

ECG of the Month

A 6-year-old 5.25-kg (11.6-lb) castrated male domestic shorthair cat was referred to the Tufts Cummings School of Veterinary Medicine Emergency Service for management of thromboembolism in the distal portion of the aorta. The cat had no history of important medical problems.

On initial evaluation, the cat was bright, alert, and responsive. Body temperature (measured by use of an aural thermometer) was 39.7°C (103.5°F). The heart rate was 180 beats/min with an intermittent cardiac gallop. A grade 2/6 left parasternal systolic murmur was ausculted. The cat was eupneic (respiratory rate, 36 breaths/min), and auscultation of the lungs revealed no abnormalities. Paraplegia was evident, and both hind limbs were cold to the touch. A weak femoral pulse was palpated in the left hind limb, and no pulse was palpated in the right hind limb.

The initial diagnostic evaluation included venous blood gas and serum biochemical (electrolytes and renal variables) analyses and urinalysis of a voided urine sample. Biochemical abnormalities included mildly high BUN concentration (39 mg/dL; reference range, 7 to 28 mg/dL) and mild hyperlactatemia (2.8 mmol/L; reference range, 0.3 to 2.0 mmol/L). Venous blood pH was 7.389 (reference range, 7.337 to 7.467). Serum creatinine concentration was 1.0 mg/dL (reference range, 0.2 to 2.1 mg/dL). The urine was concentrated (specific gravity, 1.037).

Initial treatment consisted of administration of dalteparin sodium^a (150 U/kg [68.2 U/lb, SC, q 12 h), clopidogrel bisulfate^b (3.75 mg/kg [1.70 mg/lb], PO, q 24 h), oxymorphone hydrochloride^c (0.05 mg/kg [0.023 mg/lb], IV, q 4 h), s-adenosyl methionine^d

(18 mg/kg [8.2 mg/lb], PO, q 24 h), and n-acetylcysteine^e (140 mg/kg [63.6 mg/lb], IV, initially and then 70 mg/kg [31.8 mg/lb], IV, q 6 h [3 doses]). The latter 2 treatments were administered as prophylactic measures against oxidant damage related to ischemia-reperfusion injury.

Echocardiographic examination revealed hypertrophic cardiomyopathy with concentric left ventricular hypertrophy and severe left atrial enlargement. Moderate spontaneous echocardiographic contrast, but no evidence of thrombus formation, was evident in the left atrium. Conservative supportive care for arterial thromboembolism was continued.

Thirty hours after admission to the hospital, the cat developed bradycardia. Electrocardiography was performed via telemetry (Figure 1). At this time, clinicopathologic analyses revealed high potassium concentration (9.74 mmol/L; reference range, 3.8 to 4.9 mmol/L) and progressive azotemia (BUN concentration, > 120 mg/dL; creatinine concentration, 2.5 mg/dL).

ECG Interpretation

Findings from evaluation of the initial ECG tracing (Figure 1) included evidence of bradycardia (heart rate, 115 beats/min), widening of the QRS complexes (0.07 seconds; reference range, \leq 0.04 seconds), and absence of P waves. The T waves were negative with a spiked appearance. Positive spiked T waves are a commonly reported ECG finding associated with hyperkalemia, but the waves may be positive or negative in affected cats.¹ The appearance of this initial ECG tracing was consistent with slow sinoventricular conduction, which is commonly detected in cats with hyperkalemia.²

Over the following 8 hours, the cat received fluids and separate bolus injections of dextrose, calcium gluconate, sodium bicarbonate, and regular insulin IV. Normal sinus rhythm (heart rate, 170 to 180 beats/min) was evident for part of the 8-hour period, but the cat became refractory to the treatments; the onset of meta-



Figure 1—Lead 2 ECG tracing obtained via telemetry from a cat that had arterial thromboembolism and that developed bradycardia 30 hours after hospitalization and initiation of conservative supportive care. At the time of this ECG evaluation, hyperkalemia (secondary to reperfusion injury) was detected. The heart rate is 115 beats/min, and the heart rhythm is atrial standstill. The QRS complexes are wide (0.07 seconds; reference range, \leq 0.04 seconds), consistent with delayed or aberrant conduction through the His-Purkinje system. There are no obvious P waves. Paper speed = 25 mm/s; 1 cm = 1 mV.

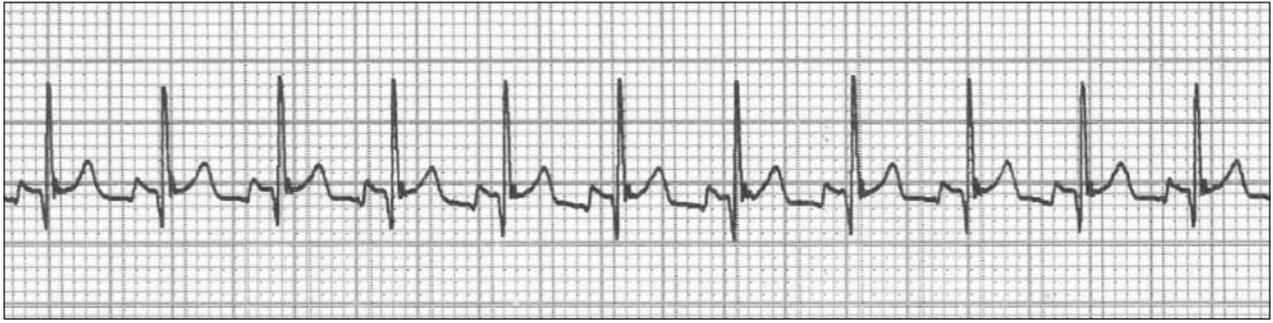


Figure 2—Lead 2 ECG tracing obtained via telemetry from the cat in Figure 1 following SC administration of terbutaline sulfate. The heart rate is 158 beats/min; the heart rhythm is regular and consistent with normal sinus rhythm. The duration of the QRS complexes appears normal (0.04 seconds). Paper speed = 25 mm/s; 1 cm = 1 mV.

bolic acidosis (venous blood pH, 7.288), progressive azotemia (BUN concentration, > 120 mg/dL [value exceeded measurement range]; creatinine concentration, 2.9 mg/dL), and persistent hyperkalemia (9.19 mmol/L) were detected. Persistent bradycardia developed as signs of depression increased.

Terbutaline sulfate^f (8 µg/kg [3.6 µg/lb], SC) was then administered and after approximately 30 minutes, an ECG recording revealed normal sinus rhythm (heart rate, 158 beats/min; Figure 2). A P wave of normal appearance preceded each QRS complex, and the corresponding PR intervals were considered normal (0.08 seconds; reference interval, 0.04 to 0.09 seconds). The duration of the QRS complexes was apparently normal (0.04 seconds), and the T waves were positive. Clinicopathologic analyses performed at this time revealed ongoing but less severe hyperkalemia (6.45 mmol/L) and progression of azotemia (BUN concentration, > 120 mg/dL; creatinine concentration, 3.5 mg/dL); venous blood pH (7.341) was within reference range. Following administration of terbutaline and the resultant increase in heart rate, the cat's attitude improved.

The cat remained in the hospital for approximately 36 hours after terbutaline was first administered. Normal sinus rhythm persisted during that period, and whole blood potassium concentrations at the times of ECG evaluation were 4.91 and 5.14 mmol/L. Terbutaline administration was continued every 6 hours for 3 additional doses, and a second dose of sodium bicarbonate was given 12 hours after the initial dose. Against medical advice, however, the cat was taken home by the owners and subsequently died.

Discussion

In cats with cardiogenic arterial thromboembolism, hyperkalemia can develop secondary to ischemia-reperfusion injury and is thought to be a consequence of reperfusion of muscles that have undergone ischemic rhabdomyolysis.³ However, development of hyperkalemia in the cat of this report was not associated with improvement in the strength of the left femoral pulse or return of a palpable right femoral pulse, although these failures did not rule out reperfusion from collateral circulation.⁴

An increase in extracellular potassium concentration decreases the resting membrane potential of atrial and ventricular myocytes, which leads to inactivation of fast sodium channels and a subsequent decrease in

maximum upstroke velocity and conduction velocity of action potentials.⁵ Atrial and ventricular myocytes are more sensitive to the effects of hyperkalemia than are the cells in the sinoatrial node, atrioventricular node, and internodal tracts. This increased sensitivity is likely attributable to the fact that the myocytes depend on fast sodium channels for phase-zero depolarization, whereas nodal tissues depend on slow calcium channels.¹ Progressive changes in ECG tracings associated with worsening hyperkalemia reflect these underlying mechanisms. Initially, a slowing of the heart rate with narrowing and peaking of T waves may be seen. As extracellular potassium concentration increases, further decreases in heart rate with widening of QRS complexes and widening and subsequent flattening of P waves reflect progressive depression of atrial and ventricular conduction. Ultimately, complete atrioventricular block, further delay in ventricular conduction, and ventricular fibrillation or asystole may result.^{1,2} However, predictable ECG changes with progressive hyperkalemia may not always develop, especially in ill cats with other electrolyte and acid base abnormalities.⁶ Additionally, ill cats that undergo continuous ECG monitoring are generally not in a standard position (ie, right lateral recumbency), and all 4 limbs may not be attached to electrodes while obtaining ECG measurements. A 6-lead ECG recording and use of precordial leads may aid in detection of P- or T-wave alterations that may not be apparent in a single-lead recording. Neither of these additional assessments was performed in the cat of this report.

Treatment of severe hyperkalemia is often multimodal and is intended to antagonize the effect of potassium on excitable cell membranes, redistribute potassium from the extracellular space to the intracellular space, and increase renal elimination of potassium.⁷ Calcium helps to stabilize the myocyte membrane through reduction of the threshold potential, thereby restoring the membrane to threshold potential gradient. Insulin promotes potassium redistribution to the intracellular space via stimulation of the Na⁺, K⁺-ATPase pump. Dextrose may be given alone or concurrently with insulin to stimulate endogenous insulin secretion and protect against insulin-induced hypoglycemia. Conventional wisdom suggests that bicarbonate causes potassium influx into cells as hydrogen ions exit cells to buffer the bicarbonate, although this has been shown not to occur in humans with dialysis-dependent renal failure.⁸ Bicarbonate may have more important effects through

enhancement of kaliuresis, secondary to increased delivery of bicarbonate to the distal tubules of the glomeruli and urine alkalinization.⁸ β -Adrenoceptor agonists such as albuterol and terbutaline reduce extracellular potassium concentrations through insulin-independent stimulation of the Na⁺, K⁺-ATPase pump.^{8,9}

To the authors' knowledge, there are no published reports of the use of terbutaline in the treatment of hyperkalemia in cats. Terbutaline may be a useful adjunctive treatment when standard treatment (administration of insulin, dextrose, calcium, and bicarbonate) is unsuccessful at controlling hyperkalemia. Terbutaline is not recommended as a first-line treatment for hyperkalemia in people,⁸ nor do the authors recommend such use in veterinary medicine at this time.

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- a. Fragmin, Pfizer Inc, New York, NY.
 - b. Plavix, Bristol-Myers-Squibb/Sanofi Pharmaceuticals, Bridgewater, NJ.
 - c. Oxymorphone hydrochloride, EndoPharmaceuticals, Chadds Ford, Pa.
 - d. Denosyl, Nutramax Laboratories, Edgewood, NJ.
 - e. n-Acetylcysteine, Hospira Inc, Lake Forest, Ill.
 - f. Terbutaline sulfate, Bedford Laboratories, Bedford, Ohio.
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