FLUID AND ELECTROLYTE STATUS IN DIARRHOEIC CONDITIONS OF DOGS

By

JABINA MARTHA PHILIP

THESIS

Submitted in partial fulfilment of the requirement for the degree of

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences

Kerala Agricultural University

Department of Clinical Medicine

COLLEGE OF VETERINARY AND ANIMAL SCIENCES

MANNUTHY, THRISSUR - 680 651

KERALA, INDIA

2003

DECLARATION

I hereby declare that the thesis entitled **"FLUID AND ELECTROLYTE STATUS IN DIARRHOEIC CONDITIONS OF DOGS"** is a bonafide record of research work done by me during the course of research and that this thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title of any other University or Society.

Mannuthy
4-8-2003.

JABINA MARTHA PHILIP

CERTIFICATE

Certified that this thesis, entitled **"FLUID AND ELECTROLYTE STATUS IN DIARRHOEIC CONDITIONS OF DOGS"** is a record of research work done independently by **Mrs.Jabina Martha Philip** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to her.

K.M. J 4/8/2003

Dr. K.M. Jayakumar
Associate Professor
Department of Clinical Medicine
College of Veterinary & Animal Sciences
Mannuthy

Mannuthy
4-8-2003

CERTIFICATE

We, the undersigned members of the Advisory Committee of **Mrs. Jabina Martha Philip,** a candidate for the degree of **Master of Veterinary Science in Clinical Medicine,** agree that the thesis entitled **"FLUID AND ELECTROLYTE STATUS IN DIARRHOEIC CONDITIONS OF DOGS"** may be submitted by **Mrs. Jabina Martha Philip,** in partial fulfillment of the requirement for the degree.

K.M.J. 4/8/03

Dr. K.M. Jayakumar
(Chairman, Advisory Committee)
Associate Professor
Department of Clinical Medicine
College of Veterinary and Animal Sciences, Mannuthy

4.8.03

Dr. P.G. Baby
Professor and Head
Department of Clinical Medicine
(Member)

4.8.03

Dr. Usha Narayana Pillai
Assistant Professor
Department of Clinical Medicine
(Member)

Mini 4/8/03

Dr. M. Mini
Assistant Professor
Department of Microbiology
(Member)

EXTERNAL EXAMINER 26/9/0

(M. SUBRAMANIAN)

ACKNOWLEDGEMENT

I wish to express my sincere gratitude to ***Dr.K.M.Jayakumar,*** *Associate Professor, Department of Clinical Medicine and Chairman, Advisory Committee for his keen interest and help.*

I am extremely thankful to ***Dr.P.G.Baby,*** *Professor and Head, Department of Clinical Medicine and* ***Dr.M.Mini,*** *Assistant Professor, Department of Microbiology for their support, invaluable suggestions and constant encouragement. Their support and guidance as members of the advisory committee is greatly acknowledged.*

My sincere thanks are due to ***Dr.P.C.Alex,*** *Associate Professor and Head, University Veterinary Hospital, Kokkalai and* ***Dr.Usha Narayana Pillai,*** *Assistant Professor, Department of Clinical Medicine, who took utmost personal interest in this work and rendered whole-hearted help. Had it not been for the sincere encouragement and intervention at critical moments my endeavor could not have been fructified.*

Encouragement given by ***Dr.S.Ajith Kumar,*** *Assistant Professor,* ***Dr.Premni Aleyas,*** *Assistant Professor,* ***Dr.Vinu David,*** *Research Associate, Department of Clinical Medicine are thankfully acknowledged.*

I am greatly indebted to ***Dr.A.M.Chandrashekaran Nair,*** *Associate Professor, Department of Pharmacology and Toxicology for carrying out the serum electrolyte assay, and* ***Mrs. M. Sujatha,*** *Assosiate Professor and Head, Department of Statistics for the help offered in analysing the data.*

The support and company given by my departmental colleagues ***Drs. Arun George, P.Muraly, O.K.Sindhu, R.Renjith, Siby Antony, Rani Gopinath*** *and* ***Udayasree*** *are sincerely acknowledged.*

I sincerely acknowledge the assistance rendered by ***Dr.Manju Soman,*** ***Dr. P.X.Antony, Dr.Chintu Ravishankar*** *and* ***Dr.M. Suresh*** *for their timely help.*

I extend my gratefulness to my friends and fellow Post-Graduate students for their help, encouragement and concern shown throughout the study.

My sincere thanks are due to the non-teaching staff of the Department of Clinical Medicine and College Hospitals for their Co-operation.

I wish to express my thanks to ***Dr. E.Nanu****, Dean, College of Veterinary and Animal Sciences, Mannuthy and* ***Dr.P.P.Balakrishnan****, the Special Officer, Pookot Veterinary College, for providing the facilities required for the work.*

I am thankful to Kerala Agricultural University for granting me contingent expenditure towards the work.

Great care and concern shown in giving good shape to its present form of this thesis by Mr.Jomon, M/s.JMJ Computer Centre, Thottappady is sincerely acknowledged.

I thankfully remember all those whom directly or indirectly helped me and contributed towards the fulfillment of my research work.

Although my husband, pappa, mummy and in-laws, who are part and parcel of my being do not require mention, my entire self is filled with love and affection for them for the support given by them to pursue the work even in the midst of the most adverse and turbulent conditions.

And, finally bowing my head in prayer before the Almighty, for all He has done.

JABINA MARTHA PHILIP

CONTENTS

LIST OF ABBREVIATIONS

CPV	-	Canine Parvo Virus
DLC	-	Differential Leucocyte Count
E. coli	-	*Escherichia coli*
ECF	-	Extra Cellular Fluid
EDTA	-	Ethylene Diamine Tetra Acetic Acid
ESR	-	Erythrocyte Sedimentation Rate
HA	-	Haemagglutination
Hb	-	Haemoglobin
PCV	-	Packed Cell Volume
RBC	-	Red Blood Corpuscle
TEC	-	Total Erythrocyte Count
TLC	-	Total Leucocyte Count
WBC	-	White Blood Corpuscle
µl	-	Micro liter
g/dl	-	Grams per deciliter
mEq/L	-	Milli equivalents per liter
m mol/L	-	Milli moles per liter
ml/kg	-	Milliliter per kilogram
mg/kg	-	Milligram per Kilogram
min	-	Minutes
ml	-	Milliliter
%	-	Per cent

LIST OF TABLES

LIST OF FIGURES

Introduction

1. INTRODUCTION

Diarrhoea, an abnormal increase in the frequency, fluidity or volume of faeces resulting from excessive faecal water content is one of the most common clinical conditions reported in the veterinary hospitals of Kerala. As it is a readily observable symptom, prompt veterinary attention is sought by pet owners. It is one of the most important clinical manifestations of intestinal diseases in dogs. Diarrhoea may be the only sign of ill health in animals or the salient sign that accompanies other abnormalities such as anorexia, weight loss, vomiting etc. and provides us a window of opportunity to evaluate the gastrointestinal tract and other systems of the body.

Diagnosis of correct etiological agents causing diarrhoea has been a subject of utmost interest from time immemorial. A multi-dimensional approach to evaluate cases of diarrhoea involve history taking, clinical examination, haematological and biochemical analysis, plasma volume estimation, parasitological and microbiological studies, including faecal culture. A thorough study of all the above aspects is very essential for the effective management and treatment of canine diarrhoea.

Regardless of the cause, diarrhoea usually leads to rapid development of dehydration and electrolyte imbalances, which are responsible for many of the clinical signs. Therefore, in the treatment of diarrhoea, the basic objectives should be to adopt a therapeutic regimen to give immediate relief, alleviate the symptoms with correction of fluid and electrolyte imbalances, if any.

The assessment of plasma volume and electrolyte status will help the clinician to find out the extent of dehydration and electrolyte imbalances, which in turn will help in formulating better therapeutic measures in the management of diarrhoea in dogs. Above all, the professional skill, competence and practical experience of a clinician has to be relied upon in the diagnosis and management of diarrhoea. Only very little work has been done in diarrhoea affected dogs in Kerala. The available reports mainly deals with parvoviral infection, which produce severe haemorrhagic-gastroenteritis and is considered to be the most important and prevalent etiological agent in Kerala.

Therefore a work on "Fluid and electrolyte status in diarrhoeic conditions of dogs" was carried out to understand the effects of diarrhoea on

i) fluid and serum electrolyte status (Na^+, K^+, Cl^-, HCO_3^-) and plasma proteins,

ii) the haemogram of the affected dogs and

iii) to find out the possible etiology of this condition for suggesting suitable therapeutic measures.

Review of Literature

2. REVIEW OF LITERATURE

2.1 DEFINITION

Diarrhoea was defined as a condition characterized by abnormal frequency and looseness of faecal matter, usually not considered primarily as a disease but as a symptom resulting from a variety of causes within or outside the gastrointestinal tract (Bloom, 1959).

Strombeck and Guileford (1991) defined diarrhoea as a change in the frequency, consistency or degree of bowel movements. Water content was recorded as 60 to 80 per cent in normal faeces and 70 to90 per cent in unformed and watery faeces.

Radostits *et al.* (1994) described diarrhoea as the increased frequency of defaecation accompanied by an elevated concentration of water and decrease in dry matter content; the consistency of faeces varied from solid to liquid.

2.2 CLASSIFICATION

Bloom (1959) classified diarrhoea as infectious or non-infectious, fermentative or putrefactive, mild or severe, functional or organic, acute or chronic, intermittent, continuous or recurrent and mucoid, watery, bloody or muco-purulent.

2.3 ETIOLOGY

Diarrhoea might be caused by protozoan, fungal, bacterial or viral infections, intestinal parasites, glandular dysfunction, mechanical factors, vitamin deficiencies, allergic factors, poisonings, psychogenic factors and a number of miscellaneous causes (Bloom, 1959).

2.3.1 Bacteria

Canine *Salmonellosis* was a bacterial infection of the intestinal tract particularly involving the posterior part of the bowel. The symptoms most often reported were diarrhoea, septicaemia, weakness, recumbency, elevated temperature and ocassionally abortion (James *et al.*, 1963). Moon (1974) classified colibacillosis in animals into four groups (i) enterotoxaemia caused by enterotoxin producer, (ii) enterotoxaemia resulting in systemic disorders following absorption of an intestinal toxin, (iii) enteroinvasive caused by invasive *Escherichia coli* (*E. coli*) and resulting in varying degrees of destruction of mucosa and (iv) septicaemic which results in extra intestinal localization of *E. coli*. Vandenberghe *et al.* (1982) isolated *Campylobacter jejuni* from faecal samples of 53.8 per cent of dogs with diarrhoea and 8 per cent of dogs without diarrhoea. Vihan and Singh (1989) demonstrated a negative correlation between the age of the kid and incidence of enteric colibacillosis. Accordingly *E. coli* infection took place immediately after birth and peaked at five to six days of age. Radostits *et al.* (1994) also opined that the occurrence of colibacillosis varied with breed, sex, litter-size, birth weight, management and climate. In 106 cases of canine diarrhoea studied by Rodrigues *et al.* (1994) revealed that 25 per cent of the cases were due to bacterial infections, of which *E. coli* was found to be the predominant one. In a study conducted on 200 goats of different age groups, Dubey and Sharda (2001) recorded highest incidence (50%) of colibacillosis in one-week old kids.

Strombeck and Guileford (1991) opined that transient bacteria became pathogenic and produced diarrhoea only when they colonize the bowel, due to a change in the homeostatic mechanisms. Ludlow and Davenport (2000) mentioned that the normal flora of the small intestine consisted of a variety of aerobic bacteria including *E. coli, Staphylococcus* sp., *Streptococcus* sp., *Pasteurella* sp., *Proteus* sp. and *Corynebacterium* sp. and also anaerobic bacteria.

Of the fifteen cases of haemorrhagic gastroenteritis in dogs studied by Spielman and Garvey (1993), *E. coli* was isolated from 13 dogs and *Clostridium* sp. from two of the diseased animals. In the control group, all were positive for *E. coli* and one for *Clostridium* sp. Clinical survey conducted by Heemyung *et al.* (1999) in pups with concurrent intestinal and respiratory tract disorders, the incidence of *E. coli* and *Staphylococcus* sp. infections was found to be 72.7 per cent and 40.9 per cent respectively.

Vandersteen *et al.* (1997) reported that severe acute diarrhoea and necrotic enteritis associated with high amounts of *C. perfringens* often developed in dogs after drastic changes in the protein content of their diet. Weese *et al.* (2001) isolated *C. difficile* from two per cent of dogs with diarrhoea but was not isolated from the faeces of normal dogs, possibly because of poor survival of the organism in faecal sample.

2.3.2 Virus

Viral gastroenteritis had emerged in the last decade as a major cause of morbidity and mortality in many animal species. Four main types of viruses namely parvovirus, rotavirus, astrovirus and corona virus have been demonstrated in the faeces of dogs and are known to be associated with canine gastroenteritis (Appel *et al.*, 1979; Hammond and Timoney, 1983). Bund and Laohasurayothin (1982) found that highest prevalence of canine parvoviral (CPV) infection was at three to four months of age in dogs. Marshall *et al.* (1984) noted that viruses of seven distinct types, some of known pathogenicity were excreted by non-diarrhoeic mature apparently healthy dogs; of 157 normal faeces studied 34 per cent contained parvovirus-like particles, five per cent contained corona virus, three per cent contained a previously undescribed "round" virus particles, two per cent contained rota virus, two per cent contained astrovirus-like particles, one per cent contained papova-like virus and one per cent contained coronavirus like particles. Mochizuki *et al.* (1993) isolated a calicivirus strain 48 of canine origin from a dog in Japan by inoculation of faecal matter or rectal swab into Madin Darby Canine Kidney (MDCK) cell monolayer. Here, the clinical manifestations recorded were watery diarrhoea, vomiting, depression and anorexia without fever. Virus strains isolated by Kokubu *et al.* (1993) from 70 dogs with diarrhoea were identified as reovirus on the basis of their biological and psysico-chemical properties. Canine parvovirus, canine corona virus and rotavirus have been incriminated as primary pathogens of viral enteritis in dogs of less than six months of age (Hoskins, 1997). Udupa and Sastry (1997) reported parvoviral enteritis in 79 dogs out of the 86 dogs screened by faecal haemagglutination (HA) or serum haemagglutination inhibition test or both. Studies conducted by Deepa (1999) among 57 clinically suspected dogs for parvoviral infection, 50.7 per cent and 19.3 per cent respectively had CPV antigen in the faecal samples by Agar Gel Immuno-Diffusion (AGID) and Counter

Immuno Electrophoresis (CIEP). Mousa *et al.* (2001) isolated canine adenovirus on Madin Darby Bovine Kidney (MDBK) cell culture from rectal swabs of six young dogs suffering from abdominal pain, bloody diarrhoea with or without vomiting. Banja *et al.* (2002) found that vomiting and diarrhoea were seen in majority of dogs suffering from corona virus infection. Haemorrahagic diarrhoea and vomiting were the most important clinical manifestations of CPV mixed infection. Clinical signs of mixed infection were indistinguishable from that of single infection and the duration of illness was prolonged in the latter. Culture and sensitivity study of parvoviral enteritis revealed that all the cases were found to be complicated by *E. coli* infection (Ramprabhu *et al.*, 2002).

2.3.3 Fungus

Rodriguez *et al.* (1998) observed that *Candida* sp. could spread systemically from gastrointestinal lesions and the risk of secondary candidosis should be kept in mind in cases of parvoviral infections in young dogs.

2.3.4 Parasites

Rachman and Pollock (1961) recognized coccidiosis as the most important protozoan disease of dogs due to its frequency of occurrence and pathogenicity. According to Lindsay (1989) most of coccidial outbreaks occur during the summer and autumn months, when high temperature and humidity favour the rapid sporulation of oocyst. Twenty five percentage of the 106 cases of canine diarrhoea studied by Rodrigues *et al.* (1994) were due to intestinal parasitic infestation. Out of these, 70.27 per cent were due to hookworms. According to Navarro *et al.* (1997), 2.26 per cent of faecal samples taken from dogs with diarrhoea were positive for *Cryptosporidium* species. The infected dogs were three to eight months of age. Duda *et al.* (1998) detected *Blastocystis* sp. from four out of six diarrhoeic dogs aged between two months and nine years. In a study of 390 dogs of various breeds of different age groups and sexes for faecal prevalence of eggs of gastro intestinal helminths in Malve region of Madhya Pradesh for two years, Asati *et al.* (1999) revealed highest incidence of *Ancylostoma caninum* in 81 dogs (20.76%) followed by *Toxocara canis* in 36 dogs (9.23%); *Dipylidium caninum* in 20 dogs (5.12%) and mixed infection of *A. caninum* and *T. canis* in five dogs (1.28%). The overall incidence of parasitism below one year of age was recorded as 22.82 per cent where as in those above one year of age it was 13.58 per cent. According to Koene and Houwers (1999) diarrhoea was a common problem in young animals living in a densely populated environment such as kennel and one possible causative agent was *Giardia.* Itoh and Muraoka (2001) also detected *Giardia* cysts in faeces of ten dogs with diarrhoea. Itoh and Muraoka (2002) detected *isospora* oocysts in 6.2 per cent of animals examined and the rates of *isospora* oocyst detection were high in soft and diarrhoeic faeces, with mucus or blood. Ruckstuhl *et al.* (2002) opined that the most often observed clinical signs in *Trichuris vulpis* infection were mild to moderate diarrhoea and weight loss. The severity of clinical signs depends on the number of parasites, the age of infection and may even breed dependent.

2.3.5 Nutritional factors

Vandersteen *et al.* (1997) concluded that a high protein diet increased faecal counts of *C. perfringens* and enhanced enterotoxin production by clostridial species, which might persist in infected dogs for months and was not reliably eliminated by antibiotic treatment. According to Deepti *et al.* (1999) diarrhoea not only occurred due to pathogens alone but also was influenced by the effects and interactions with nutrition and managemental practices.

2.3.6 Miscellaneous factors

Jergens *et al.* (1992) observed diarrhoea, vomiting, melena, abdominal pain and weight loss in idiopathic inflammatory bowel disease associated with gastroduodenal ulcertaion-erosion in dogs. Diarrhoea was reported to be the predominant clinical sign in dogs and cats with colitis associated with inflammatory bowel disease (Leib and Matz, 1995).

2.4 CLINICAL MANIFESTATIONS ASSOCIATED WITH DIARRHOEA

Weaver (1977) described the commonest triad of presenting signs in intestinal intussusception as vomiting, diarrhoea and a palpable abdominal mass in dogs. Severe anorexia, weakness, moderate or high fever, recurrent vomiting, abdominal pain, watery and blood stained diarrhoea were observed in dogs with parvo viral enteritis in Hungary (Voros *et al.*, 1981). Experimentally induced CPV infection in young pups by Sullivan *et al.* (1984) were characterized by mild to moderate depression in early stages; anorexia and fever were first noted on day five. Pups were moderately to severely affected by day six, when diarrhoea and vomiting commenced. All animals were dehydrated and developed very foul smelling watery, reddish mucoid diarrhoea by day seven. According to Udonsi and Agunama (1991), the clinical signs observed in canine ancylostomosis were diarrhoea some times with occult blood, general inappetance, whitish woolly tongue, loss of hair, vomiting, enlarged abdomen and oedema of legs. Levitt and Bauer (1992) reported that the presenting signs in dogs and cats with intussusception were varied and non-specific and could be seen in a number of acute abdominal conditions and comprised of vomiting, bloody mucoid diarrhoea and a palpable abdominal mass. Based on clinical and laboratory assessment of hydration status of neonatal calves with diarrhoea, Constable *et al.* (1998) suggested that the best predictors of degree of dehydration were extent of exophthalmous, skin elasticity on neck and thorax and the plasma protein concentration. Leib and Zajac (1999) reported that acute small bowel diarrhoea was the most common clinical syndrome associated with giardiasis in dogs. Joshi *et al.* (2001) demonstrated that experimental CPV infection produced mild to moderate enteric form of infection characterized by diarrhoea of varying severity, fever and leucopaenia. Infected pups were dull and anorectic. Haemorrhagic gastroenteritis in young pups manifested as high rise of body temperature, which declined to subnormal later, inappetance, polydipsia, froathy yellow vomitus, retching and restlessness. The faeces were brownish, semisolid and mixed with excess mucus which later became foetid and fluidy. All the animals had varying degree of dehydration and exhaustion (Ramprabhu *et al.*, 2002).

2.5 HAEMATOLOGY

The reference range of haematological parameters for normal dogs postulated by Brar *et al.* (2000) were

RBCs ($x10^6/\mu l$)	- 5-8
Haemoglobin (g/dl)	- 10-16
PCV (%)	- 30-50
WBC count (per µl)	- 6,000-16,000
Neutrophils (per µl)	
Mature (%)	- 60-70
Bands (%)	- 0-30
Lymphocytes (%)	- 15-30
Monocytes (%)	- 3-8
Eosinophils (%)	- 2-10
Basophils	- rare

2.5.1 Parasitic conditions

Morgan (1967) studied the effects of helminth parasites on the haemogram of dogs and reported that hookworms like *Ancylostoma caninum* causing enteritis resulted in low packed cell volume (PCV), haemoglobin (Hb) and total erythrocyte count (TEC). The erythrocyte sedimentation rate (ESR) was increased probably due to the result of both anaemia and concurrent hypoalbuminaemia. Udonsi and Agunama (1991) noted a negative correlation between parameters like PCV and haemoglobin with faecal egg counts in dogs infected with *Ancylostoma caninum*. Leucocytosis was observed by Rodrigues *et al.* (1994) in canine diarrhoeal cases due to parasitic infection. Maiti *et al.* (1999) conducted clinico-haematological and therapeutic studies in an outbreak of parasitic gastroenteritis in sheep and the investigations revealed a significant reduction in haemoglobin and TEC in infected sheep than non-infected group. The eosinophil and neutrophil counts were significantly higher whereas the lymphocytic counts were lower in the infected animals.

2.5.2 Non specific conditions.

2.5.2.1 Bovine

Michell (1974) opined that the loss of extra cellular fluid (ECF) in cases of diarrhoea in calves led to haemoconcentration with an increase in PCV. A fall in sodium ion concentration allows more water to enter into the cells reinforcing the rise in PCV caused by reduced plasma volume without changing Hb level. Deshpande *et al.* (1993) found a significant increase in Hb level, PCV and TEC in diarrhoric calves.

2.5.2.2 Canine

Haematological changes in dogs suffering from diarrhoea were characterized by significant increase in PCV, TEC and protein values possibly due to dehydration (Jani *et al.*, 1992). Similar findings were reported by Cornelius (1972) and Jones and Liska (1986) in dehydrated dogs. Zafar *et al.* (1999) reported that the TEC and PCV of diarrhoeic dogs were significally higher when compared with the healthy control group. They also observed that all the dogs with diarrhoea exhibited macrocytic normochromic anaemia.

2.6 HAEMATOLOGY AND HYDRATION STATUS

Chen and Shien (1981) observed that levels of erythrocytes, Hb, PCV, plasma proteins and percentages of neutrophils and lymphocytes were positively related to the degree of dehydration in dogs. The environmental temperature and relative humidity should also be considered while evaluating dehydration in dogs by single regression equation. PCV and total plasma protein concentrations were easily accessible data that could be used to estimate the relative state of hydration of most animals (Roussel and Kasari, 1990). According to Radostits *et al.* (1994), increase in PCV and total serum protein levels indicate the degree of dehydration.

2.6.1 Viral enteritis

In a study of dogs with varying degrees of severity of parvoviral infection, Prange *et al.* (1983) recorded leucopenia, increasing with severity of infection in 29 per cent of cases. Carman and Povey (1985) recorded leukocytosis on day zero, two and five post exposure in experimentally induced CPV-2 infected dogs. Total neutrophil counts remained within normal range. Monocyte and eosinophil numbers varied for individual dogs, but when the group means were assessed, little variations were apparent. Leucopenia was observed by Rodrigues *et al.* (1994) in canine diarrhoea due to parvoviral infection. Hoskins (1997) stated that severe infection with CPV-2 destroyed mitotically active precursors of circulating leukocytes and lymphoid cells; resulting in neutropenia and lymphopenia. In an experimental study conducted on twelve nondescript pups with CPV infection by Joshi *et al.* (2001), the blood picture revealed mild increase in Hb, PCV and TEC. There was leucopenia on day four and six post-infection followed by leucocytosis on day ten post infection in the experimental animals.

2.6.2 Bacterial enteritis

Leucocytosis was observed by Rodrigues *et al.* (1994) in canine diarrhoeal cases due to bacterial infection. In a study conducted by Kumar and Mandial (2002) on six cross bred calves of either sex, between one to ten days of age suffering from *E. coli* infection, haematological findings revealed a significant increase in PCV and non-significant increase in Hb concentration, TEC and Total leucocyte count (TLC). Ramprabhu *et al.* (2002) observed an increased ESR in pups affected with haemorrhagic gastroenteritis indicative of non-specific inflammation. RBC count was very low. The leukogram revealed leucocytosis with relative neutrophilia and lymphopenia.

2.7 SERUM BIOCHEMISTRY

The measurement of serum electrolyte concentrations provided an accurate assessment of the ionic composition of the ECF, but failed to reflect total body electrolyte status (Roussel and Kasari, 1990).

2.7.1 Sodium

The normal serum sodium level in healthy dog was found to be 143 (range 137-149) mEq/L (Benjamin, 1985).

Tennant *et al.* (1972) noticed a reduction in plasma sodium level in majority of diarrhoeic calves, probably due to an increase in excretion of sodium and water through faeces. Alikutty and Rajamani (1973) reported that even though in mild cases of diarrhoea there was no marked change in plasma sodium, prolonged cases resulted in decreased level. Benjamin (1985) opined that intestinal contents contain large amount of sodium bicarbonate which were lost in the faeces due to diarrhoea resulting in depletion of sodium ions. In animals with diarrhoea there was increased loss of sodium, potassium, chloride and bicarbonate with a concurrent decrease in plasma sodium concentration, resulting in hypo-osmotic ECF (Constable *et al.*, 1998). Sayed *et al.* (1998) noted significant decrease in blood serum sodium ions in diarrhoeic camels. Alone *et al.* (2000) observed hyponatraemia in calves with eight per cent dehydration due to diarrhoea. Biochemical studies on blood by Kumar and Mandial (2002) on six cross bred calves of either sex aging between one to ten days suffering from *E. coli* infection, indicated significantly decreased plasma sodium concentration. Ramprabhu *et al.* (2002) observed hyponatraemia in pups affected with haemorrhagic gastroenteritis.

2.7.2 Potassium

Benjamin (1985) recorded normal serum potassium level in dog as 4.4 (range 3.7-5.8) mEq/L.

Alikutty and Rajamani (1973) observed normal or below normal plasma potassium level in mild to moderately severe forms of diarrhoea in calves. Sayed *et al.* (1998) noted significant increase in serum potassium ion levels in diarrhoeic camels. Alone *et al.* (2000) reported hypokalemia at eight per cent dehydration due to diarrhoea in calves. Ramprabhu *et al.* (2002) observed hypokalemia in pups affected with haemorrhagic gastroenteritis. The biochemical studies on blood by Kumar and Mandial (2002) on six cross bred calves of either sex ageing between 1 to 10 days suffering from *E. coli* infection, indicated non-significant increase in potassium concentration.

2.7.3 Chloride

According to Benjamin (1985) the normal serum chloride level in dog is recorded as 90-110 mmol/L.

Significant decrease in blood serum chloride ions were noted in diarrhoeic camels by Sayed *et al.* (1998) whereas Alone *et al.* (2000) stated that in calves with eight per cent dehydration due to diarrhoea, serum electrolyte profile indicated only non-significant changes in the chloride concentration.

2.7.4 Bicarbonate

Kaneko *et al.* (1997) stated that the normal reference range for serum bicarbonate in dog as 18-24 mEq/L.

According to Greco (1998) acid-base status could be reflected by changes in serum bicarbonate concentrations. It was also stated that hyper bicarbonatemia (>30 mEq/L) was associated with metabolic alkalosis and hypobicarbonatemia (<10 mEq/L) was indicative of metabolic acidosis. Alone *et al.* (2000) observed a reduction in plasma bicarbonate level in diarrhoeic calves with eight per cent dehydration.

2.7.5 Acid-Base Status

Roussel and Kasari (1990) mentioned that acidaemia was a consistent complication of diarrhoea and dehydration, generally becoming more severe as dehydration worsened. Typical electrolyte disturbances included a total body deficit of sodium, chloride and potassium, which were lost primarily through the faeces. Acidaemia was caused by loss of bicarbonate ions through faeces; lactic acid accumulation in poorly perfused tissues; reduced acid excretion by poorly perfused kidneys and organic acid production in the colon as a result of fermentation of unabsorbed nutrients. According to Michell (1994) metabolic acidosis was a well recognized potentially life-threatening consequence of diarrhoea. Grove-White (1997) observed metabolic acidosis in diarrhoeic calves. Acidaemia due to metabolic acidosis was the commonest abnormality detected by Koutinas *et al.* (1999) in 17 dogs out of 32 with clinical signs suggestive of parvoviral enteritis.

2.7.6 Serum Proteins

Benjamin (1985) recorded total protein and albumin values in normal dogs as 5.3-7.3 (mean 6.3) g/dl and 3.1-4.0 (mean 3.56) g/dl respectively.

Schalm *et al.* (1975) suggested that the catabolism of body protein in dehydration and water loss from body could lead to an increase in plasma protein per unit volume of blood. Cornelius (1980) stated that there would be an increase in the total serum protein level in dehydration. Jani *et al.* (1992) found that diarrhoeic dogs showed significant increase in total protein values possibly due to dehydration. Hypoproteinaemia and hypoalbuminemia were recorded by Jergens *et al.* (1992) in idiopathic inflammatory bowel diseases associated with gastro-duodenal ulceration-erosion in dogs and cats. According to Radostitis *et al.* (1994) increase in PCV and total serum protein levels indicated the degree of dehydration. Inappetance with resultant reduction of dietary protein and plasma loss from damaged intestinal mucosa appeared to be the main cause of hypoproteinaemia. Study on serum biochemical changes in canine gastroenteritis by Suresh *et al.* (1994) showed that there was a significant decrease in total serum proteins when compared to that of healthy ones. Investigations by Maiti *et al.* (1999) on clinico haematological and therapeutic studies on parasitic gastroenteritis in sheep revealed significantly lower total protein levels in affected sheep. Ramprabhu *et al.* (2002) observed hypoglobulinaemia in pups affected with haemorrhagic gastroenteritis.

2.8 PLASMA VOLUME

A rapid method for determination of plasma volume by using Evan's blue dye (T-1824) was described by Constable (1958). Dilution of Evan's blue

(T-1824) following intravenous injection is the simplest method for estimation of plasma volume (Young, 1964). According to Hunsaker (1965), Evan's blue (T-1824) is the most widely used dye for plasma volume determination and developed a simple procedure for analysis of Evan's blue in plasma based on precipitation of the dye-protein complex with ten per cent trichloroacetic acid followed by extraction with n-butanol. No correlation was observed in many of the cases between plasma volume and PCV in diarrhoeic calves (Alikutty and Rajamani, 1973). Also no consistent reduction in plasma volume was found in mild to moderately dehydrated diarrhoeic calves. Diarrhoea resulted in dehydration and hypovolaemic shock leading to death in calves (Jones and Liska, 1986). Simultaneous determination of plasma volume with Evan's blue and 125 iodine labeled bovine plasma albumin in five to nine days old calves by Wagstaff *et al.* (1992) showed that the use of Evan's blue and isotopic techniques provided equally acceptable measurements of plasma volume in young calves. Dehydration in calves with diarrhoea was accompanied by a large reduction in ECF and smaller increase in intra cellular fluid volumes (Constable *et al.*, 1998).

2.9 FAECAL HAEMAGGLUTINATION

Janthur and Kokkles (1984) used supernatent fluid from chloroform extracted suspensions of faeces as antigen in the HA test using piglet erythrocytes. HA test using faecal samples of dogs with CPV infection was developed by Mohan *et al.* (1992). Hoskins (1997) stated that the period of faecal antigen shed was brief and somewhat cyclical and corresponded to five to seven days of clinical illness. Out of twelve animals experimentally infected with parvovirus, eight showed HA titre ranging between 64 and 1024 on day three, four, five and six, post infection. The HA titre gradually declined and proved negative on day nine and ten post infection. The high faecal HA titre between day four and six post infection reflected the severity of clinical manifestations of disease (Joshi *et al.*, 2001). Praveen *et al.* (2003) suggested that HA test can be used easily and successfully for the detection of parvovirus in faecal samples of dogs excreting the virus.

2.10 THERAPY

Rachman and Pollock (1961) reported the anticoccidial activity of nitrofurazone preparation in canine coccidiosis. Treatment for seven days with this preparation was found to be superior. Coates and Hoopes (1980) emphasized the use of *in vitro* antibiotic sensitivity test as a guideline for proper selection of antibiotic in diarrhoeic conditions. Leib and Zajac (1999) in their study on giardiasis in dogs and cats recommended the use of either metronidazole or fenbendazole.

Shivanandiah (1970) suggested that tender coconut water was a ready and handy boon to combat dehydration of patients suffering from severe diarrhoea and vomiting and a cheap substitute to glucose saline or plasma. An investigation undertaken by Alone *et al.* (2000) on diarrhoeic calves to assess the effectiveness of oral rehydration fluid to correct acid-base imbalance in diarrhoeic dehydration, in comparison to normal intravenous infusion suggested that oral rehydration fluid could be used under field conditions effectively in correcting electrolyte imbalances in view of its easiness in preparation, administration, efficiency and cost.

The main objective of treatment in parvoviral enteritis was to correct the fluid and electrolyte deficits and to re-establish the acid-base equilibrium (Voros *et al.*, 1981). Hoskins (1997) suggested that the primary goals of treatment for CPV-2 enteritis were restoring fluid and electrolyte balance and providing rest to the gastro-intestinal tract followed by antimicrobials, motility modifiers and antiemetics. According to Constable *et al.* (1998), treatment of dehydrated calves required rapid replacement of the existing fluid deficit and provision of additional fluid for ongoing losses. Banja *et al.* (2002) suggested symptomatic treatment with intravenous administration of ringers lactate solution @ 45-90 ml/kg body weight, metaclopramide @ 1 mg/kg body weight, adrenochrome mono semicarbazone @ 0.1-0.2 mg/kg and gentamicin @ 4 mg/kg body weight for dogs suffering from either canine parvo virus or canine corona virus or both.

In a study of commercially available parenteral solutions, by Groutides and Michell (1990) for assessing their effectiveness in correcting the disturbances associated with diarrhoea, they have suggested that solutions for intravenous administration should probably contain about 150 mmol/L sodium ion; five mmol/L potassium ions and about 50 mmol/L of a mixture of bicarbonate and precursors. In a study to determine the therapeutic effects of electrolyte solution with oil emulsions on serious diarrhoea in Holstein calves by Koiwa *et al.* (1990), it was demonstrated that oral fluid therapy with glucose-glycine-electrolyte solution could be effective for calves with serious diarrhoea which had suffered from hypo-lipaemia due to deterioration of nutritional conditions. Roussel and

Kasari (1990) while elaborating on treatment methods of calf diarrhoea agreed the finding that total body sodium and chloride concentrations are decreased in diarrhoeic calves. In dehydrated calf ECF volume might be reduced by 8 to 13 per cent. Therefore even if plasma sodium and chloride concentrations remained within normal limits the total body sodium and chloride were reduced by about the same percentage of decreased ECF volume. Therefore it was essential to replace the ECF volume with solutions that contained these ions in concentrations similar to those in normal plasma so that existing ions were not diluted. Acidaemia due to diarrhoea could be corrected by administering bicarbonate ions or so called bicarbonate precursors that generate bicarbonate ions when metabolized. Bicarbonate precursors included lactate, acetate, gluconate and citrate.

Materials and Methods

3. MATERIALS AND METHODS

The study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy for a period of one and a half years from June 2001 to October 2002. Dogs with the history of diarrhoea presented to Veterinary College Hospitals were selected and utilized for the study. Dogs with clinical signs suggestive of parvoviral enteritis were not considered for the study.

3.1 SELECTION OF ANIMALS

Twenty dogs belonging to different breeds, sex and over four months of age with history of marked diarrhoea were chosen for the study. The selected cases were subjected to detailed clinical examination and various parameters studied.

Normal (control) values of various parameters were worked out by collecting samples from healthy animals brought to Veterinary hospitals for general check up or vaccination.

3.2 OUTLINE OF STUDY

After noting the patient data viz. the age, breed and sex, the present and past history were recorded including nature and frequency of defecation, consistency of faeces or any other allied complications. Details of vaccination and deworming were also collected.

Animals under study were subjected to general clinical examination as proposed by Kelly (1974).

3.3 ISOLATION OF BACTERIAL AGENTS AND IDENTIFICATION

Rectal swab samples were collected and inoculated on Mac Conkey's agar (Hi-media) and Tryptic soya agar (Hi-media) for isolation of bacteria. The inoculated media were incubated at 37°C aerobically for 24 hours. The swab samples were also cultured in selenite F broath (Hi-media) and incubated at 37°C overnight. After the incubation period smears were prepared from the medium and examined for the presence of bacteria. The colonies produced on the Mac Conkey's agar and Tryptic soya agar were stained by gram's method and were identified based on cultural, morphological and biochemical characteristics (Barrow and Feltham, 1993).

3.4 HAEMAGGLUTINATION TEST FOR DETECTION OF CANINE PARVOVIRUS

Rectal swab samples were collected in sterile phosphate buffered saline (pH 7.2) and faecal HA test was carried out as per the procedure of Carmichael *et al.* (1980) to monitor the involvement of Canine parvovirus.

3.5 PARASITOLOGICAL STUDIES

Faecal samples were thoroughly mixed with water using a mortar and pestle, then sieved and the filtrate was collected and centrifuged at 3000 rpm for five min. The sediment was examined under microscope for the presence of ova of parasites.

3.6 SAMPLING AND ANALYSIS

Blood was collected in clean and dry vials, with EDTA at a rate of one mg/ml from the cephalic/saphenous vein for the estimation of haematological parameters as described by Schalm *et al.* (1975).

Blood was collected without anticoagulant in clean, dry, screw capped vials and non-haemolysed serum separated for determination of biochemical parameters. Photometer 5110 (Boehringer Mannheim) under standard conditions of operation was used for biochemical analyses.

Serum chloride was estimated by calorimetric method as cited by Schales and Schales (1941). Determination of serum total protein was done by modified Biurette method (Gornall *et al.*, 1949). Bromocresol green dye binding method as described by Doumas (1971) was used to estimate serum albumin. Serum sodium and potassium levels were estimated by emission flame photometry as described by Oser (1971). Serum bicarbonate was estimated by phosphoenol pyruvate carboxylase method (Tiezt, 1976).

A drop of blood collected from the ear tip was used to prepare blood smear on a clean, grease free and dry glass slide for differential leucocyte count.

3.7 PLASMA VOLUME ESTIMATION

Clean glass vials with EDTA at the rate of one mg/ml were used to collect blood samples from saphenous vein. One per cent solution of Evans blue (T-1824) was injected through the same site with the needle *in situ* as elucidated by Cornelius and Kaneko (1963). To make sure complete delivery of the contents of syringe into the circulation, the syringe was flushed by drawing the piston back and forth.

Calculation of expected plasma volume was done from the postulation that there is 45 ml of plasma per kg body weight. The dosage of dye solution was one ml/L of the expected plasma volume to give a final concentration of ten μg of the dye per ml of plasma. Ten min after the infusion of the dye, five ml of blood was again drawn into a EDTA added vial from the saphenous vein of opposite side.

Blood samples were centrifuged at 3000 rpm for five min to separate plasma. Protein precipitation and extraction method of Hunsaker (1965) was followed to estimate plasma volume.

3.8 REAGENTS USED

- Stock solution of Evans blue (T-1824; 1%)
- Dilute solution of Evans blue (0.001%)
- Trichloro acetic acid (10%)
- n-Butanol

- Hydrochloric acid (concentrated)
- Physiological saline (0.85%)

Plasma volume was then determined from the final concentration of dye per ml of plasma as per the protein precipitation and extraction method of Hunsaker(1965).

Pipetted out one ml of plasma into a test tube. Added one ml of physiological saline and five ml of 10 per cent trichloro acetic acid. Mixed well and allowed to stand for five min. Centrifuged at 3000 rpm for five min. The supernatent fluid was decanted and the test tube kept upside down on a filter paper to drain for few more min. The pad of precipitate in the test tube was broken by using a clean glass rod. Added one ml of concentrated hydrochloric acid and eight ml of n-butanol and mixed well. Kept in a water bath, incubated at 65 to 70°C for two min and then centrifuged at 5000 rpm for ten min.

The butanol dye extract was carefully transferred into the cuvette. The colour was compared with a standard in a spectrophotometer using a red filter of wavelength 620 nm. The calorimetric readings were recorded and the final concentration of the dye in the plasma was calculated using the formulae.

$$\text{Conc. of unknown} = \frac{\text{Reading of unknown x Con. of standard}}{\text{Reading of standard x Vol. of unknown}}$$

Total plasma volume of individual animal was calculated as follows.

$$\text{Plasma volume} = \frac{\text{Amount of dye injected}}{\text{Concentration of dye per ml of plasma}}$$

For calorimetric comparison of test samples, a standard dye solution was made by adding 0.001 per cent Evan's blue to one ml of plasma prepared from the blood collected before the injection of dye. Other procedures performed on this sample were same as explained above.

3.9 STATISTICAL ANALYSIS

For comparison of diarrhoeic animals with normal group, student 't' test was used. Where heterogeneity of variances is observed the test suggested by Snedecor and Cochran (1980) was used.

Results

4. RESULT

Twenty diarrhoeic dogs above four months of age belonging to either sex were selected randomly from the cases presented at Veterinary hospitals, Mannuthy or Kokkalai and were subjected to detailed clinical examination with essential laboratory supports such as faecal sample examination, haematological and microbiological studies.

4.1 ETIOLOGY

Forty five per cent of the total cases studied were due to intestinal parasites of which 35 per cent were due to ancylostomosis and rest ten per cent due to *Isospora.* Diarrhoea due to dietary causes like overfeeding and feeding of unconventional food stuffs constituted 15 per cent of the cases and the rest due to non-specific causes wherein the etiology could not be identified. Bacteria could be isolated from the rectal swabs of all the cases under study. The different bacterial isolates included *Escherichia coli* (50%), *Klebsiella sp.* (20%), *Pseudomonas sp.* (15%) and *Proteus* sp. (15%). *Escherichia coli* was isolated from all the healthy dogs also. Among the animals studied, none were detected to have CPV antigen in the faecal sample, the results of which (HA titre) are given in the Fig.1.

4.2 CLINICAL MANIFESTATIONS

Most of the cases of diarrhoea encountered were mild and the faeces were soft and semisolid in consistency, sometimes mixed with blood and mucus. In seven cases, faeces were tarry or blood tinged. Owners reported that the animals defaecated three to four times per day and in some cases even five to six times. Panting type of respiration was noted in all the dogs studied. Pulse rate of the affected dogs ranged from 88/min to 120/min with a mean value of 102.2/min. The normal dogs showed pulse rate of 90/min to 120/min. Temperature varied from 101.2°F to 105.6°F. Mucous membrane was either pale roseatte or towards the paler side in majority of the cases with a few exceptions in which it was congested. Skin and muzzle regions were relatively dry in all the cases. When assessment of degree of dehydration was done by skin fold test, the skin fold remained as such for three seconds in ten cases. Tenting of the skin could not be noticed in the rest of the cases. Tenderness of the abdominal muscles could be felt on palpation in majority of the cases. In some, intestinal loops were palpable and a few evinced pain on palpation. Either inappetance or anorexia was reported in majority (70%) of cases, while others were taking food and milk normally. Along with diarrhoea vomiting was also reported in ten cases.

4.3 HAEMATOLOGY

Haematological values (mean±SE) of the normal healthy animals and the diarrhoeic dogs are given in the Table 1.

4.3.1 Haemoglobin (Hb)

The mean value of Hb was 13.53 ± 0.35 g/dl in normal healthy animals and 12.29 ± 0.28 g/dl in the diarrhoeic dogs. A significant reduction ($P \leq 0.05$) was noticed in Hb value in the diarrhoeic group when compared to the healthy group. Although statistically significant difference was noticed, the values were within the normal range (Fig.2).

4.3.2 Total Erythrocyte Count (TEC)

The mean value of TEC was 6.763 ± 0.22 x 10^6 cells/µl in normal healthy animals and 6.455 ± 0.22 x 10^6 cells/µl in the diarrhoeic dogs. No statistically significant difference was noticed in TEC between the healthy group and the diarrhoeic group.

4.3.3 Erythrocyte Sedimentation Rate (ESR)

The mean value of ESR was 3.17 ± 0.48 mm in 30 min. in normal healthy animals and 4.75 ± 0.6 mm in 30 min. in the diarrhoeic group. A non-significant increase was observed in the ESR in the diarrhoeic group when compared to the healthy group.

4.3.4 Volume of packed red cells

The mean value of volume of packed red cells was 38.67 ± 1.54 per cent in normal healthy animals and 41.55 ± 1.09 per cent in the diarrhoeic animals. A non-significant increase was observed in the volume of packed red cells of diarrhoeic dogs when compared to healthy group.

4.3.5 Total Leucocyte Count (TLC)

The mean value of TLC was 11000 ± 2.45 cells/µl in normal healthy animals and 11320 ± 426.14 cells/µl in the diarrhoeic dogs. No statistically significant difference was noticed in TLC between the healthy group and the diarrhoeic group.

4.3.6 Differential Leucocyte Count (DLC)

Differential leucocyte count revealed neutrophils (70.50±1.56%), lymphocytes (26.33±0.85%), Eosinophils (2.33±0.85%) and monocytes (0.83±0.4%) in normal healthy dogs. Corresponding values obtained from the diarrhoeic group were neutrophils (73.95±1.78%), lymphocytes (23.30±1.76%), Eosinophils (2.10±0.39%) and monocytes (0.70±0.21%). Basophils were not detected in any of the samples. No statistically significant difference was noticed in differential leucocyte counts between the healthy group and the diarrhoeic group.

4.4 SERUM BIOCHEMISTRY

The serum biochemical values (mean±SE) of the normal healthy animals and the diarrhoeic dogs are given in the Table 2.

4.4.1 Sodium

The mean value of serum sodium level recorded was 152.653 ± 3.199 mEq/L in normal healthy animals and 137.047 ± 4.07 mEq/L in the diarrhoeic dogs. No statistically significant difference was noticed in serum sodium level between the healthy group and the diarrhoeic group.

4.4.2 Potassium

The mean value of serum potassium level recorded was 4.427 ± 0.198 mEq/L in normal healthy animals and 5.059 ± 0.194 mEq/L in the diarrhoeic group. No statistically significant difference was noticed in serum potassium level between the healthy group and the diarrhoeic group.

4.4.3 Chloride

The mean value of serum chloride level was 102.33 ± 1.26 mmol/L in normal healthy animals and 86.90 ± 2.98 mmol/L in the diarrhoeic group. A statistically significant reduction ($P \leq 0.01$) was noticed in serum chloride levels in the diarrhoeic group when compared to the healthy group (Fig.3).

4.4.4 Bicarbonate

The mean value of serum bicarbonate was 77.67 ± 6.37 mEq/L in nomral healthy animals and 58.10 ± 5.79 mEq/L in the diarrhoeic group. No statistically significant difference was noticed in the serum bicarbonate levels between the healthy group and the diarrhoeic group.

4.4.5 Total protein

The mean value of serum total protein was 8.6 ± 0.32 g/dl in normal healthy animals and 7.46 ± 0.18 g/dl in the diarrhoeic group. A statistically significant reduction was noticed in serum total protein level of diarrhoeic dogs when compared to the healthy group (Fig.4).

4.4.6 Albumin

The mean serum albumin level was 3.13 ± 0.28 g/dl in normal healthy animals and 2.75 ± 0.11 g/dl in the diarrhoeic group. No statistically significant difference was noticed in serum albumin level between the healthy group and the diarrhoeic group (Fig.4).

4.4.7 Albumin Globulin Ratio (A/G Ratio)

The mean value of albumin globulin ratio was 0.632 ± 0.02 in normal healthy dogs and 0.592 ± 0.05 in the diarrhoeic group. No statistically significant difference was noticed in the albumin globulin ratio between the healthy group and the diarrhoeic group.

4.8 PLASMA VOLUME

The mean value of plasma volume was found to be 59.93 ± 3.04 ml/kg body weight in normal healthy animals and 52.86 ± 1.14 ml/kg body weight in the diarrhoeic dogs. A statistically significant fall ($P \leq 0.05$) in the mean value of plasma volume was noticed in diarrhoeic group when compared with normal healthy group (Fig.5).

Table 1. Haematology of dogs - Healthy and Clinical cases (mean±SE)

Parameters	Healthy group	Diarrhoeic group
Plasma volume (ml/kg)	59.93±3.04[a]	52.86±1.14[b]
Total Erythrocyte count (million cells/μl)	6.763±0.22[a]	6.455±0.22[a]
Total Leucocyte count (cells/μl)	11,000±2.45[a]	11,320±426.14[a]
Neutrophils (%)	70.50±1.56[a]	73.95±1.78[a]
Lymphocyte (%)	26.33±0.85[a]	23.30±1.76[a]
Eosinophils (%)	2.33±0.85[a]	2.10±0.39[a]
Monocyte (%)	0.83±0.4[a]	0.70±0.21[a]
Haemoglobin (g/dl)	13.53±0.35[a]	12.29±0.28[b]
Erythrocyte Sedimentation rate (mm/30 min)	3.17±0.48[a]	4.75±0.60[a]
Volume of packed red cells (%)	38.67±1.54	41.55±1.09

Significant ($P<0.05$) when compared with healthy

Means within same row with different superscript differ significantly.

Table 2. Serum biochemical values of dogs - Healthy and Clinical cases (mean±SE)

Parameters	Healthy group	Diarrhoeic group
Sodium (m Eq/L)	152.653±3.199	137.047±4.07
Potassium (mEq/L)	4.427±0.198	5.059±0.194
Chloride (mmol/L)	102.33±1.26	**86.90±2.98
Bicarbonate (mEq/L)	77.67±6.37	58.10±5.79
Total protein (g/dl)	8.6±0.32	**7.46±0.18
Albumin (g/dl)	3.13±0.28	2.75±0.11
Albumin - globulin ratio	0.632±0.02	0.592±0.05

** Highly significant (P<0.01) when compared with healthy

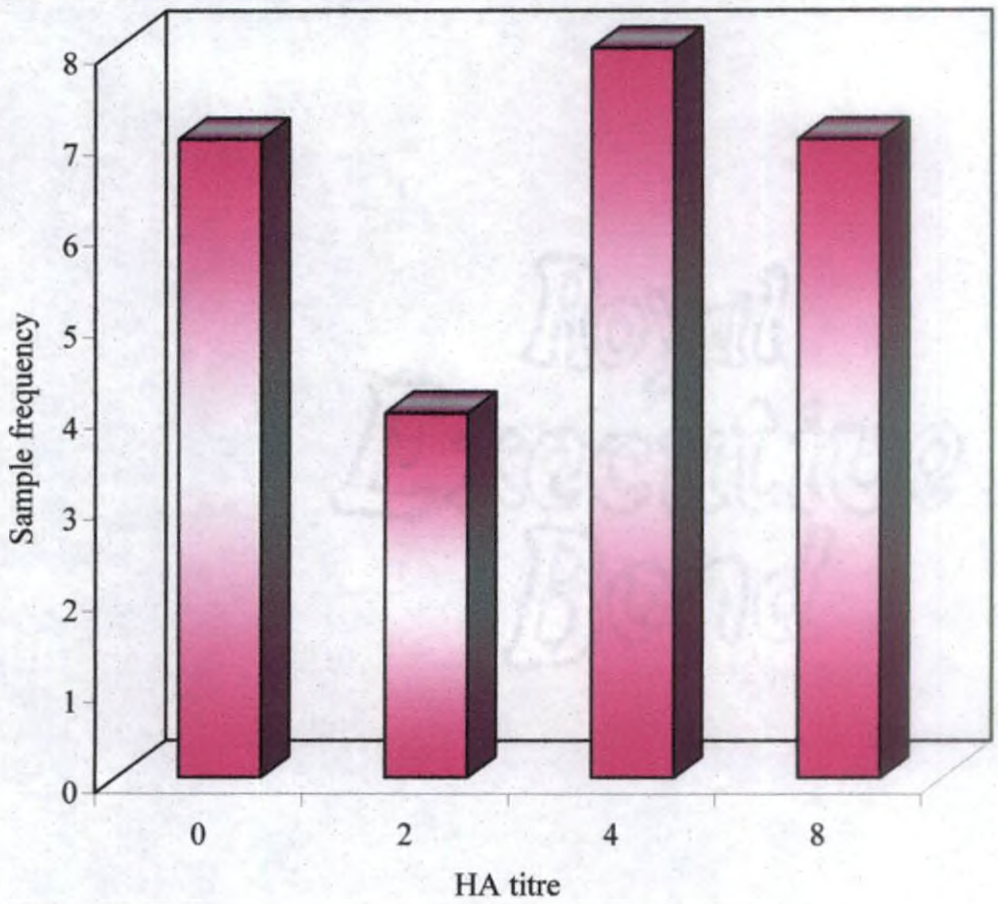

Fig. 1. Frequency of Canine Parvovirus HA titre in faecal samples

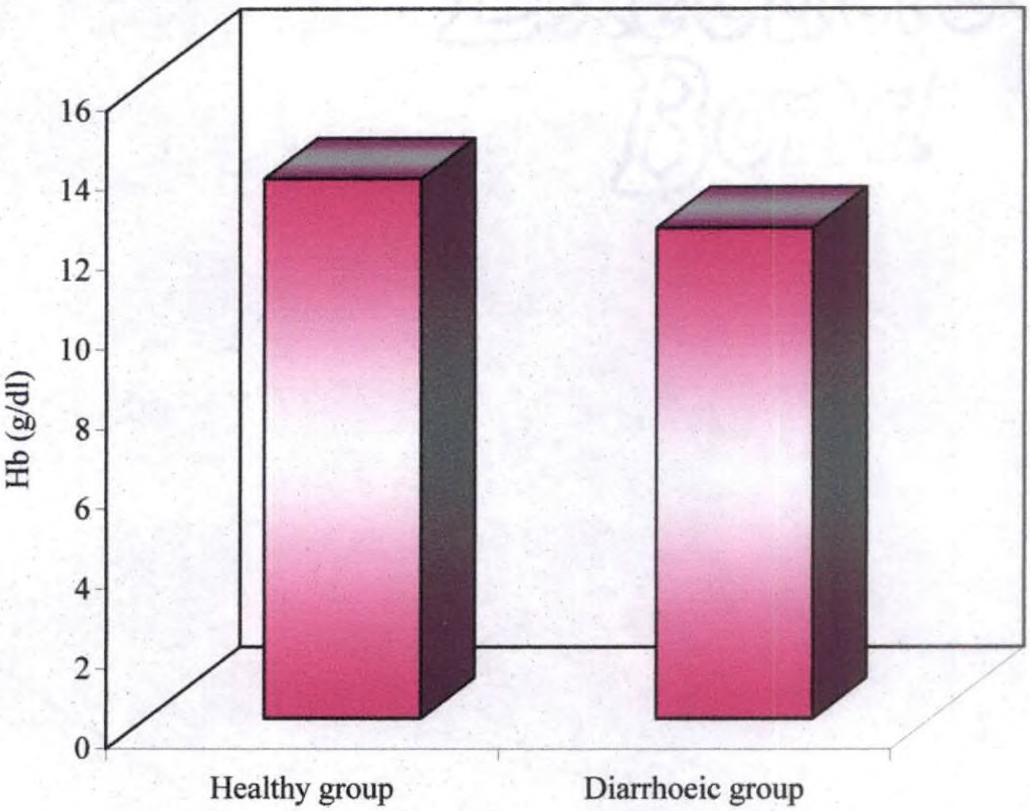

Fig. 2. Haemoglobin in healthy and diarrhoeic groups

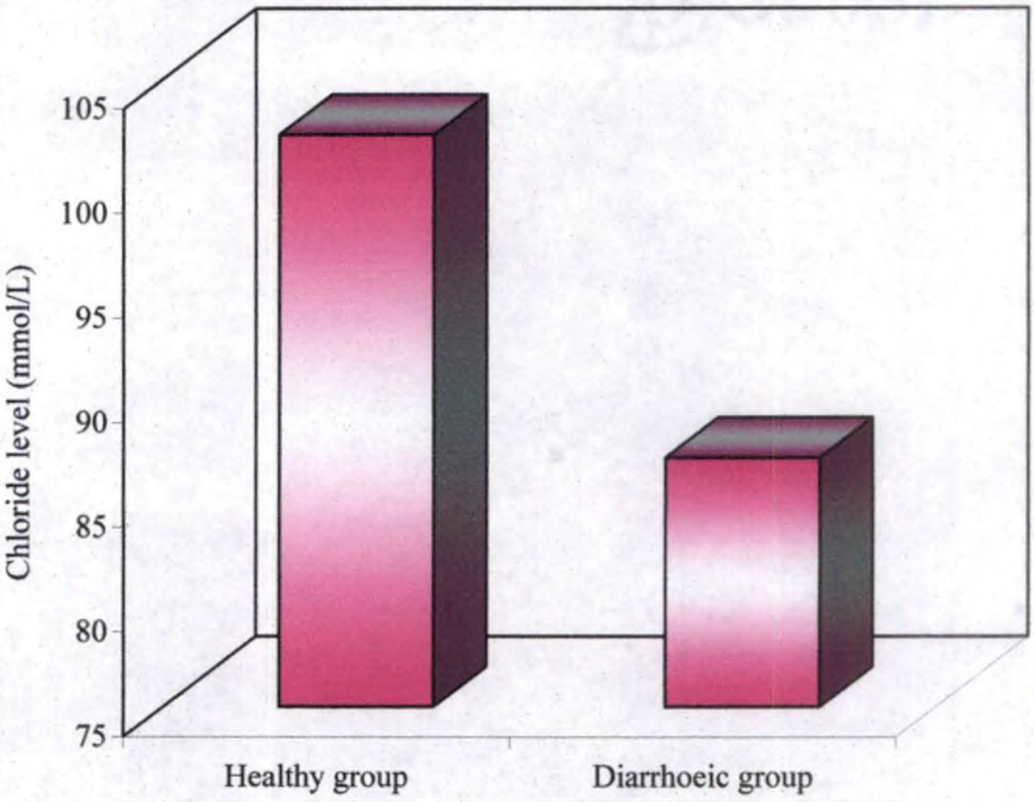

Fig. 3. Chloride level in healthy and diarrhoeic groups

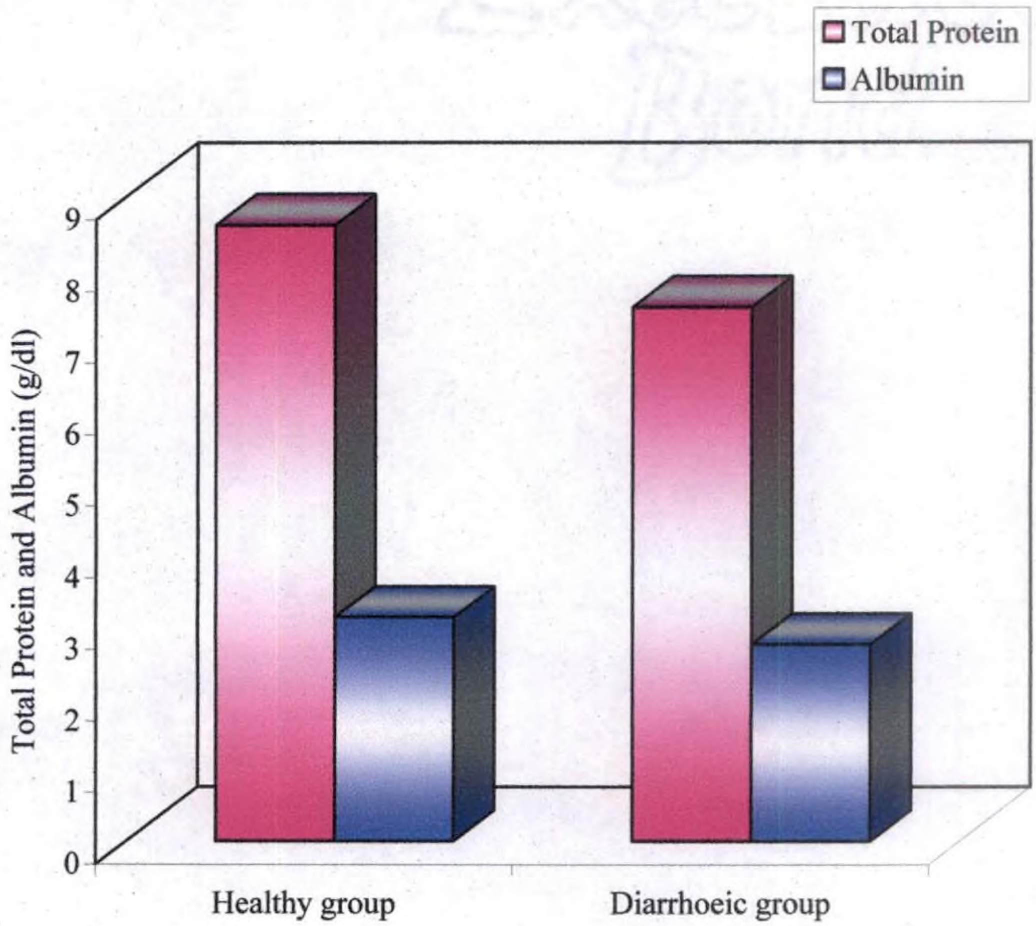

Fig. 4. Total Protein and Albumin in healthy and diarrhoeic groups

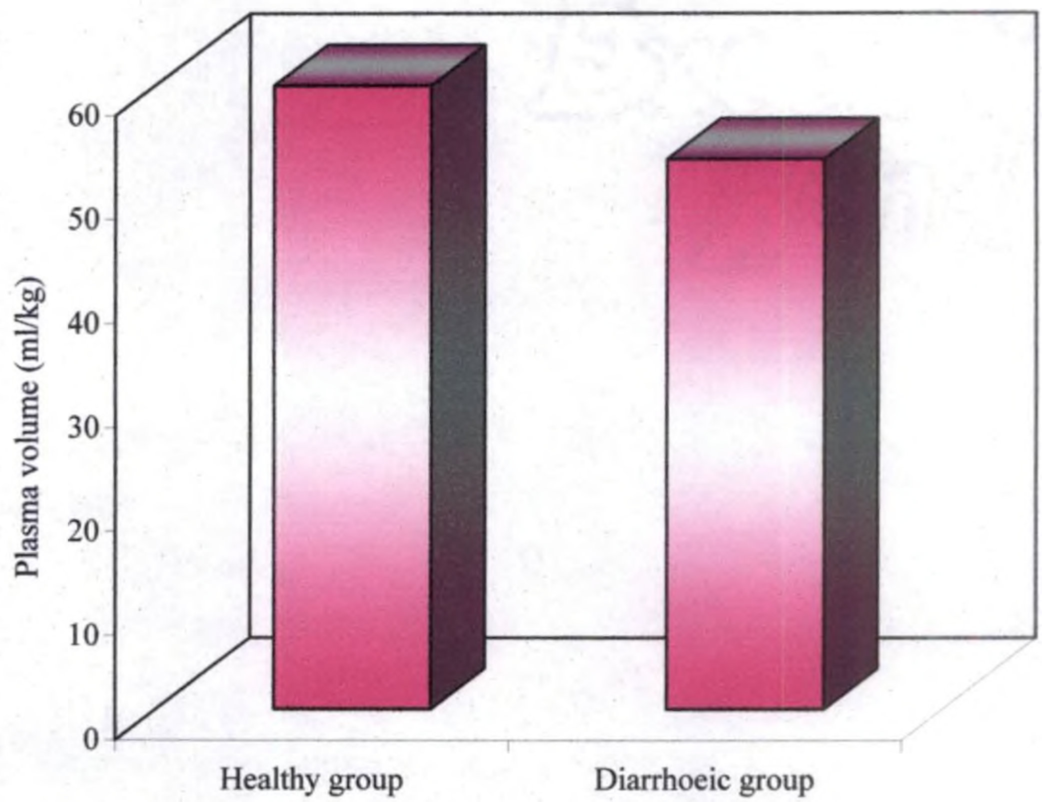

Fig. 5. Plasma volume in healthy and diarrhoeic groups

Discussion

5. DISCUSSION

Diarrhoea is one of the most common clinical conditions encountered among dogs in day to day practice and is a prominent symptom of digestive disturbances. Etiology and clinico-pathological aspects of diarrhoea have been a subject of worldwide research. Available literature have revealed that diarrhoea was a multi-factorial syndrome affecting a wide variety of animal species (Deepti *et al.*, 1999). Parasitic gastroenteritis is a common and important problem in dogs. Apart from infectious causes, nutritional and management practices also play a vital role in the occurrence of diarrhoea. Diarrhoea if not properly attended may progress to life threatening fluid and electrolyte disturbances leading to severe dehydration, hypovolaemic shock and death. The degree of dehydration, haematological and serum biochemical changes in diarrhoea were assessed in this study.

5.1 ETIOLOGY

Intestinal parasites were responsible for diarrhoea in 45 per cent of the cases studied. According to Rodrigues *et al.* (1994) diarrhoea due to parasites alone was encountered in 25 per cent of the cases studied. Similar observations were made by Niphadkar *et al.* (1979). Ancylostomes dominated the parasitic diarrhoea cases in their study. These findings were in agreement with the observations of Asati *et al.* (1999) wherein highest incidence of *Ancylostomum caninum* was demonstrated among helminths in dogs. Intestinal parasites usually create real problems, as some of them are bloodsuckers, others produce nodules in gastrointestinal tract, still others cause inflammatory reactions in gastrointestinal tract resulting in impaired digestion and assimilation. This may cause profuse watery or blood tinged diarrhoea, anaemia and emaciation.

Escherichia coli dominated among bacterial isolates from faecal samples of diarrhoeic cases followed by *Klebsiella* sp., *Pseudomonas* sp. and *Proteus* sp. *Escherichia coli* could also be isolated from the healthy group. Coliforms isolated from the clinical cases need not necessarily be the pathogenic ones as demonstrated by Spielman and Garvey (1993) who isolated *E. coli* from all the control dogs. Bacterial infections were responsible for diarrhoea in 25 per cent of the cases (Rodrigues *et al.*, 1994) and *E. coli* was found to be the major cause. Renault *et al.* (1977) found *E. coli* to play a major role in the development of diarrhoea in dogs.

5.2 CLINICAL MANIFESTATIONS

In the present study, cases of diarrhoea in which the faeces mixed with blood and mucus were mostly affected with ancylostomes. This could be corroborated with the findings of Udonsi and Agunama (1991) where in occult blood was reported in the faeces. A few dogs affected with bloody diarrhoea showed moderate rise in body temperature and vomiting. Ramprabhu *et al.* (2002) reported that haemorrhagic gastroenteritis in young pups manifested high rise of body temperature, inappetance, froathy yellow vomitus, retching and restlessness.

5.3 HAEMATOLOGY

5.3.1 Haemoglobin

The diarrhoeic group showed a significant reduction in the mean value of haemoglobin compared to the healthy group. These results were corroborated by Morgan (1967) who established reduced haemoglobin level in intestinal parasitism in dogs and was attributed to secretion of haemolysins, production of myelotoxins which injure the organs of blood formation, blood sucking activities of the parasites and depletion of haemopoietic hormones. Maiti *et al.* (1999) also reported lower haemoglobin level in an outbreak of parasitic gastroenteritis in sheep.

Deshpande *et al.* (1993) found a significant increase in haemoglobin level in diarrhoeic calves. According to Benjamin (1985) the increase in haemoglobin level in diarrhoea might be due to haemoconcentration as a result of decrease in the plasma volume without appreciable change in the total mass of erythrocyte brought about by dehydration. The cause of reduced haemoglobin level could be attributed to ancylostomosis. As the degree of dehydration was mild, it did not markedly affect the heamoglobin status.

5.3.2 Total Erythrocyte Count (TEC)

A moderate reduction in TEC was observed in diarrhoeic dogs. This result was on the similar lines of Ramprabhu *et al.* (2002) who also have observed a reduction in erythrocyte count in haemorrhagic gastroenteritis in pups. According to Morgan (1967) hook worm infestation caused decrease in red blood cells due to the secretion or elaboration of haemolysins, the production of myelotoxins which injure the organs of blood formation, the substances secreted by adult worms, the substances associated with the activity or death of larvae, blood sucking activities of the parasites, depletion of haemopoietic hormones or the combination of all the above. Similar results were obtained by Maiti *et al.* (1999) in an outbreak of parasitic gastroenteritis in sheep. But a significant increase in TEC in diarrhoeic dogs was reported by Cornelius (1972), Jones and Liska (1986), Jani *et al.* (1992), Zafar *et al.* (1999), possibly due to dehydration. The lack of marked variation in the erythrocyte count in the present study could be due to incorporation of both haemorrhagic and nonhaemorrhagic diarrhoeic cases.

5.3.3 Erythrocyte Sedimentation Rate (ESR)

A non-significant increase in ESR was evident in diarrhoeic dogs in comparison with the normal healthy group. Anaemia and concurrent hypoalbuminaemia resulted in elevation of ESR (Morgan, 1967). Ramprabhu *et al.* (2002) also observed increased ESR in pups affected with haemorrhagic gastroenteritis, and the increase was attributed to nonspecific inflammation.

5.3.4 Volume of Packed Red Cells

Non-significant elevation of volume of packed red cells observed in the present study agreed with the findings obtained in dogs suffering from diarrhoea (Cornelius, 1972; Jones and Liska, 1986; Jani *et al.*, 1992; Zafer *et al.*, 1999).. According to Michell (1974) loss of ECF in diarrhoeic calves led to haemoconcentration with an increase in PCV. Reduced sodium ion concentration due to diarrhoea causes more water to enter cells reinforcing the rise in PCV. Similar findings were obtained by Alikutty and Rajamani (1973) and Deshpande *et al.* (1993) in calves. The absence of marked elevation in PCV showed that the fluid loss caused by mild cases of diarrhoea is often compensated and do not reflect in blood.

5.3.5 Total Leucocyte Count (TLC)

Statistically the non-significant increase in TLC observed in the present study was in agreement with the findings of Rodrigues *et al.* (1994) who could demonstrate leucocytosis in canine diarrhoeal cases associated with parasitic infection. The leukogram in haemorrhagic gastroenteritis in dogs revealed leucocytosis (Macartney *et al.*, 1984; Ramprabhu *et al.*, 2002). According to Benjamin (1985) greater the severity of infection, greater was the degree of leucocytosis. Kumar and Mandial (2002) also could record a non-significant increase in TLC in a case of colibacillosis of calves.

Benjamin (1985) suggested that leucopaenia could occur in early stages of an infectious disease or localized severe infection where there is depletion in peripheral blood of its leucocytes with concentration in the area of inflammation coupled with the lysis of lymphocytes in the circulation until the bone marrow catches up with the production.

5.3.6 Differential leucocyte count

Differential leucocyte counts varied for individual dogs but when all the animals in the group were assessed a non-significant increase was observed in neutrophil count and a non-significant decrease in lymphocyte count in diarrhoeic dogs. Eosinophil and monocyte counts registered statistically non-significant changes. According to Chen and Shien (1981) percentages of neutrophils and lymphocytes were positively related to the degree of dehydration in dogs. Hoskins (1997) stated that severe infection with CPV-2 destroys mitotically active precursors of circulating leucocytes and lymphoid cells resulting in neutropaenia and lymphopaenia. Ramprabhu *et al.* (2002) observed leucocytosis with relative neutrophilia and lymphopaenia in haemorrhagic gastroenteritis in pups.

5.4 SERUM BIOCHEMISTRY

5.4.1 Sodium

A non-significant reduction in serum sodium was recorded in diarrhoeic group. Intestinal contents contain large amount of sodium bicarbonate which are lost in the faeces due to diarrhoea resulting in the depletion of sodium ions (Benjamin, 1985). Ramprabhu *et al.* (2002) observed hyponatraemia in pups affected with haemorrhagic gastroenteritis coinciding with the reports of Heald *et al.* (1986) and Mason *et al.* (1987).

Tennant *et al.* (1972) also observed a reduction in plasma sodium level in diarrhoeic calves probably due to an increased excretion of sodium and water through faeces. According to Constable *et al.* (1998) increased intestinal loss of sodium in diarrhoeic cases resulted in concurrent decrease in plasma sodium concentration. Significant fall in serum sodium concentration was observed by Sayed *et al.* (1998) in diarrhoeic camels. Alone *et al.* (2000) and Kumar and Mandial (2002) also could note hyponatraemia in diarrhoeic calves. The absence of marked hyponatraemia in the present study suggested that the cases were not severe and life threatening.

5.4.2 Potassium

The increase in serum potassium concentration in diarrhoeic group was non-significant and concurred with the findings of Kumar and Mandial (2002).

Hyperkalaemia recorded in calves simulated the earlier observations made by Deshpande *et al.* (1993) and Grove-white and White (1999). This was attributed to metabolic acidosis, which might have induced the translocation of potassium ions from intracellular to extracellular compartment thus raising the plasma potassium concentration. Whereas Ramprabhu *et al.* (2002) reported hypokalaemia in haemorrhagic gastroenteritis in pups and suggested that it was due to loss of potassium ions through diarrhoeic faeces as the intestinal contents were rich in potassium. In the present study the conditions were not serious enough to induce marked changes in the serum potassium level and the little effect could have been nullified by different disease conditions.

5.4.3 Chloride

Significantly lower values of serum chloride observed in diarrhoeic dogs agreed with the findings of Sayed *et al.* (1998) who could note a significant decrease in serum chloride ions in diarrhoeic camels. Loss of acid radicals may happen due to vomiting. Statistically significant lower values of chloride could be due to the fact that some of the diseased animals had shown vomiting also.

5.4.4 Bicarbonate

Reduction in serum bicarbonate level observed in diarrhoeic group was non-significant. Alone *et al.* (2000) noted a reduction in plasma bicarbonate level in diarrhoeic calves. Hypobicarbonataemia is indicative of metabolic acidosis (Greco, 1998). According to constable *et al.* (1998) animals with diarrhoea have increased intestinal loss of bicarbonate. The findings of the present study indicated that only severe diarrhoea would induce a marked pH change in blood.

5.4.5 Total protein

Significantly reduced total protein values were obtained in diseased dogs compared to the normal healthy group. This confirmed that there was no serious dehydration in the dogs studied. The study of Suresh *et al.* (1994) in gastroenteritis in dogs and that of Maiti *et al.* (1999) in sheep were on the similar lines and this was attributed to the reduced nutritional status of animals.

Schalm *et al.* (1975), Cornelius (1980) and Jani *et al.* (1992) found that diarrhoeic dogs showed significant increase in total protein values possibly due to dehydration. The catabolism of body protein in dehydration and water loss from body could lead to an increase in plasma protein per unit volume of blood.

5.4.6 Albumin

Non-significantly reduced albumin level in diarrhoeic dog fairly agreed with the findings of Jergens *et al.* (1992) who recorded hypoalbuminaemia in inflammatory bowel diseases in dogs. According to Benjamin (1985) decreased albumin level (hypoalbuminaemia) could also be due to impaired absorption resulting from diarrhoea.

5.4.7 Albumin Globulin Ratio (A/G Ratio)

No significant change could be observed in albumin globulin ratio between the affected and normal healthy group. The severity of dehydration was not high to effect a change in albumin globulin ratio. Benjamin (1985) reported albumin globulin ratio of 0.59 to 1.11 as normal values in dogs.

5.5 PLASMA VOLUME

The study showed a significant fall in the mean value of plasma volume which corroborates with the findings of Fisher (1965) who reported a significant reduction in the plasma volume of diarrhoeic calves. No consistent reduction in plasma volume was found in mild to moderately dehydrated diarrhoeic calves by Alikutty and Rajamani (1973). Constable *et al.* (1998) demonstrated that dehydration in calves with diarrhoea is accompanied by drastic reduction in ECF and smaller increase in intracellular fluid volumes. Extracellular hypo-osmolality causes free water to move from the extracellular to intracellular space, thereby increasing intra cellular fluid volumes. Because water loss is from the extracellular space, estimation of ECF is a valuable and commonly used method to decide about the fluid therapy in dehydrated animals.

5.6 HAEMAGGLUTINATION TEST

The HA activity of canine parvovirus was utilized diagnostically in estimating amounts of viral haemagglutinins in faecal samples (Carmichael *et al.*, 1980). HA can be considered as a rapid and convenient test for the detection of CPV in faecal samples at the field level as suggested by Sherikar *et al.* (1989). Treatment of faecal samples with chloroform as suggested by Carmichael *et al.* (1980) to remove nonspecific haemaglutinins was adopted in this study. In the present study HA test on faecal materials of 20 dogs with diarrhoea and six normal healthy dogs revealed that none of them were shedding CPV in the faeces. HA titre of 64 and above was considered positive as suggested by Carmichael *et al.* (1980). The CPV titre in this study ranged from zero to eight. While, Carmichael *et al.* (1980) observed HA titre ranging between 320 to 10,240 between four and seven days after experimental infection. Janthur and Kokkles (1984) found HA titre ranging from 40 to 40,960. Rai *et al.* (1994) recorded HA activity of 320 to 20,480 from faecal samples collected three to six days after the onset of clinical illness. The absence of positive cases in the present study may be due to the fact that all the animals subjected to the present study were protected against parvoviral infection.

Based on the findings supportive therapy with balanced electrolyte solutions was recommended for correction of the mild electrolyte and fluid imbalances that occurred in diarrhoeic cases studied. Depending upon the specific etiology, treatment schedules such as deworming, antimicrobial therapy and managemental changes were also adopted.

Summary

6. SUMMARY

Diarrhoea is the increased frequency of defecation accompanied by an increased concentration of water and decrease in dry matter content. Faeces vary in consistancy from being soft to liquid. Loss of water and electrolyte through the faeces causes fluid and electrolyte imbalances hence a detailed investigation was undertaken to assess the fluid and electrolyte imbalances resulting from diarrhoea and to suggest a suitable therapeutic regimen.

The present study included twenty dogs of different breeds of either sex over four months of age with history of marked diarrhoea as clinical cases and six apparently healthy animals as the normal control group. Signalment and detailed clinical examination was carried out in all the animals. Blood and faecal samples were collected on day of admission to the hospital and utilized for the haematological and biochemical analysis including plasma volume estimation, parasitological and microbiological studies including faecal culture. Haematological studies include estimation of Hb, TEC, ESR, volume of packed red cells, TLC and DLC.

Most of the cases of diarrhoea encountered were mild some times mixed with blood and mucus with four to five defecation per day. Temperature varied from 101.2°F to 105.6°F. Pulse and respiration registered no appreciable changes. Either inappetance or anorexia was reported in majority of cases.

Though diarrhoea is of multi-factorial etiology the possible predominant etiological agent for diarrhoea was identified as intestinal parasites (45%). Among the intestinal parasites the predominant one was ancylostomes. Though bacteria could be isolated from all the cases, it could not give a true reflection of the actual disease. The bacteria isolated cannot be considered as a primary etiological factor in diarrhoea, but it played a major role in aggravating the clinical cases. Viral etiology cannot be ruled out from the list of major etiological factors causing diarrhoea. Canine parvoviral titre was assessed by HA test and none was found to be positive. A low titre was obtained from HA test and hence concludes that the animals under study could have been properly immunized against the disease.

The mean values of haemoglobin level in diarrhoeic group and normal group were 12.29±0.25 g/dl and 13.53±0.35 g/dl respectively. The significant reduction recorded in haemoglobin level in diarrhoeic group ($P \leq 0.05$) could be attributed to helminthes parasitism. Parasitological evaluation indicating worm load and a moderate reduction in erythrocyte count were complementary findings. Diarrhoea and haemoconcentration are attributable to elevated PCV. A non-significant increase in TLC also was in expected lines indicating infectious etiology.

Loss of sodium ions through faeces resulted in reduced serum sodium level in the diarrhoeic dogs (137.047±4.07 mEq/L) when compared to the normal healthy group (152.653±3.199 mEq/L). Hyperkalaemia and hypobicarbonaetaemia observed in the diarrhoeic groups could indicate mild metabolic acidosis. Statistically significant lower values of chloride in diarrhoeic group ($P \leq 0.001$) may be due to the fact that some of the selected animals have shown vomiting also. Lowered total protein values obtained in diarrhoeic group (7.46±0.18 g/dl) when compared to normal healthy group (8.6±0.32 g/dl) were attributable to the low level of nutrition and partial or complete anorexia in clinical cases. A significant fall in plasma volume in the diarrhoeic dogs (52.86±1.14 ml/kg) when compared to normal (59.93±3.04 ml/kg) was due to mild dehydration associated with fluid loss in diarrhoea.

Studying the changes in serum biochemical and haematological parameters helped in formulating the right supportive therapy. The choice of electrolyte solution should be in such a way that it should contain these electrolytes in the right proportion. Choice of a suitable deworming agent could be made based on the parasitological findings. Haematological findings could help in choosing right supportive therapy in diarrhoea due to different etiologies.

References

REFERENCES

Alikutty, K.M. and Rajamani, S. 1973. Studies on calf diarrhoea-II plasma sodium and potassium levels and plasma volume in diarrhoeic calves. *Indian Vet. J.* 50(12): 1182-1190

Alone, S.A., Kolte, A.Y., Sadekar, R.D., Mode, S.G. and Joshi, M.V. 2000. Comparative efficacy of different rehydration therapies in restoring electrolyte imbalance in diarrhoeic dehydration. *Indian Vet. J.* 77(2): 124-126

Appel, M.J.G., Scott, F.W. and Carmichael, L.E. 1979. Isolation and immunization studies of canine parvo-like virus from dogs with haemorrhagiec enteritis. *Vet. Rec.* 105(8): 156-59

Asati, A.K., Shukla, P.C. and Sisodia, R.S. 1999. Faecal prevalence of eggs of gastro-intestinal helminthes. *Vet. Med. J.* **23**: 237-238

Banja, B.K., Sahoo, N., Das, P.K. and Ray, S.K. 2002. Clinico-therapeutic aspects of gastro-enteritis in dogs. *Indian Vet. J.* 79(9): 837-840

Barrow, G.I. and Feltham, R.K.A. 1993. *Cowan and Steel's Manual for the Identification of Medical Bacteria.* Second edition. Cambridge University, Press, New York. p.331

Benjamin, M.M. 1985. *Outline of Veterinary Clinical Pathology.* Third edition. Indian Reprint 2001. Kalyani Publishers, New Delhi. p.351

Bloom, F. 1959). Functional diarrhea in dog. *Vet. Med.* **54**(4):200-203

Brar, R.S., Sandhu, H.S. and Singh, A. 2000. Normal blood values. *Veterinary Clinical Diagnosis by Laboratory Methods.* Kalyani Publishers, New Delhi. p.334

*Bund, K. and Laohasurayothin, P. 1982. Diagnosis of parvovirus infection in the dog. *Kleintier Prax* **27**: 411-413

Carman, P.S. and Povey, R.C. 1985. Pathogenesis of canine parvovirus-2 in dogs: haematology, serology and virus recovery. *Res. Vet. Sci.* **38**(2): 134-140

Carmichael, L.E., Joubert, J.C. and Pollock, R.V.H. 1980. Haemagglutination by canine parvovirus: serological studies and diagnostic applications. *Am. J. Vet. Res.* **41**(1): 784-91

*Chen, C.L. and Shien, Y.S. 1981. Studies on dehydration in dogs-1. Evaluation of the dehydration status of dogs by some haematological findings. *Chinese Society Vet. Sci.* 7(2):121-129.

Coates, S.R. and Hoopes, K.H. 1980. Sensitivities of *Escherichia coli* isolated from bovine and porcine enteric infections to antimicrobial antibiotics. *Am. J. Vet. Res.* 41(11):1882-1883.

Constable, B.J. 1958. *Clin. Sci.* Cited in the article Determination of Evan's blue in plasma and plasma dextran mixture by protein precipitation and extraction. *Proc. Soc. Exp. Biol. Med.* 1964, **115**: 564-567

Constable, P.D., Walker, P.G., Morin, D.E. and Foreman, J.H. 1998. Clinical and laboratory assessment of hydration status of neonatal calves with diarrhoea. *J. Am. Vet. Med. Assoc.* **212**(7): 991-995

Cornelius, C.E. and Kaneko, J.J. 1963. *Clinical Biochemistry of Domestic Animals.* Academic press, New York. p.508.

Cornelius, L.M. 1972. A study of water and electrolyte balance in dogs with experimental diarrhoea. *Diss. Abst. Int.* **32**: 5526

Cornelius, L.M. 1980. Fluid therapy in small animal practice. *J. Am. Vet. Med. Assoc.* 176(2):110-114.

Deepa, P.M. 1999. Seroprevalence and diagnosis of canine parvoviral infection. M.V.Sc. thesis, Kerala Agricultural University, Mannuthy, p.159

Deepti, C., Katoch, R.C., Jasial, S. and Mahajan, A. 1999. Enteropathogenecity of *E. coli* from diarrhoeic ruminants in Himachal Pradesh. *Indian J. Anim. Sci.* 69(7): 469-471

Deshpande, A.P., Anantwar, L.G., Digraskar, S.U. and Deshpande, A.R. 1993. Clinico-pathological and biochemical alterations in calf scour. *Indian Vet. J.* 70(7): 679-680

*Doumas, B.T. 1971. *Clin. Chem. Acta.* **31**: 87. Cited in the operation instructions for estimation of albumin - Qualigens Fine Chemicals, Mumbai.

Dubey, S. and Sharda, R. 2001. Prevalence, serotypes and pathogenicity of *Escherichia coli* associated with diarrhoea in goats. *Indian J. Anim. Sci.* 71(2): 92-94

Duda, A., Stenzel, D.J. and Boreham, P.F.L. 1998. Detection of *Blastocystis* sp. in domestic dogs and cats. *Vet. Parasitol.* **76**: 9-17

Fisher, E.W. 1965. Death in neonatal calf diarrhoea. *Br. Vet. J.* **121**(3): 132-138

Gornall, A.G., Bardawill, C.J. and David, M.M. 1949. Determination of serum protein by means of biuret reaction. *J. Biol. Chem.* **177**: 1751-1752

*Greco, D.S. 1998. The distribution of body water and general approach to the patient. *Vet. Clin. North. Am. Small Anim. Pract.* **28**: 473-482

Groutides, C.P. and Michell, A.R. 1990. Intravenous solutions for fluid therapy in calf diarrhoea. *Res. Vet. Sci.* **49**(3): 292-297

*Grove-White, D.H. 1997. Pathophysiology and treatment of metabolic acidosis in the diarrhoeic calf. *Bovine practitioner*. **31**: 56-60

Grove-white, D.H. and White, D.G. 1999. Abdominal distension in collapsed diarrhoeic calves: biochemical findings and treatment. *Vet. Rec.* **144**(23):639-642.

Hammond, M.M. and Timoney, P.J. 1983. An electron microscopic study of viruses associated with canine gastroenteritis. *Cornell Vet.* **73**: 82-97

Heald, R.D., Jones, B.D. and Schmidt, D.A. 1986. Blood gas and electrolyte concentrations in canine parvoviral entritis. *J. Am. Anim. Hosp. Assoc.* **22**(6): 745-748

*Heemyung, P., Taeho, O.H., Keun, Y. and Hongryul, H. 1999. Studies on the etiological agents in pups with the concurrent intestinal and respiratory tract disorders. *Korean J. Vet. Res.* **39**(4): 825-831

Hoskins, J.D. 1997. Update on canine parvoviral enteritis. *Vet. Med.* **92**(8): 697-709

Hunsaker, W.G. 1965. Determination of Evan's blue in avian plasma by protein precipitation and extraction. *Proc. Soc. Exptl. Biol. Med.* **120:** 747-749

*Itoh, N. and Muraoka, N. 2001. *Giardiasis* in dogs. *J. Japan Vet. Med. Assoc.* **54**(9): 713-716

*Itoh, N. and Muraoka, N. 2002. Detection of Isospora oocysts in house hold dogs. *J. Japan Vet. Med. Assoc.* **55**(1): 21-25

James, G.F., Winkler, W.G., Peacock, F.L. and Jackson, V. 1963. Diagnosis and management of salmonellosis in grey hounds. *Vet. Med.* **58**(2):142-145

Jani, R.G., Dave, M.R. and Jani, B.M. 1992. Hematological and biochemical changes in dogs suffering from diarrhoea. *Indian J. Vet. Med.* **12**(1): 36

Janthur, I. and Kokkles, R. 1984. Haemagglutination test for parvovirus infection in dogs. *Vet. Med.* **39**(5): 512-516

Jergens, A.E., Moore, F.M., March, P. and Milce, K.G. 1992. Idiopathic inflammatory bowel disease associated with gastroduodenal ulceration-erosion: A report of nine cases in the dog and cat. *J. Am. Anim. Hosp. Assoc.* **28**: 21-26

Jones, B.D. and Liska, W.D. 1986. Medical diseases of the small intestine. *Canine and Feline Gastroenterology.* W.B. Saunders Company, Philadelphia, p.531

Joshi, D.V., Singh, S.P., Sharma, S.N. and Patel, B.J. 2001. Clinico-pathology of experimental canine parvovirus infection. *Indian J. Anim. Sci.* **71**(6): 543-544

Kaneko, J.J., Harvey, J.W. and Bruss, M.L. 1997. Blood analyte reference values in small and some laboratory animals. *Clinical Biochemistry of Domestic Animals.* Fifth edition. Academic Press, San Diego. p.932

Kelly, W.R. 1974. *Veterinary Clinical Diagnosis*. Second edition. Bailliere Tindall, London, p.374

*Koene, M.G.J. and Houwers, D.J. 1999. Giardia, a potential cause of diarrhoea. *Tijdschrift-voor-Diergeneeskunde.* **124**(10): 310-312

*Koiwa, M., Hatsugaya, A., Abe, T. and Minami, S. 1990. Therapeutic effects of electrolyte solution with oil emulsion on serious diarrhoea in Holstein calves. *Jpn. J. Vet. Sci.* **52**(3): 639-641

Kokubu, T., Takahashi, T., Takamura, K., Yasuda, H., Hiramatsu, K. and Nakai, M. 1993. Isolation of reovirus type 3 from dogs with diarrhoea. *J. Vet. Med. Science.* **55**(3): 453-454

*Koutinas, A.F., Michelakis, M.N., Rallis, T., Kritsepi, M. and Moraitoo, K. 1999. Acid-Base status and electrolyte concentrations in 32 dogs with parvoviral enteritis. *Bull. Hellenic Vet. Med. Soc.* **50**(4):305-309

Kumar, R. and Mandial, R.K. 2002. Clinico-biochemical and therapeutic studies on clinical colibacillosis in cross bred calves. *Indian Vet. J.* 79(7): 672-676

Leib, M.S. and Matz, M.E. 1995. Diseases of the large intestine. *Textbook of Veterinary Internal Medicine: Diseases of the Dog and Cat.* Vol.2 (eds. Ettinger, S.J. and Feldman, E.C.). Fourth edition. W.B. Saunders Company, Philadelphia. pp.1232-1260

Leib, M.S. and Zajac, A.M. 1999. Giardiasis in dogs and cats. *Vet. Med.* **94**(9):793-800

Levitt, L. and Bauer, M.S. 1992. Intussusception in dogs and cats: A review of 36 cases. *Can. Vet. J.* **33**: 660-664

Lindsay,D.S. 1989. Diagnosing and controlling *Isospora suis* in nursing pigs. *Vet. Med.* **84**(4): 443-446

Ludlow, C.L. and Davenport, D.J. 2000. Small intestinal bacterial overgrowth. *Kirk's Current Veterinary Therapy. Small Animal Practice* (ed. Bonagura, J.D.). Thirteenth edition. W.D. Saunders Company, Philadelphia, pp.637-641

Macartney, L., McCandlish, I.A.P., Thompson, H. and Ornwell, H.J.C. 1984. Canine parvovirus enteritis. 1. Clinical, haematological and pathological features of experimental infection. *Vet. Rec.* **115**(10): 201-10

Maiti, S.K., Rao, V.N., Pal, S. and Ali, S.L. 1999. Clinico-haematological and therapeutic studies in parasitic gastroenteritis in sheep. *Indian Vet. J.* 76(5): 435-437

Marshall, J.A., Healey, D.S., Studdert, M.J., Scott, P.C., Kennett, M.L., Ward, B.K. and Gust, I.D. 1984. Viruses and virus-like particles in the faeces of dogs with and without diarrhoea. *Aust. Vet. J.* **61**(2): 33-38

Mason, M.J., Gillett, N.A. and Muggenburg, B.A. 1987. Clinical, pathological and epidemiological aspects of canine parvoviral enteritis in an unvaccinated closed beagle colony. *J. Am. Anim. Hosp. Assoc.* **23**: 183-186

Michell, A.R. 1974. Body fluids and diarrhoea : dynamics of dysfunction. *Vet. Rec.* 94(14): 311-315

*Michell, A.R. 1994. Salt, water and survival:acid tests and basic advances in fluid therapy. *Irish Vet. J.* 47:3-8.

Mochizuki, M., Kawanishi, A., Sakamoto, H., Tashiro, S., Fujimoto, R. and Ohwaki, M. 1993. A calicivirus isolated from a dog with fatal diarrhoea. *Vet. Rec.* **132**(11): 221-222

Mohan, R., Nauriyal, D.C., Singh, K.B., Mangat, A.P.S. and Singh, G.K. 1992. Haemagglutination and Haemaglutination inhibition tests for diagnosis of parvo viral infection in dogs. *Ind. J. Vet. Med.* **12**(1): 1-3

*Moon, H.W. 1974. Pathogenesis of enteric diseases caused by *E. coli. Adv. Vet. Sci. Comp. Med.* **18**: 179-211

Morgan, H.C. (1967). The effect of helminth parasitism on the hemogram of the dog. *Vet. Med. Small Anim. Clinician.* **62**(5): 218-221

Mousa, H.A.A., Ahmed, L.A., Shahein, M.A. and Ali, N.M. 2001. Isolation and identification of canine adenovirus. *Vet. Med. J.* **49**(3):371-376.

*Navarro, I.T., Kano, F.S., Ogawa, L., Freire, R.L. and Vidotto, O. 1997. Occurrence of *Cryptosporidium* spp. in diarrhoeic dogs seen at the Veterinary Hospital of the State University of Londrina, Parana, Brazil. *Semina-Londrina.* **18**(1): 23-25

*Niphadkar, S.M., Narsapur, V.S. and Deshpande, V.S. 1979. Incidence of intestinal parasites in pet dogs in Bombay. Personal communication.

Oser, B.L. 1971. *Hawk's Physiological Chemistry.* Fourteenth edition. Tata McGraw Hill Publishing Ltd., New Delhi. p.1472

*Prange, H., Gunther, H., Katenkamp, D., Keil, E., Schimke, E., Schmidt, A., Schneider, R., Schubert, H. and Zieger, M. 1983. Laboratory diagnosis and histopathology of canine parvovirus infection. *Monatsheftefur Veterinar medizin.* **38**(22): 856-861

Praveen, K., Garg, S.K., Bandyopadhyay, S.K., Rashmi, S. and Sameer, S. 2003. Haemagglutinating activity of canine parvovirus. *Indian J. Anim. Sci.* **73**(2):123-125

Rachman, M. and Pollock, S. 1961. Treatment on canine coccidiosis. *Vet. Med.* **56**(2):75-76

Radostits, O.M., Blood, D.C. and Gay, C.C. 1994. *Veterinary Medicine.* Eighth edition. Bailliere-Tindal, London. UK. p.1720

Rai, A., Nauriyal, D.C. and Mohan, R. 1994. Faecal examination for diagnosis of canine parvovirus haemorrhgic gastroenteritis. *Int. J. Anim. Sci.* 9(2):195 196

Ramprabhu, R., Prathaban, S., Nambi, A.P. and Dhanapalan, P. 2002. Haemorrhagic gastroenteritis in dogs - a clinical profile. *Indian Vet. J.* 79(5): 374-376

Renault, L., Schaelz, N., Vozenin, N. and Guillot, M.H. 1977. Role of *E.coli.* in gastroenteritis in dog. *Anim. de Comp.* **11**:95-100

Rodrigues, C., Jagadish, S. and Bhalerao, D.P. 1994. Clinical studies on canine Diarrhoea. A report on 106 cases. *J. Bombay Vet. Coll.* 5(1&2): 67-69

Rodriguez, F., Fernandez, A., Monteros, A.E., Wohisein, P. and Jensen, H.E. 1998. Acute disseminated candidiasis in a puppy associated with parvoviral infection. *Vet. Rec.* **142**(16):434-436

Roussel, A.J. and Kasari, T.R. 1990. Using fluid and electrolyte replacement therapy to help diarrheic calves. *Vet. Med.* **85**(3):.303-311

*Ruckstuhl, N., Deplazes, P. and Reusch, C. 2002. Clinical signs and course of disease in dogs with *Trichuris vulpis* infection. *Kleintierpraxis.* 47(1):19-26

*Sayed, A.S., Sadiek, A.H., Ali, A.A. and Ismail, M.N. 1998. Clinical and laboratory investigations on diarrhoea in camels in association with stress factors in assiut governorate. *Assiut Vet. Med. J.* **40**(79): 83-96

*Schales, O. and Schales, S.S. 1941. *J. Biol. Chem.* **140**:879. Cited in the operation instructions of the reagent kit to estimate chloride-Qualigens fine chemicals, Mumbai.

Schalm, O.W., Jain, N.C. and Carrol, E.J. 1975. *Veterinary Haematology.* Fifth edition. Lea and Feibiger, Philadelphia. p.1344

Sherikar, A.A., Paranjape, V,L. and Konish, S.I. 1989. Incidence of canine parvoviral enteritis in Bombay and its diagnosis. *J. Bombay Vet Coll.* 1(1):7-15.

*Shivanandiah, T.M. 1970. Use of tender coconut water in gastro-enteritis. *Cev. Cocon. Quart.* **21**:113-114.

Snedecor, G.W. and Cochran, W.G. 1980. *Statistical Methods.* Ninth edition. Oxford-IBH Publishing Co., Calcutta. p.584

Spielman, B.L. and Garvey, M.S. 1993. Hemorrhagic gastroenteritis in 15 dogs. *J. Am. Anin. Hosp. Assoc.* **29**(4): 341-44

Strombeck, D.R. and Guileford, W.G. 1991. *Small Animal Gastroenterology.* Second edition. Wolfe Publishing Ltd., London. p.745

Sullivan, G., Durham, P.J.K., Smith, J.R. and Campbell, R.S.F. 1984. Experimentally induced severe canine parvoviral enteritis. *Aust. Vet. J.* **61**(1): 1-3

Suresh, R.V., Rajan, T.S.S. and Gnanaprakasam, V. 1994. Haematological and serum biochemical changes in canine gastroenteritis. *Indian Vet. J.* **71**(6): 518-519

Tennant, B., Harrold, D. and Reina, G.M. 1972. Physiologic and metabolic factors in the pathogenesis of neonatal enteric infections of calves. *J. Am. Vet. Med. Assoc.* **161**(8): 993-1007

Tiezt, N.W. 1976. *Fundamentals of Clinical Chemistry.* WB Saunders Co., Philadelphia. p.885

*Udonsi, J.K. and Agunama, L. 1991. Experimental studies on canine ancylostomiasis : morbidity, fecundity and haematological changes in the dog host. *Bull. Anim. Health and Production in Africa.* **32**(2): 177-179

Udupa, K.G. and Sastry, K.N.V. 1997. Canine parvovirus infections: Part-II. Prevalence of clinical cases of gastroenteritis. *International J. Anim. Sci.* **12**(1): 79-82

Vandenberghe, J., Lauwers, S., Plehier, P. and Hoorens, J. 1982. *Campylobacter jejuni* related with diarrhoea in dogs. *Br. Vet. J.* **138**(4): 356-361

*Vandersteen, I., Rohde, J., Zentek, J. and Amtsberg, G. 1997. Dietary effects on the occurrence of *clostridium perfringens* and its enterotoxin in the intestine of dogs. *Kleintierpraxis.* **42**(11): 855-936

Vihan, V.S. and Singh, N. 1989. Strains of *Escherichia coli* serotypes isolated from diarrhoeic kids and their resistance to drugs. *Indian J. Anim. Sci.* **59**:655-656

*Voros, K., Papp, L. and Horvath, Z. 1981. Parvovirus enteritis in the dog : clinical symptoms. *Magyar Allatorvosok Lapja.* **36**(4): 240-246

Wagstaff, A.J., Maclean, I., Mitchell, A.R. and Holmes, P.H. 1992. Plasma and extracellular volume in calves: comparison between isotopic and 'cold' techniques. *Res. Vet. Sci.* **53**(3): 271-273

Weaver, A.D. 1977. Canine intestinal intussusception. *Vet. Rec.* **100**(19): 524-527

Weese, J.S., Staempfli, H.R., Prescott, J.F., Kruth, S.A., Greenwood, S.J. and Weese, H.E. 2001. The roles of *Clostridium difficile* and enterotoxigenic *Clostridium perfringens* in diarrhoea in dogs. *J. Vet. Internal Med.* **15**(4): 374-378

Young, D.A.B. 1964. A method for extraction of Evan's blue from plasma and tissues. *Proc. Soc. Exp. Biol. Med.* **116**: 220-221

Zafar, M.S., Khan, S.A. and Rabbani, A. 1999. Haematological studies and estimation of electrolytes in dogs exhibiting diarrhoeal signs. *Pakistan Vet. J.* **19**(1): 35-39

* Originals not seen

FLUID AND ELECTROLYTE STATUS IN DIARRHOEIC CONDITIONS OF DOGS

By

JABINA MARTHA PHILIP

ABSTRACT OF THE THESIS

Submitted in partial fulfilment of the requirement for the degree of

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences

Kerala Agricultural University

Department of Clinical Medicine
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY, THRISSUR – 680 651
KERALA, INDIA

2003

ABSTRACT

The study "Fluid and electrolyte status in diarrhoeic conditions of dogs" was conducted in the Department of Clinical Medicine during the period of one and a half years from June 2001 to October 2002, to evaluate plasma volume and electrolyte status, haemogram and the possible etiological factors in diarrhoeic dogs. Detailed investigations of twenty clinical cases and six healthy normal cases were done after making a tentative diagnosis based on history. Blood and faecal samples were collected on the day of admission to the hospital. Detailed laboratory examination of faecal samples, blood and microbiological studies were carried out. Haematological parameters were estimated as described by Schalm *et al.* (1975). Biochemical analyses were done under standard conditions of operation as described, using photometer 5110 (Boehringer Mannheim). Plasma volume was determined as per the protein precipitation and extraction method of Hunsaker (1965).

Ancylostomes was identified as the major parasitic cause for diarrhoea. Though bacteria could be isolated from all the cases, it could not give a true reflection of the actual disease. A low HA titre was obtained for the canine parvoviral infection.

Haemogram revealed a significantly lower value for haemoglobin. Biochemical analysis revealed reduced sodium and bicarbonate level indicative of their excess loss through faeces. Significantly lower level of plasma protein

observed in the study suggested low level of nutrition and partial or complete anorexia in clinical cases.

Based on the haematological and biochemical findings suitable formulations were suggested with inclusion of deficient electrolytes, haematinics or other supportive measures in the therapeutic management of diarrhoea.