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	LVT-positive	LVT-negative		
Age (years)	61 ± 10	67 ± 7*		
Male %	100 (8/8)	92 (33/36)		
Smoking %	25 (2/8)	8 (6/36)		
Diabetes %	38 (3/8)	33 (12/36)		
LVEF %	$37~\pm~14$	39 \pm 11		
LVEDVI (ml/m ²)	136 \pm 40	114 \pm 31		
LVESVI (ml/m ²)	$89~\pm~41$	$72~\pm~31$		
Warfarin	25 (2/8)	6 (2/36)		
EURO score	$7.9~\pm~4.6$	10.3 \pm 9		
	95% CI for exp b			
	B (SE)	Lower	exp B	Upper
Included				
Constant	5.8 (3.5)*		2510	
Age	-0.12 (0.06)*	0.760	0.892	0.978

LVEDVI, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESVI, left ventricular end-systolic volume; LVT, left ventricular thrombus.

contrast-enhanced cardiac magnetic resonance imaging (CE-CMR) has emerged as an important non-invasive tool for the assessment of heart failure and has the capability to diagnose LVT with greater ease than transthoracic echocardiography (TTE).

Aims: To examine the prevalence of LVT as detected by CE-CMR in a consecutive series of patients with chronic left ventricular dysfunction referred for coronary artery bypass surgery (CABG). To compare the rates of detection of LVT between CMR and TTE/ ventriculography in this population of patients.

Methods: 44 patients with impaired ventricular function (CMR ejection fraction <50%) scheduled for isolated elective CABG underwent conventional left ventricular assessment (TTE and left ventricular angiography) as part of the clinical preoperative assessment and CMR as part of a research protocol. CE-CMR images were acquired by a 1.5 Tesla Siemens Sonata scanner in three long axis planes using steady state free precession imaging (flip angle 60° TE/TR 1.6/700 TI 450 ms) or ultrafast gradient echo imaging (flip angle 25° TE/TR 4.3/750 TI 450 ms) following a bolus injection of Omniscan 0.1/mmol/kg body weight. The CMR images were analyzed for the presence of LVT by two blinded observers working in consensus. LVT was defined as a filling defect characteristically present at the apex, or associated with an area of wall motion abnormality that was seen in two image planes. Artefact was characterised by a filling defect seen within the wall of the myocardium (no-reflow phenomenon) or was visualised in only one plane.

Results: The overall mean age was 66 ± 8 years and 93% were men. Eight out of 44 patients (18%) had LVT identified by CE-CMR. Patients with LVT tended to have a lower ejection fraction and increased left ventricular volumes; however, none of these comparisons were statistically significant, see table. Patients with LVT were significantly younger (61 ± 10 years) than patients in whom thrombus was not identified (67 ± 7 years) p = 0.03, table. Logistic regression analysis demonstrated age to be the only predictor for LVT, with younger age indicating a higher risk. Conventional imaging identified the presence of LVT in only three patients, two by left ventricular angiography and one by TTE. Two patients with LVT identified by CE-CMR had sustained a stroke within 6 months of their CMR scan, patients A and C, fig.

Conclusions: LVT is difficult to diagnose by conventional imaging techniques and CE-CMR may play a role in the preoperative assessment of this group, both for viability assessment and LVT detection. LVT was more likely to be present in younger patients presenting with heart failure and factors that are associated with premature ischaemic heart disease may also influence the prevalence of LVT.



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203 LEFT AND RIGHT VENTRICULAR TISSUE VELOCITIES ARE REDUCED IN HYPERTROPHIC CARDIOMYOPATHY BUT NOT IN ATHLETE'S HEART

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Introduction: Left ventricular hypertrophy due to athlete's heart and hypertrophic cardiomyopathy (HCM) can be difficult to distinguish clinically and can affect both ventricles. Cine phase contrast velocity magnetic resonance imaging (tissue phase

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Abstract 203 Myocardial velocities adjusted for covariates. White, control; light grey, athlete's heart; dark grey, hypertrophic cardiomyopathy. LV, left ventricle; RV, right ventricle. * and # denote statistical significance for hypertrophic cardiomyopathy versus controls and hypertrophic cardiomyopathy versus athlete's heart, respectively, with the number of symbols representing the significance level p < 0.05, **/##p < 0.01, ***/##p < 0.001).

mapping; TPM) allows comprehensive assessment of myocardial velocities. Magnetic resonance imaging is superior to echocardiography in the assessment of right ventricular volumes and mass and may also be a powerful tool to assess right ventricular tissue velocities. We hypothesised that myocardial velocities would be altered in the left ventricle and also in the right ventricle in HCM, but not in athlete's heart.

Methods: 79 sedentary controls (36 \pm 12 years), 26 HCM patients (42 \pm 17 years) and 35 elite athletes (25 \pm 4 years) underwent cardiac phase contrast imaging using a black blood k-space

segmented gradient echo sequence for the analysis of myocardial velocities with high spatial resolution at 1.5 Tesla. Peak systolic and diastolic radial and longitudinal velocities in both the left and right ventricle were determined. Velocities were averages of a midventricular short axis slice with the exception of left ventricular longitudinal velocities, which are based on averages of basal short axis slices. Left and right ventricular masses and volumes were determined with standard steady state free precession cine imaging. A univariate general linear model with fixed effects for group and covariates, including age, heart rate, and appropriate end-diastolic left ventricular or right ventricular dimensions was used.

Results: Myocardial mass indexed to body surface area (g/m²) was significantly higher in HCM (80 ± 26 , p<0.001) and athletes (83 ± 17 , p<0.001) when compared with controls (61 ± 11) in the left ventricle, but the right ventricular mass index was only significantly increased in athletes (26 ± 4 versus controls: 21 ± 4 , p = 0.009). In HCM, but not in athletes, radial and longitudinal left ventricular peak diastolic velocities were significantly reduced when compared with controls (fig). There was a 27% lower right ventricular peak longitudinal diastolic velocity in HCM compared with controls (p<0.001), but systolic radial and longitudinal and diastolic radial right ventricular myocardial velocities were not different among the groups.

Conclusions: Left ventricular hypertrophy leads to reduced diastolic left ventricular myocardial velocities in HCM, but not athletes. TPM also allows an assessment of right ventricular myocardial velocities and in HCM the right ventricular relaxation pattern was abnormal even in the absence of significant right ventricular hypertrophy. TPM may help differentiate athlete's heart from HCM in clinically difficult cases.

204 DIAGNOSTIC YIELD IN FIRST DEGREE RELATIVES OF VICTIMS OF SUDDEN ADULT DEATH SYNDROME FOLLOWING SYSTEMATIC CLINICAL EVALUATION IN AN EXPERT SETTING

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Objectives: Sudden adult death syndrome (SADS) accounts for at least 4% of all sudden cardiac deaths in the United Kingdom. Most causal disorders are potentially inherited. However, routine genetic assessment of relatives of victims of SADS is not widely available in the United Kingdom. Given the genetic heterogeneity and incomplete understanding of conditions implicated in SADS, a negative genetic test cannot exclude a familial disorder. The aim of this study was to identify the prevalence of familial cardiac disorders in first degree relatives of SADS victims based on systematic, purely clinical evaluation.

Methods: Between March 2006 and September 2007, 22 families of victims of SADS were evaluated in a tertiary inherited cardiac clinic. All victims' hearts were examined by an expert cardiac pathologist and a structural cause was excluded. A total of 71 individuals underwent comprehensive cardiac clinical evaluation, consisting of 12-lead ECG, echocardiography, exercise stress testing, 24-hour Holter and, when applicable, an Ajmaline provocation test and/or cardiac magnetic resonance scan. All investigations were interpreted by a cardiac expert in SADS.

Results: All family members had a structurally normal heart. Of the 22 families, 14 (64%) had objective evidence of an ion channel. In particular, 11 (79%) had clinical evidence of the Brugada phenotype either on resting ECG or after ajmaline provocation and three (21%) demonstrated clinical manifestation of the long QT syndrome. A total of 23 of 71 (32%) asymptomatic family members were identified with an ion channel disorder.

Conclusions: Systematic clinical evaluation of families of victims of SADS in an expert setting is associated a high diagnostic yield (64%). In our series, ion channel disorders account for almost