"Clinico-Biochemical and Therapeutic Studies of Experimental Ruminal Acidosis in Goats"

THESIS

SUBMITTED TO THE

RAJENDRA AGRICULTURAL UNIVERSITY

(FACULTY OF VETERINARY SCIENCE AND ANIMAL HUSBANDRY)

PUSA, (SAMASTIPUR), BIHAR

In partial fulfillment of the requirement

FOR THE DEGREE OF

Master of Veterinary Science

(VETERINARY MEDICINE)

By

Abhishek Kumar

Reg. No. M/VM/29/1999-2000

DEPARTMENT OF VETERINARY MEDICINE

BIHAR VETERINARY COLLEGE

PATNA-800014, BIHAR (INDIA)

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Department of Veterinary Medicine, Bihar Veterinary College, Patna-14

Rajendra Agricultural University, Pusa, Samastipur, Bihar.

CERTIFICATE –II

We, the undersigned, members of the advisory committee of **Dr. Abhishek Kumar,** a candidate for the degree of master of veterinary science with major in veterinary medicine have gone through the manuscript of the thesis and agree that the thesis entitled *"CLINICO-BIOCHEMICAL AND THERAPEUTIC STUDIES OF EXPERIMENTAL RUMINAL ACIDOSIS IN GOATS"* may be submitted by **Dr. Abhishek Kumar** in partial fulfillment of the requirements for the degree.

S Verma 2-9-02

(Dr. S.P. Verma)

Chairman of Advisory Committee

Members of advisory committee:

1. **Dr. B.P. Sinha** 2.9.2002
   Assoc. Prof. & Head, Deptt.
   of Vety. Clinical Medicine

2. **Dr. C. Jayachandran** Jayachandra 2/9/02
   Assoc. Prof., Deptt. of Vety.
   Pharmacology & Toxicology

3. **Dr. S.R.P. Sinha** 2/9/02
   Assoc. Prof. & Head Deptt. of Vety. Parasitology
   (Nominee, Dean, Post-Graduate Studies)

Department of Veterinary Medicine, Bihar Veterinary College, Patna-14

Rajendra Agricultural University, Pusa, Samastipur, Bihar.

CERTIFICATE –I

This is to certify that the thesis entitled *"CLINICO-BIOCHEMICAL AND THERAPEUTIC STUDIES OF EXPERIMENTAL RUMINAL ACIDOSIS IN GOATS"* submitted in partial fulfillment of the requirements for the award of master of veterinary science (veterinary medicine) in the faculty of Post-Graduate studies, Rajendra Agricultural University, Bihar is the record of bonafide research work carried out by **Dr. Abhishek Kumar** under my supervision and guidance. No part of the thesis has so far been submitted for any other degree or diploma.

It is further certified that the assistance and help received during the course of this investigation and sources of literature have been fully acknowledged.

S Verma 2.9.02

(Dr. S.P. Verma)

Major Advisor

Department of Veterinary Medicine, Bihar Veterinary College, Patna-14

Rajendra Agricultural University, Pusa, Samastipur, Bihar.

CERTIFICATE –III

This is to certify that thesis entitled *"CLINICO-BIOCHEMICAL AND THERAPEUTIC STUDIES OF EXPERIMENTAL RUMINAL ACIDOSIS IN GOATS"* submitted by **Dr. Abhishek Kumar** in partial fulfillment of the requirements for the degree of master of veterinary science (Veterinary Medicine) of the faculty of Post-Graduate studies, Rajendra Agricultural University, Pusa, Samastipur, Bihar was examined and approved on ..20.-01.-.. 2003

S Verma 20-01-03

(Dr. S.P. Verma)

Chairman, Advisory/Examination Committee

Members of advisory committee:

1. **Dr. B.P. Sinha** 20.1.2003
   Assoc. Prof. & Head, Deptt.
   of Vety. Clinical Medicine

2. **Dr. C. Jayachandran** Jayachandran 20-01-03
   Assoc. Prof., Deptt. of Vety.
   Pharmacology & Toxicology

3. **Dr. S.R.P. Sinha** 20-01-03
   Assoc. Prof. & Head Deptt. of Vety. Parasitology
   (Nominee, Dean, Post-Graduate Studies)

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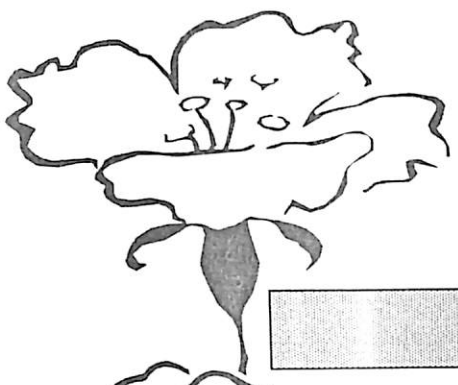
Last but not least, I would like to express my heartiest belief to Almighty God and thank God for whatever he has given me and also for whatever he did not given me.

Abhishek Kumar

Abhishek Kumar

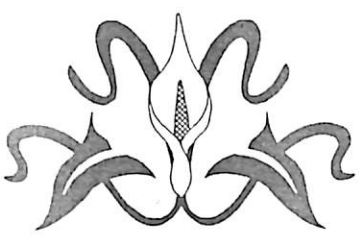
Place : Patna

Date : 2-09-02



CONTENTS

CHAPTER - I



INTRODUCTION

INTRODUCTION

India is predominantly an agricultural country where animal husbandry practices contribute significant role in the survival and betterment of the people in general and rural population in particular. In pastoral and agricultural subsistence society in India, goats are kept as a source of additional income and as an insurance against disaster.

The goat keeping is an enterprise, which have been practiced by a large section of population in rural areas. It plays a significant role in the economy and nutrition of landless, small and marginal farmers in the country. Its multipurpose utility in producing meat, milk, fiber, skin and manure make this animal admirably suited in the mixed farming system of small and marginal farmers. Because of its ability to serve the mankind even under very harsh ecological and economical conditions, the species has earned the names as "poor man's cow", "kitchen cow", "cottage industry" and "gandhian animal". Not only that, goat has been also termed as walking refrigerator for the storage of milk and can be milked number of times in a day. With a total population of 115.278 million goats, Indian ranks first in the world and with a total of 17461 thousand goats, Bihar stands first among the states in our country (Livestock census, 1992, Govt. of India). The goat contributes about 2.20 million tonnes of milk, 0.47 million tonnes of meat and 0.109 million tonnes of skin. The slaughter rate of goat is also at the level of 39.7% as compared to 31.8% and 11% for sheep and buffaloes respectively (FAO production year book, 1992). Looking to these economical importance it becomes imperative that proper attention will have to be paid for the maintenance of their health.

Goats, like other ruminants have well defined compartments namely rumen, reticulum, omasum and abomasum. Functionaly, rumen is the most momentus organ which takes major work of gastro-intestinal tract. The function of rumen is mainly fermentive and digestive. The fermentation of rumen is maintained by numerous micro-organisms, these microflora of rumen includes cilliates, polymastigates, oscillospires, flagellates, selenomonads, small bacteria mainly gram negative and fungi predominating the rumen culture (Hungate, 1966). Besides these for distinct fermentive digestion in ruminants, optimal rumen motility, concentration of living micro-organism, anaerobiasis, correct pH of rumen liquor, fluidity of rumen mass and balanced substrate contributions are essential for normal digestion (Randhawa, 1979). Optimal microbial activities can be achieved, when buffering capacity of the rumen works satisfactory because pH is the determining point, change in which causes destruction of ruminal microbial population and impedes the digestive and fermentive phenomenon.

Normally goat utilizes grasses, forage and leaves better than cattle and buffalo and able to maintain their normal digestive physiology with forage. However, many a times farmers use to give more feeds or make sudden changes in various feeds and fodder constituents for obtaining more benefit in terms of meat, milk and other products. Such incidence are very frequently occurring in stall fed goats under intensive managemental system, which are gaining popularity due to non-availability of adequate grazing pastures. In fact, for distinct fermentative digestion in ruminants any accidental change in feed, fodder and management leads to alteration of physio-chemical condition of rumen resulting to numerous rumen dysfunctions. This dysfunction sometimes are severe enough to cause death

of the animals. One of the most important dysfunctions of rumen is "lactic acidosis", other wise known as ruminal acidosis, toxic indigestion, acute carbohydrate engorgement, acute impaction, over eating disease, grain over load or founder disease etc. (Chakarborti, 1988).

Ruminal lactic acidosis is an accidental problem which occurs when ruminants consume excessive amount of readily fermentable carbohydrate rich feed. After ingestion the ruminal environment get altered and gram positive bacteria like *Streptococcus bovis, Lactobacillus* proliferate which leads to the production of large quantities of lactic acid in the rumen (Blood *et al.*, 1983). As the lactic acid as well as other acids accumulates in the rumen, the pH get decline ultimately causing a shift in the ruminal microbial population. The accumulation of excessive amount of acid in the rumen results into absorption of acid into blood (Ahrens, 1967) which provides a tremendous changes to the buffering capacity of rumen & other body fluid, leading to systemic and metabolic acidosis or lactacedemia.

Lactic acidosis damages almost all the vital organ of the body and this damage is beyond repair if not treated well in time which renders significant economic loss in terms of wasted feed, delayed marketing, condemnation of rumen, liver or entire carcass, lowered nutritional value, reduced water binding capacity of meat and several organoleptic deficiencies (Blood and Radostits, 1989). Rumen acidosis also hampers the growth rate, production and reproductive performance of the affected animals.

The management of acidosis is a challenging task to the clinicians due to its high fatality rate and it may extend upto 90% in untreated and 30 to 40% in treated animals (Blood and Radostits, 1989). Many workers tried different combination of therapy to combat the problem of lactic acidosis

and used Sodium bicarbonate (Kesari and Naylor, 1984, Okhrimenko *et al.*, 1989), Magnesium carbonate (Tanwar *et al.*, 1983) and Calcium carbonate (Amstel, 1983) as alkalizing agents both orally and parentraly. Besides these antibiotics viz. Oxytetracycline (Schukken *et al.*, 1985) & Benzyl penicillin (Tanwar & Mathur, 1983b) were used to kill gram positive organisms. B-complex with liver extract and anti-histaminic preparations were used as supportive therapy (Flachowsky *et al.*, 1974) and for the restoration of ruminal microflora, cud transplantations (Hoflund, 1967) were tried.

Rumen dysfunction syndrome has been extensively studied in details for its bio-chemical aspects in bovine (Prasad *et al.*, 1972, Vihan, 1978) and in sheep (Juhas'z and Szegedi, 1968a). However, there is paucity of information regarding the bio-chemical changes in experimentally induced acidosis in goats & their proper treatment. Hence, keeping in view the above facts into the consideration & its economic importance particularly for the people living below the poverty line, its contribution to the nations economy, paucity of information regarding the clinico-biochemical changes & proper treatment to ailing animals, the work has been undertaken with following objectives :-

(1) To induce experimental ruminal acidosis in goats by oral feeding with whole wheat grains.

(2) To study the course of ruminal acidosis thus produced (clinical aspects).

(3) To study some of the various bio-chemical alterations in rumen fluid, blood and serum of acidotic goats.

(4) To evolve a suitable therapeutic management for acidotic goats.

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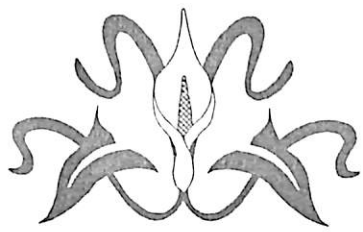
CHAPTER - II



REVIEW OF LITERATURE

REVIEW OF LITERATURE

Ruminal acidosis is the principal manifestation of ruminal pH alteration in the farm animals where ruminal digestion occurs. When ruminents consume excessive amount of readily fermentable carbohydrate rich diet, the rate of lactic acid production may exceed the rate of its metabolism to volatile fatty acid. As the lactic acid as well as other acids accumulate in the rumen, the rumen pH decline, causing a shift in the ruminal microbial population. Bacteria capable of decarboxylating the amino-acids, proliferate at lower pH leading to the acidosis. The disease is characterised by anorexia, ruminal atony, dullness, diarrhoea, sluggish eye reflexes, staggering gait, dehydration, increased pulse and respiration rates. **The literature on acidosis has been reviewed under the heads given below :-**

(1) Production and occurrence of acidosis with toxic feeds and compound.

(2) Clinical manifestations.

(3) Physical examination of rumen liquor.

(4) Biochemical changes in rumen liquor.

(5) Biochemical changes in blood and serum.

(6) Therapeutic management.

2.1. Production and occurrence of acidosis with toxic feeds and compound :

Shinosaki (1959) dosed 0.2 moles of lactic acid in goats which developed ruminal acidosis.

Gnanaprakasam (1970) reported 47 clinical cases of rumen acidosis in goats caused by accidental over eating of paddy rice.

Dunlop (1972) reviewed clinical cases of acid indigestion attributable to ingestion of wheat, barley, oats, bakery products, apple, mixed concentrate feeds & unripe green corn standing in the field. He mentioned rye, sorghum, potatoes, fodder beets, sugar beets, pears, whey, molasses and brewer grains as being responsible for causing acidosis.

Allison *et al.* (1975) induced rumen acidosis in sheep (weighing 40 to 50 kg) by administering 1.36 kg of crushed wheat suspended in 1 litre of water into rumen for two successive days.

Terashima *et al.* (1978) produced lactic acidosis in hay fed sheep by injecting lactic acid intravenously or intra ruminally.

Kezar and Church (1979b) induced rumen acidosis in sheep by feeding wheat at the dose rate of 50gm/75kg body weight.

Muir *et al.* (1980) reported wheat induced acidosis in lamb @ 40gm/kg body weight following starvation.

Sen (1982) induced rumen acidosis in goat by administering crushed rice at the dose rate of 80gm/kg body weight through rumen fistual after 36 hours of fasting.

Vihan *et al.* (1982) produced experimental rumen acidosis in goats by three treatments viz. administration of 1.00 kg crushed barley, 1.0 kg crushed grain (barley 50% and wheat 50%) and 500.00 gms sugar cane through rumen fistula.

Tanwar *et al.* (1983) were able to produce rumen acidosis in three groups of goats by administering whole wheat grain directly in to the rumen through rumen canula at the dose rates of 80, 100 and 120gm/kg body weight respectively.

Sen *et al.* (1984) induced acute ruminal acidosis in goats using crushed rice @ 80 gm/kg body weight.

Sobhanan and Aleyas (1986) noted that goats suffering with convulsive seizures in Kerala was due to excess feeding of cooked rice. The cases were attributed to ruminal acidosis associated with encephalomalacia.

Cao *et al.* (1987) fed sucrose at the dose rate of 18 gm/kg body weight to goat to induce lactic acidosis.

Crichlow (1989) produced acidosis in sheep by intraruminal administration of finely ground wheat @ 50gm/kg body weight.

Lal *et al.* (1989) experimentally induced ruminal acidosis in six goats by intraruminal adminstration of whole wheat grains @ of 100gm/kg body weight following fasting for 24 hours.

Das and Misra (1991) produced rumen acidosis experimentally in Black Bengal goat by intraruminal administration of crushed rice @ 80gm/kg body weight.

Patra (1991) produced acute ruminal acidosis in adult sheep by feeding crushed wheat @ 90gm/kg body weight.

Das *et al.* (1992) induced experimental acidosis by intra-ruminal administration of crushed rice @ 80gm/kg body weight after 36 hours of fasting in Black Bengal goats.

Lal *et al.* (1992) induced ruminal acidosis infistulated goat by giving whole wheat grains intraruminally @ 100gm/kg body weight after 24 hours of fasting with *adlib* water.

Tripathy and Mishra (1992) induced acid indigestion in 3 goats by giving flour and water via a stomach tube after overnight fasting.

Lal *et al.* (1993) induced acute rumen lactic acidosis in goats by intra ruminal administration of whole wheat grain @ 100gm/kg body weight.

Patra *et al.* (1993) induced ruminal acidosis in 6 adult sheep by oral feeding of wheat grain @ 90gm/kg body weight.

Sen *et al.* (1993) induced acute ruminal acidosis in 6 female Barbari goats by the intraruminal administration of 80gm/kg body weight of crushed rice, after 36 hours fasting.

Angelov *et al.* (1995a) induced acute rumen acidosis in 8 adult goats by a single oral administration of beet molasses @ 20ml/kg of body weight.

Aslan *et al.* (1995) induced ruminal acidosis in 10 month old west African dwarf goats by feeding a suspension of 80gm wheat flour/kg body weight through a stomach tube.

Angelov *et al.* (1996) induced acute acidosis with oral administration of a single dose of beet molasses, 20ml/kg body weight, through a stomach tube, immediately after the morning feeding.

Patra *et al.* (1996) induced acidosis in 6 ewes by feeding soaked wheat @ 90gm/kg body weight after 24 hours of starvation.

Patra *et al.* (1997) induced lactic acidosis in healthy female sheep by feeding with soaked wheat grain @ 90g/kg body weight after a 24 hours fasting.

Nour *et al.* (1998) successfully produced experimental lactic acidosis in Nubian goats using sorghum flour at a dose rate of 50gm/kg body weight given intraruminally through a fixed rumen cannula.

Shukla *et al.* (1999) induced experimental lactic acidosis in 16 calves by feeding wheat flour after 24 hours fasting.

Dwivedi *et al.* (2000) induced rumen dysfunction by oral feeding of crushed wheat grain @ 40gm/kg body weight in 12 male calves, aged 8-12 months and weighing between 40-70 kg, after 12 hours of fasting.

Gupta *et al.* (2000) experimentally induced acidosis in 6 calves by administering crushed wheat @ 50gm/kg body weight.

Chand *et al.* (2001) experimentally induced ruminal acidosis by administering crushed wheat grains @ 50gm/kg body weight orally in 6 calves.

Hawa Singh *et al.* (2001) reported 24 clinical cases of indigestion in goats due to ingestion of either excessive whole wheat grains or wheat flour or stale bread or rice or molasses.

2.2. Clinical manifestation :

Joshi (1969) reported that a goat ingested accidentally wheat grains exhibited the clinical signs such as anorexia, distended abdomen, tympanic sound on auscultation of left flank, pasty faeces, dyspnoea, weak pulse disinclined to move & lateral recumbencey etc.

Gnanaprakasam (1970) reported the clinical manifestations in goats engorged accidentally with grains within 24 hours & classified the symptoms in to mild, moderate and severe forms. In mild form, the important symptoms observed were inappetance, enlarged abdomen, constipation, firm and doughy rumen on palpation, dull sound on percussion and sluggish or absence of rumen motility on auscultation. Moderate cases were characterised by anorexia with either constipation or diarrhoea, dyspnoea, tachycardia, oliguria, absence of rumen motility and gurgling sound on auscultation. Severely affected goats were manifested with dull appearance,

inability to stand for a long time, grinding of teeth, yawning, increased heart rate (120-160/min.) and prominent costal type of respiration.

Das *et al.* (1972) observed rumen acidosis in Indian cattle due to abrupt change in diet from normal to decomposed starchy food and found ruminal pH ranged from 5.0 to 5.7. The animals had mild tympany, abdominal pain and dehydration. The pulse and respiration rates were increased.

Kelly (1974) recorded the pulse, respiration & temp. in normal healthy goats as 70-90/min., 20-30/min. & 102.9^{0}F respectively. The healthy goats & cattle were found to be alert & active in appearance, had glossy skin coat normal posture & gait, pink to rosy conjunctival mucous membrane and good appetite. The normal cattle and goats were found to ruminate during resting period both in standing and lying down position. The frequency of urination was observed to be 1 to 3 times per day.

Vestweber *et al.* (1974) recorded signs and symptoms of rumen overload in sheep which indicated depression, tachycardia, increased respiration rates, ruminal atony, congested mucus membranes, increased rectal temperature, diarrhoea and death.

Allison *et al.* (1975) observed diarrhoea, anorexia, wekness and death in sheep suffering from rumen acidosis.

Rai and Pandey (1980b) reported that total rumination time, frequency of urination and frequency of defaecation in healthy goats as 400.00 minutes,11 & 9.0/day respectively.

Sen (1982) observed symptoms as rise in body temperature, rapid pulse, heart and respiration rates, complete ruminal atony, pasty faeces and diarrhoea, fullness of rumen with water, inappetance, dullness, occasional

grinding of teeth, dyspnoea, dehydration, nasal discharge and death in goats induced rumen acidosis experimentally.

Sen *et al.* (1982) observed the clinical manifestations, like anorexia, rumen stasis, dullness, diarrhoea, constipation, abdominal pain head pressing and grinding of teeth etc. in rumen acidosis of goats.

Tanwar and Mathur (1983a) recorded clinical signs of wheat induced acid indigestion in goats as dullness, depression, anorexia, lack of rumination, atony of rumen, grinding of teeth, increased respiration, increased pulse, anuria, tachycardia, dehydration, sunken eyes, diarrhoea followed by constipation, unsteady gait, lameness, purulent nasal discharge and subnormal temp.

Sen *et al.* (1984) observed the symptoms of acute ruminal acidosis in goats as ruminal stasis, inappetance, dullness, depression, severe dehydration, polydipsia, oliguria.. increased pulse and respiration rates, sunken eyes, roughened body coat and death between 25 and 106 hours post inducement with crushed rice.

Cao *et al.* (1987) observed the signs of weakness, lieing in sternal recumbency from 12 to 48 hours after experimentally induced lactic acidosis in goats. Both mean heart rate and respiration rates gradually rose from 98 and 12/minute respectively at '0' hour to 174 and 17/minute respectively at 24 hours. The rectal temp. remained 38^0C to 39^0C throughout the experiment. All goats exhibited abdominal distension with passage of soft pasty faeces in small quantity.

Chakrabarti (1988) reported that healthy goat have bright eyes, alert and glossy, responding ears, skin with hairs lying evenly over the body coat. They had normal posture and gait. The goats were passing pellety faeces

with dark green colour. The urine had a clear straw colour appearance. He also reported that the normal range of body temp., pulse and respiration rate in healthy goats as 101.5-103.5^{0}F, 60-70/min. & 18-30/min. respectively.

Lal *et al.* (1989) observed symptoms of experimental rumen acidosis in goats which suffered from various degree of anorexia and ruminal stasis following induction of acidosis, as dullness, diarrhoea, constipation, abdominal pain, nasal discharge, head pressing and grinding of teeth etc. There was gradual fall of body temperature associated with marked increase in pulse and respiration rates with significant decrease in ruminal movement.

Das (1990) experimentally induced rumen acidosis in goats & observed the symptoms as rapid pulse & respiration rates, rise in body temp., complete rumen atony, pastey faeces and diarrhoea, inappetence, distended abdomen, dehydration and death.

Hanumanthaiah *et al.* (1990) induced ruminal acidosis in Buffalo calves by drenching ragi slurry and observed general depression, anorexia, whitish to blackish diarrhoea, laboured breathing, suspended rumination increased body temperature, pulse and respirations rates but decreased in ruminal pH, motility and concentration of protozoa, which were returned to normal by 48 to 72 hours.

Patra (1991) reported anorexia, nasal discharges, severe diarrhoea, staggering gait, convulsion and death in acute ruminal acidosis in sheep fed with wheat @ 90 gm/kg body weight orally.

Aslan *et al.* (1995) induced ruminal acidosis in goats and noted the clinical signs as loss of rumination and appetite, trembling gait and watery diarrhoea during the first 24 hours.

Bhikane *et al.* (1996) clinically observed the case of ruminal lactic acidosis in buffalo and recorded the symptoms of depression, milk fever like posture, subnormal temp., tachycardia, dyspnoea, dry muzzle, cold extremities, suspended defaecation and frequent micturition. Rumen was impacted and atonic. Per-rectal examination revealed blackish and foul smelling faeces containing sorghum grain.

Dwivedi *et al.* (2000) experimentally induced rumen dysfunction in calves and found the symptoms of inappetence, dullness and depression by 12 hours post feeding. Dirrhoea and nasal discharge were noted in both the control and treated groups by 36 hours. Both heart and respiration rates were increased moderately.

Nikolov (2000) noted decreased temperature and increased heart and respiration rates with a weak, soft, barely detectable pulse. The mucous membrane became cyanotic. The refusal of food intake, suspended rumination, grinding of teeth and polydypsia were also observed. All sheep suffered with profuse diarrhoea upto 6 hours. Prior to death they were in a comatose state with no reactions to light or sound. The rate and intensity of rumen movement was 2 ± 1/5 minutes at 6 hours and 0/5 minutes at 10 hours.

Chand *et al.* (2001) noticed ruminal atony, loss of appetite, dullness, sluggish eye reflexes, diarrhoea, staggering gait in ruminal acidosis in calves.

Hawa Singh *et al.* (2001) observed symptoms as dullness, depression, anorexia, suspended rumination, decreased in milk yield and constipation or diarrhoea in goats suffering from indigestion.

2.3. Physical examination of rumen liquor :

2.3.1. Macroscopic Examination :

2.3.1.1. Smell/odour :

Gnanaprakasam (1970) observed, putrid smell in cases of moderate and severe rumen acidosis, but not in mild acidosis cases.

Telle and Preston (1971) reported that the content became vile smelling in experimental lactic acidosis in ewes.

Misra *et al.* (1972) observed rumen fluid of animals suffering from indigestion caused by feeding of poor to good quality foods and noted greenish yellow colour with liquid consistency emiting faint sour odour. The infusarial concentration varied from (+) low to (++) moderate. The motility was (+) slow. In case of animals suffering from indigestion caused by feeding good to poor quality foods, rumen fluid emitted faint putrefied (ammoniacal) smell, the colour was brown and consistency was thin watery. The protzoal concentration and motility were same as in indigestion caused by poor to good quality foods.

Wilson *et al.* (1975) observed that the odour of rumen fluid in cattle became progressively sourer as lactic acidosis developed.

Jenkins (1982) described that the rumen fluid emitted a sour odour in ruminal acidosis.

Sen (1982) observed in grain overload goat, that the rumen content became sour at 20^{th} hours.

Sen *et al.* (1982) reported that the increased sourness in smell of rumen liquor was due to increased lactic acid content in acute ruminal acidosis in goats.

Chakrabarti (1988) mentioned that in healthy animals the odour of rumen liquor was aromatic, vinegar like, while in acid indigestion it was pungent and sour.

Pradhan *et al.* (1988) noted that the smell of rumen liquor in healthy Black Bengal goats was aromatic.

Lal *et al.* (1989) reported that the odour of rumen liquor was aromatic at '0' hour and sourness smell increased gradually upto 48 hours showing significant rise in the acid content of rumen liquor which declined from 72 hours in goats wich recovered from rumen acidosis.

Bhikane *et al.* (1996) reported that rumen fluid was greyish white, thick, & sour smelling with pH 4.0 in a case of ruminal lactic acidosis in buffalo.

2.3.1.2. Consistency :

Telle and Preston (1971) reported that the rumen liquor became very fluid and frothy in experimental rumen acidosis in ewes.

Das *et al.* (1972) stated that in acute indigestion, the consistency of rumen liquid became watery.

Misra and Singh (1974) reported thicky consistency of rumen liquor in healthy cattle which became watery in acid indigestion.

Rosenberger *et al.* (1979) observed the consistency of rumen liquor changed from slight viscous to watery in rumen acidosis in cattle.

Sen (1982) reported that the consistency of rumen liquor changed from viscous to watery liquid at 20^{th} hours onward in experimental acid indigestion of goat.

Pradhan *et al.* (1988) observed semi liquid consistency of rumen liquid in healthy Black Bengal goats.

Lal *et al.* (1989) observed that there was change in consistency of rumen liquor from thick to thin upto 72 hours followed by improvement from 96 hours in experimental acidosis in goats.

Randhawa *et al.* (1989a) observed that in lactic acidosis of buffalo calves the consistency of rumen liquor changed to watery by 12 and 6 hours in sub-acute and acute conditions respectively.

Das (1990) observed watery consistency of rumen liquor in experimental rumen acidosis in goats.

Kemal Irmak *et al.* (2000a) observed the colour, consistency and odour of rumen fluid samples in cattle manifested with simple indigestion & noted as yellow, very viscous, watery and devoid of the normal aromatic odour.

Kemal Irmak *et al.* (2000b) observed the ruminal fluid samples of calves clinically reported with forestomach flora and motility insufficiency and noted dark yellow colour, mostly putrid odour, very viscous and watery in consistency and contained undigested plant fibres.

2.3.1.3. Colour :

Gnanaprakasam (1970) observed cases of rumen acidosis in goats & found that the rumen liquor became dark green or grey in colour and had no gas bubbles.

Das *et al.* (1972) stated that in acute indigestion, the colour of rumen fluid changed, and varied from golden yellow to grey colour.

Misra and Singh (1974) observed yellowish brown colour of rumen fluid in healthy cattle which became yellowish in acid indigestion.

Sethuraman and Rathor (1979a) found that the colour of rumen fluid changed from a normal of green, in to brown at 24 hours and creamy at 48 hours & to white at 96 hours post engorgement in cattle and buffaloes.

Jenkins (1982) found milky grey colour of rumen liquor in ruminal acidosis.

Sen (1982) recorded that the colour of rumen liquor changed from dark green or greenish brown to light brown at 4^{th} hours of rumen acidosis in goats and subsequently changed to creamish white.

Sen *et al.* (1982) reported that in acute ruminal acidosis in goats the colour of rumen liquor changed from greenish brown to light grey at 12 hours and greyish at 24 hours.

Chakrabarti (1988) stated that the colour of rumen liquor varied according to types of food and types of indigestion. In green fodder/grass, fodder beat, straw, acid indigestion, alkaline indigestion and impaction the colours of rumen liquor were pure green to greenish olive, grey, yellowish brown, milk grey, dark brown and greenish black respectively.

Pradhan *et al.* (1988) observed green to greenish brown colour of rumen liquor in healthy Black Bengal goats.

Lal *et al.* (1989) observed that the colour of rumen liquor became greyish from 24 hours onwards in experimental rumen acidosis of goats. The normal colour of greenish brown was restored by 120 hours of experiment.

Randhawa *et al.* (1989a) noted that in lactic acidosis of buffalo calves, the colour of rumen liquor changed in to brownish yellow to greyish-yellow between 6^{th} and 12^{th} hours but became normal after 12 hours of the induction of subacute lactic acidosis. However, in acute lactic acidosis, the colour of

rumen liquor changed to greyish-yellow at 60 hours which persisted throughout the experimental period.

Dwivedi *et al.* (2000) observed that the rumen liquor became brownish in colour, after induction of ruminal dysfunction in calves.

2.3.2. Microscopic examination :

2.3.2.1. Motility of ruminal microflora :

Allison *et al.* (1964) reported that feeding of 450gm of cracked wheat through the ruminal fistula in lambs made the protozoa immotile.

Joshi (1969) found that in a case of acid indigestion in bullock, large infusoria were dead and others were feebly motile in a drop of fresh rumen liquor.

Gnanaprakasam (1970) did not find any motile protozoa in rumen liquor of moderate and severe cases of clinical acidosis in goats.

Das *et al.* (1972) reported that in acute indigestion in cattle, all protozoa were non-motile. No living protozoa could be found from rumen fluid having ruminal pH below 5.5.

Misra *et al.* (1972) examined fresh rumen fluid samples from healthy Indian cattle under the microscope and graded protozoal motility from moderate (++) to vigorous (+++). But in case of acute carbohydrate engorged cattle the motility of protozoa changed to absent (-).

Misra and Singh (1974) reported that motility of rumen protozoa of healthy cattle was moderate (++) to high (+++), whereas in acid indigestion it was low (+).

Nauriyal and Baxi (1978) observed that within 48 hours of induction of ruminal acidosis in crossbred cattle and buffalo, all rumen protozoa had died.

Randhawa (1979) noted complete disappearance of ruminal protozoa in rumen fluid in acidotic buffalo calves.

Sandha (1980) produced acidosis in buffalo calves and found vigorous (+++) motility of protozoa at '0' hours. At 12 hours post induction, sluggish (+) motility was observed with no living protozoa in the rumen liquor after 18-72 hours. Only a few protozoa started appearing slowly and gradually in surviving animals.

Sen (1982) found that the protozoal motility was moderate to vigorous at '0' hour and moderate at 4^{th} hours and absent at 8^{th} hours onwards of grain overload in goats.

Sen *et al.* (1982) found complete absence of protozoa by 12 hours and their re-appearance after 64-72 hours of post treatment. However, vigorous motility of protozoa was only noted after 80 hours of treatment.

Li *et al.* (1984) induced experimental acidosis in sheep and found loss of protozoal motility.

Sinha *et al.* (1985) reported a complete disappearance of protozoal activity by 24 hours after engorging buffalo calves with wheat.

Pradhan *et al.* (1988) stated that moderate (++) to vigorous (+++) motility and moderate (++) to high (+++) concentration of rumen protozoa in healthy Black Bengal goats.

Lal *et al.* (1989) observed that there was complete disappearance of ruminal protozoa from 12 to 72 hours in experimental rumen acidosis in goats.

Randhawa *et al.* (1989a) observed that in sub acute acidosis of buffalo calves, the protozoal motility gradually became sluggish at 6 hours and diminished to nil by 12 hours. The motility of the reestablished rumen

protozoa was sluggish at 120 hours and moderate at 144 hours. In acute acidosis complete absence of protozoal motility was observed at 6 hours.

Das (1990) observed that the concentration, motility and iodophilic nature of rumen protozoa in healthy goats were ++/+++ (moderate to high), ++/+++ (moderate to vigoruous) and ++/+++ (moderate to high) respectively. But these were all nil or absent in rumen acidosis.

Mukherjee and Sinha (1990) observed that the total protozoal count in Black Bengal goats varied from 0.25 to 2.83 million/ml with an average of 0.96 million/ml of rumen fluid.

Basak *et al.* (1993a) stated absence of rumen protozoal motility from 12 to 84th hours of experimentally induced lactic acidosis in goats.

Bhikane *et al.* (1996) reported that the microscopic examination of ruminal fluid revealed absence of the live ruminal protozoa.

Shukla *et al.* (1999) found complete absence of protozoal motility and rumen movement at 36 hours post induction of acidosis.

Dwivedi *et al.* (2000) reported that there was decreased ruminal motility with respect to frequency and amplitude by 36 hours post feeding.

Chand *et al.* (2001) reported that following induction of ruminal acidosis in calves, bacterial and protozoal count were significantly ($P<0.01$) decreased to 12.53 ± 0.25 and 0.00 ± 0.00 respectively at 48 hours of observation.

Hawa Singh *et al.* (2001) noticed that the total protozoal count was significantly low (25.0 ± 1.30 X 10^4/ml of RL) in indigestion in goats, whereas in healthy goats the values was 42.09 ± 2.13 X 10^4/ml of RL.

2.4. Biochemical changes in rumen liquor :

2.4.1. pH of rumen liquor :

Brandly and Jungher (1955) noted that pH of normal ruminal ingesta ranged from 5.9 to 7.4.

Ahrens (1967) reported that engorgement with wheat reduced the pH of ruminal ingesta to 4.0, while engorgement with pears reduced the pH to 4.28.

Hoflund (1967) stated that a ruminant was said to be suffering from acid indigestion if its ruminal pH was found in the range of 4.0 to 5.5.

Joshi (1969) reported a case of accidentally access of wheat during thrashing operation, in which the ruminal fluid pH was 5.5.

Eadie *et al.* (1970) fed concentrate diets *adlibitum* to cattle and observed that the rumen pH drooped from 6.4 to 5.2.

Gnanaprakasam (1970) recorded rumen pH value (4.6 to 5.2) in severe cases and (5.2 to 5.5) in moderate cases and no marked change in mild cases of ruminal acidosis in goats.

Reddy and Nair (1971) observed that the pH of rumen liquor at 2^{nd}, 4^{th}, 6^{th} and 8^{th} hours had 6.05 ± 0.04, 5.70 ± 0.04, 6.00 ± 0.04 and 6.35 ± 0.03 respectively in healthy Jamunapari-Malabari cross-bred female goats.

Allison *et al.* (1972) fed wheat grains to 3 adult sheep on two successive days. They found that both the ruminal and caecal pH dropped below 5.0 within 48 hours of the first rumen overload. Caecal pH returned to more than 7.0 within 48 hours of the second dosage with wheat but ruminal pH remained low for extended periods.

Das *et al.* (1972) reported that acute indigestion in cattle due to sudden changes in feed was accompanied by a drop in the ruminal pH to 6.3 after consumption of whole wheat.

Chaplin and Jones (1973) observed that the pH of rumen liquor decreased to 4.4 after 12 hours of induction of rumen acidosis with barley in sheep.

Shinosaki and Nakabayashi (1974) noted fall in rumen pH to 4.64 and that of blood to 7.3 in sheep suffering from acidosis.

Vestweber *et al.* (1974) reported that there was fall of pH of rumen liquor to 4.52 ± 0.52 in experimental rumen acidosis in sheep.

Wilson *et al.* (1975) engorged 2 rumen fistulated cattle with sorghum-wheat molasses mixture. Rumen pH was found to drop from 7.1 pre-engorgement to 3.8 at 30 hours and 3.6 at 48 hours post-engorgement.

Koer *et al.* (1976) observed that the mean pH of rumen liquor dropped from 6.77 to 4.74 at 4^{th} hours of corn engorgement in sheep and remained low untill about 60^{th} hours and then rose gradually to 5.90 by 99^{th} hours of post-engorgement.

Beede and Farlin (1977) induced acidosis in sheep by engorging ground wheat @ of 34.5gm/kg body wt. and reported that ruminal pH decreased steadily from 6.22 to 5.22 in the first 8 hours.

Prasad (1977) observed positive correlation between pH and TVFA in sugar induced ruminal acidosis in cattle.

Irwin *et al.* (1979) noted fall of the pH of rumen liquor from 6.97 to 4.70 in glucose induced acidotic sheep.

Kezar and Church (1979a) observed that in sucrose induced acidosis in sheep the pH of rumen liquor was within 4.13 and 4.53.

Rai and Pandey (1980a) reported that the pH of rumen liquor in three groups of goats maintained on free range, range supplemented with

concentrate and concentrate mixture. The mean pH values of rumen liquor was 5.313 ± 0.038, 6.155 ± 0.045 and 6.29 ± 0.059 respectively.

Sen (1982) observed, fall in mean pH of rumen liquor from 6.86 ± 0.17 to lowest mean value 4.37 ± 0.12 at 12 hours in grains overload goats.

Vihan *et al.* (1982) observed lowest pH values as 5.5, 4.5 and 4.0 at 12^{th}, hours of barley, 42^{th} hours of grain, and 12^{th} hours cane sugar induced rumen acidosis respectively in goats.

Tanwar and Mathur (1983a) reported that the pH of rumen liquor changed from 7.33 ± 0.04 to 4.96 ± 0.03 at 12^{th} hours, from 7.38 ± 0.03 to 4.85 ± 0.02 at 12^{th} hours and from 7.18 ± 0.07 to 4.29 ± 1.12 at 72 hours of wheat grain induced acidosis in three groups of goats with the dose rate of 80, 100 and 120 gm/kg body weight respectively.

Huber *et al.* (1984) reported that the pH of rumen liquor decreased to 4.15 ± 0.34 in glucose induced acidosis in sheep.

Sen *et al.* (1984) induced acute ruminal acidosis in goats and noted minimal pH of 4.37 ± 0.12 within 12 hours of engorgement with rice.

Sinha *et al.* (1985) reported fall in ruminal pH from 6.83 to 4.45 and in blood from 7.41 to 7.15 at 36 hours of experimental acidosis in buffalo calves.

Vihan and Rai (1985) reported that pH of rumen liquor varied between 7.2 to 4.5 in clinical cases of rumen acidosis in sheep and goats.

Cao *et al.* (1987) reported that rumen pH changed from 7.35 ± 0.302 to 4.54 ± 0.119 at 24^{th} hours of sucrose induced acidosis in goats at the dose rate of 16g/kg body weight.

Pradhan *et al.* (1988) reported that the pH of rumen liquor varied from 6.4 to 7.4 with an average of 6.8 ± 0.15 in healthy Black Bengal goats.

Randhawa *et al.* (1988) observed fall in rumen pH from 6.78 to 4.78 at 24 hours in subacute from in buffalo calves.

Lal *et al.* (1989) reported significant drop in rumen pH at 12 hours (4.7 ± 0.045) with a further drop at 24 hours (4.54 ± 0.023) after experimentally induced ruminal acidosis in goats by feeding whole wheat grains @ 100gm/kg body weight.

Patra *et al.* (1993) observed that the decrease in rumen pH was associated with decreased pH in blood, CSF and urine and increased total lactic acid conc. in rumen liquor, blood, CSF, and urine.

Angelov *et al.* (1995a) observed the lowest pH of rumen liquor 3.68 at 12 hours after induction of acute rumen acidosis in goats.

Angelov *et al.* (1995b) reported that the rumen pH decreased rapidly and fell below normal limits in the period between 2 hours to 4 hours after the administration of molasses.

Angelov *et al.* (1996) noted that the rumen pH decreased rapidly after the molasses administration in half-marino sheep. The mean values recorded at 12 hours after the treatment was (3.24 ± 0.20).

Patra *et al.* (1996) induced acidosis in sheep by feeding soaked wheat @ 90g/kg body weight and recorded decreased pH of rumen liquor, blood, and urine within 12 hours.

Gokce and Imren (1998) observed marked decrease in rumen pH at 4 hours after feeding crushed wheat straw in sheep.

Nikolov (1998a) determined the rumen pH (6.1 ± 0.1) at 4 hours and (5.0 ± 0.3) at 8 hours after induction of acute ruminal acidosis in buffalo-calves. They returned to initial value at 72 hours.

Nikolov (1998b) noted the normal pH or rumen content (7.1 ± 0.2) prior to experiment. However, pH of rumen liquor decreased significantly as early as post-treatment at 4 hours; reaching 4.8 ± 0.3 and 5.0 ± 0.2 respectively between 8 and 24 hours.

Shukla *et al.* (1999) reported that following feeding of wheat flour, there was a significant reduction in pH of rumen liquor after 36 hours in bovine calves.

Dwivedi *et al.* (2000) observed that after induction of ruminal dysfuction, the pH of rumen liquor decreased and the mean value recorded was 5.75 ± 0.281 at 12 hours of post induction.

Chand *et al.* (2001) observed that the lowest rumen pH was 4.75 ± 0.19 at 36 hours post induction of ruminal acidosis by administering crushed wheat grains @ 50gm/kg body weight orally.

Hawa Singh *et al.* (2001) recorded a slightly low pH of rumen liquor (6.59 ± .0.08) in simple digestion cases as compared to healthy goats (6.83 ± 0.09).

2.4.2. Total volatile fatty acid :

Stone (1949) found the ingesta from inactive bovine rumen to produce less gas and volatile acid than the normal rumen.

Phillipson (1952) reported low VFA production in rumen of lamb fed on flaked maize ration leading to acidosis.

Scarisbrick (1954) reported low level of VFA production which maintained almost positive correlation with decreasing value of rumen pH in sheep.

Reid *et al.* (1957) studied the effect of high starch diet viz. wheatish starch, oats and cracked maize on VFA content in the rumen of sheep and

noticed that there was an increased level of propionic acid in animals kept on different kinds of diet. However, the level of butyric acid changed with variation in diet.

Eadie *et al.* (1970) fed a barley ration *adlib* to some heifers, and found decreased level of TVFA from 125 to 197 mM/L in one heifer.

Joshi and Ludri (1970) recorded the normal level of TVFA concentration between 64 to 83 mEq/L and in a clinical case of acid indigestion, a level of 145mEq/L was recorded in buffaloes.

Reddy and Nair (1971) estimated that the mean TVFA content in rumen liquor of Jamunapari-Malabari crossbred goats at 2^{nd}, 4^{th}, 6^{th} and 8^{th} hours of feeding to 106.60 ± 1.92, 127.95 ± 1.17, 114.55 ± 1.52 and 96.40 ± 1.75 mEq/L respectively.

Prasad *et al.* (1972) found high level of TVFA (136.03 ± 16.50 mEq/L in buffaloes and 149.20 ± 13.05 mEq/L in cattle) in clinical cases of acid indigestion.

Rai *et al.* (1972) estimated the mean TVFA value in rumen liquor of Barbari goats on maintenance ration and observed as 72.08 ± 1.89 mEq/L.

Chaplin and Jones (1973) reported that the concentration on of TVFA in rumen liquor decreased to 20 mEq/L after 24 hours of ground barley overload in sheep.

Prasad *et al.* (1973) observed that the TVFA concentrations increased up to 2 hours and then declined. This was probably due to change in the microbial population brought about by the low pH.

Svendsen (1973) observed marked inhibition of ovine ruminal motility when the concentration of undissociated VFA exceeded 5mM/L. Butyric

acid was found to be the most potent inhibitor of the 3 individual VFA, but the lactic acid showed no inhibitory effect on motility.

Joshi and Mishra (1975) estimated the TVFA concentration in clinical cases of acid indigestion and found 141.6 ± 9.47 mEq/L and 139.0 ± 12.44 mEq/L in Zebu cattle & buffaloes, respectively.

Verma *et al.* (1975) estimated the mean TVFA values in rumen liquor of Barbari bucks at '0' hour, 2nd, 4th and 6th hours of concentrate mixture feeding which were 67.84 ± 4.66, 90.89 ± 7.44, 62.72 ± 5.00 and 81.70 ± 6.47 mEq/L respectively.

Wilson *et al.* (1975) found that TVFA concentration increased from 67 mM/L pre-engorgement to 105 mM/L at 12 hours and then decreased to 15 mM/L at 36 hours.

Mullen (1976) stated that the decrease in ruminal pH during the first 8 hours of over feeding was not caused by increase in lactic acid concentration, but by the increased proportions of VFA.

Prasad (1977) found a negative correlation between ruminal pH and TVFA in clinical cases of ruminal acidosis in both cattle and buffaloes.

Sethuramen and Rathor (1979c) noted inverse relationship between ruminal pH and VFA level in both cattle and buffalo suffering from acute acid indigestion.

Sethuraman and Rathor (1979c) observed that the mean value of TVFA in rumen liquor of control group was 66.73 ± 2.04 mEq/L which gradually increased to 85.3 ± 5.6, 128.2 ± 5.2, 159.3 ± 6.2 and 162.4 ± 6.2 mEq/L at 24 hours, 48 hours, 72 hours and 96 hours respectively in experimental rumen acidosis in calves.

Muir *et al.* (1980) reported that, there was an increase in concentration of TVFA in initial stage followed by gradual decrease in experimental rumen acidosis in sheep.

Rai and Pandey (1980a) observed that mean values of TVFA of rumen liquor were 76.060 ± 3.228 mEq/L, in range group, 98.246 ± 2.829 mEq/L in range supplemented with concentrate group and 96.180 ± 2.698 mEq/L in concentrate mixture in stall-fed group of adult goats.

Nagaraja *et al.* (1982) found decreased concentration of TVFA in rumen fluid in rumen acidosis.

Sen (1982) noted the mean value of TVFA in rumen liquor changed from 50.0 ± 9.49 mEq/L at '0' hour to 30.00 ± 1.79 mEq/L at 4^{th} hours of grain engorgement in goats. There after in subsequent hours the TVFA decreased to a minimum of 7.00 ± 2.11 mEq/L by 32 hours of grain overload.

Sinha *et al.* (1983) found that ruminal TVFA increased from 62.3 ± 7.34 mEq/L to a maximum of 119.66 ± 10.3 mEq/L at 12 hours and then decreased to 22.5 ± 3.53 mEq/L by 48 hours.

Tanwar and Mathur (1983b) concluded that the increase in the total volatile fatty acid concentration in early stage of rumen acidosis was due to fermentation of carbohydrates by rumen micro-organisms. Later on it decreased due to changed microbial population of rumen due to low pH.

Li *et al.* (1984) drenched maize flour @ 30g/kg body weight with water to sheep and noted that with the onset of acidosis the pH & volatile fatty acids in the rumen declined suddenly.

Sinha *et al.* (1985) reported initial rise in TVFA level at 12 hours and then declined from 36 hours onward in rumen liquor of buffalo calves during ruminal acidosis induced experimentally.

Lal (1988) reported a significant rise in TVFA concentration at 12 hours and followed by decline in the concentration throughout the experiment in acid indigestion in goats.

Pradhan *et al.* (1988) recorded the mean concentration of TVFA in rumen liquor as 78.5 ± 3.86 mEq/L in healthy Black Bengal goats after feeding.

Lal *et al.* (1989) observed that, at 12 hours the mean TVFA level in the rumen liquor increased significantly from 64.83 ± 2.891 to 148.33 ± 15.47 mEq/L which later on decreased to 44.00 ± 2.251 mEq/L at 24 hours and 31.66 ± 1.839 mEq/L at 48 hours of experimental rumen acidosis in goats.

Randhawa *et al.* (1989a) noted that in subacute cases of lactic acidosis the TVFA level gradually increased during 6 to 96 hours while in acute cases there was a sudden fall in TVFA level at 24 hours in buffalo calves.

Ahuja *et al.* (1990) recorded gradual decrease in TVFA value in buffalo calves suffering from subacute lactic acidosis.

Das (1990) observed significantly low concentration of TVFA upto 36 hours and there after it gradually increased in experimental rumen acidosis in goats.

Patra (1991) showed an increase in TVFA concentration at 12 hours followed by decrease in the value in acute ruminal acidosis in sheep.

Krehbeel *et al.* (1995) recorded decreased TVFA following intra-ruminal administration of glucose at different dose levels.

Patra *et al.* (1996) recorded the total VFA concentration in strained rumen liquor, increased between '0' hours (44.2 mmol/L) and 12 hours (83.0 mmol/L), but decreased there after.

Dwivedi *et al.* (2000) reported that there was an initial increase in TVFA concentration after induction of acidosis in calves. The mean values recorded at 12 hours post-induction was (5.53 ± 0.27 mEq/L).

Chand *et al.* (2001) reported initial increase in TVFA concentration (122.40 ± 6.59) upto 12 hours which decreased in later period of experiment.

Gurumoorthy *et al.* (2002) recorded, a significant increase in concentration of TVFA to 120 ± 3.24 mmol/L as compared to healthy animals (102.7 ± 4.36 mmol/L) in case of acid indigestion.

2.4.3. Rumen lactic acid :

Phillipson (1952) observed high lactate production (7.25mM/L) after seven hours of flaked maize feeding in the rumen of Lambs.

Scarisbrick (1954) reported elevated lactic acid (90mM/L) in the rumen of sheep, following ingestion of 15 lb. of mangold.

Broberg (1960) noted high amount of lactate (1000 mg) and pyruvic acid in cases of acute rumen overload in sheep.

Ahrens (1967) estimated increased level of ruminal lactate (17.3 mM/L at 24 hours in wheat fed, 130.5 mM/L at 21.33 hours in pear fed) in cattle in ruminal acidosis.

Eadie *et al.* (1970) reported that lactic acid concentration remained below 5 mg% when cattle were offered barley *adlibitum*.

Chaplin and Jones (1973) observed that in barley induced rumen acidosis in sheep, rumen lactate increased from 1.0 mM/L to 100 mM/L from '0' hour to 18 hours respectively.

Vihan *et al.* (1973b) found the lactic acid concentration of 21.97 ± 10.3 mg% in Zebu cattle and 10.53 ± 4.17 mg% in buffaloes suffering from acute indigestion.

Cakala *et al.* (1974) found appearance of lactic acid in the rumen as earlier sign of acidosis in sheep.

Shinosaki and Nakabayashi (1974) recorded the highest concentration of lactic acid in rumen liquor as 115 mM/L at 12 hours in glucose induced acidosis in sheep.

Verma *et al.* (1975) estimated the mean lactic acid concentration of rumen liquor in Barbari bucks at '0' hour, 2nd hours, 4th hours and 6th hours of concentrate mixture feeding as 3.80 ± 0.40, 19.62 ± 1.10, 3.50 ± 0.30 and 3.90 ± 0.10 mg/100 ml respectively.

Wilson *et al.* (1975) found no lactic acid prior to engorgement while increased lactate to 6.2 mg/ml at 24 hours and 10.7 mg/ml at 48 hours post engorgement were noted.

Koer *et al.* (1976) noted one peak concentration of ruminal lactate at 4 to 6 hours of post engorgement and the second at 60 hours.

Elam (1976) reported high level of lactic acid in rumen and blood in feedlot cattle suffering from ruminal acidosis.

Mullen (1976) stated that in protracted cases of ruminal acidosis, lactic acid concentration reached to a peak at 24 hours after overload and declined thereafter. If not treated after 24 hours of rumen acidosis, the lactic acid concentration declined and there was reversion to volatile fatty acid fermentation.

Beede and Farlin (1977) found one peak level of ruminal lactate at 3 hours and the another at 7 hours.

Sethuraman and Rathor (1979a) induced acute indigestion in cattle and buffalo and noted that the rumen lactic acid levels increased from 1.99 mg% in control animals to 24.4 mg% at 24 hours then to 36.3 mg% at 36 hours and finally 60.8 mg% at 96 hours of post engorgement.

Muir *et al.* (1980) noted that the peak concentration of rumen lactate was 130 M/ml by 12 hours and decreased to normal level of less than 1 M/ml by 30 hours of experimental acidosis in lambs.

Randhawa *et al.* (1981b) induced acidosis in 12 buffalo calves out of which 4 calves died and the mean lactic acid concentration before death, was 122 mg%.

Nagaraja *et al.* (1982) produced acidosis in cattle by intraruminal administration of glucose and reported that total lactate increased from 17.0 mg% to 46.6 mg% in 12 hours.

Sen *et al.* (1982) reported higher lactic acid in rumen liquor and blood of goats at 4 hours after induction of ruminal acidosis.

Bide (1983) collected a large number of rumen fluid samples during a course of feeding trial from 15 hay and 15 grain fed cattle, and found that the rumen fluid of grain fed animals contained significantly higher ($P<0.01$) levels of lactate (17.6 ± 2.9 mg% against 8.5 ± 8.0 mg%) than those of hay fed animals.

Tanwar and Mathur (1983b) recorded the peak concentration of lactic acid in rumen liquor, 748.21 ± 22.86 mg% at 72^{th} hours in wheat grain induced acidotic goat.

Huber *et al.* (1984) observed higher concentration of lactic acid in rumen liquor as 1048.0 ± 368 mg/100ml in glucose induced acidosis in sheep.

Sen *et al.* (1984) induced ruminal acidosis in goats and found maximum value of lactic acid (521.59 ± 39.5mg%) at 12 hours.

Sinha *et al.* (1985) found that the mean level of lactic acid in rumen fluid in buffalo calves rose from 2.05 ± 1.29 mg% before engorgement with crushed wheat to a maximum of 128.0 ± 16.7 mg% at 36 hours post-engorgement.

Vihan and Rai (1985) noted that the mean lactic acid concentrations of rumen liquor to 49.9 ± 3.83 mg% in clinical cases of acidosis in goats.

Lal *et al.* (1989) found that mean concentration of lactic acid in rumen liquor rose from 4.08 ± 0.313 mg/dl at '0' hour to 295.13 ± 16.826 mg/dl at 12 hours and then gradually declined in experimental ruminal acidosis in goats.

Randhawa *et al.* (1989b) noted increased lactic acid concentration in rumen liquor of buffalo calves in subacute cases.

Ahuja *et al.* (1990) observed an increased lactic acid concentration (13.55 mM/L) at 12 hours in subacute cases in buffalo calves.

Patra (1991) reported a higher lactic acid content in rumen liquor of sheep suffering from acute acid indigestion.

Lal *et al.* (1992) reported that the level of lactic acid in rumen increased significantly at 12 hours and then declined at 24 hours, but remained many fold higher than the normal values even upto 96 hours.

Lal *et al.* (1993) reported an increased rumen lactic acid concentration in acute ruminal lactic acidotic goats.

Patra *et al.* (1993) found an increased lactic acid concentration in rumen liquor following induction of ruminal acidosis in sheep by oral feeding of wheat grain at 90 gm/kg body weight.

Shukla *et al.* (1999) reported increased level of lactic acid in rumen liquor at 36 hour after feeding of wheat flour and it remained high upto 12 hours post therapy.

Dwivedi *et al.* (2000) found increased lactic acid concentration 163.2 ± 15.61 mg/dl at 12 hours & 206.7 ± 17.14 mg/dl at 36 hours after induction of rumen dysfunction in calves.

Chand *et al.* (2001) noted increased rumen lactic acid concentration (131.88 ± 5.82 mg/dl) at 36 hours of post-induction of rumen lactic acid and remained higher till the termination of experiments.

2.5. Biochemical changes in blood and serum :

2.5.1. Blood urea nitrogen :

Dirksen (1967) reported normal blood urea level in peracute cases of experimental ruminal acidosis in cattle but the level was found to be elevated in protracted cases.

Rai *et al.* (1972) recorded the average value of blood urea nitrogen in Barbari goats to 18.96 ± 0.69 mg/dl, maintained on 300 gm concentrate mixture (gram, ground nut cake, barley and bran 25 parts) and on grazing.

Coles (1974) reported that dehydration, haemoconcentration, anuria, & catabolism with body toxaemia, raised the blood urea nitrogen.

Mullen (1976) observed increased concentrations of blood urea in carbohydrate overfeeding in cattle and this led to the metabolic disturbance.

Jagos *et al.* (1977) observed increased concentration of plasma urea in chronic metabolic acidosis in dairy cows.

Melvin (1977) reported that the normal concentration of blood urea nitrogen of goat were 13-28 mg/dl.

Nauriyal and Baxi (1978) observed a significant rise of blood urea nitrogen in experimentally induced ruminal lactic acidosis in cattle and buffaloes.

Sethuraman and Rathor (1979c) observed an increase in mean blood urea nitrogen from 21.4 ± 2.1 mg% at '0' hour to 65.1 ± 2.4 mg% at 96 hours in cattle and from 21.4 ± 3.4 mg% at '0' hour to 62.4 ± 2.2 mg% at 96 hours in ruminal acidosis of buffaloes.

Anderson (1980) reported prerenal azotemia with the increase in BUN upto 150 mg/dl in acid indigestion of ruminent.

Nauriyal and Baxi (1981) observed, raised level of blood urea nitrogen in cross-bred cattle and buffalo following molasses and grain induced ruminal acidosis.

Randhawa *et al.* (1981a) reported increase in blood urea nitrogen in peracute lactic acidosis in crossbred calves.

Sastry (1983) reported that blood urea nitrogen level in normal goat were 13-28 mg/100ml of blood.

Sandha and Choudhary (1985) noted higher blood urea nitrogen level from 18 hours in buffalo calves following induced ruminal acidosis. The level returned to normal at 96 hours.

Cao *et al.* (1987) observed that mean plasma urea concentrations remained within normal ranges upto 48 hours of study in experimentally induced lactic acidosis in goat.

Singh *et al.* (1989) reported that normal mean blood urea concentration in buffaloes varied between 24.40 ± 0.97 and 40.30 ± 2.23 mg/dl in different seasons while in indigestion cases, the values always remained beyond 41 mg/dl.

Smith (1990) reported that, decreased renal function is reflected by elevated blood urea nitrogen concentration in ruminal acidosis.

Lal *et al.* (1992) reported an increase in blood urea nitrogen level which remained significant upto 24 hours and thereafter almost constant value was observed.

Patra *et al.* (1996) recorded higher concentration of serum urea, 14.0 mmol/L at 48 hours than all other times in experimental acidosis in sheep.

Nikolov (1998b) reported that the amount of urea nitrogen in buffaloes before the experiment was normal (6.3 ± 0.2 mmol/L) and after the treatment of experimental animals with 20 ml/kg body wt. of molasses the values increased reaching to 12.1 ± 0.8 mmol/L at about 24 hours.

2.5.2. Blood lactic acid :

Dunlop and Hammond (1965) reported abnormal amount of lactate accumulation in blood in lactic acidosis in ruminents.

Juhas'z and Szegedi (1968a) observed an increase in blood lactate concentration in sheep suffering from ruminal acidosis.

Walker (1968) noted that blood lactate values rose to above 200mg/100ml after sheep were forcibly engorged with wheat.

Huber (1969) noted that in ruminal lactic acidosis of sheep, the blood lactic acid level increased to 17.7 mM/L or greater.

Vihan *et al.* (1973a) estimated the lactic acid concentration in the serum of cattle and buffaloes suffering from acid indigestion, and found 24.3 ± 6.88mg% in Zebu cattle and 6.8 ± 0.93 mg% in buffaloes.

Shinosaki and Nakabayashi (1974) induced ruminal acidosis in sheep and recorded increased lactic acid concentration in rumen (115mM/L) and blood (4.1 mEq/L).

Vestweber *et al.* (1974) observed that the mean concentration of blood lactic acid was inversely proportional to the rumen pH. The mean concentration of blood lactate were 1.84 ± 0.95 mM/L and 3.02 ± 1.77 mM/L at rumen pH of 6 or above and 5.99 or less respectively in maize sugar induced acidosis in sheep.

Dougherty *et al.* (1975) observed that blood lactic acid concentration increased from 4 to 79.5 mg/100ml in grain overload sheep.

Verma *et al.* (1975) estimated the lactic acid concentration in blood of Barbari buks at '0' hour, 2^{nd} hours, 4^{th} hours and 6^{th} hours of concentrate mixture feeding, which were 9.53 ± 0.45, 28.51 ± 0.90, 8.35 ± 0.30 and 8.55 ± 0.25 mg/100ml respectively.

Elam (1976) reported higher level of lactic acid in rumen and blood of feedlot cattle suffering from ruminal acidosis.

Stangassinger .and Giesecke (1978) reported that when blood lactic acid level exceeded 1.9 mmol/L in goat and 4.25 mmol/L in sheep and cow, the excretion rate increased greatly. Above this threshold concentration the maximum backtransport rate of the kidney is exceeded, and lactic acid excretion became proportional to the glomerular filtration rate.

Sethuraman and Rathor (1979c) induced acute indigestion in bovine and found that blood lactate values increased from 14.5 ± 2.2 mg% and 18.6 ± 2.16 mg% in cattle and buffaloes respectively at '0' hour, through 42.4 ± 3.5 mg% and 42.2 ± 2.0 mg% at 48 hours to 61.1 ± 1.7 mg% and 60.2 ± 2.7 mg% at 96 hours.

Nauriyal and Baxi (1981) induced lactic acidosis in cross-bred cattle and buffaloes and recorded high ruminal and blood lactate level.

Randhawa *et al.* (1981b) produced ruminal acidosis in 12 buffalo calves, out of which 2 animals died after 60 hours and another 2 after 96 hours. The mean lactic acid concentration prior to death was 86 mg/100 ml of blood.

Sen *et al.* (1982) recorded increased lactic acid level in rumen liquor and blood of goats at 4 hours after induction of acidosis.

Tanwar *et al.* (1983) found increased mean blood lactic acid concentration from 9.45 ± 0.54 mg% pre-engorgement to 27.30 ± 1.32 mg% at 12 hours and further increased to 40.57 ± 5.20 mg% at 72 hours post-engorgement.

Sen *et al.* (1984) produced acute ruminal acidosis in goats by feeding rice and found that the highest lactic acid concentration (41.37 ± 2.42 mg%) occurred at 32 hours.

Sandha and Chaudhary (1985) noticed elevated blood lactic acid level at 12 hours of induced acidosis in buffalo calves. The value remained elevated throughout the acidotic phase.

Sinha *et al.* (1985) induced experimental acidosis in buffalo calves and observed that the mean concentration of lactic acid increased from 17.38 ± 1.93 mg% pre-engorgment to a maximum of 60.00 + 8.94 mg% at 36 hours post- engorgement.

Tanwar and Mathur (1985) found a marked rise in the rumen and blood lactic acid level in goats following experimental acidosis.

Vihan and Rai (1985) recorded that the lactic acid level of blood in acidotic goats increased to 30.8 ± 2.30 mg%.

Cao *et al.* (1987) noted that the mean concentration of blood lactate was increased from 1.78 ± 0.737 mmol/L to 2.72 ± 0.363 mmol/L at 12

hours, then declined and again elevated to 2.79 ± 1.223 mmol/L at 48 hours of experimentally induced lactic acidosis in goats.

Randhawa *et al.* (1988) observed increased blood lactic acid concentration in subacute cases of lactic acidosis in buffalo calves.

Lal *et al.* (1989) noted rise in blood lactic acid level in experimentally induced acidosis in goats.

Das (1990) observed a significant increase in blood lactic acid in experimental rumen acidosis in goats. Highest blood lactic acid concentration of 38.06 ± 4.11 mg/100ml was observed at 36^{th} hours.

Patra (1991) found increased blood and ruminal lactic acid levels in acidotic sheep.

Lal *et al.* (1992) observed that the lactic acid level in blood significantly rose from 12 to 48 hours & decrease towards normalcy by 72 hours.

Lal *et al.* (1993) observed decreased blood pH and an increase in blood lactic acid concentration in acidotic goats.

Patra *et al.* (1993) noted an increased lactic acid concentration in blood after 12 hours of oral feeding of wheat grain in sheep.

Sen *et al.* (1993) reported, increased lactic acid 21.3 + 4.5 mg% from normal of 13.0 ± 2.9 mg% by 8 hours of induction and the peak blood lactic acid concentration (41.4 ± 12.4 mg%) was recorded at 32 hour of acidosis.

Angelov *et al.* (1995a) noted increased blood lactate 5.2 mmol/L at 12 hours following administration of 20 ml/kg of body weight beet molasses in goats.

Angelov *et al.* (1996) recorded the mean values of blood lactate 8.1 ± 0.9 mmol/L at 12 hours after treatment in experimental acute rumen acidosis in sheep.

Patra *et al.* (1996) found an increased blood lactic acid concentration at 12 hours after feeding of soaked wheat in sheep.

Shukla *et al.* (1999) reported increased blood lactic acid level in calves after 36 hours of feeding of wheat flour.

Dwivedi *et al.* (2000) estimated increased concentration of blood lactic acid following grain feeding. It showed a rising trend till 36 hours of the observation.

2.5.3. Blood bicarbonate :

Shinosaki and Nakabayashi (1974) produced a slight acidosis in sheep by intraruminal administration of glucose @ 6.75 gm/kg body wt. The effect on rumen fluid indicated a fall in rumen pH to 4.64, increase in concentration of lactic acid to 115 mM/L, increase in concentration of calcium and magnesium ions and a decrease in sodium and carbonate concentrations. The plasma changes included, a fall in pH to 7.3, an increase in lactic acid concentration to 4.1 mEq/L, an increase in concentration of sodium and phosphate ions and decrease in concentration of potassium, chloride and carbonate ions.

Dougherty *et al.* (1975) engorged grains mixture (75% shelled corn and 25% oats) @ 70 gm/kg body weight and found a marked changes in rumen pH (from 7.2 to 4.2), blood pH (from 7.52 to 7.01) and HCO_3 (from 20.1 to 3.6 mmol/L) at 53 hours of experimental induction in sheep.

Heijlasz *et al.* (1984) studied experimental acute metabolic acidosis in cow and observed reduction in blood pH and bicarbonate level after 24 hours of induction.

Li *et al.* (1984) observed that the CO_2 combing power of the plasma and the pH of the urine fell rapidly when maize flour @ 30 gm/kg body wt. with added water was given by drench to sheep.

Sen *et al.* (1993) reported that serum bicarbonate levels decreased significantly from normal 24.6 ± 2.2 mEq/L to 22 ± 1.2 mEq/L after 4 hours of induction of acidosis and the lowest value (9.01 ± 3.13 mEq/L) was observed at 44 hours of grain overload.

Angelov *et al.* (1995a) induced acute ruminal acidosis in goats and noted the lowest value of blood pH (7.19), bicarbonate (14.5 mmol/L), ABE (-16.8 mmol/L) and rumen pH (3.68) after 12 hours of administration of beet molasses.

Angelov *et al.* (1995b) measured the blood pH and bicarbonate level 7.23 and 15.40 mmol/L respectively in buffalo calves at 12 hours, 7.18 and 13.3 mmol/L at 24 hours, 7.19 and 14.5 mmol/L in goats at 24 hours, 7.20 and 12.6 mmol/L in sheep at 12 hours after the administration of beet molasses @ 20 ml kg body wt.

Angelov *et al.* (1996) induced ruminal acidosis in sheep and observed a decreased values of blood pH, blood bicarbonate, ABE and rumen fluid pH rapidly. After 12 hours of molasses administration, the mean value recorded were, blood pH 7.201 ± 0.028, blood bicarbonate 12.6 ± 1.8 mmol/L, blood ABE - 16.3 ± 1.8 mmol/L and rumen pH 3.24 ± 0.20.

Shukla *et al.* (1999) reported that the bicarbonate level in blood (19.45 mmol/L) showed a significant (1%) fall at 36 hours post induction of lactic acidosis and remained low throughout the study period in calves.

2.5.4. Serum transaminase activity (ALT / AST) :

Jagos *et al.* (1973) reported fall in dehydrogenase activity of ruminal fluid in cattle during acidosis which was parallel with fall in pH.

Mullen (1973) noted an increased activities of enzymes viz. aspartate amino transferase, alanine amino transferase , alkaline phosphatase and glutamate dehydrogenase in calves fed on barley.

Vihan *et al.* (1973a) recorded a moderate rise in alanine amino transferase activity in cattle and buffalo while, aspartate amino transferase activity remained constant in clinical cases of acid indigestion.

Cakala *et al.* (1974) recorded higher AST value in the various forms of ruminal acidosis in sheep following sucrose administration.

Slyter (1976) observed the influence of acidosis on rumen function in ruminants and suggested that the amylase activity was reciprocal to pH, in the range of 6.5-5.0 while the activity decreased below pH 5.

Vihan (1978) recorded a significant positive correlation between the serum alkaline phosphatase activity and ruminal pH in buffalo, while non significant change in lactic dehydrogenase activity was noted.

Sethuraman and Rathor (1979b) recorded increased levels of transaminases (SGPT and SGOT) and icterus index in both the cases of experimentally produced acute ruminal acidosis and alkalosis in bovines

Nauriyal and Baxi (1981) reported a significant rise in the SGOT and SGPT activity in cross-bred cattle and buffalo, while no change was observed in alkaline phosphatase activity in rumen acidosis.

Bieniek (1982) recorded increased serum aspartate aminotransferase activity in cattle following ruminal acidosis.

Randhawa *et al.* (1988) noted, rise in ALT, AST, arginase and GDH activities in subacute lactic acidosis in buffalo calves.

Lal *et al.* (1989) reported a significant rise in AST, GGTP, LDH, amylase and CPK values in serum of acidotic goats, following intraruminal administration of wheat.

Lal *et al.* (1991) found significantly higher activities of amylase (at 12 hours), lactate dehydrogenase (12 to 48 hours), creatine phosphokinase (12 to 48 hours), aspartate aminotransferase (12 to 24 hours) and gamma-glutamyl transferase (12 to 96 hours) in serum samples of goats with acidosis.

Das and Misra (1992) reported that GOT and GPT increased markedly from 24 hours onward till death or recovery and highest value (60.36 ± 5.20 µg of pyruvic acid/ml) was recorded at 72 hours following engorgement with crushed rice @ 80 g/kg body wt. after 36 hours of fasting in goats.

Lal *et al.* (1992) reported increased AST, and GGTP activity in acidotic goats to 117.33 ± 8.143 RFU/ml and 59.00 ± 1.238 µg/L at 12 hours after the induction.

Gaikward *et al.* (1993) reported, increased level of SGOT to 59.56 ± 1.19 from normal level of 30.05 ± 0.69 in buffaloes suffering from anorexia.

Patra *et al.* (1996) recorded the maximum serum creatinine phosphokinase (54.0 IU/L), GGTP (68.3 IU/L), AST (60.8 IU/L) and serum amylase (159.5 IU/L) activities at 96, 24, and 24 hours respectively in acidotic sheep.

Nikolov (1998b) recorded the concentration of pyruvate (95.5 to 104.5 mmol/L) before the experiment, which increased maximum to (162.3 ± 8.4 mmol/L) at 24 hours of development of acute rumen acidosis in buffalo.

Dwivedi (2000) reported that there was non-significant increase in AST level in acidotic calves where as the activity of ALT remained almost unaltered.

Hawa Singh *et al.* (2001) reported significant increase in ALT and AST level from 16.83 ± 2.15 and 56.75 ± 4.56 of healthy goats to 21.95 ± 1.04 and 66.54 ± 2.72 in simple in digestion cases.

2.5.6. Total serum protein :

Gorczyca *et al.* (1960) reported that mean normal value of total serum protein in goat (both male and female) aged 7-9 months was 6.25 ± 0.35 gm/dl.

Altman and Dittmer (1961) reported that normal levels of total serum protein in goat to be 6.67 gm/100ml, but total plasma protein was 7.27 gm/100ml.

Juhas'z and Szegedi (1968b) reported that plasma protein value increased in acid indigestion of sheep produced by ground wheat and barley.

Jonson and Liberg (1974) found decrease in the level of total protein in intensively fed calves.

Mullen (1976) reported that the concentration of total plasma protein varied with the degree of dehydration present in carbohydrate overfeeding in cattle.

Nauriyal and Baxi (1978) observed that total plasma proteins showed no change in experimentally induced ruminal lactic acidosis of cross bred cattle and buffaloes.

Kessabi and Lamnaquer (1981) found increase in total protein and globulin levels in liver disorders when albumin was lower.

Vihan *et al.* (1982) observed that total serum protein, albumin and globulin levels were within the normal range in experimental rumen acidosis in goats.

Sastry (1983) reported that in healthy goat plasma protein level was 6.25 gm/100ml.

Cao *et al.* (1987) recorded the mean total plasma protein level in experimental lactic acidosis in goat which increased from 80.4 ± 5.34 gm/litre at '0' hour to 88.0 ± 3.37 gm/litre at 24 hours and then again declined to 81.4 ± 6.65 gm/litre at 48 hours.

Randhawa *et al.* (1988) noted non-significant decrease in total protein in subacute lactic acidosis in buffalo calves.

Smith (1990) reported that normal values of total serum protein in goat had ranged from 2.7 to 3.9 gm/dl.

Patra *et al.* (1996) observed significantly higher level of total protein (78.8 gm/L) at 24 hours than at all other times in acidotic sheep.

2.6. Therapeutic management :

Hungate (1966) concluded that heavy dosing with rumen liquor obtained from normal animals was usefull in recovery of acute acid indigestion in ruminants.

Hoflund (1967) suggested the use of fresh rumen juice to correct the ruminal environment in acidosis.

Dirksen (1970) recommended the treatment like discontinuance of the feed, oral feeding of alkalizers, antibiotics, baker's or brewer's yeast at early stage of indigestion. In severe cases, he advocated the parenteral

administration of antihistamics, thiamine, physiological saline & calcium and magnesium gluconate was given. For restoration of normal flora and fauna, rumen cud tramplant was adviced.

Gnanaprakasam (1970) treated clinical cases of acidosis in goats by clearing toxic ruminal contents and transplanting fresh rumen cud from healthy slaughtered goats along with antacid, antihistamincs, antibiotics, thiamine fluid therapy with electrolytes.

Dunlop (1972) found that the addition of ingesta from healthy animal, as useful material in restoration of normal fermentation in acidotic ruminants.

Prasad *et al.* (1973) used dessicated rumen liquor with mineral and ruminotoric drugs, for establishing rumen microbial activity in acidotic buffaloes.

Flachowsky *et al.* (1974) stated that the intramuscular administration of 300 mg of thiamine (10 mg/kg body weight) not later than 2 hrs before infusion of 200 gm mixture of starch, glucose and casein in to the rumen of lambs, prevented the increase in heart rate, despite the development of sever acidosis.

Prasad and Rekib (1975) did not find any satisfactory result in severe cases of acidosis (rumen pH between 4.6 to 4.5) in lambs when treated with 7.5% sodium bicarbonate intravenously and sodium bicarbonate along with rumen cud orally. But sheep, given same line of treatment with nuxvom. orally was cured.

Sethuraman (1976) treated acidotic buffalo and calves by oral therapy with antibiotics, antacids, ruminal cud transplantation, parentral therapy with

5% sodium bicarbonate, Ringer's sodium lactate, vallergan, thiamine and liver extract.

Beede and Farlin (1977) reported that capreomycin disulphate reduced lactate concentration by about 69% suggesting enhanced utulization of lactate yielding propionate.

Muir *et al.* (1980) used thiopeptin to prevent lactic acidosis in ovine.

Sandha (1980) treated experimental ruminal acidosis in buffalo calves with intraruminal administration of Gelusil tablet, Liv-52 powder, 5% sodium bicarbonate (I/v), Berin (I/m), Insulin (I/m) and Vetycillin (I/m). Cud transplantation from healthy animals was also recommended.

Mahadeven (1982) studied the keeping quality of bovine rumen fluid at room and refrigeration tamperature without adding any preservative and he observed that at room temperature, fluid was suitable for transfusion for 1.80 days and at refrigeration temperature for 4.80 days.

Sen (1982) treated experimentally induced ruminal acidosis in goats by evacuating rumen content through fistula, intraruminal administration of tetracycline hydrochloride, aluminium hydroxide, fresh rumen cud, antihistaminics, rumenton tablets & intravenous administration of sodium bicarbonate, sodium chloride (0.9%), thiamine hydrochloride and calborol.

Amstel (1983) compared and found that magnesium oxide was very potent (with the potential danger of causing severe rumen alkalosis) than magnesium hydroxide, magnesium trisilicate, calcium carbonate, aluminium hydroxide and sodium bicarbonate in alkalinizing ability in treatment of clinical cases of rumen acidosis, whereas calcium hydroxide and magnesium carbonate showed slow and unsatisfactory results.

Tanwar and Mathur (1983a) treated experimentally induced acid indigestion in goats by using magnesium carbonate and Benzyl penicillin intraruminally, Berin, Avil and Belamyl intramuscularly, Ringer's lactate solution intravenously and fresh ruminal cud intraruminally.

Tanwar and Mathur (1983b) reported that magnesium carbonate (1 gm/kg), Benzyl penicillin (50, 000IU/kg b.wt.), fresh rumen content from healthy goats and intravenous infusion of electrolytes were effective in management of experimentally induced acidosis in goats.

Karunanidhi *et al.* (1985) reported that the use of fresh rumen liquor preserved at 34^0C for a period of 8 hours was effective in the management of bovine ruminal disorders.

Howard (1986) treated acidosis in ruminents, by emptying rumen contents oral administration of antacids such as magnesium carbonate or magnesium hydroxide, intravenous administration of balanced electrolytes & 5% sodium bicarbonate solution along with antihistaminic I/m.

Cao *et al.* (1987) treated, experimentally induced lactic acidosis in goats by administering calcium hydroxide through stomach tube for precipitating lactic acid in the form of insoluble calcium lactate in the rumen & by administering bicarbonate (intravenously) to counteract the metabolic acidosis.

Ahuja *et al.* (1990) treated subacute acidotic buffalo calves with monensin @ 3mg/kg body weight. It was found to be effective in improving rumen liquor pH and significantly decreased the lactic acid content.

Tripathy and Mishra (1992) treated induced acid indigestion in goats with Rumbion herbal remedy (0.5 gm tablet, twice daily) which brought about recovery within 86 hours while untreated goats took 168 hours to recover.

Appaji Rao (1993) successfully treated the case of anorexia, impection, flatulence, indigestion and dyspepsia in dairy cattle by using rumentonic powder.

Basak (1993) successfully treated a case of moderate ruminal lactic acidosis in a crossbred heifer with 7.5% sodium bicarbonate, Dextrose saline and ruminotorics.

Aslan *et al.* (1995) treated acute ruminal acidosis of goats with 1 gm sodabicarb/kg body weight and 1gm baking yeast/kg or their combination @ 0.5 gm each/kg body weight and reported moderate effect of sodabicarb, where as no effect of yeast preparation was found.

Patra *et al.* (1997) stated that cud transplant was the best therapeutic approach along with parenteral administration of alkalizer, thiamine and anti-histamine.

Gokce and Imren (1998) concluded that the addition of 3,6 and 9% $NaHCO_3$ to carbohydrate rich diets for sheep prevented rumen acidosis.

Misraulia *et al.* (1998) evaluated the efficacy of Appevet bolus @ 4 bolus BID, for the first time in treating a clinical cases of indigestion and anorexia in ruminents (12 cows and 8 she buffaloes) and reported that the indigestion and ruminal atony was cured within 3-5 days after starting the treatment without developing any untoward symptom.

Shukla *et al.* (1999) used Ruchamax and Pachoplus along with fresh rumen liquor in rumen dysfunction found much quicker recovery with these drugs than the standard sodium bicarbonate treatment combined with either fresh or preserved rumen liquor.

Sinha and Mallikarjuna Swamy (1999) concluded that the Yeasacc^{1026} bolus supplementation was highly benificial and a natural way to overcome anorexia associated with ruminal disturbances (like ruminal dysfunction and acidosis) unlike other chemical ruminotorics.

Binding *et al.* (2000) reported that the amount of sodium bicarbonate necessary to correct the existing blood acidosis was determined on the basis of formula : base deficit x body mass x factor = mmol $NaHO_3$. The factor for the correction of the acidosis within 24 hours was 0.65 lt./kg.

Dwivedi *et al.* (2000) concluded that the Gastricare had rumenotoric potential in alleviating mild to moderate ruminal dysfunction and can be used as an adjunct drug in the treatment of rumen indigestion and acidosis.

Gupta *et al.* (2000) suggested that thiamine, insufficiency may occur in various gastrointestinal disorder, therefore, for therapeutic management and for early restoration of disorder, thiamine administration along with specific therapy may help in early recovery.

Kemal Irmak *et al.* (2000b) stated that, thiamine supplementation should be considered as a part of treatment of impaired ruminal motility.

Shu *et al.* (2000) suggested that the risk of lactic acid can be reduced by immunization against *S.bovis*, and that the immunization primed I/m is more effective than the immunization primed I/p.

Malleswar Rao *et al.* (2002) concluded that the average time required for total revival of rumen protozoal activity in untreated defaunated buffalo calves were noticed to be 10.50 ± 0.60 days. Following homologus (buffalo) and heterologus (Ovine) rumen liquor transplantation to defaunated buffalo calves, the normal protozoal activity could be established on an average of 4.16 ± 0.36 days in the former while it took an average of 5.33 ± 0.45 days in the later.

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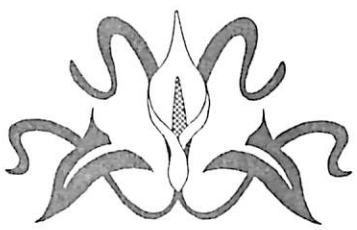
CHAPTER - III



MATERIALS AND METHODS

MATERIALS AND METHODS

The present investigation was conducted in the Department of Veterinary Medicine, Bihar Veterinary College, Patna; under RAU (Pusa), Samastipur, Bihar using the following materials and methods.

3.1. Experimental animals and its preparations :

Twelve apparently healthy, locally available breed (Black Bengal) of goats of either sex, weighing about 10 to 15 kg, between 2 to 3 years of age, were procured from the local animal supplier. These animals were kept under close observation for a week to acclimatize, during which the faeces and blood were examined and treated accordingly to ascertain their normal physiological status. A thorough clinical examinations were done to make sure that they were quite in healthy conditions.

Before start of the experiment, the experimental goats were randomly divided into two groups viz. group A and group B (six animals in each group). All the twelve goats of both the groups were kept under identical managemental condition and were provided with green and dry fodders & fresh water.

3.2 Experimental Design :

The animals were alloted with group colour ribbons (Red ribbon to group A & Green ribbon to group B) to facilitate the study. The animals of both the groups (A and B) were fasted for 24 hours and during fasting only *adlib*. drinking water was provided. After fasting, ruminal acidosis were induced in both the groups of animals at the same time by oral feeding with whole wheat grains @ 100 gm/kg body weight. The animals of group A were served as untreated acidotic control in which clinical signs, & biochemical changes in ruminal fluid, blood and serum were determined,

whereas, in animals of group B, all the above parameters including therapeutic response were observed till recovery or death. The animals were kept under close observation until acidosis started and feed and water were given to them during the entire experimental period.

Following clinical and biochemical aspects were studied in animals belonging to both the groups.

3.3 Clinical aspects (Symptomatology) :-

Pulse rate, respiration rate, temperature and other symptoms like rumination, defaecation and rumen motility were recorded as per the method described by Rosenberger *et al.* (1979). The degree of dehydration were assessed as described by Mir and Shakoor (1999).

3.4 Biochemical aspect :

Test materials :

Rumen fluid, blood and serum were collected from both the groups of animals before fasting (i.e. at '0' hour) and thereafter at 12, 24, 48, 72, 96 and 120 hours following administration of whole wheat grains after 24 hours of fasting, to record changes in various parameters.

3.5 Collection and preservation of bio-materials :

3.5.1 Rumen liquor :

Fifteen ml of rumen liquor was collected with the help of stomach tube fitted with negative pressure pump (method used by Misra and Tripathy, 1963). The fluid, was then strained out through double fold musclin cloth into a test tube. To some amount (5 ml) of strained rumen liquor (SRL), mercuric chloride @ 1mg/5ml of SRL as preservative (Brar *et al.*, 2000) was added and mixed through vigorous shaking and kept in a refrigerator at 4^0C after adding 1 ml of liquid paraffin for the analysis of

3.6. Procedure :

3.6.1. Examination of rumen liquor :

3.6.1.1. Physical examinations :

3.6.1.1.1. Macroscopic Examination :

The colour, consistency and odour of rumen liquor were noted immediately after the collection (as method adopted by Misra and Tripathy, 1963).

3.6.1.1.2. Microscopic Examination :

3.6.1.1.2.1. Motility of rumen protozoa :

Motility of rumen protozoa was determined microscopically as per the method described by Turgut (1995) and the number of motile protozoa in a microscopic area (X100); 20-30, 10-20, 1-10 and no protozoa was graded and represented as 4 (Vigorous), 3 (moderate), 2(slow) and 1 (absent) respectively.

3.6.1.2 Biochemical examination :

3.6.2.1. Rumen pH :

The pH of rumen liquor was recorded with the help of grip pH meter (Systronic GRIpH – pH meter) before adding any preservative. The filteration or aspiration of rumen fluid had no effect on pH (Garrett *et al.*, 1999).

3.6.1.2.2. Total volatile fatty acid :

TVFA in rumen liquor of goats were determined as per the method of Barnett and Reid, 1957.

Briefly, one ml of SRL, one ml of oxalic acid (5%) and one ml of potassium oxalate (10%) was taken and steam distilled in Markham's Microkjeldahl distillation apparatus. About 80 ml of distillate was collected

TVFA. One ml of SRL was taken for the preparation of protein free filtrate (as per the method of Folin and Wu, 1920 for blood) and the filtrate was kept in a refrigerator at 4^0C after adding a drop of xylene (to prevent bacterial decomposition) for the estimation of rumen lactic acid (RLA).

3.5.2. Blood :

Ten ml of blood was collected from jugular vein without exposure to air with the help of clean, dry glass syringe and needle containing little liquid paraffin. The blood was then, transferred into a clean, dry centrifuge tube containing some finely powdered potassium oxalate (1-2 mg/ml of blood as anticoagulent) under a thin layer of liquid paraffin. Out of this 1 ml of blood was pipetted out for the preparation of protein free filtrate (as per the method described by Folin and Wu, 1920). The filtrate was stored in refrigerator at 4^0C after adding a drop of xylene in it, for the estimation of blood lactic acid (BLA) and blood urea nitrogen (BUN). The remaining blood sample was centrifuged at 3000 r.p.m. for 10 minutes to separate the plasma. The supernatant plasma was removed anaerobically and stored in refrigerator at 4^0C after placing it in a tube containing thin layer of liquid paraffin for the analysis of blood bicarbonate (B.Bi).

3.5.3. Serum :

Five ml of blood was drawn in a sterilized serum separating tube, without any anticoagulant content. It was then allowed to clot at room temp. in a slanting position and then, after chilling over night in an up right position in the refrigerator at 4^0C, the serum was then poured off from the side of the tube opposite the slanting clot for the analysis of SGPT, SGOT and total serum protein (TSP).

in a conical flask and titrated against 0.01N NaOH by adding drop by drop from a burette using phenolpthalein (0.1%) as an indicator. As soon as light but stable colour developed, the reaction was stoped and the concentration of TVFA (mEq/L) of SRL was calculated using the formula :

$$\text{mEq of TVFA/L of SRL} = \frac{\text{ml of alkali used X strength of alkali}}{\text{Vol. of SRL taken}} \text{ X } 1000$$

3.6.1.2.3. Rumen lactic acid :

Lactic acid concentration of rumen fluid was estimated according to the method adopted by Barker and Summerson, 1941. Method in brief :

10 ml test tube :	**Test (A)**	**Standard (B)**	**Blank (C)**
Taken, deproteinized sample :	2 ml	--	--
Added, standard lactic acid (0.01mg/ml)	--	5 ml	--
Added, distilled water :	--	--	5 ml
Added, 20% $CuSO_4$ Soln. :	1 ml	1 ml	1 ml
Added, distilled water to each tube to make the vol. upto 10 ml			
Added, calcium hydroxide :	1 gm	1 gm	1 gm

After vigorously shaking untill solids uniformly disperesed, allow to stand them for one half hours repeating the shaking at least once in the intrim.

Centrifuged down the precipitates and transferred 1ml of supernatant to another 3 tubes A, B & C respectively.

Taken, another 10 ml test tube :	A (Test)	B(Standard)	C(Blank)
Transferred, supernatant fluid made above	1 ml	1 ml	1 ml
Added, 4% $CuSO_4$ Soln. :	0.05 ml	0.05 ml	0.05 ml
Added, conc. H_2SO_4 drop by drop from pipette with mixing :	6 ml	6 ml	6 ml

Placed, upright in boiling water for 5 minutes and then cooled to 20^0C or below.			
Added, p-hydroxidiphenyl reagent drop by drop by lateral shaking :	0.1ml	0.1ml	0.1ml
Allowed, to stand them for 30 minutes in water at 30^0C.			
Finally, placed the tubes in vigorous boiling water for 30 sec., removed and cooled to room temp.			
Coloured soln. were transferred to suitable container for the measurement of O.D. at 560 mμ using water for setting the photometer at zero density. The concentration of lactic acid (mg/dl of SRL) were calculated after subtracting blank from standard and unknown, (to obtain their true density) using the formula :			

mg lactic acid/dl of SRL =

$$\frac{\text{O.D. of test}}{\text{O.D. of standard}} \text{ X concentration of standard X } \frac{\text{Vol. of sample}}{\text{Vol. of standard}} \text{ X 100}$$

i.e. in this case, $\frac{\text{O.D. of unknown}}{\text{O.D. of standard}}$ X 0.005 X 50 X 100

= mg lactic acid/dl of SRL. (O.D. is optical density)

3.6.2. Analysis of blood :

3.6.2.1. Blood Urea Nitrogen :

Concentration of urea nitrogen in blood was estimated colorimetrically as per the method described by Netelson, 1957.

Briefly,

Test tube	: Blank (B)	Test (T)	Standard (s)
Distilled water :	1.0 ml	--	--
Deprotenized sample :	--	1.0 ml	--
Standard urea :	--	--	1.0 ml
Diacetyle monoxine :	1.0ml	1.0 ml	1.0 ml
Thiosemicarbazide :	1.0 ml	1.0 ml	1.0 ml
Acid reagent :	3.0 ml	3.0 ml	3.0 ml

All the above reagent was taken accordingly into three respective test tube, mixed and kept in a boiling water bath for 15 minutes. Thereafter, the tubes was cooled down in water to room temp. The O.D. was measured at 540 nm in photoelectric colorimeter. The concentration of urea nitrogen (mg/dl of blood) were calculated using the formula :

$$\text{mg of urea nitrogen/dl of blood} = \frac{\text{O.D. of unknown}}{\text{O.D. of standard}} \times 0.01 \times \frac{100}{0.02} \times 0.467$$

3.6.2.2. Blood Lactic Acid :

Concentration of lactic acid in blood was determined as per the method of Barker and Summerson, 1941 elaborated above for rumen lactic acid and value were expressed in mg/dl blood.

3.6.2.3. Blood Bicarbonate :

Bicarbonate concentration in blood were estimated by titration method described by Van Slyke, 1922. Briefly; described as below :

1. A standard for end point matching was prepared by mixing 20 ml of saline phenol red soln. (pH 7.4) with 1 ml of plasma sample under a thin layer of liquid paraffin in a test tube.

2. Another 1 ml of plasma sample and 5 ml of 0.01 N HCl were taken in a small round bottom flask (without paraffin) and whirled for 1 to 2 minutes to allow the CO_2 to escape out. Further, 10 ml of 0.9% sodium chloride & 7 drops of phenol red soln. (0.03%) were added and titrated with 0.01 N NaOH until the colour matches with the standard. As the end point approached, sufficient amount of 0.9% NaCl was added to bring the volume to 20 ml.
3. A blank was prepared, by taking 5 ml of 0.01N HCl, 10ml 0.9% NaCl and 7 drops of phenol red soln. (0.03%) and titrated with 0.01N NaOH untill the colour matches with standard.
4. The concentration of bicarbonate (mEq/L of blood) was calculated by using the formula given below :

   Reading of blank – Reading of unknown = x

   x = ml of 0.01N or 0.01M $NaHCO_3$ / ml of plasma, or M bicarbonate/dl of plasma.

Therefore, mEq/L plasma bicarbonate = x X 10.

3.6.3. Analysis of serum :

3.6.3.1. SGPT and SGOT :

The activity of transaminases (GPT and GOT) were determined spectrophotometrically by the method of Reitman and Frankel, 1957.

Briefly,

For calibration (Standard Curve)

1. Pyruvate standard, GOT substrate and water were added into 5 tubes as follows :

No. of tubes	ml pyruvate (2.0mmol/L)	ml GOT substrate	ml water	GPT unit/ml	GOTunit/ml
1	0	1.00	0.2	0	0
2	0.10	0.90	0.2	28	24
3	0.20	0.80	0.2	57	61
4	0.30	0.70	0.2	97	114
5	0.40	0.60	0.2	150	190

2. Added, 1 ml DNPH to each tube, mixed and let stand for 20 minutes, then added 10ml of 0.4 N NaOH, mixed by inversion and let stand for further 10 minutes.
3. Sated, the spectrophotometer to zero absorbance with distilled water as blank at 505 nm.
4. Ploted, the reading against the corresponding unit for GOT and GPT on X-axis Vs. O.D. on Y-axis.
5. Connected, the point by a smooth curve.

For Test :

1. Pipetted, 1 ml of GPT or GOT substrate into a test tube & placed in a constant temperature water bath at 37^0C for 10 minutes.
2. Added, 0.2ml serum and mixed.
3. Incubated, exactly 60 minutes for GOT or 30 minutes for GPT.
4. At the end of incubation period, added 1 ml of 2,4-DNPH reagent, removed the tube from water bath and mixed.
5. Let stand at room temp. for 20 minutes.
6. Added, 10 ml of 0.4 N NaOH soln. mixed by inversion and let stand for 10 minutes.

7. Measured, the absorbance using water as blank with a spectrophotometer at 505 nm.
8. Obtained, the units of activity from respective standard curve. Sera with values which exceed the limit of standard curve were diluted with distill water and analysed repeatedly.

3.6.3.2. Total serum protein :

The concentration of total serum protein was measured quantitatively by Biuret method. Briefly,

Test tube :	**Blank (B)**	**Standard (S)**	**Test (T)**
Serum Sample :	--	--	0.1 ml.
Protein standard :	--	3 ml	--
Distilled Water :	3 ml	--	--
0.9% Nacl :	--	--	2.9 ml.
Biuret reagent :	3 ml	3 ml	3 ml

Mixed and allowed to stand at 37^0C for 10 minutes. The O.D. were taken on colorimeter at 540 nm or using green filter against blank. The total protein concentration (gm/dl) was calculated as the formals given below.

$$\text{Total protein in gm/dl} = \frac{\text{O.D. of test}}{\text{O.D. of standard}} \times 6.0 \times \frac{100 \times 1}{0.1 \times 1000}$$

3.7. Preparation of Protein free filtrate by the method of folin and Wu, 1920. Briefly,

1. Transferred 1 ml of blood sample to a flask.
2. Added, 7 ml of water and 1 ml of 10% sodium tungstate soln. and mixed.
3. Added, slowly with shaking, 1ml of 2/3 N Sulfuric acid.

4. Let stand for 5 minutes.
5. Poured, the mixture on filter paper and filtrate were collected in a clean dry tubes.

3.8 Collection of fresh rumen liquor from healthy slaughtered goat and its preparations for ready to use :

Two liter of rumen liquor from healthy slaughtered goats (slaughtered not more than 5 minutes before), after avisceration at butcher's shop from near market, was collected. After thorough mixing, the rumen fluid was then strained out, through double fold of musclin cloth in a sterillised screw capped vial. After observing the pH, colour and odour of rumen liquor, the fluid was used for therapeutic purpose. Fresh rumen liquor was collected for each time of treatment.

3.9. Therapeutic management:

The therapeutic measures were undertaken (in group B) at the onset of clinical signs & biochemical changes at 24 hrs of post-induction. The treatment scheduled adoped for each goat till recovery or death was as followed : -

(1) Sodium bicarbonate (7.5%w/v) Soln. Intravenously @ 4ml/kg b. wt. twice daily for 2 days.

(2) Normal saline (0.9%) Soln. approx. 100-200ml Intravenously once daily for three consecutive days.

(3) Sodium bicarbonate powder : 5 gms orally, once daily for 3 days suspended in water.

(4) Rumec powder : 15gms orally, twice daily for 3 days suspended in water.

(5) Tetracycline HCl Powder : orally @ 20 mg/kg b.wt. as single dose on 1st day suspended in water.

(6) Fresh rumen liquor orally @ 15 ml/kg b. wt. twice daily for 3 days.

(7) B-complex (Belamyl) inj.: 2ml Intramuscularly daily for 5 days.

(8) Avilvet inj. : 1ml Intramuscularly daily for 3 days.

(9) Dexona inj. : @1ml, 0.75ml and 0.5ml Intramuscularly on 1st, 2nd & 3rd day respectively.

3.10. Evaluation of Therapy :

The evaluation of therapy was based on recovery from clinical illness, restoration of physico-microbial and bio-chemical changes in rumen liquor, blood & serum towards normalcy.

3.11. Statistical analysis :

The data were analysed statistically to find out any significant difference in different parameters calculated at different time intervals using the analysis of variance as per the standard method described by Snedecor and Cochran (1967).

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(1) Avilvet injection (Intervet) contains 22.75 mg of pheniramine maleate/1ml of solvent.

(2) Dexona injection (Sarabhai Zydus) contains 4.4 mg of Dexamethasone sodium phosphate /1ml of solvent.

(3) Belamyl injection (Sarabhai Zydus) Each 1ml contains : Thiamine HCl I.P. : 10 mg, Riboflavin I.P. : 3 mg, Niacinamide I.P. : 100 mg, Vitamin B_{12} I.P.. : 10 mcg, & Crude Liver I.P. : 0.66 ml (equiv. to 2 mcg of cyanocobolamine)

(4) Rumec powder (Rakesh Pharmaceuticals) Each 100 gms. Contains : Zingiber Officinate 1.00 gm., Cassia Fistula 3.5 gm, Swertia Chirata 10.00 gm, Trachyspermum Ammi 2.00 gm, Aegle Marmelos 4.00 gm,

Copper Sulphate 0.2 gm, Black Salt 9.5 gm, Coriandrum Sativum 1.75 gm, Curcuma Longa 5.00 gm, Emblica Officinalis 9.00 gm, Tinospora Cordifolia 8.00 gm, Alpina Galanga 5.00 gm, Carryo Phyllus Aromaticus 6.00 gm, Sodium Chloride 9.50 gm, Melia Azadirachta 3.00 gm, Terminalia Chebula 4.00 gm, Terminalia Belerica 9.00 gm, Peucedanum Graveolens 2.00 gm, Cinnamomum Tamala 6.00 gm, Vernonia Anthelminitica 1.50 gm and Asafotida Compound 0.05 gm.

(5) Tetracycline HCl powder (Intervet) Each one gm contains 50 mg of Tetracycline HCl.

(6) Sodium bicarbonate powder 100 gm pack, A product of Medikem PVT. Ltd. Math Laxamanpur, Gulzarbagh, Patna-7.

(7) Sodium bicarbonate (7.5%) soln., a product of M/S Bengal Chemicals and Pharmaceutical Ltd., Kolkata, containing 7.5 gms sodium bicarbonate/100 ml of solvent.

(8) Normal saline (Albert and David), It contains 0.9 % NaCl.

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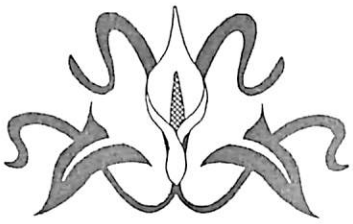
CHAPTER - IV



RESULTS AND DISCUSSION

RESULTS & DISCUSSION

4.1. Induction of ruminal acidosis :

For induction of experimental ruminal acidosis wheat grains has been commonly used in different species of farm animals (Tanwar and Mathur, 1983a; Sinha *et al.*, 1985 Crichlow, 1989, Lal *et al.*, 1992 and Chand *et al.*, 2001). Wheat grains being more potent to cause acidosis than other farm grains (Slyter, 1976; Elam, 1976), in the present study the ruminal acidosis was induced in both the groups of goats at a time by feeding whole wheat grains orally @ 100 gm/kg body weight after 24 hours of starvation and allowed free access to water. At 12 hours post-induction, the low pH of rumen fluid, reduced ruminal motility, high level of lactic acid in the rumen liquor & blood, increased pulse and respiration rates as compared to '0' hour value (healthy animals) suggested the establishment of ruminal acidosis. But, more pronounced symptoms were recorded at 24 hours onwards. Several workers have produced ruminal acidosis in different ruminants by administering wheat (Sinha *et al.*, 1985; Lal *et al.*, 1989, Patra *et al.*, 1997 Shukla *et al.*, 1999; Dwivedi *et al.*, 2000; Chand *et al.*, 2001), barley (Juhasz and Szegedi, 1968a; Dshurov, 1976; Nauriyal and Baxi, 1981; Shishkov, 1984), maize (Vestweber *et al.*, 1974; Dshurov, 1976), rice (Das *et al.*, 1992; Sen *et al.*, 1993), mangold (Scarisbrick, 1954), cereals (Broberg, 1960), glucose (Shinosaki and Nakabayashi, 1974; Dshurov, 1976), sucrose (Cakala *et al.*, 1974), lactic acid (Shinosaki, 1959; Juhasz and Szegedi, 1968a), ammonium chloride (Augustinson and Johanson, 1986), molasses (Angelov *et al.*, 1996, Nikolov, 1998a), apple (Teli *et al.*, 1986) & Sorghum flour (Nour *et al.*, 1998).

Table – 1. Clinical signs observed in both untreated (A) and treated (B) groups of acidotic goats.

Clinical singn	Group	Number of animals affected						
		Different hours of observations						
		0	12	24	48	72	96	120
Dullness	A	0/6	2/6	4/6	5/5	3/3	1/3	1/3
	B	0/6	2/6	4/6	4/5	2/5	1/5	0/5
Weakness	A	0/6	1/6	3/6	3/5	2/3	2/3	2/3
	B	0/6	1/6	2/6	2/5	1/5	0/5	0/5
Anorexia	A	0/6	6/6	6/6	5/5	3/3	3/3	3/3
	B	0/6	6/6	6/6	3/5	2/5	1/5	0/5
Distended rumen	A	0/6	2/6	3/6	3/5	2/3	1/3	1/3
	B	0/6	2/6	4/6	3/5	1/5	0/5	0/5
Diarrhoea	A	0/6	3/6	3/6	4/5	3/3	2/3	2/3
	B	0/6	2/6	3/6	2/5	1/5	1/5	0/5
Constipation	A	0/6	3/6	3/6	1/5	0/3	0/3	0/3
	B	0/6	3/6	2/6	0/5	0/5	0/5	0/5
Abdominal pain	A	0/6	2/6	4/6	3/5	1/3	1/3	1/3
	B	0/6	3/6	3/6	2/5	1/5	0/5	0/5
Nasal discharge	A	0/6	0/6	2/6	2/5	1/3	1/3	1/3
	B	0/6	0/6	2/6	1/5	1/5	0/5	0/5
Lateral recumbency	A	0/6	1/6	2/6	3/5	1/3	1/3	1/3
	B	0/6	1/6	2/6	1/5	0/5	0/5	0/5
Stand quietly	A	0/6	2/6	2/6	2/5	2/3	1/3	1/3
	B	0/6	2/6	4/6	2/5	1/5	0/5	0/5

Cont. .....

Table 1. cont. ...

Lameness	A	0/6	0/6	0/6	1/5	1/3	1/3	1/3
	B	0/6	0/6	0/6	0/5	0/5	0/5	0/5
Staggering gait	A	0/6	1/6	4/6	4/5	2/3	2/3	1/3
	B	0/6	1/6	2/6	1/5	0/5	0/5	0/5
Apparent	A	0/6	1/6	2/6	1/5	1/3	1/3	1/3
blindness	B	0/6	2/6	2/6	1/5	0/5	0/5	0/5
Pupil dilated	A	0/6	0/6	2/6	2/5	1/3	1/3	0/3
	B	0/6	0/6	2/6	1/5	1/5	0/5	0/5
Grinding of	A	0/6	2/6	4/6	3/5	2/3	1/3	1/3
teeth	B	0/6	2/6	3/6	2/5	1/5	1/5	0/5
Head pressing	A	0/6	1/6	2/6	2/5	2/3	1/3	1/3
	B	0/6	2/6	2/6	1/5	1/5	0/5	0/5
Regurgitation of	A	0/6	1/6	3/6	3/5	0/3	0/3	0/3
undigested wheat	B	0/6	1/6	2/6	1/5	0/5	0/5	0/5
Thirst	A	0/6	2/6	3/6	3/5	2/3	2/3	2/3
	B	0/6	3/6	3/6	3/5	2/5	1/5	0/5
Sunken eye	A	0/6	0/6	1/6	2/5	2/3	2/3	1/3
	B	0/6	0/6	1/6	2/5	1/5	1/5	0/5
Mortality	A	0/6	0/6	0/6	1/6	2/5	0/3	0/3
	B	0/6	0/6	0/6	1/6	0/5	0/5	0/5

4.2. Clinical symptoms :

As depicted in different tables of clinico-biochemical observations at '0' hour all the animals were physiologically normal before feeding of whole wheat grains. The clinical and bio-chemical status of goats (groupA) at various sampling hours before and after induction of acidosis was observed and evaluated under different tables.

As evident from table-1 (group A) results may be explained & discussed under the following heads :

4.2.1. Dullness :

Dullness was observed in 33, 66 and 100 percent of animal at 12, 24 hours and 48, 72 hours of post-induction of acidosis respectively. Thereafter, 1 out of 3 survivors still continued to show dullness throughout the study. Similar manifestations were reported in goats (Tanwar and Mathur, 1983a; Lal *et al.*, 1989; Hawa Singh *et al.*, 2001), sheep (Vestweber *et al.*, 1974; Albrycht *et al.*, 1986) and calves (Chand *et al.*, 2001; Dwivedi *et al.*,2000). It is possibly due to toxic effect of lactic acid on CNS (Dunlop and Hammond, 1965) or due to polioencephalomalacia owing to thiamine (Vit.-B_1) antagonism (Brent, 1976).

4.2.2. Weakness :

Weakness was noticed in one of the animal at 12 hours of induction of ruminal acidosis while 3 animals were noticed being weak from 24 to 48 hours. From 72 to 120 hours of experiment, 2 out of 3 survivors were found to remain weak. Weakness is one of the principal symptom in ruminal acidosis (Blood *et al.*, 1983) and is associated with dehydration due to increased osmotic pressure of rumen fluid resulting into passage of water from the blood into rumen (Parthasarathy and Phillipson 1953). This

observation collaborates with the earlier findings of Gnanaprakasam (1970), Sen *et al.* (1982) and Tanwar and Mathur (1983a) in goats.

42.3. Anorexia :

All the animals under experiment became anorectic following induction of ruminal acidosis and remained so throughout the period of observations. Similar observation was reported in goat (Tanwar and Mathur, 1983a and Das, 1990), sheep (Broberg, 1960) & in calves (Shukla *et al.*, 1999; Dwivedi *et al.*, 2000; Chand *et al.*, 2001). However, Shinosaki (1959) did not notice any reduction in appetite in acidotic sheep. In rumen acidosis anorexia occurs due to excessive production and absorption of toxic factors like lactic acid, histamine and amines (Huber, 1976) or due to decreased number of flora (Phillipson, 1955) or partial absence of bile salts coupled with distended liver in acute hepatitis (Blood *et al.*, 1983).

4.2.4. Distended rumen :

33, 50, 60 and 66 percent of the experimental animal showed distended rumen at 12, 24, 48 and 72 hours respectively. Thereafter, only 33 percent survivors continued to show this symptom. This was an important clinical manifestation noted in ruminal acidosis. The rumen was full, firm and doughy on palpation. The present finding was in accordance with those described by Gnanaprakasam (1970), Sen (1982) and Das (1990). This might be due to excessive feeding of grains and accumulation of fluid in the rumen after its withdrawal from circulatory system due to increased osmolarity of rumen fluid (Blood *et al.*, 1983; Chakrabarti, 1988).

4.2.5. Diarrhoea :

It was a prominent symptom in the present study. 50 percent of animal showed diarrhoea between 12 to 24 hours. Thereafter, 80 to 100 percent of surviving animal showed diarrhoea between 48 to 72 hours, which again from 96 hours, only 33 percent of survivor remained to show this symptom to the end of experiment. Similar observation was noticed in goat (Gnanaprakasam, 1970; Tanwar and Mathur, 1983a), sheep (Nisbet *et al.*, 1963) & in calves (Dwivedi *et al.*, 2000). Hypertonicity and depletion of bicarbonate ions in the intestine which was the sole factor for causing acidosis may be the cause for diarrhoea or reduced net absorption of water from colon may be the reason of diarrhoea (Lee, 1977) or, due to inflammatory and ulcerative changes of wall of the intestine due to lactic acid (Castello, 1968) or due to secondary bacterial infection of the intestinal wall may be the cause of diarrhoea.

4.2.6. Constipations :

50 percent of the animal developed constipation following induction of acidosis in this study upto 24 hours of observation. Thereafter, 20 percent of experimental animal still continued to show constipation only upto 48 hours. Similar observation was reported by Gnanaprakasam (1970) and Tanwar and Mathur (1983a) in ruminal acidosis in goats. This may be due to hypotonicity of intestine following ruminal stasis under the influence of low ruminal pH.

4.2.7. Abdominal pain :

33, 67 and 60 percent of the experimental animal showed abdominal pain evidenced by kicking at the belly at 12, 24 and 48 hours of observation respectively. Thereafter, 1 out of 3 survivors still continued to show

abdominal pain throughout the study. Almost similar observation has been reported by Ahrens (1967). The abdominal pain might have resulted due to damage of ruminal epithelium on account of corrosive action of accumulated lactic acid in the rumen (Dunlop and Hammond, 1965; Ahrens, 1967). Excessive accumulation of fluid in rumen from general circulation along with distension, creates pressure on the nerve endings which may also be the cause of abdominal pain (Blood and Radostits, 1989).

4.2.8. Nasal discharge :

It was observed that 33 to 40 percent of the animal showed mucoid to purulent nasal discharge at 24 and 48 hours respectively after the induction of acidosis. Again from 72 hours, 1 out of 3 survivors still continued to show nasal discharge to the end of experiment. Almost similar finding was reported by Tanwar and Mathur (1983a) in goats, Dunlop and Hammond (1965) in holstein heifers and Dwivedi *et al.* (2000) in calves following acidosis. The nasal discharge may be due to hyperacidity causing nasal irritations or may be due to secondary bacterial infection of respiratory system.

4.2.9. Lateral recumbency :

1, 2 and 3 animals developed lateral recumbency at 12, 24 and 48 hours respectively. Thereafter, 1 of the animal remained to show lateral recumbency throughout the study period. This finding coincides with the previous observation recorded by Scarisbrick (1954) and Nisbet *et al.* (1963) in sheep. Blood *et al.* (1983) pointed that lateral recumbency usually followed after 48 hours in large animal but it might be present as an early sign of ruminal acidosis. This may be due to dehydration, muscular weakness, hypercalcaemia and possible toxic effect on the CNS.

4.2.10. Stand quietly:

Two of the experimental animals were disinclined to move and remained to stand quietly upto 72 hours of experiment, whereas 1 out of 3 survivors were remained to stand quietly upto 120 hours of observation. Such symptom has also been described in affected animals by Blood *et al.* (1983) and it might be due to the toxic effect of lactic acid on the CNS of the affected animals in acidosis.

4.2.11. Lameness :

One of the animal showed lameness from 48 hours and remained so throughout the period of experiment. This type of symptom has been reported in acidosis in goats (Tanwar and Mathur, 1983a), Sheep (Scarisbrick, 1954) and in other ruminants (Dunlop and Hammond, 1965). Laminitis in acidotic goats might be possible due to excessive production and absorption of lactic acid. It might also be due to increased histamine toxicity in acidotic animals (Ahrens, 1967; Dunlop, 1972).

4.2.12. Staggering gait :

Out of six, 1 and 4 animals showed staggering gait at 12 and 24, 48 hours respectively, where as 2 animals remained to show staggering gait upto 96 hours. Thereafter, only 1 survivor showed this symptom of staggering gait upto 120 hours of observations. Similar observation has been recorded in goat (Gnanaprakasam, 1970; Tanwar and Mathur, 1983a), sheep (Nisbet *et al.*, 1963) and in ruminants (Mackenzie, 1967). This may be due to CNS disturbances on account of histamine toxicity and lactacidaemia (Dunlop and Hammond, 1965).

4.2.13. Apparent blindness :

Apparent blindness was noticed in one of the experimental animal at 12 hours and in 2 animals at 24 hours of experiment, out of which one animal remained blind from 48 hours to the end of experiment. Similar observation was recorded by Nisbet *et al.* (1963) and it might be due to the toxic effect of lactic acid on the CNS.

4.2.14. Pupil dilated :

Two of the experimental animals showed pupillary dilatation from 24 to 48 hours. Whereas, only 1 animal remained to show pupillary dillatation upto 96 hours of observation period. However, this symptom disappeared at 120 hours. Blood *et al.* (1983) noted that in severely affected animals the pupillary light reflection was usually present but slower than normal. Dunlop and Hammond, 1965 and Chand *et al.*, 2001 have observed loss of pupillary reflex in cattle suffering from acidosis. This might be due to toxic effect of lactic acid, histamine and amines on the CNS.

4.2.15. Grinding of teeth :

33 to 67 percent of the animal showed this symptom between 12 to 48 hours of the observation. Thereafter, the number declined but one of the survivor remained to show this symptom till the experiment ended. The present observation finds similarity with the observation reported by other workers in goat (Gnanaprakasam, 1970; Tanwar and Mathur, 1983) and in sheep (Scarisbrick, 1954). This might be due to the toxic effect causing irritation on the CNS or may also be due to toxic effect of lactacedemia (Lal *et al.*, 1989).

4.2.16. Head pressing :

This was seen in 1 animal at 12 hours while the number increased to 2 at 24 hours which remained upto 72 hours of experiment. Thereafter, the number of animals affected, declined to 1 from 96 hours to 120 hours of experiment. This was also seen by Dunlop and Hammond (1965) in heifers following wheat engorgement. This may be attributable to the effect of lactic acid toxicity.

4.2.17. Regurgitation of undigested wheat :

This was recorded in one of the animal at 12 hours and later on 3 of the animals suffered at 24 hours which remained upto 48 hours of experiment, when the effect of acidosis was maximum. This observation get support with the observation recorded by Tanwar and Mathur (1983a) in goat with wheat induced acidosis which they noticed on third day of experiment. This may be due to engorged rumen under the influence of low rumen pH.

4.2.18. Thirst :

This was noticed in 33, 50 and 60 percent of experimental animal at 12, 24 and 48 hours respectively. Thereafter, 2 out of 3 survivors remained to show this symptom upto the observation completed. This finding was similar to the observation of Broberg (1960) in sheep. This may be due to progressive diarrhoea and sequential effect of dehydration following acidosis.

4.2.19. Shunken eye :

One of the experimental animal developed this symptom at 24 hours while 2 of the animals developed this symptom from 48 hours and remained

upto 96 hours of observation. Thereafter, only 1 survivor showed this symptom upto the end of observation. This finding was similar to the observation noted by Tanwar and Mathur (1983a). This indicated that the animals had suffered from dehydration was due to increased osmotic pressure of rumen fluid resulting into passage of water from blood into the rumen (Parthasarathy and Phillipson, 1953).

4.2.20. Mortality :

In the present study 3 of the experimental animals could not survive due to severity of the disease produced in them and one had died between 24 to 48 hours and another 2 animals died between 48 to 72 hours while 3 of the animals surived the period of experiment. Before death, animals were in lateral recumbency with head turned towards the flank and did not respond to stimuli indicated comatose condition simulating milk fever posture. The incidence of death due to lactic acidosis was also previously reported by Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983a) and Das (1990) in goats. The cause of death might be due to toxaemia and dehydration which caused cardio-respiratory failure and hepato-renal failure. Acute carbohydrate over load cause secondary paralysis of respiratory centre due to accumulation of CO_2 (Castello, 1968).

The variation in percentage of death at different time intervals might be due to the individual variation in the bio-chemical changes in blood and rumen fluid in the particular animal as reported by Dougherty *et al.* (1975). Slyter (1976) reported that individual animal differs in respect of salivation, ingestafill, intestinal motility and ability to excrete or use large quantities of potentially toxic compound. Nisbet *et al.* (1963) reported flock mortality to be 5-20%. Sandha and Chaudhary (1985) reported 66% mortality in buffalo

calves between 24 to 48 hours while Sinha *et al.* (1985) observed mortality to be 66% at 48 hours of induced rumen acidosis. Blood *et al.* (1983) reported that the death usually occured within 24 to 74 hours of induction of acidosis and it might go upto 90% in untreated case. Thus, this finding was in close agreement with the above workers.

4.3. Clinical observations:

The mean along with their standard error (S.E.) and coefficient of variation percentage (C.V.%) of different clinical observations noted in untreated acidotic goats (group A) at various intervals of time has been presented in table 2 and 4 (group A). The results of analysis of variance of different clinical observations noted between hours are also shown in table-3 and 5 (group A).

4.3.1. Pulse/min. :

As evident from table-2 (group A) and fig. – 1 (group A) it was observed that mean pulse rate increased from '0' hours level of 73.00 ± 0.730 to a significant level ($P \leq 0.01$) of 112.16 ± 0.945 at 24 hours and thereafter, declined subsequently throughout the study period, but remained significantly higher than '0' hours value. Similar trend was also reported by different workers in sheep (Juhas'z and Szegedi, 1968a; Vestweber *et al.*, 1974; Nikolov, 2000), goat (Gnanaprakasam, 1970; Sen *et al.*, 1982; Tanwar and Mathur, 1983a; Cao *et al.*, 1987; Lal *et al.*, 1989 and Das, 1990) and in cattle (Dunlop and Hammond, 1965). The increased pulse rate might be due to the toxic effect of accumulated lactic acid which after absorption causes severe acidosis, fall in plasma volume and circulatory failure (Blood *et al.*, 1983).

Table – 2. Mean ± S.E. along with their C.V. % of different attributes of clinical observations recorded periodically in both untreated (A) and treated (B) groups of acidotic goats.

Hrs.	Pulse/min.		Respiration /min.		Temperature (^{O}F)	
	Group A	Group B	Group A	Group B	Group A	Group B
0	73.00^{a}±0.730 (2.450)	71.00^{a}±0.516 (1.781)	21.00^{a}±0.632 (7.377)	22.00^{a}±0.516 (5.749)	102.70^{a}±0.073 (0.174)	102.60^{a}±0.154 (0.369)
12	96.83^{b}±0.980 (2.480)	99.16^{b}±1.058 (6.579)	34.16^{b}±0.304 (5.041)	35.00^{b}±0.577 (4.040)	103.50^{b}±0.169 (0.400)	103.60^{b}±0.139 (0.328)
24	112.16^{c}±0.945 (2.065)	111.00^{c}±1.100 (6.110)	38.33^{c}±0.241 (3.564)	37.66^{c}±0.333 (2.168)	102.10^{c}±0.177 (0.424)	103.50^{b}±0.161 (0.381)
48	101.20^{d}±0.440 (2.460)	90.20^{d}±0.701 (4.794)	31.60^{d}±0.201 (3.608)	30.80^{d}±0.860 (6.245)	102.24^{ac}±0.203 (0.446)	103.40^{b}±0.209 (0.453)
72	90.00^{e}±0.707 (4.444)	83.20^{e}±0.530 (3.931)	29.00^{e}±0.353 (6.896)	25.00^{e}±0.316 (2.828)	102.50^{ac}±0.057 (0.097)	102.00^{c}±0.388 (0.851)
96	80.66^{f}±0.620 (4.353)	76.80^{f}±0.806 (6.471)	26.00^{f}±0.353 (7.692)	23.00^{ae}±0.316 (3.074)	100.23^{d}±0.622 (1.076)	102.30^{ac}±0.240 (0.526)
120	77.33^{f}±0.444 (3.254)	75.00^{af}±0.658 (5.416)	24.00^{f}±0.353 (8.333)	21.40^{a}±0.678 (7.086)	102.60^{ac}±0.230 (0.389)	102.40^{ac}±0.141 (0.308)

Note: - 1. Means (column wise) superscripted with different letters were significantly different from each other.
2. Figure in parentheses indicate C.V.%.

Table – 3. Analysis of variance of periodic variation in different attributes of clinical observations recorded in both untreated (A) and treated (B) groups of acidotic goats.

Parameter	Group	S.V.	D.F.	M.S.	F
Pulse / Min.	A	Between hours	6	1035.345	156.113**
		Error	25	6.632	
	B	Between hours	6	1207.079	51.033**
		Error	31	23.652	
Respiration / min.	A	Between hours	6	196.355	75.097**
		Error	25	2.614	
	B	Between hours	6	249.501	156.781**
		Error	31	1.591	
Temperature (^{O}F)	A	Between hours	6	3.783	17.327**
		Error	25	0.218	
	B	Between hours	6	2.321	9.545**
		Error	31	0.243	

** Significant (P≤0.01)

4.3.2. Respiration rate :

On perusal of table-2 (group A) and fig. 2 (groupA) it was noticed that the mean respiration rate recorded at '0' hours was 21.00 ± 0.632/min. which continued to increase significantly ($P \leq 0.01$) upto 24 hours (38.33 ± 0.241/min) then, declined at subsequent hours, but the values remained significantly higher than that of '0' hours. This finding has got the similarity to the observation of other workers in sheep (Vestweber *et al.*, 1974; Patra, 1991; Nikolov, 2000) & in calves (Dwivedi *et al.*, 2000). The increase in respiration rate might be due to stimulation of respiratory centre by increased CO_2 tension of blood and decreased blood pH bringing about an increased rate of CO_2 elimination (Huber, 1976).

4.3.3. Rectal Temp (^{0}F) :

On perusal of table-2 (group A) and fig. 3 (group A) it was observed that the mean rectal temperature increased from '0' hours level of 102.70 ± 0.073 to a significant level ($P \leq 0.01$) of 103.50 ± 0.169 at 12 hours which again decreased to the level of 102.10 ± 0.177 at 24 hours. Thereafter increased subsequently, but, not so markedly from 24 hours except at 96 hours, where subnormal temp. (100.23 ± 0.622) was found. The present finding coincide with the finding of Dunlop and Hammond (1965), Gnanaprakasam (1970), Cao *et al.* (1987) and Lal *et al.* (1989) but, did not properly coincide with the finding of Sen (1982) in goat and Vestweber *et al.* (1974) in sheep. The initial rise in temp. might be due to increased metabolic turnover following feeding of whole wheat grains. The subsequent rise in temp. might be due to bacterial ruminitis (Blood *et al.*, 1983). The overall significant fall of rectal temp. throughout the period of study might be due to

Table – 4. Mean ± S.E. along with their C.V. % of different attributes of clinical observations recorded periodically in both untreated (A) and treated (B) groups of acidotic goats.

Hours	Rumen motility/5 minutes		Skin inelasticity (Sec.)	
	Group A	Group B	Group A	Group B
0	6.50^{a}±0.223 (8.426)	6.66^{a}±0.210 (7.745)	2^{a}±0.447 (54.772)	2^{a}±0.365 (44.721)
12	2.83^{bd}±0.166 (14.408)	3.00^{b}±0.365 (29.814)	2^{a}±0.365 (44.712)	2^{a}±0.516 (63.245)
24	1.66^{c}±0.210 (30.983)	1.66^{c}±0.210 (30.985)	20^{b}±0.930 (11.401)	22^{b}±1.064 (11.853)
48	1.40^{bc}±0.244 (39.123)	2.60^{b}±0.244 (21.066)	30^{c}±1.673 (12.472)	24^{b}±2.167 (20.198)
72	2.00^{ce}±0.000 (0.000)	3.40^{b}±0.244 (16.109)	6^{d}±1.154 (33.333)	4^{c}±0.547 (30.681)
96	2.66^{de}±0.333 (21.650)	5.20^{d}±0.374 (16.089)	10^{e}±1.154 (20.000)	2^{ac}±0.447 (50.000)
120	3.33^{d}±0.333 (17.320)	6.40^{a}±0.244 (8.558)	3^{ad}±0.577 (33.333)	2^{ac}±0.316 (35.355)

Note: - 1. Means (column wise) superscripted with different letters were significantly different from each other.
2. Figure in parentheses indicate C.V.%.

Table – 5. Analysis of variance of periodic variation in different attributes of clinical observations recorded in both untreated (A) and treated (B) groups of acidotic goats.

Parameter	Group	S.V.	D.F.	M.S.	F
Rumen motility / 5 minutes	A	Between hours	6	16.794	67.720**
		Error	25	0.248	
	B	Between hours	6	21.418	50.814**
		Error	31	0.421	
Skin inelasticity (Sec.)	A	Between hours	6	588.828	133.824**
		Error	25	4.400	
	B	Between hours	6	554.035	112.993**
		Error	31	4.903	

** Significant (P≤0.01)

lactic acidosis leading to dehydration, fall in plasma volume, severe depression of cardiovascular system and fall in blood pressure (Dunlop and Hammond, 1965).

4.3.4. Rumen motility / 5 min. :

As noted in table-4 (group A) and fig.-4 (group A) the motility of rumen was 6.50 ± 0.223 at '0' hours which significantly ($P \leq 0.01$) decreased to 1.40 ± 0.244 at 48 hours following induction of ruminal acidosis. Thereafter, increased rumen motility was recorded, but the values were always lowered than '0' hours value throughout the investigation. This was in agreement with other workers in goats (Gnanaprakasam, 1970; Sen *et al.*, 1982; Tanwar and Mathur, 1983) and sheep (Broberg, 1960; Juhas'z and Szegedi, 1968; Vestweber *et al.*, 1974; Nikolov, 2000). The decreased rumen motility may be due to elevated hydrogen ion concentration in the rumen ingesta following induction of acidosis which inhibited some metabolic pathways, destroyed some species of organisons, damaged rumen epithelium and helped in absorption of organic acid leading to ruminal atony (Ahrens, 1967; Kay *et al.*, 1969). Huber (1976) suggested involvement of hydrogen ion receptors in gastrointestinal tract, central inhibition of absorbed acid, an inhibition by absorbed amines and thus possibly leading to reduced ruminal motility. Stern *et al.* (1970) were of the opinion that the increased plasma immuno reactive insulin was the reason for decreased ruminal motility. Svendsen (1973) observed that butyric acid was the most potent inhibitor of ruminal motility. The entry of lactic acid in duodenum exerts extra reflex inhibitory action on rumen, which may lower the ruminal motility, however, recent evidences suggest that the increased molar concentration of butyrate causes the ruminal stasis (Blood & Radostits, 1989). Crichlow (1988)

suggested that the non-dissociated volatile fatty acid concentration may have been responsible for the reduced rumen motility.

4.3.5. Skin inelasticity :

As depicted in table-4 (group A) and fig.-5 (group A) no retention of skin fold within '0' hours to 12 hours, indicated no or mild (4-6%) dehydration. Retention of skin fold for 20-30 seconds from 24 to 48 hours of induction of acidosis indicated 10% dehydration, retention for 6-10 seconds from 72 to 96 hours indicated 8% dehydration and retention for 3 seconds at 120 hours indicated 6% dehydration. Similar observation was noticed by Gnanaprakasam (1970) and Tanwar and Mathur (1983a). The retention of skin fold might be due to loss of skin elasticity occured due to less perfusion of water in the peripheral region. The less perfusion of water in the peripheral region might be due to heavy drainage of water from blood in to the rumen content.

4.4. Examination of Rumen Liquor :

4.4.1. Macroscopic examination :

4.4.1.1. Smell :

As depicted in table-6 (group A), it was observed that the smell of rumen liquor of goats was aromatic at '0' hours which after wheat feeding changed at first to faintly sour to sour from 12 to 24 hours then, to intense sour upto 72 hours and again to sour and faintly sour from 96 hours to the end of study period. This findings were close to the findings of Sen (1982), Sen *et al.* (1982) & Lal *et al.* (1989) in goats. The sourness smell of rumen liquor might be due to rise in the concentration of lactic acid.

Table – 6. Physical and microbial changes in rumen liquor recorded periodically in both untreated (A) and treated (B) groups of acidotic goats.

Hrs.	Colour		Odour/Smell		Consistency		Protozoal motility	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
0	Greenish Brown	Greenish Brown	Aromatic	Aromatic	+++	+++	4	4
12	Light Grey	Light Grey	Faintly Sour	Faintly Sour	++	++	2	2
24	Greyish	Greyish	Sour	Sour	++	++	1	1
48	Greyish	Light Grey	Intense Sour	Sour	+	+	1	2
72	Greyish	Light Grey	Intense Sour	Faintly Sour	+	++	1	3
96	Greyish	Greenish Brown	Sour	Aromatic	++	+++	2	4
120	Light Grey	Greenish Brown	Faintly Sour	Aromatic	++	+++	2	4

Note :- 1, represents absent
2, represents slow
3, represents moderate
4, represents vigorous
+++, represents viscous
++, represents Slight viscous
+, represents watery.

4.4.1.2. Colour :

As depicted in table-6 (group A) it was observed that the colour of rumen liquor in the study was greenish brown at '0' hours which changed to light grey to greyish in colour from 12 to 96 hours thereafter, it turned towards light grey at the end of observation. Similar observations were also reported by Gnanaprakasam (1970), Sen *et al.* (1982) and Lal *et al.* (1989) in goats. This variation in colour might be occurred due to type of feed taken by the animal and species variation (Rosenberger *et al.*, 1979; Das *et al.*, 1972).

4.4.1.3. Consistency :

As depicted in table-6 (group A) it was observed that the consistencyof rumen liquor of the acidotic group was gradually reducing from viscous (+++) at '0' hours to slight viscous (++) at 12 and 24 hours and then to watery (+) from 48 to 72 hours which was again became semisolid from 96 hours to the end of experiment. These results were in close agreement with the observations reported by Sen *et al.* (1982), Lal *et al.* (1989) and Das (1990) in goats and Rosenberger *et al.* (1979) in cattle. This phenomenon might be due to passage of fluid from vascular bed to rumen as a result of increased osmomolality of the rumen content.

4.4.2. Microscopic examination :

4.4.2.1. Motility of rumen protozoa :

As depicted in table-6 (group A) it was observed that the motility of rumen protozoa was vigorous (4) at '0' hours which became slow (2) at 12 hours to absent (1) at 24 hours and remained so upto 72 hours thereafter, it became slow (2) to the end of experimental period. Many workers reported a gradual loss in the rumen protozoa as the rumen pH falls (Ahrens, 1967;

Eadie *et al.*, 1970; Das *et al.*, 1972; Prasad *et al.*, 1973; Sinha *et al.*, 1985). Marked reduction in the motility of the protozoa might be due to low intracellular and environmental pH (Prins and Van Hoven, 1977) and high toxicity of rumen encountered in lactic acidotic animals (Ahrens, 1967). Hungate *et al.* (1952) and Krogh (1959) reported that factors like low rumen pH and high rumen toxicity killed protozoa. It was also been suggested that lysis of the rumen protozoa occurred when the rumen pH falls below 5.5 (Hungate, 1966). The reappearance of rumen protozoa from 96 hours synchronized with an improvement in rumen pH (Dunlop, 1972).

4.4.3. Biochemical changes in rumen liquor :

The mean along with their standard error (S.E.) and cofficient of variation percentage (C.V.%) of different bio-chemical attributes of rumen liquor of untreated acidotic goats (group A) at different interval of study has been presented in table-7(groupA). The results of analysis of variance of different bio-chemical attributes of rumen liquor between hours are also shown in table-8 (group A).

4.4.3.1. pH of rumen liquor :

On perusal of table – 7 (group A) and fig.-6 (groupA) it was observed that the ruminal pH in the present study decreased gradually from 6.97 ± 0.025 at '0' hours to a significant level ($P \leq 0.01$) of 4.95 ± 0.023 at 12 hours and to 4.52 ± 0.152 at 24 hours of post-induction. Thereafter, from 48 hours to the end of observation there was gradual improvement of ruminal pH, but values were still significantly ($P \leq 0.01$) remained lower to that of '0' hours value. A similar trend was reported by Dougherty *et al.* (1975), Cao *et al.* (1987)& Lal *et al.*(1989). The decrease in ruminal pH might be due to complete and faster fermentation of whole wheat grains by amylolytic

Table – 7. Mean ± S.E. along with their C.V. % of different bio-chemical attributes of rumen liquor recorded periodically in both untreated (A) and treated (B) groups of acidotic goats.

Hrs.	pH		Lactic acid (mg/dl)		TVFA (mEq/L)	
	Group A	Group B	Group A	Group B	Group A	Group B
0	6.97^{a}±0.025 (0.907)	6.92^{a}±0.024 (0.857)	3.96^{a}±0.067 (4.152)	3.87^{a}±0.062 (3.962)	65.80^{a}±0.814 (3.030)	66.30^{a}±0.186 (0.687)
12	4.95^{b}±0.023 (1.142)	4.90^{b}±0.338 (1.692)	118.60^{b}±3.099 (6.401)	119.83^{b}±1.672 (8.605)	110.58^{b}±1.141 (2.529)	115.99^{b}±1.250 (6.647)
24	4.52^{c}±0.152 (8.259)	4.55^{c}±0.169 (9.127)	150.91^{c}±2.640 (4.285)	151.64^{c}±1.713 (6.965)	58.83^{c}±0.406 (3.911)	58.64^{c}±1.162 (12.225)
48	4.68^{c}±0.032 (1.562)	5.30^{d}±0.036 (1.555)	82.94^{d}±1.387 3.739)	70.60^{d}±0.758 (6.619)	32.03^{d}±0.512 (3.577)	35.28^{d}±0.827 (14.457)
72	5.12^{b}±0.011 (0.390)	6.10^{e}±0.036 (1.484)	36.68^{e}±1.662 (5.507)	26.06^{e}±0.754 (17.974)	47.23^{e}±0.242 (2.900)	49.76^{e}±1.472 (18.246)
96	5.94^{d}±0.023 (0.673)	6.88^{a}±0.282 (0.919)	17.76^{f}±0.555 (5.419)	12.26^{f}±0.637 (32.071)	50.95^{f}±0.358 (3.840)	58.56^{ac}±0.956 (10.071)
120	6.45^{e}±0.028 (0.775)	6.98^{a}±0.038 (1.232)	6.56^{a}±0.598 (15.804)	4.26^{af}±0.168 (24.338)	55.80^{g}±0.364 (3.696)	66.20^{ac}±0.912 (8.496)

Note: - 1. Means (column wise) superscripted with different letters were significantly different from each other.
2. Figure in parentheses indicate C.V.%.

Table - 8. Analysis of variance of periodic variation in different bio-chemical attributes of rumen liquor recorded in both untreated (A) and treated (B) groups of acidotic goats.

Parameter	Group	S.V.	D.F.	M.S.	F
pH	A	Between hours	6	4.593	150.476**
		Error	25	0.030	
	B	Between hours	6	5.968	118.383**
		Error	31	0.032	
RLA	A	Between hours	6	17341.131	790.854**
		Error	25	21.927	
	B	Between hours	6	20653.152	481.069**
		Error	31	42.931	
TVFA	A	Between hours	6	3309.669	747.847**
		Error	25	4.425	
	B	Between hours	6	3604.743	89.059**
		Error	31	40.475	

** Significant (P≤0.01)

bacteria leading to production of lactic acid (Ahrens, 1967), reduction in concentration of rumen ammonia nitrogen and also high concentration of TVFA (Phillipson, 1942). The improvement of the rumen pH from 48 hours might be due to reappearance of greater number of cellulolytic and other types of bacteria (Eadie and Mann, 1970).

4.4.3.2. Rumen lactic acid :

On perusal of table-7 (group A) and fig.-7 (groupA)it was observed that following induction of ruminal acidosis, the lactic acid concentration increased significantly ($P \leq 0.01$) to 118.60 ± 3.099 mg/dl at 12 hours as against '0' hours. value of 3.96 ± 0.067 mg/dl. The maximum concentration of lactic acid was 150.91 ± 2.640 mg/dl at 24 hours of induction of acidosis which gradually decreased at subsequent hours. But,the values always remained significantly ($P \leq 0.01$) higher than that of '0' hours value throughout the period of experiment except at 120 hours of observation, were non-significant ($P \leq 0.01$) elevated value (6.56 ± 0.598) was found. These findings agreed with Sen (1982), Tanwar and Mathur (1983b), Vihan and Rai (1985), Cao *et al.*(1987) and Lal *et al.* (1989) in goats. The rise in lactic acid concentration was due to faster and complete fermentation of starch by amylolytic bacteria in the rumino-reticulum compartment of the engorged animals which led to the production of large amount of lactic acid in the rumen (Dunlop and Hammond, 1965; Dunlop, 1972; Slyter, 1976). The predominance of gram positive bacteria particularly *Streptococcus bovis* and *Lactobacilli* which fermented soluble sugar at a faster rate to produce lactic acid (Mac Pherson, 1953) and stopage of growth of some of the lactate utilizing bacteria at low pH (Slyter *et al.*, 1976) may also increased lactic acid concentration in the rumen. Normal ruminal contents remove added

lactate rapidly and the lactic acid get absorbed rapidly at low pH, but, once ruminal acidosis sets in, lactic acid get absorbed at relatively slow rates (William and Mackenzie, 1965). Gradual decline in concentration of lactic acid in the rumen might be due to buffering of some lactic acid by rumen buffers, absorption of lactic acid through ruminal wall into circulation and decrease in fermentative process by amylolytic bacteria.

4.4.3.3. Total volatile fatty acids :

On perusal of table-7 (group A) and fig.-8 (groupA) it was noted that total volatile fatty acid value rose significantly ($P \leq 0.01$) to 110.58 ± 1.141 mEq/L as against the '0' hours value of 65.80 ± 0.814 mEq/L at 12 hours, of induction of acidosis. This level declined significantly ($P \leq 0.01$) to the lowest concentration of 32.03 ± 0.512 mEq/L at 48 hours thereafter from 72 hours, to the end of experiment the concentration of TVFA increased gradually but remains lower than that of '0' hours value. This observations were very close to the findings of Sinha, *et al.* (1985), Rai and Pandey (1978) and Blood *et al.* (1983). However, Sen, *et al.* (1982 and Phillipson (1952) and Scarisbrick (1954) reported lower value of TVFA following induction of ruminal acidosis in goat and sheep respectively. Abrupt rise in the value of TVFA at 12 hours may be attributable to the rapid fermentation of starch by amylolytic bacteria in the paunch and subsequent decrease in the value might be due to increased absorption at low ruminal pH (Danielli *et al.*, 1945; Gray, 1948) or due to reduction of rumen microbial fermentation (Randhwa *et al.*, 1989a). Gradual increase in concentration of TVFA from 72 hours might be due to improvement of normal rumen fermentative process synchronized with the improvement of rumen pH. However, Reid *et al.* (1957) pointed out that increase in TVFA after 48 hours was due to

development of a group of TVFA producing bacteria which were resistant to low rumen pH and capable of producing TVFA.

4.5. Analysis of blood :

The mean along with their standard error (S.E.) and cofficient of variation percentage (C.V.%) of different bio-chemical attributes of blood of untreated acidotic goats (group A) at different hours of observations has been presented in table-9 (group A). The results of analysis of variance of different bio-chemical attributes of blood between hours are also shown in table-10 (group A).

4.5.1. Blood urea nitrogen :

On perusal of table-9 (group A) and fig.-9 (group A) it was noticed that the level of blood urea nitrogen at '0' hours was 14.85 ± 0.200 mg/dl which gradually rose significantly ($P \leq 0.01$) to 38.90 ± 0.186 mg/dl at 72 hours and then showed the tendency to fall down but the values remained elevated throughout the period of observation. Other workers have also reported higher BUN in acidosis in cattle and buffalo (Dirksen, 1970; Nauriyal and Baxi, 1981; Sandha and Choudhary, 1985) & in goat (Lal *et al.*, 1992). However, in peracute cases, Dirksen (1970) did not notice any rise in the BUN level.

As earlier reported by Telle and Preston (1971) the kidneys could develop insufficiency only after a time more than ten hours after the onset of acidosis as observed in the present study. This increase in BUN may be explained as a result of myocardial insufficiency, renal degeneration and subsequently decreased urea clearance by kidney (Randhawa *et al.*, 1981a). Dehydration, haemoconcentration, anuria and catabolism with body

Table – 9. Mean ± S.E. along with their C.V. % of different bio-chemical attributes of blood recorded periodically in both untreated (A) and treated (B) groups of acidotic goats.

Hrs.	BUN (mg/dl)		BLA (mg/dl)		B. Bi. (mEq/L)	
	Group A	Group B	Group A	Group B	Group A	Group B
0	14.85^{a}±0.200 (3.314)	14.82^{a}±0.138 (2.290)	9.19^{a}±0.099 (2.656)	9.15^{a}±0.018 (0.488)	22.54^{a}±0.230 (2.522)	22.78^{a}±0.223 (2.400)
12	21.36^{b}±0.384 (10.175)	19.28^{b}±0.748 (23.929)	21.08^{b}±0.364 (9.772)	22.02^{be}±0.387 (10.837)	16.72^{b}±0.322 (4.724)	17.12^{b}±0.652 (23.494)
24	27.16^{c}±0.310 (6.468)	27.10^{c}±0.544 (12.385)	35.75^{c}±0.249 (3.950)	37.16^{c}±0.665 (11.033)	14.80^{c}±0.342 (5.666)	13.98^{c}±0.291 (12.841)
48	36.95^{d}±0.367 (5.622)	25.60^{c}±0.515 (12.411)	40.05^{d}±0.362 (5.120)	25.80^{b}±0.825 (19.715)	7.20^{d}±0.740 (22.989)	10.16^{d}±0.331 (20.088)
72	38.90^{d}±0.186 (2.708)	19.63^{b}±0.492 (15.471)	34.10^{c}±0.345 (5.724)	18.90^{de}±0.839 (27.374)	8.86^{e}±0.150 (2.934)	12.62^{cd}±0.284 (13.908)
96	29.20^{c}±0.223 (4.327)	15.72^{ab}±0.691 (27.113)	27.58^{e}±0.345 (7.077)	14.20^{df}±0.753 (32.731)	10.60^{f}±0.288 (4.716)	16.94^{bc}±0.434 (15.800)
120	26.68^{c}±0.350 (7.422)	14.92^{a}±0.469 (19.416)	19.98^{b}±0.355 (10.060)	9.66^{af}±0.278 (17.790)	15.20^{c}±0.254 (2.894)	21.34^{a}±0.514 (14.857)

Note: - 1. Means (column wise) superscripted with different letters were significantly different from each other.
2. Figure in parentheses indicate C.V.%.

Table – 10. Analysis of variance of periodic variation in different bio-chemical attributes of blood of untreated (A) and treated (B) groups of acidotic goats.

Parameter	Group	S.V.	D.F.	M.S.	F
BUN	A	Between hours	6	334.112	118.011**
		Error	25	2.831	
	B	Between hours	6	139.895	12.511**
		Error	31	11.188	
BLA	A	Between hours	6	618.065	215.695**
		Error	25	2.865	
	B	Between hours	6	562.211	41.366**
		Error	31	13.591	
B.Bi.	A	Between hours	6	137.822	168.204**
		Error	25	0.819	
	B	Between hours	6	111.865	17.660**
		Error	31	6.334	

** Significant (P≤0.01)

toxaemia may increased the BUN (Coles, 1974). If the kidney is malfunctioning or if the glomerular filteration rate is reduced because of extra-renal factors such as dehydration or shock then the rate of urea excretion will fall and the circulating level of urea will rise and also an increased rate of protein catabolism will also raise the BUN level (Hall, 1983). One of the consequences of rumen acidosis included reduced salivation (Slyter, 1976) this caused failure of urea recycling process. The increased level of blood urea in indigestion might be due to failure of urea recycling process through salivery glands and its un-utilization by microbes in the rumen (Singh *et al.*, 1989). Singh *et al.* (1983) also reported that rise in the blood urea was an index of less utilization of ammonia produced by the rumen microbes for the synthesis of their own cellular proteins. Increase in BUN level may also be due to reduction in the effective renal blood flow and fall in the arterial blood pressure which resulted in subnormal renal function (Dunlop and Hammond, 1965).

4.5.2. Blood lactic acid :

On perusal of table-9 (group A) and fig.-10 (group A) it was observed that the average concentration of lactic acid in blood gradually increased significantly ($P \leq 0.01$) from 9.19 ± 0.099 mg/dl at '0' hours to a maximum concentration of 40.05 ± 0.362 mg/dl at 48 hours of induction of acidosis. Thereafter, it decreased, but these values were remained significantly higher than the '0' hours value till the experiment ended. The rise in lactic acid concentration in blood during rumen acidosis was also previously noted in goats (Vihan and Rai ,1985;Cao *et al.* 1987; Lal *et al.* 1992 & Sen *et al.* 1993), sheep (Shinosaki and Nakabayasi, 1974 & Angelov *et al.*,1996), buffalo (Sandha and Choudhary, 1985 and Sinha *et al.* 1985), cattle (Dunlop

and Hammond, 1965, Ahrens, 1967 & Dirksen, 1967) & calves (Shukla *et al.*, 1999 & Dwivedi *et al.*, 2000). The rise in level of lactic acid in blood might be due to excessive fermentation of wheat grains in the rumen and its subsequent absorption through damaged ruminal wall (Dunlop, 1972; Slyter, 1976). Lowered capacity of liver to metabolise the D-isomer of lactic acid may also contribute to higher blood lactic acid (Dunlop, 1972). It may be mentioned that lactic acid get absorbed more rapidly at low pH (Williams and Mackenzie, 1965) and decrease in perfusion pressure and oxygen supply to peripheral tissues due to dehydration may also resulted in further increase in lactic acid from cellular respiration (Blood *et al.*, 1983). The blood levels were maintained subsequently higher by constant absorption from the rumen and inability of liver to convert lactic acid into glucose and also due to decreased tissue oxidation of lactic acid (Juhas'z and Szegedi, 1968a). Gradual decrease in the level of lactic acid in blood from 48 hours might be due to less production and absorption of lactic acid from rumen and increased rate of metabolism and elimination of lactic acid from the tissue. At very lower pH the rumen became static, which inhibits absorption of lactic acid. It appears that the peak entry of lactate into the circulation occurs in the early phases of disease (Smith, 1990).

4.5.3. Blood bicarbonate :

On perusal of table-9 (group A) and fig.-11 (group A) it was noticed that the bicarbonate level in blood showed a gradual fall from normal level of 22.54 ± 0.23 mEq/L at '0' hours to a significant ($P \leq 0.01$) level of 7.20 ± 0.740mEq/L at 48 hours of post-induction of acidosis. Thereafter, this value showed increasing trend, but remained significantly ($P \leq 0.01$) lower than, the '0' hours value till the experiment ended. This finding coincide with the

observation recorded by Dougherty *et al.* (1975), Sen *et al.* (1993), Angelov *et al.* (1996) and Shukla *et al.* (1999). The decrease in bicarbonate level in blood might be due to absorption of lactate from the rumen, decreases the dept. of HCO_3 (Howard, 1981) or the regulatory mechanism of blood gas homeostasis could neutralize a considerable part of the acid products and maintained the blood pH within the normal range. H^+, released during the ionisation of lactate in blood, combined with HCO_3 to form H_2CO_3, that dissociated to water and carbondioxide which is removed in the expired air (Dunlop, 1972).

4.6. Analysis of blood serum :

The mean along with their standard error (S.E.) and cofficient of variation percentage (C.V.%) of different bio-chemical attributes of serum of untreated acidotic goats (group A) at different interval of study has been presented in table-11 (group A) the results of analysis of variance of different bio-chemical attributes of serum between hours are also shown in table-12 (group A).

4.6.1. Total serum protein :

On perusal of table-11 (group A) and fig.-12 (group A) it was observed that the average concentration of serum total protein was 6.40 ± 0.138 gm/dl at '0' hours which gradually increased to the highest significant ($P \leq 0.01$) level of 7.20 ± 0.042 gm/dl at 48 hours. Thereafter, it gradually declined below '0' hours value. The total serum protein in this study showed that at first there was rise in serum total protein and afterwards it declined below the base level. This hyperproteinaemia in acid indigestion was also previously reported by Cao *et al.* (1987) in goats, Juhas'z and Szegedi (1968b) in sheep and Mullen (1976) in Cattles. However, Vihan *et al.* (1982)

Table – 11. Mean ± S.E. along with their C.V. % of different biochemical attributes of serum recorded periodically in both untreated (A) and treated (B) groups of acidotic goats.

Hrs.	TSP (gm/dl)		SGOT/AST (RFU/ml)		SGPT/ALT (RFU/ml)	
	Group A	Group B	Group A	Group B	Group A	Group B
0	6.40^{ab}±0.138 (5.284)	6.39^{a}±0.028 (1.093)	71.20^{a}±0.276 (0.925)	70.60^{a}±0.124 (0.430)	20.64^{a}±0.366 (4.352)	21.20^{a}±0.245 (2.834)
12	6.45^{a}±0.053 (2.049)	6.42^{a}±0.012 (0.462)	175.33^{b}±0.763 (2.462)	170.00^{b}±0.747 (2.708)	27.60^{b}±0.712 (14.597)	25.86^{b}±0.709 (16.914)
24	6.55^{a}±0.065 (2.467)	6.55^{b}±0.083 (3.140)	91.60^{c}±0.718 (4.439)	92.50^{c}±0.791 (5.274)	36.82^{cc}±0.965 (14.838)	37.52^{c}±0.615 (10.108)
48	7.20^{c}±0.042 (1.317)	7.00^{c}±0.028 (0.925)	81.66^{d}±1.104 (7.652)	86.66^{d}±0.980 (6.972)	40.10^{c}±0.927 (13.080)	39.16^{c}±1.067 (16.806)
72	6.55^{a}±0.017 (0.458)	6.35^{a}±0.040 (1.421)	79.00^{d}±0.883 (6.329)	75.60^{a}±0.632 (5.153)	59.26^{d}±1.209 (11.542)	44.32^{d}±0.617 (8.595)
96	6.20^{bd}±0.011 (0.322)	6.38^{a}±0.050 (1.759)	76.33^{ad}±0.973 (7.215)	73.80^{a}±0.683 (5.712)	52.34^{d}±1.025 (11.084)	31.54^{e}±0.595 (11.631)
120	6.00^{d}±0.069 (2.000)	6.40^{a}±0.063 (2.209)	74.66^{ad}±0.830 (6.295)	72.40^{a}±0.800 (6.816)	33.32^{e}±0.573 (9.731)	23.46^{ab}±0.517 (13.607)

Note: - 1. Means (column wise) superscripted with different letters were significantly different from each other.
2. Figure in parentheses indicate C.V.%.

Table – 12. Analysis of variance of periodic variation in different biochemical attributes of serum in both untreated (A) and treated (B) groups of acidotic goats.

Parameter	Group	S.V.	D.F.	M.S.	F
TSP	A	Between hours	6	0.592	17.272**
		Error	25	0.034	
	B	Between hours	6	0.268	19.790**
		Error	31	0.013	
SGOT/ AST	A	Between hours	6	7648.655	390.859**
		Error	25	19.568	
	B	Between hours	6	7377.430	380.658**
		Error	31	19.380	
SGPT/ ALT	A	Between hours	6	728.561	34.601**
		Error	25	21.056	
	B	Between hours	6	413.829	25.901**
		Error	31	15.977	

** Significant (P≤0.01)

Table – 11. Mean ± S.E. along with their C.V. % of different biochemical attributes of serum recorded periodically in both untreated (A) and treated (B) groups of acidotic goats.

Hrs.	TSP (gm/dl)		SGOT/AST (RFU/ml)		SGPT/ALT (RFU/ml)	
	Group A	Group B	Group A	Group B	Group A	Group B
0	6.40^{ab}±0.138 (5.284)	6.39^{a}±0.028 (1.093)	71.20^{a}±0.276 (0.925)	70.60^{a}±0.124 (0.430)	20.64^{a}±0.366 (4.352)	21.20^{a}±0.245 (2.834)
12	6.45^{a}±0.053 (2.049)	6.42^{a}±0.012 (0.462)	175.33^{b}±0.763 (2.462)	170.00^{b}±0.747 (2.708)	27.60^{b}±0.712 (14.597)	25.86^{b}±0.709 (16.914)
24	6.55^{a}±0.065 (2.467)	6.55^{b}±0.083 (3.140)	91.60^{c}±0.718 (4.439)	92.50^{c}±0.791 (5.274)	36.82^{ce}±0.965 (14.838)	37.52^{c}±0.615 (10.108)
48	7.20^{c}±0.042 (1.317)	7.00^{c}±0.028 (0.925)	81.66^{d}±1.104 (7.652)	86.66^{d}±0.980 (6.972)	40.10^{c}±0.927 (13.080)	39.16^{c}±1.067 (16.806)
72	6.55^{a}±0.017 (0.458)	6.35^{a}±0.040 (1.421)	79.00^{d}±0.883 (6.329)	75.60^{a}±0.632 (5.153)	59.26^{d}±1.209 (11.542)	44.32^{d}±0.617 (8.595)
96	6.20^{bd}±0.011 (0.322)	6.38^{a}±0.050 (1.759)	76.33^{ad}±0.973 (7.215)	73.80^{a}±0.683 (5.712)	52.34^{d}±1.025 (11.084)	31.54^{e}±0.595 (11.631)
120	6.00^{d}±0.069 (2.000)	6.40^{a}±0.063 (2.209)	74.66^{ad}±0.830 (6.295)	72.40^{a}±0.800 (6.816)	33.32^{e}±0.573 (9.731)	23.46^{ab}±0.517 (13.607)

Note: - 1. Means (column wise) superscripted with different letters were significantly different from each other.
2. Figure in parentheses indicate C.V.%.

Table – 12. Analysis of variance of periodic variation in different biochemical attributes of serum in both untreated (A) and treated (B) groups of acidotic goats.

Parameter	Group	S.V.	D.F.	M.S.	F
TSP	A	Between hours	6	0.592	17.272**
		Error	25	0.034	
	B	Between hours	6	0.268	19.790**
		Error	31	0.013	
SGOT/ AST	A	Between hours	6	7648.655	390.859**
		Error	25	19.568	
	B	Between hours	6	7377.430	380.658**
		Error	31	19.380	
SGPT/ ALT	A	Between hours	6	728.561	34.601**
		Error	25	21.056	
	B	Between hours	6	413.829	25.901**
		Error	31	15.977	

** Significant (P≤0.01)

in goats and Nauriyal and Baxi (1978) in cattle and buffaloes found no change in total serum proteins in rumen acidosis. The increase in concentration of total serum protein might be as a result of haemoconcentration due to dehydration (Mullen, 1976; Anderson, 1980). Water deprivation might results in an increase in total plasma protein (Khan *et al.*, 1978). The decrease in total serum protein concentration as observed from 72 hours in this study might be due to liver dysfunction which occurred in rumen acidosis.

4.6.2. SGOT/AST :

On perusal of table-11 (group A) and fig.-13 (group A) it was observed that the average concentration of AST in serum increased significantly ($P \leq 0.01$) from 71.20 ± 0.276 RFU/ml at '0' hours to a maximum concentration of 175.33 ± 0.763 RFU/ml at 12 hours of induction of acidosis. Further, this values showed decreasing trend, but remained significantly higher than the '0' hours value upto 72 hours. Thereafter from 96 hours, to the end of experiment non-significant elevation was recorded. The rise in concentration of AST was previously reported by Cakala *et al.* (1974) in acidotic sheep, Nauriyal and Baxi (1978) in buffalo, Mullen (1973) and Bieniek (1982) in cattle. However, Vihan *et al.* (1973) in buffalo and Dwivedi *et al.* (2000) in calves did not noticed any change in AST activity in indigestion. The rise in AST activity may be due to hepato-cellular damage (Mullen, 1973). Toxic products such as histamine, tyramine, alcohol, thiaminase and endotoxin like products, might also be produced, absorbed and cause hepatic damage (Vestweber *et al.*, 1974). The non-significant elevation in AST at 96 hours onwards might be due to effect of enhanced blood lactic acid concentration on liver function (Dwivedi *et al.*, 2000).

4.6.3. ALT/SGPT :

On perusal of table-11 (group A) and fig.-14 (group A) it was noticed that the level of ALT increased gradually from 20.64 ± 0.366 RFU/ml at '0' hours to a significant ($P \leq 0.01$) level of 59.26 ± 1.209 RFU/ml at 72 hours and then showed declining tendency towards normalcy but, the values remained significantly ($P \leq 0.01$) elevated throughout the period of observation. A similar trend was reported by Sethuraman and Rathor (1979b) in bovines, Randhawa *et al.* (1981a) in buffalo calves and Das and Misra (1992) in goats. However, Dwivedi *et al.* (2000) in calves noted almost unaltered activity of ALT. The increase in transaminase level might be due to hepatic necrosis or altered membrane permeability of hepatic cells (Sethuraman & Rathor, 1979b) or due to hyper acidity, which broke down the integrity of rumen epithelium and permitted bacterial and fungal entry into portal circulation, resulting into liver damage and liver abscesses (Blood *et al.*, 1983).

4.7. Experimental therapeutic management :

The adoption of treatment at the earliest possible time i.e. at the first appearance of clinical symptoms were recommended for faster and fuller recovery in both experimental and clinical cases of ruminal acidosis in cattle (Sethuramen and Rathor, 1979a; Sinha *et al.*, 1985). A suitable therapeutic management with conventional therapy [Sod. bicarbonate (7.5%w/v) solution I/v @ 4ml/kg b. wt. twice for 2 days, Normal saline (0.9%) solution 100-200 ml I/v once daily for 3 consecutive days, Sod. bicarbonate powder 5gms orally once daily for 3 days, B. complex (Belamyl) inj.- 2ml I/m daily for 5 days, Avilvet inj.- 1 ml I/m daily for 3 days, Tetracycline HCl powder- orally @ 20 mg/kg b. wt. as single dose on 1st days & Dexona inj. @ 1ml,

0.75 ml & 0.5 ml I/m on 1st, 2nd and 3rd days respectively] along with fresh rumen liquor @ 15ml/kg b. wt. orally twice daily for 3 days and some herbal preparations (Rumec powder 15gms orally twice daily for 3 days) were started at the onset of clinical symptoms & biochemical changes at 24 hours & continued till recovery or death taking into the considerations as intravenous injection of Sod. bicarbonate (7.5% w/v) might have helped to combat the loss of alkali reserve of the body while the oral administration neutralised the acidity of the rumen (Prasad and Rekib, 1975).

In ruminants rumen microbial population are responsible for vit.-B synthesis. As in acidosis, ruminal microflora are disturbed severely so the Vit.-B complex production may be hampered, and it was reported that thiamine HCl deficiency occurred following grain engorgement which is helpfull in absorption of lactic acid. Thiamine HCl was alone used by many workers (Patra *et al.*, 1997; Gupta *et al.*, 2000; Kemal Irmak *et al.*, 2000b) but there is paucity of litrature on the changes of riboflavin, niacin and cyanocobolamin. Riboflavin acts as an intermediate in biological oxidation-reduction system in mammals and this involves in all areas of mammalian metabolism as a co-factor, co-enzyme (FAD, riboflavin-5-phosphate) for generation of ATP (Keneko, 1989). There was no such findings to confirm the use of riboflavin in acidosis. Niacin is also essential for the regulation of pyridine dinucleotide system in cell and virtually it is essential for mammalian metabolism (Keneko, 1989). In that way vit-B complex (contains thiamine HCl, ribofavin, niacin and cyanocobolamine) may be helpful, but no litrature was available to correlate the finding.

In many rumen dysfunctions, abnormal changes occur in the rumen microbial population besides other complications and mere correction of

rumen pH may not be enough for the proper functioning of the recticulo-ruman and revival of appetite (Prasad, 1971). There fore it is essential to restore the activities of the rumen microbes and for this purpose transfaunation with cud transplantation have been recommended in the restoration of rumen environment and for normal fermentative processes (Gnanaprakasam, 1970; Sen, 1982; Sandha, 1980; Patra 1997). Under the transfaunation therapy fresh rumen liquor was also tried by many workers (Hungate, 1966; Hoflund, 1967; Prasad *et al.*, 1973; Karunanidhi *et al.*, 1985; Shukla *et al.*, 1999).

Besides these some herbal preparation was added with the prospective view of to induce some secretion of gastric enzymes for quick restoration of rumen functions. Although therapeutic trial with various herbal preparation have been previously performed by several workers (Tripathy and Mishra, 1992; Pradhan, 1995; Phalphale *et al.* 1997), but still there was a need to surch for another product which should be cost effective and eco-friendly and fore that Rumec powder, a herbal preparation marketed by Rakash pharmaceutical was used in the present study of rumen acidosis in goats.

The pharmacological actions of "Rumec powder" content ingredient viz *Zingiber Officinate, Cassia Fistula, Swertia Chirata, Trachyspermum Ammi, Aegle Marmelos, Copper Sulphate, Black Salt, Coriandrum Sativum, Curcuma Longa, Emblica Officinalis, Tinospora Cordifolia, Alpina Galanga, Carryo Phyllus Aromaticus, Sodium Chloride, Melia Azadirachta, Terminalia Chedula, Terminalia Belerica, Peucedanum Graveolens, Cinnamomum Tamala, Vernonia Anthelminitica, & Asafotida Compound* are not known, however as per company litrature it was recommended as digestive tonic and it helps in digestion & restoration of appetite probably by

stimulating gastric enzymes secretions and exerting beneficial effect in restoration of normal ruminal functions.

Anti-histaminic was given in present study with the thought of increased histamine concentration of rumen fluid. In acute lactic acidosis, symptom of laminitis was of severe degree and was related to progressive increase in histamine concentration of rumen liquor and blood (Randhawa *et al.*, 1988) in buffalo calves. Excessive production of endotoxin in the rumen due to destruction of gram negative bacteria might have also contributed to the release of endogenous histamine from body cells (Ahrens, 1967; Huber, 1976). Anti-histaminics were also noted by Irwin *et al.* (1979) to counteract the effects of toxic amines produced during acidosis.

Corticosteroid was used to overcome shock (Blood *et al.*, 1983) due to lactic acidosis.

Use of Tetracycline HCl probably checked the growth of gram positive bacteria and further production of lactic acid (Nakumara *et al.*, 1971) in rumen and for sterilization of rumen contents.

Fluid therapy was followed to check dehydration and to correct haemoconcentrations. Fluid therapy has been also recommended by many workers (Sethuraman, 1976, Sen, 1982; Tanwar & Mathur, 1983a; Blood and Radostits, 1989) during the treatment of acidosis.

The clinical and bio-chemical status of goats (group B) at various sampling hours before and after treatment was observed and evaluated under different tables.

As evident from table -1 (group B) symptomatic results observed may be explained under the following heads :

4.7.1. Dullness

Dullness was observed in 33 to 66 percent of animal between 12 and 24 hours of post-induction of acidosis. Thereafter following treatment 80, 40 and 20 percent of animal showed dullness at 48, 72 and 96 hours respectively. At 120 hours of observation, no sign of dullness was observed in any of the treated animals.

4.7.2. Weakness

Weakness was observed in 1 and 2 of the experimental animals at 12 and 24 hours of ruminal acidosis respectively. Thereafter, following therapy, 2 animals at 48 hours and 1 animal at 72 hours showed the sign of weakness. Further from 96 hours, no sign of weakness was observed in any of the treated animals.

4.7.3. Anorexia :

After the induction of acidosis all the animals were found in anorectic condition. But, following treatment 3,2 & 1 of the treated animals showed anorectic state at 48, 72 and 96 hours respectively. At 120 hours of observation, no sign of anorexia was observed in any of the treated animals.

4.7.4. Distended rumen :

About 33 to 66 percent of experimental animals showed distended rumen between 12 to 24 hours of induction of acidosis. Whereas following treatment, 60 to 20 percent of treated animals were found distended rumen between 48 to 72 hours. Further from 96 hours, none of the treated animals were found distended rumen.

4.7.5. Diarrhoea :

This prominent symptom was present in 33 to 50 percent of animals between 12 to 24 hours of induction of acidosis. Thereafter following treatment, 40 to 20% of animal showed diarrhoea between 48 to 96 hours. At

120 hours of observation, none of the treated animals showed sign of diarrhoea.

4.7.6. Constipation :

Following induction of acidosis about 50 to 33 percent of experimental animals showed the symptom of constipation between 12 to 24 hours. Thereafter, following treatment, no sign of constipation was observed in any of the experimental animals to the end of study.

4.7.7. Abdominal pain :

Abdominal pain was observed in 50 percent of animals upto 24 hours of induction of acidosis. Thereafter following treatment, 40 to 20 percent of animals remained to suffer from abdominal pain between 48 to 72 hours. Further from 96 hours, no sign of abdominal pain was observed to any one of the treated animals.

4.7.8. Nasal discharge :

Following induction of acidosis 33 percent of animal showed the symptom of nasal discharge at 24 hours of observation. Thereafter following treatment, 40 to 20 percent of surviving animal remained to show the symptom of nasal discharge between 48 to 72 hours. Further from 96 hour no sign of nasal discharge was observed in any of the treated animals.

4.7.9. Lateral recumbency :

1 and 2 animals between 12 to 24 hours of post-induction of acidosis were observed to be in lateral recumbency. But, following treatment, none of the treated animals were found to be in lateral recumbency except at 48 hours where only 1 animal was found to be in lateral recumbency.

4.7. 10. Standquitely :

2 to 4 animals were found disinclined to move and remained standing between 12 to 24 hours of induction of acidosis. Whereas, following treatment only 2 animals at 48 hours followed by 1 animal at 72 hours were found disinclined to move and remained in standing position. Further from 96 hours, none of the treated animals were found disinclined to move.

4.7.11. Lameness :

None of the experimental animals were found to suffer from lameness at any interval of observations following treatment.

4.7.12. Staggering gait :

Staggering gait was observed in 1 and 2 of the experimental animals between 12 to 24 hours of post-induction of acidosis. Thereafter following treatment, none of the treated animals showed the symptom of staggering gait except at 48 hours where only 1 animal was found to show staggering gait.

4.7.13. Apparent blindness :

Apparent blindness was noticed in 2 of the experimental animals upto 24 hours of induction of acidosis. Thereafter following treatment, none of the treated animals were found apparently blind except at 48 hours where only 1 animal found to show apparent blindness.

4.7.14. Pupil dilated :

Two of the experimental animals was found to show pupillary dilatation at 24 hours of induction of acidosis. Whereas only 1 animal remained to show pupillary dilatation upto 72 hours following therapy. Further from 96 hours, none of the treated animals showed dilated pupil till the experiment ended.

4.7.15. Grinding of teeth :

33 to 50 percent of animals showed grinding of teeth between 12 to 24 hours of induction of acidosis. Thereafter following treatment, 2 animals at 48 hours and 1 animal upto 96 hours remained to show grinding of teeth. At 120 hours of observation, none of the treated animals showed grinding of teeth.

4.7.16. Head pressing :

2 of the experimental animals showed pressing of head between 12 to 24 hours after the induction of acidosis. Thereafter following therapy, 1 of the animal was found continued to show pressing of head upto 72 hours. Further from 96 hours, none of the treated animals showed pressing of head.

4.7.17. Regurgitation of undigested wheat :

This symptom was recorded in 1 of the animal at 12 hours whereas 2 of the experimental animals suffered from regurgitation of undigested wheat grains at 24 hours after the induction of acidosis. Thereafter following therapy, none of the treated animals showed the symptoms of regurgitation of undigested wheat grains, except at 48 hours where only 1 animal showed this symptom.

4.7.18. Thirst :

Thirstness was observed in 50 percent of animal between 12 to 24 hours of post-induction of acidosis. Thereafter following treatment, 3,2 and 1 animal showed thirstiness at 48, 72 & 96 hours respectively. At 120 hours of observation, none of the treated animals found suffered from thirstiness.

4.6.19. Sunken eye :

One of the experimental animal developed the symptom of sunken eye at 24 hours after the induction of acidosis. Thereafter following therapy, 2 animals at 48 hours and 1 animal between 72 to 96 hours had sunken eye.

Further at 120 hours of experiment, no sign of sunken eye was observed in any of the treated animals.

4.7.20. Mortality :

In this study 1 of the experimental animal could not survive even after the therapy was followed and died within 48 hours of observation. Thereafter, 5 animals remained survived the period of experiment.

4.7.21. Clinical observations:

The mean alongwith their standard error (S.E.) and coefficient of variation percentage (C.V.%) of different clinical observations noted in treated acidotic goats (group B) at various intervals of time has been presented in table 2 and 4 (group B). The results of analysis of variance of different clinical observations noted between hours are also shown in table-3 and 5 (group B).

4.7.21.1. Pulse/min. :

As evident from table-2 (group B) and fig.-1 (group B) it was observed that the mean pulse rate increased from '0' hours level of 71.00 ± 0.516 to a significant level ($P \leq 0.01$) of 111.00 ± 1.100 at 24 hours following induction of acidosis. Thereafter following treatment, decreasing trend was observed and non-significant difference in pulse rate as compared to '0' hours value was observed at 120 hours of experiment.

4.7.21.2. Respiration rate / min. :

On perusal of table-2 (group B) and fig.-2 (group B) it was noticed that there was significant ($P \leq 0.01$) increase in respiration rate from 12 hours and the mean respiration at 24 hours was 37.66 ± 0.333. Following treatment, this value decreased towards normalcy and non-significant difference in respiration rate as compared to '0' hours value was observed from 96 hours till the experiment was over.

4.7.21.3. Rectal Temp. (^{0}F) :

On perusal of table-2 (group B) and fig.-3 (group B) it was observed that the mean rectal temp. increased from 102.60 ± 0.154 at '0' hours to a significant ($P \leq 0.01$) level of 103.60 ± 0.139 at 12 hours which decreased to the level of 103.50 ± 0.161 at 24 hours following the induction of acidosis. Thereafter following treatment, non-significant changes in temp. as compared to '0' hours value was observed from 96 hours till the experiment ended.

4.7.21.4. Rumen motility/5 min. :

As noted in table- 4 (group B) and fig-4 (group B) the motility of rumen was 6.66 ± 0.210 at '0' hours which decreased significantly ($P \leq 0.01$) to 1.66 ± 0.210 at 24 hours following induction of acidosis. Thereafter following treatment, this value increased towards normalcy and non-significant changes in rumen motility as compared to '0' hours value was found at 120 hours of experiment.

4.7.22. Examination of rumen liquor :

4.7.22.1. Macroscopic examination :

4.7.22.1.1. Smell :

As depicted in table-6 (group B) it was observed that the smell of rumen liquor was aromatic at '0' hours which changed from faintly sour to sour between 12 to 24 hours of induction of acidosis, which again turned towards normal from 96 hours where aromatic smell was found following the therapy.

4.7.22.1.2. Colour :

As depicted in table- 6 (group B) it was observed that the colour of rumen liquor in this study was greenish brown at '0' hours which changed to

light grey to greyish in colour between 12 to 24 hours following induction of acidosis. Thereafter following treatment, this greyish colour changed towards light grey to greenish brown in colour which was similar to '0' hours observation from 96 hours to the end of experiment.

4.7.22.1.3. Consistency :

As depicted in table-6 (group B) it was observed that the consistency of rumen liquor gradually reduced from viscous (+++) at '0' hours to slight viscous (++) at 12 and 24 hours after the induction of acidosis. Thereafter following the treatment, consistency of rumen liquor changed from watery (+) at 48 hours to slight viscous (++) at 72 hours. Further from 96 hours, consistency of rumen liquor was similar to '0' hours observation to the end of experiment.

4.7.22.2. Microscopic examination :

4.7.22.2.1. Motility of rumen Protozoa :

As depicted in table-4 (group B) it was observed that the motility of rumen protozoa was vigorous (4) at '0' hours which became slow (2) at 12 hours to absent (1) at 24 hours following induction of acidosis. Thereafter following treatment, motility of rumen protozoa improved to slow (2) at 48 hours and to moderate (3) at 72 hours. Further from 96 hours motility of protozoa was vigorous (4) and remained so to the end of experiment which was similar to '0' hours observation.

4.7.22.3. Biochemical changes in rumen liquor :

The mean along with their standard error (S.E.) and coefficient of variation percentage (C.V.%) of different biochemical attributes of rumen liquor of treated acidotic goats (group B) at different interval of study has

been presented in table-7 (group B). The results of analysis of variance of different biochemical attributes of rumen liquor between hours are also shown in table-8 (group B).

4.7.22.3.1. pH of rumen liquor : On perusal of table-6 (group B) and fig-6 (group B) it was observed that rumen pH decreased gradually from 6.92 ± 0.024 at '0' hours to a significant level ($P \leq 0.01$) of 4.90 ± 0.338 at 12 hours and to 4.55 ± 0.169 at 24 hours of post-induction of acidosis. Following treatment, this valve increased significantly ($P \leq 0.01$) to 5.30 ± 0.036 at 48 hours which later on from 96 hours non-significant difference was recorded as compared to '0' hours observation till the experiment ended.

4.7.22.3.2. Rumen lactic acid :

As evident from table-6 (group B) and fig.-7 (group B) it was observed that following induction of acidosis the concentration of lactic acid increased gradually from 3.87 ± 0.062 mg/dl at '0' hours to a significant level ($P \leq 0.01$) of 119.83 ± 1.672 mg/dl at 12 hours and further to 151.64 ± 1.713 mg/dl at 24 hours of induction of acidosis. Thereafter following treatment, this value showed declining trend towards normalcy which became non-significant at 120 hours as compared to '0' hours observation.

4.7.22.3.3. Total volatile fatty acid :

As evident from table-6 (group B) and fig.-8 (group B) it was noticed that the total volatile fatty acid value rose significantly ($P \leq 0.01$) to 115.99 ± 0.186 mEq/L as against '0' hours value of 66.30 ± 0.186 mEq/L at 12 hours which declined significantly ($P \leq 0.01$) to 58.64 ± 1.162 mEq/L at 24 hours of post-induction. Thereafter following treatment, this value gets further declined to 35.28 ± 0.827 mEq/L at 48 hours which was gradually increased from 72 hours towards normal value and non-significant difference was observed from 96 hours as compared to '0' hours observation.

4.7.23. Analysis of blood :

The mean along with their standard error (S.E.) and coefficient of variation percentage (C.V.%) of different biochemical attributes of blood of treated acidotic goats (group B) at different hours of study has been presented in table-9 (group B). The results of analysis of variance of different biochemical attributes of blood between hours are also shown in table-10 (group B).

4.7.23.1. Blood urea nitrogen :

As depicted in table-9 (group B) and fig.9 (group B) it was observed that the level of blood urea nitrogen at '0' hours was 14.82 mg/dl which gradually increased to a significant ($P \leq 0.01$) level of 19.28 ± 0.748 mg/dl at 12 hours and to 27.10 ± 0.544 mg/dl at 24 hours of post-induction of acidosis. Thereafter following treatment, the value get declined towards normalcy and non-significant difference was found from 96 hours as compared to '0' hours observation upto the end of experiment.

4.7.23.2. Blood lactic acid :

On perusal of table-9 (group B) and fig.-10 (group B) it was noticed that the average concentration of lactic acid in blood gradually rose significantally ($P \leq 0.01$) from 9.15 ± 0.01 mg/dl at '0' hours to 22.02 ± 0.387 at 12 hours and further to 37.16 ± 0.665 mg/dl at 24 hours of induction of acidosis. Thereafter following therapy, this value get declined towards normalcy and at 120 hours the value 9.66 ± 0.278 mg/dl of blood showed non-significant difference as compared to '0' hours value.

4.7.23.3. Blood bicarbonate :

On perusal of table-9 (group B) and fig.-11 (group B) it was noticed that the concentration of bicarbonate in blood decreased from 22.78 ± 0.223 mEq/L at '0' hours to a significant ($P \leq 0.01$) level of 17.12 ± 0.652 mEq/L at

12 hours and to 13.98 ± 0.291 mEq/L at 24 hours of induction of acidosis. Thereafter following treatment, this value remain decreased to 10.16 ± 0.331 mEq/L at 48 hours. But from 72 hours this value showed gradualy increasing trend towards normalcy and non-significant difference was observed at 120 hours of experiment in comparision to '0' hours value.

4.7.24. Analysis of blood serum :

The mean along with their standard error (S.E.) and coefficient of variation percentage (C.V.%) of different bio-chemical attributes of serum of treated acidotic goats (group B) at different interval of study has been presented in table-11 (group B). The results of analysis of variance of different bio-chemical attributes of serum between hours are also shown in table-12 (group B).

4.7.24.1. Total serum protein :

On perusal of table-11 (group B) and fig. 12 (group B) it was observed that the average concentration of serum total protein was 6.39 ± 0.028 gm/dl at '0' hours which was non-significantly ($P \leq 0.05$) increased to 6.42 gm/dl at 12 hours and significantly ($P \leq 0.05$) to 6.55 ± 0.083 gm/dl at 24 hours of induction of acidosis. Thereafter following treatment, this value was further increased to 7.00 ± 0.028 gm/dl at 48 hours which latter on showed declining trend towards normalcy and non-significant difference from 72 hours was noted as compared to '0' hours value.

4.7.24.2. SGOT/AST :

On perusal of table-11 (group B) and fig.13 (group B) it was observed that the average concentration of AST was 70.60 ± 0.124 RFU/ml at '0' hours which gradually rose to 170.00 ± 0.747 RFU/ml at 12 hours and to 92.50 RFU/ml at 24 hours of post-induction of acidosis. Thereafter following

therapy, this value showed declining trend towards normalcy which became non-significant from 72 hours as compared to '0' hours value.

4.7.24.3. SGPT/ALT :

On perusal of table-11 (group B) and fig. 14 (group B) it was observed that the average concentration of ALT was 21.20 ± 0.245 RFU/ml which increased significantly ($P \leq 0.01$) to 25.86 ± 0.709 RFU/ml at 12 hours and to 37.52 ± 0.615 RFU/ml at 24 hours of post-induction of acidosis. Thereafter following treatment, this value remained significantly higher to 39.16 ± 1.067 RFU/ml at 48 hours to and 44.32 ± 0.617 RFU/ml at 72 hours which lateron showed declining trend towards normalcy and non-significant difference was observed at 120 hours in comparison to '0' hours value.

Hence, on perusal of above clinico-biochemical changes observed at different hours of treatment, it was seen that following therapy all the clinical signs and bio-chemical status of goats clearly indicated ameliorative effect of the treatment and almost all the alterations occurred in rumen liquor, blood and serum returned to '0' hour value i.e. to normal value at a rapid pace. These findings get conformity with Lal (1988) who reported recovery based on bio-chemical studies like that of lactic acid and blood urea nitrogen in experimental acidosis in goats. Nauriyal and Baxi (1981) reported the recovery of total plasma protein and blood urea nitrogen following therapy in acidotic buffalo calves. Shukla *et al.* (1999) who also reported almost normal return of serum bicarbonate, serum lactic acid and rumen pH following treatment in bovine calves.

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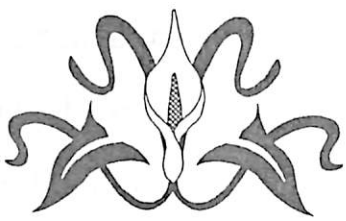
CHAPTER - V



SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSIONS

The present investigation was undertaken to study the clinical manifestations, systemic effects of ruminal acidosis and its clinical management.

The present study was conducted on twelve healthy goats, which were kept under identical managemental conditions & were divided into two groups (A and B) consisting of six animals in each group. Whole wheat grains were given at a time to both the groups of animals orally @ 100gm/kg b. wt. after 24 hours of starvation and during fasting only fresh water ad lib was provided. The animals of group A were kept as untreated acidotic control to study the clinical signs and bio-chemical changes in rumen liquor, blood & serum, whereas in group B all the above parameters including therapeutic responses were studied before ('0' hours) and after at 12, 24, 48, 72, 96 and 120 hours of induction of acidosis to know the systemic response of these animals to ruminal acidosis and success of therapy.

Following wheat grains feeding to animals of group A, the clinical manifestations observed were dullness, weakness, anorexia, distended rumen, diarrhoea, constipation, abdominal pain, lateral recumbence, stand quietly, staggering gait, apparent blindness, grinding of teeth, head pressing, regurgitation of undigested wheat & thirst at 12 hours of induction of acidosis. Later on the severity in above symptom increased with addition signs of nasal discharge, lameness, dilated pupil and sunken eye between 24 to 48 hours of post-feeding. Thereafter, the symptoms gradually returned towards normalcy but remained apparent throughout the period of study. Three goats, (one within 48 hours and another two within 72 hours) in this

group died, while 3 goats over come the acute phase of lactic acidosis with very slow rate of recovery in the survivors.

In clinical observations it was found that pulse and respiration rates remained significantly ($P \leq 0.01$) elevated, but rectal temp. decreased mildly from 24 hours which became normal as disease advanced. Rumen motility remained significantly ($P \leq 0.01$) decreased from 12 hours to the end of experiment. Skin inelasticity time increased with increase in severity of symptom which after 72 hours, followed decreasing trend after an initial rise at 96 hours towards normalcy as experiment ended.

The physico-microbial and bio-chemical status of rumen liquor showed noticeable changes. The colour of rumen liquor changed from greenish brown to light grey at 12 hours and to greyish in colour from 24 to 96 hours, which again returned towards normalcy showing light grey colour at 120 hours of observation. The odour of sourness increased with the severity of disease, which became intense sour between 48 to 72 hours & thereafter it turned towards normalcy, but failed to reach normalcy even at 120 hours of study. Consistency of rumen liquor was lost due to advancement of lactic acidosis. On microscopic examination of rumen liquor, the protozoal motility was found reduced to absent from 24 to 72 hours & thereafter, reappearance of protozoa in very low concentration from 96 hours was observed. The pH of rumen liquor fell drastically, following wheat grains feeding form 12 hours, which remained significantly ($P \leq 0.01$) low through out the study, although it showed increasing trend slightly from 48 hours towards normalcy but failed to show normalcy even at 120 hours of observation. The rumen lactic acid concentration increased many folds right from 12 hours of grains feeding which remained increasing upto 24 hours &

thereafter, it showed declining trend and reached to statistically normal level at 120 hours of study but the level remained higher then that of 0 hour value. The TVFA concentration in rumen liquor decreased significantly ($P \leq 0.01$) from 24 hours after an initial rise at 12 hours and remained decreasing upto 48 hours, thereafter, increasing trend towards normalcy was found but the value remained significantly ($P \leq 0.01$) low even at 120 hours of experiment.

Blood bio-chemical picture showed increased lactic acid concentration from 12 hours, which again showed decreasing trend from 72 hours towards normalcy but remained significantly ($P \leq 0.01$) elevated upto the end of experiment. The level of BUN showed significant ($P \leq 0.01$) increase from 12 hours and remained elevated till the observation ended. Blood bicarbonate level showed decreasing trend from 12 hours of grains feeding and remained so upto 48 hours, thereafter, increasing trend towards normalcy was found but the value could not reach to normal level even at 120 hours of observation.

Blood serum exhibited increased total serum protein from 12 hours, which get decreased towards normalcy from 48 hours but remained significantly ($P \leq 0.01$) elevated by the end of 120 hours of feeding. Significant increase in SGOT/AST activities in serum of acidotic goat were observed at 12 hours, which later on showed declining trend but could not returned to normalcy even at 120 hours of study. SGPT/ALT activities in serum increased gradually upto 72 hours & thereafter, the activities decreased to normalcy by 120 hours of observation.

A suitable therapeutic management with conventional therapy [Sod. bicarbonate (7.5%w/v) solution I/v @ 4ml/kg b. wt. twice for 2 days, Normal saline (0.9%) solution 100-200 ml I/v once daily for 3 consecutive

days, Sod. bicarbonate powder 5gms orally once daily for 3 days, B-complex (Belamyl) inj.- 2ml I/m daily for 5 days, Avilvet inj.- 1 ml I/m daily for 3 days, Tetracycline HCl powder-orally @ 20 mg/kg b. wt. as single dose on 1st days & Dexona inj. @ 1ml, 0.75 ml & 0.5 ml I/m on 1st, 2nd and 3rd days respectively] along with fresh rumen liquor @ 15ml/kg b. wt. orally twice daily for 3 days and some herbal preparations (Rumec powder, 15gms orally twice daily for 3 days) were started at the onset of clinical symptoms & biochemical changes at 24 hours & continued till recovery or death.

Following grains feeding, almost similar types of severity in clinical and bio-chemical status were observed in animals of group B as compared to animals of group A upto 24 hours of post-feeding. Thereafter, following treatment from 24 hours the severity in clinical and bio-chemical status of goats showed gradual returned towards complete and quicker recovery. However, one animal in this group had died within 48 hours, while 5 animals survived showing the ameliorative effect of treatment.

The signs of constipation, lateral recumbence, lameness, staggering gait, apparent blindness, regurgitation of undigested wheat disappeared as early as 48 hours, while weakness, distended rumen, abdominal pain, nasal discharge, dilated pupil & head pressing disappeared by 96 hours. The signs of dullness, anorexia, diarrhoea, grinding of teeth, thirst and sunken eye disappeared by 120 hours of observation.

The respiration & pulse rates returned to normal by 96 hours and 120 hours respectively whereas rectal temp. remained unaffected.

Smell, colour, consistency and protozoal motility of rumen liquor showed aromatic smell, greenish brown colour, viscous (+++) consistency and vigorous motility respectively by the end of experiment. The value of

rumen pH (6.98 ±0.038), rumen lactic acid (4.26 ± 0.168) & TVFA (66.20 ± 0.912) statistically returned to normal value by the end of observation.

The level of blood lactic acid (9.66 ± 0.278), BUN (14.92 ± 0.469) and bicarbonate (21.34 ± 0.514) statistically showed normal level by the end of observation.

The level of total serum protein (6.40 ± 0.063), activities of AST (72.40 ± 0.800) and ALT (23.46 ± 0.517) also returned to statistically normal levels by the end of observation.

Therefore, on the basis of clinical and bio-chemical status of untreated and treated groups of acidotic goats at various sampling hours the observation of investigations can be summarised and conclusion can be drawn as follows :

(i) Experimental ruminal acidosis can successfully be produced in goats by oral feeding with whole-wheat grains @ 100 gm/kg b.wt. after 24 hours of fasting.

(ii) The clinical signs and observations in lactic acidotic goat may be characterised by dullness, weakness, anorexia, distended rumen, diarrhoea, constipation, dilated pupil, grinding of teeth, pressing of head, regurgitation of undigested wheat, thirst, shunken eye, increased skin inelasticity time, abdominal pain, nasal discharge, lateral recumbency, stand quitely, lameness, staggering gait, apparent blindness, increased respiration and pulse rates, decreased or absent rumen motility and death.

(iii) There are characterstics alterations in physico-microbial & biochemical observation of rumen liquor like colour, consistency, odour, concentration of rumen microbes, concentration of TVFA, lactic acid and pH.

(iv) Marked alterations of blood bio-chemical observations like concentration of lactic acid, bicarbonate, blood urea nitrogen are observed.

(v) Marked alterations of serum AST and ALT activity and total serum protein level are observed.

(vi) In ruminal lactic acidosis in goat therapeutic attention is needed immediately, otherwise animal succumb to death.

(vii) The therapeutic measures adopted in experimental ruminal acidosis have been most successful and indicates better & faster recovery.

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"Clinico-Biochemical and Therapeutic Studies of Experimental Ruminal Acidosis in Goats".

ABSTRACT

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BY

ABHISHEK KUMAR

DEPARTMENT OF VETERINARY MEDICINE

BIHAR VETERINARY COLLEGE

P A T N A - 800014, BIHAR (INDIA)

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ABSTRACT

Clinico-biochemical and therapeutic studies of experimental ruminal acidosis in goats were carried out in the Department of Vety. Medicine, Bihar Veterinary College, Patna-14 with the aim –

(i) To induce experimental ruminal acidosis in goats by oral feeding with whole wheat grains.

(ii) To study the course of ruminal acidosis thus produced.

(iii) To study some of the various bio-chemical alterations in ruminal fluid, blood and serum of acidotic goats.

(iv) To evolve a suitable therapeutic management for acidotic goats.

The investigations were conducted on twelve healthy goats which were kept under identical managemental condition and were divided into two groups (A and B) consisting of six animals each. Whole wheat grains were given at a time to both the groups of animals orally @ 100gm/kg b.wt. after 24 hours of starvation & during fasting only *adlib* fresh water was provided. The animals of group A were kept as untreated acidotic control to study the clinical signs & bio-chemical changes in rumen fluid, blood and serum, whereas, in animals of group B, all the above parameters including therapeutic responses were studied before fasting ('0' hours) and after at 12, 24, 48, 72, 96 and 120 hours of wheat feeding to know the systemic responses of these animals to ruminal acidosis and effectiveness of therapy. After the induction of acidosis, feed and water were given to all the goats if they take during the entire period of study.

The clinical signs (resp., pulse, temp. & rumen motility) were recorded as per method described by Rosenberger *et al.* (1979), the physico-

microbial changes in rumen liquor was noted immediately after the collection, the pH of rumen liquor was recorded with the help of portable grip pH meter (systronic GRIpH-pH meter), the TVFA concentration in rumen liquor was estimated as per the method of Barnett and Reid (1957), the concentration of lactic acid in rumen liquor and blood was estimated as per method of Barker and Summerson (1941), the concentration of BUN in blood was estimated as per method of Netalson (1957), the level of blood bicarbonate was estimated by method of Van Slyke (1922), the activities of transaminase (SGPT & SGOT) was estimated by the method of Reitman and Frankel (1957) and the concentration of total serum protein was estimated by using Biuret method.

Following wheat grains feeding to animals of group A, the clinical manifestations observed were dullness, weakness, anorexia, distended rumen, diarrhoea, constipation, abdominal pain, lateral recumbency, stand quietly, staggering gait, apparent blindness, grinding of teeth, head pressing, regurgitation of undigested wheat & thirst at 12 hours of induction of acidosis. Later on the severity in above symptom increased with additional signs of nasal discharge, lameness, dilated pupil and sunken eye between 24 to 48 hours of post-feeding. Thereafter, the symptoms gradually returned towards normalcy but remained apparent throughout the period of study. Three goats, (one within 48 hours and another two within 72 hours) in this group died, while 3 goats over come the acute phase of lactic acidosis with very slow rate of recovery in the survivors.

In clinical observations it was found that pulse and respiration rates remained significantly ($P \leq 0.01$) elevated, but rectal temp. decreased mildly from 24 hours which became normal as disease advanced. Rumen motility

remained significantly ($P \leq 0.01$) decreased from 12 hours to the end of experiment. Skin inelasticity time increased with increase in severity of symptom which after 72 hours, followed decreasing trend after an initial rise at 96 hours towards normalcy as experiment ended.

The physico-microbial and bio-chemical status of rumen liquor showed noticeable changes. The colour of rumen liquor changed from greenish brown to light grey at 12 hours and to greyish in colour from 24 to 96 hours which again returned towards normalcy showing light grey colour at 120 hours of observation. The odour of sourness increased with the severity of disease, which became intense sour between 48 to 72 hours & thereafter it turned towards normalcy, but failed to reach normalcy even at 120 hours of study. Consistency of rumen liquor was lost due to advancement of lactic acidosis. On microscopic examination of rumen liquor, the protozoal motility was found reduced to absent from 24 to 72 hours & thereafter, reappearance of protozoa in very low concentration from 96 hours was observed. The pH of rumen liquor fell drastically, following wheat grains feeding from 12 hours which remained significantly ($P \leq 0.01$) low throughout the study, although it showed increasing trend slightly from 48 hours towards normalcy but failed to show normalcy even at 120 hours of observation. The rumen lactic acid concentration increased many folds right from 12 hours of grains feeding which remained increasing upto 24 hours & thereafter, it showed declining trend and reached to statistically normal level at 120 hours of study but the level remained higher than that of 0 hour value. The TVFA concentration in rumen liquor decreased significantly ($P \leq 0.01$) from 24 hours after an initial rise at 12 hours and remained decreasing upto

48 hours, thereafter, increasing trend towards normalcy was found but the value remained significantly ($P \leq 0.01$) low even at 120 hours of experiment.

Blood bio-chemical picture showed increased lactic acid concentration from 12 hours which again showed decreasing trend from 72 hours towards normalcy but remained significantly ($P \leq 0.01$) elevated upto the end of experiment. The level of BUN showed significant ($P \leq 0.01$) increase from 12 hours and remained elevated till the observation ended. Blood bicarbonate level showed decreasing trend from 12 hours of grains feeding and remained so upto 48 hours, thereafter, increasing trend towards normalcy was found but the value could not reach to normal level even at 120 hours of observation.

Blood serum exhibited increased total serum protein from 12 hours which get decreased towards normalcy from 48 hours but remained significantly ($P \leq 0.01$) elevated by the end of 120 hours of feeding. Significant increase in SGOT/AST activities in serum of acidotic goat were observed at 12 hours which later on showed declining trend but could not returned to normalcy even at 120 hours of study. SGPT/ALT activities in serum increased gradually upto 72 hours & thereafter, the activities decreased to normalcy by 120 hours of observation.

A suitable therapeutic management with conventional therapy [Sod. bicarbonate (7.5%w/v) solution I/V @ 4ml/kg b. wt. twice for 2 days, Normal saline (0.9%) solution 100-200 ml I/V once daily for 3 consecutive days, Sod. bicarbonate powder 5gms orally once daily for 3 days, B. complex (Belamyl) inj.- 2ml I/m daily for 5 days, Avilvet inj.- 1 ml I/m daily for 3 days, Tetracycline HCl powder-orally @ 20 mg/kg b. wt. as single dose on 1st days & Dexona inj. @ 1ml, 0.75 ml & 0.5 ml I/m on 1st, 2nd and 3rd

days respectively) along with fresh rumen liquor @ 15ml/kg b. wt. orally twice daily for 3 days and some herbal preparations (Rumec powder, 15gms orally twice daily for 3 days) were started at the onset of clinical symptoms&biochemical changes at 24 hours & continued till recovery or death.

Following grains feeding, almost similar types of severity in clinical and bio-chemical status were observed in animals of group B as compared to animals of group A upto 24 hours of post-feeding. Thereafter, following treatment from 24 hours the severity in clinical and bio-chemical status of goats showed gradual returned towards complete and quicker recovery. However, one animal in this group had died within 48 hours, while 5 animals survived showing the ameliorative effect of treatment.

The signs of constipation, lateral recumbency, lameness, staggering gait, apparent blindness, regurgitation of undigested wheat disappeared as early as 48 hours, while weakness, distended rumen, abdominal pain, nasal discharge, dilated pupil & head pressing disappeared by 96 hours. The signs of dullness, anorexia, diarrhoea, grinding of teeth, thirst, sunken eye disappeared by 120 hours of observation.

The respiration & pulse rates returned to normal by 96 hours and 120 hours respectively whereas rectal temp. remained unaffected.

Smell, colour, consistency and protozoal motility of rumen liquor showed aromatic smell, greenish brown colour, viscous (+++) consistency and vigorous motility respectively by the end of experiment. The value of rumen pH (6.98 $\pm$0.038), rumen lactic acid (4.26 $\pm$ 0.168) & TVFA (66.20 $\pm$ 0.912) statistically returned to normal value by the end of observation.

The level of blood lactic acid (9.66 ± 0.278), BUN (14.92 ± 0.469) and bicarbonate (21.34 ± 0.514) statistically showed normal level by the end of observation.

The level of total serum protein (6.40 ± 0.063), activities of AST (72.40 ± 0.800) and ALT (23.46 ± 0.517) also returned to statistically normal levels by the end of observation.

Therefore, on the basis of clinical recovery and restoration of bio-chemical parameters it can be summarized that the goats of group B not only had better recovery rate but also restored biochemical alterations, towards normalcy at a rapid pace.

Finally, it was concluded that oral feeding of whole wheat grains @ 100 gm/kg b. wt. can produce ruminal acidosis and it has shown its impact on different physiological and bio-chemical status of goats. The adoption of treatment at earliest possible time (i.e. at first appearance of clinical signs) with conventional therapy along with fresh rumen liquor and some herbal preparations (Rumec powder) in the management of ruminal acidosis in goats, the therapy was most successful and best approach in the management of acidosis in goats.

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Photograph of healthy goats of both the groups.

Photograph showing, recording of rumen motility.

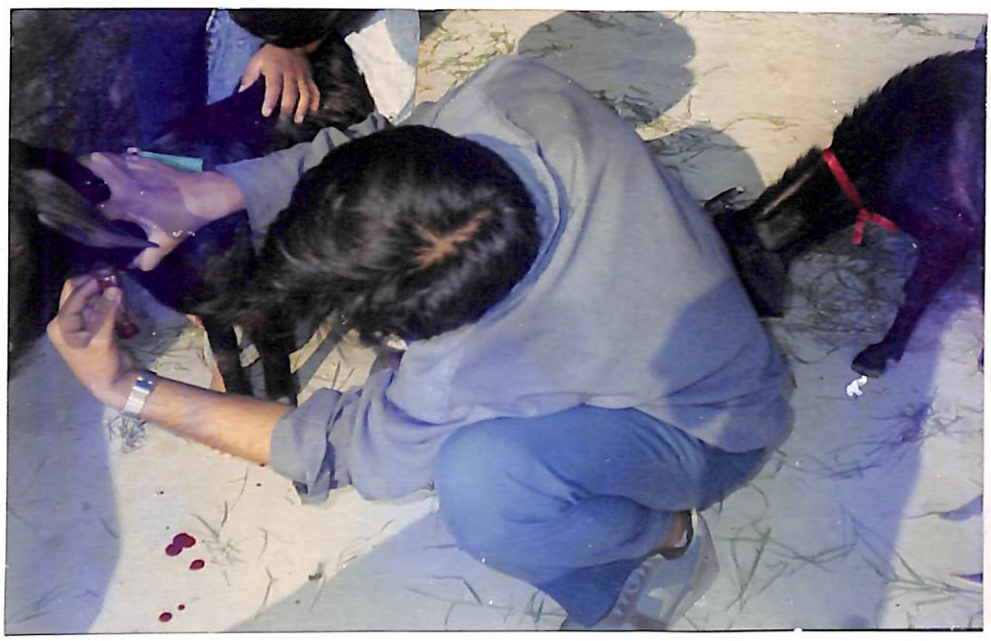
Photograph showing, feeding of whole wheat grains to goats of group A.

Photograph showing, feeding of whole wheat grains to goats of group B.

Photograph showing, acidotic goats of both the groups.

Photograph showing, collection of rumen liquor by stomach tube.

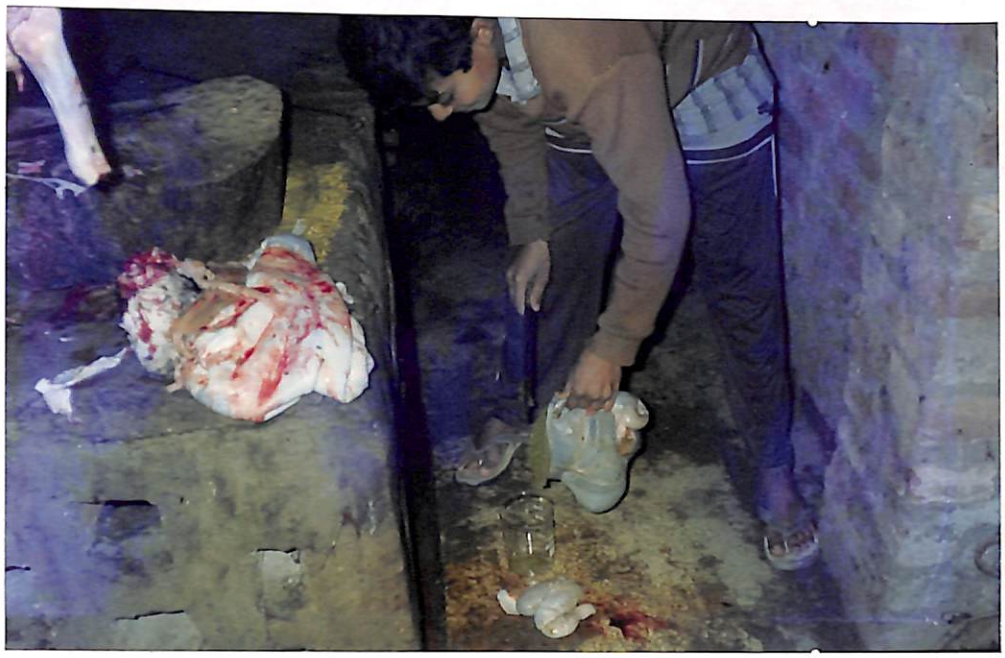
Photograph showing, recording of rectal temperature.



Photograph showing, collection of blood.

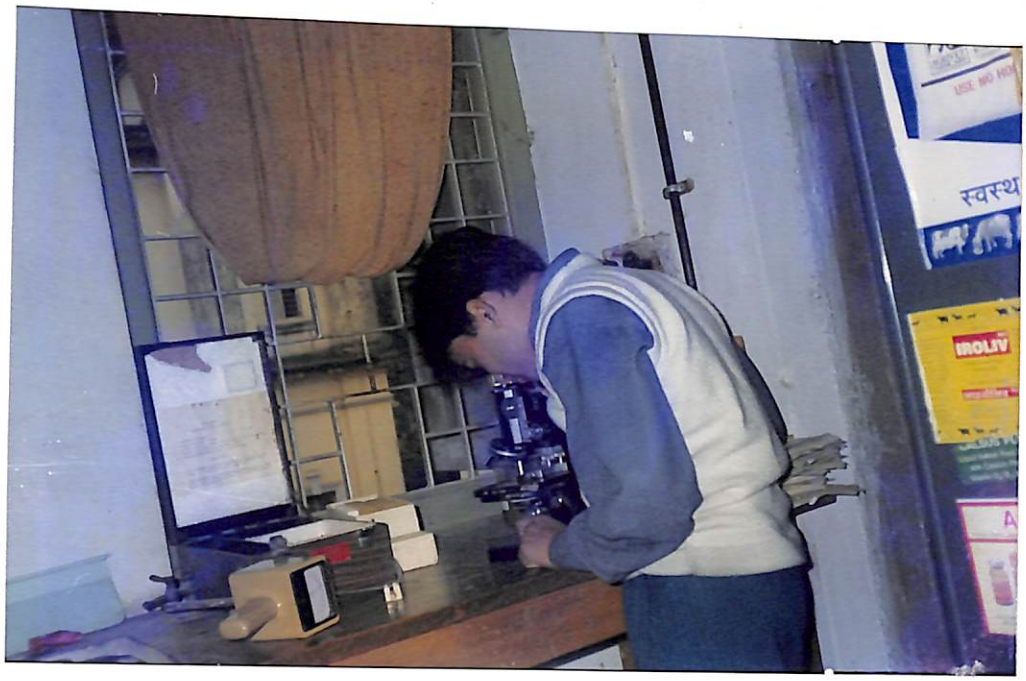
Photograph showing, milkfever posture in affected goat.

Photograph showing, recording of rumen pH by griph pH meter.



Photograph showing, collection of rumen liquor at butcher's shop.

Photograph showing, I/V infusion of fluid therapy.



Photograph showing, recording of motility of rumen protozoa.