

Misconceptions in the Treatment of Dehydration in Children

In the July issue of *Pediatrics in Review*, Dr Powers discusses the recognition and management of various forms of dehydration: isotonic, hypotonic, and hypertonic. (1) We would like to draw attention to certain aspects of her recommendations that are incorrect and could pose serious complications if followed, that is, the use of hypotonic fluids with the addition of 40 mEq/L (40 mmol/L) of potassium chloride for the treatment of dehydration and the use of isotonic fluids for the treatment of hyponatremic encephalopathy. Our main contentions are:

- Hypotonic fluids are inappropriate for the treatment of volume-depleted patients because their use is associated with a high incidence of hyponatremia and could result in fatal hyponatremic encephalopathy.
- High-concentration potassium solutions risk the development of dangerous hyperkalemia.
- Isotonic saline is inappropriate for the treatment of hyponatremic encephalopathy because it is insufficiently hypertonic to result in a consistent increase in plasma sodium.

The term “dehydration” is frequently employed in children with gastroenteritis who have normal or low plasma sodium concentrations. The term dehydration means the loss of total body water and in the strict sense can only be applied to children who have hypernatremia. The more appropriate terminology is volume depletion, which refers to extracellular fluid loss. (2)(3) We, therefore, use the term volume depletion for children with gastroenteritis and signs of hypovolemia.

Dr Powers' calculations for fluid therapy are based on the principle that replacement fluids should be a combination of the fluid deficit plus maintenance fluid requirement replaced over 24 hours. She incorrectly assumes, though, that the sodium concentration of both the deficit and maintenance fluid is hypotonic and, therefore, her calculations are always for an extremely hypotonic fluid. She derives this by assuming that the fluid deficit in isotonic volume depletion is 60% from the extracellular space and 40% intracellular space, thereby calculating the use of a hypotonic sodium concentration of 84 mEq/L (84 mmol/L) to replace the volume deficit. For the maintenance fluid requirement, she uses a sodium concentration of 30 mEq/L (30 mmol/L), as originally put forth by Holiday and Segar in 1957. (4) Using this approach, her calculations recommend a sodium concentration of 30 mEq/L (30 mmol/L) (0.2% saline) to treat hypertonic volume depletion, 40 mEq/L (40 mmol/L) (0.3% saline) to treat isotonic volume depletion, and 80 mEq/L (80 mmol/L) (0.45% saline) to treat hypotonic volume depletion. There are various problems with this reasoning and there are data to suggest that using hypotonic fluids to treat volume depletion results in a high incidence of hyponatremia and could lead to fatal hyponatremic encephalopathy.

AUTHOR DISCLOSURE Drs Moritz and Ayus have disclosed no financial relationships relevant to this article. This commentary does contain a discussion of an unapproved/investigative use of a commercial product/device.

In isotonic volume depletion, the fluid deficit is 100% from the extracellular space, and when replacing the fluid deficit, the sodium concentration should be similar to 0.9% saline (154 mEq/L [154 mmol/L]). (2)(3) Even though patients with gastroenteritis may have both sodium and potassium losses, in order for potassium to leave the intracellular space, it must exchange with sodium from the extracellular space, with a net effect of a nearly 100% extracellular volume deficit and a sodium concentration that is isotonic with plasma water. Therefore, Dr Powers's calculation significantly underestimates the sodium deficit for correcting volume depletion.

The maintenance requirements of sodium and potassium, as initially proposed by Holiday and Segar, are based on the electrolyte content of human breast and cow milk (4) and are not applicable to the treatment of volume depletion or for maintenance fluids in acutely ill patients. (5) Volume depletion is a potent stimulus for arginine vasopressin (AVP) secretion, and, therefore, volume-depleted patients have an impaired ability to excrete free water. They retain free water if hypotonic fluids are administered and have a fall in plasma sodium concentration. Only isotonic fluids should be administered to volume-depleted patients until the extracellular deficit is replaced and good tissue perfusion with urine output is established. Hypotonic fluids are primarily indicated for the correction of hypernatremia following the correction of volume depletion. In 2003, we proposed the use of isotonic maintenance fluids for children to prevent hospital-acquired hyponatremia, (6) and more than 15 subsequent prospective studies in more than 2000 children have demonstrated that isotonic fluids are superior to hypotonic fluids for the prevention of hyponatremia and are not associated with an increase in the incidence of hypernatremia or fluid overload. (7) Isotonic maintenance fluids do not produce hypernatremia (8) in the absence of a renal concentrating defect or large extrarenal free water losses because a normally functioning kidney can generate free water by excreting hypertonic urine. (9)(10)

It has been previously demonstrated that patients with gastroenteritis have elevated AVP levels (11) and a high incidence of hyponatremia when administered hypotonic fluids. (12) Prospective studies have demonstrated that isotonic fluids are superior to hypotonic fluids in preventing a fall in plasma sodium in patients with gastroenteritis. (13) Deaths have been reported in children with gastroenteritis who received hypotonic fluids, (14)(15) so hypotonic fluids should be avoided.

Patients with hypertonic volume depletion may require a hypotonic fluid to correct hypernatremia, but this should only be used after attaining appropriate volume expansion

with 0.9% saline. (15)(16) The volume deficit is difficult to determine in patients with hypernatremia and gastroenteritis. It is safer to use 0.45% saline to correct the hypernatremia, rather than 0.2% saline, in order to avoid an excessive fall in plasma sodium or inadequate volume expansion. (17)

Dr Powers' calculation for potassium replacement is 8 to 11 mEq/kg per day at a concentration of 40 mEq/L (40 mmol/L). Historic recommendations for potassium replacement in dehydration have advocated no more than 3.5 mEq/kg per day due to concerns for the development of hyperkalemia because of limitations in intracellular potassium transport. (18) (19) Unless hypokalemia is present, there is no pressing reason to administer such large quantities of potassium. A potassium concentration of no more than 20 mEq/L (20 mmol/L) should be safe and sufficient, provided that there is good urine output, normal renal function, and no hyperkalemia present.

Finally, Dr Powers recommends the use of 0.9% saline as an option for the treatment of hyponatremic encephalopathy. We would like to point out that 0.9% saline (154 mEq/L [154 mmol/L]) is not of sufficient tonicity to result in a consistent increase in plasma sodium to treat hyponatremic encephalopathy, which is a medical emergency. Only 3% saline (513 mEq/L [513 mmol/L]) should be used to treat hyponatremic encephalopathy. (20)(21)(22) There is actually a potential risk for a fall in plasma sodium if 0.9% saline is administered in states of AVP excess because natriuresis could develop with an acute lowering in plasma sodium. (23)

In summary, isotonic saline should be used for the treatment of volume depletion, regardless of the plasma sodium concentration. Hypotonic fluids should be avoided because their use could result in acute hyponatremia. Hypotonic fluids may be indicated for the correction of hypernatremia once the volume depletion has been corrected. Potassium should not be administered at a concentration of greater than 20 mEq/L (20 mmol/L) in order to prevent the development of hyperkalemia. Hyponatremic encephalopathy is a medical emergency that should be treated with 3% sodium chloride. Isotonic saline should not be used to treat hyponatremic encephalopathy because it is of insufficient tonicity to consistently increase the plasma sodium and could lead to a fall in plasma sodium if AVP concentrations are persistently elevated.

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