computational electrophysiology as a powerful non-invasive tool for patient-specific risk stratification. Moving beyond the non-conductive approximation by linking arrhythmogenic risk to substrate heterogeneity is essential to refine clinical treatment criteria.

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