As expected, native T1 was higher at 3T than 1.5T (1213 ±57 versus 1050±48 ms, p<0.001); ECV% did not vary by scanner manufacturer, field strength or T1 mapping sequence (all p>0.30); therefore, only ECV-based measures were analysed.

ECV% correlated with increasing age, Society of Thoracic Surgeons Predicted Risk of Mortality score, known coronary artery disease, lower aortic valve peak velocity, increased LV mass, presence of late gadolinium enhancement (LGE) and reduced LVEF (p<0.05 for all). Following adjustment for demographic and clinical variables, ECV% remained associated with both LVEF (p<0.001) and mass index (p=0.043). Similar associations were seen with iECV.

A progressive increase in all-cause mortality was seen across tertiles of ECV% (14.0, 28.5 and 53.7 deaths per 1000 patient-years; log-rank test, p=0.003). ECV% was independently associated with all-cause mortality following adjustment for age, sex, peak velocity, impaired LVEF and presence of LGE (HR per% increase in ECV%: 1.13, 95%, 1.04–1.24, p=0.006).

Conclusion In patients with severe AS scheduled for AVR, extracellular volume-based T1 mapping measures are robust, track with LV decompensation, and independently predict late all-cause mortality.

Background Conventional bright-blood late gadolinium enhancement (BB LGE) provides excellent contrast between areas of LGE and normal myocardium. Conversely, contrast between LGE and epicardial fat is frequently poor making the detection of sub-epicardial LGE difficult. Sub-epicardial LGE is a sensitive and specific pattern of LGE classically described in myocarditis. However, in practice, patients with a clinical presentation consistent with myocarditis often have no evidence of LGE. Fat water phase sensitive inversion recovery (PSIR) LGE (FW PSIR LGE) is a novel sequence that enables delineation of pericardial fat and may have a role in improving detection of sub-epicardial LGE.

Objective To compare the diagnostic utility of the FW PSIR LGE sequence to standard BB LGE in patients with suspected myocarditis.

Methods Thirty-one patients referred for clinical CMR for suspected myocarditis were studied. A full left ventricle short axis stack was performed using both techniques. Two experienced observers analyzed all BB LGE and FW PSIR LGE stacks. A scoring system was devised to quantify the presence and extent of gadolinium enhancement.

Results All patients (mean age 43±20 years) presented with chest pain and raised troponin (median high sensitivity troponin T 706 ng/L, interquartile range 104–1185 ng/L) and a normal coronary angiogram or very low probability of coronary artery disease. A total of 496 LV segments were analysed. Significantly more segments demonstrated sub-epicardial LGE using FW PSIR LGE compared to BB LGE (122/496 (25%) vs 44/496 (9%), p<0.01). Twelve patients (39%) with no BB LGE (classified as no myocarditis) were found to have sub-epicardial LGE on FW PSIR LGE (therefore reclassified as positive for myocarditis). There was good agreement between the two observers using both sequences (BB LGE: global agreement 80%, kappa 0.72; FW PSIR LGE: global agreement 80%, kappa 0.78, both p<0.001).

Conclusion Conventional bright blood late gadolinium enhancement images (left panels) and corresponding separated fat water PSIR LGE and fusion imaging (left panels) of 2 different patients showing no definite evidence of subepicardial enhancement in conventional imaging and evidence of subepicardial LGE in separated fat water LGE (red arrows)
Conclusions FW PSIR LGE significantly increases sub-epicardial LGE detection in patients with suspected myocarditis compared to standard bright blood LGE and importantly changes the clinical diagnosis in a third of patients.

Methods In total, 216 subjects were recruited. This included 3 cohorts: 1) Derivation cohort (22 healthy volunteers) to identify a stress MBF threshold value representative of the normal minimum response to adenosine; 2) Validation cohort (37 patients with suspected coronary disease) who underwent stress CMR and invasive coronary physiological assessment on the same day, to validate the stress MBF threshold value against invasive markers of hyperaemia; 3) Clinical cohort (159 patients undergoing clinically-indicated adenosine stress CMR) to assess the presence of stress MBF-defined hyperaemia and other physiological markers of hyperaemia (SSO, HHR and BPR).

Results From the derivation cohort, maximum stress MBF (SMBFmax) >1.43 ml/g/min was derived as the threshold value of hyperaemia (defined as 1.96 standard deviations below the sample mean of lowest stress MBF values). This threshold was tested in the validation cohort: 100% of patients with invasive evidence of hyperaemia demonstrated SMBFmax >1.43 ml/g/min, 81% had SSO and 81% had HRR >10 bpm. Of the clinical cohort, 93% had hyperaemia defined by SMBFmax compared to 71% using SSO and 81% using HRR. SMBFmax was no different in those with or without SSO (2.58±0.89 ml/g/min vs 2.54±1.04 ml/g/min, p=0.84) but those with HRR had significantly higher SMBFmax (2.69 ml/g/min vs 1.95 ml/g/min, p<0.001).

HRR >16 bpm was able to predict SMBFmax >1.43 ml/g/min with sensitivity 63% and specificity 91% (AUC 0.87, p<0.001) and performed better than SSO (AUC 0.62, p<0.001 for comparison of methods).