expression *in vivo*, although there were significant inter-patient variations. There was a significant reduction in circulating T cells following CMR.
Conclusion CMR was not associated with DNA damage *in vivo*. γ-H2AX expression varied markedly between individuals, therefore small studies using γ-H2AX as a marker of DNA damage should be interpreted with caution. CMR was associated with a statistically significant reduction in viable leukocytes, although the clinical relevance of the magnitude is unclear. Further work is warranted to contextualise these findings and delineate their impact.

010 GENERATION OF A FORMULA FOR CAROTID-FEMORAL PATHLENGTH DETERMINATION FOR USE IN PWV ASSESSMENT

Liam Brown. *University of Dundee, UK*

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Background Aortic arterial stiffening is an independent predictor for future cardiovascular events. Calculation of Carotid-Femoral Pulse Wave Velocity (cfPWV) is currently the clinical gold standard for measuring arteriosclerosis, with cfPWV now recommended by the European Society of Cardiology for the guidance of initiating preventative treatments. Despite this, no consensus exists on how best to obtain the pathlength component of the calculation with large intercentre variation in how this is performed. The aim of the current study was to generate a calculation to produce a standardised pathlength that can be generated from easily obtainable clinical measures.
Methods 1183 participants free from cardiovascular disease (CVD) underwent whole body MRI as part of the TASC-FORCE study. The distance between the carotid and femoral vessels was obtained by tracing the arterial centreline between the two. Backward linear regression was then used to generate a formula for calculating pathlengths based on easily obtainable clinical metrics. This calculation was then validated in an external cohort of 128 individuals with and without CVD who had also undergone MRI.
Results Various allometric and cardiovascular values were included in the analysis. Carotid-femoral pathlength could be calculated as follows:

Distance=100.36+(0.70×Age[years]) + (137.89×Height[m]) + (0.52×Weight[kg]) − (0.17×Pulse) + (46.16[female], 54.32 [male]).

When compared with the actual measured distance in the original cohort this differed by −0.05 mm SD +28.5 mm, p=0.962 for difference. When this formula was then applied in the external validation cohort there was a small overestimation of the pathlength by 10.07±25 mm (p>0.001). This is comparable to clinically accepted techniques: measuring the direct distance from the carotid-femoral arteries and subtracting the measured to distance to the carotid-sternal notch, by tape measure or calliper on the body surface, results in mean overestimation of pathlength by −23.5±38 mm: a value greater than that of the generated formula technique.
Conclusion Using simple allometric measures, carotid-femoral pathlength can be calculated with good accuracy. This holds promise for improving interstudy and intercentre reproducibility, thus expanding the utility and applicability of PWV calculation in clinical practice. In future, the predictive ability of the formula can be tested in disease discrimination cohorts to further assess its clinical applicability

011 ADENOSINE STRESS T1 MAPPING: A NOVEL CONTRAST FREE METHOD TO ASSESS MYOCARDIAL PERFUSION AND ISCHAEMIA IN HYPERTROPHIC CARDIOMYOPATHY

[1]Betty Raman, [1]Rina Ariga, [1]Masliza Mahmod, [1]Stefan Piechnik, [1]Alessandra Borlotti, [3]Michael Jerosch-Herold, [1]Jane M Francis, [1]Erica Dall'Armellina, [1]Sanjay Sivalokanathan, [1]Vanessa Ferreira, [2]Hugh Watkins, [1]Stefan Neubauer. *[1]Oxford Centre for Clinical Magnetic Resonance Research, Division of Cardiovascular Medicine, UK; [2]Radcliffe Department of Medicine, University of Oxford, UK; [3]Cardiology, Brigham and Women's Hospital, UK*

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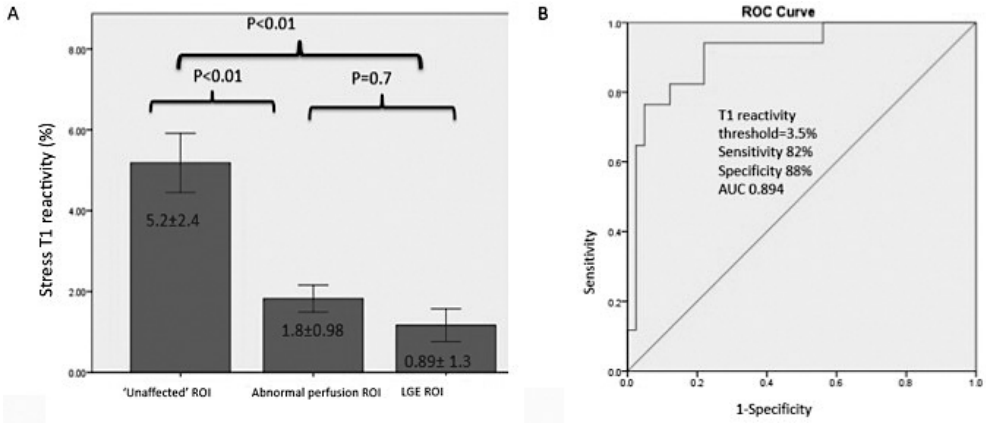
Aim The aim of this study was to assess the ability of stress T1 mapping to detect perfusion abnormalities and inducible ischaemia in hypertrophic cardiomyopathy (HCM).
Background Impaired perfusion reserve in HCM has been shown to be an independent predictor of adverse cardiovascular outcomes. CMR perfusion imaging currently requires the administration of gadolinium based contrast agents, which are contraindicated in allergy and renal failure, making non-contrast methods such as T1 mapping a safer and more affordable alternative. As adenosine stress T1 mapping has been shown to detect inducible ischaemia in patients with coronary disease, we hypothesised that stress T1 may be used to detect perfusion abnormalities in HCM with reasonable accuracy and T1 reactivity correlates with impaired myocardial blood flow (MBF) reserve and global longitudinal strain (GLS).
Method 62 subjects with no known history of coronary disease (31 controls and 31 HCM patients) underwent CMR at 3T including cine imaging, tagging, rest and stress (adenosine 140 mcg/kg/min) blood oxygen level dependent imaging (BOLD), T1 mapping (ShMOLLI), first-pass perfusion imaging and late gadolinium imaging (LGE). Rest and stress T1 values from mid ventricular slice were derived and T1 reactivity determined. [1,2] MBF was estimated using Fermi function deconvolution method as previous described.[3,4]
Results Baseline characteristics are listed in Table 1. T1 reactivity was significantly reduced in regions of interest (ROI)

Abstract 011 Table 1 Baseline characteristics of subjects including comparison of left ventricular indices on CMR.

Baseline Characteristics	Healthy controls	HCM	P value
Age (years)	45 ± 14	45 ± 13	0.8
Male gender [no (%)]	71 (17)	74 (18)	0.7
Rest pulse, bpm	59 ±10	58±11	0.4
Systolic BP	116±10	116±12	0.4
Hypertension	0	0	
Smoker, %(n)	19(6)	25(8)	0.54
Diabetes, %(n)	0 (0%)	0 (0%)	
Atrial fibrillation	0	0	
NYHA functional class	1	1.2±0.4	0.6
Beta blockers	0	42 (13)	
ARB/ACE inhibitor,%(n)	0	0	
LV Ejection Fraction (%)	68 ± 5	71 ± 13	0.09
LVEDV Index ml/m²	80±14	84±21	0.5
LVESV Index ml/m²	59 ±10	58±11	0.4
Septal thickness (mm)	9±2	15±6	<0.01
LV mass index (g/m²)	54±12	72±28	0.02
Presence of LGE, n(%)	0(0%)	21(67%)	
LGE fibrosis volume(%)	0(0%)	7.3±6.1	



Abstract 011 Figure 1 A. Comparison of T1 reactivity profiles for 'Unaffected' myocardium versus regions with abnormal perfusion versus LGE; B. ROC curve depicting sensitivity and specificity of stress T1 reactivity to predict perfusion deficits in HCM.

with LGE and perfusion defects compared to the remaining 'unaffected' myocardium (Figure 1). Using a diagnostic threshold of 3.5% on ROC analysis for detecting impaired MBF in HCM, T1 reactivity had a sensitivity and specificity of 82% and 88% respectively (AUC 0.894, p<0.01) (Figure 1). Adenosine induced rise in MBF, T1 reactivity, oxygenation and global longitudinal strain (GLS) were significantly reduced in HCM compared with controls (p<0.01 for all comparisons) Stress T1 reactivity moderately correlated with MBF reactivity (r=0.55, p<0.01) and GLS (r=−0.44, p<0.01).

Conclusions

1. Stress T1 mapping is capable of distinguishing regions with perfusion defects versus normal perfusion in HCM without the need of contrast.
2. Stress T1 reactivity, MBF reactivity, oxygenation and GLS were significantly reduced in HCM compared to controls.

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012 LONGITUDINAL CARDIAC MAGNETIC RESONANCE ASSESSMENT OF DIFFUSE AND REPLACEMENT MYOCARDIAL FIBROSIS IN AORTIC STENOSIS

RJ Everett, CWL Chin, J Kwiecinski, WJ Jenkins, S Mirsadraee, A White, SK Prasad, S Semple, DE Newby, MR Dweck.

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Objective To investigate whether cardiac magnetic resonance (CMR) can assess the progression and regression of myocardial fibrosis in patients with aortic stenosis (AS).

Methods Sixty-three patients with aortic stenosis were followed up for 2 years with serial CMR and echocardiography: 28 patients (63±13 years, 68% male, 50% asymptomatic severe AS) did not undergo intervention (*natural history cohort*) and 35 patients (67±8 years, 73% male, 83% symptomatic severe AS) underwent aortic valve replacement (AVR) within the first year (*AVR cohort*). Replacement and diffuse myocardial fibrosis were assessed using the late gadolinium enhancement (LGE) and T1 mapping techniques respectively. Annualised change was calculated for all measures.

Results In the natural history cohort, left ventricular mass index (LVMi) increased over time (6%±1%, p<0.0001) and was accompanied by a fall in longitudinal systolic function (−4%±2% p=0.03). The indexed extracellular volume (iECV, a measure of diffuse fibrosis) also increased over time (7% ±2%, p<0.0001). However, no changes were observed in native T1, post-contrast T1 or extracellular volume (ECV) fraction on serial imaging. Mid-wall LGE was observed in nine patients (32%). Absolute LGE mass increased by 3.8 ±0.8g (p<0.0001) amongst all the patients with baseline mid-wall LGE. Four patients with mid-wall LGE at baseline developed new areas of LGE in different myocardial segments. No patients without pre-existing mid-wall LGE developed new LGE during follow up.

The AVR cohort displayed a fall in LVMi following surgery (−15%±2%, p<0.0001) mirrored by a reduction in diffuse fibrosis (iECV, −9%±2%, p<0.0001). The ECV fraction however was observed to increase (5% [2, 11], p<0.0001), with no change in native T1. Mid-wall LGE was present in 9 patients (26%). No patient went on to develop new LGE nor did existing LGE resolve in any patient and LGE mass did not change. Longitudinal systolic function increased following AVR (16%±6%, p=0.01).

Conclusion Changes in diffuse fibrosis can be tracked using iECV and increase with time in patients with AS alongside LV mass and replacement fibrosis. Following AVR there is a fall in LV mass driven predominantly by regression of cellular