The Case for a Revised Definition of Myocardial Infarction—The Ongoing Conundrum of Type 2 Myocardial Infarction vs Myocardial Injury

The Task Force for the Universal Definition of Myocardial Infarction published international consensus documents simultaneously in the Journal of the American College of Cardiology, the European Heart Journal, and Circulation in 2007 and 2012. The 2007 and 2012 documents were updated revisions of the original document, published by this task force in 2000.

From the very outset, the task force has sought a clinical definition consistent with the pathological definition for myocardial infarction (MI). Accordingly, the clinical definition of infarction requires tissue necrosis secondary to severely reduced or absent blood circulation. Thus, an MI as defined by the task force involves myocardial cell death due to prolonged ischemia. As stated in the 2000 document, an MI caused by coronary artery occlusion begins to develop after approximately 15 to 30 minutes of severe ischemia and progresses from the subendocardium to the subepicardium in a time-dependent fashion (the wave-front phenomenon).

In the 2 most recent task force publications, various types of MIs were defined. A type 1 MI was defined as an MI resulting from atherosclerotic coronary artery disease with an occluded or partially occluded coronary artery secondary to an atherosclerotic plaque rupture or fissuring. Patients with type 1 MI were defined as individuals presenting with the typical clinical picture for MI.

However, patients meeting the MI criteria without having coronary thrombi require a different management strategy compared with those with coronary occlusion. The task force created the category of type 2 MI, which is defined as myocardial necrosis resulting from a marked increase in myocardial oxygen demand or a marked decrease in myocardial blood flow leading to myocardial ischemic necrosis. Patients might have atherosclerotic coronary artery disease or they might have angiographically normal coronary arteries.

In this respect, we agree with Nagele when he states that we have broadened the definition of MI in the 2007 and 2012 documents by putting more emphasis on acute myocardial ischemia that results in necrosis by adding the category of type 2 MI. However, we lack the earlier criterion of acute coronary artery occlusion. The term coronary artery occlusion was included in the pathology section in the 2000 document but omitted in the corresponding sections in the 2007 and 2012 documents. Thus, we have extended the definition of MI by adding type 2 MI in the 2007 and 2012 documents and by writing more about myocardial ischemia than about coronary occlusion in these latter documents.

To narrow the MI definition to the same extent as in the 2000 definition, it seems appropriate to lay greater emphasis on the concept of myocardial injury, whether ischemic or nonischemic. Moreover, a narrower definition of MI and a broader application of the term myocardial injury would mirror the nomenclature of infarction and injury of other organs, such as acute kidney injury. Importantly, a more restricted MI definition would certainly diminish the confusion of physicians regarding type 2 MI.

Patients with type 2 MI often have serious comorbid illnesses during which the heart is presumably injured as an innocent bystander. However, many of these individuals have not had clinically important changes in their myocardial supply-and-demand status. The task force has defined these latter patients as not having experienced a type 2 MI. Rather, they are said to have had a myocardial injury in the setting of a serious comorbid condition. Suggested causative factors of myocardial necrosis include elevated circulating levels of inflammatory cytokines, such as tumor necrosis factor-α, as well as catecholamines. Since the publication of the 2012 document, there has been a considerable volume of clinical investigations defining the incidence and clinical course of type 2 MIs and other nonischemic myocardial injuries.

As pointed out by Nagele in his commentary, and as we have experienced as well, the multiple clinical settings, the complexity of the patients involved, and the potential for differing interpretations of the universal MI definition document by physicians have resulted in some uncertainty about what constitutes a type 2 MI vs a myocardial injury. To some extent, we agree with Nagele that it might be less confusing to include all of these events—both type 2 MIs and myocardial injuries—under a single term such as acute myocardial injury. This more general definition would resemble the syndrome of an acute kidney injury, which may accompany the findings of a type 2 MI or myocardial injury in seriously ill medical and surgical patients.

There will still be problems with coding for this new entity because it is not a defined term within the International Classification of Diseases, Ninth Revision or Tenth Revision. Work has already begun to introduce this terminology into future coding systems. In the immediate future, the Task Force for the Universal Definition of Myocardial Infarction will meet again to consider revisions to be included in the fourth revised document, with publication most likely in 2017 or 2018. We will take Nagele’s insightful comments to this meeting. We believe that his
more general definition for acute myocardial injury, whether ischemic or nonischemic in complex medical and surgical patients, requires serious further study and consideration. The term *acute myocardial injury* seems a likely solution that would narrow the definition of MI and would be similar to the terminology used with injury to other organs.

**ARTICLE INFORMATION**

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