Method and result: A 64-year-old male with standard indication for CRT-D implantation was referred to our institution for a CRT-D implantation. Unfortunately, intubation of the CS was not possible. TEE showed a congenital CS anomaly with complete drainage of the CS into the left atrium. Because of contra-indication for general anaesthesia and thus for surgical epicardial implantation, we proposed to implant the LV lead by a transseptal approach. After the patient had given his consent, a transseptal puncture was performed via the right femoral vein. A conventional screw-in lead was implanted at the laterobasal segment of the LV using a deflectable catheter guide introduced via the left subclavian vein through the transseptal puncture. Post-implantation parameters of the LV lead were acceptable: pacing threshold 0.7 V-0.4 ms, impedance 435 ohms, R wave amplitude 5 mvolts. Right ventricular and right atrium leads were then implanted (fig1)

A post-implant CT scan confirmed the ectopic location of the CS ostium without persistent superior vena cava (fig 2).

The patient was discharged on anticoagulation with a targeted international normalized ratio between 3 and 4. At the one- and three-month follow-up, no complication had occurred. He was in class 2 of the NYHA and had not been hospitalised. Electrical parameters remained steady.

Conclusion: Complete drainage of the CS in the left atrium without persistent vena cava is a rare congenital abnormality. When cardiac resynchronisation therapy is needed, transseptal implantation could be a feasible alternative.

Gene-specific effect of beta-adrenergic blocker on QT duration in the Long QT syndrome

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Background: In the long QT syndrome (LQTS) the clinical efficacy of beta-blocker treatment differs according to the genotype. We aimed to assess the effect of beta-blocker treatment in LQT1 and LQT2 patients.

Patients and methods: 24-hour Holter ECG were recorded before and after beta-blocking therapy initiation in genotyped LQT1 (n=30, 8 males, mean age 21±17) and LQT2 patients (n=16, 8 males, mean age 19±15). QT duration was measured on consecutive 1-minute averaged QRS-T complexes leading up to 1440 QT-RR pairs for each recording. Then, we computed subject- and condition-specific log/log QT/RR relationships which were used to calculate QT interval duration at RR=1000 ms (QT1000).

Results: Before treatment, coefficients were higher in LQT2 than in LQT1 patients (0.53±0.10 vs. 0.40±0.11, p<0.001) and QT1000 was longer in LQT2 than in LQT1 patients (521±38 vs. 481±39 ms, p<0.01).

Beta-blockers significantly prolonged the mean RR interval (RR=827±161 ms before treatment and 939±197 ms on beta-blocker, p<0.0001). The coefficients were not significantly modified by beta-blockers (0.41±0.09 in LQT1 patients and 0.52±0.12 LQT2 patients). Beta-blocker treatment was associated with a prolongation of the QT1000 interval (from 481±39 to 498±43 ms, p<0.01) in LQT1 patients but with a shortening in LQT2 patients (from 521±38 to 503±32 ms, p<0.01).

Conclusions: Our results confirm the elevated coefficient of the QT/RR relationship in LQTS patients. LQT2 patients showed higher coefficient and longer QT1000 when compared to LQT1 patients. The effect of beta-adrenergic blocker on QT1000 duration was gene-specific. Given the demonstrated efficacy of beta-blockers in LQT1 and 2 patients, our data suggest that QT1000 might be a poor predictor of outcome under anti-adrenergic therapy.
was measured and programmed atrial stimulation with 1 and 2 ES performed in control state (CS) and after isoproterenol.

Results: At 2nd study, among pts studied for syncope at study 1, 1 has still syncope, 2 have AVRT, 1 has rapid AF, and 2 are asymptomatic. Among pts with AVRT at study 1, 25 have AVRT, 7 are asymptomatic and 2 have AF. Among pts with AF, 4 have still AF and 1 is asymptomatic. Among asymptomatic pts 3 have a spontaneous malignant form, 7 remain asymptomatic, 3 have AVRT, 1 has syncope and 1 has AF. All AVRT or AF occurred in pts with inducible AVRT or AF at EPS 1. The higher rate conducted by AP was significantly lower in CS and after isoproterenol at study 2 (157±45 b/min, 193±113) than at study 1 (199±65, 257±65). AP has lost anterograde conduc- tion properties in 17 pts aged from 17 to 67 years (47±15); all of them but one had initially 1/1 conduction through AP 170/min. However 8 of them had still AVRT. Among pts with initially rapid conduction through AP (250/min), all but one have a rapid conduction at EPS 2, 3 of them which were asymptomatic developed rapid AF.

Conclusion: The study confirms that a benign form of WPW without inducible AVRT or AF remains benign. Pts with AVRT and AF and long refractory period become asymptomatic in 20 % of cases. Pts with inducible rapid AF remain at high risk of events in most cases.

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Low stress-induced ST segment / Heart rate hysteresis as a predictor of low microvolt T-wave alternans

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Background: ST segment / heart rate hysteresis can improve the diagnostic performance of the exercise ECG for prediction of coronary artery disease and cardiovascular mortality. As this simple variable integrates the heart rate adjusted ST segment depression during exercise and recovery phase, ST/HR hysteresis is related both to ischemia and sympathovalvular balance. Microvolt T wave alternans (TWA) measurement during stress testing has been suggested as a predictor of life-threatening cardiac arrhythmia. According to the Coumel’s triangle hypothesis, the sympathovalvular balance could modulate TWA. Dipyridamol-induced ischemia does not disturb significantly this balance.

Purpose: We aimed to assess the absence of an elevated TWA under a dipyridamol SPECT imaging.

Methods and Results: All 339 consecutive patients who were referred for a pharmacological stress test with dipyridamol. The maximum TWA was determined with the modified moving average method. Using for ST/HR hysteresis a cutoff of 0.038 mV, yields a 75 % negative predictive value for a TWA above the reported threshold of 47 µV (from holter recordings). ST/HR hysteresis negative predictive value was even better reaching 90 % to rule out the occurrence of a higher TWA (65 µV derived from ECG stress testing).

Conclusion: The absence of significant alterations in sympathovalvular balance seems to be associated with low T-wave alternans. ST/HR hysteresis might be useful to exclude the possibility of an elevated T wave alternans.

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Six-hour holter recording of microvolt T-wave alternans and heart rate turbulence in the CCU compared to classical 24-hour ambulatory ECG

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Background: T-wave alternans (TWA) and heart rate turbulence (HRT) measured during 24 hour ECG recordings are 2 powerful non-invasive tools to risk-stratify cardiovascular patients. The aim of our study is to assess whether a fast ECG-holter scan yields different information from classical 24-hour ambulatory ECG regarding TWA and HRT measurements.

Methods: All consecutive 21 patients with a non-ST-elevated myocardial infarction and admitted in intensive care unit of cardiology, have been monitored with an ECG-holter for classical, TWA and HRT analysis over 24 hours. TWA has been measured with the modified moving average method. Routine reading of the holters has been followed by a specific analysis. Each 24-hour period has been divided into four equal periods. Maximal TWA, T-onset and T-slope for HRT over those four 6-hour periods have been analyzed and compared with full day results using a repeated measures analysis of variance (ANOVA).

Results: 16 men and 5 women aged between 31 and 90 (mean 57.5 +/- 24.8) have been included. Mean maximal TWA was 73 ± 25 µV. 6-hour max- imal TWA was 59 +/- 23 µV, 55 +/- 26 µV, 56 +/- 30.01 and 48 +/- 23.72 µV (p=0.11). HRT as assessed by T onset and T slope were -0.00619 % +/- 0.02, 0.0033 % +/- 0.04, 0.00571 % +/- 0.03, -0.00952 % +/- 0.03 (p=0.46) and 3.75 +/- 3.99, 5.46 +/- 7.29, 6.32 +/- 9.13, 5.96 +/- 10.85 (p=0.46) respectively for each time period.

Conclusion: This preliminary study suggests that a 6-hour ECG-Holter recording could be a reliable and feasible method to assess cardiovascular mortality and risk of SCD in patients admitted for an acute coronary syndrome in intensive care unit by studying TWA and HRT. Faster risk stratification could thus be done during hospitalization in order to optimize therapeutics and better identify candidate for fast ICU discharge.

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C-reactive protein: a new marker of arrhythmic event in Brugada syndrome?

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Background: We studied the relationship between C-reactive protein (CRP), and clinical manifestation of Brugada syndrome.

Methods and Results: All patients underwent physical examination and detailed cardiac tests. At admission, prior to any intervention, they had blood samples drawn for CRP. Among 54 patients, 37% were symptomatic (17 syncope and 3 aborted sudden death) and 63% were asymptomatic. Mean CRP level was 2.4x1.42mg/l in symptomatic group and 1.41x0.92mg/l in asymp- tomatic group (P=0.03). In a multivariate model, CRP concentrations >2 mg/l remained an independent marker for being symptomatic (P=0.004; 95% CI: 1.8 to 21.7) and a predictor of ICD implantation (P=0.008; 95% CI: 2.2 to 19.8).

Conclusion: C-reactive protein is significantly higher in symptomatic patients with Brugada syndrome and seems to be a biomarker for cardiac events in high risk Brugada patients.

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P wave signal analysis is able to recognize with a good accuracy patients with or without previous atrial fibrillation

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Purpose: Paroxysmal atrial fibrillation (PAF) is a common cardiac arrhythmia but difficult to diagnose: frequently asymptomatic and when symptomatic too sporadic to be captured by electrocardiogram (ECG) or even long duration Holter monitoring. However this diagnosis carries a major importance as it could induce key changes in diagnosis and therapy (anticoagulation). Our