but not sensitive for identifying mutation carriers (Abstract 073 table 1). Lateral annular late diastolic velocities were increased in group 2 but all other tissue Doppler indices were similar between the two groups (Abstract 073 table 2). The proposed cut-off values (from previous studies) were tested in our larger study and found to be clinically unhelpful; lateral Ea velocity <14 (sensitivity 40%, specificity 50%), lateral Sa velocity <13 (sensitivity 50%, specificity 50%), septal Sa <12 (sensitivity 100%, specificity 0%) and septal Ea <13 (sensitivity 70%, specificity 30%).

Abstract 073 Table 1

	Group 1 (mutation negative)	Group 2 (unaffected mutation carriers)	p Value
Sa lateral annulus (cm/s)	9.8±2.6	10.1±2.4	0.60
Ea lateral annulus (cm/s)	13.4±4.9	13.3±3.9	0.97
Aa lateral annulus (cm/s)	7.5±1.9	8.9±2.3	0.005
E/Ea ratio lateral annulus	6.0±1.8	5.8±1.2	0.62
Isovolumic relaxation time (lateral annulus, ms)	75.8±17.5	81.3±16.3	0.17
Sa septum (cm/s)	8.1±1.4	7.9±1.6	0.72
Ea septum (cm/s)	10.1±2.7	9.0±2.8	0.33
Aa septum (cm/s)	8.1±2.1	7.3±1.3	0.25
Isovolumic relaxation time (septum, ms)	92.0±21.1	92.5±13.8	0.95

Abstract 073 Table 2

	Sensitivity	Specificity
Sokolow-Lyon (%)	3	95
Cornell (%)	18	95
Romhilt-Estes ≥ 4 (%)	8	95
Abnormal T wave inversion (%)	21	93

Conclusions In previous small studies, tissue Doppler indices have been reported to be both sensitive and specific for identifying unaffected mutation carriers. However in our larger study, tissue Doppler imaging was unhelpful in predicting genotype and with current imaging techniques it is not possible to reliably identify unaffected mutation carriers before the development of ventricular hypertrophy.

074 THE PREVALENCE AND SIGNIFICANCE OF LEFT VENTRICULAR HYPERTRABECULATION IN HIGHLY TRAINED ATHLETES

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Introduction Left ventricular non-compaction (LVNC) cardiomyopathy is characterised by increased myocardial trabeculation and recesses. Clinical manifestations of the disorder include progressive left ventricular dilatation, systolic impairment, predilection to fatal arrhythmias and thrombo-embolic events. Studies in heart failure patients demonstrate a high prevalence (up to 30%) of myocardial

trabeculations irrespective of the criterion used, and raise the potential diagnosis of LVNC. Given the high prevalence compared with other primary cardiomyopathies, it is unclear whether the myocardial morphology is representative of LVNC or merely an epiphenomenon associated with increased cardiac preload. The large cardiac preload associated with regular participation of intensive exercise results in physiological cardiac remodelling including increased left ventricular wall thickness and cavity size. Isolated case findings have also revealed increased trabeculations in some athletes but the significance of the anomaly is unclear. The distinction between cardiac remodelling from athletic training and LVNC is important when one considers that primary cardiomyopathies are the most commonly implicated cause of exercise related sudden cardiac death in young athletes. The aim of this study was to identify the prevalence and significance of hypertrabeculation in highly trained young athletes.

Method Between 2003 and 2011, 1146 athletes, aged 14–35 years, underwent 12-lead ECG and echocardiography. Echocardiograms were analysed in accordance with ASE guidelines and hypertrabeculation was defined as >3 localised protrusions of the ventricular wall >3 mm in thickness associated with intertrabecular recesses. Results were compared with 415 healthy controls of similar age.

Results Athletes displayed a higher prevalence of LV HTC compared with controls (18.3% vs 9.0%; $p<0.0001$). Of the athletes, 10.1% fulfilled conventional criteria for LVNC. African/Afro-Caribbean athletes exhibited a higher prevalence of LV HTC compared with Caucasians (28.8% vs 16.3%; $p=0.002$). Left ventricular hypertrabeculation was associated with T-wave inversion and lower indices of systolic function, however, assessment with 48 h ECG, exercise stress test and cardiac MRI failed to identify broader features of LVNC phenotype. Follow-up during the ensuing 48.6 ± 14.6 months did not reveal adverse events.

Conclusion The high prevalence of LV HTC in athletes, particularly among African/Afro-Caribbean athletes, suggests that the morphological anomaly represents an ethnically determined physiological epiphenomenon secondary to increased cardiac preload and afterload. Associated marked repolarisation changes and lower LV fractional shortening cannot exclude a myocardial disorder in a small minority. Prolonged longitudinal follow-up in a larger cohort of athletes should identify the precise significance of LV HTC.

075 EFFECT OF AORTIC VALVE REPLACEMENT ON LV REMODELLING AND MATRIX METALLOPROTEINASES AND THEIR TISSUE INHIBITORS IN ISOLATED SEVERE AORTIC STENOSIS

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Introduction Surveillance of valvular heart disease accounts for a significant proportion of outpatient attendances in cardiology. A biomarker that correlates with disease severity and helps guide the optimum timing for intervention would be of great interest. NT-proBNP has been extensively examined in cardiac disease but focus has turned to enzymes that control collagen turnover, specifically matrix metalloproteinases (MMPs) and their tissue inhibitors (TIMPs). There is little data, other than from tissue samples, in aortic stenosis. We sought to examine the relationship between LV geometry and remodelling and serum MMPs and TIMPs in isolated severe aortic stenosis.

Methods 46 patients with isolated severe AS (peak velocity >4 m/s or mean pressure gradient >40 mm Hg or aortic valve area <1 cm²) without obstructive coronary artery disease (on angiography) were