Original Study

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Electrocardiographic assessment of hyperkalemia in dogs and cats

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Abstract

Objective: To determine if electrocardiogram (ECG) changes induced by hyperkalemia in clinical patients correspond with previously reported changes in experimental animals.
Design: Prospective clinical study.
Setting: Two private practice 24-hour emergency and critical care facilities.
Animals: Fifteen dogs and 22 cats with serum potassium levels >5.5 mEq/L.
Interventions: None.
Measurements: The following data were collected when hyperkalemia was documented: ECG ($n = 37$), sodium and chloride (mEq/L) ($n = 35$), total magnesium (mg/dL) ($n = 18$), total calcium (mg/dL) ($n = 30$), and venous pH ($n = 18$). Animals were divided into five groups based on severity of hyperkalemia and ECG interpretation included rate, rhythm and P-QRS-T evaluation.
Main Results: Twenty-two of 37 (59%) of the ECGs were normal or revealed abnormalities that have not been previously described in conjunction with hyperkalemia. In dogs, there was no correlation ($r = 0$) between potassium blood levels and heart rate ($n = 15$). There was weak correlation ($r = 0.40$; $P = 0.06$) between potassium blood levels and heart rate in cats ($n = 22$). The correlation was stronger ($r = 0.64$; $P < 0.05$) when data were compared in cats with serum potassium level >8.5 mEq/L (Groups 4 and Group 5; $n = 11$).
Conclusions: ECGs obtained from ill dogs and cats with hyperkalemia are inconsistent with ECGs from experimentally induced hyperkalemia. It is difficult to determine the clinical relevance of heart rate differences between cats with serum potassium levels >8.5 mEq/L and animals with experimentally induced hyperkalemia; this may be due to the presence of other biochemical abnormalities in diseased animals.

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Introduction

Hyperkalemia can result in life-threatening cardiac arrhythmias[1–6] and must be rapidly diagnosed. Numerous disease processes can result in hyperkalemia in dogs and cats including acute renal failure, urethral obstruction, hypoadrenocorticism, pseudohypoadrenocorticism (e.g., caused by *Trichuris vulpis* infestation or Salmonellosis), and over-supplementation of potassium. Electrocardiographic alterations have been previously observed in experimental trials where hyperkalemia was induced in healthy animals[2,3,7] and include the following electrocardiogram (ECG) alterations at different levels of potassium concentration: serum potassium concentrations of 5.5–6.5 mEq/L led to an increase in the T wave amplitude; serum potassium concentrations of 6.6–7.0 mEq/L led to a decrease in amplitude of the R wave, prolonged QRS and P–R intervals, and S–T segment depression; serum potassium concentrations of 7.1–8.5 mEq/L led to a decreased amplitude of the P wave, increased P wave duration, and prolongation of the Q–T interval; serum potassium concentrations of 8.6–10.0 mEq/L led to a lack of P waves (atrial standstill) and sinoventricular rhythm; and serum potassium concentrations >10.1 mEq/L led to a widening of the QRS complex, eventual replacement of the QRS with a smooth biphasic waveform, ventricular flutter, fibrillation, or asystole.[2,3,7] While such ECG responses have been elicited experimentally, clinicians generally recognize that these characteristic ECG changes may not always occur in clinical practice.[1,7,8]

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There may be a number of associated biochemical abnormalities occurring concurrently with hyperkalemia in clinically ill patients and these abnormalities affect the ECG.[1,7,9–13] Examples of such abnormalities include changes in serum sodium, chloride, magnesium, calcium, and venous pH. The primary objective of this study was to determine to what extent previous experimentally documented ECG changes secondary to hyperkalemia occur in clinical patients. Other biochemical data is included to evaluate the degree to which other abnormalities may also be present in ill hyperkalemic animals.

Materials and Methods

A total of 37 privately owned dogs and cats from two private practice 24-hour emergency and critical care facilities were included in the study that extended from January 1, 2003, to December 31, 2004. Owner consent was obtained before inclusion in the study. Inclusion criteria were non-anesthetized dogs and cats with a serum potassium level >5.5 mEq/L and a simultaneous ECG recorded before therapy. Exclusion criteria included the presence of known or induced cardiac disease and exposure to cardiovascular toxins such as digitalis glycosides and *Bufo marinus*. Blood collected was analyzed on a portable analyzer[a] or was centrifuged at 3300 r.p.m. (619.6 g) for 10 minutes to facilitate serum removal for processing on a point-of-care analyzer.[b] Potassium, chloride, and sodium analyses were performed on both analyzers depending on the individual clinician's orders. Total calcium and magnesium values were calculated using the point-of-care analyzer.[b] Blood gas samples were analyzed using the portable analyzer.[a] The collected ECG rhythm strips were performed in sternal recumbancy for a minimum of 15 seconds in length with a standard paper speed (25 mm/s) and calibration (1 mV = 1 cm).

Animals were placed in 1 of 5 groups based on their serum potassium elevation: Group 1, 5.5–6.5 mEq/L; Group 2, 6.6–7.0 mEq/L; Group 3, 7.1–8.5 mEq/L; Group 4, 8.6–10.0 mEq/L; and Group 5, >10.1 mEq/L. ECGs were analyzed without knowledge of the severity of hyperkalemia and were then compared with previously reported hyperkalemia-induced ECG changes.[2,3,6,7] ECG parameters evaluated included overall rate and rhythm, P wave size and duration, P–R interval, QRS complex configuration and duration, Q–T interval, and T wave configuration.

Additional laboratory data was collected when possible including serum sodium, chloride, total magnesium, total calcium, and venous pH. These samples were collected before the institution of any patient therapy and were not required to be measured from blood collected during the same venipuncture from which documentation of serum hyperkalemia occurred. Samples were processed and analyzed in a similar fashion as the potassium samples.

Statistical Methods

The data was analyzed using a commercially available computer software program.[c] Linear regression was used to compare potassium blood level and heart rate in both dogs and cats. No further inferential statistical analyses were performed due to the small group sizes, which causes high within-group variability, making it difficult to evaluate the significance between group variation. The group intervals that were used were derived from previous experiments and subsequent descriptions in the literature. Descriptive analysis was used when comparing ECG alterations with previously reported alterations[2,3,7] in healthy animals at comparable levels of hyperkalemia.

Results

Fifteen dogs and 22 cats with serum potassium levels >5.5 mEq/L were included. Disease processes resulting in hyperkalemia included urethral obstruction ($n = 15$; two dogs, 13 cats), acute and chronic renal failure ($n = 10$; three dogs, seven cats), hypoadrenocorticism ($n = 4$ dogs), pseudo-hypoadrenocorticism secondary to *T. vulpis* infestation ($n = 2$ dogs), acute tumor lysis syndrome ($n = 2$; one dog, one cat), over-supplementation ($n = 2$; one dog, one cat), diabetes mellitus and pancreatitis ($n = 1$ dog), biliary obstruction ($n = 1$ dog), diabetic ketoacidosis ($n = 1$ cat), and diabetic ketoacidosis with acute renal failure ($n = 1$ cat). Fifty-nine percent (22/37) of the electrocardiographic changes were not consistent with previously reported abnormalities at the corresponding level of hyperkalemia.[2,3,7]

Four cats and one dog met the criteria for Group 1 ($K^+ = 5.5$–6.49 mEq/L). None of the ECGs in this group were consistent with previously described[2,3,7] ECG changes seen with hyperkalemia at this level. Two of the cats and one dog had a normal sinus rhythm and one cat had a normal ECG configuration and sinus bradycardia (104 bpm). The ECG of the second cat revealed a sinoventricular rhythm (124 bpm), which has previously been described to occur at higher levels of serum potassium.[2,3,7]

Two dogs met the criteria for Group 2 ($K^+ = 6.5$–6.99 mEq/L). Neither animal's ECG was consistent with ECG changes previously described[2,3,7] at this level of hyperkalemia, and both ECGs demonstrated a normal sinus rhythm.

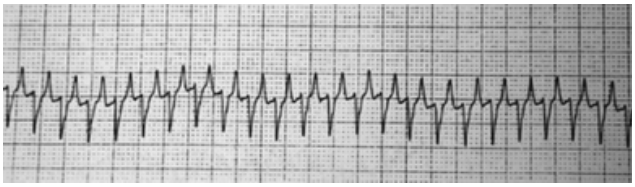

Figure 1: Ventricular tachycardia in a cat with urethral obstruction and hyperkalemia ($K^+ > 9.0$ mEq/L). Ventricular rate = 260 bpm, paper speed = 25 mm/s, 1 mV = 1 cm.

Six cats and 6 dogs met the criteria for Group 3 ($K^+ = 7.0$–8.49 mEq/L). Seventy-five percent (9/12) of the ECGs in Group 3 were not consistent with previously described[2,3,7] ECG changes at this severity of hyperkalemia. The ECGs of five cats and three dogs revealed a normal sinus rhythm. The ECG of one cat revealed a sinus tachycardia. The ECG of one dog demonstrated tall T waves, which have been previously described for this level of serum potassium.[2,3,7]

Five cats and two dogs met the criteria for Group 4 ($K^+ = 8.5$–9.99 mEq/L). Forty-three percent (3/7) of the ECGs in this group were not consistent with previously described[2,3,7] ECG changes at this elevation of serum potassium. The ECG of one cat and one dog revealed a normal sinus rhythm with heart rates of 194 and 133 bpm, respectively, and the ECG of another cat revealed ventricular tachycardia (Figure 1). The ECG of one cat was consistent with previously described changes including tall T waves, atrial standstill, and bradycardia.[2,3,7]

Seven cats and four dogs met the criteria for Group 5 ($K^+ \geq 10$ mEq/L). Twenty-seven percent (3/11) of the ECGs in Group 5 were not consistent with previously described[2,3,7] ECG changes at this elevation of serum potassium. The ECG of one dog revealed a normal sinus rhythm, the ECG of one cat revealed sinus tachycardia and the ECG of one dog revealed atrioventricular dissociation with second-degree atrioventricular block (Figure 2). The ECG of one cat revealed sinoventricular rhythm, which has been previously described.[2,3,7]

Other laboratory abnormalities documented included hyponatremia in seven of 35 patients (49%), hypochloridemia in 15 of 35 patients (43%), hypermagnesemia in 12 of 18 patients (67%), hypocalcemia in six of 30 patients (20%), and venous acidemia in 17 of 18 patients (94%) (Table 1).

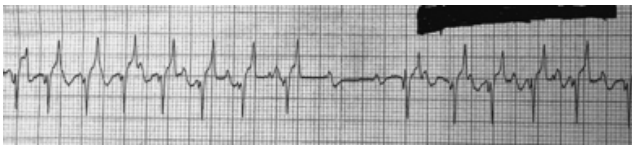

Figure 2: Atrioventricular (AV) dissociation with second degree AV block in a dog with hypoadrenocorticism and hyperkalemia (K+ = 12.1 mEq/L). Ventricular rate = 130 bpm Paper speed = 25 mm/s, 1 mV = 1 cm.

Linear regression was use to compare potassium blood level and heart rate in both dogs (Figure 3) and cats (Figure 4). There was no significant correlation ($r = 0$) between potassium blood levels and heart rate in dogs ($n = 15$) or between potassium blood levels and heart rate ($r = 0.40$; $P = 0.06$) in cats ($n = 22$). The correlation was stronger and reached significance ($r = 0.64$; $P < 0.05$) in cats with serum potassium level > 8.5 mEq/L (Groups 4 and Group 5; $n = 11$).

Discussion

Veterinarians often use ECGs as a readily available, non-invasive, cost-effective way to begin a preliminary evaluation of a dog or cat's cardiovascular stability. The ECG waveforms are created as cardiac cell action potentials depolarize or repolarize specific areas of the heart. The stimulation for a normal heart beat begins when the sinus node pacemaker cells depolarize; which, in a normal cardiac cell occurs at a resting membrane potential of approximately -90 mV. The threshold membrane potential is reached when sodium's entry into the cell exceeds that of potassium leaving the cell. This increases the cell's transmembrane potential to -60 mV. As a result an action potential is created. During phase 0, the sodium channels open leading to an influx of sodium that changes the transmembrane potential temporarily to approximately +35 mV. Repolarization then occurs during three phases until the resting membrane potential returns to -90 mV: Phase 1 – sodium channels close, Phase 2 (prolonged plateau of depolarization) – potassium channels close decreasing potassium permeability and calcium channels open allowing calcium to enter the cell, Phase 3 – potassium channels reopen allowing potassium to exit from the cell and calcium channels close stopping calcium influx, and Phase 4 – the return of the cell to the stable resting potential.[14,15]

The intracellular to extracellular potassium ratio is the primary determinate of the cardiac resting membrane potential because the potassium channels are mostly open at rest.[1,7] The concentration gradient of potassium across the cardiac cell membrane is reduced when serum potassium levels increase resulting in a less negative resting membrane potential.[14] This change in resting membrane potential should increase the cell membrane excitability because it decreases the difference between the resting and threshold potentials. However, this is not the only change that occurs when extracellular potassium levels are increased. Elevated serum potassium concentrations inactivate the sodium–potassium channels that are integral to allowing potas-

Table 1: Collected study data

Signalment	Diagnosis	Potassium (mEq/L)	ECG – heart rate	Do ECG abn. match what is expected?	Sodium (mEq/L)	Chloride (mEq/L)	Magnesium (mg/dL)	Total calcium (mg/dL)	Venous blood gas (pH)
Group 1 Serum K^+ 5.5–6.49 mEq/L									
2 yrs, M/N, Feline	Urethral obstruction	5.6	180	No	146	121			7.373
9 yrs, M/N, Feline	Iatrogenic	6.2	220	No	161	123		9.5	
12 yrs, M/I, Feline	Diabetes ketoacidosis, acute renal failure	5.8/6.3	104	No	148.5	120.2	3.35	8.03	7.113
13 yrs, F/S, Feline	Lymphosarcoma	5.68	205	No	167.3	125.6		9.99	
10 yrs, M/N, Mix canine	Acute renal failure	5.80	124	No	152.7	111.3		9.55	
Group 2 Serum K^+ 6.5–6.99 mEq/L									
2 yrs, MN, Mix canine	Hypoadrenocorticism	6.79	127	No	139.6	105.5	3.19	13.88	7.20
3 yrs, M/I, Miniature Pinscher	Urethral obstruction	6.99	110	No	128.6	90.2	>5.20	9.55	
Group 3 Serum K^+ 7.0–8.49 mEq/L									
15 yrs, F/S, Feline	Chronic renal failure	7.21	200	No	164.9	121	2.37	11.54	
14 yrs, F/S, Yorkshire Terrier	Diabetes mellitus, Pancreatitis	7.31	107	Yes	130.6	85.7	3.67	8.77	7.009
10 yrs, F/I, West High-land White Terrier	Iatrogenic	7.45	105	Yes	154.3	112.6	3.96	7.67	7.283
13 yrs, F/S, Shih Tzu	Biliary obstruction	7.50	122	No	151.3	121.5	1.76	8.8	7.008
8 yrs, F/S, Feline	Acute renal failure	7.57	200	No	153.5	118.8	0.00	iCa 1.10	7.182
3.5 yrs, F/I, Siberian Husky	Pseudo-hypoadrenocorticism	7.75	147	No	121.6	91.7	>5.2	8.35	7.114
5 yrs, F/S, Feline	Acute renal failure	7.9	200	No	152	109	4.7	9.0	7.108
Feline, M/N	Urethral obstruction	8.2	180	No					
3.5 yrs, M/N, Mix canine	Hypoadrenocorticism	8.35	169	No	136.3	104.8		10.89	
8.5 yrs, M/N, Feline	Urethral obstruction	7.5	170	No	139	112			7.236
2 yrs, M/N, Mix canine	Urethral obstruction	7.85	150	Yes	134.7	107.8		10.24	
8 yrs, M/N, Feline	Urethral obstruction	8.1	250	No	157	121		7.26	
Group 4 Serum K^+ 8.5–9.99 mEq/L									
Adult, Feline	Urethral obstruction	8.5	220	Yes	153	114	2.49	8.20	7.142
13 yrs, F/S, Toy Poodle	Acute renal failure	8.51	130	Yes	153.4	105.7		8.96	
12 yrs, M/N, Persian	Hyperosmolar diabetes ketoacidosis	8.68	194	No	156.5	120.4		9.81	
13 yrs, M/N, Mix canine	Lymphoma, acute tumor lysis syndrome	8.70	133	No	142.6	113.7	2.66	7.95	
8 yrs, M/N, Feline	Urethral obstruction	>9.0	140	Yes	135	112			7.026
6 mo. F DSH	Acute renal failure	9.73	129	Yes	149.1	105.1			
7 yrs, M/N, Feline	Urethral obstruction	>9.0	260	No	140	117			7.109
Group 5 Serum $K^+ > 10.0$ mEq/L									
10 yrs, M/N, Feline	Urethral obstruction	11.2	165	Yes	151	116	3.08		6.912
11 yrs, M/N, Feline	Acute renal failure	10.00	143	Yes	154.6	107.2		10.08	6.971
Geriatric Feline	Urethral obstruction	10.12	240	No	155.7	117.7	1.8	7.68	7.112
10 yrs, F/S, Australian Shepherd	Acute renal failure	10.48	134	No	125.1	91.3	>5.20	11.01	7.125
7 yrs, M/N, Maine Coon	Urethral obstruction	11.13	155	Yes	158.2	116.9	2.18	7.31 L	
2.5 yrs, M/N, Feline	Acute renal failure	11.17	130	Yes	152.2	107.3	4.82	11.13	7.029
6 yrs, F/S, Australian Shepherd	Hypoadrenocorticism	11.5	120	Yes	140	100		11.6	
3 yrs, M/I, Feline	Urethral obstruction	11.60	122	Yes	157.4	113.5	4.07	6.87	
6 yrs, F/S, Samoyed	Hypoadrenocorticism	12.1	170	No	138	95		11.9	
2 yrs, M/I, Chow	Pseudohypoadrenocortism	12.3	80	Yes	128	80		10.9	
Adult Feline	Acute renal failure	>13.1	102	Yes				6.77	

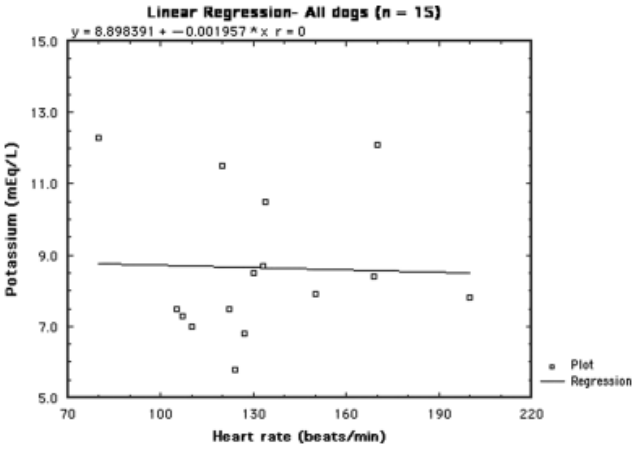

Figure 3: Linear regression analysis comparing potassium blood level and heart rate in dogs.

sium efflux during the resting phase, so cells are much slower in reaching threshold potential.[7,16] The decreased permeability of potassium will also slow the efflux of potassium out of the cell during phase 3 of repolarization, prolonging the muscle's recovery. Potassium's altered permeability ends when the slow calcium channels close and an influx of potassium returns the membrane potential to its resting state (approximately -70 mV to -80 mV during serum hyperkalemia).[3,14,17] These changes depress upstroke velocity and progressively prolong duration of the cardiac action potential as extracellular potassium concentration increases, and they explain the ECG alterations in experimentally derived hyperkalemia.[2,3,7,18]

As a dynamic system, the body has two ways to protect against hyperkalemia. The first is an intracellular shift of potassium mediated by insulin, β_2-adrenergic receptors, and potassium itself. The second compensatory mechanism in dealing with hyperkalemia involves increased renal excretion of potassium from increased activity of the potassium channels in the principle cells. This is due to a direct effect of increased serum potassium on the protein components of these channels and indirectly due to increased aldosterone release.[6,16] Once the body's ability to maintain homeostasis through potassium adaptation has been exhausted, which more commonly occurs with drastic, acute, and peracute increases in potassium, clinical evidence of hyperkalemia is more likely. Enhanced cardiotoxicity can occur with simultaneous hypocalcemia, hyponatremia, and acidemia.[1,7,16]

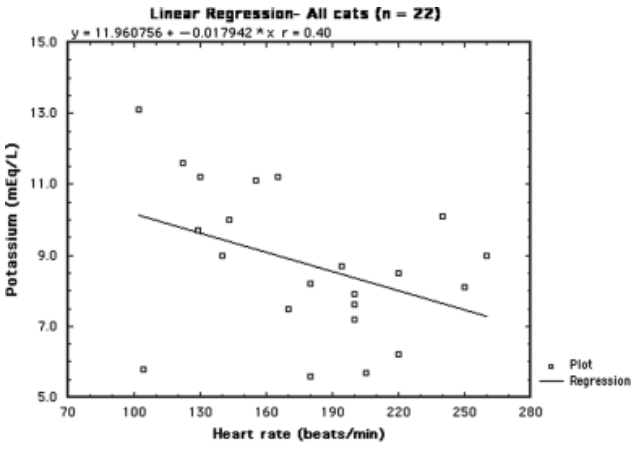

Figure 4: Linear regression analysis comparing potassium blood level and heart rate in cats.

Our hypothesis was that the ECGs of critically ill hyperkalemic dogs and cats are different than those of healthy animals with experimentally induced hyperkalemia. To evaluate this hypothesis, the animals that were selected for this study had a variety of disease processes. In turn, we anticipated that these disease processes would increase the likelihood that other abnormalities such as hypocalcemia, acidemia, hypermagnesemia, hyponatremia, and hypochloridemia would be present. The use of privately owned animals, varying severity of illness, and medical limitations posed by each animal determined what additional diagnostics and the volume of blood able to be drawn from each animal.

Another factor affecting an animal's ECG is calcium, which moves from the extracellular fluid into the cell during the plateau phase (phase 2) of the cardiac cell's action potential. Hypocalcemia prolongs phase 2 of the action potential and is postulated to down-regulate myocardial B-receptors causing weakness of the cardiac muscle. The effect of hypocalcemia on the ECG includes prolongation of the S–T segment and the Q–T interval.[7,13,17,19] Even though potassium and calcium affect different phases of the action potential, they can cause similar ECG effects. Hypocalcemia has been documented as a co-occurence in diseases with hyperkalemia,[20] and enhances the cardiotoxicity of hyperkalemia not only as mentioned above but by lowering the cell's threshold potential, which brings it closer to the resting membrane potential. The resting membrane potential is elevated (less negative) during hyperkalemia, further exacerbating the cell's hyperexcitability.[14,16] Hypocalcemia occurred in seven of 30 (23%) of dogs and cats in our study. Four of seven (57%) animals with hypocalcemia had ECG changes similar to those previously described with hyperkalemia.[2,3,7] However, eight of 12 (67%) of the animals with normal total calcium measurements had ECG changes similar to those previously described with hyperkalemia.[2,3,7] This does not clearly depict an exaggeration of ECG changes as has been described when hypocalcemia and

hyperkalemia occur simultaneously.[7,8] It is difficult to assess if a co-occurrence between other factors and ECG changes occurred in our study due to other abnormal variables present from the underlying disease process and the small population of animals that had both parameters evaluated. Ionized calcium was not measured due to the lack of availability during part of the study, but would have provided a more accurate assessment of hypocalcemia.

A second factor potentially effecting hyperkalemia and the ECG is venous acidemia, which was documented in 17 of 18 (94%) of the dogs and cats evaluated in our study. Fourteen of 17 (82%) of these animals were significantly acidemic (blood pH <7.2). Extracellular pH will directly affect cell membrane excitability by decreasing the number of β-adrenergic receptors available in cardiac nodal tissue.[9–11] Acidemia also indirectly affects cell membrane stability by causing potassium to shift extracellularly, potentially worsening any effects of pre-existing hyperkalemia and increasing ionized and total serum calcium.[1,2,8,16] The effect that blood pH has on potassium and calcium complicates assessment of whether electrocardiographic changes are due to a direct effect of the blood pH, an indirect effect via alteration of the cell's homeostasis, or both.[2] Seven of 18 (39%) animals with measured venous blood gases had ECG changes previously described with hyperkalemia. It is difficult to attach significance to venous acidemia when there are small numbers of animals within this group and multiple co-existing abnormalities.

Hypermagnesemia (>2.38 mg/dL [0.98 mmol/L] in dogs and >3.0 mg/dL [1.23 mmol/L] in cats) prolongs the QRS complex, shortens the Q–T interval, and prolongs the P–R interval.[7,8,21] Eight of 12 (67%) dogs and cats in this study had serum magnesium levels in the range where cardiovascular effects are most commonly observed (>3.6 mg/dL).[8] Some of the ECG abnormalities associated with hypermagnesemia are similar to changes with hyperkalemia. However, the shortened Q–T interval caused by hypermagnesemia may negate some of the changes expected from hyperkalemia that may explain why fewer then expected changes occurred. It is important to understand that total serum magnesium does not accurately reflect tissue or cellular levels. In addition, ionized magnesium, direct tissue assays, and measurement of intracellular magnesium are a more accurate reflection of active levels than total magnesium measurement[12,21]; however, these measurements are not widely available to private practice clinicians.

Surprisingly, hyponatremia alone does not alter the ECG even though sodium is critical for the two channels of the action potential.[7] Hyponatremia would need to be severe and inconsistent with life to exacerbate cardiotoxicity with hyperkalemia.[16] Hyponatremia was identified in 17 of 35 (49%) dogs and cats. Hyponatremia did not seem to have exacerbated the ECG alterations. Only five of 17 (29%) animals with hyponatremia revealed the characteristic ECG alterations seen with hyperkalemia. The presence of hyponatremia and its effect on homeostasis does not explain the inconsistency found between the ECGs and expected alterations with hyperkalemia.

The characteristic ECG changes described with hyperkalemia can occur with other clinical problems. Peaked T waves can occur with bradycardia, cerebrovascular accidents, left ventricular diastolic overload, subendocardial ischemia, hypothermia, and death.[2] Similarly, wide QRS complexes can occur with increased parasympathetic tone, hypoglycemia, hypothermia, hypothyroidism, and drug administration (e.g., digoxin, propranolol).[22]

Cardiac arrhythmias not previously described with hyperkalemia occurred in our study, which may provide further evidence that multiple factors are affecting the ECG. Arrhythmias including sinus tachycardia, ventricular premature contractions, and AV disassociation were revealed in 11 of 37 (30%) dogs and cats. These arrhythmias have not been described as part of the characteristic ECG changes due to hyperkalemia alone.[1–3,7] Many of these arrhythmias are often a secondary cardiac response to disruptions in cell membrane homeostasis or neuromuscular and hormonal interference. Ventricular premature complexes occur in the normal heart and are secondary to hypoxia, uremia, autonomic imbalances, infection/sepsis, inflammation, nutritional deficiencies, or thyrotoxicosis, many of which occurred in the disease processes in this study.[17]

Statistical analysis of laboratory values such as sodium, chloride, magnesium, calcium, and pH was difficult due to the small size of the study. This limitation was further pronounced when groups of dogs and cats were further divided into groups that had ECG tracings consistent or not consistent with those previously reported with hyperkalemia.[2,3,7] It is impossible to make an accurate assessment of the contributions these abnormalities have in regards to changes in the ECG secondary to hyperkalemia without more data.

Our study revealed that the ECGs of dogs and cats with a variety of disease processes do not follow a definitive sequence of changes with hyperkalemia and it is not reasonable to expect ECG derangements to follow the patterns previously described in healthy animals with experimentally induced hyperkalemia. Animals at all levels of serum hyperkalemia revealed normal ECGs, which should not influence the clinician's confidence regarding an unmeasured patient's serum potassium level. Some of the ECGs revealed sinus

tachycardia or other arrhythmias not previously described with serum hyperkalemia. This variation in ECG changes that occur with hyperkalemia has not been previously described in the literature but are clinically observed. The data collected during our study confirmed that diseased animals have multiple abnormalities other than serum hyperkalemia that may play a role in cardiac electrophysiology. Hopefully, in the future, a correlation between the other abnormalities identified with ill animals and the ECG alterations seen will lead us to a greater understanding and therapeutic approach in critically ill patients.

Acknowledgements

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Footnotes

[a] I-stat, Heska Corporation, Fort Collins, CO.
[b] IDEXX VetLab Analyzer #8008, IDEXX Laboratories Inc., Westbrook, ME.
[c] GB-Stat for Macintosh, Dynamic Microsoft Systems Inc., Silver Spring, MD.

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