Effect of packed red blood cell transfusion on thromboelastographic tracings in dogs with naturally occurring anemia

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OBJECTIVE
To assess the effect of packed RBC (pRBC) transfusion on thromboelastographic (TEG) tracings in dogs with naturally occurring anemia.

ANIMALS
22 clinically anemic dogs that received a pRBC transfusion.

PROCEDURES
For each dog, a blood sample was collected before and within 3 hours after completion of the pRBC transfusion for a CBC, nonactivated TEG analysis, and measurement of blood viscosity. Wilcoxon signed rank tests were used to compare CBC, viscosity, and TEG variables between pretransfusion and posttransfusion blood samples. Multivariable linear regression was used to assess the effects of pretransfusion-posttransfusion changes in Hct, WBC count, and platelet count on changes in TEG variables.

RESULTS
Median posttransfusion Hct (21%; range, 13% to 34%) was significantly greater than the median pretransfusion Hct (12.5%; range, 7% to 29%). Packed RBC transfusion was associated with a median increase in Hct of 6.2% (range, 1.2% to 13%). Maximum amplitude significantly decreased from 74.9 to 73.8 mm and clot strength significantly decreased from 14,906 to 14,119 dynes/s after pRBC transfusion. Blood viscosity significantly increased, whereas platelet and WBC counts significantly decreased after transfusion. Multivariable linear regression revealed that pretransfusion-posttransfusion changes in Hct, WBC count, and platelet count were not associated with changes in TEG variables.

CONCLUSIONS AND CLINICAL RELEVANCE
Results indicated that pRBC transfusion had only small effects on the TEG tracings of hemodynamically stable dogs. Therefore, large changes in TEG tracings following pRBC transfusion are unlikely to be the result of the transfusion and should be investigated further. (*Am J Vet Res* 2020;81:499–505)

Viscoelastic methods of coagulation are used for the assessment of hemostasis in blood samples. Mechanisms of action for VEMs and interpretation of VEM tracings have been extensively reviewed elsewhere.[1,2] In both human and veterinary medicine, there has been a recent resurgence of interest in VEMs for diagnostic assessment of hemostasis in patients with hemorrhagic and thrombotic disorders.[1–4] However, as with any methodology, appropriate interpretation of VEM assay results (VEM results) is dependent on understanding the limitations and biases of the assay. It is well established in both in vitro and in vivo applications that there is an inverse relationship between RBC mass and VEM results, whereby anemic blood samples appear hypercoagulable and polycythemic samples appear hypocoagulable relative to blood samples with Hcts within reference limits.[5–9] This is despite the clinical observation that anemia often prolongs bleeding time and polycythemia makes patients prone to thrombus formation. Thus, the apparently incongruous relationship between RBC mass and VEM results may be secondary to local conditions intrinsic to the technology associated with clot formation by VEMs.[5,10,11]

ABBREVIATIONS

G	Clot strength as measured by thromboelastography
IMHA	Immune-mediated hemolytic anemia
K	Clot kinetics as measured by thromboelastography
MA	Maximum amplitude as measured by thromboelastography
pRBCs	Packed RBCs
PROVETS	Partnership on Rotational ViscoElastic Test Standardization
R	Reaction time as measured by thromboelastography
RoTEM	Rotational thromboelastometry
TEG	Thromboelastography or thromboelastrographic
VEM	Viscoelastic method
VMC	Veterinary Medical Center

Owing to the inconsistency between VEM results and clinical observations, particularly for anemic and polycythemic patients, there is concern that serial use of VEM assays to monitor patients with a rapidly changing RBC mass could lead to misinterpretations regarding the hemostatic status of the patient because the changes observed in the VEM results might be associated with the specific technology used to generate those results. For example, many anemic dogs receive blood transfusions, and the subsequent changes in Hct for those dogs might make interpretation of serial VEM results challenging. To our knowledge, the effect of a changing Hct subsequent to pRBC transfusion in dogs with naturally occurring anemia on the interpretation of serial VEM results has not been investigated. The objective of the study reported here was to assess the effect of pRBC transfusion on TEG tracings in dogs with naturally occurring anemia. We hypothesized that, because of the increase in Hct after pRBC transfusion, TEG tracings obtained after the transfusion would appear less hypercoagulable than TEG tracings obtained before transfusion despite minimal change in the underlying disease status of the patient.

Materials and Methods

Animals

All study procedures were reviewed and approved by The Ohio State University Institutional Animal Care and Use Committee, Veterinary Medical Clinical Trials Office, and VMC Clinical Research Advisory Committee. Dogs examined at the university VMC that had naturally occurring anemia and required a pRBC transfusion were considered for study enrollment. Dogs were excluded from the study if they had received any type of blood product within 30 days of evaluation at the VMC, the pRBC transfusion was initiated before a blood sample was obtained for TEG analysis, the pRBC transfusion was administered rapidly (< 2 hours) owing to the hemodynamic instability of the patient, other interventions to address hemodynamic instability (eg, surgery, massive transfusion, volume fluid resuscitation, or administration of other types of blood products) were performed at any time during the pRBC transfusion, or the attending clinician thought that study enrollment was contrary to the clinical interest of the patient. Dogs that weighed < 4 kg were also excluded from study enrollment. Dogs that were receiving medications that might affect hemostasis (eg, corticosteroids or clopidogrel) for the underlying disease process were not excluded from the study; however, per the VMC transfusion protocol, no medications were administered during the pRBC transfusion or between collection of pretransfusion and posttransfusion blood samples. Informed owner consent was obtained for each dog prior to its enrollment in the study.

Sample collection and processing

For each dog, the signalment, body weight, underlying disease process, volume of nonleukoreduced pRBCs received, pretransfusion, and posttransfusion CBC results were recorded. The age of the transfused pRBCs was not recorded, but owing to the dynamic nature of the VMC blood bank, most pRBCs were used within 14 days after collection. A blood sample (approx 6 to 10 mL) was obtained 1 hour before initiation (pretransfusion sample) and between 1 and 3 hours after completion (posttransfusion sample) of the pRBC transfusion. The posttransfusion sample was collected at the same time when blood was collected to measure posttransfusion PCV and total protein concentration to minimize phlebotomy events. Per the VMC intensive care unit transfusion protocol, the pRBC transfusion was generally administered over a period of 4 hours, with the transfusion rate gradually increasing over the first hour and frequent monitoring of the patient's temperature, heart rate, and respiratory rate. Because the study subjects were clinical patients, the site and technique used to collect blood samples were not always consistent between pretransfusion and posttransfusion samples. When blood was obtained from an IV catheter or central line, care was taken to acquire the blood sample immediately after initial catheter placement. When blood was collected via an IV line, care was taken to flush the line with nonheparinized saline (0.9% NaCl) solution and aspirate an appropriate waste sample of blood (> 3 mL) before the study sample was collected.[12] Blood samples collected by direct venipuncture were obtained by use of a butterfly catheter or needle (18 to 22 gauge) that was attached to a syringe.[a] All venipunctures were performed by a trained intensive care unit technician or the primary investigator (ACB). Immediately after sample collection, 1.8 mL of blood was added to a sterile blood collection tube[b] that contained 0.2 mL of sodium citrate as an anticoagulant (3.2% citrated tube), thus resulting in a sample with a 1:9 citrate-to-blood ratio. The time when the blood sample was collected was recorded on the tube.

Prior to TEG analysis, each blood sample was analyzed with a hematologic analyzer[c] to determine the Hct, total and differential WBC counts, and platelet count. Because samples were stored in 3.2% citrated tubes, a dilution correction factor of 1.1 was applied to all parameters reported on a per unit volume basis.[13]

TEG

All blood samples were analyzed on channel 1 of a TEG unit.[d] Blood samples were stored at room temperature (approx 22°C) for 30 minutes immediately after collection and prior to TEG analysis. For each sample, 340 µL of citrated blood was mixed with 20 µL of 0.2M calcium chloride in a TEG cup that was prewarmed to 37°C, yielding a final sample volume of 360 µL. The TEG analysis was started immediately and allowed to run until it stopped. No activator was

used because at the time the samples were run, the TEG reference range established by the VMC Clinical Pathology Laboratory was determined on the basis of results for blood samples from healthy dogs that were analyzed without an activator. The TEG values for R, K, angle, MA, and G were recorded. Clot strength was calculated by the TEG unit software by use of the following equation: G = ([5,000 × MA]/[100 – MA])/1,000.

For the study reported here, hypercoagulable and hypocoagulable TEG tracings were defined as tracings for which the G was above and below, respectively, the reference range established by the VMC Clinical Pathology Laboratory.[9,14,15] All TEG values were reported and assessed independently as recommended.[16]

Viscosity measurement

Immediately after the TEG analysis was begun, the viscosity of each blood sample was measured with a cone and plate viscometer.[e] The cup was prewarmed to 37°C via a circulating warm water bath.[f] A 0.5-mL aliquot of blood was placed in the center of the cup, which was then immediately placed on the viscometer to begin cone rotation. Viscosity was measured after 10 seconds of equilibration at 20 rpm (ie, shear rate of 150 seconds^{-1}). This time period was chosen because the viscosity of blood will decrease over time owing to the sedimentation of RBCs.[17] The viscosity of each blood sample was determined in triplicate, and the mean was calculated and recorded for that sample.

Statistical analysis

The data distribution for each continuous variable was assessed for normality by means of the Shapiro-Wilk test. Because many TEG variables were not normally distributed, results were summarized as the median and range. Pretransfusion and posttransfusion sample results were compared by means of the Wilcoxon signed rank test.

Multivariable linear regression without Bonferroni correction was used to assess the effect of changes in Hct, WBC count, and platelet count on changes in individual TEG variables. For each dog, the change (Δ) in each TEG variable as well as the changes in Hct, WBC count, and platelet count were calculated as the difference between pretransfusion and posttransfusion values. For each respective linear regression model, the change in the TEG variable was the dependent variable and the changes in Hct, WBC count, and platelet count were independent variables. All analyses were performed with a commercial statistical software package.[g] Values of $P \leq 0.05$ were considered significant.

Results

Dogs

Twenty-two dogs were enrolled in the study, including 10 spayed females, 1 sexually intact female, 10 castrated males, and 1 sexually intact male. The study population included 5 mixed-breed dogs, 3 Labrador Retrievers, 1 pit bull–type dog, and 1 each of the following breeds: American Bulldog, Basset Hound, Cocker Spaniel, Doberman Pinscher, Giant Schnauzer, Golden Retriever, Greyhound, Miniature Schnauzer, Pekingese, Pembroke Welsh Corgi, Pomeranian, Staffordshire Terrier, and Wirehaired Fox Terrier.

Anemia was caused by hemorrhage for 10 dogs, destruction of RBCs for 6 dogs, and inadequate production of RBCs (ie, nonregenerative anemia) for 6 dogs. For the 10 dogs with hemorrhage-induced anemia, the hemorrhage was the result of gastrointestinal bleeding not associated with immune-mediated thrombocytopenia (n = 3), bleeding prior to surgery for removal of a left adrenal gland tumor (1), postoperative bleeding following liver lobectomy for removal of a hepatocellular carcinoma (1) and splenectomy for removal of a splenic hematoma (1), severe facial trauma subsequent to being hit by a car (1), and hemangiosarcoma-induced bleeding into the lungs (1), thigh (1), and urinary tract with concomitant lymphosarcoma (1). The 6 dogs that were anemic owing to RBC destruction had IMHA. Nonregenerative anemia was the result of pure RBC aplasia for 3 dogs, B-cell lymphoma for 1 dog, Waldenstrom macroglobulinemia for 1 dog, and undetermined (potential rule outs included chronic kidney disease, pure RBC aplasia, and phenobarbital toxicosis) for 1 dog.

Hematologic and TEG findings

Results for TEG variables before and after pRBC transfusion were summarized **(Table 1)**. The median Hct for the study population was 12.5% (range, 6.7% to 28.7%) before and 20.7% (range, 13.2% to 33.9%) after pRBC transfusion. The median dose of pRBCs transfused to each dog was 11.7 mL/kg (range, 3.9 to 14.2 mL/kg), and the median increase in Hct was 6.2% (range, 1.2% to 13%) following pRBC transfusion. The median duration between collection of pretransfusion and posttransfusion blood samples was 5.62 hours (range, 3.25 to 8.17 hours). The median Hct ($P < 0.001$) and blood viscosity ($P < 0.001$) increased, whereas the median platelet count ($P < 0.008$) and WBC count ($P < 0.008$) decreased following transfusion. The median R ($P = 0.70$), K ($P = 0.20$), and angle ($P = 0.07$) did not differ significantly between pretransfusion and posttransfusion samples. The median MA ($P = 0.05$) and G ($P = 0.007$) for posttransfusion samples were significantly decreased from the corresponding values for pretransfusion samples. The median difference between pretransfusion MA and posttransfusion MA (ΔMA) was –1.7 mm (range, –6.9 to 3.8 mm), and the median difference between pretransfusion G and posttransfusion G (ΔG) was –1,271.9 dynes/s (range, –14,546.8 to 1,871.0 dynes/s; **Figure 1**). Eighteen of the 22 (82%) dogs had a decrease in MA following pRBC transfusion; however, 4 (18%) dogs had a small (clinically negligible) increase in MA following transfusion. All 4 of those dogs had a neoplastic process.

Results of multivariable linear regression analysis revealed that the ΔHct ($P = 0.95$), Δplatelet count ($P =$

Table 1—Median (range) for select hematologic and TEG variables for 22 dogs with naturally occurring anemia before and after pRBC transfusion.

Variable	VMC reference range	Before transfusion	After transfusion
Hct (%)	45–54	12.5 (6.7–28.7)	20.7 (13.2–33.9)*
Platelet count ($\times 10^9$ platelets/L)	108–433	176 (19–767)	150 (19–469)*
WBC count ($\times 10^9$ WBCs/L)	4.1–15.2	21.1 (4.9–76.5)	20.5 (4.4–61.3)*
Viscosity (cP)	3.74–4.94	2.03 (1.5–4)	2.66 (1.8–4.4)*
R (min)	1.0–6.1	3.15 (1.7–8.9)	3.55 (1–7.8)
K (min)	0.9–3.6	0.9 (0.8–18.6)	1.0 (0.8–11.1)
α Angle (°)	51.8–73.4	77.1 (20.1–85)	75 (25.8–82)
MA (mm)	43.9–67.9	74.9 (20.8–91)	73.8 (24.6–89.3)*
G (dynes/s)	3,104–10,020	14,906 (1,316–50,652)	14,119 (1,635–41,852)*

For comparison purposes with the reference ranges for the nonactivated TEG variables assessed in this study, the VMC-determined reference ranges for kaolin-activated TEG variables were as follows: R, 1.3 to 4.2 minutes; K, 1.0 to 3.6 minutes; α angle, 9.8° to 76.2°; MA, 44.0 to 64.9 mm; and G, 3,900 to 11,300 dynes/s.

*Value differs significantly ($P \leq 0.05$) from the corresponding value before transfusion.

cP = Centipoise.

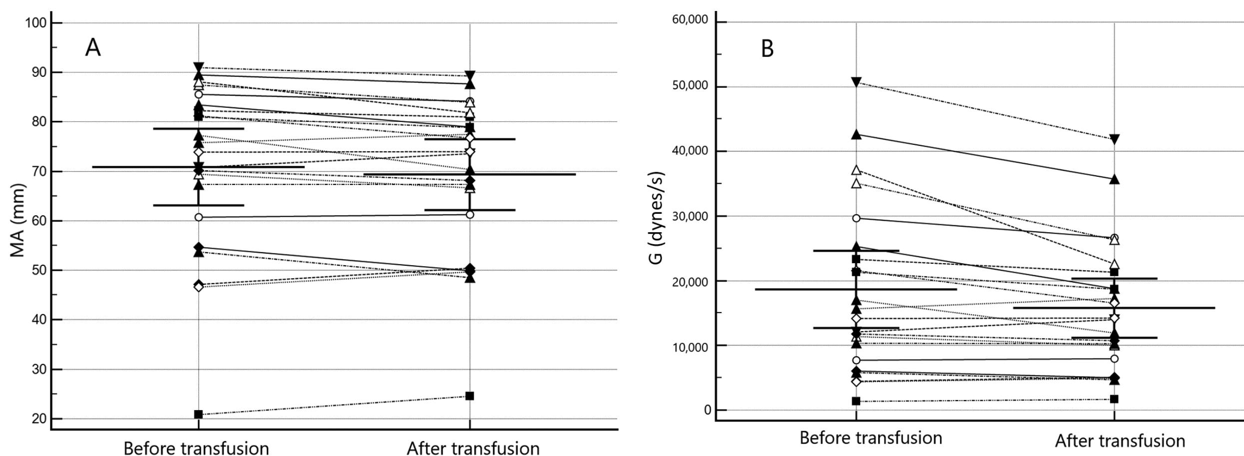

Figure 1—Scatterplots of the MA (A) and G (B) determined by nonactivated TEG analysis for 22 dogs with naturally occurring anemia before and after pRBC transfusion. Within each panel, matching symbols represent the pretransfusion and posttransfusion results for 1 dog, and the line connecting the matching symbols indicates the direction of change in that variable. Within each period (before and after transfusion), the long horizontal line represents the median and the short horizontal lines delimit the interquartile (25th to 75th percentile) range.

0.17), and ΔWBC count ($P = 0.91$) had no effect on the ΔMA. Likewise, the ΔHct ($P = 0.93$), Δplatelet count ($P = 0.16$), and ΔWBC count ($P = 0.68$) had no effect on the ΔG.

Prior to pRBC transfusion, 16 dogs were classified as hypercoagulable, 5 dogs were classified as normocoagulable, and 1 dog was classified as hypocoagulable. The coagulability category did not change following transfusion for any dog. Five of the 6 dogs that were classified as normocoagulable or hypocoagulable were thrombocytopenic, with a median platelet count of 44,000 platelets/µL (range, 19,000 to 90,000 platelets/µL) before transfusion and 26,000 platelets/µL (range, 19,000 to 79,000 platelets/µL) after transfusion.

Discussion

For the anemic dogs of the present study, there appeared to be an inverse relationship between Hct and both MA and G. The median Hct increased, whereas the median MA and G decreased significantly following pRBC transfusion. Given that G is a mathematical derivation of MA, it was expected that the nature of the respective relationships between MA and other hematologic variables would be mirrored for G. None of the other TEG variables assessed in this study (R, K, and angle) differed significantly between pretransfusion and posttransfusion blood samples.

The inverse relationship between Hct and MA (and G) observed for the dogs with naturally occurring anemia of the present study was consistent with in vitro and in vivo findings reported for canine,[5,8] equine,[6] and human[18,19] blood samples. However, in the present study, unlike those other studies,[5,6,8,18,19] we did not find evidence of a decrease in hypercoagulability on the basis of increasing Hct when other TEG variables were evaluated. In a study[8] involving blood samples obtained from healthy dogs, correlation was

strongest between Hct and the RoTEM variables for clot formation time, α angle, and maximum clot firmness, which are analogous to the TEG variables K, angle, and MA, respectively. For the present study, the small and heterogenous population of anemic dogs and the use of a different method (nonactivated TEG) for assessment of coagulation variables likely limited our ability to detect subtle changes in K and angle between pretransfusion and posttransfusion samples.

The inverse relationship between Hct and VEM variables has been well documented.[5–9,18–22] Results of the present study suggested that changes in Hct and viscosity have minimal, albeit significant, effects on TEG tracings. Three mechanisms have been proposed for those effects. An increase in RBC mass may truly impair coagulation, effectively decrease the plasma volume (and hence decrease the concentrations of fibrinogen and other coagulation factors carried in plasma), or increase blood viscosity, which affects the mechanics of VEM assays.[5,20,21,h] In another study[9] involving canine blood samples, blood factors that had the greatest effects on G included platelet count, platelet function, Hct, and fibrinogen, antithrombin, and C-reactive protein concentrations. Results of studies involving blood samples obtained from dogs with experimentally induced anemia[5] and horses with experimentally induced polycythemia[6] indicate that other biomarkers associated with coagulation, such as thrombin-antithrombin complex concentration, remain within reference limits despite changes in the TEG tracings induced by alterations in RBC mass. Thus, it seems likely that the inverse relationship between Hct and VEM variables is the result of the VEM itself, either secondary to Hct-induced changes in concentrations of plasma components or changes in blood viscosity. Red blood cell mass is the main determinant of blood viscosity, and results of another study[21] indicate that blood viscosity affects TEG variables independent of Hct. In the present study, although the viscosity increased between pretransfusion and posttransfusion blood samples, we could not assess the effects of Hct and viscosity on TEG tracings separately because those 2 properties are intrinsically linked.

Results of the present study indicated that both the WBC and platelet counts decreased between pretransfusion and posttransfusion blood samples. A possible explanation for that finding was a dilution effect secondary to pRBC transfusion, which is a WBC- and platelet-poor product. A similar effect was observed in human patients, whereby the platelet count progressively decreased for blood samples obtained before and 1 and 24 hours after transfusion of pRBCs.[22] The decrease in WBC count observed between the pretransfusion and posttransfusion blood samples of the present study was unexpected because transfusion of blood products is usually associated with an increase in WBC count.[23–25] The observed decrease in WBC count was most likely a function of the duration between pRBC transfusion and posttransfusion sample collection (ie, within 1 to 3 hours after transfusion completion) because the blood transfusion–induced increase in WBC count typically peaks 12 hours after transfusion completion.[23–25] Information regarding the effects of WBC count on VEM variables is sparse. Results of 1 study[26] in which RoTEM was used to assess blood samples suggest that polymorphonuclear WBCs have no effect on clot strength but do shorten clot formation time. Consequently, we were not surprised by the fact that results of the multivariable linear regression analysis in the present study suggested that the change in MA was not significantly affected by the change in WBC count. A decrease in platelet count is associated with a decrease in clot formation time, angle, and maximum clot firmness for blood samples obtained from healthy dogs that were analyzed by RoTEM.[8] Another study[27] indicates there is a linear relationship between decreasing platelet count and decreasing MA in human blood samples when the platelet count is manipulated in vitro. In the present study, the changes in WBC and platelet counts between pretransfusion and posttransfusion samples were not significantly associated with the change in MA or G, which suggested that the dilutional decreases in those variables were not responsible for the small changes observed in MA or there was a type 2 error present owing to the small and heterogenous population of anemic dogs evaluated.

Although the median MA (and consequently G) for posttransfusion samples was significantly lower than that for pretransfusion samples in the present study, we believe that the fairly small decrease in MA observed between pretransfusion and posttransfusion samples would have minimal effects on clinical decisions for individual patients. For example, in the present study, the maximum decrease in MA was 7 mm, which was observed for a dog in which the Hct increased by 12% between the pretransfusion and posttransfusion samples. For dogs with IMHA of another study,[28] the odds of survival for 30 days increased by 13% (OR, 1.13; 95% confidence interval, 1.02 to 1.25) for each 1-mm increase in MA at hospital admission. On the basis of the results of the present study and given that dogs with IMHA are likely to be treated with an RBC transfusion, small changes in MA that are likely the result of changes in Hct rather than the underlying disease status of a patient should be interpreted with caution, particularly if TEG variables are being used as prognostic indicators. More importantly, it is unlikely that an increase in Hct, particularly within the range observed in the present study, would cause large changes in the MA of TEG tracings.

Limitations of the present study included the heterogeneous nature of the study dogs and lack of standardization for the technique used to acquire blood samples. Additionally, although no medications were administered during the pRBC transfusion, some dogs had received medications that can alter TEG tracings, such as corticosteroids,[29] prior to study enrollment. However, because the focus of the present study was

to compare variables before and shortly after transfusion, those medications should have had little effect on the TEG tracings over that fairly short duration. Further, dogs treated with clopidogrel were not excluded from this study because there is evidence that clopidogrel does not alter TEG tracings for blood samples obtained from dogs[30] and humans.[31] Also, in an effort to minimize the inherent variation in the study population, we used paired statistical methods to compare results between pretransfusion and posttransfusion blood samples within the same subject. Analyses were not performed for subgroups of dogs with certain types of anemia (eg, dogs with IMHA or nonregenerative anemia) owing to the small number of dogs within each group.

Another limitation of the present study was the fact that the fibrinogen concentration was not measured for any of the blood samples. Measurement of fibrinogen concentration in blood samples undergoing TEG is recommended by PROVETS guidelines.[16] Fibrinogen significantly affects G, and it is possible that the inverse relationship between Hct and G observed in this study might have been affected by fibrinogen concentration.[9,h] However, results from a study[32] of biomarkers for inflammation and coagulation status following pRBC transfusion in critically ill human patients indicate that fibrinogen concentration is not significantly affected in the short-term after transfusion. Given that all posttransfusion blood samples analyzed in the present study were obtained within 3 hours after completion of the pRBC transfusion, we do not believe measurement of fibrinogen concentration would have significantly affected our results.

Changes in TEG variables associated with pRBC transfusion–induced inflammation versus an increase in Hct have yet to be defined. In mice, transfusion of pRBCs initiates an inflammatory cytokine response.[33] Results of studies into inflammatory cytokine release following pRBC transfusion in dogs[23,24] and humans[32,34] are equivocal, with some of those studies not documenting cytokine release until > 3 hours after completion of the transfusion (ie, outside the time frame during which the posttransfusion blood samples were collected in this study). Transfusion of pRBCs may also affect other factors associated with coagulation, such as thrombin-antithrombin complex, or the generation of thrombin, although it does not appear to do so in human patients.[35] However, results of in vitro studies[5,6] suggest that the effects of those changes on TEG tracings are likely to be minimal.

The present study was conducted prior to the release of current PROVETS guidelines,[36] and nonactivated TEG analysis was performed in this study because that was the only type of TEG analysis for which our clinical pathology laboratory had established reference ranges at that time. The PROVETS guidelines[36] recommend that activated TEG methods be used because activators decrease variation in TEG tracings. Sodium citrate was used as the anticoagulant for the blood samples analyzed in the present study, and the use of citrate-anticoagulated blood in the nonactivated TEG analysis may have limited our ability to detect transfusion-induced changes in all TEG variables other than MA.[36] However, we believe that was unlikely because the reference ranges for nonactivated and kaolin-activated TEG analyses currently established by our clinical pathology laboratory are similar with the exception of the reference range for R, which is expected given the mechanistic differences between the 2 methods.

Finally, we can only comment on the effect of Hct on TEG variables within the Hct range (6% to 31%) evaluated in the present study and the volume of pRBCs transfused (approx 11 mL/kg). It is possible that the respective relationships between Hct and TEG variables may differ from those observed in this study for dogs that receive a larger volume of pRBCs or for which higher Hcts are achieved. However, given that the Hct for most dogs with anemia that receive a pRBC transfusion will fall within the Hct range observed for the dogs of this study, we believe our findings are clinically relevant.

Results of the present study indicated that, for dogs with naturally occurring anemia, transfusion of pRBCs resulted in an increase in Hct, which appeared to cause the MA and G determined by nonactivated TEG analysis to decrease. The pRBC transfusion–induced increase in Hct did not appear to have a significant effect on any of the other TEG variables assessed. However, the magnitudes of the respective inverse relationships between Hct and MA and G were small, and it remains unknown how those relationships might affect clinical decisions. Nevertheless, the overall effect of pRBC transfusion on TEG tracings of dogs appeared to be fairly small. Therefore, for individual patients, large changes in TEG tracings likely reflect a true change in the coagulation status of that patient. Caution should be used when TEG variables are used for prognostication in dogs that receive blood transfusions. Although VEMs are useful for monitoring the global hemostasis status of a patient, it is important to be cognizant of the strengths and limitations of those methods when interpreting test results.

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Footnotes

a. Monoject, Covidien, Mansfield, Mass.
b. Becton, Dickinson and Co, Franklin Lakes, NJ.
c. Procyte Dx Hematology Analyzer, Idexx Laboratories, Westbrook, Me.

d. TEG Haemoscope Corp, Niles, Ill.
e. DV-II+ Pro Viscometer with CPE-40 cone (0.8°; 2.8-cm radius) and CPE-44Y plate, Brookfield, Middleboro, Mass.
f. Isotemp circulator, Model 900, Fischer Scientific, Pittsburg, Pa.
g. Stata, version 14.2, StataCorp LP, College Station, Tex.
h. Hanel RM, Jack J, Ruterbories L, et al. Heat labile proteins, not packed cell volume, contribute to thromboelastographic changes in anemia (abstr). *J Vet Emerg Crit Care* 2017;27:S12.

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