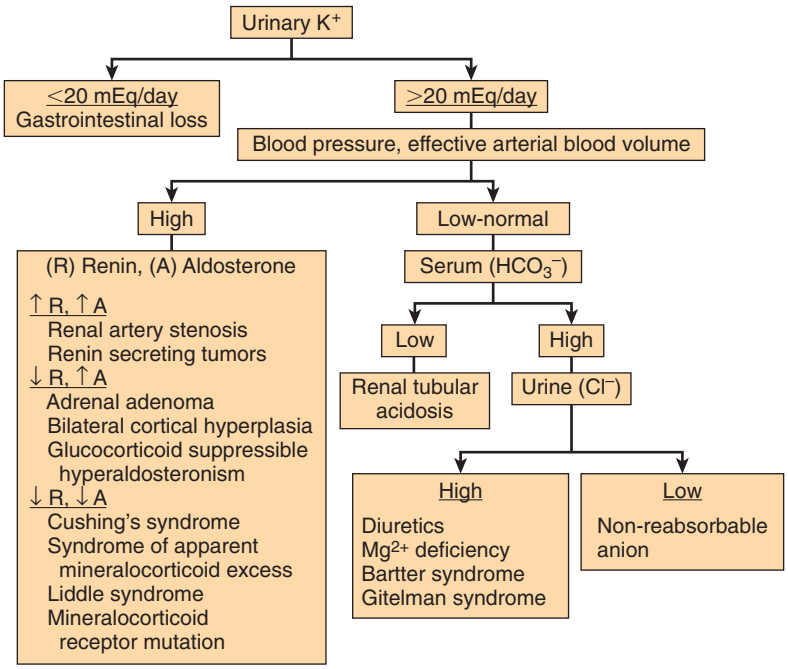

FIGURE 27-5 Approach to the hypokalemic patient. HCO_3^-, Bicarbonate; ↓, decreased; ↑, increased.

Increases in distal Na^+ delivery most frequently result from diuretics that act proximal to the cortical collecting duct. Increased delivery can also be the result of non-reabsorbed anions such as bicarbonate (HCO_3^-), as occurs with active vomiting, or of type II proximal renal tubular acidosis. Ketoanions (i.e., β-hydroxybutyrate and acetoacetate) and the Na^+ salts of penicillin are other examples. The inability to reabsorb these anions in the proximal tubule results in increased delivery of Na^+ to the distal nephron. Because these anions also escape reabsorption in the distal nephron, a more lumen-negative transepithelial voltage develops, and the driving force for K^+ excretion into the tubular fluid is enhanced. Disorders of hypokalemia due to primary increases in distal Na^+ delivery can best be categorized by the finding of metabolic acidosis or metabolic alkalosis.

Clinical Presentation

The most important clinical manifestations of hypokalemia occur in the neuromuscular system. A low serum K^+ concentration leads to cell hyperpolarization, which impedes impulse conduction and muscle contraction. Typically, a flaccid paralysis develops in the hands and feet that moves proximally to include the trunk and respiratory muscles. Death may occur from respiratory insufficiency. Myopathy may also occur, which in its most severe form can lead to frank rhabdomyolysis (i.e., muscle cell lysis) and renal failure. Hypokalemia can also lead to smooth muscle dysfunction, including paralytic ileus.

Changes in the electrocardiogram (ECG) include ST depression, T-wave flattening, and an increase in the amplitude of the U wave. Patients treated with cardiac glycosides are at increased risk for premature ventricular contractions and for supraventricular and ventricular tachyarrhythmia when hypokalemic.

Hypokalemia causes a renal concentrating defect due to a decrease in the medullary gradient and resistance of the cortical collecting tubule to AVP. This leads to polyuria and polydipsia. Prolonged hypokalemia can also lead to tubulointerstitial nephritis and renal failure. Because insulin release is regulated partially by the serum K^+ concentration, hypokalemia can lead to glucose intolerance.

Treatment

The serum K^+ levels can sometimes be misleading about the degree of deficit because a normal or increased K^+ level can occur with significant total body K^+ depletion. In the absence of significant K^+ shifts, a decline in the serum K^+ level from 4 to 3 mEq/L usually is associated with a deficit of 300 to 400 mEq of intracellular K^+ per 70 kg of body weight. A serum K^+ concentration of 2 mEq/L reflects a deficit of roughly 600 mEq. Along with these guidelines, the serum K^+ level should be monitored frequently during replacement therapy.

Supplemental K^+ can be given orally or intravenously as the potassium chloride (KCl) salt. Potassium bicarbonate or citrate can be given if there is concomitant metabolic acidosis. The safest way to administer KCl is orally. KCl can be given in doses of 100 to 150 mEq/day. Liquid KCl is bitter tasting and, like the tablet, can irritate the gastric mucosa. The microencapsulated or wax-matrix forms of KCl are better tolerated.

Intravenous administration of K^+ may be necessary if the patient cannot take oral medications or the K^+ deficit is large and is causing cardiac arrhythmias, respiratory paralysis, or rhabdomyolysis. Intravenous KCl should be given at a maximum rate of 20 mEq/hour and maximum concentration of 40 mEq/L. Higher concentrations result in phlebitis. Replacement of KCl in dextrose-containing solutions can lower the serum K^+ further due to insulin release. Saline solutions are preferred.

Depending on the specific cause, additional therapy for chronic hypokalemia involves the use of K^+-sparing diuretics such as amiloride, spironolactone, or triamterene. Caution is