CLINICO-THERAPEUTIC STUDY OF RUMINAL LACTIC ACIDOSIS IN GOATS

By

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THESIS

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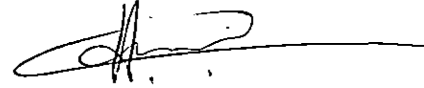

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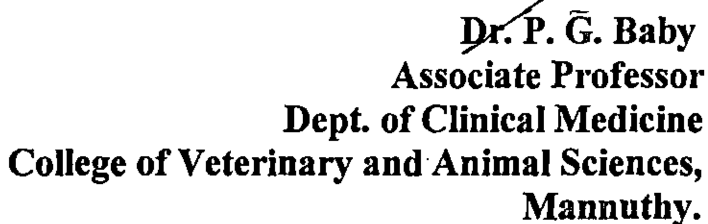

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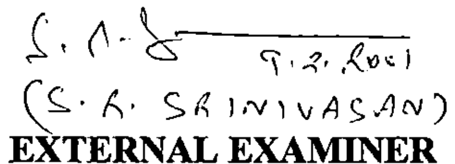

EXTERNAL EXAMINER

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Dr. Anil J. Thachil

DEDICATED

TO

MY PARENTS

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LIST OF ABBREVIATIONS

DLC	-	Differential leukocyte count
ESR	-	Erythrocyte sedimentation rate
h	-	hour
Hb	-	Haemoglobin
MBRT	-	Methylene blue reduction time
min	-	minute
PCV	-	Packed cell volume
SAT	-	Sedimentation activity time
TEC	-	Total erythrocyte count
TLC	-	Total leukocyte count
TVFA	-	Total volatile fatty acid

INTRODUCTION

1. INTRODUCTION

Ruminal acidosis is caused mainly by the accidental or excessive ingestion of large quantities of feeds rich in highly fermentable carbohydrates without adaptation.

In the homestead farming system of goat rearing in Kerala, the occurrence of ruminal lactic acidosis in goats is very high.

In a study conducted at the veterinary hospitals in the suburbs of Thrissur, Pillai and Alikutty (1989a) found that 14.28 per cent of the total animals treated were goats. Digestive disorders constituted 57.69 per cent of the cases treated in goats and 18.07 per cent of this were ruminal acidosis.

The important causes of ruminal acidosis in goats in Kerala were reported to be due to accidental ingestion of large quantities of rice, paddy, grains, payasam and jackfruits (Aleyas and Vijayan, 1981).

Since the body weight for goats is less, even a small quantity of carbohydrate can cause severe ruminal

acidosis. The mortality rate is very high in acute and peracute cases of acidosis and can be upto 30 to 40 per cent in treated cases (Radostits *et al.*, 1994). This is because of the serious damage inflicted on vital organs by the lactic acid, severe metabolic acidosis, endotoxins, functional disturbances caused by low pH, dehydration, hypotension and biochemical alterations.

Keeping in view of the non-availability of fodder especially during summer months and the severity and frequency of occurrence of ruminal acidosis in goats in Kerala, the objectives of the study were directed to record the clinical signs, changes in rumen fluid and blood and to suggest a better therapeutic management for ruminal acidosis in goats.

The main therapeutic targets in ruminal acidosis are to prevent further lactic acid production, correct the ruminal and systemic acidosis, correct dehydration, hypovolaemia and shock and to restore normal gastro intestinal function. Though numerous studies on treatment of ruminal acidosis have been carried out, reports regarding a therapeutic trial covering all the above aspects are scanty.

In correcting systemic acidosis, intravenous injections of sodium bicarbonate solutions were considered safe and effective and were widely accepted. But many authors like Graf *et al.* (1985) and Stacpoole (1986) reported that in metabolic acidosis of dogs, treated with sodium bicarbonate solution intravenously, the intracellular pH further reduced because of increased carbon dioxide load. Filley and Kindig (1984) observed that use of equimolar solution of sodium carbonate and sodium bicarbonate (carbicarb) intravenously did not raise blood carbon dioxide levels and it also improved the cardiovascular function when compared to intravenous sodium bicarbonate solution in treatment of metabolic acidosis in dogs.

On the basis of the above reports, it was decided to undertake a study in clinical cases of ruminal acidosis in goats to illustrate the following:

1. Clinical signs in ruminal acidosis
2. Changes in the rumen fluid
3. Biochemical changes in blood
4. Compare the effects of parenteral sodium bicarbonate against carbicarb.

❁

REVIEW OF LITERATURE

2. REVIEW OF LITERATURE

It is estimated that about 18 per cent of the cases of anorexia in sheep and goats in India were due to acidosis (Prasad *et al.,* 1976). Acidosis caused by carbohydrate engorgement is an economically important disease of ruminants clinically characterised by severe toxemia, dehydration, ruminal stasis, weakness, recumbency and high mortality rate (Radostits *et al.*, 1994). Excessive ruminal microbial multiplication and subsequent absorption of lactic acid were responsible for the ruminant lactic acidosis syndrome (Huber *et al.,* 1984).

2.1 Clinical signs

Hoflund (1967) reported that in bovine acid indigestion the skin began to adhere firmly to the underlying tissue, the eyes became sunken in the sockets, the amount of urine diminished, recumbency with inability to get up by third day and death occurred within next 24 h if the treatment was not started on the first day.

Depending upon the severity of clinical signs, Gnanaprakasam (1970) classified ruminal acidosis in goats as mild, moderate and severe. In mild cases anophagia, abdominal enlargement, sluggishness or absence of rumen motility, dullness on percussion and constipation were noticed. Moderate cases showed anorexia with either constipation or diarrhoea, dyspnoea, tachycardia, soft, small and weak pulse, absence of rumen motility and mild tympany. Severe cases were characterized by anorexia, dullness, recumbency, low carriage of head with extended neck, hard and leathery skin, dyspnoea with grunting, foul smell from mouth, wobbling gait, injected mucous membranes, retracted eye balls, imperceptible pulse, dry muzzle and anuria. In all those cases clinical signs were manifested within 24 h of grain engorgement.

Dunlop (1972) reported hypotension and reduced renal blood flow in ruminal acidosis of cattle.

Mild to severe depression of central nervous system, head pressing, drooping of ears, recumbency, salivation, serous nasal discharge, diarrhoea, increased heart and respiratory rates were observed by Vestweber *et al.* (1974) in acidotic sheep.

According to Dougherty *et al.* (1975), the rumino-reticular movements did not become static until the rumen pH was less than 5.0 in induced ruminal acidosis in sheep.

Tanwar *et al.* (1983) reported depression, anorexia, grinding of teeth, regurgitation of rumen contents, disinclination to move, anuria, dehydration and an increase in pulse and respiratory rates in experimental ruminal acidosis in goats.

According to Pillai (1988), the clinical signs shown in induced ruminal acidosis of goats were complete cessation of reticulo-ruminal movements, cold extremities, tachycardia, increased pulse and respiratory rates, dehydration and purulent nasal discharge.

Lal *et al.* (1989) observed various degrees of anorexia, ruminal stasis, dullness, diarrhoea, constipation, abdominal pain, nasal discharge, pressing of head and grinding of teeth, fall in body temperature and increased pulse and respiratory rates in goats with induced ruminal acidosis.

Kussmaul breathing (rapid and deep), haemo concentration and laminitis were reported in acidotic cattle by Michell (1990).

In clinical cases of lactic acidosis in sheep and goats, Braun *et al.* (1992) noted anorexia, apathy, teeth grinding, muscle twitching, ruminal stasis and excretion of soupy or watery faeces .

A significant increase in the heart and respiratory rates 12 h after induction of acidosis in goats were noticed by Lal *et al.* (1993). The authors further reported changes in amplitude of P wave, QRS complex and increased T wave in ECG.

Suda *et al.* (1996) observed depression, diarrhoea and anorexia in induced acidosis in goats.

In induced lactic acidosis in goats, Shihabudheen (1998) noticed anorexia, rise in pulse and respiratory rate by 12 h; and dullness, depression mild tympany, gurgling sound on auscultation of rumen by 24 h. Foul smelling diarrhoea was noticed by 48 h and dehydration by 72 h. The author also observed reluctance to get up, cold extremities and fluid thrill on palpation of rumen. Electrocardiographic studies showed increased

heart rate, reduction in P amplitude, P duration, QT and PR intervals and increased T amplitude and T duration.

2.1.1 Respiration

The normal mean respiratory rate reported was 8.60 ± 0.52 per min (Pillai, 1988) and 21.55 ± 0.86 per min (Shihabudheen, 1998) in healthy goats.

In induced lactic acidosis in sheep, Huber (1969) observed accelerated respiration characterised by panting.

Gnanaprakasam (1970) reported simple dyspnoea in moderate cases and intermediate dyspnoea with grunting and coastal type of respiration in severe lactic acidosis of goats.

Vestweber *et al.* (1974) attributed increased respiratory rate in sheep with lactic acidosis as a compensatory reaction to systemic acidosis.

Respiratory rate increased from 12 per min at zero hour to 17 per min at 24 h in induced lactic acidosis in goats (Cao *et al.*, 1987).

According to Pillai (1988), the mean respiratory rate increased from 16.50 ± 0.70 per min at zero hour to 29.33 ± 0.66 per min at 96 h of induction of lactic acidosis in goats. Michell (1990) reported that severe acidosis caused clinical signs like rapid and deep respiration and cardio vascular disturbances in goats.

In sheep and goats with lactic acidosis, the mean respiratory rate recorded was 53.2 ± 32.5 per min and was greater than 45 per min in 38 per cent of the clinical cases. (Braun *et al.*, 1992).

Shihabudheen (1998) noticed an increase in the mean respiratory rate from 23.50 ± 0.43 per min at zero hour to 35.00 ± 1.00 at 48 h of induction of lactic acidosis in goats.

2.1.2 Pulse

The normal mean pulse rate reported in healthy goats was 58.50 ± 0.97 per min by Pillai (1988);

whereas Shihabudheen (1998) reported the rate as 85.00 ± 8.50 per min.

Gnanaprakasam (1970) noticed soft, small and weak pulse in moderate cases and imperceptible pulse in severe cases of lactic acidosis in goats.

According to Pillai (1988), the mean pulse rate increased from 56.66 ± 0.75 per min at zero hour to 88.88 ± 4.67 per min at 96 h after induction of lactic acidosis in goats.

Shihabudheen (1998) noticed an increase in pulse rate from 98.33 ± 9.80 per min at zero hour to 148.33 ± 16.62 per min at 24 h of induction of lactic acidosis in goats.

2.1.3 Temperature

The normal rectal temperature recorded in healthy goats were 103.24 ± 0.28°F (Pillai, 1988) and 101.97 ± 0.18 °F (Shihabudheen, 1998).

Gnanaprakasam (1970) observed a normal rectal temperature in most of the cases and 1 or 2°F increase

was noticed in few cases of severe lactic acidosis in goats.

A decrease in mean rectal temperature from 102.81 ± 0.14°F at zero hour to 101.04 ± 0.20°F at 72 h was observed by Pillai (1988) in induced lactic acidosis in goats.

Braun *et al.* (1992) noticed a rectal temperature of less than 40°C (104°F) in 68 per cent of the clinical cases of lactic acidosis in sheep and goats.

In induced lactic acidosis in goats, Shihabudheen (1998) noticed a decrease in mean rectal temperature from 102.10 ± 0.36 °F at zero hour to 101.33 ± 0.38 °F at 48 h of study.

2.1.4 Heart rate

The normal heart rate noticed in healthy goats ranged from 71.67 ± 7.92 to 95.00 ± 10.88 per min (Shihabudheen, 1998).

Gnanaprakasam (1970) reported tachycardia in moderate cases and in severe cases of lactic acidosis

in goats, heart rate increased to 120 to 160 beats per min.

An increase in heart rate was noticed by Vestweber *et al.* (1974) in sheep with severe acidosis. Cao *et al.* (1987) also reported an increase in heart rate from 98 to 174 per min at 24 h after induction of lactic acidosis in goats.

2.1.5 Mucous membrane

Gnanaprakasam (1970) reported injected mucous membrane in severe cases of lactic acidosis in goats whereas Vestweber *et al.* (1974) reported a congested and dark red mucous membrane in sheep with severe acidosis.

2.1.6 Rumen motility

Pillai (1988) recorded a normal rumen motility rate of one per min in healthy goats.

Gnanaprakasam (1970) reported sluggish or absence of ruminal contractions in mild cases and absence of rumen motilty in moderate and severe cases of lactic acidosis in goats.

A pH range of 3.9 to 4.5 was noticed in ruminal acidosis and that all the protozoa and cellulolytic bacteria were destroyed as pH fell from 5.00 towards 4.00. Lactic acid producing *Streptococcus bovis* was inhibited below pH 4.5 (Dunlop, 1972).

Allison *et al.* (1975) noticed a mean pH of 4.24 in ruminal contents of sheep after 24 h of induction of lactic acidosis.

According to Vihan and Rai (1985), the mean rumen fluid pH was 7.2 ± 0.12 in simple indigestion and 6.21± 0.15 in clinical cases of lactic acidosis in goats.

In experimental ruminal acidosis in goats, Pillai and Alikutty (1989b) reported a fall in pH of rumen liquor to 4.13 ± 0.16 at 24 h of induction, whereas the observation by Lal *et al.* (1991) was 4.54 ± 0.02 at 24 h.

Braun *et al.* (1992) found a moderately low rumen pH range of 5.1 to 5.8 in 23 per cent and a very low ruminal pH range of 4.1 to 5.0 in 53 per cent of the

clinical cases of sheep and goats with acute ruminal lactic acidosis.

In induced lactic acidosis of goats, Lal *et al.* (1992) noticed a significant drop in rumen pH from 4.71 ± 0.05 to 4.54 ± 0.02 between 12 h and 24 h and thereafter it increased to 5.69 ± 0.07 at 120 h. However Basak *et al.* (1993a) reported a significant decrease in rumen liquor pH from 12 h, which reached the lowest value of 4.37 ± 0.09 at 48 h and then increased to 5.60 at 120 h of study in induced lactic acidosis in goats.

A drastic and significant decline in rumen pH was observed by Suda *et al.* (1996) from 6 h to 72 h of induction of ruminal acidosis in goats.

Shihabudheen (1998) observed a significant reduction in mean pH from the zero hour value of 6.87 ± 0.22 to 4.16 ± 0.05 at 48 h and reached 4.78 ± 0.13 at 96 h in induced lactic acidosis in goats.

2. 2. 2 Physical characters

2. 2. 2. 1 Colour

Normal colour of rumen liquor in goats varied from grey to olive or brownish green (Rosenberger, 1979), brown to green (Dirksen, 1983), greenish yellow (Pillai, 1988) and olive green (Shihabudheen, 1998).

The colour of the rumen liquor in acidosis changed to milky green (Dirksen 1965), dark green or grey (Gnanaprakasam, 1970), milky grey (Dirksen, 1983) brown to grey colour (Pillai, 1988) and greenish brown to light grey (Lal *et al.* 1992). Basak *et al.* (1993a) reported that the colour of the rumen liquor changed to creamish brown at 12 h to 24 h of induction and later to greyish white at120 h of lactic acidosis in goats.

Shihabudheen (1998) reported a change in colour of rumen liquor from olive green to milky grey at 12 h and creamy from 24 h onwards of induction in lactic acidosis of goats.

2. 2. 2. 2 Consistency

Rumen liquor from healthy cattle was slightly viscous (Rosenberger, 1979), thin gruel like (Dirksen,

1983) and thick in consistency in goats (Pillai, 1988; Shihabudheen, 1998).

Consistency of rumen liquor from animals with acidosis was reported as watery (Dirksen, 1983; Braun *et al.*, 1992; Basak *et al.*, 1993a).

Shihabudheen (1998) reported thick consistency of rumen liquor even after 12 h of induction and became semiliquid at 24 h and watery by 48 h of induction of lactic acidosis in goats.

2 .2 .2 .3 Odour

Normal odour of rumen liquor was aromatic, depended on the nature of rumen contents (Rosenberger, 1979;Dirksen, 1983; Pillai, 1988; Shihabudheen, 1998).

In lactic acidosis of ruminants, rumen liquor had putrid smell (Gnanaprakasam, 1970), penetrating lactic acid odour, pungent sour smell (Dirksen, 1983), faintly to distinctly sour (Pillai, 1988; Lal *et al.*, 1989) and penetrating acid odour (Braun *et al.*, 1992).

Shihabudheen (1998) reported a faintly sour odour by 12 h, which changed to intense sour or penetrating

acid smell from 24 h to 96 h after induction of lactic acidosis in goats.

2 .2 .3 Microbial activity

2 .2 .3 .1 Rumen protozoa

Gnanaprakasam (1970) noticed no motile organisms in moderate and severe cases of rumen acidosis in goats.

According to Dunlop (1972) inhibition and death of protozoa in rumen liquor occurred at pH below 5.

In induced lactic acidosis in buffaloes, large sized protozoa was the first one that disappeared from rumen liquor followed by medium and small ones (Prasad *et al.,* 1973).

Dirksen (1983) stated that among the protozoa, large species were the first to vanish with disorders of ruminal digestion, and next the medium sized and small protozoa.

In induced lactic acidosis of goats, Pillai (1988) observed complete disappearance of protozoa from rumen liquor after 24 h of induction.

Total disappearance of protozoa from rumen liquor was noticed between 12 h and 72 h of induction, and protozoa of entodinium species reappeared in the rumen liquor in much smaller numbers at 96 h (Lal *et al.*, 1992).

Basak *et al.* (1993a) reported that average protozoal count and motility was reduced at 12 h and no protozoa were observed from 24 h to 84 h after induction of acidosis. However reappearance of rumen protozoa noticed from 96 h along with the improvement of rumen pH.

In induced lactic acidosis of goats, Suda *et al.* (1996) found that the total number of bacteria and protozoa were decreased from 6 h onwards.

Shihabudheen (1998) reported that in experimentally induced lactic acidosis in goats the concentration of protozoa was moderate to high at 12 h. From 24 h to 96 h the rumen motility was absent to low and the protozoa was totally absent.

no floatation after induction of lactic acidosis in goats.

2.2.3.3 Methylene Blue Reduction Time. (MBRT)

Methylene blue reduction time in healthy animals ranged from 3.33 to 4.33 min in goats (Shihabudheen, 1998).

In cattle with lactic acidosis, Rosenberger (1979) observed MBRT of more than five min and Dirksen (1983) noted prolongation of the MBRT at pH 5 or below.

Basak *et al.* (1993a) found that MBRT was 11.48 ± 0.45 min in healthy goats which increased to 54.34 ± 5.00 min after 12 h of induction of acidosis in goats and was absent by 24 h.

In induced lactic acidosis in goats, Shihabudheen (1998) noticed a significant increase in MBRT from 11.36 ± 1.45 min at 12 h to 38.33 ± 4.77 min at 72 h of study.

2.2.3.4 Total Volatile Fatty Acid. (TVFA)

Total volatile fatty acid concentration of rumen liquor in normal goats were reported as 72.08 ± 1.89

mEq/l (Rai *et al.*, 1972), 67.84 ± 4.66 mEq/l (Verma *et al.*, 1975), 69.07 ± 2.07 mEq/l (Pillai, 1988), 58.60 ± 8.10 mEq/l (Basak *et al.*, 1993a), 64.83 ± 2.89 mEq/l (Lal *et al.*, 1993) and 47.17 to 65.83 mEq/l (Shihabudheen, 1998).

A decrease in rumen liquor TVFA concentration from 96.2 mEq/l at zero hour to 72.4 mEq/ l at 24 h was reported in lactic acidosis of buffaloes (Prasad, 1977).

Rai and Pandey (1980) reported a rumen fluid TVFA concentration of 96.18 ± 2.69 mEq/l in stall fed goats and 76.06 ± 3.22 mEq/l in range browsing goats.

Dirksen (1983) noticed a decline in the rumen fluid TVFA in cattle with lactic acidosis.

An initial increase in TVFA to 77.35 ± 4.02 mEq/l at 24 h and then a reduction to 42.98 ± 2.24 mEq/ l by 48 h after induction of acidosis in goats was reported by Pillai (1988).

An increase in TVFA concentration of rumen liquor was recorded as 148.33 ± 15.47 mEq/l at 12 h, which

later decreased to 44.00 ± 2.25 mEq/ l at 24 h. It was then decreased to 31.66 ± 1.82 mEq/l at 48 h of induction of lactic acidosis in goats (Lal *et al.*, 1989).

Basak *et al.*, (1993a) observed a reduction in TVFA content to 6.00 ± 0.71 at 36 h and then increased to 60.00 mEq/ l at 120 h of induction of acidosis in goats.

According to Suda *et al.*, (1996) there was no variation in rumen liquor TVFA concentration at 6 h after induction of acidosis in goats but was significantly lowered at 24 h and 48 h .The TVFA concentration then increased to the initial level at 72 h.

In induced lactic acidosis in goats, Shihabudheen (1998) reported an initial rise in TVFA content of rumen liquor from the zero hour value of 57.50 ± 8.24 mEq/l to 86.17 ± 7.39 mEq/l at 12 h. The author further reported a reduction in TVFA content of rumen liquor to 24.83 ± 2.45 mEq/ l at 24h and to 23.85 ± 2.48 mEq/l at 72 h of study.

2.2.3.5 Rumen liquor lactic acid

Verma *et al.* (1975) reported normal lactic acid level of rumen in goats ranged from 3.0 to 4.5 mg / dl, while in bovines it was found to be 10.0 to 30.0 mg / dl (Rosenberger, 1979). Pillai (1988) reported that normal lactic acid concentration in rumen liquor ranged from 19.52 ± 0.95 to 21.33 ± 1.35 mg / dl in healthy goats.

The lactic acid in rumen reached maximum concentration at lower pH values and lactate was metabolised much more slowly at pH less than 6.3 (Bruno and Moore, 1962).

A peak ruminal lactic acid concentration of 130.50 mg / dl was reported by Ahrens (1967) in induced lactic acidosis in heifers.

The absorption of lactic acid and its salts from rumeno-reticulum is a relatively slow process and it is high when the pH of the rumen contents falls below pH 5 (Mackenzie, 1967).

Vihan *et al.* (1973) reported a mean lactic acid level in rumen fluid of 21.97 ± 10.3 mg / dl in clinical cases of acid indigestion in zebu cattle.

An elevated rumen lactic acid concentration of 21.0 mg / dl, two hours after feeding of concentrates in goats was reported by Verma *et al.* (1975).

Sen *et al.*, (1982) reported that in experimentally induced acid indigestion in goats, lactic acid level increased to 499.55 ± 148.33 mg / dl during the first 12 h of induction.

Huber *et al.*, (1984) reported a rumen lactic acid concentration of 1048 ± 368 mg / dl at 48 h in lactic acidosis in sheep induced with glucose administration.

According to Vihan and Rai (1985), the level of lactic acid in acidotic sheep and goat was higher (40.10 ± 5.12 mg / dl and 49.90 ± 3.83 mg / dl respectively), when compared to that of simple indigestion (24.00 ± 5.72 and 21.20 ± 2.08 mg / dl).

In induced lactic acidosis in goats, Cao *et al.* (1987) reported that the rumen liquor lactate

concentration progressively increased to 36.00 mg / dl at 24 h of study.

Pillai (1988) observed mean lactic acid concentration in rumen liquor as 20.53 ± 1.06 mg / dl at zero hour and it increased to 374.14 ± 31.98 mg / dl at 48 h of induction of lactic acidosis in goats.

Lal *et al.* (1989) reported that the level of lactic acid increased from 4.08 ± 0.31 mg / dl at zero hour to 295.13 ± 16.83 mg / dl at 12 h and was 13.39 ± 1.53 mg / dl at 96 h of induction of lactic acidosis in goats.

In subacute lactic acidosis, the lactic acid concentration in rumen liquor was 181.65 ± 12.76 mg / dl, whereas in acute lactic acidosis it was 337.81 ± 13.49 mg / dl after 24 h of induction in buffalo calves (Randhawa *et al.*,1989).

An increase in rumen liquor lactic acid concentration from 2.66 ± 0.10 mg / dl at zero hour to 184.18 ± 14.22 mg / dl at 24 h in molasses induced subacute lactic acidosis in buffalo calves was reported by Ahuja *et al.* (1990).

In induced lactic acidosis in goats, Basak *et al.* (1993a) reported lactic acid concentration in rumen liquor as 6.06 ± 0.32 mg / dl at zero hour and it rised to a maximum of 326.08 ± 10.96 mg / dl at 24 h and then reduced to 53.13 mg / dl at 120 h.

The L (+) lactate concentration in rumen liquor of acidotic goats was 1395.70 ± 490.40 mg / dl and D (-) lactate was 1381.30 ± 791.80 mg / dl at 12 h of induction and were 571.00 ± 777.50 mg / dl and 1737.30 ± 2411.00 mg / dl respectively at 72 h of induction (Suda *et al.*, 1996).

2.3 Haematology

2.3.1 Packed Cell Volume.

Haematocrit values in healthy goats were reported as 31.96 per cent (Bhargava, 1980), 24 to 48 per cent (Jain, 1986), 34.55 ± 0.57 per cent (Lal *et al.*, 1990), 34.01 ± 0.68 per cent (Das and Misra, 1991), 22.28 per cent (Radostits *et al.*, 1994) and 26.00 to 29.83 per cent (Shihabudheen, 1998).

Dirksen (1983) and Michell (1990) observed a rise in haematocrit value in cattle with acidosis.

Tanwar and Mathur (1983) reported an increase in PCV up to 72 h of induction of acute lactic acidosis in goats. Lal *et al.* (1990) also observed an increased packed cell volume from 34.55 ± 0.57 at zero hour to 40.60 ± 0.61 at 72 h of induced ruminal acidosis in goats.

An elevation in packed cell volume was noticed by Das and Misra (1991) to 34.32 ± 0.95 per cent at 12 h of induction of acidosis in goats.

According to Braun *et al.* (1992), an increase in haematocrit was observed in 38 per cent of clinical cases of acute lactic acidosis in sheep and goats.

An increase in packed cell volume was reported by Basak *et al.* (1993b) from 22.66 ± 0.33 per cent at zero hour to 41.40 ± 0.61 per cent at 48h of induction of lactic acidosis in goats.

Sen *et al.* (1993) observed an increase in PCV after induction of acidosis and recorded a highest value of 37.70 ± 1.90 per cent at 24 h of induction in goats.

An elevation of packed cell volume to 34.32 ± 0.95 per cent at 12 h of induction of acidosis in goats (Radostits *et al.*, 1994).

Shihabudheen (1998) reported an increase in haematocrit to 33.83 ± 0.98 per cent and a cell fluid ratio of 0.52 ± 0.02 per cent at 48 h of induction of lactic acidosis in goats.

2.3.2 Haemoglobin

In normal healthy goats the haemoglobin content was reported to be 7.70 g / dl (Vaidya *et al.*, 1970), 10.09 g/ dl (Bhargava, 1980), 8 to 14 g / dl (Benjamin 1985), 8.76 ± 0.32 g/ dl (Basak *et al.*, 1993b) and 12.77 to 14.0 g/ dl (Shihabudheen, 1998).

A rise in haemoglobin level up to 17.8 g per cent was reported in acidotic cattle by Dirksen (1965).

Tanwar *et al.* (1983) reported an increase in haemoglobin up to 72 h of induction of acute lactic acidosis in goats.

The haemoglobin level increased from 8.76 ± 0.32 g/ dl at zero hour to 10.76 ± 2.50 g/ dl at 48 h of

induced acid indigestion in goats (Basak *et al.*, 1993b).

According to Angelov *et al.* (1995) an increase in haemoglobin concentration to 12.28 g/ dl was observed at 12 h of induction of acidosis in goats.

Shihabudheen (1998) reported that the haemoglobin concentration increased to 16.23 ± 0.65 g / dl at 24 h, remained high up to 72 h and reached normal at 96 h in induced lactic acidosis in goats.

2.3.3 Total erythrocyte count

Erythrocyte count in normal goat was reported to be 10.96 x 10^6 /µl (Sharma *et al.*, 1973), 10.12 x 10^6 /µl (Bhargava, 1980), 12.35 ± 0.35 x 10^6 /µl (Pyne *et al.*, 1982), 8 to 18 x 10^6 /µl (Jain, 1986), 8.82 ± 0.38 x 10^6 /µl (Basak *et al.*, 1993b), and 14.70 to 16.14 x 10^6 /µl (Shihabudheen, 1998).

Tanwar *et al.* (1983) reported that there was an increase in erythrocyte count in goats up to 36 h of induction of acidosis.

An increase in erythrocyte count to 10.20 ± 0.32 x 10^{6} /µl at 48 h was noticed after induction of lactic acidosis in goats by Basak *et al.* (1993b). Shihabudheen (1998) also reported an increase in erythrocyte count from 15.03 ± 0.53 at zero hour to 17.97 ± 0.83 at 48h of induction of lactic acidosis in goats.

2.3.4 Total leukocyte count

Total leukocyte count in healthy goats were reported to be 12.50 x 10^{3}/µl (Sharma *et al.,* 1973), 10.09 x 10^{3}/µl (Bhargava, 1980), 10.18 ± 0.24 x 10^{3}/µl (Pyne *et al.*, 1982), 6 to 16 x 10^{3} /µl (Benjamin, 1985), 4.13 x 10^{3}/µl (Radostits *et al.*, 1994) and 15.15 ± 0.86 x 10^{3}/µl (Shihabudheen, 1998).

In experimental lactic acidosis in goats, Tanwar *et al.,* (1983) observed an increase in leukocyte count from 12 h to 36 h.

A rise in leukocyte count in induced acidosis in goats was reported by Basak *et al.* (1993b) and observed the highest value of 18.35 ± 0.72 x 10^{3} /µl at 60 h of the experiment.

Shihabudheen (1998) noticed an increase in leukocyte count from 15.92 ± 1.80 x 10^3/ µl at zero hour to 24.83 ± 5.24 x 10^3/ µl at 96 h of induction of lactic acidosis in goats.

2.3.5 Differential leukocyte count

Pyne *et al.* (1982) reported differential leukocyte count in healthy goats as neutrophils 34.61 ± 0.65 per cent, eosinophils 2.04 ± 0.14 per cent, basophils 0.04 ± 0.02 per cent, lymphocytes 60.25 ± 0.14 per cent and monocytes 3.06 ± 0.16 per cent. According to Benjamin (1985) the values were 30 to 48 per cent, 3 to 8 per cent, 0 to 2 per cent, 50 to 70 per cent and 1 to 4 per cent, respectively.

The range of neutrophils, eosinophils, basophils, lymphocytes and monocytes in healthy goats were 35.67 ± 1.82 to 39.67 ± 1.65 per cent, 2.50 ± 0.56 to 3.83 ± 0.54 per cent, 0.83 ± 0.31 to 1.83 ± 0.65 per cent, 55.67 ± 2.23 to 59.83 ± 1.87 per cent and 0.17 ± 0.17 to 0.50 ± 0.22 per cent respectively (Shihabudheen, 1998).

A significant reduction in lymphocytes and an increase in neutrophils with a left shift were reported by Dash *et al.* (1972) in clinical cases of lactic acidosis in cattle.

Tanwar *et al.*(1983) reported no appreciable changes in differential leukocyte count of acidotic goats.

In ruminal acidosis of goats, Basak *et al.* (1993b) observed an increase in neutrophils from 42.83 ± 2.24 per cent at zero h to 84.75 ± 3.77 per cent at 42 h and a reduction in lymphocytes from 53.33 ± 2.53 at zero hour to 14.80 ± 1.3 per cent at 48 h along with significant reduction in monocytes and eosinophils.

Shihabudheen (1998) reported neutrophilia and lymphopenia with no significant changes in eosinophils, monocytes and basophils in induced lactic acidosis in goats.

2.4 Serum biochemistry

2.4.1 Serum bicarbonate

Braun *et al.* (1992) observed normal venous blood bicarbonate as 24.7 ± 2.0 mmol / l in sheep and goats.

Radostits *et al.* (1994) reported that the normal bicarbonate level in blood of sheep was 12 to 24 mmol/l.

Estimation of serum bicarbonate can be done inexpensively and with good clinical precision to assess the degree of metabolic acidosis using venous whole blood (Michell, 1990). The author further reported that in severe acidosis a deficit in plasma bicarbonate of up to 20 mEq/ l occurred in ruminants.

Braun *et al.* (1992) observed a venous blood bicarbonate level of less than 20 m mol/l in 50 per cent of the clinical cases of lactic acidosis in sheep and goats.

A progressive decrease in serum bicarbonate level in induced lactic acidosis was reported by Randhawa *et al.* (1993) in buffalo calves.

Sen *et al.* (1993) reported a decrease in serum bicarbonate level from a normal value of 24.6 ± 2.2 mEq/l to 22.0 ± 1.2 mEq / l after 4 h of induction of

ruminal acidosis in goats. The lowest value recorded was 9.0 ± 3.13 mEq / l at 44 h of grain overload.

2.4.2 Total serum protein

Total serum protein in normal goats ranged from 7.78 ± 0.26 g / dl (Varma, 1967), 6.40 to 7.90 g / dl (Benjamin, 1985), 6.43 ± 0.06 g / dl (Pillai, 1988) and 7.06 ± 0.25 g / dl (Braun *et al.*, 1992).

In clinical cases of indigestion in cattle and buffaloes, Prasad *et al.* (1972) observed a mean total serum protein of 9.9 ± 1.42 g / dl.

In acid indigestion Prasad *et al.* (1973) noticed a decrease in total protein from 8.3 g / dl at zero hour to 6.90 g / dl at 72 h in buffaloes.

Vihan *et al.* (1982), Vihan and Rai (1985) and Teli *et al.* (1986) could not observe any changes in the values of total serum protein in acidotic goats.

No significant variation in total serum protein was noticed in induced lactic acidosis in goats (Pillai, 1988).

Braun *et al.* (1992) reported that only 35 per cent clinical case of ruminal acidosis in sheep and goats showed hypoproteinemia.

2.4.3 Serum lactic acid.

Normal level of blood lactic acid in goats reported by various authors, ranged from 8.25 to 10.45 mg / dl (Varma *et al.*, 1975), 13.02 ± 1.02 mg / dl (Pillai, 1988), 10.72 ± 0.84 to 11.76 ± 0.91 mg / dl (Das and Misra, 1991), 9.27 ± 0.39 mg / dl (Lal *et al.*, 1992), 9.8 mg / dl (Lonkar and Prasad, 1993) and 15.20 ± 0.5 to 16.78 ± 0.78 mg / dl (Shihabudheen, 1998).

In rumen acidosis of cattle, Dirksen (1965) reported a rise in blood lactic acid and recorded a maximum of 69.80 mg / dl.

Huber (1969) noticed reduced renal blood flow with blood lactic acid concentration greater than 17.70 mg / dl and glomerular filtration rate reduced with blood lactic acid concentation greater than 30.60 mg / dl in sheep.

In acid indigestion, Vihan *et al.* (1973) observed a serum lactic acid concentration of 24.30 ± 6.88 mg / dl in zebu cattle.

Vihan *et al.* (1982) reported that serum lactic acid concentration increased to 38.30 mg / dl in 42 h of grain fed acidosis and to 48.0 mg / dl in 12h in cane sugar acidosis in goats.

According to Tanwar *et al.* (1983) the blood lactic acid concentration of 9.45 ± 0.54 mg / dl at zero hour rose to 27.30 ± 1.32 mg / dl at 12 h and then to 40.57 ± 5.20 mg / dl in severe lactic acidosis in goats.

Huber *et al.* (1984) observed blood lactate of 78.2 ± 20.00 mg / dl of which D-lactate was 27.10 ± 21.60 mg / dl and L-lactate was 51.10 ± 13.50 mg / dl in sheep with lactic acidosis.

Vihan and Rai (1985) reported serum lactic acid concentration of 15.00 ± 2.41 mg / dl in simple indigestion and 30.80 ± 2.30 mg / dl in clinical cases of lactic acidosis in goats.

An increase in lactic acid level to 30.98 ± 2.06 mg / dl at 48 h of induction of acidosis and a reduction to 17.08 ± 0.64 mg / dl at 96 h was noted by Pillai (1988) in goats.

Ahuja *et al.* (1990) reported an increase in blood lactic acid concentration from 18.52 ± 0.36 at zero hour to 98.60 ± 5.39 at 24 h in induced lactic acidosis in buffalo calves.

An increase in blood lactic acid concentration from 9.27 ± 0.39 at zero hour to 20.10 ± 1.82 at 12 h of induced lactic acidosis in goats (Lal *et al.*, 1990).

Das and Misra (1991) reported an increase in blood lactic acid concentration up to 24 h of induction of lactic acidosis in goats.

In acute lactic acidosis in buffalo calves, the rapid rise in blood lactic acid concentration between 24 h and 48 h following induction was not only of gastro intestinal origin, but also from tissues as a result of failure in aerobic metabolism in view of the

decreasing systemic blood pressure leading to a decrease in tissue perfusion. (Randhawa *et al.*, 1993)

According to Sen *et al.* (1993) an increase in lactic acid concentration from 13.0 ± 2.9 mg / dl at zero hour to 21.3 ± 4.5 mg/ dl at 8 h and the peak blood lactic acid concentration recorded was 41.4 ± 12.4 mg / dl at 32 h of induced acidosis in goats.

Radostits *et al.* (1994) reported a rise in blood lactate level in acidosis in ruminants.

A rise in lactate level to 8.30 mmol / l at 12 h after induction of acidosis in goats (Angelov *et al.*, 1995)

According to Suda *et al.* (1996) the serum D(-) and L(+) lactate levels were elevated by 6 h and 12 h respectively after induction of ruminal acidosis in goats with sucrose. The values reached four fold and twenty five fold of the initial levels respectively at 32 h after induction.

Shihabudheen (1998) recorded a rise in serum lactate from 15.74 ± 0.88 at zero hour to 69.01 ± 4.56 at 48 h of lactic acidosis in goats.

2.4.4 Serum potassium

The serum potassium level in healthy goats were reported as 4.56 mEq/l (Boss and Wanner, 1977), 4.68 ± 0.41 to 4.95 ± 0.43 mEq/l (Das and Misra, 1991) and 4.85 ± 0.08 to 4.96 ± 0.11 mEq/l (Shihabudheen, 1998).

In cattle with ruminal acidosis, Dirksen (1965) observed blood potassium values at the lowest limit of normal or below in 14 animals.

Sinha *et al.* (1985) reported a decrease in serum potassium level from 38.31 ± 5.44 to 29.05 ± 4.81 mEq/l at 24 h in buffalo calves with induced ruminal acidosis.

A decrease in serum potassium level was noticed from 6.26 ± 0.08 mEq/l at zero hour to 4.98 ± 0.26 at 24 h and then increased to 5.91 ± 0.14 at 192 h of induction of lactic acidosis in buffalo calves (Ahuja *et al.*, 1990).

According to Das and Misra (1991) a decrease in serum potassium level was noticed at 72 h in induced lactic acidosis in goats.

Braun *et al.* (1992) reported a normal serum potassium level in 73 per cent of clinical cases of acute ruminal acidosis in sheep and goats.

A reduction in potassium level in acidotic sheep was reported by Patra *et al.* (1993).

Sen *et al.* (1993) found that serum potassium decreased to 3.7 ± 0.2 mEq/l by 44 h of induction of acidosis in untreated goats whereas no change was noticed in animals treated with parenteral sodium bicarbonate and fluids. However Shihabudheen (1998) reported that serum potassium level increased from 4.71 ± 0.17 mEq/ l at zero hour to 5.97 ± 0.08 mEq/ l at 96 h of induction of lactic acidosis in goats.

2.5 Therapeutic management

2.5.1 Systemic alkalinizers

Prasad *et al.* (1973) reported effective use of 7.5 per cent sodium bicarbonate solution intravenously at

24 h and 36 h in treatment of induced rumen acidosis in buffaloes.

Administration of 7.5 per cent solution of sodium bicarbonate 20-100ml in sheep and goat and 100-300ml in cattle intravenously once or twice daily for one to two days was effective in treating moderate to severe cases of rumen acidosis (Prasad and Rekib, 1975).

Sethuraman and Rathor (1979) reported complete recovery at 192 h of treatment with Ringers sodium lactate solution and sodium bicarbonate intravenously in two separate groups along with oral alkalinizers and supportive therapy in induced acid indigestion in bovines.

According to Arieff *et al.* (1982), the lactate production increased with sodium bicarbonate, but not with NaCl infusion in treatment of lactic acidosis in dogs. Bicarbonate decreased hepatic portal vein blood flow and caused significant decrease in liver and erythrocyte pH and cardiac output.

Tanwar *et al.* (1983) noticed effective use of Ringer's lactate solution @ 25ml/Kg body weight

intravenously for three days in treating rumen acidosis in goats.

Filley and Kindig (1984) reported that carbicarb not only did not raise PCO_2 but can lower it under certain conditions. They also noticed that carbicarb raised blood pH more, than one molar sodium bicarbonate in the same dose.

Graf *et al.* (1985) noticed a reduction in liver intracellular pH on administration of sodium bicarbonate parenterally due to increased carbon dioxide load to the liver in hypoxic lactic acidosis in dogs.

Speedy recovery was noticed with five per cent sodium bicarbonate solution at the rate of 1.50 ml per Kg body weight as slow intravenous infusion at 24 h of induction of ruminal acidosis in buffalo calves along with other treatments (Sinha *et al.*, 1985).

In lactic acidosis, when sodium bicarbonate is given intravenously, the carbon dioxide generated diffuses more rapidly than bicarbonate across cell membranes and into the cerebrospinal fluid. Thus

intracellular and cerebrospinal fluid pH can actually fall in response to sodium bicarbonate administration (Stacpoole, 1986).

Bersin and Arieff (1988) reported that a mixture of sodium carbonate and sodium bicarbonate called 'carbicarb' which buffered similar to sodium bicarbonate, but has the advantage of reduced net generation of CO_2 and was superior to sodium bicarbonate in treating hypoxic lactic acidosis in dogs. Its administration results in improved arterial blood gases, tissue pH, lactate production, increased muscle glycogen consumption and cardiac haemodynamics like improved stroke volume and myocardial contractility and also suggested that these effects were due to reduced CO_2 generation.

Effective treatment of acidotic calves having a base deficit of 16.30 mEq/l with 2.4 litre of 1.3 per cent sodium bicarbonate parenterally was reported by Naylor (1989).

Michell (1990) suggested parenteral use of a safer solution called carbicarb, a mixture of sodium

carbonate and sodium bicarbonate for lactic acidosis in sheep and goats.

Successful recovery was noticed by Das and Misra (1991) using 2.50 per cent solution of sodium bicarbonate parenterally at the rate of 3 ml per kg body weight twice on 1st day and once on 2nd and 3rd day to correct systemic acidosis in goats at 24 h with induced lactic acidosis.

In the absence of blood gas analysis base deficit may be estimated as -4 to -6 m mol/l in mild acidosis, -8 to -11 m mol/l in moderate acidosis and -14 to -16 m mol/l in severe acidosis and use of five per cent sodium bicarbonate solution intravenously in clinical case of lactic acidosis in sheep and goats were recommended by Braun *et al.* (1992).

White (1993) observed 20 m mol/l of bicarbonate deficit in peracute lactic acidosis in a calf weighing 60 Kg and successfully treated with 240 m mol of 0.5M sodium bicarbonate solution intravenously at the rate of 4 m mol/kg in first half an hour, and 250 m mol of sodium bicarbonate over next 30 min, after estimating the bicarbonate deficit as 13 m mol/l after first

treatment. The bicarbonate concentration in blood became normal after 15 h of treatment.

White and White (1993) reported that in calves, administration of 5-10 litres of electrolyte solution containing 144 m mol/l sodium, 4 m mol/l potassium, 113 m mol/l chloride, 35 m mol/l bicarbonate with up to 450 ml of 1 molar sodium bicarbonate was effective in treating severe metabolic acidosis.

According to Radostits *et al.* (1994), the systemic acidosis could be corrected with 5 per cent sodium bicarbonate solution intravenously at the rate of 5 litre for 450 Kg animal over a period of 30 min followed by 1.3 per cent isotonic sodium bicarbonate at 150ml per Kg body weight over 6-12 h in severe lactic acidosis in cattle.

2.5.2 Oral alkalinizers

For treating ruminal acidosis in cattle, Dirksen (1965) suggested administration of antacids like 200g calcium carbonate or 100g magnesium oxide orally.

Joshi (1969) reported successful recovery of a case of acid indigestion in bullock with two ounce each

of Bismuth carbonate and sodium bicarbonate orally and repeated after two hours.

Complete recovery in moderate cases of lactic acidosis in goats was noticed with sodium bicarbonate 20 g, calcium carbonate 20 g and Magnesium carbonate or Bismuth carbonate 4g orally along with other supportive therapy (Gnanaprakasam, 1970).

Prasad *et al.* (1973) reported use of sodium bicarbonate 60 g orally at 24 h and 36 h for treatment of induced rumen acidosis in buffaloes.

Oral dosing of magnesium carbonate or sodium bicarbonate at half pound initially followed by an ounce daily for two days were found to be effective in correction of pH of rumen liquor in acid indigestion in cattle (Misra and Singh, 1974).

Prasad and Rekib (1975) reported use of sodium bicarbonate in sheep, goats and cattle orally for one to two days in treating clinical cases of rumen acidosis.

In cattle and buffaloes with induced acid indigestion, Sethuraman and Rathor (1979) reported effective therapy with sodium bicarbonate and magnesium carbonate orally in separate groups.

Tanwar and Mathur (1983) reported use of magnesium carbonate at the rate of 1g per Kg body weight intraruminally in treatment of lactic acidosis in goats.

Magnesium hydroxide 120 g intraruminally and 60 g orally every 6 h till rumen pH became normal in induced ruminal acidosis in buffalo calves was found to be effective (Sinha *et al.,* 1985).

Michell (1990) suggested administration of magnesium hydroxide at the rate of 1g / kg body weight orally for treating lactic acidosis in ruminants.

Use of magnesium carbonate at the rate of 1g / kg body weight intraruminally for four times a day to correct rumen pH was effective in treating goats with ruminal acidosis (Das and Misra, 1991).

2.5.3 Rehydration therapy

In clinical cases of rumen acidosis in cattle, Dirksen (1965) reported administration of physiological saline parenterally to correct dehydration.

Gnanaprakasam (1970) reported use of normal saline 500 ml intravenously for treating severe cases of lactic acidosis in goats.

Sinha *et al.* (1985) suggested effective use of 4 liters of 0.85 per cent sodium chloride as slow intravenous infusion at 24 h followed by two litre at 36 h in induced ruminal acidosis in buffalo calves.

Use of 0.9 per cent sodium chloride injection at the rate of 10ml/Kg body weight parenterally 3-4 times daily was effective in treatment after 24 h of induced lactic acidosis in goats (Das and Misra, 1991).

Braun *et al.* (1992) reported administration of isotonic sodium chloride solution at the rate of 300 ml per hour for one to two days and an average volume of 3.9 liters ranging from one to eight liters in clinical cases of lactic acidosis in sheep and goats.

2.5.4 Supportive therapy

In treatment of ruminal acidosis in cattle, Dirksen (1965) observed beneficial effects in administration of rumen fluid from healthy animals, vitamin B_1 injection and suggested that improved medical therapy could be applied successfully in severe cases. The author further reported that rumenotomy should be limited to fresh cases with severely overloaded rumen.

Gnanaprakasam (1970) reported use of 25 per cent calcium borogluconate injection 60ml subcutaneously, 0.5g strepto penicillin injection intramuscularly and rumen cud transplant in severe cases of lactic acidosis in goats, and in moderate cases antihistaminics and 100mg thiamine intramuscularly along with yeast tablets and oral antacids.

Administration of desiccated rumen liquor at 12 h of treatment was beneficial in rumen acidosis in buffaloes (Prasad *et al.*, 1973).

Vestweber *et al.* (1974) noticed return of ruminal pH to normal in 24 h after removal of ruminal contents through a rumen fistula in acidotic sheep. He also reported that parenteral thiamine therapy had a

protective effect against increased lactate in ruminal acidosis of sheep.

Fresh rumen cud transplant 150ml to one litre orally for one to three days helped in treatment of clinical cases of rumen acidosis in ruminants (Prasad and Rekib, 1975).

Sethuraman and Rathor (1979) reported effective treatment with penicillin, cud from healthy animals and rumenotoric drugs orally, thiamine hydrochloride and liver extract parenterally in cattle and buffaloes with induced acid indigestion.

According to Tanwar and Mathur (1983), use of benzyl penicillin 50,000 IU/Kg body weight intraruminally, thiamine hydrochloride injection 10mg/Kg body weight, antihistaminics for three days, liver extract injection intramuscularly and 250 ml of fresh rumen liquor intraruminally on 4th and 5th days were effective in treatment of induced rumen acidosis in goats.

Successful treatment was reported by Sinha *et al.*, (1985) with removal of rumen contents through rumen

fistula, oxytetracycline 10 mg/kg body weight orally, thiamine hydrochloride injection, one liter of fresh rumen cud transplant, 100 g molasses and 50 mg cobalt chloride intraruminally, liver extract with vitamin B_{12} injection intramuscularly for three days and thiamine hydrochloride injection one gram intravenously at 24 h of induction of ruminal acidosis in buffalo calves.

Michell (1990) suggested treatment with corticosteroids, antihistaminics, antibiotics, thiamine and calcium borogluconate injection in lactic acidosis of ruminants.

Das and Misra (1991) reported use of tetracycline hydrochloride powder at the rate of 20 mg per kg body weight, intraruminal administration of 500 ml of rumen cud along with 150 ml rice gruel and 50 g molasses after correction of rumen pH, antihistaminics intraruminally and intramuscular injection for 2 days and liver extract injection for 5 days made quick recovery in ruminal acidosis in goats.

According to Braun *et al.* (1992), use of 500mg thiamine hydrochloride intravenously, yeast orally, three to five injections of oxytetracycline

intravenously at the rate of 10mg per kg at 24 h intervals and 500 ml to one litre of rumen fluid orally one to five times at one day intervals were effective in treatment of clinical cases of ruminal acidosis in sheep and goats.

3. MATERIALS AND METHODS

3.1 Clinical study

The study was conducted from January 1999 to December1999 in adult goats of either sex about 1-4 years of age weighing 15-25 Kg brought to the Veterinary college hospitals at Mannuthy and Kokkalai with history and symptoms suggestive of ruminal lactic acidosis. A complete history, signalment followed by a detailed clinical examination of each case was carried out as suggested by Rosenberger (1979). They were divided into two groups of six each (Group I and Group II) at random. Different therapeutic management mentioned under **3.3** was given for four days to each group. Clinical data was recorded at zero hour, one hour, six hour, 12h, 24h 48h and 72h of study. Values from six healthy animals maintained under similar conditions served as controls.

3.2 Sampling and analysis

Sampling and analysis of rumen liquor and blood was done from the control animals only once. In groups I and II, the samples of rumen liquor and blood were collected at zero hour, one hour, six hour, 12 h, 24 h, 48 h and 72 h of

clinical study for analysis. During this period the clinical signs and other parameters of the disease were recorded in animals of group I and II.

3.2.1 Rumen liquor

Rumen liquor was collected from the animals in all the groups into glass bottles through a stomach tube connected to a rumen liquor pump. Approximately 100 ml was collected each time. The pH of rumen liquor was noted immediately after collection at zero hour, one hour, six hour, 12 h, 24 h, 48 h and 72 h by placing a drop directly on a strip of standard wide range pH paper. Examination of physical, microbial and biochemical characters were carried out at zero hour, 24 h, 48 h and 72 h of the study. After noting the physical characters like colour, consistency and odour, one drop of the sample was examined under microscope to determine protozoal concentration and motility (Rosenberger, 1979). Rumen contents were strained through a four layer muslin cloth and the strained rumen liquor (SRL) was used for determining the sedimentation activity time (SAT) according to the method described by Nichols and Penn (1958). Methylene blue reduction time (MBRT) was noted by the method suggested by Dirksen (1983). Total volatile fatty acid (TVFA) was estimated by the method of Barnett

and Reid (1957) and lactic acid estimation was done as per Noll (1974).

3.2.2 Blood

Approximately 5 ml of blood was collected from the jugular vein in a dry glass vial with EDTA (1-2 mg/ml) as anticoagulant for hematological studies. 10 ml of blood was collected separately in a screw capped centrifuge tube for separation of serum. Haematocrit and serum bicarbonate were estimated in controls and at zero hour, one hour, six hour, 12 h, 24 h, 48 h and 72 h in animals of groups I and II. Haemoglobin, total RBC count, total WBC count, differential leukocyte count, serum lactic acid, serum protein and serum potassium was estimated at zero hour, 24 h, 48 h and 72 h of the study in animals of group I and II.

Haematology was done as per the procedures described by Jain (1986). Estimation of serum bicarbonate was done as per Norris *et al.* (1975). Serum lactate was estimated as per Noll (1974). Serum protein was estimated by modified biuret method (Weichselbaum, 1946) and serum potassium was estimated using Atomic absorption Spectrometry (Perkin - Elmer model-3380).

3.3 Therapeutic management.

Dexamethasone (20 mg) was given intravenously in all animals of group I and II. The animals were held in sternal recumbency and the stomach tube was passed to collect the rumen liquor.

The animals of group I were given five per cent sodium bicarbonate solution intravenously at a dose rate of 5 ml per Kg body weight (3 mEq / kg body weight) (Braun *et al.*, 1992).

The rumen liquor was then evacuated through a stomach tube connected to a vacuum pump and removed as much contents after irrigating with 1000 ml of normal saline.

Magnesium hydroxide powder at the dose rate of 1 g per kg body weight and oxytetracycline powder 500 mg was given orally through a stomach tube. Dehydration was then corrected using 1000 ml to 2000 ml (50 ml per kg body weight) of normal saline intravenously. Supportive treatment was done with vitamin B complex injection (Beplex forte- 3 ml) and transfer of fresh rumen fluid was done on 2nd or 3rd day. Parenteral fluids and Vitamin B complex

injection were continued every 24 h till the appetite and rumen motility became normal.

For the second group sodium bicarbonate infusion was replaced by a 5 per cent mixture of sodium carbonate and sodium bicarbonate (carbicarb) (Bersin and Arieff, 1988) intravenously at a dose rate of 4 ml per kg body weight (3 mEq / kg body weight). All the other treatments as in group I were repeated in group II also.

3.4 Statistical analysis

The control values were compared with the pre treatment (zero hour) and post treatment values of group I and group II animals. The results obtained from group I and group II at different intervals were also compared. Statistical analysis was done according to the method described by Snedcor and Cochran (1967).

RESULTS

4. RESULTS

4.1. Clinical signs

All the animals in group I and II were either inappetant or anorectic. They were dull and depressed, had low carriage of head and the muzzle was dry. Except in one animal in group I, all animals had sunken eyes, tenting of skin and grinding of teeth.

One animal in group I and two animals in group II were presented with severe acidosis. They were on lateral recumbency with retracted eyeballs, dry muzzle and distended abdomen. The extremities were cold and showed grunting on respiration. One of the animals in group II had frequent regurgitation of rumen fluid through mouth and nostrils even during treatment, and it passed on to coma stage, died at 8th hour of treatment.

In all other animals of group I and II, there were mild tympany. On palpation of rumen, the ruminal contractions were feeble or absent. On auscultation of rumen there was gurgling sound and had fluid thrill on percussion of rumen.

Oliguria was noticed in all the cases and presence of profuse foul smelling diarrhoea noticed in seven animals. Soft dung was noticed in five animals. Out of these five animals three turned diarrhoeic after 24th h of treatment.

The clinical data recorded are presented in Table 1,2 and 3.

4.1.1. Respiration

The normal respiratory rate recorded in control animals was 21.33 ± 0.67 per minute.

In group I, the respiratory rate was significantly increased ($p \leq 0.01$) to 41.33 ± 2.23 per minute at zero hour, reached a maximum of 42.50 ± 2.22 per min at 1 h and reduced to a normal value of 21.83 ± 0.48 per min at 72 h of treatment (Table 2, Fig. I). In group II respiratory rate reduced from a significantly increased ($p \leq 0.01$) value of 43.00 ± 1.52 per min at zero hour to a normal value of 21.83 ± 0.61 at 48th h of treatment (Table 3, Fig, I).

At zero hour, no significant difference was noticed in the respiratory rate between groups I and II. In group II, at 1 h, 6 h, 12 h, 24 h and 48 h of treatment there was

significant reduction ($p \leq 0.01$) in the respiratory rate when compared to group I. From 72 h onwards, there was no significant difference in the respiratory rate between groups I and II.

4.1.2.Pulse

In control animals, the pulse was stronger in all animals with a mean pulse rate of 88.33 ± 1.96 per minute.

At zero hour of study, the pulse was imperceptible in all animals except one each in both groups I and II, where it was very weak. At one hour of study the pulse became palpable but was rapid and weak in one animal in group I and three animals in group II.

At 24th hour of study a weak pulse was noticed in four animals in group I and one animal in group II, whereas a strong pulse was noticed in two animals in group I and four animals in group II. At 24 h a significantly increased ($p \leq 0.01$) pulse rate of 103.67 ± 2.34 and 100.80 ± 2.33 per minute in group I and II respectively were recorded when compared to the normal values of control animals.

At 48 h also a significant increase ($p< 0.05$) in pulse rate of 97.83 ± 1.22 and 95.60 ± 2.56 per minute in group I and II respectively were noticed when compared to control values.

At 72 h, no significant difference was noticed in the pulse rate among group I and II when compared to control values.

4.1.3. Temperature

The mean rectal temperature recorded in control animals was 39.35 ± 0.22°C. A non significant decrease in mean rectal temperature was noticed from zero hour to 48 h in all the animals of group I and II when compared to control values (Table 1,2 and 3).

4.1.4. Heart rate

The mean heart rate recorded in control animals was 88.67 ± 1.98 per min. At zero hour the heart rate was significantly increased ($p\leq0.01$) to 114.0 ± 4.32 per min in group I and 118.33 ± 5.25 per min in group II. No significant difference noticed in the heart rate after treatment, from 72 h onwards among control values, group I and group II (Table 1,2 and 3, Fig. I).

4.1.5. Mucous membrane

Mucous membrane in all the control animals was pale roseate. At zero hour, the mucous membrane was congested in one animal in group I and in two animals in group II. At 24 h, the mucous membrane was pale roseate in all the animals in both groups I and II (Table 1,2 and 3).

4.1.6. Rumen motility

The amplitude of rumen contraction was strong and the motility recorded was one per minute in all control animals (Table 1). Rumen motility was absent in all the animals in group I and II at zero hour. At 24 h a feeble rumen motility of one per four minute was noticed in one animal in group II.

At 48 h, rumen motility was one per three minute in two animals of group I, and three animals of group II. At 72 h, five animals in group I and all animals of group II had a rumen motility of one per minute. No rumen motility was noticed in one animal in group I even after 72 h of treatment (Table 2,3).

4.2. Changes in rumen liquor

4.2.1. pH

The mean pH of the rumen liquor in control animals was 7.08 ± 0.08. At zero hour, the pH of the rumen liquor was found to be reduced significantly ($p<0.01$) to 4.50 ± 0.18 in group I and 4.08 ± 0.30 in group II. At one hour the pH of the rumen liquor was 6.75 ± 0.11 in group I and 6.58 ± 0.16 in group II. No significant difference noticed in the pH of the rumen liquor from 24 hour onwards in groups I and II when compared to control values (Table 4,5 and 6, Fig. II).

4.2.2. Physical characters

4.2.2.1. Colour

The colour of the rumen liquor was olive green in control animals. It was milky grey in all the animals of group I and II at zero hour and 24 h. At 48 h it turned to green in one animal in group II. At 72 h the colour of rumen liquor was green in two animals of group I and in three animals of group II (Table 7, 8 and 9).

4.2.2.2. Consistency

The consistency of rumen liquor was thick in all control animals. It was watery in all the animals in group I and II at zero hour (Table 7,8 and 9). At 24 h the consistency was semiliquid in three animals in group I and four animals in group II. At 48 h the rumen liquor turned to thick in three animals in group I and four animals in group II. At 72 h the rumen liquor was thick in five animals of group I and in all animals of group II.

4.2.2.3. Odour

The odour of the rumen liquor was aromatic in all control animals. At zero hour, it was intense sour in all the animals in group I and II. It became faintly sour at 24 h in both group I and II. At 72 h the odour of rumen liquor in all the animals became aromatic (Table 7,8 and 9).

4.2.3. Microbial activity

4.2.3.1. Rumen protozoa

In control animals, the concentration and motility of protozoa in rumen liquor was high (+++ to ++++). In all the animals of group I and II no protozoal motility was detected at zero hour. Few dead protozoa were also noticed in the rumen liquor. At 24 h, a few live small protozoa (+)

was noticed in one animal in group I and in two animals in group II.

At 48 h in group I animals, the protozoal motility ranged from ++ to nil, whereas in group II it was ++ to +++. Along with small protozoa a few large protozoa also started to appear at this stage. At 72 h the protozoal motility ranged from ++ to +++ in both groups I and II, respectively (Table 7,8 and 9).

4.2.3.2. Sedimentation activity time (SAT)

In control animals, the sedimentation activity time was 14.63 ± 0.35 min. In group I and II there was rapid sedimentation but no floatation at zero hour and 24 h.

In group I one animal showed a slight sedimentation activity in 16 minutes at 48 h. One animal in group II also showed a sedimentation activity time of 13 minutes with slight floatation at 48 h. In group I, only two animals showed normal sedimentation activity time whereas in group II, three animals showed normal sedimentation activity time at 72 h comparable to control animals (Table 8 and 9). More tendency for floatation was noticed in the rumen liquor in group II when compared to group I at 72 h of treatment.

4.2.3.4. Methylene blue reduction time (MBRT)

In control animals, the methylene blue reduction time was 3.83 ± 0.15 min (Table 7). In group I and II the methylene blue reduction time was more than 60 minutes at zero hour in all animals. In group I the MBRT ranged from 45 minutes to 60 minutes at 24 h and in group II it was 40 minutes to 60 minutes.

In group I at 48 h MBRT was 29.16 ± 4.15 minutes whereas in group II it was 27.20 ± 2.25 min. In group I at 72 h the MBRT was 7.83 ± 0.76 min and in group II it was 6.40 ± 1.15 min (Table 8 and 9).

4.2.3.5. Total volatile fatty acids (TVFA)

The total volatile fatty acid concentration in the rumen liquor of control animals was 54.83 ± 2.24 mEq/l (Table 7).

At zero hour the TVFA concentration was 40.00 ± 14.26 mEq/l in group I and 38.63 ± 10.49 mEq/l in group II. In group I the TVFA concentration was 20.0 ± 2.77 mEq/l at 24 h and in group II it was 20.40 ± 3.44 mEq/l.

At 48 h, the TVFA concentration in group I was 27.33 ± 2.96 mEq/l and in group II it was 29.88 ± 4.78 mEq/l. In group I at 72 h the TVFA concentration was 30.17 ± 3.26 mEq/l and in group II it was 23.26 ± 2.04 mEq/l.

In groups I and II there was a non significant reduction in TVFA noticed at zero hour when compared to TVFA concentration in control animals. At 24 h, 48 h and 72 h there was a significant reduction ($p \leq 0.01$) of TVFA in groups I and II when compared to normal (Table 8 and 9, Fig. III).

4.2.4 Lactic acid in rumen liquor

The lactic acid content of rumen liquor in control animals was 21.87 ± 1.08 mg/dl (Table 7). In animals of group I and II,it was 235.70 ± 17.12 mg/dl and 275.97 ± 28.17 mg/dl respectively at zero hour. In groups I and II the lactic acid concentration reduced to 77.85 ± 11.12 mg/dl and 85.10 ± 5.20 mg/dl respectively, at 24 h.

At 48 h in group I and II, it was 46.97 ± 9.08 mg/dl and 45.64 ± 4.10 mg/dl respectively. In group I at 72 h,

the lactic acid concentration was 24.38 ± 3.79 mg/dl and in group II it was 23.86 ± 2.04 mg/dl. A significant increase was noticed in the lactic acid concentration in groups I and II at zero hour and 24 h ($p \leq 0.01$) and at 48 h ($p \leq 0.05$), when compared to control values.

At 72 h, no significant difference was noticed in the rumen liquor lactic acid concentration in group I and II when compared to the control values (Table 8 and 9, Fig. III).

4.3. Haematology

4.3.1. Haemoglobin (Hb)

The haemoglobin concentration in control animals was 12.17 ± 0.54 g/dl. In animals of group I and II it was 13.83 ± 0.79 g/dl and 14.83 ± 0.91 g/dl at zero hour. In group I at 24 h the haemoglobin concentration reduced to 11.83 ± 0.75 g/dl and in group II it was 12.20 ± 0.49 g/dl.

At 48 h the haemoglobin concentration was 10.00 ± 0.97 g/dl in group I and 11.40 ± 0.51 g/dl in group II. In group I at 72 h the haemoglobin concentration was 10.83 ± 0.79 g/dl and in group II it was 12.00 ± 0.84 g/dl. No significant difference noted in the haemoglobin level

between group I and II at zero hour, 24 h, 48 h and 72 h (Table 10, 11 and 12) when compared to control values.

4.3.2. Packed cell volume (PCV)

The packed cell volume obtained in control animals was 27.67 ± 0.96 percent (Table 10). In groups I and II at zero hour the PCV was found to be increased significantly ($p \leq 0.01$) to 43.00 ± 3.13 and 47.67 ± 2.89 per cent respectively when compared to the control values. At one hour also significant increase ($p \leq 0.01$) noticed in the PCV in group I to 40.00 ± 2.63 percent and in group II to 43.00 ± 2.29 percent.

No significant difference noticed at 6 h, 12 h, 24 h, 48 h and 72 h in the PCV of groups I and II (Table 4,5 and 6, Fig IV) when compared to control values.

4.3.3. Total erythrocyte count (TEC)

The total erythrocyte count in control animals was 14.0 ± 0.47 x $10^6/\mu l$. In group I and II a non-significant increase in total erythrocyte count was noticed at zero hour when compared to control values. From 24 h to 72 h no significant difference in total erythrocyte count was

noticed in group I and II when compared to control values (Table 10, 11 and 12).

4.3.4. Total Leukocyte Count (TLC)

The total leukocyte count noticed in control animals was 12.57 ± 0.90 x 10^3/ µl. In groups I and II it was slightly increased to 13.50 ± 0.33 x 10^3/ µl and 13.71 ± 0.54 x 10^3/ µl at zero hour. No significant difference noticed in total leukocytes in group I and II at zero hour, 24 h, 48 h and 72 h (Table 10, 11 and 12) when compared to control values.

4.3.5. Differential leukocyte count (DLC)

4.3.5.1. Neutrophils

In control animals the percentage of neutrophils was 35.83 ± 2.26 (Table 10). In group I it was 57.33 ± 3.67 and in group II it was 58.67 ±3.78 at zero hour. At 24 h, the percentage of neutrophils in group I was 54.50 ±3.12 and in group II it was 54.40 ± 4.34.

In group I and II, significant increase ($p \leq 0.01$) was noticed in the percentage of neutrophils at zero hour and 24 h when compared to control values. In group I there was significant increase ($p \leq 0.05$) in the percentage of

neutrophils at 24 h when compared to group II and control values. But no significant difference noticed in group II at 48 h and 72 h when compared to control values (Table 11 and 12, Fig. V).

4.3.5.2. Eosinophils

The percentage of eosinophils in control animals was 3.67 ± 0.67 (Table 10). In group I it was 2.33 ± 0.33 and in group II 1.50 ± 0.34. No significant difference noticed in the percentage of eosinophils between group I and II at zero hour, 24 h, 48 h and 72 h (Table 11, 12) when compared to control values.

4.3.5.3. Basophils

The percentage of basophils in control animals was 0.33 ± 0.19 (Table 10). No significant difference noticed in the percentage of basophils between groups I and II at zero hour, 24 h, 48 h and 72 h (Table 10, 11, 12) when compared to the control values.

4.3.5.4. Lymphocytes

The percentage of lymphocytes in control animals was 58.50 ± 1.70 (Table 10). In group I at zero hour and 24 h the lymphocyte percentage significantly got reduced

($p \leq 0.01$) to 38.33 ± 3.29 and 37.33 ± 3.14 respectively and then increased to 45.17 ± 2.94 at 48 h. In group II also the percentage of lymphocytes significantly reduced ($p \leq 0.01$) to 37.83 ± 3.72, 39.20 ± 4.59 and then increased to 48.20 ± 3.45 at zero hour, 24 h and 48 h respectively.

In group I, at 72 h the percentage of lymphocytes significantly reduced ($p \leq 0.05$) to 44.17 ± 2.09 whereas no significant difference noticed in group II when compared to control values (Table 11 and 12 Fig. V).

4.3.5.5. Monocytes

The percentage of monocytes in control animals was 1.67 ± 0.33 (Table 10). No significant difference noticed in the percentage of monocytes at zero hour, 24 h, 48 h and 72 h in group I and II (Table 11, 12) when compared to control values.

4.4. Serum biochemistry

4.4.1. Serum bicarbonate

Serum bicarbonate content in control animals was 26.83 ± 0.38 mEq/l (Table 4).

In group I there was significant reduction ($p \leq 0.01$) in the serum bicarbonate to 15.48 ± 1.23 mEq/l and in group II 14.12 ± 1.40 mEq/l at zero hour. At zero hour, the serum bicarbonate in group I ranged from 11.2.mEq/l to 19.4 mEq/l. In group II it was from 8.4 mEq/l to 17.8 mEq/l at zero hour. At 1 h also serum bicarbonate was significantly reduced ($p \leq 0.01$) in groups I and II.

At 12 h, significant reduction ($p \leq 0.01$) in serum bicarbonate was noticed only in group I when compared to control values. A significant increase ($p \leq 0.01$) in serum bicarbonate was noticed in group II when compared to group I at 12 h.

At 24 h, a significant decrease ($p \leq 0.01$) was noticed in group I when compared to group II and control values. In group II at 24 h, a significant increase ($p \leq 0.01$) was noticed when compared to group I and also control values.

At 48 h, a significant increase ($p \leq 0.01$) was noticed in the serum bicarbonate in group I and II when compared to control values. But at 72 h, no significant difference was noticed among groups I and II when compared to control values (Table 5 and 6, Fig. VI).

4.4.2. Serum lactic acid

The serum lactate content in control animals was 15.45 ± 0.38 mg/dl. Significant increase ($p \leq 0.01$) was noticed in the serum lactate level at zero hour, 24 h and 48 h in both groups I and II when compared to the control values.

In group I the serum lactate reduced from a peak value of 50.95 ± 5.43 mg percent at zero hour to 22.35 ± 2.69 mg percent at 72 h. In group II it got reduced from a peak value of 60.80 ± 6.87 mg percent at zero hour to 16.88 ± 1.05 mg percent at 72 h.

At 72 h also significant increase ($p \leq 0.05$) was noticed in the serum lactate level in group I when compared to group II and control values. In group II serum lactate became normal when compared to control values at 72 h (Table 13, 14, 15 and Fig. VII).

4.4.3. Total serum protein

The total serum protein in control animals was 6.68 ± 0.13 g/dl (Table 13). A significant increase ($p \leq 0.05$) in the serum protein was noticed at zero hour, whereas no significant difference was noticed at 24 h, 48 h and 72 h

in group I and II when compared to control values (Table 14 and 15).

4.4.4. Serum potassium

The serum potassium in control animals was 4.72 ± 0.06 mEq/l (Table 13). No significant difference noticed in the serum potassium level at zero hour, 24 h, 48 h and 72 h between groups I and II (Table 14 and 15) when compared to control values.

4.5. Therapeutic management

In both groups I and II 200ml of fresh rumen liquor was given orally using stomach tube at 24 h. In one animal of group I 100ml of rumen liquor was again given at 48 h as there was no live protozoa in the rumen liquor at 48 h. All the other animals revealed sufficient live protozoa in their rumen liquor in subsequent days after single administration of rumen liquor orally.

All the animals urinated within one to four hours of fluid therapy; diarrhoeic faeces of the affected animals became normal in 48 to 72 h except in one animal in which diarrhoea persisted for 96 hours.

At 24 h, one animal in group II started taking little quantity of grass but all the animals in group I was anorectic. At 48 h all the animals except one animal in group I and all animals in group II started taking little quantity of grass and water. At 72 h all the animals in group I and II showed increased appetite except one in group I which regained the appetite only 96 h after treatment.

The distension of abdomen was reduced in all animals of group I and II after evacuation of the rumen liquor using a vacuum rumen liquor aspirator.

All the animals showed clinical recovery except one animal of group II which died at 8th hour of treatment.

The clinical assessment at 48 h revealed that although all animals were dull, they appeared to be more alert and active than at 12 h and 24 h. Due to successful treatment, all the animals in group I and II recovered except one animal in group II which died at 8th hour of treatment.

Table 1

CLINICAL DATA OF CONTROLS (MEAN ± SE)

Respiration (per min)	Pulse (per min)	Heart rate (per min)	Temperature (°C)	Mucous membrane	Rumen motility (per min)
21.33 ± 0.67	88.33 ± 1.96	88.67 ± 1.98	39.35 ± 0.22	Pale roseate	1

Table 2

CLINICAL DATA OF GROUP I (MEAN ± SE)

Time interval	Respiration (per min)	Pulse (per min)	Heart rate (per min)	Temperature (°C)	Mucous membrane	Rumen motility (per min)
zero hour	(**c) 41.33 ± 2.23	Imperceptible	(**c) 114.0 ± 4.32	38.67 ± 0.28	Pale roseate to congested	Nil
1 h	(**c,**II) 42.50 ± 2.22	,,	(**c) 113.33 ± 3.04	38.72 ± 0.22	,,	,,
6 h	(**c,**II) 38.67 ± 1.69	,,	(**c) 107.33 ± 2.56	38.78 ± 0.19	,,	,,
12 h	(**c,**II) 32.0 ± 1.71	,,	(**c) 105.00 ± 2.67	38.92 ± 0.24	,,	,,
24 h	(**c,**II) 26.33 ± 0.62	(**c) 103.67 ± 2.34	(**c) 103.67 ± 2.34	39.07 ± 0.18	pale roseate	0-1
48 h	(**c,**II) 24.17 ± 1.28	(**c) 97.83 ± 1.22	(*c) 97.83 ± 1.22	39.26 ± 0.13	,,	,,
72 h	21.83 ± 0.48	93.17 ± 1.64	93.17 ± 1.64	39.33 ± 0.11	,,	,,

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

TABLE 4
PCV, RUMEN LIQUOR pH AND SERUM BICARBONATE OF CONTROLS
(MEAN ± SE)

PCV (%)	RUMEN LIQUOR pH	SERUM BICARBONATE (mEq/l)
27.67 ± 0.96	7.08 ± 0.08	26.83 ± 0.38

TABLE 5
PCV, RUMEN LIQUOR pH AND SERUM BICARBONATE OF GROUP I
(MEAN ± SE)

Time interval	PCV (%)	RUMEN LIQUOR pH	SERUM BICARBONATE (mEq/l)
Zero hour	(**c) 43.00 ± 3.13	(**c) 4.50 ± 0.18	(**c) 15.48 ± 1.23
1 h	(**c) 40.00 ± 2.63	(*c) 6.75 ± 0.11	(**c) 18.53 ± 1.05
6 h	31.33 ± 1.76	(**c) 6.50 ± 0.13	(**c) 20.30 ± 2.19
12 h	29.67 ± 1.40	(*c) 6.67 ± 0.10	(**c, **II) 22.35 ± 0.10
24 h	28.00 ± 1.27	6.75 ± 0.11	(**c, **II) 24.18 ± 0.64
48 h	27.33 ± 0.85	6.83 ± 0.11	(**c) 30.03 ± 0.69
72 h	24.67 ± 0.85	6.83 ± 0.10	28.53 ± 1.22

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

TABLE 6
PCV, RUMEN LIQUOR pH AND SERUM BICARBONATE OF GROUP II (MEAN ± SE)

Time interval	PCV (%)	Rumen liquor pH	Serum bicarbonate (mEq/l)
Zero hour	(**c) 47.67 ± 2.89	(**c) 4.08 ± 0.30	(**c) 14.12 ± 1.40
1 h	(**c) 43.00 ± 2.29	(*c) 6.58 ± 0.16	(**c) 20.50 ± 2.14
6 h	34.33 ± 3.32	(**c) 6.50 ± 0.13	(*c) 20.67 ± 2.32
12 h	30.40 ± 1.17	(*c) 6.70 ± 0.12	(**I) 26.18 ± 0.87
24 h	26.40 ± 0.75	6.80 ± 0.05	(**c, **I) 30.02 ± 0.92
48 h	26.40 ± 1.47	6.90 ± 0.10	(**c) 29.44 ± 0.38
72 h	25.20 ± 1.02	6.90 ± 0.10	27.60 ± 0.64

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

TABLE 7

RUMEN LIQUOR CHARACTERS OF CONTROLS (MEAN ± SE)

Colour	Olive green	SAT (min)	14.63 ± 0.35
Consistency	Thick	MBRT (min)	3.83 ± 0.15
Odour	Aromatic	TVFA (mEq/l)	54.83 ± 2.24
Protozoal Activity	+++to ++++	Lactate (mg/dl)	21.87 ± 1.08

TABLE 8

RUMEN LIQUOR CHARACTERS OF GROUP I (MEAN ± SE)

Parameters	zero hour	24 h	48 h	72 h
Colour	Milky grey	Milky grey	Milky grey	Milky grey to green
Consistency	Watery	Watery to semiliquid	Semiliquid to thick	Thick
Odour	Intense sour	Faintly sour	Faintly sour to aromatic	Aromatic
Protozoal activity	Nil	+ to Nil	++ to Nil	+++ to ++
SAT (min)	Rapid sedimentation but no flotation		18 min to nil	16 min to nil
MBRT (min)	>60 min	45 to 60 min	29.16 ± 4.15	7.83 ± 0.76
TVFA (mEq/l)	40.00 ± 14.26	(**c) 20.00 ± 2.77	(**c) 27.33 ± 2.96	(**c) 30.17 ± 3.26
Lactate (mg/dl)	(**c) 235.70 ± 17.12	(**c) 77.85 ± 11.12	(*c) 46.97 ± 9.08	24.38 ± 3.79

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

TABLE 9
RUMEN LIQUOR CHARACTERS OF GROUP II (MEAN ± SE)

Parameters	Zero hour	24 h	48 h	72 h
Colour	Milky grey	Milky grey	Milky grey to green	Milky grey to green
Consistency	Watery	Watery to semiliquid	Semiliquid to thick	Thick
Odour	Intense sour	Faintly sour	Faintly sour to aromatic	Aromatic
Protozoal activity	Nil	+ to Nil	++ to +++	+++ to ++
SAT (min)	Rapid sedimentation but no flotation		13 min to nil	10 min to nil
MBRT (min)	> 60 min	40 min to 60	27.20 ± 2.25	6.40 ± 1.15
TVFA (mEq/l)	38.63 ± 10.49	(**c) 20.40 ± 3.44	(**c) 29.88 ± 4.78	(**c) 39.00 ± 3.86
Lactate (mg/dl)	(**c) 275.97 ± 28.17	(**c) 85.10 ± 5.20	(*c) 45.64 ± 4.10	23.86 ± 2.04

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

TABLE 10
HAEMATOLOGY OF CONTROLS (MEAN ± SE)

Haemoglobin (g/dl)	Total erythrocytes (x 10^6/ µl)	Total leukocytes (x 10^3/ µl)	Differential leukocyte count				
			Neutro-phils (%)	Eosino-phils (%)	Baso-phils (%)	Lympho-cytes (%)	Mono-cytes (%)
12.17 ± 0.54	14.0 ± 0.47	12.57 ± 0.90	35.83 ± 2.26	3.67 ± 0.67	0.33 ± 0.19	58.50 ± 1.70	1.67 ± 0.33

TABLE 11
HAEMATOLOGY OF GROUP I

Parameters	zero hour	24 h	48 h	72 h
Haemoglobin (g/dl)	13.83 ± 0.79	11.83 ± 0.75	10.00 ± 0.97	10.83 ± 0.79
Total erythrocytes (x 10^6/ µl)	16.11 ± 0.35	14.15 ± 0.60	13.14 ± 0.66	12.60 ± 0.75
Total leukocytes (x 10^3/ µl)	13.50 ± 0.33	13.17 ± 0.29	13.10 ± 0.44	12.27 ± 0.66
Neutrophils (%)	(**c) 57.33 ± 3.67	(*c,*II) 54.50 ± 3.12	50.17 ± 2.44	44.67 ± 2.31
Eosinophils (%)	2.33 ± 0.33	4.17 ± 0.54	3.67 ± 0.71	3.17 ± 0.48
Basophils (%)	0.16 ± 0.15	0.50 ± 0.20	0.16 ± 0.15	0.33 ± 0.19
Lymphocytes (%)	(**c) 38.33 ± 3.29	(**c) 37.33 ± 3.14	(*c) 45.17 ± 2.94	(**c,*II) 44.17 ± 2.09
Monocytes (%)	1.69 ± 0.21	3.67 ± 0.42	3.33 ± 0.42	2.67 ± 0.76

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

TABLE 12
HAEMATOLOGY OF GROUP II (MEAN ± SE)

Parameters	zero hour	24 h	48 h	72 h
Haemoglobin (g/dl)	14.83 ± 0.91	12.20 ± 0.49	11.40 ± 0.51	12.00 ± 0.84
Total erythrocytes (x 10^6/ µl)	16.45 ± 0.88	14.68 ± 0.97	13.98 ± 0.99	12.54 ± 0.80
Total leukocytes (x 10^3/ µl)	13.71 ± 0.54	13.72 ± 0.72	13.24 ± 0.54	12.88 ± 0.44
Neutrophils (%)	(**c) 58.67 ± 3.78	(*I) 54.40 ± 4.34	44.80 ± 4.13	38.20 ± 1.65
Eosinophils (%)	1.50 ± 0.34	3.00 ± 0.32	3.40 ± 0.51	2.20 ± 0.49
Basophils (%)	0.16 ± 0.15	0.60 ± 0.21	0.40 ± 0.21	0.40 ± 0.21
Lymphocytes (%)	(**c) 37.83 ± 3.72	(**c) 39.20 ± 4.59	(*c) 48.20 ± 3.45	(*I) 58.00 ± 1.14
Monocytes (%)	1.83 ± 0.31	2.80 ± 0.49	3.20 ± 0.49	1.40 ± 0.25

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

TABLE 13
SERUM BIOCHEMISTRY OF CONTROLS (MEAN ± SE)

Serum lactate (mg/dl)	15.45 ± 0.38
Serum protein (g/ dl)	6.68 ± 0.13
Serum potassium (mEq/l)	4.72 ± 0.06

TABLE 14
SERUM BIOCHEMISTRY OF GROUP I (MEAN ± SE)

Parameters	zero hour	24 h	48 h	72 h
Serum lactate (mg/dl)	(**c) 50.95 ± 5.43	(**c) 34.98 ± 4.21	(**c) 28.75 ± 3.41	(*c,*II) 22.35 ± 2.69
Serum protein (g/ dl)	(*c) 8.33 ± 0.38	6.98 ± 0.29	6.75 ± 0.23	6.18 ± 0.22
Serum potassium (mEq/l)	4.62 ± 0.48	4.55 ± 0.42	4.95 ± 0.42	4.52 ± 0.34

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

TABLE 15
SERUM BIOCHEMISTRY OF GROUP II (MEAN ± SE)

Parameters	zero hour	24 h	48 h	72 h
Serum lactate (mg/dl)	(**c) 60.80 ± 6.87	(**c) 33.68 ± 4.85	(**c) 24.20 ± 3.20	(*I) 16.88 ± 1.05
Serum protein (g/ dl)	(*c) 8.07 ± 0.46	6.80 ± 0.46	6.66 ± 0.25	6.46 ± 0.29
Serum potassium (mEq/l)	4.73 ± 0.59	4.58 ± 0.49	4.80 ± 0.41	4.50 ± 0.44

**c significant at $p \leq 0.01$ level when compared to controls.
*c significant at $p \leq 0.05$ level when compared to controls.
**I significant at $p \leq 0.01$ level when compared to group I.
*I significant at $p \leq 0.05$ level when compared to group I.
**II significant at $p \leq 0.01$ level when compared to group II.
*II significant at $p \leq 0.05$ level when compared to group II.

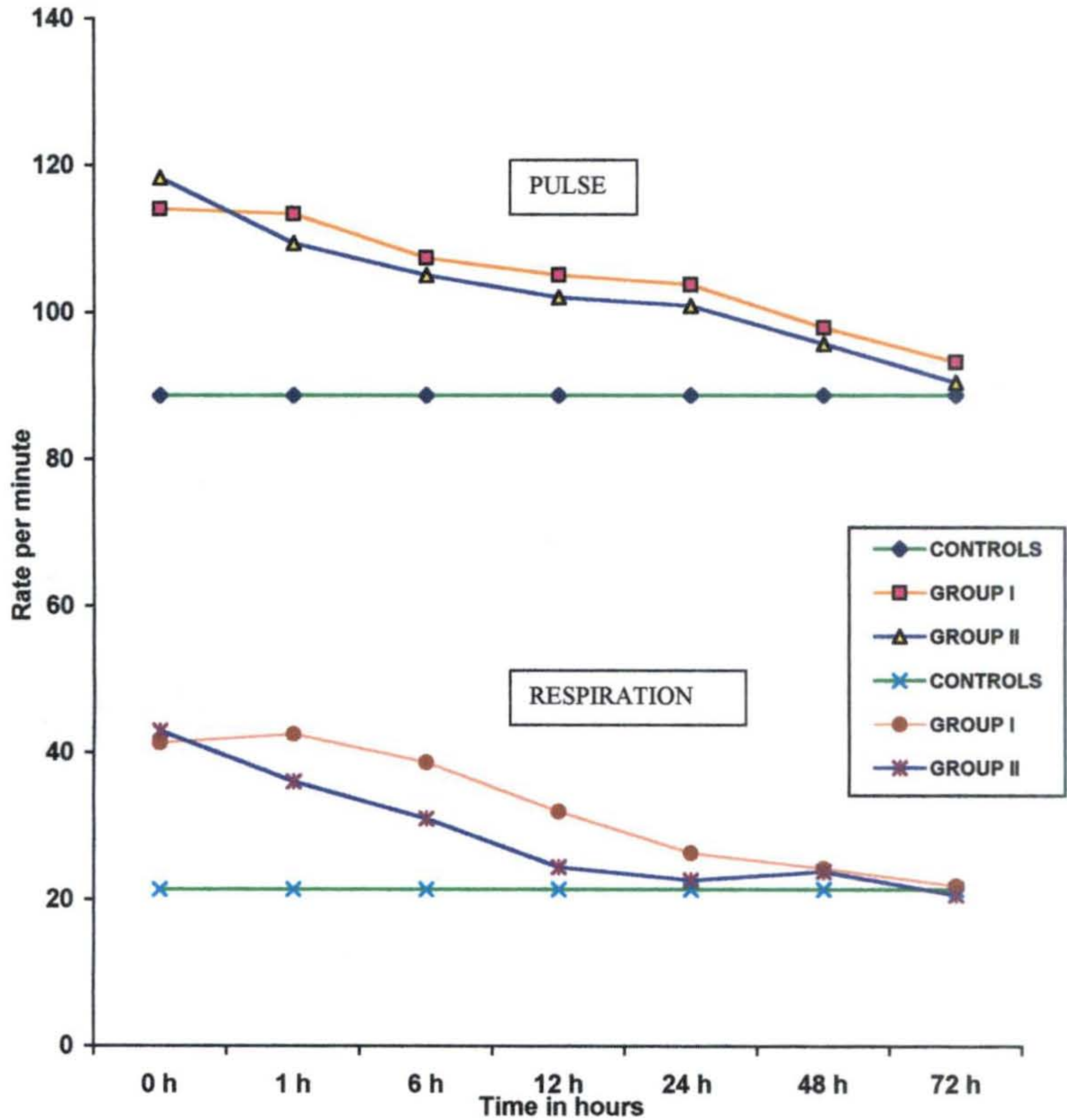


FIG. I. COMPARISON OF CLINICAL DATA OF CONTROLS, GROUP I AND GROUP II
140
120
100
80
60
40
20
0
Rate per minute
PULSE
RESPIRATION
CONTROLS
GROUP I
GROUP II
CONTROLS
GROUP I
GROUP II
0 h
1 h
6 h
12 h
24 h
48 h
72 h
Time in hours

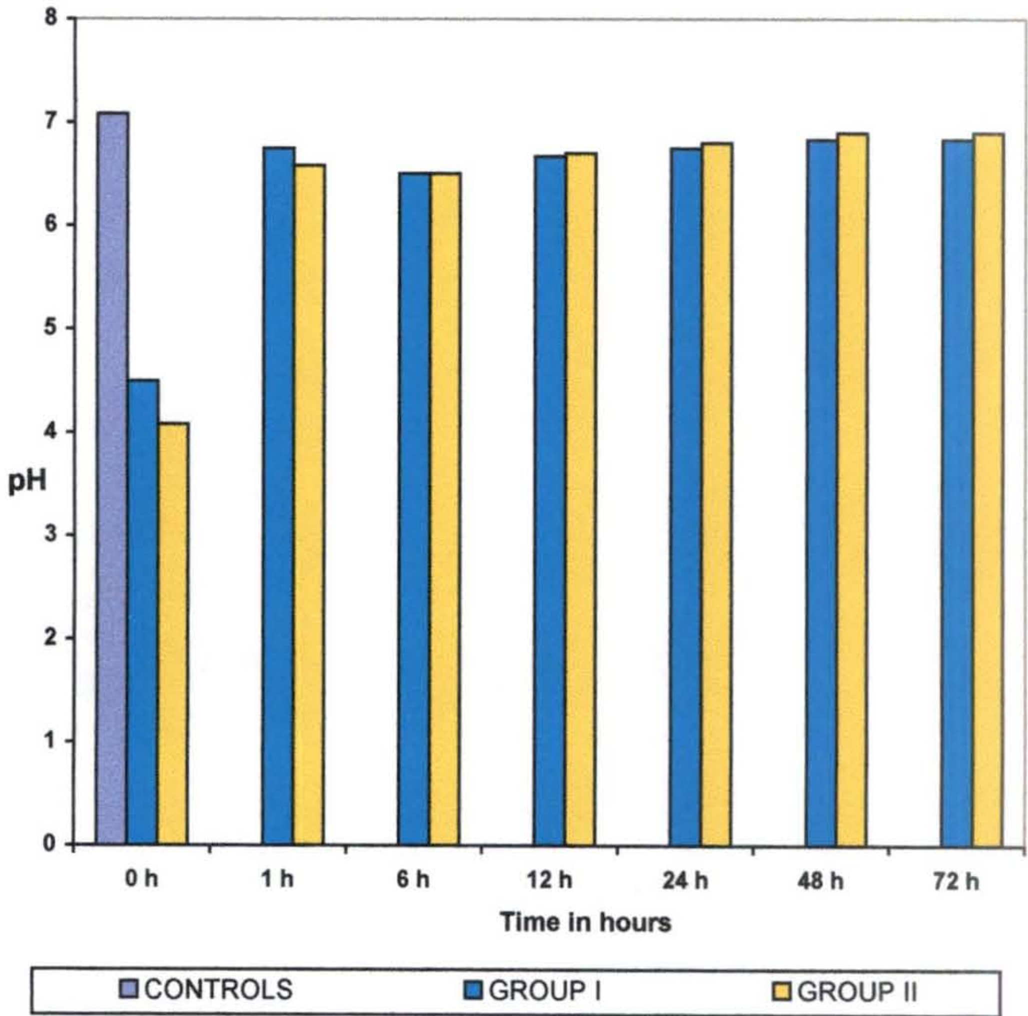
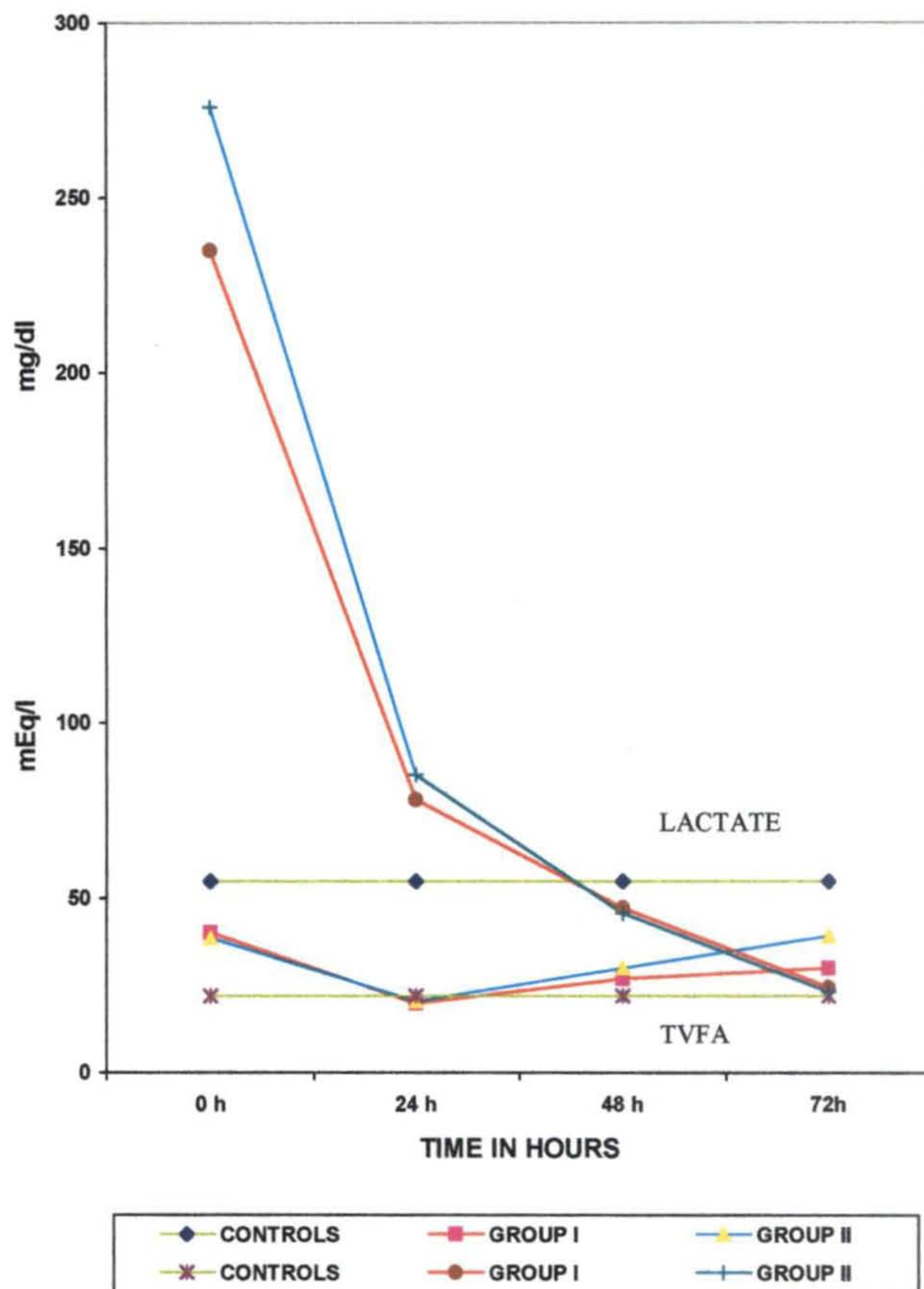


FIG. II. COMPARISON OF RUMEN pH OF CONTROLS, GROUP I AND GROUP II
pH
0
1
2
3
4
5
6
7
8
0 h
1 h
6 h
12 h
24 h
48 h
72 h
Time in hours
CONTROLS
GROUP I
GROUP II

FIG. III. COMPARISON OF RUMEN LIQUOR TVFA AND LACTATE AMONG CONTROLS,GROUP I AND GROUP II



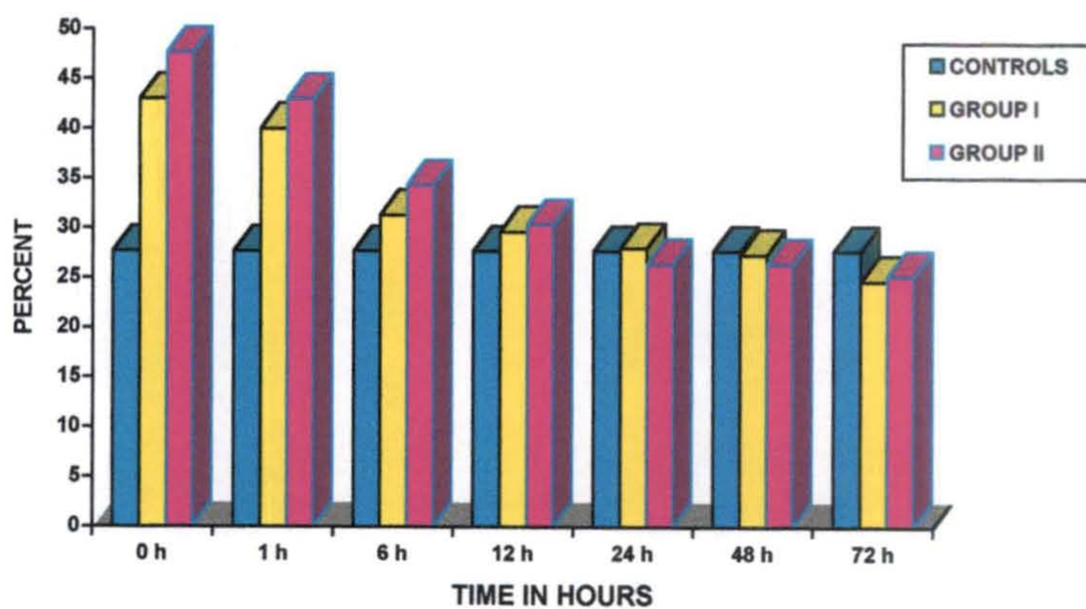
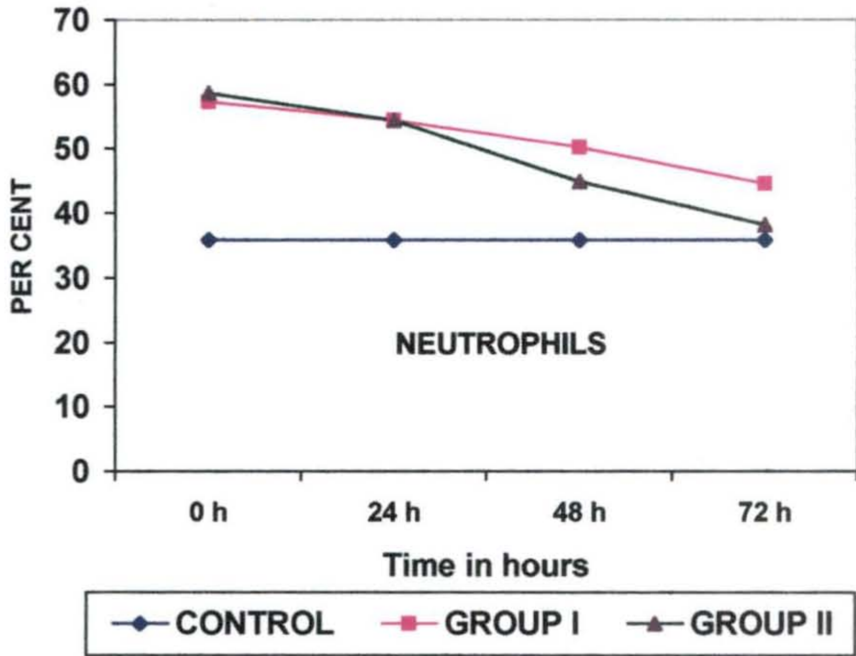


FIG. IV. COMPARISON OF PCV OF CONTROLS, GROUP I AND GROUP II
PERCENT
50
45
40
35
30
25
20
15
10
5
0
0 h
1 h
6 h
12 h
24 h
48 h
72 h
TIME IN HOURS
CONTROLS
GROUP I
GROUP II

FIG. V. DIFFERENTIAL COUNT: COMPARISON AMONG CONTROLS,GROUP I AND GROUP II



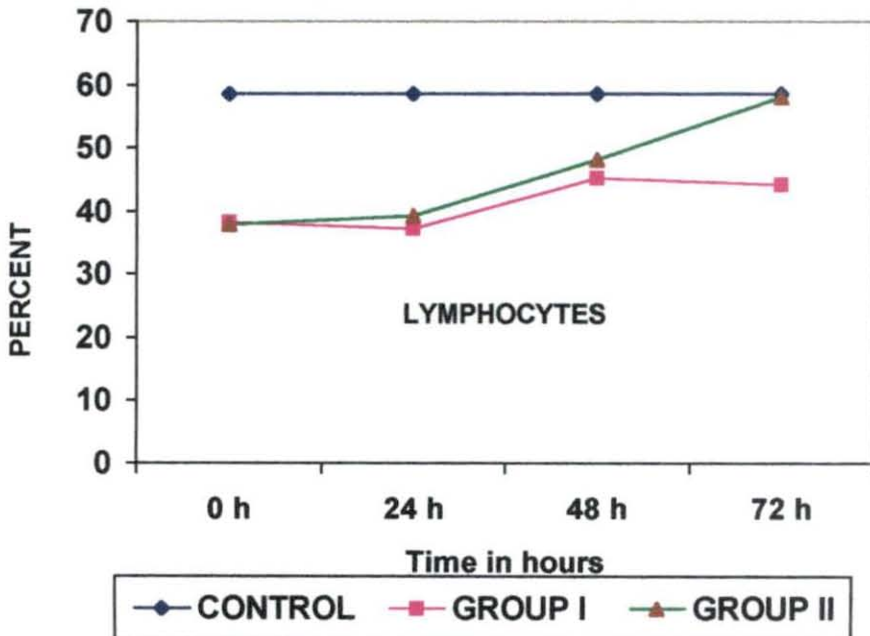


FIG. VI. COMPARISON OF SERUM BICARBONATE OF CONTROLS, GROUP I AND GROUP II

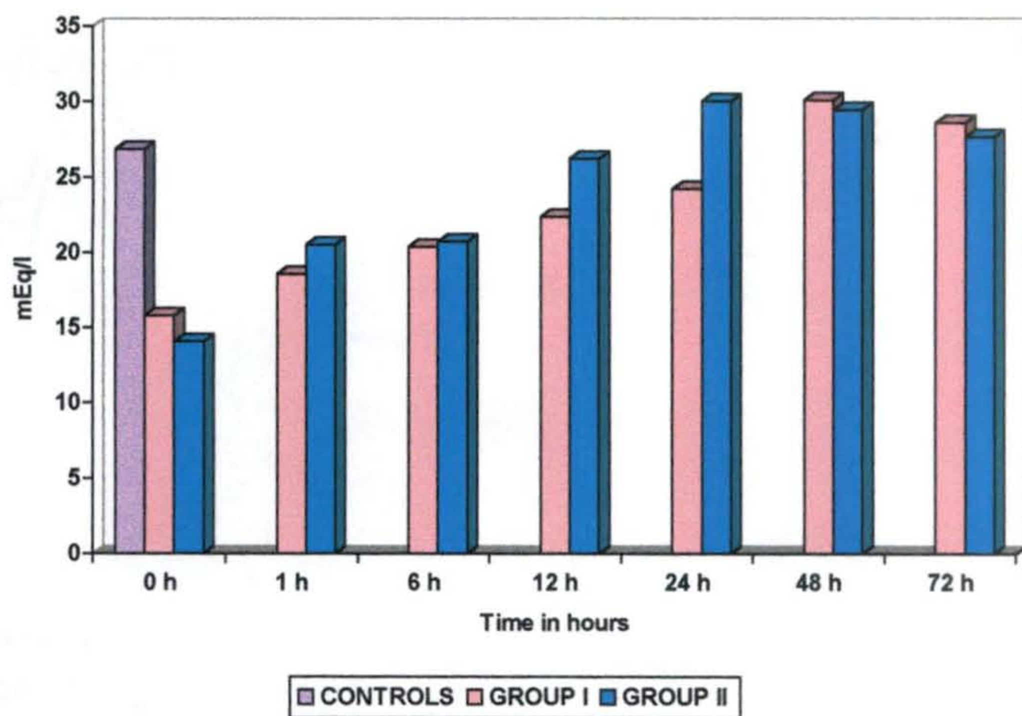
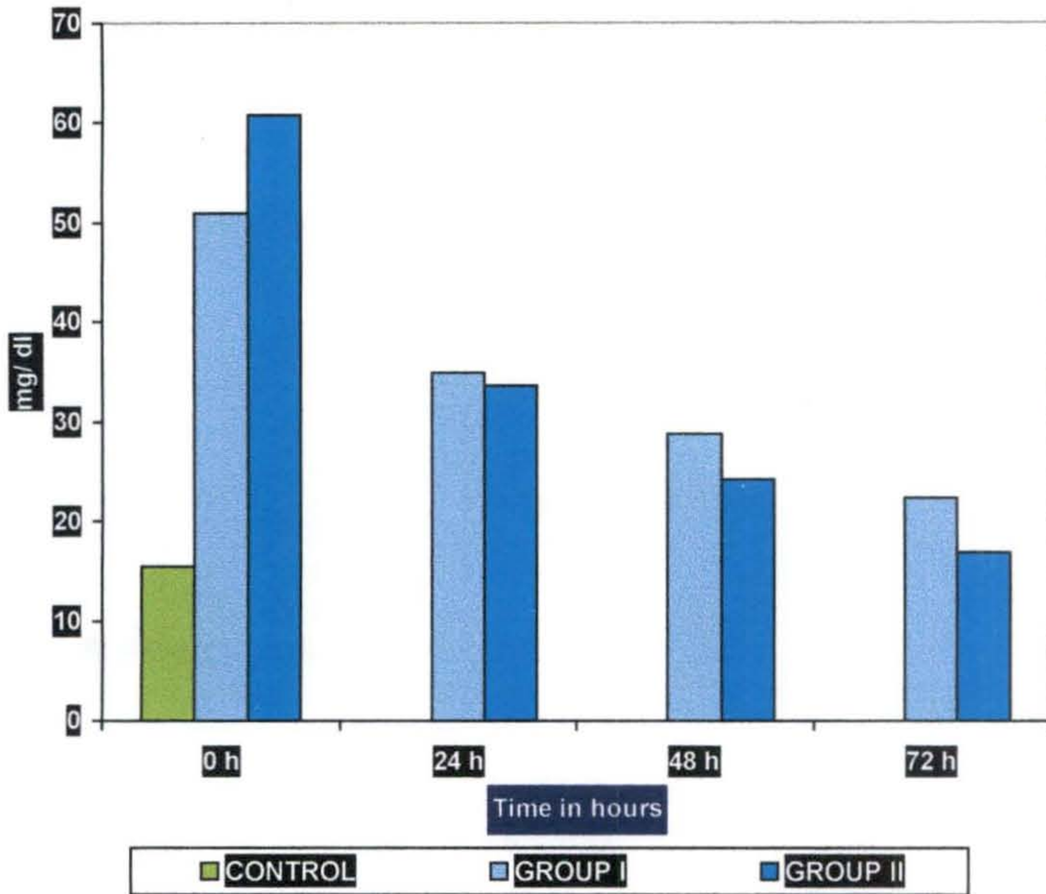


FIG. VII. COMPARISON OF SERUM LACTATE AMONG CONTROLS,GROUP I AND GROUP II



DISCUSSION

5. DISCUSSION

5.1. Clinical signs

The clinical signs observed in the present study like anorexia, loss of appetite, increased pulse and respiratory rate, mild tympany, reduced ruminal contractions, fluidy rumen on percussion, diarrhoea, oliguria and dehydration were similar to the findings of Gnanaprakasam (1970), Pillai (1988), Lal *et al.* (1989) and Shihabudheen (1998).

Anorexia and reduced appetite noticed in the present study might be due to accumulated lactic acid in the rumen (Dunlop, 1972).

The clinical signs exhibited like retracted eyeballs, lateral recumbency and cold extremities in one animal of group I and two animals of group II were suggestive of severe acidosis as referred by Gnanaprakasam (1970).

The fluidity of the rumen contents together with impairment of colonic absorptive activity might be

responsible for the diarrhoeic syndrome (Michell, 1990).

Anuria found in the present study might be due to reduced renal blood flow and glomerular filtration resulting from dehydration, heamoconcentration, hypertension and renal failure (Huber, 1969; Dunlop, 1972).

The dehydration occurred was due to the increased osmolality associated with rise in production of lactic acid which inturn was responsible for withdrawal of water from the circulation (Dunlop, 1972; Radostits et *al.*, 1994) and severity increased if associated with diarrhoea (Michell, 1990).

The clinical assessment at 24 h revealed that the dyspnoea and respiratory rate were considerably reduced in animals of group II when compared to animals of group I. This may be due to reduced PCO_2 and increased pH in the blood (Huber, 1976).

Another finding at 24 h of the study was that, in group II all animals appeared more alert and active than animals of group I.

All the animals in group I and group II, responded well to the treatment except one animal in group II which was in an advanced stage of the disease with severe symptoms and grave prognosis.

All recovered animals of group II regained appetite earlier than those animals in group I indicating that the clinical recovery was faster with carbicarb treated animals than with sodium bicarbonate alone.

5.1.1. Respiration

The normal respiratory rate observed in control animals of group I was 21.33 ± 0.67 per minute which is slightly higher than the findings of Pillai (1988). But it concurs with the findings of Shihabudheen (1998).

The increased respiratory rates in group I and group II, 41.33 ± 2.23 per min and 43.00 ± 1.52 per min respectively, at zero hour were comparable with the observations made by Cao *et al.* (1987) and Braun *et al.* (1992) in goats.

The increased carbon dioxide tension of blood and decreased blood pH stimulates the respiratory centre bringing about an increased rate of carbondioxide elimination through increased respiration (Huber, 1976). Thus the increased respiratory rate would be to compensate the rise in hydrogen ion concentration in blood (Rumsey *et al.*, 1970) by removing excess carbon dioxide (Braun *et al.*, 1992; Michell, 1990).

From one hour to 48h of treatment, the animals of group II showed significant reduction in the respiratory rate when compared to group I animals which indicates reduced carbondioxide tension in blood consequent to the infusion of carbicarb as systemic alkaliniser and resulted in early clinical recovery in animals of group II.

5.1.2. Pulse

At the beginning of the study, the pulse was imperceptible in all animals except one each in both groups I and II where it was very weak. The character of the pulse observed at this stage concurred with the observations made by Gnanaprakasam (1970).

At one hour of study the pulse was palpable but rapid and weak in three of the animals of group II and in one animal of group I. The increased pulse rate noticed in the present study agrees with the findings of Lal *et al.* (1989). Increased pulse rate may be due to the accumulated lactic acid in the system causing activation of the sympathetic nervous system (Radostits *et al.,* 1994).

A strong pulse obtained early in animals of group II than in animals of group I suggests improved cardiac haemodynamics (Bersin and Arieff, 1988).

5.1.3. Temperature

The mean rectal temperature of 39.35 ± 0.22°C recorded in control animals was comparable with the observations of Pillai (1988) and Shihabudheen (1998).

A non significant decrease in mean rectal temperature from zero hour to 48 h in animals of group I and II agrees with the findings of Lal *et al.* (1989) and it could be due to dehydration, fall in plasma volume, fall in blood pressure and peripheral circulatory failure.

5.1.4. Heart rate

The normal heart rate of 88.67 ± 1.98 per min recorded in animals of group I agrees with the findings of Shihabudheen (1998).

At zero hour a significant increase ($p \leq 0.01$) in heart rate was noticed in animals of group I and II. The increased heart rate in the present study concurs with earlier reports of Pillai (1988), Braun *et al.* (1992) and Shihabudheen (1998). This could be attributed to the activation of sympathetic nervous system in response to acidosis causing increased heart rate (Radostits *et al.*, 1994).

5.1.5. Mucous membrane

In groups I and II the congested mucous membrane noticed at zero hour agrees with the findings of Gnanaprakasam (1970) and Vestweber *et al.* (1974).

5.1.6. Rumen motility

The amplitude of rumen contraction was stronger and the motility recorded was one per minute in control

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animals. These were similar to the reports of Pillai (1988).

Absence of rumen motility at zero hour and reduced rumen motility at 24 h and 48 h in animals of group I and II were in agreement with the findings of Prasad (1979) and Lal *et al.* (1989).

The ruminal stasis observed in the present study would probably be due to increased molar concentration of butyrate or because of lactic acid entering the duodenum exerting a reflex inhibitory action on the rumen (Radostits *et al.*, 1994); it might be a protective response, that would minimise absorption of toxic materials (Dunlop, 1972).

5. 2.Changes in the rumen liquor

5.2.1 pH

The rumen liquor pH noticed in control animals was comparable to the earlier reports (Tanwar *et al.* 1983, Pillai 1988, Lal *et al.* 1989 and Basak *et al.* 1993a).

At zero hour, the pH of rumen liquor in group I significantly got reduced to 4.50 ± 0.18 and in group

II 4.08 ± 0.30. These findings were in accordance with the findings of Ahrens (1967), Basak *et al.* (1993a) and Shihabudheen (1998).

The reduction in pH of rumen liquor could be due to excess and rapid fermentation of carbohydrates (Prasad *et al.*,1972) by amylolytic bacteria (Lal *et al.*, 1989) and production of volatile fatty acids and large quantity of lactic acid (Michell, 1990), the accumulation of which exceeds buffering capacity of rumen (Radostits *et al.*, 1994).

The return of rumen liquor pH to normal by 12 h may be due to partial removal of ruminal contents and oral administration of magnesium hydroxide.

5.2.2 Physical characters

5.2.2.1 Colour

The milky grey colour of rumen liquor in group I and II at zero hour agrees with the findings of Pillai (1988); Braun *et al.* (1992) in goats and Radostits *et al.* (1994) in cattle which could be due to the presence of partially fermented starch combined with deprivation of normal feeds and fodder (Shihabudheen, 1998).

5.2.2.2 Consistency

The consistency of the rumen liquor was thick in all the control animals, whereas it was watery in all the animals of group I and II at zero hour.

The watery consistency of rumen liquor at zero hour in the present study may be due to the increased osmolality associated with excess production of lactic acid which is responsible for withdrawal of water from systemic circulation to the rumen (Braun *et al.* 1992; Radostits *et al.*, 1994).

The change in consistency of rumen liquor from watery to semifluid at 24 h and to thick consistency at 48 h in three animals of group I and in four animals of group II indicates reduced osmolality and reduced production of lactic acid and suggests clinical improvement.

5.2.2.3 Odour

Intense sour odour of rumen liquor noticed at zero hour in animals of group I and II agrees with the earlier findings of Lal *et al.* (1989) and Shihabudheen

(1998). Braun *et al.* (1992) also reported a penetrating acid odour for the rumen liquor of acidotic goats.

The intense sour odour of rumen liquor is suggestive of reduced rate of production of volatile fatty acids, variations in protein degradation in the rumen (Thomas, 1983) and also increased lactic acid content in rumen liquor (Basak *et al.* 1993).

5.2.3 Microbial activity

5.2.3.1 Rumen protozoa

The normal protozoal motility in control animals varied from +++ to ++++. The number of protozoa in the rumen liquor depends mainly on rations, feeding time and site from which the rumen liquor samples were collected (Rosenberger, 1979).

The complete absence of rumen protozoa at zero hour in the present study is supported by Gnanaprakasam (1970), Pillai (1988) and Basak *et al.* (1993a). Variations in rumen conditions like abnormal changes in pH and high lactic acid content is detrimental for the survival of microorganisms, which cannot thrive at a pH below 5 (Dunlop, 1972; Shihabudheen, 1998).

The gradual change of protozoal motility from sluggish (+) to moderate (++) to normal (+++) motility over a period of 24 h to 72 h in both groups I and II indicated a progressive and gradual return of disturbed internal environment of rumen to normal.

5.2.3.2 Sedimentation activity time (SAT)

The sedimentation activity time of rumen liquor noticed in control animals was 14.63 ± 0.35 min. This was comparable to the values reported by Pillai (1988) and Shihabudheen (1998). But Basak *et al.* (1993a) reported a higher value of 25.94 ± 1.70 min in rumen liquor of healthy goats.

Rapid sedimentation without floatation was observed in animals of group I and II at zero hour and 24h. These findings were similar to the findings of Rosenberger (1979) and Shihabudheen (1998). The rapid sedimentation and absence of floatation could be due to the interference in the normal fermentation and gas production caused by abnormal feeding and indigestion which causes settling of particles without floatation (Nichols and Penn, 1958).

5.2.3.3 Methylene blue reduction time (MBRT)

The methylene blue reduction time (MBRT) noticed in control animals was in agreement with the findings of Shihabudheen (1998).

The significant increase noticed in the MBRT in group I and II at zero hour, were comparable with earlier reports of Rosenberger (1979), Dirksen (1983) and Braun *et al.* (1992). The destruction of normal microflora (Basak *et al.*, 1993a) or decreased activity and oxidation reduction potential of microflora in lactic acidosis lead to an increase in MBRT (Dirksen, 1983).

5.2.3.4 Total volatile fatty acids (TVFA)

Total volatile fatty acid level in the rumen liquor of control animals was 54.83 ± 2.24 mEq /l. This was comparable with the values reported by Lal *et al.* (1992) and Basak *et al.* (1993a).

The mean TVFA concentration in the rumen liquor in animals of group I and II were found to be decreased at zero hour which was further reduced at 24 h. From 48 h onwards, TVFA concentration was found to be increased

and it continued till 72 h of study. Sinha *et al.* (1985), Lal *et al.* (1989) and Shihabudheen (1998) reported that in the initial stages of lactic acidosis the TVFA concentration increased till 24 h and then declined. From the above findings in the present study it could be presumed that the animals in group I and II were presented to the hospital, only after 24 h of ingestion of highly fermentable carbohydrate diet.

A decreased mean TVFA concentration in the rumen liquor in animals of group I and II at 24 h could be due to it's increased absorption at low ruminal pH (Danielli *et al.* 1945) or changes in the microbial population of rumen brought about by low pH in lactic acidosis (Prasad *et al.*, 1975) resulting in insufficient microbial population.

Subsequent increase in TVFA concentration noticed in the rumen liquor from 48 h onwards in both groups may be due to multiplication of bacteria resistant to low pH as reported by Basak *et al.* (1993a) and also due to the transplantation of fresh rumen liquor.

These findings suggest a positive role of the present therapy in normalising TVFA due to improvement in the rumen microbial activity.

5.2.4. Lactic acid in rumen liquor

In control animals, the normal lactic acid concentration in rumen liquor was 21.87 ± 1.08 mg/ dl. This was comparable with the values reported by Pillai (1988).

The lactic acid content of rumen liquor increased significantly to 235.70 ± 17.12 mg/ dl and 275.97 ± 28.17 mg./ dl respectively in group I and II at zero hour of study. The increase in rumen liquor lactate in the present study is consistent with the findings of Lal *et al.* (1989), Braun *et al.* (1992) and Pillai (1988).

Accumulation of lactic acid in rumen may be due to ingestion of highly fermentable carbohydrate feeds (Dunlop, 1972) like grains, cooked rice etc. (Aleyas and Vijayan, 1981).

The return of rumen liquor lactic acid to almost normal level by 72 h appears to have been accomplished by partial removal of ruminal contents, administration of tetracycline and fresh rumen liquor. These results were comparable with the findings of Sinha *et al.* (1985).

5.3. Haematology

5.3.1. Haemoglobin (Hb)

The normal haemoglobin content of blood in control animals was comparable with the values reported by Radostits *et al.* (1994) and Shihabudheen (1998).

A non significant increase in haemoglobin content of blood was noticed in animals of group I and II at zero hour of study. This finding agrees with the reports of Tanwar *et al.* (1983) and Angelov *et al.* (1995).

The rise in haemoglobin content of blood could be due to haemoconcentration caused by dehydration, which was evident from the clinical signs and elevated haematocrit. The release of blood cells from spleen due to stress might have also contributed to the elevated

haemoglobin level (Turner and Hodgetts, 1959; Dash *et al.*, 1972).

5.3.2. Packed cell volume (PCV)

The packed cell volume recorded in control animals were 27.67 ± 0.96 per cent which was comparable with the values of 24.0 to 48.0 per cent reported by Jain (1986) and 22.28 per cent by Radostits *et al.* (1994) in healthy goats.

A significant increase in packed cell volume was noticed at zero hour and one hour in groups I and II. These findings were comparable to the observations made earlier in cattle by Dunlop (1972), Dirksen (1983) and in goats by Lal *et al.* (1990) and Das and Misra (1991).

The increase in haematocrit could be due to the haemoconcentration and systemic dehydration caused by drawing of fluid from the circulation to rumen, when the osmolaltiy of the rumen is increased in acidosis (Michell, 1990). This could also be due to the release of red blood cells from the spleen due to the stress in lactic acidosis(Sen *et al.*, 1993).

The packed cell volume returned to normal after treatment, and from six hour onwards no significant difference was noticed in PCV in groups I and II when compared to control values.

5.3.3. Total erythrocyte count (TEC)

The total erythrocyte count in control animals was 14.0 ± 0.47 x 10^6/ µl. This was comparable to the values reported by Radostits *et al.* (1994).

The non-significant increase in total erythrocyte count observed in animals of groups I and II at zero hour were similar to the observations made earlier by Basak *et al.* (1993b). The rise in total erythrocyte count may be due to the release of red blood cells from spleen due to stress (Dash *et al.*,1972) or due to haemoconcentration (Dunlop, 1972).

5.3.4. Total leukocyte count (TLC)

A total leukocyte count of 12.57 ± 0.90 x 10^3/ µl obtained in control animals were in agreement with the values reported by Sharma *et al.* (1973) and Radostits *et al.* (1994).

The total leukocyte count slightly increased to $13.50 \pm 0.33 \times 10^3$ / µl and $13.71 \pm 0.54 \times 10^3$/ µl in groups I and II at zero hour respectively. This non-significant increase in total leukocyte count at zero hour of this study concur with the findings of Basak *et al.* (1993b). This could be due to the effect of endotoxins of ruminal origin (Dunlop, 1972).

5.3.5. Differential leukocyte count (DLC)

The differential leukocyte count obtained in control animals was comparable to the values reported by Benjamin (1985) and Shihabudheen (1998).

There were significant neutrophilia and lymphopenia noticed in group I and II at zero hour and 24 hour of this study. These observations were in agreement with the findings of Prasad *et al.,* (1972) and Basak *et al.* (1993b).

The significant neutrophilia and lymphopenia noticed at zero hour and 24 h in the present study could be due to the increased heart rate (physiological leukocytosis) or because of the release of corticosteroids due to stress (Jain, 1986).

5.4. Serum biochemistry

5.4.1. Serum bicarbonate

The serum bicarbonate level obtained in the control animals was 26.83 ± 0.38 mEq/ l. These were comparable with the values reported earlier by Braun *et al.* (1992).

In groups I and II, the serum bicarbonate level were 15.48 ± 1.23 mEq/ l and 14.12 ± 1.40 mEq/ l at zero hour and it was found to be significantly reduced ($p \leq 0.01$) when compared to control values. Michell (1990) stated that the generation of abnormal acid (H^+) load due to excess production and absorption of lactic acid in the rumen resulted in depletion of serum bicarbonate.

At 12 h, a significant increase ($p \leq 0.01$) was noticed in the serum bicarbonate level to 26.18 ± 0.87 mEq/ l in group II (comparable with control values) when compared to group I where it was only 22.35 ± 0.10 mEq/ l. This indicates early clinical recovery in animals of group II when compared to the animals of group I.

A significant increase noticed in the serum bicarbonate in group I and II to 30.03 ± 0.69 and 29.44 ± 0.38 mEq/ l respectively at 48 h is suggestive of conversion of excess lactate to bicarbonate in the liver (Dunlop, 1972).

When compared to the normal serum bicarbonate level of control animals, the base deficit in animals of group I varied from -7.43 to -14.73 mEq/ l whereas in group II it was -8.03 to -18.43 mEq/ l.

In the present study, five animals in group I and four animals in group II had moderate acidosis and one animal in group I and two animals in group II had severe acidosis based on their serum bicarbonate level and the base deficit derived from it. This classification was based on the report of Braun *et al.* (1992) who stated that a base deficit of -14 and above comes under severe acidosis and a base deficit ranging from -6 to -14 comes under moderate acidosis in goats.

5.4.2. Serum lactic acid

The normal level of serum lactic acid obtained in control animals was 15.45 ± 0.38 mg/ dl, which was comparable to the values reported by Pillai (1988) and Shihabudheen (1998).

A significant increase ($p \leq 0.01$) was noticed in the serum lactic acid level at zero hour, 24 h and 48 h in groups I and II. These were in agreement with the findings of Vihan *et al.* (1982), Tanwar *et al.* (1983) and Pillai (1988) in experimental ruminal acidosis in goats.

Increased serum lactic acid may be due to the release of high amount of lactic acid following excessive fermentation of carbohydrate in the rumen and its subsequent absorption (Dunlop, 1972; Sen *et al.*, 1993).

The serum lactic acid level in animals of group I and II reduced over a period of 24h to 48h and became almost normal by 72h of study. Parenteral administration of systemic alkalinisers might have helped in increasing the alkali reserve of the blood

along with improved metabolism which could reduce the blood lactic acid concentration gradually (Prasad and Rekib, 1975; Das and Misra, 1991).

At 72 h of study, the serum lactic acid became normal in animals of group II whereas in group I a significant increase ($p \leq 0.05$) was still noticed when compared to the control values. This indicates the efficacy of carbicarb infusion in correcting the systemic acidosis in group II animals much earlier than the sodium bicarbonate treated group I animals.

5.4.3. Total serum protein

The total serum protein obtained in control animals was 6.68 ± 0.13 g/ dl which was comparable with the values reported by Benjamin (1985) and Pillai (1988).

A significant increase ($p \leq 0.05$) in the serum protein was noticed at zero hour in groups I and II, suggested haemoconcentration in the affected animals.

5.4.4. Serum potassium

The normal level of serum potassium in control animals was 4.72 ± 0.06 mEq/ l which was in agreement with the values reported by Das and Misra (1991) and Shihabudheen (1998).

In the present study no significant changes were noticed in the serum potassium level through out the observation period. This finding agrees with the observations made in clinical cases by Braun *et al.* (1992). But Das and Misra (1991) reported a decreased serum potassium level in induced lactic acidosis in goats.

5.5. Therapeutic management

5.5.1. Systemic alkalinisers

Systemic alkalinisers used in the present study included five per cent sodium bicarbonate infusion in group I and a five per cent mixture of sodium carbonate and sodium bicarbonate each at a dose rate of 3 mEq/ kg body weight.

In animals of group I, successful recovery in goats with serum bicarbonate level ranging from 12.1 to

19.4 mEq/ l was observed whereas in group II (carbicarb treated) successful recovery was noticed in animals with serum bicarbonate ranging from 11.3 to 18.8 mEq/ l. These findings agree with similar reports of Braun *et al.* (1992).

In the present study, carbicarb and sodium bicarbonate at a dose rate of 3 mEq/ kg was found to be effective in treating acute acidosis with base deficit ranging from -7.43 to -14.73 mEq/ l and -8.03 to -15.53 mEq/l respectively.

The above dose requirement of 3 mEq/ kg is the requirement for acute cases of lactic acidosis (Radostits *et al.* 1994). For peracute cases of lactic acidosis where the base deficit reaches above -15 mEq/ l a dose rate of 4.5 mEq./kg is required (Radostits *et al.* 1994). This could be the reason why in one animal in group II with serum bicarbonate of 8.4 mEq/ l and a base deficit of -18.43 mEq/l, use of carbicarb at a dose rate of 3 mEq/kg was not sufficient to raise the blood bicarbonate level to normal, leading to the death of the animal.

Another finding in the carbicarb treated group II animals was that one animal with a base deficit of -15.53 mEq/l (severe acidosis) was treated effectively with carbicarb at a dose rate of 3 mEq/ kg which prove the better efficacy of carbicarb as a systemic alkaliniser in the therapy of lactic acidosis in goats.

The earlier clinical recovery noticed in animals of group II than in group I animals could be due to the elevation of blood bicarbonate concentration without changing the blood PCO_2 on administration of carbicarb parenterally (Bersin *et al.*,1988; Filley and Kindig, 1984). Thus the use of carbicarb as systemic alkaliniser in group II animals was found to be more effective than use of sodium bicarbonate in group I animals in the treatment of acute lactic acidosis in goats.

5.5.2. Oral alkalinisers

Following administration of magnesium hydroxide as oral alkaliniser at a dose rate of one gram per kg body weight, the rumen pH increased and came to a normal range of 6.5 to 7.0 at 24 h of study in all animals of group I and II. These findings agree with the observations of Tanwar *et al.* (1983). This normal rumen

pH might have helped in the multiplication of the microflora and also it reduced the absorption of lactic acid (Dunlop, 1972).

5.5.3. Rehydration therapy

Restoration of the circulatory volume using normal saline helped in restoring the peripheral circulation and also improved hepatic perfusion which in turn helped to resolve associated lactic acidosis (Michell, 1990).

5.5.4. Supportive therapy

Injection of dexamethasone have helped in reducing the development of shock in affected animals (Radostits *et al.*, 1994). The doughing of rumen with normal saline and partial evacuation of rumen contents might have helped in the removal of ingesta causing lactic acidosis and also much of lactic acid awaiting absorption (Michell, 1990). This finding was also supported by the reports of Radostits and Dunlop (1971) who stated that a satisfactory treatment for lactic acidosis include emptying of rumen in order to permit normal fermentation to be re-established.

Partial removal of ruminal contents probably have also helped in recovery from acidosis by elimination of some of the toxic metabolites and endotoxins present in the rumen, which concurred with the earlier findings of Sinha *et al.* (1985).

Administration of oxytetracycline orally helps in controlling the growth of bacteria, which produce lactic acid in the rumen (Radostits *et al.*, 1994). The transfer of fresh rumen liquor from healthy animals and administration of oxytetracycline orally in the present study also have helped in restoring the normal fermentation as reported by Dunlop (1972) and Sinha *et al.* (1985).

SUMMARY

6. SUMMARY

Ruminal acidosis is the most common digestive disorder in goats and it occurs due to accidental ingestion of large quantity of highly fermentable carbohydrates without adaptation.

Adult goats of either sex, weighing 15-25 kg brought to the veterinary college hospitals at Mannuthy and Kokkalai with history and symptoms suggestive of ruminal acidosis were utilised for the present study. The animals were divided into two groups of six each (group I and II) at random. Six healthy animals served as controls.

In group I and II animals, detailed clinical examination of each case was done and recording of clinical data, sampling and analysis of rumen liquor and blood were also conducted at zero hour, one hour, six hour, 12 h, 24 h, 48 h and 72 h of the study.

Therapeutic management of group I was done by administering five per cent sodium bicarbonate solution intravenously at a dose rate of five millilitre per kg body weight (3 mEq/ kg body weight), whereas in group

II, it was replaced with five per cent mixture of sodium carbonate and sodium bicarbonate (carbicarb) intravenously at a dose rate of four millilitre per kg body weight (3 mEq/ kg body weight).

In both groups I and II, Dexamethasone 20 mg was given intravenously. The rumen contents were then evacuated after irrigating with one litre of normal saline. Magnesium hydroxide powder one gram per kilogram body weight and oxytetracycline 500 mg were given orally. Supportive treatments were done with fluids, vitamin B complex injection daily and transfer of rumen liquor at 2nd or 3rd day till the appetite and rumen motility regained to normal.

The clinical signs exhibited by animals with ruminal lactic acidosis were anorexia, dullness, low carriage of head, dry muzzle, sunken eyes, tenting of skin, grinding of teeth, oliguria and distended abdomen. In some animals, cold extremities, foul smelling diarrhoea, retracted eyeballs and grunting on respiration was also noticed.

Although the respiratory rate was high when the treatment was initiated, significant reduction in

respiratory rate was noticed in animals of group II when compared to group I from one hour to 24 h of treatment. Improvement in the quality of pulse was also noticed during this period. The rumen motility was initially absent in all animals of group I and II, but regained to normal after treatment.

In animals of group I and II, the rumen liquor showed a significant reduction in pH and it was milky grey in colour with watery consistency and had an intense sour smell. Because of effective treatment in both groups, the rumen liquor regained the normal pH and became olive green in colour with thick consistency and aromatic smell. The protozoal motility also regained after treatment in both groups. The rumen liquor revealed improvement in SAT and MBRT at the end of treatment in group I and II.

The TVFA concentration of the rumen liquor showed a gradual increase from zero hour to 72 h of the study in animals of group I and II. The mean lactic acid concentration in the rumen liquor in animals of group I and II recorded a very high value at zero hour when compared to control animals and the values became comparable in all groups by 72 h of treatment.

Haemoconcentration was noticed in animals of group I and II at zero hour as evinced by increased blood haemoglobin, packed cell volume, total erythrocyte count and total leukocyte count. In differential leukocyte count neutrophilia was noticed in group I and II.

A significant reduction in the serum bicarbonate was noticed in group I and II at zero hour when compared to control values. At 12 h and 24 h serum bicarbonate level in group II was significantly higher than in group I.

The serum lactic acid content showed a significant increase at zero hour to 48 h in group I and II when compared to control animals. By 72 h no significant difference noticed between group II when compared to controls, but in group I it was still higher. The total serum protein level revealed a significant increase from zero hour to 48 h of study, but became normal by 72 h. No significant changes noticed in the serum potassium level among controls, group I and group II.

The clinical assessment at 48 h revealed that all animals in group II appeared more active and alert than

group I at 12 h and 24 h. All the animals in group I and II recovered after treatments except one animal in group II which died at eighth hour of treatment.

From this study it can be concluded that five per cent carbicarb is a very effective and safe parenteral alkaliniser solution and can replace five per cent sodium bicarbonate infusion in the treatment of lactic acidosis in goats.

Further studies are warranted with the help of blood gas analyser in more number of clinical cases of lactic acidosis in goats.

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CLINICO-THERAPEUTIC STUDY OF RUMINAL LACTIC ACIDOSIS IN GOATS

By

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ABSTRACT OF THE THESIS

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ABSTRACT

Twelve goats with history and symptoms suggestive of ruminal acidosis were divided into two groups (I and II). Six healthy goats were also served as controls. Detailed clinical examination, recording of clinical data, sampling and analysis of rumen liquor and blood were done at zero hour, one hour, 12 h, 24 h, 48 h and 72 h of the study. Therapeutic management of group I was done by giving five per cent sodium bicarbonate solution intravenously at a dose rate of 5 ml / kg ; whereas in group II it was replaced with a mixture of sodium carbonate and sodium bicarbonate (carbicarb) intravenously at a dose rate of 4 ml / kg. Animals of group I and II were given dexamethasone 20 mg intravenously, evacuated rumen contents and given magnesium hydroxide and oxytetracycline orally. Supportive treatments were done with fluids, vitamin B complex injection daily and transfer of fresh rumen liquor on second or third day in both groups. The clinical signs noticed were anorexia, dullness, low carriage of head, dry muzzle, sunken eyes, tending of skin, oliguria, distended abdomen, cold extremities and retracted eye balls. There was improvement in the

pulse quality and also considerable reduction in respiratory rate noticed in animals of group II than in group I at 24 h of treatment. In both groups, the rumen liquor colour, odour, consistency and protozoal motility became normal, SAT and MBRT showed improvement and elevated rumen liquor lactic acid became normal by 72 h. Elevated haemoglobin, PCV, TEC, TLC and neutrophilia also became normal. Improvement in serum bicarbonate level was marked in group II than in group I at 24 h and 48 h of treatment. Serum lactic acid showed significant increase at zero hour in group I and II, but came to normal in group II at 72 h, but not in group I. Elevated serum protein level at zero hour also came to normal. Clinical assessment throughout the study revealed that all animals of group II appeared more alert and active and recovered earlier than in group I.