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 = (Δ[1/T1myo] / Δ[1/T1blood]) * [1-haematocrit]

Synthetic Hct was approximated from the linear relationship between Hct and native T1blood, 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 T1blood and Hct showed a linear relationship (R2=0.45; p < 0.001, Figure 1). This was used to derive synthetic Hct = 0.88 – (T1blood/3240). Synthetic ECV correlated well with ECV (R2 = 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 R2 = 0.86).

On histological validation, synthetic and conventional ECV both correlated well with collagen volume fraction (R2 = 0.61 and 0.69, p < 0.001).

Conclusion Synthetic ECV allows instantaneous non-invasive quantification of the myocardial extracellular space without blood sampling. In-line 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 – (T1blood/3240).

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 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.

Ischaemia and viability assessment with adenosine stress CMR in high risk patients: Safety, feasibility and tolerability

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