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Alkalinizing Effect of Sodium Acetate Administered by Ororuminal Route in Adult Cattle

Paulo C. A. Amorim[1], Fabrício L. Valente[1], Samuel R. Alves[1], Felipe S. Mattos[1], Caio M. Costa[1], Lorena C. Monteiro[1], Marcel F. B. Avanza[1], Raffaella B. C. Teixeira[1], João V. M. Mota[1], Nathalia S. Rosse[1], Júlio A. N. Lisbôa[2], Hélio C. Manso Filho[3], Elizabeth H. Melo[4], José D. Ribeiro Filho[1*]

[1]Department of Veterinary, Federal University of Viçosa, Viçosa, Minas Gerais, Brazil
[2]Department of Veterinary Clinics, State University of Londrina, Londrina, Paraná, Brazil
[3]Zootecnics Department, Federal Rural University of Pernambuco, Recife, Pernambuco, Brazil
[4]Veterinary Medicine Course, Federal University of Roraima, Boa Vista, Roraima, Brazil
Email: *jdribeirofilho@gmail.com

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Abstract

This study aimed to evaluate the alkalizing potential of sodium acetate administered via ororuminal tubing in adult cattle. Six clinically healthy crossbred cows, with an average age of 5 years and average weight of 500 kg were used in a 6 × 2 crossover design, with seven-day intervals between treatment cycles, and each animal receiving both treatments. The animals were distributed into two treatments as described: TREAT1g: 1 g·kg^{-1} of sodium acetate diluted in 5 liters of water administered by ororumenal route; TREAT2g: 2 g·kg^{-1} of sodium acetate diluted in 5 liters of water administered by ororumenal route. The physical examination included measuring rectal temperature, heart rate, respiratory rate, and ruminal motility. The following biochemical and hematological parameters were measured: packed cell volume, plasma proteins, serum sodium, potassium, chloride, calcium, magnesium, phosphorus, urea, creatinine, and serum osmolarity; plasma glucose and lactate; and blood gas analysis (pH, pCO_2, $ctCO_2$, HCO_3^-, and BE). The anion gap and SID_3 were calculated, while urine pH, sodium, potassium, chloride, calcium, and magnesium were measured. In animals from the TREAT1g and TREAT2g groups, decreases were recorded in ruminal motility, serum potassium and total calcium, urinary chloride, calcium, and magnesium, while increases were detected in serum and urinary Na^+ values, plasma glucose, pH, and osmolarity of ruminal fluid, blood pH, HCO_3^-, $ctCO_2$, BE, SID_3, and urine pH. Conversely, increases in serum creatinine and osmolarity values were recorded only in an-

imals from the TREAT2g. In conclusion, both treatments caused metabolic alkalosis by strong ions and a slight increase in the pH value of the ruminal fluid.

Keywords

Alkalizing, Metabolic Acidosis, Hyperchloremia, Ruminal Fluid

1. Introduction

In adult cattle, infectious processes, gastrointestinal and metabolic diseases are common and may cause alterations in the hydroelectrolytic and acid-base balance. Among these, metabolic acidosis can be highlighted, which in this species is usually treated with the administration of sodium bicarbonate intravenously or ororuminally, and oral magnesium hydroxide [1]-[4].

Despite the variety of substances with alkalizing potential, sodium bicarbonate and magnesium hydroxide remain the main options to be used via ororuminal route in adult cattle. However, when they are administered by this route, in addition to the systemic alkalizing effect, alkalization of the digestive system occurs, leading to changes in rumen fluid pH and resident microbiota [4].

The administration of sodium acetate via ororuminal or nasoruminal routes in adult cattle has received little attention as an alkalizing agent to replace sodium bicarbonate and magnesium hydroxide. Since acetate exhibits alkalizing potential after metabolization in specific organs such as the liver and muscles [5], this means that this compound primarily exerts systemic buffering with a low capacity to alter ruminal pH.

In adult horses, Waller & Lindinger [6] described that sodium acetate administered nasogastrically can be applied as an alkalizing agent. Currently, sodium acetate has been used as a component of enteral electrolyte solutions to exert an effect on alkaline reserves in calves, foals, and adult horses [7]-[10]. However, in adult cattle, the effects of this metabolizable base have not yet been defined.

Therefore, this study aims to evaluate the effects of sodium acetate administered intraruminally by bolus at different doses on ruminal fluid, biochemical profile, acid-base and urinary balance, as well as packed cell volume, total protein, and physiological parameters in adult cattle. It is hypothesized that both doses of sodium acetate will influence alkaline reserves without causing alkalinization of the ruminal fluid.

2. Materials and Methods

The experimental procedures were approved by the ethics committee in animal use of Universidade Federal de Viçosa (CEUA/UFV process number 882/2019) following the guidelines of Brazilian legislation edited by the National Council for

the Control of Animal Experimentation (CONCEA). The experimental trial was conducted in the municipality of Viçosa, Minas Gerais, at an altitude of approximately 640 meters, latitude 20˚45'14"S and longitude 42˚52'54"W.

Study Design: Six crossbred cows were used in this study. The cows were multiparous, aged between 3 and 6 years, with an average body weight of 500 kg ± 52 kg, and were not lactating during the experimental period. They underwent physical examination, blood count, biochemical analysis, and urinalysis prior to the experiment period, and no abnormalities were found. The experimental design consisted of a crossover (6 × 2) design. Each of the six animals was subjected to two different treatments with a seven-day interval between them. The order of treatments was defined by simple randomization.

For the study, the animals were housed in Brachiaria brizantha grass paddocks with good availability of natural shade and water. The diet was supplemented with chopped elephant grass (Pennisetum purpureum) and concentrate with 20% crude protein provided at a rate of 1.5 kg·animal^{-1}·day^{-1}. During the twelve hours preceding the start of the treatments, the animals were subjected to water and food deprivation and kept in individual pens covered with sawdust bedding.

The treatments employed were Treatment 1 g (TREAT1g)—the animals received 1 g of sodium acetate per kg of body weight diluted in 5 liters of water, while those in Treatment 2 g (TREAT 2 g) received 2 g of sodium acetate per kg of body weight diluted in 5 liters of water. Each animal received a bolus of 5 liters of solution.

The solutions were administered via ororuminal probing using a 2-meter-long tube of 11 mm in diameter. Animals were monitored at predetermined intervals for 36 hours, through physical examinations and collection of samples for laboratory tests, as described: T0h (immediately before the administration of the solution containing sodium acetate), at T6h (six hours), T12h (twelve hours), T24h (twenty-four hours) and T36h (thirty-six hours) after the administration of the solutions with sodium acetate. The animals remained on a water-food fast for the 12 hours following the administration of the treatments (T0h to T12h).

Clinical Assessments: The clinical assessment of the animals consisted of: rectal temperature—measured in Celsius degrees (˚C) using a veterinary mercury column thermometer (Walmur®); heart rate—measured by auscultation of heart sounds in the region of the 4th and 5th left intercostal spaces with the aid of a stethoscope for one minute (beats per minute, bpm); respiratory rate—measured by auscultation of tracheobronchial sounds with the aid of a stethoscope for one minute (movements per minute—bpm); and ruminal motility—measured by auscultation of ruminal movements in the left paralumbar fossa with the aid of a stethoscope for five minutes.

Packed Cell Volume and Biochemical Tests: Blood samples were collected by puncture of the external jugular vein after prior antisepsis, using disposable hypodermic needles (25 mm × 0.8 mm) and a vacuum collection system. Tubes con-

taining the anticoagulant EDTA were used to determine packed cell volume and total protein. Tubes containing sodium fluoride with a capacity to obtain 4 mL of blood were used to determine plasma glucose and lactate concentrations. At the time, samples were also collected in siliconized glass tubes without an anticoagulant, with a capacity of 10 mL of blood. After obtaining serum, this was frozen at −20˚C and subsequently subjected to analysis of the following variables: sodium, potassium, chloride, calcium, magnesium, phosphorus, urea, creatinine, and osmolarity. Biochemical tests were performed using commercial kits on an Automated Biochemical Analyzer (Human Star300). Sodium and potassium measurements were performed using a Celm F250 flame photometer. Serum osmolarity was measured by the freezing point depression method using an Advanced Instruments 3320 Osmometer.

Blood Gas Analysis: For blood gas analysis, blood samples were collected by jugular vein puncture with a heparinized syringe (80 IU of lithium heparin) and immediately sent for analysis in an ABL80-Flex blood gas analyzer (Radiometer). The following variables were measured: pH; partial pressure of carbon dioxide (pCO_2); total carbon dioxide concentration ($ctCO_2$); bicarbonate concentration (HCO_3^-); titratable base excess (BE). The anion gap (AGap) and the strong ion difference (SID_3) were calculated using the following equations: $AGap = (Na^+ + K^+) - (Cl^- + HCO_3^-)$; $SID_3 = (Na^+ + K^+) - (Cl^-)$ [11].

Urine Analysis: Urine samples were obtained after perineal massage. Urinary pH was determined using an Akso portable digital pH meter. The urine was then centrifuged and frozen at −20˚C. Subsequently, the following variables were measured: sodium, potassium, chloride, calcium, and magnesium using commercial kits on an Automated Biochemical Analyzer (Human Star300).

Analysis of Rumen Fluid: Rumen fluid samples were obtained using a manual suction pump coupled to a 2-meter-long and 8 mm diameter probe inserted into the rumen through the cow's mouth. Immediately after collection, the pH of the rumen fluid was determined using an Akso portable digital pH meter. It was then centrifuged and frozen at −20˚C. Subsequently, osmolarity was evaluated (3320 Advanced Instruments Osmometer).

Statistical Analysis: Data were analyzed using two-way repeated-measures ANOVA, with treatment and time as fixed factors. Because each animal received both treatments and served as its own control in a balanced crossover design with complete follow-up, repeated-measures ANOVA was considered adequate to account for within-animal dependence. Tukey's test was used for post hoc comparisons. Statistical significance was set at $P < 0.05$.

3. Results

Respiratory rate increased ($P < 0.001$) at T6h and T36h in animals from both treatments (Table 1). Rectal temperature increased ($P < 0.001$) at T6h, T12h, and T36h in both treatments (Table 1).

Rumen motility showed a difference over time, characterized by a reduction in

Table 1. Mean values and standard mean errors of physiological parameters in cows subjected to treatments with sodium acetate at concentrations of 1 g·kg^{-1} of body weight (TREAT1g) and 2 g·kg^{-1} of body weight (TREAT2g) at various times (0 h to 36 h).

Treatment	T0h	T6h	T12h	T24h	T36h
Heart rate (beats per minute)					
TREAT1g	72.50 ± 4.33	78.30 ± 4.33	71.30 ± 4.33	72.50 ± 4.33	76.20 ± 4.33
TREAT2g	66.30 ± 4.33	71.70 ± 4.33	63.80 ± 4.33	73.30 ± 4.33	70.10 ± 5.3
Respiratory rate (breaths/minute)					
TREAT1g	25.90 ± 1.98^{Ab}	31.30 ± 1.98^{Aa}	23.50 ± 1.98^{Ab}	21.50 ± 1.98^{Ab}	30.00 ± 1.98^{Aa}
TREAT2g	24.70 ± 1.98^{Ab}	29.20 ± 1.98^{Aa}	21.00 ± 1.98^{Ab}	20.30 ± 1.98^{Ab}	29.70 ± 2.43^{Aa}
Rectal temperature (˚C)					
TREAT1g	38.10 ± 0.14^{Ab}	38.70 ± 0.14^{Aa}	38.80 ± 0.14^{Aa}	38.10 ± 0.14^{Ab}	38.60 ± 0.14^{Aa}
TREAT2g	38.00 ± 0.14^{Ab}	38.80 ± 0.14^{Aa}	38.70 ± 0.14^{Aa}	38.30 ± 0.14^{Ab}	38.60 ± 0.17^{Aa}
Rumen motility (rumen movements/5 minutes)					
TREAT1g	6.17 ± 0.74^{Aa}	5.00 ± 0.74^{Ab}	3.67 ± 0.74^{Ab}	6.83 ± 0.74^{Aa}	8.00 ± 0.74^{Aa}
TREAT2g	6.50 ± 0.74^{Aa}	3.17 ± 0.74^{Ab}	2.50 ± 0.74^{Ab}	5.33 ± 0.74^{Aa}	7.31 ± 0.9^{Aa}
Ruminal fluid pH					
TREAT1g	6.33 ± 0.10^{Ab}	6.66 ± 0.10^{Ab}	7.55 ± 0.10^{Aa}	6.71 ± 0.10^{Ab}	6.52 ± 0.10^{Ab}
TREAT2g	6.49 ± 0.10^{Ab}	7.04 ± 0.10^{Aa}	6.75 ± 0.10^{Ab}	6.67 ± 0.10^{Ab}	6.68 ± 0.10^{Ab}
Rumen fluid osmolarity (mOsmol/L)					
TREAT1g	239 ± 10.3^{Ab}	272 ± 10.3^{Aa}	267 ± 10.3^{Aa}	243 ± 10.3^{Ab}	225 ± 10.3^{Ab}
TREAT2g	241 ± 10.3^{Ab}	296 ± 10.3^{Aa}	292 ± 10.3^{Aa}	245 ± 10.3^{Ab}	233 ± 12.6^{Ab}
Packed cell volume (%)					
TREAT1g	33.20 ± 1.05	30.80 ± 1.05	32.50 ± 1.05	32.10 ± 1.05	32.00 ± 1.05
TREAT2g	32.50 ± 1.05	33.70 ± 1.05	34.70 ± 1.05	33.50 ± 1.05	32.00 ± 1.28
Total plasma protein (g/dL)					
TREAT1g	8.03 ± 0.15	7.33 ± 0.15	7.87 ± 0.15	7.83 ± 0.15	7.82 ± 0.15
TREAT2g	7.77 ± 0.15	7.87 ± 0.15	8.22 ± 0.15	7.97 ± 0.15	7.59 ± 0.19

Mean values followed by different uppercase letters in the same column or by different lowercase letters in the same row differ from each other ($P < 0.05$) by Tukey's test.

rumen movements ($P < 0.001$) at T6h and T12h in TREAT1g and TREAT2g (Table 1). There was no significant variation in rumen fluid pH values between treatments (Table 1). However, variation over time was recorded in both treatments. There was an increase at T12h in animals from TREAT1g, and at T6h in those from TREAT2g ($P < 0.001$). No difference was recorded in ruminal fluid osmolarity values between treatments ($P > 0.05$), but a difference was detected in treatments throughout the experimental phase ($P < 0.001$). The highest values were rec-

orded at 6 h and 12 h in animals from both treatments (Table 1).

Table 2. Mean values and standard mean errors of blood electrolytes in cows subjected to sodium acetate treatments at concentrations of 1 g·kg^{-1} body weight (TREAT1g) and 2 g·kg^{-1} body weight (TREAT2g) at various times (0 h to 36 h).

Treatment	T0h	T6h	T12h	T24h	T36h
Serum sodium (Na^+ mMol/L)					
TREAT1g	142.00 ± 2.79^{Ab}	147.00 ± 2.79^{Aa}	152.00 ± 2.79^{Aa}	143.00 ± 2.79^{Ab}	141.00 ± 2.79^{Ab}
TREAT2g	140.00 ± 2.79^{Ab}	148.00 ± 2.79^{Aa}	155.00 ± 2.79^{Aa}	144.00 ± 2.79^{Ab}	144.00 ± 3.42^{Ab}
Serum potassium (K^+ mMol/L)					
TREAT1g	4.65 ± 0.19^{Aa}	3.48 ± 0.19^{Ab}	3.23 ± 0.19^{Ab}	4.02 ± 0.19^{Ab}	4.03 ± 0.19^{Ab}
TREAT2g	4.42 ± 0.19^{Aa}	3.1 ± 0.19^{Ab}	3.05 ± 0.19^{Ab}	3.47 ± 0.19^{Ab}	3.65 ± 0.23^{Ab}
Serum chloride (Cl^- mMol/L)					
TREAT1g	98.2 ± 1.59	98.3 ± 1.28	101.11 ± 1.28	98.63 ± 1.28	102.7 ± 1.28
TREAT2g	97.8 ± 1.28	101.63 ± 1.28	102.21 ± 2.28	100.05 ± 1.28	101.25 ± 1.59
Serum magnesium (Mg^{2+} mg/dL)					
TREAT1g	2.43 ± 0.15	2.22 ± 0.15	2.32 ± 015	2.33 ± 0.15	2.82 ± 0.15
TREAT2g	2.25 ± 0.15	2.22 ± 0.15	2.28 ± 0,15	1.97 ± 0.15	2.16 ± 0.19
Total serum calcium (tCa^{2+} mg/dL)					
TREAT1g	9.67 ± 0.45^{Aa}	8.67 ± 0.45^{Ab}	8.03 ± 0.45^{Ab}	9.33 ± 0.45^{Ab}	8.33 ± 0.45^{Ab}
TREAT2g	10.2 ± 0.45^{Aa}	8.83 ± 0.45^{Ab}	8.17 ± 0.45^{Ab}	8.97 ± 0.45^{Ab}	8.19 ± 0.55^{Ab}
Serum phosphorus (P mg/dL)					
TREAT1g	5.82 ± 0.37	5.63 ± 0.37	5.53 ± 0.37	6.22 ± 0.37	6.03 ± 0.37
TREAT2g	5.93 ± 0.37	6.33 ± 0.37	5.47 ± 0.37	5.70 ± 0.37	5.82 ± 0.45

Mean values followed by different uppercase letters in the same column or by different lowercase letters in the same row differ from each other ($P < 0.05$) by Tukey's test.

In both treatments, sodium levels increased at T6h and T12h ($P = 0.012$), while potassium levels decreased at T6h, T12h, T24h, and T36h ($P < 0.001$). Likewise, calcium levels decreased from T6h to T36h in animals from both treatments ($P = 0.015$) (Table 2).

Plasma glucose showed an increase in its indices ($P < 0.001$) at T12h in animals from both treatments (Table 3). There was a difference between treatments in creatinine values. At T6h and T12h, animals from the TREAT2g showed the highest values when compared to those from the TREAT1g ($P = 0.021$), while over time within the group TREAT2g, the highest values were recorded at T12h ($P < 0.001$).

No difference was recorded in serum osmolarity values between treatments ($P > 0.05$), but a difference was detected in TREAT2g throughout the experimental phase ($P < 0.001$). The highest values were recorded at 6 h and 12 h (Table 3).

Table 3. Mean values and standard mean errors of biochemical parameters in cows subjected to treatments with sodium acetate at concentrations of 1 g·kg^{-1} of body weight (TREAT1g) and 2 g·kg^{-1} of body weight (TREAT2g) at various times (0 h to 36 h).

Treatment	T0h	T6h	T12h	T24h	T36h
Plasma lactate (mg/dL)					
TREAT1g	14.50 ± 1.27	14.00 ± 1.27	12.30 ± 1.27	15.30 ± 1.27	11.80 ± 1.27
TREAT2g	14.30 ± 1.27	13.70 ± 1.27	14.00 ± 1.27	12.30 ± 1.27	12.10 ± 1.27
Plasma glucose (mg/dL)					
TREAT1g	75.10 ± 2.27^{Ab}	78.50 ± 2.27^{Ab}	82.20 ± 2.27^{Aa}	77.00 ± 2.27^{Ab}	72.10 ± 2.27^{Ab}
TREAT2g	75.00 ± 2.27^{Ab}	82.40 ± 2.27^{Ab}	90.30 ± 2.27^{Aa}	77.80 ± 2.27^{Ab}	74.80 ± 2.78^{Ab}
Serum urea (mg/dL)					
TREAT1g	33.80 ± 4.70	42.70 ± 4.70	32.30 ± 4.70	34.30 ± 4.70	31.50 ± 4.70
TREAT2g	46.50 ± 4.70	46.50 ± 4.70	46.00 ± 4.70	45.30 ± 4.70	46.00 ± 5.76
Serum creatinine (mg/dL)					
TREAT1g	1.42 ± 0.06^{Aa}	1.31 ± 0.06^{Ba}	1.45 ± 0.06^{Ba}	1.42 ± 0.06^{Aa}	1.45 ± 0.06^{Aa}
TREAT2g	1.47 ± 0.06^{Ab}	1.56 ± 0.06^{Ab}	1.75 ± 0.06^{Aa}	1.36 ± 0.06^{Ab}	1.41 ± 0.07^{Ab}
Serum osmolarity (mOsmol/L)					
TREAT1g	286 ± 1.08^{Aa}	299 ± 1.08^{Aa}	298 ± 1.08^{Aa}	287 ± 1.08^{Aa}	285 ± 1.08^{Aa}
TREAT2g	289 ± 1.08^{Ab}	312 ± 1.08^{Aa}	313 ± 1.08^{Aa}	289 ± 1.08^{Ab}	289 ± 1.32^{Ab}

Mean values followed by different uppercase letters in the same column or by different lowercase letters in the same row differ from each other ($P < 0.05$) according to Tukey's test.

Regarding pH values, there was a difference over time, for both treatments, at T6h and T12h ($P < 0.001$) (Table 4). As can be seen in Table 4, there was a difference between treatments in HCO_3^-, $ctCO_2$, and BE values. At T6h, T12h, and T24h, animals from the TREAT2g showed the highest values when compared to those from the TREAT1g, while over time within the group TREAT2g, the highest values were recorded at T6h and T12h ($P < 0.031$).

In the AGap indices, a difference was recorded between treatments and within treatments over time. The animals in TREAT1g showed the highest values compared to those in TREAT2g at T6h, while over time, the lowest values were recorded in the animals in TREAT1g at T36h and in the animals in TREAT2g at T6h ($P < 0.004$) (Table 4). In turn, in SID_3, a difference was recorded over time ($P < 0.008$) in both treatments, with the highest values detected at T6 and T12h (Table 4).

There was an increase in urine pH values in animals from both treatments at T12h ($P = 0.005$) (Table 5). Also, differences were seen between treatments in urine density values, with the lowest values recorded in animals from the Treat2g group at T6h and T12h ($P = 0.005$) (Table 5).

Table 4. Mean values and standard mean errors of blood gas analysis in cows subjected to treatments with sodium acetate at concentrations of 1 g·kg^{-1} of body weight (TREAT1g) and 2 g·kg^{-1} of body weight (TREAT2g) at various times (0 h to 36 h).

Treatment	T0h	T6h	T12h	T24h	T36h
Blood pH (pH)					
TREAT1g	7.41 ± 0.01^{Ab}	7.48 ± 0.01^{Aa}	7.47 ± 0.01^{Aa}	7.43 ± 0.01^{Ab}	7.43 ± 0.01^{Ab}
TREAT2g	7.42 ± 0.01^{Ab}	7.53 ± 0.01^{Aa}	7.51 ± 0.01^{Aa}	7.47 ± 0.01^{Ab}	7.45 ± 0.01^{Ab}
Partial pressure of carbon dioxide (pCO_2, mmHg)					
TREAT1g	42.5 ± 1.23	44.1 ± 1.23	44.7 ± 1.23	43.5 ± 1.23	41.3 ± 1.23
TREAT2g	42.8 ± 1.23	45.2 ± 1.23	47.5 ± 1.23	44.6 ± 1.23	41.7 ± 1.51
Bicarbonate concentration (HCO_3^-, mEq/L)					
TREAT1g	27.83 ± 0.89^{Ab}	33.13 ± 0.89^{Ba}	33.25 ± 0.89^{Ba}	28.83 ± 0.89^{Bb}	27.5 ± 0.89^{Ab}
TREAT2g	27.08 ± 0.89^{Ab}	37.85 ± 0.89^{Aa}	37.63 ± 0.89^{Aa}	32.38 ± 0.89^{Ab}	28.83 ± 1.09^{Ab}
Total carbon dioxide concentration ($ctCO_2$, mEq/L)					
TREAT1g	28.50 ± 0.91^{Ab}	34.50 ± 0.91^{Ba}	34.58 ± 0.91^{Ba}	30.21 ± 0.91^{Bb}	28.76 ± 0.91^{Ab}
TREAT2g	28.38 ± 0.91^{Ab}	39.25 ± 0.91^{Aa}	39.03 ± 0.91^{Aa}	33.23 ± 0.91^{Ab}	29.38 ± 1.12^{Ab}
Base excess (BE, mEq/L)					
TREAT1g	2.86 ± 0.83^{Ab}	9.06 ± 0.83^{Ba}	9.03 ± 0.83^{Ba}	4.10 ± 0.83^{Bb}	3.05 ± 0.83^{Ab}
TREAT2g	2.68 ± 0.83^{Ab}	13.55 ± 0.83^{Aa}	12.80 ± 0.83^{Aa}	7.36 ± 0.83^{Ab}	3.11 ± 1.01^{Ab}
Anion Gap (Agap, mEq/L)					
TREAT1g	20.62 ± 2.93^{Aa}	19.05 ± 2.93^{Aa}	20.87 ± 2.93^{Aa}	19.56 ± 2.93^{Aa}	14.83 ± 2.93^{Ab}
TREAT2g	19.54 ± 2.93^{Aa}	11.62 ± 2.93^{Bb}	18.21 ± 2.93^{Aa}	15.04 ± 2.93^{Aa}	17.57 ± 3.58^{Aa}
Strong ion difference (SID_3, mEq/L)					
TREAT1g	48.45 ± 2.85^{Ab}	52.18 ± 2.85^{Aa}	54.12 ± 2.85^{Aa}	48.39 ± 2.85^{Ab}	42.33 ± 2.85^{Ab}
TREAT2g	46.62 ± 2.85^{Ab}	49.47 ± 2.85^{Aa}	55.84 ± 2.85^{Aa}	47.42 ± 2.85^{Ab}	46.40 ± 3.49^{Ab}

Mean values followed by different uppercase letters in the same column or by different lowercase letters in the same row differ from each other ($P < 0.05$) according to Tukey's test.

$UrNa^+$ increased in animals from the TREAT1g at T6h and T12h, while in animals from the TREAT2g, the increase was observed at T6h, T12h, and T24h ($P < 0.001$) (Table 5). A difference was found over time, in both treatments, in the values of urCl, urCa, and urMg at T6h, T12h, T24h, and T36h ($P < 0.001$). However, the difference between treatments was recorded only in the urMg values at T6h, T12h, and T24h ($P = 0.003$) (Table 5).

Table 5. Mean values and standard mean errors of urinary parameters in cows subjected to treatments with sodium acetate at concentrations of 1 g·kg^{-1} of body weight (TREAT1g) and 2 g·kg^{-1} of body weight (TREAT2g) at various times (0 h to 36 h).

Treatment	T0h	T6h	T12h	T24h	T36h
Urinary pH (urpH)					
TREAT1g	8.18 ± 0.03^{Ab}	8.35 ± 0.03^{Ab}	8.39 ± 0.03^{Aa}	8.31 ± 0.03^{Ab}	8.26 ± 0.03^{Ab}
TREAT2g	8.19 ± 0.03^{Ab}	8.38 ± 0.03^{Ab}	8.47 ± 0.03^{Aa}	8.36 ± 0.03^{Ab}	8.23 ± 0.04^{Ab}
Urine specific gravity					
TREAT1g	1029 ± 1.81^{Aa}	1025 ± 1.81^{Aa}	1028 ± 1.81^{Aa}	1025 ± 1.81^{Aa}	1027 ± 1.81^{Aa}
TREAT2g	1026 ± 1.81^{Aa}	1020 ± 1.81^{Ba}	1021 ± 1.81^{Ba}	1023 ± 1.81^{Aa}	1026 ± 2.22^{Aa}
Urinary sodium (urNa^{+}, mMol/L)					
TREAT1g	58.70 ± 21.8^{Ab}	135.00 ± 21.8^{Aa}	197.00 ± 21.8^{Aa}	120.00 ± 21.8^{Ab}	89.00 ± 21.8^{Ab}
TREAT2g	38.20 ± 21.8^{Ac}	162.00 ± 21.8^{Ab}	237.00 ± 21.8^{Aa}	196.00 ± 21.8^{Ab}	85.90 ± 26.7^{Ac}
Urinary potassium (urK^{+}, mMol/L)					
TREAT1g	33.90 ± 12.4	23.60 ± 12.4	22.70 ± 12.4	22.80 ± 12.4	29.10 ± 12.4
TREAT2g	25.00 ± 12.4	21.30 ± 12.4	18.00 ± 12.4	20.00 ± 12.4	26.50 ± 15.2
Urinary chloride (urCl^{-}, mMol/L)					
TREAT1g	107.00 ± 6.44^{Aa}	33.60 ± 6.44^{Ab}	25.90 ± 6.44^{Ab}	39.00 ± 6.44^{Ab}	48.20 ± 6.44^{Ab}
TREAT2g	82.90 ± 6.44^{Aa}	26.60 ± 6.44^{Ab}	32.70 ± 6.44^{Ab}	37.10 ± 6.44^{Ab}	32.20 ± 7.89^{Ab}
Urinary magnesium (urMg^{2+}, mg/dL)					
TREAT1g	31.30 ± 3.56^{Aa}	18.00 ± 3.56^{Ab}	20.70 ± 3.56^{Ab}	14.80 ± 3.56^{Ab}	12.20 ± 3.56^{Ab}
TREAT2g	24.40 ± 3.56^{Aa}	8.85 ± 3.56^{Bb}	8.45 ± 3.56^{Bb}	8.98 ± 3.56^{Bb}	12.80 ± 4.36^{Ab}
Urinary calcium (urCa^{2+}, mg/dL)					
TREAT1g	9.83 ± 2^{Aa}	4.50 ± 2^{Ab}	2.50 ± 2^{Ab}	3.00 ± 2^{Ab}	4.40 ± 2^{Ab}
TREAT2g	8.70 ± 2^{Aa}	1.33 ± 2^{Ab}	1.00 ± 2^{Ab}	1.17 ± 2^{Ab}	3.50 ± 2.45^{Ab}
Urinary phosphorus (urP, mg/dL)					
TREAT1g	1.47 ± 1.89	1.88 ± 1.89	1.90 ± 1.89	1.37 ± 1.89	1.12 ± 1.89
TREAT2g	3.50 ± 1.89	5.67 ± 1.89	9.23 ± 1.89	10.90 ± 1.89	6.01 ± 2.31

Mean values followed by different uppercase letters in the same column or by different lowercase letters in the same row differ from each other ($P < 0.05$) by Tukey's test.

4. Discussion

Changes in heart rate (HR) are frequently associated with stressful environmental conditions and pain, which triggers the release of norepinephrine and thus stimulates the sympathetic nervous system [12]. The fact that HR did not change significantly over time and across treatments suggests that the experimental procedures and the concentrations of sodium acetate used did not cause stress in the animals. Respiratory rate (RR) also remained within the normal range for cattle

[12], although variations were observed over time. A small increase in RR was recorded at T6h and T36h ($P < 0.05$) in animals from both treatments. This increase may be associated with ambient temperature, as the respiratory rates measured at T6h and T36h occurred during the typically warmest time of day, around 1:00 PM.

Although rectal temperature showed the lowest values in animals from both treatments at T0h and T24h, which corresponded to the morning period, at around 07:00 AM, still the values remained within the normal range [13], which makes them clinically insignificant.

Ruminal hypomotility was recorded at T6h in animals from the TREAT2g and at T12h in those from the TREAT1g. This decrease in ruminal motility was possibly caused by sodium acetate, since after its infusion via ororuminal route, a modest, but significant, increase in the pH and osmolarity values of the ruminal fluid was also recorded at T6h and T12h. As Grünberg & Constable [14] stated, some factors can alter rumen motility, among them we can mention the modification in the osmolarity and pH of the ruminal fluid and in the calcium levels in the blood. As can be seen in **Table 1** and **Table 2**, there was a significant increase in the osmolarity and pH values of the ruminal fluid and a decrease in serum calcium. These changes may have favored the suppression of rumen muscle function, leading to a state called paresis, which is the loss of muscle strength [15], expressed by a decrease in ruminal motility, confirming the data obtained in this research.

The changes caused by the infusion of sodium acetate in the osmolarity and pH of the ruminal fluid in animals from both treatments were discreet, even in animals that received the highest dose, 2 $g \cdot kg^{-1}$ (TREAT2g), demonstrating that this compound exerts important systemic buffering with a low capacity to alter ruminal pH, as cited by Schumann *et al.* [5]. This slight capacity to cause changes in the ruminal fluid differentiates sodium acetate from sodium bicarbonate, because when comparing the alkalizing effect of both substances administered orally in calves, Marshall *et al.* [16] recorded a greater alkalizing effect on the gastrointestinal tract of calves that received an isotonic solution of sodium bicarbonate.

Although less significant, it should be noted that other causes may also have contributed to the appearance of ruminal hypomotility, including water and feed deprivation from T0h to T12h, as well as the decrease in serum calcium and potassium values recorded from T6h onwards in animals from both treatments. At T24h, twelve hours after the end of fasting, ruminal motility and the osmolarity and pH of the ruminal fluid were restored.

Packed cell volume (PCV) and total protein (TP) are commonly used markers in assessing the degree of dehydration in animals [17] [18]. During the experimental phase, the values of both parameters remained without significant changes ($P > 0.05$) in animals from both treatments (**Table 1**).

Serum sodium did not differ between treatments, but a difference was recorded within animals from both treatments over time. At T6h and T12h, the highest Na^+ values were detected in animals from both treatments (**Table 2**). This increase was

due to intestinal absorption of the large sodium load administered to the animals. The absorption of this electrolyte occurs along the forestomachs and intestines. Under normal conditions, 100% of ingested sodium is absorbed through active and passive mechanisms in the rumen, small intestine, and large intestine [19], confirming the findings of this trial.

To date, there appears to be no studies reporting the effects of sodium acetate administered by ororuminal route on serum electrolytes in adult cattle, but results similar to those of the present trial were recorded in adult horses by Waller & Lindinger [6] after administration of a solution containing 500 grams of sodium acetate via the nasogastric route, and by Kline *et al.* [20] when comparing the effect of sodium bicarbonate and sodium acetate.

The results of the present trial demonstrate the alkalizing potential of sodium acetate, since, as cited by Constable [21], solutions with high Na^+ content cause an increase in the value of strong ion difference (SID), which in turn will cause the appearance of metabolic alkalosis. In addition, sodium acetate may also be an adequate option for correcting hyponatremia in adult cattle and calves. However, since its administration, depending on the dose, can lead to hypernatremia, it is important to monitor serum or plasma sodium levels in patients treated with sodium acetate, as well as keep them hydrated, since an increase in blood sodium, in addition to its therapeutic effect, can also cause adverse effects. Despite the increase in serum sodium, osmolarity, and creatinine values recorded in the TREAT2g animals, the 2 $g \cdot kg^{-1}$ dose of sodium acetate is safe, as the observed increases in these variables returned to baseline levels (T0h) twenty-four hours (T24h) after administration. Furthermore, despite the increase in serum creatinine values observed at T6h and T12h, they remained within the normal range for adult cattle.

Regardless of the dose of sodium acetate administered to the animals, one (TREAT1g) or two grams (TREAT2g) per kilogram of body weight, all resulted in a significant decrease in serum potassium values from T6h until the end of the experimental phase (T36h). However, the most pronounced decreases were recorded at T6h and T12h in animals from both treatments. This decrease is consistent with the increase in serum sodium and BE (**Table 2** and **Table 4**). As mentioned earlier, the increase in serum or plasma sodium determines an increase in the strong ion difference (SID), which in turn causes the appearance of metabolic alkalosis [22]. Once established, metabolic alkalosis, through a compensatory mechanism, depending on its intensity, may determine the translocation of potassium from the extracellular fluid to the interior of the cell, producing a decrease in the blood [11], as can be seen in **Table 2**. Results like those of the present research were described by Kline *et al.* [20] and Waller & Lindinger [6] in adult horses.

As can be seen in **Table 2**, no difference was detected in the absolute values of chloride, magnesium, and phosphorus between treatments, nor in the treatments over time in the animals of TREAT1g and TREAT2g. Although the decrease or

increase in serum or plasma sodium values may cause relative hypo- or hyperchloremia.

Serum levels of total calcium showed similar behavior to potassium in animals from both treatments, decreasing from T6h and remaining stable until T36h (**Table 2**). This finding can be interpreted as an indirect consequence of the alkalinization promoted by sodium acetate. In metabolic alkalosis, alterations in the values of various electrolytes can occur, including calcium [6] [10] [23].

Calcium-carrying plasma proteins have a buffering effect in the extracellular fluid [24]. While metabolic acidosis is characterized by an increased affinity for H^+ ions and consequent release of blood calcium, resulting in hypercalcemia, metabolic alkalosis involves an increased avidity of carrier proteins for free calcium, reducing its plasma availability [20], manifesting as serum or plasma hypocalcemia. Although sodium acetate therapy is intended for the treatment of metabolic acidosis, monitoring calcium levels is important due to the possibility of altered blood values.

Plasma lactate and serum urea levels did not show a statistically significant difference between treatments and over time ($P > 0.05$), remaining within the reference values for cattle [25], as can be seen in **Table 3**.

Regardless of the dose of sodium acetate provided, an increase in blood glucose was observed, with a maximum value reached at 12 h in animals from both treatments (**Table 3**). As the animals had already been fasting from water and food for several hours, this increase in plasma glucose values can be considered an effect of sodium acetate, since, as cited by Goularte *et al.* [26] in cattle, acetate is produced by microbial fermentation in the rumen. After its production, it is absorbed through the rumen wall, reaching the bloodstream. In the blood, it is transported to body tissues, where it is converted into acetyl-CoA, entering the citric acid cycle to produce energy, contributing to gluconeogenesis. When administering sodium acetate to horses, Waller & Lindinger [6] recorded similar results to those obtained in the present trial.

Measuring creatinine is important to the evaluation of renal function in cattle [18]. Although its values remained within the normal range during the experimental phase [25], there was an increase in its values at 6 h and 12 h in the animals of the TREAT2g, returning to baseline values from T24h onwards. Normally, when there is a decrease in blood volume, the glomerular filtration rate decreases; consequently, there will be less renal excretion of this metabolite. However, it was confirmed that the increase in creatinine at T6h and T12h in the animals of the TREAT2g was clinically irrelevant, as the globular volume and plasma protein remained unchanged throughout the experimental phase.

Serum osmolarity increased in animals from the TREAT2g at T6h and T12h. The animals in the TREAT2g received twice as much sodium as those in the TREAT1g, and since ingested sodium is absorbed throughout the digestive system, an increase in its blood concentration was recorded, as can be seen in **Table 2**. In turn, the increase in serum sodium values generated an increase in serum

osmolarity, because, as Carlson & Bruss [22] cited, sodium can contribute up to 80% to the value of serum osmolarity, justifying the result of this assay.

A similar pattern was observed in the values of pH, HCO_3^-, $ctCO_2$, BE, and SID_3. The highest pH and SID_3 values were obtained in animals from both treatments at T6h and T12h. In turn, HCO_3^-, $ctCO_2$, and BE, besides showing the highest values at T6h and T12h, were found in animals that received TREAT2g at higher concentrations than those in Trat1g at T6h, T12h, and T24h. The increase in the values of these variables characterizes a scenario of metabolic alkalosis, highlighting that the TREAT2g group of animals presented greater intensity. When administering sodium bicarbonate orally to adult cows, Bigner *et al.* [2] also recorded an increase in blood pH and HCO_3^- values. However, its oral use has marked capacity to alkalize the animal's digestive system, generating changes in intraluminal pH and resident microbiota, in addition to increasing gas production, which is why its oral use has declined in prescription.

The main factor that contributed to the development of metabolic alkalosis in the animals of this assay was the increase in the concentration of the strong ion difference [SID_3], as a result of the increase in the serum concentration of [Na^+] without alteration of the serum concentration of [Cl^-]. As cited by Constable [21], solutions with a high Na^+ content without alteration in Cl^- cause an increase in the value of the strong ion difference (SID), which in turn determines the appearance of metabolic alkalosis due to strong ions.

These results were expected, since according to the physicochemical approach, the independent variables that determine the acid-base state are the concentration of strong ions in solution, defined as the difference in strong ions [SID], partial pressure of carbon dioxide [pCO_2], and the concentration of weak acids in solution, called the total concentration of weak acids [A_{tot}]. Thus, the dependent acid-base variables [H^+], bicarbonate concentration [HCO_3^-], and total carbon dioxide concentration [$ctCO_2$] only change when at least one of the independent variables is modified [27] [28].

As shown in **Table 4**, no changes were recorded in the pCO_2 values. A significant decrease in anion gap values was detected at T6h in the animals of the TREAT2g; however, this decrease is devoid of clinical significance as the values remained within the normal range [22]. The increase in urinary pH observed at 12 h in animals from both treatments confirms the systemic alkalizing effect of sodium acetate. Since the kidneys play a fundamental role in acid-base balance by contributing to the control of acid and base concentrations in the extracellular fluid that are not of respiratory origin [29], an increase in urinary pH values resulting from excess blood bicarbonate was recorded, confirming the compensatory effect of the kidneys.

Urine density remained within the normal range [30], despite the small, but not significant, decrease observed in the animals in the TREAT2g at T6h and T12h (**Table 5**). However, significantly lower values were recorded in the 2 g Treatment group animals when compared to those in the TREAT1g at 6 h and 12 h, demonstrating

that urinary dilution was greater in the TREAT2g animals during this period.

The greater urinary dilution recorded in the TREAT2g animals was caused by the larger amount of sodium acetate that these animals received. According to DiBartola [11], when there is greater absorption of Na^+, hypernatremia, an increase in blood volume, and an elevation in blood pressure usually develop, which in turn stimulate the kidneys to excrete excess Na^+ and water. This renal compensation mechanism is called natriuretic pressure, by which a group of hormones, called natriuretic peptides, acts by regulating blood volume and blood pressure. They are natural antagonists of the renin-angiotensin-aldosterone system, acting by promoting natriuresis and diuresis. Confirming the mechanism described above, in the present study, **Table 5** shows an increase in urinary sodium values in animals that received the highest amount of sodium acetate at 6 h, 12 h, and 24 h (TREAT2g).

To ensure that urinary dilution explained above occurred, a significant decrease can be observed over time, from T6h to the end of the experimental phase, in the values of chloride, calcium, and magnesium (**Table 5**). It should be noted that the decreases recorded in the urinary concentrations of these three electrolytes may also be associated with the metabolic alkalosis caused by the administration of sodium acetate. As cited by Constable [21] and Carlson & Bruss [22], absolute or relative decreases in serum chloride, calcium, and magnesium are observed in metabolic alkalosis. Therefore, as a compensatory mechanism, urinary excretion of these electrolytes decreases, confirming the results obtained in the present assay (**Table 5**).

It can be concluded that administration of sodium acetate via the ororuminal route in adult cattle at doses of one (TREAT1g) and two (TREAT2g) grams per kilogram of body weight resulted in the development of metabolic alkalosis due to strong ions and a slight increase in ruminal fluid pH. Therefore, sodium acetate administered via the ororuminal route can be used as an alkalizing agent in adult cattle. The results of this trial offer new perspectives for the use of ororuminal alkalizing agents in this class of animals.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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