

# Usefulness of whole blood, plasma, peritoneal fluid, and peritoneal fluid supernatant glucose concentrations obtained by a veterinary point-of-care glucometer to identify septic peritonitis in dogs with peritoneal effusion

Amie Koenig, DVM, and Lindsey Lane Verlander, DVM

**Objective**—To evaluate the usefulness of a veterinary point-of-care glucometer for identification of septic peritonitis in dogs with peritoneal effusion (PE).

**Design**—Prospective clinical evaluation.

**Animals**—39 dogs with PE.

**Procedures**—Blood and peritoneal fluid convenience samples were collected concurrently in all dogs at the time of initial evaluation. A veterinary point-of-care glucometer was used to measure glucose concentration in heparinized whole blood, plasma, peritoneal fluid, and peritoneal fluid supernatant samples. Seventeen dogs had confirmed septic peritonitis, and 22 dogs had nonseptic PE. Sensitivity, specificity, positive and negative predictive values, and accuracy of identification of dogs with septic peritonitis were calculated for glucose concentration differences for whole blood versus peritoneal fluid (WB-PF), plasma versus peritoneal fluid (P-PF), and plasma versus peritoneal fluid supernatant (P-PFS).

**Results**—With a cutoff of > 20 mg/dL, the glucose concentration difference for WB-PF was an insensitive indicator of septic peritonitis (sensitivity, 41.2%; specificity, 100%). In comparison, the glucose concentration differences for P-PF and P-PFS had a higher sensitivity for septic peritonitis (88.2% and 82.4%, respectively) but a lower specificity (80% and 77.8%, respectively). With a glucose concentration difference cutoff of  $\geq$  38 mg/dL, specificity, positive predictive value, and accuracy of P-PF and P-PFS improved.

**Conclusions and Clinical Relevance**—Determination of the glucose concentration difference for WB-PF with the veterinary point-of-care glucometer was not useful in identifying all dogs with septic peritonitis. A glucose concentration difference of  $\geq$  38 mg/dL for P-PF or P-PFS, however, supported an accurate diagnosis of septic peritonitis in dogs with PE. (*J Am Vet Med Assoc* 2015;247:1027–1032)

Rapid and accurate diagnosis of septic peritonitis is critical to patient survival. Many diagnostic tests that evaluate peritoneal fluid samples can be used to identify septic peritonitis, including cytologic identification of intracellular bacteria or high total nucleated cell counts and positive bacteriologic culture results. Although bacterial culture is the gold standard for identifying septic peritonitis, results take several days to obtain, which may delay appropriate, timely therapeutic interventions. Previous research has shown the difference between blood and peritoneal fluid glucose concentrations has 100% sensitivity and specificity in identifying septic peritonitis in dogs if the peritoneal fluid has a glucose concentration that is at least 20 mg/

## ABBREVIATIONS

PE	Peritoneal effusion
POC	Point of care
P-PF	Plasma versus peritoneal fluid
P-PFS	Plasma versus peritoneal fluid supernatant
TP	Total protein
WB-PF	Whole blood versus peritoneal fluid

dL lower than that of the peripheral blood,<sup>1</sup> and this has become a popular method of attempting to diagnose septic peritonitis. Glucose concentration in that study<sup>1</sup> was measured with a blood chemistry analyzer, which may not be available for immediate, POC use in many practices. Point-of-care handheld glucometers provide rapid, inexpensive, and readily available data and thus are commonly used in veterinary patients to measure glucose concentrations.

Most POC glucometers use one of several electrochemical reactions to detect glucose. Enzymes within the test strips react with glucose in the blood and produce an electrical current proportional to the amount of glucose present in the sample. The meter senses the current (amperometric meter) or total charge (coulometric meter)

From the Department of Small Animal Medicine and Surgery, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.

Dr. Verlander's present address is Eastside Animal Medical Center, 1835 Grayson Hwy, Grayson, GA 30017.

Abbott Animal Health donated the glucometer and test strips.

Presented in part as an oral presentation at the University of Georgia Science of Veterinary Medicine Research Symposium, Athens, Ga, October 2011.

The authors thank Dr. Deborah Keys for statistical assistance.

Address correspondence to Dr. Koenig (akoenig@uga.edu).

produced by the reaction and reports the corresponding estimated plasma glucose concentration.<sup>2</sup>

Point-of-care glucometers designed for humans have variable degrees of accuracy when used with veterinary species.<sup>3-5</sup> Inaccuracies may stem, in part, from species differences in the ratio of glucose that is bound to hemoglobin versus that which is free in the plasma.<sup>6</sup> To minimize these inaccuracies, a glucometer<sup>a</sup> that uses a species-specific algorithm that accounts for the variable percentage of glucose bound to hemoglobin to provide the plasma equivalent of the glucose concentration has been marketed specifically for veterinary use. The algorithm generally renders the glucometer more accurate than most human glucometers when used in veterinary species.<sup>3,5,7</sup>

Point-of-care glucometers are subject to numerous influences that impact their ability to provide accurate results, including physical or environmental factors, patient or operator factors, and the presence of certain drugs in the blood. A low Hct falsely increases glucose measurements reported by POC glucometers.<sup>2,7-10</sup>

The goal of the study reported here was to evaluate the veterinary POC glucometer as a tool for identifying dogs with septic peritonitis. Given that most peritoneal fluid samples are hemodilute, we hypothesized that the veterinary POC glucometer would fail to reliably identify dogs with septic peritonitis on the basis of the previously reported cutoff value of 20 mg/dL for the glucose concentration difference for WB-PF, whereas the glucose concentration difference for P-PF would more reliably identify septic peritonitis in dogs with PE. We also hypothesized that use of a higher cutoff value for the glucose concentration differences for WB-PF and P-PF would achieve greater specificity.

## Materials and Methods

**Study design**—Acquisition of convenience blood and peritoneal samples was in compliance with institutional clinical research guidelines. Samples were obtained from dogs with PE identified at the time of initial evaluation at the veterinary teaching hospital or from dogs that developed PE during hospitalization.

For inclusion in the study, dogs had to have had heparinized whole blood and peritoneal fluid samples collected concurrently as part of a standard diagnostic evaluation. Venous blood was collected either by direct venipuncture or via an IV catheter immediately following placement. This blood was placed into a microcollection tube containing lyophilized lithium heparin<sup>b</sup> and analyzed within 5 minutes after collection. Peritoneal fluid samples were treated in a similar manner and analyzed within 5 minutes following acquisition by aseptically abdominocentesis with a needle and syringe.

Exclusion criteria included lack of peritoneal fluid or blood evaluation, nonconcurrent acquisition of peritoneal fluid and blood samples, peritoneal fluid collection via indwelling drainage tubes, delayed time between sample collection and analysis, and hemoperitoneum. Inability to confirm or rule out septic peritonitis (eg, cytologic findings of neutrophilic inflammation in peritoneal fluid but no bacteria seen and no bacteriologic culture performed) was also an exclusion criterion.

Medical records were retrospectively evaluated to confirm the determined cause of PE in dogs. Presence

of septic peritonitis was ultimately confirmed via identification of bacteria on cytologic evaluation of peritoneal fluid, positive peritoneal fluid bacteriologic culture results, identification of a perforation in the gastrointestinal tract during surgical exploration, or necropsy confirming a septic focus. Presence of nonseptic PE was identified via cytologic evaluation, necropsy, surgical exploratory confirming a nonseptic cause, or final diagnosis of a nonseptic etiology.

**Measurements**—Portions of whole blood and peritoneal fluid samples were placed in microhematocrit tubes; PCV was determined, and total protein concentration was measured by refractometry. The POC glucometer<sup>a</sup> was used to measure glucose concentration in whole blood and peritoneal fluid immediately after collection and in plasma and peritoneal fluid supernatant immediately after centrifugation.

The glucose concentration differences for WB-PF, P-PF, and P-PFS were calculated, and sensitivity, specificity, positive predictive value, negative predictive value, and accuracy for the identification of septic peritonitis were calculated for each calculated difference.

**Statistical analysis**—A commercial statistical software package<sup>c</sup> was used to perform statistical tests. Data were tested for normality. All hypothesis tests were 2-sided, with a significance threshold of  $\alpha = 0.05$ . Mean  $\pm$  SD or median and range were reported for each variable for dogs with septic peritonitis and dogs with nonseptic PE. Student *t* tests were used to compare mean PCV and total protein concentration in whole blood and peritoneal fluid between dogs with septic peritonitis and dogs with nonseptic PE. The folded form *F* statistic was used to test whether variances were equal between dogs with septic peritonitis and dogs with nonseptic PE. Paired *t* tests were used to compare mean PCV and TP concentration in WB and PF, mean glucose measurements in whole blood versus plasma and peritoneal fluid versus peritoneal supernatant, and mean glucose concentration differences for WB-PF, P-PF, and P-PFS. Glucose concentrations in whole blood, plasma, peritoneal fluid, and peritoneal fluid supernatant were compared between dogs with septic peritonitis and dogs with nonseptic PE by means of either a Mann-Whitney test or *t* test, depending on whether data were normally distributed. A cutoff value for diagnosis of septic peritonitis was chosen for the veterinary POC glucometer on the basis of identification of a natural breakpoint in the data.

## Results

Of the 39 dogs with PE, 17 had septic peritonitis and 22 had nonseptic PE confirmed after sample collection by review of medical records. Four additional dogs with PE were excluded from the study because of the inability to confirm or rule out septic peritonitis; these dogs had neutrophilic peritoneal fluid without visible organisms on cytologic examination, and peritoneal fluid had not been submitted for bacteriologic culture.

Causes of septic peritonitis included gastrointestinal perforation (perforated gastric or duodenal ulcers [*n* = 3], neoplastic rupture of intestinal wall [1], and necrosis of intestinal wall [1]), postoperative dehiscence

of gastrointestinal incisions (3), septic uroperitoneum (3), extension of bacterial hepatitis or hepatic abscess (2), and unknown septic focus (4). In the 3 dogs that developed septic peritonitis after surgery, the original surgery was not for septic peritonitis but involved biopsy or resection of portions of the gastrointestinal tract. Six of the 17 dogs with septic peritonitis had peritoneal fluid samples submitted for bacteriologic culture, yielding 1 to 3 bacterial isolates each, with 8 genera represented overall. Eleven dogs with septic peritonitis did not have bacteriologic culture of peritoneal fluid samples performed because they were euthanized. Six dogs with septic peritonitis had been receiving antimicrobials for various durations (hours to days) at the time of peritoneal fluid collection.

In dogs with nonseptic PE, diagnoses included pancreatitis ( $n = 4$ ), nonperforating gastrointestinal foreign body (3), hepatobiliary disease or failure (3), renal failure (3), gastroenteritis (2), uroperitoneum (1), intussusception (1), congestive heart failure (1), pericardial effusion (1), postoperative resection and anastomosis with systemic inflammatory response and hypoproteinemia (1), hepatic neoplasia (1), and anaphylaxis (1). Three of 22 dogs with nonseptic PE had peritoneal fluid samples submitted for bacteriologic culture; results were negative for each of these 3 dogs. Ten of 22 dogs with nonseptic PE were receiving antimicrobials for variable periods prior to peritoneal fluid collection.

For each dog, there was a significant and clinically relevant difference in glucose concentration as measured with the veterinary POC glucometer in whole blood versus plasma ( $P < 0.001$ ) but not between peritoneal fluid and peritoneal supernatant ( $P = 0.140$ ). In 5 dogs with septic peritonitis, the glucose concentration measured in peritoneal fluid was higher than that in whole blood. All 4 dogs that had peritoneal fluid or peritoneal fluid supernatant glucose concentrations that were below the limits of detection of the glucometer had septic peritonitis.

Median whole blood glucose was 96 mg/dL (range, 28 to 153 mg/dL) for dogs with septic peritonitis and 111 mg/dL (range, 38 to 209 mg/dL) for dogs with nonseptic PE. Mean plasma glucose concentration was  $153.6 \pm 54.0$  mg/dL for dogs with septic peritonitis and  $172.2 \pm 61.8$  mg/dL for dogs with nonseptic PE. There was no significant difference in glucose concentration between whole blood or plasma between dogs with septic peritonitis and dogs with nonseptic PE. Four dogs with nonseptic PE and 1 dog with septic peritonitis were considered hyperglycemic (whole blood glucose concentration,  $> 140$  mg/dL). Of the 4 dogs with nonseptic PE, 3 were anemic (PCV, 23%, 23%, and 13%) and the dog with septic peritonitis had a PCV of 35%. Hypoglycemia (glucose concentration,  $< 60$  mg/dL) was seen in only 1 dog with nonseptic PE (whole blood glucose concentration, 38 mg/dL) and 2 dogs with septic peritonitis (whole blood glucose concentration, 59 and 28 mg/dL). One additional dog with septic peritonitis had severe hypoglycemia (whole blood glucose concentration, 34 mg/dL) and had received an IV bolus of dextrose approximately 30 minutes prior to collection of the paired whole blood and peritoneal fluid samples. Mean glucose concentration in peritoneal fluid was 82

$\pm 52.3$  mg/dL for dogs with septic peritonitis and  $173.8 \pm 64.4$  mg/dL for dogs with nonseptic PE. Mean glucose concentration in peritoneal fluid supernatant was  $86.1 \pm 57.4$  mg/dL for dogs with septic peritonitis and  $176.6 \pm 65.7$  mg/dL for dogs with nonseptic PE. Glucose concentration in peritoneal fluid and peritoneal supernatant did differ significantly ( $P < 0.001$ ) between dogs with septic peritonitis and dogs with nonseptic PE. The mean PCV and TP concentration in blood was  $46 \pm 12.7\%$  and  $6.0 \pm 1.6$  mg/dL in dogs with septic peritonitis, respectively, and  $36 \pm 12.4\%$  and  $5.6 \pm 1.6$  mg/dL for dogs with nonseptic PE, respectively. In peritoneal fluid samples, mean PCV was  $5.1 \pm 4.0\%$  and  $4.2 \pm 5.4\%$  for dogs with septic peritonitis and dogs with nonseptic PE, respectively, and mean TP concentration was  $3.7 \pm 1.4$  mg/dL and  $3.3 \pm 1.4$  mg/dL for dogs with septic peritonitis and dogs with nonseptic PE, respectively. There was no significant difference in PCV ( $P = 0.089$ ) or TP concentration ( $P = 0.6$ ) in blood between dogs with septic peritonitis and dogs with nonseptic PE. There was no significant difference in PCV ( $P = 0.66$ ) or TP concentration ( $P = 0.7$ ) in the peritoneal fluid between dogs with septic peritonitis and dogs with nonseptic PE. There was a significant difference in PCV ( $P < 0.001$ ) and TP concentration ( $P < 0.001$ ) between whole blood and peritoneal fluid in all dogs.

The mean glucose concentration difference for WB-PF was  $14.4 \pm 45.9$  mg/dL for dogs with septic peritonitis and  $-59.5 \pm 41.9$  mg/dL for dogs with nonseptic PE. The mean glucose concentration difference for P-PF was  $71.6 \pm 45.8$  mg/dL for dogs with septic peritonitis and  $-17.3 \pm 49.6$  mg/dL for dogs with nonseptic PE. The mean glucose concentration difference for P-PFS was  $67.5 \pm 48.9$  mg/dL for dogs with septic peritonitis and  $-5.6 \pm 23.6$  mg/dL for dogs with nonseptic PE (Figure 1). There was no significant difference between calculated mean glucose concentration differences for P-PF and P-PFS for each dog.

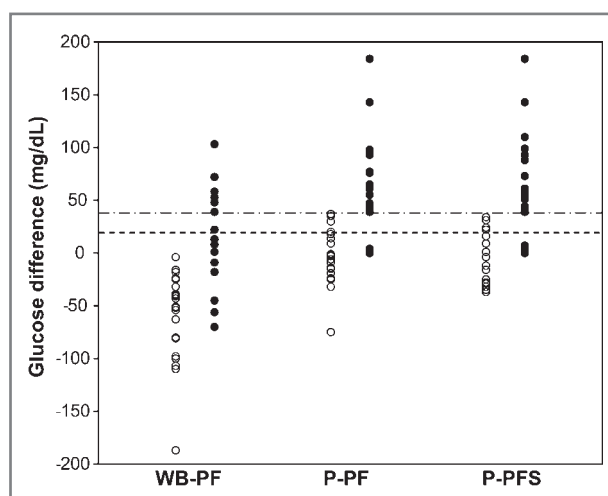


Figure 1—Calculated glucose concentration differences for WB-PF, P-PF, and P-PFS for whole blood and peritoneal fluid samples obtained from 39 dogs with PE and confirmed septic peritonitis (black circles) or nonseptic PE (white circles). The glucose concentration difference cutoff values of 20 mg/dL (dashed line) and 38 mg/dL (dashed and dotted line) are indicated.

With a cutoff value of 20 mg/dL for the mean glucose concentration difference in WB-PF, 10 of 17 dogs with septic peritonitis were incorrectly identified as having nonseptic PE. Of the 10 dogs, 3 had perforated gastroduodenal ulcers, 2 had septic uroperitoneum, 1 had dehiscence of intestinal incisions, 1 had extension of a hepatic infection, and 3 had an unknown source of septic peritonitis (2 of the 3 were suspected of having NSAID-induced perforating ulcers). Among the 10 dogs, 5 were receiving antimicrobials for various durations at the time of peritoneal fluid collection. Similarly, with a cutoff value of 20 mg/dL for mean glucose concentration difference in P-PF, 2 of 17 dogs with septic peritonitis were incorrectly identified as having nonseptic PE (1 of the 2 was receiving antimicrobials at the time of peritoneal fluid collection). With the same cutoff value to investigate the mean glucose concentration in P-PFS, 3 of 17 dogs with septic peritonitis were incorrectly identified as having nonseptic PE (1 of the 3 was receiving antimicrobials at the time of peritoneal fluid collection). One of the 3 dogs, a dog with postoperative dehiscence of intestinal incisions, was misidentified as having nonseptic PE on the basis of glucose concentration difference for WB-PF and P-PFS, but was correctly identified as having septic peritonitis on the basis of glucose concentration difference for P-PF. Two of the 3 dogs with septic peritonitis were misclassified as having nonseptic PE by glucose concentration differences for WB-PF, P-PF, and P-PFS. One of these 2 dogs had a perforated gastroduodenal ulcer (suspected to be NSAID induced), and the other had an unknown cause of septic peritonitis. This unknown cause was also suspected to be secondary to an NSAID-induced gastrointestinal perforation, and the dog was euthanized prior to treatment and a necropsy was not performed. This latter dog had a whole blood glucose concentration of 28 mg/dL and plasma, peritoneal fluid, and peritoneal fluid supernatant glucose concentrations below the limits of detection of the glucometer, so these measures were assigned a value of 20 mg/dL (the lower limit of detection of the glucometer).

Three additional dogs had peritoneal fluid and peritoneal fluid supernatant with glucose concentrations below the limit of detection of the glucometer, and these measures were assigned a value of 20 mg/dL. All 3 of these dogs, 1 with a ruptured liver abscess, 1 with postoperative dehiscence of a resection and anastomosis site, and 1 with septic peritonitis of unknown cause, were correctly identified as having septic peritonitis on the basis of all 3 (ie, P-PF, P-PFS, and WB-PF) calculated glucose concentration differences. One of these dogs had severe hypoglycemia, and an IV bolus of dextrose had been administered approximately 30 minutes prior

to identification of PE and acquisition of paired blood and peritoneal fluid samples. Because equilibration of glucose across the peritoneal membrane can take hours depending on the condition of the membrane, use of the glucose concentration (34 mg/dL) in whole blood obtained prior to the dextrose administration and 30 minutes prior to acquisition of the peritoneal fluid sample to calculate the glucose concentration difference for WB-PF (14 mg/dL) would have yielded a classification of nonseptic PE. Plasma glucose concentration was not measured when blood was drawn for the initial glucose concentration measurements in this dog, so the glucose concentration difference for P-PF and P-PFS prior to dextrose administration could not be calculated.

For dogs with nonseptic PE, all 22 dogs were correctly identified as having nonseptic PE on the basis of a cutoff value of 20 mg/dL for the glucose concentration difference for WB-PF. Five of the 22 dogs with nonseptic PE were incorrectly categorized as having septic peritonitis on the basis of a cutoff value of 20 mg/dL for the glucose concentration difference for P-PF, P-PFS, or both. One of these dogs was referred 24 hours after surgery for a ruptured bladder. The dog had cystoliths and an ongoing uroabdomen from a leaking bladder incision and was not receiving antimicrobials at the time of peritoneal fluid collection. For this dog, urine bacteriologic culture results were negative. The second dog had a foreign body without gastrointestinal perforation, and bacteriologic culture was not performed; the dog was not treated with antimicrobials before or after surgery. The third dog had pancreatitis and negative peritoneal fluid bacteriologic culture results and was receiving antimicrobials at the time of hospital admission. The fourth dog, subsequent to resection and anastomosis for a nonperforating foreign body, had severe hypoproteinemia and myocardial dysfunction, with a serosanguineous peritoneal fluid that had a glucose concentration difference for P-PF of 20 mg/dL and glucose concentration difference for P-PFS of 16 mg/dL. A final dog with bile peritonitis had a glucose concentration difference for P-PFS of 24 mg/dL and negative peritoneal fluid bacteriologic culture results.

A glucose concentration difference for WB-PF  $\geq$  20 mg/dL had 100% specificity for identification of septic peritonitis, but many dogs with septic peritonitis were misidentified as having nonseptic PE (ie, low sensitivity). The sensitivity, negative predictive value, and overall accuracy for identifying septic peritonitis on the basis of a cutoff value of 20 mg/dL were better for glucose concentration differences for P-PF and P-PFS, compared with the glucose concentration difference for WB-PF (Table 1). There was no calculated glucose con-

Table 1—Comparison of diagnostic attributes of glucose concentration differences for WB-PF, P-PF, and P-PFS measured on a POC glucometer at 2 cutoff values ( $>$  20 mg/dL and  $\geq$  38 mg/dL) for the detection of septic peritonitis in 39 dogs with PE.

Glucose difference	WB-PF (n = 39)		P-PF (n = 37)		P-PFS (n = 35)	
	$>$ 20 mg/dL	$\geq$ 38 mg/dL	$>$ 20 mg/dL	$\geq$ 38 mg/dL	$>$ 20 mg/dL	$\geq$ 38 mg/dL
Sensitivity (%)	41.2	35.3	88.2	88.2	82.4	82.4
Specificity (%)	100	100	80	100	77.8	100
Positive predictive value (%)	100	100	78.9	100	77.8	100
Negative predictive value (%)	68.8	66.7	88.9	90.9	82.4	85.7
Accuracy (%)	74.4	71.8	83.8	94.6	80	91.4



centration difference for WB-PF, P-PF, or P-PFS that differentiated between all dogs with septic peritonitis and dogs with nonseptic PE. Use of a cutoff value of 38 mg/dL for either glucose concentration difference for P-PF or P-PFS improved the specificity, positive predictive value, negative predictive value, and accuracy without a large impact on sensitivity. A cutoff value could not be established for the glucose concentration difference for WB-PF because of the wide overlap in glucose values between these measurements.

## Discussion

Results of the study presented here indicated that use of the difference in glucose concentrations, as measured with a veterinary POC glucometer, between whole blood and peritoneal fluid was an unacceptable method of identifying septic peritonitis when a cutoff value of 20 mg/dL was used. Ten of the 17 dogs with confirmed septic peritonitis were misclassified as having nonseptic PE. This finding is different from a previous study<sup>1</sup> that shows the difference in glucose concentration, as measured with a biochemical analyzer, between whole blood and peritoneal fluid has 100% sensitivity and specificity in identifying septic peritonitis in dogs.

Compared with WB-PF, glucose concentration differences for P-PF and P-PFS had a higher sensitivity for identifying septic peritonitis, with only 3 dogs with septic peritonitis being improperly classified as having nonseptic PE. Changing the mean glucose concentration difference cutoff value to 38 mg/dL for P-PF and P-PFS improved the overall accuracy and specificity of the test without a substantial reduction in sensitivity. Of the 3 misclassified dogs with septic peritonitis, 1 was misidentified as having nonseptic PE by all 3 glucose concentration differences (WB-PF, P-PF, and P-PFS); glucose concentrations in plasma, peritoneal fluid, and peritoneal fluid supernatant were below the limits of detection of the glucometer (thus, assigned a value of 20 mg/dL), and the whole blood glucose concentration was 28 mg/dL. These markedly subnormal glucose concentrations and minimal differences between whole blood, plasma, peritoneal fluid, and peritoneal fluid supernatant glucose concentrations precluded identification of glucose concentration differences > 20 mg/dL and thus prevented identification of septic peritonitis in the dog by use of any cutoff value for glucose concentration difference. It is possible that this dog's classification may have been different if the glucometer had a lower minimum limit of detection and was able to report the actual peritoneal fluid glucose concentrations. Removal of this dog's data from the analysis would increase sensitivity and the negative predictive value to 94%, without affecting the positive predictive value and specificity.

In the hypoglycemic dog with septic peritonitis that received an IV bolus of dextrose 30 minutes before paired sample acquisition, a blood sample obtained after dextrose administration was used to calculate the glucose concentration difference. However, equilibration of glucose across the peritoneal membrane varies with the quality of the membrane and can take hours. It is possible that use of the glucose concentration (34

mg/dL) in whole blood obtained prior to the dextrose administration may have better reflected the actual glucose concentration difference for WB-PF (14 mg/dL), which would have resulted in this dog with septic peritonitis being misclassified as having nonseptic PE. Plasma glucose concentration was not measured prior to dextrose administration, so glucose concentration differences for P-PF and P-PFS prior to the dextrose administration could not be calculated.

The total cell mass (cellularity) in samples may contribute to the inability to identify septic peritonitis in dogs by means of glucose concentration difference for WB-PF when the POC glucometer is used. Hematocrit is known to influence results determined with POC glucometers by altering the degree of plasma displacement by cells at the test strip. Typically, low Hcts yield elevated glucose concentrations and high Hcts yield lower glucose concentrations, compared with the gold standard,<sup>2,7-10</sup> because the low number of cells allows penetration of a greater volume of plasma into the test strip reagent layer, thus generating a greater electrochemical reaction and subsequently a higher reported glucose concentration (ie, pseudohyperglycemia). For dogs with severe anemia or hemoconcentration at the time of sample collection, whole blood glucose concentration as measured by the veterinary POC glucometer will be falsely elevated or reduced, respectively, making it more difficult to ascertain the true difference between whole blood and peritoneal fluid glucose readings. In peritoneal fluid samples from dogs with septic peritonitis, the total number of cells (both RBCs and WBCs) is low, compared with that of whole blood, providing a condition of water (plasma) excess in regard to the veterinary POC glucometer measurements. This would falsely elevate the peritoneal fluid glucose concentration and would diminish the calculated glucose concentration difference for WB-PF.

Laboratory-based biochemical analyzers are not subject to the inaccuracies generated by water exclusion or excess in whole blood or peritoneal fluid samples. When the veterinary POC glucometer was used, application of plasma and either peritoneal fluid or peritoneal fluid supernatant to the test strips allowed comparison of samples with similar water content; thus, more relevant glucose concentration difference calculations were obtained. In addition to Hct, partial pressure of oxygen and partial pressure of carbon dioxide, blood pH, and certain drugs including mannitol and dopamine can affect POC glucometer measurements.<sup>2,11</sup> Essentially, anything that occupies space within the blood, including excessive lipid or proteins, excludes water from the volume of blood in a sample and can yield a lower reported glucose concentration (ie, pseudohypoglycemia). The presence or influence of these additional substances was not investigated for the dogs of the study presented here.

In a pilot study, all 7 dogs with hemoperitoneum (6 with ruptured hemangiosarcoma and 1 with ruptured hepatocellular carcinoma) were falsely identified as having septic peritonitis by all 3 calculations (ie, WB-PF, P-PF, and P-PFS), with glucose concentrations measured by the POC glucometer (data not shown) on the basis of either cutoff value (ie, > 20 mg/dL or ≥ 38 mg/dL).

Dogs with hemoperitoneum were excluded from the present study because diagnosis is rarely a challenge in affected dogs and we do not routinely perform biochemical evaluation of the hemorrhagic peritoneal fluid. Cellularity of the samples may have contributed to these findings as well, given that glucose may be consumed by blood cells present within the peritoneal fluid, thus decreasing the measured glucose concentrations.

In summary, the glucose concentration difference for WB-PF as measured with this veterinary POC glucometer cannot be used to reliably identify septic peritonitis, regardless of the cutoff value chosen. Obtaining a P-PF or P-PFS glucose concentration difference of  $> 20$  mg/dL with the veterinary POC glucometer may have an acceptable sensitivity for identifying septic peritonitis in dogs, whereas glucose concentration differences for P-PF and P-PFS  $\geq 38$  mg/dL had greater specificity. In conjunction with fluid analysis, fluid cytologic evaluation, physical examination, and other clinical findings, the glucose concentration difference should be used as supportive evidence of septic peritonitis because a negative result (glucose concentration difference  $\leq 20$  mg/dL) does not rule out septic peritonitis for all affected dogs.

- 
- a. AlphaTRAK 2, Abbott Laboratories, Abbott Park, Ill.
  - b. Capiject, Terumo Medical Corp, Somerset, NJ.
  - c. SAS, version 9.2, SAS Institute Inc, Cary, NC.
- 

## References

1. Bonczynski JJ, Ludwig LL, Barton LJ, et al. Comparison of peritoneal fluid and peripheral blood pH, bicarbonate, glucose, and lactate concentration as a diagnostic tool for septic peritonitis in dogs and cats. *Vet Surg* 2003;32:161–166.
2. Tonyushkina K, Nichols JH. Glucose meters: a review of technical challenges to obtaining accurate results. *J Diabetes Sci Technol* 2009;3:971–980.
3. Cohen TA, Nelson RW, Kass PH, et al. Evaluation of six portable blood glucose meters for measuring blood glucose concentration in dogs. *J Am Vet Med Assoc* 2009;235:276–280.
4. Johnson BM, Fry MM, Flatland B, et al. Comparison of a human portable blood glucose meter, veterinary portable blood glucose meter, and automated chemistry analyzer for measurement of blood glucose concentrations in dogs. *J Am Vet Med Assoc* 2009;235:1309–1313.
5. Zini E, Moretti S, Tschuor F, et al. Evaluation of a new portable glucose meter designed for the use in cats. *Schweiz Arch Tierheilkd* 2009;151:448–451.
6. Coldman MF, Good W. The distribution of sodium, potassium and glucose in the blood of some mammals. *Comp Biochem Physiol* 1967;21:201–206.
7. Paul AE, Shiel RE, Juvet F, et al. Effect of hematocrit on accuracy of two point-of-care glucometers for use in dogs. *Am J Vet Res* 2011;72:1204–1208.
8. Lane SL, Koenig A, Brainard BM. Formulation and validation of a predictive model to correct blood glucose concentrations obtained with a veterinary point-of-care glucometer in hemodiluted and hemoconcentrated blood samples. *J Am Vet Med Assoc* 2015;246:307–312.
9. Pidcoke HF, Wade CE, Mann EA, et al. Anemia causes hypoglycemia in intensive care unit patients due to error in single-channel glucometers: methods of reducing patient risk. *Crit Care Med* 2010;38:471–476.
10. Mann EA, Salinas J, Pidcoke HF, et al. Error rates resulting from anemia can be corrected in multiple commonly used point-of-care glucometers. *J Trauma* 2008;64:15–20.
11. Tang Z, Du X, Louie RF, et al. Effects of drugs on glucose measurements with handheld glucose meters and a portable glucose analyser. *Am J Clin Pathol* 2000;113:75–86.