Decreased sodium:potassium ratios in cats: 49 cases

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Background: Sodium:potassium (Na:K) ratios are often reported in feline biochemical panels, although the importance of this measurement has not been investigated. **Objectives:** The aims of this study were to document the range of feline disease states associated with a decreased Na:K ratio, to determine the prevalence of this biochemical abnormality in a referral hospital population, and to identify any particular disease that was more likely to have a decreased Na:K ratio. **Methods:** A group of 49 cats with decreased Na:K ratios was compared with a group of 50 cats with normal Na:K ratios that were randomly selected from the same hospital population. **Results:** Twelve of the 49 cats (24.5%) had gastrointestinal disease, 10 (20.4%) had urinary disease, 8 (16.3%) had endocrine disease, 8 (16.3%) had cardiorespiratory disease, and 5 (10.0%) had diseases affecting other body systems. Six (12.2%) had artifactually decreased Na:K ratios. No cat was identified with hypoadrenocorticism. Statistical analysis revealed that, although none of these disease states was significantly over- or under-represented in the affected group, a significantly higher proportion of cats with decreased Na:K ratio had body cavity effusions ($P = .025$). Serum potassium concentrations were significantly higher in the affected group ($P < .0001$), but there was no significant difference in mean sodium concentration between the 2 groups. **Conclusions:** Decreased Na:K ratios frequently occur in cats with diseases other than hypoadrenocorticism, including cats with effusions. These findings should be considered when evaluating cats with this biochemical abnormality. (*Vet Clin Pathol.* 2005;34:110–114)

Key Words: Addison's disease, effusion, electrolytes, endocrine diseases, hypoadrenocorticism

A decreased sodium:potassium (Na:K) ratio can result from hyponatremia or hyperkalemia. Plasma sodium and potassium concentrations are normally under tight homeostatic control,[1] and alterations in the concentrations of either sodium or potassium can have life-threatening consequences. Hyperkalemia can result in generalized muscle weakness and slowed cardiac conduction with bradycardia leading to ventricular fibrillation or asystole. Hyponatremia can result in cerebral edema.[2]

The expression of the concentrations of these electrolytes as a ratio has been used in the diagnosis of adrenocortical insufficiency in both the dog[3] and the cat.[4] However, several other conditions also have been reported in association with a decreased Na:K ratio in both species.[5–7] In case reports of dogs, decreased Na:K ratios have been described with such diseases as hypoadrenocorticism,[3] gastrointestinal disease,[5,8] renal disease,[9] pleural effusions,[6,10] peritoneal effusions,[11] and pregnancy.[12] In cats, decreased Na:K ratios have been most often described in hypoadrenocorticism[4,13] and urethral obstruction.[14] Reports of this biochemical abnormality in cats affected with other disease conditions are sparse. Decreased Na:K ratios have been reported in cats with peritoneal effusions,[7] following repeated thoracic drainage of an idiopathic chylothorax,[15] and uterine torsion.[16]

There have been relatively few surveys of dogs with decreased Na:K ratios. In a study by Neiger and Gunderson,[17] 14 of 50 dogs had hypoadrenocorticism (28%). Urinary tract disease (20%), pleural and pericardial effusions (12%), gastrointestinal disease (8%), and a variety of other diseases (20%) including diabetes mellitus and drug ingestion were also reported. A smaller study by Roth and Tyler[18] found 14 of 34 dogs had urinary tract disease (41%). Hypoadrenocorticism (24%), pancreatic disease (12%), disseminated neoplasia (9%), and circulatory disease (6%) were also recorded. To our knowledge, there are no published surveys of cats with decreased Na:K ratios.

Decreased Na:K ratios are not always associated with disease. Marked hyperproteinemia or hyperlipidemia may cause spurious underestimation of plasma or serum sodium concentrations if the assay is performed using flame photometry or with indirect potentiometry with ion-selective electrodes.[19] Specimen contamination with potassium EDTA can cause marked pseudohyperkalemia or pseudohypocalcemia.[20]

It is apparent that decreased Na:K ratios do occur in cats, but their prevalence and disease associations have not been investigated as fully as in dogs. The first aim of this study was to identify the prevalence and diseases associated with this biochemical abnormality in a hospital-based population. The

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Table 1. The number of cats in each disease subgroup based on Na:K ratio. Indicated in parentheses are the number of cats in the group with effusions and the site of the effusion.

Subgroup	Affected Cats (Na:K <27)	Control Cats (Na:K >27)
Gastrointestinal	12 (2 ascites)	10 (1 ascites)
Urinary	10 (1 ascites, 1 pleural)	7
Endocrine	8	15
Cardiorespiratory	8 (4 pleural)	7
Other	5	11
Artifact	6	0
Total	49	50

second aim was to identify whether any disease state was significantly associated with a decreased Na:K ratio.

Materials and Methods

Clinical records of cats presenting to the University of Glasgow Small Animal Hospital (UGSAH) from May 2000 to February 2003 were searched. Criteria for inclusion were a Na:K ratio below the reference value (<27) and complete clinical records. A separate group of 50 cats with a Na:K ratio within the reference interval and with complete clinical records was randomly selected from the same hospital-based population to serve as controls.

Blood samples were collected from all cats into tubes containing potassium EDTA for a CBC, lithium heparin for plasma biochemistry, and sodium fluoride for plasma glucose concentration. All cats had a CBC performed using a Cell-Dyn hematology analyzer (Abbott Laboratories, North Chicago, IL, USA) and a plasma biochemical profile using an Olympus AU600 biochemistry analyzer (Olympus Corporation, Tokyo, Japan). All plasma sodium and potassium measurements were made by indirect potentiometry using ion-selective electrodes on diluted samples (1:25 dilution). Further diagnostic testing was performed on an individual basis as deemed appropriate to achieve a clinical diagnosis.

To facilitate comparisons, cats were grouped into 6 categories based on the primary organ system involved or the suspected artifactual error. No cat was placed in more than 1 group. The presence of body cavity effusions was recorded separately. Results were deemed to be artifactual if the samples were lipemic or hyperproteinemic or if the results appeared to indicate EDTA contamination. The results of repeat samples, when available, were assessed if artifactual changes were suspected. Hypoadrenocorticism was excluded in each cat with a decreased Na:K ratio using at least 2 of the following 3 criteria: 1) adrenocorticotropic hormone stimulation test results incompatible with hypoadrenocorticism, 2) lack of appropriate clinical signs, and 3) resolution of the decreased Na:K ratio following therapy that did not include either mineralocorticoids or glucocorticoids. Cats were excluded from the study if they were receiving exogenous potassium at the time of sampling.

Comparisons with control cats were made using χ^2, Fisher's exact, or 2 sample *t*-tests, as appropriate. Significance was assumed when $P < .05$.

Results

Between May 2000 and February 2003, 1636 cats had plasma sodium and potassium concentrations assayed. Fifty-two cats had a Na:K ratio of <27 (a prevalence of 3.1%). Complete clinical records were available for 49 of these cats. No cat presenting to the UGSAH during the study period was diagnosed with hypoadrenocorticism. No cat with a decreased Na:K ratio was receiving exogenous potassium at the time of sampling. There were no associations between age, breed, sex, results of feline immunodeficiency virus or feline leukemia virus tests and a decreased Na:K ratio when affected cats were compared with control cats.

Gastrointestinal disease

Twelve of the 49 cats (24.5%) with decreased Na:K ratio had gastrointestinal disease (Table 1). All of the cats in this group had either vomiting or diarrhea with inappetance. One cat tested positive, by polymerase chain reaction, for feline panleukopenia virus. No cat had confirmed gastrointestinal parasitism. Two cats had ascites; both were hyponatremic. One cat had a rectal stricture.

Urinary disease

Ten cats (20.4%) with a decreased Na:K ratio had urinary disease, 8 with primary renal disease and 2 with urinary outflow obstruction (Table 1). Four of the cats with primary renal disease had end-stage renal failure. Only 1 cat (with urethral obstruction and pleural effusion secondary to pulmonary trauma) had hyponatremia. One cat had ascites. Two of the cats with renal disease that were were included in the urinary disease subgroup also had some clinical evidence of gastrointestinal dysfunction. Two animals with renal disease were receiving angiotensin-converting enzyme (ACE) inhibitors.

Endocrine disease

Eight cats (16.3%) had endocrine disease, 5 with diabetes mellitus and 3 with hyperthyroidism (Table 1). Among the 5 cats with diabetes mellitus, 2 were both hyponatremic and hyperkalemic. Three of the 5 cats with diabetes mellitus had increased plasma concentrations of urea (18.2–25.9 mmol/L, reference interval 2.7–9.2 mmol/L) and creatinine (215–257 μmol/L, reference interval 91–180 μmol/L), and a fourth cat had a moderate increase in urea concentration alone (22.9 mmol/L). The fifth cat was not azotemic. One of the 3 cats with hyperthyroidism was azotemic (urea 17.9 mmol/L, creatinine 225 μmol/L), another cat had a moderate increase in serum urea concentration (13.9 mmol/L), and the third cat had protein-losing nephropathy. Two of the cats with

hyperthyroidism also had thyrotoxic cardiomyopathy; 1 of these was receiving ACE inhibitors and atenolol.

Cardiorespiratory disease

Eight cats (16.3%) had cardiorespiratory disease (Table 1). Five had primary cardiac disease, 3 of which had pleural effusion (modified transudate) secondary to congestive heart failure. Two of the cats had a distal aortic thromboembolus. Two cats had hyponatremia, of which 1 had radiographic evidence of congestive heart failure and the other was receiving furosemide. Three of the cats with cardiac disease were receiving ACE inhibitors and 2 were receiving furosemide. The 3 remaining cats were diagnosed with bacterial bronchopneumonia, idiopathic chylothorax, and suspected pulmonary contusion respectively. Six of the 8 cats had increased plasma urea concentrations (10.6–18.5 mmol/L); however, creatinine concentrations were within the reference interval. Urine specific gravity was recorded in 1 of these cats and was hypersthenuric (specific gravity >1.050).

Body cavity effusions

Eight cats (16.3%) with a decreased Na:K ratio had body cavity effusions. Of these, 5 had pleural effusions. One cat had idiopathic chylothorax. Pleural effusions in the remaining 4 cats were modified transudates secondary to congestive heart failure (3 cats) and pulmonary trauma (1 cat). Three cats had peritoneal effusions, including 1 with uroperitoneum secondary to bladder rupture, 1 with high protein ascites secondary to a Budd-Chiari–like syndrome, and 1 with a modified transudate secondary to hepatic carcinoma. Effusions had not been drained before blood sampling in any of the cats. Four of these cats had both hyponatremia and hyperkalemia. Two cats with ascites were receiving furosemide at the time of sampling; both of these cats were hyponatremic. Only 1 of the cats in the control group had a body cavity effusion; this cat had abdominal effusion because of bacterial peritonitis. There was a significant association between effusive diseases and a decreased Na:K ratio ($P = .025$).

Other system involvement

Five cats (8%) were grouped as having "other" system involvement. Two cats had ocular disease, 1 had neurological disease, 1 had a dislocated hock, and 1 had atopic dermatitis. Comparison with the control group revealed that none of these other disease states were significantly over- or underrepresented within the group.

Artifactual results

Six cats (12.2%) had an artifactually decreased Na:K ratio. One sample was grossly lipemic (serum triglyceride concentration 5.93 mmol/L; reference interval <0.6 mmol/L) because of diabetes mellitus. Repeat samples obtained from this cat showed plasma sodium, calcium, and potassium concentrations within reference intervals, so the cat was not included in the endocrine group. The remaining 5 cats all had profound hypocalcemia (total plasma calcium 0.29–0.96 mmol/L, reference interval 1.60–2.56 mmol/L) and hyperkalemia (7.5–8.5 mmol/L, reference interval 2.6–5.2 mmol/L) consistent with EDTA contamination of the samples. None of these cats had clinical signs of disease or detectable underlying disorders that may have caused these electrolyte imbalances. In 3 of these 5 cats, the plasma sodium, calcium, and potassium concentrations were immediately reassayed on a fresh sample and found to be within reference intervals.

Sodium and potassium concentrations

There was a significant difference ($P < .001$) between mean serum potassium concentrations of affected and control cats. All of the cats with decreased Na:K ratios had hyperkalemia. Only 1 of the cats in the control group had hyperkalemia (5.5 mmol/L, reference interval 2.6–5.2 mmol/L), with a Na:K ratio of 27.3. There was no significant difference in the mean serum sodium concentrations of affected and control cats. Nine cats with decreased Na:K ratios had hyponatremia and hyperkalemia; of these, 2 had insulin-dependent diabetes mellitus, 3 had gastrointestinal disease (1 with abdominal carcinoma, 1 with Budd-Chiari–like syndrome, and 1 with a rectal stricture), 2 had cardiac disease (hypertrophic cardiomyopathy), 1 had urinary disease (traumatic abdominal hernia with bladder entrapment), and 1 had artifactual hyponatremia due to sample lipemia. Four of these 9 cats had body cavity effusions. The sodium concentrations in cats with effusions ranged from 125.0–143.8 mmol/L (reference interval 145–160 mmol/L). Four control cats had mild hyponatremia, with sodium values between 140–144 mmol/L.

Discussion

In this study, hyperkalemia was the most common cause of a decreased Na:K ratio in cats, whereas hyponatremia was a relatively uncommon finding. In contrast, in a survey of decreased Na:K ratios in 34 dogs,[18] all of the dogs had hyperkalemia and 53% had hyponatremia.

Almost a quarter of the cats with a decreased Na:K ratio had gastrointestinal disease. The majority of dogs with decreased Na:K ratios and gastrointestinal disease have gastrointestinal parasitism.[5,8,21] A similar finding was not observed in the cats in this study. The development of Na:K imbalances in gastrointestinal disease in small animals has been linked to hypovolemia as a result of the loss of isotonic, sodium- and bicarbonate-rich fluid with compensatory free-water retention resulting in hyponatremia and acidosis. Increased iso-osmotic sodium and water reabsorption in the proximal tubule will occur secondary to activation of the renin-angiotensin-aldosterone system (RAAS) and antidiuretic hormone (ADH) release. This results in decreased sodium delivery to the distal renal tubules and inappropriately low kaliuresis because of a decrease in both the concentration and electrochemical gradients for potassium excretion into the distal tubular lumen.[1] An inability to excrete potassium in

addition to transcellular shifts of potassium in response to acidosis can result in hyperkalemia.

Nineteen percent of cats with a decreased Na:K ratio in this study had urinary disease, including 4 with end-stage renal failure. Because distal tubular flow rate is one of the major determinants of urinary potassium excretion,[1] animals with renal disease that have a sudden reduction in urine output due to the progression of their renal disease can become hyperkalemic.[22] Hyperkalemia, possibly as a result of an acute reduction in glomerular filtration rate and kaliuresis, is a common finding in cats with urethral obstruction,[14] and a likely mechanism for the hyperkalemia observed in the 2 cats with urinary tract obstruction.

Sixteen percent of cats with a decreased Na:K ratio in this study had endocrinopathies. The majority had diabetes mellitus, and a number of these cats also were azotemic. Decreased Na:K ratios have been recorded in canine diabetes mellitus.[17] Increased plasma osmolality has been thought to be responsible for hyponatremia and hyperkalemia in diabetic animals[2]; however, compromised renal function resulting in a failure to excrete potassium may also be responsible in some cases. In surveys of diabetic cats, hyperkalemia has been an uncommon finding,[23] but when it does occur, it has been associated with concurrent kidney disease.[24]

In the cats with cardiorespiratory disease, hyperkalemia and hyponatremia could have occurred via a variety of mechanisms. A loss of effective circulating volume in congestive heart failure can result in dilutional hyponatremia secondary to activation of the RAAS, ADH release, and free-water retention.[2] The loss of effective circulating volume in advanced cardiac disease also can result in renal underperfusion and a consequent azotemia.[25] A reduced distal tubular flow rate and reduced sodium delivery can compromise the kidney's ability to excrete potassium,[1] resulting in hyperkalemia. A high proportion of the cats in the cardiorespiratory group were azotemic, suggesting that this is an important mechanism. Two cats with cardiac disease had a distal aortic thrombus. Hyperkalemia is a recognized complication of this condition[26] and results from reperfusion of lysed muscle tissue downstream to the thrombus and concurrent metabolic acidosis.

Effusive disorders are a recognized cause of decreased Na:K ratios in dogs[6] and cats[7] through mechanisms described above for gastrointestinal diseases. Sixteen percent of cats in this case series had body cavity effusions, which is a similar prevalence to that reported in the dog.[17] This prevalence of effusions in cats with decreased Na:K ratios was significantly higher than that in the control group. Furosemide administration may have accounted for hyponatremia in 2 of the cats with ascites. Five cats had pleural effusions without ascites or pericardial effusion. Decreased Na:K ratios have not previously been reported in cats with pleural effusions alone.

In 6 cats, the decreased Na:K ratio was thought to be artifactual and caused by either marked lipemia or EDTA contamination. EDTA binds calcium and other divalent ions, causing artifactual hypocalcemia.[27] The dipotassium salt of EDTA can cause artifactual hyperkalemia. EDTA is an anticoagulant used for routine hematological studies, and all affected cats had samples submitted in EDTA concurrent with the submission of samples in lithium heparin for plasma biochemical analysis. The lack of appropriate clinical signs or underlying etiology for the electrolyte imbalances, as well as the findings on repeat samples obtained from 3 of the cats, supported the likelihood that these changes were artifactual.

In dogs, hypoadrenocorticism is one of the most common causes of decreased Na:K ratio.[17,18] In 10 cats with spontaneous primary hypoadrenocorticism reported by Peterson et al,[4] all cats had a decreased Na:K ratio. Hypoadrenocorticism was excluded as a cause of a decreased Na:K ratio in all of the cats in this study. Furthermore, no cat presenting to the UGSAH during the study period was diagnosed with hypoadrenocorticism. It is reasonable to suggest that not only is hypoadrenocorticism a rare condition in cats,[28] but it also is a rare cause of decreased Na:K ratio in this species.

The allocation of cats into various subgroups was made on the basis of the primary body system involved. These subgroups were derived from previous studies in dogs[17] and also from the relevant literature. However, many of the cats had multiple disease processes that may have had additive contributions to their decreased Na:K ratio. Furthermore, many of the cats were receiving medication that may have contributed to their decreased Na:K ratio. In particular, ACE inhibitors, by blocking the formation of angiotensin, may reduce the secretion of aldosterone sufficiently to produce hyperkalemia.[1] Diuretics such as furosemide can result in natriuresis and hyponatremia,[1] which may account for the hyponatremia observed in some of the cats receiving this drug. Atenolol can also potentially cause hyperkalemia[29] and was being given to a cat with hyperthyroidism. From the high percentage of cats with decreased Na:K ratios that had multiple disease conditions or concurrent medications, it was concluded that decreased Na:K ratio was most likely to occur when multiple stresses are placed on sodium and potassium regulatory mechanisms.

In conclusion, a number of disease states other than hypoadrenocorticism, particularly effusions, appear to be associated with decreased Na:K ratios in cats. When more than 1 of these disease states is present, then the likelihood of a decreased ratio seems to increase. Given the potentially serious clinical consequences of either hyponatremia or hyperkalemia, the findings in this study emphasize the importance of monitoring ill cats for this biochemical abnormality. However, the lack of specificity of a decreased Na:K ratio for any disease state seriously limits its use as a diagnostic tool in feline medicine.

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References

1. DiBartola SP, Autran De Morais H. Disorders of potassium: hypokalemia and hyperkalemia. In: DiBartola SP, ed. *Fluid Therapy in Small Animal Practice.* 2nd ed. Philadelphia, PA: WB Saunders Co; 1992:83–107.

2. DiBartola SP. Disorders of sodium and water: hypernatremia and hyponatremia. In: DiBartola SP, ed. *Fluid Therapy in Small Animal Practice.* 2nd ed. Philadelphia, PA: WB Saunders Co; 1992:45–72.
3. Peterson MA, Kintzer PP, Kass PH. Pretreatment clinical and laboratory findings in dogs with hypoadrenocorticism—225 cases. *J Am Vet Med Assoc.* 1996;208:85–91.
4. Peterson ME, Greco DS, Orth DN. Primary hypoadrenocorticism in 10 cats. *J Vet Intern Med.* 1989;3:55–58.
5. DiBartola SP, Johnson SE, Davenport DJ, et al. Clinicopathologic findings resembling hypoadrenocorticism in dogs with primary gastrointestinal disease. *J Am Vet Med Assoc.* 1985;187:60–63.
6. Willard MD, Fossum TW, Torrance A, et al. Hyponatremia and hyperkalemia associated with idiopathic or experimentally induced chylothorax in 4 dogs. *J Am Vet Med Assoc.* 1991;199:353–358.
7. Bissett SA, Lamb M, Ward CR. Hyponatremia and hyperkalemia associated with peritoneal effusion in four cats. *J Am Vet Med Assoc.* 2001;218:1590–1592.
8. Ruckstuhl N, Hoerauf A, Tomsa K, et al. Pseudohypoadrenocorticism in 2 Siberian Huskies with gastrointestinal parasitosis. *Schweiz Arch Tierheilkd.* 2002;144:75–81.
9. Willard MD, Refsal K, Thacker E. Evaluation of plasma aldosterone concentrations before and after ACTH administration in clinically normal dogs and dogs with various diseases. *Am J Vet Res.* 1987;48: 1713–1718.
10. Zenger E. Persistent hyperkalemia associated with nonchylous pleural effusion in a dog. *J Am Anim Hosp Assoc.* 1992;191:411–413.
11. Kitchell BE, Fan TM, Kordick D, et al. Peliosis hepatitis in a dog infected with *Bartonella henselae*. *J Am Vet Med Assoc.* 2000;216:519–523.
12. Schaer M, Halling KB, Collins KE, et al. Combined hyponatremia and hyperkalemia mimicking acute hypoadrenocorticism in 3 pregnant dogs. *J Am Vet Med Assoc.* 2001;218:897–899.
13. Parnell NK, Powell LL, Hohenhaus AE, et al. Hypoadrenocorticism as the primary manifestation of lymphoma in two cats. *J Am Vet Med Assoc.* 1999;214:1208–1211.
14. Finco DR, Cornelius LM. Characterization and treatment of water, electrolyte, and acid base imbalances of induced urethral obstruction in the cat. *Am J Vet Res.* 1997;38:823–830.
15. Thompson MD, Carr AP. Hyponatremia and hyperkalemia associated with chylous pleural and peritoneal effusion in a cat. *Can Vet J.* 2002;43: 610–613.
16. Ridyard AE, Welsh EA, Gunn-Moore DA. Successful treatment of uterine torsion in a cat with severe metabolic and haemostatic complications. *J Feline Med Surg.* 2000;2:115–119.
17. Neiger R, Gundersen HC. Decreased sodium potassium ratio in dogs: 50 cases. In: *Proceedings of the 46th British Small Animal Veterinary Association Annual Congress.* Birmingham, United Kingdom; 2003:528.
18. Roth L, Tyler RD. Evaluation of low sodium:potassium ratio in dogs. *J Vet Diagn Invest.* 1999;11:60–64.
19. Stockham SL, Scott MA. Monovalent electrolytes and osmolality. In: Stockham SL, Scott MA, eds. *Fundamentals of Veterinary Clinical Pathology.* Ames, IA: Iowa State Press; 2002:340–349.
20. Naguib MT, Evans N. Combined false hyperkalemia and hypocalcemia due to specimen contamination during routine phlebotomy. *South Med J.* 2002;95:1218–1220.
21. Malik R, Hunt G, Hinchliffe J, et al. Severe whipworm infestation in the dog. *J Small Anim Pract.* 1990;31:185–188.
22. Vaden SL. Differentiation of acute from chronic renal failure. In: Bonagura JD, ed. *Kirk's Current Veterinary Therapy; Small Animal Practice.* 13th ed. Philadelphia, PA: WB Saunders; 2000:856–858.
23. Crenshaw KL, Peterson ME. Pretreatment clinical and laboratory evaluation of cats with diabetes mellitus: 104 cases (1992–1994). *J Am Vet Med Assoc.* 1989;209:943–949.
24. Schaer M. A clinical survey of thirty cats with diabetes mellitus. *J Am Anim Hosp Assoc.* 1977;13:23–27.
25. The COVE study group. Controlled clinical evaluation of enalapril in dogs with heart failure: results of the cooperative veterinary enalapril study group. *J Vet Intern Med.* 1995;9:243–252.
26. Fox PR. Feline cardiomyopathies. In: Fox PR, Sisson D, Moise NS, eds. *Textbook of Canine and Feline Cardiology: Principles and Clinical Practice.* 2nd ed. Philadelphia, PA: WB Saunders; 1999:621–678.
27. Stockham SL, Scott MA. Basic hematologic assays. In: Stockham SL, Scott MA, eds. *Fundamentals of Veterinary Clinical Pathology.* Ames, IA: Iowa State Press; 2002:31–48.
28. Reusch CE. Hypoadrenocorticism. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine.* 5th ed. Philadelphia, PA: WB Saunders; 2000:1488–1499.
29. Kintzer PP. Differential diagnosis of hyperkalemia and hyponatremia in dogs and cats. In: Bonagura JD, ed. *Kirk's Current Veterinary Therapy: Small Animal Practice. 13th ed.* Philadelphia, PA: WB Saunders; 2000:374–376.