13.1 - Pathophysiology and Mechanisms

A novel workflow for advanced mapping of repolarization dispersion from endocardial unipolar electrograms in patients with coved-type brugada syndrome

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Background: Accentuation of the action potential notch in the epicardium causes the prolongation of the epicardial action potentials in the right ventricular outflow tract (RVOT) and is the basis for arrhythmogenesis in patients affected by Brugada syndrome (BrS). Activation-recovery interval (ARI) approximates the action potential duration and may be used to study repolarization dispersion in BrS.

Purpose: Our aim was to investigate repolarization dispersion within the RVOT in spontaneous coved-type ECG BrS patients and in controls, evaluating also the relation between repolarization dispersion and conduction abnormalities to better define the electro-anatomical substrate of the disease.

Methods: BrS probands (n=13) and control subjects (n=4) underwent endocardial mapping of RV with the CARTO 3 system. BrS patients were also studied with programmed ventricular stimulation (PVS). Data was exported from the CARTO system and converted into MatLab format using OpenEP. A specific region of interest (ROI) including the sub-pulmonary RVOT and entire RV free wall was then selected using Paraview. With an automated algorithm, we measured ARI and ARIc (corrected using the "Bazett formula") for each point of the ROI, which were then interpolated to obtain ARI maps. We also acquired right ventricular activation time (RVAT) and maps for the assessment of slow conduction zones. By dividing RVAT color scale in 5 ms steps (isoSteps), isochronal activation areas were identified in the ROI.

Results: Out of the 13 BrS subjects, 4 had VT or VF inducible during PVS (PVS+) while 9 did not (PVS-). One patient had appropriate ICD shocks during follow-up. BrS patients had higher mean ARI and ARIc compared to controls (288.5$\pm$22.2 vs 251.5$\pm$8.4 ms, $p<0.001$; 312.0 $\pm$30.4 vs 281.8$\pm$10.3 ms, $p=0.023$). ARI was found to be higher in PVS+ than in PVS- patients (294.8$\pm$27.1 vs 285.8$\pm$20.1 ms, $p=0.5$), as well as ARIc (327.7$\pm$49.9 vs 304.6$\pm$16.4 ms, $p=0.49$) although these differences were not significant. BrS patients showed a longer RVAT (106.0 $\pm$21.4 vs 74.5$\pm$5.5 ms, $p=0.003$), and increased zones of crowding, as expressed by isochronal steps (13.85$\pm$2.94 vs 9.25$\pm$1.26, $p=0.01$). Notably, a strong correlation was found between ARIc and RVAT (Pearson R coefficient = 0.83, $p<0.05$), independently from the results of PVS (PVS+ R=0.99, $p=0.01$; PVS- R=0.83, $p=0.06$).

Conclusions: We introduced a novel workflow for the electrical substrate characterisation of subjects with BrS phenotype. Our preliminary data show the presence of repolarization dispersion along with conduction slowing in the RVOT of BrS patients compared to controls. In our population, repolarization dispersion and impairment of RVOT conduction were strongly related especially in inducible BrS patients.