Cardiovascular magnetic resonance (CMR) is emerging as an important imaging tool for sub-phenotyping HF. However, currently we cannot estimate LVFP from CMR. This study sought to investigate if CMR can estimate LVFP in patients with suspected HF, whether this has increased diagnostic power beyond TTE and if CMR modelled LVFP has prognostic power.

**Methods** Suspected HF patients underwent RHC, TTE and CMR within 24 hours of each other. RHC measured pulmonary capillary wedge pressure (PCWP) was used as a reference for LVFP. CMR included left/right heart volumetric assessment and left atrial area. Patients were split into derivation (85%) and validation (15%) cohorts (figure 1). In the derivation cohort, multivariate regression was used to determine predictors of LVFP. The CMR-derived model was then applied to the validation cohort and diagnostic accuracy was compared with TTE. Association of CMR modelled LVFP with mortality was determined using Kaplan-Meier (KM) survival analysis.

**Results** We enrolled 835 patients (mean age 66±13 years, 38% male). Two CMR metrics were incorporated in the final model: LV mass and left atrial area. When applied to the validation cohort, CMR modelled PCWP had good correlation with RHC PCWP (R=0.6). The diagnostic accuracy of CMR modelled PCWP to predict elevated filling pressures (RHC PCWP > 14 mmhg) was 73%. TTE was non-diagnostic in 75% of cases (incorrect classification or indeterminate result). Of these, 71% were reclassified to a correct diagnosis by CMR (figure 2). CMR modelled PCWP was identified as an independent predictor of death on KM analysis (HR 2.18 (95% CI 1.1 to 4.3), P=0.02) (figure 3).

**Conclusion** A physiological CMR model can estimate LVFP in patients with suspected HF. Our model demonstrated good diagnostic accuracy providing additive value to TTE assessment. In addition, CMR modelled LVFP has a prognostic role.

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20 **APICAL ISCHAEMIA IS UBIQUITOUS IN APICAL HYPERTROPHIC CARDIOMYOPATHY AND OCCURS BEFORE OVERT HYPERTROPHY**

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**Background** Small vessel disease and associated microvascular ischaemia is a key feature of hypertrophic cardiomyopathy (HCM). Perfusion defects are described in 41–79% of cases. Apical HCM (ApHCM) has features suggesting distinction
from other HCM phenotypes (ECG, arrhythmogenicity, genotype, scar pattern, natural history). Clinically, apical hypoperfusion is frequently observed in ApHCM. We hypothesised that quantitative perfusion would be different and distinct in apical HCM.

**Methods and Results**

**Patients:** 100 subjects with ApHCM, 50 with conventional HCM and 28 healthy volunteer controls. 33 of the ApHCM had ‘relative ApHCM’ – MWT<15 mm but other key features of the disease – typical ECG, apex thicker than septum with loss of apical tapering and apical systolic cavity obliteration.

**CMR:** Quantitative perfusion mapping CMR using adenosine vasodilator stress. A visual read plus global and regional map segmentation was performed, with results expressed as myocardial blood flow (MBF, ml/g/min) and myocardial perfusion reserve (MPR).

**Results**

Of 100 ApHCM, all (100/100) had apical perfusion defects on visual read. There was also a high prevalence of perfusion defects in conventional HCM (45/50, 90%, p=0.012), typically in hypertrophied areas. There were no defects in controls (P<0.005). Compared with HCM, global stress MBF in ApHCM was equivalent (mean (IQ range) 1.71 ml/g/min (1.48–2.06) vs 1.61 ml/g/min (1.31–2.08), P=0.436) and lower than controls (2.59 ml/g/min (2.28–2.96), P<0.005). The reduction in MBF was most pronounced in the apical segments in ApHCM vs HCM (1.27 ml/g/min (1.05–1.64) vs 1.59 ml/g/min (1.25–2.06 ml/g/min), P<0.005) and controls (2.54 ml/g/min (2.34–3.35), P<0.005). The defects were profound with flow reduction during stress being common (MPR<1). Flow reductions were less for relative ApHCM than overt ApHCM (global stress MBF 2.03 ml/g/min (1.71–2.24) vs 1.62 (1.36–1.89), P<0.005), and similarly, relative ApHCM had less scar than overt ApHCM (FWHM LGE 0.0g (0.0–7.0) vs 24.1g (14.2–35.7), P<0.005; 0.0% (0.0–7.6) vs 17.4% (11.0–23.4%), P<0.005).

**Conclusions**

Apical microvascular ischaemia appears the hallmark feature of ApHCM, occurring even when the apical hypertrophy does not reach conventional diagnostic criteria (relative ApHCM).

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**Abstract 20 Figure 1**

Perfusion in relative apical hypertrophic cardiomyopathy with discrete apical chamber. 2-chamber cine in end-diastole(Ai) and end-systole(Ai). Short-axis view showing maximum wall thickness of 10.4 mm at the apex (B). Apical subendocardial late gadolinium enhancement (C). Perfusion mapping following adenosine-stress in the 3 short-axis slices and 2-chamber (Di-iv) demonstrate dense perfusion defect most notable in the apical subendocardium but extending to the basal anteroseptum. Rest perfusion images for comparison (Ei-iv).

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**Exploring the Ethnic Differences in Cardiac Function and Outcomes: A UK Biobank Study**

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**Background**

Risk factors for heart failure (HF) are more prevalent among South Asians (SA). It is unclear whether this translates to a higher risk of HF. Some evidence suggests that there are baseline cardiovascular differences between SA and White Europeans (WE), however this has been previously based on echocardiography imaging which is less accurate compared to cardiac magnetic resonance (CMR).

**Aims**

To investigate the differences in left ventricular (LV) volumes, cardiac output, CO, (both determined by CMR), incidence of HF and risk of HF between SA and WE, in a cohort without cardiovascular disease (CVD).