CORRESPONDENCE

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Saline Infusion, Acidosis, and the Stewart Approach

To the Editor:—The report by Scheingraber *et al.*[1] highlights the phenomenon of acidemia after infusion of 0.9% saline in the perioperative period. The accompanying editorial[2] discusses several relevant points; however, we are disappointed that neither the article nor the editorial addresses the central issue of the relative merits of the Stewart approach[3] in describing acid-base physiology and pathophysiology.

Compared with the Henderson-Hasselbalch approach, the Stewart approach has a number of appealing features. (1) The control of acid-base and water homeostasis can be explained in terms of both sodium and chloride regulation. (2) Acid-base status is partly controlled by a number of plasma electrolytes, notably sodium and chloride. These electrolytes can be manipulated in the clinical setting to optimize acid-base status. (3) The factors controlling acid-base status are independent. Criticisms of the Henderson-Haselbalch approach include a lack of independence between carbon dioxide and bicarbonate.[4] (4) The Henderson-Hasselbalch approach does not allow assessment of nonvolatile buffers, whereas the Stewart approach explicitly includes assessment of weak acids.[4]

Comparison of the Stewart and Henderson-Hasselbalch approaches is complicated by the fact that both approaches adequately describe the acid-base end point, as Scheingraber *et al.* demonstrate.[1] Further study is required to determine which approach better describes the mechanisms of acid-base physiology.

Previous animal studies[5] have suggested that the alkalinizing effect of lactate-containing solutions in acute resuscitation is time dependent, which underscores the concept of lactate as a strong ion. The removal of lactate from the circulation will increase the strong ion difference and reduce acidosis.[3] This effect may be supplemented by further increases in the strong ion difference associated with lactate metabolism[6]; in contrast, added chloride ions appear to persist longer in the circulation. Subsequently, a smaller strong ion difference is maintained along with greater acidosis, as seen in the report by Scheingraber *et al.*[1]

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References

1. Scheingraber S, Rehm M, Sehmisch C, Finsterer U: Rapid saline infusion produces hyperchloremic acidosis in patients undergoing gynecologic surgery. ANESTHESIOLOGY 1999; 90:1265–70

2. Prough DS, Bidani A: Hyperchloremic metabolic acidosis is a predictable consequence of intraoperative infusion of 0.9% saline (editorial). ANESTHESIOLOGY 1999; 90:1247–9

3. Stewart PA: Modern quantitative acid-base chemistry. Can J Physiol Pharmacol 1983; 61:1444–61

4. Fencl V, Leith DE: Stewart's quantitative acid-base chemistry: Applications in biology and medicine. Respir Physiol 1993; 91: 1–16

5. Traverso LW, Lee WP, Langford MJ: Fluid resuscitation after an otherwise fatal hemorrhage: I Crystalloid solutions. J Trauma 1986; 26:168–75

6. White SA, Goldhill DR: Is Hartmann's the solution? Anaesthesia 1997; 52:422–7.

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Article Supports Findings of Previous Comparison

To the Editor:—The article by Scheingraber *et al.*[1] supports the findings of a previous comparison of saline with a balanced salt solution carried out by McFarlane and Lee in 1994.[2] The accompanying editorial by Prough and Bidani described this study as a clinical report of the administration of "unusually large volumes of saline."[3] The study was, in fact, a randomized-controlled comparison of saline with a balanced salt solution, both of which were administered at 15 $ml \cdot kg^{-1} \cdot h^{-1}$. This rate of administration was half the rate used by Scheingraber *et*

al.[1] McFarlane and Lee studied 30 patients with a mean weight of approximately 60 kg who were undergoing surgery that lasted approximately 200 min; therefore, the mean volume of fluid administered would be approximately 3 l, which cannot be considered "unusually large." Unfortunately, neither the title of this article nor the key words include the terms "randomized comparison," "acidosis," or "hyperchloremia," which may explain why the article was neglected. The report is completely substantiated by the subsequent article by Scheingraber *et al.*,[1] although the acidosis caused by the administration of the saline solution was less severe because the dose of saline was less. In addition, McFarlane and Lee[2] reported that the plasma chloride values had returned to normal after 24 h.

I agree wholeheartedly with the editorial comment[3] that the Stewart approach to acid-base balance contributes greatly to understanding these phenomena, and that current thinking is often muddled, as shown by a recent survey[4] and the subsequent correspondence.[5] Much of this debate, regardless of whether it acknowledges previous studies, fails to properly address the potential harm from hyperchloremia. Some argue that hyperchloremia is harmful,[6] whereas others, including the authors of the editorial, consider that hyperchloremia is not harmful,[2] but cite no supporting evidence. If hyperchloremia has important adverse effects, why have they not yet become apparent? A recent volunteer study suggests that subjective mental changes can occur more readily after sodium chloride administration.[7] A prospective randomized study of clinical outcome may be justified because it is outcome rather than surrogate measures, such as biochemical values, that are of clinical importance.

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References

1. Scheingraber S, Rehm M, Sehmisch C, Finsterer U: Rapid saline infusion produces hyperchloremic acidosis in patients undergoing gynecologic surgery. ANESTHESIOLOGY 1999; 90:1265–70

2. McFarlane C, Lee A: A comparison of Plasmalyte 148 and 0.9% saline for intra-operative fluid replacement. Anaesthesia 1994; 49:779–81

3. Prough DS, Bidani A: Hyperchloremic metabolic acidosis is a predictable consequence of intraoperative infusion of 0.9% saline. ANESTHESIOLOGY 1999; 90:1247–9

4. White SA, Goldhill DR: Is Hartmann's the solution? Anaesthesia 1997; 52:422–7

5. Drummond GB: Is Hartmann's the solution? (correspondence) Anaesthesia 1997; 52: 918–9

6. Russo MA: Dilutional acidosis: A nonentity? ANESTHESIOLOGY 1997; 87:1010–1

7. Williams EL, Hildebrand KL, McCormick SA, Bedel MJ: The effect of intravenous lacated Ringer's solution versus 0.9% sodium chloride solution on serum osmolality in human volunteers. Anesth Analg 1999; 88:999–1003

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Avoiding Iatrogenic Hyperchloremic Acidosis—Call for a New Crystalloid Fluid

To the Editor:—Scheingraber *et al.*[1] have provided further evidence that hyperchloremia causes acidosis and draw attention to this clinical problem. However, the authors suggest that iatrogenic hyperchloremic acidosis may be benign. This may be true in relatively healthy patients subjected to limited hyperchloremic insults, because the hyperchloremia is corrected by the subsequent chloruresis. The concern is the effect of more severe hyperchloremia secondary to aggressive fluid resuscitation in acutely ill patients undergoing major trauma surgery, burn debridements, vascular surgery, and liver transplantation. In vascular surgery, lactic and carbonic acid load from the distal segment may be superimposed on the iatrogenic hyperchloremic acidosis at the time of unclamping the aorta.

Animal studies suggest that hyperchloremia causes renal vasoconstriction[2,3] and its affect on other organ functions are not known.

It is a matter of concern that hyperchloremia may be playing a contributory if not a major role in the pathogenesis of renal insufficiency or failure that may be frequently seen in patients requiring massive resuscitation. Until the safety of hyperchloremic acidosis is established, it seems prudent to avoid 0.9% saline during massive resuscitation. This avoidance may be more easily said than done; one consequence of massive resuscitation is increasing hyperkalemia caused by the use of blood products. The hyperkalemia is of special concern if the patient is already in renal failure. Substituting 0.9% saline by the commercially available normochloremic fluids such as lactated Ringer's injection, Normosol (Abbott), and Plasma-Lyte (Baxter) is likely to compound the problem of hyperkalemia, because these fluids contain potassium. This situation is best exemplified by the the case report where a patient undergoing bilateral nephrectomy for polycystic kidney disease required 20 l normal saline, along with blood products.[4]

One means of avoiding hyperchloremia and hyperkalemia is to use a fluid with the following composition: Na^+ = 140 mEq/l, Cl^- = 100 mEq/l, and lactate or bicarbonate = 40 mEq/l. Currently, the only way one can get such normochloremic- and potassium-free fluid is to have the hospital pharmacy prepare it on request from the physician.

Clearly, further studies are needed to better understand the pathophysiology of hyperchloremic metabolic acidosis in acutely ill patients. We think that until such data are available, the conservative and logical approach should be to avoid iatrogenic hyperchloremia. This is more