therapy for these conditions is different. Vigorous salt replacement is indicated for patients with cerebral salt wasting, and fluid restriction is the treatment of choice for patients with SIADH.

Common causes of hyponatremia outside the hospital setting include overhydration, diarrhea, vomiting, central nervous system infection, extreme exercise, liver failure, renal failure, congestive heart failure, drugs, SIADH, and combinations of these and other factors. Thiazide diuretics are the most common cause of drug-induced hyponatremia. Hyponatremia typically develops in the first 2 weeks of drug initiation and is most likely to occur in elderly women and during the summer months because of the increased ingestion of hypotonic fluids when it is hot. Concomitant use of nonsteroidal anti-inflammatory drugs (NSAIDs) and selective serotonin reuptake inhibitors (SSRIs) can further increase the risk of thiazide-induced hyponatremia.

Treatment

Symptoms of hyponatremia include nausea and malaise, which can be followed by headache, lethargy, muscle cramps, disorientation, restlessness, and obtundation. When treating a patient with hyponatremia, the Na^+ concentration should be raised at the rate at which it fell. In patients with chronic hyponatremia (>48 hours' duration), the serum Na^+ concentration has fallen slowly. Neurologic symptoms are minimal, brain size is normal, and the number of intracellular osmoles is decreased. Sudden return of ECF osmolality to normal values produces cell shrinkage and possibly precipitates osmotic demyelination. This complication can be avoided by limiting correction to between 10 and 12 mEq/L in 24 hours and to less than 18 mEq/L in 48 hours. In a patient whose serum Na^+ concentration has decreased rapidly (<48 hours), neurologic symptoms are common, and cerebral edema occurs. In this case, there has not been sufficient time to remove osmoles from the brain, and rapid return to normal ECF osmolality merely returns brain size to normal.

Development of hyponatremia in the outpatient setting is commonly chronic in duration and should be corrected slowly. In contrast, hyponatremia of short duration is more likely to be encountered in hospitalized patients receiving intravenous free water. Use of the illicit drug ecstasy, exercise-induced hyponatremia, or primary polydipsia can cause patients to develop acute hyponatremia, and if symptomatic, they may require rapid correction.

HYPERNATREMIA

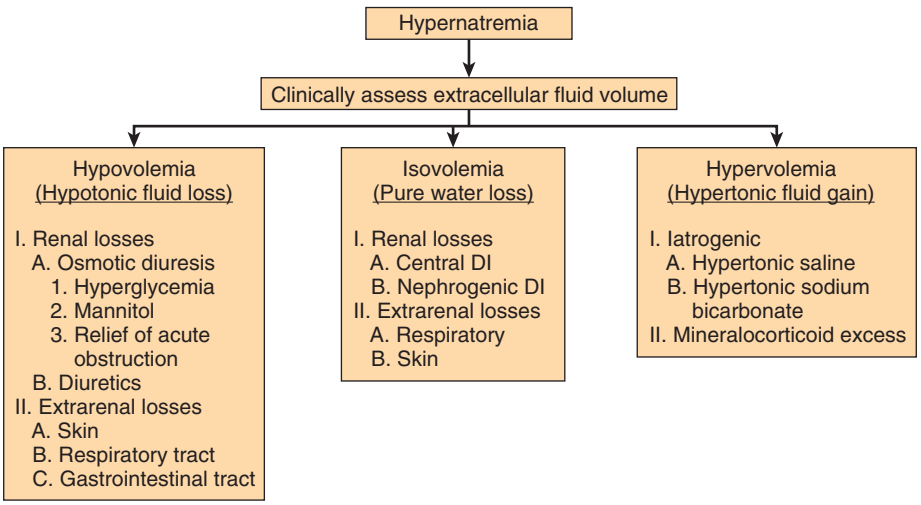
Definition

Hypernatremia is a relatively common problem, particularly among the elderly and critically ill. Hypernatremia always indicates hypertonicity and is associated with shrinkage of cells. It is an independent risk factor for mortality in the intensive care unit setting.

The initial approach to any patient with hypernatremia is to determine why there has been inadequate intake of water (Fig. 27-2). Hypernatremia is rare in conscious patients who have free access to water because of the extreme sensitivity of the thirst mechanism. Usually, there is inadequate water intake due to an alteration in the level of consciousness. Patients become unaware of thirst or cannot adequately communicate the need for water, or there is restricted access to water. Rarely, there is a specific lesion of the thirst center. A reduced sensation of thirst occurs in otherwise normal individuals as a feature of increasing age.

The next step is to search for accelerated water loss or Na^+ gain, both of which increase the likelihood of a patient developing hypernatremia. This can best be accomplished by clinical assessment of the EABV. Hypovolemic hypernatremia results from fluid losses in which the Na^+ concentration is less than the plasma concentration. Hypervolemic hypernatremia can result from iatrogenic administration of hypertonic NaCl or hypertonic sodium bicarbonate ($NaHCO_3$) or from mineralocorticoid excess.

Pure water loss, whether from mucocutaneous routes or from the kidneys, causes isovolemic hypernatremia. Because two thirds of pure water loss is sustained from within cells, patients do not become clinically volume depleted unless the water deficit becomes substantial. Insensible losses from the respiratory tract or skin result in concentrated urine. Inappropriate water loss by the kidney, whether from central or nephrogenic diabetes



*All are associated with impairment of thirst or access to water.

FIGURE 27-2 Approach to the patient with hypernatremia. DI, Diabetes insipidus.