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

Viktor Rosival

SYNLAB Department of Laboratory Medicine, Dérer's Hospital, Limbová 5, SK-833 05 Bratislava, Slovakia, Europe.

Correspondence: Viktor Rosival, SYNLAB Department of Laboratory Medicine, Dérer's Hospital, Limbová 5, SK-833 05 Bratislava, Slovakia, Europe.
E-mail: rosivalv@hotmail.com

Electrolyte Blood Press 9:38, 2011 doi: 10.5049/EBP.2011.9.1.38

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

3) Rhee KH, Toro LO, McDonald GG, Nunnally RL, Levin DL: Carbicarb, sodium bicarbonate, and sodium chloride in hypoxic lactic acidosis. Effect on arterial blood gases, lactate concentrations, hemodynamic variables, and myocardial intracellular pH. Chest 104:913-918, 1993

4) Rose BD, Post TW: Metabolic acidosis. In: Clinical physiology of acid-base and electrolyte disorders. 5th ed., Philadelphia, WB Saunders, 2001, p578-646

5) Charfen MA, Fernandez-Frackelton M: Diabetic ketoacidosis. Emerg Med Clin North Am 23:609-628, 2005.

6) Palmer BF, Alpern RJ: Metabolic acidosis. In: Comprehensive clinical nephrology. 4th ed., St. Louis, Elsever Saunders, 2010, p155-175

Yun Kyu Oh

Department of Internal Medicine, Seoul National University Boramae Medical Center; Department of Internal Medicine, College of Medicine, Seoul National University, Seoul, Korea

Correspondence: Yun Kyu Oh M.D., Department of Internal Medicine, Seoul National University Boramae Medical Center, Seoul National University College of Medicine, 425, Shindaebang 2-Dong, Dongjak-Gu, Seoul, 156-707, Korea.
E-mail: yoonkyu@snu.ac.kr

Electrolyte Blood Press 9:38-39, 2011 doi: 10.5049/EBP.2011.9.1.39