function (ejection fraction (LVEF). The results were externally validated with a MINAP dataset from the Bristol Heart Institute collected over the same time period.

Results A development dataset comprising 2232 patients was used; 931/1668 (42%) had good LV function. Factors entered in the model were decided a priori and were: site of infarction, previous myocardial infarction, heart rate, systolic BP, Killip class, peak troponin, ECG determining treatment, age and gender. The final model had an area under receiver operator curve c-statistic = 0.79 (95% CI 0.75, 0.82). The model was externally validated on 2238 patients from the Bristol Heart Institute. The discrimination of the model was moderate with c-statistic = 0.66 (95% CI 0.64, 0.68); 73% sensitivity and 59% specificity.

Conclusion This clinical risk score predicts the likelihood of having good LV function on transthoracic echocardiogram post myocardial infarction moderately well. Further work to improve the accuracy of the model could enable a move to earlier discharge and out patient echocardiography in more than a third of all AMI patients.

Conflict of Interest Nil

19 MANGANESE-ENHANCED T1 MAPPING FOR THE DETECTION OF LEFT VENTRICULAR REMODELLING AND VIABILITY AFTER MYOCARDIAL INFARCTION

[1]Nick Spath*, [1]Marc Dweck, [1]Andrew Baker, [2]David Newby, [3]Scott Semple. *[1]University of Edinburgh; [2]British Heart Foundation Centre for Cardiovascular Science, University of Edinburgh; [3]Edinburgh Imaging facility, Queens Medical Research Institute*

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Background Delayed-enhancement magnetic resonance imaging (DEMRI) using gadolinium overestimates infarct size acutely and indirectly estimates myocardial viability by quantifying scar burden. Manganese, an essential trace element and paramagnetic calcium-analogue (administered as chelated manganese dipyridoxyl diphosphate, MnDPDP), defines myocardial viability directly by imaging functional calcium-handling mechanisms. Our previous preclinical work demonstrated manganese-enhanced MRI (MEMRI) has potential to detect altered calcium-handling in remodelling myocardium following infarction.

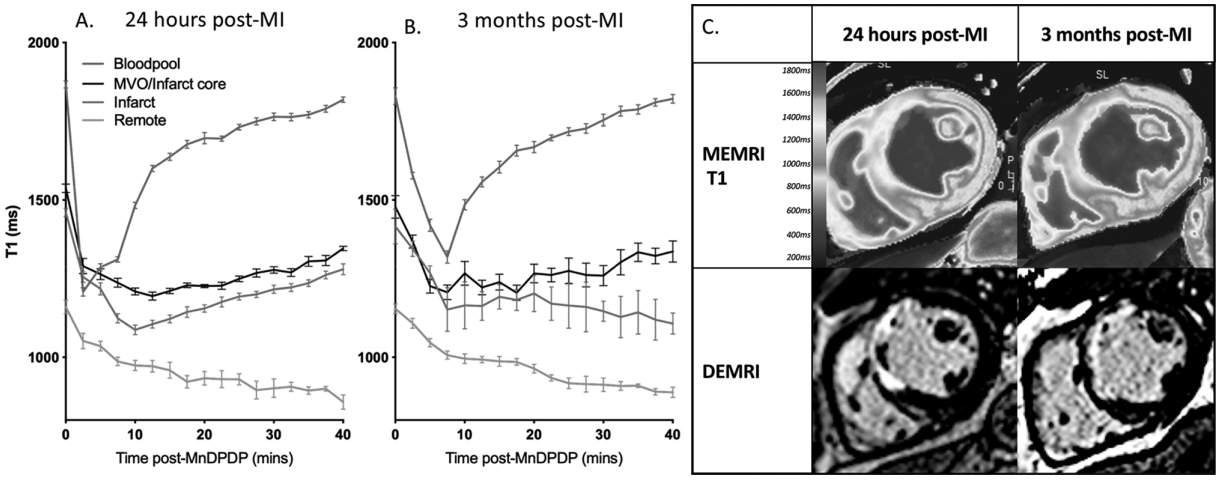
Purpose To assess whether MEMRI detects changes in myocardial calcium-handling after myocardial infarction (NCT03607669).

Methods Healthy volunteers (HV, n=20, 13 male, 42±11 years) and patients with acute ST-segment elevation myocardial infarction with reduced ejection fraction (n=8, recruitment ongoing) underwent dual DEMRI and MEMRI, 48 hours apart, with T1 mapping. Patients returned for repeat imaging at 3 months. Myocardial T1 was measured every 2.5 min for 40 min after intravenous MnDPDP (5 umol/kg) administration. Images were acquired at 3T (Siemens Magnetom Skyrafit) with T1 imaging performed with Shortened Modified Look-Locker Inversion recovery (WIP #1048 Siemens Healthcare Ltd). Scanner-generated T1 maps are analysed to quantify T1 within regions of interest.

Results No adverse effects were observed in any subject. In HV, MnDPDP rapidly reduced bloodpool T1 over 5 min (mean reduction 25.7%, 453±22 ms), followed by prompt normalisation to baseline by 40 min. Myocardial T1 also demonstrated a rapid initial descent (infusion phase) but this was followed by a slower, more gradual decrease which continued throughout the 40 min imaging period (mean reduction 25.2%, 283±9 ms) as evidence of Mn uptake by viable cardiomyocytes.

In patients with acute myocardial infarction, the profile of myocardial T1 following MnDPDP was markedly different in the infarct compared to remote myocardium. In particular, areas of transmural infarction with microvascular obstruction demonstrated a recovery of T1 values similar to bloodpool (figure 1A), whilst T1 in regions of less extensive infarct plateaued after the infusion phase. In contrast to both, a sustained reduction was seen in the remote myocardium, similar to healthy volunteers.

Three months post-myocardial infarction, two distinct T1 profiles were identifiable within the infarct zone (defined by DEMRI): (i) a central region demonstrating partial recovery of T1 following MnDPDP similar to microvascular obstruction and bloodpool, although less rapid, and (ii) the wider infarct region where T1 continued to decrease throughout the 40 min imaging period (figure 1B). This would suggest ongoing manganese uptake and viability despite near transmural late-enhancement (figure 1C), supported by improved wall thickening on cine imaging.



Abstract 19 Figure 1

Overall, T1 values at 40 min post-MnDPDP were 35.9% higher in regions of infarction compared to remote and healthy myocardium (1134±88 versus 843±28 ms, P<0.0001). All infarcts had T1 >1050 ms, whereas remote and healthy myocardium had T1 <950 ms.

Conclusion MEMRI of the myocardium with T1 mapping not only identifies myocardial infarction but also demarcates viability and delineates regions of viability within the infarct zone. This novel contrast imaging technique has exciting potential in ischaemic cardiomyopathy.

Conflict of Interest None

20 REAPPRAISING REMODELLING PATTERN OF LEFT VENTRICLE IN AORTIC STENOSIS: AXIS ORIENTATION AS A UNIQUE SIGNATURE OF POSITIVE REMODELLING

[1]Kenneth Chan*, [1]Masliza Mahmod, [1]Ernesto Zacur, [1]Marzia Rigolli, [1]Michelle D'Souza, [1]Jane Francis, [1]Rina Ariga, [1]Betty Raman, [1]Sairia Dass, [1]Theodoros Karamitsos, [1]Stefan Neubauer, [1]Saul Myerson, [2]Pablo Lamata. *[1]Oxford University; [2]Kings College London*

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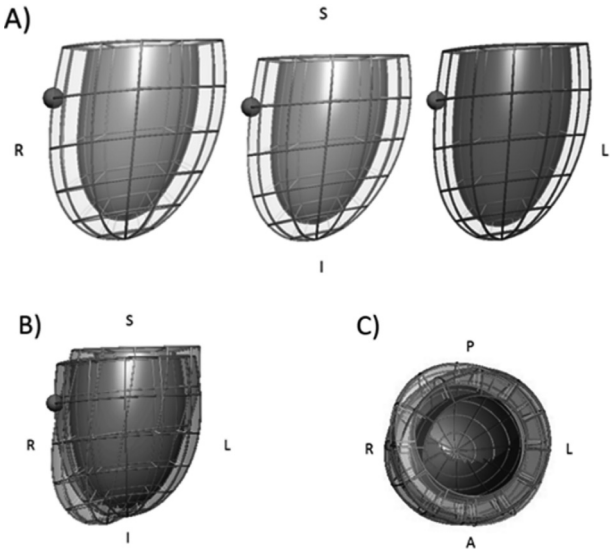
Introduction In aortic stenosis (AS), characterisation of ventricular (LV) remodelling beyond left ventricular mass measurements is lacking. We sought to study the 3-dimensional (3D) geometric LV remodelling pattern in severe AS pre- and post-surgical aortic valve replacement (AVR), and compared it with hypertensive and healthy controls.

Methods Ninety-one subjects (36 severe AS, 19 hypertension and 36 healthy controls) underwent cardiac magnetic resonance (CMR). 18 AS patients had a repeat CMR eight-month post-AVR. 3D meshes were reconstructed from the myocardial contours of the CMR cine images. Principle component analysis and linear discrimination analysis were used to derive shape coefficients.

Results AS patients had a significant shift in LV axis and apex orientation towards the septum, and more spherical LV shape which were not seen in the hypertensive and healthy control groups. As expected severe AS was associated with thicker and larger LV compared to the other two groups. Post AVR, despite significant reduction in LV thickness and sphericity, interestingly the shift in the LV axis/orientation was unchanged/irreversible (Figure 1).

Conclusion Severe AS is characterised by unique remodelling pattern which is not reversible post AVR. The novel shape metrics that comprehensively quantify the LV morphology may be a potential marker for risk stratification in the management of AS.

Conflict of Interest none



Abstract 20 Figure 1 A) Comparison between average LV shape in AS (blue), AS post-AVR (green), and controls (red). B) Overlying shape between AS (orange) and control (purple) showing shift of LV axis to septum. C) Overlying shape of AS post-AVR (orange) and healthy control (purple) showing focal dilatation in the postero-septal region post AVR

Cardiac Rhythm Management

21 THE CARDIOVASCULAR PREDICTIVE VALUE AND GENETIC BASIS OF T-WAVE MORPHOLOGY

[1]Julia Ramirez*, [2]Stefan van Duijvenboden, [1]Andrew Tinker, [3]Pier Lambiase, [3]Michele Orini, [1]Patricia B Munroe. *[1]Queen Mary University of London; [2]University College London; [3]UCL*

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Background Early prediction of cardiovascular (CV) events in the general population remains an important issue. The T-wave morphology restitution (TMR), an ECG marker quantifying ventricular repolarization dynamics, is strongly associated with CV mortality in heart failure patients. Our objective was to evaluate the CV prognostic value of TMR in the general population and identify any genetic contribution.

Methods ECG recordings from 56,780 healthy individuals undergoing exercise stress testing in the UK Biobank study (EST-UKB) were analyzed. TMR was computed for exercise (TMRex) and recovery from exercise (TMRrec). The primary endpoint was CV death or hospitalizations for CV reasons. The secondary and tertiary endpoints were (1) all-cause mortality or hospitalizations for CV reasons and (2) arrhythmic mortality or hospitalizations for arrhythmic reasons. The median follow-up time was 70.7 months. Genome-wide association studies for TMRex and TMRrec were also performed and genetic risk scores (GRSs) were derived and tested for association with endpoints in the full cohort (FULL-UKB; N=402,746, median follow-up time of 85.3 months).

Results 1,727 (3.0%) individuals met the primary endpoint in EST-UKB, and 2,326 (4.1%) and 120 (0.2%) met the secondary and tertiary endpoints, respectively. TMRrec was significantly associated with the primary endpoint (hazard ratio (HR) 1.15, P=2 × 10–10, table 1), and both secondary and tertiary endpoints (HR 1.13, P=2 × 10–11 and HR 1.28, P=1 × 10–4, respectively) independent of resting