Personalized Computational Framework to Study Arrhythmia Mechanisms on top of ECGI-Detected Substrate

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Electrocardiographic Imaging (ECGI) can unmask electrical abnormalities that were difficult to detect with the standard 12-lead ECG. However, it is still challenging to interpret the potential arrhythmogenic consequences of such ECGI-based electrical substrate. Here, we introduce a computational framework that enables personalized simulations of cardiac electrophysiology (EP). We apply it to study the virtual interaction between premature ventricular complexes (PVCs) and the ventricular recovery substrate of a patient with idiopathic ventricular fibrillation. With ECGI, we had identified pronounced local dispersion of recovery (55 ms/cm) in the right ventricle of this patient (i.e., a steep recovery gradient; panel A, red arrow).

A computational model of ventricular EP was developed based on the Mitchell-Schaeffer equations. Virtual pacing was applied in this model to simulate activation and recovery isochrones, which were qualitatively validated with invasive experimental canine data (panel B). This model was then used to mimic the recovery gradient and PVC (simulated by an extra stimulus "S2") found in the patient. We found that only the combined presence of 1) recovery gradients, and 2) a PVC could result in re-entry (panel C). We then studied a variety of gradients (6-98 ms/cm) and coupling intervals (-70 to +260 ms relative to end of local recovery) of the extra stimulus (panel D), which showed that re-entry can only occur when dispersion of recovery is large (≥76 ms/cm), and the extra stimulus occurs just after local recovery ended (~ +40 ms).

In conclusion, this computational framework enables to identify the specific conditions under which ECGI-detected substrates and PVCs can lead to re-entry in a personalized approach.