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 cm^2) without obstructive coronary artery disease (on angiography) were

recruited. Investigations were performed on the same day once listed for AVR and 6 months following surgery; venous blood samples were drawn, centrifuged within 30 min and frozen at −80°C until assayed. MMP 2, 3 and 9 and TIMPs 1, 2 and 4 and additionally NT-proBNP were measured using similarly designed non-competitive immunoluminometric assays. All samples both pre- and post-operative were thawed and analysed at the same time to ensure consistency of assays. LV geometry, function and remodelling were assessed with transthoracic echocardiography for diastolic function and cardiac magnetic resonance for cardiac mass/volumes and myocardial fibrosis on late gadolinium enhancement.

Results Three patients did not attend follow-up. The remaining 43-paired data-sets were used for analysis. Data that were not normally distributed (including all biomarker data) were log transformed before analysis. Change in biomarker values between pre- and post-op were examined using paired t-tests and results are shown in Abstract 075 table 1. The relationship between baseline biomarkers and functional parameters (both at baseline and the ratio of change; pre/post-operative) were examined using bivariate correlations and are shown in Abstract 075 table 2. There are significant falls in MMP 2 and 9 and TIMPs 1 and 2 despite no significant change in NT-proBNP by six months post AVR.

Abstract 073 Table 1

Log10	Pre-operative	Post-operative	p Value
MMP2	1.56 (0.13)	1.49 (0.12)	<0.001
MMP3	1.43 (0.13)	1.44 (0.13)	0.45
MMP9	2.19 (0.19)	2.11 (0.21)	0.03
TIMP1	2.53 (0.12)	2.50 (0.11)	<0.001
TIMP2	2.20 (0.08)	2.18 (0.07)	0.02
TIMP4	0.72 (0.29)	0.74 (0.29)	0.20
NT-proBNP	2.20 (0.73)	2.23 (0.56)	0.70

Abstract 075 Table 2

	Pre-operative	Ratio of pre-operative/ post-operative
MMP2	Sep E/e' e' r=0.4, p=0.006; RVMI r=0.37, p=0.01	RVMI r=0.46, p=0.003
MMP3	Sep E/e' e' r=0.31, p=0.04	Sep E/e' e' r=0.38, p= 0.01; LAVI r=−0.34, p=0.03; RVMI; r=0.33, p=0.03
MMP9	Nil	Nil
TIMP1	Nil	LAVI r=−0.31, p=0.047
TIMP2	Sep E/e' e' r=0.44, p=0.002; LAVI r=0.3, p=0.047	RVMI r=0.43, p=0.006
TIMP4	Nil	LAVI r=−0.57, p<0.00; RVMI r=0.47, p=0.002
NT-proBNP	Sep E/e' e' r=0.44, p=<0.001; LVMI r=0.44, p=0.002; LVEDVI r=0.32, p=0.03; LAVI r=0.29, p=0.048	LVEF r=−0.35, p=0.03

Discussion NT-proBNP is perhaps unsurprisingly associated with mainly left ventricular geometry. In contrast the MMPs and TIMPs seem to be more closely related to measures of diastolic function, namely septal E/e' (a measure of LVEDP) and left atrial volume along with right ventricular mass. The role of MMPs and TIMPs as biomarkers in aortic stenosis is of interest and warrants study in a larger patient cohort including asymptomatic patients under surveillance.

076 FAILURE OF AORTIC VALVE REPLACEMENT TO IMPROVE OBJECTIVE FUNCTIONAL CAPACITY IN PATIENTS WITH ISOLATED SEVERE AORTIC STENOSIS

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Introduction Aortic stenosis (AS) is the commonest valve disease requiring surgery in the developed world. Development of symptoms is a Class 1 indication for aortic valve replacement (AVR) which reduces mortality. However, regression of left ventricular hypertrophy may be incomplete, myocardial fibrosis and diastolic dysfunction may be irreversible which may limit the functional improvement seen following AVR. We sought to examine the impact of AVR on cardiac remodelling and objective improvements in functional capacity.

Methods 46 patients with isolated severe AS (peak velocity >4 m/s or mean pressure gradient (MPG) >40 mm Hg or aortic valve area (AVA) <1 cm^2) without obstructive coronary artery disease (on angiography) were recruited. Investigations were performed on the same day once listed for AVR and 6 months following surgery; transthoracic echocardiography (TTE) for stenosis severity, diastolic function; cardiac magnetic resonance (CMR) for ventricular geometry, left atrial volumes, myocardial fibrosis on late gadolinium enhancement (LGE), adenosine stress/rest perfusion for absolute quantification of myocardial blood flow and calculation of myocardial perfusion reserve (MPR); cardiopulmonary exercise testing (CPEX) for objective exercise capacity (peak VO_2); venepuncture for NT-proBNP.

Results 46 patients were studied pre-AVR. Baseline characteristics; mean age 66±9 years, 74% male, peak velocity 4.4±0.57 m/s, MPG 49±14 mm Hg and AVA 0.86±0.22 cm^2. 43 patients returned for follow-up, of these 2 did not have CMR due to permanent pacemaker implantation and three had CMR without gadolinium (renal impairment/refused/extravasation). Non-normal data were log-transformed before analysis, paired t-tests were used for continuous variables, χ^2/McNemar tests for categorical. 42/43 had a biological prosthesis. Comparison between pre- and post-operative values; Abstract 076 tables 1 and 2. There were significant improvements in symptomatic status and reductions in LVMI, LVEDVI and left atrial volume index (LAVI) but this was not associated with improvement in objective exercise capacity (or NT-proBNP), significant reduction in E/e' or increase in MPR.

Discussion The majority of patients had minimal or no symptoms at the time of AVR. As expected there is a significant fall in LV mass and volumes by 6 months. The fall in left atrial volumes likely reflects the fall in left atrial pressure (and LVEDP consistent with a reduced, albeit not significantly, E/e'). Despite an improvement in NYHA Class there is no significant improvement in objective exercise capacity (peak VO_2) at 6 months. Over half the patients have

Abstract 076 Table 1

	Pre-operative	Post-operative	p Value
Septal E/e'	13.7 [11.1−18.6]	12.5 [10.6−17.1]	0.76
LVMI (g/m^2)	71.5 (17.5)	60.5 (16.1)	<0.001*
LVEDVI (ml/m^2)	97.4 (15.2)	80.6 (13.3)	<0.001*
LVEF (%)	56.4 (6.2)	57.8 (5.6)	0.15
LAVI (ml/m^2)	59.6 [49.2−66.4]	51.5 [45.6−59.9]	<0.001*
RVEDVI (ml/m^2)	80.1 (10.8)	79.6 (15.7)	0.79
RVEF (%)	61.6 (6.8)	57.7 (7.5)	<0.001*
LGE present	26 (57%)	24 (63%)	0.62
MPR	2.05 (0.51)	2.29 (0.60)	0.06