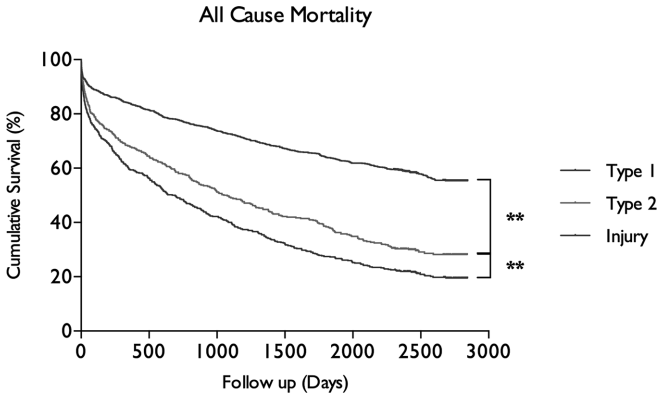


**Abstract 113 Figure 1** Kaplan Meier curve displaying time to death stratified by classification of myocardial infarction or myocardial injury. Log-rank test for comparison. ** denotes p < 0.01

## 114 PERSISTENCE OF HAEMOGLOBIN DEGRADATION PRODUCTS WITHIN INFARCT SCAR TISSUE AFTER ST-ELEVATION MYOCARDIAL INFARCTION: INCIDENCE, CORRELATES AND IMPLICATIONS FOR LEFT VENTRICULAR REMODELLING

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**Background** Myocardial haemorrhage is a prognostically important complication of acute ST-elevation myocardial infarction (STEMI). Persistence of haemoglobin degradation products within infarct scar tissue and the potential clinical significance have not been investigated.

**Methods and Results** Patients who sustained an acute STEMI were enrolled in a cohort study (BHF MR-MI; ClinicalTrials.gov: NCT02072850). 211 patients (mean (SD) age 57 (11) years, 77% male) had evaluable T2* cardiac magnetic resonance (CMR) imaging (1.5 Tesla) 2 days 6 months post-MI. Myocardial haemorrhage was defined as a hypointense infarct core with T2* signal <20ms. At 2 days, 79 (37%) patients had evidence of myocardial haemorrhage. At 6 months, 47 (59%) patients had a hypointense infarct core and 32 (41%) did not. None of the patients had *de novo* haemorrhage after the first CMR scan. Clinical associates of a persistent hypointense core at 6 months included hypertension (odds ratio (95% confidence interval) 0.28 (0.08, 0.95); p = 0.040), heart rate (1.08 (1.03, 1.13); p = 0.001), systolic blood pressure (1.06 (1.01, 1.12); p = 0.015), neutrophil count (2.19 (1.01, 4.74); p = 0.048), left anterior descending as culprit artery (9.26 (1.26, 67.99); p = 0.029), infarct size (1.10 (1.03, 1.17); p = 0.004) and initial haemorrhage size (1.34 (1.06, 1.66); p = 0.012). A hypointense infarct core with T2*-mapping at 6 months was associated with worsening LV end-diastolic volume (regression coefficient (95% confidence interval) 15.43 (1.35, 29.50); p = 0.032) and worsening left ventricular ejection fraction (-4.15 (-7.40, -0.89); p = 0.013).

**Conclusion** Persistence of haemoglobin degradation products at 6 months post-STEMI is common and prognostically important.

## 115 PERSISTENCE OF INFARCT ZONE OEDEMA AT 6 MONTHS AFTER ACUTE ST-ELEVATION MYOCARDIAL INFARCTION: INCIDENCE, PATHOPHYSIOLOGY AND ASSOCIATION WITH LEFT VENTRICULAR REMODELLING

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**Background** The natural history of persistent myocardial oedema after ST-elevation myocardial infarction (STEMI) is uncertain.

**Methods** and ResultsPatients who sustained an acute STEMI were enrolled in a cohort study (BHF MR-MI; ClinicalTrials.gov: NCT02072850). Cardiac magnetic resonance (CMR) imaging with T2-mapping of myocardial oedema was performed at 1.5 Tesla 2 days and 6 months post-MI. Myocardial oedema was defined as infarct signal intensity (S. I.) >2 standard deviations from the mean S. I. within a remote reference region. 283 STEMI patients (mean (SD) age 59 (12) years, 75% male) were enrolled. Infarct size was 18 (13)% of left ventricular (LV) mass. At 2 days, infarct zone T2 (ms) was higher than remote zone T2 (62.9 (5.2) vs. 49.7 (2.1); p < 0.001), and this relationship persisted at 6 months (56.2 (4.1) vs. 49.7 (2.3); p < 0.001). Mean remote zone T2 did not change over time (p = 0.840) whereas infarct zone T2 decreased (p < 0.001). At 6 months, infarct zone oedema persisted in 177 (63%) patients who were more likely to have a history of hypertension (regression coefficient (95% confidence interval) 1.77 (0.99, 3.13); 0.053) and a larger initial size of infarction (1.02 (1.00, 1.05); p = 0.020) but less likely to have a history of previous PCI (0.28 (0.08, 0.99); p = 0.049), compared to patients without persistent oedema. In a multivariate analysis, infarct zone oedema at 6 months was associated with an increase in LV end-diastolic volume (9.68 (3.76, 15.61); p = 0.001).

**Conclusion** Persistence of oedema within the infarct zone affected the majority of patients at 6 months post-STEMI, and was associated with the initial severity of STEMI and adverse LV remodelling.

# Imaging

## 116 PHYSIOLOGIST-DIRECTED STRESS MYOCARDIAL PERFUSION SCINTIGRAPHY PROTOCOL IS SAFE: A SINGLE SITE PRE- AND POST-INTERVENTION STUDY

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**Introduction** Vasodilator stress as part of MPS has existed for decades. There is wide national variation in procedural protocols with many centres advocating physician presence during stress MPS. The suggested safety profile of the adenosine-2A agonist Regadenoson lends itself to physiologist-led stress MPS without the need for physician presence. We investigated the