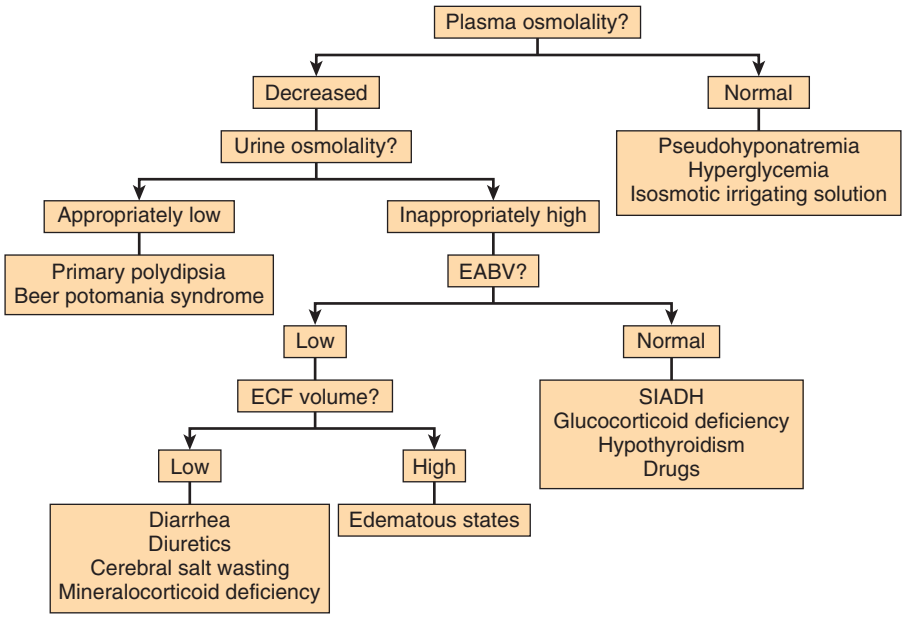

FIGURE 27-1 Approach to the patient with hyponatremia. Assessment of effective arterial blood volume (EABV) is key to understanding the mechanism of renal NaCl retention and whether it is primary or a response to a low EABV. The EABV is the arterial volume sensed by the kidney. If the kidney is working normally and is retaining NaCl, the EABV must be low, and if a normally functioning kidney is excreting large amounts of NaCl, the EABV is large. The physical examination is the most reliable way to assess EABV. Useful findings include the presence or absence of edema and orthostatic changes in blood pressure and pulse. Laboratory tests include collection of a spot urinary sample for sodium (Na^+), chloride (Cl^-), and creatinine to calculate the fractional excretion of Na^+ or fractional excretion of Cl^- using the following equations: FE_{Na} (%) = [(urine Na^+ × plasma creatinine)/(plasma Na^+ × urine creatinine)] × 100 and FE_{Cl} (%) = [(urine Cl^- × plasma creatinine)/(plasma Cl^- × urine creatinine)] × 100. If these parameters are low (<0.5-1%), a low EABV is indicated. Other findings suggesting a low EABV include an increase in the blood urea nitrogen (BUN)/creatinine ratio (>20 : 1), increased serum uric acid concentration (due to increased proximal tubular reabsorption), and increased hematocrit and serum albumin concentration due to hemoconcentration. SIADH, Syndrome of inappropriate secretion of antidiuretic hormone.

tonicity also stimulates thirst and AVP secretion, both of which contribute to water retention. As the plasma osmolality returns toward normal, the decline in the serum Na^+ level is 2.8 mEq/L for every 100-mg/dL rise in glucose. The net result is a normal plasma osmolality but a low serum Na^+ concentration.

The third cause of hyponatremia in the absence of a hypoosmolar state is the addition of an isosmotic or near-isosmotic, non-Na^+–containing fluid to the extracellular space. This situation typically occurs during transurethral resection of the prostate or during laparoscopic surgery, when large amounts of a nonconducting flushing solution containing glycine or sorbitol are reabsorbed systemically.

Despite these exceptions, hypotonic hyponatremia in most cases implies that water intake exceeds the ability of the kidney to excrete water. Because the normal kidney can excrete 20 to 30 L of water per day, hyponatremia with normal renal water excretion implies that the patient is drinking at least this volume of water. This condition is referred to as *primary polydipsia*. Urine osmolality is less than 100 mOsm/L in this setting. Hyponatremia associated with a maximally dilute urine can also result from more moderate fluid intake combined with extremely limited solute intake, a condition often referred to as *beer potomania syndrome*.

In the absence of primary polydipsia, hypotonic hyponatremia results when water intake exceeds the renal capacity for water excretion due to an inappropriately concentrated urine (>100 mOsm/L). The effective arterial blood volume (EABV) is defined in this setting. Decreased EABV causes baroreceptor stimulation of AVP secretion and decreases distal delivery of filtrate to the tip of the loop of Henle, accounting for the inability to maximally dilute the urine. If the EABV is low, ECF volume can be low in the volume-depleted patient (i.e., hypovolemic hyponatremia) or can be high in the edematous patient (i.e., hypervolemic hyponatremia). A normal EABV points to the euvolemic causes of hyponatremia (e.g., isovolemic hyponatremia).

Approximately two thirds of diagnosed hyponatremia cases are acquired in the hospital, where the common practices of monitoring daily fluid intake, patient weight, and Na^+ levels normally allow prompt diagnosis. Administration of hypotonic fluids in the postoperative period is a risk factor for acute iatrogenic hyponatremia, especially because AVP levels remain increased several days after surgical procedures. Iatrogenic cases can be prevented by close monitoring of electrolytes and urine output and by fluid restriction and avoidance of solutions with low-Na^+ content; this approach applies particularly to elderly patients.

In neurosurgical patients, the syndrome of inappropriate antidiuretic hormone (SIADH) secretion and cerebral salt wasting are two causes of hyponatremia. Differentiating these disorders can be challenging because there is considerable overlap in the clinical presentation. The primary distinction lies in the EABV assessment. SIADH is a volume-expanded state due to AVP-mediated renal water retention. Cerebral salt wasting is characterized by a contracted EABV resulting from renal salt wasting. Making an accurate diagnosis is important because