CRITICAL CARE

COMMENTARY

Management of acidosis: the role of buffer agents

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52cc-1-2-051

Full text

For more than 50 years, continuing up to about 1980, sodium bicarbonate was used for the treatment of metabolic acidosis. The rationale was that administration of an alkaline fluid would correct an acidotic state. However, the potential value of sodium bicarbonate was called into question when more recent studies demonstrated that it induced venous hypercarbia, and decreases in tissue and cerebrospinal fluid pH, as well as provoking tissue hypoxia, circulatory congestion, hypernatremia, and hyperosmolality, with consequent brain damage [1-6]. Bicarbonate buffers may intensify rather than ameliorate cellular acidosis because sodium bicarbonate generates CO_2 and thereby increases intracellular (hypercarbic) acidosis [7].

Sodium bicarbonate administered to patients with diabetic ketoacidosis failed to favorably alter the clinical course or outcome. More specifically, the survival rate was similar in patients who did not receive bicarbonate [8]. During hypoxic lactic acidosis, sodium bicarbonate produced a decline in both systemic arterial pressure and cardiac output without improvement in outcome [9]. The declines in arterial pressure and cardiac output were associated with the hypertonicity of buffer agents which produced arterial vasodilation [10].

Several other agents have been investigated for the treatment of lactic acidosis. The intent was to increase blood pH during hypoxic states, without reducing oxygen delivery or increasing blood and tissue CO_2. Among the most promising are the organic buffers, including TRIS (THAM), and a mixture of equimolar concentrations of sodium carbonate and bicarbonate named Carbicarb. Both of these agents are CO_2-consuming, rather than CO_2-generating, bicarbonate buffers. In animal studies, both THAM and Carbicarb appeared to have advantages over sodium bicarbonate in the treatment of lactic acidosis and diabetic ketoacidosis [5,11]. However, no well-controlled human trials are available at this time and neither agent is as yet regarded as appropriate for routine management.

Only in exceptional circumstances, particularly in cases of poisoning, drug intoxication, life threatening hyperkalemia or acute epinephrine-fast broncho-constriction, is there likely to be an indication for the reversal of acidosis by the administration of buffer agents.

In settings of cardiac arrest, there is currently no secure evidence of improved outcome after buffer administration. Moreover, measurements indicate that buffer agents including sodium bicarbonate, THAM and Carbicarb do not alter myocardial pH during cardiac resuscitation [12]. Nevertheless, a minority of data, based on experimental cardiopulmonary resuscitation (CPR) studies in dogs, are cited to encourage continued use of sodium bicarbonate during CPR [13,14]. A very recent study in our laboratory may be of interest as it indicates that buffer agents, when administered during CPR, may reduce the severity of post-resuscitation myocardial dysfunction and prolong survival in animals [15] We must await confirmation from studies on patients before we see whether this will prove to be clinically applicable.

In conclusion, we cannot, at the time of writing, recommend routine bicarbonate or other buffer administration for the reversal of acidosis associated with low flow states.

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Published: 26 November 1997

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doi:10.1186/cc102
Cite this article as: Weil and Tang: **Management of acidosis: the role of buffer agents.** *Critical Care* 1997 **1**:51.