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RETROSPECTIVE STUDY

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Retrospective evaluation of jugular venous blood variables and mortality in critically ill hospitalized cats

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Abstract

Objective: To evaluate clinicopathological variables associated with hospital mortality in critically ill cats with compromised hemodynamics and tissue hypoperfusion.

Design: Retrospective observational study.

Setting: Private referral center.

Animals: Fifty-seven critically ill cats with compromised hemodynamics or tissue hypoperfusion.

Interventions: None.

Measurements and Main Results: The electronic medical records were searched for all cats admitted from June 2014 to November 2020. Cats were included in the study if the medical records clearly identified the presence of compromised hemodynamics and tissue hypoperfusion. Blood samples were obtained by percutaneous puncture of the external jugular vein, and blood gases, electrolytes, L-lactate concentration, and glucose were measured by a point-of-care analyzer. A predictive logistic regression model for mortality was performed. A total of 57 cats were ultimately included in the study. Thirty-five cats died. Eighteen of them were euthanized because of the severity of illness, and 17 died naturally. Twenty-two cats were discharged alive from the hospital. After adjusting for the Acute Patient Physiologic and Laboratory Evaluation (APPLE) fast score and disease category, jugular venous partial pressure of oxygen

Abbreviations: APPLE, Acute Patient Physiologic and Laboratory Evaluation; AUROC, area under the receiver operating characteristic; BIC, Bayesian Information criteria; LACA-24, fractional decrease in L-lactate over 24 hours; $Pcvo_2$, central partial pressure of oxygen; $Pvjo_2$, jugular venous partial pressure of oxygen; $Scvo_2$, central venous oxygen saturation; $Smvo_2$, mixed venous oxygen saturation.

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($PvjO_2$) and HCT at admission were independent predictors of hospital mortality (HCT: odds ratio [OR], 0.763, 95% confidence interval [CI]: 0.625–0.930; $P = 0.008$; $PjvO_2$: OR, 0.858; 95% CI: 0.749–0.984; $P = 0.029$). The association of these variables with mortality was maintained after conducting a sensitivity analysis and excluding cats that died by euthanasia.

Conclusions: In cats with hemodynamic instability and tissue hypoperfusion, HCT and $PvjO_2$ behaved as independent predictors of mortality. Both variables seem to reflect the magnitude of oxygen debt and tissue hypoperfusion.

KEYWORDS
feline, jugular venous oxygenation, packed red blood cells

1 | INTRODUCTION

Circulatory shock is a life-threatening condition, in which the cardiovascular system is unable to deliver enough oxygen to satisfy tissue demand.[1] The persistence of the resulting anaerobic metabolism predictably results in multiple organ failure and death.[1]

Even though the main determinant of tissue hypoxia in shock states is hypoperfusion, alterations in the other components of oxygen delivery—hemoglobin and arterial oxygen saturation and content—can additionally hamper tissue oxygenation.[2] Because a critical level of hemoglobin is needed to maintain microvascular patency, anemia can not only decrease arterial oxygen content but can also compromise tissue perfusion.[3]

Mixed venous oxygen saturation ($SmvO_2$), as a surrogate for mixed venous oxygen content, reflects the balance between oxygen transport and consumption.[4] Given the difficulties in sampling blood from a pulmonary artery, the measurement of central venous oxygen saturation ($ScvO_2$) is a common approach to tissue oxygenation in critically ill people.[5] Despite the fact that $ScvO_2$ might not always accurately represent $SmvO_2$, low values in shock patients usually express reductions in oxygen availability.[4] In fact, low $ScvO_2$ has been linked to a poor outcome of shock, both in dogs and in people.[6–8] Although the sampling of central venous blood in cats is technically challenging, a suitable alternative is the measurement of jugular vein samples.

The identification of clinical and laboratory variables, which are associated with outcome, could be useful for selecting high-risk patients, who eventually require a more aggressive treatment. Despite the relevance of this question, these bedside variables have not been extensively studied in cats. The objective of the present retrospective study was to identify independent predictors of outcome in cats admitted to the hospital because of arterial hypotension and manifestations of tissue hypoperfusion. Our hypothesis was that, on admission, L-lactate concentration, jugular venous partial pressure of oxygen ($PjvO_2$), and HCT would be independent predictors of in-hospital mortality in cats with shock.

2 | METHODS

This is a retrospective cohort study of cats presenting to a single veterinary referral hospital with hemodynamic and tissue perfusion abnormalities. The electronic medical records were searched for all cats admitted to the Cooperative Veterinary Intensive Care Unit (UCI-COOP) Buenos Aires, Argentina, from June 2014 to November 2020. The principal investigator (P.D.) performed the data collection. Cats were included in the study if the medical records clearly identified the presence of 1 of the following criteria on admission: Arterial hypotension, along with at least 1 sign of tissue hypoperfusion, such as a prolonged capillary refill time (>2 s), pale mucous membranes, altered mental status, and L-lactate concentration >2.5 mmol/L. Arterial hypotension was defined as the absence of palpable pulse (femoral or metatarsal) or a systolic arterial pressure ≤90 mm Hg.[9–11] Altered mental status was defined as a score >0 in the mentation score of the feline Acute Patient Physiologic and Laboratory Evaluation fast score (APPLE fast score).[12] We also included cats without arterial hypotension but showing hyperlactatemia (L-lactate concentration >2.5 mmol/L) on admission together with at least 1 other sign suggestive of tissue hypoperfusion, such as prolonged capillary refill time (>2 s), mucosal pallor, or altered mentation.

We excluded patients with severe respiratory diseases and seizures because the clinical manifestations of these conditions might overlap with those of shock and hypoperfusion. We also excluded cats with incomplete medical records.

2.1 | Measurements

In all cases, a blood sample was obtained by percutaneous puncture of the external jugular vein. A veterinarian trained in emergency and critical care performed this procedure. Blood samples were extracted with nonheparinized syringes and analyzed immediately. Venous blood gases and electrolytes,[a] L-lactate concentration,[b] and blood glucose[c] were measured by point-of-care analyzers.

Indirect blood pressure measurements were obtained using a Doppler device.[d] Doppler cuffs were placed on the forelimb just below the elbow. For each cat, a cuff with a width between 30% and 40% of the limb circumference was chosen.[13] Core temperature was digitally registered with a rectal probe.[e]

In the absence of a metatarsal pulse and Doppler blood pressure measurements, a systolic blood pressure of 75 mm Hg was imputed, as previously reported.[10] In case the glucometer showed a blood glucose value of HIGH, a value of 33.3 mmol/L (600 mg/dl) was assigned to the cat based on the manufacturer's suggestion. In case the thermometer showed the word LOW as a result, a value of 32°C (89.6°F) was assigned to the cat based on the manufacturer's suggestion. Finally, if only tachypnea was registered in the medical records, a respiratory rate of 60/min was assumed.

The value of the mental score of the APPLE score together with the rest of the variables included in the study was collected at the time of hospital admission. To complete the APPLE fast score, the worst values of temperature, L-lactate concentration, HCT, and blood pressure during the first 24 hours were used, as previously suggested.[13] When possible, fractional decrease in L-lactate over 24 hours (LACA-24) was evaluated.

The primary disease process was determined by the attending clinicians and categorized according to the compromise of major organ systems: gastrointestinal and pancreatic, hepatobiliary, renal, or metabolic-endocrine.[14] Because there was a low incidence of the rest of the pathologies, a category named "others," which included trauma, intoxication, neoplasia, and neurological diseases, was added.

All data were passed from the medical records to a standardized data sheet that was later exported to commercial statistical software[f] for further analysis.

2.2 | Statistical analysis

For the descriptive analysis, the quantitative variables were reported as mean ± standard deviation or median and interquartile range (25th to 75th percentile) according to their distribution. To assess data distribution, histograms were evaluated along with the Shapiro–Wilk test for normality. Comparisons between 2 groups for nonnormally distributed continuous variables were performed with the Wilcoxon rank-sum test and with Student's *t*-test for normally distributed variables. Qualitative variables were reported as absolute and relative frequencies with proportions. Comparisons between qualitative variables were performed using Fisher's exact test or the chi-square test, as appropriate. To construct a predictive model, a logistic regression was used. Potentially predictive variables were initially included in the model if they had a *P*-value < 0.2 in the univariate analysis. Variables with a significant number of missing data were not included in the multivariable analysis. To select the final model, the Bayesian information criteria (BIC) was used. The model with the lowest (closest to zero) value for BIC was chosen. The variables of category of illness and APPLE fast score were entered into the model as adjusting variables. The interaction among significant risk factors was examined.

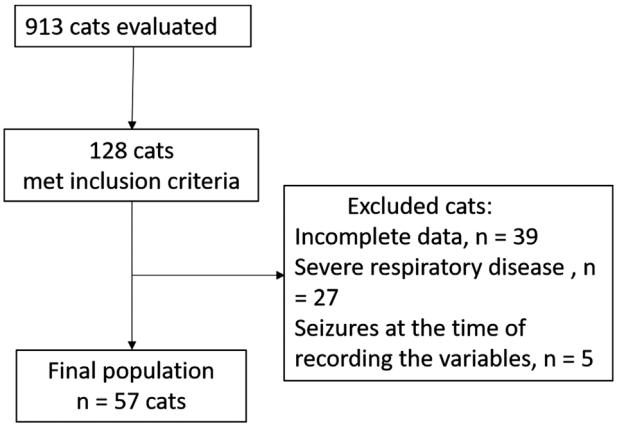


FIGURE 1 Flowchart. Description of the numbers of cats evaluated and the reasons for exclusion are shown

The Hosmer–Lemeshow test and the area under the receiver operating characteristic (AUROC) were used to assess the calibration of each model and its discriminatory power. Odds ratios and the 95% confidence interval were reported for the selected variables in the final models. Because the use of humane euthanasia might be a potential source of bias, especially in retrospective studies, in which the decision might be based on reasons unrelated to the illness progression, a sensitivity analysis was performed. In this analysis, we tried to rule out that the exclusion of euthanized cats was able to modify the results. All the statistical calculations were performed using commercial statistical software.[f]

3 | RESULTS

A total of 913 medical records were evaluated. Inclusion criteria were met in 128 cases. After the exclusion of 71 patients, 57 cats were included in the study (Figure 1). The breed was undefined in 47 (82%) of them, Siamese in 6 (11%), Exotic in 2 (4%), Persian in 1 (2%), and Russian Blue in 1 (2%). Of the 35 cats that died 18 (51 %), were euthanized because of the severity of illness, and 17 (49 %) died naturally. Twenty-two (39%) cats were discharged alive from the hospital. Table 1 shows the main demographic, clinical, and biochemical characteristics of survivors and nonsurvivors.

The results of the univariate analysis are shown in Table 1. Jugular venous partial pressure of oxygen and HCT were significantly lower in nonsurvivors compared to survivors. Interestingly, HCT was not significantly different between cats that had pale mucous membranes at admission and those that did not (0.30 L/L ± 0.10 [30% ± 10%] vs. 0.32 L/L ± 0.08 [32% ± 8%], $P = 0.428$), suggesting that pallor was related to an altered perfusion status instead of a low HCT.

In the logistic regression model, $Pjvo_2$ and HCT variables were included, according to the best model selection based on the lowest BIC (Table 2). The adjusting variables category of illness and APPLE fast score were also included. No collinearity between $Pjvo_2$ and HCT was found ($Rs = 0.04$; $P = 0.764$). The model showed an adequate goodness

TABLE 1 Results of demographics and clinicopathological variables in a cohort of critically ill hospitalized cats with hemodynamic instability or tissue hypoperfusion

Variable	Overall $n = 57$	Nonsurvivors $n = 35$	Survivors $n = 22$	*P*-value
Age (years)	8.6 ± 5.4	9.6 ± 4.9	7.0 ± 4.9	0.074
Females, *n* (%)	24 (42)	15 (43)	9 (41)	0.885
APPLE score fast	31.4 ± 4.9	32.4 ± 5.0	30.0 ± 4.5	0.073
Heart rate (/min)	151 ± 46	147 ± 44	158 ± 51	0.405
Respiratory rate (/min)	24 (20–32)	24 (20–36)	24 (20–32)	0.470
Systolic arterial pressure (mm Hg)	84 ± 28	80 ± 24	90 ± 33	0.182
Capillary refill time ≥2 seconds, *n* (%)	11 (42) $n = 27$	9 (60) $n = 15$	5 (42) $n = 12$	0.343
Pale mucous membranes, *n* (%)	30 (53)	22 (63)	8 (36)	0.051
Temperature (°C)	35.60 (33.60–36.40)	35.60 (33.00–36.90)	35.45 (34.60–36.40)	0.628
L-lactate concentration (mmol/L)	3.86 ± 2.51	4.19 ± 2.80	3.35 ± 1.90	0.217
LACA-24 (%/h)	4.2 (2.8–6.5)	4.2 (3.3–8.2) $n = 19$	3.8 (0.0–6.2) n = 9	0.121
HCT (L/L)	0.29 (0.25–0.36)	0.25 (0.20–0.32)	0.35 (0.32–0.40)	<0.001
Base excess (mmol/L)	−11 (−16 to −4)	−12 (−17 to −4)	−10 (−16 to −3)	0.688
pH	7.260 (7.06–7.34)	7.26 (7.02–7.35)	7.25 (7.14–7.33)	0.831
$Pjvo_2$ (mm Hg)	42 ± 11	40 ± 11	46 ± 9	0.033
Pco_2 (mm Hg)	39 (36–52)	40 (35–52)	40 (37–48)	0.606
Sodium (mmol/L)	146 (142–152)	146 (139–153)	146 (143–152)	0.716
Potassium (mmol/L)	3.6 (2.4–4.5)	3.6 (2.3–4.8)	3.7 (2.8–4.4)	0.426
Chloride (mmol/L)	109 ± 13	107 ± 15	112 ± 10	0.206
Anion gap (mmol/L)	15 (11–22)	16 (11–21)	14 (13–23)	0.755
Ionic Ca (mmol/L)	1.10 ± 0.20	1.081 ± 0.22	1.13 ± 0.17	0.083
Blood glucose (mmol/L)	8.16 (5.16–13.88)	7.33 (4.27–14.99)	8.77 (5.72–13.88)	0.387
Bicarbonate (mmol/L)	16 (14–22)	16 (12–22)	16 (14–22)	0.795
Category of illness				
Renal	23 (40)	15 (43)	8 (36)	0.627
Hepatobiliary	9 (16)	7 (20)	2 (9)	0.458
Gastrointestinal-pancreatic	8 (14)	4 (11)	4 (18)	0.697
Metabolic-endocrine	3 (5)	2 (6)	1 (5)	1.000
Others	14 (25)	7 (20)	7 (32)	0.181

Abbreviations: CRT, capillary refill time; HCT, hematocrit; LACA-24, fractional decrease in L-lactate over 24 hours; $Pjvo_2$, jugular venous partial pressure of oxygen.

of fit (Hosmer–Lemeshow test: $P = 0.899$) and discrimination capacity (AUROC = 0.89). After excluding the animals that were euthanized, the association between $Pjvo_2$ and HCT with mortality was maintained (Supporting Information).

4 | DISCUSSION

The main finding of this study was that HCT and $Pjvo_2$ behaved as independent predictors of mortality in cats admitted to the hospital because of shock and signs of hypoperfusion. This predictive ability remained even after adjustments to the severity (APPLE score) and the category of the illness.

Regardless of its cause, anemia can primarily affect the outcome because of the decrease in convective oxygen transport.[15] In addition, anemia can also alter tissue oxygenation by other mechanisms related to the microcirculatory oxygen transport. Because a critical level of viscosity is needed to maintain microvascular patency, hemodilution induces microvascular alterations.[16] Therefore, anemic hypoxia is associated with a microcirculatory disorder that hampers tissue oxygen extraction and oxygenation. For the same degree of reduction in systemic oxygen transport, these alterations are worse in anemic than in ischemic hypoxia.[3] The association between HCT and mortality is probably related to a combination of factors. Low HCT contributes to the decrease in the tissue oxygen transport already compromised by hypoperfusion. In addition, anemia can also affect tissue oxygenation

TABLE 2 Results of the predictive logistic regression model with the whole population

Whole population ($n = 57$)			
Variable	**Odds ratio**	**95% CI**	***P*-value**
HCT	0.763	[0.625–0.931]	0.008
$Pjvo_2$	0.917	[0.846–0.995]	0.037
APPLE fast score	1.142	[0.960–1.357]	0.132
Category of illness			
Renal (Basal)			
Hepatobiliary	0.890	[0.085–9.315]	0.923
Gastrointestinal-pancreatic	0.890	[0.071–11.028]	0.928
Metabolic-endocrine	1.383	[0.055–34.377]	0.843
Others	0.285	[0.038–2.091]	0.217

Abbreviations: APPLE, Acute Patient Physiologic and Laboratory Evaluation; HCT, hematocrit; $Pjvo_2$, jugular venous oxygen pressure.

by different mechanisms unrelated to the oxygen-binding ability. Normovolemic anemia induces a microcirculatory disorder characterized by reduction in capillary density and red blood cell velocity, which are dependent on the low plasma viscocity.[15] In addition, recent studies have shown that a microscopic area, called the cell-free layer, critically regulates microvascular perfusion.[16] This cell-free layer determines the distance between the circulating oxygen supply and the tissue and the degree of shear stress on the endothelial wall caused by the passing blood. Small changes in HCT result in large changes in the cell-free layer, thus affecting arteriolar wall nitric oxide bioavailability and wall shear stress.[16]

Interestingly, we found a trend to a greater proportion of nonsurviving patients with pale mucous membranes. Because HCT levels were not significantly different between cats with and without pale mucous membranes at admission, these findings suggest that pallor was related to an altered perfusion status instead of a low HCT.

In the present study, we also included cats with evidence of tissue hypoperfusion, albeit blood pressure was normal. This condition has been previously characterized as occult shock.[17] Even in the absence of arterial hypotension, tissue hypoperfusion can compromise aerobic metabolism. Moreover, people with septic shock have a worse outcome.[6]

In the present study, no differences were found in L-lactate concentration at admission between survivors and nonsurvivors, which is in line with previous data on cats with shock.[18,19]

The reason why neither the L-lactate concentration at admission nor LACA-24 was associated with in-hospital mortality in the present study is not entirely clear. In a previous study, a relationship was observed between the struggling experienced during blood sampling and the presence of hyperlactatemia.[20] Thus, struggling could generate increases of L-lactate concentration, even in the absence of tissue hypoxia, making interpretation difficult. Considering that our study was retrospective and struggling was not registered, this mechanism cannot be ruled out. On the other hand, in a previous study carried out in cats with arterial hypotension, it was observed that hypotensive normolactatemic cats had a significantly greater chance of survival to hospital discharge than their hyperlactatemic counterparts.[21] These findings suggest that plasma lactate concentration could play a role as a prognostic indicator. Likewise, a previous study on 185 cats admitted to an emergency room showed that L-lactate concentration was an independent predictor of mortality.[22]

Taking into consideration all these studies, we consider that further studies are required to draw definitive conclusions about the role of lactate as a prognostic marker in critically ill cats.

The blood gas analyzer used in the present study measures the partial pressure of oxygen directly, whereas it calculates the oxygen saturation using the human oxyhemoglobin dissociation curve. Therefore, the $Pvjo_2$ was used instead of the $Scvo_2$. Although the value of $Scvo_2$ yields more information than the $Pvjo_2$ both in critically ill people[5,23,24] and dogs,[5,7,8] 2 recent studies in critically ill human patients have shown a high correlation between the central partial pressure of oxygen ($Pcvo_2$) and $Scvo_2$ measured when measured by co-oximetry.[25,26] Because the agreement between $Scvo_2$ measured by co-oximetry and the $Scvo_2$ calculated from blood gases in those studies was low, the authors suggested using $Pcvo_2$ instead of $Scvo_2$ to guide therapy if measuring $Scvo_2$ by co-oximetry is not possible.[25,26]

Jugular venous partial pressure of oxygen was a strong independent predictor of mortality in the present study. The prognostic ability of venous oxygenation should be linked to the magnitude of hypoperfusion and anaerobic metabolism. Low venous oxygen saturation and partial pressure primary express a compromised balance between oxygen transport and consumption.[4] Even though the main alteration in shock is the decrease in oxygen transport, an increased respiratory workload in spontaneously breathing animals might have also contributed to the reduction in venous oxygenation.[27] As these variables are relatively easy to obtain at bedside and are useful to evaluate oxygenation,[4] our findings suggest that $Pvjo_2$ could be useful to establish prognosis in critically ill cats admitted to the hospital.

When interpreting this study, it should be considered that blood gas samples were not corrected by the patient's temperature. The solubility of oxygen and carbon dioxide in the blood increases as temperature decreases and vice versa; therefore, temperature changes can cause shifts in the oxyhemoglobin dissociation curve. Because most patients presented with marked hypothermia, the lack of correction of blood gases with temperature may have affected the results. Thus, Po_2 values that are not corrected by the current body temperature may be higher than during normothermia.[28]

We adjusted our predictors to both the illness category and severity. For this last purpose, we used the feline APPLE score, which is a severity of illness stratification system for hospitalized cats that accurately predicts mortality.

This study has some limitations. We sampled blood from the external jugular vein, instead of an actual central vein. Venous oxygen saturation and partial pressure may be different in the pulmonary artery, superior vena cava, or other regional veins.[4] Unlike the internal jugular vein, which can display distinct anatomic conformations, the

external jugular vein shows a regular morphology.[29] Thus, sampling is simpler from this location. Regardless of the issue of interchangeability among different veins, $PvjO_2$ behaved as an independent predictor of outcome.

Another important limitation is that in approximately 30% of cases, we were not able to measure blood pressure. Because these patients had an absent metatarsal pulse, a systolic arterial blood pressure value of 75 mm Hg was assigned. This decision was based on a previous study carried out in 102 cats showing that the absence of metatarsal pulses correctly identified cats with a blood pressure of ≤75 mm Hg 84% of the time.[10]

It should be considered that patients with absence of a metatarsal pulse to whom a systolic arterial blood pressure value of 75 mm Hg was imputed may had an even lower systolic arterial blood pressure value, which may have affected the results of the APPLE score by underestimating the degree of hypotension.

In conclusion, this retrospective study showed that HCT and $PjvO_2$ are independent predictors of outcome in cats that present in shock and signs of hypoperfusion. Larger studies are required to confirm our findings.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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ENDNOTES

[a] EPOC Blood Analysis System, Siemens Healthcare Diagnostics Inc, Newark, NJ.
[b] Accutrend Plus System, F. Hoffmann-La Roche Ltd, Basel, Switzerland.
[c] One touch ultra, LifeScan Inc, Milpitas, CA.
[d] Ultrasonic Doppler Flow Detector, Model 811 Series, Parks Medical Electronics, OR.
[e] TERMÓMETRO DIGITAL FEVER BLUE MT-31, Aspen salud, Atanasio Girardot 1449 C1427 AKE - CABA - Argentina.
[f] STATA 13.0, Stata Corporation, College Station, TX.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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