

A Patient-Specific Modeling Pipeline for Non-Ischemic Cardiomyopathy

Riccardo Mariani, Chiara Celotto, Elvio Heidenreich, Diego Penella, Jose F Rodriguez Matas

Politecnico di Milano, Milano, Italy

Introduction: Non-ischemic cardiomyopathy (NICM) encompasses a heterogeneous group of myocardial diseases ranging from genetic to inflammatory etiologies, not related to coronary artery disease. Despite therapeutic advances, NICM is associated with dangerous arrhythmias such as ventricular tachycardia (VT) and sudden cardiac death. Although catheter ablation is widely used to control VT, current clinical imaging often fails to accurately localize patient-specific arrhythmogenic regions. This work proposes a patient-specific computational modeling pipeline to characterize the electrophysiological substrate of a subject affected by NICM secondary to acute myocarditis.

Methods: The patient-specific left ventricle (LV) geometry, reconstructed from late gadolinium-enhanced magnetic resonance images (LGE-MRI), was discretized with a high-resolution hexahedral mesh, incorporating myocardial fiber orientation and transmural heterogeneity. The scar identified in the LGE-MRI dataset was mapped onto the ventricular mesh. Three distinct modeling approaches were analyzed: i) a purely structural model with localized conductivity reduction; ii) a purely cellular model incorporating myocyte-fibroblast electrotonic coupling; and iii) a full model integrating both modifications. Simulations were performed using the Ten Tusscher-Panfilov model for human ventricular myocytes.

Results: The full model successfully replicates the electrophysiological alterations observed in clinic, while structural and cellular modifications alone only capture partial aspects of the pathology. The model exhibits a total activation time consistent with clinical expectations and reproduces a slow-conducting channel compatible with a re-entrant pathway, providing a suitable substrate for VT induction simulations (see Figure 1). The developed pipeline represents a foundational step toward in-silico clinical tools for the identification of critical arrhythmogenic targets and personalized ablation strategies to improve patient outcomes.

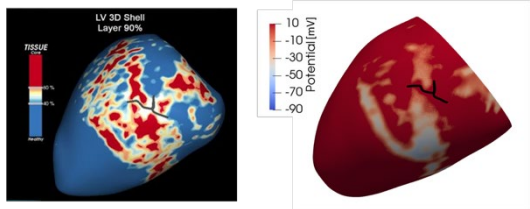


Figure 1. LGE-MRI of the patient's scar with the slow conduction channel (left panel) and the numerical model (right panel)