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

¹J Carberry*, ^{1,2}D Carrick, ³C Haig, ¹SM Rauhalampi, ¹K Mangion, ¹D Corcoran, ¹A Radjenovic, ^{1,2}C Berry. ¹BHF Glasgow Cardiovascular Research Center, Institute of Cardiovascular and Medical Sciences, University of Glasgow, Glasgow, UK; ²West of Scotland Heart and Lung Center, Golden Jubilee National Hospital, Dumbartonshire, UK; ³Robertson Center for Biostatistics, University of Glasgow, Glasgow, UK

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 $25.6 \pm 2.9\%$. There was no overall segmental variation in ECV. ECV in females was higher than in males ($27.6 \pm 3.1\%$ vs. $23.9 \pm 1.3\%$; $P = 0.003$). The percentage difference was 14.5%. ECV was higher in septal segments in females (anteroseptal: $28.0 \pm 3.3\%$ vs. $24.2 \pm 1.5\%$; $P = 0.004$; inferoseptal: $27.3 \pm 3.8\%$ vs. $23.5 \pm 1.6\%$; $P = 0.011$), whereas no differences were observed for other segments (Table 1).

Abstract 6 Table 1 Sex and segmental variation in ECV. ECV presented as Mean $\pm$ SD

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

Alexander Carpenter, Amardeep Ghosh Dastidar, Catherine Wilson, Jonathan Rodrigues, Anna Baritussio, Chris Lawton, Alberto Palazzuoli, Nauman Ahmed, Mandie Townsend, Andreas Baumbach, Angus Nightingale, Chiara Bucciarelli-Ducci. NIHR Bristol Cardiovascular Biomedical Research Unit, Bristol Heart Institute, Bristol, UK

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 need 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 Predictive 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

¹DJA Carrick, ²C Haig, ¹N Ahmed, ³H Eteiba, ³M McEntegart, ³S Watkins, ³M Lindsay, ¹A Radjenovic, ¹KG Oldroyd, ¹C Berry. ¹Cardiovascular Research Centre of Glasgow, Institute of Cardiovascular and Medical Sciences, Glasgow, UK; ²University of Glasgow, Robertson Centre for Biostatistics, Glasgow, UK; ³Golden Jubilee National Hospital, Cardiology, Glasgow, UK

10.1136/heartjnl-2015-307845.8