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# Venous glucose and arterial lactate as biochemical predictors of mortality in clinically severely injured trauma patients—A comparison with ISS and TRISS☆

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ABSTRACT

*Background:* Early assessment of injury severity is important in trauma. Trauma scores are calculated after the fact and are useful for audit and research, but not in the emergency clinical setting. Glucose metabolism is altered in trauma, and we hypothesised that alterations in glucose and lactate levels would be an early predictor of mortality.
*Methods:* Review of trauma registry data identified 1197 patients between May 2000 and September 2006 who had a trauma-team call out. Data collected included trauma scores, venous glucose (gluc), and arterial lactate (lact) on arrival. The predictive value of these variables was compared by ROC curves.
*Results:* The mortality rate for patients with gluc >11.0 mmol/L was 13.4% compared to 1.8% in those with gluc ≤11.0 mmol/L ($p < 0.0001$). Gluc had a specificity of 93.2% and a sensitivity of 37.9% for death. 13.0% of patients with lact >2.0 mmol/L died, versus 2.7% with lact ≤2.0 mmol/L, ($p$ 0.0003, specificity 56.8% and sensitivity 81.0%). Glucose was the better biochemical predictor of mortality compared to lactate (ROC area 0.845 and 0.716, respectively). The TRISS (trauma and injury severity score) was a very accurate predictor (ROC 0.963), whereas the ISS (injury severity score) significantly less so (ROC 0.854). There was a significant correlation between gluc, ISS, and TRISS ($p$ 0.01), as well as lactate and ISS ($p$ 0.01).
*Conclusion:* Glucose and lactate can predict mortality in severe trauma. The predictive value of glucose is comparable to that of ISS, and can be more easily employed in the clinical setting.


## Introduction

Early assessment of injury severity is important in trauma. Patient treatment and disposition (ward versus intensive care unit) is influenced by these initial assessments, and the ability to predict the burden of a particular injury should lead to improved patient care.

Several trauma scores have been devised to predict injury severity and risk of mortality. The injury severity score (ISS)[13] is the most commonly used internationally,[14,18,28,29,32] despite criticism in the literature regarding the statistical limitations of this score,[15] and the fact that it does not include any measure of physiological compromise, which is a fundamental component of clinical severity assessment. The trauma and injury severity score (TRISS) was developed to compensate for this. It includes various physiological markers and is regarded as the international standard in trauma scoring, but requires as many as 10 variables to compute, which increases the likelihood of missing data.[15] Regardless of the accuracy of trauma scores, it is abundantly clear that their use in clinical decision making is limited. They are complex to calculate, and therefore are usually determined after the fact for the purposes of audit and research. To this end, several studies have attempted to identify biochemical and physiological markers that reflect physiological compromise, in order to predict morbidity and mortality.

It has long been established that glucose metabolism is altered in trauma. In 1955 Howard showed that hyperglycaemia in trauma patients was proportional to the degree of injury.[11] The physiological mechanisms behind this are multi-factorial; increased levels of stress hormones result in an overproduction of endogenous glucose.[34] There is also an inappropriately low insulin level for the degree of glycaemia, and this has been shown to be proportionate to trauma severity.[22] Recently, there has been much discussion in the literature on the effectiveness of aggressive glucose control with insulin to improve outcomes.[8,30,31,35] The publication of the Host

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Response to Injury Large-Scale Collaborative Research Program stated that to date there is no level 1 data to support tight glycaemic control in severely injured patients, but given the plethora of data linking hyperglycaemia to mortality, this would be a "prudent component of ICU care".[10]

In 1971, McNamara et al. demonstrated a correlation between lactate and elevated blood sugar among combatants in the Vietnam War who had sustained an injury.[21] Initially it was thought that lactate levels were a reflection of anaerobic metabolism caused by tissue hypoperfusion, combined with a reduction in lactic acid elimination by the hepato-renal system in a shocked patient.[27] Recent evidence suggests that while lactate levels are increased in times of stress, this is not necessarily accounted for by tissue hypoxia, and may reflect increased aerobic glycolysis in skeletal muscle secondary to adrenaline stimulated $Na^+$, $K^+$-ATPase activity.[13,23]

Thus, we hypothesised that alterations in admission glucose and lactate levels would be an early predictor of mortality. We sought to measure this predictive value in a subset of clinically severely injured trauma patients, and compare it to the predictive value of ISS, and TRISS scores in this subset of patients.

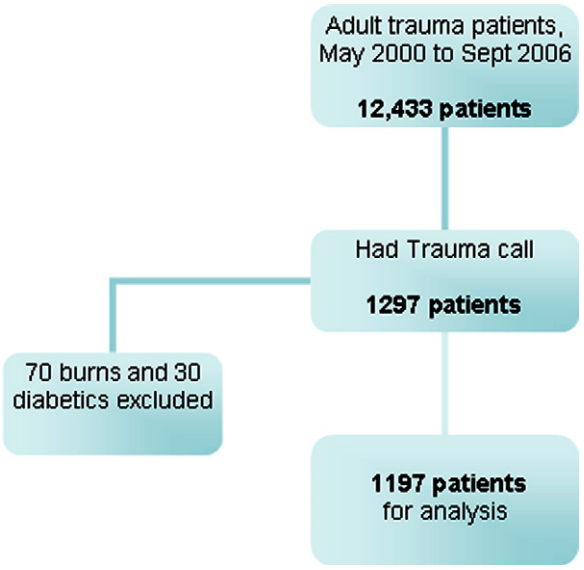


**Figure 1.** Outline of patient selection and exclusion.

## Materials and methods

Middlemore is a tertiary hospital in Auckland, New Zealand covering a population of 460,000 people.[1] The trauma registry (Collector for Windows Data Management System version 3.37, Digital Innovation Inc, Maryland, USA), is a prospectively collected database maintained by a specialist trauma nurse and trauma fellow. Trauma patients 15 years of age and older, who presented between May 2000 and Sept 2006 were identified. This subset was then searched for patients who had a trauma team call-out on arrival to the emergency department (ED). This call-out is activated by emergency department staff when community paramedics alert them by phone, or when the patient arrives in the ED if no such alert took place. An ED physician or senior nurse will activate the call-out if they think that specialty general surgery or intensive care input is needed at the outset. This decision is generally based on mechanism of injury, GCS, estimated blood loss, and/or evidence of physiological compromise on vital sign measurement. We used this as our criteria for defining severe injury, as we felt that using a definition based on trauma score or physiological parameters alone would introduce a prediction bias which would be inappropriate given the aim of the study. Additionally, this is the subset of patients for whom clinical prediction of mortality would be most useful in the clinical setting. Known diabetics and burns patients were excluded, but patients on steroid treatment were not.

Trauma registry data and electronic laboratory results were analysed for demographics, mechanism of injury, ISS score, TRISS score, venous glucose on admission (gluc), arterial lactate on admission (lact), and mortality. Mortality in our study was defined as all-cause death directly related to the index trauma event, based on data in the registry as well as review of electronic patient records. We did not look at the timing of death or analyse specific causes.

Statistical analysis was performed using SPSS (Statistical Package for the Social Sciences version 13.0 for Windows, Lead Technologies Inc, United States). Categorical data were compared using Fisher's exact test, and continuous data were correlated using the Pearson method for correlation. $p$-Values $<0.05$ were considered statistically significant. The predictive value of these variables was determined using receiver operating characteristic curves (ROC).

## Results

In total, 12,433 trauma patients presented between May 2000 and Sept 2006. 1297 of these had a trauma call out. 70 burns patients and 30 diabetics were excluded leaving 1197 patients for analysis (see Fig. 1). The relatively small percentage of diabetics is not surprising given the young median age of 31 years. 892 were male (74.5%), 305 were female (25.5%), and there were 44 deaths—a mortality rate of 3.7%. A breakdown of the mechanism of injury is shown in Table 1. The distribution of age, ISS, TRISS, venous glucose, and arterial lactate is demonstrated in Table 2. Note that a significant proportion of patients did not have lactates measured on admission.

A glucose level greater than 11.0 mmol/L (200 mg/dL) was defined as being elevated.[37] In our analysis, the mortality rate for patients with a venous glucose >11.0 mmol/L was 13.4%, compared to a mortality rate of 1.8% in those with gluc ≤11.0 mmol/L ($p < 0.0001$). At this level of measurement, the glucose level had a specificity of 93.2% and a sensitivity of 37.9% for death. The positive predictive value (PPV) was low at 13.4%, but the negative predictive value (NPV) was higher than expected at 98.2%. Fig. 2 displays a graph of mortality versus glucose level grouped into increments of 1 mmol/L. The line of best fit demonstrates an increase in mortality with increasing glucose level.

Abnormal lactate was defined as >2 mmol/L.[12] 13.0% of patients with a lact >2 mmol/L died, versus 2.7% with a lact ≤2.0 mmol/L ($p = 0.0003$). At this threshold, an abnormal lactate was 56.8% specific and 81.0% sensitive for death. PPV was once again low at 13.0%, and NPV was suitably high at 97.4%.

Elevated glucose was considerably more specific than elevated lactate, whereas the lactate level was much more sensitive than the glucose level for mortality. Combining these two values in the analysis: 25.5% of patients who had gluc >11.0 and lact >2.0 died, compared to 7.1% of patients who had one or neither parameter elevated. This had a specificity of 60.7% and a sensitivity of 70.0% for death, (PPV 14.1%, NPV 93.4%).

While these results do confirm a significant correlation between gluc, lact, and mortality, they do not demonstrate the value of these variables as predictors of mortality. Receiver operating characteristic (ROC) curves are required for this (see Fig. 3). Gluc was the better biochemical predictor of mortality compared to lact (ROC area 0.845 and 0.716, respectively). The

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**Table 1**
Demographics and mechanism of injury.

| Mechanism of injury | Number | Percentage (%) |
|---|---|---|
| Road traffic accident | 746 | 62.3 |
| Assault | 152 | 12.7 |
| Fall | 102 | 8.5 |
| Self inflicted | 60 | 5.0 |
| Work related | 38 | 3.2 |
| Sport | 18 | 1.5 |
| Animal related | 7 | 0.6 |
| Unknown | 74 | 6.2 |
| Total | 1197 | 100 |

**Table 2**
Missing values and distribution of age, ISS, TRISS, gluc, and lact.

| | No. of missing values | Percentage of missing values (%) | Median | Highest value | Lowest value |
|---|---|---|---|---|---|
| Age | 0 | 0 | 31 | 90 | 15 |
| ISS | 7 | 0.6 | 9 | 75 | 1 |
| TRISS | 201 | 16.8 | 0.972 | 0.997 | 0.001 |
| Gluc | 120 | 10.0 | 6.9 | 31.4 | 3.1 |
| Lact | 847 | 70.8 | 1.9 | 18 | 0.5 |

ISS, injury severity score; TRISS, trauma and injury severity score.

TRISS score was by far the most accurate predictor (ROC 0.963), whereas the ISS significantly less so (ROC 0.854). Subset analysis of the 323 patients who went to the intensive care unit (ICU) showed that gluc was second only to TRISS as a predictor of mortality (ROC 0.744 and 0.926, respectively), with a significant reduction in the value of ISS in this regard (ROC 0.667). Lact was also less predictive in these patients (ROC 0.637).

Using the Pearson method for correlation (Table 3), venous glucose correlated significantly with ISS, TRISS, number of days in hospital, number of days in ICU, and arterial lactate ($p < 0.01$). There was also a significant correlation between arterial lactate and TRISS ($p < 0.01$).

## Discussion

We sought to examine the predictive value of markers of glucose metabolism as measured by a simple blood test in this setting. Our results demonstrate a significant correlation between venous glucose, arterial lactate and mortality in trauma. While the positive predictive values were low, it is of clinical interest that for non-diabetic trauma calls, approximately 1 in 7 patients with a gluc >11.0 mmol/L died compared to approximately 1 in 56 of those with a gluc ≤11.0 mmol/L. The same can be said for arterial lactate level: approximately 1 in 8 patients with a lact >2.0 mmol/L died versus approximately 1 in 37 of those with a lact ≤2.0 mmol/L.

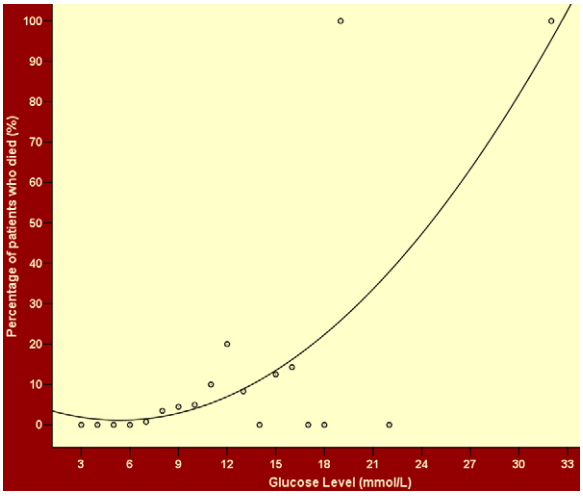


**Figure 2.** Graph of mortality versus glucose levels grouped into increments of 1 mmol/L, with line of best fit.

The ROC curve analysis demonstrates that glucose and lactate levels are *predictive* of mortality. The generally accepted threshold for a good predictive test is 0.8.[25] Based on this, the TRISS score is an exceptional predictor and ISS somewhat less so. The glucose level also meets the threshold, with a predictive value similar to that of the ISS score.

Several trauma scores have been devised to predict injury severity and risk of mortality. The ISS was developed in 1974 by Baker et al.[3] Despite the fact that there have been numerous recent publications questioning its accuracy, it is still the most commonly used trauma score internationally.[14,18,28,29,32] The ISS is derived from abbreviated injury scale (AIS) coding. The body is divided into six regions, and the ISS is the sum of the squares of the single worst AIS severity measure in the three most injured body regions. The major criticism of ISS is its statistical limitations. ISS is scaled from 1 to 75 but actually only takes 44 distinct values which are not uniformly distributed, with only six ISS values above 50. Because of this, ISS cannot be treated as a continuous measure but rather as an ordinal scale. Furthermore, as Kilgo et al.[15] demonstrated in analysis of over 350,000 trauma patients, ISS scores do not represent monotonically increasing functions of mortality. In fact, they can produce sharp declines in mortality with successive values, with a mortality of 43%, for example, with an ISS of 25, and only 14% with an ISS of 27 (a worse ISS value).

A more subjective criticism of ISS is that it does not include any measure of physiological compromise; a fundamental component of clinical severity assessment.[32] The TRISS has therefore been developed, and uses calibrated weights from logistic regression to score mortality as a function of ISS, Glasgow coma score, systolic blood pressure, respiratory rate, and age. It is regarded as the international standard in trauma scoring, but requires at least 8 and as many as 10 variables to compute, increasing the likelihood of missing or unreliable data.[15] Furthermore, trauma scores in general have limited usefulness in the acute situation, because they are complex to calculate and difficult to obtain in that situation.

There are several previously published studies examining the significance of glucose in trauma.[31] In a study by Yendamuri,[37] a significantly higher mortality, intensive care unit and hospital length of stay was found in trauma patients with both mild (gluc >135 mg/dL) and moderate hyperglycaemia (gluc >200 mg/dL). That study did not exclude diabetics. However, a study by Laird et al did, and found a gluc ≥200 mg/dL to be an independent predictor of both infection and mortality (but not gluc ≥110 mg/dL or ≥150 mg/dL).[16] This is consistent with findings from several publications by Scalea's group who have correlated hyperglycaemia with higher mortality and infection rates.[5,6,33] There has also been considerable interest in the temporal trend of glucose levels, with some studies showing that a persistent rise after admission independently predicts morbidity and mortality.[6,19,36]

The correlation between glucose and ISS has also been previously established. Pomerantz et al reported a significant direct relationship between serum glucose and ISS in 185 patients in the paediatric population. This was consistent with findings by Duane et al, who correlated gluc >150 mg/dL and ISS in 226 trauma patients.[9,26]

Lactate as a marker of physiological stress has also garnered much interest over time. An example is its usefulness in bowel

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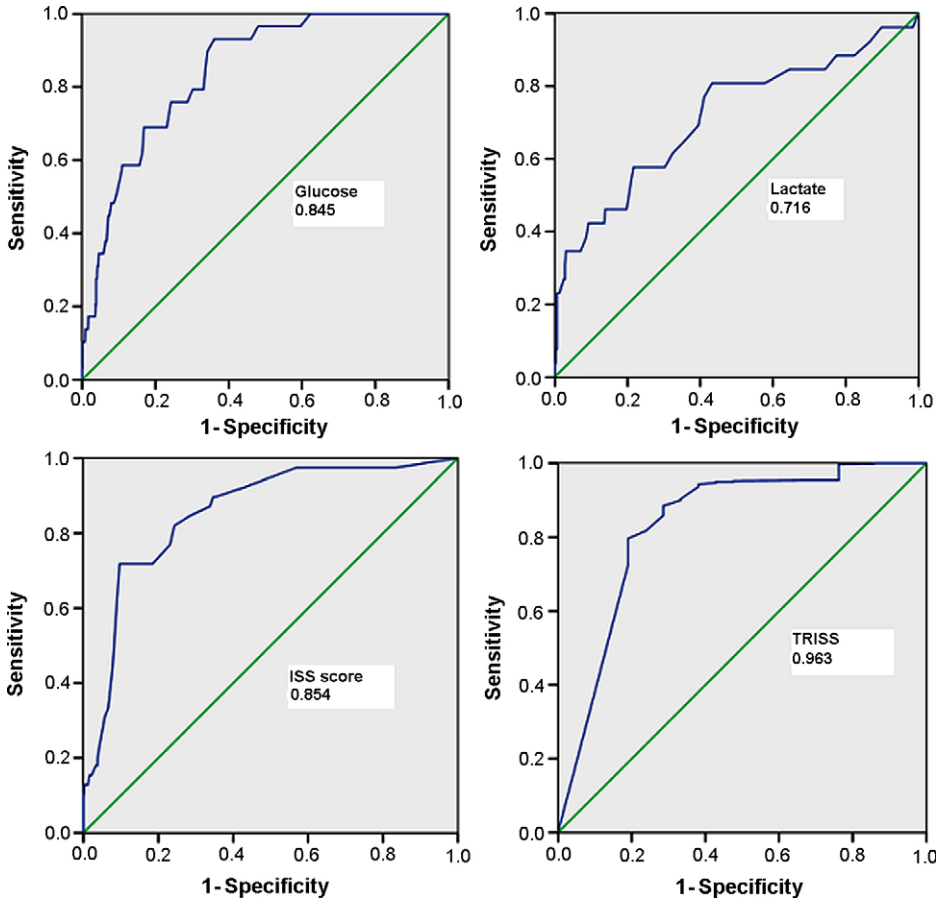


**Figure 3.** ROC curves for mortality. ISS, injury severity score; TRISS, trauma and injury severity score.

ischaemia in the absence of clinical signs and symptoms.[17] In terms of its use in trauma, this has also been investigated[2,4,7,20] although perhaps not as thoroughly as glucose. An article by Pal et al. in 2006 evaluated lactate levels in just under 6000 patients, and concluded that although elevated lactate levels were associated with death, lactate as a variable was not predictive of mortality (because the ROC curve area was only 0.72). Patients were then stratified by ISS >20, age, sex, GCS, RTS, and mechanism of injury in an attempt to see whether lactate predicted survival in these groups. It did not. Interestingly, the ROC for ISS in that series

**Table 3**
Pearson correlations.

| | Blood glucose | Lactate | ISS score | TRISS score | No. of days in hospital | No. of days in ICU |
|---|---|---|---|---|---|---|
| Blood glucose | | | | | | |
| Correlation | 1 | .356 | .399** | −.223** | .198** | .207** |
| *n* | 1077 | 349 | 1075 | 899 | 1077 | 284 |
| Lactate | | | | | | |
| Correlation | .356** | 1 | .007 | −.234** | −.066 | .047 |
| *n* | 349 | 350 | 349 | 263 | 350 | 257 |
| ISS score | | | | | | |
| Correlation | .399** | .007 | 1 | −.468** | .320** | .276** |
| *n* | 1075 | 349 | 1190 | 996 | 1190 | 322 |
| TRISS score | | | | | | |
| Correlation | −.223** | −.234** | −.468** | 1 | −.018 | −.062 |
| *n* | 899 | 263 | 996 | 996 | 996 | 250 |
| No. of days in hospital | | | | | | |
| Correlation | .198** | −.066 | .320** | −.018 | 1 | .597** |
| *n* | 1077 | 350 | 1190 | 996 | 1197 | 323 |
| No. of days in ICU | | | | | | |
| Correlation | .207** | .047 | .276** | −.062 | .597** | 1 |
| *n* | 284 | 257 | 322 | 250 | 323 | 323 |

ISS, injury severity score; TRISS, trauma and injury severity score.
** Correlation is significant at the 0.01 level (2-tailed).

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was 0.94, and this may be because all patients were included with no selection made for the more physiologically unstable as in our analysis.[25] Pal et al. also made the point that previous studies linking lactate with mortality had not included ROC curve analyses, reporting only correlations and descriptive statistics.[2,4,7,24]

Our study has several limitations. Firstly, it is a retrospective review with inherent biases as a result. Secondly, many patients did not have a lactate measured on arrival as presumably it was not clinically indicated. Thirdly, as in all trauma registries, there will always be variation in precision of the recorded data.[24] It should be noted however, that biochemical data is somewhat less subjective, and therefore less prone to human error variation. This may mean that the values recorded for gluc and lact were more accurate than those of the ISS score. While this weakens the comparison between the variables, it strengthens the argument for the use of biochemical scores to predict mortality.

Bearing the above in mind, we believe our findings will have some impact on trauma patient management within our clinical setting. We expect a higher rate of compliance for our protocol of checking venous glucose and performing an arterial blood gas in trauma resuscitations, as we have shown that these can provide useful information. More specifically, in the subset of non-diabetic patients who appear physiological well, a high glucose level on arrival might prompt emergency and surgical staff to undertake closer clinical observation, or employ further imaging as precautionary measure against a missed injury.

In summary, this is a retrospective analysis of prospectively collected data, which indicates that glucose and lactate can predict mortality in clinically severely injured patients. Venous glucose as a predictive test is at least equivalent to the ISS score, while being a simple and objective biochemical marker that is easier to obtain and more practical to use in the emergency setting.

## Conflict of interest statement

None.

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