left ventricular ejection fraction 28 ± 14%). Cardiomyopathy was ischemic in 62 and nonischemic in 38 pts. At the time of referral, 73 pts had electrical storm, defined as 3 or more shocks within 24 hours. Importantly, pts received an average of 6 shocks (range 0-90) and 17 attempts of anti-tachycardia pacing (0-148) in the month preceding referral for Abl. Eighty pts were either being treated with (73) or intolerant of (7) amiodarone at the time of referral, with use of high dose amiodarone in 57 pts (intravenous infusion in 32 and oral ≥ 400 mg daily in 25 pts). Sixty-three pts were being treated with a different antiarrhythmic medication, either alone or in combination with amiodarone.
Conclusions: Despite advances in catheter Abl of VT, consensus guidelines suggesting treatment earlier in the course of disease, significant toxicity of amiodarone and risks of ICD shocks, Abl remains a treatment of last resort for VT in structural heart disease, with the majority of pts in electrical storm at the time of referral, having had multiple VT episodes in the preceding month and frequently being treated with high dose amiodarone. Additional effort is required to change practice patterns and encourage earlier consideration of VT ablation.

PO5-111

LEFT VENTRICULAR NONCOMPACTION IS ASSOCIATED WITH PROGRESSIVE RISK FOR CHF, STROKE, AND VENTRICULAR TACHYCARDIA

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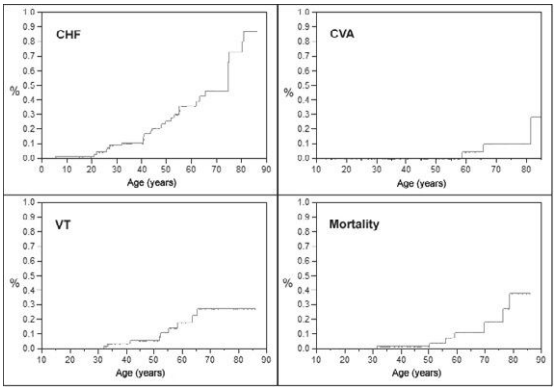
Introduction: Left ventricular noncompaction (LVNC) is an inherited cardiomyopathy with arrest of normal embryogenesis of the endomyocardium and myocardium. Our understanding of the natural history of LVNC continues to evolve.
Methods: The cohort included LVNC diagnosed by echocardiography or CMR using noncompacted/compacted ratio of > 2:1 in end-systole or 2.4:1 in end-diastole respectively. The data is presented as mean±SD and percentages.
Results: Eighty-two patients were found to have LVNC. Only 25.6% were diagnosed first on echocardiography. Mean age at diagnosis was 49.4±20.4 years, with 51% males and 89.5% Caucasians. Associated right ventricular noncompaction was present in 24.7%. The mean LVEF was 52.4±17.5% and clinical congestive heart failure was present in 37.5%. Left ventricular dysfunction (EF <50%) was present in 35.4%. Ventricular tachycardia was observed in 12.7% of patients. CVA occurred in 4.2%. The risks for unfavorable clinical events as a function of age are shown in the Kaplan Meier curves (Figure).
Conclusions: LVNC is associated with CHF, CVA, and ventricular arrhythmias. The risk for developing these outcomes appears to progress over time.



PO5-112

IS THERE A BENIGN VARIANT OF CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA?

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Introduction: Exercise-induced polymorphic ventricular tachycardia (PMVT) is associated with malignant heritable arrhythmias such as long-QT Syndrome (LQTS) and catecholaminergic polymorphic ventricular tachycardia (CPVT), which often present with syncope or sudden death in children or young adults. The pathophysiology and prognosis of asymptomatic adrenergic PMVT is less clear.
Methods: This case series describes the characteristics of 5 patients presenting with effort-induced nonsustained PMVT discovered incidentally during treadmill exercise stress testing as part of a work up for benign or unrelated symptoms.
Results: Five patients (age 51.6+/-5.4 years, all male) with incidental detection of ventricular ectopy without a cardiac history were referred to the London Inherited Heart Rhythm Clinic over a period of 4 years. 17 additional patients (age 37+/-19 years, 13 female) presented with typical findings of CPVT, including symptoms of cardiac arrest (n=7), syncope (n=7), a positive family history (n=9) and a disease causing RyR2 mutation (n=10). All 5 "benign" patients had asymptomatic non-sustained PMVT during exercise stress testing. None had prior effort syncope or presyncope, a family history of sudden death, or a reversible cause. All baseline ECGs were normal and noninvasive imaging (echo, CT, MRI) excluded any significant structural heart disease. Ischemic precipitants were also excluded with cardiac catheterization or combined coronary CT angiography and nuclear stress perfusion imaging. Adrenaline infusions were entirely negative in 2 patients and provoked frequent multifocal PVCs and QT prolongation in 2 other patients. Genetic screening for LQTS and CPVT performed in 4 patients, was negative. Treatment with a long-acting beta-blocker resulted in a significant reduction in the frequency and complexity of ectopy on repeat exercise stress testing, in each of the 5 patients. All remained asymptomatic throughout an average follow up period of over 2 years.
Conclusions: We describe apparent asymptomatic effort-induced PMVT in middle-aged males that may represent a benign variant of CPVT.

PO5-113

A RISK SCORE BASED ON SPECTRAL TURBULENCE ANALYSIS OF THE SIGNAL AVERAGED ECG FOR PREDICTING CARDIAC DEATH IN SUBJECTS WITH CHRONIC CHAGAS HEART DISEASE

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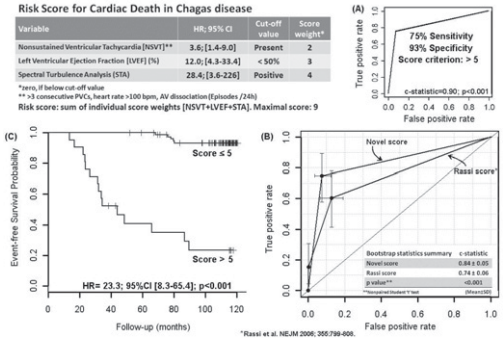
Introduction: Cardiac involvement is the main cause of death in chronic Chagas disease (CCD). This study prospectively investigated signal-averaged ECG (SAECG), 24h-ambulatory ECG and echocardiogram parameters in CCD aiming at developing a predictive score for death.
Methods: Clinically stable outpatients with CCD (34 to 74 y.o, 38 males) staged according to Los Andes (Class I: N=28; II: N=48; III: N=24) were enrolled. Deaths were ascertained by review of medical records. SAECG was acquired on admission. Spectral turbulence analysis (STA) was carried out on XYZ leads after

short-time Fourier transform mapping of ventricular activation, and applying intersegment spectral correlation technique.
Results: During a follow-up of (mean±SEM) 95.3 ± 3.1 months, 20 patients died (rate: 26.4 ± 1.4/year). In multivariate Cox proportional-hazard model, NSVT/24h (p=0.006), LVEF<50% (p<0.001), and positive STA (p=0.001) were independent predictors of death (Table inset). A prognostic score was developed by calculating weighted points proportional to beta coefficient in Cox model (Table inset). ROC analysis showed optimal cut-off value at 5 (Figure A). KM curves of novel score for deaths are presented in Figure B (proportional hazard test: rho=-0.5; p=0.06). In 1,000 bootstraps, ROC c-statistic of novel score was superior to Rassi score (Figure C).
Conclusions: In CCD, NSVT, LVEF<50% and high SAECG spectral turbulence are independent predictors of death. A novel risk score improves predictive accuracy in this population.



PO5-114

INTRAVENOUS SOTALOL INDUCED QT PROLONGATION PREDICTS SERUM SOTALOL CONCENTRATION

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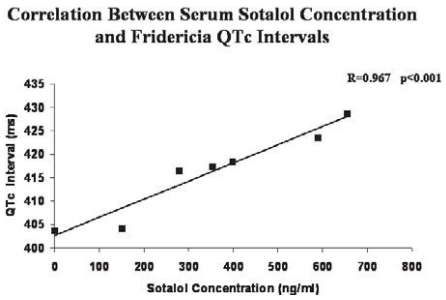
Introduction: Sotalol (S) prolongs the cardiac action potential that can be observed as QT interval (QT) prolongation on the surface EKG. It is known that S causes Torsade de Pointes ventricular tachycardia (TdP) that is due to dose related QT prolongation. We were able to develop a correlation between QT prolongation and S serum concentration.
Methods: 15 healthy volunteers (age: 32±8 y) received 75 mg intravenous S over 2.5 hr at a constant infusion rate. Serum S concentrations were determined and a 12-lead EKG was recorded at baseline, 0.5, 1, 2, 3, 4, and 5 hrs following dosing. The QT and RR intervals were measured by two blinded investigators. Heart rate corrected QT (QTc) was calculated by the Bazett (B), Fridericia (Fi), and Framingham (Fh) formulas. Linear regression analysis was performed between S concentration and QT measurements.
Results: The analysis showed close correlation between S concentration and QT (r=0.938, p<0.02), B QTc (r=0.860, p<0.02), Fh QTc (r=0.960, p=0.001), and Fi QTc (r=0.967, p<0.001). The equation of the regression line was: QTc= 0.0388 x (S concentration) + 402.63, which closely predicted actual QTc at any S concentration. Conversely any degree of QT prolongation can predict the measured serum S concentration.
Conclusions: A simple equation was found that predicts serum S concentration from QT prolongation. Measuring QT prolongation can yield a quick method of estimating serum S concentration as well as the electrophysiologic effects of serum S. Excessive serum S levels that causes increased QT prolongation can be avoided, possibly reducing the risk of TdP.



PO5-116

ELEVATED C-REACTIVE PROTEIN IS ASSOCIATED WITH ELECTROCARDIOGRAPHIC MARKERS OF SUDDEN CARDIAC DEATH IN PATIENTS UNDERGOING PROPHYLACTIC ICD IMPLANTATION

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Introduction: Inflammation may produce arrhythmia by a number of mechanisms. We examined the relationship between inflammation and ECG metrics that may predispose to lethal arrhythmias in patients with post-myocardial-infarction or non-ischemic cardiomyopathies.
Methods: The study population was selected from the Prospective Observational Study of the ICD (PROSE-ICD) in primary prevention of sudden cardiac death. QT variability index (QTVI), a marker of ventricular arrhyhthmia, was calculated as the log ratio of the coefficients of variance of the QT interval and heart rate from a 5-10 minute digital ECG recorded in each participant at enrollment. Only sinus rhythm ECGs were included. Serum C-reactive protein (CRP) at enrollment was used as a marker of inflammation.
Results: The study population of 429 patients (age: 59±13 years; 73% male; 32% non-white; EF 21±11%; NYHA 2.2±0.8) was divided into quartiles based on the CRP levels. With increasing quartiles of CRP, an increase in the coefficient of variance of QT interval (p=0.036), a decrease in the coefficient of variance of the heart rate (p=0.011), and an increase in QTVI (p<0.0001) were noted. The associations remained significant even after adjusting for potential confounders.
Conclusions: Increasing baseline CRP levels at ICD implant are associated with increased QT- and depressed HR-variability, both of which are established markers for SCD. These findings provide insights into the possible pathways by which inflammation may lead to SCD.