**Biomarkers in Acute Cardiac Disease**

In their state-of-the-art paper on biomarkers in acute cardiac disease (1), Dr. Jaffe and colleagues list creatine kinase-MB (CK-MB) as a “potentially outdated marker.” However, CK-MB has a specific utility in the diagnosis of reinfarction (2), and it cannot be replaced by the cardiac troponins for this purpose. By following up the time course of rise and fall of CK-MB, an interruption in the progressive decline in the level of the biomarker (to levels below upper reference limit) can be detected (2–4). Re-elevations in CK-MB by more than 50%, can be used to diagnose re-infarctions as early as 18 h after the index event (2). Both cardiac troponin T (cTnT) and cardiac troponin I (cTnI) on the other hand are continuously released from degenerating contractile apparatus in necrotic cardiomyocytes and may show persistent elevations, 7 to 10 days in the case of cTnI and up to 10 to 14 days in the case of cTnT, after the index event (2). The protracted time course of kinetic release of cTnI and cTnT limit their ability to diagnose reinfarction even several days after the index ST-segment elevation myocardial infarction (STEMI) because the cardiac troponin levels will still be on the rise during this period as a result of their normal kinetics, and it is not possible to be sure whether the rise is due to a re-infarction or not. It is because of this important difference in the kinetics between CK-MB, which shows a rapid rise and fall, and the troponins, the American College of Cardiology/American Heart Association Practice guidelines for STEMI specifically state that CK-MB is superior for diagnosing reinfarction even several days after the index event (2). This is very relevant as superimposed on the time course of rise and fall of CK-MB, detects cardiac injury in many situations which are not due to primary coronary abnormalities. Thus, many patients with elevations do not require the extensive workups Dr. Fye correctly indicates can occur if one obviates common sense. Some may need an evaluation for pulmonary embolism or myocarditis and some only watchful waiting. Some of this can occur in the outpatient setting in our view, but we often see cardiologists assume that once the patient is discharged that they can forget about elevations. Neglecting such a potent risk factor for mortality is not good common sense either. Regardless of the etiology, elevations of troponin are indicative of significant cardiovascular disease and usually are associated with an adverse prognosis in the short term (2) and over time once recovery occurs (3) and in those who are more compensated (4). When such elevations occur in critically ill individuals, first efforts should be focused on the primary disease process which very often is the stimulus for the cardiac injury. Additional work is necessary to distinguish when we as cardiologists should address cardiac issues acutely over and above treating the underlying disease state. Many of us are actively involved in trying to define such subsets at present. Whether or not acute intervention is needed, it is clear from most studies that elevations of troponin also predict adverse long term events (3,4). Thus, those who were critically ill and recover and those in whom “incidental” elevations of troponin were detected, require careful evaluation. That could mean an evaluation for ischemic heart disease but as Dr. Fye suggests, it is prudent to consider other etiologies for elevations as well.

It is also good common sense to upgrade our clinical judgment periodically. It is thus prudent in considering the possibility in a given patient of ischemic heart disease to take note of information concerning the lack of perfection of the angiogram, (5) differences in the way in which women present with infarction, (6) and the recent article in the Journal of the American College of Cardiology suggesting the high frequency of unrecognized myocardial infarctions detected in older individuals by MRI (7).

The issue of reinfarction is one where we have used common sense. The state of the art and guidelines are not the same. The later are often much more conservative. It was presumed in the initial studies, that increases in CKMB post-infarction were indicative of reinfarction. There was no independent validation of this. There has been no validation of the use of increases on the