Quantitative Bedside Assay for Cardiac Troponin T: A Complementary Method to Centralized Laboratory Testing

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Background: In the evaluation of chest pain patients, whole blood bedside assays of highly specific cardiac molecules may be an attractive alternative to centralized clinical chemistry testing. We now report on an optimized test strip device for cardiac troponin T (cTnT) that can be analyzed by a cardiac reader for quantitative assessment of the test result.

Methods and Results: The cTnT test strip reader measures, via a CCD camera, the reflectance of the signal line. For quantitative analysis, a calibration curve was constructed from 1030 samples of 252 consecutive patients with acute coronary syndromes. In a method comparison of 140 samples, the quantitative results of the cTnT test strip reader correlated closely with the results of the cTnT ELISA ($r = 0.98; y = 0.85x + 0.002$). Within-run and day-to-day (n = 10) mean CVs were between 11% and 16%, respectively. The cross-reactivity with skeletal troponin T was <0.02%. In patients with myocardial infarction, 45% and 91% of all samples were positive on admission and at 4–8 h after the onset of symptoms, respectively. ROC curve analysis demonstrated a comparable efficiency of the cTnT test strip reader and the laboratory-based cTnT ELISA in patients with suspected myocardial infarction.

Conclusions: It is now possible to quantitatively determine cTnT at the patient’s bedside with a rapid and convenient test device. This will facilitate the diagnostic work up of patients with suspected myocardial cell necrosis.

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Since 1994, the diagnostic work up of chest pain patients has been facilitated by the availability of a visual rapid assay for the bedside testing of cardiac troponin T (cTnT)4 from whole blood specimens. In three developmental modifications, the detection limit of this assay was improved from 0.3 µg/L for the first generation (1), to 0.2 µg/L for the second generation (2, 3), and finally, to 0.1 µg/L for the most recent assay. During this optimization process, cross-reactivity was also eliminated by the selection of a new pair of cardiac-specific monoclonal antibodies (4).

The visual assessment of the available whole blood assay allows a yes or no statement without definite information regarding the concentration of cTnT in blood. Although this qualitative assay is sufficient for clinical decisionmaking processes, some prognostic information inherent to the concentrations of circulating cTnT is lost. Furthermore, interindividual variability of visual assessment of the test strip at the detection limit of the assay may cause substantial analytical error (2). We now present an improved bedside assay for cTnT and a newly developed reader to overcome the limitations of previous tests and to enable reliable quantitative and rapid testing for cTnT. This diagnostic tool will facilitate the triage and therapeutic decisionmaking process in the treatment of chest pain patients.

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4 Nonstandard abbreviations: cTnT, cardiac troponin T; AMI, acute myocardial infarction; CK, creatine kinase; CK-MB, MB isoenzyme of CK; and ECG, electrocardiogram.
Materials and Methods

cTnT test strip
The cTnT test strip assay was improved by combining two cardiac-specific antibodies and by optimizing the labeling of the antibodies. In the new assay, the same two cardiac-specific monoclonal antibodies used in the improved version of the cTnT ELISA, M7 and M11.7, were used. The selection and characterization of these antibodies has been described in detail elsewhere (4). The cardiac-specific monoclonal antibody M7 was conjugated with biotin, using the same procedure as outlined previously (2). One test strip contained 0.23 µg of biotinylated anti-cTnT antibody M7. The cardiac-specific monoclonal antibody M11.7 was labeled with gold particles. The amount of gold-labeled antibodies per test strip was 0.11 µg.

Quantitative test strip reader
The measuring unit of the quantitative test strip reader (Cardiac reader; Boehringer Mannheim) contains a CCD camera that optically records the detection zone of the immunochemical test strip. The signal and control lines in the detection zone are recognized by an evaluation algorithm. The intensity of the signal line, determined by measuring its reflectance, is directly proportional to the concentration of cTnT. A negative test result (absence of a signal line) produces a reflectance of 100%. The test requires a sample volume of 150 µL of heparin-treated venous blood and has a reaction time of 12 min.

Calibration curve
To establish a calibration curve for the cardiac reader, we measured 1030 samples by ELISA and by a functional model of the cardiac reader, which worked on the basis of the reflectance only. The samples were obtained from 252 patients with chest pain and suspected myocardial infarction (for details, see the section on clinical analysis below). The cTnT concentrations measured by ELISA were 0–71 µg/L (median, 0.02 µg/L; 25th percentile, 0.0 µg/L; 75th percentile, 0.66 µg/L).

The calibration between the percentage of remission measured with the test strip reader and the cTnT concentration measured with the comparison method ELISA cTnT curve revealed a nonlinear relationship. The measured reflectance was converted via the calibration curve into a cTnT concentration.

Analytical method comparison
The analytical performance of the cardiac reader was compared with the standard ELISA by the use of 140 heparin-treated blood and serum samples from 140 patients with acute coronary syndromes.

Imprecision experiments
The within-run imprecision was determined by 10-fold measurements with lyophilized control materials and with different blood samples supplemented with cTnT. The day-to-day imprecision was obtained from 18 repetitive measurements on 18 subsequent days with controls.

Cross-reactivity testing
Blood samples were supplemented with skeletal troponin T up to a concentration of 500 µg/L in combination with three different cTnT concentrations (0, 0.2, and 1 µg/L) [preparation as described in Katus et al. (5)].

Clinical analysis
The clinical study group comprised 64 healthy volunteers and 252 consecutive patients with suspected acute coronary syndromes seen at the emergency room. The clinical diagnoses in the patients, based on WHO criteria (6, 7), with chest pain were as follows: (a) acute myocardial infarction (AMI; 37 patients, 351 samples), (b) unstable angina pectoris (44 patients, 354 samples), or (c) no evidence of acute myocardial ischemia (171 patients, 325 samples).

All patients gave informed consent to participate in the study after thorough explanation of the study protocol. This investigation was approved by the ethics committee of the University of Heidelberg.

Blood sampling
From each patient, heparin-treated blood and serum were collected on admission; 3, 6, 12, and 24 h after admission; and once per day until day 10 or until discharge. All bedside assay measurements and creatine kinase (CK) activity determinations were performed immediately after a blood specimen was obtained. cTnT ELISA and CK-MB mass measurements were performed from frozen serum within 2 weeks after the samples were collected.

Determination of cardiac marker molecules
The cTnT rapid assay (Boehringer Mannheim) was measured quantitatively on the test strip reader (Boehringer Mannheim). Each test strip was rechecked visually.

The cTnT ELISA (Enzymun Troponin T-Test; Boehringer Mannheim) was performed according to the manufacturer’s instruction for use (4). The cutoff value used for the cTnT ELISA was 0.1 µg/L.

Total CK activity was determined with a ClinChem Analyzer (Miles) and the reagents provided by the manufacturer. The upper reference limit for total CK activity is 80 U/L in men and 75 U/L in women (25 °C).

CK-MB mass concentrations were determined using the Creatine Kinase-MB Fluorometric Enzyme Immunoassay adapted to the Stratus analyzers (Baxter Diagnostics). The upper limit of normal used for CK-MB mass determinations is 4.8 µg/L. As a cutoff value for clinical evaluations, we used 9.6 µg/L, which is twice the upper limit of normal.

Statistics
ROC curve analyses were performed using ASTUTE software (DDU Software).
The relationship between the percentage of remission values of the rapid assay and the concentrations of cTnT ELISA was described using nonlinear regression analysis. The regression function was established using Easy software (Boehringer Mannheim).

Concentration values are given as means and SD or as medians and ranges (25th and 75th percentile).

**Results**

**Analytical evaluation**

*Method comparison and calibration curve.* The method comparison of the quantitative cTnT rapid assay, measured as reflectance on the reader, vs the cTnT ELISA, measured as concentration, is shown in Fig. 1. The data pairs were used for nonlinear regression analysis to obtain a calibration curve for the rapid assay, which is shown in Fig. 1. The cutoff concentration of 0.1 \( \mu \text{g/L} \) for the cTnT ELISA corresponds to a remission value of 97% in this calibration model. This was also confirmed by ROC curve analysis.

**Imprecision.** The within-run imprecision experiments \((n = 10)\) with the cTnT rapid assay yielded CVs between 9% and 14% for cTnT concentrations between 0.16 and 1.24 \( \mu \text{g/L} \). The CV for day-to-day imprecision, performed as 18 repetitive measurements on 18 subsequent days, was between 15% and 18% (Table 1).

**Correlation and accuracy in method comparison.** Compared with the reference method, cTnT ELISA, the results obtained with the cTnT rapid assay show a high correlation at concentrations of 0.1–3 \( \mu \text{g/L} \) \((r = 0.98)\). The accuracy of the test was \( \pm 15\% \) compared with the cTnT ELISA \((y = 0.85x + 0.002; \text{Fig. 2})\).

**Cross-reactivity with skeletal troponin \( T \).** The addition of up to 500 \( \mu \text{g/L} \) of troponin \( T \) purified from human skeletal muscle to cTnT-free samples did not lead to a detectable signal line on the test strip. The cross-reactivity with skeletal troponin \( T \) is therefore \(<0.02\% \) or lower (Table 2).

**Clinical evaluation**

**Healthy subjects.** The mean remission value for 64 healthy individuals was 99.5% \((\text{SD} = 0.6\%)\). When this remission value was converted into a concentration by the use of the established algorithm for reader measurements, the respective cTnT value was \(<0.05 \mu \text{g/L} \). The visual reassessment of the test strip yielded only negative results. The mean concentrations with the cTnT ELISA and CK-MB mass were 0.00 \( \mu \text{g/L} \) \((\text{SD}, 0.01 \mu \text{g/L}) \) and 1.35 \( \mu \text{g/L} \) \((\text{SD}, 0.56 \mu \text{g/L}) \), respectively.

![Fig. 1. Method comparison and establishment of a calibration curve.](image1)

Values of the quantitative bedside assay readings, measured as reflectance, were compared with the values of cTnT ELISA measurements. The data pairs were used to establish a nonlinear regression analysis to obtain a calibration curve.

![Fig. 2. Correlation and accuracy in method comparison.](image2)

Heparin-treated blood samples (140) from patients with suspected coronary syndrome were tested with the cTnT ELISA and the quantitative bedside assay. The results of both methods showed a high correlation in the concentration range 0.1–3 \( \mu \text{g/L} \) \((r = 0.96)\); the accuracy of the bedside assay was in the range of \( \pm 15\% \) compared with the cTnT ELISA \((y = 0.85x + 0.002)\).
Patients with chest pain but no clinical evidence for acute myocardial ischemia. The mean remission value for 325 blood samples of 171 patients was 99.5% (SD, 1.4%), which corresponds to a calculated concentration of <0.05 µg/L. The mean concentrations for the cTnT ELISA was 0.01 µg/L (SD, 0.02 µg/L). The respective values for CK activity and CK-MB mass were 47.2 U/L (SD, 42.4 U/L) and 1.36 µg/L (SD, 2.20 µg/L).

In six blood samples from three patients, the calculated cTnT concentrations were >0.1 µg/L (97% remission value). These tests were also positive when visual assessment of the rapid assay device was performed. When cTnT was measured with the ELISA technique, all but one (0.17 µg/L) sample had serum cTnT concentrations below 0.1 µg/L. Two of these samples had cTnT ELISA concentrations near the detection limit (0.08 µg/L). Clinical diagnosis was severe aortic valve stenosis in one case; in the other two cases, the diagnosis was known coronary artery disease but no evidence for acute ischemia based on cardiac enzymes and electrocardiogram (ECG).

Patients with definite AMI. The mean remission value in the 351 samples from the 37 patients with definite AMI was 74.0% (SD, 21.9%), which corresponds to a calculated concentration of 4.67 µg/L. The mean concentrations for the cTnT ELISA was 4.99 µg/L (SD, 9.36 µg/L). The respective values for CK activity and CK-MB mass were 304.5 U/L (SD, 489.5 U/L) and 55.5 µg/L (SD, 140.2 µg/L).

Negative results were found in 21 of 351 measurements for the visual assessment of the rapid assay. When cTnT ELISA measurements were performed, results below 0.1 µg/L were found in 39 samples, and the mean concentration was 0.03 µg/L (SD, 0.03 µg/L). This number corresponded to the reader measurements (mean remission, 98.88%; SD, 1.5%). All samples with cTnT values <0.1 µg/L were obtained earlier than 5 h or later than 145 h after the onset of symptoms.

Patients with unstable angina pectoris. The mean remission value for patients with unstable angina pectoris was 97.8% (SD, 4.0%), which corresponded to a calculated reader cTnT concentration of 0.14 µg/L. The mean concentration for the cTnT ELISA was 0.09 µg/L (SD, 0.28 µg/L). The respective values for CK activity and CK-MB mass were 45.6 U/L (SD, 66.1 U/L) and 1.78 µg/L (SD, 2.82 µg/L).

One hundred twenty-six of 354 samples had cTnT values >0.1 µg/L with the ELISA technique, indicating the presence of minor myocardial damage. Ninety-four percent of the samples that were positive in the cTnT ELISA were also positive when the measurement was performed with the cardiac reader. In the visual reassessment, all 126 test strips were rated positive.

Kinetics of cTnT release in AMI. The determination of cTnT in sequential blood samples of AMI patients by the use of ELISA and the cardiac reader produced similar time-dependent concentration changes (Fig. 3).

Diagnostic performance in the early period of AMI. The diagnostic sensitivity and specificity of the quantitative bedside assay were evaluated according to the time

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Table 2. Percentages of recovery with the cTnT test strip reader in cross-reactivity testing.

<table>
<thead>
<tr>
<th>Skeletal troponin T, µg/L</th>
<th>0</th>
<th>0.2</th>
<th>1.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>500</td>
<td>100%</td>
<td>110%</td>
<td>109%</td>
</tr>
</tbody>
</table>

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Fig. 3. Comparative analysis of time-dependent cTnT serum concentration changes.

cTnT was determined in sequential blood samples of 37 AMI patients with the troponin T ELISA and the quantitative bedside assay. Fig. 3 shows an example of time-dependent changes in troponin T in one patient suffering from AMI.
interval from the onset of symptoms to analysis (Tables 3 and 4). Because of the debatable classification of cTnT-positive patients with unstable angina, the groups were analyzed both with (Table 3) and without (Table 4) the unstable angina patients. Within the first 24 h, the diagnostic sensitivity was comparable in all four diagnostic assays. The diagnostic specificities increased for both cTnT determination methods to 100% when patients with unstable angina pectoris were omitted from the analysis. As expected, cTnT remained increased for longer time periods than CK and CK-MB.

The areas under the ROC curves for analyses with and without patients suffering from unstable angina pectoris are shown in Table 5. Samples of different time intervals 0–24, 0–48, and 0–96 h after onset of pain were evaluated. Again, similar performance of measurements with the reader and the cTnT ELISA could be shown.

Discussion

The potential role of cTnT in chest pain patients
cTnT testing in chest pain patients markedly aids risk stratification of patients (8–13). This holds true for the group of patients with definite AMI based on ECG as well as for patients with chest pain but unspecific ECG alterations. Stubbs et al. (13) confirmed the results of the GUSTO II trial (10) inasmuch as patients with a positive cTnT and definite AMI on ECG have a threefold higher complication rate compared with cTnT-negative AMI patients on admission. cTnT-positive patients suffering from unstable angina have a cardiac event rate similar to patients with definite AMI acutely and at a 6-month follow-up (9, 11). On the other hand, cTnT-negative patients with unstable angina represent a low-risk group and may undergo exercise testing for further evaluation (14).

Recently it was shown that cTnT determinations may provide valuable information not only for better risk stratification but also for improved therapeutic decision-making in chest pain patients. The data of the FRISC trial convincingly showed that only cTnT-positive, but not cTnT-negative, patients with acute coronary syndromes benefit from low-molecular weight heparin (15). These findings may only be the beginning of a selected treatment of patients with acute coronary syndromes based on cTnT concentrations.

Potential advantages of biochemical bedside testing

There are several potential advantages of bedside measurement of biochemical markers with the new assay and

Table 3. Diagnostic sensitivity and specificity of the cTnT test strip reader, cTnT ELISA, CK activity, and CK-MB mass for the detection of myocardial infarction in all patients with suspected acute coronary syndromes.

<table>
<thead>
<tr>
<th>Hours after onset of symptoms</th>
<th>Number of samples</th>
<th>cTnT reader (97% rem.)</th>
<th>cTnT ELISA (0.1 μg/L)</th>
<th>CK activity (80 U/L)</th>
<th>CK-MB mass (9.6 μg/L)</th>
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</thead>
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<tr>
<td>Admission</td>
<td>238</td>
<td>54</td>
<td>54</td>
<td>56</td>
<td>50</td>
</tr>
<tr>
<td>0–4</td>
<td>128</td>
<td>35</td>
<td>38</td>
<td>37</td>
<td>24</td>
</tr>
<tr>
<td>&gt;4–8</td>
<td>122</td>
<td>91</td>
<td>86</td>
<td>76</td>
<td>83</td>
</tr>
<tr>
<td>&gt;8–12</td>
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<td>100</td>
<td>100</td>
<td>94</td>
<td>100</td>
</tr>
<tr>
<td>&gt;12–24</td>
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<td>98</td>
<td>100</td>
<td>100</td>
<td>96</td>
</tr>
<tr>
<td>&gt;24–48</td>
<td>160</td>
<td>100</td>
<td>100</td>
<td>90</td>
<td>87</td>
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<tr>
<td>&gt;48–96</td>
<td>173</td>
<td>98</td>
<td>100</td>
<td>59</td>
<td>21</td>
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</table>

* Values in parentheses are upper reference limits for normal or cutoff values.

Table 4. Diagnostic specificity of the cTnT test strip reader, cTnT ELISA, CK activity, and CK-MB mass for the detection of myocardial infarction in patients without unstable angina pectoris.

<table>
<thead>
<tr>
<th>Hours after onset of samples</th>
<th>Number of samples</th>
<th>cTnT reader (97% rem.)*</th>
<th>cTnT ELISA (0.1 μg/L)</th>
<th>CK activity (80 U/L)</th>
<th>CK-MB mass (9.6 μg/L)</th>
</tr>
</thead>
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<tr>
<td>Admission</td>
<td>197</td>
<td>98</td>
<td>99</td>
<td>87</td>
<td>99</td>
</tr>
<tr>
<td>0–4</td>
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<td>99</td>
<td>100</td>
<td>91</td>
<td>99</td>
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<tr>
<td>&gt;4–8</td>
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<td>100</td>
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<td>100</td>
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<td>&gt;48–96</td>
<td>94</td>
<td>93</td>
<td>100</td>
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<td>100</td>
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</table>

* Values in parentheses are upper reference limits for normal or cutoff values.

rem., remission.
reader device in patients with chest pain: (a) A biochemical test result is immediately available at the hands of the responsible physician and may thus aid in the triage of chest pain patients, particularly if a positive cTnT result is obtained. Even in university hospitals, the time delay from venipuncture to the availability of a first result may account for several hours. (b) There are many smaller hospitals that do not provide 24-h stat biochemical testing. These institutions may utilize a simple bedside test that can be quantified conveniently by the developed reader. (c) The variability of test results attributable to the reading of the test by untrained personnel is excluded, and information about cTnT serum kinetics may be obtained in consecutive testing of individual patients. (d) Cardiac risk (9, 10) and therapeutic efficiency (15) are influenced by the degree of the increase in cTnT. It is therefore preferable to determine cTnT quantitatively. The development of the quantitative test strip reader represents a further advancement in bedside testing because a time-saving and sensitive assay is now combined with a quantitative cTnT measurement. (e) The reader can be linked to the hospital data management system, and it will provide a numerical documentation of a quantitative result. The reader printout can easily be included into the patient’s chart, taking care of the necessity of proper documentation.

**Analytical Characteristics**

Several substantial improvements of the previous test strip have been implemented to provide a highly efficient testing of whole blood samples for acute myocardial damage.

The antibody combination on the test strip now corresponds to the antibodies that are used in the cTnT ELISA system. This has produced a marked improvement in specificity of the test strip, reducing cross-reactivity with skeletal troponin T to <0.02%. Furthermore, the composition and quantitative relationship of dry chemistry constituents has been modified to ensure high sensitivity. Thus, even by visual assessment, the detection limit of the new test device has been reduced to 0.1 μg/L.

Analysis of the remission of the test strip signal demonstrated that the cardiac reader can detect cTnT concentrations below this cutoff value of 0.1 μg/L. The within-series imprecision and day-to-day imprecision studies yielded CVs of 9–14% and 15–18%, respectively. The method comparison showed a high correlation for concentrations of 0.1–3 μg/L; however, the accuracy was ±15%. These values are acceptable, but clearly higher than those observed for the laboratory-based method. Thus, further improvements of the test relating to imprecision and accuracy are certainly needed. As a consequence, the measuring range of the reader was restricted in the final version to a concentration range of 0.1–3 μg/L; values between 0.05 and <0.1 μg/L are indicated as “low”, values <0.05 μg/L are indicated as “negative”.

When higher cTnT values are present in blood, dilution of the blood specimen would be required if a quantitative result is needed. Twelve minutes are needed for completion of the reading process. However, as soon as a positive line is detected on the test strip, the signal light on the cardiac reader is activated. Thus, therapeutic consequences can be taken before the quantitative result is printed by the reader.

In patients with AMI, unstable angina, and chest pain of noncardiac origin, this quantitative cTnT assay performs comparably to the more sophisticated laboratory-based assay. This also relates to the qualitative and quantitative recovery of cTnT at different days after the onset of AMI symptoms. With the rapid assay and the laboratory ELISA method, a similar time concentration curve was found in these patients.

**Table 5. Area under the curve for ROC analysis of the cTnT test strip reader, cTnT ELISA, CK activity, and CK-MB mass for the detection of myocardial infarction.**

<table>
<thead>
<tr>
<th>Hours after onset of symptoms</th>
<th>Number of samples</th>
<th>cTnT reader (97% rem.)</th>
<th>cTnT ELISA (0.1 μg/L)</th>
<th>CK activity, (80 U/L)</th>
<th>CK-MB mass (9.6 μg/L)</th>
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<tr>
<td>Patients with unstable angina pectoris included</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Admission</td>
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<td>0.97</td>
<td>0.92</td>
<td>0.94</td>
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<tr>
<td>Patients with unstable angina pectoris excluded</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Admission</td>
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<td>0.98</td>
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<td>522</td>
<td>0.97</td>
<td>0.98</td>
<td>0.92</td>
<td>0.95</td>
</tr>
</tbody>
</table>

* Values in parentheses are upper reference limits for normal or cutoff values for assays.

*b* rem., remission.
More than 10 years ago, we first established myosin light chains and then troponin T as markers to overcome some of the limitations of biochemical testing for the detection of myocardial cell necrosis. With the development of the quantitative bedside assay for cTnT, we have completed these developments. As a result, it is now possible to measure within 12 min highly specific and sensitive cTnT without the dependency on a sophisticated clinical chemistry environment.

We do not believe that bedside testing and clinical chemistry testing are mutually exclusive methods. Clearly the laboratory-based method has a better analytical performance (4). Thus, if time is not critical and the appropriate equipment is available, the laboratory-based method may still be preferable. However, for many clinical situations and for many smaller hospitals, bedside testing may be the preferred method, particularly when the results are connected to the hospital data management system as is possible with the cardiac reader.

References