A Numerical Study of Atrial Fibrillation Electrophysiological Substrate based on High-Density Mapping

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Background

Catheter ablation guided by high-density electroanatomical (EA) maps constitutes an established procedure for managing atrial fibrillation (AF). Electrogram signals collected in sinus rhythm allow the reconstruction of the electrical wavefront propagation and the localization of electrical abnormalities. However, the interpretation of EA maps and the consequent definition of ablation strategies in the different forms of AF are subject to continuous debate.

Objective

This study aimed to quantitatively characterize the electrophysiological substrate of patients suffering from paroxysmal and persistent AF using numerical simulations parametrized with EA activation maps acquired during sinus rhythm.

Methods

We constructed a computational pipeline to simulate the formation and sustainment of localized reentrant circuits in patients affected by paroxysmal and persistent AF. We first processed the EA activation maps by computing conduction velocities with a least-square approach, providing a quantitative characterization of the main electrophysiological properties (slow conduction corridors, wavefront collisions, and pivot points). Conduction velocities were then adopted to parametrize a coupled model for cardiac electrophysiology formed by the monodomain equation and a system of ordinary differential equations describing the ionic species dynamics. This parametrization provided a detailed characterization of the heterogeneous electrical and structural properties responsible for different reentry mechanisms. Finally, we numerically predicted with the parametrized model the dynamics of localized reentrant circuits by simulating ectopic beats originating from the pulmonary veins.

Results

The numerical results highlighted the role of conduction properties' distribution in the formation and sustainment of different localized reentries. Specifically, heterogeneous areas led to head-tail interactions, which generated unstable localized reentries with moving rotors. Instead, areas with severe slow conduction corridors led to the anchoring of the rotors and, consequently, to the sustaining of the phenomenon.

Discussion

The quantitative analysis of the electrophysiological substrate showed a progression in the number and the severity of slow conduction corridors moving from paroxysmal to persistent AF. Numerical simulations associated the latter form of AF with an augmented probability of localized reentries anchoring in severe slow conducting areas. On the contrary, the less frequency of these areas, together with head-tail interactions, determined unstable localized reentries in the paroxysmal case.

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