

patients with severe hypovolemic shock may develop lactic acidosis with an elevated anion gap.

The neurohumoral response to hypovolemia stimulates an increase in renal tubular Na^+ and water reabsorption. Therefore, the urine Na^+ concentration is typically $<20 \text{ mM}$ in nonrenal causes of hypovolemia, with a urine osmolality of $>450 \text{ mOsm/kg}$. The reduction in both GFR and distal tubular Na^+ delivery may cause a defect in renal potassium excretion, with an increase in plasma K^+ concentration. Of note, patients with hypovolemia and a hypochloremic alkalosis due to vomiting, diarrhea, or diuretics will typically have a urine Na^+ concentration $>20 \text{ mM}$ and urine pH of >7.0 , due to the increase in filtered HCO_3^- ; the urine Cl^- concentration in this setting is a more accurate indicator of volume status, with a level $<25 \text{ mM}$ suggestive of hypovolemia. The urine Na^+ concentration is often $>20 \text{ mM}$ in patients with *renal* causes of hypovolemia, such as acute tubular necrosis; similarly, patients with DI will have an inappropriately dilute urine.

TREATMENT

Hypovolemia

The therapeutic goals in hypovolemia are to restore normovolemia and replace ongoing fluid losses. Mild hypovolemia can usually be treated with oral hydration and resumption of a normal maintenance diet. More severe hypovolemia requires intravenous hydration, tailoring the choice of solution to the underlying pathophysiology. Isotonic, “normal” saline (0.9% NaCl, 154 mM Na^+) is the most appropriate resuscitation fluid for normonatremic or hyponatremic patients with severe hypovolemia; colloid solutions such as intravenous albumin are not demonstrably superior for this purpose. Hypernatremic patients should receive a hypotonic solution, 5% dextrose if there has only been water loss (as in DI), or hypotonic saline (1/2 or 1/4 normal saline) if there has been water and $\text{Na}^+\text{-Cl}^-$ loss; changes in free water administration should be made if necessary, based on frequent measuring of serum chemistries. Patients with bicarbonate loss and metabolic acidosis, as occur