

Abstract 026 Figure 2 Bland Altman analysis and scatter plots for the assessment of peak velocity using all the acceleration methods.

pulsatile phantoms (Figure 1) and 25 volunteers. In volunteers, the CMR protocol included: cines, 2D phase contrast (PC) at the aortic valve (AV) and mitral valve (MV) and three whole-heart free-breathing (no respiratory motion correction) 4D flow CMR pulse sequences. Field-of-view, slices, phases (30), voxel size and VENC were the same for each subject. In volunteers, net acquisition time for each 4D flow sequence was recorded, as well as a visual grading of image quality on a four-point scale: 0, no artefacts to 3, non-evaluable.

Results For the pulsatile phantom experiments, the mean error against the reference flow by time beaker measurements for 4D-TFE was $4.9\% \pm 1.3\%$, for 4D-EPI $7.6\% \pm 1.3\%$ and for 4D-k-t BLAST $4.4\% \pm 1.9\%$. *In vivo*, acquisition time was shortest for 4D-EPI at $7 \text{ min } 59 \text{ s} \pm 2 \text{ min } 30 \text{ s}$. 4D-EPI and 4D-k-t BLAST had minimal artefacts, while for 4D-TFE, 40% of AV and MV assessments were non-evaluable because of phase dispersion artefacts. Peak velocity assessment using 4D-EPI demonstrated best correlation to 2D PC (AV: $r=0.78$, $p<0.001$; MV: $r=0.71$, $p<0.001$). Coefficient of variability (CV) for net forward flow (NFF) volume was least for 4D-EPI (7%) (2D PC:11%, 4D-TFE: 29%, 4D-k-t BLAST: 30%, respectively) (Figure 2, 3).

Conclusion Of the three 4D flow CMR methods tested, 4D-EPI demonstrated the least susceptibility to artefacts, good image quality, modest agreement with the current reference standard for peak intra-cardiac velocities and the highest consistency of intra-cardiac flow quantifications.

Competing interests The authors declare that they have no competing interests.

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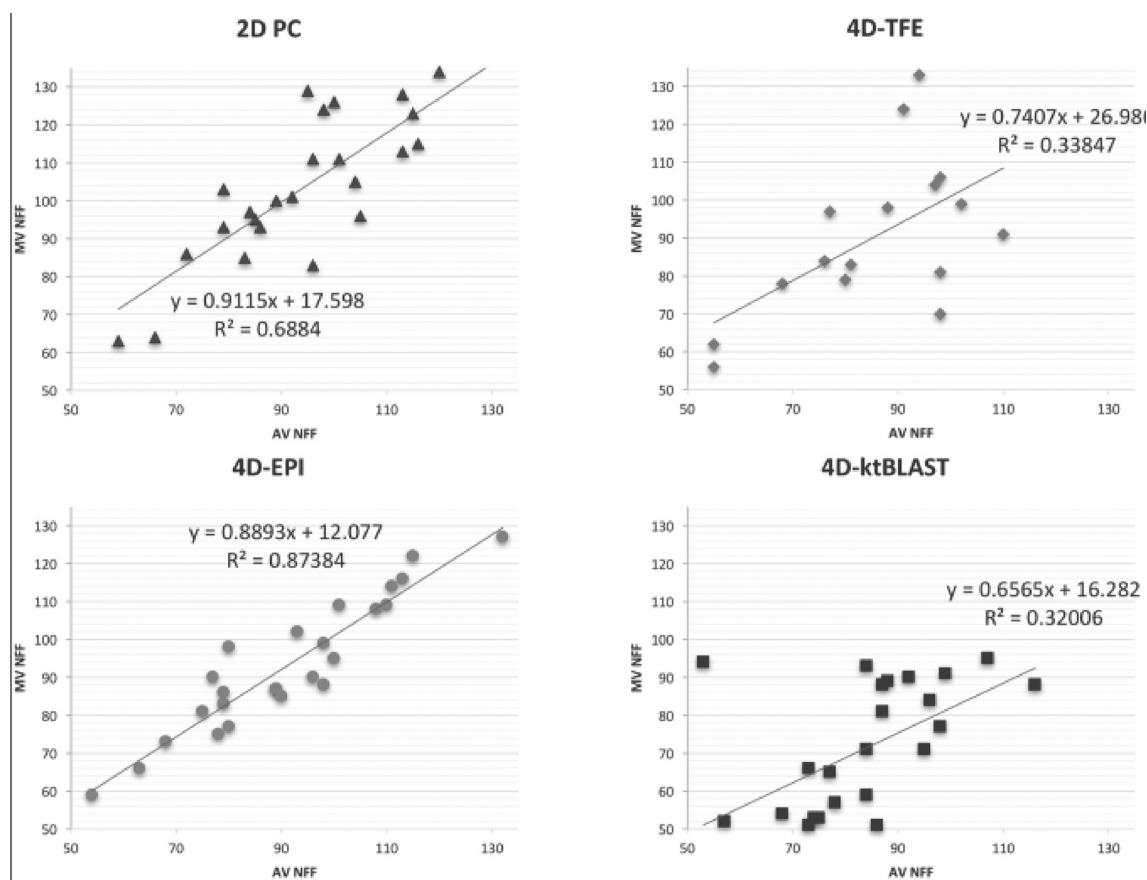
T2 MAPPING IN ACUTE AND RECOVERED MYOCARDITIS: POTENTIAL ROLE IN CLINICAL SURVEILLANCE

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Background Acute myocarditis (AM) remains a challenging diagnosis with poorly defined markers of chronic active disease and/or progression to dilated cardiomyopathy. T2 mapping allows quantitative assessment of low-level myocardial oedema¹ but requires further clinical evaluation alongside conventional biomarkers (troponin and BNP) prior to large-scale application.²

Aim To prospectively evaluate the role of T2 mapping in the clinical surveillance of acute myocarditis.



Abstract 026 Figure 3 Scatter plots of net forward flow (NFF) through the mitral and aortic valve to investigate consistency between all the four methods.

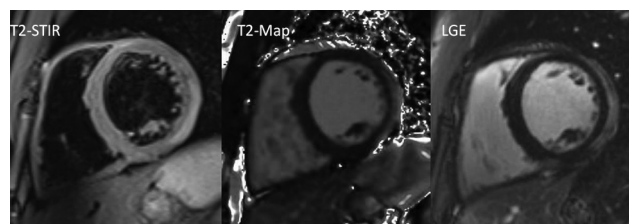
Methods T2 mapping was performed using T2-prepared balanced steady-state free-single shot-images on a 3 Tesla system (Skyra, Siemens) in the following patient groups:

- Prospective patients with AM defined by clinical presentation and 2 out of 3 Lake Louise Criteria (n=11, mean age 34 ± 10 years, all male) scanned at baseline and 3 months.
- Retrospective patients with a history of AM (n=12, mean age 35 ± 13 years, all male) scanned 5.7 ± 3.9 years from acute presentation.
- Healthy volunteers (n=9, mean age 27 ± 6 years, 56% male).

Troponin-I and BNP were measured at the time of CMR in all patients. A global region of interest was manually drawn by a single blinded observer in the basal short-axis slice (see figure 1).

Results In patients with acute myocarditis, mean global T2 was 42 ± 1.4 msec compared to 40 ± 1.3 msec in healthy volunteers ($p < 0.005$). No difference was found between T2 values at presentation and 3 month follow-up ($p = 0.1$), although the range of T2 values was greater at 3 months (see figure 2). In the retrospective group, mean T2 normalised to 40 ± 1.5 msec with preserved ventricular function, comparable to healthy volunteers. Troponin normalised at follow-up in all patients, whereas BNP remained elevated in 3 patients (mean 23 ng/mL).

Conclusion Our findings suggest that T2 values remain persistently elevated at 3 months following acute presentation despite normalisation of cardiac troponin levels. Increased variability in T2 values at 3 months is likely to have arisen from



Abstract 027 Figure 1 T2-STIR and T2 mapping at the basal short-axis level in a patient with acute myocarditis affecting the lateral wall. The late gadolinium enhancement image is provided for reference.

heterogeneity in tissue changes, unlikely to be reflected by standard AHA segments. Statistical tools such as mean absolute standard deviation (madSD)³ may improve the potential benefit of T2 mapping beyond single T2 cut-off values in this small but important subsets of patients.

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