H012

QT DYNAMIVITY PREDICTS SHORT TERM MORTALITY IN ACUTE HEART FAILURE PATIENT

F. DESPAS  1, D. FRUIT  1, C. BAIXAS  2, M. CASTEL  1, C. ALQUIER  1, M. GALINIER  2, J.-M. SENARD  1, A. PATHAK  1

1 Equipe 8 Inserm U858, I2MR, Hôpital de Rangueil, CHU, Toulouse, France
2 Service de Cardiologie, Hôpital de Rangueil, CHU, Toulouse, France

Introduction — Sympathetic overactivity increases the risk of ventricular arrhythmias. Previous studies showed that alteration of QT dynamicity, reflecting abnormal sympathetic modulation of ventricular repolarisation, is independently predictive of sudden cardiac death among patients with chronic heart failure. The aim of this study was to determine whether impaired adaptation of the QT interval to changes in heart rate also predicts death at one month in patients with acute heart failure.

Methods — In this study, we prospectively included 99 patients (mean age 71±13 years) with acute heart failure (EF = 35.2±13.4%, BNP = 1045±83 pg/ml). Heart rate variability and QT dynamicity was evaluated by analyzing 24-h Holter recordings. The linear regression slope of QT interval measured to the apex and to the end of T wave plotted against RR intervals was calculated using dedicated Holter algorithm. Clinical, biological and morphological data were collected. Follow-up was performed by direct examination or.

Results — After a follow up of one month 21 patients died. Non survivors were older (76±8 vs. 69±15 years, p<0.05), had significantly decreased ejection fraction (28.8±12.7% vs. 36.9±13.1%, p<0.05), lower hemoglobin levels (11.4±1.7 g/dl vs. 12.5±2.3 g/dl, p<0.05) and red blood cells (3650±458 1000/µl vs. 4287±771 1000/µl, p<0.05). Among parameters issued from 24-h Holter-EKG analysis only day QTe slope was significantly decreased in non survivors (0.33±0.23 vs 0.56±0.64, p<0.05).

Conclusion — These results suggest that clinical, biological and electrical parameters issued from 24 hours Holter-ECG recording are able to predict short term mortality in patients with acute heart failure. These preliminary results need to be confirmed by a more extensive statistical approach currently ongoing. These predictive parameters are simple to use and their clinical or pharmacological interest need to be confirmed.

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HEART RATE VARIABILITY ANALYSIS IS NOT EFFECTIVE FOR SLEEP-APNOEA-SYNDROME SCREENING IN PATIENTS WITH CHRONIC HEART FAILURE

T. DAMY  1, M.-P. D’ORTHO  1, B. ESTRUGO  1, L. MARGARIT  1, G. MOUILLET  1, F. ROUDOT-THORAVAL  1, E. VERMES  1, F. ROCHE  2, I. MACQUIN-MAVIER  1, J.-L. DUBOIS-RANDE  1, L. HITTINGER  1

1 Hôpital Henri Mondor-Inserm U841, Créteil, France
2 CHU Nord, St Etienne, France

Frequency-domain analysis of heart rate variation has been extensive statistical approach currently ongoing. These predictive parameters are simple to use and their clinical or pharmacological interest need to be confirmed.

Frequency-domain analysis of heart rate variation has been suggested as an effective screening tool for sleep apnoea syndrome (SAS) in the general population. The aim of this study was to determine whether this method could be effective in patients with chronic congestive heart failure (CHF). 84 patients with stable CHF, left ventricular ejection fraction (LVEF) <45%, and sinus rhythm were included prospectively. Patients underwent simultaneous polygraphy to measure apnoea/hypopnoea index (AHI) and ECG Holter monitoring to measure the power spectral density of the very low frequency component of the heart rate increment, expressed as the percentage of total power spectral density (% VLFI).

VLFI could be determined in 54 patients (mean age 52.8+/9.13 years and LVEF 33.5±9.8%). SAS defined as AHI >=15/h-1 was diagnosed in 57.4% of patients. % VLFI was not correlated with AHI (R = 0.12). Receiver operating characteristic curves constructed using various AHI cut-offs (5 to 30/h-1) failed to identify a % VLFI cut-off associated with SAS. The 2.4% VLFI cut-off recommended for the general population of patients with suspected SAS had low values for specificity (35%), positive and negative predictive value (35% and 54% respectively). Heart rate increment analysis demonstrated several limitations in CHF. At this time, HRV methodology could not be proposed as the first step of SAS screening in CHF population.

H014

PDE V POLYMORPHISM MODULATE NO-INHALED RESPONSE

T. DAMY  1, P.-F. LESAUT  1, L. TU  1, S. ADNOT  1, J.-L. DUBOIS-RANDE  1, S. EDDAHIBI  1, L. HITTINGER  1

1 Inserm U841-Hôpital Henri Mondor, Créteil, France

Pulmonary Hypertension (PAH) is frequent in patient with advanced Heart Failure (HF). One of the underlying mechanisms of this pathogenic pathway could be a reduced ability of the arteriolar vascular smooth muscle to relax. The cGMP plays an important role in the regulation of pulmonary vascular tone and its clearance is dependent of phosphodiesterase 5 (PDE5). The aim of this study was to investigate whether or not the PDE5G(-1142)T genotype was associated with PAH and could affect the NO-inhaled response in HF.

Methods — HF patients underwent VO2, echocardiography 24h breath before the right side cardiac catheterization. Hemodynamic parameters, cGMP plasma level (n=12) were made after Air and 20ppm NO gas breathing. The PCR-restriction fragment length polymorphism method was used to determine the PDE5G/T gene.

Subjects: We included 72 HF patients. Most of the patients had an ischemic cardiomyopathy (54%). The mean LVEF was 29±1 and age was 53±1. The mean pulmonary artery pressure was 25.5±1.3mmHg. The genotype TT, GT, GG and G(-1142)T allele frequency distribution was respectively 38.9%, 41.7%, 19.4% and 40.3%.

Results — Baseline characteristics were similar between the three genotypes except for the cGMP which was significantly decrease in TT genotype versus GG-GT group (p=0.02). Furthermore the pulmonary capillary wedge pressure (PCWP) trend to decrease in TT genotype (p=0.09). These results suggested that PDE 5 activity increase in patient with TT genotype and may increase the pulmonary artery tone. NO-inhaled PVR was significantly different between the three subgroups (Anova for repeated measures: p=0.002) and was more important in the TT group with a decrease of PVR of 33 % versus 1.6% in GG and 0% in GT subgroups. The PVR decrease is explained by a superior increase in PCWP in TT genotype after NO inhalation. Our hypothesis is that the greatest NO-inhaled response in TT genotype is due to a greater degree of pulmonary vasoconstriction tone at baseline.

Conclusion — We first demonstrate that G(-1142) polymorphism of PDE 5 is functional and may regulate the basal pulmonary artery tone at baseline and increase the NO-inhaled response in Heart Failure.