Dehydration: Isonatremic, Hyponatremic, and Hypernatremic Recognition and Management

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Educational Gap

Clinicians need to recognize the signs and symptoms of dehydration to safely restore fluid and electrolytes.

Objectives

After completing this article, readers should be able to:

1. Understand that the signs and symptoms of dehydration are related to changes in extracellular fluid volume.
2. Recognize the different clinical and laboratory abnormalities in isonatremic, hyponatremic, and hypernatremic dehydration.
3. Know how to manage isonatremic dehydration.
4. Know how to manage hyponatremic dehydration.
5. Know how to manage hypernatremic dehydration.
6. Recognize how to avoid as well as treat complications of fluid and sodium repletion.
7. Understand which patients are candidates for oral rehydration.
8. Know the proper fluids and methods for oral rehydration.

INTRODUCTION

Dehydration is one of the leading causes of pediatric morbidity and mortality throughout the world. Diarrheal disease and dehydration account for 14% to 30% of worldwide deaths among infants and toddlers. (1) In the United States, as recently as 2003, gastroenteritis was the source for more than 1.5 million office visits, 200,000 hospitalizations, and 300 deaths per year. The rotavirus vaccine has significantly decreased the incidence of rotaviral gastroenteritis, and now norovirus is the leading cause in the United States.

Water, which is essential for cellular homeostasis, comprises about 75% of body weight in infants and up to 60% in adolescents and adults. Without water intake, humans would die within a few days. (2) The human body has an efficient mechanism of physiologic controls to maintain fluid and electrolyte balance, including thirst. These mechanisms can be overwhelmed in disease states such as gastroenteritis because of rapid fluid and electrolyte losses, leading to dysnatremia, which is the most common electrolyte abnormality in hospitalized patients. (3)

AUTHOR DISCLOSURE Dr Powers has disclosed no financial relationships relevant to this article. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.
Infants and young children are especially vulnerable because they lack the ability to relate their thirst to caregivers or to access fluids on their own. They also have increased insensible losses due to a higher body surface area.

Hypovolemia occurs when fluid is lost from the extracellular space at a rate exceeding replacement. The typical sites for these losses are the gastrointestinal tract (diarrhea and vomiting), the skin (sweat, burns), and urine (glycosuria, diuretic therapy, obstructive uropathies, interstitial disease, neurogenic and nephrogenic diabetes insipidus). The body tries to maintain water and mineral balance by shifting fluid from the intracellular compartment into the extracellular space and promotes urinary retention of water via secretion of antidiuretic hormone (ADH). In response to losses, receptor cells in the hypothalamus shrink, causing the release of a hormonal message to drink and enhance the appetite for salt. If salt and water are not adequately replenished, the effective circulating volume is diminished, compromising organ and tissue perfusion (Fig 1).

**SIGNS OF DEHYDRATION**

Assessing the extent of volume depletion can be difficult. Ideally, the clinician would have a baseline weight for comparison; each gram of weight loss corresponds to one milliliter of water loss. Unfortunately, such baseline weight rarely exists. Therefore, the clinician should use clinical signs and symptoms as well as laboratory data to assess the degree of dehydration. Dehydration is generally classified as mild (3%–5% volume loss), moderate (6%–9% volume loss), or severe (≥10% volume loss) (Table 1).

Infants and children with mild dehydration often have minimal or no clinical changes other than a decrease in urine output. Along with decreased urine output and tearing, children with moderate dehydration often have dried mucous membranes, decreased skin turgor, irritability, tachycardia with decreased capillary refill, and deep respirations. A systematic review of the accuracy of clinically predicting at least 5% dehydration in children found prolonged capillary refill, abnormal skin turgor, and abnormal respiratory pattern to be the best predictors. (4) Children with severe dehydration present in near-shock to shock with lethargy, tachycardia, hypotension, hyperpnea, prolonged capillary refill, and cool and mottled extremities. They require immediate aggressive isotonic fluid resuscitation. Hypotension is a very late sign of dehydration, occurring when all compensatory mechanisms to maintain organ perfusion are overwhelmed.

The clinical assessment of dehydration is only an estimate. Therefore, the child must be continually reevaluated during therapy to ensure that appropriate replacement volumes are being administered. Children with hyponatremic dehydration have hypotonic body fluids with serum osmolarity less than 270 mOsm/kg (270 mmol/kg) that can lead to fluid shifts from the extracellular to the intracellular space. The degree of dehydration may be overestimated because these patients have diminished intravascular volume that is manifested by more severe clinical symptoms. They are very likely to require immediate circulatory support. On the other hand, children with hypernatremic dehydration have hypertonic body fluids with serum osmolarity, often in excess of 300 mOsm/kg (300 mmol/kg). Fluid shifts from the intracellular to the extracellular space to maintain intravascular volume. The degree of dehydration in these children is often underestimated, contributing to late presentation for medical care.

**LABORATORY TESTS**

Results of laboratory tests, including measurements of serum electrolytes and acid/base balance, are typically normal in infants and children with mild dehydration. Therefore, laboratory testing is generally indicated only for children requiring intravenous fluid repletion, typically with greater than 10% dehydration. Assessment of serum bicarbonate is one of the most sensitive tests to help determine the degree of dehydration. A value of less than 17 mEq/L (17 mmol/L) on presentation to the emergency department was shown in one study to differentiate moderate-to-severe dehydration from mild dehydration. (5) Although the blood urea nitrogen rises with increasing severity of dehydration, it also can be increased by other factors, such as excessive protein catabolism, increased protein in the diet, and gastrointestinal bleeding. Accordingly, this value may not be clinically relevant. It is important to measure the serum sodium in moderate-to-severe dehydration because it determines the type and speed of repletion.

Potassium values can be low or high. Typically, potassium measurements are low because of losses in the stool. However, with worsening degrees of hypovolemia and an increase in metabolic acidosis, they can be elevated following a net shift from the intracellular to the extracellular space. The values generally normalize and even become low with the correction of acidosis. Potassium concentrations should be followed and the mineral replenished to avoid cardiac arrhythmias as well as a functional ileus.

Children who are dehydrated often present with metabolic acidosis. This is typical in those who have gastroenteritis and bicarbonate losses in the stool. In more severe
cases, lactic acidosis can develop from poor tissue perfusion and ketosis. If renal perfusion is decreased, acid excretion by the kidneys can be compromised. Metabolic alkalosis can develop in children with significant losses from vomiting due to hydrochloric acid losses.

In response to hypovolemia, the kidneys conserve water and sodium. Urine sodium concentrations are low, generally less than 20 mEq/L (20 mmol/L). Urine osmolality and specific gravity are typically elevated. Urine osmolality is often greater than 400 mOsm/kg (400 mmol/kg) in the absence of diuretics, diabetes insipidus, or an osmotic diuresis. A specific gravity of greater than 1.015 is suggestive of concentrated urine, but this is a less accurate predictor because it depends on the number of solute particles in the urine. Because most dehydrated patients have elevated creatinine, calculating the fractional excretion of sodium (\( \text{FENa} \)) can help determine the source of the elevated level:

\[
\text{FENa} = \frac{(\text{Urinary sodium} \times \text{Plasma creatinine})}{(\text{Urinary creatinine} \times \text{Plasma sodium})} \times 100
\]

An \( \text{FENa} \) of less than 1% suggests a prerenal or hypovolemic state that should respond to volume replacement. (6)

**TYPE OF DEHYDRATION**

In dehydration, serum sodium values vary, depending on the relative loss of solute to water. Isonatremic dehydration is defined by sodium of 130 to 150 mEq/L (130 to 150 mmol/L). This reflects an equal proportion of solute and water loss. Isonatremic dehydration typically occurs in patients with...
secretory diarrhea where the solute concentration of the
diarrhea is the same as the plasma solute concentration.

Hyponatremic dehydration with a sodium concentration of
less than 130 mEq/L (130 mmol/L) occurs when diarrheal
losses are replaced with hypotonic fluids. With solute and
water loss, ADH is secreted, triggering the body to enhance
water absorption. As the patient drinks fluids that are rela-
tively hypotonic to the stool losses, the serum sodium con-
centration falls. Hypernatremic dehydration, with serum
sodium greater than 150 mEq/L (150 mmol/L), re-
fl

tects water

loss in excess of solute loss. This is common with viral
gastroenteritis, such as that caused by rotavirus, and in neo-

nates with inadequate breastfeeding in whom diarrheal and
insensible water losses are inadequately replaced.

GENERAL PRINCIPLES OF TREATMENT

The goal of therapy is to recognize the degree and type of
dehydration and to restore any water and electrolyte deficits
while meeting maintenance needs and replacing ongoing
losses. The degree of dehydration is clinically determined
from a change in weight or estimated from signs and symptoms,
as described previously. In moderate or severe dehydration,
a serum sodium value can help to determine the appropriate
fluids to use and the suitable time course of replacement. In
developed countries, this is generally achieved with intravenous
fluids, but oral rehydration for mild-to-moderate isonatremic
dehydration can be successful.

On presentation, the clinician needs to determine if the
child has signs and symptoms of intravascular compromise
that necessitate emergent intravenous therapy. If so, vascular
access must be secured. Fluids can be administered effec-
tively via an intraosseous route in hemodynamically unstable
children in whom peripheral access cannot immediately be
obtained. (7)

REPLACING ONGOING FLUID AND ELECTROLYTE
LOSSES

Most children presenting with dehydration due to diarrhea
or emesis have ongoing losses until the gastroenteritis
resolves. Therefore, in addition to providing fluids and
electrolytes to meet maintenance and deficit needs, ongoing
losses must be replaced to achieve normovolemia. The
ongoing losses generally should be replaced milliliter-for-
milliliter with fluids that have the same electrolyte compo-
sition. Losses from emesis or nasogastric drainage typically
are replaced with 0.45% normal saline (NS) plus 10 to
15 mEq/L of potassium chloride (KCl). Diarrheal losses also
contain bicarbonate, and replacement may be beneficial for
severe acidosis. (8)

CALCULATING FLUID AND ELECTROLYTE LOSSES

The fluid deficit can be determined either from a change in
baseline weight or estimated from the clinical signs and
symptoms. The deficit volume should be replaced in addition
to the patient's maintenance fluid and electrolyte require-
ments and ongoing fluid and electrolyte losses. Approximately 60% of acute fluid and electrolyte losses come from

**TABLE 1. Clinical Signs and Symptoms of Dehydration***

<table>
<thead>
<tr>
<th>CLINICAL SIGNS</th>
<th>MILD (3%–5%)</th>
<th>MODERATE (6%–9%)</th>
<th>SEVERE (≥10%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic Signs</td>
<td>Increased thirst</td>
<td>Irritable</td>
<td>Lethargic</td>
</tr>
<tr>
<td>Urine Output</td>
<td>Decreased</td>
<td>Decreased (&lt;1 mL/kg/hr)</td>
<td>Decreased (oliguria/anuria)</td>
</tr>
<tr>
<td>Mucous Membranes</td>
<td>Tacky</td>
<td>Dry</td>
<td>Parched</td>
</tr>
<tr>
<td>Skin Turgor</td>
<td>Normal</td>
<td>Reduced</td>
<td>Tenting</td>
</tr>
<tr>
<td>Capillary Refill</td>
<td>Normal</td>
<td>Mildly delayed</td>
<td>Markedly delayed</td>
</tr>
<tr>
<td>Skin Temperature</td>
<td>Normal</td>
<td>Cool</td>
<td>Cool, mottled</td>
</tr>
<tr>
<td>Anterior Fontanelle</td>
<td>Normal</td>
<td>Sunken</td>
<td>Markedly sunken</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>Normal</td>
<td>Increased</td>
<td>Markedly increased or ominously low</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Normal</td>
<td>Normal to low</td>
<td>Low</td>
</tr>
<tr>
<td>Respiration†</td>
<td>Normal</td>
<td>Deep, may be increased</td>
<td>Deep and increased or decreased to absent</td>
</tr>
</tbody>
</table>

*These findings for isonatremic dehydration overestimate the degree of dehydration with hyponatremia and underestimate the degree of dehydration with hypernatremia.

†Best predictors of dehydration.
the extracellular space, with about 40% of fluid and electrolyte shifts coming from the intracellular space. Sodium in the extracellular space (content of about 140 mEq/L [140 mmol/L]) and potassium in the intracellular space (content of 150 mEq/L [150 mmol/L]) are the major electrolyte components that are lost. For every 100 mL of water lost, 8.4 mEq (8.4 mmol) of potassium [(150 mEq/L [150 mmol/L] × 0.60) ÷ 10] and 6 mEq (6 mmol) of potassium [(150 mEq/L [150 mmol/L] × 0.40) ÷ 10] are lost. Hyperacute losses over a few hours are nearly 100% from the extracellular space because not enough time has elapsed to allow for fluid shifts. For chronic losses over weeks, such as with pyloric stenosis, fluid losses are about 50% from the intracellular space, with relatively more potassium losses.

ISONATREMIC DEHYDRATION

Isonatremic dehydration occurs as a result of equal solute and water losses, thus maintaining a normal sodium concentration of 130 to 150 mEq/L (130 to 150 mmol/L). This is the most common presentation of dehydration and has the best prognosis. In general, oral rehydration can safely and effectively restore intravascular volume in children with mild-to-moderate isonatremic dehydration. Oral rehydration is commonly used for children treated at home but is often underused in the hospital or emergency department. Even with ongoing diarrhea, water can be absorbed across the intestinal lumen by the cotransport of sodium and glucose via the SGLT1 protein and by active transport via the sodium-potassium ATPase pump. This discovery, which led to the development of oral rehydration solutions by the World Health Organization (WHO), has been described as “potentially the most important medical advancements this century.” (9) The use of these solutions in the early 1970s decreased the mortality rate associated with cholera from 30% to 50% to less than 3%. (10) A systematic review and meta-analysis found no clinical difference in rehydration recommendations have a lower sodium and osmolarity, decreasing the frequency of stools and the incidence of hypernatremia. In developed nations, where secretory diarrhea from cholera is rare, oral rehydration solutions have lower sodium contents while maintaining a low osmolarity (Table 2). Commonly used beverages, such as apple juice, tea, ginger ale, colas, and chicken broth, are inappropriate to use for rehydration because they do not contain the correct sodium and glucose ratio to promote salt and water reabsorption across the intestinal lumen. Sports drinks, designed to maintain adequate hydration from fluid and electrolyte losses caused by sweating with prolonged exercise, also do not have the appropriate glucose and sodium ratio to be used as rehydration fluids, especially because losses are from the gastrointestinal tract.

General Principles of Oral Rehydration

Regular feedings and nutrition should be continued to meet the infant’s or child’s maintenance needs. For ongoing losses, replace one milliliter of fluid for every gram of output, stool, emesis, or urine. In the hospital, diapers can be weighed. If measurements are not available, then the guidelines of replacing 10 mL/kg body weight for each watery stool or 2 mL/kg body weight for each episode of emesis can be used (Table 3).

Oral Rehydration for Mild and Moderate Isonatremic Dehydration

The amount of fluid deficit should be calculated based on change in weight or clinical signs. This typically calculates as 50 to 100 mL/kg body weight replaced over 2 to 4 hours. Using a teaspoon, syringe, or dropper, 5 mL should be administered every few minutes, with the volume increased as tolerated (Table 4). Nasogastric tubes can be used to administer continuous volume replacement in patients with severe vomiting or oral ulcers. Of note, only about 4% of patients fail oral rehydration therapy and require intravenous repletion. (13) Such children usually have a paralytic ileus or intractable vomiting. Ondansetron administration to children with severe vomiting can reduce the need for intravenous therapy and hospital admission. (14) Oral rehydration is contraindicated for infants and children who have circulatory instability or shock, altered mental status, intractable vomiting, bloody
diarrhea or ileus, abnormal serum sodium values, or glucose malabsorption.

**Intravenous Rehydration for Moderate or Severe Isonatremic Dehydration**

Maintenance fluid and electrolyte needs should be based on the child’s normovolemic weight, either obtained from a prior weight or calculated based on estimated percentage of dehydration. Children who are clinically unstable should receive repeated single fluid bolus(es) of 20 mL/kg with 0.9% NS to attain adequate tissue perfusion. More judicious volumes may be considered in children with congestive heart failure or cerebral edema. Lactated Ringer solution should not be used routinely because it is relatively hypotonic (130 mEq/L [130 mmol/L]) of sodium and could adversely lower the patient’s serum sodium. In addition, it contains 4 mEq/L (4 mmol/L) of potassium that could contribute to hyperkalemia. Finally, children with significant emesis may have a contraction alkalosis (increase in blood pH) as a result of fluid losses that could be worsened by the lactate content of the fluid being converted to bicarbonate. Many recommend initial replacement of 50% of deficit fluids over 8 hours followed by replacement of the remaining 50% deficit over the subsequent 16 hours. However, this can be clinically impractical and may be associated with an increased risk of too-rapid replacement if the fluid rate is not adjusted at the correct time. If the child received adequate initial resuscitation, replacing the total deficit over 24 hours is generally acceptable.

Ideally, maintenance and deficit fluid and electrolytes should be combined into one solution that is infused over 24 hours. Regardless of the percentage of dehydration, this calculates as 5% dextrose (D5) 1/3 NS + 40 mEq/L (40 mmol/L) of KCl (Fig 2). However, because this is not a standard fluid available in most hospitals, D5 ½ NS + 40 mEq/L (40 mmol/L) KCl can be safely substituted. If there are concerns about renal insufficiency, potassium should not be added to the fluids until the patient has

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**TABLE 2. Composition of Oral Rehydration Solutions and Commonly Used Beverages**

<table>
<thead>
<tr>
<th>SOLUTION/BEVERAGE</th>
<th>CARBOHYDRATE (g/L)</th>
<th>SODIUM (mEq/L [mmol/L])</th>
<th>POTASSIUM (mEq/L [mmol/L])</th>
<th>BASE (mEq/L [mmol/L])</th>
<th>OSMOLARITY (mOsm/kg [mmol/kg])</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pedialyte®</td>
<td>Dextrose</td>
<td>25</td>
<td>45</td>
<td>20</td>
<td>30</td>
</tr>
<tr>
<td>Enfalyte®</td>
<td>Corn Syrup</td>
<td>30</td>
<td>50</td>
<td>25</td>
<td>30</td>
</tr>
<tr>
<td>CeraLyte®</td>
<td>Rice</td>
<td>40</td>
<td>70</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>World Health Organization (2002)</td>
<td>Glucose</td>
<td>13.5</td>
<td>75</td>
<td>20</td>
<td>30</td>
</tr>
</tbody>
</table>

**Not Appropriate for Rehydration**

- Gatorade®
  - Carbohydrate: 45
  - Sodium: 20
  - Potassium: 3
  - Base: 3
  - Osmolarity: 280–360

- Powerade®
  - Carbohydrate: 58
  - Sodium: 10
  - Potassium: 3
  - Base: 1
  - Osmolarity: 403

- Apple Juice
  - Carbohydrate: 100–150
  - Sodium: 3
  - Potassium: 20
  - Base: 0
  - Osmolarity: 700

- Tea
  - Carbohydrate: 0
  - Sodium: 0
  - Potassium: 0
  - Base: 0
  - Osmolarity: 5

- Ginger Ale
  - Carbohydrate: 90
  - Sodium: 3.5
  - Potassium: 0.1
  - Base: 3.6
  - Osmolarity: 565

- Cola
  - Carbohydrate: 100–150
  - Sodium: 2
  - Potassium: 0.1
  - Base: 13
  - Osmolarity: 550

- Chicken Broth
  - Carbohydrate: 0
  - Sodium: 250
  - Potassium: 5
  - Base: 0
  - Osmolarity: 450

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**TABLE 3. Oral Replacement of Ongoing Losses**

<table>
<thead>
<tr>
<th>LOOSE STOOL REPLACEMENT</th>
<th>EMESIS REPLACEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>mL/mL if measured</td>
<td>mL/mL if measured</td>
</tr>
<tr>
<td>Or about 10 mL/kg per stool</td>
<td>Or about 2 mL/kg per episode</td>
</tr>
</tbody>
</table>

Vol. 36 No. 7  JULY 2015  279
voided. Ongoing fluid and electrolyte losses also should be replaced. Gastric losses should be replaced with 0.45% NS plus 10 to 15 mEq/L (10 to 15 mmol/L) of KCl. For diarrheal losses, bicarbonate substitution for chloride should be considered. (15)

**HYPONATREMIC DEHYDRATION**

Hyponatremic dehydration most typically occurs in older infants and children with gastrointestinal infections. These children are often given fluids with low sodium content such as water, juice, ginger ale, sodas, or tea. In addition, ADH is often released, further diluting the intravascular solute with the reabsorption of water. (17) As serum osmolality falls, fluid is shifted from the extracellular to the intracellular space, causing earlier and more severe intravascular compromise. Affected children are most likely to require immediate volume resuscitation. Normal saline should be rapidly infused in 20-mL/kg aliquots to restore intravascular volume. Lactated Ringer solution should be avoided because its lower sodium content may worsen the hyponatremia and the potassium content may contribute to hyperkalemia.

Cerebral salt wasting can lead to hyponatremic dehydration. This poorly understood, rare condition occurs in patients with central nervous system disorders, most commonly associated with intracranial surgery, meningoencephalitis, and head injury. (18) It typically occurs in the first 10 days of the illness or injury and resolves in 3 to 4 weeks. Cerebral salt wasting is characterized by hyponatremia and intravascular...

<table>
<thead>
<tr>
<th>WEIGHT (kg)</th>
<th>MILD DEHYDRATION (3%–5%)</th>
<th>MODERATE DEHYDRATION (6%–9%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TOTAL VOLUME OVER 4 HOURS</td>
<td>VOLUME PER ADMINISTRATION</td>
</tr>
<tr>
<td>5</td>
<td>150–250 mL</td>
<td>5 mL every 5–8 min</td>
</tr>
<tr>
<td>10</td>
<td>300–500 mL</td>
<td>6–10 mL every 5 min</td>
</tr>
<tr>
<td>15</td>
<td>450–750 mL</td>
<td>10–15 mL every 5 min</td>
</tr>
<tr>
<td>20</td>
<td>600–1,000 mL</td>
<td>12–20 mL every 5 min</td>
</tr>
<tr>
<td>25</td>
<td>750–1,250 mL</td>
<td>15–25 mL every 5 min</td>
</tr>
<tr>
<td>30</td>
<td>900–1,500 mL</td>
<td>18–30 mL every 5 min</td>
</tr>
<tr>
<td>40</td>
<td>1,200–2,000 mL</td>
<td>25–40 mL every 5 min</td>
</tr>
</tbody>
</table>

A 10-month-old infant presents to the emergency department with a 4-day history of frequent watery stools. He is now refusing to drink. He is listless in his mother’s arms. On physical examination, his mucous membranes are dry and the skin on his abdomen is tenting. His heart rate is 160 beats/min and blood pressure is 80/40 mm Hg. His current weight is 9 kg. One week ago, when he was seen in clinic for a routine examination, he weighed 10 kg. His serum sodium measures 138 mEq/dL (138 mmol/L). After failing a trial of oral therapy, intravenous access is obtained and he is given 20 mL/kg (200 mL) of normal saline. Following the infusion, his heart rate, perfusion, and mental status improve.

The sample calculation of intravenous rehydration for isonatremic dehydration.

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Figure 2. Sample calculation of intravenous rehydration for isonatremic dehydration.
fluid depletion related to inappropriate renal sodium wasting. Affected children can have very high-volume urine outputs containing up to 300 mEq/L (300 mmol/L) of sodium and often require replacement with hypertonic saline solutions. Administration of salt tablets and the mineralocorticoid fludrocortisone has been used to help abate sodium and fluid losses.

Intravenous Rehydration for Hyponatremic Dehydration

Oral rehydration solutions are inappropriate to treat hyponatremic dehydration. As with other forms of dehydration, the degree of dehydration should be estimated from a change in weight or from clinical signs. As noted before, the degree of dehydration may be overestimated in hyponatremic dehydration due to the osmotic shift of fluid out of the intravascular space into the tissues. Affected infants and children are most likely to need fluid boluses of 20 mL/kg of 0.9% NS administered rapidly and repeated as needed to improve tissue perfusion. In addition to fluid deficits, sodium and potassium deficits should be calculated. However, sodium losses generally exceed the usual 8 mEq/L (8 mmol/L) of sodium loss per 100 mL of water loss. Additional sodium deficit should be calculated using the formula:

\[
\text{mEq Na deficit} = (\text{desired Na} - \text{measured Na}) \times 0.6(\text{volume of distribution of Na}) \times \text{weight in kg}
\]

Using the child’s baseline weight, maintenance and deficit fluid and electrolytes are calculated and generally replaced over 24 hours. Regardless of the presenting serum sodium value, when combining maintenance and deficit water and electrolytes into one solution, this generally calculates to be 25 ½ NS + 40 mEq/L (40 mmol/L) KCl (Fig 3).

The serum sodium should not rise more than 12 to 15 mEq/L (12 to 15 mmol/L) over the 24-hour period, so frequent monitoring is recommended, generally every 4 to 6 hours. Very rarely, precipitous correction of the sodium can result in central pontine myelinolysis (19).

Occasionally, infants and children can present with seizures related to a rapid drop in the serum sodium concentration. This rapid decrease, generally to less than 120 mEq/L (120 mmol/L), overpowers the cerebral osmoregulatory mechanisms, resulting in cerebral edema. The seizures can be difficult to abate without partial correction of the serum sodium, usually to 120 mEq/L (120 mmol/L). Using the previously cited formula to calculate the sodium replacement, either 0.9% NS or hypertonic 3% saline is given. The choice of solution is generally determined by the volume of saline correction. The correction with 0.9% NS, containing 154 mEq/L (154 mmol/L) of sodium, usually equates to about 30 to 40 mL/kg, which generally can be well tolerated in a dehydrated patient. Administration of hypertonic saline, with a sodium content of 513 mEq/L (513 mmol/L) (~0.5 mEq/mL [0.5 mmol/mL]), requires approximately one-third of the volume of isotonic saline. However, hypertonic saline may necessitate central access because peripheral administration can be painful and lead to cutaneous tissue necrosis with any extravasation.

HYPERNATREMIC DEHYDRATION

Hypernatremic dehydration is defined as serum sodium greater than 150 mEq/L (150 mmol/L). Despite elevated
sodium concentrations, the child actually has total body sodium deficiency, but the water loss exceeds the sodium loss. Hypernatremic dehydration is most commonly seen in young infants receiving inadequate water replacement, typically associated with diarrheal illnesses or poor breastfeeding.

Because the intravascular contents are hypertonic, fluid shifts from the cells into the intravascular space. Thus, the children may be less hemodynamically compromised, resulting in underestimation of the degree of dehydration. In general, an additional 3% to 5% degree of dehydration should be added to the clinical estimate from Table 1. Because intravascular volume is relatively preserved, affected infants often present late for medical care. They are usually somnolent but become hyperirritable when stimulated, often with a high-pitched cry. Their skin feels “doughy” or “velvety.” The major concern is cerebral cellular dehydration in the presence of hypertonicity. Resulting brain shrinkage can cause rupture of bridging veins, leading to subdural, subarachnoid, and intraparenchymal hemorrhage. In addition, thrombosis of the small veins or dural sinuses can occur. (20) Mortality can be high, ranging from 3% to 20%. Up to 40% to 50% of infants can have neurologic sequelae, and in 5% to 10%, the sequelae are severe. Infants with presenting serum sodium values of 150 to 160 mEq/L (150 to 160 mmol/L) and with sodium correction of 0.5 mEq/L (0.5 mmol/L) per hour or less over 48 hours fare the best. Infants with presenting serum sodium values greater than 160 mEq/L (160 mmol/L) and sodium correction greater than 0.5 mEq/L (0.5 mmol/L) per hour over 48 hours have significantly higher morbidity and mortality. (21)

Therefore, these infants need to be monitored intensively and receive meticulous care while slowly correcting the serum sodium and fluid deficit over 48 hours. As with other forms of dehydration, fluid bolus(es) of 20 mL/kg with 0.9% sodium chloride should be administered rapidly if there are any signs of vascular compromise. Water and electrolyte maintenance and deficit needs should be calculated as before. Additional free water deficit can be calculated by:

\[
\text{Na(actual)} - \text{Na(desired)} \times \frac{1000 \text{ mL/L}}{\text{Na(actual)}}
\]

Alternatively, 4 mL/kg of free water can be administered for every milliequivalent (millimole) of sodium greater than 145 mEq/L (145 mmol/L) or 3 mL/kg of free water administered for every milliequivalent (millimole) of sodium greater than 170 mEq/L (170 mmol/L).

The calculation ordinarily equates to 0.2% NS. Potassium should be added once the infant is voiding and is clearly without intrinsic renal disease. Thus, D5 or D10 0.2% NS + 20 to 40 mEq/L (20 to 40 mmol/L) KCl is usually appropriate for replacement over 48 hours (Fig 4). Frequent monitoring, generally every 4 to 6 hours, for the change in serum sodium is paramount to a good clinical outcome. Overall, the rate of fluid replacement should be adjusted rather than the composition of the fluid to ensure the appropriate rate of correction because brain cells generate idiosyncratic osmols in response to hyperosmolality to maintain intracellular toxicity and size. These substances are not diffusible or transportable out of the brain cells. Therefore,
too rapid correction of the sodium can result in too much water acutely entering the cells, causing cerebral edema and seizures. If seizures do occur, the serum sodium should be acutely increased. An infusion with 3% saline can raise the serum sodium most efficiently while providing the least amount of free water. In general, 1 mL/kg of 3% saline increases the serum sodium concentration by about 1 mEq/L (1 mmol/L). Most seizures abate following administration of 4 mL/kg of 3% saline. (15)

Infants and children with diabetes insipidus can also develop hypernatremic dehydration. Central diabetes insipidus is caused by a lack of ADH related to damage to the hypothalamus or pituitary gland. Nephrogenic diabetes insipidus results from ADH unresponsiveness of the kidney. The most common causes of central diabetes insipidus are idiopathic, possibly due to autoimmune injury to the ADH-producing cells, brain tumors, pituitary surgery, or brain trauma. There are rare familial cases. With the loss of ADH, the child is unable to reabsorb water, consequently voiding large amounts of unconcentrated urine. The resultant water loss leads to hyperosmolarity and hypernatremia. Treatment is exogenous vasopressin and replacement of the free water losses.

CONCLUSION

Dehydration is common in infants and children, especially following gastrointestinal illnesses. Oral rehydration can be safely and effectively accomplished in children with mild-to-moderate dehydration and normal serum sodium values. Children with more severe dehydration or with abnormal serum sodium values should be treated with intravenous infusions. It is important for the clinician to understand how to determine the correct fluid and electrolyte solutions to meet the child’s maintenance, deficit, and ongoing losses. In addition, the clinician must recognize how to monitor patients safely while controlling the rate of rehydration.

Summary

- Maintenance, deficit, and ongoing fluid and electrolyte losses need to be calculated.
- Based on strong research evidence, mild-to-moderate isonatremic dehydration can be treated effectively with oral rehydration solutions. (10)
- Based on expert opinion, children with moderate-to-severe dehydration should have electrolytes measured to determine content and rate of fluid replacement.
- Based on expert consensus opinion, children with altered perfusion should receive immediate fluid bolus(es) with normal saline.
- Based on expert opinion and reasoning from first principles, in children with moderate-to-severe isonatremic dehydration, maintenance plus deficit fluid and electrolyte needs generally calculate to be 5% dextrose (D5) 1/3 normal saline (NS) + 40 mEq/L (40 mmol/L) potassium chloride (KCl). Because this is not a readily available fluid, D5½ NS + 40 mEq/L (40 mmol/L) KCl can generally be safely substituted. Maintenance plus deficit volumes can be infused over 24 hours.
- Based on expert opinion and reasoning from first principles, children with moderate-to-severe hyponatremic dehydration are most likely to need immediate circulatory support. Fluid and electrolyte maintenance and deficit needs usually calculate to be D5½ NS + 40 mEq/L (40 mmol/L) KCl. Maintenance plus deficit volumes can be infused over 24 hours, with goal correction of sodium not to exceed 12 to 15 mEq/L (12 to 15 mmol/L) over the 24 hours.
- Infants with moderate-to-severe hypernatremic dehydration are at highest risk for morbidity and mortality, including risk for cerebral hemorrhage, thrombus, or edema. Their intravascular volume is generally spared. Based on expert opinion and reasoning from first principles, fluid and electrolyte maintenance and deficit needs usually calculate to be D5¾ NS + 20 to 40 mEq/L (20 to 40 mmol/L) KCl. Deficit replacement should occur over 48 hours, with goal correction of sodium not to exceed 0.5 mEq/L (0.5 mmol/L) per hour. (15)

References for this article are at http://pedsinreview.aappublications.org/content/36/8/274.full.
1. You are on call on the pediatric ward one weekend and a group of medical students is discussing pediatric dehydration. They are confused about the pathophysiology on a cellular level. Which of the following statements regarding dehydration is correct?
   A. Children have hypertonic body fluids and are very likely to require immediate circulatory support.
   B. Children have hypertonic body fluids, resulting in fluid shifts from the extracellular to the intracellular space.
   C. Children have hyponatremic body fluids, resulting in fluid shifts from the extracellular to the intracellular space.
   D. Children typically do not require circulatory support as fluid shifts to the intracellular space.
   E. Serum osmolality in children typically is 300 to 330 mOsm.

2. An 8-month-old infant is brought to the emergency department because he has been vomiting for 36 hours. He has had 1-oz of formula in the previous 12 hours. You estimate that he is 11% dehydrated. As you order intravenous fluid repletion, you consider which laboratory tests to order. Which of the following statements regarding laboratory values is correct?
   A. Metabolic acidosis ensues in a child with persistent vomiting, requiring immediate therapy.
   B. Serum bicarbonate values less than 20 mEq/L (20 mmol/L) measured in children in the emergency department successfully differentiated mild from moderate dehydration.
   C. Blood urea nitrogen is increased only in dehydration, making it the only clinically relevant laboratory test for dehydration.
   D. The serum bicarbonate is very sensitive in determining the degree of dehydration.
   E. With worsening degrees of dehydration and acidosis, potassium levels become dangerously low.

3. You are attending in the emergency department when a 4-month-old infant presents with emesis for the past 36 hours. Her weight is 4.5 kg. She is sleeping in her car seat and difficult to arouse. She has had no urine output in the previous 12 hours. Her blood pressure is 68/35 mm Hg and heart rate is 166 beats/min. She appears to be approximately 10% dehydrated. Her serum sodium measures 140 mEq/L (140 mmol/L). You diagnose isonatremic dehydration and administer a 20-mL/kg (100 mL) infusion of normal saline. Which of the following are the correct calculations for this child’s maintenance, deficit, and total fluid requirements to be administered within 24 hours?

   - MAINTENANCE
   - DEFICIT
   - TOTAL

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4. A 6-month-old infant presents to your office with diarrhea for 1 week. He weighs 8 kg. He initially maintained an appetite but has not had intake over the past 24 hours. He is lethargic and sleepy. His blood pressure is 60/40 mm Hg and his heart rate is 140 beats/min. He has delayed capillary refill. His serum sodium is 122 mEq/L (122 mmol/L). You place an intravenous catheter. You desire a sodium concentration of 130 mEq/L (130 mmol/L). What is the child’s calculated sodium deficit?
A. 3.8 mEq (3.8 mmol).
B. 4.8 mEq (4.8 mmol).
C. 38 mEq (38 mmol).
D. 46 mEq (46 mmol).
E. 64 mEq (64 mmol).

5. A 3-month-old infant presents to your office with watery stools for the past 5 days. He has little energy but has a shrill, high-pitched cry when stimulated. He has had little liquid intake or urine output in the past day. He appears to be 10% dehydrated. Which of the following statements regarding hypernatremic dehydration is correct?

A. Central nervous system morbidity can be high (40%–50%), resulting in intracranial hemorrhage and sinus thrombosis.
B. Hypernatremic dehydration is defined as serum sodium greater than 145 mEq/L (145 mmol/L).
C. Infants whose serum sodium is 150 to 160 mEq/L (150 to 160 mmol/L) should have their sodium corrected at less than 0.5 mEq/L (0.5 mmol/L) per hour over 24 hours.
D. Infants with hypernatremic dehydration may be less hemodynamically compromised, causing overestimation of the degree of dehydration.
E. In hypernatremic dehydration, intravascular volume is not preserved, which results in elevated serum sodium.

Parent Resources from the AAP at HealthyChildren.org

- https://www.healthychildren.org/English/health-issues/injuries-emergencies/Pages/Dehydration.aspx
- Spanish: https://www.healthychildren.org/Spanish/health-issues/injuries-emergencies/Paginas/Dehydration.aspx
Dehydration: Isonatremic, Hyponatremic, and Hypernatremic Recognition and Management
Karen S. Powers
*Pediatrics in Review* 2015;36;274
DOI: 10.1542/pir.36-7-274

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of differentiation that cannot be further classified based on histologic features. Malignant undifferentiated tumors exhibit unpredictable clinical behavior. These may be present at birth or develop over time. Size may remain stable or tumors may undergo rapid proliferation beyond the period of growth anticipated for IHs, which may serve as a diagnostic clue. Atypical clinical characteristics or growth patterns of a presumed IH may warrant further investigation.

Similar cases of soft-tissue tumors mimicking IHs have been reported. In 2006, three cases of congenital vascular-appearing tumors diagnosed as ulcerated IH and treated with systemic corticosteroids were described. After reevaluation of unusual characteristics, including congenital presence and ulceration present at birth, tissue was obtained for pathology, leading to the diagnosis of a congenital infantile fibrosarcoma in each instance. A recently reported case of congenital infantile fibrosarcoma of the lip that underwent rapid proliferation was treated as a presumed IH. Because of the refractory response to therapy, biopsy was performed, confirming the diagnosis. To our knowledge, no case has been reported describing a malignant undifferentiated soft-tissue tumor misdiagnosed as IH of the eyelid in a neonate.

Management
Malignant undifferentiated tumors often require full excision and possibly chemotherapy and radiation. Treatment is multidisciplinary and may include ophthalmology, dermatology, oncology, and pathology services to ensure optimal therapy. The girl in this case underwent evaluation for metastatic disease, which was negative. Treatment was initiated with a sarcoma-based chemotherapy protocol (ifosfamide, etoposide, vincristine, doxorubicin, and cyclophosphamide) and proton therapy. She had an excellent clinical response to treatment by 2½ months of age (Fig 2B).

Lessons for the Clinician
• Cutaneous malignant soft-tissue tumors can clinically mimic infantile hemangiomas, which poses a diagnostic dilemma because treatment and prognosis drastically differ between the two conditions.
• Clinicians should consider diagnostic possibilities other than infantile hemangioma if the clinical history, behavior, or appearance of the lesion is not typical; atypical growth pattern or lack of response to treatment should raise suspicion for a possible malignant soft-tissue tumor.

Suggested Readings for this article are at http://pedsinreview.aappublications.org/content/36/9/420.full.

Correction
In the July 2015 article “Dehydration: Isonatremic, Hyponatremic, and Hypernatremic Recognition and Management” (Powers KS. Pediatrics in Review. 2015;36(7): 274–285, doi: 10.1542/pir.36-7-274), key phrases were deleted from the Question 1 answer options, which should begin as follows:
A. In hypernatremic dehydration ...
B. In hypernatremic dehydration ...
C. In hyponatremic dehydration ...
D. In hypernatremic dehydration ...
E. In hyponatremic dehydration ...
The phrases have been restored in the online quizzes, a correction has been attached to the article online. The journal regrets the copyediting error.
Dehydration: Isonatremic, Hyponatremic, and Hypernatremic Recognition and Management
Karen S. Powers

Pediatrics in Review 2015;36:274
DOI: 10.1542/pir.36-7-274

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://pedsinreview.aappublications.org/content/36/7/274

An erratum has been published regarding this article. Please see the attached page for:
http://pedsinreview.aappublications.org/content/36/9/422.full.pdf

Data Supplement at:
http://pedsinreview.aappublications.org/content/suppl/2015/07/17/36.7.274.DC1