

Commentary

The origin and interpretation of hyperlactataemia during low oxygen delivery states

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See related research by Ranucci *et al.*, <http://ccforum.com/content/10/6/R167>

Abstract

The origin of hyperlactataemia during critical illness is complex but its presence can provide an indicator of inadequate tissue oxygen delivery. Cardiopulmonary bypass (CPB) represents a unique situation where systemic oxygen delivery can be directly measured and controlled. In the previous issue of *Critical Care*, Ranucci and colleagues use this phenomenon to identify independent variables associated with the development of hyperlactataemia during CPB. In doing so they highlight the complexity of interpreting hyperlactataemia during critical illness and provide further evidence of its association with worse postoperative morbidity.

Introduction

The association of hyperlactataemia and acidosis with worsened clinical outcome has been demonstrated in numerous patient and disease states [1,2]. However, hyperlactataemia itself has complex origins and is variably associated with acidosis and with patient morbidity and mortality. The latter depends greatly on the origin of lactate production. 'Type A' hyperlactataemia is associated with anaerobic respiration, inadequate tissue oxygen delivery, acidosis, and increased morbidity and mortality [3]. 'Type B' hyperlactataemia occurs in the presence of adequate oxygen delivery with increased substrate utilisation. Differentiation between these aetiologies is important because they represent discrete metabolic processes with differing therapies and prognoses. Hyperlactataemia developing during cardiopulmonary bypass (CPB) is associated with worsened postoperative outcome [4]; although 'type A' lactic acidosis is the more commonly cited origin, this has been questioned [5].

Origin of hyperlactataemia during cardiopulmonary bypass

In the previous issue of *Critical Care*, Ranucci and colleagues report a prospective observational study undertaken to identify the source of hyperlactatemia developing during CPB

and its association with outcome [1]. Data were analysed to assess independent association between the tested variables and the peak blood lactate concentration. They concluded that hyperlactataemia was more likely during prolonged CPB time, that it was independently associated with low oxygen delivery, that it was almost invariably associated with hyperglycaemia, and that it was a predictor of worse postoperative morbidity (although not mortality).

The association with low oxygen delivery is suggestive that this underlies the mechanism for 'type A' hyperlactataemia, but is not conclusive. Reduced oxygen delivery is compatible with aerobic respiration if oxygen consumption is simultaneously decreased. Increased endogenous catecholamine production during times of such 'stress' stimulates increased blood glucose concentrations and glycolysis [6], with a resultant increase in lactate production due to 'flooding' of pyruvate dehydrogenase [7]. This is not an acidifying process because it develops under aerobic conditions; it has been termed 'stress hyperlactataemia' [8]. The presence or absence of acidosis is therefore an important mechanism for separating the aerobic and anaerobic aetiologies of hyperlactataemia.

In Ranucci's study [1], base excess was maintained within a normal range during CPB by administering bicarbonate solution. No association can therefore be made between the presence of hyperlactataemia and that of acidosis. This information may be available in surrogate form as the dose of bicarbonate required to maintain normal base excess levels in patients with or without hyperlactataemia. If the dose requirement for bicarbonate is significantly higher in the hyperlactataemia group, then the presence of 'type A' lactic acidosis can be inferred. However, if the bicarbonate requirements are the same, it raises the possibility that this is

CPB = cardiopulmonary bypass.

stress-induced 'type B' hyperlactataemia. Another marker that can separate the anaerobic and aerobic aetiologies of raised lactate is the lactate:pyruvate ratio. Under aerobic conditions with stimulated glycolysis, pyruvate synthesis increases in proportion to lactate, giving a normal lactate:pyruvate ratio of 10:1. However, once anaerobic conditions develop, lactate increases at a rate in excess of that for pyruvate, with a resultant increase in their ratio [9]. The presence of an increased lactate:pyruvate ratio in association with acidosis, low oxygen delivery and hyperlactataemia during CPB would be conclusive evidence that inadequate oxygen delivery and anaerobic mechanisms are predominating.

Conclusion

The study by Ranucci and colleagues [1] highlights the complexity involved in interpreting the significance of raised serum lactate concentrations. It eloquently demonstrates the association of reduced oxygen delivery in the development of hyperlactataemia during CPB and illustrates the association of the latter with greater morbidity during the postoperative period. Further insight is gained into the significance of monitoring serum lactate as a means of guiding oxygen delivery during CPB. Future studies may definitively identify 'type A' lactic acidosis during critical illness by demonstrating the concurrent association of hyperlactatemia, reduced tissue oxygen delivery, increased lactate:pyruvate ratio and acidosis.

Competing interests

The author declares that they have no competing interests.

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