Modeling the interplay between acute myocardial ischemia and arrhythmogenesis

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Abstract:
Acute Myocardial Ischemia (AMI), especially in early stages, significantly increases the risk of ventricular reentrant arrhythmias. The reduced blood flow distal to a coronary occlusion triggers a cascade of ionic and metabolic effects such as hypoxia (reduced supply of oxygen), hyperkalemia (increased extracellular potassium concentration) and acidosis (reduced intracellular pH). The spatial heterogeneity arising in the myocardial tissue may result in the formation of reentries.

In this context, computational models could simulate the generation of arrhythmias in presence of AMI regions. Since quantitative information about the latter are hardly available from clinics, to date, computational models only integrate imaging data from chronic infarcted ventricles. This may not accurately reflect the acute condition.

In our study we develop an electrophysiological framework based on patient-specific myocardial blood flow maps acquired during a pharmacologically induced acute ischemic event, with the aim to analyze the arrhythmic propensity of the patients under investigation. Specifically, we consider a multiscale model, coupling the monodomain model for macroscopic propagation with the Ten Tusscher-Panfilov model suitably modified to account for the ionic alterations induced by AMI. Additionally, we partition the geometry of the left ventricle using myocardial blood flow maps to identify AMI regions, then assuming a correlation between the least perfused region and ischemic ionic parameters. This inter-individual analysis allows us to examine the impact of the volume of border and ischemic zones on arrhythmogenesis.

Moreover, in order to assess the influence of the dimension of the AMI region on arrhythmogenesis, for one specific patient we build virtual scenarios corresponding to less severe levels of AMI. By performing this intra-patient sensitivity analysis where different levels of AMI are virtually depicted, we can suggest that the distribution of ischemic regions, not just their volume, is critical in assessing the risk of arrhythmia.