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.

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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 anions\(^2\):

\[
\text{Na}^+ + \text{K}^+ + \text{Ca}^{2+} + \text{Mg}^{2+} + \text{H}^+ = \text{Cl}^- + \text{OH}^- + \text{HCO}_3^- + \text{CO}_3^{2-} + \text{albumin} + \text{inorganic anions} + \text{organic anions}
\]

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.\(^4\) 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 SIAH 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, hypotremia caused by SIAH 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 50 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.

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Treatment of dilutional acidosis with sodium bicarbonate should be approached cautiously. We agree with Russo that severe metabolic acidosis such as lactic acidosis combined with worsening acidosis from normal saline infusion should likely be treated with conservative amounts of sodium bicarbonate and by changing to a lower chloride solution such as Ringer's lactate, if not contraindicated. However, we disagree on the necessity for treating dilutional acidosis alone without any other metabolic insult. We are not aware of any studies showing dilutional acidosis to be harmful.

Traverso et al. in the hemorrhagic swine model did find statistically nonsignificant improved survival in swine resuscitated with Ringer's lactate over 0.9% saline. Russo in his letter implied the lower chloride level with higher pH and bicarbonate level in the Ringer's lactate group to be the reason for improved survival. However, the group of swine who received Plasmalyte had a lower survival rate than the 0.9% saline group despite having lower chloride and higher bicarbonate and pH levels.

In our case report, no further sodium bicarbonate was given after the diagnosis of dilutional acidosis was made. Rapid extracellular changes in bicarbonate and chloride do not cause such changes at the intracellular level. Rosenbaum et al. found no change in intracellular pH in dogs with extracellular expansion from 0.9% saline. Sodium bicarbonate therapy has the potential for increasing intracellular acidosis by increasing the diffusion of molecular CO2.

Much investigation still needs to be performed on the etiology of dilutional acidosis and the need for treatment. Clinical studies on extracellular volume expansion comparing the effects of 0.9% saline and lower physiologic chloride solutions without any base buffer need to be done to clarify whether dilutional acidosis is caused by an increase in chloride or an actual dilution of bicarbonate. These studies should also examine changes in strong ion difference to see if Stewart's analysis is applicable to explain dilutional acidosis. Further, the measurement of intracellular pH before and after sodium bicarbonate is needed to determine if dilutional acidosis by itself is potentially harmful or should be corrected by pharmacologic means.

We appreciate Khorasani and Appavi's comments concerning other possible causes of metabolic acidosis. Although there is no way to measure the amount of bicarbonate lost secondary to blood loss or from electrolyte exchange across the open abdominal surgical field, we believe these losses were negligible and in no way accounted for the hyperchloremic metabolic acidosis. Nine units of packed erythrocytes were given for blood replacement, and we are not aware of any literature supporting the loss of significant bicarbonate through the bowel and peritoneum from surgical irritation with saline.

In our case report, the patient had a normal bicarbonate at the start of surgery, and we believe the fixed acid load was not significant after only 8 h without dialysis. We certainly recognize the potential for multiple causes of a metabolic acidosis to occur simultaneously such as dilutional acidosis combined with a lactic acidosis, ketones, or inorganic acids. Dilutional acidosis from isotonic saline has the potential for hiding an underlying anion gap acidosis by decreasing the existing anion gap from chloride elevation. Hence, we believe other causes of a metabolic acidosis such as lactic acidosis should be excluded before a clinician attributes a metabolic acidosis solely to the infusion of isotonic normal saline and extracellular volume expansion.

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