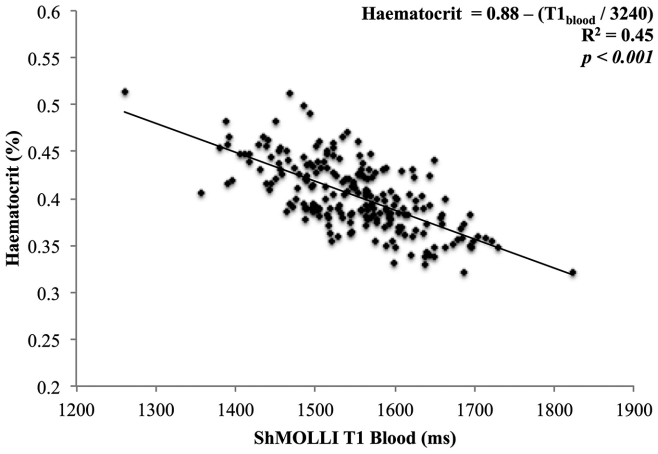
Methods 427 subjects with a wide range of health and disease were divided into derivation (n = 214) and validation (n = 213) cohorts (Table 1 for patient characteristics). All subjects underwent T1 mapping with ShMOLLI at 1.5 Tesla for ECV quantification. Venous blood for Hct was obtained prior to scanning 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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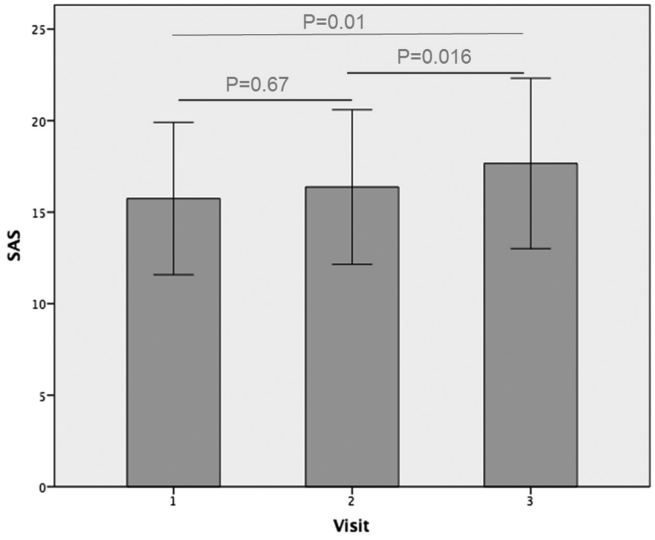
Aim To determine the ability of whole body magnetic resonance angiography (WB-MRA) to measure global atheroma burden progression.

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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