seven non-experts. The vFFRs were computed using the VIRTUheart™ tool (University of Sheffield). Figure 1 shows an example from the workflow. The vFFR results of the expert and non-expert analysed were compared on the basis of the recommendation for percutaneous coronary intervention vs medical therapy and the reason for the differences were documented. Inter- and intra-expert differences and the impact of the expert decisions upon potential clinical management were also assessed.

**Results**
The angiograms from 1098 patients with CAD were screened, from which 316 cases for vFFR analysis were identified as being suitable for processing. From these, one expert selected 264 consecutive cases for re-processing at random, of which 214 were successfully re-processed. Reasons for unsuccessful segmentation included inadequate images, poor opacification, overlap of vessels and unworkable geometry. The expert mean vFFR was 0.76 and the non-expert was 0.75 (mean per case difference 0.11, SD 0.12), with 73% agreement and 27% disagreement about treatment strategy (see figure 2). Of those, 18% would have been incorrectly revascularised and 9% incorrectly managed conservatively. The mean inter-observer (1st vs 2nd expert) and intra-observer (1st vs 1st expert) differences were 0.06 and 0.09 respectively, and agreement in management interpretations 89% and 90% respectively (p <0.0001). The management interpretation, based upon expert vFFR analysis vs the original cardiologist’s decision based upon the angiogram alone, revealed 37% disagreement, with 23% incorrectly revascularised and 14% incorrectly managed conservatively.

**Conclusion**
There is a large difference in vFFR modelling between expert and less expert modellers. The differences are due to errors in 3-D vessel construction. There is little inter- or intra-observer variation between expert modellers. However good the modelling system, training is required to produce accurate vFFR results. Expert vFFR can improve the clinical management of patients with CAD, altering revascularisation decision in 37% cases.

**Conflict of Interest**
None

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**18 CLINICAL RISK MODEL TO PREDICT LIKELIHOOD OF HAVING GOOD LV FUNCTION POST MYOCARDIAL INFARCTION**

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**Background**
A national shortage of sonographers and NHS hospital beds challenges us in providing timely inpatient echocardiography to patients admitted to hospital with acute myocardial infarction (AMI). A clinical risk model to identify clinical predictors of having good LV function post AMI could potentially help risk-stratify patients for early discharge and expedited outpatient echocardiography.

**Aim**
To develop a clinical risk score to predict the likelihood of having good LV function on transthoracic echocardiography post AMI.

**Methods**
Data that had been collected for patients presenting to Worcestershire Acute Hospitals NHS Trust with AMI between July 2014 and November 2017 were used. These data had been collected as part of the Myocardial Infarction National Audit Project (MINAP). A clinical risk model was developed. Multiple imputation methods were used to deal with missing data. Logistic regression was used to determine to effect of these factors upon the outcome of good LV
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 outpatient echocardiography in more than a third of all AMI patients.

**Conflict of Interest**

Nil

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

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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.