Introduction
Infants and young children have a greater need for water and are more vulnerable to alterations in fluid and electrolyte imbalances than adults. Water and electrolyte imbalances occur more frequently and more rapidly in children, and children adjust less promptly to those disturbances. Infants and children also have a greater proportional amount of extracellular fluid volume.

Normal Distribution of Body Water and Electrolytes
Total body water (TBW) varies with an individual’s age and amount of muscle mass and body fat; the more fat an individual has, the smaller the proportion of body weight attributed to body water
- 80%–85% of a premature infant’s body weight is attributed to water
- About 70% of a full-term infant’s body weight is attributed to water
- Body weight attributed to water in young adults differs between males and females:
  - Males: ~ 65% total body weight
  - Females: ~ 52% of total body weight
During infancy, a larger proportion of body water is extracellular; about half an infant’s extracellular fluid is exchanged daily

Changes in Fluid Composition and Distribution During Critical Illness
Critically ill infants and children tend to retain fluids because of increased secretion of antidiuretic hormone (ADH) and aldosterone
- Catecholamine release, hypotension, fright, and pain stimulate the release of ADH, renin, and aldosterone
- ADH release is also stimulated by any condition that reduces left atrial pressure (eg, hemorrhage, positive-pressure ventila-
tion, or severe pulmonary hypertension), general anesthetics, morphine, and barbiturates

- Critically ill pediatric patients often exhibit decreased urine volume and increased urine concentration in the presence of hemodilution
- A newborn’s kidney has a limited ability to concentrate urine; a neonate may have decreased urine volume and only moderate urine concentration

**Administering Maintenance Fluid**

Fluid administration must be tailored to prevent fluid overload or sodium balance (Tables 22-1 and 22-2)

- Fluid and electrolyte losses in urine most closely resemble 0.45 normal saline (½NS); insensible losses are more similar to 0.2 NS

**Table 22-1. Pediatric Daily Fluid Requirements**

<table>
<thead>
<tr>
<th>Age/Weight</th>
<th>Fluid Goal</th>
<th>Calories (kcal/kg)</th>
<th>Dextrose*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants (&gt;1 mo)</td>
<td>100–120 mL/kg</td>
<td>~ 120</td>
<td>5%–10%</td>
</tr>
<tr>
<td>&lt; 10 kg</td>
<td>100 mL/kg</td>
<td>~ 110</td>
<td>5%</td>
</tr>
<tr>
<td>10–20 kg</td>
<td>1,000 mL + 50 mL/kg &gt; 10 kg</td>
<td>~ 80</td>
<td>5%</td>
</tr>
<tr>
<td>&gt; 20 kg</td>
<td>1,500 mL + 20 mL/kg &gt; 20 kg</td>
<td>≧ 45</td>
<td>5%</td>
</tr>
</tbody>
</table>

*Percent of dextrose in water.

\[
\frac{1}{2} \text{NS}
\]

- ½NS is usually administered with 5% or 10% glucose immediately postoperatively to replace insensible and urine losses
- Excessive gastrointestinal losses are generally replaced with ½NS
- All infusions should be connected to a constant infusion pump
  - Specific electrolyte needs and therapies require different concentrations
    - Generally use D\(_{10}\) ½NS for neonates; D\(_{5}\) ½NS for other infants and children
    - Add potassium (20 mEq/L) if urine output is documented or after evaluating serum electrolytes
### Table 22-2. Pediatric Daily Electrolyte Requirements*

<table>
<thead>
<tr>
<th>Age</th>
<th>Sodium</th>
<th>Potassium</th>
<th>Magnesium †</th>
<th>Calcium †</th>
<th>Phosphorus †</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants/children</td>
<td>3.0 mEq/kg ‡</td>
<td>2.0 mEq/kg</td>
<td>0.25 mEq/kg</td>
<td>1.0 mEq/kg</td>
<td>0.50 mEq/kg</td>
</tr>
<tr>
<td>Adolescents</td>
<td>2.0 mEq/kg</td>
<td>1.0 mEq/kg</td>
<td>0.25 mEq/kg</td>
<td>0.25 mEq/kg</td>
<td>0.25 mEq/kg</td>
</tr>
</tbody>
</table>

* A standard pediatric fluid is D$_5$$\frac{1}{2}$NS with 20 mEq of KCl/L.
† Consider adding if using intravenous fluids for more than 3–5 days.
‡ 1 mEq potassium phosphate = 0.68 mmol phosphorus (1 mEq sodium phosphate = 0.75 mmol phosphorus).

### Table 22-3. Differentiating Sources of Sodium Disturbances

<table>
<thead>
<tr>
<th>Condition</th>
<th>Intravascular</th>
<th>Serum Sodium</th>
<th>Urine Volume</th>
<th>Urine Sodium</th>
<th>Net Sodium Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyponatremic dehydration</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>SIADH</td>
<td>Normal or ↑</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>Normal</td>
</tr>
<tr>
<td>CSW</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
</tbody>
</table>

↓: decreased
↑: increased
CSW: cerebral salt wasting
SIADH: syndrome of inappropriate antidiuretic hormone
Monitor urine volume closely, using a Foley if necessary
- Should average > 1 mL/kg/h if fluid volume is adequate
- If fluid is severely restricted, urine volume may average 0.5–1 mL/kg/h

**Electrolyte Management**

- **Hypokalemia**
  - Encountered most commonly following use of loop and thiazide diuretics
  - Also found following vomiting, diarrhea, intestinal fistulas, ileostomy drainage, or gastric suctioning
  - Excessive renal excretion of potassium is associated with metabolic alkalosis, renal tubular acidosis, and diabetic ketoacidosis
  - Cardiac dysrhythmias occur infrequently unless the hypokalemia is severe
  - Electrocardiogram (ECG) findings include low voltage, flattened T waves, and prolonged QT interval
- **Management**
  - Use intravenous (IV) potassium replacement if the child is nauseated and vomiting; continuous replacement is preferred over potassium chloride bolus therapy
  - Potassium chloride infusions should not exceed 1 mEq/kg and should not be delivered faster than over 60–90 minutes (maximum 25 mEq KCl)
  - Patient should be observed closely on a monitor
  - Peripheral veins will routinely tolerate up to 40–60 mEq KCl/L in IV fluids
  - Consider potassium chloride supplements to enteral feeds in the absence of vomiting
    - Administer 1 mEq/kg/dose 3–4 times daily, depending on the degree of hypokalemia
    - Do not exceed 1 mEq/oz of enteral formula during tube feeds
- **Hyperkalemia**
  - Etiologies include excessive potassium administration, significant cell destruction, and reduced renal excretion of potassium
Common signs include generalized muscle weakness and flaccidity

ECG findings include tall, peaked T wave on ECG initially, followed by widened QRS, ST segment depression, and increasing R wave amplitude

As serum potassium level rises, PR interval prolongs

Ventricular fibrillation may occur

Management includes frequent reassessments, careful monitoring of “ins and outs” (ie, fluids in and urine and drainage out)

- Reexpand intravascular volume with NS 10–20 mL/kg
- Administer calcium gluconate 100 mg/kg over 5 minutes (maximum dose is 2 g). Give IV calcium through central line, if available; calcium preparations can cause skin burns if peripheral IV is infiltrated
- Use sodium bicarbonate (1 mEq/kg over 5 min) in the presence of metabolic acidosis
- Glucose and insulin therapy are very effective in children; give 0.5 g/kg of glucose (5 cc/kg of 10% dextrose in water) and 0.1 unit/kg of insulin over 30 minutes
- Administer sodium polystyrene sulfonate 1 g/kg/dose diluted in 3–4 mL of water every 6 hours orally, nasogastrically or rectally
- Consider giving an albuterol treatment, which will begin to lower the serum potassium level in 30 minutes

Hyponatremia

- Common etiologies for hyponatremia include hyponatremic dehydration due to excessive sodium loss or diuretic use; syndrome of inappropriate antidiuretic hormone (SIADH) and cerebral salt wasting can be seen with major head trauma or meningitis (Table 22-3)

- Hyponatremic dehydration can be corrected over 24 hours, with half the deficit replaced in the first 8 hours

- The sodium (Na⁺) deficit can be calculated as follows:

\[ \text{Na⁺ deficit} = (140 - \text{serum Na⁺}) \times \text{weight (kg)} \times 0.6 \]

- Add the deficit sodium and water to the daily maintenance sodium and water to derive the most appropriate fluid (usually at least \( \frac{1}{2} \)NS)
The serum sodium should rise no more than 0.5 mEq/L/h
Potassium chloride (20 mEq/L) may be added when patient voids

Managing SIADH involves relative fluid and free water restriction; use NS to avoid giving free water

Managing cerebral salt wasting involves aggressively replacing ongoing salt and water loss
  - Check urine electrolytes if possible
  - Replace sodium deficit with a combination of NS and 3% hypertonic saline, ideally through a central line; maximum infusion rate 2 mL/kg/h (1 mEq/kg/h)

- Hypernatremia (Na⁺ > 165)
  - Etiologies include hypernatremic/hypertonic dehydration and diabetes insipidus
  - Management of hypernatremic dehydration, like all severe hypertonic states (eg, diabetic ketoacidosis) requires a slower correction, usually over 48-72 hours, depending on the duration and severity of the hypernatremia (eg, a 10-day-old breast-fed infant whose serum sodium rose to 165 mEq/dL over many days)
  - Calculating the sodium deficit can be complex; if all volume lost is assumed to be isotonic saline 140 mEq/L, the most likely calculation error will be avoided
    - For severe hypernatremic dehydration (Na⁺ ≥ 165), do not use anything less tonic than NS for at least the first 12 hours
    - The sodium level should fall no more than 0.5 mEq/L/h