Association between outcome and changes in plasma lactate concentration during presurgical treatment in dogs with gastric dilatation-volvulus: 64 cases (2002–2008)

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Objectives—To determine whether changes in presurgical plasma lactate concentration (before and after initial fluid resuscitation and gastric decompression) were associated with short-term outcome for dogs with gastric dilatation-volvulus (GDV).

Design—Retrospective case series.

Animals—64 dogs.

Procedures—Medical records were reviewed, and signalment, history, resuscitative treatments, serial presurgical lactate concentrations, surgical findings, and short-term outcome were obtained for dogs with confirmed GDV.

Results—36 of 40 (90%) dogs with an initial lactate concentration ≤ 9.0 mmol/L survived, compared with only 13 of 24 (54%) dogs with a high initial lactate (HIL) concentration (> 9.0 mmol/L). Within HIL dogs, there was no difference in mean ± SD initial lactate concentration between survivors and nonsurvivors (10.6 ± 2.3 mmol/L vs 11.2 ± 2.3 mmol/L, respectively); however, there were significant differences in post-treatment lactate concentration, absolute change in lactate concentration, and percentage change in lactate concentration following resuscitative treatment. By use of optimal cutoff values within HIL dogs, survival rates for dogs with final lactate concentration > 6.4 mmol/L (23%), absolute change in lactate concentration ≤ 4 mmol/L (10%), or percentage change in lactate concentration ≤ 42.5% (15%) were significantly lower than survival rates for dogs with a final lactate concentration ≤ 6.4 mmol/L (91%), absolute change in lactate concentration > 4 mmol/L (86%), or percentage change in lactate concentration > 42.5% (100%).

Conclusions and Clinical Relevance—Calculating changes in plasma lactate concentration following initial treatment in dogs with GDV may assist in determining prognosis and identifying patients that require more aggressive treatment. (J Am Vet Med Assoc 2010;236:892–897)

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Abbreviations

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<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>CI</td>
<td>Confidence interval</td>
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<tr>
<td>GDV</td>
<td>Gastric dilatation-volvulus</td>
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<td>HIL</td>
<td>High initial lactate</td>
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<tr>
<td>OR</td>
<td>Odds ratio</td>
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<td>ROC</td>
<td>Receiver operating characteristic</td>
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Gastric dilatation-volvulus is an acute life-threatening condition that primarily affects large-breed dogs. By definition, the initiating event of the syndrome involves an abnormal entrapment and accumulation of fluid and air within the gastric lumen that results in extreme, traumatic distention of the stomach (dilatation) and variable rotation of the stomach on its long axis (volvulus). Early clinical signs (discomfort, pacing, panting, drooling, retching, and abdominal distention) are indicative of the initial events.

However, the pathogenesis of GDV involves a more complex progression of physiologic disturbances that can rapidly (within hours) culminate in death unless immediate medical and surgical treatment is provided. Increased intragastric pressure obstructs blood flow in the caudal vena cava and portal vein, which results in decreased venous return and cardiac output (ie, obstructive shock). Although compensatory mechanisms are activated initially to maintain arterial pressure, corrective measures must be taken, or inadequate tissue perfusion will progress and compromise organ function. Eventually, the shock will become refractory, multiple organs will fail, and death will be imminent. Even with intensive treatment, including pre-surgical stabilization, surgery, and postsurgical care, mortality rates for dogs with GDV range from 13% to 43%.2,6

To lower morbidity and mortality rates, a clear understanding of the progression of a disease is required, and the ability to accurately assess and address factors most critical to survival is needed. Ideally, prognostic factors can be identified to assess critical components in the progression of disease, which can then be used to guide alternative or aggressive treatment strategies when appropriate. In numerous studies, investigators have identified prognostic indicators with regard to death of dogs with GDV. However, many of these indicators are of limited utility with regard to improving morbidity and mortality rates because they do not have alternative treatment strategies (eg, gastric necrosis, partial gastrectomy, and combined splenectomy and partial gastrectomy) or are complications that develop later in the course of the disease and alternative treatment strategies are limited (eg, peritonitis, sepsis, and disseminated intravascular coagulation).8
Early recognition and treatment are often required to prevent a disease from developing and acquiring severity. Risk factors for the likelihood of dying as a result of GDV that can be evaluated early in the course of the condition (eg, prior to surgical intervention) have been identified and include clinical status at time of admission, duration of clinical signs, and a single pretreatment plasma lactate concentration. Dogs that have signs of depression or that are comatose at admission are 3 or 36 times as likely to die, respectively. In 1 study, dogs with clinical signs for ≥ 5 hours prior to examination at a veterinary hospital had a mortality rate of 46%, whereas in another study, dogs with clinical signs for > 6 hours prior to examination at a veterinary hospital were significantly more likely to die. An initial plasma lactate concentration < 6 mmol/L has been reported to be associated with a survival rate of 99% in dogs with GDV, whereas an initial plasma lactate concentration > 6 mmol/L in that same study was associated with a survival rate of 58% in dogs with GDV.

Of the early prognostic indicators, the only accurately measurable, objective assessment is plasma lactate concentration at admission. In 1 study, a single lactate measurement could be used to accurately predict survival in dogs with an initial lactate concentration < 6 mmol/L, but it could not be used to accurately predict survival within the group of dogs with an initial lactate concentration > 6 mmol/L. In typical physiologic conditions, lactate concentration is maintained in a narrow range by a balance between production and consumption. Elevated blood lactate concentration is most commonly a result of tissue hypoperfusion and hypoxia that develop as a result of shock. During anaerobic conditions, pyruvate is converted to lactate for energy. If anaerobic conditions are severe, such as in a state of shock, lactate production will exceed the rate of metabolism. Once aerobic conditions are restored, lactate should be cleared via metabolism. However, during more advanced stages of shock (eg, decompensating or refractory shock), lactate may not be cleared even when adequate tissue perfusion is achieved. For this reason, evaluating serial lactate concentrations could potentially be used to help determine response to treatment and overall prognosis.

In humans, the use of lactate concentration as a diagnostic, therapeutic, and prognostic marker of global tissue hypoxia during shock has been established in numerous studies. Lactate concentration has also been evaluated as a marker of disease severity and as a prognostic indicator in veterinary medicine. However, the diagnostic value of a single lactate concentration as a marker of tissue hypoperfusion is debatable. Currently, serial measurement of lactate concentrations or lactate clearance in humans is becoming accepted as a more precise marker of cellular hypoxia and shock. (ie, effective lactate clearance implies reversible shock and a higher likelihood of survival vs poor lactate clearance seen with more advanced stages of shock) and a better prognosticator of organ failure and death in patients with circulatory and septic shock. Consistently, patients whose lactate concentrations began to return to the reference range after treatment had a better outcome, compared with the outcome for patients whose lactate concentrations remained high after treatment. Therefore, the objective of the study reported here was to determine whether there was an association between the response of plasma lactate concentration following presurgical treatment (fluid resuscitation and gastric decompression) and survival in dogs with GDV. Given that untreated GDV involves a complex progression of circulatory disturbances advancing through the stages of shock, our hypothesis was that changes in preoperative lactate concentration in response to initial treatment would be associated with outcome in dogs with GDV. We believed information from our institution was appropriately suited for investigation of changes in lactate concentration because a defined resuscitation protocol for use in dogs with GDV was applied uniformly. To our knowledge, no prior studies have examined changes in serial lactate measurements following presurgical treatment as prognostic indicators for dogs with GDV.

**Materials and Methods**

**Case selection**—Medical records of all dogs with GDV examined at the Cummings School of Veterinary Medicine at Tufts University between February 2002 and February 2008 were reviewed. Dogs were included in the study only when duration of clinical signs prior to admission was < 24 hours, no fluids had been administered IV prior to admission, > 1 plasma lactate concentration had been measured before surgery, fluid resuscitation excluded the use of blood products, and the diagnosis of GDV was confirmed radiographically and during surgery. Three dogs with initial plasma lactate concentrations within the reference range (each of which survived) were excluded from the study.

**Medical records review**—The following information was obtained from the medical records: signalment, history, IV administration of fluids, serial plasma lactate concentrations, use of anti-inflammatory medications, surgical findings, and short-term outcome (survived to be discharged from the hospital vs died or were euthanized because of severity of clinical signs or perceived prognosis). Dogs were presumed to have gastric necrosis when gastric resection or invagination was performed during surgery.

**Change in plasma lactate concentration and percentage**—Plasma lactate concentration was determined in heparinized blood samples by use of an amperometric autoanalyzer, as described elsewhere. Absolute change in lactate concentration was assessed as the initial lactate concentration minus the postresuscitation lactate concentration. Percentage change in lactate concentration was calculated as: (Initial lactate concentration – postresuscitation lactate concentration)/Initial lactate concentration × 100.

**Statistical analysis**—All measurements were expressed as range and median or as mean ± SD. Logistic regression analysis was used to determine those variables that were important predictors of outcome after adjusting for the effects of other variables associated with outcome (survival). The OR and 95% CI were reported for variables with significant associations. Variables included in the logistic regression analysis were percentage change in lactate concentration, interval between lactate measurements while the dog received fluids via IV administration, and volume of fluids administered IV. No multicollinearity was detected among these variables. The dependent variable was survival. Backward stepwise selection was applied to the variables in the regression

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**References**

1. Smith, J. et al. JAVMA, Vol 236, No. 8, April 15, 2010

2. Scientific Reports

3. Small Animals
model. After determination of variables that significantly affected outcome, a 2-tailed unpaired Student t test was used to compare differences between groups. A commercial software package was used for all statistical calculations. Values of P < 0.05 were considered significant.

The ROC curves were generated by use of data from all 64 dogs and from a subset of 24 dogs with an initial lactate concentration > 9 mmol/L to determine optimal values of initial plasma lactate concentration, final plasma lactate concentration, absolute change in lactate concentration, and percentage change in lactate concentration for use in predicting survival until discharge from the hospital. The sensitivity (y-axis) versus 1 – specificity (x-axis) for every possible threshold value was plotted graphically, and the prediction result closest to the top left corner of each ROC curve was used to define the optimal cutoff point.

Results

One hundred forty-one dogs were treated for GDV during the study period. Of these, 64 met the criteria for inclusion in the study. Age ranged from 1.3 to 14.6 years (median, 8.2 years). Thirty-nine (61%) dogs were males and 25 (39%) were females; 28 (72%) of the males and 22 (88%) of the females were neutered. The most common breeds were the German Shepherd Dog (n = 13 dogs) and Akita (8); all other breeds were represented by < 4 dogs.

Forty-nine of 64 (77%) dogs survived until discharge from the hospital. Of the 15 (23%) dogs that did not survive, 8 were euthanized during surgery because of severe gastric necrosis that was considered nonresectable, 3 died after surgery as a result of cardiopulmonary arrest, and 4 were euthanized after surgery because of a deteriorating clinical status (coagulopathy, hyperproteinemia, and hemoabdomen 2 days after surgery [n = 1 dog]; respiratory distress and hypoxemia necessitating ventilatory support 2 days after surgery [1]; acute renal failure, refractory arrhythmias, and hemoabdomen 1 day after surgery [1]; and neurologic deterioration from a suspected thromboembolic event resulting in respiratory arrest necessitating ventilatory support [1]). Twenty-six of 64 (41%) dogs had gastric necrosis at the time of surgery. Of the 26 dogs with gastric necrosis, 12 (46%) were euthanized or died during surgery or in the immediate postoperative period. Three of the 4 dogs euthanized during the postoperative period because of a deteriorating clinical status had no signs of gastric necrosis at the time of surgery. Fourteen dogs underwent gastric resection and were discharged from the hospital. None of the dogs were administered anti-inflammatory medications while hospitalized.

Logistic regression analysis revealed that the percentage change in lactate concentration was a significant predictor of outcome (P = 0.034; OR, 1.03; 95% CI, 1.002 to 1.054), after adjusting for fluid administration prior to surgery and interval between preoperative lactate measurements while being administered fluids. Mean ± SD volume of fluid administered IV prior to surgery was 89 ± 36 mL/kg (40.4 ± 16 mL/lb), and mean interval between preoperative lactate measurements was 82 ± 32 minutes. Fluids administered IV included crystalloid solutions alone (n = 56 dogs) or in combination with hetastarch (5), hypertonic saline (7.5% NaCl solution (1), or hetastarch and hypertonic saline solution (2). Total fluid volume administered was calculated by use of conversions to an equivalent crystalloid volume for hetastarch (3.6 X volume) and hypertonic saline solution (7.7 X volume) to account for increased expansion.1 Decompression of the stomach of each dog was accomplished during the interval between lactate measurements by passage of a stomach tube, percutaneous gastrocentesis, or a combination of both.

Data acquired from the serial measurements of plasma lactate concentration for all dogs with GDV were summarized (Table 1). For all dogs with GDV, the initial and final plasma lactate concentrations were significantly lower and the percentage change in lactate concentration significantly greater in survivors, compared with results for nonsurvivors. However, there was no significant difference for all dogs with GDV in the absolute change in lactate concentration between survivors and nonsurvivors.

Receivers operating characteristic data for all dogs with GDV were determined (Table 2). Optimal survival cutoff values were as follows: pretreatment lactat-

### Table 1—Mean ± SD serial plasma lactate concentration data for 64 dogs with GDV.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Survivors (n = 60)</th>
<th>Nonsurvivors (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial lactate concentration (mmol/L)</td>
<td>6.2 ± 2.2</td>
<td>10.3 ± 3.2*</td>
</tr>
<tr>
<td>Final lactate concentration (mmol/L)</td>
<td>3.3 ± 2.3</td>
<td>8.0 ± 3.3*</td>
</tr>
<tr>
<td>Absolute change in lactate concentration (mmol/L)</td>
<td>2.9 ± 3.3</td>
<td>2.6 ± 2.0</td>
</tr>
<tr>
<td>Percentage change in lactate concentration (%)</td>
<td>49.1 ± 28.8</td>
<td>24.6 ± 19.4*</td>
</tr>
</tbody>
</table>

Survivors were defined as dogs that underwent surgery to correct GDV and were subsequently discharged from the hospital; nonsurvivors were dogs that died or were euthanized during surgery to correct GDV or during the postoperative period.

*Within a row, value differs significantly (P < 0.05) from the value for survivors. n = Number of dogs.

### Table 2—Optimal cutoff values (on the basis of ROC data) for predicting survival of 64 dogs with GDV.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Optimal cutoff</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Value &gt; cutoff</th>
<th>Value ≤ cutoff</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial lactate concentration (mmol/L)</td>
<td>9.0</td>
<td>0.735</td>
<td>0.733</td>
<td>54 (13/24)*</td>
<td>90 (36/40)</td>
</tr>
<tr>
<td>Final lactate concentration (mmol/L)</td>
<td>5.6</td>
<td>0.837</td>
<td>0.800</td>
<td>40 (8/20)*</td>
<td>93 (41/44)</td>
</tr>
<tr>
<td>Percentage change in lactate concentration (%)</td>
<td>42.2</td>
<td>0.612</td>
<td>1.000</td>
<td>100 (30/30)*</td>
<td>56 (19/34)</td>
</tr>
</tbody>
</table>

Values in parentheses are number of dogs that survived/number of dogs in the group. *Within a row, value differs significantly (P < 0.05) from the value for survival > cutoff.
Posttreatment lactate concentration ≤ 9.0 mmol/L, posttreatment lactate concentration ≤ 5.6 mmol/L, and percentage change in lactate concentration > 42.2% for all dogs with GDV, the proportion of dogs surviving of those that had an initial lactate concentration ≤ 9.0 mmol/L, final lactate concentration ≤ 5.6 mmol/L, or percentage change in lactate > 42.2% was significantly greater than the proportion of dogs surviving of those that had an initial lactate concentration > 9.0 mmol/L (ie, HIL concentration), final lactate concentration > 5.6 mmol/L, or percentage change in lactate ≤ 42.2%, respectively. An optimal cutoff for absolute change in lactate concentration for all dogs with GDV was not determined because there was no significant difference between survivors and nonsurvivors (Table 1). The area under the curve of these data (based on the ROC curve of the logistic regression analysis) was 0.75, which indicated this was a good model for use in predicting survival.

Data from the subset of 24 HIL dogs (ie, dogs with an initial lactate concentration > 9 mmol/L), which was determined from the initial ROC curve, were evaluated to determine whether serial lactate measurements could be used to more accurately predict survival within this group. Logistic regression analysis for this subset of HIL dogs revealed that the percentage change in lactate concentration was a significant predictor of outcome (P = 0.048; OR, 1.45; 95% CI, 1.03 to 2.088), after adjusting for fluids administered IV prior to surgery and interval between preoperative lactate measurements while being administered fluids. Mean ± SD amount of fluids administered IV prior to surgery was 100 ± 43 mL/kg (45 ± 19 mL/lb), and mean interval between preoperative lactate measurements was 80 ± 36 minutes.

Data acquired from the serial measurements of plasma lactate concentration for HIL dogs with GDV were summarized (Table 3). Within the HIL dogs, there was no significant difference between initial mean ± SD plasma lactate concentrations for survivors (10.6 ± 2.3 mmol/L) and nonsurvivors (11.2 ± 2.3 mmol/L). However, final preoperative plasma lactate concentration after treatment was significantly lower, whereas absolute change in lactate concentration and percentage change in lactate concentration were significantly higher, in survivors than in nonsurvivors.

Receiver operating characteristic data for dogs with GDV with an initial lactate concentration > 9 mmol/L (HIL dogs) were determined (Table 4). Optimal survival cutoff values were as follows: lactate concentration after treatment ≤ 6.4 mmol/L; absolute change in lactate concentration > 4.0 mmol/L; and percentage change in lactate concentration > 42.5%. For HIL dogs, the proportion of dogs surviving of those with a final lactate concentration > 6.4 mmol/L, absolute change in lactate concentration ≤ 4 mmol/L, or percentage change in lactate concentration ≤ 42.5% was significantly lower than the proportion of dogs surviving of those with a final lactate concentration ≤ 6.4 mmol/L, absolute change in lactate concentration > 4 mmol/L, or percentage change in lactate concentration > 42.5%, respectively. The area under the curve of these data was 0.98 (determined on the basis of the ROC curve of the logistic regression analysis), which indicated that this was an excellent model for use in predicting survival.

### Discussion

In the study reported here, changes between serial plasma lactate measurements obtained during presurgical treatment (ie, before and after initial fluid resuscitation and gastric decompression) were significantly associated with survival in dogs with GDV. Analysis of the data from all dogs with GDV admitted to our veterinary hospital that met the inclusion criteria revealed that there was a positive association between survival and initial plasma lactate concentration, final plasma lactate concentration, and percentage change in plasma lactate concentration. Dogs that survived until discharge from the hospital had a significantly lower initial and final plasma lactate concentration and a larger percentage decrease in plasma lactate concentration than did those that were euthanized or died prior to discharge. However, the absolute change in lactate concentration did not differ significantly between survivors and nonsurvivors, likely because of the fact that some of the dogs had a relatively low initial plasma lactate concentration and therefore could not have a substantial absolute reduction. Survival rate for dogs with an initial plasma lactate concentration ≤ 9.0 mmol/L (the optimal cutoff...
for all dogs with GDV) was significantly higher than that for dogs with an initial plasma lactate concentration > 9 mmol/L.

Importantly, within the subset of 24 HIL dogs (ie, dogs with an initial plasma lactate concentration > 9 mmol/L), changes between serial measurements of plasma lactate concentration (ie, before and after fluid resuscitation and gastric decompression) were significantly associated with survival. Specifically, final lactate concentration, absolute change in lactate concentration, and percentage change in lactate concentration were all good predictors of survival within HIL dogs despite the fact that the initial lactate concentration did not differ significantly between survivors and nonsurvivors. This finding is notable because it traditionally has been considered difficult to predict survival within this category of dogs with GDV.

In veterinary medicine, lactate concentration has been used as a marker of disease severity or as a prognostic indicator. Hyperlactatemia has been associated with a higher mortality rate among dogs in an intensive care unit.19 horses with colic,10,12,29 dogs with bacte-
sis,3,30 and dogs with GDV.3 Contrary to the situation for human medicine, the concept of lactate clearance in the veterinary literature is sparsely mentioned. Two studies10,31 in systemically ill dogs revealed that serial lactate measurements obtained during the course of treatment were important for evaluating prognosis. In both of those studies, mortality rates were increased in dogs in which the lactate concentration at a predetermined time point after treatment did not decrease by more than 50% from the initial concentration.

As previously mentioned, preoperative plasma lactate concentration in dogs with GDV is reported1 to be a good indicator of gastric necrosis and outcome. By the use of cutoff values determined from ROC curves, investigators in 1 study2 identified an ideal predictive value of 6 mmol/L with survival rates of 99%. Interestingly, use of a cutoff value of 6 mmol/L in the study reported here yielded a similar survival rate (23/24 [96%]). However, our optimal initial cutoff value for lactate concentration was 9 mmol/L. One possible explanation for this difference could be that 23 of 102 (23%) dogs in that other study2 received fluids IV prior to initial measurement of the lactate concentration, whereas none of the dogs in the present study received fluids before initial measurement of the lactate concentration.

Gastric necrosis is an important factor for use in predicting postoperative complications and death. Mortality rates for dogs with GDV but without gastric necrosis vary from 2% to 14%,2–4 whereas dogs with GDV and gastric necrosis have higher mortality rates (ranging from 31% to 63%).2,5 The mortality rate of dogs with gastric necrosis in our study (12/26 [46%]) agrees with those data. Although gastric necrosis has been significantly associated with death in numerous studies,2,3,5,32 it was interesting that 3 of 15 dogs in the study reported here that did not have a response in lactate concentration after fluid resuscitation had no signs of gastric necrosis at the time of surgery but deteriorated clinically after surgery and were ultimately euthanized. The cause of death in these dogs included acute renal failure and coagulopathies consistent with disseminated intra-vascular coagulation that were not evident at the time of admission to the hospital. The persistently elevated plasma lactate concentration in these dogs is consistent with hypoperfusion and with decompensatory shock that progressed to refractory shock. Whether more aggressive treatment of these dogs could have prevented this progression is unknown.

Although our study was retrospective in nature, analysis of our data revealed a strong consistency for treatment with regard to IV administration of fluids as well as interval between plasma lactate measurements (ie, there were no significant differences between groups). None of the dogs in this study received fluids IV prior to the initial measurement of plasma lactate concentration. A small percentage of dogs (8/64 [13%]) received hetastarch or hypertonic saline solution (or both) in addition to crystalloid solutions. Most of those dogs were nonambulatory at the time of admission to the hospital, were considered to be in extremely critical condition, and were considered likely to benefit from the volume-expanding properties of these products. Administration of solutions consisting of colloids and hypertonic crystalloids does not apparently adversely affect the outcome of patients in critical condition and may potentially help with cardiovascular compromise. Animals receiving hetastarch have higher cardiac output and oxygen delivery and a lower blood lactate concentration than do those receiving lactated Ringer's solution alone.33 Additionally, the use of synthetic colloids or hypertonic saline solution in dogs with GDV was associated with a significant decrease in the risk of developing hypotension.8

Although it is standard practice at our veterinary hospital to aggressively treat dogs with GDV by IV administration of fluids until certain resuscitation endpoints (such as return of heart rate and blood pressure to within the respective reference ranges) are achieved, a weakness of the study reported here is that no definitive endpoint for treatment was confirmed. The use of plasma lactate concentrations may be a better marker of adequate resuscitation than are endpoints based on results of physical examination because lactate concentrations are an assessment of resolution of hypoperfusion. Critically ill humans with occult hypoperfusion or elevated blood lactate concentrations without signs of clinical shock have higher morbidity and mortality rates.34–37 Most deaths in intensive care units in human hospitals are secondary to multiple organ failure as an end result of systemic inflammatory response syndrome, which is most often a result of continuing hypoperfusion.37

Another limitation to our study was the inclusion of dogs euthanized on the basis of a subjective decision about gastric resectability and deterioration of the clinical condition after surgery. It is possible that had the surgeon attempted resection or had the owner pursued extensive treatment after surgery, some of these dogs may have survived.

Analysis of our data suggested that initial plasma lactate concentration is a good indicator of survival but that changes in lactate concentration in response to resuscitative treatment are also important indicators of survival in dogs with GDV. Importantly, even in the HIL
dogs, the final lactate concentration, absolute change in lactate concentration, and percentage change in lactate concentration were all good indicators of survival. It is important to exercise caution in applying these results to individual patients. The small sample size makes it difficult to determine a precise cutoff value for survival of individual dogs with GDV. We believe our cutoff values are useful for comparing populations for research and resource allocation. Additional studies that involve more patients may help to more accurately determine precise cutoff values.

Obtaining serial plasma lactate concentrations as part of the preoperative point-of-care testing can be achieved with relative ease at most veterinary referral hospitals. Additionally, portable lactate analyzers are now available that can be used to quickly obtain blood and plasma lactate concentrations. Analyzing the changes in plasma lactate concentration in response to initial fluid resuscitation and gastric decompression in dogs with GDV may assist in determining an earlier and more accurate prognosis and in identifying patients that require alternative or more aggressive treatment.

**References**