

**DIAGNOSTIC AND THERAPEUTIC STUDIES ON
INTESTINAL OBSTRUCTION IN CALVES**

THESIS

BY

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OF

**DOCTOR OF PHILOSOPHY IN VETERINARY SCIENCE
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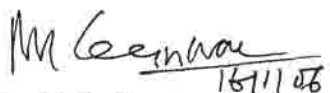
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CERTIFICATE I

This is to certify that the thesis entitled "**Diagnostic and therapeutic studies on intestinal obstruction in calves**" submitted in partial fulfillment of the requirements for the award of the degree of **Doctor of Philosophy in Veterinary Science** in the subject of **Veterinary Surgery and Radiology** of CSK Himachal Pradesh Krishi Vishvavidyalaya, Palampur (H.P.) is a *bonafide* research work carried out by **Dr. Adarsh Kumar** son of Shri Baldev Raj Kanaujia under my supervision and that no part of this thesis has been submitted for any other degree or diploma.

The assistance and help received during the course of this investigation have been fully acknowledged.



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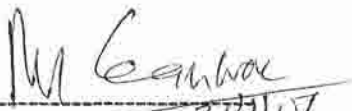
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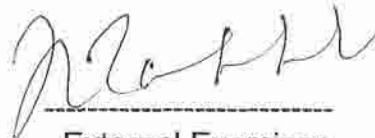
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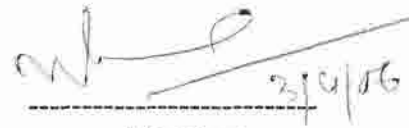
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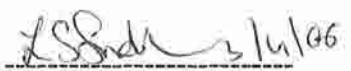
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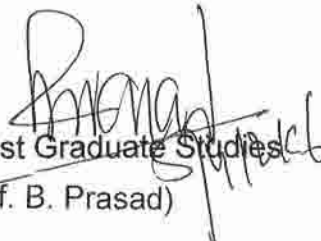
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**Dedicated to my parents, without
whom none of this would have
been even possible**

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ABBREVIATIONS

Abbreviation	Meaning
°F	Degree Fahrenheit
%	Per cent
/	Per
@	At the rate of
-ve	Negative
+ve	Positive
<	Less than
>	Greater than
µl	microlitre
ALKP	Alkaline Phosphatase
ALT	Alanine aminotransferase
ANOVA	Analysis of variance
APC	Atrial premature complexes
AST	Aspartate aminotransferase
b.i.d.	<i>bis in die</i> (Twice a day)
BUN	Blood urea nitrogen
BW	Body weight
Ca	Calcium
CK	Creatinine Kinase
Cl ⁻	Chloride anion
cm	Centimeter
Co.	Company
CRT	Capillary refill time
cu mm	Cubic millimeter
d	Day(s)
dl	Decilitre
DLC	Differential leucocytic count
ECG	Electrocardiography
Ed(s).	Editor(s)
EDTA	Ethylenediaminetetraacetic acid
<i>et. al.</i>	<i>et alli</i> (and others)
<i>etc.</i>	<i>et cetera</i> (and the rest)
F	Female
g	Gram(s)
H ₂ O	Water
Hb	Haemoglobin
HCO ₃ ⁻	Bicarbonate ion
HEX	Hexosaminidase
Hg	Mercury
<i>i.e.</i>	<i>Id est</i> (that is)
IM	Intramuscularly
Inj.	Injection
IU	International units

IV	Intravenously
K ⁺	Potassium cation
Kg	Kilogram
L or l	Litre
LDH	Lactate dehydrogenase
M	Male
mEq	Milli equivalents
mg	Milligram
Min	Minute(s)
ml	Millilitre
mm	Millimeter
mmol	Millimole
MOF	Multiple organ failure
mV	Millivolt
n	Number
Na ⁺	Sodium cation
NSAIDS	Non-steroidal anti-inflammatory drugs
p.	page
p.r.n.	<i>pro re nata</i> (as needed)
P<0.01	Statistical significance at 1% level
P<0.05	Statistical significance at 5% level
PCV	Packed cell volume
pH	P(otential of) H(drogen)
PO	<i>Per os</i> (Orally)
pp.	Pages
RBC	Red blood cells
RES	Reticulo endothelial system
rpm	Rotations per minute
S.E.	Standard error of mean
sec	Second(s)
SIRS	Systemic inflammatory response syndrome
<i>sp.</i>	Species (singular)
<i>spp.</i>	Species (plural)
TC	Time constant
TEC	Total erythrocytic count
TLC	Total leucocytic count
TPR	Total protein
<i>viz.</i>	<i>videlicet</i> (namely)
WBC	White blood cells

CHAPTER - I

INTRODUCTION

Cattle rearing is a major source of livelihood for the marginal farmers of Himachal Pradesh. About 91.5% families in this hilly state are rearing one type of livestock or other (1991 census) thus represents a significant individual financial investment. Hybridization and upgrading of Zebu cattle in combination with conventional rearing system has created some concurrent problems and one of the major problems in cross bred cattle is the obstruction of intestinal tract. Intestinal obstruction is a trivially life threatening abdominal disorder of large ruminants, which require prompt investigation and treatment. The acute abdomen arising out of intestinal obstruction represents a significant workload. Furthermore, patients with intestinal obstruction have a significant morbidity and mortality affecting the economy of the farmers.

Intestinal obstruction can be simple or strangulated in nature and presents a challenge for veterinarians. Once the nature of the condition has been ascertained, decision regarding prognosis and treatment can be made. Mortality is inevitable if the patient is not attended in time. The complex pathophysiological changes that occur during gastrointestinal disturbances in ruminants are remarkably different than those in simple stomached animals. Most of the studies conducted in ruminants are limited to the analysis of blood following intestinal obstruction. Hence it was considered worthwhile to study changes in relation to alterations in peritoneal and ruminal fluid in cattle during and subsequent to treatment of proximal simple and strangulated intestinal obstruction. Cytological and biochemical analysis of the peritoneal fluid is an important indicator in determining the severity and duration of obstruction. Since it is difficult to

determine the onset of intestinal obstruction in clinical cases therefore it is necessary to create various models of intestinal obstruction, to determine the time changes that take place following obstruction. The effect of intestinal obstruction varies widely, not only among the various species but within the same species as well, depending on the type and site of obstruction (Radostits *et al.*, 1994). Most cases of intestinal obstruction are chronic in nature which may or may not respond to treatment either due to lack of proper diagnosis or advanced stage of obstruction.

Thorough understanding of secondary pathophysiologic events following obstruction of the gastrointestinal tract, coupled with a systemic evaluation of each patient, facilitates interpretation of clinical findings. With this information, management decisions can be made about the need for surgical intervention. Most forms of intestinal obstruction result in depletion of the body's fluid compartments producing hypovolemic shock. Manifestation of clinical signs in animals with acute abdominal disease often includes secondary changes brought about by hypovolemic shock plus a number of specific variables. First, the location of lesion, the more cranial is the location, the more rapid and severe onset of clinical signs occurs. A second variable influencing outward signs of pain and shock in animals is the amount of damaged tissue. In general, greater the amount of damaged tissue, the more dramatic and fulminant clinical signs are manifested. The third variable is the type of intestinal accident involved. With simple obstructions, the mesenteric blood supply is intact but the lumen is severely occluded so severity of clinical pathology is late to develop in comparison to strangulated obstructions which imply both luminal occlusion and compromised vascular supply.

Various impediments such as partially understood etiology, ignorance and lack of awareness on the part of farmer and indiscriminate use of purgatives without proper investigations results in the deterioration of the condition which ultimately lead to death of the animals. In these cases, the importance of identification of obstruction and timing of the intervention performance in regard to patient's survival is explicitly the principal and life saving concern. Surgery alone may not be beneficial in many of the cases unless it is supplemented with specific supportive therapy. The supplementation of fluids has to be designed according to the specific requirement of the affected animal due to its altered physiological status following obstruction.

Medical records of Veterinary Clinical Complex and surrounding areas, indicates that intestinal obstruction is a frequent presentation to practioners. In large animal surgery, it's always been considered as an exigent presentation. Although some information on the clinical manifestations, diagnosis and pathophysiology of simple and strangulated intestinal obstruction in ruminants is available (Pearson, 1971; Papadopoulos *et al.*, 1985b; Avery *et al.*, 1986; Braun *et al.*, 1989b, Constable *et al.*, 1997, Dennison *et al.*, 2002; Anderson and Ewoldt, 2005), but detailed pathophysiological alterations and treatment converging at logical end is lacking in bovines.

Keeping in view the significance of the problem and paucity of work available on diagnostic, prognostic and therapeutic aspects, the present study was experimentally undertaken in cow calves to study the simple and strangulated obstruction of cranial jejunum. The study also includes the details pertaining to management of clinical cases of intestinal obstruction. The result of this study will be helpful in correlating the findings with specific obstructions of small intestine

and also have direct application in the field, thereby reducing the economic losses to the livestock owners.

The proposed study was conducted with the following objectives

1. To record clinical, hematological and biochemical alterations in blood, ruminal and peritoneal fluid following creation of high simple and strangulated intestinal obstruction in calves.
2. To compare the pathophysiological alterations following simple and strangulated intestinal obstruction in calves.
3. To correct surgically the high simple and strangulated intestinal obstruction and to formulate the suitable therapeutic regimen.
4. To apply and recommend the diagnostic and therapeutic strategy in clinical cases of intestinal obstruction.

CHAPTER - II

REVIEW OF LITERATURE

Mechanical obstruction arises from physical barrier to the passage of intestinal contents. Firstly, simple obstruction occurs when only the intestinal lumen is occluded. This can sometime lead to another form of mechanical obstruction called strangulation. This occurs when the blood supply is impaired resulting in necrosis of the bowel wall (White *et al.*, 1980).

The relevant review is summarized under following heads

2.1 Experimental model of intestinal obstruction

2.2 Incidence

2.3 Etiology

2.4 Clinical signs

2.5 Haemato -biochemical changes following intestinal obstruction

2.5.1 Haematological changes

2.5.2 Biochemical changes

2.6 Peritoneal fluid analysis

2.7 Ruminal fluid analysis

2.8 Microbiological studies

2.9 Electrocardiographic studies

2.10 Histopathological studies

2.11 Conservative and surgical management of intestinal obstruction

2.1 EXPERIMENTAL MODEL OF INTESTINAL OBSTRUCTION

Experimental creation of intestinal obstruction has been tried by many authors to simulate the clinical cases in different species of animals. Cohn and Atik (1961) used closed loop technique to study intestinal obstruction in dogs. Hammond *et al.* (1964) produced obstruction of duodenum in calves by passing inflated rubber balloon into the lumen. Upper and lower intestinal stricture in calves were created by using a woven cotton tape passed through polyvinyl tubing and placed firmly over duodenum (Hammond *et al.*, 1964).

Yale (1969) divided the proximal and distal ends of intestine by placing a layered suture closure at each end. The occlusion of the duodenum, colon and ileum in horses has been produced (Datt and Usenik, 1975) surgically by placing the rubber tube in loop fashion. Comparative studies on strangulated intestinal obstruction in bovines and equines have been described (Singh, 1971). Assorted obstructions like invagination of small intestines, invagination of ileum into colon, obstruction of duodenum and ileum, volvulus, adhesions and constriction of lumen (Ruthkowiak, 1973) have been studied.

Krishnamurthy *et al.* (1980) occluded both arterial and venous supply of jejunum to produce strangulated obstruction in buffalo calves. Experimental endotoxemia was studied by Moore *et al.* (1981) following strangulated intestinal obstruction by placing loose umbilical tape ligatures around arterial and venous jejunal loops. Strangulated obstruction model of the jejunum was produced by ligating the venous channels of the segment and keeping the arterial supply intact in buffalo and cow calves (Sahay and Kohli, 1983). Experimental models of ischemic and hemorrhagic strangulated intestinal obstruction in horses (Sullins *et*

al., 1985) and goats (Parvathamma, *et al.*, 1992) have been established. Papadopoulos^{*et al.*} (1987) used a constricted ligature consisting of flexible plastic tubing loosely placed around the bowel in double loop fashion with plastic discs on the interior and exterior of the abdominal wall. Strangulated Intestinal obstruction was induced by occluding the superior mesenteric veins to study the pathophysiology in mongrel dogs (Shikata *et al.*, 1989).

Ligation of duodenum (Avery *et al.*, 1986) at a level distal to pylorus and proximal to common bile duct through a paracostal incision was studied in cow. Pyloric obstruction (Basu, 1987) was induced in bovine calves to simulate simple intestinal obstruction. Strangulated obstruction of jejunum was created experimentally in ponies (Freeman *et al.*, 1988a) by clamping jejunal segments and/or arteries and veins.

Ischemic and hemorrhagic strangulated obstructions of jejunum were created in buffalo calves (Tank and Parsania, 1991) to mimic the clinical situations of obstructions. Pathophysiological effects of venous strangulated obstruction of the jejunal and colonic segments were studied in horses (Ruggles *et al.*, 1993) at different time intervals. Makhdoomi (1994) and Singh *et al.* (1997b) have evaluated the role of sepsis and dehydration by ligating the mesenteric veins in buffalo calves.

Falcrios *et al.* (2002) created an intra luminal intestinal obstruction by placing a latex balloon inflated to a pressure of 40 mm Hg and studied the microvascular perfusion in mucosal, submucosal, muscular and serosal layers of equine intestine. Transient intestinal ischemia was produced by clamping the aorta

at subrenal level and above the branching of the inferior mesenteric artery (Lammers *et al.*, 2003) to study the inflammatory parameters.

2.2 INCIDENCE

Medical records of cattle admitted to 17 Veterinary Medical Teaching Hospitals in North America were analyzed and 336 cattle with intestinal obstruction were identified (Constable *et al.*, 1997). In a span of 30 years, out of 336 cattle 281 had small intestinal, 7 had ileocolic, 12 had caecocolic and 36 had colocolic intussusceptions. Among the 51 calves (up to the age of three months) admitted to Medical Animal Clinic of the University of Munich (Doll *et al.*, 1998) from 1986 to 1994, the incidence of intussusception was Caecocaecal (n=12), caecocolic (n=22), ileiocaecocolic(n=6), ileocaecal (n=8) and jejunoileocolic (n=3). The most important clinical signs recorded were scanty faeces, that often contained blood and/or mucous, and the presence of hard viscus upon abdominal palpation (Doll *et al.*, 1998). Pearson and Pinsent (1977) reviewed the diagnosis and treatment in 100 clinical cases of intestinal obstruction in cows.

Dennison *et al.* (2002) reviewed the data of four years (1997-2000) and found that 22 cases in dairy cattle had acute intestinal disorder characterized by intraluminal hemorrhages and obstruction of small intestine. He named this disease as Hemorrhagic bowel syndrome. From the Medical records of Kansas State University (1967-1992), 35 cases of small intestine volvulus were reported (Anderson *et al.*, 1993). Surgical correction was performed on 32 cattle, and 17 of these cattle were discharged from the hospital. The survival rate for dairy cattle (63 per cent) was significantly higher than survival rate for beef cattle (22 per cent). Hoogewijs *et al.* (2004) reported 900 cases of intestinal obstruction (1983-

2003) presented in Ghent University Large Animal Clinic, Belgium. Substantially good number of cases pertaining to intestinal obstruction in cattle has been reported from India (Rathore *et al.*, 1977, Dixit *et al.*, 1975; Kumar *et al.*, 1980; Tayal *et al.*, 1986; Singh *et al.*, 1990; Dhaliwal *et al.*, 1992; Singh, 1995; Kanwar *et al.*, 1996; Shinde, 1996; Sarma *et al.*, 1996; Padile *et al.*, 2002; Ramprabhu *et al.*, 2002; Saini and Anand, 2002; Kumar *et al.*, 2003; Sharma *et al.*, 2003).

Thirty cases of caecocolic and caecocaecal intussusception in horses were recorded in a span of 30 years at University of Pennsylvania (Martin *et al.*, 1999). The range of age of these horses was 7 months to 30 years, but 63 per cent were < or = 3 years. Twenty six horses had acute to subacute disease and 4 had a chronic wasting disease. Surgery was performed on 24 horses, 15 horses survived and 9 horses were euthanized following surgery because of peritonitis, irreducible intussusception and rupture of gut. Gayle *et al.* (2001) reported 9 cases of strangulated intestinal obstruction in horses. The ileum and jejunum were strangulated in 8 horses, where as in one horse the small intestine and the left ascending colon were incarcerated in a rent of caecocolic fold. Twenty eight cases of ileal impactions in horses were studied to evaluate the seasonal influence, signalment, type of hay consumed, clinical exam findings and outcome of surgery. He concluded that ileal impactions can successfully be reduced by celiotomy and extraluminal massage and injection techniques to soften the ingesta. Changes in weather and feeding practices in the autumn may account for an increased risk of ileal impactions in southeastern United States (Hanson *et al.*, 1998).

Morton and Blikslager (2002) reviewed 92 cases of horses from 1994-2001 in North Carolina University, the study included surgical and post operative factors influencing short term survival of horses following small intestinal resection.

2.3 ETIOLOGY

The cause of intestinal obstruction is currently unknown, and no consistent predisposing factor has been identified. The occurrence of intestinal obstruction in large animals has though been related to many conditions.

Simple obstruction in cattle has been commonly related to formation of phytobezoars (Clem and Johnson, 1977) and can occur due to ingestion of indigestible fibres of onion grass (*Romulea rosea*) a dominant plant in pastures during lean season (Pitt, 1976). The undigested leaf of *Acacia sp.* has produced intestinal obstruction in langur monkeys (Ensley *et al.*, 1982). Intestinal torsion in a cow was apparently caused by the administration of acaprin as a babesicide (Vanzini *et al.*, 1981). Feeding of animals with large quantities of dry hydrophilic fibre sources, such as psyllium husk or guar gum resulted in intestinal obstruction (Struthers, 1986). Two cases of acute obstruction of the small intestine by solid aggregations of wood splinters were described in horses which had the habit of wood chewing (Green and Tong, 1988).

Hemp bedding used for animals has also been associated with intestinal obstruction, its ingestion in large quantity absorbed large volumes of intestinal fluid and has proved intractable to peristalsis (Green, 1996; Smith and Papworth, 1996). Fatal intestinal obstruction with ingestion of uncut maize stalks has been reported (Han and Li, 1995). Feeding of bamboo leaves was a common history in

cases of intestinal obstruction in giant pandas because of raw leaves (Chen *et al.*, 1998).

Small intestinal obstruction in 4 cows was reported due to a fibrous band extending from the free edge of the greater omentum to the ovary or the tip of uterine horn (Richardson, 1984). Persistent round ligament of the liver of cow resulted in the small intestinal obstruction (Duchrame *et al.*, 1982). The trypanosome was observed in blood smears (Kalra *et al.*, 1984) of a cow with intestinal obstruction but its significance was unknown. Allen *et al.* (1989) reported ganglioneuroma as a cause of small intestinal obstruction in the horse. Colic of parasitic origin in horses (Kopf, 1987) was attributed to embolism and thrombosis caused by *Strongylus vulgaris*, intestinal obstruction caused by masses of roundworms (*Parascaris equorum*) and cases of peritonitis following intestinal rupture due to roundworm lesions.

Various tumors like intestinal lipoma (Singh *et al.*, 1990) in cows, pedunculated lipomas in horses (Blikslager *et al.*, 1991; Edwards and Proudman, 1994), hamartomatous polyp (Colbourne *et al.*, 1996) in horses and lymphoma (Ljiljana *et al.*, 2003) in cattle were found to be associated causative factors in producing intestinal obstruction. Entrapment of intestine within the mesenteric rents resulted in intestinal obstruction in horses (Gayle *et al.*, 2000a). Prenatal intussusception in calf has also been reported to be caused by ingestion of excessive amount of amniotic fluid resulting in late natal death (Astiz Blanco *et al.*, 2004). Intraperitoneal bacterial wall lipopolysacchride caused intussusception in mice by disturbing the gastrointestinal motility (Linz *et al.*, 1998). As much as 25.9

per cent animals suffered intussusception after intra peritoneal injection of lipopolysacchride of *E. Coli* and *Salmonella*.

2.4 CLINICAL SIGNS

The most important clinical signs of intestinal obstruction were scanty faeces, that often contained blood and/or mucous, and the finding of hard viscus upon abdominal palpation (Doll *et al.*, 1998). Haemorrhagic bowel syndrome due to intestinal obstruction was characterized by acute clinical signs like profound depression, decreased milk production, tachycardia, ruminal stasis, abdominal distension and dark clotted blood in faeces (Dennison *et al.*, 2002).

The bovines suffering from intestinal obstruction usually exhibited signs of colic characterized by forceful kicking at belly with hind legs, frequent sitting and standing up, paddling of limbs, stretching in recumbency, arching of back (Wheat, 1947; Shields, 1965; Pearson, 1971; Pearson and Pincent, 1977; Bhokre and Deshpande, 1987; Anderson *et al.*, 1993).

Camelids demonstrated clinical signs like kicking at the abdomen, thrashing, rolling, restlessness, lying down and getting up frequently, vocalizing, grinding of teeth, straining to urinate and defecate, flagging the tail and lying in abdominal cush position (Anderson, 1996). The nature and intensity of clinical signs following intestinal obstruction varied in different species of animals (Radostits *et al.*, 1994) and also dependent upon the site, duration and type of obstruction (Edwards and Proudman, 2001). Complete obstruction of the intestine in ruminants produced similar clinical signs irrespective of cause. In general, cranial obstruction with compromised vascular supply incited acute clinical signs and disease (Singh *et al.*, 1995). Sheep showed signs of greater severity with

obstructed duodenum (Coker and Dzuik, 1968) than those affected with omaso-abomasal obstruction. Intensity of colicky signs was more in strangulated obstruction than in simple obstruction (Singh *et al.*, 1975).

Complete ruminal impaction and reduced ruminal movements have been reported (Brown and Parkes, 1961; Pearson, 1971) in animals following intestinal obstruction. Tympanitic resonance sound on right paralumbar fossa has been used as an aid in diagnosing intestinal obstruction in adult cattle (Smith *et al.*, 1982). Mild tympany, impaction and distension of abdomen was recorded (Makhdoomi, 1994) in buffalo calves following intestinal obstruction. Accumulation of ingesta in higher segment of obstructed intestine resulted in perceptible distension of abdomen (Dzuik and Usenik, 1964, Hammond ^{*et al.*}, 1964; Pearson and Pinsent, 1977) however, Papadopoulos *et al.* (1985a) advocated that abdominal distension may not be present in all the animals suffering from intestinal obstruction. Sharma (1999) observed unilateral right abdominal distension in cow calves in experimentally induced strangulated intestinal obstruction.

The fluid, gas and ingesta that accumulates proximal to the obstruction arise from several sources including saliva, gastric secretions, swallowed air, pancreatic secretions, bile, gas from bacterial fermentation, ingested fluid and solids and secretions from intestinal mucosa (Allen *et al.*, 1986). The affected animals showed severe dehydration and depression (Pearson and Pinsent, 1974). Increased skin tenting time (14-16 sec) was recorded by Makhdoomi (1994) in buffalo calves. Papadopoulos ^{*et al.*} (1985a) observed skin tenting of 4-6 sec on the very first day after creation of high simple and strangulated obstruction and just

before death it was 20 sec. Increase in capillary refill time following simple intestinal obstruction in cattle has been reported (Papadopoulos^{et al.}, 1985a).

Amendment in defecation is distinctive diagnostic sign in intestinal obstruction. Primarily during colic stage small faecal balls were passed (Pearson, 1971). Scanty stools or mucous in the rectum were palpated on per rectal examination (Anderson *et al.*, 1993). The last faecal material passed by the bovines was more mucoid and in some cases consisted of only mucous plugs (Brown and Parkes, 1961 and Radostits^{et al.}, 1994) which gradually changed to foul smelling, tarry or dark hard pellets consisting mucous and mucosal shreds with failure to void the faeces (Doll *et al.*, 1998). Faecal material was not evident (Constable *et al.*, 1997) in adult cattle affected with intussusception. Corke and Glenister (2001) reported that, the epithelial cells of the villi become swollen and slough into the intestinal lumen following vascular occlusion. This process advanced from the tip to the base of the villus, and at the end of a period of total ischemia lasting 120 minutes, there was complete destruction of the villi as well as the upper portions of the crypts.

Fickle changes have been seen in body temperature in ruminants following intestinal obstruction. Pearson (1971) and Papadopoulos^{et al.} (1985a) reported that in some cases the temperature became subnormal while Datt and Usenik (1975) in a similar experimental study in horses observed increase in temperature. Like wise hyperthermia was reported in bovines following intestinal obstruction (Murthy *et al.*, 1983).

Tachycardia and increase in pulse rate was observed in cattle suffering from intestinal obstruction (Anderson *et al.*, 1993). Elevation in heart rate during

the last stages of intestinal obstruction has been reported in cattle (Singh, 1971, Papadopoulos ^u ^{e^t al.}, 1985a) and buffaloes (Singh *et al.*, 1989). Air hunger was observed in equines suffering from simple and strangulated intestinal obstructions (Singh *et al.*, 1975). No alteration was seen in respiratory rate but with increase in duration of disease (Pearson, 1971; Pearson and Pinscent, 1977), an involuntary grunt accompanying each expiration was recorded in bovines. Makhdoomi (1994) observed mild and insignificant increase in respiration following simple and strangulated intestinal obstruction in buffalo calves.

2.5 HAEMATO-BIOCHEMICAL CHANGES FOLLOWING INTESTINAL OBSTRUCTION

2.5.1 HAEMATOLOGICAL CHANGES

Haematological alterations in cattle suffering from intestinal obstruction are general in nature and seldom designate the type or location of lesion. In addition, haematological status of adult cattle may not necessarily display the gravity of gastrointestinal disease (Smith, 1985a).

The obvious sign observed primarily in intestinal obstruction was haemoconcentration in all the species of animals (Hammond ^{e^t al.}, 1964; Yale, 1969; Gingerich and Murdick, 1975; Koch *et al.*, 1978; Vander Velden, 1983; Avery *et al.*, 1986; Celly and Prasad, 1997). The packed cell volume remained below 45 per cent and rarely exceeded 50 per cent in adult cattle (Pearson, 1971). A rise in haematocrit value was observed in simple and strangulated bowel obstruction (Singh *et al.*, 1975) in equines. Increased haematocrit has also been reported in torsion of caecum (Braun *et al.*, 1989b) and intussusception (Nath *et al.*, 1991) in cattle.

Elevated haemoglobin values in experimental and clinical intestinal obstruction have been reported in calves (Bhokre and Deshpande, 1988). On the contrary decreased levels of Hb have been reported (Singh and Kohli, 1980; Singh, 1987) in buffaloes. Papadopoulos *et al.* (1985a) found a positive correlation with haemoglobin and haematocrit as increase in haemoglobin values occurred in all obstructive disorders of intestinal tract in cattle.

Leukocytosis and neutrophilia with shift to left occurred in simple and strangulated intestinal obstruction in equines (Singh *et al.*, 1975). Haemoconcentration, neutrophilia and lymphocytopenia was a common finding in cases of induced intussusception in cattle (Nath *et al.*, 1991). Inflammatory leukogram with increased number of mature neutrophils may be seen if ischemic necrosis of the intestinal wall has occurred (Anderson *et al.*, 1993). In human beings the leukocytic count $< 15,000/\mu\text{l}$ suggested simple obstruction; counts $>15,000/\mu\text{l}$ suggested impaired circulation; counts $>25,000/\mu\text{l}$ suggested infarction (Hoslink, 2003).

Significant increase in total erythrocytic count has been recorded (Bhokre and Deshpande, 1988) following simple intestinal obstruction in calves. Jonson and Hogstrom (1991) documented that reduction in early wound margin strength is neutrophil dependent. Vural *et al.* (1999) concluded that polymorphnuclear cells have a major role to play in modulating post operative adhesion from previous abdominal surgery whereas neutropenia lowers the degree of post operative adhesion formation.

2.5.2 BIOCHEMICAL CHANGES

Metabolic derangement in cattle suffering from intestinal obstruction occurs as a result of underlying pathology and is more pronounced if seat of obstruction is proximal.

A close response relationship was indicated between the extent of small intestinal damage and clinical symptoms and same was applicable to biochemical parameters (Hjortkjaer and Svendsen, 1979). Increase in levels of plasma total proteins has been recorded following simple intestinal obstruction in cattle (Pearson and Pingent, 1977; Braun *et al.*, 1990; Nath *et al.*, 1991). Variable changes in plasma total proteins were observed following simple and strangulated intestinal obstruction in equines (Datt and Usenik, 1975; Johnston and Morris, 1987).

Blood urea nitrogen was found significantly increased in experiment and clinical cases of intestinal obstruction in ruminants (Parsania *et al.*, 1983; Papadopoulos *et al.*, 1985b and Singh, 1987) and equines (Datt and Usenik, 1975). Hammond *et al.* (1964) reported that calves with proximal obstruction had significant increase in BUN values, while in those with obstruction of small colon, the BUN values remained within normal limits. The azotemia has been reported in cattle affected with clinical volvulus and intussusception (Andersson *et al.*, 1993; Constable *et al.*, 1997). Significant increase in blood creatinine levels was noticed in strangulated obstructions of cattle (Makhdoomi *et al.*, 2002) and horses (Hjortkjaer and Svendsen, 1979).

Inconsistent results have been obtained in blood glucose concentration following intestinal obstruction in ruminants. Hypoglycemia has been observed in

buffalo calves (Krishnamurthy *et al.*, 1980; Singh and Kohli, 1980). Hyperglycemia has been reported in ruminants following intestinal obstruction (Parsania *et al.*, 1983; Constable *et al.*, 1997). No change in blood glucose was seen in the camels suffering from acute abdomen (Anderson, 1996).

Hyponatremia has been a usual finding in cases of intestinal obstruction. In strangulated intestinal obstruction the decrease in plasma sodium concentration was not as severe as in simple obstruction (Singh, 1971). A pronounced hypochloremic alkalosis has been reported following intestinal obstruction in cattle (Hammond *et al.*, 1964, Smart *et al.*, 1977; Papadopoulos *et al.*, 1985b and Constable *et al.*, 1997). Decrease in plasma chloride values has also been recorded in buffalo calves (Makhdoomi *et al.*, 2002) and cow calves (Sharma, 1999). Braun *et al.* (1988) documented a significant correlation between serum chloride concentrations and the rate of cure. Cows with chloride concentration above 80 mmol/litre had a 74 per cent good prognosis, but when the concentration fell below this level prognosis rapidly became grave. Plasma potassium concentrations were low in strangulated intestinal obstruction in cattle (Papadopoulos *et al.*, 1985b; Avery *et al.*, 1986 and Doll, 1991), equines (Datt and Usenik, 1975), buffaloes (Celly and Prasad, 1997) and camels (Anderson, 1996).

The plasma phosphorous concentration did not show significant alteration from normal in simple and strangulated intestinal obstruction (Avery *et al.*, 1986, Makhdoomi *et al.*, 2002). Contrariwise, hyperphosphatemia was noticed following experimental strangulated obstruction in dogs (Shikata *et al.*, 1989), cattle (Braun, 1989) and equines (Murray *et al.*, 1994). Significant decrease in plasma calcium concentration was documented in buffalo calves (Parsania *et al.*, 1983) and cattle

(Braun *et al.*, 1989). Insignificant changes in calcium following strangulated obstruction have also been recorded by Avery *et al.* (1986).

Combined with haematology and biochemical status the serum enzyme profile forms the data base for most diagnostic investigations. Many serum enzymes have specificity for an organ and/or a limited range of pathological processes. There is no documentation regarding serum enzyme profile of cattle suffering from intestinal obstruction, therefore the reviewed literature pertains to non ruminant species.

Intestinal obstruction caused reversible changes in the liver and in hepatic metabolism which normalized after relief of obstruction (Kausz and Nemesanszky, 1976). The character of changes in the activity of the alkaline phosphatase isoenzyme can serve as reliable evidence (Eriukhin *et al.*, 1981) of the predominant localization, the degree of the disease and of the inflammatory destructive process, thus contributes to earlier recognition of acute disease of organs of the abdominal cavity in humans. De Toma *et al.* (1983) reviewed the serum changes of number of markers in an animal model of bowel ischemia and noted that mesenteric infarction resulted in significant increase in serum lactic dehydrogenase (LDH), aspartate amino transferase (AST), alkaline phosphatase (ALKP) and creatinine kinase (CK). However, none of these enzymes was specific for intestinal infarction.

The elevated levels of ALKP during intestinal ischemia did not originate from liver as all the tests of liver function were normal, furthermore on electrophoresis the "ischemic ALKP" migrated faster than a known liver ALKP standard but more slowly than an intestinal ALKP sample (Williams and Wilson,

1981). The intestinal mucosa is most sensitive to ischemia, serum levels of mucosal enzymes such as diamine oxidase and ALKP and seromuscular enzymes like creatinine kinase (CK), lactic dehydrogenase (LDH) and Aspartate transaminase (AST) may indicate the severity of intestinal ischemia (Thompson *et al.*, 1990). Horses with proximal enteritis and small intestinal strangulated obstruction had higher serum AST and ALKP (Davis *et al.*, 2003).

Significant increase was found in the activities of aminotranferases, creatine kinases and alkaline phosphatases (Kazmierczak *et al.*, 1988) in the intestinal infarction and these changes were less pronounced in simple intestinal obstruction in animal models. Intestinal ALKP released into the circulation in some babies with bowel necrosis (Mclachlan *et al.*, 1993) but its detection did not have the diagnostic sensitivity and specificity as a reliable marker of the condition. Oruc *et al.* (2004) evaluated the serum hexosaminidase (HEX) levels in recognition of strangulation in an experimental model of closed loop small bowel obstruction and advocated that increased level of serum HEX levels may indicate irreversible transmural infarction therefore, it was not useful for detecting the reversible and/or irreversible ischemia in the early period of strangulation.

Amylase is found in number of organs and tissues. The greatest concentration is present in the pancreas. The normal serum amaylase present in serum and urine is predominantly of pancreatic and salivary origin. Hyperamylasemia has been found in intestinal obstructions and other intestinal disorders in man (Moss and Henderson, 1994). Afferent loop syndrome combined with duodenal phytobezoar resulted in abnormal blood biochemistry in humans (Hui *et al.*, 1997) comprising of increased levels of amylase, lipase, alkaline

phosphatase, total bilirubin, AST and ALT in which amylase was markedly elevated to 1188U/L. Petrov *et al.* (1999) while treating the patients of peritonitis and intestinal obstruction established that there was an increase in the quantity of enterobacteria and unfermenting gram-ve bacteria in intestinal paresis alongwith increase in ALKP, amylase, bilirubin, transaminases and potassium content as well.

2.6 PERITONEAL FLUID ANALYSIS

Evaluation of the cattle with intestinal obstruction has always been challenging since the patient's large size precludes many of the diagnostic imaging procedures commonly used in humans. Diagnostic modality like peritoneal fluid examination is an important consideration in presurgical evaluation (Fischer, 1989). However, this predictive method is also not 100 per cent accurate and clinicians must continue to rely on clinical evidence and instinct and should use these diagnostic and prognostic procedures only as guides for case management.

The technique for collection of peritoneal fluid in horses (Coffman, 1973) and bovines (Krishnamurthy *et al.*, 1980) have been well documented. Abdominocentesis can be performed from ventral para median area, several inches caudal to the xiphoid and to either side of midline or at the linea alba just caudal to umbilicus (Smith, 1985a) or midway between xiphoid and umbilicus on the ventral midline (Krishnamurthy *et al.*, 1980) in calves.

Comparative analysis of the normal peritoneal fluid with respect to cellular and total protein concentration in calves and adult cattle have been reported (Anderson *et al.*, 1995). Colic being frequent affection in equines also warrants the peritoneal fluid analysis especially the total protein concentration which rose to 2.8

mg/dl and 5.4 mg/dl respectively in simple and strangulated obstruction (Allen *et al.*, 1986).

Discoloured peritoneal fluid (suggestive of intestinal strangulation) may be present in horses with anterior enteritis or nonstrangulating intestinal infarction (Mair and Edwards, 2003). Changes in the peritoneal fluid in colicky horses with their relationship to the site and type of lesion, before and after medical or surgical treatment showed discolouration of the peritoneal fluid as an important indicator of intestinal strangulation (Baccarin *et al.*, 1995). Discoloration commenced early in the course of intestinal obstruction even while the segment of bowel was still viable (Swanwick and Wilkinson, 1976).

The features of peritoneal fluid like clear yellow colour and WBC count upto 3000/ μ l with 60 per cent neutrophils and 40 per cent mononuclear cells and total protein content of 0.7-1.2g/dl has been considered normal where as in abnormal conditions like simple obstruction (Increased protein), peritonitis/thrombotic colic (Increased protein and WBC) and strangulation (Increased protein, WBC, RBC and bacteria) were found in horses (White and Randolph, 2003).

Mean nucleated cell count, neutrophil and total protein concentration in the peritoneal fluid of horses with small intestine obstruction (Johnston and Morris, 1987; Swanwick and Wilkinson, 1976) and cattle with intussusception (Smith, 1985a) has been reported. The effect of omentopexy and laparotomy in adult dairy cows (Anderson *et al.*, 1994) and enterocentesis in horses (Schumacher *et al.*, 1985) on peritoneal fluid constituents have been studied. Latson *et al.* (2005) suggested analysis of peritoneal fluid's gross appearance, chloride, pH, and

lactate levels for diagnosis of intestinal ischemia secondary to strangulated intestinal obstruction in horses.

A difference between blood to peritoneal fluid glucose concentration should be used as more reliable diagnostic indicator of septic peritoneal effusion than peritoneal fluid glucose concentration alone (Bonczynski *et al.*, 2003) in dogs. Increased urea nitrogen concentration in buffalo calves (Krishnamurthy *et al.*, 1980) and ammonia content in horses (Datt and Usenik, 1975) have been recorded in peritoneal fluid following strangulated intestinal obstruction. Hypoglycemia and insignificant changes in total protein and alkaline phosphatase values have been observed in the peritoneal fluid of buffalo calves during strangulated obstruction (Krishnamurthy *et al.*, 1980). Lower pH, higher protein, higher fibrinogen and lower glucose concentrations were the typical findings in the peritoneal fluid of horses suffering with septic peritonitis (Van Hoogmoed *et al.*, 1999).

2.7 RUMINAL FLUID ANALYSIS

Ruminal fluid studies combined with serum biochemistry have been used to differentiate between the different causes of disturbance of the digesta outflow and promote more accurate therapy and prognosis.

Reflux of abomasal contents into the rumino-reticulum has been reported in many cases of digestive disturbance in ruminants. Abomasal reflux was diagnosed by measuring ruminal chloride concentrations and buffering capacity. Abomasal reflux was established in cattle with intestinal obstruction and intussusception of the small intestine. The ruminal fluid examination was compared with the results of blood examination. It was shown that abomasal reflux (Breukink and Kuiper, 1976,

Vander Valden, 1983; and Singh *et al.*, 1995) was found not only in cases of acute intestinal obstruction, but also in other types of gastrointestinal disorder in cattle. The hypochloraemic alkalosis as a result of this reflux severely aggravated the condition of the patients. Braun *et al.* (1988) coined the term internal vomiting to explain the abomasal reflux occurring in small intestinal obstruction, pyloric stenosis and abomasal displacement which was characterized by ruminal acidosis with increased levels of chloride ions and manifested by dehydration, impairment of heart function and blood circulation, weakness and apathy. Continued secretion of hydrochloric acid from abomasum, inflow of saliva into rumen and impaired reabsorption due to obstruction led to accumulation of chloride in ruminal fluid of cattle (Dobson and Phillipson, 1958; Smart *et al.*, 1977; Papadopoulos *et al.*, 1985b and Avery *et al.*, 1986, Makhdoomi *et al.*, 2002) and sheep (Doll, 1991).

The decrease in ruminal fluid sodium concentration was recorded following intestinal obstruction in buffalo (Makdoomi, 1994) and cow calves (Avery *et al.*, 1986, Papadopoulos *et al.*, 1985a). Significant increase in the concentration of urea nitrogen and creatinine was observed in ruminal fluid of buffalo calves following strangulated obstruction (Makhdoomi *et al.*, 2002). Reductions in rumen fluid potassium indicated the absorption of this ion from rumen against concentration gradient in an attempt to moderate hypokalemia (Tasker, 1980) but Avery *et al.* (1986) concluded that duodenal obstruction in heifers had no effect on rumen sodium, calcium and magnesium concentrations whereas potassium concentrations increased. Avery *et al.* (1986) found that the degree of alkalosis and electrolyte changes in ruminal fluid was greater in the steer than calves.

The colour of the ruminal fluid changed with the feeding habits of animals (Radostits *et al.*, 1994) it can vary from green to grey. The pH of rumen contents varied with feed. The normal range was 6.2-7.2. Ruminal fluid pH decreased initially on duodenal obstruction in cattle (Avery *et al.*, 1986) and showed no change afterwards. Decreased rumen pH has been observed (Sharma, 1999) in strangulated intestinal obstruction in calves.

2.8 MICROBIOLOGICAL STUDIES

The gastrointestinal tract besides being the organ responsible for nutrient absorption is also a metabolic and immunological system which functions as an effective barrier against endotoxin and bacteria in the intestinal lumen. The passage of viable bacteria from the gastrointestinal tract through the epithelial mucosa is called bacterial translocation. Equally important may be the passage of bacterial endotoxin through the mucosal barrier.

Bacterial flora studied by direct sampling techniques (Gupta *et al.*, 1980) indicated that bacterial contamination of peritoneal fluid and dissemination of microorganisms can be indirect and can occur from combination of factors like ingestion of food, retrograde spread from the bowel, lymphatics and/or haematogenous spread. Bacteriologic studies have been conducted on the peritoneal fluid, heart blood and on the content of obstructed segment of intestine in dogs (Cohn and Atik, 1961) and *Coliforms*, *Streptococcus spp.*, *Staphylococcus spp.* and *Clostridium spp.* from lumen and peritoneal fluid were isolated. Isolation of different species of microorganisms has been reported from the strangulation fluid, peritoneal fluid and blood in dogs (Yale, 1969) after experimental creation of intestinal obstruction. Antequera *et al.* (2000) proposed that translocation of

enteric bacteria may be the cause of infection that brings about the death at 48 hours (24 per cent) and 72 hours (33 per cent) post intestinal obstruction in lab animals. *Clostridium perfreingens* was consistently isolated from faeces of 17 out of 22 cows suffering from hemorrhagic bowel syndrome (Dennison *et al.*, 2002).

In buffalo calves, *Proteus spp.*, *E.Coli* and *Staphylococcus spp.* were isolated on the bacteriological examination of blood (Singh and Kohli, 1980) in strangulated jejunal obstruction. A spectrum of bacteria viz. *E.Coli*, *Citrobacter frucidil*, *Alakligens feaclis*, *Pseudomonas aeruginosa* have been isolated from fecal, intestinal, peritoneal fluid and mesenteric lymph node in goats following ischemic and haemorrhagic strangulated intestinal obstruction (Parvathamma, 1992). Obstruction induced intestinal injury (Deitch *et al.*, 1990) was very much attributed to disruption of ecology of the normal gut microflora leading to intestinal overgrowth with certain enteric bacilli and mucosal damage. The relationship between reperfusion injury, bacterial translocation and multiple system organ failure in bowel ischemia and reperfusion injury has been studied in dogs (Sheng *et al.*, 1991).

A light electron microscopy conducted on rabbits (Kabaroudis *et al.*, 2003) revealed that the disruption of the mucosal barrier begins 4 hours after complete intestinal obstruction and at 12 hours after complete intestinal occlusion, the disruption was total with different degree of severity.

2.9 ELECTROCARDIOGRAPHICAL STUDIES

Electrocardiography is an important tool in delineating the structural or functional cardiac as well as non cardiac disorders. The main purpose of ECG in present study was to find out any abnormality in cardiac functioning during the

course of obstruction consequent to electrolyte disturbance. Very scanty literature pertaining to cattle is available.

In a three year period McGuirk *et al.* (1983) recorded atrial fibrillation in 16 dairy cows suffering from gastrointestinal problem and most common electrolyte disorders were hypocalcemia, hypochloremia and hypokalemia. Some of the cows had no acid base disturbance but metabolic alkalosis, respiratory alkalosis was found in other cows. Like wise Atrial premature complexes (APC) were identified in 16 cows out of which 14 had gastrointestinal diseases. Ten cows were determined to be hypocalcemic and 4 cow's hypokalemic when APC were identified. Constable *et al.* (1990a) concluded that APC's may be a functional disorder in cattle unrelated to structural heart disease and the potential of APC to progress into sustained atrial arrhythmias such as atrial fibrillation should be considered.

Neostigmine used to promote the intestinal peristalsis in post surgical ileus had produced atrial fibrillation (Constable *et al.*, 1990b) and it converted to normal sinus rhythm after cessation of treatment. Intestinal obstruction in non ruminants produced hypokalemia (Butler, 1973) in relation to ECG findings manifested by shallow T wave, prominent "U" wave, ST segment depression, slightly peaked P wave with a slightly prolonged PR interval. Intestinal obstruction produced atrial flutter (Sobti, 1996) which was almost same as atrial fibrillation except for the oscillations which were bigger than those in atrial fibrillation in cattle.

2.10 HISTOPATHOLOGICAL STUDIES

The gastrointestinal mucosa forms a barrier between the body and a luminal environment which not only contains nutrients, but also laden with potentially hostile microorganisms and toxins. Many of gastrointestinal diseases

lead to disruption of the mucosal barrier thus allowing escalation to systemic diseases thereby adding further insult to an already compromised system.

Intestinal obstruction produced occlusive ischemia of intestine where the intestinal flow was directly disrupted and resulted in insufficient delivery of oxygen and nutrients necessary for maintenance of cell integrity (Snyder, 1989). In addition the intestinal edema and luminal distension were common findings which aggravated intestinal damage but severity of ischemia was a limiting factor in determining the clinical outcome of the obstruction (Snyder, 1989). The microscopic studies in rats (Toskes *et al.*, 1975) and dogs (Ashraf *et al.*, 1982) revealed hypertrophy of both crypts and villi with focal abnormalities of villus architecture. Ten to twenty per cent of the columnar cells in the upper half of the villi were swollen and vesiculated.

Ultrastructural changes in the small intestine of dogs (Yamamoto *et al.*, 1980) after mechanical obstruction showed alteration in microvilli with fragmentation into vesicles, narrowing of apical microvilli and decrease in number. A comparable complete necrosis of the small intestine mucosa was detected in the rat after 7 hours and in man after 44 hours of ischemia (Wagner and Gabbert, 1983).

On visual examination the experimentally induced venous strangulated obstruction in ponies was found haemorrhagic with edematous bowel wall and mesentery which turned dark in colour as the duration progressed (Freeman *et al.*, 1988a). Papadoulous *et al.* (1987) in his experimental study on intestinal obstruction in cattle found the mucosa of abomasum was edematous with petechiae or less frequently ulcers. At the point of ligation the intestinal wall was

atrophied with no sign of peritonitis and the section of intestine cranial to the obstruction as well as abomasum was found to be distended and full of watery contents and gases. The angiographic evaluation (Dabareiner *et al.*, 1993) of luminal distension in equine jejunum during obstruction identified the short villi separated by expanded crypts and had mesothelial cell loss, neutrophil infiltration and edema in the seromuscular layer, the subsequent decompression did not reverse the altered vascular density.

Surgical manipulation of intestines resulted in generation of oxygen free radicals which lead to mucosal damage as evidenced by ultrastructural and biochemical changes (Thomas *et al.*, 2002). The most common morphologic diagnosis at necropsy of cows suffering from haemorrhagic bowel syndrome (Dennison *et al.*, 2002) was severe necrohemorrhagic enteritis or jejunitis with intraluminal blood clots and most prominent histologic finding was severe, segmental submucosal hemorrhage and edema of small intestine.

2.11 CONSERVATIVE AND SURGICAL MANAGEMENT OF INTESTINAL OBSTRUCTION

Small intestinal obstruction remains a frequently encountered problem in abdominal surgery. Although modern day surgical management continue to focus appropriately on avoiding operative delay whenever surgery is indicated, not every patient is best served by immediate operation. Certain entities such as small bowel obstruction complicated by intestinal perforation, prolonged intestinal ischemia and fluid/electrolyte disturbances do require prompt operative intervention. At the same time surgeon should continue their aggressive attitude towards correction of fluid

and electrolyte disturbances, which continue to account sizeable post operative mortality.

Many intestinal surgeries are constructed in an emergency setting. In this context, careful preoperative preparation, including adequate fluid resuscitation, is important and should be carried out to the extent possible. Elective patients should be as fit as is feasible, and any other active coexisting illnesses should be stabilized or controlled as well as possible (Britton, 2003). To maximize the chances that the anastomosis would heal uneventfully, patients should be well nourished and not anemic. Adequate preoperative antibiotic prophylaxis has been shown to reduce the risk of postoperative infection in all types of bowel surgery and must be given at the start of the operation. Some patients require additional administration of steroids preoperatively.

The treatment protocol for intestinal obstruction included right flank laparotomy, removal of the obstruction and institution of fluid therapy. Surgery was found essential to relieve the obstruction that varied from simple separation of adhesions to resection of large segment of intestine (Singh *et al.*, 1995). The post operative care was aimed at correction of dehydration, acid–base and electrolyte disturbance, restoration of normal gastrointestinal tract motility and prevention of infection (Smith, 1985c). Adequate and appropriate fluid therapy, administration of antibiotics, use of gastrointestinal stimulants and oral administration of ruminal fluid helped in speedy recovery of affected animals (Singh *et al.*, 1995).

Exploratory laparotomy through right flank incision to correct bowel obstruction in cattle has been performed under local analgesia by nerve block or linear infiltration which provided optimum access to intestine (Pearson and

Pinsent, 1977). On entering the abdomen, the gaseous distension was relieved by decompression of abomasum and caecum to facilitate further exploration (Tulleners, 1981). Intestinal resection and anastomosis following intestinal obstruction in cattle in left lateral recumbency has been recommended (Smith, 1985c). To avoid intestinal resection in standing animals, a layer side to side jejunal anastomosis in small intestinal obstruction in a cow has been performed using 2-0 chromic catgut (Koch *et al.*, 1978).

Assessment of intestinal viability is important because intestine's capacity to survive and function normally after injury and to heal without residual changes that could cause clinical problems is foremost mandate. The decision to resect the bowel of questionable viability was based on certain considerations like risk of malabsorption, accessibility of viable intestinal margins for anastomosis, complexity of anastomosis, overall effects of additional surgery time and experience of surgeon (Duchrame *et al.*, 1992). Final decisions should be tampered with realization that most types of anastomosis could lead to adhesions and stenosis and the risk was greater with the development of peritonitis, shock and diffuse intestinal hypoperfusion (Duchrame *et al.*, 1992).

All dynamic processes are obscured due to edema and congestion so inspection of the mucosal surface was not advised (Shah and Anderson, 1981 and Freeman *et al.*, 1988a). Various methods like Florescein dye test, Doppler ultrasonography (Freeman *et al.*, 1988b), Thermography (Purohit *et al.*, 1982) and Surface oximetry (Snyder *et al.*, 1985) have been employed. To assess the viability of affected segment of intestine it has been concluded (Duchrame *et al.*, 1992) that short segments of small intestine (less than 45 cm) with ischemic

damage characterized largely by venous congestion, hemorrhage and edema and if there is some improvement in appearance, presence of spontaneous or evoked peristalsis (more appropriately after strangulated obstruction is corrected) and bathing the involved segment in warm saline for few minutes can improve color and peristalsis can be judged as viable. Evidence of peristalsis of the affected bowel segment on careful inspection was the only reliable and visible criterion of bowel viability (Pearson and Pinsent, 1977).

Britton (2003) recommended that the segment of bowel to be removed must be isolated with an adequate margin and the key consideration should be to preserve the blood supply to the two remaining ends of bowel. The ties placed close to the bowel can bunch tissues excessively causing angulations and distortion of the free edges leading to difficult anastomosis and threatened blood supply and the mesenteric ligation should not slip as it can result in formation of haematoma with in leaves of mesentery which can itself threaten the viability of bowel.

Skin, subcutaneous tissue and viscera takes 14 to 21 days (Peacock, 1984) for sufficient healing. Long lasting monofilament absorbable sutures like polydioxanone, polyglyconate or nylon have been preferred as they had low albumin level, minimum tissue drag and tensile strength upto 45 days. Catgut was considered questionable as it rapidly degenerated (Deveney and Way, 1977). Stainless steel no. 5-0 has also been used because of its excellent strength and relative inertness. Shackelford and Zuidema (1989) recommended that goal of enteroanastomosis should be to obtain watertight closure without stricture and to prevent adhesion formation an accurate serosal closure is essential. Repeated

perforation with needle and mucosal protrusion is to be avoided as it resulted in localized peritonitis and adhesions.

The technical options of fashioning anastomosis have inherent variations. Like perianastomotic oxygen tension was more with interrupted pattern than continuous pattern but water tight seal with less chances of eversion of mucosa and less exposure of suture material was more with continuous pattern (Shandall *et al.*, 1985). Single layer suturing offered less narrowing of lumen and fostered rapid vascularization than double layer pattern but double layer was indicated when the tissues were edematous and friable and were under minimal tension and located in highly vascular areas like jejunum (Jiborn *et al.*, 1980; Orr, 1969 and Khoury and Waxman, 1983). The inversion pattern ignored the basic principle of opposing clean cut tissues where as eversion pattern has lower bursting pressure, slower healing, more severe inflammation and bacteria laden mucosa is exposed to peritoneal surfaces resulted in adhesions and stricture (Undre, 1983).

End to end anastomosis has been evaluated as satisfactory technique to appose the cut ends of the bowel (Pearson and Pingent, 1977). Six intestinal anastomotic techniques have been compared experimentally in calves (Singh *et al.*, 1985). In short duration of intestinal obstruction enterectomy may not be necessary but in long standing cases with serious lesions of the bowel segment enterectomy and end to end anastomosis has been recommended (Papadopoulos *et al.*, 1987). Single layer inversion anastomosis technique has proved to be the best, double layer inversion being the second best followed by double layer eversion and single layer eversion techniques (Singh *et al.*, 1989) in experimental colonic anastomosis in cow calves. Eversion technique and

mersilene (Singh, 1971) has been found to be superior to end to end anastomosis in buffalo calves. Safety of intestinal anastomosis with single layer inversion technique using Connell sutures in goats have been reported (Parvathamma, 1992). Some stapled intestinal anastomosis techniques in equines (Freeman, 2001, Fugaro^{and coles}, 2001), biofragmentable ring in calf (Iselin and Steiner, 1993), fibrin-collagen plates (Gorskii, 2001) and cyanoacrylate tissue adhesive (Tebala *et al.*, 1995) in high risk intestinal anastomosis in humans have been reported.

Fleming and Remington (1982) proposed that intestinal failure should be defined as "the reduction of the functioning gut mass below the minimum necessary for adequate digestion and absorption of nutrients. The water flux towards and from the intestine in a healthy calf was estimated at about 90-100 liters/day, while the net absorption was only 3-4 liters (Bywater and Logan, 1974).

Post operative fluid therapy (oral and intravenous) complemented with administration of analgesics has been advocated to treat dehydration, shock, electrolyte imbalance, prerenal azotemia and to moderate the vigor of peristalsis of the bowel (Pearson and Pinsent, 1977; Smith, 1985c and Johnston and Morris, 1987). Intravenous infusion of polyionic solutions like lactated ringers solution in horses (Murray *et al.*, 1994), fluids containing potassium chloride, sodium chloride, ammonium chloride, ringer's solution, glucose in cattle (Tulleners, 1981; Smith, 1985d, Papadopoulos *et al.*, 1987, Braun *et al.*, 1990) and dextran, electrolytes and whole blood in dogs (Cohn and Atik, 1961) following correction of simple and strangulated intestinal obstructions have been recommended. Ammonium chloride and dilute hydrochloric acid (Myron, 1982) has been suggested to counteract the alkalosis in intestinal obstruction. Lopes *et al.* (2002) in his comparative study

found that enteral fluid therapy was more effective in promoting ingesta hydration and produced less pronounced systemic effects than the intravenous fluids combined with magnesium sulphate used in large intestine impactions of horses.

Barton (2002) proposed that the best fluids to give in proximal intestinal obstruction and abomasal displacement were 0.9 per cent normal saline or Ringers (has no lactate) and hypertonic saline (7 per cent at 4 ml/kg) can also be used as it is cheap, fast and a temporary way to increase circulatory volume. He further recommended that it should be made sure to follow hypertonic saline with isotonic fluids or animals should resume water intake postoperatively. Lactated or acetated ringers solution could be used as a third choice in case of unavailability of fluid of choice but obstruction should be corrected immediately as it has been found that correction of the obstruction resulted in lowering of plasma HCO_3^- and increase in Cl^- . The best and preferred route (Barton, 2002) was IV (with intermittent boluses) until displacement/obstruction is corrected. He recommended that if clinically, the patient is stable, the recommendations for the economic approach was to first correct the obstruction, followed by oral fluids by intubating 1 to 2 times and 20 liters can be give in 400 kg of cow per day.

Prokinetic drugs have played an important role in the treatment of post operative ileus in horses (Dart *et al.*, 1996). Effects of flunixin meglumine on plasma prostonoid concentration in colic horses with in perioperative time frame have been evaluated (Gerdemann *et al.*, 1997). The use of preoperative parenteral antibiotics, sulphonamides and flunixin meglumine (Smith, 1985c and Papadopoulos *et al.*, 1987) has been suggested in intestinal obstruction in cattle. The use of parasympathomimetic drugs like neostigmine, metoclopramide for

management of atonic bowel in post operative period has been reported (Pearson and Pinsent, 1977; Adams, 1988; Braun *et al.*, 1990, Blikslager *et al.*, 1994) in bovines and equines.

Post operative care enabling speedy recovery included adequate fluid therapy, administration of antibiotics, and use of gastrointestinal stimulants such as calcium solution and neostigmine and oral administration of ruminal cud from healthy animals (Bhatia, 1998). Post operative paralytic ileus was a common complication in equines (Boom and Valden, 2001) and cattle (Bhatia, 1998), which was managed in cattle by giving calcium solutions and neostigmine in smaller doses (2 mg SC or IM every 4-5 hour). Roussel *et al.* (2001) advocated that early surgical intervention, short surgery time and enterotomy have decreased the probability of post operative ileus in horses. Topical use of 1 per cent sodium carboxymethylcellulose during celiotomy in horses has been recommended (Hay *et al.*, 2001) to decrease the frequency of adhesion formation. Dart *et al.* (1996) concluded that metoclopramide given as continuous IV infusion decreased the incidence and severity of ileus following small intestinal resection and anastomosis in horses.

Acute reversible intestinal failure has been reported after abdominal surgery when oral intake was prevented for longer period of time (Klein, *et al.*, 1997). Recent reports suggested that undue delay in introduction of oral intake after an uncomplicated abdominal surgery might result in prolonged recovery and the patients tolerated oral feeding as early as 24 hours of laparotomy (Hessove *et al.*, 1988). In humans, prolonged intravenous administration of 5 per cent dextrose and saline was associated with intestinal failure (Sandstrom *et al.*, 1993) due to

intraabdominal infection and ileus, hypoalbuminemia, edema and deterioration of critical physiological functions such as the immunity, wound healing and respiratory muscle strength. Randomized trials conducted by Nicola (2003) on humans provided evidence that the use of enteral feeding after gastrointestinal surgery did not increase mortality and morbidity.

CHAPTER - III

MATERIALS AND METHODS

3.1 EXPERIMENTAL DESIGNS

The present study was conducted on 24 healthy male cross bred cow calves, 8 to 15 months of age, with their body weight ranging between 70 to 150 Kg. The calves were stall fed and were kept under uniform management conditions throughout the course of study. Prior to experimentation, deworming was performed with Albendazole (Albomar suspension, Agrivet Farmcare) @ 7.5 mg/Kg body weight and acclimatization period of 15 days was given. These animals were randomly divided into six groups of four animals each. The necessary permission to carry out the experimental work was taken from Institutional Animal Ethics Committee.

3.1.1 PILOT TRIALS

Pilot trials were conducted on five calves to establish a suitable model of strangulated intestinal obstruction of the jejunum, to evolve a therapeutic regimen (conservative therapy) based on the pathophysiological response depicted in blood, plasma, peritoneal fluid and clinical parameters and to record the total survival time following strangulated obstruction.

3.1.2 CREATION OF MODEL OF SIMPLE AND STRANGULATED INTESTINAL OBSTRUCTION

In all the animals, right paralumbar fossa was prepared for aseptic surgery. The animals were restrained in left lateral recumbency for undertaking laparotomy through right flank incision. The abdominal cavity was entered through a linear incision at the centre of the right paralumbar fossa under local infiltration analgesia using two per cent lignocaine hydrochloride (Xylocaine 2 per cent injection, Astra-

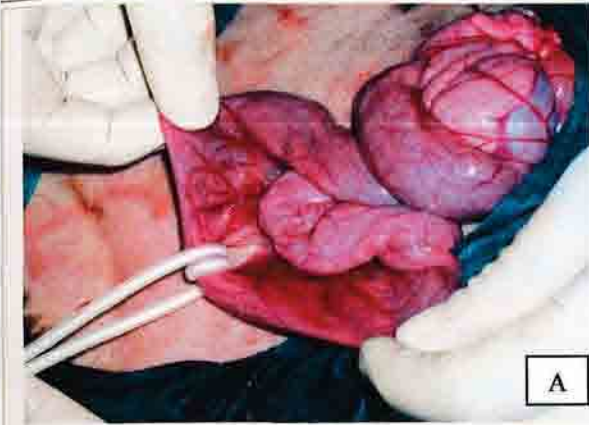
Zencea, India). Following the entrance to the abdominal cavity, the layers of omentum were incised to locate the cranial jejunum. To create the simple intestinal obstruction a constricting ligature consisting of flexible silicon tubing braced with umbilical tape suture material in its lumen was loosely placed around the cranial jejunum in a double loop fashion without interfering with the mesenteric vasculature (Plate1). The strangulated intestinal obstruction was created with similar technique but in addition the blood vessels supplying to the constricted segment were ligated with the help of 1-0 catgut (Ethicon) suture (Plate 2).

Two separate holes were created through the omentum and from these the silicon tubing was passed outside the omentum (Plate 2C). This was done to keep the jejunum within the omental aquarium and to prevent the direct exposure of the jejunum to the abdominal wall. The incised omental layers were then sutured using 1-0 Polyglactin 910 (Vicryl, Ethicon) in simple continuous fashion. Two plastic discs were used on the interior and exterior side of the abdominal wall. These two plastic discs were then fixed tightly to the lateral wall with the Stainless steel wire threads, which were then passed through corresponding small holes, at the periphery and opposite positions of the two plastic discs (Plate 2F). In a standing position, the ligature was tightened by pulling the free ends of the plastic tubing protruding from abdominal wall, immediately after closure of wound.

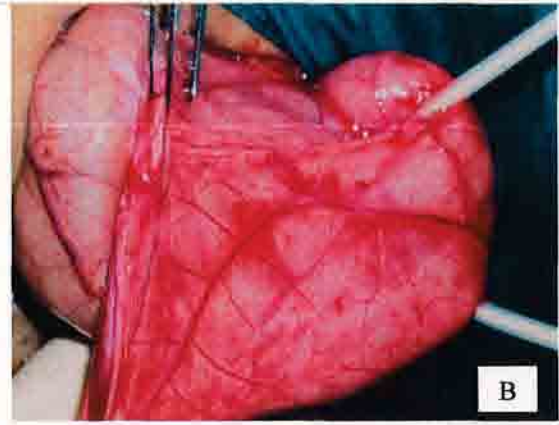
3.1.3 COMPOSITION OF GROUPS

Simple Obstruction	Group I	• Four animals served as untreated control
	Group II	• Four animals were given conservative treatment
	Group III	• Four animals were treated surgically and were maintained on conservative treatment post-operatively
Strangulated obstruction	Group I	▪ Four animals served as untreated control
	Group II	▪ Four animals were given conservative treatment
	Group III	▪ Four animals were treated surgically and were maintained on conservative therapy post operatively

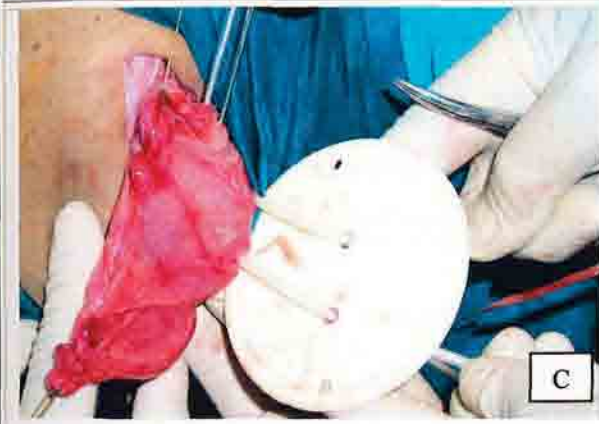
PLATE: 1 CREATION OF SIMPLE JEJUNAL OBSTRUCTION IN CALVES



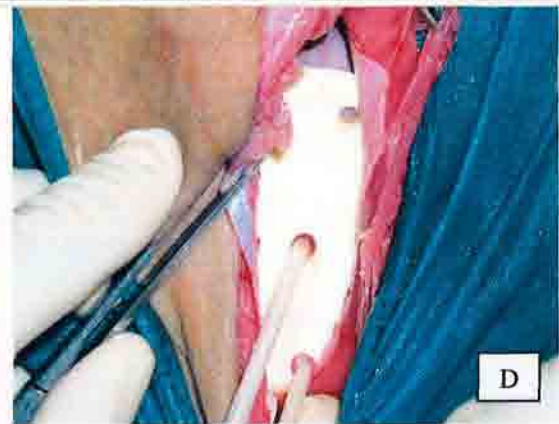
Silicon catheter braced with umbilical tape was looped around the segment of bowel



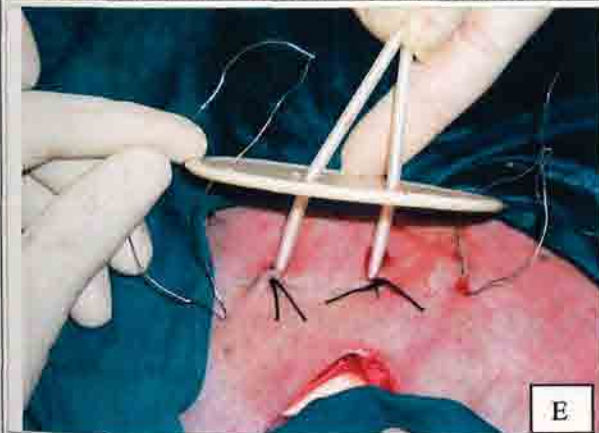
Looped segment of bowel placed in the omentum and the tubing was egressed after creating holes in the omentum



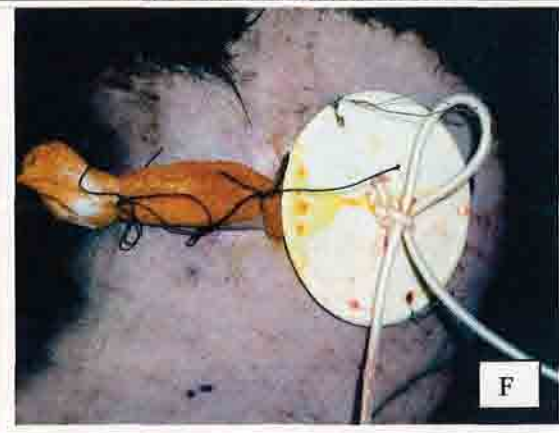
The omental incision was sutured and tubing was transfixed with a plastic disc



The plastic disc transfixed to tubing was placed intra-abdominally



Stainless steel sutures were applied through the abdominal wall to secure internal and external plate in proximity



The silicone catheter was tied in a shoe lace fashion to obstruct the intestinal lumen

3.2 THERAPEUTIC REGIMENS

3.2.1 SURGICAL TREATMENT

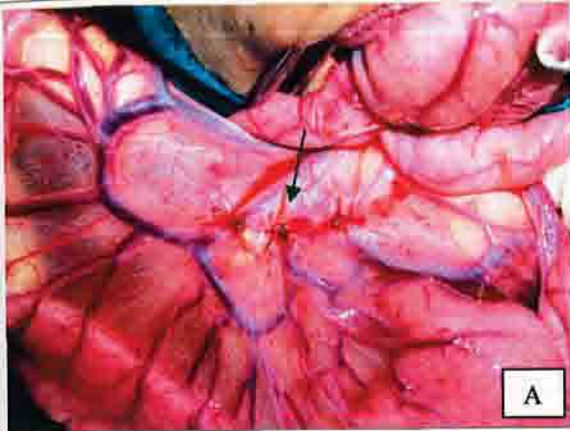
Following the creation of strangulated intestinal obstruction the corrective surgery was performed at 24 hours interval and following the creation of simple jejunal obstruction surgery was performed at 3rd day interval. The four animals each of group III, belonging to both simple and strangulated obstructions, were reopened from the same site of laparotomy incision to relieve the obstruction by resecting the non viable segment of the jejunum.

The criterion for resection of the segment was based on

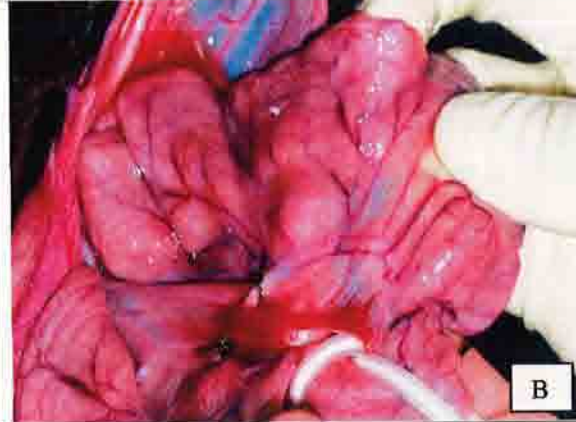
- (i) Colour of the affected segment
- (ii) Peristalsis of the affected segment
- (iii) Arterial pulsation in the jejunal segment
- (iv) Motility evoked by snapping the finger against intestinal wall

Following resection of the mesentery and affected segment of the jejunum, end to end anastomosis was achieved using a two layer anastomosis with a simple continuous pattern for the submucosal/mucosal layer, followed by a continuous Lambert in the seromuscular layer (Eggleston, 2001) using 2-0 Polyglactin 910 (Vicryl, Ethicon)) suture (Plate 3). The defect in the mesentery was closed in simple continuous suture pattern, using 2-0 Synthetic Polyglactin 910 suture. The absence of any leakage and luminal patency at the anastomotic site was checked by gentle milking of the intestinal contents. After rinsing the anastomosed segment of the jejunum with a lukewarm saline solution, it was replaced back into the abdominal cavity. The abdominal incision was closed in a routine manner.

PLATE 2: CREATION OF STRANGULATED JEJUNAL OBSTRUCTION IN CALVES



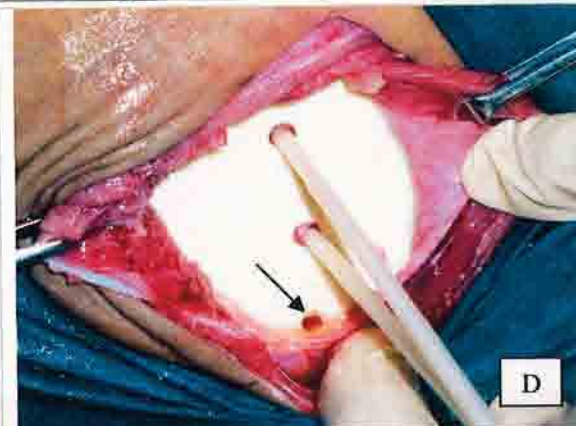
Ligature applied to strangulate the mesenteric vessels



Silicon tubing looped around the bowel segment to constrict the lumen



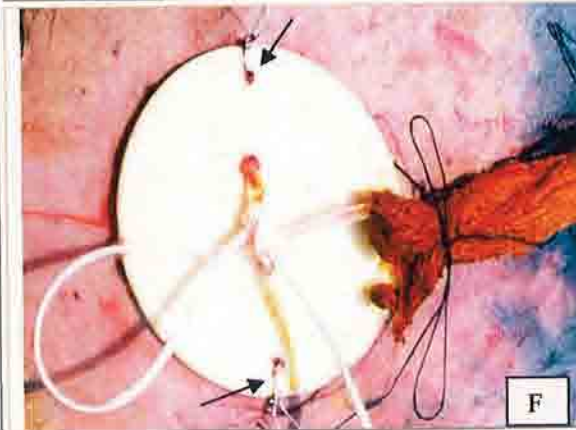
Omentum sutured as such and two additional holes are created to take the tubing out of the omental cavity



Internal disc looped with silicone tubing was placed intra-abdominally (Additional holes [→] were used to transfix the internal and external plate using stainless steel sutures



External disc looped with the tubing was fixed extra-abdominally



[→] indicates external disc transfixed with the internal disc using stainless steel sutures to immobilize the looped bowel

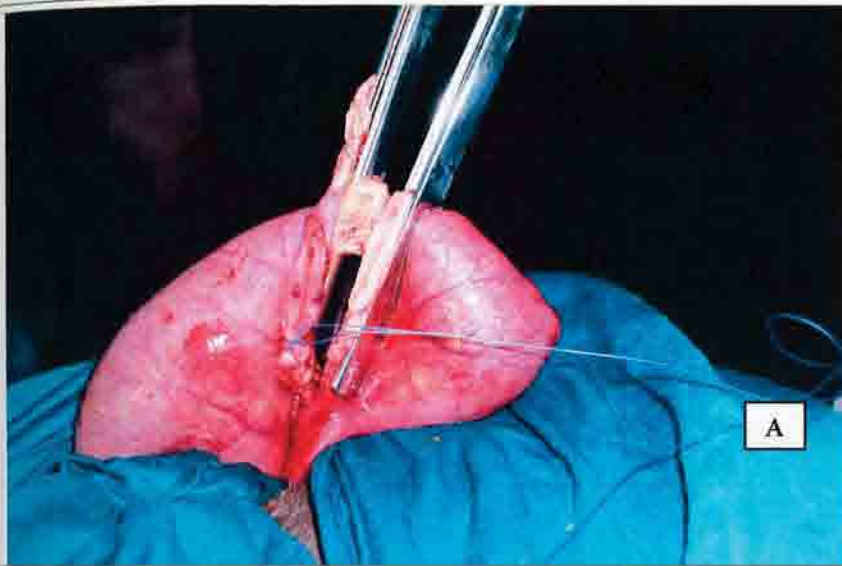
3.2.2 CONSERVATIVE TREATMENT

Following the creation of strangulated intestinal obstruction the medical treatment was instituted at 24 hours interval in the animals of group II and III and continued upto 96 hours and following the creation of simple jejunal obstruction, the treatment was instituted at 3rd day interval in the animals of group II and III and continued till 8th day interval.

The formulation of conservative treatment was based on the pathophysiological alterations observed in the animals of group I. In all of the animals, metabolic alkalosis accompanied by hypochloreaemia and hypokalemia was observed. For this reason, the following treatment schedule was instituted in the animals of group II and III.

1. 5 per cent Dextrose Normal Saline, IV.
2. Ringer Solution, IV.
3. Normal Saline solution, IV.
4. Amoxicillin & Cloxacillin (Inimox, Indian Immunologicals) - 8 mg/Kg BW., 8 hourly.
5. Potassium Chloride – 1gm/ liter of Nacl, IV.
6. Liver Extract/ B complex Vitamins(Belamyl, Sarabhai Zydus) - 5 ml alternate days, IM.
7. Vitamin C (Revici, Kee Pharma) - 5 ml, IV.
8. Dexamethasone (Dexona Vet, Cadilla Health Care) - 16 mg, (8 mg, IM and 8mg, IV).
9. Neostigmine (Myostgmin, Neon labs) 2 mg, IM at 6 hour interval, p.r.n. (Group III only).

**PLATE 3 : ENTEROANASTOMOSIS IN SIMPLE AND STRANGULATED
JEJUNAL OBSTRUCTION IN CALVES**



The viable part of intestine held together with intestinal clamps for enteroanastomosis



The luminal integrity was monitored by milking the contents from proximal to distal segments to rule out anastomotic leakage

10. *Sacchromyces cerevisiae*, *Lactobacillus sporogenes*, multiminerals and UGF's (Biobloom, Sarabhai Zydus) – 5 gm/day, orally.
11. Antimony Potassium tartrate and ferrous sulphate (Bovinum bolus, Sarabhai Zydus) – I bolus orally, b.i.d.
12. Ruminal cud - Oral transfaunation post operatively.

Composition of Ringer's solution

Sodium Chloride	-	0.84 per cent
Potassium chloride	-	0.03 per cent
Calcium chloride	-	0.033 per cent

The Ringer's solution, 5 per cent Dextrose normal saline and potassium chloride solution were prepared using pyrogen free water from Millipore filter, taking all the necessary precautions.

Formula for calculation of fluid therapy

The amount of intra-venous administered in the animals of group II and III was assessed as per the following formula

$$\text{Fluid required to overcome dehydration (ml)} \\ = \text{Patient PCV} \times 0.66 \times \text{body weight (in Kg)} \times 4 \quad (\text{Kumar, 1995})$$

3.3 COLLECTION OF SAMPLES

The blood, peritoneal and ruminal fluid samples were collected for analysis of various physio-chemical and haematobiochemical alterations.

1. At 0, 2, 3, 4, 6 and 8 days time intervals for simple obstruction.
2. At 0, 24, 48, 72 and 96 hours time interval for strangulated obstruction.

3.3.1 COLLECTION OF BLOOD

10 ml of blood was collected by jugular venipuncture and subjected for centrifugation at 5000 rpm for 15 minutes for separation of plasma. This plasma

was used for analysis of various biochemical constituents. One ml of blood was also collected separately and mixed with Ethylene Diamine Tetra Acetic Acid (EDTA) containing vial for haematological studies.

3.3.2 COLLECTION OF PERITONEAL FLUID

For collection of peritoneal fluid, the animals were comfortably secured in standing position. The area between the xiphoid and umbilicus was aseptically prepared for abdominocentesis. Local analgesia at the site was achieved by infiltration of 2 per cent lignocaine hydrochloride. A sharp 18 gauge, two inch long needle was advanced into the abdominal cavity through the skin in a perpendicular direction to the linea alba. The direction of needle was adjusted at different angles till the flow of clear to opalescent pale yellow fluid was noticed (Plate 4). The required amount of peritoneal fluid was collected in a heparinized vial for cytological and biochemical estimations. For microbiological test the peritoneal fluid was collected separately in sterilized syringe and sealed.

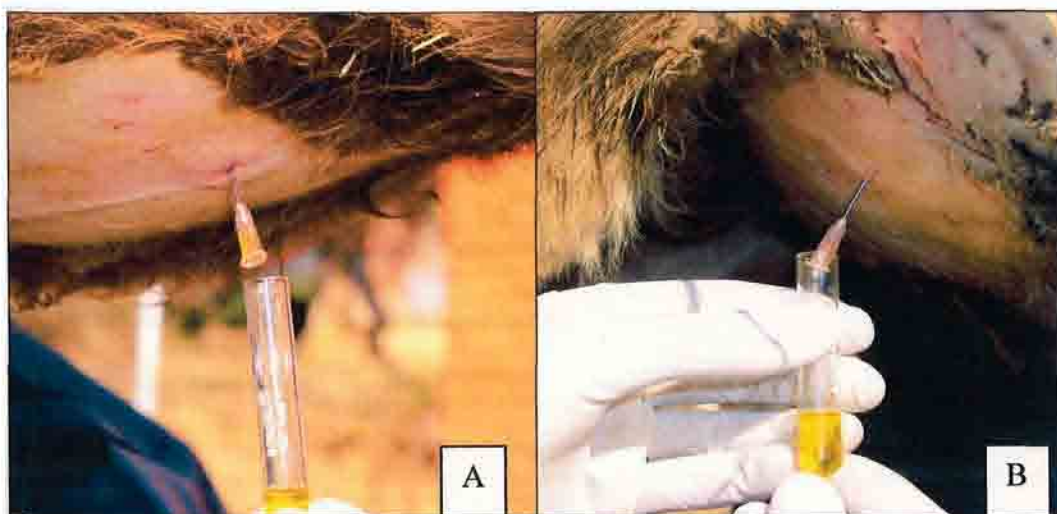


PLATE 4 COLLECTION OF PERITONEAL FLUID FROM
A. PRE UMBILICUS REGION
B. POST UMBILICUS REGION

3.3.3 COLLECTION OF RUMINAL FLUID

The ruminal fluid was aspirated before morning feeding by rumenocentesis through the left paralumbar fossa using a six inch long 18 gauge needle.

3.4 PARAMETERS INVESTIGATED

3.4.1 CLINICAL OBSERVATIONS

Various clinical parameters recorded daily were rectal temperature, heart rate, pulse rate, respiratory rate, capillary refill time, rumen motility, signs of abdominal pain, signs of dehydration (skin tenting time, eyes, status of muzzle and mucous membranes). The consistency, pattern and frequency of defecation along with feed and water intake, intestinal borborygmi, abdominal distention, muscular weakness and temperament of animal was recorded periodically.

3.4.2 PHYSICAL AND CYTOLOGICAL STUDIES

Colour, pH and status of microflora in the ruminal fluid samples were recorded. The rumen and peritoneal fluid pH was recorded using narrow range pH paper strips. The peritoneal fluid samples were also evaluated for its colour and cellular constituents.

3.4.3 HAEMATOLOGICAL STUDIES

The collected blood samples were subjected for estimation of haemoglobin packed cell volume, total leukocyte count, total erythrocyte count and differential leukocyte count as per standard methods.

3.4.4 BIOCHEMICAL STUDIES

3.4.4.1 PLASMA

The separated plasma was subjected for the analysis of various blood biochemical parameters like total plasma protein, glucose, urea nitrogen, creatinine, total bilirubin, chloride, inorganic phosphorus and calcium using

reagent kits of Autopak manufactured by Bayer Diagnostics on Semi computerized Blood Chemistry analyzer (RA 50 - Chemistry Analyzer, Bayer Diagnostics, Baroda, India). Plasma sodium and potassium were analyzed by Flame photometer (Systronics Mediflame 127).

Plasma enzymes like Alanine transaminase, Aspartate aminotransferase, Alkaline Phosphatase and Amylase were estimated using reagent kits of Autopak manufactured by Bayer Diagnostics on Semi computerized Blood Chemistry Analyzer.

3.4.4.2 PERITONEAL FLUID

The peritoneal fluid was observed for its colour and pH. The pH of peritoneal fluid was recorded using narrow range pH paper strips. The peritoneal fluid nucleated cell count was done as per the procedure described by Anderson *et al.* (1995).

Peritoneal fluid proteins, chloride, calcium and phosphorus were estimated using reagent kits of Autopak manufactured by Bayer Diagnostics on Semi computerized Blood Chemistry Analyzer. Peritoneal fluid sodium and potassium were analyzed by Flame photometer.

3.4.4.3 RUMINAL FLUID

The ruminal fluid pH was recorded by using narrow range pH paper strips. Chloride, calcium and phosphorus were estimated using reagent kits of Autopak manufactured by Bayer Diagnostics on Semi computerized Blood Chemistry Analyzer. Ruminal fluid sodium and potassium were analyzed by Flame photometer.

3.5 MICROBIOLOGICAL STUDIES

Isolation and culture sensitivity test of peritoneal fluid was carried out periodically.

3.6 ELECTROCARDIOGRAPHIC STUDIES

Electrocardiographic studies were performed on the animals for correlating the fluid and electrolyte imbalance with cardiovascular system. The bipolar base apex lead system was used with positive electrode placed at the centre of the sternal pad and negative electrode at posterior point of caudal border of scapula. The electrocardiograms were analyzed with calibration as 1mv =1cm, 0.03 T.C. and paper speed of 5/25mm/sec on physiograph (Polyrite, Recorder and Medicare System, INCO, Chandigarh).

3.7 HISTOPATHOLOGICAL STUDIES

Normal and diseased loop of intestine was subjected for histopathological study. Tissues fixed in 10 per cent neutral formalin were processed for paraffin section by standard histological techniques and were stained with routine haematoxylin-eosin method.

3.8 STATISTICAL ANALYSIS

The statistical analysis of data was carried out to evaluate the pathogenesis and effect of treatment in each group and within the group. The pre treatment values were compared with respective base values and the post treatment values were compared with the respective values on the day of treatment intervention in respective groups (3rd day in simple and 24 hours in strangulated intestinal obstruction). The data was subjected to repeated measures ANOVA, Student 't' test and Dunnett's test as per the requirement using Instat software (Graph Pad). The results were evaluated at 5 per cent and 1 per cent level of significance.

CHAPTER - IV

RESULTS

4.1 SIMPLE INTESTINAL OBSTRUCTION

4.1.1 CLINICAL OBSERVATIONS

4.1.1.1 PRE TREATMENT

All the animals showed mild signs of discomfort such as reflex guarding of abdomen while standing, grinding of teeth and mild pain during movement immediately after creation of simple intestinal obstruction. These symptoms abolished within 6-8 hours and thereafter the animals resumed their normal activity. The signs of mild abdominal pain reappeared after two days in two animals of group I and one animal of group II. These animals exhibited groaning, kicking at the abdomen and restlessness on 3rd day. The animals showed a tendency to lie down for a longer period of time and tried to seek the shady areas. These signs disappeared at 4th day onward and thereafter the symptoms of weakness were observed.

Defecation was normal on the day of creation of simple intestinal obstruction but as the time passed stools became insufficient and desiccated (Plate 5). On 3rd day the calves of all the three groups strained to defecate and passed very little amount of faeces with slightly off colour. On 4th day hard dehydrated dung balls were voided, which later at 5th day were laced with foul smelling mucoid discharge. At 6th day the faeces consisted more of the mucous discharge and very less dung. As the duration of obstruction continued from 7th day onward the mucous clogged the anus and mucoid plugs with fetid odour were voided. The frequency of defecation for initial four days was 2-3 times a day, which

PLATE 5: PATTERN AND CHARACTERISTIC OF FAECES AFTER CREATION OF SIMPLE JEJUNAL OBSTRUCTION IN CALVES



2nd day post creation



3rd day post creation



4th day post creation



5th day post creation



6th day post creation



7th day post creation



8th day post creation

later decreased to once a day. Urination was normal in the animals of group II and III but oligouria was noticed in group I as the duration of obstruction progressed.

The feed and water intake was normal in all the animals of three groups upto 3rd day of creation of obstruction, thereafter appetite reduced considerably but browsing on small amount of fodder was continued throughout the period of study till 7th day. Subsequently on 8th day all the animals of group I and II showed the tendency of prolonged recumbency and were totally reluctant to eat even when the fodder was offered.

Generalized frailty in terms of muscular debility was appreciated constantly in all the animals. These animals showed a weak footing on 2nd day of creation of intestinal obstruction which increased after 3rd day and eventually the animals showed a perceptible reluctance to move and a tendency to keep head down for an extended period of time. In the terminal part of study the animals were avert to stand and assumed sternal and often a lateral recumbency.

Rumen motility decreased to almost half at 2nd post obstruction day and later from 3rd day onward the rumen became totally atonic. In one animal, regurgitation of ruminal contents was noticed at 3rd day. Intestinal borborygmi were audible on auscultation upto 4th post obstruction day. However, these sounds diminished progressively from 6th day onward. A persisting absence of gut sounds was evident in the terminal part of study. Abdominal distention (Plate 6A) was seen on 2nd day onward which gradually increased with time and in later part of the study markedly distended abdomen posed much difficulty in carrying the weight of the animal by itself. This distention was bilateral and doughy on palpation.

There was a significant ($P < 0.01$) decrease in the rectal temperature at 3rd post obstruction day when compared to the base values in the animals of all three

PLATE 6: CLINICAL SIGNS DURING SIMPLE JEJUNAL OBSTRUCTION IN CALVES



Abdominal
distension



Eye ball
recession during
terminal stage
of obstruction

groups. The decrease in the rectal temperature also remained highly significant ($P < 0.01$) throughout the period of study in the animals of group I (Table 1). A significant increase in the respiratory rate was recorded in the animals of group II ($P < 0.05$) and this increase was highly significant in group III ($P < 0.01$) at 3rd post obstruction day when compared to their respective base values. However, a significantly higher ($P < 0.01$) increase in respiration rate was observed in the animals of the group I at 4th day onward when compared to its base value (Table 1). An increase in heart rate was seen in all the animals of three groups from 2nd post obstruction day which was significant ($P < 0.01$) on 3rd post obstruction day when compared to base values. The highly significant tachycardia persisted throughout the period of obstruction in the animals of group I (Table 1, Fig. 1). The pulse rate in the animals of all the three groups also showed the similar trend.

A comprehensive lassitude was evident in all the animals after 2-3 days following the creation of the obstruction. The hair coat appeared muffled, dry and the elasticity of skin gradually diminished. The eyes began to retract inside the orbital cavity and at 7th day the recession of eye balls was very much conspicuous (Plate 6B). These symptoms were exaggerated in the animals of group I as compared to group II. The ongoing deterioration in the condition of animals of group I was grievous, late at 8th day. The elasticity of skin was moderately decreased at 3rd post obstruction day and skin became almost inelastic at 6th day onward. Capillary refill time (CRT) was increased significantly at 3rd post obstruction day and was highly significant at 4th and 6th post obstruction day in the animals of group I (Table 2).

There was significant decrease in ruminal fluid pH in the animals of group I ($P < 0.05$) and II ($P < 0.01$) at 3rd post obstruction day when compared to base

TABLE 1: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON RECTAL TEMPERATURE, RESPIRATION RATE, HEART RATE AND PULSE RATE IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
RECTAL TEMPERATURE (°F)						
Group I	101.95 ±0.263 (n=4)	101.65 ±0.276 (n=4)	99.95** ±0.263 (n=4)	99.40** ±0.455 (n=4)	98.10** ±0.742 (n=4)	98.20 ^{N.I.} ±1.2 (n=2)
Group II	102.15 ±0.263 (n=4)	101.95 ±0.189 (n=4)	100.65** ±0.35 (n=4)	99.70 ±0.656 (n=4)	98.55 ^a ±0.922 (n=4)	97.33 ^b ±0.067 (n=3)
Group III	101.65 ±0.238 (n=4)	101.50 ±0.265 (n=4)	100.60** ±0.183 (n=4)	101.05 ±0.499 (n=4)	101.65 ^{de} ±0.427 (n=4)	101.80 ^{de} ±0.559 (n=4)
RESPIRATION RATE (/min)						
Group I	12.5 ±0.28 (n=4)	13.0 ±0.58 (n=4)	14.3 ±0.629 (n=4)	15.5** ±1.04 (n=4)	15.5** ±0.29 (n=4)	17.5 ^{N.I.} ±0.50 (n=2)
Group II	13.0 ±0.58 (n=4)	15.5 ±0.96 (n=4)	16.0* ±1.155 (n=4)	17.5 ±0.5 (n=4)	19.5 ^d ±0.96 (n=4)	21.3 ^{N.I.} ±1.33 (n=3)
Group III	12.5 ±0.5 (n=4)	13.5 ±1.5 (n=4)	16.5** ±0.957 (n=4)	14.5 ^f ±0.5 (n=4)	15.5 ^e ±0.5 (n=4)	14.0 ^f ±0.82 (n=4)
HEART RATE (/min)						
Group I	71.5 ±3.30 (n=4)	75.5 ±3.304 (n=4)	81.0** ±1.915 (n=4)	84.5** ±2.22 (n=4)	89.0** ±2.08 (n=4)	91.0 ^{N.I.} ±3.0 (n=2)
Group II	67.0 ±2.65 (n=4)	71.0 ±2.38 (n=4)	79.3** ±2.926 (n=4)	81.5 ±3.12 (n=4)	88.3 ^b ±4.37 (n=4)	96.0 ^a ±7.57 (n=3)
Group III	69.5 ±0.957 (n=4)	73.5 ±0.5 (n=4)	82.0** ±2.449 (n=4)	71.0 ^{bc} ±4.359 (n=4)	68.0 ^{bdf} ±2.16 (n=4)	66.5 ^{bce} ±3.76 (n=4)
PULSE RATE (/min)						
Group I	70.0 ±3.464 (n=4)	74.5 ±3.403 (n=4)	77.5* ±0.957 (n=4)	80.0** ±1.633 (n=4)	85.5** ±1.50 (n=4)	87.0 ^{N.I.} ±1.0 (n=2)
Group II	66.0 ±2.16 (n=4)	69.5 ±1.5 (n=4)	78.0** ±2.449 (n=4)	81.0 ±2.887 (n=4)	86.5 ^b ±3.862 (n=4)	93.3 ^b ±5.696 (n=3)
Group III	68.0 ±1.633 (n=4)	72.5 ±0.958 (n=4)	80.5** ±1.5 (n=4)	69.0 ^{bce} ±3.697 (n=4)	67.5 ^{bdf} ±2.062 (n=4)	65.5 ^{bcd} ±3.202 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

TABLE 2: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON CAPILLARY REFILL TIME AND RUMINAL FLUID pH IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
CAPILLARY REFILL TIME (sec)						
Group I	0.62 ±0.125 (n=4)	1.25 ±0.144 (n=4)	1.38* ±0.125 (n=4)	1.75** ±0.323 (n=4)	2.38** ±0.239 (n=4)	2.50 ^{N.I.} ±0.50 (n=2)
Group II	0.75 ±0.145 (n=4)	1.00 ±0.204 (n=4)	1.13 ±0.125 (n=4)	1.50 ±0.204 (n=4)	1.25 ^d ±0.144 (n=4)	1.67 ^{N.I.} ±0.167 (n=3)
Group III	0.63 ±0.125 (n=4)	1.00 ±0.204 (n=4)	1.13 ±0.239 (n=4)	1.00 ±0.204 (n=4)	0.88 ^d ±0.239 (n=4)	0.63 ^{df} ±0.125 (n=4)
RUMINAL FLUID pH						
Group I	7.25 ±0.144 (n=4)	7.05 ±0.166 (n=4)	6.85* ±0.087 (n=4)	6.63** ±0.075 (n=4)	6.55** ±0.086 (n=4)	6.20 ^{N.I.} ±0.200 (n=2)
Group II	7.13 ±0.125 (n=4)	6.93 ±0.075 (n=4)	6.65** ±0.050 (n=4)	6.55 ±0.15 (n=4)	6.43 ±0.075 (n=4)	6.23 ±0.145 (n=3)
Group III	6.85 ±0.086 (n=4)	6.65 ±0.05 (n=4)	6.60 ±0.058 (n=4)	6.73 ±0.075 (n=4)	6.85 ±0.866 (n=4)	6.77 ^e ±0.075 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

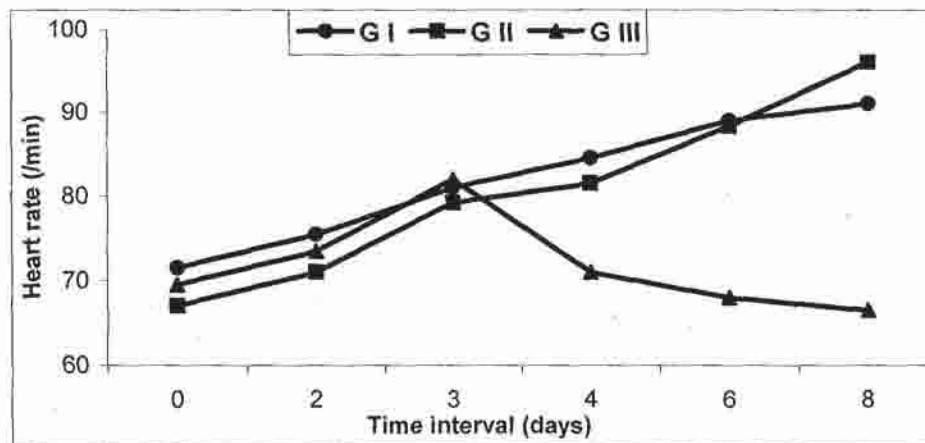
N.I. - Value not included for statistical analysis within group.

values (Table 2). The significant ($P < 0.01$) decreasing trend in the ruminal fluid pH continued throughout the period of study upto 6th day in the animals of group I. There was a moderate loss (++) of ruminal microflora at 3rd post obstruction day but as the duration of obstruction progressed, the loss became sluggish (+) at 4th and 5th day and afterwards complete loss (-) was evident.

4.1.1.2 POST TREATMENT

The animals of group II voided faeces upto 7th post obstruction day. The amount of faeces decreased and the mucoid content persistently increased even after institution of medical therapy at 3rd post obstruction day. Complete cessation of defecation was observed at 8th post obstruction day onward. The animals of

FIG. 1: VARIATIONS IN HEART RATE FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.



group III passed scanty faeces in the form of dung balls with a little amount of mucous after 4-5 hours of the surgical treatment. The consistency of faeces was watery on 4th day and later from 5th day onward the consistency was almost normal. The frequency of defecation was 3-4 times on 4th and 5th day and 2-3 times from 6th day onward. The stools passed immediately after the operation had a foul odour, which vanished in due course of time. The frequency of urination was 4-5 times a day in the animals of group II and III after the institution of treatment.

Feed and water intake was reduced progressively in the animals of group II. All the animals showed the tendency of occasional grasps at the fodder but after 8th post obstruction day the feed and water intake was absolutely absent, whereas the animals of group III resumed normal feed and water intake from 5th day onward.

Signs of muscular weakness persisted in all the animals of group II but the severity was less in comparison to the animals of group I. Signs of debility manifested by prolonged recumbency during night hours, inability to stand during early morning hours and adoption of frequent sternal recumbency with staggering

gait were noticed after 6th day and remained so till the completion of the study. The animals of group III did not show any such signs after the institution of surgical treatment except the animals were reluctant to walk on 4th post obstruction day. These signs attenuated as the animals recovered.

Complete cessation of ruminal movements was recorded in all the animals of group II even after the institution of medical treatment at 3rd day, whereas, in the animals of group III a feeble contraction of rumen was observed at 4th day when compared with 3rd post obstruction day interval. From 6th day onward appreciable ruminal motility (1/3min) was present but the rumen was hypotonic. Intestinal borborygmi in the animals of group II was audible on auscultation up to 5th post obstruction day and was clinically unappreciable from 6th day onward, whereas in the animals of group III the bowel sounds were very much appreciable at 3rd day onward. There was a progressive enlargement of the abdomen in the animals of group II and at the end of the study marked bilateral abdominal distention was observed.

A significant fall in the rectal temperature was recorded at 6th ($P < 0.05$) and 8th day ($P < 0.01$) in the animals of group II when compared to 3rd post obstruction day, whereas no significant alteration was noticed in the animals of group III when compared to day three. A significant increase in rectal temperature was recorded at 6th and 8th day in the animals of group III when compared with the animals of group I ($P < 0.01$) and II ($P < 0.05$). Respiratory rate did not register any significant change in the animals of group II and III but increasing trend was seen in the animals of group II during post treatment period. The comparison within groups showed a significant increase in respiration rate in animals of group II at 6th day when compared to group I. Whereas, a significant decrease ($P < 0.05$: 6th day and

$P < 0.01$: 8th day) in respiration rate was noticed in the animals of group III when compared with the animals of group II in the post treatment period (Table 1). The increase in heart rate in the animals of group II was significant at day 6th ($P < 0.01$) and 8th ($P < 0.05$) day when compared to 3rd post obstruction day. Contrariwise, heart rate decreased significantly ($P < 0.01$) during post treatment intervals in the animals of group III when compared to 3rd post obstruction day. The inter group comparison revealed a statistically significant decrease ($P < 0.01$) in heart rate attaining normalcy following treatment in group III when compared to group I and II. Similar changes were observed in pulse rate (Table 1).

The skin turgor was affected mildly during the entire post treatment period in the animals of group II. There was no sign of enophthalmia during the entire period of observation but muffling of hair coat and dryness of muzzle were constant findings in all the animals of group II. The animals of group III did not show such sign except the muzzle was dry at 4th and 5th day which became wet in later stages. Submandibular oedema was a constant finding in all the animals of group II and in one animal of group III. Conjunctival mucous membrane was pale in the terminal part of study in the animals of group II. Statistically non-significant increase in CRT was seen in group II whereas it was normal in post treatment period in the animals of group III. The increase in CRT in the animals of group I in comparison to group II and III was significant ($P < 0.01$) at 6th day and 8th day (Table 2).

Non-significant alteration in the pH of ruminal fluid was recorded in the animals of group II and III following the treatment. On comparative basis a significant shift in ruminal pH at 8th day interval was recorded in the animals of group III (Table 2). The loss of ruminal microflora in the animals of group II was

almost comparable to group I with a difference of slow pace in former, whereas in group III sluggish (+) to moderate (++) loss of micro flora was present upto 6th day and afterwards complete rejuvenation (+++) of microflora was observed.

4.1.2 HAEMATOLOGICAL ALTERATIONS

4.1.2.1 PRE TREATMENT

A significant ($P < 0.05$) increase in the haemoglobin concentration was recorded 3rd post obstruction day and it was highly significant ($P < 0.01$) at 4th and 6th post obstruction day in the animals of group I. This increase in haemoglobin was highly significant ($P < 0.01$) at 3rd post obstruction day in the animals of group II and III when compared to base values. Inconsistent variations were found during inter group comparisons (Table 3). An increase in PCV was seen from 2nd post obstruction day onward and this rise in PCV became significant ($P < 0.01$) at 3rd post obstruction day in the animals of all the three groups when compared with base values. In the animals of group I, a significant ($P < 0.01$) increase in the PCV was observed throughout the period of study (Table 3, Fig. 2).

A highly significant increase in total erythrocytic count (TEC) was found on 3rd post obstruction day in all the three groups when compared to their base values. A significant ($P < 0.01$) rise in TEC persisted throughout the period of study in the animals of group I (Table 4). Similar observations were recorded in total leukocytic count (TLC) in all the animals of three groups but leukocytosis was significant ($P < 0.01$) from 2nd post obstruction day when compared to base value (Table 4, Fig. 3). A significant ($P < 0.01$) neutrophilia was evident on 3rd day after the creation of simple intestinal obstruction. An unrelenting neutrophilia compared to base value persisted in the entire post obstruction period in the animals of Group I (Table 4). Contrariwise, a highly significant post obstruction decrease in

TABLE 3: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON HAEMOGLOBIN, PACKED CELL VOLUME AND TOTAL ERYTHROCYTE COUNT IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
HAEMOGLOBIN (g%)						
Group I	7.05 ±0.443 (n=4)	7.70 ±0.404 (n=4)	8.15* ±0.403 (n=4)	8.50** ±0.387 (n=4)	9.20** ±0.274 (n=4)	9.20 ^{N.I.} ±0.40 (n=2)
Group II	8.85 ±0.25 (n=4)	9.65 ±0.222 (n=4)	10.25** ±0.126 (n=4)	10.25 ^c ±0.419 (n=4)	10.30 ±0.519 (n=4)	10.87 ^c ±0.176 (n=3)
Group III	9.90 ±0.723 (n=4)	10.15 ±0.619 (n=4)	10.80** ±0.559 (n=4)	10.00 ^b ±0.668 (n=4)	9.90 ^b ±0.655 (n=4)	9.80 ^b ±0.627 (n=4)
PACKED CELL VOLUME (%)						
Group I	29.5 ±2.86 (n=4)	33.0 ±2.915 (n=4)	38.0** ±1.826 (n=4)	39.5** ±1.71 (n=4)	48.0** ±2.71 (n=4)	50.0 ^{N.I.} ±2.0 (n=2)
Group II	36.0 ±0.82 (n=4)	38.0 ±0.82 (n=4)	39.5** ±0.957 (n=4)	39.0 ±0.58 (n=4)	40.5 ±1.5 (n=4)	43.3 ^a ±1.764 (n=3)
Group III	31.8 ±2.72 (n=4)	34.8 ±1.109 (n=4)	37.5** ±1.893 (n=4)	33.0 ^{bce} ±1.732 (n=4)	31.5 ^{bdf} ±1.893 (n=4)	31.0 ^{bdf} ±1.732 (n=4)
TOTAL ERYTHROCYTE COUNT (millions/cu mm)						
Group I	5.26 ±0.736 (n=4)	5.53 ±0.69 (n=4)	6.12** ±0.804 (n=4)	6.31** ±0.797 (n=4)	6.74** ±7.387 (n=4)	5.95 ^{N.I.} ±0.530 (n=2)
Group II	6.32 ±0.073 (n=4)	6.43 ±0.025 (n=4)	6.51** ±0.061 (n=4)	6.56 ±0.062 (n=4)	6.65 ^b ±0.081 (n=4)	6.82 ^a ±0.075 (n=3)
Group III	6.02 ±0.438 (n=4)	6.24 ±0.324 (n=4)	6.49** ±0.359 (n=4)	5.94 ^b ±0.444 (n=4)	5.88 ^b ±0.490 (n=4)	5.89 ^b ±0.465 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

lymphocytic count was noticed on 3rd day following creation of simple intestinal obstruction in the animals of all the three groups when compared to base values. A highly significant decrease in lymphocytes was observed throughout the period of obstruction in the animals of group I (Table 4).

TABLE 4: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON TOTAL LEUKOCYTE COUNT, NEUTROPHILS AND LYMPHOCYTES IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
TOTAL LEUKOCYTIC COUNT (x 10³/cu mm)						
Group I	8.03 ±0.304 (n=4)	11.54** ±0.665 (n=4)	11.39** ±0.562 (n=4)	11.14** ±0.620 (n=4)	10.74** ±0.617 (n=4)	11.38 ^{N.I.} ±0.425 (n=2)
Group II	8.05 ±0.439 (n=4)	11.43** ±0.506 (n=4)	11.54** ±0.467 (n=4)	11.36 ±0.498 (n=4)	10.65 ^b ±0.478 (n=4)	10.22 ^{N.I.} ±0.261 (n=3)
Group III	7.10 ±0.417 (n=4)	10.14** ±0.626 (n=4)	10.26** ±0.515 (n=4)	9.68 ±0.542 (n=4)	9.05 ^b ±0.517 (n=4)	8.48 ^{bd} ±0.287 (n=4)
NEUTROPHILS (% of TLC)						
Group I	32.8 ±1.11 (n=4)	36.0 ±0.707 (n=4)	40.3** ±0.75 (n=4)	41.8** ±2.56 (n=4)	44.5** ±0.87 (n=4)	48.5 ^a ±0.5 (n=2)
Group II	30.5 ±1.04 (n=4)	33.5 ±0.866 (n=4)	39.3** ±1.315 (n=4)	38.8 ±1.70 (n=4)	43.0 ±0.41 (n=4)	44.0 ±1.155 (n=3)
Group III	30.0 ±1.83 (n=4)	35.0 ±1.472 (n=4)	37.8** ±0.250 (n=4)	38.0 ±1.35 (n=4)	36.0 ^{df} ±1.47 (n=4)	34.0 ^{adf} ±1.83 (n=4)
LYMPHOCYTES (% of TLC)						
Group I	65.8 ±1.44 (n=4)	62.7 ±1.031 (n=4)	57.8** ±1.031 (n=4)	56.8** ±2.56 (n=4)	54.0** ±1.00 (n=4)	50.5 ^{N.I.} ±1.5 (n=2)
Group II	68.8 ±0.85 (n=4)	65.8 ±0.75 (n=4)	58.8** ±1.031 (n=4)	59.0 ±0.58 (n=4)	55.5 ±0.65 (n=4)	55.7 ^a ±1.453 (n=3)
Group III	68.5 ±2.06 (n=4)	63.5 ±1.258 (n=4)	60.3** ±0.629 (n=4)	60.3 ±1.79 (n=4)	62.8 ^{df} ±1.31 (n=4)	65.5 ^{ade} ±1.94 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

4.1.2.2 POST TREATMENT

The animals of group II during post treatment period showed a mild increase in haemoglobin (Hb) concentration which was statistically non-significant when compared with 3rd post obstruction day, whereas, in the animals of group III,

a significant ($P < 0.01$) decrease in Hb concentration was noticed when compared with its 3rd day values (Table 3). There was non-significant alteration in PCV values at 4th and 6th day in the animals of group II when compared to its 3rd post obstruction day but significant ($P < 0.05$) increase in PCV was recorded at 8th day in the animals of group II. The animals of group III showed a highly significant decrease in PCV during the entire post treatment period when compared to 3rd post obstruction day. The inter group comparison of group I and II with group III revealed statistically significant ($P < 0.01$) decrease at corresponding intervals (Table 3, Fig. 2).

A significant increase in TEC at 6th ($P < 0.01$) and significant increase at 8th ($P < 0.05$) day was recorded in the animals of group II when compared to 3rd post obstruction day, whereas the TEC decreased significantly ($P < 0.01$) following surgical treatment when compared to 3rd post obstruction day in the animals of group III (Table 4). Similar trends were found in TLC but on comparative basis the animals of group III showed significant ($P < 0.01$) decrease in leukocytic count at 8th day (Table 4, Fig. 3). No significant change in neutrophils was appreciated both in group II and III upto 6th day following treatment but, significant reduction in neutrophils was seen at 8th day in the animals of group III as compared to its 3rd post obstruction day value. The inter group comparison showed a statistically significant ($P < 0.01$) tendency of normalization in the values of neutrophils at 6th and 8th days in the animals of group III (Table 4). Following conservative therapy a gradual decline in the lymphocytic count was noticed which was significantly ($P < 0.05$) low at 8th day when compared to 3rd post obstruction day in the animals of group II. The inter group comparison illustrated a statistically significant ($P < 0.01$) tendency of normalization in the values of lymphocytes at 6th and 8th

FIG. 2: VARIATIONS IN PACKED CELL VOLUME FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.

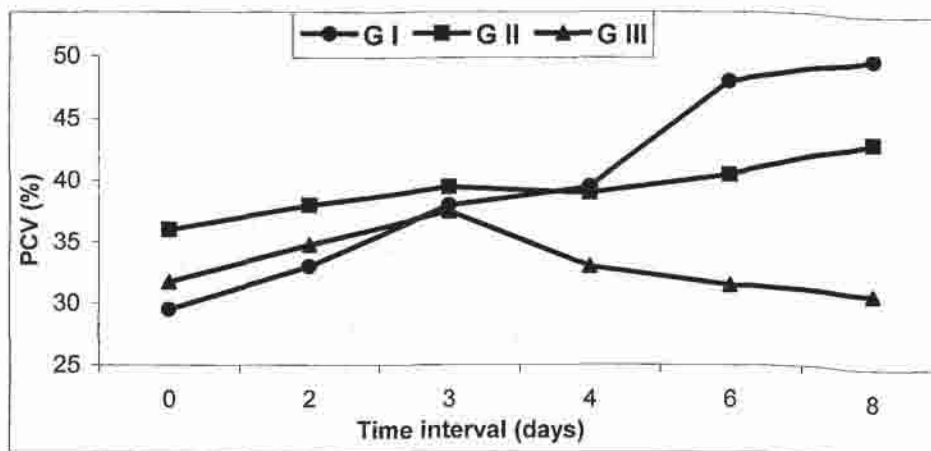
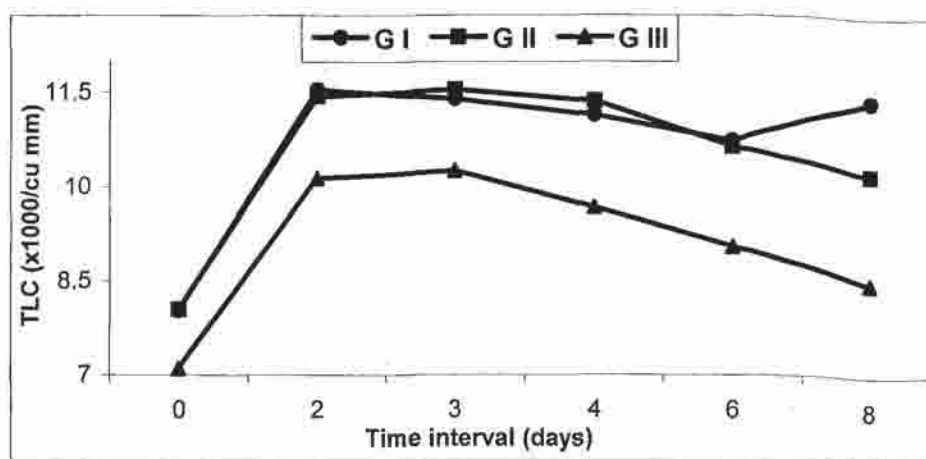


FIG. 3: VARIATIONS IN TOTAL LEUKOCYTIC COUNT FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.



in the animals of group III (Table 4).

4.1.3 BIOCHEMICAL CHANGES IN PLASMA

4.1.3.1 PRE TREATMENT

Blood glucose concentration did not show any significant alteration throughout the period of study in the animals of group I, whereas significant ($P < 0.05$) increase in blood glucose concentration was noticed at 2nd post obstruction day in the animals of group II and significant ($P < 0.01$) increase at 2nd

and 3rd post obstruction day in the animals of group III when compared to base values (Table 5).

There was an increase in total plasma protein from on 2nd day onward following creation of simple intestinal obstruction and it was found significantly high ($P < 0.01$) at 3rd post obstruction day in all the animals of three groups in comparison to base values. A highly significant increase in the total plasma protein concentration continued throughout the period of study in the animals of group I (Table 5, Fig. 4).

The blood urea nitrogen concentration increased significantly ($P < 0.05$) at 3rd post obstruction day in the animals of all the three groups. This concentration remained constantly higher ($P < 0.01$) in the entire post obstruction period in the animals of group I (Table 5). The plasma creatinine concentration increased significantly in animals of group I ($P < 0.01$), group II ($P < 0.05$) and III ($P < 0.05$) at 3rd day following creation of simple intestinal obstruction. In the animals of group I significant ($P < 0.01$) rise in the concentration of plasma creatinine was observed throughout the period of study (Table 5).

The increase in plasma total bilirubin concentration was significant ($P < 0.05$) at 4th post obstruction day and subsequently it was significantly ($P < 0.01$) higher on 6th and 8th post obstruction day in the animals of group I when compared with base value. There was no alteration in plasma total bilirubin in the animals of group II and III at 3rd post obstruction day when compared with base values (Table 6).

A significant ($P < 0.01$) decrease in plasma sodium concentration was recorded in the animals of group I at 3rd, 4th and 6th day following the creation of simple intestinal obstruction (Table 7). As compared to base value a significant decline ($P < 0.01$) in plasma potassium concentration was recorded at 2nd and 3rd

TABLE 5: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON GLUCOSE, TOTAL PLASMA PROTEINS, BLOOD UREA NITROGEN AND CREATININE IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
GLUCOSE (mg/dL)						
Group I	65.5 ±3.50 (n=4)	69.5 ±2.50 (n=4)	67.8 ±2.25 (n=4)	63.5 ±4.113 (n=4)	70.75 ±8.097 (n=4)	67.0 ±9.00 (n=2)
Group II	70.5 ±3.43 (n=4)	84.3* ±4.33 (n=4)	80.5 ±2.217 (n=4)	73.0 ±7.69 (n=4)	72.5 ±13.45 (n=4)	70.0 ±16.17 (n=3)
Group III	68.3 ±5.14 (n=4)	83.8** ±3.119 (n=4)	85.0** ±2.082 (n=4)	88.5 ^d ±2.754 (n=4)	79.8 ±2.84 (n=4)	83.0 ±2.38 (n=4)
TOTAL PLASMA PROTEIN (mg/dL)						
Group I	6.60 ±0.408 (n=4)	7.22 ±0.098 (n=4)	8.65** ±0.515 (n=4)	8.96** ±0.520 (n=4)	10.77** ±0.4250 (n=4)	13.00* ±0.200 (n=2)
Group II	6.88 ±0.179 (n=4)	7.25 ±0.065 (n=4)	7.63** ±0.103 (n=4)	7.75 ±0.155 (n=4)	8.15 ^{bd} ±0.125 (n=4)	8.87 ^{ad} ±0.233 (n=3)
Group III	6.63 ±0.132 (n=4)	7.03 ±0.165 (n=4)	7.85** ±0.156 (n=4)	7.35 ^{ac} ±0.096 (n=4)	6.95 ^{bdf} ±0.132 (n=4)	6.63 ^{bdf} ±0.179 (n=4)
BLOOD UREA NITROGEN (mg/dL)						
Group I	11.37 ±0.67 (n=4)	19.98 ±3.916 (n=4)	26.10* ±5.5 (n=4)	44.80** ±2.042 (n=4)	72.45** ±3.638 (n=4)	92.35* ±4.05 (n=2)
Group II	8.18 ±0.698 (n=4)	14.35 ±4.2 (n=4)	14.35* ±4.2 (n=4)	35.83 ^{bc} ±1.485 (n=4)	51.78 ^{bd} ±1.44 (n=4)	63.83 ^a ±1.88 (n=3)
Group III	10.33 ±0.206 (n=4)	19.38 ±4.651 (n=4)	24.53* ±4.386 (n=4)	24.23 ^{df} ±0.994 (n=4)	14.95 ^{df} ±1.135 (n=4)	12.33 ^{adf} ±0.541 (n=4)
CREATININE (mg/dL)						
Group I	0.96 ±0.165 (n=4)	2.10 ±0.434 (n=4)	3.08** ±0.325 (n=4)	3.30** ±0.353 (n=4)	4.45** ±0.206 (n=4)	6.25* ±0.150 (n=2)
Group II	1.26 ±0.083 (n=4)	1.46 ±0.039 (n=4)	1.69* ±0.081 (n=4)	2.33 ^{bc} ±0.084 (n=4)	2.58 ^{bd} ±0.1025 (n=4)	3.03 ^{bc} ±0.57 (n=3)
Group III	0.98 ±0.283 (n=4)	1.54 ±0.333 (n=4)	1.74* ±0.209 (n=4)	2.02 ^{ce} ±0.075 (n=4)	1.19 ^{df} ±0.017 (n=4)	1.06 ^{af} ±0.171 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

TABLE 6: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON PLASMA TOTAL BILIRUBIN (mg/dL) IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
Group I	0.08 ±0.1633 (n=4)	0.13 ±0.236 (n=4)	0.15 ±0.015 (n=4)	0.23* ±0.170 (n=4)	0.33** ±0.017 (n=4)	0.43** ±0.015 (n=2)
Group II	0.10 ±0.103 (n=4)	0.14 ±0.014 (n=4)	0.15 ±0.011 (n=4)	0.21 ^a ±0.108 (n=4)	0.24 ^b ±0.119 (n=4)	0.30 ^a ±0.12 (n=3)
Group III	0.11 ±0.015 (n=4)	0.15 ±0.019 (n=4)	0.16 ±0.014 (n=4)	0.16 ±0.005 (n=4)	0.13 ^d ±0.009 (n=4)	0.11 ^{ad} ±0.010 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

day following induction of simple intestinal obstruction and this decreasing trend continued ($P < 0.01$) throughout the period of study in the animals of group I (Table 7, Fig. 5). Likewise a significant hypochloraemia was noticed on 2nd post obstruction day ($P < 0.05$: group I and $P < 0.01$: group II and III) and it was highly significant ($P < 0.01$) at 3rd day in the animals of all the three groups in comparison to their base values. A significant ($P < 0.01$) hypochloraemia was observed in the animals of group I throughout the period of study (Table 7, Fig. 6). Inconsistent changes were recorded in plasma calcium and phosphorus concentration in all the animals of three groups when compared to base values (Table 7, 8).

Plasma Alkaline phosphatase, aspartate amino transferase and alanine amino transferase concentrations were elevated ($P < 0.05$) at 3rd day following creation of simple intestinal obstruction in the animals of all three groups when compared to their base values (Table 9, Fig. 7). The concentration of these enzymes remained significantly ($P < 0.01$) elevated during the entire course of obstruction in the animals of group I. Serum amylase concentration in comparison

TABLE 7: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON PLASMA SODIUM, POTASSIUM, CHLORIDE AND CALCIUM IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
SODIUM (mEq/L)						
Group I	144.0 ±0.81 (n=4)	140.5 ±1.5 (n=4)	133.5** ±0.957 (n=4)	131.0** ±0.57 (n=4)	128.0** ±0.81 (n=4)	119.0 ^{N.I.} ±2.00 (n=2)
Group II	145.0 ±0.577 (n=4)	142.5 ±0.957 (n=4)	142.0 ±0.817 (n=4)	142.0 ^d ±1.63 (n=4)	144.0 ^d ±1.41 (n=4)	142.0 ^d ±1.155 (n=3)
Group III	144.0 ±0.817 (n=4)	143.5 ±0.5 (n=4)	143.5 ±0.5 (n=4)	145.0 ^d ±0.577 (n=4)	142.0 ^d ±0.82 (n=4)	143.0 ^d ±0.58 (n=4)
POTASSIUM (mEq/L)						
Group I	4.95 ±0.493 (n=4)	3.93** ±0.144 (n=4)	3.33** ±0.170 (n=4)	3.13** ±0.170 (n=4)	2.93** ±0.201 (n=4)	2.65 ^{N.I.} ±0.150 (n=2)
Group II	4.98 ±0.296 (n=4)	4.35** ±0.222 (n=4)	4.00** ±0.216 (n=4)	3.80 ^c ±0.163 (n=4)	3.53 ^c ±0.103 (n=4)	3.25 ^c ±0.065 (n=3)
Group III	5.05 ±0.171 (n=4)	4.35 ±0.15 (n=4)	4.08 ±0.125 (n=4)	4.13 ^d ±0.180 (n=4)	4.38 ^{df} ±0.132 (n=4)	4.63 ^{df} ±0.085 (n=4)
CHLORIDE (mEq/L)						
Group I	99.08 ±0.953 (n=4)	87.00* ±1.861 (n=4)	75.25** ±4.469 (n=4)	71.78** ±2.918 (n=4)	68.38** ±2.363 (n=4)	65.90* ±1.4 (n=2)
Group II	102.70 ±1.914 (n=4)	87.60** ±0.964 (n=4)	75.10** ±3.712 (n=4)	92.48 ^{bd} ±0.819 (n=4)	91.55 ^{bd} ±0.839 (n=4)	94.40 ^d ±2.65 (n=3)
Group III	104.40 ±0.406 (n=4)	85.28** ±3.023 (n=4)	75.00** ±3.584 (n=4)	94.13 ^{bd} ±1.365 (n=4)	98.75 ^{bd} ±0.928 (n=4)	100.80 ^{bde} ±0.408 (n=4)
CALCIUM (mg/dL)						
Group I	6.61 ±0.987 (n=4)	6.78 ±0.545 (n=4)	6.63 ±0.487 (n=4)	6.70 ±0.531 (n=4)	6.58 ±0.448 (n=4)	6.45 ±0.15 (n=2)
Group II	6.65 ±0.863 (n=4)	6.68 ±0.496 (n=4)	6.58 ±0.347 (n=4)	6.50 ±0.492 (n=4)	6.65 ±0.676 (n=4)	6.80 ±0.702 (n=3)
Group III	6.90 ±0.123 (n=4)	6.95 ±0.065 (n=4)	6.78 ±0.075 (n=4)	6.80 ±0.71 (n=4)	6.83 ±0.063 (n=4)	6.88 ±0.048 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

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TABLE 8: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON PLASMA PHOSPHORUS IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
Group I	5.42 ±0.259 (n=4)	5.45 ±0.232 (n=4)	5.40 ±0.173 (n=4)	5.45 ±0.171 (n=4)	5.58 ±0.193 (n=4)	5.75 ±0.050 (n=2)
Group II	5.85 ±0.484 (n=4)	5.8 ±0.528 (n=4)	5.83 ±0.492 (n=4)	5.78 ±0.521 (n=4)	5.95 ±0.609 (n=4)	5.90 ±0.764 (n=3)
Group III	5.58 ±0.278 (n=4)	5.73 ±0.319 (n=4)	5.60 ±0.418 (n=4)	5.63 ±0.364 (n=4)	5.55 ±0.353 (n=4)	5.75 ±0.226 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

to base value was high in the animals of group I ($P<0.05$), II and III ($P<0.01$) at 3rd day following creation of intestinal obstruction. A constant rise ($P<0.01$) in serum amylase concentration persisted throughout the period of obstruction in the animals of group I (Table 9, Fig. 8).

4.1.3.2 POST TREATMENT

Variable changes in plasma glucose concentration during post treatment period were found inconclusive. Overall, an increase in the glucose concentration was recorded both in the animals of group II and III. The inter group comparison revealed significant ($P<0.01$) increase in glucose concentration in animals of group III as compared to group I at 4th day (Table 5).

A constant increase in total plasma protein concentration was recorded in the entire post treatment period at 6th ($P<0.01$) and 8th ($P<0.05$) days in the animals of group II when compared to 3rd post obstruction day value. There was a significant decrease ($P<0.05$) in plasma protein concentration in the animals of

TABLE 9: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON PLASMA ALKP, AST, ALT AND AMYLASE IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
ALKALINE PHOSPHATASE (IU/L)						
Group I	99.75 ±2.955 (n=4)	103.00 ±2.739 (n=4)	110.50* ±2.901 (n=4)	125.75** ±1.750 (n=4)	141.00** ±1.291 (n=4)	150.50* ±1.5 (n=2)
Group II	88.25 ±6.811 (n=4)	100.75 ±3.301 (n=4)	113.00* ±4.203 (n=4)	126.00 ^a ±4.243 (n=4)	133.75 ^b ±4.049 (n=4)	147.30 ^a ±2.667 (n=3)
Group III	98.75 ±4.09 (n=4)	107.50 ±2.958 (n=4)	114.25* ±4.404 (n=4)	130.00 ^b ±2.483 (n=4)	116.75 ^d ±1.887 (n=4)	106.75 ^{df} ±2.562 (n=4)
AST (IU/L)						
Group I	81.0 ±8.81 (n=4)	92.5 ±5.852 (n=4)	104.3* ±8.33 (n=4)	121.5** ±8.38 (n=4)	151.0** ±4.44 (n=4)	189.0 ^{N.I.} ±3.0 (n=2)
Group II	85.0 ±2.517 (n=4)	97.5 ±1.708 (n=4)	104.8* ±3.902 (n=4)	119.5 ±2.754 (n=4)	148.0 ^b ±6.48 (n=4)	166.7 ^a ±9.615 (n=3)
Group III	80.5 ±1.323 (n=4)	92.3 ±3.705 (n=4)	102.5* ±5.752 (n=4)	117.0 ±3.512 (n=4)	87.0 ^{df} ±3.536 (n=4)	57.5 ^{bdf} ±2.96 (n=4)
ALT (IU/L)						
Group I	29.3 ±4.99 (n=4)	39.8 ±2.097 (n=4)	47.0* ±3.109 (n=4)	60.0** ±5.148 (n=4)	76.3** ±4.131 (n=4)	101.0* ±3.00 (n=2)
Group II	29.63 ±3.092 (n=4)	38.0 ±3.028 (n=4)	44.5* ±3.014 (n=4)	55.5 ±2.630 (n=4)	69.5 ^b ±2.217 (n=4)	82.0 ^a ±5.292 (n=3)
Group III	28.5 ±0.956 (n=4)	35.8 ±2.78 (n=4)	43.0* ±4.021 (n=4)	31.8 ^{df} ±2.25 (n=4)	27.5 ^{adf} ±1.555 (n=4)	24.5 ^{bdf} ±1.555 (n=4)
AMYLASE (IU/L)						
Group I	19.00 ±2.858 (n=4)	24.50 ±2.986 (n=4)	29.25* ±3.224 (n=4)	31.50** ±3.227 (n=4)	40.25** ±1.75 (n=4)	48.50* ±4.500 (n=2)
Group II	18.25 ±1.887 (n=4)	22.25 ±0.479 (n=4)	26.26** ±2.097 (n=4)	28.50 ^b ±2.179 (n=4)	32.25 ^{bc} ±2.097 (n=4)	35.33 ^a ±3.712 (n=3)
Group III	18.50 ±1.555 (n=4)	23.75 ±1.436 (n=4)	29.00** ±1.225 (n=4)	30.50 ±1.893 (n=4)	25.75 ^{ade} ±1.315 (n=4)	22.00 ^{bde} ±1.08 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

group III at 4th day and this decrease was significantly higher ($P<0.01$) at 6th and 8th day. The animals of group III revealed a statistically significant ($P<0.05$: 4th day and $P<0.01$: 6th and 8th day) decrease of plasma protein during post treatment period when compared to the corresponding intervals of animals of group I. Similarly, this decrease was significant ($P<0.01$) in group III in comparison to group II at 6th and 8th day of recording after treatment (Table 5, Fig. 4).

Blood urea nitrogen in comparison to 3rd day's value remained significantly elevated (4th and 6th day: $P<0.01$ and 8th day: $P<0.05$) even after institution of conservative therapy in the animals of group II. However, significant ($P<0.05$) decrease in BUN concentration was noticed at 8th day as compared to 3rd post obstruction day in the animals of group III. The inter-group comparison inferred a constantly significant ($P<0.05$) rise in the concentration of BUN in the animals of group II when compared to group I. Whereas, animals of group III showed highly significant ($P<0.01$) recovery in BUN values after surgical correction when compared to animals of group I and II (Table 5). The plasma creatinine concentration remained significantly ($P<0.01$) higher in the animals of group II throughout the post treatment period when compared to 3rd post obstruction day. On the contrary, after surgical treatment in the animals of group III the plasma creatinine concentration showed decreasing trend and this decrease was significant ($P<0.05$) at 8th day. The significant ($P<0.05$) increase in plasma creatinine concentration was gradual in group II in comparison to group I. Whereas, a significant (4th day: $P<0.05$ and 6th day: $P<0.01$) decrease in plasma creatinine concentration was recorded in group III when compared to corresponding values of other two groups (Table 5).

The plasma total bilirubin concentration remained significantly high ($P < 0.05$: 4th day and $P < 0.01$: 6th day) during entire treatment period when compared to 3rd post obstruction day. In the animals of group II reduction in plasma bilirubin concentration was significant ($P < 0.05$) at 8th day in the animals of group III when compared to 3rd post obstruction day. The animals of group III showed significant ($P < 0.01$) lowering of bilirubin concentration in comparison to group I at 6th and 8th day (Table 6).

The blood sodium concentration remained in normal range in the animals of group II and III in comparison to respective 3rd post obstruction day. A significant ($P < 0.01$) increase in sodium concentration was recorded in the animals of group II and III when compared to group I in the post treatment period (Table 7). Similarly, a slow pace drop in the plasma potassium concentration was evident in the animals of group II when compared to 3rd post obstruction day. Whereas, in the period after surgery as compared to 3rd post obstruction day a gradual revival of plasma potassium concentration (6th day: $P < 0.05$ and 8th day: $P < 0.01$) was observed in the animals of group III. A significant ($P < 0.01$) increase in plasma potassium concentration towards normalization was noticed during the post treatment period in the animals of group III when compared to group I and II (Table 7, Fig. 5). A significant ($P < 0.01$) increase in plasma chloride concentration at 4th and 6th day persisted in the animals of group II when compared to its 3rd day value. Whereas, a significant ($P < 0.01$) recuperation in plasma chloride concentration (Table 7, Fig. 6) in the animals of group III occurred during entire post surgical period when compared to 3rd post obstruction day. The inter-group comparison revealed that decline in plasma chloride concentration was significant ($P < 0.01$) in the untreated animals of group I in comparison to group II and III. Furthermore,

FIG. 4: VARIATIONS IN TOTAL PLASMA PROTEIN FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.

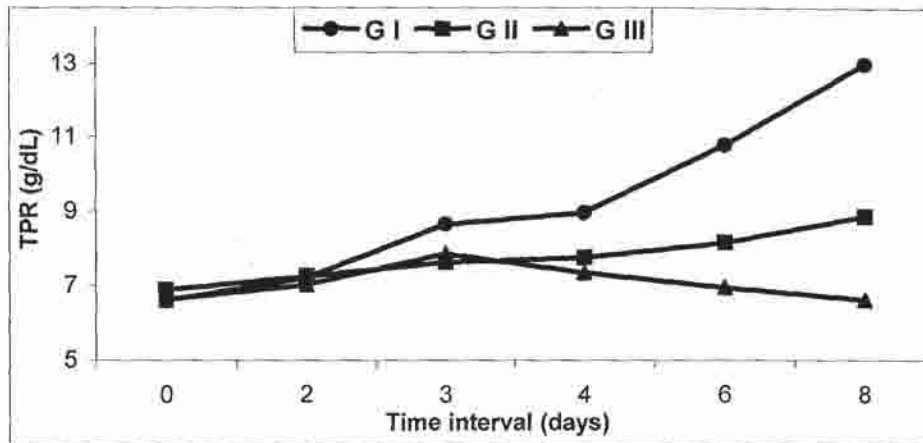


FIG. 5: VARIATIONS IN PLASMA POTASSIUM FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.

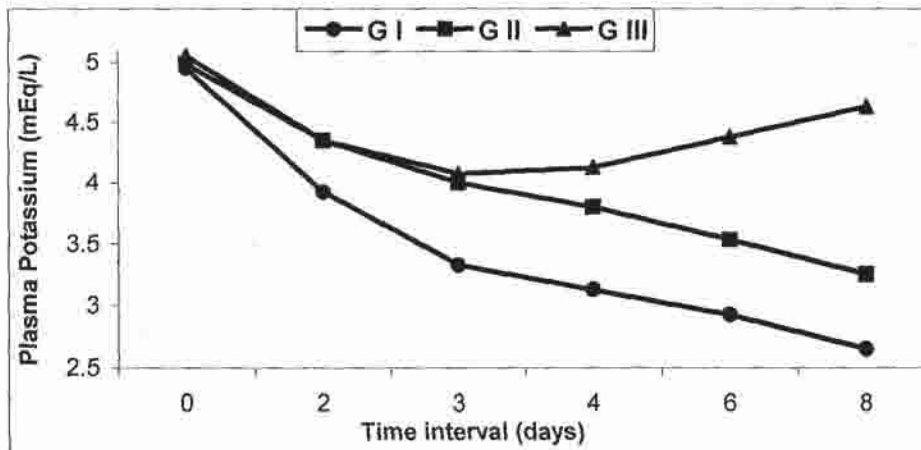
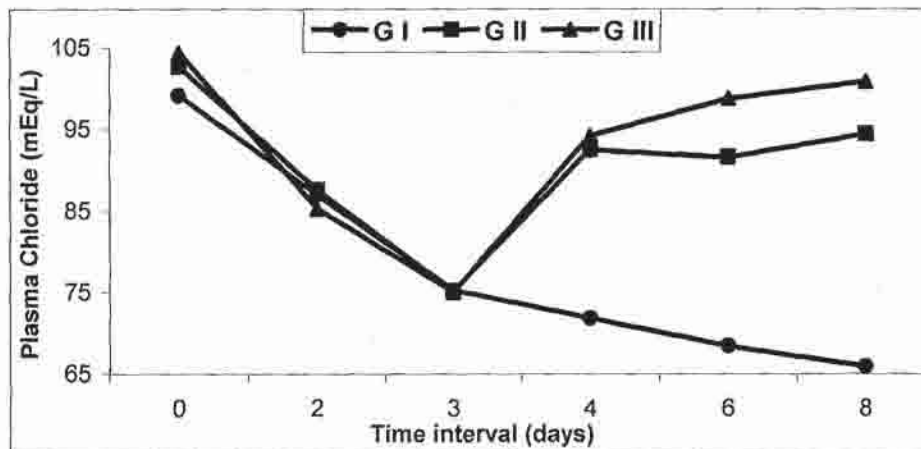


FIG. 6: VARIATIONS IN PLASMA CHLORIDE FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.



the comparison of group II and III depicted that the recovery of chloride concentration was significant ($P < 0.01$) in the animals of group III in comparison to group II at 6th day. Incoherent alterations in the values of plasma calcium and phosphorus following medical and surgical treatment were observed in the animals of group II and III (Table 7, 8).

A significant to highly significant rise in plasma Alkaline phosphatase (ALKP) was recorded throughout the entire post treatment period in the animals of group II when compared to 3rd post obstruction day value. The ALKP remained higher ($P < 0.01$) on the day after surgery when compared to 3rd post obstruction day and thereafter it decreased and was almost comparable to its base value in the terminal part of observations in the animals of group III. There was marked ($P < 0.01$) improvement in ALKP concentration at 6th and 8th day in the animals of group III when compared to the corresponding intervals of group I and II (Table 9, Fig. 7). The plasma AST and ALT concentration was higher in the animals of group II at 6th ($P < 0.01$) and 8th ($P < 0.05$) days when compared to 3rd post obstruction day. Whereas a significant drop in AST concentration (8th day: $P < 0.01$) and ALT (6th day: $P < 0.05$ and 8th day: $P < 0.01$) was noticed during post treatment period in the animals of group III when compared to respective 3rd post obstruction day values. A significant ($P < 0.01$) decline in AST and ALT values was seen at the 6th and 8th post treatment day in the animals of group III when compared to group I and II (Table 9). An unrelenting increase ($P < 0.01$) in plasma amylase concentration persisted continuously during post treatment period in the animals of group II as compared to 3rd post obstruction day. Following surgery the decrease in plasma amylase concentration started at 6th ($P < 0.05$) day onward and was significant ($P < 0.01$) at 8th day. Comparison within groups showed gradual increase

FIG. 7: VARIATIONS IN PLASMA ALKALINE PHOSPHATASE FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.

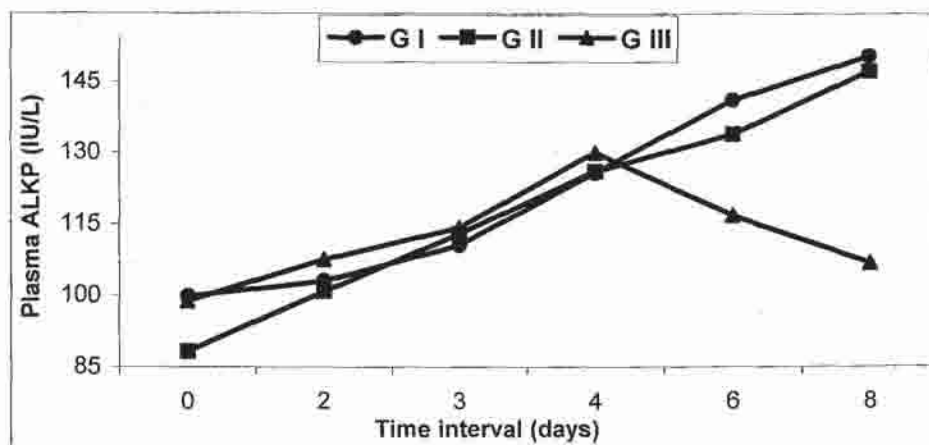
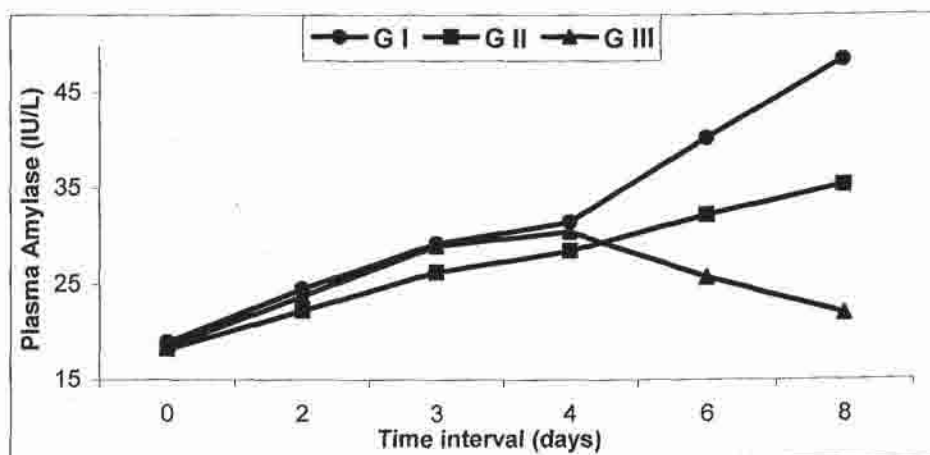


FIG. 8: VARIATIONS IN PLASMA AMYLASE FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.



($P < 0.05$; 6th day) in the amylase concentration of the animals of group II when compared to group I. The improvement in plasma amylase concentration following surgery in group III was significant ($P < 0.01$) in comparison to group I and it was significant ($P < 0.05$) in comparison to group II at 6th and 8th day respectively (Table 9, Fig. 8).

4.1.4 CHANGES IN PERITONEAL FLUID

4.1.4.1 PRE TREATMENT

The peritoneal fluid in the animals of group I was straw colour before creation of simple intestinal obstruction. It remained same upto 2nd post obstruction day but later at 3rd and 4th day, slight increase in the yellowish tinge was seen which discoloured to deep yellow from 6th day onward (Plate 7). A slight decrease in the pH was observed on narrow range pH paper in the animals of group III, however, there were no alterations in peritoneal fluid pH in the animals of group I and II (Table 10).

A significant ($P < 0.01$) increase in the peritoneal fluid total protein and cell count was recorded throughout the period of study when compared to base values in the animals of group I. The significant ($P < 0.01$) rise in proteinaceous (3rd day) and cellular components (2nd and 3rd day) was noticed in all the animals of three groups following creation of simple intestinal obstruction when compared to their base values (Table 10, Fig. 9, 10).

A significant decrease in peritoneal fluid sodium concentration was recorded at 3rd day in the animals of group I ($P < 0.05$) and III ($P < 0.01$) when compared to their '0' hour concentrations respectively. However, this decline in sodium concentration was significant ($P < 0.01$) throughout the period of obstruction in the animals of group I (Table 11). The potassium concentration in peritoneal fluid decreased significantly at 3rd day following creation of intestinal obstruction in the animals of group I, II ($P < 0.05$) and III ($P < 0.01$) as compared to respective base values (Table 11, Fig. 9). This decline in peritoneal fluid potassium concentration was consistently significant ($P < 0.01$) during the whole period of observation in the animals of group I. The changes in peritoneal fluid chloride concentration as

PLATE 7: PERITONEAL FLUID CHANGES AFTER CREATION OF SIMPLE JEJUNAL OBSTRUCTION IN CALVES



**"0" Day
(Base Value)**



**2nd day
post creation**



**3rd day
post creation**



**4th day
post creation**



**6th day
post creation**



**8th day
post creation**

TABLE 10: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON PERITONEAL FLUID pH, TOTAL PROTEINS AND NUCLEATED CELL COUNT IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
PERITONEAL FLUID pH						
Group I	7.18 ±0.284 (n=4)	7.25 ±0.25 (n=4)	7.00 ±0.123 (n=4)	6.83 ±0.214 (n=4)	6.75 ±0.206 (n=4)	6.75 ±0.206 (n=2)
Group II	7.36 ±0.239 (n=4)	7.30 ±0.286 (n=4)	7.13 ±0.175 (n=4)	7.05 ±0.206 (n=4)	6.98 ±0.188 (n=4)	6.73 ±0.145 (n=3)
Group III	7.10 ±0.308 (n=4)	6.90 ±0.2 (n=4)	6.70 ±0.123 (n=4)	7.03 ±0.347 (n=4)	7.10 ±0.308 (n=4)	7.17 ±0.284 (n=4)
TOTAL PERITONEAL FLUID PROTIENS (mg/dL)						
Group I	2.55 ±0.1258 (n=4)	3.18 ±0.278 (n=4)	4.15** ±0.096 (n=4)	4.53** ±0.125 (n=4)	5.40** ±0.178 (n=4)	6.00 ^{N.I.} ±0.30 (n=2)
Group II	2.70 ±0.216 (n=4)	3.15 ±0.287 (n=4)	3.50** ±0.248 (n=4)	3.80 ^c ±0.187 (n=4)	4.23 ^{bd} ±0.193 (n=4)	4.46 ^{ac} ±0.203 (n=3)
Group III	2.38 ±0.263 (n=4)	2.75 ±0.185 (n=4)	3.75** ±0.272 (n=4)	3.78 ^d ±0.144 (n=4)	3.38 ^{ade} ±0.149 (n=4)	2.93 ^{bd} ±0.170 (n=4)
NUCLEATED CELL COUNT (x 10³/cu mm)						
Group I	3.01 ±0.080 (n=4)	4.41** ±0.281 (n=4)	5.44** ±0.116 (n=4)	5.93** ±0.119 (n=4)	6.63** ±0.116 (n=4)	7.35* ±0.10 (n=2)
Group II	3.19 ±0.103 (n=4)	4.84** ±0.229 (n=4)	5.43** ±0.601 (n=4)	6.10 ^b ±0.552 (n=4)	6.46 ^b ±0.481 (n=4)	6.88 ^a ±0.627 (n=3)
Group III	3.24 ±0.186 (n=4)	4.55** ±0.203 (n=4)	5.43** ±0.573 (n=4)	6.18 ^b ±0.318 (n=4)	6.83 ^b ±0.215 (n=4)	7.13 ^b ±0.171 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

compared to base value were significantly low ($P < 0.01$) at 3rd post obstruction day and continued till death supervened in the animals of group I. A significant ($P < 0.05$) decrease in peritoneal fluid chloride concentration at 3rd post obstruction day was also observed in group III when compared to base value (Table 11, Fig. 11). The

TABLE 11: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON PERITONEAL FLUID SODIUM, POTASSIUM, CHLORIDE AND CALCIUM IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3*	4	6	8
SODIUM (mEq/L)						
Group I	142.5 ±0.96 (n=4)	140.0 ±0.816 (n=4)	137.5* ±0.957 (n=4)	136.0** ±0.82 (n=4)	132.0** ±0.82 (n=4)	131.0 ^{N.I.} ±1.0 (n=2)
Group II	143.0 ±0.577 (n=4)	142.0 ±0.817 (n=4)	142.5 ±0.5 (n=4)	142.0 ^c ±1.83 (n=4)	143.5 ^d ±1.71 (n=4)	141.3 ^d ±0.67 (n=3)
Group III	145.5 ±1.5 (n=4)	143.0 ±1.0 (n=4)	141.0** ±1.0 (n=4)	141.5 ^c ±1.71 (n=4)	141.5 ^d ±1.26 (n=4)	143.0 ^d ±1.29 (n=4)
POTASSIUM (mEq/L)						
Group I	4.80 ±0.294 (n=4)	4.23 ±0.085 (n=4)	3.73* ±0.232 (n=4)	3.45** ±0.175 (n=4)	2.97** ±0.137 (n=4)	2.40* ±0.20 (n=2)
Group II	5.00 ±0.548 (n=4)	4.35 ±0.287 (n=4)	4.05* ±0.388 (n=4)	3.85 ±0.366 (n=4)	3.53 ^b ±0.312 (n=4)	3.26 ^a ±0.409 (n=3)
Group III	4.83 ±0.132 (n=4)	4.25 ±0.155 (n=4)	3.75** ±0.065 (n=4)	4.20 ^{ac} ±0.187 (n=4)	4.63 ^{bde} ±0.193 (n=4)	4.65 ^{bde} ±0.096 (n=4)
CHLORIDE (mEq/L)						
Group I	98.95 ±2.693 (n=4)	91.18 ±2.307 (n=4)	82.70** ±2.53 (n=4)	77.92** ±3.113 (n=4)	71.42** ±1.392 (n=4)	65.65* ±1.850 (n=2)
Group II	98.50 ±3.096 (n=4)	95.03 ±2.29 (n=4)	94.75 ±1.556 (n=4)	96.40 ^d ±0.40 (n=4)	98.18 ^d ±1.312 (n=4)	96.13 ^d ±0.884 (n=3)
Group III	101.95 ±1.012 (n=4)	96.68 ±2.152 (n=4)	94.13* ±0.214 (n=4)	94.38 ^d ±0.853 (n=4)	99.05 ^{bd} ±1.093 (n=4)	101.53 ^{bde} ±1.229 (n=4)
CALCIUM (mg/dL)						
Group I	6.05 ±0.444 (n=4)	5.98 ±0.2872 (n=4)	6.03 ±0.193 (n=4)	5.90 ±0.294 (n=4)	6.05 ±0.311 (n=4)	6.05 ±0.071 (n=2)
Group II	5.30 ±0.374 (n=4)	5.33 ±0.386 (n=4)	5.35 ±0.328 (n=4)	5.03 ±0.492 (n=4)	5.05 ±0.448 (n=4)	5.33 ±0.203 (n=3)
Group III	5.45 ±0.499 (n=4)	5.45 ±0.357 (n=4)	5.53 ±0.397 (n=4)	5.43 ±0.371 (n=4)	5.40 ±0.576 (n=4)	5.28 ±0.449 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

changes in the concentration of calcium and phosphorus in the peritoneal fluid were inconclusive following creation of intestinal obstruction in all the animals of three groups (Table 11, 12).

4.1.4.2 POST TREATMENT

The discolouration in peritoneal fluid showed gradual improvement towards normalcy after institution of the surgical treatment and was near normal at 8th day in the animals of group III. No changes were recorded in peritoneal fluid pH in group II animals (Table 10).

The total protein concentration of peritoneal fluid remained elevated ($P < 0.01$) in the animals of group II as compared to 3rd post obstruction day. However, decrease in total protein was noticed at 6th ($P < 0.05$) and 8th ($P < 0.01$) day when compared to 3rd post obstruction day in the animals of group III. The comparison of group I and II revealed a significantly slower ($P < 0.01$) increase of peritoneal fluid total protein in the animals of group II. On the contrary, a significant decrease was observed in group III when compared to group I and II at 6th ($P < 0.05$) and 8th ($P < 0.01$) day (Table 10, Fig. 9). A significant increase in peritoneal fluid cell count was recorded (4th, 6th day: $P < 0.01$ and 8th day: $P < 0.05$) in the animals of group II when compared to 3rd post obstruction day. Similarly, a statistically significant ($P < 0.01$) increase in the cell count was present throughout the post treatment period in the animals of group III as compared to 3rd post obstruction day (Table 10, Fig. 10).

There was no significant change in the peritoneal fluid sodium concentration in the animals of group II and III respectively. The inter-group comparison revealed that the conservative (group II) and surgical (group III)

TABLE 12: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON PERITONEAL FLUID PHOSPHORUS IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
Group I	4.52 ±0.356 (n=4)	4.33 ±0.259 (n=4)	4.43 ±0.357 (n=4)	4.37 ±0.419 (n=4)	4.60 ±0.324 (n=4)	4.95 ±0.150 (n=2)
Group II	4.40 ±0.455 (n=4)	4.35 ±0.494 (n=4)	4.50 ±0.532 (n=4)	4.45 ±0.413 (n=4)	4.40 ±0.392 (n=4)	4.60 ±0.231 (n=3)
Group III	4.13 ±0.132 (n=4)	4.10 ±0.204 (n=4)	4.20 ±0.283 (n=4)	4.00 ±0.324 (n=4)	4.15 ±0.321 (n=4)	4.03 ±0.229 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

therapy offsets ($P < 0.01$) the sodium deficit which was quite evident in group I (Table 11). There was a continuous fall (6th day: $P < 0.01$ and 8th day: $P < 0.05$) in the peritoneal fluid potassium concentration in the animals of group II. The potassium concentration increased significantly ($P < 0.05$) immediately a day after the operation and significant ($P < 0.01$) increase was recorded in its concentration at 6th and 8th days in the animals of group III when compared to 3rd post obstruction day. The animals of group III showed significant ($P < 0.05$) recovery of potassium at 4th day and it was significantly higher ($P < 0.01$) at 6th and 8th day in comparison to group I. The potassium concentration remained significantly ($P < 0.05$) low at 6th and 8th day in the animals of group II when compared to group III (Table 11). The chloride concentration in peritoneal fluid remained statistically unaffected in the animals of group II when compared to 3rd post obstruction day. The chloride concentration in peritoneal fluid was elevated significantly ($P < 0.01$) at 6th and 8th day in the animals of group III as compared to 3rd post obstruction day.

FIG. 9: VARIATIONS IN PERITONEAL FLUID PROTEIN FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.

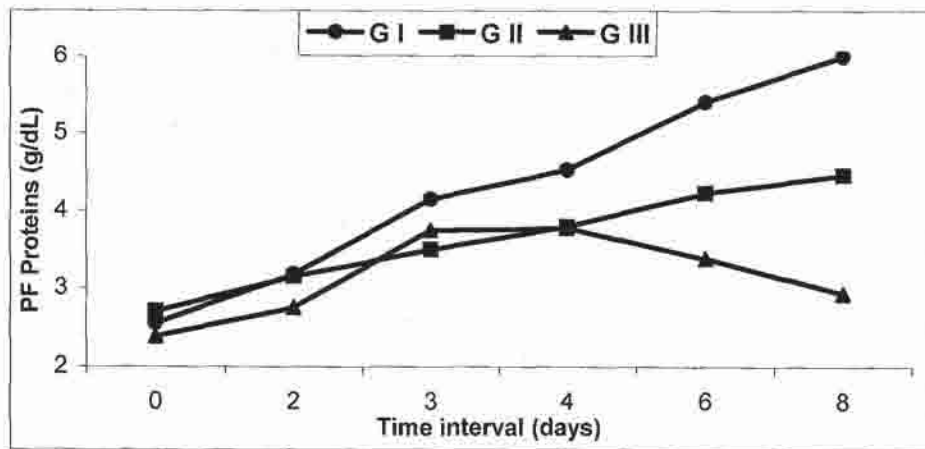


FIG. 10: VARIATIONS IN PERITONEAL FLUID NUCLEATED CELLS FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.

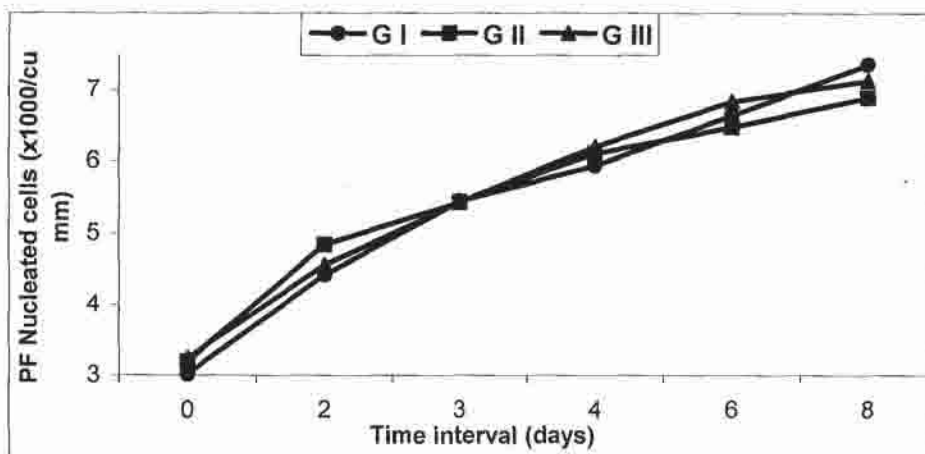
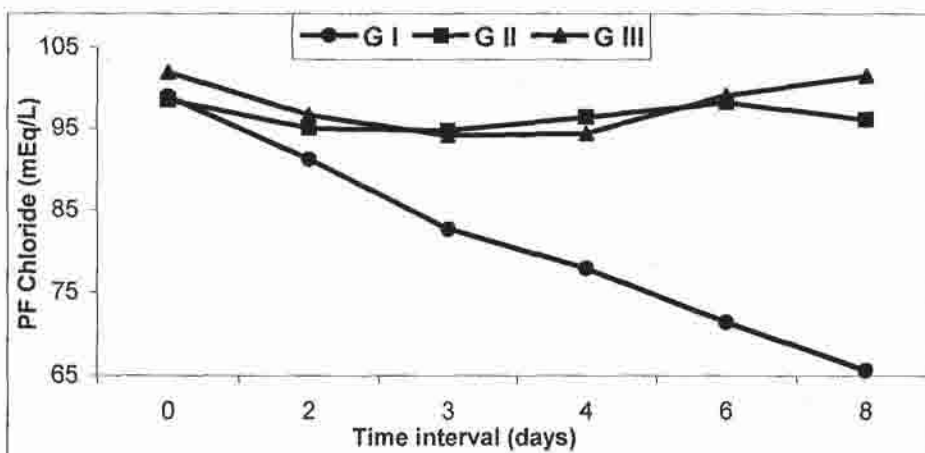


FIG. 11: VARIATIONS IN PERITONEAL FLUID CHLORIDE FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.



There was a significant ($P<0.01$) loss of peritoneal fluid chloride in group I as compared to group II and III. Furthermore, a significant recuperation of chloride in peritoneal fluid occurred on 8th day in the animals of group III when compared to group II (Table 11, Fig. 11). No alterations in peritoneal fluid calcium and phosphorus were seen at different time intervals in the animals of group II and III (Table 11, 12).

4.1.5 ALTERATIONS IN RUMINAL FLUID BIOCHEMISTRY

4.1.5.1 PRE TREATMENT

A decreasing trend in the ruminal fluid sodium and potassium concentration was seen from 2nd day onward following creation of simple intestinal obstruction. In comparison to their respective base values its drop was highly significant at 3rd post obstruction day in the animals of all the three groups. A significant ($P<0.01$) decrease in ruminal fluid sodium and potassium concentration was observed throughout the period of obstruction in the animals of group I as compared to their base values (Table 13, Fig. 12). Contrarily, the ruminal fluid chloride concentration rose significantly ($P<0.01$) in all the groups on 3rd day following simple intestinal obstruction when compared to base value. A significant ($P<0.01$) progressive rise in ruminal fluid chloride concentration persisted throughout period of study in the animals of animals of group I (Table 13, Fig. 13).

Inconsistent changes were encountered in ruminal fluid calcium concentration following creation of intestinal obstruction in all the animals of three groups (Table 13). The rumen fluid phosphorus concentration increased significantly ($P<0.05$) in the animals of group II and III and it was significantly higher ($P<0.01$) in group I at 2nd post obstruction day which later on increased significantly ($P<0.01$) in all the groups at 3rd day. A progressive and significant

TABLE 13: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON RUMINAL FLUID SODIUM, POTASSIUM, CHLORIDE AND CALCIUM IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
SODIUM (mEq/L)						
Group I	112.0 ±5.66 (n=4)	104.5 ±5.058 (n=4)	94.0** ±2.160 (n=4)	91.5** ±2.22 (n=4)	82.0** ±0.82 (n=4)	76.0 ^{N.I.} ±2.00 (n=2)
Group II	98.5 ±4.03 (n=4)	92.0 ±1.155 (n=4)	86.0** ±2.582 (n=4)	85.0 ^c ±1.29 (n=4)	76.0 ^{bd} ±0.83 (n=4)	64.0 ^{ac} ±1.155 (n=3)
Group III	102.5 ±1.708 (n=4)	96.5 ±1.5 (n=4)	85.5** ±4.717 (n=4)	86.5 ±6.70 (n=4)	88.0 ^e ±4.76 (n=4)	94.0 ^{cf} ±4.08 (n=4)
POTASSIUM (mEq/L)						
Group I	27.97 ±2.042 (n=4)	23.90 ±0.545 (n=4)	20.50** ±1.443 (n=4)	19.57** ±1.722 (n=4)	15.75** ±1.109 (n=4)	14.5 ^{N.I.} ±0.500 (n=2)
Group II	29.55 ±1.926 (n=4)	25.38 ±1.346 (n=4)	21.38** ±1.609 (n=4)	21.38 ±1.61 (n=4)	18.10 ^b ±1.56 (n=4)	14.77 ^a ±1.748 (n=3)
Group III	27.75 ±1.548 (n=4)	23.43 ±1.105 (n=4)	17.75** ±1.031 (n=4)	20.75 ^a ±1.493 (n=4)	24.00 ^{bde} ±1.472 (n=4)	26.25 ^{bdf} ±1.493 (n=4)
CHLORIDE (mEq/L)						
Group I	37.17 ±1.756 (n=4)	45.05 ±3.025 (n=4)	55.08** ±2.116 (n=4)	64.27** ±2.823 (n=4)	86.23** ±5.062 (n=4)	99.15* ±0.350 (n=2)
Group II	31.30 ±1.714 (n=4)	40.35 ±2.739 (n=4)	52.10** ±1.663 (n=4)	60.53 ±2.555 (n=4)	72.98 ^b ±5.68 (n=4)	82.90 ^a ±5.351 (n=3)
Group III	27.88 ±2.631 (n=4)	37.40 ±3.219 (n=4)	53.30** ±2.627 (n=4)	34.15 ^{bdf} ±3.973 (n=4)	30.70 ^{bdf} ±2.980 (n=4)	28.95 ^{bdf} ±3.013 (n=4)
CALCIUM (mg/dL)						
Group I	8.80 ±0.572 (n=4)	8.70 ±0.420 (n=4)	8.93 ±0.263 (n=4)	8.38 ±0.175 (n=4)	8.63 ±0.328 (n=4)	9.15 ±0.45 (n=2)
Group II	8.30 ±0.168 (n=4)	8.28 ±0.214 (n=4)	8.35 ±0.524 (n=4)	8.25 ±0.296 (n=4)	8.33 ±0.319 (n=4)	8.03 ±0.219 (n=3)
Group III	8.95 ±0.263 (n=4)	8.95 ±0.226 (n=4)	8.90 ±0.204 (n=4)	9.10 ±0.379 (n=4)	9.03 ±0.218 (n=4)	9.05 ±0.296 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

TABLE 14: EFFECT OF SIMPLE INTESTINAL OBSTRUCTION ON RUMINAL FLUID PHOSPHORUS IN CALVES (MEAN±S.E).

GROUPS	DAYS AFTER CREATION OF INTESTINAL OBSTRUCTION					
	0	2	3 [#]	4	6	8
PHOSPHORUS (mg/dL)						
Group I	12.80 ±0.628 (n=4)	27.2** ±1.655 (n=4)	36.70** ±2.286 (n=4)	44.15** ±1.825 (n=4)	58.10** ±2.964 (n=4)	73.00* ±0.60 (n=2)
Group II	9.80 ±1.582 (n=4)	26.85* ±4.918 (n=4)	33.75** ±5.555 (n=4)	39.80 ^a ±5.283 (n=4)	53.48 ^b ±4.794 (n=4)	68.73 ^a ±3.083 (n=3)
Group III	9.83 ±1.59 (n=4)	23.96* ±5.042 (n=4)	32.58** ±4.693 (n=4)	22.30 ^{bde} ±4.049 (n=4)	16.28 ^{bdf} ±2.744 (n=4)	12.30 ^{bdf} ±0.892 (n=4)

- Day of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 'hour' value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 3 day value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding day.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding day.

N.I. - Value not included for statistical analysis within group.

($P < 0.01$) increase in ruminal fluid phosphorus continued throughout the entire period of study in all the animals of group I when compared to base value (Table 14, Fig. 14).

4.1.5.2 POST TREATMENT

A progressively significant ($P < 0.01$) decline in ruminal fluid sodium concentration persisted at 6th and 8th day in group II in comparison to 3rd post obstruction day. However, the animals of group III did not show such change but maintained constancy in ruminal fluid sodium concentration during the entire post operative period. The inter-group comparison revealed a significant recovery of sodium on 8th day in the animals of group III in comparison to group I (Table 13). Whereas, at 6th ($P < 0.05$) and 8th ($P < 0.01$) day sodium concentration increased significantly in the animals of group III as compared to group II. The deficit in ruminal fluid potassium concentration as compared to 3rd post obstruction day was significantly ($P < 0.01$, 6th day and $P < 0.05$, 8th day) apparent in the animals of group

FIG. 12: VARIATIONS IN RUMINAL FLUID POTASSIUM CONCENTRATION FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.

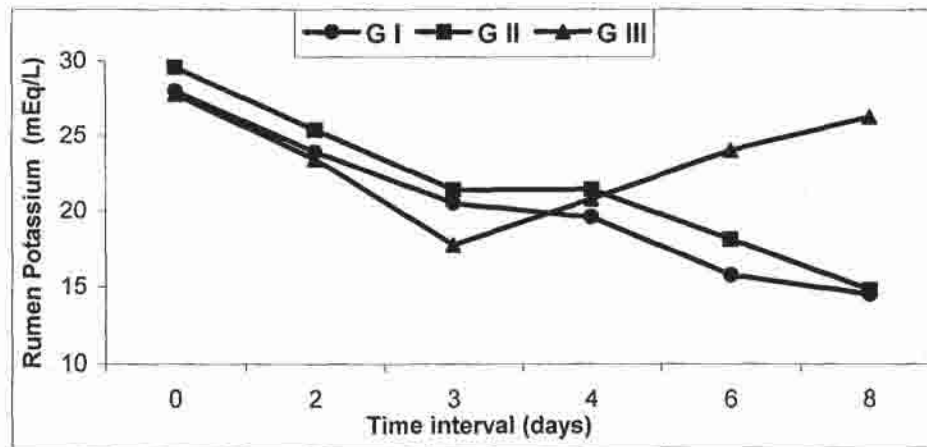


FIG. 13: VARIATIONS IN RUMINAL FLUID CHLORIDE CONCENTRATION FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.

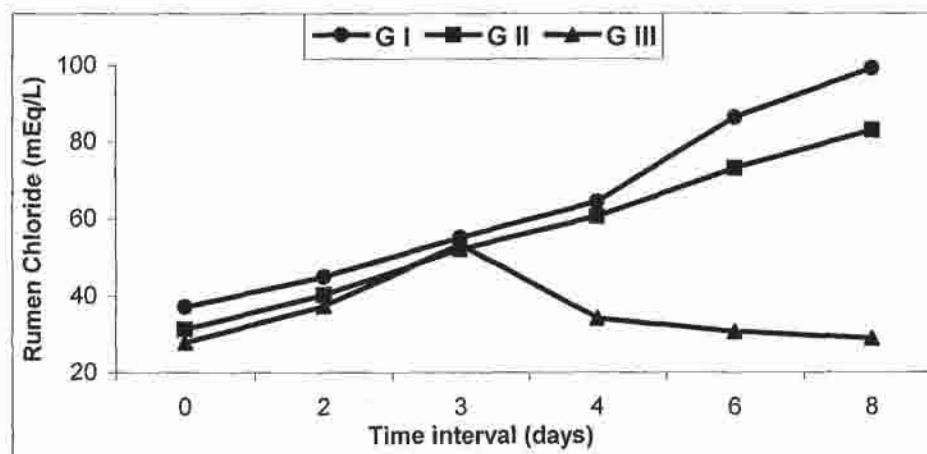
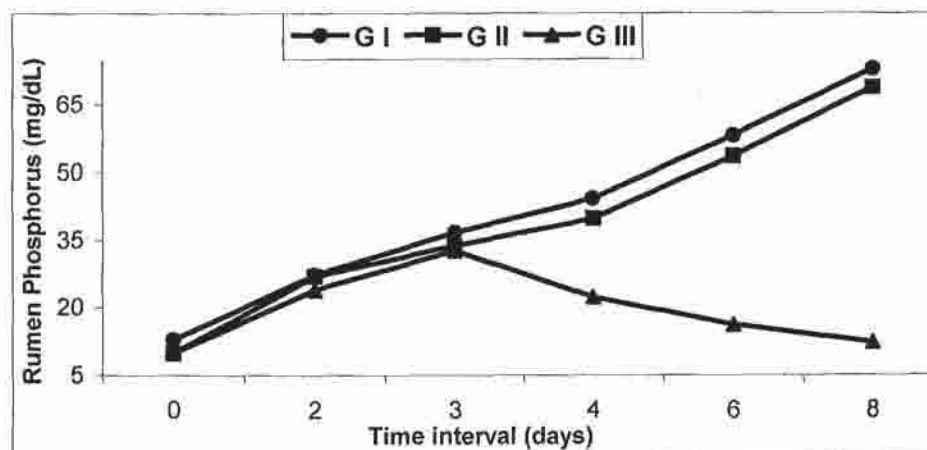


FIG. 14: VARIATIONS IN RUMINAL FLUID PHOSPHORUS CONCENTRATION FOLLOWING SIMPLE INTESTINAL OBSTRUCTION IN CALVES.



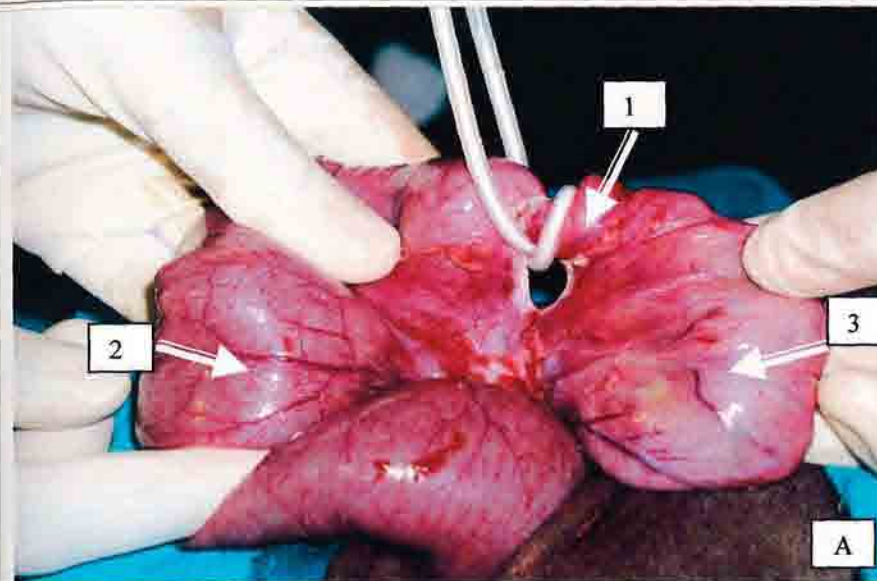
II. However, the potassium concentration in ruminal fluid of group III significantly ($P<0.01$) recuperated to near normal at 8th post treatment day. The comparison of group III with group I and II revealed a significant ($P<0.01$) recovery in ruminal potassium concentration after surgical treatment (Table 13, Fig. 12). The ruminal fluid chloride concentration remained significantly high ($P<0.01$: 6th day and $P<0.05$: 8th day) in post treatment period when compared to 3rd post obstruction day in the animals of group II. Whereas, a highly significant decrease as compared to 3rd post obstruction day's value was noticed during post surgery period in the animals of group III. The inter-group comparison revealed significant ($P<0.01$) normalization of ruminal fluid chloride concentration in the animals of group III as compared to group I and II (Table 13, Fig. 13).

No changes in ruminal fluid calcium were noticed during post treatment period in the animals of group II and III (Table 13). A significant ($P<0.05$: 4th day and $P<0.01$: 6th day) rise in ruminal fluid phosphorus concentration was recorded in the animals of group II but a significant ($P<0.01$) fall in ruminal fluid phosphorus concentration was observed in group III when compared to their respective 3rd post obstruction day values. In comparison to group I and II, the normalization in ruminal fluid phosphorus concentration was significant ($P<0.01$) in the animals of group III (Table 14, Fig. 14).

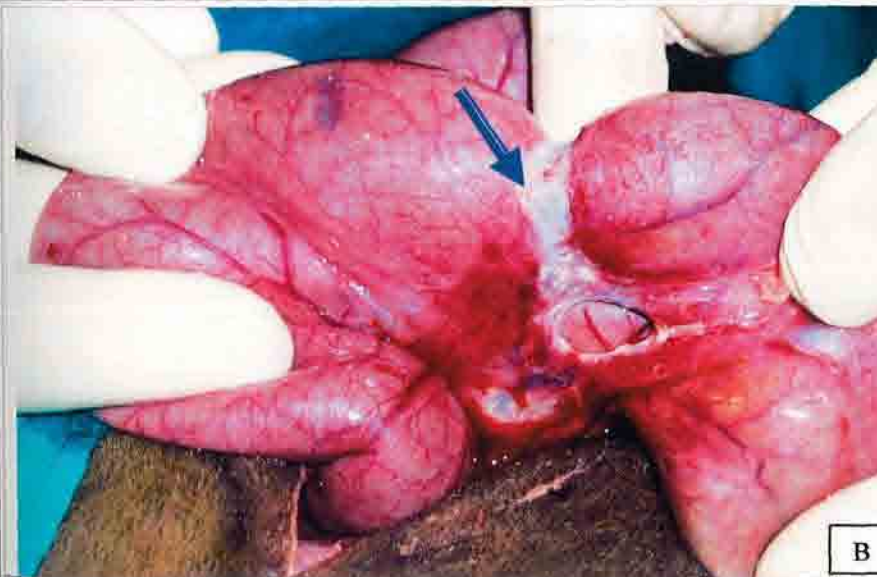
4.1.6 OPERATIVE FINDINGS

The site of obstructed loop was discolored and appeared grayish in colour with irreversible stricture. The segment of intestine proximal to the site of obstruction was greatly distended and showed bluish discolouration with no peristalsis upto considerable length. The distal segment was collapsed and the mesentery was hemorrhagic with adhesive reaction (Plate 8).

PLATE 8: INTESTINAL CHANGES JUST BEFORE CORRECTIVE SURGERY OF SIMPLE JEJUNAL OBSTRUCTION IN CALVES



1. Site of obstructed loop
2. Proximal distended segment
3. Distal collapsed segment



The view of intestine after removal of silicon tubing

→ The bowel wall showed discoloration and ischemic changes at obstructed site

4.1.7 TOTAL SURVIVAL TIME

The animals of group I served as diseased control succumbed at different time interval following creation of simple intestinal obstruction. Three animals of group I survived upto 8 days. The average survival time in the animals of group I was 7.99 ± 0.793 days. The average survival time in the animals of group II with conservative treatment was 9.29 ± 0.49 days. All the animals of group III survived following surgical treatment.

4.2 STRANGULATED INTESTINAL OBSTRUCTION

4.2.1 CLINICAL OBSERVATIONS

4.2.1.1 PRE TREATMENT

All the animals exhibited clinical signs of acute pain within half to two hours of creation of strangulated intestinal obstruction which were manifested by kicking at the abdomen, restlessness, lying down and getting up frequently, vocalization, bruxism, straining to urinate and defecate, lying their head and neck flat against the ground or down across their back. These signs persisted upto 4-5 hours and the intensity of pain was severe during initial 2-3 hours of obstruction.

The majority of animals in all the three groups defecated immediately after creation of strangulated obstruction but as the time passed, defecation became scanty. After 24 hours, the water content of faeces decreased and assumed the shape of balls in all the animals of three groups. Initially, the animals of group I made unsuccessful attempts to defecate (Plate 9A) and there was complete cessation of defecation after 24 hours and only the mucous with foul odour was voided. The frequency of passing the mucous was 2-3 times a day till 48 hours in group I. Later on the nature of faeces became crupous with diphtheritic shreds and remained so till the death of animals (Plate 10). Urination was normal in all the animals of group I, II and III but was reduced at terminal stages in group I.

Calves of group I and II resumed almost normal feed and water intake at 12 post obstruction hours but appetite was reduced after 24 – 36 hours in the animals of group I. Water intake was normal in the animals of group I, II and III upto 24 hours and thereafter in the animals of group I the water intake reduced progressively.

PLATE 9: CLINICAL SIGNS DURING STRANGULATED JEJUNAL OBSTRUCTION IN CALVES



Straining while defecation



Dryness of Muzzle during early course of disease

Signs of muscular weakness were constantly observed in all the animals of three groups which were manifested initially by reluctance to move at 12 hours, instability of hind limbs whenever animals tried to shift the position at 24 hours. Eventually the animals exhibited difficulty while assuming the sternal recumbency and even greater difficulty when tried to rise at 72 hours. As the time period of obstruction was progressed the animals of group I and II became recumbent and were unable to rise even with assistance at 96 hours.

The rumen was usually hypotonic in the animals of group I and II on 24 hours and virtually no contraction could be detected on and after 48 hours. In all the animals rumination ceased completely at 24 hours following the intestinal obstruction. On auscultation intestinal borborygmi revealed tinkling and clicking sounds from the abdomen which were evident upto 48 hours in the animals of group I and later on quiescence in abdomen was appreciated. Abdominal distention was not very much appreciable during initial period of obstruction in all the groups however, in group I progressive bilateral distention was observed after 48 hours and it became prominent at 72 and 96 post obstruction hours.

Rectal temperature showed a significant ($P < 0.05$) decrease at 24 hours and this decline was significantly ($P < 0.01$) higher at 48 hours when compared to base values in the animals of group I (Table 15). The respiration rate (Table 15) in the animals of group I increased significantly ($P < 0.05$) at 24 hours and then this increase was significantly ($P < 0.01$) higher at 48 hours in comparison to base values. There was a significant ($P < 0.01$) increase in heart (Fig. 15) and pulse rate at 24 post obstruction hours in animals of group I, II and III when compared to base values. The heart and pulse rate remained significantly ($P < 0.01$: 48 hours

PLATE 10: PATTERN AND CHARACTERISTIC OF FAECES AFTER CREATION OF STRANGULATED JEJUNAL OBSTRUCTION IN CALVES



8 hours post creation



12 hours post creation



24 hours post creation



48 hours post creation



96 hours post creation

and $P < 0.05$: 72 hours) elevated upto 72 post obstruction hours in the animals of group I (Table 15).

The generalized listlessness and depression following creation of obstruction was seen in all the animals of group I and II accompanied by cold extremities and sunken eye balls. These symptoms were more pronounced in group I and their severity increased with the passage of time. The gradual worsening in attentiveness was observed in animals of group I as the time of death approached. All the animals in three groups manifested the signs of dehydration i.e. the moderate recession of eye ball by 48 hours which was marked at the end of the period of obstruction especially in the animals of group I. The elasticity of skin decreased from 24 hours (3-4 seconds) and became inelastic after 48 (10-12 seconds) hours onward. Usually after 24 hours the muzzle was mildly wet and became dry from 48 hours onward (Plate 9B). A significant increase in capillary refill time (CRT) was observed at 24 ($P < 0.05$) and 48 ($P < 0.01$) post obstruction hours in the animals of group I. Its value was highest (3 seconds) at the end of the period of obstruction. In the animals of group III, the CRT was significant ($P < 0.05$) at 24 post obstruction hours (Table 16).

Ruminal fluid pH decreased significantly ($P < 0.05$) in the animals of group I and II and this decline in the ruminal fluid pH remained significant ($P < 0.05$) in the entire post obstruction period in the animals of group I (Table 16). The large ruminal protozoa suffered moderate (++) loss at 24 hours in the animals of all the three groups. The protozoal motility in animals of group I was sluggish (+) comprising of only the smaller protozoa at 48 hours with complete (-) loss at 96 hours.

TABLE 15: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON RECTAL TEMPERATURE, RESPIRATION RATE, HEART RATE AND PULSE RATE IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
RECTAL TEMPERATURE (°F)					
Group I	102.15 ±0.25 (n=4)	101.60* ±0.316 (n=4)	100.60** ±0.337 (n=4)	100.20 ±0.8 (n=2)	98.60 ^{N.I.} ±0.0 (n=1)
Group II	102.10 ±0.129 (n=4)	101.50 ±0.369 (n=4)	100.50 ±0.387 (n=4)	99.40 ^a ±0.116 (n=3)	99.00 ^{N.I.} ±0.2 (n=2)
Group III	101.75 ±0.479 (n=4)	102.25 ±0.096 (n=4)	102.15 ^{df} ±0.126 (n=4)	102.05 ^{df} ±0.263 (n=4)	102.00 ^f ±0.245 (n=4)
RESPIRATION RATE (/min)					
Group I	12.25 ±0.75 (n=4)	14.00* ±0.71 (n=4)	16.25** ±0.629 (n=4)	17.50 ±1.5 (n=2)	18.00 ^{N.I.} ±0.0 (n=1)
Group II	13.00 ±0.58 (n=4)	14.50 ±0.96 (n=4)	15.50 ±0.96 (n=4)	16.67 ±1.764 (n=3)	18.00 ^{N.I.} ±2.00 (n=2)
Group III	14.50 ±0.96 (n=4)	15.00 ±1.29 (n=4)	14.50 ±0.5 (n=4)	15.00 ±1.0 (n=4)	15.00 ±0.58 (n=4)
HEART RATE (/min)					
Group I	69.5 ±0.957 (n=4)	89.5** ±2.5 (n=4)	85.5** ±0.957 (n=4)	90.0* ±3.0 (n=2)	80.0 ^{N.I.} ±0.0 (n=1)
Group II	65.5 ±0.957 (n=4)	87.5** ±1.708 (n=4)	86.0 ±1.414 (n=4)	80.6 ±6.566 (n=3)	86.0 ±6.0 (n=2)
Group III	69.5 ±2.5 (n=4)	84.5** ±2.363 (n=4)	71.5 ^{bdf} ±3.304 (n=4)	71.0 ^{bd} ±2.38 (n=4)	71.5 ^{be} ±2.217 (n=4)
PULSE RATE (/min)					
Group I	68.5 ±0.5 (n=4)	87.5** ±4.193 (n=4)	83.5** ±2.63 (n=4)	86.5* ±6.50 (n=2)	78.0 ^{N.I.} ±0.0 (n=1)
Group II	62.5 ±0.957 (n=4)	85.5** ±0.957 (n=4)	85.5 ±1.5 (n=4)	80.0 ±6.11 (n=3)	84.0 ±4.0 (n=2)
Group III	69.0 ±2.517 (n=4)	83.0** ±2.082 (n=4)	70.5 ^{bdf} ±2.986 (n=4)	70.5 ^{bc} ±2.5 (n=4)	70.5 ^{be} ±1.258 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

TABLE 16: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON CAPILLARY REFILL TIME AND RUMINAL FLUID pH IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
CAPILLARY REFILL TIME (sec)					
Group I	0.63 ±0.125 (n=4)	1.25* ±0.144 (n=4)	1.63** ±0.125 (n=4)	2.25 ±0.25 (n=2)	3.00 ^{N.I.} ±0.0 (n=1)
Group II	0.63 ±0.125 (n=4)	0.75 ±0.144 (n=4)	0.75 ^d ±0.144 (n=4)	0.67 ^d ±0.167 (n=3)	0.50 ±0.0 (n=2)
Group III	0.50 ±0.0 (n=4)	0.88* ±0.125 (n=4)	0.75 ^d ±0.144 (n=4)	0.50 ±0.0 (n=4)	0.50 ±0.0 (n=4)
RUMINAL FLUID pH					
Group I	7.20 ±0.123 (n=4)	6.93 ±0.075 (n=4)	6.78* ±0.075 (n=4)	6.55** ±0.15 (n=2)	6.40 ^{N.I.} ±0.0 (n=1)
Group II	7.20 ±0.123 (n=4)	7.00* ±0.123 (n=4)	6.93 ±0.144 (n=4)	6.80 ±0.10 (n=3)	6.55 ±0.15 (n=2)
Group III	6.70 ±0.123 (n=4)	6.48 ±0.075 (n=4)	6.70 ±0.123 (n=4)	6.78 ±0.75 (n=4)	6.85 ±0.15 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

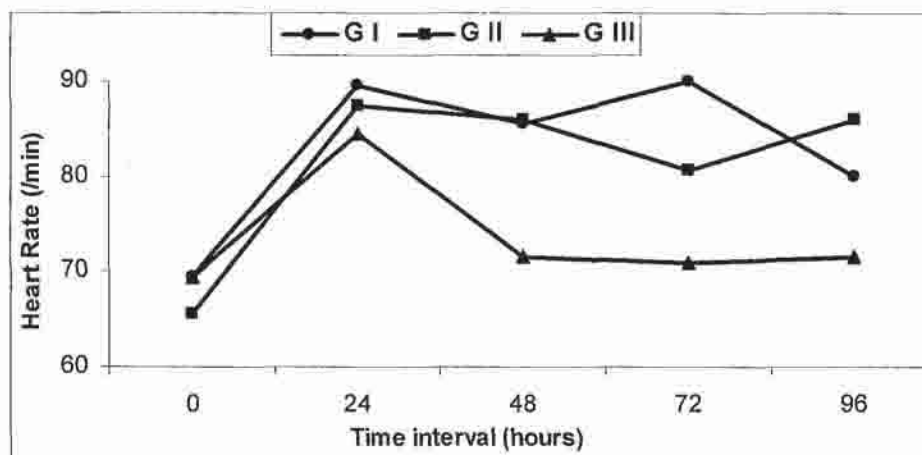
4.2.1.2 POST TREATMENT

The animals of group II made attempts to defecate at almost all the intervals. Animals passed dehydrated dung balls with small amount of mucous at 24 hours and the faeces were malodourous, majorly mixed with the mucous at 48 hours, later at 72 and 96 hours only thick mucous was voided which often clogged the anus. The animals of group III passed dung balls after 5-6 hours of treatment. The consistency of the faeces was semisolid with fair amount of moisture and was almost well formed at 72 and 96 hours of treatment, but slight mucous was persistently present. Normal consistency of faeces without foul odour was

regained at 96 hours post treatment in the animals of group III. The frequency and quantity of urination was normal in the animals of group II and III during the entire post treatment period.

The animals of group II had normal appetite upto 24 hours and thereafter it reduced, but intermittent intake of fodder was observed even upto the end of post treatment period. All the animals of group III resumed normal feed intake at 72 post obstruction hours. Water intake was normal in the animals of group II upto 24 hours but later it reduced to almost nil as the duration of obstruction increased, whereas in the animals of group III, water intake was almost normal after 24 hours of treatment.

FIG. 15: VARIATIONS IN HEART RATE FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.



Signs of muscular weakness were observed during the post treatment period in the animals of group II which were evident by frequent assumption of recumbency after 48 hours of creation of strangulated intestinal obstruction. The animals assumed prolonged sternal recumbency with inability to stand at 72 hours, eventually lead to digging of head into the flank. Whereas, the animals of group III were able to get up and walk without assistance.

Rumen motility decreased at 24 hours ($\frac{1}{2}$ per 3 min) onward in group II and rumen became atonic at 72 hours onward till the end of the study whereas in group III the tonicity of rumen increased slightly after 48 hours (1 per 3 min) but the rumen remained hypotonic till 96 hours (1 per 3 min). On auscultation intestinal borborygmi comprising of rumbling noise of fluids were present in the animals of group II upto 72 hours, whereas in the animals of group III unique sounds of peristaltic rushes were heard at 24 hours onward. Marked bilateral distention of the abdomen was observed after 48 hours in the animals of group II, whereas the abdominal distention observed prior to treatment in the animals of group III subsided within 24 hours after the treatment.

There was significant ($P < 0.05$; 72 hours) decrease in the rectal temperature in the animals of group II when compared to 24 hours, whereas the rectal temperature remained almost normal throughout the post treatment period in the animals of group III. On inter-group comparison the decline in rectal temperature in the animals of group I was significant ($P < 0.01$) at 48 and 72 hours when compared to group III. Likewise, a significant ($P < 0.01$) decrease in rectal temperature was evident during entire post treatment period in the animals of group II in comparison to group III (Table 15). An increasing trend in the respiration rate was observed in group II but respiratory movements became progressively shallow with occasional groaning as the period of illness increased, whereas in the animals of group III after the surgical treatment the respiratory rate remained unchanged (Table 16). An increasing trend in heart rate was seen in the animals of group II upto 96 hours whereas a significant ($P < 0.01$) decrease with a tendency of restitution towards normalcy was seen in the entire post treatment period. The inter-group comparison between group I and group III indicated significant ($P < 0.01$) decrease,

which almost came to normal at 96 hours in group III (Table 15, Fig. 15). Similar trend in pulse rate was observed in the animal of group II and III. A stronger pulse was appreciated in group III after the treatment was ensued (Table 15).

The animals of group II were active upto 48 hours and thereafter they became dull and depressed. The animals of group III remained active and attentive throughout the post treatment period. Hydration status in all the animals of group II and III was normal as evident by normal skin tenting time and condition of eye ball but dryness of muzzle was seen at 48 hours of obstruction which remained as such till the terminal part of study in the animals of group II. Only one animal of group II showed some signs of dehydration with increased skin tenting time and retraction of globe in the orbital cavity at 72 post obstruction hours. Conjunctival mucous membrane remained pink throughout the period of study. Capillary refill time (CRT) remained unchanged both in the animals of group II and III in comparison to base values. The inter-group comparison between group I, II and III indicated that there was significant ($P < 0.01$) decrease in the CRT during post treatment period in the animals of group II and III when compared with group I at 48 and 72 hours (Table 16).

The pH of ruminal fluid in the animals of group II and III did not show any significant changes (Table 16). Ruminal microflora showed moderate (++) motility at 24 hours interval and there was sluggish (+) motility upto 72 hours with complete loss at 96 hours in group II, whereas in group III there was mild loss of ruminal microflora at 48 hours but its livability increased at 72 hours onward.

4.2.2 HAEMATOLOGICAL OBSERVATIONS

4.2.2.1 PRE-TREATMENT

A significant ($P < 0.01$) increase in the hemoglobin concentration was recorded at 24 post obstruction hours when compared to base values in the animals of all the three groups. Whereas, in the animals of group I a statistically significant ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) increase was observed in haemoglobin concentration when compared to its base value (Table 17). There was significant ($P < 0.01$) increase in packed cell volume at 24 post obstruction hours in the animals of all the three groups when compared to respective base values. The significant elevation (48 hours: $P < 0.01$ and 72 hours: $P < 0.05$) in the PCV continued through out the period of study in the animals of group I (Table 17, Fig. 16). The total erythrocytic count showed significant ($P < 0.01$) increase at 24 post obstruction hours in comparison to base values, in the animals of group I and II. A progressive increase in total erythrocytic count was noticed in the animals of group I throughout the period of observation (Table 17).

A significant ($P < 0.01$) increase in total leukocyte count (TLC) was recorded at 24 hours in all the animals of group I, II and III in comparison to base values. A statistically significant ($P < 0.05$) increase in TLC continued through out the period of study in the animals of group I (Table 18, Fig. 17). A significant ($P < 0.01$) increase in neutrophils was observed in the animals of group I and II at 24 post obstruction hours when compared to base values, whereas this increase in neutrophils continued through out the period of study in group I (Table 18). There was significant ($P < 0.01$) decrease in lymphocytes at 24 hours period both in the animals of group I and II when compared to base values. The pace of decrease in

lymphocytes of group I was significant through out the period of observation (Table 18).

4.2.2.2 POST TREATMENT

There was statistically significant ($P < 0.01$) increase in haemoglobin in the animals of group II at 72 hours when compared to 24 hours value, where as in group III, significant ($P < 0.01$: 72 hours onward) reduction was observed following surgery which at 96 hours approached near normal base value. The inter-group comparison revealed a significant ($P < 0.05$) decrease in haemoglobin at 72 hours in the animals of group III when compared to the corresponding value of group I (Table 17). The packed cell volume increased significantly ($P < 0.05$) at 72 hours onward in comparison to 24 hours value in the animals of group II, whereas in group III animals at 24 hours onward the decrease in packed cell volume was significant ($P < 0.01$) and it persisted upto 96 hours. The inter-group comparison revealed significant ($P < 0.01$) decrease in PCV in the animals of group III from 48 to 72 hours as compared to group I, similarly the comparison between group II and III revealed significant ($P < 0.01$) decrease in packed cell volume in the animals of group III after the treatment (Table 17, Fig. 16). An increasing trend in total erythrocytic count (TEC) was observed in the animals of group II in comparison to 24 hours value, whereas in group III a decrease in TEC was noticed following surgery in comparison to 24 post obstruction hours. The inter-group comparison revealed a significant ($P < 0.05$) decrease in TEC at 72 hours in the animals of group III when compared to group I.

There was an increase in TLC in the entire post treatment period in the animals of group II, whereas, in the animals of group III a significant decrease

TABLE 17: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON HAEMOGLOBIN, PACKED CELL VOLUME AND TOTAL ERYTHROCYTE COUNT IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
HAEMOGLOBIN (g%)					
Group I	9.75 ±0.310 (n=4)	11.60** ±0.216 (n=4)	11.95** ±0.25 (n=4)	12.00* ±0.2 (n=2)	12.60 ^{N.I.} ±0.0 (n=1)
Group II	8.85 ±0.331 (n=4)	11.50** ±0.238 (n=4)	11.65 ±0.331 (n=4)	11.67 ^a ±0.291 (n=3)	11.80 ^{N.I.} ±0.20 (n=2)
Group III	10.20 ±0.408 (n=4)	12.10** ±0.238 (n=4)	11.70 ±0.173 (n=4)	10.90 ^{bc} ±0.192 (n=4)	10.45 ^b ±0.359 (n=4)
PACKED CELL VOLUME (%)					
Group I	30.50 ±0.957 (n=4)	39.25** ±1.797 (n=4)	44.50** ±1.708 (n=4)	49.00* ±1.0 (n=2)	50.00 ^{N.I.} ±0.0 (n=1)
Group II	30.25 ±1.931 (n=4)	39.75** ±1.436 (n=4)	41.00 ±0.577 (n=4)	43.67 ^a ±1.856 (n=3)	47.50 ^a ±0.5 (n=2)
Group III	32.00 ±1.472 (n=4)	42.75** ±1.25 (n=4)	36.50 ^{bd} ±0.957 (n=4)	33.00 ^{bd} ±1.291 (n=4)	32.50 ^{bf} ±0.50 (n=4)
TOTAL ERYTHROCYTE COUNT (millions/cu mm)					
Group I	5.86 ±0.294 (n=4)	7.57** ±0.777 (n=4)	7.66** ±0.751 (n=4)	7.72 ±0.40 (n=2)	8.04 ^{N.I.} ±0.0 (n=1)
Group II	5.06 ±0.215 (n=4)	6.08** ±0.332 (n=4)	6.22 ±0.278 (n=4)	6.37 ±0.393 (n=3)	6.17 ±0.29 (n=2)
Group III	6.25 ±0.281 (n=4)	6.81 ±0.181 (n=4)	6.65 ±0.119 (n=4)	6.53 ^c ±0.084 (n=4)	6.35 ±0.098 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

(P<0.05: 48, 72 hours and P<0.01: 96 hours) was recorded when compared to 24 hours value. The inter-group comparison revealed a significant (P<0.05) decrease in TLC at 72 hours in the animals of group III when compared to group I and significant (P<0.05) decrease in TLC when compared to group II at 96 hours

TABLE 18: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON TOTAL LEUKOCYTE COUNT, NEUTROPHILS AND LYMPHOCYTES IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
TOTAL LEUKOCYTE COUNT (x10³/cu mm)					
Group I	6.00 ±0.333 (n=4)	13.11** ±0.279 (n=4)	13.31** ±0.386 (n=4)	13.73* ±0.275 (n=2)	14.10 ^{N.I.} ±0.0 (n=1)
Group II	6.19 ±0.519 (n=4)	12.81** ±0.621 (n=4)	12.89 ±0.535 (n=4)	13.07 ±0.577 (n=3)	13.28 ±0.491 (n=2)
Group III	6.95 ±0.801 (n=4)	12.85** ±0.644 (n=4)	12.36 ^a ±0.503 (n=4)	11.67 ^{ac} ±0.368 (n=4)	10.83 ^{ba} ±0.367 (n=4)
NEUTROPHILS (% of TLC)					
Group I	32.8 ±1.25 (n=4)	41.5** ±1.5 (n=4)	46.8** ±1.89 (n=4)	53.5* ±2.5 (n=2)	62.0 ^{N.I.} ±0.0 (n=1)
Group II	31.3 ±1.25 (n=4)	39.8** ±1.32 (n=4)	44.3 ^b ±1.8 (n=4)	45.7 ^a ±1.764 (n=3)	46.0 ^{N.I.} ±0.5 (n=2)
Group III	32.5 ±1.44 (n=4)	40.8 ±1.65 (n=4)	41.5 ±1.94 (n=4)	39.3 ^{ac} ±1.89 (n=4)	37.3 ^{be} ±1.89 (n=4)
LYMPHOCYTES (% of TLC)					
Group I	65.5 ±1.26 (n=4)	57.5** ±1.5 (n=4)	53.0** ±1.87 (n=4)	45.0* ±2.0 (n=2)	38.0 ^{N.I.} ±0.0 (n=1)
Group II	68.3 ±1.11 (n=4)	59.8** ±1.49 (n=4)	54.5 ^b ±1.19 (n=4)	54.0 ^{bc} ±1.732 (n=3)	53.0 ^{N.I.} ±1.0 (n=2)
Group III	66.3 ±1.70 (n=4)	59.8 ±2.78 (n=4)	57.5 ±1.44 (n=4)	58.5 ^d ±1.32 (n=4)	62.3 ^e ±2.02 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

(Table 18, Fig. 17). A significant ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) neutrophilia was observed in the animals of group II when compared with 24 hours value. The decrease in the neutrophils was statistically significant ($P < 0.01$: 72 hours and $P < 0.05$: 96 hours) in the animals of group III when compared to its 24

FIG. 16: VARIATIONS IN PACKED CELL VOLUME FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

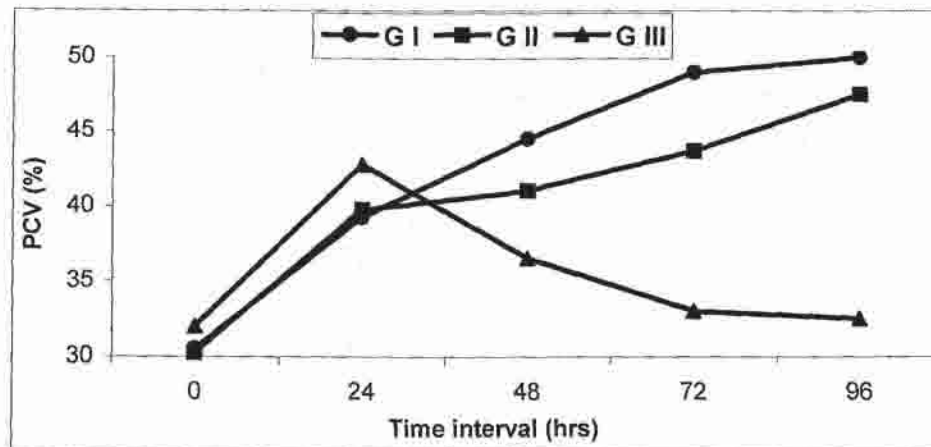
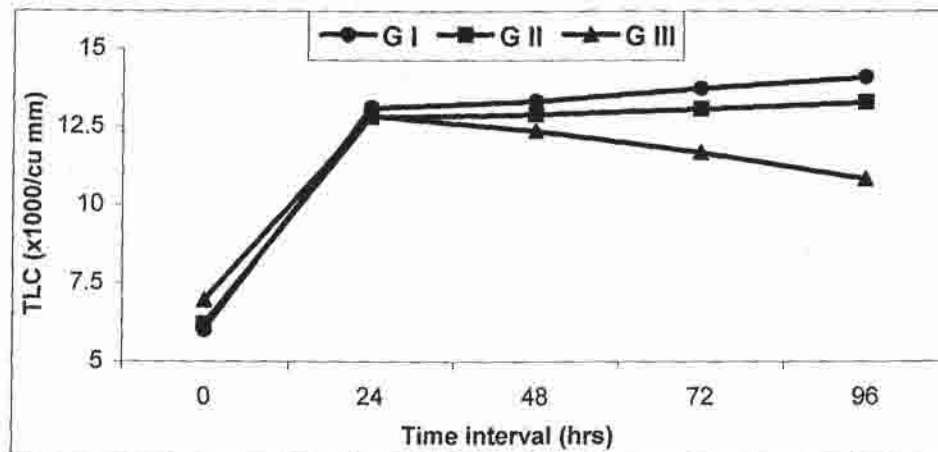


FIG. 17: VARIATIONS IN TOTAL LEUKOCYTIC COUNT FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.



hours value (Table 18). A statistically significant ($P < 0.01$) decrease in the lymphocytes was seen in the animals of group II when compared to its 24 hours value. The inter-group comparison between group I, II and III revealed significant decrease in lymphocytes in animals of group I as compared to group II ($P < 0.05$) and III ($P < 0.01$) at 72 hours. Whereas, a significant ($P < 0.05$) decrease in lymphocytes was seen in the animals of group II when compared with group III at 96 hours (Table 18).

4.2.3 BIOCHEMICAL CHANGES IN PLASMA

4.2.3.1 PRE TREATMENT

There was significant ($P < 0.01$) increase in blood glucose concentration at 24 post obstruction hours when compared to base values in the animals of group I and III. A steady elevation ($P < 0.01$) in the blood glucose concentration was recorded at 48 post obstruction hours in the animals of group I when compared to 24 hours (Table 19).

The increase in total plasma protein (TPR) concentration was significant ($P < 0.01$) at 24 post obstruction hours in the animals of group I, II and III when compared to base values and this significant ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) increase in total plasma protein continued throughout the period of study in the animals of group I (Table 19, Fig. 18).

Increase in blood urea nitrogen was significant ($P < 0.01$) at 24 post obstruction hours in comparison to base values of the animals of group I, II and III. A progressive elevation in the values of BUN was noticed at 48 hours ($P < 0.01$) onward in all the animals of group I as the time period of obstruction increased (Table 19). A significant ($P < 0.01$) increase in the values of plasma creatinine was found in the animals of group II and III at 24 post obstruction hours, whereas significant ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) increase in plasma creatinine was recorded in the animals of group I when compared to base values (Table 19).

Plasma total bilirubin concentration increased significantly ($P < 0.05$) in the animals of group II and III after 24 hours following creation of strangulated obstruction when compared to base values, whereas a significant ($P < 0.01$) increase was observed in total bilirubin at 48 hours interval in the animals of group I (Table 20).

There was significant ($P < 0.01$) decrease in plasma sodium concentration in all the animals of three groups at 24 hours interval when compared to base values. A significant ($P < 0.01$) decrease in plasma sodium concentration was recorded at 48 post obstruction hours in the animals of group I (Table 21). A significant ($P < 0.01$) hypokalemia was observed in the animals of all the three groups at 24 post obstruction hours in comparison to their base values. A consistently decreasing trend ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) in potassium concentration was noticed in all the animals of group I as the duration of obstruction increased (Table 21, Fig. 19).

A significant ($P < 0.01$) hypochloraemia was observed in the animals of all the three groups at 24 hours in comparison to base value. A significant perpetual ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) decline in plasma chloride concentration was recorded in the animals of group I till the terminal part of study when compared to base value (Table 21, Fig. 20). Incoherent changes in relation to base values were recorded in plasma calcium and phosphorus concentration in the animals of all the groups (Table 21, 22).

The elevation in plasma alkaline phosphatase (ALKP) concentration was significant ($P < 0.01$) in the animals of all three groups at 24 hours of creation of strangulated obstruction and this significant ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) rise in the values of ALKP was consistent throughout the period of obstruction in comparison to base value in the animals of group I (Table 23, Fig. 21). A significant ($P < 0.01$) increase in comparison to base value was observed in plasma Aspartate amino transferase (AST) concentration in the animals of all three groups at 24 hours period and this rise was continual ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) in group I throughout the period of study (Table 23).

TABLE 19: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON GLUCOSE, TOTAL PLASMA PROTEIN, BLOOD UREA NITROGEN AND CREATININE IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
GLUCOSE (mg/dL)					
Group I	57.5 ±2.22 (n=4)	69.0** ±1.29 (n=4)	73.5** ±2.22 (n=4)	81.0 ±7.0 (n=2)	82.0 ^{N.I.} ±0.0 (n=1)
Group II	59.3 ±2.29 (n=4)	75.5 ±7.47 (n=4)	94.8 ±13.36 (n=4)	99.7 ±9.35 (n=3)	113.0 ±4.0 (n=2)
Group III	62.5 ±4.63 (n=4)	78.0** ±4.163 (n=4)	80.8 ±4.715 (n=4)	83.0 ^a ±3.697 (n=4)	84.5 ^{be} ±4.992 (n=4)
TOTAL PLASMA PROTEIN (g/dL)					
Group I	5.63 ±0.301 (n=4)	9.13** ±0.256 (n=4)	10.73** ±0.170 (n=4)	11.85* ±0.25 (n=2)	12.60 ^{N.I.} ±0.0 (n=1)
Group II	6.40 ±0.238 (n=4)	8.83** ±0.246 (n=4)	8.93 ^d ±0.218 (n=4)	9.30 ^d ±0.231 (n=3)	9.55 ±0.150 (n=2)
Group III	5.90 ±0.216 (n=4)	9.25** ±0.233 (n=4)	7.80 ^{bdf} ±0.147 (n=4)	6.55 ^{bdf} ±0.456 (n=4)	6.30 ^{bf} ±0.303 (n=4)
BLOOD UREA NITROGEN (mg/dL)					
Group I	14.25 ±1.548 (n=4)	26.25** ±1.702 (n=4)	36.00** ±1.472 (n=4)	45.00 ±1.0 (n=2)	52.00 ^{N.I.} ±0.0 (n=1)
Group II	6.90 ±1.168 (n=4)	12.60** ±1.374 (n=4)	21.83 ^{bd} ±1.969 (n=4)	30.93 ^{ad} ±1.026 (n=3)	35.30 ^{N.I.} ±1.1 (n=2)
Group III	6.15 ±0.776 (n=4)	19.48** ±0.890 (n=4)	17.38 ^d ±2.027 (n=4)	14.00 ^{adf} ±1.268 (n=4)	9.25 ^{bf} ±0.457 (n=4)
CREATININE (mg/dL)					
Group I	1.03 ±0.34 (n=4)	1.22 ±0.025 (n=4)	1.50** ±0.071 (n=4)	2.12* ±0.065 (n=2)	2.64 ^{N.I.} ±0.0 (n=1)
Group II	0.96 ±0.639 (n=4)	1.28** ±0.051 (n=4)	1.48 ^b ±0.030 (n=4)	1.78 ^{ad} ±0.012 (n=3)	2.27 ^a ±0.065 (n=2)
Group III	0.90 ±0.029 (n=4)	1.19** ±0.069 (n=4)	1.19 ^{df} ±0.026 (n=4)	1.05 ^{df} ±0.037 (n=4)	0.94 ^{af} ±0.062 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

The plasma alanine amino transferase (ALT) recorded a significant ($P < 0.01$) increase at 24 post obstruction hours in the animals of group I as compared to base value and this rise continued till the end of study (Table 23). A significant ($P < 0.01$) rise in plasma amylase concentration was noticed at 24 hours post obstruction in the animals of all the three groups and this increase persisted ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) throughout the period of obstruction in group I (Table 23, Fig. 22).

4.2.3.2 POST TREATMENT

A non-significant increase in plasma glucose concentration was observed in the animals of group II, whereas there was significant increase in the blood glucose concentration at 72 hours ($P < 0.05$) and 96 hours ($P < 0.01$) when compared to the 24 post obstruction hours value in the animals of group III (Table 19). A significant ($P < 0.01$) decrease in blood glucose concentration was noticed at 96 hours in the animals of group III when compared to the corresponding value of group II.

A non-significant increase in total plasma protein concentration was recorded in the animals of group II when compared to its 24 hour value. Whereas, a significant ($P < 0.01$) decline in the TPR concentration was observed in the animals of group III when compared to 24 post obstruction hour. The inter group comparison revealed a significant ($P < 0.01$) increase in total protein concentration in the animals of group II when compared to group I. Similarly, the animals of group III showed a significant ($P < 0.01$) decrease in plasma protein concentration when compared to group I and II (Table 19, Fig. 18).

The increased blood urea nitrogen concentration was significant ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) in the animals of group II, whereas there was a

TABLE 20: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON PLASMA TOTAL BILIRUBIN IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
Group I	0.16 ±0.020 (n=4)	0.18 ±0.013 (n=4)	0.26 ^{**} ±0.015 (n=4)	0.42 ±0.025 (n=2)	0.52 ^{N.I.} ±0.0 (n=1)
Group II	0.11 ±0.125 (n=4)	0.18* ±0.012 (n=4)	0.22 ±0.019 (n=4)	0.29 ±0.035 (n=3)	0.41 ±0.05 (n=2)
Group III	0.12 ±0.013 (n=4)	0.18* ±0.014 (n=4)	0.25 ^b ±0.026 (n=4)	0.19 ±0.206 (n=4)	0.17 ^{af} ±0.028 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

significant ($P < 0.05$: 72 hours and $P < 0.01$: 96 hours) decrease in BUN in the animals of group III when compared to 24 hours value. The inter-group comparison revealed a significant ($P < 0.01$) decrease in BUN values in the animals of group II and III when compared to group I at 48 and 72 hours and a significant ($P < 0.01$) fall in BUN concentration was noticed at 72 and 96 hours in the animals of group III when compared with group II (Table 19). The plasma creatinine concentration remained significantly ($P < 0.01$: 48 hours and $P < 0.05$: 72, 96 hours) high when compared to its 24 hours value in the animals of group II, whereas there was a significant ($P < 0.05$) decrease in plasma creatinine at 96 hours when compared to 24 post obstruction hours in the animals of group III. The value recorded at 96 hours was almost comparable to its base value. On inter-group comparison there was a significant ($P < 0.01$) decline in plasma creatinine concentration in the animals of group II at 72 hours as compared to corresponding interval of group I. Likewise, the animals of group III showed statistically significant

TABLE 21: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON PLASMA SODIUM, POTASSIUM, CHLORIDE AND CALCIUM IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
SODIUM (mEq/L)					
Group I	144.0 ±1.63 (n=4)	122.5** ±3.30 (n=4)	116.5** ±1.71 (n=4)	108.0 ±4.0 (n=2)	104.0 ^{N.I.} ±0.0 (n=1)
Group II	142.5 ±0.957 (n=4)	136.0** ±0.816 (n=4)	134.5 ^d ±1.708 (n=4)	124.6 ^{bc} ±1.764 (n=3)	119.0 ^{N.I.} ±1.0 (n=2)
Group III	145.0 ±1.00 (n=4)	136.0** ±0.817 (n=4)	137.5 ^d ±1.258 (n=4)	141.0 ^{adf} ±1.291 (n=4)	142.0 ^{bf} ±1.155 (n=4)
POTASSIUM (mEq/L)					
Group I	5.18 ±0.409 (n=4)	3.40** ±0.071 (n=4)	3.18** ±0.095 (n=4)	2.90* ±0.30 (n=2)	2.80 ^{N.I.} ±0.0 (n=1)
Group II	5.05 ±0.222 (n=4)	3.40** ±0.071 (n=4)	3.25 ^a ±0.065 (n=4)	3.03 ^b ±0.088 (n=3)	2.95 ^{N.I.} ±0.15 (n=2)
Group III	5.15 ±0.171 (n=4)	3.55** ±0.096 (n=4)	3.80 ^{df} ±0.071 (n=4)	4.08 ^{adf} ±0.085 (n=4)	4.90 ^{bf} ±0.129 (n=4)
CHLORIDE (mEq/L)					
Group I	103.45 ±0.913 (n=4)	68.63** ±3.035 (n=4)	66.75** ±3.064 (n=4)	64.75* ±4.95 (n=2)	63.50 ^{N.I.} ±0.0 (n=1)
Group II	104.28 ±0.485 (n=4)	67.58** ±2.677 (n=4)	72.08 ^a ±1.687 (n=4)	75.83 ^a ±2.061 (n=3)	76.55 ^{N.I.} ±3.85 (n=2)
Group III	105.25 ±0.184 (n=4)	68.30** ±3.684 (n=4)	77.35 ^{bcd} ±1.169 (n=4)	97.70 ^{bdf} ±1.092 (n=4)	102.38 ^{bf} ±0.826 (n=4)
CALCIUM (mg/dL)					
Group I	7.53 ±0.359 (n=4)	7.48 ±0.435 (n=4)	7.30 ±0.314 (n=4)	7.10 ±0.7 (n=2)	7.33 ^{N.I.} ±0.0 (n=1)
Group II	7.83 ±0.266 (n=4)	7.43 ±0.366 (n=4)	7.45 ±0.429 (n=4)	7.63 ±0.536 (n=3)	7.55 ±0.75 (n=2)
Group III	6.90 ±0.367 (n=4)	6.73 ±0.165 (n=4)	6.63 ±0.333 (n=4)	7.07 ±0.272 (n=4)	7.05 ±0.218 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

TABLE 22: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON PLASMA PHOSPHORUS IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
Group I	5.28 ±0.349 (n=4)	5.15 ±0.312 (n=4)	5.25 ±0.296 (n=4)	5.10 ±0.6 (n=2)	5.20 ^{N.I.} ±0.0 (n=1)
Group II	5.03 ±0.304 (n=4)	5.25 ±0.384 (n=4)	5.18 ±0.401 (n=4)	5.20 ±0.493 (n=3)	5.55 ±0.25 (n=2)
Group III	5.40 ±0.092 (n=4)	5.43 ±0.188 (n=4)	5.13 ±0.103 (n=4)	5.23 ±0.111 (n=4)	5.40 ±0.108 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

($P < 0.01$) fall in the values of plasma creatinine from 48 hours onward when compared to animals of group II and I (Table 19).

A gradual and non-significant increase in plasma total bilirubin concentration recorded during the entire post treatment period in the animals of group II when compared to 24 post obstruction hour value. The animals of group III recorded a significant ($P < 0.01$) increase in total bilirubin at 48 hours when compared to 24 hours value, thereafter, the concentration decreased and the drop was significant ($P < 0.05$) at 96 hours in comparison to 24 post obstruction hour value. On comparative basis a significant ($P < 0.01$) decrease in plasma bilirubin concentration was observed at 96 hours in the animals of group III as compared to group II (Table 20).

There was a gradual decrease in plasma sodium concentration in the animals of group II throughout the period of study when compared with 24 post obstruction hours value, this decrease at 72 hours was significant ($P < 0.01$) and it continued upto 96 hours. A significant ($P < 0.05$: 72 hours and $P < 0.01$: 96 hours)

TABLE 23: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON PLASMA ALKP, AST, ALT AND AMYLASE IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
ALKALINE PHOSPHATASE (IU/L)					
Group I	92.25 ±1.652 (n=4)	131.25** ±2.428 (n=4)	145.25** ±1.436 (n=4)	154.00* ±4.00 (n=2)	156.00 ^{N.I.} ±0.0 (n=1)
Group II	93.00 ±2.646 (n=4)	130.75** ±2.78 (n=4)	138.25 ^{bc} ±1.887 (n=4)	142.67 ^a ±2.333 (n=3)	147.50 ^{N.I.} ±1.50 (n=2)
Group III	90.25 ±6.033 (n=4)	131.50** ±4.481 (n=4)	137.50 ^{bc} ±2.102 (n=4)	121.50 ^{bdf} ±1.443 (n=4)	107.50 ^{bf} ±1.708 (n=4)
AST (IU/L)					
Group I	82.3 ±2.955 (n=4)	149.5** ±4.924 (n=4)	161.0** ±3.582 (n=4)	172.5* ±1.50 (n=2)	178.0 ^{N.I.} ±0.0 (n=1)
Group II	80.0 ±5.164 (n=4)	154.8** ±3.351 (n=4)	161.8 ^b ±3.172 (n=4)	170.0 ^a ±3.215 (n=3)	174.5 ^{N.I.} ±1.5 (n=2)
Group III	82.5 ±7.932 (n=4)	156.5** ±8.49 (n=4)	139.5 ^{bce} ±5.172 (n=4)	121.0 ^{bdf} ±2.858 (n=4)	106.3 ^{bf} ±2.394 (n=4)
ALT (IU/L)					
Group I	21.8 ±1.377 (n=4)	30.0** ±1.225 (n=4)	39.8** ±1.601 (n=4)	50.5* ±4.95 (n=2)	60.0 ^{N.I.} ±0.0 (n=1)
Group II	24.0 ±1.225 (n=4)	26.8 ±0.75 (n=4)	30.3 ^b ±2.109 (n=4)	35.0 ^a ±1.0 (n=3)	50.5 ^a ±0.5 (n=2)
Group III	23.5 ±1.190 (n=4)	33.0 ±0.189 (n=4)	31.5 ±1.19 (n=4)	29.0 ±0.707 (n=4)	24.8 ^b ±1.493 (n=4)
AMYLASE IU/L					
Group I	11.25 ±0.854 (n=4)	25.75** ±1.25 (n=4)	31.75** ±2.25 (n=4)	38.50* ±1.5 (n=2)	44.00 ^{N.I.} ±0.0 (n=1)
Group II	14.25 ±0.854 (n=4)	24.00** ±1.472 (n=4)	29.25 ^a ±2.175 (n=4)	32.00 ^a ±3.215 (n=3)	36.50 ^a ±3.5 (n=2)
Group III	16.25 ±0.75 (n=4)	33.25** ±0.125 (n=4)	33.50 ±0.194 (n=4)	24.50 ^{bc} ±2.394 (n=4)	20.00 ^{bf} ±1.414 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

FIG. 18: VARIATIONS IN TOTAL PLASMA PROTEIN FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

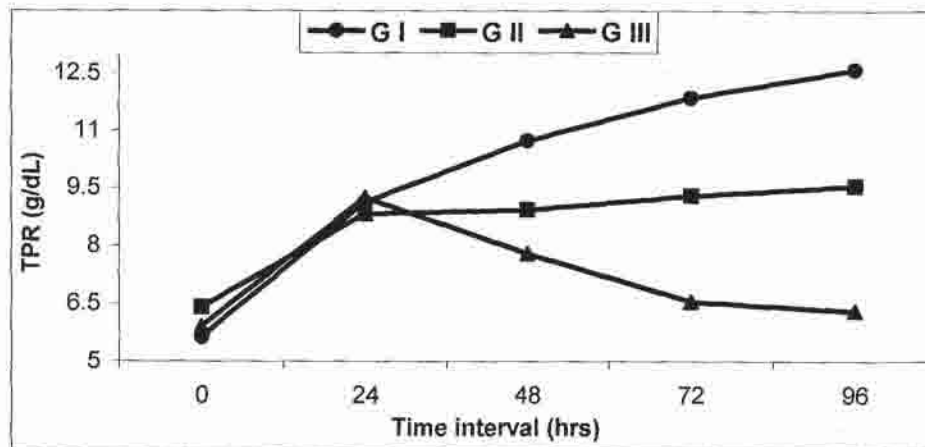


FIG. 19: VARIATIONS IN PLASMA POTASSIUM CONCENTRATION FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

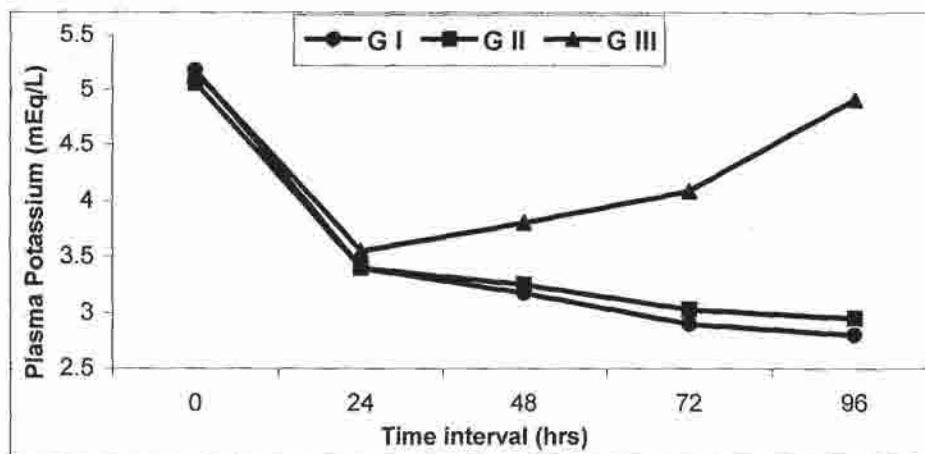


FIG. 20: VARIATIONS IN PLASMA CHLORIDE FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

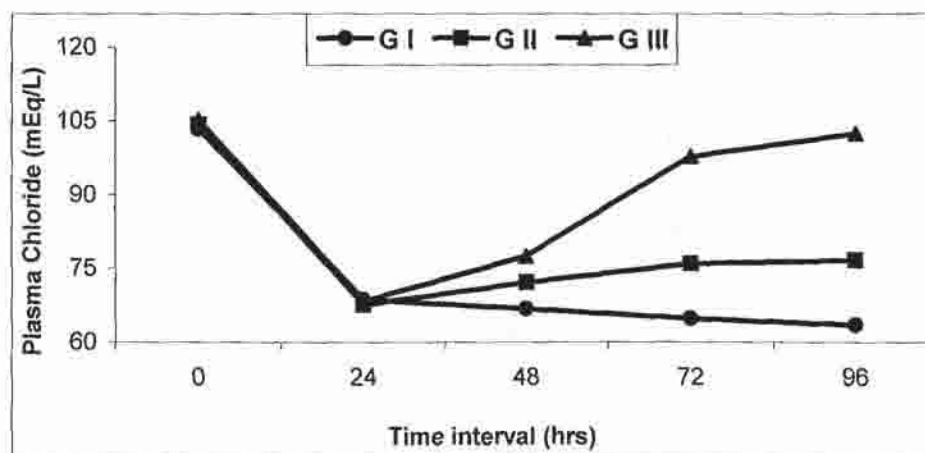


FIG. 21: VARIATIONS IN PLASMA ALKALINE PHOSPHATASE FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

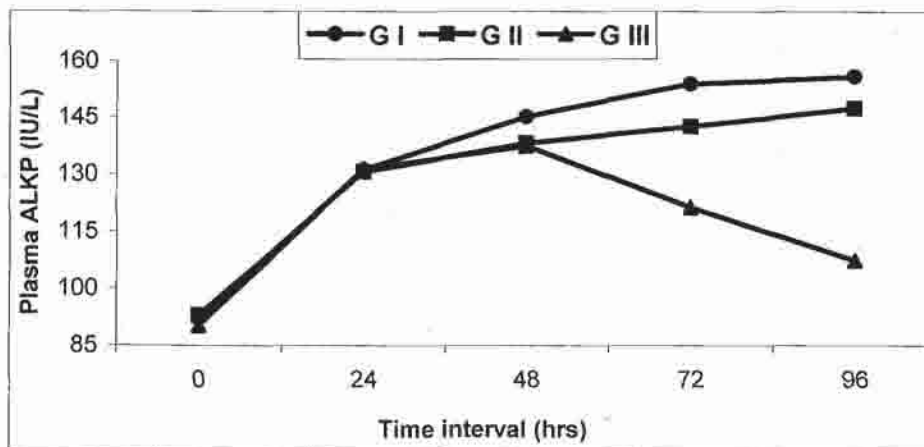
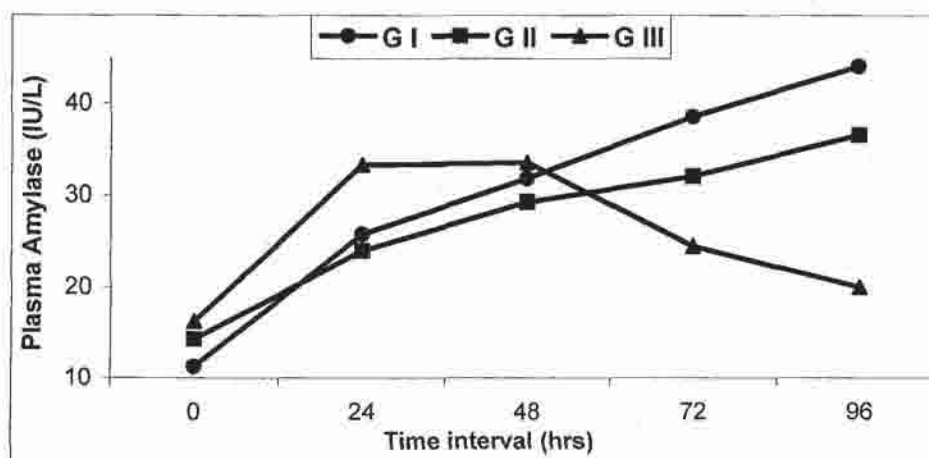


FIG. 22: VARIATIONS IN PLASMA AMYLASE FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.



rise in sodium concentration was recorded when compared to its 24 post obstruction hours value in the animals of group III. At the end of study it reached almost to normal in comparison to base value. A significant hyponatraemia was present in group I at 48 ($P < 0.01$) and 72 ($P < 0.05$) hours as compared to group II. Comparison between group I and III revealed that there was a significant ($P < 0.01$) increase in the sodium concentration in the animals of group III at 48 and 72 hours onward till the end of study. A significant ($P < 0.01$) increase in plasma sodium concentration was recorded in the animals of group III when compared to animals

of group II at 72 and 96 hours (Table 21). The plasma potassium concentration dropped significantly ($P<0.05$) at 48 and 72 hours ($P<0.01$) in the animals of group II when compared with 24 post obstruction hours. In the animals of group III the potassium concentration was significantly high in the post treatment period ($P<0.05$: 72 hours and $P<0.01$: 96 hours) when compared to 24 hour value. The inter-group comparison revealed significant ($P<0.01$) increase in the potassium concentration in the animals of group III as compared to group II (Table 21, Fig. 19). A significant ($P<0.05$) increase in chloride concentration was noticed in the animals of group II when compared to 24 post obstruction hour. Similarly, increase in plasma chloride concentration was significantly ($P<0.01$) higher in the animals of group III when compared to 24 hour value. The inter-group comparison revealed a significant recovery in plasma chloride concentration in the animals of group III as compared to group II ($P<0.01$) and I ($P<0.05$: 48 hours and $P<0.01$: 72 hours) (Table 21, Fig. 20). No statistically significant alterations were seen in plasma calcium and phosphorus concentration during post treatment period in the animals of group II and III (Table 21, 22).

A significant increase in plasma alkaline phosphatase (ALKP) was recorded in the animals of group II at 48 ($P<0.01$) and 72 hours ($P<0.05$), whereas, the decrease in ALKP was significant ($P<0.05$: 48 hours and $P<0.01$: 72, 96 hours) in the animals of group III when compared to 24 post obstruction hours. On comparative basis a significant ($P<0.05$) increase was noticed in alkaline phosphatase concentration in the animals of group II when compared to group I at 48 hours. A significant ($P<0.01$) decrease in alkaline phosphatase was seen in the animals of group III at 72 hours when compared to group I and II. Likewise, a significant ($P<0.01$) decrease in ALKP in the animals of group III was observed at

96 hours when compared with group II (Table 23, Fig. 21). A statistically significant ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) rise in plasma AST concentration was maintained in the animals of group II, whereas, a significant ($P < 0.01$) decrease in AST concentration was noticed in the animals of group III as compared to 24 hour values. The inter-group comparison revealed a statistically significant decrease in AST concentration in the animals of group III as compared to group I ($P < 0.05$: 48 hours and $P < 0.01$: 72 hours) and II ($P < 0.05$: 48 hours and $P < 0.01$: 72, 96 hours) (Table 23). A rise in plasma ALT concentration in the animals of group II was statistically significant ($P < 0.01$: 48 hours and $P < 0.05$: 72, 96 hours) in the entire post treatment period as compared to its 24 hours value. A gradual decline approaching to normalcy was observed in the animals of group III as evidenced by a significantly ($P < 0.01$) lower concentration of ALT at 96 hour when compared to 24 hour value (Table 23).

A significant ($P < 0.05$) rise in plasma amylase concentration was recorded during the entire period of study in the animals of group II as compared to 24 post obstruction hours. In comparison to 24 hours value a significant ($P < 0.01$) decline in plasma amylase was observed at 72 and 96 hours in the animals of group III. The inter-group comparison revealed a significant drop in amylase concentration in the animals of group III as compared to group I ($P < 0.05$) at 72 hours and group II ($P < 0.01$) at 96 hours (Table 23, Fig. 22).

4.2.4 CHANGES IN PERITONEAL FLUID

4.2.4.1 PRE TREATMENT

The normal straw colour of peritoneal fluid showed a yellowish tinge following creation of intestinal obstruction in all the animals of three groups at 24 post obstruction hours. As the duration of obstruction progressed in the animals of

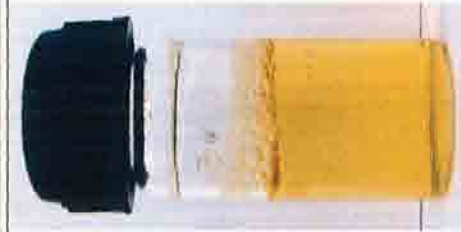
group I the peritoneal fluid appeared deep yellow in colour (Plate 11). A decreasing trend in the peritoneal fluid pH was observed immediately after creation of strangulated intestinal obstruction in all the animals of three groups and this decrease was significant ($P < 0.01$) at 72 hours in the animals of group I (Table 24).

A significant ($P < 0.01$) increase in the total protein of peritoneal fluid (Table 24, Fig. 23) was observed at 24 hours in comparison to base value in all the animals of three groups. The increase in total protein of peritoneal fluid remained significantly ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) high throughout the period of study in the animals of group I (Table 24). A significant ($P < 0.01$) increase in peritoneal fluid nucleated cell count was observed in all the animals of three groups at 24 hours when compared to base values. A continual increase in the nucleated cell count was recorded during entire period of observation in the animals of group I which was significant ($P < 0.01$) at 48 hours (Table 24, Fig. 24).

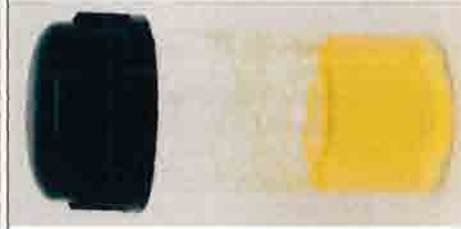
A significant decrease in peritoneal fluid sodium concentration was recorded at 24 post obstruction hours in the animals of group II ($P < 0.05$) and III ($P < 0.01$), when compared with base values. However, in the animals of group I a significant ($P < 0.01$) decrease was noticed at 48 hours (Table 25). The decrease in peritoneal fluid potassium was significant ($P < 0.01$) in all the animals of three groups at 24 hours when compared to respective base values. This decrease was consistent in the animals of group I till the end of the period of observation but the decrease was significant ($P < 0.01$) only at 48 hours (Table 25).

The chloride concentration in peritoneal fluid decreased significantly ($P < 0.01$) at 24 hours when compared with base values in the animals of group I and

**PLATE 11: PERITONEAL FLUID CHANGES AFTER CREATION OF STRANGULATED JEJUNAL OBSTRUCTION
IN CALVES**



"0" Day (Base Value)



24 hours post creation



48 hours post creation



72 hours post creation



96 hours post creation

TABLE 24: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON PERITONEAL FLUID pH, TOTAL PROTEIN AND NUCLEATED CELL COUNT IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
PERITONEAL FLUID Ph					
Group I	6.93 ±0.075 (n=4)	6.85 ±0.087 (n=4)	6.78 ±0.075 (n=4)	6.55 ^b ±0.15 (n=2)	6.40 ^{N.I.} ±0.0 (n=1)
Group II	6.85 ±0.087 (n=4)	6.78 ±0.075 (n=4)	6.60 ±0.058 (n=4)	6.60 ±0.1 (n=3)	6.85 ±0.15 (n=2)
Group III	6.70 ±0.123 (n=4)	6.55 ±0.194 (n=4)	6.63 ±0.075 (n=4)	6.55 ±0.027 (n=4)	6.63 ±0.075 (n=4)
TOTAL PERITONEAL FLUID PROTEIN (g/dL)					
Group I	2.93 ±0.155 (n=4)	4.80 ^{**} ±0.178 (n=4)	5.35 ^{**} ±0.144 (n=4)	6.65 [*] ±0.150 (n=2)	7.30 ^{N.I.} ±0.0 (n=1)
Group II	2.73 ±0.125 (n=4)	4.10 ^{**} ±0.265 (n=4)	4.73 ^b ±0.218 (n=4)	5.37 ^{bc} ±0.296 (n=3)	6.10 ^a ±0.2 (n=2)
Group III	2.40 ±0.147 (n=4)	4.48 ^{**} ±0.16 (n=4)	4.10 ^d ±0.204 (n=4)	3.38 ^{def} ±0.085 (n=4)	2.98 ^{df} ±0.165 (n=4)
NUCLEATED CELL COUNT (x 10³/cu mm)					
Group I	2.94 ±0.083 (n=4)	6.38 ^{**} ±0.188 (n=4)	6.68 ^{**} ±0.222 (n=4)	7.15 ±0.300 (n=2)	6.90 ^{N.I.} ±0.0 (n=1)
Group II	3.04 ±0.116 (n=4)	7.33 ^{**} ±0.364 (n=4)	7.48 ^a ±0.350 (n=4)	7.68 ±0.460 (n=3)	8.35 ±0.200 (n=2)
Group III	2.95 ±0.184 (n=4)	7.43 ^{**} ±0.259 (n=4)	7.73 ^{bc} ±0.249 (n=4)	7.88 ^b ±0.247 (n=4)	8.11 ^d ±0.263 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

II. The drop ($P < 0.01$: 48 hours) in chloride concentration of peritoneal fluid was constant throughout the period of study in the animals of group I (Table 25, Fig. 25). The peritoneal fluid calcium and phosphorus concentration did not reveal any

TABLE 25: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON PERITONEAL FLUID SODIUM, POTASSIUM, CHLORIDE AND CALCIUM IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
SODIUM (mEq/L)					
Group I	138.5 ±0.96 (n=4)	135.5 ±1.26 (n=4)	128.0** ±2.16 (n=4)	121.0 ±3.0 (n=2)	112.0 ^{N.I.} ±0.0 (n=1)
Group II	144.5 ±2.06 (n=4)	140.0* ±1.83 (n=4)	135.0 ^{ac} ±1.29 (n=4)	128.6 ^a ±1.764 (n=3)	126.0 ±2.0 (n=2)
Group III	142.5 ±0.957 (n=4)	139.0** ±0.577 (n=4)	140.5 ^{df} ±0.50 (n=4)	141.0 ^{df} ±0.58 (n=4)	141.5 ^f ±0.50 (n=4)
POTASSIUM (mEq/L)					
Group I	4.68 ±0.193 (n=4)	3.98** ±0.86 (n=4)	3.60** ±0.141 (n=4)	3.60 ±0.2 (n=2)	3.10 ^{N.I.} ±0.0 (n=1)
Group II	4.58 ±0.132 (n=4)	4.08** ±0.111 (n=4)	3.73 ^a ±0.049 (n=4)	3.40 ^a ±0.115 (n=3)	3.10 ^{N.I.} ±0.30 (n=2)
Group III	4.68 ±0.138 (n=4)	4.05** ±0.096 (n=4)	4.30 ^{df} ±0.041 (n=4)	4.38 ^{df} ±0.085 (n=4)	4.43 ^e ±0.155 (n=4)
CHLORIDE (mEq/L)					
Group I	96.33 ±2.401 (n=4)	83.55** ±2.021 (n=4)	81.78** ±1.624 (n=4)	75.80 ±1.4 (n=2)	69.30 ^{N.I.} ±0.0 (n=1)
Group II	92.15 ±2.408 (n=4)	86.20** ±2.043 (n=4)	81.30 ^a ±1.369 (n=4)	75.87 ^b ±1.374 (n=3)	74.45 ^{N.I.} ±0.85 (n=2)
Group III	93.97 ±3.772 (n=4)	94.28 ±1.512 (n=4)	96.18 ^{df} ±2.116 (n=4)	93.15 ^{df} ±3.365 (n=4)	93.18 ^f ±1.894 (n=4)
CALCIUM (mg/dL)					
Group I	6.53 ±0.272 (n=4)	6.30 ±0.279 (n=4)	6.57 ±0.243 (n=4)	6.70 ±0.50 (n=2)	6.60 ^{N.I.} ±0.0 (n=1)
Group II	6.23 ±0.111 (n=4)	5.97 ±0.063 (n=4)	6.20 ±0.108 (n=4)	6.16 ±0.145 (n=3)	6.15 ±0.05 (n=2)
Group III	6.48 ±0.293 (n=4)	6.38 ±0.236 (n=4)	6.85 ±2.72 (n=4)	6.43 ±0.309 (n=4)	6.43 ±0.206 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

significant alteration following strangulated intestinal obstruction in all the animals of three groups (Table 25, 26).

4.2.4.2 POST TREATMENT

The peritoneal fluid retained its deep yellow colour in the animals of group II during the entire post treatment period, whereas, the colour of peritoneal fluid in the animals of group III was dark yellow at 48 hours following treatment but gradually it returned to normal straw colour from 72 hours onward.

The concentration of peritoneal fluid total protein increased significantly ($P < 0.01$: 48, 72 hours and $P < 0.05$: 96 hours) in the animals of group II when compared to 24 hour value, whereas, it decreased significantly ($P < 0.01$) from 72 hours onward in the animals of group III as compared to 24 post obstruction hours. The comparison within groups revealed significant ($P < 0.05$) rise in the peritoneal fluid protein concentration in the animals of group I as compared to group II at 72 hours. Similarly, a significant ($P < 0.01$) decrease in peritoneal fluid protein concentration was observed in the animals of group III when compared to group I (48 and 72 hours) and II (72 and 96 hours) (Table 24, Fig. 23). In all the animals of group II the nucleated cell count of peritoneal fluid remained high ($P < 0.01$: 48 hours) throughout the post treatment period when compared to 24 post obstruction hours. In the animals of group III a significant ($P < 0.01$) increase in the cell count was recorded throughout the post operative period when compared to 24 hours. The inter-group comparison revealed a significant rise in the cell count in the animals of group III when compared to group I at 48 hours interval (Table 24, Fig. 24).

There was a significant ($P < 0.05$: 48, 72 hours) decrease in peritoneal fluid sodium concentration in the animals of group II, whereas, no significant change in

TABLE 26: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON PERITONEAL FLUID PHOSPHORUS IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
Group I	5.08 ±0.375 (n=4)	5.03 ±0.349 (n=4)	5.03 ±0.446 (n=4)	4.95 ±0.15 (n=2)	5.10 ^{N.I.} ±0.0 (n=1)
Group II	4.80 ±0.358 (n=4)	4.77 ±0.240 (n=4)	4.88 ±0.368 (n=4)	4.73 ±0.41 (n=3)	4.65 ±0.55 (n=2)
Group III	3.93 ±0.602 (n=4)	3.90 ±0.652 (n=4)	4.05 ±0.792 (n=4)	3.80 ±0.803 (n=4)	3.63 ±0.662 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

peritoneal fluid sodium concentration was observed in the animals of group III when compared to 24 post obstruction hours. The animals of group III when compared to group II showed significantly ($P<0.01$) increasing trend in peritoneal fluid sodium concentration during post treatment period and this increase was significant ($P<0.01$) in comparison to group I at 48 and 72 hours (Table 25).

A significant ($P<0.05$) decrease in peritoneal fluid potassium concentration was observed in the animals of group II when compared to 24 hours value but it continued to rise from 48 hours onward in the animals of group III. The inter-group comparison revealed a significant recovery in potassium concentration in the animals of group III at 48 ($P<0.01$) and 72 ($P<0.05$) hours when compared with group I. Similarly, a significant increase in peritoneal fluid potassium was observed in the animals of group III ($P<0.01$: 48, 72 hours and $P<0.05$: 96 hours) when compared with group II (Table 25). In all the animals of group II a significant decrease in the peritoneal fluid chloride concentration was noticed at 48 ($P<0.05$)

FIG. 23: VARIATIONS IN PERITONEAL FLUID PROTEIN FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

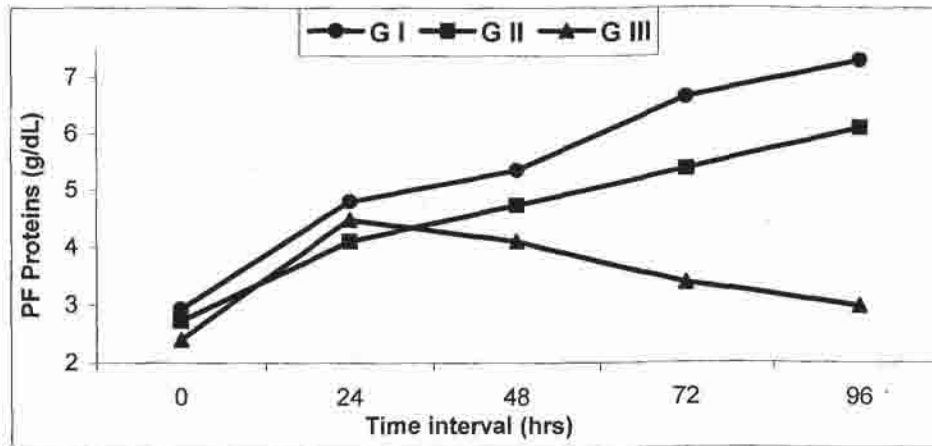


FIG. 24: VARIATIONS IN PERITONEAL FLUID NUCLEATED CELLS FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

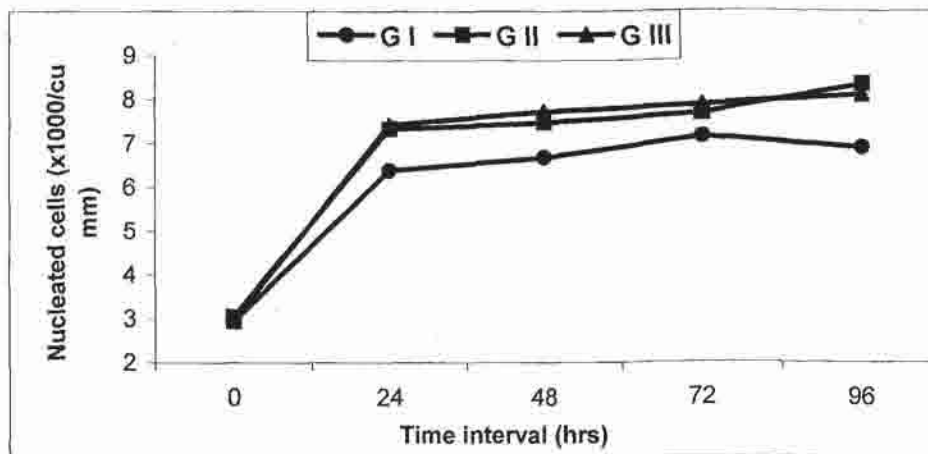
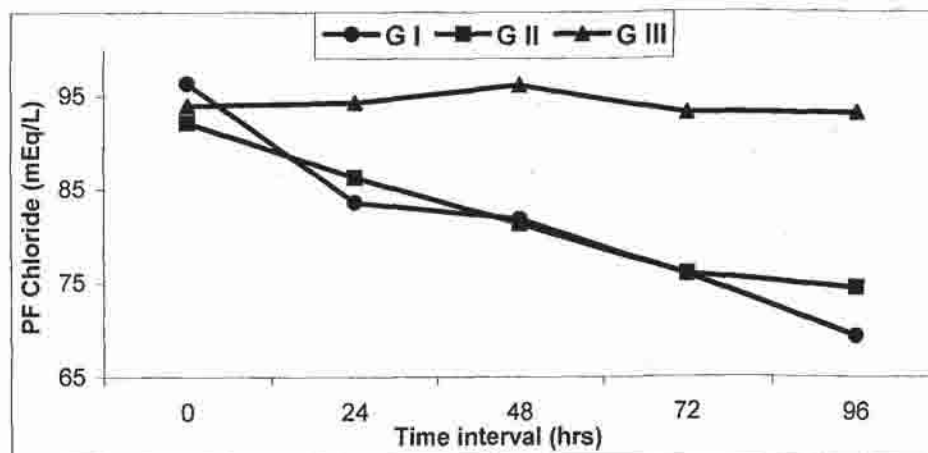


FIG. 25: VARIATIONS IN PERITONEAL FLUID CHLORIDE FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.



and 72 ($P<0.01$) hours, when compared to 24 post obstruction hours. The peritoneal fluid chloride level in the animals of group III almost remained unaltered. On comparative basis (Table 25) the animals of group III showed significant recovery in peritoneal fluid chloride concentration when compared to group I ($P<0.01$: 48 hours and $P<0.05$: 72 hours) and II ($P<0.01$: 48, 72, 96 hours). No significant changes in peritoneal fluid calcium and phosphorus concentration were seen in the animals of group II and III (Table 25, 26, Fig. 25).

4.2.5 RUMINAL FLUID BIOCHEMISTRY

4.2.5.1 PRE TREATMENT

There was significant ($P<0.01$) decrease in ruminal fluid sodium concentration at 24 post obstruction hours when compared to base values in the animals of group I and II. This decrease was gradual during rest of the post obstruction period in the animals of group I but it was significant ($P<0.01$) at 48 hours (Table 27). The ruminal fluid potassium (Table 27, Fig. 26) concentration dropped significantly ($P<0.01$) at 24 hours when compared to base value in the animals of group I and II. This decline in ruminal fluid potassium concentration was significant ($P<0.01$: 48 hours and $P<0.05$: 72 hours) throughout the period of obstruction in the animals of group I.

The increase in ruminal fluid chloride concentration was significant ($P<0.01$) at 24 hours in all the animals of three groups when compared to respective base values. The concentration of chloride remained significantly ($P<0.01$) high throughout the period of study in the animals of group I (Table 27, Fig. 27). Ruminal fluid calcium concentration did not show any significant changes at 24 post obstruction hour in the animals of group I, II and III (Table 28). A significant ($P<0.01$) increase in ruminal fluid phosphorus was recorded at 24 post obstruction

TABLE 27: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON RUMINAL FLUID SODIUM, POTASSIUM AND CHLORIDE IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
SODIUM (mEq/L)					
Group I	134.5 ±1.5 (n=4)	126.5** ±2.06 (n=4)	124.5** ±2.217 (n=4)	125.0 ±1.0 (n=2)	122.0 ^{N.I.} ±0.0 (n=1)
Group II	134.0 ±0.41 (n=4)	130.0** ±0.41 (n=4)	127.5 ^a ±0.96 (n=4)	126.7 ±0.67 (n=3)	124.5 ±0.50 (n=2)
Group III	131.5 ±1.71 (n=4)	133.5 ±2.5 (n=4)	131.5 ^c ±1.5 (n=4)	130.0 ^{ce} ±0.817 (n=4)	133.5 ^e ±1.71 (n=4)
POTASSIUM (mEq/L)					
Group I	27.5 ±0.958 (n=4)	24.25** ±0.63 (n=4)	17.75** ±0.75 (n=4)	15.5* ±1.5 (n=2)	12.0 ^{N.I.} ±0.0 (n=1)
Group II	26.5 ±0.87 (n=4)	22.0** ±1.08 (n=4)	18.8 ^b ±1.25 (n=4)	15.67 ^b ±1.202 (n=3)	12.5 ^{N.I.} ±0.50 (n=2)
Group III	26.5 ±2.63 (n=4)	23.5 ±2.53 (n=4)	27.4 ^{df} ±3.81 (n=4)	26.25 ^{cf} ±3.64 (n=4)	28.0 ^f ±3.37 (n=4)
CHLORIDE (mEq/L)					
Group I	29.50 ±1.19 (n=4)	40.75** ±0.854 (n=4)	53.00** ±1.291 (n=4)	72.50** ±1.5 (n=2)	88.00 ^{N.I.} ±0.0 (n=2)
Group II	27.80 ±1.89 (n=4)	43.45** ±2.374 (n=4)	56.70 ^a ±3.34 (n=4)	79.20 ^a ±3.564 (n=2)	92.60 ^{N.I.} ±2.0 (n=2)
Group III	29.30 ±0.78 (n=4)	42.15** ±0.788 (n=4)	41.18 ^{de} ±2.87 (n=4)	37.95 ^{df} ±1.168 (n=4)	31.60 ^{df} ±1.786 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

hours in all the animals of three groups when compared with respective base values. A consistently high ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) concentration of phosphorus was conspicuous in the ruminal fluid throughout the entire period of study in the animals of group I (Table 28, Fig. 28).

TABLE 28: EFFECT OF STRANGULATED INTESTINAL OBSTRUCTION ON RUMINAL FLUID CALCIUM AND PHOSPHORUS IN CALVES (MEAN±S.E).

GROUPS	HOURS AFTER CREATION OF INTESTINAL OBSTRUCTION				
	0	24 [#]	48	72	96
CALCIUM (mg/dL)					
Group I	8.00 ±0.365 (n=4)	7.98 ±0.103 (n=4)	7.88 ±0.253 (n=4)	8.05 ±0.15 (n=2)	8.10 ^{N.I.} ±0.0 (n=1)
Group II	8.23 ±0.511 (n=4)	8.03 ±0.433 (n=4)	8.10 ±0.473 (n=4)	8.26 ±0.441 (n=3)	8.30 ±0.9 (n=2)
Group III	8.08 ±0.249 (n=4)	7.90 ±0.354 (n=4)	8.15 ±0.353 (n=4)	8.05 ±0.296 (n=4)	7.90 ±0.204 (n=4)
PHOSPHORUS (mg/dL)					
Group I	7.68 ±0.673 (n=4)	14.75 ^{**} ±0.644 (n=4)	27.25 ^{**} ±1.448 (n=4)	38.10 [*] ±2.4 (n=2)	51.20 ^{N.I.} ±0.0 (n=1)
Group II	10.00 ±1.255 (n=4)	18.25 ^{**} ±1.438 (n=4)	30.45 ^b ±1.962 (n=4)	43.23 ^b ±2.795 (n=3)	49.75 ^b ±3.05 (n=2)
Group III	13.68 ±1.180 (n=4)	22.53 ^{**} ±3.644 (n=4)	24.08 ±4.613 (n=4)	29.90 ±4.353 (n=4)	23.05 ^e ±4.565 (n=4)

- Time of institution of treatment

*, ** - 5% and 1% level of significance respectively when compared to 0 hour value of the same group.

a, b - 5% and 1% level of significance respectively when compared to 24 hour value of the same group.

c, d - 5% and 1% level of significance respectively when compared to group I value of corresponding hour.

e, f - 5% and 1% level of significance respectively when compared to group II value of corresponding hour.

N.I. - Value not included for statistical analysis within group.

4.2.5.2 POST TREATMENT

A significant ($P < 0.05$) decrease in the ruminal fluid sodium was recorded at 48 hours in the animals of group II but there was no significant change in the ruminal fluid sodium concentration in the animals of group III. The inter-group comparison revealed significant ($P < 0.05$) loss of sodium in group I as compared to group III at 48 and 72 hours. Whereas, a significant ($P < 0.05$) increase was noticed in the animals of group III at 72 and 96 hours when compared to group II (Table 27). A significant ($P < 0.01$) decrease in ruminal fluid potassium concentration was observed from 48 hours onward in the animals of group II when compared to 24

hours value, whereas, non-significant alteration was evident in potassium concentration in the animals of group III. On comparative basis a significant ($P < 0.01$: 48 hours and $P < 0.05$: 72 hours) loss of potassium was noticed in the animals of group I as compared to group III. Whereas, a significant ($P < 0.01$) recovery during post treatment period was observed in the animals of group III when compared to group II (Table 27, Fig. 26). The increase in ruminal fluid chloride concentration in the animals of group II was significant ($P < 0.05$) at 48 and 72 hours of treatment when compared with 24 hours in the animals of group II. However, a significant ($P < 0.01$) decline in ruminal fluid chloride was observed at 96 post obstruction hours in the animals of group III when compared to 24 hours. The comparison within groups (Table 27, Fig. 27) revealed a significantly low ruminal fluid chloride concentration in the animals of group III as compared to group I ($P < 0.01$: 48, 72 hours) and II ($P < 0.05$: 48 hours and $P < 0.01$: 72, 96 hours).

The ruminal fluid calcium did not show any significant alterations in the animals of group II and III in post treatment period (Table 28). The ruminal fluid phosphorus concentration continued to increase significantly ($P < 0.01$) throughout the post treatment period in the animals of group II, whereas, no significant alteration was observed in the animals of group III when compared to 24 post obstruction hours. At 96 hours in group III, the ruminal fluid phosphorus concentration was significantly ($P < 0.05$) low as compared to its corresponding value in the animals of group II (Table 28, Fig. 28).

FIG. 26: VARIATIONS IN RUMINAL FLUID POTASSIUM FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

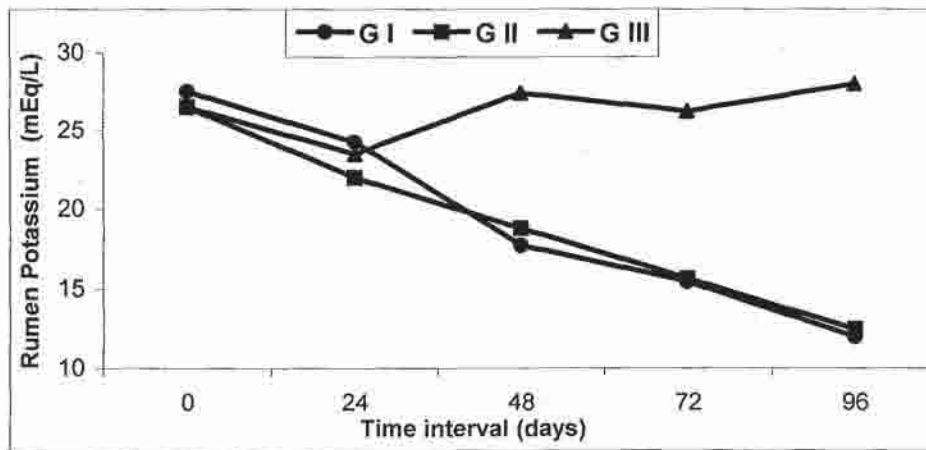


FIG. 27: VARIATIONS IN RUMINAL FLUID CHLORIDE FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.

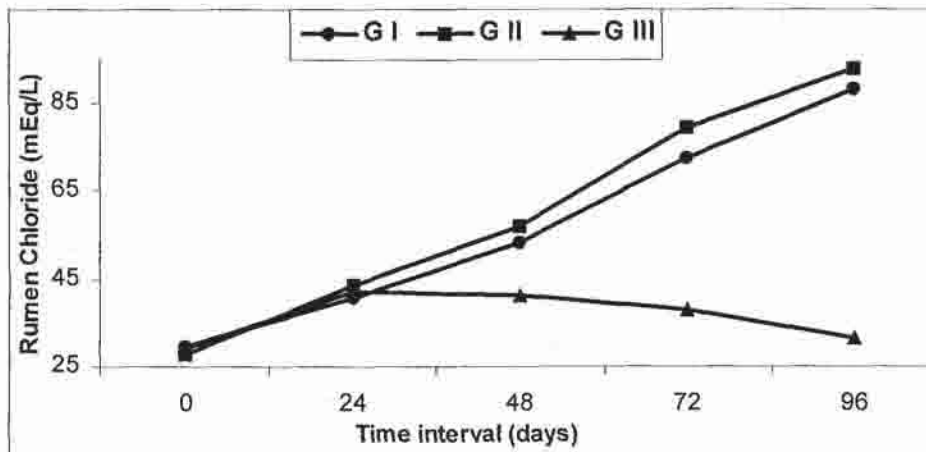
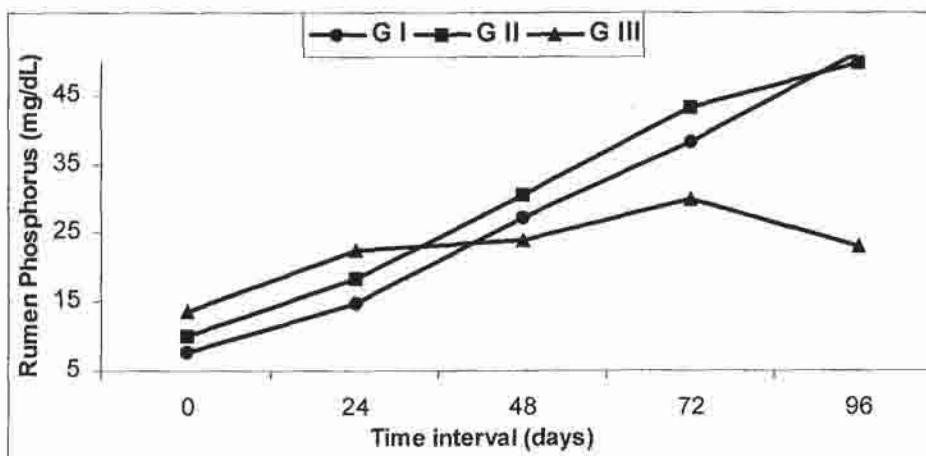


FIG. 28: VARIATIONS IN RUMINAL FLUID PHOSPHORUS FOLLOWING STRANGULATED INTESTINAL OBSTRUCTION IN CALVES.



4.2.6 OPERATIVE FINDINGS

The site of obstructed loop was discolored and appeared grayish in colour with irreversible stricture. The segment of intestine proximal to the site of obstruction was distended and was heavily congested with stagnated venous channels. The peristalsis was not seen in the immediate proximal. The distal segment was collapsed. The mesentery was extensively hemorrhagic and arterial pulsation was not present in the vicinity of affected intestine (Plate 12).

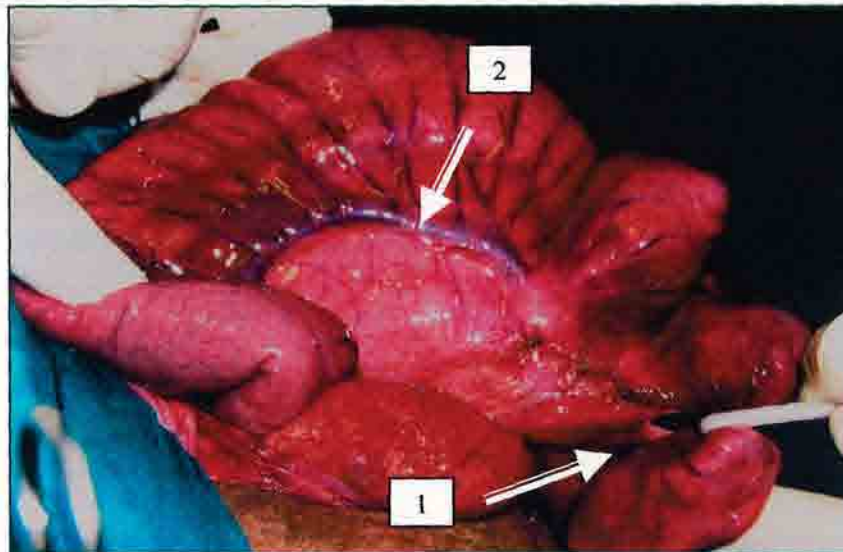
4.2.7 TOTAL SURVIVAL TIME

The animals of group I served as disease control succumbed at different time following creation of strangulated intestinal obstruction. Two animals survived upto 72 hours and only one animal survived upto 96 hours. The average survival time in the animals of group I was 78.0 ± 9.49 hours. The survival time in the animals of group II with conservative treatment was 92.25 ± 8.67 hours. All the animals of group III survived following surgical treatment.

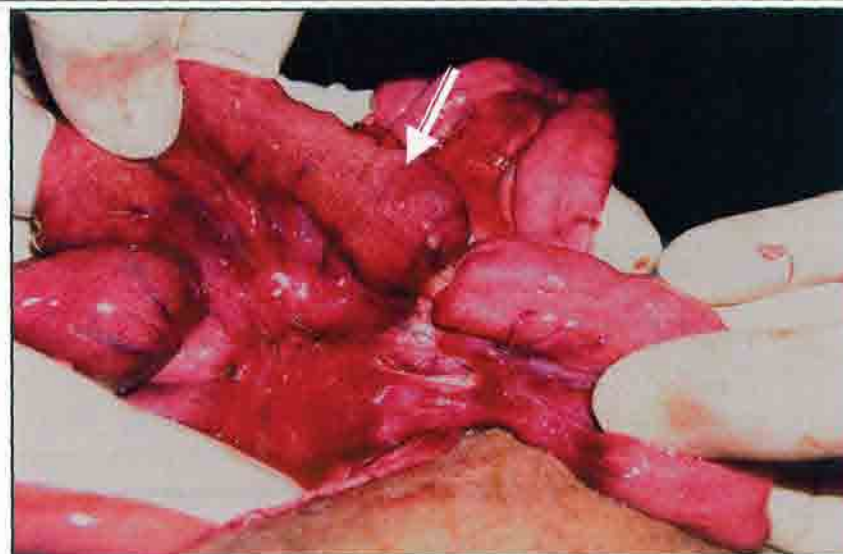
4.3.1 MICROBIOLOGICAL STUDIES

The peritoneal fluid was collected periodically before (0hour), during pretreatment and post treatment period for isolation and culture sensitivity tests. No microbial growth was observed in '0' hour samples of peritoneal fluid. Gram +ve bacilli, Gram -ve rods and Gram -ve cocobacilli were found during post treatment especially at 24 and 48 hours of strangulated and 4th day onwards in simple intestinal obstruction. The drug sensitivity profile revealed the sensitivity of the isolates to Amoxycillin, Gentamicin, Tetracycline, Ciprofloxacin, Cloxacillin, Erytromycin and Penicillin.

PLATE 12: INTESTINAL CHANGES JUST BEFORE CORRECTIVE SURGERY OF STRANGULATED JEJUNAL OBSTRUCTION IN CALVES



1. Site of obstructed loop
2. Stagnated and dilated venous channels



After removal of silicon tubing (Venous stasis and distended proximal loop)

4.3.2 ELECTROCARDIOGRAPHIC STUDIES

The electrocardiograms of group I and II revealed shallow T wave and ST segment depression at 48 hours of creation of strangulated intestinal obstruction. These changes on electrocardiogram were more pronounced in strangulated obstruction and appeared very late (6th day) in simple intestinal obstruction. One animal with simple obstruction exhibited atrial flutter and was found regurgitating the ruminal contents on the same day.

4.3.3 COMPARISON OF PATHOPHYSIOLOGICAL CHANGES BETWEEN SIMPLE AND STRANGULATED INTESTINAL OBSTRUCTION

The pathophysiological alterations which occurred in blood following creation of strangulated and simple intestinal obstruction were compared at 24 and 72 hours post obstruction respectively. All the haematological parameters were almost comparable at this stage but there was a slight increase in packed cell volume and TLC in the strangulated obstruction when compared to simple obstruction (Table 29). The plasma electrolyte concentration showed early and severe hypochloraemia, hypokalemia in the strangulated obstruction at 24 hours post obstruction as compared to 72 hours of simple obstruction. The increase in plasma total proteins concentration was marked in strangulated obstruction than in the simple one. The plasma enzymes though remained in normal range but there was considerable elevation in the concentration of ALKP, AST and ALT in all the animals of three groups subjected to strangulated intestinal obstruction when compared to simple intestinal obstruction (Table 30).

TABLE 29: COMPARISON OF CLINICAL AND HAEMATOLOGICAL PARAMETERS BETWEEN SIMPLE AND STRANGULATED INTESTINAL OBSTRUCTION (MEAN \pm S.E.)(n=12).

S. No.	Parameter	Strangulated (24 hours)	Simple (3rd day)
1	Rectal temperature ($^{\circ}$ F)	101.78 \pm 0.18	100.4 \pm 0.172
2	Respiration rate (/min)	14.5 \pm 0.544	15.58 \pm 0.570
3	Heart rate (/min)	87.17 \pm 1.313	80.75 \pm 1.332
4	CRT (sec)	0.96 \pm 0.096	1.21 \pm 0.096
5	Ruminal fluid pH	6.8 \pm 0.085	6.7 \pm 0.047
6	Haemoglobin (g%)	10.7 \pm 0.153	9.73 \pm 0.404
7	PCV (%)	36 \pm 0.749	38.33 \pm 0.882
8	TEC ($\times 10^6$ /cu mm)	6.28 \pm 0.142	6.37 \pm 0.271
9	TLC ($\times 10^3$ /cu mm)	7879.17 \pm 281.73	11062.5 \pm 319.69
10	Neutrophils	40.67 \pm 0.81	39.08 \pm 0.556
11	Lymphocytes	59 \pm 1.101	58.92 \pm 0.57
12	Ruminal Motility (/2min)	0.495 \pm 0.118	0.248 \pm 0.109

The total proteins concentration and total nucleated cell count of peritoneal fluid were higher in strangulated obstruction as compared to simple obstruction. All other parameters viz. clinical parameters, blood biochemical profile, peritoneal fluid biochemical profile and ruminal fluid biochemical profile indicated comparable pathological alterations after 24 hours of creation of strangulated obstruction and following 72 hours of simple obstruction (Table 29, 30).

TABLE 30: COMPARISON OF PATHOPHYSIOLOGICAL CHANGES IN BODY FLUIDS BETWEEN SIMPLE AND STRANGULATED INTESTINAL OBSTRUCTION (MEAN \pm S.E.) (n=12).

S. No.	Parameter	Strangulated (24 hours)	Simple (3rd day)
1	Plasma glucose (mg/dL)	74.167 \pm 2.847	77.75 \pm 2.481
2	Plasma total Protein (g/dL)	7.07 \pm 0.139	8.04 \pm 0.211
3	Plasma BUN (mg/dL)	19.44 \pm 1.825	22.83 \pm 2.786
4	Plasma creatinine (mg/dL)	1.23 \pm 0.029	2.17 \pm 0.227
5	Plasma total bilirubin (mg/dL)	0.18 \pm 0.007	0.15 \pm 0.007
6	Plasma sodium (mEq/L)	131.5 \pm 2.19	139.67 \pm 1.389
7	Plasma potassium (mEq/L)	4.13 \pm 0.041	4.0 \pm 0.136
8	Plasma chloride (mEq/L)	98.17 \pm 1.645	89.12 \pm 2.058
9	Plasma calcium (mg/dL)	7.14 \pm 0.180	6.66 \pm 0.182
10	Plasma phosphorus (mg/dL)	5.28 \pm 0.164	5.61 \pm 0.208
11	Plasma ALKP (IU/L)	109.17 \pm 2.088	112.58 \pm 2.087
12	Plasma AST (IU/L)	111.08 \pm 2.569	103.83 \pm 3.284
13	Plasma ALT (IU/L)	29.92 \pm 0.965	44.83 \pm 1.850
14	Plasma amylase (IU/L)	27.67 \pm 1.394	28.17 \pm 1.284
15	Peritoneal fluid pH	6.73 \pm 0.077	6.94 \pm 0.091
16	Peritoneal fluid total protein (g/dL)	3.98 \pm 0.134	3.8 \pm 0.14
17	Peritoneal fluid cell count (x 10 ³ /cu mm)	3.67 \pm 0.127	3.41 \pm 0.92
18	Peritoneal fluid sodium (mEq/L)	138.17 \pm 0.903	140.33 \pm 0.772
19	Peritoneal fluid potassium (mEq/L)	4.075 \pm 0.052	3.84 \pm 0.145
20	Peritoneal fluid chloride (mEq/L)	88.01 \pm 1.688	90.53 \pm 1.895
21	Peritoneal fluid calcium (mg/dL)	6.22 \pm 0.124	5.63 \pm 0.187
22	Peritoneal fluid phosphorus (mg/dL)	4.57 \pm 0.276	4.475 \pm 0.251
23	Ruminal fluid sodium (mEq/L)	130 \pm 1.371	88.5 \pm 2.105
24	Ruminal fluid potassium (mEq/L)	23.25 \pm 0.897	20.53 \pm 0.981
25	Ruminal fluid chloride (mEq/L)	42.12 \pm 0.864	53.49 \pm 1.192
26	Ruminal fluid calcium (mg/dL)	7.97 \pm 0.172	8.73 \pm 0.203
27	Ruminal fluid phosphorus (mg/dL)	18.51 \pm 1.534	34.34 \pm 2.357

4.4 AUTOPSY CHANGES

4.4.1 Simple obstruction

4.4.1.1 Gross changes

The affected segment of intestine was dark purple to bluish in colour. The segment oral to obstruction was extremely distended with fluidy and foul smelling ingesta while the distal part appeared pallor and collapsed and the mucosa was peeling off easily. The omasum contained dehydrated ingesta. Petechial hemorrhages were observed in the mesentery and peritoneum. The quantity of peritoneal fluid was more with its colour appearing high yellow. The rumen epithelium was found sloughed at certain areas.

4.4.1.2 Histopathological changes

The mucosa of the affected part of intestine showed severe necrosis and infiltration with predominantly the neutrophils. The submucosa was heavily thickened due to oedema and infiltration with lymphocytes and macrophages. Serosa was also congested and oedematous (Plate 16A).

4.4.2 Strangulated obstruction

4.4.2.1 Gross changes

The affected segment of the bowel appeared dark red, with marked wall thickening due to oedema and infiltration of blood. Mucosa was severely congested, oedematous and exhibited ulcerated surface. The Serosal surface of the distal bowel segment also appeared severely congested and haemorrhagic. Coprostatic obstruction within the distended loop of intestine was evident. The mesentery was thick and rubbery with ecchymotic areas. The peritoneal fluid in the abdominal cavity was serosanguinous. The lymph nodes within the involved

mesentery were swollen. The mucosal wall of abomasum was denuded and it contained more of the fluidy ingesta. Omasum contained dehydrated feed particles. Adhesions were present between omentum and peritoneum also involving loops of intestine.

4.4.2.2 Histopathological changes

The affected part of intestine showed complete mucosal damage and sloughing of the epithelial lining to the extent that even muscular layers were exposed at some places. The submucosa was heavily congested and oedematous. Atrophic and necrotic changes in muscularis layer were conspicuous. The serosa appeared thick due to edema and severely congested blood vessels (Plate 16B).

4.5 CLINICAL CASE STUDIES OF INTESTINAL OBSTRUCTION

A total of 15 cases suffering from intestinal obstruction in bovines, 14 at Veterinary Teaching Clinical Complex and one at Veterinary Polyclinic, Shahpur were treated during the period 2002-2005. All the patients were females except one male and their age ranged from 2-10 years.

4.5.1 HISTORY AND CLINICAL SIGNS

The history of failure of defecation ranged from 3 to 8 days. All the cases referred to University Clinics were undiagnosed but suspected for intestinal obstruction. The progression of ailment started initially with symptoms like acute intestinal colic manifested by vigorous kicking at belly, paddling of limbs, semi crouching, shifting lateral and sternal recumbencies, tremors of muscles of hind quarter. These symptoms were abolished after 2-3 days. Repeated attempts to void feces with flatus were seen. The nature of faeces became increasingly scanty with hard dung balls covered with mucous. Melena was seen frequently with mucosal shreds from rectum. All the affected animals were treated with purgatives and rumenotorics before they were referred for treatment. The animals presented after 4 days of above mentioned symptoms had reduced appetite but water intake was sufficient. Partial to complete anorexia developed over a period of hours to days in some cases. Only 3 animals were presented in recumbent posture. Twelve out of 15 animals had a history of being fed with mature bamboo leaves.

4.5.2 PHYSICAL EXAMINATION

All the animals presented with intestinal obstruction did not show any sign of acute colic. All the animals had a typical finding of soiling of tail with mucoid tarry colored faecal material. The muzzle was dry with symptoms of dehydration. The animals were not agile in walking and tended to guard their abdomen while in

motion. The rectal temperature was subnormal. The respiratory rate was often elevated with shallow respiration. In five animals the abdominal wall palpation and percussion revealed a generalized rigidity of muscles to the extent that it interfered in normal respiration and resulted in difficult thoracic respiration. The heart rate of the affected animals was found to be slightly elevated initially but as the chronicity of the case developed the heart rate increased considerably (Table 31).

TABLE 31: PREOPERATIVE CLINICAL OBSERVATIONS IN CLINICAL CASES SUFFERING FROM INTESTINAL OBSTRUCTION (MEAN \pm S.E.).

S. NO.	PARAMETER	SURVIVORS (N=6)	NON SURVIVORS (N=9)
1	Rectal Temperature ($^{\circ}$ F)	100.7 \pm 0.657	100.71 \pm 0.478
2	Heart Rate (/min)	83.33 \pm 2.459	77.11 \pm 3.128
3	Respiration Rate (/min)	25 \pm 1.693	27.88 \pm 1.399

Ruminal contractions were totally abolished with tympanitic sound on light palpation and impacted on deep palpation. Bilateral distension of abdomen was found with tightness of abdominal musculature. Affected animals were depressed and reluctant to move. The extent of dehydration in few animals was marked with eyeball recession and increased skin tent. Intestinal borborygmi on auscultation revealed mild tinkling and fluid splashing sounds but in the cases presented after 4-5 days the quiescence of abdominal cavity was conspicuous.

Per rectal examination revealed dryness and edema of the rectal mucosa with lot of tarry colored mucous and occasionally mucosal shreds. The lumen of rectum was collapsed in some animals whereas it was gas filled in other animals. The rumen had a doughy consistency and had reached almost to the brim of pelvic inlet almost in all the animals. The more cranial palpation towards right side

of the abdomen revealed gas filled intestinal loops along with an obstructed loop which appeared as thick, slightly movable and impacted mass of intestinal tract. The manipulation of this segment elicited a severe pain to the animal. In three animals thick bands of mesentery running dorso-ventrally in the abdominal cavity were palpated. Three animals were presented during gestation period of more than 5 months where the obstructed loop within the abdominal cavity could not be located by per rectal examination.

4.5.3 LABORATORY DATA

The preoperative laboratory data of the patients was obtained and has been divided into two groups namely the survivors (n=6) and non survivors (n=9). The values presented in the table are before undertaking surgery.

The haematological parameters revealed increased haematocrit with elevated total leukocytic count and total erythrocytic count towards higher range (Table 32).

TABLE 32: PREOPERATIVE HAEMATOLOGICAL OBSERVATIONS IN CLINICAL CASES SUFFERING FROM INTESTINAL OBSTRUCTION (MEAN \pm S.E.).

S. NO.	PARAMETER	SURVIVORS (N=6)	NON SURVIVORS (N=9)
1	Haemoglobin (7.5-12.5 g/dl)*	9.43 \pm 0.448	9.53 \pm 0.551
2	PCV (23-36%)*	42 \pm 1.789	44.78 \pm 2.235
3	TEC (5-8 x 10 ⁶ /ul)*	6.34 \pm 0.338	6.42 \pm 0.531
4	WBC count (4-20 x 10 ³ /ul)*	9.24 \pm 0.418	7.31 \pm 0.301
5	Neutrophils	41.17 \pm 2.088	39.89 \pm 1.968
6	Lymphocytes	56.83 \pm 1.922	59.11 \pm 1.806

*Normal references ranges, University of Florida Veterinary Teaching Hospital.

The blood biochemistry analysis revealed hypokalemia, hypochloreaemia and azotemia (only in severely dehydrated animals). The plasma phosphorus, calcium, bilirubin and total proteins were towards higher range (Table 33).

The plasma creatinine level was high and mild decrease in plasma sodium concentration was also appreciated. The plasma Alkaline phosphatase, Alanine amino transferase and Aspartate amino transferase were elevated to their maximum range with a very high increase in plasma amylase concentration (Table 33).

TABLE 33: PREOPERATIVE BIOCHEMICAL OBSERVATIONS IN CLINICAL CASES SUFFERING FROM INTESTINAL OBSTRUCTION (MEAN \pm S.E.).

S. NO.	PARAMETER	SURVIVORS (N=6)	NON SURVIVORS (N=9)
1	Glucose	64.3 \pm 4.24	63.88 \pm 12.534
2	BUN(10-22 mg/dl)*	30.05 \pm 1.733	39.04 \pm .899
3	Creatinine(0.5-1.5 mg/dl)*	3.72 \pm 0.141	4.76 \pm 0.344
4	Total Bilirubin(0.1-0.4 mg/dl)*	0.42 \pm 0.064	0.45 \pm 0.048
5	Plasma Total Proteins (6.5-8.0 g/dl)*	6.98 \pm 0.323	7.63 \pm 0.273
6	Sodium	133.33 \pm 1.43	134.44 \pm 1.937
7	Potassium(4-5.5 mEq/L)*	4.06 \pm 0.333	3.54 \pm 0.173
8	Chloride(99-110 mEq/L)*	90.32 \pm 3.031	78.85 \pm 2.62
9	Calcium	5.97 \pm 0.385	6.15 \pm 0.288
10	Phosphorus(4-7 mg/dl)*	5.92 \pm 0.254	7.11 \pm 0.285
11	ALKP(19-126 IU/L)*	127.8 \pm 7.731	126.67 \pm 9.833
12	SGOT (AST)	146 \pm 13.653	152.33 \pm 13.427
13	SGPT (ALT)	130.33 \pm 7.923	133.77 \pm 8.188
14	Amylase(4-38 IU/L)*	51.17 \pm 4.308	32.67 \pm 3.169

* Normal references ranges, University of Florida Veterinary Teaching Hospital.

4.5.4 DIAGNOSIS

The history of the course of illness comprising symptoms of colic and failure to defecate, reflex guarding of abdomen, crouching, rigidity of abdominal muscles with abdominal distention and postural abnormalities were indicative of acute abdomen. Auscultation and ballottement of abdominal wall was observed to be important tool for diagnosis of intestinal obstruction. The presence of fluid splashes along with resonant tinkling sounds during early phase of obstruction and relative quietness during later phase of obstruction aided in diagnosis.

Digital rectal examination proved to be the most informative diagnostic procedure in cases of intestinal obstruction. Almost 80 per cent of the cases suffering from intestinal obstruction involving jejunum, ileum, colon and caecum were easily diagnosed per rectally. The intestinal loops could not be palpated per rectally in the pregnant animals with gravid uterus of more than 5 months, thus restricting diagnostic options to only exploratory laparotomy and subsequent surgical correction. The presence of tympanitic loops of intestine, tight mesenteric bands and discrete pain on palpation of the obstructed loops helped in confirmation of intestinal obstruction.

The laboratory data indicating hypochloraemia, hypokalemia and azotemia were characteristic of bowel obstruction. The other findings like elevated plasma enzymes especially amylase, bilirubin and total proteins also followed the intestinal obstruction. Other laboratory tests indicated a very high increase in ruminal fluid chloride concentration with increased protein and cellular content of peritoneal fluid. The discolouration of the peritoneal fluid also pointed towards the intestinal pathology (Table 34).

TABLE 34: PREOPERATIVE RUMINAL AND PERITONEAL FLUID ANALYSIS IN CLINICAL CASES SUFFERING FROM INTESTINAL OBSTRUCTION (MEAN \pm S.E.).

S. NO.	PARAMETER	SURVIVORS (N=6)	NON SURVIVORS (N=9)
1	Ruminal fluid – Chloride (30mEq/L)*	66.1 \pm 8.036	73.68 \pm 5.119
2	Ruminal fluid – pH	6.48 \pm 0.210	6.05 \pm 0.13
3	Peritoneal fluid – Proteins (1-5 mg/dl)*	5.17 \pm 0.499	6 \pm 0.294
4	Peritoneal fluid - Cell count (1 - 20,000 cells/mm ³)*	3591.67 \pm 548.55	5266.7 \pm 289.88
5	Peritoneal fluid - pH	6.87 \pm 0.117	6.87 \pm 0.0954

* Normal references ranges, University of Florida Veterinary Teaching Hospital

4.5.5 TREATMENT

The existence of pre operative dehydration and hypochloraemia indicated the vital need for rapid parenteral fluid replacement therapy, started preferably before surgery or as soon as possible afterwards. The administration of Ringer's solution and additional potassium chloride by IV route was preferred in these patients to offset the impending and ongoing metabolic alkalosis.

The amount of intra-venous fluid administered in the patients was calculated as per the following formula

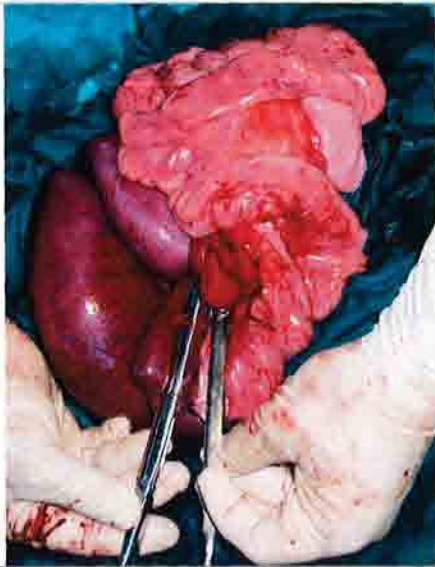
Fluid required to overcome dehydration (ml)

$$= \text{Patient PCV} \times 0.66 \times \text{body weight (in Kg)} \times 4 \quad (\text{Kumar, 1995})$$

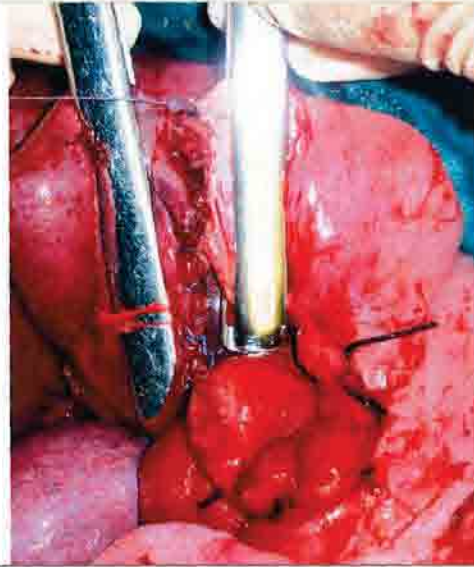
The pre operative administration of corticosteroids, NSAIDS and antibiotics helped to reduce the stress induced by surgery. The corticosteroids were not given in pregnant animals because of the possibilities of potential complications.

Right flank laparotomy was performed under linear infiltration analgesia in standing position. The per rectal findings were corroborated to locate the obstructed loop of intestine. Usually the omental covering was reflected forward to

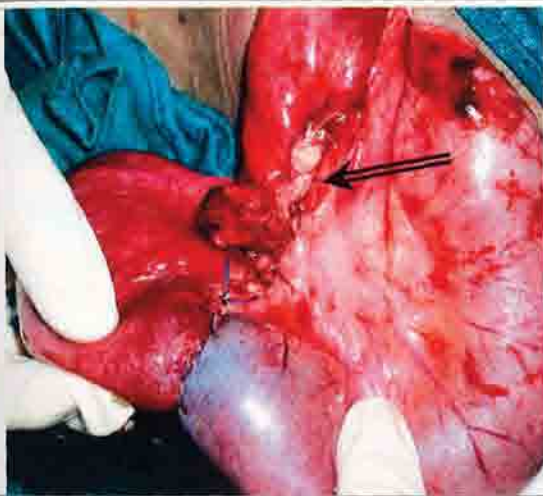
**PLATE 13: CORRECTIVE SURGERY FOR INTUSSUSCEPTION
COMPRISING OF ENTEROANASTOMOSIS IN CLINICAL CASES**



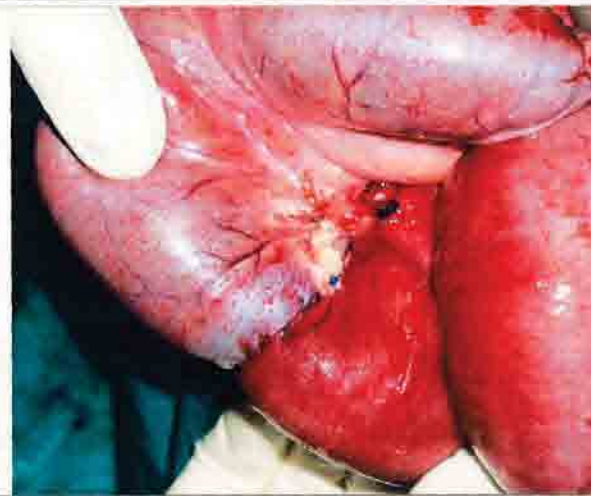
A



B



C



D



E

- A. Placement of Doyen's intestinal clamp for anastomosis
- B. First simple continuous layer to join the lumens
- C. Mesenteric rent joined by suturing
- D. Complete enteroanastomosis
- E. Copious washing of the setting with normal saline before reposing back into intestinal cavity

locate and exteriorize the affected segment but in certain cases there was considerable tightening of mesentery enforcing difficult or even impossible exposure of the affected loop, in these cases the omental layers were incised for easy exteriorization. The viable segments of intestine proximal and distal to the obstruction were identified and the affected/obstructed segment was removed by ligating the mesentery in between the two healthy segments. The mesentery selected for resection of the intestine was infiltrated with 2 per cent xylocaine. Following resection, intra operative decompression of the distended segment was performed and end to end anastomosis (Plate 13) was achieved using two layer anastomotic technique with a simple continuous pattern for the submucosal/mucosal layer, followed by a continuous Lambert pattern in the seromuscular layer using 2-0 Vicryl (Polyglactin 910). The laparotomy wound was closed in a standard routine manner.

The post operative care included administration of Ringer's solution, normal saline solution, 5 per cent dextrose normal saline, calcium borogluconate, antibiotics, Vitamin C, analgesics, corticosteroids, metronidazole (intraperitoneally) and Nuxvomica (PO). The animals were fed, rice gruel, soft hay and treacle after 6 hours of surgery followed by a handful of hay mixed with green supple grass after every 3-4 hour interval in post operative period. The full ration was asked to resume within 3-4 days.. The transfaunation of ruminal cud 12 hours after surgery in the present study was done in all the cases.

Specific therapy included administration of neostigmine in the patients those were unable to defecate within 12 hours of surgery. Additional potassium chloride was included in the treatment protocol where the animals resumed eating but were unable to defecate and developed ruminal tympany. The patients with

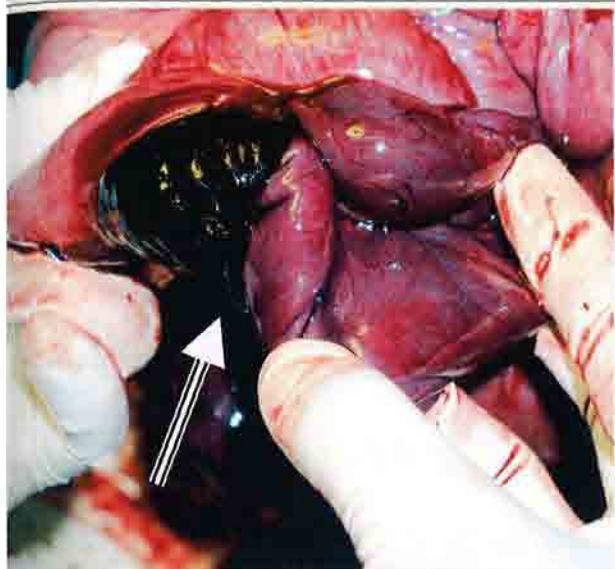
feeble or nil ruminal motility combined with sluggish/nil protozoal motility were given ruminotorics, biobloom and ruminal cud.

4.5.6 OPERATIVE FINDINGS AND OUTCOME OF CASES

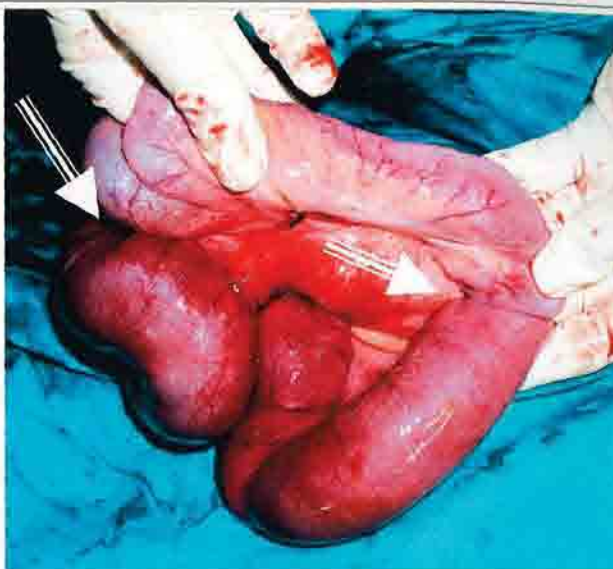
One of the 15 affected animals was bull and 3 out of 14 cows were pregnant with gestation period of 5 months or more. All the cases suffered from intussusception (Plate 14) at the following locations, Jejunojejunal (n=5), Jejunoileocaecocolic (n=1), Ileioacaecocolic (n=1), Caecocolic (n=7) and Colocolic (n=1). Three animals had intussusception with rupture of the intestine at the affected site. Surgical intervention was attempted in all the cases. Out of 15 cases 6 animals survived and 9 animals died. Non survivors also included 3 pregnant animals and 3 with intestinal rupture and peritonitis (Table 35).

In all the animals, failure to defecate was associated with telescoping of the alimentary tract (Plate 14) with severe oedema of the intestine and particularly of mesentery which was grossly infiltrated and distended with oedematous fluid and noticeably heavy and painful on palpation. In three cases loose adhesions of affected segment were found with the mesentery and peritoneal fluid contained free floating and grossly visible masses of fibrin. The segment of intestine distal to obstruction was collapsed and proximal segment was greatly distended with ischemic changes (Plate 15).

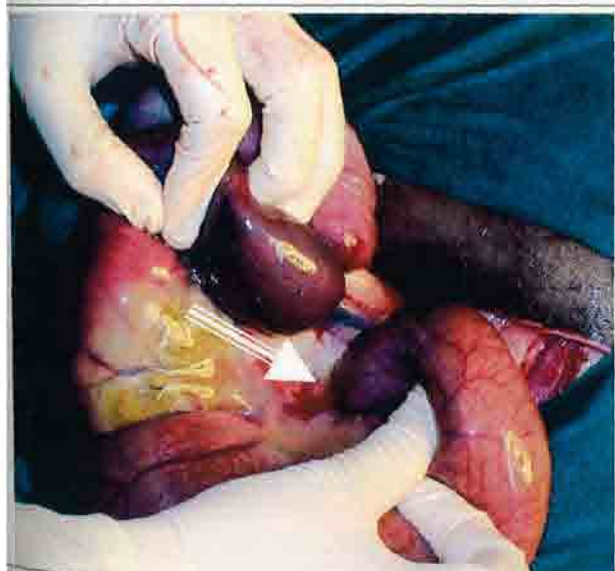
PLATE 14: INTRA-OPERATIVE FINDINGS IN CLINICAL CASES



A



B



C

A. Ileocecolic Intussusception
(Note the extent of mucosal damage)

B. Colocolic Intussusception
(Telescoping at two points)

C. Colocolic Intussusception
(Bowel wall necrosis upto mucosa)

→ (ARROW INDICATES SITE OF
TELESCOPING)

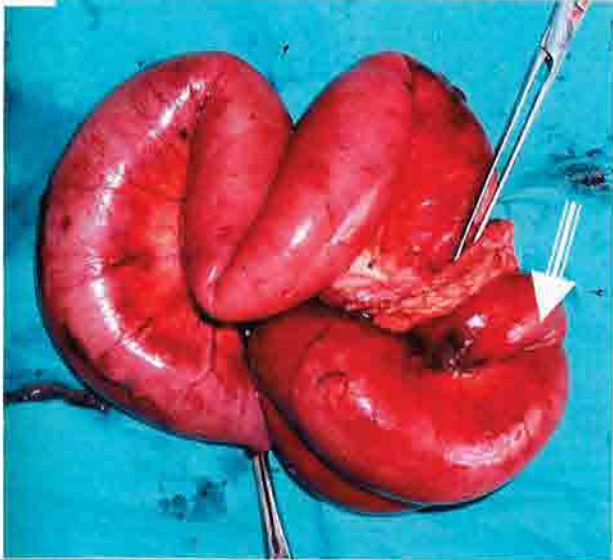
TABLE 35: SUMMARY OF CLINICAL CASES OF INTESTINAL OBSTRUCTION WITH RESPECT TO SIGNALMENT AND OUTCOME IN CATTLE.

S. No.	Species	Age (Years)	Sex	Day of Surgery*	Diagnosis	Outcome
1	Cow	4	F	6	Intussception Caeco colic junction	Died
2	Buffalo	7	F	6	Jejunum Intussception	Died
3	Cow	10	M	8	Jejunum Intussception	Died
4	Cow	7	F	2	Intussception Jejuno ileocaecocolic junction	Died
5	Cow	4	F	4	Intussception Caeco colic junction	Died
6	Cow	2	F	3	Intussception Caeco colic junction	Survived
7	Cow	5	F	3	Intussception Caeco colic junction	Survived
8	Cow	3	F	5	Intussception at ileocaecal junction	Survived
9	Cow	3	F	5	Intussception at caeco colic junction	Died
10	Cow	2	F	4	Intussception Jejunum	Died
11	Cow	7	F	6	Torsion and Intussception	Died
12	Cow	5½	F	5	Intussception of caecum into colon	Survived
13	Cow	6	F	5	Intussception Rupture at Caecocolic junction	Died
14	Cow	6	F	7	Intussception Colon	Survived
15	Cow	5	F	3	Intussception Jejunum	Survived

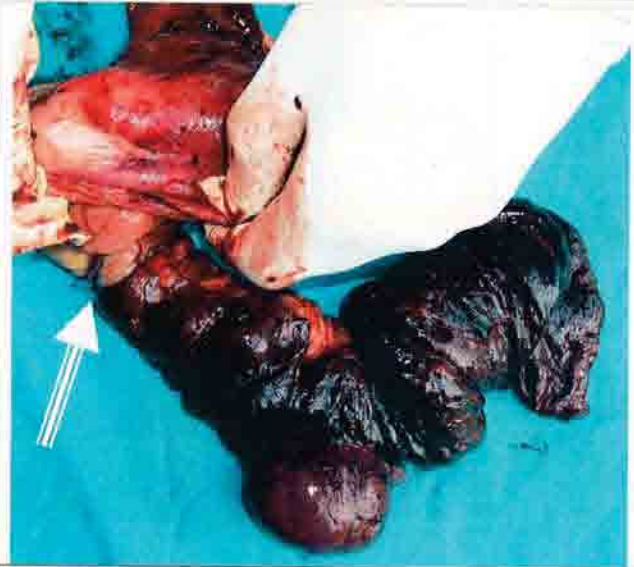
4.5.7 POST MORTEM FINDINGS

On autopsy of the non survivors, adhesions were noticed in the abdominal cavity and the affected segment of the bowel appeared dark red. The mesentery was thick and rubbery with ecchymotic areas. Serosanguinous peritoneal fluid was usually present in the abdominal cavity. Coprostatic obstruction within the distended loop of intestine was evident. The lymph nodes within the involved mesentery were swollen. There was no anastomotic leakage but the serosa of the

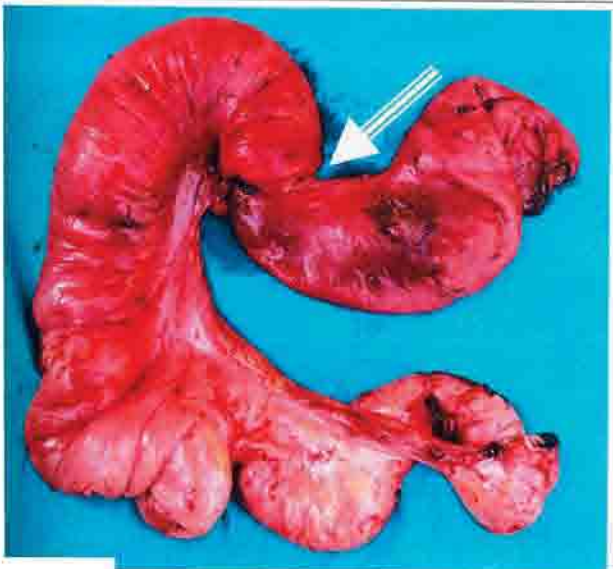
PLATE 15: RESECTED PARTS OF VARIOUS INTUSSUSCEPTION DURING CORRECTIVE SURGERY



A



B



C

A. Colocolic Intussusception
(Resected part of intestine)

B. Jejunojejunal Intussusception
(Intussusceptum dissected with
transmural injury)

C. Jejunoileal Intussusception
(Intussusceptum is engorged as compared
to collapsed part of intestine)

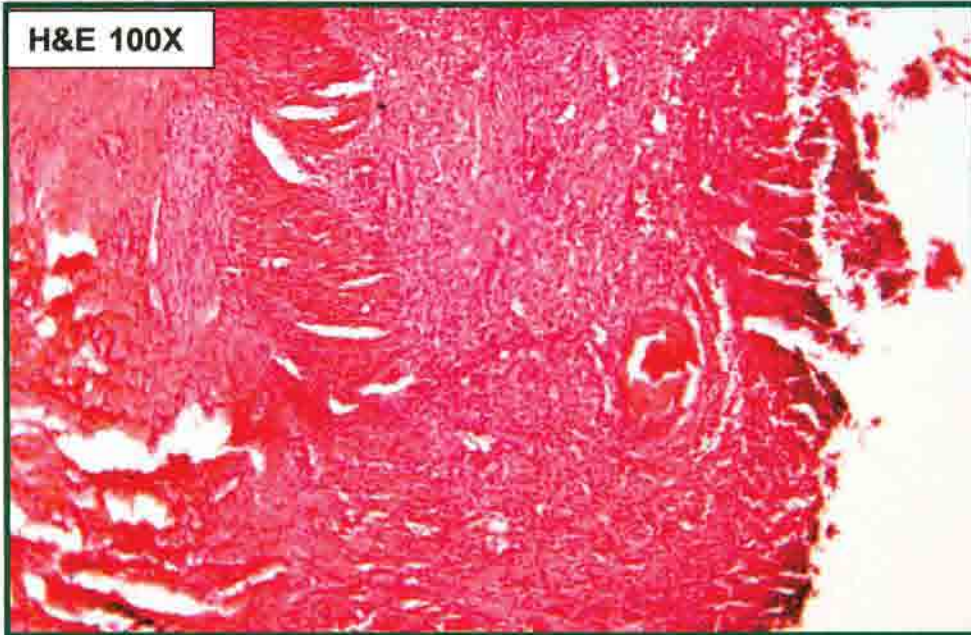
→ **ARROW INDICATES SITE OF
TELESCOPING**

Plate 16 : Photomicrograph of intestine affected with intestinal obstruction

SIMPLE JEJUNAL OBSTRUCTION

A

H&E 100X

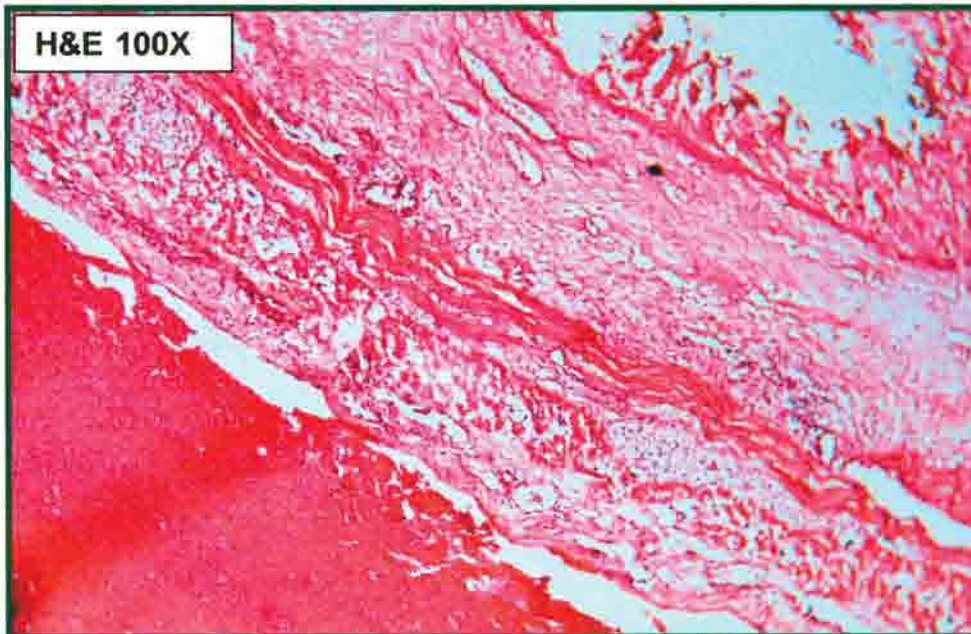


1. Sloughing of villi from mucosal surface
2. Increase in eosinophilic matrix in the submucosa

STRANGULATED JEJUNAL OBSTRUCTION

B

H&E 100X



1. Denudation of epithelial lining
2. Severe oedema in submucosa
3. Atrophic and necrotic changes in muscularis layer
4. Serosa heavily congested and oedematous

reconstructed intestinal segment was denuded and focal ulcers were seen on the mucosal surface. The mucosa of abomasum was edematous and it contained more of fluidy ingesta, whereas, omasum contained dehydrated feed particles.

CHAPTER - V

DISCUSSION

Intestinal obstruction is a familiar presentation to most of the practitioners and it belongs to highly severe conditions in gastroenterology, namely from the viewpoint of quick and correct diagnosis as well as determining rational and effective therapy. In these cases, the importance of identification of obstruction and timing of the intervention performance in regard to patient's survival is explicitly the principal and life saving concern. Obstruction can occur in any part of the intestinal tract but most often develops in the small intestine as a result of its narrower luminal diameter (Strombeck and Guilford, 1979). Obstructions are classified as either "simple", where the obstruction is not complicated initially by a vascular compromise of the bowel or "strangulated", characterized by interruption of vascular supply of the intestinal tract with simultaneous blockage of the intestinal lumen (White *et al.*, 1980). Anatomical intestinal obstruction (mechanical ileus) can result from intraluminal foreign objects, intramural masses or extramural compression due to abdominal adhesions or entrapment. Functional intestinal obstruction can result from transient hypomotility (paralytic or adynamic ileus) or from persistent intestinal neuromuscular derangements that cause hypomotility (pseudo obstruction) or spasticity (spastic or hyperdynamic ileus).

In adult cattle the passage of ingesta through the digestive tract takes one and half to four days (Pearson, 1974). Failure to defecate for 24 hours or more is therefore abnormal and the continued absence of faeces leads to the well recognized and usually fatal syndrome of bowel obstruction. The specific metabolic disturbances which occur during intestinal obstruction are to be corrected and the patency of the intestinal tract is to be restored to increase the

longevity of the animal. Therefore, the present study was aimed to create a suitable model of simple and strangulated intestinal obstruction in cow calves and to formulate an effective therapeutic regimen based on the clinico pathologic findings in different body fluids following simple and strangulated intestinal obstruction.

The pathogenesis of intestinal obstruction has been studied intensively in man and experimentally in simple stomached animal, especially the horses (Datt and Usenik, 1975; Hjortkjaer and Svendsen, 1979 and Lucke, 1970) and dog (Warner *et al.*, 1966 and Wetterfors, 1965). These experiments indicated that high obstructions of the small intestine cause more acute and serious symptoms than low obstructions of large intestine. In cattle there is experimental data, even though limited, concerning duodenum and ileum obstruction. Since it is difficult to assess the actual duration of onset of the closure of intestinal lumen in clinical cases on the basis of apparent clinical signs, therefore, it became necessary to create and standardize a suitable model of simple and strangulated intestinal obstruction in the present study.

Several experimental models have been designed to produce either simple or strangulated intestinal obstruction in bovines (Krishnamurthy *et al.*, 1980; Singh and Kohli, 1980; Parsania *et al.*, 1983; Papadopoulos *et al.*, 1985a; Avery *et al.*, 1986; Bhokre and Deshpande, 1988; Tank and Parsania, 1991 and Makhdoomi, 1994) and equines (Yale, 1969; Datt and Usenik, 1975; Singh *et al.*, 1975; Adams *et al.*, 1980; Sullins *et al.*, 1985; Freeman *et al.*, 1988; Ruggles *et al.*, 1993 and Falcrios *et al.*, 2002). An experimental model of closed loop strangulated intestinal obstruction at the level of proximal ileum in equines (Singh *et al.*, 1975) and bovines (Krishnamurthy *et al.*, 1980) has been evaluated to simulate the clinical

presentation of intestinal obstruction. Intestinal obstruction at the level of duodenum, jejunum or colon has been created by Papadopoulos *et al.* (1985a) in cattle by placing flexible plastic tubing around the bowel in a double loop fashion. In the present study the technique used by Papadopoulos *et al.* (1985a) has been modified as it allowed the extra omental fixation of the part of bowel which is normally covered by omentum in the abdominal cavity. In present study, the incision on the omentum for locating the cranial jejunum was sutured as such and instead extra small holes were created in the omentum to pass the silicon tube and thus making the bowel to stay back in the normal aquarium of peritoneal fluid with in the omental covering. This procedure benefited in less adhesion formation and the leakage of the peritoneal fluid was prevented from the holes communicating outside the skin.

5.1 PRE TREATMENT (SIMPLE AND STRANGULATED INTESTINAL OBSTRUCTION)

5.1.1 CLINICAL OBSERVATIONS

The abdominal pain manifested by the affected animals in the present study following both simple and strangulated intestinal obstruction has also been documented in bovines (Pearson, 1971; Papadopoulos *et al.*, 1985a; Radostits *et al.*, 1994 and Sharma, 1999) and camel (Anderson, 1996). The cause of pain in small intestinal obstruction could be related to ischemia, the increase in the intraluminal pressure and the resultant increase in bowel wall tension and increased pull of the viscera on the mesentery. The intensity of pain was probably proportional to the rapidity with which the intestinal tension developed and to the magnitude of the tension (Allen *et al.*, 1986). The magnitude of the increase in intraluminal hydrostatic pressure in intestine proximal to the obstruction appeared

to have a direct effect on prognosis for survival. Allen *et al.* (1986) observed that the horses which lived following surgery had a significantly lower intraluminal pressure (6.3 cm H₂O) as compared to horses that died (15 cm H₂O).

The underlying inflammation must be suspected when a colicky pain does not disappear between spasms, or becomes continuous. In the case of intestinal obstruction this might mean strangulation and urgent surgery is required. The intensity of pain was less severe in simple as compare to strangulated obstruction in the present study. The clinical signs of colic disappeared after 24 hours in strangulated and after 4 days in simple intestinal obstruction which could be due to passing of crucial point of intestinal distension and diminishing ability of the muscularis layer to respond to distension (Radostitis *et al.*, 1994). Signs of anterior abdominal pain may be elicitable with the "Skooch test" (Garry, 1990) i.e. bovine drops it back ventrally when the withers and back are pinched but animal with abdominal pain usually resists and refuses to drop the back.

The pattern of defecation characterized by initial scanty and passing of hard dung balls followed by passage of thick mucous in strangulated obstruction was similar to the findings of Pearson and Pinsent (1977), Braun *et al.* (1990), Anderson *et al.* (1993) and Doll *et al.* (1998). These signs showed the impact of the mechanical obstruction on the intestinal tract (Singh *et al.*, 1975), dehydration and loss of vascular and cellular integrity of the intestinal wall (Anderson *et al.*, 1993) due to venous occlusive ischemia and low flow state causing mucosal damage (Snyder, 1989). The difference between simple and strangulated obstruction in the present study was that crupous shreds were voided as early as 48 hours in strangulated obstruction as compared to 6th day in simple intestinal obstruction. The passing of mucous after the intestinal obstruction could be due to

empty intestine distal to obstruction where the normal peristalsis continued to occur (Singh *et al.*, 1997b).

Normal urination both in frequency and quantity, in the animals of group II and group III of simple and strangulated intestinal obstruction in the pretreatment period may be attributed to a shorter time between the induction of obstruction and institution of fluid therapy. The gradual decline in urine volume in the animals of control group may be due to the increased secretion of anti diuretic hormone and aldosterone through the rennin-angiotensin system and subsequent retention of water (Ganong, 1975; Papadopolous *et al.*, 1985a).

Progressive anorexia and normal water intake in the calves suffering from simple and strangulated obstruction in the present study could be due to intense colic initially followed by cessation of rumination and abdominal distension, reduced extracellular fluid and dry mucous membrane (Papadopoulos *et al.*, 1985a). In the present study the animals with simple obstruction continued to eat for a longer period of time upto 7 days and were not able to do so later because of generalized weakness.

The muscular frailty was severe in strangulated intestinal obstruction than simple intestinal obstruction as there was early recumbent posture following strangulated obstruction. This muscular weakness may be attributed to decreased concentration of potassium which interferes with the normal contractility of smooth, skeletal and cardiac muscles.

Bowel sounds alongwith other clinical signs such as scanty mucoid feces, ruminal atony and colic served as a criteria to undertake surgery in cases of obstruction in cattle (Smith *et al.*, 1982). Bowel sounds upto 48 hours following strangulated and 4th day after simple intestinal obstruction in the present study

were similar to those described by Smith (1990). The tinkling and clicking sounds could be due to pressure of gas under pressure in the dilated loop of intestine. The normal bowel sounds were low pitched and occur every few seconds. Their absence over 30 seconds in human beings suggests that peristalsis has ceased resulting into ileus. The absence of bowel sounds in later part of both types of obstruction could be due to atony of bowel smooth muscle tone as a result of prolonged period of obstruction. MacHarg *et al.* (1986) confirmed that horses with simple obstruction often revealed an increase in borborygmi early in disease process, followed by progressive decrease as a result of initial increase in electric activity in the oral segment followed by a progressive decrease later in the obstructive phase. It has been suggested by Miar and Edwards (2003) that auscultation was not a good indicator of small intestinal activity and sounds of colon and caecal motility may be present without any small intestinal activity in horses. However the sounds may be reduced temporarily in many cases of abdominal pain but a persisting absence of gut sounds is expected in strangulated obstructions. During simple obstruction, pain may be related to hearing of a progressive peristalsis whereas strangulation causes cessation of all motility (White and Randolph, 2003).

The bilateral abdominal distension observed in the animals of group II following simple and strangulated obstruction in the present study was similar to that described by Dzuik and Usenik (1964), Hammond *et al.* (1964) and Papadopoulos *et al.* (1985a). Unilateral distension in experimentally induced strangulated obstruction of ileum has been reported in cow calves (Sharma, 1999). The distension might be attributable to the accumulation of ingesta, gas from

bacterial fermentation, saliva, swallowed air, pancreatic and bile secretions and secretions from intestinal mucosa proximal to the obstruction (Allen *et al.*, 1986).

The cold extremities, lassitude exhibited by dullness and depression in the present study were similar to the findings observed in buffalo calves following jejunal obstruction (Singh and Kohli, 1980). The progression of deteriorating signs was of less intensity in simple and of severe intensity in strangulated obstruction. These signs often persisted due to fluid abnormalities and probably because of persistent absorption of toxins from the obstructed loop (Cohn, 1985).

Progressive decrease in rectal temperature following simple and strangulated obstruction might be due to the reduced metabolic activity and muscular weakness. Similar observations have been reported by Postal and Schoerb (1977), Pearson and Pinsent (1977), Basu *et al.* (1990), Braun *et al.* (1990) and Papadopoulos *et al.* (1985a). Excessive and prolonged secretion of catecholamines leads to lack of response from tissues and hence contribute to hypothermia (Willmore *et al.*, 1974). Negligible changes in rectal temperature following jejunal obstruction in buffalo calves have been observed (Makhdoomi, 1994). Occasionally fever concomitant with abdominal pain has been reported in horses (Steckel, 1992) which were related to regional or generalized peritonitis due to extreme muscle exertion associated with violent abdominal pain particularly in summer months.

Increased heart rate observed in all the animals following simple and strangulated jejunal obstruction in the present study might be attributable to the decrease in plasma volume and decreased activity of baroreceptors (Papadopoulos *et al.*, 1985a). Increased pulse rate was a consistent finding in all the animals of the present study and it might be due to intense visceral pain or

dehydration with accompanying shock (Singh, 1971; Smith, 1985a and Nath *et al.*, 1991). Steckel (1992) reported that horses exhibiting a heart rate of 60 beats per minute after systemic medication for pain were experiencing cardiovascular shock. In general, a heart rate of 80 beats per minute or greater was a definite indication of shock due to hypovolemia but individual horses manifesting heart rate of 100-120 beats/minute may have progressed to irreversible fatal shock regardless of the inciting cause or treatment administered.

The increase in respiration rate following simple and strangulated obstruction was in agreement with the other studies conducted in bovines (Murthy *et al.*, 1983; Bhokre and Deshpande, 1987 and Singh, 1987), donkeys (Singh *et al.*, 1975) and sheep (Singh *et al.*, 1983). However, these observations were not in agreement with the findings of Papadopoulos *et al.* (1985a), Steckel (1992) and White and Randolph (2003) ascribed the increase in respiration rate to abdominal distension, metabolic alkalosis and restriction of diaphragm. They further elucidated that elevated heart and respiratory rate can serve as indicators of abdominal pain, shock and toxemia.

Progressive and gradual increase in CRT was recorded in simple intestinal obstruction whereas abrupt increase occurred following strangulated obstruction. These observations were congruous with the observations made by Papadopoulos *et al.* (1985a) and Makhdoomi *et al.* (2002). This increase indicated compromised cardiovascular dynamics and low flow states due to peripheral vasoconstriction and decrease peripheral perfusion as has also been reported by Singh and Kohli (1980).

Cessation of ruminal motility at 24 hours of strangulated and 3rd day after simple jejunal obstruction could be due to visceral pain, dehydration, electrolyte

and acid base imbalance (Radostitis *et al.*, 1994). It has been explicated that splanchnic sensory nerves affects reticuloruminal motility by direct innervation and by nonhumoral effects of adrenal secretion. These nerves are not required for generation of normal contractions and the effect of splanchnic stimulation is inhibition of reticuloruminal motility. These nerves also innervate sensory receptors in other areas of gastrointestinal system, and some abnormalities such as intestinal distension produce reticuloruminal inhibition by means of reflex from splanchnic afferent activity (Kay, 1983). Reduced contractibility of the rumen and complete ruminal impaction has also been recorded by Pearson (1971) and Papadopoulos *et al.* (1985a) respectively.

The gradual decrease in the ruminal pH was recorded in both types of obstructions in the present study. The decrease in pH could be attributed to abomasal reflux into the reticulorumen resulting from abomasal disease, vagal indigestion or intestinal obstruction because of acidic nature of abomasal contents. The cause of low rumen pH has been best assessed by measurement of rumen chloride concentration (Garry, 1990) which was very high in the present study. The protozoal motility in the ruminal fluid showed a slight sensitivity to change in ruminal fluid pH as the large protozoa's were abolished at 24 hours of strangulated and 4th day after simple intestinal obstruction. In the present study the ruminal fluid pH has not fallen below 5, so sluggish motility of smaller protozoa was appreciable almost upto terminal part of study as has also been described by Garry (1990). At the same time the protozoal motility is not a prerequisite of normal digestive function but the importance from clinical viewpoint is their sensitivity to abnormalities in fluid milieu Garry (1990).

The classical signs of dehydration such as enophthalmia, increased skin tenting time and dry muzzle are reliable indices of body fluid depletion and were recorded in later stages of simple and strangulated obstruction in the present study. Similar clinical signs of dehydration following creation of simple intestinal obstruction in calves have been described (Hammond *et al.*, 1964; Sharma, 1999 and Sharma, 2000). It has been established that although the skin pliability is not a sensitive test for the state of dehydration (Steckel, 1992), but it provides an idea about the duration of the problem as skin tenting will be present in protracted cases when slow fluid loss due to reduced intake is a component of the disease process whereas, with fulminant diseases such as intestinal volvulus, skin turgor is usually normal (Steckel, 1992).

5.1.2 HAEMATOLOGICAL CHANGES

The haematological changes were more pronounced in strangulated than simple intestinal obstruction. The classical signs of shock were manifested in later stages following the simple intestinal obstruction. Increase in hemoglobin and packed cell volume in the present study might be due to haemoconcentration and hypovolemia as a result of dehydration and exudation of fluid in peritoneal cavity. The loss of fluid and electrolytes into the bowel lumen occurs due to dehydration and cardiovascular compromise which is a prominent feature of intestinal obstruction. In rats, it has been found that the volume of fluid that collected proximally to an experimental obstruction corresponded to 91 per cent of the plasma water volume (Larsson *et al.*, 1981) and distension of oral segment might be due to pancreatic, abdominal and biliary secretions which increases during obstruction (Shields, 1965). The plasma to lumen flow of fluid and electrolytes is the result of a passive filtration secretion mechanism and is less in the

obstructions where blood flow complications to the bowel wall were less as seen in simple intestinal obstruction (Swabb *et al.*, 1982). Ischemic occlusion in strangulated obstruction causes loss in bowel integrity which leads to exudation of fluid into the peritoneal cavity causing dehydration. At the same time disruption in mucosal barrier allows absorption of endotoxins and later enteric bacteria access to peritoneal cavity and then into systemic circulation leading to cardiovascular collapse.

The increase in total erythrocytic count in both types of obstructions could be due to decrease in the fluid component of blood subsequent to loss of fluid into peritoneal cavity and dehydration. These observations in the present study corroborate with the findings of Avery *et al.* (1986), Bhokre and Deshpande (1988), Basu *et al.* (1990) and Nath *et al.* (1991).

Neutrophilia and leucocytosis were seen in all the animals with simple and strangulated jejunal obstructions. These findings were in accordance with the findings of Smith (1985c) and Nath *et al.* (1991). In human beings it has been reported that when there is ischemic damage to mucosal barrier there is always increase in white blood cells thus inciting systemic inflammatory response syndrome (SIRS) which, if not counteracted can lead to multiple organ failure (Takayuki *et al.*, 2001). The increase in neutrophils might be due to neutrophil attractants such as leukotrienes, interleukins and activated complements that lead to accelerated production of neutrophils which eventually, migrate out of the capillaries and infiltrate the subepithelial mucosa by breaking the junctional complexes and inducing significant damage to barrier function. In addition neutrophils release reactive oxygen species and proteases that accentuate the damage to epithelium and microvasculature (Gayle *et al.*, 2000b).

5.1.3 BIOCHEMICAL CHANGES

The increase in total plasma proteins levels was observed following simple and strangulated intestinal obstruction and these observations were in agreement with the findings of Papadopoulos *et al.* (1985a), Braun *et al.* (1989a), Basu *et al.* (1990) and Nath *et al.* (1991) in bovine. However, Krishnamurthy *et al.* (1980) and Singh and Kohli (1980) reported no change in total plasma protein concentration following strangulated intestinal obstruction in buffalo calves. The increase in total protein concentration can be attributed to haemoconcentration and dehydration (Schalm *et al.*, 1975). Evaluation of the diagnostic triad of PCV, WBC count and total protein in animals with strangulated and simple intestinal obstruction is mandatory. Increase in PCV and total plasma proteins or haemoconcentration which developed slowly from 24 to 48 hours in simple obstructions was due to lack of water intake. In contrast, marked haemoconcentration developed within hours following strangulating obstruction which could be due to extracellular fluid shifts from the plasma water compartment into obstructed bowel. In conjunction to these the leucogram of cases of intestinal obstruction often reveals a stress response, with elevated or with in upper limits values.

The increase in blood glucose concentration was seen in both types of obstructions in the present study. These observations were similar to the findings of Parsania *et al.* (1983); Avery *et al.* (1986); Anderson *et al.* (1993) and Constable *et al.* (1997) but do not correlate with the findings of Krishnamurthy *et al.* (1980), Basu *et al.* (1990) and Nath *et al.* (1991). The increase however may be related to over activity of the cortisones or it may be secondary to increased hepatic glycogenolysis occurring in early stage of shock or sepsis. Hypoglycemia at later

stages can occur because of decreased gluconeogenesis and increased peripheral glucose utilization (Kuesis and Spier, 1999).

The majority of blood urea is synthesized in the liver from ammonia. Once formed, it diffuses freely throughout all body fluids. Increase in blood urea nitrogen has also been attributed to prerenal diseases like fever, infection, tissue necrosis including corticosteroid administration, decreased glomerular filtration rate and increased protein digestion. All these factors might have contributed to increased BUN in simple and strangulated intestinal obstruction in the present study. The findings encountered in bovines (Hammond *et al.*, 1964; Krishnamurthy *et al.*, 1980; Papadopoulos *et al.*, 1985b and Anderson *et al.*, 1993) and equines (Watts and Campbell, 1971 and Finco, 1980) were also akin. They attributed this increase to prerenal causes associated with hypochloremic alkalosis and starvation.

Simultaneous elevations of urea and creatinine on a biochemical profile denote azotemia. In the present study, the rise in creatinine concentration was more pronounced in strangulated than simple intestinal obstruction. This azotemia might be because of prerenal i.e. reduced glomerular filtration rate due to hypovolemia which usually is readily reversible on restoration of normovolemia. Clinical differentiation between the renal and prerenal azotemia is usually based on urine specific gravity since most patients with prerenal azotemia (intestinal obstruction) have highly concentrated urine while patients with renal azotemia have isothermic urine (Axiom, 2005).

The significant increase in total bilirubin was seen at later stages (6th day) in simple obstruction and at an early stage (48 hours) in strangulated obstruction in the present study. Anderson and Ewoldt (2005) advocated that bile acid concentration in cattle with proximal duodenal or jejunal obstruction was higher as

compared to animals affected with reticuloperitonitis, abomasal displacement or caecal dilation. Since bile duct is closely associated with the pancreas and empties into duodenum, various disorders causing swelling or constriction of the liver, pancreas or duodenum can also cause increased pressure within the biliary tree and thus cholestasis (Axiom, 2005). In addition to this the large animals are least efficient at excreting bilirubin in the urine. In horses most of the serum bilirubin is indirect and elevations has been reported to occur in anorexia, hepatic diseases, hemolytic anemia, endotoxemia and colic (Axiom, 2005).

The accumulation of fluid in the gut lumen is referred to as third space problem. Serum electrolytes vary depending upon the severity and duration of illness, fluid and acid- base disturbance, intake and renal function. The major site of fluid absorption is the proximal jejunum. The direction of water movement across the intestinal mucosa is dependent on osmotic gradients. Hypertonic solutions within lumen cause net secretion whereas a hypotonic solution leads to absorption. In case of luminal distension as a result of intestinal obstruction the fluid accumulated orad to obstruction is hypertonic thus restricting the absorption and promoting the net secretion into lumen.

Hyponatremia was seen in both types of intestinal obstruction in the present study but the severity was less in simple obstruction as has also been documented by Singh (1971). Sodium is a primary cation in extracellular fluid and its depletion may be attributed to the loss of ability of the obstructed intestine to absorb electrolytes and water (Shields, 1965). Similar findings have been recorded by Singh (1971), Parsania *et al.* (1983), Makhdoomi (1994) and Sharma (1999). Moreover due to ischemia subepithelial vacuoles created in the intestinal wall, the extrusion of fluid from the cell to these vacuoles is thought to occur to remove

excess cellular water and sodium accumulating intracellularly as the Na⁺, K⁺ pump becomes deenergized (Wagner *et al.*, 1979).

The abnormalities of the digestive tract which obtund the movement of gastric juices into the intestine for reabsorption produces metabolic alkalosis since hydrogen ions are continuously contributed to the extracellular fluid (Tasker, 1980). The intestinal secretions at the site of obstruction gradually lose capability to function normally when the intestine is distended by the accumulation of ingesta, gases and saliva. In the present study, hypochloraemia was observed following simple and strangulated jejunal obstruction. This was due to the impaired reabsorption of chloride ions from the strangulated segment of the intestine following their movement from abomasum to the intestinal tract, despite the constant secretion of chloride from the acid secreting cells of abomasum. Hypochloraemia associated with intestinal obstruction in bovines has been documented by Papadopoulos *et al.* (1985b), Smith (1985a), Sharma (2000) and Makhdoomi^{et al.} (2002). The chloride concentration in blood (<80mEq/L) is considered to have a poor prognosis for survival in cattle (Anderson and Ewoldt, 2005). Low concentrations of chloride have also been associated with gastrointestinal motility functions. Decreased gastrointestinal motility in humans and smooth muscles contraction in guinea pig (Rangachari *et al.*, 1982) have been associated with hypochloraemia and the tentative mechanism responsible for this is the requirement of chloride for release of calcium from sarcoplasmic reticulum or failure to provide the necessary osmotic forces that are needed for release of calcium.

Plasma potassium may influence intestinal motility (Adams, 1988). A highly significant decline in the plasma potassium concentration was observed in simple

and strangulated intestinal obstruction in the present study. Avery *et al.* (1986) attributed hypokalemia to hypophagia and intracellular shifts of potassium ions due to metabolic alkalosis. Whereas Hammond *et al.* (1964) correlated such hypokalemia due to shift of potassium from extracellular fluid to intracellular fluid or to gut or bone following intestinal obstruction in bovines. Hypokalemia observed in the present study might be due to reduced feed intake, shift of potassium to intracellular fluid and continuous loss through urine. The overall lassitude and muscular weakness as discussed earlier may also well be linked to the deficit of potassium in the blood to carry out the normal physiological functions like transmission of nerve impulses, maintenance of intracellular tonicity and muscular functions. Contrarily, it has also been established that even during long standing simple intestinal obstruction, if peritonitis develops and organic acids are released into blood stream, the serum biochemistry changes to metabolic acidosis with relative hyperkalemia (Anderson and Ewoldt, 2005).

Inconsistent changes were found in the concentrations of plasma phosphorous and calcium in both types of obstructions. The studies conducted by Parsania *et al.* (1983), Basu *et al.* (1990) and Braun *et al.* (1989a) found hypocalcaemia and hypophosphatemia associated with intestinal obstruction in bovines because of malabsorption from affected gut. Avery *et al.* (1986) and Makhdoomi ^{et al.} (2002) did not find any significant changes in the levels of phosphorous following duodenal obstruction in cattle. In contrast hyperphosphatemia was encountered in dogs (Shikata *et al.*, 1989), equines (Murray *et al.*, 1994) and cattle (Braun *et al.*, 1989) with strangulated obstruction.

Increase in liver enzymes is a frequent clinical problem of varying significance. In the present study, significant increase in the ALKP, AST and ALT

was recorded. Though the increase was significant but was on higher limit of the prescribed range. Krakovskii *et al.* (1990) in his studies on acute obstructive and strangulation ileus in rats found that the obstructive diseases are attended by marked diminution of hepatic detoxifying mechanism and suppression of hepatic microsomal enzymes p50 and b5. Intoxication by organisms and hypoxia developing in liver thus elevates liver specific enzymes. Horses with proximal enteritis and small intestinal strangulated obstruction were found to show higher serum AST and ALKP concentrations which Davis *et al.* (2003) attributed to hepatic injury due to ascending infection from the common bile duct and absorption of endotoxin or inflammatory mediators from the portal circulation, or hepatic hypoxia resulting from systemic inflammation and endotoxic shock. Damage to intestinal mucosa releases the 'ischemic ALKP' into circulation, which migrates faster than a known liver ALKP standard and exhibited elevated plasma levels (Williams and Wilson, 1981). Significant increase in the activity of aminotransferases, creatine kinase and alkaline phosphatase was found in the intestinal infarction (Kazmierczak *et al.*, 1988) and these changes were less pronounced in simple intestinal obstruction in animal models. Since AST is not liver specific and present in many extrahepatic tissues like muscles in domestic species so in the present study its elevation could have been due to the muscle injury during ischemia of bowel wall (Axiom, 2005). The rise in ALT can be due to altered membrane permeability, which is potentially reversible, or may lead to hepatocellular necrosis, which is essentially an irreversible change. The causes for rise in liver enzymes can be metabolic disturbances, inflammation and toxemia as a sequel of intestinal obstruction (Axiom, 2005).

Amylase is majorly a part of pancreatic secretion and very less amount is present in the saliva of the ruminants (Ruckebusch *et al.*, 1991a). The increase in amylase concentration was appreciated in the present study which could be attributed to luminal distension and altered permeability of the bowel wall resulting in leakage of accumulated amylase to the peritoneal cavity, which subsequently after absorption into blood stream produced a rise in the serum level and is thus a helpful diagnostic test in intestinal obstruction (Ellis *et al.*, 2002). Chiemprabha and Donelson (2005) reported that the human gut diseases like perforated bowel, mesenteric infarction, intestinal obstruction, appendicitis and peritonitis usually resulted in increased amylase because of increased absorption of amylase from intestinal lumen.

5.1.4 PERITONEAL FLUID ANALYSIS

Changes in the peritoneal fluid occur rapidly in response to inflammation involving the peritoneum or intestinal tissues. Fluid transudation occurs from lymphatics or venous obstruction or increased capillary permeability. The nature and composition of peritoneal fluid depends on the extent of vascular occlusion or the severity of inflammatory changes. Normal peritoneal fluid is odorless, straw colored, and may be slightly opaque (Oheme, 1969). In the present investigations the color, cytology, protein and electrolyte content of peritoneal fluid showed slow pace deterioration in simple than strangulated intestinal obstruction. The normal straw colour of peritoneal fluid changed to yellow, deep yellow with visible particles in the terminal part of study following creation of intestinal obstruction. Since normal and diseased bovine peritoneal fluid clots over a period of time (Payne, 1990) so it was collected in EDTA. The quantity of peritoneal fluid increased as the duration of obstruction progressed, suggestive of abdominal effusion. (Wilson *et*

al., 1985). The turbidity with particulate matter occurs due to the appearance of high cellular content, inflammation and presence of fibrin in the exudate. Discoloration commences early in the course of intestinal obstruction even while the segment of bowel is still viable (Swanwick and Wilkinson, 1976).

The pH of peritoneal fluid decreased slightly after creation of strangulated intestinal obstruction but no significant change was seen after simple intestinal obstruction which is in contrast with the findings of Van Hoogmoed *et al.* (1999). The total proteins and nucleated cell count have been reported to contribute good evidence to whether the peritoneal fluid is exudate or transudate (Coffman, 1973). In the present study, following simple and strangulated obstruction a marked elevation in total proteins and nucleated cell count was observed. The total proteins content in normal bovine peritoneal fluid can range from less than 1 mg/dl to greater than 5 mg/dl (Rosenberger, 1979). Elevated peritoneal fluid protein concentration is a sensitive indicator of early inflammation, while increased RBC counts in the presence of normal WBC counts suggests vascular damage without marked tissue ischemia (Hunt *et al.*, 1986). The increase in the WBC count usually indicates severe tissue inflammation or intestinal injury (Moore and White, 1982). The rise in these parameters might be because of sequence of events taken place during acute vascular injury to the intestine, reflected in the peritoneal fluid. First, the peritoneal fluid protein concentration increased, followed by increase in RBC count and fibrinogen (Moore and White, 1982). A transudative process (Morris and Johnston, 1986) resulting from vascular congestion and increased endothelial permeability allows small molecules (albumin) to escape into peritoneal fluid, followed by larger molecules (globulin and fibrinogen) and finally diapedesis of cells (RBC's then WBC's). As the degree of irreversible damage to the intestine

increases, the peritoneal fluid characteristics become more exudative (Hunt *et al.*, 1986). The normal WBC count in bovine peritoneal fluid can range from 1 - 20,000 cells/cu mm (Wilson *et al.*, 1985). Similar observations have been documented by Smith (1985a), Anderson *et al.* (1994), Sharma (1999) and Sharma (2000) in bovine intestinal obstruction.

The decrease in peritoneal fluid sodium, potassium and chloride was recorded in the present study. Since peritoneal fluid is an ultrafiltrate of plasma, so the changes exhibited in the plasma electrolyte concentration were reproduced in the peritoneal fluid.

5.1.5 RUMEN FLUID STUDIES

The epithelium of the forestomachs absorbs inorganic ions (e.g., sodium, chloride, ammonia) with water from the digesta in the rumino-reticular fermentation vat. Sodium and chloride ions are absorbed from the rumino-reticulum against a concentration gradient. The transfer of sodium across the rumino-reticulum wall, occurring against its electrochemical gradient, is stimulated when potassium intake is increased. Chloride is absorbed passively in the rumino-reticulum but is secreted in the omasum (Ruckebusch, 1991b). The decreased sodium concentration of ruminal fluid in the present study indicated an attempt by homeostatic mechanism to conserve it. Similar observations were made by Papadopoulos *et al.* (1985a) and Sharma (2000) in cow calves and Makhdoomi *et al.* (2002) in buffalo calves. The decrease in potassium concentration in the present study indicated absorption of this ion from rumen against concentration gradient in an attempt to moderate the hypokalemia. Normal chloride concentration in the ruminal fluid is 30 mEq/L. The increased value of chloride concentration demonstrates reflux of abomasal ingesta as seen in the present study (Above 40

mEq/L). In the clinical evaluation of forestomach dysfunction, elevated rumen chloride level suggests secondary indigestion caused by abomasal reflux or obstruction of intestinal flow (Garry, 1990). The continuous secretion of chloride ion as hydrochloric acid from abomasal mucosa, inflow of saliva in to the rumen complicated by impaired reabsorption and retrogression of gastrointestinal contents, contribute to accumulation of this chloride rich fluid in a compartment anterior to obstruction. Therefore, the chloride ion concentration tended to increase in ruminal fluid (Papadopoulos *et al.*, 1985b). Similar increasing trend in ruminal fluid chloride concentration has also been reported in intestinal obstruction by Avery *et al.* (1986) and Makdoomi *et al.* (2002).

In the present study the phosphorous concentration in ruminal fluid remained elevated in simple and strangulated intestinal obstruction. It is well known that main route of phosphorus excretion in ruminants is through saliva into the gut (Smith, 1985b), since the rumino-reticulum wall does not favour transfer of phosphate and it is mainly absorbed by small intestine both by active and passive processes. Therefore in the present study, its concentration might have increased due to disruption in absorptive surface of small intestine and retrogression of intestinal fluids back into forestomachs. Inconsistent changes were found in ruminal calcium concentration in the present study. Contrary to this, the decrease in ruminal fluid calcium and phosphorous concentration has been reported (Avery *et al.*, 1986 and Makhdoomi, 1994) following strangulated intestinal obstruction in bovine calves. As per studies of Avery *et al.* (1986) intestinal obstruction did not affect ruminal fluid minerals much.

5.1.6 MICROBIOLOGICAL STUDIES

Translocation of bacteria in diminutive amounts is a physiologically important phenomenon to boost the reticuloendothelial system (RES), especially the Kupffer cells in the liver. Breakdown of both the mucosal barrier and the RES capacity results in systemic endotoxemia which, produces organ dysfunction, impairs the mucosal barrier, the clotting system, the immune system and depresses Kupffer cell function. Furthermore multiple organ failure is more probably triggered by the combination of tissue damage and systemic endotoxemia (Van Leeuwen *et al.*, 1994). In the present study, existence of Gram +ve bacilli, Gram -ve rods and Gram -ve cocobacilli in the peritoneal fluid is in agreement with the studies of Cohn and Atik (1961) in dogs, Singh and Kohli (1980) in bovines and Parvathamma (1992) in goats.

Contamination of peritoneal fluid and dissemination of microorganisms can occur from combination of factors such as ingestion of food, retrograde spread from the bowel, lymphatics and/or hematogenous spread (Gupta *et al.*, 1980). Whereas, the contamination of peritoneal fluid has also been attributed to obstruction induced intestinal injury and disruption of ecology of normal gut flora (Deitch *et al.* (1990). Administration of large amount of fluids to animals with strangulated obstruction normalizes the arterial pressure and improves the intestinal blood flow thus minimizing damage to the intestinal mucosa and subsequent bacterial translocation (Fevang *et al.*, 2004).

5.1.7 ELECTROCARDIOGRAPHIC STUDIES

The electrocardiographic changes like shallow T wave and ST segment depression following intestinal obstruction in the present study were indicative of

electrolyte disturbance because of generation of third space due to intestinal distension. These changes were consequent to hypokalemia seen both in simple and strangulated intestinal obstruction in the present study. The other changes recorded in cows affected with gastrointestinal problem are atrial fibrillation (McGurik *et al.*, 1983) and atrial premature complexes (Constable *et al.*, 1990a) associated with metabolic alkalosis, hypocalcemia and hypokalemia. Atrial flutter which is almost same as atrial fibrillation except for the oscillations which are bigger than those in atrial fibrillation has also been reported following intestinal obstruction in bovines (Sobti, 1996).

5.1.8 HISTOPATHOLOGICAL FINDINGS

The gross and histopathological changes both in simple and strangulated intestinal obstruction in the present study were comparable on autopsy but the severity of changes were rapid and tremendous in strangulated obstruction. Generally, autolytic changes were found in strangulated obstruction and necrotic changes were evident in simple intestinal obstruction. The severity in strangulated obstruction can be attributed to occlusive ischemia of intestine where the intestinal flow was directly disrupted and resulted in insufficient delivery of oxygen and nutrients necessary for maintenance of cell integrity (Snyder, 1989). Like wise, in simple intestinal obstruction the intestinal edema and luminal distension were common findings, which aggravated intestinal damage (Snyder, 1989). The epithelial shedding might have resulted because of the appearance of membrane enclosed cytoplasmic blebs which arise at the cell base of the enterocytes and detach the epithelium from the basement membrane and this process begins at

the tip of villi and extends to the base making the changes irreversible (Wagner *et al.*, 1979).

5.1.9 TOTAL SURVIVAL TIME

The total survival period in strangulated jejunal obstruction (78.0 ± 9.49 hours) was less than simple jejunal obstruction (7.99 ± 0.793 days). The severity of strangulated obstruction was more pronounced due to vascular occlusion and low flow states. These changes were similar to simple intestinal obstruction in long standing cases (Snyder, 1989). In the small intestinal villi, a countercurrent exchange mechanism exists due to hairpin like vascular arrangement. During low flow states, oxygen and other nutrients may short circuit the villus tip, leaving the tip relatively anoxic compared with the villus base. During venous occlusion, this mechanism would explain the initial and most severe changes seen at the villus tip. The mucosal damage accompanies fluid and electrolyte disturbances causing clinical signs of shock. Transmural necrosis occurs, allowing absorption of endotoxin and later enteric bacteria access to peritoneal cavity (Moore *et al.*, 1981). Endotoxins cause further deterioration in the hemodynamic, hematologic and blood biochemical systems including hypotension, disseminated intravascular coagulation and disruption of glucose metabolism (Ewert *et al.*, 1985). In humans, endotoxemia after bowel necrosis is known as systemic inflammatory response syndrome (SIRS) which during critical illness produces multiple organ failure (MOF), and results in death of an individual (Davis and Hagen, 1997).

Simple intestinal obstruction alters the absorption of normal fluid and solute thus, contributing to luminal distension and clinical dehydration. Abnormal fluid dynamics with net fluid and electrolyte loss into the lumen may contribute to

disturbances of motility and further propagate the obstructive process. In simple obstruction, septic and endotoxic shock is unusual and does not occur until late when necrosis of the intestinal segment occurs. The depletion of plasma volume and reduction in cardiac output occurs, causing clinical signs of dehydration, elevation of the heart rate and poor peripheral perfusion (Billig and Jordan, 1969). These changes generally occur late in animals with simple obstruction and may be due to systemic changes related to pain rather than to the haemodynamic of fluid loss.

The administration of fluid therapy alongwith the antibiotics in the animals of group II produced only marginal difference in the survival time (14 hours in strangulated and one and half day in simple obstruction) as compared to control group I. Adequate fluid therapy during initial stages of intestinal obstruction is mandatory as it improves the intestinal blood flow and slows down the pathophysiological alterations as described earlier. The most important beneficial effect of adequate fluid therapy is preservation of mucosal barrier for longer period of time enabling success of corrective surgery (Fevang *et al.*, 2004).

5.2 POST TREATMENT (SIMPLE AND STRANGULATED INTESTINAL OBSTRUCTION)

The formulation and institution of conservative treatment in the animals of group II was based on the pathophysiological alterations recorded in the animals of group I. In the present study, metabolic alkalosis accompanied by hypochloraemia and hypokalemia were observed in both types of obstruction.

The time for surgical intervention in the present study was 3rd day following simple and 24 hours following strangulated intestinal obstruction and was decided

on the basis of decrease of chloride concentration in plasma and specifically when it decreased below 75 mEq/L, the decrease in plasma potassium concentration and specifically when it decreased below 3.5 mEq/L, degree of dehydration, inelasticity of skin and increased haematocrit value more than 40 per cent and muscular weakness.

5.2.1 CLINICAL STUDIES

In the present study, complete cessation of dung in the animals of group II (Simple and strangulated obstruction) was quite obvious due to occlusion of the intestinal lumen. Passage of scant, dry mucoid malodorous feces with fibrinous/crupous shreds coincides with findings of Smith (1985b) and as such indicated damage to bowel wall. Voiding of feces 5-6 hours (Strangulated) and 4-5 hours (Simple) in the animals of group III after corrective surgery confirmed the restoration of normal passage of intestine after surgery. The presence of mucous in the loose faeces initially in the post treatment period signifies the onset of reparative process and coincides with the findings of Smith (1985b). Two layered anastomosis was preferred for anastomosing the jejunum in the present study as it has been reported that collagen synthetic activity is relatively less in small intestine and collagen is single most molecule which determines the intestinal strength (Martens and Hendriks, 1992).

The urine output remained normal in the animals of group II and III irrespective of obstruction during the entire post treatment period which could be due to the effect of hydration achieved by IV fluid therapy. Similar findings have been reported by Sharma (1999) in bovines after treatment of experimentally induced ileal obstruction.

Hypophagia persisted in all the animals of group II (Simple and strangulated intestinal obstruction) even after institution of conservative therapy which might be attributed to the non correction of pathology of intestinal obstruction. The return of appetite to normal was seen at 72 hours in strangulated and at 5th day in simple obstruction in the animals of group III in present study and this has been reported to be an excellent prognostic indication of return of the normal digestive function and metabolic state (Constable *et al.*, 1991). The animals of group III were offered meal comprising of rice gruel, treacle and soft hay 6 hours after surgical correction of simple and strangulated intestinal obstruction to prevent the development of acute intestinal failure after abdominal surgery due to complications of withholding the oral diet (Klein *et al.*, 1997). Moreover, prolonged intravenous administration of 5 per cent dextrose and saline also produces intestinal failure due to ileus and deterioration of critical physiological functions such as immunity, wound healing and respiratory muscle strength (Sandstrom *et al.*, 1993). In humans, small intestinal motility recovers 6-8 hours after surgical trauma and moderate absorptive capacity exists even in the absence of normal peristalsis (Woods *et al.*, 1978). It has been shown that feeding in patients undergoing gastrointestinal resection is safe and well tolerated even when started within 12 hours of surgery (Reissman *et al.*, 1995). After doing corrective surgery on intussusception in bovines the food should not be withheld to avoid postoperative ileus (Anderson and Ewoldt, 2005).

The severity of muscular weakness in comparison to control diseased groups was less in the conservative groups of either type of obstruction which could be due to the correction of electrolyte and fluid imbalances in the post treatment period. The surgically corrected groups were also provided the fluid

therapy and showed normal appetite too, thus precluding the likelihood of hypokalemia which resulted in muscular weakness. Similar improvement in these signs following oral administration of potassium chloride has been observed by Hammond *et al.* (1964).

The rumbling noises of high pitch were observed in the group II in either type of obstruction. This might be due to the distension of the segment oral to obstruction and movement of fluid within the lumen. Similar observations were also made by Pearson and Pinsent (1977) suggesting that auscultation is an important diagnostic aid in intestinal disorders. In the later stages i.e. 72 hours after strangulation and 6th day onward after simple intestinal obstruction the abdomen was quiescent indicating ileus in the animals of group II. In surgically treated groups peristaltic rushes were heard in the entire post treatment period which qualifies good clinical progress and adequate therapy (Braun *et al.*, 1989a). The progressive bilateral enlargement of abdomen in the animals of group II in either type of obstruction could be due to the non correction of underlying pathology and perpetual accumulation of ingesta proximal to obstruction. The continuous abomasal secretion without significant absorption causes a reflux of gastric fluid into the forestomachs, particularly the ventral sac. This leads to pushing of distended intestine dorsad and the ventral sac of rumen encroaches upon the right side of the abdomen (Smith, 1985b). Whereas, in group III of either obstruction the gradual decrease in size of abdomen was obvious as soon as the luminal passage was made patent.

The rectal temperature showed a progressive decrease in the animals treated with medical therapy in both type of obstruction in the present study despite institution of adequate fluid therapy. This may be due to ensuing bowel

necrosis, electrolyte disturbance and circulatory insufficiency reflecting on the metabolic rate of the animal. Such signs have been observed by Braun *et al.* (1989b) following dilatation and torsion of caecum in cattle due to circulatory insufficiency. The restoration of normothermia, warm extremities and marked improvement in temperament of surgically treated groups in the entire post treatment period were mainly due to restitution of circulatory efficiency, absorptive function of intestine and administration of adequate fluid therapy to assist the metabolic functions of the body.

An increasing though statistically non significant, trend of increased heart and pulse rate persisted through out the post treatment period upto 96 hours in the animals of group II (Strangulated obstruction) whereas the heart and pulse rate were found elevated only at 6th and 8th day in the animals of group II (Simple obstruction). These results indicated the severity of type of obstruction and unremitting pathophysiological alterations despite fluid therapy in conservatively treated animals. In Simple obstruction, rise in the heart and pulse rate at late stages indicated bowel necrosis due to prolonged obstruction resulting in inflammatory response. Whereas significant decrease in the heart and pulse rates was found after surgery in both type of obstruction in the present study. This decrease could be due to the intensive chloride therapy, since the plasma chloride level is the most sensitive prognostic aid in cattle with bowel obstruction and low levels of chloride are usually associated with a corresponding increase in pulse rate (Pearson and Pinsent, 1977).

An increasing trend in the respiration rate was recorded in the calves treated with conservative therapy in either type of obstruction. The medical treatment though makes the animal haemodynamically stable yet underlying

pathology progresses insidiously. The weakness in respiratory muscles and air hunger might have resulted into compensatory increase in the respiration. Similar observations have been recorded in acute functional stenosis of pylorus in cattle (Braun *et al.*, 1990). A progressively significant decrease in respiration rate was seen in surgically treated animals as compared to other two groups indicating a signified response to therapy.

Initially the skin tenting time and eyeball attained normal state following treatment in the animals of group II and III in both types of obstructions, indicating rehydration. In the animals of group II (Simple and strangulated), the increase in skin tenting time, muffling of hair coat and dry muzzle were seen in the terminal which probably could be due to failure of haemodynamic system to maintain hydration in the body and dietary deficiencies resulting from abnormal fermentation and malabsorption. Pale conjunctiva in the later part of study in the animals of group II could be due to the hemodilution and progressive anemia (Kelly, 1984). The submandibular oedema in one of the animals of group II might be attributed to the impending shock and leakage of fluid in the extracellular spaces. The animals of group III (Simple and strangulated) showed fairly good hydration status because of correction of surgical affection and administration of fluid therapy.

Comparative CRT of group I, II and III (Simple and strangulated) inferred that the hypotension and decreased peripheral circulation was intense in the diseased control group followed by the conservatively treated groups. The satisfactory tissue perfusion in the animals of group III might be as a result of adequate intravenous therapy along with correction of obstruction and restoration of normal appetite.

The rumen motility either remained completely ceased or feeble in the animals given conservative treatment (Simple and strangulated). The very feeble ruminal motility could be due to rumenotorics instituted in the treatment protocol but overtly, the progressive accumulation of ingesta anterior to the site of obstruction and increased ruminal distension along with high chloride concentration in the rumen might have contributed to cessation of rumen motility despite administration of fluid therapy. After surgical correction in the animals of group III (Simple and strangulated) the rumen motility showed a plodding return within 24. Largely, rumen though showed motility yet remained hypotonic for longer period of time therefore transfaunation was invariably done after 12 hours of surgery in all the animals as also recommended by Anderson and Ewoldt (2005).

The ruminal pH did not show any significant alteration. It decreased slightly but remained on the basic side. Ruminal microflora suffered gradual loss in the animals of group II in both type of obstructions which could be ascribed to stagnation of ruminal fluid for a longer period of time resulting in accumulation of acids and chloride. Surgery increased livability in the animals of group III due to passage of stagnated ingesta and secretions, and also due to transfaunation and administration of biobloom along with fluids.

5.2.2 HAEMATOLOGICAL STUDIES

The hydration status in all the animals of group II of simple and strangulated obstruction remained good during initial stages as reflected by the marginally affected PCV values. However, at 72 hours of strangulated obstruction and 8th day of simple obstruction the PCV increased significantly indicating deterioration of haemodynamic status of the body leading to shock. Similarly, concurrent

haemoconcentration increased hemoglobin concentration at similar stages of obstruction. Whereas, in group III (Simple and strangulated obstruction) after surgery, these parameters showed appreciable recovery towards normal as compared to other two groups.

The increase in TEC remained gradual in the animals of group II (Simple and strangulated obstruction) as compared to their respective diseased control group I whereas substantial decrease in TEC was seen in surgically treated group indicating improvement in hydration status following surgery and supportive therapy. The significant increase of TEC in the terminal part of observations in the animals of group II may be explained on the basis of losses due to vascular insult occurred because of obstruction. These observations are in agreement with the findings of Smith (1985a), Bhokre and Deshpande (1988) and Sharma (1999).

The increase in the count of leucocytes in blood was observed in the animals of group II in both type of obstruction. These increased levels of TLC might be due to the administration of corticosteroids in the post treatment period. Besides this, neutrophilia was a major finding and the initiation of inflammatory process as evidenced by leucocytosis and neutrophilia can perhaps be due to the systemic inflammatory response syndrome (Takayuki *et al.*, 2001) which indicates a warning symptom of bowel necrosis. The decrease in TLC occurred in the animals of group III (Simple and strangulated) following surgery, but it remained higher than the base values which could be ascribed to repeated surgical intervention for correction of lesion in the animals of group III and use of corticosteroid therapy (Benjamin, 1985). Leucocytosis following corticosteroid therapy has been reported during last stage of endotoxic shock in ponies (Frauenfelder *et al.*, 1982).

5.2.3 BIOCHEMICAL STUDIES

The total plasma protein concentration remained elevated in the animals of group II (Simple and strangulated) despite medical therapy but the increase was not drastic as recorded in the animals of group I. In the present study the plasma protein concentration decreased significantly in the animals of group III when compared to the value on the day of surgical intervention (24 hours; strangulated and 3rd day; simple obstruction). This decrease in plasma protein concentration could be due to correction of abnormality supported by rehydration, analgesics and antibiotics resulting in improved ability of the body to utilize them for assimilation (Nath *et al.*, 1991) and reduced cellular lysis. Similarly Blikslager *et al.* (1994) found that institution of fluid therapy in horses helps in maintaining the total plasma protein concentration.

In strangulated jejunal obstruction an increasing trend was noticed in plasma glucose concentration after the treatment of animals of group II, whereas the blood glucose concentration increased significantly in the post treatment period when compared to the 24 hours post obstruction value in the animals of group III. The elevated levels of blood glucose could be due to the infusion of 5 per cent dextrose. Moreover, use of corticosteroids also mobilizes hepatic glycogen and increases lactate utilization as glucose precursor (Morill and Spitzer, 1978). Contrarily, in simple jejunal obstruction, a decrease in the glucose concentration was recorded in group II which might be due to longer disease process with incomplete loss of appetite and decreased gluconeogenesis and increased peripheral glucose utilization (Kuesis and Spier, 1999). However, in the animals of group III glucose concentration though remained high yet the increase was much less as compared to group II.

The azotemia observed in the present study might be consequent to dehydration, increased protein digestion resulting from intestinal bleeding, hypochloremic alkalosis and starvation (Watts and Cambell, 1971). This azotemia was counteracted by adequate fluid therapy in the animals treated conservatively (Group II) in either type of obstruction. The decrease in BUN and creatinine in the surgically treated groups III (Simple and strangulated) indicated that the treatment has lead to improvement in renal perfusion and glomerular filtration rate. Similar restoration of blood urea nitrogen to normal following fluid therapy and conventional treatment has been reported in uremic calves (Kulkarni *et al.*, 1985).

The increased bilirubin concentration following intestinal obstruction remained high regardless of fluid therapy provided to the conservatively treated groups II (Simple and strangulated). Similarly, in surgically treated groups considerable time (8th day; simple and 96 hours; strangulated obstruction) requisite was found to be obligatory for normalization of bilirubin concentration.

Since intestinal obstruction was marked by hypochloremic and hypokalemic metabolic alkalosis in the present study, the adequate fluid therapy to the treated animals furnished the deficit arising out of the lesion. In the animals of group II of both type of obstructions, the gravity of loss of these electrolytes was moderated but as the duration of obstruction advanced the deficit turned obvious. Whereas, in the animals of group III (Simple and strangulated), the removal of devitalized organ and restoration of luminal passage, resulted in rejuvenation of intestinal mucosa which helped in counteracting the electrolyte disturbance along with fluid therapy. Similar amendments in the concentration of these ions were also observed following the correction of abomasal displacement in cattle (Singh *et al.*, 1986).

The plasma enzymes like ALKP, AST, ALT and amylase showed progressive rise in the animals of group II of either type of obstruction with a difference that the pace of increasing concentration was much higher in the diseased control group I, indicating that the medical therapy tried to preserve and retard the destructive changes occurring in the hepatic metabolism, intestinal mucosa, pancreas and muscles making it reversible (Fevang *et al.*, 2002). The plasma concentration of these enzymes decreased significantly after surgical correction (Group III of simple and strangulated) of obstruction supported by adequate fluid therapy, liver tonics etc. Similar observations were recorded by Kausz and Nemesanszky (1976) after relief of intestinal obstruction in humans.

5.2.4 PERITONEAL FLUID STUDIES

The pathophysiological state of the parietal, visceral and mesothelial surface is reflected upon the peritoneal fluid (Hanson *et al.*, 1992). The discolouration of the peritoneal fluid assuming deep yellow colour during obstruction showed some improvement in color from 72 hours (strangulated obstruction) and 8th day (simple obstruction) following treatment. The amount of peritoneal fluid increased in the conservatively treated animals (group II) but deviation from normal straw colour persisted till the death supervened. Presence of hemolyzed blood in peritoneal fluid was observed by Coffman (1973). Progressive change in colour of peritoneal fluid assuming its normal straw colour after treatment in the animals of group III (Simple and strangulated) indicated the onset of reparative process. The peritoneal fluid pH showed no change in post treatment period in animals of group II and group III of either type of obstruction. These findings are in contrast to the findings of Van Hoogmoed *et al.* (1999) who

observed a lower pH than normal value of peritoneal fluid in equines following septic peritonitis.

Elevated peritoneal total proteins and nucleated cell count were seen throughout the period of study in the calves of group II of simple and strangulated obstruction. This could be attributed to the presence of obstructed bowel segment in the animals of group II. The total proteins though showed decreasing trend in the animals of group III, but nucleated cell count continued to rise throughout the entire period of study even after surgical correction of the obstruction in the animals of group III. This decrease in protein content might be due to hemodilution with fluid therapy after surgical correction and increase in cellular content could be attributed to the inflammation caused by resection and repair of intestine and laparotomy incision. Similar findings have been reported after exploratory celiotomy and omentopexy in cattle (Anderson *et al.*, 1994).

The peritoneal fluid electrolytes such as sodium, potassium and chloride concentrations in the animals of group II (Simple and strangulated) were almost similar like that of in pretreatment phase where, these ions confirmed decrease in their concentration. Surgical treated groups of both simple and strangulated obstruction showed recovery in their concentrations during the entire post treatment period which might be attributed to the resolution of third space (Luminal distension) responsible for electrolyte disturbance due to abomasal reflex, disruption of absorptive surface and increased secretory activity of the affected intestine.

5.2.5 RUMINAL FLUID STUDIES

The decreasing trend in ruminal fluid sodium and potassium was seen throughout the period of study in the animals of group II of either type of

obstruction. However, on intergroup comparison, it was found that a persistent loss of sodium and potassium throughout the period of obstruction was more in the animals of group I and group II as compared to group III in either type of obstruction. The reduction in ruminal fluid sodium and potassium might be ascribed to the absorption of this ion from rumen against concentration gradient in an attempt to moderate hypokalemia (Tasker, 1980).

The ruminal fluid chloride concentration is an important diagnostic tool of outflow obstruction. The increase in chloride concentration in conservatively treated groups II (Simple and strangulated) remained high throughout the period of study. Similarly chloride concentration in the surgically treated groups III (Simple and strangulated) showed a decreasing trend following surgery but remained high as compared to the base values. Similar findings were true for phosphorus but its concentration declined after surgery in either type of obstruction. The reason for increased ruminal fluid chloride could be due to continuous secretion of hydrochloric acid from abomasum, inflow of saliva into rumen and intensive chloride therapy given to animals to counteract the hypochloremia (Dobson and Philipson, 1958 and Makhdoomi *et al.*, 2002). It was observed from the present study that as soon as the motility of rumen and normal appetite was resumed, the rumen electrolytes concentration normalized.

5.3 CONSERVATIVE AND SURGICAL MANAGEMENT STUDIES

The medical therapy in the present study was primarily aimed at correction of fluid deficit, electrolyte imbalance and acid base abnormalities. The use of Ringers solution or a solution containing sodium chloride, potassium chloride, calcium borogluconate and dextrose intravenously has been suggested (Braun *et al.*, 1990) in cattle suffering from stenosis of pylorus and functional stenosis of the

duodenum at the level of sigmoid curve (Vander Velden, 1983). Use of specific fluid, antibiotics, analgesics and corticosteroids has been advocated by Tulleners (1981) and Smith (1985d) in cattle affected with intestinal volvulus.

Hammond *et al.* (1964) suggested that the correction of hypochloraemia and dehydration would probably be of primary importance, if relief of an obstruction was not immediately feasible. In the present study, attempt has been made to utilize the usefulness of a therapeutic regimen aimed at restoration of body fluids and electrolytes and use of antibiotics, analgesics, B- complex vitamins and rumenotronics to delay the progression of septic shock in the animals of group II of either type of obstruction and to aid in the reversal of shock and correction of specific metabolic disturbances following surgical treatment in group III (simple and strangulated). Intravenous administration of potassium chloride in the present study was included to reverse the metabolic alkalosis as reported by Papadopoulos *et al.* (1987).

The role of intravenous polyionic therapy is pertinent to treat dehydration, shock, electrolyte imbalance and prerenal azotemia in animals. A popular method of end to end anastomosis (Eggleston *et al.*, 2001) of small intestine comprising of two layer anastomosis with a simple continuous pattern for the submucosal/mucosal layer, followed by continuous Lembert pattern in the seromuscular layer was used in the present study, as it caused less inversion and lesser exposure of suture material to serosal surface, which reduced the risk of adhesions. This technique was faster and considered to produce large stomal diameter (Eggleston *et al.*, 2001).

The infusion of specific fluids and supportive therapy is therefore, important to prevent the dehydration and shock in the animals suffering from simple and

strangulated intestinal obstruction under field conditions. From economic point of view the fluid therapy instituted in this study was cheaper if the facilities of autoclaving exist in the veterinary hospitals in the field. Few studies are available in the literature wherein systemic therapy was used to treat intestinal obstruction in cattle (Smith, 1985c and Papadopoulos *et al.*, 1987) by using the intravenous fluids as per the status of dehydration and shock. The survival rate in the animals suffering from intestinal obstruction could be enhanced in the field conditions before referring such cases for surgical intervention. For a successful treatment or even for prolonging the life of an individual suffering from intestinal obstruction before being brought for specialized surgery, early observations of clinical signs by the owner, recognition of severity of the problem through history and physico-clinical examination of blood and peritoneal fluid by the veterinarian is absolutely necessary.

In the present study Ringer's solution was given to counteract metabolic alkalosis, dextrose and normal saline were given to cater the demand of energy and fluid deficit. The potassium chloride was given to offset the hypokalemia, metabolic alkalosis and muscular weakness associated with inappetance. Calcium borogluconate was instituted for maintenance of neuromuscular excitability, permeability of cell membranes and muscle contractions. The dextrose included in the fluid therapy enhances intracellular movement of potassium (Smith, 1990). Biobloom and antimony potassium tartrate (blend of *Sacchomyces cerevisiae*, *Lactobacillus sporogenes* fortified with phytase, calcium, phosphorous, proteins, carbohydrates, vitamins and growth factors) were given as rejuvenator of ruminal flora and ruminal motility respectively. Transfaunation with ruminal cud was done in all the animals 12 hours after surgical correction of intestinal obstruction.

Neostigmine is a prokinetic drug used 18 hours after surgery in animals to increase the peristalsis. Dexamethasone was used as an anti-inflammatory and as an adjunct to fluid therapy to correct the fluid disturbance. Vitamin C was given in surgically treated animals to prevent oxygen reperfusion injury. In the present study in both types of obstructions the treatments (conservative and surgical) were instituted when plasma chloride has not fallen below 75 mEq/L, plasma potassium has not declined below 3.5 mEq/L and haematocrit has not raised above 40 per cent. During treatments efforts were being made to maintain the concentrations at above mentioned optimal levels. Ringer's solution and potassium chloride were being given initially to maintain the concentration of plasma chloride and potassium at concentrations mentioned *ut supra*. When the concentration of these electrolyte were restored to optimal levels, the normal saline, dextrose normal saline (5 per cent) and Ca borogluconate were administered to cater the fluid deficit due to dehydration.

5.4 CLINICAL CASE STUDIES

The most common pathology identified in the clinical cases was intussusception, which refers to the invagination of one segment of intestine into the adjacent segment of intestine. Intussusception occurred sporadically in cattle of all ages, breeds and gender and may be seen anytime during the year (Constable *et al.*, 1997 and Pearson, 1971). In the present study the sexually intact females aging between 2 to 8 years especially during summers with lean fodder period were presented. Although the inciting cause could not be identified but intussusception has been related to many inciting factors such as enteritis, intestinal parasitism, sudden change in diet, mural granuloma or abscess, intestinal neoplasia, mural haematoma and administration of drugs that affect the

intestinal motility (Andersson and Ewoldt, 2005). Any focal disturbance of intestinal motility may facilitate the invagination of an oral segment into abroad segment of intestine. In the present study, the common history with most of the cases was feeding of bamboo leaves and enteritis. Intussusception most commonly occurred in distal portion of jejunum but may also be found affecting the proximal jejunum, ileum, caecum and spiral colon (Constable *et al.*, 1997 and Strand *et al.*, 1993). These findings were in concordance with the present study where Jejunojejunal (n=5), Jejunoileocaecocolic (n=1), Ileocaecocolic (n=1), Caecocolic (n=7) and Colocolic (n=1) obstructions were encountered. In a review of 336 intussusceptions in cattle 281 affected the small intestine, 7 were ileocolic, 12 were caecocolic and 36 were colocolic (Constable *et al.*, 1997).

Cattle affected with intussusception showed initial symptoms of pain. Later after 24 hours these symptoms subsided and affected cattle became progressively lethargic, recumbent with apparent depression and abdominal distension by 24-48 hours. Similar observations were seen by Anderson and Ewoldt (2005). Fecal production was normal for 12 hours after the occurrence of the intussusception, but minimal fecal production was noted after 24 hours. Passage of blood and mucous from the rectum is common at this time (Anderson and Ewoldt, 2005) and the same has also been observed in the present study.

Haemoconcentration, inflammatory leuckogram, hypochloremic alkalosis, hyponatraemia, hypokalemia, and azotemia were found in the present study. Similar observations have been documented by many authors (Pearson, 1971; Constable *et al.*, 1997; Anderson and Ewoldt, 2005). Proximal jejunal intussusception has been shown to cause rapid and severe dehydration, electrolyte sequestration and metabolic alkalosis (Anderson and Ewoldt, 2005).

The pathophysiologic events that result in intussusception remain to be elucidated, but an important feature would appear to be failure of coordinated motor activity in the affected segment. This can result from diseases that produce discontinuous segmental intestinal flaccidity or induration or a mechanical linkage of non adjacent segments due to linear foreign bodies or fibrin adhesions that result in kink in the bowel (Lewis and Ellison, 1987). Peristaltic activity continues the invagination once it has begun. Mesenteric tension limits the length of the invagination. The consequences of the intussusception may be intestinal obstruction and ischemia. The lymphatic and venous drainage are obstructed early in process, resulting in vascular engorgement, intramural oedema and hemorrhage, and loss of plasma and blood into lumen of the gastrointestinal tract. Exudation of fibrin, with in a short time can render the intussusception irreducible. Mural necrosis may eventually result, but perforation is rare because the ensheathing intestinal segments usually retain their viability (Lewis and Ellison, 1987).

In the present study, the diagnosis of intussusception was usually made by per rectal examination with presence of tympanitic loops of intestine, tight mesenteric bands and discrete pain on palpation of the obstructed loops. Per-rectal diagnosis was almost difficult in the pregnant animals of more than 5 months. Differential diagnosis included primary indigestion, functional ileus, trichobezoar, foreign bodies, intestinal incarceration or strangulation, vagal syndrome, intestinal neoplasia, fat necrosis and jejunal flange volvulus.

Affected cattle were stabilized before surgical intervention. Fluid therapy was administered to replace fluid and electrolyte deficits. Most of the surgeries were performed in standing position. The tension on the mesentery of the small

intestine resulted in pain and cattle made attempt to lie down during surgery. Of 35 cattle having standing right paralumbar fossa laparotomy for resection of intussusception, 14 per cent became recumbent during the surgery (Constable *et al.*, 1997). In such type of cases Anderson and Ewoldt (2005) has recommended that operative procedure should be performed under general anesthesia. Surgical removal by resection and anastomosis is the treatment of choice for intussusception. Manual reduction of the intussusception is not recommended because of risk of rupture of the intestine during manipulation. Probable ischemic necrosis of the intestine after surgery and prolonged ileus may be caused by motility disturbance and swelling in the affected segment of bowel. In the present cases, the margins for excision were selected in healthy appearing intestine. Hermann (2002) recommended that the larger (30cm) proximal segment from the lesion and short (10cm) distal to lesion should be selected for resection because the injury to proximal segment is more due to luminal distension, microvascular thrombosis, relative ischemia and noxious ingesta accumulated in the proximal segment that may cause severe and prolonged ileus.

The mesentery selected for resection of the intestine was infiltrated with 2 per cent xylocaine to decrease the pain of traction in the present study. The mesenteric vessels were ligated using mass ligation with absorbable suture. Mass ligation is required because cattle did not have an arcuate vascular anatomy as do horses, and the fatty mesentery renders vessels identification difficult and time consuming. The anastomosis was performed in two layer technique in the present study as per technique described by Eggleston (2001) using 2-0 Polyglactin 910. The mesentery was sutured for restoring the continuity. The anastomosed intestine washed thoroughly with sterile lukewarm normal saline, checked for

leakage and replaced back in the abdomen. In the present study, metronidazole (100 mg) was infused into the peritoneal cavity to prevent adhesions and anaerobic infection.

Post operative management in the present study was directed to prevent dehydration (Use of ringer's solution, normal saline, Ca borogluconate and 5 per cent dextrose), maintain optimal blood electrolyte concentration, control of infection/ inflammation and to stimulate appetite. Administration of intravenous fluids was found beneficial during the first 24 hours after surgery (Anderson and Ewoldt, 2005). The transfaunation of ruminal cud 12 hours after surgery in the present study was done in all the cases to stimulate forestomach motility and appetite. The animals were fed, rice gruel, soft hay and treacle after 6 hours of surgery to prevent ileus. Neostigmine was given in the animals where the animal has not defected with in 12 hours of surgery.

The favorable prognostic factors in the present study were the passage of feces with in 12 hours of corrective surgery. The affected animals, if operated with in 24 to 48 hours of obstruction responded favorably to surgery as also been reported by Anderson and Ewoldt (2005). In the present study the cattle presented with dehydration (PCV >45 per cent), hypochloraemia (<78 mEq/L). Hypokalemia (< 3.5 mEq/L) with severe abdominal distension and recumbency along with pregnancy had a poor prognosis for survival. Young non lactating animals responded more favorably to surgery than adult and high yielding cattle. The clinical cases with rupture of viscera presented at the time of surgery made the prognosis grievous. In the present study out of 15 cases 6 animals survived and 9 animals died. Non survivors also included 3 pregnant animals and 3 with intestinal rupture and peritonitis.

CHAPTER - VI

SUMMARY AND CONCLUSIONS

Simple and strangulated obstructions of cranial jejunum were created in 24 calves, which were randomly divided into six groups of four animals each. The animals of group I served as untreated control. In strangulated obstruction the animals of group II were treated with conservative therapy alone at 24th post obstruction hour whereas in simple obstruction they were treated at 3rd post obstruction day. Similarly, the strangulated and simple intestinal obstruction of cranial jejunum in the animals of group III were corrected by surgical intervention at 24th post obstruction hour and 3rd post obstruction day respectively and these animals were also maintained on conservative therapy till the end of study. The blood, peritoneal fluid and ruminal fluid samples were collected at 0, 24, 48, 72 and 96 post obstruction hours (Strangulated obstruction) and at 0, 2, 3, 4, 6 and 8th post obstruction day (Simple obstruction). Clinical, physical, cytological, haematological and biochemical alterations in body fluids were studied before and after induction of simple and strangulated intestinal obstruction and following its treatment.

All the animals immediately after creation of simple intestinal obstruction showed mild signs of discomfort like reflex guarding of abdomen while standing, grinding of teeth and mild pain during movement. These symptoms abolished within 6-8 hours. The mild abdominal pain reappeared after two days exhibited by groaning, kicking at the abdomen and restlessness. These signs disappeared at 4th day onward and thereafter the symptoms of weakness were observed. Stools became insufficient, desiccated and mucoid with strained defecation. Progressive generalized muscular debility was observed constantly in all the animals. Total atony of rumen was seen alongwith the sluggish protozoal motility.

Intestinal borborygmi were audible on auscultation upto 4th post obstruction day. The abdomen was bilaterally distended and a comprehensive lassitude marked by diminished elasticity of skin, eyeball recession and increased capillary refill time was recorded. Elevated haemoglobin, packed cell volume (38.33 ± 0.88 per cent) and total erythrocytic count was observed in all the animals. At 3rd day, following simple intestinal obstruction the increase in total leukocytic count (11062.5 ± 319.69 cells/ μ l) with absolute neutrophilia, hypochloraemia (75.12 ± 2.05 mEq/L), hypokalemia (3.8 ± 0.13 mEq/L), azotemia (BUN: 22.83 ± 2.78 mg/dl and Creatinine: 2.17 ± 0.27 mg/dl), hyperproteinemia (8.04 ± 0.21 mg/dl) and increased level of alkaline phosphatase (112.58 ± 2.08 IU/L) and amylase (28.17 ± 1.28 IU/L) were observed. High yellow colour discoloration of peritoneal fluid with increased levels of total protein concentration (3.8 ± 0.14 mg/dl), nucleated cell count (3412.5 ± 91.93 cells/ μ l), decreased sodium (140.33 ± 0.77 mEq/L), potassium (3.84 ± 0.14 mEq/L) and chloride (90.53 ± 1.89 mEq/L) concentration were recorded. The decrease in ruminal fluid sodium and potassium and substantial increase in ruminal fluid chloride (53.49 ± 1.19 mEq/L) concentration was a prominent finding. The electrocardiograms revealed shallow T wave and ST segment depression at 6th day in simple intestinal obstruction.

All the animals following creation of strangulated intestinal obstruction exhibited clinical signs of acute abdominal pain manifested by kicking at the abdomen, restlessness, lying down and getting up frequently, bruxism within half to two hours and these signs persisted upto 4-5 hours. Intensity of these signs was more severe during 2-3 hours following creation of intestinal obstruction. Defecation became scanty after 12 hours of creation and after 24 hours only the mucous with foul odour was voided which later after 48 hours contained the mucosal and

diphtheric shreds till the death of animals. Signs of muscular weakness were constantly observed in all the animals of three groups which were manifested initially by reluctance to move after 12 hours and instability of hind limbs at 24 hours, difficulty while assuming the sternal recumbency and getting up on feet at 72 hours. The rumen was usually hypotonic on 24 hours and became atonic on and after 48 hours. There was an appreciable loss in large ruminal protozoa at 24 hours and later after 48 hours onward the protozoal motility comprising of only the smaller species of protozoa was feebly appreciable. Intestinal borborygmi were audible upto 48 hours and progressive bilateral abdominal distention was observed from 48 hours onward. All the animals manifested the signs of dehydration like the recession of eye ball from 48 hours and became markedly so at the end of the period of obstruction. The elasticity of skin decreased from 24 hours (3-4 seconds) and became inelastic after 48 (7-8 seconds) hours onward. Following 24 hours of creation of strangulated intestinal obstruction serious physiological alterations characterized by haemoconcentration (40.58 ± 0.91 per cent), leucocytosis (7879.17 ± 281.73 cells/ μ l) with neutrophilia were observed. The striking characteristics were hyperproteinemia (9.07 ± 0.13 mg/dl), azotemia (BUN: 19.44 ± 1.82 mg/dl and Creatinine: 1.23 ± 0.02 mg/dl), hypochloremia (68.17 ± 1.64 mEq/L), hypokalemia (3.45 ± 0.04 mEq/L) with raised levels of alkaline phosphatase (131.17 ± 1.75 IU/L) and amylase (27.67 ± 1.394 IU/L). The peritoneal fluid colour exhibited high yellowish colored tinge in the post obstruction period. A highly significant increase in the total proteins (4.45 ± 0.13 mg/dl) and nucleated cell count (3666.67 ± 127.38 cells/ μ l) of peritoneal fluid was observed at 24 hours. There was a significant decline in sodium, potassium and chloride concentrations of peritoneal fluid. The ruminal fluid chloride concentration

which rose to 42.12 ± 0.86 mEq/L can be an important diagnostic test to be included in biochemical profile.

The criteria for surgical and medical intervention were based on above findings which have a significant prognostic importance. In either type of obstructions the treatments were instituted when plasma chloride has not fallen below 75 mEq/L, plasma potassium has not declined below 3.5 mEq/L and haematocrit has not raised above 40 per cent. During treatments efforts were being made to maintain the concentrations at above mentioned optimal levels. Ringer's solution and potassium chloride were being given to maintain the optimal concentration of chloride and potassium as mentioned *ut supra*. When the electrolyte levels were restored, the normal saline, dextrose normal saline (5 per cent) and calcium borogluconate were administered to cater the fluid deficit due to dehydration. Post operative supportive therapy comprising of antibiotics, anti-inflammatory drugs, ruminal motility rejuvenators, and transfaunation aided in improving the prognosis in intestinal obstruction.

The effectiveness of conservative treatment in group II and surgical treatment alongwith conservative treatment in the animals of group III of either type of obstruction was evidenced clinically. The conservative treatment given to the animals of group II decreased the pace of deterioration of the pathophysiology and increased the life span of the animals as compared to the animals of group I. Haemoconcentration and other signs of dehydration also subsided in the animals of group II and III following treatment. The concentrations of total protein decreased and blood electrolytes increased in the post treatment period when compared to the day of treatment (24 hours in strangulated and 3rd day in simple obstruction). The peritoneal fluid samples collected before obstruction revealed no growth. However,

Gram +ve bacilli, Gram –ve rods and Gram –ve cocobacilli were found during post treatment period. The isolates were found sensitive to Amoxycillin, Gentamicin, Tetracycline, Ciprofloxacin, Cloxacillin, Erythromycin and Penicillin. The histopathological changes in the strangulated intestine revealed autolytic changes whereas in simple obstruction necrotic changes were evident.

CONCLUSIONS

Based on the results of present study, the following conclusions were drawn

1. The Strangulated and simple cranial jejunal obstruction produced severe pathophysiology within 24 hours and 72 hours of creation respectively manifested by scant feces with mucosal/diphtheric shreds, hypochloraemia, hypokalemia, haemoconcentration, azotemia with increased plasma alkaline phosphatase and amylase concentration.
2. In strangulated and simple obstruction the prognosis is favourable if
 - i. Plasma Chloride is not < 75 mEq/L
 - ii. Plasma Potassium is not < 3.5 mEq/L
 - iii. Hematocrit is not > 40 per cent
3. Increased ruminal fluid chloride (> 40 mEq/L) indicated outflow obstruction thus is an important parameter to be included in a diagnostic profile.
4. Conservative treatment following simple and strangulated intestinal obstruction in the animals of group II decreased the pace of deterioration of pathology as compared to diseased control group I. It increased the survival time by 14 hours in strangulated and one and half day in simple intestinal obstruction as compared to control group I.

5. Blood chloride concentration can serve as an index for the type of fluid to be administered in both types of obstruction. To correct the hypochloreaemia, Ringer's solution combined with potassium chloride can be given and when the blood chloride level are restored optimally, normal saline and dextrose normal saline can be given for maintenance.
6. The clinical cases of intussusception in cattle presented with dehydration (PCV > 45 per cent), hypochloreaemia (<78 mEq/L), hypokalemia (<3.5 mEq/L) and increased ruminal concentration (74 mEq/L) with severe abdominal distension, recumbency along with pregnancy had poor prognosis.
7. To avoid post surgical mortality the supportive treatment should comprise of Ringer's solution, potassium chloride, isotonic saline, dextrose normal saline, calcium borogluconate, antibiotics, corticosteroids and transfaunation with rumen microflora rejuvenators.

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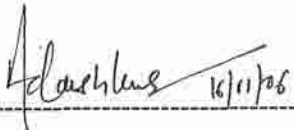
ABSTRACT

Simple and strangulated obstructions of cranial jejunum were created in 24 calves, which were randomly divided into six groups of four animals each. The animals of group I in both type of obstructions served as untreated control. The animals of group II and III of either type of obstructions were treated with conservative therapy and surgical intervention at 24th post obstruction hour in strangulated and 3rd day in simple intestinal obstruction respectively. The blood, peritoneal and ruminal fluid samples were collected at 0, 24, 48, 72 and 96 post obstruction hours (Strangulated obstruction) and at 0, 2, 3, 4, 6 and 8th post obstruction day (Simple obstruction). Clinical, physical, cytological, haematological and biochemical alterations in different body fluids were studied before and after induction of simple and strangulated intestinal obstruction and following its treatment.

Following creation of simple intestinal obstruction, mild signs of abdominal colic and scant mucoid faeces were recorded. Muscular debility, atony of rumen alongwith the sluggish protozoal motility was observed. Intestinal borborygmi were present upto 4th post obstruction day. The abdomen was bilaterally distended with signs of dehydration. Increased haematocrit (38.33 ± 0.88 per cent), total leukocytic count (11062.5 ± 319.69 cells/ μ l) with absolute neutrophilia, hypochloraemia (75.12 ± 2.05 mEq/L), hypokalemia (3.8 ± 0.13 mEq/L), azotemia (BUN: 22.83 ± 2.78 mg/dl and Creatinine: 2.17 ± 0.27 mg/dl), hyperproteinemia (8.04 ± 0.21 mg/dl) and increased level of alkaline phosphatase (112.58 ± 2.08 IU/L) and amylase (28.17 ± 1.28 IU/L) were observed. High yellow colour discoloration of peritoneal fluid with increased levels of total protein concentration (3.8 ± 0.14 mg/dl), and nucleated cell count (3412.5 ± 91.93 cells/ μ l) were recorded. The increase in ruminal fluid chloride (53.49 ± 1.19 mEq/L) concentration was a prominent finding. Following creation of strangulated intestinal obstruction signs of acute abdominal colic were observed. Defecation became scanty and contained the mucosal and diphtheric shreds. Muscular weakness, ruminal atony with signs of dehydration like the recession of eye ball and increased skin tent (7-8 seconds) after 48 hours were observed. Within 24 hours of creation, haemoconcentration (40.58 ± 0.91 per cent), leucocytosis (7879.17 ± 281.73 cells/ μ l) with neutrophilia was seen. The striking characteristics were hyperproteinemia (9.07 ± 0.13

mg/dl), azotemia (BUN: 19.44 ± 1.82 mg/dl and Creatinine: 1.23 ± 0.02 mg/dl), hypochloremia (68.17 ± 1.64 mEq/L), hypokalemia (3.45 ± 0.04 mEq/L) with raised levels of alkaline phosphatase (131.17 ± 1.75 IU/L) and amylase (27.67 ± 1.394 IU/L). The peritoneal fluid colour exhibited high yellowish colored tinge in the post obstruction period. A highly significant increase in the total proteins (4.45 ± 0.13 mg/dl) and nucleated cell count (3666.67 ± 127.38 cells/ μ l) in peritoneal fluid was observed at 24 hours. The ruminal fluid chloride concentration significantly rose to 42.12 ± 0.86 mEq/L.

In either type of obstructions the treatments were instituted when plasma chloride has not fallen below 75 mEq/L, plasma potassium has not declined below 3.5 mEq/L and haematocrit has not risen above 40 per cent. During treatments, efforts were being made to maintain the concentrations at above mentioned optimal levels. Ringer's solution and potassium chloride were being given to maintain the optimal concentration of chloride and potassium as mentioned *ut supra*. When the electrolyte levels were restored, the normal saline, dextrose normal saline (5 per cent) and calcium borogluconate were administered to cater the fluid deficit due to dehydration. The surgical treatment comprised of right flank laparotomy, resection of non viable intestine and enteroanastomosis. Post operative supportive therapy comprising of antibiotics, anti-inflammatory drugs, ruminal motility rejuvenators, and transfaunation aided in improving the prognosis in both types intestinal obstruction. The conservative treatment given to the animals of group II decreased the pace of deterioration of the pathophysiology and increased the life span of the animals as compared to control group. The diagnostic and therapeutic strategy which was applied in 15 clinical cases, out of which 6 animals survived and 9 animals died since they were presented late with dehydration (PCV >45 per cent), hypochloreaemia (<78 mEq/L) and Hypokalemia (< 3.5 mEq/L). Three animals were pregnant (more than 5 months) and three had rupture and peritonitis. The histopathological changes in the strangulated intestine revealed autolytic changes whereas in simple obstruction necrotic changes were evident.



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