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From Creatine Kinase-MB to Troponin: Do We Really Need to Differentiate between Myocardial Injury and Infarction?

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To the Editor

We commend Archan *et al.*¹ for their excellent review on creatine kinase-MB fraction and troponin for the diagnosis of perioperative myocardial infarction (MI) in noncardiac surgery patients. We, too, recently investigated the utility of creatine kinase-MB and cardiac troponin I for predicting clinically relevant myocardial injury in two cohorts of patients who had undergone coronary artery bypass surgery (N = 1,576).² Similar to the studies the authors¹ reviewed, we also found cardiac troponin I to be superior to creatine kinase-MB in its association with increased hospital length of stay and mortality.²

When creating a universal definition for MI guidelines, the Joint European Society of Cardiology, American College of Cardiology, American Heart Association, and World Heart Federation Task Force identified five clinical classifications, although MI associated with coronary artery bypass surgery is the only category for perioperative MI.³ From a mechanistic point of view, noncardiac surgical perioperative MI would likely be classified as a type 2 MI, “myocardial infarction secondary to ischemia due to either increased oxygen demand or decreased supply, for example coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, or hypotension.”⁴

As Archan *et al.*¹ correctly note, the universal definition requires a combination of biomarker elevation and angina symptoms, electrocardiogram, imaging, or angiography to diagnose MI. This definition is problematic in the perioperative setting, however, because angina symptoms are not reliable in patients undergoing general anesthesia and receiving analgesics and sedatives. In addition, as a diagnostic tool, electrocardiogram is often not sensitive enough to detect ischemia—particularly after cardiac surgery.² Therefore, even in the absence of outcome data, we suggest using troponin as the primary criteria for identifying clinically significant myocardial injury in all perioperative settings.

We respectfully disagree with Archan *et al.*¹ regarding the relevance of differentiating between myocardial injury and myocardial infarction. They correctly point out that troponin elevation may result from a variety of etiologies, including physiologic stress associated with marathon running or mountain climbing.⁵ Furthermore, the extraordinary sensitivity of currently available biomarker assays permits detection of a single troponin molecule release even after minimal exercise.⁶ At present, however, imaging modalities and cellular detection technology are unable to differentiate between troponin release from the cytosol or damaged cells that are likely to recover (myocardial injury) and irreversible cellular necrosis

(myocardial infarction). Therefore, we suggest that increased concentrations of circulating troponin, in fact, reflect a spectrum of myocardial injury. Consequently, the assignment of a specific cutoff point in an attempt to differentiate between injury and infarction may be counterproductive to efficient identification of therapeutic interventions.

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