Abstract: 1348

Analysis of amplified sinus-p-wave to localise left atrial low voltage substrate and guide pulmonary vein isolation in persistent atrial fibrillation

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Background: A subgroup of patients with persistent atrial fibrillation (AF) has advanced left atrial (LA)-low voltage substrate (LVS), resulting in significantly reduced success rates following pulmonary vein isolation (PVI). Duration of amplified p-wave was demonstrated before to relate to the presence of LA-LVS.

Purpose: To investigate the spatial distribution of LA-LVS in persistent AF, its relation to anatomical structures involved in the inter- and intra-atrial electrical conduction, and its impact on morphology/duration of amplified p-wave and PVI-outcome.

Methods: 95 persistent AF-patients underwent high-density (>1200 sites) LA voltage mapping in sinus rhythm. Extent of LA-LVS (bipolar voltage <1.5mV, <1.0mV and <0.5mV) was analyzed in nine LA regions and related to underlying anatomical structures. Impact of LA-LVS distribution patterns/extent on p-wave morphology and duration were evaluated using digitized amplified 12-lead-ECG (40mm/mV, 100-200mm/s). PVI outcome during a 12 month-follow up was assessed in persistent AF-patients who underwent their first PVI.

Results: LA-LVS at <1.0mV was most frequently found at the anteroseptal LA (59%), followed by LA roof (46%) and posterior LA (35%). Comparable results were found for LVS <0.5mV and <1.5mV. Characteristic changes of amplified p-wave morphology related to the distinct spatial distribution of LA-LVS: early anteroseptal LA-LVS involving the typical anatomic location of the inter-atrial connecting myocyte bundles of Bachmann was associated with diffuse inter-and intra-atrial conduction delay leading to p-wave prolongation. Advanced anteroseptal LA-LVS resulted in significant conduction delay/block of Bachmann bundle and development of positive-negative p-wave in the inferior leads D2, D3 and aVF. Extensive LA-LVS involving both the anteroseptal and posterior LA resulted in monophasic-positive (right atrial) p-waves with isoelectric second (left atrial body) p-wave portion and apparition of a late positive deflection in the lateral leads (D1, aVL, V5-6), reflecting the late activation of the lateral LA and LA-appendage. Based on these findings, an algorithm involving amplified p-wave-duration (<150 ms excludes relevant LA-LVS, mean 4.9cm² at <1.0 mV, stage 1; 150-180 ms in advanced LA-LVS, 28.6cm², stage 2; >180 ms in extensive LA-LVS, 42.3cm², stage 3) and -morphology (positive-negative inferior p-waves or late-terminal positive lateral p-wave in stage 3-LA-LVS) was developed. This algorithm correctly stratified 139 persistent AF-patients according to the observed arrhythmia freedom rate within 12 months following PVI (figure 1).

Conclusions: LA-LVS develops predominantly at the anteroseptal LA. The regional distribution affects morphology and duration of the amplified p-wave in characteristic ways. Careful analysis of amplified p-wave-duration and -morphology allows for rapid, non-invasive estimation of LA-LVS and arrhythmia freedom following PVI in persistent AF.
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