

Buffy Coat and Serum Concentration of Potassium Related to the Acid-base Status in Dogs Following Induced Hypokalemia

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Abstract

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Electrolyte and acid-base disturbances are frequent in sick dogs. The objective of this study was to determine the concentration of K^+ in blood serum and buffy coat K^+ (BC K^+) and its relation to other serum electrolytes and acid-base values in blood of healthy dogs and those with hypokalemia induced by furosemide. The study was performed on 42 dogs of both sexes, aged 3 to 8 years, divided into two groups. Group 1 (control, $n = 21$) included healthy dogs, Group 2 (experimental, $n = 21$) consisted of healthy dogs in which hypokalemia had been induced using furosemide at a dose of 12 mg/kg PO BID during 21 days. Physical examination, haemogram (16 analytes), biochemical profile (22 analytes) and urinalysis (15 analytes) were performed to evaluate the health status of all dogs. In this study, the most important serum analytes were Na^+ , K^+ and Cl^- ; BC K^+ and blood pH, HCO_3^- , base excess (BE), as well as pCO_2 . A method for obtaining BC and determining intracellular K^+ was developed. Between groups 1 and 2, serum K^+ had a significant correlation with pH, HCO_3^- and BE ($r > 0.6$, $p < 0.01$); in addition, in Group 2 there was a significant correlation with Na^+ and Cl^- ($r > 0.7$, $p < 0.01$), due to the furosemide effect. The BC K^+ presented low correlation with serum K^+ , as well as with other variables, and thus could not be considered a good indicator of intracellular K^+ . The correlation coefficients in groups 1 and 2 were significant between strong ion difference (SID), K^+ , Cl^- , HCO_3^- and BE ($r > 0.7$, $p < 0.01$), suggesting SID as a good indicator of K^+ translocation in metabolic alkalosis. The anion gap (AG) did not present significant correlation with BC K^+ , with serum Na^+ , K^+ and Cl^- , or with pH, HCO_3^- , BE and pCO_2 . For the evaluation of electrolyte alterations and metabolic acid-base disorders it is necessary to include in laboratory profiles the determination of K^+ , Na^+ , Cl^- , HCO_3^- in blood serum, as well as to calculate AG, SID, and relate them to the physical examination of the animal.

Dog, furosemide, buffy coat potassium, serum electrolyte, acid-base status

Acid-base and electrolyte disturbances are frequently present in sick dogs. Approximately 95% of the total potassium (K^+) is intracellular, of which 60 to 75% is contained in the muscles, thus causing it to be the main ion at this level (MacIntire 1997; Phillips and Polzin 1998). K^+ maintains intracellular volume and a normal membrane potential (Bilbrey et al. 1973; Field et al. 1987). In healthy animals, daily intake of K^+ equals the losses. In extracellular fluid (ECF) the average sodium (Na^+) concentration is 150 mmol/L and that of K^+ is 4 mmol/L. In dogs, the $Na^+ : K^+$ proportion in intracellular fluid (ICF) is inverted. Experimental studies in healthy dogs have muscle tissue Na^+ and K^+ levels ranging from 8.4 to 13.7 mmol/L and 139 to 142 mmol/L, respectively (Phillips and Polzin 1998).

One study, in dogs, determined total K^+ concentration to be 47.1 mmol/kg of body weight, ranging from 39.8 to 61.1 mmol/kg (Di Bartola and De Moraes 2000). The normal proportion of extracellular and intracellular potassium is maintained by the sodium-

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potassium pump found in cell membranes. This pumps Na^+ ions out and K^+ ions into the cell at a 3:2 ratio (Di Bartola and De Morais 2000; Hendrix and Raffae 1994). The ability of specialized cells to develop a membrane potential is crucial for normal cardiac conduction, muscular contraction and the transmission of nervous impulses (Di Bartola and De Morais 2000; Kornegay 1995). K^+ homeostasis in the organism is maintained through dietary intake and excretion in urine (Di Bartola and De Morais 2000). Changes in pH can produce K^+ translocation between ICF and ECF. In general, acidosis is associated with the movement of K^+ from ICF to ECF, while during alkalosis K^+ ions move in the opposite direction (Ilkiw et al. 1989; Schaer 1993; Di Bartola and De Morais 1993; Di Bartola et al. 1994).

Hypokalemia is one of the most frequently encountered electrolyte disturbances in critical patients. It presents when there is a lack of nutrients, such as in anorexia, or when there are excessive losses during vomiting (Bellenger et al. 1990). Polyuria also causes hypokalemia, be it from chronic renal failure or any type of polyuric diuresis, such as: release of a urinary tract obstruction, nephrogenic diabetes insipidus (Phillips and Polzin 1998; Di Bartola and De Morais 2000); prostatic abscesses or pyometra, both of which cause *E. coli* toxin deposition in renal tubules with subsequent interference on Na^+ and Cl^- absorption; medullary washout; and the use of loop diuretics (Di Bartola and De Morais 2000). Finally, hypokalemia is also seen with translocation of potassium into ICF during respiratory or metabolic alkalosis (MacIntire 1997).

In animals with diabetic ketoacidosis, the level of K^+ in serum can drop after insulin treatment, since this favors the entrance of potassium into the ICF. Correcting the acidotic state also reverts the translocation; thus the importance of monitoring serum K^+ in diabetic patients (Schaer 1993a). Hypokalemia, induced by K^+ translocation to ICF, is observed in dogs with hyperventilatory respiratory alkalosis (Muir et al. 1990). Both in hypo- and hyperkalemia, the proportion of intracellular (IC) and extracellular (EC) K^+ is altered, and the membrane potentials in excitable heart tissue, nerves and muscles are affected, producing altered conduction rates (MacIntire 1997). The reference range for kalemia in dogs is from 3.92 to 5.54 mmol/L, thus defining hypokalemia as values lower than those referred and hyperkalemia as values higher than reference values (Kirk 1980; Núñez 2001).

The only practical measurement of the change of body K^+ is the concentration of K^+ in serum; representing only 5% of the total given that 95% is found in the IC level (Phillips and Polzin 1998). With this in mind, the aim of the present study was to determine the relationship between K^+ in the buffy coat (BC) with that observed in serum, as well as of both of these with the acid-base evaluation in healthy dogs and those in which hypokalemia had been induced.

Materials and Methods

Forty-two clinically healthy dogs of both sexes, aged three to eight years, were divided into two groups. Group 1 (control) consisted of 21 healthy dogs, ten German shepherds (seven males and three females) and 11 mixed-breed dogs (three females and eight males). Physical examination, haemogram (16 analytes), biochemical profile (22 analytes) and urinalysis (15 analytes) were performed to evaluate the health status of all dogs. None of them showed any alterations in the haematological indices, serum biochemistry or urinalysis (Lees et al. 1994; Núñez 2001). Group 2 (experimental) was made up of 21 mixed-breed dogs (nine females and 12 males) in which hypokalemia (< 3.8 mmol/L) had been induced using furosemide at a dosage rate of 12 mg/kg PO BID during 21 days (Bonagura et al. 2000; Senior 1995).

Blood samples were obtained from either the jugular or cephalic vein, after an eight-hour fast. The urine samples were collected via catheterization or cystocentesis. Procedures were developed to obtain the buffy coat (BC) and determine intracellular K^+ . Venous blood samples were collected in vacuum tubes (Vacutainer®) containing sodium heparin. Blood components were separated using a relative centrifugal force (RCF) of 1200 g during 10 min, thus obtaining red blood cells, buffy coat and plasma. The plasma was decanted and approximately one mL of BC containing red blood cells was removed to fill a Wintrobe tube. A second centrifugation at 1200 g for 10 min was carried out, this separated the components leaving the red blood cells on the bottom and the buffy coat on the

top. A Hamilton syringe was used to obtain 20 μL of BC, to which 380 μL of demineralized water was added in microtubes, thus obtaining a 1/20 dilution.

Intracellular K^+ was obtained by successively freezing at $-10\text{ }^\circ\text{C}$ and heating to $60\text{ }^\circ\text{C}$ three times, thus causing cell destruction. The cellular destruction in the BC was confirmed through cytological examination of each sample in which cell debris was the only finding, thus indicating complete cell destruction. The concentration of K^+ was determined by means of Electrolyte analyzer, Model 644 (Ciba Corning). To confirm procedure reliability, a ten-minute estimate of the same sample was carried out.

Acid-base status was determined in blood taken in a heparinized syringe, removing air bubbles and the needle was inserted into a rubber stopper to avoid contact with atmospheric air and then the syringe was immersed in a mixture of ice and water. These samples were analysed by pH/blood gas analyser, Model 238 (Ciba Corning) within 1 h after blood sampling (obtaining pH, partial pressure of CO_2 (pCO_2), bicarbonate (HCO_3^-) and base excess (BE).

The biochemical profile was carried out on blood obtained in plain vacuum tubes (Vacutainer[®]). After the clot had fully formed, the samples were centrifuged at 1200 g during 10 min and serum was transferred into microtubes. In the following 4 hours a Biochemical analyser (Cobas Mira, Roche Switzerland) was used to determine the following serum analytes: glucose, urea, creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatin kinase (CK), total proteins, albumin, globulins, calcium, phosphate and total CO_2 (TCO_2), while potassium (K^+), sodium (Na^+) and chloride (Cl^-) were determined by the electrolyte analyzer model 644 Ciba Corning). Anion gap (AG) was calculated using the following formula: $\text{AG} = (\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$. The strong ion difference (SID) was calculated by clinical approach and corresponded to the difference between Na^+ and Cl^- (Russell et al. 1996).

Venous blood samples for haemogram (packed cell volume, haemoglobin, red blood cell count, mean corpuscular volume, mean corpuscular haemoglobin concentration, reticulocytes, white blood cell count, platelet count, plasma proteins, leukocyte differential count - neutrophils, band neutrophils, lymphocytes, monocytes, eosinophils, basophils, red blood cell and leukocyte abnormal morphology) were obtained in vacuum tubes (Vacutainer[®]) containing EDTA. Cells counts were carried out less than three hours after sampling, using automated equipment CellDyn Analyser 3500R, Abbott Laboratories. Differential leukocyte count was determined employing the manual method upon a Wright stained blood smear (Kno11 2000).

Urinalysis included physical exam (appearance, density), chemical exam (pH, protein, glucose, ketones, bilirubin, urobilinogen, blood) using urine strips, Multistix[®] Ames and microscopic examination of the sediment (leucocytes, erythrocytes, epithelial cells, casts, crystals, bacteria. Urine sediment examination was performed by standardized procedures according to Brobst (1989) and Meyer et al. (1992).

The statistical analysis determined the variation coefficient of K^+ in BC in order to calculate its repeatability. The difference between groups was carried out using Student's t -test. The relationship between the concentration of K^+ in serum and BC with that of other electrolytes and the values of acid-base status in blood, was determined using the Pearson correlation coefficient. Pearson's coefficient correlation was also calculated for SID, AG and Na^+/K^+ relationship with serum electrolytes and acid-base values. This was done in order to understand the relationship of the traditional and non-traditional methods with the electrolyte concentration. Statistical analyses were carried out using the SAS statistical package (1990).

Results

Means for serum Na^+ , K^+ and Cl^- , BC K^+ as well as values for acid-base status in blood (pH, HCO_3^- , BE and pCO_2) for healthy animals are shown in Table 1.

Buffy coat values of K^+ in the control group were 32.84 ± 2.34 mmol/L, while for K^+ in serum, they were 4.77 ± 0.59 . In Group 2, furosemide administration caused a decrease of K^+ in BC, as well as hypokalemia, hyponatremia, hypochloremia and an increase in pH, HCO_3^- and BE, due to metabolic alkalosis and an increase in pCO_2 (Table 1).

Differences for serum Na^+ , K^+ , and Cl^- , BC K^+ and for acid-base values ($p < 0.05$) were observed. The correlation coefficient between groups, for serum K^+ with BC K^+ , was non-significant ($r < 0.6$). There was a negative correlation greater than 0.6 with pH, HCO_3^- and BE; and a positive correlation greater than 0.7 with Na^+ and greater than 0.8 with Cl^- . The correlation coefficient between K^+ in BC and serum K^+ with the rest of the electrolytes and acid-base values was lower than 0.6, negative with pH, HCO_3^- and BE and positive with the rest of the electrolytes (Table 2).

Pearson's correlation coefficient for SID, AG and Na^+/K^+ with Na^+ and Cl^- in serum, as well as with pH, HCO_3^- , BE and pCO_2 produced the following results: SID has a positive and significant correlation with pH which is greater than 0.6; likewise for HCO_3^- and BE, though in this case greater than 0.7; SID correlations with serum K^+ and Cl^- are significant

Table 1. Means for serum Na⁺, K⁺ and Cl⁻, buffy coat K⁺ as well as values for acid-base status in blood (Group 1, control, n = 21) and in dogs with hypokalemia caused by furosemide administration (Group 2, experimental, n = 21)

Analyte	K ⁺	BC K ⁺	HCO ₃ ⁻	Cl ⁻	Na ⁺	BE	pH	pCO ₂	SID	AG	Na ⁺ :K ⁺
			mmol/L					mm/Hg		mmol/L	
GROUP 1											
Mean	4.73a	33.19a	18.0a	113.7a	147.5a	-7.04a	7.38a	30.7a	33.8a	20.4a	31.5a
SD	0.54	2.44	1.4	3.3	3.6	.72	0.031	3.6	2.8	3.0	3.3
Lower	3.84	30	16	108	140	-11.9	7.31	25	30	16.1	25.3
Upper	5.99	37.4	20.7	119	153	-4.0	7.44	39	40	26.1	37.6
GROUP 2											
Mean	3.45b	27.69b	24.7b	94.4b	137.6b	-1.68b	7.43b	34.4b	43.1b	21.9b	39.9b
SD	0.22	6.42	2.8	4.3	5.4	2.7	0.041	4.5	3.3	3.7	3.0
Lower	2.84	13.2	19.4	88.0	131.0	-6.8	7.34	29.0	39	14.5	36.1
Upper	3.77	37.0	29.5	106.0	156.0	4.6	7.5	44.0	50	27.7	47.1

K⁺; serum potassium, BC K⁺; buffy coat potassium, Na⁺; serum sodium, Cl⁻; serum chloride, HCO₃⁻; bicarbonate, BE; base excess, pCO₂; partial pressure of CO₂ in blood, SID; strong ion difference, AG; anion gap, Na⁺:K⁺; ratio sodium-potassium; a, b values (*p* < 0.5)

Table 2. Pearson's coefficient correlation of serum electrolytes, buffy coat potassium and blood acid-base values in healthy animals (Group 1, control, n = 21) and in dogs with hypokalemia caused by furosemide administration (Group 2, experimental, n = 21)

Analyte	K ⁺	BC K ⁺	pH	HCO ₃ ⁻	Cl ⁻	Na ⁺	BE	pCO ₂
K ⁺	1.00000	0.40569	-0.63820	-0.67244	0.84461	0.70701	-0.60396	-0.32784
	0	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.0340
BC K ⁺		1.00000	-0.32218	-0.47798	0.56655	0.58889	-0.40064	-0.22933
			0.0375	0.0014	<0.0001	<0.0001	0.0086	0.1440
pH			1.00000	0.65936	-0.61042	-0.44589	0.70806	-0.12729
				<0.0001	<0.0001	0.0031	<0.0001	0.4218
HCO ₃ ⁻				1.00000	-0.78388	-0.57529	0.93571	0.55814
					<0.0001	<0.0001	<0.0001	<0.0001
Cl ⁻					1.00000	0.87122	-0.71601	-0.34219
						<0.0001	<0.0001	0.0265
Na ⁺						1.00000	-0.43771	-0.15125
							0.0037	0.3390
BE							1.00000	0.58291
								<0.0001
pCO ₂								1.00000

K⁺; serum potassium, BC K⁺; buffy coat potassium, Na⁺; serum sodium, Cl⁻; serum chloride, HCO₃⁻; bicarbonate, BE; base excess, pCO₂; partial pressure of CO₂ in blood.

and negative, being -0.7 and -0.6, respectively; there were no significant correlations with BC K⁺, or with pCO₂, values being less than 0.5. The AG did not show a significant correlation with BC K⁺, serum Na⁺, K⁺ or Cl⁻, nor with pH, HCO₃⁻, BE or pCO₂ (Table 3).

Discussion

The values for acid-base status, Na⁺, K⁺, Cl⁻, haematology, biochemical profile and urinalysis in dogs from Group 1 were similar to reference values described by Willard et al. (1994) and Núñez (2001). In dogs of Group 2, with furosemide-induced hypokalemia, K⁺ losses were increased given that this diuretic acts upon the ascending portion of the loop

Table 3. Pearson's coefficient correlation of serum electrolytes, buffy coat potassium and blood acid-base values with strong ion difference, anion gap and ratio $\text{Na}^+:\text{K}^+$ in blood of healthy animals (Group 1, control, $n = 21$) and in dogs with hypokalemia caused by furosemide administration (Group 2, experimental, $n = 21$)

Analyte	K^+	BC K^+	pH	HCO_3^-	Cl^-	Na^+	BE	pCO_2
SID	-0.71470	-0.34084	0.63934	0.76108	-0.80493	-0.40999	0.80127	0.45298
	<0.0001	0.0272	<0.0001	<0.0001	<0.0001	0.0070	<0.0001	0.0026
AG	0.16181	0.08960	0.09695	-0.07037	-0.21369	0.15658	0.08476	0.01710
	0.3060	0.5725	0.5413	0.6579	0.1742	0.3221	0.5935	0.9144
$\text{Na}^+:\text{K}^+$	-0.95840	-0.27339	0.47038	0.63050	-0.74394	-0.52504	0.59853	0.32450
	<0.0001	0.0798	0.0017	<0.0001	<0.0001	0.0004	<0.0001	0.0360

K^+ ; serum potassium, BC K^+ ; buffy coat potassium, Na^+ ; serum sodium, Cl^- ; serum chloride, HCO_3^- ; bicarbonate, BE; base excess, pCO_2 ; partial pressure of CO_2 in blood, SID; strong ion difference, AG; anion gap, $\text{Na}^+:\text{K}^+$; ratio sodium-potassium

of Henle, thus blocking the $\text{Na}^+ - \text{K}^+ - 2\text{Cl}^-$ transporter (Gamba 1992; Bonagura et al. 2000). Previous experiments have described decreased serum concentrations of Na^+ and K^+ when administering 4 to 8 mg/kg of furosemide BID for 35 days (Pereira et al. 1998). Animals of Group 2 showed results similar to those already described, with a significant decrease of K^+ concentration from day 21 onwards, which can be verified by looking at the mean and standard deviation compared to those of Group 1. Furosemide administration caused metabolic alkalosis due to a loss of water, Na^+ , K^+ and Cl^- , as well as an increase in HCO_3^- , results which are similar to those found by other authors (Rose 1994; Senior 1995). The analysis of variance carried out between groups showed significant differences between pH, HCO_3^- and BE, due to metabolic alkalosis induced by furosemide.

In order to consider BC K^+ as an indicator of intracellular K^+ and the translocation process, the correlation coefficients of BC K^+ with serum K^+ , as well as with other analytes used for acid-base status, were calculated. Upon determining the correlations between groups, serum K^+ had a positive correlation with Cl^- and Na^+ , a negative correlation with pH, HCO_3^- and BE, while BC K^+ did not show high correlations with serum K^+ or with any other variables. The correlation found between serum K^+ and acid-base status could be explained by the changes in acid-base status that produce translocation of K^+ between ICF and ECF (Di Bartola and De Morais 1993, 2000; De Morais 1993). In general, metabolic acidosis is associated with the movement of K^+ from ICF to ECF, while alkalosis is associated with the movement of potassium ions from ECF to ICF (Ilkiw et al. 1989; Di Bartola and De Morais 1993). Experimental studies have found a decrease of serum K^+ during metabolic alkalosis, in dogs these were 0.18 mmol/L for each 0.1 increase in pH, and up to 0.4 mmol/L for each 0.1 of pH seen in respiratory alkalosis induced by hyperventilation (Muir et al. 1990).

The significant positive correlation of serum K^+ with Na^+ and Cl^- in Group 2 can be explained by the action of the diuretic, furosemide, which causes the loss of two Cl^- ions for each Na^+ and K^+ (Bonagura et al. 2000). The low correlation coefficient found between BC K^+ and serum K^+ , as well as with acid-base status, means that BC K^+ cannot be considered a good indicator of total intracellular potassium, despite having found a greater concentration of intracellular K^+ in BC when compared with the concentrations of K^+ in dog erythrocytes described in previous studies (Miseta et al. 1992; Catchpole and Engel 1996).

In both groups 1 and 2, BC K^+ was found in greater concentration than that found (Di Bartola and De Morais 2000). The lower concentration of BC K^+ does not reflect the translocation process from ECF to ICF during metabolic alkalosis, as is reflected by serum K^+ and acid-base values, when comparing both groups.

Acid-base status determines the translocation of K^+ . Evaluation of acid-base status with a blood gas analyzer allows one to know the origin of the problem, be it respiratory, metabolic or mixed; as well as the severity of these alterations and thus allows the proper diagnostic criteria for therapy and outcome (Di Bartola 2000). In clinical practice, blood gas analyzers are not always available, thus SID and HCO_3^- are so frequently used for diagnosing metabolic alkalosis and acidosis, when there is no gain of acids, and AG is used to diagnose metabolic acidosis with a gain in acids (Figge et al. 1998; Reilly and Anderson 1998)

Stewart (1983) presented a novel quantitative non-traditional method for the evaluation of acid-base disturbances of metabolic origin. This is based upon the existence of independent variables in body fluids, which include pCO_2 , strong ions (Na^+ and Cl^-) and weak acids (proteins). The dependent variables are HCO_3^- , OH^- and pH. According to this theory, the dependent variables are determined by the independent variables and can be calculated through specific equations (Russell et al. 1996). Only the independent variables can cause hydrogen ion movement in body fluids (Stewart 1983). Acid-base status can be understood in quantitative terms through the independent variables and their regulation by the lungs, kidneys, gastrointestinal tract and liver. In the last few years there has been a tendency to apply Stewart's concepts in evaluating acid-base status in human medicine (Fencil and Leith 1993).

It was deemed important to establish the relationship between SID, AG and $Na^+ : K^+$ with BC K^+ , and serum K^+ , Na^+ and Cl^- , as well as with the values used to determine acid-base status (pH, HCO_3^- , BE and pCO_2) in both the control and furosemide-induced hypokalemic group.

In the results of the statistical analysis for the groups, SID presented a significant positive correlation coefficient with HCO_3^- and BE, thus corroborating its utility in the diagnosis of metabolic alkalosis (Russell et al. 1996). A significant negative correlation coefficient with serum K^+ indicated that SID is a good parameter for evaluating the translocation that occurs due to metabolic translocation.

The AG in the experimental group did not show significant correlation with any of the electrolytes, or with the values of acid-base status. This can be explained due to the fact that AG is only useful for the diagnosis of metabolic acidosis when there is an overall gain in organic acids, sulphates and phosphates (Reilly and Anderson 1998).

In conclusion, under the conditions of the present study, BC K^+ did not turn out to be a better index than serum K^+ in diagnosing deficit or excess of total body K^+ . BC K^+ did not have a significant correlation with acid-base status, as was found to be the case for serum K^+ . Acid-base status determines the process of translocation, thus explaining the significant correlation coefficients found with serum K^+ . SID is a good indicator of the translocation process in metabolic alkalosis but not in metabolic acidosis concurrent with acid gain. AG is only a good indicator of the translocation process if metabolic acidosis involves a gain in acids.

Koncentrace draslíku v buffy coat, krevním séru a její vztah k acidobazickému stavu zdravých psů a psů s hypokalemií

Poruchy metabolismu elektrolytů a acidobazické rovnováhy jsou u psů časté. Cílem práce bylo stanovit koncentrace K^+ v krevním séru a koncentrace K^+ v buffy coat (BC K^+) a jejich vztah k elektrolytům v krevním séru a acidobazickým hodnotám zdravých a hypokalemických psů. Studie byla provedena na 42 psech obou pohlaví, stáří 3-8 roků. Psi byli

rozdělení do dvou skupin. Skupina 1 (kontrolní, n = 21) byla tvořena zdravými psy, skupina 2 (pokusná, n = 21) sestávala ze zdravých psů, u kterých byla vyvolána hypokalemie, a to perorální aplikací furosemidu v dávce 12 mg/kg ž. hm. po dobu 21 dnů. Klinické vyšetření, hematologický profil (16 analytů), biochemický profil (22 analytů) a analýzy moči (15 analytů) byly hodnoceny u všech psů. Nejdůležitější analyty v krevním séru byly Na⁺, K⁺ a Cl⁻; BC K⁺ a v krvi pH, HCO₃⁻, base excess (BE) a pCO₂. Byla vypracována metoda pro stanovení intracelulárního K⁺ v BC. Mezi skupinami 1 a 2 koncentrace K⁺ v krevním séru korelovala signifikantně s pH, HCO₃⁻ a BE (r > 0.6, p < 0.01); navíc ve skupině 2 byla zjištěna v důsledku účinku furosemidu signifikantní korelace s koncentracemi Na⁺ a Cl⁻ (r > 0.7, p < 0.01). Koncentrace K⁺ v BC vykazovala nízkou korelaci s K⁺ v krevním séru a ostatními sledovanými krevními analyty a tudíž nemůže být považována za vhodný ukazatel funkce intracelulárního K⁺. Korelační koeficienty ve skupinách 1 a 2 byly signifikantní mezi diferencí silných iontů (SID), K⁺, Cl⁻, HCO₃⁻ a BE (r > 0.7, p < 0.01), z čehož vyplývá, že SID je vhodným ukazatelem translokace K⁺ při metabolické alkalóze. Anion gap (AG) nekoreloval s BC K⁺, se Na⁺, K⁺ a Cl⁻ v krevním séru a rovněž s pH, HCO₃⁻, BE a pCO₂ v krvi. Pro vyhodnocení acidobazických poruch je nutné zařadit do laboratorních profilů stanovení K⁺, Na⁺, Cl⁻, HCO₃⁻ v krevním séru, stejně jako výpočet AG a SID a interpretovat je v návaznosti na klinické vyšetření zvířat.

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