Potential Roles of Purkinje Fibers in Ischemia-Induced Arrhythmias

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Aims: This study aimed to assess the potential roles of the inclusion of the His-Purkinje-System (HPS) in arrhythmia dynamics of a ventricular computational model with a myocardial ischemic (MI) region.

Methods: A patient-specific bi-ventricular model was segmented from MRI data, including a HPS topology on the endocardial surface generated by a semi-automatic fractal method to produce a realistic ventricular activation. A MI region was incorporated in the ventricles by modelling the main aspects of ischemia (*i.e.*, hyperkalemia, hypoxia, and acidosis) in a gradually increasing fashion, from the peripheral to the central MI region.

Sub-epicardial voltage optical mapping of Langendorf-perfused rabbit hearts during regional ischemia was performed to assess the plausibility of the computational findings.

Results: Simulations with His-node pacing were able to produce re-entry through the ischemic region, which resulted in short-term ventricular tachycardia (VT) involving retrograde conduction in the HPS and was terminated due to synchronous activation of the ventricles by the Purkinje network. When the HPS was disabled once the ischemia-induced re-entry was generated, self-sustained VT was triggered.

Similar patterns of re-entry and termination of short-term VT were observed from optical mapping of isolated rabbit hearts. A premature highly homogeneous activation of the ventricles, before an also premature activation of the atria, could be caused by re-entry within the HPS and retrograde atrioventricular-node conduction.

Conclusion: Inclusion of the HPS in computational models is essential for studying arrhythmia maintenance and termination in the context of ventricular ischemia. HPS seems to have an important role, enabling arrhythmia termination. Further experiments targeting the ablation of the endocardial part of the HPS could offer better insights of its role in arrhythmia termination.