volume, intravenous (IV) fluids also correct and maintain normal acid-base and electrolyte balance. A thorough understanding of the appropriate selection, timing, and goals of fluid therapy is vital to optimize patient care.

PATHOPHYSIOLOGY

OXYGEN DELIVERY AND TISSUE PERFUSION

Oxygen is delivered to cells via the circulation as a function of red blood cell mass and cardiorespiratory function. Oxygen enables continuous production of energy by cells in the form of adenosine triphosphate. Poor oxygenation compromises cell energetics and function and results in the clinical manifestations of organ dysfunction and failure.

Cardiac output is the most important determinant of oxygen delivery, and it has sufficient flexibility to compensate for reduced oxygen-carrying capacity, as well as increased metabolic demands. The physiologic response to a decrease in cardiac output is catecholamine-induced tachycardia and enhanced cardiac contractility, which attempt to maintain oxygen delivery in the face of falling stroke volume. Concomitant venoconstriction maintains intrathoracic blood volume (preload), whereas arterial vasoconstriction shunts perfusion to vital organs and maintains critical organ perfusion pressure.

Cardiac output and organ perfusion vary dramatically under changing physiologic, pathologic, and pharmacologic stimuli. Organ blood flow is directly proportional to perfusion pressure in most vascular beds. In hypovolemia, protection of arterial (organ perfusion) pressure occurs via peripheral vasoconstriction at the expense of reduced flow to noncritical circulations (e.g., hepatosplanchnic, renal, cutaneous). Consequently, arterial pressure is maintained despite hypovolemia and organ hypoperfusion.

Effective circulating volume (ECV) conceptualizes the portion of intravascular volume contributing to organ perfusion. ECV decreases with hypovolemia but does not necessarily correlate with volume status because organ perfusion is also dependent on cardiac output, vasomotor tone, and circulatory distribution. As an example, ECV may be compromised by limited cardiac output despite optimized intravascular preload status.

Volume depletion describes a state of contracted extracellular fluid with clinical implications of compromised ECV, tissue perfusion, and function. This is distinguished from

**KEY POINTS**

- Shock is defined by inadequate tissue perfusion and not by systemic blood pressure.
- The majority of emergency department patients who require resuscitation are in compensated shock with normal blood pressure.
- Volume expansion with isotonic fluid is the most important immediate therapy for most patients with circulatory insufficiency.
- Resuscitation and maintenance fluids should be tailored to an individual patient’s acid-base and electrolyte status.
- The fluid dose titrated to appropriate end points is more important than the individual fluid class (i.e., crystalloid versus colloid) used for resuscitation.
- Normal vital signs do not guarantee adequate systemic perfusion and should not be the ultimate end point of resuscitation.
- Dynamic measures of fluid responsiveness should be used to assist in the management of patients who remain hypoperfused despite initial empiric volume therapy.

**PERSPECTIVE**

Hypovolemia is a common crisis in acute care medicine. Loss of volume is often a direct consequence of acute fluid or blood loss, but relative hypovolemia complicates many clinical conditions. Its severity ranges from mild compensated hypovolemia to shock and hypotension that place end-organ perfusion and function at risk. Fluid therapy to optimize cardiac performance and restore fluid and electrolyte balance is a cornerstone of medical support. Timely and appropriate fluid therapy maintains macrocirculatory and microcirculatory support and reduces mortality. In contrast, both underresuscitation and overly aggressive fluid therapy can have an adverse impact on organ function and outcome. Inadequate resuscitation risks leaving a patient in compensated shock. Overly aggressive fluid administration results in volume overload without improving oxygen delivery and is associated with worse clinical outcomes. In addition to sustaining circulating blood volume, intravenous (IV) fluids also correct and maintain normal acid-base and electrolyte balance. A thorough understanding of the appropriate selection, timing, and goals of fluid therapy is vital to optimize patient care.

**PATHOPHYSIOLOGY**

OXYGEN DELIVERY AND TISSUE PERFUSION

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Volume depletion describes a state of contracted extracellular fluid with clinical implications of compromised ECV, tissue perfusion, and function. This is distinguished from
dehydration, which implies an intracellular water deficit characterized by plasma hypernatremia and hyperosmolarity.

**WATER**

Water is the most abundant constituent of the body. An adult man weighing 70 kg (154 lb) contains approximately 45 L of water, which accounts for 60% of body mass (Table 159.1). Total body water (TBW) is proportional to lean body mass and affects maintenance fluid requirements. TBW is physiologically compartmentalized into intracellular and extracellular spaces. The extracellular compartment is anatomically and conceptually divided into vascular and interstitial spaces.

Water freely crosses cell membranes, and osmotic forces determine the distribution of water within the body. The intracellular and extracellular fluid environments remain isosmolar but physiochemically distinct via tight regulation of dissolved solutes and proteins. Membrane-bound sodium-potassium adenosine triphosphatase pumps compartmentalize sodium and potassium to the extracellular and intracellular spaces, respectively. Active restriction of sodium to the extracellular space is the foundation of isotonic sodium-based resuscitation solutions.

Starling’s law describes the forces governing fluid flux across vascular endothelial membranes. In healthy persons, transcapillary hydrostatic force is nearly opposed by colloid oncotic pressure. Small net loss from the vascular space is ultimately returned to the systemic circulation via lymphatics. Albumin normally accounts for 80% of colloid oncotic pressure, whereas large cellular moieties such as red blood cells and platelets contribute little oncotic pressure effect. Positive hydrostatic pressure, hypoalbuminemia, and pathologic endothelial permeability are common clinical conditions that enhance extravasation of fluid from the vascular compartment. The clinical consequences include large and ongoing volume resuscitation requirements coupled with tissue (e.g., lung, gut, brain) edema, which may compromise function.

**PRESENTING SIGNS AND SYMPTOMS**

Absence hypovolemia occurs as a consequence of loss of water, electrolytes, or blood (or any combination of the three) (Box 159.1). Patients with hypovolemia most often have symptoms related to reduced cardiac output such as fatigue, dyspnea, postural dizziness, and near or true syncope. Tolerance is variable and depends on the acuity and severity of the hypovolemia, associated anemia, individual physiologic reserve, and primary cause. Organ dysfunction is often a heralding sign of hypovolemia and may occur in the absence of global hypoperfusion or frank hemodynamic instability.

Shock is defined as a state of inadequate tissue perfusion in which oxygen delivery does not meet metabolic requirements. The term does not reflect perfusion pressure—shock may occur with low, normal, or elevated blood pressure. Unfortunately, clinical signs are unreliable indicators of oxygen delivery and blood volume.  

Compensated shock refers to inadequate perfusion in the setting of normal blood pressure. The majority of critically ill patients are in compensated shock. The difficulty in identifying these patients prompted the terms occult and cryptic shock to describe normotensive patients with alternative evidence of cardiovascular insufficiency. Hyperlactatemia (<3 mmol/L) is an important marker to aid in identification of these high-risk patients. Left unresuscitated, these patients often progress to frank hypotension.
Brief episodes of hypotension are important markers of hypoperfusion and herald progressive hemodynamic deterioration. These self-limited episodes of transient hypotension represent progressive exhaustion of cardiovascular compensation and are the first sign of uncompensated shock.\textsuperscript{8} Uncompensated shock is characterized by hypotension that occurs when physiologic attempts to maintain normal perfusion pressure are overwhelmed or exhausted. Sustained hypotension signifies a late stage of shock.

Volume status and perfusion should be evaluated during every emergency department (ED) examination (Box 159.2). Delayed capillary refill, dry axillae and mucous membranes, abnormal skin turgor, sunken eyes, and a depressed fontanelle are classic but imperfect hallmarks of hypovolemia.\textsuperscript{9,10} Peripheral cyanosis, cool extremities, and cutaneous mottling (cutis marmorata) characterize classic hypodynamic shock but are not a primary indication of hypovolemia. In contrast, early hyperdynamic septic shock may be manifested as peripheral vasodilation with warm extremities and brisk capillary refill.

Generalized tissue edema reflects total body sodium and fluid excess but does not quantify intravascular status and may be accompanied by hypovolemia, especially in acute illness. Acute weight change implies loss of fluid rather than lean body mass and is helpful in patients with a reliable comparison weight.

**BOX 159.2 Clinical Indicators of Hypoperfusion That Warrant Consideration for a Rapid Monitored Fluid Challenge**

- Mean arterial pressure (MAP) < 65 mm Hg
- Systolic blood pressure < 90 mm Hg
- Decrease in MAP > 20 mm Hg from baseline
- Shock index > 0.9
- Sinus tachycardia > 100 beats/min
- Serum lactate > 3 mmol/L
- Base deficit ≤ 6
- Oliguria (urine output < 0.5 mL/kg/hr)
- Abnormal peripheral perfusion

**TREATMENT**

Circulatory failure is the final common pathway of many diseases. Inadequate circulating volume is the most common and immediately reversible factor in patients with acute circulatory insufficiency. Accordingly, fluid therapy is the initial management of undifferentiated shock. Pathologic vasodilation compounds the fluid deficit in common conditions such as sepsis, anaphylaxis, adrenal failure, neurogenic shock, and toxin-induced shock. Acute cardiac decompensation and pulmonary embolism are two exceptional situations in which limited volume resuscitation takes secondary priority to catecholamine and mechanical support.

Early recognition of hypovolemia and shock must be coupled with aggressive resuscitation to have an effect on patients. Timely resuscitation is important because the window to reverse critical organ hypoperfusion and affect clinical outcome is measured in hours and often transpires in the ED.\textsuperscript{11} Equivalent but delayed resuscitation yields greater morbidity and mortality.\textsuperscript{12-15} The immediate goal of fluid resuscitation is intravascular expansion to optimize stroke volume and oxygen delivery.

**FLUID RESUSCITATION**

**Resuscitation Targets (End Points)**

The target end point of resuscitation guides the dose and selection of interventions, including fluid therapy. In high-risk patients, fluid selection appears to be less important than volume dosage titrated to an appropriate therapeutic end point.\textsuperscript{1,16} Rapid restoration of systemic pressure (mean arterial pressure > 65 mm Hg) is a first priority to support vital organ autoregulated blood flow. However, stabilization of blood pressure does not guarantee adequate organ perfusion—resuscitation aimed primarily at normotension risks leaving the patient in persistent compensated shock.\textsuperscript{17} Unfortunately, there is no single best resuscitation end point for all clinical circumstances. An approach seeking to normalize a combination of both global and regional perfusion markers is most prudent (Box 159.3).

**Empiric Fluid Challenge**

Empiric volume challenges remain the standard means of early fluid resuscitation. Volume expansion is achieved by infusing serial aliquots of isotonic fluid under direct observation. This strategy is appropriate for acute undifferentiated shock or for obvious or suspected hypovolemia. Crystalloid (10 to 20 mL/kg) or colloid (4 to 7 mL/kg) should be infused rapidly over a period of 15 minutes with serial boluses titrated to the clinical end point objective while monitoring for adverse effects. A positive clinical response to volume loading confirms volume responsiveness but does not predict further response to therapy. Failure to recognize patients not

**BOX 159.3 Conditions Warranting a Strategy of Limited-Volume Resuscitation Pending Surgical Control of Hemorrhage**

- Penetrating thoracoabdominal trauma
- Traumatic aortic injury
- Ruptured abdominal aortic aneurysm
- Severe pelvic fracture
- Gastrointestinal bleeding
- Ectopic pregnancy
- Major hemoperitoneum
- Postpartum hemorrhage

**PRIORITY ACTIONS**

**Prioritized End Points of Fluid Resuscitation**

- Adequate intravenous access
- Mean arterial pressure > 65 mm Hg
- Systemic perfusion
  - Central venous oxygen saturation ($S_{CO_2}$) > 70%
  - Serum lactate clearance (>5%/hr) and normalization (<2 mmol/L)
- Regional perfusion
  - Urine output >0.5 mL/kg/hr
  - Normalized peripheral perfusion
responding to fluid may contribute to overly aggressive volume therapy and delay alternative care measures.

Total volume requirements are difficult to predict at the onset of resuscitation and are often underestimated. Classic hypovolemia, as occurs with acute hemorrhage or fluid loss, may stabilize rapidly with appropriate volume expansion. The 3:1 rule of hemorrhage resuscitation suggests that 3 volumetric units of crystalloid are required to replete an extracellular fluid deficit of 1 unit of blood loss. However, experimental models have confirmed that with severe hemorrhage, volume requirements exceed the 3:1 suggestion. Pathologic vasodilation and capillary leak contribute to the need for ongoing volume replacement. Exaggerated transcapillary fluid shifts are common with early burns, peritonitis, and pancreatitis. Crystalloid requirements average greater than 60 mL/kg in the first hours of septic shock but may be as high as 200 mL/kg to normalize perfusion.18,19

**Predicting Volume Responsiveness**

The utility of fluid administration to improve stroke volume depends on a number of variables, including venous tone and ventricular function. Critically ill patients may manifest continued hypoperfusion despite initial empiric volume resuscitation. As an example, only half of hypotensive patients with sepsis are stabilized with volume resuscitation alone.20 In the absence of overt clinical hypovolemia or ongoing fluid loss, volume responsiveness should not be assumed. Continued fluid therapy does not have any impact on macrocirculatory flow in up to 50% of critically ill patients.20

Volume or preload responsiveness refers to the ability to augment stroke volume with fluid administration. In contrast to an empiric volume challenge, volume responsiveness is gauged before fluid administration, and the resulting information is used to guide whether fluid administration is a solution to reverse clinical hypoperfusion. Fluid loading in volume-unresponsive patients is optimally avoided because it delays appropriate therapy and contributes to fluid excess that contributes to organ dysfunction, including hypoxemic respiratory failure and abdominal compartment syndrome.21 A more rational approach for patients who remain hypoperfused after initial empiric management incorporates selection and titration of subsequent therapy under the guidance of objective cardiovascular monitoring.

A target central venous pressure (CVP) of 8 to 12 mm Hg is recommended to maximize preload before instituting pressor and inotropic support.22 Unfortunately, there is no consistent threshold CVP to reliably estimate response to fluid administration.22 Values that are considered low, normal, or high can be found in patients who respond positively to fluid.20

Dynamic hemodynamic indices are more dependable signs of volume responsiveness. Respiratory variation in stroke volume during positive pressure mechanical ventilation is among the most useful signs of fluid responsiveness.23 Respiratory collapse of the inferior vena cava (IVC) is another helpful indicator. Minimal IVC variation is associated with supranormal CVP and a low probability of fluid responsiveness. Inspiratory IVC collapse greater than 50% indicates a high probability of augmenting stroke volume with fluid therapy.

**FLUID SELECTION**

Early resuscitation and ongoing replacement of fluid deficits may be performed with a variety of fluids. Because each possesses specific benefits and potential disadvantages in given clinical scenarios, an understanding of fluid composition is important. Isotonic solutions are effective volume expanders (Table 159.2). Electrolyte disorders (including hypernatremia and hyponatremia) take secondary priority to isotonic volume loading in hypoperfused patients. Hypotonic solutions are ineffective and inappropriate volume expanders.

**Crystalloids**

Isotonic sodium-based crystalloids are distributed to the extracellular compartment, which includes the vascular space. Partitioning within the extracellular fluid leaves 25% of the infused volume within the circulation. Normal saline and lactated Ringer (LR) solution are two isotonic resuscitation solutions in common use. LR solution, also known as Hartmann solution, was developed as a more physiologic alkalinizing replacement solution; bicarbonate is generated by lactate metabolism via the Cori cycle.

The clinical superiority of any single crystalloid remains unproved, and selection of fluid should be based on the source of the hypovolemia, associated electrolyte derangements, and volume requirements. Normal saline provides a supraphysiologic chloride load that induces metabolic acidosis when administered in large volumes. This is advantageous in correcting the volume and electrolyte disturbances (hypochloremic metabolic alkalosis) associated with loss of gastric secretions (e.g., profuse vomiting, gastric outlet obstruction, nasogastric suctioning). LR solution provides a more physiologic electrolyte balance and is often preferred for large-volume resuscitation. LR is recommended for trauma resuscitation but is incompatible with blood. Isotonic bicarbonate (i.e., three ampules of sodium bicarbonate in 1 L of sterile water) is an alternative resuscitation fluid for patients with coexisting metabolic acidosis.

**Colloids**

Colloid solutions are composed of electrolyte preparations reinforced with macromolecules designed to preserve colloid oncotic pressure. Vascular retention of colloid makes these formulations more efficient volume expanders with a longer duration of action than is the case with crystalloids. Crystalloid solutions require two to four times more volume for equivalent resuscitation. Dilutional hypoalbuminemia, transcapillary fluid shift, and interstitial and pulmonary edema may therefore be limited with colloid use. When dosed to the same end points, colloids and crystalloids are equally effective. Randomized clinical trials have failed to prove clinical superiority of one fluid class, with comparable rates of mortality and lung dysfunction.23,24 Traumatic brain injury remains one important exception in which isotonic albumin is associated with a higher risk for adverse outcomes when compared with equivalent crystalloid.25,26 Colloid solutions remain the standard ED choice for resuscitation and confer a significant cost advantage over colloid solutions.

Human albumin and hydroxyethyl starch (HES) are the colloids primarily used in clinical practice in the United States. Human albumin solutions are heat-sterilized...
derivatives of donor plasma. Isotonic 5% albumin is recommended for resuscitation of patients with severe hypoalbuminemia and end-stage liver disease, but there is little outcome evidence to support this position. HES, a semisynthetic polymerized amylopectin compound, has supplanted dextran and gelatin-based colloids. A 6% solution provides volume expansion equivalent to that of 5% albumin. The renal dysfunction and coagulopathy that complicated early-generation synthetic colloids do not appear to be clinically significant with new-generation HES solutions.

**Hypertonic Solutions**

**HYPERONCOTIC ALBUMIN** Infusions of hyperoncotic albumin (20% to 25%) result in vascular expansion greater than two times the volume administered. Besides the obvious benefits of small-volume resuscitation, improved portability, and more rapid hemodynamic stabilization, hyperoncotic albumin has additional advantages. Synergistic interaction with administered drugs and primary antioxidant effects are explanations hypothesized for the improved morbidity linked to hyperoncotic albumin administered to patients with complicated hypoalbuminemic states. End-stage liver disease is one important example in which hyperoncotic albumin attenuates renal dysfunction and death in patients with spontaneous bacterial peritonitis or those undergoing large-volume paracentesis.

**HYPERTONIC SALINE** Hypertonic sodium solutions rapidly expand intravascular volume by mobilizing water from the interstitial and intracellular spaces. A small infusion expands plasma several times the volume infused. Used alone, the hemodynamic impact of hypertonic crystalloid is transient. Hypertonic crystalloid is often used in combination with hyperoncotic colloid (6% dextran or 10% HES) to sustain vascular expansion. Animal resuscitation models have demonstrated additional benefits, including enhanced cardiac output, improved microcirculatory flow, and attenuated inflammatory response. Hypertonic saline is safe, but data are insufficient to conclude that it is better than isotonic crystalloid for the resuscitation of patients with burns, trauma, or sepsis. Severely traumatized patients with associated traumatic brain injury remain an exceptional use for hypertonic saline, but outcome evidence is mixed.

**SPECIAL TREATMENT CONSIDERATIONS**

**Minimal-Volume Resuscitation of Hemorrhagic Shock**

Traditional resuscitation of hemorrhagic shock prioritized rapid restoration of circulating blood volume with crystalloid and blood products. Strategic limited-volume resuscitation for uncontrolled hemorrhage dates back to the early 1900s and reemerged in the 1980s. The rationale is that the increased intravascular pressure and hemodilution resulting from aggressive fluid resuscitation compounds the blood loss by precipitating rebleeding from hemostatic sites. Animal models of uncontrolled hemorrhage reveal that aggressive fluid administration reduces oxygen delivery and results in higher mortality. The widely recognized Houston experience showed a mortality benefit in penetrating trauma victims, but the results have yet to be matched.

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**Table 159.2 Intravenous Fluid Composition and Distribution**

<table>
<thead>
<tr>
<th>SOLUTION</th>
<th>ELECTROLYTES (mEq/L)</th>
<th>DISTRIBUTION</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Na</td>
<td>K</td>
</tr>
<tr>
<td>Crystalloid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NS (0.9%)</td>
<td>154</td>
<td>154</td>
</tr>
<tr>
<td>LR</td>
<td>130</td>
<td>4</td>
</tr>
<tr>
<td>1 L water with 3 ampules HCO₃ (150 mEq)</td>
<td>130</td>
<td></td>
</tr>
<tr>
<td>3% NaCl</td>
<td>513</td>
<td>513</td>
</tr>
<tr>
<td>7.5% NaCl</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.45% NaCl</td>
<td>77</td>
<td>77</td>
</tr>
<tr>
<td>0.20% NaCl</td>
<td>34</td>
<td>34</td>
</tr>
<tr>
<td>D₂W</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Colloid**

<table>
<thead>
<tr>
<th>SOLUTION</th>
<th>ELECTROLYTES (mEq/L)</th>
<th>DISTRIBUTION</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Na</td>
<td>K</td>
</tr>
<tr>
<td>Hextend</td>
<td>143</td>
<td>3</td>
</tr>
<tr>
<td>Hespan</td>
<td>154</td>
<td>154</td>
</tr>
<tr>
<td>Human albumin 5%</td>
<td>145</td>
<td>95</td>
</tr>
</tbody>
</table>

D₂W, 5% Dextrose in water; LR, lactated Ringer solution; NS, normal saline.
by other investigators.\textsuperscript{34,35} In this strategy, hypotensive patients with a source of uncontrolled life-threatening hemorrhage receive IV fluids titrated to sustain critical organ flow until definitive surgical control is achieved (see Box 159.3). Conventional resuscitation ensues once surgical hemostasis is achieved.

The degree and duration of permissive hypotension remain to be clarified, although current recommendations target a systolic blood pressure of 70 mm Hg. Patients with concomitant traumatic brain injury are not candidates for this strategy.

**BURN RESUSCITATION**

Patients with partial-thickness and full-thickness burns exhibit marked fluid shifts related to denuded skin, injured tissue, and the systemic inflammatory response. Early anticipation of these large fluid requirements prevents underresuscitation. The Parkland formula remains the most commonly used guide for acute burn resuscitation. Formula calculations are based on the time of injury rather than the time until medical attention and incorporate prehospital fluid administration. All burn formulas provide only estimates of fluid requirements. The requirements must be modified and individualized based on patient response because volume needs may substantially exceed formula approximation.\textsuperscript{36} LR solution is the resuscitation crystalloid preparation of choice for acute burn management. In addition to burn formula replacement, maintenance fluid requirements should be allocated. Urine output greater than 1 mL/kg/hr is a traditional end point of acute burn resuscitation and may be augmented by the perfusion end points discussed earlier.

**Oral Rehydration Therapy**

Oral rehydration therapy is a valuable tool for maintenance and correction of mild to moderate dehydration secondary to gastroenteritis in children and adults. It remains underused in the United States despite worldwide success, guideline support, and evidence from controlled trials.\textsuperscript{37} When used as per published guidelines, oral rehydration therapy is comparable with IV therapy, but with reduced hospitalization and improved safety and expense. Small aliquots of fluid (as low as 5 mL, depending on patient size and tolerance) are administered by bottle, spoon, syringe, or nasogastric tube at regular 2- to 5-minute intervals to meet deficit and maintenance goals. After patient and family education, appropriate fluid selection is the most important factor in successful oral rehydration therapy.

Many common household fluids, including fruit juice, sport drinks, carbonated beverages, and soups, contain poorly tolerated concentrations of sugar and salt. Commercial (e.g., Rehydralyte, Pedialyte) and reconstituted liquids (oral rehydration salts or home recipe) are balanced, low-carbohydrate enteral solutions. One recipe for an oral rehydration solution containing home ingredients consists of 1 L of water, 8 teaspoons of sugar, and 1 teaspoon of salt.

**Maintenance Fluid Therapy**

The goal of maintenance fluid therapy is normal body fluid composition and volume. Fluid orders anticipate daily fluid requirements, ongoing losses, and coexisting electrolyte abnormalities. Though often ordered concurrently, the estimated physiologic fluid (true maintenance) should be consciously distinguished from therapy aimed to replace an existing fluid deficit.

Routine water and electrolyte maintenance is based on normal energy expenditure, sensible loss from urine and stool, and insensible loss from the respiratory tract and skin. Calculations assume euvolemia and are adjusted for body mass. The greater per-kilogram fluid requirements in children are proportional to their TBW and metabolism (Table 159.3).

All maintenance prescriptions should be individualized—energy expenditure, fluid losses, and electrolyte status vary with disease and dictate the rate and electrolyte modifications. For example, exfoliative skin disease, increased work of breathing, and fever enhance insensible loss. Measurable nasogastric, fistula, ostomy, and urinary drainage can be estimated and replaced per drained volume. Limitation of fluid and potassium is an important disease-specific modification for patients with renal insufficiency.

Hypotonic solutions (e.g., 0.45% and 0.2% sodium chloride) with or without dextrose and potassium are popular fixed-combination maintenance solutions. Hospitalized patients often suffer impaired free water excretion because of nonosmotic release of antidiuretic hormone, which makes them vulnerable to hyponatremia. The serum sodium concentration provides a simple and accurate marker of hydration status. Isotonic maintenance solutions should be considered in all patients (including children), especially those with a serum sodium concentration of less than 138 mEq/L.\textsuperscript{38-40} Glucose infusions are best formulated by adding dextrose to an electrolyte solution (e.g., LR solution, 0.45% or 0.9% normal saline) rather than using 5% dextrose in water, which behaves as electrolyte-free water on sugar metabolism.

### REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.

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**Table 159.3** Pediatric Maintenance Fluid Estimate Formulas

<table>
<thead>
<tr>
<th>BODY WEIGHT</th>
<th>DAILY MAINTENANCE (mL/DAY)</th>
<th>HOURLY MAINTENANCE (mL/HR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-10 kg</td>
<td>100 mL/kg</td>
<td>5 mL/kg</td>
</tr>
<tr>
<td>10-20 kg</td>
<td>1000 mL plus 50 mL/kg</td>
<td>40 mL plus 2 mL/kg</td>
</tr>
<tr>
<td>20-80 kg</td>
<td>1500 mL plus 20 mL/kg\textsuperscript{1}</td>
<td>60 mL plus 1 mL/kg\textsuperscript{1}</td>
</tr>
</tbody>
</table>

\textsuperscript{1}To a maximum of 2400 mL/day or 100 mL/hr.

*Note that the following two formulas calculate disparate rates. The difference between these calculated rates is clinically insignificant. Sodium and chloride—2 to 3 mEq per 100 mL water; potassium—1 to 2 mEq per 100 mL water. D\textsubscript{5}NS with 20 mEq KCl is a common maintenance solution for most euolemic pediatric patients and provides 20% of the daily calories at a routine maintenance rate. Comorbid conditions or electrolyte abnormalities may require modification.

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