Prehospital elevation of serum lactate is associated with worse clinical outcomes

An elevated serum lactate level is an important marker of global tissue hypoxia. Prehospital lactate measurement could warn paramedics of pending organ failure despite normal global haemodynamic parameters. PA van Beest et al. studied the feasibility of implementing prehospital lactate measurements and the relation between lactate levels and clinical outcomes.

This was a retrospective ambulance chart review from prospectively obtained data. Ambulance personnel were given a 2-month training program before prehospital lactate measurement was started. Patients were divided into a clinical shock group and a clinical non-shock group. The clinical shock group was subdivided into those with a prehospital lactate < 4 mmol/l and ≥ 4 mmol/l. Capillary or venous lactate levels were measured using the Accutrend lactate meter, Roche Diagnostic GmbH. In 50% of potential cases, prehospital lactate was actually measured. The most important barriers were the fact that lactate measurement was considered time consuming, there was little experience in handling the apparatus and, there was non-compliance with the inclusion criteria. A total of 216 charts were considered fit for inclusion (non shock N = 81, shock N = 135). The median lactate level was significantly higher in the shock group (3.9 versus 2.8 mmol/l, p < 0.0001). Length-of-stay in the ICU and the hospital was also significantly longer in the shock group as was the mortality rate (26.7 versus 1.2%, p < 0.0001). In the shock group, 74 patients had a lactate level < 4 mmol/l and 61 had a lactate ≥ 4 mmol/l. patients with a lactate level ≥ 4 mmol/l more often needed endotracheal intubation, had a longer length-of-stay in the ICU and in the hospital and had a higher mortality (44.3 versus 12.2%, p = 0.002). The ROC curve for lactate predicting in-hospital mortality was high (0.827) with an optimal cut-off point of 3.2 mmol/l. Normotensive patients with a lactate level ≥ 4 mmol/l also had an increased mortality compared to normotensive patients with a lactate level < 4 mmol/l.

Despite the evident shortcomings in design, this study clearly shows the feasibility and potential importance of prehospital serum lactate measurements. The authors thoroughly discuss the barriers for implementation and give suggestions for further improvement. However, the most important issue still needs clarification: will aggressive prehospital treatment of elevated lactate levels improve outcome? As van Beest et al. already have experience with implementing prehospital lactate measurements, they would appear perfectly suited to perform such a study.


Somatosensory evoked potentials may also predict a poor neurologic outcome during mild hypothermia after cardiopulmonary resuscitation

The bilateral absence of the cortical N20 responses of median nerve somatosensory evoked potentials (SSEP) 24 hours after cardiopulmonary resuscitation (CPR), invariably predicts a poor neurologic outcome. It is unclear whether SSEP is also helpful in patients treated with mild hypothermia and subsequent prolonged use of sedatives. Bouwes et al. performed a pilot study to find the answer to this question.

This was a prospective cohort study conducted in two centres that included comatose patients admitted to the ICU after CPR and treated with mild hypothermia (32 - 34 °C). The authors tested the following hypotheses: absence of bilateral cortical N20 responses during hypothermia persists during normothermia and is invariably associated with a poor neurologic outcome. The results of the hypothermic SSEP were not available for the treating physician.

After the exclusion of one patient with a subarachnoid haemorrhage, a total of 77 patients were included. After 30 days, 51 (66%) patients had a poor neurologic outcome. During hypothermia bilateral cortical N20 responses were absent in 13 patients (median time from resuscitation = 20 hours, range 14 - 24). Three patients died before a SSEP during normothermia could be performed. Of the 10 remaining patients, 9 also had absent bilateral cortical N20 responses during normothermia (median time from resuscitation 63 hours, range 49.5 - 90.5) and in one patient responses could not be determined (PPV 1.0, 95% CI 0.7 - 1.0).

The neurologic outcome in patients with absence of bilateral cortical N20 responses during hypothermia was invariably poor (PPV 1.0, 95% CI 0.7 - 1.0). Only one patient with presence of bilateral cortical N20 responses during hypothermia had absent responses during normothermia.

This is an important pilot study suggesting that absence of bilateral cortical N20 responses during mild hypothermia after CPR is also associated with a dismal neurologic prognosis. These results should be confirmed in a larger study before we change clinical practice. Furthermore, only a small percentage of patients with a poor neurologic outcome after CPR actually have absence of bilateral cortical N20 responses. Therefore, other early indicators of a poor neurologic outcome after CPR are still needed.