provides further reassurance that complete revascularisation can be considered at PPCI.

Abstract 19 Table 1 Baseline, angiographic and CMR characteristics

Variable	IRA-only revascularisation (n = 105)	Complete revascularisation (n = 98)	p
Baseline characteristics			
Age (y)	64.1 ± 10.8	63.1 ± 11.3	0.53
Male sex (n,%)	83/105 (79.0)	87/98 (88.8)	0.06
Anterior infarct (n,%)	37/105 (37.2)	35/98 (35.7)	0.94
Diabetes Mellitus (n,%)	13/105 (12.4)	15/98 (15.3)	0.55
Angiographic markers			
TIMI pre PCI grade 0–2 (n,%)	97/105 (92.4)	89/98 (90.8)	0.69
SYNTAX score (total)	18 (14–22)	17.3 (13–23.5)	0.81
Symptom-PCI time (TTR, min)	171 (127–268)	192 (131–302)	0.20
TIMI post PCI grade 3 (n,%)	100/105 (95.2)	89/98 (90.8)	0.21
Acute CMR			
Time to acute CMR (d)	2.8 (1.8–3.4)	3.0 (2.0–4.3)	0.13
LV ejection fraction (%)	45.1 ± 9.5	45.9 ± 9.9	0.60
Peak LV circumferential strain (Ecc,%)	−18.1 ± 6.0	−18.6 ± 6.1	0.86
Total infarct size (% LV mass)	13.5 (6.2–21.9)	12.6 (7.2–22.6)	0.57
Patients with >1 infarct	11/105 (10.5)	22/98 (22.4)	0.02
Patients >1 acute infarct	5/105 (4.8)	17/98 (17.1)	0.004
Myocardial salvage index (%)	60.5 (40.6–81.9)	58.5 (32.8–74.9)	0.14
Follow-up CMR			
Time to follow-up CMR (CMR2, mth)	9.3 (8.9–9.9)	9.4 (9.0–10)	0.20
LV ejection fraction (%)	50.8 ± 8.7	49.7 ± 9.4	0.42
Peak LV circumferential strain (Ecc,%)	−23.6 ± 6.3	−22.5 ± 6.3	0.28
Total infarct size (% LV mass)	7.6 (3.2–15.1)	7.3 (3.0–14.4)	0.41
Patients with >1 infarct (%)	9/80 (11.2)	20/84 (23.8)	0.035
Presence of ischaemia (n,%) in all pats	16/77 (20.8)	17/82 (20.7)	0.99
Global ischaemic burden (%) all pats	4.3 ± 11.3	3.4 ± 8.9	0.81

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MYOCARDIAL EXTRACELLULAR VOLUME PREDICTS FUNCTIONAL RECOVERY IN ACUTE MYOCARDIAL INFARCTION MORE ACCURATELY THAN THRESHOLD-BASED MEASURES OF LATE GADOLINIUM ENHANCEMENT TRANSMURAL EXTENT

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10.1136/heartjnl-2015-307845.20

The transmural extent of late gadolinium enhancement (LGE) CMR predicts functional recovery in acute myocardial infarction (AMI). Automated methods are recommended¹ to define infarct extent on LGE imaging, such as ‘n-standard deviations’ (SD) and ‘full width at half maximum’ (FWHM). These define infarcted myocardium by signal intensity as compared to remote myocardium, which in turn depends on signal-to-noise and contrast. Individual variability in these parameters makes a single thresholding technique unlikely to be universally suitable. Extracellular volume (ECV) estimation by T1-mapping CMR is theoretically less affected by sequence and contrast variations. We compared infarct ECV with threshold-based measures of LGE transmural extent to predict contractile recovery in reperfused AMI.

Consecutive patients with reperfused first ST-elevation AMI underwent acute (day 2) and convalescent (3 months) CMR. Cine imaging, modified Look-Locker inversion T1 mapping natively and 15 min post gadolinium-contrast administration and LGE imaging were performed. Five LGE thresholding techniques were compared: 2, 5 and 6 SD, FWHM and a histogram-based technique (Otsu).² The ability of acute infarct ECV to predict improvement in segmental wall motion was compared with these thresholding techniques.

n = 35 (28(80%) male, age 57 ± 11 years). Infarct characteristics are shown in Table 1. ECV showed modest correlation with all threshold measures of LGE ($r^2 = 0.16$ – 0.31 , $p < 0.01$).

Abstract 19 Figure 1 Multiple infarcts on late gadolinium imaging in complete revascularisation patients

The 2 images on left and 2 images on right are 2 different patients.

A = main infarct-related artery territory infarct

B = infarct in non-infarct related artery territory

Reduced convalescent wall motion score correlated with acute ECV ($p < 0.01$), and acute LGE for 5 SD ($p < 0.01$), 6 SD ($p < 0.01$) and FWHM ($p = 0.01$), but not 2 SD ($p = 0.2$) or Otsu ($p = 0.6$). Acute infarct ECV demonstrated a significantly higher c-statistic for prediction of improved segmental convalescent wall motion score than all threshold measures of acute transmural LGE extent ($p \leq 0.02$ for all, Figure 1).

Acute infarct ECV outperforms threshold-based LGE transmural extent to predict segmental LV functional recovery in reper-fused AMI.

Abstract 20 Table 1 Infarct characteristics

Characteristic	Acute visit	Convalescent visit	P value
Ejection fraction, %	48 ± 9	58 ± 7	<0.01
LV EDV ¹ , ml/m ²	81 ± 16	85 ± 21	ns
LV ESV ¹ , ml/m ²	41 ± 12	36 ± 13	<0.01
LGE transmural extent, %	79 ± 12	61 ± 16	<0.01
LGE infarct volume, ml	16 ± 11	10 ± 8	<0.01
LGE MO volume, ml	2 ± 2		
Area at risk, ml	41 ± 17		
Myocardial salvage index	0.55 ± 0.25		
Infarct native T1	1333 ± 110	1244 ± 124	<0.01
Remote native T1	1189 ± 71	1146 ± 72	ns
Infarct ECV	0.56 ± 0.14	0.57 ± 0.19	ns
Remote ECV	0.30 ± 0.06	0.29 ± 0.06	ns

Data as mean ± SD. ¹LV EDV¹ Left ventricular end diastolic volume, indexed to body surface area, ¹LV ESV¹ Left ventricular end systolic volume, indexed to body surface area. LGE measurements made with visual analysis

Abstract 20 Figure 1 Receiver operator characteristic (ROC) curve comparing infarct ECV and LGE thresholds in dysfunctional segments ($n = 163$) with improvement in wall motion score at 90 days. Remote segments not shown. C-statistic and 95% confidence intervals are shown in the legend for each method. ECV had a significantly higher c-statistic than all threshold-based measures ($p \leq 0.02$ for all).

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21 LEFT ATRIAL REMODELLING FOLLOWING TREATMENT OF SYMPTOMATIC SEVERE AORTIC STENOSIS

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10.1136/heartjnl-2015-307845.21

Background Left atrial (LA) size is an adverse prognostic marker. Aortic stenosis results in increased filling pressures with progressive left atrial enlargement and dysfunction. Intervention for aortic stenosis results in LV reverse remodelling, however the effect of TAVI or SAVR upon LA function remains poorly understood and the two treatments have not been directly compared.

Aim We assessed LA size and function in patients with severe symptomatic aortic stenosis before and 6 months TAVI and SAVR.

Methods All patients underwent an identical 1.5T CMR protocol and were in sinus rhythm at time of imaging. LA volumes were derived using the biplane area-length method (Figure 1). The total LA emptying fraction was derived as: (LAVmax–LAVmin)*100/LAVmax.

Results 23 SAVR patients (age 72.7 ± 7.5 years, 83% male, EuroSCORE II 1.40 ± 1.11%) and 23 TAVI patients (age 80.7 ± 6.9 years, 57% male, EuroSCORE II 4.99 ± 2.97%). The left atria of the TAVI group were significantly more dilated at baseline than those of the SAVR group ($p = 0.039$) however both groups were comparable at 6 months ($p = 0.227$). Similarly, the LA emptying fraction of the TAVI group was significantly lower than the SAVR group at baseline ($p = 0.003$) with comparable function seen at 6 months ($p = 0.08$) (Table 1).

Abstract 21 Table 1 LA volumes and emptying fraction before and after TAVI and SAVR

TAVI	Baseline	6 months	p Value
Maximum LA volume (mls/m ²)	63.3 ± 17.0	52.8 ± 14.0	0.001
Total LA emptying fraction (%)	36.9 ± 12.6	43.4 ± 10.4	0.011
SAVR	Baseline	6 months	p Value
Maximum LA volume (mls/m ²)	53.2 ± 15.1	48.2 ± 11.7	0.09
Total LA emptying fraction (%)	48.5 ± 12.8	48.7 ± 9.1	0.945

Conclusions TAVI, but not SAVR, was associated with a significant reduction in LA volume and concomitant improvement in emptying fraction at 6 months. These preliminary findings may reflect worse LA function at baseline in the TAVI group or improved valvular haemodynamics with TAVI compared to SAVR.

22 SURGICAL AORTIC VALVE REPLACEMENT (SAVR) UPON RIGHT VENTRICULAR FUNCTION: A CARDIAC MRI STUDY

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10.1136/heartjnl-2015-307845.22

Background Right ventricular function is of prognostic importance in a variety of clinical settings but its complex anatomic geometry can pose a challenge to 2-dimensional imaging modalities. Right ventricular dysfunction is thought to occur following cardiac surgery and independently predicts adverse outcomes. However a clear mechanism for this dysfunction remains undefined.

Aim To accurately assess the effect of SAVR upon right ventricular function in patients treated for severe symptomatic aortic stenosis.

Methods All patients underwent an identical 1.5T CMR protocol before and 6 months after surgery (Intera, Phillips Healthcare, Best, The Netherlands or Avanto, Siemens Medical Systems, Erlangen, Germany).