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Reticulocyte Hemoglobin Content in Critically Ill Patients

To the Editor:

We read with interest the article by Fernandez *et al.*[1] Reticulocyte hemoglobin content (CHr) is a promising marker of iron metabolism, particularly in the intensive care unit setting where usual markers are often disrupted by inflammation. In a small cohort of critically ill patients with no evidence of real iron deficiency, we observed a correlation between C-reactive protein and CHr, suggesting that inflammation rapidly reduces iron availability for erythropoiesis.[2] We agree that CHr should be used in future research protocols to monitor the response to iron therapy in critically ill patients. However, CHr measurements are not routinely available in most hospitals. In our study, blood samples had to be stored on ice and sent to an external laboratory within 72 h.[2] In the study by Fernandez *et al.*,[1] it would have been interesting to know the iron status of the patients based on the ferritin concentration, serum iron concentration, and transferrin saturation to ensure that there was no coexistence of true iron deficiency. Also, because CHr is the product of cellular volume and cellular hemoglobin concentration, other variables such as mean cellular volume may have affected the CHr values.[3,4] In our study, two patients had a high CHr value (≥35 pg) probably secondary to their high mean cellular volume values (more than 100 fl).[2] CHr seems to be useful to predict transfusions or to monitor iron therapy but should be interpreted in the context of folates or vitamin B_{12} deficiencies that may coexist in critically ill patients.[4,5]

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The above letter was sent to the authors of the referenced report. The authors did not wish to reply. —James C. Eisenach, M.D., Editor-in-Chief.

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

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 authors[1] 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,

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