# Disorders of Potassium: Hypokalemia and Hyperkalemia

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Over 90% of the potassium in the body is located within cells. External balance for potassium is maintained by matching output to input. Internal balance is maintained by translocation of potassium between intracellular and extracellular fluid. Any change in plasma potassium concentration must arise from a change in intake, distribution, or excretion.

## Hypokalemia

### Causes

Decreased intake of potassium alone is unlikely to cause hypokalemia but, in chronically ill animals, prolonged anorexia, loss of muscle mass, and ongoing urinary potassium losses may combine to cause hypokalemia. Alkalemia contributes to hypokalemia as potassium ions enter cells in exchange for hydrogen ions. Insulin promotes uptake of glucose and potassium by hepatic and skeletal muscle cells. A syndrome characterized by recurrent episodes of limb muscle weakness and neck ventroflexion, increased creatine kinase concentrations, and hypokalemia has been reported in related young Burmese cats.

Gastrointestinal loss of potassium (especially vomiting of stomach contents) is an important cause of hypokalemia in small animals. Chloride depletion and sodium avidity due to volume depletion contribute to perpetuation of potassium depletion and metabolic alkalosis by enhancing urinary losses of potassium and hydrogen ions. Urinary loss of potassium is another important cause of hypokalemia and hypokalemia is common in cats with chronic renal failure. Hypokalemia also may occur in distal renal tubular acidosis in cats. Finally, hypokalemic nephropathy characterized by tubulointerstitial nephritis may develop in cats fed diets marginally replete in potassium and containing urinary acidifiers. Hypokalemia commonly occurs during the postobstructive diuresis that follows relief of urethral obstruction in cats. Mineralocorticoid excess is a rare cause of urinary potassium loss and hypokalemia in dogs and cats. Administration of loop or thiazide diuretics may cause hypokalemia by increased flow rate in the distal tubules and increased secretion of aldosterone secondary to volume depletion. Peritoneal dialysis can be complicated by hypokalemia if potassium-free dialysate is used over an extended period of time.

### Clinical signs

Muscle weakness may be observed when serum potassium concentration falls below 2.5-3.0 mEq/L. Rear limb weakness and, in cats, weakness of neck muscles with ventroflexion of the head are commonly observed. Cardiac arrhythmias may develop because hypokalemia increases automaticity and delays ventricular repolarization. In dogs and cats, the electrocardiographic changes associated with hypokalemia are inconsistent but ventricular arrhythmias may be observed. Polyuria, polydipsia, and defective urinary concentrating ability may be observed in hypokalemia.

### Diagnosis

The clinical history often will provide information about the likely source of potassium loss (e.g. vomiting, diuretic administration). Determination of the fractional excretion of potassium (FEK as a percentage = UKPCr/PKUCr X 100) may help differentiate renal and non-renal sources of potassium loss. The FEK should be < 4% for non-renal sources of loss and values > 4% indicate inappropriate renal loss in the face of hypokalemia. The occurrence of hypokalemia with metabolic alkalosis suggests vomiting of stomach contents or diuretic administration as likely causes of potassium loss.

### Treatment

Potassium chloride is the additive of choice for parenteral therapy because chloride repletion also is very important if vomiting or diuretic administration is the underlying cause of hypokalemia. When administered intravenously, potassium should not be infused at a rate greater than 0.5 mEq/kg/hr. Infusion of potassium-containing fluids initially may be associated with a decrease in plasma potassium concentration as a result of dilution, increased distal tubular flow, and cellular uptake of potassium, especially if the infused fluid also contains glucose. This effect may be minimized by using a fluid that does not contain glucose and by administering it at an appropriate rate. Potassium gluconate is recommended for oral supplementation. In cats with hypokalemic nephropathy, the initial oral dosage of potassium gluconate is 5-8 mEq per day divided BID or TID whereas the maintenance dosage can usually be reduced to 2-4 mEq per day.

Careful potassium supplementation is very important when using insulin to treat diabetic ketoacidosis. Chronic potassium depletion usually is present in affected patients as a result of loss of muscle mass, anorexia, vomiting, and polyuria. Serum potassium concentrations, however, often are normal or even increased due to the effects of insulin deficiency and hyperosmolality on serum potassium concentration. As blood glucose concentration falls with insulin treatment, marked hypokalemia may develop if supplementation is not diligent.

## Hyperkalemia

### Causes

Hyperkalemia occurs uncommonly if renal function is normal. Even in chronic renal failure, potassium excretion is maintained by enhanced tubular secretion in remnant nephrons so that hyperkalemia only develops if oliguria supervenes. Thus, chronic hyperkalemia is almost always associated with impaired renal excretion. Increased intake is likely to be contributory only during excessive infusion of potassium-rich fluids or in the face of impaired renal excretion.

Serum potassium concentrations exceed plasma concentrations because potassium is released from platelets during clotting. The difference between serum and plasma potassium concentrations is most pronounced in animals with thrombocytosis. Hemolysis can result in hyperkalemia in species with high red cell potassium content. Normal canine and feline red cells contain potassium in concentrations similar to those of plasma and hemolysis usually is not associated with hyperkalemia. In some Akitas, however, red cell potassium concentration may be as high as 70 mEq/L and hemolysis may result in progressive hyperkalemia during storage of blood. Hemolysis in Akitas and thrombocytosis cause what has been called pseudohyperkalemia because these effects occur in vitro.

Translocation of potassium from intracellular to extracellular fluid can cause hyperkalemia. Metabolic acidosis due to mineral acids (e.g. $NH_4Cl$, HCl) but not organic acids (e.g. lactic acid, ketoacids) causes potassium to shift out of cells in exchange for hydrogen ions that enter cells to be buffered. The effect of inorganic metabolic acidosis on serum potassium concentration is very variable. Insulin deficiency and hyperosmolality contribute to hyperkalemia in diabetic patients. Acute tumor lysis syndrome complicated by renal failure and hyperkalemia has been reported in dogs with lymphoma after radiation or chemotherapy.

Decreased urinary excretion is the most important cause of hyperkalemia in small animal practice. The most common associated disorders are urethral obstruction, ruptured bladder, anuric or oliguric renal failure, and hypoadrenocorticism. The time required for development of hyperkalemia in cats after urethral obstruction is variable but it may occur within 48 hours. After relief of obstruction, hyperkalemia resolves within 24 hours whereas azotemia and hyperphosphatemia require 48-72 hours to resolve. After experimental bladder rupture in dogs, azotemia, hyperphosphatemia, and mild hyponatremia developed within 24 hours whereas hyperkalemia did not develop until after 48 hours. Hyperkalemia, hyponatremia, and Na/K ratios < 27:1 are usually (but not always) found in dogs and cats with hypoadrenocorticism and similar findings can occur in dogs with gastrointestinal disease due to trichuriasis, salmonellosis, or perforated duodenal ulcer. Hyperkalemia only occurs in renal failure when anuria or oliguria develops. This occurs more commonly in acute renal failure (e.g. ethylene glycol ingestion) but may occur terminally in chronic renal failure. Potassium-sparing diuretics (e.g. spironolactone) reduce urinary excretion of potassium and can cause hyperkalemia.

### Clinical signs

Muscle weakness develops with hyperkalemia, usually when serum potassium concentration exceeds 8 mEq/L. The electrocardiographic findings caused by hyperkalemia include peaked narrow T waves and shortened QT interval reflecting abnormally rapid repolarization. These changes are followed by widened QRS complexes and decreased amplitude, abnormally wide or absent P waves associated with delayed depolarization. A sinoventricular rhythm develops followed by ventricular fibrillation and cardiac standstill. These electrocardiographic findings represent the life-threatening functional consequences of hyperkalemia.

### Diagnosis

Increased intake, translocation from intracellular to extracellular fluid, and impaired excretion must all be considered when evaluating a patient with hyperkalemia. Except in a hospital setting where infusion of potassium-containing fluids may be the source of hyperkalemia, decreased urinary excretion of potassium or translocation are the usual causes.

### Treatment

Hyperkalemia can be treated by antagonizing its effects on cell membranes with calcium gluconate, driving extracellular potassium into cells with sodium bicarbonate or glucose, or by removing potassium from the body with a cation exchange resin or dialysis. First, any source of intake must be discontinued (e.g. potassium-containing fluids, potassium penicillin). Hyperkalemia decreases the resting potential of cells. By administering calcium gluconate, the extracellular fluid concentration of calcium is increased and the threshold potential is decreased thus normalizing the difference between the resting and threshold potential and restoring normal membrane excitability. Administered calcium begins to work within minutes but its effect lasts less than an hour. The dosage of calcium gluconate is 2-10 ml of a 10% solution to be administered slowly with electrocardiographic monitoring. Glucose works by increasing endogenous insulin release and moving potassium into cells. Its effects begin within an hour and last a few hours. Glucose-containing fluids (5 or 10% dextrose) or 50% dextrose (1-2 ml/kg) can be used for this purpose. Unless the patient is diabetic, administration of insulin with glucose usually is unecessary and may cause hypoglycemia. Sodium bicarbonate also works by moving potassium into cells as hydrogen ions come out to titrate the administered bicarbonate. Bicarbonate begins to work within an hour and its effects last a few hours. The usual dosage is 1-2 mEq/kg intravenously and it can be repeated if necessary. Loop or thiazide diuretics increase distal tubular flow rate and potassium secretion and may have adjunctive value in the treatment of hyperkalemia. The cation exchange resin polystyrene sulfonate can be used to bind potassium and release sodium in the gastrointestinal tract. Each gram will bind one mEq of potassium and release 1-2 mEq of sodium. If these measures fail, the clinician must consider peritoneal dialysis.

**References**

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