increase in the sodium concentration. Although water balance usually is regulated by osmolality, volume depletion stimulates thirst, ADH secretion, and renal conservation of water. In fact, volume depletion takes precedence over osmolality; volume depletion stimulates ADH secretion, even if a patient has hyponatremia.

The excretion of sodium by the kidney is not determined by the plasma osmolality. The patient's effective plasma volume regulates the amount of sodium in the urine through a variety of regulatory systems, including the renin-angiotensin-aldosterone system. In hyponatremia or hypernatremia, the underlying pathophysiology determines the urinary sodium concentration, not the serum sodium concentration.

HYPONATREMIA

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Hyponatremia

Etiology

Different mechanisms can cause hyponatremia (Fig. 35-1). **Pseudohyponatremia** is a laboratory artifact that is present when the plasma contains high concentrations of protein or lipid. It does not occur when a direct ion-selective electrode determines the sodium concentration, a technique that is increasingly used in clinical laboratories. In true hyponatremia, the *measured* osmolality is low, whereas it is normal in pseudohyponatremia. **Hyperosmolality**, resulting from mannitol infusion or hyperglycemia, causes a low serum sodium concentration because water moves down its osmotic gradient from the intracellular space into the extracellular space, diluting the sodium concentration. For every 100 mg/dL increment of the serum glucose, the serum sodium decreases by 1.6 mEq/L. Because the manifestations of hyponatremia are due to the low plasma osmolality, patients with hyponatremia caused by hyperosmolality do not have symptoms of hyponatremia and do not require correction of hyponatremia.

Classification of true hyponatremia is based on the patient's volume status (see Fig. 35-1). In **hypovolemic hyponatremia,** the child has lost sodium from the body. Water balance may be positive or negative, but there has been a higher net sodium loss than water loss; this is often due to oral or intravenous (IV) water intake, with water retention by the kidneys to compensate for the intravascular volume depletion. If the sodium loss is due to a nonrenal disease (e.g., diarrhea), the urine sodium concentration is very low, as the kidneys attempt to preserve the intravascular volume by conserving sodium. In renal diseases, the urine sodium is inappropriately elevated.

Patients with hyponatremia and no evidence of volume overload or volume depletion have **euvolemic hyponatremia.** These patients typically have an excess of total body water and a slight decrease in total body sodium. Some of these patients have an increase in weight, implying that they are volume overloaded. Nevertheless, they usually appear normal or have only subtle signs of fluid overload. In **syndrome of inappropriate ADH (SIADH),** there is secretion of ADH that is not inhibited by either low serum osmolality or expanded intravascular volume. Retention of water causes hyponatremia, and the expansion of the intravascular volume results in an increase in renal sodium excretion. Hyponatremia in hospitalized patients is often due to SIADH secondary to stress in the presence of hypotonic fluids.

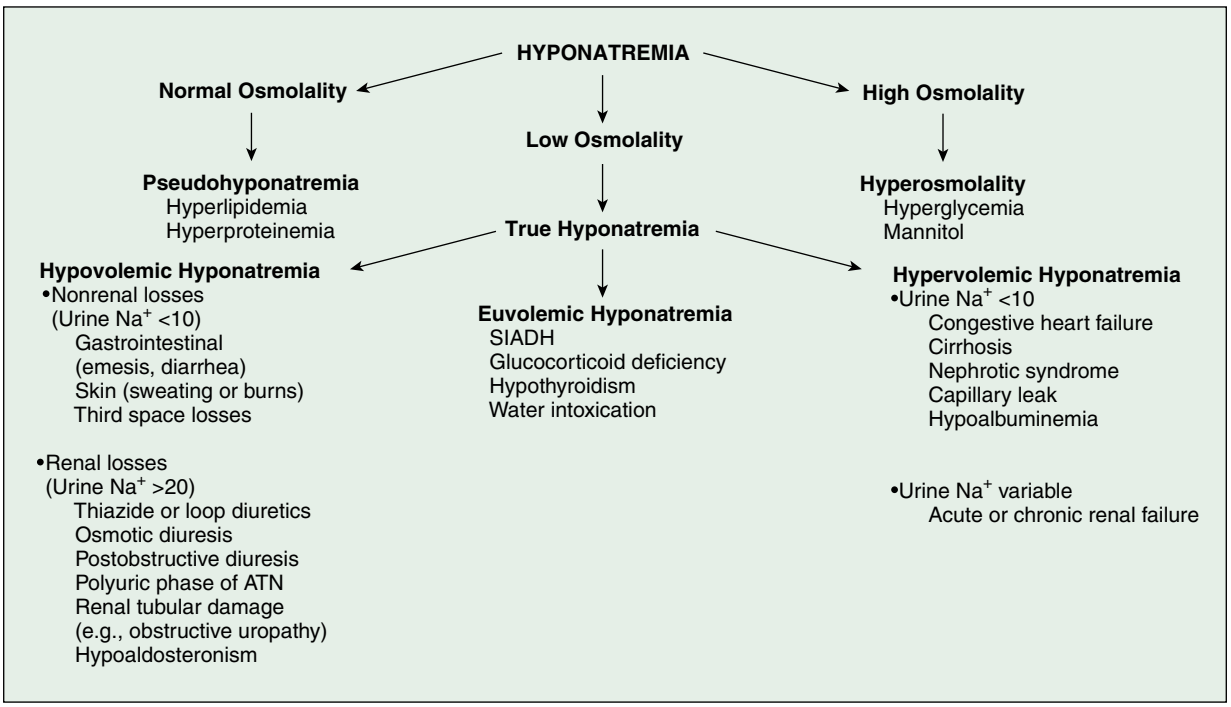

Figure 35-1 Differential diagnosis of hyponatremia. Assessment of hyponatremia is a three-step process: (1) Determine if the osmolality is low; if yes, the patient has true hyponatremia. (2) Evaluate the patient's volume status. (3) Determine the urine sodium concentration to help narrow the differential diagnosis. *ATN,* Acute tubular necrosis; *SIADH,* syndrome of inappropriate secretion of antidiuretic hormone.