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## POTASSIUM DISORDERS IN CATS: MYTHS AND FACTS

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Potassium is the major intracellular cation in mammalian cells and is largely responsible for maintenance of intracellular volume. Serum potassium concentrations slightly exceed plasma concentrations because potassium is released from platelets during the clotting process. Potassium is responsible for maintaining resting cell membrane potential. Therefore, disorders of potassium concentration affect excitable membranes. Clinical signs are related to disturbances in skeletal (weakness) and cardiac (arrhythmia) muscles.

### Potassium and Acid-Base Balance: Myths and Facts

Internal distribution of potassium is affected by acid-base equilibrium. These changes, however, are only clinically relevant during acute mineral acidosis (e.g. infusion of HCl). Extrapolation of information obtained in experimental animals to the clinical setting has led to misconceptions regarding the effects of acid-base changes in potassium balance (Tab. 1).

**Table 1.** Myths and facts about potassium and acid-base disorders

<b>Myth 1</b>	Acid-base changes will clinically affect potassium concentration	<b>Fact 1</b>	Only clinical relevant in acute mineral acidosis (IV infusion of NH <sub>4</sub> Cl or HCl)
<b>Myth 2</b>	Oral administration of NH <sub>4</sub> Cl and HCl causes hyperkalemia	<b>Fact 2</b>	It causes hypokalemia
<b>Myth 3</b>	Potassium increases 0.5 mEq/l for each 0.1 unit decrease in pH	<b>Fact 3</b>	See fact 1. Based on a 1954 study in dogs (nephrectomized, anesthetized, infused with HCl)
<b>Myth 4</b>	Hyperkalemia in renal failure or ketoacidosis is due to metabolic acidosis	<b>Fact 4</b>	It is due to poor renal function (renal failure) or lack of insulin (ketoacidosis)
<b>Myth 5</b>	Hypokalemia causes metabolic alkalosis	<b>Fact 5</b>	True in rats. Not true in cats, dogs and human beings.

### Hypokalemia

Hypokalemia arises from decreased intake, translocation of potassium from ECF to ICF, and excessive loss of potassium by either gastrointestinal or urinary routes. The history often provides information about the likely source of potassium loss (e.g. chronic vomiting, diuretic administration) or the possibility of translocation (e.g. insulin administration). Muscular weakness, polyuria and polydipsia, anorexia, and tachycardia are the most common clinical signs associated with hypokalemia. Cats are usually presented with ventroflexion of the neck. Electrocardiographic changes in hypokalemia are not specific, although supraventricular and ventricular arrhythmias may occur. Hypokalemia renders arrhythmias refractory to therapy and favors digitalis toxicity. Other complications of hypokalemia include polymyopathy and respiratory muscle paralysis. Cats fed highly acidifying diets also low in potassium content may develop hypokalemic nephropathy.

Decreased intake of potassium alone is unlikely to cause hypokalemia, but it may be a contributing factor. Administration of potassium-deficient fluids to an anorectic animal also may result in hypokalemia.

Translocation of potassium into cells may occur with sodium bicarbonate administration, and insulin and catecholamine release. Insulin promotes uptake of glucose and potassium by hepatic and skeletal muscle cells and may contribute to hypokalemia when glucose-containing fluids are administered.

Gastrointestinal loss of potassium from vomiting or diarrhea is a very important cause of hypokalemia in cats. Urinary loss of potassium is another important cause of hypokalemia. Chronic renal disease was the most common associated disorder observed in a survey of cats with hypokalemia. Urinary losses of potassium also may occur in renal tubular acidosis and during the

postobstructive diuresis that follows relief of urethral obstruction and post administration of loop or thiazide diuretics.

Treatment of hypokalemia should be directed at correcting the underlying disease process and providing potassium supplementation. Parenteral therapy is usually attempted with KCl, although  $\text{KH}_2\text{PO}_4$  also can be used. Potassium-containing fluids can be administered intravenously or subcutaneously, although slow intravenous infusion usually is preferred. Maintenance supplementation (20-30 mEq/K/L) usually corrects mild hypokalemia (serum K > 3,0 mEq/L). Higher maintenance values should be used in patients with serum K close to 3,0 mEq/L. When potassium concentration is < 3,0 mEq/L, potassium supplementation in mEq of KCl per liter of fluid can be estimated as:  $\text{K supplement} = 4 - (\text{patient's K}) \times 35$ . Potassium should not be given at a concentration greater than 80 mEq/L or at a rate faster than 0,5 mEq/Kg/h to avoid acute hyperkalemia and its adverse effects. Fluids containing > 60 mEq/L potassium should be administered carefully because of the potential for sclerosis of peripheral vessels. Infusion of potassium-containing fluids may be associated with an initial decrease in serum potassium concentration. This can be minimized by using a glucose-free solution and by starting oral potassium supplementation as soon as possible. Potassium gluconate usually is recommended for oral supplementation. In cats, the initial dose is 5-8 mEq/day divided BID or TID, followed by a maintenance dose of 2-4 mEq/day. The dose should be adjusted based on clinical improvement and serial measurements of potassium concentration.

### **Hyperkalemia**

Chronic hyperkalemia almost always is associated with impairment in urinary potassium excretion. Clinical manifestations of hyperkalemia reflect changes in cell membrane excitability with muscle weakness and cardiac electrical conduction abnormalities. ECG changes in mild hyperkalemia include increased amplitude and narrowing of the T wave and shortening of the QT interval. Moderate hyperkalemia causes prolongation of the PR interval and widening of the QRS. As hyperkalemia progresses, P waves decrease in amplitude, become wide and eventually disappear. Bradycardia due to a sinoventricular rhythm may be observed, although is less pronounced in cats. The QRS may merge with the T wave creating a sine wave appearance. Clinical and ECG signs do not necessarily correlate with potassium concentration, but ECG and muscle strength reflect the functional consequences of hyperkalemia.

Increased intake of potassium only causes sustained hyperkalemia if renal excretion of potassium is abnormal. Exceptions include iatrogenic hyperkalemia resulting from calculation errors during IV infusion of potassium and administration of drugs known to predispose to hyperkalemia (e.g. beta-blockers or ACE inhibitors) with concurrent potassium supplementation.

Translocation of potassium from ICF to ECF can occur in diabetic patients. Insulin deficiency and hyperosmolality contribute to the development of hyperkalemia in diabetic patients. Massive tissue breakdown may lead to transient hyperkalemia until released potassium is excreted by the kidneys. This may occur after reperfusion of aortic thromboembolism. Nonspecific beta-blockers (e.g. propranolol) decrease cellular uptake of potassium, and may cause hyperkalemia in the presence of a potassium load or decreased renal function.

Decreased urinary excretion of potassium is the most important cause of hyperkalemia in feline practice. It usually results from urethral obstruction, ruptured bladder, and anuric or oliguric renal failure. Oliguria or anuria with hyperkalemia are more likely to occur in acute renal failure, but they may be observed terminally in chronic renal failure. Patients with chronic renal disease have reduced ability to tolerate an acute potassium load and may require 1-3 days to reestablish external potassium balance when intake of potassium is abruptly increased (e.g., fluid therapy). Several drugs may reduce renal excretion of potassium. ACE inhibitors interfere with angiotensin II-mediated aldosterone secretion, whereas spironolactone competitively blocks aldosterone.

Treatment of hyperkalemia will depend on the magnitude and rapidity of onset of the hyperkalemia. Patients with symptomatic hyperkalemia or ECG abnormalities should be treated. Mild chronic hyperkalemia ( $\text{K} < 6,5$  mEq/L) may not require immediate therapy.

*Asymptomatic, non-oliguric patient:* The underlying disease process should be treated and any source of potassium intake or drugs that cause potassium retention should be discontinued, if possible. Fluid therapy with potassium-free solutions ameliorates mild hyperkalemia by improving

renal perfusion, enhancing urinary excretion of potassium, and by diluting the potassium in ECF.

*Mild Hyperkalemia:* Administration of glucose or NaHCO<sub>3</sub> can be attempted in patients with mild hyperkalemia that did not respond to potassium discontinuation and fluid therapy. Administration of glucose (1-2 ml/kg of the 50% solution) will cause release of insulin and move potassium into the cells. The effects begin within an hour and last a few hours. Concurrent administration of insulin may improve the response but it increases the risk of hypoglycemia. Sodium bicarbonate (1-2 mEq/Kg, repeated if necessary) also will move potassium inside cells within an hour. All potassium driven inside the cells will come back out after a few hours. Therefore, drugs that translocate potassium have a short duration of action.

*Moderate to Severe Hyperkalemia:* are emergencies. Administration of calcium gluconate (2-10 ml of a 10% solution given slowly with ECG monitoring) may antagonize the adverse electrophysiologic effects of hyperkalemia. Potassium concentration, however, will remain increased and the beneficial effects of calcium are short-lived (less than one hour). Administration of glucose or NaHCO<sub>3</sub> is recommended to lower potassium concentration.

*Adjunctive therapy:* in selected cases of hyperkalemia, loop diuretics may be used to enhance the flow rate in the distal nephron and increase potassium excretion. Patients with renal failure are unlikely to benefit from this maneuver.

*When Everything Failed:* Atropine may be attempted in patients that are bradycardic. During the hyperkalemia-induced bradycardia, the SA-node still works (the so-called sinoventricular rhythm) and is under vagal influence. Atropine injection may cause a mild increase of heart rate and temporarily reestablish cardiac output and blood pressure. Refractory cases may require hemodialysis or continuous renal replacement therapy.

#### **Suggested Reading**

DiBartola SP, de Morais HA: Disorders of potassium: Hypokalemia and hyperkalemia, in DiBartola SP (ed): *Fluid, Electrolyte, and Acid-Base Disorders*. 3<sup>rd</sup> ed., Philadelphia, Elsevier, 2006, pp 91-121.

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