



CUTOFF FOR HIGH-SENSITIVITY CARDIAC TROPONIN T IS NOT ARBITRARY BUT ACCORDING TO USUAL CLINICAL PRACTICE



To the Editor:

We were pleased to read a comment written by Bianchi¹ regarding our article previously published in *The Journal of Thoracic and Cardiovascular Surgery*.² Bianchi¹ raised several important issues related to the use of high-sensitivity cardiac troponin T (hs-cTnT) for the diagnosis of acute myocardial infarction (MI). First, we shall respond to his comment on the electrocardiographic and transthoracic echocardiographic approach. The combination of biomarkers with electrocardiographic or transthoracic echocardiographic criteria to diagnose perioperative MI, as suggested by the “Third Universal Definition of Myocardial Infarction,”³ is more specific and has a high negative predictive value for the diagnosis of perioperative MI after heart valve surgery than the use of a single criterion.^{1,2}

The elevation of hs-cTnT is a frequent finding in the postoperative period of uncomplicated cardiac surgery.⁴ This could be due to several factors that may provide some insights into the mechanism of perioperative MI after heart valve surgery: (1) direct trauma of the myocardium, which can be produced by sutures or direct manipulation of the heart; (2) myocardial ischemia related to an inadequate intraoperative cardioprotection; (3) microvascular embolisms related to reperfusion; and (4) myocardial damage induced by the release of oxygen free radicals.⁴ We found cutoffs for hs-cTnT and creatine kinase isoenzyme MB that could be diagnostic of perioperative acute MI after heart valve surgery by means of a rigorous methodology applied according to usual clinical practice and not arbitrarily.

Criteria for diagnosing perioperative MI after heart valve surgery are controversial, because chest pain is frequently removed by analgesics in these patients. To diagnose perioperative MI appropriately, a stable baseline of hs-cTnT before heart valve surgery is required, because if patients have elevated, rising hs-cTnT levels, it is not possible to distinguish this

accurately from a recent index MI.⁵ Accordingly, we excluded patients with recent medical history of coronary artery disease, and all patients included in our study had normal, stable baseline hs-cTnT. Data about preoperative hs-cTnT are not available in the article, although we think that this would have been very interesting. We agree with the editorial comment that the diagnosis could have been made earlier to institute the appropriate treatment.¹ The diagnosis of perioperative acute MI after heart valve surgery should be based on enzymatic curves of hs-cTnT and creatine kinase isoenzyme MB suggestive of ischemia, however, with peak values reached at similar times as described in literature for spontaneous MI, and also on electrocardiographic or transthoracic echocardiographic criteria, rather than solely on the elevation of the hs-cTnT within 3 hours after cardiac surgery, as Bianchi¹ suggests.

Finally, we consider that it would be advisable to group the efforts of several centers and compare their results with ours to conduct an external validation of the study. Our findings do improve on the criteria of the “Third Universal Definition of Myocardial Infarction”³ to diagnose perioperative MI early and with rigorously by providing new cutoffs for hs-cTnT and creatine kinase isoenzyme MB above the upper reference limit, with the highest sensitivity and specificity seen in patients with electrocardiographic or transthoracic echocardiographic criteria for MI after heart valve surgery.

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5. Miller WL, Garratt KN, Burritt MF, Lennon RJ, Reeder GS, Jaffe AS. Baseline troponin level: key to understanding the importance of post-PCI troponin elevations. *Eur Heart J*. 2006;27:1061-9.

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**PERIOPERATIVE
MYOCARDIAL
INFARCTION AFTER
HEART VALVE SURGERY,
WHERE ARE WE GOING?
Reply to the Editor:**



We are pleased to provide a response to the letter to the Editor entitled “Cut-off for High-Sensitivity Cardiac Troponin T Not Arbitrarily but Accordingly to Usual Clinical Practice,”¹ regarding our article previously published in the *Journal*.² In this letter to the Editor, Cubero-Gallego and colleagues¹ raised several important issues related to the combination of biomarkers with electrocardiographic (ECG) or transthoracic echocardiographic criteria to diagnose perioperative myocardial infarction (MI), the mechanism of perioperative MI after heart valve surgery, and the requirement of a stable baseline of high-sensitivity cardiac troponin T (hs-cTnT) before surgery to distinguish an acute event from a recent index MI.

First, we shall add a comment about the ECG approach because new 2017 European Society of Cardiology (ESC) guidelines for the management of acute MI with ST-segment elevation have been published.³ In these new ESC guidelines,³ patients with persistent ischemic symptoms and right bundle branch block should be considered to undergo an emergency coronary angiography and percutaneous coronary intervention if indicated.³ In our article previously published in the *Journal*,² ECG criteria for diagnosing perioperative MI after heart valve surgery included (1) new pathologic Q-waves and (2) new left bundle branch block, as suggested by the Third Universal Definition of MI to diagnose MI after coronary artery bypass grafting.⁴ Therefore, the incidence of perioperative MI after heart valve surgery reported in our article might be higher on the basis of the new ESC guidelines,³ because the onset of new right bundle branch block after heart valve surgery was not considered as an ECG criterion of perioperative MI.

Cutoffs for hs-cTnT and creatine kinase isoenzyme MB for diagnosing perioperative MI after heart valve surgery were reached at our study following a rigorous

methodology according to usual clinical practice^{1,2} and the Third Universal Definition of MI.⁴ However, the elevation of biomarkers is a frequent finding in the perioperative period of uncomplicated heart valve surgery, which could be due to several factors related to direct trauma of the myocardium, inadequate cardioprotection, and reperfusion injury.¹ Another clinical scenario where high levels of hs-cTnT have been reported is in patients undergoing noncomplicated cardiac surgery with end-stage renal failure.⁵ The interference of renal function with hs-cTnT levels might be explained because troponins are large macromolecules that are small enough to be cleared by healthy kidneys, but renal impairment leads to clearance decrease.⁵

Patients undergoing cardiac surgery are usually under treatment with drugs that may remove ischemic symptoms. Therefore, criteria for diagnosing perioperative MI after cardiac surgery should be based on the elevation of biomarkers (requiring a stable baseline of hs-cTnT before surgery) and following the new cutoffs for hs-cTnT and creatine kinase isoenzyme MB provided by Cubero-Gallego and colleagues,^{1,2} with enzymatic curves suggestive of ischemia, peak values reached at similar times for spontaneous MI, and ECG or transthoracic echocardiographic criteria (the onset of new pathologic Q-waves or new bundle branch block,^{3,4} or the imaging evidence of new wall motion abnormality).^{2,4}

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