Effect of inhaled albuterol on whole blood potassium concentrations in dogs

Andrzej Ogrodny | Jared A. Jaffey | Rachael Kreisler | Mark Acierno | Teela Jones | Renata S. Costa | Anderson da Cunha | Emily Westerback

1Department of Specialty Medicine, College of Veterinary Medicine, Midwestern University, Glendale, Arizona, USA
2Department of Primary Care, Shelter, and Community Medicine, College of Veterinary Medicine, Midwestern University, Glendale, Arizona, USA

Correspondence
Jared A. Jaffey, Department of Specialty Medicine, College of Veterinary Medicine, Midwestern University, Glendale, Arizona, USA.
Email: jjaffe@midwestern.edu

Abstract
Background: Albuterol by inhalation (IH) is a common treatment for hyperkalemia in humans but its effect on blood potassium concentrations in dogs is unknown.
Objective: Determine whether albuterol (IH) decreases blood potassium concentrations in healthy normokalemic dogs and if effects are dose-dependent.
Animals: Ten healthy dogs.
Methods: Prospective, crossover experimental study. Albuterol sulfate was administered at a low-dose (90 μg) in phase I and, 7 days later, high-dose (450 μg) in phase II. Blood potassium and glucose concentrations (measured via blood gas analyzer) and heart rates were obtained at baseline and then 3, 5, 10, 15, 30, 60, 90, 120, 180, and 360 minutes after inhaler actuation.
Results: Blood potassium concentrations decreased rapidly after albuterol delivery with a significant reduction compared to baseline within 30 minutes in both phases (P = .05). The potassium nadir concentration of phase I occurred at 60 minutes (mean, SD; 4.07 mmol/L, 0.4) and was significantly decreased from baseline, (4.30 mmol/L, 0.3; t(9) = 2.40, P = .04). The potassium nadir concentration of phase II occurred at 30 minutes (mean, SD; 3.96 mmol/L, 0.39) and was also significantly decreased from baseline, (4.33 mmol/L, 0.4; t(9) = 2.22, P = .05). The potassium nadir concentration decreased by 0.1 mmol/L for each 10 μg increase in dose of albuterol (P = .01). Five dogs had ≥1 hyperglycemic measurement (ie, >112 mg/dL). No median heart rate was tachycardic nor was any mean blood glucose concentration hyperglycemic at any time point.
Conclusion and Clinical Importance: Albuterol IH decreases blood potassium concentrations in a dose-dependent manner without clinically meaningful alterations to heart rate or blood glucose concentrations in healthy dogs. The mean decrease in potassium concentration at the high-dose of albuterol was modest (0.38 mmol/L).

Keywords
blood gas, electrolytes, hyperkalemia, β2-agonist

Abbreviations: ICC, intraclass correlation; IQR, interquartile range.

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.
© 2022 The Authors. Journal of Veterinary Internal Medicine published by Wiley Periodicals LLC on behalf of American College of Veterinary Internal Medicine.
1 | INTRODUCTION

Hyperkalemia is a common electrolyte disturbance in dogs evaluated on an emergency basis. Urinary diseases are the most common cause for hyperkalemia in dogs with other etiologies including hypoadreno-corticism, diabetes mellitus, intestinal parasites, acute tumor lysis syndrome, and secondary to several drugs. Hyperkalemia is a risk factor for death in dogs and humans in an emergency setting because of its role in causing potentially life-threatening arrhythmias.

Moderate-to-severe hyperkalemia requires urgent intervention. The goals of treatment are to mitigate cardiac conduction disturbances, eliminate excess potassium, shift potassium into cells, and resolve the underlying disease process. Therapies used to eliminate excess potassium include IV fluid therapy, loop or thiazide diuretics, hemodialysis, and continuous renal replacement therapy. Fluid diuresis is effective at rapidly lowering blood potassium concentrations but is contraindicated after initial hemodynamic stabilization in some of the most common conditions that cause hyperkalemia in dogs including oliguric or anuric kidney injury, ureabdomen, and complete urinary obstruction until after diversion or resolution. Loop and thiazide diuretics could have adjunctive value to promote renal excretion of potassium but should be avoided in the presence of dehydration or hypovolemia.

Hemodialysis and continuous renal replacement therapy can rapidly decrease blood potassium concentrations but are rarely used for the purpose of treating hyperkalemia alone in dogs. Therapeutic options in veterinary medicine to promote intracellular shifting of potassium are limited and include dextrose with or without insulin or sodium bicarbonate with the latter typically reserved for cases with severe acidosis. The β2-agonist terbutaline decreases serum potassium concentrations in dogs when administered as a continuous infusion; however, its safety as well as alternative dosing strategies have not been investigated.

Albuterol, like terbutaline, is a β2-agonist that promotes an intracellular shift of potassium via stimulation of endogenous insulin release and the induction of extracellular membrane-bound Na/K-ATPase pumps in an insulin-independent fashion. Albuterol is commonly used alone or with regular insulin and dextrose to treat moderate-to-severe hyperkalemia in humans and can be administered via inhalation or as an IV infusion. In humans, albuterol decreases serum potassium concentrations within 15 to 30 minutes of administration, with duration of effect lasting up to 180 minutes, making it an ideal intervention. Tachycardia and hyperglycemia are reported potential adverse effects to albuterol administration in humans. There have been no published studies that have investigated the effect of inhaled albuterol on blood potassium concentrations in dogs.

Our study had 3 objectives: (i) to determine whether inhaled albuterol decreases blood potassium concentrations in dogs; (ii) to determine if nadir blood potassium concentrations are associated with delivered albuterol dose; and (iii) to assess if inhaled albuterol affects heart rate or blood glucose concentrations. We hypothesized that inhaled albuterol would decrease blood potassium concentrations and the nadir potassium concentration would be associated with the delivered dose. Furthermore, we hypothesized that inhaled albuterol would not affect heart rate or blood glucose concentrations.

2 | MATERIALS AND METHODS

2.1 | Animals

Dogs of any age, breed, or sex owned by faculty, staff, and students at the Midwestern University College of Veterinary Medicine that weighed 5 to 20 kg with whole blood potassium concentrations of 3.5 to 5.5 mmol/L (ie, normokalemic) were eligible for inclusion. Dogs were excluded if there was previously documented history of cardiac disease or if either a heart murmur or an arrhythmia (brady- or tachyarrhythmia) were identified on physical examination performed by a board-certified small animal internist (JAJ). In addition, dogs were excluded if 1 or more of the following medications with potential blood potassium altering effects were administered within 60 days of enrollment, enalapril, benazepril, telmisartan, losartan, diuretics, trilostane, mitotane, potassium gluconate/citrate, fludrocortisone, desoxy-corticosterone pivalate, or insulin. Client consent was obtained. This study was approved by the Midwestern University Animal Care and Use Committee (protocol #3010).

2.2 | Study design

The study was performed with a prospective, open-label, 2-way crossover design.

Pharmaceutical grade albuterol sulfate (metered-dose inhaler, 90 μg/actuation; Teva Pharmaceuticals Ireland, Waterford, Ireland) was delivered via an AeroDawg spacer (Trudell Animal Health, London, Ontario, Canada) attached to a tightly sealed silicone facemask (Figure 1) at a dose of 90 μg (1 metered actuation) in phase I (ie, low-dose) and 450 μg (5 metered actuations) in phase II (ie, high-dose), executed 7 days later.

FIGURE 1 Illustration of albuterol sulfate delivery via “AeroDawg” AeroDawg spacer attached to a tightly sealed silicone facemask
In both phases, heart rates were recorded followed by blood sample acquisition at baseline (time \( t = 0 \); before delivery of inhaled albuterol) and at 3, 5, 10, 15, 30, 60, 90, 120, 180, and 360 minutes after the tenth breath following inhaler actuation. Blood was collected by venipuncture and immediately transferred to lithium heparin-containing tubes. Whole blood was analyzed by a veterinary benchtop blood gas analyzer (Stat Profile Prime Plus Vet, Nova Biomedical, Waltham, Massachusetts) within 5 minutes of collection. Whole blood potassium and glucose concentrations were recorded for each time-point. Throughout the study period, the blood gas analyzer underwent daily quality control and routine maintenance as instructed by the manufacturer. Dogs were housed in a quiet room under continuous observation by investigators, water was available ad libitum, and food was withheld through the duration of each phase. Hyperglycemia and hypoglycemia were defined as blood glucose concentrations of >112 and <65 mg/dL, respectively. Hypokalemia was defined as blood potassium concentrations of <3.5 mmol/L. Tachycardia was defined as a heart rate of >180 beats/min (bpm).

### 2.3 Statistical analysis

Statistical analyses were performed using proprietary software (Stata Statistical Software version 17, StatCorp LLC, College Station, Texas). Normality was assessed using tests of skewness and kurtosis. Normally distributed values were reported as mean, SD, and range and values at each time point compared to baseline values using T-tests, with the \( t \) statistic (\( t \)-value), an indication of the difference between 2 samples, and degrees of freedom (df) reported as \( t \). Non-normally distributed values were reported as median and range and compared using Wilcoxon rank-sum tests. In each phase, the nadir value for variables was defined as the time point with the lowest mean or median while similarly the apex value for variables was defined as the time point with the highest mean or median. The effect of inhaled albuterol dose (ie, \( \mu \)g/kg) on nadir potassium concentration was assessed using multilevel mixed-effects linear regression with patient as a random effect. The model was validated through visual inspection of the residuals. The contribution of interpatient variability to the dose-effect was assessed using the intraclass correlation (ICC), with values <0.5 considered poor and values \( \geq 0.5 \) considered moderate or better. A \( P \)-value of \( \leq 0.05 \) was considered significant.

### 3 RESULTS

#### 3.1 Dogs

Ten dogs fulfilled the inclusion criteria and were enrolled. No dogs were excluded. There were 6 mixed breed dogs. Purebred dogs included were Miniature schnauzer (n = 2), and 1 each of Cattle dog and Australian shepherd. The median age and weight were 5 years (range, 2.5-9.8) and 15.9 kg (range, 5.6-19.6). There were 5 castrated males, 4 spayed females, and 1 intact female.

#### 3.2 Effect on potassium concentration

Potassium concentrations decreased rapidly after administration of inhaled albuterol in both phases, before reaching a nadir and slowly increasing toward baseline (Figure 2). There was no difference in baseline potassium concentration (\( t(18) = 0.22, P = .83 \)) between phases, with mean baseline potassium concentrations of 4.30 mmol/L (SD, range; 0.3, 3.99-4.87) and 4.33 (SD, range; 0.4, 3.90-5.14) for

![Figure 2](image1.png) Scatterplot and LOWESS curves of the potassium concentration at baseline (ie, before administration of inhaled albuterol) and at 3, 5, 10, 15, 30, 60, 90, 120, 180, and 360 minutes after the tenth breath following albuterol inhaler actuation for phase I (1 inhaler actuation, 90 \( \mu \)g) and phase II (5 inhaler actuations, 450 \( \mu \)g). Solid blue vertical line at the potassium nadir concentration of phase II (30-minutes time point) and dashed vertical red line at nadir concentration of phase I (60-minutes time point)

![Figure 3](image2.png) Relationship between nadir potassium concentration and albuterol IH dose (\( \mu \)g/kg) overlaid by linear best fit line and 95% CI. Phase I values as red squares and phase II values as blue diamonds. Nadir potassium concentration was inversely associated with inhaled albuterol dose (\( P = .01 \))

Nadir potassium concentration was inversely associated with inhaled albuterol dose (\( P = .01 \)) for
phase I and II, respectively. The potassium nadir concentration of phase I occurred at $t = 60$ minutes (mean, SD, range; 4.07 mmol/L, 0.4, 3.54-4.53) and was significantly decreased from baseline, $t(9) = 2.40, P = .04$. The potassium nadir concentration of phase II occurred at $t = 30$ minutes (mean, SD, range; 3.96 mmol/L, 0.39, 3.36-4.53) and was also significantly decreased from baseline, $t(9) = 2.22, P = .05$. Mean potassium concentrations remained decreased at $t = 360$ minutes as compared to baseline, although this difference was not statistically significant for either phase I ($t(18) = 0.86, P = .4$) or phase II ($t(18) = 0.93, P = .36$).

A multivariable mixed-effects linear regression model with potassium nadir as the dependent variable, dose and baseline potassium as independent variables and patient as the random effect found a significant inverse association between the inhaled albuterol dose and potassium nadir concentration ($P = .01, Figure 3$). The potassium nadir concentration decreased by 0.1 mmol/L (95% CI: -0.12, -0.02) for each 10 $\mu$g/kg increase in inhaled albuterol dose, while each 0.1 mmol/L increase in baseline potassium concentration resulted in a 0.04 mmol/L (95% CI: 0.01-0.08) increase at nadir ($P = .03$). The ICC for the random effect of patient was 0.5 (95% CI: 0.1-0.9; Figure 4).

Hypokalemia occurred in 3% (6/200) of time points (phase I, $n = 1$; phase II, $n = 5$). The potassium concentration reflective of hypokalemia in phase I was 3.42 mmol/L. The median potassium concentration of the 5 time points associated with hypokalemia in phase II was 3.41 mmol/L (range, 3.36-3.44). Hypokalemia resolved without intervention in all cases.

### 3.3 | Effect on glucose concentration

Glucose concentration decreased briefly in both phases, with the nadir (mean, SD, range; 91.7 mg/dL, 16.5, 66-116) for phase I occurring at $t = 10$ minutes before rebounding to an apex at $t = 180$ minutes (109.8 mg/dL, 15.8, 92-134). Phase II reached the nadir (mean, SD, range; 87.2 mg/dL, 20.2, 65-122) at $t = 5$ minutes before subsequently increasing to the apex (106.9 mg/dL, 9.0, 94-119) at $t = 360$ minutes (Figure 5). The mean glucose concentration at baseline was 95.8 mg/dL (SD, range; 134, 67-111) for phase I and 100.1 mg/dL (143, 77-127) for phase II, and these were not different ($t(18) = 0.69, P = .5$). In phase I, the glucose nadir was not different from baseline ($t(18) = 0.61, P = .55$). The apex mean concentration achieved statistical significance ($t(18) = -2.14, P = .05$). There was no difference in the glucose nadir ($t(18) = 1.65, P = .12$) or apex ($t(18) = -1.27, P = .22$) concentration as compared to baseline in phase II.

Hyperglycemia occurred in 21% (42/200) of time points (phase I, $n = 22$; phase II, $n = 20$). In both phases, there were 5 dogs that had 1 or more hyperglycemic measurements. Three of these dogs had hyperglycemic measurements in both phases, while the remaining 2 in each phase were unique to their phase and did not experience hyperglycemia in the alternate phase. The median glucose concentration for the hyperglycemic values were 117.5 mg/dL (range, 114-141, $n = 22$) and 122 mg/dL (114-134, $n = 20$) for phase I and phase II, respectively. Hypoglycemia occurred in 0.5% (1/200) of time points (phase II, $n = 1$). The blood glucose concentration associated with hypoglycemia was 64 mg/dL.
In addition, similar to results from our study, a tachycardia can occur with high doses of albuterol. One potential explanation for the variability in the potassium lowering effect of inhaled albuterol in humans is that the potassium lowering effect of albuterol in normokalemic dogs reported in the current study might underestimate the expected magnitude of reduction in dogs with hyperkalemia. There have been no studies comparing the potassium lowering effects of albuterol in normokalemic and hyperkalemic human patients. However, 2 studies have shown a more modest decrease in serum potassium concentrations in normokalemic humans after nebulized albuterol administration (0.5 mmol/L) compared to what is generally reported in hyperkalemic patients (0.61-0.9 mmol/L).

The potassium lowering effects of inhaled albuterol in normokalemic dogs reported in the current study might underestimate the expected magnitude of reduction in dogs with hyperkalemia. There have been no studies comparing the potassium lowering effects of albuterol in normokalemic and hyperkalemic human patients. However, 2 studies have shown a more modest decrease in serum potassium concentrations in normokalemic humans after nebulized albuterol administration (0.5 mmol/L) compared to what is generally reported in hyperkalemic patients (0.61-0.9 mmol/L). The cause for this lack of expected potassium lowering effect of albuterol in normokalemic dogs reported in the current study might be related to elevated circulating concentrations of endogenous catecholamines. One potential explanation for the variability in the potassium lowering effect of albuterol in a subset of patients is unknown although 1 theory suggests it could be related to elevated circulating concentrations of endogenous catecholamines. Therefore, shallow breaths might have affected the amount of delivered albuterol, even after 10 breaths.

There were no clinically relevant changes to mean blood glucose concentration, with the single statistically significant mean apex reading found in phase I below the pre-determined cutoff for hyperglycemia and the highest blood glucose value recorded for any dog at any time point was 141 mg/dL. There were no statistically significant changes to median heart rate after delivery of inhaled albuterol in either phase of this study. Mild transient increases in heart rate and blood glucose concentration can occur after inhaled/albuterol administration in normokalemic dogs. Tachycardia can occur with high doses of albuterol because of direct stimulation of atrial β1-receptors, myocardial β1-receptors, as well as from reflex cardiac stimulation from peripheral vasodilation. The reason relevant changes in heart rate and blood glucose were not identified in our study could be related to the dose of delivered albuterol. The majority of studies in humans that have investigated the hypokalemic effects of inhaled/nebulized albuterol have used high doses ranging from 10 to 20 mg.
It is possible the incidence of adverse effects could increase with higher doses of albuterol IH in dogs. This theory is supported by the results from 2 recent retrospective studies that revealed tachycardia (81%-94%), hypokalemia (21%-69%), and hyperglycemia (67%) were common sequela to intoxication by high-dose salbutamol (albuterol) exposure in dogs.40,41

This study had several limitations that require further elucidation. The dogs in this study were healthy and normokalemic. The safety and potassium lowering effect of inhaled albuterol could vary in critically ill dogs and in those that are stable with various comorbid disorders. Therefore, our results should not be extrapolated for use in clinical situations until future studies investigating the safety and effectiveness in unhealthy hyperkalemic dogs are performed. Ours, was an exploratory study aimed at determining whether inhaled albuterol lowered blood potassium concentrations in dogs; therefore, optimal dose and frequency of administration were not determined. In addition, we did not pre-specify whether our cutoff for significance would be P < or ≤ .05. Lastly, randomized order of treatment protocols was not utilized in the design of this study. The lack of randomization could have had unknown effects on results. However, the wash out period was sufficient to minimize carry over biologic effects between phases and all dogs readily accepted the AeroDawg chamber and facemask in both phases.

5 | CONCLUSION

Our findings revealed that inhaled albuterol caused a rapid decrease in blood potassium concentrations in healthy normokalemic dogs. The potassium lowering effect of inhaled albuterol was dose-dependent, and caused no adverse effects. Additional studies with larger sample populations are needed to better understand the safety and efficacy of inhaled albuterol in unhealthy hyperkalemic dogs.

ACKNOWLEDGMENT

No funding was received for this study. The authors thank Paige Hunsinger for her technical assistance.

CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

This study was conducted in accordance with guidelines for clinical studies and approved by the Midwestern University Animal Care and Use Committee (protocol# 3010).

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

REFERENCES

1. Hoehne SN, Hopper K, Epstein SE. Retrospective evaluation of the severity of and prognosis associated with potassium abnormalities in dogs and cats presenting to an emergency room (January 2014-August 2015): 2441 cases. J Vet Emerg Crit Care (San Antonio). 2019;29:653-661.
2. Khanagavi J, Gupta T, Aronow WS, et al. Hyperkalemia among hospitalized patients and association between duration of hyperkalemia and outcomes. Arch Med Sci. 2014;10:251-257.
3. An JN, Lee JP, Jeon HJ, et al. Severe hyperkalemia requiring hospitalization: predictors of mortality. Crit Care. 2012;16:R225.
4. Riordan LL, Schaer M. Potassium disorders. In: Silverstein DC, Hopper K, eds. Small Animal Critical Care Medicine. 2nd: Elsevier; 2015;269-273.
5. Liu M, Rafique Z. Acute management of hyperkalemia. Curr Heart Fail Rep. 2019;16:67-74.
6. O’Malley CM, Frumento RJ, Hardy MA, et al. A randomized, double-blind comparison of lactated Ringer’s solution and 0.9% NaCl during renal transplantation. Anesth Analg. 2005;100:1518-1524.
7. Cole LP, Jepson R, Dawson C, Humm K. Hypertension, retinopathy, and acute kidney injury in dogs: a prospective study. J Vet Intern Med. 2020;34:1940-1947.
8. Bouchard J, Soroko SB, Chertow GM, et al. Fluid accumulation, survival and recovery of kidney function in critically ill patients with acute kidney injury. Kidney Int. 2009;76:422-427.
9. Joseph WB, Delmar RF, David JP, Cari AO, Jeanne AB, Scott AB. Pathophysiology of urethral obstruction. Vet Clin North Am Small Anim Pract. 1996;26:255-264.
10. Balakrishnan A, Drobatz KJ. Management of urinary tract emergencies in small animals. Vet Clin North Am Small Anim Pract. 2013;43:843-867.
11. Gordon RD. The syndrome of hypertension and hyperkalemia with normal GFR. A unique pathophysiological mechanism for hypertension? Clin Exp Pharmacol Physiol. 1986;13:329-333.
12. Bragg-Gresham JL, Fissell RB, Mason NA, et al. Diuretic use, residual renal function, and mortality among hemodialysis patients in the dialysis outcomes and practice pattern study (DOPPS). Am J Kidney Dis. 2007;49:426-431.
13. Langstone C. Hemodialysis in dogs and cats. Compendium. 2002;24:540-548.
14. Clausen T, Kohn P. The effect of insulin on the transport of sodium and potassium in rat soleus muscle. J Physiol. 1977;265:19-42.
15. adrogué HJ, Madias NE. Changes in plasma potassium concentration during acute acid-base disturbances. Am J Med. 1981;71:456-467.
16. Hiatt N, Morgenstern L, Davidson M, Bonorris G, Miller A. Role of insulin in the transfer of infused potassium to tissue. Horm Metab Res. 1973;5:84-88.
17. Hurlbert BJ, Edelman JD, David K. Serum potassium levels during and after betabutaline. Anesth Analg. 1981;60:723-725.
18. Mandelberg A, Krumnik Z, Houri S, et al. Salbutamol metered-dose inhaler with spacer for hyperkalemia: how fast? How safe? Chest. 1999;115:617-622.
19. Pierre SV, Xie Z. The Na,K-ATPase receptor complex: its organization and membership. Cell Biochem Biophys. 2006;46:303-316.
20. Allon M, Dunlay R, Copkney C. Nebulized albuterol for acute hyperkalemia in patients on hemodialysis. Ann Intern Med. 1989;110:426-429.
21. Wong SL, Maltz HC. Albuterol for the treatment of hyperkalemia. Ann Pharmacother. 1999;33:103-106.
22. Singh BS, Sadiq HF, Naguchi A, Keenan WJ. Efficacy of albuterol inhalation in treatment of hyperkalemia in premature neonates. J Pediatr. 2002;141:16-20.
23. Orgel HA, Kemp JP, Welch MJ, et al. 230 Single-dose comparison of subcutaneous (SC), intramuscular (IM) and intravenous (IV) injectable albuterol in acute asthma. J Allergy Clin Immunol. 1985;75:162.
24. Montoliu J, Almirall J, Ponz E, et al. Treatment of hyperkalaemia in renal failure with salbutamol inhalation. *J Intern Med*. 1990;228:35-37.
25. King WD, Holloway M, Palmisano PA. Albuterol overdose: a case report and differential diagnosis. *Pediatr Emerg Care*. 1992;8:268-271.
26. Duane M, Chandran L, Morelli PJ. Recurrent supraventricular tachycardia as a complication of nebulized albuterol treatment. *Clin Pediatr*. 2000;39:673-677.
27. Lam S, Chen J. Changes in heart rate associated with nebulized racemic albuterol and levalbuterol in intensive care patients. *Am J Health Syst Pharm*. 2003;60:1971-1975.
28. Schmitz KL, Jeffery U, Heinz JA, Rutter CR. Evaluation of two benchtop blood gas analyzers for measurement of electrolyte concentrations in venous blood samples from dogs. *Am J Vet Res*. 2021;82:105-109.
29. Shea EK, Hess RS. Assessment of postprandial hyperglycemia and circadian fluctuation of glucose concentrations in diabetic dogs using a flash glucose monitoring system. *J Vet Intern Med*. 2021;35:843-852.
30. Hackett TB. Physical examination and daily assessment of the critically ill patient. In: Silverstein DC, Hopper K, eds. *Small Animal Critical Care Medicine*. St. Louis, Missouri: Elsevier; 2015:6-10.
31. Portney LG, Watkins MP. *Foundations of Clinical Research: Applications to Practice*. 3rd ed. Upper Saddle River, NJ: Pearson/Prentice Hall; 2009.
32. Allon M, Copkney C. Albuterol and insulin for treatment of hyperkalemia in hemodialysis patients. *Kidney Int*. 1990;38:869-872.
33. Allon M, Shanklin N. Effect of albuterol treatment on subsequent diuretic potassium removal. *Am J Kidney Dis*. 1995;26:607-613.
34. McClure R, Prasad V, Brocklebank J. Treatment of hyperkalaemia using intravenous and nebulised salbutamol. *Arch Dis Child*. 1994;70:126-128.
35. Pau C, LaFlamme M, Evans E, Reed J. Levalbuterol is as effective as racemic albuterol in lowering serum potassium. *J Emerg Med*. 2003;25:13-16.
36. Zitek T, Cleveland N, Rahbar A, et al. Effect of nebulized albuterol on serum lactate and potassium in healthy subjects. *Acad Emerg Med*. 2016;23:718-721.
37. Liou HH, Chiang SS, Wu SC, et al. Hypokalemic effects of intravenous infusion or nebulization of salbutamol in patients with chronic renal failure: comparative study. *Am J Kidney Dis*. 1994;23:266-271.
38. Woodward S, Mundorff M, Weng C, Gamboa DG, Johnson MD. Incidence of supraventricular tachycardia after inhaled short-acting beta agonist treatment in children. *J Asthma*. 2021;58:471-480.
39. Du Plooy W, Hay L, Kahler C, et al. The dose-related hyper- and hypokalaemic effects of salbutamol and its arrhythmogenic potential. *Br J Pharmacol*. 1994;111:73-76.
40. Meroni ER, Khorzad R, Bracker K, Sinnott-Stutzman V. Retrospective evaluation of albuterol inhalant exposure in 36 dogs: 36 cases (2007-2017). *J Vet Emerg Crit Care (San Antonio)*. 2021;31:86-93.
41. Crouchley J, Bates N. Retrospective evaluation of acute salbutamol (albuterol) exposure in dogs: 501 cases. *J Vet Emerg Crit Care (San Antonio)*. 2022;32:500-506.

How to cite this article: Ogrodny A, Jaffey JA, Kreisler R, et al. Effect of inhaled albuterol on whole blood potassium concentrations in dogs. *J Vet Intern Med*. 2022;36(6):2002-2008. doi:10.1111/jvim.16552