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 editorial2 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 approach3 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-Hasselbalch 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 studies5 have suggested that the alkalizing 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.5 This effect may be supplemented by further increases in the strong ion difference associated with lactate metabolism6; 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

David A. Story, M.B.B.S(Hon), B.M.Sci(Hon), F.A.N.Z.C.A.
Staff Anaesthetist
Department of Anaesthesia
davids@austin.unimelb.edu.au
Frank Liskaser, M.B.B.S., F.A.N.Z.C.A.
Staff Anaesthetist
Department of Anaesthesia
Rinaldo Bellomo, M.B.B.S., M.D., F.R.A.C.P.
Associate Professor
Intensivist and Director of Research
Department of Intensive Care
Austin and Repatriation Medical Centre
Austin Hospital
Heidelberg
Melbourne, Victoria 3084, Australia

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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 ⋅ kg⁻¹ ⋅ h⁻¹. This rate of administration was half the rate used by Scheingraber et al.
CORRESPONDENCE

G. B. Drummond, M.B., Ch.B., F.R.C.A.
Department of Anaesthetics
Royal Infirmary
Edinburgh, Scotland

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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 debridement, vascular surgery, and liver transplantation. In vascular surgery, lactate and carbonic acid load from the distal segment may be superimposed on the iatrogenic hyperchloremic acidosis at the time of undamping the aorta.

Animal studies suggest that hyperchloremia causes renal vasoconstriction2,3 and its effect on other organ functions are not known.

It is a matter of concern that hyperchloremia may be playing a contributory role in the pathogenesis of renal insufficiency or failure, thus 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 isotonic crystalloid fluids such as lactated Ringer's solution, 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 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 hyperkalemia and hyperchloremia is to use a fluid with the following composition: Na+ = 140 mEq/l, Cl− = 100 mEq/l, 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

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