drive pacing (S2-S2), we examined an initial cyclic variation after the induction of the tachycardia by measuring intertrial intervals of H2-H3, H3-H4 and H4-H5.

Results: As shown in table, the tachycardia cycle length immediately after the induction was paradoxically shorter than the subsequent tachycardia cycle length in F-S AVNRT only. Temporal P-QRS variation during F-S AVNRT and a combination of S-F AVNRT with the same earliest atrial site in 5 F-S AVNRT (83%) suggested the presence of low common pathway (LCP) located below the AVN reentry circuit.

Conclusions: Paradoxic shortening of H2-H3 in F-S AVNRT may be explained by DVR in which H2 and H3 are antegradely captured over FP and SF, respectively, followed by slow-slow AVNRT with a long LCP, presenting long RP tachycardia.

P4970 | BENCH
Entraînement from the right ventricle distinguishes fast-slow AV nodal reentrant tachycardia from permanent junctional reciprocating tachycardia
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Introduction: The response to entrainment from the right ventricular apex (RVA) is useful to distinguish typical AV node reentrant tachycardia (AVNRT) from atrioventricular reentrant tachycardia (AVRT). The purpose of this study was to determine whether this response is useful for differentiating fast-slow AVNRT from the permanent form of junctional reciprocating tachycardia (PJRT).

Methods: We studied 33 patients (P) with fast-slow AVNRT or PJRT with successful entrainment from the RVA. All tachycardias had a VA interval which was longer than the AV interval. Diagnoses of fast-slow AVNRT or PJRT were made according to conventional electrophysiologic criteria and confirmed by the outcome of the ablation. Fifteen P (mean age 55±24 years, 6 males, tachycardia cycle length -TCL- 446 ±72 ms) had fast-slow AVNRT, and 18 P (mean age 41±22 years, 10 males, TCL 353±59 ms) had PJRT. The following responses to entrainment were analysed: difference between the pacing interval after entrainment and TCL (PPI-TCL), difference between the Stimulus-Atrial electrogram interval during entrainment and the VA interval during tachycardia (SA-VA interval), and the A-V response upon cessation of ventricular overdrive pacing (A-V versus pseudo A-A-V response). Optimal cut-off values of continuous variables were determined by ROC curve analyses.

Results: Significant differences in mean values for SA-VA interval, PPI-TCL and A-V responses were observed between the two groups (table). The diagnostic yield of the selected cut-off values was also shown. For PPI-TCL, area under ROC curve 0.85; p<0.01. For SA-VA: area under ROC curve: 0.9; p<0.004.

Table 1

| Variable | PPI-TCL 115±98 211±62 0.006 130 0.76 0.90 0.93 0.76 | PPI-TCL 194±53 <0.001 100 0.76 0.93 0.93 0.76 | AV response 30% 18% -0.01 0.92 0.82 0.86 0.90 |

Conclusions: The response to tachycardia entrainment from the RVA is useful in order to distinguish fast-slow AVNRT from PJRT. A PPI-TCL lower than 130 ms and an SA-VA interval lower than 100 ms are highly suggestive of PJRT and make fast-slow AVNRT unlikely.

P4971 | BEDSIDE
Long-term outcomes of ivabradine in inappropriate sinus tachycardia patients: appropriate efficacy or inappropriate patients?

Background: Inappropriate Sinus Tachycardia (IST) is characterized by persistent and disproportional elevation of Heart Rate (HR). Ivabradine has been successfully used in some patients.

Methods: 24 patients (16 women, 41±13 year-old) were diagnosed of IST according to current guidelines criteria. Patients were treated with 5 to 7.5 mg of Ivabradine twice a day. 24-h Holter recordings and the SF-36 Health Survey were performed at 6 months to evaluate both HR control and clinical status.

Results: Holter recordings before and after 6 months on treatment showed a significant reduction in the average maximal HR of 155±18 vs 132±16 bpm, mean HR of 97±6 vs 79±8 bpm (mean day-time HR of 103±5 vs 84±10rpm) and minimal HR of 58±12 vs 48±7rpm (Wilcoxon analysis, p<0.05). The SF-36 mean score showed a significant improvement on Ivabradine treatment (57±23 vs 76±20), with a better physical and mental status scores (56±25 vs 74±22 and 58±24 vs 78±18, respectively) (Wilcoxon analysis, p<0.001). Mean dose of Ivabradine was 5.6±1.4 mg. No episodes of severe bradycardia or syncope were reported. After 1 year, patients were asked to stop treatment to reevaluate the situation. 20 patients were on treatment and only 10 patients accepted to stop Ivabradine. Only two patients (20%) remained on IST criteria.

Conclusions: IST patients treated with Ivabradine showed both HR normalization and quality of life improvement maintained in the long-term follow-up. Stopping Ivabradine after one year unexpectedly showed that HR remained in the normal limits in 80% of the patients.

P4972 | BEDSIDE
High density mapping of ventricular scar: a comparison of ventricular tachycardia supporting channels with channels that do not support VT
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Introduction: Surviving myocytes within scar may form channels that become critical components of a ventricular tachycardia (VT) reentrant circuit. There is little data on the physiognomies of channels that comprise VT circuits and those which are non-VT channels.

Methods: Twenty-two patients with ischemic cardiomyopathy (LVEF 32±8%) and multiple inducible VT (n=73) were evaluated. Left ventricular endocardial mapping was done with high-density Pentara™ catheter connected to the Ensite Velocity™ system. Pacing was done from biopoles showing low voltage (<1.5V) with late potentials and/ or fractionation. A channel was defined as series of matching pace-maps with stimulus (S) to QRS time ≥40ms. Sites were determined to be part of VT channel if there were matching pace-maps to the induced VT during endocardial mapping. This was confirmed with entrainment mapping when possible. The anatomical and electrophysiologic properties of these channels were evaluated.

Results: A mean of 760±205 voltage points were taken, with 431±137 within scar and 329±137 outside scar (<0.5mV). Overall 2507 pace-maps (114±62 per patient) were performed. Of the 238 channels identified, 57 channels corresponded to an inducible VT. Channels that were part of a VT circuit were of greater length (mean 52±31 vs 36±26mm), had longer S-QRS time (31 vs 25ms), longer conduc-tion time (108±90 vs 35±25ms) and slower conduction velocity (CV) (0.7±0.5 vs 1.3±1.1mm/s) than non-VT channels (p<0.01). The probability of finding a longest S-QRS time >80ms was 5.3 times higher in a VT channel relative to an unshared non-VT channel (95% CI 2.6 to 10.6, p<0.001). VT channels were more commonly located within dense scar (95% vs 81% non-VT channels, p<0.04). Of all the fractionated (mean 130±105) late (mean 10±5) and very late (mean 4±3) potentials located in scar, only 22%, 30% and 35% respectively were recorded within VT channels. Compared to elsewhere within the scar, in the region of VT channels fractionated potentials were longer in duration (104±26ms vs 95±26ms, p<0.001), and very late potentials were poorly coupled (309±159ms vs 240±119ms, p<0.04) to the intrinsic QRS. Following ablation targeting channels, the clinical VT was abolished in 95% of patients, and no VT could be induced in 64%.

Conclusion: High density mapping shows substantial differences among channels in ventricular scar. Channels supporting VT are more commonly located in dense scar, longer than non-VT channels, and have slower CV. Only a minority of scar related potentials participate in the VT supporting channels. These findings have pertinent implications for substrate based ablation strategies.

P4973 | BEDSIDE
Treatment strategy for Ebstein anomaly with accessory pathways occurring in infancy
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Background: There are few reports on Radiofrequency Catheter Ablation (RFA) in Ebstein’s Anomaly (EA) associated with tachycardias in infancy. This study aimed to clarify the characteristics of the Accessory Pathways (AP) related tachycardias in EA and to find the strategy to treat EA in infancy.

Methods: Patients with APs in EA undergoing RFA during an 18 yr. period (1995-2012) were enrolled. The age at the time of the RFCA, surgical intervention before the RFCA, and RFA results were analyzed and compared between patients less than one y.o. and those more than one y.o.

Results: Thirty-one surgeries in 27 patients (median age 7.6 y.o., female 17, male 10) were performed. The arrhythmias in 12 patients with APs (group Y) occurred at <1 y.o. (including fetal tachycardias) and in 15 with APs (group O) at >1 y.o. The age at onset of the tachycardia was 1.7 months and 9.8 y.o. in each group, and the median age and average body weight at the time of the RFA were 3.8 y.o. and 10.3 and 11.9 y.o. and 35.6kg, respectively. The tachycardias were refractory to multiple Anti-Arrhythmic Drugs (AADs), which were used more in group Y (2.5) than group O (0.5). Brain damage caused by arrhythmias was seen

Supraventricular tachycardias

913