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Letter to the Editor

Cardiac troponin I plasma levels for diagnosis and quantitation of perioperative myocardial damage in patients undergoing coronary artery bypass surgery

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Reply to: Volker Sadony, Michael Korber, Guido Albes, Volker Podtschaske, Thorleif Etgen, Thomas Trosken, Ursula Ravens, Max Ernst Schuelen. Cardiac troponin I plasma levels for diagnosis and quantitation of perioperative myocardial damage in patients undergoing coronary artery bypass surgery.

We congratulate Doctor Sadony and colleagues for their interest in cardiac troponin I as a marker of perioperative myocardial ischemia. This has been our main field of research since 1994. The authors confirm the conclusions of our works, the first of which was published in 1995.

In our first study [1], we demonstrated cardiac troponin I (CTn I) to be a marker of ischemia during human heart surgery. We compared CTn I release after aortic valve replacement in patients with normal coronary arteries (AVR) with release after coronary artery bypass grafting (CABG). CTn I concentration was significantly higher in the CABG group. In the AVR group, a positive correlation ($r = 0.6$, $P < 0.01$) was found between aortic cross-clamping time and CTn I release. There was no such relationship in patients undergoing CABG because, as opposed to AVR, where myocardial ischemia is only due to cardioplegic arrest, in CABG, it is multifactorial. These results were confirmed by an experimental study [2]. We measured CTn I release in three groups of six rat hearts perfused according to the Langendorff method, immediately after excision, after 3 h of immersion, and after 6 h of immersion in St. Thomas solution at 4°C. We observed a highly significant linear increase in CTn I release during the course of the three ordered periods of ischemia. These two studies showed CTn I to be a reliable marker of cardiac damage during heart surgery. One application of this characteristic was to compare different methods of myocardial protection with the idea that the best method would induce the lowest release of CTn I. We subsequently conducted a trial [3] in which 60 patients undergoing CABG were randomly assigned to one of two crystalloid cardioplegia routes of delivery: antegrade only or combined with retrograde. The combined route of delivery was shown to be more effective in patients with significant left main coronary artery stenosis, as shown by a significantly lower release of CTn I.

In a further trial [4], we randomly assigned 70 patients undergoing CABG either to cold crystalloid or to cold blood cardioplegia followed by warm reperfusion. The total amount of CTn I released was higher in the crystalloid group, showing that cold blood cardioplegia followed by warm reperfusion provided better myocardial protection than cold crystalloid cardioplegia in an unselected group of patients undergoing an elective first CABG, and having a preserved left ventricular function.

None of these different works directly linked to the subject of the article have been referred to or by Doctor Sadony and colleagues. This oversight in the review of the literature has lead the authors to state as new, findings which have already been reported.

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