Hypokalemia: A Quick Reference

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Potassium is the main intracellular cation. Intracellular potassium represents
approximately 95% of the total body potassium.

Despite low extracellular content, extracellular potassium concentration is
maintained within narrow limits to avoid the life-threatening effects of hypoka-
lemia or hyperkalemia.

Potassium maintains resting membrane potential. Changes in potassium con-
centration are associated with a decrease in the excitability of membranes,
especially in cardiac and skeletal muscles.

Changes in serum potassium concentration attributable to acid-base imbal-
ance are variable and mostly clinically irrelevant.

ANALYSIS

• Indications: Serum potassium should be measured in patients at high risk to
have or develop hypokalemia. This includes dogs and cats with chronic or fre-
quent vomiting, diarrhea, marked polyuria, muscle weakness, and unex-
plained cardiac arrhythmias and those receiving insulin, total parenteral
nutrition, and diuretics.

• Typical reference range: The mean normal value expected for dogs and cats
is 4.5 mEq/L (range: 3.5–5.5 mEq/L) but may vary slightly among
laboratories.

• Fractional excretion potassium (FE钾) can be used to rule out the kidneys as
the source of potassium losses. FE钾 is calculated as follows:

\[ FE_钾 = \frac{U_钾/S_钾}{U_肌酐/S_肌酐} \times 100 \]

where \( U_钾 \) is the urine concentration of potassium (mEq/L), \( S_钾 \) is the serum
concentration of potassium (mEq/L), \( U_肌酐 \) is the urine concentration of cre-
atinine (mg/dL), and \( S_肌酐 \) is the serum concentration of creatinine (mg/dL).

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**Box 1: Principal causes of hypokalemia**

Pseudohypokalemia (infrequent and rarely causing significant change)

Increased loss (most common and important category)
- Gastrointestinal (FEK <6%)
  - Vomiting of gastric contents (common and important)
  - Diarrhea (common and important)
- Urinary (FEK >20%)
  - Chronic renal failure in cats (common and important)
  - Diet-induced hypokalemic nephropathy in cats (important)
  - Postobstructive diuresis (common and important)
  - Inappropriate fluid therapy (especially with inadequate potassium supplementation) (common and important)
- Diuresis caused by diabetes mellitus/ketoacidosis (common and important)
  - Dialysis (uncommon)

Drugs
- Loop diuretics (eg, furosemide) (common and important)
- Thiazide diuretics (eg, chlorothiazide, hydrochlorothiazide)
- Amphotericin B
- Penicillins (rare)
  - Albuterol overdose (rare)
- Distal (type I) renal tubular acidosis (RTA) (rare)
- Proximal (type II) RTA after NaHCO₃ treatment (rare)
- Mineralocorticoid excess (rare)
- Hyperadrenocorticism (mild changes)
- Primary hyperaldosteronism (ie, adenoma, hyperplasia)
- Translocation (extracellular fluid → intracellular fluid)
- Glucose-containing fluids with or without insulin (common and important)
- Total parenteral nutrition solutions (uncommon but important)
- Catecholamines (rare)
- Hypokalemic periodic paralysis (Burmese cats) (rare)

Decreased intake
- Unlikely to cause hypokalemia by itself unless diet is severely deficient
  - Administration of potassium-free fluids (eg, 0.9% sodium chloride [NaCl], 5% dextrose in water)

The FEK should be less than 6% for nonrenal sources of potassium loss. Increased values are difficult to interpret and do not necessarily mean that the kidneys are the source of potassium losses.

Danger values: Concentrations less than 3.5 mEq/L may be associated with clinical signs. A potassium serum concentration less than 3.0 mEq/L may result in muscle weakness, cardiac arrhythmias, and polyuria, whereas rhabdomyolysis may be observed when potassium serum concentrations decrease to less than 2.0 mEq/L. Respiratory muscle paralysis may occur if potassium decreases to less than 2.0 mEq/L.

Artifacts: Pseudohypokalemia, an in vitro decrease in potassium concentration, is uncommon and rarely leads to substantial changes in potassium concentration.

Drug effect: Hypokalemia may occur in patients receiving diuretics, insulin, mineralocorticoids, potassium-free fluids, and sodium bicarbonate. The intracellular shift of potassium induced by sodium bicarbonate is not caused by an increase in pH.

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**Fig. 1.** Algorithm for the clinical approach to hypokalemia. (From DiBartola SP, de Morais HA. Disorders of potassium: hypokalemia and hyperkalemia. In: DiBartola SP, editor. Fluid, electrolyte, and acid-base disorders. 3rd edition. St. Louis (MO): Elsevier; 2006. p. 102; with permission.)
HYPOKALEMIA

- Causes: Hypokalemia may result from the causes listed in Box 1.
  - Increased potassium loss
    - From gastrointestinal tract (FEK <6%) in patients with vomiting or diarrhea
    - From the kidneys (FEK >20%) in patients that have renal failure or polyuria or in those receiving diuretics or potassium-free fluids
    - Most hypokalemic patients have increased potassium losses.
    - Potassium translocation from extracellular fluid to intracellular fluid in patients receiving insulin or glucose-containing fluids
  - Decreased intake is unlikely to cause hypokalemia unless the diet is severely deficient or if potassium-free fluids are being given intravenously.

- Signs: Clinical signs vary with the severity and acuteness of $K^+$ depletion. Anorexia, muscular weakness, and polyuria or polydipsia are the most common signs. Generalized weakness may be observed in dogs and cats, whereas flaccid ventroflexion of the neck, forelimb hypermetria, and a broad-based hind limb stance are seen in cats with polymyopathy. Hypokalemia can lead to ventricular or supraventricular tachyarrhythmias. Hypokalemia does not cause metabolic alkalosis in dogs or cats, however.

- Stepwise approach: An algorithm for the differential diagnosis of hypokalemia is presented in Fig. 1.

Further Readings