Acta vet. scand. 1995, *36*, 65-77.

Induced Acute Ruminal Acidosis in Goats Treated with Yeast *(Saccharomyces cerevisiae)* and Bicarbonate

By *V. Aslan*[1], *S. M. Thamsborg*[1], *R. J. Jørgensen*[1] and *A. Basse*[2]

[1]Department of Clinical Studies, and [2]Department of Pharmacology and Pathobiology, The Royal Veterinary and Agricultural University, Frederiksberg, Denmark.

Aslan, V., S. M. Thamsborg, R. J. Jørgensen and A. Basse: Induced acute ruminal acidosis in goats treated with yeast (Saccharomyces cerevisiae) and Bicarbonate. Acta vet. scand. 1995, 36, 65-77. – Ruminal acidosis was induced in twenty-one 10-month-old West African Dwarf Goats by feeding a suspension of 80 g wheat flour per kg bodyweight (day 0) through a stomach tube. Ruminal and systemic acidosis was diagnosed on day 1 in all goats. Clinical signs included loss of rumination and appetite, trembling, and watery diarrhoea. The detection of acidic faeces during the first 24h was considered of diagnostic importance. Subgroups were treated orally on days 1, 2, and 3 either with 1 g of sodium bicarbonate per kg bodyweight, with 1 g of baking yeast per kg, or with a combination of these treatments at 0.5 g of each per kg. A fourth group served as untreated controls.

Peroral bicarbonate neutralization was highly effective in the treatment of rumen acidosis, whereas the use of yeast was found ineffective. The combined treatment had a moderate effect probably due to the bicarbonate.

Three fatal cases (60%) occurred in the untreated group compared with none in the bicarbonate group, and 2 in each of the remaining groups. This corresponded to 33% of the yeast treated group and 40% of the combined treated group. Details were given on post mortem examinations performed on all survivors on day 11. Lesions included subacute rumenitis and abomasal ulcers. No lesions were found in 3 of the bicarbonate treated goats and in 2 of the animals receiving combined treatment.

rumen pathology; therapy.

Introduction

In ruminants, overeating with easily fermentable carbohydrates leads to acute lactic acidosis *(Dirksen* 1986) which is often fatal if left untreated. The treatment aims at correcting the ruminal and systemic acidosis, preventing further production of lactic acid, correcting fluid and electrolyte losses, maintaining circulating blood volumes, and restoring forestomach and intestinal motility *(Radostits et al.* 1994). Such therapy basically relies on neutralization with bicarbonate, but may also include intraruminal administration of antibiotics, water and electrolytes, intravenous fluid therapy including alkalinizing agents and, as a last resort, rumenotomy *(Radostits et al.* 1994). *Raz & Landau* (1970) concluded that neutralization with bicarbonate given perorally was essential in acute cases, whereas *Börkü & Imrein* (1989) found that intravenous fluid therapy, including bicarbonate, was sufficient as the only treatment of experimen-

Table 1. Experimental groups and treatments. Treatments were given by stomach tube daily for 3 days, starting 24h after wheat flour drenching on day 0.

Group	No. of animals	Treatment on day 1+2+3
C	5	Untreated controls
B	5	Sodium bicarbonate 1g/kg* bodyweight/day
Y	6	Baking yeast 1g/kg/day
BY	5	Sodium bicarbonate 0.5g/kg** plus baking yeast 0.5g/kg/day

* equals 12 mmol/kg
** equals 6 mmol/kg

tally induced acidosis in sheep. Care must be taken with regard to bicarbonate concentration and infusion speed *(Hyldgaard-Jensen & Simesen* 1966).

Among the less known suggestions for treatment is baking or beer brewing yeast given orally. Such therapy was first mentioned by *Broberg* (1960) who claimed a satisfactory therapeutic effect in natural cases of grain overeating among sheep and cattle. According to *Dirksen* (1986), a dose of 2 to 4 kg of non-desiccated yeast is suggested for adult cattle. Although the therapeutic value and possible mechanism is unclear *(Dirksen* 1986), yeast was part of the standard therapy of cases of ruminal lactic acidosis in sheep and goats reported by *Braun et al.* (1992). No dosage was given, and the therapeutic effect was not measured separately.

Similarly, the combined effect of yeast and penicillin was reported by *Hejlasz & Nicponn* (1977) who gave 0.5 kg of yeast and 3 mill IU of penicillin to mild experimental acidosis in cows 6h after the administration of sugar. This prevented the development of clinical acidosis with full acid-base equilibrium in venous blood.

In recent years, the probiotic effect of adding yeast products to the ration of dairy cows has been described (e.g. *Williams et al.* 1991).

The possible therapeutic effect of yeast, given as the only treatment, does not appear to have been tested against ruminal lactic acidosis. Therefore, an experiment was designed to compare the effect of treatment with baking yeast, bicarbonate, or with a combination of yeast and bicarbonate of wheat flour induced acidosis in goats.

Materials and methods

Animals, feeding and housing

Twenty-one castrated West African Dwarf goats, approximately 10 months old with a mean bodyweight of 20 kg (range 17-21 kg) were used. The animals were offered hay ad libitum before and during the trial. Food was withheld for 24h prior to the start of the experiment to ensure sufficient ruminal capacity for the experimental ration. During the trial, all groups were penned separately on concrete floor with no straw bedding.

Design and treatments

On day 0, all animals were given 80 g of wheat flour per kg of bodyweight. The flour was given as a suspension in lukewarm water (1:2 w/v) by stomach tube. The animals were allocated into 4 groups according to bodyweight. Details of the treatments are given in Table 1.

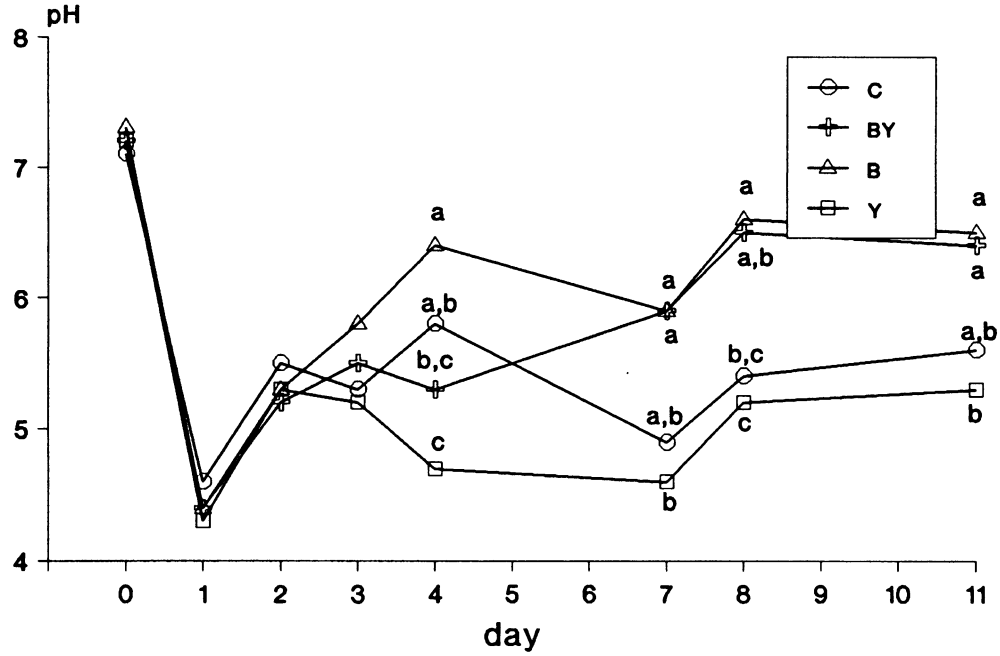

Figure 1. Mean pH of rumen fluid in the control (C), bicarbonate + yeast (BY), bicarbonate (B) and yeast (Y) treated group. Groups with different letters day 4, 7, 8 and 11 are significantly different.

Commercial non-dessiccated baking yeast was used. According to the producer (Danisco, De Danske Spritfabrikker, Copenhagen, Denmark), it consisted of *Saccharomyces cerevisiae* grown on molasses.

Clinical observations and post mortem examinations

All animals were examined 3 times daily with regard to general appearance and behaviour, appetite, rumination and diarrhoea. Animals with a poor prognosis, based on clinical grounds, were euthanized using pentobarbital intravenously. The trial was terminated on day 11 where all survivors were killed with pentobarbital and subjected to examination post mortem for gastro-intestinal tract lesions.

Sampling and analyses

The animals were sampled on days 0, 1, 2, 3, 4 and 7, and rumen pH was also measured on days 8 and 11. Rumen samples were collected by stomach tube. The pH was measured by an electronic pH-meter (ATC pH-meter, model Piccolo, Hannah, Singapore). The protozoal activity was evaluated by microscopical examination of warm samples at 40x magnification. Bacterial activity was evaluated by the methylene blue test *(Dirksen 1969, Radostits et al.* 1994). Osmolality was measured after the termination of the experiment on rumen samples stored at –18°C. On the day of examination, the samples were thawed. Particle-free, transparent samples were obtained by centrifugation at 4000 rpm for 15 min followed by filtration of the supernatant through a 0.45 μm disposable filter assembly (Acrodisc, product

Table 2. Appetite/rumination scores during the experiment.

Group	Day no.										
	0	1	2	3	4	5	6	7	8	9	10
C	2	0	0	0	0	0	0	0	0	0	0
B	2	0	0	0	1	1	1	2	2	2	2
Y	2	0	0	0	0	0	0	0	0	0(1)	0(1)
BY	2	0	0	0	0	0	0	1	1	1	1

0/1/2: No/reduced/normal appetite/rumination

no. 4184, Colman Sciences, Missouri, USA). Samples of 0.25 ml were processed in a freeze-point osmometer (Advanced DigiMatic model 3D2, Advanced Instruments, Massachusetts, USA).

Blood samples were collected from the jugular vein into 2 ml syringes containing 0.1 ml heparin solution (5000 IU/ml). The samples were examined immediately on a blood gas analyser (Model ABL4, Radiometer, Danmark). Haematocrit was determined by standard microhaematocrit centrifugation. The glutaraldehyde test *(Sandholm* 1974, *Doll et al.* 1985) was performed in vacutainer tubes (Glutavac Test, J. Kruuse a/s, Marslev, Denmark). Equal volumes of whole venous blood and 1.4% glutaraldehyde solution were mixed, and observed for max. 15 min for gel formation. Gel formation within 15 min is considered indicative of hypergammaglobulinaemia or increased fibrinogen level *(Jørgensen et al.* 1990) and is thus an indicator of inflammation.

Statistical analysis

Blood parameters from the blood gas analyser, haematocrit, rumen and faecal pH were analysed one day at a time by using analysis of variance, and the different treatments were ranked using the Duncan multiple range test using the PC-SAS, *(SAS Institute Inc.* 1988). Data from animals euthanized or dying during the trial were not excluded from the analysis. $P < 0.05$ was considered statistically significant.

Results

Clinical observations

During the first few days of the experiment, a uniform clinical picture was observed in all 4 groups. On day 1, all animals showed CNS depression, developed watery diarrhoea, and exhibited periods of trembling. All were able to stand, most animals with their head and tail kept in a low position. Rumination was not observed during days 1 to 3, and all animals were anorectic (Table 2).

The first fatal cases occurred in Group C. Two animals were excluded on day 2 and a 3rd case on day 8. The remaining 2 animals of the group did not regain their appetite.

No animals were lost in Group B, and at termination of the trial all had regained their appetite.

The responses of goats in Group Y were similar to those in Group C. Of the 6 animals, 1 was lost on day 5 and 1 on day 10. Individuals were observed to eat some hay at the trial termination. A single animal, which had persistent diarrhoea until day 5, suddenly died on day 10. A large penetrating abomasal ulcer with diffuse peritonitis was found at necropsy (Goat No. 16, Table 5).

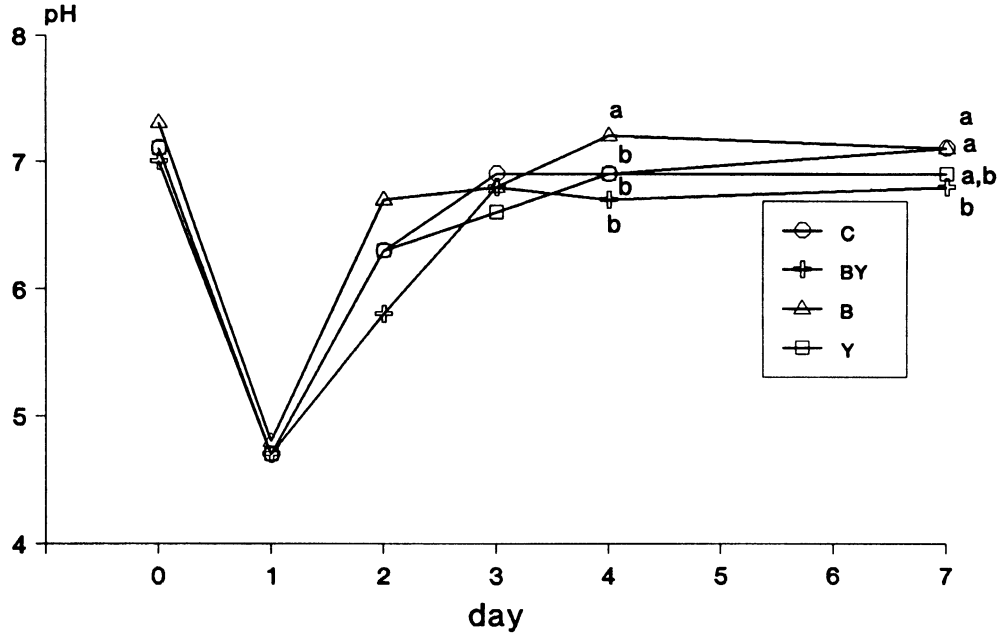

Figure 2. Mean pH of faecal samples in the control (C), bicarbonate + yeast (BY), bicarbonate (B) and yeast (Y) treated group. Groups with different letters day 4 and 7 are significantly different.

The goats of Group BY showed an intermediate response with regard to appetite when compared to Groups B and Y. One goat died on day 2, and 1 was euthanized on day 3.

Teeth grinding and plaintive sounds were noted in 2 animals which collapsed late in the experiment (days 8 and 10), but not in animals which died or were euthanized during the acute depressive stage before day 4, characterized by profound ruminal acidosis.

Rumen and faecal pH

From a relatively high rumen pH of 7.16 (SD 0.26, n = 21), probably due to the preceding period of starvation, all groups experienced severe rumen acidosis from day 1 (Fig. 1). Very few animals regained this initial level during the experiment, but pH of 6.5 or above was recorded in all goats in Group B, in 2 out of 3 in Group BY, and in none in the remaining groups.

Faecal pH showed a similar and simultaneous decrease followed by an increase, but in contrast to rumen pH, the values normalized in all groups during the next couple of days (Fig. 2). The faecal pH of Group B was significantly higher than Group BY on day 0 before the experimental meal. pH of groups C and Y were between these. The difference is not shown in Fig. 2. However, comparison of the ruminal and faecal pH of all animals on day 0, before onset of symptoms, failed to show any significant correlation between the 2 parameters.

Rumen osmolality

Rumen osmolality was not measured on day 0. However, the values on day 1 (Fig. 3) prob-

Table 3. Blood pH and standard base excess (SBE) of fatal cases. The measurements were made on the day of euthanasia or death.

Group/ goat no.	Day no.	Blood pH	Blood SBE
C 1	2	6.997	-19.2
C 5	2	6.931	-21.6
BY 9	2	6.768	-24.5
BY 6	3	7.025	-22.1
Y 20	5	7.285	- 5.3
C 2	8	7.164	-10.0
Y 16	10	7.171	-10.3

ably reflected an increase from physiological levels of 270-300 mOsm/l *(Carlson* 1989). The next 2 days, the level declined in all groups. On day 7, groups B and BY were at a considerably lower level than the 2 other groups, but no differences were significant.

Protozoal activity

Before the experiment, rumen protozoa were abundant in all animals. On day 1, protozoa were not detected. In groups C and Y, the protozoa did not reappear, whereas in groups B and BY small protozoa reappeared in low numbers after day 6.

Methylene blue test

Before the experiment, decolorization took place in approximately 5 min. During the first days of acidosis, reaction time was reduced to 8-10 min whereafter it became shorter than 5 min. No apparent differences were recorded between the groups.

Blood parameters

The increase in haematocrit from 35% to 45% the first day (Fig. 4) indicates that a pronounced haemoconcentration took place in all groups, but while the 2 bicarbonate treated groups compensated and in fact exhibited haemodilution and then returned to normal range, the haematocrite of groups Y and C remained elevated throughout the trial.

However, large variation was noted in this parameter as reflected in few significant differences in spite of large average differences. Standard base excess (SBE) is shown in Fig. 5. Blood pH and standard bicarbonate had a very similar trend and are not shown. As can be seen, all groups experienced severe systemic acidosis on day 1. During days 2, 3, and 4, Groups C and Y approached normal values, whereas, Groups B and Group BY showed marked alkalosis, apparently parallel to the amount of bicarbonate given. Maximum levels were recorded on day 4, one day after the third treatment and coinciding with maximum haemodilution.

The blood pH and SBE values of fatal cases demonstrate severe acidosis as the cause of the fatal clinical condition in the early part of the experiment, whereas the cases which occurred on days 5, 8 and 10 were only moderately acidotic (Table 3).

The partial pressure of CO_2 was very low at the start of the trial (Fig. 6). Normally pCO_2 in venous blood of ruminants is in the range 35-44 mm Hg *(Carlson* 1989). The pCO_2 fell markedly on day 1, probably due to hyperventilation.

In Groups C and Y, the pCO_2 levelled out, whereas it increased drastically in Groups B and BY, probably in response to and apparently proportional to the bicarbonate treatment.

Glutaraldehyde test

The glutaraldehyde test was negative in all animals on days 0, 1, and 2 (Table 4). On day 3, the 3 remaining animals of Group C became positive. In these, which all were severely affected and did not eat, the reaction time became significantly shorter. In group B, the an-

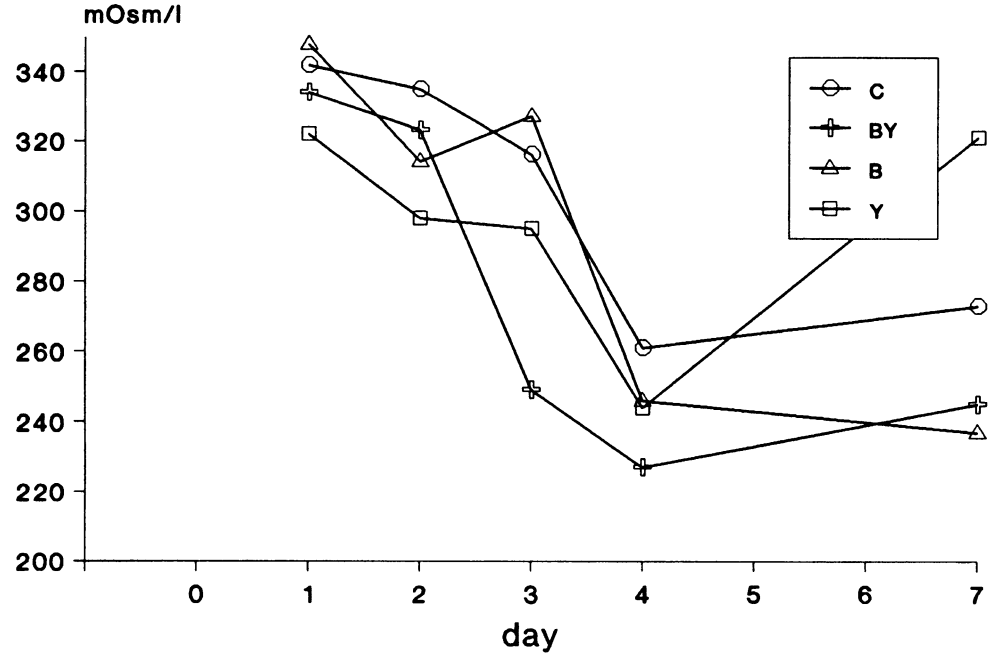

Figure 3. Mean osmolality of rumen samples in the control (C), bicarbonate + yeast (BY), bicarbonate (B) and yeast (Y) treated group. No significant differencies between groups.

imals responded later and apparently at a lower level as compared to those of Group C, and the test became negative again on day 10 except in 1 animal which developed subacute pneumonia (goat no. 15, Table 5). Goat no. 14 in Group B remained negative throughout the trial. As in Group C, the goats of Group Y became positive and remained so, apparently with even shorter reaction times than Group C. Fast positive reactions were recorded late in the trial among the animals in Group BY, the exception being goat no. 10 which remained negative throughout the trial.

Post mortem examination

The details of the post mortem findings are given in Table 5. From the table it can be deduced that in Group C, lethal acute rumenitis occurred in 3 goats, whereas subacute rumenitis persisted in the 2 survivors on day 11. One of these also had abomasal ulcers. In Group B, either no or slight signs only of rumenitis were recorded on day 11 with the exception of goat No. 11 which had subacute rumenitis and abomasal ulcers. Group Y had 1 fatal case in the acute phase plus 1 goat dying on day 10 with a perforating abomasal ulcer, and subacute rumenitis in the 4 survivors. The severity of the lesions seen in Group Y may thus be characterized as intermediate to Group C and B. In Group BY, 2 lethal cases occurred in the acute phase, whereas the survivors only had mild lesions. Group BY may therefore also be considered intermediate to Group B and C in severity, but it differed from Group Y in that those who survived the acute phase appeared similar to those of Group B on day 11.

Table 4. Glutaraldehyde reaction time in minutes.

Group	Goat No.	Day no. 0	1	2	3	4	7	8	9	10
C	1	–	–	*						
	2	–	–	–	15	10	4	2	*	
	3	–	–	–	12	12	10	10	4	5
	4	–	–	–	10	13	13	10	2	2
	5	–	–							
B	11	–	–	–	–	–	14	14	10	–
	12	–	–	–	–	–	12	12	13	–
	13	–	–	–	–	15	14	12	10	–
	14	–	–	–	–	–	–	–	–	–
	15	–	–	–	–	–	10	–	5	7**
Y	16	–	–	–	–	–	2	2	2	2
	17	–	–	–	–	5	2	2	4	4
	18	–	–	–	–	–	2	2	2	2
	19	–	–	–	–	–	5	5	5	2
	20	–	–	–	–	15	*			
	21	–	–	–	–	–	6	5	5	5
BY	6	–	–	–	*					
	7	–	–	–	–	–	10	5	5	–
	8	–	–	–	–	–	12	5	6	–
	9	–	–	–	*					
	10	–	–	–	–	–	–	–		

–: Negative (more than 15 min)
*: euthanized or dead
**: Subacute pneumonia

In some of the goats which had not started to eat or ruminate, a conglomerate of flour was found in the ventral sac of the rumen.

Discussion

Although goats have been reported tolerant to grain engorgement *(Mgasa & Mbassa* 1988), forced feeding of 80 g wheat flour pr kg bodyweight obviously caused severe acute ruminal and systemic acidosis. According to *Dirksen* (1986), the recorded SBE values of –14 to –16 mMol/l are considered severe acidosis.

The low level of pCO_2 at start could perhaps be explained by the starving period resulting in loss of bicarbonate to saliva. The fact that rumen pH was high initially as compared to the reference range of 5.9-7.0 given by *Braun et al.* (1992) may support this.

Wheat flour was used for ease of feeding through a stomach tube. The finding of a conglomerate in some of the severely affected goats at post mortem may leave wheat flour less ideal for experimental purposes than more coarsely grinded grain.

Braun et al. (1992) found teeth grinding and muscle twitching in 37 hospitalized sheep and goats considered to be suffering from acute lactic acidosis. Initially, we saw depression and muscle twitching, whereas teeth grinding

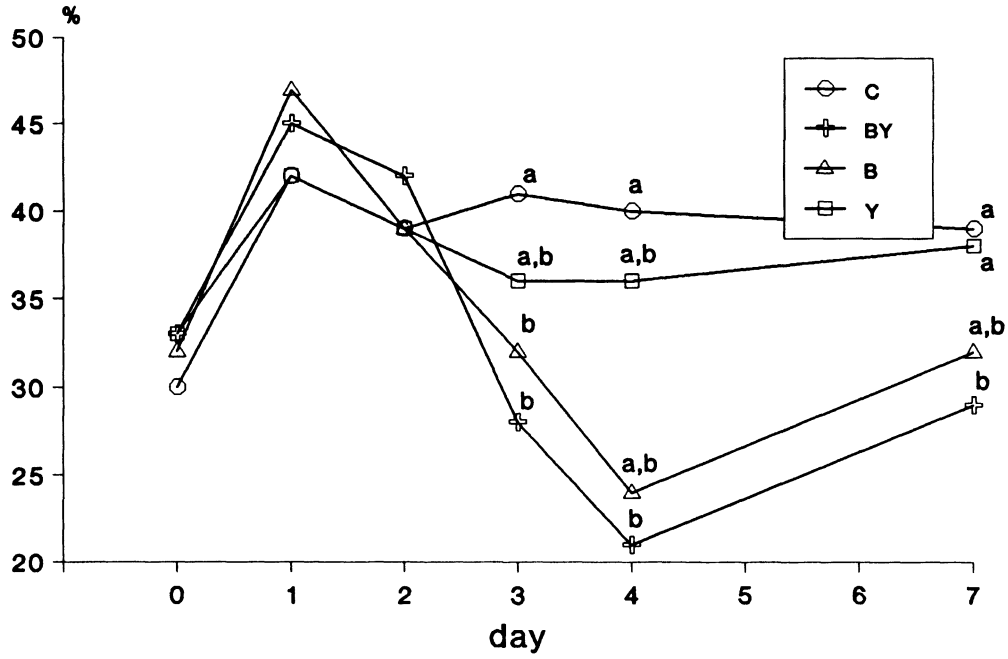

Figure 4. Mean haematocrit in the control (C), bicarbonate + yeast (BY), bicarbonate (B) and yeast (Y) treated group. Groups with different letters day 3, 4, and 7 are significantly different.

appeared later on, particularly in individuals with a doubtful or poor prognosis. The depression coincided with the peak of acidosis and may be linked with acidosis in the central nervous system *(Lal et al.* 1991), whereas teeth grinding may result from painful lesions in the forestomachs and abomasum. Based on the clinical, the pathophysiological, and pathological findings, the observed course of the acidosis may be divided into an acute and a subacute phase. While the former can be explained by severe acidosis alone, the latter can not. The cause of fatal clinical cases on days 5, 8, and 10, the acidosis being only moderate, may be linked with shock caused by the severe lesions in the gastrointestinal canal.

Of the general post mortem findings it was of particular interest to find that abomasal ulcers, together with rumenitis, were sequelae to the feed induced acidosis.

The drop in faecal pH appears to be a useful diagnostic tool within the first 24h. *Kovac et al.* (1986) made similar observations within 8-10h but not 24h after sucrose induced rumen acidosis in calves.

The glutaraldehyde test reaction indicated inflammatory lesions in nearly all animals as confirmed at post mortem, whereas the 2 goats which remained negative were without lesions. A short reaction time was, however, not recorded before 7-8 days in Group C. The test may therefore be of prognostic value in later stages, but not in the early phase of acute ruminal acidosis.

Yeast treatment did not influence the gross clinical picture and its influence, if any, on ap-

Table 5. Post mortem findings. Goat no.'s 1, 2, 5, 6, 9, 16 and 20 either died or were euthanized due to poor prognosis before termination on day 11.

Group	Goat No.	Day No.	Gross diagnosis	Details
C	1 and 5	2	Rumenitis, acute	Hyperperaemia of papillac in atrium ruminis
C	2	8	Rumenitis, subacute	Epithelial necrosis and dyskeratosis in all forestomachs ulcers
C	3	11	Abomasitis rumenitis, subacute	Epithelial necrosis and dyskeratosis in all forestomachs; depapillation and conglomeration of papillae
C	4	11	Rumenitis, subacute	Epithelial necrosis, and dyskeratosis in all forestomachs, ulcerations to serosa
B	11	11	Rumenitis, subacute abomasitis	Widespread dyskeratosis, depapillation and epithelial necrosis; focal peritonitis ulcers
B	12 and 13	11	None	Few hyperaemic papillac in atrium ruminis
B	14	11	None	
B	15	11	Rumenitis, focal pneumonia subacute	Depapillation with ulcers in rumen
Y	20	5	Rumenitis, acute	Epithelial necrosis, hyperaemia and dyskeratosis
Y	16	10	Rumenitis, subacute	Rumen focal necrosis and peritonitis. Pronounced dyskeratosis, depapillation and epithelial necrosis perforating abomasal ulcers
Y	17	11	Rumenitis, subacute	Necrosis, dyskeratosis and depapillation
Y	18	11	Rumenitis, subacute	Ulcers, focal peritonitis, papillar necrosis and dyskeratosis
Y	19	11	Rumenitis, subacute	Moderate hyperkeratosis and depapillation
Y	21	11	Rumenitis, subacute	Hyper- and dyskeratosis; depapillation; papillar conglomeration
BY	6 and 9	2 and 3	Rumenitis, acute abomasitis	Hyperaemia, dyskeratosis, acute superficial ulcers
BY	7	11	Rumenitis, low grade	Hyperkeratosis; papillar conglomeration in ventral sac; hyperaemia in dorsal sac
BY	8	11	None	Exfoliation on a few rumen papillae
BY	10	11	None	Depapillation on 3 areas (1×1 cm) of rumen wall; minor catarrhal bronchopneumonia.

petite, and the relative number of survivors was far from that recorded in response to oral bicarbonate treatments.

The good results obtained with oral sodium bicarbonate treatment was in accordance with the finding of *Raz & Landau* (1970), who emphazised the importance of neutralizing the rumen content in acute cases. The systemic alcalosis and haemodilution seen in the bicarbonate treated groups may be interpreted as overdosing, although spontaneous alcalosis is known to occur following episodes of severe acidosis.

It is generelly recommended to aim at neutralization when bicarbonate is given by the intravenous route. This may be achieved by multiplying the SBE at the time of treatment by 0.3 *(Carlson* 1989), or by 0.6 in young calves with diarrhoea *(Tromp* 1990), in which case 5 to 7.5 and 3 to 5 mMol/kg should have been given on days 1 and 2 respectively, and none on day 3. However, no indication of ill effects from the resulting base excess was recorded in Group B as compared to group BY which only received half the dose.

Our experiment shows that the often pre-

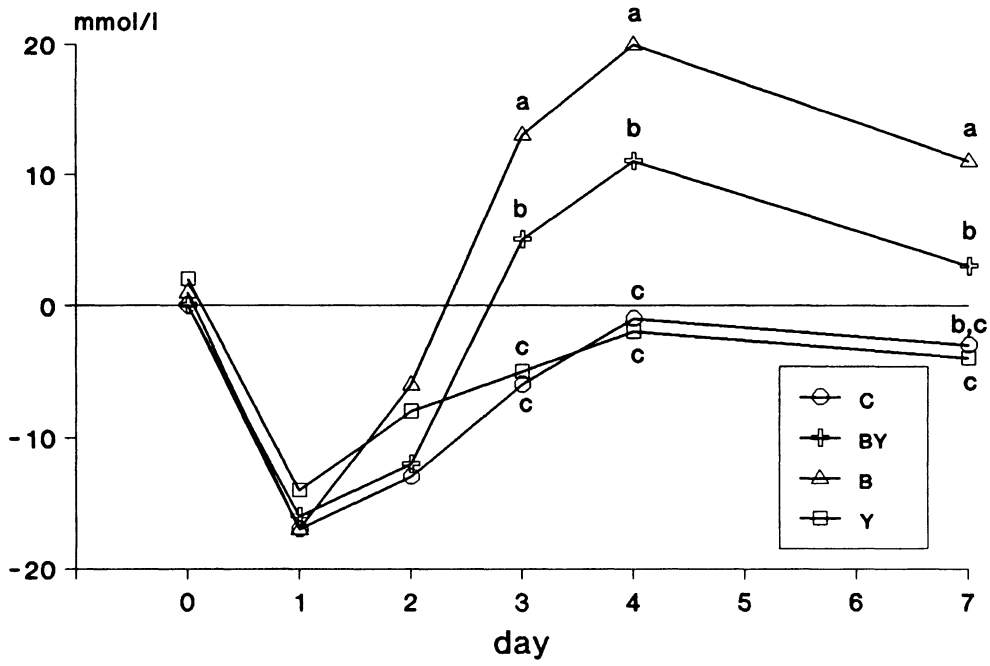

Figure 5. Mean standard base excess (SBE) in blood in the control (C), bicarbonate + yeast (BY), bicarbonate (B), and yeast (Y) treated group. Groups with different letters day 3, 4, and 7 are significantly different.

ferred intravenous route of neutralization is not essential, even in cases with ruminal stasis, and that moderate overdosing by the peroral route was well tolerated. It may be argued that under field conditions severe overdosing with bicarbonate is more likely to occur when neutralization is attempted by intravenous treatment.

Conclusion

The present experiment shows that peroral bicarbonate neutralization was highly effective in the treatment of rumen lactic acidosis, whereas the use of yeast was found ineffective. It thus appears that yeast treatment of acute ruminal acidosis is not justified.

Acknowledgement

This study was supported by the Danish Agricultural and Veterinary Research Council (grant no. 13-4374).

References

Börkü MK & Imren HY: Clinical, haematological changes and fluid therapy in experimentally induced ruminal acidosis in sheep. Turkish J. vet. anim. Sci., 1989, *13,* 414-431.

Braun U, Rihs T, Schefer U: Ruminal lactic acidosis in sheep and goats. Vet. Rec. 1992, *130,* 343-349.

Broberg G: Acute overeating with cereals in ruminants. *Thesis.* Lovisa (Helsinki), Finland 1960.

Carlson GP: Fluid, electrolyte and acid-base balance. In: Kaneko JJ (ed): Clinical biochemistry of domestic animals, 4th ed., Academic Press Inc., San Diego. 1989, 543-575.

Dirksen G: Ist die "Methylenblauprobe" als Schnelltest für die klinische Pansensaftuntersuchung

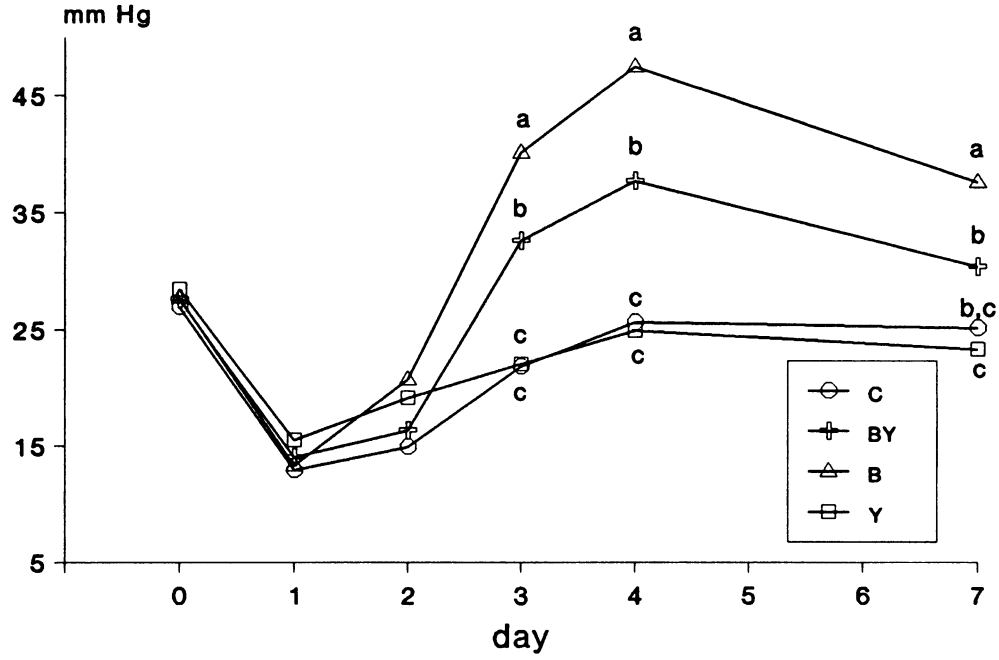

Figure 6. Mean pCO_2 of blood in the control (C), bicarbonate + yeast (BY), bicarbonate (B), and yeast (Y) treated group. Groups with different letters day 3, 4 and 7 are significantly different.

geeignet? (Is the "methylenblue-reduction-probe" useful as quick-test for clinical examination of rumen fluid?). Deutsche Tierärztl. Wochenschr., 1969, *76,* 305-309.

Dirksen G: Der Pansenazidose-Komplex – neuere Erkenntnisse und Erfahrungen (2). (The rumen acidosis complex. (Recent progress and experiences). Tierärztl. Prax., 1986, *14,* 23-33.

Doll K, Schillinger D, Klee W: The glutaraldehyde test in cattle – its usefulness for diagnosis and prognosis of internal inflammatory conditions. Zentralblatt Vet. Med. A, 1985, 581-593.

Hejlasz Z, Nicponn J: Uklady buforowe krwi w doswiadczalnej kwasicy krow i proby jej zapobiegania. (Blood buffer systems in experimental acidosis of cows and attempts at preventing it). Polskie Archiwum Weterynaryjne, 1977, *20,* 103-118.

Hyldgaard-Jensen J, Simesen, MG: Grutforgiftning hos kvæg (Rumen overload in cattle). Nord. Vet.-Med. 1966, *18,* 73-94.

Jørgensen RJ, Basse A, Aslan V: Sequelae to oral calcium chloride gel dosing of cows. XVI World Buiatrics Congress. Salvador, Bahia, Brasil, 1990. Proceedings, 511-515.

Kovac G, Mudron P, Bartko P: Comparative studies on the pH of blood, rumen and abomasal fluid, faeces and urine in cattle. Folia Veter., 1986, *30,* 21-34.

Lal SB, Awarup D, Dwivedi SK, Sharma MC: Biochemical alterations in serum and cerebrospinal fluid in experimental acidosis in goats. Res. vet. Sci. 1991, *50,* 208-210.

Mgasa MN, Mbassa GK: Tolerance of goats to experimental grain engorgement and intraruminal lactic acid injection. Vet. Res. Comm. 1988, *12,* 143-147.

Radostits OM, Blood DC, Gay CC: Veteriary Medicine 8th ed., Baillière Tindall, London 1994.

Raz A, Landau M: The treatment of experimentally induced acute overeating with cereals in sheep. Refuah vet. 1970, *27,* 143-147.

Sandholm M: A preliminary report of a rapid

method for the demonstration of abnormal gammaglobulin levels in bovine whole blood. Res. Vet. Sci. 1974, *17,* 32-35.

SAS Institute Inc: SAS/Stat™ Users Guide, Release 6.03 Edition. Cary, NC: 1988, 1028 pp.

Tromp AM: A practitioner's views on fluid therapy in calves. Veterinary Clinics of North America: Food Animal Pract. 1990, *6,* 103-110.

Williams PE, Tait CAG, Innes GM, Newbold CJ: Effects of the inclusion of yeast culture *(Saccharomyces cereviciae* plus growth medium) in the diet of dairy cows on milk yield and forage degradation and fermentation patterns in the rumen of steers. J. Anim. Sci. 1991, *69,* 3016-3026.

Sammendrag

Oral behandling af induceret akut vomacidose med gær henholdsvis bikarbonat.

Vomacidose blev induceret på 21 ti måneder gamle dværggeder ved indgivelse af 80 g hvedemel per kg legemsvægt (dag 0). Vomacidose og systemisk acidose diagnosticeredes på dag 1 hos alle gederne, idet der konstateredes ophørt appetit og vomkontraktioner, rystelser og vandig diarre. Påvisning af sur fæces de første 24 timer vurderedes at være af diagnostisk værdi.

Undergrupper behandledes oralt på dag 1, 2 og 3 med enten 1g Na-bicarbonat per kg legemsvægt, med 1 g bagegær per kg, eller med en kombination bestående af 0,5 g af hver per kg legemsvægt. En fjerde gruppe fungerede som ubehandlet kontrol. Peroral bikarbonatneutralisering var effektivt medens indgivelse af gær alene var værdiløst. Effekten af den kombinerede behandling var moderat og kunne tilskrives bikarbonaten. Alle i bikarbonatgruppen rettede sig. I den ubehandlede gruppe optrådte 3 fatale tilfælde (60%) imod 2 i hver af de resterende grupper, svarende til 33% af gærgruppen og 40% af gruppen, der fik den kombinerede behandling. Obduktion blev foretaget på dag 11. Subakut rumenitis og løbesår dominerede. Tre af de bikarbonatbehandlede og 2 af de, der modtog kombineret behandling, var helt uden forandringer.

(Received August 18, 1994; accepted October 7, 1994).

Reprints may be requested from: R. J. Jørgensen, Department of Clinical Studies, The Royal Veterinary and Agricultural University, Bülowsvej 13, DK-1870 Frederiksberg C, Denmark.