

FLUIDS AND ELECTROLYTES

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CHAPTER 32

Maintenance Fluid Therapy

BODY COMPOSITION

Water is the most plentiful constituent of the human body. **Total body water (TBW)** as a percentage of body weight varies with age. The fetus has a high TBW, which gradually decreases to about 75% of birthweight for a term infant; TBW is higher in premature infants. During the first year of life, TBW decreases to about 60% of body weight and basically remains at this level until puberty. At puberty, the fat content of females increases more than that of males, who acquire more muscle mass than females. Because fat has low water content and muscle has high water content, by the end of puberty, TBW in males remains at 60%, but it decreases to 50% of body weight in females. During dehydration, TBW decreases and is a smaller percentage of body weight.

TBW has two main compartments: **intracellular fluid (ICF)** and **extracellular fluid (ECF)**. In the fetus and newborn, the ECF volume is larger than the ICF volume. The normal postnatal diuresis causes an immediate decrease in the ECF volume, which is then followed by continued expansion of the ICF volume because of cellular growth. By 1 year of age, the ratio of the ICF volume to the ECF volume approaches adult levels. The ECF volume is 20–25% of body weight, and the ICF volume is 30–40% of body weight (Fig. 32.1). With puberty, the increased muscle mass of males results in a higher ICF volume than in females.

The ECF is divided further into **plasma water** and **interstitial fluid** (see Fig. 32.1). Plasma water is about 5% of body weight. The blood volume, given a hematocrit of 40%, is usually 8% of body weight, although it is higher in newborns and young infants. The interstitial fluid, normally 15% of body weight, can increase dramatically in diseases associated with edema, such as heart failure, liver failure, and nephrotic syndrome.

The composition of solutes in the ICF and ECF is different. Sodium and chloride are the dominant cation and anion in the ECF. Potassium is the most abundant cation in the ICF, and proteins, organic anions, and phosphate are the most plentiful anions in the ICF. The dissimilarity between the anions in the ICF and the ECF is determined largely by the presence of intracellular molecules that do not cross the cell membrane, the barrier separating the ECF and the ICF. In contrast,

the difference in the distribution of cations—sodium and potassium—is due to the activity of the Na^+, K^+ -ATPase pump, which extrudes sodium from cells in exchange for potassium.

REGULATION OF INTRAVASCULAR VOLUME AND OSMOLALITY

Proper cell functioning requires close regulation of plasma osmolality and intravascular volume; these are controlled by independent systems for water balance which determines osmolality, and sodium balance which determines volume status. Maintenance of a normal **osmolality** depends on control of water balance. Control of **volume status** depends on regulation of sodium balance.

The plasma osmolality is tightly controlled between 285 and 295 mOsm/kg through regulation of water intake and urinary water losses. A small increase in the plasma osmolality stimulates thirst. Urinary water losses are regulated by the secretion of **antidiuretic hormone (ADH)**, which increases in response to an increasing plasma osmolality. ADH, by stimulating renal tubular reabsorption of water, decreases urinary water losses. Control of osmolality is subordinate to maintenance of an adequate intravascular volume. When significant volume depletion is present, ADH secretion and thirst are stimulated, regardless of the plasma osmolality.

Volume depletion and volume overload may cause significant morbidity and mortality. Because sodium is the principal extracellular cation and is restricted to the ECF, adequate body sodium is necessary for maintenance of intravascular volume. The kidney determines sodium balance because there is little homeostatic control of sodium intake, although salt craving occasionally occurs, typically in children with chronic renal salt loss or adrenal insufficiency. The kidney regulates sodium balance by altering the percentage of filtered sodium that is reabsorbed along the nephron. The **renin-angiotensin system** is an important regulator of renal sodium reabsorption and excretion. The juxtaglomerular apparatus produces renin in response to decreased *effective* intravascular volume. Renin cleaves angiotensinogen, producing angiotensin I, which angiotensin-converting enzyme converts into angiotensin II. The actions of angiotensin II include direct stimulation of the proximal tubule to increase sodium reabsorption and stimulation of the adrenal gland to increase aldosterone secretion, which increases sodium reabsorption in the distal nephron. In contrast, volume expansion stimulates the synthesis of **atrial natriuretic peptide**, which increases urinary sodium excretion.

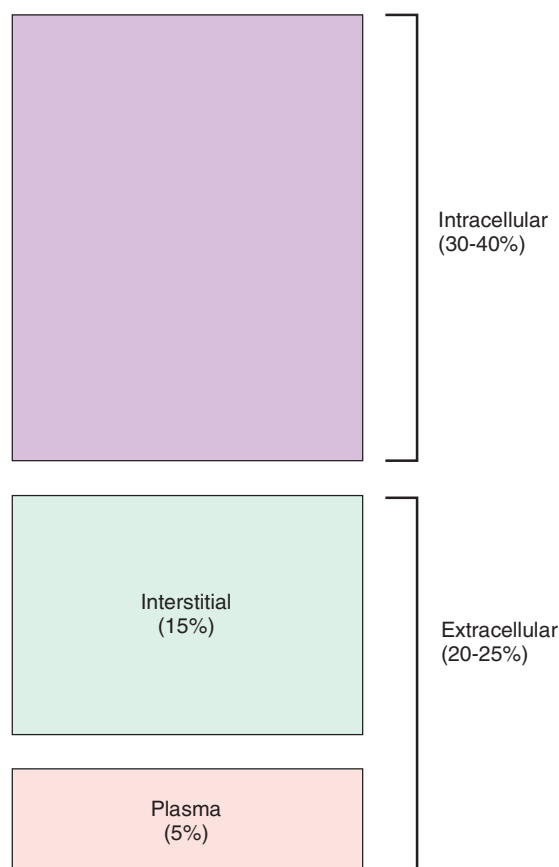


FIGURE 32.1 Compartments of total body water, expressed as percentage of body weight, in an older child or adult. (From Greenbaum LA. Pathophysiology of body fluids and fluid therapy. In Kliegman RM, St Geme JW, eds. *Nelson Textbook of Pediatrics*. 20th ed. Philadelphia: Elsevier, 2020:390.)

MAINTENANCE FLUIDS

Maintenance intravenous (IV) fluids are used in children who cannot be fed enterally. Along with maintenance fluids, children may require concurrent **replacement fluids** if they have excessive **ongoing losses**, such as may occur with diarrhea, vomiting, or drainage from an enteral feeding tube. In addition, if dehydration is present, the patient also needs to receive deficit replacement (see [Chapter 33](#)).

Maintenance fluids are composed of a solution of water, glucose, sodium, potassium, and chloride. This solution replaces electrolyte losses from the urine and stool, as well as water losses from the urine, stool, skin, and lungs. The glucose in maintenance fluids provides approximately 20% of normal caloric needs. This is enough to prevent the development of starvation ketoacidosis and diminishes the protein degradation that would occur if the patient received no calories.

Maintenance fluids do not provide adequate calories, protein, fat, minerals, or vitamins. Patients should not remain on maintenance therapy indefinitely; parenteral nutrition (see [Chapter 34](#)) should be used for children who cannot be fed enterally for more than a few days.

Daily water losses are measurable (urine and stool) and not measurable (*insensible losses* from the skin and lungs). Failure to replace these losses leads to a thirsty child and, ultimately,

TABLE 32.1 Body Weight Method for Calculating Maintenance Fluid Volume and Rate

BODY WEIGHT (kg)	VOLUME PER DAY	HOURLY RATE
0–10	100 mL/kg	4 mL/kg/hr
11–20	1,000 mL + 50 mL/kg for each 1 kg >10 kg	40 mL/hr + 2 mL/kg/hr × (wt–10)
>20	1,500 mL + 20 mL/kg for each 1 kg >20 kg*	60 mL/hr + 1 mL/kg/hr × (wt–20) [†]

*The maximum total fluid per day is normally 2,400 mL.

[†]The maximum fluid rate is normally 100 mL/hr.

a dehydrated child. [Table 32.1](#) provides a system for calculating **24-hour maintenance water** needs based on the patient's weight.

After calculation of water needs and electrolyte needs, children typically receive D5 in Normal Saline plus 20 mEq/L of KCl as a maintenance solution. This assumes that there is no disease process present that would require an adjustment in either the volume or the electrolyte composition of maintenance fluids. Children with renal insufficiency may be hyperkalemic or unable to excrete potassium and may not tolerate 20 mEq/L of KCl. In children with complicated pathophysiologic derangements, it may be necessary to adjust the electrolyte composition and rate of maintenance fluids empirically based on electrolyte measurements and assessment of fluid balance.

PEARLS FOR PRACTITIONERS

See *Fluids and Electrolytes: Pearls for Practitioners* at the end of this section.

CHAPTER 33

Dehydration and Replacement Therapy

REPLACEMENT THERAPY

There are three sources of normal water loss—the components of maintenance water (see Chapter 32): urine (60%), **insensible losses** from the skin and lungs (35%), and stool (5%) (Table 33.1). Sweating is not insensible and, in contrast to evaporative losses, sweat contains water and electrolytes.

A variety of clinical situations modify normal maintenance water balance (Table 33.2). Evaporative skin water losses can be higher in neonates, especially premature infants who are

TABLE 33.1 Components of Maintenance Water

Urine	60%
Insensible losses (skin and lungs)	35%
Stool	5%

TABLE 33.2 Adjustments in Maintenance Water

SOURCE	CAUSES OF INCREASED WATER NEEDS	CAUSES OF DECREASED WATER NEEDS
Skin	Radiant warmer Phototherapy Fever Sweat Burns Extensive exfoliative dermatitis*	Incubator (premature infants)
Lungs	Tachypnea Tracheostomy	Humidified ventilator
Gastrointestinal	Diarrhea Emesis Nasogastric suction	
Renal	Polyuria	Oliguria/anuria
Miscellaneous	Surgical drain Third space losses	Hypothyroidism

*Epidermolysis bullosa, toxic epidermal necrolysis.

TABLE 33.3 Adjusting Fluid Therapy for Gastrointestinal Losses

AVERAGE COMPOSITION	APPROACH TO REPLACEMENT
DIARRHEA	REPLACEMENT OF ONGOING STOOL LOSSES
Sodium: 55mEq/L	Solution: 5% dextrose in ¼ normal saline + 20mEq/L sodium bicarbonate + 20mEq/L potassium chloride Replace stool mL/mL every 1–6 hr
Potassium: 25mEq/L	
Bicarbonate: 15mEq/L	
GASTRIC FLUID	REPLACEMENT OF ONGOING GASTRIC LOSSES
Sodium: 60mEq/L	Solution: normal saline + 10mEq/L potassium chloride Replace output mL/mL every 1–6 hr
Potassium: 10mEq/L	
Chloride: 90mEq/L	

under radiant warmers or undergoing phototherapy. Burns can result in massive losses of water and electrolytes (see Chapter 44). Fever increases insensible losses. Tachypnea or a tracheostomy increases evaporative losses from the lungs.

The gastrointestinal tract is potentially a source of considerable water and electrolyte losses. A child who has large amounts of gastrointestinal losses should have these losses measured and replaced with an appropriate **replacement solution** (Table 33.3).

Urine output is normally the largest cause of *water* loss. Diseases such as renal failure and the syndrome of inappropriate antidiuretic hormone (SIADH) can lead to a decrease in

TABLE 33.4 Adjusting Fluid Therapy for Altered Renal Output

OLIGURIA/ANURIA	POLYURIA
Place the patient on insensible fluids (~1/3 maintenance)	Place the patient on insensible fluids (~1/3 maintenance)
Replace urine output mL/mL with half normal saline	Measure urine electrolytes
	Replace urine output mL/mL with a solution that is based on the measured urine electrolytes

urine volume. Maintenance fluids in a patient with oliguria or anuria may produce fluid overload if the cause is acute kidney injury. In contrast, other conditions produce an increase in urine volume; these include the polyuric phase of acute kidney injury, diabetes mellitus, and diabetes insipidus. When the urine output is excessive, the patient must receive more than standard maintenance fluids to prevent dehydration.

The approach to decreased or increased urine output is similar (Table 33.4). Insensible losses are replaced by a solution that is administered at a rate one third of the normal maintenance rate. Placing the **anuric** child on “insensibles” theoretically maintains an even fluid balance, with the caveat that one third of maintenance fluid is only an *estimate* of insensible losses. This rate may need to be adjusted based on monitoring of the patient’s weight and hydration status. An **oliguric** child needs to receive a urine replacement solution. Most children with **polyuria** (except for children with diabetes mellitus [see Chapter 171]) should be placed on insensible fluids plus urine replacement.

Output from surgical drains and chest tubes, when significant, should be measured and replaced. **Third space losses** manifest with edema and ascites and are due to a shift of fluid from the intravascular space into the interstitial space. Third space losses cannot be quantitated. Nonetheless, these losses can be large and lead to intravascular volume depletion, despite weight gain from edema or ascites. Replacement of third space fluid is empirical but should be anticipated in patients who are at risk, such as children who have sepsis, shock, burns, or abdominal surgery. Third space losses and chest tube output are isotonic and usually require replacement with an **isotonic fluid**, such as normal saline (NS) or Ringer lactate. Adjustments in the amount of replacement fluid for third space losses are based on continuing assessment of the patient’s intravascular volume status (heart rate, blood pressure, urine output).

DEHYDRATION

Dehydration, most often due to gastroenteritis, is common in children. The first step in caring for a dehydrated child is to assess the degree of dehydration. The degree of dehydration dictates the urgency of the situation and the volume of fluid needed for rehydration. Table 33.5 summarizes the clinical features that are present with varying degrees of dehydration.

A patient with **mild dehydration** has few clinical signs or symptoms. The history may describe decreased intake but more often increased fluid losses. An infant with **moderate dehydration** has demonstrable physical signs and symptoms. The patient needs fairly prompt intervention. A patient with **severe dehydration** is gravely ill. The decrease in blood pressure

TABLE 33.5 Assessment of Degree of Dehydration

	MILD	MODERATE	SEVERE
Infant	5%	10%	15%
Adolescent	3%	6%	9%
Infants and young children	Thirsty, alert; restless	Thirsty; restless or lethargic; irritable	Drowsy; limp, cold, sweaty, cyanotic extremities; may be comatose
Older children	Thirsty, alert	Thirsty, alert (usually)	Usually conscious (but at reduced level), apprehensive; cold, sweaty, cyanotic extremities; wrinkled skin on fingers and toes; muscle cramps
SIGNS AND SYMPTOMS			
Tachycardia	Absent	Present	Present
Palpable pulses	Present	Present (weak)	Decreased
Blood pressure	Normal	Orthostatic hypotension	Hypotension
Cutaneous perfusion	Normal	Normal	Reduced and mottled
Skin turgor	Normal	Slight reduction	Reduced
Fontanelle	Normal	Slightly depressed	Sunken
Mucous membrane	Moist	Dry	Very dry
Tears	Present	Present or absent	Absent
Respirations	Normal	Deep, may be rapid	Deep and rapid
Urine output	Normal	Oliguria	Anuria and severe oliguria

Data from World Health Organization.

indicates that vital organs may be receiving inadequate perfusion (shock) (see [Chapter 40](#)). Such a patient should receive immediate and aggressive intravenous (IV) therapy. Clinical assessment of dehydration is only an estimate; the patient must be continually re-evaluated during therapy. The degree of dehydration is underestimated in hypernatremic dehydration because the osmotically driven shift of water from the intracellular space to the extracellular space helps to preserve the intravascular volume.

Laboratory Evaluation

Serum blood urea nitrogen (BUN) and creatinine concentrations are useful in assessing a child with dehydration. Volume depletion without renal insufficiency may cause a disproportionate increase in the BUN, with little or no change in the creatinine concentration. This is secondary to increased passive reabsorption of urea in the proximal tubule caused by appropriate renal conservation of sodium and water. This increase in the BUN may be absent or blunted in a child with poor protein intake because urea production depends on protein degradation. Conversely, the BUN may be disproportionately increased in a child with increased urea production, as occurs in a child with a gastrointestinal bleed or a child who is receiving glucocorticoids. A significant elevation of the creatinine concentration suggests renal injury.

The urine specific gravity is usually elevated (≥ 1.025) in cases of significant dehydration but decreases after rehydration. With dehydration, a urinalysis may show hyaline and

granular casts, a few white blood cells and red blood cells, and 30–100 mg/dL of proteinuria. These findings usually are not associated with significant renal pathology, and they remit with therapy. Hemoconcentration from dehydration increases the hematocrit and hemoglobin.

Calculation of Fluid Deficit

A child with dehydration has lost water; there is usually a concurrent loss of sodium and potassium. The fluid deficit is the percentage of dehydration multiplied by the patient's weight (for a 10-kg child, 10% of 10 kg = 1 L deficit).

Approach to Dehydration

The child with dehydration requires acute intervention to ensure that there is adequate tissue perfusion (see [Chapter 40](#)). This resuscitation phase requires rapid restoration of the circulating intravascular volume, which should be done with an isotonic solution, such as NS or Ringer lactate. Blood is an appropriate fluid choice for a child with acute blood loss but is not always available at the start of fluid resuscitation. The child is given a fluid bolus, usually 20 mL/kg of the isotonic solution, over about 20 minutes. A child with severe dehydration may require multiple fluid boluses and may need to receive fluid at a faster rate. The initial resuscitation and rehydration is complete when signs of intravascular volume depletion resolve. The child typically becomes more alert and has a lower heart rate, normal blood pressure, and improved perfusion.

TABLE 33.6 Fluid Management of Dehydration

Restore intravascular volume
Normal saline: 20 mL/kg over 20 min
Repeat as needed
Rapid volume repletion: 20 mL/kg normal saline (maximum = 1 L) over 2 hr
Calculate 24-hr fluid needs: maintenance + deficit volume
Subtract isotonic fluid already administered from 24-hr fluid needs
Administer remaining volume over 24 hr using D5 normal saline + 20 mEq/L KCl
Replace ongoing losses as they occur

TABLE 33.7 Monitoring Therapy

Vital signs
Pulse
Blood pressure
Intake and output
Fluid balance
Urine output and specific gravity
Physical examination
Weight
Clinical signs of fluid volume depletion or overload
Electrolytes

With adequate intravascular volume, it is now appropriate to plan the fluid therapy for the next 24 hours (Table 33.6). To ensure that the intravascular volume is restored, the patient receives an additional 20 mL/kg bolus of isotonic fluid over 2 hours. The child's total fluid needs are added together (maintenance + deficit). The volume of isotonic fluids the patient has received as acute resuscitation is subtracted from this total. The remaining fluid volume is then administered over 24 hours. Potassium usually is not included in the IV fluids until the patient voids, unless significant hypokalemia is present. *Children with significant ongoing losses also need to receive an appropriate replacement solution.*

MONITORING AND ADJUSTING THERAPY

All calculations in fluid therapy are only approximations. Thus the patient needs to be monitored during treatment with therapy modifications based on the clinical situation (Table 33.7).

Hyponatremic dehydration occurs in children who have diarrhea and consume a hypotonic fluid (water or diluted formula). Volume depletion stimulates secretion of antidiuretic hormone, preventing the water excretion that should correct the hyponatremia. Some patients develop symptoms, predominantly neurologic, from the hyponatremia (see Chapter 35). Most patients with hyponatremic dehydration do well with

the same general approach outlined in Table 33.6. Overly rapid correction of hyponatremia (>10 mEq/L per 24 hours) should be avoided because of the remote risk of **central pontine myelinolysis**.

Hypernatremic dehydration is usually a consequence of an inability to take in fluid because of a lack of access, a poor thirst mechanism (neurologic impairment), intractable emesis, or anorexia. The movement of water from the intracellular space to the extracellular space during hypernatremic dehydration partially protects the intravascular volume losses. Urine output may be preserved longer, and there may be less tachycardia. Children with hypernatremic dehydration are often lethargic and irritable. Hypernatremia may cause fever, hypertonicity, hyperreflexia, and seizures. More severe neurologic symptoms may develop if cerebral bleeding or dural sinus thrombosis occurs.

Overly rapid treatment of hypernatremic dehydration may cause significant morbidity and mortality. **Idiogenic osmoles** are generated within the brain during the development of hypernatremia. Idiogenic osmoles increase the osmolality within the cells of the brain, providing protection against brain cell shrinkage secondary to movement of water out of cells into the hypertonic extracellular fluid. These idiogenic osmoles dissipate slowly during correction of hypernatremia. With rapid lowering of the extracellular osmolality during correction of hypernatremia, a new gradient may be created that causes water movement from the extracellular space into the cells of the brain, producing **cerebral edema**. Possible manifestations of the resultant cerebral edema include headache, altered mental status, seizures, and potentially lethal brain herniation.

To minimize the risk of cerebral edema during correction of hypernatremic dehydration, the serum sodium concentration should not decrease more than 12 mEq/L every 24 hours (Fig. 33.1). The choice and rate of fluid replacement are not nearly as important as vigilant monitoring of the serum sodium concentration and adjustment of the therapy based on the result (see Fig. 33.1). Nonetheless, the initial resuscitation-rehydration phase of therapy remains the same as for other types of dehydration.

Oral Rehydration

Mild to moderate dehydration from diarrhea of any cause can be treated effectively using a simple, oral rehydration solution (ORS) containing glucose and electrolytes (see Chapter 112). The ORS relies on the coupled transport of sodium and glucose in the intestine. Oral rehydration therapy has significantly reduced the morbidity and mortality from acute diarrhea but is underused in developed countries. It should be attempted for most patients with mild to moderate diarrheal dehydration. Oral rehydration therapy is less expensive than IV therapy and has a lower complication rate. IV therapy may still be required for patients with severe dehydration; patients with uncontrollable vomiting; patients unable to drink because of extreme fatigue, stupor, or coma; or patients with gastric or intestinal distention. Rapidly absorbed sublingual ondansetron may be used to treat vomiting, thus facilitating oral rehydration.

As a guideline for oral rehydration, 50 mL/kg of the ORS should be given within 4 hours to patients with mild dehydration, and 100 mL/kg should be given over 4 hours to patients

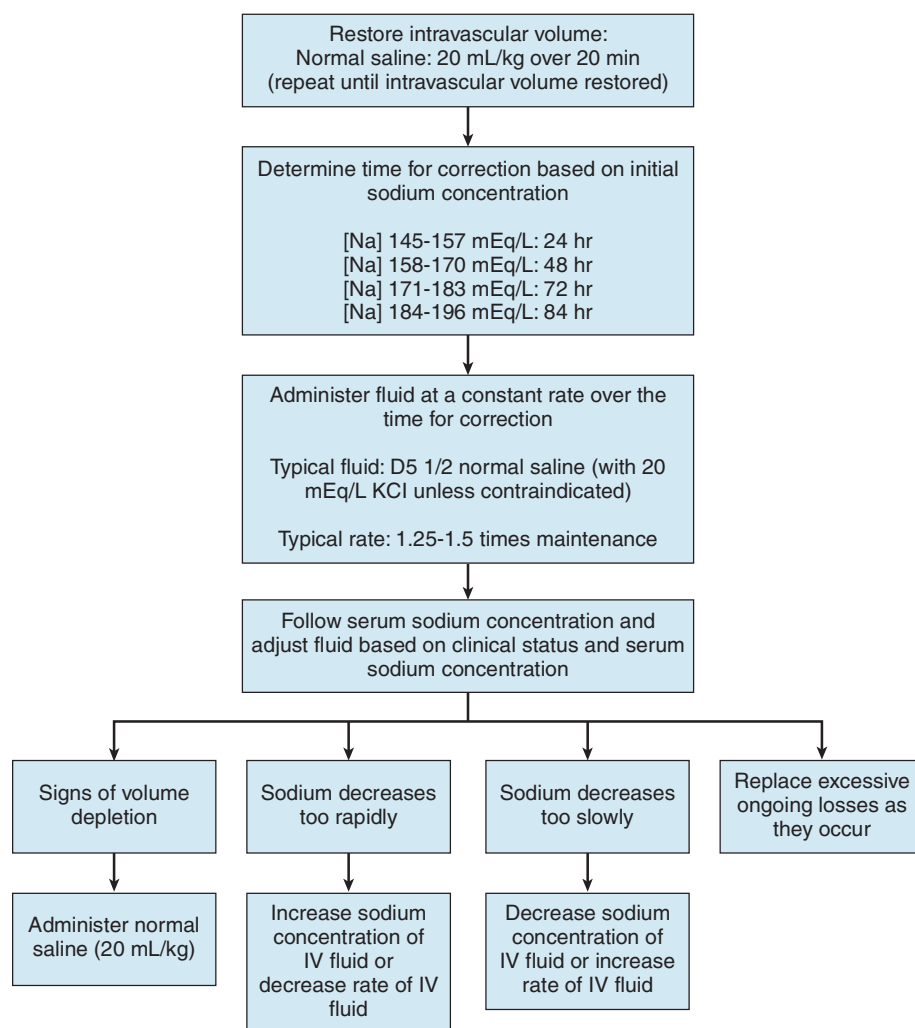


FIGURE 33.1 Strategy for correcting hypernatremic dehydration.

with moderate dehydration. Supplementary ORS is given to replace ongoing losses from diarrhea or emesis. An additional 10 mL/kg of ORS is given for each stool. Fluid intake should be decreased if the patient appears fully hydrated earlier than expected or develops periorbital edema. After rehydration, patients should resume their usual diet (breast milk, formula).

When rehydration is complete, maintenance therapy should be started, using 100 mL of ORS/kg in 24 hours until the diarrhea stops. Breast-feeding or formula-feeding should be maintained and not delayed for more than 24 hours. Patients with more severe diarrhea require continued supervision. The volume of ORS ingested should equal the volume of stool losses. If stool volume cannot be measured, an intake of 10–15 mL of ORS/kg per hour is appropriate.

PEARLS FOR PRACTITIONERS

See *Fluids and Electrolytes: Pearls for Practitioners* at the end of this section.