T2-weighted magnetic resonance imaging to assess myocardial oedema

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To the editor: We read with interest the comprehensive review article on T2-weighted cardiovascular magnetic resonance imaging (CMRI) by Edwards et al.1 We wish to make one comment for the sake of clarity and also to bring to the reader’s attention important new data, which have become available in the meantime, and shed light on some of the issues raised by the authors. The authors cite one of the first reports employing T2-weighted CMRI to assess acute myocardial injury at a very early stage2 but fail to bring out one key aspect of that study. We used polyvinyl alcohol alcohol foam particles (contour emboli) rather than ethanol injections to induce therapeutic infarction in patients undergoing septal artery embolisation. As a result, these ischaemic insults were fairly comparable with the clinical event of atherothrombotic coronary occlusions. Although an elevated T2 signal was not consistently observable within the first 60 minutes after the onset of ischaemia, it was present on the next day and remained elevated for up to 4 weeks. Whereas this study demonstrated the ability of T2-weighted CMRI to identify acute myocardial injury, the ‘early blind spot’ was possibly related to the experimental design with embolisation of a very small coronary territory. Furthermore, the impact of such particles on the T2 signal was not investigated, but may have been a confounder.

Recently, Abdel-Aty et al.3 added a few more pieces to the jigsaw; one of the 108 dogs were instrumented with a snare around the first diagonal artery. Ischaemia duration was deliberately restricted so as to avoid relevant cell destruction (ie, infarction). While contractility was immediately affected (reflecting energetic depletion), the pathological T2 signal (reflecting tissue oedema) did not visually become apparent before 28±4 minutes after the onset of ischaemia and was further increased after reperfusion. The marked increase in T2 signal was paralleled by a relatively small, yet relevant and significant, increase in myocardial water content (~2%), probably reflecting cellular rather than interstitial oedema, with disruption of cell membranes. This experiment elucidates the T2-weighted CMRI profile of non-lethal ischaemia in a dog model with some, albeit inherently limited, transferability into the human scenario. Moreover, the relationships of the T2 signal with conditions such as stunning, repetitive ischaemia–reperfusion and peri-ischaemic inflammation are still waiting to be unravelled.

Incremental technical advances in T2-weighted CMRI will facilitate that and are expected to enhance our understanding of the ischaemia–reperfusion sequence and improve clinical decision-making.

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Competing interests None.

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The authors’ reply: We thank Bohl et al1 for their additional comments relating to the timing of T2-weighted imaging to detect acute ischaemia.2 They reference their own experimental data that has been instrumental in the development of T2-weighted cardiovascular magnetic resonance imaging (CMRI) and emphasise the methodological differences that might explain the failure to detect myocardial oedema within the first 24 h. This paper was important not only in raising the issue of the earliest time point for the detection of myocardial oedema but also for the long-term 180-day follow-up period assessing the duration of the changes detectable on T2-weighted CMRI. Recent data from animal models and clinical human studies have provided more information regarding the ‘time window’ in which the earliest appearances of myocardial oedema occur.3,4 The recent paper by Abdel-Aty et al,3 which became available during revision of our

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