28 SEX- AND AGE-DIFFERENCES IN NATIVE T1 RELAXATION TIMES IN HEALTHY ADULTS AT 1.5 AND 3.0 TESLA

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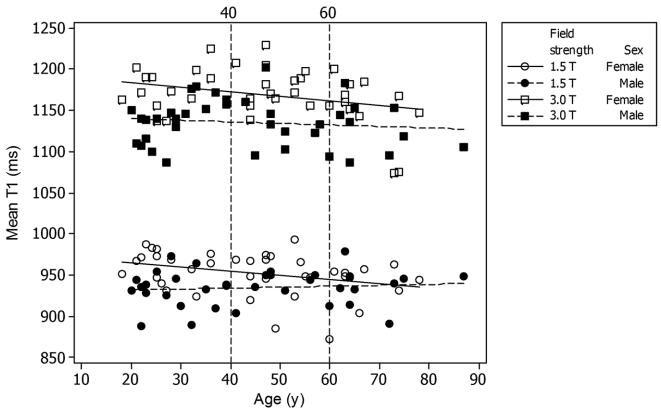
Background Limited information is available on sex differences in myocardial T1 relaxation times over age ranges. We used cardiac magnetic resonance (CMR) imaging at two field strengths to assess myocardial T1.

Methods Healthy adults underwent CMR at 1.5 Tesla (T) (Avanto) and 3.0 T (Verio). T1 maps were acquired in three short axis slices, using an optimised MOLLI investigational prototype sequence (Siemens Healthcare WIP 448). Global mean T1, in milliseconds (ms), was calculated from evaluable regions-of-interest using 16-segment model.

Results 84 volunteers (43 male) underwent scans 1.4 ± 1.4 days apart. Because of artefacts related to cardio-respiratory motion and susceptibility effects, 47 (3.9%) segments were excluded at 1.5 T and 81 (6.3%) segments at 3.0 T, with a preponderance occurring at the distal slice.

Age-related decrease in T1 was observed in females, whereas male T1 remained reasonably constant (Figure 1 and Table 1). At 1.5 T, amongst those <40 years T1 was higher for females (961.3 ± 19.3 ms) than males (932.0 ± 22.8 ms, p < 0.001), whereas there was no difference in those ≥60 years (937.2 ± 28.7 vs. 934.3 ± 24.3 ms, respectively, p = 0.807). Results were similar at 3.0 T; female T1 was higher at <40 years (1166.0 ± 41.7 vs. 1139.6 ± 26.1 ms, p = 0.044), but not at ≥60 years (1151.0 ± 39.3 vs. 1124.3 ± 31.6 ms, p = 0.087).



Abstract 28 Figure 1 Global myocardial T1 relaxation times.

Regression analysis shows that at 1.5 T average T1 decreases by 5.13 ms for each additional decade (p = 0.038). An identical trend was observed at 3.0 T, with regression coefficient −0.564 ms/year approaching statistical significance (p = 0.064).

Conclusions In healthy adults, sex difference in global myocardial mean T1 relaxation times are observed amongst younger. This pattern is consistent across CMR field strengths. Pre- vs. post-menopausal differences in myocardial structure and function of females may explain these differences and this possibility merits further assessment.

Abstract 28 Table 1 Global myocardial T1 relaxation times (mean ± SD, ms)

	1.5 T		3.0 T	
	Men	Women	Men	Women
Age <40, y (n = 36)	932.0 ± 22.8	961.3 ± 19.3	1139.6 ± 26.1	1166.0 ± 41.7
Age 40–59, y (n = 25)	939.8 ± 16.5	951.7 ± 27.6	1135.8 ± 31.9	1180.1 ± 24.4
Age ≥60, y (n = 23)	934.3 ± 24.3	937.2 ± 28.7	1124.3 ± 31.6	1151.0 ± 39.3

29 SYNTHETIC ECV – SIMPLIFYING ECV QUANTIFICATION BY DERIVING HAEMATOCRIT FROM T1 BLOOD

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Background Extracellular volume (ECV) quantification by cardiovascular magnetic resonance (CMR) measures the extracellular space. Current methodologies require blood haematocrit (Hct) correction, a barrier to easy clinical use. We hypothesised that the relationship between Hct and longitudinal relaxation time of blood ($T1_{blood}$) could be calibrated and used to generate a *synthetic* ECV without Hct.

Abstract 29 Table 1 Patient characteristics

	Derivation	Validation	*p-value*
Total	214	213	
Male	107	104	*0.8*
Age	60 ± 15	60 ± 15	*0.6*
BSA (m^2)	1.87 ± 0.23	1.87 ± 0.23	*0.8*
Healthy Volunteer	33	33	
Aortic Stenosis	62	61	
Cardiac Amyloidosis	37	37	
Hypertrophic Cardiomyopathy	34	34	
Anthracycline	48	48	
Cardiac			
EDVi (ml/m^2)	71 ± 21	70 ± 20	*0.9*
ESVi (ml/m^2)	25 ± 14	24 ± 13	*0.5*
LV mass index (g/m^2)	90 ± 35	92 ± 35	*0.5*
SVi (ml/m^2)	47 ± 12	47 ± 13	*0.7*
LVEF (%)	66 ± 12	67 ± 12	*0.7*
LAAi (cm^2/m^2)	14 ± 3	14 ± 5	*0.5*
RAAi (cm^2/m^2)	12 ± 4	12 ± 4	*0.9*
Clinical			
Hematocrit	0.40 ± 0.04	0.40 ± 0.04	*0.4*
Createnine	79 ± 24	78 ± 21	*0.9*
eGFR	80 ± 23	78 ± 22	*0.4*
Systolic BP (mmHg)	110 ± 44	108 ± 49	*0.7*
Diastolic BP (mmHg)	65 ± 28	62 ± 31	*0.5*
T1 Mapping			
SHMOLLI ECV (%)	33 ± 10	33 ± 11	*0.9*

BSA, body surface area; BP, blood pressure; ECV, extracellular volume fraction; EDVi, indexed end-diastolic volume; ESVi, indexed end-systolic volume; eGFR, estimated glomerular filtration rate; LAAi, indexed left atrial area; LVEF, left ventricular ejection fraction; RAAi, indexed right atrial area; ShMOLLI, Shortened Modified Look-Locker Inversion recovery; SVi, indexed stroke volume

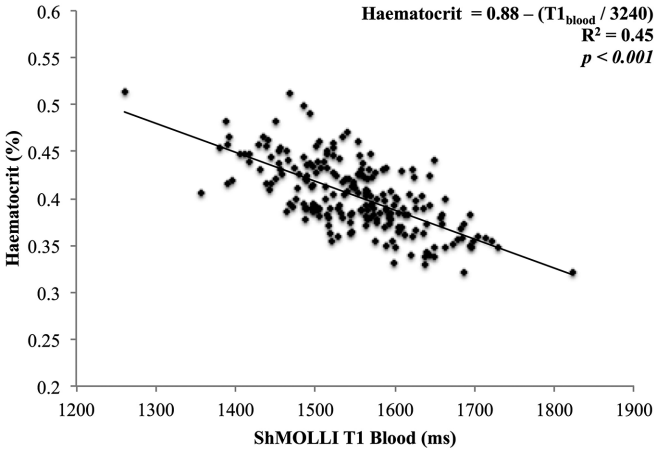
Methods 427 subjects with a wide range of health and disease were divided into derivation (n = 214) and validation (n = 213) cohorts (Table 1 for patient characteristics). All subjects underwent T1 mapping with ShMOLLI at 1.5 Tesla for ECV quantification. Venous blood for Hct was obtained prior to scanning with 44 patients having a repeat Hct within 6 h.

ECV was calculated as: ECV = ($\Delta[1/T1_{myo}]$ / $\Delta[1/T1_{blood}]$) * [1-haematocrit]).

Synthetic Hct was approximated from the linear relationship between Hct and native $T1_{blood}$, and used to calculate *synthetic* ECV. Histological validation was performed on 18 patients with severe aortic stenosis (age 71 ± 10 years, 78% male). ECV was compared with collagen volume fraction from intra-operative biopsies taken during surgical valve replacement.

Results In the derivation cohort, native $T1_{blood}$ and Hct showed a linear relationship (R^2=0.45; $p < 0.001$, Figure 1). This was used to derive *synthetic* Hct = 0.88 – ($T1_{blood}$/3240). *Synthetic* ECVcorrelated well with ECV (R^2 = 0.99; $p < 0.001$). These results were maintained in the validation cohort. Test:retest variability of haematocrit was higher than expected (n = 44, variability 10% with Hct:Hct R^2 = 0.86). On histological validation, *synthetic* and conventional ECV both correlated well with collagen volume fraction (R^2 = 0.61 and 0.69, $p < 0.001$).

Conclusion *Synthetic* ECV allows instantaneous non-invasive quantification of the myocardial extracellular space without blood sampling. Inline application of *synthetic* ECV may be an attractive alternative in clinical practice.



Abstract 29 Figure 1 Correlation between T1 blood and haematocrit. In the derivation cohort (n = 214), native T1 blood and hematocrit (Hct) showed a linear relationship (R2 = 0.45; p < 0.001). This was used to derive synthetic Hct = 0.88 – ($T1_{blood}$/3240).

30 WHOLE BODY CONTRAST ENHANCED MRA CAN QUANTIFY AND MONITOR ATHEROSCLEROSIS PROGRESSION

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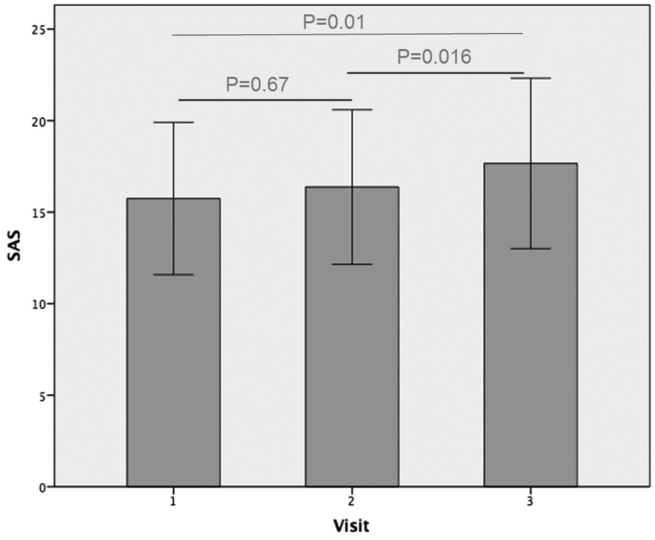
Aim To determine the ability of whole body magnetic resonance angiography (WB-MRA) to measure global atheroma burden progression.

Methods 50 consecutive patients with symptomatic peripheral arterial disease referred for clinical MRA were recruited. WB-MRA was performed at baseline, 6 months and 3 years. WB-MRA data was analysed by dividing the vasculature into 31 anatomical arterial segments. Each segment was scored according to degree of luminal narrowing: 0=normal, 1 = <50%, 2 = 50–70%, 3 = 71–99%, 4 = vessel occlusion. From this a standardised atheroma score (SAS) was calculated with a maximum score of 100 and minimum score of 0. Progression was assessed with repeat measure ANOVA.

Results 36 patients were scanned at 0 and 6 months, with 26 patients scanned at the three year follow up. Only those who completed all 3 visits were included in the final analysis. At 3 years, n = 18 demonstrated atheroma progression while n = 8 showed stable or improved disease. Those with no progression had significantly lower baseline SAS, and were more likely to be on statin therapy (p < 0.05 for both). Baseline SAS was 15.7 ± 10.3 at baseline with no progression at 6 months (SAS=16.4 ± 10.5, p = 0.67). At 3 years there was significant progression in atheroma (SAS = 17.7 ± 11.5, p = 0.01) (Figure 1). On multiple linear regression, age (β 0.14 p = 0.014), pulse pressure (β −0.12 p = 0.005) and ankle-brachial pressure index (β −7.7 p = 0.036) were independently associated with the rate of progression.



Abstract 30 Figure 1 Comparison of atheroma score at baseline, 6 months and 3 years. Visit 1 = Baseline, Visit 2 = 6months, Visit 3 = 3 years. T-bars represent 95% confidence intervals.

Conclusion Whole body contrast enhanced MRA can quantify and monitor atherosclerosis progression at 3 year follow-up even in a small cohort.

31 ISCHAEMIA AND VIABILITY ASSESSMENT WITH ADENOSINE STRESS CMR IN HIGH RISK PATIENTS: SAFETY, FEASIBILITY AND TOLERABILITY

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