DETAILED PATHOPHYSIOLOGIC STUDIES OF INTESTINAL OBSTRUCTIONS IN GOATS

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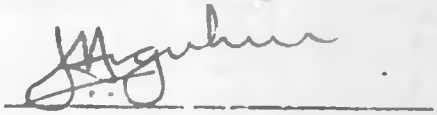
DECLARATION:

(a) This thesis is my original work and has not been presented for a degree in any other University.



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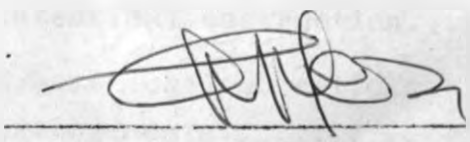

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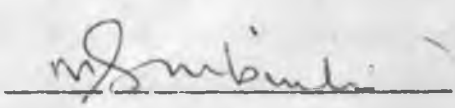

Dr. S.M. Mbiuki

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To my loving wife Purity N. Nguhiu,

and daughter Grace W. Nguhiu.

ABSTRACT OF THESIS

Literature is available on studies of physical signs and blood biochemistry in horses with intestinal obstruction (Datt and Usenik, 1975). In cattle, Hammond et al. (1964), studied the effects of obstruction on water and electrolyte metabolism. Studies on effects of strangulated intestinal obstruction on peritoneal fluid and blood parameters in buffalo calves was conducted by Krishnamurthy et al. (1980). In sheep, Gingerich and Murdick (1975), studied paradoxical aciduria during intestinal obstruction. However, these studies are not exhaustively comprehensive about different ruminant species.

No literature was available to us on studies in goats. The present investigation is designed to study the pathogenesis of intestinal obstruction at different sites in the goat.

Eighteen Masai goats were used in these experiments. They were divided into 3 groups. Group A consisted of 6 female goats which were subjected to simple duodenal obstruction. Group B consisted of 6 female goats which underwent strangulating obstruction of jejunum. Finally, group C consisted of 6 male goats which were subjected to strangulating obstruction involving the ileocaecocolic junction.

Obstruction was achieved in each case by ligation of the intestine and the mesentery with a double-loop of plastic tubing.

Parameters taken from each goat over the 48 hour-period immediately preceding the obstructions, served as control for each of these goats.Three other goats which were not subjected to any obstruction, were used for histopathologic control.

Clinical features were monitored every 6 hours. Blood parameters including: sodium, potassium, chloride, blood urea nitrogen, packed cell volume, erythrocyte count, haemoglobin concentration, leucocyte count, plasma protein and serum albumin, were all monitored every 12 hours.

Also monitored every 12 hours were the urinary parameters including: colour, transparency, quantity, specific gravity, urinary pH, protein, glucose, ketones, and the rate of excretion of sodium, potassium and chloride.

Peritoneal fluid was sampled every 12 hours, and analysed for colour, transparency, odour, erythrocyte and leucocyte count.

After death, necropsy was performed with more emphasis on the gastrointestinal tract. Histopathologic studies on intestinal tract were carried out.

Dehydration was a main clinical feature in the 3 groups, but reached a higher degree in group A.

Gastrointestinal stasis was evident terminally in all the groups. All the goats in these groups had splashing abdominal fluid sounds, but those in group A had symmetrical abdominal distension in addition.

The survival order in increasing survival time, was B. C & A respectively.

There was hypernatraemia in group A, hypokalaemia in group B, while hypochloraemia, and elevated blood urea nitrogen were observed in the 3 groups.

Oliguria, elevated urinary specific gravity, lowered urinary pH, and decreased rate of urinary excretion of sodium and chloride were observed in the 3 groups.

Increased peritoneal fluid, as well as elevated leucocyte count in this fluid was an invariable finding in all the groups.

Congestion and haemorrhages were grossly evident on intestinal serosal surface, and it was more severe in the strangulated loops. Mucosal haemorrhages were grossly seen in the strangulated segments. The lumen of the strangulated loops was filled with dark-red fluid contents.

Histologically, there was loss of villus epithelium in group A, and loss of villus architecture in groups B & C.

Congestion, haemorrhage and oedema in the lamina propria, submucosa, and sometimes in the tunica muscularis and serosa were evident in the 3 groups. These lesions were more marked in groups B & C.

Cellular infiltration into the intestinal wall layers, especially the tunica mucosa, included neutrophils and macrophages.

1 INTRODUCTION

Any mechanical or functional interference with progression of intestinal contents, with or without occlusion of the blood supply to the intestine is termed "intestinal obstruction".

Extensive studies on the effect of intestinal obstruction on various body parameters in man and simple-stomached animals, have been carried out. Information on clinical manifestation, diagnosis, management and treatment of simple and strangulated intestinal obstruction in the dog (Cohn, 1961); cattle (Hammond et al., 1964); sheep (Gingerich and Murdick, 1975); horse (Datt and Usenik, 1975); and in the buffalo (Krishnamurthy et al., 1980) is available.

Of the ruminants, there is no available literature on a comprehensive study, or on detailed systematic clinical reports on the effect of intestinal obstruction in the goat.

The field veterinarian in practice has often been faced with difficulty in making

quick and accurate clinical diagnosis on cases of intestinal obstruction in ruminants. This is because the condition is more rare and less severe clinically in the ruminants as compared to its manifestation in other species (Weipers, 1965; Hofmeyr, 1974), and also due to unavailability of comprehensive literature on the effect of the condition in ruminants. The difficulty is particularly common in cases where rectal exploration is not possible, for example in the small ruminants.

In most cases, diagnosis is made only on radiography, exploratory laparotomy, or at necropsy. Radiographic diagnosis is almost always restricted to the canine, feline and man.

At the time the veterinarian observes convincing signs in ruminants to decide to perform an exploratory laparotomy, it is often too late to reverse the effects of the intestinal obstruction on various body systems, even after relief and correction of this obstruction (Gingerich and Murdick, 1975). The prognosis at this stage is usually unfavourable due to fluid, electrolyte, and

acid-base imbalances. Exploratory laparotomy may at this stage, aggravate the condition of the patient due to enhanced gross imbalance and toxaemia.

The present research was therefore designed with the following objectives:

a) to undertake a comprehensive systematic study of the effects of surgically induced
   (i) simple duodenal obstruction,
   (ii) strangulation obstruction of jejunum, and
   (iii) strangulation obstruction of ileocaecocolic area, in the goat.

b) to compare the effects of duration and the site of intestinal obstruction in the goat.

c) to investigate whether there are any differences between the effects of intestinal obstruction in the goat and the reported effects in other species.

These objectives were achieved by carrying out a detailed study on clinical features, blood picture, urine changes,

peritoneal fluid changes, gross pathological, and histopathological changes in goats with induced intestinal obstruction.

The results obtained, will in conjuction with the already available information in other ruminants, be a future aid in making early clinical diagnosis of intestinal obstructions before the disease process progresses to an irreversible state.

2. LITERATURE REVIEW

2.1 Anatomy of the Intestine

This anatomical description is restricted to sheep and goat.

The small intestine is generally divided into three parts namely: duodenum, jejunum, and ileum. It is 18.0-35.0 metres long, with jejunum as the longest.

The duodenum is the first part and begins from the pylorus of the abomasum. It is suspended by a short mesentery, the mesoduodenum which becomes longer at the duodenojejunal flexure. It is divided into three parts. The cranial duodenum is a sigmoid loop closely related to the liver by hepatoduodenal ligament attachment and it receives the bile and pancreatic ducts. The descending and ascending duodenum form a U-shaped loop which is closely related to the kidneys (Nickel et al., 1973; Habel, 1975).

The jejunum forms numerous tight coils arranged along the border of the long mesentery. It forms a series of U-shaped loops just

before joining the ileum (Nickel et al., 1973; Habel, 1975)

The ileum starts from ileocaecal fold which arises from antimesenteric border to ileocaecal orifice, which is on the medial surface. It is adherent to the caecum and colon cranially (Nickel et al., 1973).

Histologically, the small intestine is divided into four main layers: tunica mucosa, tunica submucosa, tunica muscularis, and tunica serosa (Trautmann and Fiebiger, 1952; Dellmann, 1971; Nickel et al., 1973).

The tunica mucosa consists of lamina epithelialis mucosae, lamina propria, and lamina muscularis mucosae. Lamina epithelialis mucosae has absorptive simple columnar epithelium, secretive goblet cells, and argentaffin cells. Lamina propria has aggregates of lymphatic tissue (Peyer's patches), blood vessels, and intestinal glands (Crypts of Lieberkuhn) which are tortous in the goat unlike in the other species where they are longer and straight. Lamina muscularis mucosae has an inner circular and outer longitudinal layers of smooth muscles. The mucous

membrane has countless finger-like microscopic projections (the intestinal villi) which give it a velvety appearance. (Trautmann and Fiebiger, 1952; Dellmann, 1971; Nickel et al., 1973).

The tunica submucosa consists of submucosal or duodenal (Brunner's) glands, which in the goat are only within 20-25 centimetres (cm) from the pylorus (Habel, 1975).

The submucosal layer has dense connective tissue, blood vascularity, lymphatics, and nerve network (Trautmann and Fiebiger, 1952; Dellmann, 1971; Nickel et al., 1973).

The tunica muscularis consists of an inner circular and an outer longitudinal layers of muscle bundles. These two layers are separated by a thin layer of connective tissue (Trautmann and Fiebiger 1952; Dellmann, 1971; Nickel et al., 1973).

The large intestine is divided into caecum, colon and rectum. It is 4.0-8.0 metres long (Nickel et al., 1973; Habel, 1975).

The caecum is slightly s-shaped and its base is attached to the mesentery cranially,

with the apex lying near the pelvic inlet (Nickel et al., 1973; Habel, 1975).

The colon is divided into ascending colon, transerve colon suspended by a short mesocolon, and descending colon with a longer mesocolon at the level of the first lumbar vertebra (Nickel et al., 1973; Habel, 1975).

The rectum has the cranial part covered by peritoneum and a caudal retroperitoneal part (ampulla recti). It has a short mesorectum. The rectococcygeus muscle from the dorsal surface of the rectum is attached to the caudal vertebrae (Habel, 1975).

The mucous membrane of large intestine has no villi (Trautmann, 1952). The other structural characteristics are similar to those of the small intestine.

Small intestine is supplied with blood by cranial mesenteric and celiac arteries; and the large intestine by caudal mesenteric and internal iliac arteries. The entire intestinal tract is drained into portal vein and caudal vena cava (Nickel, et al., 1973).

The intestine is innervated by autonomic nervous system (Trautmann and Fiebiger, 1952; Bloom and Fawcett, 1970; Nickel et al., 1973).

2.2 Physiology of the Intestine

(Breazile, 1971)

The intestinal villi are covered by columnar cells which absorb nutrients, goblet cells that produce protective mucus, and enterochromaffin (argentaffin) cells whose function is unknown.

The intestinal glands (Crypts of Lieberkuhn) have undifferentiated cells which replace the surface mucosal cells that are disquamated. These glands also have paneth cells which secrete amylase and enterokinase enzymes.

Duodenal glands have the major constituent of their secretion as mucoprotein, but other proteins are present - probably proteases, amylases, enterokinases and pepsin. They also produce a protective secretion. The pancreas secretes enzymes for digestion of proteins, lipids, and carbohydrates, and also a secretion for regulating pH of the duodenum. The bile

juice from the gall bladder contains bile pigments, bile salts, cholesterol, lecithin and inorganic salts which are emptied into the duodenum through the bile duct.

Chyme is the undigested mixture of lipid, protein and carbohydrates reaching the small intestine from the stomach.

Protein is digested and absorbed as amino acids in the small intestine. Carbohydrate complex molecules reaching the small intestine undigested are digested and absorbed as monosaccharides. Lipids are hydrolysed by lipases and absorbed as monoglycerides and fatty acids.

Sodium and chloride are actively absorbed; potassium and bicarbonate are passively absorbed.

Most of the water is absorbed within the colon, but a considerable amount is also absorbed within the small intestine.

The mucosa of the large intestine is similar to that of the small intestine except it has no villi. Intestinal glands are present but no paneth cells.

In ruminants, a considerable quantity of cellulose material is digested within the large intestine by microbial fermentation. Some fatty acids and vitamins are produced by microbial activity within the large intestine.

Small intestine have propulsive movements known as segmental intermittent contractions where short segments of intestine contract alternatively and progressively in a proximo-distal direction as a result of muscular coat activity. These contractions aid in pushing the small intestinal contents towards the large intestine.

Rhythmic to-and-fro contractions of the longitudinal muscles of small intestinal wall produce pendular movements which mix the contents of the small intestinal lumen with the digestive juices.

Peristaltic waves in the small intestine are unphysiological movements resulting from irritation of the mucosa and progressing proximally and distally from the point of irritation. These occur with a higher frequency than the physiological movements, and therefore interfere with digestion.

Stimulation of parasympathetic nerves accelerates intestinal activity, while sympathetic stimulation depresses its activity.

Little is known about motor activity of large intestine of ruminants. But it is thought to have similar activity to small intestine with variation of the degree of activity. Unlike for small intestine, peristaltic waves are physiological movements for the large intestine, and are normally more frequent than in the small intestine.

2.3 Clinical Pathology

Several authors have described the normal values of goat's blood.

2.3.1. Haematology

The erythrocytes (RBC) values range from 7-22 x 10^{12}/L; the haemoglobin (Hb) values range from 8-16 gm% (Coffin, 1947; Bentinck-smith, 1969; Coles, 1974; Schalm et al., 1975; Greenwood, 1977; Benjamin, 1979).

Total leucocyte (WBC) count ($X10^3/mm^3$) ranges from 4-16 (Coffin, 1947; Coles, 1974; Benjamin, 1979). The differential leucocyte count in percentage ranges from 20-50 for neutrophils, 43.7-70 for lymphocytes, 1-10 for eosinophils, and 0-2 for basophils (Coles, 1974; Schalm et al., 1975; Greenwood, 1977; Benjamin, 1979).

2.3.2. Blood Biochemistry

Normal sodium ion value in blood is 142-155 mEq/L. Herbivores may have a deficiency in sodium unless there is supplementation (Coles, 1974).

The normal range of serum chloride concentration is 99-118 mEq/L (Meier, 1963; Coles, 1974). Chloride follows the sodium pattern. Hyperchloraemia may occur in some cases of dehydration (Coles, 1974).

The normal potassium range in serum is 3.5-6.7 mEq/L (Meier, 1963; Coles, 1974). According to Coles (1974), most potassium is in the intracellular fluid. The serum levels of potassium do not necessarily reflect the true status of body potassium concentration because

there is no constancy of relation between extracellular fluid and intracellular fluid potassium. Serum potassium levels can be misleading especially during massive cellular necrosis. In metabolic acidosis, the cells take up hydrogen ion and release potassium ion, while the reverse is true during metabolic alkalosis. Hence in metabolic alkalosis, hypokalaemia is experienced while there is an increase in intracellular fluid potassium.

Blood urea nitrogen (BUN) values range from 13.0-28.0 mg/100 ml. (Coffin, 1947; Bentinck-smith, 1969; Coles, 1974). As a rule, any time there is hyperkalaemia there is almost always retention of urea nitrogen (Meier, 1963). Some of the conditions that result in BUN elevation are diminished glomerular filtration, increased protein catabolism, secondary to internal haemorrhage, and necrosis (Duncan and Prasse, 1978).

The reported plasma protein range in gm/100 ml is 6.0 - 7.5 (Schalm et al., 1975). Plasma protein values are elevated during inflammatory conditions. Values can be lowered in blood loss conditions, and protein loss

from the intestinal tract (Bentinck-smith, 1969).

2.3.3 Urinalysis

This is a general description on most species.

Oliguria may be caused by reduced fluid intake, dehydration, or gastrointestinal disorders with vomiting and diarrhoea, among other causes (Benjamin, 1979).

Normal urine colour may be yellow to amber, with the depth of colour related to volume (Duncan and Prasse, 1978; Benjamin, 1979); colourless to pale-yellow urine is dilute with low specific gravity, while dark-yellow to yellow-brown urine is concentrated and has high specific gravity. Small quantity of urine could be a result of reduced fluid intake, dehydration, or prolonged vomiting and diarrhoea (Coles, 1974; Benjamin, 1979). Normal urine is clear when freshly voided. Cloudiness does not necessarily indicate pathological condition, because long standing urine may become cloudy (Bentinck-smith, 1969; Coles, 1974; Duncan and Prasse, 1978;

Benjamin, 1979). According to Bentinck-smith (1969), precipitation on cooling of urine is a common finding in dogs and horses.

Specific gravity of urine is inversely related to volume, the number and weight of solute molecules in the urine (Bentinck-smith, 1969; Coles, 1974; Duncan and Prasse, 1978). The normal value of specific gravity of urine in goats ranges from 1.015-1.045 (Bentinck-smith, 1969; Coles, 1974; Benjamin, 1979).

Herbivores void alkaline urine due to their vegetable diet (Bentinck-smith, 1969; Coles, 1974; Benjamin 1979). Duncan and Prasse (1978) reported that urine becomes alkaline on standing, due to loss of carbon dioxide as well as conversion of urea to ammonia by bacteria.

Under normal circumstances, protein should not be present in urine (Bentinck-smith, 1969; Coles, 1974; Duncan and Prasse, 1978; Benjamin, 1979). Renal insufficiency generally brought about by shock resulting from such diseases as pancreatic necrosis, intestinal obstruction, and trauma, causes

large quantities of protein to be voided in urine (Bentinck-smith, 1969). The author further reports that products of tissue destruction elsewhere in the body permit protein leakage from the renal capillaries into the urinary tract.

Glucose should not be present in normal urine (Bentinck-smith, 1969; Coles, 1974; Duncan and Prasse, 1978).

Ketones appear in urine only after their accumulation in blood (Bentinck-smith, 1969; Coles, 1974). However, according to Duncan and Prasse (1978), ketonuria appears earlier than ketonaemia. Ketonuria may be observed in conditions causing severe anorexia, and in cases of prolonged starvation (Bentinck-smith, 1969; Coles, 1974; Duncan and Prasse, 1978; Benjamin, 1979). Benjamin (1979) reported that prolonged vomiting and diarrhoea cause ketonuria.

Decreased urinary excretion of chloride has been observed during starvation, excessive vomiting and diarrhoea (Benjamin, 1979).

2.4 Intestinal Obstruction

2.4.1 Classification, aetiology, and pathogenesis

Any interference with normal intestinal motility, or progressive distal passage of intestinal contents is termed "intestinal obstruction" (Jubb and Kennedy, 1970).

Any mechanical, or functional interference with progression of intestinal contents, constitutes "intestinal obstruction" (Larsen and Bellenger, 1974).

Volvulus has been defined differently by various authors. It has been defined as rotation of the intestine about the long axis of the mesentery (Runnells et al., 1965); axial rotation of a portion of the intestine (Larsen and Bellenger, 1974). The Merck vet. Mannual (1973) defines it as intestinal obstrctuion due to twisting of the bowel on its mesenteric axis.

The definitions of intussusception include: telescoping of a section of the intestine into the distal portion (Runnells

et al., 1965); telescoping or invagination of a portion of intestine into an adjacent one (The Merck vet. Mannual, 1973); invagination of a portion of intestine into the segment that follows or precedes it (Larsen and Bellenger, 1974).

Torsion of the intestine is defined as twisting of the bowel on its own or long axis (The Merck Vet. Mannual, 1973), or a twist in the mesentery (Larsen and Bellenger, 1974).

Strangulation is when part of the intestine slips through an opening in the mesentery, omentum or abdominal wall (Larsen and Bellenger, 1974).

Incarceration is the occlusion of intestinal lumen by pressure from the serosal surface (The Merck Vet. Mannual, 1973).

Intestinal hernia is the displacement of the intestine through normal or pathological foraminae within the abdominal cavity without hernial sac (Jubb and Kennedy, 1970).

The Merck Vet. Mannual (1973), defines impaction as formation of enteroliths, or coproliths in the colon, signifying inadequate

propulsion of faecal material.

Intestinal obstructions can be divided into simple occlusions, strangulating obstructions, and functional obstructions (paralytic ileus) (Weipers, 1965; Jubb and Kennedy, 1970). They are also classified into high (proximal) obstructions whose clinical onset is rapid and acute, and includes obstructions of pylorus, duodenum, and proximal jejunum. Low (distal) obstructions whose clinical manifestation is chronic and includes obstructions of distal jejunum, ileum, colon, and rectum (Jubb and Kennedy, 1970; Palminteri, 1972; Larsen and Bellenger, 1974).

Intestinal obstructions can also be classified according to the degree of obstruction as complete, or incomplete obstruction (Larsen and Bellenger, 1974; Anderson, 1975).

Simple occlusions are mechanical in origin with intraluminal or external compression (Jubb and Kennedy, 1970), while strangulation obstructions have luminal and vascular (usually veins) occlusion. Paralytic

ileus is a functional obstruction due to reflex inhibition, or of metabolic origin.

Jubb and Kennedy (1970), classify the causes of intestinal obstruction as mechanical, nervous imbalance, and vascular causes.

Several factors have been reported as causes of simple intestinal obstruction. These factors are: foreign bodies, intraluminal or extraluminal abscesses, impaction with feed or faecal material, concretions, enteroliths, and haematomas (Runnells et al., 1965; Weipers, 1965; Jubb and Kennedy, 1970; Hofmeyr, 1974; Anderson, 1975; Hornbuckle and Kleine, 1977; Pearson and Pinsent, 1977; Blood et al., 1983).

Several causes of intestinal strangulation obstruction have been observed. These are: Hernias-inguinal, scrotal, ventral, mesenteric tear, and omental tear (Rooney, 1965; Runnells et al., 1965; Weipers, 1965; Fox, 1970; Jubb and Kennedy, 1970; Palminteri, 1972; Hofmeyr, 1974; Hornbuckle and Kleine,

1977; Foo et al., 1978; Blood et al., 1983); volvulus (Espersen, 1961; Rooney, 1965; Runnells et al., 1965; Weipers, 1965; Jubb and Kennedy, 1970; Hornbuckle and Kleine, 1977; Pearson, and Pinsent, 1977; Foo et al., 1978; Barclay et al., 1980). Intussusception, common at jejunoileal and ileocaecocolic junctions, and rarely colonic or jejunojejunal, is also one of the causes of intestinal strangulation obstruction. Factors predisposing to intussusception are bowel inflammation, leading to hyperperistalsis, drinking very cold water, nematode infestation, recent dietary change after weaning in puppies, tumour growths, and sometimes intussusception may occur during normal intestinal movements and then correct itself (physiological intussusception). But when it fails to correct itself it becomes pathological after circulatory interference results (Espersen, 1961; Rooney, 1965; Runnells et al., 1965; Lowe, 1966; Fox, 1970; Jubb and Kennedy, 1970; Palminteri, 1972; Hofmeyr, 1974; Anderson, 1975; Rees and Lari, 1976; Hornbuckle and Kleine, 1977; Pearson and Pinsent, 1977; Foo et al., 1978; Dutoit et al.,

1981; Blood et al., 1983). Other causes of strangulation obstruction are: thrombosis and embolism, common in the dog as a result of accidents, and in horses as verminous thromboarteritis of anterior mesenteric artery as a result of migration of strongylus vulgaris larvae, ending up with infarction which may also lead to paralytic ileus (Weipers, 1965; Jubb and Kennedy, 1970; Pearson and Pinsent, 1977; White, 1981); torsion of the mesentery, or intestinal mass around the cranial root of mesentery or of caecum (Runnells et al., 1965; Jubb and Kennedy, 1970; Palminteri, 1972; Hofmeyr, 1974; Anderson, 1975; Blood et al., 1983); caecal dilatation and dislocation-this could be sequela to intussusception, stenosis of colon, or volvulus of the intestine (Espersen, 1961; Pearson, 1963; Fox, 1970; Tehghani and Townsend, 1982).

Paralytic ileus is a functional intestinal obstruction. The causes of paralytic ileus are: peritonitis (Weipers, 1965; Blood et al., 1983); extensive trauma in surgical operations, and prolonged intestinal distension (Jubb and Kennedy, 1970; Blood

et al., 1983); and reflex inhibition of metabolic origin e.g. in hypokalaemia (Littlejohn and Brown, 1963; Jubb and Kennedy, 1970). Other causes of paralytic ileus are: vagal indigestion (Pearson and Pinsent, 1977); fast fermentation and stasis of the caecum (Espersen, 1961). Paralytic ileus may be a sequela to intestinal infarction (Jubb and Kennedy, 1970). Sometimes this functional obstruction may occur and not be diagnosed postoperatively, or no known cause is attributed to it (Weipers, 1965).

2.4.2 Clinical Features

Severity of intestinal obstruction depends on various factors: site-the proximity of obstruction to duodeno-pyloric junction, and the completeness of obstruction (Weipers, 1965; Anderson, 1975); circulatory involvement, and animal species (Blood et al., 1983).

The higher (closer to duodenopyloric junction) obstructions have a rapid and more acute clinical onset, while low obstructions have a chronic and less acute clinical manifestation (Jubb and Kennedy, 1970; Blood et al.,

1983). When blood supply to the obstructed segments is compromised, the syndrome is acute (Blood et al., 1983).

Weipers (1965), has reported differences in clinical manifestation with reference to different animal species. He has reported the horse as manifesting the most severe syndrome. Acute and strangulating obstructions in the dog may exhibit only dull pain which may or may not be continous, and sometimes this pain may fail to show until just before death. The author further reports that ruminants and pigs have an intermediate manifestation of the syndrome between horses and dogs.

Irrespective of the species, animals with simple intestinal obstruction show signs of depression and progressive weakness. Sometimes the syndrome has a sudden clinical manifestation. Loss of body weight and progressive deterioration of body condition until death, is an invariable feature in cases of intestinal obstruction (Hammond et al., 1964; Dixon, 1965; Corker and Dziuk, 1968; Fox, 1970; Hofmeyr, 1974; Pearson and Pinsent, 1977; Sherman, 1981; Ducharme et al., 1982).

Recumbency ensues in the terminal stages (Blood et al., 1983). According to Anderson (1975), incomplete intestinal obstruction presents similar signs as low intestinal obstructions.

It has been reported that ruminants with intestinal obstruction, first lose appetite for hay, then for grains, and finally they develop complete anorexia (Hammond et al., 1964; Corker and Dziuk, 1968; Fox, 1970; Hofmeyr, 1974; Pearson and Pinsent, 1977; Ducharme et al., 1982; Blood et al., 1983). Sherman (1981) observed complete anorexia on the second day of duodenal obstruction in a goat. Decreased appetite, then complete anorexia has been observed in dogs with complete intestinal obstruction (Anderson, 1975). Anorexia has also been reported in pigs with bowel obstruction (Blood et al., 1983).

It has been reported that defaecation is initially present during intestinal obstruction, but it later ceases (Corker and Dziuk, 1968; Fox, 1970; Hofmeyr, 1974; Pearson and Pinsent, 1977; Ducharme et al., 1982; Blood et al., 1983). According to Fox (1970),

defaecation in bovine, ceases immediately if obstruction is near the rectum, and later if near the duodenum. Hammond et al. (1964), did experimental work in calves, and reported that 24 hours after complete duodenal obstruction, defaecation either ceased totally, or there were few and dry small faecal pieces, or dark-liquid that had foul-smell. In cases of intussusception, there is passage of blood-stained mucus. This has been noticed in children (Rees and Lari, 1976; Dutoit et al., 1981).

Datt and Usenik (1975), have reported that there is straining to defaecate in later stages of intestinal obstruction in the horse. According to Johnston (1962), passage of small amount of faeces, either semi-solid mixed with blood and mucus (foetid), or foul-smelling with pasty consistency, is a positive diagnostic feature of intestinal obstruction in cattle.

Pain receptors on abdominal viscera, are either absent, or very few, and therefore pain is only exhibited when organs are stretched, bruised, torn, or spastic

(Hofmeyr, 1974).

In the horse with intestinal obstruction, pain is severe and is manifested by colicky signs of pawing on the ground, rising up and lying down, looking at the flank, tremors, sweating, ataxia, and rolling. This pain is usually of sudden onset in acute obstruction, and continous or intermittent colic in chronic, or subacute obstructions (Dixon, 1965; Weipers, 1965; Datt and Usenik, 1975; Barclay et al., 1980; Dobson and Lopez, 1981; Blood et al., 1983).

Pain in dogs with intestinal obstruction has been reported to be of a dull form, and sometimes it fails to show until a few hours to death (Weipers, 1965). The author further reports that in dogs with strangulation obstruction, pain may be continous. But Anderson (1975), reported that dogs with complete intestinal obstruction, were restless, showed signs of abdominal pain, and had arched back. According to Dutoit et al.(1981), dogs with intussusception manifested periodic painful attacks. Michell (1967), reported that during intestinal obstruction, pain is

less marked in dogs than in ruminants, or pigs, and is much less marked than in horses.

Johnston (1962), reported that cattle with intestinal obstruction, had signs of abdominal pain for 1-2 days. Some of the signs of pain observed in cattle are restlessness, kicking at the belly, looking at the flank, switching of the tail, hind limb paddling and stamping, depressed back, bellowing, groaning may or may not be present, frequent lying and rising, and stretching in recumbency (Johnston, 1962; Fox, 1970; Pearson, and Pinsent, 1977; Ducharme et al., 1982; Blood et al., 1983). This pain does not last for long, but disappears later (Fox, 1970; Pearson and Pinsent, 1977).

In man, pain is reported to be constant during strangulation obstruction, but repeated sudden painful attacks have been observed in chronic intussusception (Kingsnorth, 1976; Rees and Lari, 1976; Foo et al., 1978; Gough, 1978).

There is usually dilatation and distension of the intestine proximal to the site of obstruction due to fluid and gas accumulation.

The extent of this distension is influenced by the degree of completeness of obstruction (Jubb and Kennedy, 1970). It has been observed in man, that when fluid alone without air is accumulated in the bowel, there is usually no abdominal distension (Kingsnorth, 1976). Abdominal distension during bowel obstruciton, has been reported in cattle (Hammond et al., 1964; Pearson and Pinsent, 1977; Blood et al., 1983); in pigs and sheep (Blood et al., 1981); in man (Foo et al., 1978). Abdominal distension due to gas and fluid accumulation is an important diagnostic feature (Johnston, 1962).

On abdominal ballotment, and auscultation in ruminants with intestinal obstruction, splashing fluid sounds are detected (Johnston, 1962; Sherman, 1981; Blood et al., 1983). The abdomen has been reported to be usually tense on ballotment during bowel obstruction (Dixon, 1965; Hofmeyr, 1974). In dogs with intussusception, abdominal mass is usually palpated through rigid abdominal wall (Dutoit et al., 1981). Gough (1978), observed that during strangulation obstruction in man, abdominal tenderness is a common

finding.

Distension of the intestine with gas and fluid causes violent bowel contractions, initially, then later paresis due to muscular fatigue (Palminteri, 1972). Blood et al. (1983) noted that in bowel obstruction, gas gurgles are heard. It is generally noted that in the later stages of intestinal obstruction in horses and ruminants, there is gastrointestinal hypoperistalsis or aperistalsis (Dixon, 1965; Fox, 1970; Hofmeyr, 1974; Datt and Usenik, 1975; Pearson and Pinsent, 1977; Ducharme et al., 1982; Blood et al., 1983). In man, increased intestinal sound has been reported (Kingsnorth, 1976; Foo et al., 1978). Kingsnorth (1976), further reports that when no air, but only fluid accumulation in the bowel, there is usually no hyperactive bowel sounds.

Clinical dehydration is evident in all species during intestinal obstruction, and is manifested by sunken eyes. glazed eye appearance, lack of skin pliability, encrustation on the muzzle, cold extremeties, elevated packed cell volume, increased capillary refill time, and oliguria among others (Johnston, 1962;

Hammond et al., 1964; Dixon, 1965; Palminteri, 1972; Hofmeyr, 1974; Gingerich and Murdick, 1975; Pearson and Pinsent, 1977; Dobson and Lopez, 1981; Dutoit et al., 1981; Ducharme et al., 1982). This dehydration has been associated with loss of fluids into the intestine, peritoneal cavity, loss in sweat, and inability to drink during bowel obstruction (Datt and Usenik, 1975).

No change in temperature has been observed in most cattle during intestinal obstruction (Fox, 1970; Blood et al., 1983). But subnormal temperature has been observed as the illness progresses (Pearson and Pinsent, 1977; Sherman, 1981). In horses, temperature elevation has been reported, and associated with pain, excitement, inflammation of the gut wall, and muscular exertion; but it may be lowered in certain obstruction. High obstructions exhibit high temperatures in horses (Datt and Usenik, 1975; Blood et al., 1983). Gough (1978), has reported pyrexia in man as a common feature of strangulation obstruction.

Pulse rate during intestinal obstruction is reported to be dependent on whether or not circulatory disturbance is present. It is usually elevated after blood and fluid is accumulated in loops of intestine and dehydration has set in (Dixon, 1965; Fox, 1970; Hofmeyr, 1974; Datt and Usenik, 1975; Pearson and Pinsent, 1977; Dobson and Lopez, 1981; Sherman, 1981; Blood et al., 1983). This pulse is usually of a weak nature. Gough (1978) reported that tachycardia is a common finding in man with strangulation obstruction. Blood et al. (1983), has reported that pulse rate could also be normal during intestinal obstruction.

Dixon (1965), and Datt and Usenik (1975) reported elevation of respiratory rate in horses during intestinal obstruction. In cattle respiratory rate is usually unchanged, or irregular, but there is grunting and groaning (Fox, 1970; Pearson and Pinsent, 1977; Blood et al., 1983). Hofmeyr (1974), stated that respiratory rate is generally elevated during intestinal obstruction in most species.

Vomiting has been reported in dogs and man (Weipers, 1965; Anderson, 1975; Kingsworth, 1976; Rees and Lari, 1976; Foo et al., 1978; Dutoit et al., 1981).

Horses with intestinal obstruction have been observed to have injected, dark-red and oedematous ocular mucous membranes, and purple, cold-dry oral mucous membrane (Datt and Usenik, 1975; Dobson and Lopez, 1981; Blood et al., 1983).

During intestinal obstruction, Hofmeyr (1974), has reported that gas-filled loops of intestine can be palpated on rectal examination in cattle and horses, with sticky mucus, or blood in the rectum, and later more blood, with no faecal material.

Other authors have observed gas distended loops of intestine and empty rectum.(Dixon, 1965; Dobson and Lopez, 1981; Blood et al., 1983). In cattle, the rectum is usually empty except thick tenacious mucus or thick dark-red pasty material (blood exuded into the lumen of the intestine) with foul-smell (Blood et al., 1983). Sherman (1981), reported passing of mucus or scanty hard faecal pellets from a

goat with duodenal obstruction.

During intestinal obstruction, urination is normal in bovine (Fox, 1970), but in sheep oliguria has been observed (Gingerich and Murdick, 1975).

Gough (1978) has observed that in man, laboratory data are non-discriminatory against non-strangulating and strangulating obstructions. The author has further observed that during intestinal obstruction, man may be radiographically normal initially. Kingsnorth (1976), has reported that in man, during intestinal obstruction, multiple air-fluid-filled levels are observed on radiograph. The author has also found that when no air is present in the bowels, there is usually no air-fluid level.

Some animals occasionally recover spontaneously from intestinal obstruction (Hofmeyr, 1974). Death in higher obstructions is usually due to fluid and electrolyte loss, and in lower obstructions, it is primarily due to starvation (Jubb and Kennedy, 1970). According to Johnston (1962), death may also depend on the physical nature of the

obstruction, site of obstruction, infective agents present, and whether or not bacterial toxins are present. Death in strangulation obstruction has been ascribed to blood loss, products from autolysis of necrotic section, and bacteria (Yale, 1969).

Untreated cases of intestinal obstruction in cattle, may be in standing posture until a few hours to death, and may survive for two weeks or more. Survival time has been reported to vary from 6-18 days (Johnston, 1962; Pearson and Pinsent, 1977; Blood et al., 1983).

Pearson and Pinsent (1977), have stated that survival time depends on development of secondary circulatory complications. Buffalo calves with intestinal strangulation obstruction survived 48-84 hours (Krishnamurthy et al., 1980). Sheep with simple duodenal obstruction did not survive even after they were relieved of the obstruction (Gingerich and Murdick, 1975). Blood et al. (1983), have recorded death period in sheep and pigs with bowel obstruction, between 3-6 days post-obstruction. In horses, the syndrome is acute and short, and death ensures within 24 hours of obstruction

(Johnston, 1962; Blood et al., 1983). Datt and Usenik (1975), observed that horses with duodenal obstruction survived shortest due to excessive fluid loss, distension and rupture of the stomachs, and finally septic shock.

According to Johnston (1962), the concept that the closer the intestinal obstruction to the stomach, the more rapidly will death ensue, does not always apply in cattle.

2.4.3 Diagnosis and Differential Diagnosis

Adequate history is an essential feature in diagnosis of intestinal obstruction. This may include change in feeding habits, signs observed by the clients on their patients, duration of symptom manifestation, previous abdominal surgery, and whether any therapy, or surgical intervention has been instituted. (Palminteri, 1972; The Merck Vet. Mannual, 1973; Larsen and Bellenger, 1974; Kingsnorth, 1976).

Complete thorough clinical examination and findings is an essental aid to diagnosis of bowel obstruction (The Merck Vet. Mannual,

1973; Larsen and Bellenger, 1974; Hornbuckle and Kleine 1977). In dogs, signs of vomiting, depression, dehydration, abdominal distension, discomfort, restlessness, passage of scanty faeces and polydypsia may suggest presence of bowel obstruction (Palminteri, 1972).

In cattle, Pearson and Pinsent (1977), observed that signs of colic, absence of faeces, postural abnormalities, abdominal distension, increased resonance and pitch in the dilated loops of the gut on ausculatation, and fluid splashes on abdominal ballotment and auscultation, may suggest bowel obstruction.

Blood et al. (1983), have reported that complete faecal absence or passage of blood- and mucus-stained faeces is a positive sign of bowel obstruction.

According to Gough (1978), clinical and laboratory data are non-discriminatory against strangulating and non-strangulating obstruction in man.

Johnston (1962), mentioned two main diagnostic features of bowel obstruction in large animals. These are: (1) passage of

small amounts of faeces that are a semisolid mixture of blood and mucus (foetid), foul-smelling, and pasty in consistency; (2) fluid and gas accumulation in the gut loops leading to abdominal distension - elicited as fluid splashes on abdominal ballotment.

Rectal palpation in cattle and the horse is an inevitable necessity in cases of suspected bowel obstruction (Fox, 1970; The Merck Vet. Mannual, 1973; Blood et al., 1983). Pearson and Pinsent (1977), on rectal palpation in cattle with intestinal obstruction found empty rectum, with only small amounts of mucus, and increased intra-abdominal pressure, with distended intestinal loops.

In the dog, Palminteri (1972), pointed out that with much experience, diagnosis of bowel obstruction can be made on abdominal palpation.

Radiography has often been employed as a diagnostic tool in small animals and man suspected to have bowel obstruction (Palminteri, 1972; The Merck Vet. Mannual, 1973; Kingsnorth, 1976; Hornbuckle and Kleine, 1977; Gough, 1978). Radiographic diagnosis of

intestinal obstruction can be made easily if there is presence of radiopaque foreign bodies in the intestine, but it is difficult when foreign bodies are radiolucent, or in cases of volvulus, intussusception and neoplasms with similar density as surrounding tissues (Larsen and Bellenger, 1974).

In man, Kingsnorth (1976), observed air-fluid levels in the bowels on x-ray during bowel obstruction. The author pointed out that diagnosis was normally delayed when no air was present in the bowels, because there was no abdominnal distension, no increased bowel sounds, and no air-fluid level on x-ray. Gough (1978), further pointed out that man with intestinal obstruction may appear radiographically normal.

Laboratory tests in bowel obstruction may not be of much value in diagnosis, but measurement of plasma electrolyte (Sodium, potassium, and chloride ion) concentration can provide useful informations (Palminteri, 1972; Larsen and Bellenger, 1974; Hornbuckle and Kleine, 1977; Blood et al., 1983).

Palminteri (1972) stated that laboratory

data may help to estimate the extent of dehydration, and duration of the obstruction. According to Blood et al. (1983), laboratory data is of value in assessing the severity of the secondary disturbances during bowel obstruction.

Gas and pH analyses; and serum electrolyte values are a helpful guide to therapy of obstruction (Anderson, 1975; Blood et al., 1983).

Confirmation of tentative diagnosis of intestinal obstruction is often made by exploratory laparotomy, especially in acute obstructions like volvulus, intussusception and torsion among others (Palminteri, 1972; The Merck Vet. Mannual, 1973; Larsen and Bellenger, 1974; Pearson and Pinsent, 1977).

Larsen and Bellenger (1974) have cautioned that exploratory laparotomy should be done before the patient's condition deteriorates, and before development of secondary circulatory failure. They have alsc warned that exploratory laparotomy could aggravate the condition of patients that might have other diseases rather than bowel obstruction.

It should be noted here that some cases of acute intestinal obstruction die before diagnosis is made, and therefore it is made only on necropsy.

Blood et al. (1983), have mentioned a few diseases affecting the alimentary tract that should be differentiated from intestinal obstruction. These are: gastric dilatation, pyloric obstruction, abomasal torsion, and non-obstructed bowel loops palpated rectally. The authors also noted that renal, ureteric and urethral problems cause abdominal pain, grunting, and straining, but defaecation is not impeded.

2.4.4. Effect on Blood Parameters

Features of bowel obstruction in ruminants are similar to those observed in simple-stomached animals except for vomiting (Hammond et al., 1964). This means that sequestration of water, electrolytes, and large volumes of intestinal secretions into the gut is the critical pathologic feature in ruminants, rather than their ejection from the animal (Hammond et al., 1964; Coles, 1974).

Corker and Dziuk (1968), while doing experimental work in sheep reported that prevention of the normal flow of abomasal secretions to the duodenum is a primary factor in pathogenesis of ionic changes during acute upper intestinal obstruction in ruminants. These authors further observed that when duodenum was ligated together with omaso-abomasal junction, fluid and electrolyte changes were not as marked as when ligated alone. They also suggested that probably reverse flow of abomasal fluid into rumino-reticulum increases net sequestration of water and electrolytes. According to these authors, fluid and electrolytes that move from the abomasum to the rumino-reticulum during upper intestinal obstruction are not readily absorbed into the blood and other body fluids. They observed that obstruction proximal to the abomasum resulted in slow and less severe water and electrolyte changes as compared to obstruction at duodenal level.

Intestinal strangulation obstructions lead to greater reduction in blood volume than simple intestinal obstructions, and therefore

shock and death ensure at an earlier stage (Sherman, 1981).

According to Michell (1967), tissue damage at the site of the obstruction leads to vascular dilatation with further loss of fluid into the gut lumen. The author suggests that extracellular fluid is lost from plasma into the gut lumen. He observes that loss of fluid and electrolytes during bowel obstruction is similar to their loss during vomiting and diarrhoea.

In the dog, fluid and electrolyte depletion is dependent on proximity of the intestinal obstruction to the pyloric sphincter, severity of vomiting, and sequestration within the distended bowel loops (Twedt and Graver, 1982).

Littlejohn (1965), and Twedt and Graver (1982), observed reduction in total body water, blood volume, and extracellular fluid in dogs and man with intestinal obstruction.

According to Coles (1974), alteration in electrolytes, and acid-base balance seen during any obstruction of the upper intestinal tract in bovine is undoubtedly a consequence

of sequestration of large volumes of abomasal juices. The author reports that when this sequestration occurs, there is almost invariably a hypochloraemia, hypokalaemia and metabolic alkalosis.

There is usually circulatory failure during intestinal obstruction, as a result of decreased blood volume (Hammond et al., 1964).

Fluid and Electrolytes loss lead to death (Jubb and Kennedy, 1970). But Johnston (1962), observed that loss of fluid and electrolytes alone, during intestinal obstruction, without bacterial growth and toxin absorption, does not lead to early death of the patient.

It has been noted by Pearson (1973), that changes in electrolytes and acid-base balance in cattle with intestinal obstruction, or right displacement of abomasum, are similar, and that clinico-pathological indices of dehydration are not markedly changed.

Metabolic alkalosis with significantly high plasma bicarbonate (HCO_3^-) ion and high pH has been observed in sheep with intestinal

obstruction (Corker and Dziuk, 1968; Gingerich and Murdick, 1975). Similar changes have also been observed in cattle (Hammond et al., 1964; Pearson, 1973); dogs (Littlejohn, 1965; Palminteri, 1972); and in man (Littlejohn, 1965). This metabolic alkalosis in ruminants results from sequestration of hydrogen ions into the stomachs due to secretion of chloride ions into the abomasum as Hydrochloric acid (HCL), and removal of chloride (Cl^-) ions from circulation while bicarbonate ions are returned back into the circulation (Hammond et al., 1964 ; Gingerich and Murdick, 1975). In the dog metabolic alkalosis results from loss of chloride ions in gastric juice through vomiting, as well as the build up of bicarbonate ions in the extracellular fluid (Palminteri, 1972).

Metabolic acidosis during intestinal obstruction has been observed in the horse (Littlejohn and Brown, 1963; Littlejohn, 1965; Coles, 1974; Datt and Usenik, 1975); and the Anderson, 1975; Twedt and Graver, 1982). s acidosis may originate from inadequate loride ion absorption from the intestinal tract (Schotman, 1971).

After experimental work of intestinal obstruction (duodenal obstruction and ileal volvulus) in the horse, Datt and Usenik (1975), observed an initial rise in blood pH and bicarbonate ions, and a fall later. The authors attributed the fall in blood pH during ileal volvulus to fluid of intestinal origin being sequestered, rather than of gastric origin, and also intestinal reabsorption ability was lost much earlier. They further attributed the initial pH rise to bicarbonate ion absorption, and failure to reabsorb hydrochloric acid in the stomach. These authors suggest that the fall in blood pH could result from intestinal damage in obstruction, which leads to continous bicarbonate depletion, excess hydrogen (H^+) ion production, and a rise in partial pressure of carbon dioxide (PCO_2). They found out that acid-base balance status in horses with small colonic obstruction remained unchanged.

Schotman (1971), has observed that during caecal and colonic dilatations in cattle, the blood pH remains within the normal range. Hornbuckle and Kleine (1977), while working with the dog, have further noted that acid-

base balance status could be normal in animals with intestinal obstruction. These authors have pointed out the importance of including at least sodium, potassium and chloride ions during electrolyte studies in cases of intestinal obstruction, to assist in differential diagnosis.

According to Pearson and Pinsent (1977), plasma chloride ion level is the most sensitive prognotic aid in cattle with bowel obstruction.

Hypochloraemia during bowel obstruction has been observed in cattle (Hammond et al., 1964; Pearson, 1973; Pearson and Pinsent, 1977); sheep (Corker and Dziuk, 1968; Gingerich and Murdick, 1975); Buffalo (Dass et al., 1981); dog (Littlejohn, 1965; Palminteri 1972; Twedt and Graver, 1982); horse (Datt and Usenik, 1975); and in man (Littlehohn, 1965). Chloride ions are lost in gastric secretions (Jubb and Kennedy, 1970). In ruminants chloride ions are lost as a result of sequestration in the gut as compared to expulsion in man and the simple-stomached animals, therefore leading to hypochloraemic

alkalosis (Littlejohn, 1965; Coles, 1974; Gingerich and Murdick, 1975). Corker and Dziuk (1968), have suggested that in sheep, a practical and reliable indicator of duodenal obstruction when considered in conjuction with clinical features is plasma chloride ion concentration. It has also been observed in bovine by Pearson (1973), and in man and the dog by Littlejohn (1965), that in bowel obstruction, of the electrolyte disturbances, chloride ion has the greatest deviation from normal. Pearson (1973) has also noted that chloride ion level remained low until normal bowel motility resumed, and that the lower the plasma chloride ion concentration, the worse the prognosis. He also observed that below a certain plasma chloride ion threshold level, therapy and surgical intervention of bowel obstruction is useless.

Gingerich and Murdick (1975), while doing experimental work in sheep with duodenal obstruction, have suggested that abomasal distension, and reflux of alkaline buffered secretions from the duodenum to the abomasum serve as stimuli for further hydrochloric

acid secretion from gastric juice, hence there is less hydrochloric acid secretion in pyloric obstruction, and therefore no hypochloraemic alkalosis. These authors have further observed that ruminal mucosa can absorb chloride ions, but concentration gradient is against this process during obstruction. Sherman (1981), has observed hypochloraemia in a goat with duodenal obstruction. Plasma chloride ion level has been found to be normal during colonic obstruction in the horse (Datt and Usenik, 1975); and in bovine with either colonic obstruction, or caecal torsion (Espersen, 1961; Hammond et al., 1964; Dehghani and Townsend, 1982). Cattle with right displacement of abomasum have also been found to exhibit hypochloraemia (Pearson, 1973; Hofmeyr, 1974).

According to Hammond et al. (1964), hypokalaemia is a common finding in metabolic alkalosis. Normally, this is because of the substitution of potassium ions for hydrogen ions during the distal renal tubular sodium ion reabsorption. Hydrogen ion is lost in the exchange. But during alkalosis these

authors have reported that there is general depletion of hydrogen ions, and hence the potassium ion substitution for hydrogen ion during the exchange. This leads to increased potassium loss in urine. The authors have suggested that the general potassium ion loss may also be due to their shift from the extracellular fluid into the intracellular fluid, the gut lumen, or bone. They have further indicated that alkalosis induces potassium ion shift from the extracellular fluid into the intracellular fluid.

Loss of intracellular potassium into extracellular fluid as a result of increased catabolism, injury, or starvation during bowel obstruction and leading to hyperkalaemia has been reported in the horse (Datt and Usenik, 1975). The authors have explained that normally, released intracellular potassium, is quickly eliminated by the kidney, but during bowel obstruction, dehydration causes decreased renal blood flow and reduced renal output, which therefore means that this potassium is retained.

In the bovine, there is usually high

concentration of potassium ions in the ruminal fluid during intestinal obstruction, and therefore potassium ion level deviation is not critical during the disease (Pearson, 1973). Jubb and Kennedy (1970) have reported that in bowel obstruction, potassium ion is lost in gastric eliminations.

During this disease hypokalaemia has been reported in cattle(Hammond et al., 1964; Pearson, 1973); Sheep (Corker and Dziuk, 1968); goat (Sherman, 1981); buffalo with abomasal torsion (Dass et al., 1981); horse with jejunal resection (Littlejohn and Brown, 1963); dog (Littlejohn, 1965; Palminteri, 1972; Twedt and Graver, 1982); and in man (Littlejohn, 1965).

Cases without significant change in plasma potassium ion concentration during bowel obstruction have been reported in sheep (Gingerich and Murdick, 1975); cattle (Hammond et al., 1964; Espersen, 1961; Dehghani and Downsend, 1982); and horses (Datt and Usenik, 1975).

In dogs and man, potassium ion loss in the disease, is due to vomiting (Littlejohn,

1965). In cattle, the loss is due to potassium sequestration (Coles, 1974). Cattle subjected to starvation and thermal stress have been found to have minimal electrolyte (sodium, potassium, calcium and magnesium) changes (Dale et al., 1954).

Elevation of plasma sodium ion levels terminally during intestinal obstructions, has been found in the horse and the rise was not explained (Datt and Usenik, 1975). Loss of sodium ion has been reported in dogs (Littlejohn, 1965; Palminteri, 1972; Twedt and Graver, 1982); and in man (Littlejohn, 1965). Cases without significant change in plasma sodium ion level have been recorded in sheep with duodenal obstruction (Gingerich and Murdick, 1975); in horse with small colonic obstruction (Datt and Usenik, 1975); and in buffalo with abomasal torsion (Dass et al., 1981).

Other plasma electrolytes like calcium ions, magnesium ions etc. are not significantly altered during bowel obstruction (Gingerich and Murdick, 1975; Twedt and Graver, 1982). But in bovine with caecal torsion, lowered

plasma magnesium ion level has been reported (Dehghani and Townsend, 1982).

Uraemia of pre-renal origin due to increased catabolism, reduced renal blood flow, and reduced glomerular filtration, has been reported in bowel obstruction (Jubb and Kennedy, 1970). Blood urea nitrogen (BUN) has been observed to be significantly elevated in sheep (Gingerich and Murdick, 1975); cattle (Pearson and Pinsent, 1977); horse (Datt and Usenik, 1975); and in the buffalo (Krishnamurthy et al., 1980). Horses with small colonic obstruction, were found to have no change in BUN value (Datt and Usenik, 1975). These authors have explained that BUN rise during bowel obstruction, is due to dehydration that leads to decreased renal blood flow, and therefore inadequate renal function. Pearson and Pinsent (1977) have further observed the degree of BUN elevation to be proportional to the degree of dehydration. Blood et al. (1983), have attributed the rise in non-protein nitrogen to gangrenous gut segments.

A rise in packed cell volume (PCV),

erythrocyte (RBC) count, and haemoglobin (Hb) concentration during bowel obstruction has been recorded in the horse (Datt and Usenik, 1975); sheep (Corker and Dziuk, 1968; Gingerich and Murdick, 1975); cattle Hammond et al., 1964); buffalo (Krishnamurthy et al., 1980); dog (Palminteri, 1972); and goat (Sherman, 1981).

Krishnamurthy et al. (1980), doing experimental work of intestinal strangulation obstruction in the buffalo calves, observed initial leucocytosis, then leucopaenia 48 hours postobstruction. The authors also observed neutrophilia. They associated leucocytosis with stress, trauma, and infection in intestinal obstruction. Leucopaenia was associated with outpouring of leucocytes from the distended bowel into the peritoneal cavity. Sherman (1981), observed leucocytosis in a goat with duodenal obstruction.

Haemoconcentration has been associated with dehydration (Palminteri, 1972; Person, 1973; Blood et al., 1983). Haemoglobin concentration and haematocrit may be within normal range during bowel obstruction

(Pearson, 1973). In advanced cases of intestinal obstruction, erythrocyte sedimentation rate has been reported to be increased (Hofmeyr, 1974).

Total plasma protein value has been observed to be elevated during upper bowel obstruction in the horse (Datt and Usenik 1975); and in cattle (Pearson and Pinsent, 1977); lowering of plasma protein value has been reported in buffalo calves with abomasal torsion (Dass et al., 1981). Cases without significant change in serum protein have been observed in buffalo with intestinal strangulation obstruction (Krishnamurthy et al., 1980); cattle with caecal dilatation and dislocation (Espersen, 1961); and in the horse with small colonic obstruction (Datt and Usenik, 1975). According to Pearson and Pinsent (1977), a rise in total plasma protein level is proportional to the degree of dehydration. During rise in total plasma protein concentration, there is usually a significant rise in fibrinogen as has been observed in the horse with obstruction (Datt and Usenik, 1975).

Hypoglycaemia has been reported in buffalo calves with intestinal strangulation obstruction (Krishnamurthy et al., 1980). This hypoglycaemia is associated with toxaemia, and its level of severity is dependent on the degree of toxaemia (Krishnamurthy et al., 1980; Blood et al., 1983). The authors have explained that toxaemia has an effect on carbohydrate metabolism. Hofmeyr (1974), on the other hand has reported that hyperglycaemia is a constant finding in bowel obstruction.

Krishnamurthy et al. (1980), observed significant change in serum alkaline phosphatase in buffalo calves during their experimental work of intestinal strangulating obstruction.

Serum creatinine has been found to have an insignificant rise during experimental work of duodenal obstruction in sheep (Gingerich and Murdick, 1975).

Cattle subjected to starvation and thermal stress were found to have increased concentration of ketone bodies in the blood (Dale et al., 1954), but usually in ruminants, secondary ketosis does not reach a significant degree as to affect the normal functioning of the animals.

2.4.5 Effect on Urinary Parameters

During intestinal obstruction, there is usually a marked decrease in urine output due to reduced renal blood flow, and glomerular filtration, in response to the reduced plasma volume and dehydration (Jubb and Kennedy, 1970; Blood et al., 1983). This reduction in urine output has been reported in sheep with duodenal obstruction (Gingerich and Murdick, 1975); and in the buffalo with intestinal strangulation obstruction (Krishnamurthy et al. 1980).

A marked and significant reduction in the rate of urinary excretion of chloride during duodenal obstruction was reported in cattle (Hammond et al., 1964); and in sheep (Gingerich and Murdick, 1975). Chloride is usually conserved by kidneys during hypochloraemia (Jubb and Kennedy, 1970).

According to Hammond et al. (1964), there is usually loss of potassium in urine during metabolic alkalosis. The authors further explain that normally, renal reabsorption of sodium ions takes place in exchange

with potassium ions, and this results in increased potassium ion loss in urine. But in their experimental work of duodenal obstruction in calves, these authors found a decrease rather than an increase in potassium ion loss in urine.

Gingerich and Murdick (1975), found no change initially until the 48th and 60th hour post-obstruction when they observed a slight decrease in the rate of urinary excretion of potassium, in sheep with experimentally induced duodenal obstruction. It was also noted by the authors that during bowel obstruction, there was depletion of potassium ion in plasma, and that the kidneys try to compensate for the loss by conserving it. Therefore in this case, they explained that hydrogen ion is lost in urine in exchange with sodium ion which is reabsorbed in the distal renal tubules. The authors give this as a reason for the paradoxical aciduria observed at the peak of metabolic alkalosis. They have observed a rise in urinary pH with the onset of metabolic alkalosis, and later it dropped below pH 7. The same authors have pointed out that some undetermined anions like phosphates,

and sulphates from tissue catabolism during starvation and dehydration; as well as lactates and other organic acids from incomplete carbohydrate metabolism; and acetoacetate, from incomplete fat metabolism could contribute to paradoxical aciduria.

The relationship between acidification of urine, and potassium, as well as hydrogen ion has been explained in the dog (Berliner and Kennedy, 1951).

There is usually renal sodium retention during intestinal obstruction (Krishnamurthy et al., 1980). Varied renal excretion of sodium, and a marked decrease in its urinary excretion after the 60th hour post-obstruction has been reported in sheep during experimental duodenal obstruction (Gingerich and Murdick, 1975). The same authors noted that there was no significant change in urinary excretion of calcium, and no ketonuria despite complete anorexia.

2.4.6 Effect on Peritoneal Fluid

Krishnamurthy et al. (1980), have found that in buffalo calves with arterio-venous

strangulation, blood-and plasma-like fluid leaks into the peritoneal cavity from the gangrenous bowel.

Blood et al. (1983), have reported that during occlusion of local blood supply to the intestine, there is usually leakage of fluid form these vessels into the peritoneal cavity and the intestinal wall.

During strangulation obstruction, blood-stained, or serosanguineous exudate has been observed in the peritoneal cavity (Runnells et al., 1965; Jubb and Kennedy, 1970; White, 1981; Blood et al., 1983).

Datt and Usenik (1975), experimentally induced intestinal obstruction in horses and found out that the horses with simple duodenal obstruction had straw coloured peritoneal fluid initially which later became pink, and then deeper pink as the condition progressed; the horses with small colonic obstruction had light-pink to deeper-pink peritoneal fluid; and those with ileal volvulus had light-red peritoneal fluid initially, but became deeper-red, then darker-red terminally. The authors also found ery-

throcytes in the peritoneal fluid, initially following abdominal surgery.

Horses with intestinal infarction due to mesenteric vascular thrombotic disease, but without strangulation obstruction were reported to have yellow or orange peritoneal fluid (White, 1981).

Straw coloured peritoneal fluid with neutrophils, was observed in peritoneal cavities of cattle with caecal torsion (Dehghani and Townsend, 1982).

According to Cohn (1961), during strangulating obstruction, peritoneal fluid is either pink, coagulable, odourless and non-haemolysed; or dark foul-smelling, non-coagulable and haemolysed. The author has reported that chemical, bacterial and spectrophotometric studies indicate similarity of peritoneal fluid and fluid in a strangulated bowel loop during strangulation obstruction. They pointed out that initially, peritoneal fluid is non-toxic, but later it becomes toxic.

2.4.7 Toxic Factors

Under some conditions, intestinal

permeability can be greatly increased and associated with absorption of considerable amounts of endotoxins. (Gans and Matsumoto, 1974). The authors have pointed out that, much of the endotoxins escape into the intestinal lymphatics. These cause systemic manifestation and death because the endotoxins bypass the clearing and detoxifying functions of the liver. The mechanism of this absorption is not elaborate, but probably is by passive diffusion.

Littlejohn (1965), has pointed out that the chief lethal factor in simple bowel obstruction, are bacteria and noxious agents absorbed after damage of the gut wall by intraluminal pressure. This lesion is the second lethal factor after blood loss in strangulating bowel obstruction. The same author has reported that clostridial toxins are the major lethal factors of the peritoneal fluid during intestinal strangulating obstruction in rabbits.

Jubb and Kennedy (1970), have reported that in lower bowel obstruction, wearing of the mucosa as well as bacterial accumulation

leads to escape of endotoxins from the bowel lumen, and this results in toxaemia. They have further reported that in strangulation obstruction, invasion by putrefactive bacteria enhances gangrene formation, which in turn enables passage of bacterial endotoxins through the intestinal wall leading to death.

Distension and stasis of the gut enhances bacterial accumulation and multiplication, as well as toxin production which results in toxaemia, septic shock, and death (Weipers et al., 1964; Michell, 1967; Palminteri, 1972).

Moore et al. (1980), have observed that loss of mucosal barrier allows bacteria and bacterial endotoxin movement across the gut wall, resulting in endotoxaemia and shock.

According to Johnston (1962), loss of fluid and electrolytes alone, without bacterial growth and toxin absorption during bowel obstruction allows the patient more days to survive. The author states that rapid death occurs when bacterial growth and absorption of toxins occur.

Moore et al. (1981), found mucosal degeneration during bowel obstruction to be

coincidental with endotoxaemia. They were able to detect endotoxins in general circulation.

Weipers et al. (1964), have pointed out that necrosis and ulceration of the mucosa increased its permeability to toxins from the obstructed loops. The authors have found Clostridium Welchii A as the only important aetiological factor present in low intestinal obstruction, each time sampled. Administration with Clostruim Welchii A antitoxin, increased chances of survival by 64%. These authors have further reported that in the late stages of intestinal strangulation obstruction, with massive necrosis and little viable tissue in the area, alphatoxin was present in the strangulated loop contents, and to a less extent in the peritoneal fluid, and thoracic duct; but it was not the only toxic component present.

The predominant organisms isolated from strangulated loop fluid during intestinal strangulation obstruction, were Bacteroides sp., Sphaerophorus sp., Clostridum perfringens, Escherichia coli, streptococcus sp., and

peptostreptococcus sp.. Bacteroides sp., E. coli and Clostridium perfringens were the major organisms (Yale, 1969). The author found that blood cultures prior to the intraperitoneal release of the strangulation fluid contained no organisms, while in the later stages, blood cultures were usually positive with several of these predominant organisms.

Peritoneal fluid contained organisms present in the lumen of the strangulated bowel segment. These were haemolytic organisms, E. coli, and non-haemolytic streptococcus (Cohn, 1961). Subsequently, the author found that some of the contents of the strangulated bowel traversed the wall of the bowel without actual perforation into the peritoneal cavity. Then they were absorbed into circulation. Toxic agents present in the strangulated bowel followed the same pattern.

Kobold and Thal (1963), have reported that during intestinal ischaemia, both vasodilator and vasoconstrictor substances are released, but the vasodilators are predominant. The source of these vasodilators

has been suggested to be intestinal contents (major source), intestinal wall, portal veinous blood, and accumulating peritoneal fluid. All these are within, or from the strangulated segment because removal of the strangulated segment brings about a fall in the level of these vasodilators.

The vasodilators that have been identified by these authors are 60-80% polypeptide-like substances, 20% histamine and 10-15% serotonin. Histamine and serotonin are normally present in the gut and therefore are expected to be released in cases of intestinal necrosis. The polypeptide-like vasodilator is thought to be Kallikrein (Kobold and Thal, 1963), or bradykinin (Weipers et al., 1964). It appears from the study of Kobold and Thal (1963), that the polypeptide vasodilator is released from intestinal contents as a result of interaction between bacterial products with plasma and tissue precursors.

Apart from the above named vasodilators, Weipers, et al. (1964), have also reported that there is evidence of an enzyme that releases a slow acting substance from a

a pseudoglobulin, which invades general circulation and causes shock.

There were no catecholamines identified during intestinal ischaemia (Kobold and Thal, 1963).

Peritonitis has also been reported as a factor contributing to fatal termination (Littlejohn, 1965; Weipers, 1965; Jubb and Kennedy, 1970). Sometimes shock and death may occur before the onset of peritonitis (Jubb and Kennedy, 1970).

There has been controversy as to whether the causes of shock during obstruction are neurogenic, cardiogenic, or bacterial factors (Littlejohn, 1965). But a decrease in the effective whole blood or plasma volume, has been agreed upon as one of the causes of shock, the author notes. He lists some factors that contribute to shock. These are: bowel distension, pain, fluid and electrolyte disturbances, bacterial and toxic factors. The author further notes that tourniquet shock may occur during correction of strangulation obstruction if arterial blood supply to the obstructed part is occluded. After

relief of strangulation, there may be sudden and fatal reduction in blood volume and pressure.

Yale (1969), has suggested the causes of death during intestinal strangulating obstruction as haemorrhagic shock due to long section of the gut whose venous return is occluded; products from autolysis of ischaemic or necrotic section or its altered metabolism; contents of the gut (partially digested diet), intestinal enzymes and bacteria; and loss of blood.

2.4.8. Necropsy and Histopathology

Invariably, the segment proximal to the obstruction site is usually dilated and distended with gas and fluid. This fluid is from swallowed saliva, gastric juice, pancreatic juice, biliary juice and parasympathetic stimulation of the intestine, causing secretion from intestinal glands. The gas is from fermentation and swallowed air (Rooney, 1965; Jubb and Kennedy, 1970; Gingerich and Murdick, 1975; Dobson and Lopez, 1981; Koike et al., 1981; Blood et al., 1983). The segment distal to the obstruction is usually

contracted and almost empty (Rooney, 1965; Corker and Dziuk, 1968; Dobson and Lopez, 1981; Sherman, 1981).

Jubb and Kennedy (1970); and Blood et al. (1983), have reported congestion of mucosa and submucosa, oedema of intestinal wall, haemorrhage, necrosis and gangrene of the affected segments during bowel obstruction. These changes have further been reported in goat (Sherman, 1981); horse (White et al., 1980; Rooney, 1965; Runnells et al., 1965); and in bovine with caecal torsion (Wynn Jones et al., 1957).

Necrosis and gangrene are reported to be more common in strangulating obstructions and in these cases the intestine involved appear either dark, blue-red or almost black, and sometimes with wall rupture (Rooney, 1965; Runnells et al., 1965; Jubb and Kennedy, 1970). In these cases the contents appear thin, watery, bloody, or blood-stained in character (Rooney, 1965).

During strangulating obstruction, venous occlusion is usually present, and if strangulation is severe, arterial occlusion as well

occurs (Jubb and Kennedy, 1970). The venous compression causes passive hyperaemia, oedema, haemorrhage and finally necrosis (Runnells et al., 1965).

Jubb and Kennedy (1970), have reported that in chronic obstruction of the large intestine, there is hypertrophy rather than dilatation of the intestinal wall. When obstructive material accumulates in the lumen, the condition becomes acute, with dilatation superimposed on hypertrophy.

Hard dry faecal pellets have been found in the colon of a goat that died of duodenal obstruction (Sherman, 1981); similar findings have been recorded in other species (Jubb and Kennedy, 1970).

Abomasal wall oedema and distension; as well as distension of the forestomachs have been observed in goat (Sherman, 1981); and in sheep (Naerland and Helle, 1962; Gingerich and Murdick 1975) that died of intestinal obstruction.

According to Jubb and Kennedy (1970), when perforation of intestine occurs, peritonitis may result.

Cases that have died of intussusception, may have fibrinous adhesions between the adjacent serosal surfaces due to inflammation (Runnells et al., 1965; Jubb and Kennedy, 1970; Foo et al., 1978).

Bloody fluid has been found in peritoneal cavities of animals that died of intestinal obstruction (Runnells et al., 1965; Jubb and Kennedy, 1970).

Cattle with caecal torsion usually live long enough to allow development of all lesions observed during small intestinal obstruction (Jubb and Kennedy, 1970).

White et al. (1980), induced intestinal strangulation obstruction in ponies and found that 30 minutes post-obstruction, the intestine turned blue in colour; 60 minutes post-obstruction, they turned blue-black in colour, were thickened and lost motility. The colour returned to normal if obstruction was relieved after 60 minutes of obstruction, but cyanosis and decreased motility recurred 120 minutes following this relief. The same authors suggested that intestinal damage should be assessed on the basis of colour, motility,

and arterial pulsation.

When the bowel is infarcted, it usually becomes thickened, suffused, gangrenous and dark with stagnant blood (Jubb and Kennedy, 1970). These infarcts are defined by congestion, subserosal oedema, and overlying peritonitis.

Carcases that have died of intestinal infarction without strangulation have been found to have yellow or orange peritoneal fluid (White, 1981). The author has classified lesions associated with infarction as ileal intussusception, ileal impaction, jejunal incarceration, and large colon impaction.

Moore et al. (1980), created experimental intestinal strangulating obstruction in ponies to evaluate the effect of intraluminal administration of oxygen, and they found that the intestinal had spastic contractions immediately after the intestinal strangulating obstruction (ISO) instead of the normal peristalsis. Some ponies, were left with intestinal strangulation obstruction for 50 minutes, and out of these, the intestines that were not treated with oxygen were purple in colour, had no motility, had thickened walls and continued to

have mucosal cell degeneration 120 minutes after release of the intestinal strangulation obstruction, while those treated with oxygen were pink in colour, had motility and thickened walls, but showed no further mucosal cell degeneration on light microscopy 120 minutes after relief of the intestinal strangulation obstruction. But on scanning electron microscopy (SEM), there was evidence of microvilli disruption and erythrocyte effusion between mucosal cells. Other ponies were left with intestinal strangulation obstruction for 90 minutes before any oxygen treatment or relief of the obstruction. Out of these the intestine treated with oxygen were blue in colour, had no motility, had thickened walls, and continued to have full mucosal degeneration. The authors further observed that intraluminal oxygen administration, prevented mucosal degeneration only if the villus lesion had not progressed beyond the stage of loss of epithelial cells from the tips and minimal haemorrhage into the lamina propria.

According to White et al. (1980), ponies with small intestinal strangulation obstruction, had all sections of the intestine cranial (unaffected parts) to obstruction, normal histologically. On further histological evaluation 30 minutes after intestinal strangulation obstruction, there was slight separation of epithelial cells from lamina propria at the tips of villi. Sixty minutes after intestinal strangulation obstruction, there was loss of epithelial cells from the tips of villi, and minimal haemorrhage into the lamina propria.

There was extension of the subepithelial space exposing $\frac{1}{3}$ to $\frac{1}{2}$ of the lamina propria, and haemorrhage in the lamina propria and the submucosa, 120 minutes after intestinal strangulation obstruction. Finally after 180 minutes, all the above changes were present, with addition of complete separation of epithelium from the lamina propria to the villus base, marked lamina propria haemorrhage, as well as submucosal haemorrhage and oedema. According to these authors, the transition of the lesion between 30 minutes and 60 minutes of intestinal strangulation

obstruction is effected by fluid accumulation in the lateral intercellular space which causes build up of pressure at the basal epithelial cell attachment, resulting in their detachment from the lamina propria. They further explain that cells lift off in sheets due to their tight junctional complexes.

Reduced rate of blood flow through the villus, and increased oxygen uptake time at the base of the villus, as well as the long distance for oxygen diffusion from the base of the villus to the tip, are the factors that cause the villus tip to receive hypoxic blood. Therefore sloughing of epithelial cells (mucosal cell degeneration) starts at the villus tips (Moore et al., 1980; White et al., 1980).

Fluid filling the submucosal space may be from the intestinal lumen (White et al., 1980). The authors have attributed intestinal wall thickening to oedema production, and have explained the continued mucosal degeneration after relief of intestinal strangulation obstruction as being consequential to local hypoxia which causes vasodilation, and hence

reduced blood flow in the mesenteric microcirculation. This according to the authors increases the time for blood perfusion of the villus, and therefore counter current exchange of oxygen between the villus central arteriole, and the surface descending capillaries, takes longer. This in turn allows hypoxic blood to reach the villus tip. Mucosal degeneration continues irrespective of the macroscopic judgement of the intestinal appearance.

Moore et al. (1981), found that mucosal degeneration with loss of villus epithelium occured coincidentally with endotoxaemia, during intestinal strangulation obstruction.

According to Barclay et al. (1980), mucosal necrosis is a consistent finding in horses with intestinal volvulus, and this is always a grave prognotic sign.

Foo et al. (1979), have observed submucosal oedema and inflammation in man with small intestinal obstruction.

Horses dying of intestinal obstruction were found to have carcasses in good body condition (Dobson and Lopez, 1981). Sheep

carcass was found to be emaciated (Naerland and Helle, 1962).

Rooney (1965), reporting on autopsy findings in horse carcasses with small intestinal volvulus observed that lesions in other organs, and tissues were non-specific, and related to asphyxiation. These were: poorly clotted, dark blood; haemorrhages in the nasal mucosae, in the cervical tissues, in the subcutaneous tissue, in the epicardium and pleura; hyperaemia of lungs, and failure of lungs to collapse (alveolar emphysema). Oedema of the lungs was present only when autopsy was delayed.

Fibrinous pneumonia was observed in a sheep carcass that had died of duodenal obstruction (Gingerich and Murdick, 1975).

3. MATERIALS AND METHODS

3.1 Experimental Animals

Twenty one Masai goats were used for this project. Eighteen of these were used for the actual experimental work. The other three were used only for gross and histopathological control. Of the eighteen goats, twelve were female and six were male.

All the goats were stall-fed for five weeks before the actual experimental work was started. During this period, their health was monitored regularly by checking on temperature, pulse and respirations; as well as observing the animals generally to see if they showed any signs of illness. Laboratory analysis of blood, faeces and urine was done regularly. They were fed on dry hay and maize bran[1/]. Water and mineral blocks[2/] were always available

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1/ Maize bran - Unga Ltd., Industrial area, Nairobi.

2/ Maclick^{R} - Twiga Chemical Industries Ltd., P.O. BOX 30172, Nairobi.

in the stalls. The animals were dewormed twice with Nilzan^{R} 3/ during this five week period. No signs of disease were observed in any animal during this period.

3.2 Experimental Groups

The goats were divided into 3 different groups of six. Each group was subjected to obstruction of different levels of the intestine.

3.2.1 Group A - simple duodenal obstruction

This group consisted of 6 female goats ear-tagged A1, A2, A3, A4, A5, and A6. They were subjected to simple duodenal obstruction at varying levels from the duodenopyloric junction. The levels from the duodenopyloric

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3/ Nilzan^{R} - (Cooper) Wellcome Kenya Ltd.,
Private bag
Kabete

junction were randomly selected with no particular criterion for their selection. A1 and A2 were obstructed at 10 centimeters (cm), A3 and A4 at 20 cm, and A5 and A6 at 30 cm from the duodenopyloric junction.

3.2.2. Group B - jejunal strangulating obstruction

This also consisted of 6 female goats ear-tagged B1, B2, B3, B4, B5, and B6. Each was subjected to a condition simulating strangulating obstruction of the jejunum. Lengths of jejunum involved in the strangulation obstruction ranged from 1-2 metres (m).

3.2.3. Group C - ileocaecocolic strangulating obstruction

In this group were 6 male goats ear-tagged C1, C2, C3, C4, C5 and C6. A condition simulating strangulating obstruction of ileocaecocolic area, or ileocaecal intussusception was induced in each of these animals.

3.3. Experimental Methods

Experimental methods used in this project were adopted from those used in the horse (Datt and Usenik, 1975); and in calves (Hammond et al., 1964).

3.3.1 General Procedures for all the Groups

Each goat was restrained manually (no chemical used) on its left lateral recumbency and the legs tied onto the operation table with strings. The right paralumbar fossa was prepared for aseptic surgery by shaving the hair over it and doing three scrubs with hibiscrub[R4/] and finally applying 95% methylated spirit on the area (Fig. 1).

4/ Hibiscrub[R] - Chlorhexidine gluconate- ICI Ltd., Alderley Park, Macclesfield, Cheshire, Gt. Britain.

Fig. 1. Method of Manual restraint of the goats by tying the limbs and the head with strings onto the surgical table.

15 millilitres (ml) of lignocaine[R] 5/ (2% lignocaine hydrochloride) was infiltrated vertically along the midpoint of the right paralumbar fossa for a length of about 12cm. Another small area in the ventral midline between xiphoid cartilage area and the area of the umbilicus was prepared for aseptic surgery in a similar manner to that described above. 2 ml of lignocaine[R] was infiltrated in this area (Fig. 2).

A surgical cloth-drape was placed over the prepared right paralumbar fossa and held in position with towel-clamps. A 10 cm skin incision was made in the paralumbar fossa vertically along the infiltration line. The laparotomy incision was then made by transecting abdominal muscle and the peritoneum (Fig. 3).

Two stab incisions penetrating through skin and body wall into the peritoneal cavity, were made 2 cm anterior to the skin incision on the paralumbar fossa, about 5 cm apart (Fig. 3).

5/ Lignocaine[R] - Sunways (India) PVT. LTD., Bombay, India.

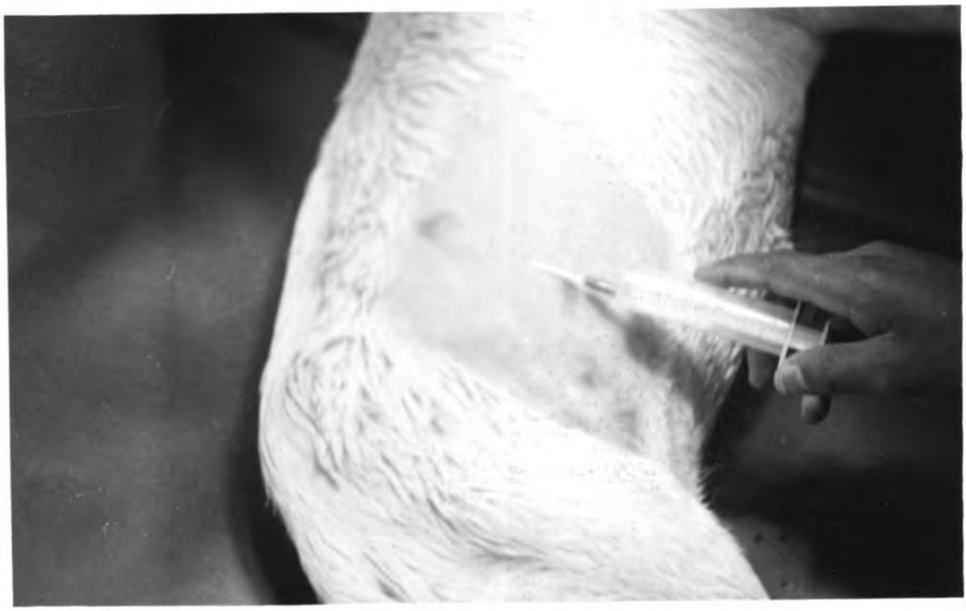

Fig. 2. Infiltration of lignocaine hydrochloride (local analgesic) along the proposed line of incision.

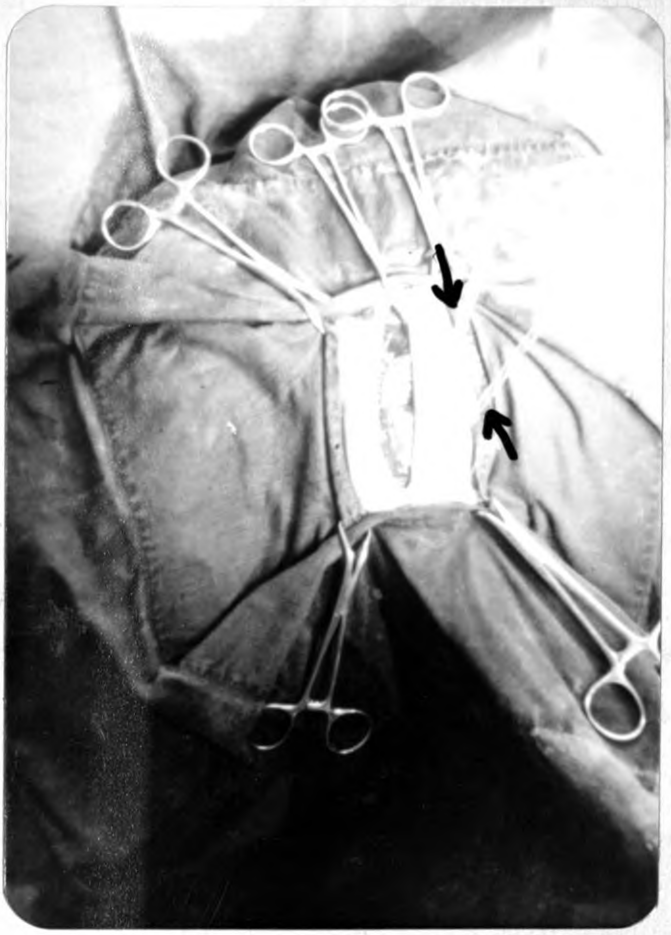

Fig. 3. Complete laparotomy incisions, and two stab incisions through which the plastic-tubing ends are exteriorized. (Arrows - Plastic-tubing).

Another stab incision also through the skin and body wall and penetrating into the peritoneal cavity, was made in the area prepared on the ventral midline. A short y-shaped plastic tubing[6/] (from the disposable infusion set), was introduced into the peritoneal cavity through the laparotomy incision to the ventral midline stab incision. The long end of the y-piece tube was exteriorized by grasping it with a mosquito forceps introduced from outside through the stab incision. A clip was then applied to the exteriorized end to keep the lumen of the y-piece tube blocked, so that no unwanted peritoneal fluid could flow out (Fig. 4 & 5). The y-piece tube was secured in position by placing a purse-string suture on the skin to constrict the opening, using number (#) 0 nylon. This tube was used for sampling of the peritoneal fluid during the experiment.

6/ Plastic tubing - Behring Institute - Germany

Fig. 4. Plastic tubing used for ligation of the intestine; plastic sheet onto which the plastic ligature was tightened; and the y-piece tubing and the clip used for peritoneal fluid collection.

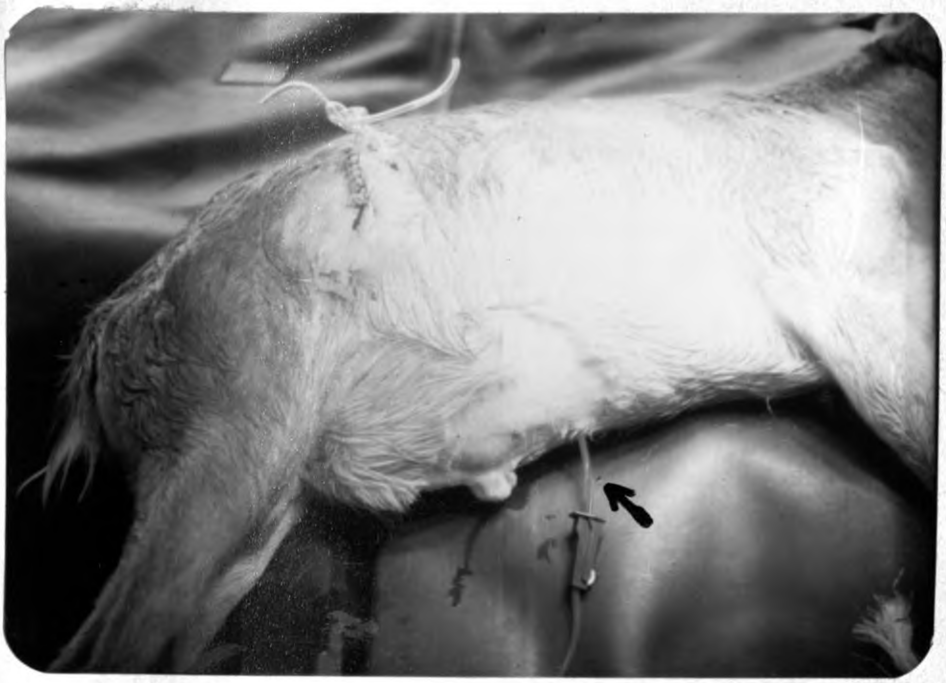

Fig. 5. Y-piece tubing and the clip in position for peritoneal fluid collection. Blood is seen in the lumen of the tubing immediately post-surgery. (Arrow)

3.3.2 Group A

For each goat in this group, the duodenum was identified by exploration, and exteriorization through the laparotomy incision. About 50 cm length of the plastic tubing (from the disposable infusion set), was placed round the duodenum in a double-loop, through an opening made in the mesentery with a mosquito forceps (Fig. 6). The double-loops were made at the levels specified earlier, from duodenopyloric junction for each animal.

A pair of forceps was introduced through one of the stab incisions in the paralumbar fossa inorder to grasp and exteriorize one of the ends of the double-looped plasting tubing (Fig. 6). The pair of forceps was also introduced through the second stab incision, to grasp and exteriorize the remaining end of the plastic tubing.

Holes corresponding to the two paralumbar fossa stab incisions, were made through a thick plastic sheet[7/] measuring about 5 cm. by 10 cm. The two exteriorized ends of the

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7/ Plastic sheet - Vifor S.A. Geneva - Suisse.

double-looped plastic tubing were introduced through these holes in the plastic sheet respectively (Fig. 6). The plastic sheet prevented the plastic tubing from severing the skin once its ends were tightened. The ends of the plastic tubing were then loosely tied together, inorder not to cause duodenal obstruction before the set time.

3.3.3 Group B

Jejunum of each goat in this group was identified by exploration and exteriorization through the laparotomy incision. Two holes apart, were made through the jejunal mesentery near the roots of the mesenteric vessels, to include lengths of jejunum within the range of 1-2m. The segment of the jejunum between these mesenteric holes was isolated by passing the ends of a 60 cm. long plastic tubing, through the two holes respectively. The two ends of the plastic tubing were then passed round the jejunum corresponding to them, in a double-loop similar to that of the duodenum (Fig. 6). The reason for passing the plastic tubing through two mesenteric

holes was to cause strangulation of the mesenteric blood vessels, as well as luminal occlusion of the jejunum between these two holes when the tubing is tightened.

The ends of the double-looped plastic tubing were then exteriorized and tied loosely in a similar manner to group A.

3.3.4. Group C

Abdominal exploration was done for each goat in this group, and the caecum was identified and exteriorized through the laparotomy incision. The ileo-caeco-colic junction was then identified.

Two holes similar to those in group B (Fig. 6) were made through the mesentery so as to incorporate the caecum, distal segment of ileum, proximal part of colon, and the ileocaecocolic junction between them.

The area incorporated was isolated by placing a 60 cm. long plastic tubing in a similar manner to group B. The reason was to create strangulation obstruction of ileocaecocolic area, or to simulate a condition similar

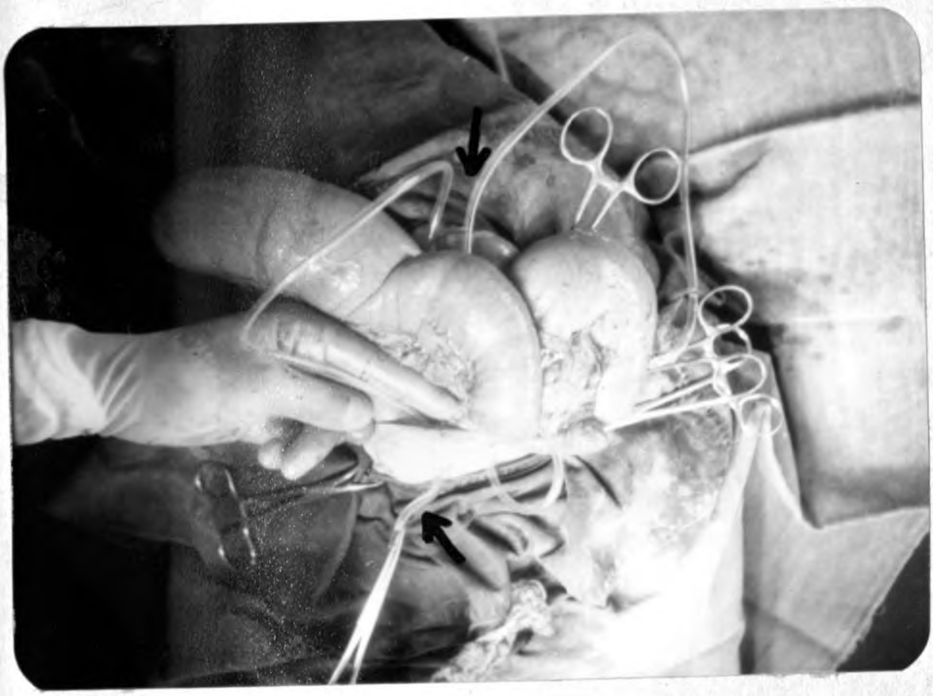

Fig. 6. Double-looped plastic-tubing immediately after its placement through the mesentery and round the intestine.

(Upper arrow - double looping round the intestine)

(Bottom arrow - Plastic-tubing end).

to ileocaecal intussusception once the plastic tubing was tightened. The ends of the plastic tubing were exteriorized and tied together loosely as in group A.

For each goat in all the groups, the laparotomy incision was closed in two layers. The peritoneum, transverse abdominal muscle, and oblique abdominal muscles were closed together with #1 chromic catgut in a cruciate suture pattern, and oversewn with a continous cushing pattern using the same suture material. The skin and the subcutis were closed together with #0 nylon in a blanket-stitch pattern.

3.3.5. Controls

The period up to 48 hours preobstruction, starting immediately after surgery, was considered as the control period for each animal. All the parameters and samples taken from each goat during this preobstruction period served as control values. Thus each animal served as its own control pre-obstructively.

For each goat in all groups, intestinal obstruction was caused by tightening the

exteriorized loose ends of the double-looped plastic tubing 48 hours postsurgery. During this 48 hour preobstruction period, the condition of the animals was left to stabilize and to recover from surgical stress without using any medication. The stability of their condition was determined by resumption of normal appetite, rumination, defaecation, temperature, respiration and pulse.

Paremeters and values from samples taken just prior to tightening of the ends of the plastic tubing, were used as baseline values.

Three independent goats were used for gross and histopathological control. From these, a goat was used for each group respectively. Each of these goats was subjected to the same procedures as the goats in the experimental group it represented, except that the plastic tubing was never tightened. In this case, no intestinal obstruction or strangulation was caused. The three goats were sacrificed after the average survival time for each respective group had elapsed. Necropsy was performed and the carcases were examined for any gross pathological changes.

Intestinal samples were collected in 10% buffered formalin and stored at room temperature to fix for histopathology.

3.3.6. Clinical Features:

Parameters of the physical condition of all the animals were monitored and recorded every six hours during the control (pre-obstructive) and the post-obstructive period until the death of each animal.

The clinical parameters monitored were: rectal temperature; heart rate per minute; respirations per minute; colour of the oral, ocular, or vulval (where applicable) mucous membranes; demeanour; appetite for hay and grains; general body condition; body hydration using skin pliability, capillary refill time, sinking of the eyes, moistening of mucous membranes and cooling of the extremeties; defaecation and character of faeces; water intake; sweating; ruminal motility per minute; intestinal motility or sound; abdominal distention; abdominal ausculatation and ballotment; rumination; signs of pain; posture of the animals; and survival time.

3.3.7 Blood Analysis

Blood samples about 10 c.c. were collected from the jugular vein every 12 hours during pre-obstruction and postobstruction period.

Blood for haematology was collected in EDTA[8/] bottles and analysed immediately or within 12 hours from collection time. Blood for biochemical analysis was collected in glass tubes free of anticoagulant and left to stand at room temperature to clot. Serum was then separated after the clotted blood-serum mixture was spinned in a centrifuge[9/] at 5,000 r.p.m. for 10 minutes. The serum was then frozen for later analysis.

The haematological analysis was done on erythrocyte (RBC) count in millions per cubic millimetres (mm^3); haemoglobin (Hb) concentration in grams per cent (gm%); mean corpuscular volume (PCV) in percentage, and total

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8/ EDTA - Ethylene-diamine tetra-acetic acid sodium salt - Howse and McGeorge Ltd., P.O. BOX 72030, Nairobi.

9/ Centrifuge - MSE minor centrifuge made in England.

leucocyte (WBC) count in thousands per cubic millimetres.

The RBC, and WBC were obtained by the use of an electronic coulter counter[10/]. The MCV was obtained by use of MCV calculator[10/]. Hb concentration was obtained by the use of a haemoglobinometre[10/]. PCV and total plasma protein (TP) were measured from a small amount of blood spinned in a capillary tube with a microcapillary centrifuge[11/] at 12,000 rpm for 5 minutes to separate blood cells from plasma. The PCV was then read from a micro-haematocrit[12/] reader, and the TP was read in grams per 100 ml from a refractometer[13/].

Differential leucocyte count (DLC) was obtained by making a blood smear on a microscope slide and staining it with 1:5 Giemsa

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10/ Coulter electronics, Inc., 590W. 20th St., Hialeah, Florida, 33010.

11/ Microcapillary centrifuge - Model M_B - International equipment Co., Boston, Mass (U.S.A.)

12/ Microhaematocrit reader - Hawksley, England (1869)

13/ Atago SPR - TZ - Japan

stain. The smear was then examined with a light microscope[14] at a x1000 magnification under oil emersion. The different leucocytes were counted by use of a blood-cell calculator[15].

Blood biochemical analysis was done from serum. Albumin in grams per 100ml. was obtained by use of a photometer[16]. Blood urea nitrogen (BUN) was measured in milligrams (mg) per 100 ml. by use of urea nitrogen test strips[17].

Chloride in milliequivalent per litre (mEq/L) was analysed in a chloridometer[18].

14/ Ernst Leitz GMBH Wetzlar
Typ. 020 - 441.003. (Germany)

15/ Blood-cell calculator - The Marbel blood-calculator Co., 30W. Washington St. Chicago 2,Ill., U.S.A.

16/ Photometer - Eppendorf Geratebau Metheler - Hinz. GMBH Germany

17/ Urastrat^{R} - General diagnostics. Morris Plains, N.J. 07950 (Ireland)

18/ Chloridometer - Buchler instruments, Inc., Fort Lee, N.J., U.S.A.

Sodium and potassium in mEq/L were obtained by use of a flame photometer[19]. All the results obtained from blood analysis were recorded for statistical analysis.

3.3.8 Peritoneal Fluid Analysis

Peritoneal fluid about 3 c.c. was collected in EDTA bottles through the y-piece tube on the ventral midline, every 12 hours during preobstruction and postobstruction period.

RBC and WBC count was done similar to blood analysis above and recorded. Colour and transparency of the fluid were observed. Oduor was also noted.

3.3.9 Urine Analysis

Urine collection was done every 12 hours preobstructively and postobstructively. This was achieved by adhering clean plastic surgical gloves over a cleaned vulval area in the female (Fig. 7a, 7b & 7c), or preputial area

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19/ Flame photometer - Evans Electroselenium Ltd., Halstead, Essex. England.

in the male with an elastic bandage. The finger part of the gloves was cut off, and the opening left was kept tightly sealed by tying it with a string. The string was untied each time urine was being collected. After each collection, the gloves were removed for cleaning, and clean ones replaced back into position.

The urine was then analysed and results recorded. Colour, transparency and quantity of urine were noted each time.

Sodium, potassium and chloride were analysed in a similar manner as described for their analysis in serum.

Glucose in mg/100 ml. was analysed using clinistix^{R} 20/ reagent strips, and the results read after 10 seconds.

Protein in mg/100 ml. was analysed by use of Albustix^{R} 21/ reagent strips.

20/ Clinistix^{R} - Ames Co., Elkhart, Indiana U.S.A.

21/ Albustix^{R} - Ames Co., Stoke Poges, Slough Bucks, England.

Fig. 7a. Surgical gloves used as a device for urine collection.

Fig. 7b. Surgical gloves in position over the vulva ready for collection of urine from the female goat.

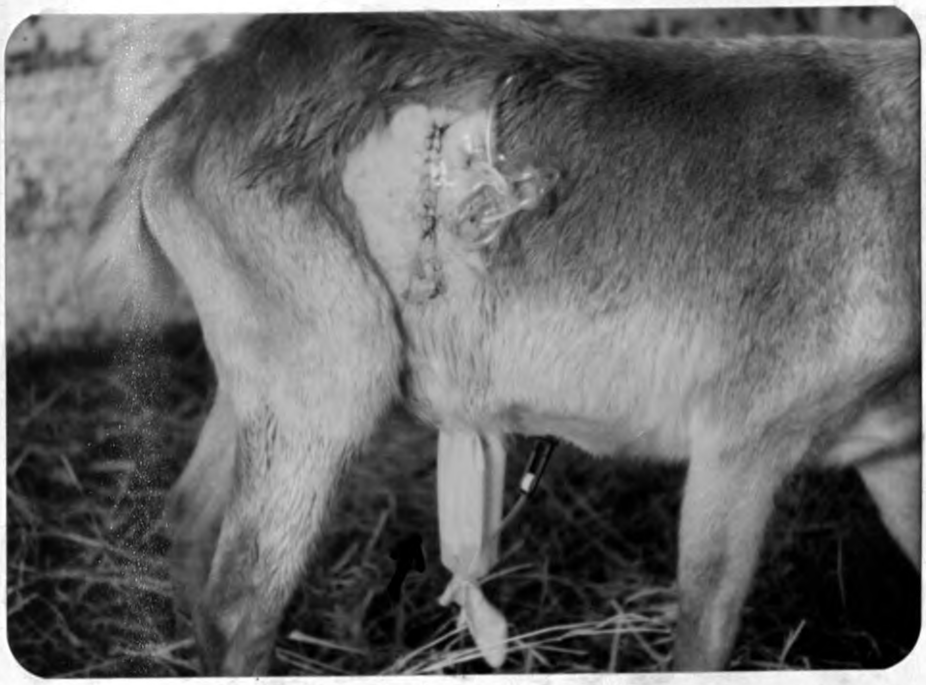

Fig. 7c. Surgical gloves in position over the prepuce ready for collection of urine from the male goat.

Ketone bodies and hydrogen ion concentration (pH) were analysed using Labstix[R] 22/ reagent strips.

3.3.10. Necropsy

Necropsy was performed on each goat carcass immediately, or a few hours after death. The general condition of the carcass, and gross pathological changes on the gastro-intestinal tract, as well as on other internal organs were evaluated and the result recorded.

Intestinal samples from the site of intestinal obstruction, from cranial and distal to the site were taken and preserved in 10% buffered formalin at room temperature to fix for histopathological examination later.

3.3.11. Histopathology

Both transverse and longitudinal sections, were cut from the formalin-fixed intestinal samples. They were mounted on blocks. Sections 5 microns (μ) were sliced from the

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22/ Labstix[R] - Ames Co., Australia Pty. Ltd., Melbourne Australia.

blocks, and mounted on microscope slides, then stained with haematoxylin and eosin (H/E) stain.

Histological interpretation was grouped into 6 grades after White et al., (1980) as follows:

Grade 0 - normal intestinal tissue

Grade I - slight separation of epithelial cells from the lamina propria at the tip of the villus.

Grade II - Loss of epithelial cells from the tip of the villus, and minimal haemorrhage into the lamina propria.

Grade III - extension of the subepithelial space exposing one third to one half of the lamina propria, and haemorrhage into the lamina propria and submucosa.

Grade IV - complete separation of epithelium from lamina propria to the villus base with marked lamina propria haemorrhage, and submucosal haemorrhage and oedema.

Grade V - loss of villus architecture and early necrosis of the crypt cells.

Any other change that is not covered in the above grading, was also noted. All the histological changes were recorded for comparison and evaluation.

4. RESULTS

4.1. General Observations

The animals were in good body condition, bright and had good appetite for both hay and grains (maize Bran) before and after surgery.

Normal defaecation with soft faecal pellets was present in all the animals both before and after surgery. Twenty four hours after obstruction, most of the animals in the three groups strained during defaecation, and passed hard and dry faecal pellets, or soft mucoid, or blood-tinged faeces. Terminally, defaecation ceased totally, in all the animals.

Intestinal hypomotility occured immediately after surgery. But normal intestinal motility was present 48 hours after the surgery. A few animals in all the groups had intestinal hypermotility immediately after obstruction. However, as all the animals approached death, they had either intestinal hypomotility, or intestinal stasis, as well as ruminal stasis. Intestinal motility was estimated by auscultation, but ruminal motility by measuring cycles per minute.

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The animals became dehydrated after obstruction, and this was more marked in animals from group A apparently because they survived longer. On abdominal ballotment, all animals had splashing fluid sounds, with animals in group A having more fluid than those in the other groups.

The animals became severely emaciated gradually and were finally unable to stand.

The animals in group A survived longest, while those in group B survived shortest. There were however few exceptions in groups A and C. One animal in group A had a ruptured duodenum after obstruction, caused by the tightened plastic tubing ligature. This animal survived longest (i.e. 122 hours). Two animals in group C, survived extraordinarily longer than the rest of the animals in the same group. These two survived 109 and 99 hours respectively.

4.2. Clinical Features

All the animals looked dull and depressed after surgery (before obstruction), but 48

hours after the surgery, they had all become bright and active. They became dull again after 24 hours of obstruction in group A, 12 hours in group B and 18 hours in group C. They were all in a coma terminally.

Temperatures varied irregularly within the limits of normal values, before obstruction in the three groups. The temperature dropped markedly from the 12th hour after obstruction, till death of the animals in group B (Fig. 8). In groups A and C, the temperature dropped but not to the same extent as in group B.

The heart rate in group A was progressively elevated from the 12th hour of obstruction. It reached the peak with a mean value of 161 beats/minute respectively at the 18th hour of obstruction. The heart rate then dropped in a similar manner to group A.

There was no difference in respiratory rate between the preobstruction and post-obstruction period in the three groups (Fig. 10). Expiratory groaning and grunting were present terminally, in all groups.

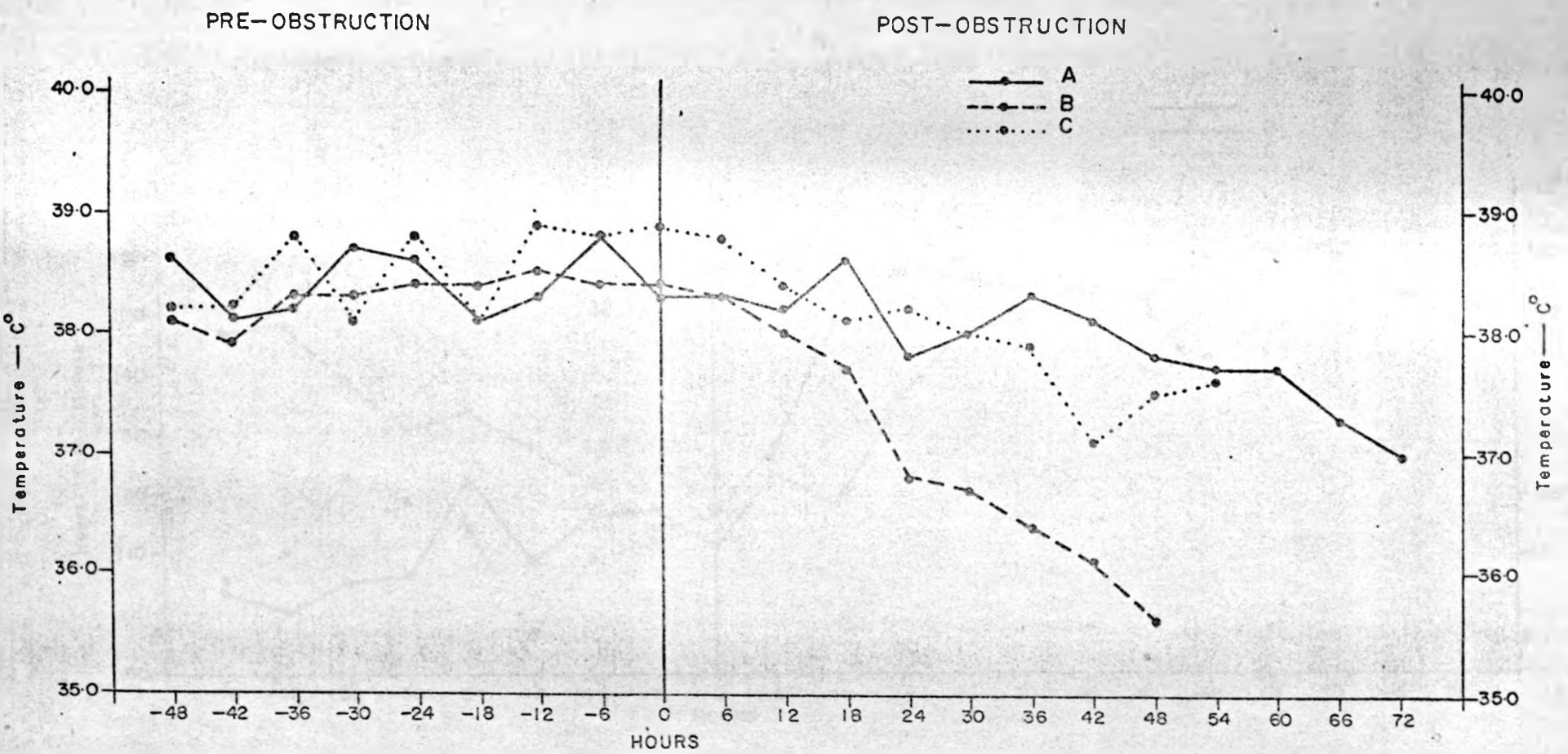


Fig. 8. Comparative mean temperatures in degrees Celcius (°C) for groups A, B and C before and after obstruction.

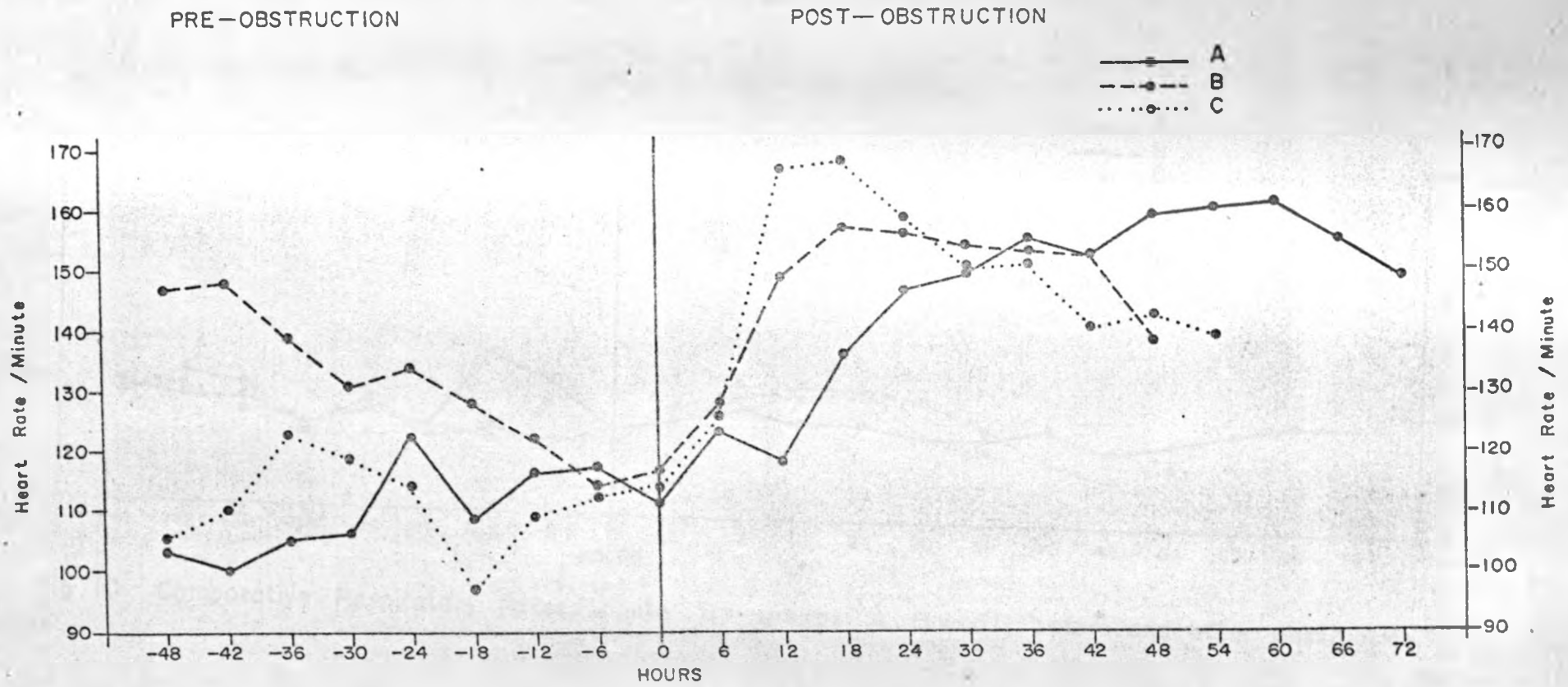


Fig. 9. Comparative mean heart rates/minute for groups A, B and C before and after obstruction.

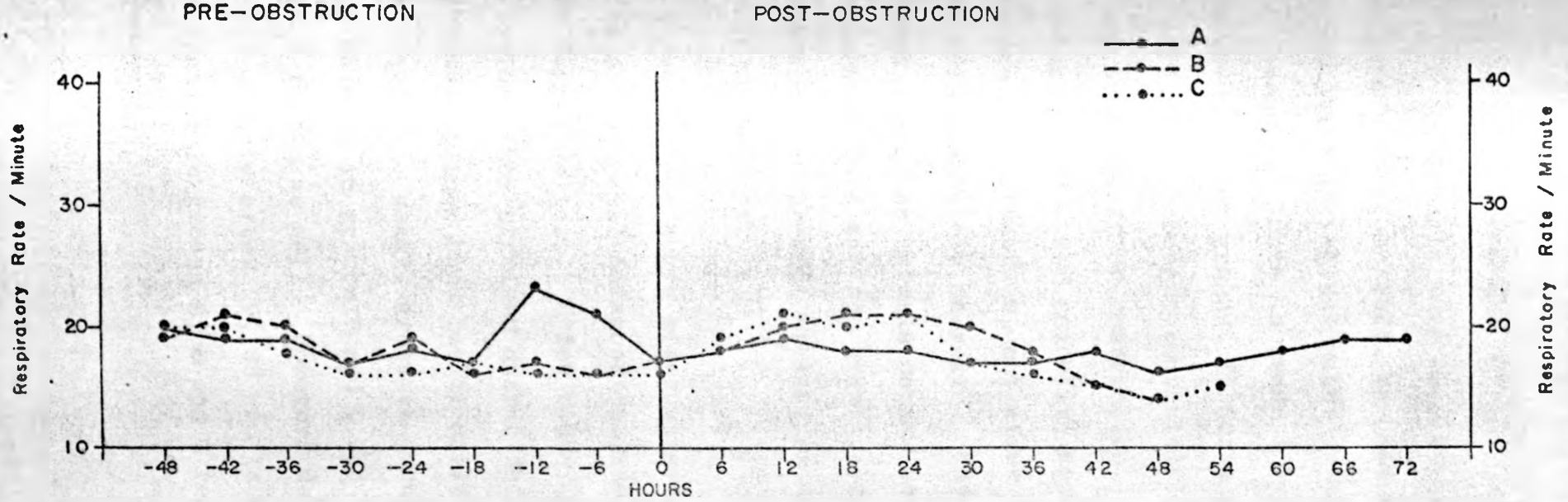


Fig. 10 Comparative Respiratory Rates/Minute for groups A, B and C before and after obstruction.

All the animals were completely anorexic immediately after surgery, but had regained good appetite and normal water intake 24 hours after surgery. Those in group A had selective appetite only for grains, 18 hours after obstruction, and became completely anorexic 48 and 60 hours after obstruction. The animals in groups B and C had selective appetite after 10 hours of obstruction, but after 18 hours, they were completely anorexic. Water intake stopped after the animals became very weak and recumbent. This was not quantitatively monitored. Rumination was present as long as the animals continued feeding.

Twenty four hours after surgery, all the animals had normal defaecation, and passed soft faecal pellets. A few hours after obstruction, they strained during defaecation, and passed either hard faecal pellets, or foul-smelling scanty greenish-mucoid faeces. Some animals in groups B and C had blood-tinged faeces as well. However, defaecation ceased totally after 60, 30 and 18 hours of obstruction in groups A, B and C respectively.

The colour of the visible mucous membrane was pink and dry after 60 hours of obstruction in group A. In groups B and C, it became pale but still moist terminally, with an exception of those that survived longer than 60 hours of obstruction, whose mucous membrane became dry.

Immediately after surgery, the animals in the three groups were grinding their teeth as a sign of pain, but this had already ceased 24 hours after the surgery. Signs of pain (grinding of teeth, restlessness, rising up and lying down, bleating), appeared immediately after tightening the plastic tubing, thus causing obstruction, in groups B and C. However, there was no sweating in any of these animals. There were no signs of pain after obstruction in group A.

Sounds of intestinal motility were reduced after surgery in all the groups, but 48 hours later normal motility was evident on auscultation. Most of the animals had intestinal hypermotility immediately following obstruction, but terminally, they all had either intestinal hypomotility, or intestinal stasis.

Ruminal motility was also reduced following surgery in all the groups, but was normal rate 48 hours later. Some animals in group A had ruminal hypermotility at one stage or another during obstruction, but all had ruminal hypomotility after 66 hours of obstruction. In groups B and C, the animals had either ruminal hypomotility, or stasis after the 12th hour of obstruction.

In group A, signs of severe clinical dehydration were evident during obstruction. These were: delay of the skin, to return to normal position after picking it, long capillary refill time, drying of the muzzle, drying of the mucous membrane, sunken eyes, and weak and fast pulse. Groups B and C had moderate dehydration, evidenced by poor skin elasticity and delayed capillary refill.

On abdominal ballotment, the animals in group A had splashing fluid sounds 48 hours after obstruction. The abdomen was symmetrically distended especially at the terminal stages. Animals in groups B and C had splashing fluid sounds after 30 and 36 hours of obstruction respectively, but no abdominal

distension.

Animals in group A, lost their body condition progressively and finally they became very emaciated and weak. Those in groups B and C were in fairer body condition, but they were weak and their bony prominences visible. The animals however, were still able to stand, but became recumbent after 60 hours of obstruction in group A, and after 30 hours in groups B and C.

The animals in group A survived longest, with an average survival time of 92 hours, while those in group B survived shortest with an average survival time of 53 hours. Group C had an average survival time of 70 hours. See Table 1 below.

Group	Average Survival time (hrs)	Maximum Survival time (hrs)	Minimum Survival time (hrs)
A	92	122	74
B	53	62	49
C	70	109	51

Table 1 - Survival time in hours for groups A, B and C.

4.3. Blood Analysis

Paired t-test was done on the mean values of blood parameters to find whether there were any significant differences from the controls. Analysis of variance for comparison of the effect of the different sites (i.e. group comparisons) and duration of obstruction on blood parameters, was done for values from 48 hours preobstruction to 48 hours post-obstruction. The ANOVA tables are included in the appendices.

4.3.1. Blood Sodium

There was a significant ($P<0.05$) rise in sodium ion concentration 36 hours after obstruction in group A, and it continued to the terminal stages. In group C, there was a significant ($P<0.05$) fall 24 hours post-obstruction which also continued to the terminal stages. No significant change in sodium concentration was observed in group B (Tables 2, 3 & 4; and Fig. 11, 12 & 13).

There was significant ($P<0.01$ and 0.05) difference in sodium values between the three

TABLE 2 - BLOOD VALUES: MEANS ± SD

Group A

	PRE-OBSTRUCTION (CONTROL) PERIOD	BASELINE VALUES	POST-OBSTRUCTION PERIOD					
Time (hrs)	-48 to -12	0	12	24	36	48	60	72
Na^+ (mEq/L)	143.83±3.60	143.67±1.97	143.50±1.97	145.33±7.09	148.00±5.76*	152.00±8.22*	156.50±8.71*	154.66±9.67*
K^+ (mEq/L)	3.88±0.68	3.50±0.55	3.83±0.75	3.67±0.52	3.50±0.55	3.67±0.52	4.00±0.63	3.33±0.82
Cl^- (mEq/L)	102.42±4.84	102.17±4.45	88.17±13.64*	88.33±12.55*	74.33±10.39*	70.33±8.76*	65.67±5.85*	60.67±5.01*
BUN (mg/100ml)	13.3 ±4.08	13 ±4.08	27 ±12.02*	36 ±14.97*	47 ±34.78*	58 ±29.63*	72 ±22.01*	99 ±32.95*
Plasma Protein (gm% ml)	7.53±0.77	7.47±0.85	7.97±0.98	8.15±1.02	8.50±1.55	8.87±1.70	8.75±1.64	8.63±1.71
Albumin (gm/100 ml)	3.03±0.31	3.23±0.21	3.12±0.25	3.21±0.79	3.04±0.66	3.29±0.57	3.59±0.37*	2.37±0.24
PCV (%)	30 ±3.64	31 ±1.10	33 ±4.38	39 ±3.29*	42 ±3.78*	44 ±3.82*	47 ±3.33*	47 ±1.72*

TABLE 2 (continued)

	PRE-OBSTRUCTION (CONTROL) PERIOD	BASELINE VALUES	POST-OBSTRUCTION PERIOD					
Time (hrs)	-48 to -12	0	12	24	36	48	60	72
RBC ($x10^6/mm^3$)	15.70± 2.23	14.42± 1.40	15.97± 1.58	15.65± 2.09	16.23± 1.78	16.28± 2.35	17.28± 3.60	18.68± 5.01
Hb (gm%)	11.20± 1.33	11.6 ± 1.63	11.7 ± 1.44	11.3 ± 2.17	12.0 ± 2.47	11.9 ± 2.28	12.5 ± 2.93	12.4 ± 2.44
WBC ($x10^3/mm^3$)	11.90± 0.92	12.00± 0.99	12.22± 0.81	13.68± 2.21	21.83± 7.02*	23.17± 9.02*	24.60±11.45*	26.33±14.73*
DLC (%) N	71±12.84	70±10.23	77± 9.46	77±10.89	78± 6.85	77±13.84	77±13.84	78±14.44
L	29±12.84	30±10.23	23± 9.46	23±10.89	22± 6.85	23±13.84	23±13.84	22±14.44
M	0	0	0	0	0	0	0	0
B	0	0	0	0	0	0	0	0
E	0	0	0	0	0	0	0	0

* Significant difference from control values at 5% significant level

SD Standard deviation
DLC Differential leucocyte count

TABLE 3 - BLOOD VALUES: MEANS' ± SD

Group B

	PRE-OBSTRUCTION (CONTROL) PERIOD	BASE-LINE VALUES	POST-OBSTRUCTION PERIOD			
Time (hrs)	-48 to -12	0	12	24	36	48
Na^+ (mEq/L)	138.29±6.54	139.17±4.75	141.33± 1.97	144.00± 3.52	144.67±10.86	138.00±6.45
K^+ (mEq/L)	4.67±0.48	4.17±0.41	4.33± 0.52	3.67± 0.52*	3.50± 0.55*	3.67±0.52*
Cl^- (mEq/L)	101.29±3.74	99.83±2.14	99.00± 6.42*	86.83± 7.28*	77.33± 0.82*	68.67±4.32*
BUN (mg/100ml)	12±2.41	12±2.58	20±11.81	35±16.12*	49±18.73*	58±9.83*
Plasma Protein (gm/100ml)	7.23±0.63	7.73±0.72	7.53± 0.43	7.65± 0.46	7.45± 0.77	7.55±0.63
Albumin (gm/100ml)	2.85±0.51	3.13±0.41	3.28± 0.50	3.16± 0.51	3.28± 0.24	3.19±0.19
PCV (%)	30±3.75	30±3.66	32± 3.69	35± 4.03	38± 5.54	38±6.77*
RBC ($X10^6/mm^3$)	13.72±2.77	13.64±2.86	16.04± 3.88	18.47± 6.32	23.99± 5.19	25.65±4.56*
Hb (gm%)	9.9 ±1.05	9.9 ±0.97	10.1 ± 1.53	10.5 ± 1.26	12.0 ± 1.86*	12.4 ±2.11*
WBC ($X10^3/mm^3$)	12.44±0.98	12.67±1.17	16.05± 1.71*	20.70± 3.08*	24.22± 3.08*	30.90±5.65*

Table 3 - continued

		PRE-OBSTRUCTION (CONTROL) PERIOD	BASE-LINE VALUES	POST-OBSTRUCTION PERIOD			
Time (hrs)		-48 to -12	0	12	24	36	48
	N	67±8.04	71±5.00	77±1.72*	76±5.74*	77±3.54*	83±3.72*
	L	33±8.04	29±5.00	23±1.72*	24±5.74*	23±3.54*	17±3.72*
DLC(Z)	M	0	0	0	0	0	0
	B	0	0	0	0	0	0
	E	0	0	0	0	0	0

* Significant difference from control values at 5% significance level.

SD standard deviation

DLC Differential leucocyte count

TABLE 4 - BLOOD VALUES: MEANS ± SD

Group C

	PRE-OBSTRUCTION (CONTROL) PERIOD	BASELINE VALUES	POST-OBSTRUCTION PERIOD			
Time (Hrs)	-48 to -12	0	12	24	36	48
Na^+ (mEq/L)	144.1 ±4.23	147.3 ±5.61	140.3 ±4.97	137.7 ±4.46*	135.3 ±163*	134.0 ± 2.53*
K^+ (mEq/L)	3.1 ±0.51	2.9 ±0.41	2.9 ±0.33	3.0 ±0.65	2.7 ± 0.24	3.0 ± 0.56
Cl^- (mEq/L)	110.3 ±4.30	108.5 ±3.51	96.3 ±1.86*	93.8 ±2.23*	89.5 ± 1.22*	88.8 ± 2.04*
BUN (mg/100ml)	16±2.32	17±4.08	16±3.76	25±5.48*	35± 7.75*	38±10.37*
Plasma Protein (gm%)	7.1 ±0.92	6.7 ±0.52	6.4 ±0.54	6.7 ±0.63	6.9 ± 0.77	7.1 ± 1.17
Albumin (gm/100ml)	2.52±0.52	2.63 ±0.67	2.58±0.55	2.49±0.50	2.82± 0.87	3.01± 0.62
PCV (%)	27±4.42	26 ±4.59	32±5.44	31±6.13	32± 6.51	32± 7.19
RBC ($X10^6/mm^3$)	9.51±1.14	9.05 ±1.81	11.24±1.71	11.09±1.83	11.00± 2.36	11.45±11.77*
Hb (gm%)	9.2 ±1.68	8.8 ±1.78	11.2 ±2.08	11.2 ±2.60	10.8 ± 1.88	11.3 ± 2.61
WBC ($X10^3/mm^3$)	11.65±1.09	11.18 ±1.40	14.52±4.10	16.42±5.40	17.40± 7.98	17.60± 7.59

Table 4 - (continued)

	PRE-OBSTRUCTION (CONTROL) PERIOD	BASELINE VALUES	POST-OBSTRUCTION PERIOD			
Time (hrs)	-48 to -12	0	12	24	36	48
N	62±14.55	65±13.59	74±18.64	74±12.04	74±12.40	78±13.34
L	38±14.55	35±13.59	26±18.64	26±12.04	26±12.40	22±13.34
DLC (%) M	0	0	0	0	0	0
B	0	0	0	0	0	0
E	0	0	0	0	0	0

* Significant difference from control values at 5% significance level.

SD standard deviation

DLC differential leucocyte count

Fig. 11 - Serum sodium concentration in mEq/litre, and rate of urinary excretion of sodium in mEq/hour before and after simple duodenal obstruction.

I - represents ± standard Errow of the mean (SEM).

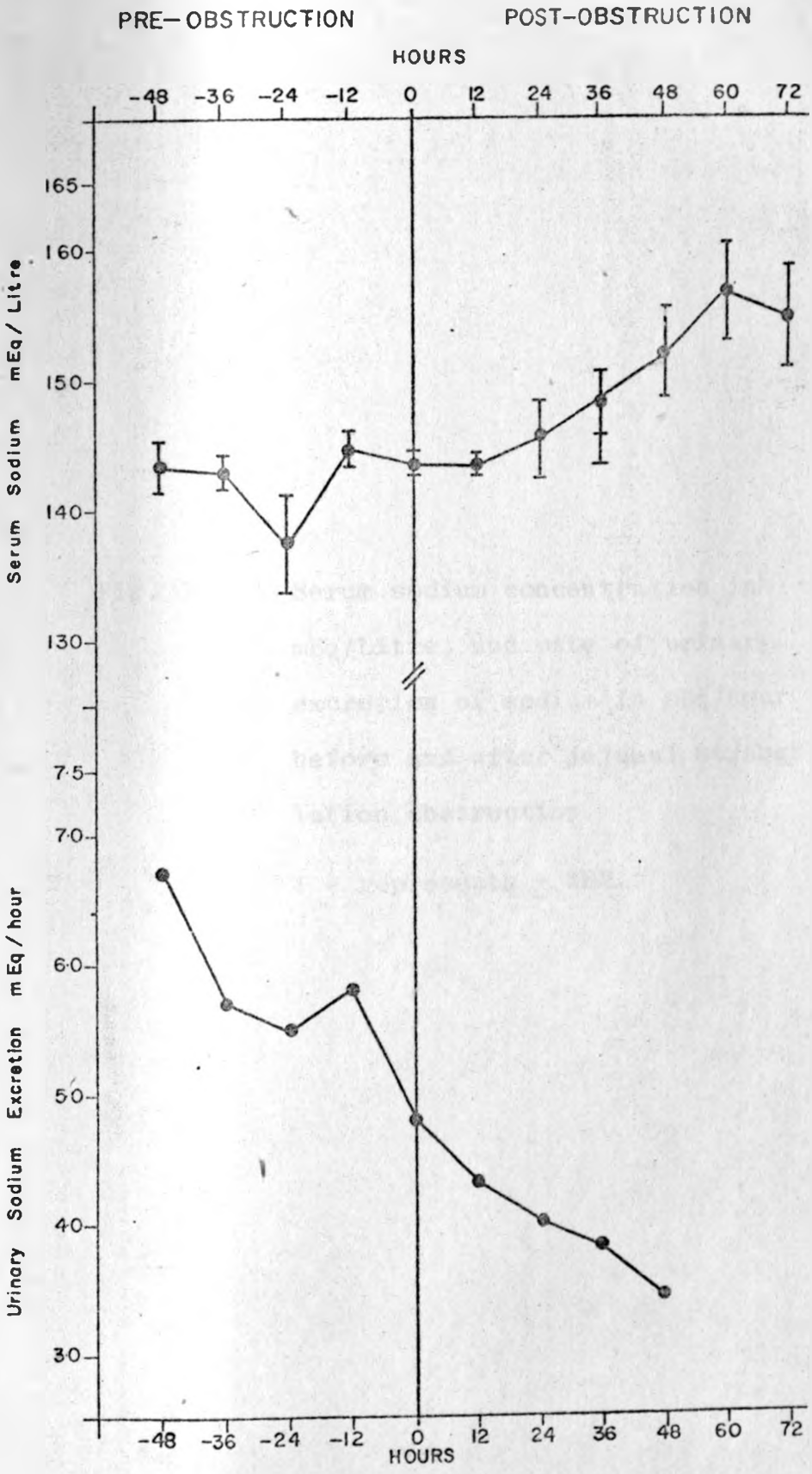

PRE-OBSTRUCTION
POST-OBSTRUCTION
HOURS
-48 -36 -24 -12 0 12 24 36 48 60 72
Serum Sodium mEq/Litre
165
160
150
140
130
Urinary Sodium Excretion mEq/hour
7·5
7·0
6·0
5·0
4·0
3·0
-48 -36 -24 -12 0 12 24 36 48 60 72
HOURS

Fig. 11.

Fig. 12 - Serum sodium concentration in mEq/Litre, and rate of urinary excretion of sodium in mEq/hour before and after jejunal strangulation obstruction.

I - represents ± SEM.

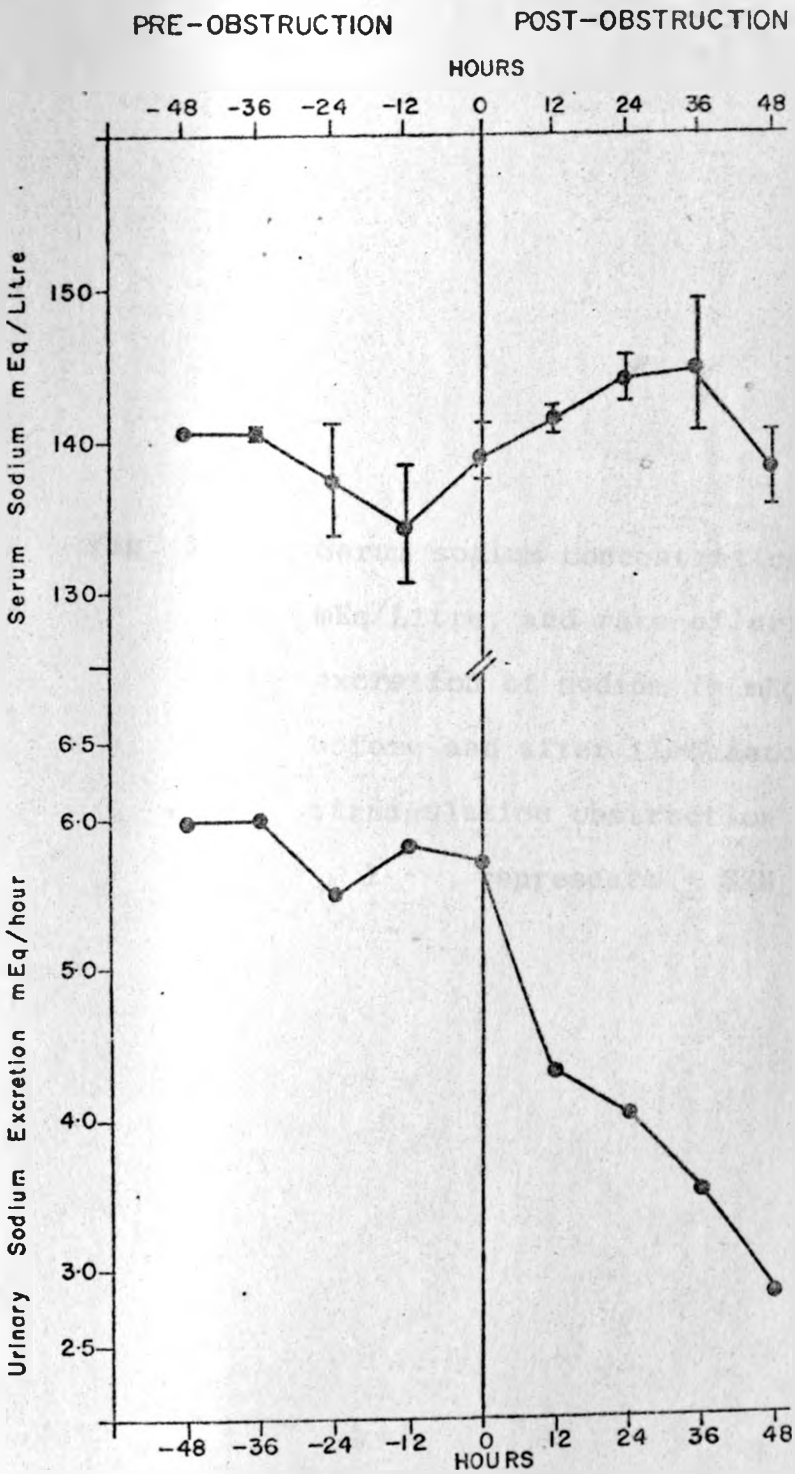

PRE-OBSTRUCTION
POST-OBSTRUCTION
HOURS
-48 -36 -24 -12 0 12 24 36 48
Serum Sodium m Eq / Litre
150
140
130
Urinary Sodium Excretion m Eq/hour
6·5
6·0
5·0
4·0
3·0
2·5
-48 -36 -24 -12 0 12 24 36 48
HOURS

Fig. 12.

Fig. 13 - Serum sodium concentration in mEq/Litre, and rate of urinary excretion of sodium in mEq/hour before and after ileocaecolic strangulation obstruction

I - represents ± SEM

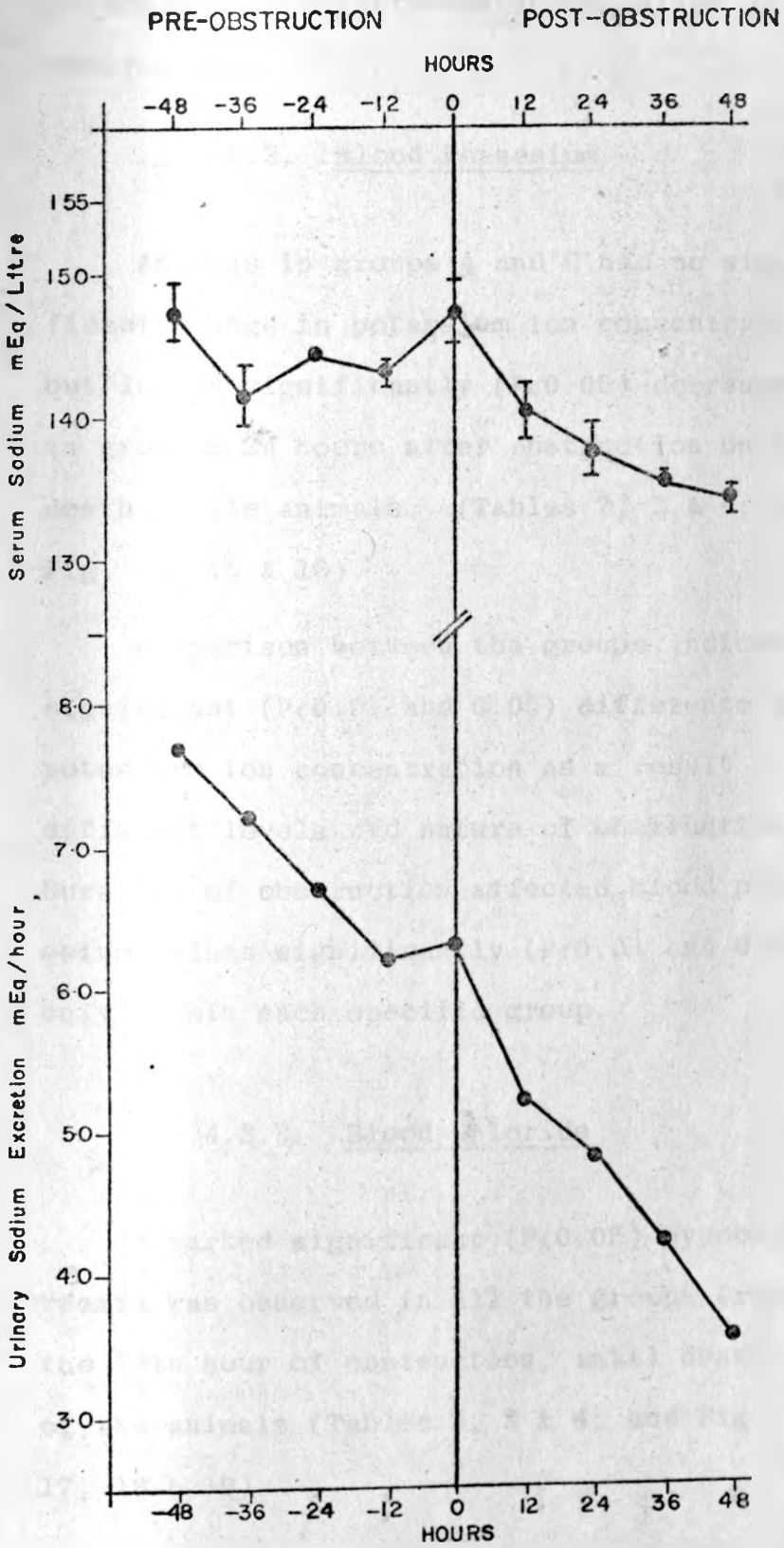

PRE-OBSTRUCTION
POST-OBSTRUCTION
HOURS
-48 -36 -24 -12 0 12 24 36 48
155
150
140
130
Serum Sodium mEq/Litre
8·0
7·0
6·0
5·0
4·0
3·0
Urinary Sodium Excretion mEq/hour
-48 -36 -24 -12 0 12 24 36 48
HOURS

Fig. 13.

groups due to differences in the sites of obstruction.

4.3.2. Blood Potassium

Animals in groups A and C had no significant change in potassium ion concentration, but it was significantly ($P<0.05$) decreased in group B 24 hours after obstruction until death of the animals. (Tables 2, 3 & 4; and Fig. 14, 15 & 16).

Comparison between the groups indicated significant ($P<0.01$ and 0.05) difference in potassium ion concentration as a result of different levels and nature of obstruction. Duration of obstruction affected blood potassium values significantly ($P<0.01$ and 0.05), only within each specific group.

4.3.3. Blood Chloride

A marked significant ($P<0.05$) hypochloraemia was observed in all the groups from the 12th hour of obstruction, until death of the animals (Tables 2, 3 & 4; and Fig. 17, 18 & 19).

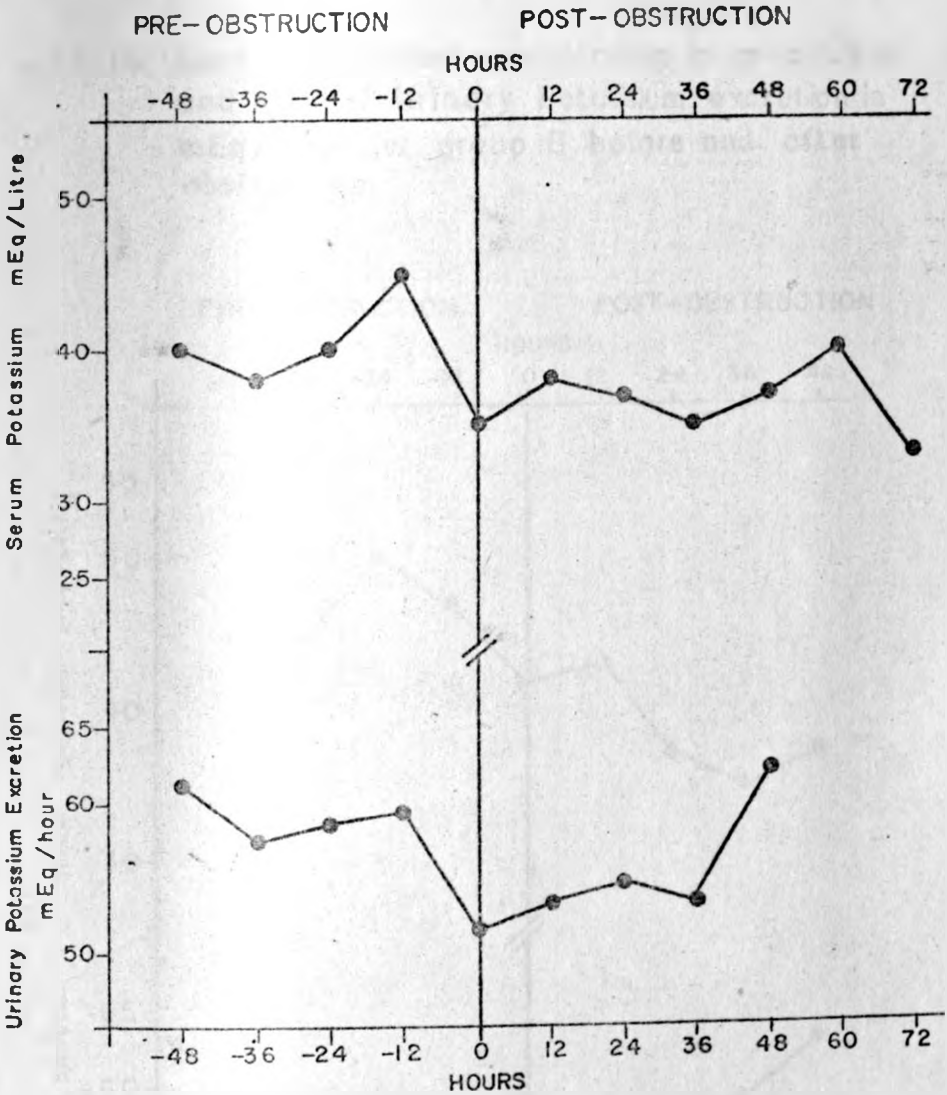


Fig.14 Serum Potassium concentration in mEq/Litre and rate of Urinary Potassium excretion in mEq/hour for group A before and after obstruction.

Fig.15 Serum Potassium concentration in m Eq/Litre and rate of Urinary Potassium excretion in mEq/hour for group B before and after obstruction.

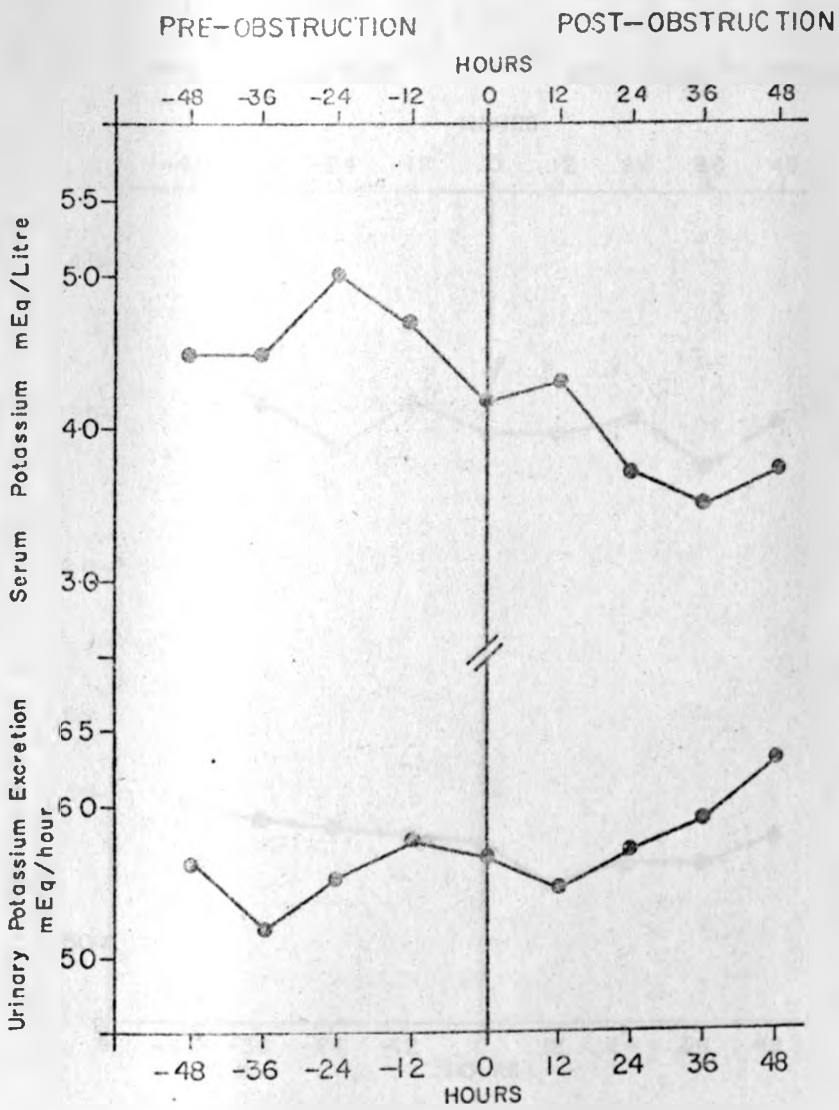
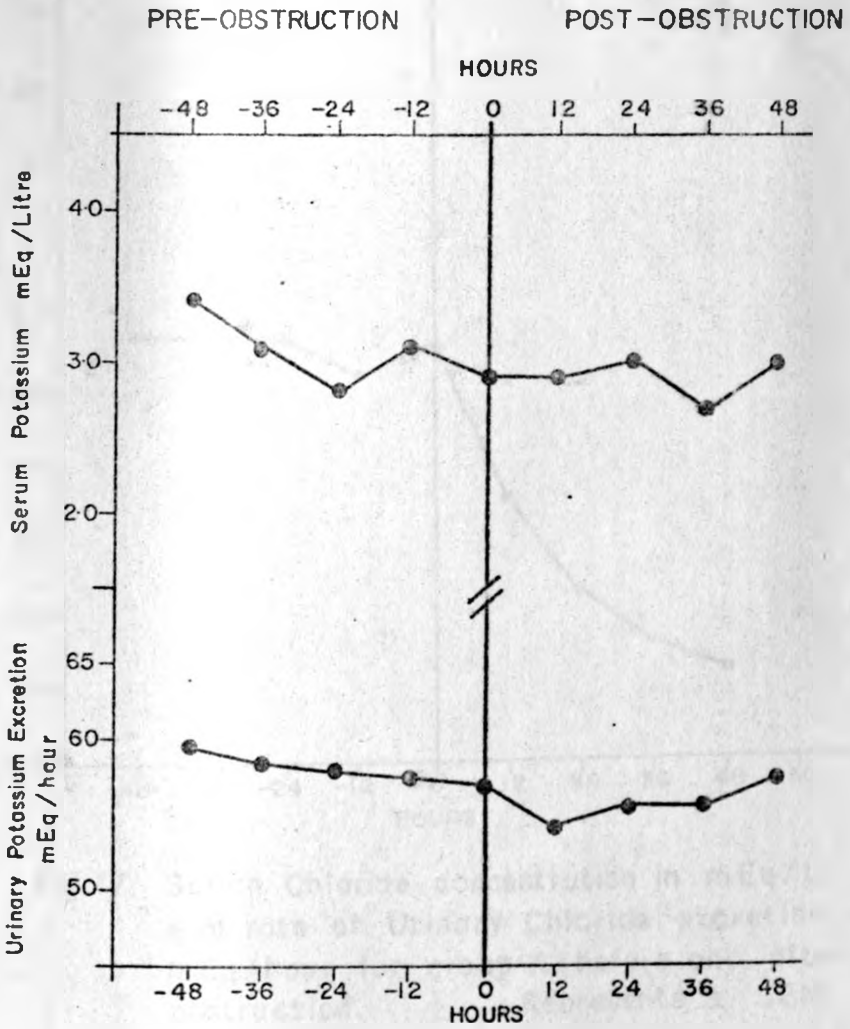


Fig.16 Serum Potassium concentration in mEq/Litre and rate of Urinary Potassium excretion in mEq/hour for group C before and after obstruction.



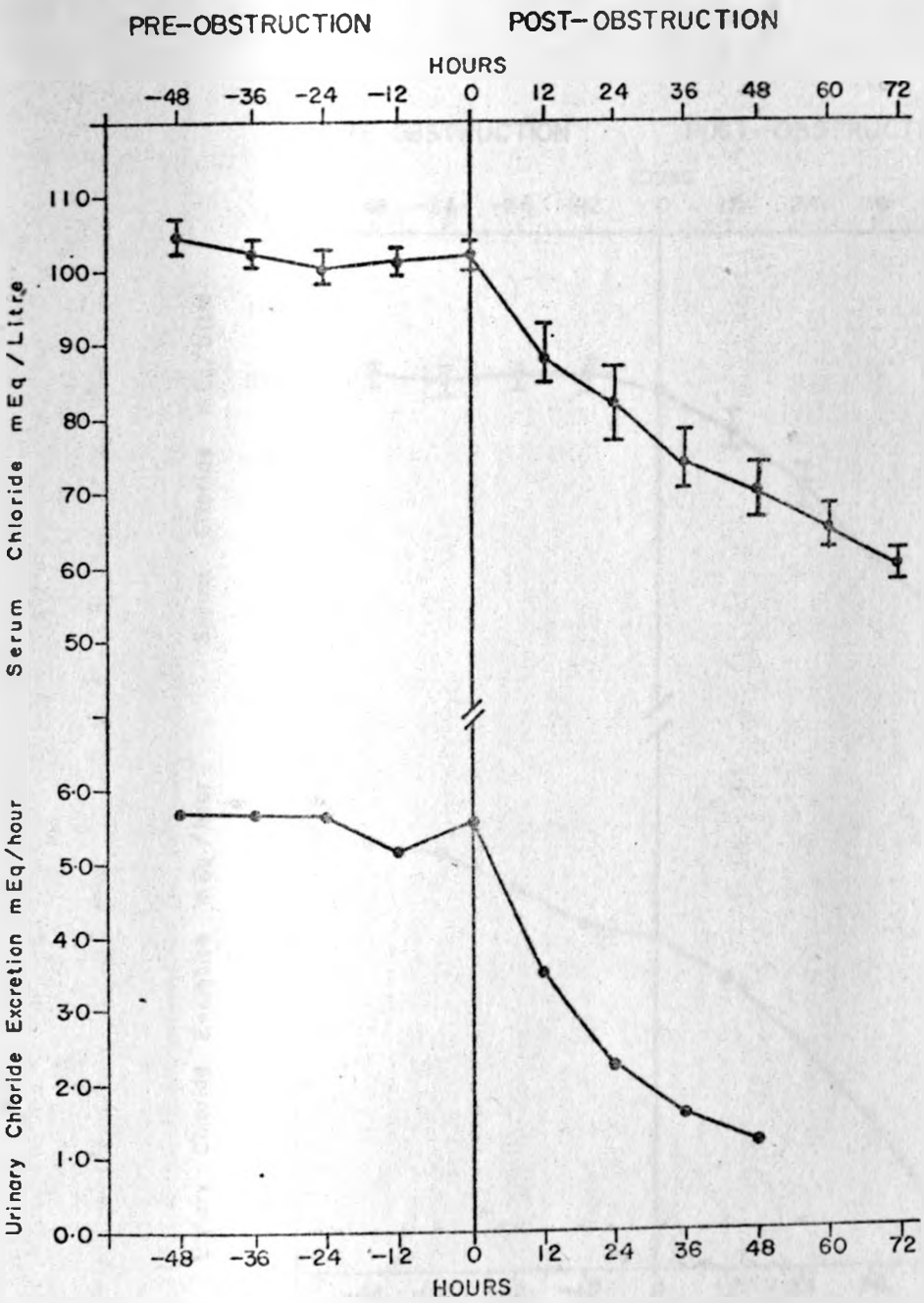


Fig.17 Serum Chloride concentration in mEq/Litre and rate of Urinary Chloride excretion in mEq/hour for group A before and after obstruction. ⊥ Represents ± SEM.

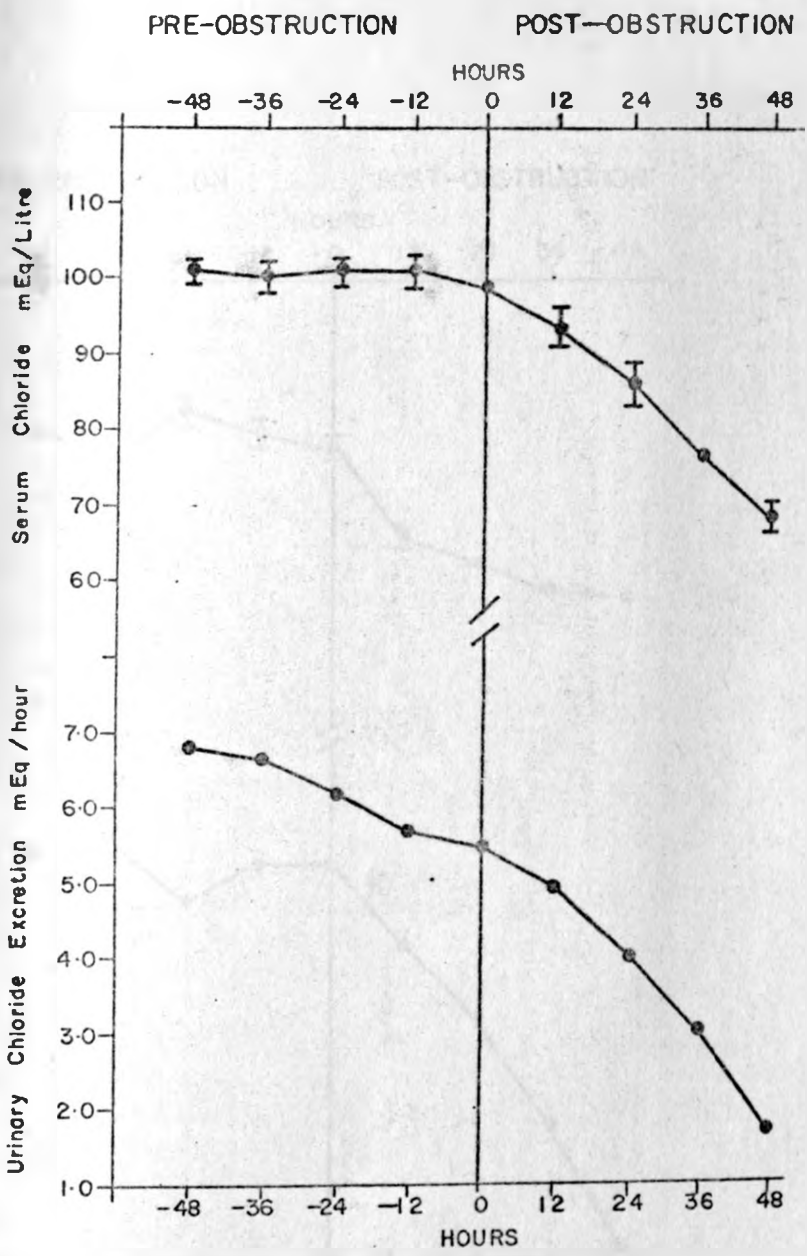


Fig. 18 Serum Chloride concentration in mEq/Litre and rate of Urinary Chloride excretion in mEq/hour for group B before and after obstruction. ⊥ Represents ± SEM.

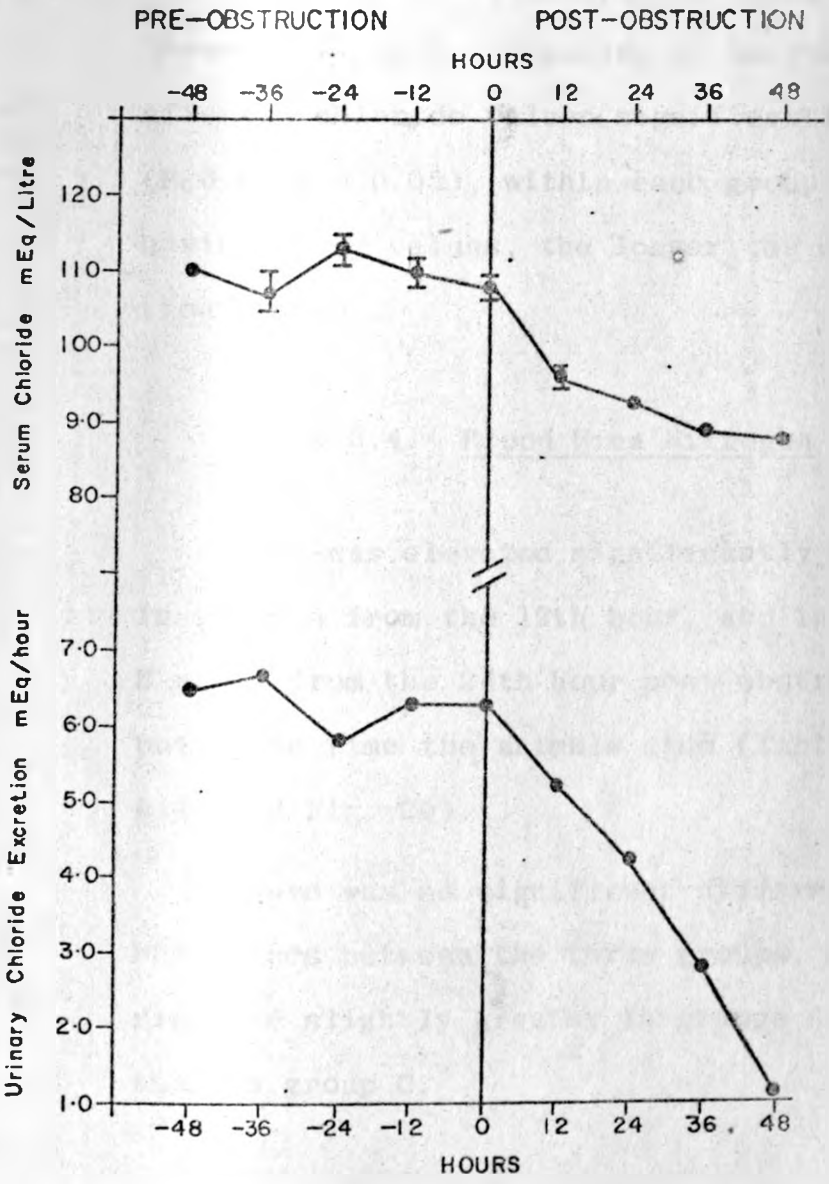


Fig.19 Serum Chloride concentration in mEq/Litre and rate of Urinary Chloride excretion in mEq/hour for group C before and after obstruction. ⊥ Represents ± SEM.

Difference in chloride ion concentration between the groups was significant ($P < 0.01$ and 0.05), with animals in groups A and B showing a more marked hypochloraemia than those in group C. Duration of obstruction affected chloride values significantly ($P < 0.01$ and 0.05), within each group by having lower values, the longer the obstruction lasted.

4.3.4. Blood Urea Nitrogen

This was elevated significantly ($P < 0.05$) in group A from the 12th hour, and in groups B and C from the 24th hour post-obstruction, until the time the animals died (Tables 2, 3 & 4; and Fig. 20).

There was no significant difference in BUN values between the three groups, but its rise was slightly greater in groups A and B, than in group C.

4.3.5. Packed Cell Volume

PCV rose significantly ($P < 0.05$) in groups A and B. In group A, it rose from the

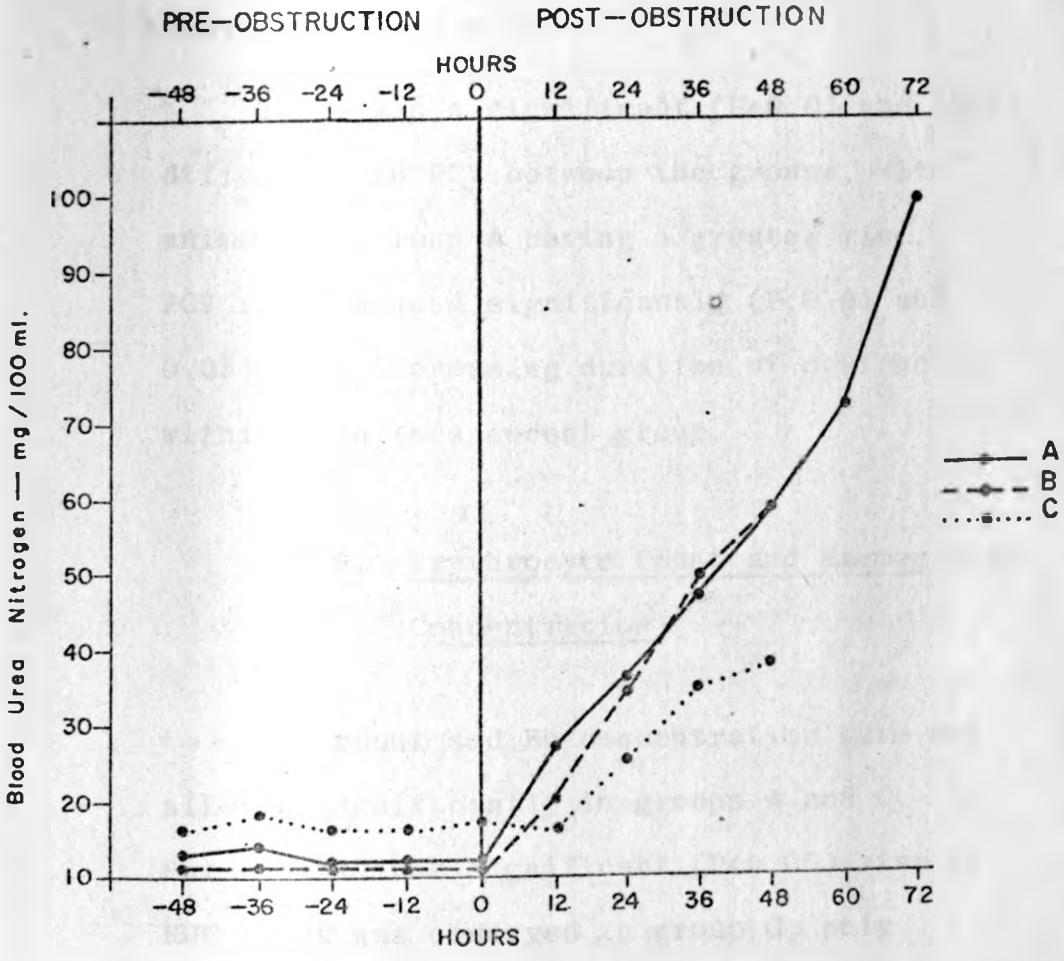


Fig. 20 Compative Blood Urea Nitrogen in mg/100ml. for groups A, B and C before and after obstruction.

24th hour and in group B from the 36th hour after obstruction, until the terminal stages. No significant change occured in group C. See Tables 2, 3 and 4.

There was a significant ($P<0.01$ and 0.05) difference in PCV between the groups, with animals in group A having a greater rise. PCV also changed significantly ($P<0.01$ and 0.05) with increasing duration of obstruction within each independent group.

4.3.6. Erythrocyte Count and Haemoglobin Concentration

RBC count and Hb concentration were not altered significantly in groups A and C. But unexplained significant ($P<0.05$) rise in RBC count was observed in group C, only at 48 hours postobstruction. In group B, significant ($P<0.05$) rise in RBC count and Hb concentration was seen on the 36th and 48th hours after obstruction (Tables 2, 3 and 4).

Significant ($P<0.01$ and 0.05) difference in RBC count and Hb concentration between the groups was observed, with group B showing a greater change than groups A and

C. Duration of obstruction also significantly ($P<0.01$ and 0.05) influenced the changes observed in the two parameters within individual groups.

4.3.7. Leucocyte Count

Animals in groups A and B, had significant ($P<0.05$) rise in WBC count. The rise in group A started on the 36th hour, and in group B on the 12th hour post-obstructively, and continued to the terminal stages. Changes in group C were not significant (Tables 2, 3 and 4).

There was a marked significant ($P<0.01$ and 0.05) difference in WBC count between the three groups, with group B having a higher count than the rest. Duration of obstruction was noted to have a significant ($P<0.01$ and 0.05) influence on the value of WBC count within the individual groups.

4.3.8. Plasma Protein

No significant changes in the values of plasma protein were noticed within independent groups (Tables 2, 3 and 4). However, comparison of plasma protein values between groups A, B

and C revealed significant ($P<0.01$ and 0.05) difference between their values at specific times, with group A showing higher values than groups B and C at each specific time.

4.3.9. Serum Albumin

Groups B and C did not show any significant changes in albumin values. Group A had unexplained significant ($P<0.05$) rise in albumin value only at the 60th hour post-obstructively (Tables 2, 3 and 4).

However, there was significant ($P<0.01$ and 0.05) difference in albumin values between the groups at specified times, with group A having higher values at these specific times.

4.4. Urinalysis

4.4.1. Colour

The urine from animals in group A was yellow initially, but gradually changed to light-yellow during obstruction, except in one animal that it remained yellow all through.

Urine from the animals in groups B and C remained yellow throughout.

4.4.2. Transparency

In all groups, the urine remained clear throughout, but in group A, it had a high viscosity terminally.

4.4.3. Quantity

In the three groups, the quantity of urine voided, reduced gradually until at the terminal stages, only few drops were collected from each animal. This reduction during the terminal stages, was more marked in group A than in groups B & C.

4.4.4. Specific gravity

Specific gravity of urine was significantly ($P < 0.05$) elevated from the 24th hour of obstruction in group A, and from the 12th hour of obstruction in groups B & C (Tables 5, 6 & 7; and Fig. 22). The highest recorded values were 1.053; 1.055 and 1.056 in groups A, B and C respectively.

Varying of the site and duration of obstruction had significant ($P<0.01$ and 0.05) influence on the values of specific gravity.

4.4.5. Urinary pH

Urinary pH was lowered significantly ($P<0.05$) from the 12th hour of obstruction in groups A & C, and from the 24th hour in group B, until the time of death of the animals. The lowest pH observed was 6.0 for groups A and C, and 5.0 for group B (Tables 5, 6 & 7; and Fig. 21). Variation of the site and duration of obstruction significantly ($P<0.01$ and 0.05) influenced the changes observed on urinary pH values.

4.4.6. Protein

Trace amount of protein was detected in urine from all the animals in group A, and 83% of the animals in group C, usually after 48 hours of obstruction. No protein was detected in urine from one animal in group C, and from all the animals in group B.

4.4.7. Glucose

Glucose was not detected in urine from any animal in the three groups.

4.4.8 Ketones

Terminally, small amount of ketones was detected in urine from all the animals in group A, and from 2 animals in group C. Urine from the rest of the animals in group C, and from all the animals in group B was negative for Ketones throughout this experiment.

4.4.9. Sodium Excretion

The rate of urinary excretion of sodium was significantly ($P<0.05$) reduced in all the three groups, from the 12th hour of obstruction to the death of the animals (Tables 5, 6 & 7; and Fig. 11, 12 & 13).

Changing the site and duration of obstruction influenced the rate of urinary excretion of sodium significantly ($P<0.01$ and 0.05), in which case groups A and B reached the lowest sodium excretion rate.

TABLE 5 - URINALYSIS: MEANS ± SD Group A

	PRE-OBSTRUCTION (CONTROL) PERIOD	BASELINE VALUES	POST-OBSTRUCTION PERIOD				
Time (hrs)	-48 to -12	0	12	24	36	48	60
SP.Gravity	1.032±0.002	1.031±0.005	1.034±0.006	1.042±0.005*	1.044±0.001*	1.048±0.005*	1.051±0.003*
pH	8.1 ±0.31	8.0 ±0.00	7.5 ±0.50*	7.0 ±0.63*	6.8 ±0.83*	6.0 ±0.00*	6.0 ±0.00*
Na^+ (mEq/hour)	5.8 ±1.20	4.8 ±0.84	4.3 ±0.82*	4.0 ±0.89*	3.8 ±0.84*	3.4 ±0.55*	2.8 ±0.41*
K^+ (mEq/hour)	58.6 ±2.76	51.2 ±9.44	53.3 ±10.01	59.7 ±4.27	58.6 ±3.58	62.0 ±3.46	63.3 ±4.84
Cl^- (mEq/hour)	5.6 ±0.60	5.6 ±0.89	3.5 ±1.05*	2.2 ±0.75*	1.6 ±0.89*	1.2 ±1.04*	0.6 ±0.68*

* Significant difference from control values at 5% significance level.

SD standard deviation

TABLE 6: URINALYSIS: MEANS ± SD Group B

	PRE-OBSTRUCTION (CONTROL) PERIOD	BASELINE VALUES	POST-OBSTRUCTION PERIOD			
Time (hrs)	-48 to -12	0	12	24	36	48
SP. Gravity	1.032±0.004	1.034±0.004	1.050±0.004*	1.050±0.003*	1.053±0.002*	1.053±0.002*
pH	8.0 ±0.00	8.0 ±0.00	7.0 ±0.00	6.5 ±0.55*	6.0 ±0.00*	5.3 ±0.52*
Na^+ (mEq/Hour	5.8 ±0.78	5.7 ±0.82	4.3 ±0.82*	4.0 ±0.63*	3.5 ±0.55*	2.8 ±0.41*
K^+ (mEq/hour)	55.2 ±6.37	56.7 ±3.93	54.7 ±7.45	57.0 ±3.52	59.0 ±2.45	60.3 ±3.44
Cl^- (mEq/hour)	6.3 ±1.02	5.5 ±0.55	5.0 ±0.63*	4.0 ±0.89*	3.0 ±0.89*	1.7 ±0.52*

* Significant difference from control values at 5% significance level.

SD standard deviation

TABLE 7 - URINALYSIS: MEANS ± SD Group C

	PRE-OBSTRUCTION (CONTROL) PERIOD	BASELINE VALUES	POST-OBSTRUCTION PERIOD			
Time (hrs)	-48 to -12	0	12	24	36	48
SP. Gravity	1.030±0.005	1.033±0.005	1.043±0.003*	1.045±0.004*	1.048±0.004*	1.050±0.005*
pH	7.7 ±0.56	7.0 ±0.00	6.8 ±0.41*	6.0 ±0.00*	6.0 ±0.00*	6.0 ±0.00*
Na^+ (mEq/hour)	6.9 ±0.93	6.3 ±1.21	5.2 ±0.41*	4.8 ±0.98*	4.2 ±0.75*	3.5 ±0.55*
K^+ (mEq/hour)	58.5 ±3.73	57.0 ±3.03	54.3 ±9.24	55.7 ±8.52	55.7 ±8.52	57.7 ±6.62
Cl^- (mEq/hour)	6.3 ±0.92	6.3 ±1.03	5.2 ±0.98	4.2 ±0.75*	2.8 ±0.41*	1.1 ±0.49*

* Significant difference from control values at 5% significance level.

SD standard deviation

Fig. 21 - Urinary pH before and after obstruction in groups A, B and C.

Fig. 22 - Urinary specific gravity before and after obstruction in groups A, B and C.

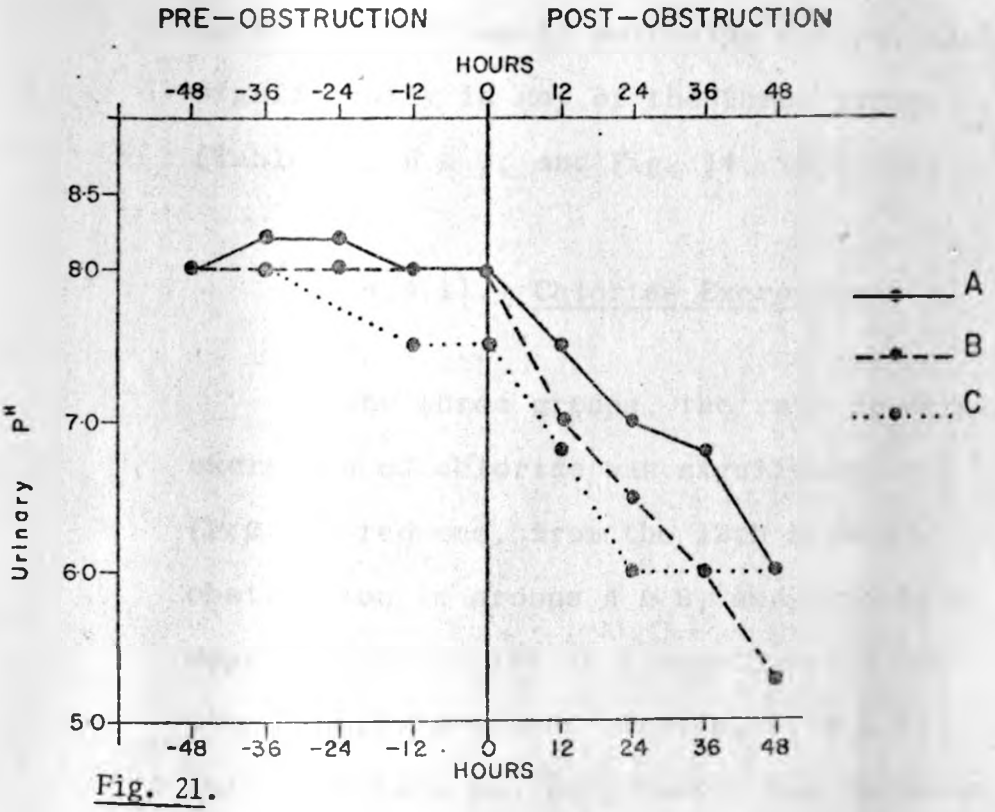

PRE—OBSTRUCTION
POST—OBSTRUCTION
HOURS
-48 -36 -24 -12 0 12 24 36 48
8·5
8·0
7·0
6·0
5·0
Urinary PH
A
B
C

Fig. 21.

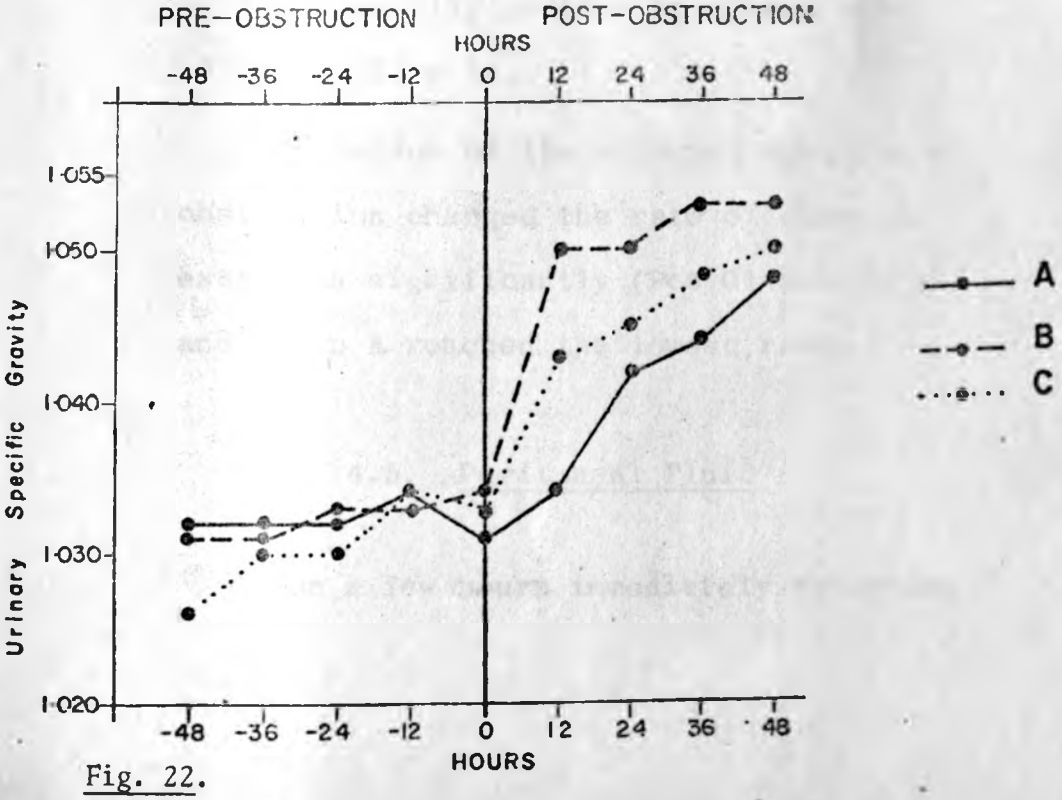

PRE—OBSTRUCTION
POST—OBSTRUCTION
HOURS
-48 -36 -24 -12 0 12 24 36 48
1·055
1·050
1·040
1·030
1·020
Urinary Specific Gravity
A
B
C

Fig. 22.

4.4.10 Potassium Excretion

Throughout the experiment, the rate of urinary excretion of potassium was not altered significantly in any of the three groups (Tables 5, 6 & 7; and Fig. 14, 15 & 16).

4.4.11. Chloride Excretion

In the three groups, the rate of urinary excretion of chloride was significantly ($P<0.05$) reduced, from the 12th hour of obstruction in groups A & B, and from 24th hour of obstruction in group C, until the time the animals died (Tables, 5, 6 & 7). This reduction was progressive and in group A, it terminally reduced to almost zero (Fig. 17, 18 & 19).

Variation of the site and duration of obstruction changed the rate of chloride excretion significantly ($P<0.01$ and 0.05), and group A reached the lowest rate.

4.5. Peritoneal Fluid

For a few hours immediately following

surgery (pre-obstruction), the peritoneal fluid was red in colour in all the groups. During the next 48-hour period after the surgery, the fluid in these three groups changed in colour from red, to light-brown, through pink to clear straw colour. After bowel obstruction, the peritoneal fluid remained straw coloured and clear throughout in group A, but became cloudy terminally. In groups B and C, it changed in colour initially, from straw and clear, to straw and cloudy, through pink, to light-red terminally.

The peritoneal fluid had the smell of fresh blood initially after surgery in all the groups, but soon became odourless, and remained odourless even after obstruction, in the three groups. In the terminal stages of obstruction, it again had the smell of blood, both in groups B & C.

It was very difficult to obtain peritoneal fluid in the period before obstruction, and also during the first 24 hours of obstruction in all the groups. In the period between 24 hours after bowel obstruction and death of the animals in the three groups, the quantity of peritoneal fluid within the

peritoneal cavity, increased gradually and progressively. Although the fluid increased faster in groups B & C, it had not accumulated to the same total quantity as in group A at the time of death of the animals. On abdominal ballotment, it was not possible to tell whether the splashing fluid sound was due to fluid in the peritoneal cavity, or in the forestomachs.

In the period immediately following surgery, peritoneal fluid from the animals in all three groups had high numbers of erythrocytes. Later, the erythrocyte count dropped until finally no erythrocytes were detected in the fluid 48 hours after surgery. There were no erythrocytes detected in the peritoneal fluid from animals in group A, throughout the period of obstruction. In the 12th hour of obstruction, few erythrocytes were detected in the peritoneal fluid from the animals in groups B & C. The erythrocyte count in the fluid from the latter groups, rose progressively until the death time of the animals.

There were few leucocytes in the peritoneal fluid from animals in all the groups immediately following surgery, but their number decreased gradually. However, 48 hours after surgery, there were still few leucocytes detected in the fluid from these animals. The number of leucocytes increased again progressively after bowel obstruction in all the three groups, until at death of the animals, the count was very high. The count was higher in groups B & C than in group A. The fluid had more neutrophils than the rest of the leucocytic-cell types in the period following 24 hours of obstruction in groups B and C, while in group A, the neutrophils exceeded other leucocytic cell-types, only after 60 hours of obstruction.

4.6. Necropsy

All the carcasses were emaciated. Carcasses from group A were more severely emaciated than those from groups B & C.

In group A, there was accumulation of straw coloured fluid in the peritoneal

cavity, while in groups B & C, the fluid was serous to light-brown in colour. This fluid was more in group A than in groups B & C. One animal in group A had a ruptured duodenum and therefore its carcass had ingesta in the peritoneal cavity, with evidence of peritonitis.

The forestomachs, especially the rumen and the abomasum, were distended with gases and fluid ingesta-like contents in the three groups (Fig. 23). The quantity of the contents was more in group A than in groups B & C. The mucosa of the forestomachs was normal in the three groups, with an exception of two carcasses in groupA, which had few ecchmotic haemorrhages and ulcerations in the abomasal mucosa.

Congestion of the intestine was seen on the serosal surface in the three groups. This congestion was more evident in groups B & C where it involved the entire intestinal length. Congestion in the three groups was more severe proximal than distal to the obstruction (Fig. 24). There was more congestion and ecchmotic haemorrhages within

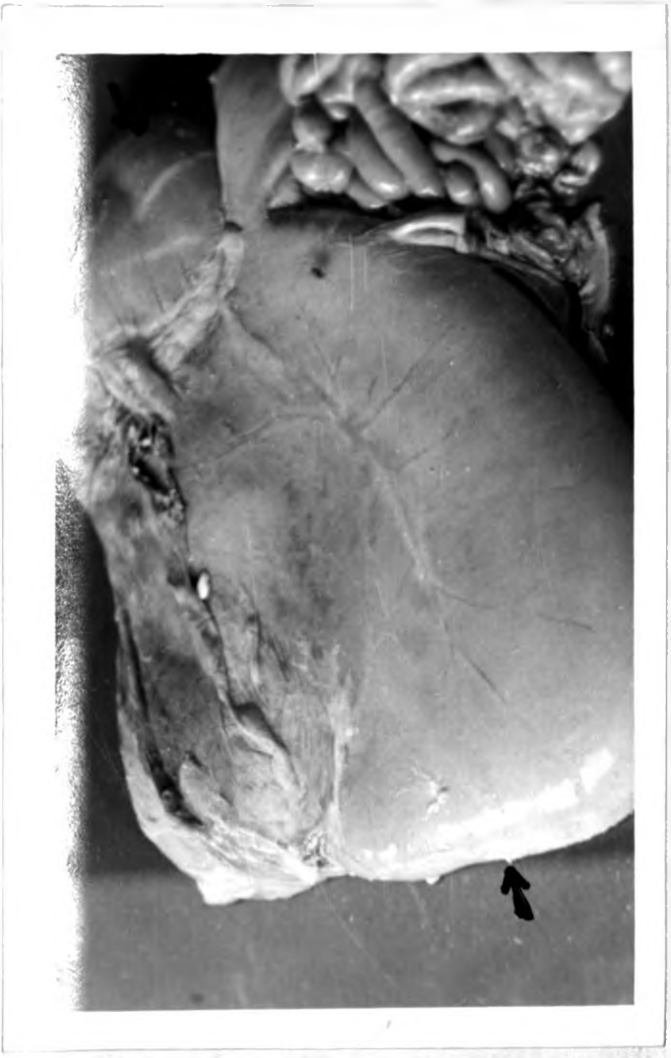


Fig. 23. Severe distension of the rumen and abomasum with fluid contents and gas during obstruction. (Upper arrow - abomasum). (Bottom arrow - rumen)

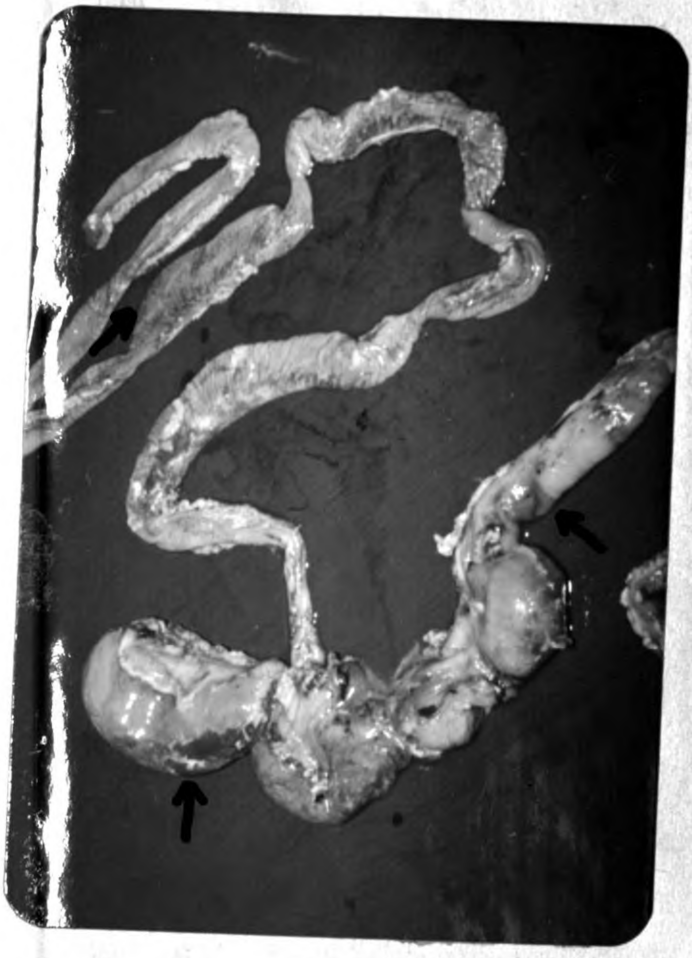


Fig. 24. Generalized congestion of the entire intestinal length evident on the serosal surfaces. (Arrows)

the strangulated loops in both groups B & C. Two carcasses in group C had congested omentum, while one carcass in the same group had oedema of the omentum.

The intestinal lumen distal to the site of obstruction in group A, had either small amount of fluidy greenish-mucoid contents, or nothing at all, but proximal to obstruction the lumen was full. The intestinal contents were progressively less in the lumen near the rectum. In groups B & C, there was blood-tinged fluid contents in the intestinal lumen distal to the strangulated loops, while proximally the lumen was full of fluid greenish contents. The lumen of the strangulated loops had a lot of red to dark-red fluid contents. In group C, the colon had dry faecal pellets.

The intestinal mucosa proximal and distal to the obstruction site in group A had normal colour, but in groups B & C, both proximal and distal to the strangulated intestinal loops, the intestinal mucosa was congested and had petechial haemorrhages (Fig. 25). The mucosa of the strangulated loops was congested, had ecchmotic haemorrhages, and was dark in appearance (Fig. 26 & 27).

Fig. 25. Mucosal congestion of the strangulated intestinal segments. (Arrows)

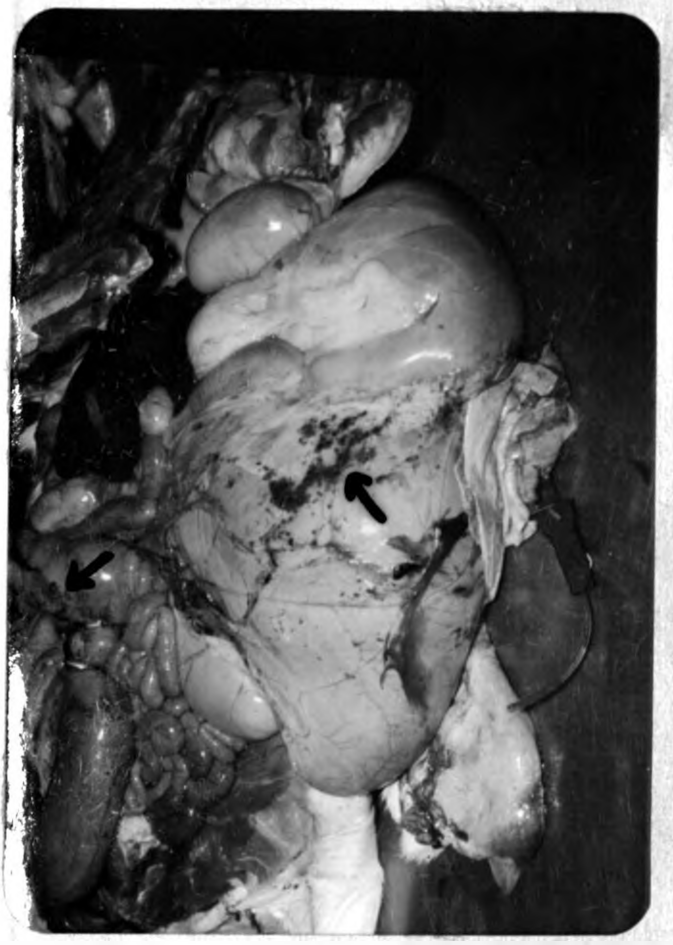


Fig. 26. Ecchmotic haemorrhages on the omentum and the strangulated intestinal segments evident on the serosal surfaces.
(Left arrow - strangulated segment
Right arrow - omentum)

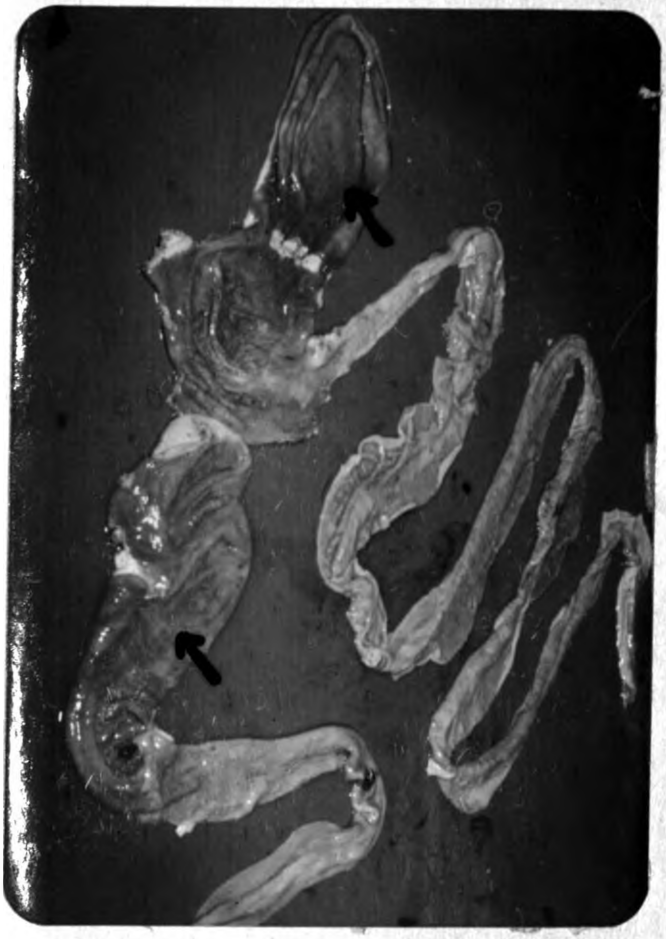


Fig. 27. Darkening of the intestinal mucosa especially in the strangulated segments, as evidence of severe necrosis.
(Arrows)

There was ulceration of intestinal mucosa corresponding to the site of the plastic tubing ligature in the three groups. The plastic tubing also caused tearing of the mesentery in all the cases.

The area around the peritoneal trocar (used to collect peritoneal fluid) in all cases, had localised peritonitis and adhesions.

Other body organs apart from the gastro-intestinal tract appeared normal, except for one carcass in group B which had congested myocardium.

The degree (completeness) of obstruction was confirmed at necropsy.

4.7 Histopathologic Changes

The histopathologic interpretation described under the materials and methods (Sec. 3.3.11) was used only as a guideline to our histopathologic studies.

4.7.1. Group A

The following observations are similar for segments proximal and distal to obstruction. The differences are mentioned where they arise. The summary for these observations is found in table 8.

In the tunica mucosa, there was complete loss of villus epithelium. Detachment of crypt epithelium was evident throughout. General vascular congestion was observed in the lamina propria, but slight haemorrhage and oedema were observed only in 50% of the goats. The cellular reaction included infiltration with macrophages, fibroblasts and lymphocytes as the predominant cell-types. Few plasma cells were present proximal to obstruction, while few neutrophils were observed distal to obstruction.

The tunica submucosa was either completely normal, or had generalized vascular congestion. Oedema and detachment of cells of Brunner's glands were observed only in 33% of the goats; usually distal to obstruction. The cellular reaction in the submucosa included fibroblast predominance, few

lymphocytes and macrophages. Neutrophils were present only distal to obstruction.

Tunica muscularis was normal distal to obstruction in all the goats, while proximal to obstruction, it was normal in 50% of the goats, and had congestion and oedema in the remaining 50% of the goats. There was infiltration with macrophages.

Tunica serosa was completely normal in 33% of the goats. Vascular congestion was observed proximal to obstruction in another 33% of the goats. Fibroblasts were the predominant cells in the serosa, and other cells present were few neutrophils and macrophages.

4.7.2. Group B

The observations below are similar proximal and distal to the point of obstruction, as well as to the strangulated segments. The differences are pointed out where they arise. A summary of the observations is included in table 9.

The tunica mucosa had loss of villus architecture. In every case, there was loss of some crypts of Lieberkuhn. The crypts that were present had detachment of some of their cells. There was however, evidence of crypt-cellular necrosis in the strangulated segments. Congestion and haemorrhage

were evident in the lamina propria, but were more severe in the strangulated segment. Fibroblasts and lymphocytes were the predominant cells in the lamina propria. Other cells present were few neutrophils and macrophages.

There was vascular congestion in the tunica submucosa and tunica-muscularis, which was more severe in the strangulated segments. Slight haemorrhages and oedema were evident in 66% of the goats, both in the submucosa and the tunica muscularis. Fibroblasts and lymphocytes were the predominant cells in these layers. Few neutrophils and macrophages were seen proximal to the strangulation in the submucosa. In 16.6% of the goats, the tunica muscularis was normal both proximal and distal to strangulation.

The tunica serosa was normal distal and proximal to strangulation in 33% of the goats. In another 33% of the goats, the serosa had congestion distal to the strangulation, and in the strangulated segment. Haemorrhages were evident proximal to the strangulation as well as in the strangulated segment. Oedema was observed distal to the strangulation, in 16.6% of the goats. The cellular reaction included infiltration with neutrophils, with predominant cells being fibroblasts and lymphocytes. Macrophages were present proximal to strangulation as well as in the strangulated segments.

4.7.3. Group C

The following observations are similar for ileum, caecum and colon. The differences between these sections are indicated. The observations are summarized in table 10.

In the tunica mucosa of the ileum, there was complete loss of villus architecture. In the caecum and the colon, there was loss of surface epithelium. There was generalized detachment of the epithelium of the crypts of Lieberkuhn. Some of these crypts were detached to the surface, in the caecum and colon. There was evidence of crypt-cellular necrosis in the caecum. Vascular congestion and haemorrhages were present in all the goats, especially in the ileum and caecum. Fibroblasts and lymphocytes were the predominant cells. Other cells present included macrophages and neutrophils, present only in the caecum.

Ileum and caecum had vascular congestion in the submucosa, with more severity in the caecum. Oedema was seen in the submucosa of the caecum and the colon, in only 16.6% of the goats. Submucosal haemorrhage was present in another 16.6% of the goats. Cellular infiltration in this layer included macrophages in the caecum. Fibroblasts were the predominant cells in the three

segments.

The tunica muscularis and serosa were normal, except in the caecum where these layers had vascular congestion and oedema. There was macrophage infiltration in both layers, and few neutrophils only in the tunica serosa.

Table 8 - A Summary of Intestinal Histopathologic Changes for Group A

Intestinal level	Tunica Mucosa	Tunica Submucosa	Tunica Muscularis	Tunica Serosa
Proximal to Obstruction	Loss of villus epithelium. Detached crypt cells. Vascular congestion, slight haemorrhages (±) in the Lamina propria. Oedema (±) in the Lamina propria and crypts. Few plasma cells (±) and macrophages (±) in the lamina propria	Normal, or Vascular congestion Macrophages (±)	Normal, or Vascular congestion (±) Oedema (±) macrophages (±)	Normal, or Vascular congestion (±) Neutrophils (±) macrophages (±)
Distal to obstruction	Loss of villus epithelium. Detached crypt cells. Vascular congestion, slight haemorrhages (±), oedema (±), neutrophils (±) and macrophages (±) in the lamina propria.	Normal, or vascular congestion oedema (±) Detached cells of	Normal	Normal, or Neutrophils (±) Macrophages (±)

Table 8 (cont'd)

Intestinal level	Tunica Mucosa	Tunica submucosa	Tunica Muscularis	Tunica Serosa
		Brunner's glands.		
		Neutrophils (±)		
		Macrophages (±)		

(±) - Not observed in all the goats.

Table 9 - A Summary of Intestinal Histopathologic Changes for group B

Intestinal level	Tunica Mucosa	Tunica Submucosa	Tunica Muscularis	Tunica Serosa
Proximal to strangulation	Complete or partial loss of villus architecture Detached crypt cells. Vascular congestion, haemorrhages, neutrophils (±) and macrophages (±) in the lamina propria.	Vascular congestion, haemorrhages (±) Oedema (±) Few neutrophils (±) and macrophages (±)	Vascular congestion slight haemorrhages (±) or Normal	Normal, or slight haemorrhages (±) Neutrophils (±) Macrophages (±)
Strangulated segment	Complete loss of villus architecture. Detached and necrotic crypt-cells. Severe congestion and haemorrhages in the lamina propria. Neutrophils (±) Macrophages	Severe vascular congestion, haemorrhages (±) and Oedema (+)	Vascular congestion and haemorrhages (±)	Vascular congestion and haemorrhages (±) Neutrophils (+)

Table 9 (cont'd)

Intestinal level	Tunica Mucosa	Tunica submucosa	Tunica muscularis	Tunica serosa
Distal to Strangulation	Complete, or partial loss of villus architecture. Detached crypt cells. Congestion and haemorrhages(±) in the lamina propria. Neutrophils(±)and macrophages (±)	Slight congestion, haemorrhages(±) and Oedema(±)	Congestion, haemorrhages(±) and Oedema(±) or Normal	Normal, or congestion (±) Oedema (±) Neutrophils(±) Macrophages(±)

(±) - Not observed in all the goats.

Table 10 - A Summary of Intestinal Histopathologic Changes for group C

Intestinal level	Tunica Mucosa	Tunica submucosa	Tunica muscularis	Tunica serosa
Ileum	Complete or Partial loss of villus architecture Detached crypt cells. Vascular congestion, and slight haemorrhages (±) in the lamina propria. Few macrophages (±)	Normal, or vascular congestion(±)	Normal, or congestion (16% of the cases).	Normal
Caecum	Loss of surface epithelium. Detached and necrotic crypt cells. Some crypts detached to the mucosal surface. Congestion and haemorrhage (±) in the lamina propria. Few neutrophils (±) and macrophages (±)	Severe vascular congestion. Oedema. Haemorrhage (16% of the cases). Neutrophils (±) macrophages (±)	Vascular Congestion. Macrophages	Vascular congestion. Oedema (±) Neutrophils(±)

Table 10 (cont'd)

Intestinal level	Tunica Mucosa	Tunica submucosa	Tunica muscularis	Tunica serosa
Colon	Loss of surface opithelium Detached and necrotic crypt cells. Some crypts detached to mucosal surface. Haemorrhages (±) Macrophages (±)	Normal, or Oedema (16% of the cases)	Normal	Normal, or presence of macrophages (±)

(±) - Not observed in all the goats.

5. DISCUSSION

5.1. Clinical Features

The clinical behaviour of the animals immediately after surgery (pre-obstruction) was due to surgical stress (especially pain), but 48 hours later, there was always an apparent complete recovery from this stress after pain had subsided. A similar behaviour was reported in sheep by Gingerich and Murdick (1975).

The clinical manifestation was evident earlier and was more severe always in group B and occasionally in group C, than in group A, because high or low strangulating obstructions are more acute than high or low non-strangulating obstructions.

Lowering of temperature similar to that observed in the course of intestinal obstruction in the three groups, has been reported by Pearson and Pinsent (1977); and Sherman (1981), in cattle and a goat respectively. In agreement with what has been

reported by Fox (1970); and Blood et al. (1983) in cattle, temperature rise did not exceed the upper limit of the normal range during intestinal obstruction in all the goats. Reduction in total blood volume, as well as the inactivity of the animals, might have been part of the contributory factors to the lowering of temperature. Shock may have contributed to the lower temperatures in the terminal stages.

The elevation of heart rate was observed in all the groups during bowel obstruction, but was noticed earlier in groups B & C, than in group A. The reason for this was dehydration, reduction of total blood volume, and loss of blood into the strangulated loops of intestine. The terminal slight reduction in heart rate in the three groups, may apparently have been due to blood electrolyte imbalance as well as failing of the cardiac function.

Apart from groaning and grunting, no other respiratory changes were observed. This agrees with the observations of Fox (1970); Pearson and Pinsent (1977); and Blood et al. (1983), in cattle.

Selective appetite, then complete anorexia that were observed in this project, resembled what was reported earlier by Hammond et al. (1964); Corker and Dziuk (1968); and Pearson and Pinsent (1977). The animals in groups B & C developed selective appetite and complete anorexia earlier than those in group A, due to pain and acuteness of strangulation intestinal obstruction. Selective feeding was due to the nature of feed. The animals lost appetite for hard feed earlier.

Defaecation ceased earlier in animals of group C, and latest in those of group A. According to Fox (1970), defaecation in bovine ceases earlier when obstruction is close to the rectum, and later when close to the duodenum. Since the goat is a ruminant, the same reasoning may be applied. Straining and defaecation of scanty foul-smelling greenish-mucoid, or blood-tinged faeces similar to what was seen by Johnston (1962) in a cow, was observed in our experiments. Presence of blood in faeces in groups B & C, was due to loss of the blood into the lumen of the strangulated intestinal loops. This blood may have escaped distally if the lumen obstruction was not fluid-tight.

The drying of the visible mucous membrane

in group A after 60 hours of obstruction, was due to the fact that animals in this group lived longer, and therefore had time to develop a higher degree of dehydration. Paleness of the visible mucous membrane in groups B & C, is associated with blood loss into the strangulated intestinal loops. Change of colour of the visible mucosa during bowel obstruction, has only been reported in the horse, where it became injected, or dark-red because the horse is more susceptible to pain, and hence becomes overexcited, therefore resulting in congestion of the mucosa (Datt and Usenik, 1975; Dobson and Lopez, 1981).

Pain observed in groups B & C was short-lived, since it occured only immediately after obstruction, contrary to the observations in cattle where pain lasted 1-2 days of obstruction (Johnston, 1962). Hofmeyr (1974), has stated that pain receptors on abdominal viscera are either absent or very few, and therefore pain is only exhibited when organs are stretched, bruised, torn, or spastic. In this project, the animals in groups B & C had tearing as well as involvement of a larger part of the mesentery and the gut, than

those in group A; and therefore pain was exhibited immediately after tightening the plastic tubing because of pressure on the mesentery and the gut.

Intestinal hypoperistalsis or aperistalsis seen after surgery, was due to handling of the intestine during the operation. The intestinal hyperperistalsis observed in the course of obstruction may probably have been due to what was reported by Palminteri (1972), that distension with fluid and gas causes violent bowel contractions. The intestinal hypoperistalsis or aperistalsis observed in the terminal stages of this experiment agrees with the observations of Hofmeyr (1974); Datt and Usenik (1975); and Pearson and Pinsent (1977), that generally in the later stages of intestinal obstruction in the horse and ruminants, there is usually gastro-intestinal hypoperistalsis, or aperistalsis. It is also possible that after violent bowel contractions caused by distension with fluid and gas, paresis of the bowel follows due to muscular fatigue resulting from prolonged distension.

Ruminal hypomotility or stasis observed in this project agrees with the general observation that in later stages of bowel obstruction in ruminants, there is usually gastrointestinal hypomotility, or no motility at all (Fox, 1970; Hofmeyr, 1974; Pearson and Pinsent, 1977; Blood et al., 1983). This may be due to distention with fluid and gas as reported by Palminteri (1972). The same reason may be the cause of occasional ruminal hypermotility observed in our experiments.

Clinical dehydration was observed in all the animals, but was more severe in group A, than groups B & C. Similar clinical dehydration has been reported (Johnston, 1962; Hammond et al., 1964; Gingerich and Murdick, 1975; Pearson and Pinsent, 1977) in ruminants. Dehydration has been associated with loss of fluids into the intestine and peritoneal cavity, together with sweating and inability to drink during intestinal obstruction (Datt and Usenik, 1975). In this project, the same reasons apply, apart from sweating which was never observed. The

severity of dehydration in group A was due to the fact that the animals in this group lived longer, and therefore lost more fluid, while those in groups B & C died earlier and as such did not reach the same degree of dehydration as group A. This was also evidenced by presence of bilateral symmetrical abdominal distension in group A, but not in groups B & C. This distension was due to fluid and gas accumulation. The splashing fluid sound heard on abdominal ballotment, was an indication of fluid within the peritoneal cavity, or lumen of gastrointestinal tract, especially the rumen and abomasum. This was confirmed at necropsy. Similar sounds have earlier been reported in ruminants (Johnston, 1962; Sherman, 1981; Blood et al., 1983).

Animals in group A became more emaciated and weaker than those in groups B & C. This was due to complete anorexia. The animals in group A lived longer in anorexic state than those in groups B & C, and therefore became more emaciated and weaker.

Animals in groups B & C had shorter survival time than those in group A. This agrees with the conclucion of Johnston (1962),

that in ruminants the concept that the closer the bowel obstruction to the stomach, the more rapidly will death ensure, does not always apply. The difference between the survival time of these groups depended on the development of secondary circulatory complications. Groups B & C had circulatory involvement since they were subjected to strangulating intestinal obstruction, while group A had non-strangulating obstruction. The survival time in group A falls in the range of survival time recorded for sheep and pigs by Blood et al. (1983). The survival time for groups B & C also falls in the range of survival time recorded for buffalo calves with intestinal strangulating obstruction by Krishnamurthy et al. (1980). However, the range of survival time for goats observed in this project, was shorter than that observed earlier in cattle (Johnston, 1962; Pearson and Pinsent, 1977; Blood et al., 1983). Death in high obstruction is usually due to fluid and electrolyte loss, while in low obstructions, it is primarily due to starvation. Death depends on physical nature and site of obstruction, infective agents

present, and whether or not bacterial toxins are present. In our opinion, fluid and electrolyt imbalances were the main cause of death.

5.2. Blood Analysis

5.2.1 Blood Sodium

The rise in sodium ion concentration observed in group A, agrees with the findings in the horse by Datt and Usenik (1975), but disagrees with the findings in sheep by Gingerich and Murdick (1975). Generally, there is normally a tendency for plasma sodium to move in the opposite from plasma potassium because the two are usually on the opposite sides of the cell-membrane. There is no apparent reason for the elevation of sodium concentration seen in group A.

Decrease in sodium ion concentration in group C may have been due to sequestration of fluid and electrolytes in the gut during bowel obstruction. The sequestered sodium ions could not be absorbed because normally sodium absorption is by an active process. Similar decrease observed in man and the dog is explained to be a result of loss through vomition (Littlejohn, 1965; Palminteri, 1972;

Twedt and Graver, 1982).

Significant difference in sodium concentration between the three groups was probably due to the fact that high and strangulating obstructions are usually more acute than low and non-strangulating obstructions.

5.2.2. Blood Potassium

Hypokalaemia observed in group B agrees with earlier observations in ruminants (Hammond et al., 1964; Corker and Dziuk, 1968; Sherman, 1981). Hypokalaemia is associated with metabolic alkalosis which occurs in bowel obstruction (Hammond et al., 1964). Jubb and Kennedy (1970), have reported that during obstruction, potassium is lost in gastric eliminations. Hypokalaemia in bowel obstruction is primarily due to sequestration of potassium ions within the gut lumen. The decrease in potassium occuring in group B, is apparently due to the fact that in strangulation obstruction there is more injury and more fluid and electrolyte sequestration within the gut lumen than in simple obstruction.

Pearson (1973), has explained that in cattle with bowel obstruction, there is normally a high potassium ion concentration in the ruminal fluid, which is readily available for absorption because absorption of potassium is by passive process, and therefore blood potassium concentration is not grossly affected in ruminants during bowel obstruction. This was true for groups A & C, but not group B, and this makes the theory not acceptable for the goat.

5.2.3. Blood Chloride

There was a marked hypochloraemia in all the groups. Similar observations were made earlier in other ruminants (Hammond et al., 1964; Gingerich and Murdick, 1975; Dass et al., 1981).

In ruminants, chloride is sequestered in the gut lumen as hydrochloric acid, leading to hypochloraemic alkalosis (Gingerich and Murdick, 1975). According to Pearson (1973), chloride change has greater deviation than other blood electrolytes during bowel obstruction in bovine species. Chloride values

remain low until normal peristalsis is resumed. The lower the plasma chloride, the worse the prognosis.

Corker and Dziuk (1968), have suggested that blood chloride concentration is a practical and reliable indicator of obstruction in sheep, when considered together with the clinical features. Our experiments with the goat tend to agree with this concept.

Gingerich and Murdick (1975), have reported that ruminal mucosa can absorb sequestered chloride ions, but the concentration gradient is against this process during bowel obstruction. These authors have noted that abomasal secretions as well as the reflux of alkaline buffered secretions from duodenum to the abomasum are a stimuli for further hydrochloric acid secretion from gastric juice, leading to more hypochloraemia. This probably explains why hypochloraemia was greater in group A because animals in this group had more fluid accumulation in the abomasum than those in groups B & C.

Group differences in chloride values are due to the fact that, high and

strangulating obstructions are more acute than low and simple obstructions. The greater the duration of obstruction, the greater the fluid and chloride sequestration.

5.2.4. Blood Urea Nitrogen

There was marked elevation of BUN in all the groups. This elevation agrees with the findings in sheep (Gingerich and Murdick, 1975); cattle (Pearson and Pinsent, 1977); and buffalo (Krishnamurthy et al., 1980).

Elevation in BUN is due to dehydration that leads to decreased renal blood flow, and therefore inadequate renal function. Pearson and Pinsent (1977), have stated that the higher the degree of dehydration the higher the BUN value.

According to Jubb and Kennedy (1970), BUN can be elevated as a result of increased catabolism, reduced renal blood flow and reduced glomerular filtration during bowel obstruction.

5.2.5. Packed Cell Volume

The rise in PCV in groups A & B was similar to that observed in other ruminants with bowel obstruction (Hammond et al., 1964; Gingerich and Murdick, 1975; Krishnamurthy et al., 1980). Sherman (1981), also observed a rise in PCV in a goat that had duodenal obstruction. This PCV elevation may be associated with dehydration. Group C had low degree of dehydration, and hence no rise in PCV.

The group differences in PCV values depends on the site and nature of obstruction.

5.2.6. Erythrocyte Count and Haemoglobin Concentration

The high RBC count in group C at 48 hours postobstruction, is difficult to explain since it is an isolated rise at only one specific time.

The rise in both RBC count and Hb concentration was similar to the findings of Pearson (1973) in cattle. Elevation is partly due to dehydration and partly due to compensatory process by the erythropoetic

system which tries to synthesize greater numbers of RBC to replace those lost into the strangulated loops. The RBC released by the erythropoetic system are then detected in peripheral circulation as an increase in their numbers.

5.2.7 Leucocyte Count

Leucocytosis with neutrophilia which was observed in groups A & B, is similar to the findings of Krishnamurthy et al. (1980), who associated it with stress, trauma, and infection in bowel obstruction. Leucocytosis was also observed earlier in a goat with duodenal obstruction (Sherman, 1981).

Animals in group B had the highest WBC count and neutrophilia, probably due to the fact that in strangulating obstruction, there is necrosis and gangrene of the gut segments which allows microorganisms and their toxins to diffuse into blood circulation from the gut lumen. This leads to an increased neutrophil count in response of normal body defence mechanism to infection.

5.2.8. Plasma Protein and Serum Albumin

The difference in plasma protein and serum albumin between the groups may be attributed to differences in the degree of dehydration as a result of different levels and nature of obstruction.

5.3. Urinalysis

5.3.1. Colour

The change in urine colour from yellow to light yellow was not of any significance, since this is the colour of normal urine due to urochromes.

5.3.2. Transparency

The high viscosity of urine observed terminally in group A, is probably due to the reduced volume of urine voided, and hence increased concentration of solutes. This was only in group A because animals in this group survived longer than those in groups B & C, and therefore time allowed their urine to be

more concentrated. This viscosity is not necessarily abnormal (Benjamin, 1979).

5.3.3. Quantity

The reduction in urine output observed in the three groups, has previously been found during bowel obstruction in sheep (Gingerich and Murdick, 1975), and in buffalo calves (Krishnamurthy et al., 1980). This is probably due to reduced renal blood flow, and glomerular filtration, in response to the reduced plasma volume and dehydration due to bowel obstruction. The greater reduction in urine output in group A, than in the other groups was due to the fact that the animals in this group survived longer, and had therefore a chance to be more dehydrated.

5.3.4. Specific Gravity

Elevation of urine specific gravity in the three groups is related to dehydration and reduced renal blood flow, and therefore the urine becomes more concentrated. This is

because the specific gravity of urine is inversely related to the volume of urine voided, and the number of solute molecules in it. Dehydration and reduced fluid intake causes elevation of urine specific gravity (Benjamin, 1979). The animals in this experiment were severely dehydrated, and reduced their water intake later during obstruction, and these factors contributed to the elevation of urine specific gravity.

5.3.5. Urinary pH

The low urine pH in the three groups, is similar to observations in sheep with duodenal obstruction (Gingerich and Murdick, 1975). Hydrogen ion is secreted in urine in exchange with sodium ion during sodium reabsorption in the distal renal tubules. Gingerich and Murdick (1975), have noted that undetermined anions like phosphates and sulphates from tissue catabolism in starvation and dehydration; lactates and other organic acids from incomplete carbohydrate metabolism during bowel obstruction, may contribute to aciduria since they are voided in urine.

The animals in this experiment were all starved as a result of complete anorexia in the course of obstruction, and therefore, the urinary pH changes related to starvation occured as described above.

The lowest recorded urine pH was in group B. This is probably because of more pain and intestinal tissue damage due to strangulating obstruction. Therefore, the animals in this group became anorexic earlier, and were eventually more starved than those in groups A & C.

5.3.6. Protein

The trace amounts of protein present in urine could have been due to renal insufficiency. Renal insufficiency is generally brought about by shock in such diseases as intestinal obstruction, and trauma. The products of tissue destruction elsewhere in the body permit protein leakage from renal capillaries. There could also be protein leakage due to increased glomerular permeability as a result of congestion of the capillaries in response to stress (Benjamin,

1979). Animals in group A lived longer and therefore had more tissue damage and stress for a longer period, than those in the other groups, and hence the trace amounts of proteins.

5.3.7. Ketones

All animals in group A and two in group C had small amounts of ketones detected in urine. This was contrary to the reports of Gingerich and Murdick (1975) in sheep, who despite complete anorexia during obstruction, did not detect any Ketonuria. However, severe anorexia and starvation are usually associated with ketonuria.

Animals in group A lived longer in starvation, and hence the resulting terminal ketonuria. Ketonuria in the two animals in group C, was due to individual variation, which could mean they starved more than the rest, possibly as a result of developing severe anorexia earlier than the rest.

5.3.8. Sodium Excretion

The reduced rate of urinary excretion

of sodium in the three groups, is similar to that reported by Gingerich and Murdick (1975), at the 60th hour post-obstruction in sheep. Since there is usually renal sodium retention in bowel obstruction (Krishnamurthy et al., 1980), the goats in this experiment seem to have taken the same pattern.

5.3.9. Potassium Excretion

Since serum potassium concentration in the three groups was not grossly altered, there follows no significant changes in the rate of urinary excretion of potassium. These findings disagree with those of Gingerich and Murdick (1975) in sheep, who found a decrease in urinary excretion of potassium during duodenal obstruction.

5.3.10. Chloride Excretion

There was marked reduced rate of urinary excretion of chloride in the three groups. This was similar to the findings of Hammond et al. (1964) in cattle; and Gingerich and Murdick (1975) in sheep, during bowel obstruction.

Chloride ion is conserved by the kidneys in hypochloraemia of any source. All animals in the three groups had hypochloraemia, and therefore reduced rate of urinary excretion of chloride.

5.4. Peritoneal Fluid

The red colour of the peritoneal fluid observed in the three groups following surgery, was due to blood in the peritoneal cavity from the surgical operation. The blood was gradually absorbed until the fluid was free of blood, and its colour changed from red, to light-brown, through pink to straw.

The peritoneal fluid changed in appearance from clear to cloudy. This was due to inflammatory cells as a result of inflammatory reaction triggered by obstructive injury to the bowels, and possibly due to infection introduced through the peritoneal trocar.

In groups B & C, the colour of the peritoneal fluid changed from straw, through pink to light-red. This was the result of leakage of contents from the lumen of

strangulated loops, and surrounding blood vessels into the peritoneal cavity. The contents were usually mixed with blood. These observations agree with the findings of Krishnamurthy et al. (1980), and Blood et al. (1983).

The smell of blood noticed in the peritoneal fluid after surgery in the three groups, and in the terminal stages of obstruction in groups B & C, was due to the presence of blood in the peritoneal cavity. Similar findings have been reported by Yale (1969).

The gradual increase in peritoneal fluid quantity observed in the period after 24 hours of obstruction in the three groups, was due to fluid leakage from the bowel lumen, local blood vessels and the surrounding tissues. The rate of accumulation of the fluid was higher in groups B & C because in addition to leakage from the bowel lumen, there was also fluid from the strangulated local blood vessles. The latter observation agrees with the findings of Krishnamurthy et al. (1980); and Blood et al. (1983).

However, the total amount of accumulated peritoneal fluid in group A, was greater than in groups B & C. The reason for this was the long life of the animals in group A which allowed for more accumulation time.

Presence of erythrocytes and leucocytes in peritoneal fluid from the three groups after surgery, was due to presence of blood from the operation injury. After reabsorption of this blood, the erythrocytes disappeared for all the groups, but reappeared in the fluid 12 hours after obstruction, only in groups B & C. The reappearance was due to presence of blood from strangulated intestinal loops as well as from the occluded local blood vessels. This is supported by similar reports from Runnells et al. (1965); Jubb and Kennedy, (1970); White (1981); and Blood et al. (1983).

The leucocytes were detected in the peritoneal fluid preobstructively and throughout the obstruction period, possibly because of the localised peritonitis around the peritoneal trocar, presence of infection introduced from outside through the fluid-

collecting y-tube, and leakage of bacteria from the gut lumen into the peritoneal cavity. This possibility is strengthened by high numbers of neutrophils detected in the peritoneal fluid from groups B & C, where there was massive necrosis and gangrene of the strangulated loops, making them more permeable to bacteria.

5.5. Necropsy

Emaciation of the carcasses was due to complete anorexia during bowel obstruction in which cases the animals in group A lived long in this anorexic status, hence severe emaciation. This supports observations in sheep that died of bowel obstruction (Naerland and Helle, 1962), but is contrary to observations in the horse by Dobson and Lopez (1981) who found carcasses of horses dying of intestinal obstruction to be in good body condition. It seems that during obstruction, the longer an animals lives, the more severe will be the emaciation.

The appearance of serous to light-brown fluid in the peritoneal cavity in groups B & C,

was due to blood leakage into the cavity as a result of strangulation of mesenteric blood vessels.

The quantity of peritoneal fluid was more in carcasses from group A because the animals survived long and this gave a chance for more fluid to leak into the peritoneal cavity. Animals in groups B & C would probably have had more peritoneal fluid than those in group A, had they lived long, since their blood vessels were also occluded.

The rupture of the duodenum in one animal of group A, which was caused by the tightened plastic tubing, resulted in peritonitis. This agrees with Jubb and Kennedy (1970), who reported that when perforation of intestine is present, peritonitis may result.

Distension of the forestomachs with fluid and gas, similar to what was observed in this project has been reported in a goat (Sherman, 1981), and sheep (Naerland and Helle, 1962; Gingerich and Murdick, 1975), that died of intestinal obstruction. The accumulation is due to the fact that the

forestomachs are proximal to the obstruction. The source of the fluid is the swallowed saliva, gastric juice, sequestration of fluid from tissues and plasma. The gas is from fermentation of ingesta and from swallowed air.

Animals in group A, lived longer than those in groups B & C and therefore had more fluid and gas accumulation. The ulceration seen in the abomasum of two carcasses in group A, may have occured prior to duodenal obstruction.

Congestion was more severe in the proximal than distal intestinal segments. This is because the segments proximal to the obstruction site are more distended and hence more venous compression. There was more venous compression in groups B & C than in group A, and hence more intestinal congestion in these groups. Distension and venous compression were greater on the strangulated intestinal loops, resulting in more congestion and haemorrhages of the loops.

Necrosis and gangrene are reported to be common in strangulating bowel obstructions. In these cases the intestine involved appear

dark (Rooney, 1965; Runnells et al., 1965; Jubb and Kennedy, 1970). This was observed in cases of groups B & C.

Blood-tinged contents seen in groups B & C in the intestinal lumen distal to the obstruction site, and in the lumen of the strangulated loops, was due to blood leaking from the occluded mesenteric vessels into the strangulated loops, and thereafter little amount passing into the lumen of segments distal to these loops..

Finding of dry faecal pellets in the colon of carcasses in group C, was similar to the observation of Sherman (1981) in a goat carcass that had duodenal obstruction.

The lumen nearer the rectum was more empty because the faecal material had been passed out, and no more was pushed in its place due to proximal obstruction. This has earlier been observed by Rooney (1965); Corker and Dziuk (1968); Dobson and Lopez (1981); and Sherman (1981).

The distension of the proximal intestinal segments in groups B & C was probably due to the fact that invariably, the segments pro-

ximal to obstruction site are usually dilated and distended with fluid and gas.

Peritonitis and peritoneal adhesions around the peritoneal trocar were due to injury, contamination, and infection in the area. The trocar made a communication between the external environment and the peritoneal cavity, which was unsealed each time fluid was collected. These contributed to introduction of contaminants and infective agents into the cavity.

5.6. Histopathologic Changes

The loss of villus epithelium, as well as loss of villus architecture, may be due to the reduced blood flow through the villi as a result of intraluminal pressure during simple obstruction. This resulted in less oxygen getting to the epithelium, and hence the latter sloughing off. It has been suggested by Bounous et al. (1966), that circulatory insufficiency depresses the biosynthesis of intestinal mucin. Finally, the epithelium becomes vulnerable to trypsin activity of the intestinal juice, and the mucosal lesions are thus created (Bounous, 1967).

During strangulating obstruction, there is complete occlusion of blood supply to the villi, and therefore results in the loss of villi (loss of villus architecture), detachment and necrosis of the crypt cells. These agree with the reports in dogs and horses by Brown et al. (1970), Moore et al. (1980), and White et al. (1980). However, the findings by these authors were only up to 180 hours after obstruction. The observations in our experiments disagree with reports by Cohn (1961), that in dogs with strangulating obstruction, the entire mucosa was unrecognizable, apart from epithelial nuclei, while the submucosa was identified only as a transition between mucosa and muscularis which could be due to species variation.

The detachment of the crypt cells to the mucosal surface, seen in groups B & C was probably due to complete occlusion of blood supply to these glands.

In simple obstruction, vascular congestion apparently resulted from pressure on blood vessels due to intestinal intraluminal pressure. When intravascular pressure build up to a certain threshold, some blood cells leak out of the capillaries, resulting in slight haemorrhage.

Congestion, haemorrhage and oedema were more severe in strangulating obstruction because venous compression during this type of obstruction causes passive hyperaemia, oedema, haemorrhage and finally necrosis (Runnells et al., 1965). These observations agree with earlier reports in horses (Moore et al., 1980; White et al., 1980), bovine (Wynn Jones et al., 1957), and those by Jubb and Kennedy (1970), as well as Blood et al. (1983).

Fibroblasts and lymphocytes are usually present in the normal intestinal wall layers, but an increase in their number during these experiments was due to severe inflammatory reaction. Presence of neutrophils, plasma cells, and macrophages was probably due to invasion of intestinal wall by bacteria and their products as a result of increased mucosal permeability. Some of the bacteria identified in the intestinal lumen have been found to traverse the intestinal wall

without actual perforation (Cohn, 1961). Masses of bacteria have been isolated from intestinal wall layers during intestinal strangulating obstructions (Yale, 1969).

6. CONCLUSIONS

The following conclusions were made about intestinal obstruction in goats:

1. Clinical dehydration occurs in both simple, and strangulating obstruction, but it is more severe in simple obstruction where the goats survive longer.

2. Defaecation ceases earliest during obstructions in the distal intestinal segments than proximal segments

3. Gastrointestinal hypermotility is consistently present in the initial stages of both simple, and strangulating obstructions, but terminally, there is gastrointestinal hypomotility.

4. Abdominal distension due to fluid accumulation in the peritoneal cavity and in the stomachs is invariably present when goats survive more than 60 hours of obstruction.

5. The average survival time for goats with strangulating obstruction is about 53 hours, and with simple obstruction, it is 92 hours. Goats with higher strangulating obstructions die earlier than those with low strangulating obstructions.

6. There is significant ($P<0.05$) hypernatraemia in simple obstruction, and hypokalaemia in higher strangulating obstructions.

7. Significant ($P<0.05$) hypochloraemia and BUN elevation are present in both simple, and strangulating obstructions.

8. The quantity of urine voided decreases with time i.e. the longer the goat survives with obstruction, the less the quantity voided. Urine specific gravity is elevated, urine pH lowered, and there is a decrease in the rate of urinary excretion of sodium and chloride.

9. There is congestion of intestinal serosal surfaces which is grossly more severe in strangulating obstructions.
   Intestinal mucosa is grossly congested, and haemorrhages in the mucosa occurs only in strangulating obstructions, and more specifically in the strangulated intestinal segments.

10. Histologically, there is loss of villus epithelium in simple obstruction, and loss of villus architecture in strangulating obstructions.
    There is vascular congestion in the lamina propria in both simple and strangulating obstruction, but haemorrhages and oedema are seen in the lamina propria only in strangulating obstructions.

Cellular reaction includes infiltration of the intestinal wall layers by neutrophils and macrophages, and the reaction is more marked in strangulation obstructions than in simple obstructions.

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KEY TO APPENDICES 1 - 18

1 - 6 - Individual Blood Parameter values for group A

7 -12 - Individual Blood Parameter values for group B

13-18 - Individual Blood Parameter values for group C

N -	Neutrophil	E -	Eosinophil
L -	Lymphocyte	M -	Monocyte
B -	Basophil		

APPENDIX 1

Animal A - 1

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	141	5	110	10	8.0	2.90	22	13.90	8.0	13600	53	47	0	0	0
-36	141	5	103	10	7.8	3.05	31	13.85	11.0	10900	68	32	0	0	0
-24	150	5	101	10	7.8	2.40	28	14.90	10.1	11200	69	31	0	0	0
-12	143	4	100	10	9.0	2.90	31	15.35	10.8	11300	69	31	0	0	0
0	141	4	100	10	8.2	3.25	31	15.60	10.3	11800	68	32	0	0	0
12	140	3	77	20	8.2	2.90	30	14.80	10.4	11300	69	31	0	0	0
24	140	3	75	35	7.8	2.40	37	14.70	9.0	11700	75	25	0	0	0
36	155	3	66	35	8.2	2.35	39	15.50	9.8	17500	78	28	0	0	0
48	155	3	63	50	8.2	3.05	38	14.95	10.2	15000	71	29	0	0	0
60	164	4	65	60	8.6	3.75	48	15.35	10.4	15000	77	23	0	0	0
72	160	3	67	72	8.2	2.25	48	15.15	10.2	14400	78	22	0	0	0

APPENDIX 2

Animal A - 2

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	141	4	99	10	7.4	3.10	30	14.70	10.3	13300	60	40	0	0	0
-36	143	4	99	10	6.2	3.30	30	13.50	10.7	11000	68	32	0	0	0
-24	143	3	99	10	6.4	3.00	31	14.90	10.6	11600	62	38	0	0	0
-12	140	4	100	10	6.4	3.00	29	13.45	12.7	11600	95	5	0	0	0
0	144	4	100	10	6.6	2.90	31	13.00	9.9	11400	71	29	0	0	0
12	146	4	100	25	6.6	3.00	28	14.25	9.6	11300	68	32	0	0	0
24	153	4	95	35	6.5	3.25	36	14.15	9.3	13100	70	30	0	0	0
36	151	4	79	35	6.5	2.90	43	13.45	9.0	19100	83	17	0	0	0
48	150	4	69	45	6.8	2.60	45	12.55	9.0	23200	57	43	0	0	0
60	161	5	63	50	6.2	3.15	45	13.60	9.4	22500	55	45	0	0	0
72	162	4	61	60	6.3	3.25	48	14.60	9.2	23100	55	45	0	0	0

APPENDIX 3

Animal A - 3

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	151	4	110	20	8.6	2.90	33	16.00	11.0	11500	80	20	0	0	0
-36	146	4	100	20	7.8	2.95	38	16.90	12.9	11500	94	6	0	0	0
-24	144	4	96	20	8.2	2.95	30	17.80	13.4	11000	91	9	0	0	0
-12	145	4	100	20	6.4	2.95	32	13.40	10.0	11200	93	7	0	0	0
0	147	3	105	20	6.4	3.14	31	15.45	13.7	11200	85	15	0	0	0
12	144	3	72	55	9.4	3.25	40	18.00	12.8	13200	90	10	0	0	0
24	154	4	64	65	9.2	3.10	40	13.60	12.7	14600	98	2	0	0	0
36	150	3	62	118	10.5	3.25	43	18.25	14.8	20400	89	11	0	0	0
48	162	4	60	118	10.6	3.25	45	19.15	13.7	25600	98	2	0	0	0
60	160	4	56	113	10.1	3.15	44	23.00	16.6	29000	95	5	0	0	0
72	160	4	56	118	10.3	3.25	45	28.25	14.8	32100	97	3	0	0	0

APPENDIX 4

Animal A - 4

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	140	4	102	15	8.2	3.15	35	19.00	11.6	11500	69	30	0	1	0
-36	141	3	104	15	8.0	3.05	30	19.75	12.3	11500	73	27	0	0	0
-24	140	3	99	10	8.0	3.30	30	17.35	12.2	11100	64	36	0	0	0
-12	143	3	99	15	8.0	3.50	32	15.65	13.0	11100	54	46	0	0	0
0	143	3	99	15	8.2	3.40	30	18.35	12.8	13600	53	47	0	0	0
12	143	5	87	20	8.4	3.45	33	17.80	13.3	13000	72	28	0	0	0
24	144	4	87	25	9.2	4.10	45	19.40	14.2	17600	67	33	0	0	0
36	140	4	77	30	10.0	3.75	48	18.00	14.6	21900	76	24	0	0	0
48	150	4	78	40	10.6	3.95	49	18.00	14.6	19600	73	27	0	0	0
60	154	4	71	76	10.6	3.75	48	20.35	14.8	19300	70	30	0	0	0
72	153	2	55	108	10.6	3.25	48	19.60	14.7	18500	71	29	0	0	0

APPENDIX 5

Animal A - 5

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	140	3	99	15	8.0	3.15	33	19.50	10.8	13500	52	48	0	0	0
-36	147	3	98	20	6.6	3.75	33	19.15	12.1	13600	77	23	0	0	0
-24	148	4	100	15	8.0	3.30	32	18.35	11.9	13100	81	19	0	0	0
-12	147	3	99	15	7.4	3.25	31	15.50	12.4	12600	75	25	0	0	0
0	144	3	99	15	8.2	3.50	33	15.80	12.7	11200	72	28	0	0	0
12	144	4	85	20	8.0	3.30	36	15.15	12.4	12200	87	13	0	0	0
24	145	4	77	25	8.4	4.10	39	16.35	12.8	13400	77	23	0	0	0
36	150	3	71	30	8.6	3.75	39	16.50	12.8	35600	71	29	0	0	0
48	157	4	69	50	9.8	4.00	41	17.05	13.3	39600	85	15	0	0	0
60	160	4	67	65	9.4	4.10	43	16.00	13.8	45600	87	13	0	0	0
72	157	4	59	82	9.2	3.85	45	17.25	14.1	53500	88	12	0	0	0

APPENDIX 6

Animal A - 6

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	147	4	110	10	7.0	2.80	22	12.90	9.0	12600	54	46	0	0	0
-36	140	4	110	10	6.8	2.90	24	12.85	10.0	11900	67	33	0	0	0
-24	141	5	110	10	6.8	2.30	28	13.90	10.1	11200	69	31	0	0	0
-12	150	4	111	10	8.0	2.80	30	14.35	11.8	12300	70	30	0	0	0
0	143	4	110	10	7.2	3.15	30	15.60	10.3	12800	69	31	0	0	0
12	144	4	108	20	7.2	2.80	31	15.80	11.4	12300	74	26	0	0	0
24	136	3	96	30	7.8	2.30	37	15.70	10.0	11700	77	23	0	0	0
36	142	4	91	35	7.2	2.25	38	15.70	10.8	16500	72	28	0	0	0
48	138	3	83	45	7.2	2.90	45	15.95	10.4	16000	78	22	0	0	0
60	140	3	72	65	7.6	3.65	52	15.35	10.2	16100	78	22	0	0	0
72	136	3	66	150	7.2	3.25	49	17.25	11.5	16400	80	20	0	0	0

APPENDIX 7

Animal B - 1

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	140	5	101	10	8.0	3.25	35	18.50	11.9	12600	56	44	0	0	0
-36	140	5	102	10	8.0	3.25	32	17.60	11.2	12800	61	39	0	0	0
-24	120	5	104	10	6.0	3.55	32	16.15	11.0	13800	63	37	0	0	0
-12	140	5	102	10	8.1	3.55	33	15.15	11.2	14600	67	33	0	0	0
0	130	4	101	10	8.0	3.75	32	15.20	11.2	13500	74	26	0	0	0
12	142	4	100	15	7.2	4.00	35	18.40	11.2	17800	78	22	0	0	0
24	144	4	85	30	8.0	3.35	38	19.20	12.1	22700	69	31	0	0	0
36	130	4	78	65	8.0	3.35	44	20.80	14.6	24100	76	24	0	0	0
48	130	4	66	65	8.2	3.35	44	21.50	14.9	31300	81	19	0	0	0

APPENDIX 8 Animal B - 2

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	140	4	101	10	7.2	2.25	32	15.35	10.2	12600	62	38	0	0	0
-36	140	5	100	10	6.0	2.75	32	15.25	10.1	12600	81	19	0	0	0
-24	140	5	103	10	6.8	2.75	33	15.15	10.2	13900	79	21	0	0	0
-12	120	4	100	10	6.8	2.80	32	15.15	10.3	13800	74	26	0	0	0
0	140	4	99	10	7.0	3.25	32	15.15	10.2	13800	70	30	0	0	0
12	143	5	96	30	7.6	3.35	33	18.50	11.2	17700	77	23	0	0	0
24	145	4	95	55	8.1	3.55	35	21.25	11.3	21200	81	19	0	0	0
36	150	4	77	66	7.7	3.60	36	29.80	12.8	27200	83	17	0	0	0
48	148	4	66	65	7.6	3.35	38	32.25	12.9	34900	89	11	0	0	0

APPENDIX 9

Animal B - 3

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	141	4	99	15	7.8	2.25	30	12.50	10.2	12100	60	40	0	0	0
-36	141	4	99	15	7.0	2.35	33	11.80	10.5	12100	68	32	0	0	0
-24	140	5	103	15	7.0	4.00	33	11.15	9.5	11400	69	31	0	0	0
-12	140	4	96	15	7.2	2.60	34	11.30	10.1	11400	69	31	0	0	0
0	140	4	96	15	7.2	3.25	33	11.30	10.2	11000	70	30	0	0	0
12	140	5	83	22	7.8	3.60	34	12.70	10.6	13300	74	26	0	0	0
24	150	3	75	30	7.2	3.50	37	14.10	10.8	24700	81	19	0	0	0
36	160	3	77	45	6.8	2.90	38	25.15	11.7	27400	75	25	0	0	0
48	140	3	76	56	6.8	2.95	38	25.20	12.8	37000	80	20	0	0	0

APPENDIX 10 Animal B - 4

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	140	5	96	15	7.8	2.60	22	9.05	8.7	12600	54	46	0	0	0
-36	140	5	96	15	7.3	2.60	23	8.55	7.9	12100	73	27	0	0	0
-24	140	5	94	15	7.2	2.75	23	9.15	8.0	13100	68	32	0	0	0
-12	140	5	102	15	7.3	3.05	22.5	9.30	8.2	12700	71	29	0	0	0
0	140	5	101	15	7.4	2.60	22.5	9.60	8.5	13500	63	37	0	0	0
12	143	4	90	20	7.4	2.55	25	10.10	7.1	15100	79	21	0	0	0
24	140	4	85	20	7.0	2.20	27	10.35	8.4	15700	77	23	0	0	0
36	150	4	78	25	6.2	3.25	29	19.15	8.9	19400	74	26	0	0	0
48	140	4	66	40	6.8	2.50	25	23.30	8.7	21100	81	19	0	0	0
60	140	4	61	55	7.0	3.00	25	22.30	8.5	28000	85	15	0	0	0

APPENDIX 11 Animal B - 5

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	145	5	110	10	6.0	2.25	34	16.50	10.9	10600	55	45	0	0	0
-36	142	4	107	10	8.0	2.25	30	15.60	10.2	10800	60	40	0	0	0
-24	144	5	102	10	7.0	2.55	30	14.15	10.0	11800	60	40	0	0	0
-12	142	5	108	10	7.1	2.55	31	15.15	10.2	12600	69	31	0	0	0
0	144	4	102	10	9.0	2.75	31	13.20	10.2	11500	72	28	0	0	0
12	138	4	96	15	8.2	3.00	34	16.40	10.2	15800	78	22	0	0	0
24	141	3	87	20	8.0	3.00	37	17.20	10.2	20700	69	31	0	0	0
36	136	3	78	30	8.0	3.15	43	18.80	12.1	22100	75	25	0	0	0
48	132	4	72	55	8.2	3.15	43	21.50	13.6	28300	82	18	0	0	0

APPENDIX 12 Animal B - 6

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	141	4	102	10	7.2	3.25	30	14.35	9.2	11500	61	39	0	0	0
-36	141	4	101	10	7.0	3.75	30	14.15	9.0	11500	80	20	0	0	0
-24	141	5	102	10	7.8	2.65	31	14.15	9.0	12800	79	21	0	0	0
-12	125	5	101	10	7.8	2.75	30	14.15	9.1	12800	72	28	0	0	0
0	141	4	100	10	7.8	3.20	30	17.40	9.0	12700	78	22	0	0	0
12	142	4	99	35	7.0	3.15	31	20.15	10.1	16600	77	23	0	0	0
24	144	4	94	55	7.6	3.35	34	28.70	10.2	19200	80	20	0	0	0
36	142	3	76	65	8.0	3.45	35	30.25	11.6	25100	80	20	0	0	0
48	138	3	66	65	7.7	3.35	37	30.15	11.7	32800	87	13	0	0	0

APPENDIX 13 Animal C - 1

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	144	3.2	111	20	7.0	2.90	22	8.10	9.8	10100	46	54	0	0	0
-36	140	3.6	108	20	7.2	3.05	24	8.90	8.4	9700	62	38	0	0	0
-24	144	2.4	116	15	8.0	2.40	25	10.15	7.3	10900	61	39	0	0	0
-12	144	3.2	110	20	7.7	2.90	25	9.15	7.6	10800	57	43	0	0	0
0	142	2.8	111	15	6.8	3.25	26	8.15	7.8	10800	72	28	0	0	0
12	138	2.8	96	20	6.6	2.90	30	11.25	10.9	14700	89	11	0	0	0
24	138	2.8	93	25	7.2	2.40	32	11.75	10.6	18900	78	22	0	0	0
36	134	2.4	88	45	7.6	2.35	33	11.35	10.7	26800	72	28	0	0	0
48	132	2.6	89	50	7.8	3.05	34	11.45	11.9	27200	73	27	0	0	0

APPENDIX 14

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %
-48	154	3.6	110	15	7.4	1.90	20
-36	142	3.6	95	15	6.2	1.55	24
-24	146	3.4	111	15	6.6	1.55	19
-12	146	3.4	105	15	6.6	1.55	19
0	148	3.2	102	15	6.0	1.55	18
12	150	3.2	96	10	6.1	1.55	23
24	144	3.0	96	25	6.6	1.60	20
36	138	3.0	91	25	6.6	1.60	20
48	138	3.2	85	25	6.0	2.05	20
60	134	2.4	91	35	6.8	2.35	22
72	130	2.4	90	40	6.8	1.55	20

Animal C - 2

RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
8.05	7.3	10900	88	12	0	0	0
8.50	7.9	12200	88	12	0	0	0
7.20	6.5	12600	62	38	0	0	0
8.05	6.5	12200	62	38	0	0	0
6.55	6.1	9100	64	36	0	0	0
8.55	7.9	16400	74	26	0	0	0
7.75	7.9	19000	56	44	0	0	0
7.30	7.0	12800	57	43	0	0	0
9.10	7.9	15200	59	41	0	00	0
8.80	7.2	15300	73	27	0	0	0
7.75	7.4	11500	78	22	0	0	0

APPENDIX 15

Animal C-3

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	144	3.2	111	15	5.9	2.05	30	9.65	10.2	12100	60	40	0	0	0
-36	152	2.8	114	20	5.7	2.65	34	11.35	11.9	12800	73	27	0	0	0
-24	144	2.4	116	15	5.7	2.65	29	9.50	10.3	14000	63	37	0	0	0
-12	144	2.4	116	15	5.6	2.75	29	10.35	10.3	13000	77	23	0	0	0
0	154	2.8	111	25	6.2	2.90	28	11.30	9.6	13200	79	21	0	0	0
12	136	2.4	100	15	5.7	2.75	39	13.75	14.0	6900	96	4	0	0	0
24	138	2.8	93	35	5.9	2.75	38	13.10	13.2	5800	92	8	0	0	0
36	134	2.8	91	40	6.0	2.75	40	13.25	12.9	5800	88	12	0	0	0
48	134	2.6	89	35	6.0	2.90	42	13.75	14.3	6200	90	10	0	0	0

APPENDIX 16 Animal C - 4

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	154	2.8	110	15	7.6	3.25	24	8.90	8.4	9750	39	61	0	0	0
-36	138	2.4	111	20	7.6	2.90	30	10.00	9.8	11300	55	45	0	0	0
-24	144	3.2	110	15	7.8	3.05	25	9.15	7.6	12500	73	27	0	0	0
-12	140	3.2	110	15	7.8	3.05	25	8.50	7.3	10900	44	56	0	0	0
0	142	2.4	108	15	7.0	3.05	25	7.85	7.9	10600	75	25	0	0	0
12	140	3.2	95	15	7.2	2.40	34	11.70	10.9	18600	72	28	0	0	0
24	130	3.8	91	20	7.6	2.40	31	11.25	10.8	19900	73	27	0	0	0
36	136	2.8	89	40	8.0	4.25	33	10.45	10.8	25800	76	24	0	0	0
48	132	4.0	90	50	9.0	4.00	31	10.35	10.6	16200	72	28	0	0	0

APPENDIX 17

Animal C - 5

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	144	4.4	108	15	9.0	2.80	33	11.60	9.9	12900	44	56	0	0	0
-36	140	3.6	108	20	7.0	2.90	34	10.10	10.8	12700	72	27	0	0	0
-24	144	3.2	110	15	7.8	2.55	30	9.55	10.0	11800	76	24	0	0	0
-12	144	3.2	110	15	7.0	2.55	30	9.95	10.7	11800	77	23	0	0	0
0	144	3.6	108	15	7.4	3.00	32	10.45	10.8	11300	42	58	0	0	0
12	138	2.8	96	15	6.2	2.80	35	10.65	11.1	13700	42	58	0	0	0
24	138	3.6	97	20	6.8	3.00	35	9.90	10.5	15700	68	32	0	0	0
36	134	2.4	89	30	6.4	3.05	33	10.80	10.0	15500	65	35	0	0	0
48	136	3.2	91	30	6.6	3.00	31	11.10	10.3	25000	78	22	0	0	0
60	134	2.4	85	45	6.4	2.90	30	11.60	10.3	34900	94	6	0	0	0
72	130	4.0	85	40	6.4	2.50	30	11.00	10.2	18600	61	39	0	0	0

APPENDIX 18 Animal C - 6

Time Hrs	Na^+ mEq/L	K^+ mEq/L	Cl^- mEq/L	BUN mg%	Plasma protein gm%	Albumin gm%	PCV %	RBC $10^6/mm^3$	Hb gm%	WBC count	N	L	B	E	M
-48	144	3.2	111	15	9.0	2.05	25	9.90	9.4	10700	32	68	0	0	0
-36	138	2.4	111	15	7.0	2.25	31	11.75	10.4	11100	57	43	0	0	0
-24	144	2.4	116	20	7.4	3.05	25	10.05	11.7	11700	63	37	0	0	0
-12	140	3.2	110	15	6.8	2.25	24	9.95	12.0	11200	61	39	0	0	0
0	154	2.8	111	15	6.6	2.05	25	10.00	10.4	12100	58	42	0	0	0
12	140	3.2	95	20	6.8	3.05	31	11.75	12.8	16800	73	27	0	0	0
24	138	2.8	93	25	6.2	2.80	30	12.05	14.6	19200	79	21	0	0	0
36	136	2.8	89	30	6.6	2.90	33	13.75	12.6	17700	88	22	0	0	0
48	132	2.6	89	35	6.9	3.05	35	13.25	13.7	15800	96	4	0	0	0

KEY TO APPENDICES 19-36

19-24 - Individual Urinary parameter values for group A.

25-30 - Individual Urinary parameter values for group B.

31-36 - Individual Urinary parameter values for group C.

Appendix: 19 Animal: A-1

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.032	8.0	6.0	62	6.0
-36	1.032	8.0	4.0	62	6.0
-24	1.031	8.0	6.0	62	5.0
-12	1.035	8.0	5.0	60	4.0
0	1.033	8.0	6.0	58	6.0
12	1.036	7.0	5.0	58	3.0
24	1.040	7.0	5.0	60	3.0
36	1.046	7.0	5.0	58	3.0
48	1.051	6.0	3.0	68	3.0
60	1.053	6.0	2.0	72	2.0

APPENDIX: 20. Animal: A-2

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	NS	NS	NS	NS	NS
-36	1.032	8.0	8.0	54	6.0
-24	1.030	8.0	6.0	60	6.0
-12	1.035	8.0	8.0	60	6.0
0	1.022	8.0	4.0	36	4.0
12	1.030	8.0	4.0	36	5.0
24	1.045	8.0	3.0	58	2.0
36	1.042	8.0	3.0	54	1.0
48	NS	NS	NS	NS	NS
60	1.045	6.0	3.0	66	0.5

NS: No urine sample available.

APPENDIX: 21 Animal: A - 3

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	NS	NS	NS	NS	NS
-36	1.030	8.0	4.0	60	6.0
-24	1.032	8.0	6.0	60	5.0
-12	1.032	8.0	5.0	62	6.0
0	NS	NS	NS	NS	NS
12	1.026	7.0	4.0	48	4.0
24	1.034	7.0	4.0	58	2.0
36	NS	NS	NS	NS	NS
48	1.042	6.0	3.0	60	1.0
60	1.052	6.0	3.0	60	0.2

NS: No urine sample available.

APPENDIX: 22 Animal: A-4

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	NS	NS	NS	NS	NS
-36	1.032	9.0	6.0	56	5.0
-24	1.038	9.0	6.0	58	6.0
-12	NS	NS	NS	NS	NS
0	1.033	8.0	5.0	52	6.0
12	1.044	8.0	5.0	54	3.0
24	1.043	8.0	5.0	54	3.0
36	1.043	7.0	4.0	68	2.0
48	1.043	6.0	4.0	62	0.5
60	1.053	6.0	3.0	62	0.3

NS: No urine sample available

APPENDIX: 23 Animal: A-5

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.031	8.0	8.0	60	6.0
-36	1.030	8.0	6.0	58	6.0
-24	1.030	8.0	5.0	58	6.0
-12	1.033	8.0	6.0	60	5.0
0	1.033	8.0	5.0	60	6.0
12	1.033	8.0	5.0	62	2.0
24	1.047	6.0	5.0	56	1.0
36	1.044	6.0	3.0	62	1.0
48	1.052	6.0	4.0	60	0.5
60	1.051	6.0	3.0	60	0.3

APPENDIX: 24 Animal: A - 6

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.033	8.0	6.0	62	5.0
-36	1.033	8.0	6.0	54	5.0
-24	1.032	8.0	4.0	54	6.0
-12	1.035	8.0	5.0	56	5.0
0	1.035	8.0	4.0	50	6.0
12	1.036	7.0	3.0	62	4.0
24	1.040	7.0	3.0	58	3.0
36	1.044	7.0	4.0	62	2.0
48	1.052	6.0	3.0	60	1.0
60	1.053	6.0	3.0	60	0.5

APPENDIX: 25 Animal: B - 1

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.030	8.0	8.0	56	6.0
-36	1.030	8.0	6.0	40	6.0
-24	1.032	8.0	6.0	48	5.0
-12	1.032	8.0	7.0	56	5.0
0	1.030	8.0	7.0	56	5.0
12	1.051	7.0	5.0	58	3.0
24	0.050	6.0	4.0	54	3.0
36	0.053	6.0	4.0	60	2.0
48	0.051	5.0	3.0	62	1.0

APPENDIX: 26 Animal: B - 2

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	NS	NS	NS	NS	NS
-36	1.031	8.0	7.0	58	6.0
-24	1.030	8.0	6.0	60	8.0
-12	1.032	8.0	6.0	60	6.0
0	1.032	8.0	6.0	60	6.0
12	1.050	7.0	5.0	40	5.0
24	1.047	7.0	5.0	56	3.0
36	1.050	6.0	4.0	60	3.0
48	1.052	5.0	3.0	60	1.0

NS: No urine sample available.

APPENDIX: 27 Animal : B - 3

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.030	8.0	5.0	48	8.0
-36	1.030	8.0	6.0	40	6.0
-24	1.030	8.0	5.0	48	6.0
-12	1.027	8.0	6.0	50	6.0
0	1.030	8.0	5.0	52	6.0
12	1.054	7.0	4.0	54	5.0
24	1.053	7.0	4.0	52	4.0
36	1.053	6.0	3.0	56	2.0
48	1.051	5.0	3.0	56	2.0

APPENDIX: 28 Animal : B - 4

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.030	8.0	6.0	58	8.0
-36	1.030	8.0	5.0	56	8.0
-24	1.030	8.0	5.0	58	7.0
-12	1.030	8.0	5.0	60	6.0
0	1.032	8.0	5.0	60	6.0
12	1.053	7.0	4.0	58	5.0
24	1.053	7.0	4.0	60	5.0
36	1.052	6.0	4.0	62	4.0
48	1.055	5.0	3.0	66	2.0

APPENDIX: 29 Animal: B - 5

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.030	8.0	6.0	58	6.0
-36	1.031	8.0	6.0	58	8.0
-24	1.037	8.0	6.0	56	6.0
-12	1.036	8.0	5.0	60	6.0
0	1.0 36	8.0	6.0	52	5.0
12	1.046	7.0	5.0	58	6.0
24	1.052	6.0	4.0	60	5.0
36	1.052	6.0	3.0	56	4.0
48	1.052	6.0	2.0	58	2.0

APPENDIX: 30 Animal: B-6

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.036	8.0	5.0	62	6.0
-36	1.036	8.0	6.0	60	6.0
-24	1.041	8.0	5.0	60	5.0
-12	1.041	8.0	6.0	60	5.0
0	1.041	8.0	5.0	58	5.0
12	1.044	7.0	4.0	60	5.0
24	1.046	6.0	3.0	60	4.0
36	1.055	6.0	3.0	60	3.0
48	1.055	6.0	3.0	60	2.0

APPENDIX: 31 Animal: C - 1

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.030	8.0	8.0	63	8.0
-36	1.032	8.0	8.0	66	6.0
-24	1.030	8.0	7.0	60	6.0
-12	1.036	6.0	6.0	62	5.0
0	1.033	7.0	7.0	60	6.0
12	1.044	7.0	6.0	58	5.0
24	1.047	6.0	6.0	62	4.0
36	1.053	6.0	5.0	62	3.0
48	1.051	6.0	4.0	60	1.0

APPENDIX : 32 Animal: C - 2

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.030	8.0	8.0	60	6.0
-36	1.026	8.0	6.0	58	5.0
-24	1.028	8.0	6.0	60	6.0
-12	1.035	7.0	6.0	62	6.0
0	1.030	7.0	8.0	60	7.0
12	1.040	7.0	5.0	60	5.0
24	1.042	6.0	4.0	58	4.0
48	1.040	6.0	4.0	48	2.0

APPENDIX: 33 Animal: C - 3

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.030	8.0	8.0	62	6.0
-36	1.032	8.0	6.0	62	8.0
-24	1.026	7.0	6.0	60	6.0
-12	1.040	7.0	6.0	56	7.0
0	1.032	7.0	5.0	56	6.0
12	1.041	6.0	5.0	60	5.0
24	1.043	6.0	6.0	62	5.0
36	1.051	6.0	5.0	58	3.0
48	0.052	6.0	4.0	66	1.0

APPENDIX: 34 Animal: C - 4

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.016	8.0	6.0	58	6.0
-36	1.030	8.0	7.0	58	8.0
-24	1.032	8.0	6.0	60	6.0
-12	1.037	7.0	6.0	56	8.0
0	1.034	7.0	5.0	58	8.0
12	1.042	7.0	5.0	58	7.0
24	1.046	6.0	4.0	60	5.0
36	1.046	6.0	3.0	62	3.0
48	1.050	6.0	3.0	62	1.0

APPENDIX: 35 Animal : C - 5

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.022	8.0	8.0	56	7.0
-36	1.025	8.0	8.0	52	7.0
-24	1.030	8.0	8.0	50	6.0
-12	1.032	8.0	7.0	52	6.0
0	1.026	7.0	6.0	52	5.0
12	1.040	7.0	5.0	54	4.0
24	1.042	6.0	5.0	52	3.0
36	1.041	6.0	4.0	60	3.0
48	1.053	6.0	3.0	58	1.0

APPENDIX: 36 Animal : C - 6

Time Hrs	Specific Gravity	pH	Na^+ mEq/hr	K^+ mEq/hr	Cl^- mEq/hr
-48	1.026	8.0	8.0	58	6.0
-36	1.032	8.0	8.0	56	6.0
-24	1.031	7.0	7.0	58	5.0
-12	1.035	7.0	6.0	58	6.0
0	1.040	7.0	7.0	56	6.0
12	1.048	7.0	5.0	36	5.0
24	1.052	6.0	4.0	40	4.0
36	1.050	6.0	4.0	52	2.0
48	1.051	6.0	3.0	52	0.5

Appendix: 37 - Erythrocyte and leucocyte count in the Peritoneal fluid for individual goats in group A.

		TIME IN HOURS									
Animal	Parameter	-48	-36	-24	-12	0	12	24	36	48	60
A1	RBC	700	634	300	190	64	0	0	0	0	0
	WBC	1190	1200	1200	1200	2000	2200	5000	4200	5340	7900
A2	RBC	799	584	364	260	100	0	0	0	0	0
	WBC	2000	2600	2600	2880	3100	3100	4600	5200	6670	9800
A3	RBC	800	600	480	80	0	0	0	0	0	0
	WBC	1100	1360	1300	2200	3460	4100	5000	5100	5100	5800
A4	RBC	560	388	559	0	0	0	0	0	0	
	WBC	990	900	780	1200	1400	1880	2200	3600	3800	
A5	RBC	590	300	260	115	0	0	0	0	0	
	WBC	1550	1500	1300	2100	2260	3300	3600	5100	6800	
A6	RBC	700	590	264	60	0	0	0	0	0	
	WBC	1200	1200	1200	2100	2300	4100	4800	6600	9000	

Appendix: 38 - Erythrocyte and Leucocyte Count in the Peritoneal fluid for individual goats in group B

		TIME IN HOURS							
Animal	Parameter	-48	-36	-24	-12	0	12	24	36
B1	RBC	890	550	210	0	0	0	200	260
	WBC	1100	1600	2600	2800	1900	9900	5800	7800
B2	RBC	780	500	264	80	0	0	115	200
	WBC	1300	1400	2200	3100	2000	2300	5000	9900
B3	RBC	580	300	120	0	0	0	210	200
	WBC	1120	1150	1500	1500	1600	2100	5100	6800
B4	RBC	500	300	260	0	0	115	120	150
	WBC	1500	1300	2200	2260	2000	3800	6000	7900
B5	RBC	700	380	120	0	0	150	120	210
	WBC	1100	1200	1360	1600	1680	2600	3400	5100
B6	RBC	660	410	210	0	0	0	200	180
	WBC	1300	1100	1300	1560	1500	2200	3900	5880

Appendix: 39 - Erythrocyte and leucocyte count in the peritoneal fluid for individual goats in group C

		TIME IN HOURS								
Animal	Parameter	-48	-36	-24	-12	0	12	24	36	48
C1	RBC	900	540	264	0	0	80	130	150	270
	WBC	1360	1300	1680	1600	1600	2300	5000	5900	7700
C2	RBC	560	380	180	0	0	0	115	120	250
	WBC	1100	1200	1100	1550	1500	1820	2200	3800	5900
C3	RBC	700	300	60	0	0	0	200	250	210
	WBC	1560	1320	1300	1360	1500	2100	2600	3300	5510
C4	RBC	780	310	170	0	0	120	210	380	300
	WBC	1220	1200	1200	1300	1550	2200	2300	5100	7800
C5	RBC	560	260	150	80	0	0	210	380	580
	WBC	1200	1250	1080	1500	2000	2500	3800	3100	5900
C6	RBC	700	300	110	0	0	0	200	260	300
	WBC	1100	1200	1200	1700	2800	3800	5900	7700	9000

Appendix 40 - ANOVA for comparison of effect of variariation of site and duration of obstruction on blood parameters.

Appendix 41 - ANOVA for comparison of effect of variation of site and duration of obstruction on urinary parameters.

Appendix: 40

Parameter	Source of Variation	Degrees of Freedom
Sodium Concentration	Treatments	2
	Time	8
	Total Observations	161
	Error	151
Potassium Concentration	Treatments	2
	Time	8
	Total Observations	161
	Error	151
Chloride Concentration	Treatments	2
	Time	8
	Total Observations	161
	Error	151

Sum of Squares	Mean Sum of Squares	F - Ratio
828.16	414.08	11.57**
1 31.90	16.49	0.46
6491		
5531.01	35.78	
40.07	20.04	64.45**
9.18	1.15	3.76**
95		
46.24	0.31	
3521.64	1760.82	48.46**
19483.38	2435.42	67.02**
28492		
5487.09	36.34	

Appendix: 40 continued

Parameter	Source of Variation	Degrees of Freedom	Sum of Squares	Mean Sum of Squares	F-Ratio
Blood Urea Nitrogen	Treatments	2	523.12	261.56	1.87**
	Time	8	31014.90	3876.86	27.77**
	Total Observations	161	52619.68		
	Error	151	21081.66		
Packed Cell Volume	Treatments	2	886.75	443.38	21.04**
	Time	8	2067.09	258.39	12.26**
	Total Observations	161	6136		
	Error	151	3182.58	21.08	
Erythrocyte Count	Treatments	2	1414.45	707.22	64.83**
	Time	8	544.66	68.08	6.24**
	Total Observations	161	3606		
	Error	151	1647.15		

Appendix: 40 continued

Parameter	Source of Variation	Degrees of Freedom	Sum of squares	Mean Sum of squares	F-Ratio
Haemoglobin Concentration	Treatments	2	58.26	29.13	9.74**
	Time	8	79.13	9.89	3.31**
	Total Observations	161	589		
	Error	151	451.70	2.99	
Leucocyte Count	Treatments	2	$1007X10^6$	$50.32X10^6$	58.24**
	Time	8	$3447X10^6$	$431X10^6$	49.87**
	Total Observations	161	$5758X10^6$		
	Error	151	$1304X10^6$	$8.64X10^6$	
Plasma Protein	Treatments	2	25.72	12.86	16.71**
	Time	8	8.08	1.01	1.31
	Total Observation	161	150		
	Error	151	116.18	0.77	

Appendix: 40 continued

Parameter	Source of Variation	Degrees of Freedom	Sum of squares	Mean Sum of squares	F-Ratio
Albumin	Treatments	2	7.47	3.74	14.87**
	Time	8	2.57	0.32	1.27
	Total Observation	161	48		
	Error	151	37.93	0.25	

Appendix: 41

Parameters	Source of Variation	Degrees of Freedom	Sum of Squares	Mean Sum of Squares	F-Ratio
Specific Gravity	Treatments	2	$0.515X10^{-3}$	$0.257X10^{-3}$	14.11^{**}
	Time	8	$9.716X10^{-3}$	$1.214X10^{-3}$	66.64^{**}
	Total Observations	153	0.012836		
	Error	143	$2.605X10^{-3}$	$0.018X10^{-3}$	
pH	Treatments	2	9.37	4.68	32.26^{**}
	Time	8	107.82	13.48	92.92^{**}
	Total Observations	153	137.93		
	Error	143	20.74	0.15	
Sodium Excretion	Treatments	2	77.69	38.84	37.13^{**}
	Time	8	107.87	13.48	12.89^{**}
	Total Observations	153	322.26		
	Error	143	136.70	1.05	

Appendix: 41 continued

Parameters	Source of Variation	Degrees of Freedom	Sum of Squares	Mean Sum of Squares	F-Ratio
Potassium Excretion	Treatments	2	39.26	19.63	0.60
	Time	8	454.20	56.78	1.78
	Total Observations	153	5139.54		
	Error	143	4646.08	32.49	
Chloride Excretion	Treatments	2	30.91	15.46	22.83**
	Time	8	464.41	58.05	85.72**
	Total Observations	153	592.16		
	Error	143	96.84	0.68	