Alkali Therapy in Patients with Metabolic Acidosis

To the Editor: In his review "Acid-Base Disorders in lCU Patients, Oh[1] writes in the summary on p 66 "The treatment target … is not the acidosis, but the underlying condition causing acidosis". In contrast to this statement, Edge et al.[2] have concluded that the immediate cause of coma in patients with acidosis is the very low blood pH. The glycolytic enzyme phosphofructokinase is pH dependent, as its activity is decreasing with decreasing pH, and, thus, glucose utilization in brain cells is impaired[3]. For the readers of "Electrolyte Blood Press" it would be perhaps interesting to know, why the author did not comment the paper of Edge et al.[2].

On p 69, the author writes "HCO_3^- therapy does not improve the outcome in diabetic ketoacidosis … " Life-threatening is the most severe stage of diabetic ketoacidosis, coma. If the treatment of diabetic ketoacidotic coma included also infusions of alkalizing solutions, lethality was zero %, e g[4]. Without alkalizing solutions, lethality was up to 100%, e g[5]. Again, it would be perhaps interesting to know why the author did not comment the papers of Wagner et al.[4] and Basu et al.[5].

1) Oh YK: Acid-Base Disorders in lCU Patients. Electrolyte Blood Press 8:66-71, 2010

2) Edge JA, Roy Y, Bergomi A, et al.: Conscious level in children with diabetic ketoacidosis is related to severity of acidosis and not to blood glucose concentration. Pediatr Diabetes 7:11-15, 2006

3) Van Nimmen D, Weyne J, Demeester G, Leusen 1: Local cerebral glucose utilization in systemic acidosis. Am J Physiol 247: R639-645, 1984

4) Wagner A, Risse A, Brill HL, et al.: Therapy of severe diabetic ketoacidosis. Zero-mortality under very-low-dose insulin application. Diabetes Care 22:674-677, 1999

5) Basu A, Close CF, Jenkins D, Krentz AJ, Nattrass M, Wright AD: Persisting mortality in diabetic ketoacidosis. Diabet Med 10:282-284, 1993

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The Authors Reply: Severe metabolic acidosis can generate detremental clinical effect such as cardiovascular depression and central nervous system dysfunction[1]. It also disturbe important key enzymes' activity[2]. The effect of bocarbonate therapy aimed at correcting the pH, however, is controversial. Bicarbonate therapy produced CO_2 and paradoxically lower the intracellular pH and cerebrospinal fluid pH[3, 4]. Bicarbonate infusion is associated with an increased blood lactate levels[4]. It might be also produce the volume expansion, hypernatremia and renbound alkalemia. The other buffer agents such as Carbicarb (Na_2CO_3 + $NaHCO_3$) and THAM (Tris-hydroxymethyl aminomethane) are available, but these agents dose not imporving outcomes of metabolic acidosis[4].

Therefore, recent articles and text books suggest that therapy is aimed at correction of the underlying disorder, volume depletion, and electrolyte imbalance[4-6]. On condition that severe acidosis (pH < 7.1) and the patient is deteriorating rapidly, bicarbonate therapy can be considered.

1) Sonnett J, Pafani FD, Baker LS, et al.: Correction of intrmyocardial hypercarbic acidosis with sodium bicarbonate. Circ Shock 42:163-173, 1994

2) Edge JA, Roy Y, Bergomi A et al.: Conscious level in children with diabetic ketoacidosis is related to severity of acidosis and not to blood glucose concentration. Pediatr Diabetes 7:11-15, 2006