Mechanisms of Initiation and Acute Termination by Non-invasive Identification of Atrial Fibrillation Drivers

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It remains unclear the mechanisms by which premature atrial complexes (PACs) induce Atrial Fibrillation (AF) and their evolution during spontaneous AF onset. We studied the anatomical distribution and nature of AF initiation drivers and their implication respect to acute termination by Pulmonary Vein Isolation (PVI) in a unique subset of patients with PAC and spontaneous AF onset characterized by ECG imaging (ECGI).

ECGI was reconstructed from 62 electrodes using the zero-order Tikhonov method in 13 patients referred for AF ablation (10 male, 67±8 years). Premature Atrial Complexes (PAC) that did and did not initiate AF were identified as well as spontaneous transitions to AF (A). Focal and re-entrant sites were identified by phase analysis on ECGI maps (B).

PACs inducing AF were more likely to arise from the PVs than non-inducing PACs, in form of focal (6±9 vs 2±9, C) and re-entrant sources (5±6 vs 1±1, D). During AF onset, Pulmonary Veins of patients with successful PVI harbored less focal (15±12% vs. 33±17%, p=0.02, AUC=0.775, E) and more reentrant sources (32±21% vs. 22±14%, p = 0.04, AUC = 0.775, F) compared to unsuccessful PVI patients.

Anatomical distribution of PACs supports the role of PV in spontaneous AF onset. Characterization of anatomical driver distribution at spontaneous AF onset showed significant differences when compared with acute termination by PVI. This may help to plan ablation procedures and provide insight on the patient-specific fibrillatory process.