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AUTHORS: Cortellini, S., Seth, M. and Kellett-Gregory, L. M.

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Plasma Lactate in septic peritonitis: a retrospective study of 83 dogs (2007-2012)

Abstract

Objective: To determine if absolute lactate or lactate clearance in dogs with septic peritonitis is associated with morbidity and mortality.

Design: Retrospective cohort study from 2007-2012.

Setting: University teaching hospital accepting referrals and primary emergencies.

Animals: Eighty-three dogs diagnosed with septic peritonitis were included. All the patients had at least one lactate measurement taken during the course of the hospitalization.

Results: Sixty-four percent of the patients survived to discharge, 22% were euthanized and 14% died during hospitalization. Lactate concentration > 2.5 mmol/l on admission (29% of the patients) was associated with mortality (p=0.001). Median admission lactate (n=81) was significantly different between non-survivors (2.5 mmol/l, range 0.5-8.4) and survivors (1.4 mmol/l, range 0.5-9.7) (p=0.007). Admission lactate higher than 4 mmol/l yielded a sensitivity of 36% and a specificity of 92% for non-survival. The inability to normalise lactate within 6 hours (n=10/24) yielded a sensitivity of 76% and specificity of 100% for non-survival. Post-operative hyperlactatemia (n=18/76) had a sensitivity of 46% and specificity of 88% for non-survival. Persistent post-operative hyperlactatemia (n=11/18) had a sensitivity of 92% and a specificity of 100% for non-survival. Regarding
the lactate clearance at 6 hours, the area under the curve (AUC) (n=20) was 0.81 and the
best cut-off was 21% (sensitivity 54% and specificity 91% for non-survival). AUC for
lactate clearance at 12 hours (n=18) was 0.92, with the best cut-off value defined as 42%
(sensitivity of 82% and a specificity of 100% for non-survival).

Conclusions: In this study absolute lactate and lactate clearance were good prognostic
indicators in septic peritonitis. Further studies are needed to assess its role as a
resuscitative end-goal parameter.
Introduction

Septic peritonitis is defined as the presence of microbial contamination of the peritoneal cavity. It carries a high mortality with a reported survival to discharge rate in dogs ranging from 29% to 71%.1-3

Septic peritonitis can be either primary or secondary to intra-abdominal organ leakage2,3 and can lead, even with early surgical treatment, to septic shock. The pathophysiology of the disease is intimately related to the complexity of sepsis, including systemic inflammation and its repercussions on major organ systems, cardiovascular instability and the underlying disease process responsible for the peritonitis. It is therefore difficult to identify a specific and reliable prognostic factor. In human medicine, there has been growing interest towards identification of biomarkers able to act not only as prognostic indicators but also as clinical indicators to guide therapy. This interest has extended also to veterinary medicine.6

Many studies, both in human and veterinary medicine, have examined absolute lactate level in the clinical context both for its diagnostic and prognostic value. Lactate is an end-product of anaerobic metabolism and its increase can indicate tissue hypoxia.9,10 Therefore its role is not only relevant in assessing the degree of overt or occult hypoperfusion but also in assessing tissue oxygenation.11,12

Several studies have examined lactate in different critically ill populations and these suggest that serial lactate measurements and the change in response to therapy are a more significant prognostic indicator than the single lactate level at admission.12-14 Lactate clearance, expressed as the percentage change over time of lactate level, seems to be the most sensitive prognostic indicator but interest has also been shown towards...
lactime, defined as time from admission taken to normalize plasma lactate concentration. The objective of this study was to assess if elevated plasma lactate concentration at admission or lactate clearance were able to predict survival in dogs with septic peritonitis. Additional study aims were to evaluate the association between plasma lactate concentration and increased morbidity, defined as longer ICU stay, transfusion administration or pressor requirements.

**Materials and Methods**

The electronic medical records databases at a university teaching hospital were searched for cases of septic peritonitis in dogs. Inclusion criteria were a diagnosis of septic peritonitis, either on presentation or at any point during hospitalization. The criteria for diagnosis were defined as the finding of intracellular bacteria or fungal agent on cytological evaluation—on abdominal fluid culture or intra-operative evidence of macroscopic leakage of the gastro-intestinal tract or intra-abdominal rupture of an abscess or pyometra. At least one lactate measurement during the course of the hospitalization was necessary to be included in the study.

Missing or incomplete records or uncertain diagnosis or euthanasia without attempted surgical treatment were exclusion criteria.

Medical records for enrolled cases were reviewed by a single author. Data including signalment, relevant history, cardio-respiratory parameters, available blood work, intraoperative findings, post-operative requirements for pressors, transfusions and colloids, and outcome were recorded. Hypotension was defined as a systolic Doppler pressure less than 90 mmHg or a mean arterial pressure less than 60 mmHg when
oscillometric or invasive measurement was used. Multiple organ dysfunction syndrome (MODS) was identified based on the criteria defined by Kenney et al\textsuperscript{16}.

APPLE scores, in full and fast form \textsuperscript{17}, were also retrospectively calculated where suitable data were available and expressed as the mortality prediction probability (score 0-1).

Blood lactate was measured with a bench-top blood gas analyzer\textsuperscript{a} using heparinized venous or arterial blood samples. Hyperlactatemia was defined as a blood lactate concentration \textgreater 2.5mmol/L\textsuperscript{18}.

Where available, serial blood lactate and base excess concentrations were recorded, together with their time relative to presentation and surgery.

Lactate clearance, defined as the percentage change in lactate, was calculated for each case that was initially hyperlactatemic at 3, 6, 12 and 24 hours from admission as \[
\frac{(\text{Lactate}_{\text{adm}} - \text{Lactate}_{\text{hour \times}})}{\text{Lactate}_{\text{pres}}} \times 100.
\]
The plasma lactate concentration at each of these time intervals were extracted from the medical records with a tolerance of +/- 2hours, except for the 3 hour time point which was given a tolerance of \pm 30 minutes.

Lac-time was defined as the time (hours) passed until the normalization (meant as return within the reference interval, 0-2.5 mmol/L) of lactate from admission was achieved.

\textbf{Statistical analysis}

The retrospective data were recorded in a spreadsheet using Microsoft Excel\textsuperscript{b} and were analyzed with SPSS\textsuperscript{c}.
Each data set was analyzed for distribution using the Shapiro-Wilk test and expressed as mean (± SD) or median (range) depending on the presence or absence of a normal distribution.

Continuous variables were compared with the Student’s t-test for parametric data and Mann Whitney-U test for non-parametric data. Discrete variables were analyzed for difference using ANOVA for parametric data and Kruskal-Wallis test for non-parametric data.

Categorical variables were analyzed using Chi-squared or Fisher’s exact test if the contingency table contained an expected value <4.

For lactate clearance and absolute plasma lactate concentration, a ROC curve was elaborated and an optimal cut-off visually defined for each time point.

**Results**

The initial medical record database search identified 363 cases. After application of the inclusion and exclusion criteria, 83 dogs with septic peritonitis were included in the study.

The median age was 60 months (range 4-142) and the median weight was 25.7 kg (range 1.7-46.1). There were a variety of breeds represented in this study with the most represented being Labradors (13), springer spaniels (8) and cross breeds (5). Males were 53/83 (64%) (36/83 (43%) neutered and 17/83 (20%) entire) and females were 30/83 (36%) of the population (spayed 20/83 (24%) and entire 10/83 (12%)).

Seventy-seven (93%) were referred from another veterinary practice whereas 6/83 (7%) were seen as primary emergency cases. Sixty-one (74%) of the patients received
intravenous fluids at another clinic prior to presentation, while 22/83 (26%) did not receive any fluids beforehand. Forty-four (53%) had had another surgery within the 10 days prior to presentation.

Regarding the focus of the septic peritonitis, in 73 dogs (89%) the gastro-intestinal system was involved, in 5 dogs (6%) there was a urogenital focus, 3 dogs (4%) had hepatobiliary disease and 1 (1%) had a primary peritonitis. In terms of underlying disease, 38 dogs (46%) had a gastro-intestinal foreign body, 11 (13%) had gastro-intestinal ulceration, leading to perforation, 6 (7%) were diagnosed with neoplasia, 7 (8%) had intestinal biopsies, 4 (6%) had a gastric-dilation volvulus. Three (4%) had an intussusception, 2 (2.4%) each had hepatic disease, pyometra, abscessation, mesenteric volvulus, trauma and 1 (1%) each had dystocia, a mesenteric thrombus, retention of a surgical swab (gossypiboma) and a primary peritonitis.

Sixty patients (72%) had a positive culture from the abdominal fluid, 3 patients had Candida Albicans isolated, 7 patients had a negative culture and 13 patients did not have a culture performed.

Fifty-three (64%) of the patients survived to discharge, 18/83 (22%) were euthanized and 12/83 (14%) died during hospitalization.

Median hospitalization time was 7 days (range 0-21) and the median time spent in the ICU was 3 days (range 0-17). MODS was identified in 15/83 (18%) of the patients. Median APPLE full score was 0.06 (range 0.00-0.95) and median APPLE fast score was 0.21 (range 0.01-0.99). APPLE full and fast score was calculated in 46/83 patients (Table 1).

On admission, the mean heart rate was 126 bpm (SD ±34), the median respiratory rate was 32 bpm (range 14-100) and the median temperature was 38.6°C (range 36-40.4). The mean systolic Doppler blood pressure recorded on admission was available for
37/83 cases and was 140 mmHg (SD ± 35), with 4/83 (5%) of these dogs being hypotensive. Oscillometric blood pressure was recorded in 6/83 dogs on admission and the mean of the mean blood pressure was 85 (SD ±13). Median blood glucose was 5.8 mmol/l (range 1.8-10.4).

Lactate on admission was available for 81/83 cases with a median of 1.7 mmol/l (range 0.5-9.7) and median base excess for 78/83 dogs was -4.9 mmol/l (range -17.1 - +5.3) (table 2).

Because of the retrospective nature of the study, plasma lactate measurements were not available for all the dogs at the different time points; complete serial lactate concentration was available for seventeen (Table 2).

Synthetic colloids (hydroxyethyl starches) were used in 46/83 (55%) of the patients, human albumin in 9/83 (11%), blood products (packed red blood cells or plasma) in 30/83 (36%) and vasopressors (dopamine or norepinephrine) in 16/83 (19%) of the patients.

Lactate level on admission was not predictive of administration of colloids (p 0.14) or albumin (p 0.53), although it was predictive for the use of blood products (p<0.01) and vasopressors (p < 0.01). Dogs with hyperlactatemia on admission had a significantly shorter ICU stay (median 2 days, range 0-11) compared to patients presenting with normolactatemia (median 4, range 0-17) (p=0.03). When comparing only patients which survived, normolactatemic patients did not have significantly higher median total stay in the hospital (8.3 days, range 5-21) compared to the hyperlactatemic patients (8.2 days, range 4-13) (p=0.72) or have a shorter ICU stay (4 days, range 1-17) compared to the hyperlactatemic patients (4.5 days, range 1-11) (p=0.73). These numbers need to be
interpreted in light of the retrospective nature of the study with the length in the ICU and the discharge time being clinician and finance-based.

Twenty-four (29%) patients had hyperlactatemia on admission and 14 of these dogs did not survive. Hyperlactatemia on admission was associated with mortality (p=0.01) (Figure 1).

Lactate on admission higher than 4 mmol/l was significantly associated with mortality (odds ratio 7.1, 95% C.I 2-25, p=0.002). 15/81 patients had lactate on admission higher than 4 mmol/l. Of these (11/15) 73% of the patients died, versus (19/68) 28% of the patients with a lactate < 4 mmol/l. A lactate higher than 4 mmol/l on admission yielded a sensitivity of 36% and a specificity of 92% for non-survival.

Lactate time could be calculated for 24 dogs of which 13 patients died and 11 survived. Of the 24 dogs, 10 failed to resolve hyperlactatemia (ranging from 1 hour to 24 hours) over the documented measurements and all of these patients did not survive. Of the remaining 14 patients that did have resolution of hyperlactatemia, 11 survived and 3 did not survive.

An ROC curve of the lactate time yielded poor sensitivity and specificity for survival with an area under the curve of 0.66.

Data regarding resolution of hyperlactatemia within 6 hours were available for 24 dogs. All the patients that did not clear lactate within 6 hours died (10/10). Of the dogs which cleared their hyperlactatemia within 6 hours (14/24), 11 (79%) survived and 3 (21%) died. The inability to clear lactate within 6 hours yielded a sensitivity of 76% and specificity of 100% for non-survival.

Lactate post-surgery was recorded in 76 dogs. Post-operative hyperlactatemia was present in 18 dogs, 12 of which died (p=0.02). Of the 58 dogs which were not
hyperlactatemic post-operatively, 14 died (24%) and 44 survived (76%). Post-operative
hyperlactatemia had a sensitivity of 46% and specificity of 88% for non-survival.

Persistent post-operative hyperlactatemia was significantly associated with mortality
(p < 0.001). Of 18 dogs which were hyperlactatemic postoperatively, 11 failed to clear
lactate. **Off these eleven dogs, 4 had normal lactate concentration on admission but**
all of them (11/11, 100%) did not survive to discharge. Of the remaining 7 dogs with
post-operative hyperlactatemia that resolved, 6 survived and 1 did not survive despite
clearing lactate. Persistent hyperlactatemia post-operatively yielded a sensitivity of 92%
and a specificity of 100% for non-survival.

An ROC curve was generated for lactate concentration at 6 hours (55/83) following
admission which had an AUC of 0.87 with a cut-off 2.3 mmol/l yielding a sensitivity of
68% and specificity of 92% for non-survival. Lactate clearance at 6 hours could be
calculated for 20 patients and ROC curve analysis produced an AUC of 0.81 with a
sensitivity 54% and specificity 91% for non-survival at 21% clearance.

Absolute lactate concentration at 12 hours post admission produced an ROC curve
with an AUC of 0.86 (n=54) with a cut-off of 2.4 mmol/l resulting in a sensitivity of 54%
and a specificity of 100% for non-survival. Lactate clearance at 12 hours post-admission
could be calculated for 18 patients and ROC curve analysis produced a curve with an AUC
of 0.90 and an optimal cut-off of 42% resulted in a sensitivity of 82% and specificity of
100% for non-survival (Figure 2).

Lactate clearance was not significantly different between survivors and non-survivors
at 3 hours post admission (n=9, p=0.25).
Discussion

Septic peritonitis is a complex and severe process with fluid loss and systemic inflammation caused by infectious agents in the peritoneal cavity and the causative underlying disease. Interactions between the inflammatory response, the coagulation and endothelial systems may lead to abnormalities in the macro- and micro-circulation, multi-organ failure and ultimately death. Hypoperfusion may occur through various pathophysiologic pathways, including hypovolemia, cardiogenic shock, vasoplegia, and splanchnic microthrombi.

Hyperlactatemia either represents an imbalance between tissue oxygen supply and demand (type A) or altered cellular metabolism (type B). In septic patients hyperlactatemia may occur due to hypoperfusion, mitochondrial dysfunction, increased leukocyte activity, organ dysfunction, decreased lactate clearance, Na⁺-K⁺ ATPase activation, hypoglycemia or dysregulation of pyruvate dehydrogenase. Recent veterinary studies have found neoplasia to be a cause of increased lactate but typically this is not clinically significant and was unlikely to be a significant confounder in our study as a neoplastic process was only identified in 7% of the enrolled patients. Similarly, the presence of a primary liver disease (2 patients), steroid administration (documented in 4 patients) or the use of cathecolamines could have increased the lactate serum levels and should be considered possible confounders of our study.

In our study lactate at admission was significantly different between survivors and non-survivors, although median lactate for non-survivors (2.5 mmol/L) was only the upper reference limit of 2.5mmol/l outlined by a previous study. This suggests that even moderate increase in lactate should be concerning, warranting immediate attention to the patient. Alternatively, a median lactate concentration within normal limits on
admission in non survivors may be suggesting that at this time point lactate is not predictive of death. In previous veterinary studies lactate on admission has achieved different significance. In a study on a heterogenous population of systemically ill dogs it was not found to be significantly associated with mortality\textsuperscript{12}; in patients with IMHA survivors had a lower lactate on admission (median 2.9 mmol/l) in comparison to non-survivors (4.8 mmol/l)\textsuperscript{14}. In dogs with Babesiosis the difference between survivors and non-survivors was more marked (2.6 mmol/l in survivors vs 8.5 mmol/l in non-survivors) and significant \textsuperscript{15}. These findings suggest that perhaps lactate values should be interpreted differently according to the underlying pathophysiologic process involved and therefore should always be considered within a disease-related context.

Severe hyperlactatemia on admission was defined as lactate higher than 4 mmol/l and was associated with a higher mortality. These results are in accordance with studies in human patients in which hyperlactatemia was associated with a higher mortality, independent of organ failure or shock\textsuperscript{25} and that a lactate higher than 4 mmol/l at admission was associated with a six-fold higher probability for death within 3 days\textsuperscript{26}. A retrospective study on dogs with IMHA showed that a cut-off of 4.4 mmol/l on admission was a good predictor of survival\textsuperscript{14}. In dogs with gastric dilation-volvulus an admission lactate of < 4.1 mmol/l was the best cut-off based on a ROC curve analysis for predicting survival\textsuperscript{27}.

Interestingly, and in contrast to another veterinary study in trauma patients\textsuperscript{6} base excess on admission was not significantly associated with survival in this study (p=0.17), highlighting that base excess can vary for reasons unrelated to lactate accumulation in septic patients. A reduction in standard base excess and consequent metabolic acidosis is likely multifactorial in sepsis, and the contribution of lactate can be less than 10%\textsuperscript{28}. 
Other contributing factors to acidosis could be represented by hyperchloremia, accumulation of inorganic unmeasured ions (e.g. keto acids or sulfate) or by a metabolic alkalosis by hypoalbuminemia\textsuperscript{29}.

Lactate on admission was associated with patient morbidity in this study, with a higher median lactate reported for dogs that \textbf{required} transfusions or vasopressors. Anemia and hypotension induce tissue hypoxia, due to reduced oxygen delivery and tissue perfusion respectively. It is assumed that patients that received transfusions or vasopressors had higher plasma lactate concentration ascribable to their anemia or hypotension but due the retrospective nature of the study we cannot exclude that high lactate was an actual indication for blood transfusion administration or vasopressor therapy. Somewhat surprisingly, hyperlactatemia was associated with a shorter ICU stay in this patient population. This finding is likely explained by the early euthanasia or death of this population.

Lactate was recorded at 3 hours and at 6, 12 and 24 +/- 2 hours after admission when available. The best area under ROC curves for absolute plasma concentration were obtained at 6 and 12 hours post admission; lactate level higher than 2.3 mmol/l at 6 hours or higher than 2.4 mmol/l at 12 hours were good predictors of mortality (specificity 92% and 100% respectively). These levels are only mildly elevated or within the reference range suggesting that the single value should be critically interpreted in consideration of the disease affecting the patient or that higher levels of lactate, but still within the normal limits established in healthy dogs, could indicate inadequate tissue oxygenation and the need for more medical attention. \textbf{In addition, the low sensitivity for mortality of the cut-offs at 6 and 12 hours suggest that lactate concentration below these...}
concentration should not decrease the clinicians’ concern for these patients which are still at risk or mortality.

The percentage clearance of lactate at 6 and 12 hours after admission was also found to be a good predictor of mortality in this study. This is in accordance with the findings of Stevenson et al.\textsuperscript{12} who examined systemically ill dogs with a variety of diseases and found that lactate clearance <50% at 6 hours after admission was associated with mortality. Similarly, Nel et al.\textsuperscript{15} found that a lactate clearance <50% at 8 and 16 hours after admission was a poor prognostic indicator in canine babesiosis. Additionally, failure to normalise lactate within 6 hours of admission has been found to be associated with mortality in dogs with immune mediated hemolytic anemia\textsuperscript{14}. Similar results have been found in septic people; lactate clearance at 6 hours was shown to be prognostically superior to central venous saturation of oxygen (ScvO\textsubscript{2})\textsuperscript{30} and a 24 hours window for lactate clearance was also a good predictor of survival\textsuperscript{31}.

There are several limitations to this study, most of which are intrinsic to its retrospective design. The majority of the patients (93%) included in the study was referred from another veterinarian and may have received treatment and in some cases even had surgery prior referral. As a result lactate measurements taken at the same time from admission may not represent the same stage of the disease in different patients. The lactate values were extracted from records rather than being prospectively timed and this likely led to more data points being available in the sickest patients. It is assumed that stable patients with normal lactate recorded continued to have normal lactate but in patients with hyperlactatemia on admission samples were serially measured to look for normalization.
The number of patients for which lactate was available at our predetermined time-points was low and therefore the power of the study when looking at lactate at time points other than admission is reduced. Also the +/- 2 hours tolerance for the time points might actually have influenced the results; a further prospective study with sampling performed at the time set could overcome the limitation of this retrospective study. The ability to euthanize patients is another limitation inherent to many veterinary studies. Given that the majority of patients were referred from another centre it is likely that financial constraints and owner motivation were less of a confounding factor in this population. The difference in APPLE scoring between the survivors and non-survivors supports the notion that mortality in this population was associated with disease severity rather than socioeconomic factors. In addition, the results regarding the hospitalization and ICU time or discharge time need to be interpreted with caution in this retrospective study, as these times can often be clinician and finance-based.

In conclusion, this study showed that in dogs with septic peritonitis lactate was associated with mortality and morbidity. In particular the results suggest that lactate clearance within a 6 to 12 hour window is associated with mortality and that post-operative hyperlactatemia should raise particular concerns. Further prospective studies are needed in order to assess the potential role of lactate-guided therapy in sepsis.
Footnotes:

a. Critical Care Xpress and Phox Ultra, Nova Biomedical

b. Microsoft Excell, version 2008, Microsoft Inc.

c. SPSS, version 20, SPSS Inc, Chicago Ill
References:


Table 1: Various parameters in dogs with septic peritonitis compared between survivors and non-survivors. Data presented as mean (+/- SD) or median (range). A p value of <0.05 was considered statistically significant.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>SURVIVORS</th>
<th>NON-SURVIVORS</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (bpm)</td>
<td>117 (±32)</td>
<td>141 (±30)</td>
<td>0.001</td>
</tr>
<tr>
<td>Respiratory rate (brpm)</td>
<td>32 (14-100)</td>
<td>32 (20-100)</td>
<td>0.6</td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>38.6 (36.3-40.3)</td>
<td>38.4 (36-40.1)</td>
<td>0.52</td>
</tr>
<tr>
<td>Doppler systolic pressure (mmHg)</td>
<td>145 (±26)</td>
<td>121 (±42)</td>
<td>0.04</td>
</tr>
<tr>
<td>Blood glucose (mmol/l)</td>
<td>5.7 (2.9-10.9)</td>
<td>6.3 (1.8-9.6)</td>
<td>0.16</td>
</tr>
<tr>
<td>Ionized Calcium (mmol/l)</td>
<td>1.16 (0.99-1.42)</td>
<td>1.1 (0.87-1.34)</td>
<td>0.013</td>
</tr>
<tr>
<td>Lactate adm. (mmol/l)</td>
<td>1.4 (0.5-9.7)</td>
<td>2.5 (0.5-8.4)</td>
<td>0.007</td>
</tr>
<tr>
<td>Base excess adm. (mmol/l)</td>
<td>-4.8 (-15-0.2)</td>
<td>-6.2 (-17.1-5.3)</td>
<td>0.17</td>
</tr>
<tr>
<td>ICU stay (days)</td>
<td>4 (1-17)</td>
<td>3 (0-11)</td>
<td>0.039</td>
</tr>
<tr>
<td>Hospital stay (days)</td>
<td>7 (4-21)</td>
<td>3 (0-12)</td>
<td>0.001</td>
</tr>
<tr>
<td>Time of administration of antibiotics from adm. (hours)</td>
<td>3 (0-48)</td>
<td>3 (1-56)</td>
<td>0.72</td>
</tr>
<tr>
<td>Time from admission to surgery (hours)</td>
<td>Survivors</td>
<td>Non survivors</td>
<td>P value</td>
</tr>
<tr>
<td>-----------------------------------------</td>
<td>-----------</td>
<td>---------------</td>
<td>---------</td>
</tr>
<tr>
<td><strong>APPLE full</strong></td>
<td>0.03 (0-0.38)</td>
<td>0.2 (0.01-0.95)</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>APPLE fast</strong></td>
<td>0.13 (0.01-0.77)</td>
<td>0.55 (0.05-0.99)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Table 2: Median plasma lactate concentration (range) in survivors and non survivors.

P value is expressed on the right side of the table.
Figure 1: Box and whiskers plot representing median lactate and interquartile range (box) at different time points from admission in survivors and non-survivors. The black dots represent outliers. Significance was determined with a p value < 0.05. A significant difference between the two groups was detected at admission, 6 hours, 12 hours and 24 hours post-admission.
Figure 2: ROC analysis of lactate clearance at 12 hours following admission for prediction of non-survival in hyperlactatemic dogs with septic peritonitis (AUC 0.90).