SIADH is also associated with pneumonia, mechanical ventilation, meningitis, and other central nervous system disorders (trauma). Ectopic (tumor) production of ADH is rare in children. Infants can develop euvolemic hyponatremia as a result of excessive water consumption or inappropriately diluted formula.

In **hypervolemic hyponatremia,** there is an excess of total body water and sodium, although the increase in water is greater than the increase in sodium. In renal failure, there is an inability to excrete sodium or water; the urine sodium may be low or high, depending on the cause of the renal insufficiency. In other causes of hypervolemic hyponatremia, there is a decrease in the effective blood volume because of either third space fluid loss or poor cardiac output (see Chapter 145). In response to the low effective blood volume, ADH causes renal water retention, and the kidneys also retain sodium, leading to a low urine sodium concentration. The patient's serum sodium concentration decreases when water intake exceeds sodium intake, and ADH prevents the normal loss of excess water.

Clinical Manifestations

Hyponatremia causes a fall in the osmolality of the extracellular space. Because the intracellular space then has a higher osmolality, water moves from the extracellular space to the intracellular space to maintain osmotic equilibrium. The increase in intracellular water may cause cells to swell. **Brain cell swelling** is responsible for most of the symptoms of hyponatremia. Neurologic symptoms of hyponatremia include anorexia, nausea, emesis, malaise, lethargy, confusion, agitation, headache, seizures, coma, and decreased reflexes. Patients may develop hypothermia and Cheyne-Stokes respirations. Hyponatremia can cause muscle cramps and weakness. Symptoms are more severe when hyponatremia develops rapidly; chronic hyponatremia can be asymptomatic because of a compensatory decrease in brain cell osmolality, which limits cerebral swelling.

Treatment

Rapid correction of hyponatremia can produce **central pontine myelinolysis.** Avoiding more than a 12-mEq/L increase in the serum sodium every 24 hours is prudent, especially in chronic hyponatremia. Treatment of hypovolemic hyponatremia requires administration of IV fluids with sodium to provide maintenance requirements and deficit correction, as well as to replace ongoing losses (see Chapter 33). For children with SIADH, water restriction is the cornerstone of therapy. Children with hyponatremia secondary to hypothyroidism or cortisol deficiency need specific hormone replacement. **Acute water intoxication** rapidly self-corrects with transient restriction of water intake, which is followed by introduction of a normal diet. Treatment of hypervolemic hyponatremia centers on restriction of water and sodium intake, but disease-specific measures, such as dialysis in renal failure, also may be necessary.

Emergency treatment of **symptomatic hyponatremia,** such as seizures, uses IV hypertonic saline to increase the serum sodium concentration rapidly, which leads to a decrease in brain edema. One milliliter per kilogram of 3% sodium chloride increases the serum sodium by approximately 1 mEq/L. A child often improves after receiving 4 to 6 mL/kg of 3% sodium chloride.

HYPERNATREMIA

Decision-Making Algorithm
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Hypernatremia

Etiology

There are three basic mechanisms of hypernatremia (Fig. 35-2). **Sodium intoxication** is frequently iatrogenic in a hospital setting resulting from correction of metabolic acidosis with sodium bicarbonate. In hyperaldosteronism, there is renal retention of sodium and resultant hypertension; the hypernatremia is mild.

Hypernatremia resulting from water losses develops only if the patient does not have access to water or cannot drink adequately because of neurologic impairment, emesis, or anorexia. Infants are at high risk because of their inability to control their own water intake. Ineffective breastfeeding, often in a primiparous mother, can cause severe hypernatremic dehydration. High insensible losses of water are especially common in premature infants; the losses increase further as a result of radiant warmers or phototherapy for hyperbilirubinemia. Children with extrarenal causes of water loss have high levels of ADH and very concentrated urine.

Children with diabetes insipidus have inappropriately diluted urine. Hereditary **nephrogenic diabetes insipidus** causes massive urinary water losses. Because it is most commonly an X-linked disorder due to a mutation in the gene for the ADH receptor, it usually occurs in boys, who may have episodes of severe hypernatremic dehydration and failure to thrive. *Acquired nephrogenic diabetes insipidus* may be secondary to interstitial nephritis, sickle cell disease, hypercalcemia, hypokalemia, or medications (lithium or amphotericin). If the defect is due to **central diabetes insipidus,** urine output decreases and urine osmolality increases in response to administration of an ADH analog. Central causes of ADH deficiency include tumor, infarction, or trauma. There is no response to an ADH analog in a child with nephrogenic diabetes insipidus.

Diarrhea results in sodium and water depletion. Most children with gastroenteritis do not develop hypernatremia because they drink enough hypotonic fluid to compensate at least partially for stool water losses. Hypernatremia is most likely in a child with diarrhea who has inadequate intake because of emesis, lack of access to water, or anorexia. Some renal diseases, including obstructive uropathy, renal dysplasia, and juvenile nephronophthisis, can cause losses of sodium and water, potentially producing hypernatremia if the patient consumes insufficient water.

In situations with combined sodium and water deficits, analysis of the urine differentiates renal and nonrenal etiologies. When the losses are extrarenal, the kidney responds to volume depletion with low urine volume, a concentrated urine, and sodium retention (urine sodium <10 mEq/L). With renal causes, the urine volume is usually high, the urine is not maximally concentrated, and the urine sodium may be inappropriately elevated.