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Original Study

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Decreased central venous oxygen saturation despite normalization of heart rate and blood pressure post shock resuscitation in sick dogs

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Abstract

Objective – To evaluate traditional and global perfusion parameters in clinical canine shock patients, and to evaluate for occult hypoperfusion as evidenced by low central venous oxygen saturation or high plasma lactate concentrations in clinical patients resuscitated to traditional endpoints.
Design – Clinical observational trial designed with a 1-year data entry period and patient follow-up of 28 days posthospital presentation.
Setting – Large, private urban teaching hospital, and emergency and critical care center.
Animals – Adult canine patients presenting to the emergency department with untreated shock.
Interventions – None.
Measurements and Main Results – Patients received fluid resuscitation to normalize perfusion parameters based on physical examination and arterial blood pressure (BP). Monitoring of central venous pressure (CVP) and central venous oxygen saturation ($ScvO_2$) was feasible with current standard of care interventions in critically ill, client-owned dogs. Decreased $ScvO_2$ was observed in 37.8% of patients resuscitated to normal traditional perfusion parameters. Hyperlactatemia was commonly recorded.
Conclusions – Decreased $ScvO_2$ exists in a significant proportion of critically ill dogs following standard fluid resuscitation for shock, providing a relevant target population for implementation of a more standardized early goal-directed therapy bundle in veterinary patients. Normalization of heart rate, blood pressure, mentation, and perfusion parameters directed by physical examination may be attained despite the persistence of significant tissue hypoperfusion and oxygen debt.

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Keywords: early goal-directed therapy, lactate, patient monitoring

Abbreviations

BE	base excess
BP	arterial blood pressure
CaO_2	arterial oxygen content
CVP	central venous pressure
DO_2	oxygen delivery
EGDT	early goal-directed therapy
ED	emergency department
HR	heart rate
MAP	mean arterial pressure
MODS	multiple organ dysfunction syndrome
PE	physical examination
$ScvO_2$	central venous oxygen saturation
$SmvO_2$	mixed venous oxygen saturation
UOP	urine output

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Introduction

Shock is defined as failure to meet the cellular metabolic demands of the body. Resuscitation is complete when aerobic metabolism is restored to all tissue beds, and oxygen debt eliminated. Persistent hypoperfusion despite resuscitation efforts is associated with increased morbidity, development of multiple organ dysfunction syndrome (MODS), and death.[1–6]

Historically, standard of care shock resuscitation has focused on normalization of traditional perfusion

parameters (heart rate [HR], mean arterial pressure [MAP], and urine output [UOP]). However, it has been demonstrated that many human shock patients have evidence of occult shock (ie, ongoing tissue hypoperfusion) despite normalization of these parameters.[7–9] For this reason, several investigators have demonstrated the utility of various physiologic markers of global perfusion to assess the success of early resuscitation efforts on shock reversal. These markers, including plasma lactate, base excess (BE), and venous oximetry (measuring central [$ScvO_2$] or mixed [$SmvO_2$] venous oxygen saturation) are more sensitive indicators of tissue hypoperfusion than are traditional hemodynamic variables.[7,9,10] Furthermore, timing of shock reversal (ie, normalization of chosen endpoints of resuscitation) is crucial to achieving a successful clinical outcome. Early achievement of set resuscitation goals (within 6 hours of presentation) has been shown to positively impact outcome in certain patient populations, and early goal-directed therapy (EGDT) in human patients with severe sepsis and septic shock is now a Grade 1C recommendation in the Surviving Sepsis Campaign Guidelines.[1,11]

In decompensated shock, cells rely on anaerobic metabolism to convert pyruvate to lactate for ATP production. Thus, in nonseptic patients with normal liver function, hyperlactatemia may reflect decreased cellular perfusion.[12] An increase in the oxygen extraction ratio is observed with inadequate oxygen delivery (DO_2).[13] Increased oxygen extraction can compensate for decreased DO_2 to a critical point (supply independence), at which time oxygen consumption will begin to decrease with diminished DO_2 (supply dependence). Increased oxygen extraction results in a decrease in $ScvO_2$, which may reflect a state of global tissue hypoperfusion and predicts increased morbidity and mortality among the human intensive care patient population.[14–16] In addition to hypoperfusion, low $ScvO_2$ may be attributed to anemia, hypoxemia, hyperthyroidism, seizure disorders, and pyrexia.

Venous oxygen saturation can be measured continuously via specialized fiberoptic catheters attached to reflectance oximetry, or serially with a cooximeter.[17] While intensivists still rely on traditional perfusion parameters to assess shock patients and success of resuscitation efforts, clearance of blood lactate, and normalization of $ScvO_2$ have become widely utilized as endpoints of resuscitation and are becoming standard of care in the human critical care arena.[18–21]

The rate of lactate clearance is both a better predictor of patient outcome than a single lactate measurement and a sensitive indicator of shock reversal.[22–24] In a recent prospective, randomized human trial evaluating goal-directed endpoints of resuscitation in severe sepsis, 6 hour lactate clearance of >10% was equivalent to achieving a $ScvO_2$ of >70% in predicting outcome.[21]

A canine study demonstrated the utility of serial lactate measurements in patients with Babesiosis, showing a correlation between blood lactate clearance of <50% at 8 and 16-hour post-presentation time points and decreased survival.[25] A retrospective study of canine IMHA patients documented 100% survival in emergent patients with hyperlactatemia that resolved within 6 hours of presentation, whereas dogs with persistent hyperlactatemia had only a 71% survival.[26] In another canine study, lactate clearance of <50% or a blood lactate >2 mmol/L at 6 hours into treatment of critically ill dogs was associated with significantly increased mortality.[27]

Shock is commonly encountered in small animal patients presenting to the emergency department (ED). However, ideal endpoints of resuscitation and the effect of EGDT on patient outcome have been minimally investigated in clinical patients. As is the case with critically ill human patients, diagnosis and treatment of occult hypoperfusion in veterinary patients is vital to successful early shock reversal and prevention of MODS and death.

The objectives of this study were to evaluate shock reversal in canine shock patients by comparing traditional perfusion parameters (physical examination [PE] and blood pressure [BP]) with indices of global perfusion (venous plasma lactate, lactate clearance, BE, and $ScvO_2$).

Materials and Methods

This prospective study evaluated 30 dogs that presented with clinical signs of shock to the ED of a large, urban, private veterinary teaching hospital. Inclusion criteria included: (1) adult dogs (greater than 6 months of age) and (2) documented hypotension (Doppler BP <90 mm Hg). Dogs were excluded if there was a preexisting diagnosis that might confound plasma lactate, lactate clearance, or venous oximetry (respiratory, hepatic, or cardiac disease; severe anemia), or if they had received treatment for shock prior to presentation to the ED. At presentation, the following data were recorded for each study participant: time of arrival; duration of illness; mentation; mucous membrane color; capillary refill time; pulse quality; HR; respiratory rate; rectal temperature; subjective perfusion of distal extremities; and Doppler BP. Concurrently, 0.5 mL of blood was collected anaerobically from a peripheral vein and placed in a syringe containing lyophilized heparin for immediate measurement of venous blood gas, sodium, potassium, chloride, ionized calcium, ionized magnesium, plasma lactate, PCV, total plasma protein via refractometry, and

blood glucose.[a] Pulse oximetry was obtained from each patient as soon as feasible to document oxygenation status.

Dogs were resuscitated to traditional end points (ie, normalization of PE perfusion parameters and indirect arterial BP using Doppler or oscillometric technology [Doppler or systolic BP>90 mm Hg]) using intravenous crystalloids or nonprotein colloids at the judgment of the primary clinician. Upon normalization of traditional endpoints judged by criticalist or senior resident, a central jugular venous catheter was placed without sedation. Preplacement measurements were taken before selecting between 3 commercially available central venous catheters: a 60 cm long silicone, 5 Fr, double lumen catheter; a 20 cm long polyurethane, 7 Fr, triple lumen catheter; and a 13 cm long polyurethane, 5 Fr, triple lumen catheter.[b,c] Location of the central venous catheter tip was confirmed via radiography to be within 1 rib space of the right atrium, after which central venous pressure (CVP) was measured with a water manometer and recorded.[d] Approximately 3 mL of blood was then collected anaerobically and placed in a blood gas syringe containing lyophilized heparin, a serum separator blood tube, and a tube containing EDTA. Central venous blood gas and cooximetry were analyzed immediately.[a] A CBC and serum biochemistry screen were analyzed within 24 hours at an on-site clinical pathology laboratory.[e,f] Lactate clearance was calculated using the following formula: [(initial – postresuscitation lactate)/initial lactate] × 100. The global perfusion values were recorded and were not used to guide ongoing therapy.

Dogs were stratified into 2 groups based on $ScvO_2$; Group A $ScvO_2$ >70% and Group B $ScvO_2$ <70%. Variables compared between the 2 groups included: signalment; body weight; rectal temperature; initial HR; postresuscitation PCV; total plasma protein; ionized magnesium; alanine aminotransferase; albumin; total bilirubin; BUN; creatinine; WBC, neutrophil and platelet counts; duration of resuscitation; volume of resuscitation fluids; venous plasma lactate (pre- and postresuscitation); lactate clearance; postresuscitation BE; postresuscitation CVP; days in the hospital; and 28 day outcome.

Paired *t*-test or Mann–Whitney rank sum test was used to compare pre- and postresuscitation continuous variables between patients groups (HR, respiratory rate, rectal temperature, BP) and markers of global tissue oxygenation ($ScvO_2$, plasma lactate, lactate clearance, and BE).[g] Mann–Whitney rank sum test was used to compare variables between patient groups, including days in the hospital, time to resuscitation, patient weight, CVP, CBC, and serum biochemistry screen variables.

Table 1: List of the breeds represented in this study

Breed	*N*
Mixed breed	6
Yorkshire Terrier	5
Chihuahua	3
Dachshund	2
Miniature Pinscher	2
Pit Bull Terrier	1
Chow Chow	1
Siberian Husky	1
Mastiff	1
Standard Poodle	1
Miniature Poodle	1
Rottweiler	1
Maltese	1
Cavalier King Charles Spaniel	1
Havanese	1
Coton de Tulear	1
Dalmation	1

Table 2: Disease categories, postresuscitation $ScvO_2$, mean lactate clearance, and survival data for the dogs included in this study

Disease	% of study population (*n*)	$ScvO_2$ <70	Mean lactate clearance (%)	Survival (%)
Gastrointestinal	66.7 (20)	6	69	90
Endocrine	10 (3)	2	68	100
Reproductive	10 (3)	1	39	0
Trauma	6.7 (2)	1	70	100
Renal	3.3 (1)	0	94	0
Sepsis	3.3 (1)	1	84	100

Results

Thirty-one dogs were initially enrolled in the study from January to November 2006. One dog was excluded because it required continued oxygen supplementation. Data for the included patients are as follows. There were 11 neutered females, 11 neutered males, 5 intact females, and 3 intact males. The median age was 6 years (range 9 months–15 years). Breeds are listed in Table 1 and underlying disease categories in Table 2. Doppler BP ranged from unmeasurable to 87 mm Hg. All dogs had hypovolemic or septic shock. Twenty-two out of 30 dogs (73.3%) were tachycardic (median HR 160; range 80–240). Hyperlactatemia, >2 mmol/L, was documented in 23/30 dogs (76.7%) [median preresuscitation venous plasma lactate was 3.82 mmol/L (range 0.8–14.6)]. A BE <–2.3 mmol/L was observed in 27/30 (90%) of dogs [median BE –8.6 mmol/L (range –22.3 to 20.6)].[28,29]

Traditional perfusion parameters (PE, HR, BP) normalized in all patients. The median duration of resuscitation was 177.5 minutes (range, 71–861 minutes). Long resuscitation times (>360 minutes) occurred (7/30 dogs; 23.3%), and were due to emergency surgery in 3/7 dogs

(42.9%), severe gastroenteritis in 3/7 dogs (42.9%), and septic arthritis in the remaining patient (14.3%). Median administered doses of isotonic crystalloids (Normosol-R, lactated Ringer's solution, or 0.9% sodium chloride) and artificial colloid fluids (hetastarch 6%) used to resuscitate to traditional perfusion parameters were 62.7 mL/kg (range 0–133.1 mL/kg) and 4.8 mL/kg (range 0–43 mL/kg), respectively.[h,i,j,k] Median postresuscitation CVP was 0 (range of –6 to 11 cm H_2O). Venous lactates and BE improved following fluid resuscitation (lactate <2 mmol/L in 77% of patients [median lactate 0.8 mmol/L (range 0.3–2.5)] and median BE –3.85 mmol/L (range –8.7 to 19.2). The median lactate clearance from pre- to postresuscitation was 71% (range 4.3–96.6%). The median $ScvO_2$ was 75.5% (range 36–88.6%).

A postresuscitation $ScvO_2$ <70% was documented in 11 of 30 patients (Group B, see Table 2 for disease categories). There was no significant difference in administered doses of crystalloids ($P = 0.485$) or colloids ($P = 0.621$) between groups. Patients with a $ScvO_2$ >70% (Group A) were significantly less anemic than patients with a low $ScvO_2$ [PCV of 41% (range 31–56) compared to 34% (range 20–46)]. Dogs with a $ScvO_2$ >70% had increased lactate clearance compared to Group B that did not reach statistical significance ($P = 0.093$) [Group A (75.7%; range 61.2–96.6%), Group B (61.5%; range 4.3–90.2%)]. Sixteen of 19 dogs (84.2%) in Group A had a postresuscitation central venous plasma lactate <2 mmol/L, compared to 8 of 11 dogs (72.7%) in Group B.

Eighty-six percent of patients survived to discharge from the hospital. Three dogs were euthanized and 1 died. It is unclear if the decision for euthanasia was based on financial constraints or prognostic information. All of these dogs had a $ScvO_2$ >70% and normal venous plasma lactate at the end of resuscitation. No further deaths by 28 days were documented. However, 5/26 discharged patients (19.2%) were lost to follow-up.

Discussion

Similar to reports in the human literature, the current veterinary study demonstrated that 38% of initially hypotensive canine shock patients with a variety of primary disease processes had a postresuscitation $ScvO_2$ <70% despite normalization of traditional perfusion parameters and clearance of venous hyperlactatemia, suggestive of occult shock.[1] These findings confirm the insensitivity of PE and BP to indicate correction of shock in clinical canine patients. Ongoing tissue hypoxia and oxygen debt can contribute to inflammation, progressive cellular dysfunction, and ultimately MODS and death.[3,30,31]

The validity of $ScvO_2$ as a surrogate marker of $SmvO_2$ has been debated. Although $ScvO_2$ is usually slightly higher than $SmvO_2$ in human patients, the values generally trend together until low flow states.[32–34] In shock states, the difference between these 2 variables is more pronounced due to heterogeneous circulation (eg, decreased splanchnic and renal perfusion), as well as the addition of blood from the coronary circulation and the besian vessels to mixed venous blood.[35] Two experimental canine models have demonstrated that venous oximetry is an accurate and sensitive marker of ongoing hemorrhage and predictor of subsequent resuscitation.[13,36] The second study demonstrated a correlation coefficient of 0.97 when comparing $ScvO_2$ with $SmvO_2$ in 22 anesthetized dogs over a wide range of volume and oxygenation states.[36] Despite $ScvO_2$ and $SmvO_2$ not being interchangeable, measuring $ScvO_2$ is more clinically accessible and recommended as part of a shock resuscitation protocol in combination with other indicators of global perfusion such as serum lactate and UOP.[11,37]

Two recent studies in clinical veterinary patients showed that $ScvO_2$ was correlated with outcome in a population of critically ill dogs.[38,39] In the Hayes patient cohort ($n = 126$), a $ScvO_2$ of less than 68% was associated with nonsurvival, with an increased mortality risk of 2.66 for each 10% drop in $ScvO_2$. Low $ScvO_2$ predicted outcome as reliably as venous plasma lactate. Patients with low $ScvO_2$ had lower MAPs in this study, suggesting that ongoing tissue hypoperfusion was in part responsible for decreased $ScvO_2$. The second group demonstrated $ScvO_2$ and BE to be reliable predictors of survival in 30 dogs with pyometra and severe sepsis or septic shock. Morbidity and mortality rates were low in this current cohort of 30 dogs and not significantly different based on postresuscitation $ScvO_2$.

In both the current prospective study and in the observational research by Hayes et al evaluating $ScvO_2$ in critically ill dogs, low PCV was associated with decreased $ScvO_2$.[38] Decreased oxygen carrying capacity will adversely impact arterial oxygen content (CaO_2) and potentially DO_2 if increases in cardiac output cannot adequately compensate. Although it is a controversial recommendation, critically ill, volume-loaded people with evidence of low $ScvO_2$ are liberally transfused to an HCT of 30% as part of EGDT.[19] However, a large, prospective, randomized trial in critically ill humans (TRICC) did not demonstrate a survival benefit of PRBCs in transfused patients without cardiovascular disease until Hb dropped below 7g/dL.[40] The risk of infectious bloodborne disease, as well as immunosuppression and other possible complications, warrants conservative transfusion practice in both human and veterinary patients. After a treatment bundle (eg, EGDT) is shown to be beneficial, prospective trials investigating specific interventions (PRBC, CVP, $ScvO_2$) are necessary to determine the survival benefit of each treatment. The survival

margin of EGDT patients might increase with more conservative transfusion targets. Evaluation for clinical signs of anemia (eg, tachycardia, tachypnea, pallor, or hyperdynamic pulse quality), in addition to the chronicity of the anemia, presence of hyperlactatemia, and high anion gap metabolic acidosis may provide helpful information to guide transfusion practice in anemic pets. Decreased $ScvO_2$ may provide additional physiologic evidence of cellular hypoxia due to anemia and could be used to guide transfusion therapy in the critically ill small animal patient.[41]

Previously established causes of a low $ScvO_2$ in human and experimental animal studies include hypothermia, decreased cardiac output, and hepatic failure. Increased oxygen consumption and subsequently decreased $ScvO_2$ may be caused by respiratory failure, stress, pain, hyperthermia, and shivering.[35,42] Although 3 of the patients in the current study had pyrexia recorded (1 pretreatment and 2 postresuscitation), they all had a normal postresuscitation $ScvO_2$. While venous oximetry provides an additional value to consider when assessing global perfusion, it must be interpreted in context of other perfusion parameters, CaO_2, and cardiopulmonary function. Hyperlactatemia with a normal to high $ScvO_2$ may indicate impaired systemic oxygen utilization as occurs with thiamine deficiency, cyanide toxicity, heterogenous microcirculatory flow, or mitochondrial dysfunction in septic patients.[8,15,43] A cut off value for high $ScvO_2$ in dogs has not yet been established.

The measured postresuscitation CVP was low in most dogs included in the current study despite normalization of lactate and $ScvO_2$. It is possible that this finding is indicative of ongoing hypovolemia and tissue hypoperfusion in the animals with a low $ScvO_2$, warranting additional volume loading (as opposed to transfusion of PRBCs). This would be supported by the fact that dogs with a normal postresuscitation $ScvO_2$ did receive a larger volume of fluid therapy although it was not statistically significant. However, static parameters of preload such as CVP are inherently insensitive and nonspecific indicators of blood volume. A recent review of the human literature, including 803 patients, concluded that CVP is not predictive of fluid responsiveness; there is no association between CVP and circulating blood volume.[44] A multicenter, randomized, controlled trial investigated people with septic shock ($n = 778$) and demonstrated that a more positive fluid balance, or CVP >8 mm Hg at 12 hours postresuscitation, were negative prognostic indicators. The authors noted that patients with high CVP had increased extravascular lung water, which decreased pulmonary compliance and may affect outcome. Also, a more positive fluid balance is associated with increased mortality in human patients with acute kidney injury requiring dialysis.[45] Research has suggested that employing CVP to assess fluid responsiveness was only useful within the first 12 hours of resuscitation in septic shock.[46]

Dynamic indices of preload (ie, stroke volume variation, pulse pressure variation, and pleth variability index), while more predictive of clinical response to fluid challenges, are widely unavailable in veterinary clinical settings. And until dynamic indices of preload become available, noting trends in CVP and MAP before and after fluid challenges may be advisable for guiding resuscitation efforts in veterinary patients. Negative consequences of excessive, large volume resuscitation with crystalloids or colloids include pulmonary edema secondary to volume overload, hypothermia, coagulopathy, abdominal compartment syndrome, worsened oxygenation in patients with acute respiratory distress syndrome, cerebral edema, hyperchloremic metabolic acidosis, and hypokalemia. Persistence of low $ScvO_2$ in the face of a high CVP and normal BP indicates the need to evaluate for and address inadequacies in CaO_2 (low PCV or PaO_2), while considering the addition of inotropic therapy in attempt to improve cardiac output and DO_2.

Several limitations exist in this study. The low mortality in this group of patients may have limited the ability to show a survival advantage in patients with a postresuscitation $ScvO_2$ >70%. The low mortality may be due to the high incidence of hypovolemia and reversible shock and low incidence of septic shock (no patients required vasopressor or inotropic therapy to maintain BP). It is possible that intervention with a central venous catheter and oximetry monitoring would be more useful at an earlier time point for some of the dogs in this study. Previous research targeting hemodynamic endpoints without strict timed-resuscitation guidelines in humans did not document the same dramatic improvements in patient outcome compared to more recent data.[1,47,48] It appears that in order to confer survival benefits, successful restoration of tissue perfusion must be accomplished with a concerted effort in a timely manner.

Other limitations include variability among clinicians treating these patients, as they may disagree on subjective parameters used to evaluate perfusion (eg, mucous membrane color, pulse quality, perfusion of distal extremities). Additionally, lactate clearance was evaluated comparing an initial venous plasma lactate obtained from a peripheral vein to a postresuscitation central venous lactate. It is likely that prior to resuscitation, venous plasma lactate in the peripheral extremities may differ from central venous plasma lactate due to vasoconstriction and centralization of circulatory flow. Venous plasma lactate from the cephalic vein in healthy dogs was shown to be significantly higher than both

Table 3: List of variables compared between patients stratified based on postresuscitation $ScvO_2$, followed by the *P* value obtained from t–test or Mann–Whitney rank sum test. Hematocrit was the only variable besides $ScvO_2$ that was statistically significant between groups

Parameter	$ScvO_2$ >70	$ScvO_2$ <70	*P* value
$ScvO_2$ (median)	81	63.8	0.001
Male versus female	6:13	6:5	0.441
Weight [median, kg (pounds)]	4.6 (10.1)	21.8 (48)	0.138
Days in hospital (median)	3	4	0.168
28 day outcome (% survival)	89	100	0.113
Resuscitation time (median, minutes)	170	180	1
Crystalloid volume (mean, mL/kg)	71.7	58.9	0.341
Colloid volume (mean, mL/kg)	6.6	11	0.621
Preresuscitation T [mean, degrees C (degrees F)]	37.2 (99)	37.8 (100)	0.293
Preresuscitation HR (mean, bpm)	151	164	0.351
Preresuscitation lactate (mean, mmol/L)	4.95	3.79	0.351
Preresuscitation BE (median, mmol/L)	−7.8	−10.6	0.132
Postresuscitation T [mean, degrees C (degrees F)]	37.7 (99.8)	38.2 (100.7)	0.267
Postresuscitation HR (mean, bpm)	119	111	0.279
Postresuscitation lactate (median, mmol/L)	0.7	1.1	0.281
Postresuscitation CVP (mean, cm H_2O)	0.4	0	0.735
Lactate clearance (mean, percent)	0.76	0.62	0.093
PCV (mean, postresuscitation)	42.6	34.8	0.006
Total plasma protein [mean, postresuscitation; g/L (g/dL)]	52 (5.2)	45 (4.5)	0.318
Ionized magnesium (median, mmol/L, postresuscitation)	0.54	0.5	0.746
Ionized magnesium (mean, mmol/L, last day)	0.51	0.5	0.59
Alanine aminotransferase (median, IU/L)	40	53	0.497
Albumin [mean, g/L (g/dL)]	20 (2.0)	21 (2.1)	0.613
Total bilirubin [median, mmol/L (mg/dL)]	3.42 (0.2)	5.99 (0.35)	0.507
Blood urea nitrogen [median, mmol/L (mg/dL)]	6.43 (18)	6.43 (18)	0.513
Creatinine [median, mmol/L (mg/dL)]	70.72 (0.8)	75.14 (0.85)	0.686
White blood cell count [median, $x10^9$/L (10^3/uL)]	13.1 (13.1)	15 (15)	0.706
Neutrophil count [median, 10^9/L (10^3/uL)]	11.5 (11.5)	11.3 (11.3)	0.781
Band neutrophils [median, 10^9/L (10^3/uL)]	0 (0)	0.14 (0.14)	0.573
Platelet count [median, 10^9/L (10^3/uL)]	214 (214)	178.5 (178.5)	0.407

arterial and jugular venous plasma lactate, although the difference was of questionable clinical relevance.[49]

This study demonstrated that a significant number of dogs suffering from hemorrhagic or hypovolemic shock have persistently low $ScvO_2$ despite resuscitation to traditional endpoints such as normalization of BP and HR. These patients were all assessed as stable and well perfused based on PE and BP but may have been experiencing regional hypoperfusion. It is possible that further resuscitation or transfusion therapy would have improved tissue perfusion and decreased morbidity in this group of dogs. Increased oxygen extraction by the tissues may indicate oxygen debt and occult shock, which is associated with poorer outcomes. Measurement of $ScvO_2$ provides the veterinary clinician an additional parameter to evaluate the relative adequacy of shock resuscitation similar to human patients. Placement of central venous catheters and cooximetry monitoring may help guide therapy and improve survival in veterinary patients undergoing major surgery, or those suffering from polytrauma or postcardiac arrest syndrome.[35,50–52] A sudden change in $ScvO_2$ can provide an early warning sign of impending deterioration in patient condition and has been validated in several trials.[20,53–56] In addition to $ScvO_2$ monitoring, central venous access provides a safe route for serial blood sampling, parenteral nutrition, CVP monitoring, and administration of vasopressor and hyperosmotic agents. The benefits of EGDT incorporating not only lactate clearance but also $ScvO_2$ to aid in rapid shock reversal in veterinary emergency and critical care patients will remain undefined until investigated in a prospective research effort.

Footnotes

[a] Critical Care Xpress, NOVA Biomedical, Waltham, MA.
[b] Arrow International, Inc., Reading, PA.
[c] Mila International, Inc., Erlanger, KY.
[d] CVP water manometer, Baxter Healthcare, Deerfield, IL.
[e] CBC Sysmex XTO 2000, Mundelein, IL.
[f] Chemistry analyzer Olympus AU400, Center Valley, PA.
[g] SigmaStat, version 3.5, Systat Software Inc., Richmond, CA.
[h] Normosol-R, Butler Schein Animal Health, Columbus, OH.
[i] Lactated Ringer's solution, Baxter Healthcare.
[j] 0.9% sodium chloride, Baxter Healthcare.
[k] Hespan 6%, Prescription Containers Inc., Secaucus, NJ.

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