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Prediction of arterial blood gas values from venous blood gas values in Asiatic black bears (*Ursus thibetanus***) anesthetized with intramuscular medetomidine and zolazepam-tiletamine**

Dong-Hyuk JEONG1), Jeong-Jin YANG1), Lyon LEE2) and Seong-Chan YEON3)*

1)Species Restoration Technology Institute of Korea National Park Service, Gurye 542-853, Republic of Korea ²⁾Laboratory of Veterinary Anesthesia and Pain Management, College of Veterinary Medicine, Western University, Pormona, CA 91766, U.S.A.

3)Laboratory of Veterinary Surgery and Behavior, College of Veterinary Medicine, Gyeongsang National University, Jinju 660-701, Republic of Korea

ABSTRACT. The objective of this study was to measure differences between arterial and venous blood gas parameters and to evaluate whether arterial blood gas values can be estimated from venous blood in Asiatic black bears (ABBs). Twelve healthy captive ABBs (8 males and 4 females; 8–16 years; 76.8–220 kg) were included in this study. The bears were immobilized with medetomidine and zolazepam-tiletamine using a dart gun. Arterial and venous samples were collected simultaneously at 5 and 35 min after recumbency (5- and 35-min points). Partial pressure of oxygen (PO₂), partial pressure of carbon dioxide (PCO₂), pH, bicarbonate (HCO₃⁻), total carbon dioxide (TCO₂), oxygen saturation of hemoglobin (SO₂) and base excess (BEecf) were analyzed using a portable blood gas analyzer. There was no marked difference in measured and calculated variables over time in both venous and arterial blood except for PO₂. However, arterial PO₂, SO₂ and pH were significantly higher and arterial PCO₂, TCO₂ and HCO₃⁻ were lower than those of venous samples at both 5- and 35-min points. In the regression analysis to estimate arterial values from venous values, PCO₂, TCO₂, HCO₃⁻, BEecf and pH significantly showed over 0.45 in coefficient of determination value (*R*2), and there were little differences between actual and predicted arterial values. Although there were limits in venous gas values replaced those of arterial blood, if we could not get the arterial samples, the regression formulas for arterial values from venous blood in this study would be useful clinically, except for $PO₂$ and $SO₂$.

KEY WORDS: anesthesia, arterial, Asiatic black bear, blood gas, venous

Chemical immobilization of zoo and wild animals is a form of veterinary anesthesia conducted under difficult circumstances. The risk of adverse side effects can never be completely eliminated. In addition, all immobilizing drugs are toxic, and some are potentially lethal to humans [[16](#page-5-0)]. Many drug combinations have been used for chemical immobilization of bears. Among them, the combinations of α_2 -adrenoceptor agonist (α_2 -agonist; xylazine, detomidine, medetomidine and dexmedetomidine) plus zolazepam-tiletamine have been used widely [[10, 17, 24, 26\]](#page-5-1). However, there were several reports that α_2 -agonists induced hypoxemia and respiratory problems in bear species [[6, 10, 11\]](#page-5-2). Thus, monitoring of immobilized animal is important, and a pulse oximeter has been simply used in the fields. The pulse oximeter is a noninvasive monitor that provides real-time values of hemoglobin saturation and pulse rate; therefore, it enables to detect hypoxemia before other clinical signs develop [\[8, 18\]](#page-5-3). However, the accuracy and reliability of the pulse oximeter can be affected by hemoglobin concentration, placement of probe, motion artifacts, ambient light, vasoconstriction and vasodilation [[2, 22\]](#page-5-4). And, vasoconstriction is commonly associated with use of α_2 -agonist in immobilization protocols for either captive or free-ranging animals [[18](#page-5-5)]. Therefore, blood gas analyzing is more accurate than the pulse oximeter for obtaining information about oxygenation, ventilation and the acid-base status of the body. In general, arterial blood is used for blood gas analysis, but some studies have demonstrated a good correlation of indicators for pulmonary function and acid base balance among arterial, capillary and venous samples in humans and animals [\[1, 15, 19](#page-5-6)]. However, the use of venous blood for blood gas analysis for clinical application still remains a controversial topic. Studies on blood gas values in polar bear [[5\]](#page-5-7), brown

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^{*}Correspondence to: Yeon, S.-C.: scyeon@gnu.ac.kr

bear [[10, 12, 26\]](#page-5-1), American black bear [[6](#page-5-2)] and sun bear [\[3](#page-5-8)], were carried out using arterial blood samples. However, little is known about blood gas values in Asiatic black bears; moreover, differences in blood gas values between arterial and venous blood have not been investigated in this or any other bear species. The objective of this study was to measure the differences between arterial and venous blood gas parameters and to find out whether arterial blood gas values can be predicted accurately from venous blood gas values in Asiatic black bears immobilized by medetomidine-zolazepam-tiletamine (MZT) administration.

MATERIALS AND METHODS

Animals

The study protocol was approved by the Animal Care and Research Committee of the Species Restoration Technology Institute, Korea National Park Service (SRTI number 16-021). Twelve clinically healthy adult Asiatic black bears (8 male and 4 female bears; age range, 8–16 years; weight range, 76.8–220 kg) housed at the Species Restoration Technology Institute were included in this study. The bears had been released into the wild for restoration of the bear population, but they were subsequently relocated into a captive facility because of conflicts with humans and potential harm to each other.

Each bear was housed individually in a small pen (approximately $3 \times 4 \times 3$ m) with a cement floor. The bears spent 4–6 hr per day in a semi-natural enclosed field (2,880 m²) that resembled the wild bear habitat. They were fed acorns, chestnuts, fruits, vegetables, sweet potatoes and commercial feed (Omnivore Diet Dry®, ZuPreem, Mission, TX, U.S.A.) twice a day based on seasonal calorie requirements. Feeding was discontinued during the hibernation period (mid-December to early March) in accordance with the unique ecology and physiology of bears.

Immobilization and physiologic monitoring

The bears were fasted overnight before anesthesia. They received an intramuscular (IM) injection of 2.0 ± 0.3 (range: 1.7–2.7) mg/kg zolazepam/tiletamine (Zoletil 50[®], Virbac Co., Ltd., Fort Worth, TX, U.S.A.) and 0.04 ± 0.01 (0.03–0.06) mg/ kg medetomidine (Domitor®, Pfizer Inc., New York, NY, U.S.A.) using a dart gun (CO₂ PI, Dan-Inject, Børkop, Denmark) in their sleeping or resting pens. During the anesthesia the bears were on the floor in the pens, and room temperature was between $12-17$ °C. All animals were weighed during anesthesia, and the actual drug doses were retrospectively calculated. At the end of the procedure, 0.20 ± 0.03 (0.2–0.3) mg/kg atipamezole (Antisedan®, Pfizer Inc.) was injected IM to reverse medetomidine and expedite the recovery from anaesthesia. During the procedures, 0.1 mg/kg of meloxicam (Metacam®, Boehringer Ingelheim Co., Ltd., Ingelheim am Rhein, German) and 5 mg/kg cefazolin (Cefazolin®, Yuhan Co., Ltd., Seoul, Korea) were administered intravenously to reduce pain and to minimize the risk of wound infection after the experiment. Rectal temperature was monitored with a digital thermometer that provided continuous reading across a measurement range of 32.0–42.9°C (MT200, Microlife AG Co., Ltd., Widnau, Switzerland). The respiratory rate was monitored by observing thoracic movements, and heart rate was checked by auscultation with a stethoscope (Master Cardiology 2160, 3MTM Littmann®, Syracuse, NY, U.S.A.). Rectal temperature, respiratory rate and heart rate were monitored throughout anesthesia and recorded at the time of blood sampling.

Blood sampling and analysis

Arterial blood samples were collected from the femoral artery, which was accessed by palpation, and confirmed by pulsating blood into syringe. Firm pressure was applied to the sample site for 3 min after sampling to avoid bleeding. Venous blood samples were collected from the jugular vein. Arterial and venous samplings were simultaneously conducted by 2 veterinarians at 5 and 35 min, respectively, after recumbency with loss of pedal and sluggish palpebral reflex. The blood samples of 10 m*l* were collected anaerobically in pre-heparinized 10 m*l* plastic syringes with 18-gauge × 38 mm needle (Nayeon Medical, Seoul, Korea) and then immediately analyzed for pH, partial pressure of carbon dioxide (PCO₂), partial pressure of oxygen (PO₂), bicarbonate (HCO₃⁻), total carbon dioxide (TCO₂), hemoglobin saturated with oxygen $(SO₂)$ and base excess (BEecf), by using a portable blood gas analyzer and cartridges (i-STAT Portable Clinical Analyzer and i-STAT cartridges EC8+ and CG4+, Abaxis Inc., Union City, CA, U.S.A.). Blood gas values and pH were corrected to the rectal temperature.

Statistical analysis

All statistical tests were performed using a statistical analysis program (IBM SPSS Statistics 18 software®, Foster City, CA, U.S.A.). A normal distribution for all analytes was confirmed using a Shapiro-Wilk analysis. The independent *t*-test was used to confirm the differences between venous and arterial variables at each 5- and 35-min point, and the changes over time in venous and arterial blood samples. Correlation between venous and arterial variables regardless of sampling timing was evaluated by Pearson's correlation test, and the parameters with correlation coefficients (*r*) exceeding 0.6 were entered into simple linear regression analysis to predict arterial values from venous values. The predicted arterial values were calculated by the regression formulas which were obtained using the simple linear regression analysis, and those calculated arterial values were compared to actual arterial values. To estimate accuracy of the regression formulas, the coefficient of determination values $(R²)$ were confirmed. The Bland-Altman plots was analyzed to determine the agreement between venous and arterial samples, and the bias and the 95% limit of agreement (LOA) were calculated using the whole data from the 5- and 35-min points. Differences were considered significant for *P*<0.05.

a) *P*<0.05, significant difference between 5- and 35- min points in arterial blood; b) *P*<0.05, significant difference between 5- and 35- min points in venous blood; c) *P*<0.05, significant difference between arterial and venous blood at the 5 min point; d) *P*<0.05, significant difference between arterial and venous blood at the 35 min point; SD, standard deviation; RR, respiratory rate; HR, heart rate; RT, rectal temperature; PCO₂, partial pressure of carbon dioxide; PO₂, partial pressure of oxygen; TCO₂, total carbon dioxide; SO₂, hemoglobin saturated with oxygen; BEecf, base excess; HCO₃⁻, bicarbonate.

Table 2. Actual values in this study and predicted values by regression formulas for base excess, bicarbonate, pH, PCO₂ and TCO₂ in Asiatic black bears (*Ursus thibetanus*) immobilized with medetomidine-zolazepam-tiletamin

	Actual values (Mean \pm SD (Median))		Predicted values	Formula	R^2	P -value
	Venous blood $(n=24)$	Arterial blood $(n=24)$				
BEecf (mmol/l)	-4.9 ± 2.1 (-5)	-5.63 ± 1.9 (-6)	-5.7	$AB=0.708 \times VB-2.145$	0.586	≤ 0.001
$HCO3- (mmol/l)$	$22.3 \pm 1.7(22.2)$	20.2 ± 1.6 (20.3)	20.1	$A HCO3=0.758 \times V HCO3 + 3.299$	0.644	≤ 0.001
pH	7.25 ± 0.05 (7.25)	$7.33 \pm 0.07(7.3)$	7.3	ApH= $1.005 \times VpH + 0.036$	0.463	≤ 0.001
$PCO2$ (mmHg)	$50.64 \pm 6.7(50.9)$	39.6 ± 7.4 (39.8)	39.7	$APCO_2=0.743 \times VPCO_2 + 1.909$	0.453	≤ 0.001
$TCO2$ (mmol/ <i>l</i>)	23.7 ± 1.9 (22.2)	21.4 ± 1.8 (21)	20.3	$ATCO_2=0.692 \times VTCO_2 + 4.973$	0.528	< 0.001

BEecf, base excess; HCO₃⁻, bicarbonate; PCO₂, partial pressure of carbon dioxide; TCO₂, total carbon dioxide; AB, arterial base excess; VB, venous base excess; AHCO₃, arterial bicarbonate; VHCO₃, venous bicarbonate; ApH, arterial pH; VpH, venous pH; APCO₂, arterial partial pressure of carbon dioxide; VPCO₂, venous partial pressure of carbon dioxide; ATCO₂, arterial total carbon dioxide; VTCO₂, venous total carbon dioxide; R^2 , coefficient of determination.

RESULTS

The bears were successfully immobilized with a single dart, and the time from darting to recumbency ranged from 1 to 15 min. During the anesthesia, the bears were maintained in dorsal recumbent position, and immobilized body conditions of 5-min point did not significantly differ from those of 35-min point. There were no side effects, such as vomit, excessive salivation, hyperthermia and conversion throughout the anesthesia, and no re-sedation after recovery. The lowest rectal temperature recorded was 36.1°C, and the mean temperature at each 5- and 35-min point was 36.6 ± 1.2 and 36.6 ± 0.9 °C, respectively.

There was no significant change in respiratory rate of the bears $(P=0.058)$, and their heart rates decreased significantly $(P=0.042)$ over time. Arterial PO₂ at 5-min point was significantly lower than that at 35 min point ($P=0.002$). However, there were no significant differences in the other parameters between 5-min and 35-min points in both venous and arterial samples. Arterial PO₂ SO2 and pH values were higher (at 5-min point: *P*<0.001, *P*<0.001, and *P*=0.002; at 35-min point, *P*<0.001, *P*<0.001 and *P*=0.014, respectively), and arterial PCO_2 , TCO_2 and HCO_3^- values were lower than those of venous samples (5-min point, $P<0.001$, *P*=0.004, and *P*=0.005; at 35-min point, *P*=0.002, *P*=0.004 and *P*=0.005, respectively) (Table 1).

There were significant positive correlations between venous and arterial samples ($r=0.67-0.80$, $P<0.05$) except for PO₂ ($r=0.26$, $P=0.204$) and SO₂ ($r=0.10$, $P=0.634$), and the estimated regression formulas of arterial parameters by venous samples are shown in Table 2. There were little differences between actual and predicted arterial values of BEecf, HCO_3^- , pH, PCO_2 and TCO_2 by regression formulas (R^2 =0.45−0.64, P <0.001) in this study (Table 2).

The Bland-Altman plots indicated that venous HCO_3^- (bias, 2.09 ± 1.06 ; 95% LOA, 0.01 to 4.16) and BEecf (bias, 0.71 \pm 1.37; 95% LOA, −1.97 to 3.38) were higher than the corresponding arterial parameters (Fig. 1). However, the bias and the 95% LOA for HCO₃⁻ and BEecf between arterial and venous blood samples did not seem to be relevant from a clinical perspective, because of marginal difference between the samples. The venous PO₂ (bias, -42.92 ± 18.83 ; 95% LOA, -79.82 to -6.01), SO₂ (bias, -27.04) \pm 14.44; 95% LOA, −55.35 to 1.27) and pH (bias, −0.06 \pm 0.03; 95% LOA, −0.11 to −0.006) were lower, and venous PCO₂ (bias, 10.46 ± 4.26 ; 95% LOA, 2.11 to 18.81) and TCO₂ (bias, 2.33 \pm 1.34; 95% LOA, -0.29 to 4.96) were higher than these values in

arterial blood based on the Bland-Altman plots (Fig. 1), but in this case, the bias and LOA seemed to be important from a clinical perspective because of substantial magnitude of differences between arterial and venous samples.

DISCUSSION

Several studies have demonstrated the reliability and accuracy of venous blood gas in acid-base monitoring as an alternative to arterial blood gas analysis including in human and canine intensive care [[1, 19, 20, 23, 25](#page-5-6)]. Some of these studies simply used correlation analysis to compare the differences between venous and arterial blood. However, Giavarina [[14](#page-5-9)] pointed out that correlation between the 2 methods was always misleading and should not be used for assessing the accuracy of different methods or obtained data in comparability. In the present study, we used both correlation analysis and the Bland-Altman method to compare differences between venous and arterial blood more objectively with less bias.

The PO₂ values are influenced by the inspired oxygen tension, adequacy of ventilation, cardiac output and blood pressure. Fahlman [[9\]](#page-5-10) defined 60–80 mmHg of arterial PO_2 was mild hypoxemia, while <60 mmHg PO_2 was considered as marked hypoxemia in brown bears. Furthermore, the arterial PO₂ of American black bears immobilized with MZT increased over time [[6](#page-5-2)], but decreased in brown bears [\[10](#page-5-1)], grizzly bears [[26](#page-6-0)] and polar bears [[5\]](#page-5-7) that had been immobilized with MZT, dexmedetomidine-ZT (dexMZT), and Xylazine-ZT (XZT) or zolazepam-tiletamine (ZT), respectively. The decreases in PO_2 values in brown bears immobilized with MZT was probably associated with impaired pulmonary gas exchange, because α_2 -agonists can increase the pulmonary vascular pressure and disturb the matching of pulmonary perfusion in relation to ventilation. However, in this study, median arterial PO₂ increased over time from 76.5 mmHg (mild hypoxemia) to 91.5 mmHg (within normal range). This is maybe related to different drug doses, because lower drug doses were used in this study compared to those in brown bears (medetomidine, 0.07−0.12 mg/kg; ZT, 3.2−6.0 mg/kg), and our doses were similar to those in American black bears (medetomidine, 0.05 mg/kg; ZT , 1.92 mg/kg), which increased PO₂ over time. Another possibility is probably due to an effect of time dependent reduction of the immobilizing drug effects through metabolism and redistribution and resultant improvement in cardiopulmonary function.

In addition, the mean values of arterial SO₂ were $88.5 \pm 2.5\%$ and $89.3 \pm 1.1\%$ at 5- and 35-min points, respectively, in our study. In free-ranging brown bears immobilized with MZT [[10](#page-5-1)], the value of arterial SO_2 was 88–89%, and there was no significant difference between 2 samples which had time difference of 30 min. Thus, the arterial SO_2 values in our Asiatic black bears immobilized with MZT seem to be similar to those in brown bears [[10](#page-5-1)]. Although further investigation is necessary to confirm the recommended target values of arterial SO₂ for hemodynamic optimization, the IM administration of MZT may provide a reliable immobilization with a transient mild hypoxemia in the Asiatic black bears.

Acidemia is defined as a blood pH below 7.35 and alkalemia is done as a pH above 7.45 in brown bears [\[9](#page-5-10)]. And, Bush *et al.* [\[3](#page-5-8)], Caulkett and Cattet [[6](#page-5-2)] and Fahlman *et al.* [\[10](#page-5-1)] discussed a pH 7.26–7.35 of arterial blood was mild acidemia in brown bears and American black bears immobilized with phencyclidine or zolazepam-tiletamine or MZT [\[3, 6, 10\]](#page-5-8). In this study, mild acidemia was presented in arterial blood (5-min point, 7.33; 35-min point, 7.30) from the Asiatic black bears immobilized with MZT.

The $PCO₂$ represents the respiratory component of the acid-base balance. Hypocapnia was defined as an arterial $PCO₂$ below 35 mmHg, and hypercapnia was defined as mild (arterial PCO₂ 45–60 mmHg) or marked (arterial PCO₂ above 60 mmHg) in brown bears [[9\]](#page-5-10). Mild hypercapnia was reported in captive brown bears immobilized with MZT [\[9, 10](#page-5-10)] or dexMZT [[26](#page-6-0)].These increased PCO₂ values may be due to hypoventilation arising from the respiratory effects of α_2 -agonists (medetomidine and dexmedetomidine). However, in this study, none of the bears were hypercapnic during the anesthesia, considering arterial PCO₂ (5-min point, 36.6 mmHg; 35-min points, 42 mmHg). This is probably because pulmonary depression was less than that in those bears in previous studies from anesthetic effects, or no observed hyperthermia, or absence of excessive exercise or exhaustion in animals before the anesthesia or combination of these factors.

The HCO_3^- automatically calculated from pH and PCO_2 by blood gas analyzer based on Henderson Hesselbach equation, is representative of both respiratory and metabolic status [[7\]](#page-5-11). The mean values of arterial HCO₃⁻ were 14–21 mmol/*l* in free ranging brown bears immobilized with MZT [[10](#page-5-1)], 17.9−23.5 mmol/*l* in free-ranging and captive grizzly bears immobilized with dexMZT [[26](#page-6-0)] and 21.3–25.1 mmol/*l* in dogs immobilized with sevoflurane [\[25](#page-6-1)]. In our study, the median values of arterial HCO₃⁻ were 19.9 and 20.3 mmol/*l* (at 5 and 35 min, respectively) which seemed to be acceptable as normal range. Finally, the IM administration of MZT provided a reliable immobilization with mild hypoxemia and academia, while respiratory and metabolic status changed minimally in the Asiatic black bear.

The arterial PO₂ values obtained at 5- and 35-min points in our study differed significantly from the venous values. The Bland-Altman plots revealed that venous PO_2 was greatly lower than arterial PO_2 , and the bias as well as the 95% LOA seemed to be important from a clinical perspective, indicating that the 2 values could not be used interchangeably. The mean difference value between venous and arterial SO2 in this study (−27.04 ± 14.44%) was large, and 95% LOA in the Bland-Altman plots (Lower, −55.35; Upper, 1.27) was too wide to allow substitution. There was no significant difference of pH between 5- and 35-min points, and the Bland-Altman plots showed that venous pH was slightly lower than arterial pH. Although the values of bias and the 95% LOA seem to be small (-0.06 ± 0.03 and -0.006 to -0.114, respectively), substituting venous pH for arterial pH may have limitations.

In the present study, venous $PCO₂$ at both the 5- and 35-min points was significantly higher than at the corresponding times of arterial blood, and the Bland-Altman plots showed that venous $PCO₂$ was higher than arterial $PCO₂$. Moreover, the bias and 95% LOA (10.46 ± 4.26 mmHg and 2.11 to 18.81, respectively) seem to be clinically relevant, suggesting that these values cannot be used interchangeably. Carter and Auton [[4\]](#page-5-12), Geers and Gros [[13](#page-5-13)], and Maren and Sanyal [[21\]](#page-6-2) reported the difference of carbonic

anhydrase activity between animal species, and Tamura *et al.* [[25\]](#page-6-1) discussed the difference might affect the buffering capacity of carbon dioxide in blood which results in different gradients between venous and arterial PCO₂ in different animal species. Thus, venous PCO₂ cannot be readily substituted for arterial PCO₂ in some species including cats and Asiatic black bears.

Although there were statistically significant differences between venous and arterial HCO_3^- , the mean differences and 95% LOA were small and comparable from a clinical perspective. $TCO₂$ is also representative of both respiratory and metabolic status as it includes HCO_3^- and dissolved CO_2 , although the contribution by dissolved CO_2 is small [[7](#page-5-11)]. In our study, the mean difference between venous and arterial blood of TCO₂ was 2.33 ± 1.34 mmol/*l*, and 95% LOA in the Bland-Altman plots was 4.96 (Upper) and −0.29 (Lower). These values in our study seem to indicate some differences between venous and arterial TCO₂, although they are interchangeably used in human medicine. However, further studies are warranted, because there are very few references for TCO₂ in bear species and small sample size used in this study.

The BEecf value describes the buffering capacity of the blood and provides a calculated assessment of the metabolic status of a patient that is almost independent of respiratory changes [\[7](#page-5-11)]. There was no significant change in BEecf over time in either venous or arterial blood, and there was also no difference between venous and arterial BEecf in the present study. Although the Bland-Altman plots revealed that venous BEecf was slightly higher than the arterial BEecf, venous and arterial BEecf can be used interchangeably from a clinical perspective.

Interestingly, although the Bland-Altman method indicated that venous pH, $PCO₂$ and $TCO₂$ could not be substituted for the corresponding arterial parameters, those parameters were highly correlated between venous and arterial blood. The $PO₂$ and $SO₂$ were the only 2 parameters that were not significantly correlated. It means that we could estimate arterial pH, $PCO₂$ and $TCO₂$ values from those of venous sample, even though we could not use interchangeably. Thus, we compared actual and predicted arterial values by regression formulas and could confirm those values had no big differences.

In conclusion, this study reports the basic venous and arterial blood gas parameters in healthy Asiatic black bears immobilized with MZT. We show that arterial gas analysis values cannot be replaced by venous blood gas analysis values, except for BEecf. However, if we could not get the arterial blood, we suggest the use of estimated regression formulas for arterial blood values based on venous blood in this study, except for $PO₂$ and $SO₂$. Although the use of regression formulas would result in differences, it would also be helpful for use as a clinical reference.

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