

Carol Rees Parrish, M.S., R.D., Series Editor

# Clinical Observations

## Correcting Hypernatremia: Enteral or Intravenous Hydration?

by Carol Rees Parrish, Mitchell H. Rosner

### BACKGROUND

**H**ypernatremia is defined as a serum sodium value  $> 145$  mEq/L. Since the serum sodium is determined by the ratio of the amount of sodium in the serum to the amount of plasma water, hypernatremia can develop from either an excess of sodium (such as due to the administration of hypertonic fluids), a loss of hypotonic fluids (free water) or a combination of both. Most commonly, it is the loss of hypotonic fluids and the failure to replace these water losses that result in hypernatremia.

In most circumstances, thirst is a powerful defense mechanism against a rise in the serum sodium level. The body defends its serum osmolality closely so that as

the serum sodium rises (and with it serum osmolality), thirst ensues along with rises in arginine vasopressin (AVP) secreted by the posterior pituitary. AVP leads to urinary concentration and conservation of renal water excretion, but ultimately it is thirst and ingestion of water that allows the serum sodium to normalize. Thus, most patients who develop hypernatremia have the common feature that water intake is restricted in some form. For instance, patients in the intensive care unit (ICU) who are intubated and sedated cannot control their water intake and the same is true for patients with impaired mental status or limited mobility. Thus, these patients are at high risk for hypernatremia (Table 1).

### Epidemiology of Hypernatremia

Given the powerful ability of thirst to defend against hypernatremia, it is not surprising that the incidence of this electrolyte disorder in patients presenting to the emergency department (ED) is uncommon (0.2%).<sup>1</sup>

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Most of these outpatients usually have either chronic or acute impairment in their mental status (such as dementia). In critically ill patients, the incidence of hyponatremia is 10-fold higher (2-6%).<sup>2,3</sup> Importantly, a large percentage of patients develop hyponatremia during the course of their hospital stays (especially in the ICU – up to 10%).<sup>4,5</sup> The reasons for this are multi-fold and include:

- Use of hypertonic fluids
- Ongoing loss of body fluid loss (gastric decompression, stool, ostomy, fistulas, biliary drains, etc., inattention to water intake and needs)
- Use of diuretics, lactulose
- Poorly controlled hyperglycemia.<sup>6</sup>

### Consequences and Complications Associated with Hyponatremia

The clinical symptoms associated with hyponatremia are alterations in central nervous system functioning, including a spectrum ranging from mild confusion to stupor and coma. These symptoms likely result from changes in cellular volume as water moves from the intracellular compartment to the more hypertonic extracellular compartment resulting in cell shrinkage.<sup>7</sup>

Mortality rates in patients with hyponatremia, especially those in the ICU, are very high (ranging from 15 to 50%), depending upon the severity of the hyponatremia.<sup>8,9</sup> While hyponatremia has an independent effect on increased mortality, the underlying disease processes driving the development of hyponatremia is more likely to blame with the higher mortality rates.<sup>10</sup>

### Etiology of Hyponatremia

Broadly speaking, the causes of hyponatremia can be divided into three categories:

- water with solute loss (with water losses in excess of solute losses)
- pure water losses
- solute (sodium) gain.<sup>11</sup>

In those cases of water with solute loss and pure water losses, most patients will have impaired mental

status and decreased thirst sensation, or the inability to obtain free water (see Table 2).

### Correction of Hyponatremia

As many patients with hyponatremia will be volume depleted as well as dehydrated, assessing the need for rapid resuscitation is critical, and if needed, intravenous isotonic solutions should be administered until the patient is hemodynamically stable. Before correction of hyponatremia, it is vital to determine if the rise in serum sodium is acute (< 48 hours) or more chronic (> 48 hours). This is because with chronic hyponatremia, brain adaptations in cellular volume have occurred such that rapid correction in these circumstances can result in cerebral edema, increased intracranial pressure and brain stem herniation with death.<sup>12</sup> In chronic states of hyponatremia the serum sodium should not be lowered by more than 8 to 10 mmol/L/24 hours. If it is unclear as to the duration of hyponatremia, it is best to assume that the condition is chronic and use a slower rate of correction.

Several formulas are available to determine the rates of infusion of hypotonic solutions and any of these can be utilized.<sup>7,13,14</sup> However, it is critical that the clinician measure serum sodium levels frequently the duration of correction (every 4-6 hours), so that over-rapid correction is avoided and infusion/replacement rates for water can be adjusted. Water replacement can be achieved in several manners: (1) intravenous hypotonic fluids which may range from 0.45% saline to 5% dextrose in water or (2) enteral water administration.

**Table 1. Patient's at Risk for Hyponatremia**

- Disturbed sensation of thirst
- Unconscious
- Water intake dependent on another clinician/individual
  - Immobile patient
  - Altered mental status
  - Enterally fed patients
  - Critically ill patients

In many cases, it is important to recognize that the free water deficit may be great and along with continuing water losses (insensible losses), the replacement rates can be substantially greater than 1-2 liter per day. When replacement rates are greater than 1 liter in a 24 hour period, administering water replacement only via the gastrointestinal route may prove challenging as the following cases highlight.

### CASE 1

46 year-old female who is post-repair of a congenital heart defect is now requiring mechanical ventilation. A percutaneous gastrostomy (PEG) tube is in place and the patient is tolerating enteral nutrition (EN) with a daily volume of 1400 ml of enteral formula. Over the past few days, she has become hyponatremic and the care team increased her water flushes to 300mL every 4 hours (1800mL per 24 hours) to address her estimated water deficit. Thus, in total, she was receiving 3200 ml of fluid enterally per day. In this case, she became quite uncomfortable and given this concern as well as concerns for gastric distention and risk for aspiration, it was recommended she be given IV replacement of her free water deficit and minimize enteral delivery to only tube feeding and necessary medications until the distension resolved. Two days after the switch to IV free water replacement, her abdominal distention had resolved.

**Table 2. Etiology of Hyponatremia**

- Extra-renal water losses (profuse sweating)
- Renal losses (osmotic diuresis)
- High-protein intake
- Lithium administration
- Vasopressin receptor antagonists
- High urine output that is hypotonic to plasma.
- Lastly, solute gain occurs in situations where hypertonic solutions are given to the patient (for example, sodium bicarbonate during a cardiac resuscitation).

### CASE 2

A 72 year-old male admitted to the intensive care unit (ICU) with sepsis developed severe diarrhea due to *Clostridium difficile* colitis. He is receiving enteral nutrition, but due to ongoing diarrhea and fluid losses he became hyponatremic. The patient was prescribed 400mL of water every 3 hours to replace the water deficit. With this increased fluid delivered enterally, he vomited leading to a change to IV water replacement.

### Practical “Water Replacement”

The GI tract is normally capable of handling a large amount of fluids and solute/nutrient administration. However, in the setting of critical illness or serious GI problems (gastroparesis, etc.), this capacity for water and nutrient adsorption may be compromised and lead to increased abdominal distention, discomfort and the risk for nausea/vomiting and aspiration events. Water given the enteral route vs. the intravenous route is equally as effective; however there may be practical limitations to the use of the GI tract especially when replacement volumes are greater than 1000mL. On the other hand, IV water replacement is safe, reliable, predictable, consistent, and avoids these complications.

### Oral versus IV Hydration

If the clinician is contemplating oral hydration, it is important to assess the ability of the GI tract to accommodate and absorb the additional fluid load. This is especially true if the patient is already receiving EN and the additional volume with hydration may prove intolerable. Certainly, if a patient is having trouble tolerating EN at the outset, then IV hydration should be the route of choice. Other key questions that need to be asked before utilizing enteral hydration include:

1. Will enteral solutions be held for periods of time (such as for procedures, lost access)?
2. Does the patient have impaired GI motility?
3. Is the patient constipated?
4. Is the patient at risk for aspiration that could be worsened by increased GI distention?

Finally, it should be noted that when large and frequent water flushes are ordered (such as 300mL every 4 hours), each time the flush is scheduled to run in, enteral feedings are stopped or automatically shut off (if the patient is on a dual pump), while the water infuses. The larger the flush, the more time it takes to

run in. If the patient has frequent and large flushes, this can translate into significant lost feeding time and the patient's nutritional status is compromised. Furthermore, if the above dual enteral pump is being used and the patient is on a nocturnal, or other cycled delivery mode, when the pump is turned off, no flushes will be infused.

### Suggested Guidelines

1. For serum sodium < 150 mmol/L, it is reasonable to try enteral water replacement up to 1 liter in divided doses (for example, 250mL every 6 hours, or 165mL every 4 hours).
2. For serum sodium > 150 mmol/L, IV hydration should be given carefully, and in a controlled and reliable fashion, using dextrose 5% in water or another hypotonic fluid as appropriate for the individual patient. ■

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