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Effect of acetate- and lactate-containing intravenous fluid on acid-base status, electrolytes and plasma lactate concentration in dehydrated cats

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Abstract

Objectives Acetate (ACE) and lactate (LAC)-containing balanced resuscitation fluids are commonly used for fluid therapy in cats. These fluids can influence acid-base and electrolyte status. This prospective randomised study compared two balanced crystalloid solutions regarding their effect on acid-base status, electrolytes and LAC concentrations in dehydrated cats after intravenous fluid therapy.

Methods A total of 100 client-owned cats presenting to the emergency service with dehydration $\geqslant$5% due to diarrhoea, vomiting or anorexia were included in this study. They were randomised to receive either ACE- (Sterofundin ISO; B Braun Vet Care) or LAC-containing (Ringer-Laktat nach Hartmann; B. Braun Vet Care) fluids for rehydration. Exclusion criteria were age <6 months, severe electrolyte abnormalities, severely increased creatinine or bolus therapy, and decompensated heart disease and liver diseases. Vital parameters were assessed and venous blood gas analysis was performed before and after fluid therapy. Data were analysed using the Mann–Whitney U-test and the Wilcoxon matched-pairs signed-rank test. The significance level was set at $P \leqslant 0.05$.

Results Post-rehydration pH normalised, and bicarbonate and base excess increased in both groups. Anion gap and LAC and potassium concentrations decreased in both groups. No difference in electrolyte, acid-base status and LAC was observed between cats receiving ACE and cats receiving LAC.

Conclusions and relevance Both fluids have similar effects on dehydrated cats' acid-base status and electrolyte and LAC concentrations. No significant differences in values were found between ACE- and LAC-containing resuscitation fluids. Blood LAC concentration decreased in both groups.

Keywords: Crystalloid solutions; acid-base disorders; fluid therapy; infusion; rehydration; base excess; bicarbonate

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Introduction

Administration of intravenous fluids in dehydrated cats is a common therapeutic measure.[1] Restoring hydration might be the most important treatment for patients experiencing dehydration.[2–4] Isotonic saline has been the standard rehydration fluid for cats.[3] It contains a higher amount of chloride compared with feline plasma. Larger doses of isotonic saline promote hyperchloraemic metabolic acidosis in human patients.[5,6] Balanced solutions are better tolerated and have a more physiologic composition.[5] Balanced fluids, such as acetate (ACE) or lactate (LAC)-containing solutions, mimic blood plasma in their electrolyte composition and contain potassium, calcium

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or magnesium as well as sodium and chloride.[7,8] In addition, balanced solutions contain buffer substances. Different buffers in resuscitation fluids can influence metabolism, oxygen consumption during the metabolism of buffer substances and acid-base status.[8] ACE is metabolised primarily in skeletal muscle and its metabolism is independent of oxygen and intact liver function. It is effective at low pH values and does not influence glucose or insulin concentration.[9,10] The use in paediatric patients whose liver metabolism is not yet fully functional and in patients with liver failure is possible without restrictions.[11] On the other hand, LAC metabolism occurs mostly in the liver and to a lesser extent in the kidneys and myocardial cells.[12] As LAC metabolism is oxygen-dependent, oxygen consumption increases during this process. Under anaerobic conditions, LAC metabolism slows down and LAC accumulates.[10] In the liver, LAC is either metabolised by lactate dehydrogenase (LDH) to pyruvate, which promotes adenosine triphosphate (ATP) production, or is used for gluconeogenesis.[13] Both consume hydrogen ions and lead to an increase in hydroxide ions, preventing metabolic acidosis.[8] The administration of large amounts of LAC-containing fluids can lead to a transient increase in plasma lactate.[12] Usually, this is not associated with acidosis because LAC is not present in the form of lactic acid but sodium lactate.[4,12,14]

In dogs, ACE metabolism was well maintained during shock, whereas LAC metabolism was significantly affected;[15] therefore, depending on the buffers included, fluids given to the patient can have different effects on metabolism, oxygen consumption and acid-base status. LAC-containing solutions are commonly used for rehydration; however, some concerns exist that these could cause hyperlactataemia or worsen lactic acidosis.[14,16] To date, studies comparing the use of ACE- and LAC-containing fluid for resuscitation or rehydration in cats are rare. Two studies compared the effect of different crystalloid infusion solutions on acid-base status and electrolytes in cats with urethral obstruction.[17,18] In one study of severely decompensated cats with experimentally induced urethral obstruction, the LAC-containing solution was more efficient than isotonic saline in restoring the acid-base and electrolyte balance.[18] In another study of cats with naturally occurring urethral obstruction, the use of a balanced ACE-containing electrolyte solution allowed for more rapid correction of blood acid-base status compared to isotonic saline.[17] A study evaluating ACE- and LAC-containing resuscitation fluids in dehydrated dogs found a decrease in heart rate after rehydration only in the ACE-containing fluid group.[8] Bolus therapy of ACE- and LAC-containing shock fluids in dogs with hypovolaemia had a minor impact on acid-base status and electrolyte concentrations.[12] LAC levels significantly decreased in both groups, with a stronger LAC reduction in the ACE-containing fluid group. Sodium and chloride levels increased during rehydration in both groups.[12]

The purpose of the present study was to compare the influence of ACE- and LAC-containing resuscitation fluids on acid-base status, electrolyte and blood LAC content in dehydrated cats.

Materials and methods

Study population

The study protocol was approved by the Ethics Committee of the Centre for Clinical Veterinary Medicine at the Ludwig-Maximilians-University (LMU) Munich (reference number 153-09-12-2018). Cats were included in the study after informed owner consent. The study was performed between June 2020 and September 2022. A total of 100 client-owned cats presenting to the emergency service of the LMU Small Animal Clinic were enrolled in this randomised prospective study. Cats aged older than 6 months and at least 5% dehydrated due to gastrointestinal clinical signs, such as vomiting, diarrhoea or anorexia, were included. Cats were scheduled for intravenous rehydration based on signs of dehydration from history and physical examination (mucous membrane moisture, skin turgor, eyeball position) according to the criteria described by Rudloff.[19] Exclusion criteria were hypovolaemia, severe electrolyte abnormalities (sodium <130 mmol/l or >160 mmol/l, potassium <3 mmol/l or >6 mmol/l), severely increased creatinine (creatinine >300 µmol/l) as well as cats with severe glucose deviations (<3.5 mmol/l or >15 mmol/l). In addition, patients with clinical signs associated with cardiac disease, dyspnoea or liver failure were excluded. During the rehydration period, no other fluids, electrolytes or vasopressors were allowed to be administered. Cats were excluded if they received any intravenous bolus therapy or subcutaneous fluid administration during the study period. In total, 100 cats were randomised to receive either an ACE-containing rehydration solution (Sterofundin ISO; B Braun Vet Care), or a LAC-containing solution (Ringer-Laktat nach Hartmann; B Braun Vet Care) for rehydration using an open-source program (www.randomizer.org) (Table 1; Figure 1).

Physical examination, blood collection and laboratory analysis

Physical examination to assess hydration status as mentioned above and collection of heparinised blood for venous blood gas analysis were performed before and immediately after calculated rehydration time when vital signs, used to assess dehydration assessment, normalised. Blood samples were collected from the cephalic or the medial saphenous vein either by venipuncture or via an indwelling catheter using a three-syringe technique

Table 1 Composition of resuscitation fluids used for rehydration in 100 dehydrated cats

Parameter (unit)	Sterofundin ISO (acetate)	Lactated Ringer's (lactate)
Sodium (mmol/l)	145	130.49
Chloride (mmol/l)	127	112
Potassium (mmol/l)	4	5.37
Calcium (mmol/l)	2.5	1.84
Magnesium (mmol/l)	1.0	–
Lactate (mmol/l)	–	27.84
Acetate (mmol/l)	24	–
Malate (mmol/l)	5	–
Osmolarity (mosmol/l)	308	277
pH	5.1–5.9	5.0–7.0

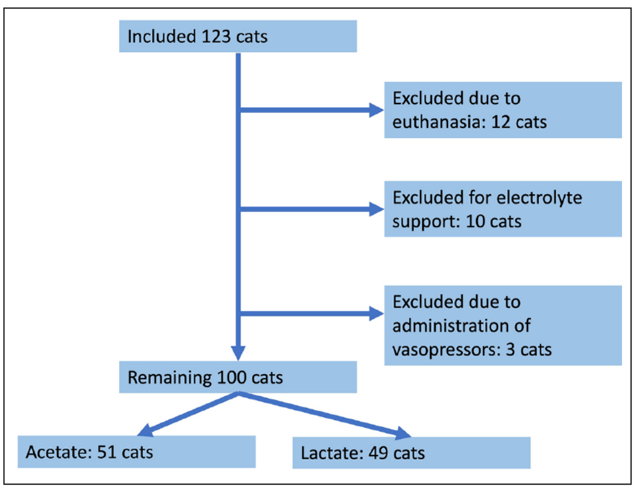

Figure 1 Cats included in the study and reasons for exclusion

without exposure to air. Venous blood gas analysis was performed within 10 mins (RAPIDpoint 500 System; Siemens Healthcare). The reference intervals used for the blood gas were established in the clinic's in-house laboratory. The results were corrected to the cat's body temperature. Analysed parameters include heart rate (beats/min), temperature (°C), pH, pCO_2 (mmHg), bicarbonate (mmol/l), base excess (BE), anion gap, sodium (mmol/l), chloride (mmol/l), potassium (mmol/l), calcium (mmol/l), LAC (mmol/l) and glucose (mmol/l).

Interventions

Intravenous rehydration was performed by constant rate infusion at the discretion of the clinician over a median time of 20 h. The rehydration rate was determined by the clinician and calculated individually for each patient according to their body weight, considering the degree of dehydration, additional losses and maintenance fluids according to the following formula: [body weight (kg) × 1000] × [percentage dehydration/100] = deficit (ml). The deficit was divided by the planned rehydration time to achieve the fluid rate in ml/h. Estimated ongoing losses in ml/h were added to this. Maintenance fluids were calculated from the formula: ml/h = body weight$^{0.75}$ × 70/24 and added to the rehydration rate and ongoing losses.[20]

Statistical analysis

A power analysis was performed with open-source software (www.powerandsamplesize.com). To detect a sodium difference of 3 mmol/l with a standard deviation of 3 mmol/l, with a power of 0.8 and an alpha error of 5%, 44 cats per group were required. A commercial software (Prism5 for Windows; GraphPad Software) was used for the statistical analysis. Normality was tested using the D'Agostino omnibus normality test. Normally distributed data are presented as mean ± SD, and non-normally distributed data are presented as median (range). The median age of cats in the ACE and LAC groups was compared using the Mann–Whitney U-test. The mean weight of cats in the ACE and LAC groups were compared using the *t*-test. A paired *t*-test was used to compare heart rate, pH, bicarbonate, BE, anion gap and sodium values in both groups before and after fluid resuscitation. A Wilcoxon matched-pairs signed-rank test was used to compare temperature, pCO_2, ionised calcium, chloride, glucose, potassium and LAC in both groups before and after fluid therapy.

Changes in heart rate, pH, pCO_2 and chloride between the two groups were compared using the *t*-test. Changes in temperature, bicarbonate, BE, anion gap, LAC, ionised calcium, glucose and potassium between the two groups were compared using the Mann–Whitney-U test. The number of cats with categorical physical parameters was analysed using the χ^2 test. A P value $\leqslant$0.05 was considered significant.

Results

Initially, 123 patients were enrolled in the study, of which 23 were subsequently excluded owing to euthanasia (n = 12), the requirement of electrolyte support (n = 10) or vasopressors (n = 3). Of the 100 cats included in the study, 29 were spayed females, nine were intact females, 47 were castrated males and 15 were intact males. The most represented breed was the domestic shorthair (n = 64), followed by Bengal (n = 7) and crossbreed (n = 7), Maine Coon (n = 5) and British Shorthair (n = 4). Other breeds were represented by a maximum of two cats per breed. The demographic data of cats in both groups were not different. The mean age was 6.0 years (range 0.5–18.0) in the ACE group and 9 years (range 0.6–16.0) in the LAC group (P = 0.429). Cats weighed a mean of 4.1 ± 1.2 kg in the ACE group and 4.5 ± 1.4 kg in the LAC group (P = 0.099) (Table 2). The main causes of dehydration were gastrointestinal disorders (n = 60), neoplasia-related illnesses (n = 12),

Table 2 Demographic data of 100 dehydrated cats receiving resuscitation fluids containing either ACE or LAC

Parameter (unit)	ACE (n = 51)	LAC (n = 49)	*P*
Age (years)	6.0 (0.5–18.0)	9.0 (0.6–16.0)	0.429
Weight (kg)	4.1 ± 1.2	4.5 ± 1.4	0.099
Dehydration (%)	6 (5–10)	6 (5–8)	0.266
Amount of resuscitation fluids (ml)	461 (190–1050)	510 (200–1155)	0.284
Amount of resuscitation fluids (ml/kg)	113 ± 24	108 ± 16	0.266
Duration of rehydration (h)	20 (12–24)	20 (15–24)	0.539
Rehydration rate (ml/kg/h)	5.3 (4.0–8.8)	5.0 (4.0–7.5)	0.204

Values are mean ± SD (*t*-test) or median (range) (Mann–Whitney U-test). $P < 0.05$ is considered statistically significant
ACE = acetate; LAC = lactate

infectious diseases (n = 6), intoxications (n = 5), fever of unknown origin (n = 4), neurological disorders (n = 3) and various other diseases (n = 10).

Intravenous rehydration was performed by constant rate infusion to the discretion of the clinician over 20 h (range 15–24) in the ACE group ($P = 0.539$) and 20 h (range 12–24) in the LAC group. The total amount of resuscitation fluids administered in ml ($P = 0.284$) and in ml/kg ($P = 0.266$) were not significantly different between the groups (Table 2). Pre- and post-rehydration heart rates did not differ in both groups (Table 3). Before rehydration, 79/100 cats presented with metabolic acidosis; after rehydration, the number of cats with metabolic acidosis decreased to 53. After rehydration, pH normalised in both groups (ACE: $P = 0.025$; LAC: $P = 0.001$). Bicarbonate (ACE: $P < 0.001$; LAC: $P < 0.001$) and BE (ACE: $P < 0.001$; LAC: $P < 0.001$) increased in both groups. Anion gap (ACE: $P < 0.001$; LAC: $P < 0.001$) and potassium concentration decreased in both groups (ACE: $P < 0.001$; LAC: $P = 0.001$). Electrolytes did not change significantly during rehydration (Table 4). After rehydration, LAC concentration (ACE: $P = 0.003$; LAC: $P < 0.001$) and glucose (ACE: $P < 0.001$; LAC: $P < 0.001$) decreased in both groups.

No significant differences in vital signs, acid-base status, LAC or electrolytes were detected between the ACE and LAC groups before and after rehydration.

Discussion

In the present study, the effect of ACE- and LAC-containing resuscitation fluids in dehydrated cats on acid-base balance, electrolyte and LAC concentrations was compared. All cats included in the study were dehydrated due to gastrointestinal clinical signs, such as vomiting, diarrhoea and anorexia. These clinical signs are a common cause of dehydration in cats, which can subsequently lead to hypovolaemia and further systemic distress if not treated appropriately.[21]

No statistically significant or clinically relevant changes in heart rate before and after rehydration in both groups were detected. A study of 90 dehydrated dogs comparing Sterofundin ISO with lactated Ringer's during rehydration found a larger decrease in heart rate in the ACE-containing fluid group. The dogs in this study were dehydrated owing to various diseases and

Table 3 Results of venous blood gas analysis, electrolytes and LAC in 100 dehydrated cats before and after rehydration, receiving resuscitation fluids containing either ACE (n = 51) or LAC (n = 49)

Parameter	Reference interval	ACE			LAC		
		Pre	Post	*P* value	Pre	Post	*P* value
Heart rate (beats/min)	140–200	193 ± 32.6	184.7 ± 25.2	0.67	186.2 ± 28.2	189.2 ± 24.7	0.473
Temperature (°C)	38.0–39.0	38.6 (37.0–40.7)	38.7 (36.4–40.1)	0.218	38.6 (36.1–41.0)	38.8 (37.3–40.5)	0.495
pH	7.34–7.44	7.31 ± 0.06	7.33 ± 0.03	0.003	7.30 ± 0.08	7.33 ± 0.05	0.001
pCO_2 (mmHg)	29–51	41.3 (26.5–61.2)	43.9 (28.1–53.7)	0.078	41.6 (27.2–59.3)	42.7 (27.2–63.4)	0.086
Bicarbonate (mmol/l)	19–24	19.1 ± 2.6	20.8 ± 1.9	<0.001	19.2 ± 3.4	21.0 ± 2.5	<0.001
Base excess	2.5–2.5	–6.1 ± 3.5	–3.6 ± 2.8	<0.001	–6.1 ± 4.8	–3.5 ± 3.3	<0.001
Anion gap	–	17.3 ± 3.8	14.9 ± 3.3	<0.001	17.1 ± 4.0	13.9 ± 3.5	<0.001
Sodium (mmol/l)	146–158	148.8 ± 3.7	149.3 ± 3.0	0.324	148.7 ± 5.0	148.4 ± 3.6	0.062
Potassium (mmol/l)	3.0–5.0	3.94 (3.03–5.78)	3.68 (2.72–4.70)	<0.001	3.94 (3.30–5.95)	3.69 (3.06–5.48)	0.002
Chloride (mmol/l)	110–124	115 (108–123)	117 (108–126)	0.301	116 (110–130)	117 (109–131)	0.313
Glucose (mmol/l)	3.7–6.9	7.0 (4.3–15)	5.3 (7.3–8.8)	<0.001	6.6 (4.4–13.7)	5.2 (3.6–9.9)	<0.001
LAC (mmol/l)	<2.5	1.99 (0.60–6.02)	1.57 (0.38–6.78)	0.009	1.85 (0.72–8.37)	1.37 (0.19–4.87)	0.004

Values are mean ± SD or median (range). $P < 0.05$ is considered statistically significant
ACE = acetate; LAC = lactate

Table 4 Changes in parameters of the venous blood gas analysis, electrolytes and LAC in 100 dehydrated cats before and after rehydration, receiving resuscitation fluids containing either ACE (n = 51) or LAC (n = 49)

Parameter (unit)	ACE	LAC	*P*
Heart rate (beats/min)	−8 ± 33.3	3.1 ± 29.7	0.064
Temperature (°C)	−0.1 (−3.9–2.8)	0.0 (−3.2–4.1)	0.717
pH	0.03 ± 0.06	0.03 ± 0.05	0.800
pCO_2 (mmHg)	1.7 ± 6.4	1.5 ± 5.9	0.907
Bicarbonate (mmol/l)	1.7 (−3.8–7.4)	1.9 (−6.6–6.4)	0.499
Base excess	2.3 (−4.4–10)	2.9 (−9.4–10.6)	0.603
Anion gap	−2.7 (−11.0–15.2)	−2.7 (−15.4–2.0)	0.539
LAC (mmol/l)	−0.6 (−3.3–4.4)	−0.6 (−8.4–1.0)	0.948
Sodium (mmol/l)	−0.3 (−1.5–0.7)	0.0 (−12.5–8.3)	0.065
Potassium (mmol/l)	−0.3 (1.5–0.7)	−0.2 (−2.6–0.8)	0.959
Chloride (mmol/l)	0.55 ± 3.77	1.55 ± 3.73	0.998
Calcium (mmol/l)	0.0 (−0.1–0.2)	0.0 (−0.2–0.2)	0.502
Glucose (mmol/l)	−1.3 (−8.4–2.6)	−1.5 (−6.1–2.5)	0.615

Values are mean ± SD or median (range). $P < 0.05$ is considered statistically significant
ACE = acetate; LAC = lactate

intravenous rehydration was performed over 6–24 h. As a possible explanation for the change in heart rate, the dogs in the LAC-containing fluid group could have been more severely ill compared with the dogs in the ACE-containing fluid group, as illness severity scores were not recorded.[8] In a study of 100 dogs in shock receiving a randomised bolus therapy of 30 ml/kg of either an ACE- (Sterofundin ISO) or LAC-containing (lactated Ringer's) solution over 10 mins, heart rates decreased in both groups after the fluid bolus.[12] Hydration and volume status are interconnected aspects of fluid balance in the body. While volume status relates to the amount of fluid in the circulatory system, hydration status refers to the overall fluid balance;[22] therefore, optimising hydration status may have influenced the volume status of the dehydrated dogs in this study to some degree.[8] As a result of increased preload, cardiac output increases and heart rate may decrease even in normovolaemic patients. Rehydration time was shorter in the dehydrated dogs compared with the dehydrated cats in the present study; therefore, the fluid rate was also higher in the dehydrated dogs compared with the dehydrated cats in the present study. This higher fluid rate could have a stronger influence on volume status; however, minor improvements in circulation status, blood flow and vascular tone cannot be demonstrated without more sophisticated monitoring. The dogs in that study could have had an increased heart rate on presentation caused by transportation and stress during initial examinations in an unfamiliar hospital environment, which later subsided.

In the present study of cats, blood pH, bicarbonate and BE increased significantly in both treatment groups after rehydration. Crystalloid fluids can modify the acid-base balance of plasma due to differences in their physicochemical composition relative to plasma.[3] Generally, BE is a calculated parameter for assessing the acid-base balance. Most of the cats (79/100) in this study were presented with metabolic acidosis (reference intervals: pH 7.34–7.44, bicarbonate 19–24 mmol/l, BE −2.5 to 2.5). After fluid therapy, the number of cats with metabolic acidosis decreased to 53/100. This suggests a partial correction of metabolic acidosis through the rehydration period that may not be caused by fluid therapy alone. However, other mechanisms and underlying comorbidities, such as cachexia, vomiting and diarrhoea, may also influence the acid-base status and can exacerbate metabolic acidosis.[12] Furthermore, without analysing creatine, urea, urine specific gravity and albumin, the possibility of pre-renal azotaemia or hyperalbuminaemia as a potential cause of metabolic acidosis in the cats in our study cannot be excluded.

In a study of 68 cats with naturally occurring urethral obstruction, the acid-base status normalised faster using balanced ACE-buffered fluid (Normosol-R) compared with isotonic saline.[17] While both crystalloid solutions appear safe and effective for fluid therapy in cats with urethral obstruction, a balanced electrolyte solution allows for more rapid correction of blood acid-base status within the first 12 h of fluid therapy.[17] Balanced solutions contain buffer molecules, being converted to bicarbonate after administration. ACE, which is included as a buffer in Normosol-R, is metabolised to acetyl coenzyme A in skeletal muscle and contributes to the alkalising properties of this fluid.[17] In humans, even small changes in pH can negatively impact various organ systems.[5] In a study on LAC- and ACE-containing fluids in human medicine, both balanced solutions preserved or increased the plasma bicarbonate.[5] In women who had undergone abdominal gynaecological surgery and received either a LAC- or ACE-containing solution no difference in hemodynamic parameters between both solutions were found.[5] Neither fluid appeared to be superior in balancing the acid-base status. The pH and BE values did not change in the group receiving ACE-containing fluids. A small but significant reduction in pH and bicarbonate was observed in women receiving LAC-containing solutions.[5] In children undergoing scoliosis surgery who were randomised to receive either 10 ml/kg/h ACE- or LAC-containing fluid intraoperatively, pH decreased in both groups, which was more pronounced in the LAC-containing fluid group.[23] The results of the acid-base status in this study may be influenced by anaesthesia, ventilation and changes in volume status due to blood loss.

In the present study, some cats had increased LAC concentrations at presentation though the mean LAC concentration of the study group was within the normal reference interval. Fluid deficits can lead to hypoperfusion. Hypoxic tissues produce energy through anaerobic glycolysis. This leads to increased hydrogen ion and LAC production.[12,24,25] Thus, hyperlactataemia often occurs with fluid deficits. Although cats with clinical signs of hypovolaemia were excluded, the possibility of type B hyperlactataemia, cryptic hypovolaemic shock or increased muscle activity during blood collection cannot be excluded. After rehydration, LAC decreased in both groups, most likely due to dilution from the intravenous fluid administration and improvement of underlying disease. In another study of dehydrated dogs rehydrated with either ACE- or LAC-containing fluids over 6–24 h, plasma LAC concentration decreased significantly in both groups, which was more substantial in the ACE-containing fluid group.[8] The larger decrease in the LAC level in the group receiving ACE-containing fluids was explained by the fact that no additional LAC was administered by the infusion solution in this group in contrast to the group with LAC-containing fluids.[8] A study comparing ACE- and LAC-containing fluids as shock boluses in dogs also concluded that the plasma LAC concentration decreased significantly only in the ACE-containing fluid group. The lack of reduction in LAC levels in the dogs receiving LAC-containing fluids was explained by the short period between the administration of fluids and blood LAC measurements, as the time was most likely insufficient to metabolise the LAC administered by the infusion solution.[12] LAC analysis in cats can be challenging as patient factors, such as stress or slow blood flow, can influence LAC values.[26] Results for the influence of LAC-containing fluids on plasma LAC concentration in humans also vary. A meta-analysis of 29 studies comparing the effects of ACE- and LAC-containing solutions in humans found increased plasma LAC concentration after the administration of LAC-containing solutions in 14 studies, while five studies found no difference between groups.[27]

Potassium concentration decreased after rehydration in the cats in both groups studied here. Potassium is the main intracellular cation and is important for cardiac and renal function, skeletal muscle metabolism and nerve conductibility. Its distribution throughout the intracellular and extracellular space is influenced by aldosterone, insulin, β-adrenergic agonists and bicarbonate.[1,8] In a study of 68 cats with naturally occurring urethral obstruction, 22 of which were initially hyperkalaemic, cats were randomised to receive 0.9% sodium chloride or an ACE- and gluconate-containing solution.[17] Potassium normalised similarly in both groups. The effect of fluid therapy with LAC-containing fluid and 0.9% sodium was evaluated in 10 cats with experimentally induced urethral obstruction. Although in the present study both solutions have a similar or slightly higher potassium content compared with feline plasma, the potassium concentration did not increase but decreased after infusion. As potassium is the main intracellular electrolyte, its distribution to the extracellular and intracellular space is influenced by various complex factors. Furthermore, the redistribution of the infused fluid into the interstitial space and cells is also associated with redistribution of the electrolytes.[10] As most cats refused food during rehydration, the decrease in blood potassium content can also be a consequence of anorexia. Despite the mild supraphysiologic potassium concentration in lactated Ringer's, it is usually inadequate to increase serum potassium concentration. In other countries, the potassium concentration of lactated Ringer's differs from that in Europe and therefore may lead to different results.

In the present study, other electrolytes, such as sodium, chloride and calcium, did not change significantly before and after rehydration. As cats with gastrointestinal diseases were included, the vomiting of gastric or intestinal content causes the loss of chloride, potassium, sodium and bicarbonate-containing fluids. These losses can lead to dehydration along with hyponatraemia, hypochloraemia and hypokalaemia.[19] Vomiting and diarrhoea usually result in isotonic dehydration;[4,19] therefore, a replacement fluid with sodium concentrations and osmolarity in the physiologic range should be selected.[4] Given the considerably lower sodium concentration in LAC, the trial was expected to show potential hyponatraemia after rehydration in cats in the LAC group. Similarly, Sterofundin ISO used in the ACE group contains higher chloride concentration compared to feline plasma and was expected to cause hyperchloraemia in patients in this study; however, after rehydration, no significant differences in both sodium and chloride concentration between the two groups were observed.[5] This lack of difference can be attributed to the body's ability to regulate and stabilise electrolyte levels efficiently, regardless of the chloride content in the rehydration fluids. This finding aligns with other studies, indicating that balanced solutions can maintain stable acid-base and electrolyte balance during and after fluid therapy.[8,23]

In the present study, glucose decreased in both study groups. Glucose increases are often stress-dependent in cats; therefore, it might have been elevated on arrival owing to transport, initial examination and blood collection and returned to normal later.

Limitations

A power analysis was performed to detect differences in sodium and chloride concentrations of 3 mmol/l. Furthermore, the assessment of dehydration was performed subjectively and is especially difficult in cats with cachexia and obesity and in cats with moist mucous membranes secondary to hypersalivation. However, dehydration was used as an indication for fluid therapy and was not a primary outcome measure. In addition, rehydration time was adapted to dehydration at the discretion of the clinician. Therefore, the individual amounts of fluids were different and influenced by ongoing losses. A standardised fluid therapy protocol using resuscitation fluids over a defined time at a defined fluid rate would be beneficial for further studies. Despite being clinically excluded, some states of hypovolaemia cannot be entirely excluded in the cats in the present study. To keep the patient population as uniform as possible, patients with hepatic dysfunction, cardiac diseases and other serious underlying diseases were excluded; therefore, the results of this study cannot be fully applied to critically ill cats.

Conclusions

Both ACE- and LAC-containing solutions equally improve acid-base status and decrease potassium and LAC concentration in cats after rehydration. Intravenous fluid therapy with ACE or LAC is an effective treatment for dehydration and ongoing losses. Both solutions are equally effective in this regard.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval The work described in this manuscript involved the use of non-experimental (owned or unowned) animals and procedures that differed from established internationally recognised high standards ('best practice') of veterinary clinical care for the individual patient. The study therefore had ethical approval from an established (or ad hoc) committee as stated in the manuscript.

Informed consent Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers, tissues and samples) for all procedure(s) undertaken (prospective or retrospective studies). No animals or people are identifiable within this publication, and therefore additional informed consent for publication was not required.

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