Should We Add More Salt, or Less Water?

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The most appropriate maintenance solution for children continues to be a furiously debated topic in the pediatric literature(1, 2). In this issue of *Indian Pediatrics*, Singhi, *et al*.(3) report that the administration of 0.18% saline in 5% dextrose was associated with 13 new episodes of hyponatremia (defined as serum sodium ≤ 130 mEq/L), occurring in 11 of their cohort of 38 patients. As serum sodium is determined by the ratio between effective osmoles (sodium and potassium) and total body water, changes in serum sodium may be predicted by calculating a tonicity balance from the net gain or loss of effective osmoles or electrolyte free water (EFW). The administration of EFW in the form of hypotonic saline should thus predictably result in a fall in serum sodium. However, contrary to previous observational studies(4), the EFW intake in those who developed hyponatremia was lower than those whose sodium remained >130 mEq/L, and therefore the magnitude of EFW intake alone could not explain the fall in serum sodium in all of the patients in this study. The authors therefore postulate alternative mechanisms for hyponatremia. A determination of EFW balance using a tonicity calculation that incorporates both sodium and potassium, and a description of intravascular volume requirements prior to maintenance fluid administration, would be important in order to make a more informed conclusion.

Hyponatremia occurs as a result of a positive balance of EFW, either through the administration, and/or the inability to excrete EFW. Antidiuretic hormone (ADH) increases the permeability of the distal renal tubule and collecting duct, resulting in renal concentration of EFW and inappropriately high urinary sodium concentration. Advocates of isotonic fluids therefore argue that maintaining plasma tonicity supercedes the need to maintain nutritional sodium intake during acute illness, at a time when the non-osmotic ADH secretion and impaired EFW excretion predominate(5). Those who argue against isotonic fluids use suggest that hyponatremia is a result of the misuse and over-administration of appropriate hypotonic solutions(6). The traditional calculation for maintenance fluid requirements in hospitalized children has been criticized for overestimating energy expenditure and subsequently free-water requirements, and the total fluid requirements during acute illness or following surgery may approximate only half of that suggested by traditional recommendations (50-60 mL/kg/day)(5). Hence, in susceptible, euvolemic patients, both isotonic or hypotonic maintenance solutions may both result in a net increase in serum sodium if the "desalination" induced by ADH is negated with fluid restriction. It is important to emphasize however, in volume depleted patients, elevated ADH secretion and increased urinary tonicity persists until adequate volume expansion is achieved(7). The significantly lower total fluid intake in hyponatremic patients in comparison to controls may be suggestive of this phenomenon in the present study(3).

Where no relationship is observed between EFW balance and the fall in serum sodium, a translocational hyponatremia with increased osmolar gap, termed the "sick cell syndrome", has been hypothesized in the development early hyponatremia in critically ill adults, while a dilutional mechanism may be more responsible for ongoing hyponatremia(8). It is yet unclear what role this

phenomenon plays in the etiology of hyponatremia in children. If hyponatremia was purely a problem of dilution, then all hypotonic solutions should be abandoned. The study by Singhi, *et al.*(3) provides further evidence of multifactorial mechanisms unique to each patient, and while a fall in serum sodium was not consistently attributable to a positive EFW balance, the risks of significant hyponatremia during hypotonic fluid administration were substantial (36.8% of patients).

There is, therefore, no simple formula or single solution of choice that will guarantee tonicity balance and minimize electrolyte disturbances in children other than vigilance with monitoring and "dose adjusting" our prescriptions according to the patient's response. It is often difficult to assess a child's extracellular volume status, and knowing the serum electrolytes at presentation may not reliably predict how these will evolve with an empiric and generic fluid prescription, particularly in complex and critically ill children. Our current maintenance fluid prescription practice in children is challenging in light of the disturbing paucity of evidence. Until more prospective trials are undertaken, the safest solution is to tailor fluid prescriptions to the individual patient, needs over time, and focus on the inherent properties of the patient's physiology, rather than the inherent properties of the fluid being used.

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