cardiovascular outcomes, including new-onset atrial fibrillation, ventricular arrhythmia, hospitalisation due to heart failure or embolic events, or cardiovascular death.

**Results**

HCM patients exhibited lower RV ejection fraction (RVEF) and global peak systolic strains (radial/circumferential/longitudinal) on feature-tracking analysis of cine images than healthy controls. On follow up CMR imaging, RVEF, peak RV circumferential and longitudinal strains decreased over time while LVEF remained preserved. All patients were followed up clinically for a median duration of 4.4 years. Both reduced RVEF and RV longitudinal strain were independent predictors of ventricular arrhythmias and composite cardiovascular endpoint (arrhythmia, cardiac hospitalisation and cardiovascular death), after adjusting for baseline NYHA class, medication use, maximal LV wall thickness, and late gadolinium enhancement. Patients with RVEF <55% had a 3-fold increase in ventricular arrhythmias (HR 3.03, 95% CI 1.63 to 5.66, figure 1A) and a 2-fold increase in composite cardiac events (HR 2.05, 95% CI 1.23 to 3.41, figure 1B). Similarly patients in the lowest quartile of RV longitudinal strain (>0.16.3%) demonstrated a higher risk of ventricular arrhythmias (HR 2.20, 95% CI 1.61 to 3.84, figure 1D) compared to others.

**Conclusions** Despite preserved LVEF, RVEF and strain decline over time in HCM, RVEF and strain predict the occurrence of adverse cardiovascular outcomes and may have a role in prognostic risk stratification in HCM.

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**DIFFUSION TENSOR CARDIOVASCULAR MAGNETIC RESONANCE IN CARDIAC AMYLOIDOSIS**

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**Introduction** Cardiac amyloidosis is a disease of infiltration, typically by light chains (AL) or transthyretin (ATTR). We studied cardiac amyloidosis using diffusion tensor cardiovascular magnetic resonance (DT-CMR), which is the only non-invasive tool able to assess cardiac microstructure in-vivo.

**Methods and results** Biphasic STEAM DT-CMR was successfully performed in 20 cardiac amyloidosis patients (10 AL, 10 ATTR) and 10 age and sex matched controls. Compared with controls, amyloid patients had higher mean diffusivity (MD); median [IQR] 1.46 [1.36–1.54] × 10⁻³ mm²/s vs 1.15 [1.03–1.20] × 10⁻³ mm²/s (p<0.001), shown in figure 1A. MD correlated with T1 (R²=0.76, p<0.001) and extracellular volume (ECV) (R²=0.47, p=0.004).

Amyloid patients had lower fractional anisotropy (FA) compared to controls; 0.43 [0.39–0.45] vs 0.55 [0.52–0.60], p<0.001), as shown in figure 1B. FA inversely correlated with T1 (R²=0.59, p<0.001). Amyloid patients had elevated diastolic E2A and reduced E2A mobility (both <0.001).

**Segmental analysis** for co-location of FA and MD with ECV amyloid infiltration showed good correlation (both p<0.001). Example maps are shown in figure 2. There was no significant difference in MD, FA or E2A mobility between amyloid subtypes.

**Conclusion** In cardiac amyloidosis, DT-CMR characterises the microstructural effects of infiltration, and increases in MD show good correlation with increases in ECV. The novel insights from DT-CMR offer a deeper understanding of pathological mechanisms in cardiac amyloidosis. With further development, DT-0CMR might offer a gadolinium free assessment of amyloid burden, which is of particular value in renal failure.
**Background**

Myositis is a systemic autoimmune condition causing skeletal muscle inflammation and fibrosis as well as extra skeletal involvement. Cardiac involvement is well described and has morbidity and mortality. It can be diffuse or focal which can make assessment using conventional imaging techniques more difficult. To date the incidence is poorly defined using conventional cardiac investigations.

**Purpose**

To investigate the reproducibility of a 12 segment ventricular myocardial model of T1 and T2 mapping in healthy volunteers and then to use this methodology in patients with myositis, comparing it to the current non invasive gold standard of troponin I level for cardiac involvement and then monitor how it changes with time and treatment.

**Methods**

19 Healthy volunteers were scanned at 1.5T. T1 (using a MOLLI sequence) and T2 mapping were performed using T1 and T2 mapping sequences and twelve-segment myocardial assessment to identify and monitor myocardial inflammation in myositis.