Table 36-1 Causes of Hypokalemia

Spurious	With metabolic alkalosis
High white blood cell count	Low urine chloride
Transcellular shifts	Emesis/nasogastric suction
Alkalemia	Pyloric stenosis
Insulin	Chloride-losing diarrhea
β-Adrenergic agonists	Cystic fibrosis
Drugs/toxins (theophylline, barium, toluene)	Low-chloride formula
Hypokalemic periodic paralysis	Posthypercapnia
Refeeding syndrome	Previous loop or thiazide diuretic use
Decreased intake	High urine chloride and normal blood pressure
Extrarenal losses	Gitelman syndrome
Diarrhea	Bartter syndrome
Laxative abuse	Loop and thiazide diuretics
Sweating	High urine chloride and high blood pressure
Renal losses	Adrenal adenoma or hyperplasia
With metabolic acidosis	Glucocorticoid-remediable aldosteronism
Distal RTA	Renovascular disease
Proximal RTA	Renin-secreting tumor
Ureterosigmoidostomy	17α-Hydroxylase deficiency
Diabetic ketoacidosis	11β-Hydroxylase deficiency
Without specific acid-base disturbance	Cushing syndrome
Tubular toxins (amphotericin, cisplatin, aminoglycosides)	11β-Hydroxysteroid dehydrogenase deficiency
Interstitial nephritis	Licorice ingestion
Diuretic phase of acute tubular necrosis	Liddle syndrome
Postobstructive diuresis	
Hypomagnesemia	
High urine anions (e.g., penicillin or penicillin derivatives)	

RTA, renal tubular acidosis.

is more cautious if renal function is decreased because of the kidney's limited ability to excrete excessive potassium. The plasma potassium level does not always provide an accurate estimation of the total body potassium deficit because there may be shifts of potassium from the intracellular space to the plasma. Clinically, this shift occurs most commonly with metabolic acidosis and as a result of the insulin deficiency of diabetic ketoacidosis; the plasma potassium underestimates the degree of total body potassium depletion. When these problems are corrected, potassium moves into the intracellular space, and these patients require more potassium supplementation to correct the hypokalemia. Patients who have ongoing losses of potassium need correction of the deficit and replacement of the ongoing losses.

Because of the risk of hyperkalemia, intravenous (IV) potassium should be used cautiously. Oral potassium is safer in nonurgent situations. The dose of IV potassium is 0.5 to 1 mEq/kg, usually given over 1 hour. The adult maximum dose is 40 mEq. Conservative dosing is generally preferred. For patients with excessive urinary losses, potassium-sparing diuretics are effective. When hypokalemia, metabolic alkalosis, and volume depletion are present, restoration of intravascular volume decreases urinary potassium losses.

HYPERKALEMIA

Decision-Making Algorithm
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Hyperkalemia

Etiology

Three basic mechanisms cause hyperkalemia (Table 36-2). In the individual patient, the etiology is sometimes multifactorial. **Factitious hyperkalemia** is usually due to hemolysis during phlebotomy, but it can be the result of prolonged tourniquet application or fist clenching, which causes local potassium release from muscle. Falsely elevated serum potassium levels can occur when serum levels are measured in patients with markedly elevated white blood cell or platelet counts; a promptly analyzed plasma sample usually provides an accurate result.

Because of the kidney's ability to excrete potassium, it is unusual for excessive intake, by itself, to cause hyperkalemia. This mechanism can occur in a patient who is receiving large quantities of IV or oral potassium for excessive losses that are no longer present. Frequent or rapid blood transfusions can increase the potassium level acutely secondary to the high