Management of Potassium Disorders

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You have asked…

What is the best practice in addressing hyper- and hypokalemia in dogs and cats?

The expert says…

With 95% of total body potassium located intracellularly, potassium is the major intracellular cation. Its most important function is generating normal resting cell membrane potential, which regulates the function of excitable tissue (cardiac and skeletal muscle and nerves). Because small changes in extracellular potassium concentrations can have life-threatening effects on excitable tissue function, plasma potassium concentration is maintained within narrow limits. Hyperkalemia and hypokalemia are common electrolyte abnormalities in hospitalized patients, although hypokalemia occurs more frequently than does hyperkalemia.

Hyperkalemia

Hyperkalemia, a serum potassium concentration greater than 5.5 mEq/L (although reference ranges can vary), is considered life threatening at concentrations greater than 7.5 mEq/L; however, an individual animal may have life-threatening clinical signs at higher or lower concentrations. The prevalence and clinical impact of hyperkalemia in veterinary patients are unknown, but the condition can be fatal.

Severe hyperkalemia requires rapid correction to prevent significant cardiovascular complications (**Table 1,** next page). Hyperkalemia (**Table 2,** next page) is generally associated with impairment in urinary excretion of potassium, although shifts in intracellular and extracellular potassium may contribute to elevated levels. Increased intake is an unlikely cause if both renal and adrenal function are normal.

Clinical Effects

Most clinical consequences of hyperkalemia are related to the effect on transmembrane resting cell potential. Cardiac and neuromuscular cells are most sensitive to changes in serum potassium

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concentrations. Changes in the cardiac conduction system are usually evidenced by ECG changes, which may indicate potentially life-threatening arrhythmias. Classic ECG changes include tented T waves, widened QRS complexes, atrioventricular conduction blocks, accelerated idioventricular rhythm, sinoatrial block, ventricular fibrillation, and asystole.

No absolute level of serum potassium concentration is associated with a particular ECG abnormality; the ECG may be normal with life-threatening hyperkalemia and ECG abnormalities need not be present for emergency treatment of severe hyperkalemia. Bradycardia is commonly seen with hyperkalemia as a result of prolonged de- and repolarization of the myocardial conduction system. Hyperkalemia may also produce mild hyperchloremic metabolic acidosis.

Table 1 **Emergency Approach to Hyperkalemia[1]**

Agent	*Dose*	*Onset*	*Duration*	*Complications*
10% Calcium gluconate	0.5–1.5 mL/kg over 5–10 min with ECG monitoring	Immediate	30–60 min	Hypercalcemia
Short-acting insulin (ie, regular insulin) with 25% dextrose	0.5 U/kg IV; dextrose at 2 g/U of insulin administered; consider 1.25%–2.5% dextrose CRI for 4–6 hr	15 min	4–6 hr	Hypoglycemia
β_2-Adrenergic agonists	Terbutaline 0.01 mg/kg IV slowly *or* 1–3 puffs of albuterol inhaler or nebulization of albuterol solution	30 min	2 hr	Tachycardia, inconsistent response
Sodium bicarbonate	1–2 mEq/kg IV over 10–15 min	30–60 min	Duration of infusion	Inconsistent response, metabolic alkalosis, paradoxical CNS acidosis

Table 2 **Causes of Hyperkalemia**

Presentation	*Potential Causes*
Decreased urinary excretion (most common)	• Anuric or oliguric renal failure • Chylothorax with repeated pleural drainage • Drugs (eg, ACE inhibitors, potassium-sparing diuretics) • Hypoadrenocorticism • Ruptured urinary bladder, ureter(s), urethra • Urethral obstruction • Whipworm infestation, salmonellosis, perforated duodenum
Translocation from intracellular fluid to extracellular fluid	• Massive tissue damage from trauma, reperfusion • Metabolic acidosis • Nonspecific β-blockers (eg, propranolol)
Pseudohyperkalemia	• Thrombocytosis

Treatment

Acute changes in serum potassium concentrations tend to produce more significant (potentially life-threatening) clinical effects than are produced by chronic changes. Treatment of hyperkalemia is intended to prevent adverse cardiac complications. Although there is no consensus for treatment of hyperkalemia, veterinary patients with acute serum potassium concentrations greater than 6.5 mEq/L or with ECG changes suggestive of hyperkalemia are typically treated. Treatment is aimed at one or more of the following mechanisms.

1 Direct Antagonism of Hyperkalemia on Cell Membrane Polarization

Infusion of calcium gluconate antagonizes the effect of hyperkalemia on the heart without lowering serum potassium concentrations. It usually starts working within minutes and should last about 30 to 60 minutes. Repeat doses (0.5–1.5 mL/kg IV over 5–10 min) can be administered until a desired effect is noted. The heart rate should be monitored, ideally via ECG, to ensure that rapid administration does not cause bradycardia. Potential complications include hypercalcemia, bradycardia, and asystole.

Hypertonic saline may reverse ECG changes of hyperkalemia in patients with concurrent hyponatremia and starts working within minutes, although its duration of action is unknown. Hypertonic saline is not commonly used for this purpose in veterinary patients. Potential complications include volume overload and hypertonicity.

2 Redistribution of Extracellular Potassium into the Intracellular Compartment

Administration of regular insulin shifts potassium into the cells about 15 minutes after administration and lasts approximately 2 to 4 hours. An IV bolus of 25% dextrose should be administered concurrently to prevent hypoglycemia. CRI of 1.25% to 2.5% dextrose for 4 to 6 hours after insulin administration is recommended at doses of 0.5 U/kg of regular insulin and 2 g of IV dextrose per each unit of insulin administered. Combination insulin and dextrose is the author's preferred treatment for hyperkalemia. Dextrose, however, may be administered alone to treat hyperkalemia. Its mechanism of action is to stimulate endogenous insulin release, which ultimately drives potassium intracellularly. Sole administration of dextrose is inconsistent for managing severe hyperkalemia and is not generally recommended.[1]

β_2-Adrenergic agonists (eg, albuterol, terbutaline) can be administered as an adjunct treatment, driving potassium intracellularly by increasing sodium and potassium ATPase activity. Albuterol can be administered through inhalation or nebulization. Terbutaline can be administered at 0.01 mg/kg IV slowly. β_2-Adrenergic agonists exert their effects about 30 minutes following administration, with a duration of action of about 2 hours.

Sodium bicarbonate can also move serum potassium intracellularly, although it is less effective than insulin or β_2-adrenergic agonists and has a delayed onset of action. In humans, a single IV injection of sodium bicarbonate has little effect on serum potassium concentrations; however, a 4-hour CRI has been shown to lower potassium.[1] Sodium bicarbonate should thus be limited to situations in which concurrent acidosis may also benefit from its administration. Complications of administering sodium bicarbonate include metabolic alkalosis, hypernatremia, and paradoxical CNS acidosis.

3 Removal of Potassium from the Body

The most effective way to remove potassium from the body is through hemodialysis or peritoneal dialysis. Onset of action is immediate; however, these procedures are limited and expensive. Oral agents, including polystyrene sulfonate (Kayexalate, covispharma.com), are used in human patients, but their use is limited in veterinary medicine.

Fluid therapy may also correct mild hyperkalemia. Traditionally recommended for patients with hyperkalemia, 0.9% sodium chloride contains no potassium. However, potassium-containing fluids (lactated Ringer's solution; PlasmaLyte A, abbottanimalhealth.com; Normosol-R, hospira.com) can be safe in patients with hyperkalemia if the underlying cause has been identified and is being treated (eg, cats with urinary obstruction).

Hypokalemia

Hypokalemia, a serum potassium concentration of less than 3.5 mEq/L, is more common than is hyperkalemia. Hypokalemia (**Table 3**, next page) occurs as a consequence of potassium depletion because of increased excretion, redistribution, or inadequate potassium intake.

Clinical Effects

Unless severe, hypokalemia typically has no clinical signs. Cardiac and neuromuscular cells are the most susceptible targets; serious and potentially fatal effects are usually related to disturbances in cardiac electrical activity.

Skeletal muscle weakness is the primary clinical sign associated with hypokalemia in dogs and cats. Ventroflexion of the head

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and neck; stiff, stilted gait; and plantigrade stance are characteristic physical examination findings. Respiratory arrest and death from diaphragmatic and/or respiratory muscle failure can occur in severely affected patients. Rhabdomyolysis has also been reported with hypokalemia.[3]

Hypokalemia effects on myocytes may predispose the patient to atrial and ventricular tachyarrhythmias, atrioventricular dissociation, and ventricular fibrillation. In addition, hypokalemia can predispose the heart to digitalis-induced arrhythmias and cause myocardial cells to be refractory to class I antiarrhythmic drugs.[4]

Table 3 **Causes of Hypokalemia[2]**

Presentation	*Potential Causes*
Increased excretion	• Aldosterone-secreting tumor • Chronic kidney disease • Diabetic ketoacidosis • Diarrhea • Postobstructive diuresis • Vomiting
Inadequate intake	• Administration of potassium-free fluids • Severely potassium-deficient diet • Prolonged anorexia
Redistribution	• Barium poisoning • β-Adrenergic therapy or intoxication • Hyperthyroidism • Insulin administration • Metabolic alkalosis • Refeeding syndrome

Table 4 **Parenteral Potassium Supplementation**

Serum Potassium Level (mEq/L)	*Potassium Supplementation Required (mEq/L)*
3.5–5	20
3–3.4	30
2.5–2.9	40
2–2.4	60
<2	80

Treatment

Treatment of hypokalemia aims to prevent or correct cardiac electrical disturbances and serious neuromuscular weakness. The long-term goal is to return total body potassium to normal levels and identify and correct the primary underlying disease. Potassium supplementation can be achieved with parenteral administration of potassium chloride or potassium phosphate; potassium chloride is used more often, except in patients that require concurrent phosphorus supplementation. Less affected patients that eat on their own may receive oral potassium supplementation. Oral potassium supplementation can also be provided to patients with chronic kidney disease or chronic hypokalemia. There are various oral potassium supplements available, and they should be initiated based on the manufacturer-recommended dose, which may be adjusted based on patient response to therapy.

Guidelines for parenteral potassium supplementation (**Table 4**) in dogs and cats with mild-to-moderate hypokalemia may not adequately resolve hypokalemia at lower fluid rates and may cause hyperkalemia at higher fluid rates. To prevent under- or oversupplementation of potassium in patients with moderate-to-severe hypokalemia, supplementation should be calculated rather than estimated. The rate of potassium infusion should not exceed 0.5 mEq/L/kg/hr. In severely affected animals with normal urine output (serum potassium <2 mEq/L), higher rates can be administered cautiously (1–1.5 mEq/L/kg/hr) with concurrent ECG monitoring. In diabetic patients with hypokalemia, insulin or sodium bicarbonate administration should be delayed until serum potassium concentrations have been replenished.

The patient may be refractory to correction of hypokalemia when hypomagnesemia and hypocalcemia exist. It is important to correct all concurrent electrolyte abnormalities to attain normal neuromuscular function. Care must be taken not to create hyperkalemia when treating for hypokalemia. Frequent monitoring (q6–8h) of serum potassium concentration is recommended when aggressive replenishment is required. ■ cb

See **Aids & Resources**, back page, for references & suggested reading.