

Low Solute Intake and Hyponatremia Hyponatremia can occasionally occur in patients with a very low intake of dietary solutes. Classically, this occurs in alcoholics whose sole nutrient is beer, hence the diagnostic label of *beer potomania*; beer is very low in protein and salt content, containing only 1–2 mM of Na⁺. The syndrome has also been described in nonalcoholic patients with highly restricted solute intake due to nutrient-restricted diets, e.g., extreme vegetarian diets. Patients with hyponatremia due to low solute intake typically present with a very low urine osmolality (<100–200 mOsm/kg) with a urine Na⁺ concentration that is <10–20 mM. The fundamental abnormality is the inadequate dietary intake of solutes; the reduced urinary solute excretion limits water excretion such that hyponatremia ensues after relatively modest polydipsia. AVP levels have not been reported in patients with beer potomania but are expected to be suppressed or rapidly suppressible with saline hydration; this fits with the overly rapid correction in plasma Na⁺ concentration that can be seen with saline hydration. Resumption of a normal diet and/or saline hydration will also correct the causative deficit in urinary solute excretion, such that patients with beer potomania typically correct their plasma Na⁺ concentration promptly after admission to the hospital.

Clinical Features of Hyponatremia Hyponatremia induces generalized cellular swelling, a consequence of water movement down the osmotic gradient from the hypotonic ECF to the ICF. The symptoms of hyponatremia are primarily neurologic, reflecting the development of cerebral edema within a rigid skull. The initial CNS response to acute hyponatremia is an increase in interstitial pressure, leading to shunting of ECF and solutes from the interstitial space into the cerebrospinal fluid and then on into the systemic circulation. This is accompanied by an efflux of the major intracellular ions, Na⁺, K⁺, and Cl⁻, from brain cells. Acute hyponatremic encephalopathy ensues when these volume regulatory mechanisms are overwhelmed by a rapid decrease in tonicity, resulting in acute cerebral edema. Early symptoms can include nausea, headache, and vomiting. However, severe complications can rapidly evolve, including seizure activity, brainstem herniation, coma, and death. A