Renal and cardiorespiratory effects of treatment with lactated Ringer's solution or physiologic saline (0.9% NaCl) solution in cats with experimentally induced urethral obstruction

Marina G. M. C. M. Cunha, MSc; Gabrielle C. Freitas, MSc; Adriano B. Carregaro, PhD; Kleber Gomes, MSc; João P. M. C. M. Cunha, DVM; Diego V. Beckmann, MSc; Ney L. Pippi, PhD

Objective—To compare the renal and cardiorespiratory effects of IV treatment with lactated Ringer's solution (LRS) or physiologic saline (0.9% NaCl) solution (PSS) in severely decompensated cats with urethral obstruction (UO).

Animals—14 cats (4 cats were used only to establish infusion rates).

Procedures—An occluded urethral catheter was used to induce UO in each cat. After development of severe metabolic acidosis, hyperkalemia, and postrenal azotemia, the obstruction was relieved (0 hours); LRS or PSS (5 cats/group) was administered IV (gradually decreasing rate) beginning 15 minutes before and continuing for 48 hours after UO relief. Ten minutes before urethral catheter placement (baseline), at start of fluid therapy (SFT), and at intervals during fluid administration, various physical and clinicopathologic evaluations were performed.

Results—Metabolic acidosis was detected in the PSS-treated group at SFT and 2 hours after relief of UO and in the LRS-treated group only at SFT. The PSS-treated group had significantly lower blood pH and bicarbonate concentrations at 8 through 48 hours and lower base excess values at 2 through 48 hours, compared with the LRS-treated group. Hypocalcemia and hypernatremia were detected in the PSS-treated group at 2 and 12 hours, respectively. Absolute serum potassium and chloride concentrations did not differ significantly between groups at any time point.

Conclusions and Clinical Relevance—Treatment with LRS or PSS appeared to be safe and effective in cats with experimentally induced UO; however, LRS was more efficient in restoring the acid-base and electrolyte balance in severely decompensated cats with UO. (*Am J Vet Res* 2010;71:840–846)

Urethral obstruction is one of the most common emergencies involving the urinary tract in cats,[1] and approximately 12% of feline patients with UO have life-threatening metabolic derangements.[2] Cats with UO for more than 36 hours are considered to be severely affected.[3] Death may occur as a result of cardiopulmonary failure, hydroelectrolytic imbalance, or acute renal failure.[4] Hyperkalemia is considered to be the most common life-threatening complication associated with this condition[2] because it impedes the myocardial resting membrane potential, thereby initiating a depolarization blockade effect and reducing the electrical conductivity.[5,6]

In cats with UO, it is recommended that administration of fluids is commenced as soon as possible to correct the hydroelectrolytic balance and replace urinary losses due to dehydration and postobstructive diuresis.[1,7,8] Fluid therapy is the most important component involved in the stabilization of postrenal azotemia because it alleviates hyperkalemia, acidosis, and azotemia in most instances.[8] Inadequate replacement of fluids during the postobstructive period can delay resolution of these electrolyte, acid-base, and uremic disturbances[7] as well as cause development of renal lesions through hypoperfusion that results from hypovolemia.[4] Several authors have indicated that the preferred electrolyte solution for treatment of hyperkalemic animals is PSS,[7,9–12] despite its acidifying property. It is also recommended that use of LRS and other solutions that contain potassium should be avoided in patients with acute renal lesions because it is believed that their administration could cause or worsen hyperkalemia.[13] This effect is improbable with the use of LRS because that solution contains

Abbreviations

LRS	Lactated Ringer's solution
PSS	Physiologic saline (0.9% NaCl) solution
SFT	Start of fluid therapy
UO	Urethral obstruction

Received December 18, 2008.
Accepted May 29, 2009.
From Departamento de Clínica de Pequenos Animais, Centro de Ciências Rurais, Universidade Federal de Santa Maria, Santa Maria, RS 97105-900, Brazil.
Address correspondence to Dr. Maria Cunha (morimv@yahoo.com.br).

a very small quantity of potassium (4 mEq/L), compared with the quantity stored in the body.[13,14] In addition, the reestablishment of the urinary flow leads to a marked excretion of potassium, and the alkalinizing effect of LRS results in shift of K^+ ions to the intracellular space.[14]

The alkalinizing effect of LRS is attributable to the presence of lactate buffer, which is biotransformed into bicarbonate in the liver and helps to stabilize the acid-base balance. Treatment of the UO-related acid-base disturbance with sodium bicarbonate should be carried out with caution because the increase in blood pH will cause more calcium to bind to negatively charged proteins, which may further decrease the concentration of its ionized form.[2]

Administration of PSS causes hyperchloremic metabolic acidosis[15] and may cause renal vasoconstriction as a result of hyperchloremia[16] and acidemia[17]; this leads to a decrease in the glomerular filtration rate, which may reduce urinary output as well as possibly induce hyperkalemia through the cation exchange with H^+ ions.[4,18]

The purpose of the study reported here was to compare the renal and cardiorespiratory effects of IV treatment with LRS or PSS in severely decompensated cats with UO. Our hypothesis was that treatment with LRS would be more effective than treatment with PSS for stabilization of cats with UO during a 48-hour period following relief of obstruction.

Materials and Methods

Animals—Fourteen neutered male adult mixed-breed cats that were donated to the university were used in the study. Four cats were used in preliminary experiments to establish infusion rates; data from these cats were not included in the analyses. Ten cats (weight range, 3 to 5 kg) were used in the main study. Cats were housed in individual cages (16 × 22 × 28 inch) for a minimum adaptation period of 15 days. They were fed dry commercial food, and water was available ad libitum. The 10 cats were randomly allocated to receive either PSS[a] or LRS[b] (5 cats/group).

Experimental induction of UO—All experimental procedures were reviewed and approved by the Federal University of Santa Maria Animal Care and Use Committee. For each cat, anesthesia was induced with isoflurane in 100% oxygen by use of a facial mask. After aseptic preparation of the preputial and peripreputial areas with an aqueous solution of 1% chlorhexidine, urethral catheterization was performed with a 3.5F polypropylene catheter[c] lubricated with lidocaine gel. The urethral catheter was occluded with the cap of a peripheral venous catheter and fixed in the peripreputial area by use of a simple interrupted suture with 3-0 monofilament nylon. Each cat was allowed to recover from anesthesia following catheter placement.

Relief of UO—Cats were monitored for development of severe metabolic acidosis, hyperkalemia, and postrenal azotemia. During this monitoring period, venous blood samples (1 mL each) were collected for analysis of pH, BUN concentration, and serum creatinine and potassium concentrations. The interval between sample collections (2 to 4 hours or 12 hours) was adjusted on the basis of the blood pH (ie, used as evidence of development of metabolic acidosis). For each cat, relief of UO was performed when any 3 of 4 clinicopathologic criteria were met: venous pH < 7.2, BUN concentration > 200 mg/dL, serum creatinine concentration > 4.5 mg/dL, and serum potassium concentration > 6.5 mEq/L.

Fluid therapy—Once a cat developed severe metabolic acidosis, hyperkalemia, and postrenal azotemia, IV administration of LRS or PSS was commenced. The treatment was administered via a 22-gauge IV catheter placed specifically for fluid administration in a cephalic vein and maintained until completion of fluid therapy. Fifteen minutes after SFT, anesthesia was induced by use of propofol (5 mg/kg, IV). The cat remained anesthetized during relief of UO (designated as 0 hours) and urinary bladder washing (a period of approx 15 minutes), similar to a clinical situation. An aseptic technique was used to perform urinary bladder drainage; the occluded urethral catheter was replaced by a patent flexible polyvinyl chloride catheter.[d] The urinary bladder was flushed with cold LRS until the effluent was clear; the urethral catheter was then connected to a low-vacuum drainage system.[e] Each cat received fluid therapy for 48 hours after relief of obstruction (ie, total duration of fluid therapy was 48.25 hours). The low-vacuum drainage system was also used during this time. Having completed the urinary bladder drainage procedures, the cat was allowed to recover from anesthesia.

The infusion rate protocol used in the study was based on preliminary data obtained from 4 cats that were administered fluids at a rate of 60 and 40 mL/kg/h after relief of experimentally induced UO. These cats died as a result of fluid overload; data were not included in any of the study analyses. For treatment of the 10 cats in the main study, the infusion rate of either LRS or PSS was gradually decreased during the 48-hour period. The initial rate was 20 mL/kg/h from 0 to 6 hours; this rate was decreased to 15 mL/kg/h from 6 to 12 hours, 10 mL/kg/h from 12 to 24 hours, and 5 mL/kg/h from 24 to 48 hours.

Cats received meloxicam (0.1 mg/kg, IM, q 24 h) beginning the day that the occluded urethral catheter was placed and continuing up to 3 days after relief of UO. At the end of the study, all cats were donated to households.

Assessments—At predetermined time points, a blood sample (3 mL) was collected from a jugular vein of each of the 10 cats. Samples were used to assess venous blood pH, P_{CO_2}, P_{O_2}, bicarbonate concentration, base excess, anion gap, Hct, serum total protein and total plasma protein concentrations, BUN concentration, and serum sodium, chloride, total calcium, potassium, creatinine, and albumin concentrations.

Physical examination (including assessment of weight, heart rate, respiratory rate, and rectal temperature) was performed 10 minutes before urethral catheter placement (ie, prior to UO [baseline]), at SFT (ie, 15 minutes prior to relief of obstruction [obstruction relief performed at 0 hours]), and at 2, 4, 6, 8, 12, 24, and 48

hours after relief of UO. Venous blood gas and serum electrolyte analyses were performed at baseline, at SFT, and at 2, 8, 12, 24, and 48 hours. Measurements of Hct; serum concentrations of total protein, creatinine, and albumin; and BUN and total plasma protein concentrations were performed at baseline, at 24 hours after placement of the occluded urethral catheter, at SFT, and at the 2-, 8-, 12-, 24-, and 48-hour time points. Additionally, these assessments were performed at the 72-hour time point and at 7 days after relief of UO. Serum chloride concentration was corrected as recommended in the literature.[19]

The urine from low-vacuum drainage system was measured at 2, 4, 6, 8, 12, 24, and 48 hours to estimate the urinary output. After relief of obstruction, a syringe was attached to the urethral catheter for the collection of urine to perform urinalysis.

Statistical analysis—All analyses were performed with standard software.[f] A repeated-measures ANOVA was used followed by the Dunnett test to compare the mean of each variable (except urinary output) at the various time points with the baseline mean value within a group. Urinary output was compared among time points by use of the Tukey test. Between groups, values for each variable at each time point were compared by use of a Student *t* test. A value of $P < 0.05$ was considered significant. Results were expressed as mean ± SD.

Results

The method of inducing UO used in the cats was successful and resulted in development of clinical signs similar to those reported for cats with naturally occurring UO. In all cats, clinical signs of renal decompensation became evident 24 hours after initiation of UO. At the time of relief of UO, all 10 cats had hyperkalemia, azotemia, metabolic acidosis, and anorexia; 5 cats (2 from the PPS-treated group and 3 from the LRS-treated group) developed vomiting, which was not observed after relief of obstruction. The mean ± SD duration of UO before the clinicopathologic criteria for relief of obstruction were met was 43 ± 3 hours. Cats began to eat 4 (n = 2), 6 (7), and 8 (1) hours after relief of UO. Because food was not withheld from the cats before collection of blood samples, some serum and plasma samples were lipemic (cloudy and white appearance).

Weight, heart rate, respiratory rate, and rectal temperature did not differ significantly between the groups at any time point (**Table 1**). In the LRS-treated group, heart rate was decreased ($P < 0.05$) at SFT, compared with the baseline value. In both groups, rectal temperature was significantly decreased from the respective baseline value at that time point. With regard to weight, values did not change in either group except for a significant ($P < 0.05$) decrease from baseline value in the LRS-treated group at the 48-hour time point.

The PSS-treated group had a lower urinary output, compared with findings in the LRS-treated group, at almost all time points, although the difference was significant ($P < 0.05$) only at 8 hours (Table 1). All samples of urine collected at SFT appeared cloudy and bloody.

At 2, 8, 12, 24, 48, and 72 hours and at 7 days after relief of UO, values of Hct were significantly ($P < 0.05$) decreased in relation to baseline values in both

Table 1—Results of physical examination and urinary output in cats with experimentally induced UO that were treated IV with LRS (n = 5) or PSS (5) for 48 hours following relief of UO.

Time point	Treatment group	Weight (percentage of baseline value)	Heart rate (beats/min)	Respiratory rate (beats/min)	Rectal temperature (C°)	Urinary output (mL/kg/h)
Baseline	LRS	100 ± 0.0	208 ± 22	40 ± 6	38.7 ± 0.6	NM
	PSS	100 ± 0.0	196 ± 25	33 ± 9	38.6 ± 0.2	NM
SFT	LRS	99.5 ± 2.9	141 ± 75*	54 ± 21	37.5 ± 1.6*	NM
	PSS	97.9 ± 2.9	215 ± 61	48 ± 10	38.42 ± 1.0*	NM
2 h	LRS	99.3 ± 1.6	218 ± 43	41 ± 7	37.6 ± 2.5	14.8 ± 5.2[a]
	PSS	99.4 ± 3.2	244 ± 54	49 ± 20	38.4 ± 1.0	11.9 ± 3.1[ab]
4 h	LRS	99.7 ± 2.8	216 ± 19	57 ± 23	38.3 ± 1.7	15.8 ± 6.4[a]
	PSS	99.8 ± 4.5	210 ± 46	48 ± 22	38.2 ± 0.8	15.7 ± 7.7[a]
6 h	LRS	99.8 ± 3.8	207 ± 10	54 ± 10	37.7 ± 1.4	15.6 ± 5.2[a]
	PSS	100.7 ± 5.2	220 ± 29	43 ± 12	38.0 ± 0.4	14.4 ± 3.7[a]
8 h	LRS	99.5 ± 2.3	210 ± 19	55 ± 11	38.4 ± 0.7	17.5 ± 2.8†[a]
	PSS	101.6 ± 5.6	232 ± 39	45 ± 12	37.8 ± 0.6	12.0 ± 5.7[ab]
12 h	LRS	99.5 ± 2.8	208 ± 21	53 ± 12	38.6 ± 0.6	13.4 ± 3.3[a]
	PSS	102.2 ± 5.6	218 ± 27	48 ± 10	37.9 ± 0.9	13.0 ± 2.7[ab]
24 h	LRS	96.8 ± 1.4	202 ± 30	55 ± 25	38.8 ± 0.7	11.4 ± 1.2[ab]
	PSS	100.5 ± 4.7	198 ± 23	42 ± 6	38.6 ± 1.0	9.0 ± 3.5[ab]
48 h	LRS	96.1 ± 3.6*	191 ± 18	35 ± 5	39.2 ± 0.3	4.1 ± 1.5[b]
	PSS	99.0 ± 3.7	211 ± 20	46 ± 13	38.4 ± 1.1	4.2 ± 1.1[b]

Assessments were made 10 minutes before placement of an occluded urethral catheter (ie, prior to induction of UO [baseline]), at SFT (15 minutes before relief of UO [obstruction relief was designated as 0 h]), and at intervals during fluid administration (discontinued 48 hours after relief of UO [total duration, 48.25 h]). The initial infusion rate was 20 mL/kg/h from 0 to 6 hours; this rate was decreased to 15 mL/kg/h from 6 to 12 hours, 10 mL/kg/h from 12 to 24 hours, and 5 mL/kg/h from 24 to 48 hours. Data are reported as mean ± SD. In cats, reference ranges for the variables of interest are as follows: heart rate, 140 to 230 beats/min; respiratory rate, 20 to 40 breaths/min; rectal temperature, 37.8° to 39.5°C; and urinary output, 1 to 2 mL/kg/h.

*Within a group, value was significantly ($P < 0.05$) different from the baseline value for this variable. †For a given variable at a given time point, value in the LRS-treated group was significantly different from the value in the PSS-treated group.

NM = Not measured.

[ab]For urinary output, identical superscript letters indicate values that do not differ significantly (regardless of group or time point).

groups (**Table 2**). Total plasma protein concentration was significantly less than the respective baseline value at 8 hours in the LRS- and PSS-treated groups (both $P < 0.05$) and at 12 hours ($P < 0.05$) in the LRS-treated group.

Concentrations of BUN and serum total protein, creatinine, and albumin were assessed at baseline, at 24 hours after placement of the occluded urethral catheter, at SFT, and at the 0-, 2-, 8-, 12-, 24-, and 48-hour time points (Table 2). These variables did not differ significantly between groups at any time point. In both groups, BUN and creatinine concentrations were significantly ($P < 0.05$) increased from the respective baseline value at SFT and 2 and 8 hours. A decrease in total protein concentration was detected at 2, 8, and 12 hours in both groups.

Values of blood pH, P_{CO_2}, P_{O_2}, and bicarbonate concentration were assessed at baseline, at SFT, and at the 2-, 8-, 12-, 24-, and 48-hour time points (**Table 3**). Compared with the LRS-treated group, the PSS-treated group had significantly ($P < 0.05$) lower values for blood pH and base excess at 8, 12, 24, and 48 hours; bicarbonate concentration was significantly ($P < 0.05$) lower at all time points from 2 to 48 hours. In the PSS-treated group, blood pH was significantly ($P < 0.05$) decreased from baseline value at SFT and 2 hours and did not return to reference values until the end of the blood gas evaluation (at 48 hours). In the LRS-treated group, blood pH was significantly ($P < 0.05$) decreased from baseline value only at SFT. In that group, values for P_{CO_2} were significantly ($P < 0.05$) reduced in relation to baseline values at SFT and 2, 8, 12, and 24 hours, but there was no difference in P_{O_2} values at any time point. Values of bicarbonate concentration were significantly ($P < 0.05$) decreased from the respective baseline value at all time points from SFT to 24 hours in the PSS-treated group and at SFT and 2 hours in the LRS-treated group. Base excess values were significantly ($P < 0.05$) decreased from baseline at all time points from SFT to 48 hours in the PSS-treated group and from SFT to 2 hours in the LRS-treated group.

Values of serum potassium, sodium, chloride, and total calcium concentrations and anion gap were also assessed at baseline, at SFT, and at the 2-, 8-, 12-, 24-, and 48-hour time points (Table 3). There was no significant difference in serum potassium or chloride concentration between the groups at any time point. Potassium concentration was increased significantly from the respective baseline value at SFT in both groups; the increase was detected only at 2 hours in the LRS-treated group. Hypokalemia (< 3.5 mEq/L) was evident in 7 of the 10 cats (3 in the LRS-treated group and 4 in the PSS-treated group) at 24 hours after SFT and in only 1 cat in the PSS-treated group at the 48-hour time point. Ventroflexion of the neck was not noticeable in those cats at those time points; thus, hypokalemia was not associated with any overt clinical sign in those cats.

After relief of UO, the PSS-treated group had higher serum sodium concentrations than the LRS-treated group, although the difference was significant only at the 12-hour time point. At SFT, serum sodium concentration was significantly ($P < 0.05$) decreased in relation to baseline values in each group; however, values at SFT were not less than the lower reference limit for cats. Serum sodium concentration returned to baseline value at 2 hours in the PSS-treated group and at 8 hours in the LRS-treated group. Compared with the LRS-treated group, the PSS-treated group had significantly lower serum total calcium concentrations at 2, 8, and 12 hours. For both groups, total calcium concentration was significantly ($P < 0.05$) decreased from baseline only at 2 hours in the PSS-treated group. Anion gap val-

Table 2—Results of hematologic and biochemical analyses in cats with experimentally induced UO that were treated IV with LRS (n = 5) or PSS (5) for 48 hours following relief of UO.

Time point	Treatment group	Hct (%)	Plasma total protein (g/dL)	BUN (mg/dL)	Serum creatinine (mg/dL)	Serum albumin (g/dL)	Serum total protein (g/dL)
Baseline	LRS	40 ± 5	9.1 ± 0.7	47 ± 19	1.3 ± 0.3	1.7 ± 0.3	8.4 ± 0.5
	PSS	41 ± 5	8.6 ± 1.0	44 ± 14	1.1 ± 0.1	1.7 ± 0.2	7.7 ± 1.4
24 h after initiation of UO	LRS	40 ± 4	8.8 ± 0.7	114 ± 56	1.9 ± 0.8	2.0 ± 0.2	9.5 ± 0.4
	PSS	41 ± 8	8.6 ± 0.6	125 ± 82	1.4 ± 0.4	1.8 ± 0.2	7.8 ± 1.2
SFT	LRS	37 ± 7	8.3 ± 1.3	278 ± 121*	7.5 ± 1.3*	1.8 ± 0.3	7.6 ± 0.6
	PSS	35 ± 8	8.6 ± 1.2	251 ± 106*	6.7 ± 2.0*	1.8 ± 0.2	7.1 ± 1.4
2 h	LRS	32 ± 6*	8.0 ± 0.7	256 ± 112*	5.9 ± 1.2*	1.6 ± 0.4	7.0 ± 1.1*
	PSS	29 ± 3*	7.6 ± 1.4	219 ± 83*	5.5 ± 2.3*	1.5 ± 0.1	6.0 ± 1.1*
8 h	LRS	27 ± 3*	7.5 ± 0.5*	143 ± 76*	3.1 ± 1.0*	1.5 ± 0.3	6.9 ± 1.3*
	PSS	24 ± 4*	7.0 ± 1.1*	134 ± 48*	3.6 ± 2.3*	1.3 ± 0.2	5.7 ± 0.9*
12 h	LRS	27 ± 5*	7.8 ± 0.7*	97 ± 58	2.0 ± 0.7	1.5 ± 0.3	6.8 ± 0.7*
	PSS	25 ± 6*	7.5 ± 1.3	115 ± 64	2.5 ± 1.7	1.5 ± 0.3	6.0 ± 1.1*
24 h	LRS	26 ± 3*	8.1 ± 0.4	44 ± 24	1.3 ± 0.2	1.6 ± 0.4	7.3 ± 1.2
	PSS	26 ± 4*	8.3 ± 1.4	64 ± 40	1.4 ± 0.6	1.6 ± 0.3	6.4 ± 0.6
48 h	LRS	26 ± 8*	7.9 ± 0.5	47 ± 14	1.3 ± 0.2	1.5 ± 0.4	6.9 ± 1.0
	PSS	26 ± 3*	8.4 ± 1.2	49 ± 24	1.3 ± 0.2	1.7 ± 0.2	6.6 ± 0.6
72 h	LRS	29 ± 6*	8.6 ± 0.2	68 ± 33	1.5 ± 0.3	1.8 ± 0.4	7.6 ± 0.8
	PSS	27 ± 3*	9.0 ± 0.9	71 ± 50	1.5 ± 0.2	1.8 ± 0.2	8.2 ± 1.4
7 d	LRS	30 ± 4*	8.6 ± 0.9	58 ± 12	1.6 ± 0.3	1.8 ± 0.2	8.0 ± 0.5
	PSS	32 ± 6*	8.7 ± 0.7	52 ± 9	1.2 ± 0.2	1.8 ± 0.2	8.3 ± 0.8

Data are reported as mean ± SD. In cats, reference ranges for the variables of interest are as follows: Hct, 24% to 45%; plasma protein concentration, 6 to 8 g/dL; BUN concentration, 20 to 65 mg/dL; serum creatinine concentration, 0.8 to 1.9 mg/dL; serum albumin concentration, 2.1 to 3.2 g/dL; and serum total protein concentration, 5.4 to 7.8 g/dL.
See Table 1 for key.

Table 3—Results of venous blood gas and electrolyte analyses in cats with experimentally induced UO that were treated IV with LRS (n = 5) or PSS (5) for 48 hours following relief of UO.

Time	Treatment group	Blood pH	P_{CO_2} (mm Hg)	P_{O_2} (mm Hg)	Bicarbonate (mmol/L)	Base excess	Potassium (mEq/L)	Sodium (mEq/L)	Chloride (mmol/L)	Total calcium (mg/dL)	Anion gap (mmol/L)
Baseline	LRS	7.31 ± 0.02	36.1 ± 5.9	39.1 ± 8.6	18.1 ± 2.6	–7.3 ± 2	3.7 ± 0.4	159 ± 2	118.2 ± 3.9	10.8 ± 0.4	27.0 ± 3.7
	PSS	7.31 ± 0.02	38.5 ± 3.9	37.2 ± 5.5	18.9 ± 2.7	–6.9 ± 2.7	4.1 ± 0.4	159 ± 2	118.6 ± 3.0	9.5 ± 0.9	26.0 ± 4.4
SFT	LRS	7.16 ± 0.11*	32.1 ± 5.1	41.9 ± 9.5	11.4 ± 2.7*	–16.1 ± 4.1*	6.7 ± 1.0*	152 ± 5*	123.3 ± 6.5	9.4 ± 1.3	23.8 ± 9.4
	PSS	7.19 ± 0.06*	31.4 ± 4.8*	38.4 ± 3.9	11.6 ± 2.5*	–15.3 ± 3.2*	6.5 ± 1.3*	150 ± 4*	124.5 ± 5.0	9.2 ± 0.6	20.0 ± 1.0
2 h	LRS	7.24 ± 0.10	33.5 ± 7.7	35.9 ± 3.8	13.8 ± 0.7*†	–12.6 ± 2.3*	5.0 ± 1.0*	153 ± 6*	120.3 ± 7.0	10.0 ± 1.5†	26.2 ± 9.5
	PSS	7.22 ± 0.05*	28.6 ± 2.4*	42.1 ± 7.1	11.3 ± 2.0*†	–15.0 ± 2.7*	5.0 ± 1.1	155 ± 3	125.0 ± 6.0	7.7 ± 0.3*†	22.0 ± 8.9
8 h	LRS	7.34 ± 0.05†	30.8 ± 5.7	38.0 ± 5.7	16.1 ± 1.5†	–8.7 ± 1.1†	3.9 ± 0.4	155 ± 4	122.0 ± 4.8	10.5 ± 1.5†	21.3 ± 6.2
	PSS	7.25 ± 0.04†	29.8 ± 2.9*	41.3 ± 8.9	12.8 ± 1.4*†	–13.2 ± 1.6*†	4.1 ± 0.3	158 ± 3	119.9 ± 4.0	8.4 ± 1.0†	29.4 ± 5.9
12 h	LRS	7.36 ± 0.05†	31.8 ± 7.0	39.2 ± 4.3	17.5 ± 2.5†	–7.8 ± 1.9†	3.9 ± 0.3	155 ± 8†	121.5 ± 3.8	10.8 ± 1.9†	19.9 ± 9.0†
	PSS	7.26 ± 0.01†	26.1 ± 2.7*	43.3 ± 2.8	11.5 ± 1.1*†	–14.0 ± 0.9*†	3.8 ± 0.3	163 ± 3†	118.4 ± 3.9	8.7 ± 0.5†	36.9 ± 6.1†
24 h	LRS	7.37 ± 0.03†	35.7 ± 5.5	35.4 ± 4.8	20.0 ± 2.4†	–4.6 ± 1.8†	3.4 ± 0.2	156 ± 4	119.8 ± 4.7	10.4 ± 1.7	19.3 ± 7.0
	PSS	7.26 ± 0.05†	32.9 ± 4.56*	40.6 ± 4.9	14.0 ± 3.4*†	–11.6 ± 3.7*†	3.5 ± 0.4	160 ± 2	119.9 ± 1.9	9.0 ± 0.8	30.0 ± 6.2
48 h	LRS	7.37 ± 0.03†	33.5 ± 2.3	36.5 ± 9.0	18.6 ± 0.7†	–5.9 ± 1.2†	3.8 ± 0.4	157 ± 3	120.2 ± 1.5	10.8 ± 1.3	21.8 ± 3.4
	PSS	7.25 ± 0.02†	38.5 ± 4.9	36.4 ± 5.8	15.6 ± 2.6*†	–10.8 ± 2.0*†	3.8 ± 0.3	159 ± 8	118.0 ± 4.8	9.8 ± 0.7	30.7 ± 10.4

Data are reported as mean ± SD. In cats, reference ranges for the variables of interest are as follows: blood pH, 7.28 to 7.41; P_{CO_2}, 32.7 to 44.7 mm Hg; P_{O_2}, 41 to 51 mm Hg; serum bicarbonate concentration, 18.0 to 23.2 mmol/L; base excess, –6 to 1; serum potassium concentration, 3.5 to 4.5 mEq/L; serum sodium concentration, 149 to 161 mEq/L; serum chloride concentration, 117 to 123 mmol/L; serum total calcium concentration, 8.4 to 11.0 mg/dL; and anion gap, 13 to 27 mmol/L.
See Table 1 for key.

ues differed significantly ($P < 0.05$) between the groups only at 12 hours.

Discussion

In the cats in the present study, clinical signs of renal decompensation became evident 24 hours after initiation of complete UO, as has been reported previously.[4] The signs were similar to those reported for cats with naturally occurring UO.[2] The interval between initiation of obstruction and clinical decompensation in the study cats varied, which is in accordance with reports[3,20] that the intensity of electrolyte, acid-base, and uremic changes is individual and depends on a number of factors, including the general health of the animal and the duration and degree of UO.

In an attempt to stabilize the decompensated cats, the procedures used in the present study included provision of fluid therapy for 15 minutes before the cats were anesthetized for relief of UO, as recommended.[8] A gradual reduction in the rate of infusion of each crystalloid solution was carried out over 48 hours, as suggested in the veterinary medical literature.[1,8] In cats, cardiotoxicosis associated with UO promotes fluid overload after the administration of small quantities of fluids.[7] This alteration was not observed in our study, indicating that the rates of administration used were adequate.

In 1 study,[g] ECG alterations were observed in 4 cats with UO, despite the fact that their serum potassium concentrations were < 5.5 mEq/L; thus, it was concluded that potassium is not the only electrolyte responsible for the ECG alterations observed in cats with UO and that other electrolytes (eg, magnesium and ionized calcium) and acidemia may be influential. Because hemodynamic destabilization is multifactorial in cases of UO, 4 clinicopathologic variables were used to determine the time for relief of UO in cats of the present study.

In the cats in the LRS-treated group, heart rate immediately before SFT was decreased, compared with the baseline value determined before induction of UO. This effect was due to hyperkalemia and metabolic acidosis.[6] The hypothermia (temperature was assessed per rectum) in the LRS-treated group at this time point was associated with uremia, which reduces the thermoregulation set point in the hypothalamus, and with low rectal perfusion, which results from cardiac dysfunction and hypovolemia.[2]

In both groups, urinary output was significantly increased at all time points evaluated, compared with the reference values for cats (1 to 2 mL/kg/h); this was a result of the increase in renal intratubular hydrostatic pressure[7] and postobstructive diuresis (which is a mechanism to maintain the electrolytic balance and increase the excretion of metabolites retained during UO[4]). Under normal conditions, the increase in blood volume leads to natriuresis,[21,22] and the magnitude of excretion of sodium is a good indicator of renal blood flow.[23] The cats in the PSS-treated group were not dehydrated at the 12-hour time point; therefore, there would be no explanation for the retention of sodium in that group. In addition, the PSS-treated group had lower urinary output in general than the LRS-treated group. It can be suggested that this higher retention of sodium and water in the PSS-treated group may be attributable to comparatively less renal perfusion caused by metabolic acidosis because low renal perfusion is the greatest stimulant of the renin-angiotensin system.[21,22] Angiotensin II, in turn, causes renal vasoconstriction followed by retention of sodium and water. This mechanism occurs in an attempt to reverse hypotension by increasing blood volume and producing an increase in renal blood flow.[23]

During the study, the cats in both groups had some weight loss (compared with baseline values) as a result of protein catabolism associated with the state of anorexia produced by discomfort and uremia.[3] After the 4-hour time point until the 48-hour time point, the mean weight of cats in the LRS-treated group was lower than the weight of cats in the PSS-treated group; however, no differences in weight were observed between the groups, and only the LRS-treated group had a significant decrease from the baseline value at 48 hours. It can be suggested that the difference in weight between

groups was associated with the lower urinary output of the PSS-treated group at 8 hours, which led to subsequent retention of fluid. Greater weight and lower urinary output were observed in people who received 2 L of PSS, compared with findings in people who received LRS.[24] In that study, excretion of urinary sodium in the PSS-treated group was significantly lower, despite the fact that LRS has a lower concentration of sodium than does PSS.

In both groups, Hct was significantly decreased from the baseline value at 8, 12, 24, 48, and 72 hours and at 7 days after relief of UO. This was likely caused by hematuria (a result of rupture of vessels subsequent to vesical hyperdistension[7]) as well as hemodilution (a consequence of fluid therapy) and the cumulative volume of blood collected for assessments. Gradual changes in serum total protein and plasma total protein concentrations were observed in both groups; a general pattern of decreasing values was evident in the period of 2 to 12 hours, which was caused by anorexia, proteinuria (associated with hematuria),[3] and, probably, hemodilution and blood sample collection.

The BUN and creatinine concentrations significantly increased, compared with baseline values, from SFT through the 8-hour time point as a result of progressive reduction of the glomerular filtration rate 24 hours after complete UO.[4] The BUN and creatinine concentrations decreased to within reference ranges 24 hours after SFT; this was a shorter interval than that reported previously[18] (normalization at 48 to 72 hours after SFT). This difference between study findings may be due to differing infusion rates.

In the PSS-treated group, P_{CO_2} was decreased from baseline value at all time points from SFT to 24 hours. This was attributed to metabolic acidosis, which results in increased CO_2 elimination as an attempt to stabilize blood pH. This also causes a decrease in the CO_2 concentration in tissues and, consequently, a decrease in P_{CO_2}.[22] The significant difference in blood bicarbonate concentration between the groups throughout the period of fluid therapy after UO relief (2 to 48 hours) was likely due to the absence of bicarbonate precursors in the PSS. A decrease in both blood pH and base excess occurred in both groups because of an accumulation of H^+ ions, lactate, and other metabolic acid wastes caused by a marked decrease in the glomerular filtration rate.[4] After relief of UO, there is a return of glomerular filtration and these acids are excreted and bicarbonate and other buffers normalize blood pH and base excess. Because the PSS does not contain a bicarbonate precursor and has a pH value less than that of blood, blood pH in the PSS-treated group was not within reference range until the end of the blood gas evaluations (at 48 hours).

Hyperkalemia (serum potassium concentration > 4.5 mEq/L) was detected at SFT and 2 hours in both groups of cats. This change was due to decreased renal excretion of K^+ ions as a result of reabsorption of K^+ ions from the injured vesical mucous membranes and the cation exchange with H^+ ions that occurred with acidosis.[2] Although acidosis theoretically induces hyperkalemia,[4,18] there was no significant increase from baseline in serum potassium concentration in the PSS-treated group at 8 hours. Hypokalemia (serum potassium concentration < 3.5 mEq/L) detected at 24 and 48 hours was a result of intense diuresis.[1,22]

Initial hyponatremia developed as a result of anorexia and vomiting.[3] Considering that the cats were dehydrated (which led to the release of ADH that resulted in water and sodium retention), the sodium deficit was probably even greater than that indicated by the serum sodium concentration data.

Hypocalcemia was not evident at SFT, which suggested that dehydration may have masked the decrease in serum calcium concentration; at 2 hours after relief of UO, serum calcium concentration in the PSS-treated group was significantly decreased, compared with the baseline value. Although the total calcium concentration is not tightly correlated with ionized calcium concentration in patients with decreased renal function, decreased total calcium concentration is a good indicator of low ionized calcium concentration.[25,26] The significant difference in total calcium concentration between the groups at 2, 8, and 12 hours was attributable to the fact that LRS contains 3 mmol of calcium/L, whereas PSS does not contain this ion.

Because food was not withheld from the cats for 12 hours before blood samples were collected, the serum derived from some samples was lipemic, which increases the serum calcium concentration.[27] Therefore, some cats in both groups had serum total calcium concentrations that were greater than the upper reference limit for the species.

Chloride transport is closely related to sodium and fluid transport as well as to the cellular acid-base balance.[19] Although hypernatremia developed in the PSS-treated group, there was no elevation from baseline in serum chloride concentration at any time point, possibly because Cl^- ions are not always reabsorbed with Na^+ ions, especially in specific regions of the nephron.[19] Hyperchloremic metabolic acidosis can develop following administration of PSS in humans.[28,29] However, in our study of cats that received fluid therapy for an extended period subsequent to relief of UO, an elevation in serum chloride concentration was not observed. The reason that hyperchloremia was not detected in the study cats may be attributable to the difference in the reference ranges between species (117 to 123 mEq/L in cats and 100 to 106 mEq/L in humans); on that basis, either the amount of chloride from the PPS is not sufficient to increase the serum chloride concentration in cats or UO causes an alteration in the mechanism of tubular reabsorption of this ion, leading to increased excretion. In the cats of the PSS-treated group, anion gap was greater than the baseline value (albeit not significantly) through the latter part of the study period (ie, 8 to 48 hours), indicating normochloremic metabolic acidosis. Thus, cats with UO that are treated with PSS have metabolic acidosis not because of an elevation in serum chloride concentration, but rather because of a decrease in bicarbonate concentration.

Because serum chloride concentration did not increase from baseline in either group of cats in the present study, it can be suggested that metabolic acidosis caused by the administration of PSS developed because of the lack of a bicarbonate precursor, thereby leading to dilution or lack of replenishment of serum con-

centrations of bicarbonate used in the stabilization of plasma. Humans with moderate renal lesions associated with metabolic acidosis may not develop serious clinical signs; however, severe renal lesions require simultaneous management of multiple clinical problems[30] because the decompensation may lead to numerous hemodynamic abnormalities.

On the basis of the results of the present study, it can be concluded that both solutions used as fluid therapy were effective in renal and cardiopulmonary stabilization in cats following relief of experimentally induced UO. However, LRS was more efficient in acid-base and electrolyte stabilization. The IV infusion of large volumes of LRS appears to be a safe treatment option in cats with UO and does not cause further increase in serum potassium concentration nor impedes its normalization to reference range values.

a. Cloreto de sódio 0.9%, Texon, Viamão, RS, Brazil.
b. Ringer com lactato de sódio, Aster Produtos Médicos Ltda, Sorocaba, Brazil.
c. Sovereign, 3.5 tom cat catheter, open end, Sherwood Medical, St Louis, Mo.
d. Sonda uretral, 04 F, Mark Méd, Bragança Paulista, SP, Brazil.
e. Biovac, Bional Indústria Biomédica, Recife, PE, Brazil.
f. GraphPad Prism 4, Graph Pad Software Inc, San Diego, Calif.
g Horta PVP. *Clinical, laboratorial and electrocardiografic abnormalities in cats with urethral obstruction.* MS thesis, Department of Veterinary Clinic, São Paulo University, São Paulo, Brazil, 2007.

References

1. Rieser TM. Urinary tract emergencies. *Vet Clin North Am Small Anim Pract* 2005;35:359–373.
2. Lee JA, Drobatz KJ. Characterization of the clinical characteristics electrolytes, acid-base, and renal parameters in male cats with urethral obstruction. *J Vet Emerg Crit Care* 2003;13:227–233.
3. Finco DR, Cornelius LM. Characterization and treatment of water, electrolyte, and acid-base imbalances of induced urethral obstruction in the cat. *Am J Vet Res* 1977;38:823–830.
4. Bartges JW, Finco DR, Polzin DJ, et al. Pathophysiology of urethral obstruction. *Vet Clin North Am Small Anim Pract* 1996;26:255–264.
5. Schaefer TJ, Wolford RW. Disorders of potassium. *Emerg Med Clin North Am* 2005;23:723–747.
6. Malouin A, Milligan JA, Drobatz KJ. Assessment of blood pressure in cats presented with urethral obstruction. *J Vet Emerg Crit Care* 2007;17:15–21.
7. Polzin DJ, Osborne CA, Bartges JW. Management of postrenal azotemia. *Vet Clin North Am Small Anim Pract* 1996;26:507–513.
8. Hostutler RA, Chew DJ, DiBartola SP. Recent concepts in feline lower tract disease. *Vet Clin North Am Small Anim Pract* 2005;35:147–170.
9. Schaer M. Disorders of potassium metabolism. *Vet Clin North Am Small Anim Pract* 1982;12:399–409.
10. Willard MD. Disorders of potassium homeostasis. *Vet Clin North Am Small Anim Pract* 1989;19:241–263.
11. McLoughlin MA. Surgical emergencies of the urinary tract. *Vet Clin North Am Small Anim Pract* 2000;30:581–601.
12. Norman BC, Côté E, Barrett KA. Wide-complex tachycardia associated with severe hyperkalemia in three cats. *J Feline Med Surg* 2006;8:372–378.
13. O'Malley CMN. Intravenous fluids and renal failure. *Transf Altern Transf Med* 2003;5:416–423.
14. Drobatz KJ, Cole SG. The influence of crystalloid type on acid-base and electrolyte status of cats with urethral obsruction. *J Vet Emerg Crit Care* 2008;18:355–361.
15. Biondo AW, de Morais HA. Chloride: a quick reference. *Vet Clin North Am Small Anim Pract* 2008;38:459–465.
16. Wilcox CS. Regulation of renal blood flow by plasma chloride. *J Clin Invest* 1983;71:726–736.
17. Gauthier PM, Szerlrip HM. Metabolic acidosis in the intensive care unit. *Crit Care Clin* 2002;18:289–308.
18. DiBartola SP. Management of hypokalaemia and hyperkalaemia. *J Feline Med Surg* 2001;3:181–183.
19. De Moraes HA, Biondo AW. Distúrbios relacionados ao cloro: hiper e hipocloremia. In: DiBartola SP, ed. *Anormalidades de fluidos eletrólitos e equilíbrio ácido-básico na clínica de pequenos animais.* 3rd ed. São Paulo, Brazil: Roca, 2007;77–86.
20. Osborne CA, Kruger JM, Lulich JP, et al. Medical management of feline urethral obstruction. *Vet Clin North Am Small Anim Pract* 1996;26:483–498.
21. Rose BD. Renal circulation and glomerular filtration rate. In: *Clinical physiology of acid-base and electrolyte disorders.* 4th ed. New York: McGraw-Hill Book Co, 1994;20–65.
22. DiBartola SP. Participação dos íons fortes nos distúrbios ácido-básicos. In: DiBartola SP, ed. *Anormalidades de fluidos eletrólitos e equilíbrio ácido-básico na clínica de pequenos animais.* 3rd ed. São Paulo, Brazil: Roca, 2007;295–306.
23. Nashat FS, Tappin JW, Wilcox CS. The renal blood flow and the glomerular filtration rate of anaesthetized dogs during acute changes in plasma sodium concentration. *J Physiol* 1976;256:731–745.
24. Reid F, Lobo DN, Williams RN, et al. (Ab)normal saline and physiological Hartmann's solution: a randomized double-blind crossover study. *Clin Sci (Lond)* 2003;104:17–24.
25. Drobatz KJ, Hughes D. Concentration of ionized calcium in plasma from cats with urethral obstruction. *J Am Vet Med Assoc* 1997;211:1392–1395.
26. Drobatz KJ, Ward C, Graham P, et al. Serum concentrations of parathyroid hormone and 25-OH vitamin D_3 in cats with urethral obstruction. *J Vet Emerg Crit Care* 2005;15:179–184.
27. Schenck PA, Chew DJ, Nagode LA, et al. Distúrbios relacionados ao cálcio: hiper e hipocalcemia. In: DiBartola SP, ed. *Anormalidades de fluidos eletrólitos e equilíbrio ácido-básico na clínica de pequenos animais.* 3rd ed. São Paulo, Brazil: Roca, 2007;115–183.
28. Scheingraber S, Rehm M, Sehmisch C, et al. Rapid saline infusion produces hyperchloremic acidosis in patients undergoing gynecologic surgery. *Anesthesiology* 1999;90:1265–1270.
29. Ho AM, Karmakar MK, Contardi LH, et al. Excessive use of normal saline in managing traumatized patients in shock: a preventable contributor to acidosis. *J Trauma* 2001;51:173–177.
30. Prough DS. Physiologic acid-base and electrolyte changes in acute and chronic renal failure patients. *Anesthesiol Clin North America* 2000;18:809–833.