STUDIES ON
PATHO-BIOCHEMICAL CHANGES
IN EXPERIMENTAL RUMEN ACIDOSIS IN GOATS
WITH SPECIAL REFERENCE TO ENDOCRINOPATHY
AND THERAPY

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DEBANANDA BASAK
B. V. Sc. & A.H.

DEPARTMENT OF VETERINARY MEDICINE AND PUBLIC HEALTH
FACULTY OF VETERINARY AND ANIMAL SCIENCES
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In reverent Dedication to

My Parents & Family Members for

their Love, Inspiration & Blessings

Dr. Amalendu Chakrabarti
B.V.Sc. & A.H. (Cal),
M.V.Sc. (Medicine), Ph.D.(Cal)
Reader, Deptt. of Vety. Medicine & Public Health
Faculty of Veterinary & Animal Sciences
Bidhan Chandra Krishi Viswavidyalaya
P.O. Krishi Viswavidyalaya - 741 252, Nadia.

C E R T I F I C A T E

This is to certify that Shri Debananda Basak, B.V.Sc. & A.H. (B.C.K.V.) has worked in the Department of Veterinary Medicine and Public Health of the Faculty of Veterinary and Animal Science under Bidhan Chandra Krishi Viswavidyalaya. The work relating to his thesis entitled "STUDIES ON PATHOBIOCHEMICAL CHANGES IN EXPERIMENTAL RUMEN ACIDOSIS IN GOATS WITH SPECIAL REFERENCE TO ENDOCRINOPATHY AND THERAPY" was carried out independently by him, under my direct guidance and supervision. It gave me much pleasure and satisfaction to watch the methodical approach to his subject. The results of investigation reported in the thesis have not so far been submitted for any other Degree or Diploma. The assistance and help received during the course of investigation have been duly acknowledged.

Dated :
The 3rd...August 1991.

(Dr. Amalendu Chakrabarti)
Signature of the Advisor.

APPROVAL OF EXAMINERS FOR THE AWARD OF
DEGREE OF MASTER OF VETERINARY SCIENCE

We, the undersigned, having been satisfied with the performance of Shri Debananda Basak in the Viva-Voce Examination conducted to-day, the ..... 19th August ..... 1991 recommend that the thesis be accepted for the award of the Degree.

Name	Signature
1. Dr. A. Chakrabarti Advisor (Chairman).	.....
2. External Examiner.	..... 19/8/91
3. Dr. S. Sarkar, Member, Advisory Committee.	..... 19/8/91
4. Dr. B.N. Mukherjee, Member, Advisory Committee.	..... 19.8.91
5. Dr. S. Pan, Member, Advisory Committee.	..... 19/8/91
6. Dr. R. K. Ghosh, Member, Advisory Committee.	..... 19/8/91

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1.0 INTRODUCTION

Goat is the most important ruminant which provides valuable materials like meat, skin, hides, caesins, pashmina etc. Goat contributes to about 35% of the total meat and 3% of total milk produced in India (Chawla et al., 1981). Export of goats' hair, skin, caesins also earns valuable foreign exchanges (Acharya, 1982). In West Bengal Black Bengal goat is mostly reared for meat production.

India ranks first in goat population in the world (Sahani, 1982). The density of goat population is highest in West Bengal followed by Uttar Pradesh, Kerala and Madras.

In Indian condition goat rearing is most popular within small, marginal and landless farmers.

Goats do not require any extra housing system, can be reared in free range system, do not require extra amount of concentrate feeding if they are allowed to graze. Calving interval is minimum, give birth of 3 - 4 kids at a time and labour cost is also negligible in goats.

Goat utilizes grass, forages and leaves better than cattle and buffaloes and can maintain normal digestive physiology with forages. However, many a time farmers use to give more feeds to them in order to achieve increased body weight and milk production. So, there is every possibility of digestive upset leading to indigestion and occasional death. Such incidence frequently occurs in stall-fed goats under intensive management system which are gaining popularity due to non-availability of adequate grazing pasture.

In ruminants, optimum rumen motility, concentration of living microorganisms, 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 incidental changes in the feed, fodder and management lead to alteration of physico-chemical conditions of the rumen resulting to rumen dysfunction. This rumen dysfunction is characterised by loss of appetite, atony of the rumen and other abnormalities. In per-acute cases there is always possibility of fatality of the animals. In recent years it has become a routine practice to provide more concentrates to ruminants in order to obtain maximum production. This type of feeding causes indigestion called "Ruminal acidosis, Lactic acidosis, Toxic indigestion, Acute Carbohydrate engorgement, Acute impaction, Over eating disease, Grain Overload, Founder etc. (Chakrabarti, 1988).

Like cattle, goats also suffer from fatal ruminal acidosis due to accidental ingestion of large quantities of carbohydrate rich diet like rice, wheat, barley, oat, rye etc. Highly fermentable carbohydrate rich diets alter the rumen environment and help in rapid multiplication of gram +ve bacteria like Streptococcus bovis, Lactobacillus which lead to production of large quantities of lactic acid in rumen and thus decrease the rumen pH and lead to condition known as acidosis (Ahrens, 1967; Dunlop, 1972; Blood et al., 1983).

Incidence of this condition in goats has frequently been reported from the field during paddy harvest seasons (Gnanaprakasam,

1970; Sen, 1982; Tanwar and Mathur, 1983 a, b; Lal et al., 1989; Das, 1990). But, no comprehensive studies have been made on the pathobiochemical changes in experimental rumen acidosis in goat with special reference to hormones. There is limited research work on the impact of ruminal acidosis on hormones specially on Thyroxin (T_4), Cortisol and Insulin level. The main object of this study is to know whether lactic acidosis has any role to alter the function of thyroid gland, adrenal gland and pancreatic endocrine system.

Alteration of level of hormones may decrease or increase the metabolic processes of the body and ultimately death of the animal. Liver dysfunction has also been recorded in rumen acidosis in goat (Das, 1990).

Horino et al. (1968) showed that glucose, propionate and butyrate stimulated insulin secretion in cows. Plasma insulin level is also increased in cattle fed with high grain. High grain fed sheep had significantly higher concentrations of insulin at 24 and 72 hours than the hay fed sheep (Trenkle, 1970). Randhawa (1979) in his study on experimental ruminal acidosis in buffalo calves observed that there was increased circulating levels of plasma insulin at 48 hours.

Mills and Jenny (1979) observed more glucocorticoids value in concentrate fed dairy heifers than those of control group.

Stress due to digestive disorders lead to release of more adrenocorticoids as anti stressors of the body (Salye, 1958).

Available literature shows no relevant information for thyroxin and thyrotrophin (TSH) hormones in relation to rumen acidosis in goats.

A rise in blood glucose level has also been recorded by Nauriyal (1975) after experimental engorgement of buffalo and cow calves with crushed wheat.

Sen (1982) and Das (1990) have also reported high blood glucose level in experimentally induced rumen acidosis in goats. This increase could be due to an increase in the glycogenolysis or gluconeogenesis or due to decreased utilization of glucose by peripheral tissues (Dirksen, 1970). It may also be due to low levels of circulating IRI as suggested by Ivanov (1974). The present study has been undertaken to measure the level of hormones such as thyroxin, cortisol and insulin etc. in the serum and histopathological examination of the organs like thyroid, pancreas and adrenal glands and also other organs to know the structural and functional changes if any, due to rumen acidosis.

Keeping in view of the above facts, the present study has been framed on the following aspects to induce experimental rumen acidosis in goats by intra-ruminal administration of crushed rice grains and make observations on the following aspects.

i) Clinical accounts

ii) Changes in rumen liquor

a) Physical changes - Colour, consistency, smell, sediment activity time (SAT), Cellulose digestion

time (CDT), methylene blue reduction time (MBRT), motility and iodophilic nature of rumen protozoa.

b) Biochemical changes - pH, total volatile fatty acid (TVFA), ammonia nitrogen (NH_3-N) and lactic acid concentration.

c) Bacterial and protozoal count of rumen liquor,

iii) Changes in blood/serum-blood pH, blood lactic acid, blood glucose, total protein, albumin, globulin, albumin : globulin ratio, calcium, phosphorus, blood urea and creatinine.

iv) Haematological changes - Haemoglobin percentage, packed cell volume (PCV) percentage, total W.B.C. and R.B.C. count and differential leukocytic cell count.

v) Hormone level in serum :

a) Thyroxin

b) Cortisol

c) Insulin

vi) Macroscopical and microscopical changes in brain, pituitary, thyroid, pancreas, adrenal, liver, kidney etc.

vii) To evolve a suitable therapy against rumen acidosis in goats.

2.0 REVIEW OF LITERATURE

2.1 Experimental Rumen Acidosis

Scarisbrick (1954) induced acid indigestion in sheep of about 150lb 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.

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.

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

Krogh (1959) induced rumen acidosis in sheep by feeding of cane sugar in excess to hay diet.

Juha'sz and Szegedi (1968b) induced rumen lactic acidosis in Hungarian Merino sheep weighing 20 - 45 kg and over 6 months of age by feeding 50 - 90 g/kg ground wheat and barley and 16.5 g/kg glucose and by intra-ruminal infusion of 6 ml/kg of 90% lactic acid solution through fistula.

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

Huber (1971) produced acute acid indigestion in sheep by introducing glucose (20 g/kg body weight) suspended in 1 litre of water through cannula into the rumen.

Irwin et al. (1979) induced ruminal acidosis in four mature ewes by intra-ruminal administration of glucose.

Chaplin and Jones (1973) induced rumen acidosis in sheep by administration of ground barley at the dose rate of 40 g/kg body weight mixed with water in the proportion of 1 : 2 through rumen fistula after 24 hours of fasting.

Hartig and Hebold (1973 a) induced experimental ruminal acidosis in sheep by giving repeated doses of sugar.

Chronic acidosis was produced in sheep by increasing dosage of sucrose by 100 g per week for 7 weeks, sub-acute acidosis by 100 g per day for 13 days and acute acidosis by single dose of 15 or 20 g/kg body weight (Cakala et al., 1974).

Flachowsky et al. (1974) produced ruminal acidosis in lambs following the infusion of a mixture of starch, glucose and casein into the rumen.

Mild rumen acidosis was produced in sheep by intra-ruminal administration of glucose at the dose rate of 6.75 g/kg body weight after 15 hours of fasting while administration of 10.4 g/kg weight had a pronounced effect (Shinosaki and Nakabayashi, 1974).

Vestweber and Leipold (1974) and Vestweber et al. (1974) produced acute rumen acidosis in sheep by administration of corn sugar @ 100 to 1000 g/day through rumen fistula or by feeding.

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

Dougherty et al. (1975 a,b) produced rumen acidosis in sheep by excess feeding with grain (75% ground corn + 25% ground oats) at the dose rate of 70 g/kg body weight.

Koer et al. (1976) over fed the sheep with 3.178 kg of the 92% finely ground concentrate corn diet to produce acute rumen acidosis.

Nokata et al. (1977) induced rumen acidosis in three sheep by giving successively several different rations having concentrate to roughage ratio of 4 : 6, 5 : 5, 6 : 4, 7 : 3, 8 : 2 or 9 : 1 each for two days and finally 10 : 0 for five days through fitted rumen fistulas.

Terashima et al. (1978) produced lactic acidosis in hay fed sheep by injection of lactic acid intravenously or into the rumen.

Kezar and Church (1979 a) produced rumen acidosis in sheep by administering sucrose (15 g/kg body weight in 700 ml of water) through rumen fistula.

Kezar and Church (1979 b) induced rumen acidosis in sheep when it was fed with wheat at the dose rate of 50 g/75 kg body weight.

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

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

Tanwar and Mathur (1983 a,b) and Tanwar et al. (1983) were able to induced rumen acidosis in three groups of goat by rapid administering whole wheat grain at the dose rates of 80, 100 and 120 g/kg body weight respectively through rumen cannula.

Teli et al. (1986) produced rumen acidosis in sheep by feeding of damaged apple (Malus sylvestris) diet.

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

Kuusksalu (1988) produced rumen acidosis in sheep by introducing glucose at the dose rate of 20 g/kg body weight into the rumen.

Lal et al. (1989) experimentally induced ruminal acidosis in six goats of either sex, aged one to two years by intraruminal administration of whole wheat grain at the dose rate of 100 g/kg body weight following fasting for 24 hours.

Randhawa et al. (1989) produced sub-acute and acute lactic acidosis in calves by oral feeding of molasses given at

the rate of 10 g/kg body weight and 15 g/kg body weight after an over night fasting.

2.2 Clinical Account and Observations

Scarisbrick (1954) observed in acid indigestion in a sheep fed on mangolds that sheep lay down, did not rise untill strongly urged and then simply stood with a dejection appearance, occasionally grinding of teeth and showed no interest to take mangolds. When turned out they walked painfully and proved to be lame in all four feet, which were hot to touch and were rested at intervals. During the whole period they did not drink water.

Juha'sz and Szegedi (1968 b) in experimental ruminal acidosis of Hungarian Merino sheep found that local acidosis led to a systemic acidosis after absorption of lactic acid from the fore stomach, resulting in decreased ruminal movements, increased heart and respiratory rates, in some cases culminating in respiratory failure and death.

Gnanaprakasam (1970) reported that goats manifested clinical symptoms within 24 hours of accidental grain overload and the symptoms were classified into 3 catagories as mild, moderate and severe. Mild cases were characterized by anophagia and enlargement of abdomen. Rumen was firm and doughy on palpation and on percussion dull sound was heard. Ruminal motility was absent or sluggish. There was constipation. Moderate cases were characterized by anorexia with either constipation or diarrhoea, simple dyspnoea tachycardia and oliguria.

Ruminal motility was absent. On auscultation of rumen, gurgling sounds were heard. In severe cases animals were dull and unable to stand for long time. Skin was hard and lethery. Costal type of respiration was prominent. Heart rate increased to the range of 120 to 160 per minute. Wobbling gait, grinding of teeth and yawning were often noticed.

Mella-Lizama (1973) reported that in molasses feeding, a condition referred to as 'borrachera' (drunkenness) or molasses toxicity has been encountered with symptoms of depression, incordination, blindness of cortical origin and later on convulsions, opisthotonus and death.

Kelly (1974) reported that normal rectal temperature, pulse rate and respiration rate of goats are 102.9°F, 70 - 90/minute and 20 - 30/minute respectively. The healthy cattle and goat, are alert, active and have normal posture and gait, glossy skin coat, pink to rosy conjunctival mucous membrane and good appetite. Normal cattle and goats ruminate during resting period both in standing and lying down position. The frequency of unination is 1 - 3 times per day.

Misra and Singh (1974) observed that five cattle out of 75 suffering from primary indigestion showed symptoms of acid indigestion having the rumen pH varying from 4 to 4.5. The animals had the history of over eating with paddy. Rise in respiration and pulse rate, absence of rumen contractions, staggering gait, restlessness were the common signs. They also reported that pH of the urine decreased to 4.2 - 4.5 (AV. 4.4) in acid indigestion in cattle.

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

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

Terashima et al. (1978) reported decreased pH of urine in intravenous injection of lactic acid or intra-ruminal infusion of lactic acid in hay-fed sheep.

Sethuraman and Rathor (1979) observed that there was a direct correlation between the pH of rumen fluid and urine which could be used as a diagnostic tool in acute acid indigestion in cattle and buffaloes. The mean pH of the urine gradually declined from 8.0 $\pm$ 0.2 at 0 hour to 5.8 $\pm$ 0.2 at 96 hours of in experimental rumen acidosis.

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

Sen (1982) observed symptoms of experimentally rumen acidosis in goats as rise in body temperature, rapid pulse, heart and respiration rate, complete ruminal atony, pasty faeces and diarrhoea, fullness of rumen with water, inappetance, dullness, occasional grinding of teeth, dyspnoea, dehydration, nasal discharge and death.

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

Kutus et al. (1983) reported that mean pH of the faeces of 45 healthy cattle was 6.66 ± 0.03. In cattle with acidosis produced by carbohydrate feeding pH was between 4.36 and 5.85.

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

Li et al. (1984) observed fall of pH of the urine in experimental maize acidosis in the rumen of Xinjang fine wool sheep.

Kovac et al. (1986) reported that measurement of faecal pH with indicator paper was a rapid method of diagnosis of acute metabolic disorder due to excessive intake of readily digestible carbohydrate or urea.

Cao et al. (1987) observed the signs of weakness, preferring to lie in sternal recumbency from 12 to 48 hours after experimentally induced lactic acidosis in goats. Both mean heart

rate and respiratory rate gradually rose from 98 and 12 per minute respectively at '0' time to 174 and 17 per minute at 24 hours. Rectal temperature remained at 38 to 39^{o}C throughout the experiment. Soft but not voluminous faeces were being passed by all goats by 12 hours and each had abdominal distension.

Chakrabarti (1988) reported that healthy cattle and goat have bright eyes, alert and responding ears, glossy, skin with hairs lying evenly over the body coat. The muzzle look moist with dew drop like appearance over it. They have normal posture and gait. The normal goats have pelleted faeces with dark green colour. The normal colour of the urine of goat is clear straw 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^{o}F, 60 - 70/minute, 18 - 30/minute respectively.

Lal *et al*. (1989) observed symptoms of experimental rumen acidosis in goats which suffered from various degree of anorexia and ruminal stasis following induction of acidosis, whereas 33 - 50 % of animals manifested other clinical symptoms like dullness, diarrhoea, constipation, abdominal pain, nasal discharge, pressing of head and grinding of teeth etc. There was significant but gradual fall of body temperature associated with marked increase in thepalse and respiration rate and significant decrease in ruminal movement.

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

2.3 Observations on Rumen Liquor

2.3.1 Macroscopic

2.3.1.1 Smell

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

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

Sen (1982) observed that the odour of rumen liquor changed to sour at 20th hour of grain overload in goat.

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

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

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

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

2.2.1.2 Consistency

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

Misra and Singh (1974) reported that the consistency of rumen liquor in healthy cattle was thicky which was changed to watery in acid indigestion.

Rosenberger (1979) observed that consistency of rumen liquor changed from slight viscous to watery in rumen acidosis in cattle.

Sen (1982) reported that the consistency of rumen liquor changed from viscous to watery liquid at 20th hour onwards in experimental acid indigestion of goat.

Pradhan et al. (1988) observed that the consistency of rumen liquor in healthy Black Bengal goats was semi liquid.

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

Randhawa et al. (1989) observed that in lactic acidosis of buffalo calves the viscosity of the normal rumen liquor changed to watery by 12 and 6 hours in subacute and acute conditions respectively.

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

2.3.1.3 Colour

In cases of rumen acidosis of goat the rumen fluid became dark green or grey in colour and had no gas bubbles (Gnanaprakasam, 1970).

Misra and Singh (1974) observed that the colour of rumen liquor of healthy cattle was yellowish brown. It became yellowish in acid indigestion.

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

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

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

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

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

Randhawa et al. (1989) observed that in lactic acidosis of buffalo calves the colour of rumen liquor changed into brownish yellow to greyish-yellow between 6th and 12th hour but became normal after 12 hours of the induction of sub acute lactic acidosis. However, in acute lactic acidosis, the colour of rumen liquor changed to greyish-yellow at 60 hours which persisted throughout the experimental period.

2.3.1.4 Sediment activity time (SAT), Cellulose digestion time (CDT) and Methylene Blue reduction time (MBRT)

Hoflund et al. (1948) did the CDT in rumen liquor to know the activeness of rumen microbes. A fully active rumen liquor

would digest the cellulose within 48 to 54 hours. In case of indigestion rumen liquor could not digest cellulose at all or took more time to digest.

Nichols and Penn (1958) reported that SAT was dependent with ration and time after feeding. There were 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) observed that there was no sedimentation or floation activity of particles of rumen liquor in acid indigestion of goat.

Rosenberger (1979) reported prolonged methylene blue reduction time of rumen liquor in clinical rumen acidosis in cattle.

Sen (1982) observed that the normal SAT of rumen liquor of healthy goat was 26.60 $\pm$ 5.86 minutes but there was complete absence of sediment activity after 4th hour onwards of grain engorgement.

Blood *et al.* (1983) reported that SAT in normal animals varied between 3 minutes if the animal has just been fed and 9 minutes if the last feeding has occurred sometime previously. Settling of the particulate materials indicate gross inactivity, less severe degrees being manifested by prolongation of time required for flotation. Cellulose digestion times in excess of 30 hours 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) recorded that SAT in rumen liquor increased significantly at 6 hours and no sedimentation activity was detected at 12 and 24 hours, which returned to normal by 168 hours of sub acute induction of lactic acidosis in buffalo calves. In acute lactic acidosis, complete absence of SAT was observed from 6 hours which persisted throughout the period of observation. No cellulytic activity was detected in the rumen liquor in calves of sub acute lactic acidosis between 6 and 144 hours. However, at 168 hours CDT values were higher than 48 hours values. In acute lactic acidosis CDT values were nill till the death of the buffalo calves.

Das (1990) reported that mean SAT and CDT of rumen liquor were 25.22 $\pm$ 1.95 minutes and 52.14 $\pm$ 3.07 hours respectively at '0' hour which were completely absent at 12th hour onwards in experimental rumen acidosis in goat.

2.3.2.1 Motility, Concentration, Count and Iodophilic nature of rumen protozoa.

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

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

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

Chou and Walker (1964) reported that diet had a considerable effect on the protozoal concentrations in sheep. Starch feeding caused unfavourable conditions resulting to reduction of protozoa.

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

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

Slyter et al. (1970) observed that cattle fed 90% corn, wheat, barley or milo all - concentrate diet (restricted to 1.5% of the Steer's body weight) contained 31, 10, 36 and 27 x 10^3 protozoa per gram of ruminal ingesta, respectively.

Dash et al. (1972) reported that in acute indigestion in cattle all protozoa were non-motile as there was no living

protozoa found from rumen fluid having ruminal pH below 5.5.

Misra et al. (1972) observed that concentration, motility and iodophilic nature of protozoa in rumen liquor of healthy cattle was ++/+++ (moderate to high) respectively. In case of acute carbohydrate engorgement the concentration, motility and iodophilic nature of protozoa in rumen liquor changed to - (absent), - (absent) and - (absent) respectively.

Rai et al. (1972) found that total protozoa count (No. x 10^4/ml SRL) in rumen liquor of goat at '0' hour, 4 hours and 6 hourswere 31.86 ± 4.24, 32.60 ± 4.16, 18.59 ± 1.83 and 19.60 ± 2.16 respectively under maintenance feeding standard. Here the daily average protozoal concentration was 25.66 ± 1.75 x 10^4/ml of SRL. A steady population was observed indicating even fermentation rate and metabolites production.

Cakala et al. (1974) observed that concentration of rumen protozoa decreased in early phage of rumen acidosis in sheep.

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

Rai and Pandey (1980 b) reported that average number protozoa of healthy range control group, stall fed group and range group of goats were 99.99 ± 7. 239 x 10^4/ml, 67.61 ± 5.919 x 10^4/ml and 28.80 ± 1.257 x 10^4/ml of rumen liquor respectively, collected at 2 hours intervals upto 8th hour.

They also reported that lowest pH values as observed at 8 hours might be one of the implicating factors for the lowest total protozoal counts.

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

Tanwar and Mathur (1983 b) observed that the mean values of total protozoal count were 23.91 $\pm$ 2.53, 20.83 $\pm$ 4.28 and 26.91 $\pm$ 2.41 x 10^4/ml of rumen liquor at '0' hour in I (at the rate of 80 g/kg whole wheat grain), II (at rate of 100 g/kg) and III (at the rate of 120 g/kg) group of goats with acid indigestion respectively and then started to decline and reached the values of 14.16 $\pm$ 2.41 and 9.28 $\pm$ 1.96 x 10^4/ml of rumen liquor at 96 hours in the group I and II respectively. In group III no protozoa were observed when examined at 12 hours.

Li *et al.* (1984) reported that following the decline in rumen pH there was gradual reduction in the number of ciliates in rumen liquor in experimental maize acidosis in Xinjiang fine wool sheep.

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

Lal *et al.* (1989) observed that there was complete disappearance of ruminal protozoa from 12 to 72 hoursin experimental rumen acidosis in goats. However, the protozoa predomi-

nated by Entodinium Spp. (85.75%) appeared in the rumen liquor in much smaller number (1.42×10^5) at 96 hours. He found that at '0' hour the total protozoal count was $4.21 \pm 0.25 \times 10^5$/ml.

Randhawa et al. (1989) observed that in sub acute acidosis of buffalo calves the protozoal motility gradually became sluggish at 6 hours and diminished to nil by 12 hours. The motility of the reestablished rumen protozoa was sluggish at 120 hours and moderate at 144 hours. In acute acidosis complete absence of protozoal motility was observed at 6 hours. The average total rumen protozoal count decreased significantly by 6 hours of the induction of subacute and acute lactic acidosis. In acute lactic acidosis no protozoa could be detected at subsequent intervals, but in sub acute condition, reappearance of protozoa was observed by 120 hours and the total protozoal count was $0.59 \pm 0.04 \times 10^5$/ml of rumen liquor at 168 hours.

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

Narendra et al. (1990) reported that there was gradual reduction of number of protozoa in rumen fluid till 18 hours and were found absent afterwards in experimental ruminal acidosis in calves.

2.3.2.2 Bacterial count and Types of Bacteria

Hungate et al. (1952) observed that the predominating Gram negative flora in the rumen liquor was replaced by a predominating Gram positive flora in animals fed with grain ration.

Gall et al. (1953) recorded that there was predominance of Gram positive organisms in rumen liquor in grain-fed animals than the animals fed with roughage.

Krogh (1963 a,b) reported that in acute rumen acidosis there was decrease in rumen pH and the Gram positive flora in the rumen multiplied at the expense of the normal Gram negative flora or fauna.

Allison et al. (1964) observed that the ingestion of excessive amount of readily fermentable carbohydrates by ruminants resulted in acute indigestion. Further, the predominating Gram negative ruminal flora was replaced by Gram positive bacteria principally Streptococcus bovis and Lactobacilli which altered the intra-ruminal environment and affected the acid base balance of the animals.

Slyter et al. (1965) reported that in the rumen of healthy concentrate-fed cattle consuming diets at ad libitum intake, the number of ruminal bacteria was increased to about 10 fold that of forage-fed animals.

Hungate (1966) reported that dense population of bacteria (4 to 88 x 10^9/ml) exist in healthy ruminants fed natural forage.

Mann (1970) reported that Gram positive flora mainly Lactobacilli predominated with the reduction in ruminal pH.

Rai et al. (1972) found that total bacterial count (No. x 10^9/ml RL) in rumen liquor of goat at 0 hour, 2 hours, 4 hours and 6 hours were 12.46 ± 0.83, 13.04 ± 1.08, 10.42 ± 0.69 and 10.31 ± 0.66 respectively under maintenance feeding standard. Here the daily average bacterial concentration was 11.56 ± 0.428 x 10^9/ml RL.

Vazquez (1976) observed that there was proliferation of Gram positive bacteria in rumen lactic acidosis.

Rai and Pandey (1980 b) reported that average number bacteria of healthy range plus concentrate group, stall-fed group and range group of goats were 44.26 ± 1.943 x 10^9/ml, 23.43 ± 1.460 x 10^9/ml and 35.91 ± 1.383 x 10^9/ml of rumen liquor respectively collected at 2 hours intervals upto 8 hours.

Tanwar and Mathur (1983 b) observed that the total bacterial count increased after the administration of whole wheat grain and showed some variation in different intervals of sampling in experimentally induced rumen acidosis in goats.

Lal et al. (1989) reported that there was a significant decrease in the rumen bacterial population at 12 hours which started improving from 96 hours onwards in experimental rumen acidosis in goats.

Randhawa et al. (1989) observed that the average total

bacterial count in both sub acute and acute lactic acidosis in buffalo calves showed a significant decrease and the minimum counts were observed at 24 hours. Subsequently, the bacterial count showed an increasing trend but concentrations in both the groups were less than the '0' hour's count. At '0' hour Gram negative bacteria were predominant while Gram positive bacteria were predominant at 6 hoursin both the groups. In acute lactic acidosis, few Gram negative bacteria were found at subsequent intervals.

Narendra et al. (1990) reported an increase in mean bacterial count per ml of strained rumen liquor from 7.89 $\pm$ 0.14 x 10^9 at '0' hour to 11.90 $\pm$ 0.372 x 10^9 at 36 hoursin experimental ruminal acidosis in calves.

2.3.3 Biochemical changes of Rumen Liquor

2.3.3.1 pH of Rumen liquor

Olsen (1941) recorded an average pH value of rumen liquor of 6.86.

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

According to Allison et al. (1975) and Wilson et al. (1975) pH of the normal ruminal ingesta varied from 6.35 to 6.95 and 6.7 to 7.1 respectively.

Pradhan et al. (1988) reported that the pH of rumen liquor varied from 6.4 to 7.4 with an average of 6.81 $\pm$ 0.15 in

healthy Black Bengal goats.

Variation in acidosis cases :

Phillipson (1942) reported that sheep fed on a ration containing high flaked maize and less roughage (150 g daily) reduced the pH of rumen liquor to 4 to 5.

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.

Gnanaprakasam (1970) observed that in moderate and severe cases of rumen acidosis in goat the pH of the rumen liquor was 5.2 to 5.5 and below 5.2 respectively.

Reddy and Nair (1971) observed that the pH of rumen liquors at 2nd, 4th, 6th and 8th hour samples were 6.05 $\pm$ 0.04, 5.70 $\pm$ 0.04, 6.00 $\pm$ 0.04 and 6.35 $\pm$ 0.03 respectively in healthy Jamunapari - Malabari cross-bred female goats.

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

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

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

Dougherty et al. (1975 a,b) that the pH of rumen liquor was 4.2 in grain induced acidosis in sheep.

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

Beede and Farlin (1977) observed that the pH of rumen liquor decreased from 6.22 to 5.22 within 8 hours in experimental lactic acidosis in sheep.

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

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

Rai and Pandey (1980 a) reported that pH of rumen liquor in three group of goats maintained on range, 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 (1982) observed fall in mean pH of rumen liquor from 6.86 ± 0.17 to lowest mean value 4.37 ± 0.12 at 12 hours of grain overload in goats.

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

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

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

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

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

Lal et al. (1989) reported that a significant drop in the ruminal pH was observed at 12 hour (4.7 ± 0.045) with a further drop at 24 hour (4.54 ± 0.023) 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 hour and 6.79 ± 0.04 to 4.24 ± 0.06 at 24th hour of sub acute and acute rumen lactic acidosis in buffalo calves respectively.

2.3.3.2 Lactic acid

Scarisbrick (1954) reported that in sheep fed an excessive quantity (15 lb) of mangolds, the usual picture in the rumen became distorted in that lactic acid accumulated to an abnormal extent.

Juha'sz and Szegedi (1968 b) reported that there was excessive production of lactic acid in the rumen in acid indigestion of Hungarian Merino sheep.

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

Vihan et al. (1973 b) recorded high level of lactic acid in rumen liquor in acid indigestion of bovine.

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

Verma et al. (1975) estimated the mean lactic acid concentration of rumen liquor in Barbari bucks at '0' hour, 2nd hour, 4th hour and 6th hour of concentrate mixture feeding as

3.80 ± 0.40, 19.62 ± 1.10, 3.50 ± 0.30 and 3.90 ± 0.10 mg/ 100 ml respectively.

Koer et al. (1976) observed the peak rumen lactate at 4th hour and 24th hour, which again rose through 60 hours and retarded to normal after 99 hours of post engorged concentrate corn diet in lambs.

Beede and Farlin (1979) recorded high concentration lactic acid in rumen liquor at 3rd hour and 7th hour in experimental rumen acidosis in sheep. The mean concentration of lactate in rumen liquor was 1.3 mM/L.

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

Sen (1982) observed that the mean concentration of lactic acid in rumen liquor was 3.38 ± 0.92 mg/100 ml at '0' hour which increased after grain overload and maximum mean concentration of lactic acid was 521.59 ± mg/100 ml at 12th hour of grain overload in goats.

Tanwar and Mathur (1983 b) recorded that 748.21 ± 22.86 mg % at 72th hour was the peak concentration of lactic acid in rumen liquor in wheat grain induced acidotic goats.

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

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

Cao et al. (1987) observed that mean ruminal lactate concentration progressively increased from 0 mmol/L at 0 hour to 36 mmol ± 5.7/L at 24 hours in experimentally induced lactic acidosis in the goat.

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

2.3.3.3 Total Volatile Fatty Acid (TVFA)

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

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

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

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

Variation in acidosis cases :

Scarisbrick (1954) observed that the TVFA concentration in the rumen liquor increased in the initial stage and gradually declined to very low level with the progress of time in acid indigestion in a sheep fed on mangolds.

Ryan (1962) reported that depressed quantities of ruminal volatile fatty acids had been noted in overfed animals.

Prasad et al. (1972) observed that in acid indigestion of cattle and buffaloes the mean concentrations of TVFA were 149 ± 13.05 and 136.03 ± 16.5 respectively which were significantly higher.

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

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

Muir et al. (1980) reported that there was increase in concentration of TVFA in initial stage followed by gradual

decrease in experimental rumen acidosis in sheep.

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

Sen (1982) observed that the mean value of TVFA in rumen liquor changed from 50.0 ± 9.49 mEq/L at '0' hour to 30.0 ± 1.79 mEq/L at 4th hour of grain engorgement in goats. In subsequent hours the TVFA decreased further reaching a minimum of 7.00 ± 2.11 mEq/L by 32 hours of grain overload.

Tanwar and Mathur (1983 b) observed that the TVFA of rumen liquor in early stages of wheat grain induced rumen acidosis in goats increased due to fermentation of carbohydrates by rumen micro-organisms. Then it decreased due to change of microbial population in rumen owing to cow pH.

Lal *et al.* (1989) observed that the mean TVFA concentration in the rumen liquor increased significantly from 64.83 ± 2.891 to 148.33 ± 15.47 mEq/L at 12 hours which decreased to 44.00 ± 2.251 mEq/L at 24 hours and 31.66 ± 1.819 mEq/L at 48 hours of experimental rumen acidosis in goats.

Randhawa *et al.* (1989) observed that in the sub acute condition the TVFA level gradually increased during 6 to 96 hours

While in acute condition there was a sudden fall in TVFA values at 24 hours in lactic acidosis of buffalo calves.

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

2.3.3.4 Ammonia nitrogen (NH_3-N)

McDonald (1948) reported that the level of ammonia in the rumen, reflecting the difference between production from nitrogenous sources and uptake by micro-organisms for growth, rose slightly at first and then gradually fell away as the balance swang in favour of uptake by microorganisms in lactic acidosis.

Scarisbrick (1954) reported that on feeding of mangolds in a sheep there was increase in ammonia unusually than that of V.F.A., after attaining a maximum value very much lower than usual, fell away.

Pant *et al.* (1962) reported a higher level of TVFA, ammonia in Jamunapari goats than in sheep at maintenance level of feeding.

Chou and Walker (1964 b) reported that there was considerable variation between diets in their effect on the concentrations of the nitrogen fractions in sheep. The concentration of ammonia nitrogen (NH_3-N) of rumen liquor was reduced conside-

rably in rice, maize and potato feeding compared to the highest value on the lucern diet.

Rai et al. (1972) experimentally showed that ammonia nitrogen accounted for 20.41% of the total nitrogen in strained rumen liquor of healthy Barbari goats. The average daily mean and peak values were 31.64 ± 1.87 and 60.12 ± 2.25 mg/100 ml of rumen liquor respectively of those goats maintained on 300 g concentrates mixture (gram, groundnut cake, barley and bran 25 parts each) and on grazing. The changes in the concentration due to time of sampling were also significant. The peak was observed at 2 hours post concentrate feeding and the concentrations thereafter declined and by sixth hour they were lowest.

Sethuraman and Rathor (1979) reported that gradual reduction in the rumen ammonia nitrogen and its complete absence at pH below 4.0 indicated high acidity in lactic acidosis in cross bred and Murrah calves.

Rai and Pandey (1980 b) reported that mean concentrations of ammonia nitrogen in rumen liquor were 22.50 ± 0.605, 18.52 ± 1.628 and 12.98 ± 0.563 mg/100 ml in range plus concentrate group, stall-fed group and range group of adult goats respectively.

Teli et al. (1986) observed increase in level of ammonia nitrogen of rumen liquor in acid indigestion of ewes due to feeding of damaged apple meal, urea and molasses.

Randhawa et al. (1989) observed that in sub acute lactic acidosis in buffalo calves there was a gradual increase in NH_3-N concentration between 6 and 48 hours which was followed by a subsequent decrease at 72 and 96 hour. However, in acute condition the NH_3-N concentration was significantly low at 6 hours but thereafter the level gradually increased.

2.4 Observation in Blood Serum

2.4.1 Haematological Studies

Brown et al. (1959) reported that there was complete reversal of the normal lymphocyte neutrophil ratio, stab neutrophils segmented neutrophil ratio and increase in total leukocyte count in chronic bovine reticuloperitonitis.

Hjerpe (1961) studied acute traumatic indigestion in cattle and found three alterations in haemological picture which were (i) elevation of the total leukocyte count in excess of 13,000/cmm (ii) increase in the incidence of unsegmented neutrophils in the differential count in excess of the upper normal limit of 5% (iii) reversal of the normal lymphocyte : neutrophil ratio of 1 : 0 to 1.7.

Hjerpe (1963) recorded the haemograms of acute indigestion in 26 dairy cows. There was no leukocytosis, however in 50% of these 26 cows there was a shift to the left in the schilling indexes evidenced by increase in non-segmented neutrophils in the differential leukocyte count to 6% or greater or both.

Castello (1968) cited the leukogram in indigestion of cattle fed on 250 lb bread daily. There was significant neutrophilia, evidently due to the increased heart rate (physiological leukocytosis) and the effect of stress. There was a variable degree of shift to the left including metamyelocytes in 50% of the cattle, apparently due to stress. Lymphopenia and eosinopenia anticipated in the first 24 to 48 hours of acute condition.

Telle and Preston (1971) observed that the haematocrit value and total erythrocytic count rose upto 4 hours of induction of lactic acidosis in sheep.

Dash et al. (1972) observed that there was increase in haemoglobin levels with simulteneous rise in total R.B.C. count and PCV in acute indigestion of cattle. Leukocytosis, neutrophilia and lymphopenia were also marked. In the differential counts, the immature neutrophils specially the metamyelocytes, was noticed along with leukocytosis, neutrophilia and shift to the left. The rise in the metamyelocyte number might be due to the stress factors in the blood forming organs during acute indigestion.

Misra and Singh (1974) reported that haemoglobin value of cattle having indigestion was found to be similar to that of healthy cattle. There was slight increase of R.B.C. and W.B.C. count in cattle having indigestion than that of healthy cattle. The packed cell volume (PCV), lymphocyte percentage and eosinophil percentage were slightly less in cattle affected with indigestion.

Shinosaki and Nakabayashi (1974) reported that haematocrit values gradually increased from 31.3% to 38% in glucose induced acidosis in sheep.

Dougherty *et al.* (1975 b) observed increased in haematocrit value in sheep with grain overload.

Randhawa *et al.* (1981 a) reported that there were increase in blood haemoglobin and PCV values in peracute lactic acidosis in calves.

Sen (1982) observed that mean PCV changed from 24.17 ± 0.75% at '0' hour to 37.67 ± 1.86% at 24th hour of grain overload in goats and then the value decreased gradually.

Sastry (1983) reported that values of PCV, Hb, total R.B.C. count, total W.B.C. count, neutrophil, lymphocyte, eosinophil, monocyte and basophil count was in between 19 - 28%, 8 - 14 g%, 8 - 18 x 10^6/cmm, 4 - 13 x 10^3/cmm, 10 - 59%, 40 - 75%, 0 - 10%, 0 - 6% and 0 - 2% respectively in normal goat.

Tanwar and Mathur (1983 a) recorded that the mean haematocrit value changed from 27.60 ± 0.74%, 24.40 ± 0.07% and 26.60 ± 1.07% to 28.40 ± 0.59%, 25 ± 0.70% and 37.80 ± 2.86% at 36th hour, 12th hour and 72th hour of wheat grain induced acidosis in three groups of goat at the dose rate of 80, 100 and 120 g/kg body weight respectively.

Tanwar *et al.* (1983) observed increase in total erythrocyte count, haemoglobin level and packed cell volume

for the first 12 hours in experimentally induced rumen acidosis in goats.

HejLasz et al. (1984) observed high haematocrit value and erythrocytosis and reduced erythrocyte volume in experimental acidosis in cows.

Schalm et al. (1986) reported that percentages of neutrophil, lymphocyte, monocytes, eosinophil and basophil in normal goat were 30 - 48%, 50 - 70%, 0 - 4%, 1 - 8% and 0 - 1% respectively. Neutrophil (band) was rare. The percentage of PCV was between 24 - 48% with an average of 35%. They also reported that stress induced by acute indigestion in cattle was found to result in a left shift without accompanying leukopenia. Leukocytosis occurred primarily due to neutrophilia and persistence of lymphocytes. There was variable degree of left shift including metamyelocytes in half of the cases.

Cao et al. (1987) observed that total white cell count significantly (P/0.05) rose from a mean value of 15.8 x 10^9/L at Zero time to 36.5 x 10^9/L at 24 hoursand the white cells differential indicated a stress response. The neutrophils increased being more than 3-fold with a 50% decrease in the lymphocyte count in experimentally induced acid indigestion in goat.

Chakrabarti (1988) reported that in normal goats, the mean neutrophil, eosinophil, lymphocyte, monocyte, basophil and PCV percentages are 40.00, 2.5, 48.00, 2.00, 1.60 and 34.00 respectively. The total R.B.C. and W.B.C count are 13. 90 million/cmm and 5.14 thousand/cmm.

Kuusksalu (1988) observed the increased of haematocrit values in glucose induced acidosis in sheep.

Mohi et al. (1990) recorded the blood picture of 16 healthy non pregnant female shami goats with a particular reference to seasonal variation. Significantly low values of TEC ($13.37 \pm 0.24 \times 10^6$/cmm), PCV ($30.38 \pm 0.85\%$) were recorded in summer than winter. On the contrary higher values of Hb (10.93 ± 0.43 g/dl) was recorded during summer than winter. Total leukocytes remained unaffected during different seasons. The total leukocytic count range from 9.03 ± 0.35 to $9.80 \pm 0.35 \times 10^3$/cmm.

Narendra et al. (1990) reported increase in haemoglobin content and PCV value from '0' hour to 36 hours of post feeding in experimental ruminal acidosis in calves, indicating development of dehydration due to acidosis.

2.4.2 Biochemical changes of Blood/Serum

2.4.2.1 Blood pH

Dirksen (1965, 1970) recorded tne pH of blood below 7.0 during first phase of acute rumen acidosis in cattle.

Juhasz and Szegedi (1968 a) recorded in blood pH decrease from 7.52 to 7.08 in acid indigestion of sheep fed with ground wheat, barley and glucose.

Juha'sz and Szegedi (1968 b) suggested that a drop in pH of blood to 7.3 signified approach of death in sheep in acid indigestion.

Telle and Preston (1971) recorded that there was inverse relationship between blood pH and blood lactate in experimental rumen acidosis in sheep.

Hartig and Hebold (1973 a) induced experimental ruminal acidosis in sheep by giving repeated doses of sugar and noticed a drop in blood pH from 7.44 to 7.18.

Shinosaki and Nakabayashi (1974) recorded fall in the plasma pH to 7.3 in glucose induced rumen acidosis in sheep.

Vestweber et al. (1974) recorded the mean pH of blood as 7.44 ± 0.06 when rumen pH was 6 or above and 7.36 ± 0.12 when rumen pH was 5 or less in corn sugar induced acidosis in sheep.

Nokata et al. (1977) experimentally induced ruminal acidosis in sheep by abruptly increasing the concentrate to roughage ratio and recorded a drop in blood pH.

Sen (1982) recorded the mean blood pH 7.48 ± 0.03 at '0' hour which gradually decreased to a lowest value of 7.19 ± 0.05 at 32th hour of grain overload in goats.

Vihan et al. (1982) observed that the pH level of blood varied with the different types of carbohydrate induced acidosis in goats. He recorded the blood pH of 5.5 at 18 hours of barley induced acidosis, 4.5 at 42 hours of grain (50% barley

+ 50% wheat) induced acidosis and 4 at 12 hours of cane sugar induced acidosis in goats.

Tanwar et al. (1983) experimentally proved that the mean blood pH of 7.5 ± 0.02 at '0' hour markedly decreased to 7.07 ± 0.04 at 72 hours of wheat grain induced acidosis in goats.

Huber et al. (1984) observed that the mean pH of blood fell from 7.37 ± 0.02 to 7.17 ± 0.05 in glucose induced acidotic sheep.

Cao et al. (1987) observed that mean blood pH gradually fell from 7.44 ± 0.052 at 0 hour to 7.20 ± 0.177 by 24 hours and again rose to 7.48 ± 0.001 at 48 hours in experimentally induced lactic acidosis in the goat.

Kuusksalu (1988) reported that there was always fall of blood pH in glucose induced acidosis in sheep.

2.4.2.2 Blood glucose

Juha'sz and Szegedi (1968 a, 1968 c) observed increase in blood glucose level in acidotic sheep.

McIntosh et al. (1973) recorded a three time rise in blood glucose concentration in acidotic sheep.

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

Ivanov (1974) studied 19 cows with recurrent subacute

or chronic ruminal acidosis for 12 years and observed hyperglycaemia (114 ± 8 mg/100 ml) and glycosuria.

Shinosaki and Nakabayashi (1974) reported that there was transitory rise in blood glucose in glucose induced acidosis in sheep.

Vestweber et al. (1974) observed that in maize-sugar induced ruminal acidosis in sheep the mean concentration of glucose in blood increased to 78 ± 17.3 mg/100 ml.

Melvin (1977) mentioned that normal blood glucose concentration in goat as 45 - 60 mg/dl.

Bieniek (1981) observed that blood glucose level decreased initially but increased later on in experimental induced rumen acidosis in six heifers.

Sen (1982) observed the maximum mean value of 172.05 ± 94.11 mg/100 ml blood glucose by 24 hours of grain overload in goats.

Vihan et al. (1982) observed that blood sugar level variably increased with the different types of carbohydrate rich feed induced acidosis in goat. The blood sugar level increased from value of control group of 57.25 mg% to 76.5 mg% at 18 hours of barley induced acidosis, to 140.00 mg% at 42th hour of grain (50% barley + 50% wheat) induced acidosis and to 350.0 mg% at 12th hour of cane sugar induced acidosis in goats.

Sastry (1983) reported that blood sugar level of

normal goat ranged from 45 to 60 mg/100 ml.

Vihan and Rai (1985) recorded the mean concentration of glucose of 81.9 ± 8.57 mg% in clinical acidotic goats.

Das (1990) observed increase in level of blood glucose in experimental rumen acidosis in goats.

2.4.2.3 Blood lactic acid

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) reported that the absorption of lactate from the rumen was evident from the increased level of blood lactate in the rumen vien after $1\frac{1}{2}$ to 2 hours of intraruminal administration D - L lactic acid in sheep.

Shinosaki and Nakabayashi (1974) observed that the plasma lactic acid concentration increased to 4.1 mEq/L in glucose induced acidosis in sheep.

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

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

Verma et al. (1975) estimated the lactic acid concentrations in blood of Barbari bucks at '0' hour, 2nd hour, 4th hour and 6th hour 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.

Sen (1982) experimentally found that the mean lactic acid concentration in blood increased from 12.98 ± 2.90 mg/100 ml to 23.69 ± 3.27 mg/100 ml by 12 hours of induction of lactic acidosis in goat and it remained increased throughout the observation period.

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

Tanwar et al. (1983) recorded that lactic acid level in blood of goat elevated to 24.4 ± 2.73, 24.46 ± 1.50 and 27.30 ± 1.32 mg% in wheat grain induced acidosis at the dose rate of 80, 100 and 120 g/kg body weight respectively.

Huber et al. (1984) observed that in glucose induced acidosis in sheep blood lactate concentration rose from 3.50 ± 1.90 mg/100 ml to 78.2 ± 20.0 mg/100 ml.

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

Cao et al. (1987) observed 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 hours, then declined and again elevated to 2.79 ± 1.223 mmo/L at 48 hours of experimentally induced lactic acidosis in the goats.

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

2.4.2.4 Serum Calcium and Phosphorus

Moodie (1960) found hypocalcaemia in a few cases of simple and acid indegestion in cattle.

Juha'sz and Szegedi (1968 b) observed that in rumen overload in sheep fed with wheat and barley meal, the plasma inorganic phosphorus concentration increased accompanied by some increase in Ca^{++} concentration in 24 hours period.

Kaneko and Cornelius (1970) mentioned that normal mean concentration of serum calcium and phosphorus in goat were 10.3 ± 0.7 mg/dl and 6.8 - 8.4 mg/dl respectively.

Telle and Preston (1971) reported that concentration of calcium in blood remained relatively constant throughout the period in experimentally intraruminal administration lactic acid to sheep.

Cakala et al. (1975) reported that there was increased concentration of serum inorganic phosphorus in cow fed a large amount of grain food.

Mullen (1976) observed that there was increase in serum inorganic phosphate level in overfeeding in cattle.

Jagos et al. (1977) observed that there was increased concentration of serum inorganic phosphorus in chronic metabolic acidosis in dairy cows.

Jones et al. (1977) reported that in the advanced stages of protracted rumen acidosis, plasma calcium levels might be decreased to less than one half of normal.

Melvin (1977) reported that serum calcium and phosphorus level in normal goat ranged from 4.5 to 6 mEq/L and 2 - 5.2 mEq/L respectively.

The phosphorus level in blood plasma tended to rise as the concentrate to roughage ratio was increased. The blood pH had negative association with plasma phosphorus in sheep (Nakota et al., 1977).

Jones and Luthman (1978) reported that addition of concentrate to the diet of hay-fed sheep decreased serum calcium 0.4 kg/kg body weight of concentrate was needed to induce this drop in serum calcium.

Nauriyal and Baxi (1978) observed that serum calcium and inorganic phosphorus level did not show any change within 48 hours of experimentally induced ruminal lactic acidosis in cattle and buffaloes.

Terashima et al. (1978) observed that injection of lactic acid i/v or into the rumen of hay-fed sheep caused increased serum lactic acid with increased excretion of calcium and phosphorus through urine.

Sethuraman and Rathor (1979) reported normal range of serum calcium level in cattle but increase serum calcium level in buffaloes, whereas serum inorganic phosphorus rose significantly both in cattle and buffalo in rumen acidosis.

Anderson (1980) reported that in acid indigestion serum calcium level decreased which might contribute to the myasthenia. The serum phosphorus was likely elevated as a result of both increased phosphate absorption and decreased renal loss.

Vihan et al. (1982) reported that serum calcium concentration decreased consistently with the change of the rumen pH and was found to have positive correlation with the pH in barley as well as in grain induced acidosis in goats.

The inorganic phosphorus level was found within the normal range however the higher values were found in early hours of acidosis in grain and cane sugar. He observed that serum calcium level decreased from 10.2 mg/dl at 0 hour to 6.5 mg/dl at 18 hours of barley induced rumen acidosis and to 3.8 mg/dl at 66 hours of grain (50% barley + 50% wheat) induced rumen acidosis in goats. At the same time serum inorganic phosphorus level increased to 9.1 mg/dl and 11.1 mg/dl in 42 hours of grain (50% barley + 50% wheat) and 12 hours of cane sugar induced rumen acidosis respectively. He also found that serum calcium and phosphorus in normal control goats were 12.75 mg/dl and 4.37 mg/dl respectively.

Blood et al. (1983) reported that inorganic phosphate level rose and in almost all cases there was a mild hypocalcaemia which was presumably due to a temporary malabsorption.

Sastry (1983) reported that mean value of normal calcium level in serum was 10.7 mg/100 ml and phosphate level ranged from 3.0 to 11 mg/100 ml in goat.

Fraser et al. (1986) reported that in rumen acidosis there was increased serum inorganic phosphate and mild hypocalcaemia.

Cao et al. (1987) observed that mean plasma inorganic phosphate rose from 1.73 $\pm$ 0.129 mmol/L to 2.97 $\pm$ 1.515 mmol/L

at 24 hour while Ca^{++} concentration remained steady throughout in experimentally induced lactic acidosis in the goat.

Robinson and Huxtable (1988) reported that a number of additional problems might occur in animals suffering from ruminal acidosis, these being hypocalcaemia resulting from calcium malabsorption which in turn caused by the low pH of digestion entering the duodenum in the acute stage of the disease.

2.4.2.5 Total serum Protein,Albumin,Globulin,Albumin:Globulin ratio

Gorczyca *et al*. (1960) reported that mean normal values of total protein and albumin in goat both male and female, aged 7 - 9 months were 6.25 ± 0.35 g/dl and 3.95 ± 0.26 g/dl respectively.

Altman and Dittmer (1961) reported that normal levels of total serum protein, albumin and globulin in goat were 6.67 g/100 ml, 3.96 g/100 ml and 2.71 g/100 ml respectively, but total plasma protein was 7.27 g/100 ml.

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

Prasad and Joshi (1971) found low albumin and A/G ratio in most cases of rumen dysfunction in buffaloes and

concluded that in rumen dysfunction varying degrees of hepatic stress was inevitable.

Prasad et al. (1972) recorded low level of serum albumin compared to the level of serum globulin in cattle and buffaloes in acid indigestion. There was also decrease in serum albumin : globulin ratio, which were 0.77 ± 0.005 in cattle and 0.66 ± 0.003 in buffaloes.

Jonson and Liberg (1974) found decrease in the level of total protein, globulin and fall of A/G ratio in intensively fed calves.

Prasad and Joshi (1975) reported that there was decrease in serum albumin percentage and albumin; globulin ratio in primary rumen impactions in Zebu cattle and buffaloes, which indicated disturbances in protein in metabolism or hepatic insufficiency.

Concentration of total plasma protein varied with the degree of dehydration present in carbohydrate overfeeding in cattle (Mullen, 1976).

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

There was reduction of serum albumin parallel to the reduction of albumin:globumin ratio in acid indigestion of heifers (Bienick, 1981).

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

Holtenius (1982) reported that animals with liver abscesses had as a rule normal liver function tests but with high gammaglobulin and low albumin level in serum.

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

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

HejLasz et al. (1984) observed that there was reduced serum albumin and increased gammaglobulin value in experimental acidosis of cow due to displacement of water from the blood and tissue to the digestive tract.

Cao et al. (1987) reported that mean total plasma protein increased from 80.4 ± 5.34 g/litre at '0' hour to 88.0 ± 3.37 g/litre at 24 hours and then again declined to 81.4 ± 6.65 g/litre at 48 hours in experimentally induced lactic acidosis in goats.

Smith (1990) reported that normal values of serum total protein in goat ranged from 2.7 to 3.9 g/dl, albumin ranged from 2.7 tc 3.9 g/dl and A/G ratio was 0.6 - 1.3.

2.4.2.6 Blood Urea and Serum Creatinine

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

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

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

Furll et al. (1977) observed that serum creatinine concentration remained within normal physiological range in sheep aged two and three years during a six week course of metabolic acidosis.

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

Melvin (1977) reported that the normal concentration of blood urea nitrogen and creatinine of goat were 13 - 28 mg/dl and 1 - 2 mg/dl respectively.

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

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

Anderson (1980) reported prerenal azotemia with the increase of BUN up to 150 mg/dl and comparable elevations in plasma creatinine in acid indigestion of ruminant.

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

Sastry (1983) reported that blood urea nitrogen and creatinine level in normal goat were 13 - 28 mg/100 ml and 0.9 to 1.82 mg/100 ml respectively.

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

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

Smith (1990) reported that decreased renal function is reflected by elevated serum creatinine and urea nitrogen concentration in rumen acidosis.

2.4.2.7 Serum Sodium and Potassium

Huber (1971) observed decrease in serum sodium and potassium in glucose induced acute indigestion in sheep.

Telle and Preston (1971) reported that the blood potassium concentration increased upto 4 hours of intraruminal infusion of lactic acid in sheep and then back to the '0' hours value. The concentration of sodium remained relatively constant.

Shinosaki and Nakabayashi (1974) reported decrease in plasma potassium and increase in plasma sodium in acidosis of sheep.

Irwin et al. (1979) reported that plasma sodium level increased slightly during glucose induced acidosis in sheep.

Sen (1982) observed that there was increase in serum sodium concentration and decrease in serum potassium concentration in experimental acidosis in goats.

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

Das (1990) reported increase in serum sodium and decrease in serum potassium level in experimental rumen acidosis in goats.

2.5 Observations of Serum Hormones

2.5.1 Cortisol

Salye (1958) reported that due to stress of digestive disorder propably more adrenocorticoids are released as anti-stressors of the body.

Linder (1967) reported that plasma cortisol level in goat ranged from 8.0 - 19.0 ng/ml (0.8 - 1.9 µg/dl) with mean value 12 ng/ml (1.2 µg/dl).

Mills and Jenny (1979) observed that high concentrate feeding decreased the molar proportion of rumen acetate and increased that of propionate in dairy heifers. Mean value for the first 12 hours after feeding (feeding period) for high concentrate and control heifer were 14.8 and 7.9 ng/ml for total glucocorticoids which indicated more glucocorticoids value in concentrate feeding animals than that of control animals. Starvation decreased plasma glucose and insulin and increased plasma free fatty acids and glucocorticoids.

2.5.2 Insulin

Horino (1968) reported that insulin concentration in adult sheep had been observed as 20 - 30 µU/ml of plasma.

Horino *et al.* (1968) showed that glucose, propionate and butyrate stimulated insulin secretion in cows and plasma insulin level increased in cattle fed with high grain diets.

Trenkle (1970) reported that feeding of grains to ruminants increased insulin secretion, which would seem to be caused by the higher proportions of propionate and butyrate

produced in the rumen of sheep and cattle fed high grain diet. The grain fed sheep has significantly higher concentrations at 24 hours and 72 hours than the hay fed sheep.

Walket and Elliot (1973) obtained insulin concentration as 20.6 μU/ml in serum of adult cow.

Jenny and Polan (1975) studied the effect of ration on serum glucose and insulin concentrations in 12 Holstein Fresian Cows. High grain feeding (15% hay and 85% concentrate) increased glucose and insulin at all hours post feeding as compared to the control cows (fed 55% corn silage, 10% hay and 35% concentrate). In the cows fed with grains, blood glucose concentration increased from 63.3 to 72.2 mg percent and insulin from 19.2 to 25.6 μU/ml serum just before feeding to three hours postfeeding.

Bueno et al. (1977) observed that plasma immunoreactive insulin (IRI) levels increased during feeding of cereal pellets and were related to plasma glucose concentration but plasma IRI levels remained unchanged during feeding of grass or hay in sheep.

Chasen et al. (1977) observed that portal insulin within 2 minutes of meal initiation (3.4 Kcal/g digestible energy) in steers remained elevated for 1st 14 minutes of meal. On control days when no feed was consumed, there were no significant changes in concentrations of metabolites or insulin.

Kolb (1977) reported that in ruminants insulin secretion was stimulated by increase in glucose, propionate, butyrate and valerate level in the blood plasma.

Pearson et al. (1977) reported that normal level of insulin in adult goat was 6.8 $\pm$ 0.3 μU/ml.

Hove and Halse (1978) studied the effect of feed intake on plasma insulin in 38 cows with plasma sugar ranging from 41 to 86 mg/100 ml and acetoacetate (ACAC) ranging from 0.2 to 18 mg/100 ml, measured before morning feeding. The animals were fed concentrates silage and hay. In animal with low blood ACAC plasma insulin concentrations began to increase as early as $\frac{1}{2}$ hour after the start of feeding and reached maximum after 2 hours. Simultaneously ACAC increased and sugar decreased markedly. Animal with ACAC of 1 mg/100 ml had low prefeeding insulin concentrations and the level of the hormone did not increase after feeding. Glucose was infused at a low rate (0.9 g/min) for 10 hours into a hypoglycaemic, ketonaemic cow. As glucose and ketone levels became normal, insulin levels also increased in response to feeding.

Konider et al. (1978) observed that infusion of glucose solution resulted in only a brief increase of plasma fructose in sheep. The highest insulin rise in blood plasma occurred after infusion of fructose solution.

Thompson et al. (1978) reported that in sheep after feeding in the neutral environment temperature there

were significant increase in portal blood flow and release of VFA into the portal blood stream. The uptake of propionate by liver increased and output of glucose increased slightly. Plasma insulin concentration also increased after feeding.

Randhawa (1979) found increased level of circulating immunoreactive insulin (IRI) during first 48 hours and then declined gradually and reached below the base values at 120 hours of experimental rumen acidosis in buffalo calves.

Hayakawa et al. (1985) observed that a rapid intravenous infusion of 50 ml of a 50% glucose solution or 100 ml of a 25% xylitol solution in ewes caused increase in level of blood insulin.

2.5.3 Thyroxin level

Kallfelz and Erali (1973) reported that in goat normal thyroxin level was 5.26 ± 2.08 µg/100 ml (52.6 ± 2.08 ng/ml).

Reep et al. (1978) reported that serum thyroxin (T_4) level in goat ranged from 3.0 to 4.23 µg/dl (30 - 42.3 ng/ml).

2.6 Pathological lesions

2.6.1 Macroscopic lesions

Ahrens (1967) reported swollen edematous rumen papillae with development of hyperemia and haemorrhage.

Udall (1972) observed that in overloading with heavy ground feed in cattle the rumen and reticulum showed large black areas with congestion and haemorrhage, their walls were thickened and friable; the mucosa of the omasum was haemorrhagic while the abomasum and small intestines were either congested or severly inflammed. The serous membrane of the heart showed haemorrhages and the heart muscle showed degeneration.

Hartig and Hebold (1973 a) on post mortem examination observed haemorrhages in abomasum, large and small intestines in sheep in rumen acidosis.

Vestweber and Leipold (1974) observed gross changes like congestion and oedema of the lungs; congestion of the meningeal blood vessels and swelling; oedema and dilatation of ventricles of the brain, effusion and ecchymotic haemorrhages of the epicardium in ovine ruminal acidosis. Ruminal papillae were swollen and larger than normal.

Dshurov (1975) reported that there were presence of haemorrahge in rumen, reticulum and omasum in rumen acidosis of calves. The abomasum was ulcerated, lymph nodes draining the fore-stomachs were enlarged.

Dshurov (1976 a) observed macroscopic lesions like hyperemia and blood leakage beneath the ruminal mucosa; in some cases this occured in the second and third stomach, prolonged oedema and hyperaemia in the lungs, meninges and brain and blood leakage from the epicardium in experimental

and spontaneous rumen acidosis in sheep.

Dshurov (1976 b) observed that there were desquamation of the ruminal villi, hyperaemia, bleeding and erosions in the propia and enlargement of gall bladder, sometimes necrotic foci in the rumen, bleeding and hyperaemia in the reticulum and omasum, ulcers in the abomasums and swelling of the lymph nodes arround the forestomachs in ruminal acidosis of fattening calves.

Nauriyal et al. (1978) observed gross changes like exfoliative changes in the rumen mucosa, peritonitis, hepatic necrosis, duodenal and abomasal haemorrhages, pulmonary oedema, necrotic changes in the kidney, degeneration the myocardium and congestion of brain and meninges in rumen lactic acidosis in buffaloes and cattle.

Sodhi et al. (1981) performed post mortem examination of the peracute cases of rumen lactic acidosis in calves and found severe cerebral haemorrhages, gastro-enteritis, pneumonia, general venous congestion and enlargement of the liver.

Sen (1982) observed gross changes like sloughing of rumen mucosa, hyperaemia of abomasal mucosa, congested liver, enlarged gall bladder in rumen acidosis in goats.

Teli et al. (1986) observed swollen and pale liver, enlarged spleen, petechial haemorrhage on the surfaces of spleen and heart in acid indigestion of ewes

by feeding of damaged apple. Gall bladder contained thick, mucoid and yellowish bile.

Das (1990) observed gross postmortem changes like sloughing of the rumen mucosa, hyperaemia over abomasal mucosa, congested mesenteric blood vessels, patecheal haemorrhages on the small intestinal mucosa, scattered congestion over liver and enlargement of gall bladder in experimental rumen acidosis in goats.

2.6.2 Microscopical lesions

Jensen et al. (1954 a) observed epithelial necrosis, vesicles and colonies of bacteria including Spherophorous necrophorus, which were penetrated, injured tissue of rumen of cattle.

Delak and Adamic (1959) observed histopathological lesions like catarrhal gastroenteritis and degenerative lesions in liver, kidney and heart muscle in sucrose intoxication in sheep.

Strafuss and Monlux (1966) reported severe congestion, mild perivascular lymphocytic cuffing, chromatolysis of neurons, satellitosis and neuronophagia in brain in digestive disturbances of ruminants.

Ahrens (1967) reported that there was rumenitis with the development of microvesicles of the stratum lucidum, hyperaemia and then epithelial desquamation and detachment of

area of mucosa in lactic acidosis of cattle.

Franklin (1967) indicated a loss of keratin and vaculation of cytoplasm of epithelial cells, rupture of the cells, microvesicle formation and neutrophilic infiltration in ruminal papillae in experimental rumen acidosis in cattle.

Dirksen (1970) and Thomson (1967) observed degenerative changes in the heart, liver, muscles and kidney following death from ruminal acidosis.

Hartig and Hebold (1973 a) observed microthrombi in almost all the organs in experimental acute rumen acidosis in sheep. Lymphatic tissues displayed swelling of the reticular cells and disappearance of lymphocytes.

Vihan et al. (1973 a) reported epithelial desquamation, superficial necrosis, formation of vesicles and vacules in the stratum lucidum, neutrophilic infiltration and capillary congestion in the rumen in experimental rumen acidosis in buffaloes. Abomasal mucosa showed degenerative changes and neutrophilic infiltration. Brain tissues also indicated congestion of capillaries and neutrophilic infiltration.

Vestweber and Leipold (1974) reported that there was microscopic changes of the brain like perivascular and perineural oedema, death of neurones, increased vascularity and congestion and gliosis in ruminal acidosis of sheep. Mycarditis was a consistent feature. The kidneys showed degenerative

changes of the tubules, Polymorphonuclear infiltration and microvesicles were observed on the ruminal mucosa. Lymphocytic infiltration occurred in the submucosal area of the small intestine.

Histologically the rumen epithelium showed necrotic inflammation and there was desquamation of the mucous membrane of omasum and reticulum. Inflammatory and degenerative changes were also present in liver, kidney, pancreas and thyroid gland in ruminal acidosis in calves (Dshurov, 1975).

Brent (1976) observed that liver abscesses were produced by the lactic acidosis in ruminants.

Histological findings like desquamation of the ruminal villi, infiltration of lymphocytes and leukocytes into the rumen and forestomach, subacute hepatitis (in 60% of the cases) degeneration of kidneys, focal myocarditis (50% of the cases), hyperaemia, oedematization and blood leakage in the lungs, perivascular and pericellular oedema in the brain and severe hyperaemia of the menings and thyroid glands in acute and subacute acid indigestion in sheep were observed (Dshurov, 1976 a,b).

Sethuraman (1976) observed degenerative changes with infiltration of polymorphonuclear cells in liver, intestine and kidney in rumen acidosis of buffalo and cow calves.

McManus (1977) reported microlesions on the rumen papillae of lambs fed with wheat grain diet.

Landsverk (1978) observed infiltrations of polymorphonuclear leukocytes with the formation of microabscesses in the rumen mucosa in barley and barley plus hay fed indigestion in calves. The ruminal papillae were thickened, club-shaped and sometimes nodular.

Nauriyal et al. (1978) observed in rumen lactic acidosis of buffaloes and cattle that there were severe diffuse coagulative changes in the renal tubules, thickening of the interstitial septa in the lungs and haemorrhages in the Virchow-Robin Space of the brain.

Randhawa et al. (1981 b) observed histological lesions which included diffuse coagulative necrosis and microabscesses in the liver parenchyma, glycogen depletion of hepatocytes, ulceration and microabscesses in the reticulum and rumen, enlargement of cells of zona fasiculata with some cells having 2 - 3 nuclei, marked infiltration of lymphoid cells in the mucous membrane of intestine, deposition of haemosiderin in the mucous membrane of intestine, congested, oedematous and emphysematous lungs, degenerated renal tubules chromatolysis of neurons, satellitosis and neuronophagia in the brain and atrophy of the exocrine epithelial cells lining the acini of the pancreas and atrophy of the endocrine beta cells with degranulation of cytoplasm of catecholamine secreting cells of adrenal medulla in ruminal acidosis in buffalo calves.

Sen (1982) observed microscopical changes like

desquamation, hydropic degeneration and accumulation of large number of heutrophils forming microabscesses over epithelial cells of reticulum and ruminal mucosa. Hydropic degeneration and microabscesses over liver parenchyma were also observed in goats died due to grain overload.

Ivanov et al. (1987) observed degenerative changes in the liver in clinical acidotic cows.

Chihaya et al. (1988) reported desquamation, necrosis of walls of forestomach, focal necrosis, diffuse haemorrhages and infiltration of neutrophils in the abomasum by barley induced acidosis in sheep.

Das (1990) observed desquamation and infiltration of neutrophils and mononuclear cells in rumen, reticulum, omasum and abomasum, congestion, haemorrhage, necrosis and microabscesses in liver in experimental rumen acidosis in goats.

2.7 Therapy

Bullen and Scarisbrick (1957) reported that treatment with crystalline sodium penicillin was effective to inhibit the growth of gram-positive flora and thus check the intraruminal pH in experimentally induced rumen acidosis in sheep.

Broberg (1960) suggested that treatment of acute overeating should aim at reducing the high blood content of

lactic acid. He recommended large oral doses of brewer's yeast and intravenous administration of thiamine for an effective recovery from acidosis and oral therapy with alkalizers.

Dirksen (1965) recommended treatment with antihistamines, vitamin B complex, yeast, prednisolone, fluid and electrolyte supplementation, rumenotomy in early cases of ruminal acidosis.

Juha'sz and Szegedi (1968 d) gave the line of treatment for acidosis in sheep viz. infusion of 50 - 100 ml of 10% solution of sodium hydroxide into rumen when rumen pH was below 4.0, 1-2 g of broad spectrum antibiotic in water in severe cases, a further infusion of reduced dose of sodium hydroxide or 200 ml of 25% sodium chloride solution had been suggested. The additional injection of large doses of thiamine was also recommended.

Dirksen (1970) recommended the treatment like discontinuance of the feed, orally alkalizers, antibiotics, baker's or brewer's yeast in early cases. In severe cases antihistamines, thiamine, physiological saling with calcium and magnesium - gluconate (subcutaneously) was recommended. For restoration of normal flora and fauna rumen cud transfer should be done.

Gnanaprakasam (1970) treated clinical cases of acidosis in goats by clearing toxic ruminal contents and transplanting fresh ruminal cud, and with antacid, antihistamine, thiamine, fluid with electrolytes.

Huber (1971) suggested isotonic electrolytes to combat dehydration in ruminal acidosis.

Vestweber et al. (1974) reported 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 sodibicarbonate was ineffective.

Prasad and Rakib (1975) did not find any satisfactory result in severe acidosis (p.H between 4.6 to 4.5) in bucks and lambs with treatment of injection sodium bicarbonate 7.5% intravenously and sodium bicarbonate orally. But sheep suffering from severe acidosis cured with the same line of treatment and nuxvom orally. The moderate acidotic group (pH between 5.00 and 5.2) cured with same line of treatment.

Juha'sz and Szegedi (1976) reported that intraruminal administration of Bykodigest antacid (a proprietory mixture of carbonates, sulphates and other compounds) resulted in normal rumen pH in experimental ovine lactic acidosis. The antacid resulted in 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 hours after overeating, administration of antibiotics such as chloramphenicol or penicillin-v mixed with grain in over feeding in cattle.

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

Beede and Farlin (1977) reported that capreomycin disulphate reduced lactate concentration by about 69% suggesting enhanced utilization of lactate yielding propionate. Ruminal pH increased and decreased the acetate (A) to propionate (P). Treatment with oxamycin increased ruminal pH and the A : P ratio increased linearly. Total volatile fatty acid concentration was unaltered by oxamycin treatment in induced acidosis in sheep.

Kezar and Church (1979 b) prevented acute acidosis and maintained higher pH in sheep 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.

Sen (1982) treated experimentally induced ruminal acidosis in goats by evacuating rumen content through fistula, intraruminal administration of tetracycline hydrochloride, intraruminal administration of aluminium hydroxide, intravenous sodium bicarbonate administration, parenteral 0.9% sodium chloride administration, intravenous administration of thiamine hydrochloride, calborol (M/s May and Baker India Pvt. Ltd.) injection, intraruminal antihistaminics administration, fresh rumen cud replacement and Rumenton (M/s Pfizer

India Pvt. Ltd.) tablet intraruminal administration.

Amstel (1983) found that magnesium oxide was very potent than magnesium hydroxide, magnesium trisilicate, calcium carbonate, aluminium hydroxide and sodium bicarbonate in alkalinising ability in treatment of clinical cases of rumen acidosis, whereas calcium hydroxide and magnesium carbonate give satisfactory result.

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

Tanwar et al. (1983) treated experimentally induced rumen acidosis in goats with magnesium carbonate orally, fresh rumen content intraruminally along with vitamins.

Howard (1986) treated acidosis in ruminants by emptying rumen contents, oral administration of antacids such as magnesium carbonate or magnesium hydroxide, intravenous administration of balanced electrolytes and 5% sodium bicarbonate solution and administration of antihistamines.

Sinha et al. (1985) followed the treatment schedule like (i) partial removal of ruminal content (ii) intraruminal administration of magnesium hydroxide, tetracycline water soluble powder and fresh rumen cud drawn from healthy buffalo cattle, molasses and cobalt sulphate (iii) intravenous

infusion of 5% sodium bicarbonate, 0.85% of sodium chloride and thiamine hydrochloride (iv) intramuscular injection of vitamin B_{12} with liver extract (v) Fresh water at frequent intervals in experimental ruminal acidosis in buffalo calves.

Cao *et al.* (1987) treated experimentally induced lactic acidosis in goat by administering calcium hydroxide 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.

Fig. 1. Fistulated male goat.

3.0 MATERIALS AND METHODS

3.1 The present study was conducted in the Department of Veterinary Medicine and Public Health, Faculty of Veterinary and Animal Sciences, Bidhan Chandra Krishi Viswa Vidyalaya, Mohanpur in the District of Nadia of West Bengal from May 1990 to June 1991.

3.2 Preparation of the Experimental Animals.

Eighteen healthy Black Bengal goats of either sexes with a body weight between 12 to 18 kg were taken up for the experimental studies. The animals were maintained in semi - intensive system for one month. They were allowed to graze at least for eight hours in a day and provided concentrate ration for their maintenance. The animals were also had free access to drinking water throughout the day. During this period haemoglobin percentage of each goat was estimated to rule out anaemia and faeces was examined to rule out parasitic burden. Deworming was done in positive cases with 'Valbazen liquid'* (2.5% suspension) at the dose rate of 6 ml per 30 kg body weight orally.

The experiment was started on clinically healthy goats. All goats were fitted with fibre glass rumen fistula (length 7 cm, diameter 1.5 cm) on the left paralumber fossa (Fig. 1 & 2) as per the method described by Wakanker et al. (1980).

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* Valbazen suspension - A product of M/s Eskayef Pharmaceuticals Pvt. Ltd., Bangalore, containing albendazale, 2.5% suspension.

Antibiotic therapy was given for the next four days and the operated area was dressed with antiseptic lotion and fly repellant solution was applied till healing was complete.

A minimum of three weeks time was allowed for them to recover after surgical operation. Leakage of no gas or fluid from rumen through fistula indicated complete healing. The external openings of fistula were always tightly capped to prevent any leakage of rumen gas or fluid or entry of outside air.

For therapeutic trials 36 numbers of natural acid indigestion cases were included.

3.3 Experimental Procedure

The above experimental goats were divided into three groups viz. 'A', 'B' and 'C' having 6 animals in each group. Group 'A' was selected as control healthy group. Group 'B' and 'C' were selected as experimental group in which rumen acidosis were produced.

After induction of acidosis group 'B' was kept without treatment. Group 'C' was again subdivided into C_1 and C_2 groups each comprising of 3 goats. C_1 is the mild acidotic and C_2 is the severe acidotic group.

Natural clinical cases were designated as group 'N' which was again sub divided into three groups viz. 'N_1', 'N_2' and 'N_3', each comprising of at least 10 - 12 numbers of

animal . The mild acidotic group was 'N_1' and moderate and severe acidotic groups were 'N_2' and 'N_3'.

Animals of group 'C', 'N_1', 'N_2' and 'N_3' were subjected to different therapy on the basis of ARD profiles (Anorexia, ruminal dysfunction and defaecation).

3.4 Experimental Induction of Rumen Acidosis by Crushed Rice

All experimental animals were weighed and fasted for 24 hours. After fasting rumen acidosis was produced by intraruminal administration of crushed rice through rumen fistula at the dose rate of 70 g/kg body weight except 'C_1' group. Mild acidosis was produced in group 'C_1' by intra-ruminal administration of crushed rice at the dose rate of 40 g/kg of body weight. The animals were kept under close observation untill acidosis started and food was not given to them during the experimental period. The goats were allowed to drink water ad libitum.

3.5 Clinical Accounts and Observations

Clinical manifestations like general condition, pulse rate, rectal temperature, rumeno-reticular contractions rate, rumination, respiration rate, abdominal condition, colour, consistency, amount of faeces and pH of faeces and colour, quantity and pH of urine were recorded as per the methods described by Chakrabarti (1988) in healthy control group of goats in the morning for 3 consecutive days. The pH

of the urine and faeces were also recorded with the help of pH paper immediately after collection. In each time urine samples were collected after proper catheterization.

In untreated group of goats with acid indigestion, the above observations and symptoms were recorded at '0' hour and every 12 hours interval till death or recovery.

3.6 Collection and Analysis of Rumen liquor

3.6.1 Collection of Rumen liquor - The goats were controlled in the standing position and the caps of the fistula were opened. A polythene tube was inserted through the fistula at various positions and depths in the rumen to have the representative samples of the rumen liquor.

With the help of plastic enema pump syringe fitted with that polythene tube (Fig. 3) about 60 ml of rumen liquor was collected in wide mouthed air tight stoppered polythene bottle under thin paraffin layer to prevent exposure to air. The rumen liquor was strained through double folded gauze cloth. Some amount of strained rumen liquor (SRL) was poured into cleaned plastic bottle containing few drops of 10% mercuric chloride solution. The bottles filled with rumen liquor upto the brim were immediately stoppered and skaken for thorough mixing of the liquor with mercuric chloride. These samples were transferred to deep freeze for further analysis of Ammonia-nitrogen (NH_3-N) and Total volatile fatty acid (TVFA). For other biochemical estimations some

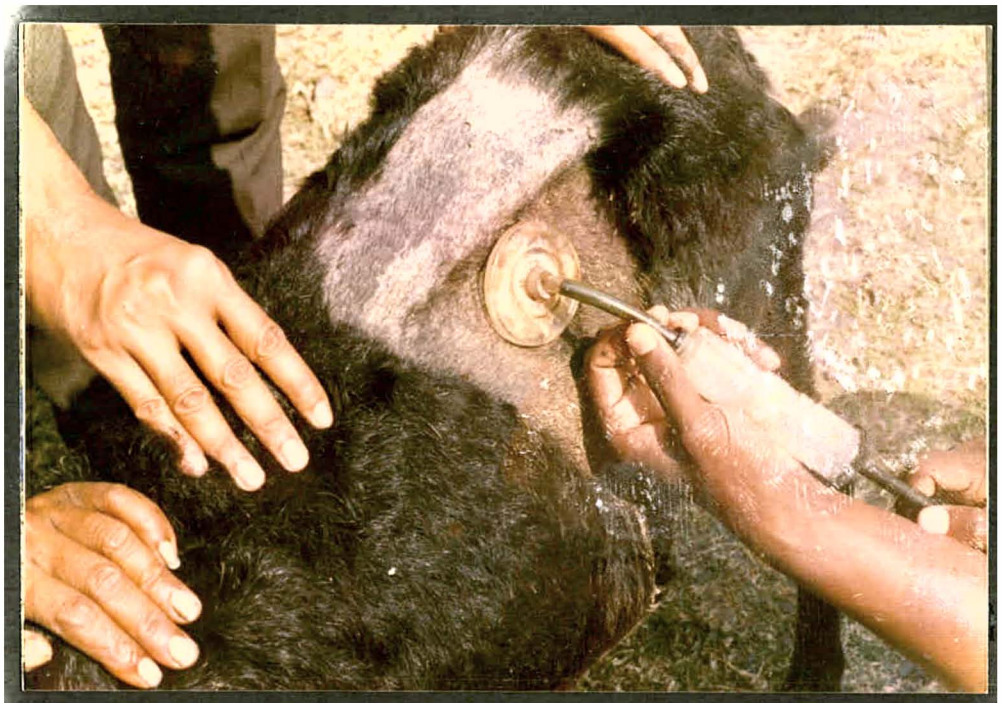

Fig. 3. Collection of rumen liquor with enema pump syringe fitted with polythene tube.

amount of rumen liquors were taken for preparation of protein free filtrate as per the method described by Nelson-Somogyi elaborated by Oser (1979).

Few amount of SRL was preserved with equal volume of 10% formaline and used for enumerating total number of protozoa and bacteria.

The remaining samples of rumen liquor were taken immediately for analysis of colour, consistency, smell, pH, sediment activity time (SAT), methylene blue reduction time (MBRT), cellulose digestion time (CDT) and concentration, motility and iodophilic nature of protozoa. Samples of rumen liquor were collected for 3 consecutive days in the morning from healthy control group of goats. In case of untreated acidotic group of goats, it was made before fasting ('0' hour) and at every 12 hours and sometimes at 6 hours interval following experimental rumen acidosis.

3.6.2 Analysis of Rumen liquor

3.6.2.1 Macroscopic

3.6.2.1.1 Colour, Consistency and Smell - The colour, consistency and smell of rumen liquor were recorded as per the method adopted by Misra and Tripathy (1963).

3.6.2.1.2 Sediment activity time (SAT), Cellulose digestion time (CDT) and Methylene Blue reduction time (MBRT)

SAT of strained rumen liquor was recorded as per the method described by Nichols and Penn (1958). It was expressed as minutes. CDT of strain rumen liquor was studied using the method described by Chakrabarti (1988). It was expressed as hours.

MBRT of strained rumen liquor was recorded as per method described by Chakrabarti (1988). It was expressed as minutes.

3.6.2.2 Microscopic

3.6.2.2.1 Concentration, Motility and Iodophilic nature of rumen Protozoa

These were studied using the method described by Misra et al. (1972) with slight modification. 1 - 10 protozoa, 11 - 30 protozoa and above 30 protozoa per microscopical field indicated +(low), ++(moderate) and +++(high) concentration of protozoa respectively.

The motility of protozoa was graded under eye and estimated as - (absent), + (slow), ++ (moderate) and +++ (vigorous).

For study of iodophilic nature of rumen protozoa a smear of fresh strained rumen liquor covered with cover-slip was stained with a drop of lugol's iodine and examined under microscope. Protozoa containing more starch showed brownish blue colour. Based on starch content of protozoa

the iodophilic nature could be graded as - (nil), + (low), ++ (moderate) and +++ (high).

3.6.2.2.2 Total Protozoal count - This was studied using the method described by Purser and Moir (1966). Protozoa present in formalinised SRL was stained by adding lugol's iodine and uniformly suspended in solution using 30% freshly prepared glycerol. Counting was made on Neuber's haemocytometer under 10× lens of compound microscope. The total protozoal count was expressed as number x 10^4/ml of SRL.

3.6.2.2.3 Total Bacterial count - This was studied as per method described by Gall et al. (1949). Formalinised SRL was serially diluted to 1 : 10,000. 0.02 ml of diluted SRL was pipetted on a glass slide and mixed with saturated nigrosine solution with the help of a platinum loop (3mm. diameter) and spread uniformly over a 2 x 2 cm area. After drying, 20 fields were counted under oil immersion lens of compound microscope. The total bacterial count was expressed as average number in twenty fields x 10^9/ml SRL.

3.6.2.2.4 Type of Bacteria in the rumen fluid - Smear for direct microscopic examination were made as per the method described by Hungate et al. (1952) by diluting the rumen contents 10 times with distilled water and spreding 0.01 ml of this over 1 square centimeter on the slide. They were dried, fixed on flame and stained with Gram's method.

3.6.2.3 Biochemical

3.6.2.3.1 pH of SRL - The pH of SRL was recorded immediately after its collection with the help of Systronics Digital pH meter type 335 (Systronics, Amedabad, India).

3.6.2.3.2 Lactic acid of SRL - The amount of lactic acid present in rumen liquor was measured as per the method described by Barker and Summerson (1941). It was expressed in mg/ 100 ml of SRL.

3.6.2.3.3 Ammonia Nitrogen (NH_3-N) of SRL - The concentration of NH_3-N in the rumen liquor was estimated as per the method of Wooton (1964). 4.75 ml of SRL and 0.25 ml of 1.0 (N) H_2SO_4 were centrifused and 2 ml of supernatant was steam distilled with an excess of alkali in Microkjeldahl apparatus. The distillate was collected in 10 ml of one percent boric acid containing mixed indicator and titrated against 0.01N H_2SO_4. The result was expressed in mg% of SRL.

3.6.2.3.4 Total Volatile Fatty Acids (TVFA) of SRL - The concentration of TVFA in the rumen liquor was determined as per method of Barnett and Ried (1957). One ml of SRL and two ml of mixture of oxalic acid 5% and potassium oxalate (10%) in equal proportions were steam distilled in Markham's Microkjeldahl distillation apparatus. About 80 ml of distillate was collected and titrated against 0.01 (N) NaoH using phenolpthalein as indicator. The concentration of TVFA was expressed as milliequivalent per litre of SRL (mEq/L).

3.7 Collection and examination of Blood/Serum

3.7.1 Collection of Blood and Serum - About 16 ml of blood was collected from each animal from jugular vein with a sterilized syringe. Out of which 9 ml of blood was collected in sterilized dry test tube without anticoagulant for seperation of serum. The separated serum was centrifused to clear any blood corpuscles . The centrifused serum was then preserved in deep freeze at - 15°C for further biochemical work.

Then 5 ml of collected blood was taken in a sterilized vial containing 5 g sodium fluoride under a layer of liquid paraffin to prevent exposure to air for measurement of pH of blood. After which the blood was stored in refrigerator at the temperature of 4°C. From this sample 1 ml of blood was taken for immediate glucose estimation and 1 ml of blood was taken to prepare protein free filtrate as per the method of Nelson - Somogyi described by Oser (1979) for estimation of lactic acid. The protein free filtrate was stored in refrigerator at 4°C.

Remaining 2 ml of blood was taken in sterilized vial containing EDTA (Ethylene diamine tetraacetic acid), as anticoagulant, at a dose of 2 g/ml of blood for Hb percentage, total erythrocytic count (TEC), total leukocytic count (TLC) and differential leukocytic count (DLC).

Samples of blood and serum were collected on every alternate day for three occasions in the morning from

healthy control group of goats. In untreated group, the sample was collected before fasting ('0' hour) and at every 12 hours intervals and sometimes at every 6 hours interval of experimental rumen acidosis till death or recovery.

3.7.2 Examination of Blood/Serum - This included haematological examination and biochemical examination.

3.7.2.1 Haematological examination

3.7.2.1.1 Packed cell volume (PCV) - PCV was determined in Wintrobe tubes as per Wintrobe method (1956) and was expressed as percentage.

3.7.2.1.2 Haemoglobin (Hb) percentage - Hb percentage was determined by Sahlis's method as described by Schalm et al. (1986).

3.7.2.1.3 Total Erythrocytic count (TEC) - TEC was done by haemocytometer as per method described by Schalm et al. (1986) and was expressed as millions per cubic milimeter (10^6/cmm).

3.7.2.1.4 Total Leukocytic count (TLC) - TLC was estimated by haemocytometer as described by Schalm et al. (1986) and was expressed as thousands per cubic milimeter (10^3/cmm).

3.7.2.1.5 Differential Leukocytic count (DLC) - DLC was done as per method described by Schalm et al. (1986) and was expressed as percentage.

3.7.2.2 Biochemical examination of Serum/Blood

3.7.2.2.1 Estimation of Serum Calcium - Serum calcium level was estimated colorimetrically by O - Cresolphthalein Complex one (O.C.P.C.) method with the help of diagnostic reagent kit* of calcium and was expressed in mg%.

3.7.2.2.2 Estimation of Serum Inorganic Phosphorus - Serum phosphorus was determined spectrophotometrically by Gomorri's method as described by Varley (1969) and was expressed in mg%.

3.7.2.2.3 Estimation of Blood Urea - Blood urea was estimated colorimetrically by Urease Nesslerization method as described by Varley (1969) and was expressed in mg%.

3.7.2.2.4 Estimation of Serum Protein, Albumin (A), Globulin (G) and A/G ratio

These were determined colorimetrically by Biuret method as described by Reinhold (1953) and were expressed in mg%.

The A/G ratio was obtained by dividing the percent of albumin by percent of globulin in the serum.

$$\text{A/G ratio} = \frac{\text{percent of albumin}}{\text{percent of globulin}}$$

3.7.2.2.5 Estimation of Blood Glucose - The concentration of blood glucose was estimated by Ortho - Toluidine method as described by Varley (1969).

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* Diagnostic reagent kit for determination of calcium in serum - Span Diagnostic Private Ltd. Udhna - 394210 (Surat).

3.7.2.2.6 Blood Lactic acid - Blood lactic acid was estimated according to the method of Barker and Summerson (1941). The result was expressed in mg%.

3.7.2.2.7 Blood pH - The measurement of pH of blood was done immediately after collection with the help of Systronics Digital pH Meter, type 335 (Systronics, Ahmedabad, India).

3.7.2.2.8 Serum Sodium and Potassium - These were determined by Flame photometry as described by Oser (1979). The results were expressed as meq/L.

3.8 Examination of Hormones - The level of hormones in serum samples like insulin, cortisol and thyroxine (T_4) were estimated by Radioimmunoassay method (RIA).

Before starting the experiment reagents and samples those were kept in refrigerator; brought to room temperature and mixed thoroughly before use and returned to refrigerator at 2 - 8°C immediately thereafter to minimize deterioration. In all the steps careful pipetting was done.

As per recommendation the standards and serum samples were run in duplicate. A standard curve was run with each set of clinical specimens.

3.8.1 Radioimmunoassay (RIA) method - Labelled 12 x 75 mm polypropylene radioimmune assay (RIA) tubes according to the following outline and determined the level of each hormone as stated below.

Assays tubes	Contents		
	Cortisol	Insulin	Thyroxin (T_4)
1,2	Blank	Blank	Blank
3,4	Standard (St.) 0 µg/dl	Standard(St.)0 µIu/ml	Standard(St.) 0 ng/ml
5,6	St. 1 µg/dl	St.2.5 µIU/ml	St. 10 ng/ml
7,8	St. 4 µg/dl	St. 5 µIU/ml	St. 30 ng/ml
9,10	St.10 µg/dl	St. 10 µIU/ml	St. 60 ng/ml
11,12	St.25 µg/dl	St. 25 µIU/ml	St.120 ng/ml
13,14	St.60 µg/dl	St. 50 µIU/ml	St.240 ng/ml
15,16	Serum Samples	St.100 µIU/ml	Serum Samples
17,18	Serum Samples	St.200 µIU/ml	Serum Samples
19,20	Serum Samples	Serum Samples	Serum Samples
21,22	....	....	....

3.8.1.1 Estimation of Serum Cortisol - The level of serum cortisol was estimated with the help of Cortisol Premix radioimmunoassay kit*. The value was expressed in µg/dl.

Reagents

a) Rabbit anti-cortisol serum (Cortisol Antiserum)

b) Cortisol ^{125}I Tracer Premix

c) Cortisol standards - 0 µg/dl, 1 µg/dl, 4 µg/dl, 10 µg/dl, 25 µg/dl, 60 µg/dl.

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* Cortisol Premix Radioimmunoassay kit - Bioticx Laboratories, INC, Texas - 77548.

Assay Procedure :

Step - I - Pipetted 20 µL of the standards and serum samples into appropriately lebelled assay tubes as cited before.

Step - II - Pipetted 100 µL of cortisol antiserum coloured yellow into all tubes (except tubes 1,2).

Step -III - Pipetted 1000 µL of Cortisol 125_I Tracer Premix, into all tubes and vortex. Set tubes 1 and 2 no. aside untill step 8. (Note : Shaked this reagent immediately before use).

Step - IV - Incubated all tubes at room temperature for 1 hour.

Step - V - Centrifused for 10 minutes at 1000xg.

Step - VI - Decanted carefully the supernatant from all tubes except 1 and 2 or a rack of tubes (when tubes are firmly held). Kept the tubes inverted and allowed them to drain for 5 - 10 minutes on absorbent paper. After draining, absorbed the last drop of liquid from the mouth of the tubes with absorbent paper.

Step -VII - Counted the radio-activity in all tubes in a Gamma Scintillation Counter for one minute.

3.8.1.2 Estimation of Serum Thyroxin (T_4) - The level of serum thyroxin (T_4) was estimated with the help of T_4 - Premix Radioimmunoassay Kit*. The value was expressed in ng/ml.

Reagents :

a) T_4 Antiserum.

b) T_4 Premix 125_I.

c) T_4 standards - 0 ng/ml, 10 ng/ml, 30 ng/ml, 60 ng/ml, 120 ng/ml and 240 ng/ml.

Assay Procedure :

Step - I - Added 25 µl of T_4 standard and 25 µL of the serum samples to the respective lebelled assay tubes as cited before.

Step - II - Added 1.0 ml of T_4 Premix to each tube. (Note : This reagent was shaken well immediately before use).

Step - III - Added 500 µL of T_4 Antiserum to tubes No. 3 through 14 No. and to all tubes containing clinical samples.

Step - IV - Vortex each tube.

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* T_4 - Premix Radioimmunoassay kit, Biotecx Laboratories, INC, Texas - 77033.

Step - V - Set tubes 1 and 2 aside and incubated the remaining tubes at ambient temperature for 30 minutes.

Step - VI - Centrifused the incubated tubes for 15 minutes at 3000 to 4000 rpm.

Step - VII - Decanted the supernatant by gently inverting each tube once.

Step -VIII - Counted the radioactivity in all tubes in a Gamma Scintillation Counters for one minutes.

3.8.1.3 Estimation of Serum Insulin - The level of serum insulin was estimated with the help of radioimmunoassay 125_I Insulin Kit*. The value was expressed in μIU/ml.

Reagents :

a) Dilulent Buffer - Normal guineapig serum in 0.01 M phosphosaline buffer.

b) Anti-Insulin - The antiserum was generated in guineapig.

c) Insulin standards- 2.5, 5, 10, 25, 50, 100 and 200 μIU/ml.

d) Precipitating antiserum (Second antibody) - Goat anti-guineapig gamma globulins.

e) Insulin 125_I.

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* Radioimmunoassay 125_I Insulin Kit - INC Biomedicals, INC Dianostic Division, Costa - Mesa, CA - 92626.

Assay Procedure :

Step - I - Added 0.4 ml of Diluent buffer to tubes number 1 and 2.

Step - II - Added 0.2 ml of Diluent buffer to tubes 3 and 4.

Step -III - Added 0.2 ml of each standard to tubes number 5 to 18.

Step - IV - Added 0.2 ml of serum unknowns to tubes number 19 to end of assay.

Step - V - Added 0.2 ml of Anti-Insulin to all tubes with the exception of tubes number 1 and 2.

Step -VI - Added 0.2 ml of Insulin ^{125}I to all assay tubes.

Step -VII - Mixed and incubated all tubes at 37°C for 1 hour.

Step -VIII- After incubation added 0.1 ml of second Antibody to all tubes and mixed. Incubated all tubes at room temperature for another 1 hour or more.

Step - IX - Centrifuged at 2300 - 2500 rpm for 15 minutes

Step - X - Decanted the supernatant.

Step - XI - Counted the precipitate in a Gamma Scintillation counter for one minute.

Fig.4: (Standard Curve of Serum Thyroxin)

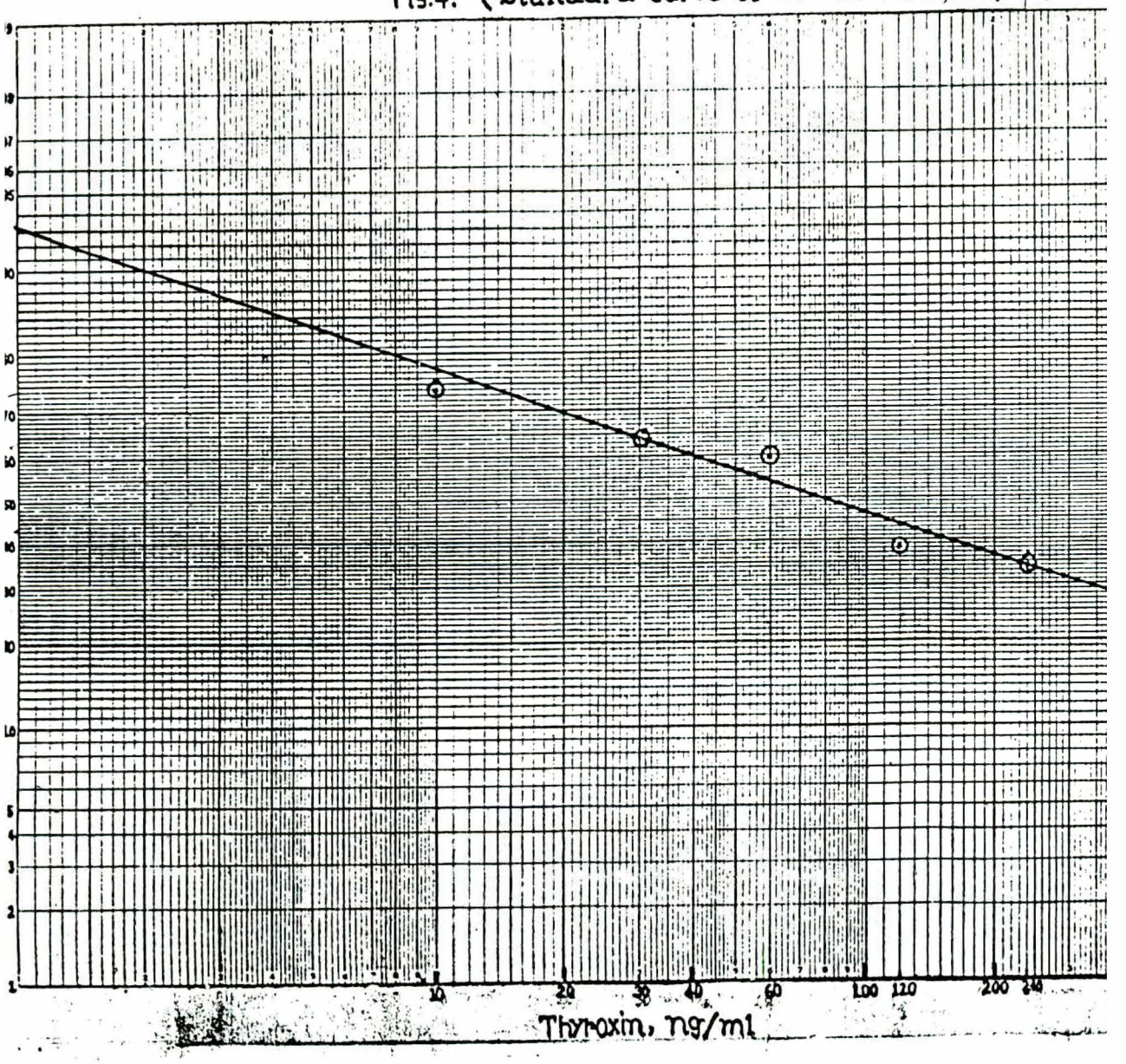


Fig.5: (Standard Curve of Serum Cortisol

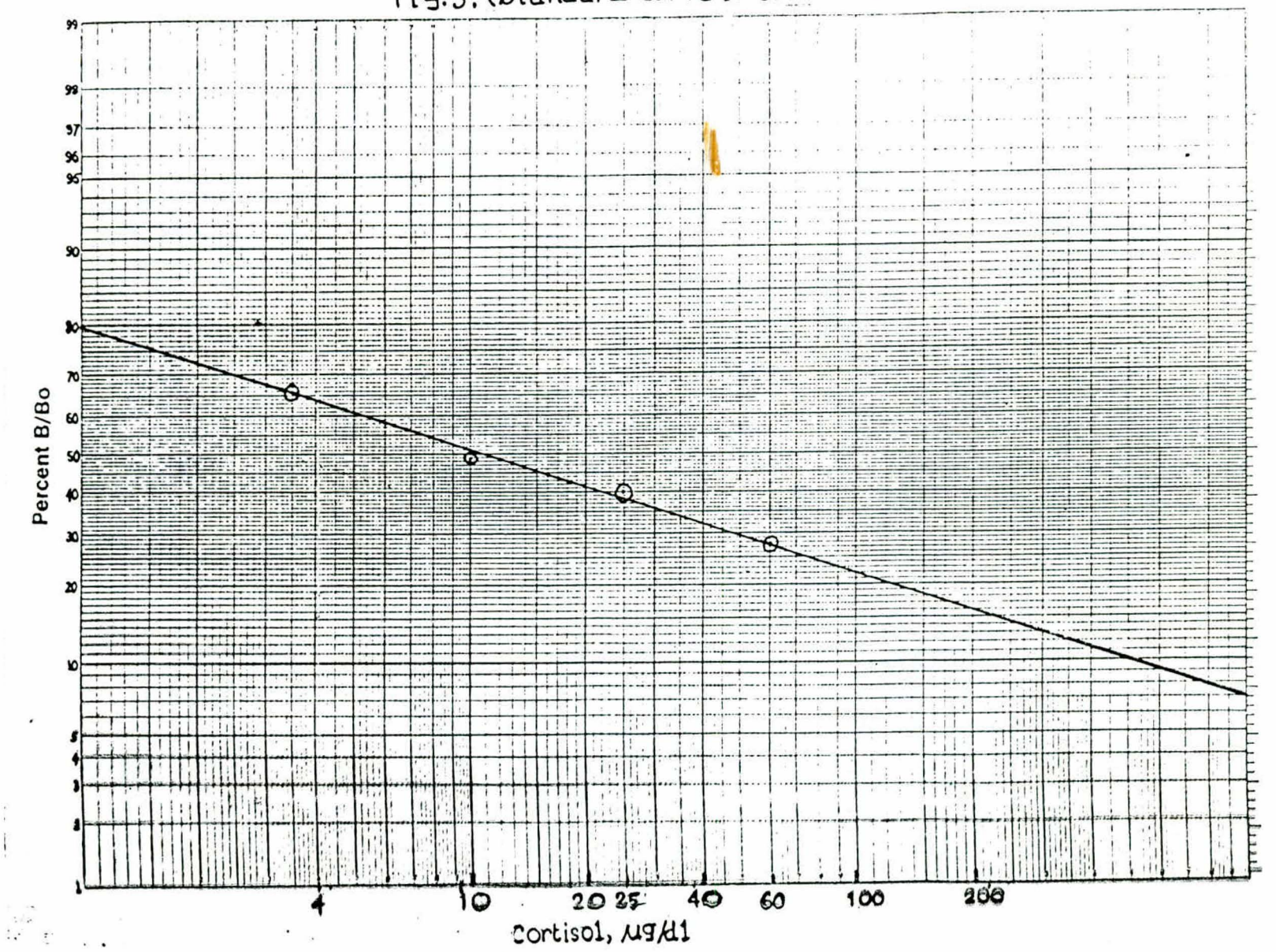
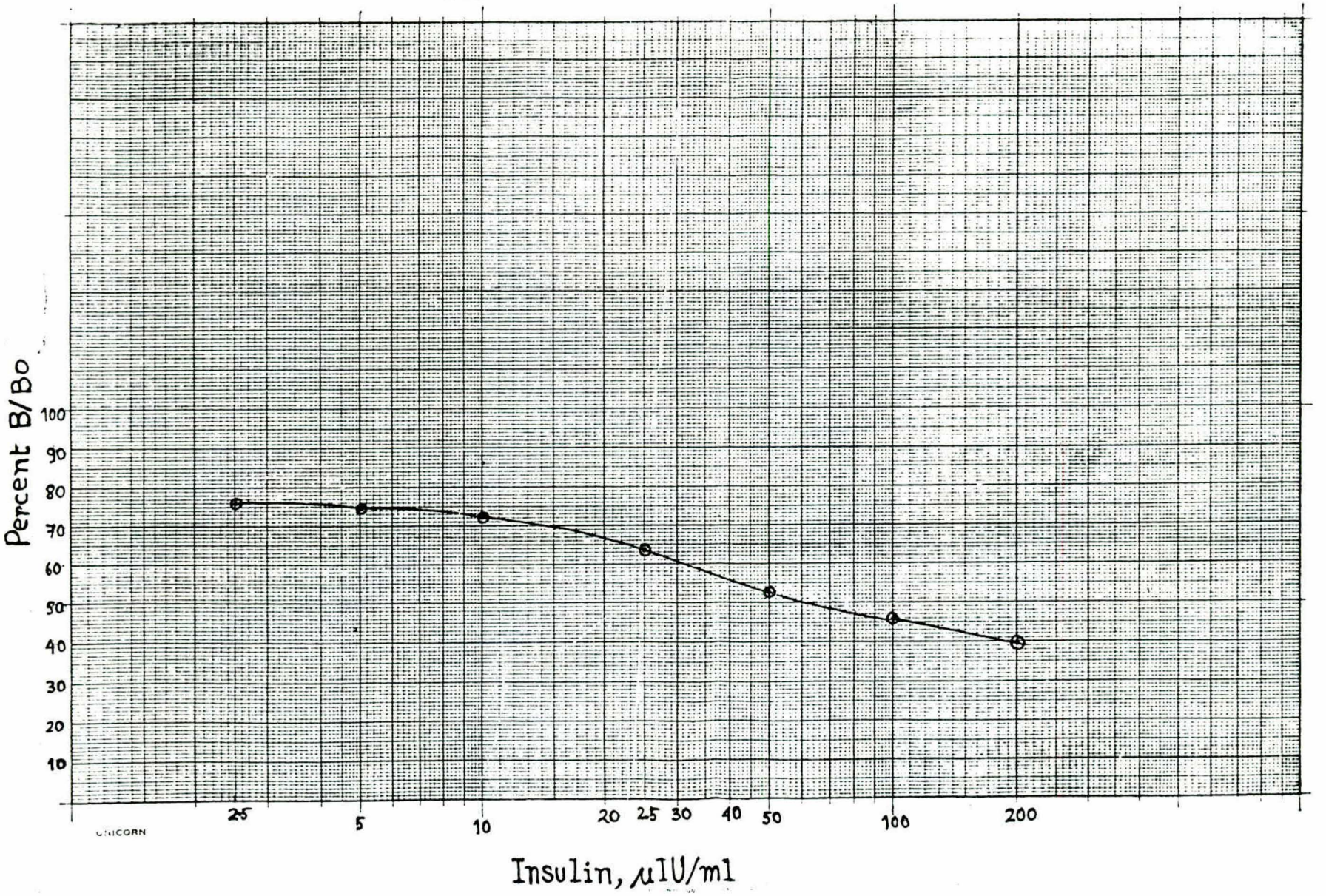


Fig.6: (Standard Curve Of Serum Insulin)



3.8.1.4 Calculation - The concentration of serum insulin, thyroxin (T_4) and cortisol were calculated as follows.

1. Calculated the average counts per minute (CPM) from duplicate samples.
2. Calculated the % B/B_o

$$\% \ B/B_o = \frac{\text{Average CPM of samples or standards - Average CPM (Blank)}}{\text{Average CPM of '0' standard - Average CPM (Blank)}} \times 100$$

3. Prepared a standard curve by plotting the % B/B_o for standard against the concentration of the standards on a linear log graph paper or logit log graph paper (Fig. 4,5 & 6).
4. The concentration of hormones of unknown samples were found out from standard curve.

3.9 Pathological Studies

3.9.1 Necrospsy Examination - Goats died during the experimental periods were immediately autopsied and gross lesions were noted after a thorough examination of all the organs like liver, kidney, rumen, intestines etc.

3.9.2 Microscopic Examination

3.9.2.1 Collection of Tissues - Tissues were collected in 10% neutral buffered formalin for histopathological examination.

3.9.2.2 Histopathological Process - Tissues were processed as per the procedure described by Griedly (1960). Formalin treated tissues were dehydrated after putting in automatic tissue processor in ascending order strength of alcohol (70%, 80%, 90%, 95%, absolute alcohol No. 1 and No. 2) for one hour in each.

Then the tissues were put in Xylene No. I and No. II each for one hour. Then tissues were placed in Xylol and paraffin mixture (equal parts) for one hour and then in paraffin No. I and No.II each for one hour. After that tissues were blocked in paraffin. Sections were cut at 5 - 6 microns thickness. Made a smear of egg albumin in glass slides. The paraffin cut sections were mounted on the smeared glass slides over a hot water bath at 80°C.

The mounted slides were air dried and then put in Xylene No. I and No. II to remove the paraffin. Slides were then passed twice through discending grades of alcohol (absolate alcohol No. I and No. II, alcohol 95%, 90%, 80% and 70%) for two minutes to remove Xylene. Excess alcohol was removed by washing the slides in running water for few minutes.

The slides were stained with haematoxylin stain by putting the slides in haematoxylin solutions for 15 minutes and then washed in running tap water. Excess haematoxylin of the sections were removed by putting the slides once or twice in acid alcohol and then washed thoroughly in running water.

Then the slides were put in eosin stain for 30 seconds. Slides were passed through 95% alcohol, absolute alcohol (absolute alcohol No. I and No. II). Finally the sections were mounted in DPX mountant and studied under microscopes.

Special stains like PAS (Periodic Acid Schiff's) for demonstration of glycogen, Pearl's stain for demonstration of haemosiderin deposits and Von-cosa stain for demonstration calcium deposits in tissues, were also used.

3.10 Therapeutic Trials

Treatment was given to the goats on the basis of ARD profiles (Anorexia, ruminal dysfunction and defaecation).

The goats of Group C received treatment at 24 hours of grain induced rumen acidosis. Goats of natural acidotic group (N) were collected from Veterinary Hospital, Bidhan Chandra Krishi Viswavidyalaya, Mohanpur and also from areas in and arround Bidhan Chandra Krishi Viswavidyalaya and were subjected to therapy.

Different schedule of treatment as adopted to treat rumen acidosis of goats were stated on next page.

3.10.1

Group 1	Rumen liquor pH 2	Experimental/ Natural 3	Treatment Schedule 4	No. of Animals treated 5
Mild acidotic	5.5 to 6.5	Experimental(C_1) and natural group (N_1)	a) Floratone bolus*. It was given intraruminally in experimental cases and orally in natural cases, @ 1 bolus twice daily for 5 days. b) Evacuation of rumen content (approximately 2/3rd of the volume) through rumen fistula in experimental cases.	15

* Floratone bolus - A product of M/s. Concept Pharmaceuticals Ltd., Bombay, containing Methionine 40 mg, Cobalt sulphate 0.88 mg, Copper sulphate 0.44 mg, Sodium phosphate (Dibasic dihydrate) 100 mg, Sodium bicarbonate 660 mg, Magnesium trisilicate 1000 mg, Gentian powder 220 mg, Ginger powder 44 mg, Vitamin B_1 145 mg, Nicotinamide 165 mg, Dried yeast 700 mg and dextrose 500 mg.

3.10.2

Group	Rumen	Experimental/ Natural	Treatment Schedule	No. of Animals treated
1	2	3	4	5
Moderate acidotic	4.5 to 5.5	Natural cases (N_2)	a) Steclin bolus*(500 mg). It was given orally @ 20 mg/kg body weight for 3 days. b) Gelusil MPS** liquid. It was given orally @ 6 t.s.f. thrice daily for 5 days. c) Injection Berin*** (10 ml). It was given intramuscularly @ 2 ml $I/_m$ daily for 5 days.	10

* Steclin bolus – A product of M/s. Sarabhai Company, Bombay, containing Tetracycline hydrochloride 500 mg per bolus.

** Gelusil MPS liquid – A product of M/s. Warner Company, Bombay, containing Methylpolysiloxane 50 mg, Dried Aluminium hydroxide gel 250 mg and Magnesium hydroxide 250 mg per 5 ml.

*** Injection Berin– A product of M/s. Glaxo Company, Bombay, containing Vit. B_1 100 mg per ml.

3.10.3

Group	Rumen liquor pH	Experimental/ Natural	Treatment Schedule	No. of animals treated
1	2	3	4	5
Severe acidotic group	4.00 to 4.5	Experimental (C_2) and natural cases (N_3)	a. Evacuation of rumen contents (Approx. 2/3rd of its volume) through rumen fistula in experimental group. b. Injection Sodium bicarbonate (7.5%)* - 25 ml. It was given intravenously @ 1 ml/kg body weight two at 1st day and once at 2nd and 3rd day. c. i. Injection Avil**(10 ml). It was given intramuscularly @ 1 ml twice daily for 3 days. ii. Avil tablet (50 mg).*** it was given intraruminally in experimental cases and orally in natural cases @ 2 tabs twice daily for 3 days. d. Steclin bolus (500 mg). It was given intraruminally in experimental and orally in natural cases @ 20 mg/kg body weight for 3 days.	12

1	2	3	4	5
			e. Injection Rintose****. It was given intravenously @ 50 ml at 12 hours interval for first day followed by half of above dose for 2nd and 3rd day.	
			f. Gelusil MPS liquid. It was given orally or intraruminally @ 6 t.s.f. thrice daily for 5 days.	
			g. Floratone bolus @ 1 bolus twice daily intraruminally in experimental group and orally in natural group for 5 days.	

* Injection Sodium Bicarbonate (7.5%) - A product of M/s. Bengal Chemicals and Pharmaceuticals Ltd., Calcutta, containing 7.5 g sodium bicarbonate per 100 ml.

**Injection Avil - A product of M/s. Hoechst Company, Bombay, containing pheniramine meleate 22.75 mg per ml.

*** Avil tablet - A product of M/s. Hoechst Company, Bombay, containing pheniramine meleate 50 mg.

**** Injection Rintose - A product of M/s. Wockhardt Company, Bombay, containing dextrose anhydrous 20 g, potchlor 0.040 g, sodium lactate 0.312 g, sodium chloride 0.600 g and dihydrate calcium-chloride 0.027 g/ 100 ml.

3.11 Statistical Methodology

3.11.1 Average Estimates of the Different Parameters

The estimates of averages of the different parameters along with the estimates of standard errors were obtained following Snedecor and Cochran (1967).

3.11.2 Analysis of Variance of different Parameters of between hours variations

For ascertaining the statistically significant variations, if any, on the different parameters due to hours, the analysis of variance was carried out adopting the model as suggested by Becker (1967).

a) Mathemetical model

$Yij = \mu + hi + eij$

Where μ = overall mean

h = hours, i = 1 .....n

eij = random error, j = 1 .....n

b) Analysis of variance table

Source of variation	d.f.	S.S.	M.S	Expected mean squares (E.M.S.)
Between hours	h-1	$\sum_i \frac{(Y_i^2)}{n_i} - \frac{Y^2}{n_i}$	MS_h	$\sigma_e^2 + K\sigma^2_h$
Within hours	n-h	$\sum_i \sum_j (Y_{ij}^2) - \sum_i \frac{(Y_i^2)}{n_i}$	MS_e	σ_e^2
Total	n-1	$\sum_i \sum_j (Y_{ij}^2) - \frac{Y^2}{n_i}$		

c) F - test

For the test of significance, the 'F' values were calculated as

$$F_{h-1,\ n-h} = \frac{\sigma_e^2 + K\sigma_h^2}{\sigma_e^2}$$ to test between hours M.S.

d) Critical difference (C.D.) tests

To test the differences between the hour's mean, critical difference test at 5% level of probability were made by using the following formula.

$$C.D. = t_{0.05}\sqrt{\frac{1}{K_1} + \frac{1}{K_2}\ \sigma_e^2}$$

Where, σ_e^2 is the error means square, K_1 and K_2 are the number of observations of the two groups (hours) to be compared and $t_{0.05}$ is a factor entered from t - table for n-h d.f. at 0.05 percent level of probability.

C H A P T E R - IV

RESULTS AND DISCUSSION

4.0 RESULTS AND DISCUSSION

4.1 Observations on Healthy Control goats (Group A)

4.1.1 Clinical accounts on healthy control goats are presented in Table - 1.

Table-1. Clinical accounts and observations on healthy control goats (Group A)

Parameters (6)		Mean ± S.E.
Rumen motility rate/5 minutes		7.16 ± 0.20
Rumination	The animals ruminated many times in a day during resting period.	
Abdominal condition	Abdomen showed normal size. On percussion no abnormality was detected.	
Defaecation	All animals passed pelleted faeces, characteristics for the species.	
Dehydration	Absence of retention of skin fold/sec indicated no dehydration.	
Urination	All animals urinated 5 - 9 times a day.	
pH of urine		8.1 ± 0.308
pH of faeces		7.28 ± 0.16
Heart rate/minute		70.66 ± 0.98
Rectal temperature ($0^{o}F$)		102.94 ± 0.085
Respiration rate/minute		18.16 ± 0.575

Figure in the parenthesis indicates number of animals.

It reveals from the table that mean rumen motility rate, pH of urine, pH of faeces, heart rate, respiration rate and rectal temperature of healthy control goats were 7.16 ± 0.20/5 minutes, 8.1 ± 0.308, 7.28 ± 0.16, 70.66 ± 0.98/minute, 18.16 ± 0.575/minute and 102.94 ± 0.085°F respectively. All animals ruminated number of times in a day, urinated 5 - 9 times in a day and had normal abdominal condition, defaecation with no sign of dehydration.

All animals grazed whole day, drunk water ad libitum which showed good appetite. They had normal posture and gait with glossy body coat. They were active and alert. The muzzles looked moist. Conjunctival mucous membranes were pale pink to rosy in colour. These findings were similar to that of Kelly (1974) and Chakrabarti (1988).

The animals ruminated several times in a day during resting period. Abdomens were normal in size and did not show abnormality on percussion. The mean ruminal motility rates were 7.16 ± 20/5 minutes. These findings were similar to that of Kelly (1974) and Rai and Pandey (1980 b).

The goats of this group urinated 5 - 9 times a day. This findings did not show any similarity with that of Kelly (1974) and Rai and Pandey (1980 b). The Variation of frequency of urination might be depended on the quantity of water and other fluid intake, environmental condition, diet, and the size and physical activity of the animal (Chakrabarti, 1988). It might depend on the amount of water lost by respiration and defaecation (Das, 1990). The mean pH of the urine was 8.1 ± 0.308 which agreed with the findings of Chakrabarti (1988).

All animals of this group defaecated pelleted faeces with dark green colour which agree with the observations of Chakrabarti (1988). The dark green colour of the faeces indicated that the animals were grass eater. The average pH of the faeces was 7.28 ± 0.16.

The mean values of normal heart rate, respiration rate and rectal temperature were 70.66 ± 0.98/minute, 18.16 ± 0.575/minute and 102.94 ± 0.085°F respectively. These findings were close to the values stated by Kelly (1974) and Chakrabarti (1988) in normal goats.

4.1.2 Observations on Rumen Liquor

4.1.2.1 Macroscopic

Macroscopic observations of rumen liquor of healthy control goats are presented in Table 2.

Table - 2. Macroscopic observations of rumen liquor in healthy control goats (Group A)

Parameters (6)	Mean ± S.E.
Smell	Aromatic
Consistency	Semiliquid or viscous
Colour	Pure green to greenish brown
SAT (min.)	26.54 ± 1.02
CDT (hrs.)	44.11 ± 2.52
MBRT(min.)	10.29 ± 0.309

Figure in the parenthesis indicates number of animals.

The above table showed that mean SAT, CDT and MBRT of rumen liquor of healthy control goats were 26.54 ± 1.02 minutes, 44.11 ± 2.52 hours and 10.29 ± 0.309 minutes respectively. The smell, consistency and colour of rumen liquor were aromatic, semiliquid or viscous and pure green to greenish brown respectively.

4.1.2.1.1 Smell

The smell of rumen liquor was aromatic. This findings agreed with the observations of Chakrabarti (1988). Pradhan et al. (1988) and Lal et al. (1989). However, smell of rumen liquor varied with the types of feed ingested (Rosenberger, 1979) and types of indigestion (Chakrabarti,1988).

4.1.2.1.2 Consistency

The consistency of rumen liquor was semiliquid or viscous. Similar observation was recorded by Sen (1982), Pradhan et al. (1988) and Lal et al. (1989) in goats. The variations of consistency of rumen liquor might be depended upon the types of feed offered (Rosenberger, 1979)

4.1.2.1.3 Colour

The colour of the rumen liquor was pure green to greenish brown. This finding was in conformity with the observation of Sen (1982), Sen et al. (1982), Pradhan et al. (1988) and Lal et al. (1989) in goats. This finding did not coinside with that of Misra and Singh (1974). The variation might be due to type of feed (Chakrabarti, 1988; Rosenberger, 1979) and types of indigestion (Chakrabarti, 1988).

4.1.2.1.4 Cellulose digestion time (CDT), Sediment activity time (SAT) and Methylene Blue reduction time (MBRT)

The average CDT of rumen liquor was 44.11 ± 2.52 hours which was nearer to the findings of Hoflund et al. (1948). The variation might be due to individual, species variation and types of feed intake. The average SAT of rumen liquor was 26.54 ± 1.02 minutes which coinsided with the observation of Sen (1982) and Pradhan et al. (1988) in goats but did not coinside with the findings of Nichols and Penn (1958) and Blood et al. (1983) in cattle which showed lower SAT value. This variation was due to individual and species variation (Nichols and Penn, 1958). The average MBRT of rumen liquor was 10.29 ± 0.309 which was similar to the observations of Pradhan et al. (1988).

4.1.2.2 Microscopic

Microscopic observations of rumen liquor of healthy control goats are presented in Table - 3.

Table - 3. Microscopic observations of rumen liquor in healthy control goats (Group A).

Parameters (6)	Mean ± S.E.
Concentration of rumen protozoa	+++
Motility of rumen protozoa	+++
Total protozoal count (10^4/ml)	39.36 ± 9.07
Iodophilic activity of rumen protozoa	++/+++
Total bacterial count (10^9/ml)	15.07 ± 1.34
Types of bacteria	Predominance of Gram negative Cocci and rods.

Figure in the parenthesis indicates number of animals.

Motility of protozoa	Iodophilic nature and concentration of protozoa
- = Absent	- = Nil
+ = Slow	+ = Low
++ = Moderate	++ = Moderate
+++ = Vigorous	+++ = High

It reveals from the table that concentration, iodophilic activity and motility of rumen protozoa of healthy control goats were high (+++), moderate to high (++/+++) and vigorous (+++) respectively. Total protozoal count and total bacterial count of strained rumen liquor were $39.36 \pm 9.07 \times 10^4$/ml and $15.07 \pm 1.34 \times 10^9$/ml. The type of bacteria of rumen liquor was always predominated by Gram negative Cocci and rods.

4.1.2.2.1 Concentration, Iodophilic nature and Motility of rumen protozoa.

The concentration, iodophilic nature and motility of rumen protozoa were +++ (high), ++/+++ (moderate to high) and +++ (Vigorous) respectively which were nearer to the findings of 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 species variations, individual variation, difference in nature and quality of feed intake and time of sampling (Purser and Moir, 1966; Rosenberger, 1979).

4.1.2.2.2 Total Protozoal count of rumen liquor

The average total protozoal count of rumen liquor of healthy control group of goats was $39.36 \pm 9.07 \times 10^4$/ml which

was nearer to the findings of Rai et al. (1972) and Rai and Pandey (1980 b) in goats. Variation in total protozoal count might be due to species variation and individual variation (Purser and Moir, 1966; Rosenberger, 1979). However, Rai et al. (1972) reported that variation in total protozoal count in rumen liquor was depended on time of sampling and types of feed intake.

4.1.2.2.3 Total Bacterial count of rumen liquor

The average total bacterial count of rumen liquor was 15.07 $\pm$ 1.34 x 10^9/ml which was similar or nearer to the findings of Rai et al. (1972) and Lal et al. (1989) in goats and Randhawa et al. (1989) in buffalo calves. The variation depended on management system i.e. stall fed animals or grazing animals (Rai and Pandey, 1980 b).

4.1.2.2.4 Type of Bacteria in rumen liquor

Type of bacteria in rumen liquor was gram negative cocci and rods. The predominance of Gram negative flora as compared to Gram positive bacteria in the rumen liquor of healthy control goats might be due to the hydrogen ion concentration of rumen fluid, which provides an optimal environment for their growth and multiplication. Similar findings was reported earlier by Hungate et al. (1952); Krogh (1963 a,b); Mann (1970); Allison et al. (1972) and Vazquez (1976).

4.1.2.3 Biochemical analysis

Biochemical analysis of rumen liquor of healthy control goats (Group A) is presented in Table - 4.

Table-4. Biochemical analysis of rumen liquor of healthy control goats (Group A)

Parameters (6)	Mean ± S.E.
pH	6.98 ± 0.03
Lactic acid (mg/100 ml)	7.09 ± 0.30
TVFA (mEq/L)	66.33 ± 1.98
NH_3-N (mg/100 ml)	14.80 ± 1.51

Figure in the parenthesis indicates numbers of animals.

It reveals from the table that average of pH, lactic acid, TVFA, NH_3-N concentration of rumen liquor of healthy control goats were 6.98 ± 0.03, 7.09 ± 0.30 mg/100 ml, 66.33 ± 1.98 mEq/L and 14.80 ± 1.51 mg/100 ml respectively.

4.1.2.3.1 pH of Rumen liquor

The average pH of rumen liquor was 6.98 ± 0.03 in healthy control goats. It was in close agreement with the earlier reports of Sen (1982), Vihan et al. (1982), Tanwar and Mathur (1983 a), 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). Quality of feed used to influence the pH of rumen liquor (Rai and Pandey, 1980 a).

4.1.2.3.2 Lactic acid

The mean lactic acid level in rumen liquor of healthy control goats was 7.09 ± 0.30 mg/100 ml. This observation was nearer to the earlier observations recorded by Verma et al. (1975), Sen (1982), Lal et al, (1989) and Das (1990) in goats. The slight variation in level of lactic acid in rumen liquor within normal range occured in ruminants maintained with grain feeding (Balch and Rowland, 1957; Ghorban et al., 1966). Variation in level was also obtained just after wetering of the animal (Verma et al., 1975).

4.1.2.3.3 Total Volatile fatty acids (TVFA)

The mean TVFA concentration in rumen liquor of healthy control goats was 66.33 ± 1.98 mEq/L. This was nearer to the values reported earlier by Rai et al. (1972), Verma et al. (1975), Rai and Pandey (1980 a), Sen (1982), Lal et al. (1989) and Das (1990). Slight variation of TVFA concentration in the rumen liquor within the normal range appeared to be due to breed variation and variation in time of collection of sample (Reddy and Nair, 1971; Verma et al., 1975) and feed (Rai and Pandey, 1980 a). Decreased concentration of TVFA in rumen liquor also obtained just after watering the animal (Verma et al., 1975).

4.1.2.3.4 Ammonia nitrogen (NH_3-N)

The mean concentration of NH_3-N in rumen liquor of healthy control goats was 14.80 ± 1.51 mg/100 ml which was close to the findings of Rai *et al.* (1972), Rai and Pandey (1980 b) in goats and Randhawa *et al.* (1989) in buffalo calves. Slight alteration of values were due to time of sampling and type of feed intake (Rai *et al.*, 1972; Rai and Pandey, 1980 b).

4.1.3 Blood analysis

4.1.3.1 Haematological examination

The haematological examination of healthy control goats (Group A) is presented in Table - 5.

Table-5. Observations on haematological studies of healthy control goats (Group A)

Parameters (6)	Mean ± S.E.
PCV (%)	23.08 ± 0.288
Hb (g/100 ml)	8.80 ± 0.176
TEC (10^6/cmm)	8.59 ± 0.29
TLC (10^3/cmm)	5.95 ± 0.364
Neutrophils (%)	41.84 ± 1.62
i) Segmented Neutrophils (mature) (%)	41.84 ± 1.62
ii) Neutrophils (Band) (%)	0.00 (nil)
iii) Metamyelocytes (%)	0.00 (nil)
Lymphocytes (%)	53.00 ± 1.50
Monocytes (%)	2.00 ± 0.258
Eosinophils (%)	3.16 ± 0.40
Basophils (%)	0.13 ± 0.08

Figure in the parenthesis indicates numbers of animal.

The table showed that average values of PCV percentage, Hb percentage, TEC, TLC, segmented neutrophils (mature) percentage, lymphocytes percentage, monocytes percentage and eosinophils percentage of healthy control goats were 23.08 ± 0.288, 8.80 ± 0.176 g/100 ml, 8.59 ± 0.29 x 10^6/cmm., 5.95 ± 0.364 x 10^3/cmm., 41.84 ± 1.62, 53.00 ± 1.50, 2.00 ± 0.258 and 3.16 ± 0.40 respectively. But, percentage of neutrophilic band cells, metamyelocytes and basophil cells were nil, nil and 0.13 ± 0.08.

4.1.3.1.1 Packed cell volume (PCV)

The mean PCV value of healthy control goats was 23.08 ± 0.288 percent which was close to the observations of Sen (1982),Sastry (1983), Tanwar and Mathur (1983 a), Schalm et al. (1986) and Mohi et al. (1990) in healthy goats. However, a slightly higher values were recorded by Das (1990) in goats. Slight variation in normal values might be due to species and breed variation and variations of general condition (Sen,1990). Variation between sexes (Kundu et al. 1991) and between seasons (Mohi et al.1990) were also reported.

4.1.3.1.2 Haemoglobin (Hb)

The average value of Hb of healthy control goats was 8.80 ± 0.176 g/100 ml. This observation was within the range of the observations of Sastry (1983) and Mohi et al. (1990) in goats. Slight deviation in normal values might be due to variations of species, breed, sex and general condition of animals. Variation due to season was also reported by Mohi et al. (1990).

4.1.3.1.3 Total Erythrocytic count (TEC)

The mean value of TEC of healthy control goat was 8.59 ± 0.29 x 10^6/cmm. This value was within the range of normal value reported by Sastry (1983) in goats. However, Chakrabarti (1988) and Mohi et al. (1990) reported higher values than those seen by the above workers. This difference might be due to sepcies variation, sex variation (Kundu et al. 1991), Seasonal variation (Mohi et al., 1990) and variations of general condition of the animals. Mohi et al. (1990) reported lower TEC in summer season.

4.1.3.1.4 Total Leukocytic count (TLC)

The mean valu TLC of healthy control goat was 5.95 ± 0.364 x 10^3/cmm. which was within the range of values reported by Sastry (1983), Chakrabarti (1988) and Mohi et al. (1990). Slight differences might be due to variation in status of health of the animals.

4.1.3.1.5 Differential Leukocytic count

The mean values of percentage of segmented neutrophils (mature), lymphocytes, monocytes, eosinophils and basophils were 41.84 ± 1.62, 53.00 ± 1.50, 2.00 ± 0.258, 3.16 ± 0.40 and 0.13 ± 0.08 which were within the normal range of values reported by Sastry (1983), Schalm et al. (1986) and Chakrabarti (1988). The percentages of metamyelocytes (immature neutrophils) and neutrophil band cells were nil which coincided with the similar observations reported by Schalm et al. (1986) in normal goats.

4.1.3.2 Biochemical examination of Serum/Blood

The biochemical analysis of serum/blood of healthy control goats (Group A) is presented in Table - 6.

Table - 6. Observations on biochemical analysis of serum/blood of healthy control goats (Group A)

Parameters (6)	Mean ± S.E.
Blood pH	7.44 ± 0.013
Blood glucose (mg/100 ml)	53.5 ± 0.85
Blood lactic acid (mg/100 ml)	12.31 ± 0.684
Serum calcium (mg/100 ml)	11.70 ± 0.256
Serum phosphorus (mg/100 ml)	5.31 ± 0.468
Serum total protein (g/100 ml)	6.45 ± 0.10
Serum albumin (g/100 ml)	3.97 ± 0.055
Serum globulin (g/100 ml)	2.48 ± 0.066
Albumin : Globulin (A/G) ratio	1.615 ± 0.040
Blood urea (mg/100 ml)	20.85 ± 0.88
Serum creatinine (mg/100 ml)	1.03 ± 0.041
Serum sodium (mEq/L)	140.75 ± 2.70
Serum potassium (mEq/L)	3.94 ± 0.12

Figure in the parenthesis indicates number of animals

It reveals from the table that mean blood pH of healthy control goats was 7.44 ± 0.013. The mean concentration of blood glucose, blood lactic, serum calcium, serum phosphorus, serum total protein, serum albumin, serum globulin, blood urea, serum creatinine were 53.5 ± 0.85 mg,

12.31 ± 0.684 mg, 11.70 ± 0.256 mg, 5.31 ± 0.468 mg, 6.45 ± 0.10 g, 3.97 ± 0.055 g, 2.48 ± 0.066 g, 20.85 ± 0.88 mg and 1.03 ± 0.041 mg per 100 ml respectively. The A/G ratio was 1.615 ± 0.040 and mean serum sodium and serum potassium concentration were 140.75 ± 2.70 and 3.94 ± 0.12 mEq/L respectively.

4.1.3.2.1 Blood pH

The mean blood pH of healthy control goats was 7.44 ± 0.013. This observation was close to the observations of Sen (1982), Vihan *et al.* (1982), Tanwar *et al.*(1983) and Cao *et al.*(1987). Mild variation in blood pH might be due to variations of species, breed, feed, method of collection and time of examination.

4.1.3.2.2 Blood Glucose

The mean blood glucose level of healthy control goats was 53.5 ± 0.85 mg/100 ml. This observation agreed with the findings of Melvin (1977), Sen (1982), Sastry (1983) and Das (1990) 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).

4.1.3.2.3 Blood Lactic acid

The mean lactic acid value in blood of the healthy control group of goats was 12.31 ± 0.684 mg/100 ml. Present findings was in accordance with the results of Verma et al. (1975), Sen (1982), Tanwar et al. (1983), Cao et al. (1987), Das (1990) in goats. Slight variation in level of lactic acid in normal animal might be due to different times of sampling and concentrate feeding (Verma et al., 1975).

4.1.3.2.4 Serum Calcium

The mean serum calcium value of healthy control goats was 11.70 ± 0.256 mg/100 ml. The present finding was in accordance with the results of Kaneko and Cornelius (1970), Melvin (1977) and Sastry (1983) in normal goat. However, Vihan et al. (1982) observed higher values in control goats in experimental rumen acidosis. This difference might be due to breed variation, general status of health of the animal and type of dietary feed supplement. Kaneko and Cornelius (1970) reported low status of calcium with increasing age due to poor calcium absorption.

4.1.3.2.5 Serum inorganic Phosphorus

The mean serum phosphorus level of healthy control group of goats was 5.31 ± 0.468 mg/100 ml and was close to the findings of Kaneko and Cornelius (1970), Melvin (1977), Vihan et al. (1982) and Sastry (1983).

4.1.3.2.6 Serum total Protein, Albumin, Globulin and Albumin : Globulin ratio

The observed mean values of serum total protein, albumin, globulin and albumin : globulin ratio in healthy control goats were 6.45 ± 0.10, 3.97 ± 0.055, 2.48 ± 0.066 g/100 ml and 1.615 ± 0.040. Present observations were similar to the observations of Gorcayca et al. (1960), Altman and Dittmer (1961), Sastry (1983) and Cao et al. (1987) in goat. However, Vihan et al. (1982) reported higher values in control goats in experimental rumen acidosis. Slight variation of normal values might be depended on age (Irfan, 1967), sex, breed, species and status of animal health and types of feed supplemented.

4.1.3.2.7 Blood Urea

The average concentration of blood urea in healthy control group of goats was 20.85 ± 0.88 mg/100 ml. This was within the normal range. Similar observation was reported by Rai et al. (1972), Melvin (1977) and Sastry (1983) in goats. Mild deviation within normal range might be due to intake of digestible crude protein (Rai et al., 1972), species and breed variation.

4.1.3.2.8 Serum Creatinine

The average concentration of serum creatinine level in healthy control group of goats was 1.03 ± 0.041mg/100 ml which was within normal range as reported previously by Melvin (1977) and Sastry (1983) in goat. Slight deviation within physiological

limit might be depended on excercise, urine volume, species, sex, breed and general condition of the animal.

4.1.3.2.9 Sodium and Pottasium

Observed value of mean sodium concentration in serum of the healthy control group of goats was 140.75 ± 2.70 mEq/L. Findings were nearer to the finding of Sen (1982), Das (1990) in goat and Irwin et al. (1979) in sheep. The mean concentration of potassium in serum was 3.94 +_0.12 mEq/L which was close to the previous findings reported by Sen (1982) and Das (1990).

4.1.3.3 Analysis of serum hormonal level of healthy control goats (Group A)

The analysis of level of different serum hormones is presented in table - 7.

Table - 7. Observations on concentration of serum cortisol, insulin and thyroxin of healthy control goats (Group A).

Parameters (6)	Mean ± S.E.
Cortisol (µg/100 ml)	BDL (/1)
Insulin (µIU/ml)	10.70 ± 1.20
Thyroxin (ng/ml)	44.11 ± 2.68

BDL = Below detection level (/1 µg/100 ml)

Figure in the parenthesis indicates No. of animals.

It reveals from the table that mean concentration of serum cortisol, insulin and thyroxin in healthy control goats were BDL (/1 µg/100 ml), 10.70 ± 1.20 µIU/ml and 44.11 ± 2.68 ng/ml.

4.1.2.3.1 Serum Cortisol

The mean serum cortisol concentration of healthy control goats was below detection level (BDL) i.e. /1 µg/100 ml. The observation was close to the observation of Linder (1967) in goats. However, slight variation in the serum cortisol status appeared to be due to age and breed variation.

4.1.3.3.2 Serum Insulin

The mean concentration of serum insulin of healthy control goats was 10.70 ± 1.2 uIU/ml. This observation was very close to the observation of Pearson et al. (1977) in adult goats. But, this observation did not coincide with that of Horino (1968) in adult sheep and Walket and Elliot (1973) in adult cow. The variation in the insulin concentration appeared to be due to age and species variation. Individual variations might be due to status of glucose level in blood. After feeding glucose level was increased which stimulated insulin secretion.

4.1.3.3.3 Serum Thyroxin (T_4)

The average concentration of serum thyroxin in healthy control goat was 44.11 ± 2.68 ng/ml. This value

agreed with the previous findings as reported by Kallfelz and Erali (1973) and Reap et al. (1978) in goat. Slight variation might be due to breed, sex, age, environmental temperature, exposure to light and status of the animal's health.

4.2 Observations on goats after induction of rumen acidosis (Group B)

4.2.1 Clinical Accounts

It was found that about 16.66% of experimental goats died within 36 hours, 33.33% died within 60 hours, 50% died within 72 hours, 66.66% died within 96 hours and 83.33% within 108 hours after intraruminal administration of crushed rice at the dose of 70 g/kg body weight. Incidence of death due to lactic acidosis was also reported previously by Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983 a) and Das (1990) in goats. The cause of death might be due to toxaemia and dehydration which caused cardio-respiratory failure and hepato-renal failure. Acute carbohydrate overload cause secondary paralysis of respiratory centre due to accumulation of Co_2 (Castello, 1968).

The variation of percentage of death in different hours might be due to the individual variation in the bio-chemical changes in blood and ruminal fluid in that particular animal as reported by Dougherty et al. (1975 b). Slyter (1976) reported that individual animal differs in respect of salivation, ingesta fill, intestinal motility and ability to excrete or use large quantities of potentially toxic compound. Blood

et al. (1983) reported that in untreated cases of grain induced acidosis the percentage of mortality may go upto 90%.

4.2.1.1 Clinical manifestations

The clinical manifestations varied among the animals based on degree of acidosis produced in them at the rate of 70 g/kg body weight. This might be due to individual variation (Slyter, 1976). The characteristic symptoms were observed after 24 hours of induction of acidosis. The goats which were examined clinically within 12 hours after engorgement; the only abnormality detected was a full rumen and occasional abdominal pain evidenced by kicking at the belly.

The goats which showed anorexia, semisolid faeces and absence of rumination along with fairly bright and alert look could last for longer duration, indicating mild to moderate form of acidosis.

Severe dullness/depression, head pressing, dyspnoea, loss of appetite, occasional grinding of teeth, mucoid to purulent nasal discharge, wobbling gait, excessive thirst, shunken eyes, dropping head, disinclination to move, reluctant to stand, reduced skin elasticity, injected mucous membranes, constipation in maximum cases, occasional diarrhoea with undigested crushed rice, drooling of saliva and excessive thirst, all indicated severe form of acidosis. The affected animals died within 24 to 48 hours. Before death animals were in lateral recumbency with head turned towards the flank and did not respond to stimuli indicated comatose condition simulating milk fever posture.

The clinical symptoms as observed after the experimental induction of ruminal acidosis in goats were similar to those described by Sen (1982), Sen et al. (1982), Tanwar and Mathur (1983 a), Cao et al. (1987), Lal et al. (1989), Das (1990) in experimental goats and by Gnanaprakasam (1970) in natural cases of goats.

Excessive production of lactic acid in the rumen give rise to variety of clinical manifestations ranging from temporary inappetence to death. Clinical symptoms resulting from ruminal accumulation and subsequent absorption of lactic acid include rumen statis, diarrhoea, dehydration, systemic acidosis and in acute form cardiovascular and respiratory failure (Huber, 1976).

Loss of appetite or inappetance may be due to lactiac-edemia as a result of derangement in the normal rumen metabolism. However, Phillipson (1955) suggested that it may be due to decreased flora. Hungate (1966) reported that appetite might be linked with microbial fermentation activity. Nervous symptoms like dullness, depression, head pressing, wobbling gait are due to toxic effect of lactic acid and alcohol poisoning on brain (Juha'sz and Szegedi, 1968 b) or due to polioencephalomacia owing to thiamine (Vitamin - B_1) antagonism (Brent, 1976).

Diarrhoea observed in some goats might be due to hypertonicity and subsequent vascular drainage (Ahrens, 1967). Constipation as observed in maximum cases might be due to severe gut atony.

Symptoms like shunken eye balls, reduced skin elasticity, thirst, comatosed condition, injected mucous membrane are indicative of severe dehydration and haemoconcentration due to increased osmotic pressure of rumen fluid resulting into passage of water from blood into the rumen (Parthasarathy and Phillipson, 1953). Reluctance to stand and unsteady gait may be due to laminitis caused by histamin release from damage tissues as described by Dirksen (1970). Drooling of saliva may be due to lack of rumination.

Mucopurulent nasal discharge might be due to secondary bacterial infection of respiratory system.

Grinding of teeth might be due to toxic effect of lactiacedemia (Lal et al., 1989).

4.2.1.2 Clinical observations

The mean with standard error as well as the result of the test for the mean difference of different clinical observations of acidotic goats (Group B) at different hours are shown in the Table - 8. The results of analysis of variance of the different clinical observations between hours are also presented in Table - 9.

4.2.1.2.1 Rumen motility rate

The average rumen contraction rate was 7.5 $\pm$ 0.428/5 minutes at '0' hour. There was no rumen contraction from 12th hour onwards. However, one animal had shown sluggish rumen contraction at the rate of 1/5 minutes at 120th hour. Present

Table – 8. Mean with standard error of different parameters on clinical observations of acidotic goats(Group 8)

Parameters \ Hours	0 (6)	12 (6)	24 (6)	36 (5)	42 (5)	48 (5)	60 (4)	72 (3)	84 (3)	96(2)	108(1)	120(1)
Rumen motility rate/5 minutes	7.50 ±0.428	-	-	-	-	-	-	-	-	-	½	-
Rumination	Rumina-ting	-	-	-	-	-	-	-	-	-	-	-
Abdominal size, shape and condition	Heavy and full abdomen, felt firm and doughy within 12th to 18th hours. Distended, heavy abdomen and accumulation of excessive fluid in the rumen on combined palpation and percussion from 24 hours onwards. Gurling sound on ausculation over rumen.											
Defaecation	Pelleted to pasty faeces with undigested crushed rice in most of the cases. Occasional diarrhoea between 42th hour to 60th hour.											
Dehydration (Skin fold test in Sec.)	-- No dehydration ---------10% dehydration ---------- -------8% dehydration ------- —6-7%dehydration											
Urination	All animals urinated 4-6 times a day upto 12th hour. Afterwards frequency and quantity decreased with high colour. Anuria between 60th hour to 84th hour.											
pH of urine	8.21[a] ±0.13	7.83[b] ±0.125	6.82[c] ±0.101	5.80[d] ±0.173	ND	5.06[e] ±0.068	NA	NA	NA	6.75[c] ±0.25	7.00	7.20

Contd ....123...

Parameters \ Hours	0 (6)	12 (6)	24 (6)	36(5)	42 (5)	48 (5)	60 (4)	72 (3)	84 (3)	96(2)	108(1)	120(1)
pH of faeces	7.41^{a} ±0.27	7.16^{af} ±0.33	6.50^{fb} ±0.22	5.30^{cd} ±0.20	ND	4.60^{c} ±0.25	5.12^{ce} ±0.13	5.16^{b} ±0.17	ND	6.00^{bde} ±0.00	5.00	5.20
Heart rate/minute	72.33^{a} ±1.33	123.66^{b} ±2.1	135.83^{ce} ±1.99	140.00^{cd} ±0.70	140.00^{cg} ±0.38	142.00^{dg} ±0.708	132.5^{eh} ±2.53	129.66^{h} ±1.2	126.33^{bh} ±1.66	116.00^{f} ±5.01	110.00	110.00
Rectal temperature(^{0}F)	102.50^{ahe} ±0.179	103.01^{b} ±0.202	102.00^{ci} ±0.057	101.40^{d} ±0.107	101.42^{di} ±0.096	102.15^{ch} ±0.169	102.80^{be} ±0.15	102.25^{gc} ±0.233	102.00^{cf} ±0.152	99.95^{g} ±0.15	100.20	100.20
Respiration rate/ minute	17.83^{a} ±0.98	19.33^{a} ±0.88	21.50^{bc} ± 0.76	22.20^{bfg} ±0.66	23.20^{boeh} ±0.862	25.20^{eh} ±0.376	23.50^{be} ±0.286	24.00^{f} ±0.578	23.00^{bde} ±0.578	20.50^{acdg} ±0.496	20.00	20.00

-- = Absent

ND = Not done

NA = Not available

Figures in the parentheses indicate the number of animals

Means with same superscripts in the same row do not differ significantly.

Table - 9. Analysis of variance of different parameters on clinical observation in acidotic goats (Group B)

Parameters	Source of variations			
	Between hours		Error	
	d.f.	M. S.	d.f.	M. S.
pH of urine	5	7.71**	24	0.088
pH of faeces	7	5.20**	29	0.324
Heart rate/minute	9	2341.36**	35	17.61
Rectal temperature	9	2.4**	35	0.113
Respiration rate/minute	9	25.96**	35	2.90

(** P/0.01)

observation was similar to the observations of Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983 a), Lal et al. (1989), Das (1990) in goats.

Atony of the rumen might be due to accumulation of toxic products like histamine, lactic acid, other amines (Huber, 1976, Miert et al., 1976), butyric acid (Blood et al., 1983) ethanol, methanol and endotoxin in the rumen. This histamine is released from damage tissues of rumen and also after conversion of histidine to histamine by Lactobacillus (Rowdell, 1953 a,b). Slight rumen contraction at 120th hour in one goat might be due to temporary improvement of rumen environment.

4.2.1.2.2 Rumination

The acidotic goats ruminated at '0' hour of induction of rumen acidosis and did not ruminate from 12th hour onward, This finding was similar to the observations of Sen (1982), Tanwar and Mathur (1983 a) and Das (1990) in goat. Lack of rumination might be due to failure of muscular contractions of rumen, reticulum due to accumulation of lactic acid.

4.2.1.2.3 Abdomen size, shape and condition

Full, firm and doughy abdomen was observed within 12th to 18th hour. Distended, heavy abdomen and accumulation of excessive fluid in the rumen on combined palpation and percussion was observed from 24th hour onward. Occassional gurgling sound was also observed on auscultation over rumen in some goats. These findings were in accordance with those

described by Gnanaprakasam (1970), Sen (1982) and Das (1990) in goats.

In the initial stage firm and doughy condition of the rumen might be due to accumulation of excessive solid mass of crushed rice impacted in the rumen. The excessive accumulation of fluid in the rumen in later stage might be due to increased osmolality of ruminal fluid which drew out fluid from the blood vascular system to the rumen (Chakrabarti, 1988).

4.2.1.2.4 Defaecation

Most of the acidotic goats defaecated pelleted to pasty faeces with undigested crushed rice throughout the whole period of experiment. Occasional diarrhoea was observed in some goats between 42th hour to 60th hour. Similar observations were also reported by Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983 a), Lal et al. (1989) and Das (1990) in goats.

The diarrhoea might be due to hyperosmolality of intestinal content or due to inflammatory and ulcerative changes of wall of the intestine due to lactic acid (Castello, 1968) or due to secondary bacterial infection of the intestinal wall.

4.2.1.2.5 Dehydration (Skin fold test in second)

In acidotic group no retention of skin fold within '0' hour to 12th hour as observed indicated no or mild (4-6%) dehydration. Retention of skin fold for 20-30 seconds from 24th hour to 48 hour of induction of acidosis indicated 10% dehydration, retention for 6-10 seconds from 60th hour to

96th hour indicated 8% dehydration and retention for 2-3 seconds from 108th hour to 120th hour indicated 6-7% dehydration. Similar was the observations of Gnanaprakasam (1970) and Tanwar and Mathur (1983 a). The retention of skin fold might be due to loss of skin elastisity occured due to less perfusion of water in the peripheral region. The less perfusion of water in the peripheral region might be due to heavy drainage of water from blood into the rumen content.

4.2.1.2.6 Urination

All the acidotic goats urinated 4-8 times a day upto 12th hour. Afterward frequency and quantity of urine decreased with high colour. Anuria between 60th hour to 84th hour was also observed. Similar finding was also recorded previously by Gnanaprakasam (1970), Tanwar and Mathur (1983 a) and Das (1990). Anuria or oliguria might be attributed to no or decreased perfusion and filtration rate of kidney (Huber, 1969; Blood et al., 1983). High coloured urine indicated concentrated urine.

4.2.1.2.7 pH of Urine

The mean pH of urine of acidotic goats was 8.21 $\pm$ 0.13 at '0' hour which decreased significantly ($P/0.01$) from 12th hour onward as compared to '0' hour. Lowest mean pH of urine was 5.06 $\pm$ 0.068 at 48 hours and again slightly rose significantly ($P/0.01$) to 6.75 $\pm$ 0.25 at 96 hours but still remained below from '0' hour value. The pH of the urine at 60th hour, 72th hour and 84th hour were not determined due to nonavailability of urine (Fig. 7). Similar finding was

reported by Li et al. (1984) and Terashima et al. (1978) in sheep and Misra and Singh (1974), Sethuraman and Rathor (1979) in cattle and buffaloes.

The decrease in pH of urine might be due to metabolic acidosis resulted from Lactiacedemia. The lowest mean pH of urine at 48th hour indicated peak level of lactic acidosis. There was direct correlation between pH of rumen fluid and urine which could be used as a diagnostic tool in acid indigestion (Misra and Singh, 1974; Sethuraman and Rathor, 1979). Urine pH may be a more reliable indicator of milder forms of lactic acidosis. Since hydrogen ion secretion by the kidney would proceed before the tubular reabsorption mechanism for lactic acid would be exceeded (Huber, 1976).

4.2.1.2.8 pH of Faeces

The mean pH of faeces of acidotic goats was 7.41 $\pm$ 0.27 at '0' hour which decreased insignificantly at 12 hours but decreased significantly ($P/0.01$) from 24 hours onwards throughout the experimental period as compared to '0' hour value. The lowest mean pH of faeces was 4.60 $\pm$ 0.25 at 48th hour which differed significantly ($P/0.01$) from '0' hour value (Fig. 8). Due to nonavailability of such information in goat, the present findings could not be compared. However, these findings agreed with the observations of Kutus et al. (1983) in cattle.

The low pH of faeces was as a result of passage of lactic acid from rumen to gut and it could be used as a

Fig. 7. CLINICAL OBSERVATIONS ON pH OF URINE IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS

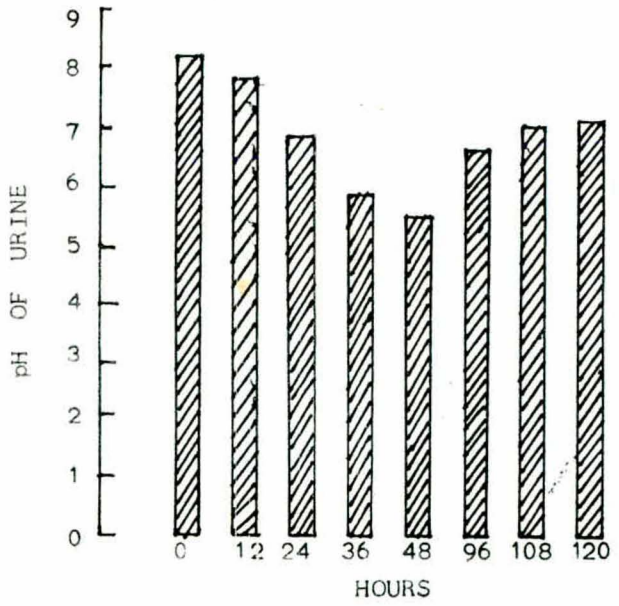
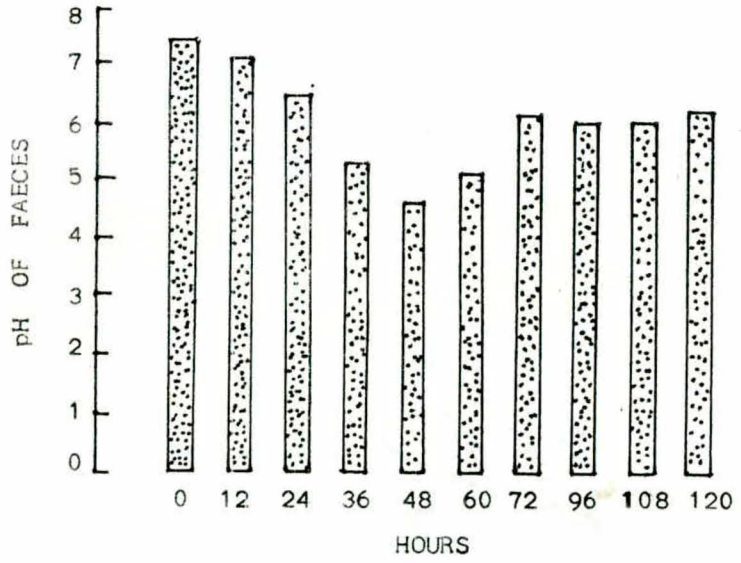


Fig.8. CLINICAL OBSERVATIONS ON pH OF FAECES IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS



dignostic tool of rumen acidosis in an indirect way. The lowest mean pH of faeces at 48th hour indicated peak level of lactic acidosis.

4.2.1.2.9 Heart rate

The mean heart rate was 72.33 ± 1.33/minute at '0' hour which gradually increased significantly (P/0.01) from 12th hour to 48th hour as compared to '0' hour value. The mean heart rate decreased from 60th hour onward towards the value of 116.00 ± 5.01/minute at 96th hour but always significantly higher (P/0.01) than that of '0' hour value. The mean heart rate at 48th hour was 142.00 ± 0.708 which was the highest of value of all hours (Fig. 9). Similar trend was also reported by Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983 a), Cao et al. (1987), Lal et al. (1989) and Das (1990) in goats.

The increased heart rate might be due to accumulated lactic acid which after absorption caused severe acidosis, fall in plasma volume and circulatory failure (Blood et al., 1983).

4.2.1.2.10 Rectal Temperature

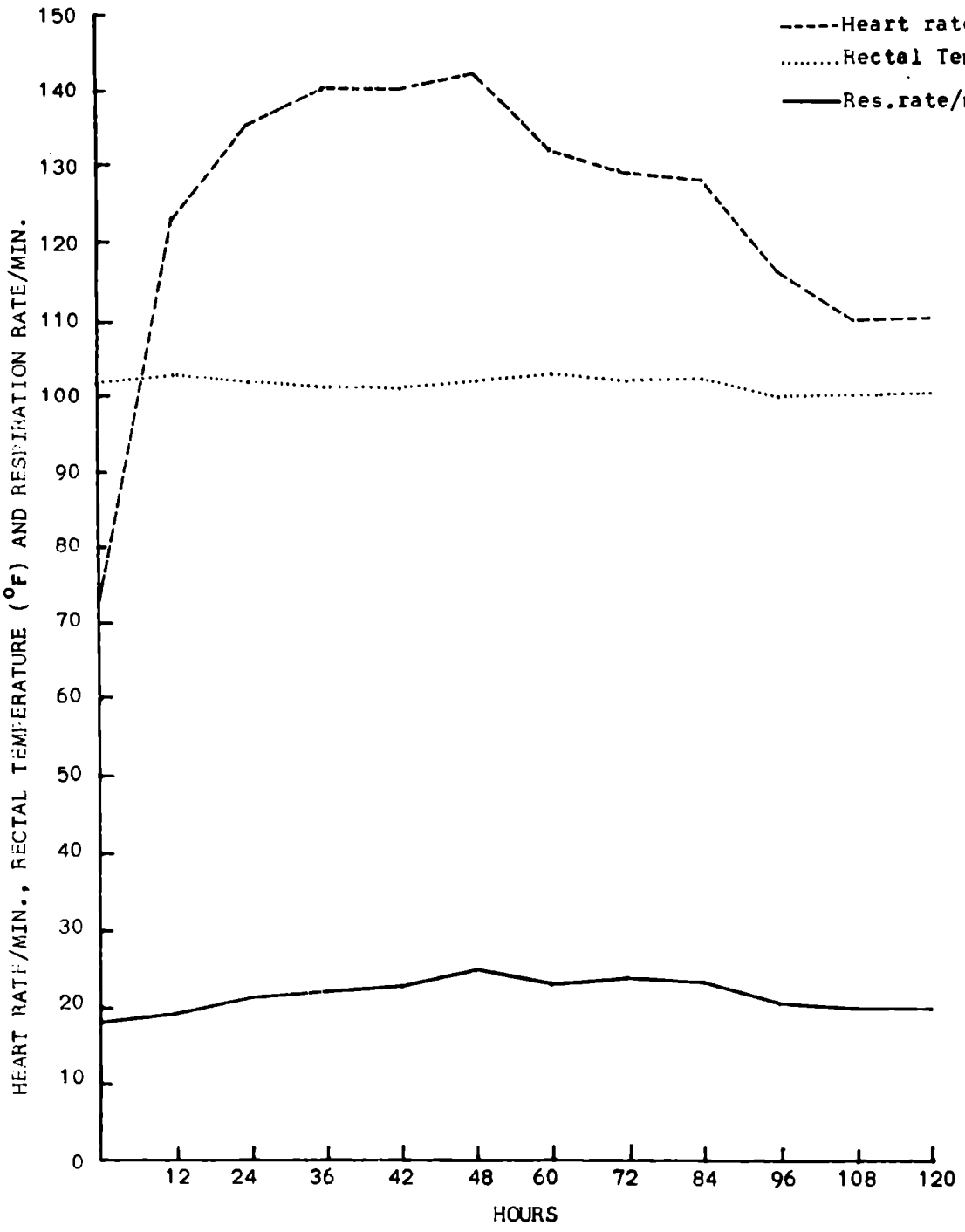
The mean rectal temperature increased from '0' hour level of 102.5 ± 0.179°F to a significant level (P/0.01) of 103.01 ± 0.202°F at 12th hour and decreased subsequently not so markedly from 24th hour onwards except at 60th hour where the level was higher than that of '0' hour (Fig. 9).

The present finding did not properly coincide with the findings of Sen (1982) in goat and Vestweber et al. (1974) in sheep, but coincided with the observations of Gnanaprakasam (1970), Cao et al. (1987) and Lal et al. (1989) in goat. Every animal showed subnormal rectal temperature at 2 - 3 hours prior to death. The initial rise of temperature might be due to increased metabolic turnover following feeding of highly fermentable carbohydrate i.e. crushed rice. The subsequent rise of temperature might be due to bacterial ruminitis (Blood et al., 1983). The overall significant fall of rectal temperature throughout the period of study might be due to lactic acidosis leading to dehydration, fall in plasma volume, severe depression of cardiovascular system and fall in blood pressure (Dunlop and Hammond, 1965).

4.2.1.2.11 Respiration rate

The mean respiration rate recorded at '0' hour was 17.83 $\pm$ 0.98/minute which continuted to increase significantly ($P/0.01$) from 24th hour onward upto 84th hour then declined at 96th hour but not below the value at '0' hour. The mean highest value at 48th hour was recorded as 25.2 $\pm$ 0.376/minute (Fig. 9). Similar findings on the increase of respiration rate was earlier reported by Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983 a), Cao et al. (1987), Lal et al. (1989) and Das (1990) in goats. The above observations of rise in respiratory rate might be due to stimulation of respiratory centre by increased Co_2 tension of blood and decreased blood pH bringing about an increased rate of Co_2 elimination (Huber, 1976).

Fig. 9. CLINICAL OBSERVATIONS ON HEART RATE, RECTAL TEMPERATURE AND RESPIRATION RATE IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS



4.2.2 Examination of Rumen liquor

4.2.2.1 Macroscopic

The mean with standard error as well as the result of the test for mean difference of different macroscopical observations of rumen liquor in acidotic goats (Group B) at different hours are shown in Table - 10. The results of analysis of variance of the different macroscopical observations between hours are also shown in Table - 11.

4.2.2.1.1 Smell

The smell of rumen liquor of the acidotic group was aromatic at '0' hour which changed at first to faintly sour from 12th to 24th hour, then to intensed sour from 36th hour onwards and again changed to sour and faintly sour from 60th hour onwards. This findings were close to the findings of Sen (1982), Sen et al. (1982), Lal et al. (1989) in goats. The sourness of smell of rumen liquor might be due to increased production of lactic acid.

4.2.2.1.2 Consistency

The consistency of rumen liquor of the acidotic group was semiliquid at '0' hour which at first changed to very thick with froth at 12th hour and then to watery from 24th hour onward throughout the whole experimental study. These were in accordance with the findings as described by Sen (1982), Lal et al. (1989) and Das (1990) in goats. The watery nature of rumen liquor might be due to passage of fluid from vascular

Table — 10 . Mean with standard error of the different macroscopical observations on rumen liquor in acidotic goats (Group 9).

Parameters \ Hours	0 (6)	12 (6)	24 (6)	36 (5)	42 (5)	48(5)	60 (4)	72 (3)	84 (3)	96 (2)	108 (1)	120 (1)
Smell	AR	FS	FS	IS	IS	IS	S	S	S	FS	FS	FS
Consistency	SL	TF	W	W	W	W	W	W	W	W	W	W
Colour	DG	CB	CB	GW	GW	GW	GW	GW	GW	GW	GW	GW
Cellulose digestion time(CDT), in hours.	47.22[a] ±3.64	77.00[b] ±4.33	-	-	-	-	-	-	-	-	-	-
Sediment activity time (SAT),in min.	25.94[a] ±1.70	65.33[b] ±4.25	-	-	-	-	-	-	-	-	-	-
Methylene blue reduction time (MBRT),inmin.	11.48[a] ±0.45	54.34[b] ±5.00	-	-	-	-	-	-	-	-	-	-

AR = Aromatic
FS = Faintly sour
IS = Intense sour
S = Sour
SL = Semiliquid
TF = Very thick with froth
DG = Dark green
W = Watery
CB = Creamish brown
GW = Grayish white
- = Absent

Figures in Parentheses indicate the number of animals

Means with same superscripts in the same row do not differ significantly.

bed to rumen as a result of increased osmomolality of the rumen content. At 12th hour, very thick with frothyness of the rumen liquor might be due to increase viscosity with heavy concentration of undigested crushed rice particles.

4.2.2.1.3 Colour

The colour of the rumen liquor of the acidotic group changed from dark green at '0' hour to creamish brown at 12th hour and 24th hour and then changed to greyish white for subsequent hours till the end of the experimental period. Similar observations were also reported by Gnanaprakasam (1970), Sen *et al.* (1982) and Lal *et al.* (1989) in goats. Variation of the colour might be due to type of feed taken by the animals and species variation (Rosenberger, 1979; Dash *et al.*, 1972).

4.2.2.1.4 Cellulose digestion time (CDT), Sediment activity time (SAT) and Methylene blue reduction time (MBRT)

The CDT, SAT and MBRT of rumen liquor of the acidotic group were 47.22 $\pm$ 3.64 hours, 25.94 $\pm$ 1.70 minutes and 11.48 $\pm$ 0.45 minutes at '0' hour respectively and increased significantly ($P/0.01$) to 77.00 $\pm$ 4.33 hours, 65.33 $\pm$ 4.25 minutes and 54.34 $\pm$ 5.00 minutes respectively at 12th hour. There was no cellulose digestion activity, sedimentation activity and methylene blue reduction activity from 24th hours onward in this study. The observed results were very similar to that of Gnanaprakasam (1970), Sen (1982) and Das (1990) in goat and Rosenberger (1979) in cattle.

The significant increase in SAT, CDT and MBRT by 12 hours of the induction of acidosis followed by the absence could be ascribed to the destruction of normal microflora (Cellulolytic bacteria) and shift in their pattern from predominantly Gram negetive to Gram positive nature (amylolytic) (Randhawa et al., 1989). This indicated gross inactivity of the rumen microbes resulted to disturbance of normal digestion process.

4.2.2.2 Microscopic

The mean with standard error as well as the result of the test for mean difference of different microscopical observations of rumen liquor in acidotic goats (Group B) at different hours are shown in Table - 12. The results of analysis of variance of the different microscopical observations between hours are also shown in Table - 11.

4.2.2.2.1 Motility, Concentration, Total count and Iodophilic nature of rumen Protozoa

In acidotic goats, the motility, concentration and iodophilic nature of rumen protozoa was totally absent from 12th hour to 84th hour of induction of rumen acidosis, as compared to '0' hour values which were +++ (Vigorous), +++ (high) and ++/+++ (moderate to high). From 96th hour onward there was reappearance of protozoa in very low concentration. The average total protozoal count was $47.92 \pm 9.41 \times 10^4$/ml at '0' hour

Table – 12. Mean with standard error of the different microscopical observations on rumen liquor in acidotic goats (Group B).

Parameters \ Hours	0 (6)	12 (6)	24 (6)	36 (5)	42 (5)	48 (5)	60 (4)	72 (3)	84 (3)	96 (2)	108(1)	120(1)
Concentration of rumen protozoa	+++	-	-	-	-	-	-	-	-	+	+	+
Motility of rumen protozoa	+++	-	-	-	-	-	-	-	-	-/+	-/+	-/+
Total protozoal count(10^4/ml)	47.92^{a} ±9.41	2.07^{b} ±0.31	-	-	-	-	-	-	-	2.10^{b} ±0.355	2.55	3.00
Iodophilic activity of rumen protozoa	++/+++	-	-	-	-	-	-	-	-	-/+	-/+	-/+
Total bacterial Count (10^9/ml)	16.65^{a} ±3.61	13.95^{ab} ±2.20	11.08^{ac} ±1.23	ND	ND	5.54^{c} ±0.83	7.80^{bc} ±0.50	8.38^{bc} ±0.46	9.27^{bc} ±0.59	9.20^{ac} ±1.00	9.50	10.00
Type of bacteria	Gram -ve	Gram-ve and Grame +ve	Gram +ve	Gram +ve	Gram +ve	Gram +ve	Gram +ve	Gram +ve	Gram +ve	Gram+ve and Gram -ve	Gram+ve and Grame-Ve	Grame+ve and Grame -ve

Concentration and Iodophilic activity of rumen protozoa

+++ = high

++ = Moderate

+ = low

-/+ = nil to low

- = nil

Motility of protozoa

+++ = Vigorous

+ = low

- = Absent

-/+ = Absent to low

Figures in Parentheses indicate the number of animals

Means with same superscripts in the same row do not differ significantly.

ND = Not done

which was markedly reduced at significant level ($P/0.01$) to $2.07 \pm 0.31 \times 10^4$/ml by 12th hour of induction of rumen acidosis and no protozoa was found from 24th hour to 84th hour. Reappearance of protozoa in very few numbers from 96th hour onward which was much less than that of '0' hour, was also noted (Fig. 10). These observations agreed with the observations of Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983 a) and Lal *et al*. (1989).

Growth, multiplication, viability and motility of the ruminal protozoa of healthy goats depended on the H^+ ion concentration and osmolality of the rumen liquor which provided an optimal environment. It was also been suggested that lysis of the rumen protozoa occured when the rumen pH fall below 5.5 (Hungate, 1966). Similarly death and disintegration of the protozoa had been reported in solution of 160 or 455 milliosmol osmotic pressure (Slyter, 1976). Marked reduction in the motility, concentration and viability of the protozoa could be due to low intracellular and environmental pH (Prins and Van Hoven, 1977) and high tonicity of rumen encountered in lactic acidotic animals (Aherns, 1967). Hungate *et al*. (1952) and Krogh (1959) observed that factors like low rumen pH and high rumen toxicity killed protozoa. No iodophilic nature occured due to improper utilization of carbohydrate and storage of glycogen by the rumen protozoa affected with toxic factors. The reappearance of rumen protozoa from 96th hour onwards synchronized with the improvement in ruminal pH (Dunlop, 1972).

4.2.2.2.2 Total Bacterial count

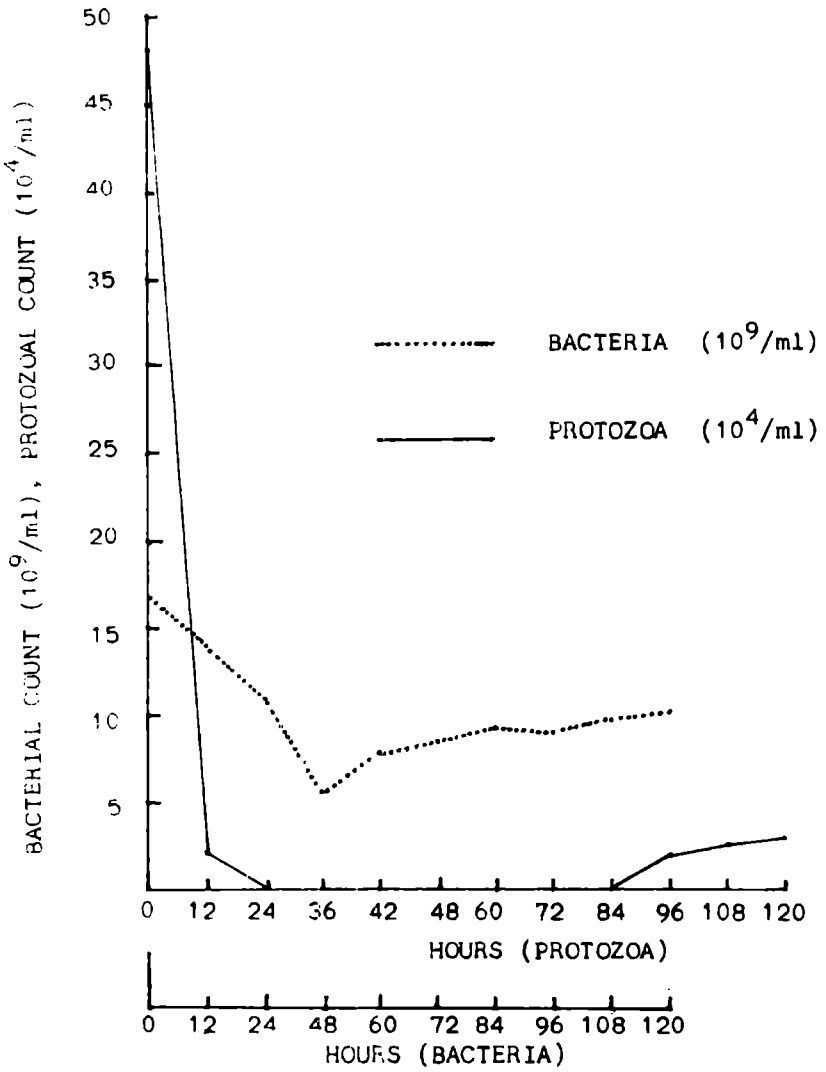
In acidotic goats average total bacterial count showed a significant ($P/0.01$) decrease and the minimum counts were observed at 48th hour of induction of lactic acidosis. Subsequently, the bacterial count showed an increasing trend but count was less than the '0' hour count (Fig. 10). Similar findings were also reported by Lal *et al.* (1989). Contrary to this Tanwar and Mathur (1983 b) observed increased total bacterial count.

The reduced total bacterial count might be due to destruction of cellulolytic bacteria at low pH (Dunlop, 1972). However, Narendra *et al.* (1990) suggested that gradual increase in total bacterial count might be due to multiplication of *Streptococci* and *Lactobacilli*.

4.2.2.2.3 Type of Bacteria

At '0' hour Gram negative bacteria were predominant while Gram positive bacteria (Coccoid and rod) were predominent from 24th hour onwards (Fig. 11). Few Gram negative bacteria were found at 96 hours and onwards. These were in agreement with the previous reports of Hungate *et al.* (1952), Krogh (1963), Allison *et al.* (1964), Mann (1970) and Vazquez (1976). Excessive ingestion of carbohydrate rich sources was associated with increase in the activity of alpha-amylase enzyme possessed by the Gram positive bacteria, particularly *Streptococcus bovis* and *Lactobacilli*. These bacteria take soluble sugar

Fig. 10. MICROSCOPICAL OBSERVATIONS OF RUMEN LIQUOR ON PROTOZOAL AND BACTERIAL COUNT IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS



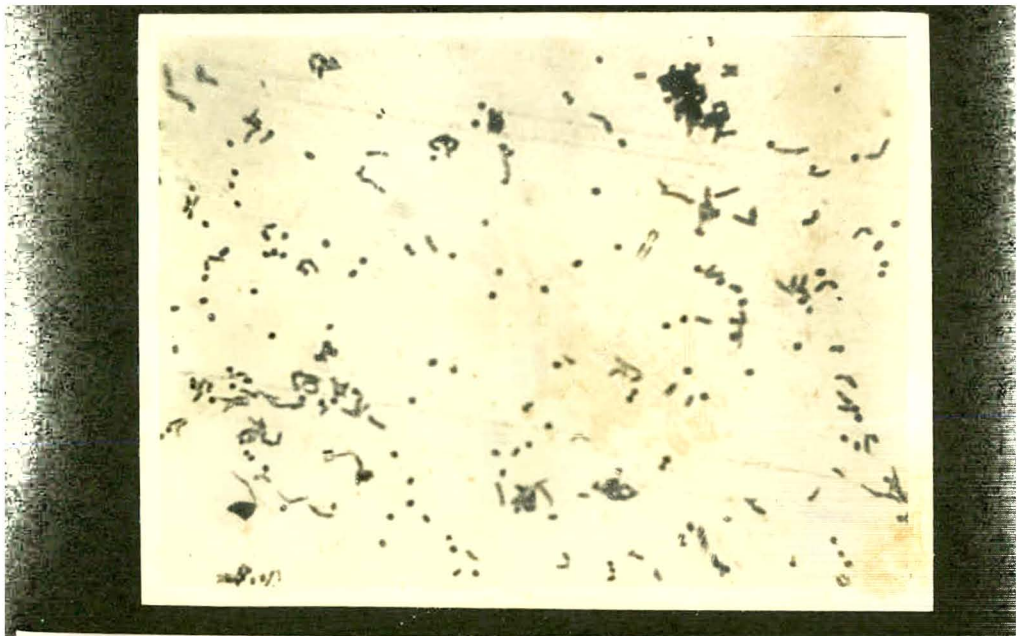


Fig. 4. Picture showing gram +ve Cocci and rod shaped bacteria of rumen liquor at 36th hour of rumen acidosis of goats (Group - B) x 1000.

especially glucose at faster rate and used to ferment it at a very high rate to produce lactic acid (Mac Pherson, 1953). Identification of few Gram negative bacteria at 96 hours and onwards might be due to improvement of ruminal pH.

4.2.2.3 Biochemical

The mean with standard error as well as the result of the test for mean difference of different biochemical attributes of rumen liquor in acidotic goats (Group B) at different hours are shown in Table - 13. The results of analysis of variance of the different biochemical attributes between hours are also shown in Table - 11.

4.2.2.3.1 pH of Rumen liquor

The average pH of rumen liquor gradually decreased at significant level ($P/\underline{0}.01$) from 6.98 $\pm$ 0.065 at '0' hour to the lowest value of 4.37 $\pm$ 0.096 at 48th hour. From 60th hour onwards there was gradual improvement of pH, but values were still significantly ($P/\underline{0}.01$) lower than that of '0' hour value (Fig. 12). Similar findings were also reported by Gnanaprakasam (1970), Sen (1982), Vihan et al. (1982), Cao et al. (1987), Lal et al. (1989). The decrease in the rumen pH might be due to complete and faster fermentation of crushed rice by amylolytic bacteria leading to production of lactic acid (Ahrnes, 1967; Tremere et al., 1968), reduction in concentration of rumen ammonia nitrogen and also high concen-

Table – 13. Mean with standard error of the different biochemical attributes on rumen liquor in acidotic goats (Group - B).

Parameters \ Hours	0(6)	12 (6)	24 (6)	36 (5)	42(5)	48 (5)	60 (4)	72 (3)	84 (3)	96 (2)	108(1)	120(1)
pH of rumen liquor	6.98^{a} ±0.065	6.57^{b} ±0.109	5.40^{c} ±0.115	4.43^{d} ±0.093	ND	4.37^{d} ±0.096	4.68^{d} ±0.08	5.15^{e} ±0.029	5.26^{c} ±0.265	5.26^{c} ±0.241	5.40	5.60
Lactic acid (mg/100ml)	6.06^{a} ±0.32	210.27^{b} ±10.34	326.08^{c} ±10.95	303.05^{c} ±9.60	ND	244.13^{d} ±10.50	175.58^{e} ±8.36	104.21^{f} ±9.45	76.76^{fg} ±6.75	65.26^{g} ±5.25	50.33	53.13
Total volatile fatty acid (TVFA)in mEq/L	58.66^{a} ±8.12	19.00^{b} ±2.36	9.83^{c} ±1.33	6.00^{c} ±0.797	ND	6.20^{c} ±1.09	7.25^{c} ±1.70	10.33^{cd} ±2.51	16.30^{bd} ±2.12	36.00^{e} ±7.07	52.00	60.00
Ammonia nitrogen(NH_3-N) in mg/100 ml.	14.60^{a} ±1.208	9.36^{b} ±1.00	5.68^{c} ±0.784	3.26^{c} ±0.312	ND	13.26^{a} ±2.28	23.13^{d} ± 1.54	32.22^{e} ±1.46	42.94^{f} ±2.74	43.75^{f} ±4.26	56.73	30.25

Figures in Parantheses indicate the number of animals

Means with same superscripts in the same row do not differ significantly.

ND = Not done.

Table – 11. Analysis of variance of different macroscopical, microscopical and biochemical parameters of rumen liquor in acidotic goats (Group B)

Parameters	Source of variations			
	Between hours		Error	
	d.f.	M. S.	d.f.	M. S.
C D T	9	4067.40**	35	27.34
S A T	9	2553.79**	35	17.94
M B R T	9	1670.92**	35	21.69
Total protozoal count	9	1306.34**	35	75.98
Total bacterial count	7	67.00**	27	22.36
pH of rumen liquor	8	4.43**	30	0.053
Lactic acid	8	59303.97**	30	412.22
T V F A	8	1613.7**	30	14.96
NH_3-N	8	718.50**	30	9.7

(** P/0.01)

tration of TVFA (Phillipson, 1942). The improvement of the pH of rumen liquor at 60th hour onwards might be due to reappearance of greater number of cellulolytic and other types of bacteria (Eadie and Mann, 1970).

4.2.2.3.2 Lactic acid

The average concentration of lactic acid in rumen liquor was 6.06 ± 0.32 mg/100 ml which increased at significant level ($P/0.01$) at 12th hour onwards in acidotic goats. The maximum concentration of lactic acid was 326.08 ± 10.95 mg/100 ml at 24th hour of induction of acidosis which gradually decreased for subsequent hours but the values always remained higher than the '0' hour value till the end of the experimental period (Fig. 12). These findings agreed with Sen (1982), Tanwar and Mathur (1983 b), Vihan and Rai (1985), Cao *et al.* (1987) and Lal *et al.* (1989) in goats. The rise in lactic acid concentration was due to predominance of Gram positive bacteria particularly *Streptococcus bovis* and *Lactobacilli* which fermented soluble sugar at a faster rate to produce lactic acid (Mac Pherson, 1953) and stoppage of growth of some of the lactate utilizing bacteria at lower pH (Slyter *et al.*, 1976). Gradual decline in concentration of lactic in the rumen from 36th hour onwards might be due to buffering of some lactic acid by rumen buffers, absorption of lactic acid through ruminal wall into circulation and decrease in fermentative process by amylolytic bacteria.

4.2.2.3.3 Total Volatile fatty acids (TVFA)

The average concentration TVFA of rumen liquor was 58.66 ± 8.12 mEq/L at '0' hour which gradually decreased from 12th hour to 48th hour at a significant level (P/0.01). The lowest mean concentration was 6.00 ± 0.707 mEq/L at 36th hour. From 60th hour onwards till the end of experiment the average concentration of lactic acid in rumen liquor gradually increased (Fig. 12).

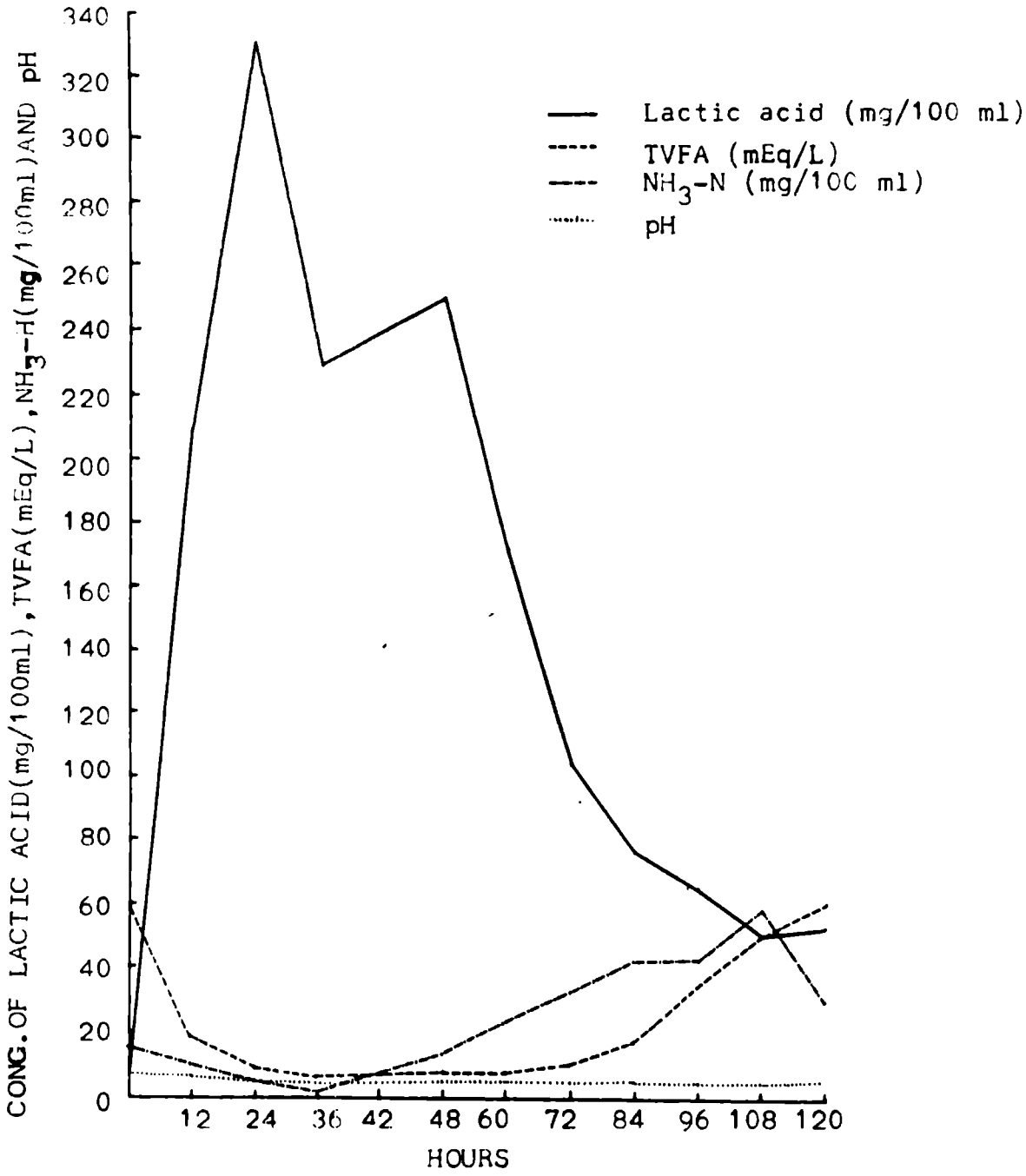
These observations were very close to the findings of Sen (1982) in goats, Chaplin and Jones (1973) in sheep and Ryan (1962) in overfed animals. However TVFA concentrations of rumen liquor was increased initially as previously reported by Tanwar and Mathur (1983 b), Lal et al. (1989) in goats, Scarisbrick (1954) and Muir et al. (1980) in sheep and Prasad et al. (1972), Sethuraman and Rathor (1979) and Randhawa et al. (1989) in cattle and buffaloes. The gradual fall of concentration of TVFA indicated reduction of rumen microbial fermentation (Randhawa et al., 1989). However, Danielli et al. (1945) and Gray, (1948) reported that abrupt rise in the TVFA concentration at 12 hours might be attributed to the rapid fermentation of starch by amylolytic bacteria in the paunch and subsequent decrease in the value might be due to increased absorption at low ruminal pH. Gradual increase in concentration of TVFA at 60th hour onwards might be due to improvement of normal rumen fermentative process synchronized with the improvement of rumen pH. However, Reid et al. (1957) pointed out

that increase in TVFA after 48 hours was due to development of a group of TVFA producing bacteria which were resistant to low rumen pH and capable of producing TVFA.

4.2.2.3.4 Ammonia nitrogen (NH_3-N)

In acidotic goats mean concentration of ammonia nitrogen in rumen liquor was 14.6 $\pm$ 1.208 mg/100 ml at '0' hour, whereas the concentrations at 12th hour, 24th hour, 36th hour and 48th hour was significantly lower ($P/0.01$) than that of '0' hour. The concentration of ammonia nitrogen gradually increased significantly ($P/0.01$) from 60th hour onwards towards the end of the experimental period as compared to the value at '0' hour of induction of rumen acidosis (Fig. 12). These observations coincided with the findings of Chou and Walker (1964 b) in sheep and Sethuraman and Rathor (1979) and Randhawa *et al.* (1989) in calves. However Mc-Donald (1948) reported that level of ammonia in the rumen rose first and then gradually fell away in ruminants while Scarisbrick (1954) reported increase in ammonia in sheep in acid indigestion. The fall of concentration of ammonia nitrogen in rumen liquor might due to depression of the fermentation at low pH and (or) a faster rate of conversion of ammonia into microbial protein. However, the ammonia was not utilized by the microbes and so it increased gradually from 60th hour onwards with some improvement of fermentation due to decrease in rumen pH. Increased blood urea nitrogen which occurs as a result of renal failure in rumen acidosis, may be recycled through saliva and in the rumen it is fermented

Fig.12. BIOCHEMICAL OBSERVATIONS ON CONCENTRATION OF LACTIC ACID, TOTAL VOLATILE FATTY ACID (TVFA), AMMONIA NITROGEN (NH_3-N) AND pH OF RUMEN LIQUOR IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS.



to produce much ammonia.

4.2.3 Analysis of Blood

4.2.3.1 Haematological examination

The mean with standard error as well as the result of the test for mean difference of different haematological observations in acidotic goats (Group B) at different hours are shown in Table - 14. The results of analysis of variance of the different haematological observations between hours are also shown in Table - 15.

4.2.3.1.1 Packed cell volume (PCV)

The average packed cell volume of acidotic goats at '0' hour was 22.66 $\pm$ 0.33 percent, which gradually increased significantly ($P/0.01$) from 12th hour to 48th hour of induction of acidosis. Subsequently, it gradually decreased towards the end of the experimental period, but the values were significantly ($P/0.01$) still higher than '0' hour value. The highest PCV wqs 41.4 $\pm$ 0.601 percent at 48th hour in this experiment (Fig. 13). Similarly the increase in PCV percentage has also been previously reported by Sen (1982), Tanwar *et al*. (1983) and Tanwar and Mathur (1983 a). The cause of increased haematocrit value was due to haemoconcentration which could be explained due to the withdrawal of fluid from the intravascular compartments into the rumen leading to hypertonicity of rumen contents (Huber, 1971). Increase in haematocrit value was also due to the release of red blood cells from the spleen as postulated during acute indigestion (Turner and Hodgetts, 1959).

Table — 14 . Mean with standard error of different haematological observations in acidotic goats (Group 8)

Parameters \ Hours	0 (6)	12 (6)	24 (6)	36 (5)	42 (5)	48 (5)	60 (4)	72 (3)	84 (3)	96 (2)	108(1)	120(1)
PCV (%)	22.66^{a} ±0.33	25.00^{b} ±0.364	31.00^{c} ±0.726	37.80^{d} ±0.973	39.40^{d} ±0.60	41.40^{e} ±0.601	35.75^{f} ±0.945	32.60^{c} ±0.33	31.66^{c} ±0.33	28.50^{g} ±0.501	28.00	29.00
Hb (g/100ml)	8.76^{a} ±0.324	9.06^{af} ±0.311	9.53^{bfeg} ±0.28	10.06^{cdeh} ±0.271	10.30^{cdh} ±0.249	10.76^{d} ±2.50	10.05^{cdei} ±0.05	9.56^{aghi} ±0.233	9.06^{ag} ±0.066	8.70^{ag} ±0.20	8.50	8.30
TEC(10^{6}/cmm) (NS)	8.82 ±0.38	9.03 ±0.37	9.33 ±0.37	9.54 ±0.36	9.72 ±0.36	10.20 ±0.32	9.87 ±0.41	9.50 ±0.50	9.20 ±0.50	8.35 ±0.15	8.50	8.50
TLC(10^{3}/cmm)	5.93^{a} ±0.55	10.97^{b} ±0.64	12.18^{bg} ±0.84	14.4^{cdg} ±1.07	15.64^{cefj} ±1.03	16.22^{cdhi} ±1.07	18.35^{eh} ±0.77	13.9^{bfgi} ±2.19	12.68^{bgj} ±2.66	15.60^{cgh} ±1.66	16.75	17.50
DLC												
Total neutrophils (%)	42.83^{a} ±2.24	51.66^{b} ±0.881	62.83^{c} ±1.70	75.40^{dfg} ±1.81	84.75^{h} ±3.77	83.60^{h} ±1.50	80.00^{dh} ±0.708	71.33^{ef} ±2.40	71.33^{eg} ±3.48	83.50^{h} ±1.50	80.00	82.00
(i)Neutrophils - Segmented(%)	41.33^{a} ±2.22	44.16^{ab} ±1.16	48.33^{abcg} ±2.60	52.00^{cefj} ±3.44	74.50^{d} ±4.21	60.00^{hi} ±1.00	52.60^{afghi} ±3.77	44.33^{af} ±4.26	59.00^{ij} ±2.08	76.50^{d} ±2.50	76.00	78.00

Contd.....146..

Parameters \ Hours	0 (6)	12 (6)	24 (6)	36(5)	42 (5)	48 (5)	60 (4)	72 (3)	84 (3)	96 (2)	108 (1)	120 (1)
ii)Neutrophils - band (%)	1.33^{a} ±0.21	7.50^{b} ±0.43	10.50^{b} ±0.67	19.00^{pde} ±1.14	8.75^{b} ±0.853	20.60^{def} ±1.36	24.60^{ef} ±4.04	25.67^{f} ±2.33	11.00^{b} ±3.51	6.00^{b} ±1.00	4.00	4.00
iii)Metamyelo-cytes (%)	0.00^{a}	0.00^{a}	4.00^{b} ±0.577	4.40^{b} ±1.16	1.50^{cd} ±0.288	3.00^{bc} ±0.549	2.80^{bcd} ±0.665	1.33^{cd} ±0.288	1.33^{cd} ±0.288	1.00^{ad} ±1.00	0.00	0.00
Lymphocytes (%)	53.33^{a} ±2.53	46.16^{b} ±0.871	36.66^{c} ±1.52	24.60^{df} ±1.81	15.25^{de} ±3.72	14.80^{e} ±1.32	20.00^{de} ±0.709	28.33^{f} ±2.18	28.00^{f} ±3.47	15.00^{e} ±1.00	15.00	13.00
Monocytes (%)	1.67^{a} ±0.42	1.00^{ac} ±0.45	0.17^{b} ±0.16	0.00^{b}	0.00^{b}	0.20^{b} ±0.20	0.00^{b}	0.00^{b}	0.67^{ab} ±0.34	0.50^{bc} ±0.50	2.00	2.00
Eosinophils (%)	2.16^{a} ±0.307	1.17^{b} ±0.307	0.34^{cd} ±0.21	0.00^{c}	0.00^{c}	0.40^{ce} ±0.40	0.00^{c}	0.34^{cf} ±0.355	0.00^{c}	1.00^{bdef} ±0.00	3.00	3.00
Basophils (%)	0.16 ±0.16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Figures in Parentheses indicate the number of animals.

Means with same superscripts in the same row do not differ significantly.

NS = Non Significant

Table - 15. Analysis of variance of different haematological parameters in acidotic goats (Group B)

Parameters	Source of variations			
	Between hours		Error	
	d.f.	M. S.	d.f.	M. S.
P C V	9	197.96**	35	1.97
Hb	9	2.15**	35	0.367
T E C (NS)	9	1.09	35	0.723
T L C	9	61.67**	35	5.43
Total Neutrophils (%)	9	409.41**	35	8.55
Neutrophils segmented (%)	9	547.75**	35	36.88
Neutrophilic band cells (%)	9	265.82**	35	9.22
Metamyelocytes (%)	9	44.53**	35	4.41
Lymphocytes (%)	9	354.99**	35	9.40
Monocytes (%)	9	10.59**	35	2.39
Eosinophils (%)	9	17.41**	35	2.055

(** P/0.01)

N S = Non Significant

4.2.3.1.2 Haemoglobin (Hb)

The average concentration of haemoglobin of acidotic goats was 8.76 ± 0.324 g/100 ml at '0' hour, which slightly increased to 9.06 ± 0.311 g/100 ml at 12th hour. Between these two values, there was no significant difference. Whereas it increased from 24th hour at a significant level ($P/0.01$) and continued to 48th hour. Thereafter, the concentration of haemoglobin gradually declined towards the normal value nearer to the '0' hour value (Fig. 13). The increase in haemoglobin concentration in acid indigestion was also previously reported by Tanwar *et al.* (1983) in goats. The increase in haemoglobin concentration had been attributed to decrease in plasma volume and to spleenic contraction (Huber, 1971). However, gradual improvement of haemoglobin value towards the '0' hour value might be due to reduction of dehydration which was attributed to increase in plasma volume.

4.2.3.1.3 Total Erythrocytic count (TEC)

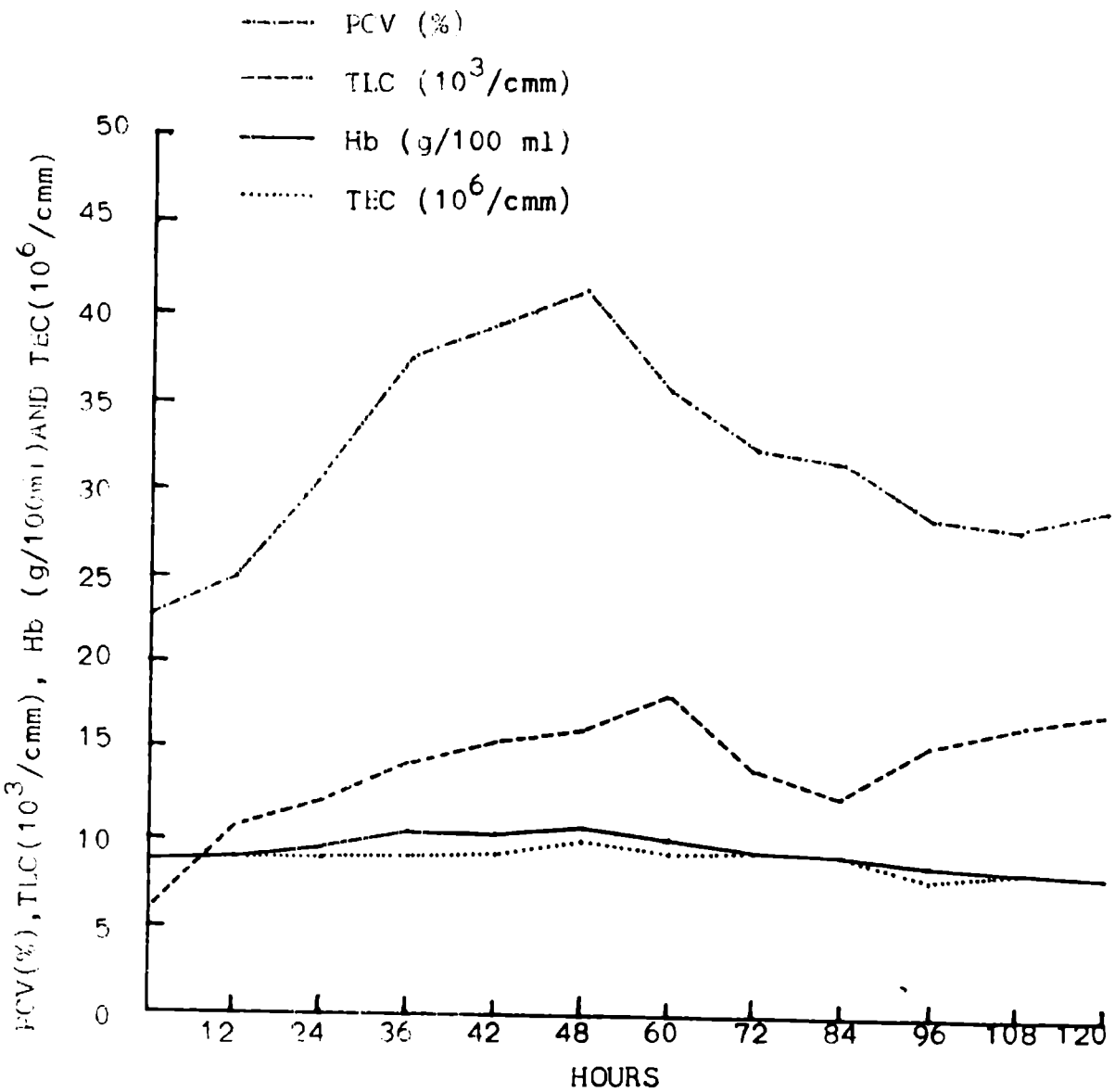
The average total erythrocytic count at '0' hour of the acidotic goats was 8.82 ± 0.38 x 10^6/cmm which gradually increased to a maximum value of 10.2 ± 0.32 x 10^6/cmm at 48th hour and thereafter gradually declined towards the base value (Fig. 13). This variation was not significant statistically. Similar observations were also reported earlier by Tanwar *et al.* (1983) in goats, Telle and Preston (1971) in sheep and Dash *et al.* (1972), Misra and Singh (1974) and HejLasz *et al.* (1984) in cattle. The increase in total

erythrocytic count in lactic acidosis might be due to two probable causes, namely the efflux of water from the blood into the rumen and the release of red blood cells from the spleen as postulated during acute indigestion (Turner and Hodgetts, 1959). The stress due to lactic acidosis has been noted to stimulate the adrenals (Kilburn, 1966) thereby causing a release of red blood cells from the spleen.

4.2.3.1.4 Total Leukocytic count (TLC)

The average total leukocytic count at '0' hour in acidotic goats was 5.93 $\pm$ 0.55 x 10^3/cmm. The count increased gradually at significant level ($P/0.01$) from 12th hour onwards upto 60th hour in which time maximum count was observed. Thereafter the count declined gradually upto 84th hour of induction of acidosis. But, the values were still significantly higher ($P/0.01$) than the value of '0' hour. Again the count sharply rose at significant level from 96th hour onwards (Fig. 13). The increase in leukocyte count at first phase has also been previously reported by Cao *et al.* (1987) in goats. At first phase the increase in total leukocytic count was evidently due to the increased heart rate (Physiologic leukocytosis) and the effect of stress due to indigestion (Castello, 1968; Dash *et al.*, 1972; Schalm *et al.*, 1986). Gradual reduction in leukocytic count from 72th hour to 84th hour might be due to withdrawal of stress factors associated with decrease in lactic acid concentration in rumen liquor and blood. However, 2nd phase rise in total leukocytic count

Fig.13. HAEMATOLOGICAL OBSERVATIONS ON PACKED CELL VOLUME(PCV),TOTAL LEUKOCYTIC COUNT (TLC), HAEMOGLOBIN (Hb) PERCENTAGE AND TOTAL ERYTROCYTIC COUNT (TEC) IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS.



might be due to bacterial rumenitis, liver abscesses and peritonitis etc.

4.2.3.1.5 Differential Leukocytic count

4.2.3.1.5.1 Total Neutrophil percentage including Segmented neutrophil, Neutrophilic Band cell and Metamyelocyte

The average percentage of total neutrophils at '0' hour was 42.83 ± 2.24 which gradually increased significantly ($P/0.01$) from the 12th hour onwards and maximum value of 84.75 ± 3.77 was observed at 42th hour. Afterwards percentage of neutrophils gradually decreased upto 84th hour, but values were significantly ($P/0.01$) higher than that of '0' hour value. Again at 96th hour onwards the neutrophilic percentage increased significantly ($P/0.01$) from the '0' hour value (Fig. 14). The pattern of increased and decreased percentage of segmented neutrophils also followed the same trend like total neutrophil percentage (Fig. 15), but the values at 36th hour and onwards differed significantly ($P/0.01$) from '0' hour value (Fig. 14). There was also significant ($P/0.01$) increase in percentage of band cells (Fig. 16, 17) from '0' hour to 72th hour at which time maximum percentage was observed. Afterwards, percentage declined towards the base value (Fig.14). The percentage of metamyelocytes was completely nil at '0' hour and 12th hour. The appearance of metamyelocytes (Fig. 18) at 24th hour onwards occurred at a significant percentage

(P/0.01). After that it gradually disappeared from 96th hour onwards (Fig. 19).

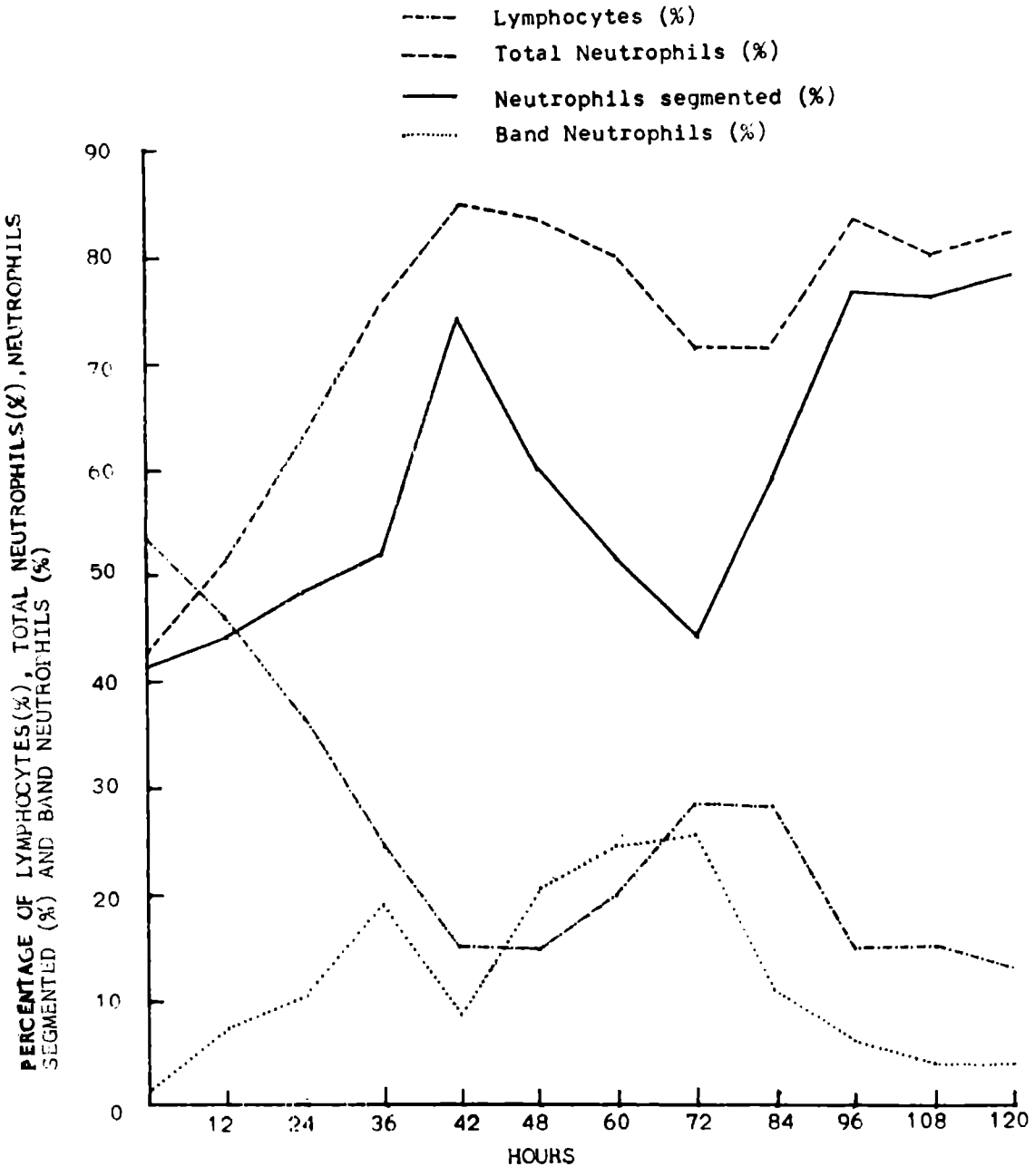
The total picture showed that there were neutrophilia, comperatively less percentage of segmented neutrophils from the total neutrophilic percentage, appearance of neutrophilic band cells in high percentage along with metamyelocytes. Similar findings were also reported by Cao et al. (1987) in goats and in bovine indigestion by Hjerpe, (1961), Hjerpe (1963), Castello (1968), Dash et al. (1972) and Schalm et al. (1986).

The increase in the number of immature neutrophils specially the metamyelocytes, band cells along with leukocytosis i.e. neutrophilia with shift to the left might be due to stress factors in blood forming organs during acute indigestion. (Castello, 1968; Dash et al., 1972). The stress factor is corticosteroids (Guyton, 1976). The observation of neutrophilia in second phase at 96th hour onwards might be due to invasion and infection of secondary bacteria in the ruminal wall, liver, lungs etc.

4.2.3.1.5.2 Lymphocyte, Monocyte, Eosinophil and Basophil percentage

The average percentage of lymphocytes at '0' hour was 53.33 ± 2.53. It gradually decreased significantly (P/0.01) to a minimum percentage of 14.8 ± 1.32 at 48 hours of induction of acidosis. Afterwards there was gradual increase in percentage at 60th hour onwards. But, the values were

Fig.14. HAEMATOLOGICAL OBSERVATIONS ON DIFFERENTIAL LEUKOCYTIC COUNT(Lymphocytes,Total neutrophils,Neutrophils segmented and Band neutrophils percentage) IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS.



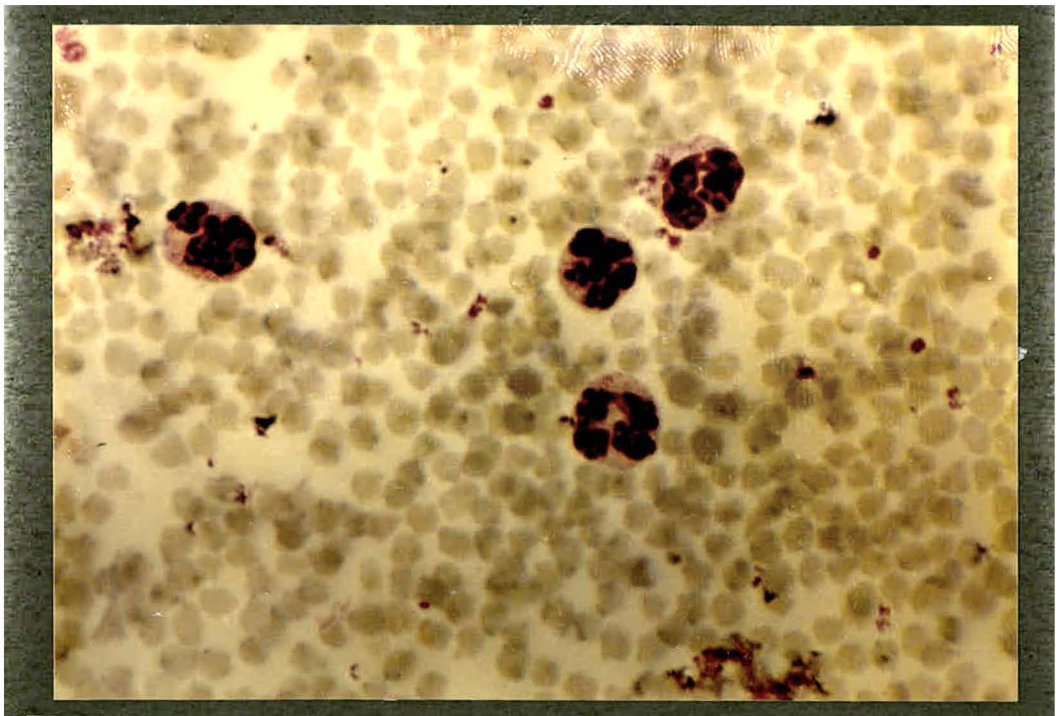


Fig. 15. Picture of blood smear showing segmented neutrophilia in experimental rumen acidosis of goats (Group - B) x 1000

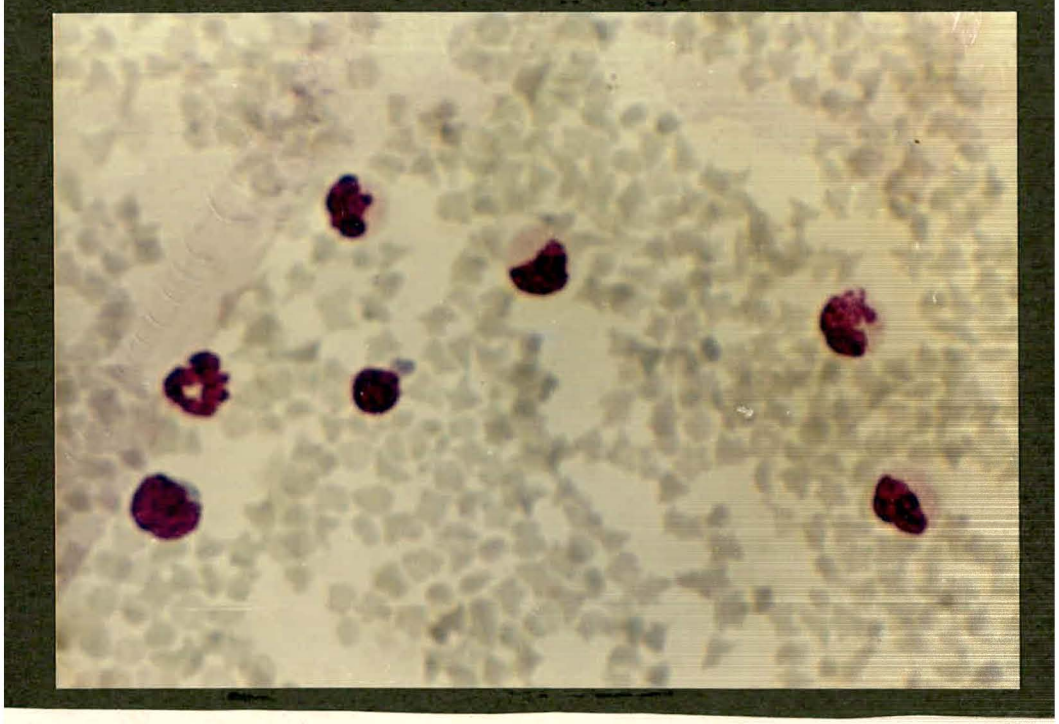


Fig. 16. Picture of blood smear showing neutrophilic band cells, unsegmented neutrophils, segmented neutrophils and lymphocytes in experimental rumen acidosis of goats (Group - B). x 1000.

still significantly (P/0.01) below the value of base level. Again there was decline of lymphocytes percentage at 96th hour onwards (Fig. 14).

The average percentage of monocytes and eosinophils were 1.67 ± 0.42 and 2.16 ± 0.307 respectively at '0' hour which showed a significant (P/0.01) fall from 24th hour and 12th hour onwards respectively. There was abscence of cells at 36th, 42th and 60th hours following induction lactic acidosis (Fig.19). Percentage of basophils was completely nil from 12th hour onwards.

The findings of lymphopaenia, eosinopaenia, monocytopaenia and reversal of normal lymphocyte : neutrophil ratio in indigestion were previously reported by Cao et al. (1987) in goats and Hjerpe (1961), Hjerpe (1963), Castello (1968), Dash et al. (1972) and Misra and Singh (1974) in bovine . The lymphopaenia, eosinopaenia might be due to release of corticosteroids due to stress as induced by digestive disorder (Castello, 1968).

4.2.3.2 Biochemical examination of Serum/Blood

The mean with standard error as well as result of the test for the mean difference of different biochemical attributes of serum or blood of acidotic goats (Group B) at different hours are shown in Table - 16. The results of analysis of variance of different biochemical attributes between hours are also shown in Table - 17.

Table-16 : Mean with standard error of the different bio-chemical attributes of blood or serum in acidotic goats (Group B)

Parameters \ Hours	0 (6)	12 (6)	24 (6)	36 (5)	42 (2)	48 (5)	60 (4)	72 (3)	84 (3)	96 (2)	108(1)	120(1)
Blood pH	7.40^{a}	7.31^{bg}	7.24^{ch}	7.19^{ode}	7.15^{d}	7.23^{cef}	7.29^{bfgh}	7.28^{bfgh}	7.25^{baf}	7.37^{ag}	7.35	7.32
	±0.018	±0.021	±0.018	±0.015	±0.017	±0.014	±0.016	±0.008	±0.014	±0.02		
Blood glucose (mg/100ml)	52.67^{a}	88.17^{b}	92.50^{c}	103.10^{d}	116.08^{e}	109.24^{f}	100.24^{d}	93.29^{o}	99.63^{d}	102.75^{d}	100.20	85.50
	±0.747	±1.79	±0.661	±0.435	±1.67	±0.565	±0.146	±0.255	±0.296	±0.751		
Blood lactic acid (mg/100 ml)	14.54^{a}	22.82^{b}	31.78^{c}	45.78^{d}	48.66^{d}	39.05^{e}	31.04^{c}	28.61^{o}	30.70^{o}	26.91^{bo}	21.21	20.11
	±0.612	±1.60	±1.73	±1.69	±1.93	±1.38	±0.91	±1.31	±0.70	±2.00		
Serum calcium (mg/100 ml)	11.39^{ab}	10.50^{o}	10.60^{od}	9.58^{e}	9.82^{ef}	10.49^{og}	10.37^{cfg}	10.77^{aogh}	11.79^{bi}	11.31^{adhi}	11.33	11.30
	±0.242	±0.166	±0.239	±0.147	±0.16	±0.178	±0.13	±0.014	±0.359	±0.069		
Serum inorganic phosphorus(mg/100ml)	5.78^{a}	8.00^{ad}	14.47^{b}	12.22^{beh}	13.03^{bh}	13.72^{bh}	13.28^{bh}	10.36^{adfgh}	8.20^{af}	7.75^{aef}	6.95	6.20
	±0.426	±0.433	±1.63	±1.18	±1.67	±1.47	±1.50	±0.68	±0.351	±0.751		
Serum total protein(g/100 ml)	6.38^{acd}	6.43^{acd}	6.56^{abd}	7.04^{fg}	7.07^{fg}	7.28^{g}	6.77^{bfh}	6.65^{ah}	6.18^{ce}	6.15^{de}	5.85	5.80
	±0.101	±0.03	±0.142	±0.094	±0.10	±0.92	±0.157	±0.132	±0.164	±0.155		
Serum albumin (g/100ml)	3.95^{d}	3.20^{a}	3.10^{ab}	3.20^{a}	3.21^{a}	3.50^{e}	3.20^{a}	2.90^{bc}	2.69^{c}	2.71^{c}	2.55	2.50
	±0.075	±0.053	±0.071	±0.051	±0.046	±0.089	±0.163	±0.036	±0.074	±0.042		

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Parameters \ Hours	0 (6)	12 (6)	24 (6)	36(5)	42(2)	48(5)	60(4)	72(3)	84(3)	96(2)	108(1)	120(1)
Serum globulin (g/100 ml)	2.42^{f} ±0.038	3.23^{a} ±0.063	3.46^{b} ±0.087	3.85^{c} ±0.045	3.86^{c} ±0.056	3.79^{cd} ±0.164	3.57^{bd} ±0.036	3.75^{cde} ±0.101	3.50^{be} ±0.09	3.44^{abe} ±0.116	3.30	3.30
Albumin:Globulin ratio(A/G)	1.63^{a} ±0.028	0.995^{b} ±0.034	0.898^{bcd} ±0.018	0.831^{de} ±0.006	0.832^{de} ±0.04	0.935^{bc} ±0.07	0.894^{bdf} ±0.049	0.775^{ef} ±0.013	0.768^{e} ±0.001	0.795^{de} ±0.014	0.772	0.751
Blood urea (mg/100 ml)	19.14^{a} ±1.86	21.19^{ab} ±1.49	22.56^{ab} ±1.77	27.55^{b} ±0.843	27.43^{b} ±0.99	39.99^{d} ±4.99	47.76^{c} ±4.42	48.57^{c} ±1.95	49.83^{c} ±2.64	53.89^{c} ±1.39	68.00	72.00
Serum creatinine (mg/100ml)	0.95^{aeg} ±0.052	0.94^{aeg} ±0.06	0.91^{a} ±0.054	0.95^{aeg} ±0.062	0.95^{aeg} ±0.088	1.10^{bg} ±0.067	1.15^{bf} ±0.061	1.31^{cf} ±0.043	1.40^{c} ±0.05	1.92^{d} ±0.075	2.10	2.00
Serum sodium (mEq/L)	141.11^{a} ±0.49	140.90^{a} ±0.85	143.84^{a} ±0.68	148.00^{bc} ±2.20	144.14^{ac} ±2.19	143.40^{a} ±1.90	143.04^{a} ±0.65	142.60^{a} ±0.63	141.14^{a} ±0.52	142.00^{a} ±1.20	141.00	140.00
Serum potassium (mEq/L)	5.10^{a} ±0.13	5.09^{a} ±0.16	5.31^{ad} ±0.16	5.40^{ade} ±0.192	5.50^{abd} ±0.19	5.89^{bde} ±0.24	5.86^{cd} ±0.24	5.47^{ade} ±0.32	5.30^{abd} ±0.36	5.48^{ad} ±0.37	5.01	4.73

Figures in Parentheses indicate the number of animals

Means with same superscripts in the same row do not differ significantly.

Table - 17. Analysis of variance of different biochemical attributes of blood or serum in acidotic goats (Group B)

Parameters	Source of variations			
	Between hours		Error	
	d.f.	M. S.	d.f.	M.S.
Blood pH	9	0.024**	35	0.0026
Blood glucose	9	1649.44**	35	6.55
Blood lactic acid	9	557.08**	35	10.8
Serum calcium	9	1.96**	35	0.194
Serum inorganic phosphorus	9	43.08**	35	7.25
Serum total protein	9	0.611**	35	0.062
Serum albumin	9	0.593**	35	0.0257
Serum globulin	9	1.015**	35	C.033

contd.. 156..

Parameters	Source of variations			
	Between hours		Error	
	d.f.	M. S.	d.f.	M. S.
Albumin : Globulin (A/G) ratio	9	0.35**	35	0.0061
Blood urea	9	720.05**	35	31.37
Serum creatinine	9	0.266**	35	0.02
Serum sodium	9	21.85*	35	9.28
Serum potassium	9	0.384*	35	0.192

(** P/0.01)

(* P/0.05)

4.2.3.2.1 Blood pH

The average blood pH of the acidotic goats at '0' hour was 7.40 ± 0.018 which significantly decreased ($P/0.01$) from 12th hour onwards. The lowest mean pH was 7.15 ± 0.017, observed at 42th hour of induction of acidosis. Afterwards it rose but these values were significantly lower ($P/0.01$) than that of '0' hour, whereas at 96th hour the value of pH was insignificantly lower (Fig. 20). Similar trend has earlier been recorded by Sen (1982), Vihan *et al.* (1982), Tanwar *et al.* (1983) and Cao *et al.* (1987). The drop of pH of blood might be due to lactate absorption from the rumen (Telle and Preston, 1971) and depletion of alkali reserve of blood. Gradual rise of pH value from 48th hour onwards might be due to improvement of alkali reserves to compensate the fall of blood pH and decrease lactic acid formation and absorption through the rumen.

4.2.3.2.2 Blood Glucose

The average concentration of blood glucose was 52.67 ± 0.747 mg/100 ml at '0' hour. The concentration increased significantly ($P/0.01$) at 12th hour onwards. The peak level was observed at 42th hour and then gradually declined from 48th hour onwards, but the concentrations remained still higher at a significant level ($P/0.01$) from '0' hour value. Again from 84th hour the concentration increased at a significant level ($P/0.01$) from the '0' hour value (Fig. 21).

The initial rise of blood glucose level was in accordance with Sen (1982), Vihan et al. (1982) and Vihan and Rai (1985) in experimental rumen acidosis in goats. This increase might have been either due to the increase glycogenolysis or gluconeogenesis 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 stresses of digestive disorder. Gradual decrease in level from 48th hour might have been coincided with the level of corticosteroids. The second phase rise in glucose level might have been due to increase in corticosteroids level and decreased concentration of circulating immunoreactive insulin (IRI).

4.2.3.2.3 Blood Lactic acid

In the acidotic goats, the average concentration of lactic acid in blood gradually increased significantly ($P/0.01$) from 14.54 $\pm$ 0.612 mg/100 ml at '0' hour to a maximum concentration of 48.66 $\pm$ 1.93 mg/100 ml at 42th hour of induction of acidosis. Afterwards it decreased but these values remained significantly higher than the '0' hour value till the end of the experimental period (Fig. 20). A rise in lactic acid concentration in blood during rumen acidosis was also previously noted by Sen (1982), Vihan et al. (1982), Tanwar et al. (1983), Vihan and Rai (1985) and Cao et al. (1987) in

goats. The rise in level of lactic acid in the blood was due to rapid rate of absorption of lactic acid from rumen through damage ruminal wall and less rate of lactic acid conversion to glycogen or glucose in the liver. 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). Gradual decrease in the level of lactic acid in blood from 48th hour might be due to less production and absorption of lactic acid from the rumen and increased rate of metabolism and elimination of lactic acid from the tissues. 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).

4.2.3.2.4 Serum Calcium

The mean concentration of serum calcium in acidotic goats at '0' hour was 11.39 ± 0.242 mg/100 ml which gradually decreased at a significant level ($P/0.01$) from 12th hour onwards. The lowest concentration of 9.58 ± 0.147 mg/100 ml was observed at 36th hour of this study. Afterwards it increased gradually to reach the value nearer to the value of '0' hour till end of the experimental period. However, the values at 72th hour and 84th hour were slightly higher than the '0' hour value (Fig.22). But this difference was statistically insignificant. The matter of hypocalcaemia in rumen acidosis was previously reported by Vihan et al. (1982) in goats, Jones and Luthman (1978), Tereshima

et al. (1978) in sheep, Moodie (1960), Anderson (1980) and Blood et al. (1983) in bovines and Jones et al. (1977), Fraser et al. (1986) and Robinson and Huxtable (1988) in animals. However, Cao et al. (1987) in goats, Telle and Preston (1971) in sheep and Nauriyal and Baxi (1978) and Sethuraman and Rathor (1979) in cattle and buffaloes did not find any change in the level of calcium in serum in rumen acidosis. Juha'sz and Szegedi (1968 b) reported some increase in calcium concentration over a 24 hours period of rumen acidosis in sheep. The cause of hypocalcaemia in rumen acidosis might be due to temporary malabsorption (Blood et al., 1983; Robinson and Huxtable, 1988). Timet et al. (1978) reported that calcium absorption by the rumen was most rapid at pH 7.4 whether from high or low concentration and absorption from the omasum was best at pH 5.2. Loss of appetite, reduced faecal excretion and reduction in alimentary activity might hamper the uptake of calcium from the alimentary tract (Moodie and Robertson, 1961, 1962 ; Robertson et al., 1960; Payne, 1964). High concentrate ration might cause increased loss of urinary calcium (Reed et al.,1955; Yano et al., 1976; Terashima et al., 1978).

The rise of calcium level in serum in the initial stages as reported by Juha'sz and Szegedi (1968 b) in rumen acidosis in sheep might be rapid absorption through gastrointestinal tract favoured by increased acidity of the gastrointestinal fluid before complete stasis of the G.I. tract started (Granstrom, 1903; Hart et al., 1931).

4.2.3.2.5 Serum Inorganic Phosphorus

The mean concentration of serum inorganic phosphorus in acidotic goats at '0' hour was 5.78 ± 0.426 mg/100 ml. The total observations of serum inorganic phosphorus level from 12th hour to 72th hour in the present study showed that the values were significantly (P/0.01) higher than the '0' hour value, which indicated hyperphosphataemia, with some little rise and fall of level between different hours. However, values at 84th hour and 96th hour were higher than that of '0' hour but did not significantly differ from the '0' hour value (Fig. 22). The matter of hyperphosphataemia in rumen acidosis agreed with the findings reported by Vihan et al. (1982) and Cao et al. (1987) in goats, Juha'sz and Szegedi (1968 b), Nokata et al. (1977) in sheep and Cakala et al. (1975), Mullen (1976), Jones et al. (1977), Jagos et al. (1977), Sethuraman and Rathor (1979), Anderson (1980) and Blood et al. (1983) in cattle and buffaloes. However, Nayriyal and Baxi (1978) did not find any change of inorganic serum phosphorus level in experimentally induced ruminal lactic acidosis in cattle and buffaloes. The increase in serum inorganic phosphate was due to renal failure (Kaneko & Cornelius, 1970; Blood et al.,1983).

The serum phosphorus was likely elevated as a result of both increased phosphate reabsorption and decreased renal loss (Reed et al., 1955; Yano et al., 1976; Anderson, 1980).

4.2.3.2.6 Serum Total Protein

The average concentration of serum total protein at '0' hour in acidotic goats was 6.38 ± 0.101 g/100 ml which significantly ($P/0.01$) increased from 12th hour onwards. The highest mean concentration was 7.28 ± 0.92 g/100 ml, observed at 48th hour of induction of acidosis. Afterwards, it gradually declined and reached the value of 6.15 ± 0.155 g/100 ml at 96th hour, which was significantly ($P/0.01$) lower than the '0' hour value. Beyond 96th hour, values of serum total protein were much lower than the '0' hour value (Fig. 23).

The picture of serum total protein in this study showed that at first there was rise of serum total protein and afterwards it declined below the base level. The hyperproteinaemia in acid indigestion was also previously reported by Cao *et al.* (1987) in goats, Juha'sz and Szegedi (1968 b) in sheep and Mullen (1976) in cattles. However, Vihan *et al.* (1982) in goats and Nauriyal and Baxi (1978, in cattle and buffaloes found no change in total serum proteins in rumen acidosis. The increase in concentration of total serum protein might be as a result of haemoconcentration due to dehydration (Mullen, 1976; Anderson, 1980). Water deprivation might result in increase in total plasma protein (Khan *et al.*, 1978). The decrease in total serum protein concentration as observed at 84th hour onwards in this study might be due to liver dysfunction which occured in rumen acidosis.

4.2.3.2.7 Serum Albumin

The average concentration of serum albumin in acidotic goats initially significantly ($P/0.01$) decreased to a minimum level of 3.10 ± 0.071 g/100 ml by 24th hour as compared to '0' hour value of 3.95 ± 0.075 g/100 ml. Afterwards, it showed tendency to rise, but the values were still significantly ($P/0.01$) lower than that of '0' hour. Again at 60th hour onwards, the concentration of serum albumin markedly reduced ($P/0.01$) in relation to the '0' hour value (Fig. 23). The overall picture showed hypoalbuminaemia throughout the whole experimental period. Similar observations were also reported by Prasad and Joshi (1971), Prasad et al. (1972), Prasad and Joshi (1975), Bienick (1981) and HejLasz et al. (1984) in cattle and buffaloes in rumen dysfunction. However, Vihan et al. (1982) did not find any change in serum albumin level in goats in experimentally induced acid indigestion. This variation might be due to species variation, indicating goats are more resistant to ruminal acidosis than cattle and buffaloes. This hypoalbuminaemia and hyperglobinaemia, reduction of A/G ratio might be due to liver dysfunction (Prasad and Joshi, 1971) which occurred in rumen acidosis, because liver played a major role in the biosynthesis of the majority of plasma proteins. Studies on liver dysfunction in acid indigestion was previously reported by Mullen (1976) in cattle and Cakale et al. (1974) in sheep. In rumen acidosis toxic products which are produced in the rumen may cause stress on the liver and sometimes liver is also damaged due to formation of liver abscesses by secondary

bacterial infection. Reduced serum albumin value in experimental acidosis of cow indicated hypertonic dehydration due to displacement of water from the blood and tissue to the digestive tract (HejLasz et al., 1984).

4.2.3.2.8 Serum Globulin

The mean concentration of serum globulin in acidotic goats at '0' hour was 2.42 ± 0.038 g/100 ml which gradually increased significantly (P/0.01) by 12th hour onwards from the '0' hour value. The highest mean concentration of serum globulin was 3.86 ± 0.056 g/100 ml observed at 42th hour. Afterwards, the values slightly decreased as compared to the value of 42th hour but were significantly (P/0.01) much higher than the value of '0' hour (Fig. 23). The total picture of level of serum globulin at different hour compared to the value at '0' hour indicated hyperglobinaemia. Similar observations were also reported by Prasad and Joshi (1971), Prasad et al. (1972), Prasad and Joshi (1975), Bienick (1981) and HejLasz et al. (1984) in cattle and buffaloes. However, Vihan et al. (1982) reported that the serum globulin level in acidotic goat remained within the normal range. Increased serum globulin level might be due to hypertonic dehydration as a result of displacement of water from the blood and tissue to the digestive tract (HejLasz et al., 1984) and also due to liver dysfunction caused by lactic acidosis.

4.2.3.2.9 Albumin/Globulin (A/G) ratio

In acidotic goats the mean albumin : globulin ratio at '0' hour was 1.63 ± 0.028 which gradually declined significantly (P/0.01) at 12th hour onwards except a slight improvement at 48th hour of induction of acidosis. But the value of A/G ratio at 48th hour was significantly (P/0.01) much lower than that of '0' hour value (Fig. 23). The picture showed a reduction of albumin and globulin ratio in this experimental study. Similar observations were also reported by Prasad et al. (1972), Prasad and Joshi (1975), Bienick (1981) in rumen dysfunction in cattle and Jonson and Liberg (1974) in intensively fed calves. The alteration of albumin : globulin ratio was due to liver dysfunction caused by toxic effect of lactic acidosis.

4.2.3.2.10 Blood Urea and Creatinine

The mean concentration of blood urea in acidotic goats at '0' hour was 19.14 ± 1.86 mg/100 ml. It gradually rose significantly (P/0.01) at 36th hour onwards and did not show any tendency to fall down (Fig. 20). The mean concentration of serum creatinine in acidotic goats at '0' hour was 0.95 ± 0.052 mg/100 ml. The serum creatinine level was within the normal range upto 42th hour of induction of ruminal acidosis. It gradually increased significantly (P/0.01) at 48th hour onwards (Fig. 20). Similar observations were also reported previously by Mullen (1976), Jagos et al. (1977), Nauriyal and Baxi (1978), Randhawa et al. (1981), Singh et al. (1989) and Smith (1990) in

cattle and buffaloes and Anderson (1980) in ruminants. However, Cao et al. (1987) did not find any variation of plasma urea and creatinine concentrations upto 48 hours of study of experimentally induced lactic acidosis in the goat. This variation might be due to the fact that the times of observation was less.

As earlier reported by Telle and Preston (1971) the kidneys could develop insufficiency only after a time more than ten hours after the onset of acidosis as observed in the present study. This increase in BUN may be explained as a result of myocardial insufficiency, renal degeneration and subsequently decreased urea clearance by kidney (Randhawa et al., 1981). Dehydration, haemoconcentration, anuria, catabolism with body toxaemia increased the BUN (Coles, 1974). If the kidney is malfunctioning or if the glomerular filtration rate is reduced because of extra-renal factors such as dehydration or shock then the rate of urea excretion will fall and the circulating level of urea will rise (Hall, 1963).

An increased rate of protein catabolism will also raise the blood urea level (Hall, 1983). One of the consequences of rumen acidosis included reduced salivation (Slyter, 1976). This caused failure of urea recycling process. The increased level of blood urea in indigestion might be due to failure of urea recycling process through salivery glands and its non utilization by microbes in the rumen (Singh et al., 1989). Singh et al. (1983) also reported that rise in the blood urea is an index of less utilization of ammonia produced

Fig. 20. BIOCHEMICAL OBSERVATIONS ON LEVEL LACTIC ACID, UREA, pH AND CREATININE OF BLOOD/ SERUM IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP) AT DIFFERENT HOURS.

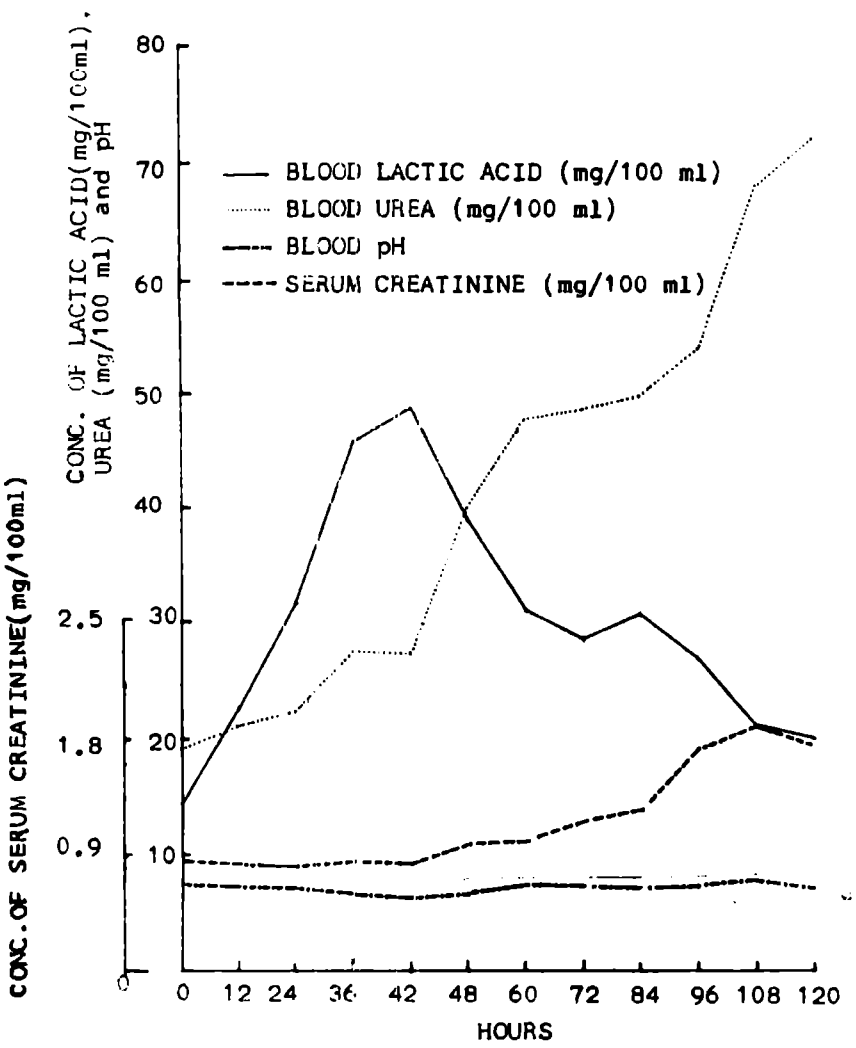


Fig. 21. BIOCHEMICAL OBSERVATIONS ON LEVEL OF SODIUM, POTASSIUM AND GLUCOSE OF BLOOD/SERUM IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS.

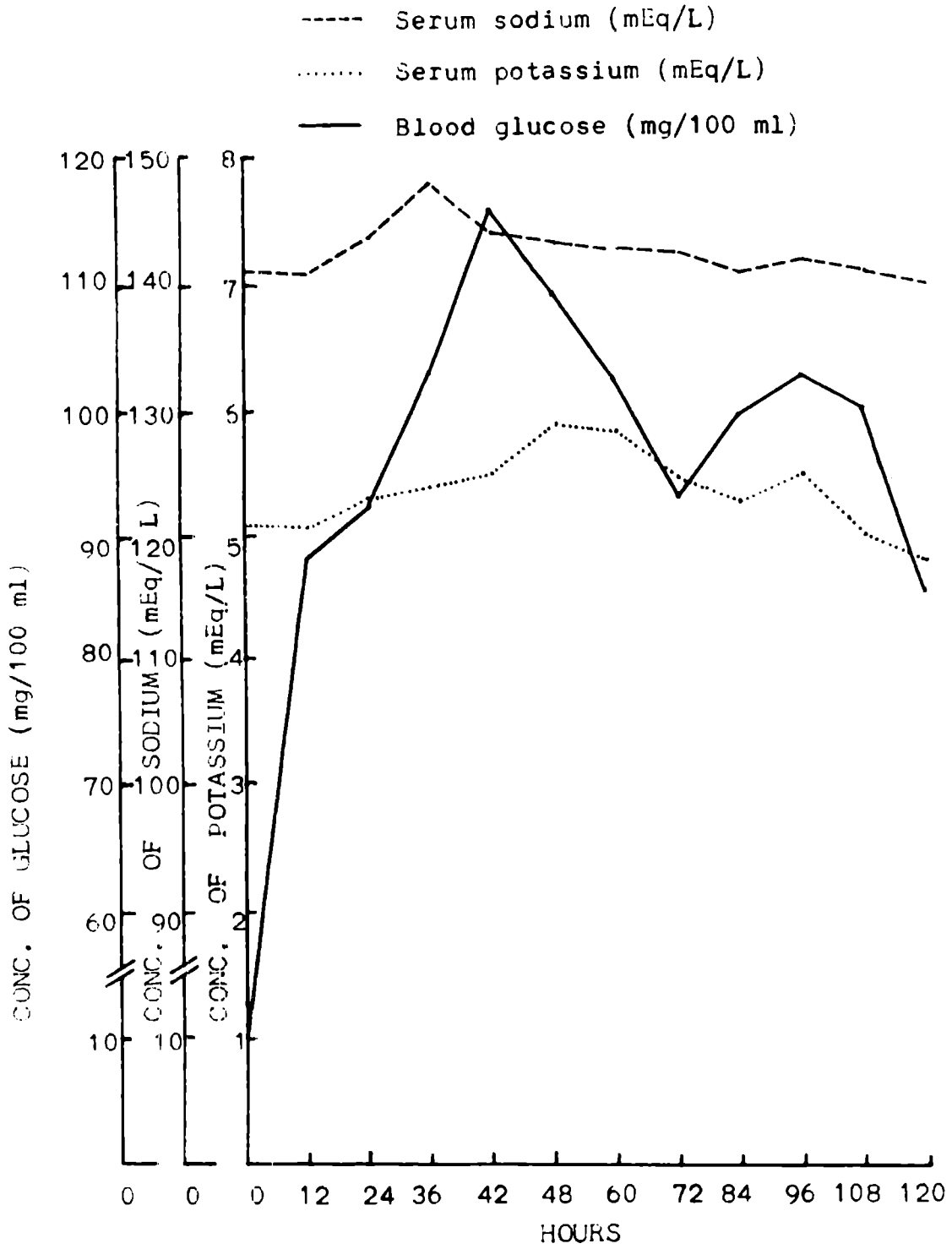


Fig. 22. BIOCHEMICAL OBSERVATIONS ON LEVEL OF PHOSPHORUS AND CALCIUM OF SERUM IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS.

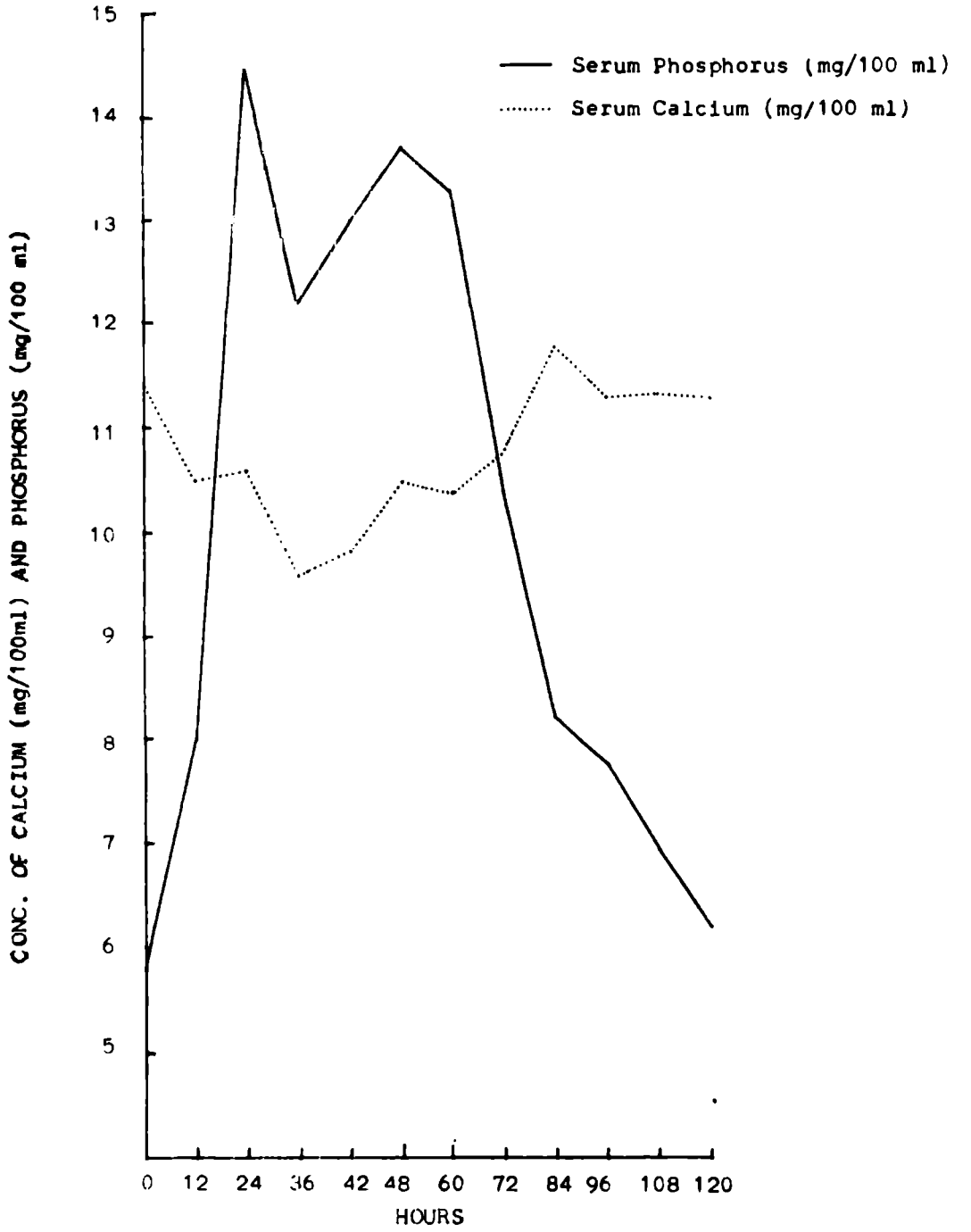
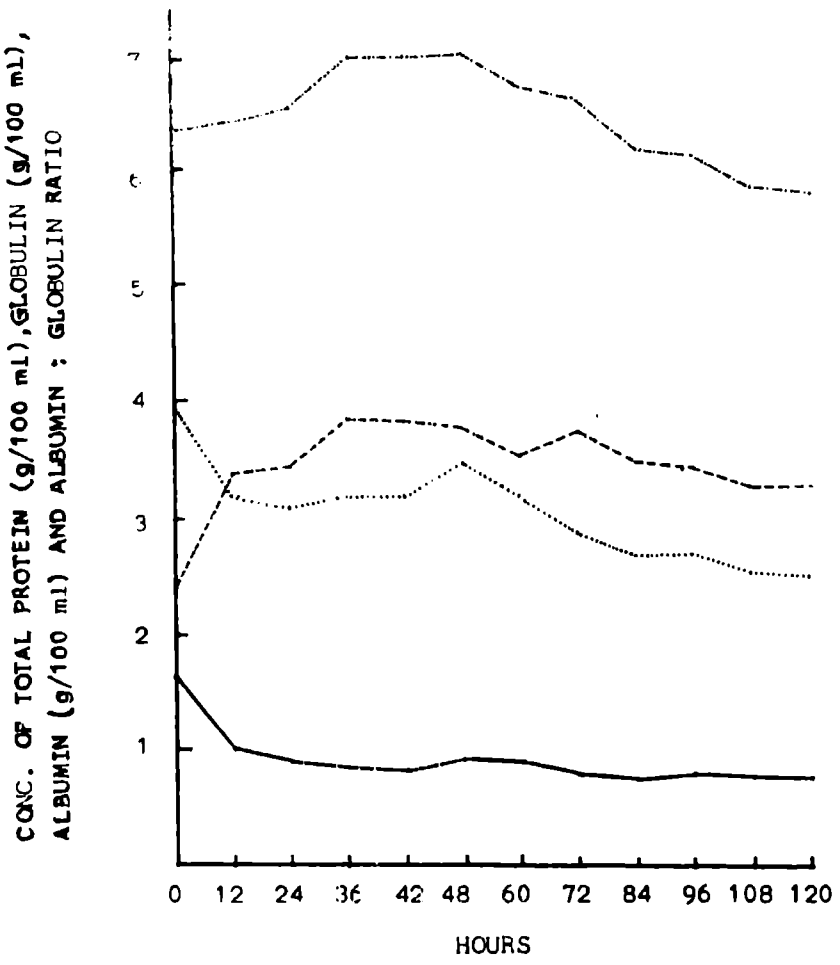


Fig. 23. BIOCHEMICAL OBSERVATIONS ON CONCENTRATION OF TOTAL PROTEIN, GLOBULIN, ALBUMIN AND ALBUMIN : GLOBULIN RATIO OF SERUM/BLOOD IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS.



by the rumen microbs for the synthesis of their own cellular proteins.

The increase in serum creatinine at 48th hour onwards might be due increased catabolism of the tissue due to toxaemia in lactic acidosis. It might also be as a result of less glomerular filtration rate due to dehydration which occured in ruminal acidosis. Serum creatinine level did not abruptly rise like blood urea. The serum creatinine level is not affected as soon as rise of serum urea level following renal damage and is used to detect the more severe types of renal lesions (Hall, 1983). Creatinine is generally considered a better guide to renal failure (Smith, 1990).

4.2.3.2.11 Serum Sodium

The average value of serum sodium was 141.11 $\pm$ 0.49 mEq/L at '0' hour. It gradually increased from 24th hour onwards and highest value was observed 148.00 $\pm$ 2.2 mEq/L at 36th hour which significantly ($P/0.05$) differed from the '0' hour value. From 42th hour onward the values were slightly higher and did not significantly differ from '0' hour value (Fig. 21). The increase in sodium concentration in serum in rumen acidosis agreed with the findings of Sen (1982), Das (1990) in goats and Shinosaki and Nakabayashi (1974) and Irwin *et al*. (1979) in sheep. The higher sodium might be due to its retention by the kidney.

4.2.3.2.12 Serum Potassium

The average concentration of serum potassium was 5.10 ± 0.13 mEq/L at '0' hour. There was significant ($P/0.05$) increase in serum potassium value at 48th and 60th hour. The mean concentrations at 24th, 36th, 42th, 72th, 84th and 96th hour were slightly higher than that of '0' hour but significantly not differed from '0' hour value (Fig. 21). These findings were nearer to the findings of Telle and Preston (1971) and Kuusksalu (1988) in sheep, but differed from the findings of Huber (1971) and Skinosaki and Nakabayashi (1974) in sheep and Sen (1982) and Das (1990) in goats. Increase in serum potassium might be due to its increase reabsorption through renal tubules in respect of secretion of H^+ ion to maintain acid base balance.

4.2.4 Analysis of Hormonal levels

The mean with standard error of different serum hormonal levels of acidotic goats (Group B) at different hours are shown in Table - 18.

4.2.4.1 Serum Cortisol

The average concentration of serum cortisol of acidotic goats at '0' hour was below detection level (1 µg/100 ml). It abruptly increased to mean value 12.4 ± 0.60 µg/100 ml by 12th hour of induction of rumen acidosis. Afterward it gradually increased and reached to a maximum level of 36.14 ± 0.47 µg/100 ml. Again from 48th hour onwards it gradually

Table — 18. Mean with standard error of different serum hormonal levels in in acidotic goats (Group B).

Parameters \ Hours	0 (6)	12 (6)	24 (6)	36 (5)	42 (5)	48 (5)	60 (4)	72 (3)	84 (3)	96 (2)	108 (1)	120 (1)
Cortisol (µg/100ml)	BDL	12.40 ±0.60	25.00 ±0.74	33.25 ±0.55	36.14 ±0.47	29.86 ±0.69	26.70 ±0.36	14.20 ±0.17	22.40 ±0.47	26.15 ±0.55	11.00	5.00
Insulin (µLU/ml)	10.41 ±1.98	48.00 ±3.47	187.17 ±4.33	39.00 ±4.31	35.00 ±4.28	20.00 ±1.45	11.25 ±1.75	5.34 ±0.34	1.94 ±0.07	BDL	BDL	BDL
Thyroxin (T_4) (ng/ml)	44.72 ±4.04	36.49 ±7.23	16.91 ±1.31	4.92 ±0.48	BDL	BDL	BDL	BDL	BDL	23.00 ±4.01	30.3	35.2

BDL = Below detection level i.e. /1 µg/dL for Cortisol
/1 µIU/ml for Insulin
and /1 ng/ml for T_4

Figures in the Parentheses indicate the number of animals.

declined to a minimum level of mean value 14.2 ± 0.17 µg/100 ml by 72th hour. But the concentrations were still higher than that of '0' hour concentration. At 84th and 96th hour the mean concentrations of cortisol in serum sharply rose again and then gradually declined (Fig. 24). This similar incidence of rise of cortisol level was previously reported by Salye (1958) in digestive disorder and Mills& Jenny(1979) in high concentrate fed cattle.

The rise of cortisol level in rumen acidosis due to heavy concentrate feeding might be due to stress which occured in digestive disorders (Salye, 1958). It is amazing that almost any type of stress, whether it be physical or neurogenic will cause an immediate and marked increase in ACTH secretion followed within minutes by greatly increased adrenocortical secretion of cortisol (Guyton, 1976).

The abrupt rise cortisol level at first phase is within 42th hour might be due to lactiacedemia, dehydration, haemoconcentration and circulatory failure etc. which induced stress. Afterwards, animals those were alive could compensate their all physiological disbalance and this was indicated by gradual fall of level of cortisol in serum. Second phase rise of cortisol level at 84th hour and 96th hour might be as a result of stress due to toxaemia, secondary bacterial infection in different tissues and degenerative or inflammatory changes in different tissues. Again in one animal at 108th hour onwards the concentration gradually come down which indicated withdrawal of stress factors and the animal came to recovery phase.

4.2.4.2 Serum Insulin

The average concentration of serum insulin at '0' hour of the acidotic goats was 10.41 ± 1.88 μIU/ml which increased to 48.00 ± 3.87 μIU/ml by 12th hour of induction of acidosis. At 24th hour maximum concentration as 187.17 ± 4.33 μIU/ml was observed. Afterward sharply declined by 36th hour and continued to decrease gradually and reached the value at 96th hour, 108th and 120th hours which was below detection level (<1 μIU/ml) i.e. below the base value (Fig. 24). Horino et al. (1968), Trenkle (1970), Jenny and Polan (1975) in cattle and Bueno et al. (1977) in sheep observed an increase in plasma insulin levels fed with high grain diets and it remained elevated during the entire period of their experimental studies which varied from one to three days. However, Randhawa (1979) found increased level of circulating immunoreactive insulin (IRI) during first 48 hours and then declined gradually and reached below the base values at 120 hours of experimental rumen acidosis in buffaloes calves. Konider et al. (1978) and Hayakawa et al. (1985) reported increase in insulin secretion by intravenous infusion of glucose solution or xylitol solution or fructose solution in sheep. The increase in the insulin level might have been resulted due to higher proportions of propionate and butyrate produced in the rumen of animals fed high grain diet which on absorption stimulated insulin secretion (Horino, et al. 1968; Trenkle, 1970). The increase in circulating level in insulin might also be as result of hyperglycaemia (Gyton, 1976) which was due to high concentration of cortisol and catecholamine

secretion induced by stress in rumen acidosis. Subsequent decrease in the level of IRI (Immuno reactive insulin) at 36th hour onward and below detection level at 96th hour onwards might be as a result of atrophy of endocrine bets cells due to over exhaustion in the early stages (Randhawa et al., 1981 b) or due to destruction of bets cells by lactic acid and other toxaemia substances produced in ruminal acidosis.

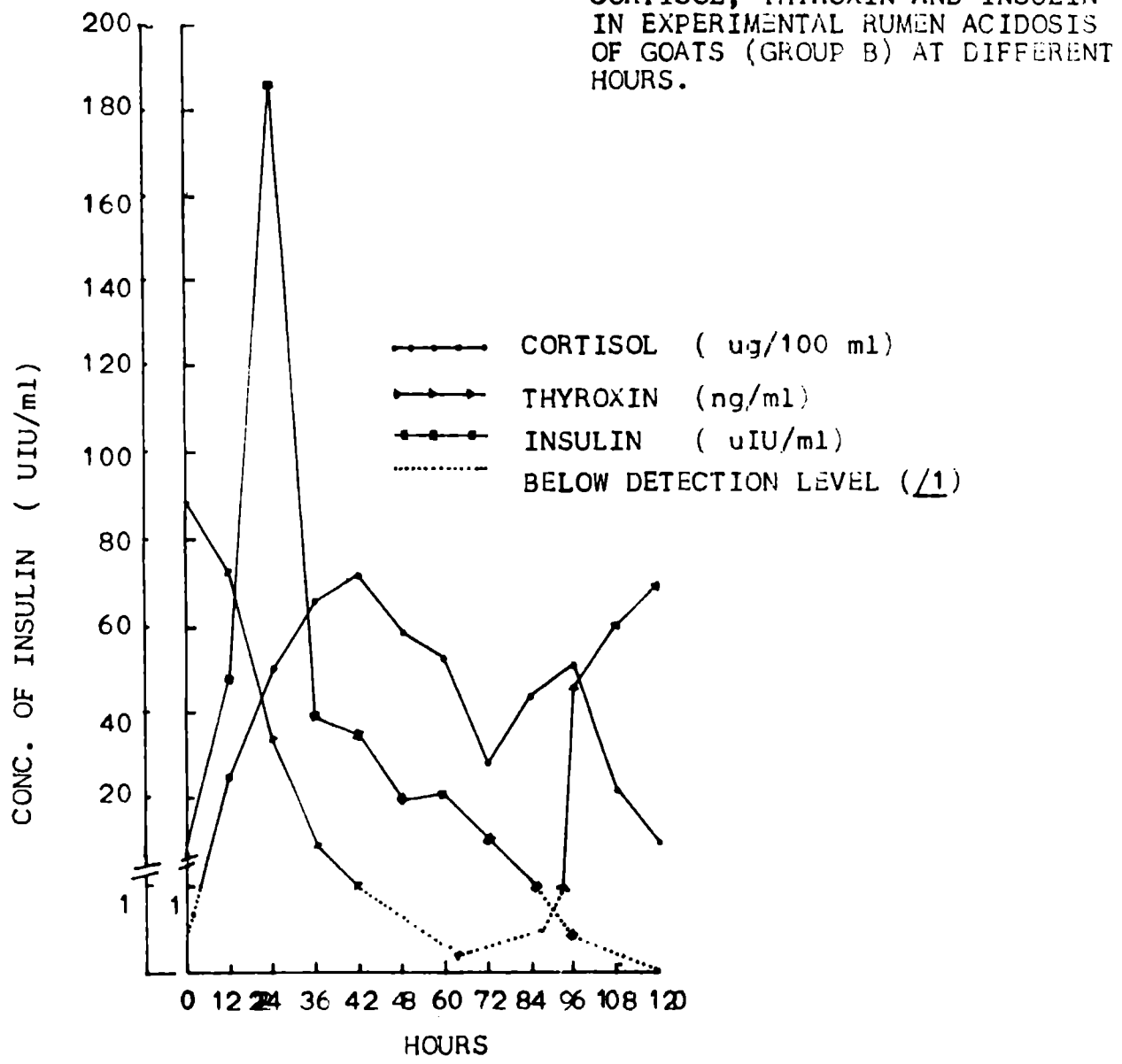
4.2.4.3 Serum Thyroxin (T_4)

The average concentration of thyroxin in serum at '0' hour was 44.72 $\pm$ 4.04 ng/ml which gradually declined at 12th hour onwards upto 84th hour. The concentrations were below detection level ($\not$1 ng/ml) at 42th hour, 48th hour, 60th hour, 72th hour and 84th hour. Afterwards it gradually increased from 96th hour onwards towards the base value (Fig. 24).

There was no such relevant reference on concentration of thyroxin in serum in relation to acid indigestion in ruminants in available literature and as such the present observations could not be compared.

The gradual decline in level of serum thyroxin in lactic acidosis and sometimes below detection level might be due to effect of glucocorticoids. Increased concentration of glucorticoids supress thyroid function at least partly by inhibiting pituitary TSH secretion (Wilber and Utiger, 1970). The fact that electrical stimulation of the mid and posterior

Fig.24. OBSERVATIONS ON LEVEL OF SERUM CORTISOL, THYROXIN AND INSULIN IN EXPERIMENTAL RUMEN ACIDOSIS OF GOATS (GROUP B) AT DIFFERENT HOURS.



median eminance results in increased thyroid activity only after adrenolectomy (Harris and Woods, 1958). It has been also suggested that endogenous glucocorticoids may exert some regulatory influence on the system (Nicoloff et al., 1970). Gradual increase of thyroxin level from 96th hour onwards might be due to gradual decrease in level of corticosteroids in serum.

4.2.5 Pathological studies

4.2.5.1 Gross examination/Necrospsy examination

4.2.5.1.1 Carcases

Emaciated, dehydrated carcase, loss of skin elasticity, shunken eye, congested conjunctival mucous membranes, purulent to mucoid nasal discharge, distended and heavy abdomen with fluid inside were observed in most of the acidotic goats died after rumen acidosis. Few animals showed massive vomiting or regurgitation of sour odorous rumen contents immediately after death.

Carcasses were opened immediately after death and following changes were observed in different organs. The lesions varied in form and degree from goat to goat and were influenced by the course and severity of the disease.

4.2.5.1.2 Rumen

Thining of the wall of the rumen was observed .

Patchy red areas indicating congestion and haemorrhage were observed over the wall of the rumen (Fig. 25). The haemorrhages were observed beneath the rumen mucosa i.e. at the level of submucous, subserosal and serosal layer. After opening the rumen, the rumen contents showed watery, undigested or partially digested ground rice with sour odour. Desquamation of the rumen vill was also observed. The rumen mucosa could be pealed easily with minimum finger pressure.

4.2.5.1.3 Reticulum

It did not show any macroscopical change. It contained only semisolid feed materials. Sloughing of the keratinised layer was observed in few goats.

4.2.5.1.4 Omasum

No abnormality was seen macroscopically.

4.2.5.1.5 Abomasum

The abomesal wall became thin, contained liquid substances. The abomesal mucosa showed slightly peticheal haemorrhages.

4.2.5.1.6 Small and large intestines

Marked venous congestion of alimentary tract was noticed with haemorrhages in the mucosa of small and large intestines (Fig. 26). The loop of the intestines were grossly distended with semiliquid contents. The wall of the intestines became very thin.

4.2.5.1.7 Uterus

One pregnant animal died after induction of rumen acidosis within 36 hours. It's uterine wall showed marked haemorrhages with discolouration (Fig. 27).

4.2.5.1.8 Liver and Gall Bladder

The liver was enlarged in size with distended gall bladder containing bluish yellow tinged fluid. Necrotic patches were noticed on external surface of the liver in some animals. The bile duct was distended. The liver showed pale colour which died between 86th to 96th hour of rumen acidosis (Fig. 28). However, congested liver was seen in those animals which died very early (Fig. 29).

4.2.5.1.9 Kidney

Kidney showed congestive changes only on its cut surfaces. Oedematous appearance of intact kidney was also seen.

4.2.5.1.10 Spleen

Spleen was pale bluish in colour on external surface. Marked hyperemic changes were observed on cut surfaces.

4.2.5.1.11 Lungs

Lungs were congested in the most of the animals (Fig. 30). Two animals showed pale and oedematous lungs, with

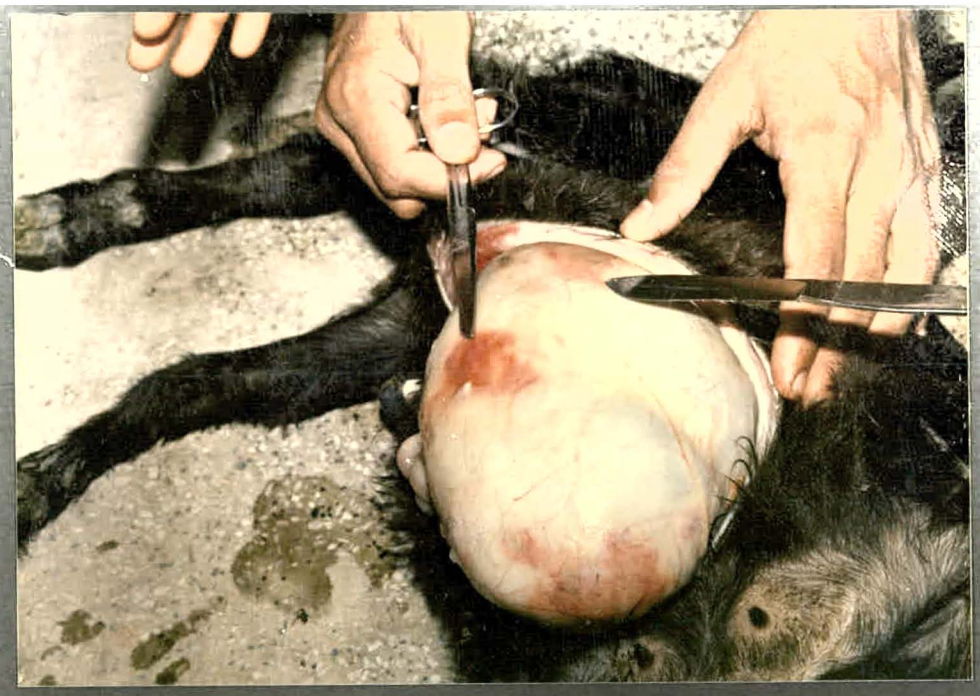


Fig. 25. Patchy red arears indicating congestion and haemorrhages over the wall of the rumen in experimental rumen acidosis of goat (Group - B)

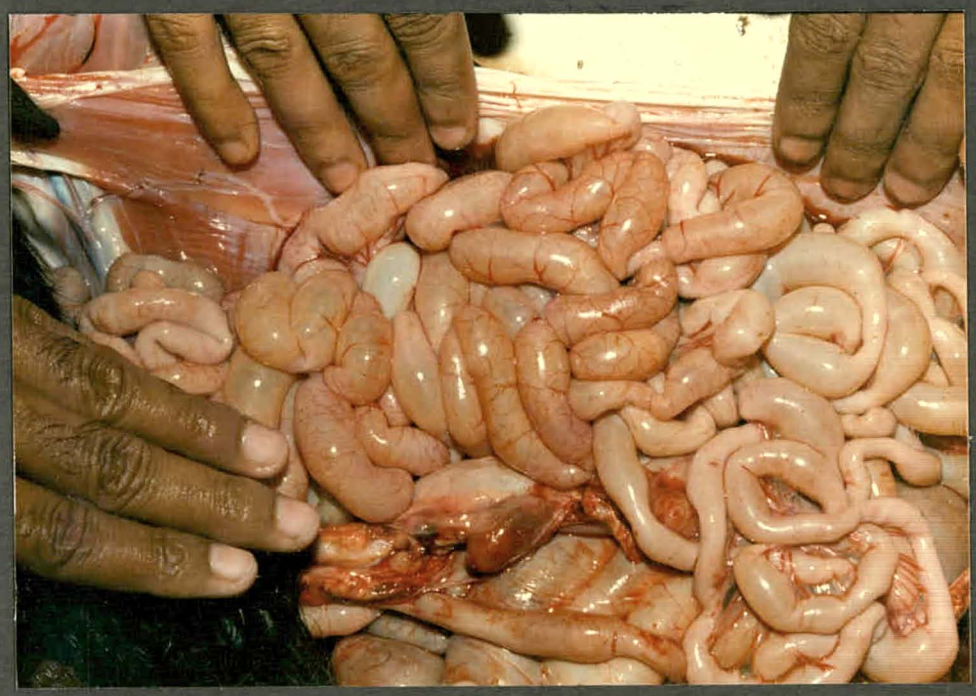


Fig. 26. Marked venous congestion with haemorrhages over the wall of the small and large intestines in experimental rumen acidosis of goat (Group_B)

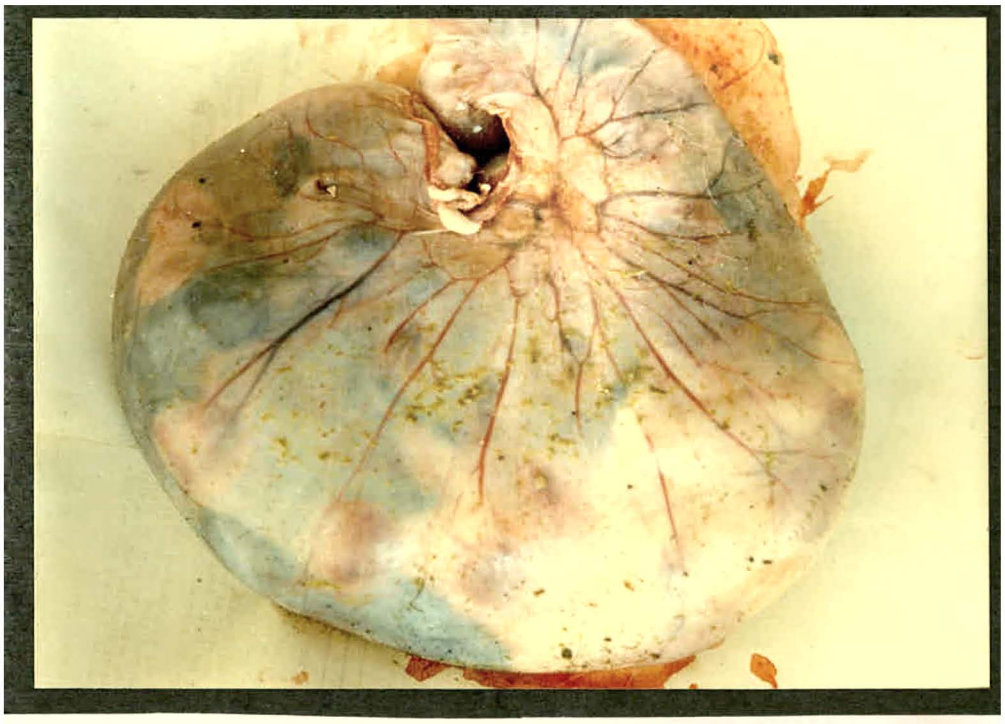


Fig. 27. Uterine wall with marked haemorrhages and discolouration of goat (Group B) in experimental rumen acidosis.

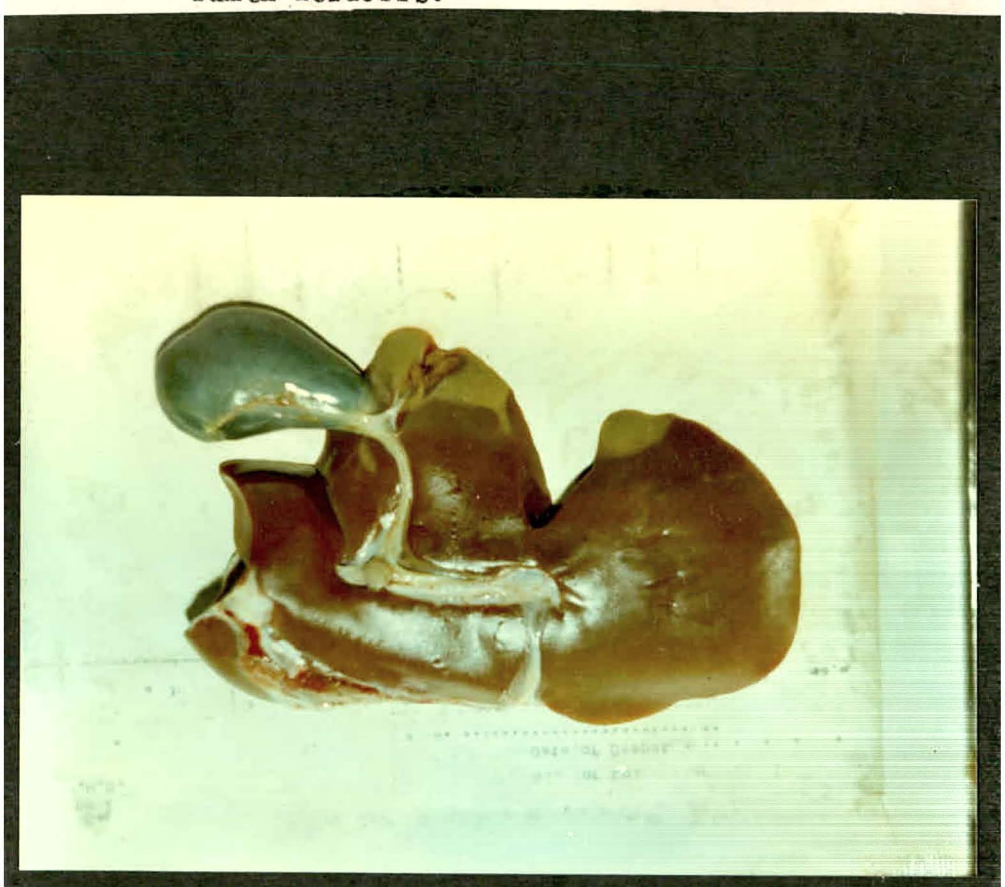


Fig. 28. Pale coloured liver of goat (Group B) in experimental rumen acidosis.

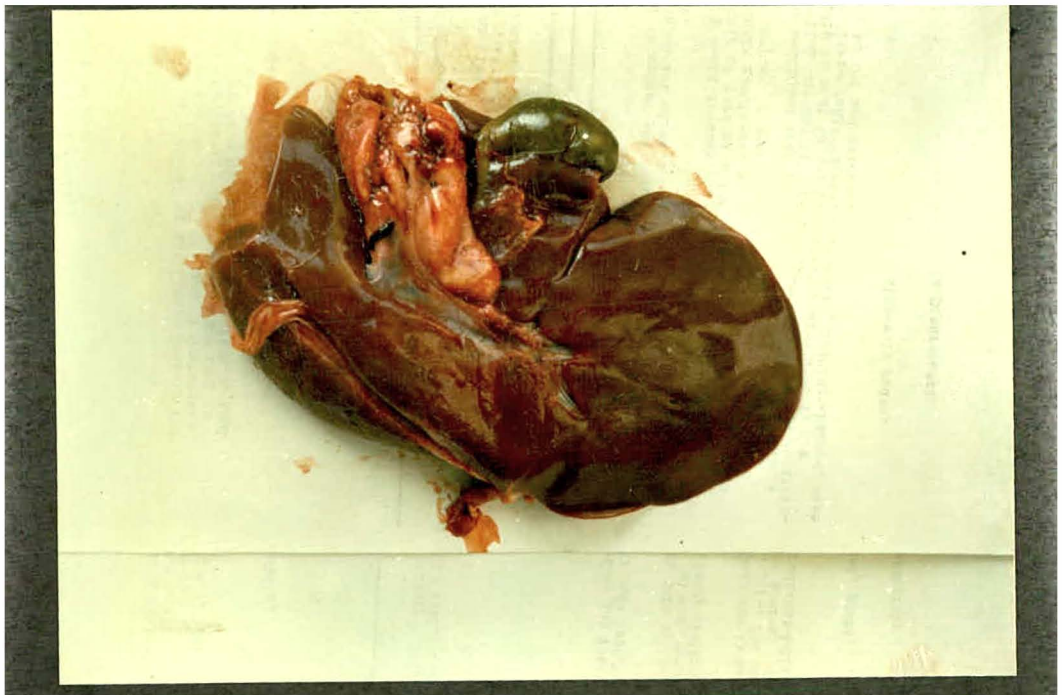


Fig. 29. Congested liver of goat (Group B) in experimental rumen acidosis.

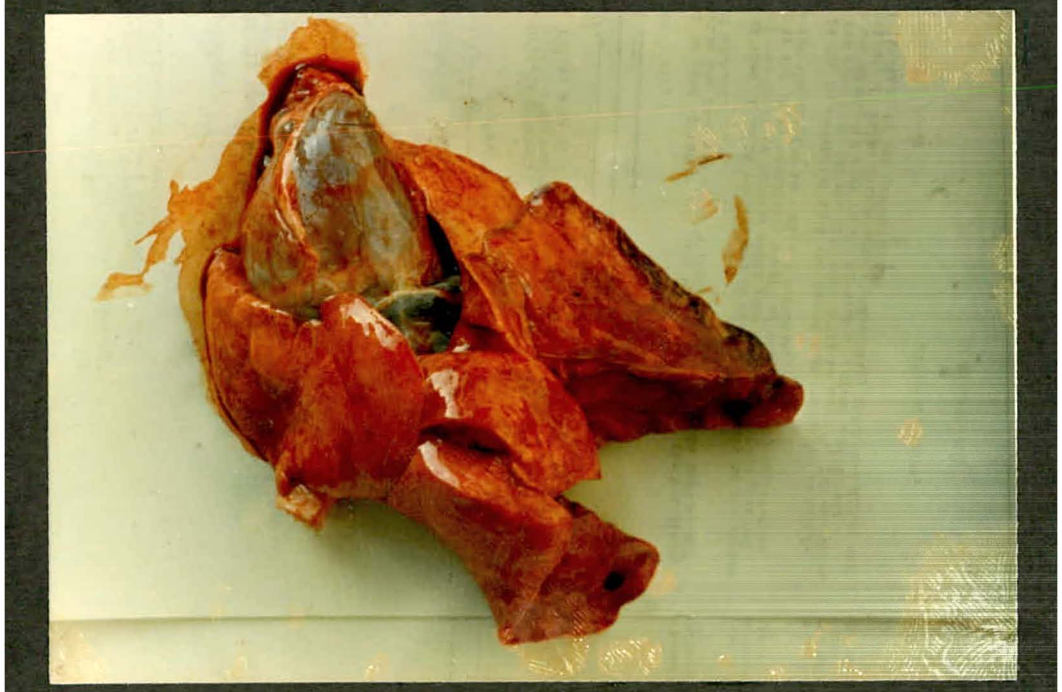


Fig. 30. Congested lungs of goat (Group B) in experimental rumen acidosis.

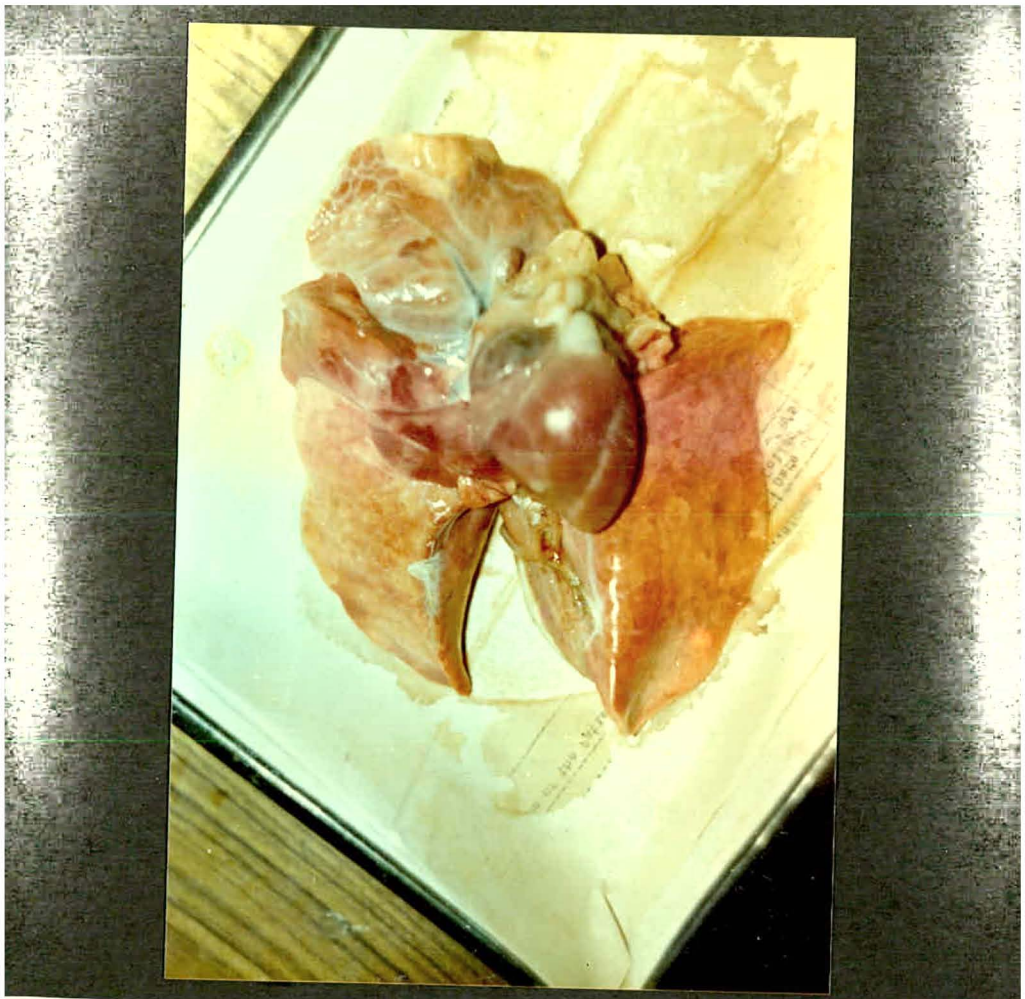


Fig. 31. Pale oedematous lungs with mucoid exudation and congested pericardium and myocardium of heart of goat (Group B) in experimental rumen acidosis.

mucoid exudation on cut surfaces of the parenchyma of the lungs and bronchioles (Fig. 31).

4.2.5.1.12 Heart

Severe congestion of pericardium and myocardium was observed from outside (Fig. 31).

4.2.5.1.13 Mesenteric lymph nodes

Mesenteric lymph nodes were swollen.

4.2.5.1.14 Pancreas, adrenal and thyroid gland

Did not show any gross changes

4.2.5.1.15 Brain

Macroscopically no apparent changes were observed in brain.

The necropsy findings of rumen, abomasum, liver and gall bladder were similar to the findings of Sen (1982), Das (1990) in goats. But there were no relevant informations of other organs in goats in lactic acidosis, So, present findings could not be compared. However, observations were similar to the observations of Hartig and Hebold (1973 a), Vestweber and Leipold (1974), Dshurov (1976 a) and Teli et al. (1986) in sheep and Udall (1972), Dshurov (1976 b) and Nauriyal et al. (1978) in cattle and buffaloes.

The emaciated carcasses with loss of skin elasticity, shunken eyes, congested conjunctival mucous membranes indicated marked hemoconcentration and dehydration. The distended, weighty abdomen indicated much withdrawal of fluid from vascular system into rumen. Sour smell rumen liquor was due to excessive lactic acid accumulation inside the rumen.

Enlargement of liver, with some necrotic patches, marked congestion and haemorrhages in the different organs might be due to local corrosive action of lactic acid (Dirksen, 1970; Vestweber and Leipold, 1974). It might not be only due to lactic acid. There were other toxic substances produced in the rumen like ethanol, methanol, histamine and bacterial endotoxin (Slyter, 1976). Congestion and oedematous changes of liver might be due inflammatory changes due to passage of bacteria through ruminal wall into liver.

4.2.5.2 Histopathological Studies

4.2.5.2.1 Pancreas

Exocrine pancreatic alveoli appeared very small and atrophic (Fig. 32,33). Cells lining the acini showed marked atrophy leading to depletion of cytoplasm and gathering of nuclei (Fig. 32). Some cases lining epithelial cells of the alveoli showed mild fatty changes and hyaline changes at places. Some atrophic pancreatic alveoli showed inflammatory changes with replacement fibrosis at places. Islets were markedly reduced in number and were mostly atrophic with degranulation

of the cytoplasm (Fig. 32, 33). These findings partly simulated the findings of Dzhurov (1975) in calves and coincided fully with the findings of Randhawa et al. (1981 b) in buffalo calves. There was no such relevant report on goats in ruminal acidosis. Hyperactivity in the early stages of ruminal acidosis might have led to the exhaustion and subsequent atrophy of endocrine beta cells and exocrine epithelial cells (Randhawa et al., 1981 b).

4.2.5.2.2 Adrenal gland

In adrenal cortex, zona glomerulosa was thin at places, sometimes appeared normal (Fig. 34). Zone fasciculata appeared hypertrophied (Fig. 34). The cells of the fascicular zone appeared pale stained and lipid rich (Fig.35), indicating hyperactivity. Some cells in this zone had more than 2 - 3 nuclei. The fascicular zone was broaded even upto the superficial capsule at some areas. Reticular Zone appeared normal. The medulla appeared normal. Similar findings were also reported by Randhawa et al. (1981 b) in buffalo calves. The hyperactivity of adrenal cortex might be due to stress and shock in lactic acidosis. The hyperactivity of adrenal cortex particularly Zona fasciculata was characterised by increase in secretion of cortisol hormone as obtained in this study.

4.2.5.2.3 Thyroid gland

Histologically thyroid gland showed atrophic changes of the follicles which indicated gross inactivity of the gland compared to normal one (Fig. 36,37). Present observation could not be compared due to nonavailability of literature in relation to acid indigestion. However, atrophy of follicles might be due to inhibition of pituitary TSH secretion by increased concentration of glucocorticoids which were released due to stress in digestive disorder.

4.2.5.2.4 Kidney

Most of the glomeruli appeared shunken with widen capsular space. Few glomeruli appeared atrophic, sometimes totally disappeared (glomeruli obsolescence) and hyalinized. Tubules showed cloudish swelling and fatty changes of lining epithelium. Most of the renal tubules of kidney showed coagulative necrosis and degenerative changes in the lining epithelial cells (Fig. 38). Hyaline casts were seen in the lumen of few tubules (Fig. 39). Blood vessels were thick walled. Patchy areas of haemorrhages were also evident (Fig. 39). Interstitial tissues were mildly oedematous with few infiltration of mononuclear cells in the interstial tissue. The above picture showed degenerative changes of kidney. This findings agreed with Delak and Adamic (1959), Vestweber and Leipold (1974),Dshurov (1976 a) in sheep and Dirksen (1970), Dshurov (1975), Nauriyal et al. (1978) and Randhawa et al. (1981 b) in buffaloes and cattle.

Atrophy of the glomeruli indicated hypoactivity and acute renal failure. Degenerative changes of renal tubules might be due to peripheral circulatory failure leading to ischemia and also due to toxic effect of lactic acid (Vestweber and Leipold, 1974).

4.2.5.2.5 Lungs

Section of lung showed accumulation of inflammatory cells mostly in the interstitial space (Fig. 40). Some showed extensive interstitial oedema (Fig. 40). Fare number of alveoli were dilated and a few appeared ruptured indicating alveolar emphysema (Fig. 41). Blood vessels were extensively congested. Bronchioles showed oedema of the wall, accumulation of inflammatory exudate in the lumen and infolding of the lining mucous membrane (Fig. 40). Some bronchiolar lining epithelium showed evidence of acute inflammatory reaction (Fig. 40). Peri-bronchiolar alveoli were packed with acute inflammatory exudate (Fig. 41). Blood vessels were extensively congested and haemorrhages (Fig. 41). Total picture was suggestive of pneumonitis. Similar findings agreed with the finding of Vestweber and Leipold (1974) and Dshurov (1976 a) in sheep and Randhawa et al. (1981 b) in cattle and buffaloes. These changes of lungs might be due to over exhausation of lungs caused by increased respiratory rate to combat acidosis. Pneumonic changes indicating multiplication of dormant bacteria in the respiratory tract due to severe stress condition.

4.2.5.2.6 Intestine

Mucous membrane appeared extensively congested with surface ulceration of the mucosa. Villous structures were completely denuded off. The inflammatory cells on the surfaces of mucous membrane consisted mostly of mononuclear cells with fare number of neutrophils (Fig. 42). Inflammatory reaction was limited to mucous membrane only. Submucous coat and muscle coat appeared normal in most cases. Serosal vessels were found congested in few cases. The mucosal glands appeared small and atrophic (Fig. 42). Similar findings were reported by Delak and Adamic (1959) and Vestweber and Leipold (1974) in sheep and Sethuraman (1976) and Randhawa *et al.* (1981 b) in calves. This indicated ulcerative enteritis due to toxic and corrosive effect of lactic acid and concurrent secondary bacterial infection.

4.2.5.2.7 Brain

Sections of brain tissues showed congestion of superficial blood vessels. Perivascular accumulation of inflammatory cells mainly lymphocytes known a perivascular cuffing (Fig. 43) was observed. Patchy areas of necrosis (Chromatolysis) with replacement glosis were also evident (Fig. 44). These were similar to those described in acidotic sheep (Vestweber and Leipold, 1974; Dshurov, 1976 a) and in cattle (Strafuss and Monlux, 1966; Vihan *et al.*, 1973 a and Randhawa *et al.*, 1981 b). The above picture of brain might be due to deficiency of thiamine (Brent, 1976). Passage of

undesirable substances like lactic acid, pyruvic acid, histamine etc. to brain through blood brain barrier might sometimes cause lesion in the brain (Losade et al., 1972; Mella Lizama, 1973).

4.2.5.2.8 Spleen

Sections of spleen tissues showed extensive vascular congestion and patchy areas of haemorrhages (Fig. 45, 46). Haemosiderin ladent cells were present in fare number throughout the whole section. Splenic capsules appeared thickened and fibrocholagenous lymphoid follicles were diminished. In one animal splenic tissue showed total loss of folicular architecture in the cortex (Fig. 46) suggestive of necrosis. Due to nonavailability of relevant informations in goats and other animals, the present study could not be compared. These changes of spleen might be suggestive of toxic effect of lactic acid and other toxic substances like amines, histamine, endotoxin produced during lactic acidosis.

4.2.5.2.9 Mesenteric Lymph node

Sections of lymph node showed congestion of capsular vessels, with pericapsular inflammatory reaction. Lymphoid follicles were very prominent, enlarged in size with prominent germ centre indicating hyperactivity. Sinusoidal spaces appeared dilated with mild proliferation of sinus lining endothelial cells. The total picture was that

of acute lymphadenitis with sinus histiocytosis. Present observations could not be compared due to nonavailability of literature in relation to acid indigestion in goats and other animals.

4.2.5.2.10 Myocardium

Sections of myocardium showed moderate degree of fatty changes with focal areas of necrosis and focal area of calcium deposition. Diffused accumulation of inflammatory cells in interstitial spaces. It is suggestive of focal myocarditis. Similar were the findings as reported by Delak and Adamic (1959), Vestweber and Leipold (1974) and Dshurov (1976 a) in sheep and Uddal (1972) and Nayriyal et al. (1978) in cattle and buffaloes. This degenerative lesions might be as a result of toxic damage caused by lactic acid or bacterial endotoxin. It might also be due to ischemic changes as a result of circulatory failure occured due to rapid dehydration.

4.2.5.2.11 Liver

Section of liver tissues showed accumulation of inflammatory cells in periportal spaces with increased periportal fibrosis. Evidence of intrahepatic cholestasis was present. Liver cells showed mild fatty changes with accumulation of bile pigment granules. Segmental coagulative necrosis of liver lobules with accumulation of chronic inflammatory cells were evident (Fig. 47, 50). Neutrophilic infiltrations

at places leading to the formation of microabscesses containing necrosed tissue within the centre lined by fibrocholagenous wall (Fig. 48). Focal granulometous lesions with foreign body giant cells formation and accumulation of histiocytes were also evident (Fig. 49). Pericholengitis and pericholengiolitis were also present. In one animal lobular architecture was present with extensive simusoidal congestion (Fig. 47). PAS (Periodic acid Schiff's) stained sections of liver revealed depletion of glycogen in the hepatocytes (Fig. 50). On pearl's stain there was no evidence of haemosiderin deposit. The above changes like, fatty changes, coagulative necrosis, microabscesses, sinusoidal congestion were previously observed by Delak and Adamic (1959), Thomson (1967), Dirksen (1970), Vestweber and Leipold (1974), Dshurov (1975), Brent (1976), Sethuraman (1976), Nauriyal et al. (1978), Randhawa et al. (1981 b), Sen (1982) and Ivanov et al. (1987). Fibrotic changes in the liver, accumulation of inflammatory cells, necrosis, fatty changes in the liver parenchyma indicated bacterial hepatitis and hepatitis due to toxic substances like lactic acid, amines, ethanol, methanol, endotoxin which were produced due to lactic acidosis. Accumulation of chronic inflammatory cells indicated chronic hepatitis or hepatosis.

Entry of pyogenic organisms like Spherophorus necrophorus (Dirksen, 1970), Corynebacterium pyogenes and Pasteurella Spp. (Nillo et al., 1967) to liver tissues from the denuded epithelial surface of the reticulo-rumen caused

abscess formation. Depletion of liver glycogen was agreed with the observations of Dunlop and Hammond (1965). The depletion may be due to hyperactivity of adrenal medulla.

4.2.5.2.12 Rumen

Necrosis and exfoliation of Stratified squamous keratinized epithelium lining the mucous membrane at places were observed in the rumen papilla (Fig. 51). Vacuolation of cytoplasm of epithelial cells in the stratum lucidum was also observed. Some microvesicles due to rupture of cell walls were also observed. Microvesicles were also present in stratum spinosum (Fig. 51,51). Massive neutrophilic infiltration was also observed in microvesicles leading to the formation of abscesses (Fig. 51,52). Neutrophilic infiltrations were sometimes observed in the lamina propria of mucous membrane. Sometimes mononuclear cells infiltration was also observed at places. Colonies of bacteria were also observed in the rumen papilla. Similar descriptions were presented by Sen (1982) and Das (1990) in goats, Hartig and Hebold (1973 a), Vestweber and Leipold (1974), Dshurov (1976 a), McManus (1977), Chihaya et al. (1988) in sheep and Jensen et al. (1954 a), Ahrens (1967), Franklin (1967), Vihan et al. (1973 b), Dshurov (1975), Landsverk (1978) and Randhawa et al. (1981 b) in cattle and buffaloes. The desquamation and ulceration of superficial layer of rumen papillae might be due to corrossive action of lactic acid and toxic metabolites (Jensen et al. 1954 a) and hypertonicity (Broberg, 1960). Vacuolated cells in the stratum lucidum

indicated hydropic degeneration. The abscess formation in the superficial layer might be due to invasion of bacteria e.g. Fusiformis necrophorus through erroded surface.

4.2.5.2.13 Reticulum

Desquamation of superficial layer of villi, marked infiltration of polymorphonuclear cells and mononuclear cells in the stratum lucidum and lamina propria of mucous membrane and formation of microvesicles in the stratum lucidum were also observed. There was formation of microthrombi in the villi (Fig. 53). Present observations similar to those described by Hartig and Hebold (1973 a) in sheep. These changes were due to corrosive action of lactic acid and invasion of bacteria like Fusiformis necrophorus through erroded surface.

4.2.5.2.14 Abomasum

The surface epithelium of tunica mucosa was completely denuded off. The glandular structures in the lamina propria became atrophic and in most places completely degenerated. The epithelial lining of the glandular structures was lost. The lamina propria was infiltrated with mononuclear cells and sometimes polymorphs (Fig. 54). Muscle coat and submucous coat remained normal. These changes might be due to corrossive effect of lactic acid and toxic metabolites. Infiltration of mononuclear and polymorphs were due to secondary bacterial invasion. These findings were similar to the finding of Vihan et al. (1973 a) in buffaloes.

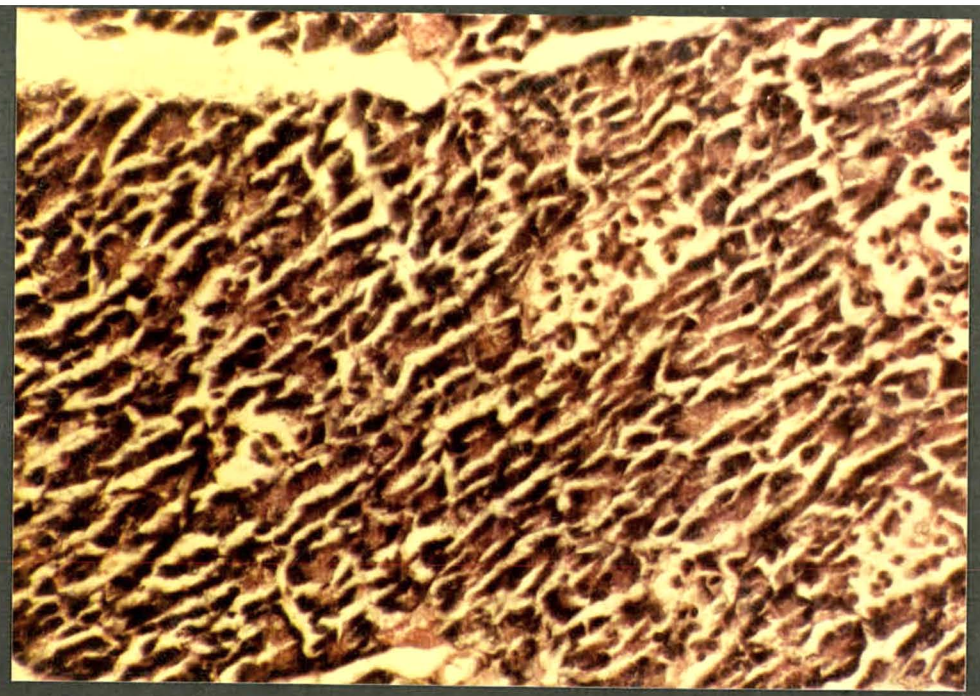


Fig. 32. Section of pancreas showing atrophic exocrine alveoli with atrophic islets containing granular appearance islet cells of goat (Group B) in experimental rumen acidosis. H & E x 100.

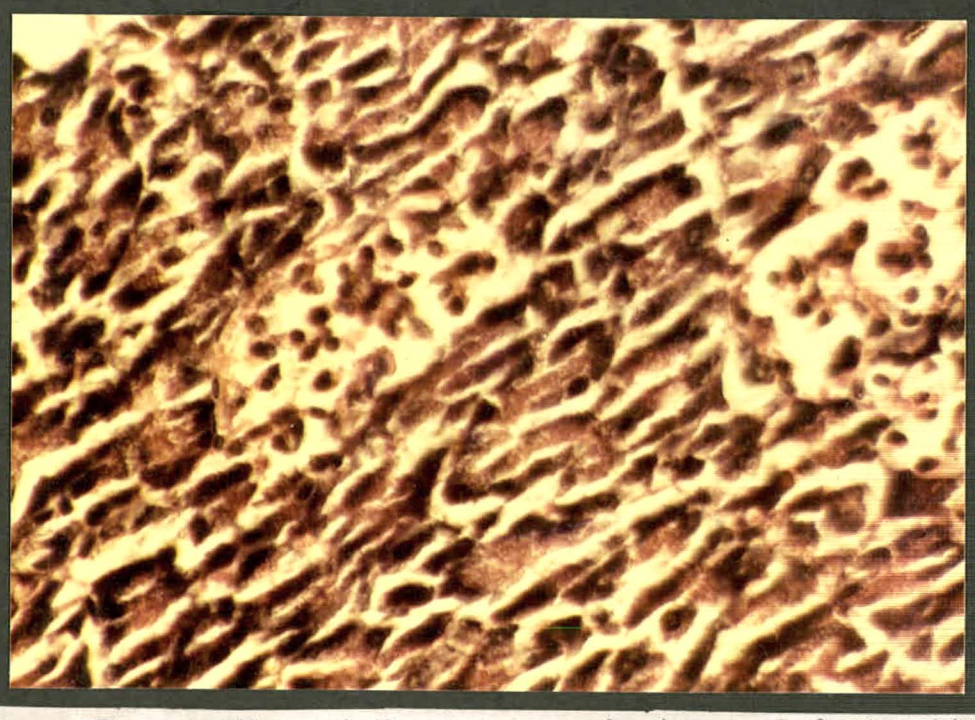


Fig. 33. Section of the pancreas showing marked atrophied acini with depletion of cytoplasm of cells lining the acini and reduced number of islet cells, which are atrophied with degranulation of the cytoplasm of goat (Group B) in experimental rumen acidosis. H & E x 450.

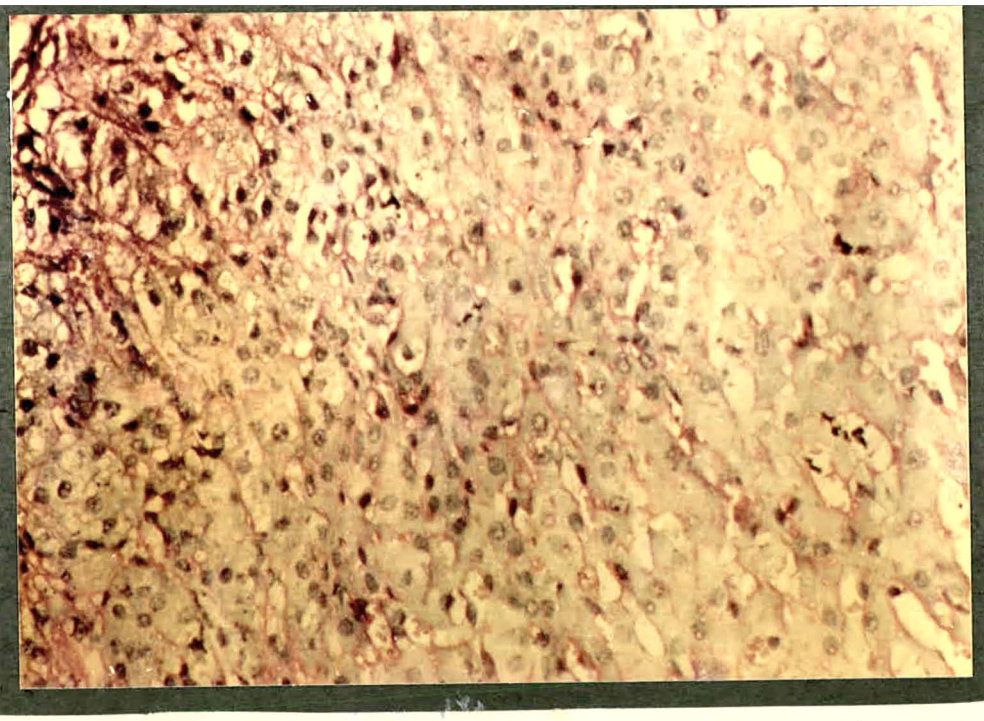


Fig. 34. Section of the adrenal cortex showing thin zona glomerulosa and hypertrophied zona fasciculata of goat (Group B) in experimental rumen acidosis. H & E x 100.

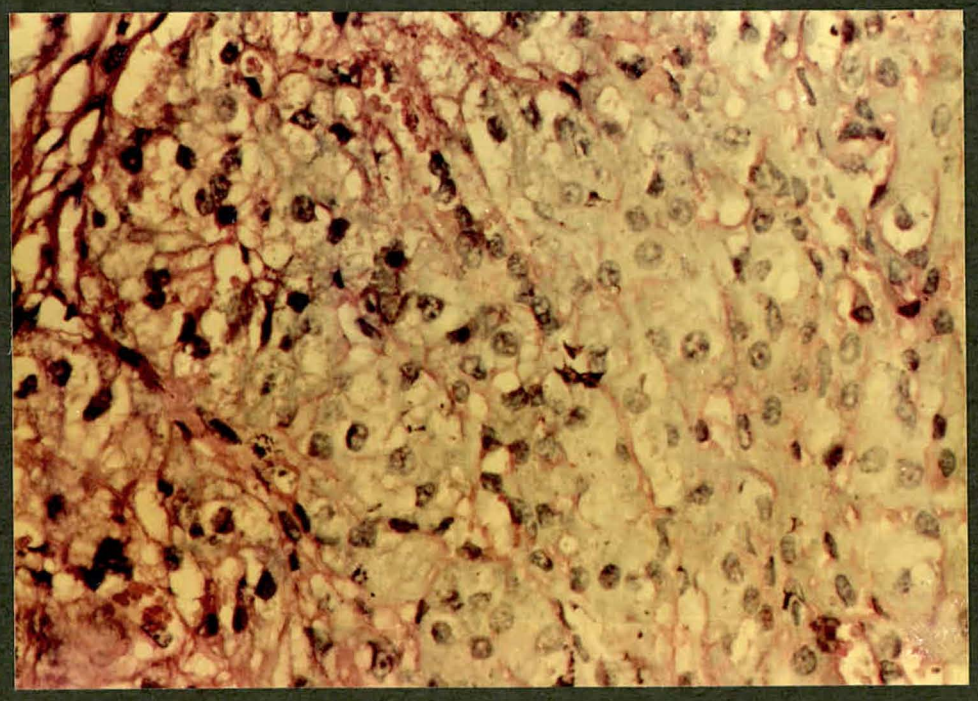


Fig. 35. Section of the adrenal cortex showing pale staining, lipid rich and hypertophied zona fasciculata in experimental rumen acidosis of goat (Group B). H & E x450.

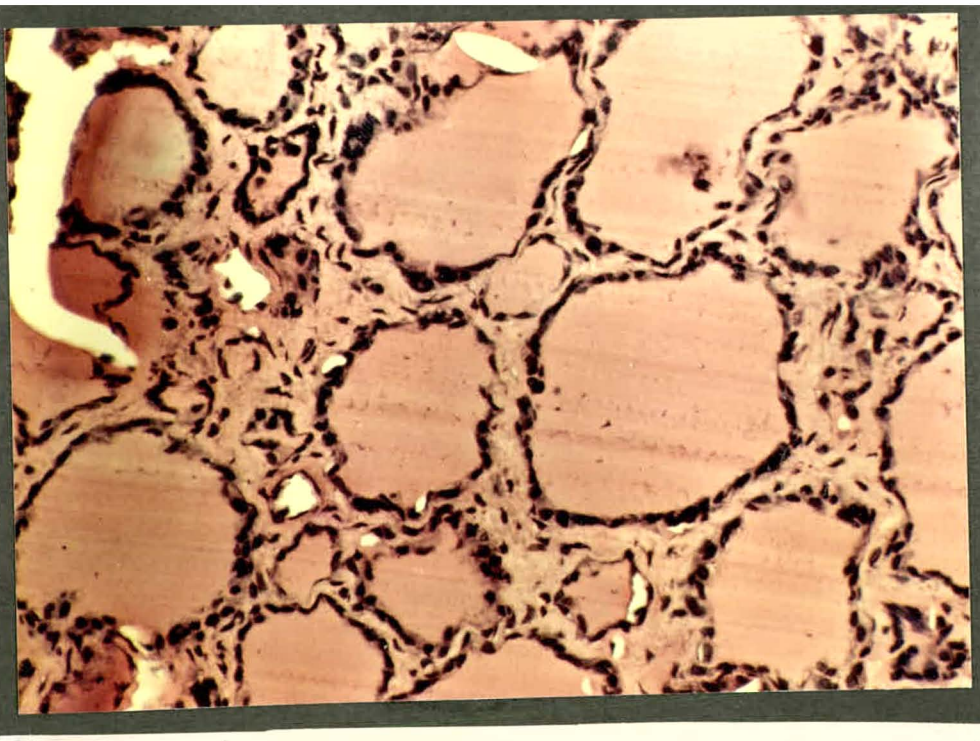


Fig. 36. Section of the thyroid gland showing normal thyroid follicles of goat. H & E x 100.

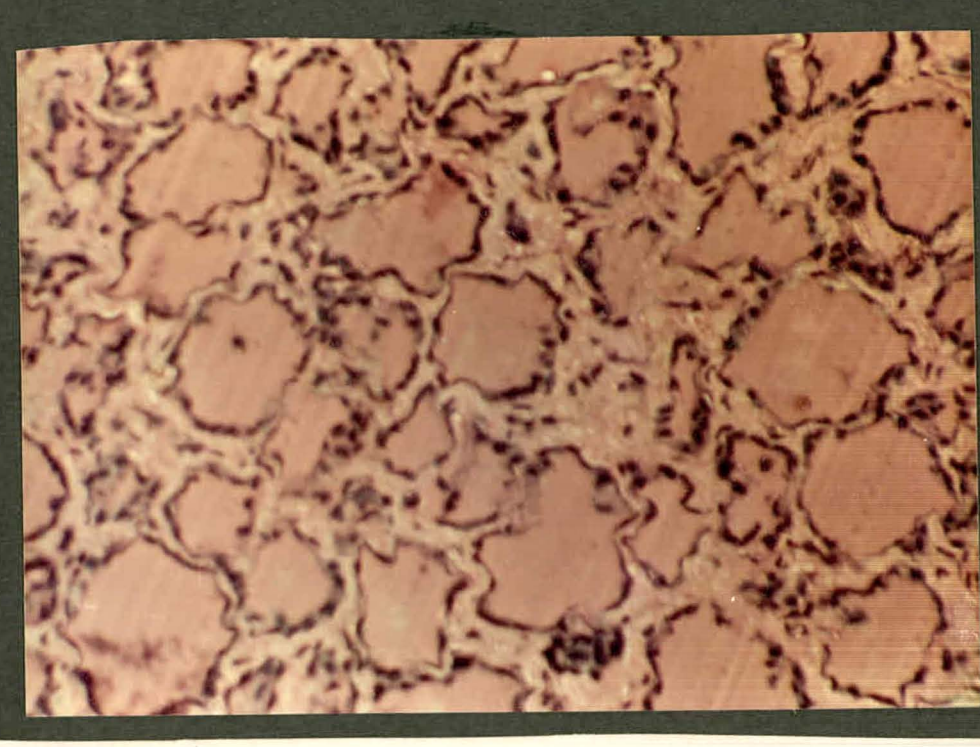


Fig. 37. Section of the thyroid gland showing atrophied thyroid follicles of goat (Group B) in experimental rumen acidosis H & E x 100.

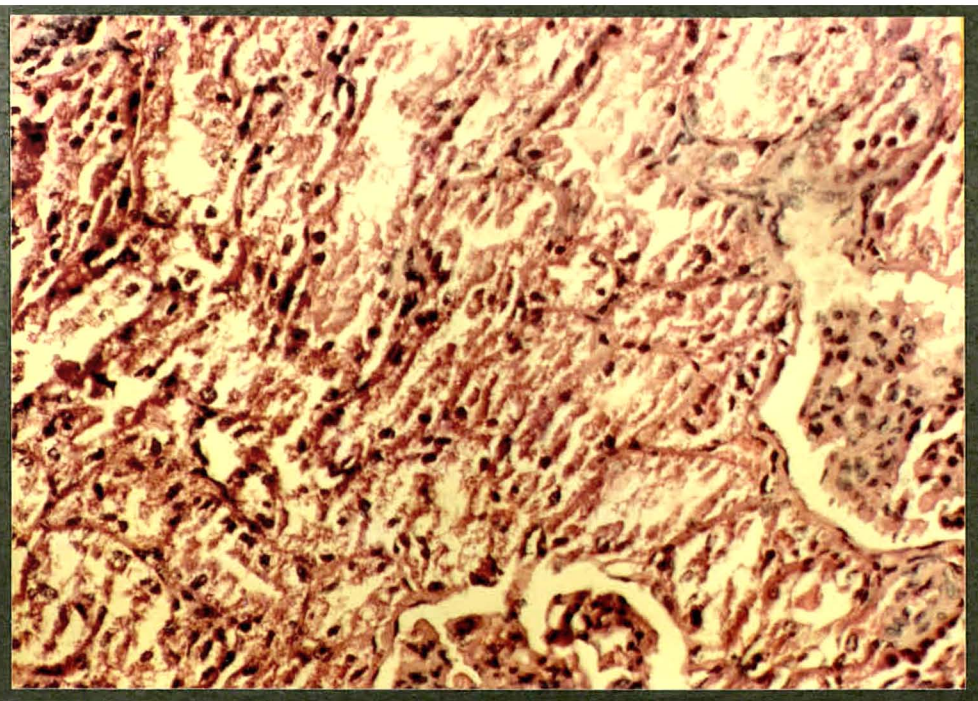


Fig. 38: Section of the Kidney showing shunken glomeruli with widen capsular spaces, few atrophied and hyalinized. Renal tubules showing coagulative necrosis in the lining epithelial cells of goat (Group B) in experimental rumen acidosis. H & E x 100.

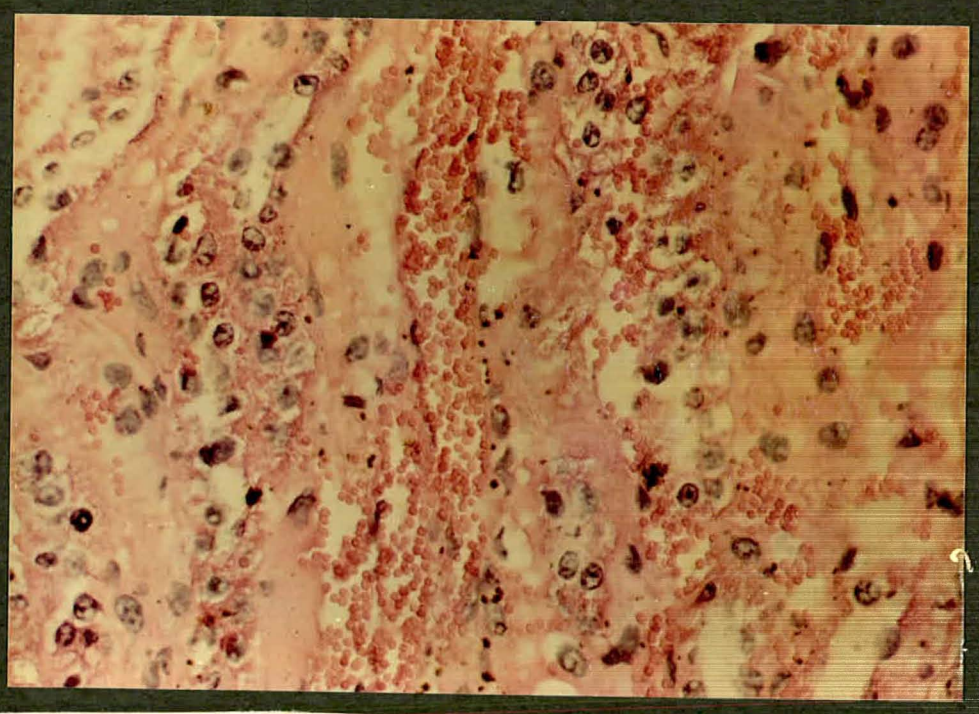


Fig. 39. Section of the kidney showing hyaline casts in the lumen of the tubules and patchy areas of haemorrhages in the parenchyma of goat (Group B) in experimental rumen acidosis. H & E x 450.

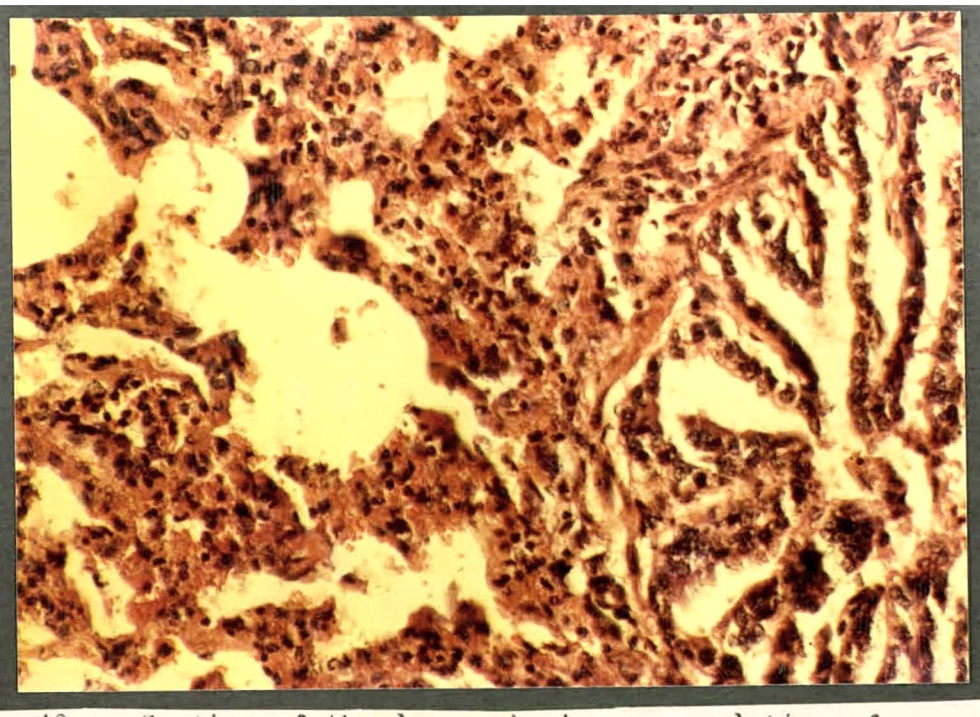


Fig. 40. Section of the lungs showing accumulation of inflammatory cells mostly in the interstitial spaces and extensive interstitial oedema. Bronchioles showing oedema of the walls, accumulation of inflammatory exudate in the lumen and in folding lining mucous membrane in experimental rumen acidosis of goat (Group B). H & E x 100.

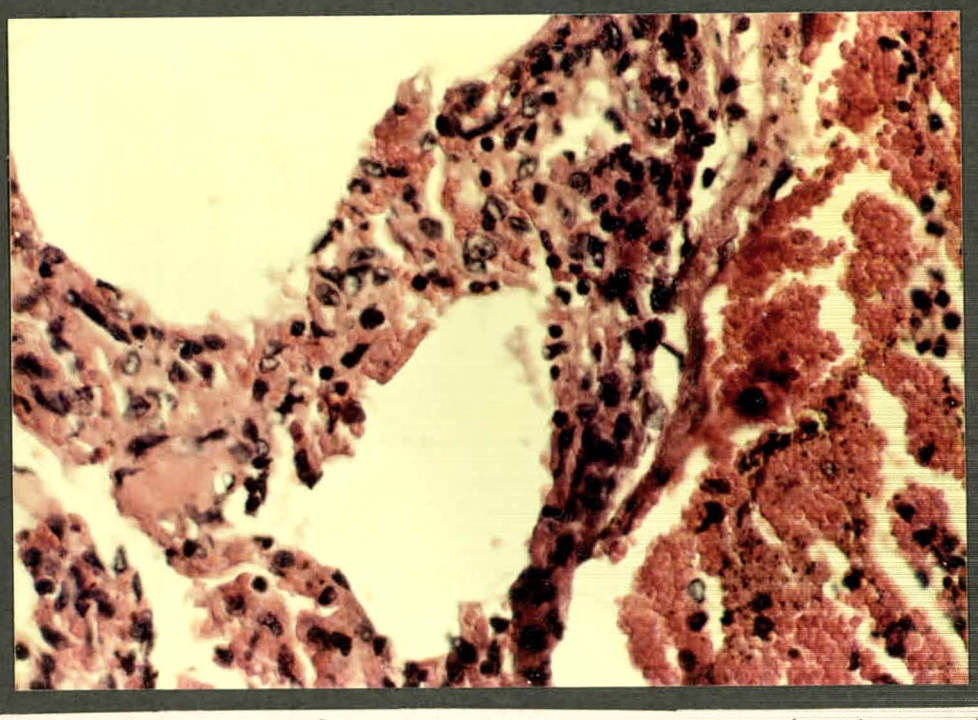


Fig. 41. Section of lungs showing severe congestion, haemorrhages accumulation of exudate in the alveoli and emphysema of alveoli of goat (Group B) in experimental rumen acidosis. H & E x 450.

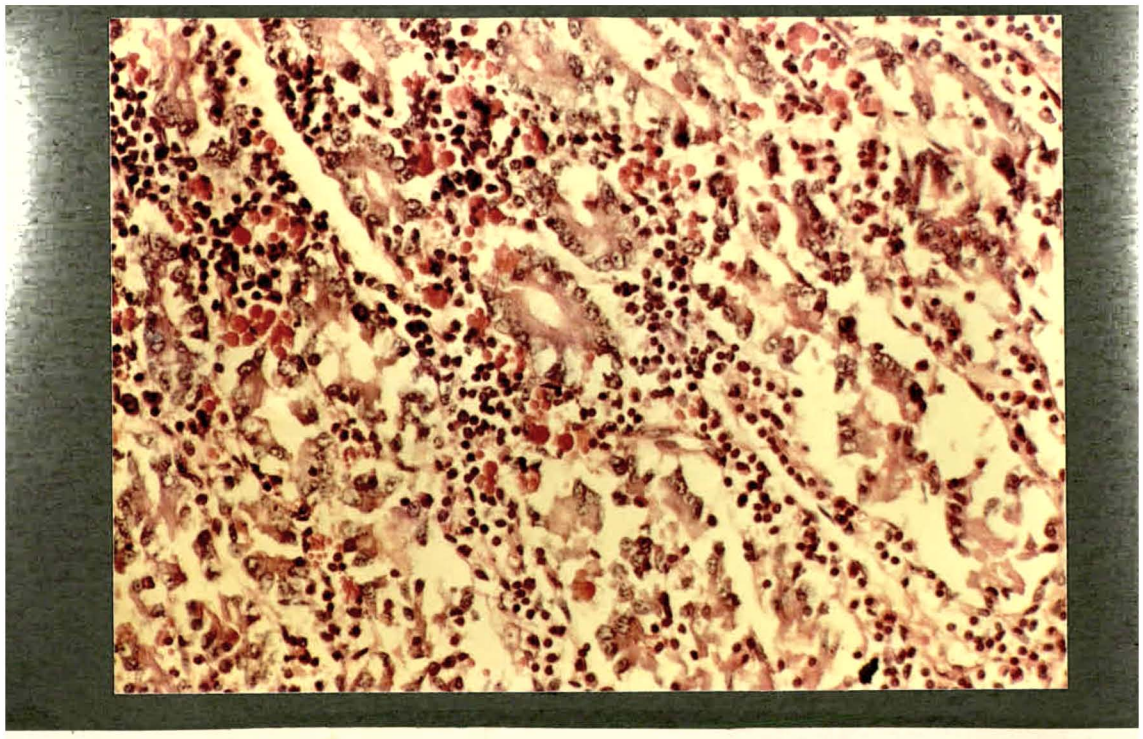


Fig. 42. Section of the intestine showing complete denudation of villus structure, infiltration of mononuclear cells with fare number of neutrophils and atrophic and degenerated mucosal glands of goat (Group B) in experimental rumen acidosis. H & E x 100.

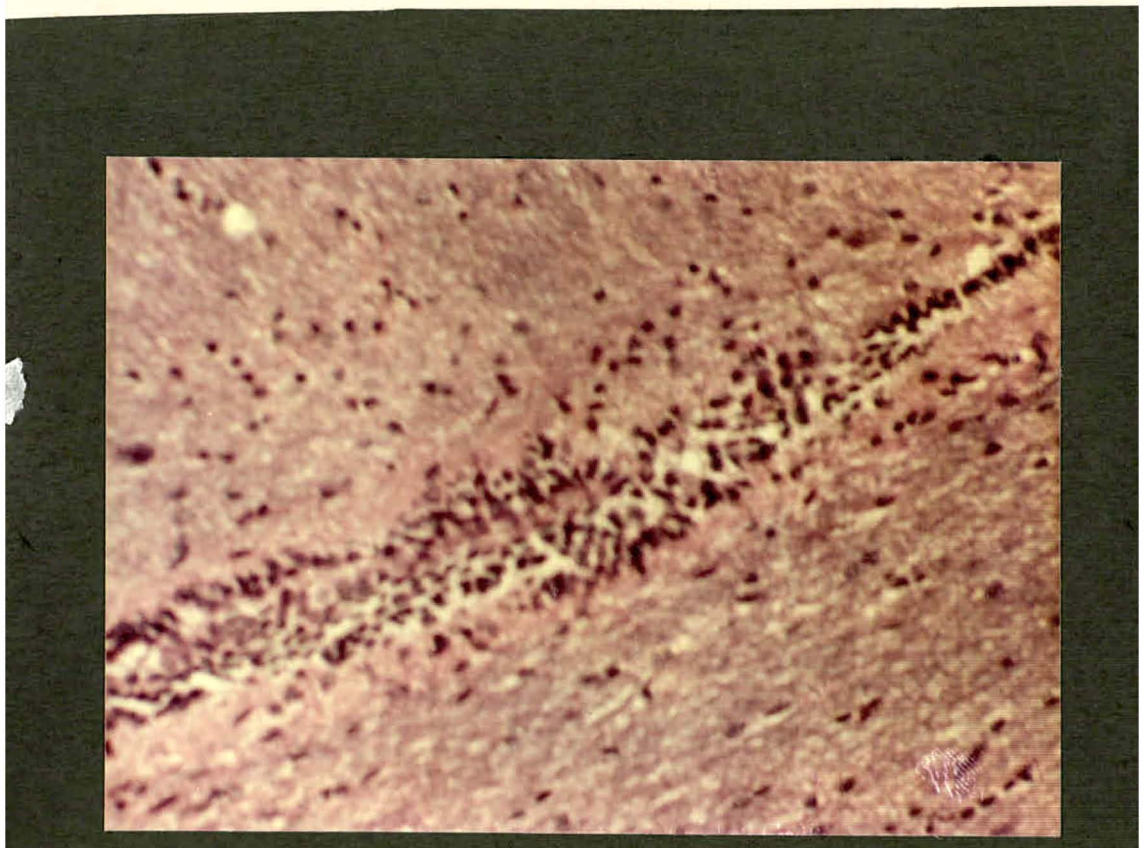


Fig. 43. Section of the brain showing perivascular cuffing of goat (Group B) in experimental rumen acidosis. H & E x 100.

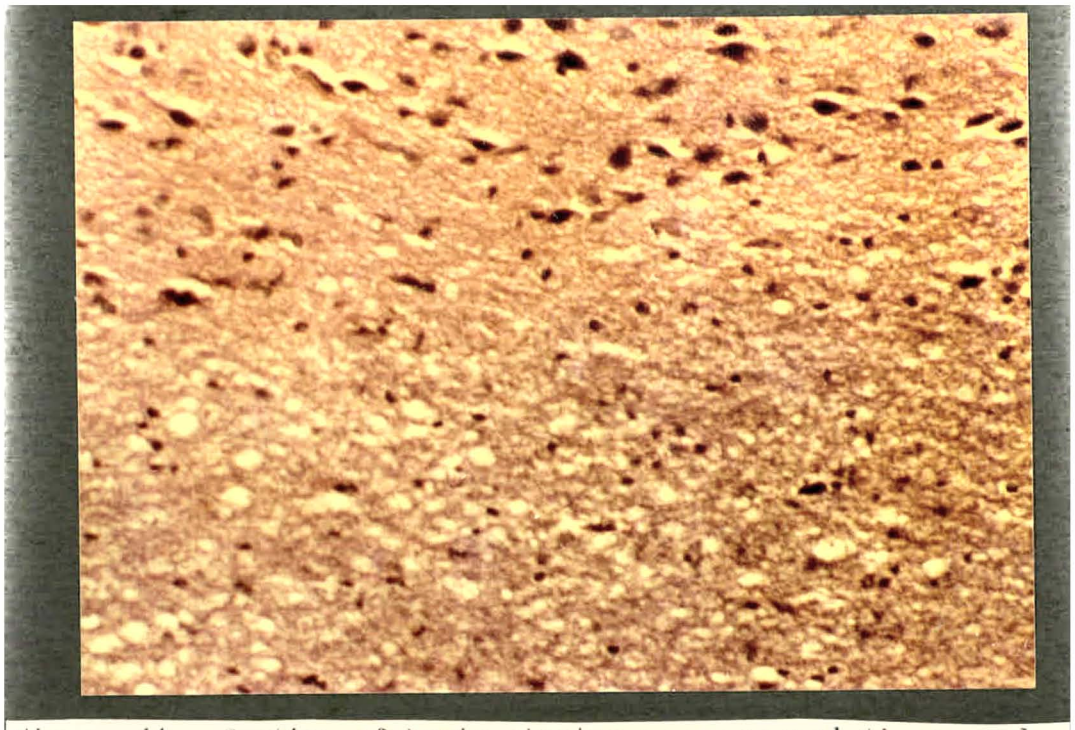


Fig. 44. Section of brain showing upper normal tissue and lower degenerated tissue containing chromatolysis, gliosis and fatty changes of goat (Group B) in experimental rumen acidosis. H & E x 100.

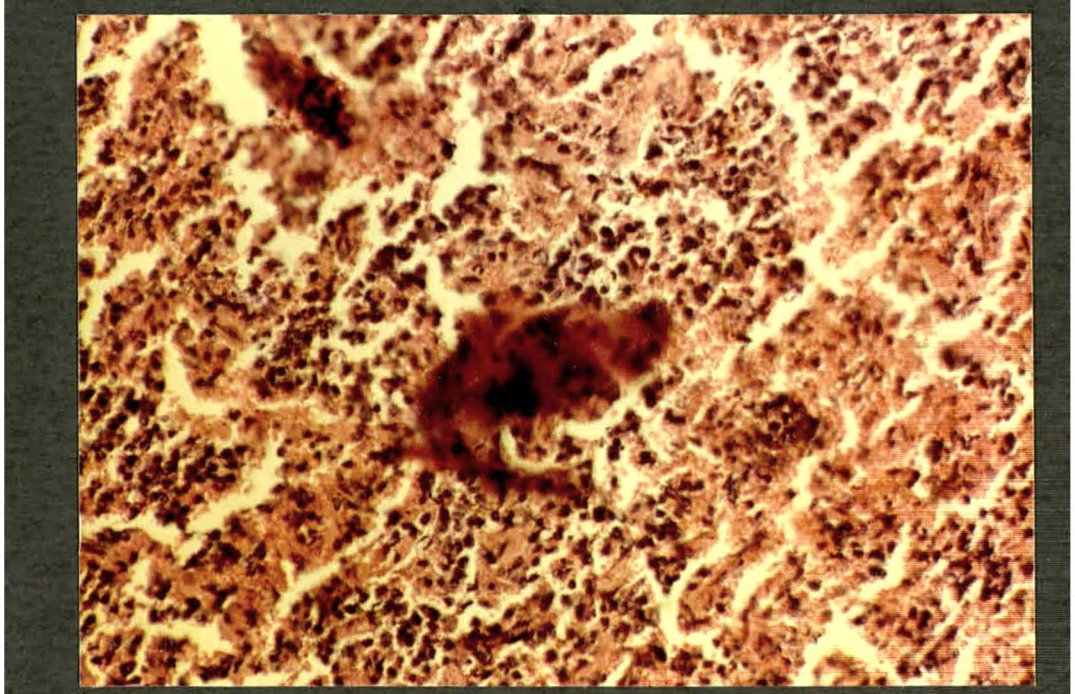


Fig. 45. Section of the spleen showing extensive vascular congestion and patchy areas of haemorrhages of goat (Group B) in experimental rumen acidosis. H & E x 100.

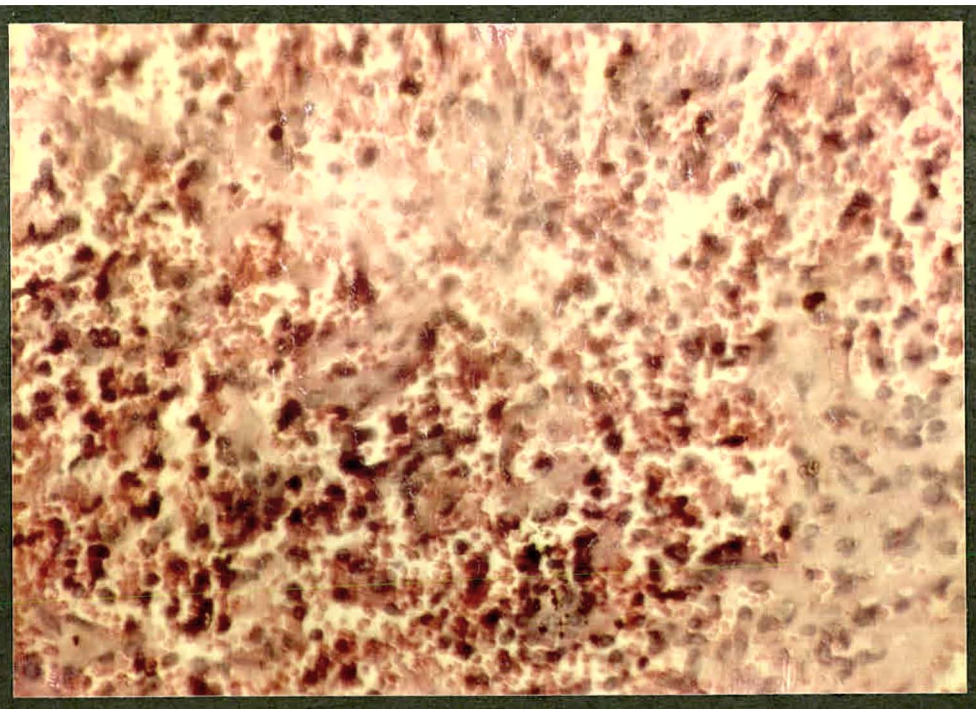


Fig. 46. Section of the spleen showing total loss of follicular architechture in the spleenic cortex with the [illegible] massive congestion in experimental rumen acidosis of goat (Group B). H & E x 450.

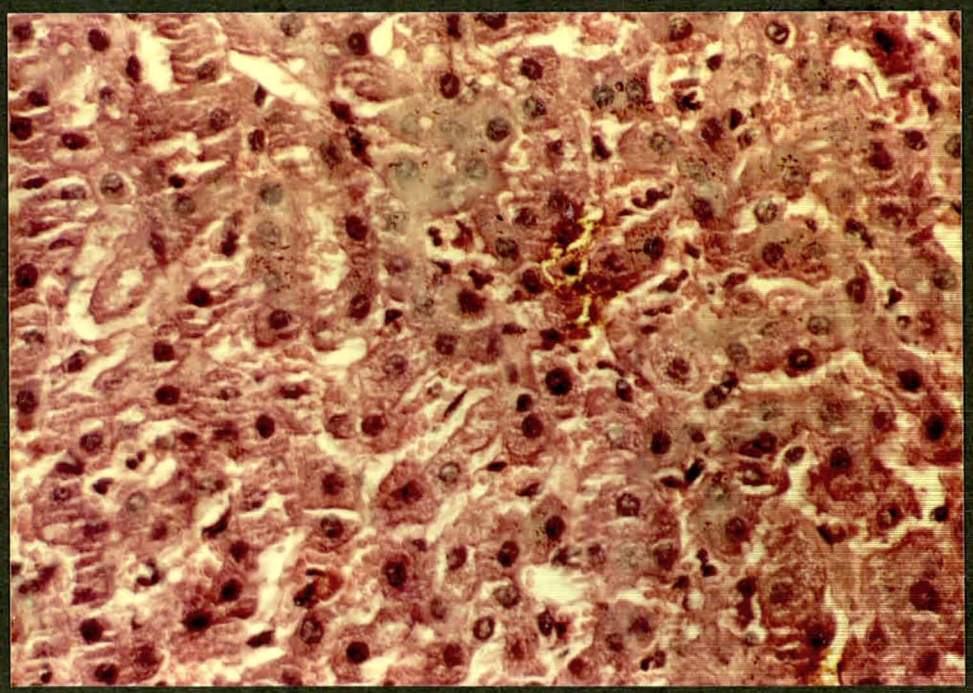


Fig. 47. Section of the liver showing coagulative necrosis of cells with sinusoidal congestion in experimental rumen acidosis of goat (Group B). H & E x 450.

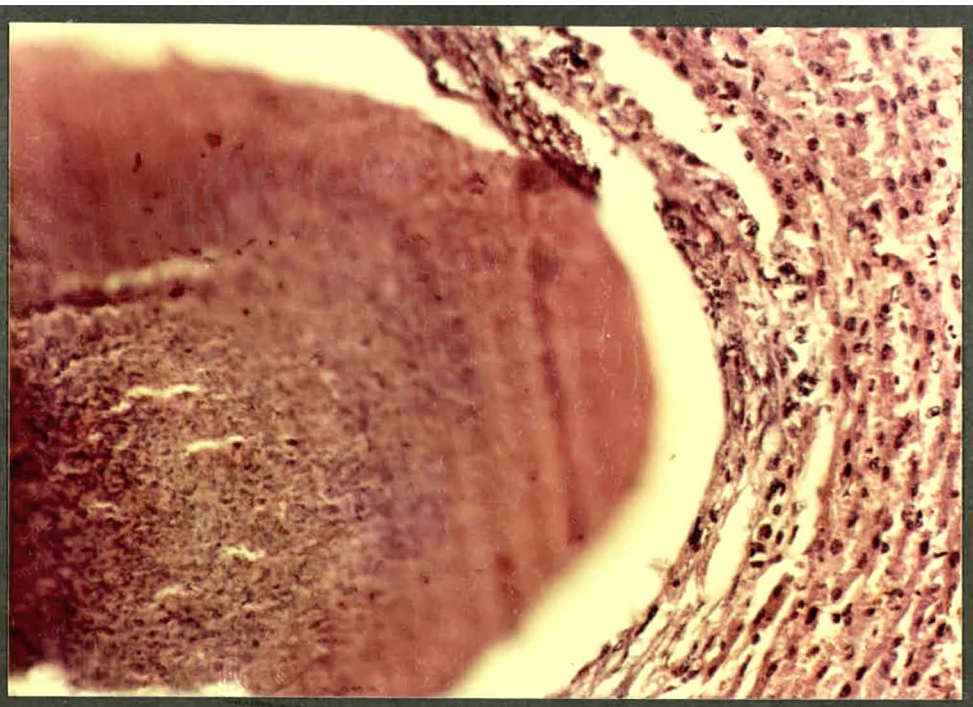


Fig. 48. Section of the liver showing neutrophilic infiltrations at places leading to the formation of microabscess containing necrosed tissues within the centre linned by fibrocholagenous wall of goat (Group B) in experimental rumen acidosis. H & E x 100.

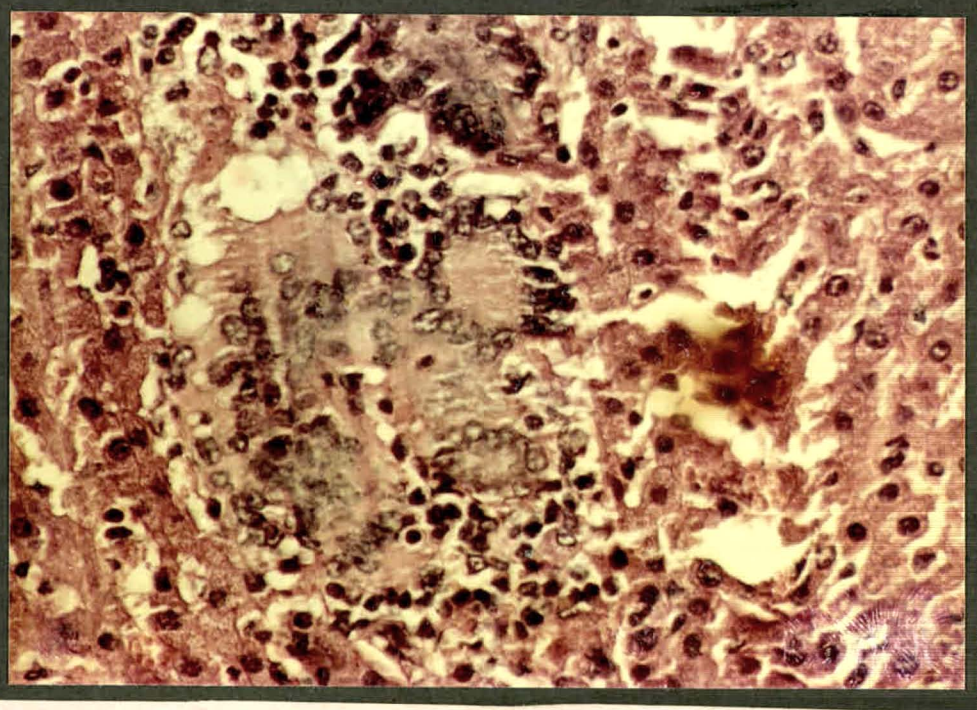


Fig. 49. Section of the liver showing focal granulomatous lesious with foreign body giant cells formation and accumulation of histiocytes with neutrophilic infiltration in experimental rumen acidosis of goat (Group B). H & E x 450.

Fig. 50. Section of the liver tissue showing depletion of glycogen in the hepatocytes and coagulative necrosis of cells on PAS staining in experimental rumen acidosis of goat (Group B). PAS x 100.

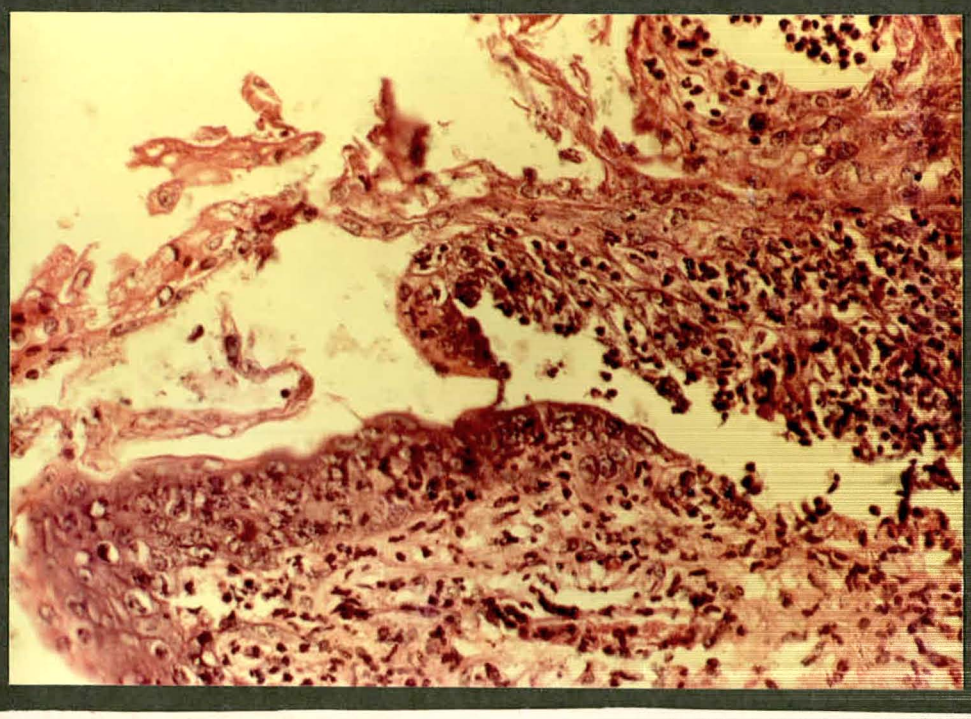


Fig. 51. Section of the rumen showing exfoliation of stratified squamous keratinized epithelium lining the mucous membrane of the rumen papillae. Infiltration of neutrophils and some times mononuclear cells of microvesicles and macrovesicles leading to the formation of abscesses in the rumen papillae are shown in experimental rumen acidosis of goat (Group B). H & E x 100.

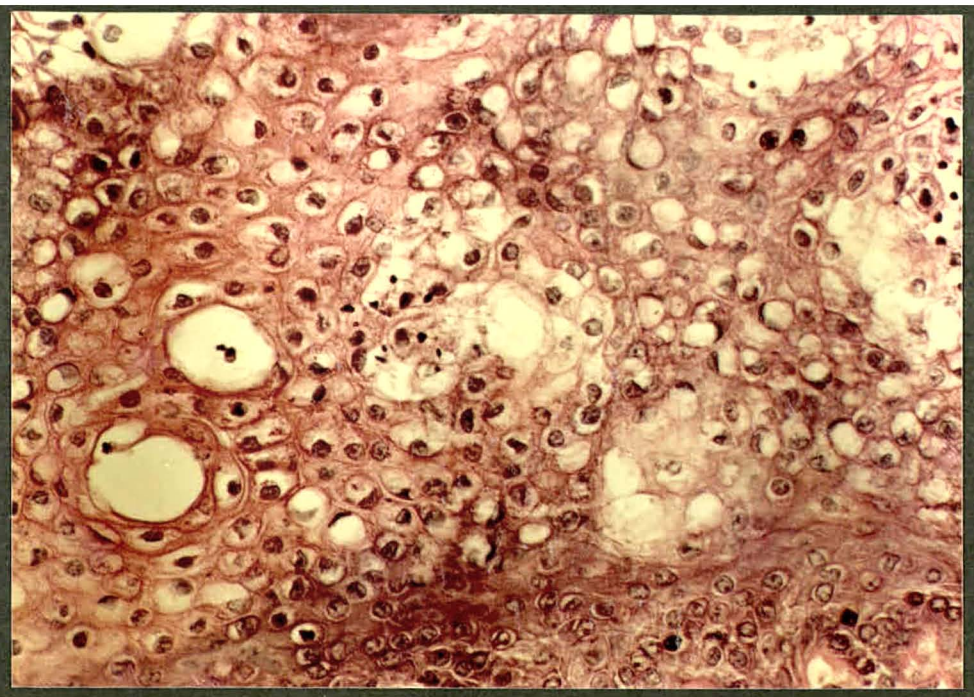


Fig. 52. Section of the rumen papillae showing extensive microvesicles formation in the stratum lucidum of rumen papillae with infiltration of neutrophils in experimental rumen acidosis of goat (Group B). H & E x 100.

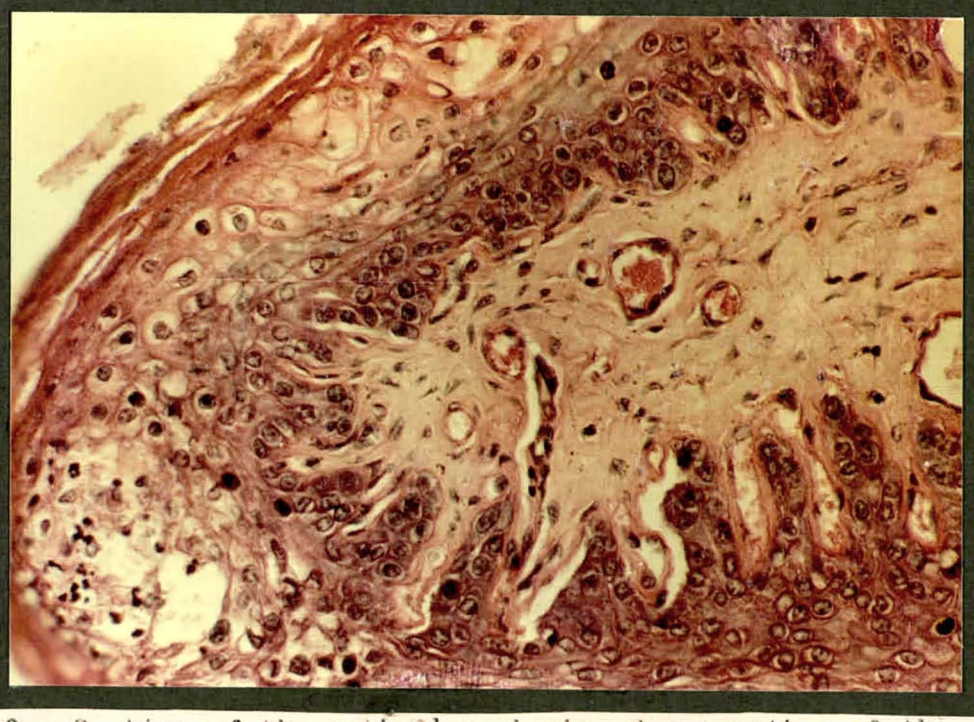


Fig. 53. Section of the reticulum showing desquamation of the superficial layer of villi, polymorphonuclear and mononuclear cells infiltration in the stratum lucidum and lamina propria and formation of microvesicles and macrovesicles in the stratum lucidum of goat (Group B) after induction of experimental rumen acidosis. H & E x 100.

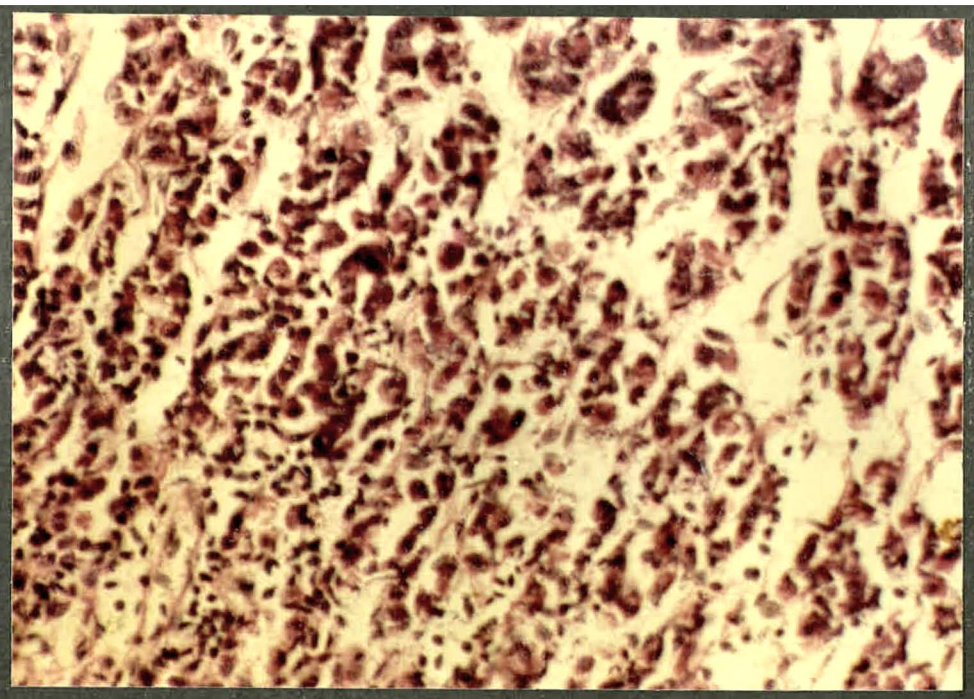


Fig. 54. Section of the abomasal tissue representing atrophic and degenesated glandular acini and infiltrations of mononuclear cells in the lamina propria of goat (Group B) after induction of rumen acidosis. H & E x 100.

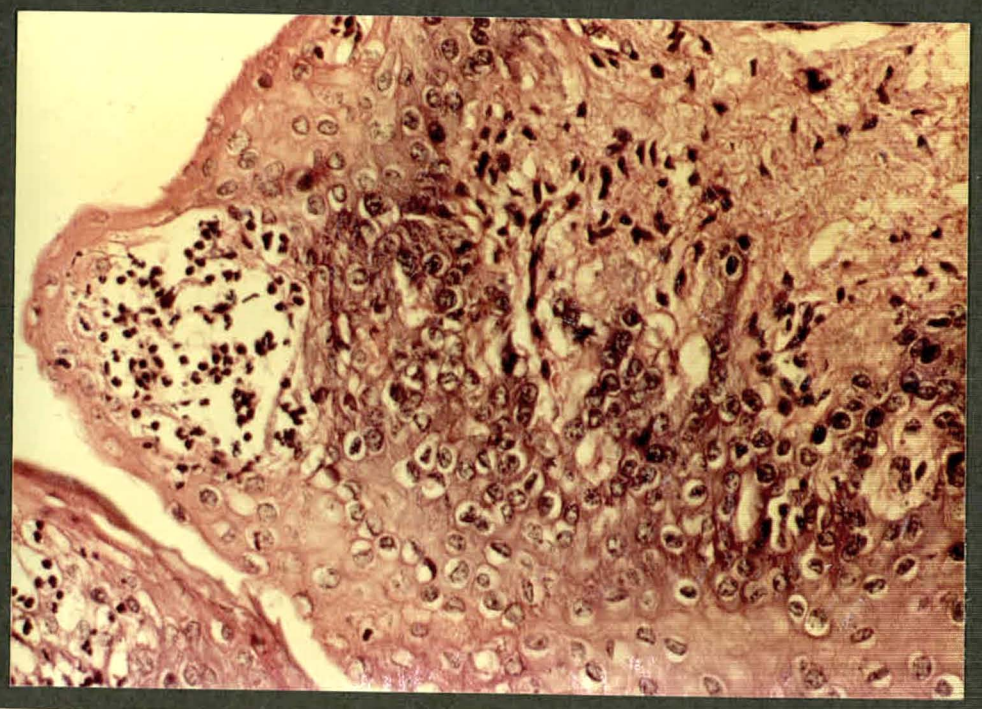


Fig. 55. Section of the omasum showing massive neutrophils infiltration of lamina propria within the microvesicles. It also showing macrovesicles formation with infiltration of mononuclear cells in epithelial surfaces of goat (Group B) in experimental rumen acidosis. H & E x 100.

4.2.5.2.15 Omasum

There was massive neutrophilic infiltration of lamina propria within the microvesicles. Stratum corneum of the papilla desquamated at places. Epithelial surfaces were massively infiltrated with mononuclear cells (Fig. 55). Sometimes colonies of bacteria were seen in the epithelial surfaces. Muscle layer of the papilla remained normal. Infiltration of inflammatory cells might be due to invasion of bacteria through the erroded surface of omasum. Due to lack of relevant literatures on goats and other animals the present findings could not be compared.

4.3 Therapy

The results of therapeutic trials on the basis of ARD profiles (Anorexia, rumen dysfunction and defaecation) of goats in three category suffering from mild (comprising of C_1 = experimental cases and N_1 = natural cases), moderate (comprising of N_2 = natural cases) and severe (comprising of N_3 = natural cases and C_2 = experimental cases) lactic acidosis respectively are presented as below.

4.3.1 The efficacy of treatment of mild acidotic group containing 12 goats as were brought under the treatment with 'Floratone bolus' intraruminally in experimental cases and orally in natural cases and evacuation of rumen content only in experimental cases. Floratone bolus as test agent is presented in Table - 19. The results of analysis of variance of different parameters between treatments (Pre and Post treatments) are also shown in Table - 20.

Table - 19. Trials with Floratone bolus as Test Agent against mild Acidosis in Goats based on ARD Profiles

No. of goats treated		12	
Duration of treatment (days)		5	
No. of goats recovered		12	
Percentage recovered		100 %	
	Pre-treatment values	Post-treatment values	
pH of Rumen liquor	5.97[a] ±0.087	6.79[b] ±0.049	Mean S.E.
Rumen motility rate/5 minutes	2.92[a] ±0.335	7.42[b] ±0.193	Mean S.E.
Protozoal motility	(+/-)	(+++)	
CDT(hrs.)	81.00[a] ±2.63	46.28[b] ±2.64	Mean S.E.
SAT(mts.)	69.00[a] ±3.20	27.43[b] ±0.85	Mean S.E.
Appetite	Inappetence	Good appetite	
Faecal Character	Semidolid	Pelleted	

+/- = Slow to absence of protozoal motility

+++ = Vigorous protozoal motility

The complete recovery was obtained in 12 (100%) of 12 affected goats with a mean pretreatment values of pH of rumen liquor, rumen motility rate, motility of protozoa, cellulose digestion time (CDT), sediment activity time (SAT), faecal character and appetite as 5.97 ± 0.087, 2.92 ± 0.335/5 minutes,

Table - 20 . Analysis of variance of pre and post treatment values of different parameters in mild acidosis of goats.

Source	d.f.	M. S.			
		pH of R.L.	Rumen motility rate	CDT	SAT
Between treatment	1	3.96**	121.47**	4114.28**	6048.64**
Error	22	0.06	0.90	18.78	38.48

(** P/0.01)

slow to absent (+/-), 81.00 ± 2.63 hours, 69.00 ± 3.20 minutes, semisolid and inappetence respectively which changed significantly (P/0.01) to 6.79 ± 0.049, 7.42 ± 0.193/5 minutes vigorous (+++) 46.28 ± 2.64 hours, 27.43 ± 0.85 minutes, good appetite and pellated in nature after treatment respectively (Fig. 56, 57, 58 and 59).

Therapeutic trial with Floratone bolus in acid indigestion in cattle was previously adopted by Chakrabarti (1990) (Personal communication). But, no comprehensive studies were made on Floratone bolus in acid indigestion of goats.

The above findings showed that there was improvement of pH of rumen liquor, rumen motility, protozoal motility, CDT, SAT, faecal character and appetite. Percentage of recovery after treatment was 100%.

In mild acidosis, animals do not suffer from severe dehydration, toxaemia, liver dysfunction and lactiacedemia. So, death rate is minimum or nil after adoption of suitable therapy.

The individual component of the drug (Floratone) was found to exert beneficial effect in restoration of normal ruminal function.

In mild acidosis, pH of rumen liquor was not changed markedly and it was corrected by alkaliser sodium bicarbonate, sodium phosphate and magnesium trisillicate. The improvement of rumen motility rate might be due to improvement of rumen pH. Methionine as used has been found to have role to resusscitate

liver functions (Misra and Singh, 1974). Copper sulphate and cobalt sulphate have been used to stimulate the growth of microbial population (Joshi, 1976). Brewer's yeast has been used as it has been pointed out that dyspepsia due to ruminal acidosis, caused by accidental over feeding of grain have been treated with success by giving vitamin B or brewer's yeast and sodium bicarbonate (Dirksen, 1970).

The improvement of character of faeces from semi-solid to pelleted might be due to correction of hyperosmolality of rumen content and intestine and reduced inflammatory conditions of intestines. The reduction of normal microflora in the rumen stimulates the growth of B. thiaminolyticus which produce thiaminase enzyme causing subclinical thiamine deficiency. Thiamine is given orally with the basis that thiamine can be inactivated by the primary substrate thiaminase. Nicotinamide has been used to correct the deficiency of nicotinamide along with thiamine in acid indigestion (Thomas, 1986).

4.3.2 Treatment of moderate acidotic goats containing 10 animals of natural cases as were brought under the treatment of Steclin bolus orally, Gelusil MPS liquid orally and injection of Berin intramuscularly as test agents are presented in Table - 21. The results of analysis of variance of different parameters between treatments (Pre and post treatments) are shown in Table - 22.

Table - 21. Trials with Steclin bolus, Gelusil MPS liquid and injection of Berin as Test Agents against moderate acidosis in goats based on ARD profiles.

No. of goats treated	10	
Duration of treatment (days)	5	
No. of goats recovered	8	
Percentage of recovery	80%	
	Pre-treatment values	Post-treatment values
pH of rumen liquor	4.94[a] ±0.08	6.53[b] ±0.068 Mean S.E.
Rumen motility	0.50[a] ±0.224	7.00[b] ±0.189 Mean S.E.
Protozoal motility	-	+++
CDT (hrs.)	A[a]	49.00[a] ± 0.757 Mean S.E.
SAT (mins.)	A[a]	30.43[b] ± 0.650 Mean S.E.
Appetite	Complete anorexia	Good appetite
Faecal character	Semisolid to pasty	Pelleted faeces

- = No protozoal motility

+++ = Vigorous protozoal motility

A = Absent.

Complete recovery was obtained in eight (80%) out of 10 affected goats with mean pretreatment value of pH of rumen liquor, rumen motility rate, protozoal motility, cellulose digestion time (CDT), sediment activity time (SAT), faecal character and appetite as 4.94 ± 0.08, 0.50 ± 0.224/5 minutes,

Table - 22. Analysis of variance of pre and post treatment values of different parameters in moderate acidosis of goats

Source	d.f.	M.S.			
		pH of R.L.	Rumen motility rate	C D T	S A T
Between treatment	1	11.35**	187.77**	8403.5**	3240.64**
Error	16	0.0525	0.406	3.43	1.48

(** P/0.01)

absent (-), absent (A), absent (A), complete anorexia and semisolid to pasty faeces respectively which changed significantly ($P/0.01$) to 6.53 ± 0.068, 7.00 ± 0.189/5 minutes, vigorous (+++), 49.00 ± 0.757 hours, 30.43 ± 0.65 minutes, good appetite and pellated faeces after treatment respectively (Fig. 56, 57, 58 & 59).

The above findings showed that there was much improvement of pH of rumen liquor, rumen motility, protozoal motility, CDT, SAT, faecal character and appetite but not returned fully towards normal level and it might be due to severe dehydration, toxaemia and lactiacedemia.

Each drug was found to exert beneficial effect in normalisation of rumen function. The pretreatment level of pH of rumen liquor was much below the level of normal value.

For better improvement of pH antacid like Gelusil MPS was given orally. It contains dried aluminium hydroxide and magnesium hydroxide as well as antifrothing agent methyl polysiloxane. The use of antacid in rumen acidosis by oral or intraruminal route has also been recommended by Gnanaprakasam (1970), Sen (1982), Tanwar and Mathur (1983 a) and Cao *et al.* (1987) in goats. However, 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). Oral

antacid may be beneficial if administered within four to six hours after overeating and before lactic acid production has commenced.

To check the growth of Gram positive bacteria, to stop further production of lactic acid in rumen and for sterilisation of rumen contents, Steclin bolus which contain tetracycline hydrochloride was administered orally. Sen (1982) and Tanwar and Mathur (1983 a) in goats adopted the use of antibiotic treatment intraruminally in rumen acidosis.

To check the thiamine deficiency vitamin B_1 injection like 'Berin' was given. Death due to polioencephalomalacia as result of deficiency of thiamine may occur. Gnanaprakasam (1970), Sen (1982) and Tanwar and Mathur (1983 a) obtained satisfactory result with thiamine in acidotic goats.

The improvement of rumen motility rate, protozoal activity, CDT and SAT might be due to improvement of rumen pH and microbial population character.

The improvement of appetite, faecal character might be due to improvement of SAT, CDT and correction of hyper osmolality of rumen and intestinal content.

4.3.3 Treatment of severe acidotic group containing 12 goats (experimental and natural cases) as were brought under the treatment of injection of Sodium bicarbonate (7.5%) and injection of Rintose intravenously along with injection of Avil intramuscularly, Steclin bolus, Avil tablet, Gelusil MPS

and Floratone bolus intraruminally in experimental cases and orally in natural cases and evacuation of rumen contents in experimental cases as test agents are presented in Table - 23. The results of analysis of variance of different parameters between treatments (Pre and post treatments) are also shown in Table - 24.

Table - 23. Trials with injection of Sodium bicarbonate (7.5%) and injection of Rintose along with injection of Avil, Steclin bolus, Avil tablet, Gelusil MPS and Floratone bolus as Test agents against severe acidosis in goats based on ARD Profiles.

No. of goats treated	12		
Duration of treatment (days)	5		
No. of goats recovered	5		
	Pre-treatment value	Post-treatment value	
pH of Rumen liquor	4.04[a] ±0.065	6.52[b] ± 0.583	Mean S.E.
Rumen motility rate/5 minutes	0.083[a] ±0.083	6.6[b] ± 2.46	Mean S.E.
Protozoal motility	-	++	
CDT (hrs.)	A[a]	50.71[b] ± 0.682	Mean S.E.
SAT (mts.)	A[a]	30.00[b] ± 0.49	Mean S.E.

contd..

Appetite	Complete anorexia	Good appetite
Faecal Character	Pasty	Pellated

\- = No protozoal motility
++ = Moderate protozoal motility
A = Absent

The recovery was obtained in five (41.67%) out of 12 goats with mean pretreatment values of pH of rumen liquor, rumel motility rate, protozoal motility, cellulose digestion time (CDT), sediment activity time (SAT), appetite and faecal character as 4.04 ± 0.065, 0.083 ± 0.083/5 minutes, absent (-), absent (A), absent (A), complete anorexia and pasty faeces respectively which changed significantly ($P<0.01$) to 6.52 ± 0.583, 6.6 ± 2.46/5 minutes, moderate (++), 50.71 ± 0.682 hours, 30.00 ± 0.49 minutes, good appetite and pellated faeces respectively after treatment (Fig. 56, 57, 58 & 59).

The present findings showed that percentage of recovery was not satisfactory which was 41.67%. More than 50% cases were refractory to treatment and it might be due to severe toxaemia, dehydration, irreversible hepatic and renal damage.

There was moderate improvement of pH of rumen liquor, rumen motility rate, protozoal motility, CDT, SAT but not upto the level of normal value.

Table - 24. Analysis of variance of pre and post treatment values of different parameters in severe acidosis of goats

Source	d.f.	M.S.			
		pH of R.L	Rumen motility rate	C D T	S A T
Between treatment	1	21.67**	149.88**	9001.79**	3148.25**
Error	15	0.0414	0.141	1.62	0.979

(** P/0.01)

Fig. 56. COMPARATIVE STUDIES ON PRE AND POST TREATMENT MEAN VALUES OF pH OF RUMEN LIQUOR IN MILD, MODERATE AND SEVERE LACTIC ACIDOSIS OF GOATS

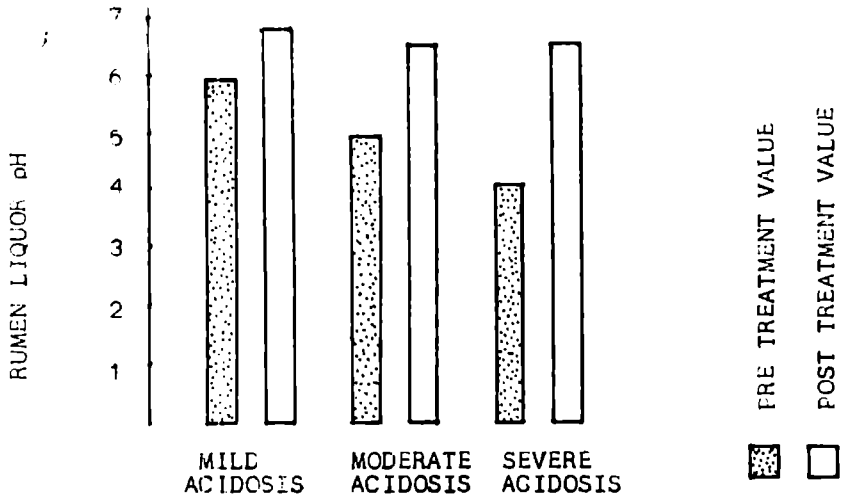


Fig. 57. COMPARATIVE STUDIES ON PRE AND POST TREATMENT MEAN VALUE OF RUMEN MOTILITY RATE IN MILD, MODERATE AND SEVERE LACTIC ACIDOSIS OF GOATS

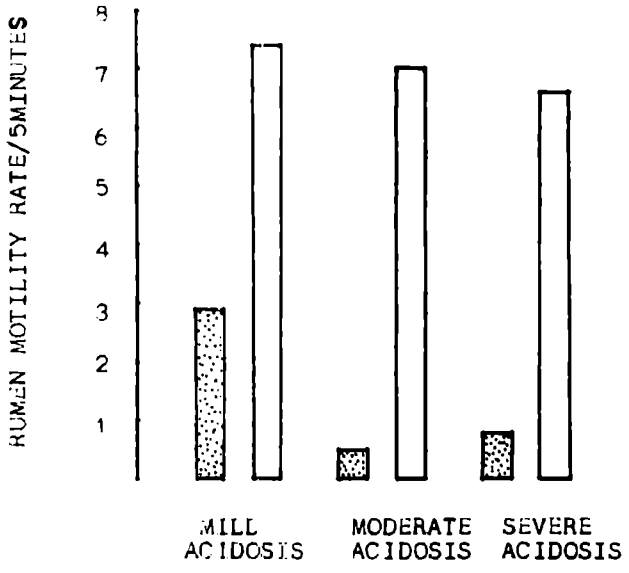


Fig. 58. COMPARATIVE STUDIES ON PRE AND POST TREATMENT MEAN VALUES OF CELLULOSE DIGESTION TIME (CDT) IN MILD, MODERATE AND SEVERE LACTIC ACIDOSIS OF GOATS

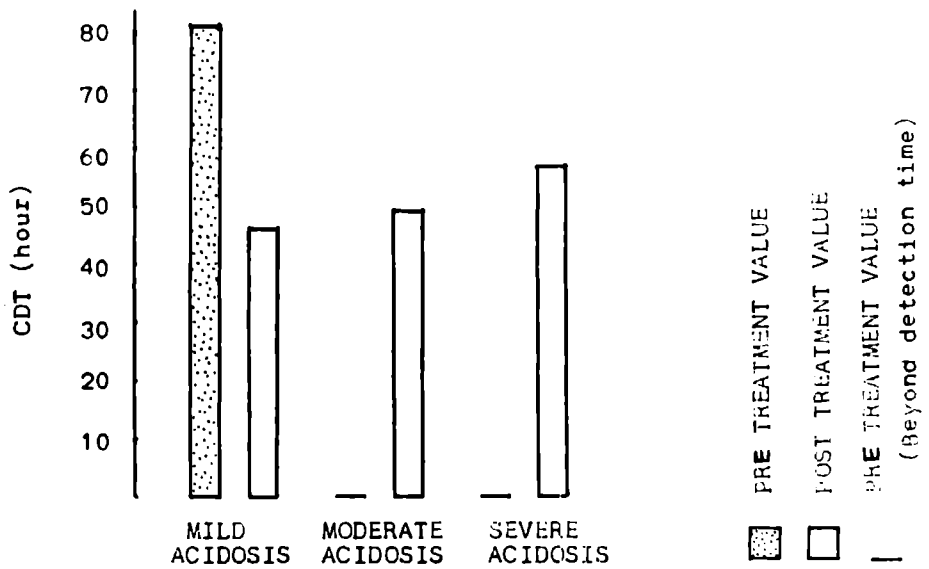
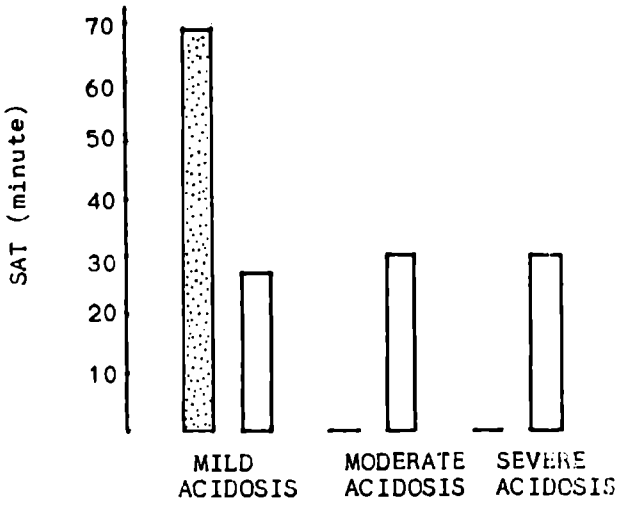


Fig.59. COMPARATIVE STUDIES ON PRE AND POST TREATMENT MEAN VALUES OF SEDIMENT ACTIVITY TIME (SAT) IN MILD, MODERATE AND SEVERE LACTIC ACIDOSIS OF GOATS



Severe lactic acidosis required prompt multi drug approach with optimum dose level to safe guard the life of the animals.

The evacuation of rumen content and rumen lavage were conducted in experimental group to curtail the amount of lactic acid and other noxious material in the rumen. This type of treatment has also been suggested by Gnanaprakasam (1970) and Sen (1982). To increase the alkali reserve of blood, sodium bicarbonate solution (7.5%) was administered intravenously. This type of treatment was also performed by Prasad and Rakib (1975) and Howard (1986) during rumen acidosis with satisfactory response.

To counteract the effect of histamine and other amines produced during rumen acidosis, antihistamine like injection Avil and Avil tablet intramuscularly and intraruminally or orally were used. Administration of antihistamine was also recommended by Gnanaprakasam (1970) and Sen (1982) in goats.

To check the growth of Gram positive bacteria, to stop further production of lactic acid in rumen and for sterilization of rumen content, tetracycline hydrochloride was administered intraruminally or orally. Similar line of treatment was adopted by Sen (1982) and Tanwar and Mathur (1983 a) in goats.

To check the electrolyte imbalance (Na, K and Ca) and also acid base balance to some extent Rintose solution

infusion was given intravenously. Similar line of treatment was adopted by Tanwar and Mathur (1983 a) in goats and Sethuraman (1976) in buffaloes and cows.

For neutralising the rumen hyperacidity, antacid like Gelusil MPS was administered intraruminally in experimental animals and orally in natural cases.

The each component of the Floratone bolus was found to exert beneficial effect to restore normal rumen function. Methionine corrected the hepatic insufficiency and restored normal appetite, Brewer's yeast corrected dyspepsia due to ruminal acidosis (Dirksen, 1970). Copper sulphate and cobalt sulphate stimulated the growth of microbial population (Joshi 1976). Thiamine deficiency due to activation of enzyme thiaminase in lactic acidosis was corrected by the thiamine present in the Floratone bolus. Niotinamide corrected the deficiency of micotinamide along with thiamine in acid indigestion (Thomas, 1988).

The improvement of rumen motility rate, protozoal motility coincided with the improvement of rumen pH. Reduction in time of cellulose digestion activity and sedimentation activity of rumen liquor might be due to reappearance of cellulolytic bacteria and destruction of amylolytic bacteria. Improvement of appetite might be due to better CDT and SAT of rumen liquor.

5.0 SUMMARY AND CONCLUSION

Studies on the pathobiochemical changes in experimental rumen acidosis in goats with special reference to endocrinopathy and therapy were carried out in the Department of Veterinary Medicine and Public Health, Faculty of Veterinary and Animal Sciences, Bidhan Chandra Krishi Viswavidyalaya, Mohanpur, District Nadia, West Bengal from May 1990 to June 1991.

Experimental rumen acidosis was produced in goats to study the following :

i) Clinical accounts
ii) Macroscopic, microscopic and biochemical changes of rumen liquor
iii) Haematological changes
iv) Biochemical changes of blood/serum
v) Changes in hormonal level
vi) Macroscopic and microscopic changes of different tissues
vii) Application of suitable therapy in mild, moderate and severe acidosis in goats on the basis of ARD profiles.

18 healthy Black Bengal goats of either sexes with an average body weight between 12 to 18 kg were taken up for experimental studies. They were fitted with fibre glass made rumen fistula and were divided into three groups viz. 'A', 'B' and 'C' having 6 animals in each group. Group 'A' was selected

as control healthy. Group 'B' and 'C' were selected as experimental without therapy and with therapy respectively. Group C was again subdivided into C_1 and C_2 groups each comprising of 3 goats. C_1 group was the mild acidotic and C_2 group was the severe acidotic. Natural clinical cases were designated as group 'N' which was again subdivided into three groups viz. 'N_1', 'N_2' and 'N_3' each comprising of 10 - 12 numbers of animals. The mild acidotic group was 'N_1', moderate was 'N_2' and severe was 'N_3'. Animals of 'N_1' category was also subjected to different therapy. Experimentally severe rumen acidosis was produced in group 'B' and 'C_2' with crushed rice through rumen fistula at the dose rate of 70 g/kg body weight and mild acidosis was produced at the dose rate with 40 g/kg body weight in 'C_1' group.

The control group of animals showed good appetite, normal posture and gait with glossy body coat. They were active and alert and their muzzles looked moist. Conjunctival mucous membranes were pale pink to rosy in colour. All animals showed normal rumination. Abdomens were normal in size and shape. The mean heart rate, rectal temperature, respiration rate, rumen motility rate were 70.66 $\pm$ 0.98/minute, 102.94 $\pm$ 0.085^{o}F, 18.16 $\pm$ 0.575/minute and 7.16 $\pm$ 0.20/5 minutes respectively. The animals urinated 5 - 9 times a day and defaecated normal faeces like pellated faeces. The smell, consistency and colour of rumen liquor were aromatic, semiliquid or viscous

and green to greenish brown respectively. The mean CDT, SAT and MBRT were 44.11 ± 2.52 hours, 26.54 ± 1.02 minutes and 10.29 ± 0.309 minutes respectively. The concentration, iodophilic nature and motility of rumen protozoa were +++ (high), ++/+++ (moderate to high) and +++ (vigorous) respectively. The normal mean protozoal count and bacterial count of rumen liquor were 39.36 ± 9.07 x 10^4/ml and 15.07 ± 1.34 x 10^9/ml respectively. The type of bacteria in normal healthy rumen liquor was always Gram negative Cocci and rods. The average values of pH, lactic acid, TVFA, NH_3-N were 6.98 ± 0.03, 7.09 ± 0.30 mg/100 ml, 66.33 ± 1.98 mEq/L and 14.80 ± 1.51 mg/100 ml respectively in normal healthy goats. In normal haematological studies, the PCV, haemoglobin, TEC, TLC, total neutrophils, segmented neutrophils, neutrophilic band cells, metamyelocytes, lymphocytes, monocytes, eosinophils and basophils were 23.08 ± 0.288%, 8.80 ± 0.176 g/100 ml, 8.59 ± 0.29 x 10^6/cmm., 5.95 ± 0.364 x 10^3/cmm., 41.84 ± 1.62%, 41.84 ± 1.62%, nil, nil, 53.00 ± 1.50%, 2.00 ± 0.258%, 3.16 ± 0.40% and 0.13 ± 0.08% respectively. The normal mean blood pH, blood glucose, blood lactic acid, serum calcium, serum phosphorus, serum total protein, serum albumin, serum globulin, A/G ratio, blood urea, serum creatinine, serum sodium and serum pottasium were 7.44 ± 0.013, 53.5 ± 0.85 mg/100 ml, 12.31 ± 0.684 mg/100 ml, 11.70 ± 0.256 mg/100 ml, 5.31 ± 0.468 mg/100 ml, 6.45 ± 0.10 g/100 ml, 3.97 ± 0.055 g/100 ml, 2.48 ± 0.066 g/100 ml, 1.615 ± 0.040, 20.85 ± 0.88 mg/100 ml, 1.03 ± 0.041 mg/100 ml, 140.75 ± 2.70 mEq/L and 3.94 ± 0.12 mEq/L respectively. The mean value of serum cortisol, insulin

and thyroxin concentration in normal healthy goats were Below Detection Level ($\angle 1$ µg/100 ml), 10.70 $\pm$ 1.20 µIU/ml and 44.11 $\pm$ 2.68 ng/ml respectively.

The clinical manifestations of experimental goats without therapy (Group B) were severe dullness, head pressing, dyspnoea, loss of appetite, grinding of teeth, mucoid to purulent nasal discharge, wobbling gait, excessive thirst, shunken eyes, injected mucous membrane, reduced skin elasticity, occassional diarrhoea and constipation and lateral recumbency before death. Five goats died between 30th to 108th hour of induction of rumen acidosis.

In clinical observations it was found that rumen motility rate and rumination were absent from 12th hour onwards. Distended heavy abdomen and occassional gurgling sound on auscultation over rumen were also observed. Most of the acidotic goats defaecated pellated to pasty faeces with occasional diarrhoea. Severe dehydration (10%) was observed between 24th hour to 48th hour of induction of acidosis. Frequency of urination gradually declined from 12th hour onwards and no urination between 60th hour to 84th hour was observed. There was decrease in pH of urine and faeces from 12th hour onwards and 24th hour onwards respectively at a significant level.

The mean heart rate rose significantly from 12th hour onwards and highest value was observed at 48th hour.

The rectal temperature increased significantly at 12th and 60th hour and in other hours it decreased. Every animal showed subnormal temperature before death. The respiration rate was increased significantly from 24th hour onwards upto 84th hour, then declined at 96th hour but not below the value of '0' hour.

On macroscopical examination of rumen liquor, the smell, consistency, colour, CDT, SAT and MBRT were found faintly sour to intensed sour, semiliquid to watery, greyish white, absent, absent and absent respectively. On microscopical examination of rumen liquor, the motility, concentration, iodophilic nature of rumen protozoa were totally absent from 12th hour to 84th hour. The average total protozoal count of rumen liquor declined significantly by 12th hour and no protozoa was found from 24th hour to 84th hour. From 96th hours onward there was reappearance of protozoa in a very low concentration. The total bacterial count of rumen liquor significantly decreased at first and rose afterward but always less than '0' hour count. There was predominence of Gram positive bacteria (coccoid and rod) from 24th hour onwards in rumen liquor.

The average pH of rumen liquor declined significantly and lowest value of 4.37 $\pm$ 0.096 was observed at 48th hour. The mean concentration of lactic acid in rumen liquor increased significantly from 12th hour onwards and reached to highest mean concentration of 326.08 $\pm$ 10.95 mg/100 ml

at 24th hour of induction of acidosis. The mean concentration of TVFA of rumen liquor decreased significantly and lowest mean concentration of 6.00 ± 0.707 mEq/L was observed at 36th hour. The mean concentrations of ammonia nitrogen (NH_3-N) were significantly lower than that of '0' hour at 12th to 48th hour. Then it gradually increased significantly from 60th hour onwards.

On haematological studies the average PCV was increased from 12th hour and highest PCV was 41.4 ± 0.601 percent at 48th hour. The average concentration of haemoglobin increased from 12th hour and gradually declined from 60th hour nearer to the '0' hour value. The average total erythrocytic count nonsignificantly increased to a maximum value of 10.2 ± 0.32 x 10^6/cmm. at 48th hour and thereafter gradually declined towards the base value. The mean total leukocytic count increased significantly throughout the experimental period and reached to highest mean concentration at 60th hour. In differential leukocytic count, the average percentage of total and segmented neutrophils were significantly higher than that of '0' hour value. There was also significant increase in percentage of band cells from '0' hour to 72th hour, afterwards percentage declined. There was appearance of metamyelocytes at a significant percentage at 24th hour onwards. It gradually disappeared from 96th hour onwards. The mean percentage of lymphocytic count decreased significantly throughout the experimental period and reached to a minimum percentage

of 14.8 ± 1.32 at 48th hour of induction of acidosis. The average percentage of monocytes and eosinophils showed a significant fall from 24th hour and 12th hour onwards respectively. But complete absence of cells was observed at 36th, 42th and 60th hour following induction of lactic acidosis.

In biochemical studies on serum/blood the average blood pH decreased throughout the experimental period. The lowest mean pH 7.15 ± 0.017 was observed at 42th hour of induction of acidosis. The mean concentration of blood glucose was significantly higher than that of '0' hour value throughout the experimental period and peak level was observed at 42th hour of lactic acidosis. The average concentration of blood lactic acid increased significantly to reach a maximum value of 48.66 ± 1.93 mg/100 ml at 42th hour. There was gradual decrease of mean serum calcium concentration at significant level from 12th hour onwards and the lowest concentration of 9.58 ± 0.147 mg/100 ml was observed at 36th hour of this study. Afterwards, it increased gradually to reach the value nearer to the value of '0' hour upto the end of experimental period. The total observations on serum inorganic phosphorus level showed significantly higher value than that of '0' hour value. The average concentration of serum total protein increased significantly and highest mean concentration of 7.28 ± 0.92 g/100 ml was observed at 48th hour. Afterwards, it gradually declined below the level of '0' hour value. The average concentration of serum albumin was decreased significantly as compared to '0' hour value. The average concentration of

serum globulin gradually increased significantly and highest mean concentration was observed at 42th hour. The average value of albumin : globulin ratio declined significantly from '0' hour value. The mean concentration of blood urea gradually rose significantly from 12th hour onwards and did not show any tendency to fall down. However, serum creatinine level was within the normal range upto 42th hour of induction of ruminal acidosis. It gradually increased significantly at 48th hour onwards. The average concentration of serum sodium gradually increased from 24th hour onwards and significantly highest value was observed 148.00 $\pm$ 2.20 mEq/L at 36th hour. The average concentration of serum potassium was increased throughout the experimental period.

On analysis of serum hormonal levels it showed that the average concentration serum cortisol increased abruptly by 12th hour and onwards. Peak level of 36.14 $\pm$ 0.47 μg/100 ml was observed at 42th hour. The average concentration of serum insulin gradually increased and reached to a maximum level as 187.17 $\pm$ 4.33 μIU/ml by 24th hour. Afterwards the values sharply declined and reached below detection level ($\underline{/1}$ μIU/ml) towards the end of experimental period. The mean concentration of thyroxin in serum gradually declined at 12 hours onwards and the concentration were below detection level ($\underline{/1}$ ng/ml) at 42th, 48th, 60th, 72th and 84th hour. Afterwards it gradually increased from 96th hour onwards.

The post mortem examination of goats that died during the experimental period revealed desquamation of

rumen mucosa, patchy congestion and haemorrhages in the rumen wall, thining of the abomasal wall, marked venous congestion in the mucosa of small and large intestines, marked haemorrhages and congestion in the uterine wall, kidney, spleen, lungs, pericardium and myocardium of heart, Mesenteric lymph nodes were swollen. The liver was enlarged in size, sometimes congested and pale with distended gall bladder. Necrotic patches were noticed on external surfaces of the liver.

Histopathological lesions revealed marked atrophy of exocrine alveoli, reduction in number and atrophy of islets of pancreas and hypertrophied zona fasciculata rich in lipid content of adrenal gland, atrophic changes of the follicles of thyroid glands and shunken glomeruli and degenerative changes of lining epithelium of tubules with full hyaline cast of the kidney. Section of lungs showed oedema and infiltration of inflammatory cells in the interstitial spaces. Section of intestine showed extensive congestion with surface ulceration of the mucosa and infiltration of mononuclear cells. Superficial congestion, perivascular accumulation of inflammatory cells, chromatolysis, gliosis were observed in brain. Sections of spleen tissues showed extensive vascular congestion and patchy areas of haemorrhage, sometimes loss of folicular architechture. Lymphoid follicles were very prominent, enlarged in size with prominent germ centre indicating hyperactivity of lymphnodes.

Myocardium showed moderate degree of fatty changes.

Periportal fibrosis, intrahepatic cholestasis, segmental coagulative necrosis, microabscesses and focal granulometous lesions were observed in liver. PAS stained sections of liver revealed depletion of glycogen in the hepatocytes. In rumen, reticulum and omasum the lesions varied from formation of microvesicles in the stratum lucidum with infiltration of neutrophils and mononuclear cells to desquamation of the mucous membrane. Section of abomasum showed degeneration or atrophy of glandular structure with infiltration of mononuclear cells in the lamina propria. Denudation of mucous layer was also evident.

Different therapeutic trials were taken up for different degree of acidosis (e.g. mild, moderate and severe) on the basis of ARD profiles. For mild degree of acidosis trial with Floratone bolus gave 100% recovery. In mild acidosis mean pretreatment values of pH of rumen liquor, rumen motility rate/5 minutes, protozoal motility, CDT, SAT, appetite and faecal character were 5.97 $\pm$ 0.087, 2.92 $\pm$ 0.335, moderate to slow (+/-), 81.00 $\pm$ 2.63 hours, 69.00 $\pm$ 3.20 minutes, inappetence and semisolid respectively. After treatment then changed to normal values of 6.79 $\pm$ 0.049, 7.42 $\pm$ 0.193, vigorous (+++), 46.28 $\pm$ 2.64 hours, 27.43 $\pm$ 0.85 minutes, good appetite and pellated faeces respectively. For moderate degree of acidosis trial with local alkaliser (Gelusil MPS), local antibiotic (Steclin bolus) and systemic injection of Berin gave 80% recovery. 20% animals did not

respond due to severe dehydration and lactiacedemia. In moderate acidosis mean pretreatment values of pH of rumen liquor, rumen motility rate/ 5 minutes, protozoal motility, CDT, SAT, appetite and faecal character were 4.94 ± 0.08, 0.05 ± 0.224, absent (-), absent (A), absent (A), complete anorexia and semisolid to pasty respectively. After treatment these changed nearer to normal values of 6.53 ± 0.068, 7.00 ± 0.189, vigorous (+++), 49.00 ± 0.757 hours, 30.43 ± 0.650 minutes, good appetite and pellated faeces respectively. But for severe degree of acidosis, trial with systemic and local alkaliser, systemic and local antihistaminic drug, systemic electrolytes therapy etc. did not give satisfactory result because only 41.6% animals recovered. More than 50% cases were refractory to treatment and it might be due to irreversible damage of liver, kidney, brain and also due to toxaemia. In severe acidosis, mean pretreatment values of pH of rumen liquor, rumen motility rate, protozoal motility, CDT and SAT were 4.04 ± 0.065, 0.083 ± 0.083/5 minutes, absent (-), absent (A) and absent (A) respectively. After treatment these changed to 6.52 ± 0.583, 6.6 ± 2.46/5 minutes, moderate (++), 50.71 ± 0.682 hours and 30.00 ± 0.49 minutes which were slightly away from the normal values.

The observations of investigation can be summarised and conclusion can be drawn as follows.

1. Experimental rumen acidosis can be produced in goats by intraruminal administrations of crushed rice at the

dose rate of 70 g/kg body weight.

2. In case of untreated acidotic goats clinical symptoms and observations are characterised by dullness, depression, anorexia, increased heart rate, respiration rate, distended abdomen, decreased or absent rumen motility and death.

3. There are characteristic alteration of macroscopical, microscopical and biochemical observations of rumen liquor like CDT, SAT, MBRT, protozoal activity and count, bacterial type and count, concentration of TVFA, NH_3-N, Lactic acid and pH.

4. Marked alterations of haematological picture like PCV, Hb, R.B.C. count, W.B.C. count and differential count are observed.

5. Marked alteration of blood biochemical observations like pH, calcium, phosphorus, glucose, urea, creatinine, protein, sodium and potassium are observed.

6. Marked alterations of serum hormonal level like insulin, cortisol and thyroxin are observed.

7. There are marked macroscopical and microscopical changes of different tissues.

8. Treatment of severe acidotic cases are not always encouraging while therapy of mild and moderate cases are encouraging.

CHAPTER - VI

FUTURE SCOPE OF RESEARCH

6.0 FUTURE SCOPE OF RESEARCH

1. Recent study has pointed out that butyric acid is the active component in producing acid indigestion. Studies may be made on the measurement of level of butyric acid in rumen liquor and serum in future.

2. Decrease in thiamine level occurs in acid indigestion cases. Studies on thiamine level in rumen liquor and blood may be made in future.

3. Efforts may be made to isolate different endotoxins produced in acid indigestion, which are deletorious to health of the animal.

4. Attempt may be made to assess the level of thyroid stimulating hormone (TSH) and levels of epinephrine and norepinephrine in serum to know their impact in relation to lactic acidosis.

5. Effort may be made to develop acid adaptative microbes for effective treatment agent in lactic acidosis.

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