phenomenon in asymptomatic white individuals without a relevant family history. Conversely, ATWI beyond V2 is rare, particularly in males and may warrant investigation. The results of this study will have a significant impact on ECG interpretation in young white athletes and non-athletes.

BCS Affiliates — Allied Professionals (BACPR/BANCC)

150 THE INCIDENCE OF LONG QTc IN ACUTE MEDICAL ADMISSIONS: AN EVALUATION OF FIVE METHODS

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Aim Long QTc is associated with poor outcomes on CCU and acute stroke units. Its incidence and outcome in unselected medical admissions is unknown. A range of drug classes and electrolyte abnormalities may lead to acquired long QTc. Given the frequency with which these are encountered in medical admissions, the incidence of long QTc requires study. We have calculated the incidence of long QTc encountered in acute medical admissions. As inaccuracies in the correction formula may give misleading data, this study compared five formulas commonly used to correct QT interval duration by heart rate (HR): Bazett (B), Fridericia (Fa), Framingham (Fm), Hodges (H), Van der Water's (VdW) as well as the automated QTc correction provided by the Welch Allyn (WA) ECG recorder.

Methods 1000 records of consecutive patients admitted to acute medical wards at a tertiary teaching hospital were reviewed by two physicians. Admission ECGs were examined. QTc was calculated using the five formulas specified. The Pearson correlation coefficient (R^2) was used to estimate the relationship between QTc and HR. Patient age and gender were recorded. QTc of ≥ 500 ms was considered significantly prolonged in both genders. ECGs with missing patient identifiers or poor trace quality were excluded. Cohen's kappa statistic, for inter-observer agreement, was made from a subgroup of 200 randomly chosen ECGs evaluated by a third observer.

Results 715 ECGs were included. 356 males, 359 females. Age 61 ± 20 years (mean $\pm$ SD). Kappa inter-observer agreement was 0.65 indicating substantial agreement. Incidence of QTc ≥ 500 ms was 1.7% (B), 0.4% (Fa), 0.4% (Fm), 0.3% (H), 0.4% (VdW) but was 6.2% with WA. Pearson correlation between QTc and HR was 0.02 (B), 0.06 (Fa), 0.5 (Fm), 0.001 (H), 0.5 (VdW) 0.009 (WA).

Conclusion The incidence of significant QT prolongation in unselected medical admissions varied by a factor of ten depending on the method used. Automated QTc software gave a far higher incidence of long QTc than manual interpretation. Framingham and Van der Water's equations show bias by HR. The clinical sequelae of long QT in unselected medical admissions needs further study.

Valve Disease/Pericardial Disease/ Cardiomyopathy

151 CARDIAC MYOSIN-BINDING PROTEIN C IS A NOVEL MARKER OF MYOCARDIAL INJURY AND FIBROSIS IN PATIENTS WITH AORTIC STENOSIS

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Background Cardiac myosin binding protein C (cMyC) is an abundant sarcomeric protein and a novel highly specific marker of myocardial injury. Given myocyte death characterises the transition from hypertrophy to replacement myocardial fibrosis in advanced aortic stenosis, we hypothesised that serum cMyC concentrations would be associated with cardiac structure and outcomes in patients with aortic stenosis.

Methods cMyC was measured in two cohorts: a mechanism cohort of patients with aortic stenosis ($n = 161$) and healthy controls ($n = 46$) who underwent cardiac magnetic resonance imaging, and an outcomes cohort with aortic stenosis ($n = 104$) followed for a median of 11.3 years. True cut myocardial biopsies were obtained from 10 patients in the mechanism

Abstract 151 Figure 1 Linear regression correlation of cMyC with indexed LV mass and fibrosis volume in aortic stenosis

Abstract 151 Figure 2 Oncosis and autophagy in biopsy samples from patients with low and high cMyC concentrations, visualised using a 3,3'-diaminobenzidine staining. Plot shows correlation between cMyC and total myocyte death rate across 10 patients tested

cohort who underwent aortic valve replacement. These samples underwent staining and analysis for myocyte death.

Results In the mechanism cohort, cMyC concentration correlated with left ventricular mass ($r = 0.59$, 95% CI 0.47–0.68, $p < 0.0001$), fibrosis volume ($r = 0.57$, 95% CI 0.45–0.67, $p < 0.0001$) and extracellular volume ($r = 0.28$, 95% CI 0.12–0.42, $p = 0.0004$) in patients with aortic stenosis but not in controls (Figure 1). In those with late gadolinium enhancement (LGE), cMyC was higher (32 [21–56] ng/L vs 17 [12–24] ng/L without LGE, $p < 0.001$). cMyC was unrelated to aortic transvalvular gradient or objective assessment of coronary disease. Unadjusted Cox proportional hazards analysis

in the outcomes cohort showed greater all-cause mortality (HR 1.53 per doubling of cMyC, 95% CI 1.13–2.06, $p = 0.006$). In exploratory analyses, a relationship was observed between cMyC and the rate of myocyte death (expressed as the sum of apoptosis, oncosis and autophagy counts) in biopsy samples ($r = 0.67$, 95% CI 0.08–0.92, $p = 0.03$). Clear differences were observed in staining patterns for oncosis and autophagy between subjects with low and high cMyC concentrations (Figure 2).

Conclusion Serum cMyC concentration is associated with myocardial hypertrophy, fibrosis and an increased risk of mortality in aortic stenosis. The quantification of serum sarcomeric protein concentrations have major potential to provide objective measures of disease progression and to guide clinical decisions in patients with aortic stenosis.

152 EXPLORATORY ASSESMENT OF SIMULTANEOUS LEFT VENTRICULAR STRAIN AND VOLUME IN CHRONIC SEVERE AORTIC VALVE DISEASE

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Introduction Aortic valve regurgitation (AR) and stenosis (AS) increase the workload of the left ventricle (LV), resulting in remodeling of chamber volume and walls that normalise wall stress. Whether functional characteristics of the heart, such as strain (ϵ), adapt as the heart remodels is unclear. Likewise, we know little about the relationship between changes in volume and strain across the cardiac cycle. This exploratory study utilised a novel technique (ϵ -volume loops) to establish the contribution of longitudinal ($L\epsilon$) and transverse strain ($T\epsilon$) to volume change throughout the cardiac cycle in patients with severe aortic valve disease.

Methods 27 participants with preserved ejection fraction (EF) were included consisting of AR ($n = 7$), AS ($n = 10$) and healthy controls ($n = 10$). Standard transthoracic echocardiography was used to obtain an apical 4 chamber image of the LV. Temporal $L\epsilon$ and $T\epsilon$ values were exported and divided into 5%-time increments. LV volumes were traced at each 5%-time increment. The ϵ -volume relationship was assessed by 1) the slope of the linear regression line in systole (Sslope) and diastole (Dslope), 2) polynomial equations to derive

Abstract 152 Figure 1 A schematic view of the methods used to assess the ϵ -volume loops. Left: a curve of a typical control ϵ -volume loop. Right: a curve of the ϵ -volume loop of a patient with AR