Clinico-Biochemical and Therapeutic Studies on Acidosis in Goats

THESIS

SUBMITTED TO THE

RAJENDRA AGRICULTURAL UNIVERSITY

(FACULTY OF POST-GRADUATE STUDIES)

PUSA, (SAMASTIPUR) BIHAR

In partial fulfilment of the requirements

FOR THE DEGREE OF

Master of Veterinary Science

IN

VETERINARY MEDICINE

By

Dr. Pradeep Kumar Ram

Registration No. M/Vety. Med./26/1998-99

Department of Veterinary Medicine

BIHAR VETERINARY COLLEGE

PATNA

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Dedicated

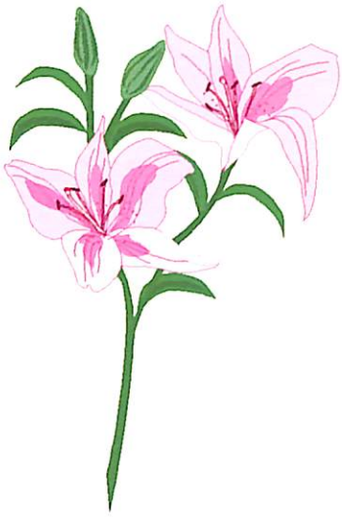
to

My Parents

Shri Dudh Nath Ram

&

Smt. Rikeba Devi



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CERTIFICATE – I

This is to certify that the thesis entitled **"CLINICO-BIOCHEMICAL AND THERAPEUTIC STUDIES ON ACIDOSIS IN GOATS"** submitted in partial fulfilment 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. Pradeep Kumar Ram** 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 the sources of literature have been fully acknowledged.

Endorsed 30.1.2001
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Chairman/Head of the Deptt.

30.01.2001
(S.P. Verma)
Major Advisor

CERTIFICATE – II

We, the undersigned, members of the Advisory Committee of **Dr. Pradeep Kumar Ram,** 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 ON ACIDOSIS IN GOATS" may** be submitted by **Dr. Pradeep Kumar Ram** in partial fulfilment of the requirements for the degree.

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ACKNOWLEDGEMENTS

I would like to express deep and sincere feeling gratitude to my guide and major advisor, Dr. S.P. Verma. Ph. D., Assoc. Prof. Deptt. of Vety. Clinical Medicine, Bihar Veterinary College, Patna, for his valuable guidance, excellent supervission, constant encouragement, keen interest unequalled suggestion and everlasting inspiration during the entire study. I am indebted to him for his kind co-operation and helpful attitude from the beginning of the investigation to the preparation of the manuscript of this thesis.

The author is very much thankful to the members of his Advisory Committee, Dr. B.P. Sinha, Ph. D.. Assoc. Prof. & Head, Deptt. of Vety. Medicine, Dr. B.K. Sinha, Ph. D., Assoc. Prof. & Head, Deptt. of Vety. Microbiology, Dr. S.R.P. Sinha, Ph. D., Assoc. Prof. Deptt. of Vety. Parasitology, Dr. C. Jayachandran. Assoc. Prof. Deptt. of Vety. Pharmacology and Toxicology, Dr. S.B. Verma, Ph. D., Assoc. Prof. Deptt. of A.B.G., Dr. J.N. Singh, Ph. D., Assoc. Prof. and Head, Deptt. of Vety. L.P.T., Vety. Biochemistry and director Resident Instruction-cum-Dean, P.G.S. for their valuable suggestions and untiring help.

Deep hearted sincere thanks to Dr. V.K. Sinha, Ph. D., Assoc. Prof. & Head, Deptt. of Epidemiology and preventive Vety. Medicine, B.V.C., Patna for his spell bound efforts, unerring criticism and keen interest to complete this work.

I am also thankful to Dr. Mani Mohan, Dean-cum-Principal, Bihar Vety. College, Patna for providing all possible facilities in time.

I am very much thankful to Late Dr. Md. Murtuza, Ex. Assoc. Prof. & Head, Deptt. of Vety. Biochemistry, Dr. Siyaram Singh, Ph. D., Assoc. Prof. Deptt. of A.B.G. and Dr. Surendra Kumar (M.V.S.c.) for their excellent guidance.

The author is obliged to Dr.Jagnnath Oraon, Dr. Ashfaque and Dr. Anjuman Ara for their help during reference collection. I am also thankful to my all friends and colleagues viz. Dr. Shrawan, Dr. P.K. Babbar, Dr. Deepak Oraon , Dr. Sadanand, Dr. Bisheshwer, Dr. Sanjeev, Dr. Rekha Teresa Kujur, Dr. Sanjay Kr., Dr. Mukesh, Dr. Arun, Dr. Param Bodh, Dr. A. Rahman, Dr. G. Verma, Dr. Sanjay Singh, Dr. Sashikant Singh, Mr. Madhukar and Mr. Satyendra for their help rendered during the study.

The author wish to record deep regards and heartfelt thanks to his respectable seniors, Dr. Yogendra Kr. Das, Dr. Arvind Kr. Das, Dr. Birendra Kr., Dr. Navin Kr., Dr. Krishna Choudhury, Dr. Vijay Mandal, Dr. Gopal Pd. Singh, Dr. Shahid Prawez, Dr. Kamakhya Das, Dr. Raychand Paswan and Dr. Omprakash Chaudhary for their constant help and moral encouragement during the study period.

Thanks are also extended to Dr. Abhishek, Dr. Sunil, Dr. Purushottam, Dr. Ankesh, Dr. Nitesh, Dr. Moin, Dr. Parmanand, Dr. Shambhu, Dr. Ajay, Dr. Anil Das, Dr. Vijay, Dr. Ramsharan, Dr. Ravindra, Dr. Ram Naresh Raman and Dr. Binod for their friendly co-operation during the course of the thesis work.

I am extremely thankful to Shri. H.P. Yadav and his wife Dr. Asha Devi for their valuable guidance and constructive suggestions during entire period of investigation.

My thanks are also extended to all technical and non-technical staff members of Deptt. of Vety. Medicine , pharmacology and B.V.C. Library, Patna for their help during study Period.

I have no words to express my gratitude towards the tremendous support and blessing that my father Shri. Dudh Nath Ram (H.M.), Mother Smt. Rikeba Devi and Dadi Maa.

Lot of thanks to my dear brother, Mr. Ravishankar, Dr. Rajesh, Mr. Divakar, Sisters, Miss. Shantee, Miss Gyantee, and Smt. Chinta Devi for

their moral encouragement and help throughout the course of study and research.

The author is also thank to his uncle Shri Shivnath, Shri Rambali and Shri Rajbali Ram including all family members for their constant encouragement, sacrifice and excellent love during entire investigation.

The Author is much thankful to his father-in-law, Shri Prameshwar Ram (H.M.), Mother-in-law, Smt. Binda Devi, Brother-in-law, Mr. Krishna and Mr. Ghanshyam , Sister-in-law. Miss. Mintu and Miss. Rintu for their close co-operation during entire study period.

Finally, I shall fail in my duty if, I do not extend special gratitude to my beloved betterhalf, 'Pinki' for her constant encouragement, sacrifice, devotion and affectionate help, without which I would not have imagined to complete the investigation smoothly.

I am also thankful to all family members of Sanjeevani Art Computers like, Mr. Monoj, Mr. Raja, Mr. Dharm Raj Chaudhary etc. for their kind support in course of typing and printing of this manuscript.

Last but not the least, I extended my sincere indebtedness to Baba Saheb Dr. Bhim Rao Ambedakar for enlighting and spritual guidance.

Pradeep Kumar Ram.

(Pradeep Kumar Ram)

Place: B.V.C. Patna.

Date: 30.01.2001.

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CHAPTER - I

INTRODUCTION

INTRODUCTION

From the time immemorial, the goat has been domesticated by man for its versatility. Throughout history, the contribution of goats in developing countries has been remained very prominent. This prominence is associated with a number of features unique to this species. Its wide distribution, across all complex agro-ecological environments & functional contribution, in terms of milk, meat fiber, skin and the socio-economic relevance includes income generation and human nutrition.

With regard to the population size and distribution, India has 110 million of total goats population which is nearly one-fourth (¼)th of the total world population and is maximum in any country of the world (F.A.O., 1991). India ranks 1st in goat population in the world (Sahni, 1982) & the maximum number of goats are found in Bihar, Rajsthan, U.P. and M.P. Nearly 40 % of country's total goats population is concentrated in these states. Goat contributes to about 35 % of total meat and 3 % of total milk produced in India (Chawla *et al.*, 1981). Exports of goats, hair, skin & caesins also earns valuable foreign exchanges (Acharya, 1982).

Goat, like other ruminants, have well defined compartments namely rumen, reticulum, omasum and abomasum. Functionally, rumen is most momentous organ, which takes major work of gastrointestinal tract. The function of rumen is mainly fermentive

and digestive. The fermentation of rumen is maintained by numerous micro-organisms. These microflora of rumen include cilliates, polymastigate, Oscillospires flagellates, selenomonads small bacteria mainly gram negative & fungi, predominating the rumen culture (Hungate, 1966). It is a continuous culture sac which maintains the homeostasis for the maximum growth of micro-organism. Besides, these functions, rumen renders detoxification of toxic substances. Optimal microbial activities can be achieved when the buffering capacity of the rumen works satisfactorily because, pH is the determining point, change in which causes destruction of ruminal micro-organisms.

Unfortunately, this voluminous structure undergoes numerous dysfunctions. These dysfunctions sometimes are severe enough to cause death of the animal. In ruminants, optimum rumen motility, concentration of living micro-organisms, anaerobiasis, correct pH of the rumen liquor, fluidity of the rumen mass and balanced substrate combinations are essential for normal digestion (Randhawa, 1979). But, for the distinct fermentative digestion in ruminants, any accidental change in the feed, fodder and management, lead to alteration of physio-chemical condition of the rumen resulting into rumen dysfunction.

Besides these, for obtaining more meat, milk and other products from goats, people introduce sudden change in various

feed constituents. They provide much more concentrates, which destabilize the ruminal flora and fauna, resulting into rumen dysfunction. This type of feeding causes indigestion, termed as "Ruminal Acidosis, Lactic Acidosis, Toxic Indigestion, Acute Carbohydrate Engorgement, Acute Impacting, Over eating disease, Grain overload or Founder etc. (Chakrabarti, 1988).

The syndrome 'Ruminal Acidosis' in goat is not uncommon which arises from accidental ingestion of large quantity of carbohydrate rich diet such as flour, crushed wheat, crushed rice or paddy, as per the greedy nature of the goats or by over feeding with grains or concentrates, to improve the milk yield as well as the weight of the goats causes high mortality rate due to proliferation of lactate producing rumen bacteria particularly the *Streptococcus lactis* on lowered ruminal pH. The excess accumulation of lactic acid in rumen results into absorption of acid in the blood further decreases in blood, pH (Dirksen, 1965; Ahrens, 1967; Dunlop, 1972; Choudhury *et al.*, 1980 and Sen *et al.*, 1982).

Ruminal lactic acidosis renders significant economic loss in terms of wasted feed, delayed marketing and condemnation of rumen, liver or entire carcass, lowered nutritional value, reduced water binding capacity of meat and several organoleptic deficiencies (Schukken *et al.*, 1985; Cleon, 1988 and Blood and Radostits, 1989).

Rumen hyperacidity has recently attracted the attention of veterinary clinicians in India due to its occurrence in all ruminant species causing mortality in untreated cases due to acidaemia and progressive dehydration (Blood and Henderson, 1968). It is reported that about 18 percent cases of anorexia in sheep and goats in India are due to ruminal acidosis (Prasad *et al.*, 1976a).

Clinically, this syndrome is manifested by severe toxemia, complete loss of appetite, suspended rumination, complete ruminal stasis, severe constipation or diarrhoea or dehydration, weakness, incordination, recumbancy and high mortality rate (Elam, 1976 and Blood and Radostits, 1989). Moreover the disease has been reported to cause ruminal and systemic acidosis, prostration, coma and high mortality among severely affected animals (Dirksen 1970 and Vestweber *et al.*, 1974).

As a matter of fact, 'Ruminal acidosis' is very common ailment in poor man's cow, due to its greedy nature. Such malady has been extensively studied in detail for its clinico-biochemical aspects in bovines (Hoflund, 1967; Prasad and Joshi, 1971; Prasad *et al.*, 1972; Vihan *et al.*, 1973; Prasad and Joshi, 1975 and Vihan, 1978) and in sheep (Scrarisbrick, 1954 and Slania, 1970). Although, in goats, some experimentally induced acidosis have been reported from different places (Das and Misra, 1992) not at all from Bihar. Since the reports, on clinical acidosis and its therapy are scanty & keeping in view i.e. the importance of this poor creature particularly for the people living below the

poverty line, initiated to under take this programme with following objectives:

(i) A thorough clinical examination of all the goats brought to B.V.C. Hospital with particular complain of inappetance, anorexia and suspended rumination without febrile reactions were made. It included the record of history of feeding and management particularly the feed provided before appearance of the syndrome of rumen dysfunction/acidosis.

(ii) Determination of ruminal motility, pH of ruminal fluid and also motility as well as activity of ruminal microflora.

(iii) Determination of the level of some important biochemical constituents viz. Lactic Acid and Glucose in Blood and Chloride, Sodium and Potassium in Serum of healthy goats, pre-treated and post-treated goats.

(iv) The therapeutic approaches were included.

(a) Neutralization of the ruminal acidosis by oral or parentral administration of antacids.

(b) Restoration of ruminal motility, ruminal tone and microbial activity by oral administration of rumenotoric drugs (Herbal preparations) and ruminal cud from healthy goats.

(c) Restoration of fluid and electrolytes on the basis of results of ionograms.

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CHAPTER - II

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Status of rumen plays an important role in maintaining health and keeping productivity of goats to optimum level. In-discriminatory feeding habits, excessive feeding to get maximum production, abrupt change in diet and use of certain drugs may hamper the microbial digestion in rumen, primarily by altering the runimal environment, which results into loss of production.

Ruminal acidosis is one of the major rumen dysfunction of goats arises from accidental ingestion of large quantities of highly fermentable carbohydrate rich diet causes a non physiological reduction in rumen pH. This reduction in ruminal pH favours the prolification of lactate producing gram+ve bacteria which ferments the readily availabe carbohydrate, resulting into an excess accumulation of lactic acid in the rumen and its subsequent absorption into blood. The syndrome "Ruminal Acidosis" is characterised by anorexia, toxaemia, ruminal stasis, constipation, diarrhoea, dehydration, generalized weakness, recumbency, incoordination, staggering gait, tachycardia, increased respiratory rate and high mortality.

The literature on acidosis in goat has been reviewed under the following heads.

1. Occurance of acidosis (Naturally and Experimentally)
2. Clinical findings (Tempt, pulse, respt. etc.)
3. Examination of ruminal fluids
   (a) pH of ruminal fluid
   (b) Motility of ruminal microflora (protozoa)
   (c) Activity of ruminal microflora, followed by
      (i) Cellulose digestion time (CDT)

(ii) Sedimentation activity time (SAT)

4. Examination of Blood/Serum

(a) Blood lactic acid

(b) Blood glucose

(c) Serum chloride

(d) Serum Sodium

(e) Serum potassium

(5) Therapeutic studies

2.1 OCCURANCE OF ACIDOSIS (NATURALLY AND EXPERIMETALLY)

Adoptation of lamb with flaked maize with low roughage diet over a period of week caused acidosis. Ground wheat, ground rye or glucose administered suddenly in large quantity could resulted in low pH, ruminal stasis and death (Phillipson, 1952).

Scarisbrick (1954) induced acid indigestion in sheep of about 150 lb body weight by providing 16 lb of mangolds for one day after maintaining the sheep on diet of 12 lb of mangolds daily for a fortnight.

Delak and Adamic (1959) experimentally produced rumen acidosis in 44 sheep by giving concentrate sucrose solution through mouth. Sheep given @ 5g/kg body weight remained healthy, those given 40g/kg body weight died within 8-13 hours.

Broberg (1960) reported that sheep allowed to eat continuously for 8 hrs. and that had consumed 4 to 5 kg of cereals developed acute over eating syndrome with typical signs of acidosis.

Gnanaprakasam (1970) reported the clinical cases of ruminal acidosis in goats due to grain overload (Paddy or rice) during the period 1968 to 1969 in Thanjavur district of Tamilnadu.

Prasad *et al.* (1976a) investigated 99 cases of anorexia in sheep and goats and found 18% of the cases to be due to acidosis.

Sen (1982) induced rumen acidosis in goat by introducing crushed rice @ 80g/kg body weight through rumen fistula after 36 hrs. of fasting.

Vihan *et al.* (1982) experimentally produced rumen acidosis in goats by three treatment viz. Administration of 1.00 kg crushed barley, 1.0 kg crushed grains (Barley 50% and Wheat 50%) and 500.00 g cane sugar through rumen fistula.

Srivastava *et al.* (1985) investigated 3253 goats for various disorders at Agricultural University, Rahuri and recorded 20% of all disorders to be the cases of digestive disorders including ruminal acidosis.

Teli *et al.* (1986) reported acidosis in ewes caused by feeding of damaged apple (*Malus sylvestris*).

Gutowski (1988) induced acidosis in 5 rams giving the aquous solution of sugar into the dorsal sac of the rumen @ 12-20 g/kg body weight through permanent fistula.

Intraruminal administration of finely ground wheat @ 50 g/kg weight produced acidosis in sheep (Crichlow, 1989).

Lal *et al.* (1989) succeeded in producing acidosis by giving wheat @ 100 g/kg body weight intraruminally in goats.

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

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

Patra *et al.* (1993) induced ruminal acidosis in six adult sheep by oral feeding of wheat grain @ 90g/kg body weight. Ruminal fluid, blood, CSF and urine samples were collected before grain feeding (0 hr.) and thereafter at 12, 24, 46, 72, 76 and 120hr intervals various parameters were studied.

Basak *et al.* (1994) induced acid indigestion in six healthy Black Bengal goats weighting 15-18 kg about 1½ to 2 years of age, by intra-ruminal administration of crushed rice @ 70 g body weight.

Aslan *et al.* (1995) induced ruminal acidosis in 21, 10 month old, west African Dwarf Goats by feeding a suspension of 80 g wheat flour/kg (day 0) through a stomach tube. Ruminal and systemic acidosis was diagnosed on day 1 in all goats.

Ruminal acidosis was produced by feeding soaked wheat at 50 g/kg body weight after 24 hr starvation in six ewes (2.5 to 3 year old) Patra *et al.* (1996).

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

Nour *et al.* (1998) experimentally induced lactic acidosis in Nubian goats using sorghum flour @ 50g/kg body weight given intraruminally through a fixed rumen canula. It resulted in the development of acute and peracute clinical signs of lactic acidosis.

Nikolov (2000) described clinical experimental studies on acute rumen acidosis in sheep with a pre-existing chronic lead and Organo-phosphorus compound intoxication. I clinical and haematological changes.

2.2 CLINICAL FINDING :

Scarisbrick (1954) observed the signs of painful walking, difficult standing, lateral recumbency, occasional grinding of teeth, rumminal atony and lamenes from all the four lambs in sheep suffering from rumen acidosis.

Shinosaki (1959) did not noticed any reduction of appetite or ruminal mortility in goats following lactic acid induced ruminal acidosis.

Broberg (1960) noted clinical picture in sheep due to over eating which comprised of anorexia, stasis of fore stomach, thirst, subnormal temperature and suppressed urinary secretion.

Clinical symptoms of incoordination, blindness, subnormal or normal temperature followed by recumbency whthin 24-48 hrs and finally coma and death were recorded in ruminal acidosis in sheep (Nisbet *et al.,* 1963). Observations revealed that greedy feeders were first to be affected and profused mucoid diarrhoea was noticed. The milder case that recovered after the barley feeding was discontinued. The flock mortality varied from 5 to 20%.

Jensen and Mackey (1965) reported that the onset of clinical signs in ruminal acidosis in feedlot cattle was sudden but the body temperature usually remained normal. Inappetance and depression developed early. Respiration and pulse were accelerated. Muzzle was dry and in severe cases, grunting was manifested. Ruminal movement ceased. In termal stages, physical weakness, prostration, coma, subnormal body temperature, low blood pressure and cynosis developed rapidly. The urine became acidic. Death occurred after a course of 1 to 3 days.

Strafuss and Monlux (1966) noted the signs of ruminal acidosis in cattle which consisted an unsteady gait, ataxia, asthesia, excessive salivation,

atony of rumen, a watery diarrhoea and apparent blindness. The animals then became recumbent and exhibited spastic paralysis, prostration and movements of the jaw. They continued to struggle in lateral recumbency with paddling movements and made repeated attempts to rise. Severely affected animals generally died between 12 and 24 hrs. after the first appearance of clinical signs. The rectal temperature in animals was within the normal range i.e. between 101 to 103^0F.

Juhasz and Szegedi (1968d) noted reduced ruminal movement, increased heart and respiratory rates culminating in respiratory failure and death in cases of ruminal acidosis in sheep.

Joshi (1969) observed anorexia, a distended left flank, profound depression, staggery gait and pasty faeces, dyspoenic respiration and weakened pulse in a clinical case of acid indigestion in a bullock.

Gnanaprakasam (1970) reported that goats manifested clinically within 24 hours of accidental grains overload. The symptoms were classified into 3 catagories as mild, moderate and severe. Mild cases were characterised by anophagia and enlargment of abdomen. Rumen was firm and doughy on palpation. Ruminal motility was absent or sluggish. There was constipation. Moderate cases were characterised by anorexia with either consitpation or diarrhoea, simple dyspnoea, tachycardia and oliguria. Ruminal motility was absent. On auscultation of rumen, gurgling sound were heard. In severe cases animals were dull and unable to stand for long time. Skin appeared rough and lethery. Costal type of respiration was prominent. Heart rate increased and pulse was imperceptable. Muzzle was dry. Anorexia was present. Temperature was slightly elevated. Rumination and rumen

motility were completely absent. Frequent watery, foul smelling diarrhoea was noticed. Anuria was present.

Kelly (1974) reported that normal rectal temperature, pulse and respiration rate of goats as 102.9^{0}F, 70-90/minute and 20-30/minute respectively. He further indicated goats were alert, active and had normal posture and gait, glossy skin coat, pink to rosy conjunctival mucous membrane, normal appetite and normal rumination with frequency of urination 1 to 3 ~~types~~ times per day in cattle and goats.

Allison *et al.* (1975) dealt symptoms of rumen acidosis in sheep as diarrhoea, anorexia, weakness and death.

Elam (1976) noted outward signs of acidosis in feedlot cattle comprising of anorexia, ruminal stasis, diarrhoic or mucoid faeces, dehydration, incoordination and many time death, following the ingestion of excessive carbohydrate rich feed.

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

Sen *et al.* (1982) noted complete absence or weak and irregular reticulo-ruminal contraction up to fifty six hrs. in ruminal acidosis. The animals remained anorectic and resumed normal appetite only after eighty hours of therapy. The colour and consistency of the rumen liquor were light brown and thin, compared to normal dark green to greenish brown and viscous at 0 hr.

Tanwar and Mathur (1983a) studied clinical manifestations in three groups of goats with ruminal acidosis induced by intra-ruminal administration of whole wheat. In mild cases there was depression, anorexia,

semisolid faeces and decreased ruminal movement, where as in moderate cases dullness, depression, anorexia, constipation, regurgitation of undigested wheat, grinding of teeth, tachycardia, increased pulse and respiration rate, purulent nasal discharge, lameness with diurnal variation were also noticed. In severe cases, depression, diarrhoea, severe dyspnoea, increased pulse and respiration, tachycardia, grinding of teeth, regurgitation of wheat grains, suspended rumination, uneasy gait, inability to stand, lameness, subnormal temperature, anuria and a sunken eyes were the prominent symptoms.

Sen *et al.* (1984) studied acute ruminal acidosis in 6 goats, the signs were observed as ruminal stasis, inappetance, dullness, depression, severe dehydration, polydispsia, oliguria, increased pulse and respiratory rates, sunken eyes, roughened body coat and death of 5 goats between 25 and 106 hrs. post inducement with crushed rice.

Cao *et al.* (1987) studied clinical manifestations of acidotic goats and reported that all goats survived, but showed clinical signs of dullness, sternal recumbency, increased mean heart and respiratory rates. Soft, but not voluminous, faeces were being passed by all animals by 12 hrs. and each had abdominal distension.

Chakrabarti (1988) reported bright eyes, alert apperance, glossy skin in healthy cattle and goat. The muzzle appeared with dew-drops like appearance overit. They had normal posture and gait. The healthy goats had pellety faeces with dark green colour. He also reported that normal pH of urine of sheep, goat and cattle range from 7.5 to 8.4. He also reported that the normal ranges of body temperature, pulse and respiration rate in healthy goats as 101.5-103.5^{0}f, 60-70/minute, 18-30/minute respectively.

Lal *et al.* (1989) noted inappetance, anorexia and ruminal stasis in experimentally induced ruminal acidosis in goats, whereas 30 to 50 % goats exhibited the signs of dullness, diarrhoea, constipation, abdominal pain, nasal discharge, head pressing and grinding of teeth etc. There was significant but gradual fall in body temperature associated with marked increased in pulse and respiration rates and significant decrease in ruminal motility.

Das (1990) observed the symptoms likes rapid pulse, respiration and rise in body temperature, complete rumen atony, pasty faeces diarrhoea, inappetence, distended abdomen, dehydration and death in experimentally induced ruminal acidosis in goats.

Lal *et al.* (1991) induced experimental acidosis in goats, the clinical signs observed were anorexia, ruminal stasis, constipation followed by diarrhoea, abdominal pain, nasal discharge, head pressing syndrome and teeth grinding.

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

Bhikane *et al.* (1996) reported ruminal lactic acidosis in a buffalo and the clinical signs observed were servere depression, milk fever like posture, subnormal temperature, tachycardia, dyspnoea, dry muzzle, cold extremitise, absence of defecation and frequent micturation. Rumen was impacted and atonic. Per-rectal examination revealed blackish and foul smelling faeces containing sorghum grains. Ruminal fluid was grayish white, thick, sour smelling with pH 4.0. The microscopic examination of ruminal fluid revealed absence of the live ruminal protoza.

Nour *et al.* (1998) experimentally induced lactic acidosis in Nubian goats and observed different clinical signs. Moderate clinical signs developed in animals of group I which were manifested with dullness, depression, anorexia, slight dehydration, ruminal stasis, reduced water intake, tachycardia and polypnea. Animals of group II showed the clinical syndromes viz. peracute signs comparised severe dehydration with sunken eyes and loss of dermal elasticity, apparent blindness, salivation, grinding of teeth contineous crying, subnormal temperature (99.5-100^{0}F) and accelerated heart rate. Respiration was shallow and rapid (35-45 min^{-1}). Finally the animals went down in lateral recumbency and died. Some of the goats showed the clinical signs of complete loss of appetite, frequent watery diarrhoea and sternal recumbency with neck extended. Animals in group III developed per acute clinical signs similar to group I and II and died later on.

Dwivedi *et al.* (2000) reported decreased ruminal motility with respect to frequency and amplitude by 36 hrs. post feeding. All the animals showed signs of inappetance, dullness and depression by 12 hrs. post feeding.

2.3 EXAMINATION OF RUMINAL FLUID :

2.3.1 pH of ruminal fluid :

Phillipson (1952) noticed lower rumen pH in lambs fed flaked maize ration and opined that, ground wheat, ground rye or glucose adminstered suddenly in large quantity could result in low pH of rumen fluid.

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

Bullen and Scarisbrick (1957) experimentally produced acidosis in sheep by engorging the rumen with wheat, oats and maize grains in one trial

and with sulphuric acid in another trial. In grain engorged sheep, the rumen pH fell to 4.0.

Ryan (1964) observed that, following the intake of soluble carbohydrate the first sign of ruminal dysfunction was the lowering of pH to 5.0 and in severe cases to below 4.0.

Joshi (1969) reported a case of acid indigestion in a bullock which accidently excessed wheat during thrashing operations, in which the ruminal fluid pH was 5.5.

Gnaprakasam (1970) observed the pH of rumen liquor between 5.2 to 5.5 and below 5.2 respectively in moderate and severe cases of ruminal acidosis in goats

Joshi and Ludri (1970) found the ruminal pH up to 5.0 in clinical cases of acid indigestion in buffalo, as against normal pH between 6.12 and 6.75 in buffaloes belonging to the same locality.

Reddy and Nair (1971) observed that the pH of rumen liquor at 2nd, 4th, 6th and 8th hrs. sampling were 6.05 ± 0.04, 5.70 ± 0.04, 6.00 ± 0.04 and 6.35 ± 0.03 respectively in healthy Jamunapari-Malbari crossbred female goats.

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

Dunlop (1972) suggested rumen pH in the range of 3.9 to 4.5 in lactic acidosis in cattle.

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

Shinosaki and Nakabayashi (1974) reported a fall of rumen liquor pH to 4.64 after 12 hrs. of glucose induced rumen acidosis in sheep.

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

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

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

Rai and Pandey (1980a) reported pH of rumen liquor in three groups of goats maintained on range, supplemented with concentrate and concentrate mixture varied. The mean values of pH of rumen liquor were 5.313 ± 0.038, 6.155 ± 0.045 and 6.29 ± 0.059 respectively.

Sen *et al.* (1982) noted the rumen and urine pH at 12 hrs. of induction of acidosis in goats to be 4.65 and 6.05 respectively.

Vihan *et al.* (1982) reported that lowest pH values as 5.5, 4.5 and 4 at 12 hrs. of barley, 42th hrs. of grain (50% barley ± 50% wheat) and 12th hrs. of cane sugar induced rumen acidosis respectively in goats.

Tanwar and Mathur (1983c) reported ruminal pH of 4.96, 4.85 and 4.29 at 12 hrs. in mild, moderate and severe cases of acidosis in goats respectively following whole wheat engorgement.

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) produced acute ruminal acidosis in goats, and recorded minimal pH of 4.37 ± 0.12 within 12 hrs. of engorgement with rice.

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

Kovac *et al.* (1986) studied pH changes in induced acidosis in calves. Blood pH of ruminating calves feel from 7.36 to 7.16 at 10 hrs., rumen pH from 6.9 to 4.7 at 8 hrs. and faecal pH from 6.55 to 5.1 at 10 hrs.

Cao *et al.* (1987) reported that the pH of rumen liquor change from 7.35 ± 0.302 to 4.54 ± 0.119 at 24th hrs. of sucrose induced acidosis in goats @ of 16 g/kg body weight.

Lal *et al.* (1989) reported, a significant drop in the ruminal pH observed to (4.7 ± 0.045) at 12 hrs. with a further drop to (4.54 ± 0.023) at 24 hrs. after experimentally induced ruminal acidosis in goats by whole wheat grain @ 100 g/kg body weight.

Randhawa *et al.* (1989) observed that the pH of rumen liquor changed from 6.80 ± 0.04 to 4.77 ± 0.77 at 24th hrs. and 6.79 ± 0.04 to 4.24 ± 0.06 at 24th hrs. in sub acute and acute rumen lactic acidosis in buffalo calves respectively.

Das and Misra (1991) reported the pH of rumen liquor to be 6.95 to 0.03 and 3.0 in healthy and acidotic goats respectively. The pH of rumen liquor decreased markedly from 12hrs. onward and the mean pH 4.48 ± 0.01 was recorded at 12 hr of the study.

Lal *et al.* (1993) experimentally induced acute rumen lactic acidosis in clinically healthy (1-2 year old) goats of either sex by intraruminal

administration of whole wheat grain @ 100 g/kg body weight. A significant reduction was observed in rumen pH (4.71 ± 0.045) at 12 hrs.

Basak *et al.* (1993) experimentally induced lactic acidosis in goats and observed biochemical changes in rumen liquor. The pH of rumen liquor gradually decreased significantly ($P<0.01$) from 12th hrs. to the lowest value at 48th hrs. from 60th hrs. onwards an improvement in pH was noted but still significantly lower ($P<0.01$) than that of 'O' hrs. value.

Angelov *et al.* (1995) carried out an experiment in acute ruminal acidosis in eight adult goats by a single oral administration of 20 ml/kg of body weight beet molasses and found ruminal pH 3.68.

Patra *et al.* (1996) induced acidosis experimentally in 6 ewes (2.5 to 3 years old) by feeding soaked wheat @ 90 g/kg body weight, after 24th hrs. starvation and reported decreased ruminal pH value.

Patra *et al.* (1997) in a study of experimental ruminla acidosis in sheep, they found decreased pH of rumen liquor (4.56 ± 0.135 to 4.91 ± 0.153).

Nour *et al.* (1998) experimentally induced lactic acidosis in Nubian goats and recorded decreased ruminal pH 4.7 at 6th hrs. after dosing with wheat and subsequently increased, until it returned to normal level (6.5) at 144 hour post-dosing.

Dwivedi *et al.* (2000) studied the physiochemical examination of rumen liquor before and after induction of ruminal dysfunction and reported decreased ruminal pH (5.75 ± 0.781).

2.3.2 Motility of Ruminal Microflora (Protozoa):

Hungate *et al.* (1952) observed that, total disappearance of rumen protozoa at 12 hrs. might be due to very high concentration of lactic acid and low pH in the rumen in acute indigestion of sheep.

Cherkasov (1959) noted a decrease in the number of rumen infusoria in cattle after addition of grains with hay ration. On the second and third day of experiment, the population of infusoria decreased and consisted only encysted forms.

Krogh (1959) observed that, most protozoa died and there was marked reduction in the number of gram negative flora in rumen liquor of sheep fed excess of cane sugar.

Krogh (1960) reported that, the disppearnce or reduction in number of protozoa in animals gradually adapted to consuming concentrates ad libitum, was probably caused by some of the same factors that killed protozoa when dosed with concentrates. The factor included low rumen pH.

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

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

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

Misra *et al.* (1972) examined fresh rumen fluid samples from healthy Indian cattle, under the microscope and graded protozoal motility as moderate (++) to vigorous (+++) ie. 10-30 protozoa per fields.

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

In early stages of acute, sub acute and chronic ruminal acidosis, decreased motility and viability of rumen protozoa was observed (Cakala *et al.*, 1975).

Sethuraman (1976) induced ruminal acidosis in buffalos calves and Zebu calves by intraruminal administration of rice, complete disappearance of rumen protozoa after 24 hrs. of induction was obtained.

In cross-bred cow and buffalo calves from the 1st to the 4th day under crushed oat grain induced acid indigestion, complete disappearance of rumen protozoa on pH 5.0 to 4.2 was reported by (Nauriyal and Baxi 1978).

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

In a experimentally induced ruminal acidosis by oral feeding of molasses in buffalo calves, Choudhuri *et al.* (1980) recorded optimal motility of rumen protozoa as vigorous (+++) prior to induction, while after 3 hrs. post-induction, it became scanty (+) with many of the protozoa classed as dead. Though there was complete disappearance of protozoa after 24 hrs. samples at 108 and 132 hrs. indicated the presence of protozoa, graded as "+" to "++" (Sluggish to moderate).

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

Sen (1982) found moderate to vigorous protozoal motility at 'O' hr., moderate at 4th hrs. and absent at 8th hrs., onwards of grain overload in goats.

Tanwar and Mathur (1983c) recorded reduced protozoal count in mild acidotic in goats, while in severe cases no protozoa could be seen at 12 hrs.

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

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) recorded. complete disappearance of ruminal protozoa from 12 to 72 hrs. in experimental rumen acidosis in goats.

Das (1990) found that, the fair number & better motility of rumen protozoa in healthy goats (moderate ++ to vigorous +++) but absent in rumen acidosis.

Narendra *et al.* (1990) observed a gradual reduction number of protozoa of rumen fluid till 18 hrs. and were found absent afterwards in experimental ruminal acidosis in calves.

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

Upadhyay and Shukla (1999) mentioned the protozoal motility was sluggish (+ or ++) in each animal before treatment but following treatment with Anigest, it became vigorous (+++ or ++++), showing action of medicine on rumen microflora in his suspected cases of ruminal disorder of clinically affected 80 sick cows.

2.3.3 Activity of ruminal microflora followed by CDT and SAT :

Hoflund *et al.* (1948) did the CDT in rumen liquor to know the activeness of rumen microbes. A fully active rumen liquor could digest cellulose within 48-54 hours. In case of indigestion, rumen liquor took more time to digest cellulose or could not digest at all.

Nicholas and Penn (1958) stated that, a good co-relation of SAT with ration and time after feeding. They observed also variations between cows and in the same cow, from day to day. The SAT of rumen liquor in healthy cattle was 3-8 minutes. Indigestion caused incomplete floating or complete settling without any floating which reflected the SAT.

Gnanaprakasam (1970) studied acid indigestion in goats and reported, no sedimentation or floatation activity of particles of rumen liquor.

Sen (1982) recorded that the normal sedimentation activity time of rumen liquor of healthy goat was 26.60 ± 5.86 minutes but there was complete absence of sedimentation activity after 4 hrs. onward of grains engorgement.

Blood *et al.* (1983) noted sedimentation activity time in normal animal which varied between 3 minutes if, the animal has just been fed and 9 minutes if the last feeding has occurred some time previously. Settling of the particulate materials indicates gross inactivity, less severe degree being manifested by prolongation of time required for floatation. CDT in excess of 30 hrs. indicate abnormality.

Pradhan *et al.* (1988) observed that average SAT of rumen liquor of healthy Black Bengal goat was 24.16 minutes.

Randhawa *et al.* (1989) noted that SAT in rumen liquor increased significantly at 6 hrs. and no sedimentation activity was detected at 12 and

24 hrs., which returned to normal by 168 hrs. of sub acute induction of lactic acidosis in buffalo calves. In acute lactic acidosis, complete absence of SAT was observed from 6 hrs. which persisted throughout the period of observation. No cellulolytic activity was detected in the rumen liquor in calves of sub acute lactic acidosis between 6 and 144 hrs. However, at 168 hrs. CDT values were higher than 48 hrs. values. In acute lactic acidosis, the CDT values were nil till the death of the buffalo calves.

Das (1990) observed that mean SAT and CDT of rumen liquor were 25.22 ± 1.95 minutes and 52.14 ± 3.07 hours respectivelly at '0' hr. which were completely absent on 12th hrs. onwards in experimental rumen acidosis in goat.

Basak *et al.* (1993) studied the CDT and SAT in experimentally induced lactic acidotic goats and found that significantly increased time factors for CDT and SAT ($P<0.01$) at 12 hrs. and was absent from 24 hrs. on ward.

2.4 EXAMINATION OF BLOOD/SERUM:

2.4.1 Blood lactic acid:

Broberg (1960) reported that sheep which over ate grains, had a higher lactic acid concentration in the blood.

Dunlop and Hammond (1965) suggested accumulation abnormal amount of lactate in the blood in lactic acidosis in ruminants.

Williams and Mackenzie (1965) studied the absorption of lactate from the isolated rumen of anaesthetized sheep. The absorption of volatile fattyacids was found to be five to ten times more rapid than lactic acid absorption from solution at pH 5.2. This difference could be accounted particularly by the fact that appreciate amounts of nonionized VFA would be

present at this pH whereas, lactate would be mainly in the ionized form due to the difference pKa (VFA = 4.8, lactate = 3.7). Absorption rate was said to be increased when concentration was higher at low pH (4.0) than at higher pH (upto 7.5).

Dirksen (1967) recorded increased level of blood lactic acid in peracute cases of ruminal acidosis in cattle.

Castello (1968) overloaded six months old sheep with wheat barley meal by rumen fistula. He observed that, though the rumen lactic acid rose sharply at once, blood levels remained steady for several hrs.

Juhasz and Szegedi (1968d) noted increased blood lactic acid concentration in the sheep suffering from ruminal acidosis.

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

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

Telle and Preston (1971) suggested that the absorption of lactate from the rumen was evident from the increased level of blood lactate in the rumen vien after 1.5 to 2 hrs. of intra-ruminal administration D-L lactic acid in sheep.

Vihan *et al.* (1973) recorded the lactic acid concentration in the serum of cattle and buffaloes suffering from acid indigestion. They observed the level of 24.3 ± 6.88 mg% in Zebu cattle and 6.8 ± 0.93 mg% in buffaloes.

Pinkiewicz *et al.* (1974) observed the blood lactic acid in normal cows whith ranged from 6.3 to 22.75 mg percent against 30.3 to 64.5 mg percent in acidotic cows.

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

Cakala *et al.* (1975) observed increased lactic acid concentration in rumen fluid in one study following saccharose administration and in another subsequent study, both rumen and blood lactate levels were elevated following large amount of grain feeding in feedlot cattle.

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

Nauriyal (1975) observed the blood lactic acid levels in acidotic buffalo calves to 19 mg percent, 33 mg%, 55 mg%, 72 mg percent and 90 mg percent at 0,1,2,3,4 and 5th day respectively as against 19.9 ± 6.2 in healthy ones.

Verma *et al.* (1975) recorded that, the lactic acid concentration in blood of Barberi buck at '0' hr., 2nd hrs., 4th hrs. and 6th hrs. of concentrate mixture feeding, which were 9.53 ± 0.45, 28.51 ± 0.90, 8.35 ± 0.30 and 8.55 ± 0.25 mg/100 ml respectively.

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

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

Nauriyal and Baxi (1981) experimentally produced lactic acidosis in crossbred cattle and buffaloes and found a high ruminal and blood lactate values.

Roychoudhary (1981) reported a rise in blood lactic acid level to 57.28 ± 8.88 in acidotic buffalo calves from the mean normal value of 19.14 ± 2.59 mg percent.

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

Vihan *et al.* (1982) stated that, the serum lactic acid concentration increased from normal 23.01 mg/100ml to 25.71 mg/100ml, 83.3 mg/100ml and 48.0 mg/100 ml by 18 hrs of barley, 42 hrs of grain (50% barley + 50% wheat) and 12 hrs of cane sugar induced acidosis in goats respectively.

Tanwar *et al.* (1983) revealed similar result when they used wheat to induce acidosis in goats. Lactic acid concentration increased from 9.45 ± 0.54 mg% in pre-engorgement to 27.30 ±1.32 mg% at 12 hrs and further increased to 40.57 ± 5.20 mg% at 72 hrs post engorgement.

Huber *et al.* (1984) stated increased blood lactate concentration from 3.50 ± 1.90 mg/100ml to 78.0 ± 20.0 mg/100ml in experimentally glucose induced acidosis in sheep.

Sen *et al.* (1984) produced acute ruminal acidosis in goats by intra-ruminal administration of rice and the highest lactic acid concentration (41.37 ± 12.42 mg%) occurred at 32 hrs.

Sandha and Choudhury (1985) reported increased blood lactate level at 12 hrs. of induced acidosis in buffalo calves. The value remained elevated throughout the acidosis phase.

Sinha *et al.* (1985) produced experimental acidosis in buffalo calves and reported that, the mean concentration of lactic acid rose from 17.38 ± 1.93 mg% pre-engorgement to a maximum of 60.00 ± 8.94 mg% at 36 hrs. post-engorgent.

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

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

Cao *et al.* (1987) reported that, the mean concentration of blood lactate was elevated from 1.78 ± 0.734 mmo/L to 2.72 ± 0.363 mmo/L at 12 hrs, then declined and again elevated to 2.79 ± 1.223 mmo/L at 48 hrs. of experimentally induced lactic acidosis in the goats.

Randhawa *et al.* (1988) reported that, the mean lactic acid concentration prior to death was 86 mg/100 ml in experimentally induced ruminal acidosis in 12 buffalo calves, as a result of which 2 animals died after 60 hrs. and another 2 after 96 hrs.

Lal *et al.* (1989) produced acidosis in goats experimentally and mentioned a rise in blood lactic acid concentration.

Das (1990) Observed significant increase in blood lactic acid in experimental rumen acidosis in goats. The highest blood lactic acid concentration of 38.06 ± 4.11 mg/100 ml was observed at 36th hrs.

Das and Misra (1991) carried out an experiment in ruminal acidosis in Black Bengal goats and found the level of lactic acid in ruminal fluid in healthy, untreated acidotic and treated acidotic goats as 4.17 ± 0.57 mg/dl, 35 mg/dl & 30 mg/dl respectively.

Patra (1991) recorded increased lactic acid concentration in both rumen fluid and blood in acidotic sheep.

Lal *et al.* (1992) observed significantally increased blood lactic acid concentration in the experimentally induced ruminal acidosis in goats.

Basak *et al.* (1993) observed significantly ($P<0.01$) increased lactic acid concentration in ruminal fluid of Black Bengal goats.

Sen *et al.* (1993) carried out an experiment in acute ruminal acidosis in 6 female Barbari goats and reported significantaly higher value of blood lactic acid.

Lal *et al.* (1993) experimentally produced acute rumen lactic acidosis in 6 clinically healthy 1-2 year old goats of either sex by intraruminal administration of whole wheat grains @ 100 g/kg body weight and observed increased value of lactic acid in ruminal fluid and blood.

Patra *et al.* (1996) noted increased lactic acid concentration in blood, in the experimentally induced acidotic ewes.

Angelov *et al.* (1996) carried out an experiment in acute ruminal acidosis in sheep and found, increased level of blood lactate.

Patra *et al.* (1997) observed increased the values of lactic acid concentration in both rumen liquor and blood in experimental ruminal acidosis in sheep.

Nour *et al.* (1998) reported experimental lactic acidosis in Nubian goats and stated that rumen lactic acid concentration increased progressively after dosing and reaches a maximum level of 52 mmol/l at 10 hrs. This level was maintained until the 25 hrs. post-dosing and then it fell, steeply at first, then gradually until reached normal level at 120 hrs post-dosing. They also stated, plasma lactic acid was not affected until 12 hrs. after dosing, but it

began to increase gradually reaching 8 ± 1.2 mmol/l by the 21hrs. post-dosing and reached a maximum level of 14.02 mmol/l by the 23 hrs. post dosing. However, the plasma lactic acid levels were maintained in the range of 11.2 – 12 mmol/l between 25-29 hrs. after dosing. The level then decreased to 8 mmol/l at 21 hrs. post-dosing and was maintained at the range of 7-9 mmol/l between 31-48 hrs. after dosing. Subsequently, the level of lactic acid declined until it resumed its normal values by the 56 hrs. post-dosing.

Dwivedi *et al.* (2000) carried out an experimental rumen dysfunction and recorded significant ($P<0.05$) increase in blood lactic acid concentration.

2.4.2 Blood Glucose :

Broberg (1960) reported that, the blood glucose content increased in sheep which, over ate grain.

Mullick (1964) recorded 79.3 mg/100ml of blood glucose level in two to three years old healthy buffalo calves.

Juha'sz and Szegedi (1968a, 1968b) mentioned increased blood glucose level in acidotic sheep.

Dirksen (1970) suggested, rise in blood glucose concentration in acidotic cattle to 214 mg%.

McIntosh *et al.* (1973) recorded three times rise in blood glucose level in sheep which was suffering from acidosis.

Cakala *et al.* (1974) recorded increased blood glucose level in sucrose induced acidosis in sheep.

Ivanov (1974) dealt hyperglycaemia (114 ± 8mg/100ml) and glycosuria in recurrent subacute or chronic ruminal acid indigestion in cows.

Shinosaki and Nakabayashi (1974) produced experimentally acidosis in sheep by glucose administration and reported a transitory increase in blood glucose level.

Vestweber *et al.* (1974) experimentally induced ruminal acidosis in sheep and found increased blood glucose level from 61.3 ± 1.7 to 78.9 ± 5.0 mg%.

Nauriyal (1975) stated that an increased in blood glucose level from normal 43.8 mg% to 102.8 mg% in acidotic calves.

Melvin (1977) determined the blood glucose level of a healthy goat which was found up to 45 to 60 mg/dl.

Nauriyal and Baxi (1978) induced acidosis in calves by intraruminal administration of molasses and reported a significant fall in blood glucose level.

Sethuraman and Rathor (1979b) experimentally induced acute acid indigestion in bovine and noted that blood glucose values rose steadily from pre-engorgement values of 45.0 ± 5.0 mg% and 48.3 ± 2.3 mg% to 72.5 ± 2.8 mg% and 81.6 ± 5.2 mg% 96 hrs., post engorgement in cattle and buffaloes, respectively.

Randhawa *et al.* (1980) produced ruminal acidosis in buffalo calves by intra-ruminal administration of wheat grains @ 50 g/kg body weight and observed a increased blood glucose level at 120 hour post-induction.

Bieniek (1981) experimentally produced acidosis in six heifers and observed decreased blood glucose level initially but increased later on.

Nagaraja *et al.* (1982) induced lactic acidosis in cattle experimentally by intra-ruminal administration of glucose. They found that the blood

glucose value increased from 32.0 mg% at '0' hr. to 33.4 mg% and 34. 3 mg% at 4 and 8 hrs, respectively to 35.3 mg% at 12 hrs. post engorgement.

Sen (1982) recorded the maximum mean value of blood glucose to 172.05 ± 94.11 mg/100 ml at 24 hrs. in goat over loaded with grains.

Vihan *et al.* (1982) noted increased blood glucose levels with decrease in ruminal pH, after rumen acidosis induction, by various treatments in goats. They observed highest blood glucose level in the barley treatment group of 76.5 mg% at 18 hrs while in the mixed grain (barley–wheat) treatment, the level was highest (140 mg%) at 42 hrs and in the cane sugar treatment, the highest level of 350 mg% at 12 hrs. post engorgement.

Sastry (1983) recorded the blood glucose level in normal healthy goat which ranged from 45 to 60 mg/100 ml.

Vihan and Rai (1985) reported a clinical cases of acidosis in goat and observed that, the mean concentration of blood glucose was 81.9 ± 8.57 mg%.

Das (1990) experimentally produced acidosis in goats and found, increased blood glucose level.

Lal *et al.* (1992) noted increased blood glucose level mg/dl (94.50 + 5.097) at 12 hrs in experimentally induced ruminal acidosis in goat.

Sen *et al.* (1993) carried out an experiment in acute ruminal acidosis in Barbari goat and reported significantly higher value of blood glucose (47.1 ± 6.8 mg% to 73.5 ± 14.6 mg%).

Angelov *et al.* (1995) found increased value of blood glucose (5.2 mml/litre) in blood sample of goats suffering from acute rumen acidosis.

Angelov *et al.* (1996) noted increased blood glucose level, (4.8 ± 0.9 mmol/l) at 12 hrs. in experimentally induced acute rumen acidosis in sheep.

Patra *et al.* (1997) found increased blood glucose level (70.65 ± 5.439 to 78.32 ± 7.36 mg/dl) in experimentally wheat grain induced rumen acidosis in sheep.

2.4.3 Serum Chloride :

Parthasarthy and Phillipson (1953) suggested the movement of chloride across the rumen epithelim of sheep and found that, the presence of 0.002 M mercuric chloride inhibited its absorption from rumen.

Dirksen (1967) recorded normal blood chloride value in peracute and hypochloraemia in the protrected cases of ruminal acidosis in cattle.

Shinosaki and Nakabayashi (1974) experimentally produced ruminal acidosis in sheep and noted, lower blood chloride level.

Cakala *et al.* (1975) recorded increased chloride level in rumen liquor of cattle and goats blood as well as its ruminal fluids.

Sinha *et al.* (1985) suggested initial rise of chloride value in the blood and CSF of acidotic buffalo calves at 24 hrs. following induction of acidosis.

Patra *et al.* (1993) found decreased level of chloride in CSF and urine in acidotic sheep.

Sen *et al.* (1993) studied acute ruminal acidosis in Barberi goats and found a significantly increase in the level of serum chloride.

2.4.4 Serum sodium :

Parthasarthi and Phillipson (1953) noted movement of sodium across the rumen epithelium of sheep and observed that, the presence of 0.002 in mercuric chloride did not affect its absorption.

Dirksen (1967) reported there was no much changes in blood sodium in ruminal acidosis in cattle.

Huber (1971) recorded decrease in serum sodium in glucose induced acute indigestion in sheep.

Telle and Preston (1971) observed that the concentration of sodium remained relatively constant when they infused lactic acid in sheep.

Shinosaki and Nakabayashi (1974) recorded increase in plasma sodium in acidosis of sheep.

Cakala *et al.* (1975) suggested decreased value of sodium ions in ruminal fluid of cattle following saccharose administration.

Irwin *et al.* (1979) noticed slightly increased level of plasma sodium in glucose induced acidosis in sheep.

Choudhuri *et al.* (1980) experimentally induced acidosis in Indian water buffaloes by feeding of molasses @ 10 g/kg body weight and found slightly increased sodium value in blood.

Sen (1982) reported increased in serum sodium concentration in experimental acidotic goats.

Heijlasz *et al.* (1984) noticed, decreased sodium value in experimental induced acute metabolic acidosis in cows.

Das (1990) reported increased serum sodium level in experimental rumen acidosis in goats.

Nikolov (1998) conducted a clinical experimental studies on acute rumen acidosis in buffaloes and found, blood sodium was decreased from the beginning up until hrs. 8 (177 ± 10 mmol/l) but after was decreased. Rumen sodium content decreased in the later hrs. of the experiment (110 ± 9 mmol/l) et post treatment at hrs. 72.

2.4.5 Serum potassium :

Parthasarthy and Phillipson (1953) reported the movement of potassium across the rumen epithelium of sheep and it was noticed that the presence of 0.002 m mercuric chloride did not affect its absorption.

Dirksen (1967) observed oscillating blood potassium value in protracted cases of lactic acidosis in cattle.

Huber (1971) mentioned decreased serum potassium in glucose induced acute indigestion in sheep.

Telle and Preston (1971) reported, increased blood potassium concentration increased upto 4 hrs. of intra-ruminal infusion of lactic acid in sheep and then back to the '0' hr. value.

Shinosaki and Nakabayashi (1974) recorded decreased plasma potassium level in acidotic sheep.

Cakala *et al.* (1975) noticed decreased potassium level in sheep.

Sethuraman and Rathor (1979b) observed increased potassium in acute ruminal acidosis in bovines.

Choudhuri *et al.* (1980) noted decreased potassium concentration in blood in experimentally induced acidosis by feeding of molasses @ 10 g/kg body weight in Indian water buffaloes.

Sen *et al.* (1982) dealt rise in the rumen and blood potassium levels in early stage of ruminal acidosis in goats with grain engorgement.

Heijlasz *et al.* (1984) noticed increased potassium value in experimental acute metabolic acidosis cows.

Kuusksalu (1988) noted increased blood potassium with decrease of blood pH in glucose induced acidosis of sheep.

Das (1990) recorded decreased serum potassium level in experimental rumen acidosis in goats.

Nikolov (1998) observed decreased potassium concentration in both blood and rumen liquid in acute rumen acidosis of buffaloes.

2.5 THERAPEUTIC STUDIES:

Rumen hyperacidity has recently attracted the attention of Veterinary Clinicians in India due to its occurence in all ruminant species causing, variable mortality in untreated cases. Death may be attributed to acidaemia and progressive dehydration in body (Blood and Henderson, 1968). Many antacids like Sodium bicarbonate or Sodium hydroxide, Magnesium oxide, Magnesium carbonate, vitamins and rumen contents (fresh or desicated) with rumenotorics, have been used for the treatment of rumen hyperacidity with variable results.

Westcott *et al.* (1953) observed a beneficial effect of "Proilovan" a synthetic rumen media in enhancing the rumen microbial growth and there activities in cows suffering from digestive disorders when used in early stages.

Pounden *et al.* (1954) studied, some substitutes for freshly obtained rumen contents or cud for use in young calves fed on high roughage ration and concluded that *in vitro* benefits of fresh rumen juice and commercial products was not comparable to those of freshly obtained rumen contents. They further suggested that indicators of typical micro-organisms could not be found in the commercial preparations and in frozen juice. The protozoa were no longer motile. In addition the *in vitro* CDT indicated, that compared to fresh rumen juice, the frozen product was 80 % efficient while the commercial product was only 30 % efficient.

Scarisbrick (1954) suggested use of ruminal contents, may lead to buffering the acid within the rumen and had beneficial effects in acidotic sheep.

Tucker *et al.* (1956) dealt the effect of dried preserved rumen inoculum on the rumen micro-organism of lambs and noted its beneficial effects in the therapeutic management of acidosis.

Bullen and Scarisbrick (1957) reported that treatment with Crystalline sodium penicillin was effective to inhibit the growth of gram-positive flora and thus checked the intra-ruminal pH in experimentally induced ruminal acidosis in sheep.

For successful treatment Broberg (1960) recommended the use of Baker's yeast and thiamine (3-4gm) in animals. But parentral use of calcium and oral alkalizers were found to be contraindicated.

Gibbon (1963) stated that for the treatment of rumen intoxication, the objectives were to evacuate the rumen, to stimulate rumen motility, to overcome intoxication, to provide supportive treatment and to restore function.

Allison *et al.* (1964) investigated the effect of intra-ruminal inoculation on adoption of lambs to a ration containing wheat. They concluded that the rate of adaptation to a new ration may be accelerated by intra-ruminal inoculation of ruminal material from animals adopted to that ration. However, a large quantity of the inoculum was required for immediate protection. Further more, ruminants do not accept raw liquor, and preparation into a palatable form might lead to a loss in the viability of the lactic acid utilizing organisms.

Dirksen (1965) recommended treatment with antihistamines, vitamin B-complex, yeast, prednisolone, fluid and electrolytes supplimentation and removal of rumen content by rumenotomy in early cases of rumen acidosis in cow.

Jensen and Mackey (1965) stated rumen evacuation, followed by administration of mineral oil in the early stage of the disease, but oral or intravenous administration of Sodium bicarbonate may also be indicated.

Hungate (1966) advocated heavy dosing with rumen liquor from normal animal for the recovery of ruminants from acute acid indigestion.

Hyldgaard-Jensen and Simesen (1966) successful treated cases of acidosis with oral administration of 8 – 10 gm Tetracycline mixed with water followed by frequent supply of small quantities of drinking water. Oral administration of Sodium bicarbonate (250 gm) in a few litres of water, to be repeated after a few hours to increased the rumen pH 4.5.

Ahrens (1967) attempted to neutralize the ruminal acidity in pear's induced ruminal acidosis in a heifer by using 450 g of Magnesium oxide, 225 g of activated charcoal and 5 million units of Crystalline penicillin. However, in this particular case the rumen pH increased to 9.72 indicating that it was given an overdose of Magnesium oxide.

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

Juhasz and Szegedi (1968c) treated severe cases of acidotic sheep by infusion of 50 – 100 ml of 10% solution of Sodium hydroxide into rumen when pH was below 4.0, 1-2g of broad spectrum antibiotic in water in severe cases, a further infusion of decreased doses of sodium hydroxide 200 ml of

25% Sodium chloride had been suggested. The additional injection of large doses of thiamine was also recommended.

Joshi (1969) treated a case of acid indigestion in a bullock by administering alkalizers (Bismuth carbonate, Magnesium carbonate and Sodium bicarbonate) orally, which raised the ruminal pH from 5.5 to 6.7.

In early Dirksen (1970) advocated discontinuation of the carbohydrate rich diet, in early cases of oral use of antibiotics with alkalizer and Baker's yeast. In severe cases parentral use of antihistamines, thiamine, normal saline with calcium magnesium gluconate and circulatory stimulant. Later on ruminal cud transplantation from healthy animal for restoration of normal flora and founa in rumen were recommended.

Gnanaprakasam (1970) treated, successfully, severe cases of ruminal acidosis in goats, by transplanting fresh rumen cud from slaughtered healthy goats alongwith antihistamines, antibiotics, thiamine, fluid and electrolyte therapy.

Treatment of ruminal acidosis of varying degree in cattle was done by Slanina *et al.,* (1970) with a proprietary preparation of buffer salts, stimulants, sodium propionate and trace elements. They found that within 48 hours of the treatment ruminal fluid and biochemical alterations in the blood returned to normal.

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

Huber (1973) achieved success in the treatment and prevention of acidosis in lamb when transplanted rumen cud from adopted sheep.

Heavy losses in terms of death in acidotic sheep in spite of treatment with calcium carbonate and agramin in mild cases, evacuation and

substitution of ruminal contents in severe cases and 10 ml of NaOH and 100 ml of water by stomach tube in acidotic lambs were recorded (Lippman, 1973).

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

Vihan *et al.* (1973) advocated the use of alkalizers only for correction of acid base balance and also suggested changes in feeding schedule and use of rumenotorics for treatment in acute acidosis of cattle.

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

Misra and Singh (1974) treated clinical cases of acid indigestion in cattle with Magnesium carbonate, ½ lb of which was given on the first day, and again on the next day if the ruminal pH had still not improved. Supportive therapy with 'Livogen' was given on alternate day for three days times. After recovery, the average ruminal fluid pH was found to increase from 4.3 to 6.2. Urine pH also showed considerable improvement from 4.4 to 7.7 after therapy.

Vestweber *et al.* (1974) mentioned that draining of the rumen contents restored normal pH in ovine ruminal acidosis. The animals given 100-750 g thiamine hydrochloride daily for 7 days was tolerated but oral dosing with a solution containing 25-75 g sodabicarbonate was ineffective.

Nauriyal (1975) treated ruminal acidosis in cross-bred cow calves and buffalo calves by removal of ruminal contents, oral administration of

antacids (Sodium hydrogen carbonate, Magnesium carbonate and Ammonium carbonate) rumenotoric drugs, broad spectrum antibiotic parentral administration of antihistaminics, thiamine and liver extract and infusion of fluid and electrolytes solution. For rapid recovery of ailing animals, transplantation of fresh ruminal cud from healthy animals was also advocated.

Prasad and Rekib (1975) managed, successfully, clinical cases of ruminal acidosis in ruminants with rumen cud and Sodium bicarbonate.

Juhasz and Szegedi (1976) used Bykodigest an antacid intraruminally for the treatment of experimentally induced ovine lactic acidosis and found in normal rumen pH, better utilization of the lactic acid in the liver and its elimination.

Mullen (1976) suggested rumenotomy, intravenous administration of 7% Sodium bicarbonate, oral antacid within four to six hrs. after overfeeding, administration of antibiotics such as Chloramphenicol or Penicillin-v mixed with grain in over feeding in cattle.

Prasad *et al.* (1976b) corrected the rumen pH with sodium bicarbonate. They administered 'Anorexon' boluses as a supportive therapy in rumen hyperacidity. One bolus was given bid daily for three days which cured acid indigestion.

Sethuraman (1976) studied acidotic buffalo and cow calves and tried oral therapy with antibiotics, antacids, ruminal cud transplantation, parentral therapy with 5% Sodium bicarbonate, Ringer's sodium lactate, 5% Vellergan, thiamine and liver extract.

Heljdasz and Nicponn (1977) prevented the appearance of symptoms of clinical acidosis and also fall of lactic acid value in rumen and blood by

the help of Penicillin (3 million IU) and Yeasts (0.5kg) administered along with beet sugar solution.

Jagos *et al.* (1977) used sodium bicarbonate and adjusted diet for treatment of chronic metabolic acidosis in dairy cows, which improved the general health, fermentation in the rumen and the milk butter fat content.

Mc Manus and Bigham (1978) stated addition of either 2% mixture of equal parts of sodium bicarbonate, potassium bicarbonate, disodium hydrogen phosphate and calcium hydrogen phosphate or one percent aluminium hydroxide helped to increase microbial population in grain-fed acid indigestion in sheep.

Kezar and Church (1979a) prevented acute acidosis in sheep and maintained higher pH value by the use of a combined therapy of Thiopeptin 0.25% plus Sodium bicarbonate 2% of the amount of wheat which were given to animal.

Muir and Barreto (1979) opined that penicillin were very active on *Streptococcus bovis* but they would not be effective when administered orally since the rumen contain an active penicillinase which will destroy the efficacy of penicillin. They considered sulphur containing peptide antibiotics to be more useful eg. Thiopeptin, sulfomycin and thistrepton.

Randhawa (1979) treated ruminal acidosis in buffalo calves with oral administration of magnesium carbonate, parentrally administration of 2.5% solution of Sodium bicarbonate, 5% Dextrose saline, Anthisan, Livogen and Berin. Oral transplantation of rumen cud from healthy animals had also been advocated.

Sethuraman and Rathor (1979a) instituted therapy at 96 hours post engorgement in experimental acid indigestion in cattle and buffaloes. In one

group, sodium bicarbonate was given orally, followed by Ringer's sodium lactate, intravenously and phenargen, intramuscularly. In the other group, magnesium carbonate was given orally followed by Sodium bicarbonate intravenously and Vellargan, intramuscularly. In both the groups, Penicillin and ruminal cud were given orally, and Thiamine hydrochloride and liver extracts, parentrally. After therapy, gradual recovery was seen. Complete recovery occurred at 192 hrs. and all treated animals were cured.

Legee (1980) successfully treated acute cases of acidosis in cows by administering a glucose-sodium chloride sodium bicarbonate mixture intravenously and chronic cases by giving freeze dried rumen juice orally.

Muir *et al.* (1980) used Thiopeptin during prevention of lactic acidosis in ovine.

Sandha (1980) treated experimental ruminal acidosis in buffalo calves with intraruminal administration of Gelusil table Live-52 powder and parentral administration of Sodium bicarbonate 5 percent solution (I/V) , Berin (I/M), Insulin (I/M) and Vetycitin (I/M). Cud transplantation from healthy animals was also recommended by him .

Mahadevan (1982) described the keeping quality of bovine rumen fluid at room and refrigeration temperatures without adding preservative. He recorded that, at room temperature fluid was suitable for transfusion for 1.80 days and at refrigeration temperature for 4.80 days .

Amstel (1983) compared the efficacy of 8 different oral antacids in clinical ruminal acidosis. Results showed, Magnesium oxide to be very potent, with the potential danger of causing severe rumen alkalosis.Calcium hydroxide and Magnesium carbonate gave satisfactony results. But the alkalinising ability of Magnesium hydroxide, Magnesium trisilicate, Calcium

corbonate, Aluminium hydroxide and Sodiom bicarbonate seemed both slow and ineffective.

Ogilvie *et al.* (1983) revealed that magnesium oxide, a commercial antacid caused systemic alkalosis in normal cattle, if they were given in therapeutic doses. Magnesium oxide was contraindicated in simple mdigestion (with a normal ; or alkaline rumen PH) and vegal indigestion, and only indicated in acidic ruminal PH.

Tanwar and Mathur (1983b) intra-ruminal administration of Magnesium carbonate (1 g/ Kg), Benzyl Penicillin (50,000 IU/ Kg) and transplantation of fresh rumen cud from healthy animal were found efficacious in acute ruminal acidosis in goats.

Schukken *et al.* (1985) claimed the recovery of acidosis in cows caused by untreated consumption of wastebakery, (a substitute for concentrates) with Sodium bicarbonate, vitamin preparation and Oxytetracycline injection. Some heifers recovered but became unthrifty and were eventually culled.

Sinha *et al.* (1985) treatment of experimentally induced ruminal acidosis in buffalo calves by intraruminal administration of Magnesium hydroxide, Teracycline soluble powder, rice gruel, molasses and cobalt sulphate along with intravenous infusion of 0.85 percent Sodium chloride and 5 percent Sodium bicarbonate proved successful in combating the malady. The use of exogenous thiamine for restoring its endogenous level and thus improving appetite was also advocated.

Howard (1986) treated acidosis in ruminats by emptying rumen contents, oral administration of antacids such as Magnesium carbonates or

Magnesium hydroxide, intravenous administration of bt balanced electrolytes and 5% sodium bicarbonate solution including antihistaminic

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

Blood and Radostits (1989) described an excellent guideline for the treatment of carbohydrate engorgement in ruminants. According to their opinion the treatment must be aimed at correction of ruminal and systemic acidosis and prevention of further production of lactic acid, restoration of fluid and electrolyte losses, maintenance of circulating blood volumes and finally restoration of forestomach and intestinal motility to normal. The rumenotomy was often indicated in peracute and acute cases, followed by sodium bicarbonate intravenously and oral as antacids.

Ahuja *et al.* (1990) produced subacute acidosis in buffaloe calves by giving molasses @ 10 g /Kg body weight orally. He treated with Monensin @ 3 mg / Kg body weight. It was found to be effective in improving rumen liquor pH and significantly decreasing its lactic acid content.

Das and Misra (1991) treated acidotic goats with Avil injection @ 1 ml I/M daily and Anorexon tablet orally daily with significant results.

Braun *et al.* (1992) in their experiment on ruminal lactic acidosis, in sheep and goat found 78.4% efficacy antacid when administered 5% $NaHCO_3$. Yeast, Chlortetracycline, I/V infusion of isotonic solulion and 5% Sodabicarb solution.

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

goats were treated with Rumbion (Herbal remedy) 0.5 tablet, twice daily which brought about recovery within 86 hrs. while the others, untreated, goat – took 168 hrs to recover.

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

Mandial *et al.* (1994). Reported good efficacy of Rumbion in experimental acid indigestion in calves.

Aslan *et al.* (1995) treated acute ruminal acidosis of goats with 1 gram Sodabicarb /kg body weight and with 1 gram Baking yeast / kg or their combination @ 0.5 gram of each per kg and reported moderate improved effect of Sodabicarbonate where as no effect of Yeast preparation.

Bhikane *et al.* (1996) treated severe ruminal lactic acidosis in a buffalo with Dextrose saline 3 lit. i.v., Ringer's lactate solution 1 lit. i.v., Sodium bicarbonate 5% 1 lit i.v, Vetalog (Sarabhai Chemicals, Baroda) 5 ml.i.m., Tonophosphon (Hoechst India Ltd., Bombay) 10ml i.m., Oxysteclin (Sarabhai Chemicals, Baroda) 20ml, i.v. within the first 5 hrs. of admission, Again The animal was 450 g each orally. The animal was again treated with Dextrose saline 1 lit. i.v., Sodium bicarbonate 1.3% 450ml. i.v., Chloropromatine (R one Poulnec Ltd., Bombay) 6 ml i.m. , Neuroxin (Cadilla Vet, Ahmadabed) 10 ml i.m. Rumen was also evecuated and washed with normal saline. During operation 2 liters of Dextrose saline i.v. and Avil (Hoechst India Ltd., Bombay) 10 ml i.m. were given.

Patra *et al.* (1997) lactic acidosis was induced in 24 healthy female sheep by feeding soaked wheat grain. One group of 6 sheep was left untreated while another group was given 5% Sodium bicarbonate solution

i.v. after 12 hrs. of grain feeding, followed by isotonic saline and Sodium bicarbonate I/V, with thiamine hydrochloride and pheniramine maleate i.m. The other 2 groups were given the therapeutic regimes and fresh cud from other sheep or floratone tablets that contain a mixture of ingredients inducing methionine, various inorganic salts, herbal preparations, vitamins and dextrose. It was concluded that the best treatment was cud transplant with parenteral treatment comprising alkalizing agents, thiamine and antihistamine.

Gokce and Imren (1998) fed four groups of sheep for 4 days with crushed wheat- wheat straw mixture supplemented with 0,3,6 and 9% Na HCO_3. Four hrs. after feeding a decrease in rumen pH was observed in all groups. At 8 hra. and there after, acidosis was observed in the control group only. It was concluded that the addition of 3, 6 and 9% $NaHCO_3$ to carbohydrate-rich diet for sheep prevented rumen acidosis.

Upadhyay and Shukla (1999) reported clinical cases to be suffering from various ruminal disorders and treated with Anigest @ 15 g orally, two time a day for 3 days and found improvement of microbial activity, ruminal tonicity and good digestion in ruminats (cow).

Dwivedi *et al.* (2000) stated that, 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, secondary rumen dysfunctions.

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CHAPTER - III

MATERIALS AND METHODS

MATERIALS AND METHODS

3.1 SITE OF PRESENT INVESTIGATION :

The present study was conducted in the Deptt. of Vety. Medicine, Bihar Veterinary College, Patna, under Rajendra Agricultural University, Pusa, Samastipur, Bihar.

3.2 SELECTION OF ANIMAL :

3.2.1 Healthy control goats :

Six apparentally healthy goats of either sex, above one year of age were procured from the Animal Supplier, B. V. College, Patna. All the goats were thoroughly examined clinically for their normal physiological status. The faeces were examined to acertain any endoparasitic infection. These goats were kept on close observation under identical, environmental and managemental conditions. During this period feeding of grasses, fodder and adlib water was provided. The temperature, pulse and respiration including the rumination and ruminal motility were observed regularly.

3.2.2 Clinically affected acidotic goats :

All the ailing goats brought to the B. V. College, Hospital with the complain of inappetance, anorexia, suspended rumination, abnormal stool, without febrile reactions were selected for the above studies. Owener's history regarding the feeding and management were recorded. A total number of 27 clinical cases showing the signs of rumen dysfunction particularly "acidotic syndrome" were selected for further detail studies as per the experimental design.

3.3 EXPERIMENTAL DESIGN :

In the present study, all the goats were divided mainly into three groups as noted below.

3.3.1 (1) Healthy control goats (group C)

This group was consisting of six healthy goats designated as group C.

3.3.2 (2) Pre-treated goats (Clinically affected)

On the basis of pH of ruminal fluid and severity of the clinical signs, all the twenty seven acidotic goats were categorised into mild moderate and severe acidotic forms.

(a) Mild acidotic goats (Group T_1)

It was consisting twelve acidotic goats and designated as group T_1.

(b) Moderate acidotic goats (Group T_2)

It was designated as group T_2 which consisting ten acidotic goats.

(e) Severe acidotic goats (Group T_3)

It was designated as group T_3 and consisting five acidotic goats.

3.3.3 (3) Post-treated goats :

The pre-treated group of goats viz. T_1, T_2 and T_3 were designated as T_1, T_2 and T_3 respectively under post treated groups.

3.4 CLINICAL FINDINGS :

The rectal temperature, pulse respiration rates etc. were recorded as described by Rosenberger *et al.* (1979). Ruminal motility and consistency were noted with the help of stethoscope or digital palpation. The degree of dehydration was noticed by feeling of the tonicity of the skin.

3.5 LABORATORY EXAMINATION :

3.5.1 Test material :

Ruminal fluid, blood and serum from healthy goats, pre-treated (clinically affected) and post-treated (recovered) goats were taken for study of different parameters.

3.6 COLLECTION AND PRESERVATION OF BIOMATERIAL OF HEALTHY CONTROL GOATS, GOATS OF PRE-TREATED AND POST-TREATED GROUPS :

3.6.1 Ruminal fluids :

About 15-20 ml of rumen fluid was obtained from left paralumber fossa by paracentesis abdominis with the help of a 6'' long needle and 10cc leur lock syringe in aseptic condition. A thin layer of liquid paraffin was spread over collected rumen fluid to prevent air contact. Sample was strained with the help of four fold muslin cloth. It was kept at 4^0C in the refrigerator.

3.6.2 Blood :

About 5 ml of blood was collected from each goat by jugular vein puncture with the help of clean, dry glass sterilized syringe and niddle and kept in vial containing sodium fluoride acted as anticoagulant.

3.6.3 Serum :

About 10 ml of the blood was collected in a sterilized serum separating tubes from each goat without any anticoagulant. The blood samples were kept in slant position for coagulation. The clot was gently separated, centrifused and clear serum was stored at 4^0C in the refrigerator.

3.7 ANALYSIS OF RUMINAL FLUID, BLOOD AND SERUM OF HEALTHY CONTROL AND PRE-TREATED AS WELL AS POST-TREATED GOATS:

3.7.1 Analysis of ruminal fluid :

3.7.1.1 pH of ruminal fluid :

The measurment of pH of ruminal fluid was done immediately after collection with the help of systronics Digital pH meter, type 335 (Systronics, Ahmedabad, India).

3.7.1.2 Motility of rumen microflora (protozoa) :

Motility of protozoa was studied as per method described by (Misra *et al.*, 1972).

3.7.1.3 Activity of rumen microflora :

Activity of rumen microflora was determined by two simple laboratory test viz.

(a) Cellulose digestion time (CDT)

(b) Sedimentation activity time (SAT)

As per the method described by Nicholas and Penn (1958). CDT and SAT were expressed in hours and minutes respectively.

3.7.2 ANALYSIS OF BLOOD :

3.7.2.1 Blood lactic acid :

Lactic acid concentration in blood was determined as per the method of Barker and Summerson (1941) and values were expressed in mg %.

3.7.2.2 Blood glucose :

Blood glucose was estimated by Folin-Wu-method as described by (Frankel *et al.*, 1970) and values were expressed in mg%.

3.7.3 ANALYSIS OF SERUM :

3.7.3.1 Serum chloride :

The serum chloride level was determined as per the method of Schales and Schales (1941) and values were expressed in mEq/l.

3.7.3.2 Serum sodium and potassium :

Sodium and Potassium concentration in the serum sample were estimated by flame-photometer as per the method described by Oser (1965) and values were expressed in mEq/l.

3.8 COLLECTION OF GROSS PHOTOGRAPH OF HEALTHY AND ACIDOTIC GOATS :

A few number of coloured photograph were collected with an exhaustive use of colour films and camera. Only good, clear and important photographs were selected.

3.9 THERAPEUTIC STUDIES :

Line of treatment adopted was as follow :

(a) Neutrilization of the ruminal acidosis by oral or parentral administration of antacids.

(b) Restoration of ruminal motility, ruminal tone and microbial motility and activity were achieved by oral administration of rumenotoric drugs (herbal preparations) and by ruminal cud from healthy goats/healthy slaughtered goats.

(c) Restoration of fluid and electrolytes on the basis of results of ionograms.

Mild acidotic goats, moderate acidotic goats and severe acidotic goats were treated 3, 5 and 7 days respectively.

3.9.1 Mild acidotic goats : (Group T_1)

All the twelve goats of this group were treated with following drugs combination.

(1) Soda bicarb @ 3-5gm once daily for 2-3 days in 50 ml of fresh water orally.

(2) Rumbion bolus @ 1 bolus bid orally for 3 days.

(3) Anigest powder 15gm. @ 5gm bid orally daily for 3 days.

(4) Vitamin B complex liquid (Ambiplex) @ 5 ml orally twice daily for 5 days.

3.9.2 Moderate acidotic goats : (Group T_2)

All the ten goats of this group were treated with following drugs.

(i) Anistamin 2 ml I/M daily for 3 days given.

(ii) Dexamithazone (Dexona) 1ml, 0.75ml and 0.5ml I/M for 3 consecutive days respectively to each goat.

(iii) A combination mixture of Soda bicarb @ 3-5 gm, Rumbion bolus @ 1 bolus and Anigest powder @ 5 gm twice daily orally given for three consecutive days. Beside these, Ambiplex, 5 ml was given orally twice daily.

(iv) Soda bicarb (7.5%) 25 ml I/V as single dose and Belamyl 2ml I/M alternate day for 3 days.

3.9.3 Severe acidotic goats : (Group T_3)

All the five goats of this group were treated with following drugs :

(i) Anistamin 10 ml. @ 2 ml I/M twice daily for three days followed by 2 ml I/M daily once for next 4 days.

(ii) Dexamithazone (Dexona) @ 1 ml, on 1st two days, days, 0.75 ml on next two days and 0.5 ml on next fifth day by I/M route to each surviving goat.

(iii) Soda bicarb (7.5 %) was given @ 50 ml I/V for two days followed by 5 gm Soda bicarb in 50 ml of fresh water twice daily by orally for two days.

(iv) A combination of rumenotoric drugs containing Rumbion, Yea-sacc bolus and Anigest powder were given @ 1 bolus, 1 bolus and 5 gm respectively, twice daily, orally for a week. Steclin bolus @ ½ bolus at 12 hrs. interval for 3 days orlly given only.

(v) Normal saline solution (0.9%) 100 ml, I/V, daily for 3 consecutive days depending upon the degree of dehydration.

(vi) Belamyl @ 2ml I/M on alternate day for 5 days were given after some improvement in the conditions of goats.

(vii) From 3rd day onward, Vit. B-Complex liquid (Ambiplex) was given @ 5 ml orally twice daily for one week.

(viii) When improvement of pH of ruminal fluid was achieved, ruminal-cud from healthy goats/healthy slaughtered goats was given by oral route @ 150 – 200 ml per day for 3 to 4 days.

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* Soda bicarb powder 100gm pocket, A product of Medikem PVT.Ltd. Math Laxamanpur, Gulgar bagh, Patna-7 (Med in India).

* Anigest Vet. powder 15gm (Sarabhai Chemicals), each 15gm contains Swertia chirata 1.50gm, Alstonia scholaris 1.15gm, Nux-vomica 0.20gm, Zingiber officinale 0.35gm, Caesalpinia bonduc 0.35gm, Vernonia anthelmintica 0.75gm, Holarrhena antidysenterica 0.75gm, Peucedanum graveolens 0.75gm, Trachys permum ammi 0.35gm, Brassica Juncea 0.75gm, Trigonella foenumgraceum 2.25gm, Sarjika kshar 1.50gm, Lavan 1.20gm, Excipients 3.15gm.

* Rumbion bolus (Indian Herbs), It contains Mahaushadh 270mg, Manjalya 180mg, Kana 135mg, Vrishketu 135mg, Amogha 135mg, Vellaj 112mg, Upkulya 112mg, Katubhadra 675mg, Chakrangi 810mg, Tiktak 540mg, Yartiktak 360mg, Dipyaka 360mg, Angark 270mg.

* Steclin bolus (Sarabhai Chemicals), it contains Tetracyeline hydrochloride 500mg per bolus.

* Injection Anistamin (Intas Pharmaceuticals Ltd.), 10ml vial, each ml contain Chorpheniramine maleate 10mg.

* Injection Sodium bicarbonate (7.5%), a product of M/S Bengal Chemicals and Pharmaceutical Ltd., Kolkota, containing 7.5g Sodium bicarbonate per 100ml.

* Vit. B-Complex liquide Ambiplex (Brihans), It contains Vit. B_1 7mg, Vit. B_2 2.5mg, Vit. B_6 1mg, Vit. B_{12} 12.5mg, biotin 25mcg, Calcium pantothenate 2.5mg, Niacin 75gm, Choline chlorinde 10mg, Methionine 10mg, lysine 20mg per 5ml.

* Yea-sac bolus (Vetcare). Division of Tetragon Chem. Ltd., Banglore.

* Injection Belamyl (Sarabhai chemicals), each ml contains thiamine hydrochloride B.Vet. C. 10 mg, Riboflavin B.Vet. C. 3mg, Niacinamide B.Vet. C 100mg, Cyanocobalamin preservative 0.57, Phenol B.Vet. C.

* Injection Dexona (Cadilla Aqrovet), It contains Dexamethazone 4mg per ml.

* Normal saline (Albert and David), It contain 0.9 %. Nacl.

3.10 STATISTICAL ANALYSIS :

The data were analysed statistically as per the method described by Snedecor and Cochran (1967).

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CHAPTER - IV

RESULTS

RESULTS

All the ailing goats brought for the treatment, at Bihar Veterinary College, Hospital with particular complain of inappetance, anorexia and suspended rumination without febrile reactions, were investigated. Owner's report viz. history related to feeding and managemental practices, type of food/fodder provided before the appearance of syndrome, were noted. In the present study it was found that, those goats which were confined to the house or kept as stall feeding or the male goats kept for ceromonial functions like 'Bakarid' etc., were suffered mostly either due to feeding of rice, boiled rice, gruel, chapati, kitchen-offals or accidental access of wheat / paddy grains.

4.1 CLINICAL FINDINGS :

On the basis of determination of pH of ruminal fluid and severity of the clinical signs, the goats, were divided into mild, moderate and severe acidotic forms.

In mild acidotic group of goats (group T_1) the ovious signs observed were, partial loss of appetite, partially suspended rumination, decreased rumen motility. Decreased rectal temperature was observed to be 102.7 ± 0.14 ^{0}F, (table 1/fig.1). Thick, firm and doughy condition of rumen on palpation of the left flank, dull sound on percussion were found and the mean pH of rumen liquor was 5.91 ± 0.10 (table 10/fig.4).

In moderate acidotic goats (group T_2), the signs observed were complete absence of rumination, dull appearance, anorexia, absence of ruminal motility, moderate constipation, rumen consistency was watery in nature, gurgling sound on auscultation of rumen, distended rumen, abdominal pain, grinding of teeth, frequent bleating, purulant nasal discharges, the mean values of rumen liquor pH and rectal temperature were

4.99 ± 0.10 and 101.9 ± 0.10 (table 10 & 1/fig.4 & 1) respectively. Increased pulse and respiration were also recorded.

In severe acidotic goats (group T_3), the signs obsorved were severe depression with pathetic appearance, anorexia, decreased ruminal pH (mean values 4.16 ± 0.09), table 10/fig.4. Complete absence of ruminal motility, continuous gurgling sound on auscultation of rumen, some goats exihibited severe constipation and other showed pasty to diarrhoric faeces. Regurgitation of undigested feed materials were also noticed. Sunken eye, staggering gait, lamness and anuria were observed. In some goats, lateral recumbency and diarrhoea were noted. Some goats of this group were showing retension of skin fold for 20 – 30 seconds. The mean value of rectal temperature was recorded 101.3 ± 0.13 (table 1/fig.1). Dysponoeic but accelerated respiration & increased pulse rate were observed.

4.1.1 Temperature :

Mean alongwith their S. E.. coefficient of variance percentage (C.V.%) and analysis of variance of temperature (^{0}F) among various groups of gaots have been given in the table 1 (fig.1) and 2 respectively. The table 1, indicates the significant ($P<0.05$) difference (columnwise) in temperature (^{0}F) among various groups of goats.

The mean value of temperature (^{0}F) in healthy control goats (group C) was observed to be 102.8 ± 0.19, whereas, in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 102.7 ± 0.14, 101.9 ± 0.10 and 101.3 ± 0.13 respectively. The mean values of temperature (^{0}F) was found to be non-significant between healthy control goats (group C) and mild acidotic goats (group T_1). However, the mean temperature (^{0}F) values in moderate

TABLE – 1. MEAN ± S. E. ALONGWITH C.V.% OF TEMPERA - TURE (^{0}F) AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$102.8^a \pm 0.19$ (06)	0.45	$102.8^a \pm 0.19$ (06)	0.45
2.	MILD ACIDOTIC (T_1)	$102.7^a \pm 0.14$ (12)	0.49	$102.7^a \pm 0.13$ (12)	0.45
3.	MODERATE ACIDOTIC (T_2)	$101.9^b \pm 0.10$ (10)	0.32	$102.5^a \pm 0.14$ (10)	0.44
4.	SEVERE ACIDOTIC (T_3)	$101.3^c \pm 0.13$ (12)	0.31	$102.4^a \pm 0.30$ (03)	0.51

Note – (i) Means (columnwise) with different superscripts differ significantly ($P<0.05$).

(ii) Figures in parentheses indicate number of observations.

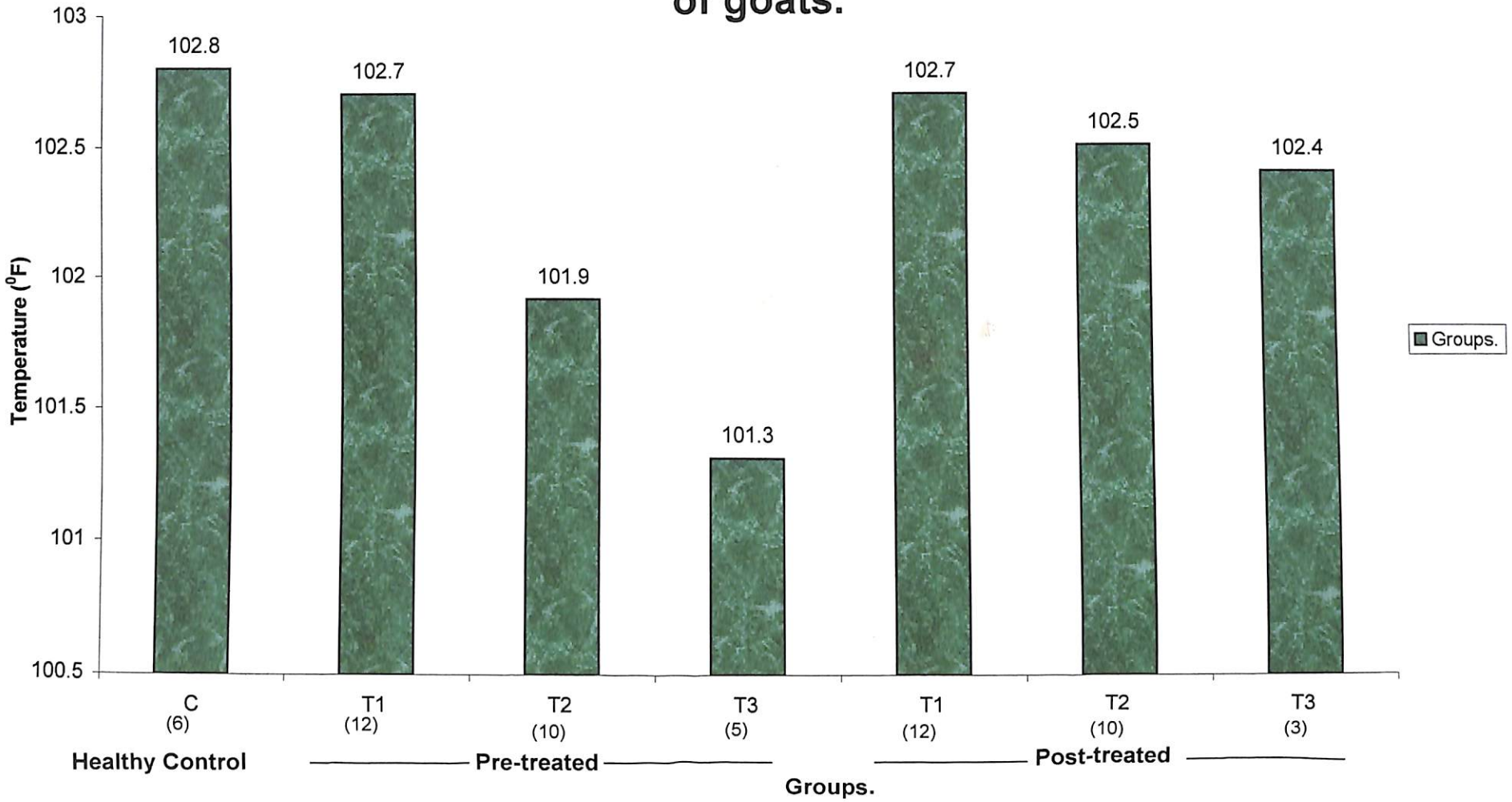
TABLE – 2. ANALYSIS OF VARIANCE OF TEMPERATURE (^{0}F) AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	3.426	18.824**	3	0.250	1.141^{NS}
ERROR	29	0.182		27	0.219	

** : Significant ($P<0.01$)

N.S. : Non-significant

Fig. 1 :Mean value of temperature (^{0}F) among various groups of goats.



acidotic goats (group T_2) and severe acidotic goats (group T_3) had been found to be decreased significantly ($P<0.05$) by 0.87 and 1.46 respectively as compared to healthy control goats (group C). Similarly, the mean values of temperature (^0F) in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be decreased significantly ($P<0.05$) by 0.87 and 1.44 respectively as compared to mild acidotic goats (group T_1). Likewise, the mean value of temperature (^0F) in severe acidotic goats (group T_3) was found to be decreased significantly ($P<0.05$) by 0.59 as compared to moderate acidotic goats (group T_2).

The mean values of temperature (^0F) in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 102.7 ± 0.13, 102.5 ± 0.14 and 102.4 ± 0.30 respectively. These values were found to be non-significant as compared to healthy control goats (group C).

4.1.2 Pulse :

Mean alongwith their S. E., coefficient of variance percentage (C.V.%) and analysis of variance of the pulse per minute among various groups of goats have been given in the table 3 (fig.2) and 4 respectively. Table 3 indicate the significant ($P<0.01$) difference (columnwise) in pulse per minute among various groups of goats.

The mean value of pulse per minute in healthy control goats (group C) was found to be 73 ± 2.06. Whereas, in pre-treated groups viz. Mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 123.5 ± 1.40, 133.9 ± 0.47 and 142 ± 1.51 respectively. The mean values of pulse per minute of mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats

TABLE – 3. MEAN ± S. E. ALONGWITH C.V.% OF PULSE PER MINUTE AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$73^{a} \pm 2.06$ (06)	6.93	$73^{a} \pm 2.06$ (06)	6.93
2.	MILD ACIDOTIC (T_1)	$123.5^{b} \pm 1.40$ (12)	3.94	$75.0^{a} \pm 1.48$ (12)	6.87
3.	MODERATE ACIDOTIC (T_2)	$133.9^{c} \pm 0.47$ (10)	1.66	$77.2^{a} \pm 1.8$ (10)	7.37
4.	SEVERE ACIDOTIC (T_3)	$142.0^{d} \pm 1.51$ (05)	2.38	$78.3^{a} \pm 1.45$ (03)	3.21

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.01$).

(ii) Figures in parentheses indicate number of observations.

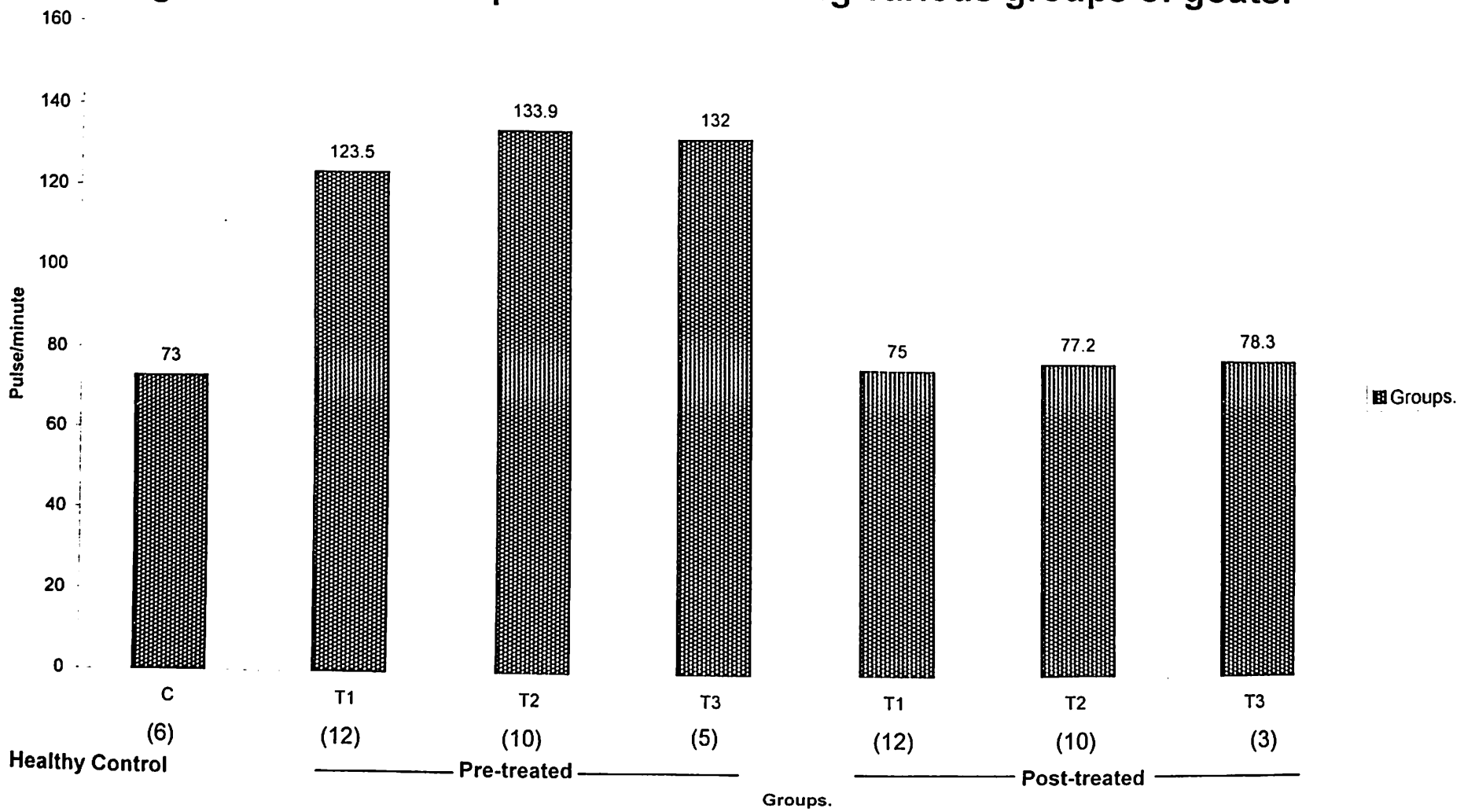
TABLE – 4. ANALYSIS OF VARIANCE OF PULSE PER MINUTE AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	5916.881	252.383**	3	30.530	1.136^{NS}
ERROR	29	23.444		27	26.856	

** : Significant ($P<0.01$)

N.S. : Non-significant

Fig. 2: Mean value of pulse/minute among various groups of goats.



(group T_3) were found to be increased significantly ($P<0.01$) by 50.5, 60.9 and 69.0 respectively, as compared to healthy control goats (group C). Similarly, the mean values of pulse per minute in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.01$) by 10.4 and 18.5 respectively, as compared to mild acidotic goats (group T_1). Likewise, the mean values of pulse per minute of severe acidotic goats (group T_3) was found to be higher significantly ($P<0.01$) by 8.1, as compared to healthy control goats (group C).

The mean values of pulse per minute in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 75.0 ± 1.48, 77.2 ± 1.8 and 78.3 ± 1.45 respectively. These values were found to be non-significant, as compared to healthy control goats (group C).

4.1.3 Respiration :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of respiration per minute among various groups of goats have been given in the table 5 (fig.3) and 6 respectively. Table 5 indicates the significant ($P<0.01$) difference (columnwise) in respiration per minute among various groups of goats.

The mean value of respiration per minute in healthy control goats (group C) was found to be 21.66 ± 1.14 whereas, in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 24.58 ± 0.55, 31.6 ± 0.142 and 36.8 ± 1.15 respectively. The mean values of respiration per minute of mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly

TABLE – 5. MEAN ± S. E. ALONGWITH C.V.% OF RESPIRATION PER MINUTE AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$21.66^{a} \pm 1.14$ (06)	12.94	$21.66^{a} \pm 1.14$ (06)	12.94
2.	MILD ACIDOTIC (T_1)	$24.58^{b} \pm 0.55$ (12)	7.84	$22.5^{a} \pm 0.75$ (12)	11.60
3.	MODERATE ACIDOTIC (T_2)	$31.6^{c} \pm 0.142$ (10)	4.27	$23.5^{a} \pm 0.67$ (10)	9.02
4.	SEVERE ACIDOTIC (T_3)	$36.8^{d} \pm 1.15$ (05)	7.03	$24.0^{a} \pm 1.15$ (03)	8.33

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.01$).

(ii) Figures in parentheses indicate number of observations.

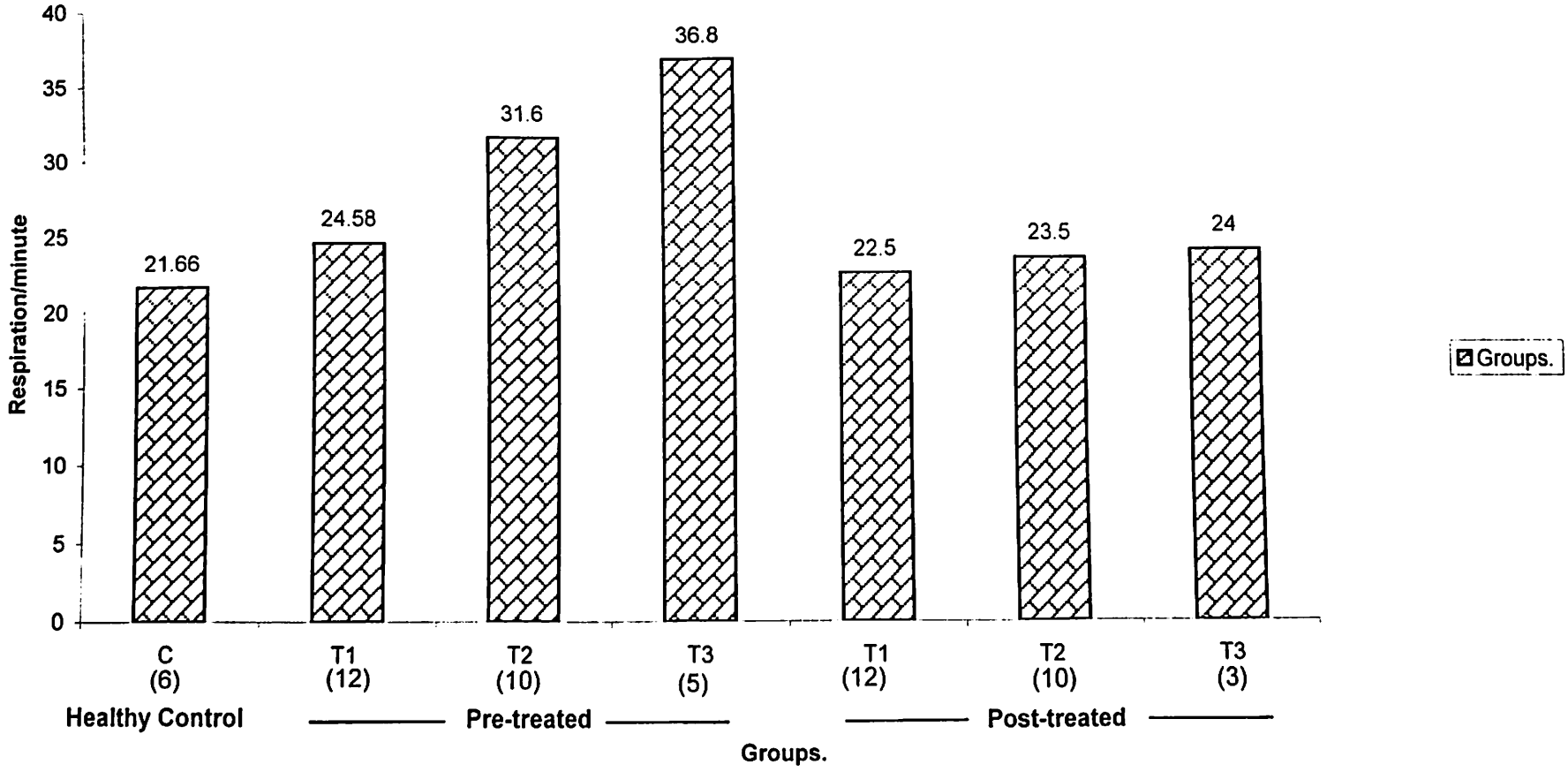
TABLE – 6. ANALYSIS OF VARIANCE OF RESPIRATION PER MINUTE AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	299.173	70.294**	3	6.001	0.995^{NS}
ERROR	29	4.256		27	6.030	

** : Significant ($P<0.01$)

N.S. : Non-significant

Fig. 3: Mean value of respiration/minute among various groups of goats.



($P<0.01$) by 2.92, 9.94 and 15.14 respectively, as compared to healthy control goats (group C). The mean values of respiration per minute in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.01$) by 7.02 and 12.22 respectively, as compared to mild acidotic goats (group T_1). Similarly, the mean values of respiration per minute of severe acidotic goats (group T_3) was found to be higher significantly ($P<0.01$) by 5.2 as compared to moderate acidotic goats (group T_2).

The mean values of respiration per minute in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 22.5 ± 0.75, 23.5 ± 0.67 and 24.0 ± 1.15 respectively. These values were found to be non-significant, as compared to the healthy control goats (group C).

4.1.4 Rumen motility :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of rumen motility/5 minutes among various groups of goats have been given in the table 7 and 8 respectively. Table 7 indicates the significant ($P<0.01$) difference (columnwise) in rumen motility/5 minutes among various groups of goats.

The mean values of rumen motility / 5 minutes in healthy control goats (group C) was observed to be 7.66 ± 0.49 whereas, in pre-treated group viz. mild acidotic goats (group T_1) was found to be 2.91 ± 0.25. But the rumen motility was found to be absent in both moderate acidotic goats (group T_2) and severe acidotic goats (group T_3).

TABLE – 7. MEAN ± S. E. ALONGWITH C.V.% OF RUMINAL MOTILITY PER 5 MINUTES AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$7.66^{a} \pm 0.49$ (06)	15.79	$7.66^{a} \pm 0.49$ (06)	15.79
2.	MILD ACIDOTIC (T_1)	$2.91^{b} \pm 0.25$ (12)	30.86	$7.41^{a} \pm 0.35$ (12)	16.72
3.	MODERATE ACIDOTIC (T_2)	--	--	$6.80^{a} \pm 0.32$ (10)	15.18
4.	SEVERE ACIDOTIC (T_3)	--	--	$6.66^{a} \pm 0.88$ (03)	22.91

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.01$).

(ii) Figures in parentheses indicate number of observations.

TABLE – 8. ANALYSIS OF VARIANCE OF RUMINAL MOTILITY PER 5 MINUTES AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	1	90.25	88.916**	3	1.440	1.009^{NS}
ERROR	16	1.015		27	1.426	

** : Significant ($P<0.01$)

N.S. : Non-significant

The mean value of rumen motility / 5 minutes of mild acidotic goats (group T_1) was found to be decreased significantly ($P<0.01$) by 4.75 as compared to healthy control goats (group C).

The mean values of rumen motility / 5 minutes in pos-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 7.41 ± 0.35, 6.80 ± 0.32 and 6.66 ± 0.88 respectively. All these values were found to be non-significant, as compared to healthy control goats (group C).

4.1.5 Rumen consistency :

The table no. 9 indicates rumen consistency among various groups of goats.

Viscous to semi-liquid rumen consistency was found in healthy control goats (group C). Whereas. thick, firm and doughy like rumen consistency was found in pre-treated group viz. mild acidotic goats (group T_1). But watery rumen consistency was found in moderate acidotic goats (group T_2) and severe acidotic groups of goats (group T_3).

Viscous or semi-liquid rumen consistency was recorded by digital palpation in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3).

4.2 EXAMINATION OF RUMINAL FLUID :-

4.2.1 pH of ruminal fluid :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of pH of ruminal fluid among various groups of goats, have been given in table 10 (fig.4) and 11 respectively. Table 10, indicates the significant ($P<0.01$) difference (columnwise) in pH of ruminal fluid among various groups of goats.

TABLE- 9. RUMEN CONSISTENCY IN HEALTHY, PRE-TREATED AND POST-TREATED GOATS.

HEALTHY CONTROL GOATS	PRE-TREATED GOATS			POST-TREATED GOATS		
C	MILD ACIDOTIC T_1	MODERATE ACIDOTIC T_2	SEVERE ACIDOTIC T_3	MILD ACIDOTIC T_1	MODERATE ACIDOTIC T_2	SEVERE ACIDOTIC T_3
1. Viscous	1. Thick/firm and doughy	1. Watery	1. Watery	1. Viscous	1. Viscous	1. Viscous
2. Semiliquid	2. Thick/firm and doughy	2. Watery	2. Watery	2.Semiliquid	2. Viscous	2.Semiliquid
3. Viscous	3. Thick/firm and doughy	3. Watery	3. Watery	3. Viscous	3.Semiliquid	2. Died
4. Viscous	4. Thick/firm and doughy	4. Watery	4. Watery	4. Viscous	4. Viscous	3. Viscous
5. Semiliquid	5. Thick/firm and doughy	5. Watery	5. Watery	5.Semiliquid	5. Viscous	4. Died
6. Vicous	6. Semiliquid	6. Watery		6. Viscous	6. Viscous	
	7. Thick/firm and doughy	7. Watery		7. Viscous	7. Viscous	
	8. Thick/firm and doughy	8. Watery		8.Semiliquid	8. Viscous	
	9. Thick/firm and doughy	9. Watery		9.Viscous	9. Viscous	
	10. Semiliquid	10.Watery		10.Viscous	10. Viscous	
	11.Thick/firm and doughy			11.Viscous		
	12.Thick/firm and doughy			12. Viscous		

TABLE – 10. MEAN ± S. E. ALONGWITH C.V.% OF pH OF RUMINAL FLUID AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$6.96^{a} \pm 0.06$ (06)	2.16	$6.96^{a} \pm 0.06$ (06)	2.16
2.	MILD ACIDOTIC (T_1)	$5.91^{b} \pm 0.10$ (12)	6.10	$6.94^{a} \pm 0.05$ (12)	2.84
3.	MODERATE ACIDOTIC (T_2)	$4.99^{c} \pm 0.10$ (10)	6.36	$6.92^{a} \pm 0.06$ (10)	2.79
4.	SEVERE ACIDOTIC (T_3)	$4.16^{d} \pm 0.09$ (05)	4.98	$6.9^{a} \pm 0.05$ (03)	1.0

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.01$).

(ii) Figures in parentheses indicate number of observations.

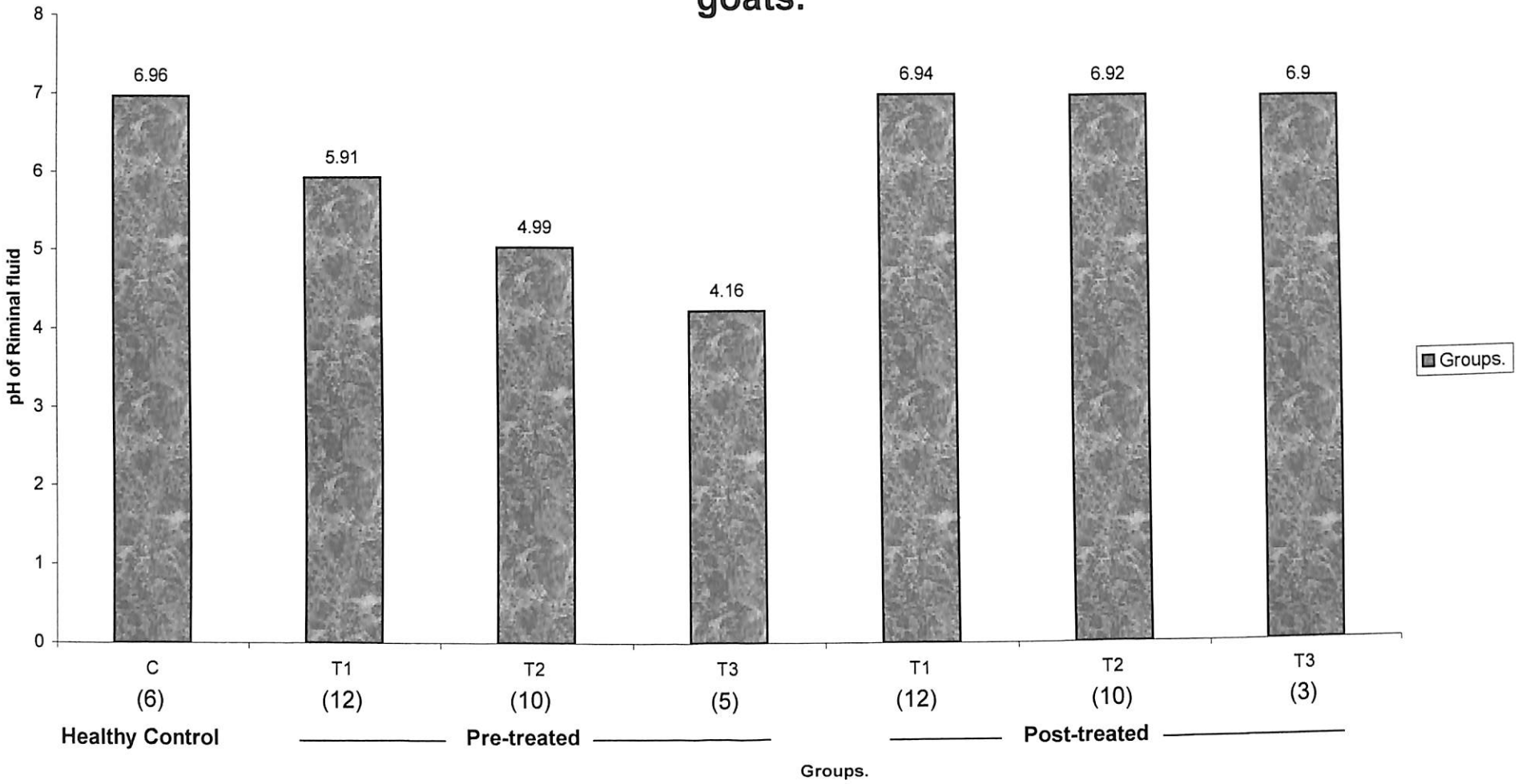
TABLE – 11. ANALYSIS OF VARIANCE OF pH of RUMINAL FLUID AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	8.815	97.944**	3	4.166	0.126^{NS}
ERROR	29	0.090		27	0.033	

** : Significant ($P<0.01$)

N.S. : Non-significant

Fig. 4: Mean values of pH of ruminal fluid among various groups of goats.



The mean value of pH, of ruminal fluid in healthy control goats (group C) was found to be 6.96 ± 0.06 whereas, in pre-treated groups viz. mild acidotic goats (group T_1) moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) the values were found to be 5.91 ± 0.10, 4.99 ± 0.10 and 4.16 ± 0.09 respectively. The mean values of pH of ruminal fluid in mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be decreased significantly ($P<0.01$) by 1.05, 1.97 and 2.8 respectively, as compared to healthy control goats (group C). Similarly, the mean values of pH of ruminal fluid in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be decreased significantly ($P<0.01$) by 0.92 and 1.75 respectively, as compared to mild acidotic goats (group T_1). Likewise, the mean value of pH of ruminal fluid in severe acidotic goats (group T_3) was found to be lowered significantly ($P<0.01$) by 0.83, when compared to moderate acidotic goats (group T_2).

The mean values of pH of ruminal fluid in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 6.94 ± 0.05, 6.92 ± 0.06 and 6.9 ± 0.05 respectively. These values were found to be non-significant as compared to healthy control goats (group C).

4.2.2 Motility of rumen microflora (Protozoa) :

The motility of rumen protozoa have been given in the table 12. The motility of rumen protozoa was examined microscopically and graded under eyes as absent (-), slow/sluggish (+), moderate (++) and vigorous (+++).

Vigorous motility (+++) was observed in healthy control goats (group C). In pre-treated groups viz. mild acidotic goats (group T_1) the motility was

TABLE- 12. MOTILITY OF RUMEN MICROFLORA (PROTOZOA) IN HEALTHY, PRE-TREATED AND POST TREATED GOATS.

HEALTHY CONTROL GOATS	PRE-TREATED GOATS			POST-TREATED GOATS		
C	MILD ACIDOTIC T_1	MODERATE ACIDOTIC T_2	SEVERE ACIDOTIC T_3	MILD ACIDOTIC T_1	MODERATE ACIDOTIC T_2	SEVERE ACIDOTIC T_3
1. +++	1. –	1. –	1. –	1. +++	1. +++	1. ++
2. +++	2. +	2. –	2. –	2. +++	2. +++	2. ++
3. +++	3. –	3. –	3. –	3. +++	3. +++	3. Died
4. +++	4. +	4. –	4. –	4. ++	4. ++	4. ++
5. +++	5. –	5. –	5. –	5. +++	5. +++	5. Died
6. +++	6. +	6. –		6. +++	6. +++	
	7. –	7. –		7. +++	7. +++	
	8. +	8. –		8. +++	8. ++	
	9. –	9. –		9. ++	9. +++	
	10. –	10. –		10.+++	10.+++	
	11. –			11.+++		
	12. +			12.+++		

Note: - - = Absent
+ = Slow/sluggish
++ = Moderate
+++ = Vigorous.

absent (-) to sluggish (+) but, complete absence of protozoal motility (-), were observed in moderate and severe acidotic goats (group T_2 &T_3).

The protozoal motility, in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) were found to be moderate (++) to vigorous (+++), but only moderate (++) protozoal motility was recorded in severe acidotic goats (group T_3).

4.2.3 Activity of rumen microflora :

4.2.3.1 Cellulose digestion time (CDT) :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of CDT (hrs.) among various groups of goats have been given in the table 13 and 14 respectively. Table 13, indicates significant ($P<0.01$) difference (columnwise) in CDT (hrs.) among various groups of goats.

The mean values of CDT (hrs.) in healthy control goats (group C) was found to be 44.33 ± 1.05 whereas, in pre-treated groups viz. mild acidotic goats (group T_1) the value observed was 76.33 ± 1.38. The mean values of CDT (hrs.) in mild acidotic goats (group T_1) was found to be significantly high ($P<0.01$) by 32.00 as compared to healthy control goats (group C). cellulose digestion time (hrs.) was not observed in moderate and severe acidotic goats (group T_2 & T_3).

The mean values of CDT (hrs.) in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 45.75 ± 0.65, 48.7 ± 1.06 and 49.66 ± 1.20 respectively. All these values were found to be non-significant, as compared to healthy control goats (group C).

TABLE – 13. MEAN ± S. E. ALONGWITH C.V.% OF ACTIVITY OF RUMEN MICROFLORA (CDT) IN HOURS VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$44.33^{a} \pm 1.05$ (06)	5.82	$44.33^{a} \pm 1.05$ (06)	5.82
2.	MILD ACIDOTIC (T_1)	$76.33^{b} \pm 1.38$ (12)	6.27	$45.75^{a} \pm 0.65$ (12)	4.94
3.	MODERATE ACIDOTIC (T_2)	--	--	$48.7^{a} \pm 1.06$ (10)	6.91
4.	SEVERE ACIDOTIC (T_3)	--	--	$49.66^{a} \pm 1.20$ (03)	4.19

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.01$).

(ii) Figures in parentheses indicate number of observations.

TABLE – 14. ANALYSIS OF VARIANCE OF ACTIVITY OF RUMEN MICROFLORA (CDT) IN HOURS AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	1	4096	229.146**	3	10.205	0.050^{NS}
ERROR	16	17.875		27	200.3492	

** : Significant ($P<0.01$)

N.S. : Non-significant

4.2.3.2 Sedimentation activity time (SAT) :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of SAT (minutes) among various groups of goats have been given in the table 15 and 16 respectively. Table 15, indicates the significantly ($P<0.01$) difference (columnwise) in SAT (minutes) among various groups of goats.

The mean value of SAT (minutes) in healthy control goats (group C) was found to be 24.83 ± 1.55 whereas, in pre-treated groups viz. mild acidotic goats (group T_1) was 67.08 ± 0.75. The mean value of SAT (minutes) in mild acidotic goats (group T_1) was found to be higher significantly ($P<0.01$) by 42.25, as compared to healthy control goats (group C). sedimentation activity time (minutes) was not observed in moderated and severe adidotic goats (group T_2 & T_3).

The mean values of SAT (minutes) in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 25.16 ± 0.72, 27.1 ± 1.04 and 29.33 ± 1.45 respectively. The values of post treated groups were found to be non-significant as compared to healthy control goats (group C).

4.3 EXAMINATION OF BLOOD / SERUM :

4.3.1 Blood lactic acid :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of blood lactic acid concentration (mg%) among various groups of goats have been given in the table 17 (fig. 5) and 18 respectively. Table 17, indicates the significant ($P<0.05$) difference (columnwise) in blood lactic acid level among various groups of goats.

TABLE – 15. MEAN ± S. E. ALONGWITH C.V.% OF ACTIVITY OF RUMEN MICROFLORA (SAT) IN MINUTES AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$24.83^{a} \pm 1.55$ (06)	15.36	$24.83^{a} \pm 1.55$ (06)	15.36
2.	MILD ACIDOTIC (T_1)	$67.08^{b} \pm 0.75$ (12)	3.89	$25.16^{a} \pm 0.72$ (12)	9.99
3.	MODERATE ACIDOTIC (T_2)	--	--	$27.1^{a} \pm 1.04$ (10)	12.23
4.	SEVERE ACIDOTIC (T_3)	--	--	$29.33^{a} \pm 1.45$ (03)	8.57

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.01$).

(ii) Figures in parentheses indicate number of observations.

TABLE – 16. ANALYSIS OF VARIANCE OF ACTIVITY OF RUMEN MICROFLORA (SAT) IN MINUTES AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	1	7140.25	773.225**	3	20.472	2.175^{NS}
ERROR	16	9.234375		27	9.409	

** : Significant ($P<0.01$)

N.S. : Non-significant

TABLE – 17. MEAN ± S. E. ALONGWITH C.V.% OF BLOOD LACTIC ACID (mg%) AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$12.91^{a} \pm 1.02$ (06)	19.42	$12.91^{a} \pm 1.02$ (06)	19.42
2.	MILD ACIDOTIC (T_1)	$18.16^{b} \pm 1.03$ (12)	19.81	$13.51^{a} \pm 0.79$ (12)	20.43
3.	MODERATE ACIDOTIC (T_2)	$25.99^{c} \pm 1.11$ (10)	13.54	$14.03^{a} \pm 1.00$ (10)	22.69
4.	SEVERE ACIDOTIC (T_3)	$41.44^{d} \pm 2.85$ (05)	15.37	$14.43^{a} \pm 1.81$ (03)	21.80

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.05$).

(ii) Figures in parentheses indicate number of observations.

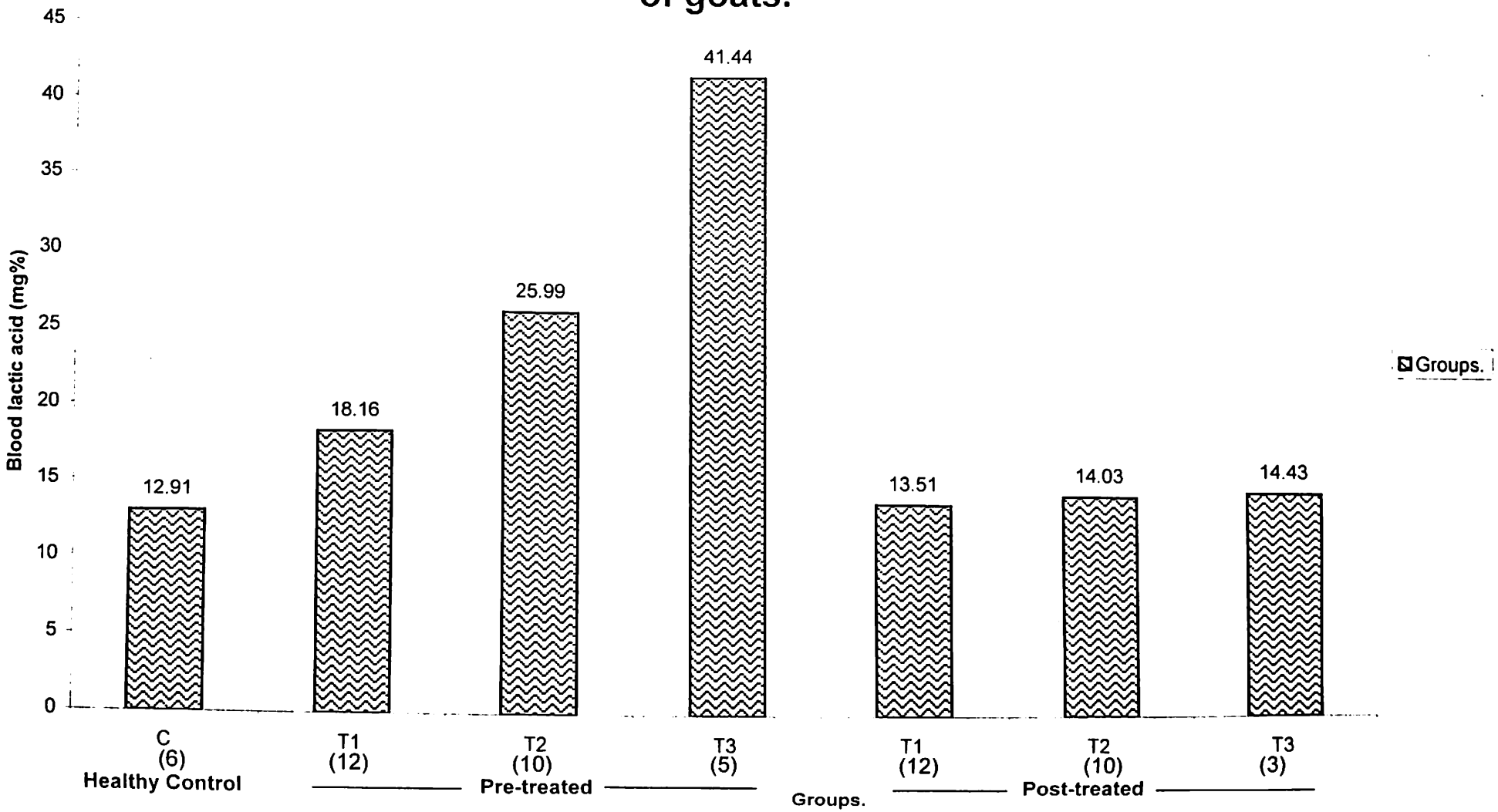
TABLE – 18. ANALYSIS OF VARIANCE OF BLOOD LACTIC ACID (mg%) AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	639.643	41.403**	3	2.246	0.267^{NS}
ERROR	29	15.449		27	8.385	

** : Significant ($P<0.01$)

N.S. : Non-significant

Fig. 5: Mean values of blood lactic acid (mg%) among various groups of goats.



The mean value of blood lactic acid concentration (mg%) in healthy control goats (group C) was found to be 12.91 ± 1.02 whereas, in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 18.16 ± 1.03, 25.99 ± 1.11 and 41.44 ± 2.85 respectively. The mean values of blood lactic acid concentration (mg%) of mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.05$) by 5.24, 13.08 and 28.53 respectively as compared to healthy control goats (group C). Similarly, the mean values of blood lactic acid concentration (mg%) of moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.05$) by 7.83 and 23.28 respectively as compared to mild acidotic goats (group T_1). The mean value of blood lactic acid concentration (mg%) of severe acidotic goats (group T_3) was found to be significantly ($P<0.05$) high by 15.45 as compared to moderate acidotic goats (group T_2).

The mean values of blood lactic acid concentration (mg%) in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 13.51 ± 0.79, 14.03 ± 1.00 and 14.43 ± 1.81 respectively. All these values were found to be non-significant, as compared to healthy control goats (group C).

4.3.2 Blood glucose :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of blood glucose level (mg%) among various groups of goats have been given in the table 19 (fig. 6) and 20 respectively. Table 19, indicates the significant ($P<0.01$) difference (columnwise) in blood glucose level among various groups of goats.

TABLE – 19. MEAN ± S. E. ALONGWITH C.V.% OF BLOOD GLUCOSE (mg%) AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$52.59^{a} \pm 2.05$ (06)	9.58	$52.59^{a} \pm 2.05$ (06)	9.58
2.	MILD ACIDOTIC (T_1)	$56.82^{b} \pm 1.44$ (12)	8.78	$53.73^{a} \pm 1.17$ (12)	7.57
3.	MODERATE ACIDOTIC (T_2)	$85.52^{c} \pm 1.46$ (10)	5.42	$54.01^{a} \pm 1.14$ (10)	6.69
4.	SEVERE ACIDOTIC (T_3)	$112.38^{d} \pm 4.22$ (05)	8.40	$54.58^{a} \pm 1.14$ (03)	4.72

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.01$).

(ii) Figures in parentheses indicate number of observations.

TABLE – 20. ANALYSIS OF VARIANCE OF BLOOD GLUCOSE (mg%) AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	4985.998	151.831**	3	3.568	0.219^{NS}
ERROR	29	32.839		27	16.270	

** : Significant ($P<0.01$)

N.S. : Non-significant

The mean value of blood glucose level (mg%) in healthy control goats (group C) was found to be 52.59 ± 2.05, whereas, in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) the values were found to be 56.82 ± 1.44, 85.52 ± 1.46 and 112.38 ± 4.22 respectively. The mean values of blood glucose level (mg%) of mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.01$) by 8.23, 32.92 and 59.78 respectively as compared to healthy control goats (group C). The mean values of blood glucose level (mg%) of moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.01$) by 28.69 and 55.55 as compared to mild acidotic goats (group T_1). The mean value of blood glucose level (mg%) of severe acidotic goats (group T_3) was found to be significantly ($P<0.01$) high by 26.86 as compared to moderate acidotic goats (group T_2).

The mean values of blood glucose level (mg%) in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 53.73 ± 1.17, 54.01 ± 1.14 and 54.58 ± 1.14 respectively. These values were found to be non-significant, as compared to healthy control goats (group C).

4.3.3 Serum chloride :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of serum chloride level (mEq/l) among various groups of goats have been given in the table 21 (fig. 7) and 22 respectively. Table 21, indicates significant ($P<0.05$) difference (columnwise) in serum chloride level (mEq/l) among different groups of goats.

TABLE – 21. MEAN ± S. E. ALONGWITH C.V.% OF SERUM CHLORIDE (mEq/l) AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$105.98^{a} \pm 1.76$ (06)	4.08	$105.98^{a} \pm 1.76$ (06)	4.08
2.	MILD ACIDOTIC (T_1)	$110.51^{a} \pm 1.79$ (12)	5.62	$105.07^{a} \pm 0.97$ (12)	3.21
3.	MODERATE ACIDOTIC (T_2)	$117.51^{b} \pm 1.34$ (10)	3.60	$106.51^{a} \pm 1.13$ (10)	3.37
4.	SEVERE ACIDOTIC (T_3)	$126.99^{c} \pm 1.89$ (05)	3.33	$107.33^{a} \pm 1.68$ (03)	2.72

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.05$).

(ii) Figures in parentheses indicate number of observations.

TABLE – 22. ANALYSIS OF VARIANCE OF SERUM CHLORIDE (mEq/l) AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	498.498	19.214**	3	6.011	0.459^{NS}
ERROR	29	25.944		27	13.079	

** : Significant ($P<0.01$)

N.S. : Non-significant

The mean value of serum chloride level (mEq/l) in healthy control goats (group C) was found to be 105.98 ± 1.76 whereas, the values in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 110.51 ± 1.79, 117.51 ± 1.34 and 126.99 ± 1.89 respectively. The mean value of serum chloride level (mEq/l) was found to be non-significant increased between healthy control goats (group C) and mild acidotic goats (group T_1). However, the mean values of serum chloride level (mEq/l) in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.05$) by 11.53 and 21.009 respectively, as compared to healthy control goats (group C). Similarly, the mean values of serum chloride level (mEq/l) in moderate acidotic goats (group T_2) and severe acidotic goat (group T_3) were found to be increased significantly ($P<0.05$) by 6.99 and 16.47 respectively, as compared to mild acidotic goats (group T_1). The mean value of serum chloride level (mEq/l) in severe acidotic goats (group T_3) was found to be increased significantly ($P<0.05$) by 9.47 as compared to moderate acidotic goats (group T_2).

The mean values of serum chloride level (mEq/l) in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were recorded to be 105.07 ± 0.97, 106.51 ± 1.13 and 107.33 ± 1.68 respectively. All these values were found to be non-significant, as compared to healthy control goats (group C).

4.3.4 Serum sodium :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of serum sodium level (mEq/l) among various groups of goats have been given in the table 23 (fig. 8) and 24 respectively.

Table 23, indicates significant ($P<0.05$) difference (columnwise) in serum sodium level (mEq/l) among different groups of goats.

The mean value of serum sodium level (mEq/l) in healthy control goats (group C) was found to be 140 ± 2.12 whereas, in pre-treated groups viz. mild acidotic goats (group T_1) moderate acidotic goats (group T_2) and severe acidotic goats (group T_3), the values were found to be 142 ± 1.18, 149 ± 1.72 and 165 ± 1.93 respectively. The mean values of serum sodium level (mEq/l) were found to be non-significant between healthy control goats (group C) and mild acidotic goats (group T_1). However, the mean values of serum sodium level (mEq/l) in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.05$) by 8.30 and 24.95 respectively as compared to healthy control goats (group C). Similarly, the mean values of serum sodium level (mEq/l) in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.05$) by 6.91 and 23.57 respectively as compared to mild acidotic goats (group T_1). Likewise, the mean values of serum sodium level in severe acidotic goats (group T_3) was found to be increased significantly ($P<0.05$) by 16.65 as compared to moderate acidotic goats (group T_2).

The mean values of serum sodium level (mEq/l) in post-treated groups viz. mild acidotic goats (group T_1), Moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were observed to be 141 ± 1.38, 143 ± 1.77 and 145 2.39 respectively. These values were found to be non-significant, as compared to healthy control goats (group C).

TABLE – 23. MEAN ± S. E. ALONGWITH C.V.% OF SERUM SODIUM (mEq/l) AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$140^{a} \pm 2.12$ (06)	3.69	$140^{a} \pm 2.12$ (06)	3.69
2.	MILD ACIDOTIC (T_1)	$142^{a} \pm 1.18$ (12)	2.89	$141^{a} \pm 1.38$ (12)	3.40
3.	MODERATE ACIDOTIC (T_2)	$149^{b} \pm 1.72$ (10)	3.65	$143^{a} \pm 1.77$ (10)	3.91
4.	SEVERE ACIDOTIC (T_3)	$165^{c} \pm 1.93$ (05)	2.60	$145^{a} \pm 2.39$ (03)	2.85

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.05$).

(ii) Figures in parentheses indicate number of observations.

TABLE – 24. ANALYSIS OF VARIANCE OF SERUM SODIUM (mEq/l) AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	766.809	33.455**	3	18.076	0.689^{NS}
ERROR	29	22.920		27	26.201	

** : Significant ($P<0.01$)

N.S. : Non-significant

4.3.5 Serum potassium :

Mean alongwith their S.E., coefficient of variance percentage (C.V.%) and analysis of variance of serum potassium level (mEq/l) among various groups of goats have been given in the table 25 (fig. 9) and 26 respectively. Table 25, indicates significant ($P<0.05$) difference (columnwise) in serum potassium level (mEq/l) among different groups of goats.

The mean value of serum potassium level (mEq/l) in healthy control goats (group C) was found to be 4.27 ± 0.22 whereas, in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 4.57 ± 0.09, 3.88 ± 0.07 and 3.29 ± 0.10 respectively. The mean values of serum potassium level (mEq/l) were found to be non-significant increased between healthy control goats (group C) and mild acidotic goats (group T_1). However, the mean values of serum potassium level (mEq/l) in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.05$) by 0.385 and 0.98 respectively, as compared to healthy control goats (group C). Similarly, the mean values of serum potassium level (mEq/l) in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be increased significantly ($P<0.05$) by 0.994 and 1.589 respectively as compared to mild acidotic goats (group T_1). The mean value of serum potassium level (mEq/l) in severe acidotic goats was found to be increased significantly ($P<0.05$) by 0.595 as compared to moderate acidotic goats (group T_2).

The mean values of serum potassium level (mEq/l) in post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 4.44 ± 0.12, 4.23 ±

TABLE – 25. MEAN ± S. E. ALONGWITH C.V.% OF SERUM POTASSIUM (mEq/l) AMONG VARIOUS GROUPS OF GOATS.

S.N.	GROUPS	PRE-TREATED		POST - TREATED	
		MEAN ± S.E.	C. V. %	MEAN ± S.E.	C. V. %
1.	HEALTHY CONTROL (C)	$4.27^{a} \pm 0.22$ (06)	12.78	$4.27^{a} \pm 0.22$ (06)	12.78
2.	MILD ACIDOTIC (T_1)	$4.57^{a} \pm 0.09$ (12)	6.80	$4.44^{a} \pm 0.12$ (12)	9.41
3.	MODERATE ACIDOTIC (T_2)	$3.88^{b} \pm 0.07$ (10)	6.24	$4.23^{a} \pm 0.13$ (10)	10.36
4.	SEVERE ACIDOTIC (T_3)	$3.29^{c} \pm 0.10$ (05)	6.83	$4.10^{a} \pm 0.40$ (03)	16.87

Note – (i) Means(columnwise) with different superscripts differ significantly ($P<0.05$).

(ii) Figures in parentheses indicate number of observations.

TABLE – 26. ANALYSIS OF VARIANCE OF SERUM POTASSIUM (mEq/l) AMONG VARIOUS GROUPS OF GOATS.

SOURCE OF VARIATION	PRE-TREATED			POST-TREATED		
	D. F.	M S	F	D. F.	M S	F
BETWEEN TREATEMENT	3	2.220	18.8**	3	0.143	0.632^{NS}
ERROR	29	.118		27	0.226	

** : Significant ($P<0.01$)

N.S. : Non-significant

0.13 and 4.10 ± 0.40 respectively. These values were found to be non-significant, as compared to healthy control goats (group C).

4.4 THERAPEUTIC STUDIES :

In the present study, the result of post-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) have been depicted in table 1 to 26.

The mild acidotic goats (group T_1) were treated with a combination mixture of Sodabicarb @ 3 – 5 gm, Rumbion bolus @ 1 bolus and Anigest powder @ 5 gm twice daily, orally, for three consecutive days. Besides these the goats were also administered Ambiplex @ 5 ml, orally, twice daily for 5 days. After completion of treatment, the mean values of various parameters studied were found to be non-significant, as compared to healthy control goats (group C) table 1 to 26. All the twelve goats of this group were recovered completely without relapses.

The goats of moderately affected group (group T_2) were treated as above. In addition to that, these goats were also given Anistamin 2 ml I/m daily for 3 days, Dexona 1 ml, 0.75ml and 0.5ml I/M for 3 conscutive days respectively to each goat, Sodabicarb (7.5 %) 10 ml I/V, single dose and Belamyl 2 ml I/M, alternate day, for 3 days. All the goats responded well to the treatment and recovered completely and the appetite returned to normal. After completion of treatment the mean values of various parameters studied were found to be non-significant as compared to healthy control goats (group C) table 1 to 26. All the ten goats of this group were recovered completely.

The goats of severe acidotic group (group T_3) were given Anistamin 2 ml I/M morning and evening for 3 days followed by 2 ml I/M daily once for rest 4 days. Dexona @ 1ml on 1st and 2nd days, 0.75ml on next two days and

0.5ml on next 5th day by I/M rout to each surviving goats. Sodabicarb (7.5%) was given @ 50 ml I/v for 2 days followed by 5 gm Sodabicarb in 50 ml of water twice daily by intra-ruminal route for 2 days. Normal saline (0.9 %), 100 ml, I/V daily, for 3 consecutive days depending upon the degree of dehydration. After some improvement the goats were also given 2 ml Belamyl I/M on alternate day for 5 days. From 3rd day onward, Vit. B complex liquid (Ambiplex) was given @ 5 ml orally twice daily for one week. A combination of rumenotoric drugs containing Rumbion, yea-sac bolus and Anigest were given @ of 1 bolus, 1 bolus and 5 gm. respectively, twice daily, orally for a week given. Steclin bolus @ ½ bolus was given twice daily for 3 days orally. When improvement of pH of ruminal fluid was achieved, ruminal-cud from healthy goats/slaughtered goats was transplanted orally @ 150 – 200 ml per day for 3 to 4 days. During the course of treatment two goats out of five severely affected groups died on 3rd day and rest three recovered gradually. After completion of treatment the mean values of various parameters were found to be non-significant as compared to healthy control goats (group C) table 1 to 26.

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CHAPTER - V

DISCUSSION

DISCUSSION

Rumen dysfunction is the common problem, incountered in ruminants particularly in goats because of several factors including change in diet or greedy nature of goats. The disease is characterized by poor appetite, alteration in pH of rumen fluid, decreased ruminal motility and reduced protozoal count (Radostits *et al.*, 1994). As a matter of fact, ruminal acidosis is one of the major managemental disease of ruminants. Primarily this syndrome arises due to heavy engorgement with easily digestable and fermentable carbohydrate rich diet. Different carbohydrate can produce lactic acidosis in acute and sub acute forms. Among them wheat has been considered as one of the potent grains that causes acidosis (Elam, 1976 and Slyter, 1976). Acute ruminal acidosis causes severe losses in terms of death. The percent mortality may reaches up to 100% in untreated cases. But the ruminants suffering from mild or sub acute acidosis the production losses are much more. In field condition 18% of indigestion cases in India are due to lactic acidosis (Prasad *et al.*, 1976a). Following on outbreak the morbidity rate may be 10-50% while the mortality rate may reach up to 90 % in untreated and 30 – 40% in treated cases (Blood *et al.*, 1983). So in the present study keeping in view a lot of benefits to the people of the society and importance of this poor creature particularly for the people living below the poverty line attention, were concentrated to protect this species from the most common and dreadful condition related to rumen dysfunction especially ruminal acidosis which directly affect the appetite and eventually the production of milk, meat and other by products. The high mortality rate and rare available information on clinical ruminal acidosis initiated to under take this programme.

5.1 OCCURANCE OF ACIDOSIS (Naturally/Experimentally) :

In the present study, on the basis of pH of ruminal fluid and severity of symptoms, all clinically affected goats were categorised into mild acidotic goats, moderate acidotic goats and severe acidotic goats.

Similarly, occurrence of clinical cases of ruminal acidosis in goats were earlier reported by other workers Gnanaprakasam (1970), Prasad and Rekib (1975), Prasad *et al.* (1976a), Bhatia *et al.* (1980), Srivastava *et al.* (1985), Sobhanan and Aleyas (1986). A moderate ruminal acidosis (pH 5.5) in a buffalo (Kumar and Joshi, 1994) and cross bred heifer (Basak, 1993) has been reported. Bhikane *et al.* (1996) reported a case of severe ruminal lactic acidosis in a buffalo. Likewise, occurrence of experimentally induced acidosis were also reported by several workers from broad and abroad. (Aslan *et al.* (1995), Patra *et al.* (1996), Nour *et al.* (1998) and Nikolov (2000).

5.2 CLINICAL FINDINGS :

In the present study, mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) showed several clinical signs and symptoms. Similar findings has been reported by Gnanaprakasam (1970); Sen *et al.* (1982) and Tanwar and Mathur (1983b).

In the present investigations, loss of appetite was observed in mild acidotic goats (group T_1). It might be due to lactic acedemia as a result of derangement in the normal rumen metabolism. However, Phillipson (1955) suggested that it may be due to decreased microflora. Hungate (1966) reported that loss of appetite might be linked with microbial fermentation activity. Loss or suspended rumination was observed in all three forms of acidotic goats. This findings is an agreement to the observation of Sen

(1982), Tanwar and Mathur (1983b) and Das (1990), in goats. Lack of rumination might be due to failure of muscular contractions of rumen and reticulum due to accumulation of lactic acid. Anorexia was observed in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). Similar observation has been recorded by different workers in goats by Gnanaprakasam (1970), Sen *et al.* (1982), Tanwar and Mathur (1983b) and in Sheep by Broberg (1960) and in ruminants by Mackenzie (1967). However, Shinosaki (1959) did not notice any reduction in appetite in acidosis in sheep. In rumen acidosis, anorexia occurs due to excessive production and absorption of toxic factors like lactic acid, histamin and amines as reported by Huber (1976) or due to complete or partial absence of bile salts coupled with distened liver in acute heptitis (Blood *et al.,* 1983). In the present study, firm and doughy rumen consistency was found in mild acidotic goats (group T_1) whereas, watery like rumen consistency was observed in both moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). These finding were in accordance with those described by Gnanaprakasam (1970), Sen (1982) and Das (1990) in goats. Firm and doughy rumen might be due to accumulation of excessive solid mass of crushed rice and wheat etc. impacted in the rumen. The excessive accumulation of fluid in the rumen or watery consistency of rumen might be due to increased osmolality of ruminal fluid which drew out fluid from the blood vascular system to the rumen. (Chakrabarti, 1988). Distended abdomen was an important clinical sysmptoms found in both moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). This finding is similar to the finding of Joshi (1969), Gnanaprakasam (1970) and Das (1990). This may be due to excessive feeding of grains and accumulation of

fluid after its with drawal from circulation (Blood *et al.*, 1983). Abdominal pain was observed in moderate acidotic goats (group T_2). Similar finding was recorded by Ahrens (1967) and Lal *et al.* (1989). The abdominal pain might have developed due to damage of ruminal epithelium on account of corrosive action of lactic acetic acid (Dunlop and Hammond, 1965 and Ahrens, 1967). Excessive accumulation of fluid in rumen from general circulation along with distension creates pressure on the nerves endings which may be the cause of abdominal pain (Blood and Radostits (1989). Nervous symptoms like, dull appearance and severe depression were observed in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) respectively. Similar menifestations have been reported in goats by Tanwar and Mathur (1983b), Lal *et al.* (1989), in sheep by Vestweber *et al.* (1974) and in cattle by Mackenzie (1967). It might be due to the change in biophysical property of the rumen liquor and toxic effect of accumulated lactic acid on the CNS (Dunlop and Hammond, 1965). It may be due to toxic effect of lactic acid and alcohol poisoning on brain Juhasz and Szegedi (1968d) or due to polioencephalomalacia owing to thiamine (Vitamin B_1) antagonism (Brent, 1976). Staggering gait like symptom was found in some severe acidotic goats (group T_3). Similar observation has been recorded in goat by Gnanaprakasam (1970), Tanwar and Mathur (1983b), in sheep by Nisbet *et al.* (1963) and in cattle by Mackenzie (1967). It may be due to CNS disturbance on account of histamine toxicity and lactic acidaemia (Dunlop and Hammond, 1965). In the present study moderate constipation was observed in moderate acidotic goats (group T_2) while severe constipation was found in severe acidotic goats (group T_3). Likewise, finding has been reported by Gnanaprakasam (1970) and Tanwar and Mathur (1983b) in

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ruminal acidosis in goats. This may be due to hypertonicity of intestine following ruminal stasis under the influence of low ruminal pH. Pasty to diarrhoic stool was also observed in some severe acidotic goats (group T_3). This finding is similar to the finding of Gnanaprakasam (1970), Tanwar and Mathur (1983b), Lal *et al.* (1989) and Das (1990) in goats and in sheep by Nisbet *et al.* (1963). Diarrhoea may be due to hypertonicity and depletion of bicarbonate ions in the intestine which are the sole factors for causing acidosis. Reduced net absorption of water may be the cause of diarrhoea (Lee, 1977). It might be due to hyperosmolality of intestinal content or due to inflammatory and ulcerative changes of wall of the intestine due to lactic acid or due to secondary bacterial infection of the intestinal wall (Castello, 1968) Nasal discharges was found in moderate acidotic goats (group T_2). Similar symptom was noted by Tanwar and Mathur (1983b) in goats and Dunlop and Hammond (1965) in Holstein heifers following acidosis. Nasal discharge might be due to hyperacidity causing nasal irritation. Grinding of teeth was an imporant symptom noticed in moderate acidotic goats (group T_2). Similar observation was recorded by other workers in goats Gnanaprakasam (1970), Tanwar and Mathur (1983a) and Nour *et al.* (1998) and in sheep by Scarisbrick (1954). This may be due to the toxic effect, causing irritation on the CNS. In the present study in mild acidotic goats (group T_1), dull sound was observed on percussion of left flank whereas, continous gurgling sound was noted in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). This observation is in agreement to the observation of Gnanaprakasam (1970). It might be due to watery consistency of ruminal content as a result of withdrawal of fluid from the vascular system to the rumen. In severe acidotic goats (group T_3) out of five goats,

two were showing symptoms like regurgitation of feed materials from the nostril & mouth which is similar to the observation noted by Tanwar and Mathur (1983a) in goats with wheat induced acidosis. This may be due to engorged rumen under the influence of low rumen pH. In the present study lamness and staggering gait was noticed in some severe acidotic goats (group T_3). Similar observations were recorded in acidotic goat by Gnanaprakasam (1970), Tanwar and Mathur (1983b) and in sheep by Scarisbrick (1954) and in other ruminants by Dunlop and Hammond (1965). This may be a clinical manifestation possibly of laminitis in severe acidotic goats (group T_3) on account of excessive production and absorption of lactic acid. It may also be due to increased histamine toxicity in experimental animals, Ahrens (1967) and Dunlop (1972). Staggering gait may be due to CNS disturbance on account of histamin toxicity and lactic acidaemia, Dunlop and Hammond (1965). Frequent bleating/crying was observed in moderate acidotic goats (group T_2). As a matter of fact crying was expression of pain, the source of which might be abodominal pain. Similar clinical signs was found in goat by Nour *et al.* (1998). In severe acidotic goats (group T_3) some goats were showing lateral recumbency. This finding gets support from the observations recorded by Scarisbrick (1954) and Nisbet *et al.* (1963) who have too reported similar lateral recumbency in sheep. Blood *et al.* (1983) had opined that recumbency usually followed after 48 hours in large animals but if might be present as an early sign in ruminal acidosis. This might be due to dehydration, muscular weakness, hypocalcaemia and the possible toxic effect on the CNS. In the present study symptoms like anuria was found in severe acidotic goats (group T_3). The present observation is in agreement with the findings recorded in goat by

Gnanaprakasam (1970); Sen *et al.* (1982) and Tanwar and Mathur (1983b) and in sheep by Broberg (1960). Anuria may be a clinical finding in acute cases of ruminal acidosis Blood *et al.* (1983). It might be due to decreased renal blood flow and glomorular filteration rate in acidosis as a result of renal damage. In severe acidotic goats (group T_3) the sunken eyes was found. This finding colloberates with the observation noted by Tanwar and Mathur (1983b). This indicated that the animals had suffered from dehydration, which was due to increased osmotic pressure of rumen fluid resulting into passage of water from blood into the rumen by Parthasarthy and Phillipson (1953). In the present study two out of five goats, severe acidotic goats (group T_3) could not stand with the severity of the disease and died during investigation period. Nisbet *et al.* (1963) reported flock mortality to be 5-20 per cent. Sandha and Choudhuri (1985) reported 66 per cent mortality in buffalo calves between 24 to 48 hours while, Sinha *et al.* (1985), observed mortality to be 66 per cent at 48 hours of induced rumen acidosis. Blood *et al.* (1983) opined that death usually occur following engorgement within 24 to 72 hours of acidosis. They also stated that, following an out break the morbidity rate may be 10-50 per cent while the mortality rate may be 10-50 per cent while the mortality rate may reached up to 90 percent in untreated and 30-40 per cent in treated cases. Thus this finding is inclose agreement with the above workers.

After the post treatment in all recovered acidotic goats passed pelleted faeces, characteristic for the species. All goats grazed whole day, drunk water ad-libitum which showed good appetite. They were active and alert. The muzzles looked moist. Conjunctival mucous membranes were pale pink to rosy in colour. This finding was similar to that of Kelly (1974) and

Chakrabarti (1988). The goats ruminated several times in a day during resting period. Abdomens were normal in size and did not show abnormality on percussion. These findings were similar to that of Kelly (1974) and Rai and Pandey (1980b).

5.2.1 Temperature :

In the present study the average rectal temperature (^{0}F) was found to be 102.8 ± 0.19 in healthy control goats (group C). This finding was close to the values stated by Kelly (1974) and Chakrabarti (1988) in normal goats. The mean values of rectal temperature (^{0}F) in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 102.7 ± 0.14, 101.9 ± 0.10 and 101.3 ± 0.13 respectively. In the present study in moderate acidotic goats (groupT_2) and severe acidotic goats (group T_3) the average values of rectal temperature were found to be significant ($P<0.05$) but gradual fall except in mild acidotic goats (group T_1) where, non-significant fall in average rectal temperature was observed as compared to healthy control goats (group C). The present finding is similar to the observation reported by Lal *et al.* (1989). Subnormal temperature were recorded by Jensen and Mackey (1965) in ruminants, Tanwar and Mathur (1983a) and Nour *et al.* (1998) in acidotic goats. However, Gnanaprakasam (1970), Sen (1982) and Das (1990) reported elevated body temperature. Decrease in rectal temperature may be due to lactic acidosis leading to dehydration, fall in total plasma volume & severe depression of cardiovascular system (Dunlop and Hammond, 1965).

5.2.2 Pulse :

The average pulse per minute was found to be 73 ± 2.06 in healthy control goats (group C). This finding has got the similarity to the observations of Kelly (1974) and Chakrabarti (1988). In the present study the mean values of pulse per minute in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 123.5 ± 1.40, 133.9 ± 0.47 and 142 ± 1.51 respectively. These values were found to be significantly ($P<0.01$) increased as compared to healthy control goats (group C). Similar trend were also reported by Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983a), Cao *et al.* (1987), Lal *et al.* (1989), Das (1990) and Nour *et al.* (1998) in acidotic goats, Patra (1991) in sheep and Bhikane *et al.* (1996) in Buffalo. The increased pulse per minute might be due to toxic effect of accumulated lactic acid, fall in plasma volume and circulatory failure (Blood and Radostits, 1989). Significantly pulse rate have been shown to increase due to energy rich diet (Thomas and Moore 1951).

5.2.3 Respiration :

The average respiration per minute was found to be 21.66 ± 1.14 in healthy control goats (group C). This observation is similar to the findings of Kelly (1974) and Chakrabarti (1988). In the present study respiration per minute in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 24.58 ± 0.55, 31.6 ± 0.142 and 36.8 ± 1.15 respectively. These values were found to be significantly ($P<0.01$) increased as compared to healthy control goats (group C). Similar findings were earlier reported by Gnanaprakasam (1970), Tanwar and Mathur (1983a), Sen *et al.* (1984), Cao

et al. (1987), Lal *et al.* (1989) Das (1990) and Nour *et al.* (1998) in acidotic goats. In the present investigation increased respiration per minute 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). In the present study goats consumed accidently a lot of energy rich diet in toxic doses which might have caused to increase respiration per minute.

5.2.4 Dehydration (Skin fold test in second) :

No retention of skin folds were observed in healthy control goats (group C) and pre-treated groups viz. mild acidotic goats (group T_1) and moderate acidotic goats (group T_2). Retension of skin folds for 20 – 30 second were seen in severe acidotic goats (group T_3) which indicated 10% dehydration. Similar observation has been reported by Gnanaprakasam (1970) and Tanwar and Mathur (1983a). The retention of skin fold might be due to loss of skin elastisity occurred due to less perfussion of water in the peripheral region. The less perfussion of water in the peripheral region might be due to heavy drainage of fluid from blood into the rumen content.

5.2.5 Rumen motility :

In the persent study, the average rumen motility/ 5 minutes in healthy control goats (group C) was found to be 7.66 ± 0.49. Similar finding was reported by Kelly (1974) and Rai and Pandey (1980b). In pre-treated group viz. mild acidotic goats (group T_1) the average rumen motility was observed to be 2.91 ± 0.25 whereas, the average rumen motility was found to be absent in both moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). The mean value of rumen motility/5 minutes was found to be decreased significantly ($P<0.01$) in mild acidotic goats (group T_1), when,

compared to healthy control goats (group C). This is in agreement with the observation made by other workers in goat (Gnanaprakasam 1970; Sen *et al.* 1982; Tanwar and Mathur 1983b and Lal *et al.* 1989) and in sheep (Scarisbrick, 1954 and Broberg, 1960). Gaikwad *et al.* (1993), stated markedly decreased rumen motility in clinical cases of indigestion in buffaloes. Dwivedi *et al.* (2000) reported decreased ruminal motility in calves following ruminal dysfunction. The decreased rumen motility might be due to elevated hydrogen ion concentration in the rumen ingesta inhibited metabolic pathways, destroyed rumen epithelium and helped in absorption of organic acids leading to ruminal atony (Ahrens, 1967). Various factors such as rumen pH, lactic acid concentration, volatile fatty acids concentration, histamine, endotoxin etc. have been suggested to influence the ruminal motility (Huber, 1976). They also suggested involvement of hydrogen ion receptors in gastroentestinal tract, central inhibition of absorbed acids, an inhibition by absorbed amines and thus possibly leading to reduced ruminal motility. Stern *et al.* (1970) found that the increased plasma immuno reactive insulin was the reason for decreased rumen motility. Svendsen (1973) observed that the butyric acid was the most potent inhibitor of ruminal motility. Many workers have reported that, as the pH of rumen ingesta approaches 5.0, the amplitude and frequency of rumen contractions progressively diminish with evantual stasis (Kezar and Church, 1979b).

5.2.6 Rumen Consistency :

In the present study viscous to semi-liquid rumen consistency was found in healthy control goats (group C). Similar finding were recorded by Sen (1982), Pradhan *et al.* (1988) and Lal *et al.* (1989). The variation of rumen consistency may be due to difference in feed comsumption

(Rosenberger, 1979). Thick, firm and doughy like rumen consistency was observed in pre-treated groups viz. mild acidotic goats (group T_1) but watery rumen consistency were found in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). Similar observation were earlier reported by Sen (1982), Lal *et al.* (1989) and Das (1990) in goats. Thick, firm and doughy like rumen consistency might be due to increased viscosity with heavy concentration of undigested feed grains particles. The watery rumen consistency might be due to passage of fluid from vascular bed to rumen as a result of increased osmolality of the rumen content.

5.3 EXAMINATION OF RUMINAL FLUID :

5.3.1 pH of ruminal fluid :

In the present study the mean value of ruminal fluid in healthy control goats (group C), was found to be 6.96 ± 0.06. It was in close agreement with the earlier reports of Sen (1982), Vihan *et al.* (1982), Tanwar and Mathur (1983c), Vihan and Rai (1985), Cao *et al.* (1987), Lal *et al.* (1989), Das (1990) in goats. The normal pH of the ruminal fluid was thus on the acidic side or neutrality. The slight variation in the pH values was due to variation of methods of collection and assaying of the ruminal fluid, the nature, quality and quantity of feed and time of collection of samples (Blake *et al.,* 1957). Rai and Pandey (1980a) stated that quality of feed used, influence the pH of rumen liquor. The average values of ruminal pH in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were recorded to be 5.91 ± 0.10, 4.99 ± 0.10 and 4.16 ± 0.09 respectively. The significantly ($P<0.01$) decreased pH of ruminal fluid were found in above pre-treated groups. Similar findings were also noted by Gnanaprakasam (1970), Sen (1982), Vihan *et al.* (1982), Cao

et al. (1987), Lal *et al.* (1989) Das and Misra (1991), Basak *et al.* (1993), and Nour *et al.* (1998) in goats, Patra *et al.* (1996) in sheep and Dwivedi *et al.* (2000) in cattle. Tanwar and Mathur (1983c) reported ruminal pH of 4.96, 4.85 and 4.29 at 12 hrs. in mild, moderate and severe cases of acidosis in goats respectively, following whole wheat engorgement. A marked decreased in the pH of ruminal fluid might be due to complete and faster fermentation of crushed grains (rice/wheat) by amylolytic bacteria, leading to production of lactic acid (Ahrens 1967; Tremere *et al.,* 1968), reduction in concentration of rumen ammonia nitrogen and also high concentration of TVFA (Phillipson, 1942). As the pH fell below 6, the salt of volatile fatty acids were converted to the free acid forms, and provided some buffering. The marked and prolonged depression of rumen pH seen in later stages must have been due to increased lactic acid concentration (Dunlop, 1972). The buffering capacity of the rumen contents below 5.5 pH has been shown to be mainly due to volatile fatty acids (Mackenzie, 1967). The increased lactic acid, converted carbonate and bicarbonate into carbonic acid and it further dissociated to water and carbon-dioxide. Thus (H lactic + Na^+ HCO^-_3 ..... Na^+ lactate $^-$ + H_2CO_3) absorption of lactic acid reduced the body's reserves of bicarbonate leading to acidosis (Dunlop, 1972).

5.3.2 Motility of rumen microflora (Protozoa) :

In the present study, vigorous protozoal motility (+++) was observed in healthy control goats (group C). Similar findings were also noted by Sen (1982), Pradhan *et al.* (1988) and Das (1990) in goats and Misra *et al.* (1972) and Misra and Singh (1974) in cattle. Slight variation might be due to individual variation, species variation, difference in nature and quality of feed intake and time of sampling (Purser and Moir, 1966; Rosenberger,

1979). In pre-treated groups viz mild acidotic goats (group T_1), the motility of rumen protozoa was found to be sluggish (+) whereas, complete absence of protozoal motility (-) was observed in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). This finding is near to the findings of other workers in goats (Gnanaprakasam 1970; Sen *et al.* 1982; Tanwar and Mathur 1983c), sheep (Hungate *et al.*, 1952; Phillipson 1952) and cattle (Dash *et al.*, 1972; Nauriyal and Baxi 1978). Li *et al.* (1984) reported that loss of protozoal motility in experimentally induced sheep. Das (1990) found that concentration of motility of rumen protozoa absent in rumen acidosis. Basak *et al.* (1993) stated that there was obsence of rumen protozoal motility in experimentally induced lactic acidosis in goats. Upadhayay and Shukula (1999) mentioned sluggish (+ or ++) protozoal motility in various ruminal disorder. Motility of the ruminal protozoa depended on the H^+ ion concentration and osmolality of the rumen liquor which provided an optimal environment. The rumen protozoa are very sensitive to abrupt change in rumen eccosystem especially to acidity. Absence of rumen motility in the present study might be due to very high concentration of lactic acid and low pH in the rumen (Hungate *et al.*, 1952; Dunlop, 1972). It was also suggested that lysis of rumen protozoa, occurred when the rumen pH fell below 5.5 (Hungate, 1966; Slyter, 1976). Marked reduction in the motility of the rumen protozoa could be due to low intracellular and environmental pH (Prins and Van Hoven, 1977) and high tonicity of the rumen encountered in lactic acidotic animals (Ahrens, 1967). Hungate *et al.* (1952) and Krogh (1959) found that factors like low rumen pH and high rumen toxicity killed protozoa.

5.3.3 Activity of rumen microfora :

5.3.3.1 Cellulose digestion time (CDT) :

The average value of CDT (hrs.) in healthy control goats (group C) was found to be 44.33 ± 1.05. This is in agreement with the observation made by other workers in goats (Das 1990; Basak *et al.*, 1993) and in sheep, (Hoflund *et al.*, 1948). The variations might be due to individual, species variation and types of feed intake. The mean value of CDT (hrs.) in pre-treated groups viz. mild acidotic goats (group T_1) was found to be 76.33 ± 1.38. This value was observed to be significantly ($P<0.01$) high as compared to healthy control goats (group C). There was no observed cellulose digestion time (hrs.) in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). Basak *et al.* (1993) found significantly ($P<0.01$) high (47.22 ± 3.64 hrs.) CDT value, whereas Gnanaprakasam (1970), Das (1990) recorded complete absence of cellulose digestion time from 24 hrs onward in acidotic goats. Blood *et al.* (1983) stated that CDT in excess of 30 hours, indicate abnormality. In the present study the time factor for CDT increased significantly ($P<0.01$) in mild acidotic goats (group T_1) and were absent in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). It might be due to destruction of normal microflora (Cellulolytic bacteria) and shift in their pattern from gram-negative to gram positive nature (Amylolytic bacteria) Randhawa *et al.* (1989). This indicated gross inactivity of the rumen microbes resulting in to disturbance of normal digestion process.

5.3.3.2 Sedimentation activity time (SAT) :

In the present study the mean value of SAT (minutes) was found to be 24.83 ± 1.55 in healthy control goats (group C). This finding is similar to the findings of Sen (1982), Pradhan *et al.* (1988), Das (1990) and Basak *et al.* (1993) in goats but did not coinside with the findings of Nicholas and Penn (1958) and Blood *et al.* (1983) in cattle which showed lower SAT (minute) value. This variation was due to individual and species variation (Nicholas and Penn, 1958). The average value of SAT (minute) was observed to be 67.08 ± 0.75 in pre-treated group viz. mild acidotic goats (group T_1). This value was found to be significantly ($P<0.01$) increased when compared to healthy control goats (group C). There was no observed sedimentation activity time (minutes) in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). This observation were very similar to that of Gnanaprakasam (1970), Sen (1982), Das (1990) and Basak *et al.* (1993) in goat and Rosenberger (1979) in cattle. The average SAT value significantly ($P<0.01$) increased in mild acidotic goats (group T_1) but absent in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) might be due to destruction of normal microflora and shift in their pattern from gram-negative to gram positive nature (Randhawa *et al.*, 1989).

5.4 EXAMINATION OF BLOOD/SERUM :

5.4.1 Blood lactic acid :

The mean lactic acid concentration (mg%) in blood of the healthy control group of goats (group C) was 12.91 ± 1.02. This finding has got the similarly to the observation of Verma *et al.* (1975), Sen (1982), Tanwar *et al.* (1983), Cao *et al.* (1987) and Das (1990). Slight difference in level of lactic acid in normal animal might be due to different times of sampling and

concentrate feeding (Verma *et al.*, 1975). In the present study the average values of blood lactic acid (mg %) in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 18.16 ± 1.03, 25.99 ± 1.11 and 41.44 ± 2.85 respectively. These values were found to be significantly ($P<0.05$) higher as compared to healthy control goats (group C). Similar observation was previously reported by Sen (1982), Vihan *et al.* (1982), Tanwar *et al.* (1983), Vihan and Rai (1985), Cao *et al.* (1987), Lal *et al.* (1989), Das (1990), Das and Misra (1991), Lal *et al.* (1992), Basak *et al.* (1993), Sen *et al.* (1993) and Nour *et al.* (1998) in goats. Patra (1991) recorded increased lactic acid concentration in both in rumen fluid and blood in acidotic sheep. Angelov *et al.* (1996) reported increased level of blood lactate in acidotic sheep. Patra *et al.* (1996) noted increased lactic acid concentration in blood of experimentally induced acidotic ewes. Dwivedi *et al.* (2000) carried out an experimental rumen dysfunction and recorded significant ($P<0.05$) increased blood lactic acid concentration. Blood lactic acid concentration was increased due to excessive fermentation of carbohydrates by Streptococcus and Lactobacillus produced by the shift of microbial population from gram negative to gram positive flora. The increased blood lactic acid concentration (mg%) was absorbed through rumen wall leading to lactacidaemia (Dunlop, 1972 and 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 is absorved more readly at low pH (William and Mackenzie, 1965). Decrease in perfusion pressure and oxygen supply to peripheral tissues due to dehydration resulted in a further increase in lactic acid from cellular

respiration (Blood *et al.*, 1983). At very lower pH the rumen become 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). Anuria was found in severe acidotic goats (group T_3) might prevent excretion of D-lactate and aggravate the systemic acidosis. As more lactic acid accumulates in the blood, the bicarbonate buffer system releases even more carbon-dioxide, in a final effort to compensate. The increased carbon-dioxide tension depresses the respiratory centre in the terminal stages and blood pressure decreases rapidly (Huber, 1976).

5.4.2 Blood glucose :

The average value of blood glucose level (mg%) in healthy control goats (group C) was found to be 52.59 ± 2.05. This observation in agreed with the observations of Melvin (1977), Sen (1982), Sastry (1983), Das (1990) and Sen *et al.* (1993) in goats. The concentration of blood glucose was maintained within a fairly optimal range and any slight deviation was influenced by several factors including hepatic and renal uptake and release of glucose, utilization of glucose by peripheral tissues, the intestinal absorption and the effect of hormonal influences on these processes (Coles, 1974). In the present study the mean values of blood glucose level (mg%) in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 56.82 ± 1.44, 85.52 ± 1.46 and 112.38 ± 4.22 respectively. These values were found to be significantly ($P<0.01$) increased as compared to healthy control goats (group C). Increased blood glucose level was previously reported by other workers Dirksen (1970), Ivanov (1974), Vestweber *et al.* (1974), Nauriyal (1975), Randhawa *et al.* (1980), Nagaraja *et al.* (1982), Sen (1982),

Vihan *et al.* (1982), Vihan and Rai (1985), Cao *et al.* (1987), Das (1990), Sen *et al.* (1993) and Angelov *et al.* (1995). However, Nauriyal and Baxi (1978) reported a significant fall in blood glucose level in molasses induced rumen acidosis in calves. Heuter *et al.* (1956) have indicated that the rise in blood glucose observed in ruminal acidosis was due to absorbed ruminal lactic acid rather than the propionate fraction of volatile fatty acids. Although appropriate pathways for gluconeogeneses from lactate have been demonstrated (Annison *et al.* 1963), it would be erroneous to attribute the hyperglycaemia to this factor alone (Mackenzie, 1967). Annison's data suggested that over 15% of the glucose pool was derived from lactate, after prior dehydrogenation to pyruvate by L-lactate acid dehydrogenese in the presence of nicotinamide adenine dinucleotide (NAD). The excess lactate (i.e. increased lactate: pyruvate ratio) generally associated with lactic acidosis led to believe that lack of sufficient NAD might be responsible for the block in pyruvate metabolism (Mackenzie, 1967). Under such condition, glucose can not be formed from lactate. The stress associated with lactic acidosis might lead to secretion of adreno-corticoid hormones, which in turn increase the blood glucose level by gluconeogenesis (Mackenzie, 1967). Transient hyperglycemia may be due to absence of immuno reactive insulin in circulation (Randhawa *et al.*, 1982). The rise in level of blood glucose might have been either due to the increase glycogenolysis or gluoneogenesis or due to the decreased utilization of glucose by peripheral tissues (Dirksen, 1970, Ivanov, 1974). The gluconeogenesis was due to high absorption of propionic acid and lactic acid, the precursors of blood glucose in ruminants. The increased glycogenolysis and gluconeogenesis might have been due to increase in serum glucocorticoids level in stress of digestive disorder.

5.4.3 Serum chloride :

The average value of serum chloride level (mEq/l) in healthy control goats (group C) was found to be 105.98 ± 1.76. This finding is close to the observation of Dutta (1992) in normal healthy sheep. In the present study the mean values of serum chloride level (mEq/l) were found to be 110.51 ± 1.79, 117.51 ± 1.34 and 126.99 ± 1.89 in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) respectively. The mean values of serum chloride level (mEq/l) was found to be increased non-significantly between healthy control goats (group C) and mild acidotic goats (group T_1). However, significant ($P<0.05$) increased mean values of serum chloride (mEq/l) found in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) as compared to healthy control goats (group C). This observation is similar to the observation of Cakala *et al.* (1975), Sinha *et al.* (1985) and Dunlop (1961) had reported that the serum chloride in cattle had tendency to rise as osmolality of plasma increased. Randhawa *et al.* (1980) have also found elevated CSF chloride in acidotic buffalo calves. Sen *et al.* (1993) have reported the serum chloride increased (113 ± 4.5 mEq/l) significantly by 4 hours of grain overload and the peak concentration (131.0 ± 4.2 mEq/l) reached by 20 hours. However, Shinosaki and Nakabayashi (1974), found lower blood chloride level in acidotic sheep. Patra *et al.* (1993) had reported decreased level of chloride in CSF and urine in acidotic sheep.

5.4.4 Serum Sodium :

In the present study the mean value of serum sodium (mEq/l) was found to be 140 ± 2.12 in healthy control goats (group C). Similar finding was noted by Sen (1982), Das (1990), in goat and Irwin *et al.* (1979) in

sheep. The average serum sodium level in pre-treated groups of goats viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 142 ± 1.18, 149 ± 1.72 and 165 ± 1.93 respectively. The mean values of serum sodium were found to be non-significantly increased between healthy control goats (group C) and mild acidotic goats (group T_1). However, a significant ($P<0.05$) increase level of serum sodium was observed in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) as compared to healthy control goats (group C). The increase in serum sodium concentration in rumen acidosis agreed with the findings of Sen (1982) and Das (1990) in goats and Shinosaki and Nakabayashi (1974) and Inwin *et al.* (1979) in sheep. The increase in serum sodium might be due to its retention of sodium by the kidneys.

5.4.5 Serum potassium :

The average value of serum potassium (mEq/l) was found to be 4.27 ± 0.22 in healthy control goats (group C). Similar findings were also earlier reported by Sen (1982) and Das (1990) in goat. In the present study the mean values of serum potassium (mEq/l) in pre-treated groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) were found to be 4.57 ± 0.09, 3.88 ± 0.07 and 3.29 ± 0.10 respectively. The mean values of serum potassium (mEq/l) were found to be non-significant increased in between healthy control goats (group C) and mild acidotic goats (group T_1). However, there was significant ($P<0.05$) decreased serum potassium level were found in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) as compared to healthy control goats (group C). The decrease in serum potassium level was similar

to that, reported by Dirksen (1965) in cattle, Huber (1971) & Cakala *et al.* (1975) in sheep, Das (1990) in goats. The decrease in serum potassium concentration might be due to its excretion through the kidney. The causes of hypokalemia may be due to retention of sodium ions from renal tubules in exchange of hydrogen ion actively secreted by renal tubules in acidotic animals (Kaneko, 1989). Hypokalemia noted in this study also supported by the finding of Choudhuri *et al.* (1980). However, Sethuraman and Rathor (1979b) observed hyperkalemia in bovine. Increase in serum potassium might be due to its increased reabsorption through renal tubules in respect of secretion of H^+ ion to maintain acid base balance.

5.5 THERAPEUTIC STUDY :

In the present study, therapy was based on the severity of clinical signs and symptom as well as alteration in biochemical (Ruminal fluid, blood and serum) findings. All the recovered goats of post-treated groups were found to be physiologically normal, having glossy skin coat, pink to rosy conjunctival mucous membrane. The muzzle appeared moist with dew-drop like appearance over it. They appeared active and alert. They showed good appetite, normal rumination, defecation and urination. These signs and symptoms were same as healthy control goats (group C) of the present study. It is also similar to the findings of Kelly (1974) and Chakrabarti (1988) in normal healthy animals. All the alterations of biochemicals (Ruminal fluid, blood and serum) occurring in rumen acidosis were found to be return back, following treatment. After completion of treatment, the mean values of various parameters were found to be 102.7 ± 0.13, 75.0 ± 1.48, 22.5 ± 0.75, 7.41 ± 0.35, 6.94 ± 0.05, 45.75 ± 0.65, 25.16 ± 0.72, 13.51 ± 0.79, 53.73 ± 1.17, 105.07 ± 0.97, 141 ± 1.38, 4.44 ± 0.12 in mild acidotic goats (group

T_1) whereas, 102.5 ± 0.14, 77.2 ± 1.8, 23.5 ± 0.67, 6.80 ± 0.32, 6.92 ± 0.06, 48.7 ± 1.06, 27.1 ± 1.04, 14.3 ± 1.00, 54.01 ± 1.14, 106.51 ± 1.13, 143 ± 1.77, 4.23 ± 0.13 and 102.4 ± 0.30, 78.3 ± 1.45, 24.0 ± 1.15, 6.66 ± 0.88, 6.9 ± 0.05, 49.66 ± 1.20, 29.33 ± 1.45, 14.43 ± 1.81, 54.58 ± 1.14, 107.33 ± 1.68, 145 ± 2.39, 4.10 ± 0.40 in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) respectively, in respect of temperature (^{0}F), pulse per minute, respiration per minute, ruminal motility per five minutes, pH of ruminal fluid, CDT in hours, SAT in minutes, blood lactic acid (mg%), blood glucose (mg%), serum chloride (mEq/l), serum sodium (mEq/l) and serum potassium (mEq/l). These average values of various parameters of post-treated groups were observed to be non-significant as compared to healthy control goats (group C). This findings is similar to the findings of Lal (1988), who reported recovery based on biochemical studies like that of lactic acid, BUN in experimental acidosis in goats. Nauriyal and Baxi (1981) also reported the recovery of total plasma protein and BUN following therapy in acidotic buffalo calves.

In all the three groups viz. mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3), the therapeutic measures consisting of neutrilization of acidity by oral or parentral or both of sodabicarb, the excesses of toxic amines & toxic gases which increase in acidosis were counteracted by parentral administration of antihistamin (Anistamin) & dexamethazone (Dexona), for the normalization of the ruminal pH, rumenotoric drugs particularly herbal preparation (Rumbion) and fresh ruminal cud from healthy slaughtered goats were given orally and fluid and electrolytes were restored on the basis of estimation of ionograms and also on the degree of dehydration.

In the present study, Anigest (Sarabhai Chemicals) was used which is a composition of Swertia Chirata, Nux Vomica, Zingiber Officinale etc. has got very good stomachic, appetiser and carminative properties (Day, 1980 and Kapoor, 1990). So Anigest helps to improve the activity of microflora, ruminal tonicity and thus facilitates digestion process. The present observation coincides with Upadhayay and Shukla (1999) who reported clinical cases to be suffering from various ruminal disorders and treated with Anigest @ 15 gm orally, two times a day for 3 days and found improvement of microbial activity, ruminal tonicity and good digestion in ruminants.

Rumbion Bolus (Indian Herbs), a herbal preparation was given orally in mild acidotic goats (group T_1), moderate acidotic goats (group T_2) and severe acidotic goats (group T_3) in the present study. As per the company literature and ingradients of the drug, it is recommendded as appetizer. Rumbion restored appetite and rumination quickly and improved cellulose digestion. It normalized the ruminal pH by promoting secretion of saliva and acted as a pH regulator. It optimises the tone and rumeno-reticular contraction by reflex stimulation of vagus nerve endings. Cobalt and Copper, in Rumbion, help in vitamin B synthesis, obviating the need for their extra supplimentation. Rumbin bolus ensures rapid growth and multiplication of rumen bacteria and protozoa for efficient digestion. It contains Chakrangi 810 mg, Katubhadra 675 mg, Mangalya 180 mg and Amogha 135 mg etc. These combinations help in increasing the appetite probably due to stimulation of the liver. Rumbion bolus had a stimulatory effect on the digestibility of nutrients. On supplementation of Rumbion bolus in vivo the tone and contraction of the rumen and reticulum improved and also the activity of rumen microflora and fauna improved (Misra, 1991). Dutta

(1992) used Rumbion bolus for treatment of sheep suffering with sub-acute lactic acidosis. Tripathy and Mishra (1992) reported beneficial effect of Rumbion bolus in experimentaly induced acid indigestion in goats.

In the present study Yea-sac bolus was used in severe acidotic goats (group T_3). Yeast tablets helps in stimulation of ruminal flora and for optimising biological and metabolic function of rumen. To reduce lactic acid concentration in blood and rumen fluid I/V administration of 3 million units of Penicillin and oral dosing with 0.5 Kg of Yeast with sugar beet was reported (Helijdasz and Nicponn, 1977). In early cases of ruminal acidosis in cow treatment with antihistamine, vitamin-B complex, Yeast, prednisolone, fluid and electrolytes supplementation and removal of rumen content by rumenotomy were suggested by Dirksen (1965). Braun *et al.* (1992) in their experiment on ruminal lactic acidosis in sheep and goat found 78.4% efficacy of antacid, Yeast, Chlortetracycline I/V and 5% Sodabicarb solution. Aslan *et al.* (1995) treated induced acute ruminal acidosis of goat with 1 gm Sodabicarb/kg body weight and 1 gm backing Yeast/kg or their combination @ 0.5 gm of each per kg and reported moderate improved effect of Sodabicarbonate where as no effect of Yeast preparation.

In the present study vitamin-B complex liquid (Ambiplex) was used in all groups. In severe acidotic goats (group T_3) was treated vitamin-B complex with liver extract (Belamyl) also which corrects the deficiency of thiamine hydrochloride and help in the absorption of lactic acid (Broberg, 1960). Vitamin – B complex preparation contains thiamine hydrochloride, riboflavin, niacin and cyanocobalamine. Thiamine hydrochloride deficiency occurs following grain engorgement but there is paucity of literature in the changes of riboflavin, niacin and cyanocobalamine. As a matter of fact,

riboflavin acts as an intermediate in biological oxidation reduction systems in mammals and this involves in all the areas of mammalian metabolism as co-factor, co-enzyme (FAD, riboflavin-5-phosphate) for generation of ATP (Kaneko, 1989). There is no such finding to confirm the use of riboflavin in acidosis. Niacin is also essential for the regulation of pyridin dinucleotide system in cell and virtually it is essential for mammalian metabolism (Kaneko, 1989). In ruminants rumen microbial population are responsible for B-vitamins productions. As the ruminal microflora are disturbed severely so the vitamin B-complex production may be hampered. In this way, vitamin B-complex may be helpful. Death due to polioencephalomalacia, as a result of deficiency of thiamin may occur. Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983b) obtained good result with thiamin in acidotic goats. Patra *et al.* (1997) also used vitamin for the treatment of acidotic sheep. Bhikane *et al.* (1996) used Neuroxin 10 ml. I/M for the treatment of ruminal lactic acidosis in buffalo. Schukken *et al.* (1985) suggested the use of vitamin preparation. They claimed the recovery of acidosis in cows caused by untreated consumed of Wastebakery, with vitamin preparation along with Soda bicarb etc.

Soda bicarb was given in mild, moderate and severe acidotic goats (group T_1, T_2 & T_3) orally whereas, in moderate and severe acidotic goats (group T_2 & T_3) it was given in both orally and I/V form. Soda bicarb @ 5 gm. once daily for 2-3 days in 50 ml. fresh water orally given in all three groups. It was also given in injection form in moderate acidotic goats (group T_2) and severe acidotic goats (group T_3). Intravenous injection of Sodium bicarbonate (7.5% W/V) might have helped to combat the loss of alkali reserve of the body while the oral administration neutrilized the acidity of the rumen (Prasad and Rekib, 1975). Gnanaprakasam (1970), did not

observe any adverse effect on the oral administration of sodium bicarbonate in goats suffering from rumen hyperacidity. In acute cases, antacids administration directly into the rumen may be contraindicated because of the risk of alkalosis which may occur during the recovery phases (Mullen, 1976). The initial metabolic alkalosis in the experimental goats indicates that the early administration of bicarbonate might be contraindicated (Cao *et al.*, 1987). The use of antacid in rumen acidosis by oral or parentral or both route has also been recommended by Sen (1982), Tanwar and Mathur (1983b), Braun *et al.* (1992) and Aslan *et al.* (1995) in goats, Patra *et al.* (1997) in sheep and Bhikane *et al.* (1996) in buffalo. Interavenous infusion of sodium bicarbonate (5%) has been recommended as a specific treatment in systemic acidosis (Blood and Radostits, 1989).

Ruminal cud were given in severe acidotic goats (group T_3) when pH of ruminal fluid improved. Many workers viz. Hoflund, 1967; Dunlop, 1972; Sethuraman and Rathor, 1979a) in cattle, (Scarisbrick, 1954; Patra *et al.*, 1997) in sheep, (Gnanaprakasam, 1970; Sen *et al.*, 1982; Tanwar and Mathur, 1983b; Lal, 1988) in goat, (Hungate, 1966; Dunlop, 1972; Prasad and Rekib, 1975) in buffalo have advocated the use of rumen cud in the restoration of rumen environment and fermentative processes. The present findings is thus in close agreement with the findings recorded by above workers. In many rumen dysfunctions the abnormal changes in the rumen microbes occur primarily in addition to the other complecations. The establishment of normal rumen microbial population is essential and can be best achieved by the cud transplantation (Hoflund 1967 and Dunlop 1972).

In the present study Steclin bolus (Sarabhai Chemicals) which contains Tetracycline hydrochloride was administered orally in severe acidotic goats (group T_3) to check the growth of gram positive bacteria, to stop further production of lactic acid in rumen and for sterilization of rumen

contents. Dirksen (1970) advocated oral antibiotic for treatment of ruminal acidosis. Nauriyal (1975) treated ruminal acidosis in cross bred cow calves and buffalo calves by broad spectrum antibiotic. Similar line of treatment was adopted by Sen (1982) and Tanwar and Mathur (1983b) in goats. Schukken *et al.* (1985) claimed the recovery of acidosis in cow by Oxytetracyclin injection along with Sodium bicarbonate and vitamin preparation. Braun *et al.* (1992) used to know the efficacy of Chlortetracycline antibiotic along with Yeast, I/V infusion of isotonic saline and 5% Sodabicarb in lactic acidosis in sheep and goats. Bhikane *et al.* (1996) reported severe forms of ruminal lactic acidosis in a buffalo and used Oxysteclin (Sarabhai Chemical, Baroda) antibiotic for treatment along with other drugs.

To counteract the effect of histamine and other toxic amine produced during rumen acidosis antihistamine like Anistamin (Intas pharmaceutical Ltd.) was given intramuscular. Antihistaminics were also recommended by Irwin *et al.* (1979). Likewise administration of antihistaminics previous noted by Gnanaprakasam (1970), Sen (1982), Das and Misra (1991) in goats. Patra *et al.* (1997) in sheep and Bhikane *et al.* (1996) in buffalo suffering from ruminal acidosis. 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 endotoxins 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). Increase in blood histamine levels could be due to the action of lactic acid and endotoxins which on absorption into circulation are considered to be responsible for release of histamine from body tissues particularly from laminitis tissue (Brent, 1976).

Dexamethazone (Dexona) as a corticosteroid was used in the present study. As per literature dexamethazone is ideal for emergencies. Bhikane *et al.* (1996) also used corticosteroid to prevent shock on ruminal lactic acidosis in a buffalo. Dirksen (1965) used prednisolone in early cases of ruminal acidosis in cow for treatment.

To check the degree of dehydration and restoration of electrolyte volumes to some extant Normal saline infusion was given intravenously in severe acidotic goats (group T_3). Similar line of treatment was adopted by Tanwar and Mathur (1983b), Patel (1990) in goats and Sathuraman (1976) and Bhikane *et al.* (1996) in buffaloes.

In spite of all treatment two goats out of five severely acidotic goats (group T_3) died on 3rd day during treatment. It might be due to the fact that the severely affected goats did not respond to systemic alkalizers and died, indicating the already set up toxaemia as a result of chemical rumenitis as reported by Blood and Radostits (1989). Kumar and Joshi (1994) were unable to cure a case of whole wheat grain engorgment with conservative treatment. Bhikane *et al.* (1996) failed to cure a five years old buffalo, with the history of having eaten, approximately 20 kg of whole sorghum grains 30-32 hours earlier suffering from lactic acidosis. However, Basak (1993) successfully treated a case of moderate ruminal lactic acidosis in a crossbred heifer with 7.5% Sodium bicarbonate, Dextrose saline and rumenotorics.

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CHAPTER - VI

SUMMARY & CONCLUSION

SUMMARY AND CONCLUSION

The present study was conducted in the Deptt. of Vety. Medicine, Bihar Veterinary College, Patna, under R.A.U, Pusa, Samastipur, Bihar. Six apparently healthy goats of either sex above one year of age were procured from the Animal supplier, B.V. College, Patna to study their normal physiological status. All the aling goats, brought to the B.V. College, Hospital with the complain of inappetance or anorexia, suspended rumination, without febrile reaction were selected for the present investigation. Altogether 27 clinically affected acidotic goats were choosen for study. Depending upon the clinical signs and on the examination of pH of ruminal fluid, all the 27 goats were categorised as mild, moderate and severe acidotic groups. The healthy control goats considered as group C consisted of 6 goats whereas, pre-treated goats (clinically affected) were divided into mild, moderate and severe acidotic goats viz. group T_1, T_2 and T_3 consisting of 12, 10 and 5 goats respectively. The pre-treated groups of goats viz. T_1, T_2 and T_3 were further designated as T_1, T_2 and T_3 respectively under post-treated groups. Clinical findings (temp, pulse, respiration etc) were recorded as described by Rosenberger *et al.* (1979). Test materials like ruminal fluid, blood/serum were collected from healthy control goats (group C), pre-treated groups (clinically affected) and post-treated groups for the present study. The pH of ruminal fluid was measured by systronics Digital pH meter, type 335 (systronics Ahmedabad India). Motility of rumen microflora (protozoa) was studied as per the method described by Misra *et al.* (1972), to know the activity of rumen microflora, two simple laboratory tests viz. CDT and SAT, were done as per the method described by Nicholas and Penn (1958). CDT and SAT were expressed in hours and minutes respectively. Lactic acid

concentration in blood was determined as per the method of Barker and Summerson (1941) and values were expressed in mg %. Blood glucose level was estimated by Folin-Wu-method as described by Frankel *et al.* (1970) and values were expressed in mg %. Serum chloride level (mEq/l) was determined as per the method of Schales and Schales (1941) whereas, serum sodium and potassium concentration were estimated by Flame-photometer as per the method described by Oser (1965) and values were expressed in mEq/l. A few number of coloured photograph were collected with an exhaustive use of colour films and camera. Only good, clear and important phtographs were selected.

In the present study, mild acidotic goats (group T_1) showed ovious signs were partial loss of appetite, partially suspended rumination, mild constipation and the mean pH of ruminal fluid was 5.91 ± 0.10. In moderate acidotic goats (group T_2), the signs observed were complete absence of rumination, complete loss of appetite, dull appearance, moderate constipation, increased pulse and respiration, decreased temperature. Ruminal motility was absent. The mean pH of ruminal fluid was 4.99 ± 0.10. Severe acidotic goats (group T_3) were characterised by severe depression with pathetic appearance, completely off-fed, no rumination and absence of ruminal motility microbial motility and activity. Some goats exhibited severe constipation and in other pasty to diarrhoic stools, ejected conjunctiva, dehydration and lateral recumbency etc. were observed. Dysponoeic but accelerated respiration and increased pulse rate were observed. The mean pH of ruminal fluid was 4.16 ± 0.09.

The mean temperature (^{0}F) in healthy control goats (group C) was found to be 102.8 ± 0.19. The rectal temperature (^{0}F) declined significantly

($P<0.05$) in pre-treated groups viz. moderate and severe acidotic goats (group T_2, T_3). After treatment it returned to normalcy in post-treated groups.

The average value of pulse per minute in healthy control goats (group C) was 73 ± 2.06 whereas, in pre-treated groups viz. mild, moderate and severe acidotic goats (group T_1, T_2 & T_3) were found to be 123.5 ± 1.40, 133.9 ± 0.47 and 142 ±1.51 respectively. All these values were observed significantly ($P<0.01$) increased and after treatment it returned to normalcy.

The mean value of respiration in per minute in healthy control goats (group C) was observed to be 21.66 ± 1.14. In per-treated groups viz. mild, moderate and severe acidotic goats (group T_1, T_2 & T_3), the mean values were 24.58 ± 0.55, 31.6 ± 0.142 and 36.8 ± 1.15 respectively and found to be significant ($P<0.01$) increased. After completion of treatment these values returned to normalcy.

The average value of ruminal motility/5 minutes was found to be 7.66 ± 0.49 in healthy control goats (group C). Complete absence of ruminal motility/5 minutes were observed pre-treated groups viz. moderate and severe acidotic goats (group T_2 & T_3) but significantly ($P<0.01$) increased values was found in mild acidotic goats (group T_1). After treatment it returned to normalcy.

Viscous to semi-liquid rumen consistency was found in healthy control goats (group C) whereas thick, firm and doughy consistency was observed in pre-treated groups viz. mild acidotic goats (group T_1) but watery rumen consistency was found in moderate and severe acidotic goats (group T_2 & T_3). Following treatment it returned to normalcy.

The average pH of ruminal fluid in healthy control goats (group C) was 6.96 ± 0.06 whereas, in pre-treated groups viz. mild, moderate and

severe acidotic goats (group T_1, T_2 & T_3), the values were found to be 5.91 ± 0.10, 4.99 ± 0.10 and 4.16 ± 0.09 respectively. These values were significantly (P<0.01) decreased in the present study and after treatment it came to normalcy.

Vigorous motility (+++) of rumen protozoa was observed in healthy control goats (group C) whereas, in pre-treated groups viz. mild acidotic goats (group T_1) the motility was sluggish (+) but complete absence of protozoal motility was found in moderate and severe acidotic goats (group T_2 & T_3). The values returned to normalcy after treatment.

The mean values of CDT (hrs.) and SAT (minutes) were found to be 44.33 ± 1.05 and 24.83 ± 1.55 respectively in healthy control goats (group C) whereas, in pre-treated groups viz. mild acidotic goats (group T_1) the CDT (hrs.) and SAT (minutes) values were observed to be 76.33 ± 1.38 and 67.08 ± 0.75 respectively. These values were significantly (P<0.01) higher than healthy control goats (group C). The cellulose digestion time (hrs.) and sedimentation activity time (minutes) were not observed in moderate and severe acidotic goats (group T_2 & T_3). After the completion of treatment the values returned to normalcy.

The mean values of blood lactic acid (mg %) and blood glucose (mg %) in healthy control goats (group C) were found to be 12.91 ± 1.02 and 52.59 ± 2.05 respectively. In pre-treated groups viz. mild acidotic goats, moderate acidotic goats and severe acidotic goats the values were (18.16 ± 1.03 & 56.82 ± 1.44), (25.99 ± 1.11 & 85.52 ± 1.46) and (41.44 ± 2.85 & 112.38 ± 4.22) respectively (group T_1, T_2 & T_3). Blood lactic acid and blood glucose were found to be increased significantly (P<0.05) and (P<0.01)

respectively as compared to healthy control goats (group C). After treatment, the values returned to normalcy.

The average values of serum chloride (mEq/l) and serum sodium (mEq/l) in healthy control goats (group C) were observed to be 105.98 ± 1.76 and 140 ± 2.12 respectively. In pre-treated groups viz. mild acidotic goats (group T_1), the values (110.51 ± 1.79 & 142 ± 1.18) were found to be increased non-significantly ($P<0.05$) as compared to healthy control goats (group C) but the values in moderate (117.51 ± 1.34 & 149 ± 1.72) and in severe acidotic goats (126.99 ± 1.89 & 165 ± 1.93) respectively, (group T_2 & T_3) were found to be increased significantly ($P<0.05$) when compared to healthy control goats (group C).. These values were found to be returned normalcy following treatment.

The mean value of serum potassium (mEq/l) in healthy control goats (group C) was found to be 4.27 ± 0.22 whereas, in pre-treated groups viz. mild, moderate and severe acidotic goats (group T_1, T_2 & T_3) were found to be 4.57 ± 0.09, 3.88 ± 0.07 and 3.29 ± 0.10 respectively. The mean value of serum potassium (mEq/l) in mild acidotic goats was found to be non-significantly ($P<0.05$) increased. However, in moderate and severe acidotic goats (group T_2 & T_3) the values were found to be significantly ($P<0.05$) decreased as compared to healthy control goats (group C).

In the present study, for therapeutic measures adopted were neutralization of the ruminal acidosis by oral or parentral or both administration of antacids, restoration of ruminal motility, ruminal tone, microbial motility, and activity by oral administration of rumenotoric drugs (herbal prepartions) and by ruminal cud from healthy or healthy slaughtered

complex liquid (Ambiplex) @ 5ml orally twice daily for one weeks was given. When ruminal fluid pH returned towards normalcy, ruminal-cud from healthy goats/slaughtered goats administered orally @ 150-200ml per day for 3-4 days. In spite of all treatment, two goats of this group which were severely affected could not survive and died. Recovered goats of this group looks active and alert. They showed sing and symptoms as like healthy control goat (group C) in the present study.

On the basis of present investigation following points have been concluded:

1. In the present study it was found that, mostly those goats, which were confined to the house or kept as a stall feeding or the male goats kept for some cerömonial function like 'Bakarid' were suffered mostly due to feeding of rice, boiled rice, gruel, chapati or accidently by wheat or the kitchen offals.
2. Basing on the determination of pH of ruminal fluid and severity of the clinical signs, the goats were exhibited mild, moderate and severe acidotic signs.
3. pH of the ruminal fluid, motility of rumen, motility and activity of rumen microflora were found to be decreased in the present study.
4. The biochemical parameters that were studied viz. the blood lactic acid and glucose was found to be highly elevated depending upon severity of acidosis.
5. The serum chloride and sodium level was found to be increased whereas, the serum potassium concentration was decreased in the present study.

6. Mild acidotic groups of goats responded well to the only oral therapy of a combination including Anigest, Rumbion and Soda bicarb along with Vit. B- Complex liquid (Ambiplex).
7. The moderate acidotic goats were successfully treated with the above combination along with parentral administration of Soda-bicarb, Anistamin and Dexamethazone (Dexona).
8. The severely acidotic goats were given all the above treatment including Steclin bolus, Yea-sac bolus, Rumigest and Normal saline as fluid therapy to overcome the degree of dehydration.
9. Two severely affected acidotic goats out of five died on 3rd day of treatment and rest recovered gradually.

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BIBLIOGRAPHY

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Acharya, R. M. (1982). Sheep and Goat Production-Role of sheep and goat diseases in causing mortality and reduce production. National Seminar on sheep and goat diseases, CSWRI, Avikanagar, Rajasthan 28-30th May, 1982.

Ahrens, F. A. (1967). Histamin, Lactic acid and hypertonicity as factors in the development of ruminitis in cattle. *Am. J. Vet. Res.* **28** : 1335-1342.

Ahuja, A. K.; Randhawa, S. S. and Rathor, S. S. (1990). Effect of monensin in ameliorating subacute lactic acidosis in buffalo calves. *Acta, Veterinaria Brono.* **59** (3-4) : 171-178.

Allison, M. S.; Bucklin, J. A. and Dougherty, R. W. (1964). Ruminal changes after overfeeding with wheat and the effect of intraruminal inoculation on adaptation to ration containing wheat, *J. Anim. Sci.* **23** : 1164 – 1171.

Allison, M. J.; Bucklin, J. A. and Dougherty, R. W. (1972). Overfeeding effect on caecum and rumen bacteria. *J. Anim. Sci.* **35** : 259.

Allison, M. J.; Robinson, I.M.; Daugherty, R. W. and Bucklin, J. A. (1975). Grain overload in cattle and sheep:changes in microbial population in caecum and rumen. *Am. J. Vet. Res.* **36** (2) : 181 – 185.

Amstel, S.R.V. (1983). Oral antacid treatment in clinical rumen acidosis. *J. South African Vet. Assoc.* **54** : 265 – 266.

Angelov, G.; Nikolov, Y. and Angelov, A. (1995). Changes in acid-base variables and some biochemical parameters in caprine acute rumen acidosis. *Vet. Arhiv.* **65** : 43 – 48.

Angelov, G.; Nikolov, Y. and Angelov, A. (1996). Changes in acid-base parameters, blood sugar and blood lactate in experimental acute rumen acidosis in sheep. *Indian Vet. J.* **73** (3) : 309 – 314.

Annison, E. F.; Lindsay, D. E. and white, R. R. (1963). Metabolic interactions of glucose and lactate in sheep. *Biol. Chem. J.* **88** : 3.

Aslan, V.; Thamsberg, S. M.; Jorgensen, R. J. and Basse, A. (1995). Induced acute ruminal acidosis in goats treated with yeast (*Sachoromyces cervisiac*) and bicarbonat. Frederiksberg, Denmar, *Acta Vet. Scand*, **36** (1) : 65 - 77.

Barker, S. B. and Summerson, W. H. (1941). The colorimetric determination of lactic acid in biological material. *J. Biol. Chem.* **138** : 535.

Basak, D. N. (1993). *Ibid*, **70** : 662 (Cited by Bhikane *et al.*, 1996. *Indian Vet. J.* **73** : 321 – 323).

Basak, D. N.; Das, S. K. and Chakrabarti, A. (1994). Studies on the endocrinal changes in experimentally induced acid indigestion in goats. *Indian Vet. J.* **71** : 587 – 589.

Basak, D. N.; Pan, S.; and Chakarabarti, A. (1993). Physiochemical and microbial changes in rumen liquor of experimentally induced lactic acidosis in goats. *Indian J. Anim. Sci.* **63** (3) : 263-267.

Bhatia, S. S.; Sra, I.S. and Dwarikanath, P.K. (1980). Plasma monoamine oxidase level in non-specific anorexia of ruminants. *Indian Vet. J.* **57** : 697-698.

Bhikane, A. U.; Ali, M.S.; Usturge, S. M.; and Salunke, V. M. (1996). A case report on ruminal lactic acidosis in a buffalo. *Indian (Vet. J.* **73** (3) : 321 –323).

Bieniek, K. (1981). Studies on liver function in experimental rumen acidosis in cattle. *Polskie Archiwum Weterynaryine* **23** : 103 – 116. (*Vet. Bull.* **52** : 764).

Blake, J. T.; Allen, R. S. and Jacobson, N. L. (1957). The influence of various factors on surface tension and pH of the ruminal fluid. *J. Anim. Sci.* **16** : 190 – 200.

Blood, D. C. and henderson, J. A. (1968). Veterinary Medicine, **3rd Edn.** Bailliers, Tindall and Cassell, London.

Blood, D. C.; Radostits, O. M.; Henderson, J. A.; Arundel, J.H. and Gay, C.C. (1983). Veterinary Medicine, **6th Edn.** The English Language Book Society and Bailliere, Tindall.

Blood, D. C. and Radostits, O. M. (1989). Veterinary Medicine. **7th Edn.** ELBS, Bailiere and Tindall, London.

Brandly, C. A., and Jungher, E. L. (1955). *Advances in Veterinary Sci.* Vol. **II**. Academic press. Inc. Publisher, New Delhi : 213 – 240.

Braun, U.; Rihs, T. and Scherfer, U. (1992). Ruminal lactic acidosis in sheep and goats. *Vet. Rec.* **130** (16) : 342 – 349.

Brent, B. E. (1976). Relationship of acidosis to other feedlot ailments. *J. Anim. Sci.* **43** : 930 – 935.

Broberg, G. (1960). Acute over eating with cereals in ruminants. Various aspects of disease resulting from the consumption of excessive amounts of carbohydrate rich feeds, including the studies of lactic acid and thiamine metabolism. PP. 83. *Lovisa Nya Fryekeri Lovisa, Finland* – AB (*Vet. Bull.* **31** : 3681).

Bullen, J. J. and Scarisbrick, R. (1957). Enterotoxaemia of sheep. Experimental production of the disease. *J. Path. Bact.* **73** : 495 – 509.

Cakala, S.; Borkowski, T. and Albrycht. A. (1974). Rumen acidosis in sheep induced by various doses of sucrose. *Polskie Archiwum Wet.* **17** (1) : 117-130. (*Vet. Bull.* **45** (6) : 3352).

Cakala, S.; Albrycht, A. and Bieniek. K. (1975). Biochemical changes in the rumen fluid and blood in the cows fed a large amount of grain food. *Bull. Vet. Inst. Pulawy* **19** : 90-96. (*Vet. Bull.* **46** : 5219).

Cao, G. R.; English, P. B.; Fillippich, L. J. and Inglis, S. (1987). Experimental induced lactic acidosis in the goat. *Aust. Vet. J.* **64** (12) : 367-370.

Castello, H. H. (1968). Indigestion in cattle. *Mod Vet. Practice.* **49** (10) : 38-42.

Chakrabarti A. (1988). Text Book of Clinical Veterinary Medicine **1st Edn.** Kalyani Publishers, New Delhi- Ludhiana.

Chaplin, R. K. and Jones, G. A. (1973). Rumen microbial changes during experimental rumen acidosis. *J. Dairy Sci.* **56** (5) : 671.

Cherkasov, V. K. (1959). Changes in the infusorial fauna of the forestomachs of cattle with ruminal atony. *Trude. Mesk. Vet. Acad.* **24** (2) : 131 – 142. (*Vet. Bull.* **31** : 1579).

Choudhuri, P. C.; Karunanidhi, P. S.; Misra, S. K. and Sen, M. M. (1985). Evaluation of rumen liquor in the goat. In : All India Conference on current concepts of various disorders of ruminant stomach. 8th – 9th January, 1985. Tamilnadu Agricultural University, Madras.

Choudhuri, P. C.; Randhawa, S. S. and Misra, S. K. (1980). Effect of lactic acidosis on electrolyte changes in blood and rumen liquor in buffalo calves. *Zentbl. Vet. Med.* **27** A : 358-363.

Chawla, D. S.; Bhatnagar, D. S. and Sunderesan, D. (1981). Dairy goats at Karnal, NDRI Publication No. 195.

Cleon, V. K. (1988). Lactic acidosis. Jensen and Swifts diseases of sheep. Lee and Febriger, Philadelphia, U.S.A. pp. 141 – 144.

Coles, E. H. (1974). Vaterinary Clinical Pathology, **2nd End.**, pp. 192 – 271. W. B. Saunder Company, Philadelphia, London, Toronto.

Crichlow, E. C. (1989). Loss of forestomach motility in sheep experiencing ruminal lactic acidosis is not dependent on duodenal acidification by lactic acid. *Journal of Veterinary Medicine*, A **36** (1) : 39-45.

Das, S. (1990). Studies on experimental rumen acidosis in goats with special reference to liver functions and therapy. *M. V. Sc. thesis* submitted to Bidhan Chandra Krishi Viswavidyalaya, Madia, West Bengal.

Das, S. K. and Misra, S. K. (1991). Studies on biochemical changes in rumen liquor before and after therapy in experimental rumen acidosis in goats. *Indian J. Vet. Med.* Vol. **11** . No. 1 and 2. 1991 pp. 26 – 27.

Das, S.K. and Misra, S.K. (1992). Liver function in experimental rumen acidosis in goats. *Indian J. Anim. Sci.* **62** (3): 243-244.

Dash, P.K.; Misra, S.K. and Mohantry, G.P. (1972). Effect of acute indigestion on the rumen protozoa population and blood picture of Indian cattle. *Indian Vet. J.* **49** (7): 672-677.

Day, A. C. (1980). Indian medical plants used in Ayurvedic preparation. Eds. B. Singh and M. P. Singh, Dehradun, India.

Delak, M. and Adamic, S. (1959). Sucrose intoxication in sheep. *Vet. Arhiv.* **29** : 214-223. (*Vet. Bull.* **30** : 1517).

Dirksen, G. (1965). Rumen acidosis in cattler *Vet. Med. Rev. Leverkusen No.* 2. pp. 98-125.

Dirksen, G. (1967). Biochemistry of ruminal acidosis in cattle. *Schweizer Archieve Fierbeilk.* **109** : 28 – 34. (*Vet. Bull.* **37** : 2788).

Dirksen, G. (1970). Acidosis. In A. T. Phillipson (Ed.) Proceedings of 3rd International symposium on the physiology of Digestion and Metabolism in the ruminant. P. 612 – 623. *Oriel Press, New Castle, England.*

Dougherty, R. W.; Rilley J. L.; Baetz, A. L.; Cook, H. M. and Coburn, R. L. (1975). Physiologic studies of experimentally grain-engorged cattle and sheep. *Am. J. Vet. Res.* **36** (6) : 833 – 835.

Dunlop, R.H. (1961). A study of factors related to the functional impairement resulting from loading the rumen of cattle with high carbohydrate feeds. *Ph. D. Thesis* Univ. Minn. St. Poul. Minn. (Cited by Dunlop, R.H. and Hamond, P.B., 1965. D. lactic acidosis of ruminants annals New York Acad. Sci., **119**: 1109-1132).

Dunlop, R. H. (1972). Pathogenesis of ruminants lactic acidosis. *Adv. Vet. Sci. Comp. Med.* **16** : 259-262.

Dunlop, R. H. and Hammond, P. B. (1965). D-lactic acidosis of ruminants. *Animals of New York Acad. Sci.* **119** : 1109 – 1132.

Dutta, S. K. (1992). Chlinico – biochemical and therapeutic studies on sub-acute lactic acidosis in sheep. *M.V.Sc. thesis* submitted in IVRI Izatnagar, U. P.

Dwivedi, S. K.; Patra, R. C.; Dey, S. and Swarup, D. (2000). Therapeutic efficacy of Gastricare against ruminal dysfunction in calves. *The Veterinarian.* **24** (1) : 22 – 25.

Elam, C. J. (1976). Acidosis in feedlot cattle. Practical observations. *J. Anim. Sci.* **42** : 898-901.

Flachowsky, G.; Lohnert, H. J.; Henning, A. (1974). Some aspects of the supply of thiamine to lambs after the induction of ruminal acidosis. *Archiv. Fur Exp. Veterinarme dizin.* **28** : 543 – 549. (*Vet. Bull.* **45** : 1969).

F.A.O. Rome (1991). Production year book **44**. F.A.O., Rome.

Folin, O. and Wu, H. (1920). *J. Biol. Chem.* **41** : 367.

Frankel, S.; Reitman, S. and Sonnenwirth, A.C. (1970). Gradwoh's Clinical Laboratory Methods and Diagnosis. **7th Edn**. The C. V. Moshy Company, Saint Louis.

Gaikwad, R. V.; Jagadish, S. and Bhalerao, D. P. (1993). Studies on primary indigestion in milch buffaloes. *Indian Vet. J.* **70** : 1046 – 1049.

Gibbons, W. J. (1963). Rumen intoxication in cattle. *Mod. Vet. Practice.* **40** (4) : 45 – 49.

Gnanaprakasam, V. (1970). Rumen acidosis in goats. *Indian Vet. J.* **47** : 904-910.

Gokce, G. and Imren, H.Y. (1998). The studies on the experiments of preventive treatment with sodium bicarbonate supplementation to feeds in sheep with ruminal acidosis. *Turk-Veterinerlik-ve-Hayvancilik-Dergisi.* **22** (4): 333-343.

Gutowaski, S. (1988). Oxygen consumption and the pH of the rumen contents in experimental acidosis in sheep. *Zeszyty Naukowe*

Akademii Rolniczej Wewroclawiu, Weterynaria **44** (172) : 9-16 (*Vet. Bull.* **59** (9) : 5408).

Helzdasz, Z. and Nicponn, J. (1977). Blood buffer system in experimental acidosis of cows and attempts at preventing it. *Polskie Arch. Weterinaygne.* **20** (2) : 103 – 118 (*Vet. Bull*, **48** (4) : *Abstr.* 2447).

Heijlasz, L.; Mazur, O. and Rauluszkiewicz, S. (1984). Effect of experimental acidosis in under function in cows. *Medycyna Weterynaryjna.* **40** (4) : 217 – 220.

Heuter, F. G.; Shaw, J. C. and Doetsch, R. N. (1956). Absorption and dissimilation of lactates added to the bovine rumen and the resulting effect on blood glucose. *J. Dairy Sci.* **39** : 1430.

Hoflund, S.; Quin, J. I. and Clark, R. (1948). Studies on the alimentary tract of Merino sheep in South Africa. XV. The influence of different factors on the rate of cellulose digestion (a) in the rumen and (b) in the ruminal ingesta as studied in vitro. Understepoort *J. Vet. Sci. Anim. Husb.* **23** (1-2) : 395 – 409 (cited by Hosenberger, G., 1979. Clinical examination of cattle, **2nd Edn**. Verlarpoulparley, Berlin and Hamburg.

Hoflund, S. (1967). Animal diseases associated with the use of detoriated feeding stuffs under Swidish conditions. (*Vet. Bull.* **37** : 701-717).

Howard, J. L. (1986). Current Veterinary Therapy : Food animal practice – 2 W. B. Saunders Company, London.

Huber, T. L. (1969). Lactic acidosis and renal Function in sheep. *J. Anim. Sci.* **29** : 612 – 615.

Huber, T. L. (1971). Effect of acute indigestion on compartmental water volumes and osmolarity in sheep. *Am. J. Vet. Res.* **32** (6) : 887 – 890.

Huber, T. L. (1973). Lactic acidosis, prevention by rumen inoculation. *J. Anim. Sci.* **36** : 226 – 227.

Huber, T. L. (1976). Physiological effects of acidosis on feedlot cattle. *J. Anim. Sci.* **43** : 902 – 909.

Huber, T. L.; Wilson, R. C. and Mc Garity, S. A. (1984). Hepatic metabolite concentration in lactic acidotic sheep. *Am. J. Vet. Res.* **45** (6) : 1209–1211.

Hungate, R. E.; Dougherty, R. W.; Brynt, M. P. and Cello, R. M. (1952). Microbiological and physiological changes associated acute indigestion in sheep. *Corneel Vet.* **42** (4) : 423 – 449.

Hungate, R. E. (1966). The rumen and its microbes. Academic Press, New York.

Hyldgaard-Jensen, J. and Simesen, M. G. (1966). Grutforgiftning hos Kvaeg. *Nord. Vet. Med.* **18** : 73 – 94.

Irwin, L. N.; Mitchell, G. E. Jr.; Tucker, R. E. and Schelling, G. T. (1979). Histamin, tyramine, Tryptamine and electrolytes during glucose induced lactic acidosis. *J. Anim. Sci.* **49** (2) : 367 – 374.

Ivanov, I. T. (1974). Studies on rumen acidosis and diabetes syndrome in cattle. *Veterinarno meditsinski* Nauki, Bulgaria. **II** : 59 – 63. (*Vet. Bull.* **44** : 6117).

Jagos, P.; Bouda, J.; Dvorak, R.; Slleck, J. and Jurajdova, P. (1977). Chronic metabolic acidosis in dairy cows. *Veterinari Medicina*, **22** : 143 – 151. (*Vet. Bull.* **48** : 343).

Jensen, R. and Mackey, D. R. (1965). Diseases of feed lot cattle. *Lea and febiger*, Philadelphia.

Joshi, B. P. (1969). Grain engorgement in bovine. A case of acid indigestion in bullock. *Indian Vet. J.* **46** : 343 – 345.

Joshi, B. P. and Ludri, R. S. (1970). A note on biochemical aspects rumen dysfunction in buffaloes. *Guj. Vet.* **4** : 102 – 104.

Juhasz, B. and Szegedi, B. (1968a). Pathogenesis of ruminal digestive disordes caused by easily digestible carbohydrate. Magy. Allatorv Lap. **23** : 60 – 68. (*Vet. Bull.* **39** : 2155).

Juhasz, B. and Szegedi, B. (1968b). Aetiology of rumen acidosis in bovines. *Arch. Exp. Vet. Med.* **22** : 969 – 993.

Juhasz, B. and Szegedi, B. (1968c). Experiments on the treatment of ruminal acidosis in sheep *Magy. Allatorv.* Lap. **23** : 625 – 630.

Juhasz, B. and Szegedi, B. (1968d). Pathogenesis of rumen over load in sheep. *Acta Vet. Acad. Sci. Hungaricae*, **18**: 63.

Juhasz, B. and Szegedi, B. (1976). Experimental studies on the prophylaxis and therapy of rumen acidosis. *Zentralblaft fur Veterinarmedizine* **23** A (6) : 485 – 501. (*Vet. Bull.* **47** (3) : 1658).

Kaneko, Jiro, J. I. (1989). Clinical biochemistry of domestic animals. **4th Edn.**, Academic Press, INC, California.

Kapoor, L. D. (1990). Handbook of Ayurvedic Medical Plants, CRC pres, Boca Raton, Florida.

Kelly, W. R. (1974). Veterinary Clinical Diagnosis. **2nd Edn.** Bailliere Tindall, London.

Kezar, W. W. and Church, D. C. (1979a). Effect of thiopeptin and sodium bicarbonate on prevention of lactic acidosis induced in sheep. *J. Anim. Sci.* **49** (5) : 1396 – 1402.

Kezar, W. W. and Church, D. C. (1979b). Ruminal changes during onset and recovery of induced lactic acidosis in sheep. *J. Anim. Sci.* **49** (5) : 1161 – 1167.

Koer, W. C.; Briton R.; Klopfenstein, T. J. and Woods, W. R. (1976). Ruminal histamine, lactate and animal performance. *J. Anim. Sci.* **43** (3) : 684 – 691.

Kovac, G.; Mudron, P. and Bartko, P. (1986). Comparative studies on the pH of blood, rumen and abomasal fluid, faeces and urine in cattle. *Folia Veterinaria.* **30** (2) : 21 – 35. (*Vet. Bull.* 1987. **51** (11) : 7228).

Krogh, N. (1959). Studies on the alterations in the rumen fluid of sheep, especially concerning the microbial composition when readily available carbohydrates are added to the food I. Sucrose. *Acta. Vet. Scand,* **I** : 74 – 79. (*Vet. Bull.* **30** : 1516).

Krogh, N. (1960). Studies on the alterations in the rumen fluid of sheep, especially concerning the microbial composition when readily available carbohydrates are added to the food. II. Lactose. *Acta. Vet. Scand.,* **2** : 383.

Kumar, A. and Joshi, B. P. (1994). *Indian Vet. J.* **71** : 735 (Cited by Bhikane *et al.,* 1996. *Indian Vet. J.* **73** : 321 – 323).

Kuusksalu, V. (1988). Pathophysiological aspects of lactic acidosis in sheep. Eesti NSV Teaduste Akadeemia Toimetised, Biologia. **37** (2) : 167 – 172. (*Vet. Bull.* **58** : 7184).

Lal, S.B. (1988). Studies on systemic effects of experimental acidosis and its clinical management with cud transplant in goats. *Ph. D. Thesis* submitted to IVRI. Izatnagar, Bareilly, U.P.

Lal, S.B.; Dwivedi, S.K.; Sharma, M.C. and Swarup, D. (1992). Biopathological studies in experimentally induced ruminal acidosis in goat. *Indian J. Anim. Sci.* **62** (3): 200-204.

Lal, S. B.; Dwivedi, S. K.; Sharma, M. C. and Swarup, D. (1989). Clinico-biochemical and microbial studies in rumen liquor in experimental acidosis in goats. *Indian J. Vet. Med.* **9** (2) : 81 – 85.

Lal, S. B.; Dwivedi, S. K.; Celly, C. S. Singh, G. R.; Sharma, M. C. and Swarup, D. (1993). Biochemical and electrocardiographic changes in experimental rumen lactic acidosis in goats. *Indian J. Anim. Sci.* **63** (9) : 952 – 956.

Lal, S. B. Swarup, D.; Dwivedi, S. K. and Sharma, M. C. (1991). Biochemical alteration in serum and cerebrospinal fluid in experimental acidosis in goats. *Res. Vet. Sci.* **50** (2) : 208 – 210.

Legee, P. (1980). Therapy of acidosis in dairy cows. Bulletin Mensuel de La Societe Veterinairee Pratique de France **64** : 185 – 194. (*Vet. Bull.* **50** : 7614).

Lee, G. J. (1977). *Aust. Agric. Res.* **28** : 1075 (Cited by Blood *et al.* 1983, Veterinary Medicine **6th Edn**. Bailliere, Tindall, London).

Lippman, R. (1973). Hints on controlling rumen acidosis in intensive fat lamb production-Monafschefte fur Veterinamedizine 28 Heft (**23**) : 900 – 904. (*Vet. Bull.* , **44** (5) : 2409).

Li, X. J.; Wang, N.; Yu, Y. C.; Liu, W. H. and Wang, L. (1984). Experimental maize acidosis in the rumen of xinjiange fine-wool sheep. *J. Vet. Sci. and Tech.. No.* **12** : 14 – 17. (*Vet. Bull.*, **55** (10) : 6471).

Mackenzie, D. D. S. (1967). Production and utilization of lactic acid by the ruminant. A review *J. Dairy Sci.* **50** : 1172 – 1737.

Mahadevan, A. R. (1982). Study on the technique of preservation and keeping quality of bovine ruminal fluid under field conditions. *M.V.Sc. thesis* submitted to Tamil-Nadu Agricultural University, Coimbatore.

Mandial, R. K.; Prasad, B. P. and Pal, B. (1994). Efficacy of Rumbion in experimental acid indigestion in calves. *Indian J. Indg. Med.* **11** (1) : 19 – 23.

Mc Manus, W. R. and Bigham, M. L. (1978). Effects of mineral buffers on the rumen flora of sheep fed grains diets. *Res. Vet. Sci.* **24** (2) : 129 – 133.

Mc Intosh, G. H.; Filsell, O. H. and Jerrett, I.G. (1973). Kidney function and net glucose production in normal and acidotic sheep. *Aust. J. Biol. Sci.* **36** (6) : 1389 – 1394.

Melvin, J. S. (1977). Dukes Physiology of Domestic Animals. **9th Edn.** Comstock Publishing Associates, Thaca and London.

Misra, S. K. (1991). Therapeutic efficacy of Rumbion, a herbal rumenotoric drug in cases of simple indigestion in ruminants. *Indian. J. Indg. Med.* **8** : 1.

Misra, S. K.; Das, P. K. and Mohenty, G. P. (1972). The protozoan fauna of the rumen and reticulum of Indian cattle. *Indian Vet. J.* **49** : 463 – 470.

Misra, S. K. and Singh, U. (1974). Studies on the clinicopathological and therapeutic aspects of indigestion in cattle. *Indian Vet. J.* **51** : 698 – 704.

Muir, L. A. and Barreto, A. Jr. (1979). Sensitivity of *Streptococcus bovis* to various antibiotics. *J. Anim. Sci.* **48** : 468 – 473.

Muir, L. A.; Duquette, P. F.; Rickes, E. L. and Smith, G. E. (1980). Thiopeptin for the prevention of ovine lactic acidosis induced by diet chenges. *J. Anim. Sci.* **51** : 1182 – 1188.

Mullen, P. A. (1976). Overfeeding in cattle : Clinical, biochemical and therapeutic aspects. *Vet. Res.* **98** : 439 – 443.

Mullick, D. N. (1964). Review of the investigation on the physiology of Indian buffalo. *Indian J. Dairy Sci.* **17** : 45.

Nagaraja, T. G.; Avery, T. B.; Bartley, E.E. and Roof, S. K. and Dayton, A. D. (1982). Effect of lasalocid or monensin or thiopeptin on lactic acidosis in cattle. *J. Anim. Sci.* **54** : 649 – 658.

Narendra, R. M.; Tripathy, S. B. and Das, P. K. (1990). A note on the treatment of ruminal acidosis in calves. *Indian J. Vet. Med.* **10** (1) : 74 – 75.

Nauriyal, D. C. and Baxi, K. K. (1978). Bio-medical profile of cross-bred cattle and buffaloes on experimentally induced ruminal lactic acidosis. II intra-ruminal administration of crushed oat grain. *J. Res.*, PAU, Ludhiana. **15** (4) : 433 – 438.

Nauriyal, D. C. (1975). Studies on clinico-biochemical and therapeutic aspects of rumen dysfunctions with particular reference to rumen acidosis in buffalo and cross-bred cattle in Punjab. *Ph. D. Dissertation* submitted to P.A.U., Ludhiana.

Nauriyal, D. C. and Baxi, K. K. (1981). Bio-medical profile of cross-bred cattle and buffaloes in experimentally induced ruminal lactic

Muir, L. A. and Barreto, A. Jr. (1979). Sensitivity of *Streptococcus bovis* to various antibiotics. *J. Anim. Sci.* **48** : 468 – 473.

Muir, L. A.; Duquette, P. F.; Rickes, E. L. and Smith, G. E. (1980). Thiopeptin for the prevention of ovine lactic acidosis induced by diet chenges. *J. Anim. Sci.* **51** : 1182 – 1188.

Mullen, P. A. (1976). Overfeeding in cattle : Clinical, biochemical and therapeutic aspects. *Vet. Res.* **98** : 439 – 443.

Mullick, D. N. (1964). Review of the investigation on the physiology of Indian buffalo. *Indian J. Dairy Sci.* **17** : 45.

Nagaraja, T. G.; Avery, T. B.; Bartley, E.E. and Roof, S. K. and Dayton, A. D. (1982). Effect of lasalocid or monensin or thiopeptin on lactic acidosis in cattle. *J. Anim. Sci.* **54** : 649 – 658.

Narendra, R. M.; Tripathy, S. B. and Das, P. K. (1990). A note on the treatment of ruminal acidosis in calves. *Indian J. Vet. Med.* **10** (1) : 74 – 75.

Nauriyal, D. C. and Baxi, K. K. (1978). Bio-medical profile of cross-bred cattle and buffaloes on experimentally induced ruminal lactic acidosis. II intra-ruminal administration of crushed oat grain. *J. Res.*, PAU, Ludhiana. **15** (4) : 433 – 438.

Nauriyal, D. C. (1975). Studies on clinico-biochemical and therapeutic aspects of rumen dysfunctions with particular reference to rumen acidosis in buffalo and cross-bred cattle in Punjab. *Ph. D. Dissertation* submitted to P.A.U., Ludhiana.

Nauriyal, D. C. and Baxi, K. K. (1981). Bio-medical profile of cross-bred cattle and buffaloes in experimentally induced ruminal lactic

acidosis. III. Intra-ruminal crushed barley grain administration. *Indian. J. Vet. Med.* **1** : 51 – 57.

Nicholas, R. E. and Penn, K. E. (1958). Simple methods for the detection of unfavorable changes in rumen ingesta. *J. Am. Vet. Med. Assoc.* **133** : 275 – 277.

Nikolov, Y. (1998). Clinical experimental studies on acute rumen acidosis in buffaloes *(Bubalus bubalis).* IV. Influence of acidosis on blood, rumen liquid and urine electrolytes. *Veteriarski Arhiv.* **68** (1): 1-9.

Nikolov, Y. (2000). Clinical experimental studies on acute rumen acidosis in sheep with a pre-existing chronic lead and organophosphorus compound intoxication. I. Clinical and haematological changes. *Vet. Arhiv.* **70** : 103 – 112.

Nisbet, A.; Beddows, A. CL. And Johne N. A. (1963). Overfeeding of barely to sheep. *Vet. Rec.* **75** : 152.

Nour, M.S.M.; Abusamra, M. T. and Hago, B.E.D. (1998). Experimentally induced lactic acidosis in Nubian goats : clinical, biochemical and pathological investigations. *Small Ruminant Research* **31** : 7 – 17.

Ogilvie, T. H.; Butter, D. G.; Gartley, C. J. and Dohov, I. R. (1983). Magnesium oxide induced metabolic alkalosis in cattle. *Canadian J. Comp. Med.* **47** : 108 – 111.

Oser, B.L. (1965). Howk's Physiological Biochemistry, **14th Edn.** The Blackiston Division. Mcgraw Hill Book Company.

Parthasarthy, D. and Phillipson, A. T. (1953). The movement of potassium, sodium, chloride and water across the rumen epithelium of sheep. *J. Physiol.* **121** : 452 – 469.

Patel, R. M. (1990). Clinico-biochemical studies on experimental ruminal acidosis and its therapy in goats. *M. V. Sc. thesis*, submitted in Gujarat Agricultural University, Sardar Krishinager.

Patra, R. C. (1991). Clinico-biochemical and therapeutic studies on experimental acidosis in sheep. *M. V. Sc. Thesis* submitted to Deemed University I.V.R.I. Izatnagar, Bareilly, U.P.

Patra, R. C.; Lal, S. B. and Swarup, D. (1993). Physiochemical alterations in blood, cerebrospinal fluid and urine in experimental lactic acidosis in sheep. *Res. Vet. Sci.* **54** (2) : 217 – 220.

Patra, R. C.; Lal, S. B. and Swarup, D. (1996). Biochemical profile of rumen liquor, blood and urine in experimental acidosis in sheep. *Small Ruminant Research* **19** : 177 – 180.

Patra, R. C.; Lal, S. B. and Swarup, D. (1997). Therapeutic management of experimental ruminal acidosis in sheep. *Indian Vet. J.* **74** : 237 – 240.

Phillipson, A.T. (1942). The fluctuation of pH and organic acids in the rumen of the sheep. *J. Exp. Biol.* **19**: 186.

Phillipson, A. T. (1952). The fatty acids present in rumen of lambs, fed on flaked maize ration. *Br. J. Nutr.* **6** : 190 – 198.

Phillipson, A.T. (1955). Rumen dysfunction. *Advance in Veterinary Sci.* **Vol-2.** Academic Press INC., Publishers, New York, NY.

Pinkiewicz, E.; Pomorski, Z.; Rukasz, I. and Szalecki, J. (1974). Histamine, lactic acid and pyruvic acid in the course of acid indigestion in cattle. *Annales Universitatis Mariae Curie-Sklodwska Sectio.* **27** : 175 – 192. (*Vet. Bull.* **44** (12): 6118).

Pounden, W. D.; Conrad, H. R. and Hibbs, J. W. (1954). Observations on some substitute for rumen inoculation. *J. Am. Vet. Med. Assoc.* **124** : 394 – 396.

Pradhan, N. R.; Misra, S. K. and Pan, S. (1988). A note on rumen liquor of healthy goats. *Indian J. Vet. Med.* **8** (1) : 66-67.

Prasad, J.; Ahluwalia, S. S. and Joshi, B. P. (1972). Clinico-biochemical studies in indigestions in cattle and buffaloes. *Indian J. Anim. Sci.* **42** (11) : 911 – 914.

Prasad, J. and Joshi, B. P. (1971). Liver function studies in ruminal derrangement in buffaloes, *Orissa Vet. J.* **6** (1) : 27 – 29.

Prasad, J.; Ahluwalia, S.S. and Joshi, B.P. (1973). A note on experimental rumen acidosis in buffalo and its treatment with sodium bicarbonate. *Indian. J. Anim. Sci.* **43** : 77 – 78.

Prasad, J. and Joshi, B. P. (1975). Biochemical exploration of primary rumen impactions in Zebu and buffaloes. *Indian Vet. J.* **52** (5) : 366 – 369.

Prasad, J.; Joshi, S. V. and Rekib, A. (1976a). Studies on physiochemical and therapeutic aspects of primary anorexia syndrome in sheep and goats. *Mahavet.* **3** (11) : 13 – 14.

Prasad, J. and Rekib, A. (1975). Clinical management of ruminal acidosis with sodium bicarbonate and rumen cud transplant. *Indian Vet. J.* **32** : 317-318.

Prasad, J.; Rekib, A. and Joshi, S. V. (1976b). Clinical trials with Anorexon (pfizer) in anorexia syndrome in ruminants. *Indian Vet. J.* **53** : 297 – 299.

Prins, R. A. and Van, Hoven, W. (1977). Carbohydrate fermentation by the rumen cilliate. *Isotriche prostoma. Prostiatologia.* **13** : 549 – 556. (Cited by Randhawa, *et al.*, 1989, *Indian J. Vet. Med.* **9** : 1 – 7).

Purser, D. B. and Moir, R. J. (1966). Dietary effects upon concentration of protozoa in the rumen. *J. Anim. Sci.* **25** : 668 – 674.

Radostits, O. M.; Blood, D. C. and Gay, G. C. (1994). Veterinary Medicine, **8th Edn**. Bailliere Tindall, London.

Rai, G. S. and Pandey, M. D. (1980a). Effect of management on rumen function in goats. I. TVFA, PH, freezing point, depression, buffering capacity and *in vitro* TVFA production. *Indian Vet. J.* **57** : 120 –125.

Rai, G. S. and Pandey, M. D. (1980b). Effect of management on rumen function in goats : II. Nitrogen functions, microbiology and transminase activity in rumen liquor. *Indian Vet. J.* **57** : 205 – 210.

Randhawa, S. S. (1979). Studies on biochemical changes in ruminal fluid, blood and cerebrospinal fluid in ruminal acidosis, *M.V.Sc. thesis* submitted to Punjab Agricultural University, Ludhiana.

Randhawa, S. S.; Ahuja, A. K. and Rathor, S. S. (1989). Effect of lactic acidosis on microbial and biochemical changes in rumen liquor of buffalo calves. *Indian J. Vet. Med.* **9** : 1-7.

Randhawa, S.S.; Choudhury, P.C. and Misra, S.K. (1980). Physio-chemical changes in cerebrospinal fluid in experimental ruminal acidosis in buffalo calves. *Res. Vet. Sci.* **29**: 118-119.

Randhawa, S. S.; Das, L. N. and Misra, S. K. (1982). Comparative biochemical and pathological studies on acute ruminal acidosis induced by molasses and grain feeding in buffalo calves. *Acta Vet. Scand.* **36**: 257-264.

Randhawa, S. S.; Gupta, P.P.; Ahuja, A. K. and Rathor, S. S. (1988). Clinico-biochemical and pathological studies on experimental subacute acidosis in buffalo calves. *Indian. J. Anim. Sci.* **58** (9) : 1024 – 1032.

Reddy, T.V.K. and Nair, S. G. (1971). Studies on rumen function in goats. *Indian J. Anim. Sci.* **41** : 462 – 467.

Rosenberger, G. (1979). Clinical examination of cattle, **2nd Edn.** Verlarpaulparley, Berlin and Hamburg.

Rosenberger, G.; Dirksen, G.; Grunder, H. D.; Grunert, E.; Krause, D. and stabber, M. (1979). Clinical Examination of Cattle, *Verlag Paul Parey, Berlin and Hamburg.*

Roychoudhury, R. K. (1981). Clinico studies on hepatic dysfunction associated with acid indigestion in Bovine. *Ph. D. Dissertation* submitted to P. A. U., Ludhiana.

Ryan, R. K. (1964). Concentrations of glucose and low molecular weight acids in the rumen of sheep following the addition of large amount of wheat to the rumen. *Am. J. Vet. Res.* **25** : 646 – 652.

Sahani, K. L. (1982). Present state and Future scope of goat industry in India. *Indian Vet. J.* **6** : 177 – 185.

Sandha, H. S. (1980). Effect of ruminal acidosis on functions of Kidney and pancreas and therapy. *M.V.Sc. thesis* submitted to Punjab Agricultural University, Ludhiana.

Sandha, H. S. and Choudhury, P. C. (1985). Status of renal function in acute ruminal lactic acidosis in Indian water buffalo (*Bubalus bubalis*). In all India convergence of current concepts of the various disorder of

ruminant stomach. 8th-9th January 1985, Tamil Nadu Agricultural University, Madras.

Sastry, G. A. (1983). Veterinary Clinical pathology. 3rd **Edn.** C.B.S. Publishers and Distributors, Shahdara, Delhi – 32.

Scarisbrick, R. (1954). Acid indigestion in a sheep fed on mangolds. *The Vet. Rec.* **66** (2) : 131 – 132.

Schales, O. and Schales, S. S. (1941). *J. of Biological Chemistry.* **140** : 879. In Hawk's physiological chemistry. Edited by Oser, B. L. (1965). **14th Edn.,** Mc Graw Hill Publishing Company, Ltd., New Delhi.

Schukken, A.; Vellinga, K. and Kamp, J. S. Van Der (1985). One persons bread is another's death. I. Ruminal acidosis in cattle fed stale bread. *Tijdschraft Noor Diergeneeskunde.* **110** (2) : 69-70. (*Vet. Bull.* **55** (5) : 3029).

Sen, M. M. (1982). Studies on grain overload in goats with reference to clinical menifestation, biochemical and microbial changes in the rumen biochemical changes in the blood and cerebrospinal fluid, pathological changes in internal organs and therapy. *Ph. D. Thesis* submitted to P.A.U., Ludhiana.

Sen, M. M.; Misra, S. K. and Choudhury, P. C. (1982). Clinico-therapeutic aspects of acute ruminal acidosis in goats. *Indian J. Vet. Med* **2** : 25 – 32.

Sen, M. M.; Misra, S. K. and Choudhuri, P. C. (1993). Blood biochemical changes in acute experimental ruminal acidosis in Barberi goat. *Indian Vet. J.* **70** (6) : 515 – 518.

Sen, M. M.; Singh, N. and Misra, S. K. (1984). Acute ruminal acidosis in goats. *Indian J. Anim. Sci.* **54** : 898 – 901.

Sethuraman, V. (1976). Studies on Bovine ruminal indigestion in natural and experimental cases and their therapy. *Ph. D. Dissertation* submitted to P. A. U., Ludhiana.

Sethuraman, V. and Rathor, S. S. (1979a). Clinical studies and therapy of experimental rumen acute acid and alkaline indigestion in bovine. *Indian Vet. J.* **56** (1) : 23 – 26.

Sethuraman, V. and Rathor, S. S. (1979b). Biochemical studies of blood in experimentally produced acute acid and alkali indigestion in bovines. *Indian J. Anim. Sci.* **49** : 699 – 702.

Shinosaki, K. (1959). Studies on experimental bloat in ruminants. 5. Effects of various volatile fatty acids induced into the rumen on the motility. *Tohoku J. Agri. Res.* **9** : 237-250.

Shinosaki, K. and Nakabayashi, K. (1974). An experimental study of rumen acidosis in ruminants. *Journal of the faculty of Agriculture, Iwate, University,* **12** (2) : 133 – 142. (*Vet. Bull.* **41** : 263).

Sinha, V. K.; Misra, S. K. and Choudhury, P. C. (1985). Clinicotherapeutic management of experimental ruminal acidosis in buffalo calves. *Indian J. Anim. Sci.* **5** : 5 – 9.

Slanina, L. (1970). Changes in the ruminal acidity of sheep produced experimentally by some rumianl metabolites. *Vet. Bull.,* **41** (3): 1936.

Slanina, L.; Cabady, R. and Assmus, G. (1970). Acid forestomach dysfunction and its treatment with Bykodigest Dt. Tierarztl, Wschr. 77 : 369-373. (*Vet. Bull.* : 3043).

Slyter, L. L. (1976). Influence of acidosis on rumen function. *J. Anim. Sci.* **43** : 910 – 929.

Smith, B. P. (1990). Large Animal Internal Medicine. The C. V. Mosby Company, St. Louis Baltimore, Philadephia, Toronto.

Snedecor, G. W. and Cochran, W. G. (1967). Statistical methods. **6th Edn.** Oxford and IBH, Publishing co. Kolkata, Bombay and New Delhi.

Sobhanan, T. A. and Aleyas, N. M. (1986). Studies on convulsive seizures in goats in Kerala. In Vth Annual Convention of ISVM and National symposium on rencent advance in animal disease management and strategy for improved health care by 2000 A. D. 9th October. 1986. *Trichur, Kerala, Abstract No.* **71**.

Srivastava, A. K.; Patil, V. K. and More, B. K. (1985). Incidence of various disorders in local Angora and cross-bred goats. *Indian Vet. J.* **62** : 935 – 939.

Stern, J.S.; Bali, C.A. and Mayer. J. (1970). Are propipnete and butyrate physiological regulators of plasma insulin in ruminants 7 *Am.J. Physiol* **219**: 84.

Strafuss, A. C. and Monlux, W. S. (1966). A central nervous system reaction to disturbances in ruminant digestion. *Cornell Vet.* **56** : 128 – 141.

Svendsen, C. (1979) *Nordisk Vet. Med.* 31, **12**: 497.

Svendsen, P. (1973). The effect of volatile fatty acids and lactic acid on rumen motility in sheep. *Nordisk Vet. Med.* **25**: 226-231.

Tanwar, R.K. and Mathur, P.D. (1983a). Studied on experimental rumen acidosis in goats. *Indian Vet. J.* **60**: 499-500.

Tanwar, R. K. and Mathur, P. D. (1983b). Studies on clinical menifestations and therapy of experimentally induced acid indigestion in goats. *Indian J. Vet. Med.* **3** (1) : 15 – 20.

Tanwar, R. K. and Mathur, P. D. (1983c). Biochemical and microbial changes in experimentally induced rumen acidosis in goats. *Indian J. Anim. Sci.* **53** (3) : 271 – 274.

Tanwar, R. K.; Mathur, P. D.; Sharma. S. N. and Gahlot, A. K. (1983). Haematological and biochemical studies of blood in experimentally induced rumen acidosis in goats. *Indian J. Anim. Sci.* **53** (12) : 1296 – 1299.

Tanwar, R.K. and Mathur, P.D. (1985). Studies on experimentally induced acid indigestion in goats. In : All India Conference on current concept of various disorders of ruminant stomach. 8th – 9th Junary. 1985- Tamilnadu Agricultural University, Madras.

Teli, A. A.; Chauhan, H. V. S.; Gupta, B. S. and Srivastava, J. P. (1986). Acidosis in ewes caused by feeding of damaged apple (*Malus Sylvestris*) diet. *Indian Vet. J.* **63** (7) : 591 – 593.

Telle, P.P. and Preston, R. L. (1971). Ovine lactic acidosis : Intraruminal and systemic *J. Anim. Sci.* **33** (3) : 698 – 705.

Thomas, J.W. and Moore, L.A. (1951). Variations in heart rate in dairy cow. *J. Dairy Sci.* **34**: 321-327.

Tremere, A.W.; Mernill, W.G. and Loosli, J.K. (1968). Adaptation to high concentrate feeding as related to acidosis and digestive disturbance in early heifers. *J. Dairy Sci.* **51** (7): 1065-1072.

Tripathy, S.B. and Mishra, D.N. (1992). Therapeutic efficacy of Rumbion in experimental acid indigestion in goats. *Pashudhan* **7** (4): 4,8.

Tucker, J.O.; Glenn, M.W. and Robertsted (1956). Effect of dried preserved rumen inoculum on rumen micro-organisms of lambs. *Am. J. Vet. Res.* **17**: 498.

Upadhyay, A.K. and Shukla, S.K. (1999). Studies on efficacy of Anigest in various ruminal disorders. *Indian Vet. Med. J.* **23** (9): 249-251.

Vihan, V.S.(1978). Studies on changes in blood, serum in experimental rumen acidosis in buffalo. *Indian Vet. J.* **55**: 377-379.

Vihan, V. S.; Joshi, B. P. and Rai, P. (1973). Observations on changes in pH and lactic acid in rumen fluid and blood in bovine indigestion. *Indian Vet. J.* **59** (12) : 1178 – 1181.

Vihan, V. S. and Rai, P. (1985). Studies on change in blood serum and rumen liquor in rumen disorders of sheep and goats. *Indian. Vet. Med. J.* **9** (3) : 16 – 20.

Vihan, V. S.; Wani, G. M. and Sahni. K. L. (1982). Observations on changes in blood serum in experimental rumen acidosis in goats. *Indian Vet. J.* **59** (12) : 998 – 1000.

Verma, D. N.; Pant, H. C.; Rai, G. S. and Rawat, J. S. (1975). Concentration of V. F. A. and lactic acid in the rumen of goat. *Indian Vet. J.* **52** : 442 – 444.

Vestweber, J. G. E.; Leipold, H. W. and Smith, J. E. (1974). Ovine ruminal acidosis ; Clinical studies. *Am. J. Vet. Res.* **35** : 1587 – 1590.

Walker, D. J. (1968). The position of lactic acid and its derivatives in the nutrition and metabolism of ruminants. *Nutr. Abstr. And Rev.* **38** : 1-11.

Westcott, R. W.; More, G. R.; Conner, G. H. and Riley, W. T. (1953). Synthetic media powder to enhances rumen microfloral growth and activity. *Preliminary report. Vet. Med.* **48** : 349 – 350.

Williams, V. J. and Mackenzie, D. D. S. (1965). The absorption of lactic acid from the reticulorumen of sheep, *Australian J. Biol. Sci.* **18** : 917 – 934.

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