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ABSTRACT

Lactate – from villain to guardian

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Elevated plasma lactate level is a useful warning sign in patients presenting with a variety of critical illnesses. Our understanding of hyperlactatemia, however, has improved and we can no longer associate lactate itself with any harmful effects. Lactate is a crucial intracellular buffer, and a central molecule in the interorgan exchange of carbon and redox potential. While lactate levels correlate with severity of acidaemia in shock there is little evidence to support the commonly held view that its origin is mainly secondary to anaerobic metabolism because of reduced tissue oxygen delivery. More commonly, lactate production is increased due to accelerated aerobic glycolysis caused by adrenergic stimulation and inflammation. Concurrently, splanchnic vasoconstriction in shock leads to reduced delivery of lactate to two major lactate consuming organs (liver, kidney) disturbing the normally operating interorgan lactate shuttle. The construct of tissue hypoxia and insufficient aerobic ATP production as a cause of "lactic acidosis" is commonly attributed to tissue hypoperfusion. While tissue perfusion e.g. assessed by capillary refill time, is crucial in treating shocked patients, targeting lactate clearance may lead to over-resuscitation and potentially harmful use of inotropes, intravenous fluids and supplemental oxygen. While it is crucial to restore perfusion to ischaemic tissues the goal should be the correction of blood flow and underlying pathology rather than treatment of the "lactic acidosis" per se.

Keywords: lactate, shock, acidosis, glycolysis

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