

## 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 hypokalemia or hyperkalemia.
- Potassium maintains resting membrane potential. Changes in potassium concentration 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 imbalance 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 frequent vomiting, diarrhea, marked polyuria, muscle weakness, and unexplained 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 ( $FE_K$ ) can be used to rule out the kidneys as the source of potassium losses.  $FE_K$  is calculated as follows:

$$FE_K = \frac{U_k/S_k}{U_{Cr}/S_{Cr}} \times 100$$

where  $U_K$  is the urine concentration of potassium (mEq/L),  $S_K$  is the serum concentration of potassium (mEq/L),  $U_{Cr}$  is the urine concentration of creatinine (mg/dL), and  $S_{Cr}$  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 ( $FE_K < 6\%$ )

Vomiting of gastric contents (common and important)

Diarrhea (common and important)

Urinary ( $FE_K > 20\%$ )

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  $\text{NaHCO}_3$  treatment (rare)

Mineralocorticoid excess (rare)

Hyperadrenocorticism (mild changes)

Primary hyperaldosteronism (ie, adenoma, hyperplasia)

Translocation (extracellular fluid  $\rightarrow$  intracellular fluid)

Glucose-containing fluids with or without insulin (common and important)

Total parenteral nutrition solutions (uncommon but important)

Catecholamines (rare)

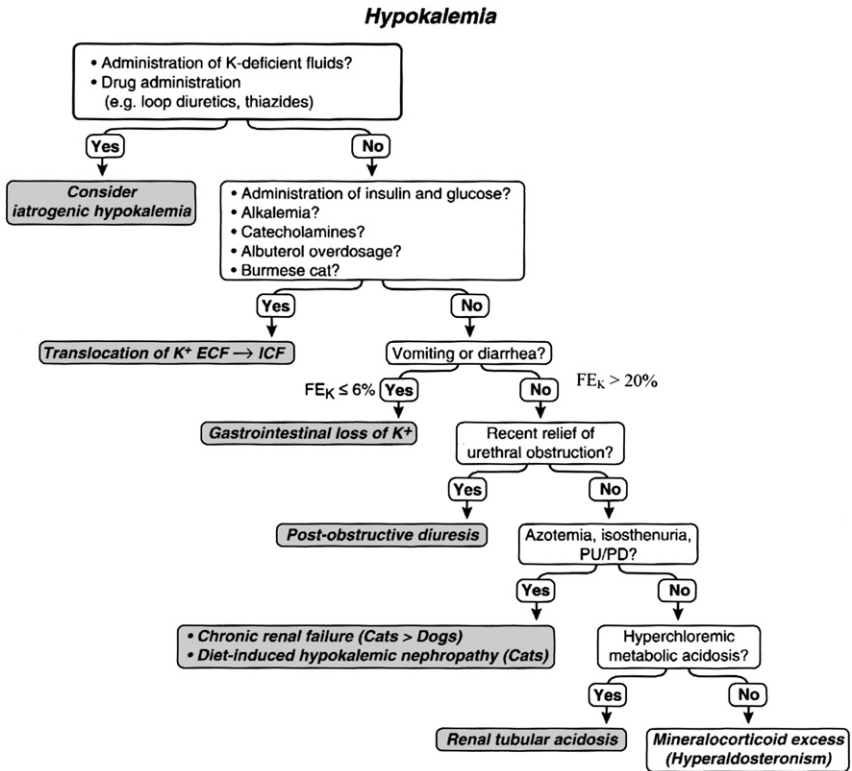
Hypokalemic periodic paralysis (Burmese cats) (rare)

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)

*Adapted from DiBartola SP, de Morais HA. Disorders of potassium: hypokalemia and hyperkalemia. In: DiBartola SP, editor. Fluid therapy in small animal practice. 2nd edition. Philadelphia: WB Saunders; 2000. p. 93; with permission.*



**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.)

- The  $FE_K$  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.

## HYPOKALEMIA

- Causes: Hypokalemia may result from the causes listed in Box 1.
  - Increased potassium loss
    - From gastrointestinal tract ( $FE_K < 6\%$ ) in patients with vomiting or diarrhea
    - From the kidneys ( $FE_K > 20\%$ ) 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

- 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. 91–121.
- DiBartola SP, Green RA, de Morais HA, et al. Electrolyte and acid base abnormalities. In: Willard MD, Tvedten H, editors. Small animal clinical diagnosis by laboratory methods. 4th edition. St. Louis (MO): Saunders; 2004. p. 117–34.