resonance imaging (CMR) using T1 MOLLI maps before and after contrast. We repeatedly measured infarct ECV in STEMI survivors, and assessed the relationships between ECV, peak troponin T and baseline infarct size.

Methods Acute STEMI survivors were enrolled in a single-centre cohort study (BHF MR-MI study – NCT02072850). Contrast-enhanced CMR was performed at 1.5 Tesla (Siemens MAGNETOM Avanto) 2 days and 6 months post-MI. T1 mapping with MOLLI was performed before and 15 min after contrast (0.15 mmol/kg gadoterate meglumine). ECV analysis was performed by outlining regions of interest (ROIs) in infarcted myocardium and left ventricular (LV) blood pool. ROIs were representative of the infarct including microvascular obstruction. ECV was calculated as the relaxation rate (R1=1/T1) for myocardium and LV blood pool before vs. after contrast, corrected for haematocrit. Infarct size was measured using late gadolinium images and expressed as a percentage of LV mass. Infarct ECV at baseline and follow up was compared with peak troponin T measured at presentation and infarct size at baseline. Peak troponin T was log-transformed to improve normality and linearity.

Results 201 STEMI patients (mean age 58 ± 11 years; 156 (77%) male) were enrolled. Infarct ECV was similar at baseline and follow-up (51.3 ± 9.5% vs. 50.3 ± 12.0%, p = 0.097). Peak troponin T was widely variable (4256.7 ug/L ± 4020.8 ug/L). Peak troponin T was positively associated with infarct ECV at baseline (p < 0.001) and follow-up (p < 0.001). Infarct size at baseline was 18 ± 13% of LV mass and was positively associated with infarct ECV at baseline (p < 0.001) and follow-up (p < 0.001) (Table 1).

Abstract 4 Table 1 Association of infarct ECV at baseline and follow-up with peak troponin T and infarct size in linear regression analysis (n = 201)

	Baseline Infarct ECV			Follow-up Infarct ECV*		
Characteristics	Coefficient (95% CI)	R^2	p	Coefficient (95% CI)	R^2	p
Log peak troponin T	4.754 (3.473, 6.034)	0.293	<0.001	5.093 (3.688, 6.498)	0.612	<0.001
Infarct size	0.420 (0.307, 0.533)	0.284	<0.001	0.421 (0.298, 0.543)	0.600	<0.001

*Accounting for baseline infarct ECV

Conclusion High peak troponin T and a large infarct size are predictors of higher infarct ECV at baseline and follow-up. Infarct ECV is associated with clinical and MRI measures of MI severity in survivors of STEMI.

5 RELATIONSHIPS BETWEEN INFARCT ZONE EXTRACELLULAR VOLUME AND CLINICAL MEASURES OF ISCHAEMIA AND REPERFUSION IN ACUTE STEMI SURVIVORS

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10.1136/heartjnl-2015-307845.5

Background The clinical significance of infarct extracellular volume (ECV) post-STEMI is unknown. ECV can be estimated by cardiac magnetic resonance imaging (CMR) using T1 MOLLI maps. We measured infarct ECV in STEMI survivors, and assessed the relationships between ECV and markers of MI severity.

Methods STEMI survivors were enrolled in a single-centre cohort study (BHF MR-MI study – NCT02072850). Culprit artery flow was described by thrombus in myocardial infarction (TIMI) classification. CMR was performed at 1.5 Tesla (Siemens MAGNETOM Avanto) 2-days and 6-months post-MI, including T1-mapping with MOLLI before and 15-minutes after contrast (0.15 mmol/kg gadoterate meglumine). ECV was analysed by outlining regions-of-interest (ROIs) in infarcted myocardium, including microvascular obstruction, and left ventricular (LV) blood pool. ECV was calculated as the relaxation rate (R1=1/T1) for myocardium and LV blood pool before vs. after contrast, corrected for haematocrit. ECV was compared with TIMI-flow pre- and post-percutaneous coronary intervention (PCI) and ST-segment resolution. A reduction in ST-segment voltage of ≥70% was considered complete ST-resolution and <70% was considered incomplete ST-resolution.

Results 201 STEMI patients (age 58 ± 11 years; 156 (77%) male) were enrolled. Infarct ECV was similar at baseline and follow-up (51.3 ± 9.5% vs. 50.3 ± 12.0%, p = 0.1). Pre-PCI, 131 (65%) patients had TIMI-flow 0 and 70 (35%) had TIMI-flow 1–3. Post-PCI, 3 (1.5%) patients had TIMI-flow 0 and 198 (98.5%) had TIMI-flow 2–3. TIMI-flow 0 pre-PCI was associated with higher infarct ECV at baseline (p < 0.001) and follow-up (p = 0.004). TIMI-flow post-PCI was not associated with infarct ECV. ST-resolution was complete in 104 (52%) patients and incomplete in 96 (48%) patients. Incomplete ST-resolution was associated with higher infarct ECV at baseline (p = 0.001) and follow-up (p = 0.026) (Table 1).

Conclusion Preserved culprit artery flow and complete ST-resolution are associated with lower infarct ECV at baseline

Abstract 5 Table 1 Association of infarct ECV at baseline and follow-up with TIMI coronary flow grade pre- and post-PCI in linear regression analysis (n = 201)

	Baseline Infarct ECV			Follow-up Infarct ECV*		
Characteristics	Coefficient (95% CI)	R^2	p	Coefficient (95% CI)	R^2	p
TIMI coronary flow grade pre-PCI 1/2/3 vs 0 (ref)	−6.743 (−9.972, −3.514)	0.111	<0.001	−4.834 (−8.092, −1.575)	0.458	0.004
TIMI coronary flow grade post-PCI 2/3 vs 0 (ref)	−5.386 (−16.308, 5.536)	0.007	0.331	1.656 (−8.037, 11.348)	0.450	0.736
ST-segment resolution: complete vs incomplete (ref)	−5.191 (−8.250, −2.133)	0.077	0.001	−3.390 (−6.358, −0.422)	0.488	0.026

*Accounting for baseline infarct ECV

and follow-up. Clinical and electrocardiographic markers of MI severity are predictors of interstitial expansion in the infarct zone in STEMI patients.

6 SEGMENTAL VARIATION IN MYOCARDIAL EXTRACELLULAR VOLUME IN HEALTHY MID-LIFE ADULTS

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10.1136/heartjnl-2015-307845.6

Background Myocardial extracellular volume (ECV) can be estimated by cardiac magnetic resonance imaging (CMR) using pre- and post-contrast T1 MOLLI maps. The age and sex associations with myocardial ECV in healthy mid-life adults are uncertain.
Methods Healthy adults without any history of cardiovascular disease or treatment underwent contrast-enhanced CMR at 1.5 Tesla (Siemens MAGNETOM Avanto). T1 mapping with MOLLI was performed before and 15 min after contrast (0.15 mmol/kg gadoterate meglumine). ECV was estimated in regions (AHA 16-segment LV model) and for the whole left ventricular (LV) myocardium (all regions). ECV was calculated as the difference in relaxation rate (R1=1/T1) for myocardium and LV blood pool before vs. after gadolinium contrast administration, corrected for haematocrit (HCT). LV segments which were not evaluable due to artefact were excluded from analysis.
Results 114 segments were assessed from 19 subjects (mean age 61 ± 12 years; 10 (53%) male). 21 (18%) segments were excluded due to blood pool artefact or signal drop-out in the pre-contrast T1 MOLLI scan. All segments were evaluable in the post-contrast T1 MOLLI scans. The remaining segments for each subject were averaged to give an overall ECV (global LV). The mean ECV for all subjects was 25.6 ± 2.9%. There was no overall segmental variation in ECV. ECV in females was higher than in males (27.6 ± 3.1% vs. 23.9 ± 1.3%; $P = 0.003$) The percentage difference was 14.5%. ECV was higher in septal segments in females (anteroseptal: 28.0 ± 3.3% vs. 24.2 ± 1.5%; $P = 0.004$; inferoseptal: 27.3 ± 3.8% vs. 23.5 ± 1.6%; $P = 0.011$), whereas no differences were observed for other segments (Table 1).

Abstract 6 Table 1 Sex and segmental variation in ECV. ECV presented as Mean ± SD

ECV (%) per Segment	All subjects (n = 19)	Male (n = 10)	Female (n = 9)	P
Anteroseptal	26.0 ± 3.1	24.2 ± 1.5	28.0 ± 3.3	0.004
Anterior	24.5 ± 3.9	22.6 ± 1.6	26.0 ± 4.6	0.063
Anterolateral	24.8 ± 3.2	24.2 ± 1.8	25.7 ± 4.5	0.407
Inferolateral	26.0 ± 3.7	24.6 ± 1.9	27.6 ± 4.6	0.112
Inferior	25.5 ± 4.4	23.6 ± 2.4	27.4 ± 5.2	0.087
Inferoseptal	25.3 ± 3.4	23.5 ± 1.6	27.3 ± 3.8	0.011
Average	25.6 ± 2.9	23.9 ± 1.3	27.6 ± 3.1	0.003

Conclusion In this preliminary analysis, myocardial ECV was higher in women than in men, which was attributable to higher ECV in the septum in females. This sex difference merits further study. If these results are confirmed by other studies, then sex-specific reference ranges for ECV would seem appropriate.

7 DIAGNOSTIC ACCURACY OF 12 LEAD ECG Q-WAVES AS A MARKER OF MYOCARDIAL SCAR: VALIDATION WITH CMR

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10.1136/heartjnl-2015-307845.7

Background Traditionally, the presence of Q-waves on 12 lead ECG is considered a marker of a large and/or transmural myocardial infarction (MI). Late gadolinium enhancement (LGE) cardiovascular magnetic resonance (CMR) accurately identifies the presence and extent of myocardial infarction and has become the gold standard for the assessment of myocardial viability.
Aim To determine the diagnostic accuracy of Q-waves on 12 lead ECG to identify myocardial scarring as compared with CMR.
Methods Data was collected on 631 consecutive patients referred for a stress CMR with suspected ischaemic heart disease (April 2013 to Mar 2014). A 12-lead ECG was recorded. Pathological Q-waves – deflection amplitude of >25% of the subsequent R wave, or being >0.04 s (40 ms) in width and >2 mm in amplitude in >1 corresponding lead. A comprehensive CMR protocol was used. Transmural infarction was defined as >50% LGE.
Results 498 patients were included (mean age of 64 ± 12 years, 71% males). 290 patients demonstrated MI of whom 157 were transmural and 133 sub-endocardial based on CMR LGE. 126 had pathological Q-waves on 12 lead ECG. The sensitivity, specificity, positive, negative predictive value and accuracy of 12 lead ECG Q-wave as a marker of transmural MI was 36%, 80%, 45%, 73% with moderate overall diagnostic accuracy (66%). The diagnostic accuracy of Q waves as a predictor of previous MI (composite of sub-endocardial and transmural) was 55% (Table 1).
Conclusion Our study demonstrates that the presence of pathological Q-waves on 12 lead ECG is not only a poor marker of myocardial scarring, but also a poor predictor of viability when compared to CMR. In their clinical decision making process, clinicians needs to be aware of the limitation of ECG Q-waves.

Abstract 7 Table 1 Sensitivity, specificity, positive predictive value, negative predictive value and diagnostic accuracy of ECG Q-waves vs LGE myocardial infarction scar

	Sensitivity (%)	Specificity (%)	Positive Predictive value (%)	Negative Predicitive value (%)	Accuracy (%)
Q waves vs transmural MI	36.3	79.8	45.3	73.1	66.1
Q waves vs any MI	32.8	85.1	75.4	47.6	54.6

8 MYOCARDIAL HAEMORRHAGE AFTER ACUTE REPERFUSED ST-ELEVATION MYOCARDIAL INFARCTION: TEMPORAL EVOLUTION, RELATION TO MICROVASCULAR OBSTRUCTION AND PROGNOSTIC SIGNIFICANCE

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10.1136/heartjnl-2015-307845.8