Blood Gas Analysis in Dogs with Heartworm Caval Syndrome

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(Received 24 September 1993/Accepted 17 May 1994)

ABSTRACT. Blood gases and cardiopulmonary function were analyzed in 67 dogs with heartworm (HW) caval syndrome (CS) and 19 HW-free dogs. Arterial oxygen tension (PaO_2) was 91.5±7.3 mmHg in the HW-free dogs, 74.9±14.3 mmHg in 46 dogs that subsequently survived surgical HW removal (surviving dogs) and 64.6±14.7 mmHg in 21 dogs which later died or were euthanatized following surgical treatment (nonsurviving dogs). PaO_2 levels less than 60 mmHg were detected in 30.4% and 38.1% dogs in the surviving and nonsurviving groups, respectively. Arterial carbon dioxide tension ($PaCO_2$) was 35.8±4.9 mmHg in the HW-free dogs, 30.7±5.6 mmHg in the surviving dogs, and 28.8±6.2 mmHg in nonsurviving dogs. PaO_2 ($p<0.01$) and $PaCO_2$ were lower ($p<0.01$) and the alveolar-arterial oxygen difference ($AaDO_2$) was higher ($p<0.01$) in CS dogs than in HW-free dogs. PaO_2 was lower ($p<0.01$) and $AaDO_2$ was larger ($p<0.01$) in the nonsurviving dogs than in the surviving dogs. Arterial blood pH and bicarbonate concentration were lower ($p<0.01$) and the anion gap was larger ($p<0.01$) in CS dogs than in the HW-free dogs. Serum lactic acid level in nonsurviving dogs (13.2±3.9 mmol/*l*) was higher ($p<0.01$) than in the HW-free (1.7±0.8 mmol/*l*) and surviving dogs (2.7±1.8 mmol/*l*). The PaO_2 correlated significantly with mean pulmonary arterial pressure ($r=-0.65$, $p<0.01$), cardiac index ($r=0.44$, $p<0.05$) and total pulmonary resistance ($r=-0.70$, $p<0.01$). Blood gas analysis was repeated 1 week after HW removal in 21 dogs that survived and 10 dogs that did not survive. PaO_2 ($p<0.05$) and $PaCO_2$ ($p<0.01$) increased, and $AaDO_2$ ($p<0.01$) decreased in the surviving dogs, but not in the nonsurviving dogs. Blood gas values reflecting circulatory disturbance and ventilation insufficiency are predictors of survival following surgical HW removal in dogs with CS.—KEY WORDS: blood gas, canine, caval syndrome, heartworm disease.

J. Vet. Med. Sci. 56(5): 861–867, 1994

Caval syndrome (CS) is a severe type of heartworm (HW) disease in dogs. Affected dogs show severe circulatory and respiratory disorders such as prostration, cardiac murmur, jugular pulsation, venous congestion, tachycardia, labored respiration, tachypnea, rough breath sounds and cyanosis. Severe anemia, liver and renal failure, and hemoglobinuria (intravascular hemolysis) are also major features of the syndrome [1, 6, 8]. By echocardiography, heartworms (HWs) can be seen moving back and forth with each beat between the right atrium and right ventricle through the tricuspid valve orifice in all dogs, and also in the pulmonary arteries of many cases [9, 10]. Relatively fresh pulmonary thromboemboli containing dead HWs and proliferative lesions of pulmonary arterial walls have been observed at necropsy [10]. The tricuspid valve dysfunction and pulmonary arterial lesions induce circulatory disturbances and ventilatory insufficiency. Therefore, it was anticipated that the determination of blood gases might contribute to a better understanding of pathophysiology of the disease. Nevertheless, there has been a lack of data on blood gases in dogs with CS.

In a previous paper [11], the authors reported blood gas tensions and acid base balance in dogs with pulmonary HW disease, in which adult HWs were residing only in the pulmonary arteries. The present study describes results of arterial and mixed venous blood gas analyses in dogs with naturally acquired CS. Relationships between blood gas tensions and laboratory or cardiopulmonary function values, and changes in blood gas tensions after HW removal are also reported.

MATERIALS AND METHODS

Dogs: Nineteen HW-free dogs and 67 dogs with naturally acquired CS were studied. The diagnosis of the disease was made on the basis of clinical signs such as prostration, cardiac murmur, jugular pulsation, anemia, hemoglobinuria, laboratory test results and echocardiographic documentation of HWs at the tricuspid valve orifice. All HW-free dogs and 19 affected dogs were obtained from the regional dog pound, and the other 48 CS dogs were patients of the Veterinary Hospital of Gifu University. During an observation period (10 to 60 days) after surgical HW removal, 46 dogs recovered (surviving dogs). Twenty-one dogs showed the terminal signs of severe prostration, anorexia, hypothermia, azotemia, oliguria, and increase in ascitic and subcutaneous fluid, and died or were euthanatized because of their poor prognosis (nonsurviving dogs).

Surgical HW removal: General anesthesia was induced with diazepam (0.1 to 0.5 mg/kg, IV, Cercine, Takeda Chemical Industries Ltd., Osaka) and ketamine hydrochloride (2 to 5 mg/kg, IM, Ketalar, Sankyo Co., Ltd., Tokyo). Heartworms were removed from the right atrium, tricuspid valve orifice and pulmonary arteries via a jugular venotomy using flexible alligator forceps (FK-380S and FK-480L, Fuji Photo-Optical Co., Ltd., Omiya, Japan) [7].

Blood gas and electrolyte analysis: Dogs were placed in right lateral recumbency under general anesthesia using diazepam and ketamine hydrochloride, and breathed room air spontaneously. Arterial blood samples were

collected by direct puncture of a femoral artery. Mixed venous blood was drawn from the pulmonary arteries through a catheter. Blood sampling was carried out immediately before and 1 week after heartworm removal. Arterial oxygen tension (PaO_2), arterial carbon dioxide tension ($PaCO_2$) and pH were determined with an automated pH/blood gas analyzer (168 pH/Blood Gas Analyzer, Corning Ltd., Halstead, England). The values for pH, PO_2, and PCO_2 were corrected for body temperature and blood hemoglobin concentration. The oxygen saturation, plasma bicarbonate concentration (HCO_3^-) and base excess were calculated from blood gas values and/or pH with the same pH/gas analyzer. Serum sodium (Na) and potassium (K) concentrations were determined by the ion-electrode method (Na/K ISE Analyzer Model 902, Corning Ltd., Halstead, England) and serum chloride concentration by Volhard chloride estimation (925 Chloride analyzer, Corning Ltd.). Serum lactic acid concentration was determined with a commercial test kit (F-kit L-lactic acid, Boehringer Mannheim GMBH, Mannheim, Germany). Alveolar-arterial oxygen difference ($AaDO_2$) was calculated from the equation; $149.73–1.25 \times PaCO_2 - PaO_2$ [19].

Other measurement procedures: Blood and serum biochemical examinations were performed by standard procedures. Cardiopulmonary function measurements were carried out by procedures described in our previous papers [10, 11].

Statistical analysis: Data are expressed as mean± standard deviation. The Duncan's new multiple range test was used for statistical comparison of the data among HW-free, surviving and nonsurviving dogs, and the Student *t*-test or Cochran-Cox test was used for comparison of the data between surviving and nonsurviving dogs. A paired *t*-test was used for comparison of the values before and after HW removal.

RESULTS

Figure 1 shows the PaO_2, $PaCO_2$ and $AaDO_2$ values. The PaO_2 was within the normal range in 24 of 46 (52.2%) of the surviving dogs, and in 4 of 21 (19.0%) of the nonsurviving dogs. A PaO_2 less than 60 mmHg was detected in 14 (30.4%) and 8 (38.1%) of the surviving and nonsurviving dogs, respectively. The PaO_2 level was significantly ($p<0.01$) lower in the surviving (74.9±14.3 mmHg) and nonsurviving (64.6±14.7 mmHg) dogs than in the HW-free dogs (91.5±7.3 mmHg), and significantly ($p<0.01$) lower in nonsurviving dogs than in surviving dogs. The $PaCO_2$ was within the normal range in many dogs of both CS groups. Nine (19.6%) surviving dogs and 7 (33.3%) nonsurviving dogs had a $PaCO_2$ less than 25 mmHg. The $PaCO_2$ level in surviving (30.7±5.6 mmHg) and nonsurviving (28.8±6.2 mmHg) dogs were lower ($p<0.01$) than in HW-free dogs (35.8±4.9 mmHg), however, there was no significant difference in $PaCO_2$ level between surviving and nonsurviving dogs. The $AaDO_2$ ranged from 6.1 mmHg to 65.5 mmHg with a mean of 36.5±17.4 mmHg in surviving dogs. In nonsurviving dogs, the $AaDO_2$ was distributed in a wider range than in surviving dogs. Twenty (43.5%) of the surviving dogs and 15 (71.4%) of the nonsurviving dogs had a $AaDO_2$

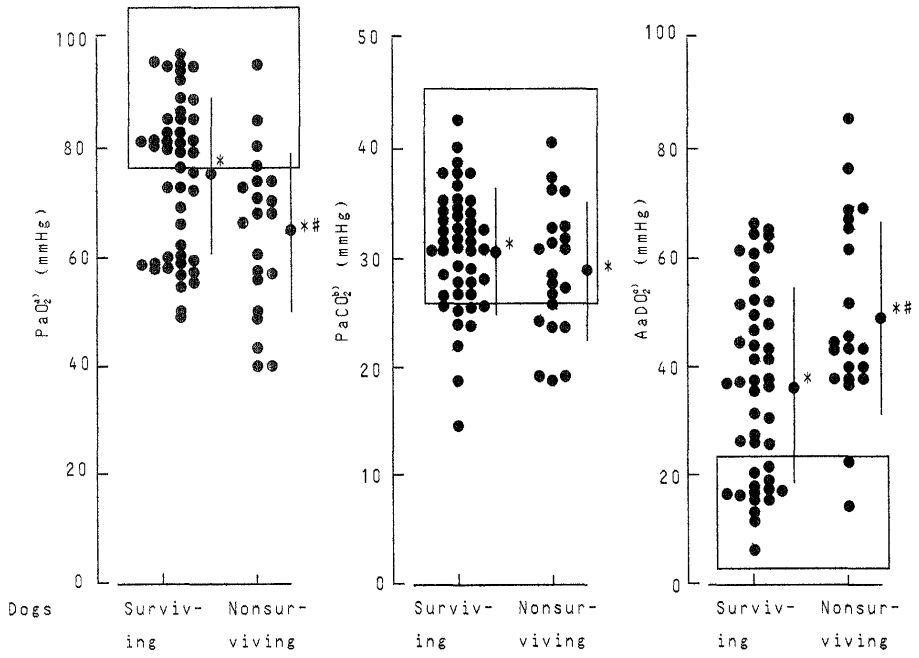

Fig. 1. Arterial blood gas values in dogs with caval syndrome. Vartical bar: mean ± standard deviation; rectangle: normal range (mean ± 2 standard deviations in HW-free dogs); *: significantly ($p<0.01$) different from the value of HW-free dogs; #: significantly ($p<0.01$) different from the value of surviving dogs. a: Arterial oxygen tension, b: arterial carbon dioxide tension, c: alveolar arterial oxygen difference.

exceeding 35 mmHg.

As shown in Table 1, the oxygen tension in mixed venous blood ($P\bar{v}O_2$) was lower in surviving and nonsurviving dogs than in HW-free dogs. In the nonsurviving group, many dogs (11/18 dogs, 61.1%) had a low $P\bar{v}O_2$ (less than 30 mmHg). The mixed venous carbon dioxide tension ($P\bar{v}CO_2$) was also lower in both surviving and nonsurviving CS dogs than in HW-free dogs, but there was no significant difference between surviving and nonsurviving dogs. The arterial oxygen saturation (SaO_2) was lower only in nonsurviving dogs. The mixed venous oxygen saturation ($S\bar{v}O_2$) was lower in surviving dogs than in HW-free dogs, and was lower in nonsurviving dogs than in surviving dogs.

Arterial blood pH, HCO_3^- concentration and base excess (Table 2) were lower ($p<0.01$), and the anion gap was higher in surviving and nonsurviving dogs than in HW-free dogs. Serum Na concentration was lower ($p<0.01$) in nonsurviving dogs than in surviving dogs. Serum K concentration was higher ($p<0.01$) in nonsurviving dogs than in HW-free dogs. Serum chloride concentration was significantly ($p<0.01$) lower in nonsurviving dogs. Serum lactic acid concentration was higher ($p<0.01$) in nonsurviving dogs.

Table 3 summarizes hematologic and cardiopulmonary function values. Statistically significant differences ($p<0.01$) between HW-free and surviving dogs were found for hematocrit (Ht), hemoglobin (Hb) concentration, white blood cell (WBC) count, plasma alanine transaminase (ALT) activity, mean pulmonary arterial

Table 1. Venous blood gas tensions and arterial and venous oxygen saturations

Variable	HW[a]-free dogs		Caval syndrome			
			Surviving dogs		Nonsurviving dogs	
Mixed venous oxygen tension (mmHg)	13	55.3±5.9 (46.0 to 63.9)	40	33.8±9.0* (10.9 to 56.8)	18	29.3±10.7* (5.7 to 48.9)
Mixed venous carbon dioxide tension (mmHg)	13	42.6±5.8 (36.1 to 57.3)	40	38.7±7.0* (19.7 to 51.9)	18	38.8±7.4* (27.1 to 57.0)
Arterial oxygen saturation (%)	11	95.7±2.2 (90.1 to 97.6)	30	88.9±7.1 (71.6 to 96.7)	13	84.8±10.4*# (63.6 to 95.7)
Mixed venous oxygen saturation (%)	11	83.0±3.8 (74.7 to 87.2)	30	47.9±17.1* (8.1 to 83.1)	13	34.7±20.1*# (7.1 to 72.7)

Data are expressed as number of dogs and mean±standard deviation (range).*: Significantly ($p<0.01$) different from the value in HW-free dogs; #: Significantly ($p<0.01$) different from the value in surviving dogs; a) Heartworm.

Table 2. Acid-base and electrolyte values

Variable	HW[a]-free dogs		Caval syndrome			
			Surviving dogs		Nonsurviving dogs	
pH	19	7.354±0.051 (7.254 to 7.439)	46	7.292±0.066* (7.136 to 7.447)	21	7.269±0.078* (7.124 to 7.3999)
Bicarbonate concentration (mmol/*l*)	19	19.5±2.0 (16.0 to 23.7)	46	14.7±3.3* (6.5 to 20.8)	21	13.2±3.9* (6.4 to 22.0)
Base excess	19	−4.2±2.2 (−7.1 to 1.4)	46	−9.8±3.9* (−3.1 to −18.5)	21	−11.7±4.6* (−1.0 to −19.5)
Sodium concentration (mmol/*l*)	10	141±12 (137 to 149)	29	144±7 (126 to 155)	11	135±11# (120 to 149)
Potassium concentration (mmol/*l*)	10	3.32±0.39 (2.54 to 3.88)	29	3.75±0.61 (2.72 to 4.88)	11	3.87±0.82* (2.82 to 5.39)
Chloride concentration (mmol/*l*)	10	114±5 (107 to 121)	29	116±11 (76 to 142)	11	107±7*# (95 to 115)
Anion gap (mmol/*l*)	10	7.5±12.4 (−7.9 to 24.2)	29	16.2±11.3* (−7.2 to 54.7)	11	17.5±10.3* (1.8 to 31.9)
Lactic acid concentration (mmol/*l*)	10	1.7±0.8 (0.7 to 3.0)	26	2.7±1.8 (0.4 to 8.6)	10	13.2±3.9*# (6.4 to 22.0)

Data are expressed as number of dogs and mean±standard deviation (range). *: Significantly ($p<0.01$) different from the value in HW-free dogs; #: Significantly ($p<0.01$) different from the value in surviving dogs; a) Heartworm.

Table 3. Hematologic and cardiopulmonary function values

Variable	HW[a]-free dogs		Caval syndrome			
			Surviving dogs		Nonsurviving dogs	
Hematocrit value (%)	19	39.4±5.6 (24.0 to 52.5)	46	28.9±8.3* (11.0 to 44.0)	21	32.6±7.1* (10.0 to 43.0)
Hemoglobin concentration (g/dl)	19	13.12±1.95 (7.96 to 17.93)	46	9.31±2.77* (3.57 to 14.96)	21	10.21±2.49* (5.05 to 13.52)
White blood cell count ($\times 10^2/\mu l$)	19	130±49 (70 to 227)	46	206±90* (86 to 513)	21	240±109* (105 to 571)
Alanine transaminase activity (IU/L)	19	37±15 (21 to 67)	44	110±145* (14 to 764)	19	70±65 (6 to 253)
Creatine kinase activity (IU/L)	10	96±29 (63 to 148)	26	255±182 (109 to 891)	13	371±401* (80 to 1591)
Urea nitrogen concentration (mg/dl)	19	16.5±7.1 (9.0 to 39.0)	44	29.7±31.8 (6.8 to 153)	18	47.7±33.1*# (14.6 to 149)
Heart rate (beat/min)	13	173±36 (103 to 221)	38	163±31 (100 to 235)	18	151±28*# (95 to 212)
Mean pulmonary arterial pressure (mmHg)	13	12.5±2.6 (8.0 to 16.8)	38	44.6±19.4* (11.6 to 85.0)	18	50.7±16.6* (19.6 to 77.5)
Cardiac index (ml/min/kg)	13	336±67 (236 to 472)	16	283±86 (181 to 514)	8	215±57* (117 to 286)
Total pulmonary resistance ($dyne \cdot sec \cdot cm^{-5} \cdot kg$)	13	3069±832 (1993 to 5222)	16	8927±4249* (3341 to 17689)	8	17716±11405*# (7974 to 38389)
Right ventricular stroke work index (g/beat/kg)	13	0.339±0.095 (0.198 to 0.481)	16	0.699±0.318* (0.282 to 1.322)	8	0.901±0.529* (0.529 to 2.135)

Data are expressed as number of dogs and mean±standard deviation (range). *: Significantly ($p<0.01$) different from the value in HW-free dogs; #: Significantly ($p<0.01$) different from the value in surviving dogs; a) Heartworm.

pressure (MPAP), total pulmonary resistance (TPUR), and right ventricular stroke work index (RVSWI). In nonsurviving dogs, Ht value, Hb concentration, WBC count, plasma creatinine kinase (CK) activity, plasma urea nitrogen (UN) concentration, heart rate (HR), MPAP, cardiac index (CI), TPUR, and RVSWI were statistically different ($p<0.01$) from those in HW-free dogs. Significant differences ($p<0.01$) between surviving and nonsurviving dogs were found in plasma UN concentration, CI, and TPUR.

Table 4 shows the number of live adult HWs removed. Four to 104 worms (23.4±19.8 worms) were removed from the right atrium and tricuspid valve orifice area in surviving dogs, and 1 to 64 worms (15.1±17.7 worms) in nonsurviving dogs. From the pulmonary arteries, 0 to 38 worms and 0 to 23 worms were removed in surviving and nonsurviving dogs, respectively. The number of worms/kg body weight in surviving dogs (4.00±2.11 worms/kg) was greater ($p<0.05$) than in nonsurviving dogs (2.07±2.53 worms/kg).

Table 5 shows correlation coefficients between arterial blood gas tensions and hematologic, blood chemical and cardiopulmonary values. The PaO_2 correlated significantly with MPAP ($r=-0.65$, $p<0.01$), CI ($r=0.44$, $p<0.05$), and TPUR ($r=-0.70$, $p<0.01$). Correlation coefficients between the $PaCO_2$ and plasma UN concentration ($r=-0.27$, $p<0.05$) or MPAP ($r=-0.35$, $p<0.01$) were significant. The $AaDO_2$ correlated significantly with plasma UN concentration ($r=0.30$, $p<0.05$), MPAP ($r=0.67$,

Table 4. Number of heartworms removed in dogs with caval syndrome

No. of heartworms	Surviving dogs (n=38)	Nonsurviving dogs (n=21)	p[a]<
Removed from right atrium and tricuspid valve orifice	23.4±19.8 (4 to 104)	15.1±17.7 (1 to 64)	NS[b]
Removed from pulmonary arteris	8.3±9.8 (0 to 38)	4.6±6.8 (0 to 23)	NS
Sum total	32.1±20.9 (4 to 104)	20.9±22.9 (2 to 74)	NS
Sum total/kg body weight of dog	4.00±2.11 (0.20 to 12.73)	2.07±2.53 (0.25 to 11.75)	0.05
Remaining[c]	3.6±2.8 (0 to 9)	6.6±2.8 (0 to 22)	NS

Data are expressed as mean±standard deviation (range). a) Probability of significant difference between surviving and nonsurviving dogs; b) Not significant; c) Number of worms remaining was examined in 10 surviving dogs and 9 nonsurviving dogs.

Table 5. Correlations between arterial blood gas values and other clinical data in dogs with caval syndrome

Variable	No. of dogs	Arterial oxygen tension		Arterial carbon dioxide tension		Alveolar arterial oxygen difference	
Hematocrit value	67	−0.002	NS[a)]	0.09	NS	−0.03	NS
Alanine transaminase activity	63	0.03	NS	−0.14	NS	0.03	NS
Creatine kinase activity	39	−0.16	NS	−0.05	NS	0.16	NS
Serum urea nitrogen concentration	63	−0.23	NS	−0.27	0.05	0.30	0.05
Mean pulmonary arterial pressure	56	−0.65	0.01	−0.35	0.01	0.67	0.01
Cardiac index	24	0.44	0.05	−0.01	NS	−0.34	NS
Total pulmonary resistance	24	−0.70	0.01	−0.02	NS	0.56	0.01
No. of heartworms	59	−0.09	NS	−0.03	NS	0.09	NS
No. of worms/kg body weight	59	0.16	NS	0.06	NS	−0.16	NS

Data are expressed as correlation coefficient and probability of significant coefficient (p value). a) Not significant.

Table 6. Changes in blood gas values after heartworm removal in dogs with caval syndrome

Group	Variable	No. of dogs	Before removal	1 week after removal	p[a)]<
Surviving	PaO_2[c)] (mmHg)	21	77.1±12.9	81.5±8.3	0.05
	$PaCO_2$[d)] (mmHg)	21	29.4±5.7	33.3±5.3	0.01
	$AaDO_2$[e)] (mmHg)	21	35.9±16.2	26.6±13.0	0.01
	$P\bar{v}O_2$[f)] (mmHg)	12	40.2±7.2	46.0±5.2	NS[b)]
	$P\bar{v}CO_2$[g)] (mmHg)	12	37.1±8.1	41.6±5.6	0.01
	$a\bar{v}DO_2$[h)] (mmHg)	12	43.9±8.6	38.8±9.8	NS
	pH	21	7.313±0.059	7.318±0.053	NS
	HCO_3^{-1} (mmol/*l*)	21	14.7±3.3	16.8±2.7	0.01
Nonsurviving	PaO_2 (mmHg)	10	62.3±10.4	61.6±9.4	NS
	$PaCO_2$ (mmHg)	10	30.9±4.8	30.5±6.8	NS
	$AaDO_2$ (mmHg)	10	48.9±14.2	49.9±14.1	NS
	$P\bar{v}O_2$ (mmHg)	5	37.1±5.5	35.3±5.1	NS
	$P\bar{v}CO_2$ (mmHg)	5	40.0±2.5	40.1±8.4	NS
	$a\bar{v}DO_2$ (mmHg)	5	31.4±6.2	27.0±5.1	NS
	pH	10	7.292±0.082	7.342±0.056	NS
	HCO_3^- (mmol/*l*)	10	15.1±4.3	16.2±3.1	NS

Data are expressed as mean±standard deviation. a) Probability of significant difference from the value before heartworm removal; b) Not significant; c) Arterial oxygen tension; d) Arterial carbon dioxide tension; e) Alveolar arterial oxygen difference; f) Mixed venous oxygen tension; g) Mixed venous carbon dioxide tension; h) Arterial mixed venous oxygen difference; i) Bicarbonate concentration.

$p<0.01$), and TPUR ($r=0.56$, $p<0.01$).

Table 6 shows changes in blood gas tensions after HW removal. In surviving dogs, the PaO_2 ($p<0.05$) and $PaCO_2$ ($p<0.01$) increased, and the $AaDO_2$ decreased ($p<0.01$). The $P\bar{v}O_2$ increased slightly (but not significantly), and the $P\bar{v}CO_2$ increased ($p<0.01$). The pH did not alter, but HCO_3^- concentration increased ($p<0.01$). In nonsurviving dogs, no blood gas variables changed significantly.

DISCUSSION

The mean minus 2 standard deviation of PaO_2 in HW-free dogs of the present study was 76.9 mmHg. Generally, hypoxemia is defined by a PaO_2 less than 60 mmHg [5, 14]. Hypoxemia indicates a disturbance in gas exchange in the lung resulting from maldistribution of ventilation and perfusion, pulmonary shunt and diffusion

insufficiency in the lung [5, 14]. Low PaO_2 is a very sensitive though non-specific sign of pulmonary embolism [17, 18]. In dogs with CS, the hypoxemia was detected in approximately 30% of surviving dogs and in approximately 40% of nonsurviving dogs, and hypoxemic dogs showed severe dyspnea. The prevalence of hypoxemia in dogs with CS was higher than in dogs with ascitic pulmonary HW disease, in which low PaO_2 less than 60 mmHg was rare [11]. In dogs with CS, thromboemboli containing dead HWs, which were formed recently, have been observed in almost all dogs [10]. It was shown in the previous study [10] that the degree of thromboembolization correlated positively with MPAP and TPUR, and consistent with a ventilation-perfusion abnormality, PaO_2 correlated negatively with MPAP as shown in the present study. Thus, hypoxemia may be caused by the abnormality in pulmonary circulation following thromboembolization with dead HWs in dogs with CS. Consolidation and pneumonia around thromboemboli and pulmonary edema, which have been observed in many dogs with CS, also might contribute to low PaO_2. Moreover, the hypoxia associated with pulmonary lesions contributes to the development of pulmonary hypertension by causing pulmonary arterial vasoconstriction [13, 16]. Thus, in dogs with CS, the ventilatory function was disturbed by pulmonary lesions. However, cardiac output was lower in many dogs with CS than in dogs with pulmonary HW disease because of severe pulmonary lesions and tricuspid stenosis and regurgitation owing to the presence of HWs. The pathogenesis of alterations in arterial blood gas tension might be decrease in the O_2 content of mixed venous blood in pulmonary thromboembolism [4]. This low $P\bar{v}CO_2$ level has also contributed to a low PaO_2 level in dogs having ventilation-insufficient lungs. The large pulmonary shunt ratio in nonsurviving dogs (unpublished data) indicated that a considerable amount of venous blood might pass through the lung without gas exchange. Therefore, many dogs with CS were hypoxemic, despite attempts to correct hypoxemia by hyperventilation (low $PaCO_2$), a characteristic response to acute pulmonary vascular occlusion [4]. Changes in blood gas tensions 1 week after HW removal might indicate improved pulmonary circulation and gas exchange. Improvement of tricuspid valve function and pulmonary circulation by HW removal may also contribute to improvement in gas exchange.

Low $P\bar{v}O_2$ and $P\bar{v}CO_2$ levels in dogs with CS might reflect low cardiac output or inability of cardiac output for tissue O_2 demand. In dogs with CS, low oxygen delivery was assumed, based on low cardiac outputs [10], low blood Hb concentrations [1, 6, 12], and low PaO_2 (present study). Generally, patients having a low $P\bar{v}O_2$ have an ominous prognosis [19]. Also, in the present study, many nonsurviving dogs had a low $P\bar{v}O_2$ just as human patients with severe respiratory disorders also have a poor prognosis. The tendency for $P\bar{v}O_2$ and $P\bar{v}CO_2$ to increase after HW removal suggests an increase in oxygen delivery and improvements in blood gas exchange in peripheral tissues.

Relatively fresh thromboemboli including dead HWs were observed in many dogs with CS [10]. Generally, acute thromboembolization results in hypocapnia and acute respiratory alkalosis [4]. However, many dogs with CS had metabolic acidosis, which was indicated from low pH and HCO_3^- and greater base excess and anion gap. The serum lactic acid level was high in many dogs with CS, especially in dogs of the nonsurviving group. The acidosis observed might involve lactic acidosis attributable to severe circulatory disturbance, as expected by Cornelius and Rawlings [3]. However, serum lactic acid concentration did not fill in completely the anion gap. Many and complicated factors such as acute and chronic renal failure, renal tubular lesions, liver failure, intravascular hemolysis, respiratory disturbance and intestinal disorders [1, 2, 6, 8, 12, 15, 16] might influence the pathophysiology of the acidosis. Moreover, correction for the acid-base imbalance through hyperventilation, which was induced by metabolic acidosis and pulmonary lesions [5, 19], makes the interpretation of data on acid-base balance more difficult. Further investigations are needed for a more precise elucidation of the metabolic acidosis in CS. After HW removal, metabolic acidosis tended to be reduced in surviving dogs owing to improved circulation.

In many dogs with CS, compensatory mechanisms keep abnormal blood gas values close to the normal range. However, abnormal blood gas values might be a reflection of circulatory disturbance and ventilation insufficiency, and might be associated with organ injuries. The authors consider that determination of blood gases is valuable for treatment of dogs with CS, because the appropriate application of pH and blood gas measurements to the clinical management of a patient is helpful, and, in some cases, vital to a successful therapeutic endeavor [5].

ACKNOWLEDGEMENTS. The authors thank Professor David H. Knight, Department of Clinical Studies, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, U.S.A. for helpful suggestion and careful review of the manuscript.

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