Table 33-6 Fluid Management of Dehydration
Restore intravascular volume
Normal saline: 20 mL/kg over 20 minutes
Repeat as needed
Rapid volume repletion: 20 mL/kg normal saline (maximum = 1 L) over 2 hours
Calculate 24-hour fluid needs: maintenance + deficit volume
Subtract isotonic fluid already administered from 24-hour fluid needs
Administer remaining volume over 24 hours using D5 ½ 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 depletion or overload
Electrolytes

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 need to receive an appropriate replacement solution.*

Monitoring and Adjusting Therapy

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Hypernatremia
Hyponatremia

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 (>12 mEq/L/24 hr) 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. 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 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 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 (Figure 33-1). The deficits in severe hypernatremic dehydration may need to be corrected over 2 to 4 days. The choice and rate of fluid are not nearly as important as vigilant monitoring of the serum sodium concentration and adjustment of the therapy based on the result (see Figure 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