Cardiac troponin I measurement with the ACCESS® immunoassay system: analytical and clinical performance characteristics

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We evaluated the ACCESS® cardiac troponin I (cTnI) immunoassay as a marker for myocardial infarction (MI). Total imprecision was 6.0% to 13.5%, the minimum detectable concentration was 0.007 μg/L, and the limit of quantitation was 0.046 μg/L. Comparison of cTnI measurement between the ACCESS and Stratus systems (n = 114) showed a proportional difference: ACCESS cTnI = 0.0996 Stratus cTnI + 0.049 μg/L (r = 0.811). Fifty-nine of 61 ambulatory patients without cardiac symptoms had no detectable cTnI (95% range, 0.00 to 0.025 μg/L). The optimum cutoff for discriminating MI (n = 289, 45 with MI) was 0.15 μg/L by receiver operator characteristic curve analysis; at this cutoff, the ACCESS cTnI assay showed a sensitivity of 88.9% (95% CI, 79.7–98.1%) and specificity of 91.8% (95% CI, 88.4–95.2%). The ACCESS cTnI assay results showed 89.4% and 93.0% concordance with the MB isoenzyme of creatine kinase (CK-MB) mass and Stratus cTnI results, respectively, for classification of patients with suspected MI. The ACCESS cTnI assay appears to show sensitivity and specificity comparable with those of both CK-MB mass and Stratus cTnI assays for the diagnosis of MI in patients presenting within 12 h of onset of symptoms.

According to the National Heart Attack Alert Program, an estimated 1.25 million Americans suffer acute myocardial infarction (MI)6 each year. Of these, 500 000 die and 700 000 MI patients are hospitalized. Further, >3 million additional patients are admitted to medical centers for whom MI eventually is ruled out. Despite this conservative approach, MI goes undiagnosed in ~30 000 to 50 000 individuals each year, accounting for the largest source of malpractice dollars in US emergency departments [1].

Biochemical markers of myocardial injury are considered the “gold standard” for the diagnosis of MI [2] and are particularly important in nondiagnostic electrocardiogram patients, a group accounting for 40–76% of MI patients at presentation [3]. The MB isoenzyme of creatine kinase (CK-MB) represents the benchmark for comparison with other biochemical markers because the characteristic rise and fall of CK-MB in serial measurements is nearly pathognomonic for MI [4]. By using CK-MB mass assays and a strategy that included sampling at presentation and after 3 h, 6 h, and 9 h, Gibler et al. [5] documented a diagnostic sensitivity of 100% and diagnostic specificity of 98.3% for MI diagnosis. CK-MB, however, is not a perfect marker because in populations with low disease prevalence, such as chest pain evaluation centers, the predictive value of a positive result is low [5]. Also, depending on the methodology used for measurement, as long as 8–12 h may be required after myocardial injury before the diagnosis of MI can be made with high sensitivity and specificity [4]. In addition, CK-MB is not cardiac specific [6]; this lack of tissue specificity is particularly problematic for interpretation in patients with concomitant myocardial and skeletal muscle injury [6–8]. The inadequacies of biochemical markers currently in use have generated

* Nonstandard abbreviations: MI, myocardial infarction; CK-MB, MB isoenzyme of creatine kinase; cTnI, cardiac troponin I; HCMC, Hennepin County Medical Center; UTSMC, University of Texas Southwestern Medical Center; UMAB, University of Maryland at Baltimore; CI, confidence interval; and CRF, chronic renal failure.

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considerable interest toward development of more cardiac-specific markers such as cardiac troponin I (cTnI).

cTnI is part of a new generation of biochemical markers that provides an additional clinical tool for assessment of the "acute coronary syndromes," a term that describes the continuum of myocardial injury ranging from angina, or so-called "reversible" ischemia, to Q-wave MI and definite tissue necrosis. Functionally, troponin I is a 24-kDa structural protein that interacts with troponin T, 37 kDa, and troponin C, 18 kDa, as part of the three-member troponin complex that is essential for contraction of striated muscle in both cardiac and skeletal tissue [9]. Troponin I molecules from cardiac and skeletal muscle have different amino acid sequences; antibodies directed at these different sequences are the basis for development of cardiac-specific immunoassays [10]. Studies have indicated that cTnI is a more specific marker in cases involving skeletal muscle injury and renal failure [11]. Also, different cTnI immunoassays have demonstrated excellent potential for clinical use in the diagnosis of MI [12–14]. Therefore, cTnI may have an important role in real-time strategies for evaluating acute coronary syndrome patients, an area that has been of intense interest, discussion, and study over recent years [15–17].

In this study, we evaluated the analytical and clinical performance for MI diagnosis of the ACCESS cTnI assay, intended for real-time measurement in clinical laboratories.

Materials and Methods
This was a multicenter prospective study conducted at Hennepin County Medical Center (HMC), Minneapolis, MN; the University of Texas Southwestern Medical Center (UTSMC), Dallas, TX; and the University of Maryland at Baltimore (UMAB), Baltimore, MD. The protocol was approved by each center’s Institutional Review Board.

PATIENT SAMPLES
Patients without cardiac symptoms. To estimate the reference range for the ACCESS cTnI assay, we studied 61 ambulatory patients scheduled for same-day surgery. None of these patients had evidence of cardiac, renal, or skeletal muscle disease.

Suspected MI patients. Serum samples from a total of 289 patients presenting within 12 h of acute chest pain suggestive of cardiac ischemia were included in the study, 45 (16%) of whom were diagnosed as having MI according to WHO criteria [18]. Table 1 lists the number of patients enrolled and the methods and markers measured at each site. Although multiple methods and markers were measured at the participating centers, only Stratus cTnI values were used clinically at HMC; CK-MB mass and total CK activity were used clinically at UTSMC and UMAB.

The mean age of the patient population was 54 years, with a range of 24–90 years; 55% of the patients were men; 39% were Caucasian, 49% were African American, 4% were Hispanic, and the remaining 8% were of other ethnicity. All patients had two or more serum samples collected within 24 h of presentation. Although the timing of blood collection was dependent on the individual hospital’s protocol, all patients had specimens collected at presentation and at least 6–10 h later. Specimens were either analyzed within 8 h of collection or stored frozen, at −70 °C, then thawed once just before analysis.

All sites in this study performed ACCESS cTnI testing. Clinicians determining the diagnosis of patients in this study were blinded to the ACCESS cTnI results. Chart reviews were performed by members of the investigation team at each site. As indicated in Table 1, two of the three sites performed CK-MB mass analysis, and two sites performed Stratus cTnI testing on suspected MI patients. Thus, the numbers of patients in each of the clinical performance evaluations are slightly different.

Skeletal muscle injury/disease. cTnI concentration was measured in specimens from 58 patients with skeletal muscle diseases (e.g., polymyositis) or skeletal muscle damage (e.g., trauma) having a total CK activity >1000 U/L. Trauma patients with evidence or suspicion of myocardial injury were excluded.

Chronic renal failure (CRF). Fifty-five CRF patients, all of whom had serum or plasma creatinine values >80.0 mg/L, were enrolled for testing. CRF patients with concomitant cardiac disease were excluded from this study.

<table>
<thead>
<tr>
<th>Study site</th>
<th>No. of patients</th>
<th>Cardiac marker assays</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>MI</td>
</tr>
<tr>
<td>HMC⁴</td>
<td>120</td>
<td>21</td>
</tr>
<tr>
<td>UMAB⁴</td>
<td>88</td>
<td>9</td>
</tr>
<tr>
<td>UTSMC⁴</td>
<td>81</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>289</td>
<td>45</td>
</tr>
</tbody>
</table>

* Diagnosis of MI was made according to WHO criteria [18].
* Only the Stratus cTnI assay was clinically used for diagnosis of MI. Clinicians were unaware of the ACCESS cTnI and ACCESS CK-MB results.
* CK-MB mass and total CK activity were used by clinicians for diagnosis of MI. Clinicians were unaware of the ACCESS cTnI results.
* CK-MB mass and total CK activity were used for diagnosis of MI. Clinicians were unaware of the Stratus cTnI and ACCESS cTnI results.
CARDIAC MARKER MEASUREMENTS

ACCESS cTnI. The ACCESS cTnI Immunoassay (Beckman Instruments) is a two-site immunoassay using monoclonal antibodies described previously [19]. The ACCESS immunoassay system is a bench-top random-access and continuous immunoassay analyzer that uses chemiluminescent detection [20]. cTnI can be measured with on-analyzer time to first result of 15 min. All specimens in this study were analyzed in duplicate with the ACCESS cTnI assay.

Stratus cTnI. As indicated in Table 1, cTnI was also assayed with the Stratus II system (Dade International) according to the manufacturer’s instructions at the HCMC and UTSMC sites. The characteristics of this two-site immunoassay have been described previously [21]. The assay time for the Stratus II system is 10 min. The minimum detection limit is 0.35 μg/L; the cutoff for MI detection used for comparison in this study was 1.5 μg/L, as determined from clinical studies described in the manufacturer’s package insert. Typical interassay CVs were 7% at cTnI concentrations of 2.0 μg/L, 8% at 1.5 μg/L, and 15% at 0.7 μg/L [22].

Stratus CK-MB. CK-MB mass was assayed with the Stratus II system at the UMAB site in accordance with the manufacturer’s instructions (Table 1). The detection limit is 0.4 μg/L, and the upper limit of the reference range determined from evaluation studies done at UMAB is 6.6 μg/L (Stratus CK-MB reference range study on 138 non-cardiac ICU patients at the University of Maryland Medical Center, unpublished data); however, 7.0 μg/L was used clinically at the UMAB site and in this study.

ACCESS CK-MB. The ACCESS CK-MB mass assay was performed by the HCMC site in this study (Table 1). This assay is based on two-site immunoassay technology with monoclonal antibodies described previously [23]. The upper limit of the reference range was determined from clinical studies of noncardiac hospitalized patients as 8.5 μg/L (data on file); the minimum detectable concentration is 0.3 μg/L.

ANALYTICAL PERFORMANCE

Effect of dilution. Three different sample pools were diluted with equal volumes of assay diluent provided by the manufacturer as recommended. The undiluted and the diluted specimens were analyzed in triplicate, and the recoveries were calculated. An average recovery within 10% of expected values was considered acceptable.

Precision study. ACCESS cTnI imprecision was examined following National Committee for Clinical Laboratory Standards protocol EP5-T [24]. Three concentrations of quality-control materials from Beckman Instruments were analyzed in triplicate, in two separate assays per day for 10 days, over a 20-day period, with use of a single calibration curve. The data were analyzed by using one-stage nested ANOVA to derive within-assay, between-assay, and total imprecision.

Detection limit. The detection limit was defined as the lowest cTnI concentration corresponding to a signal 2 SD above the mean of 10 replicates of the zero calibrator. This was evaluated in four assays, each using different calibration curves and reagent packs. The analytical signal equivalent to 2 SD above the mean signal of the zero calibrator was translated into cTnI concentration by interpolation between the zero and 0.1 μg/L calibrators.

Limit of quantification. Precision was assessed in serial dilutions of a serum with known cTnI concentration both with diluent, supplied by the manufacturer, and with a cTnI-negative serum from a healthy individual. The limit of quantification was defined as the lowest concentration having a within-run CV ≤20%.

cTnI assay correlation. The relation between cTnI measurements with the ACCESS and Stratus systems was compared in 502 samples after exclusion of specimens with cTnI amounts outside the linear reportable range of the test methods. Of the 502 samples, 114 (22.7%) had results both above each cTnI assay’s detection limit and within each respective assay’s dynamic range; 122 (24.3%) had one of the two results below the detection limit, and 266 (53.0%) had both results below the respective detection limits. Regression analysis was performed for results that were both above the detection limit and within the dynamic range for each assay (n = 114).

Evaluation of ACCESS cTnI cutoff for MI. ROC curves were constructed for the peak ACCESS cTnI concentrations attained within 24 h of presentation in samples collected prospectively from all 289 patients (Table 1). The first result of duplicate testing was used for all analyses.

Comparison between ACCESS cTnI and other markers for MI diagnosis. Clinical performance of the ACCESS cTnI assay was compared with CK-MB mass measurements by the Stratus or ACCESS systems. As indicated in Table 1, 208 patients were included in this analysis, 30 of whom were diagnosed as having MI. After categorization according to each assay’s cutoff, Stratus CK-MB mass and ACCESS CK-MB mass results were pooled to represent the performance of CK-MB mass in data analysis.

Performance of the ACCESS cTnI assay was also compared with results for the Stratus cTnI system in 201 patients, 36 of whom were diagnosed as having MI, as indicated in Table 1.

Peak concentrations within 24 h of presentation were used for analysis of the ACCESS cTnI assay and other markers. Diagnostic sensitivity and specificity were calculated at each marker or assay’s respective cutoff concentration. Agreement between two markers or assays in
classifying patients based on dichotomized test results being positive (above CK-MB mass or cTnI cutoffs) or negative (below CK-MB mass or cTnI cutoffs) for MI was analyzed by using the McNemar test [25].

ACCESS cTnI and Stratus cTnI data in serial specimens were plotted for eight MI patients, all of whom had four or more specimens collected for >24 h after presentation.

**STATISTICAL ANALYSIS**

Correlation between ACCESS cTnI and Stratus cTnI assays was analyzed by least-squares regression. The McNemar test was used for comparison of correlated proportions such as diagnostic sensitivity and diagnostic specificity data between markers or assays. The McNemar test was also used to evaluate groups of paired dichotomous results [25]. Briefly, the McNemar test evaluates whether the number of patients with paired results that are discrepant, i.e., positive/negative or negative/positive, are distributed evenly between the two assays. When the McNemar test shows statistically significant “uneven” distribution of the discrepant results, performances of the two assays are considered different. For a two-sided test with a significance of 0.05, the sample size used in this study would be sufficient to detect a difference up to 15% in specificity and sensitivity between two markers with a power of 0.8 [26]. All statistical tests were two-tailed, with significance set at \( P < 0.05 \).

**Results**

**ANALYTICAL PERFORMANCE**

Data for the ACCESS cTnI assay listed in Table 2 shows total imprecision ranging from 6.0% to 13.5% for the three quality-control materials used in this study.

<table>
<thead>
<tr>
<th>cTnI conc., ( \mu g/L )</th>
<th>Within assay</th>
<th>Between assays</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SD</td>
<td>CV, %</td>
<td>SD</td>
</tr>
<tr>
<td>0.19</td>
<td>0.010</td>
<td>5.1</td>
<td>0.024</td>
</tr>
<tr>
<td>6.66</td>
<td>0.209</td>
<td>3.1</td>
<td>0.45</td>
</tr>
<tr>
<td>28.63</td>
<td>0.689</td>
<td>2.4</td>
<td>1.56</td>
</tr>
</tbody>
</table>

ACCESS cTnI <1.0 \( \mu g/L \) and Stratus cTnI <10.0 \( \mu g/L \).

The subset of data with ACCESS cTnI values <0.25 \( \mu g/L \) and Stratus cTnI values <2.5 \( \mu g/L \) were not significantly correlated (\( r = 0.156 \)).

Figure 2 shows serial cTnI data from eight MI patients, all of whom had sampling over >24 h after presentation. Note that panels A, B, C, and D of Fig. 2 show parallel patterns for the ACCESS and Stratus cTnI methods; panels A, B, and D also show similar cTnI increases, expressed as multiples of the diagnostic cutoff. On the other hand, panels E, F, G, and H show patterns of rise and fall that differ substantially in their ACCESS and Stratus cTnI values.

**CLINICAL PERFORMANCE EVALUATION**

ACCESS cTnI for 61 ostensibly healthy ambulatory individuals were undetectable in 59 (96.7%; 95% CI, 92.2–100%); cTnI values were 0.03 and 0.10 \( \mu g/L \) in the remaining two.

Figure 3, top, shows ACCESS cTnI data for the overall population of 289 patients enrolled in the study (Table 1); the corresponding ROC curve for these data is displayed
in Fig. 3, bottom. Analysis of this ROC curve yielded an optimum cutoff of 0.15 μg/L for the diagnosis of MI with the ACCESS cTnI assay. At this 0.15 μg/L cutoff, the diagnostic sensitivity was 88.9% (95% CI, 79.7–98.1%), and the diagnostic specificity was 91.8% (95% CI, 88.4–95.2%) for the ACCESS cTnI assay.

Diagnostic sensitivity and specificity for the ACCESS cTnI and CK-MB mass assays were compared by using
each marker’s respective cutoff in the 208 patients indicated in Table 1. Diagnostic sensitivity was 90% (95% CI, 79.3–100%) for both markers; diagnostic specificity was 93.8% (95% CI, 90.3–100%) for ACCESS cTnI and 91.6% (95% CI, 87.5–100%) for CK-MB mass (P >0.05, power = 0.8).

The agreement in classifying patients as either MI or non-MI with the ACCESS cTnI and CK-MB mass markers was examined with the McNemar test (see Materials and Methods). The markers were in agreement for 186 (89.4%) of the 208 patients. Among the 22 patients having discordant results, 9 (1 MI and 8 non-MI) were ACCESS cTnI positive (>0.15 μg/L) but CK-MB mass negative (<7.0 μg/L); the remaining 13 (1 MI and 12 non-MI) were ACCESS cTnI negative but CK-MB mass positive. There was no statistically significant difference between the ACCESS cTnI and CK-MB mass makers by the McNemar test (power = 0.8).

Diagnostic sensitivity and specificity of the ACCESS cTnI and Stratus cTnI assays were compared for 201 patients (Table 1) by using respective cutoff values of 0.15 and 1.5 μg/L. With these cutoffs, diagnostic sensitivity was 86.1% (95% CI, 74.8–97.4%) for both cTnI assays; diagnostic specificities were 92.1% (95% CI, 88.0–97.4%) for the ACCESS cTnI assay and 94.5% (95% CI, 91.1–97.4%) for the Stratus cTnI assay (no statistically significant difference; power = 0.8).

Agreement between the ACCESS cTnI and Stratus cTnI assays with regard to classifying individual patients as either MI or non-MI was examined in 34 of the 36 MI patients and in 153 of 165 non-MI patients (93.0% agreement overall). Among the 14 patients having discordant results shown in Fig. 4, 9 patients (1 MI and 8 non-MI) were ACCESS cTnI positive (>0.15 μg/L) but Stratus cTnI negative (<1.5 μg/L); five patients (1 MI and 4 non-MI) were ACCESS negative but Stratus positive. Note that in Fig. 4, the magnitude of difference between the ACCESS and Stratus discordant cTnI results was substantial; all but two results were beyond the 95% CI of each respective assay’s cutoff. There was no statistically significant difference between the cTnI assays in diagnostic performance by the McNemar test (power = 0.8).

Figure 5 shows cTnI concentrations in specimens from 113 patients with either skeletal muscle disease/injury or CRF, each of which was measured with both the ACCESS and Stratus assays. Four patients with skeletal muscle disease having Stratus cTnI results >1.5 μg/L also showed ACCESS cTnI results >0.15 μg/L. There was no statistically significant difference between the two cTnI assays in these CRF or skeletal muscle injury patients (power = 0.8).

**Discussion**

Biochemical markers, in particular CK-MB, have become the “gold standard” for diagnosis of MI over the past two decades [2]. cTnI is part of a new generation of cardiac markers that provides a more tissue-specific diagnostic tool for the laboratory-assisted diagnosis of MI patients and possibly for risk stratification of patients within the continuum of acute coronary syndromes [22, 27]. For the diagnosis of MI, the ideal cTnI assay must have both good analytical characteristics, as indicated by accuracy (correlation), recovery, and precision, as well as good clinical performance, showing discrete separation between MI and non-MI patients. In this multicenter study, we examined analytical and clinical characteristics in suspected MI patients for a new cTnI assay available on the ACCESS Immunoassay system.
The ACCESS cTnI assay demonstrated acceptable analytic performance based on our examination of detection limit and limit of quantification, imprecision, and cTnI recovery. The detection limit, the minimum concentration that can be distinguished from zero, was in the range of 0.01 $\mu$g/L. Such a low limit is favorable, this characteristic having been shown to be important in clinical applications such as risk stratification [22]. The limit of quantification of the ACCESS assay, indicating the minimum cTnI concentration having within-assay imprecision $\leq 20\%$ CV, was 0.046 $\mu$g/L, well below the determined cutoff for MI diagnosis.

The ACCESS and Stratus cTnI assays were highly correlated ($r = 0.811$); however, there was a 10-fold difference between cTnI results, as indicated by both the slope of 0.0996 and the comparable diagnostic sensitivity and specificity at a cutoff of 0.15 $\mu$g/L for the ACCESS system and 1.5 $\mu$g/L for the Stratus. This apparent 10-fold difference in assays measuring the same analyte clearly points out a need for standardization. In addition, however, there was substantial residual scatter indicated by the large $s_{yx}$, suggesting that some fundamental factor other than a straightforward difference in standardization between the two assays might be involved. This issue will be discussed further later.

Specificity of the ACCESS cTnI assay was examined in noncardiac populations, which included ambulatory surgery patients, patients with skeletal muscle disease or...
injury, and patients with CRF. For ambulatory surgery patients, cTnI was undetectable in 96.7% of the subjects with the ACCESS cTnI assay. The two ambulatory patients in whom cTnI was detectable had values of 0.03 and 0.10 μg/L, substantially lower than the cutoff value of 0.15 μg/L for MI diagnosis. Although cTnI is rarely increased in conditions other than cardiac injury [27,28], high cTnI results have been observed among CRF and skeletal muscle damage patients [29,30]. Therefore, it was not surprising that some of these patients in this study had detectable ACCESS cTnI concentrations, as were also seen in concurrent Stratus cTnI measurements. There was no statistically significant difference, however, between these two cTnI assays in the occurrence of above-normal cTnI results. Because cTnI has been recognized as highly specific for cardiac tissue on grounds of biochemical understanding and clinical experience [27,31], speculation exists that the increased cTnI results of patients with skeletal muscle injury or renal failure may indicate occult minor cardiac injury and that patients showing such cTnI increases are at higher risk for an adverse outcome [32]. However, this issue cannot be substantiated without additional invasive testing or large outcome-based studies.

ROC curve analysis of all 289 suspected MI patients enrolled at the three independent study sites identified a cutoff value of 0.15 μg/L with the ACCESS cTnI assay for MI diagnosis. By using this cutoff value, the ACCESS cTnI assay demonstrated diagnostic sensitivity of 88.9% (95% CI, 79.7–98.1%) and diagnostic specificity of 91.8% (95% CI, 88.4–95.2%) for this patient population.

CK-MB mass and the ACCESS cTnI assay results appeared comparable in terms of diagnostic sensitivity, diagnostic specificity, and ability to classify individual patients as MI or non-MI in the population studied. Also, the ACCESS and the Stratus cTnI assays appeared comparable in the population studied, showing no statistically significant difference in either diagnostic sensitivity, diagnostic specificity, or ability to classify individual patients as MI or non-MI at respective cutoffs of 0.15 and 1.5 μg/L. Although the sensitivity and specificity of the ACCESS cTnI appear to be similar to the CK-MB mass or Stratus cTnI assays, it is important to note, based on type I error analysis, that the number of patients included in this study limited the power to state that the assays were equivalent. In fact, although the observed differences in sensitivity and specificity were small, the statistical power of 0.8 used here meant there was a one-in-five (20%) chance that the actual differences in sensitivity or specificity between the assays could be as large as 15%. The potential for a 15% difference in sensitivity or specificity dictated by both the power of 0.8 and the number of patients included in this study may be viewed as relatively large.

Although this study was not designed to elucidate the issue of concomitant skeletal and cardiac muscle injury, considering the high specificity of cTnI for cardiac muscle [33–35], the ACCESS cTnI assay should have the advantage of providing a more accurate MI diagnosis in patients having conditions associated with skeletal muscle injury and increased CK-MB mass, such as may occur in perioperative MI [8].

Despite 93.0% concordance between the ACCESS and Stratus assays, there were apparent differences in the cTnI measurements. Data presented in Fig. 4 demonstrated that virtually all discordant results between ACCESS cTnI and Stratus cTnI assays showed large pair-wise differences, which was in accordance with the poor correlation (r = 0.156) at relatively low cTnI values and the large SD of the residuals at higher cTnI concentrations. Evidently, fundamental differences in epitope recognition or differences in interaction with antibodies between the ACCESS and Stratus cTnI immunoassays exist. Reportedly, the release of cTnI by damaged myocardium is similar to other myofibril components such as tropomyosin and myosin light chain [36]. In this model, the cytosolic pool of the free component is released first, shortly after myocyte damage, followed by the release of the components as products of myofibrillar breakdown [36]. Evidently, the molecular structures of these two released forms differ; hence, the cTnI composition in circulation changes with time after MI. This hypothesis is also supported by the obvious differences between patterns in ACCESS and Stratus cTnI immunoassays exist. Reportedly, the release of cTnI by damaged myocardium is similar to other myofibril components such as tropomyosin and myosin light chain [36].

In conclusion, the data presented here show that the ACCESS cTnI assay provides a useful clinical tool for the diagnosis of MI. The ACCESS cTnI assay demonstrated acceptable analytical performance and, in the populations studied here, appeared to be diagnostically comparable with both CK-MB mass and the Stratus cTnI assays for the diagnosis of MI. The ACCESS cTnI also showed comparable specificity to the Stratus cTnI assay in non-MI patients with skeletal muscle injury and CRF. Although this study was not designed to compare real-time availability of cardiac markers, the ACCESS cTnI assay is available on a random-access platform designed for rapid turnaround of results.

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References
