Methods: EPS was performed in 51 patients aged from 13 to 50 years (mean 30±16) for a PS. Atrial stimulation was performed in control state (CS) at a cycle length of 400ms and coupling was decreased until APRP or atrial RP. Measurement was repeated after isoproterenol when APRP≥250ms in CS. Measurement was performed by transesophageal route and then by intracardiac route, with a delay not ≥3 months.

Results: APRP’s were 267±50ms in CS, 233±49ms after isoproterenol at transesophageal EPS, 309±72ms in CS, 280±64ms after isoproterenol at intracardiac EPS (p<0.002). Among 31 patients with initially APRP ≥250ms at transesophageal EPS, APRP was ≤220ms after isoproterenol in 12 patients. All, but one had an APRP≥250ms in CS at intracardiac EPS; one patient with APRP of 270ms at esophageal EPS had a value of 240ms at intracardiac EPS in CS but value was 250ms after isoproterenol. Three patients with transesophageal APRP in CS and after isoproterenol ≥230ms had lost their anterograde conduction. At transesophageal EPS, APRP was ≤250ms in 20 patients of which only 8 had a short AP-RP at intracardiac EPS and 3 had a short AP-RP after isoproterenol. There was no significant difference for the induction of orthodromic tachycardia at transesophageal and intracardiac EPS (72%) and there was a similar induction of atrial fibrillation at transesophageal EPS (32%) and intracardiac EPS (28%).

Conclusions: A long AP refractory period measured at transesophageal EPS excluded the presence of an AP with a short refractory period at intracardiac study even after isoproterenol. There was an adrenergic factor during transesophageal EPS and isoproterenol produced a significant shortening of AP refractory periods in only 39% of patients with APRP≥250ms. When APRP is ≥280ms at transesophageal EPS, the loss of anterograde conduction in APRP can be expected in the following weeks (15%).

0049

Long-term follow-up of AV conduction disturbances after slow pathway ablation in patients with AV node reentrant tachycardia

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AV block following radiofrequency (RF) ablation for the treatment of ativoventricular nodal reentrant tachycardia (AVNRT) is a rare but well recognised complication of the procedure. The purpose of the study was to report the long-term follow-up of patients a first degree AVB (AVB1), second degree AVB (AVB2), or third degree AVB (AVB3) during ablation of AVNRT.

Methods: 930 patients, 615 females, aged from 12 to 92 years, mean age 52±18, had AVNRT. RF energy, 65%, 40 watts was delivered on the slow pathway, until AVNRT was not induced.

Results: 94 patients presented a transitory or permanent AVB1,2,3. In 8, mean age 53±21.5 years, AVB was of vagal origin generally occurring at femoral puncture (group 1). In 26 patients, mean age 46±21, it was traumatic and regressive occurring either in young patients with a normal conduction system or in 3 patients with a left bundle branch block. In remaining 60 patients, AVB was directly related to the RF application; AVB was of first degree in 22 patients aged 56±17 years; it was of 2nd or third degree AVB in 38 patients: in 2 patients AVB3 remained permanent and in all other patients it was partially or totally regressive. After a follow-up of 2.1±2 years, pace-maker implantation was implanted in 15 patients. 1 patient with traumatic AVB3 aged 81 years, 5 patients with AVB3 during ablation, 2 with permanent AVB3 (0.2%) and 3 with transitory AVB3 and 9 patients without AVB during ablation. In these last patients, 2 had spontaneous long HV interval. Age of these patients differed from age of patients with RF-related AVB (73±14 vs 56±17) (p<0.04). 5 patients with transitory AVB3 remained symptomatic with alternating slow junctional rhythm and sinus tachycardia.

Conclusions: AVB remains frequent during AVNRT ablation (10%) but it is frequently benign and not directly related to the RF application. Permanent complete AVB is exceptional (0.2%). Patients with transitory complete AVB remain at high risk of later events as conduction disturbances or sinus tachycardia. Another AVB’s are age-related and probably without relation with ablation. Permanent or transitory 1 degree AVB seems without clinical significance.

0372

Outcomes of patients with unexplained syncope, bundle branch block and normal electrophysiological study

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Background: Little is known about predictors of high-degree atrioventricular block (AVB) in patients without evidence of advanced His-Purkinje conduction disturbances at electrophysiological study (EPS) performed for unexplained syncope associated with bundle branch block (BBB).

Aims: Identify electrocardiographic predictors of high-degree AVB during follow-up of these patients. Methods. In this multicenter cohort, patients were included if they had: (1) unexplained syncope, (2) bundle branch block (≥210ms), (3) no HPCD at EPS, i.e. baseline HV interval <70ms, and absence of 2nd- or 3rd-degree AVB induced with atrial pacing or ajmaline challenge.

Results: Among the 150 studied patients (72±14 years, 62% male, mean left ventricular ejection fraction 57±8%), index electrocardiograms showed right BBB (70%; n=105), left BBB (2.7%; n=4), and nonspecific intraventricular conduction disturbance (3%; n=4). A first-degree AVB was noted in 62 patients (44.3%). During a mean follow-up of 584±88 days, high-degree AVB was documented in 25 patients (17%). The presence of a first-degree AVB on the index electrocardiogram was associated with an increased risk of subsequent high-degree AVB [72% vs. 35%; p<0.01]. Conversely, no patients with isolated right BBB developed high-degree AVB after a normal EPS.

Conclusion: In patients with unexplained syncope associated with BBB, 16% of patients subsequently developed high-degree AVB despite no evidence of advanced HPCD at EPS. This was more likely to occur in patients with first-degree AVB at presentation. Conversely, this finding never occurred in patients with isolated BBB and normal PR interval.

0392

Prospective evaluation of QT duration in eating disorder patients correlations with morphological and biological parameters

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Introduction: long QT and related ventricular arrhythmias may complicate the evolution of Eating Disorders (ED) but duration of QT interval and prevalence of long QT in large series of ED patients has not been reported.

Methods: QT intervals were measured in 100 consecutive ED patients (anorexia nervosa, bulimia nervosa or mixed) in lead II, V2 and V5 and corrected using Bazett’s formula. 95 healthy subjects matched in age and gender forms the control group.

Results: mean age was 30±12 yo and 93% were females. Mean Body Mass Index (BMI) was 16.9±3kg/m². Heart rate was significantly lower in patients with ED (64±14 bpm) compared to controls (78±15 bpm) (p<0.001).

QT were significantly longer in ED patients than in controls in leads II (384±33 vs 366±32, p=0.0003), V2 (377±37 vs 367±35ms, p=0.04) and V5 (381±34 vs 365±30ms; p=0.0005). Corrected QT were significantly shorter in patients with ED compared to controls in leads II (389±28 vs 412±22ms), V2 (383±27 vs 413±32ms) and V5 (387±26 vs 410±28ms) (p<0.001 for each comparison). None of the ED patients had a corrected QT >480 msec (max