

ions present in the solution that is balanced by the net charge on all other weak ions. In normal plasma, SID has a numerical value of about 40. Lower values of SID lead to metabolic acidosis, and higher values lead to metabolic alkalosis. In Mathes' case report, the high chloride load may have caused a reduction in the SID, which resulted in the metabolic acidosis.

One of the interesting questions resulting from Stewart's approach relates to how sodium bicarbonate corrects the metabolic acidosis. The metabolic acidosis may be corrected not so much by its bicarbonate content but by its sodium content. The increased sodium concentration resulting from bicarbonate therapy corrects the reduced SID toward normal, thereby correcting the acidosis. According to Stewart, bicarbonate is a dependent variable and therefore cannot bring about a change in another dependent variable like hydrogen ion concentration.

In conclusion, we believe that hyperchloremia caused the metabolic acidosis by altering the SID, and we do not believe that dilution of bicarbonate is a likely cause.

Pema Dorje, M.D.
Assistant Professor
Gaury Adhikary, M.D.
Lecturer
Ian D. McLaren, M.D.

Anesthesiology
1997; 87:1012-3
© 1997 American Society of Anesthesiologists, Inc.
Lippincott-Raven Publishers

Decrease in the Total Amount of Extracellular Bicarbonate Is Not Dilution

To the Editor:—Mathes *et al.*,¹ in their case report titled "Dilutional Acidosis: Is it a Real Clinical Entity?" have reported a case of metabolic acidosis that they believe is due to dilutional acidosis developing as a direct result of infusion of a large volume of isotonic saline. Although we agree that dilutional acidosis can occur when the plasma bicarbonate concentration is decreased by extracellular fluid expansion, we disagree with the authors on the following issues:

1. This case report is not a true example of "dilutional acidosis." Dilutional acidosis occurs when the plasma bicarbonate concentration decreases as a result of volume expansion with solutions that contain neither acid nor alkali.² The intraoperative metabolic acidosis that occurred in this case may not be the direct result of giving a large volume of isotonic saline. Instead, the following factors may have significantly contributed to the generation of this metabolic acidosis:

- Bicarbonate loss in conjunction with 3.5-l blood loss
- Bicarbonate loss due to electrolyte exchange from the normal saline that was used to moisten laparotomy sponges and to irrigate the surgical field
- Decreased "buffer power" of blood due to blood loss

Assistant Professor
Stephen Bogush, M.D.
Resident
Department of Anesthesiology
University of Michigan Medical Center
Ann Arbor, Michigan

References

1. Mathes DD, Morell RC, Rohr MS: Dilutional acidosis: Is it a real clinical entity? *ANESTHESIOLOGY* 1997; 86:501-3
2. Goodkin Da, Raja RM, Saven A: Dilutional acidosis. *South Med J*, 1990; 83:354-5
3. Azzam FJ, Steinhardt GF, Tracy TF, et al: Transient perioperative metabolic acidosis in a patient with ileal bladder augmentation. *ANESTHESIOLOGY* 1995; 83:198-200
4. Miller LR, Waters JH, Provost C: Mechanism of Hyperchloremic Metabolic Acidosis. *ANESTHESIOLOGY* 1996; 84:482-3
5. Stewart PA: Modern quantitative acid-base chemistry. *Can J Physiol Pharmacol* 1983; 61:1444-61
6. Fencel V, Leith DE: Stewart's quantitative acid-base chemistry: Applications in biology and medicine. *Resp Physiol* 1993; 91:1-16.

(Accepted for publication May 21, 1997.)

• Accumulation of fixed acids

Fixed acids are responsible for the formation of 50-100 mmol of hydrogen ions per day. In the acute setting, the magnitude of contribution of the kidney to the pH homeostasis may be insignificant. In this case, however, the operative procedure lasted longer than 8 h, and this length of time cannot be regarded as an acute situation. Accumulation of fixed acids during this extended period of time can contribute significantly to this metabolic acidosis. To rule out the possibility of fixed acids playing a role in the metabolic acidosis, the authors emphasized the presence of nonanion gap metabolic acidosis in this patient. Although increased anion gap may be used as a criterion to differentiate the various causes of metabolic acidosis, the possibility of nonanion gap or even decreased anion gap (as compared with baseline anion gap) exists in the presence of organic acid acidosis in conjunction with extreme hyperchloremia. In this patient, the chloride increased from 90 to 128 mEq/l.

2. Discrepancy in the proposed treatment of metabolic acidosis: The treatment of acidosis depends on its severity, any associated electrolyte/hemodynamic disturbance, and the judgment of the clinician. Although the authors state that there is no need to manage dilutional metabolic acidosis with bicarbonate, yet their patient

CORRESPONDENCE

was treated with bicarbonate dialysate in the ICU. We are in agreement in this therapy. We believe that the correction of metabolic acidosis that followed the dialysis in this case is in part due to the removal of the unmeasured fixed acids.

Arjang Khorasani, M.D.

Assistant Professor
Department of Anesthesiology
Rush University Medical College
Chairman, Division of Critical Care Anesthesiology
Department of Anesthesiology & Pain Management
Cook County Hospital
Chicago, Illinois

Samuel K. Appavu, M.D.

Assistant Professor of Surgery
Department of Surgery
University of Illinois College of Medicine at Chicago
Chairman, Division of Surgical Critical Care

Cook County Hospital

Chicago, Illinois

Simin Saatee, M.D.

Assistant Professor
Department of Anesthesiology
Rush University Medical College
Department of Anesthesiology & Pain Management
Cook County Hospital
Chicago, Illinois

References

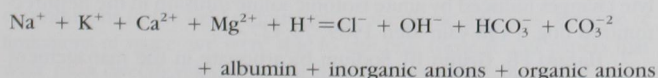
1. Mathes DD, Morell RC, Michael RS. Dilutional acidosis: Is it a real clinical entity? *ANESTHESIOLOGY* 1997; 86:501-3
2. Garella S, Chang BS, Khan SI. Dilutional acidosis and contraction alkalosis: Review of a concept. *Kidney Int* 1975; 8:279-83

(Accepted for publication May 21, 1997.)

Anesthesiology
1997; 87:1013-4
© 1997 American Society of Anesthesiologists, Inc.
Lippincott-Raven Publishers

In Reply:—We appreciate the insightful comments concerning our case report on dilutional acidosis. The primary purpose of this case report was for the readers of *ANESTHESIOLOGY* to be aware that dilutional acidosis (presenting as a hyperchloremic nonanion gap metabolic acidosis) should not be mistaken for inadequate volume resuscitation and poor end organ perfusion. Stewart's analysis of acid-base chemistry was not mentioned because currently it is not the standard measurement for pH of blood gases in clinical practice. We explained the cause of dilutional acidosis by the traditional Henderson-Hasselbalch equation.

Stewart's analysis and subsequent modifications on acid-base chemistry is based on the law of electroneutrality in aqueous solutions, in which the total number of cations equal the total number of anions¹²:



Only the independent variables of strong ions, PCO_2 (molecular CO_2) or proteins can change acid-base equilibrium. The primary independent strong ions involved in electroneutrality are sodium, chloride, and organic anions. Changes in these ions result in the dependent variables of H^+ and HCO_3^- changing to maintain electrical neutrality. The dependent variables in themselves do not change unless there is a change in the strong ions or other independent variables such as molecular CO_2 or total protein (primarily albumin).

We agree that following Stewart's analysis of acid-base chemistry, one must conclude that the infusion of high chloride solutions such as 0.9 normal saline causes an elevation in the strong ion Cl^- , which in turn causes the dependent variables of H^+ to increase and HCO_3^- to decrease to maintain electroneutrality.

Traverso *et al.* and McFarlane and Lee found a higher pH and bicarbonate level with the use of the lower chloride solutions Ringer's lactate or Plasmalyte compared with 0.9% saline.^{3,4} By Stewart's analysis,

the higher pH and bicarbonate levels from Ringer's lactate or Plasmalyte is contributed to a lower chloride load compared to 0.9% saline. However, the etiology of the higher pH and bicarbonate levels with these lower chloride solutions is difficult to interpret because lactate found in Ringer's lactate and gluconate and acetate found in Plasmalyte are metabolized to bicarbonate by the liver, and hence, act as a bicarbonate buffer raising pH and measured bicarbonate.

Such states as SIADH and psychogenic polydipsia do not cause dilutional acidosis. These states cause a chronic mild extracellular volume expansion. Dilutional acidosis is seen with acute and large increases in extracellular volume such as in trauma resuscitation. Also, as illustrated in our case report, dilutional acidosis can occur with intravascular volume depletion, and in such situations as trauma resuscitation, in which the total extracellular volume commonly increases despite a low intravascular volume. Of interest, hyponatremia caused by SIADH should cause a metabolic acidosis by Stewart's analysis. This is due to a decreased strong ion difference causing an increase of H^+ and a decrease of HCO_3^- to maintain electroneutrality.

We are skeptical that Stewart's analysis of acid-base chemistry offers a complete explanation for dilutional acidosis. The question of whether an elevation in chloride or a dilution of bicarbonate causes a metabolic acidosis with extracellular volume expansion from isotonic saline was a question investigated over 30 yr ago. Asano *et al.* compared the acidification effects of extracellular volume expansion in dogs with 0.9% saline, 5% glucose, and 5% mannitol.⁵ They found all three solutions caused the same degree of acidosis and decrease in bicarbonate. Asano *et al.* concluded the dilution of bicarbonate and not chloride elevation is the cause of metabolic acidosis with extracellular volume expansion. Rosenbaum *et al.* found in dogs that the total extracellular bicarbonate actually increased with large infusions of isotonic saline, but the relative concentration decreased due to extracellular volume expansion.⁶ By Stewart's analysis, total extracellular bicarbonate should decrease with hyperchloremia to maintain electroneutrality.