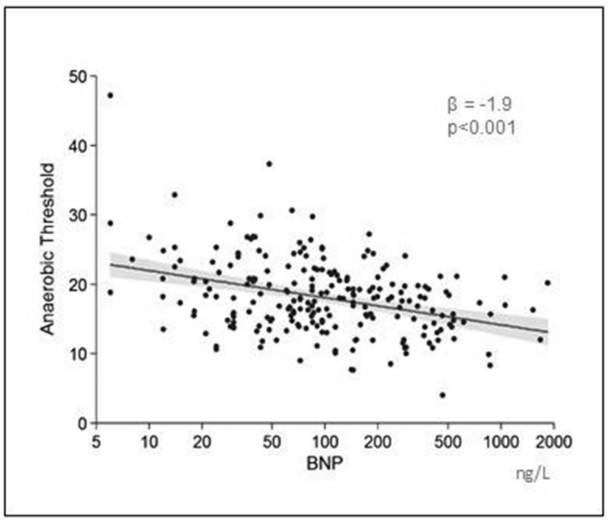
indexed RV end-diastolic volume and body weight, whilst BNP was independently associated with VE/VCO2 (β^2=0.8; 95% CI 0.1 – 1.6; p<0.001), and to a lesser extent, anaerobic threshold (β^2=−1.4; 95% CI −2.2 – −0.6; p=0.001).

Conclusion In this cohort, BNP is independently associated with VE/VCO2, a marker of ventilation-perfusion mismatch, which is known to provide a more reliable assessment of exercise intolerance irrespective of effort level. These results therefore support a role for BNP as an useful marker of submaximal exercise capacity and for consideration in guiding prognosis.



Abstract 127 Figure 2 Association between BNP and anaerobic threshold n HCM

128 GENOMIC INSIGHTS OF ECG STRAIN PATTEN IN AORTIC STENOSIS: T WAVE INVERSION AND ST-SEGMENT DEPRESSION ARE UNDERLINED BY DIFFERENT MOLECULAR PATHWAYS

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Background ECG strain pattern is frequently presented in AS patients as a marker of LV hypertrophy and is emerging as one of the predictors for AVR. Our previous study had demonstrated that the normalisation of ST depression occurred within first 24 hours after AVR for AS while that of inverted T wave took 6–12 months in parallel to the time course of LVH regression. We hereby studied myocardium gene expression profiling and tested the hypothesis that different molecular mechanisms could have underlined the ST depression and T wave inversion.

Material and methods We studied 17 AS patients with age 73 ±8.5 years and 12 males. LV biopsy was taken during AVR. Myocardial gene expression profiling was studied using Stanford Human Exonic Evidence Based Oligonucleotide (HEEBO) array. T wave inversion was defined by mean voltage of T wave in ECG lead I, aVL, V5 and V6 below −50uV (T-invert, n=8; T-norm, n=9). ST depression was defined by a mean value of ST in the same leads being below –50 uV (ST-dep, n=9; ST-norm, n=8). Differential gene expression profiling within ST and T wave sub-group were carried out by parametric permutative (permutation times=1000) t-test using the p-value cut-off 0.01 respectively. Further gene functional annotation clusters were performed using Database for Annotation, Visualisation and Integrated Discovery (DAVID, NIH) and pathway analysis of GeneSpring GX10.

Results We identified 588 differentially expressed genes (DEGs) between the T-invert and T-norm with 308 genes up-regulated and 280 genes down-regulated in T-invert. Using Direct Interaction Network, genes with more intensive interactions were PLCL1, FGF1, MGP, FABP4, IL31 and IFI27 in up regulation and EGF, PAK2 SYT1, PPA1 and CSH1 in down-regulation, in T-invert. Between ST-dep and ST-normal, we identified 202 DEGs, with 95 up and 107 down regulated in ST-dep. Using Direct Interaction Network analysis, we identified interactive connexion of genes, including ARHGEF1, ARRB1, PPM1A, CD28, SNX9, PRSS27, ASXL1, HDAC9 and BCOR, with no centre nodes orientation. The Significantly Enriched Core Pathways Analysis and DAVID gene functional annotation clustering further revealed that the top pathways underlined the genes of T-invert were oxidative phosphorylation, mitochondria, cell cycle/mitosis, cell adhesion, EGF/ERBB pathway, fatty acid metabolism; whilst for the genes of ST-dep, the main pathways were G protein signalling pathway, transcription regulation of adipocyte differentiation and membrane trafficking/endocytosis.

Conclusions Our study provides new molecular insights into ECG strain pattern in LV hypertrophy due to AS. T wave inversion appears to be involved in down regulation of energy metabolism and cell cycle but ST-depression is mainly associated with changes in signalling transduction. Understanding these different biological significance will help a more precise clinical interpretation of ECG strain pattern and its changes following AVR for AS.

129 ASSOCIATION OF ADVERSE VENTRICULAR REMODELLING AND GENDER IN AORTIC STENOSIS

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Introduction Previous echocardiography studies have demonstrated smaller left ventricles(LV) with more hypertrophy and concentric remodelling in females with Aortic Stenosis (AS) compared to males. However, more recent cardiac magnetic resonance(CMR) studies have shown lower LV mass and mass/volume in females. We utilised CMR to assess myocardial perfusion and tissue characterisation, in addition to remodelling and function, and looked at biomarkers of fibrosis (Syndecan-4 and MMP-3) in male and female patients.

Methods Subjects with asymptomatic moderate to severe AS (2 of: aortic valve area <1.5 cm^2, peak gradient >36 mm Hg or mean gradient >25 mm Hg) were recruited in this prospective, multi-centre, observational study. All patients underwent venepuncture, echocardiogram and a comprehensive stress CMR.

Results 174 patients (133 male) were recruited. Females were slightly younger but there was no difference in resting haemodynamics, co-morbidities or AS severity between the genders (Table 1). Male patients had significantly higher LV volumes