Abstract:

BACKGROUND: We have previously investigated the fate of administered bicarbonate infused as a hypertonic solution in animals with each of the 4 chronic acid-base disorders. Those studies did not address the fate of sodium, the coadministered cation.

METHODS: We examined baseline total body water (TBW), Na+ space, HCO3- space, and urinary sodium and bicarbonate excretion after acute hypertonic NaHCO3 infusion (1-N solution, 5 mmol/kg body weight) in dogs with each of the 4 chronic acid-base disorders. Observations were made at 30, 60, and 90 min postinfusion. Retained sodium that remains osmotically active distributes in an apparent space that approximates TBW. Na+ space that exceeds TBW uncovers nonosmotic sodium storage.

RESULTS: Na+ space approximated TBW at all times in normal and hyperbicarbonatemic animals (metabolic alkalosis and respiratory acidosis), but exceeded TBW by ~30% in hypobicarbonatemic animals (metabolic acidosis and respiratory alkalosis). Such osmotic inactivation was detected at 30 min and remained stable. The pooled data revealed that Na+ space corrected for TBW was independent of the initial blood pH but correlated with initial extracellular bicarbonate concentration ($y = -0.01x + 1.4$, $p= 0.002$). The fate of administered sodium and bicarbonate (internal distribution and urinary excretion) was closely linked.

CONCLUSIONS: This study demonstrates that hypobicarbonatemic animals have a Na+ space that exceeds TBW after an acute infusion of hypertonic NaHCO3 indicating osmotic inactivation of a fraction of retained sodium. In addition to an expanded Na+ space, these animals have a larger HCO3- space compared with hyperbicarbonatemic animals. Both phenomena appear to reflect the wider range of titration of nonbicarbonate buffers (Δ pH) occurring during NaHCO3- loading whenever initial e is low. The data indicate that the fate of administered bicarbonate drives the internal distribution and the external disposal of sodium, the co-administered cation, and is responsible for the early, but non-progressive, osmotic inactivation of a fraction of the retained sodium.

Reference:

Adrogué, H.J., Awan, A.A. and Madias, N.E. (2020) Sodium Fate after Sodium Bicarbonate Infusion: Influence of Altered Acid-Base Status. American Journal of Nephrology. 51(3), p.182-191. doi: 10.1159/000506274.