Assessment of Clinical and Laboratory Variables as a Guide to Packed Red Blood Cell Transfusion of Euvolemic Anemic Dogs

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**Background:** There are no standardized guidelines for determining the likelihood that euvolemic anemic dogs will benefit from transfusion of packed red blood cells (pRBC).

**Objectives:** To report clinical and laboratory variables of dogs receiving pRBC transfusion, which could guide transfusion of other anemic dogs.

**Animals:** Twenty-four client-owned anemic dogs receiving pRBC transfusion.

**Methods:** Prospective study; 30 transfusions assessed. Clinical findings (mucosal color, pulse quality, heart rate, respiratory rate, mentation/exercise tolerance) before and after transfusion were evaluated by the anemic dog clinical assessment score (ADCAS). Hemoglobin concentration, hematocrit, venous oxygen content (CvO₂), and lactate concentration were measured from blood samples taken before and after transfusion. These results were not used for case management.

**Results:** All ADCAS variables decreased significantly with transfusion (P < .001); the total score was ≥5/12 before transfusion, and ≤3/12 in all cases that were deemed to no longer require transfusion. Hematocrit and CvO₂ were <17% and <5 mL/dL, respectively, in 83% of cases before transfusion and hematocrit concentration was <5.8 g/dL in 80%. Hemoglobin concentration, hematocrit, and CvO₂ increased significantly with transfusion (P < .001); lactate concentration decreased significantly (P = .006).

**Conclusions and Clinical Importance:** Clinical and laboratory variables improved significantly after transfusion of pRBC. By identifying how transfusion affected these variables, it was possible to recognize clinical (ADCAS) and laboratory (hemoglobin, CvO₂, lactate) variables, which could be useful in guiding the decision to transfuse dogs with similar presentations.

**Key words:** Anemia; Hemoglobin; Objective clinical assessment; Venous oxygen content.

Blood transfusions are important for the management of severe anemia in dogs. Transfusion of blood restores tissue perfusion by increasing blood oxygen carrying capacity and improving oxygen delivery (DO₂) to tissues. Several factors should be considered when deciding to transfuse blood including the risks of transfusion reactions and disease transmission, costs involved, and difficulties acquiring blood products. Transfusions should therefore be restricted to animals with clear indication of the need for transfusion.¹ ⁴

There are no universal, standardized, objective guidelines established for assessing whether people or dogs could benefit from transfusion. A critical hemoglobin concentration, a level below which cellular metabolism becomes compromised, signifying that transfusion would be beneficial, remains undetermined despite clinical and experimental studies in people and an experimental study in dogs.⁵ ¹⁰ Current procedures involve assessing hemoglobin concentration and subjectively interpreting clinical examination findings, with the possibility of interobserver variability.⁵ ¹¹ Few studies record the use of the clinical signs of severe anemia as part of the decision-making process.³ ⁶ ¹²

Markers of impaired cellular oxygenation, such as increased lactate concentration, and decreased DO₂ and cellular oxygen extraction, have been investigated alongside hemoglobin concentration to better determine whether transfusion might be required in anemic people.¹³ ¹⁴ Hyperlactatemia develops as a result of decreased DO₂ and subsequent tissue hypoxia and therefore in euvolemic, anemic dogs, it could suggest a requirement for blood transfusion.¹⁵ Central venous oxygen saturation has been investigated as an approximation of DO₂ in critically ill dogs, but requires cranial vena cava sampling.¹⁶ Venous oxygen content (CvO₂) has not been reported for evaluation of tissue oxygen status or DO₂; however, it could provide a less invasive surrogate measure of DO₂. An increase in CvO₂ after transfusion, in the absence of supplemental

**Abbreviations:**

ADCAS anemic dog clinical assessment score
bpm beats per minute
CvO₂ venous oxygen content
DO₂ oxygen delivery
Hb hemoglobin
IMHA immune-mediated hemolytic anemia
IMTP immune-mediated thrombocytopenia
pRBC packed red blood cell
PvO₂ venous partial pressure of oxygen
rpm respirations per minute
SvO₂ venous oxygen saturation of hemoglobin

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oxygen administration and hypovolemia, would suggest improved DO₂.

As there are no standardized approaches to determine when anemic dogs require pRBC transfusion, this study aimed to evaluate the effect of pRBC transfusion on hemoglobin concentration, hematocrit, CvO₂, lactate concentration, and standardized clinical assessment of anemic dogs, all of which were hypothesized to improve after transfusion, with a view to identifying levels of these variables, common to all included dogs, which could be extrapolated to other anemic dogs as guidelines to assist with the decision to transfuse.

Materials and Methods

This study was approved by the University Ethics and Welfare Committee. Anemic (hematocrit <37%) client-owned dogs admitted to the hospital that were deemed to require pRBC transfusion were included in this prospective study (October 2010–July 2012). Decision to transfuse and pRBC volume administered were independently determined by the attending clinician, who was aware of hematocrit and clinical examination findings, but blinded to anemic dog clinical assessment score (ADCAS), lactate concentration, and oxygenation values; these results were not used for case management. Dogs receiving supplemental oxygen and those with primary cardiorespiratory disease or dyspnea were excluded from the study. Dogs with neuromuscular or orthopedic disease affecting ambulation were excluded. Dogs deemed to be dehydrated, hypovolemic, or hyperbilirubinemic, and any cases of suspected transfusion reaction requiring specific treatment were also excluded. These were all considered possible confounding factors that could influence blood oxygenation, laboratory variable evaluation, or clinical examination assessment.

Age, sex, weight, diagnosis, and duration of anemia were recorded. An experienced clinical pathologist assessed for signs of regeneration (polychromasia, anisocytosis) on blood smear examination when reticulocyte count was not measured.17 The duration of anemia was determined based on the duration of clinical signs, documented anemia, or both (whichever was greater); dogs were separated into acute (<2 weeks) or chronic (>2 weeks) groups.

Dogs were blood typed (Rapid Vet-H Canine blood typing kit®) (DEA-1.1 positive or negative) before transfusion and type compatible pRBC units® were administered (DEA-1.1 negative blood was administered to dogs whose blood type could not be determined). Blood volume administered was based on the formula: volume (mL) of pRBC = (desired PCV−recipient PCV)/donor PCV × body weight (kg) × 90.18 The target PCV was determined at the discretion of the attending clinician; arbitrary hospital recommendation was to achieve a PCV of 20–30% after transfusion. Cross-matching (Rapid Vet-H Companion animal crossmatch kit®) was performed in dogs that had previously received a transfusion. Transfusions were completed without concurrent medications or intravenous fluids. Volume of blood administered, transfusion duration, and transfusion reactions were recorded.

Clinical Assessment

An anemic dog clinical assessment score (ADCAS), scored out of 12, evaluating 5 clinical variables, was designed (Table 1). Variables included mucus membrane color (compared to photographs depicting each category) (Fig 1), pulse quality, heart rate, respiratory rate, and mentation and exercise tolerance. Scores were given for each variable based on the severity of the abnormality (brackets indicate score): normal (0), mild (1), moderate (2), and severe (3). Heart and respiratory rates were categorized as normal, mildly increased (1), and moderately to severely increased (2). The same clinician examined every dog, immediately before and after transfusion, to avoid interobserver variation and was not involved in transfusion decision, but was not blinded to transfusion status.

Laboratory Variables

A venous blood sample was taken before and within 1 hour after transfusion for PCV measurement. Both blood samples were taken from the same vein. Lactate concentration (Accutrend Lactate Meter®) and oxygenation values (venous partial pressure of oxygen, P VeniceO₂, and venous oxygen saturation of hemoglobin, SvO₂), hemoglobin concentration and hematocrit, from blood gas analysis (Opti-Critical Care Blood Gas Analyser®), were assessed within 5 minutes of sample collection from surplus blood. CvO₂ was calculated from the formula: (hemoglobin × SvO₂/100 × 1.34) + (P VeniceO₂ × 0.003).19 Blood samples were taken anaerobically with heparinized syringes.d

Statistical Analysis

Descriptive statistics were performed on all data. The normality of distribution of continuous variables was assessed with the Anderson–Darling test. Normally distributed data were summarized with mean ± standard deviation, and nonparametric and categorical data with median (range). Clinical scores and variables were compared by the Wilcoxon signed rank test. The actual increase in hematocrit was compared with the estimated increase with a paired t-test. Laboratory variables measured before and after transfusion were compared by paired t-tests. Associations between the duration, and etiology of anemia as well as the presence of regeneration and clinical variables before transfusion were assessed with the Kruskal–Wallis test. P values < .05 were considered statistically significant. All analyses were performed by a statistical software package.e

Results

Twenty-four dogs of various breeds were included, with a total of 30 transfusion cases; 2 dogs received 2

### Table 1. Anemic dog clinical assessment score (ADCAS).

| Variable                        | Normal (0)   | Mild (1)    | Moderate (2) | Severe (3)   |
|---------------------------------|--------------|-------------|--------------|--------------|
| Mucosa color                    | Salmon pink (Fig 1A) | Slightly pale (Fig 1B) | Moderately pale (Fig 1C) | Severely pale (Fig 1D) |
| Pulse quality                   | Normal       | Bounding    | Weak         | Weak         |
| Heart rate                      | 65–109 bpm   | 110–140 bpm | >140 bpm     | >140 bpm     |
| Respiratory rate                | 15–24 rpm    | 25–40 rpm   | >40 rpm      | >40 rpm      |
| Mentation/exercise tolerance    | BAR, walking | Quiet, able to walk | Lethargic, able to stand | Lethargic, unable to stand |

bpm, beats per minute; rpm, respiration per minute.
transfusions and 2 dogs received 3 (as several days elapsed between transfusions, these were treated as unrelated cases). There were 14 female neutered, 1 female entire, 7 male neutered, and 2 male entire dogs. Dogs were a median of 6 years of age (range 0.5–9 years) and weighed a median of 21 kg (range 6–50 kg). Ten dogs were blood type DEA-1.1 positive and 12 were DEA-1.1 negative; auto-agglutination interfered with blood type interpretation in 2 dogs. Cross-matching was performed in 6 dogs before transfusion. Twenty-one dogs (27/30 transfusions) had jugular venipunctures; the 3 dogs with immune-mediated thrombocytopenia (IMTP) had cephalic venipunctures.

Anemia duration was 5 days, 2–40 days (median, range); further categorization of dogs based on duration, type, and etiology of anemia is given in Table 2.

Transfusion duration was 5 hours (3–6 hours) with a volume of 320 mL (160–450 mL) equating to 14.8 mL/kg (7.1–41.7 mL/kg) of pRBC; mean age of pRBC units was 12.7 ± 6.7 days (mean ± SD). Transfusion was deemed by the attending clinician, independently of the ADCAS, lactate concentration, and oxygenation values, to be successful in 21 dogs (27/30 transfusions) based on improvement in clinical examination findings and increase in hematocrit. Dogs 6 (immune-mediated hemolytic anemia, IMHA), 13, and 18 (IMTP) were independently thought, by the attending clinician, to have unsuccessful transfusions based on insufficiently increased hematocrits and persistent tachycardia, tachypnea, and bounding pulses suggesting ongoing tissue hypoxia and requirement for further transfusion.

**Clinical Variables**

All variables in the ADCAS, including the total score, decreased significantly with transfusion ($P < .001$; Table 3). All cases had a total ADCAS of ≥5/12 before transfusion. After transfusion, all cases, except for those that remained transfusion-dependent (dogs 6, 13, and 18), had a total score ≤3/12.

Heart rate was 131 bpm before transfusion (100–196 bpm), 96 bpm after transfusion (66–148), with a decrease of 45 bpm (~88 to 0 bpm). It was >100 bpm in all dogs, and >120 bpm in 27/30 cases before transfusion and this decreased to ≤100 bpm in all except for dogs 6, 13, and 18. Respiratory rate was 48 rpm before transfusion (16–112 rpm), 30 rpm after transfusion (12–112 rpm), with a decrease of 22 rpm (~68 to 12 rpm). Heart and respiratory rates decreased significantly with transfusion ($P < .001$).

Dogs with regenerative IMHA had higher heart and respiratory rates before transfusion, $P < .001$ and

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**Table 2. Duration, type, and etiology of anemia for 30 transfusion cases.**

| | Regenerative Anemia | Nonregenerative Anemia | Total Dogs (Transfusions) |
|---|---|---|---|
| **Acute anemia** | | | |
| 2 GI hemorrhage | 2 IMHA | | 19 (23) |
| 3 IMTP | 1 GI hemorrhage | | |
| 11 IMHA | | | |
| **Chronic anemia** | | | |
| 1 GI hemorrhage | 2 myelodysplasia | 2 IMHA | 5 (7) |
| | | | |
| **Total dogs** | 16 (20) | 8 (10) | 24 (30) |

GI, gastrointestinal; IMHA, immune-mediated hemolytic anemia; IMTP, immune-mediated thrombocytopenia; IMHA*, immune-mediated hemolytic anemia affecting bone marrow precursors.
Table 3. Change in laboratory variable values after 30 transfusions. $P$ value is for the comparison between values before and after transfusion.

| Variable          | Before Transfusion   | After Transfusion   | $\Delta$ Transfusion | RI     | $P$ Value |
|-------------------|----------------------|---------------------|-----------------------|--------|-----------|
| $PvO_2$ (mmHg)    | 36 ± 8.1             | 35.3 ± 6.9          | −0.7 ± 11.5           | Unknown| .733      |
| SvO$_2$ (%)       | 68.3 ± 9.3           | 66.6 ± 8.5          | −1.7 ± 12.8           | Unknown| .471      |
| Hb (g/dL)         | 4.8 ± 1.2            | 8.4 ± 1.7           | 3.6 ± 2.1             | 12–18  | <.001     |
| Hematocrit (%)    | 14.6 ± 3.4           | 25.4 ± 5.1          | 10.8 ± 6.0            | 37–55  | <.001     |
| CvO$_2$ (mL/dL)   | 4.5 ± 1.2            | 7.7 ± 1.9           | 3.1 ± 2.3             | 14–14.5| <.001     |
| Lactate (mmol/L)  | 3.0 ± 1.9            | 2.4 ± 1.3           | −0.6 ± 1.1            | <2.5   | .001      |

Values denote mean ± SD.

$\Delta$ Transfusion, change with transfusion; RI, reference interval; $PvO_2$, venous partial pressure of oxygen; SvO$_2$, venous oxygen saturation of hemoglobin; Hb, hemoglobin; CvO$_2$, venous oxygen content.

$P = .004$, respectively. Dogs with acute onset and regenerative anemia had a higher pulse quality score ($P = .001$) and were more lethargic ($P = .02$) with a higher total ADCAS before transfusion ($P < .001$).

**Laboratory Variables**

Hemoglobin concentration, hematocrit, and CvO$_2$ increased significantly with transfusion ($P < .001$), and lactate concentration decreased significantly ($P = .006$), whereas $PvO_2$ and SvO$_2$ did not change significantly ($P = .733$ and $P = .471$, respectively) (Table 4). The increase in hematocrit was not significantly different from the estimated increase ($P = .056$). Hemoglobin concentration, hematocrit, and CvO$_2$ increased in all cases except for dog 6, where these variables decreased from 6.3 g/dL, 20%, and 6.88 mL/dL to 6.1 g/dL, 19%, and 4.63 mL/dL, respectively. Eighty-three percent (25/30) of transfusions were administered to dogs with a hematocrit $<17\%$, and CvO$_2$ $<5$ mL/dL before transfusion. Eighty percent of transfusion cases (24/30) had a hemoglobin concentration $<5.8$ g/dL before transfusion. After transfusion, CvO$_2$ remained $<5$ mL/dL in dogs 6 (4.6 mL/dL) and 13 (3.6 mL/dL), but not in dog 18 (5.5 mL/dL); these dogs were still deemed to be transfusion-dependent. Dog 11, which had acute-onset, regenerative IMHA, had a hemoglobin concentration and hematocrit of 5.2 g/dL and 16.5%, respectively, after transfusion, but was not considered clinically transfusion-dependent despite these levels. All other dogs had hemoglobin concentration $>5.8$ g/dL and hematocrit $>17\%$ after transfusion. Lactate concentration was abnormal in 16/30 (53%) cases before transfusion. Hyperlactatemia resolved in 4/16 cases after transfusion and improved, but remained high in 12/16 cases after transfusion. Results from cephalic samples were not higher than those acquired from jugular samples.

**Discussion**

The results of this study show that both clinical examination findings, assessed by the ADCAS, and laboratory variables (hemoglobin concentration, hematocrit, CvO$_2$, and lactate concentration) improve significantly after transfusion of pRBC to anemic dogs. Clinical and laboratory variables were recorded before transfusion and values of these variables were identified at a time that corresponded to a clinical decision to administer a transfusion, thereby fulfilling the study objectives.

The ADCAS was designed to assist with the identification of anemic dogs that would likely benefit from transfusion by allowing a more objective and standardized assessment of clinical examination findings. A mild abnormality for an assessed variable received a lower score compared with more severe abnormalities. The higher the total score achieved, the more likely a dog in this study was to require transfusion when compared with scores after transfusion. All scores improved significantly after transfusion. A “transfusion-need scale” was previously designed to guide transfusion decision using the clinical signs of anemia; however, it relies heavily on the PCV of the dog, which is an unreliable indicator of the need for transfusion. There are few studies in human and veterinary medicine, which have investigated using the easily recognizable clinical examination findings of anemic patients for making decisions regarding transfusion. This has led to what are described as “weak” recommendations, by the American Association of Blood Banks, based on this limited evidence, to consider clinical signs of anemia in addition to hemoglobin concentration assessment to guide transfusion...
therapy of anemic people. Clinical examination findings attributable to reduced oxygen carrying capacity of a population of anemic dogs before transfusion are reported in this study. By correlating a change in these variables with a clinical interpretation of whether transfusion had been deemed successful or otherwise, the usefulness of assessing these variables in combination with laboratory variables when deciding to transfuse dogs has been demonstrated.

All ADCAS variables improved significantly after transfusion. Abnormalities in individual variables could nonspecifically indicate a variety of conditions, but considered together, they are the most commonly assessed clinical variables in anemic dogs and people when deciding whether a transfusion could be beneficial. Classification of tachycardia and tachypnea based on severity is rarely described and so differs from the other variables. Subjectivity was minimized as mucosal color was compared with photographs. Mentation and exercise tolerance were assessed together as these were likely to be equally affected in anemic dogs. Pulse quality was the most subjective variable and therefore the same clinician performed all clinical examinations, thus minimizing subjectivity.

Most dogs were initially tachycardic and tachypneic, and these variables decreased significantly with transfusion. Despite the wide body weight variation in the included dogs, resting heart and respiratory rates in normal dogs have been determined to be unrelated to bodyweight. Classification of tachycardia and tachypnea based on severity is rarely described and so ranges were arbitrarily chosen.

The total score decreased significantly in all cases. It remained high in 3 dogs after transfusion and within the range of scores seen for cases before transfusion. These dogs were deemed to have continued signs of severe anemia (tachycardia, tachypnea, bounding pulses), suggesting further need for transfusion. Although each individual ADCAS variable improved significantly after transfusion, the scoring system provides a stronger guideline for transfusion decision when assessed as a whole, as there are likely less differential diagnoses possible when all 5 variables are abnormal.

Dogs with acute, regenerative anemia were more lethargic, tachycardic, and tachypneic, with more severe pulse quality abnormalities before transfusion compared with dogs with chronic, nonregenerative anemia; mentation/exercise tolerance was similar between groups. Hematocrits before transfusion were not markedly different between regenerative and nonregenerative anemia cases. These findings reflect the understanding that dogs with acute anemia require days to develop systemic compensatory mechanisms to anemia, which become exhausted quicker than in cases of chronic anemia, and that clinical signs of anemia are more apparent in dogs with acute anemia than those with chronic anemia at a similar hematocrit. Despite the variation in clinical examination findings before transfusion, the response to transfusion was comparable in all dogs; underlying disease was therefore an unlikely explanation of the difference in ADCAS variables between cases of acute and chronic anemia before transfusion.

Hemoglobin concentration, hematocrit, and subsequently CvO₂ increased significantly after pRBC transfusion in this study. Hemoglobin concentrations at which dogs in this study were deemed to require transfusion, based on assessment by the attending clinician, were recorded. The difference between the hematocrits of cases of acute, regenerative and chronic, nonregenerative anemia before transfusion was minimal (data not shown). Hemoglobin is the major determinant of both blood oxygen content and DO₂. Various studies have attempted to identify the hemoglobin concentration or hematocrit threshold level, suggesting that transfusions should be administered at the latest when hemoglobin concentration is <5–8 g/dL in people and hematocrit <10% in dogs. Hemoglobin was <5.8 g/dL in 80% of the included cases before transfusion and although not a confirmed threshold level, this concentration could assist with transfusion decision in dogs in combination with ADCAS findings.

CvO₂ increased significantly after transfusion. There was no clear difference between values for cases of acute compared with chronic anemia before and after transfusion (data not shown). This variable was selected as a potential surrogate marker of DO₂ because of ease of sample collection and measurement. An increase in CvO₂ after transfusion suggests concurrent DO₂ improvement, which itself is considered a surrogate marker for tissue oxygenation. Mathe-
after transfusion. Lactate concentration before transfusion of dogs with hemolysis appeared to be higher than that seen in cases of hemorrhage or chronic anemia (data not shown). As a surrogate marker of tissue oxygenation, lactate allows approximation of severity of tissue hypoperfusion and hypoxia, and monitoring serial measurements over time enables assessment of treatment response.\textsuperscript{14} Lactate could guide transfusion therapy.\textsuperscript{15} Lactate concentration did not reliably differentiate between cases that would benefit from further blood transfusion in this study as only 10% of transfusion cases (25% of those with ongoing hyperlactatemia) were deemed to have continuing clinical signs of anemia (tachycardia, tachypnea, bounding pulses). The hyperlactatemia after transfusion was unlikely because of hypoperfusion, as hypovolemic dogs were excluded. It could have resulted from persisting tissue hypoxia caused by anemia, which might only have resolved hours after transfusion or it was possibly a result of microcirculatory perfusion derangements related to underlying disease.\textsuperscript{25,26} Lactate concentrations do not vary significantly between arterial and venous samples, but have been shown to be lower in jugular compared with cephalic venous samples,\textsuperscript{27} a finding that was not mirrored in this study.

There were several limitations to this study. Only 30 transfusion cases were included during the study period. Four dogs had multiple transfusions in this study; however, as the clinical examination and laboratory variables were assessed immediately before and after transfusion, previous transfusions were unlikely to have a marked impact on the results of these. Type II statistical error could have influenced the lack of significance of \( \text{PvO}_2 \) and \( \text{SvO}_2 \) changes after transfusion. Despite low case numbers, there were statistically significant changes in hemoglobin concentration, hematocrit, \( \text{CvO}_2 \), lactate concentration, and the ADCAS variables. The ADCAS contained some subjective variables, including pulse quality and mucus membrane color in particular. Subjectivity was minimized as the same clinician performed all clinical examinations, but bias could have been introduced as the clinician was not blinded to the transfusion status of dogs. Future studies are needed to validate the ADCAS to statistically prove its reproducibility and reliability; the same score should be achieved in a particular dog (taking exclusion criteria into consideration), whatever the experience of the veterinary professional assessing it. Anemia cases of different etiologies were not specifically excluded, so as to reflect the variety of cases seen in this hospital. There were, however, extensive exclusion criteria applied to minimize confounding factors, which restricted the population of dogs in the study and could limit the usefulness of these variables for cases of anemia from other causes. Consideration should be given to these criteria if applying the results from this study to other anemic dogs. The dogs in this study were presented to a referral institution, which could have introduced bias into case selection. As many veterinary practices do not store blood transfusion products, cases of severe anemia are often referred. Cases with less severe anemia would not likely require transfusion and therefore, it is likely that the dogs in this study reflect the general population of anemic dogs that would likely require transfusion. The authors were the primary clinicians of <17% of the cases included, which minimized any bias toward deciding to initiate transfusion. All other cases were independently managed by other clinicians.

In summary, this study describes the clinical examination and laboratory parameter changes that occur with transfusion of \( \text{pRBC} \) to anemic dogs. The ADCAS allowed clinical examination findings to be more objectively included in the decision-making process; the higher the score, the more likely a blood transfusion would be clinically beneficial. Concentrations of hemoglobin, hematocrit, and \( \text{CvO}_2 \) below which, most included dogs were given a transfusion, were reported. These variables could be used to provide objective criteria of transfusion dependency in dogs. A single universal transfusion trigger does not exist, but the variables described in this study could be extrapolated to other anemic dogs and collectively used to guide the decision to transfuse alongside clinician judgment, providing that the exclusion criteria are taken into consideration.

### Footnotes

\begin{itemize}
\item \textsuperscript{a} DMS Laboratories, Flemington, NJ
\item \textsuperscript{b} Canine \( \text{pRBC} \), Pet Blood Bank, Loughborough, UK
\item \textsuperscript{c} Roche Diagnostics, West Sussex, UK
\item \textsuperscript{d} Pre-heparinized syringe sampler, Sanguis Counting, Numbrecht, Germany
\item \textsuperscript{e} Minitab 16, Coventry, UK
\end{itemize}

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**Conflict of Interest Declaration:** Authors disclose no conflict of interest.

### References

1. Callan MB, Oakley DA, Shofer FS, Giger U. Canine red blood cell transfusion practice. J Am Anim Hosp Assoc 1996; 32:303–311.
2. Howard A, Callan B, Sweeney M, Giger U. Transfusion practices and costs in dogs. J Am Vet Med Assoc 1992;201:1697–1701.
3. Prittie JE. Triggers for use, optimal dosing, and problems associated with red cell transfusions. Vet Clin North Am Small Anim Pract 2003;33:1261–1275.
4. Wardrop KJ, Reine N, Birkenheuer A, et al. Canine and feline blood donor screening for infectious disease. J Vet Intern Med 2005;19:135–142.
5. Carson JL, Noveck H, Berlin JA, Gould SA. Mortality and morbidity in patients with very low postoperative Hb levels who decline blood transfusion. Transfusion 2002;42:812–818.

6. Carson JL, Terrin ML, Noveck H, et al. Liberal or restrictive transfusion in high-risk patients after hip surgery. N Engl J Med 2011;365:2453–2462.

7. Hebert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. Transfusion Requirements in Critical Care Investigators, Canadian Critical Care Trials Group. N Engl J Med 1999;340:409–417.

8. Viele MK, Weiskopf RB. What can we learn about the need for transfusion from patients who refuse blood? The experience with Jehovah’s Witnesses. Transfusion 1994;34:396–401.

9. Weiskopf RB, Viele MK, Feiner J, et al. Human cardiovascular and metabolic response to acute, severe isovolemic anemia. J Am Med Assoc 1981;241:864–871.

10. Schwartz S, Frantz RA, Shoemaker C. Sequential hemodynamic and oxygen transport responses in hypovolemia, anemia, and hypoxia. Am J Physiol Heart Circ Physiol 1981;241:864–871.

11. Carson JL, Grossman BJ, Kleinman S, et al. Red blood cell transfusion: A clinical practice guideline from the AABB. Ann Intern Med 2012;157:49–58.

12. Kerl ME, Hohenhaus AE. Packed red blood cell transfusion in dogs: 131 cases (1989). J Am Vet Med Assoc 1993;202:1495–1499.

13. Saugel B, Klein M, Hapfelmeier A, et al. Effects of red blood cell transfusion on hemodynamics in patients: A prospective study in intensive care unit patients. Scand J Trauma Resusc Emerg Med 2013;21:1–7.

14. Holahan ML, Brown AJ, Drobatz KJ. The association of blood lactate concentration with outcome in dogs with idiopathic immune-mediated hemolytic anemia: 173 cases (2003-2006). J Vet Emerg Crit Care 2010;20:413–420.

15. Cassut M, Seifert B, Pash T, et al. Factors influencing the individual effects of blood transfusions on oxygen delivery and oxygen consumption. Crit Care Med 1999;27:2194–2200.