and mass, with more concentric remodelling, lower systolic (LV ejection fraction, longitudinal and circumferential global peak systolic strain) and diastolic function (longitudinal and circumferential peak early diastolic strain rates) than females. Late gadolinium enhancement (LGE) was more prevalent (51.1% *vs*. 34.1%, p=0.038) and extent of LGE was also higher in males, as was Syndecan-4 and MMP-3 levels. Extracellular volume (ECV) was marginally higher in female patients. Stress and rest myocardial blood flow (MBF) were significantly lower in males, with no difference in myocardial perfusion reserve.

**Conclusions** Male patients with AS have more concentric remodelling, worse cardiac function and more fibrosis than females, with biomarkers associated with fibrosis being significantly higher as well, for a similar degree of AS.

**Abstract 129 Table 1**

| | Male (n=133) | Female (n=41) | p=value |
|---|---|---|---|
| Age (years) | 67.3±12.64 | 62.9±15.08 | 0.042* |
| Mean PG (mmHg) | 34.5±12.05 | 38.0±13.66 | 0.491 |
| AVAI ($cm^2/m^2$) | 0.58±0.14 | 0.55±0.15 | 0.595 |
| LVEDVI ($ml/m^2$) | 90.00±18.67 | 79.74±14.50 | <0.001* |
| LVEF (%) | 55.9±4.84 | 59.2±4.49 | <0.001* |
| LVMI ($g/m^2$) | 60.54±13.70 | 48.45±9.74 | <0.001* |
| LV mass/volume (g/ml) | 0.68±0.11 | 0.61±0.11 | <0.001* |
| $PSS_L$ (%) | −17.85±2.80 | −20.52±2.81 | <0.001* |
| $PSS_C$ (%) | −27.64±4.83 | −29.56±3.74 | 0.002* |

**Abstract 129 Table 1**

| | Male (n=133) | Female (n=41) | p-value |
|---|---|---|---|
| Stress MBF (ml/min/g) | 2.09±0.66 | 2.39±0.80 | 0.009* |
| Rest MBF (ml/min/g) | 0.93±0.21 | 1.14±0.36 | <0.001* |
| Global MPR | 2.29±0.70 | 2.18±0.70 | 0.449 |
| % LGE (%) | 4.6±3.87 | 2.9±3.06 | 0.001* |
| ECV (%) | 24.57±2.54 | 25.64±1.85 | 0.007* |
| Log10.Syndecan-4 (pg/ml) | 2.33 [IQR 0.72] | 2.14 [IQR 2.39] | 0.044* |
| Log10.MMP-3 (pg/ml) | 4.41 [IQR 0.88] | 4.24 [IQR 0.71] | 0.041* |

## 130 THE CONVERGENCE AND DIVERGENCE OF MOLECULAR PATHWAYS IN LV HYPERTROPHY DEFINED BY ECG VOLTAGE VERSUS LV MASS IN PATIENTS WITH AORTIC STENOSIS

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10.1136/heartjnl-2017-311726.129

**Background** Left ventricular hypertrophy (LVH) assessment by ECG voltage or image based mass volume is commonly used in clinical practice and research. Our previous study of LVH regression after AVR for AS demonstrated a more complete regression in ECG voltage than in LV mass, thus indicated that ECG and Echo could be quantifying different aspects of LVH. In this study, we used gene expression profiling to examine whether different molecular pathways are involved in LV hypertrophy defined by ECG voltage or Echo LV mass.

**Material and Methods** We studied 17 patients with aortic stenosis, aged 73±8.5 years with 12 males. Gene expression profiling of LV myocardium biopsy during AVR was carried out using Stanford Human Exonic Evidence Based Oligonucleotide (HEEBO) array. Using ECG QRS voltage cut-off 3.0 mv, 8 patients had LVH (ECG-LVH) and 9 had not (ECG-Norm ). Using Echo LV mass index 125 g/m2 for man and 105 g/m2 for woman, 9 cases had LVH (Echo-LVH) and 8 had not (Echo-Norm). The gene expression profiling comparisons were carried out between ECG-LVH vs. ECG-Norm as well as Echo-LVH vs. Echo-Norm, respectively, by parametric permutative (permutation times=1000) t-test using the p-value cut-off 0.01. Further gene functional annotation clusters were performed using Database for Annotation, Visualisation and Integrated Discovery (DAVID, NIH) and pathway analysed by GeneSpring GX10.

**Results** There are 163 genes significantly differentially expressed (DEGs) between ECG-LVH and ECG-Norm. Among which, 81 genes were up-regulated and 82 genes down-regulated in ECG-LVH. Between Echo-LVH and Echo-Norm, 256 DEGs were identified, of which 141 genes were up and 115 genes were down regulated in Echo-LVH. The Direct Interaction Network of 163 DEGs in ECG-LVH revealed several connected island hubs with genes of HGF, CDK5RAP2, FYN (up-regulated) and IL6, PTBP2 and IRF1 (down-regulated). Of 257 DEGs in Echo-LVH, there were more abundant island-like connexions. The hubs of the connexion include PTEN, GNL3, AREG, ADAM17 and PPP1CA (up-regulated) and DCTN6, TWIST1 and ITGAM (down regulated). Further data mining and analysis of the two DEGs lists using DAVID and the Significantly Enriched Core Pathways analysis has demonstrated that the DEGs of ECG-LVH and ECHO-LVH have similar regulation directions in pathways/annotation clustering, including transcription regulation, nucleotide binding, zinc finger, focal adhesion. In addition, ECG-LVH has down regulation in wounding healing/inflammatory/cytokines pathway indicating a lower interstitial fibrosis activity compared with ECG-Norm; In Echo-LVH, mitochondrial protein importing, metalloprotease/ECM remodelling and cell division pathways were up-regulated compared to Echo-Norm.

**Conclusions** Cardiac hypertrophy diagnosed by ECG voltage or Echo muscle mass share many common molecular pathways that have been implicated in LVH. The unique pathways associated with ECG voltage based LVH imply a predominant hypertrophy of myocytes; in contrast, Echo mass based LVH involves the unique pathways of myocardial energy and ECM remodelling. The convergence and divergence of underlying molecular pathways between ECG based and Echo based LVH suggest the two methods may provide complimentary clinical value in the assessment of LVH and its response to treatment.

## 131 LEFT VENTRICULAR INDEXED MASS ASSOCIATED WITH VENTRICULAR ARRHYTHMIAS IN PATIENTS WITH HYPERTROPHIC CARDIOMYOPATHY – A TERTIARY CENTRE MRI REGISTRY

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10.1136/heartjnl-2017-311726.130