membranes (e.g., fractional anisotropy with LV GFI: Beta= 0.052; 95% CI= [0.040, 0.064]; p-value= 1.79×10^{-17}). Healthy white matter microstructure was associated with less concentric LV hypertrophy (LVM: LVEDV: Beta= -0.051; 95% CI= [-0.064, -0.038]; p-value= 2.94×10^{-15}) and better GLS (Beta= -0.032; 95% CI= [-0.044, -0.019]; p-value= 4.89×10^{-7}).

Conclusion In this large population cohort, we demonstrate independent links between heart and brain health using detailed MRI. Participants with healthier hearts had significantly less brain atrophy, less brain microvascular injury, and healthier neuronal structure.

7 PREDICTORS OF MYOCARDIAL FIBROSIS AND RESPONSE TO ANTI-FIBROTIC THERAPY IN HEART FAILURE WITH PRESERVED EJECTION FRACTION

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Objectives To identify baseline characteristics that associate with baseline myocardial fibrotic burden, predict change in myocardial fibrosis over a year, and predict response to the novel anti-fibrotic agent, pirfenidone, in patients with heart failure with preserved ejection fraction (HFpEF).

Background Myocardial fibrosis, measured using cardiovascular magnetic resonance extracellular volume (ECV), is associated with adverse outcome in HFpEF. In the PIROUETTE (The Pirfenidone in Patients with Heart Failure and Preserved Left Ventricular Ejection Fraction) trial, pirfenidone reduced myocardial fibrosis.

Methods Amongst patients enrolled in the PIROUETTE trial (n=107), linear regression models were used to assess the relationship between baseline variables and baseline myocardial ECV, and with change in myocardial ECV (week 52 value

minus baseline value), adjusting for treatment allocation. Linear regression models were used to identify baseline variables that modified pirfenidone treatment effect.

Results Body mass index, left atrial reservoir strain, haemoglobin and aortic distensibility were associated with baseline ECV in stepwise modelling (table 1). QRS duration, left ventricular mass and presence of an infarct at baseline were associated with an increase in ECV from baseline to week 52 in stepwise modelling. Whilst QRS duration, presence of an infarct, global longitudinal strain and left atrial strain modified the treatment effect of pirfenidone when considered individually, no baseline variable modified treatment effect on multivariable modelling.

Conclusions Baseline characteristics were identified that associate with myocardial fibrosis and can predict change in myocardial fibrosis over 1 year. No variables that independently modify the treatment effect of pirfenidone were identified (PIROUETTE, NCT02932566).

8 INTELLIGENT LOCALISERS: AN INTEGRATED TIME-SAVING DEEP LEARNING SOLUTION FOR THE PLANNING OF CINE IMAGING AND IDENTIFICATION OF UNEXPECTED FINDINGS FROM A SINGLE TRANSAXIAL STACK

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Background CMR scans typically begin with a transaxial stack for anatomical evaluation, but current strategies require (1) additional localiser images for cine planning and (2) expert staff present to identify unexpected findings and adjust protocols for maximum yield.

We present an integrated deep learning solution to (1) automate scan plane prescription without need for localisers and (2) identify important protocol-changing findings at the earliest stages of the scan.

Methods Task 1: We trained a neural network on 1,300 CMR transaxial stacks to identify the spatial co-ordinates of the cardiac apex and the 6 locations on the mitral annulus corresponding to 2-, 3-, and 4-chamber views. By identifying these points, the network proposed long-axis scanning planes, which were compared with a gold-standard of experienced radiographers who had access to the full conventional range of localiser images. 130 scans were used as a testing set.

Task 2: We trained a neural network on 1,500 transaxial stack slices to perform automated segmentation of important cardiac structures, great vessels, and other pathologies. The system assembled a 3D model of the thorax from which it made clinical measurements which were compared to those

Abstract 7 Table 1 Associations between selected patient characteristics and myocardial extracellular volume (ECV) at baseline

Baseline Covariate	Univariable Model			Multivariable Model		
	β -coefficient	95% CI	P-value	β -coefficient	95% CI	P-value
BMI - kg/m ²	-0.18 (0.05)	-0.28 to -0.08	<0.001	-0.19 (0.05)	-0.29 to -0.10	<0.001
Haemoglobin - g/dL	-0.49 (0.20)	-0.89 to -0.09	0.02	-0.69 (0.18)	-1.05 to -0.34	<0.001
LA strain (reservoir) -%	-0.13 (0.04)	-0.20 to -0.06	<0.001	-0.11 (0.04)	-0.20 to -0.03	0.01
Aortic distensibility - 10 ⁻³ /mmHg	0.43 (0.35)	-0.27 to 1.13	0.22	0.69 (0.29)	0.11 to 1.27	0.02

BMI - body mass index; CI - confidence interval; LA - left atrial.

Abstract 8 Figure 1

obtained from the full scans. 200 scans were used as a testing set.

Results Task 1: The network successfully learned the 7 key points on the transaxial stack, and the scan planes it proposed closely matched those chosen by radiographers using dedicated localisers, with a median out-of-plane distance at the key points of only 3.4 mm (IQR 1.6–6.3) - less than half the slice thickness (8 mm) of the gold standard cines.

Task 2: The system was successful in segmenting the transaxial slices (Dice 0.91). Diagnostic accuracy was high for abnormalities including left and right ventricular dilatation (90.5%, 85.5%), LVH (85%) and ascending aorta dilatation (94.4%). The ROC AUC for diagnosing pleural effusions was 0.91.

Conclusion A neural network can use the standard transaxial stack of images to identify important pathology and accurately predict scan planes close to those of expert radiographers using dedicated localisers. This system could speed up the delivery of CMR and lead to faster and more dynamic scanning protocols.

9

IDENTIFICATION OF THIRTY NOVEL LOCI FOR CARDIOVASCULAR MAGNETIC RESONANCE DERIVED AORTIC DISTENSIBILITY IN THE UK BIOBANK

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Introduction Cardiovascular magnetic resonance (CMR)-derived aortic distensibility (AoD) is a validated tool to measure arterial stiffness¹. This local stiffness marker is an independent predictor of cardiovascular events and all-cause mortality². Our knowledge of genes modulating aortic stiffness remains limited. We conducted genome-wide association studies (GWASs) for AoD in the ascending aorta (AA) and proximal descending aorta (PDA).

Methods 34,039 UK Biobank (UKB) participants of European ancestry with transverse cine images of pulmonary trunk and right pulmonary artery acquired using a 1.5T CMR scanner were included. AoD is defined as the relative change in cross-sectional aorta area for a given change in central pulse pressure, they were either manually derived or processed through a developed automated algorithm³. GWASs were performed using linear mixed models⁴ using ~7 million imputed variants adjusted for age, sex, height, weight, systolic blood pressure, aortic area segmentation method (manual/automated), imaging centre, genotype array, smoking, diabetes mellitus and top 10 principal components. Bioinformatic analyses were performed including investigation of trait pleiotropy. Polygenic risk scores (PRS) for AA and PDA were calculated using PRSice⁵ and tested for association with major adverse cardiovascular events (MACE) in the UKB cohort without imaging data (n=98,559).

Results and discussion Variants at 18 independent loci for AA and 16 loci for PDA AoD were genome-wide significant, $P < 5 \times 10^{-8}$ (table 1 and figure 1) with four shared loci between the two phenotypes. The heritability for both AA and PDA AoD was ~27%, much higher than arterial stiffness index (6%)⁶ but comparable to pulse wave velocity (36–40%)^{7,8}. We captured a modest percentage of the genetic variance for each trait (2.7% for AA; 2.1% for PDA). Several candidate genes are highlighted including *ELN* for both traits, with *ISL1* and *PCSK1* both involved in insulin regulation that may explain the interplay with diabetes^{9,10}. Variants at 13 loci were significant with blood pressure and/or coronary artery