Myocardial Injury in Critically Ill Patients*
Relation to Increased Cardiac Troponin I and Hospital Mortality

Jean-Pierre Quenot, MD; Gwénaël Le Teuff, PhD; Catherine Quantin, MD, PhD;
Jean-Marc Doise, MD; Michal Abrahamowicz, PhD; David Masson, PhD; and
Bernard Blettery, MD

Objective: To examine the relationship between myocardial injury, assessed by cardiac troponin I (cTnI) levels, and outcome in selected critically ill patients without acute coronary syndromes or cardiac dysfunction.

Design and setting: Prospective, observational study in the emergency ICU of a university teaching hospital.

Population: Over a 6-month period, 217 consecutive patients admitted to the ICU were studied.

Methods and results: cTnI assays were performed in all patients on admission to the ICU. The incidence of myocardial injury, defined by cTnI level > 0.1 ng/mL, was 32% (69 of 217 patients). Overall mortality was 27% (58 of 217 patients). Patients with myocardial injury had a mortality rate of 51%, compared with only 16% mortality for those without myocardial injury (p < 0.001). The hospital mortality rate was highest among older patients (71 ± 14% vs 58 ± 20%, p < 0.0001) and patients with higher simplified acute physiology scale (SAPS) II score (62 ± 25% vs 37 ± 17%, p < 0.0001). Mechanical ventilation was associated with higher in-hospital death (50% vs 31%, for patients who died in the hospital vs those who were discharged alive; p = 0.03). Elevated blood levels of cTnI were found to be independently associated with hospital mortality, regardless of the presence of SAPS II score and mechanical ventilation, in the logistic regression analysis (odds ratio, 2.09; 95% confidence interval, 1.06 to 4.11; p = 0.01).

Conclusions: This study demonstrates the high frequency of myocardial injury (32%) in critically ill patients without acute coronary syndromes or cardiac dysfunction on admission to ICU. Myocardial injury is an independent determinant of hospital mortality. Assessment of myocardial injury on admission to ICU would make it possible to identify patients at increased risk of death.

(CHEST 2005; 128:2758–2764)

Key words: cardiac troponin I; hospital mortality; ICU; myocardial injury

Abbreviations: ALP = alkaline phosphatase; CI = confidence interval; cTnI = cardiac troponin I; LV = left ventricular; LVED = left ventricular end-diastolic; OR = odds ratio; SAPS = simplified acute physiology score

Adult patients admitted to an ICU appear to be at increased risk of cardiac injury due to the underlying presence of atherosclerosis in their coronary circulation in combination with noncardiac stresses, including anemia, increased tissue oxygen demands, mechanical ventilation, sepsis, and hemodynamic instability. However, cardiac injury is difficult to assess, both clinically and by echocardiography, because it is not always an acute condition in an ICU.6

An unexpectedly high incidence of clinically unrecognized myocardial injury, assessed by elevated cardiac troponin I (cTnI) levels, has previously been reported in critically ill patients.7–9 cTnI is a regulatory protein that is unique to the heart muscle and has been proposed as a highly sensitive and specific marker of myocardial injury.10

To our knowledge, only two studies7,8 have examined the prognostic role of cTnI, and these studies suggested that an elevated blood level of cTnI was not an independent determinant of in-hospital mor-
tality. Furthermore, in both studies, all patients admitted to the ICU were included and no selection was performed prior to inclusion.

Elevated cTnI levels are mainly observed in acute coronary syndromes and in other diseases associated with cardiac dysfunction.1,2,9–15 Therefore, we performed a prospective cohort study in which the main goal was to identify the contribution of myocardial injury, assessed by measurement cTnI levels, to outcome in a selected population of critically ill patients presenting without acute coronary syndrome or cardiac dysfunction.

**Materials and Methods**

**Study Population**

The study was conducted prospectively within the medical ICU (eight beds) of the university teaching hospital in Dijon, France, from June 2003 through December 2003. Patients who received external heart massage for cardiac arrest or symptoms and/or ECG signs typical of acute myocardial infarction or unstable angina at enrollment were excluded from the study. All patients with myocardial systolic dysfunction (ejection fraction < 50%) were also excluded.

The study was approved by the local Human Research Ethics Committee of the Teaching Hospital of Dijon. As required by French law, verbal consent was obtained from all patients (or from their next of kin) after detailed explanations and a letter of information were given. All patients underwent continuous ECG monitoring of leads II and V5, with 12-lead ECG performed on ICU admission and on ST-segment change.

**Echocardiography**

Two-dimensional, real-time echocardiographic studies were performed with an echocardiogram and color Doppler images were recorded with a 2.5/3.5 MHz transducer (Sonos 2500; Hewlett Packard; Andover, MA). A transthoracic approach via an apical or a left subcostal window was used to obtain a long-axis four-chamber view of the heart. Using a microcomputer, stop-motion frames were digitized and displayed on the screen to delineate the endocardial outlines of both ventricles. Left ventricular (LV) end-diastolic (LVED) area and right ventricular end-diastolic area were automatically processed, and the global LV ejection fraction was calculated from LVED and LV end-systolic volumes by the following formula: LVED volume − LV end-systolic volume/LVED volume.16–19 The cutoff point for detecting myocardial LV dysfunction was set at 0.5.

For all patients, we systematically checked the presence of echocardiographic right ventricular dysfunction.19,20 The echocardiographic results were reviewed off-line by two senior intensive care physicians who were blinded to the cTnI results.

**Data Collection**

At admission to the medical ICU, baseline clinical variables were recorded, including age, gender, SAPS II (calculated from the first 24 h data); use of mechanical ventilation, and disease diagnosis. The SAPS II system incorporates physiologic variables such as age and a chronic health evaluation into a measure of the risk of in-hospital mortality.21 The clinical end point was death at any time during hospitalization. The length of stay in the ICU and in the hospital were also recorded. Clinical and laboratory data were collected prospectively for all patients.

**Measurements of cTnI**

Blood samples were collected in ICU patients at admission. All cTnI assays were performed by individuals blinded to the clinical data and mortality outcomes. cTnI levels were measured by means of a one-step enzyme immunoassay based on the sandwich principle22 (Dimension Xpand; Dade Behring; Deerfield, IL). The method used is, briefly, as follows: a plasma sample is incubated with chromium dioxide particles coated with a monoclonal antibody specific for the cTnI molecule and with an alkaline phosphatase (ALP)-labeled second monoclonal antibody specific for cTnI, to form a particle/cTnI/ALP sandwich. Unbound conjugate is removed by magnetic separation and washing. In a further step, the particle/cTnI/ALP complex is transferred to a cuvette where ALP triggers an amplification cascade that leads to the production of hydrogen peroxide. In the presence of peroxidase, a-aminantipyrine, and 3.5-dichlorobenzenesulfonic acid, a colored product reaction that absorbs at 510 nm is produced. The intensity of observed color changes is directly proportional to the concentration of cTnI present in the patient sample. The upper limit of the reference range is 0.1 ng/mL, and the lower limit of detection of this assay is 0.01 ng/mL.22 The sensitivity of the method is 0.04 μg/L. The assay has been designed to minimized interference from heterophilic antibodies.23 The nursing staff and physicians providing care for the study patients in the medical ICU were completely blinded to the nature of this investigation.

**Statistical Analysis**

Categorical variables were compared by χ² statistic and by Fisher Exact Test when expected frequencies were < 5. Continuous variables were compared using Student t test for normally distributed variables and homoscedasticity and using the Mann-Whitney U test when one of the two conditions above were not respected. For all tests, a p value of 0.05 was considered significant. To assess the association between cTnI levels at ICU admission and risk of in-hospital mortality, logistic regression was performed. The statistical significance of the selected variables was tested using a Wald test. Dichotomization of cTnI with a cutoff point equal to the upper range of “normal”²² (ie, 0.1 ng/mL) was also performed. All data analysis was performed using statistical software (SAS version 8.2; SAS Institute; Cary, NC).

**Results**

Among the 293 patients admitted during the study period, 217 patients were included in the study. Seventy-six patients were excluded because of acute coronary syndromes (n = 13), cardiopulmonary arrest (n = 9), myocardial LV dysfunction (n = 35), or inability to perform echocardiography (n = 19). The cause of myocardial LV dysfunction was mainly due to severe sepsis or septic shock (n = 25)²¹ and congestive heart failure (n = 10).

**Diagnosis of Myocardial Injury**

The incidence of myocardial injury as determined by cTnI level > 0.1 ng/mL was 32% (69 of 217
patients). Details of the baseline characteristics are given in Table 1. Patients with cTnI > 0.1 ng/mL were significantly older (69.3 ± 14 vs 61.5 ± 20 years, p = 0.02) and had a significantly higher SAPS II score (55 ± 25 vs 43 ± 22, p < 0.001).

Relationship of Myocardial Injury to Clinical Course

During their ICU stay, patients with myocardial injury were more likely to require mechanical ventilation (62% vs 28%, p < 0.001) than patients without injury, and the average ICU stay was significantly longer (11.3 ± 5.4 days vs 5.4 ± 7.5 days, p < 0.01), although overall hospital stays were of similar duration (Table 1).

Overall mortality was 27% (58 of 217 patients). Patients with myocardial injury had a mortality rate of 51% compared with a mortality rate of 16% for those without myocardial injury (p < 0.001). The in-hospital mortality rate was highest among older patients (71 ± 14% vs 58.5 ± 20%, p < 0.0001) and patients with a higher SAPS II score (62 ± 25% vs 37 ± 17%, p < 0.0001). Mechanical ventilation was associated with higher in-hospital death (50% vs 31%, for patients who died in-hospital vs those discharged alive; p = 0.03) [Table 2]. When cTnI is considered in quartiles (0 to 0.1, > 0.1 to 1, > 1 to 2 and > 2), there were statistically significant increases in mortality with increasing levels of cTnI (p < 0.001, χ² test for trend) [Fig 1].

Multivariate Analysis

Logistic regression analysis confirmed that a higher SAPS II score and cTnI levels above the cut-off point of 0.1 ng/mL are significantly associated with a higher risk of in-hospital mortality (Table 3). After adjusting for the SAPS II score and mechanical ventilation, logarithmic transformation of the continuous cTnI level and dichotomization at 0.1 ng/mL yielded a very similar goodness of fit (Akaide Information Criterion, 678.0 vs 679.2) and, therefore, the model with a dichotomous variable was retained because of easy interpretation.

Elevated blood levels of cTnI were found to be independently associated with hospital mortality regardless of the presence of SAPS II score and mechanical ventilation in the logistic regression analysis (odds ratio [OR], 2.09; 95% confidence interval [CI], 1.06 to 4.11; p = 0.01). Mechanical ventilation was not found to be independently associated with hospital mortality. This could be explained by possible collinearity, as it is included in the SAPS II score.

Table 1—Comparison of cTnI Level and Baseline Characteristics at ICU Admission*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>cTnI &lt; 0.1 ng/mL (n = 69)</th>
<th>cTnI ≤ 0.1 ng/mL (n = 148)</th>
<th>p Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>69.3 ± 14</td>
<td>61.5 ± 20</td>
<td>0.02</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td>0.78</td>
</tr>
<tr>
<td>Male</td>
<td>39 (56)</td>
<td>82 (55)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>30 (44)</td>
<td>66 (45)</td>
<td></td>
</tr>
<tr>
<td>SAPS II</td>
<td>55 ± 25</td>
<td>43 ± 22</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>38 (62)</td>
<td>41 (28)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asthma/COPD</td>
<td>12 (18)</td>
<td>33 (22)</td>
<td>0.34</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>7 (10)</td>
<td>3 (2)</td>
<td>0.05</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>14 (20)</td>
<td>21 (14)</td>
<td>0.34</td>
</tr>
<tr>
<td>GI hemorrhage</td>
<td>2 (3)</td>
<td>11 (7)</td>
<td>0.26</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>3 (4)</td>
<td>2 (1)</td>
<td>0.36</td>
</tr>
<tr>
<td>Diabetic ketoacidosis</td>
<td>1 (1)</td>
<td>5 (3)</td>
<td>0.73</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>8 (12)</td>
<td>6 (4)</td>
<td>0.06</td>
</tr>
<tr>
<td>Cerebral vascular accident</td>
<td>8 (12)</td>
<td>19 (13)</td>
<td>0.90</td>
</tr>
<tr>
<td>Meningitis</td>
<td>1 (1)</td>
<td>6 (4)</td>
<td>0.56</td>
</tr>
<tr>
<td>Status epilepticus</td>
<td>3 (4)</td>
<td>16 (11)</td>
<td>0.16</td>
</tr>
<tr>
<td>Drug overdose</td>
<td>5 (7)</td>
<td>18 (12)</td>
<td>0.34</td>
</tr>
<tr>
<td>Hypovolemic shock</td>
<td>5 (7)</td>
<td>3 (2)</td>
<td>0.12</td>
</tr>
<tr>
<td>Head injury</td>
<td>0 (0)</td>
<td>5 (3)</td>
<td>0.30</td>
</tr>
<tr>
<td>ICU stay, d</td>
<td>11.3 ± 5.4</td>
<td>5.4 ± 7.5</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Hospital stay, d</td>
<td>18.6 ± 29</td>
<td>15.8 ± 23.2</td>
<td>0.38</td>
</tr>
<tr>
<td>Hospital death</td>
<td>35 (51)</td>
<td>23 (16)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD or No. (%).
†Student t test for two-tailed independent groups, Mann-Whitney U test for quantitative variables, and χ² test for dichotomous variables.

Table 2—Baseline Characteristics of the Study Population According to Whether They Survived or Died in the Hospital*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Discharged Alive (n = 159)</th>
<th>Died in Hospital (n = 58)</th>
<th>p Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>58.5 ± 20</td>
<td>71 ± 14</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>SAPS II</td>
<td>37 ± 17</td>
<td>62 ± 25</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>50 (31)</td>
<td>29 (50)</td>
<td>0.03</td>
</tr>
<tr>
<td>cTnI &lt; 0.1 ng/mL</td>
<td>34 (21)</td>
<td>35 (60)</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD or No. (%).
†Student t test for two-tailed independent groups, Mann-Whitney U test for quantitative variables, and χ² test for dichotomous variables.

Table 2—Baseline Characteristics of the Study Population According to Whether They Survived or Died in the Hospital*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Discharged Alive (n = 159)</th>
<th>Died in Hospital (n = 58)</th>
<th>p Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>58.5 ± 20</td>
<td>71 ± 14</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>SAPS II</td>
<td>37 ± 17</td>
<td>62 ± 25</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>50 (31)</td>
<td>29 (50)</td>
<td>0.03</td>
</tr>
<tr>
<td>cTnI &lt; 0.1 ng/mL</td>
<td>34 (21)</td>
<td>35 (60)</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD or No. (%).
†Student t test for two-tailed independent groups, Mann-Whitney U test for quantitative variables, and χ² test for dichotomous variables.

Table 2—Baseline Characteristics of the Study Population According to Whether They Survived or Died in the Hospital*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Discharged Alive (n = 159)</th>
<th>Died in Hospital (n = 58)</th>
<th>p Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>58.5 ± 20</td>
<td>71 ± 14</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>SAPS II</td>
<td>37 ± 17</td>
<td>62 ± 25</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>50 (31)</td>
<td>29 (50)</td>
<td>0.03</td>
</tr>
<tr>
<td>cTnI &lt; 0.1 ng/mL</td>
<td>34 (21)</td>
<td>35 (60)</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD or No. (%).
†Student t test for two-tailed independent groups, Mann-Whitney U test for quantitative variables, and χ² test for dichotomous variables.

Discussion

This prospective study showed that myocardial injury is common (32%) among critically ill medical patients and is independently associated with significantly higher in-hospital mortality (p = 0.01), even after adjusting for the SAPS II score.21 The originality of this article is to avoid possible confusion coming from the enrollment of patients with acute coronary syndromes or who presented with cardiac dysfunction on echocardiography, as these patients were purposely excluded from the study (26% were excluded in total).
In this study, patients with myocardial injury were more likely to require mechanical ventilation (62% vs 28%, \( p < 0.001 \)). This observation has been reported by other authors. The presence of myocardial injury was confirmed by cTnI levels above the threshold value, but there was no clear explanation of whether the cTnI elevation was present at ICU admission, or whether it occurred during mechanical ventilation.

Other studies describe elevated cTnI among different groups of critically ill patients. Guest and coworkers measured cTnI levels in 209 critically ill and 260 critically ill patients, respectively. All patients admitted to the ICU were included, and no selection was performed prior to inclusion. In these two studies, 15% and 15.8% of the included patients were cTnI positive. cTnI levels have also been studied in selected groups of critically ill patients with sepsis, in whom the reported incidence of cTnI elevations ranged from 50 to 85%. We found altered LV systolic performance in 35 patients (12%), especially in septic patients. A possible direct cardiac myocytotoxic effect of bacterial endotoxins or of local and circulating mediators (e.g., cytokines or reactive oxygen species) and ischemia and reperfusion damage associated with microvascular dysfunction or resuscitation procedures (e.g., the use of vasopressors) could be involved.

In observational studies, tachycardia, arrhythmia, hypotension, and inotropic drugs were associated with higher concentrations of cTnI in ICU patients. Physiologic stresses can occur in the form of either increased myocardial oxygen demands (e.g., fever, tachycardia) or decreased myocardial oxygen delivery (e.g., anemia, hypotension, hypoxemia) resulting in cardiac dysfunction, cardiac injury, or both. This potential for an imbalance between oxygen supply and demand and the known propensity of critically ill patients to develop acute thrombosis may explain the increase in the risk of myocardial injury.

The previous published data on critically ill patients suggest an association between enhanced systemic oxygen delivery (achieved with the use of dobutamine) and increased mortality from cardiac events, which may be related to secondary myocardial injury. Therefore, it is not surprising that a diagnosis of cardiac injury should be relatively common among critically ill medical pa-

---

Table 3—Relative Value of cTnI, SAPS II, and Mechanical Ventilation as Predictors of Hospital Mortality

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Adjusted OR</th>
<th>95% CI</th>
<th>( p ) Value, Wald Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>cTnI, ng/mL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \leq 0.1 )</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 0.1</td>
<td>2.09</td>
<td>1.06–4.11</td>
<td>0.01</td>
</tr>
<tr>
<td>SAPS II (1 point)</td>
<td>1.04</td>
<td>1.01–1.42</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>0.91</td>
<td>0.58–1.43</td>
<td>0.68</td>
</tr>
</tbody>
</table>
tients even in the absence of known cardiac diseases. Furthermore, these factors may be of greater significance in patients with underlying ischemia or nonischemic heart disease.14,15

Echocardiogram-derived LV ejection fraction is widely used to assess LF function because it is noninvasive and commonly available.30,31 Echocardiography is particularly difficult to perform in some conditions, particularly in obese patients, or in patients with chronic respiratory insufficiency at emphysema stage, but also among patients receiving mechanical ventilation who cannot be properly positioned.32 This explains why it was impossible to perform echocardiography in 19 patients (6%). In the article by Bellenger et al,33 comparing echocardiography, radionuclide ventriculography, and MRI for the measurement of ejection fraction and LV volume in stable heart failure patients, it was shown that these three techniques are not interchangeable, and their respective indications depend on the medical context and their facility of application. In the ICU, echocardiography is the simplest and quickest technique, albeit operator dependent. In our study, the echocardiographic images were reviewed by two senior physicians not concerned by the outcome of the study. No right ventricular dysfunction was noted among the patients of our study,19,20 particularly among those with pulmonary embolism.

In another study,34 the authors demonstrated the interest of cTnI in detecting cardiac myolysis in heart failure, independent of the presence of coronary artery diseases. The mechanisms of myocyte loss include necrosis and apoptosis,35 which could be due to a variety of factors, such as interstitial changes reducing capillary density, a reduced coronary reserve and subendocardial ischemia,36 excessive activation of vasoconstrictive neurohormonal factors,37 and cytokine activation.38

In our study, myocardial injury was shown to be an independent determinant of in-hospital mortality by multivariate analysis (OR, 1.85; 95% CI, 1.15 to 2.95; p = 0.01). In the two studies7,8 that included larger numbers of critically ill patients (260 patients and 219 patients, respectively), cTnI-positive patients had a greater hospital mortality rate, but elevated cTnI concentrations did not independently contribute to the prediction of hospital mortality.

A possible explanation for the discrepancy in the magnitude of the relative risks of death for cTnI between our study and previous studies may be the populations considered. The reported differences in cTnI positivity could also be the consequence of different cardiac troponin assays.

In the literature,39–43 the common causes of false-positive troponin I measurements are heterophilic antibodies, rheumatoid factor, fibrin clots, microparticles, and analyzer malfunction. The incidence of this interference varies considerably, ranging from 0.17 to 40%.44 In the study by Fleming and co-workers,45 a new serum centrifugation method is proposed, as well as the use of heterophilic blocking agents, which made it possible to attain an overall prevalence of false-positive serum cTnI of 3.1%.

The finding, therefore, of a significant level of false-positive cTnI is of clinical importance and raises the real possibility that some low-risk patients may be incorrectly labeled and managed. We recommended repeated serum cTnI estimation in equivocal cases. A special treatment is used in our laboratory to avoid such false results among the samples tested.23 The prevalence and clinical significance of elevated troponins in patients with renal failure have been reviewed elsewhere.46

The majority of studies assessed levels of troponin T in patients with chronic renal disease; therefore, the results cannot easily be extrapolated to our study, where cTnI was assessed in patients with acute renal insufficiency. The exact causes of cTnI elevation in renal failure remain debatable; patients with elevated troponin levels generally have worse clinical outcome than those without elevated levels.46

The ability to detect myocardial injury in a noninvasive and readily available way might open a new avenue that would allow early identification of myocardial involvement in critically ill patients, independent of indexes that measure overall cardiovascular function. Our findings have two potentially important implications. Firstly, cardiac injury is common in patients hospitalized in an ICU, and the ability of cTnI to predict cardiac injury and the outcome is interesting in the absence of clinical signs. Secondly, a number of treatment strategies such as medical therapies in the form of IV fluids, administration of drugs with known negative inotropic effects, and the influence of positive pressure mechanical ventilation, may lead to cardiac injury.14,22,23 The early identification of myocardial injury would make it possible to take appropriate medical interventions to reverse myocardial injury, thereby decreasing patient morbidity and mortality in intensive care.

Future studies are required to determine the benefits of strategies aimed at prevention and more aggressive treatment of myocardial injury. These investigations should also attempt to identify whether any subgroup of critically ill patients would benefit from establishing a diagnosis of acute cardiac injury, using cTnI measurements, early in their ICU stay.

Study Limitations

Several limitations of our study must be acknowledged. Firstly, this study considered only medical
patients admitted to a single institution presenting without acute coronary syndromes and cardiac dysfunction. Therefore, these results may not be directly applicable to nonmedical patients. Secondly, no investigations were conducted to determine whether patients had myocardial injury that would predispose them to myocardial ischemia when an increase in cTnI occurred. Lastly, our relatively small sample size limited our ability to identify other independent determinants of in-hospital mortality.

Conclusions

In summary, this study demonstrates the high frequency of myocardial injury (32%) on admission to ICU in critically ill patients presenting without acute coronary syndromes or cardiac dysfunction. Myocardial injury is an independent determinant of in-hospital mortality, even when adjusted for the SAPS II score. Systematic assessment of cTnI levels on ICU admission would allow the early identification of patients at increased risk of death. Further studies are required to confirm these results in a larger series of patients, and to identify other determinants of increased cTnI levels in ICU patients and prognostic implications.

References

3. Farmley WW, Tyberg JV. Determination of myocardial oxygen demand. Prog Cardiol 1976; 5:19
mittee on Clinical Application of Echocardiography); developed in collaboration with the American Society of Echocardiography. Circulation 1997; 95:1686–1744
33 Bellenger NG, Burgess MI, Ray SG, et al. Comparison of left ventricular ejection fraction and volumes in heart failure by echocardiography, radionuclide ventriculography, and cardiovascular magnetic resonance; are they interchangeable? Eur Heart J 2000; 21:1387–1396
36 Vatner SF. Reduced subendocardial myocardial perfusion as one mechanism for congestive heart failure. Am J Cardiol 1988; 62:94–98
40 Nosanchuk JS. False increases of troponin I attributable to incomplete separation [letter]. Clin Chem 1999; 45:714
42 Dasgupta A, Banjiree SK, Datta P. False positive troponin I in the MEIA due to the presence of rheumatoid factors in serum. Am J Clin Pathol 1999; 112:753–756

Downloaded From: http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chester/22032/ on 06/26/2017