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THE POISON PEN

Don't Throw the Sodium Bicarbonate Out with the Correlation

Alexander M. Sidlak[1] · Anthony D. Scoccimarro[2] · Ryan T. Marino[3]

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Dear Editors,

We read Shastry et al.'s paper on antidotal sodium bicarbonate with great interest and have concerns regarding the implications of this study for the practice of medical toxicology [1]. We fear that despite some limitations which have been noted by the authors themselves, practitioners may limit their use of sodium bicarbonate, which has previously been shown to benefit toxicities from multiple compounds with sodium channel–blocking properties [1, 2]. The present study makes a claim similar to a historic one—a claim later disproven as a clinical susceptibility bias—("Levophed, leave em' dead") purporting the danger of vasopressors whereas what was being identified was that those patients requiring vasopressors were more critically ill [3].

Although the authors correct for some confounding variables, we wonder if more strict inclusion criteria may have strengthened the conclusions. Many of the inclusion criteria were overly broad. For instance, a salicylate level of 20 mg/dL would not prompt treatment with sodium bicarbonate nor would a QRS of 101 ms [4, 5]. By only choosing those criteria by which most toxicologists initiate sodium bicarbonate therapy (SBT), we wonder if more equal groups would have been created and whether the same conclusions would have been reached.

Secondly, we question if specific additional data could have been obtained that would have better identified key differences between the control and treatment groups and solidified any claims of causation. Many potentially important confounding variables are not identified. For instance, there is no accounting fora patient's initial hemodynamics, no information on whether a patient's QRS widening was new or preexisting, and no data addressing differences in the initial pH of patients with salicylate toxicity or of their initial mental status. All of these factors help to determine the need for therapy and would help us better determine if like groups were being compared. Likewise, as the proposed pathophysiologic mechanism for harm was SBT-induced hypokalemia, additional data on the development of hypokalemia or of torsade de pointes would have strengthened the causative link made in this study.

In summary, we do not believe that this correlative evidence is cause for reappraisal of our use of bicarbonate. Certainly, patients with more significant toxicity requiring a longer duration of sodium bicarbonate therapy need close monitoring, but in our opinion, patients with more significant toxicity have worse outcomes because they are sicker and withholding any therapy from them would simply increase this mortality. We should certainly reconsider our use of therapies in toxicology where evidence is limited, but the future analysis we hope can be conducted is at what cutoff of salicylate level, QRS, or pH do the benefits of SBT outweigh the risks.

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Declarations

Competing interests None.

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✉ Alexander M. Sidlak
alex.sidlak@envisionhealth.com

[1] Department of Emergency Medicine, Inova Fairfax Medical Campus, 3300 Gallows Road, Falls Church, VA 22042, USA

[2] Department of Emergency Medicine, Lincoln Medical and Mental Health Center, Bronx, NY, USA

[3] Division of Medical Toxicology, Department of Emergency Medicine, University Hospitals, Cleveland, OH, USA

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