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.

**Abstract 18 Figure 1**

### QUANTITATIVE CMR MYOCARDIAL PERFUSION MAPPING TO ASSESS HYPERAEMIC RESPONSE TO ADENOSINE STRESS: A NEW REFERENCE STANDARD?

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**Background** Assessment of hyperaemia during adenosine stress CMR remains a clinical challenge with lack of a gold standard marker of adequate stress. Multiple parameters including the splenic switch off (SSO) sign, heart rate response (HRR) and blood pressure response (BPR) are used in clinical practice. Perfusion mapping provides a pixelwise representation of myocardial blood flow (MBF), allowing for measurement of MBF at a regional level. This may provide an alternative tool for assessment of hyperaemia.

**Objectives** To validate the use of stress MBF for assessment of hyperaemic response and compare this to currently used clinical markers.

**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-induced 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).

**Abstract 18 Figure 1**
Conclusion Adenosine-induced increase in MBF measured using perfusion mapping is accurate for the confirmation of hyperaemia during stress CMR studies and is superior to traditional, clinically used markers of adequate stress.

### Abstract 19

**MYOCARDIAL PERFUSION MAPPING IN CARDIAC AMYLOIDOSIS- UNEARTHING THE SPECTRUM FROM INFILTRATION TO ISCHAEMIA**

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**Abstract 19**

Short axis cine SSFP images in end-diastole, corresponding native T1 mapping, late gadolinium enhancement (LGE) images, ECV Mapping and stress myocardial blood flow mapping in a normal subject, a patient with early cardiac infiltration (raised ECV, no LGE), a patient with cardiac amyloidosis, a patient with severe three vessel coronary disease.

### Abstract 20

**ENDOTHELIAL LOSS AS A CAUSE OF IMPAIRED MYOCARDIAL PERFUSION RESERVE ON CMR IN SEVERE AORTIC STENOSIS**


**Abstract 20**

CD31 + for endothelium, smooth muscle actin (SMA) for smooth muscle, and picrosirius red for fibrosis.