Results Echocardiographic global longitudinal strain (GLS) and extracellular volume (ECV) measured by CMR were the only variables able to independently stratify between the three groups of patients. ECV was the best technique for differentiation between hypertensive heart disease and HFPEF (AUC 0.88; GLS AUC 0.78, p<0.001 for both). Using ECV, an optimal cut-off of 31.2% gave 100% sensitivity and 75% specificity. ECV was significantly higher and GLS was significantly reduced in subjects with reduced exercise capacity (lower peak VO2 and higher VE/VCO2).

Conclusions Both GLS and ECV are able to independently discriminate between hypertensive heart disease and HFpEF and identify patients with prognostically significant functional limitation. ECV is the best diagnostic discriminatory marker of HFpEF and could be used as a surrogate end-point for therapeutic studies.

008 DEMONSTRATION OF CARDIAC AL AMYLOIDOSIS REGRESSION AFTER SUCCESFUL CHEMOTHERAPY. A CMR STUDY

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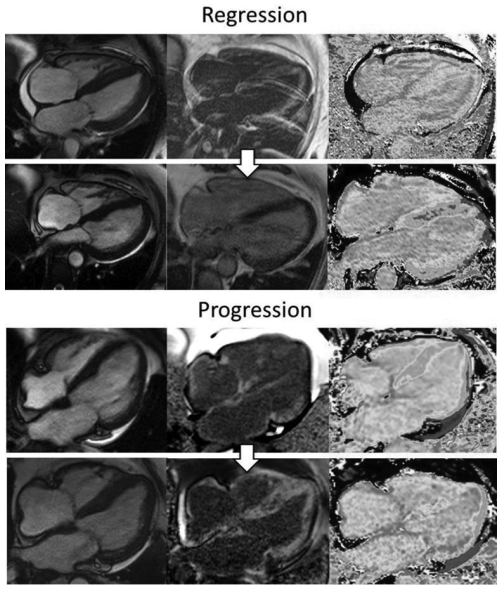
Background Cardiac involvement in immunoglobulin light chain (AL) amyloidosis is the major determinant of survival; Cardiac response to chemotherapy is conventionally assessed by serum brain natriuretic peptide (NT-proBNP) and echocardiography, but neither quantify amyloid burden. The aim of this study was to evaluate cardiac AL amyloid serially using cardiovascular MR (CMR) including extracellular volume measurement (ECV), which is the site of the amyloid deposits.

Methods 31 patients with cardiac AL amyloidosis who had chemotherapy were studied serially using ECG, echocardiography, ^{123}I-labelled serum amyloid P component (SAP) scintigraphy, NT-proBNP measurements and CMR with T1 mapping and ECV measurements (mean interval 20±11 months). Nineteen patients achieved a complete or very good partial haematological response (CR n=10; VGPR n=9). Twelve patients attained a partial response (PR) or no response (NR).

Results At follow-up (mean 20±11 months), the amyloid burden had decreased substantially in 6 of the 10 (60%) attaining a CR, 6 of the 9 (67%) in VGPR and 1 of the 8 (13%) in PR. Changes in the ECV consistent with regression of amyloid were concordant with the changes in native T1, reduction in amyloid volume and in 5 patients with changes in late gadolinium enhancement pattern (figure 1). Overall there was significant reduction in NT-proBNP concentration, LV mass, left atrial area and improvement in diastolic function in patients whose amyloid burden decreased. Regression of cardiac amyloid by CMR correlated with regression of amyloid in other organs measured by SAP scintigraphy.



Abstract 008 Figure 1 Top: four chamber SSFP cine images in disatole, corresponding late gadalinium anhancement (LGE) images and ECV mapping before and after chemotherapy in a patient who had regression of amyloid burden after chemotherapy. Bottom: four chamber SSFP cine images in disatole, corresponding LGE images and ECV mapping before and after chemotherapy in a patient who had progression of amyloid burden after chemotherapy.

009 THE EFFECT OF CARDIAC MAGNETIC RESONANCE ON HUMAN CIRCULATING LEUKOCYTES

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Aims Investigators have proposed that cardiovascular magnetic resonance (CMR) should have restrictions similar to those of ionising imaging techniques due to proposed alterations to leukocytes. We aimed to investigate the acute effect of CMR on leukocyte DNA integrity and cell viability *in vitro*, and in a large cohort of patients *in vivo*.

Methods and results *In vitro* study: Peripheral blood mononuclear cells (PBMC) were isolated from healthy volunteers and assessed: 1) immediately following PBMC isolation, 2) after standing on the benchside as a temperature and time control, 3) after a standard CMR scan. Histone H2AX phosphorylation (γ-H2AX), an indicator of DNA damage, and leukocyte counts were quantified using flow cytometry. *In vivo* study: Blood samples were taken from 64 consecutive consenting patients immediately before and after a standard clinical scan. Samples were analysed for T cell count and γ-H2AX expression.

CMR scanning was associated with a significant increase in leukocyte γ-H2AX expression, indicating DNA damage occurs. We also observed a trend towards a significant decrease in absolute leukocyte numbers *in vitro* following CMR. CMR was not associated with a significant change in γ-H2AX

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

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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

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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	