Ohman et al² did compare troponin T to troponin I as predictors of adverse cardiac events in more than 600 patients with the acute coronary syndrome. Using receiver-operator curves and a multiple regression model, they concluded that troponin T had a greater sensitivity than troponin I in detecting patients who would eventually have a myocardial infarction and/or die within 30 days. Furthermore, the concern that troponin T is inappropriately elevated in patients with renal failure has been clarified and rejected in an abstract by Haller et al.³ They assessed the cardiac troponin T levels in 97 patients with renal failure and classified them into three groups: 22 with coronary artery disease proved by angiography or prior myocardial infarction, 40 with two or more recognized risk factors, and 28 with no risk factors. In each, the troponin T value was correlated with cardiac risk; mean±SD values were 0.26±0.08 ng/mL, 0.23±0.06 ng/mL, and 0.07±0.02 ng/mL for the three groups, respectively. Increased troponin T in patients with renal disease was a function of severity of cardiac risk and most likely indicated minimal cardiac injury in this patient population.

Another benefit of troponin T that has not been discussed is the low clinical cutoff (0.1 ng/mL) of troponin T assays, allowing detection of even minor infarcts. Cutoffs for troponin I assays vary by manufacturer and are often too high to detect microinfarcts, and lower cutoffs can result in false positives. According to Ohman et al.,⁴ troponin T has been shown to be an excellent prognostic indicator of adverse cardiac events in patients with unstable angina, due to its analytical sensitivity at very low concentrations.

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To the Editor:

We thank Dr. Statland for his comments. We have examined the abstract by Haller et al.¹ According to the standardized troponin T test, each group had elevated levels of troponin T. However, according to a new more specific enzyme-linked immunosorbent assay (ELISA), patients with proven coronary artery disease (CAD) and patients with two or more cardiac risk factors but without CAD had elevated levels. Therefore, in patients with and without CAD and chronic renal failure, cardiac troponin T is unable to distinguish patients with acute myocardial injury, which is the purpose of the test.

In the abstract by Ohman et al.,² both cardiac troponins T and I were helpful in stratifying patients with acute coronary syndromes by risk. In order to make further comments regarding the comparison of troponins I and T performed by these investigators, we will wait for peer review and publication of the data.

Regardless, these and other emerging articles support the use of cardiac troponins as more sensitive and specific tools/markers for acute myocyte injury than creatine kinase (CK) or CK-MB.

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Cardiac Troponins in the Diagnosis of Myocardial Contusion
An Emerging Controversy

To the Editor:

I am writing in response to the excellent study and presentation entitled “Circulating Cardiac Troponin T in Myocardial Contusion,” by Ferjani et al (February 1997).¹

In spite of their well-defined attempts in the methods of selection and their exemplary efforts in the hemodynamic control of the 29 patients diagnosed as having myocardial contusion, I feel that certain comments and questions are in order. Even though the study appears to be statistically overburdened for the small number of patients presented, the results of the cardiac troponin T (cTnT) assays seem to be in general agreement with the sensitivity and specificity of others,² although to my knowledge, all studies to date are limited in design and by the small numbers of patients available.

The receiver operating characteristic (ROC) curves and associated areas under them were beautifully done, but I feel the reader would have been better served by a master table indicating each patient’s cardiac diagnosis and abnormality, extent of trauma, results of the enzyme analysis, hemodynamic parameters, etc., so that a more reasonable evaluation of the conclusions reached by the authors could be ascertained.

I would also question the authors’ use of 0.5 μg/L as the clinical cutoff for the cTnT analysis, when the normal reference range is <0.1 μg/L. Mair et al.³ have reported positive cTnT values of >1.0 μg/L in 64% of patients with myocardial contusion, in contrast to the 31% of Ferjani et al, who used one half the assay cutoff point. Furthermore, Mair et al.³ reported increases of cTnT in 72% of patients with suspected myocardial injury and peak levels of 0.6 to 5.1 μg/L. It should also be noted that the authors did not use the more well-defined CK-MB(MASS) determination in their analysis of this enzyme. Would this have made a difference in their statistical analysis?⁵

More importantly, I would question the use of the troponin T enzyme-linked immunosorbent assay (ELISA) measurement rather than the more updated, recent versions of this assay. It has been well documented that the original ELISA test has exhibited analytic and biological nonspecificity.⁵

The ROC curve from the article by Ferjani et al.¹ (shown in Figure 1) suggests that a lower cutoff may be more appropriate. Would this have altered the sensitivity and specificity calculations to a noticeable degree? The cardiac markers were performed serially on admission, and at 4 and 24 h thereafter. Which measurement did the authors utilize in the calculation of their

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