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Metabolic and electrolyte disturbance after cardiac arrest: How to deal with it

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Cardiac arrest (CA) is a sudden, severe event that causes a cascade of metabolic and electrolyte disturbances throughout the body triggered by a loss of cardiac output. Metabolic disturbances are primarily in the form of mixed metabolic and respiratory acidosis; dysglycaemia; and states of deficiency or excess in potassium, calcium, magnesium and lactate. It is known that persistent metabolic disturbances are associated with poor patient outcome following resuscitation from CA, but this might simply be a reflection of the severity of illness. Moreover, contemporary evidence for the management of metabolic disturbances to improve outcomes in these patients is scarce. Moreover, metabolic disturbances during the early post-resuscitation period remain poorly understood in terms of severity, duration and the influence of their post-resuscitation care and treatment on outcome. Although sufficient data suggest that extreme metabolic disturbances such as hypoglycaemia, severe hyperglycaemia, severe hypokalaemia and hyperkalaemia and hypomagnesaemia likely have a devastating effect and should be avoided, randomised controlled trial evidence is clearly need for the management of metabolic and electrolyte derangements in resuscitated CA patients.

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Introduction

Cardiac arrest (CA) is a common, severe event with an incidence of up to 2.0 per 1000 person years [1–3]. CA in itself carries a high mortality rate [4,5]. Among resuscitated CA patients admitted to the hospital after return of spontaneous circulation (ROSC), the reported rates of live discharge remain disappointingly low at 30–40% [6–8]. Even fewer survivors have sufficient neurological recovery to return to their premorbid status, and many have persistent long-term cognitive impairment [6,9,10].

Without prompt ROSC and adequate perfusion, CA results in irreversible organ injury, particularly of the brain and heart, subsequent multi-organ failure and eventual death [11,12]. Following ROSC, the period of whole-body ischaemia creates an abnormal physiological state and marks the onset of reperfusion injury [13]. Reperfusion injury is a complex constellation of pathological processes particularly affecting heart and brain function as well as the rest of the body [13]. Irrespective of being an in-hospital cardiac arrest (IHCA) or an out-of-hospital cardiac arrest (OHCA), the event initiaties a cascade of metabolic and electrolyte disturbances throughout the body triggered by a loss of cardiac output and a likely shift from aerobic to anaerobic metabolism [6,14,15]. Metabolic and electrolyte disturbances occur as a result of both the CA and post-resuscitation care and treatment.

The management of metabolic disturbances is an important component of post-CA care as severe derangements are associated with poor outcome [13,16]. Although metabolic processes are likely to be important for maintaining stability after ROSC, it is possible that inappropriate management of metabolic disturbances after ROSC exacerbates reperfusion injury [17–19] by contributing to mirco-vascular injury and cerebral and cardiac cellular dysfunction [20,21]. However, contemporary evidence for the management of metabolic disturbances to improve patient outcomes is scarce and largely based on observational study findings and limited to experimental animal models of organ injury [20]. Moreover, such derangements may simply be a reflection of illness severity, and the physiological effects of metabolic disturbances, including electrolyte abnormalities, during the immediate and early post-resuscitation period remain poorly understood.

In this article, we review the current data and recent advances in knowledge related to the management of metabolic disturbances following CA. We particularly focus on acid–base derangement, serum lactate and glucose metabolism and their management. We also describe and discuss electrolyte disturbances with a focus on serum potassium, calcium and magnesium.

Metabolic disturbance following CA

Metabolic disturbances commence within seconds of the CA and worsen during the immediate period of hypoperfusion and lack of flow to the body tissues. The magnitude of the disturbance is proportional to the extent of the injury, but it may be influenced by the components of care delivered during the resuscitation effort and post-resuscitation care, such as drugs, ventilation and oxygen targets and nutrition. In resuscitated CA patients, metabolic disturbances are evident as impaired acid–base status, worsening serum lactate levels and dysglycaemia. Recognising these disturbances will help clinicians make evidence-based decisions to optimise patient outcomes.

Acid-base disturbance after CA

Acidaemia may be a key contributor to cell death following an ischaemic insult [22,23]. Acidaemia following CA occurs as a result of interrupted blood flow to metabolically active tissues [24]. The cellular response to uncontrolled acidaemia is the further loss of intracellular function, accumulation of lactate and overwhelming endoplasmic reticulum stress leading to cell death [25,26]. Previous studies defined severe acidaemia as a serum arterial pH < 7.20 [14,27,28], a state that is not uncommon after CA.

During the immediate and early post-resuscitation periods, many CA patients have a low pH and either a metabolic acidaemia or combined respiratory and metabolic acidaemia [24]. Metabolic acidosis occurs at the tissue level due to the excessive accumulation of hydrogen ions (H^+), carbon dioxide (CO_2), lactate and unmeasured anions [14,19,24,29]. Respiratory acidosis is less common following CA, and its occurrence is often associated with specific ventilatory management during the active