

Commentary

Increased blood lactate levels: an important warning signal in surgical practice

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Abstract

Both in emergency and elective surgical patients increased blood lactate levels warn the physician that the patient is at risk of increased morbidity and decreased chances of survival. Prompt therapeutic measures to restore the balance between oxygen demand and supply are warranted in these patients.

Keywords lactate, lactic acid, morbidity, mortality, trauma, surgery

When first described by Gaglio in 1886, measurement of lactate levels required the collection of 100–200 ml blood and took several days to complete. The labour-intensive nature of early lactate measurement techniques limited their clinical use, because results were not available until long after therapeutic decisions had to be made. In 1964 Broder and Weil [1] were the first to use a photospectrometric method to measure lactate levels in whole blood decreasing turnaround times greatly. Current handheld devices and mobile blood gas analyzers have decreased turnaround time to less than 2 min using a minimal amount of blood [2]. With this technology it is possible to diagnose, treat and monitor critically ill patients rapidly and easily.

A relationship between increased blood lactate levels and the presence of oxygen debt (tissue hypoxia) in patients with circulatory shock was suggested as early as 1927 [3]. In patients with clinical shock, associated with tachycardia, hypotension, cold and clammy skin and decreased urine output, lactate levels have been referred to as the best objective indicator of the severity of shock [4]. Can increased blood lactate levels serve an indicator function in patients without clinical signs of circulatory failure also? If so then what would increased blood lactate levels in these circumstances indicate, and what would be the most appropriate therapy in these patients?

The circulation is a demand driven system in which increases in oxygen demand are met by increases in oxygen delivery

through increases in blood flow. Tissue hypoxia can thus be defined as a state in which tissue oxygen demands are not met by tissue oxygen delivery. Decreases in haemoglobin levels and arterial oxygen saturation are usually compensated for by an increase in cardiac output to maintain global oxygen delivery [5], and so tissue hypoxia usually does not occur [6]. When cardiac function is limited this compensatory mechanism fails and tissue hypoxia can occur rapidly [7]. Many experimental and clinical studies have shown that blood lactate levels start to rise when tissue hypoxia occurs [8–11].

Because blood pressure is maintained over a rather wide range of cardiac output values, limited compensatory increases in cardiac output may fail to be noticed clinically, and thus the presence of tissue hypoxia may not always be noticed. In this issue, Meregalli and coworkers [12] show that lactate levels in haemodynamically stable postsurgical patients discriminated between survivors and nonsurvivors within the first 12 hours of admission, despite similar global haemodynamics in both patient groups. These and other authors refer to this situation of hyperlactataemia in the presence of stable haemodynamics as a state of occult hypoperfusion [13,14].

Several authors have studied the importance of increased blood lactate levels in surgical patients. Waxman and coworkers [15] studied lactate levels during and after surgery. Although cardiac output and blood pressure did not

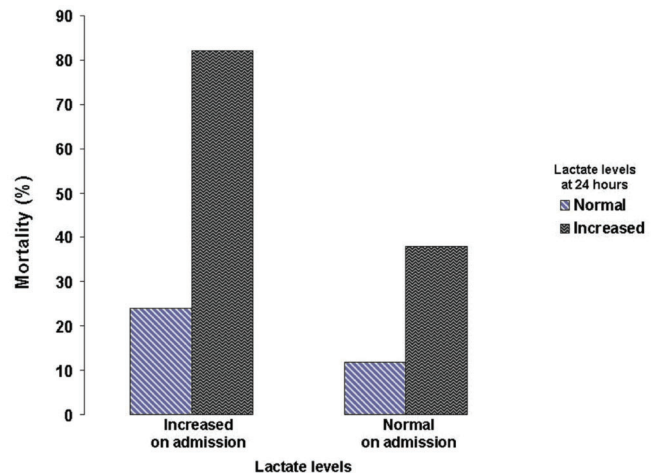
change intraoperatively, lactate levels increased. Following surgery cardiac output increased, and those authors suggested that this represented physiological compensation for the intraoperative oxygen deficits, because a significant linear relationship between calculated intraoperative oxygen deficit and lactate levels was found in that study. In a study conducted by Smith and coworkers [16] (50% of patients studied were surgical patients), those investigators showed that patients with increased blood lactate levels on admission had significant mortality (24%), even when blood lactate levels normalized within the first 24 hours. When increased blood lactate levels on admission could not be normalized within 24 hours, mortality in these patients increased to 82% (Fig. 1).

Referring to the 'golden hour' and the 'silver day' of trauma resuscitation, Blow and coworkers [14] showed that normalizing blood lactate levels within 24 hours of admission in haemodynamically stable trauma patients was associated with improved survival. Resuscitation in these patients was aimed at improving global blood flow whenever lactate levels remained above 2.5 mmol/l. Both morbidity (organ failure) and mortality were increased among those patients in whom blood lactate levels failed to normalize with these therapeutic efforts. In patients with major trauma, Claridge and coworkers [17] showed that occult hypoperfusion was associated with an increased rate of infection and mortality. In patients with a femur fracture, Crowl and coworkers [13] showed that patients with occult hypoperfusion, defined as an increased blood lactate level, had no clinical signs of shock. Nevertheless, these patients had increased morbidity compared with patients without occult hypoperfusion.

Abramson and coworkers [18] showed that normalization of lactate levels within 24 hours after resuscitation aimed at increasing oxygen delivery, and cardiac output was associated with a 100% survival. In patients requiring 24–48 hours to normalize lactate levels, mortality was 25%. None of the patients with increased blood lactate levels at 48 hours survived. Only one prospective study has shown that normal lactate levels as a therapeutic goal in surgical patients (cardiac surgery) and a resuscitation protocol aimed at increasing oxygen delivery (mainly cardiac output) was associated with improved outcome (morbidity) in the protocol group [19].

From these studies it is clear that increased blood lactate levels in critically ill surgical patients are not always related to clinical signs of circulatory failure. Treatment aimed at increasing oxygen delivery (usually aimed at increasing cardiac output) can normalize blood lactate levels in these patients. Failure to normalize increased blood lactate levels with these interventions is generally associated with increased morbidity and mortality. Limited prospective data are available, but these data also indicate that maintaining normal blood lactate levels or rapid normalization of

Figure 1



Both in patients with normal and in those with increased blood lactate levels on admission, changes in blood lactate levels during the first 24 hours of treatment were related to ultimate survival. Data from [16].

increased blood lactate levels is an important therapeutic goal in critically ill patients. Adequate fluid resuscitation and inotropes to increase cardiac output have consistently been found to improve tissue oxygen delivery in patients with tissue hypoxia, and thus remain the mainstay of therapy in these circumstances [20–24].

Where Allardyce and coworkers [25] urged referral centres for critically ill surgical patients to monitor blood lactate levels, we would urge clinicians to monitor lactate levels in all patients at risk for (occult) hypoperfusion related either to decreases in oxygen delivery or to increases in oxygen demand.

Competing interests

None declared.

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