received pacemakers with AF diagnostics and 4 preventive algorithms (Selection and Prevent AF series, Vitatron). A 3-month Diagnostic Phase with conventional pacing identified a Substrate Group (>70% of AF episodes with <2 premature atrial contractions [PACs] before AF onset) and a Trigger Group (≤70% of AF episodes with <2 PACs before AF onset). This was followed by a 3-month Therapy Phase when in the Trigger Group algorithms were enabled aimed at avoiding or preventing a PAC and in the Substrate Group continuous atrial overdrive pacing was enabled.

Results: 126 patients were evaluable, in the Trigger Group (n=73) a statistically significant 28% improvement in AF burden (median AF burden: 2.60hrs/day, Diagnostic Phase vs. 1.49hrs/day, Therapy Phase; p=0.03304 signed rank test), and reduced PAC activity could be documented. There was no significant improvement in AF burden in the Substrate Group (median AF burden: 1.82hrs/day, Diagnostic Phase vs. 2.38hrs/day, Therapy Phase; p=0.12095, signed rank test), and little change in PAC activity.

Conclusions: We identified a subgroup of patients for whom the selection of appropriate pacing algorithms, based on individual diagnostic data, translated into a reduced AF burden. Trigger AF patients were more likely responders to preventive pacing algorithms as a result of PAC suppression.

738 Spontaneous augmentation of ST elevation is related to clinical predictors of outcome in Brugada syndrome
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Typical ECG pattern of Brugada syndrome is dynamic and often concealed and it can change over time related to many influences. Predictors of outcome have been proposed based on clinical evaluation, ECG pattern and ventricular arrhythmias induced at PES. Relation between ST-segment elevation and occurrence of ventricular arrhythmias has been reported. Nevertheless, relation between spontaneous augmentation of ST elevation and clinical profile of patients has not been elucidated.

Objectives: The aim of this study was to determine this relation by analyzing spontaneous augmentation of ST-segment elevation during usual daily life.

Methods: 21 patients and 25 non-affected first degree relatives were included. Demographic and clinical profile of the patients were assessed. A 24-hour digital Holter ECG with 12 leads was recorded during daily life. Beat to beat ST level at 40, 60 and 80 ms after J point was analyzed. Episodes of spontaneous augmentation of ST level (>2 mm from baseline = absolute episode or from ST level at ECG baseline = delta episode) were assessed.

Results: Clinical diagnosis was done based on typical ECG pattern at baseline or unmasked by drugs (71% with positive drug test). 17 were men (81%) with mean age at diagnosis of 44.5 ± 16.9 (15-73). Family history of sudden death in 24%, 62% symptomatic at diagnosis, 29% “coved type” (type 1) ECG pattern and 33% “saddle back” type (type 2) ECG pattern. PES was done in all patients. ICD was implanted in 76%. 60% of patients showed transient episodes of absolute ST augmentation and 42% of them (5p) also showed delta episodes. No episodes were assessed in any relatives.

A transient elevation of ST was related to patients with abnormal ST at baseline. 80% of patients with type 1 ECG, 70% of patients with “saddle back” type ECG and 25% of type 3 ECG at baseline showed ST absolute episodes during Holter monitoring. Patients with normal ECG at baseline showed no episodes. Contingency table of the relationship between basal ECG-type and assessment of ST absolute events at 60 ms to J point showed statistical significance (P<0.05). Absolute episodes at 60 and 80 ms occurred more frequently in patients with an abnormal ECG at baseline (p<0.05). Episodes of absolute ST-level augmentation occurred more frequently in men (p=0.08)

Conclusion: 12 lead ECG Holter monitoring is a useful tool to assess ST absolute events in patients with Brugada Syndrome. Furthermore, spontaneous augmentation of ST elevation is related to sex and ECG pattern at baseline, two clinical predictors associated with a worse clinical outcome.

739 Positive ajmaline challenge in arrhythmogenic right ventricular dysplasia: Brugada syndrome or both?
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In patients with typical arrhythmogenic right ventricular dysplasia-cardiomyopathy (ARVD/C) ajmaline challenge can induce right bundle branch block (RBBB) and coved ST elevation in >1 right precordial leads in about 15% of cases. On the other hand biopsies or autopsies in Brugada syndrome (BS) have revealed histological findings of ARVD/C in a third of cases superseding a certain overlap.