

CORRESPONDENCE

Anesthesiology
1997; 87:1009
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In Reply:—We would like to thank Dr. Appleyard for his gracious comments regarding our report "Asystole and Severe Bradycardia during Epidural Anesthesia in Orthopedic Patients." One of our main objectives in presenting this report was to make the anesthesia community aware of the severity and scope of bradyarrhythmias that may occur during an epidural anesthetic. The importance of vigilance and "heightened awareness" of common complications on the part of the anesthesiologist *intraoperatively* is expected as a component of good clinical care. Dr. Appleyard's comments highlight the important role of the anesthesiologist as a *perioperative* physician. Health care workers who provide coverage for patients in PACU settings also

should be aware of the type and severity of complications occurring after spinal and epidural anesthetics and be extensively trained in the most effective therapeutic interventions.

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(Accepted for publication May 15, 1997.)

Anesthesiology
1997; 87:1009-10
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Mechanism of Hyperchloremic Nonanion Gap Acidosis

To the Editor:—We read with great interest the case report, "Dilutional Acidosis: Is It a Real Clinical Entity?"¹ Although we praise the authors for their recognition of the development of an unusual metabolic acidosis during this surgical procedure, we question their explanation for the generated acidosis. They pointed out that their patient was persistently hypovolemic and state that, "dilutional acidosis can occur in the presence of intravascular volume depletion." By definition, dilutional acidosis occurs with volume expansion. It is this volume expansion that results in dilution of plasma bicarbonate and generation of the metabolic acidosis. This acidosis is maintained because of the effects of volume expansion on the ability of the proximal tubules to handle bicarbonate. The increased filtrate across the proximal tubules as a result of volume expansion overwhelms the proximal tubule's ability to reabsorb bicarbonate, and bicarbonaturia results.² Without this volume expansion, this classic explanation for the mechanism responsible for dilutional acidosis does not work, and another cause for the normal anion gap hyperchloremic metabolic acidosis should be explored.

We, too, have recognized that the development of a hyperchloremic metabolic acidosis is common in patients undergoing surgical procedures with large fluid shifts. In 12 patients, we found no significant decrease in tissue oxygen delivery or increased lactic acid concentrations during the course of the operative procedure. Similar to Mathes' one patient, we found no evidence of volume expansion either by direct plasma volume measurements or pulmonary artery pressure monitoring. Therefore, we were unable to explain the development of the nonanion gap hyperchloremic acidosis by the development of a dilutional acidosis. It was only when we considered the Stewart approach to acid-base balance that we were able to propose a mechanism for this hyperchloremic acidosis.³

Stewart's approach is a mathematically based model of acid-base

balance where the independent variables affecting $[H^+]$ are clearly determined.⁴ These variables are the pCO_2 , the albumin concentration, and the strong ion difference (SID). Of these independent variables, the SID is the most important in determining the final pH of body fluids.⁵ The SID is the difference between the strong cations (Na^+, K^+) and the strong anions ($Cl^-,$ lactic acid). Strong electrolytes are molecules that completely dissociate when in water, examples being HCl and NaOH. In a solution containing any collection of strong electrolytes, the $[H^+]$ is determined by the difference between the positively charged and the negatively charged strong ions. To satisfy the law of electroneutrality, any difference in the strong ions should be balanced by changes in $[H^+]$ or $[OH^-]$. A decrease in SID is associated with acidosis, and an increase in SID is associated with alkalosis.

The most dramatic finding among our patients was the development of a highly significant hyperchloremia and a decrease in SID during the course of these prolonged operations. We suggest that the hyperchloremic acidosis consistently observed in these patients undergoing prolonged surgical procedures is related to the chloride load inherent in the intravenous fluid (normal saline) they received. In this case report, the patient received 20 l of normal saline solution intraoperatively. We admit that the literature concerning hyperchloremic metabolic acidosis after saline infusion is relatively sparse,⁶⁻⁸ although reports of hyperchloremic acidosis with the use of hypertonic saline for volume resuscitation would support this hypothesis.⁹⁻¹⁰ We have observed this hyperchloremic acidosis in virtually every patient who undergoes large fluid shift procedures where large volumes of normal saline solution are administered.

Some anesthesiologists interpret intraoperative acidosis to represent hypovolemia, tissue hypoperfusion, and lactic acidosis. The common practice of chasing this acidosis with more fluid may be worsening the acidosis rather than correcting it. Metabolic acidosis can