

## CLINICAL INVESTIGATIONS

# Diagnostic Performance of Venous Lactate on Arrival at the Emergency Department for Myocardial Infarction

Mathieu Gatien, MD, Ian Stiell, MD, MSc, Andreas Wielgosz, MD, PhD, Daylily Ooi, MD, Jacques S. Lee, MD, MSc

## Abstract

**Objectives:** To determine the sensitivity of the venous lactate level at presentation for acute myocardial infarction (AMI) in emergency department (ED) patients with chest pain. **Methods:** A prospective, double-blind observational study was done in a tertiary care ED. From January to April 2000, all consecutive patients presenting with chest pain were eligible. Lactate level was obtained on arrival and compared with two criterion standards for the diagnosis of AMI: the World Health Organization (WHO) and the Joint European Society of Cardiology/American College of Cardiology Committee (ESC/ACC) classifications. A lactate level greater than 1.50 mmol/L was considered positive. **Results:** Between January and April 2000, 718 patients were enrolled. By the WHO criteria, 64 patients suffered an AMI, of whom 59 had an elevated lactate level, yielding a sensitivity of 92% (95% CI = 86% to 99%), a specificity of 44% (95% CI = 40% to 48%), and a negative predictive value

(NPV) of 98% (95% CI = 97% to 99%). For all patients presenting with more than two hours of chest pain ( $n=34$ ), the lactate level was elevated. When using the ESC/ACC criteria, 100 patients sustained an AMI, of whom 88 had an elevated lactate level, yielding a sensitivity of 88% (95% CI = 82% to 94%), a specificity of 46% (95% CI = 42% to 50%), and an NPV of 96% (95% CI = 94% to 98%). **Conclusions:** Venous lactate level at presentation is highly sensitive for the diagnosis of AMI, particularly in patients with more than two hours of chest pain. Given its limitations in specificity and ability to detect creatine kinase-MB-negative/troponin-positive microinfarcts, further research is needed to determine how lactate can complement other cardiac enzymes in risk-stratifying all acute coronary syndromes. **Key words:** lactates; chest pain; myocardial infarction. *ACADEMIC EMERGENCY MEDICINE* 2005; 12:106–113.

More than five million patients visit emergency departments (EDs) in Canada and the United States each year because of chest pain.<sup>1</sup> The disposition of the patient with chest pain is one of the more difficult and inherently risky decisions encountered by the emergency physician. Even though a minority of ED chest pain patients are actually suffering from cardiac ischemia, a much larger number of patients are being admitted out of concern of sending a cardiac patient home. Despite admission rates as high as 86%, large studies reveal that between 2% and 6% of acute myocardial infarctions (AMI) are missed in the ED and the patients are inadvertently discharged.<sup>2–4</sup> The morbidity and mortality of these missed patients are

consistently worse than those of a similar cohort of admitted AMI patients.<sup>4</sup>

Emergency physicians are keenly aware of the limitations of present laboratory investigations, particularly in the early phases of an AMI. The initial electrocardiogram (ECG) is diagnostic in only about 50% of AMIs.<sup>5</sup> Biochemical markers, including levels of creatine kinase (CK), CK-MB, and the troponins, are often initially within normal range and may take up to six to 12 hours to rise to a diagnostic level.<sup>6</sup> Myoglobin level has an earlier peak, but its lower specificity makes it a less useful test in the ED.<sup>6</sup> Thus, patients are often observed in the ED for hours, awaiting serial biochemical markers or cardiac stress testing, despite the fact that the majority of these patients are not suffering from cardiac ischemia. A rapidly available and highly sensitive screening test for AMI, available at presentation, could reduce the rate of unnecessary admission for patients with non-cardiac chest pain, and simultaneously reduce the rate of missed AMIs—those patients sent home in error.

Lactic acid is a byproduct of anaerobic glycolysis that rises in states of tissue hypoperfusion, and is quickly buffered in the circulation to lactate.<sup>7</sup> States of regional hypoperfusion frequently occur in AMI, despite maintenance of normal blood pressure.<sup>8</sup> In

From the Department of Emergency Medicine (MG, IS, JSL), Department of Pathology and Laboratory Medicine (DO), and Division of Cardiology (AW), The Ottawa Hospital, Ottawa, Ontario, Canada.

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Address for correspondence and reprints: Mathieu Gatien, MD, Emergency Department, The Ottawa Hospital, General Campus, 501 Smyth Road, Ottawa, Ontario, Canada K1H 8L6. e-mail: m.gatien@sympatico.ca.

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addition, under basal conditions, the myocardium extracts lactate from the circulation, but in conditions of cardiac ischemia, its ability to do so is compromised.<sup>9-12</sup> Therefore, myocardial ischemia could cause an elevation in the level of circulating lactate through both of these mechanisms.

Intensive care unit (ICU)-based studies<sup>13,14</sup> have previously shown that the venous lactate level on admission correlates well with prognosis and survival from an AMI. Only one prior study<sup>15</sup> has addressed the value of using venous lactate level in the ED as a point-of-care test to exclude myocardial infarction in patients presenting with chest pain. This study found that venous lactate had a sensitivity for AMI of 96% and a negative predictive value (NPV) of 98%, even in patients presenting within two hours of the onset of chest pain. However, a subsequent study published in abstract form<sup>16</sup> reported a lower sensitivity for lactate of 54%.

The goals of this investigation were to assess whether the venous lactate level obtained at the time of presentation is clinically useful in excluding AMI among patients presenting to the ED with acute chest pain, and to resolve these previous conflicting findings.

## METHODS

**Study Design.** This was a prospective, double-blind observational study. The ethics committee of our institutional review board approved the study without requiring formal written consent.

**Study Setting and Population.** This study was conducted in the ED of a Canadian university-affiliated teaching center, whose ED has an annual census of 55,000 patient visits. From January 2000 to April 2000, all consecutive patients presenting to the ED with a primary complaint of chest pain (or neck/shoulder/back pain suspicious for cardiac ischemia) were considered for enrollment in the study.

To be eligible, patients had to be triaged to the acute care area of the department (or reassigned to a higher acuity by the treating physician) and have an ECG and any type of blood work ordered as part of their investigations. The aim of these requirements was to eliminate extremely low-risk patients who had a clear alternative diagnosis for their chest pain upon clinical assessment. Patients were excluded by the following criteria: age less than 25 years, cardiac arrest prior to blood sampling, traumatic cause for pain, inability to provide history, unavailability for follow-up, more than three ED visits for chest pain during the study period, and referral from another institution directly to the cardiology service.

**Study Protocol.** Upon arrival in the ED, a sample of venous blood was collected in a heparinized evacuated tube (Becton-Dickinson PST, Franklin Lakes, NJ),

placed on ice, and sent immediately to the laboratory for analysis of lactate level, total CK and CK-MB levels, or cardiac troponin T (cTnT) level. Plasma lactate and CK levels were measured on the Roche Diagnostics BM/Hitachi 917 analyzer (Indianapolis, IN), CK-MB on the Abbott AxSYM (Abbott Laboratories, Abbott Park, IL), and cTnT on the Roche Diagnostics Elecsys 1010. In order to achieve greater precision of the lactate analyses, each sample was measured in duplicate and the averaged value was used. The coefficient of variation of lactate analysis was 2.5% at 1.45 mmol/L. A lactate concentration greater than 1.50 mmol/L was prospectively chosen as a positive test based on previous studies.<sup>15</sup> Physicians were blinded to the lactate concentration.

Data from the patients' ED visits were prospectively collected by the attending emergency physician and recorded on standardized patient data collection sheets, and further information was obtained from the patients' ED and inpatient records. All information was then entered into an SAS 6.0 (SAS Institute, Cary, NC) computerized database.

**Outcome Measures.** The diagnosis of myocardial infarction was made by the attending cardiologist based on World Health Organization (WHO) criteria,<sup>17</sup> requiring at least two of the following three criteria: typical history, typical ECG changes, and rise in CK to above twice the upper limit of the reference range (with concomitant elevation of CK-MB). During the study period, the hospital laboratory replaced CK-MB with the cTnT as the specific cardiac marker. A CK level >430 U/L in males and >340 U/L in females, a CK-MB level >15 µg/L with a relative index >5%, and a cTnT level >0.10 µg/L were considered positive. Each patient's final diagnosis was confirmed by a study author (MG) using the above criteria while remaining blinded to lactate results. Patients discharged from the ED were contacted four weeks postdischarge by telephone interview to ensure that no patients with myocardial infarction were inadvertently sent home.

**Data Analysis.** We report sensitivity and specificity with 95% confidence intervals (95% CIs) to assess the diagnostic performance of the venous lactate level for AMI. Positive and negative predictive values (PPVs, NPVs) are also reported with 95% CIs. Based on conservative assumptions that the lactate level would have a sensitivity of 85%, and that the AMI rate would be 12% in our ED population, we calculated that a sample size of 600 patients would yield a confidence interval of less than 10% for our primary endpoint, the sensitivity of the lactate level.

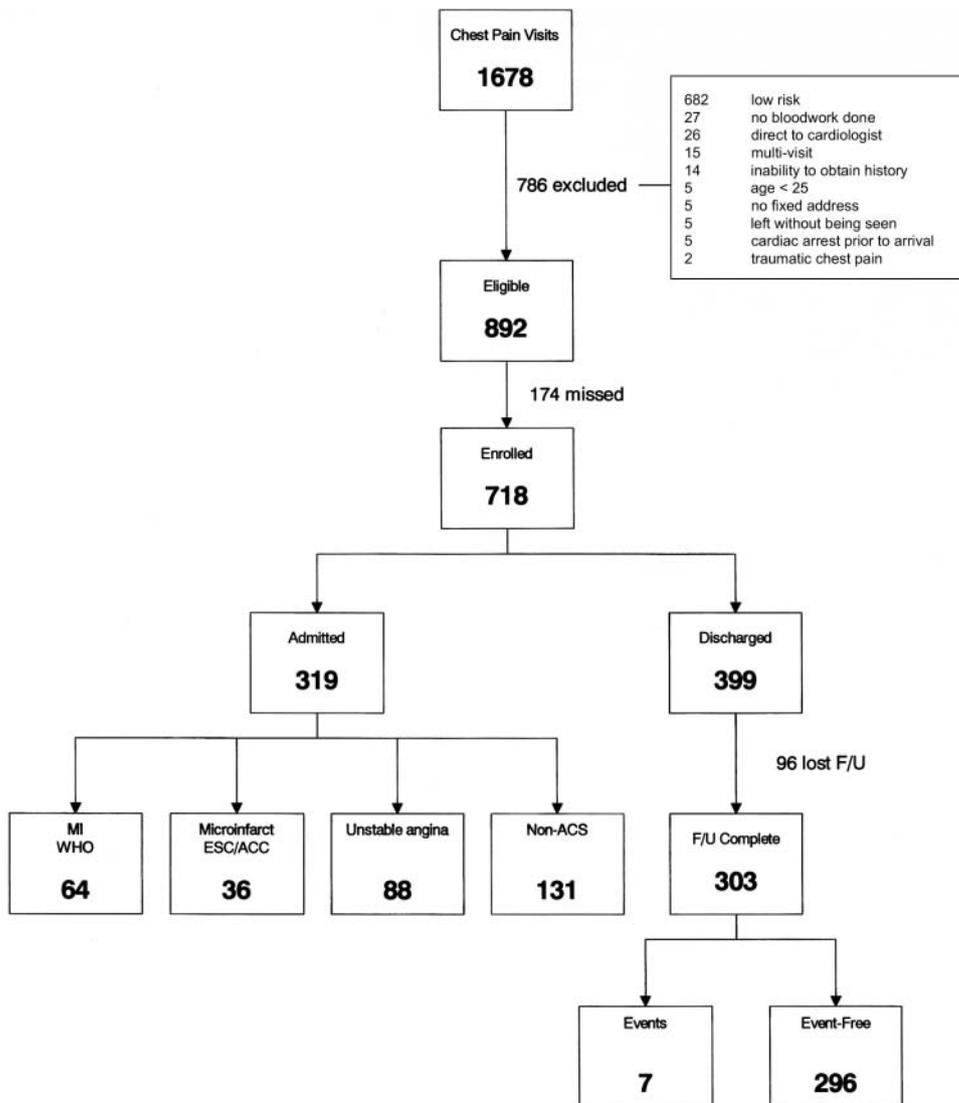
After completion of the study, new criteria for the diagnosis of AMI were released in September 2000 by the Joint European Society of Cardiology/American College of Cardiology Committee (ESC/ACC),<sup>18</sup>

whereby the laboratory diagnosis of myocardial infarction is based on a positive troponin level alone, even if a patient's CK-MB remains normal. Patients who are CK-negative but troponin-positive are deemed to have suffered "microinfarcts." This new definition in effect changes the criterion standard by which AMI is diagnosed. As such, the performance of the lactate level was compared with both standards—the previous WHO classification and the newer, more inclusive, ESC/ACC definition for AMI.

### RESULTS

From January 2000 to April 2000, there were 1,678 patient visits to our ED with a primary complaint of chest pain, of which 786 were excluded (Figure 1). Of the remaining 892 patients, 174 were missed at the time of enrollment (blood was not sent for lactate analysis), leaving 718 patients enrolled in the trial.

There was no significant difference between the baseline characteristics or the outcomes of missed and enrolled patient groups. Patient demographics for the study population are given in Table 1. Of the 718 enrolled patients, 319 (44%) were admitted to hospital. Sixty-four patients (8.8%) suffered an AMI according to the WHO classification (cTnT-positive, CK-positive) and an additional 36 patients (5.0%) suffered a microinfarct (cTnT-positive, CK-negative). Of the 399 patients discharged from the ED, follow-up was completed for 303 patients, and of those patients, seven had events within 30 days of ED discharge (two AMIs, one death, four unstable angina). Only one of those patients was given an inaccurate diagnosis for his chest pain in the ED and returned three days later with an AMI. The plasma lactate level for that patient was elevated. The other six patients were correctly identified as having a cardiac etiology for pain (stable or unstable angina), but a clinical decision was made



**Figure 1.** Patient enrollment. F/U = follow-up; MI = myocardial infarction; WHO = World Health Organization; ESC/ACC = the Joint European Society of Cardiology/American College of Cardiology Committee; ACS = acute coronary syndrome.

**TABLE 1. Patient Characteristics**

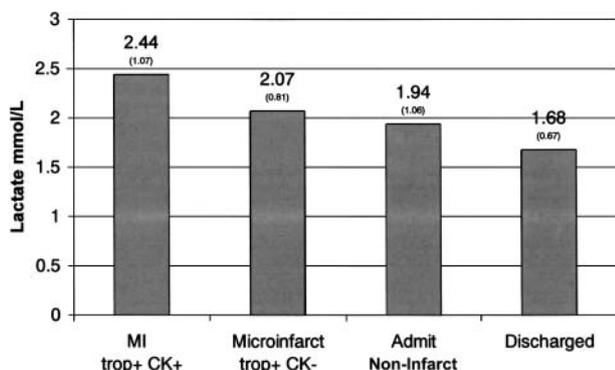
| Characteristic             | Total Number (%) (n = 718) |
|----------------------------|----------------------------|
| Gender                     |                            |
| Male                       | 403 (56%)                  |
| Female                     | 315 (44%)                  |
| Age                        |                            |
| 25–35 years                | 9 (1%)                     |
| 36–45 years                | 60 (8%)                    |
| 46–55 years                | 144 (20%)                  |
| 56–65 years                | 136 (19%)                  |
| >65 years                  | 369 (51%)                  |
| Outcomes                   |                            |
| Disposition                |                            |
| Discharged                 | 399 (56%)                  |
| Admitted                   | 319 (44%)                  |
| Ward/chest pain unit       | 160                        |
| CCU                        | 142                        |
| Catheterization unit       | 14                         |
| Death in ED                | 3                          |
| Diagnosis                  |                            |
| AMI (cTnT+, CK+)           | 64 (9%)                    |
| Microinfarct (cTnT+, CK–)  | 36 (5%)                    |
| All MI                     | 100 (14%)                  |
| Unstable angina (enzymes–) | 88 (12%)                   |
| Stable angina/noncardiac   | 530 (74%)                  |

CCU = coronary care unit; cTnT = cardiac troponin T; CK = creatine kinase; MI = myocardial infarction.

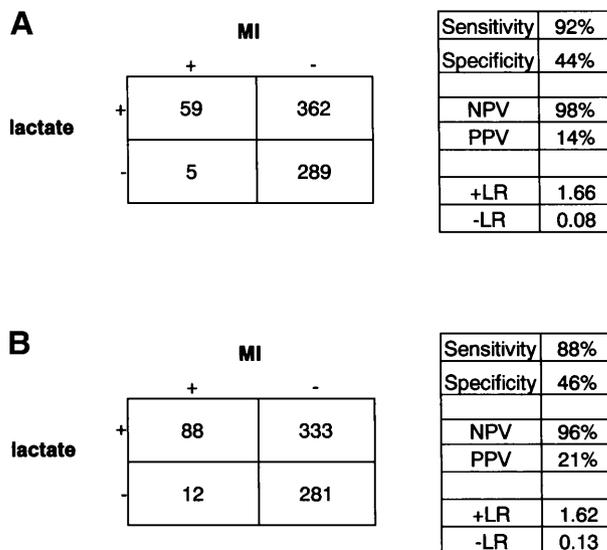
to treat them with outpatient therapy and they were discharged from the ED.

Mean plasma lactate levels for the following patient groups decreased according to the severity of illness (Figure 2): those who had AMI by WHO criteria (cTnT-positive, CK-positive), those who had microinfarcts (cTnT-positive, CK-negative), admitted patients with no infarction (non-ACSs), and discharged patients. It is interesting to note that for all groups, including those with noncardiac chest pain, the mean plasma lactate level was higher than the “normal” cutoff of 1.50 mmol/L suggested by the manufacturer.

Figure 3 contains two 4 × 4 tables comparing the lactate values against the final diagnosis given to each patient, by either the WHO classification or the ESC/ACC classification.



**Figure 2.** Mean lactate values for each group of diagnoses ( $\pm$  standard deviation). MI = myocardial infarction; trop = cardiac troponin; CK = creatine kinase.

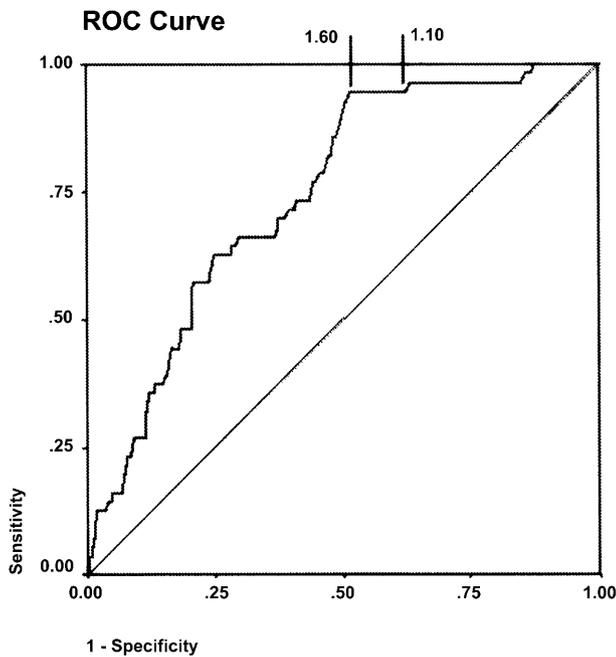


**Figure 3. A.** Performance of the lactate level against the World Health Organization criteria for detecting acute myocardial infarction (MI). **B.** Performance of the lactate level against the Joint European Society of Cardiology/American College of Cardiology Committee definition of acute myocardial infarction. NPV = negative predictive value; PPV = positive predictive value; +LR = positive likelihood ratio; -LR = negative likelihood ratio.

tion, there were 64 patients with an AMI, of whom 59 had elevated lactate levels, yielding a sensitivity of 92.2% (95% CI = 85.6% to 98.7%), a specificity of 44.4% (95% CI = 40.6% to 48.2%), an NPV of 98.3% (95% CI = 96.8% to 99.8%), and a PPV of 14.0% (95% CI = 10.7% to 17.3%). When using the ESC/ACC classification (AMI + microinfarcts), there were 100 patients with infarction, of whom 88 had elevated lactate levels, yielding a sensitivity of 88.0% (95% CI = 81.6% to 94.4%), a specificity of 45.8% (95% CI = 41.8% to 49.7%), an NPV of 95.9% (95% CI = 93.6% to 98.2%), and a PPV of 20.9% (95% CI = 17.0% to 24.8%).

The receiver operating characteristic (ROC) curve for lactate level in the diagnosis of AMI according to WHO criteria is presented in Figure 4. Lactate values corresponding to specific points of interest are labeled. The area under the curve is 0.704 (95% CI = 0.643 to 0.766).

The sensitivities of lactate level and other cardiac markers at the point of presentation in relation to duration of chest pain are shown in Figure 5. The time of onset of symptoms could not be determined for one patient, and data are presented for the remaining 63 patients who had AMI. When using the WHO classification for AMI, for patients with chest pain of two to six hours' duration, lactate level at presentation had 100% sensitivity in the diagnosis of AMI, as compared with 14% for CK and 45% for cTnT. For patients with less than two hours of chest pain, the sensitivity of the lactate level was 86%, compared with 4% and 25% for CK and cTnT levels, respectively. When including microinfarcts, lactate level had a 97%

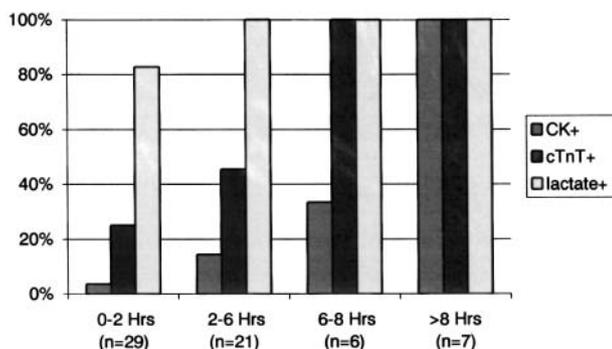


**Figure 4.** The receiver operating characteristic (ROC) curve for lactate level in the diagnosis of myocardial infarction.

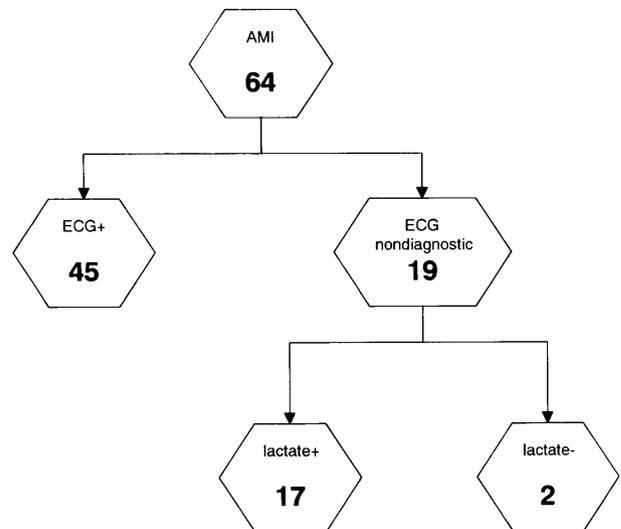
sensitivity in patients with more than two hours of chest pain.

The sensitivity of the first ECG to diagnose AMI is shown in Figure 6. Of the 64 patients with AMI by WHO criteria, 45 (71%) had a diagnostic ECG at point of care. Of the 19 patients with a normal or non-diagnostic ECG, 17 had elevated lactate levels, indicating that the lactate level added to the diagnostic ability of the ECG at the point of presentation.

When using 1.50 mmol/L as a cutoff, there were 326 patients with false-positive lactate levels, of whom 205 were discharged from the ED. Of the 121 admitted patients who had a falsely elevated lactate level, 68 had significant comorbidities requiring admission, such as hypotension, significant dysrhythmia, pulmonary edema, or need for urgent revascularization. An additional 15 of those 121 patients required admission



**Figure 5.** The sensitivity of cardiac markers for acute myocardial infarction by duration of chest pain. CK = creatine kinase; cTnT = cardiac troponin T.



**Figure 6.** The sensitivity of the first electrocardiogram (ECG) to diagnose acute myocardial infarction (AMI).

for intravenous therapy with either heparin or nitroglycerin, and 38 patients neither had comorbidities nor received intravenous therapy.

## DISCUSSION

Patients presenting to the ED with chest pain utilize substantial resources in their workups. Even though only 20–30% of those patients are suffering from an ACS,<sup>1</sup> many more are admitted in chest pain units for prolonged observation and may undergo expensive investigations, such as exercise electrocardiography or nuclear imaging. Despite this aggressive approach to the investigation of chest pain in the ED, 2–6% of AMI patients are missed and discharged home.<sup>2–4</sup> The outcomes for those missed patients are worse than those for a similar age-matched cohort of admitted AMI patients.<sup>4</sup> To avoid discharging patients who have an AMI, an even more aggressive approach to the investigation of chest pain might be needed and such an approach could lead to higher hospitalization rates and an even greater consumption of health care resources. A point-of-care test that is readily accessible and highly sensitive could reduce the inadvertent discharge of AMI patients while allowing for earlier discharge of low-risk patients. Physicians would, however, have to know that the test is highly sensitive and easy to use.

Such a test does not yet exist. Present cardiac markers are not sufficiently sensitive in the first six hours of symptoms.<sup>6</sup> More aggressive investigative tools, such as exercise electrocardiography, echocardiography, and nuclear imaging, may be more sensitive, but they lack specificity and are not readily available in many centers.<sup>19</sup>

The study by Schmiechen et al.<sup>15</sup> was the first to examine the use of the lactate level in the ED for the

evaluation of chest pain patients. They found that a venous lactate level greater than 1.50 mmol/L at presentation was highly sensitive for AMI, with moderate specificity. If the sensitivity of lactate for AMI is confirmed, the use of this simple test could fulfill the above criteria and allow safe discharge of a larger number of chest pain patients directly from the ED. The test would also have to be sensitive for non-AMI ACSs (previously termed microinfarcts or troponin-positive unstable angina) now included in the broader definition of AMI as proposed by the newer ESC/ACC criteria, as those patients have a short-term morbidity and mortality approaching that of AMI.<sup>20</sup> The above study did not examine the value of the lactate level in the diagnosis of unstable angina.

In our study, we separately compared the performance of the lactate level at presentation in the exclusion of AMI with two criterion standards. The first criterion standard defined by WHO is largely determined by a positive CK-MB level, and the second, newer criterion standard proposed by the ESC/ACC is based largely on a positive troponin test and would therefore appear to be a more sensitive and all-inclusive definition of AMI.

When compared with the WHO criteria, we confirmed that the lactate level at presentation is highly sensitive for AMI. It detected 59 of 64 patients with AMI. The lactate test appeared to be more sensitive than CK and cTnT levels in patients presenting within all time periods. In patients with more than two hours of symptoms, it detected all 34 patients with AMI with a sensitivity of 100% and an NPV of 100%. In comparison, for patients with two to eight hours of symptoms ( $n = 27$ ), CK and cTnT levels were only 19% and 60% sensitive, respectively. All five AMI patients with false-negative lactate results presented early within two hours of symptoms, suggesting that a certain amount of time must elapse before the pathophysiologic derangements result in significant hyperlactatemia.

As may be expected, when the performance of the venous lactate level is compared with the ESC/ACC criteria for AMI, which include microinfarcts, its sensitivity is reduced to 88% and its NPV is reduced to 96%. The pathophysiologic changes caused by these smaller infarcts may not be sufficient to cause either regional hypoperfusion or impaired cardiac extraction of lactate from the circulation, and would therefore be expected to cause a lesser rise in the level of venous lactate. However, when excluding patients who presented within two hours of symptoms, the sensitivity of the lactate level to detect microinfarcts rises to 97%. In contrast, the sensitivity of the cTnT level to detect AMI and microinfarcts prior to six hours after chest pain onset is poor.<sup>21</sup>

The lack of specificity of the venous lactate level in our study was problematic. We had 326 patients with false-positive lactate levels, of whom 205 were discharged from the ED. This is unlike Schmiechen and

colleagues' study, in which most of the false-positive lactate levels occurred in patients requiring admission for significant comorbidities.<sup>15</sup> Hence, if the lactate result had been available to the attending physician, and its limitations not well understood, it could have led to the unnecessary admission or observation of many of those discharged patients and a paradoxical increase in the use of health care resources. Readjusting the cutoff point for a positive lactate could improve the specificity of the test, but as the ROC curves demonstrate, the 1.50-mmol/L threshold closely approximates the point of maximum specificity without a significant decrease in sensitivity. It is unclear why the test had a poorer specificity than in previous studies. It is known that other disease processes can lead to hyperlactatemia, but many of the false-positive results in our population were in seemingly healthy patients who were discharged home. It is possible that the threshold established for normality (1.50 mmol/L) is too low for a given population, but elevating this threshold would detract from the test's sensitivity.

Could the addition of the lactate test have prevented the inappropriate discharge to home of a high-risk patient in our study? Of 398 patients discharged home, seven patients had an event, as defined by AMI, revascularization, or death within four weeks of discharge. Of those seven patients, only one patient received an inaccurate diagnosis of atypical chest pain in the ED, while the other six were correctly diagnosed as having cardiac pain and treated as outpatients. The missed patient's lactate level was 2.03 mmol/L, which could have prevented his discharge. However, the above-mentioned limitations regarding lactate's lack of specificity might have rendered this result meaningless in the clinical picture of this patient.

## LIMITATIONS

There were some potential limitations in our study. The first is that only chest pain patients triaged to the higher-acuity area of the department were eligible for enrollment in the study. This requirement eliminated many low-risk patients for whom a clear alternative diagnosis was available for their chest pain. Telephone follow-up was not done on these low-risk patients, and it is possible that an undetected myocardial infarction occurred in some of these patients. However, none of the low-risk patients had return visits to our institution with a cardiac complication within the study period. Excluding clinically low-risk patients from any particular test will always optimize the specificity of the given test. As such, if the test were indiscriminately used on all chest pain patients (including the lowest-risk patients), its specificity would be further reduced and the ratio of false positives to true positives would be unacceptably high. Clinical judgment is always

warranted to identify those patients who are sufficiently low-risk as to warrant no investigations at all.

A second limitation is that follow-up of discharged patients was not complete—302 of 398 discharged patients were successfully contacted at four weeks post-discharge. ED logs were reviewed for those patients lost to follow-up, and none returned to our ED with a cardiovascular event or complication within the next six months. However, it remains possible that some of them either sustained an occult AMI or sought care at a different institution.

Third, plasma lactate results may have been spuriously high in some patients due to prolonged transportation time. The analysis of plasma lactate was added to the patients' routine blood samples sent to the laboratory. Although all efforts were made to ensure that the samples were chilled following collection, it is possible that some samples were not handled under ideal conditions. It is known that cellular glycolysis generates lactate, and at room temperature, lactate can increase by 0.4 mmol/L each 30 minutes. Even on ice, lactate can increase by 0.1 mmol/L in 30 minutes.<sup>22</sup>

The criterion standard for the diagnosis of AMI changed after the completion of this trial when the ESC/ACC released their criteria in September 2000, whereby the diagnosis of AMI is now largely driven in the right context by a positive troponin measurement alone. We tried to accommodate for this change by comparing the performance of venous lactate with both criterion standards. However, only 80% of patients had a troponin measurement. Some admitted patients who did not have troponin measurements could have sustained a small AMI as defined by the ESC/ACC criteria. Consequently, we have incomplete data to determine the true performance of lactate when compared with a troponin-based definition of AMI.

Finally, we did not specifically examine the accuracy of the lactate test in patients with unstable angina (non-AMI ACS). We had decided a priori that our main interest was to determine the lactate level's sensitivity for AMI. As well, we did not have a uniform definition for unstable angina. Many patients were given that clinical diagnosis while in hospital without objective confirmation with stress testing, echocardiography, or angiography. As such, the criterion standard for those patients was rather weak and we elected not to report the performance of the lactate level for those patients.

The practical implications of this study's findings for daily clinical practice are yet to be determined. It appears that venous lactate is highly sensitive for detecting AMI, particularly after two hours of symptoms, and yields additional information to that offered by the ECG. Its high NPV in such patients can exclude the presence of AMI, but at a cost of poor specificity. However, it is not yet apparent how it

would fit in a diagnostic strategy that involves the use of other biochemical markers. It is clear that its sensitivity is greater than both CK and cTnT levels in the initial hours of presentation, but we have not compared it with another early marker such as the myoglobin level to assess whether it could provide superior diagnostic performance in that initial stage. If the treating physician did not understand the limitations of its specificity, inappropriate interpretation of results could lead to increased use of investigations. More importantly, we do not yet know whether lactate can provide important prognostic information for all types of ACSs and for chest pain patients in general. Since the time of our study, there has been a recent shift in the cardiovascular literature toward assessing a test's ability to risk-stratify chest pain patients in addition to providing diagnostic information. Further studies should examine the above questions before we decide to incorporate lactate testing in current practice.

## CONCLUSIONS

When comparing the ability of the venous lactate level to detect AMI with the criterion standard given by the WHO classification, lactate level at presentation is highly sensitive in its ability to exclude AMI in ED patients with chest pain, particularly in patients presenting with more than two hours of symptoms. However, its poor specificity would restrict its role to a screening tool in the risk-stratification of chest pain patients in the ED. Further study is required to determine its utility in diagnosing and risk-stratifying all types of ACSs within the existing framework of cardiac markers.

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