STUDIES ON ACUTE RUMINAL ACIDOSIS IN GOATS WITH SPECIAL REFERENCE TO THERAPEUTIC AND SURGICAL MANAGEMENT

BHAGAVANTAPPA B.

DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
VETERINARY COLLEGE, BIDAR
KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY BIDAR-585401

JUNE, 2017

STUDIES ON ACUTE RUMINAL ACIDOSIS IN GOATS WITH SPECIAL REFERENCE TO THERAPEUTIC AND SURGICAL MANAGEMENT

Thesis submitted to the

Karnataka Veterinary, Animal and Fisheries Sciences University, Bidar

In partial fulfilment of the requirements for the award of the

Degree of

DOCTOR OF PHILOSOPHY

in

VETERINARY SURGERY AND RADIOLOGY

By

BHAGAVANTAPPA B.

DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
VETERINARY COLLEGE, BIDAR
KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY BIDAR-585401

JUNE, 2017

KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY (KVAFSU), BIDAR
DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
VETERINARY COLLEGE, BIDAR

CERTIFICATE

This is to certify that the thesis entitled "**STUDIES ON ACUTE RUMINAL ACIDOSIS IN GOATS WITH SPECIAL REFERENCE TO THERAPEUTIC AND SURGICAL MANAGEMENT**" submitted by **Mr. BHAGAVANTAPPA B.** I. D. No. **DVNK-1102** in partial fulfillment of the requirements for the award of **DOCTOR OF PHILOSOPHY** in **VETERINARY SURGERY AND RADIOLOGY** of the Karnataka Veterinary, Animal and Fisheries Sciences University, Bidar is a record of bonafide research work carried out by him during the period of his study in this University under my guidance and supervision, and the thesis has not previously formed the basis for the award of any degree, diploma, associate ship, fellowship or other similar titles.

Bidar
June, 2017

(SHIVSHANKAR M. USTURGE)
Major Advisor, Professor (V S R)
and Director of Research, KVAFSU, Bidar

Approved by:

Chairperson : _____
(SHIVSHANKAR M. USTURGE)

Nominated External Examiner : _____
(R.V. SURESH KUMAR)

Members :

1. _____
(B. V. SHIVAPRAKASH)

2. _____
(D. DILIP KUMAR)

3. _____
(VIVEK R. KASARALIKAR)

4. _____
(ASHOK PAWAR)

5. _____
(VINAY P. TIKARE)

AFFECTIONATELY DEDICATED

TO

PARENTS

ACKNOWLEDGEMENT

I take this opportunity to acknowledge the chairman of advisory committee, **Dr. Shivshankar M. Usturge**, Director of Research, KVAFSU, Bidar for allotting me this topic of research, who extended keen interest, untiring guidance in surgical techniques and writing skills, critical appreciation, advice, support and encouragement throughout the tenure of my study.

I am thankful to **Dr. B.V. Shivaprakash,** Dean, Veterinary College, Bidar, Professor and Head, Department of Veterinary Surgery and Radiology, Veterinary College, Bidar and advisory committee member for the advice, strict guidance in surgical techniques, and critical appreciation during the period of this research with deep sense of gratitude.

I am thankful to **Dr. Mohamed Nadeem Fairoze,** Director of instruction (PGS) for providing all the requirements and guidance during this research work.

I am immensely thankful to **Dr. D. Dilipkumar,** Professor, Department of Veterinary Surgery and Radiology, Veterinary College, Bidar and as a member of advisory committee for helping me in many ways to conduct my research, his valuable suggestion and supervision rendered throughout the period of research.

I am thankful to **Dr. Vivek R. Kasaralikar**, Professor, Department of Veterinary Medicine, Veterinary College, Bidar and advisory committee member for critical appreciation, advice, guidance, support and encouragement and suggestions during this course of the study.

I am thankful to **Dr. Ashok Pawar,** Professor and Head, Department of Veterinary Anatomy and Histology, Veterinary College, Bidar and advisory committee member, for valuable help, advice, support and suggestions during this course of study.

I am thankful to **Dr. Vinay P. Tikare,** Assistant Professor, Department of Veterinary Pharmacology and Toxicology, Veterinary College, Bidar and advisory committee member, for valuable help and suggestions during this course of study.

I am thankful to **Dr. Balappanavar** and **Dr. Jahangir**, Assistant Professors, Department of Veterinary Surgery and Radiology, Veterinary College, Bidar for their timely help, support and assistance throughout the study.

I am also thankful to **Dr. U. S. Jadhav**, Assistant Librarian, Veterinary College, Bidar, for their kind help in collecting references of the study. I am highly thankful to library staff for their co-operation throughout the course of study.

I am thankful to **Dr. Patil,** Professor and Head, **Dr. Thirumalesh**, Professor, **Dr, Ramachandra,** Professor and Head, **Dr. Basavaraj Awati**, Professor and Head, **Dr. Shrikant Kulkarni**, associate professor, **Dr. Bijurkar**, associate professor, **Dr. Ravindra, Dr. Sandeep, Dr. Vijyakumar, Dr. Sunilchandra, Dr. Kiran, Dr. Yathish, Dr. Girish, Dr. Pradeep, Dr. Shrinivasreddy, Dr. Venkangouda, Dr. Sathish**, **Dr. Mallinath, Dr. Manjunath, Dr, Mahesh, Dr. Arun**, and **Dr**. **Harish**, assistant professor of Veterinary College, Bidar and **Dr. Panchasheel, Dr. Yallappa Ingale, Dr. Kumarswamy, Dr. Vijay, Dr. Sarvagnya, Dr. Somshekar, Dr. Vishwanath** and **Dr. Basavaraj** Veterinary Officers for their whole hearted encouragement, advices and ever wiling support during research work.

I am thankful to my departmental postgraduates **Dr. Venkatgiri, Dr. Kareppa, Dr. Vineet, Dr. Nikith, Dr. Sagar, Dr. Beerappa Dr. Sachin and Dr. Chamanvali** for wonderful companion, help extended to me on the way to completion, and who have shared the moments of laughter and sorrow can never be forgotten.

I respectfully concede to my departmental staff, **Mr. Shekhsavali,** Sr. Lab Tech, **Mr. Sangareddy**, Pharmacist, **Mr. Manikappa, Mr. Shantappa, Mr. Hussian** and **Smt. Lakshmi** attender, for always providing friendly atmosphere and made my working time pleasant.

Indeed the words are at my command are not adequate either in form of spirit to convey the depth of gratitude, regards and respect to my grandfather Shri. **Shivaray,** grandmother Late Smt. **Sharanamma,** my parents, Shri. **Basavannappa**, Smt. **Kalavati**, my uncles and aunts Shri. **Gurushantappa**, Smt. **Mahadevi**, Shri. **Bhimashankar**, Smt. **Nagamma**, Shri. **Shrimanth**, and Smt. **Mahadevi** for their blessings, affection, encouragement and unceasing moral support to accomplish this study. I am extremely thankful to my beloved wife **Anita**, smiling daughter **Nishkala** and cheerful son **Satvik**, for their moral support and sacrifices made during entire doctoral research. My sincere thanks to my brothers **Mallikrajun, Sharanbasappa, Prabhuling, Hanumanth, Siddharama, Reavnsiddha, Somlinga, Madival, Mahanthesh,** my sister **Mukta,** and sister in laws**, Rekha, Jyoti, Jayashree, Shobha, Surekha and Lalita,** and their daughters and sons for providing moral support.

I acknowledge the sacrifice and moral support shared by friends and relatives during my study.

Bidar
June, 2017 **[Bhagavantappa B.]**

CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF PLATES

LIST OF ABBREVIATIONS

%	-	Per cent
@	-	At the rate of
>	-	Greater than
±	-	Plus or minus
≤	-	Lesser than or equal to
^{0}F	-	Degrees Fahrenheit
µl	-	microlitre
ALT	-	Alanine amino transaminase
AST	-	Aspartate transaminase
dL	-	Decilitre
DLC	-	Differential leucocyte count
et al.	-	Co workers
g	-	Gram
Gram +ve	-	Gram positive bacteria
Gram –ve	-	Gram negative bacteria
H^+	-	Hydrogen ion concentration
Hb	-	Haemoglobin
HCO^-_3	-	Bicarbonate
IU	-	International unit
kg	-	Kilogram
L	-	Litre
Ltd.	-	Limited
MBRT	-	Methylene blue reduction time

mg	-	milligram
ml	-	millilitre
mmHg	-	Partial pressure of mercury
mmol	-	Millimoles
n	-	Number of animals
nmol	-	Nanomoles
No.	-	Number
P	-	Level of significance
PCV	-	Packed cell volume
Pvt.	-	Private
RL pH	-	Rumen liquor pH
SAT	-	Sedimentation activity test
SE	-	Standard Error
TEC	-	Total erythrocyte count
TLC	-	Total leucocyte count
TPC	-	Total protozoal count
viz.	-	Namely
$VpCO_2$	-	Venous partial pressure of carbon dioxide
VpH	-	Venous blood pH
VpO_2	-	Venous partial pressure of oxygen

Introduction

I. INTRODUCTION

A large number of farmers in India depend on animal husbandry for their livelihood and it plays an important role in the rural economy. As per 19th Livestock census, 2012 (GOI, 2014) India's livestock sector is one of the largest in the world with a holding of 11.6% of world livestock population. Livestock sector contributed about 27.25% out of the total agricultural GDP during 2012-13 (Islam *et al*., 2016). Worldwide interest in goats has continued to increase drastically from last decade. It has been recognized that the value of using goats as a tool in rural development programmes to improve the social and economic conditions of subsistence farmers and the rural poor. Goat production in India is the primary occupation of poor and landless laboures. The expansion interest in goats husbandry practice has increased the demand for goat related veterinary services in the areas of clinical medicine, research and extension. Goat is a browsing animal; readily feed on shrubs, bushes and trees. However, in the past twenty to thirty years, the improper management in the feed offered to ruminants, urbanization, lesser availability of the grazing land and accidental ingestion of ration rich in carbohydrate resulted in the providing a high energy diet. In ruminants, acidosis is defined as the biochemical and physiological stresses caused by rapid production and absorption of ruminal organic acids (volatile fatty acids and lactic acid) that arise from the over consumption of readily fermentable carbohydrates (Britton and Stock, 1986). Prasad *et al*. (1976) reported in India lactic acidosis contributes 18% cases of ruminal indigestion under field condition. Mahmood *et al*. (2013) conducted a survey and recorded tentatively a total of 265 adult goats of various breeds had lactic acidosis. However, on ruminal fluid analysis they found that 24 (1.2 %) animals were positive for

lactic acidosis with 50% of case fatality rates. Ruminal acidosis is the greatest nutritional problem for farmers which attributes to substantial economic losses arising directly from the treatment of acidotic animals, mortality and /or morbidity and indirectly from the reduction of milk, meat and reproduction.

In India, the feeding of cereal grains is the most common readily fermentable carbohydrates source contributing to acidosis. Cereal grains vary in their starch fermentability and potential to cause acidosis (Dunlop, 1972 and Huntington, 1988). For example, cooked rice, chapati, wheat flour, vegetable waste, ceremonial leftover food are highly fermentable and therefore diets based on these ingredients are conducive to acidosis, while sorghum grain based diets are less fermentable and the least likely to cause acidosis (Radostits *et al*., 1994). Ruminal acidosis is not one disease, rather a continuum, with degree of severity often separated into acute and the more economically important, chronic or subacute ruminal acidosis (Slyter, 1976 and Britton and Stock, 1986).

Acute ruminal acidosis symptoms are depression or listless appearance, diarrhoea, absence of ruminal movements, gurgling sound of rumen on auscultation, grinding of teeth, regurgitation of rumen contents, unable to move, tachycardia, increase in pulse, respiratory rate, distension of abdomen, founder, laminitis, cessation or abrupt reduction of feed intake and potentially polioencephalomalacia, which occurs from thiamine deficiency (Tremere *et al*., 1968; Koers *et al*., 1976 and Owens *et al*., 1998) and degree of signs intensified with acuteness of the disorder in induced acidotic goats. Other responses to rapid ingestion of high grain diets include a dramatic reduction of ruminal

pH (5.2 or less) (Cooper and Klopfenstein, 1996), increased concentration of VFA and lactic acid in the rumen (Huntington, 1988) and a significant decline in total protozoa (Hristov *et al*., 2001). In severely acute cases, death may occur within 24 to 72 hours following grain engorgement (Glock and DeGroot, 1998). When the grain levels ingested exceed protozoal capacity to remove starch, protozoa populations will decline (Hristov *et al*., 2001), a drastic increase in bacterial counts will occur, fermentation to VFA and lactic acid will proceed and ruminal pH will decline (Mackie *et al*., 1978). As the ruminal environment becomes increasingly acidic and approaches pH 5.0, growth of *S. bovis* gradually declines (Slyter, 1976; Russell and Hino, 1985). A niche is created for other lactate producing bacterial populations, such as *Lactobacillus spp*., ultimately reducing ruminal pH below 5.0 and leading to systemic and metabolic acidosis (Braun *et al*., 1992; Radostits *et al*., 1994 and Owens *et al*., 1998). The systemic impact of acidosis can have several physiological implications including decreased ruminal motility and complete ruminal stasis occurring at pH of 5.0 or less (Huber, 1976 and Radostits *et al*., 1994), hyperkeratosis (Krehbiel *et al*., 1995), liver abscesses (Nagaraja and Chengappa, 1998), rumenitis and laminitis (Nocek, 1997).

Treatment is aimed at correcting the cardiovascular shock, dehydration, acidosis, toxemia and removing or neutralizing the offending feed stuffs. Intravenous fluid containing sodium bicarbonate should be administered. Oral administration of magnesium hydroxide and magnesium oxide may neutralize the acidic pH and is sufficient in mild cases. However, if much of the feed is still in the rumen, these two alkalizing agents will only work momentarily (Navarre *et al*., 2002). Oral antibiotics have been recommended to kill rumen microflora and stop fermentation. Removing the

substrate for growth of lactobacillus organisms is more effective line of treatment. Orogastric tubes with large enough bores for reflux of feed stuffs are too large for goats; rumenotomy is indicated in severe cases to remove the feed (O' Connor, 1985; Frank, 2002; Fubini and Duchrame, 2004; Tyagi and Singh, 2004 and Das *et al*., 2011). After the rumen pH is corrected transfaunation of the rumen microflora is beneficial (Nour, 2006). The systemic antimicrobial of choice is penicillin as anaerobes are the most likely offending organisms. Several feed additives are commonly used in conjunction with good management practices and include antibiotics (chlortetracycline, tylosin) that have either systemic effects or ruminal effects (ionophores such as monensin and lascalocid.) and dietary buffers (e.g. bicarbonates, hydroxides and silicates) designed to neutralize acidic conditions in the rumen (Huntington, 1988).

Research Objectives

From a welfare point of view and inconsistent recovery by therapeutic management of acute ruminal acidosis, surgical approach has been reported beneficial in severe cases of ruminal acidosis in order to save the life of the animal. However, the available literature on surgical approach for ruminal acidosis in goats appears to be limited. Keeping these aspects in view, the research was under taken with following objectives.

Thus, the objectives of the research were:

1. To study the prevalence of ruminal acidosis in goats of last five years from January 2011 to December 2016 at five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar.

2. To study the changes in clinical, haematological, biochemical and rumen liquor parameters of the affected animals before and after different treatment.

3. To compare the therapeutic and surgical management of acute ruminal acidosis in goats.

Review of Literature

II. REVIEW OF LITERATURE

Ruminal acidosis is recognized as a significant disorder in ruminants. When ruminant ingests large quantity of readily fermentable carbohydrates results in the over production of volatile fatty acids thereby decreases rumen fluid pH to 5.5 and causes the condition called acute ruminal acidosis. The conservative medicinal approach had been practiced to treat acute forms of ruminal acidosis in goats with inconsistent results. Whereas goats with rumen fluid pH less than 5.5, distension of abdomen, recumbent position are preferred to be treated by the surgical approach i.e. rumenotomy to save the life of animal (Radistits *et al*., 2007). However, the available literature on surgical treatment for acute ruminal acidosis in goats is few. Therefore the present literature is reviewed and discussed under the following headings.

2.1. Incidence of ruminal acidosis

2.2. Etiology of ruminal acidosis

2.3. Clinical signs of ruminal acidosis

2.4. Diagnostic methods of ruminal acidosis

2.5. Ruminal fluid changes in ruminal acidosis

2.6. Haematological changes in ruminal acidosis

2.7. Biochemical changes in ruminal acidosis

2.8. Blood gas changes in ruminal acidosis

2.9. Treatment methods for ruminal acidosis

2.1 Incidence of ruminal acidosis

Prasad *et al*. (1976) reported that lactic acidosis contributes 18% cases of ruminal indigestion under field condition in India.

Nour *et al*. (1998) and Kleen *et al*. (2003) stated that incidence of ruminal acidosis was 0.6 % in Nubian goats and 40 % in bovine herds respectively.

Ashok Kumar *et al*. (2005) observed 20 per cent incidence of ruminal acidosis in a retrospective study for 14 years conducted on organized goat farm at Makhdoom (UP).

Darwin *et al*. (2007a) found that 278 out of 2123 (13.09%) had incidence of ruminal acidosis in goats presented to the clinics for treatment. Among 278 goats, females (86.69 %) had higher incidence than the males (13.30 %). The winter season had higher incidence of acidosis (32.73%) compared to north east monsoon (27.34 %), south west monsoon (21.94 %) and summer (17.99 %) in goats. The incidence of acidosis was higher in goats of 1-2 years of age (52.16 %) followed by one year age group (18.71 %), 2-3 years age group (16.90 %) and above three years age group (12.23 %).

Gonzalez *et al*. (2010) reported 18 % incidence of ruminal acidosis in goat herds.

Kasaralikar *et al*. (2012) recorded the incidence of caprine ruminal acidosis was 9.67 per cent. The incidence was highest in goats aged between 2-3 years (48.77%) followed by 1-2 years (33.81%) and least in more than 3 years (11.48%). The highest occurrence of ruminal acidosis was recorded in the month of October (12.50%), followed by November (10.66%) and least in March (6.15%).

Panchasheel (2013) reported the incidence of ruminal acidosis in 157 goats (11.12 %) among the total 1413 cases reported for digestive disorders during the years 2010, 2011 and 2012. Highest incidence was in the goats aged between 1 to 3 years (87.80%) followed by more than 3 years of age (8.28%) and least in the goats less than 1 year of age. The higher incidences of ruminal acidosis in female (85.98%) as compared to males goats (14.01%).

Mahmood *et al*. (2013) conducted a survey and recorded tentatively a total of 265 adult goats of various breeds having lactic acidosis. On confirmation based on clinical signs, history and ruminal fluid analysis 24 (1.2 %) animals were found positive for lactic acidosis with 50% of fatality rates.

Tufani *et al*. (2013) treated 42 small ruminants (26 sheep and 16 goats) with ruminal acidosis and they have been graded as mild (18), moderate (16) and severe (8) based on rumen fluid pH as 6.32, 5.71 and 4.54 respectively.

Singh *et al*. (2016) reported 18 goats of 2-4 years age of either sex developed ruminal acidosis after accidental consumption of wheat and rice.

2.2 Etiology of ruminal acidosis

Experimentally rumen acidosis had been produced by feeding wheat (Allison *et al*., 1972), barley (Chaplin and Jones, 1973), glucose (Hungate *et al*., 1952), sucrose (Krogh, 1959) in sheep and cattle.

Tanwar and Mathur (1983a) produced ruminal acidosis in goats by administration of whole wheat grain through the rumen fistula at the rate of 80g, 100g and 120g/kg body

weight in group I, II and III respectively. The acidosis was more severe in group III followed by group II and least in group I.

Rowe *et al*. (1989) reported that lactic acid accumulation occured in the rumen due to proliferation of lactate producers (Gram - positive bacteria) and failure of lactate utilizers (Gram - negative bacteria) in rumen with reduced pH and accumulated volatile fatty acids.

Nocek (1997); Krause and Oetzel (2006) stated that ruminants when ingest larger quantity of grain, starch or other feeds which (rich in readily fermentable carbohydrates) produced more lactic acid and reduce pH of rumen which is referred as fermentative acidosis or ruminal acidosis.

Singh *et al*. (2001) mentioned that excessive ingestion of whole wheat grains or wheat flour or stale bread or rice or molasses result into acidic indigestion in goats.

Darwin *et al*. (2007a) recorded that over eating of kitchen waste, market waste, eating vegetables like cabbage, cauliflower leaves and grains as common contributory factors. They opined that severe acidosis was due to ingestion of large quantity of rice gruel, overnight soaked grain and left over food at functions.

Padmaja and Praveena (2011) reported that history of accidental ingestion of left over ceremonial waste by a flock of 18 goats as a cause of rumen acidosis.

Choudhary *et al*. (2011) observed acute ruminal acidosis in 5 goats with a history of accidental ingestion of rice.

Tufani *et al*. (2013) noted that 19, 12, 6 and 5 animals had a history of accidental ingestion of apple, cooked rice, turnip and chapatti respectively that resulted into rumen acidosis in small ruminants.

Wankhede *et al*. (2016) observed that sukadi, a mixture containing various pulses eaten by a 14 year old female Osmanabadi goat which showed anorexia, diarrhoea and acid indigestion.

2.3 Clinical signs of ruminal acidosis

Tanwar and Mathur (1983a) reported that depression, diarrhoea, absence of ruminal movements, gurgling sounds of rumen on auscultation, grinding of teeth, regurgitation of rumen contents, unable to move, tachycardia, increase in pulse and respiration rate and degree of signs intensified with acuteness of the disorder in induced acidotic goats.

Braun *et al*. (1992) stated that clinical signs of acidosis vary depends on the severity of the disease. It could be acute, life threatened situation, or chronic with reduced feed intake and weight gain.

Rowe (1997) mentioned that a new condition, acid gut syndrome, characterized by accumulation of acids in the digestive tract at concentration that had not previously been considered harmful to the animals.

Jani *et al*. (2001) reported that pasty, grey coloured, scanty and soft feaces and nystagmus, grinding of teeth, regurgitation of food, in coordination, increased pulse and respiration rate in severe acidotic goats.

Abhishek Kumar and Verma (2005) reported that the rumen motility was decreased to 1.40 ± 0.24 / 5 min at 48 hr and reached towards normal (6.50 ± 0.22 / 5min) at 120 hr in acidotic goats. The ruminal stasis might had occurred due to increased molar concentration of butyrate in the rumen (Radostitis *et al*., 2000).

Hajikolaei *et al*. (2006) observed that the mean heart rate increased significantly at 9, 12, 15, 18, 21, 24, 30, 36 and 48 hr (66.60 ± 9.80 to 100.20 ± 9.96 and 98.00 ± 6.05), rectal temperature decreased significantly at 24, 30 and 36 hr (22.60 ± 3.45 to 19.00 ± 2.30 and 19.00 ± 1.89) and respiration rate remained without changes significantly throughout the experimentation of ruminal lactic acidosis in sheep.

Krause and Oetzel (2006) stated that repeated exposure of rumen wall to sub-acute form of acidosis damages the rumen. Once the rumen wall gets damaged, bacteria and toxins produced by bacteria can enter the portal circulation, causing liver abscesses and an inflammatory response (Gozo *et al*., 2005, 2006 and 2007).

Nour (2006) reported that the mean values of heart rate (58 ± 2.60 to 132 ± 8.90) and respiration rate (15 ± 0.80 to 27 ± 3.10) increase progressively by four hour and highest observations made at 21 hour of post induced lactic acidosis in Nubian goats.

Darwin *et al*. (2007b) found that mild distention of abdomen, reduced rumen motility, semisolid faeces in mild acidosis whereas distended abdomen, fluid splashing sound on percussion, wobbling gait and pasty diarrhoea in moderate form acidosis. They also observed signs of regurgitation of rumen contents, anuria, lateral recumbancy, sub - normal temperature and watery diarrhoea in severe acidotic goats.

Lindinger and Heignhauser (2008) opined that the severity of the acidosis related to many factors and not the only lactic acid production. The importance also should be given to the other contributory acids which disrupts acid base status in acidosis condition (Harmon *et al*., 1983).

Parrah *et al.* (2010) observed acute tympany, restlessness, anorexia, diarrhoea, excessive salivation, tachycardia, oligourea and ruiminal stasis in 18 cattle suffering with acute ruminal tympanitis and acidosis. The similar findings had been recorded in a fallow deer and ruimanl acidotic goats by Sahinduran (2003) and Jani *et al*. (2001) respectively.

Sharma *et al*. (2010) reported increased respiration, pulse rate, bloat, reduced rumen motility, pasty fecal material around anal region, mucopurulant nasal discharge, dehydration and subnormal temperature in acidotic goats.

Radostits *et al*. (2000) mentioned that general weakness is one of the principle sign in ruminal acidosis.

Eldin *et al*. (2014) recorded decreased feed intake, depression, weakness, semisolid feaces, increased pulse and respiration rates, decreased ruminal movement and distension of abdomen in an experimentally induced lactic acidosis in sheep.

Meena *et al*. (2016) observed projectile regurgitation, distended abdomen and laboured breathing in a goat due to ingestion of excess concentrate feed.

Singh *et al*. (2016) recorded atony of rumen, tympany, subnormal body temperature, increased respiration rate, pulse rate and heart rate in acidotic goats.

Somashekar Reddy *et al*. (2016) reported that signs of depression, laboured breathing and sudden collapse in sheep flock affected with grain overload.

Tawheed *et al*. (2017) observed ruminal distension, respiratory distress, tachycardia, sunken eyes, dehydration and dullness in goats affected with acute ruminal acidosis.

2.4 Diagnostic methods of ruminal acidosis

Sharma *et al*. (2010) diagnosed lactic acidosis in goats based on decreased ruminal pH ranged from 4 to 6.

Arora *et al*. (2011) diagnosed grain over load in buffaloe based on reduced rumen pH (5), blood pH (6) and complete absence of the live microflora in ruminal fluid.

Choudhary *et al*. (2011) diagnosed rumen acidosis on the basis of reduced rumen pH (3 to 5), milky grey colour, watery consistency with foul odour and absence of microflora in ruminal fluid of goats and sheeps.

Padmaja and Praveena (2011) diagnosed rumen acidosis based on dark brown colour, acidic in odour, pH (4.5 to 5.0) and absence of rumen microflora in ruminal fluid of 18 goats.

Singh *et al*. (2016) diagnosed rumen acidosis based on dark brown colour, presence of more gram positive organism, pH 4-5 and absence of protozoal activity in acidotic goats.

Tawheed *et al*. (2017) recorded that decreased pH (4.0-5.0), diminished prtotozoal count and motility in ruminal fluid of goats affected with acute ruminal acidosis by ingestion of jawar, rice and chapati.

2.5 Ruminal fluid changes in ruminal acidosis

2.5.1 Colour, Consistency and Odour

Sen *et al*. (1982) observed change in colour and consistency of ruminal fluid from dark green and viscous before induction to light brown and liquid by 12 hr after experimental induction of acidosis in goats. Presence of faint sour odour, absence of sedimentation activity and complete absence of protozoa at 12 hr post induction of acidosis was recorded.

Lal *et al*. (1989) reported ruminal fluid became greyish, watery and sour from 24 hr after induction of acidosis in goats.

Braun *et al*. *(*1992) observed milky coloured and profoundly acidic odour of ruminal fluid in clinical cases of acute ruminal acidosis in sheep and goats.

Basak *et al*. (1993) reported that the smell of ruminal fluid changed from aromatic to faintly sour within 12 – 24 hr after induction of acidosis, which became intense sour from 36 hr onwards. The colour of rumen liquor changed from dark green to creamy brown, by 24 hr and to greyish white by 36 hr and the consistency changed to watery after 24 hr of induction of acidosis.

Desai *et al*. (1999) observed normal physical changes like normal greenish brown colour, viscous consistency and aromatic odour of ruminal fluid which were turned to milky gray, watery and sour odour respectively after 24 hr post induction to acidosis in calves.

Abhishek Kumar and Verma (2003) reported that the ruminal fluid had greyish colour, sour odour, watery viscosity, absence of protozoa, and drastic fall in pH within 24 hr post induction of acidosis. They also found increased values of total volatile fatty acids in ruminal fluid at 12 hr to 24 hr post induction of acidosis.

Abhishek Kumar and Verma (2005) induced experimentally ruminal acidosis in six goats aged 1-2 year and weighing 10-15 kg by providing whole wheat grains @ 100g/kg body weight and they were observed at 12, 24, 48, 72, 96 and120 hr post induction of acidosis. The rumen fluid colour changed from greenish to grayish between 12-96 hr. The odour and sourness increased between 48-72 hr with increase in severity of acidosis. The consistency was changed from viscous to watery as severity of acidosis was advanced. The protozoal motility was absent between 12-24 hr post induction and then reappeared at 96 hr.

Darwin *et al*. (2007b) reported physical characteristic changes in ruminal fluid of healthy and different stages of rumen acidosis in goats. Ruminal fluid was milky grey with watery consistency in both moderate and severe cases of acidosis. The odour of ruminal fluid was sour in mild and acidic in moderate and severe acidotic goats.

Kasaralikar *et al*. (2007) mentioned that the ruminal fluid changed to milky white colour, sour odour and watery consistency in acute ruminal acidosis in goats.

Pradeep Kumar Ram *et al*. (2007) found that consistency of ruminal fluid as viscous to semisolid in healthy and semi liquid to watery in mild, moderate and severe acidotic goats.

Chaudhary *et al*. (2011) observed that milky grey coloured ruminal fluid with watery consistency and foul odour in acidotic goats.

Padmaja and Praveena (2011) observed dark brown coloured ruminal fluid with acidic odour in rumen acidosis of goats.

Rahima *et al*. (2012) found milky green coloured ruminal fluid with watery consistency and acidic odour in acute ruminal acidosis in goats.

Tufani *et al*. (2013) observed milky coloured ruminal fluid with sour odour in acidotic sheep and goats.

2.5.2 Ruminal fluid pH and Protozoal activity

Dirksen (1965) reported decreased pH of 4-6 in ruminal acidosis of cattle.

Dash *et al*. (1972) reported absence of protozoa in ruminal acidosis contents at pH below 5.5 in Indian cattle.

Randhawa *et al*. (1981) reported decreased pH of ruminal fluid which was associated with significant increase in the level of TVFA’s along with lactic acid in per acute ruminal acidosis in calves.

Sen *et al*. (1982) observed drop in pH of ruminal fluid to 4.65 ± 0.02 with complete absence of protozoa at 12 hr after induction of acidosis in goats and their reappearance at 72 hr after therapy.

Tanwar and Mathur (1983b) observed significant fall in the pH of ruminal fluid to 4.29 ± 1.12 by 72 hr post induction of acidosis in goats.

Pradhan *et al*. (1988) and Braun *et al*. (1992) recorded normal ruminal fluid pH 7.11± 0.07 in apparently healthy goats.

Narendra *et al*. (1990) found reduced protozoal activity with increased bacterial count in experimental ruminal acidosis in calves.

Basak *et al*. (1993) observed pH of ruminal fluid was gradually decreased from 12^{th} hr (6.57) to the lowest value at 48^{th} hr (4.37) and absence of protozoal motility, concentration and iodophilic nature from 12^{th} to 84^{th} hr post induction of acidosis in goats.

Lal *et al*. (1993) reported significant fall in the pH of the ruminal fluid was from 7.08 ± 0.60 to 4.54 ± 0.23 by 24 hr post induction of lactic acidosis in goats.

Patra *et al*. (1993) noticed decreased ruminal fluid pH from 6.70 ± 0.11 to 4.70 ± 1.20 by 24 hr post induction of acidosis with wheat grain in sheep and which might be attributed to rapid utilization of carbohydrate rich diet by amylolytic bacteria (*Strep. bovis*) led to production of large quantity of lactic acid in rumen. Such variation might be

seen due to quick fermentation of wheat grains by amylolytic bacteria in the rumen leading to production of large amount of lactic acid (Dunlop, 1971).

Desai *et al*. (1999) recorded normal ruminal fluid pH (6.90 ± 0.30) which decreased significantly to 6.56 ± 0.11 at 12 hr and change of vigorous movement of protozoa to complete absence of protozoal concentration till 72 hr post induction to acidosis in calves.

Shukla *et al*. (1999) observed absence of rumen protozoa, rumen movements and reduction of ruminal fluid pH at 36 hr post induced acidosis in calves.

Jani *et al*. (2001) found decreased ruminal fluid pH and protozoal counts in the acidotic goats at Anand Veterinary College (Gujarat).

Metkari *et al.* (2001) observed decreased ruminal fluid pH 5.40 ± 0.20 at 12 hr, absolute absence of protozoal motility at 24 hr post induction with forced feeding of wheat grains @100g/kg body weight and restored to normalcy by 96 hr after treatment in experimental acidosis of goats.

Nikolov (2003) observed declination of pH of ruminal fluid after 12 hr of post treatment with molasses and the normal values were recorded on 130^{th} hr after treatment.

Abhishek Kumar and Verma (2005) reported ruminal fluid pH declined to 4.52 ± 0.15 at 24 hr and reached to 6.45 ± 0.02 at 120 hr in experimentally induced ruminal acidosis in goats.

Hajikolaei *et al*. (2006) mentioned the mean ruminal fluid pH decreased significantly at 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 hr (6.91 ± 0.11 to 4.04 ± 0.14 and 4.19 ± 0.14) of post induction rumen lactic acidosis respectively in sheep.

Darwin *et al*. (2007b) reported based on ruminal fluid pH graded as mild (6 to 6.80), moderate (5.50 to 5.80) and severe (4 to 5.20) in goats presented to clinics with ruminal acidosis. They observed changes in colour of ruminal fluid from green to milky grey, consistency from thick and viscous to watery and aromatic odour to sour odour in acidotic goats.

Darwin *et al*. (2007b) observed abundant protozoan density in healthy goats and it will be reduced in mild (less than 10 protozoa per field) and completely absent in moderate and severe acidotic goats.

Pradeep Kumar Ram *et al*. *(*2007) reported decreased ruminal fluid pH (5.19 ± 1.10 in mild, 4.99 ± 0.10 in moderate, 4.16 ± 0.09 in severe and absence of motility and activity of ruminal micro flora in acidotic goats as compared to normal once.

Kumar (2010) found highest population of total protozoa (3.69X 10^5 /mL) in healthy goats than the cattle, buffaloe and sheep.

Parrah *et al.* (2010) found that ruminal fluid pH ranged from 3.90 to 4.90 and protozoal count was nil in all the cases of cattle with acidosis.

Chaudhary *et al*. (2011) observed decreased ruminal fluid pH (3-5 range) and nil concentration of protozoa in ruminal acidosis of goats.

Padmaja and Praveena (2011) recorded decreased pH (4.50 to 5.00) and absence of protozoal activity of ruminal fluid in acidotic goats.

Rahima *et al*. (2012) recorded low pH (4) and nil protozoal concentration in ruminal fluid of acute ruminal acidotic goats.

Mahmood *et al*. (2013) found no protozoa in ruminal fluid with pH (4.00) whereas drastic reduced number of protozoa and motility with pH (5.00) in goats.

Tufani *et al*. (2013) observed significant decrease in mean pH of ruminal fluid (4.54 ± 0.159) and entire rumen protozoa ceased in severe acidosis of small ruminants.

Ullah *et al*. (2013) stated that the ruminal fluid pH of lactic acidotic goats was significantly lower (4 to 5) than the normal range (6 to 7) and absence of rumen protozoa in acidotic goats.

Meena *et al*. (2016) noticed brown colour ruminal fluid with pH 5.00 and regurgitation in an acidotic goat.

2.5.3 Methylene Blue Reduction Time (MBRT)

Basak *et al*. (1993) found significant increased MBRT at 12 hr post induction of acidosis in goats.

Desai *et al*. (1999) recorded significant increase in methylene blue reduction time (61.00 ± 1.47 min) at 96 hr post induction to acidosis in calves.

Darwin *et al*. (2007b) observed normal mean MBRT value 6.65 ± 0.23 in apparently healthy goats where as significant increase in values noticed 11.05 ± 0.63 in mild to 32.55 ± 1.00 severe acidotic goats.

Kasaralikar *et al*. (2007) reported drastic fall in pH (4.72 ± 0.07) within 12-24 hr post induction of acidosis. Similar findings recorded by Garry (1990) and Basak *et al*. (1993). Significant decreased MBRT (6.08 ± 0.24 to 52.50 ± 3.35 min) and absence of sedimentation activity time (18.00 ± 1.00 to nil) observed at 12-24 hr post induction of acidosis in goats.

2.5.4 Sedimentation Activity Time (SAT)

Sen *et al*. (1982) reported complete absence of sedimentation activity at 4 hr post induction of acidosis in goats and normal SAT was observed at 72 hr after therapy.

Basak *et al*. (1993) recorded significantly increase sedimentation activity time (65.33 ± 4.25 minutes) at 12^{th} hr and which was absent from 24^{th} hr post induction to ruminal acidosis in goats.

Pal *et al*. (1994) noticed increased SAT and decreased GFT and TVFA values because of suppressed microbial fermentation in rumen. Restoration of rumen protozoal motility and count, SAT, GFT and TVFA to normal by 72 hr had beneficial effects with Ruchmax on rumen protozoa and carbohydrate fermentation and similar findings was also reported by the Tripathy and Mishra (1972) by administering Rumbion in experimental acid indigestion in goats.

Desai *et al.* (1999) recorded significant increase in sedimentation activity time (50.00 ± 2.41 min) at 72 hr post induction to acidosis in calves.

Jani *et al*. (2001) reported increased sedimentation activity time of ruminal fluid of acidotic goats (38.25 ± 0.25 min) compared to healthy goats (12.80 ± 0.15 min).

Pradeep Kumar Ram *et al*. (2007) found increased sedimentation activity time significantly in mild acidotic goats (67.08 ± 0.09 min) compared to normal once (24.83 ± 1.55 min) and was absent in moderate and severe cases.

Eldin *et al*. (2014) mentioned colour, odour and consistency of ruminal fluid which showed significant changes. Sedimentation activity time showed highly significant increase and significant decrease in rumen pH and absence of protozoa in an experimentally induced lactic acidosis in sheep.

2.5.5 Rumen Bacteria

Basak *et al*. (1993) stated that gram positive bacteria were predominant from 24th hr post induction to rumen acidosis in goats.

Desai *et al*. (1999) observed presence of gram positive and gram negative bacteria changed to presence of only gram positive bacteria till 120 hr post induction to acidosis.

Darwin *et al*. (2007b) recorded complete absence of ruminal microflora and predominance of gram positive bacteria in acidotic goats when compared with apparently healthy goats.

Arora *et al*. (2011) noticed markedly increase in number of gram positive bacteria (*Streptococcus bovis*) which resulted in the production of large quantities of lactic acid in the rumen of acidotic buffaloes.

Padmaja and Praveena (2011) observed more number of gram positive bacteria (*cocci*) in rumen liquor of acidotic goats.

Tufani *et al*. (2013) observed significant increase in gram positive bacteria (*Streptococcus spp*.) in ruminal acidosis of small ruminants.

2.5.6 Total Volatile Fatty Acids (TVFA)

Randhawa *et al*. (1981) reported decrease in pH of ruminal fluid and significant increase in levels of TVFA along with lactic acid in per acute ruminal acidosis of calves.

Pal *et al*. (1994) reported increased SAT and decreased GFT and TVFA were the results of suppressed microbial fermentation in rumen.

Nocek (1997) found that ingestion of feeds rich in ruminal fermentable carbohydrates resulted in production of large quantity of TVFA, lactic acids, decreased pH, weakened the buffering capacity of rumen that resulted in decreased efficiency of rumen microflora and fermentation.

Singh *et al*. (2001) observed significantly decrease in the total volatile fatty acids (46.42 ± 2.16 mEq/L of rumen liquor) in goats with simple indigestion, compared to healthy goats (69.50 ± 3.15 mEq/L of rumen liquor). The similar observations also made by the Lal *et al*. (1989) and Singh *et al*. (1996).

Abhishek Kumar and Verma (2003) reported increased values of TVFA in ruminal fluid at 12 hr to 24 hr post induction of acidosis.

Abhishek Kumar and Verma (2005) reported increased TVFA concentration at12 hr post induction (110.58 ± 1.14 mEq/L) and tended to decrease by 55.80 ± 0.36 mEq/L at 120 hr and remained higher than normal. The abrupt rise in TVFA at12 hr post induction might be attributable to rapid fermentation of starch by amylolytic bacteria in the rumen and subsequent decreased value might be due to increased absorption at low pH (Gray, 1948).

2.6 Heamatological changes in ruminal acidosis

Wildenthal *et al*. (1968) reported that in ruminal acidosis it was very important to evaluate severity of disorder by evaluating haematological changes since severe dehydration and involvement of cardiovascular system.

Dunlop (1972) and Basak *et al*. (1993) opined that significant increased total leukocyte count could be due to endotoxins of ruminal origin.

Randhawa *et al*. (1981) attributed that increased Hb and PCV levels in lactic acidosis to withdrawal fluid from vascular system in to the rumen in peracute ruminal acidosis in calves.

Tanwar *et al*. (1983) found steady rise in haematocrit after induction of acidosis. In the untreated control group PCV which reached to 37.80 ± 2.86 per cent by 72 hr. Mean hematological values of hemoglobin and packed cell volume in untreated control

goats increased till 72 hr post induction, reaching maximum of 12 g (haemoglobin) and 38 per cent (PCV).

Lal *et al*. (1990), Michell (1990) and Das and Mishra (1991) reported raise in haematocrit levels which could be due to haemoconcentration and systemic dehydration caused by drawing of fluid from the circulation to rumen as increased osmolality in ruminal acidosis.

Dunlop (1971) and Basak *et al*. (1993) observed leucocytosis and neutrophilia which was due to endotoxins of rumen origin in ruminal acidosis.

Jani *et al*. (2001) reported increased haemoglobin and packed cell volume values in goats with acidosis.

Sandhu *et al*. (2001) found from day 0 to up to 120^{th} day mean values of 8.55 ± 0.10 (g/dL), 27.32 ± 2.03 (%), 10.85 ± 0.12 ($10^6/mm^3$), 7.04 ± 0.24 ($10^3/mm^3$), 71.16 ± 0.52 (%) and 26.32 ± 0.58 (%) for Hb, PCV, TEC, TLC, lymphocytes and neutrophils respectively in healthy goats.

In cattle significant increased erythrocytic count and Hb was observed by Parrah *et al.* (2010) and it was attributed to haemoconcentration due to dehydration. The similar finding was also made by the Shihabudheen *et al*. (2003).

Shihabudheen *et al*. (2003) observed significant increased erythrocyte count and Hb (16.23 ± 0.65 g/dL) concentration at 24, 48 and 72 hr post induction ruminal acidosis with rice in six goats. It could be attributed to the haemoconcentration as a result of

dehydration in ruminal acidosis and also the release of blood cells from spleen due to stress might contributed to the elevated Hb was said by Dash *et al*. (1972).

Nour (2006) found total white blood cells count, neutrophil, lymphocyte percentages and the PCV returned to normal level after 22 hr and 2 hr respectively by surgical treatment in acidotic goats.

Sharma *et al*. (2010) found significantly increased haematological values viz. Hb, PCV, TLC, TEC, DLC, only PCV, Hb and TLC values were before treatment in acidotic goats.

Arora *et al*. (2011) found significantly increased Hb, PCV and TEC values in ruminal acidosis of buffaloes.

Rahima *et al*. (2012) recorded significant increase in haemoglobin (9.47 ± 0.36 g/dL), packed cell volume (27.30 ± 0.73%), total leucocyte count (11.78 ± $2.50\times10^3/mm^3$) levels in acute ruminal acidotic goats.

Mahmood *et al*. (2013) recorded no substantiate changes in the Hb and TEC levels whereas marked rise in the PCV, TLC and MCV values in acidotic goats.

Tufani *et al*. (2013) recorded increased heart rate, respiratory rate and significant increased blood glucose, Hb PCV and TEC before treatment in small ruminants. They observed significant increase in haemoglobin (g/dL) 14.09 ± 0.16 in mild, 15.06 ± 0.22 in moderate, 15.90 ± 0.20 in severe acidosis, packed cell volume (%) 41.28 ± 0.65 in mild, 44.88 ± 0.60 in moderate, 49.63 ± 0.73 in severe acidosis and total erythrocyte count

(million/mm^3) 9.60 ± 0.127 in mild, 10.79 ± 0.17 in moderate and 12.50 ± 0.37 in severe acidotic small ruminants.

Eldin *et al*. (2014) observed significant increase in Hb, PCV levels, non-significant increase in WBCs, lymphocyte, granulocyte, monocyte counts and RBCs count remain within normal change in an experimentally induced lactic acidotic sheep.

2.7 Biochemical changes in ruminal acidosis

2.7.1 Glucose

Randhawa *et al*. (1981) found significant increase in blood glucose levels in acidosis which could be due to increased glycogenolysis or gluconeogenesis or due to decreased utilization of glucose by peripheral tissue (Dirkson, 1970).

As a result of increasing carbohydrate rich feed, large amounts of volatile fatty acids were produced and they reach liver via portal vein and utilized for glucose synthesis. Therefore the glucose level increased in large amount in lactic acidosis condition (Radostits *et al*., 1995). The hyperglycemic condition in cattle was also reported by Parrah *et al.* (2010).

Metkari *et al.* (2001) observed decreased blood pH from 7.61 ± 0.05 to 7.45 ± 0.05 and increased blood glucose from 60.50 ± 1.64 to 84.79 ± 1.29 after post induction with forced feeding of wheat grains @100 g/kg body weight in goats.

Sandhu *et al*. (2001) found mean value of plasma glucose was 54.29 ± 2.51 (mg/dL) in healthy goats.

Singh *et al*. (2001) found significant increase in mean glucose values (74.97 ± 2.73 mg/dL) in goats with acid indigestion compared to healthy goats (58.85 ± 5.10 mg/dL).

Nikolov (2003) found increase in levels of blood glucose 12 hr after molasses feeding and which returned to normalcy at 130th hr after treatment.

Nour (2006) recorded immediate decrease in plasma glucose levels after treatment and which reached towards normalcy by 8 hr post treatment in acidotic goats.

Kasaralikar *et al*. (2007) found increased value of glucose (58.36 ± 1.05 to 94.83 ± 1.65) at 24 hr post induction in acidotic goats.

Sharma *et al.* (2010) reported the reduced serum glucose levels in acidotic goats which might be attributed to anorexia due to ruminal stasis. However, the values reduced and remained within the normal physiological limits after 72 hr after the treatment.

Arora *et al*. (2011) found increased level of blood glucose in acidotic buffaloes.

Tufani *et al*. (2013) observed significant increase in blood glucose level (mg/dL) in mild (67.89 ± 1.07), moderate (83.25 ± 0.95) and severe (92.88 ± 1.28) acidotic small ruminants.

Eldin *et al*. (2014) observed significant increase in glucose, total protein levels in an experimentally induced lactic acidosis in sheep.

2.7.2 Aspartate Amino Transferase (AST) and Alanine Amino Transferase (ALT)

Randhawa *et al*. (1981) found increased blood levels of AST in acidosis and it could be due to damage to hepatic parenchyma by corrosive action of lactic acid leading to hepatic necrosis. The rise in AST and ALT concentrations also reported by the Cakala *et al*. (1974) and Das and Mishra (1992) in goats affected with acidosis.

Patra *et al*. (1996) observed significant increase in aspartate amino transferase (60.80 ± 7.65 IU/L) at 24 hr post induction to acidosis in sheep.

Sandhu *et al*. (2001) reported mean value of alkaline phospatase activity was 432.79 ± 20.00 (IU/L) in healthy goats.

Singh *et al*. (2001) found significant increase in values of AST and ALT were in goats with simple indigestion.

Sharma *et al*. (2010) observed a significant increase in AST (108.99 ± 5.40), ALT and serum alkaline phospatase levels compared to base values in acidotic goats before treatment. It might be due to hepatocellular damage as a result of toxic product like alcohol, histamine, thiaminase and other endotoxins produced in the rumen epithelium and thus entering the portal circulation or due to dehydration resulting in to liver damage.

Kasaralikar *et al*. (2007) found increased values of AST (28.92 ± 2.16 to 56.25 ± 2.73) at 24 hr post induction of acidosis goats.

Rahima *et al*. (2012) observed elevated levels of AST (80.33 ± 1.42 IU/L) in blood of acute ruminal acidotic goats.

Eldin *et al*. (2014) observed significant increase in ALT activity and lactic acid and non-significant increase in AST in an experimentally induced lactic acidosis sheep.

2.8 Blood Gas changes in ruminal acidosis

2.8.1 Venous Blood pH

Patra *et al*. (1996) observed significant decrease in blood pH values (7.24 ± 0.006) at 24 hr post induction of acidosis in sheep.

Metkari *et al*. (2001) noticed decline in blood pH values from 7.61 ± 0.05 to 7.45 ± 0.049 by the end of 48 hr grain feeding in all acidotic goats.

Nikolov (2003) reported decrease in venous blood pH from 12 hr post treatment with molasses which returned to normal at 130^{th} hr post treatment.

Sobiech *et al*. (2005) reported significantly higher average arterial blood pH (7.37 ± 0.04) than the average pH of venous blood (7.33 ± 0.03) in healthy sheep.

Leal *et al.* (2010) recorded average venous blood pH (7.35 ± 0.01) in a 14 apparently healthy goats.

Rahima *et al*. (2012) found significant decrease in blood pH values (7.17 ± 0.03) in acute ruminal acidotic goats.

Ullah *et al*. (2013) recorded mean blood pH values as 7.18 ± 0.04, 7.10 ± 0.05, in acidotic goats before treatment and which returned to normalcy (7.35 ± 0.03, 7.20 ± 0.06 respectively) post treatment.

Ghanem *et al*. (2015) reported the average venous blood pH (7.41 ± 0.02) in ten apparently healthy Boer goats.

2.8.2 Venous Bicarbonate (HCO^-_3, mmol/L)

Cao *et al*. (1987) recorded the mean normal values of bicarbonate as (24.2 ± 1.52 mmol/L), pH (7.35 ± 0.30) and CO_2 pressure (36.7 ± 4.81 mm of Hg) of venous blood in goats.

Shukla *et al*. (1999) reported significant fall in blood bicarbonate was noticed at 36 hr post induction of lactic acidosis in all acidotic calves. The values returned to normalcy from 48 hr to 120th hr post treatment.

Sobiech *et al*. (2005) reported the average values of HCO^-_3 and content CO_2 which were lower in arterial blood, compared with venous blood (HCO^-_3, 24.61 ± 3.12); in the case of arterial blood this difference was statistically significant and correlated with pCO_2 in a healthy sheep.

Nour (2006) recorded minimum plasma bicarbonate as 9.2 mmol / L before treatment which increased at 6 hr post-surgical treatment and reached to normalcy by the 6 days of post treatment in acidotic goats.

Leal *et al.* (2010) recorded average bicarbonate concentration as (22.58 ± 0.59) in fourteen apparently healthy goats.

Rahima *et al*. (2012) observed significant decrease in bicarbonate (11.33 ± 0.42) concentration in acute ruminal acidotic goats.

Ghanem *et al*. (2015) reported average bicarbonate concentration was 18.36 ± 1.632 in ten apparently healthy Boer goats.

2.8.3 Venous Partial Pressure of Carbon Dioxide ($VpCO_2$) (mm of Hg)

Sobiech *et al*. (2005) reported lower level of partial pressure of carbon dioxide in arterial blood (4.91 ± 0.46) was than in venous blood (6.19 ± 0.41) in a healthy sheep**.**

Hajikolaei *et al*. (2006) observed decreased blood pCO_2 at 24 hr (40.20 ± 2.17 to 35.04 ± 0.81), blood pH decreased at 18, 21, 24, 30, 36 and 48 hr (7.41 ± 0.03 to 7.15 ± 0.08 and 7.23 ± 0.06) and blood bicarbonate decreased at 15, 18, 21, 24, 30, 36 and 48 hr (25.85 ± 2.08 to 12.1 ± 1.61 and 35.25 ± 1.70) compared to 0 hr in an experimentally induced ruminal lactic acidosis in sheep.

Leal *et al.* (2010) observed average level of pCO_2 in 14 apparently healthy goats was 42.92 ± 1.083.

Rahima *et al*. (2012) found elevated level of pCO_2 (47.50 ± 0.50) in acute ruminal acidotic goats.

Ghanem *et al.* (2015) mentioned that the average level of pCO_2 was reported to be 38.12 ± 0.752 in 10 apparently healthy Boer goats.

2.9 Treatment methods for ruminal acidosis

2.9.1 Conservative Medicinal Approach

Sen *et al*. (1982) and Patra *et al*. (1997) treated acidotic goats with hypertonic sodium bicarbonate intravenously to obtain rapid clinical recovery.

Tanwar and Mathur (1983a) suggested intra ruminal administration of antibiotics, antacids and fresh ruminal fluid transplant for early recovery in ruminal acidosis of goats.

Erdman *et al*. (1988) recommended administration of alkali or buffer as sodium bicarbonate for restoring decreased bicarbonate and increased lactic acid in animals.

Pal *et al*. (1994) reported improvement in ruminal movements, appetite within 72 hr, increased total protozoal count, motility and speedy restoration of rumen dysfunction was observed in calves treated with Ruchamax (herbal formulation).

Shukla *et al*. (1999) conducted an experiment in 16 calves with feeding wheat flour and divided them into four groups with four in each group. They observed that use of herbal agent Ruchamax and Pachoplus along with fresh ruminal fluid had better recovery than the standard sodium bicarbonate treatment combined with either fresh or preserved ruminal fluid.

Metkari *et al.* (2001) concluded that combined treatment containing blood alkalizer, oral antibiotics and vitamin B-complex along with supplementation of fresh rumen cud could be effective in treatment of ruminal lactic acidosis in goats.

Jani *et al*. (2001) treated acidotic goats with sodium pencillin @ 40,000 IU / kg body weight intrarumenally, inj. pheniramine maleate @ 0.2 mg / kg body weight intramuscular and crude liver extract @ 3 ml / animal for three days.

Abhishek Kumar and Verma (2003) treated the goats with sodium biocarbonate (7.5%) solution intravenous @ 4 ml / kg body weight bid for two days, sodium

biocarbonate powder 5 g orally once for 3 days, liver extracts 2 ml intramuscular for 5 days, Avil inj. 1 ml intramuscular for 3 days, Tetracycline HCl powder orally @ 20 mg / kg body weight as single dose on first day, Fresh rumen liquor @ 15 ml / kg body weight orally for 3 days in an experimentally induced acidotic goats with whole wheat grains @100 g / kg body weight.

Abhishek Kumar and Verma (2003) reported that in goats recovery from ruminal acidosis was faster which received the Rumecpowder @ 15 g in water orally for 3 days for restoration of normal ruminal physiological status than the group which has not given.

Nema *et al*. (2003) studied that Appevet, a poly herbal preparation found to be effective to correct the simple indigestion in goats by restoring the altered rumen and biochemical parameters.

Kasaralikar *et al*. (2007) reported higher therapeutic efficacy (94.12%) in clinical cases of ruminal acidosis in goats was observed in treatment regimen which included intravenous sodium bicarbonate with thiamine and oral administration of magnesium hydroxide, chloromphenicol along with cud transplantation.

RAGFAR (2007) stated that the contents of the yeast cell wall aid in the binding of pathogenic bacteria within the rumen, reducing the risk of colonization of detrimental bacteria and, consequently, ruminal upsets and also the actions of metabolites produced by the yeasts in culture to stimulate the rumen microflora and increase the digestive efficiency of the animal.

RAGFAR (2007) mentioned that antibiotics including penicillins, tylosin, potentiated sulphonamides and tetracycline should be given to reduce the risk of liver abscessation. Other supportive treatments included flunixin meglumine (1 mg / kg) for endotoxaemia, antihistamines to control the adverse effects of histamine release and calcium or magnesium solutions either intravenously or subcutaneously to counteract secondary hypocalcaemia and hypomagnesaemia. Thiamine (10 mg / kg) intravenously every 24 to 48 hr for up to three doses may also be helpful to prevent polioencephalomalacia.

Selvaraj *et al*. (2009) suggested oral administration of sodium bicarbonate for 3-5 days resulted in better stabilization of rumen pH in all the animals indicating the dietary buffers in acid indigestion.

Parrah *et al.* (2010) treated cattle with isotonic fluid, electrolyte, sodium bicarbonate 0.8% intravenous, intra ruminal antacids and antibiotics to restore fluid, electrolyte and pH balance and inj. Vitamin B-complex to prevent the damage of liver and central nervous system by hyper acidic condition.

Rohilla *et al*. (2010) conducted an experiment and found that effect of probiotic (*sacchromyces cerevisiae*) alone and with nutri-mix feeding had a significant effect on growth of kids and milk in lactating goats. Live cell present in probiotic caused better ammonia rumen utilization and it improved production of microbial protein and volatile fatty acids, where as decreased lactic acid peaks in the rumen and satbilised the rumen pH (Kander *et al*., 2000 and Kander *et al*., 2005).

Sharma *et al*. (2010) treated goats with sodium bicarbonate (7.5%) solution intravenous @ 4 ml / kg body weight bid for two days, sodium bicarbonate powder 5 g orally once for 3 days, vitamin B-complex inj. 3 ml intramuscular for 5 days, avilvet inj. 2 ml intramuscular for 3 days, tetracycline HCl inj. intra ruminal and intravenously @ 20 mg / kg body weight for three days, and dexamethasone inj. @ 4 mg / kg body weight first day followed by 2 mg and 1 mg / kg body weight respectively for two days along with stomachic powder (Rumec powder) @ 15 g daily for five days in acidotic goats.

Arora *et al*. (2011) treated a buffaloe with hypertonic sodium bicarbonate (7.5 %) @ 5 ml / kg body weight intravenous, inj. Vitamin $B_{1,}$ $B_{6,}$ and B_{12} 30 ml intravenous, antihistamine 15 ml intravenous, four steclin boli (oxytetracycline) intra ruminal, Ecotas bolus @ 2 boli twice daily orally for 3 days and fresh 5 liters of rumen liquor orally from second day onwards once daily for three days for grain over load.

Choudhary *et al*. (2011) treated sheep and goats with inj. Ringers lactate 500 ml intravenous, sodium bicarbonate intra ruminal @ 1 g / kg body weight, inj. chlorpheniramine maleate 2.5 ml intramuscular, inj. Vitamin B-complex 2.5 ml intramuscular, Tetracycline bolus and Provissac bolus orally to restore rumen acidosis.

Choudhary *et al*. (2011) reported complications like polioencephalomalacia from an induced thiamine deficiency, laminitis, ruminitis and liver abscessation (Bolton and Pass, 1988) could be prevented by giving the antibiotics, antihistamine and vitamins.

Padmaja and Praveena (2011) treated goats with rumen acidosis by administration of tribivet 2 ml intramuscular, anistamin 2 ml intramuscular, Bufzone 50 g and Tyrel 20 ml orally which recovered very well.

Rahima *et al*. (2012) concluded that haemodialysis in addition to standard treatment consisting of fluid therapy, administration of sodium bicarbonate, antibiotic, rumenotorics was able to provide 100 per cent recovery in acute ruminal acidosis.

Eldin *et al*. (2014) inferred treatment with sodium bicarbonate and yeast restore ruminal pH at faster rate and stabilized floura and thereby it had given good result and improved general health condition of induced lactic acidosis in sheep.

2.9.2 Surgical Approach: Rumenotomy and Ruminal Lavage

Tyagi and Singh (2004) reported acid indigestion occur due to over feeding of carbohydrates and severely affected cattle or buffaloe with acid indigestion might require emergency rumenotomy to remove the contents of rumen. O' Connor (1985), Frank (2002), Fubini and Duchrame (2004) and Das *et al*. (2011) had the similar opinion of performing rumenotomy in emergency condition where distension of rumen occurred after acid indigestion in ruminants.

Nour (2006) opined in acute cases treated by medicines to correct rumen acidity and fluid loss accompanied the syndrome resulted in signs of alkalosis and considerable erythrocyte destruction respectively. So immediate after appearance of the clinical signs (21 hr) surgical treatment by rumenotomy had been performed to evacuate the ruminal contents, followed by ruminal lavage with normal saline and replacement with fresh ruminal fluid along with presoaked hay to restore ruminal microfloura and fauna in experimentally induced lactic acidotic goats.

RAGFAR (2007) mentioned treatment of mild cases of ruminal acidosis included oral antacids such as magnesium hydroxide, magnesium oxide or sodium bicarbonate at 1

g / kg body weight orally initially to alkalinize the rumen and then oral electrolyte solutions, preferably those containing additional sodium bicarbonate. Severe cases of ruminal acidosis should be treated by giving intravenous hypertonic saline fluids or balanced electrolyte solutions not containing lactic acid. Lavaging the rumen with a wide bore stomach tube in combination with transfaunation from a healthy animal was preferable.

Parrah *et al.* (2010) reported that out of 18 cattle suffering from ruminal acidosis, 10 did not respond to the medicinal treatment. Among 10 cattle 6 were subjected to rumenotomy within 4-6 hr and all of these survived and rest 4 animals died.

Das *et al*. (2011) performed emergency surgical rumenotomy for acute distension of abdomen in a goat which had not responded for routine standard medicinal therapy using stomachics, antibloat powder, treacle suspension, sodium bicarbonate, liver stimulant and Vitamin B-complex.

Tufani *et al*. (2013) treated with oral or intravenous use of isotonic @ 50 ml / kg body weight (1.30 %) and hypertonic @ 10 ml / kg body weight (5 %) to neutralize acidosis. The Rumentas bolus @ 1 boli bid for 3 days, inj. Vitamin B_1, B_6 and B_{12} for three days @ 5 ml intravenous, a course of antihistaminic inj. 3 ml intramuscular and in severe cases along with above medications emergency rumenatomy had been performed to evacuate the rumen contents, lavaging of rumen with 5 % sodium bicarbonate and transplantation of fresh rumen cud into rumen for early recovery.

Tawheed *et al*. (2017) found faster clinical recovery by emptying of rumen using stomach intubation compared to without intubation in six acute acidotic goats.

Materials and Methods

III. MATERIALS AND METHODS

The "studies on acute ruminal acidosis in goats with special reference to therapeutic and surgical management" was carried out at Department of Veterinary Surgery and Radiology, Veterinary College, Bidar.

3.1 INCIDENCE

Incidence of acute ruminal acidosis in goats of different parts of Bidar district was evaluated by analyzing the data of clinical cases presented to Veterinary Clinical Complex, Veterinary College, Bidar, APMC (Peripheral) hospital Bidar and five taluka Veterinary Hospitals of Bidar district *viz*., Aurad, Bidar, Basavakalyan, Bhalki, and Humnabad for a period of five years (2011-2016). The retrospective data was analyzed statistically to draw the inference related to breed, age, sex, season and prevalence of the ruimanl acidosis in goats.

3.2 SELECTION OF ANIMALS

The research was conducted in clinical cases of eighteen goats suffering from acute ruminal acidosis referred for treatment. The animals were randomly divided into three groups of six animals in each group. The therapeutic efficacy of various drugs in comparison with surgical treatment was evaluated for management of acute ruminal acidosis in goats.

3.3 CLINICAL EXAMINATION

The history from the cases was collected with respect to etiology, duration of illness and any previous medication had been given to treat the disorder. The clinical

signs shown by the animal were recorded and systematic clinical examination was carried out to diagnose acute ruminal acidosis.

The vital signs in clinical cases of acute ruminal acidosis were examined from the day of presentation (0 hour, before treatment) and after initiation of treatment at 12 hours, 24 hours, 72 hours and 120 hours. The temperature, heart rate and respiratory rate were recorded in all the goats subjected for therapeutic and surgical treatment.

3.3.1 Respiratory rate (breaths / min)

The respiratory rate was measured by counting the movements of the rib cage and abdomen walls during inspiration and expiration and recorded in terms of breaths / minutes (Chakrabarti, 2006).

3.3.2 Temperature (^{0}F)

The body temperature was recorded with a digital clinical thermometer. The thermometer was gently inserted into rectum in a rotatory manner and kept for two minutes with care that bulb of mercury (thermometric sensor) touches the wall of the rectum. After two minutes the thermometer was taken out of the rectum and reading of the body temperature was recorded (Chakrabarti, 2006). The recordings were done in ^{0}F.

3.3.3 Heart rate (Beats / min)

The heart rate was recorded with the help of stethoscope at 3rd to 5th left intercostals space. The heart rate was measured in beats/minute.

3.3.4 Ruminal contractions

Ruminal contractions were measured by placing a closed fist in left para-lumbar fossa and counting the number of contractions, ruminal contractions were expressed as number of contractions per two minutes. The ruminal motility was observed in goats of all the three groups on 0 hour (before treatment), 72 hours and 120 hours after the initiation of the treatment.

3.4 RUMINAL FLUID ANALYSIS

The ruminal fluid was collected from the clinical cases at the time of presentation for therapeutic and surgical management for evaluation of following parameters. The collection of rumen fluid was done with the help of rumen fluid collection apparatus (Plate 15, 16 and 17). The rumen liquor was collected (0 hour, before treatment) and after initiation of treatment at 12 hours, 24 hours, 72 hours and 120 hours. In total volatile fatty acids estimation the samples were collected in flask kept in ice bath, 2 drops of 20% H_2SO_4 was added to 20 ml of rumial fluid immediately after collection and stored at -20 ^{0}C until further use for the estimation.

3.4.1 Physical Examination

The colour, consistency and odour of ruminal fluid were evaluated as described by Gnanaprakasam (1970).

3.4.2 pH of Ruminal Fluid

The pH of ruminal fluid was estimated immediately after collection with the help of digital pH meter and was expressed in single decimal (*viz*., 7.1, 7.2….) as shown in Plate 18.

3.4.3 Gram Positive to Gram Negative Ratio

Ten drops of strained ruminal fluid was taken and evenly spread on a clean glass slide and heat fixed. The smear was stained with Gram's staining solution. A total 100 bacteria were counted and Gram + ve to Gram - ve ratio was noted and was expressed in per cent value (Rogosa, 1964).

3.4.4 Protozoal Density

Protozoal density was estimated (Shankaranarayana and Nambiar, 1972). A drop of fresh ruminal fluid was placed on a clean slide, covered with a cover slip and observed under microscope with low (10 X) power objective lence. It was concluded that +++ when the more than 30 protozoa were seen per low power field, ++ when the number of protozoa were ranging between 10 to 20, + when the number was between 1 to 10 and nil when no protozoa have been observed.

3.4.5 Methylene Blue Reduction Time (MBRT, minutes)

Methylene blue reduction time of ruminal fluid was estimated by adding 1.0 ml of 0.03 per cent methylene blue dye to 10ml of ruminal fluid. Time taken for the dye to get reduced was taken as methylene blue reduction time and was expressed in minutes (Smith, 1996).

3.4.6 Sedimentation Activity Time (SAT, minutes)

Freshly collected ruminal fluid was filtered through gauze and was observed as it settled in a glass cylinder. The values were expressed in minutes (Dirksen, 1970).

3.4.7 Total Volatile Fatty Acids (TVFA, mmol / dL)

Total volatile fatty acids concentration was analysed using acidified strained ruminal fluid with Markham's apparatus as per the procedure and the value obtained was expressed as mmol per 100 ml (mmol / dL) of ruminal fluid (Barnett and Reid, 1956).

3.5 Haematological Examination

The animal head was stretched and raised from the body. The neck of the animal was prepared aseptically for blood collection. Jugular vein was raised from the furrow by pressing with thumb against the neck and punctured with the 5 ml disposable syringe to get 5ml of blood in all animals. Blood was collected (0 hour, before treatment) and after initiation of treatment at 12 hours, 24 hours, 72 hours and 120 hours. 2.0 ml was transferred to the blood collecting tubes containing 10 per cent of ethylene diamine tetra acetate (EDTA) for heamatological studies. The remaining 3.0 ml was transferred to a clot activator tube for separation of serum and serum was stored at -20 ^{0}C until further use for the estimation of biochemical parameters.

3.5.1 Total Erythrocyte Count (X 10^6 / µL)

Total erythrocyte count was determined by the standard method and expressed in millions permicrolitre (Jain, 2000).

3.5.2 Total Leucocyte Count (X 10^3 / µL)

Total leucocyte count was done by the standard method and expressed in thousands permicrolitre (Jain, 2000).

3.5.3 Haemoglobin (g/dL)

Haemoglobin was estimated by Sahli's haemoglobinometer as per standard method and the values obtained were expressed as g / dL (Schalm *et al*., 1975).

3.5.4 Packed Cell Volume (PCV, %)

The packed cell volume was estimated by microhaematocrit method and values were expressed in per cent (Benjamin, 1985).

3.5.5 Differential Leucocyte Count (DLC, %)

The differential leucocyte count was done by Battlement method in a blood smear stained with Geimsa stain and 100 leucocytes were counted and the individual cells expressed in percentage (Jain, 2000).

3.6 SERUM BIOCHEMISTRY

3.6.1 Serum Glucose (mg/dL)

Serum glucose was estimated by glucose oxidase-peroxidase (OP) method and expressed in mg / dL (Henry, 1963).

3.6.2 Aspartate Amino Transferase (AST, IU/L)

Aspartate amino transferase levels were estimated by modified IFCC method and expressed in IU / L (Reitman and Frankel, 1957).

3.6.3 Alanine Amino Transferase (ALT, IU/L)

Alanine amino transferase levels were estimated by modified IFCC method and expressed in IU / L (Reitman and Frankel, 1957).

3.7 BLOOD GAS ANALYSIS

2 ml of blood was collected at (0 hour, before treatment) and after initiation treatment at 12 hours, 24 hours, 72 hours and 120 hours from the jugular vein in Na EDTA vial and immediately taken to the laboratory for blood gas analysis to estimate VpO_2, $VpCO_2$, VpH, HCO^-_3, H^+ and base deficit with the help of automatic blood gas electrolyte analyser (ESCHWEILER, Combi line, machine serial number, CL 1241, Supplied by CL MICROMED PVT. LTD., a product of Eschweiler GmbH and Co. KG Holzkoppelweg 35 / D-24118 Kiel / Germany) as shown in Plate 19 and 20.

3.8 THERAPY

Eighteen goats with acute ruminal acidosis were selected for the present study and randomly divided into three groups, consisting of six animals in each group.

3.8.1 Therapeutic Treatment

In group I six acute acidotic goats received intravenous alkalizing agent as isotonic sodium bicarbonate as per base deficit and the rumen buffer (Bufzone® from the Intas Pharmaceuticals Ltd., Ahmadabad) 50 g /day per oral for five days (unique rumen buffer enriched with yeast and metabolic boosters) as buffering agent (Plate 1). The fluid therapy was given to all the animals for correction of dehydration and the fluid loss in rumnial acidotic condition.

3.8.2 Surgical Treatment

3.8.2.1 Pre-operative Considerations

The acute ruminal acidotic goats of group II and III were subjected for the clinical examination and decided to treat with surgery i.e. rumenotomy along with medicinal therapy to treat the condition (Plate 2).

In Group II six goats with acute acidosis were subjected to rumenotomy and received the intravenous alkalizing agent as isotonic sodium bicarbonate as per base deficit and probiotics (Ecotas® from the Intas Pharmaceuticals Ltd., Ahmadabad) 1 bolus bid orally for five days were used as symbiotic bolus to restore ruminal microflora (Plate 24).

In Group III six goats with acute acidosis were operated for rumenotomy and received the intravenous alkalizing agent as isotonic sodium bicarbonate as per base deficit and fresh rumen fluid was given @ 10 ml / kg per orally for two days to restore ruminal microflora (Plate 21, 22 and 23). The fluid therapy was given to all the animals both group II and III for correction dehydration and the fluid loss in rumenal acidotic condition before and after surgery.

3.8.2.2 Preparation of Patient

The left flank was prepared for aseptic surgery by clipping the hairs, cleaning with soap water followed by antiseptic chlorhexidine solution. The operative site was scrubbed with surgical spirit and painted with povidone-iodine solution before surgery

(Plate 3). Inj. streptopencillin @ 10 mg / kg body weight was administered intramuscular prior to surgery as pre-operative antibiotic coverage.

3.8.2.3 Anaesthetic Technique

In both the groups II and III surgery was performed under regional block using local linear infiltration and inverted "L" block in the left flank region of the animal. 2 % Lignocaine Hydrochloride® (Inj. Regan Laboraoteries, Hisa) was used as local anesthetic agent. As goats were sensitive to lignocaine, so 1 % was prepared from 2 % by mixing equal volume of drug and distilled water as V/V (1:1). The two finger width away from the transverse process of lumbar vertebrae and two finger widths away from the last rib linear infiltration were done to produce the analgesia at the site of operation (Plate 4).

3.8.2.4 Surgical Procedure

The animals were restrained in sternal recumbancy on the operating table. The surgical site was draped and prepared for the surgery. 10 minutes after regional block the left flank laparotomy was performed. The vertical skin incision was given about 5 cm length with the following landmarks i.e. two finger width away from the transverse process of lumbar vertebrae and two finger widths away from the last rib. Subcutaneous fascia was dissected to expose the external abdominal muscle. All three abdominal muscles were incised vertically similar to skin to expose the peritoneum. Then the peritoneum was incised along with transverse abdominal muscle layer to enter into the abdominal cavity. The distended rumen wall was pulled outside the incision site and stay sutures using nylon were put at two opposite sites, one proximal and another distal to the expected line of incision on the rumen without piercing the mucosa of rumen. The

laparotomy wound was covered with surgical drapes to prevent the seepage of the ruminal contents into the abdomen (Plate 5).

The rumen was incised avoiding the blood vessels of rumen wall (Plate 6). The incision was sufficient enough to allow the hand to pass inside. The acidotic liquid contents along with other undigested food materials of the rumen were evacuated completely (Plate 7 and 8). The rumen was lavaged with normal saline so that the substances adhering to ruminal papillae got cleaned off (Plate 9). A 100 g of presoaked soft dried chopped fodder, 50 g jaggery and 5 g of sodium bicarbonates in 500 ml of water was replaced back into the rumen (Plate 10). The Streptopencillin antibiotic powder was also put inside the rumen and then rumen wall was closed by using Cushing's sutures followed by Lembert's sutures with chromic catgut no.1 (Plate 11). After closer of rumen wall thorough cleaning with normal saline was carried out, stay sutures were removed and rumen was placed back into the abdomen. Peritoneum along with the internal transverse muscle sutured in one layer using chromic catgut no. 1 with lock stitch pattern. Both external and internal abdominal muscles were sutured in one layer with simple continuous pattern using chromic catgut no. 1 (Plate 12). The skin was sutured using nylon with interrupted suture pattern (Plate 13). Surgical wound was dressed with ointment as shown in Plate 14 (Loraxene®, Virbac India Pvt. Ltd.).

3.8.2.5 Post-operative Care

Surgical wound dressing was carried out for each animal of group II and III by using five percent povidone iodine solution and Loraxene® ointment till the healing of the surgical wound. Inj. streptopencillin (10 mg / kg body weight, intramuscular, once a

day for seven days) and Inj. tolfenamic acid (2 mg / kg body weight, intramuscular, once daily for three days) were used as post-operative antibiotics and analgesia to control secondary bacterial infection and inflammation respectively. Owners of the patients were advised for regular inspection of surgical wound for any presence of infection at the site, excess exudates, wound dehiscence and signs of edema. All the animals of each group were given complete rest for initial two weeks after operation. The owners were advised for controlled feeding and water to avoid the stress on the ruminal sutures. After complete healing of laparotomy wound, sutures were removed on 12^{th} day of after operation.

3.8.2.6 Common supportive treatments for all affected animals of each group

Inj. streptopencillin @ 10 mg / kg body weight intraruminally every 12 hours for 3 days in group I only

Inj. thiamine hydrochloride @ 10 mg /kg body weight by intramuscular route once a day for 3 days

Inj. chlorphenaramine maleate @ 2 ml /animal by intramuscular route once a day for 3 days

Inj. calboral (Calcium Borogluconate) 20 ml / goat by intravenous route once in two days for 3 days

Therapeutic efficacy of each group was evaluated based on recovery from clinical illness, physicochemical changes in rumen fluid and blood. Ruminal fluid and blood

samples were collected following standard methods, on different intervals 0 hours (before treatment), 12, 24, 72 and 120 hours after the initiation of the treatment.

3.9 Statistical Analysis

Statistical analysis of data obtained was carried out by employing paired "t" test and ANOVA, as per Snedecor and Cochran (1994).

Table 1. Design of detailed technical programme of clinical study

Sl. No.	Group	Number of animals	Therapeutic line of treatment
1	I	6	1. Isotonic $NaHCO_3$-1.3 % intravenous once a day for 3 days at 12 hours interval for 3 days 2. Bufzone® powder @ 50 g / day oral at 12 hours for 5 days
2	II	6	1. Isotonic $NaHCO_3$-1.3 % intravenous once a day for 3 days at 12 hours interval for 3 days 2. Rumenotomy 3. Ecotas® boli-1bolus orally twice a day for 5 days
3	III	6	1. Isotonic $NaHCO_3$-1.3% intravenous once a day for 3 days at 12 hours interval for 3 days 2. Rumenotomy 3. Cud transplantation -@ 10 ml / kg per oral at 24 hour after surgery for two days

1. Bufzone®: A product of Intas Pharmaceuticals Ltd.,
   Composition: Unique rumen buffer enriched with yeast and metabolic boosters
2. Ecotas®: A product of Intas Pharmaceuticals Ltd.,
   Composition: Each coated bolus contains, Saccharomyces cerevisiae - 25×10^6 CFU, Lactobacillus sporogenes - 20×10^6 CFU, Aspergillus oryzae - 20×10^6 CFU, Biotin - 10 mg, DL-Methionine - 1 g, Zinc sulphate - 200 mg, Cobalt sulphate - 40 mg, Copper sulphate -100 mg and Fructo-oligosaccharide - 250 mg.

Plate 1: Photograph showing oral administration of Bufzone with 10 ml disposable syringe in a goat with acute ruminal acidosis of group I

Plate 2: Clinical case presentation with distended abdomen in a goat with acute ruminal acidosis of group II

Plate 3: Aseptic preparation of surgical site (left flank) for rumenotomy in a goat of acute ruminal acidosis of group II and III

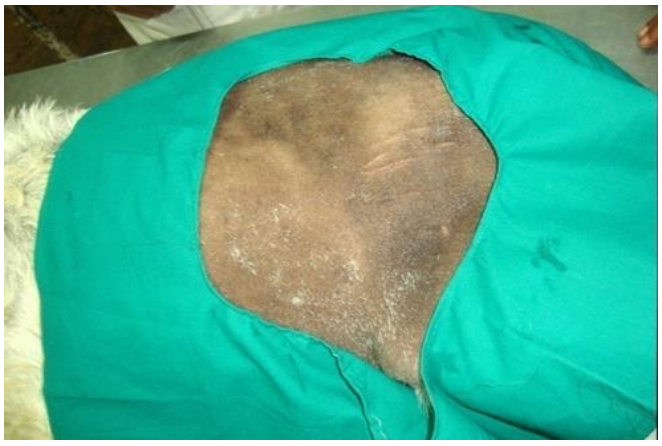


Plate 4: Induction of analgesia in the flank region with regional block using inj. Lignocaine 1% at the surgical site in a goat of acute ruminal acidosis of group II and III

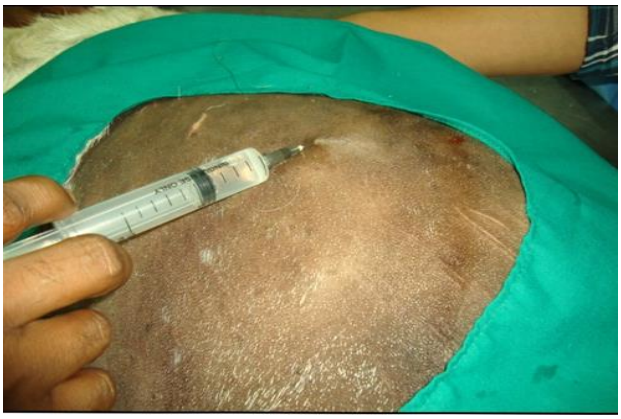


Plate 5: Exteriorization of the rumen, held in position with stay sutures and proper draping of the laparotomy wound at the surgical site in a goat of acute ruminal acidosis of group II and III

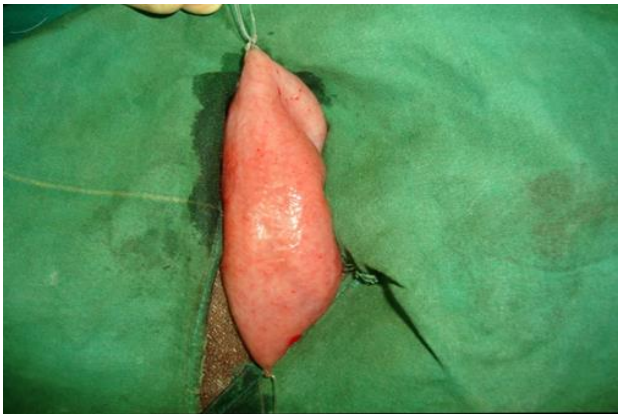


Plate 6: Photograph showing rumenotomy along with ruminal contents from the surgical site in a goat of acute ruminal acidosis of group II and III

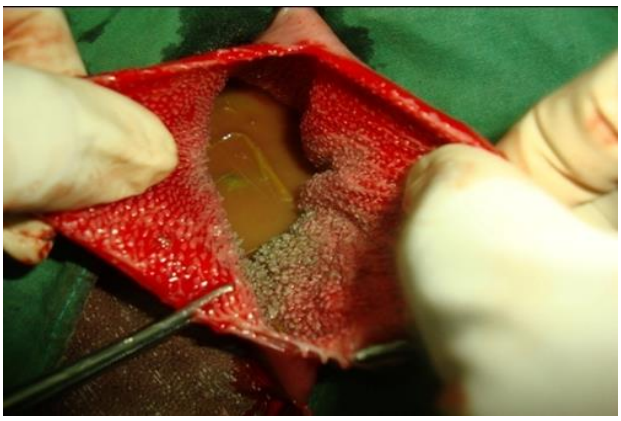


Plate 7: Photograph showing removal of acidotic ruminal fluid and ruminal contents from the rumen in a goat of acute ruminal acidosis of group II and III

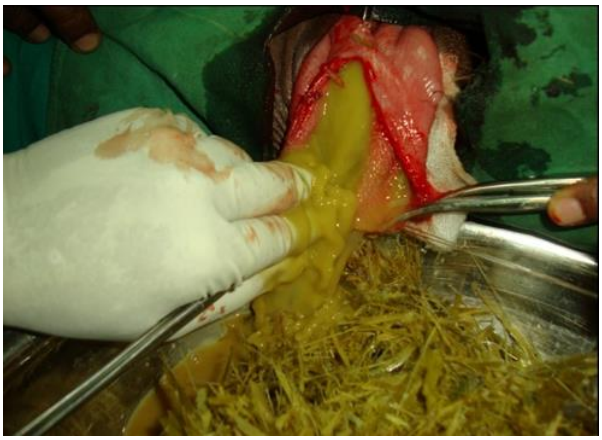


Plate 8: Photograph showing removal of undigested feed from the rumen in a goat of acute ruminal acidosis of group II and III

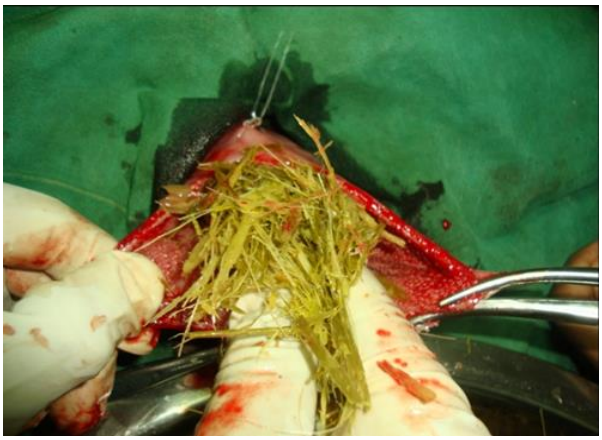


Plate 9: Thorough lavaging of the ruminal wall with normal saline in a goat of acute ruminal acidosis of group II and III

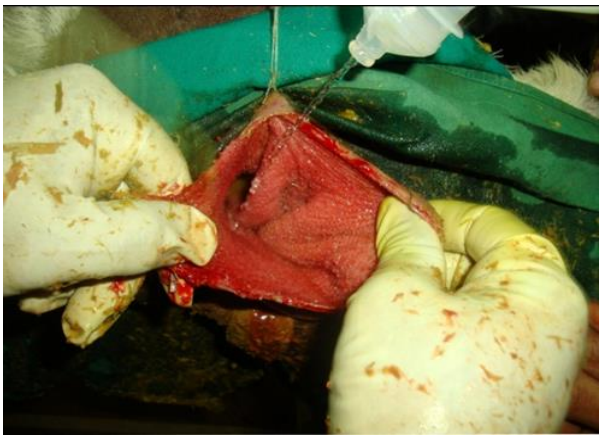


Plate 10: Photograph showing replacement of presoaked chopped fodder in to the rumen in a goat of acute ruminal acidosis of group II and III

Plate 11: Rumen sutured with Cushings and Lemberts suture pattern by chromic catgut no.1 in a goat of acute ruminal acidosis of group II and III

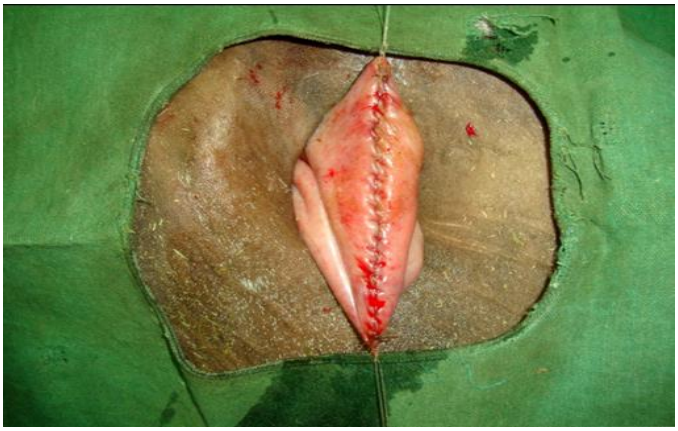


Plate 12: Photograph showing sutured abdominal muscles with lockstitch pattern in two layers using chromic catgut no.1 in a goat of acute ruminal acidosis of group II and III

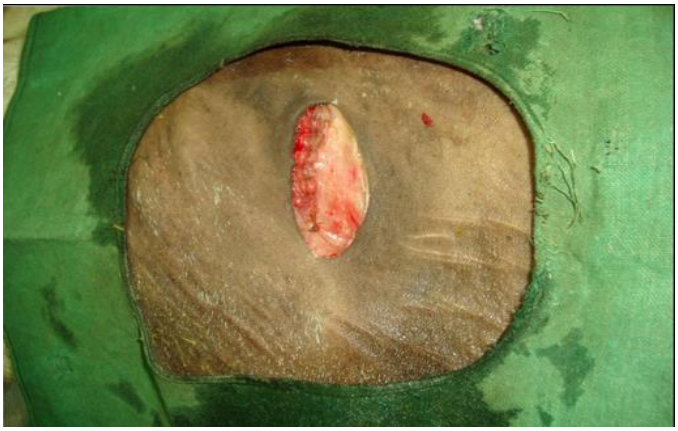


Plate 13: Photograph showing sutured skin wound with vertical mattress using nylon in a goat of acute ruminal acidosis of group II and III

Plate 14: Photograph showing standing of goat immediate after rumenotomy operation and dressing with Loraxene ointment in an acute ruminal acidosis of group II and III

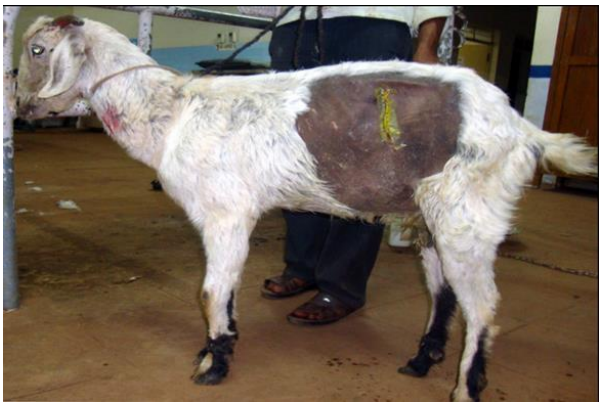


Plate 15: Specially designed orogastric rumen liquor extraction pipe used in a goat of acute ruminal acidosis of group I, II and III

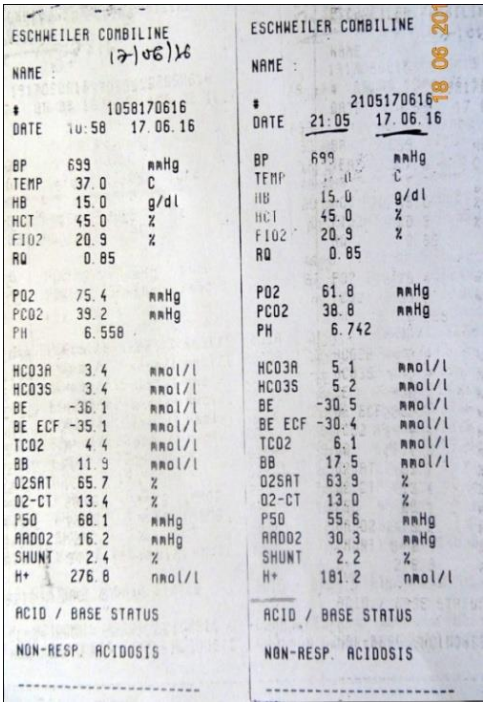
Plate 16: Photograph showing after oral insertion of orogastric rumen liquor extraction pipe passed into the rumen and collection of rumen liquor in a jar of suction pump in a goat of acute ruminal acidosis of group II

Plate 17: Photograph showing collected 150 ml of acidotic rumen liquor in a jar of suction pump from the goat of acute ruminal acidosis of group II

Plate 18: Photograph showing measured pH of 4.3 in ruminal fluid by digital pH meter immediate after collection in a goat of acute ruminal acidosis of group II

Plate 19: Photograph showing ESCHWEILER, Combiline Machine serial number, CL 1241, (CL MICROMED PVT. LTD.) for venous blood gas analysis in a goat of acute ruminal acidosis of group I, II and III

Plate 20: Photograph showing actual print out indicating metabolic acidosis after venous blood gas analysis in Combiline Machine of acute ruminal acidosis of group I, II and III



ESCHWEILER COMBILINE

NAME :

\# 1058170616

DATE 10:58 17.06.16

BP	699	mmHg
TEMP	37.0	C
HB	15.0	g/dl
HCT	45.0	%
FIO2	20.9	%
RQ	0.85	
PO2	75.4	mmHg
PCO2	39.2	mmHg
PH	6.558	
HCO3A	3.4	mmol/l
HCO3S	3.4	mmol/l
BE	-36.1	mmol/l
BE ECF	-35.1	mmol/l
TCO2	4.4	mmol/l
BB	11.9	mmol/l
O2SAT	65.7	%
O2-CT	13.4	%
P50	68.1	mmHg
AADO2	16.2	mmHg
SHUNT	2.4	%
H+	276.8	nmol/l

ACID / BASE STATUS

NON-RESP. ACIDOSIS

ESCHWEILER COMBILINE

NAME :

\# 2105170616

DATE 21:05 17.06.16

BP	699	mmHg
TEMP	[illegible]	C
HB	15.0	g/dl
HCT	45.0	%
FIO2	20.9	%
RQ	0.85	
PO2	61.8	mmHg
PCO2	38.8	mmHg
PH	6.742	
HCO3A	5.1	mmol/l
HCO3S	5.2	mmol/l
BE	-30.5	mmol/l
BE ECF	-30.4	mmol/l
TCO2	6.1	mmol/l
BB	17.5	mmol/l
O2SAT	63.9	%
O2-CT	13.0	%
P50	55.6	mmHg
AADO2	30.3	mmHg
SHUNT	2.2	%
H+	181.2	nmol/l

ACID / BASE STATUS

NON-RESP. ACIDOSIS

Plate 21: Photograph showing healthy goat slaughtered and exposure of rumen and other visceral organs in a slaughter house

Plate 22: Photograph showing straining of collected fresh ruminal fluid with a muslin cloth into a beaker

Plate 23: Photograph showing oral administration of strained fresh ruminal fluid with 10 ml disposable syringe after rumenotomy in a goat of acute ruminal acidosis of group III

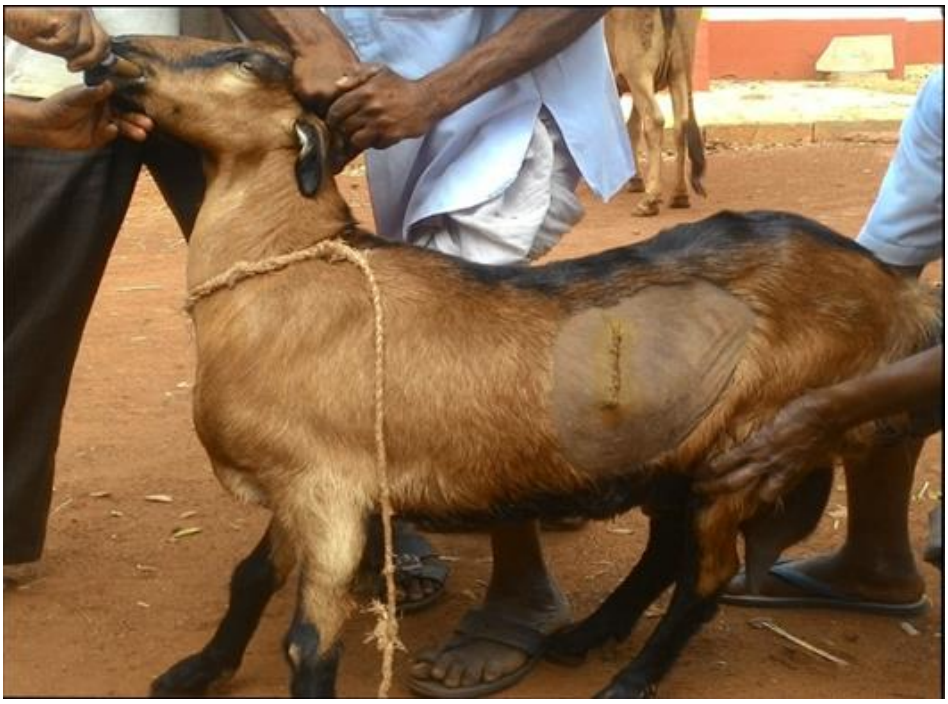
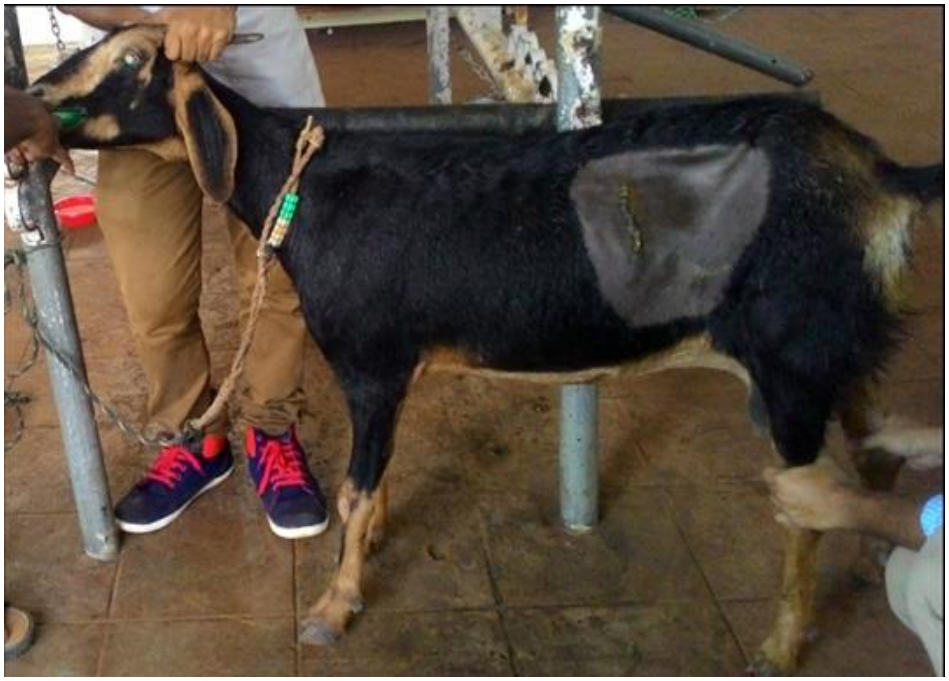


Plate 24: Photograph showing oral administration of Ecotas bolus solution with 10ml disposable syringe in a goat of acute ruminal acidosis of group II



Results

IV. RESULTS

4.1 Prevalence

Prevalence of ruminal acidosis in goats of Bidar district was evaluated by analyzing the data of clinical cases presented for a period of five years from January to December (2011-2016) to Veterinary Clinical Complex, Veterinary College, Bidar, and five taluka Veterinary Hospitals of Bidar district *viz*., Aurad, Bidar, Basavakalyan, Bhalki, and Humnabad. Among the goats presented during the period of study, a total 1,93,217 clinical cases presented to five taluka Veterinary Hospitals and Veterinary College, Bidar, out of them the total goat cases presented were 66,667. The total cases of digestive disorders with various systemic disorders recorded were 25,693. An overall 1071 (0.55%) cases of ruminal acidosis in goats were recorded as shown in Table: 2 and 3 and Fig. 1 and Fig. 2.

4.1.1 Prevalence of ruminal acidosis in goats with respect to sex at five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Prevalence of ruminal acidosis in goats with respect to sex at five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years recorded were presented in table: 2. A total of 1071 cases of ruminal acidosis were recorded in goats. Among 1071 cases 954 (89.08%) females and 117 (10.92 %) males were recorded. The higher prevalence of ruminal acidosis was recorded in females than in male goats as shown in Fig. 3.

4.1.2 Prevalence of ruminal acidosis in goats at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

A total of 33,885 clinical cases were presented to Veterinary Clinical Complex, Veterinary College, Bidar out of them the total goat cases presented were 8,829. Total cases of digestive disorders with various systemic disorders recorded were 2,316. An overall 304 (0.89%) cases of ruminal acidosis in goats were recorded as shown in Table: 4 and Fig. 2.

4.1.3 Prevalence of ruminal acidosis in goats with respect to age at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Age wise prevalence of ruminal acidosis in goats at Veterinary Clinical Complex, Bidar for a period of five years were recorded and presented in table: 5. A total of 304 cases of ruminal acidosis were recorded in goats. Among 304 cases 128 (42.10%) 1 -2 years, 106 (34.87%) less than or equal to 1 year and 70 (23.03 %) more than 2 years of age were observed. The highest prevalence of ruminal acidosis were recorded in 1-2 years of age groups, followed by less than 1 year and least in more than 2 years of age groups in goats as shown in Table: 5 and Fig. 4.

4.1.4 Prevalence of ruminal acidosis in goats with respect to sex at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Sex wise prevalence of ruminal acidosis in goats at Veterinary Clinical Complex, Bidar for a period of five years were recorded and presented in Table: 6. A total of 304

cases of ruminal acidosis were recorded in goats. Among 304 cases 237 (77.96%) females and 67 (22.04 %) males were observed. The higher prevalence of ruminal acidosis was recorded in females than the male goats as shown in Table: 6 and Fig. 5.

4.1.5 Prevalence of ruminal acidosis in goats with respect to source of carbohydrate at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Prevalence of ruminal acidosis in goats with respect to source of carbohydrate at Veterinary Clinical Complex, Bidar for a period of five years were recorded and presented in Table: 7. A total of 304 cases of ruminal acidosis were recorded in goats. Among 304 cases 62 (20.40 %) rice, 32 (10.52 %) wheat, 21(6.90 %) jawar, 6 (1.98 %) vegetables, 4 (1.32 %) grains, 4 (1.32 %) maize, 1(0.33 %) fruits and 174 (57.23 %) not known cases were observed. The highest prevalence of ruminal acidosis were recorded with rice, followed by wheat, jawar, vegetables, grains, maize and lastly fruits in goats among the known causes as shown in Table: 7, Fig. 6 and 7.

4.1.6 Prevalence of ruminal acidosis in goats with respect to season at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Prevalence of ruminal acidosis in goats with respect to season at Veterinary Clinical Complex, Bidar for a period of five years were recorded and presented in Table: 8. A total of 304 cases of ruminal acidosis were recorded in goats. Among 304 cases 111 (36.51 %) in monsoon, 79 (25.99 %) in summer, 64 (21.05%) in post monsoon and 50 (16.45 %) in winter was observed. The highest prevalence of ruminal acidosis was

recorded in the monsoon season followed by summer, post monsoon and least in winter season as shown in Table: 8 and Fig. 8.

4.1.7 Prevalence of ruminal acidosis in goats with respect to breed at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Prevalence of ruminal acidosis in goats with respect to breed at Veterinary Clinical Complex, Bidar for a period of five years (2011-2016) were observed in non-descript and Osmanabadi breed of goats, as the population of this breed in and around Bidar district were more compared to other breeds of goats.

4.2 Selection of animals

The research was conducted in clinical cases of eighteen goats suffering from acute ruminal acidosis. The animals were randomly divided into three groups of six animals in each group.

4.3 CLINICAL EXAMINATION

The history from the cases was collected with respect to etiology, duration of illness and any previous medication had been given to treat the disorder. The details of the etiology, duration of illness and major clinical sign shown by the animals were recorded. In most of the cases presented were treated for anorexia and distension of abdomen with Vitamin B complex inj. and carminative mixtures. However, with a lag period of 12 to 24 hours as there was no improvements in the conditions the animals were presented to the Veterinary College, Bidar for specialized treatment. The clinical signs

shown by the goats were recorded followed by systematic clinical examination was performed and diagnosed as acute ruminal acidosis.

The onsets of illness after consumption of readily fermentable carbohydrates in goats observed were 24 to 48 hours. Out of eighteen cases, 12 cases with 24 hours followed by 5 cases with 48 hours and least with 12 hours in 1 case with acute ruminal acidosis in goats were recorded.

The clinical signs shown by the animal were anorexia, distension of abdomen, passing of loose feaces, regurgitation of cud from the mouth and nostril, fluid thrill on palpation of abdomen, dull, recumbent, hurried respiratory rate and heart rate, grinding of teeth, dehydration and death in goats affected with ruminal acidosis as shown in plate (25, 26, 27, and 28).

The diagnosis of the cases was confirmed by correlating the history, clinical signs, clinical examination and ruminal fluid examination. Acute case of ruminal acidosis was confirmed pH of the ruminal fluid has shown less than 5.5 pH reading in digital pH meter as shown in plate (47 and 48). However, the detailed clinico-physiological, heamato-biochemical changes in ruminal fluid and venous blood changes in the acute ruminal acidosis were elaborated in detail as discussed below with tables and figures.

The vital signs in clinical cases of acute ruminal acidosis were examined from the day of presentation (0 hour, before treatment) and after initiation of treatment at 12 hours, 24 hours, 72 hours and 120 hours. The temperature, heart rate and respiratory rate were recorded in all the goats subjected for therapeutic and surgical treatment.

4.3.1 Temperature (^{0}F)

The Mean ± SE., values of rectal temperature (^{0}F) before and after treatment in different groups are given in Table: 9.

The Mean ± SE., values of rectal temperature of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 101.33 ± 0.35, 101.80 ± 0.28, 101.95 ± 0.32, 101.81 ± 0.23 and 101.91 ± 0.30 respectively.

The Mean ± SE., values of rectal temperature of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 102.48 ± 0.45, 100.26 ± 0.25, 101.38 ± 0.16, 101.98 ± 0.14 and 102.76 ± 0.12 respectively.

The Mean ± SE., values of rectal temperature of group III animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 102.13 ± 0.26, 100.65 ± 0.23, 101.71 ± 0.42, 102.11 ± 0.46 and 101.98 ± 0.27 respectively.

The results have shown significant difference in the temperature within the group and in between the groups. The temperature in all the groups from 0 hours to 120 hours has fluctuated within the normal range in all intervals within the group and between the groups. However, significantly lower ($P \leq 0.01$) temperature was observed at 12 hours interval after treatment in the group II and III compared to before treatment (0 hour). Whereas in group I no changes were found at 12 hour interval after treatment and it

significantly ($P \leq 0.05$) had higher temperature from the other two groups (II and III) at corresponding interval. In group II at 24 hours significantly ($P \leq 0.05$) had higher temperature compared to 12 hours after treatment. In group II higher temperature was recorded at 120 hours interval compared to other two groups I and III at corresponding interval.

4.3.2 Heart rate (beats/minute)

The Mean ± SE., values of heart rate (beats / minute) before and after treatment in different groups are given in Table: 10 and Fig. 9.

The Mean ± SE., values of heart rate of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 108.00 ± 4.89, 103.33 ± 4.30, 98.16 ± 3.57, 89.83 ± 3.35 and 76.33 ± 2.15 respectively.

The Mean ± SE., values of heart rate of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 110.00 ± 6.17, 101.66 ± 4.37, 99.5 ± 3.61, 92.16 ± 2.99 and 82.50 ± 3.07 respectively.

The Mean ± SE., values of heart rate of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 116.33 ± 3.55, 102.33 ± 4.85, 100.66 ± 6.56, 90.50 ± 1.25 and 79.83 ± 1.97 respectively.

Increased heart rate from 0 hours before treatment decreased significantly at 120 hours after treatment in all groups. Initial decrease in heart rate was observed in the treated groups I, II and III at 12 hours interval and there after a significant ($P \leq 0.05$) drop in the heart rate was observed in group I, II and III at 72 hours interval after treatment. In

group III significant ($P \leq 0.01$) drop in the heart rate was observed ($P \leq 0.01$) at 72 hours interval after treatment.

There was no significant difference found between the groups I, II and III at all corresponding treatment intervals. However, the values have reached near to normal physiological range at 120 hours of after treatment.

4.3.3 Respiratory rate (breaths /minute)

The Mean ± SE., values of respiratory rate (breaths / minute) before and after treatment in different groups are given in Table: 11

The Mean ± SE., values of respiratory rate of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 44.50 ± 4.68, 38.83 ± 2.71, 35.50 ± 2.36, 34.00 ± 1.03 and 29.00 ± 0.85 respectively.

The Mean ± SE., values of respiratory rate of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 45.83 ± 2.94, 39.33 ± 3.44, 30.66 ± 2.67, 27.50 ± 1.74 and 29.83 ± 1.60 respectively.

The Mean ± SE., values of respiratory rate of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 43.33 ± 2.40, 33.66 ± 4.99, 31.83 ± 3.18, 25.83 ± 1.86 and 28.33 ± 0.98 respectively.

Respiratory rate decreased significantly from 0 hours to 120 hours in all the three groups. The significant ($P \leq 0.01$) decrease was observed at 24 hours onwards in group II

and III compared to the group I at corresponding interval after treatment. In group I the significant ($P \leq 0.01$) lower respiratory rate was observed at 120 hours after treatment.

In group II and III at 72 hours of treatment interval the respiratory rate differed significantly ($P \leq 0.05$) lower compared with group I at corresponding interval. However in group II at 72 hours after treatment the higher respiratory rate was observed compared to group III at corresponding interval.

4.3.4 Rumen contractions (per two minutes)

The Mean ± SE., values of rumen contractions (per two minutes) before and after treatment in different groups are given in Table: 12

In all the groups the rumen contractions were nil at 0 hour before treatment interval. The Mean ± SE., values of rumen contractions at 72 hours and 120 hours after the treatment were: 1.75 ± 0.11, 2.25 ± 0.11 of group I, 2.08 ± 0.27, 2.50 ± 0.22 of group II and 2.75 ± 0.11 and 2.91 ± 0.08 of group III animals respectively.

Rumen contraction rates were nil at 0 hour interval and observed only at 72 hours and 120 hours after treatment in all the groups. The values were significantly ($P \leq 0.01$) higher at 72 hours and 120 hours compared to 0 hour interval in all the three groups. At 120 hours they reached towards normal rate in all the three groups.

In group II and III at 72 hours and 120 hours of after treatment interval the rumen contraction rate was significantly ($P \leq 0.05$) higher compared with group I at corresponding interval. However, in group II and III at 120 hours after treatment the significant difference was not observed compared to group I at corresponding interval.

4.4 RUMINAL FLUID ANALYSIS

4.4.1 Physical Examination

4.4.1.1 Colour of rumen fluid

The changes in the rumen fluid colour before and after the treatment interpreted in Table: 13

The colour of ruminal fluid was milky white at ‘0’ hour before treatment in all acidotic goats.

In group I the colour changed to light green at 72 hours after treatment and reached to normal green at 120 hours after treatment compared to milky white at 0 hour before treatment.

In group II animals the colour changed to light green at 12 hours and persisted up to 72 hours there after reached to normal green at 120 hours after treatment compared to milky white at 0 hour before treatment.

In group III animals the colour changed to light green at 12 hours after treatment and reached to normal green at 24 hours to 120 hours after treatment compared to milky white at 0 hour before treatment.

4.4.1.2 Odour of ruminl fluid

The changes in the odour of rumen liquor before and after the treatment are shown in Table: 14

In group I the odour of the ruminal fluid was sour at ‘0’ hour before treatment, remained up to 24 hours after treatment and then mild aromatic odour was observed at 72 hours after treatment interval. The odour was moderate aromatic at 120 hours after treatment interval.

In group II the odour of the ruminal fluid was sour at ‘0’ hour before treatment and mild aromatic odour was observed at 12 hours after treatment, remained up to 72 hours after treatment interval. The odour was moderate aromatic at 120 hours after treatment interval.

In group III the odour of the ruminal fluid was sour at ‘0’ hour before treatment and mild aromatic odour was observed at 12 hours after treatment. There after it was changed to moderate aromatic and persisted up to 72 hours after treatment interval. The odour was normal aromatic at 120 hours after treatment interval.

4.4.1.3 Consistency of ruminl fluid

The changes in the consistency of rumen liquor before and after the treatment are shown in Table: 15

In group I the consistency of the ruminal fluid was watery at ‘0’ hour before treatment, remained up to 24 hours after treatment and then mild viscous was observed at 72 hours after treatment interval. The consistency was moderately viscous at 120 hours after treatment interval.

In group II the consistency of the ruminal fluid was watery at ‘0’ hour before treatment. It was observed that a clear water like consistency from 12 hours to 24 hours

after treatment and then shifted to mild viscous consistency at 72 hours after treatment interval. The consistency was moderate viscous at 120 hours after treatment interval.

In group III the odour of the ruminal fluid was sour at '0' hour before treatment. It was observed that clear water like consistency at 12 hours after treatment and then shifted to mild viscous consistency at 24 hours after treatment interval. The consistency was moderately viscous at 72 hours after treatment interval there after normal viscosity seen at 120 hours after treatment interval.

4.4.2 pH of Rumen fluid

The Mean ± SE., values of rumen fluid pH before and after treatment in different groups are given in Table: 16 and Fig. 10.

The Mean ± SE., values of rumen fluid pH of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 4.78 ± 0.25, 5.63 ± 0.05, 6.00 ± 0.07, 6.40 ± 0.05 and 6.81 ± 0.07 respectively.

The Mean ± SE., values of rumen fluid pH of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 4.68 ± 0.19, 6.53 ± 0.09, 6.81 ± 0.05, 6.76 ± 0.08 and 6.88 ± 0.06 respectively.

The Mean ± SE., values of rumen fluid pH of group III animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 4.70 ± 0.26, 6.60 ± 0.06, 6.81 ± 0.06, 6.78 ± 0.09 and 6.90 ± 0.06 respectively.

In group I, II and III animals the significant ($P \leq 0.01$) higher values of pH was observed at 12 hours, 24 hours, 72 hours and 120 hours after treatment compared to 0 hour before treatment intervals. The pH values were lower before treatment and reached towards normal at 72 hours after treatment in group I and at 12 hours after treatment in group II and III.

Comparison between the groups showed significant difference at 12 hours, 24 hours and 72 hours after treatment intervals. The group I showed significant ($P \leq 0.05$) lower value at 12 hours, 24 hours and 72 hours after treatment compared to group II and III at corresponding treatment intervals. The group II not showed any significant difference at any given interval compared to group III at corresponding treatment intervals.

4.4.3 Gram Positive to Gram Negative Ratio

4.4.3.1 Gram positive bacteria count (%)

The Mean ± SE., values of gram positive bacteria count (%) before and after treatment in different groups are given in Table: 17 and Fig. 11.

The Mean ± SE., values of gram positive bacteria count of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 81.84 ± 1.64, 60.16 ± 2.03, 48.50 ± 1.43, 30.50 ± 1.66 and 22.66 ± 0.66 respectively.

The Mean ± SE., values of gram positive bacteria count of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment

were: 82.83 ± 1.49, 29.33 ± 1.45, 20.66 ± 0.88, 19.66 ± 0.80 and 22.33 ± 0.66 respectively.

The Mean ± SE., values of gram positive bacteria count of group III animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 81.83 ±1.53, 30.66 ± 1.70, 21.50 ± 1.33, 22.00 ± 0.85 and 21.83 ± 1.16 respectively.

In group I, II and III animals the significant ($P \leq 0.01$) lower values of gram positive bacteria count was observed at 12 hours, 24 hours, 72 hours and 120 hours after treatment compared to 0 hour before treatment intervals. In all the groups the values were higher before treatment reached towards normal at 120 hours after treatment.

Comparison between the groups showed significant difference at 12 hours, 24 hours and 72 hours after treatment intervals. The group I showed significant ($P \leq 0.05$) higher value at 12 hours, 24 hours and 72 hours after treatment compared to group II and III at corresponding treatment intervals. The group II not showed any significant difference at any given interval compared to group III at corresponding treatment intervals.

4.4.3.2 Gram negative bacteria count (%)

The Mean ± SE., values of gram negative bacteria count (%) before and after treatment in different groups are given in Table: 18 and Fig. 12.

The Mean ± SE., values of gram negative bacteria count of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment

were: 18.16 ± 1.64, 39.83 ± 2.03, 53.16 ± 2.27, 69.50 ± 1.66 and 77.33 ± 0.66 respectively.

The Mean ± SE., values of gram negative bacteria count of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 17.16 ± 1.49, 70.66 ± 1.45, 79.33 ± 0.88, 80.33 ± 0.80 and 77.66 ± 0.66 respectively.

The Mean ± SE., values of gram negative bacteria count of group III animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 18.16 ± 1.53, 69.33 ± 1.70, 78.50 ± 1.33, 78.00 ± 0.85 and 78.16 ± 1.16 respectively.

In group I, II and III animals the significant ($P \leq 0.01$) higher values of gram negative bacteria count was observed at 12 hours, 24 hours, 72 hours and 120 hours after treatment compared to 0 hour before treatment intervals. In all the groups the values were lower before treatment reached towards normal at 120 hours after treatment.

Comparison between the groups showed significant difference at 12 hours, 24 hours and 72 hours after treatment intervals. The group I showed significant ($P \leq 0.05$) lower value at 12 hours, 24 hours and 72 hours after treatment compared to group II and III at corresponding treatment intervals. The group II not showed any significant difference at any given interval compared to group III at corresponding treatment intervals.

4.4.4 Protozoal Density

The changes in the protozoal density of rumen liquor before and after the treatment are shown in Table: 19

In group I the protozoal density of the ruminal fluid was nil at '0' hour before treatment, remained up to 24 hours after treatment and then 1-10 protozoal concentration was observed at 72 hours after treatment interval. The protozoal concentration was 10-20 at 120 hours after treatment interval.

In group II the protozoal density of the ruminal fluid was nil at '0' hour before treatment. It was observed that 1-10 protozoal concentration from 12 hours to 24 hours after treatment and then shifted to 10-20 protozoal concentration from 72 hours to 120 hours after treatment interval.

In group III the protozoal density of the ruminal fluid was nil at '0' hour before treatment. It was observed that 1-10 protozoal concentration at 12 hours after treatment and then shifted to 10-20 protozoal concentration at 24 hours after treatment interval. The normal protozoal concentration of 20-30 was observed from 72 hours to 120 hours after treatment interval.

4.4.5 Methylene Blue Reduction Time (in minutes)

The Mean ± SE., values of methylene blue reduction time (in minutes) before and after treatment in different groups are given in Table: 20 and Fig. 13.

The Mean ± SE., values of methylene blue reduction time of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the

treatment were: 64.61 ± 1.07, 52.22 ± 0.73, 37.99 ± 2.25, 26.95 ± 1.11 and 10.34 ± 0.24 respectively.

The Mean ± SE., values of methylene blue reduction time of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 63.72 ± 0.94, , 28.68 ± 2.06, 21.28 ± 0.88, 13.70 ± 0.99 and 9.99 ± 0.34 respectively.

The Mean ± SE., values of methylene blue reduction time of group III animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 66.40 ± 0.98, 27.76 ± 1.09, 18.80 ± 1.01, 10.95 ± 0.39 and 9.12 ± 0.32 respectively.

In group I, II and III animals the significant ($P \leq 0.01$) lower values of methylene blue reduction time was observed at 12 hours, 24 hours, 72 hours and 120 hours after treatment compared to 0 hour before treatment intervals. In all the groups the values were higher before treatment reached towards normal at 120 hours after treatment.

Comparison between the groups showed significant difference at 12 hours, 24 hours, 72 hours and 120hours after treatment intervals. The group I showed significant ($P \leq 0.05$) higher value at 12 hours, 24 hours 72 hours and 120hours after treatment compared to group II and III at corresponding treatment intervals. The group II showed significant ($P \leq 0.05$) higher value at 12 hours, 24 hours 72 hours and 120 hours after treatment compared to group III at corresponding treatment intervals.

4.4.6 Sedimentation Activity Time (in minutes)

The Mean ± SE., values of sedimentation activity time (in minutes) before and after treatment in different groups are given in Table: 21

In all the groups the sedimentation activity time was absent at 0 hour before treatment, 12 hours and 24 hours after treatment interval.

The Mean ± SE., values of sedimentation activity time of group I animals at 72 hours and 120 hours after the treatment were: 22.22 ± 0.95 and 10.80 ± 0.19 respectively.

The Mean ± SE., values of sedimentation activity time of group II animals at 72 hours and 120 hours after the treatment were: 15.61 ± 0.51 and 11.12 ± 0.31respectively.

The Mean ± SE., values of sedimentation activity time of Group III animals at 72 hours and 120 hours after the treatment were: 11.75 ± 0.60 and 10.76 ± 0.23 respectively.

In group I, II and III animals the significant ($P \leq 0.01$) difference of Sedimentation activity time was observed at 72 hours and 120 hours after treatment compared to 0 hour before treatment intervals. In all the groups the values reached towards normal at 120 hours after treatment.

Comparison between the groups showed significant difference at 72 hours after treatment intervals. The group I showed significant ($P \leq 0.05$) higher value at 72 hours after treatment compared to group II and III at corresponding treatment intervals. The group II showed significant ($P \leq 0.05$) higher value at 72 hours after treatment compared to group III at corresponding treatment intervals.

4.4.7 Total volatile fatty acids (mmol / dL)

The Mean ± SE., values of total volatile fatty acids (mmol / dL) before and after treatment in different groups are given in Table: 22 and Fig. 14.

The Mean ± SE., values of total volatile fatty acids of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 66.40 ± 1.73, 62.81 ± 2.03, 50.38 ± 0.92, 40.70 ± 2.25 and 24.61 ± 1.74 respectively.

The Mean ± SE., values of total volatile fatty acids of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 68.60 ± 1.48, 8.38 ± 0.41, 12.37 ± 0.33, 16.89 ± 0.72 and 25.20 ± 1.70 respectively.

The Mean ± SE., values of total volatile fatty acids of group III animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 70.87 ± 1.59, 8.93 ± 0.34, 17.90 ± 0.57, 31.22 ± 1.32 and 23.26 ± 0.85 respectively.

In group I, II and III animals the significant ($P \leq 0.01$) lower values of total volatile fatty acids was observed at 12 hours, 24 hours, 72 hours and 120 hours after treatment compared to 0 hour before treatment intervals. In all the groups the values were higher before treatment reached towards normal at 120 hours after treatment.

Comparison between the groups showed significant difference at 12 hours, 24 hours and 72 hours after treatment intervals. The group I showed significant ($P \leq 0.05$) higher value at 12 hours, 24 hours and 72 hours after treatment compared to group II and III at corresponding treatment intervals. The group II showed significant ($P \leq 0.05$)

lower value at 24 hours and 72 hours after treatment compared to group III at corresponding treatment intervals.

4.5 HAEMATOLOGICAL ANALYSIS

4.5.1 Total erythrocyte count ($\times 10^6/\mu L$)

The Mean ± SE., values of total erythrocyte count ($\times 10^6/\mu L$) before and after treatment in different groups are given in Table: 23 and Fig. 15.

The Mean ± SE., values of total erythrocyte count of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after treatment were: 12.21 ± 0.19, 10.81 ± 0.15, 10.23 ± 0.21, 9.53 ± 0.30 and 8.73 ± 0.20 respectively.

The Mean ± SE., values of total erythrocyte count of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after treatment were: 12.48 ± 0.32, 11.15 ± 0.18, 10.13 ± 0.19, 9.26 ± 0.19 and 8.65 ± 0.15 respectively.

The Mean ± SE., values of total erythrocyte count of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after treatment were: 12.81 ± 0.30, 11.35 ± 0.23, 10.68 ± 0.26, 9.55 ± 0.27 and 8.66 ± 0.23 respectively.

In all the three groups the total erythrocyte count values showed declined trend from 0 hour before treatment to 120 hours after treatment and the values ranged were within the physiological limits in all the groups. The significant ($P \leq 0.01$) declinations in the values were observed at 12 hours to up to 120 hours interval after treatment in all the three groups.

When compared between the groups no significant difference was observed in the total erythrocyte count values at any corresponding intervals. The values of TEC were within the physiological limits from '0' hour before treatment to 120 hours after treatment in all the groups.

4.5.2 Total Leucocytes Count ($\times 10^3/\mu L$)

The Mean ± SE., values of total leucocytes count ($\times 10^3/\mu L$) before and after treatment in different groups are given in Table: 24 and Fig. 16.

The Mean ± SE., values of total leucocytes count of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after treatment were: 13.19 ± 0.47, 13.20 ± 0.48, 12.51 ± 0.43, 10.98 ± 0.32 and 10.48 ± 0.28 respectively.

The Mean ± SE., values of total leucocytes count of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after treatment were: 13.41 ± 0.26, 12.7 ± 0.29, 11.21 ± 0.27, 10.38 ± 0.21and 9.88 ± 0.13 respectively.

The Mean ± SE., values of total leucocytes count of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after treatment were: 13.73 ± 0.19, 12.73 ± 0.23, 11.15 ± 0.32, 10.00 ± 0.14 and 9.85 ± 0.16 respectively.

In group I animals, significant ($P \leq 0.01$) lower values of TLC observed at 72 hours and 120 hours compared to before treatment intervals. In Group II animals, significant ($P \leq 0.01$) lower values of TLC observed at 24 hours, 72 hours and 120 hours compared to before treatment intervals. In group III animals, significant ($P \leq 0.01$) lower values of TLC observed at 12 hours, 24 hours, 72 hours and 120 hours compared to

before treatment intervals. In all the groups the values were within normal physiological range at all corresponding intervals.

Comparison between the groups showed significant difference at 24 hours and 72 hours after treatment intervals. In group I showed significant ($P \leq 0.05$) higher value of TLC at 24 hours and 72 hours after treatment compared to group II and III at corresponding treatment intervals. In group II the higher TLC were observed significantly ($P \leq 0.05$) at only 72 hours after treatment intervals when compared with group III.

4.5.3 Heamoglobin (g / dL)

The Mean ± SE., values of heamoglobin (g / dL) before and after treatment in different groups are given in Table: 25 and Fig. 17.

The Mean ± SE., values of heamoglobin of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 10.85 ± 0.18, 9.91 ± 0.16, 10.36 ± 0.19, 10.16 ± 0.20 and 9.93 ± 0.13 respectively.

The Mean ± SE., values of heamoglobin of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 11.40 ± 0.26, 10.58 ± 0.30, 10.21 ± 0.23, 9.75 ± 0.14 and 9.26 ± 0.13 respectively.

The Mean ± SE., values of heamoglobin of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 11.11 ± 0.31, 10.00 ± 0.30, 10.13 ± 0.20, 9.53 ± 0.19 and 8.96 ± 0.20 respectively.

The values of haemoglobin compared within the group have decreased significantly from 0 hours to 120 hours in all the three groups. In group I ($P \leq 0.01$) and group III ($P \leq 0.05$) haemoglobin decreased significantly at 12 hours after treatment where as in group II ($P \leq 0.01$) it decreased significantly at 24 hours after treatment. There after the values decreased up to 120 hours after treatment in all the three groups. The haemoglobin level was significantly ($P \leq 0.01$) lower, when compared to before treatment; however, these Hb values were within lower physiological limits.

When compared the haemoglobin values between the groups, in group II and III significant ($P \leq 0.05$) lower Hb was observed at 120 hours after treatment than the group I at corresponding interval. When compared Hb value at 120 hours between group II and III no significant difference was observed at corresponding interval after treatment.

4.5.4 Packed Cell Volume (%)

The Mean ± SE., values of packed cell volume (%) before and after treatment in different groups are given in Table: 26 and Fig. 18.

The Mean ± SE., values of packed cell volume of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 34.81 ± 0.83, 30.63 ± 0.45, 31.00 ± 0.59, 29.48 ± 0.55 and 26.23 ± 0.42 respectively.

The Mean ± SE., values of packed cell volume of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 36.88 ± 0.90, 32.33 ± 0.67, 30.96 ± 0.57, 28.28 ± 0.74 and 26.15 ± 0.38 respectively.

The Mean ± SE., values of packed cell volume of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after treatment were: 38.65 ± 0.64, 32.73 ± 0.88, 30.55 ± 0.86, 27.86 ± 0.43 and 25.65 ± 0.56 respectively.

The packed cell volume values within the groups have showed significant decline from the day of presentation i.e. 0 hour before treatment to 120 hours intervals after treatment in all the groups. In all the three groups the significant ($P \leq 0.01$) decline in the values of packed cell volume was observed at 12 hours after treatment and continued up to 120 hours after treatment and reached lower physiological limits at 120 hours compared to before treatment.

When compared between the groups, in group I and II the packed cell volume differ significantly ($P \leq 0.05$) lower than group III at 0 hour corresponding intervals before treatment. However the significant ($P \leq 0.05$) lower PCV was observed at 72 hours after treatment in group III when compared to group II at corresponding intervals. The values were declined at 120 hours without significant difference between the groups in all the three groups.

4.5.5 Differential Leucocyte Count (DLC)

4.5.5.1 Neutrophils (%)

The Mean ± SE., values of neutrophils (%) before and after treatment in different groups are given in Table: 27 and Fig. 19.

The Mean ± SE., values of neutrophils group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 24.00 ± 0.99, 26.66 ± 1.64, 25.50 ± 1.20, 23.66 ± 0.55 and 23.83 ± 0.47 respectively.

The Mean ± SE., values of neutrophils of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 24.66 ± 1.11, 32.33 ± 0.95, 31.16 ± 0.83, 27.16 ± 0.60 and 23.33 ± 1.08 respectively.

The Mean ± SE., values of neutrophils of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 23.33 ± 0.98, 34.83 ± 1.53, 31.66 ± 1.45, 26.16 ± 1.13 and 24.16 ± 0.74 respectively.

In group I animals there was no significant difference found at 0 hours to 120 hours before and after treatment intervals. In group II and III animals, neutrophils showed significant difference at 12 hours after treatment, where it increased significantly ($P \leq 0.01$) and continued up to 24 hours after treatment compared to value before treatment. In all the groups the values fluctuated within normal physiological range at all corresponding intervals.

Comparison between the groups showed significant difference at 12 hours, 24 hours and 72 hours after treatment intervals. The group I showed significant ($P \leq 0.05$) lower value at 12 hours, 24 hours and 72 hours after treatment compared to group II and III at corresponding treatment intervals. In group II higher neutrophils were observed significantly ($P \leq 0.05$) at only 72 hours after treatment intervals compared with group III.

4.5.5.2 Lymphocytes (%)

The Mean ± SE., values of lymphocytes (%) before and after treatment in different groups are given in Table: 28 and Fig. 20.

The Mean ± SE., values of lymphocytes of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 69.16 ± 0.60, 67.50 ± 1.30, 68.00 ± 1.15, 69.00 ± 0.81 and 69.66 ± 0.61 respectively.

The Mean ± SE., values of lymphocytes of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after treatment were: 69.00 ± 0.57, 60.83± 1.35, 62.5 ± 1.60, 67.00 ± 0.96 and 69.5 ± 0.56 respectively.

The Mean ± SE., values of lymphocytes of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after treatment were: 69.16 ± 0.60, 58.00 ± 1.78, 61.66 ± 1.81, 67.5 ± 1.17 and 68.50 ± 1.05 respectively.

In group I animals there was no significant difference found at 0 hours to 120 hours before and after treatment intervals.

In group II and III animals, lymphocytes showed significant difference at 12 hours after treatment, where it decreased significantly ($P \leq 0.01$) and continued up to 24 hours after treatment compared to value before treatment. In all the groups the values fluctuated within normal physiological range at corresponding intervals.

Comparison between the groups showed significant difference at 12 hours and 24 hours after treatment intervals. The group I showed significant ($P \leq 0.05$) higher value at 12 hours and 24 hours after treatment compared to group II and III at corresponding treatment intervals. There was no significant difference between group II and III at all corresponding intervals.

4.5.5.3 Monocytes (%)

The Mean ± SE., values of monocytes (%) before and after treatment in different groups are given in Table: 29

The Mean ± SE., values of monocytes of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 3.16 ± 0.47, 2.33 ± 0.40, 2.66 ± 0.36, 3.16 ± 0.47 and 3.16 ± 0.47 respectively.

The Mean ± SE., values of monocytes of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 2.83 ± 0.30, 3.33 ± 0.49, 2.66 ± 0.36, 2.33 ± 0.29 and 2.66 ± 0.29 respectively.

The Mean ± SE., values of monocytes of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 3.33 ± 0.55, 2.83 ± 0.47, 3.16 ± 0.60, 2.66 ± 0.36 and 3.00 ± 0.51respectively.

The monocyte count was within normal limits on all the days of observation. There was no significant difference observed in monocytes count within and in between the groups at all corresponding treatment intervals.

4.5.5.4 Basophils (%)

The Mean ± SE., values of basophils (%) before and after treatment in different groups are given in Table: 30

The Mean ± SE., values of basophils of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 0.50 ± 0.10, 0.33 ± 0.06, 0.33 ± 0.06, 0.66 ± 0.21 and 0.16 ± 0.00 respectively.

The Mean ± SE., values of basophils of group II animals at ‘0’ hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 0.33 ± 0.01, 0.33 ± 0.01, 0.50 ± 0.12, 0.33 ± 0.01 and 0.33 ± 0.01 respectively.

The Mean ± SE., values of basophils of group III animals at ‘0’ hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 0.33 ± 0.01, 0.50 ± 0.02, 0.33 ± 0.01, 0.50 ± 0.02 and 0.50 ± 0.02 respectively.

The basophil count was within normal limits on all the days of observation. There was no significant difference observed in basophil count within and in between the groups at all corresponding treatment intervals.

4.5.5.5 Eosinophils (%)

The Mean ± SE., values of eosinophils (%) before and after treatment in different groups are given in Table: 31

The Mean ± SE., values of eosinophils of group I animals at ‘0’ hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 3.50 ± 0.76, 3.16 ± 0.65, 3.50 ± 0.76, 3.50 ± 0.76 and 3.16 ± 0.70 respectively.

The Mean ± SE., values of eosinophils of group II animals at ‘0’ hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 3.00 ± 0.57, 3.16 ± 0.47, 3.16 ± 0.47, 3.16 ± 0.65 and 4.16 ± 0.47 respectively.

The Mean ± SE., values of eosinophils of group III animals at ‘0’ hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 3.83 ± 0.60, 3.83 ± 0.70, 3.66 ± 0.66, 2.66 ± 0.61 and 3.83 ± 0.47 respectively.

The eosinophil count was within normal limits on all the days of observation. There was no significant difference observed in eosinophil count within and in between the groups at all corresponding treatment intervals.

4.6 SERUM BIOCHEMICAL ANALYSIS

4.6.1 Glucose (mg / dL)

The Mean ± SE., values of glucose (mg / dL) before and after treatment in different groups are given in Table: 32 and Fig. 21.

The Mean ± SE., values of glucose group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 100.48 ± 2.96, 88.10 ± 2.91, 74.51± 4.23, 64.55 ± 3.30 and 53.58 ± 1.82 respectively.

The Mean ± SE., values of glucose of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 95.64 ± 4.77, 81.58 ± 3.29, 61.93 ± 3.22, 53.44 ± 2.72 and 48.66 ± 2.35 respectively.

The Mean ± SE., values of glucose of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 94.37 ± 5.74, 78.48 ± 3.20, 66.86 ± 3.10, 58.75 ± 2.91and 53.01 ± 1.03 respectively.

In group I, II and III animals, significant ($P \leq 0.05$) and ($P \leq 0.01$) lower values of glucose observed at 12 hours and at 24 hours respectively compared to before treatment intervals. In all the groups the values were higher before treatment and reached normal physiological range at 72 hours to 120 hours after treatment intervals.

Comparison between the groups showed significant difference at 24 hours and 72 hours after treatment intervals. In group I showed significant ($P \leq 0.05$) higher value of glucose at 24 hours and 72 hours after treatment compared to group II and III at corresponding treatment intervals. In group II significantly ($P \leq 0.05$) lower glucose observed at 24 hours and 72 hours after treatment intervals compared with group III.

4.6.2 Aspartate Amino Transferase (AST, IU/L)

The Mean ± SE., values of AST (IU/L) before and after treatment in different groups are given in Table: 33 and Fig. 22.

The Mean ± SE., values of AST group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 104.84 ± 5.15, 78.71 ± 1.90, 63.96 ± 1.91, 53.45 ± 2.58 and 27.87 ± 2.05 respectively.

The Mean ± SE., values of AST of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 106.53 ± 7.56, 83.60 ± 3.44, 64.53 ± 2.13, 49.50 ± 5.21 and 32.72 ± 1.38 respectively.

The Mean ± SE., values of AST of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 97.73 ± 4.56, 77.00 ± 1.99, 59.73 ± 2.00, 42.03 ± 1.15 and 27.16 ± 1.30 respectively.

In group I, III animals, significant ($P \leq 0.01$) lower values of AST were observed at 12 hours to 120 hours compared to before treatment intervals. In Group II animals, significant ($P \leq 0.05$) lower values of AST was observed at 12 hours compared to before treatment intervals and there after significant ($P \leq 0.01$) lower values of AST were

observed from 24 hours to 120 hours compared to before treatment intervals. In all the groups the glucose values were higher before treatment and reached within the physiological limit at 120 hours after treatment onwards.

Comparison between the groups showed no significant difference at all treatment intervals.

4.6.3 Alanine Amino Transferase (ALT, IU/L)

The Mean ± SE., values of ALT (IU/L) before and after treatment in different groups are given in Table: 34 and Fig. 23.

The Mean ± SE., values of ALT group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 58.16 ± 2.67, 39.83 ± 2.46, 27.83 ± 1.55, 22.5 ± 0.84 and 14.83 ± 1.01 respectively.

The Mean ± SE., values of ALT of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 55.16 ± 2.49, 38.83 ± 1.92, 31.33 ± 1.47, 24.16 ± 0.87 and 18.5 ± 1.70 respectively.

The Mean ± SE., values of ALT of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 58.83 ± 2.27, 39.66 ± 1.66, 30.33 ± 1.17, 24.16 ± 1.44 and 17.16 ± 1.53 respectively.

In group I, II and III animals, significant ($P \leq 0.01$) lower values of ALT were observed at 12 hours to 120 hours compared to before treatment intervals. In all the

groups the ALT values were higher before treatment and reached within the physiological limit at 120 hours after treatment onwards.

Comparison between the groups showed no significant difference at all treatment intervals.

4.7 BLOOD GAS ANALYSIS

4.7.1 Bicorbonate ions (HCO_3^-- mmol/L)

The Mean ± SE., values of bicarbonate ions (mmol / L) before and after treatment in different groups are given in Table: 35 and Fig. 24.

The Mean ± SE., values of bicarbonate ions of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 8.61 ± 0.84, 12.41 ± 0.52, 17.91 ± 0.80, 18.65 ± 0.40 and 19.96 ± 0.38 respectively.

The Mean ± SE., values of bicarbonate ions of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 8.26 ± 0.75, 12.86 ± 0.94, 20.03 ± 0.35, 21.60 ± 0.07 and 21.81 ± 0.03 respectively.

The Mean ± SE., values of bicarbonate ions of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 8.00 ± 0.75, 13.45 ± 0.79, 20.41 ± 0.14, 21.28 ± 0.11 and 21.83 ± 0.03 respectively.

In all the groups the significant lower bicarbonates values were observed at 0 hour before treatment. In group I, II and III animals, significant ($P \leq 0.01$) higher values of bicarbonates were observed at 12 hours to 120 hours after treatment compared to 0 hour

before treatment and reached within the physiological limit at 72 hours after treatment in group II and III whereas in group I at 120 hours after treatment.

Comparison between the groups showed, significant difference at 24 hours 72 hours and 120 hours after treatment intervals. In group I the significant ($P \leq 0.01$) lower value of bicarbonates at 24 hours, 72 hours and 120 hours after treatment compared to group II and III at corresponding intervals. A significant ($P \leq 0.05$) higher value in group II than group III was observed at 72 hours after treatment. However, compared between group II and III no significant difference was seen at 120 hours after treatment interval.

4.7.2 Hydrogen ions (H^+- nmol/L)

The Mean ± SE., values of hydrogen ions (nmol / L) before and after treatment in different groups are given in Table: 36 and Fig. 25.

The Mean ± SE., values of hydrogen ions of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 147.83 ± 7.84, 125.73 ± 20.35, 68.01 ± 9.96, 68.16 ± 9.37and 48.81 ± 3.10 respectively.

The Mean ± SE., values of hydrogen ions of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 143.21 ± 9.61, 98.16 ± 3.62, 55.25 ± 9.43, 58.10 ± 7.33 and 46.16 ± 2.41respectively.

The Mean ± SE., values of hydrogen ions of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 226.85 ± 43.30, 163.95 ± 37.32, 147.38 ± 51.35, 73.31 ± 18.24 and 56.45 ± 6.94 respectively.

In all the groups the significant higher values of hydrogen ions were observed at 0 hour before treatment. In group I the significant ($P \leq 0.01$) lower hydrogen ions was observed at 24 hours after treatment compared to before treatment. In group II the significant ($P \leq 0.01$) lower hydrogen ions was observed at 12 hours after treatment compared to before treatment. In group III the significant ($P \leq 0.01$) lower hydrogen ions was observed at 72 hours after treatment compared to before treatment and reached within the physiological limit at 120 hours after treatment in all the groups.

Comparison among the groups showed no significant difference was observed at any given corresponding intervals.

4.7.3 Venous partial pressure of oxygen (mm of Hg)

The Mean ± SE., values of venous partial pressure of oxygen (mm of Hg) before and after treatment in different groups are given in Table: 37

The Mean ± SE., values of venous partial pressure of oxygen group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 65.03 ± 5.10, 85.16 ± 14.64, 54.33 ± 5.82, 57.91 ± 3.73 and 103.63 ± 25.19 respectively.

The Mean ± SE., values of venous partial pressure of oxygen of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 61.41 ± 5.18, 66.36 ± 9.97, 72.31 ± 12.48, 55.88 ± 3.72 and 76.73 ± 9.91 respectively.

The Mean ± SE., values of venous partial pressure of oxygen of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 82.48 ± 12.72, 77.16 ± 6.37, 60.50 ± 4.35, 54.95 ± 10.04 and 52.15 ± 6.70 respectively.

In all the groups the venous pressure of oxygen values was lower at 0 hour before treatment. In group I the significant ($P \leq 0.01$) higher value was observed at 12 hours, 24 hours, 72 hours and 120 hours after treatment compared to before treatment. In group II the significant ($P \leq 0.01$) higher value was observed at 24 hours, 72 hours and 120 hours after treatment compared to before treatment. In group III the significant ($P \leq 0.01$) higher value was observed at 12 hours, 24 hours, 72 hours and 120 hours after treatment compared to before treatment and reached within the physiological limit at 120 hours after treatment in all the groups.

Comparison between the groups showed no significant difference was observed at any given corresponding intervals.

4.7.4 Venous partial pressure of carbon dioxide (mm of Hg)

The Mean ± SE., values of venous partial pressure of carbon dioxide (mm of Hg) before and after treatment in different groups are given in Table: 38 and Fig. 26.

The Mean ± SE., values of venous partial pressure of carbon dioxide of group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 38.73 ± 0.62, 40.43 ± 0.32, 42.21 ± 0.31, 43.18 ± 0.35 and 43.58 ± 0.24 respectively.

The Mean ± SE., values of venous partial pressure of carbon dioxide of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 38.58 ± 1.00, 40.95 ± 0.99, 42.60 ± 0.29, 43.73 ± 0.29 and 44.26 ± 0.22 respectively.

The Mean ± SE., values of venous partial pressure of carbon dioxide of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 39.25 ± 0.38, 40.98 ± 0.22, 42.36 ± 0.42, 43.26 ± 0.29 and 44.08 ± 0.22 respectively.

In all the groups the venous partial pressures of carbon dioxide values were lower at 0 hour before treatment. In group I and III animals, showed significant ($P \leq 0.01$) higher values of venous partial pressure of carbon dioxide were observed from 12 hours to 120 hours where as in group II from 24 hours to 120 hours after treatment compared to before treatment intervals and reached within the physiological limit at 120 hours after treatment in all the three groups..

Comparison among the groups showed no significant difference was observed at all treatment intervals.

4.7.5 Base deficits

The Mean ± SE., values of base deficit before and after treatment in different groups are given in Table: 39 and Fig. 27.

The Mean ± SE., values of base deficit group I animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 13.38 ± 0.84, 9.58 ± 0.52, 4.08 ± 0.79, 3.35 ± 0.40 and 2.03 ± 0.38 respectively.

The Mean ± SE., values of base deficit of group II animals at '0' hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 13.73 ± 0.75, 9.16 ± 0.94, 1.96 ± 0.35, 0.40 ± 0.07 and 0.18 ± 0.03 respectively.

The Mean ± SE., values of base deficit of group III animals at '0' hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after the treatment were: 14.00 ± 0.75, 8.55 ± 0.79, 1.58 ± 0.14, 0.71± 0.11 and 0.16 ± 0.03 respectively.

In all the groups the base deficit values were higher at 0 hour before treatment. In group I, II and III animals, significant by ($P \leq 0.01$) decreasing trend in base deficit values was observed from 12 hours to 120 hours after treatment compared to before treatment intervals and reached within the physiological limit at 72 hours after treatment in group II and III where as in group I at 120 hours after treatment.

Comparison between the groups showed, significant difference at 24 hours, 72 hours and 120 hours after treatment intervals. In group I the significant ($P \leq 0.05$) higher value of base deficits at 24 hours, 72 hours and 120 hours after treatment were observed compared to group II and III at corresponding intervals. A significant ($P \leq 0.05$) lower value in group II than group III was observed at 72 hours after treatment. However, compared between group II and III no significant difference was seen at 120 hours after treatment interval.

4.7.6 Venous pH

The Mean ± SE., values of venous pH before and after treatment in different groups are given in Table: 40 and Fig. 28.

The Mean ± SE., values of venous pH group I animals at ‘0’ hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after the treatment were: 6.86 ± 0.05, 6.95 ± 0.03, 6.98 ± 0.03, 7.11 ± 0.05 and 7.22 ± 0.03 respectively.

The Mean ± SE., values of venous pH of group II animals at ‘0’ hour before treatment and 12 hours, 24 hours, 72 hours and 120 hours after treatment were: 6.89 ± 0.02, 6.98 ± 0.01, 7.02 ± 0.01, 7.07 ± 0.02 and 7.30 ± 0.02 respectively.

The Mean ± SE., values of venous pH of group III animals at ‘0’ hour before treatment and 12 hours, 24 hours,72 hours and 120 hours after treatment were: 6.76 ± 0.09, 7.13 ± 0.07, 7.12 ± 0.06, 7.16 ± 0.05 and 7.35 ± 0.10 respectively.

In all the groups the venous pH values were lower 0 hour before treatment. In group I the significant ($P \leq 0.01$) higher venous pH was observed at 72 hours after treatment compared to before treatment. In group II the significant ($P \leq 0.01$) higher venous pH was observed at 12 hours after treatment compared to before treatment and there after continued up to 120 hours after treatment. In group III the significant ($P \leq 0.05$) higher venous pH was observed at 12 hours after treatment compared to before treatment and there after significant ($P \leq 0.01$) higher venous pH was from 24 hours to 120 hours after treatment than before treatment interval and reached within the physiological limit at 120 hours after treatment in all the groups.

Comparison between the groups showed significant ($P \leq 0.05$) higher values of venous pH in group II and III compared to group I at 120 hours after treatment. In group

II the venous pH showed significant ($P \leq 0.05$) lower values at 120 hours after treatment compared to group III at given corresponding intervals.

4.8 Therapy

4.8.1 Conservative medicinal approach

In group I six acute acidotic goats received intravenous isotonic sodium bicarbonate as alkalizing agent as per base deficit and the rumen buffer (Bufzone® from the Intas Pharmaceuticals Ltd. Ahmedabad) 50 g / day per oral for five days (unique rumen buffer enriched with yeast and metabolic boosters) as buffering agent. All the six cases of ruminal acidosis recovered without any complications. However, the recovery was very slow compared to other two groups as shown in plates no. 29, 30, 31, 32 and 33.

4.8.2 Surgical approach: Rumenotomy

The acute ruminal acidotic goats of group II and III were subjected for the clinical examination and decided to treat with surgery i.e. rumenotomy along with medicinal therapy to treat the condition.

In group II six goats with acute acidosis were subjected to rumenotomy and received the intravenous alkalizing agent as isotonic sodium bicarbonate as per base deficit and probiotics (Ecotas® from the Intas Pharmaceuticals Ltd., Ahmadabad) 1 bolus bid orally for five days were used as probiotic bolus to restore ruminal microflora.

In group III six goats with acute acidosis were operated for rumenotomy and received the intravenous alkalizing agent as isotonic sodium bicarbonate as per base

deficit and fresh rumen fluid was given @ 10 ml / kg per orally for two days to restore ruminal microflora.

4.8.2.1 Pre-operative Considerations

As there was an emergency situation with holding of food and water pre-operatively was not practicable in this study. All the twelve goats of group II and III had undergone similar procedure of pre- operative preparation.

4.8.2.2 Preparation of Patient

The aseptic preparation of the surgical site on left flank with antiseptic chlorhexidine and povidone-iodine solution before surgery was beneficial in controlling infection during and after post-operative surgery. Inj. streptopencillin @ 10 mg / kg body weight was administered intramuscular prior to surgery as pre-operative antibiotic coverage.

4.8.2.3 Anaesthetic Technique

In both the groups of II and III animals, surgery was performed under regional block using local linear infiltration and inverted “L” block in the left flank region of the animal. The 2% lignocaine hydrochloride® was used as local anesthetic agent. As goats were sensitive to lignocaine the 1% was prepared from 2% by mixing equal volume of drug and distil water as V/V (1:1). The anaesthetic procedure performed in this study was excellent. There was no anaesthetic emergency during the surgical procedure or lack of analgesia in either of the group II and III.

4.8.2.4 Surgical Procedure

In both the groups of II and III, the standard laparotomy and rumenotomy was performed in sternal position. Then intra-operatively removal of acidotic liquid contents along with other undigested food materials of the rumen, lavaging of rumen with normal saline, replacement of mixture containing (100g of presoaked soft dried chopped fodder, 50g joggery and 5g of sodium bicarbonates in 500ml of water) into the rumen, precaution during surgery for prevention of abdominal contamination with ruminal contents and proper suturing of the rumen and abdominal muscles with suture material were effective in speedy recovery from the surgery of acute ruminal acidosis in goats. All the six cases of each group II and III with ruminal acidosis were recovered without any complications. The surgical procedure followed in both the group of II and III were shown in the plates no.34, 35, 40 and 41.

4.8.2.5 Post-operative Care

Surgical wound dressing was carried out for each animal of group II and III by using five percent povidone iodine solution and Loraxene® ointment till the healing of the surgical wound. Inj. streptopencillin (10 mg / kg body weight, intramuscular, once a day for seven days) and Inj. tolfenamic acid (2 mg / kg body weight, intramuscular, once daily for three days) were given. The animals of the both the groups had excellent recovery from the surgery. In two cases of group III had wound dehiscence at the surgical site and it was managed by open wound dressing with antiseptic povidone iodine solution till healing was observed. After complete healing of laparotomy wound, sutures were removed on 12^{th} day of after operation as shown in plate (39 and 46). The resumption of

feed intake was more active in group III than the group II animals as shown in plates no. 36, 37, 38, 42, 43, 44 and 45.

4.8.2.6 Common supportive treatments for all affected animals of each group

The common therapy for all the animals of irrespective of group helped in faster recovery from the acidotic condition. In group I animals Inj. streptopencillin @ 10 mg / kg body weight intraruminal every 12 hours for 3 days was given to stop the over fermentation and restriction of the growth of gram positive organisms. The early resumption of feed and water intake by all the animals was aided by giving Inj. thiamine hydrochloride @ 3 ml / animal by intramuscular route once a day for 3 days, Inj. chlorphenaramine maleate @ 2 ml / animal by intramuscular route once a day for 3 days, and Inj. calboral (calcium borogluconate) 20 ml / goat by intravenous route once in two days for 3 days.

In order to know the impact of ruminal acidosis in affected goats the mean ± SE values of fourteen important parameters were compared between the acidotic goats. The details are enumerated in Table: 41 and 42. In group I out of fourteen parameters four parameters (rumen liquor pH, TVFA, MBRT and gram positive organism) significantly ($P \leq 0.05$) differs from other two groups II and III. Whereas, no significant difference was observed between group II and III at all the fourteen parameters. This shows the values of fourteen parameters of group I, II and III were improved compared to 0 hour interval before treatment and the much better improvement was observed at 12 hours interval after treatment in group II and III as compared to group I. The values of important parameters of different groups of goats at 72 hours intervals after treatment

were compared. In group I out of fourteen parameters eight parameters (rumen liquor pH, TVFA, MBRT, gram positive organism, PCV, HCO^{-}_{3}, Base deficit and glucose) significantly ($P \leq 0.05$) differs from other two groups II and III. The significant ($P \leq 0.05$) difference was observed in four parameters (MBRT, PCV, Base deficit and glucose) out of fourteen compared between group II and III. This shows the values of fourteen parameters of group I, II and III were improved compared to 0 hour interval before treatment, 12 hours after treatment interval and the much better improvement was observed at 72 hours interval after treatment in group II and III as compared to group I.

Among three groups, in group III the recovery in the form of normalcy attained in physical, heamatobiochemical and ruminal fluid parameters was earlier compared to group I and II as shown in table 43. Hence it can be concluded that therapeutic protocol used in group III consisting of rumenotomy, isotonic sodium bicarbonate intravenous as alkalizing agent along with cud transplantation can be used as preferred therapy for the acute ruminal acidosis for early recovery. Alternatively group II treatment protocol consisting of rumenotomy, isotonic sodium bicarbonate intravenous as alkalizing agent along with Ecotas® (oral) can be used. However, group I treatment protocol consisting of Sodium bicarbonate as alkalizing agent, intra-ruminal antibiotic and Bufzone®, rumen buffering agent can also be used for the treatment of ruminal acidosis in goats at dispensary level where surgical intervention not feasible.

Table 2. Prevalence of ruminal acidosis in goats at five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Sl. No.	Year	Total Clinical cases presented	Total goat cases presented	Total goat cases of digestive disorders	Ruminal acidosis in goats	Male	Female
1	2011	33814	14644	5463	228	30	198
2	2012	32374	13428	4787	176	20	156
3	2013	29763	9554	3361	177	19	158
4	2014	31939	10021	4152	148	14	134
5	2015	32490	9269	3882	211	20	191
6	2016	32837	9751	4048	131	14	117
Total		**193217**	**66667**	**25693**	**1071**	**117**	**954**
Percentage		**0.55**	**1.60**	**4.16**		**10.92**	**89.08**

Table 3. Hospital wise prevalence of ruminal acidosis in goats at five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Bidar for a period of five years (2011-2016)

Sl. No	Name of the Veterinary Hospital	Total Clinical cases presented	Total goat cases presented	Total goat cases of digestive disorders	Ruminal acidosis in goats	Male	Female
1	Veterinary Hospital, Aurad	17599	4097	1204	50	00	50
2	Veterinary Hospital, Basavakalyan	45956	18995	10235	166	07	159
3	Veterinary Hospital, Bhalki	24357	4867	2484	37	01	36
4	Veterinary Hospital, Bidar	43493	18325	6554	434	32	402
5	Veterinary Hospital, Humnabad	27927	11554	2900	80	09	71
6	Veterinary Clinical Complex, Veterinary College, Bidar	9626	2611	1159	130	24	106
7	APMC Veterinary Hospital (Peripheral), Bidar	24259	6218	1157	174	44	130
	Total	**193217**	**66667**	**25693**	**1071**	**117**	**954**
	Percentage	**0.55**	**1.60**	**4.16**		**10.92**	**89.08**

Table 4. Prevalence of ruminal acidosis in goats at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Sl. No.	Year	Total Clinical cases presented	Total goat cases presented	Total goat cases of digestive disorders	Ruminal acidosis in goats	Male	Female
1	2011	5771	1891	559	34	09	25
2	2012	6033	1708	420	55	12	43
3	2013	6361	1665	369	50	13	37
4	2014	5505	1450	399	55	10	45
5	2015	5286	1190	332	51	12	39
6	2016	4929	925	237	59	12	47
Total		**33885**	**8829**	**2316**	**304**	**68**	**236**
Percentage		**0.89**	**3.44**	**13.12**		**22.37**	**77.63**

Table 5. Prevalence of ruminal acidosis in goats with respect to age at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Sl. No.	Name of the Hospital	≤ 1 year age	1-2 year age	≥ 2 years age	Total
1	Veterinary Clinical Complex, Veterinary College, Bidar	39	58	33	130
2	APMC Peripheral Veterinary hospital, Bidar	67	70	37	174
	Total	**106**	**128**	**70**	**304**
	Percentage	**34.87**	**42.10**	**23.03**	**100**

Table 6. Prevalence of ruminal acidosis in goats with respect to sex at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Sl. No.	Name of the Hospital	Male	Female	Total
1	Veterinary Clinical Complex, Veterinary College, Bidar	24	106	130
2	APMC Peripheral Veterinary hospital, Bidar	44	130	174
	Total	**68**	**236**	**304**
	Percentage	**22.37**	**77.63**	**100**

Table 7. Prevalence of ruminal acidosis in goats with respect to source of carbohydrate at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

Sl. No.	Name of the Hospital	Rice	Chapati or Wheat flour	Jawar or flour	Vegetables	Grains	Maize	Fruits	Not Known	Total
1	Veterinary Clinical Complex, Veterinary College, Bidar	34	17	12	02	03	03	01	58	130
2	APMC Peripheral Veterinary hospital, Bidar	28	15	09	04	01	01	00	116	174
	Total	**62**	**32**	**21**	**06**	**04**	**04**	**01**	**174**	**304**
	Percentage	**20.39**	**10.53**	**6.91**	**1.97**	**1.32**	**1.32**	**0.33**	**57.23**	**100**

Table 8. Prevalence of ruminal acidosis in goats with respect to season[**] at Veterinary Clinical Complex, Bidar for a period of last years (2011-2016)

Sl. No.	Name of the Hospital	Summer (March, April and May)	Monsoon (June, July, August and September)	Post Monsoon (October, November and December)	Winter (January and February)	Total
1	Veterinary Clinical Complex, Veterinary College, Bidar	42	48	22	18	130
2	APMC Peripheral Veterinary hospital, Bidar	37	63	42	32	174
	Total	**79**	**111**	**64**	**50**	**304**
	Percentage	**25.99**	**36.51**	**21.05**	**16.45**	**100**

[**]As per the Indian Meteorological Department of Bengaluru (Karnataka), the state is divided into four seasons in a year *viz*., summer (March, April and May), monsoon (June, July August and September), post monsoon (October, November and December) and winter (January and February).

Table 9: Mean ± SE., values of rectal temperature (^{0}F) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	101.33 ±0.35	102.48 ± 0.45	102.13 ± 0.26
12	101.80 ± 0.28^a	100.26 ± 0.25^b**	100.65 ± 0.23^b**
24	101.95 ± 0.32	101.38 ± 0.16*	101.71 ± 0.42
72	101.81 ± 0.23	101.98 ± 0.14	102.11 ± 0.46
120	101.91 ± 0.30^a	102.76 ± 0.12^b	101.98 ± 0.27^a

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

$^{a\,b}$ Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 10: Mean ± SE., values of heart rate (beats/minute) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	108.00 ± 4.89	110.00 ± 6.17	116.33 ± 3.55
12	103.33 ± 4.30	102.83 ± 4.69	102.33 ± 4.85*
24	98.16 ± 3.57	99.50 ± 3.61	100.66 ± 6.56
72	89.83 ± 3.35*	92.16 ± 2.99*	90.5 ± 1.25**
120	76.33 ± 2.15**	82.5 ± 3.07**	79.83 ± 1.97**

Table 11: Mean ± SE., values of respiratory rate (breaths/minute) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	44.50 ± 4.68	45.83 ± 2.94	43.33 ± 2.40
12	38.83 ± 2.71	39.33 ± 3.44	33.66 ± 4.99
24	35.50 ± 2.36	30.66 ± 2.67**	31.83 ± 3.18**
72	34.00 ± 1.03[a]	27.5 ± 1.74[b] **	25.83 ± 1.86[b]**
120	29.00 ± 0.85**	29.83 ± 1.60**	28.33 ± 0.98**

Table 12: Rumen contractions (in minutes) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	Nil	Nil	Nil
72	1.75 ± 0.11[a]**	2.08 ± 0.27[ac]**	2.75 ± 0.11[b]**
120	2.25 ± 0.11[a]**	2.50 ± 0.22[b]**	2.91 ± 0.08[b]**

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

[a b] Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 13: Colour of ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	Milky white	Milky white	Milky white
12	Milky white	Light green	Light green
24	Milky white	Light green	Normal Green
72	Light green	Light green	Normal Green
120	Normal Green	Normal Green	Normal Green

Table 14: Odour of ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	Sour	Sour	Sour
12	Sour	Mild aromatic	Mild aromatic
24	Sour	Mild aromatic	Moderate aromatic
72	Mild aromatic	Mild aromatic	Moderate aromatic
120	Moderate aromatic	Moderate aromatic	Normal aromatic

Table 15: Consistency of ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	Watery	Watery	Watery
12	Watery	Watery	Watery
24	Watery	Watery	Mild viscous
72	Mild viscous	Mild viscous	Moderate viscous
120	Moderate viscous	Moderate viscous	Normal viscous

Table 16: Mean ± SE., values of ruminal fluid pH in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	4.78 ± 0.25	4.68 ± 0.19	4.70 ± 0.26
12	5.63 ± 0.05[a]**	6.53 ± 0.09[b]**	6.60 ± 0.06[b]**
24	6.00 ± 0.07[a]**	6.81 ± 0.05[b]**	6.81 ± 0.06[b]**
72	6.4 ± 0.05[a]**	6.76 ± 0.08[b]**	6.78 ± 0.09[b]**
120	6.81 ± 0.07**	6.88 ± 0.06**	6.90 ± 0.06**

Table 17: Mean ± SE., Gram positive bacteria count (%) in ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	81.84 ± 1.64	82.83 ± 1.49	81.83 ± 1.53
12	60.16 ± 2.03[a]**	29.33 ± 1.45[b]**	30.66 ±1.70[b]**
24	48.50 ± 1.43[a]**	20.66 ± 0.88[b]**	21.50 ± 1.33[b]**
72	30.50 ± 1.66[a]**	19.66 ± 0.80[b]**	22.00 ± 0.85[b]**
120	22.66 ± 0.66**	22.33 ± 0.66**	21.83 ± 1.16**

Table 18: Mean ± SE., Gram negative bacteria count (%) in ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	18.16 ± 1.64	17.16 ± 1.49	18.16 ± 1.53
12	39.83 ± 2.03[a]**	70.66 ± 1.45[b]**	69.33 ± 1.70[b]**
24	53.16 ± 2.27[a]**	79.33 ± 0.88[b]**	78.5 ± 1.33[b]**
72	69.50 ± 1.66[a]**	80.33 ± 0.80[b]**	78.00 ± 0.85[b]**
120	77.33 ± 0.66**	77.66 ± 0.66**	78.16 ± 1.16**

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

[a b] Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 19: Protozoal density of ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	Nil	Nil	Nil
12	Nil	+	+
24	Nil	+	++
72	+	++	+++
120	++	++	+++

Table 20: Mean ± SE., Methylene blue reduction time (in minutes) of ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	64.61 ± 1.07	63.72 ± 0.94	66.40 ± 0.98
12	52.22 ± 0.73^{a}**	28.68 ± 2.06^{b}**	27.76 ± 1.09^{b}**
24	37.99 ± 2.25^{a}**	21.28 ± 0.88^{b}**	18.80 ± 1.01^{b}**
72	26.95 ± 1.11^{a}**	13.70 ± 0.99^{b}**	10.95 ± 0.39^{c}**
120	10.34 ± 0.24^{a}**	9.99 ± 0.34^{ab}**	9.12 ± 0.32^{b}**

Table 21: Mean ± SE., Sedimentation activity time (in minutes) of ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	Absent	Absent	Absent
12	Absent	Absent	Absent
24	Absent	Absent	Absent
72	22.22 ± 0.95^{a}**	15.61 ± 0.51^{b}**	11.75 ± 0.60^{c}**
120	10.80 ± 0.19**	11.12 ± 0.31**	10.76 ± 0.23**

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

a b Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 22: Mean ± SE., Total volatile fatty acids (mmol/dL) of ruminal fluid in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	66.4 ± 1.73	68.60 ± 1.48	70.87 ± 1.59
12	62.81 ± 2.03^{a}	8.38 ± 0.41^{b}**	8.93 ± 0.34^{b}**
24	50.38 ± 0.92^{a}**	12.37 ± 0.33^{b}**	17.90 ± 0.57^{c}**
72	40.70 ± 2.25^{a}**	16.89 ± 0.72^{b}**	31.22 ± 1.32^{c}**
120	24.61 ± 1.74**	25.20 ± 1.70**	23.26 ± 0.85**

Table 23: Mean ± SE., values of Total erythrocyte count (×10^{6}/µL) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	12.21± 0.19	12.48 ± 0.32	12.81± 0.30
12	10.81± 0.15**	11.15 ± 0.18**	11.35 ± 0.23**
24	10.23 ± 0.21**	10.13 ± 0.19**	10.68 ± 0.26**
72	9.53 ± 0.30**	9.26 ± 0.19**	9.55 ± 0.27**
120	8.73 ± 0.20**	8.65 ± 0.15**	8.66 ± 0.23**

Table 24: Mean ± SE., values of Total leucocyte count (×10^{3}/µL) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	13.19 ± 0.47	13.41 ± 0.26	13.73 ± 0.19
12	13.20 ± 0.48	12.70 ± 0.29	12.73 ± 0.23**
24	12.51 ± 0.43^{a}	11.21 ± 0.27^{b}**	11.15 ± 0.32^{b}**
72	10.98 ± 0.32^{a}**	10.38 ± 0.21^{a}**	10.00 ± 0.14^{b}**
120	10.48 ± 0.28**	9.88 ± 0.13**	9.85 ± 0.16**

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

$^{a\,b}$ Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 25: Mean ± SE., values of Haemoglobin (g/dL) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	10.85 ± 0.18	11.4 ± 0.26	11.11 ± 0.31
12	9.91 ± 0.16**	10.58 ± 0.30	10.00 ± 0.30*
24	10.36 ± 0.19	10.21 ± 0.23**	10.13 ± 0.20*
72	10.16 ± 0.20*	9.75 ± 0.14**	9.53 ± 0.19**
120	9.93 ± 0.13[a]**	9.26 ± 0.13[b]**	8.96 ± 0.20[b]**

Table 26: Mean ± SE., values of Packed Cell Volume (%) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	34.81± 0.83[a]	36.88 ± 0.90[a]	38.65 ± 0.64[b]
12	30.63 ± 0.45**	32.33 ± 0.67**	32.73 ± 0.88**
24	31.00 ± 0.59**	30.96 ± 0.57**	30.55 ± 0.86**
72	29.48 ± 0.55[a]**	28.28 ± 0.74[a]**	27.86 ± 0.43[b]**
120	26.23 ± 0.42**	26.15 ± 0.38**	25.65 ± 0.56**

Table 27: Mean ± SE., values of Neutrophils (%) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	24.00 ± 0.99	24.66 ± 1.11	23.33 ± 0.98
12	26.66 ± 1.64[a]	32.33 ± 0.95[b]**	34.83 ± 1.53[b]**
24	25.50 ± 1.20[a]	31.16 ± 0.83[b]**	31.66 ± 1.45[b]**
72	23.66 ± 0.55[a]	27.16 ± 0.60[b]	26.16 ± 1.13[a]
120	23.83 ± 0.47	23.33 ± 1.08	24.16 ± 0.74

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

[a b] Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 28: Mean ± SE., values of Lymphocytes (%) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	69.16 ± 0.60	69.00 ± 0.57	69.16 ± 0.60
12	67.5 ± 1.30[a]	60.83 ± 1.35[b]**	58.00 ± 1.78[b]**
24	68.00 ± 1.15[a]	62.5 ± 1.60[b]**	61.66 ± 1.81[b]**
72	69.00 ± 0.81	67.00 ± 0.96	67.50 ± 1.17
120	69.66 ± 0.61	69.50 ± 0.56	68.50 ± 1.05

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

[a b] Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 29: Mean ± SE., values of Monocytes (%) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	3.16 ± 0.47	2.83 ± 0.30	3.33 ± 0.55
12	2.33 ± 0.40	3.33 ± 0.49	2.83 ± 0.47
24	2.66 ± 0.36	2.66 ± 0.36	3.16 ± 0.60
72	3.16 ± 0.47	2.33 ± 0.29	2.66 ± 0.36
120	3.16 ± 0.47	2.66 ± 0.29	3.00 ± 0.51

Table 30: Mean ± SE., values of Basophils (%) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	0.50 ± 0.10	0.33 ± 0.01	0.33 ± 0.01
12	0.33 ± 0.06	0.33 ± 0.01	0.50 ± 0.02
24	0.33 ± 0.06	0.50 ± 0.12	0.33 ± 0.01
72	0.66 ± 0.21	0.33 ± 0.01	0.50 ± 0.02
120	0.16 ± 0.00	0.33 ± 0.01	0.50 ± 0.02

Table 31: Mean ± SE., values of Eosinophils (%) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	3.50 ± 0.76	3.00 ± 0.57	3.83 ± 0.60
12	3.16 ± 0.65	3.16 ± 0.47	3.83 ± 0.70
24	3.50 ± 0.76	3.16 ± 0.47	3.66 ± 0.66
72	3.50 ± 0.76	3.16 ± 0.65	2.66 ± 0.61
120	3.16 ± 0.70	4.16 ± 0.47	3.83 ± 0.47

Table 32: Mean ± SE., values of Glucose (mg/dL) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	100.48 ± 2.96	95.64 ± 4.77	94.37 ± 5.74
12	88.10 ± 2.91*	81.58 ± 3.29*	78.48 ± 3.20*
24	74.51 ± 4.23^{a}**	61.93 ± 3.22^{b}**	66.86 ± 3.10^{a}**
72	64.55 ± 3.30^{a}**	53.58 ± 1.82**	58.75 ± 2.91^{a}**
120	53.58 ± 1.82**	48.66 ± 2.35**	53.01 ± 1.03**

Table 33: Mean ± SE., values of AST (IU/L) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	104.84 ± 5.15	106.53 ± 7.56	97.73 ± 4.56
12	78.71 ± 1.90**	83.60 ± 3.44*	77.00 ± 1.99**
24	63.96 ± 1.91**	64.53 ± 2.13**	59.73 ± 2.00**
72	53.45 ± 2.58**	49.50 ± 5.21**	42.03 ± 1.15**
120	27.87 ± 2.05**	32.72 ± 1.38**	27.16 ± 1.30**

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

[a b] Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 34: Mean ± SE., values of ALT (IU/L) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	58.16 ± 2.67	55.16 ± 2.49	58.83 ± 2.27
12	39.83 ± 2.46**	38.83 ± 1.92**	39.66 ± 1.66**
24	27.83 ± 1.55**	31.33 ± 1.47**	30.33 ± 1.17**
72	22.5 ± 0.84**	24.16 ± 0.87**	24.16 ± 1.44**
120	14.83 ± 1.01**	18.5 ± 1.70**	17.16 ± 1.53**

Table 35: Mean ± SE., values of Bicarbonate ions (HCO_3^- - mmol/L) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	8.61 ± 0.84	8.26 ± 0.75	8.00 ± 0.75
12	12.41 ± 0.52**	12.83 ± 0.94**	13.45 ± 0.79**
24	17.91 ± 0.79^{a}**	20.03 ± 0.35^{b}**	20.41 ± 0.14^{b}**
72	18.65 ± 0.40^{a}**	21.60 ± 0.07^{b}**	21.28 ± 0.11^{c}**
120	19.96 ± 0.38^{a}**	21.81 ± 0.03^{b}**	21.83 ± 0.03^{b}**

Table 36: Mean ± SE., values of Hydrogen ions (H^+- nmol/L) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	147.83 ± 7.84	143.21 ± 9.61	226.85 ± 43.30
12	125.73 ± 20.35	98.16 ± 3.62**	163.95 ± 37.32
24	68.01 ± 9.96**	55.25 ± 9.43**	147.38 ± 51.35
72	68.16 ± 9.37**	58.10 ± 7.33**	73.31 ± 18.24**
120	48.81 ± 3.10**	46.16 ± 2.41**	56.45 ± 6.94**

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

a b Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 37: Mean ± SE., values of VpO_2 (mm of Hg) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	65.03 ± 5.10	61.41 ± 5.18	82.48 ± 12.72
12	85.16 ± 14.64	66.36 ± 9.97	77.16 ± 6.37
24	54.33 ± 5.82	72.31 ± 12.48	60.50 ± 4.35
72	57.91 ± 3.73	55.88 ± 3.72	54.95 ± 10.04
120	103.63 ± 25.19	76.73 ± 9.91	52.15 ± 6.70

Table 38: Mean ± SE., values of $VpCO_2$ (mm of Hg) in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	38.73 ± 0.62	38.58 ± 1.00	39.25 ± 0.38
12	40.43 ± 0.32**	40.95 ± 0.99	40.98 ± 0.22**
24	42.21 ± 0.31**	42.60 ± 0.29**	42.36 ± 0.42**
72	43.18 ± 0.35**	43.73 ± 0.29**	43.26 ± 0.29**
120	43.58 ± 0.24**	44.26 ± 0.22**	44.08 ± 0.22**

Table 39: Mean ± SE., values of base deficits in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	13.38 ± 0.84	13.73 ± 0.75	14.00 ± 0.75
12	9.58 ± 0.52**	9.16 ± 0.94**	8.55 ± 0.79**
24	4.08 ± 0.79^{a}**	1.96 ± 0.35^{b}**	1.58 ± 0.14^{b}**
72	3.35 ± 0.40^{a}**	0.40 ± 0.07^{b}**	0.71 ± 0.11^{c}**
120	2.03 ± 0.38^{a}**	0.18 ± 0.03^{b}**	0.16 ± 0.03^{b}**

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

a b Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 40: Mean ± SE., values of venous pH in different groups of goats at different intervals

Groups / Hours	Group I	Group II	Group III
0	6.86 ± 0.05	6.89 ± 0.02	6.76 ± 0.09
12	6.95 ± 0.03	6.98 ± 0.01**	7.13 ± 0.07*
24	6.98 ± 0.03	7.02 ± 0.01**	7.12 ± 0.06**
72	7.11± 0.05**	7.07 ± 0.02**	7.16 ± 0.05**
120	7.22 ± 0.03[a]**	7.30 ± 0.02[ac]**	7.35 ± 0.10[b]**

* Means bearing superscript differ significantly at 5 % level (P ≤ 0.05) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level (P ≤ 0.01) from '0' hr interval within the Group

[a b] Means bearing superscript differ significantly at 5 % level (P ≤ 0.05) between the groups at corresponding intervals

Table 41: Mean ± SE., values of important parameters of different groups of goats at 12 hour intervals

Sl. No.	Parameter	Group I	Group II	Group III
1	Heart rate	103.33 ± 4.30	102.83 ± 4.69	102.33 ± 4.85*
2	RL pH	5.63 ± 0.05^{a}**	6.53 ± 0.09^{b}**	6.60 ± 0.06^{b}**
3	TVFA	62.81 ± 2.03^{a}	8.38 ± 0.41^{b}**	8.93 ± 0.34^{b}**
4	MBRT	52.22 ± 0.73^{a}**	28.68 ± 2.06^{b}**	27.76 ± 1.09^{b}**
5	Gram +ve	60.16 ± 2.03^{a}**	29.33 ± 1.45^{b}**	30.66 ± 1.70^{b}**
6	Hb	9.91 ± 0.16**	10.58 ± 0.30	10.00 ± 0.30*
7	PCV	30.63 ± 0.45**	32.33 ± 0.67**	32.73 ± 0.88**
8	HCO^{-}_{3}	12.41 ± 0.52**	12.83 ± 0.94**	13.45 ± 0.79**
9	VpH	6.95 ± 0.03	6.98 ± 0.01**	7.13 ± 0.07*
10	Base deficit	9.58 ± 0.52**	9.16 ± 0.94**	8.55 ± 0.79**
11	$VpCO_2$	40.43 ± 0.32**	40.95 ± 0.99	40.98 ± 0.22**
12	H^{+}	125.73 ± 20.35	98.16 ± 3.62**	163.95 ± 37.32
13	Glucose	88.10 ± 2.91*	81.58 ± 3.29*	78.48 ± 3.20*
14	AST	78.71 ± 1.90**	83.60 ± 3.44*	77.00 ± 1.99**

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

a b Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 42: Mean ± SE., values of important parameters of different groups of goats at 72 hour intervals

Sl. No.	Parameter	Group I	Group II	Group III
1	Heart rate	89.83 ± 3.35*	92.16 ± 2.99*	90.5 ± 1.25**
2	RL pH	6.40 ± 0.05^{a}**	6.76 ± 0.08^{b}**	6.78 ± 0.09^{b}**
3	TVFA	40.70 ± 2.25^{a}**	16.89 ± 0.72^{b}**	31.22 ± 1.32^{b}**
4	MBRT	26.95 ± 1.11^{a}**	13.70 ± 0.99^{b}**	10.95 ± 0.39^{c}**
5	Gram +ve	30.50 ± 1.66^{a}**	19.66 ± 0.80^{b}**	22.00 ± 0.85^{b}**
6	Hb	10.16 ± 0.20*	9.75 ± 0.14**	9.53 ± 0.19**
7	PCV	29.48 ± 0.55^{a}**	28.28 ± 0.74^{a}**	27.86 ± 0.43^{b}**
8	HCO^{-}_{3}	17.91 ± 0.79^{a}**	20.03 ± 0.35^{b}**	20.41 ± 0.14^{b}**
9	VpH	7.11 ± 0.05**	7.07 ± 0.02**	7.16 ± 0.05**
10	Base deficit	3.35 ± 0.40^{a}**	0.40 ± 0.07^{b}**	0.71 ± 0.11^{c}**
11	$VpCO_2$	43.18 ± 0.35**	43.75 ± 0.29**	43.26 ± 0.29**
12	H^{+}	125.73 ± 20.35	98.16 ± 3.62**	163.95 ± 37.32
13	Glucose	64.55 ± 3.30^{a}**	53.44 ± 2.72^{b}**	58.75 ± 2.91^{a}**
14	AST	53.45 ± 2.58**	49.50 ± 5.21**	42.03 ± 1.15**

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

$^{a\,b}$ Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Table 43: Comparison of mean recovery time after treatment in different groups of acute ruminal acidosis in goats

Sl. No.	Parameter	Group I		Group II		Group III	
		Values	Time	Values	Time	Values	Time
1	Heart rate	76.33 ± 2.15**	**1**120	82.5 ± 3.07**	120	79.83 ± 1.97**	120
2	RL pH	6.4 ± 0.05^{a}**	72	6.53 ± 0.09^{b}**	12	6.60 ± 0.06^{b}**	12
3	TVFA	24.61 ± 1.74**	120	12.37 ± 0.33^{b}**	24	17.90 ± 0.57^{c}**	24
4	MBRT	10.34 ± 0.24^{a}**	120	9.99 ± 0.34^{ab}**	120	10.95 ± 0.39^{c}**	72
5	Gram +ve	22.66 ± 0.66**	120	20.66 ± 0.88^{b}**	24	21.50 ± 1.33^{b}**	24
6	Hb	9.93 ± 0.13^{a}**	120	9.26 ± 0.13^{b}**	120	9.53 ± 0.19**	72
7	PCV	26.23 ± 0.42**	120	28.28 ± 0.74^{a}**	72	27.86 ± 0.43^{b}**	72
8	HCO^{-}_{3}	19.96 ± 0.38^{a}**	120	20.03 ± 0.35^{b}**	24	20.41 ± 0.14^{b}**	24
9	VpH	7.22 ± 0.03^{a}**	120	7.30 ± 0.02^{ac}**	120	7.35 ± 0.10^{b}**	120
10	Base deficit	3.35 ± 0.40^{a}**	72	1.96 ± 0.35^{b}**	24	1.58 ± 0.14^{b}**	24
11	$VpCO_2$	43.58 ± 0.24**	120	44.26 ± 0.22**	120	44.08 ± 0.22**	120
12	H^{+}	48.81 ± 3.10**	120	46.16 ± 2.41**	120	56.45 ± 6.94**	120
13	Glucose	53.58 ± 1.82**	120	53.58 ± 1.82**	72	53.01 ± 1.03**	120
14	AST	27.87 ± 2.05**	120	32.72 ± 1.38**	120	27.16 ± 1.30**	120
Mean recovery time in hours			**113.14 ± 4.65**		**78.00 ± 12.50**		**74.57 ± 12.07**

* Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) from '0' hr interval within the Group

** Means bearing superscript differ significantly at 1 % level ($P \leq 0.01$) from '0' hr interval within the Group

$^{a\,b}$ Means bearing superscript differ significantly at 5 % level ($P \leq 0.05$) between the groups at corresponding intervals

Fig. 1: Prevalence of ruminal acidosis in goats at five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

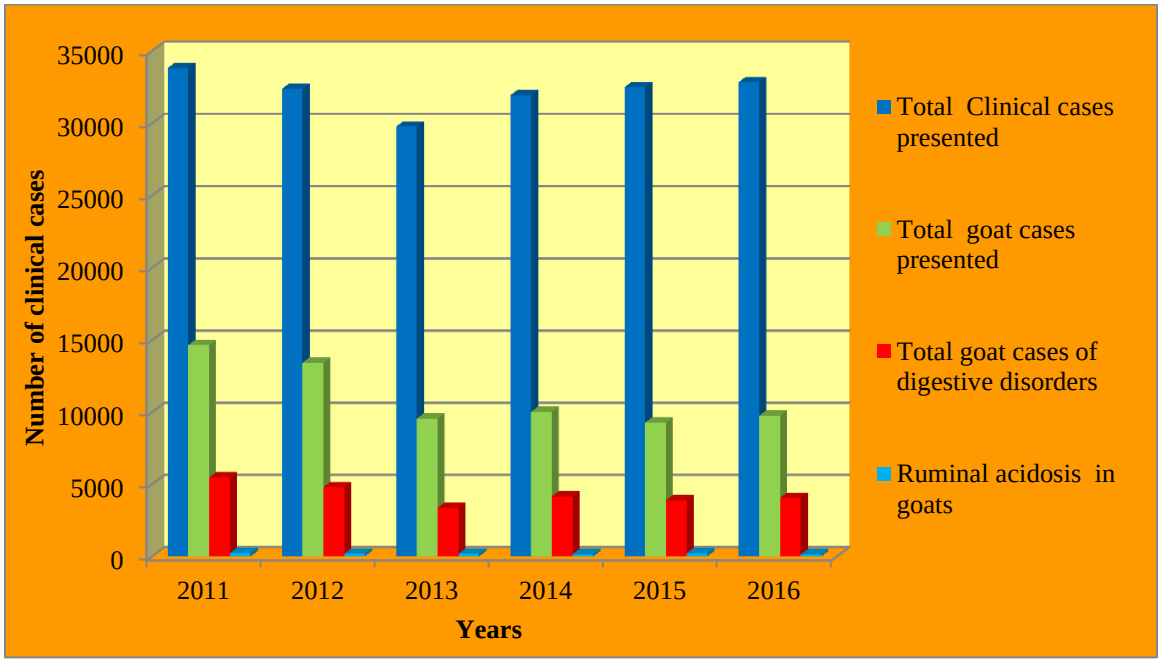


Fig. 2: Hospital wise prevalence of ruminal acidosis in goats at five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

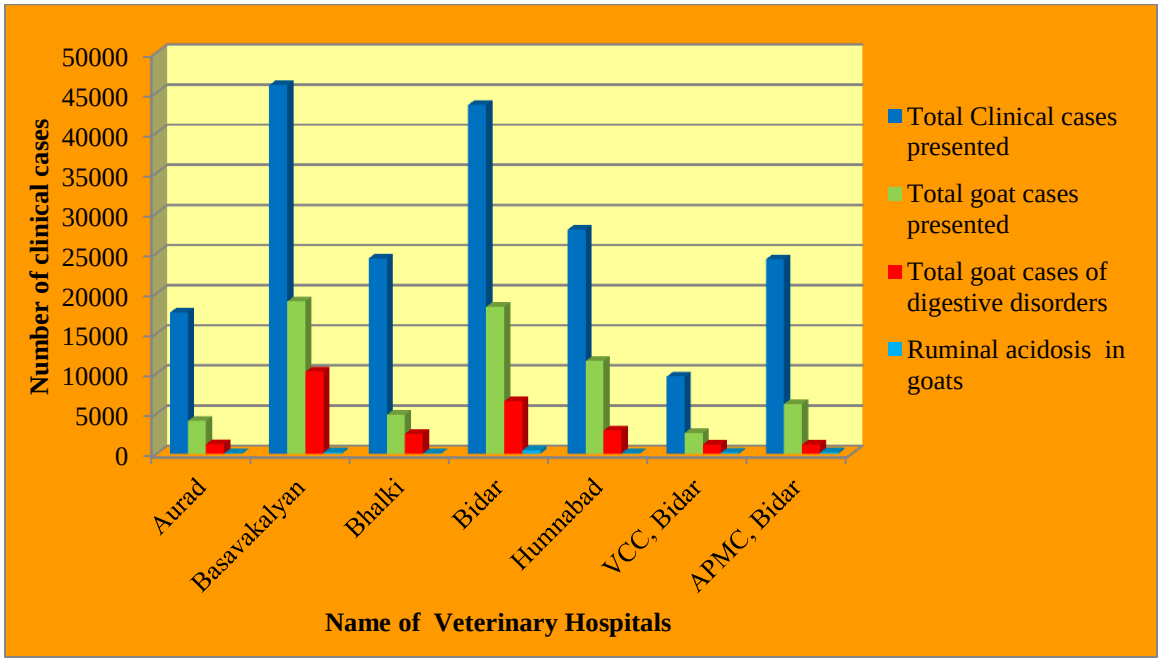


Fig. 3: Prevalence of ruminal acidosis in goats with respect to sex at five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

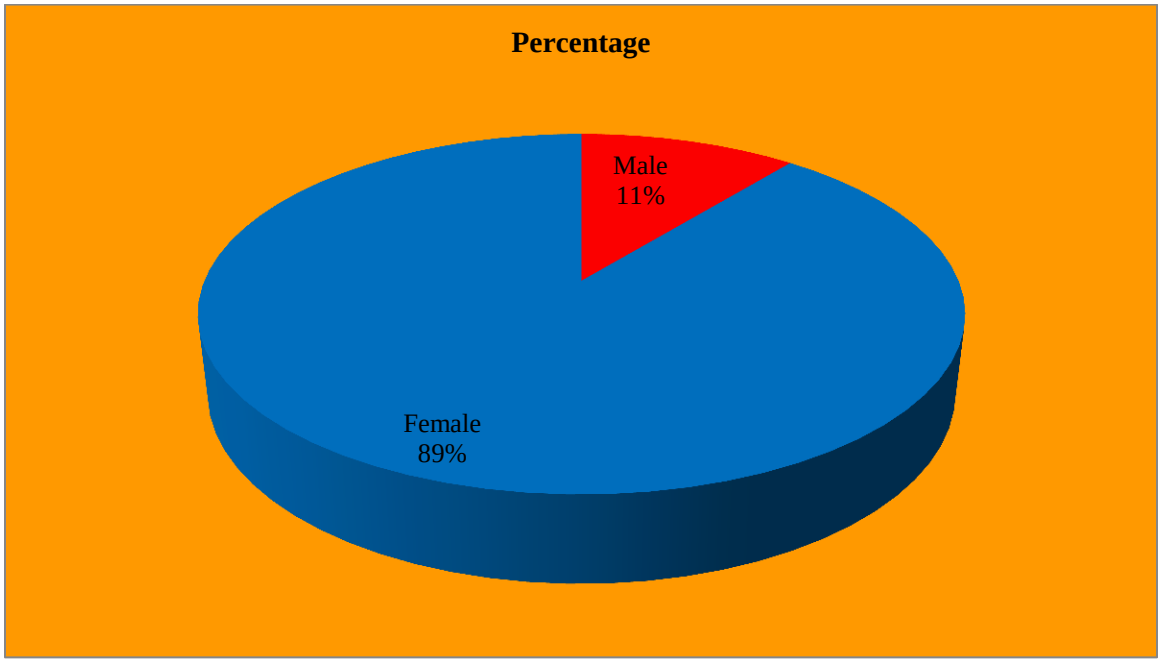


Fig. 4: Prevalence of ruminal acidosis in goats with respect to age at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

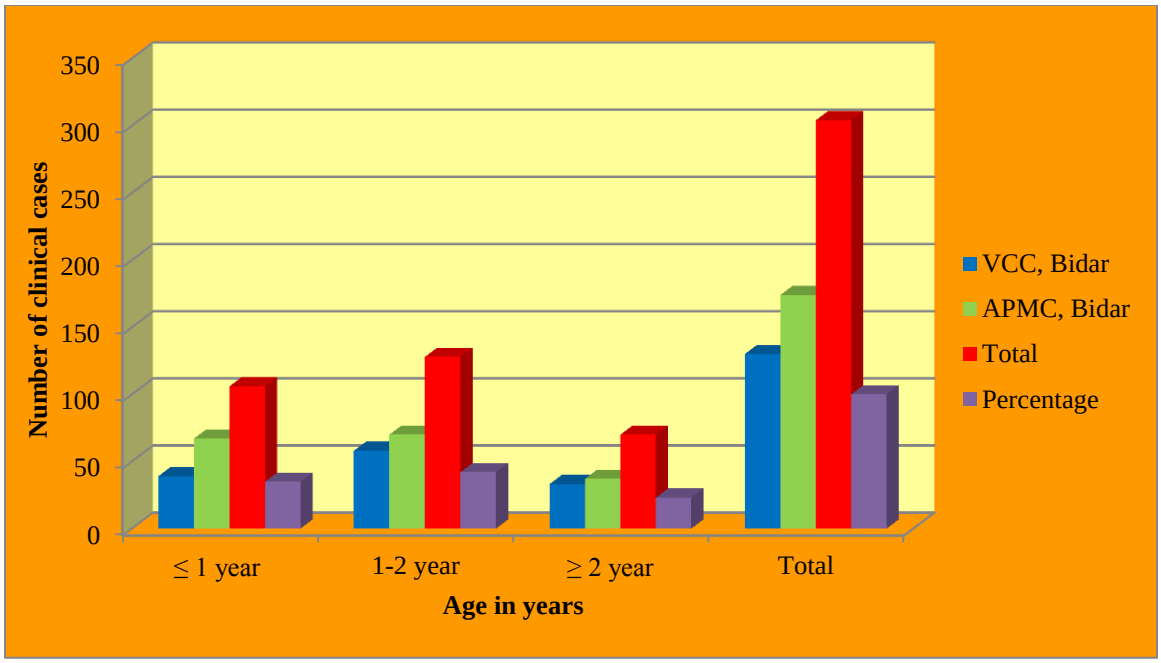


Fig. 5: Prevalence of ruminal acidosis in goats with respect to sex at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

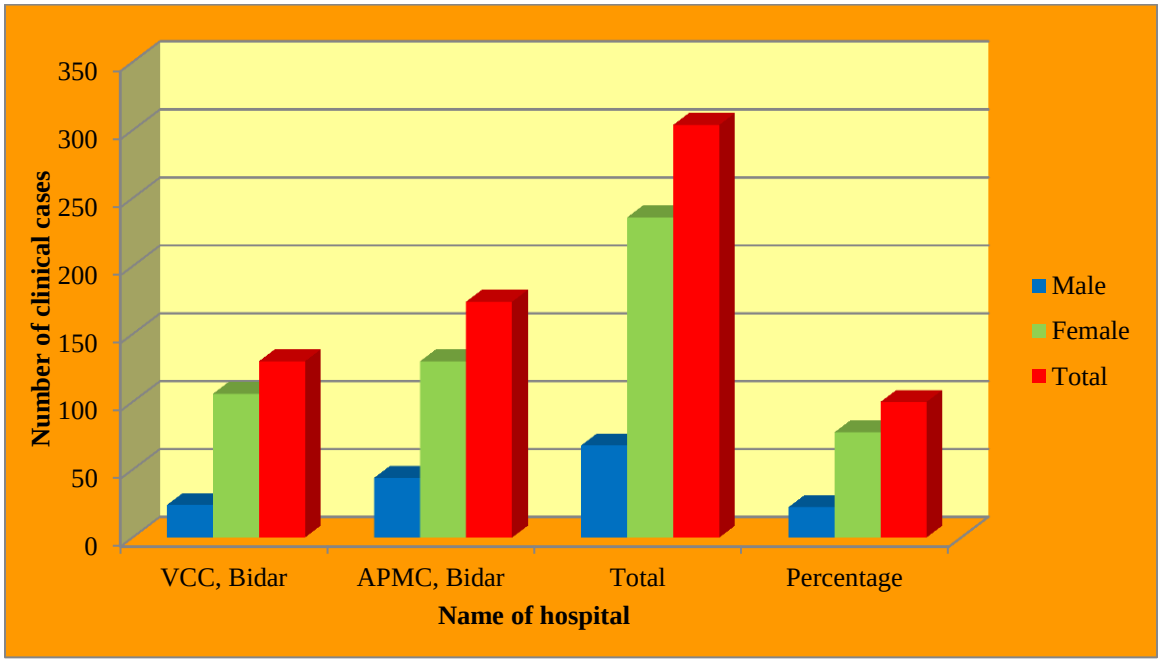


Fig. 6: Prevalence of ruminal acidosis in goats with respect to source of carbohydrate at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years (2011-2016)

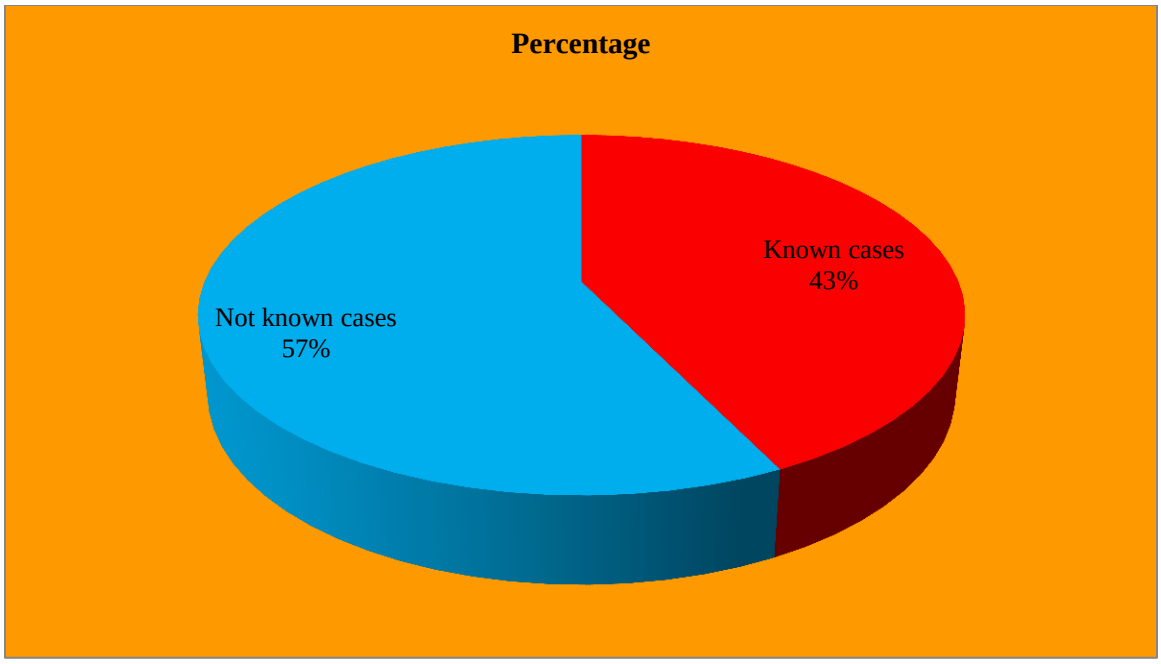


Fig. 7: Prevalence of ruminal acidosis in goats with respect to source of carbohydrate at Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years among known cases (2011-2016)

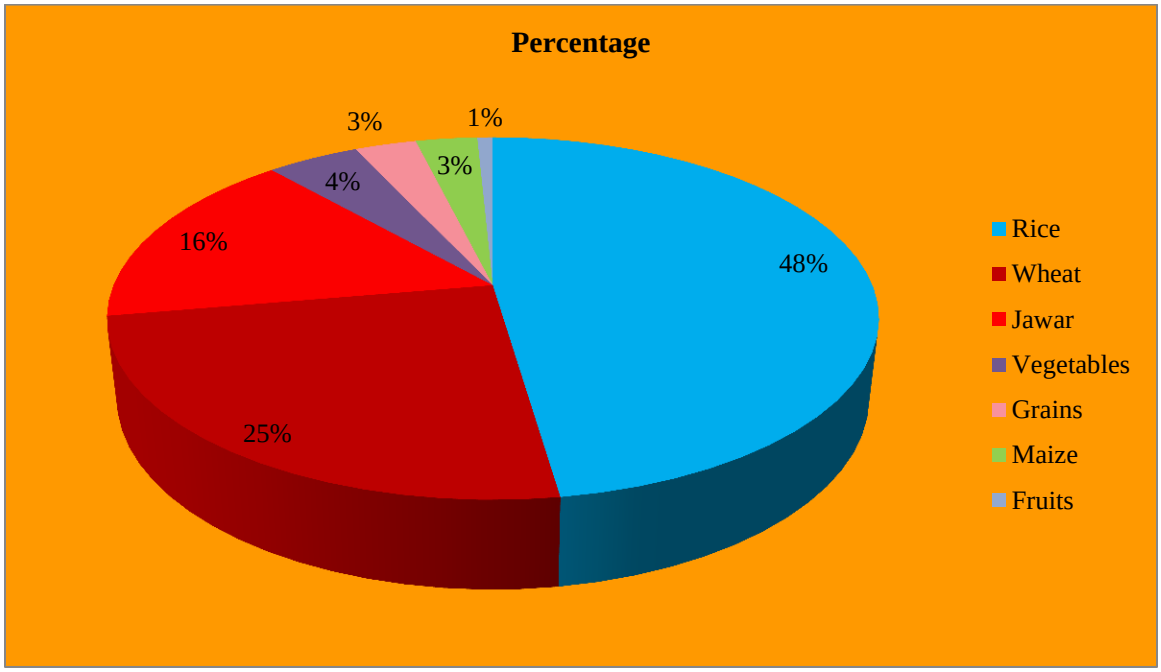


Fig. 8: Prevalence of ruminal acidosis in goats with respect to season at Veterinary Clinical Complex, Bidar for a period of five years (2011-2016)

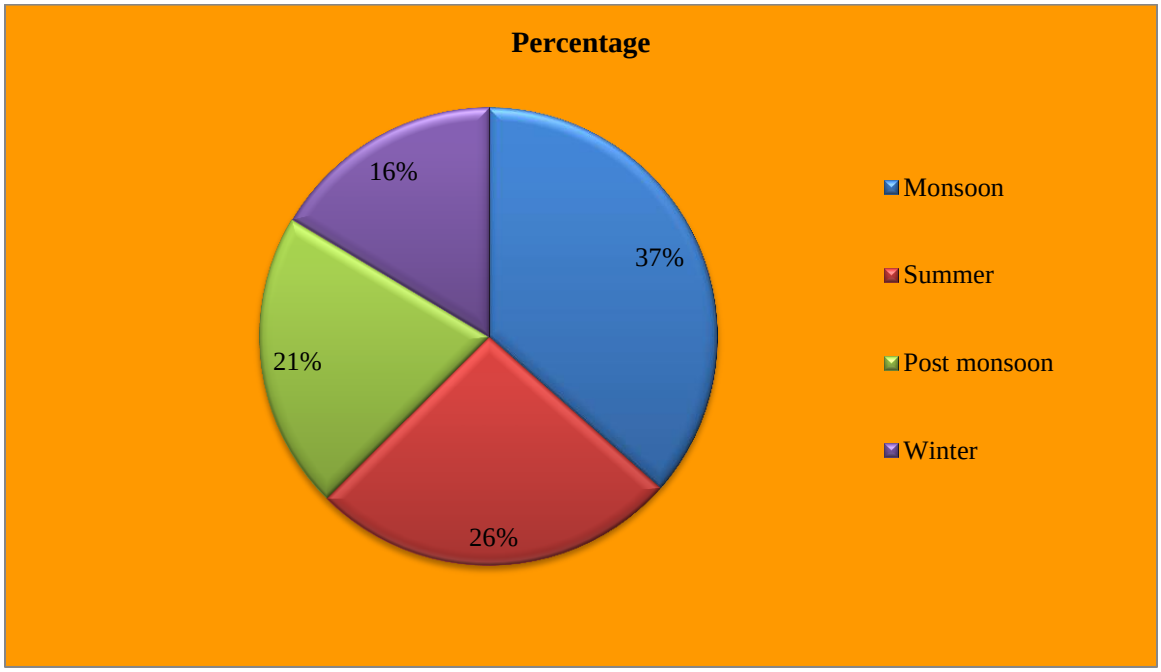


Fig. 9: Heart rate (beats/minute) in different groups of goats at different intervals

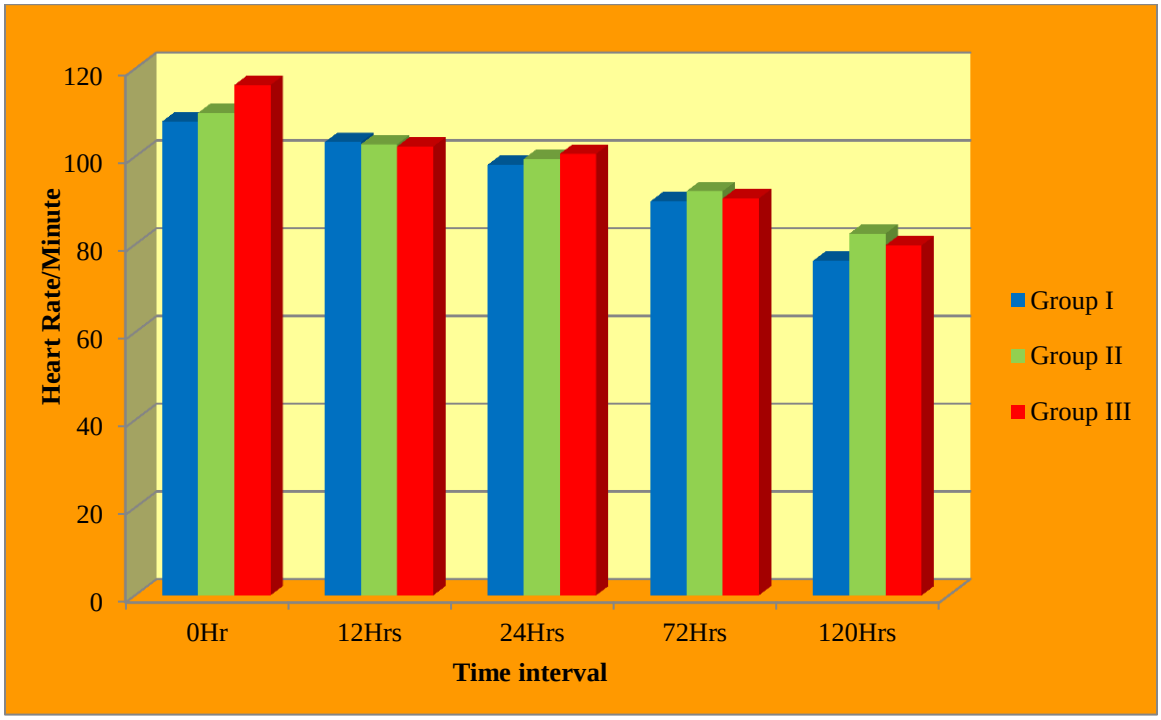


Fig. 10: Ruminal fluid pH in different groups of goats at different intervals

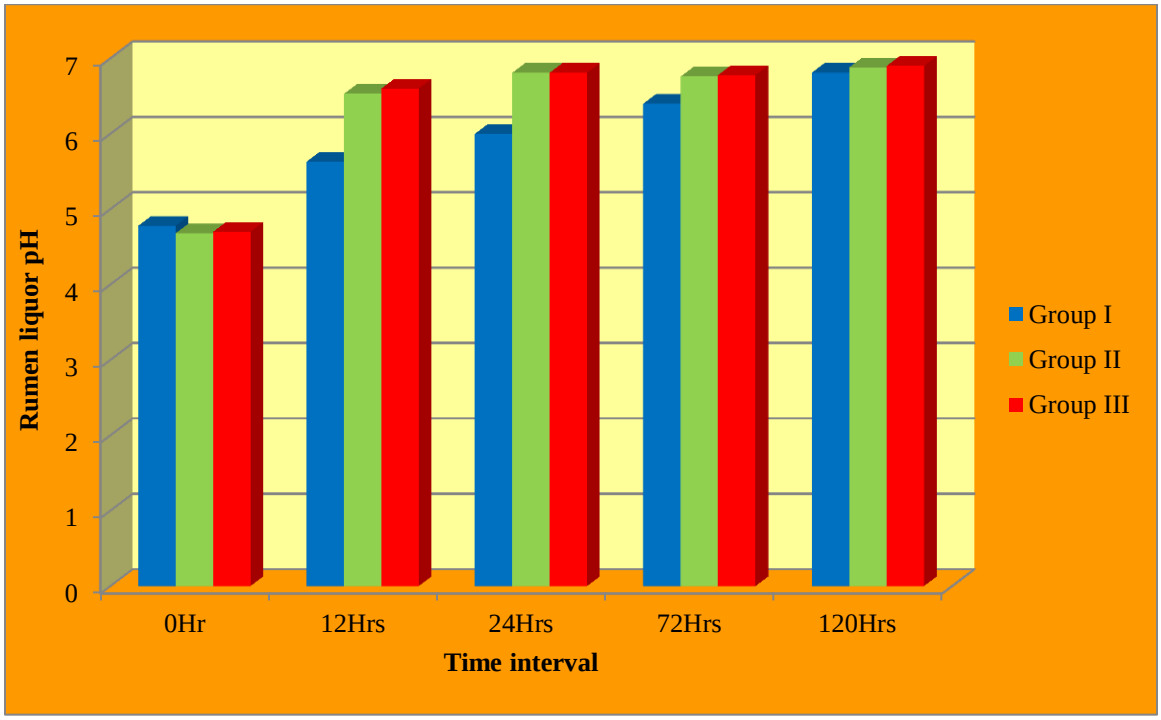


Fig. 11: Gram positive bacteria count (%) in ruminal fluid in different groups of goats at different intervals

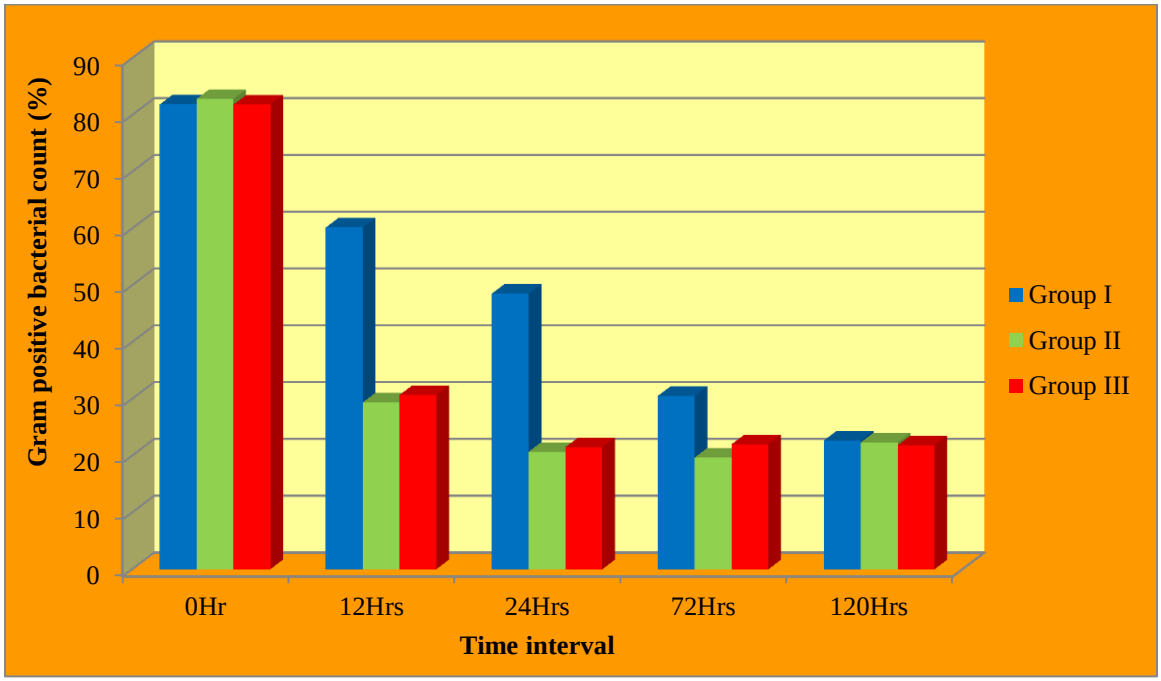


Fig. 12: Gram negative bacteria count (%) in ruminal fluid in different groups of goats at different intervals

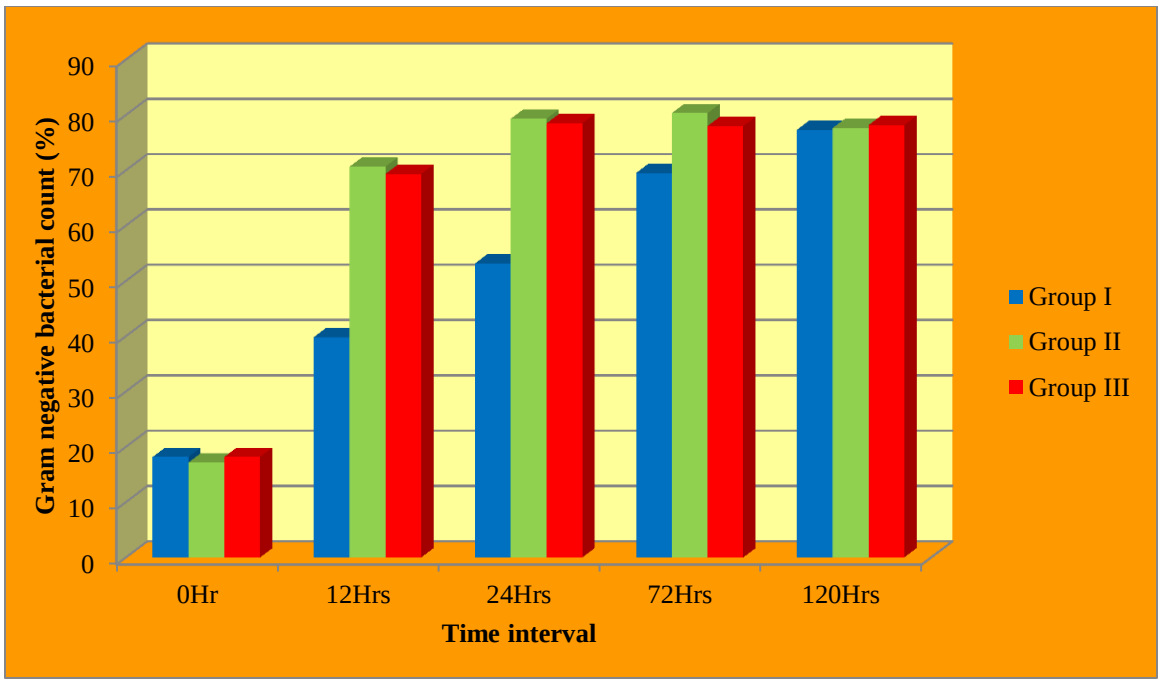


Fig. 13: Methylene blue reduction time (in minutes) of ruminal fluid in different groups of goats at different intervals

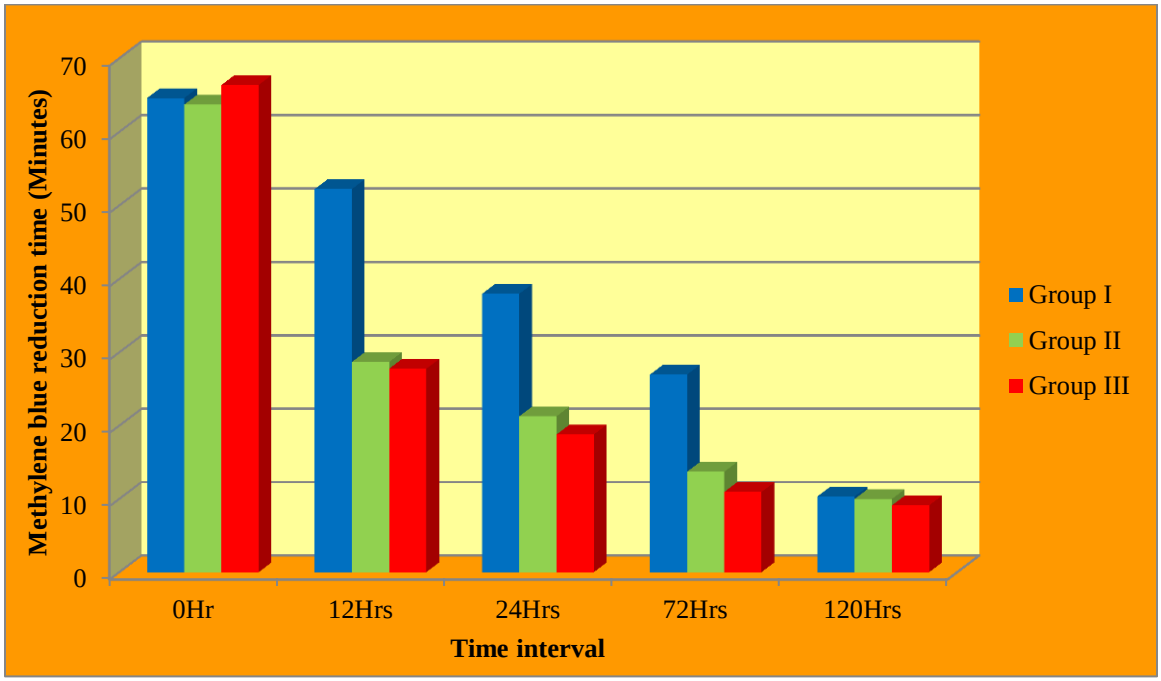


Fig. 14: Total volatile fatty acids (mmol/dL) of ruminal fluid in different groups of goats at different intervals

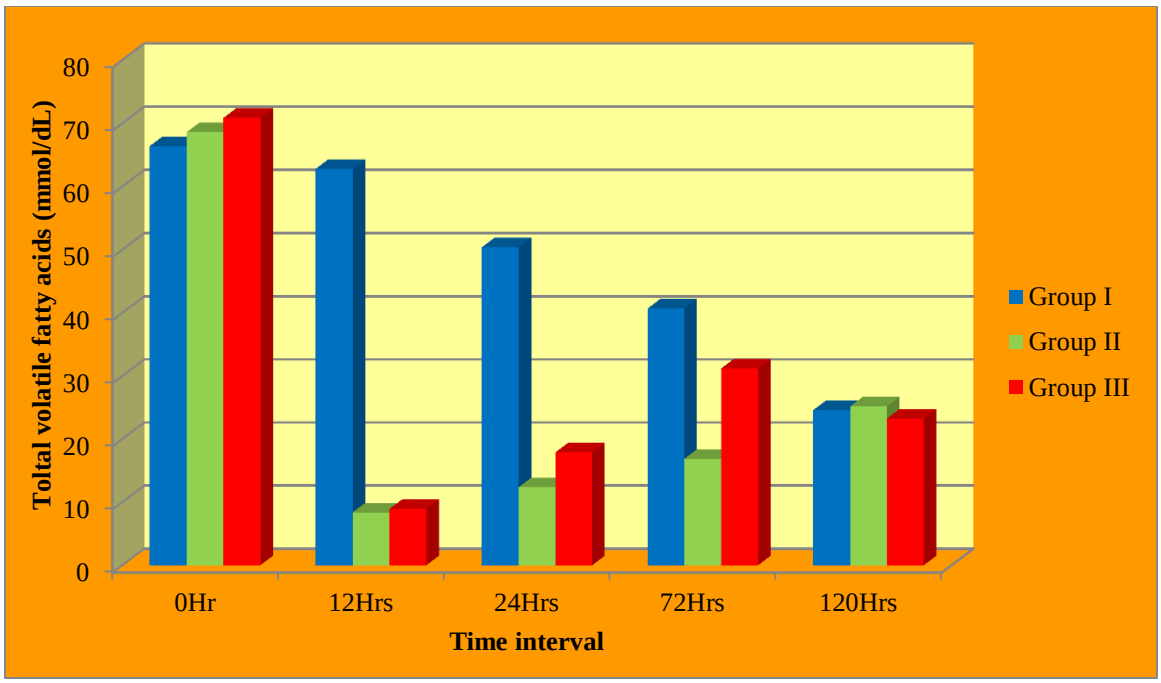


Fig. 15: Total erythrocyte count ($\times 10^6$/µL) in different groups of goats at different intervals

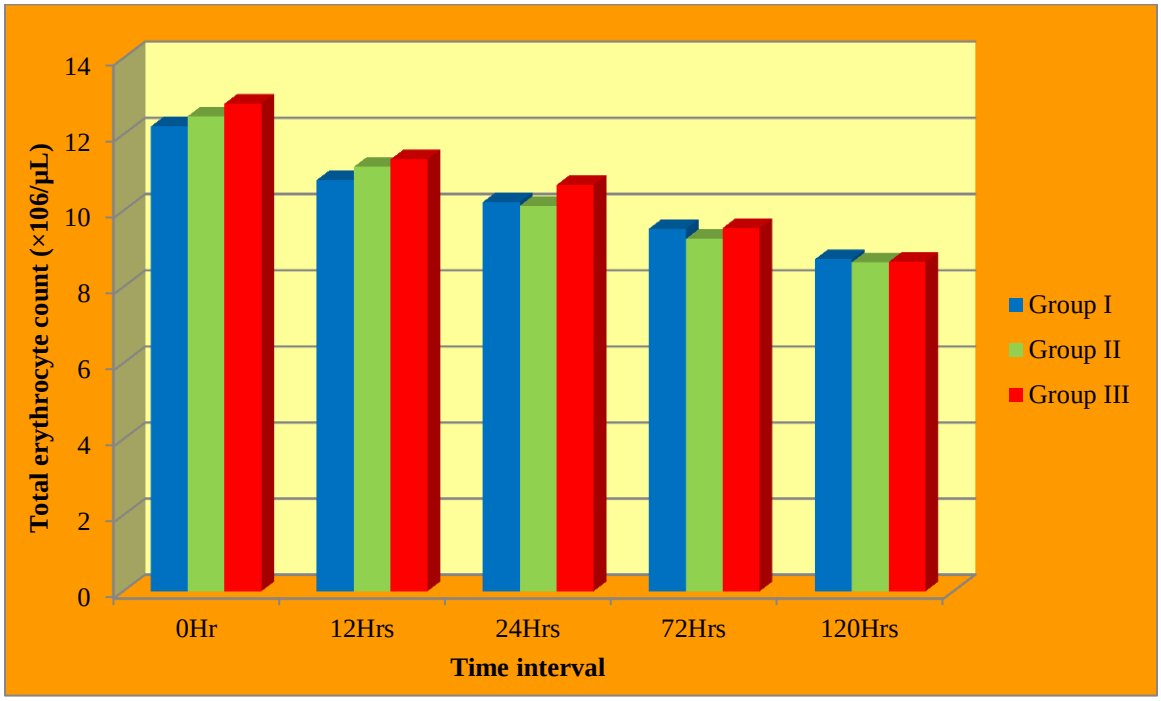


Fig. 16: Total leucocyte count ($\times 10^3$/µL) in different groups of goats at different intervals

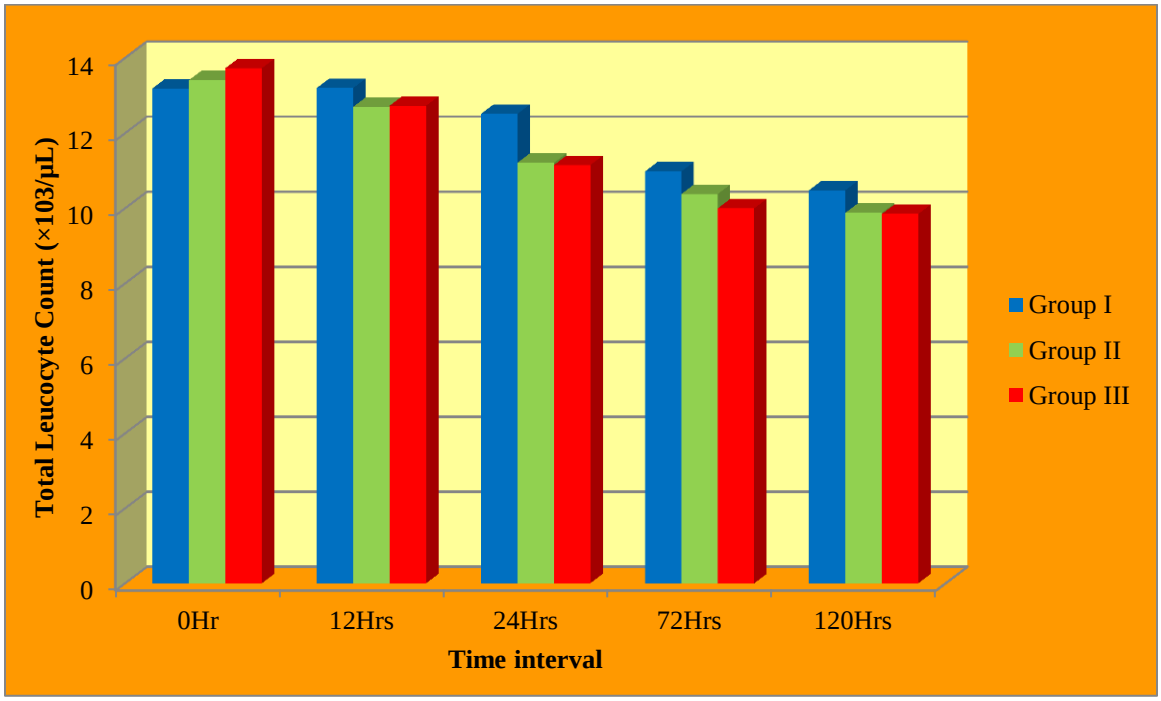


Fig. 17: Haemoglobin (g/dL) in different groups of goats at different intervals

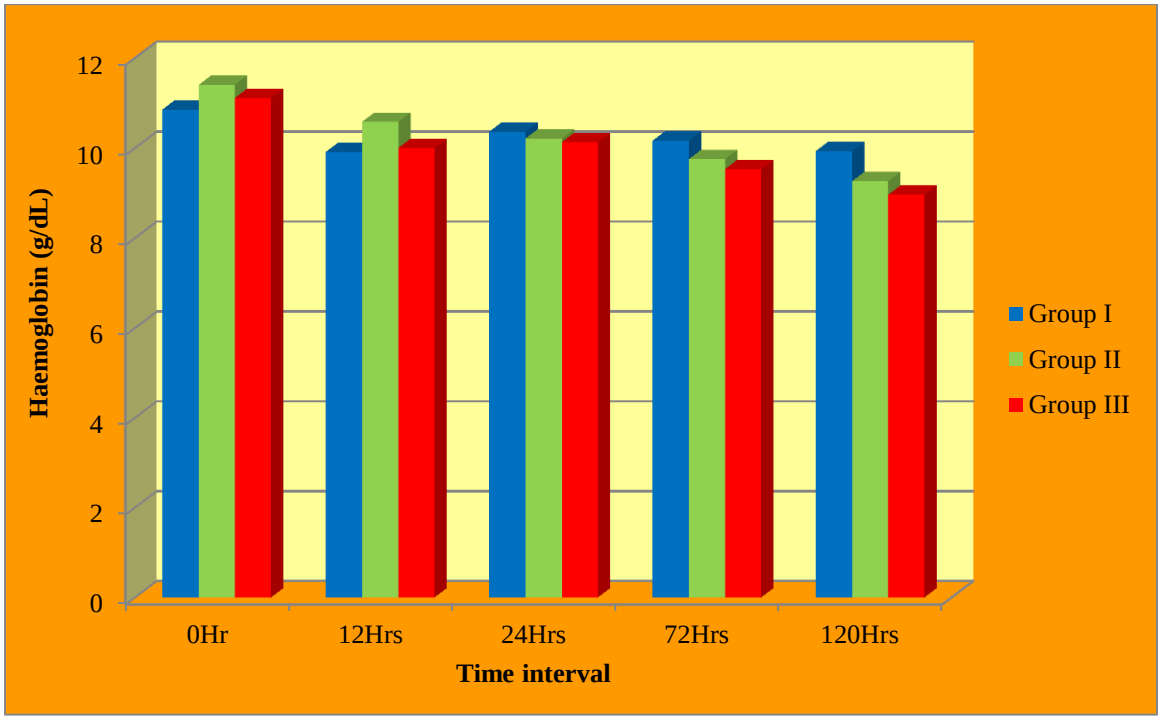


Fig. 18: Packed Cell Volume (%) in different groups of goats at different intervals

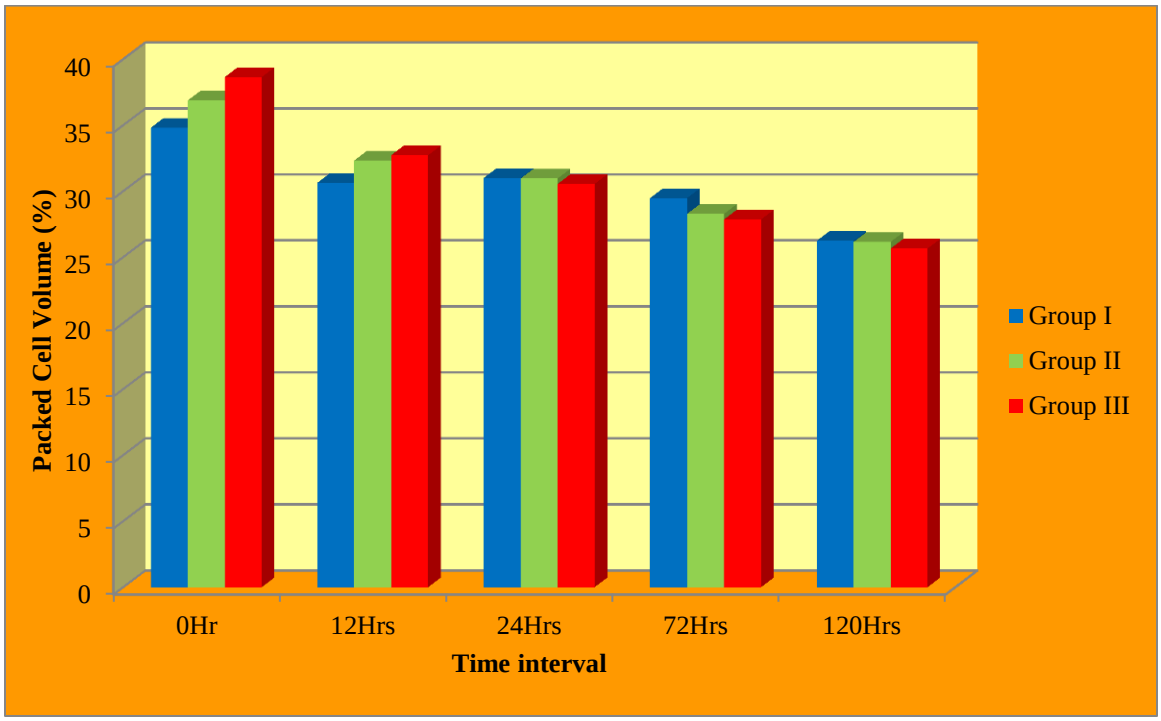


Fig. 19: Neutrophils (%) in different groups of goats at different intervals

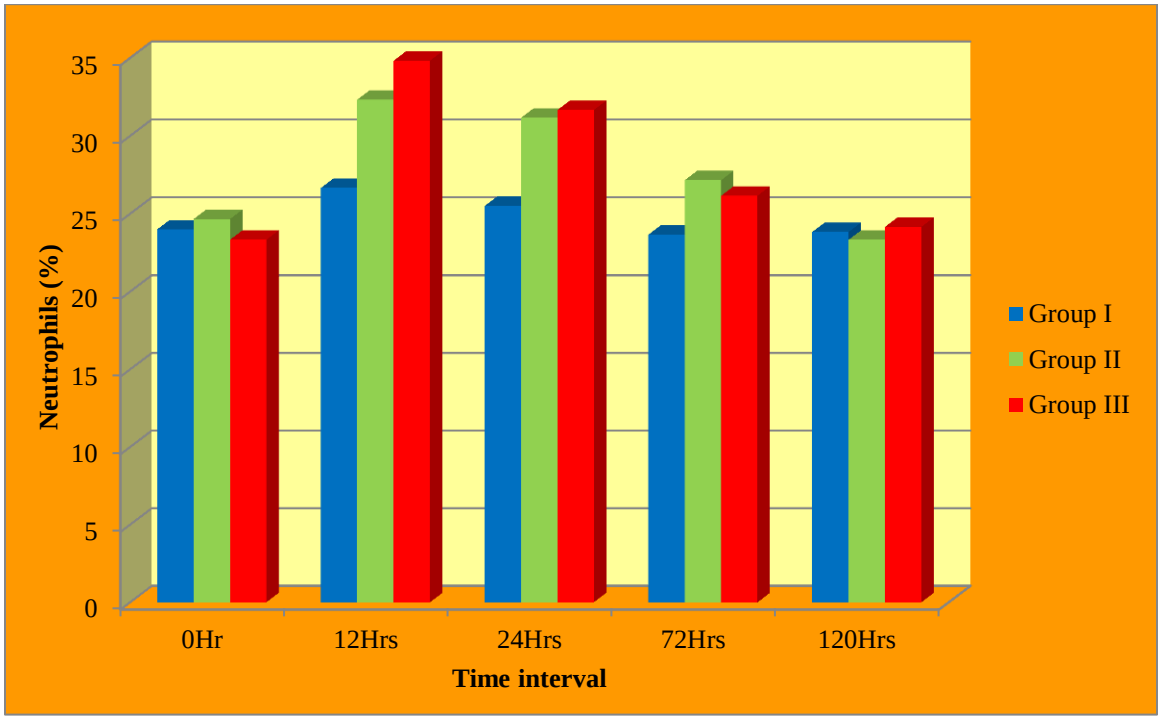


Fig. 20: Lymphocytes (%) in different groups of goats at different intervals

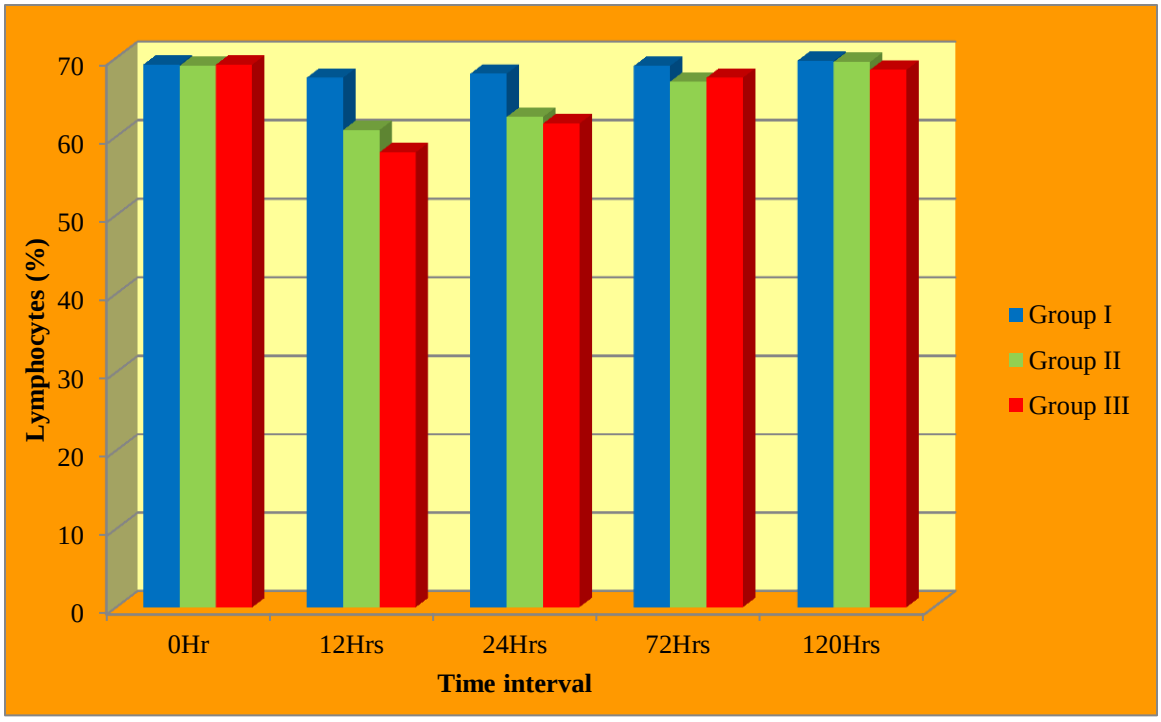


Fig. 21: Glucose (mg/dL) in different groups of goats at different intervals

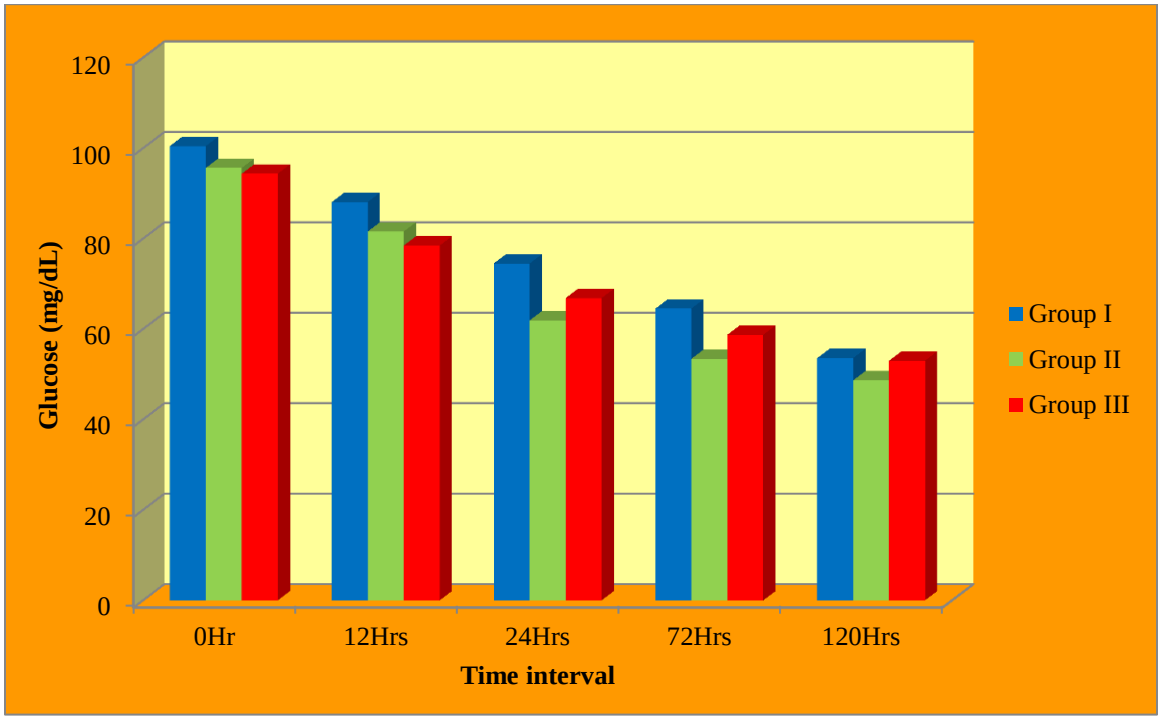


Fig. 22: AST (IU/L) in different groups of goats at different intervals

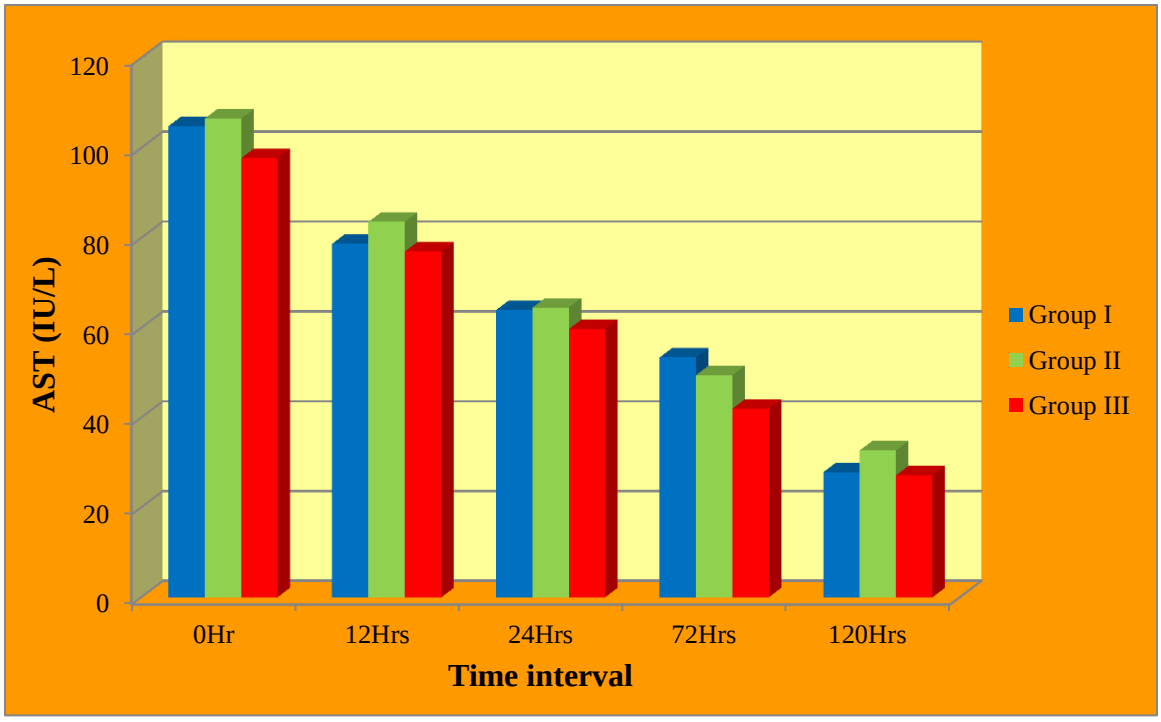


Fig. 23: ALT (IU/L) in different groups of goats at different intervals

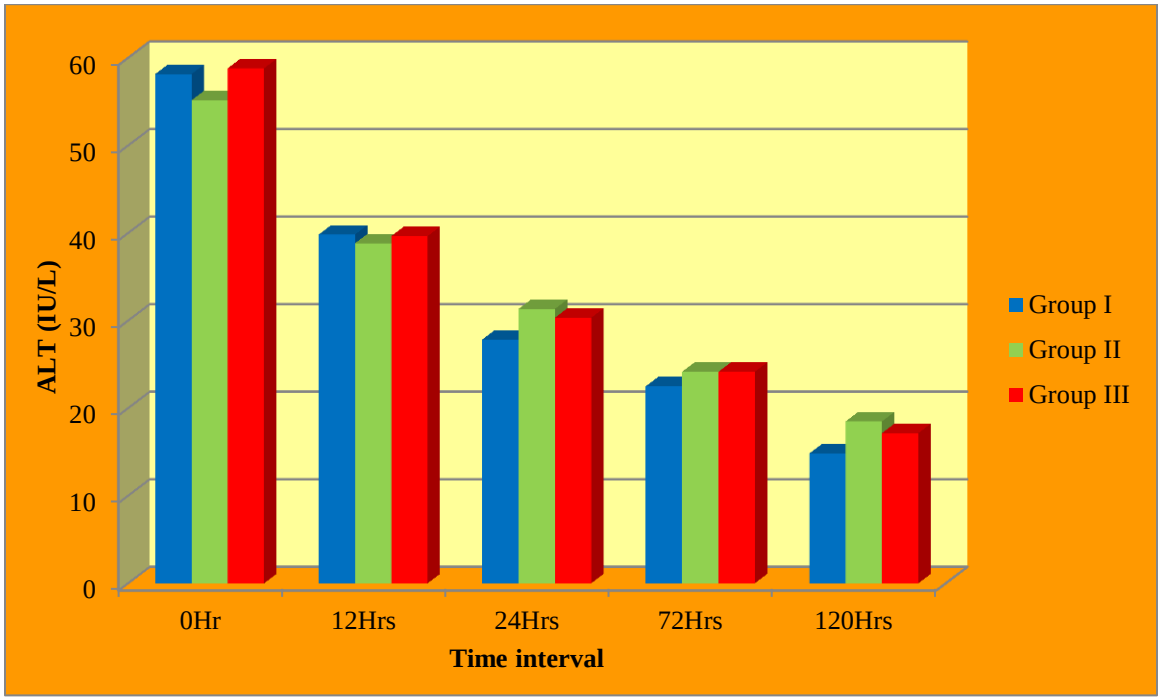


Fig. 24: Bicarbonate ions (HCO_3^- - mmol/L) in different groups of goats at different intervals

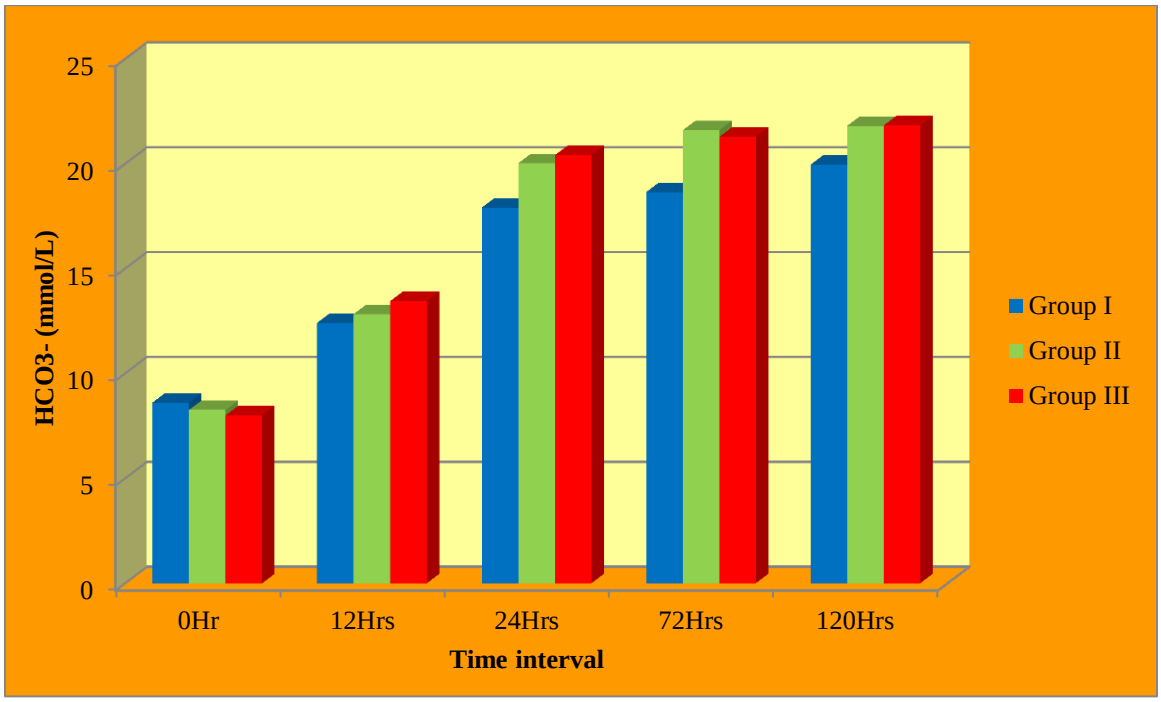


Fig. 25: Hydrogen ions (H^+- nmol/L) in different groups of goats at different intervals

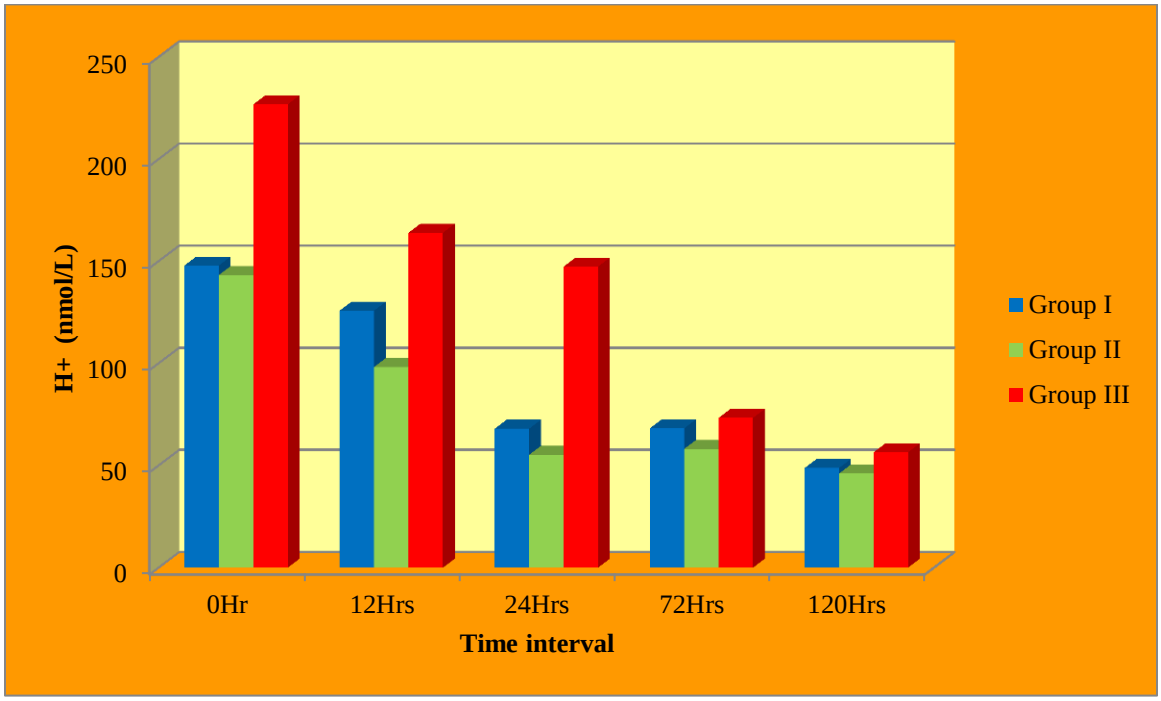


Fig. 26: $VpCO_2$ (mm of Hg) in different groups of goats at different intervals

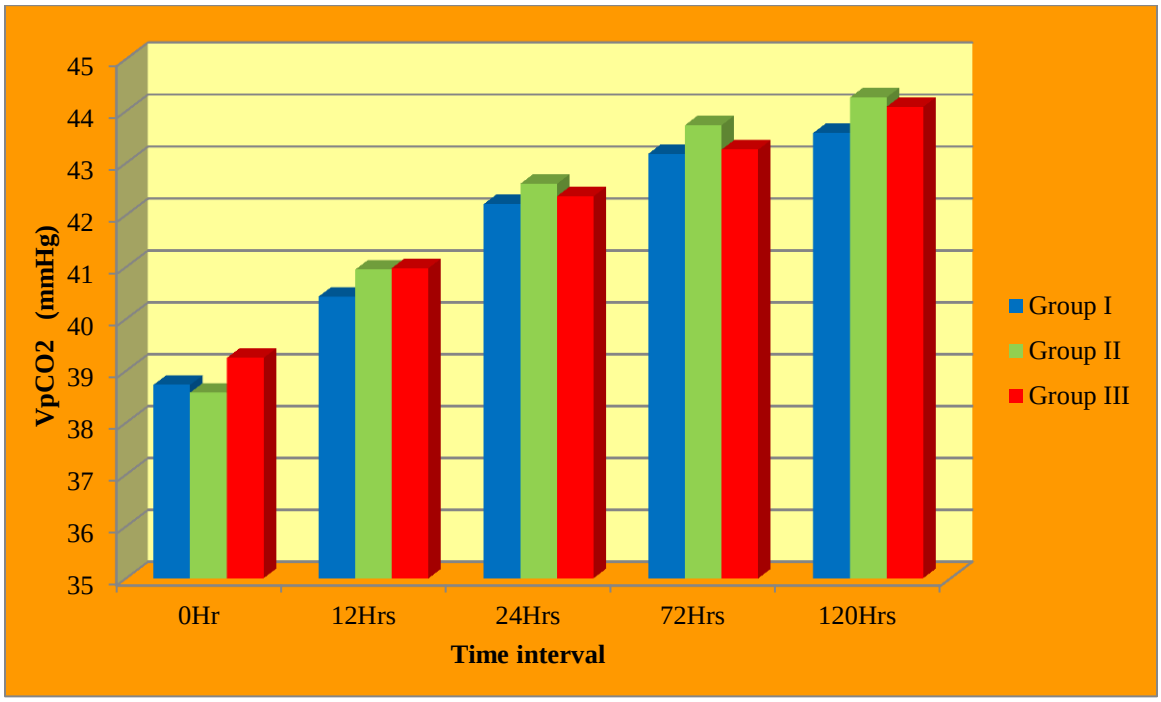


Fig. 27: Base deficits in different groups of goats at different intervals

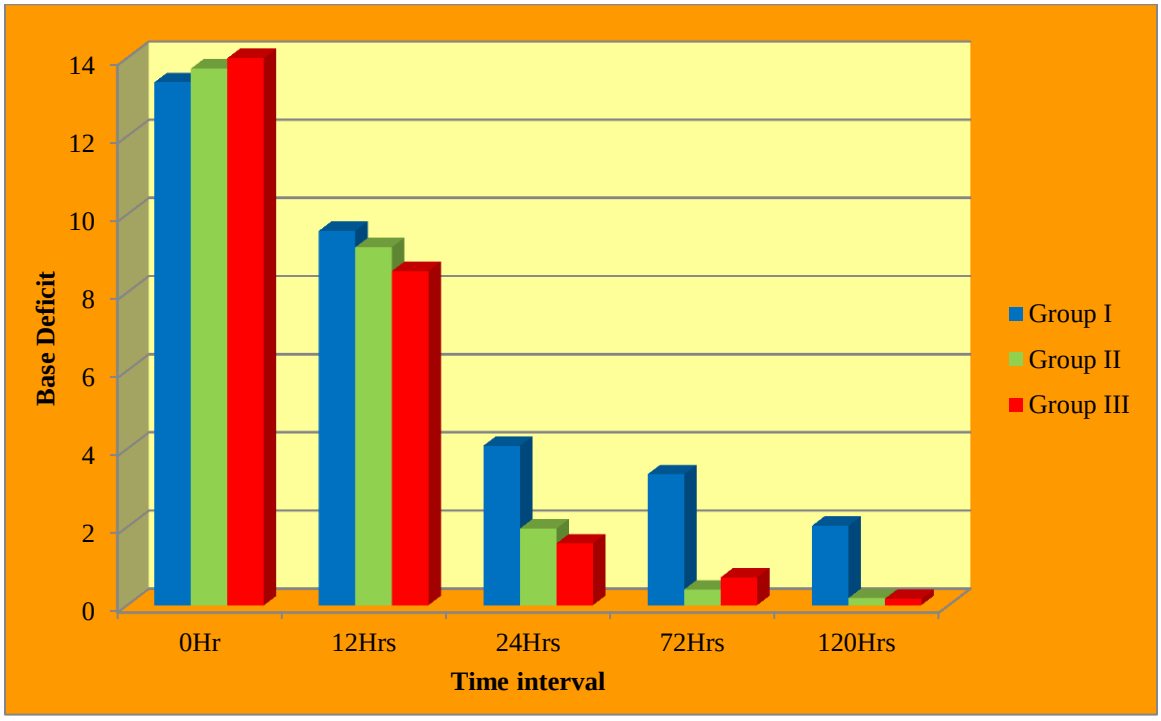


Fig. 28: Venous pH in different groups of goats at different intervals

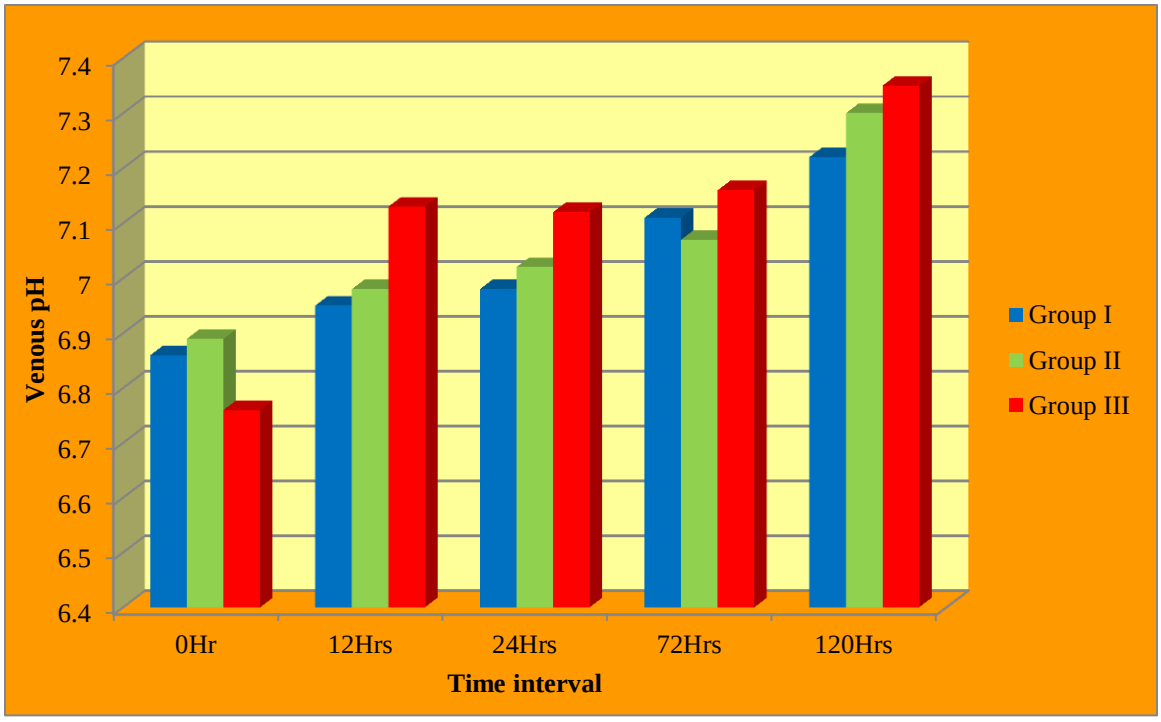


Plate 25: A goat showing clinical signs as distension of abdomen and sternal position in an acute ruminal acidosis

Plate 26: A goat showing clinical signs as distension of abdomen, and right lateral position in an acute ruminal acidosis

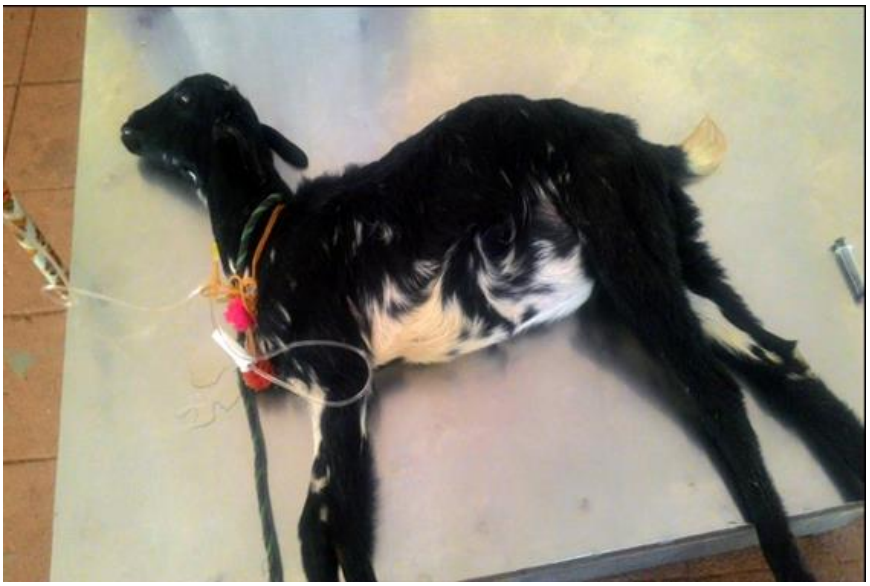


Plate 27: A goat showing clinical signs as thick pasty diarrhoea in an acute ruminal acidosis

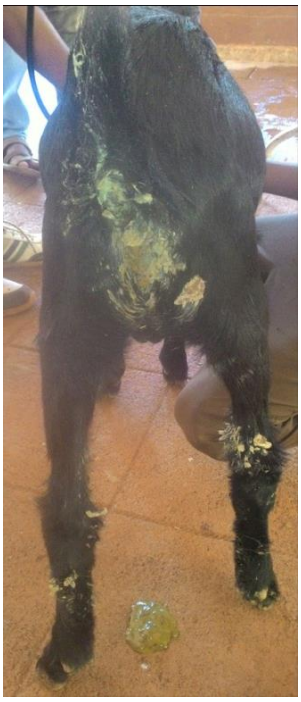


Plate 28: A goat showing clinical sign as distension of abdomen in an acute ruminal acidosis

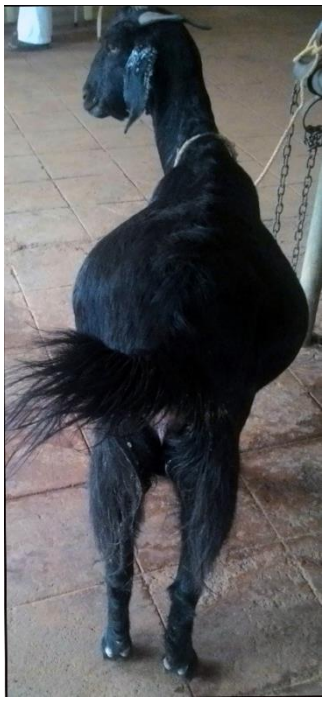


Plate 29: A goat showing distension of abdomen and diarrhoea in an acute ruminal acidosis at 0 hour before treatment (Group I)

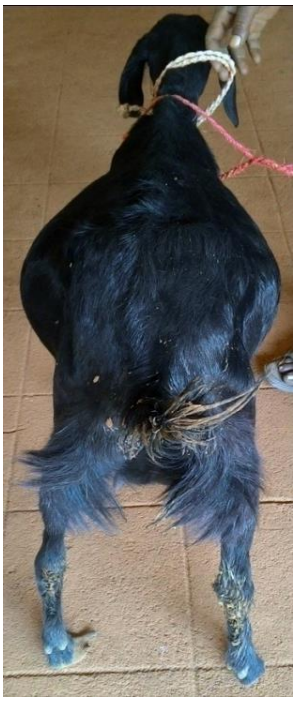


Plate 30: A goat showing slight distension of abdomen and watery diarrhoea at 24 hours after medicinal therapy in an acute ruminal acidosis (Group I)

Plate 31: A goat showing passing semisolid feaces at 72 hours after medicinal therapy in an acute ruminal acidosis (Group I)

Plate 32: A goat showing taking feed at 96 hours after medicinal therapy in an acute ruminal acidosis (Group I)

Plate 33: A goat showing taking feed at 120 hours after medicinal therapy and passing normal feaces in an acute ruminal acidosis (Group I)

Plate 34: A goat showing distended abdomen in an acute ruminal acidosis at 0 hour before treatment (Group II)

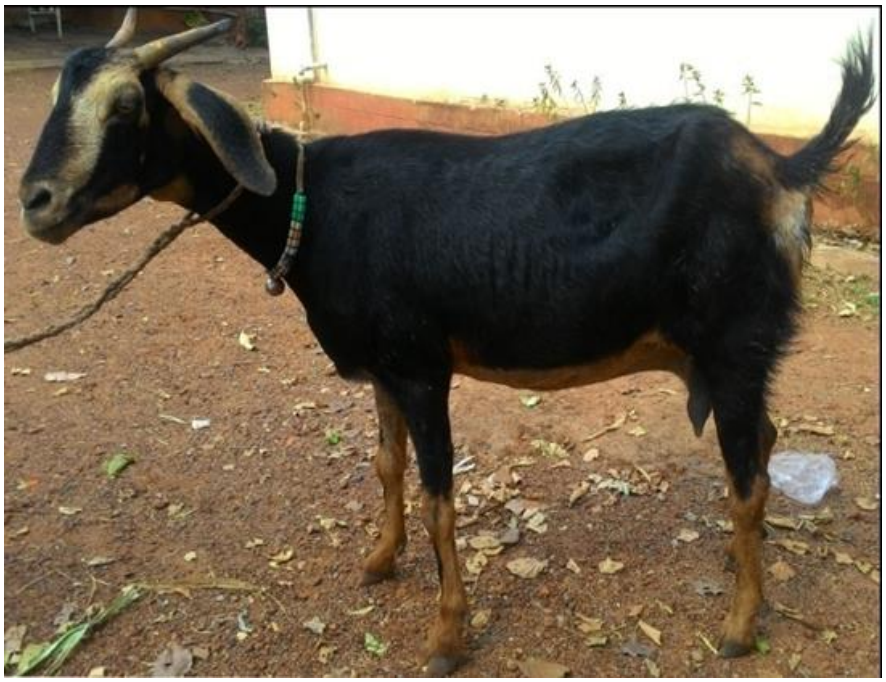


Plate 35: Rumenotomy showing the acidotic contents of the ruminal fluid, discolouration of the ruminal epithilium in an acute ruminal acidosis of goat (Group II)

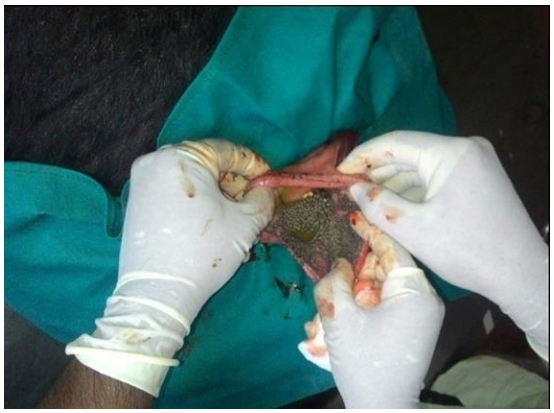


Plate 36: Animal standing at 24 hours after rumenotomy operation, taking food and water in an acute ruminal acidosis of goat (Group II)

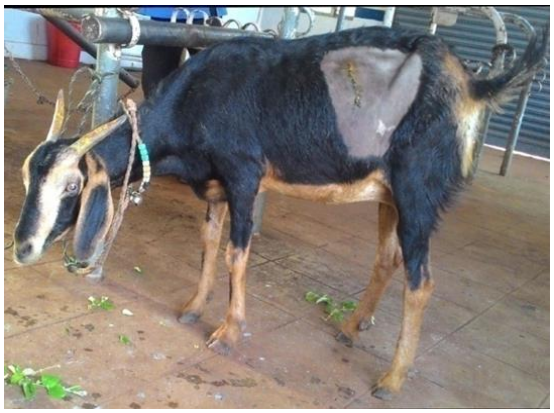


Plate 37: Photograph showing recovered animal in an acute ruminal acidosis of goat at 72 hours after surgery (Group II)

Plate 38: Photograph showing recovered animal with sutures intact in an acute ruminal acidosis of goat at 120 hours after surgery (Group II)

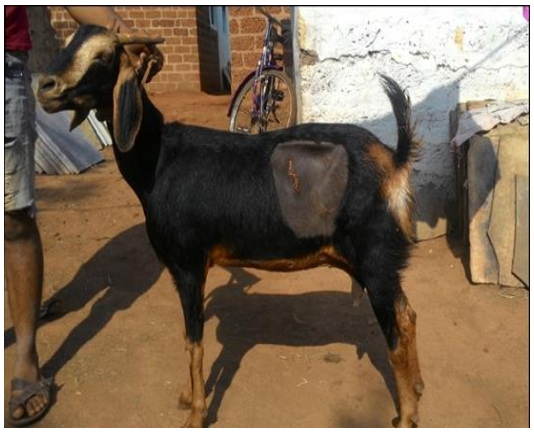


Plate 39: Photograph showing complete healed wound with sutures removed on 12th day in an acute ruminal acidosis of goat (Group II)

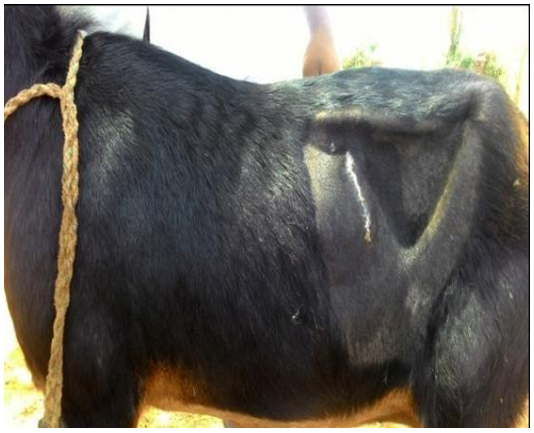


Plate 40: A goat showing distended abdomen in an acute ruminal acidosis at 0 hour before treatment (Group III)

Plate 41: Rumenotomy showing the acidotic contents of the ruminal fluid with frothy in nature in an acute ruminal acidosis of goat (Group III)

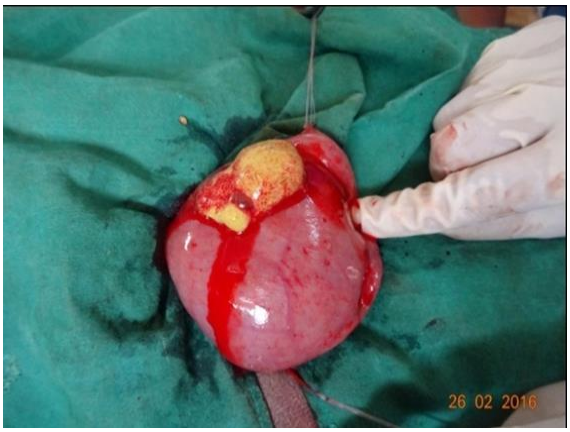


Plate 42: Animal standing immediately after rumenotomy operation in an acute ruminal acidosis of goat (Group III)

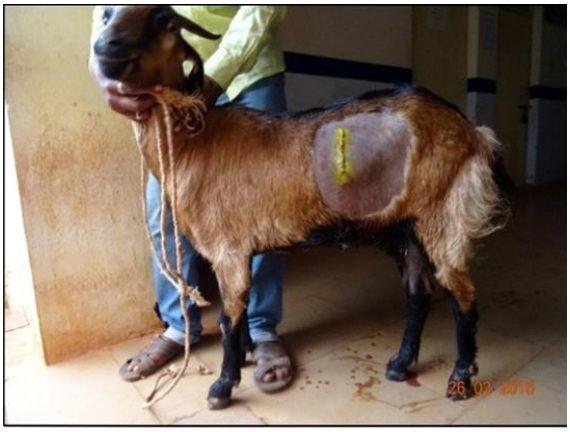


Plate 43: Photograph showing animal taking water in an acute ruminal acidosis of goat at 24 hours after surgery (Group III)

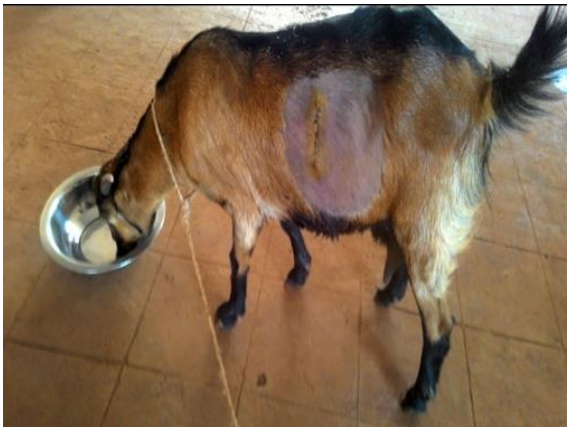


Plate 44: Photograph showing animal taking tree leaves in an acute ruminal acidosis of goat at 72 hours after surgery (Group III)

Plate 45: Photograph showing recovered animal with intact sutures in an acute ruminal acidosis of goat at 120 hours after surgery (Group III)

Plate 46: Photograph showing complete healed wound with sutures removed on 12th day in an acute ruminal acidosis of goat (Group III)

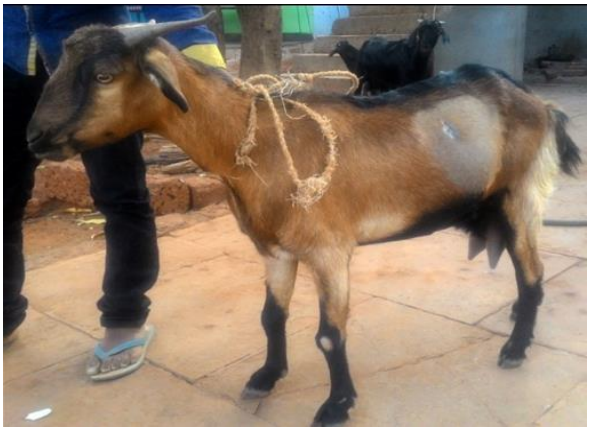


Plate 47: Photograph showing measured pH 3.7 of acidotic goat ruminal fluid by digital pH meter immediate after collection in a goat of acute ruminal acidosis of group III

Plate 48: Photograph showing measured pH 4.2 of acidotic goat ruminal fluid by digital pH meter immediate after collection in a goat of acute ruminal acidosis of group I

Discussion

V. DISCUSSION

5.1 PREVALENCE

Prevalence of ruminal acidosis in the present study were recorded 0.55 per cent among total number of clinical cases, 1.60 per cent out of total number of goat cases and 4.16 per cent out of total number of digestive disorders in goats presented to five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar from 2011 to 2016.

The prevalence of ruminal acidosis was in agreement with Nour *et al*. (1998) reported 0.6 per cent of incidence of acidosis in Nubian goats and Mahmood *et al*. (2013) reported 1.2 per cent were positive for lactic acidosis in adult goats.

On the contrary 18, 20, 13.09, 18, 9.67 and 11.12 per cent of ruminal acidosis were reported by Prasad *et al*. (1976) under field condition, Ashok Kumar *et al*. (2005) in a organized goat farm, Darwin *et al*. (2007a) in clinical cases, Gonzalez *et al*. (2010) in goat herds, Kasaralikar *et al*. (2012) in clinical cases and Panchasheel (2013) in a goats cases presented to clinics respectively.

However, incidence of ruminal acidosis was 13.12 per cent out of total number of digestive disorders in goats presented to Veterinary Clinical Complex, Veterinary College, Bidar from 2011 to 2016. This finding is in agreement with Darwin *et al*. (2007a), Kasaralikar *et al*. (2012) and Panchasheel (2013).

In the present study prevalence of ruminal acidosis was higher in female 954 (89.08%) when compared to male 117 (10.92 %) goats presented to five taluka Veterinary Hospitals of Bidar district and Veterinary Clinical Complex, Veterinary College, Bidar from 2011 to 2016. This skewed representation of sex wise incidence could be due to a definitive bias in the population under study. Earlier reports also suggest that there are more female per male (10:1) as per the standard goat husbandry practice (Kasaralikar *et al.*, 2012).

On further analysis of recorded data the highest prevalence of ruminal acidosis were recorded in 1-2 (42.10 %) years of age groups, followed by less than 1 (34.87 %) year and least in more than 2 (23.03 %) years of age groups in goats presented to Veterinary Clinical Complex, Veterinary College, Bidar for a period of last five years. This finding was in agreement with Darwin *et al*. (2007a), Kasaralikar *et al*. (2012) and Panchasheel (2013). The reason is higher prevalence in growing could be due to their less selectivity in feed as compared to older flock mates.

In this study, the source of carbohydrate was traced in 130 (42.77 %) cases among 304 cases presented to Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years 2011-2016. The highest prevalence of ruminal acidosis was recorded for rice (20.40 %), followed by wheat (10.52 %), jawar (6.90 %), vegetables (1.98 %), grains and maize (1.32 %) and lastly fruits (0.33 %) as known source. This finding is in close agreement with the findings of Darwin *et al*. (2007a), Padmaja and Praveena (2011), Choudhary *et al*. (2011), Tufani *et al*. (2013) and Wankhede *et al*. (2016). They observed that over eating of kitchen waste, market waste, eating vegetables like cabbage,

cauliflower leaves and grains as common contributory factors. However, for severe acidosis the ingestion of large quantity of rice gruel, over night soaked grain, and left over food at functions as main causative factors (Darwin *et al*., 2007a).

It was found that highest prevalence of ruminal acidosis was recorded in the monsoon season (36.51 %) followed by summer (25.99 %), post monsoon (21.05%) and least in winter season (16.45 %) in goats presented to Veterinary Clinical Complex, Veterinary College, Bidar for a period of last five years. On the contrary Darwin *et al*. (2007a) reported winter season had highest prevalence of ruminal acidosis and least in summer. Whereas, Kasaralikar *et al*. (2012) and Panchasheel (2013) reported similar findings and they observed that October and September month had higher incidence of ruminal acidosis and least in March and April respectively. In the present study the higher prevalence in monsoon and summer could be due to consumption of waste food that is available in plenty at the time of festivals and marriage seasons.

Prevalence of ruminal acidosis was observed in non descript and Osmanabadi breed of goats presented to Veterinary Clinical Complex, Veterinary College, Bidar for a period of five years 2011-2016. This could be attributed to the maximum population of non descript and Osmanabadi goats in the area of present study.

5.2 CLINICAL EXAMINATION

The clinical cases presented with a lag period of 12 to 24 hours post ingestion of food rich in carbohydrate *viz*., rice, jawar, wheat, bread, vegetables and grains. In the present study the duration of illness was 24 hours in 12 cases followed by 48 hours in 5 cases and 12 hours in 1 case with acute ruminal acidosis. The above finding is in

agreement with the Nour (2006) and reported that duration required to develop signs of ruminal acidosis was 24-36 hours post ingestion of carbohydrate rich food.

Present investigation found that frequently observed clinical signs in ruminal acidosis of goat were anorexia, distension of abdomen, passing loose feaces, fluid thrill on palpation of abdomen, dullness and tachycardia. These signs could be used as a diagnostic aid for ruminal acidosis in goats. Less commonly observed clinical signs were regurgitation of cud from the mouth, grinding of teeth, dehydration, recumbency and sudden death in goats affected with ruminal acidosis.

Clinical signs of profound dullness, recumbency, polypnoea, tachycardia in ruminal acidosis had been attributed to the toxaemia associated with distension of abdomen, regurgitation of ruminal contents and fluid splashing sounds on abdominal ballottement were due to accumulation of fluid in rumen. Similar clinical findings in acidotic goats were also observed by Tanwar and Mathur (1983a), Darwin *et al.* (2007a), Sharma *et al*. (2010), Tufani *et al.* (2013) and Ullah *et al.* (2013).

Hyperosmolarity of ruminal content due to increased lactic acid concentration and accumulation of extra cellular fluid in to rumen has been quoted as the important reason for overdistension of abdomen in animals affected with ruminal acidosis (Dunlop, 1965; Underwood, 1992 and Radostits *et al*., 2007).

In the present study the diagnosis of acute ruminal acidosis was confirmed by correlating the history, clinical signs, clinical examination and ruminal fluid examination. The acute case of ruminal acidosis was confirmed when pH of the ruminal fluid had

shown less than 5.5 pH reading in digital pH meter. The similar observations for diagnosis of ruminal acidosis in goats were reported by Sharma *et al*. (2010), Arora *et al*. (2011), Choudhary *et al*. (2011) and Singh *et al*. (2016). They concluded that confirmative diagnosis of acute ruminal acidosis could be ensured based on reduced rumen pH (3 to 5), milky grey colour, watery consistency with foul odour and absence of live microflora in rumen liquor of goats.

5.3 EFFECT OF RUMINAL ACIDOSIS ON CLINICAL PARAMETERS

The vital signs in clinical cases of acute ruminal acidosis were examined from the day of presentation (0 hour, before treatment) and after initiation of treatment at 12 hours, 24 hours, 72 hours and 120 hours.

The results have shown significant difference in the temperature within the group and in between the groups. The significant lower temperature was observed at 12 hours interval after treatment in the group II and III when compared to before treatment (0 hour). Whereas, in group I no changes were found at 12 hour interval after treatment and it significantly had higher temperature from the other two groups (II and III) at corresponding interval. Decrease in temperature of surgical group (II and III) in the present study could be attributed to the evacuation of the ruminal contents, ruminal wall lavage by normal saline and post-surgical stress by rumenotomy. On the contrary Nour (2006) had found no significant changes in rectal temperature by surgical treatment in lactic acidotic goats. Whereas in group I which had conservative medicinal approach no significant changes in the rectal temperature was observed at corresponding interval. This was in agreement with findings of Braun *et al*. (1992) and Sharma *et al*. (2010). On the

contrary Ullah *et al.* (2013) observed significant decrease in rectal temperature which later showed elevation of temperature to normal after treatment. However, the temperature in all the groups at 120 hours after treatment reached towards normal range within the group and between the groups.

In the present study there was significant increase in the heart rate at 0 hour before treatment in all the groups of animals. These findings are in accordance with the observations by Patra *et al*. (1996) Metkari *et al*. (2001) and Tufani *et al.* (2013). Radostits *et al.* (2000) who had reported that tachycardia in ruminal acidosis could be in response to ongoing dehydration, systemic absorption of lactic acid and low circulatory plasma volume.

Initially steady decrease was observed in the treated groups I, II and III at 12 hours interval after treatment and there after a significant drop in the heart rate was observed in group I, II and III at 72 hours interval after treatment. The reason could be attributed to fluid therapy for correcting the dehydration and removing the abdominal distension stress by therapeutic intervention. These findings are in accordance with Padmaja and Praveena (2011) and Tufani *et al.* (2013). However, the values had reached near to physiological range at 120 hours of after treatment in all the groups. On the contrary Nour (2006) reported that normal heart rate at 7-8 days after surgery in acidotic goats.

Respiratory rate decreased significantly from 0 hours to 120 hours in all the three groups. The significant decrease was observed at 24 hours onwards in group II and III when compared to the group I at corresponding interval after treatment. This finding was

in accordance with Nour (2006), Tanwar and Mathur (1983a), Radostits *et al.* (2007), Pradeep Kumar Ram *et al.* (2007), Tufani *et al*. (2013) and Ullah *et al*. (2013). In group I the significant lower respiratory rate was observed at 120 hours after treatment. Huber (1976) attributed elevation of respiratory rate due to stimulation of respiratory centre by increased carbon dioxide tension of blood and decreased blood pH. The respiratory rate was reached to physiological range on 72^{nd} hour after the treatment in groups (II and III) and at 120 hours after treatment in group I.

In the present study rumen contraction rate was nil at 0 hour interval and observed only at 72 hours and 120 hours after treatment in all the groups. The values were significantly higher at 72 hours and 120 hours when compared to 0 hour interval in all the three groups. At 120 hours they reached towards normal rate in all the three groups. This finding was in agreement with the Tufani *et al*. (2013) and Eldin *et al*. (2014). As in severe acidotic cases rumen motility was discernible and exhibited complete distension of left flank with complete absence of primary contractions of rumen, although the fluid filled splashing sound was usually audible on auscultation.

In group II and III at 72 hours and 120 hours of after treatment interval the rumen contraction rate was significantly higher when compared with group I at corresponding interval. This observation was in agreement with the Tufani *et al*. (2013) as improvement in rumen motility after medicinal and surgical treatment.

5.4 RUMINAL FLUID PARAMETERS

5.4.1 Changes in physical parameters

In the present study, the colour of ruminal fluid was milky white with watery consistency and sour odour on the day of presentation. These changes in rumen fluid due to acidosis are in agreement with earlier reports of Braun *et al.* (1992), Basak *et al.* (1993), Metkari *et al.* (2001), Kasaralikar *et al*. (2007), Darwin *et al*. (2007b), Rahima *et al.* (2012) and Tufani *et al.* (2013).

Gross changes in the physical properties of ruminal fluid observed in the present study are suggestive of acute ruminal acidosis in all the groups. The physical changes in ruminal fluid after treatment in all the groups could be due to therapeutic and surgical intervention which helped in creating positive ruminal environment.

5.4.2 Chemical changes in ruminal fluid

The decrease in pH of the ruminal fluid has been attributed to increased production of lactic acid by Gram +ve bacteria which predominate in acidic pH was observed on the day of presentation in all the affected groups. This is in agreement with reports of Kasaralikar *et al*. (2007), Chaudhary *et al.* (2011), Padmaja and Praveena (2011), Tufani *et al.* (2013) and Ullah *et al*. (2013).

Following therapy, increase in pH of ruminal fluid was observed in all the groups and pH returned to normal at 72 hours after treatment in group I and 12 hours after treatment in group II and group III respectively. Observations of the present study of group I are similar to the reports of Sen *et al*. (1982), Tanwar and Mathur (1983a), Braun

et al. (1992), Basak *et al*. (1993) Patra *et al*. (1993), Desai *et al*. (1999), Tufani *et al.* (2013) and Ullah *et al.*(2013). Whereas, in group II and III the early changes in the rumen pH at 12 hours after treatment could be due to surgical treatment. This finding was in agreement with the Tufani *et al.* (2013) and Nour (2006).

The significant increase for MBRT and SAT values were recorded in all the animals before treatment. Similar observations were also seen by Basak *et al.* (1993) Desai *et al.* (1999) and Pradeep Kumar Ram *et al.* (2007). This could be due to destruction of normal cellulolytic bacteria and shift in the pattern from gram-negative to gram-positive microbes (Randhawa *et al.*, 1989 and Braun *et al.*, 1992).

The group I showed significant higher MBRT value after treatment when compared to group II and III at corresponding treatment intervals. Similarly the group II showed significant higher value after treatment when compared to group III at corresponding treatment intervals. This difference in the present study could be attributed to the differences in the line of therapy. Wherein group III had earlier recovery in MBRT value, as it was response to therapy by surgical with transfaunation as compared to other groups. This finding was in agreement with Panchasheel (2013).

Gross inactivity of micro flora and decline in normal cellulolytic bacteria and shift in their pattern from gram negative to gram positive (Randhwa *et al.*, 1989) could be possible reason for this change.

In the present study the values of SAT started from 24 hours after treatment in all groups of animals. However, improvement in treated groups could be attributed to response to therapy. This was in agreement with (Desai *et al.*, 1999).

In this study, the total protozoal density was absent on the day of presentation in all the groups. Sensitivity of ruminal protozoa to the change in ruminal pH is well documented and their number declines as the pH falls below 6. The absence of protozoa in ruminal fluid with pH $\leq$ 5.5 has been reported by earlier workers viz., Garry (1990), Chaudhary *et al.*,(2011), Padmaja and Praveena (2011), Rahima *et al.* (2012), Tufani *et al.* (2013) and Ullah *et al*. (2013)**.**

In this study the protozoa appeared normal at 24 hours after treatment in group III than group I and II wherein the appearance of protozoa was observed on 120 hours after treatment. This difference between the groups in the present study could be attributed to the differences in the line of therapy. Wherein group III had earlier reappearance of ruminal protozoa, as it was response to therapy by surgical with transfounation as compared to other groups. This is in agreement with reports of Desai *et al.* (1999) and Nour *et al.* (1998) suggestive of reappearance of small protozoa 5^{th} day post treatment in acidotic goats.

The present study observed more number of gram positive bacteria as compared to gram negative bacteria in rumen liquor of all the acidotic goats. Similar results were observed by Desai *et al.* (1999), Padmaja and Praveena (2011), Arora *et al.* (2011) and Tufani *et al.* (2013) in ruminal acidosis.

The normal ratio of gram positive to gram negative bacteria was obtained by 72 hours after treatment in group I, which is in accordance with Desai *et al*. (1999), Padmaja and Praveena (2011) and Tufani *et al*.(2013). Whereas in group II and III normal ratio of gram positive to gram negative bacteria was obtained at 24 hours after treatment.

In all the groups the TVFA values were higher before treatment and this is in agreement with Randhawa *et al*. (1981), Nocek (1997), Abhishek Kumar and Verma (2003) and Abhishek Kumar and Verma (2005). The abrupt rise in TVFA could be attributable to rapid fermentation of starch by amylolytic bacteria in the rumen and subsequent decrease in the value might be due to increased absorption at low pH (Gray, 1948).

In all the three groups the value reached towards physiological range at 120 hours after treatment. However, the difference at 12, 24 and 72 hours after treatment could be attributed to differences in the line of therapy and responses to surgical therapy in group II and III.

5.5 HAEMATOLOGICAL PARAMETERS

Total erythrocyte count, haemoglobin, and packed cell volume in present study revealed significant increase on the day of case presentation. This was in agreement with Shihabudhin *et al.* (2003), Nour (2006), Sharma *et al*. (2010), and Tufani *et al.* (2013). It could be due to haemoconcentration as a result of dehydration following drawing of systemic fluid in the rumen and profuse diarrhoea (Radostits *et al*., 2000).

In group I and group III haemoglobin decreased significantly at 12 hours after treatment where as in group II it decreased significantly at 24 hours after treatment. The values of haemoglobin were reached to normal at 24 hours after the treatment in Group I, II and III compared to before treatment, values which were in agreement with earlier reports, Sharma *et al*. (2010), Rahima *et al.* (2012), Nour (2006) and Tufani *et al*. (2013).

In all the three groups the significant decline in the values of packed cell volume was observed at 12 hours after treatment and continued up to 120 hours after treatment. The PCV values were returned to normal at 24 hours after treatment in all the groups. This finding was in accordance to Sharma *et al*. (2010), Arora *et al.* (2011), Padmaja and Praveena (2011), Tufani *et al*. (2013) and Ullah *et al*. (2013). On the contrary Nour (2006) reported that PCV dropped to normal level by 2 hours post surgery in acidotic goats.

The significant declinations in the TEC values were observed at 12 hours to up to 120 hours interval after treatment in all the tee groups. The TEC normal values were obtained 24 hours after treatment onwards; this was in agreement with (Parrah *et al.,* 2010; Nour, 2006 and Sharma *et al.,* 2010).

The changes in haemoglobin, PCV and TEC values after therapeutic intervention could be attributed to inclusion of fluid therapy counteracting the ongoing dehydration.

In the present study total leucocyte count showed significant increase in acidotic goats before treatment. Similar results were also observed by Parrah *et al.* (2010), Nour (2006) and Sharma *et al.* (2010). Dunlop (1971) suggested that the elevated TLC in

ruminal acidosis could be due to release of endotoxins in rumen. In group II and III total leucocyte count differs significantly at 24 hours after treatment from group I which has higher value compared to surgical groups. The reason for this could be removal of the toxic substances from the rumen and systemic use of antibiotics in controlling the infections. The total leucocyte count reached to normal in all the groups at 72 hours after treatment. This finding is in agreement with Nour (2006).

In group I animals, neutrophils and lymphocytes values had not shown significant difference at 0 hours to 120 hours before and after treatment intervals. This finding is in agreement with Panchasheel (2013).

In group II and III animals, neutrophils showed significant difference at 12 hours after treatment, where it increased significantly and continued up to 24 hours after treatment when compared to value before treatment. This finding was in agreement with Nour (2006). Significant neutrophilia was similar to the report of Basak *et al*. (1993).

In group II and III animals, lymphocytes showed significant difference at 12 hours after treatment, where it decreased significantly and continued up to 24 hours after treatment when compared to value before treatment. This finding was in agreement with the Nour (2006). Significant lymphopaenia are similar to the report of Basak *et al*. (1993).

The values of both neutrophils and lymphocytes were returned to normal by 72 hours after treatment. This finding is in agreement with Nour (2006).

There was no significant difference found in the values of monocytes, basophils and eosinophils counts in all the treatment intervals in all the groups, similar results were observed by other workers. This finding was in agreement with the Nour (2006).

5.6 BIOCHEMICAL PARAMETERS

Increased concentration of glucose (mg / dL) in serum was recorded in all the acute ruminal acidotic goats. This was also observed by Randhawa *et al.* (1981), Metkari *et al.* (2001), Nikolov (2003), Kasaralikar *et al*. (2007), Sharma *et al.* (2010), Arora *et al.* (2011) and Tufani *et al.* (2013). Elevated glucose in lactic acidosis has been attributed to hepatic glycogenolysis due to adrenal medulla in response to stress and decreased immune-reactive insulin (Randhawa *et al.*, 1981; Basak *et al*., 1984 and Kaneko *et al*., 1999).

There was significant decrease in glucose in all the groups after treatment compared to 0 hour before treatment. In all the groups the values were higher before treatment and reached normal physiological range at 72 hours to 120 hours after treatment intervals. These present results were in close agreement with Lal *et al*. (1990), Sen *et al*. (1993), Angelov *et al*. (1996), Patra *et al*. (1997), Metkari *et al*. (2001), Nikolov (2003), Sharma *et al.* (2010), Arora *et al.* (2011) and Tufani *et al.* (2013). Comparison between the groups showed significant difference at 24 hours and 72 hours after treatment intervals.

The present study found significant increase in serum AST level in acidotic goats which could be due to hepatocellular damage as a result of toxic product like alcohol, histamine, thiaminase and other endotoxins produced in the rumen epithelium and thus

entering the portal circulation (Radostits *et al*., 2000). Patra *et al.* (1996) and Kasaralikar *et al*. (2007) reported rise in AST level in similar way in acidotic sheep and goats.

The levels were found on declining trend in all the groups and reached to physiological range at 120 hours after treatment in group I, II and III. These observations are in agreement with Braun *et al.* (1992), Alone *et al.* (2005) in goats, and Randhawa *et al.* (1981) in calves.

In all the groups the ALT values were higher at 0 hour before treatment. This was in agreement with Singh *et al*. (2001) and Sharma *et al*. (2010) found that the values of AST and ALT increased significantly in acidotic goats. It could be due to hepatocellular damage as a result of toxic product like alcohol, histamine, thiaminase and other endotoxins produced in the rumen epithelium and thus entering the portal circulation or due to dehydration resulting in to liver damage. The values of ALT reached within the physiological limit at 120 hours after treatment onwards. These responses could be due to the therapy adopted in treatment.

5.7 BLOOD GAS PARAMETERS

The blood pH of acute ruminal acidotic animals of all the groups was found to be lower than normal value which is in agreement with (Angelov *et al*., 1996 and Ullah *et al.*, 2013). Two important reasons for decrease in the pH is due to over-distension of rumen which impedes venous return to heart and impaired hepatic perfusion there by poorer lactic acid utilization which in turn leads to systemic lactic acidosis, resulting decrease in blood pH (Ullah *et al.*, 2013). Similar findings were observed by Patra *et al.*

(1996) in sheep. The decline in blood pH in ruminal acidosis in goats is also reported by Angelov *et al*. (1996), Hajikolaei *et al*. (2006) and Metkari *et al.* (2001).

There was significant increase in the venous blood pH in all the groups from 12 hours after treatment compare to 0 hour before treatment. The values were reached to normal at 120 hours after treatment in all the groups. The similar finding was observed by Nikolov (2003) that decreasing tendency in venous blood pH from 12 hour post treatment with molasses. The normal values were obtained on 130^{th} hour after starting the treatment.

In the present study, venous partial pressure of oxygen (VpO_2) showed no significant change in their values before treatment compared to after treatment in all the animals. The values fluctuated within the normal physiological limits.

The $VpCO_2$ values in the present study were lower before treatment. This is in agreement with Hajikolaei *et al*. (2006) stated that blood pCO_2 at 24 hours was 35.04 ± 0.81 when compared to 0 hour in an experimentally induced ruminal lactic acidosis in sheep. On the contrary Rahima *et al*. (2012) found elevated level of pCO_2 in acute ruminal acidotic goats.

In group I, II and III animals, showed significant higher values of $VpCO_2$ were observed from 12 hours after treatment when compared to before treatment intervals and reached within the physiological limit at 120 hours after treatment in all the three groups. This is in agreement with the Leal *et al.* (2010).

Significant decrease in bicarbonate (HCO^-_3) concentration was recorded 0 hour before treatment in goats of all the groups. This was in accordance to report of low bicarbonate by Shukla *et al.* (1999), Hajikolaei *et al*. (2006) and Rahima *et al.* (2012). Significant decrease in plasma bicarbonate on the day of presentation was suggestive of systemic metabolic acidosis.

There was significant and steady increase in the venous HCO^-_3 in all the groups after treatment compare to 0 hour before treatment. The values were reached to normal at 120 hours in all the groups. This was in agreement with Shukla *et al*. (1999).

In all the groups, increased concentration of H^+ was seen 0 hour before treatment in all acidotic goats. There was significant decrease of H^+ in all the groups after therapeutic intervention and reached within the physiological limit at 120 hours after treatment intervals.

Perusal of available literature could not find any report on the level of H^+ concentration in ruminal acidosis. However, its increased levels in present study are corroborative with decrease in venous bicarbonate suggestive of metabolic acidosis.

In the present study the base deficit values were significantly increased at 0 hour before treatment. This was also observed by Rahima *et al*. (2012). The values were reached to physiological range at 72 hours after treatment in group II and III and at 120 hours after treatment in group I. This difference could be due to the early setting of ruminal environment and responses to surgical therapy in group II and III.

5.8 TREATMENT

The study was conducted with different treatment trials in order to evaluate the treatment efficacy on clinical cases of acute ruminal acidosis in goats.

Oral antibiotics, alkalizing agents, rumenotorics, probiotics and intravenous isotonic or hypertonic sodium bicarbonate has been recommended as a therapeutic regimen for ruminal acidosis on basis of experimental trials on sheep and goats (Sen *et al*., 1982; Tanwar and Mathur, 1983a; Patra *et al*., 1997 and Metkari *et al*., 2001).

Use of combination of prebiotic and probiotic has been used to treat ruminal disorder including ruminal acidosis as it creates a positive ruminal environment for rejuvenating essential microflora (Fuller, 1989; Rohilla, 2005; Boodoor *et al.*, 2010; Padmaja and Praveena, 2011; Kasaralikar, 2005 and Tufani *et al.*, 2013).

Treatment in group I included oral and intravenous administration of sodium bicarbonate as alkalizer and bufzone as buffering agent. The usage of intravenous administration of sodium bicarbonate as alkalizer is in agreement with Patra *et al*. (1997). The usage of buffzone as buffering agent has helped in stabilization of ruminal pH and improvement digestion. This was in agreement with Padmaja and Praveena (2011) and Rohilla *et al*. (2010). It was found that effect of probiotic (saccomyces cerevisiae) alone and with nutri-mix feeding had a significant effect on lactating goats (Rohilla *et al*., 2010). The live cell present in probiotic causes better ammonia rumen utilization and it improved production of microbial protein and volatile fatty acids, whereas decreased lactic acid peaks in the rumen, stabilised the rumen pH (Kander *et al*., 2000 and Kander *et al*., 2005). All animals of this group recovered without any complications.

Treatment in group II included rumenotomy followed by intravenous administration of sodium bicarbonate as alkalizer and Ecotas bolus as probiotic drug to normalize rumen ecosystem. Earlier reports also justify use of intravenous sodium bicarbonate in the place of other electrolyte as preferred protocol for the treatment of ruminal lactic acidosis to counteract metabolic acidosis (Sen *et al*., 1982; Patra *et al*., 1997; Radostits *et al*., 2000, Kasaralikar, 2005 and Tufani *et al.*, 2013). In this group Ecotas bolus was used for improvement in digestion and stabilisation of ruminal environment. This was in agreement with Rohilla *et al*. (2010), Choudhary *et al*. (2011) and Eldin *et al*. (2014). As per use of probiotics and symbiotics in the ruminants Kander *et al*. (2000) and Kander *et al*. (2005) found that live cell present in probiotic causes better ammonia rumen utilisation and it improved production of microbial protein and volatile fatty acids, whereas decreased lactic acid peaks in the rumen and stabilised the rumen pH.

Treatment in group III included rumenotomy followed by intravenous administration of sodium bicarbonate as alkalizer and transfaunation to rejuvenate rumen environment. Similar opinion was reported by Sen *et al*. (1982), Patra *et al*. (1997) and Nour (2006). They had treated with hypertonic sodium bicarbonate intravenous to obtain rapid clinical recovery in acidotic goats. Tanwar and Mathur (1983a), Shukla *et al*. (1999) Metkari *et al.* (2001) and Kasaralikar *et al*. (2007) suggested intra ruminal administration of antibiotics, antacids and fresh rumen liquor transplant for early recovery in ruminal acidosis.

As there was an emergency situation with holding of food and water pre-operativley was not practicable in this study. All the twelve goats of group II and III underwent similar procedure of pre-operative preparation. The aseptic preparation of the surgical site on left flank with antiseptic Betadine® solution before surgery were beneficial in controlling infection during and after post-operative surgery. Inj. streptopencillin @ 10 mg / kg body weight was administered intramuscular prior to surgery as pre-operative antibiotic coverage. In both the groups of II and III animals, surgery was performed under regional block using local linear infiltration and inverted "L" block in the left flank region of the animal. As goats were sensitive to lignocaine the 1% was prepared from 2% by mixing equal volume of drug and distil water as V/V (1:1) this is in agreement with Fubini and Ducharme (2004). The anaesthetic procedure performed in this study was excellent. There was no anaesthetic emergency during the surgical procedure or lack of analgesia in either of the group II and III.

The surgery was performed in sternal position on the raised operation table. It was convenient for the surgeon to perform surgery without any difficulty. This was in agreement with the Frank (2002).

Animals of group II and III presented with acute ruminal acidosis were subjected to emergency rumenotomy to remove the source of carbohydrate and undigested feed material from the rumen to save the life. Same treatment method was followed by O' Connor (1985), Frank (2002), Tyagi and Singh (2004), Fubini and Ducharme (2004) and Das *et al*. (2011) for removing the acidotic contents from the rumen of acute ruminal acidosis in animals. Thorough lavaging of the rumen with normal saline after

rumenotomy was performed to remove the contents adhering to the rumen wall. Same method was followed by RAGFAR (2007) and preferred that in severe cases of ruminal acidosis should be treated by giving intravenous hypertonic saline fluids and lavaging of rumen with a wide bore stomach tube in combination with transfaunation from a healthy animal. It was also supported and reported by Parrah *et al.* (2010), Das *et al*. (2011), Tufani *et al*. (2013) and Tawheed *et al*. (2017). In acidotic goats of group II and III, rumenotomy was performed, followed by ruminal lavage with normal saline and replacement with fresh ruminal fluid along with presoaked chopped fodder to restore ruminal microfloura and fauna. This was in agreement with Nour (2006) who had followed emergency rumenotomy to evacuate the ruminal contents, followed by ruminal lavage and replacement with fresh ruminal fluid along with presoaked hay to restore ruminal microfloura and fauna in experimentally induced lactic acidotic goats.

Surgical wound dressing was carried out for each animal of group II and III by using five percent povidone iodine solution and Loraxene® ointment till the healing of the surgical wound. Inj. streptopencillin (10 mg / kg body weight, intramuscular, once a day for seven days) and Inj. tolfenamic acid (2 mg / kg body weight, intramuscular, once daily for three days) were given. The animals of the both the groups had excellent recovery from the surgery. In two cases of group III had wound dehiscence at the surgical site and it was managed by open wound dressing with antiseptic povidone iodine solution till healing was observed. After complete healing of laparotomy wound, sutures were removed on 12^{th} day of after operation. All the six cases of each group II and III with ruminal acidosis were recovered without any complications.

Ancillary therapy in this study included streptopencillin, thiamine hydrochloride, chlorphenaramine maleate and calboral and these were given as common treatment protocol to all affected goats. The supportive therapy also helped in early recovery and prevention of complications during treatment period or post-opreative period.

In order to evaluate the comparative efficacy among the therapeutic trial, mean values of thirteen sensitive parameters at 12 hours and 72 hours after treatment in group I, II and group III were compared.

At 12 hours after treatment, in group I out of fourteen parameters four parameters (ruminal fluid pH, TVFA, MBRT and gram positive organism) significantly differ from other two groups II and III. Whereas, no significant difference was observed between group II and III at all the fourteen parameters. This shows the values of fourteen parameters of group I, II and III were improved when compared to 0 hour interval before treatment and much better improvement was observed at 12 hours interval after treatment in group II and III as compared to group I.

The values of important parameters of different groups of goats at 72 hours intervals after treatment were compared. In group I out of fourteen parameters eight parameters (ruminal fluid pH, TVFA, MBRT, gram positive organism, PCV, HCO^-_3, Base deficit and glucose) significantly differed from other two groups II and III. The significant difference was observed in four parameters (MBRT, PCV, Base deficit and glucose) out of fourteen when compared between group II and III. This showed the values of fourteen parameters of group I, II and III were improved when compared to 0 hour interval before treatment, 12 hours after treatment interval and the much better

improvement was observed at 72 hours interval after treatment in group II and III as compared to group I.

Among three groups, in group III the mean recovery hour (74.57 ± 12.07) in the form of normalcy attained in physical, heamatobiochemical and ruminal fluid parameters was earlier compared to group II (78.00 ± 12.50) and I (113.14 ± 4.65). Hence it can be concluded that therapeutic protocol used in group III consisting of rumenotomy, isotonic sodium bicarbonate intravenous as alkalizing agent along with cud transplantation can be used as preferred therapy for the acute ruminal acidosis for early recovery. Alternatively group II treatment protocol consisting of rumenotomy, isotonic sodium bicarbonate intravenous as alkalizing agent along with Ecotas® (oral) can be used. However, group I treatment protocol consisting of sodium bicarbonate as alkalizing agent, intraruminal antibiotic and oral Bufzone®, as rumen buffering agent can also be used for the treatment of ruminal acidosis in goats at dispensary level where surgical intervention is not feasible.

Summary

VI. SUMMARY

The “studies on acute ruminal acidosis in goats with special reference to therapeutic and surgical management” was carried out at Department of Veterinary Surgery and Radiology, Veterinary College, Bidar. The research was conducted in clinical cases of eighteen goats suffering from acute ruminal acidosis referred for treatment. The animals were randomly divided into three groups of six animals in each group. The therapeutic efficacy of various drugs in comparison with surgical treatment was evaluated for management of acute ruminal acidosis in goats.

Prevalence of ruminal acidosis in the present study were recorded 0.55 per cent among total number of clinical cases and 4.16 per cent out of total number of digestive disorders in goats. It was higher occurrence in females (237) 77.96 per cent than males (67) 22.04 per cent, higher in non-pregnant 209 (88.56 %) than pregnant 27 (11.44 %) and highest in 1-2 (42.10 %) years of age groups followed by less than 1 (34.87 %) year and least in more than 2 (23.03 %) years of age groups in goats. The source of carbohydrate rich food was highest for rice (20.40 %), followed by wheat (10.52 %), jawar (6.90 %), vegetables (1.98 %), grains (1.32 %), maize (1.32 %) and lastly fruits (0.33 %) and occurrence of disorder was found highest in the monsoon season (36.51 %) followed by summer (25.99 %), post monsoon (21.05%) and least in winter season (16.45 %) in goats presented to Veterinary Clinical Complex, Veterinary College, Bidar.

The most frequently observed clinical signs were anorexia, distension of abdomen, passing loose faeces, fluid thrill on palpation of abdomen, dullness and tachycardia whereas less common were regurgitation of cud from the mouth, grinding of

teeth, dehydration, recumbent and sudden death in goats affected with ruminal acidosis. The diagnosis of acute ruminal acidosis was confirmed by correlating the history, clinical signs, clinical examination and ruminal fluid examination. The acute case of ruminal acidosis was confirmed when pH of the ruminal fluid had shown less than 5.5 pH reading in digital pH meter.

The results showed significant difference in the temperature. The significant lower temperature was observed in the surgical group (II and III) after surgery when compared to medicinal group (I). However, the temperature in all the groups reached towards normal range at 120 hours after treatment. The increased heart rate and respiratory rate was observed on the day of presentation in all the groups before treatment and followed by decreased trend was observed after treatment. The significant decrease was observed at 24 hours onwards in group II and III when compared to the group I at corresponding interval after treatment indicated that early recovery was observed in surgical group compared to medicinal group.

In the present study rumen contraction rate were nil at 0 hour interval and at 120 hours they reached towards normal rate in all the three groups. In group II and III at 72 hours and 120 hours of after treatment interval the rumen contraction rate was significantly higher when compared with group I at corresponding interval indicating improvement in response to therapy.

The physical properties of ruminal fluid showed milky white with watery consistency and sour odour on the day of presentation and were suggestive of acute ruminal acidosis in all the groups. The normal colour, odour and consistency were

observed in ruminal fluid after treatment at 24 hours in group III and at 72 to 120 hours in group II and I respectively. The decrease in pH of the rumen fluid was observed on the day of presentation in all the affected groups. Following therapy increase in pH of ruminal fluid was observed in all the groups and pH returned to normal at 72 hours after treatment in group I and 12 hours after treatment in group II and group III respectively. The earlier changes in the ruminal fluid pH at 12 hours after treatment could be due to surgical treatment.

The chemical properties of ruminal fluid showed changes in total protozoal density was absent on the day of presentation in all the groups. The protozoa appeared normal at 24 hours after treatment in group III than group I and II wherein the appearance of protozoa was observed on 120 hours after treatment. In group III had earlier reappearance of ruminal protozoa, as it was response to therapy by surgical with transfaunation as compared to other groups. The present study observed more number of gram positive bacteria as compared to gram negative bacteria in rumen liquor of all the acidotic goats. The normal ratio of gram positive to gram negative bacteria was obtained by 72 hours after treatment in group I and whereas in group II and III normal ratio of gram positive to gram negative bacteria was obtained at 24 hours after treatment. The significant increase in the MBRT and SAT values were recorded in all the animals before treatment. The values for MBRT after treatment decreased in all the three groups however, the differences among the groups were observed. Wherein group III had earlier recovery in MBRT value than other groups. The values of SAT started to reduce from 24 hours and reached to normal 120 hours after treatment in all groups of animals. In all the

groups the TVFA values were higher before treatment. In all the three groups the value reached towards normal at 120 hours after treatment.

Total erythrocyte count, haemoglobin, and packed cell volume in present study revealed significant increase on the day of case presentation. The values of haemoglobin were reached to normal at 24 hours after the treatment in group I, II and III compared to before treatment. The significant decline in the values of packed cell volume was observed at 12 hours after treatment and continued up to 120 hours after treatment. The PCV values were returned to normal at 24 hours after treatment in all the groups. The significant declinations in the TEC values were observed at 12 hours to up to 120 hours interval after treatment in all the three groups. The TEC normal values were obtained 24 hours after treatment onwards.

The total leucocyte count showed significant increase before treatment. In group II and III total leucocyte count differs significantly at 24 hours after treatment from group I which has higher value compared to surgical groups. The total leucocyte count reached to normal in all the groups at 72 hours after treatment.

In group I animals, neutrophils and lymphocytes values had not shown significant difference at 0 hour to 120 hours before and after treatment intervals. In group II and III animals, neutrophils showed significant difference at 12 hours after treatment, where it increased significantly and continued up to 24 hours after treatment when compared to value before treatment. In group II and III animals, lymphocytes showed significant difference at 12 hours after treatment, where it decreased significantly and continued up to 24 hours after treatment when compared to value before treatment. The values of both

neutrophils and lymphocytes were returned to normal by 72 hours after treatment. There was no significant difference found in the values of monocytes, basophils and eosinophils counts in all the treatment intervals in all the groups.

On biochemical examination of serum from the affected goats, increased concentration of glucose was recorded before treatment. There was significant decrease in glucose in all the groups after treatment and reached normal physiological range at 72 hours to 120 hours after treatment intervals. In all the groups the AST and ALT values were higher before treatment. The levels were found on declining trend and reached to normal levels at 120 and 12 hours after treatment in group I, II and III respectively.

Blood gas analysis was done to estimate the level of metabolic acidosis in affected goats. The blood pH of acute ruminal acidotic animals of all the groups was found to be lower than normal value. The significant increase in the venous blood pH from 12 hours after treatment compare to before treatment. The values were reached to normal at 120 hours after treatment. Venous partial pressure of oxygen (VpO_2) showed no significant change in their values before treatment compared to after treatment. The $VpCO_2$ values were lower before treatment. In group I, II and III animals, showed significant higher values of $VpCO_2$ were observed from 12 hours after treatment and reached within the physiological limit at 120 hours after treatment in all the three groups.

The significant decrease in bicarbonate (HCO^-_3) concentration in acute ruminal acidotic goats of all the groups on the day of presentation was suggestive of systemic metabolic acidosis. There was significant increase in the venous HCO^-_3 in all the groups after treatment compare to before treatment. The significant difference between the

groups was observed after treatment indicating the response to therapy and values reached to normal at 120 hours in all the groups. In all the groups, increased concentration of H^+ was seen before treatment. There was significant decrease of H^+ in all the groups after therapeutic intervention and reached within the physiological limit at 120 hours after treatment. The base deficit showed significantly increased values at before treatment. The values were reached to normal at 72 hours after treatment in group II and III and at 120 hours after treatment in group I. This difference could be due to the early setting of ruminal environment and responses to surgical therapy.

The study was conducted with different treatment trials in order to evaluate the treatment efficacy on clinical cases of acute ruminal acidosis. Among three groups, in group III the recovery in the form of normalcy attained in biochemical, heamatobiochemical and ruminal fluid parameters was earlier at 72 hours after treatment compared to group I and II. Hence it can be concluded that therapeutic protocol used in group III consisting of rumenotomy, isotonic sodium bicarbonate intravenous as alkalizing agent along with cud transplantation can be used as preferred therapy for the acute ruminal acidosis for early recovery. Alternatively group II treatment protocol consisting of rumenotomy, isotonic sodium bicarbonate intravenous as alkalizing agent along with Ecotas® (oral) can be used. However, group I treatment protocol consisting of sodium bicarbonate as alkalizing agent, intraruminal antibiotic and oral Bufzone®, as rumen buffering agent can also be used for the treatment of ruminal acidosis in goats at dispensary level where surgical intervention is not feasible.

Ancillary therapy in this study included streptopencillin, thiamine hydrochloride, chlorphenaramine maleate, calboral and fluid therapy. This supportive treatment helped early recovery from the condition and had prevented complications in goats affected with ruminal acidosis.

CONCLUSIONS:

1. Overall prevalence of ruminal acidosis was 1.60 per cent out of total number of goat cases with higher occurrence in females than males and highest in 1-2 years of age groups than 2 years of age groups in goats.

2. Anorexia, distended abdomen with fluid splashing sound, dyspnoea, tachycardia, absence of ruminal motility and passing loose faeces were more frequently observed clinical signs in goats with acute ruminal acidosis.

3. The rumen liquor showed milky white colour, watery consistency, sour odour, reduced pH, absence of ruminal protozoa, more percentage of gram positive organisms, increased TVFA and MBRT in goats with acute ruminal acidosis.

4. Increased PVC, Hb, TLC and TEC were observed in goats with acute ruminal acidosis suggestive of systemic dehydration and haemoconcentration and blood gas analysis showed decreased blood pH, bicarbonate ions, VpCo2 with increased H^+ ions and base deficit in goats with acute ruminal acidosis.

5. Least mean recovery time ($\leq$ 75 hours) with normalisation of important bench mark parameters was observed in group III. Hence recommended as first line of treatment as life saving measures in acute ruminal acidosis of goats.

6. In absence of skilled help for surgical intervention therapeutic intervention with sodium bicarbonate as alkalizing agent, intraruminal antibiotic and oral Bufzone®, as rumen buffering agent as found in group I could be alternative treatment regimen for acute ruminal acidosis of goats.

Bibliography

VII. BIBLIOGRAPHY

ABHISHEK KUMAR and VERMA, S.P., 2003. Efficacy of Rumec powder in the treatment of acidotic goats. *Indian J. Vet. Med.*, **23**(2): 113-114

ABHISHEK KUMAR and VERMA, S.P., 2005. Changes in rumen liquor in experimental ruminal acidosis in goats. *Indian J. Vet. Med.*, **25**(2): 100-101

ALLISON, M.J., ROBINSON, I.M., BUCKLIN, J.A. and DOUGHERTY, R.W., 1972. *J. Anim. Sci.* **35**:259, Cited from TANWAR, R.K. and MATHUR, P.D., 1983a. Studies on clinical manifestations and therapy of experimentally induced acid indigestion in goats. *Indian J.Vet. Med*., **3**: 15-20

ALONE, S.D., ROAD, A.M., SARODE, D.B. and GHANWAT, D.V., 2005. Studies on biochemical profile in induced acid indigestion in goats. Round Table Conference on Rumenology. 5th Indian Veterinary Conference at Bikaner, *pp* 249-251

ANGELOV, G., NIKOLOV, Y. and ANGELOV, A., 1996. Changes in acid-base parameters, blood sugar and blood lactate, experimental acute rumen acidosis in sheep. *Indian Vet. J.*, **73**(3): 85

ARORA, N., TUFANI, N.A. and TARUN KUMAR., 2011. Grain overload in a buffalo and its therapeutic management. *Intas Polivet*, **12**(II): 315-317

ASHOK KUMAR VIHAN, V.S., CHOUDHARY, U.B., GUPTA, V.K. and TEWARI, H.A., 2005. Rumen disorders in goats under organised farming system - A retrospective study. Round Table Conference on Rumenology. 5th Indian Veterinary Conference at Bikaner, 221-236.

BARNETT, J.G.A. and REID, R.L., 1956. Studies on the production of volatile fatty acids from the grass by rumen liquor in an artificial rumen. *J. Agri. Sci.*, **48**: 315

BASAK, D.N., PAN, S. and CHAKRABARTI, A., 1993. Physico-chemical and microbial changes in rumen liquor of experimentally induced lactic acidosis in goats. *Indian J. Anim. Sci.*, **63**(3): 263-267

BENJAMIN, M.M., 1985. Outline of Veterinary Clinical Pathology. *Edn*. 3rd., Kalyani Publishers., Ludhiana, India.

BOLTON, J.R. and PASS, D.A., 1988. The Elimentary Tract. *In*: Clinicopathologic Princiles of Veterinary Medicine. *Edt.* Robinson, W.F. and Huxtable, C.R.R., Cambridge University, Cambridge, Press, *pp* 99-121

BOODOOR, P., SHIVAPRAKASH, B.V., KASARALIKAR, V.R. and DILIPKUMAR, D., 2010. Rumen impaction in bovines with indigestible foreign bodies and its surgical and therapeutic management. *Intas Polivet*, **11**(2): 184-188

BRAUN, U., RIHS, T. and SCHEFER, U., 1992. Ruminal lactic acidosis in sheep and goats. *Vet. Rec*. **130**: 343-349

BRITTON, R.A. and STOCK, R.A., 1986. Acidosis, rate of starch digestion and intake. *In*: Symposium Proceedings: Feed Intake by Beef Cattle. Oklahoma State University, *pp* 125-137

CAKALA, S., BORKOWSKI, T. and ALBRYCHT, A., 1974. *Polskie Arch. Wet*., **17**: 117, Cited from SHARMA, A.K., CHOUDHARY, C.K., ABHISHEK KUMAR, HEMANT KUMAR and SANJAY YADAV, 2010. Lactic acidosis in goats. *Indian Vet. J*., **87**: 1148-1150

CAO, G.R., ENGLISH, P.B., FILIPPISH, L.J. and INGLIS, S.S., 1987. Experimentally induced lactic acidosis in the goat. *Australian Vet. J*., **64**: 367-370

CHAKRABARTI, A., 2006. Textbook of Clinical Veterinary Medicine. *Edn*. 4th., Kalyani Publishers, Ludhiana, India

CHAPLIN, R.K. and JONES, G.A., 1973. *J. Dairy. Sci*., **56**: 671, Cited from TANWAR, R.K. and MATHUR, P.D., 1983a. Studies on clinical manifestations and therapy of experimentally induced acid indigestion in goats. *Indian J.Vet. Med*., **3**: 15-20

CHOUDHARY, S., MURALIDHARA, A. and RAVINDRANATH, B.M., 2011. Ruminal acidosis in small ruminants and its therapeutic management. *Intas Polivet*, **12** (II): 320-321

COOPER, R. and KLOPFENSTEIN, T., 1996. Effect of Rumensin® and feed intake variation on ruminal pH. *In*: Scientific Update on Rumensin® / Tylan® / Micotil® for the Professional Feedlot Consultant. Elanco Animal Health, Indianapolis, *pp* 1 -14

DARWIN, L., SURESH, R.V., THANGATHURAI, R. and DHANAPALAN, P., 2007a. Retrospective Study on Ruminal Acidosis in Goats. *Indian Vet. J.*, **84**: 432-433

DARWIN, L., SURESH, R.V., THANGATHURAI, R. and DHANAPALAN, P., 2007b. Rumen liquor profile examination in rumen acidosis in goats. *Indian Vet. J.*, **84**: 882-883

DAS, J., BEHERA, S.S. and PATI, S., 2011. Acute bloat in a goat and its surgical management by rumenotomy. *Intas Polivet*, **12**(II): 322-324

DAS, S.K. and MISHRA, S.K., 1991. Studies on blood composition during the onset and recovery phases of therapy in experimental rumen acidosis in goats. *Indian Vet. Med. J*. **15**: 275-279

DAS, S.K. and MISHRA, S.K., 1992. *Indian Journal of Animal Sciences*, **62**:243, Cited from SHARMA, A.K., CHOUDHARY, C.K., ABHISHEK KUMAR, HEMANT KUMAR. and SANJAY YADAV., 2010. Lactic acidosis in goats. *Indian Vet. J*., **87**: 1148-1150

DASH, P.K., MISRA, S.K. and MOHANTHY, G.P., 1972. Effect of acute indigestion on the rumen protozoa population and blood picture of indian cattle. *Indian Vet. J.*,**49**: 672-679

DESAI, V.F., BIJWAL, D.L., MODE, S.G., SADEKAR, S.Z. and DHORE, R.N., 1999. Rumen liquor profile following rumen lactic acidosis in calves. *Indian J. Anim. Sci.*, **69**(8): 586-587

DIRKSEN, 1965. Cited from JANI, R.G., JANI, B.M. and PATEL, P.R., 2001. Clinicopathological studies in ruminal acidosis in goats. *Indian J.Vet. Med*., **21**(2): 105

DIRKSEN, G., 1970. Acidosis. *In*: Physiology of Digestion and Metabolism in ruminants. *Edt.* Phillipson, A.T., Oriel Press, New Castle -Tyne, UK

DUNLOP, R.H. and HAMMOND, P.B., 1965. D-lactic acidosis of ruminants. Annals of New York Academy of Science, **119**: 1109

DUNLOP, R.H., 1972. Pathogenesis of ruminant lactic acidosis. *In*: advances in veterinary sciences and comparative medicine. Academic press, New York., *pp* 252-302

DUNLOP, R.W., 1971. Pathogenesis of ruminant lactic acidosis. *Adv. Vet. Sci. Comp. Med.,* **16**: 289

ELDIN, M.M., GHANEM, M.M., ABD EL, R.Y.M., EL-ATTAR, H.M. and EL-KHAIAT, H.M., 2014. Clinical, haematobiochemical and ruminal changes during the onset and recovery of induced lactic acidosis in sheep. *Biotechnol. Anim. Husbandry*, **30**(4): 647-659

ERDMAN, R.A., 1988. Dietary buffering requirement of the lactating dairy cattle: A review. *J. Dairy Sci.*, **71**: 3246-66

FRANK, E.R., 2002. Veterinary Surgery, CBS Publishers and Distributers, New Delhi., *pp* 235-236

FUBINI, S.L. and DUCHARME, N.G., 2004. Surgery of the ruminant fore stomach compartments. *In*: Farm animal surgery, Saunders, Missouri, *pp* 522

FULLER, 1989. A review : Probiotic in man and animals. *J. Appl. Bact.*, **66**: 365-378

GARRY, F., 1990. Diagnosing and treating indigestion caused by fermentative disorders. Symposium on bovine digestive disorders. *Vet. Med.*, **85**: 660

GHANEM, M.M., YOUSIF, H.M., ABD EL-GHANY, A.H., ABD EL-RAOF, Y.M., EL-ATTAR, H.M., 2015. Evaluation of pulmonary function tests with hemato-biochemical alterations Boer goats affected with klebsiella pneumoniae. *Benha Vet. Med. J.*, **29**(1):53-62

GLOCK, R.D. and DEGROOT, B.D., 1998. Sudden death of feedlot cattle. *J. Anim. Sci.*, **76**: 315-319

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

GONZALEZ, F.H.D., RUIPEREZ, F.H., SANCHEZ, J.M., SOUZA, J.C., MARTINEZ-SUBIELA, S. and CERON, J.J., 2010. Haptoglobulin and serum amyloid A in subacute ruminal acidosis. *Rev. Med. Vet. Zoot*., **57**: 159-167

GOZO, G.N., PLAIZER, J.C. and KRAUSE, D.O., 2006. Effects of grain induced subacute ruminal acidosis on ruminal lipopolysaccharide and inflammation in Holstein cows. *J. Dairy Sci*., **89**(1): 404

GOZO, G.N., PLAIZER, J.C. and KRAUSE, D.O., 2007. Ruminal lipopolysacharide concentration and inflammatory response during grain induced subacute ruminal acidosis in dairy cows. *J. Dairy Sci*., **90**: 856-866

GOZO, G.N., PLAIZER, J.C., KRAUSE, D.O., KENNEDY, A.D. and WITTENBERG, K.M., 2005. Subacute ruminal acidosis induces ruminal lipopolysaccharide release and triggers an inflammatory response. *J. Dairy Sci*., **88**: 1399-1403

GRAY, F.V., 1948. The absorption of VFA from the rumen. II. The influence of pH on the absorption. *J. Exp. Biol*., **25**: 135

HAJIKOLAEI, H.M.R., NOUR, M., SABERI, A.F. and JAFARI, D.A., 2006. Effects of experimentally induced ruminal lactic acidosis on blood pH, Bicarbonate and pCO2 in the sheep. *Pakistan J. Bio. Sci.*, **9**(10): 2003-2005

HARMON, D., BRITTON, R. and PRIOR, R., 1983. D(-) lactate metabolism in beef cattle. *Beef Cattle Rep*. *pp* 51-52

HENRY, R. J., 1963. Estimation of Glucose. Standard Methods of Clinical Chemistry

HRISTOV, A.N., IVAN, M., RODE, L.M. and MCALLISTER. T.A., 2001. Fermentation characteristics and ruminal ciliate protozoal populations in cattle fed medium- or highconcentrate barley-based diets. *J. Anim. Sci*., **79**: 515-524

HUBER, T.L., 1976. Physiological effects of acidosis on feedlot cattle. *J. Anim. Sci*., **43**(4): 902

HUNGATE, R.E., DOHGHERTY, R.W. BRYANT, M.P. and CELLO, R.M., 1952. *Cornell. Vet*., **42**.423, Cited from TANWAR, R.K. and MATHUR, P.D., 1983a. Studies on clinical manifestations and therapy of experimentally induced acid indigestion in goats. *Indian J.Vet. Med*., **3**: 15-20

HUNTINGTON, G.B., 1988. Nutritional problems related to the gastrointestinal tract. Acidosis. *In*: *Edt*. Church, D.C. The ruminant animal. Waveland Press, IL. *pp* 474-480

ISLAM, M.M., ANJUM, S., MODI, R.J. and WADHWANI, K.N., 2016. Scenario of livestock and poultry in India and their contribution to national economy. *Int. J. Sci. Env. Technol.*, **5**(3): 956-965

JAIN, N.C., 2000. Schalm's Veterinary heamatology *Edn*. 5th., Lea and Febiger Publisher, Philadelphia.,USA

JANI, R.G., JANI, B.M. and PATEL, P.R., 2001. Clinicopathological studies in ruminal acidosis in goats. *Indian J.Vet. Med*., **21**(2): 105

KANDER, M., DEPTA, A. and NIERADAKA, R., 2005. *Indian Vet. J.*, **82**: 370, Cited from ROHILLA, P.P., PATIL, N.V. and BOHRA, H.C., 2010. Effect of probiotic and nutrimix on growth and productivity of Marvari kids and goats. *Indian Vet. J.*, **87**: 367-369

KANDER, M., DEPTA, A., RYCHLIK, A. and BOMBA, A., 2000. Conference: The prospects of probiotics in prevention of and therapy of diseases of young. 11-14 October, High Tatras, Slavok, Republic, *pp* 57

KANEKO, J.J., HARVEY, J.W. and BRUSS, M.L., 1999. Clinical biochemistry of domestic animals. Harcourt Brace and Company, Asia Pvt. Ltd. Singapore.

KASARALIKAR, V.R., 2005. Primary ruminal acidosis in goats with particular reference to lactic acidosis. Ph.D. thesis submitted to Acharya N. G. Rajendra Agricultural University, Rajendranagar, Hydaerabad, India

KASARALIKAR, V.R., SINGARI N.A., HAFIZ, M.D., PRASAD, P.E. and KUMAR, S.P., 2007. Alterations in ruminal fluid and blood in acute ruminal acidosis of goats. *Indian J. Vet. Med.*, **27:** 111-114

KASARALIKAR, V.R., SINGARI, N.A., NALINI KUMARI, K. and PRASANNA KUMAR, S., 2012. Incidence of Acute Ruminal Acidosis in Goats in and around Bidar, Karnataka: A four year review. *Frontier J. Vet. Anim. Sci.*, **1**: 92-94

KLEEN, J.L., HOOIJER, G.A., REHAGE, J. and NOORDHUIZEN, J.P.T., 2003. Subacute ruminal acidosis: A review. *J. Vet. Med. A.*, **50**: 406-414

KOERS, W.C., BRITTON, R., KLOPFENSTEIN, T.J. and WOODS, W.R., 1976. Ruminal histamine, lactate and animal performance. *J. Anim.Sci.*, **43**: 684-691

KRAUSE, M.K. and OETZEL, G.R., 2006. Understanding and preventing subacute ruminal acidosis in dairy herds: a review. *Anim. Feed. Sci. Technol*., **126**: 215-236

KREHBIEL, C.R., BRITTON, R.A., HARMON, D.L., WESTER, T.J. and STOCK, R.A., 1995. The effects of ruminal acidosis on volatile fatty acid absorption and plasma activities of pancreatic enzymes in lambs. *J. Anim. Sci.*, **73**: 3111-3121

KROGH, N., 1959. *Acta. Vet. Scand*. **1**: 74, Cited from TANWAR, R.K. and MATHUR, P.D., 1983a. Studies on clinical manifestations and therapy of experimentally induced acid indigestion in goats. *Indian J.Vet. Med*., **3**: 15-20

KUMAR, A., 2010. Rumen protozoa in different species of livestock. *Indian Vet. J.*, **87**: 1166-1167

LAL, S. B., DWIVEDI, S.K., CELLY, C.S., SINGH, G.R., SHARMA, M.C. and SWAROOP, D., 1993. Biochemical and electrocardiographic changes in experimental rumen lactic acidosis in goats. *Indian J. Anim. Sci.*, **63**(9): 952-956

LAL, S.B., DWIVEDI, S.K., SHARMA, M.C. and SWARUP, D., 1989. Clinico-biochemical and microbial studies in rumen liquor in experimental acidosis in goats. *Indian J. Vet. Med.*, **9**(2): 81-85

LAL, S.B., DWIVEDI, S.K., SHARMA, M.C. and SWARUP, D., 1990. Influence of acidosis on blood biochemical values in goats. *Indian J. Vet. Med*., **40**: 125-128

LEAL, L.M.R., SOARES, P.C., CYRILLO, F.C. and BENECI, F.J., 2010. Influence of refrigeration on blood gas analysis of caprine venous blood. *Braz. J. Vet. Res. Anim. Sci*., 47 (2): 105-110

LINDINGER, M.I. and HEIGENHAUSER, G.J.F., 2008. Lactate is not the only physicochemical contributor to the acidosis of exercise. *J. Appl. Physiol*., **105**(1): 363-367

MACKIE, R.I., GILCHRIST, F.M.C., ROBBERTS, A.M., HANNAH, P.E. and SCHWARTZ, H.M., 1978. Microbiological and chemical changes in the rumen during the stepwise adaptation of sheep to high concentrate diets. *J. Agric. Sci. Camb.,* **90**: 241-254

MAHMOOD, A.K., KHAN, M.S., KHAN, M.A., KHAN, M.A., BILAL, M. and FAROOQ, U., 2013. Lactic acidosis in goats: prevalence, intra-ruminal and haematological investigations. *J. Anim. Plant Sci.*, **23**(6): 1527-1531

MEENA, V.K., BIHANI, D.K., ANKITA, S. and MEENA, S.K., 2016. Rumen acidosis in goat and its clinical management. An abstract in 34th Annual convention of Indian Society for Veterinary Medicine and National Symposium on "Newer approaches in diagnosis and management of animal diseases for sustainable health and production" cum workshop. *pp* 101

METKARI, S.M., SALABAT, ALI., RAJGURU, D.N. and MOHD. SALEEM., 2001. Management of experimentally induced lactic acidosis in goats. *Indian Vet. J.*, **78**(8): 692-694

MICHELL, B., 1990. Ruminant acidosis. *In Practice,* **12**: 245-249

NAGARAJA, T.G. and CHENGAPPA, M.M., 1998. Liver abscesses in feedlot cattle: a review. *J. Anim. Sci.,* **76**: 287-298

NARENDRA, R.M., THRIPHATY, S.B. and DAS, P.K., 1990. A note on the treatment of ruminal acidosis in calves. *Indian J. Vet. Med.*, **21** (2): 105

NAVARRE, B.C., BAIRD, A.N. and PUGH, D.G., 2002. Diseses of the gastrointestinal system. Sheep and goat medicine. Elsevier Publisher, *pp* 71

NEMA, R.K., TIWARI, S.K., ROY, S., GALDHAR, C.N. and BHANDEKAR, S.K., 2003. Studies on the simple indigestion in goats and its treatment with appevet. *Indian Vet. J.*, **80**: 564-566

NIKOLOV, Y., 2003. Biochemical alterations in rumen liquor, blood, cerebrospinal fluid and urine in experimental acute ruminal lactic acidosis in sheep. *Indian Vet. J.*, **80** (1): 36-39

NOCEK, J.E., 1997. Bovine acidosis: implications on laminitis. *J. Dairy Sci.*, **80**: 1005-1028

NOUR, M., ABUSAMAR, M.T. and HAGO, B.E.D., 1998. Experimentally induced lactic acidosis in Nubian goats: clinical, biochemical and pathological investigations. *Small Rum. Res*., **31**: 7-17

NOUR, M.S.M., 2006. Surgical treatment of experimentally induced lactic acidosis in Nubian goats. *J. Anim. Vet. Adv.*, **5**(1): 49-52

O' CONNER, J.J., 1985. Dollar's veterinary surgery, *Edn*. 4th., CBS Publishers and Distributors, New Delhi., *pp* 349-351

OWENS, F.N., SECRIST, D.S., HILL, W.J. and GILL, D.R., 1998. Acidosis in cattle: a review. *J. Anim. Sci.,* **76**: 275-286

PADMAJA, K. and PRAVEENA, G., 2011. Rumen acidosis in goats. *Intas Polivet,* **12**(II): 318-319

PAL, B., PRASAD, B., SHARMA, S.K. and WADHWA, D.R., 1994. *Indian J. Vet. Med.*, **14**: 62, Cited from SHUKLA, G.K., MAHESH KUMAR and SHARMA , S.P., 1999. Management of lactic acidosis in bovine calves with rumen liquor and herbal therapeutic agents. *Indian Vet. J.*, **76**: 428-431

PANCHASHEEL, 2013. Clinicopathological and therapeutic studies in acute ruminal acidosis of goats. M.V.Sc., thesis submitted to Karnataka Veterinary Animal and Fisheries Sciences University, Bidar, India

PARRAH, J.D., MOULVI, B.A., MALIK, H.U., BUCHOO and BILAL, S., 2010. A clinical study on ruminal acidosis in cattle. *Indian J. Vet. Med.*, **30**(2): 134-135

PATRA, R.C., LAL, S.B. and SWAROOP, D., 1993. Physico-chemical alterations in blood, cerebro-spinal fluid and urine in experimental lactic acidosis in sheep. *Res. Vet. Sci.*, **54**(2): 217-220

PATRA, R.C., LAL, S.B. and SWAROOP, D., 1996. Biochemical profile of rumen liquor, blood and urine in experimental acidosis in sheep. *Small Ruminant Res.*, **19**:177-180

PATRA, R.C., LAL, S.B. and SWAROOP, D., 1997. Therapeutic management of experimental ruminal acidosis in sheep. *Indian Vet. J.*, **74**(3): 237-240

PRADEEP KUMAR RAM, VERMA, S.P., ANUP KUMAR AGRAWAL and JAYACHANDRAN, C., 2007. Effect of severity of acidosis on ruminal activity in goats. *Indian J. Anim. Res*., **41**(4): 256-260

PRADHAN, N.R., MISRA, S.K. and PAN, S., 1988. *Indian J. Vet. Med.*, **8**: 66, Cited from DARWIN, L., SURESH, R.V., THANGATHURAI, R. and DHANAPALAN, P., 2007b. Rumen liquor profile examination in rumen acidosis in goats. *Indian Vet. J.*, **84**: 882-883

PRASAD, J., JOSHI, S.V. and REKIB, A., 1976. *Mahavet*., **3**:13 Cited from SHUKLA, G.K., MAHESH KUMAR and SHARMA , S.P., 1999. Management of lactic acidosis in bovine calves with rumen liquor and herbal therapeutic agents. *Indian Vet. J.*, **76**: 428-431

RADOSTITS, O.M., BLOOD, D.C. and GAY, C.C., 1994. Acute carbohydrate engorgement of ruminants (rumen overload) *In*: Veterinary Medicine. *Edn*. 8th., W. B. Saunders, Philadelphia, *pp* 262-269

RADOSTITS, O.M., BLOOD, D.C. and GAY, C.C., 1995. Veterinary Medicine. *Edn*. 8th., Blair-Tindall, London, *pp* 262-69

RADOSTITS, O.M., GAY, C.C., BLOOD, D.C. and HICHCLIFF, K.W., 2000. Veterinary Medicine. *Edn*. 9th., Book Power, Elsevier Science Limited, London (UK), *pp* 284-292

RADOSTITS, O.M., GAY, C.C., HINCHCLIFF, K.W., and CONSTABLE, P.D., 2007. Veterinary Medicine. A Text Book of Disease of Cattle, horses, Sheep, Pig and Goats. *Edn*. 10th., *pp* 318-321

RAGFAR (Reference Advisory Group of Fermentative Acidosis of Ruminants), 2007. A Review for Veterinarians and Nutritional Professionols, *pp* 3-56

RAHIMA, A., NAMBI, A.P., VIJAYKUMAR, G. and VAIRAMUTHU, S., 2012. Efficacy Of haemodialysis in goats affected with acute ruminal acidosis. *Indian Vet. J.*, **89**(6): 128-130

RANDHAWA, S.S., AHUJA, A.K. and RATHORE, S.S., 1989. Effect of lactic acidosis on microbial and biochemical changes in rumen liquor of buffalo calves. *Indian J. Vet. Med*., **9**: 1-7

RANDHAWA, S.S., SINGH, K.B. and MISRA, S.K., 1981. Some studies on biochemical changes in peracute lactic acidosis in crossbred calves. *Indian J.Anim. Health*, **20**(2): 87-89

REITMAN, S. and FRANKEL, S., 1957. Estimation of Aspartate amino-transaminase in serum. *Am. J. Clin. Pathol.*, **28**: 56.

ROGOSA, M., 1964. The genus Veillonella I. General, cultural, ecological and biochemical consideration. *J. Bacteriol*., **1**: 162-170

ROHILLA, P.P., 2005. Role of probiotic and prebiotic in livestock production. *Indian Vet. J.*, **29**: 101-103

ROHILLA, P.P., PATIL, N.V. and BOHRA, H.C., 2010. Effect of probiotic and nutria-mix on growth and productivity of Marwari kids and goats. *Indian Vet. J.*, **87**: 367-369

ROWE, J.B., 1997. Acidic gut syndrome: is it a problem for animal and humans? In: Recent advances in animal nutritionin Australia 97 (Carbett, J.L., Choct, M., Nolan, J.V. and Rowe, J.B. *Edn.*) University of New England, Armidale, Australia, *pp* 47-54

ROWE, J.B., WINSLOW, S.G. and MURRAY, P.J., 1989. Effect of basal dieght and feed additives on the susceptibility of sheep rumen fluid to lactic acid production. *Asian-Aust. J. Anim. Sci*., **2**: 346-348

RUSSELL, J.B. and HINO, T., 1985. Regulation of lactate production in Streptococcus bovis: a spiraling effect that contributes to rumen acidosis. *J. Dairy Sci.*, **68**: 1712-1721

SAHINDURAN, S., 2003. Ruminal acidosis in fallow deer a case report. *Indian Vet. J.*, **80**: 1169-1170

SANDHU, A.K., SAINI, A. and RANDHAWA, S.S., 2001. Haematobiochemical studies in healthy goats. *Indian Vet. J.*, **78**: 590-593

SCHALM, O.W., JAIN, N.C. and CARROL, C.J., 1975. Veterinary Heamatology, *Edn*. 4th., Lea and Febiger Publisher, Philadelphia, *pp* 21-48

SELVARAJ, P., SRINIVASAN, S.R., NAMBI, A.P. and DHANAPALAN. P., 2009. Efficacy of feed additives in management of ruminal acidosis in dairy cattle. *Indian J. Vet. Med.*, **29**:32-34

SEN, M.M., MISRA, S.K. and CHOUDHURI, P.C., 1982. Clinico-therapeutic aspects of acute ruminal acidosis in goats. *Indian J. Vet. Med.*, **2**(2): 25-32

SEN, M.M., MISRA, S.K. and CHOUDHURI, P.C., 1993. Blood chemical changes in acute experimental acidosis in Barbari goats. *Indian Vet. J.*, **70**(6): 515-518

SHANKARNARAYANA, G. and NAMBIAR, K.H., 1972. Physiological studies on liquor. *Cherion*, **1**: 45-47

SHARMA, A.K., CHOUDHARY, C.K., ABHISHEK KUMAR, HEMANT KUMAR and SANJAY YADAV, 2010. Lactic acidosis in goats. *Indian Vet. J.*, **87**: 1148-1150

SHIHABUDEEN, P.K., PILLAI, U.N., AJITKUMAR, S. and ALEX, N.P.C., 2003. Haematological changes in experimental ruminal acidosis. *Indian J. Vet. Med.*, **23**: 93-95

SHUKLA, G.K., MAHESH KUMAR and SHARMA , S.P., 1999. Management of lactic acidosis in bovine calves with rumen liquor and herbal therapeutic agents. *Indian Vet. J.*, **76**: 428-431

SINGH, H., TANWAR, R.K., SINGH, A.P. and GAHLOT, A.K., 2001. Haemato-biochemical profiles in indigestion in goats. *Indian Vet. J.*, **78**: 130-132

SINGH, K.P., SINGH, R.V., PRANEETA, S., SINGH, J.P. and SINGH, S.K., 2016. Theraputic management of ruminal acidosis in goats. An abstract in 34th Annual convention of Indian Society for Veterinary Medicine and National Symposium on “Newer approaches in diagnosis and management of animal diseases for sustainable health and production” cum workshop. *pp* 101

SINGH, N., AKBAR, M.A., KUMAR, R. and KHANNA, B.M., 1996. *Indian J. Vet. Med.*, **20**: 115, Cited from SINGH, H., TANWAR, R.K., SINGH, A.P. and GAHLOT, A.K., 2001. Haemato-biochemical profiles in indigestion in goats. *Indian Vet. J.*, **78**: 130-132

SLYTER, L.L., 1976. Influence of acidosis on rumen function. *J. Anim. Sci.*, **43**(4): 910-929

SMITH, B.P., 1996. Large animal Internal Medicine. The C.V. Mosby Company, St. Louis, Missouri.

SNEDECOR, G.W. and COCHRAN, W. G., 1994. *Statistical Methods. Edn.* 8th., Affiliated East - West Press, Pvt. Ltd., New Delhi.

SOBIECH, P., STOPYRA, A., KULETA, Z., ZBANYSZEK, M. and MILEWSKI, S., 2005. Acid-base balance parameters of arterial, venous and capillary blood in sheep. *Bull. Vet. Inst. Pulawy*, **49**: 125-127

SOMASHEKAR REDDY, B., SUDHAKAR REDDY, B., SHIVAKUMAR, R.V. and DHANUNJAYA REDDY. R., 2016. An outbreak of acute rumen acidosis in sheep and its management. An abstract in 34th Annual convention of Indian Society for Veterinary Medicine and National Symposium on "Newer approaches in diagnosis and management of animal diseases for sustainable health and production" cum workshop, *pp* 72

TANWAR, R.K. and MATHUR, P.D., 1983a. Studies on clinical manifestations and therapy of experimentally induced acid indigestion in goats. *Indian J.Vet. Med*., **3**: 15-20

TANWAR, R.K. and MATHUR, P.D., 1983b. Biochemical and microbial changes in experimentally induced rumen acidosis in goats. *Indian J. Anim. Sci.*, **53**(3): 271-274

TANWAR, R.K., MATHUR, P.D., SHARMA, S.N. and GAHLOT, A.K., 1983. Haematological and biochemical studies of blood in experimentally induced rumen acidosis in goats. *Indian J. Anim. Sci.*, **53**(12): 1296-1299

TAWHEED, A.S., DIGRASKAR, S.U., BORIKAR, S.T., SYED, A.M. and NITHIN, B.S., 2017. Emptying of rumen in acute ruminal acidosis with intubation in goats. An abstract in 35th Annual convention of Indian Society for Veterinary Medicine and National Symposium on "Innovative techniques, emerging issues and advancement in veterinary medicine to meet the challenges: present and the feature" cum workshop, *pp* 254

THACHIL, A.J., 2000. Clinico-therapeutic study of ruminal lactic acidosis in goats. M.V.Sc., thesis submitted to Kerala Agricultural University, India

TREMERE, A.W., MERRILL, W.G. and LOOSLI. J.K., 1968. Adaptation to high concentrate feeding as related to acidosis and digestive disturbances in dairy heifers. *J. Dairy Sci*., **51**(7): 1065-1072

TRIPATHY, S.B. and MISHRA, D.N., 1972. Theraputic efficacy of Rumbion in experimental acid indigestion in goats. *Pashudhan*, **7**: 4-8

TUFANI, N.A., MAKHDOOMI, D.M. and HAFIZ, A., 2013. Rumen acidosis in small ruminants and its management. *Iranian J. Applied Anim. Sci.*, **3**(1): 19-24

TYAGI, R.P.S. and SINGH, J., 2004. The digestive system. Ruminant Surgery, CBS Publishers and Distributors, New Delhi, *pp* 190

ULLAH, H.A., KHAN, J.A., KHAN, M.S., SADIQUE, U., SHAH, M. IDREES, M. and SHAH. Z., 2013. Clinico-therapeutical trials of Lactic Acidosis in Small Ruminants. *J. Anim. Plant Sci.*, **23**(1): 80-83

UNDERWOOD, W.J., 1992. Rumen lactic acidosis. Part- II: Clinical signs, diagnosis, treatment and prevention. *Compound Cont. Practice*, **14**: 1265-1270

WANKHEDE, G.D, SWAMI, S.B., BORIKAR, S.T., APTE, N.P. and BORSE, M.R., 2016. Acidic indigestion with worm infestation in goat - a case report. An abstract in 34th Annual convention of Indian Society for Veterinary Medicine and National Symposium on "Newer approaches in diagnosis and management of animal diseases for sustainable health and production" cum workshop, *pp* 96

WILDENTHAL, K., MIERZWIAT, D.S., MYERS, R.W. and MITCHELL, J.H., 1968. Effect of acute lactic acidosis on left ventricular performance. *Am. J. Physiol*., **214**: 1352-1359

Abstract

STUDIES ON ACUTE RUMINAL ACIDOSIS IN GOATS WITH SPECIAL REFERENCE TO THERAPEUTIC AND SURGICAL MANAGEMENT

Student **June 2017** **Major Advisor**
Bhagavantappa B. **Dr. Shivashankar M. Usturge**

ABSTRACT

The research was conducted in eighteen clinical cases of goats suffering from acute ruminal acidosis at Department of Veterinary Surgery and Radiology, Veterinary College, Bidar. They were randomly divided into three groups of six animals in each group. The treatment protocol for group I included oral and intravenous administration of sodium bicarbonate along with oral Bufzone powder, in group II included rumenotomy followed by intravenous administration of sodium bicarbonate and oral Ecotas bolus and in group III included rumenotomy followed by intravenous administration of sodium bicarbonate and cud transplantation. In the present study overall prevalence of ruminal acidosis was recorded 1.60 per cent out of total number of goat cases presented. The higher occurrence was recorded in females than males, highest in 1-2 years of age groups followed by less than 1 year and least in more than 2 years of age groups in goats. The source of carbohydrate rich food was highest for rice, followed by wheat, jawar, vegetables grains, maize and fruits and occurrence of disorder was found highest in the monsoon season followed by summer, post monsoon and least in winter season in goats. The more frequent clinical signs observed were complete anorexia, distended abdomen with fluid flashing sound, diarrhea, dyspnoea and tachycardia in acidotic goats. The physiological parameters like heart rate and respiratory rate were significantly elevated in the acidotic goats. The physico-chemical properties of ruminal fluid showed significant changes in almost all the parameters. The ruminal fluid showed milky white colour, watery consistency, sour odour, decreased pH, absence of ruminal protozoa, increased TVFA, increased MBRT, absence of SAT and more percentage of gram positive organisms were observed. Haematological, biochemical and blood gas analysis showed significant increased Hb, PCV, TEC, TLC, glucose, AST, H^+ and base deficit values whereas, decreased VpH and HCO^-_3 values were seen. Least mean recovery time ($\leq$ 75 hours) with normalisation of important bench mark parameters was observed in group III, hence recommended as first line of treatment as life saving measures in acute ruminal acidosis of goats. In absence of skilled help for surgical intervention therapeutic intervention with sodium bicarbonate as alkalizing agent, intraruminal antibiotic and oral Bufzone®, as rumen buffering agent as found in group I could be alternative treatment regimen for acute ruminal acidosis of goats. In this study rumen lavage with normal saline by rumenotomy in combination with transfaunation from a healthy animal yielded better results without any post-operative complications. Ancillary therapy with balanced electrolyte solutions, streptopencillin, thiamine hydrochloride, chlorphenaramine maleate and calboral augmented the recovery without any complications in goats of acute ruminal acidosis in all the three groups. Surgery is considered as emergency therapy to save the life of goats.