correspond to the amount of peri-operatively infarcted myocardium as measured by delayed-enhancement cardiac MRI (DE-MRI). They found that 18 out of 23 (78%) patients studied demonstrated delayed hyper-enhancement (HE) on MRI, with a median mass of 4.4 g. While we applaud the use of state of the art imaging techniques to address fundamental questions regarding the pathophysiology of peri-operative myocardial injury, this study suffers from a number of methodological flaws.

Firstly, the lack of a pre-CABG MRI is a significant weakness in the study. It is highly likely that some of the 18 patients, had HE prior to surgery (despite the negative clinical history), and hence, both the incidence and magnitude of presumed peri-operative myocardial injury is significantly over-estimated in the study cohort. In a recent randomized study comparing on-pump and off CABG, where patients had systematic use of both pre- and post-CABG MRI scans and serial biochemical markers, we reported that 21 out of 55 (38%) patients had evidence of new HE quantified at 6.5 ± 4.1 g or 4.6 ± 2.9% of absolute LV mass. Furthermore, 30 (50%) patients in our cohort had pre-existing HE (i.e. HE in their pre-CABG scan), with 6 of these patients (20%) having HE in the absence of impaired LV function, prior history of MI, or ECG Q waves. Hence, serial imaging is vital in the design of studies of this nature.

Second, the selection of patients for post-operative MRI scans based on an elevated 12–18 h post-operative CK-MB concentration, as opposed to a consecutive series of CABG patients, potentially limits the applicability of the study results to general clinical practice.

Third, serial assessment of post-operative biochemical markers permits area under the curve (AUC) quantification of biochemical marker release post-surgery. It would be interesting to see if the correlation between biochemical marker release and new DE-MRI findings change if AUC analysis (over 3–5 days) rather than single time point measurements (i.e. <24 h) of CK-MB/Troponin measurements are made. We have previously reported in a series of 150 patients that the predominantly early pattern of cTnI release after on-pump cardiac surgery, with peak values observed within 6–12 h after cardiopulmonary bypass, was inconsistent with true myocardial necrosis (in which case peak levels would not be seen for 48–72 h) but more likely to represent a cytoplasmic 'washout' phenomenon. Finally, while the sub-endocardial and transmural distribution of (presumed) peri-operative HE seen in this study supports our recent findings, the patchy (sometimes epicardial) distribution found in some 7 out of 18 patients is surprising and has not been previously reported in the setting of coronary disease/vascularization.

References