Comparative sensitivities and specificities of the
mass measurements of CK-MB2, CK-MB, and
myoglobin for diagnosing acute myocardial
infarction

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Timothy Pelletier,1 and Magdalena Usategui3,4

We evaluated the clinical utility of the mass measurement
of the tissue isoform of creatine kinase MB isoenzyme
(CK-MB2) in the diagnosis of an acute myocardial infarction
(AMI) by determining its sensitivity, specificity, and
predictive value relative to those of CK-MB mass and
myoglobin. Samples were obtained at 0, 4, 8, and 16 h
postpresentation from 100 patients (41% with AMI). The
order of sensitivity for the sample proportions taken at 0–2 h
from the onset of symptoms was myoglobin > CK-MB2 >
CK-MB. At all other time points, the sensitivity of CK-
MB2 either equaled or surpassed that of both CK-MB and
myoglobin, although the 95% confidence intervals for the
population proportions each of these markers overlapped.
Of the 41 AMI patients, 31 (76%) exhibited concurrent
abnormal increases of CK-MB and %CK-MB2; the other
10 (24%); 8 non-Q wave, 2 Q wave) exhibited abnormal
values for %CK-MB2 before their CK-MB exceeded the
upper limit of normal. The specificity of myoglobin was
statistically lower than that for either CK-MB2 or CK-MB
at all time points.

INDEXING TERMS: laboratory management • diagnostic efficien-
cy • isoenzymes • isoforms

Many patients presenting to hospital emergency departments
for the evaluation of chest pain have symptoms and electrocar-
diograms that are both nonspecific and inconclusive [1, 2].
During the past 30 years, the development of sophisticated
serum assays for specific proteins has greatly assisted in the
diagnosis of both acute and subtle manifestations of coronary
artery disease. Despite the technological advances in diagnostic
laboratory medicine and noninvasive cardiac imaging, however,
the etiology of acute chest pain remains a difficult diagnostic
problem [3].

Currently, the diagnosis of acute myocardial infarction
(AMI), as defined by the World Health Organization (WHO), is
based on the presence of any two of the following in the patient:
clinical features and chest pain; electrocardiographic changes;
and abnormal values for creatine kinase (CK) and the MB
isoenzyme of creatine kinase (CK-MB) [4]. Although the tem-
poral changes in the serum concentrations of CK-MB are
relatively specific for an AMI, the lack of complete tissue
specificity of this enzyme often results in a misdiagnosis (false-
ly positive) of myocardial infarction [5]. Additionally, the reported
lack of diagnostic sensitivity of this marker early in the course
of an AMI precludes its use as a means of early diagnosis [6]. Given
the aggressive surgical and medical interventions associated with
an evolving AMI, and the cost of hospitalization of non-AMI
patients with acute chest pain syndrome until the presence
of myocardial damage has been excluded [2, 7], the need for an
accurate and rapid diagnosis of AMI is crucial for the proper,
effective, and efficient management of these patients. As a result,
several biochemical markers have been proposed as potential
supplements to the measurement of CK-MB to assist in the
diagnosis of AMI. Three such proteins are myoglobin, the
tissue-associated isoform of CK-MB (CK-MB2), and troponin.

Myoglobin is found in the cytosolic fraction of both cardiac
and skeletal muscle tissue. In the absence of skeletal muscle
trauma or other factors associated with non-cardiac-related
release of myoglobin [8, 9], rapid and pronounced increases in
myoglobin serum concentrations have been used as an early
marker for AMI. Several investigators have reported the rapid
increase of serum myoglobin after AMI, with peak values occurring between 6 and 9 h after the occlusion of a coronary artery [10, 11].

CK-MB exists in serum as a polymorphic mixture of isoforms [12, 13] produced by the catabolism of CK-MB2, the tissue form of CK-MB [14]. Puleo et al. have proposed [15] that an assay of CK-MB isoforms sufficiently sensitive to detect diagnostic changes in the CK isoform composition in serum when concentrations of CK-MB are normal would clearly be of clinical utility. Currently, all of the reported methods for separating and quantifying the isoforms of CK-MB [15-20] are based on the analysis of the activity of CK-MB2 and (or) CK-MB1 and reportedly lack either the low-end sensitivity or the specificity [21, 22] required for the rapid and accurate diagnosis of AMI.

We recently reported the development of a sensitive and specific immunochemical method for direct measurement of the mass of CK-MB2 in serum [23]. Here we report our evaluation of the clinical utility of the mass measurement of CK-MB2 for detecting an AMI, determined by analyzing the sensitivity and specificity of this isoform assay, relative to the values for CK-MB and myoglobin, in serial samples from patients presenting to the Emergency Department at the Memorial Hospital of Rhode Island within 6 h from the onset of symptoms suggestive of an AMI.

**Materials and Methods**

**Patients and Samples**

Patients presenting with symptoms consistent with myocardial ischemia or infarction of <6 h in duration were eligible for inclusion in this study. Additionally, the initial suspicion of infarction had to be of sufficient magnitude to merit hospital admission to either the coronary care unit, the telemetry unit, or the observation unit. Patients with a prior history of a documented AMI within 2 weeks of admission and patients receiving thrombolytic therapy were eliminated from this study.

Of the 100 patients retained in the study, 61 were men and 39 were women; their median age was 70 years. The mean time from onset of chest pain to presentation was 2.3 h.

Blood samples from study participants were drawn at 0, 4, 8, and 16 h after presentation; the serum was processed, frozen, and stored at −80 °C within 60 min of collection, according to established laboratory procedures. Patient inclusion and sample acquisition were conducted in accordance with the policies and procedures of the Institutional Review Board for the Use of Human Subjects in Research at the Memorial Hospital of Rhode Island. All enrolled patients requiring phlebotomy in excess of normal hospital protocol provided additional written informed consent.

**Clinical Diagnosis**

The triage and care of all patients were conducted by physicians who were unaware of the assay results for CK-MB2 and myoglobin, and the serum samples were analyzed by laboratory personnel in a manner that was blinded to the patients' diagnosis and outcome. The clinical diagnosis of AMI was determined by the patient’s admitting physician and the primary physician (often a cardiologist) and confirmed by at least one full-time cardiology faculty member (Department of Cardiology, Brown University School of Medicine, Providence, RI) at the Memorial Hospital of Rhode Island, in accordance with established WHO criteria [4]. The type of infarction (non-Q wave or Q wave) was determined from the electrocardiogram.

**Analytical Methods**

**CK-MB and myoglobin assays.** Serum CK-MB and myoglobin mass concentrations were determined by immunofluorometric assays (Dade International, Miami, FL) based on two-site immunometric sandwich methods using two monoclonal antibodies. The upper limits of normal for these assays were determined to be 6 and 110 µg/L, respectively, in a healthy adult population.

**Troponin T assay.** Serum troponin T concentrations were determined by using the CardiAC T ELISA on the ES 300® analyzer (Boehringer Mannheim, Indianapolis, IN). The performance of this method, relative to its high specificity for cardiac injury [24], has been reported by Ravkilde et al. [25]. The upper limit of normal for this assay was 0.2 µg/L in a healthy adult population.

**CK-MB2 assay.** We determined serum concentrations of CK-MB2 as previously described [23]. The upper limit of normal for the CK-MB2 assay was determined to be 3 µg/L of CK-MB and 70% as CK-MB2 in both hospitalized noncardiac patients and healthy ambulatory adults.

**Effect of storage temperature on analyte stability.** Six patients' samples, drawn into siliconized glass blood-collection tubes, were analyzed for initial (0 h) CK-MB2 concentration. The samples were aliquoted; stored at 4 °C, room temperature (25 °C), and 37 °C; and reanalyzed for CK-MB2 concentration after 6 h of storage. All values obtained represent the mean of three determinations.

**Statistics**

The results of serial CK-MB and CK-MB2 determinations were correlated with the eventual clinical diagnosis. The sensitivity (true-positive rate in patients with infarction) and specificity (true-negative rate in patients without infarction) were determined for both assays at each of the postpresentation time points analyzed. A serum marker was judged to be positive if its concentration in any of the four specimens was greater than the cutoff limit. The positive predictive value was defined as the number of true-positive results divided by the total number of positive results, and the negative predictive value was defined as the number of true-negative results divided by the total number of negative results. The 95% confidence intervals for the population proportions were calculated from the sample proportions by using the appropriate upper-tail probability [26]. A paired t-test was used to compare numerical data obtained from the CK-MB2 stability study.

**Results**

**Stability of CK-MB2**

We investigated the in vitro stability of CK-MB2 in serum samples stored at various temperatures. As shown in Table 1,
losses of CK-MB2 after 6-h incubations at either 4 °C (P > 0.24) or 25 °C (P > 0.39) were statistically insignificant. However, after 6 h at 37 °C, CK-MB2 was significantly degraded (P < 0.025). These results suggest that (a) the carboxypeptidase-mediated conversion of CK-MB2 is inhibited at temperatures of 25 °C or less, (b) CK-MB2 concentrations can be determined in serum, and (c) serum samples can be stored at either room temperature or refrigerated for up to 6 h without significant loss of CK-MB2 concentration.

CLINICAL EVALUATION
Because of the strict inclusion criteria for the patients from whom serial serum samples were obtained, the prevalence of AMI in this study group was fairly high, 41 of 100. Of these, 29 had non-Q wave and 10 had Q wave infarction. Another 19 patients had unstable angina, and 10 had stable angina. Additionally, relatively few (in comparison with general hospital populations) patients in this group were diagnosed with noncardiac chest pain: 30 of 100.

The relative sensitivities and specificities of CK-MB2, CK-MB, and myoglobin for the diagnosis of an AMI are summarized in Table 2. Using as upper limits of normal for CK-MB, CK-MB2, and myoglobin values of >3 μg/L (as CK-MB) and 70% MB2, 6 μg/L, and 110 μg/L, respectively, we determined the order of diagnostic sensitivity for the samples taken at 0-2 h from the onset of symptoms as: myoglobin > CK-MB2 > CK-MB. At all other time points, the sensitivity of CK-MB2 equaled or surpassed that of both CK-MB and myoglobin, although the 95% confidence intervals overlapped for the population proportions for each of these markers.

Of the 41 AMI patients, 31 (76%) exhibited concurrent abnormal increases of CK-MB and %CK-MB2, and 10 (24%: 8 non-Q wave, 2 Q wave) exhibited abnormal values for %CK-MB2 before demonstrating CK-MB values in excess of the upper limit of normal. Also, the sensitivity of CK-MB was adversely affected by one patient who demonstrated a transient increase in CK-MB concentration within the normal range. Similarly, the sensitivity of myoglobin was adversely affected by another patient who demonstrated decreasing but “normal” myoglobin values over this timeframe. The specificity of myoglobin was statistically lower than that for either CK-MB2 or CK-MB at all time points.

Table 3 describes the positive and negative predictive values of CK-MB, CK-MB2, and myoglobin for the diagnosis of an AMI. In general, the positive predictive value of myoglobin was significantly less than that of either CK-MB2 or CK-MB. Additionally, the negative predictive value of CK-MB2 was 100% at 8-10 h after the onset of symptoms. However, the positive predictive values obtained for all three tests may be favorably biased because of the relatively low number of nonAMI patients in this study group.

The specificities of CK-MB2, CK-MB, and myoglobin for each diagnostic category were determined. CK-MB2 was increased in 4 of the 30 noncardiac chest pain patients, 0 of the 10 stable angina patients, and 5 of the 19 unstable angina patients. CK-MB was increased in 2 of the 30 noncardiac chest pain

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<tr>
<th>Table 1. Stability of CK-MB2.</th>
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<td>Patient</td>
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* P > 0.24; # P > 0.39; % P < 0.025

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<tr>
<th>Table 2. Relative sensitivities and specificities of CK-MB, CM-MB2, and myoglobin for diagnosis of AMI.</th>
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<td>Sensitivity/specificity, % (and 95% confidence interval)</td>
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<td>Time from onset, h</td>
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<td>0-2</td>
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<th>Table 3. Positive and negative predictive values of CK-MB, CM-MB2, and myoglobin for diagnosis of AMI.</th>
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<td>Predictive value, %: positive/negative (and 95% confidence interval)</td>
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<tr>
<td>Time from onset, h</td>
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patients, 0 of the 10 stable angina patients, and 1 of the 19 unstable angina patients. Myoglobin was increased in 13 of the 30 noncardiac chest pain patients, 3 of the 10 stable angina patients, and 8 of the 19 unstable angina patients.

Because of the relatively large number of unstable angina patients with increased CK-MB2 values (26.3%) and the reported ability of CK-MB isoforms to detect myocardial damage in noninfarct clinical conditions [27], patients diagnosed with unstable angina, angina, or noncardiac chest pain were examined for independent evidence of ischemic myocardial damage by a commercially available troponin T assay [27, 28-30]. Serum concentrations of troponin T exceeded the reference interval (>0.2 μg/L) in one patient diagnosed with noncardiac chest pain, and in four patients diagnosed with unstable angina. In two of these patients (one diagnosed with noncardiac chest pain and one with unstable angina), CK-MB2, CK-MB, and myoglobin were also increased. The remaining three patients diagnosed with unstable angina exhibited abnormal increases of both CK-MB2 and myoglobin. Thus, given the high probability that myocardial damage was present in these patients, the relative sensitivities, specificities, and predictive values for the diagnosis of potential myocardial injury were as shown in Tables 4 and 5.

**Discussion**

**STABILITY OF CK-MB2**

The relative stability of the isoforms of CK remains controversial; it is not clear how quickly degradation occurs either in vivo or in vitro. Several investigators have indicated the need for preservatives to maintain the integrity of isoform activity. Nohara et al. [31] added ethylene glycol-bis(β-aminoethyl ether) at a final concentration of 7.5-10 mmol/L and mercaptoethanol at 5 mmol/L to the sample to maintain activity. Chapelle [32] collected samples for isoform analysis in tubes containing disodium EDTA, 5 mmol/L. Additionally, Davies et al. [33] reported the loss of CK-MB2 activity in the absence of 15 mmol/L EDTA and β-mercaptoethanol, as measured by the Helena REP® electrophoresis system (Helena Labs., Beaumont, TX). Their data indicated a significant difference between the relative stability of CK-MM3 and CK-MB2 activity under identical storage conditions. Other investigators have reported successful analysis of isoforms of CK in the absence of a preservative [34-37]. Previously, Prager et al. [38], using anion-exchange chromatography and a monospecific antibody, reported that CK-MB2, in contrast to the tissue isoform of CK-MM (CK-MM3), undergoes slow cleavage of lysine in vivo to form CK-MB1. Our results are in agreement with those of Prager et al., but neither study directly addresses the stability of the activity of CK-MB2. Rather, the data suggest that the mass concentration of this isoform is stable for limited times in isolated samples when measured by specific monoclonal antibodies.

**CLINICAL EVALUATION**

The similar release kinetics of CK-MB2 and CK-MB precludes the use of the isoforms of CK-MB2 as a sensitive marker of myocardial damage early in the course of an AMI. Furthermore, although the diagnostic sensitivity of CK-MB2 may be slightly greater than that of CK-MB for diagnosing an AMI in a small subset of patients, the majority of our patients exhibited abnormal increases of both CK-MB and CK-MB2 at identical times after the onset of chest pain. Thus, determination of the serum concentration of CK-MB2 in all patients with acute chest pain is of limited utility. Additionally, myoglobin was shown to lack sufficient diagnostic specificity.

| Table 4. Relative sensitivities and specificities of CK-MB, CM-MB2, and myoglobin in diagnosing potential myocardial injury. |

<table>
<thead>
<tr>
<th>Time from onset, h</th>
<th>CK-MB</th>
<th>CK-MB2</th>
<th>Myoglobin</th>
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<tbody>
<tr>
<td>0-2</td>
<td>6.5 (0.0, 13.6)/100 (100)</td>
<td>17.4 (6.4, 28.4)/100 (100)</td>
<td>21.7 (9.8, 33.6)/92.6 (85.6, 99.6)</td>
</tr>
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<td>2-4</td>
<td>10.9 (1.9, 19.9)/98.1 (94.5, 100)</td>
<td>32.6 (19.1, 46.1)/100 (100)</td>
<td>30.4 (17.1, 43.7)/83.3 (73.4, 93.2)</td>
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<td>4-6</td>
<td>67.4 (53.9, 80.9)/98.1 (94.5, 100)</td>
<td>80.4 (68.9, 91.9)/98.1 (94.5, 100)</td>
<td>78.3 (66.4, 90.2)/72.2 (60.3, 84.1)</td>
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<tr>
<td>6-8</td>
<td>84.8 (74.4, 95.2)/98.1 (94.5, 100)</td>
<td>93.5 (86.4, 100)/96.3 (91.3, 100)</td>
<td>91.3 (83.2, 99.4)/64.8 (52.1, 77.5)</td>
</tr>
<tr>
<td>8-10</td>
<td>91.3 (83.2, 99.4)/98.1 (94.5, 100)</td>
<td>97.8 (93.6, 100)/92.6 (85.6, 99.6)</td>
<td>95.6 (89.7, 100)/64.8 (52.1, 77.5)</td>
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<tr>
<td>10-12</td>
<td>91.3 (83.2, 99.4)/98.1 (94.5, 100)</td>
<td>100 (100)/92.6 (85.6, 99.6)</td>
<td>97.8 (93.6, 100)/64.8 (52.1, 77.5)</td>
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| Table 5. Positive and negative predictive values of CK-MB, CM-MB2, and myoglobin in diagnosing potential myocardial injury. |

<table>
<thead>
<tr>
<th>Time from onset, h</th>
<th>CK-MB</th>
<th>CK-MB2</th>
<th>Myoglobin</th>
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<tbody>
<tr>
<td>0-2</td>
<td>100 (100)/55.7 (46.0, 65.4)</td>
<td>100 (100)/58.7 (49.0, 68.4)</td>
<td>71.4 (62.5, 80.3)/58.1 (48.4, 67.8)</td>
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<td>2-4</td>
<td>83.3 (76.0, 90.6)/56.4 (46.7, 66.1)</td>
<td>100 (100)/63.5 (54.1, 72.9)</td>
<td>60.9 (51.3, 70.5)/58.4 (48.7, 68.1)</td>
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<tr>
<td>4-6</td>
<td>96.9 (93.5, 100)/77.9 (69.8, 86.0)</td>
<td>97.4 (94.3, 100)/85.5 (78.6, 92.4)</td>
<td>70.6 (61.7, 79.5)/79.6 (71.7, 87.5)</td>
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<tr>
<td>6-8</td>
<td>97.5 (94.4, 100)/88.3 (82.0, 94.6)</td>
<td>95.6 (91.6, 99.6)/94.5 (90.0, 99.0)</td>
<td>68.9 (59.8, 78.0)/89.7 (83.7, 95.7)</td>
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<td>8-10</td>
<td>97.7 (94.8, 100)/93.0 (88.0, 98.0)</td>
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<td>97.7 (94.8, 100)/93.0 (88.0, 98.0)</td>
<td>92.0 (86.7, 97.3)/100 (100)</td>
<td>70.3 (61.3, 79.3)/97.2 (94.0, 100)</td>
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These conclusions are in general agreement with those published by other investigators. Gibler et al. [39] reported that the sensitivity of CK-MB immunochemical methods were 50-62.1% on emergency department presentation and increased to 92.0-96.7% 3 h later; these tests also demonstrated specificities of 81.0-96.4% at 3 h. In a study of 189 patients presenting to the emergency department within 12 h from the onset of chest pain and without a history of trauma, renal failure, or muscular dystrophy, Brogan et al. [40] reported the sensitivities of CK-MB and myoglobin at presentation to be 23% and 55%, respectively, increasing to 73% and 82%, respectively, at 3 h postpresentation. Both CK-MB and myoglobin were highly specific (98-100%) at presentation and 3 h after presentation. In a similar study of 133 patients, Tucker et al. [41] reported the sensitivity, specificity, negative predictive value, and positive predictive value of myoglobin at presentation to be 44%, 96%, 81%, and 82%, respectively. These values increased to 78%, 94%, 91%, and 83% after 4 h. CK-MB was slightly less sensitive (34% and 63% at presentation and 4 h) and had a lower negative predictive value (79% at presentation and 87% at 4 h) than myoglobin but was more specific (99% and 100% at presentation and 4 h) with a higher positive predictive value (88% at presentation and 100% at 4 h). More importantly, in a prospective study of 37 patients with an AMI presenting within 4 h after the onset of chest pain, Mair et al. [42] found that differences in the early sensitivities of myoglobin, CK-MB mass, and CK isoform (activity) ratios were small and of little clinical relevance. Given the differences in the demographics, the inclusion and exclusion criteria, the diagnoses of the patients in these studies, the analytical methods used, and the relative inaccuracy in the time of onset of chest pain, the results of these studies appear to be in agreement.

Previous investigators have indicated that the isoforms of CK-MB are both more sensitive and more specific than CK-MB for the diagnosis of AMI. Puleo et al. [16] determined the sensitivity of the CK-MB2/CK-MB1 ratio to be 8%, 56%, and 96% at 2, 4, and 6 h after onset of symptoms, respectively, compared with the sensitivity of CK-MB (0%, 23%, and 48% at the same times). Although our results may suggest a minor improvement in the sensitivity of CK-MB2 relative to CK-MB in a small subset of patients with acute chest pain, we did not observe the differences in sensitivity and specificity noted by Puleo et al. This apparent inconsistency may be caused in part by differences between the assay methods used for CK-MB2 and CK-MB. We found the isoform immunochemical mass assay [23] to be equivalent to the activity assay for measuring and quantifying CK-MB2 in patients’ sera when present in high concentrations. Thus, our anti-B subunit capture and anti-(M + lysine) subunit conjugate assay system measures all of the CK-MB isoform associated with the CK-MB2 electrophoretic band. In both assay systems, the exact nature of the B subunit of CK-MB2 relative to the presence or absence of a terminal lysine group is unknown. However, as was evident in the previously described quantification of CK-MB2 within the normal reference interval [23], the immunochemical mass assay for CK-MB2 utilized herein is more sensitive than the activity-based assay previously reported by Puleo et al. [16]. This increased sensitivity also does not appear to result from equivalent assays with different reference ranges, because the upper limit of normal for %CK-MB2 (70%) as determined by the immunochemical assay is much higher than that used (CK-MB2/CK-MB1 ratio of 1.5, corresponding to a %CK-MB2 of 60%) in the activity assay. Thus, if the two assay systems were equivalent for measuring low concentrations of CK-MB2, the mass determination would exhibit a lower diagnostic sensitivity than the existing CK-MB2 activity method.

Of equal importance in the comparison of these studies is the nature of the CK-MB assay used. Our results suggest that the differences between the sensitivities of CK-MB2 and CK-MB are substantially less than those reported by Puleo et al. This is probably a direct result of the use of a highly sensitive mass assay for CK-MB in our study rather than the relatively insensitive activity-based method they used.

Our results indicate that CK-MB2 may have a potential role in diagnosing minor myocardial injury. A similar observation was recently reported by Hossein-Nia et al. [27]. Thus, CK-MB isoforms, like troponins T and I, may be a sensitive marker of myocardial cell damage in patients in whom the extent of ischemia is less overt than that associated with AMI. However, because of the limited number of patients examined in these studies, more-extensive analyses involving unstable angina patients are required to substantiate this potential role.

Financial support for this project was provided, in part, by Roche Diagnostic Systems, Branchburg, NJ, and Dade International, Miami, FL. In addition, troponin T reagents were provided by Boehringer Mannheim, Indianapolis, IN.

References


