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Integration of acid-base and electrolyte disorders

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Integration of Acid–Base and Electrolyte Disorders

Richard D. Vaughan-Jones, Ph.D. and Walter F. Boron, M.D., Ph.D.

University of Oxford, Oxford, United Kingdom
Case Western Reserve University, Cleveland, OH

Walter F. Boron: wfb2@case.edu

TO THE EDITOR

In his review on acid–base disorders, Seifter (Nov. 6 issue) attempts to integrate the “traditional, bicarbonate-centered model” described by Davenport and Boron with Stewart’s strong-ion-difference model:

\[
\text{strong ion difference (in mmol per liter)} = [\text{Na}^+] + [\text{K}^+] + [\text{Ca}^{2+}] + [\text{Mg}^{2+}] - [\text{Cl}^-].
\]

In unbuffered salt solutions,

\[
\text{strong ion difference} = [\text{OH}^-] - [\text{H}^+] 
\]

and is thus a pH surrogate. However, adding buffers as strong ion salts (e.g., sodium bicarbonate equilibrated with carbon dioxide, or sodium lactate equilibrated with lactic acid) increases the strong ion difference while having variable or even no effects on pH. Thus the strong ion difference does not uniquely define pH. Changes in the strong ion difference are a consequence of adding acids or bases as strong ion salts; they do not cause pH to change. Stewart makes the fundamental error of mistaking correlation for causation. Proteins are generally sensitive to pH per se, not the strong ion difference. No biologic mechanisms exist for directly sensing or regulating the strong ion difference in cells, blood, or other compartments (e.g., the cerebrospinal fluid) — domains where pH is both sensed and regulated. Seifter advocates the strong ion difference as a diagnostic tool, rather like the anion gap. Although this analysis may indirectly provide information on acid–base control, the strong ion difference offers no new mechanistic insight because it does not have a causal role in pH changes.

References


No potential conflict of interest relevant to this letter was reported.
