

A Quick Reference on Hypokalemia

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KEYWORDS

• Hypokalemia • Dog • Cat • Fractional excretion • Potassium

KEY POINTS

- Hypokalemia is usually caused by excessive losses of potassium in the urine or in the gastrointestinal tract.
- Iatrogenic hypokalemia is common with use of diuretics and insulin or glucose-containing fluids.
- Clinical signs are observed when hypokalemia is moderate or severe; life-threatening cardiac arrhythmias may occur.

INTRODUCTION

- In mammalian cells, potassium is the major intracellular cation, representing approximately 90% to 95% of the total body potassium. The remaining 5% to 10% is extracellular, being close to three-quarters in bone and one-quarter in plasma and interstitial fluid.¹⁻³
- One of the most important functions of intracellular potassium is generation of normal resting cell membrane potential. Hypokalemia hyperpolarizes the cell decreasing its membrane excitability. This effect is noticed mostly in cardiac and skeletal muscles.¹⁻³
- Potassium is removed primarily by the kidneys; 90% to 95% is excreted in urine.¹⁻³
- Hypokalemia is more common than hyperkalemia in dogs and cats.^{1,4-6}

ANALYSIS

- Reference values for dogs and cats range from 4.0 to 5.5 mEq/L and may vary slightly among laboratories owing to the methodology and type of sample (plasma, blood, or serum).^{1,4,7}

The authors have nothing to disclose.

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- Potassium concentration in plasma or blood is lower (≤ 0.5 mEq/L) than in serum because potassium is released from platelets during the clotting process.^{1,4,7}
- Methodology: most laboratories and point-of-care testing devices use ion-selective electrode methods; unlike with flame photometry, hyperlipemia and hyperproteinemia do not interfere with potassium measurement when ion-selective electrode methods are used.^{1,4}
- Artifacts: pseudohypokalemia is an in vitro decrease in potassium concentration; it occurs infrequently in samples with hyperlipemia or hyperproteinemia if the potassium concentration is measured by flame photometry.^{1,4}
- Indications: serum potassium should be measured in patients developing chronic or frequent vomiting or diarrhea, marked polyuria, muscle weakness, or unexpected cardiac arrhythmias, as well as in those being treated with insulin, diuretics, or receiving total parenteral nutrition.¹
- Danger values: clinical signs are associated with potassium serum concentrations of less than 3.5 mEq/L (moderate hypokalemia is defined as 2.5–3.0 mEq/L and severe as < 2.5 mEq/L). Muscle weakness, cardiac arrhythmias, and polyuria may occur when serum potassium is less than 3.0 mEq/L, whereas rhabdomyolysis and respiratory muscle paralysis may be observed if potassium concentration is below 2.0 mEq/L.^{1,4}
- Hypokalemia does not cause metabolic alkalosis in dogs and cats.¹
- Drug effect/iatrogenic: hypokalemia may occur in patients receiving diuretics, insulin, mineralocorticoid analogs (eg, fludrocortisone), potassium-free fluids, and sodium bicarbonate.¹
- Fractional urinary excretion of potassium (FE_K) can be used to help localize the source of potassium loss, whether renal loss or not.¹ It is calculated as:

$$FE_K = \frac{U_K/S_K}{U_{cr}/S_{cr}} \times 100 (\%)$$

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) 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.

- Complementary examinations: an electrocardiogram helps to characterize arrhythmias associated with hypokalemia and monitor its resolution after potassium supplementation.

CAUSES AND CLINICAL SIGNS

- **Tables 1** and **2** list potential causes of hypokalemia in dogs and cats.
 - The main mechanism leading to hypokalemia is increase in potassium losses through the gastrointestinal tract (vomiting or diarrhea) or the kidneys. Kidney losses can occur owing to intrinsic renal disease (impairment of tubular potassium absorption, tubular acidosis, increased losses through polyuria) or from drugs that affect sodium, chloride, and potassium handling in renal tubular cells (diuretics and potassium-free fluids).^{1–3,5}
 - Potassium translocation from extracellular fluid to intracellular fluid can occur with use of insulin or glucose-containing fluids.¹

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