

# Quantification of infarct size and myocardium at risk: evaluation of different techniques and its implications

Elisa McAlindon<sup>1</sup>, Maria Pufulete<sup>2</sup>, Chris Lawton<sup>1</sup>, Gianni D. Angelini<sup>1</sup>  
and Chiara Bucciarelli-Ducci<sup>1\*</sup>

<sup>1</sup>NIHR Bristol Cardiovascular Biomedical Research Unit, Bristol Heart Institute, Level 7 Queens Building, Bristol Royal Infirmary, Bristol BS2 8HW, UK; and

<sup>2</sup>Clinical Trial and Evaluation Unit (CTEU), University of Bristol, Bristol, UK

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## Aims

The aim of this study was to evaluate seven methods for quantifying myocardial oedema [2 standard deviation (SD), 3 SD, 5 SD, full width at half maximum (FWHM), Otsu method, manual thresholding, and manual contouring] from T<sub>2</sub>-weighted short tau inversion recovery (T2w STIR) and also to reassess these same seven methods for quantifying acute infarct size following ST-segment myocardial infarction (STEMI). This study focuses on test–retest repeatability while assessing inter- and intraobserver variability. T2w STIR and late gadolinium enhancement (LGE) are the most widely used cardiovascular magnetic resonance (CMR) techniques to image oedema and infarction, respectively. However, no consensus exists on the best quantification method to be used to analyse these images. This has potential important implications in the research setting where both myocardial oedema and infarct size are increasingly used and measured as surrogate endpoints in clinical trials.

## Methods and results

Forty patients day 2 following acute reperfused STEMI were scanned for myocardial oedema and infarction (LGE). All patients had a second CMR scan on the same day >6 h apart from the first one. Images were analysed offline by two independent observers using the semi-automated software. Both oedema and LGE were quantified using seven techniques (2 SD, 3 SD, 5 SD, Otsu, FWHM, manual threshold, and manual contouring). Interobserver, intraobserver and test–retest agreement and variability for both infarct size and oedema quantification were assessed. Infarct size and myocardial quantification vary depending on the quantification method used. Overall, manual contouring provided the lowest inter-, intraobserver, and interscan variability for both infarct size and oedema quantification. The FWHM method for infarct size quantification and the Otsu method for myocardial oedema quantification are acceptable alternatives.

## Conclusions

This study determines that, in acute myocardial infarction (MI), manual contouring has the lowest overall variability for quantification of both myocardial oedema and MI when analysed by experienced observers.

## Keywords

Cardiac magnetic resonance • Infarct size • Myocardial oedema • Quantification

## Introduction

Myocardial oedema,<sup>1</sup> myocardial salvage<sup>2</sup> (calculated as myocardial oedema minus infarct size), and infarct size<sup>3–5</sup> assessed by cardiovascular magnetic resonance (CMR) are prognostic indicators following myocardial infarction (MI). Myocardial oedema and myocardial

salvage are increasingly being used as surrogate endpoints in clinical trials to test different reperfusion strategies. However, there is no standardization as to the technique used to quantify the myocardial oedema and the debate continues.<sup>6,7</sup> There is some evidence as to which analysis technique to use when assessing chronic infarct size; suggesting the full-width at half-maximum (FWHM) method or 5

\* Corresponding author. Tel: +44 117 3423145; Fax: +44 117 9299737, E-mail: c.bucciarelli-ducci@bristol.ac.uk

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standard deviations (SD) from remote myocardium,<sup>8</sup> although this study did not include the Otsu method. FWHM has also been validated against histology in an animal model for the quantification in acute MI,<sup>9</sup> whose phenotype differs from the chronic scar. In fact, the concomitant presence of myocardial oedema, and often of microvascular obstruction and intramyocardial haemorrhage (all hallmarks of acute infarctions and absent in chronic infarctions), alters myocardial signal intensity (SI) and can affect a signal intensity threshold-based method of analysis.

For myocardial oedema, many centres use 2 SD from remote myocardium or manual contouring.<sup>10</sup> There are many software options available for image post-processing analysis that allow 2 SD, 3 SD, 5 SD, FWHM, Otsu method, manual thresholding, and manual contouring. The aim of this study was to evaluate these seven available methods for quantifying myocardial oedema from  $T_2$ -weighted short tau inversion recovery (T2w STIR) imaging following ST-segment myocardial infarction (STEMI), and also to reassess these same seven methods for quantifying acute infarct size following STEMI. This study focuses on test–retest repeatability while assessing inter- and intraobserver variability.

## Methods

### Patient population

Thirty patients day 2 following a first acute reperfused MI treated with successful primary angioplasty, and agreeable to undergo repeated scans on the same day, were approached. All patients were diagnosed with STEMI according to guidelines.<sup>11</sup> Exclusion criteria were: general contraindications to MRI, chronic atrial fibrillation, renal impairment with eGFR <30, cardiogenic shock, and patients with special communication needs. This study was approved by the local ethics committee. All patients gave informed written consent. These patients were recruited to the CMR repeatability in the STEMI study NCT 01468662 ([www.clinicaltrials.gov](http://www.clinicaltrials.gov)).

### Image acquisition

Patients had two CMR scans at least 6 h apart on Day 2 following STEMI, interval for the second scan was  $8 \pm 2$  h [median of 7 h (range 6–10 h)]. Six hours were chosen to allow enough time for the contrast to wash out between scans, while minimizing the time between scans for patients' physiological parameters or imaging characteristics to alter. Patients were studied using a 1.5-T scanner (Magnetom Avanto, Siemens) with a standard 12-channel matrix coil configuration. The same operator acquired all images.

The imaging protocol included three long axis and a full stack of short axis (8 mm, no gap) from base to apex using both the T2w STIR and late gadolinium enhancement (LGE) techniques following 0.1 mL/kg gadobutrol (Gadovist, Bayer Schering Pharma).

For T2w STIR imaging,<sup>12</sup> a breath-hold black-blood segmented turbo spin echo technique was adopted, using a triple inversion recovery preparation module in order to suppress signal from flowing blood as well as from fat, with surface coil normalization. Typical imaging parameters were TR 2 R-to-R intervals, TE 75 ms, flip angle 90°, TI 170 ms, slice thickness 8 mm, no interslice gap, field of view 340–400 mm, matrix 208 × 256, and a voxel size of 2.3 × 1.4 × 8 mm. T2w STIR images were acquired on short-axis planes covering the entire left ventricle. Each slice was obtained during a breath-hold of 10–15 s depending on the patient's heart rate. To accommodate poor breath-holders, turbo factor was increased as necessary.

For LGE imaging, a standard inversion recovery gradient-echo sequence was adopted. Acquisition planes were identical to that of T2w STIR and were acquired at least 10 min following administration of 0.1 mL/kg gadobutrol (Gadovist, Bayer Schering Pharma). LGE images were obtained using an inversion recovery prepared breath-hold gradient-echo technique. Typical image parameters were TR 700 ms, TE 4.33 ms; matrix 256 × 256; flip angle 30°; slice thickness 8.0 mm, no interslice gap, and voxel size 1.7 × 1.4 × 8 mm. The inversion time was progressively optimized to null normal myocardium (typical values, 250–350 ms). Images were acquired on short-axis planes covering the entire left ventricle. Each slice was obtained during a breath-hold of 10–15 s depending on the patient's heart rate. To accommodate poor breath-holders, segments for acquisition were increased as necessary.

The imaging planes were planned *de novo* for the second; however, care was taken to start the short-axis slice at the mitral valve annulus in both scans to achieve as close as possible scan planes.

### Image analysis

Images were randomized for analysis. All measurements on the first scan were performed by two observers (E.M. and C.B.-D., level 3 SCMR) blinded to clinical and angiographic data, and previous image analysis. For intraobserver variability, Observer 1 analysed 'Scan A' and re-analysed 'Scan A' at an interval of 1 month. For interobserver variability, both Observer 1 and Observer 2 analysed 'Scan A'. For test–retest repeatability, Observer 1 analysed 'Scan A and Scan B'. The observers were blinded to the previous analysis results. Hypointense areas in the T2w STIR and LGE images (representing intramyocardial haemorrhage and microvascular obstruction, respectively), when present, were included in the contoured areas. Image quality was subjectively visually graded 1 or 2 (1 = good and 2 = suboptimal/non diagnostic). Images were suboptimal/non diagnostic if there was artefact or signal loss that interfered with the ability of the observers to interpret the image. No images were excluded because they were non diagnostic.

### Image analysis methods

Images were analysed offline using the semi-automated software (CMR42 Circle Cardiovascular Imaging, Canada, a certified CMR analysis software). The endocardium and epicardium were delineated on each slice. For 2, 3, and 5 SD, a region of interest (ROI) was drawn in the remote myocardium, deemed unaffected with the absence of regional wall motion abnormalities or LGE. In addition, an ROI was drawn in the high SI myocardium in the affected myocardium for the FWHM technique. Manual thresholding (a technique where the operator adjusts a threshold bar to include the affected myocardium) was subjective as was manual contouring. The myocardial oedema and infarct size were expressed as a mass in grams (g). The FWHM threshold<sup>13</sup> estimates an intensity threshold from the remote myocardium as midway between the mean intensity within the remote region and the maximal intensity within the affected tissue. The Otsu method<sup>14</sup> estimates the intensity threshold from the histogram of all intensities to get minimal variance both above and below threshold. For T2w STIR and LGE, the optimal window setting was defined as the sum of the mean myocardial SI of the unaffected area plus 2 SD for this area. The level setting was set at the mean SI of the unaffected area.<sup>15</sup> Where the left ventricular outflow tract was present within the slice, this was manually excluded.

### Statistical analysis

Intraobserver, interobserver, and test–retest agreement were assessed using the 95% limits of agreement method (Bland–Altman).<sup>16</sup> Means and SDs of the differences between repeat measurements and 95% limits of agreement [ $\text{mean} \pm (1.96 \times \text{SD})$ ] were calculated. Bland–Altman plots

of the differences between the measurements against their mean were constructed. The variability data were graphically displayed, showing the variability attributed to intraobserver, interobserver, and test–retest effects as a proportion of total variability for each method [ $1 - \text{intraclax correlation coefficient (ICC)}$ ].

## Results

Fifty-three patients were initially enrolled in the study. Of the 53 patients initially enrolled, 40 completed the protocol successfully. Thirteen patients (25%) withdrew following the first scan due to claustrophobia (11 patients), fatigue (1 patient), or inability to continue with the second scan (1 patient). Forty patients completed the two scans protocol. The median time difference between the two scans was 7 h [interquartile range (IQR) 7, 8]. Most patients were scanned Day 2 (range 1–4), following acute reperfused STEMI. Mean age was  $60 \pm 11$  years, 80% male, 20% female. The infarct-related artery (IRA) was the left anterior descending artery (LAD) in 13 patients, the left circumflex artery (LCx) in 6 patients, and the right coronary artery (RCA) in 21 patients. Time to reperfusion was  $4.2 \pm 3.5$  h. CMR characteristics of the population included mean LV end diastolic volume  $138 \pm 27$  mL, LV end systolic volume  $58 \pm 19$  mL, and mean ejection fraction  $58 \pm 10\%$ . The mean troponin T was  $3976 \pm 3017$  ng/L. *Figures 1 and 2* provide examples of the region of myocardial oedema and infarct size as identified by each technique, respectively.

### Infarct-related artery

For the purposes of analysis, IRAs by territory were assigned according to standardized myocardial segmentation:<sup>17</sup> the anteroseptum and anterior wall were assigned to the LAD territory, the lateral wall to the LCx territory, and the inferior wall and inferoseptum to the RCA territory. Many patients had a large amount of myocardial salvage (minimal LGE) from early intervention. Both LGE and T2w STIR agreed on the IRA. When compared with angiography, both techniques mislabelled one patient IRA as LCx when the IRA by angiography was diagonal territory.

*Figures 3 and 4* shows that there were differences in the mass of myocardial oedema and infarct size quantified by the seven techniques. Five SD produced the smallest mass of myocardial oedema ( $20 \pm 16$  g), and FWHM the largest ( $104 \pm 91$  g). Five SD produced the smallest mass of infarct size ( $30 \pm 16$  g) and 2 SD the largest ( $66 \pm 24$  g). *Table 1* summarizes the correlation of infarct size against peak troponin T. Manual contouring has the best correlation with peak troponin T of all seven methods for quantification. *Table 2* reports the correlation of myocardial oedema with area at risk (AAR) calculated by an angiographic score (APPROACH) in a subset of 23 patients. Manual contouring has the best correlation with ARR by APPROACH among the seven techniques tested.

### Agreement

Agreement was assessed using the Bland–Altman method. Bland–Altman plots for intraobserver, interobserver, and test–retest agreements were acquired for the seven analysis techniques for both myocardial oedema (see Supplementary data online, *Figures S1–3*, respectively) and infarct size (see Supplementary data online, *Figures S4–6*, respectively). Intraobserver agreement was

good for both oedema and infarct size with a minimal bias for all techniques and a good distribution of points (see Supplementary data online, *Figures S1 and S4*). Interobserver agreement showed a systematic bias for both oedema and infarct size quantification (see Supplementary data online, *Figures S2 and S5*). This was most marked for 2, 3, and 5 SD of myocardial oedema. In addition, the interobserver Bland–Altman plots for myocardial oedema when using 2, 3, and 5 SD, manual threshold, and manual contouring showed a greater bias with increasing oedema mass (see Supplementary data online, *Figure S2*). The test–retest Bland–Altman plots (see Supplementary data online, *Figures S3 and S6*) showed a systematic bias for both infarct size and myocardial oedema for all, except the 2, 3, and 5 SD of myocardial oedema techniques. There is a degree of funnelling of the points, suggesting greater variability as oedema and infarct size increase, for all test–retest plots except the FWHM and Otsu myocardial oedema plots.

Overall, bias is most marked on interobserver quantification of myocardial oedema with a greater bias as the mass of oedema increases.

### Myocardial oedema variability

Variability was assessed using the ICC. Variability is shown in *Figure 5*. Compared with any other technique, taking into account inter/intraobserver variability and test–retest repeatability, manual contouring has the lowest variability overall for myocardial oedema; manual thresholding has the highest variability. The Otsu method also has low interobserver, intraobserver variability, and test–retest repeatability. Although intraobserver variability was good for 2, 3, and 5 SD, interobserver variability and test–retest repeatability is higher for these methods, there is a significant bias for interobserver agreement on Bland–Altman analysis as described above.

### Infarct size variability

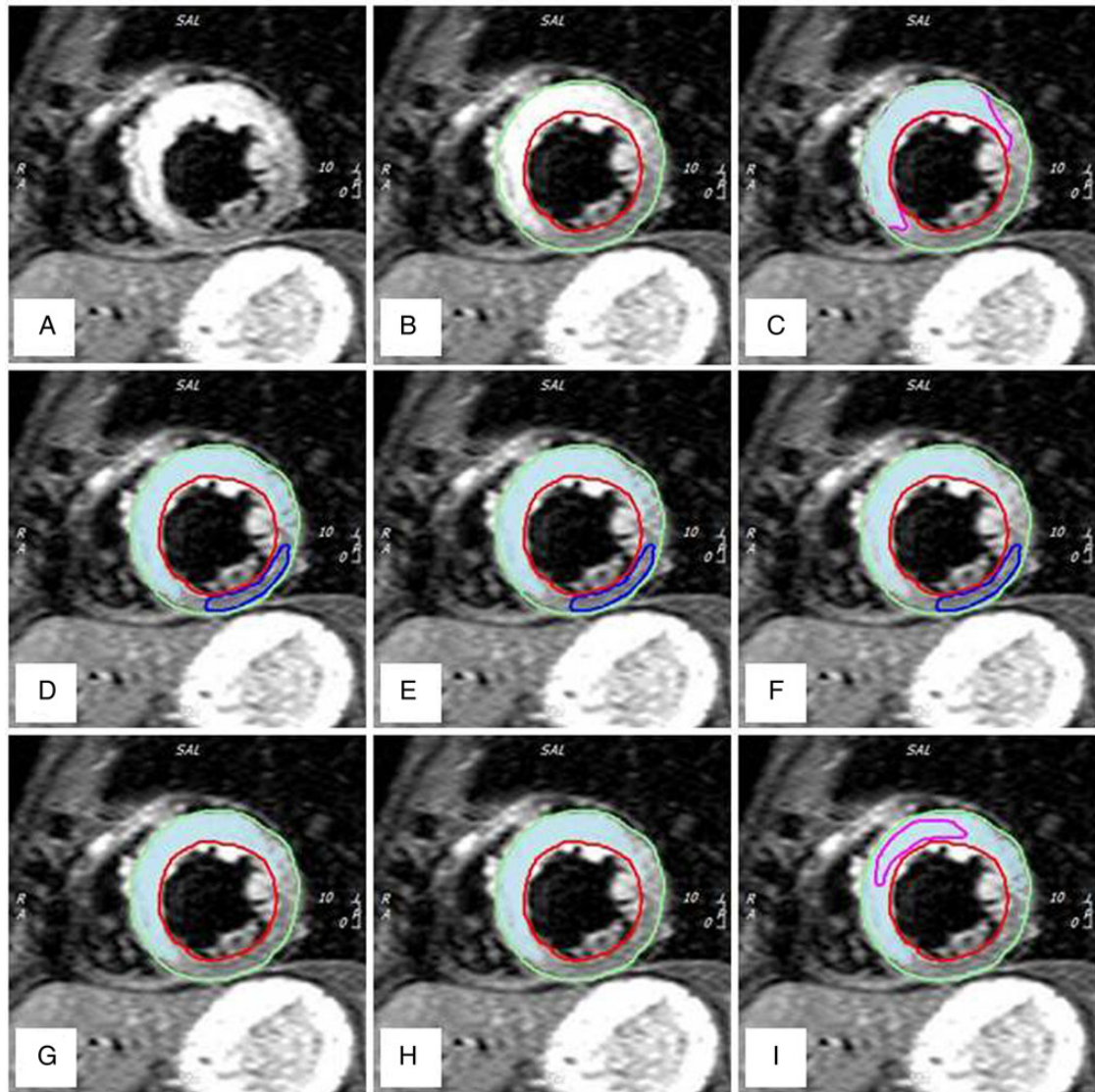
Variability for infarct size analysis is shown in *Figure 6*. Again, manual contouring has the lowest variability overall followed by FWHM and Otsu methods. 2SD had the highest variability.

## Discussion

This study assesses intraobserver, interobserver, and importantly, test–retest repeatability using numerous (7) available analysis methods for the quantification of myocardial oedema and infarct size in a population with reperfused acute myocardial infarction (STEMI). Most of the previous studies compared few methods of analysis either in patients with chronic MIs or cardiomyopathies. This is also the first study that investigated test–retest repeatability in patients with acute MI.

We demonstrated that in acute MI, among the various methods, manual contouring has the lowest overall variability for quantification of both myocardial oedema and MI when analysed by experienced observers. The FWHM method for infarct size quantification and the Otsu method for myocardial oedema quantification are acceptable alternatives.

We also demonstrated that different quantification methods result in different mass of myocardial oedema and infarct size, suggesting that the different methods are not interchangeable. In fact, if myocardial salvage is calculated from the highest and lowest methods for



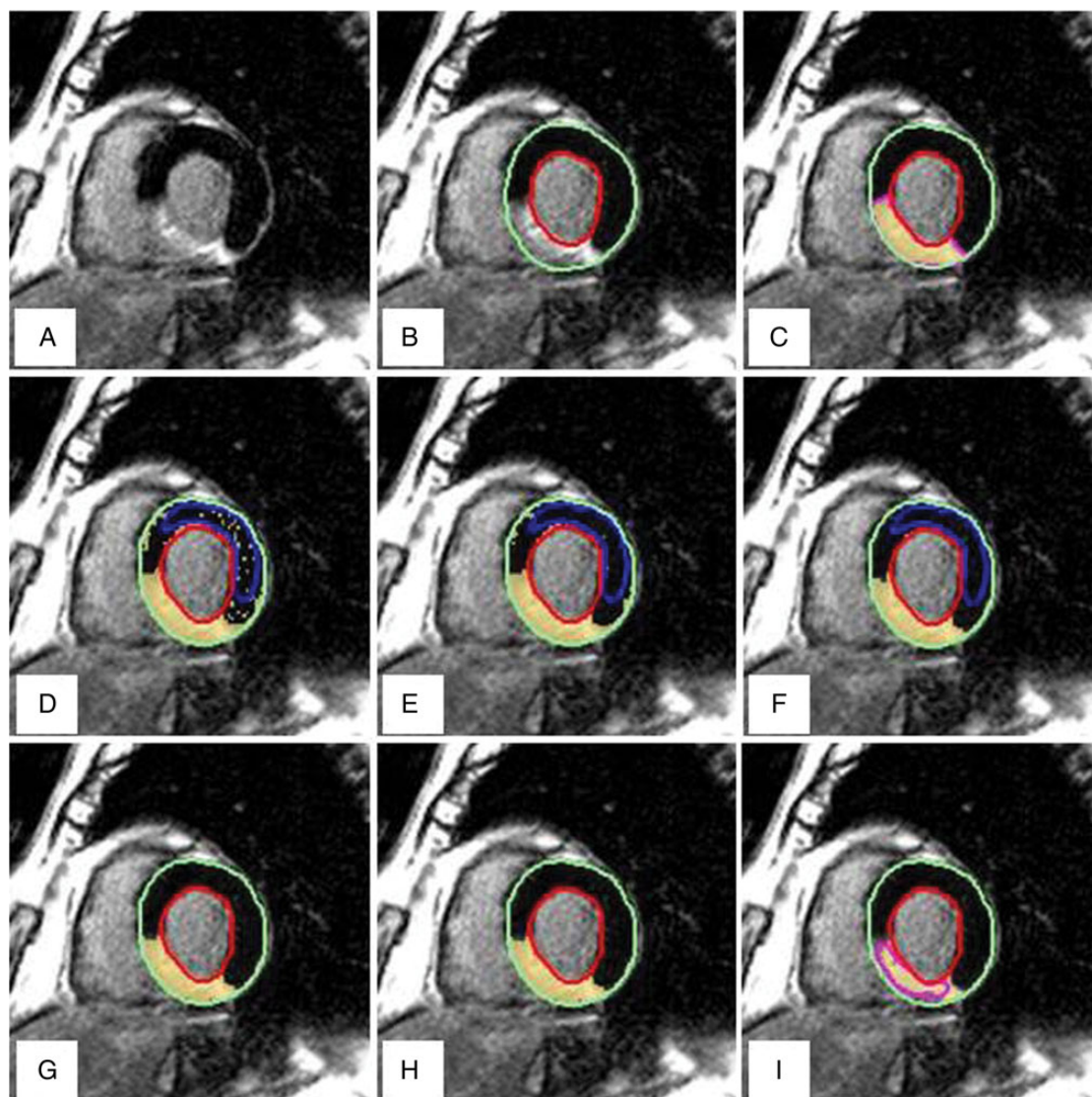
**Figure 1** Myocardial oedema in the LAD territory as quantified by each technique: (A) raw image, (B) endocardium and epicardium demarcated, (C) manual contouring, (D) 2 SD, (E) 3 SD, (F) 5 SD, (G) Otsu, (H) manual threshold, and (I) FWHM. The oedema is highlighted in light blue. For 2, 3, and 5 SD, an ROI is identified (dark blue contour) in the reference remote myocardium. For FWHM, an ROI is identified in the affected myocardium (pink contour).

infarct size and myocardial oedema, mean myocardial salvage can vary from 18 to 39% LV (if FWHM used for oedema to calculate the AAR and 2 or 5 SD are used for infarct size). Similarly, if the quantification technique used for oedema imaging is 5 SD, generating the smallest amount of oedema for AAR, mean myocardial salvage would be negative. When manual contouring is used for quantification for both, salvage is 12% LV.

An acute MI differs significantly from a chronic MI. Features typical of an acute injury like myocardial oedema, microvascular obstruction, and intramyocardial haemorrhage make the acute scar a much more complex entity to analyse compared with a chronic scar. This complexity challenges the methods of analysis, which rely mostly on SI threshold and SD. In particular, this could represent an issue

especially in acute infarctions where the hypointense regions of microvascular obstruction and intramyocardial haemorrhage will affect the hyperintense infarcted and oedematous myocardial signals.

Although both myocardial oedema and MI are important consequences of STEMI with prognostic importance, their best method of quantification remains unclear, with many laboratories using different methods. The lack of evidence and indeed of head-to-head comparison of different methods have been highlighted by the recent consensus document 'Standardized image interpretation and post processing in cardiovascular magnetic resonance: Society for Cardiovascular Magnetic Resonance (SCMR) Board of Trustees Task Force on Standardized Post Processing'.<sup>18</sup> Our study addressed this issue demonstrating that among the currently available methods, manual



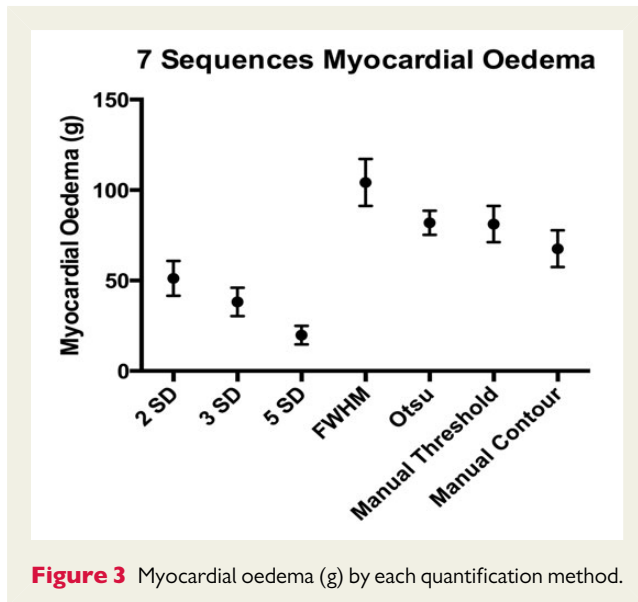
**Figure 2** MI in the RCA territory as quantified by each technique: (A) raw image, (B) endocardium and epicardium demarcated, (C) manual contouring, (D) 2 SD, (E) 3 SD, (F) 5 SD, (G) Otsu, (H) manual threshold, and (I) FWHM. The infarct is highlighted in yellow. For 2, 3, and 5 SD, an ROI is identified in the reference remote myocardium (dark blue contour). For FWHM, an ROI is identified in the affected myocardium (pink contour).

contouring performed by experienced operators is overall (taking into account variability and agreement), the most reproducible method available for both myocardial oedema and infarct size.

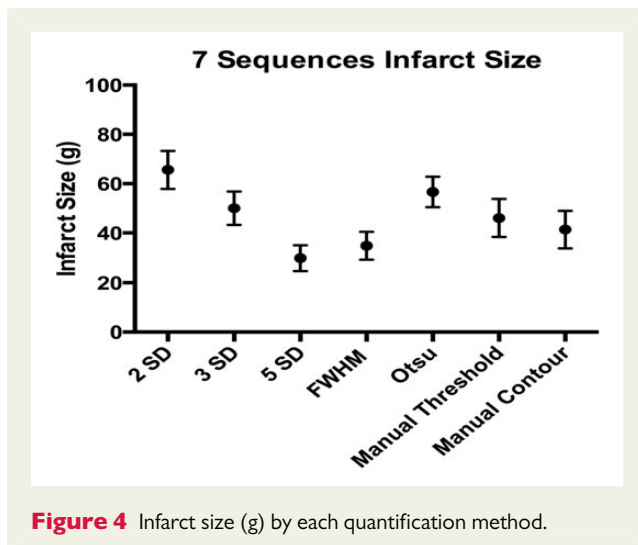
### On quantifying infarct size

Previous studies demonstrated that FWHM<sup>8,9</sup> might be the appropriate method for LGE quantification in MI. Other studies suggest 5 SD,<sup>19</sup> 6 SD,<sup>20</sup> or manual contouring<sup>19</sup> as suitable methods. None of these studies included test–retest repeatability. Other investigators have tried to determine the optimal method for LGE quantification in cardiomyopathies,<sup>8</sup> and found that in this setting the areas of the LGE have a lower SI compared with LGE in MIs, making manual contouring challenging and unsuitable and FWHM the optimal quantification method. A recent study investigated the most reproducible

method of analysis LGE across multiple pathologies: acute MI, chronic MI, and hypertrophic cardiomyopathy.<sup>8</sup> However, this study did not investigate test–retest repeatability. In keeping with this previous work,<sup>8</sup> our study agrees that manual contouring and FWHM have low intra- and interobserver variability and would be suitable methods also to use in the setting of acute MI. Manual contouring has been validated against SPECT for assessment of infarct size in acute MI.<sup>21</sup> In addition, our study expands on this work by also assessing test–retest repeatability showing that, overall, manual contouring is the most robust method for assessing infarct size in acute MI. Although FWHM remains a good technique in acute STEMI and is validated against histology in acute MI,<sup>9</sup> it may be limited as it assumes a bright centre of infarction; microvascular obstruction in STEMI may impact on this.



**Figure 3** Myocardial oedema (g) by each quantification method.



**Figure 4** Infarct size (g) by each quantification method.

## On quantifying myocardial oedema

As opposed to infarct size, there is no current consensus on which technique to use for quantifying myocardial oedema.<sup>18</sup> Important prognostic data using myocardial oedema have been assessed using manual contouring,<sup>2,22</sup> Manual contouring,<sup>21</sup> 2 SD<sup>23,24</sup> from the remote myocardium, FWHM,<sup>25</sup> and the Otsu method<sup>26</sup> have all been proposed. A previous head-to-head comparison investigating an additional proposed automatic segment algorithm (not tested in this study) also investigated interobserver agreement of manual contouring, 2 SD, FWHM, and Otsu methods.<sup>27</sup> The interobserver agreement was better for manual contouring than the 2 SD, FWHM, and Otsu in keeping with our findings. However, the study by Sjogren *et al.*<sup>27</sup> found that the interobserver bias was relatively smaller with Otsu and 2 SD than FWHM. It is perhaps surprising that the Otsu method had a higher interobserver variability than manual contouring: this may be explained in that differing observers

**Table 1** The correlation of infarct size against peak troponin T (ng/L)

Quantification method	R-value	P-value
2 SD	0.31	0.05
3 SD	0.37	0.02
5 SD	0.46	0.003
FWHM	0.44	0.005
Otsu	0.12	0.24
Manual threshold	0.45	0.004
Manual contour	0.59	<0.0001

Manual contouring has the best correlation with peak troponin T of all seven methods for quantification.

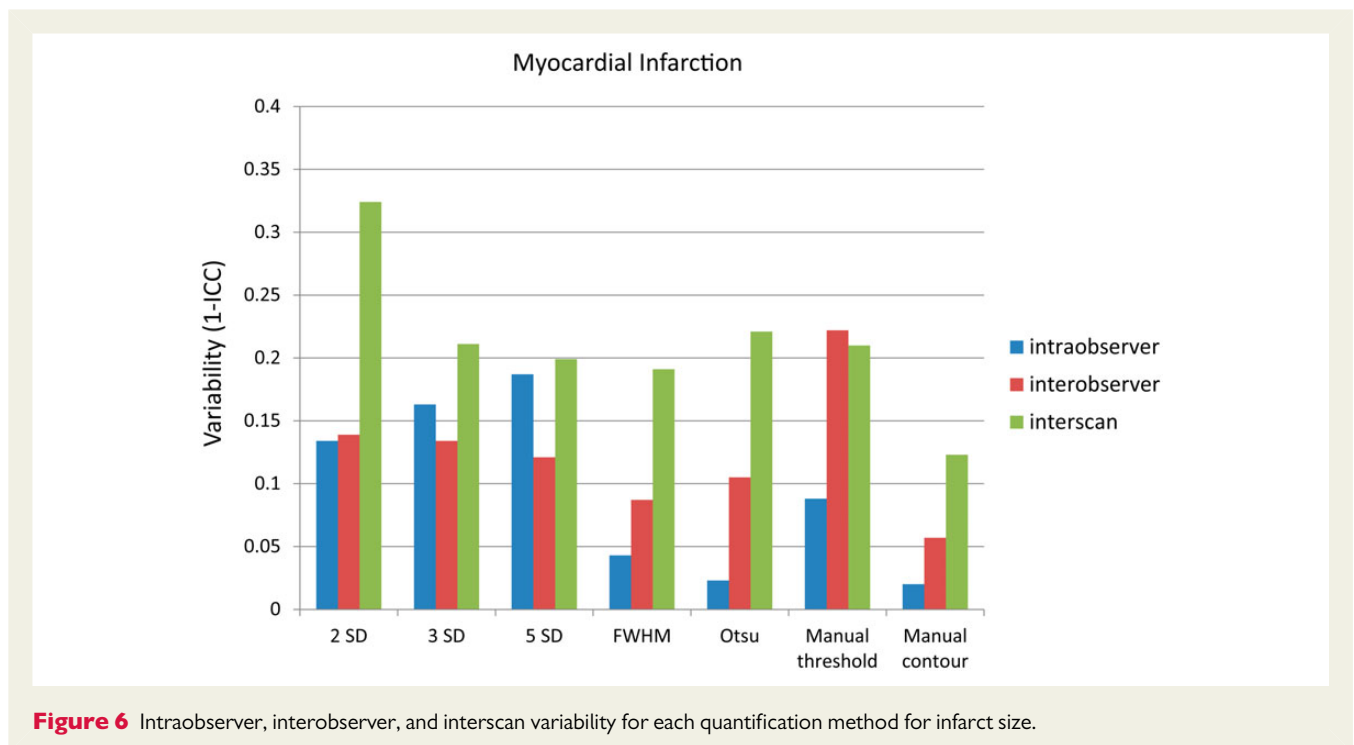
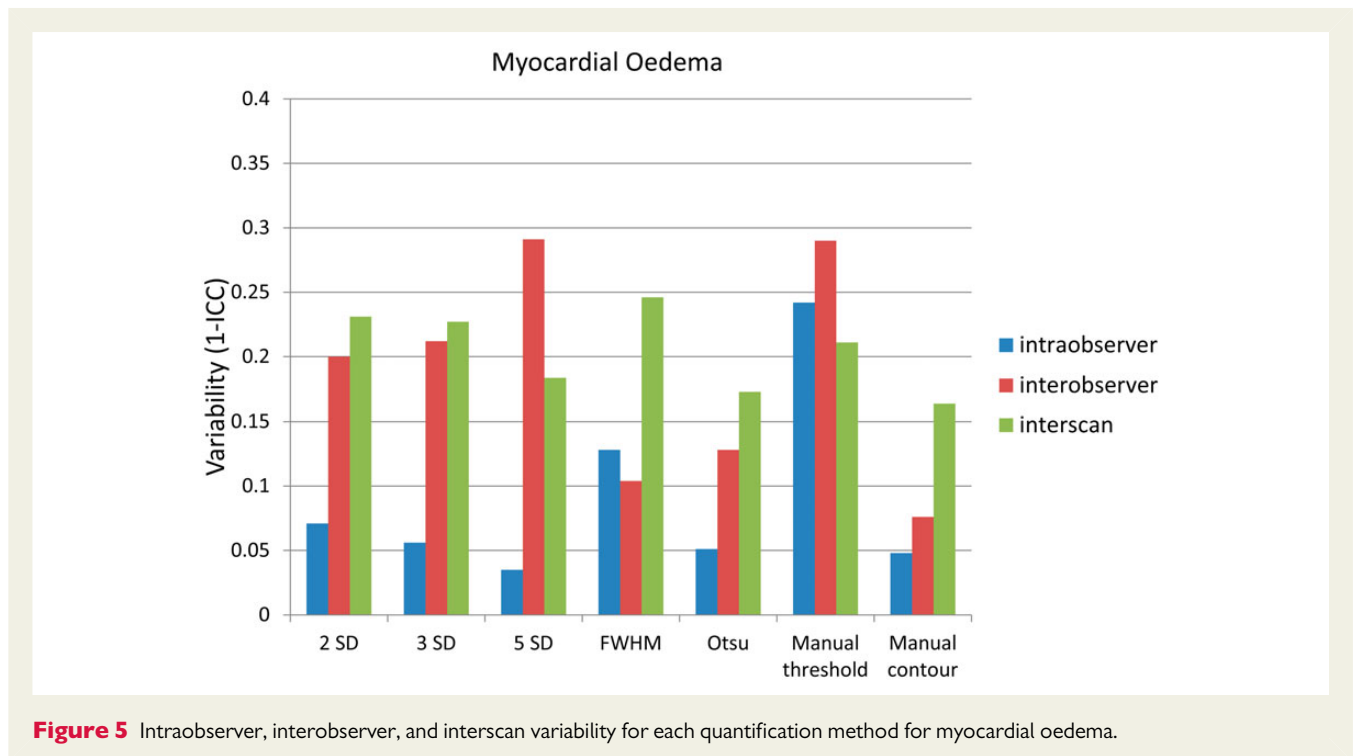
**Table 2** The correlation of myocardial oedema with AAR calculated by an angiographic score (APPROACH) in a subset of 23 patients

Quantification method	R-value	P-value
2 SD	0.31	0.15
3 SD	0.25	0.26
5 SD	0.06	0.78
FWHM	0.09	0.68
Otsu	0.35	0.11
Manual threshold	0.01	0.99
Manual contour	0.57	0.005

Manual contouring has the best correlation with AAR by APPROACH among the seven techniques tested.

may have had variation in the delineation of the endocardium and epicardium. Our study confirms that manual contouring by an expert observer is the most robust technique (when considering overall interobserver, intraobserver, and test–retest variability and agreement) for assessing myocardial oedema following STEMI, followed by the Otsu method.

Contouring pixels with a threshold of a certain number of SDs from the remote myocardium on T2w STIR has limitations when compared with using this technique in LGE imaging. There is a relatively lower contrast to noise ratio between normal and abnormal myocardium in T2w STIR<sup>28</sup> than that of the myocardium in LGE imaging.<sup>29</sup> This can possibly represent a limitation when applying a thresholding technique, and especially when using a low SD cut off from the unaffected myocardium. This can explain our findings of systematic bias with increasing bias at higher oedema mass with these methods. Additional challenges in analysing T2w STIR images are slow flow artefacts adjacent to regions of hypokinesia, which will also cause inaccurate results if not appropriately excluded.<sup>30</sup> In our study, we included regions with a hypointense core suggesting intramyocardial haemorrhage;<sup>2,31</sup> these regions, due to the heterogeneous signal distribution, are difficult to contour.



The FWHM threshold<sup>13</sup> uses an intensity threshold as  $>50\%$  of the peak SI within the ROI in the infarct. Therefore, this technique may overestimate the area of myocardial oedema due to the relatively lower contrast to noise ratio between normal and abnormal myocardium in T2w STIR. Additionally, the SD techniques rely on

placing an ROI in the remote myocardium; this has limitations again due to a relatively lower contrast to noise ratio between normal and abnormal myocardium and also can be overly affected by artefact of problem due to coil inhomogeneity. Again, this could explain our findings of interobserver systematic bias with thresholding

techniques with increasing bias at higher oedema mass. Our study confirms that the Otsu method is an acceptable alternative to manual contouring and this semi-automated technique has low overall variability. The Otsu method objectively estimates the intensity threshold from the histogram of all intensities to get minimal variance both above and below threshold. It does not require any ROI to be subjectively demarcated, which can be a source of error/artefact introduced into the other methods.

Overall, when considering the variability of a measurement, all three parameters (intraobserver, interobserver, and test–retest) variability are important in combination with accuracy. This study has no histological comparison data to comment on accuracy. However, perhaps the most important measurement is the test–retest repeatability, which has an important role when determining sample size for clinical studies. For myocardial oedema assessment, the test–retest variability is lowest for manual contouring followed by Otsu. For infarct size calculation, the test–retest variability is lowest for manual contouring followed by FWHM. This finding is in keeping with the study's conclusions if considering all three variability parameters.

## Limitations

The major limitation of this study is that there is no clinical gold standard for the assessment of myocardial oedema/infarct size and, being a clinical study, comparison with histological gold standards is unavailable and therefore accuracy cannot be assessed. However, when CMR findings are compared with biochemical markers of infarct size (troponin T) and angiographic measures of myocardial oedema (AAR), manual contouring has the best correlation (Tables 1 and 2). Serial troponin measurements would have been a better measure of infarct size than a single peak troponin; however, these measurements were unavailable in this study.

An ROI for the normal myocardium reference was drawn in the remote myocardium with no regional wall motion abnormalities for both infarct and oedema quantification methods. We did not use skeletal muscle as a reference for T2w STIR as the  $T_2$  values for skeletal muscle can alter depending on physiology and remote myocardium is widely used in the literature. In contrast to previous studies,<sup>32</sup> no patients were excluded for poor image quality in this study.

This was a single centre, single vendor study. Myocardial oedema was imaged and analysed using T2w STIR. Other newer oedema sequences appear promising<sup>33–35</sup> and may supersede T2w STIR.

## Conclusion

Different methods for oedema and infarct size quantification are not interchangeable. In acute MI, manual contouring has the lowest overall variability for quantification of both myocardial oedema and MI when analysed by experienced observers. The FWHM method for infarct size quantification and the Otsu method for myocardial oedema quantification are acceptable alternatives.

## Supplementary data

Supplementary data are available at *European Heart Journal – Cardiovascular Imaging* online.

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## IMAGE FOCUS

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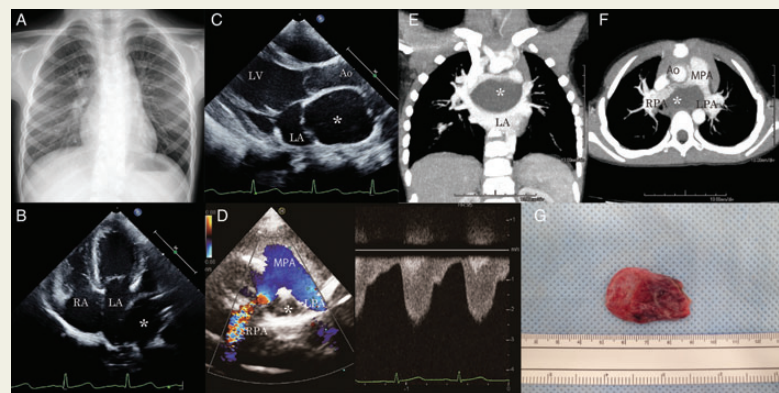
# Bronchogenic cyst compressing the pulmonary artery and the left atrium

Yasunobu Hayabuchi\*, Miho Sakata, and Shoji Kagami

Department of Pediatrics, Tokushima University, Kuramoto-cho-3, Tokushima 770-8503, Japan

\* Corresponding author: Tel: +81 886 33 7135; fax: +81 886 31 8697, Email: hayabuchi@tokushima-u.ac.jp

A 6-year-old boy was referred to our hospital because of the presence of a heart murmur. A chest roentgenogram did not demonstrate cardiomegaly or pulmonary congestion (Panel A). Echocardiographic examination revealed a membranous structure dividing the left atrium (Supplementary data online, Video S1). Both sides had the same echo density, indicating fluid density. Several possible diagnoses were initially possible, such as cor triatriatum, left atrial dissection, cardiac tumor, or an external mass. Colour Doppler examination showed no communication between the sides of the abnormal membrane, and there



was no blood flow within the upper side of the membrane (Supplementary data online, Video S2). Therefore, the examination revealed an extracardiac homogeneous mass located on the cranial aspect of the left atrium, compressing the pulmonary artery and left atrium. The mass was shown as an echolucent space with a smooth surface (Panels B and C, asterisk). The right pulmonary artery was compressed and the peak systolic velocity was increased to 2.40 m/s (Panel D). In addition, there was an unusual antegrade diastolic flow across this narrowing, which indicates vessel compression in both systole and diastole. A chest computed tomographic scan was subsequently performed showing the presence of a 45 × 35 × 30 mm, thin-walled homogeneous lesion, which was not contrast-enhanced, located in the middle mediastinum behind the pulmonary artery and in front of the descending aorta (Panels E and F). Surgery was conducted via a right posterolateral thoracoscopic approach in the left lateral position (Panel G). The histopathological examination of the section revealed a bronchogenic cyst.

Supplementary data are available at *European Heart Journal – Cardiovascular Imaging* online.