A CLINICAL STUDY ON THE DISEASES OF ESOPHAGUS AND STOMACH WITH SPECIAL REFERENCE TO ENDOSCOPY IN DOGS

By

SURYAWANSHI RAVIRAJ VINAYAK
M.V.Sc

THESIS SUBMITTED TO
SRI VENKATESWARA VETERINARY UNIVERSITY
IN PARTIAL FULFILMENT OF THE REQUIREMENTS
FOR THE AWARD OF THE DEGREE OF

DOCTOR OF PHILOSOPHY

DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
COLLEGE OF VETERINARY SCIENCE, RAJENDRANAGAR,
HYDERABAD-500030

JULY-2010

CERTIFICATE

Dr. SURYAWANSHI RAVIRAJ VINAYAK has satisfactorily prosecuted the course of research and the thesis entitled "**A CLINICAL STUDY ON THE DISEASES OF ESOPHAGUS AND STOMACH WITH SPECIAL REFERENCE TO ENDOSCOPY IN DOGS"** submitted is the result of original research work is of sufficiently high standard to warrant its presentation to the examination. I also certify that the thesis or part thereof has not been previously submitted by him for a degree of any university.

DATE : 03 / 07 / 2010.

PLACE: HYDERABAD

(Dr.K.B.P. RAGHAVENDER)

Major Advisor

CERTIFICATE

This is to certify that the thesis entitled "**A CLINICAL STUDY ON THE DISEASES OF ESOPHAGUS AND STOMACH WITH SPECIAL REFERENCE TO ENDOSCOPY IN DOGS"** submitted in partial fulfilment of the requirement for the degree of **DOCTOR OF PHILOSOPHY (VETERINARY SURGERY AND RADIOLOGY)** of Sri Venkateswara Veterinary University, Tirupati, is a record of the bonafide research work carried out by **Dr. SURYAWANSHI RAVIRAJ VINAYAK** under our guidance and supervision. The subject of the thesis has been approved by the student's Advisory Committee.

No part of the thesis has been submitted by the student for any other degree or diploma. The published part has been fully acknowledged. All the assistance and help received during the course of investigation have been duly acknowledged by the author the thesis.

(Dr.K.B.P.RAGHAVENDER)
Chairman of the Advisory Committee

Thesis approved by the Student Advisory Committee

Chairman : (Dr.K.B.P.RAGHAVENDER)
Professor and University Head
Department of Veterinary Surgery and Radiology
College of Veterinary Science
Rajendranagar, Hyderabad-500030. ……....

Member : (Dr. V. GIREESH KUMAR)
Associate Professor,
Department of Veterinary Surgery and Radiology
College of Veterinary Science
Rajendranagar, Hyderabad-500030. …….

Member : (Dr. G.PURUSHOTHAM)
Associate Professor
Department of Veterinary Anatomy and Histology
College of Veterinary Science
Rajendranagar, Hyderabad-500030. …….

Member : (Dr. M.SHANTHI LAKSHMI)
Associate Professor and Head
Department of Veterinary Anatomy and Histology
College of Veterinary Science
Rajendranagar, Hyderabad-500030. …….

TABLE OF CONTENTS

LIST OF ILLUSTRATIONS

LIST OF TABLES

LIST OF VIDEOS

ACKNOWLEDGEMENTS

I would like to offer millions of thanks to lord almighty from the depth of my heart for blessings on me and gave me an opportunity as well as capability to complete present research work.

I feel an immense pleasure to express my deep sense of gratitude and sincere thanks to my erudite teacher and mentor Chairman of Advisory committee ***Dr. K.B.P. Raghavender****, Professor and University Head, Department of Veterinary Surgery and Radiology, College of Veterinary Science, Rajendranagar, Hyderabad for his guidance, suggestions in planning, constant inspiration and encouragement for executing my thesis work.*

It's my great honour to offer sincere thanks to ***Dr. V. Gireesh Kumar****, Associate Professor, Department of Veterinary Surgery and Radiology, College of Veterinary Science, Rajendranagar, Hyderabad, member of advisory committee for his keen interest, noble ideas, valuable and timely co-operation during the prosecution and representation of this research work.*

I deem it my honour to offer thanks to ***Dr.M Shanthi Lakshmi*** *Associate Professor and Head, Department of Veterinary Anatomy and Histology, College of Veterinary Science, Rajendranagar, Hyderabad member of my advisory committee for her co-operation, plan of work and valuable suggestions during my thesis work.*

I am extremely grateful for to ***DR. G. Purushotham****, Associate Professor, Department of Veterinary Anatomy and Histology, College of Veterinary Science, Rajendranagar, Hyderabad member of my advisory committee for his guidance, valuable suggestions and constant inspiration for executing my research work.*

I would like to thanks to ***Dr.P.Ameer Hamza,*** *Associate Dean****,*** *College of Veterinary Science, Rajendranagar, Hyderabad for providing the necessary facilities and cooperation during the present study.*

I am extremely thankful to ***Dr.A.Anand Kumar****, Professor and Head,* ***Dr M Lakshman****, Assistant Professor, Ruska Laboratory, Department of Veterinary Pathology and* ***Dr. M. Gnyanaprakash****, Associate Professor, Department of Animal*

Genetics and Breeding, College of Veterinary Science, Rajendranagar, Hyderabad for their timely cooperation during the present study.

I am also extremely thankful to ***Dr.E.L.Chandrasekhar,*** *and* ***Dr. T. Madhavarao****, Associate Professor,* ***Dr. C. Latha, Dr. S. Bharathi****, and* ***Dr.Radhakrishna****, Assistant Professor, Department of Surgery and Radiology, College of Veterinary Science, Rajendranagar, Hyderabad for their help and kind co-operation in successful completion of this research work.*

I would like to thanks my colleagues and juniors ***Dr.S.Verghese,Dr. Abhisekh, Dr.V.Kadambari, Dr.M Vijaykumar, Dr.Sunil Ghomase, Dr. Swapnil Jintpure, Dr. Suresh Naik, Dr. Alok Bharathi*** *and those who one really have missed and contributed in the completion of present research work.*

Last but not the least I would like to thank ***Sri. G. Madhava Reddy****,* ***Sri. K. Sataiah****,* ***Sri Bhaskar****,* ***Sri kumar****,* ***Smt. Anasuja*** *and* ***Late B.V.Raja*** *for their co-operation during the research work. I wish to extend my thanks to one who really have missed and contributed in the completion of this study.*

I feel sorts of words in expressing my deep feeling of affection and love to my father ***Vinayak*** *and mother* ***Jayashri*** *who inspired me to make things always better and for their hearty blessings, constant encouragement and efforts that lead my career to present stage. I am extremely grateful to my younger brother* ***Kiran*** *and sister* ***Kalpana*** *and* ***Mayuri*** *for their heartful support, encouragement and cooperation.*

(Dr Ravi Suryawanshi)

DECLARATION

I, **Dr. Suryawanshi Raviraj Vinayak**, hereby declare that the thesis entitled "**A CLINICAL STUDY ON THE DISEASES OF ESOPHAGUS AND STOMACH WITH SPECIAL REFERENCE TO ENDOSCOPY IN DOGS"** submitted to Sri Venkateswara Veterinary University for the degree of **DOCTOR OF PHILOSOPHY** in **SURGERY AND RADIOLOGY,** is the result of original research work done by me. I also declare that the thesis or part thereof has not been published earlier elsewhere in any manner.

DATE : 03 / 07/ 2010

PLACE: HYDERABAD **(DR.SURYAWANSHI R. V.)**

Name	**: SURYAWANSHI RAVIRAJ VINAYAK**
Title of thesis	**: A CLINICAL STUDY ON THE DISEASES OF ESOPHAGUS AND STOMACH WITH SPECIAL REFERENCE TO ENDOSCOPY IN DOGS**
Degree to which It is submitted	**: DOCTOR OF PHILOSOPHY**
Faculty	**: VETERINARY SCIENCE**
Discipline	**: VETERINARY SURGERY AND RADIOLOGY**
Major Advisor	**: DR. K.B.P.RAGHAVENDER**
University	**: SRI VENKATESWARA VETERINARY UNIVERSITY, TIRUPATI**
Year of submission	**: JULY-2010**

ABSTRACT

The present study was undertaken in 42 cases out of the 120 clinical cases of dogs presented with diseases of esophagus and stomach with the history of persistent vomiting or regurgitation. A total of 120 dogs were presented with the chief complaint of recurrent vomiting or regurgitation.

Out of the total of 120 cases selected for closer examination, 78 dogs (65%) responded to routine medical treatment with antiemetics, antibiotics and fluid therapy and hence, they were excluded from the purview of the present clinical study. The remaining 42 dogs (35%), which continued to vomit or regurgitate for more than one week and showed signs consistent with the primary diseases of the esophagus and stomach formed the patient material for the present clinical study. All the 42 clinical cases of dogs underwent routine physical, clinical, haematological and biochemical examinations as well as plain and contrast radiographic, Ultrasonographic and endoscopic assessment to rule out the concurrent diseases prior to the consideration in the present study.

The results of the present study revealed that out of the total 42 cases selected for the present study, 31dogs (73.81%) had diseases of the esophagus while the rest

(11dogs; 26.19%) had diseases of the stomach. During the present study, megaesophagus was diagnosed in 18 (42.86%) dogs, reflux esophagitis in 2 dogs (4.76%), vascular ring anomalies in 2 dogs (4.76%), esophageal diverticula and esophageal nodule in one dog each (2.38% each), esophageal foreign bodies in 5 dogs (11.90%), esophageal erosion or ulcers in 2 dogs (4.76%), chronic gastritis in 3 dogs (7.14%), gastric foreign bodies in 4 dogs (9.52%), gastric erosion/ ulcer in 2 dogs (4.76%) and gastric dilatation and Volvulus in 2 dogs (4.76%). The results also showed that the occurrences of these diseases were seen in dogs ranging from two and a half months to thirteen years of age. No sex wise occurrence of the esophageal or stomach diseases was reported. All the dogs afflicted with the various diseases exhibited similar symptoms like emaciation, shrunken abdomen, chronic weight loss, prominent rib cage, regurgitation, vomiting or both.

In the present clinical study, 18 clinical cases of megaesophagus were recorded. The disease was encountered in Labrador (2 dogs), Golden Retriever (2 Dogs), German Shepherd (5 Dogs), Doberman (2 Dogs), Mongrel (3 Dogs), Pomeranian (2 Dogs), Cocker Spaniel (1 Dog) and Boxer (1 Dog) breeds of dogs. The mean age of occurrence was found to be 6.25 ± 0.88 years. Out of these 18 dogs, 11were found to be males (61.12 %) and the rest were females (7 dogs; 38.88%). All these 18 dogs showed the signs of regurgitation of the food soon after food consumption. The haematological and biochemical studies showed that all the parameters studied were with in the normal range, except for haemoglobin. Ultrasonography did not reveal any sort of esophageal pathology.

Radiographic features of megaesophagus in the seven dogs were air filled dilated esophagus and tracheoesophageal stripe sign. Esophagography clearly revealed generalized distention of esophagus in all the 18 dogs. While endoscopy revealed markedly dilated, flaccid esophagus and pooling of retained fluid, in the most dependent segment of the esophagus. The results of the present study indicated that among the three treatment regimens tested, Metoclopramide combined with feeding the dogs in an upright position from an elevated platform improved the esophageal function to the maximum extent.

Post mortem examination of the seven dogs died or euthanized showed severe dilatation and thinning of the esophageal wall, while histopathological examination showed scanty muscle bundles, infiltration of polymorphonuclear cells with submucosal congestion and enlargement of submucosal glandular pattern with

epithelial irregularity. Scanning electron microscopy revealed destruction of blood vessels, loss of normal architecture and direction of inner circular as well as outer longitudinal muscle fibers while, transmission electron microscopic examination showed complete loss of cellular architecture, mitochondrial destruction and complete loss of architecture of myoneuronal plate at the neuromuscular junction suggestive of neuromuscular disorder.

Two clinical cases of dogs were tentatively diagnosed to be suffering from reflux esophagitis. Endoscopic examination characterized by mucosal erythema, haemorrhage, mucosal irregularity, erosion with thickened mucosal folds especially at the lower esophageal area confirmed reflux esophagitis. Endoscopic examination showed the evidence of reflux esophagitis in these two dogs. Both the dogs recovered uneventfully following the treatment adopted.

Esophagoscopy of a nine year old male Pomeranian dog with sings of recurrent cough and retching revealed a lemon sized nodular growth appearing as broad based protuberance with nipple like orifice located just caudal to the base of the heart suggestive of Spirocerca lupi infestation. No surgical exploration was conducted as the dog was not available for further investigation.

Plain radiography of five cases out of 42 cases (11.90%) revealed osseous foreign bodies in the esophagus of four dogs and radiolucent foreign bodies (socks) in an other dog. In three dogs, osseous foreign bodies located in the thoracic esophagus were pushed in to the stomach successfully by endoscope itself. In dog, osseous foreign bodies located in the post pharyngeal was retrieved by cheatle forceps under the endoscopic illumination. The dog with socks in the cervical esophagus vomited the socks out due to emetic effects of xylazine prior to endoscopic manoeuvre. All dogs recovered uneventfully.

Barium swallow of the dogs revealed stricture of esophagus over the base of heart and cranial distention of esophagus suggestive of vascular ring anomalies. Despite of giving supportive therapy, both the dogs died. Necropsy revealed distention of esophagus cranial to the constructive vascular band confirmed vascular ring anomalies. Histopathological examination further confirmed presence of concurrent gastritis.

Plain radiography of three year old female German shepherd dog with post prandial regurgitation, revealed saclike out pouching of cranial thoracic esophageal

wall that rested on floor of sternum which was further confirmed by endoscopy. No treatment was initiated in this case on the owner was reluctant for surgery.

The two dogs with vomiting of about three weeks duration that did not respond to routine medication were diagnosed to be suffering from esophageal erosions/ulcers, both by endoscopy and by positive contrast radiography. The dogs responded well to the treatment adopted.

Gastroscopy of all three dogs revealed generalized congestion as well as petechial hemorrhagic spots on gastric mucosa with minor areas of mucosal erosions suggestive of chronic gastritis. Cytological examination of stomach fluid collected during endoscopy further confirmed chronic gastritis. No *Helicobacter* Like Organisms (HLO) was encountered. These three dogs responded well to the treatment protocol adopted i.e. combination of amoxicillin, clarithromycin, Metronidazole and ranitidine.

In the present study, gastroscopy of two dogs revealed gastric ulcerative patches with marked thickening and irregular edges confirmed gastric erosion or ulcers. Both the dogs treated with sucralfate and omperazole responded well to complete recovery.

In present clinical study, four dogs were diagnosed to be affected with gastric foreign bodies. Out of four cases, two dogs that underwent plain radiographic examination revealed presence of sewing needle in the stomach of one dog and a pair of metal caps of cool drink bottle and a safety pin in another dog. The metallic foreign bodies whose removal was considered to be too risky by endoscope were removed by standard gastrotomy. In the remaining two dogs that swallowed dog nylon belt, plain radiograph did not confirm their presence. Endoscopic examination revealed presence of dog collar made up of nylon in the stomach. Endoscopic retrieval of the nylon belt in the stomach of one dog was successfully done while, in the second case, the radiolucent foreign body (another nylon belt), which turned out later on to be a linear foreign body, was removed successfully by standard gastrotomy and enterotomy operation. All the dogs recovered uneventfully.

In the two dogs with gastric dilatation and volvulus (GD-V) in the present study, the clinical and radiographic signs helped in confirmation of the disease and the clinical out come of their treatment with surgical derotation and gastropexy was uneventful.

From the results of the present clinical study, it was concluded that about 35% of dogs (42 out of 120 dogs) that were refractory to general medical treatment for vomiting or regurgitation were appeared to be affected with primary diseases of esophagus and stomach. The most common malady affecting the esophagus in dogs was found to be megaesophagus (18), and this can be reasonably well managed by using metoclopramide and feeding the dogs in an upright position from an elevated platform. The disease is considered to be a neuromuscular disease as seen during scanning and transmission electron microscopy. The neuromuscular junctions as seen by transmission electron microscopy appeared to be destroyed in cases of megaesophagus.

Esophagography was found to be more useful procedure in confirming the cases of megaesophagus than endoscopy. This is because it is sometimes difficult in mild cases to be sure whether the esophagus was really dilated or it appeared so due to insufflation during endoscopy. On endoscopy examination, a large cavernous lumen extending the length of the esophagus is vividly suggestive of megaesophagus. But a normal esophagus can also appear flaccid and distended under anaesthesia. The other diseases encountered in the present study in their order frequency of occurrence are esophageal foreign bodies (5), gastric foreign bodies (4), chronic gastritis (3), reflux esophagitis (2), esophageal erosion/ulcer (2), gastric erosion/ ulcer (2), vascular ring anomalies (2), gastric dilatation and volvulus (2), esophageal nodule (1) and esophageal diverticulum (1).

Endoscopy is not always useful in treatment of esophageal or gastric foreign bodies in dogs for the shape and size of foreign body, duration of obstruction and nature and location of foreign body in the esophagus and stomach determine the use and success of endoscopy for therapeutic purpose. Endoscopy proved to be a valuable and useful aid in the diagnosis of esophageal and gastric ulcers/ erosions, esophageal nodule and esophageal diverticulum. Albeit veterinarians are still in the early phase of adoption of minimally invasive procedures, endoscopy proves to be vital diagnostic and therapeutic tool of the future veterinarian’s armamentarium.

LIST OF ABBREVIATIONS

%	-	Percentage
dl	-	Decilitre
µm	-	micro millilitre
ALT	-	Alanine amino Transferase
AST	-	Asparate amino Transferase
BUN	-	Blood Urea Nitrogen
CBP	-	Complete Blood Picture
Cumm	-	Cubic millimetre
Fig	-	Figure
GD-V	-	Gastric Dilatation- Volvulus
GERD	-	Gastro-Esophageal Reflux Disease
GES	-	Gastroesophageal sphincter
GIT	-	Gastro-Intestinal Tract
H & E	-	Haematoxylin and Eosin
Hb	-	Haemoglobin
i/m	-	Intramuscular
HLO	-	Helicobacter Like Organism
Kg	-	Kilogram
Mg/dl	-	milligram per decilitres
MHz	-	Meter hertz
ml	-	millilitres
NMJ	-	Neuro-Muscular Junction
PRAA	-	Persistent Right Aortic Arch
S.E	-	Standard Error
SEM	-	Scanning Electron Microscopy
TEC	-	Total Erythrocyte Count
TEM	-	Transmission Electron Microscopy
TLC	-	Total Leukocyte Count
U/L	-	Unit per liter
VRA	-	Vascular Ring Anomalies

CHAPTER-I

INTRODUCTION

Vomiting is one of the most common symptoms reported by the clients to the practicing veterinarians. It is also not uncommon for the practicing veterinarians to think of vomiting as a sign of gastritis. Most of the times, the clients cannot even distinguish between vomiting and regurgitation. It is common veterinary practice to treat vomiting dogs by routine medication, especially when this symptom occurs in a season that coincides with outbreaks of diseases like canine Parvoviral gastroenteritis. It is quiet uncommon for a field veterinarian to examine vomiting dogs in detail and as a result, dogs continue to be treated routinely in an attempt to control vomiting or regurgitation. Attempts are seldom made to zero down on primary diseases of the esophagus or the stomach.

Vomiting is also a common clinical sign of many diseases of the esophagus and stomach (Guilford and Strombeck 1996; Ramprabhu *et al* 2001; Ettinger and Feldman 2005; Raekallio *et al* 2006; Luna *et al* 2007 and Harvey 2008) in addition to being one of the symptoms of many infectious and other diseases. Esophagus and stomach lend themselves for closer detailed examination in several ways. Palpation, plain and contrast radiographic examination, ultrasonography and endoscopy are some of the commonly employed diagnostic methods.

Plain and contrast radiography are useful for detection of many diseases including esophageal or gastric foreign bodies (Chiang and Chou 2005; Guilford 2005; Mylonakis *et al* 2004; Glazer and Walters 2008 and Ponnuswamy *et al* 2009) and are performed before endoscopy to make sure that there are no masses, lesions or unexpected foreign bodies which are beyond the reach of the endoscope. Contrast radiographic procedures do not need anaesthesia and offers a better estimation of

esophageal diameter and function, and gastric motility and emptying. However, endoscopy is more sensitive for detection of mucosal diseases and renders the clear advantage of a potential, definitive diagnosis through biopsy (Tams 2003; Gualtieri 2001; Guilford 2005 and Hall 2008).

Today in veterinary medicine ‘minimally invasive’ usually refers to flexible endoscopy for diagnosis and rigid endoscopy for both diagnosis and surgery. An endoscopy technique makes diagnosis and therapy possible and effective because it gives magnification and visualization of cavernous areas of body.

Upper gastrointestinal (GI) endoscopy is frequently performed to diagnose diseases of the esophagus and stomach. It is relatively direct, safe, accurate, non-invasive, atraumatic and effective technique that permits visual examination of the esophageal and gastric lesions and allows the detection of gross abnormalities of mucosa and allows descriptive or photographic documentation of their severity and extent (Guilford 1990; Gualtieri 2001; Moore 2003; Tams 2003 and Rychlik *et al* 2007).

The upper gastrointestinal endoscopy is useful of evaluation of dysphagia, recurrent regurgitation, chronic vomiting, hematemesis, esophageal and gastric foreign bodies, reflux esophagitis, esophageal stricture, intramural mass and diverticula, etc. (Guilford 2005; Rychlik *et al* 2007; Valentine 2007 and Harvey 2008).

The primary causes of esophageal dysfunction in the dog include motility disorders (megaesophagus), inflammatory diseases (esophagitis, gastroesophageal reflux and hiatal hernia), obstructive lesions (foreign body, stricture, vascular ring anomalies and neoplasia) and miscellaneous conditions (diverticula and bronchoesophageal fistula). The common disorders of stomach include chronic

vomiting, gastric erosion/ ulcers, gastric neoplasia, gastric foreign bodies, gastric dilatation and volvulus, etc (Guilford and Strombeck 1996; Gualtieri 2001 and Guilford 2005).

Endoscopy provides for diagnostic interventions such as collection of biopsy, cytology as well as retrieval of gastric foreign bodies (Michels *et al* 1995; Moore 2003; Rychlik *et al* 2007; Valentine 2007; Dutta *et al* 2008 and Ponnuswamy et al 2009). However, none of the techniques may be capable of providing a conclusive diagnosis and is bereft of complications and limitations.

A thorough search of published literature revealed scanty information on the primary diseases of the esophagus and stomach and their therapeutic management. There is also a paucity of published literature as far as such diseases of the esophagus like megaesophagus is concerned.

Therefore, the present clinical study on the diseases of esophagus and stomach with special reference to endoscopy in dogs was contemplated with the following objectives:

1. To study the occurrence of the diseases of the esophagus and stomach and to evaluate cases of chronic vomiting by endoscopy in dogs.
2. To detect intra-luminal pathological lesions of the oesophagus and stomach and to collect biopsy / endoscopic biopsy.
3. Retrieval of esophageal and gastric foreign bodies by endoscopy, if possible.
4. To assess the clinical outcome of the cases treated.

CHAPTER II

REVIEW OF LITERATURE

2.1 DISEASES OF ESOPHAGUS AND STOMACH IN DOGS

2.1.1 DISEASES OF ESOPHAGUS IN DOGS

2.1.1.1 Megaesophagus

Clifford *et al* (1971) reported the congenital achalasia of esophagus in four cats having common ancestry. Dilatation of the esophagus subsequent to congenital persistence of right aortic arch, congenital achalasia and acquired stricture was reported.

Barber *et al* (1983) reported that ectasia (dilatation) is a sign of esophageal dysfunction rather than a specific disease and has been associated with a number of etiological factors. They also recorded achalasia of the oesophagus in man is a specific smooth muscle disorders characterised by aperistalsis of the lower two third of the oesophagus and failure of the lower esophageal sphincter to relax after swallowing. In dogs, the condition characterized by neuromuscular dysfunction is referred to as achalasia or megaesophagus. This may develop due to developmental immaturity of the innervations and or musculature.

Ross *et al* (1995) stated that, myasthenia gravis (MG), is an autoimmune disease characterised by extreme muscular weakness, the acetylcholine receptors on the sarcolemma become blocked by antibodies to the receptor protein. Thus the number of functional receptors sites is reduced and the muscle fiber can respond to the nerve stimuli in only a feeble manner leads to muscle atrophy and total loss of function in the denervated muscle.

Randelia *et al* (1990) reported that megaesophagus is a neuromuscular disorder characterised by an absence of peristalsis, flaccid dilatation resulting in retention of ingesta in the dilated segment of oesophagus. They also reported that, in the dog, rat and mouse, the muscle coat of entire esophagus consists mainly of striated muscles which are innervated by motor axons of both vagus nerves.

Sherding *et al* (1999) reported that dilation of the esophagus is associated with diffuse hypomotility. Megaesophagus can be either congenital or acquired in nature. They also stated that congenital idiopathic megaesophagus is characterized by severe dilation of the oesophagus and persistent regurgitation of food from the time of weaning while in acquired type, the onset of megaesophagus can be either idiopathic or secondary to various identifiable causes including myasthenia gravis, polymyositis, neuropathies, CNS disease, dyautonomia, lead toxicity and hypothyroidism.

Gualtieri (2001) studied esophageal hypomotility in detail and stated that the organ was diffusely enlarged, flaccid and peristalsis was ineffective. Megaesophagus could be congenital idiopathic or acquired idiopathic irrespective of breeds of dogs. It was also reported that clinically, megaesophagus was characterised by regurgitation, occurring at variable intervals after feeding is the primary sign followed by cachexia, dehydration and respiratory sings. Further, defective afferent neural response to esophageal distention and neuromuscular dysfunction i.e. myasthenia gravis, polymyositis, peripheral neuropathies, central nervous diseases, dysautonomia, hypothyroidism and hypoadrenocortisism may lead to megaesophagus.

Watrous (2002) studied that generalised megaesophagus may be due to congenital (hereditary) or acquired (idiopathic), although it may be secondary to one of many possible causes which include chest trauma, tetanus, autoimmune disease, lead toxicity, myasthenia gravis, polymyositis, polymyopathy,

hypoadrenocorticocism, thymoma, esophagitis and GDV. He also noted that, a defect in the afferent innervations might be present in both congenital and acquired form of idiopathic megaesophagus. A segmental motility disturbance affected any portion of the esophagus. Reflux esophagitis is probably the most common form of segmental dysfunction. It usually involves the caudal portion of the esophagus and may or may not be associated with haital hernia while diffuse esophagitis may produce generalized dilatation.

Michael and Goldstein (2004) reported a case of five years old castrated male German shepherd dog with chief complaint of anorexia, lethargy and regurgitation secondary to a previously diagnosed megaesophagus. The cervical esophagus was found enlarged and palpable and the lung sounds were muffled bilaterally. It was felt that megaesophagus was due to poor muscle tone and hypomotility. The congenital form is believed to be the result of aberrancies in the afferent innervations of the muscular esophagus; while in the acquired disease, it can be consequent upon innumerable anatomic and metabolic conditions which obstruct or disturb normograde peristalsis resulting in loss of muscle tonicity. The onset of acquired megaesophagus is usually seen in middle –aged dogs (mean age 8 years); predisposed breeds include German shepherd, Irish Setters and Golden retriever.

Guilford (2005) stated that dogs with megaesophagus usually had fluid and fermenting food retained in the oesophagus. The mucosal folds of over distended esophagus were often so voluminous that it was difficult to pass the endoscope to the lower esophageal sphincter. Megaesophagus is commonly seen in middle age group of the dogs irrespective to breeds of dog.

Kealy and McAllister (2005) stated that megaesophagus is dilatation of the esophagus. It may be congenital or acquired, generalised or local (segmental). A

generalized distention is more common. They also noted that, congenital megaesophagus may be hereditary in cats and within certain breeds of dogs such as German shepherd dogs, Miniatures, Labrador retriever, etc.

Keles *et al* (2007) and Johnson *et al* (2009) stated that megaesophagus is a condition characterised by decreased or absent esophageal motility that usually results in diffuse dilatation of the oesophagus. Megaesophagus occurs as a congenital disorder that becomes clinically apparent at or shortly after weaning or it can occur as an acquired disorder in a previously normal adult. Acquired megaesophagus can be secondary to a variety of diseases that because neuromuscular dysfunction or it can occur as a primary disorder for which the cause is unknown (idiopathic megaesophagus).

Johnson *et al* (2009) stated that myasthenia gravis is the most common cause of acquired megaesophagus in the dog. It occurs rarely as a congenital disease and more frequently as an acquired disease; both can cause megaesophagus. Acquired myasthenia gravis is an autoimmune disorder that interferes with normal neuromuscular transmission and also production of auto antibodies against nicotinic acetylcholine receptors; decrease the number of receptors available for normal neuromuscular transmission, resulting in skeletal muscle weakness.

2.1.1.2 Reflux Esophagitis

Fossum and Hedlund (1999) reported that persistent esophagitis may lead to chronic regurgitation because of hypomotility, delayed clearing of the distal oesophagus and gastroesophageal reflux. They also stated that, gastroesophageal reflux, subsequent esophagitis and megaesophagus are responsible for most of the clinical signs.

Sherding *et al* (1999) and Tams (1999) listed out the possible causes of esophagitis. They included injury from esophageal foreign bodies, gastroesophageal reflux secondary to general anaesthesia, persisting vomiting of any cause, hiatal hernia, gastric emptying disorders, ingestion of caustic irritants and thermal injury from micro waved food. They also reported that, gastroesophageal reflux means the movement of gastro-esophageal contents into the esophageal region unassociated with eructation or vomiting. Esophageal damage caused by gastro-esophageal reflux is primarily attributed to the duration of the mucosal contact with refluxed gastric acid, bile salts and trypsin. They also reported that factors contributing for GES relaxation include anaesthetic agents (Anticholinergic/tranquilizers), impaired swallowing and secondary peristalsis, increased abdominal pressure during surgical manipulation, gravitational effects of a tilted surgery table and impaired function of LES due to hiatal hernia.

Gualtieri (2001) reported that esophagitis in relatively common in small animals due to gastroesophageal reflux, injuries from foreign bodies, acute or chronic vomiting, anaesthesia induced esophagitis, NSAIDs induced esophagitis and chemical injuries. They noted that reflux of gastric acid into the oesophagus is the most common cause of esophagitis followed by anaesthetic drugs (atropine, meperidine, diazepam, pentobarbital, halothane, etc.) induced esophageal hypomotility, increased abdominal pressure during surgery and impairment of mechanical function of lower esophageal sphincter.

Shaheen and Ransohoff (2002) stated that gastroesophageal reflux disease (GERD) involved chronic mucosal damage produced by the abnormal reflux of gastric contents into the esophagus; Guilford (2005) reported that esophagitis may or may not be visually apparent even when marked histological changes are present.

Gross abnormalities suggestive of esophagitis include erythema, erosions, irregularity and stricture.

Watrous (2002) Stated that reflux esophagitis occurs when acidic gastric contents remain within the esophageal lumen for a prolonged period, resulting mural inflammation. Reflux may be a normal consequence of swallowing, but the retrograde flow of gastric acid should be rapidly cleared by the esophagus by either primary or secondary peristaltic contraction.

Gualtieri and Olivero (2006) reported that gastroesophageal reflux is the passage of gastric juices from the stomach into the oesophagus, a condition that may cause reflux esophagitis in people or animals. Gastroesophageal reflux is usually secondary to alteration of the lower esophageal sphincter or the diaphragmatic esophageal hiatus. They also noted that some factors that are associated with altered function of these structures are general anaesthesia, hiatal hernia, cardial incompetence, chronic vomiting, positioning of animal during surgery, delayed gastric emptying and rhino gastric tube placement.

Rousseau *et al* (2007) reported 30 dogs with mild esophagitis and other 30 with moderate to severe esophagitis due to esophageal foreign body. Twenty two of 30 dogs with mild esophagitis showed signs of lethargy, regurgitation, vomiting, gagging, coughing, dysphagia, ptyalism and retching. Duration of clinical signs before presentation was significantly shorter for dogs in mild cases when compared with moderate to severe esophagitis.

Glazer and Walters (2008), Meineri *et al* (2008) and Willard and Carsten (2009) stated that esophagitis may involve disruption of the esophageal mucosa with resultant exposure of the submucosa. Causes of the esophagitis includes gastroesophageal reflux, vomiting, ingestion of foreign bodies, caustic substances or

medication and anaesthesia induced gastroesophageal reflux is the most commonly reported cause in veterinary literature. It can cause severe complications such as esophagitis and a precancerous lesion called “barrets esophagus”.

2.1.1.3 Esophageal Neoplasm

Wandera (1976) reported 1607 cases of *Spirocerci lupi* infection from 1964 to 1974 with an incidence rate were 12.8% in autopsied dogs and revealed the close association of the parasite *Spirocerci lupi* with esophageal sarcomas suggest aetiological involvement of *Spirocerci lupi*. Thirty nine had clinical signs referable to Spirocercosis and esophageal lesions ranged from chronic inflammation to fibrosarcoma and osteosarcoma. Forty three sarcoma cases were encountered of which 29 showed symptoms of Spirocercosis characterised by aortic aneurysm, deformative spondylitis of thoracic vertebrae and hypertrophic pulmonary osteoarthropathy.

Crow (1985) and Sherding *et al* (1999) studied the incidence of esophageal neoplasm and concluded that the tumours of the canine and feline oesophagus are very rare, accounting for less than 5 per cent of alimentary tract neoplasm in both species. A strong causal relationship between esophageal sarcomas and esophageal parasite - *Spirocerca lupi*, has been established. The other neoplasm of canine and feline oesophagus included squamous cell carcinoma, undifferentiated carcinoma, fibrosarcoma, osteosarcoma and leiomyosarcoma.

Berry (2000) **re**ported that seven dogs with *Spirocerca lupi* esophageal granulomas were identified based on the site of involvement (distal oesophagus) and characteristic endoscopic appearance. On thoracic radiographs, 4 dogs had evidence of a caudodorsal mediastinal mass and 2 of these dogs had spondylitis of midthoracic vertebrae. On endoscopy single esophageal nodules were observed in 5 dogs. One dog

had 3 nodules and 1 dog had 6 nodules. He treated *Spirocerci lupi* in 7 dogs with doramectin @ 200µg/kg s/c at 14 days interval for three treatments. By six weeks, clinical signs had resolved in six dogs and esophageal nodules had completely resolved in 4 dogs; incompletely resolved in 3 dogs which was confirmed endoscopically.

Lobetti (2000) reported that a questionnaire survey of 716 veterinary practices was undertaken to determine the incidence of Spirocerci lupi in dogs in South Africa and revealed no seasonal incidences of Spirocerci lupi was reported by 48% of respondents. They also stated those large breeds were considered to be at higher risk by 43% of respondents. No specific age or sex was identified to be at higher risk. The most common complaints by owners and clinical findings were vomiting (46%), weight loss (27%), coughing (21%) and regurgitation (20%), although 14% of respondents reported no abnormal findings.

Gualtieri (2001), Fossum and Hedlund (2002) and Head *et al* (2002) reported that adult *Spirocerci lupi* worm occasionally is seen protruding into the lumen from the mass. Identification of granulomas with nipple like orifice is characteristics of Spirocercosis. They also reported that esophageal neoplasia is rare and usually malignant. The most common types of tumour are sarcomas, squamous cell carcinoma and leiomyoma. Clinically characterised by regurgitation, dysphasia, melena, salivation, cough, neurological signs and weight loss. Esophagoscopy allows direct visualization of intraluminal masses and biopsy for definitive diagnosis.

Mylonakis *et al* (2004) and Mylonakis *et al* (2008) stated that clinical Spirocercosis occur more often in young-adult, large breed dogs and may transform to sarcomas; aortic aneurysms; mid-thoracic spondylitis; hypertrophic osteopathy; salivary gland necrosis and pyothorax.

Kealy and McAllister (2005) stated that primary neoplasia of the esophagus is rare in dog, Osteosarcoma or fibrosarcoma may occur secondary to *Spirocerca lupi* granuloma leads to partial or complete obstruction is a possible sequel. They also stated that Spirocerca lupi provokes a grannulomatous reaction, which appears on radiographs as an area of increased opacity between the base of the heart and the diaphragm.

2.1.1.4 Esophageal Foreign Bodies

Beitzel and Brinker (1956) reported the case of esophageal foreign body in a female boxer dog with complaint of sudden onset of illness, lethargy, loose stools and vomiting. A plain radiograph of cervical region revealed a pork chop bone piece lodged in the proximal oesophagus just above the cardia region.

Michels *et al* (1995) stated that the criteria for selection of cases regarding retrieval of fishhooks from oesophagus and stomach includes patient signalment, body weight, and time of year the animal was examined, clinical signs, type of fish hook, and previous attempt at removal performed by the owner or referring veterinarian, radiographic location of the fishhook, orientation (for fishhook in oesophagus and stomach), amount of time taken for removal, type of endoscope equipment, method of retrieval, hospitalization time and complications. They also reported medical records of 3 cats and 72 dogs that had a fishhook endoscopically or surgically retrieved from the stomach or esophagus. Endoscopic retrieval was successful in 41 of 62 (66%) animals and retrieval time and hospitalization time for endoscopic retrieval were shorter those times for surgical retrieval.

Fossum and Hedlund (1999) stated that esophageal foreign bodies generally lodge into mucosa because they are too large to pass or they have puts excessive pressure on the oesophagus or if it remains at one location for several days, they cause

pressure necrosis and ulcers and can cause esophageal perforation. They also stated that sharp objects may also perforate the esophageal wall and allow bacteria, ingesta and secretions to contaminate the perioesophageal area.

Sherding *et al* (1999), Griffiths *et al* (2000), Gualtieri (2001) and Fossum and Hedlund (2002) stated that esophageal foreign bodies are common cause of esophagitis. Bones are the most frequent foreign bodies to become lodged in the esophagus. Other esophageal foreign bodies include pins, needles, fish hooks, string, toys and vomited hairballs in cats. They also reported that most esophageal foreign bodies lodge at the thoracic inlet, the base of heart and the hiatus of the diaphragm. The extent of secondary esophageal mucosal injury depends upon the type of objects, its size and shape, length of time it is in contact with the mucosa. Foreign bodies with sharp edges and points such as bones may lacerate the mucosa or be come embedded in it, whereas tightly wedged objects can cause local pressure necrosis of the mucosa.

Watrous (2002) reported entrapment of esophageal foreign bodies within the esophageal lumen is a common cause of esophageal dysphagia due to ingestion of non-digestible material or pieces of food too large to pass through the esophagus can lead to entrapment. Typical feature of obstructing foreign bodies include a firm or rigid consistence; a sharp, angular or speculated shape; or a large size relative to the least distensible region of esophagus. Bones, fishhooks and needles are common esophageal foreign bodies comparative to trichobezoars and linear foreign bodies. He also mentioned usual sites of obstruction of foreign bodies include cranial cervical esophagus, thoracic inlet, base of heart, caudal thoracic esophagus and cranial to esophageal hiatus.

Lieb and Sartor (2008) reported that esophageal obstruction with dental chew treat occurred primarily in small dogs (26/31). The most common clinical signs were

gagging, regurgitation, vomiting, anorexia and lethargy. Diagnosis was made via survey thoracic radiography.

Esophageal foreign bodies are frequent causes of dysphagia and regurgitation in the dog because esophageal foreign bodies pose sustained risk for injury when lodged. Damage to the esophagus is influenced by the foreign body type, size, shape, sharpness and duration of obstruction. Clinical signs associated with esophageal foreign bodies are variable. They also noted that esophageal foreign body may cause early complications such as esophagitis, esophageal perforation, mediastinitis, pneumothorax and aortic perforation. Esophageal foreign bodies can be safely extracted during esophagoscopy. However, various factors including impaction if several days, sharp foreign bodies and those with protruding parts, increase the danger of perforation when pulled out at esophagoscopy (Lieb and Sartor 2008; Rousseau *et al* 2007 and Weissberg 2008).

2.1.1.5 Vascular Ring Anomalies (VRA)

Fossum and Hedlund (2002) and Koc *et al* (2004) stated that vascular ring anomalies are congenital malformation of the great vessels and their branches that cause constriction of the oesophagus and signs of esophageal obstruction. Persistent right aortic arch, the most common type of vascular ring anomaly in dog and cat. Clinically, acute onset of regurgitation starts when solid or semisolid food is fed. Regurgitation of undigested food occurs soon after eating early in the disease. The other symptoms are postprandial regurgitation of solid food after weaning, stunted growth and unthriftyness. They also noted that tentative diagnosis is made on the basis of history, physical examination and contrast esophagogram with megaesophagus and constriction of the esophagus at the base of the heart.

Watrous (2002) stated that the most common malformation leading to entrapment of the esophagus is persistent right aortic arch; this connects to the main or left pulmonary artery via ductus arteriosus (or ligamentum arteriosus after birth), which forms the left sixth arch. Constriction occurs with the esophagus dorsal to the heart base but ventral to the vascular created by the aortic arch located right ward, connecting to the leftward pulmonary artery via the ligamentum arteriosum.

Haverly (2003) stated that vascular rings anomaly is the result of developmental anomalies of which there are six anatomical variations that will result in an esophageal stricture. It is common in medium to large breed dogs with much higher incidences in German Shepherd Dogs, Irish Setters, Great Danes and Botstein Terriers. He also noted that, diagnosis of vascular ring anomalies is based on history, physical examination, clinical signs, radiographic signs, esophagogram and esophagoscopy.

Kealy and McAllister (2005) reported that congenital anomalies of the vascular system within the thorax may result in vessel or vessel remnant forming bands that constrict the esophagus near the base of the heart. Persistent right aortic arch is the most common anomalies encountered, although double aortic arch and aberrant subclavian vessels are occasionally seen.

Saini *et al* (2009) reported a case of persistent right aortic arch and patent ductus arteriosus in a three months old Labrador pup with history of regurgitation after taking feeding. Regurgitation after weaning especially solid food and no response to medical treatment are generally diagnostic.

2.1.1.6 Esophageal Diverticulum

Sherding *et al* (1999) stated that esophageal diverticula are large circumscribed sacculation of esophageal wall that interfere with the orderly

movement of ingesta through the esophagus. The wall sacculation are relatively rare and mostly occur in the cranial and epiphrenic regions. Congenital diverticula are caused by abnormalities of embryonic development that result in herniation of mucosa through a defect in to muscularis layer of esophageal wall, while acquired diverticula occurs due to external traction and distortion of oesophagus caused by peri-oesophageal adhesions (traction diverticula) or from increased intraluminal pressure. Food impaction caused by hypo motility (VRA, foreign body, tumour etc.,) leads to pulsative diverticula.

Gualtieri (2001) and Fossum and Hedlund (2002) reported that esophageal diverticula are sac like dilations of esophageal wall that can interfere with normal function of oesophagus. They are rare in small animals and are found most commonly in proximal thoracic oesophagus and distal thoracic oesophagus. Diverticula may be classified as true (when all wall layers are involved) and false diverticula (when only mucosa is involved). They also studied the pathophysiology of formation of esophageal diverticula. The congenital diverticula results from defective embryologic development of oesophagus whereas acquired diverticula due to traction or pulsion type based on their cause. Traction diverticula occur in the cranial and mid thoracic oesophagus due to secondary inflammatory process involving perioesophageal structure. The fibrous tissue that forms between the oesophagus and adjacent structures distort the esophageal wall to form a pouch. Pulsion diverticula results from increased intraluminal pressure and accumulation of food secondary to esophageal hypomotility or obstruction.

Kealy and McAllister (2005) stated that esophageal diverticula are not common. A traction diverticulum results from adhesions and contraction associated with a perioesophageal lesion. A pulsion diverticulum results from increased

intraluminal pressure such as may arise with long standing foreign body obstruction or esophageal stenosis.

2.1.1.7 Esophageal Erosion/Ulcer

Paddleford (1999) stated that the adverse effects from toxicity of NSAIDs included ulcers of gastrointestinal tract, renal dysfunction and haemorrhages caused by platelet function inhibition. He also quoted that, dogs and cats seem more susceptible than humans to these side effects and not all NSAIDs are equal in their ability to produce toxic side effects.

Tams (1999) reported that esophageal foreign bodies are common cause of esophagitis. The extent of secondary esophageal mucosal injury depends upon the type of objects with sharp edges and points such as bones which may lacerate the mucosa and get embedded in it, where as, tightly wedged objects can cause local pressure necrosis/ulcers of the mucosa.

Gualtieri (2001) stated that major benefits from the advent of endoscopy are corriprehnding the esophageal pathology by direct visualization. Esophagoscopy is a highly reliable diagnostic method for evaluating esophageal disorders such as esophagitis, reflux esophagitis, stricture, ulcers, erosion, fistulas, neoplasia and foreign bodies that affect the mucosa or alter the lumen of organ. He reported that oesophageal ulceration is common in dogs due to severe inflammation, reflux esophagitis, torn and irregular sized foreign bodies or neoplastic growth. He also reported that esophagoscopy is the best way to establish a definitive diagnosis of esophagitis and oesophageal ulcers which are characterised by mucosal hyperaemia arranged in linear streaks on the top of mucosal folds or round localised hyperaemic

areas. Increased mucosal friability and easily bleeding mucosa with erosion or ulcer may be seen in chronic form of esophagitis.

Kovacs *et al* (2001) studied the esophageal anastomosis and its healing in thirty mongrel dogs via endoscopy (4^{th}, 7^{th}, 14^{th} and 21^{st}). They also looked for bleeding, ulceration, erosion, haematoma and granulation of the tissue in anastomosis site. They concluded that overall sensitivity of esophagoscopy to discover mucosal lesions were 73% without any complications. Therefore it is a useful method of follow-up and may help to predict the normal or compromised healing of esophageal anastomosis.

Fossum and Hedlund (2005) stated that enlargement of the stomach associated with rotation on its mesenteric axis is referred to as GD-V. The terms simple dilatation refers to a stomach that is engorged with air or froth but not malpositioned. Dilatation refers to a condition in which an organ or structure is starched is beyond its normal dimensions. They also stated that, clinically GD-V is an acute condition with gastric enlargement due to aerophogia, although bacterial fermentation of carbohydrates, diffusion from blood stream and metabolic reaction may contribute. They also noted that, GDV is primarily occurs in large deep chested breeds like Great Dane, weimaraner, saint Bernard, German shepherd, etc. GD-V may occur in a dog of any age, but it most common in middle aged or older animals.

Jergens (2005) reported that, esophageal erosion or ulcer may be due to chronic inflammation caused by foreign bodies, chemical injuries, gastroesophageal reflux and persisting vomiting.

2.1.2 Diseases of Stomach in Dogs

2.1.2.1 Gastritis

Happe *et al* (1982) reported that examination of dogs with duodeno-gastric reflux endoscopically showed bile stained gastric material; thick red rugal folds; petechial in antral mucosa in three of the five dogs with signs of gastritis.

Warren (1983) reported that gastric microbiology has been sadly neglected and half of the patients coming to gastroscopy and biopsy show bacterial colonisation of their stomach, a colonisation remarkable for the constancy of both bacteria involved and associated histological changes in human beings.

Vander Gaag (1988) performed endoscopic gastric biopsies of 501 dogs consisting, of 19 clinically healthy dogs and 482 vomiting dogs. Slight to severe gastritis was found in 168 vomiting dogs whereas in five dogs of clinically healthy group, a sign of mild gastritis was observed. Superficial and diffuse gastritis were the most prominent findings in the dogs with gastritis.

Jergens *et al* (1992) stated that inflammatory bowel disease (IBD) refers to a group of idiopathic, chronic gastrointestinal disorders, characterised by infiltration of the gastrointestinal tract by inflammatory cells. These inflammatory cellular infiltrates with a population of lymphocytes, plasma cells, eosinophils and neutrophils generally confined to mucosa. They also stated that, the mean age of 58 dogs affected with IBD was 6.3 years; with most of them were males (32 dogs) and remaining were females (26 dogs). Most commonly affected breed of dogs in his study were cocker spaniels, German shepherd and mixed breeds.

Hall and Washabau (1999) reported that chronic hypertrophic gastritis may be associated with diffuse or focal macroscopic thickening of the gastric mucosa, leading to progressive obstruction to gastric outflow.

Strauss-Ayali and Simpson (1999) studied gastric mucosal pathology by light microscopy and revealed that infection with large helicobacter like organisms (HLOs) was accompanied by mild gastritis and infiltration of lymphocytes and plasma cells in some but not in all infected dogs. Interestingly, experimentally infection of gnotobiotic dogs with *H. felis and H. pylori* resulted in chronic gastritis in all dogs after four weeks of infection.

Endoscopic appearance in dogs with gastritis is characterised by marked mucosal irregularity and friability in patients with severe gastritis. Mucosal folds appeared oedematous and bled easily. *Helicobacter* organisms have long been known to be present in the stomach of animals, and it is speculated that these organism are normal inhabitants. However, in some animals, it is likely that helicobacter infection can cause chronic gastritis and in rare cases gastric ulceration. *Helicobacter felis* and *Helicobacter heilmanii* appears to be the most common species affecting dogs and cats (Tams, 1999; Zoran, 2001; Jones *et al* 2002 and Shabestari *et al* 2008.

Ettinger and Feldman (2005), Guilford (2005) and Akhtardanesh *et al* (2006) stated that chronic non-specific gastritis was commonly seen in dogs.

Valentine (2007) reported that fluid aspiration is most useful when evaluating the gastric or duodenal samples for presence of organism such as *Helicobacter, Giardia* and *Ollulanus*, or when submitting samples for bacterial culture to identify bacterial disorders of the gastrointestinal tract.

Day *et al* (2008) and Dutta *et al* (2008) stated that the characterization of inflammatory changes in endoscopic biopsy samples of the gastrointestinal mucosa is

an increasingly important component in the diagnosis and management of canine and feline gastrointestinal disease.

Leib and Duncan (2009) reported that spiral bacteria were common in the stomach of clinically normal dogs and cats, as well as those with signs of gastrointestinal disease. They also found that, experimental infection in both dogs and cats resulted in lymphoid follicular gastritis; however, clinical signs were absent or very mild. Currently the direct causal relationship between spiral bacteria and chronic gastritis and vomiting has not been firmly established in dogs and cats.

2.1.2.2 Gastric Erosion /Ulcer

Miwa *et al* (1981); Sorjonen *et al* (1983); Meddings *et al* (1995) and Boston *et al* (2003) studied experimental gastric ulcers due to NSAIDs in canine stomach by endoscopic evaluation, Roth *et al* (1990) observed that ulcers or erosions were characteristic of inflammatory and neoplastic lesions. Luminal masses often had ulcerated or eroded surface.

While Tams (1999) , Ramprabhu *et al* (2001) and Borer *et al* (2003) stated that NSAIDs caused gastric ulceration and haemorrhage, Rohrer *et al* (1999) noticed gastric haemorrhages in dogs due to administration of methylprednisolone succinate (MPSS) by intravenous route@ 30mg/kg body weight.

Fossum and Hedlund (2007) and Luna *et al* (2007) reported gastric ulceration/erosion (GUE) in small animals often is iatrogenic (NSAIDs) or occurs secondary to an underlying diseases process (mast cell disease, shock, tumour, hepatic diseases) characterised by vomiting with undigested, fresh blood or blood clots. Digested blood looks like “coffee ground.”

Neiger (2009) reported that ulcers in the stomach form when damage from acid and pepsin outweighs the ability of the mucosa to protect itself and replace damaged cells. Superficial injury of the mucosa results in haemorrhages and erosion; ulcers formation is only seen when muscular layer is affected. He also reported the prevalence of gastric ulcers in dogs and cats is unknown; however, pets are regularly presented with hematemesis and in these cases breach of the gastric mucosa is likely.

2.1.2.3 Gastric Foreign Bodies

Michels *et al* (1995) reported eight of 36 animals with stomach fish hooks with various types of fishhook include single barb and treble barb. Percentage of treble type of fishhook (15/19) was significantly greater than the percentage with single-barb hook (4/19). Fish hooks were found to be embedded in the esophageal or gastric wall in 26 of the 62 dogs in which endoscopic removal were attempted. The percentage of embedded fishhooks that were successfully removed (11/26; 42%) was significantly less than the percentage of non-embedded fishhooks that were successfully removed (30/36; 83%).

Fossum and Hedlund (1999) reported that a gastric foreign body causes vomiting as a result of outflow obstruction, gastric distention and or mucosal irritation. Dogs are indiscriminate eaters and often ingest plastic toys, cooking bags and other objects. They also noted that noxious stimuli or distention of the duodenum or pyloric antrum stimulates vomiting, where similar stimulation of the gastric body often does not. Therefore vomiting often is intermittent and occurring when the objects are traced into the pyloric antrum.

Kumar and Ameerjan (2001) found gastric foreign such as tile pieces, black stones, chicken bones, sewing needle and mango turnips in seven dogs.

Den (2003) stated that foreign bodies in the airway and gastro intestinal tract in dogs and cat are common problems. Improved endoscopic techniques together with their greater availability mean that it is often possible to remove the foreign bodies endoscopically, without surgery.

Kealy and McAllister (2005) stated that gastric foreign bodies may obstruct the pylorus, provoking persistent vomiting. Intermittent obstruction of the pylorus by foreign bodies may provoke occasional vomiting. They also stated that, a foreign body might be present despite the absence of any clinical signs.

2.1.2.4 Gastric Dilatation and Volvulus (GD-V)

Fossum and Hedlund (1999) stated that clinically GDV syndrome is an acute condition with a mortality rate of 20% to 45% in treated animals. The gastric enlargement is thought to be associated with a functional or mechanical gastric outflow obstruction. The initiating cause of the outflow obstruction is gas or fluid or both that accumulates in the lumen.

Eggertsdottir *et al* (2001) stated that acute gastric dilatation with or without volvulus is an acute, life threatening condition, which predominantly occurs among the large, deep chested dogs. Presurgical treatment for shock and before gastric decompression is vital to a successful outcome. Surgical intervention is necessary for correction of visceral displacement and to prevent recurrence of GDV.

Mahaffey and Barber (2002) stated that acute gastric dilation and gastric volvulus produce gaseous distension of the stomach due to a complex variety of causes, secondary due to aerophagia. Gastric volvulus is differentiated with acute gaseous distension of stomach by the presence of stomach rotation depending on the type and degree of rotation and amount of distension. They also stated that, as the stomach dilates, the greater curvature rotates to lie along the ventral abdominal wall.

Kealy and McAllister (2005) stated that dilatation of the stomach may result from an obstruction of the outflow tract or from atony of the stomach wall. Acute gastric dilatation may occur with or without torsion. Chronic dilatation also occurs as a result of prolonged low grade of pyloric obstruction. They further stated that, swallowed air, often associated with esophageal abnormalities, may cause mild degree of gastric distention.

Simpson (2005) stated that gastric dilatation is characterised by the dramatic distention of the stomach with air whereas in volvulus the stomach twists about its axis, moving dorsally and left side of the fundus. Both the dilatation and volvulus cause caudal caval obstruction and impairs venous return to the heart. He also stated that GD-V is common in large breeds like Great Dane, Labrador, and Rottweiller.

2.2 SYMPTOMS OF THE DISEASES OF ESOPHAGUS AND STOMACH

2.2.1 Symptoms of Esophageal Diseases

Richard *et al* (1967) studied that in congenital achalasia; the dog failed to eat a normal diet and after each meal, vomited undigested large bolus of food without gaining the body weight.

Crow (1985) reported that animals with esophageal neoplasms usually present with a history of dysphasia and weight loss. In advance stage tumours which obstruct the passage of food ingesta, inability to swallow may be seen. The thoracic esophageal neoplasms cause regurgitation, haemoptysis, persistent swallowing efforts and excessive salivation.

Michels *et al* (1995) reported five dogs with a fishhook in the esophagus. Clinical signs included haemorrhage, odynophagia, cough, poor appetite and regurgitation.

Griffiths *et al* (2000) observed that large breeds of dogs with oropharyngeal foreign bodies were presented with signs of dysphagia, excessive salivation, coughing, halitosis, stertor, epistaxis and oral pain.

Gualtieri (2001) stated that hiatal hernia can cause clinical signs by itself, but of more significance is its role in the development of gastroesophageal reflux and esophagitis. Factors responsible for esophagitis are many including composition of reflux material, volume and frequency of reflux, contact time of reflux with mucosa, integrity of esophageal mucosal barrier and efficacy of esophageal clearance. Clinical signs of esophagitis include anorexia, regurgitation, dysphagia, odynophagia and excessive salivation. Signs of depression, fever, dyspnoea and cough suggest perforation of esophageal wall. He reported that clinical signs associated with esophageal tumour can be insidious in onset and slowly progress and characterised by regurgitation of solid food shortly after intake, excessive salivation, anorexia, dysphagia, hematemesis and loss of body weight.

Fossum and Hedlund (2002) and Katerina *et al* (2002), Shaheen and Ransohoff (2002) stated that in esophageal obstruction, acute onset of dysphasia and regurgitation is the most common initial signs. Others include gagging, retching, salivation, inappetance, restless, depression, dehydration, and respiratory distress. Clinical signs vary somewhat depending upon duration, location and type of obstruction. Weight loss and emaciation sometimes are seen in patients with long term esophageal obstruction. The latter further stated that the dogs with these symptoms should undergo endoscopy.

Mylonakis *et al* (2004) studied 39 clinical cases of *Spirocerca lupi* infection. Most of the common complaints included regurgitation (27 dogs,69%), odynophagia (23 dog,59%), excessive salivation (23dog,33%), anorexia or poor appetite(12 dog,31%), progressive weight loss (5 dogs,13%), coughing (3 dogs,8%), weakness(3 dogs,8%) and vomiting (1 dog,3%).

Koc *et al* (2004) reported two months and 10 days old female German shepherd pup weighing 4 kg with history of regurgitation of 20 day duration before the time of weaning to solid food. The dog had no serious medical problem and its vaccination status was good. However, over the previous 20 days, the dog had persistent regurgitation soon after feeding and retarded growth compared with its littermate and also showed postprandial swelling in the thoracic inlet on palpation. Post mortem examination established a vascular ring anomaly.

Guilford (2005) stated that esophageal strictures appear as circumferential narrowing of the esophageal lumen. There may be an associated esophagitis cranial to the stricture, presumably secondary to the fermentation of food, or caudal to the stricture suggestive of gastroesophageal reflux. The lumen of strictures oesophagus is often narrows (1-2mm) and length usually ranges from 1-6 cm. If the lumen is sufficiently wide, radial fibrotic striations are often apparent in the stricture wall.

Kealy and McAllister (2005) stated that vascular ring anomalies in dogs characterised by regurgitation following feeding and failure of to thrive the common presenting signs. Occasionally the dilated esophagus causes a bulge in the lower cervical area. The bulge is more noticeable after feeding. They also noted that, appetite is maintained in case of PRAA anomalies. There may be coughing and dyspnoea due to aspiration pneumonia.

Meineri *et al* (2008) reported that a typical sign of gastroesophageal reflux disease (GERD) includes abundant salivation, regurgitation, and vomiting after meal with or without accompanying respiratory symptoms. They also pointed out food stuffs traditionally considered to causes GERD are chocolate, cheese, tomato sauce, pizzas, mints, coke and spirits and coffee.

Glazer and Walters (2008) studied the timing of events with regard to eating may be helpful in distinguishing the regurgitation and vomiting because regurgitation most often occurs shortly after ingestion. However, animals with distal esophageal diseases or esophageal hypomotility may not regurgitate until hours after eating. Animals with esophageal obstruction or strictures may tolerate liquids better than solids, whereas animals with motility disorders are likely to have problems with liquids as well as solids and loss of body weight was common to both disorders.

Mylonakis *et al* (2008) observed that *Spirocerca lupi* infection is frequently sub clinical and characterized by regurgitation, repeated attempts to swallow (odynophagia), hypersalivation, vomiting, melena, weakness, dyspnoea, decreased appetite, cough, fever, salivary gland enlargement, swollen distal limbs and lameness.

Johnson *et al* (2009) reported that regurgitation is the most common clinical sign observed with megaesophagus. Regurgitation is characterised as a passive evacuation of fluid, mucus, and undigested food from oesophagus. Other signs observed with megaesophagus include ptyalism, halitosis, and vomiting and nasal discharge and dyspnoea caused by aspiration pneumonia.

Saini *et al* (2009) reported a case of persistent right aortic arch and patent ductus arteriosus in a Labrador pup. The symptoms were regurgitation of solid food, noticed immediately after weaning.

2.2.2 Symptoms of Stomach Diseases

Crow (1985) stated that the clinical signs of gastric cancer include vomiting, anorexia, weight loss, abdominal pain and occasionally hematemesis. Frank blood or coffee coloured/ ground material in the vomitus indicated upper digestive tract bleeding.

Roth *et al* (1990) studied the endoscopic observation of gastrointestinal tract in 58 dogs and 17 cats which included normal mucosa, alteration in mucosal colour and texture and luminal mass. It was concluded that endoscopy alone is a useful technique for detecting the alterations of the gastrointestinal mucosa.

Dennis *et al* (1992) studied the lymphocytic or plasmocytic gastroenteritis in 14 cats with vomiting, weight loss and diarrhoeal as common clinical observations in 10(71%) out of 14 cats. Vomiting which was recurrent in six cats since three weeks contained food, bile or mucus. Emaciation was the most consistent finding noted.

Kapatkin *et al* (1992) studied thirteen cases of gastric leiomyosarcoma in dogs characterised by vomiting, lethargy (5 dogs), anorexia and diarrhoea (4 dogs), weight loss and distended abdomen (1dog).

Michels *et al* (1995) reported that eight out of 36 animals with fishhooks in the stomach showed clinical symptoms such as gagging (2), haemorrhages (1), ptyalism (1), tenesmus (1), anorexia (1), vomiting (1), lethargy (3) and melena (1).

Baez *et al* (1999) stated that the clinical signs commonly associated with IBD are vomiting, diarrhoea, anorexia and weight loss, but these may vary depending upon site of inflammation. The most common clinical signs of IBD were vomiting (24/33), weight loss (18/33), diarrhoea (16/33), partial to complete loss of appetite (6/33) and lethargy (3/33). The duration of clinical signs ranged from 1 week to 5 years (mean, 12.5 months).

Gualtieri *et al* (1999) reported that clinical signs associated with gastric malignancies chronic vomiting, weight loss and anorexia. Vomiting is usually persistent, progressive and often not related to food intake may consist of mucus and swallowed saliva. In case of bleeding gastric lesions, vomiting of partially digested blood with classic "coffee grounds" appearance was noted.

Lamb (1999) reported that abnormal gastric emptying is an important cause of vomiting in dogs and cats that can occur as a result of variety of conditions like foreign bodies, mural masses, ulcers, neoplasms and hypertrophic pyloric gastropathy which bring morphological changes.

Gualtieri (2001) observed that the presence of large amount of bile reflux may suggest disorders of motility of the stomach, pylorus or duodenum or an intestinal obstruction.

Ramprabhu *et al* (2001) observed that the effect of non-steroidal anti-inflammatory drugs (Ibuprofen@15mg/kg, orally) on gastric mucosa were severe and caused vomiting, melena, varying degrees of lethargy, paler colour of mucous membrane and hematemesis. One dog in group III was noted with perforated ulcers in the pyloric region and same animal was scarified on 8^{th} day and lesions were confirmed at necropsy examination. Hence they concluded that dogs are more sensitive than human beings to the adverse effects of ibuprofen and therefore should be used judiciously.

Chiang and Chou (2005) reported the case of gastrointestinal foreign body in a one year old cat which showed sings of anorexia, constipation, loss of body weight and appeared normal on physical examination.

Simpson (2005) stated chronic gastritis was previously defined as chronic inflammatory changes occurring within the gastric mucosa coexisting with clinical

symptoms such as anorexia, weight loss and chronic vomiting, but recent studies suggested that chronic gastritis is very common in asymptomatic dogs and cats. The major clinical signs of chronic gastritis are vomiting of food or bile. He also stated that decreased appetite, weight loss, melena or hematemesis are variably encountered in chronic gastritis.

Raekallio *et al* (2006) stated that all NSAIDs have the potential for causing gastrointestinal, renal and hepatic problems; carprofen is generally considered to be well tolerated by dogs. In several studies, the drug was not found to induce substantial lesions in the gastric mucosa of dogs and when administration of carprofen is combined with general anaesthesia in healthy dogs. They also reported that the dogs treated with carprofen, few of them vomited or had diarrhoea during the fallow up period. Four carprofen treated and three placebo treated dogs had signs (vomiting, diarrhoea and development of skin reaction) that had not been detected by the owner during the month before initiation of treatment.

Harvey (2008) studied one case of pyloric stenosis in female boxer dog with history of episodes of regurgitation beginning at the time of weaning and continued at the age of 12 weeks. He also noted that, the regurgitation involves the passive ejection of material that usually includes undigested food that is in tubular shape and devoid of bile.

2.3 PATHOPHYSIOLOGY OF DISEASES OF ESOPHAGUS AND STOMACH

Richard *et al* (1967) stated that in achalasia of the esophagus, the body of the esophagus lacks peristalsis and is usually appear dilated while lower esophageal sphincter fails to respond normally to swallowing. They also stated that, achalasia is described as a failure of the sphincter to relax and contract in a coordinated manner

which is related to loss of the enteric neurons or ganglion cells in Auerbach's myenteric plexus. The etiology of the loss of enteric neurons is unknown. The absence or reduction in number of these nerve cells is a common finding in this disease and occurs mainly in the body of the esophagus but may affect the lower sphincter and the body in varying proportions.

Willard (1984) studied the mechanism of vomiting and explained that medullary vomiting centre is the final common pathway from which efferent impulses arise to cause contraction of abdomen and diaphragmatic muscles. This contraction, coupled with gastric atony, causes forceful expulsion of gastric content. Input from CRTZ, the vestibular centre, viscera, higher centres (cerebrum), or direct stimulation from intracranial processes is necessary for the vomiting centre to initiate this reflex.

Hamir (1986) studied the life cycle of Spirocerci lupi. He explained that adult worms were found in the terminal part of esophagus where they deposit embryonated eggs which are then voided through faeces. The eggs are then ingested by various species of caprophagic beetles in which they hatch and develop infected stage of larvae. In the final host, larvae migrate through stomach wall and along the adventitia and facia of regional arteries to ascend the aorta and then migrate to the oesophagus.

Michels *et al* (1995) examined 88 animals during the study period for ingestion of fish hook. However, 10 of these were excluded from the study because the fishhook located in the caudal part of pharynx (3 dogs), the intestine (5 dogs), or the perioesophageal tissues (1 dog and 1 cat). Of the remaining 78 animals 38 had a fishhook in the esophagus, 39 had a fishhook in stomach. Thirteen of 72 (18%) dogs included in the study were miniature or toy poodles, and poodles were overrepresented, compared with hospital population during the study period. The remaining 59 dogs were of a variety of breeds.

Tams (1996) stated that during pregnancy in human beings, the likelihood of gastroesophageal reflux is increased as a result of the elevated progesterone level, which decrease lower esophageal sphincter pressure and not by the increased gastric pressure from an enlarged uterus. Besides, gastroesophageal reflux during anaesthesia, other causes of esophageal stricture are chemical burn, foreign body trauma, persistent vomiting and hiatal hernia.

Boothe (1999) studied the pathophysiology of emesis which is controlled through the emetic centre located in the lateral reticular formation of the medulla; as such, in this site, the centre is protected by the blood –brain barrier. Although several afferent pathways may be responsible for initiating the emesis, all signals are coordinated by the emetic centre. Impulses to the emetic centre in the medulla may arise from higher centre such as cerebral cortex and limbic system. Psychogenic vomiting and that arising from visual and olfactory stimuli originate in the cerebral cortex. Acetylcholine is the primary afferent neurotransmitter that mediates emesis from the higher centre, although histamine acts as a secondary transmitter via H_1 receptors. Blood borne chemical compounds may stimulate the chemoreceptor trigger zone (CTZ), which is located in the area postrema in the lateral wall of third ventricles.

Paddleford (1999) reported that NSAIDs mediated analgesia has been thought to be entirely the result of the anti-inflammatory effect of these drugs at the site of tissue injury. However, evidence now suggests, these drugs may also have significant central analgesic effects. The central effects may be independent of the inhibitors of prostaglandins formation. Also he stated that NSAIDs inhibitors of cyclooxygenase, which is an enzyme necessary to convert arachidonic acid into prostanoids such as prostaglandins, thromboxane and prostacyclines.

Griffiths *et al* (2000) reported that a penetrating foreign body can drive a bacterial inoculum deep into the soft tissues, which may subsequently form an abscess, even if the foreign material is removed. Alternatively, the foreign body may be retained within the sublingual, intermandibular, retrobulbar, pharyngeal, retropharyngeal and cervical soft tissues.

Eggertsdottir *et al* (2001) concluded that there was no significant difference in the treatment of GDV between two groups. At the end of study, the difference rate was 9% and 20% in group A and in group B, respectively. Hence both the techniques (circumcostal gastropexy and gastrocolonopexy) are effective in preventing recurrence of GDV.

Jones *et al* (2002) reported that the therapeutic action and toxicity of NSAIDs are primarily thought to be the result of an inhibition of cyclooxygenase (COX) and subsequent prostaglandin formation. The discovery of two separate isoforms of COX has revolutionized NSAIDs development. Cyclooxygenase -2, the inducible isoforms, is believed to be primarily responsible for inflammatory activity of prostaglandins. Cyclooxygenase -1, the consecutive isoforms, is believed to be responsible for basal physiological functions, or overall maintenance, provided by prostaglandins. These actions include gastric mucosal protection, renal function and platelet activity.

Boston *et al* (2003) studied the mechanism of NSAIDs in clinically healthy dogs; the major mechanism of action of NSAIDs is to prevent the production of PGs from arachidonic acid by inhibition of cyclooxygenase enzymes (COX). Along with the desired effects like analgesic, anti-inflammatory and antipyretic, the systemic inhibition of PGE2 production inhibits the normal protective mechanism of the gastric mucosa. In the absence of protective mechanism, the acidic environment of the stomach damages the mucosa and leading to gastric ulceration. He also pointed out

the COX-1, is responsible for the protective effect on the gastric mucosa, renal function and renal blood flow whereas, COX-2, is responsible for the production of PGs that cause pain and inflammation.

Gualtieri and Olivero (2006) studied the factors affecting the occurrence of reflux esophagitis include the nature of the refluxate, esophageal clearance, volume and frequency of the refluxate, duration of refluxate in oesophagus and esophageal mucosal integrity. The resulting lesions may be limited to the mucosa in mild cases or may involve the muscular layer and lead to stricture, ulceration and perforation of the oesophagus.

Fossum and Hedlund (2007) studied the mechanism of ulcer formation secondary to NSAIDs which is probably multifactorial but inhibition of prostaglandin synthesis is important. Prostaglandin exerts a protective effect on the mucosal barrier by stimulating mucus and bicarbonate production. They also noted that gastric adenocarcinoma and lymphoma are probably the most gastric tumours, but leiomyomas are especially prone to ulcerate and bleed.

Luna *et al* (2007) reported that NSAIDs is the world health organizations (WHO) first drug of choice for treating the mild to moderate chronic pain. The NSAIDs reduce inflammation by inhibiting the action of cyclooxygenase, which convert arachidonic acid into prostanoids (PGG2, H2 and prostacyclines). Prostaglandins H2 is converted into various eicosanoids (PGD2, PGE2, PGF2α and thromboxaneA2), which are inflammation mediators and amplify nociceptive input and transmission to the spinal cord.

Chen *et al* (2008) stated that esophageal food impaction is the most common type of foreign body ingestion in adults. Patients presenting with food bolus impaction commonly have underlying esophageal abnormalities like peptic stricture,

esophageal motility disorders such as achalasia, diffuse esophageal spasm and nutcracker oesophagus.

Mylonakis *et al* (2008) studied that pathogenesis of *Spirocerca lupi* and elucidated proposed mechanisms of Spirocercosis that include the malignant transformation of fibroblasts within the grannulomatous reaction, the inciting role of an oncogenic stimulus originating from the parasite itself and even environmental factors. Granulomas and sarcomas may result in esophageal dysphagia due to mechanical obstruction and possibly functional impairment. Later esophageal dysphagia worsened by concomitant salivary gland necrosis associated with *S.lupi* infection. Respiratory distress and coughing have been attributed to the space occupying nature of voluminous grannulomatous or neoplastic masses ,pulmonary metastasis of the latter, dysphagia induced aspiration bronchopneumonia and occasionally esophageal perforation leads to pyothorax. Ulceration of Granulomas or sarcomas may lead to malena without hematemesis and chronic haemorrhagic anaemia.

Johnson *et al* (2009) stated that the pathogenesis of congenital megaesophagus is unclear, and esophageal function studies of affected dogs indicate that defects in the vagal afferent innervations of the oesophagus and decreased esophageal motor function, possibly secondary to abnormal biochemical properties of the esophageal muscle. They also reported that, myasthenia gravis (MG) is the most common cause of acquired megaesophagus in the dog due to an autoimmune disorder that interfere with normal neuromuscular transmission. Productions of autoantibodies against nicotinic Ach receptors decrease number of receptors available for normal neuromuscular transmission, resulting in skeleton muscle weakness.

Neiger (2009) reported that NSAIDs are thought to cause gastrointestinal mucosal injury by several mechanisms. Primarily these drugs impair prostaglandin dependent mucosal protective mechanism by non-selective inhibition of the two isomers of cyclooxygenase (COX), COX-1 and COX-2. He also noted that, COX-1 inhibition leads to reduced bicarbonate secretion, reduced mucus formation and vascular action, mitochondrial injury by a direct toxin action. In his largest study of gastric ulcers in dogs only 4 of 43 cases were thought to have peptic ulcers primarily caused by NSAIDs.

Willard and Carsten (2009) stated that when the esophagus becomes inflamed, motility can be impaired, allowing food to be retained and ultimately regurgitated. More important, poor esophageal motility allows gastric acid refluxed into the esophagus to remain longer, worsening the esophagitis. They also noted that, LES dysfunction occur due to esophagitis allowed more GER. Chronic esophagitis caused by GER can be associated with severe histologic changes somewhat comparable to barrette's oesophagus in human.

2.4 DIAGNOSIS OF DISEASES OF OESOPHAGUS AND STOMACH

2.4.1 Haematological Examination

Dennis *et al* (1992); Jergens *et al* (1992) and Baez *et al* (1999) studied the haematological changes in 14 cats suffering with plasmacytic or lymphocytic gastroenteritis and revealed non-regenerative anaemia, leucocytosis, and high numbers of segmented and non-segmented neutrophils. Additionally neutrophillia, eosinophilia, lymphopenia and basophilia may be observed.

Luna *et al* (2007) reported that there were no changes in the values of haemoglobin, RBC, WBC, platelet count and differential leucocyte count in dogs with drug induced gastritis.

2.4.2 Serum Biochemical Examination

Hoenig *et al* (1990) studied the biochemical changes in two cats with megaesophagus and revealed leucopoenia, high concentration of BUN (42mg/dl), high glucose (297mg/dl) and low concentration of chloride (99 mEq/L).

Dennis *et al* (1992) reported that cats suffering with plasmacytic or lymphocytic gastroenteritis showed hypoproteinemia, low serum globulin values, hypokalemia, hypernatremia, hyperchloremia, hyperglycemia, high serum phosphatase and serum alanine transaminase activity.

Baez *et al* (1999) studied the biochemical changes in 33 cats with IBD and revealed hypocholelesterolemia which was one of the most abnormalities on serum biochemical analysis and was detected in10 out of 33 cases of dogs.

Ramprabhu *et al* (2001) stated that dogs treated with Ibuprofen @15 mg/kg orally three times daily showed changes in biochemical profile i.e. lowered plasma protein and elevated blood urea nitrogen on 8^{th}day, post treatment.

Raekallio *et al* (2006) observed the effect of oral administration of carprofen in dogs and revealed mean serum protein and albumin concentrations were significantly lower in dogs treated with carprofen than in dogs that received the placebo at 4th week, but not at 8th week. None of the dogs had serum protein concentration outside reference range. The mean values of serum urea and creatinine concentration, serum ALP and ALT activities; and urinary ALP to creatinine, GGT to creatinine and protein to creatinine ratios did not change and no difference in those variables were observed between the groups.

Luna *et al* (2007) studied the effect of flunixin on gastrointestinal tract and biochemical changes revealed increased serum alanine transferase activity, acute renal

failure and gastric ulcers. Also they reported the ketoprofen induce 30% rate of adverse effects, including gastrointestinal changes, dizziness, headache and nephritis in humans.

2.4.3 Plain Radiographic Examination

Richard *et al* (1967) and Crow (1985) reported that congenital esophageal and esophageal obstruction achalasia appears to be more common in the German shepherd dog. The condition was diagnosed in pups on the basis of radiographic and esophageal motility studies.

Hoenig *et al* (1990) revealed generalised dilatation of esophagus (megaesophagus) and gastric distention with gas on plain radiography in dogs with idiopathic megaesophagus.

Michels *et al* (1995); Fossum and Hedlund (1999) and Lamb (1999) stated that survey radiography is a useful tool for diagnosis of esophageal and gastric lesions. The most common location of fish hook was the pharyngeal portion of oesophagus (13). Fish hook was also located at or around the base of heart (11), at thoracic inlet (4), and in the distal portion of oesophagus at level of hiatal region (2).

Sherding *et al* (1999) and Michael and Goldstein (2004) stated that normal esophagus is not usually visualized on survey radiographs but megaesophagus can be identified when esophagus becomes distended with gas, fluid, food or foreign material. They also reported that diverticula can be diagnosed on plain radiography characterized by an air filled or food filled mass in the area of the esophagus and contrast enhanced radiograph demonstrate filling of the pouch with barium.

Griffiths *et al* (2000) reported the radiographic abnormalities in 42 dogs having oropharyngeal injuries which included presence of gas, either subcutaneously

or between facial planes, soft tissue swelling, pneumomediastenium, metallic foreign bodies and vertebral periosteal proliferative reaction.

Kumar and Ameerjan (2001); Gualtieri (2001) and Chiang and Chou (2005) reported gastric foreign bodies in seven dogs and they were diagnosed by plain radiography. They revealed abnormal opaque shadows on plain radiograph indicated the location of the foreign bodies.

Fossum and Hedlund (2002); Meyer and Levine (2008) and Ponnuswamy *et al* (2009) stated that most of the foreign bodies (99%) can be seen as a density on good quality radiographs provided that they are radiopaque, but it is not always obvious that they are in oesophagus. The foreign body usually found at or cranial to base of heart and diaphragm. In addition to the foreign body, there may be an adjacent soft tissue opacity, and dilated, air filled cranial oesophagus may be seen. They also studied radiographic appearance of the esophageal diverticula includes pear shape, air filled or food filled masses in the area of the esophagus.

Mahaffey and Barber (2002) observed the radiographic feature of gastric Volvulus is gas and fluid dissention of the stomach. Additionally, the pylorus is usually displaced dorsally and to the left on left lateral abdominal radiographic view. Lateral views are usually of most valuable because stomach filled with gas, the pylorus portion of the stomach appears more tabular and narrower than the rest of stomach. Thus, the radiographic findings that, the pyloric portion fills with fluid on the left lateral recumbent view whereas pylorus fills with gas on right recumbent lateral view indicates that the pylorus is on the left side and that the stomach ahs rotated.

Watrous (2002) and Guilford (2005) stated that segmental diseases of esophagus is identified on survey radiograph by focal air accumulation. Abnormal

regional air accumulation may occur anywhere along the esophagus just cranial to or at the site of localized diseases like intramural foreign bodies, esophagitis and segmental esophageal hypomotility. They also noted that, obstruction of the esophageal lumen by vascular ring anomalies, acquired strictures, extramural and intrinsic masses and chronic foreign bodies may result in air accumulation. They also stated that the ventral wall projects lateral and often ventral to the trachea. The draping of the ventral wall of the esophagus over the dorsal wall of the trachea results in summation (silhouetting) of the two walls which creates tracheal band or "tracheoesophageal stripe signs" indicated generalized megaesophagus.

Kealy and McAllister (2005) explained the radiographic signs of megaesophagus included radiographs of thorax show the esophagus to be dilated with air, fluid or food material with a mixture of these. They also observed that, when the esophagus is dilated with air, it drapes itself over the dorsal trachea, giving a "tracheal stripe sign".

Fossum and Hedlund (2007) stated that radiographs are necessary to differentiae simple dilatation from dilatation plus Volvulus. On a right lateral view of dog the GD-V the pylorus lies cranial to the body of the stomach and is separated from the rest of the stomach by soft tissue band i.e. reverse C sign or double gas bubble appearance.

Rousseau *et al* (2007) studied radiographic examination of cervical, thoracic and abdominal radiographs in 43 dogs. Esophageal foreign bodies were radiographically visible in all patients. Thoracic radiographs from dog with mild esophagitis and moderate to severe esophagitis were reviewed for evidence of pulmonary, mediastinal or pleural pathology.

Hall (2008) stated that plain thoracic and abdominal radiographs and abdominal ultrasound examination should always be performed before endoscopy to identify the intraluminal masses or lesions and large foreign bodies beyond the reach of the endoscope. However, the place of contrast radiography has been changed since the advent of endoscopy method; although it should be considered a complementary procedure and not necessarily redundant. Contrast procedure does not require anaesthesia and provide esophageal luminal diameter, function, gastric motility and emptying. He also stated that, endoscope is more sensitive for detection of mucosal diseases and gives a potential, definitive diagnosis through biopsy.

2.4.4 Contrast Radiographic Examination

Crow (1985) Contrast radiography is useful in identifying the diseased portion of esophagus and may give indication regarding the size of lesions. Gastric contrast radiography also permits more thorough evaluation of the stomach for wall thickness, filling defects, abnormal motility and emptying time.

Hoenig *et al* (1990) and Rousseau *et al* (2007) reported that barium sulphate suspension were lodged throughout the lumen of oesophagus and occupied approximately half of thoracic cavity in cases of generalised megaesophagus.

Guilford (1990) reported that upper GI contrast radiography and endoscopy are complimentary procedures and contrast procedure do not require anaesthesia and they provide a better estimation of luminal diameter (esophagus), mural masses, extramural compressive lesions, jejunal diseases, GI motility and gastric emptying.

Baez *et al* (1999) and Fossum and Hedlund (1999) stated that flocculation of barium contrast material, irregular barium and mucosal surface, delayed transit time and persistent adherence of barium to the mucosa were considered abnormal findings.

Gualtieri *et al* (1999); Lamb (1999) and Hall and Washabau (1999) studied the contrast technique to outline the intramural masses, thickening of the gastric wall, loss of derangement of rugal folds, ulcers, filling defects, and delayed gastric emptying. Most gastric ulceration identified radiographically is caused by gastric carcinoma.

Sherding *et al* (1999); Gualtieri (2001); Lavin (2003) and Keles *et al* (2007) suggested that barium contrast radiography is more reliable than endoscopy for the diagnosis of megaesophagus, diverticula, vascular ring anomalies and hiatal disorder of oesophagus. They also reported that the contrast material is accumulating into the dilated oesophagus cranial to the wall delineated narrowing of lumen caused by stricture.

Lobetti (2000); Chiang and Chou (2005) and Mylonakis *et al* (2008) stated that the most diagnostic methods used for diagnosis of esophageal *Spirocerca lupi* were radiology (74%), endoscopy (27%), post mortem examination (34%) and faecal floatation (4%).

Michael and Goldstein (2004); Vlasin *et al* (2004) and Glazer and Walters (2008) suggested that the diagnosis of megaesophagus, independent of the underlying cause, requires imaging studies which demonstrate the anatomic and physiologic abnormalities. They also suggested that, barium swallow may be performed to outline the organ but should be avoided if survey radiographs are convincing as aspiration is a serious concern.

Johnson *et al* (2009) suggested that megaesophagus is diagnosed on radiographic identification of a dilated or hypomotile oesophagus. Survey thoracic radiograph confirm the presence of generalised megaesophagus in most cases and usually reveal dilated esophagus with air, fluid or ingesta. They also stated that, barium liquid or barium contrast studies must be performed to detect subtle hypomotility and to rule out foreign body, stricture or other obstructive lesions.

Saini *et al* (2009) reported a case of persistent right aortic arch and patent ductus arteriosus in a Labrador pup and contrast radiography revealed accumulation of contrast material in the esophageal diverticulum near the thoracic inlet and cranial to the base of heart.

2.4.5 Ultrasonographic Examination

Washababu (1996) stated that ultrasonography does not have a traditional role in the diagnosis of esophageal diseases, but it has proved to e useful in diagnosis of periesophageal mass or other mediastium diseases.

Fossum and Hedlund (1999) reported that some radiolucent gastric foreign bodies can be identified ultrasonographically if the stomach is fully filled and an appropriate acoustic window is obtained.

Chiang and Chou (2005) and Fossum and Hedlund (2007) stated that ultrasonography is not the modality of choice for evaluation of GI-tract. However foreign bodies may occasionally be identified acoustic shadows.

Kealy and McAllister (2005) stated that a 5- 7.5 MHz high resolution transducer is used particularly for evaluating gastric wall details. Animals are usually positioned in ventro-dorsal position with the transducer placed caudal to the costal

arch in sagittal plane. He expressed that right lateral recumbency is useful for pylorus examination while left lateral recumbency for fundus examination.

Kealy and McAllister (2005) stated that the cervical esophagus may be examined from a ventrodorsal or left lateral aspect. A transverse image identifies it to the left of the trachea, which is a hyperechoic region with associated acoustic shadowing. They also noted that, the esophagus is poorly identified, round structure, with concentric rings and a central hyperechoic area representing intraluminal air.

2.4.6 Endoscopic Examination

Johnson *et al* (1976); Crow (1985); Happe (1985); Davies (1986) and Twedt (1993) and Moore (2003) reported that endoscopy in small animal practice is safe and new diagnostic and therapeutic concept and it has clearly wide spread popularity among the specialist. Upper gastro intestinal endoscopy is a non invasive and atraumatic technique that permits visual examination of esophagus and stomach to allow therapeutic interventions as well as collection of biopsy samples for detection of mucosal abnormalities.

Roth *et al* (1990) and Baez *et al* (1999) observed hyperaemia and haemorrhages during endoscopic examination of stomach (n=3) and duodenum (n=5) that did not show appreciable haemorrhages or hyperaemia in histologically examined tissue samples obtained from the same site. They also described that appearance of the mucosa as normal (noninflammed) or as areas of inflammation with hyperaemia, haemorrhages, friable on the basis of the ease at which it was damaged by the endoscope or by biopsy forceps. Other abnormalities, such as erosion (superficial defect of the mucosa), plaques, or ulcers (deep mucosal defect with raised margin) were also recorded.

Dennis *et al* (1992); Ramprabhu *et al* (2001) and Luna *et al* (2007) studied the gastroscopy of gastric extramedullary plasmacytoma in dog that showed, crateriform mass on the greater curvature of stomach, approximately 2.5cm from the pylorus. The mass consist central ulcer surrounded by crater wall 0.5cm high. They also reported that, gastric mucosa had a "cobble stone" appearance and multifocal sites of haemorrhages in 2 of 5 cats with histological evidence of gastric inflammation.

Jergens *et al* (1992) collected the biopsy specimens via endoscopy in dogs and cats with IBD were histologically evaluated for severity of mucosal epithelial damage, mucosal erythema, friability, enhanced granularity and erosion and ulceration were predominant endoscopic lesions among the others.

Washabau (1996) Gualtieri (2001) and Guilford (2005) reported that endoscopy is useful for therapeutic i.e. retrieval of foreign bodies from the esophagus and stomach. They also stated that the esophageal foreign bodies can be pushed into the stomach if failed to retrieve endoscopically.

Sherding *et al* (1999); Gualtieri (2001) and Fossum and Hedlund (2007) studied the endoscopic appearance of severely dilated oesophagus described as markedly dilated, flaccid esophagus extending from the cranial region to the gastroesophageal sphincter and pooling of retained saliva with fermenting food in the lumen. The esophageal mucosa in megaesophagus is usually normal in appearance, but secondary esophagitis is occasionally observed. They also stated that endoscopic examination of the esophageal mucosa is the most sensitive method for diagnosing esophagitis. The endoscopic findings of this inflammatory condition include mucosal erythema, haemorrhage, friability, irregularity.

Tams (1999); Gualtieri (2001) and Fossum and Hedlund (2007) studied the endoscopic appearance of gastric haemorrhages of reddish, discrete petechiae, yellow or bright-red confluent streaks that are not associated with any visible break in the mucosa. The intactness of the mucosa can be ascertained from the appearance of mucosal folds.

Gualtieri (2001) stated that the patient for endoscopy examination of esophagus and stomach should be positioned in left lateral recumbency with an endotracheal tube and mouth gag in place.

Tams (2003) stated that the diagnosis of the esophageal dilatation (megaesophagus) mostly done by contrast radiography hence, the esophagoscopy is not a routine diagnostic tool because general anaesthetics may sluggish the esophageal motility.

Mylonakis *et al* (2004) observed that the parasitic nodules were seen during esophagoscopy in all 39 dogs at distal aspect of thoracic part of oesophagus between 2 to 17 cm diameters (mean 5 cm) from the lower esophageal sphincter. Ulcerative nodules were observed in 4 dogs (10%) and, 3 of the 4 had melena. In 5 (13%) dogs, adult S.lupi worms were seen to protrude from the nodules. However, unless there is a visual evidence of adult worm protrusion into the esophageal lumen or typical nipple like orifice associated with parasitic nodule, differentiating between granulomas, sarcomas and other primary nodules is rather difficult because of their macroscopic similarities.

Guilford (2005) recommended that for routine upper gastrointestinal endoscopy, the patient is positioned in left lateral recumbency which facilitates examination of pylorus by bringing the pylorus to the top f abdomen.

Gualtieri (2001) and Jergens (2005) stated that esophagoscopy is rarely required for diagnosis of megaesophagus except any obstructive lesion in the lumen of the esophagus like esophageal foreign bodies, intramural lesions, etc.

Gualtieri and Olivero (2006); Fossum and Hedlund (2007) and Glazer and Walters (2008) reported that upper gastrointestinal endoscopy in reflux esophagitis revealed a severe, localized, chronic esophagitis on the right ventro-lateral wall of the middle to distal third of the oesophagus. Also they reported that, the lesions were characterized by white irregular, raised mucosa that easily bleed during the passage of endoscope and close to the cardia, a localized proliferation of the esophageal mucosa assumed a nodular form.

Hall (2008) stated endoscopy is indicated in the investigation of suspected esophageal, gastric and small intestinal diseases if the lesion can be reasonably being expected to be accessible from GI lumen. He also stated that surgery is always associated with increased risk to life and an inevitable convalescence period whilst wounds heal. In addition, patient with protein losing enteropathies often have impaired healing and therefore endoscopy provides the safest method of obtaining a definitive diagnosis in most cases. From the clinical advantages of endoscopy to patients are (1) can be discharged on the same day as the procedure, (2) No convalescence or wound healing and (3) immediately treated with steroids if indicated.

Ponnuswamy *et al* (2009) reported gastric erosion in one dog and endoscopic examination revealed erosion in the middle and caudal oesophagus, lower esophageal sphincter and gastric fundus with circumscribed bleeding ulcers at fundus and pylorus region.

2.4.7 Necropsy Examination

Richard *et al* (1967) and Barber *et al* (1983) studied the necropsy findings of megaesophagus and revealed thin and dilated throughout its whole length and filled with water, the water passed readily through the lower sphincter from both directions, thus excluding stricture.

Clifford *et al* (1971) found on necropsy examination of 6 month old kitten with congenital PRAA distention of esophagus containing recently consumed food where the vascular ring had obstructed the esophagus and also revealed large blood clots at the ventral border of the esophagomyotomy incision. Several millilitres of blood were noted on both sides of the thoracic cavity death. Death was attributed to haemothorax, atelectasis and aspiration of vomitus.

Hoenig *et al* (1990) reported one cat with megaesophagus. The post mortem findings included wide and flaccid esophagus approximately 1.5cm in width cranial to the heart and 2.5cm caudal to the heart.

Brunnert *et al* (1992) observed gross structure of gastric extramedullary plasmacytoma during laparotomy operation consisting of a tan infiltrating mass which was protruding from the mucosal and serosal surfaces of greater curvature of stomach. The gastric mucosa of greater curvature was focally ulcerated, thick and haemorrhagic.

Head *et al* (2002) reported that the canine gastric smooth muscle tumours range in size from 0.5 to 24 cm in diameter. They are round or oval with a thin capsule, and bulge out of the surface, and appear pink to white and has a slightly

whorled pattern of fibres, in contrast to the more homogenous granular cut surface of carcinoma.

Keles *et al* (2007) observed necropsy lesions in a calf with acquired megaesophagus, revealed thoracic part of esophagus filled with feed, therefore thoracic and cervical esophagus appeared enlarged. On longitudinal incision of esophagus revealed feedstuff containing rough fibres.

2.4.8 Cytological Examination

Guilford (1990) reported that brush cytology is an adjunct to endoscopic biopsy. It should be performed prior to biopsy so that the cytological specimen is not contaminated with blood from the biopsy site. It may improve the diagnostic yield because superficial material that otherwise may be lost during processing is easily sampled. Thus, organisms such as unidentified bacteria and protozoa that reside in the mucus of the gastrointestinal tract may occasionally be detected by brush cytology but not well seen in biopsies.

Jergens *et al* (1998) and Gualtieri (2001) collected cytological samples from 85 dogs and 23 cats undergoing routine endoscopic examination of the gastrointestinal tract. Cytological smears were prepared by using brush and touch technique. The brush technique was particularly useful for detection of benign inflammation of the lamina propria of stomach, small intestine and colon. When the diagnostic accuracies of the two cytological techniques were compared, use of brush technique yielded specimens of greater diagnostic value in 84% of the organs sampled.

Guilford (2005) reported that brush cytology is a useful adjunct to endoscopic biopsy. Thus organism such as bacteria and protozoa that reside in the mucus of the

gastrointestinal tract may occasionally be detected by brush cytology but not seen on biopsy.

Akhtardanesh *et al* (2007) and Stromar *et al* (2008) examined cytological analysis of helicobacteria species and revealed a GHLO (Gastric Helicobacteria Like Organism) infection rate of antrum, body of stomach as 63.2% and 77.2% of cases, respectively. Only large spiral organisms, morphologically resembling *H.felis* or *H.heilmanni* (but not *H.pylori*) were detected in cytology examination.

Leib and Duncan (2009) and Rogers (2009) stated that gastric cytology is the least expensive and most practical diagnostic method with the quickest turnaround time. A guarded cytology brush is passed through the biopsy channel of the endoscope into the gastric body along the greater curvature. After completion of endoscopic procedure and collection of biopsy sample from stomach and duodenum, gently rubbed across several glass microscope slide, which are air dried, and stained with a rapid Wright stain (Dip Quick stain).

Cytological examination of exfoliative specimens obtained during endoscopy is a useful and reliable adjunct to histological examination of biopsy specimens in the diagnosis of gastrointestinal tract diseases in dogs and cats as suggested by Jergens *et al* (1998); Guilford (1999); Hall and Washabau (1999); Lamb (1999); Gualtieri (2001); Mansell and Willard (2003); Guilford (2005).

2.4.9 Histopathogical Examination

Richard *et al* (1967) studied the histopathological examination of dilated esophagus, which revealed thinned muscle layer, little inter-muscular tissue and no signs of ganglion cells were seen on serial section of esophagus.

Hoenig *et al* (1990) observed the histopathological changes in first cat with megaesophagus which included purulent esophagitis consistent with secondary reaction to static ingesta. The submucosa was infiltrated with neutrophils and plasma cells. The lymphocytes appeared dilated and many contain neutrophils. In second case oesophagus had diffuse lymphocytic plasmocytic esophagitis with focal ulceration and diffusely hyperplastic. The gastric cardia mucosa had lymphocytic plasmocytic gastritis.

Randelia *et al* (1990) reported that in both dog and rat with megaesophagus, histological studies have indicated a normal or decreased numbers of ganglion cells in the myenteric plexus. He also reported that, histochemical or ultra structural studies have not been carried out, thus pathogenesis of this condition in animals has not yet been elucidated.

Brunnert *et al* (1992) observed on histological examination of multiple gastric mucosal biopsy specimens, a heterogeneous infiltrate composed of neutrophils, macrophages, lymphocytes, and extensive necrotic debris with moderate hyperplasia of gastric epithelium. Also transmural, highly cellular, infiltrate of neoplastic round cells are found in cords and nests and separated by a fine reticular stromal network.

Gualtieri *et al* (1999) studied the cytology of gastric fluid and revealed gastric epithelial cells in both normal and abnormal conditions and are seen as cohesive clusters of columnar cells. The nucleus is small and round, with a finely granular chromatin pattern.

Strauss-Ayali and Simpson (1999) suggested that HLOs can be visualized easily using May-Grunwald-Giemsa or Diff Quick stains. HLOs were demonstrated in 100% of positive canine sample when using touch biopsy. Impression smears of gastric biopsies were found to be equally sensitive to modified Steiner stain in experimentally H. Felis infected dogs; hence it is sensitive and easily performed.

Valentine (2007) stated that cytological preparations using endoscopic techniques such as fluid aspiration for cytology or bacterial culture, brush cytological preparation and impression smears made by rolling biopsy samples on a glass slide before fixation in formalin suggested by him that appears to be promising for identification of various inflammatory and neoplastic conditions of gastrointestinal, respiratory and urogenital tract.

Harvey (2008) and Shabestari *et al* (2008) reported that the incidence of chronic gastritis was 40% (n=12) by histopathological examination, but only 13.3% (4 dogs) of these cases showed macroscopic lesions of chronic gastritis via gastroscopy. Gross lesions includes congestion of the mucosa (8 cases; 26.6%) and erosions and ulcers (4 cases; 13.3%). They also noted that in most cases diffuse chronic gastritis with lymphoid follicle formation and connective tissue replacement. They concluded that, superficial and diffuse gastritis, lymphocytic-plasmacytic gastritis were the most prevalent type of chronic gastritis in dogs.

2.4.10 Scanning Electron Microscopy (SEM) Examination of Megaesophagus

Barber *et al* (1983) studied the scan electron microscopy of dilated oesophagus in colt and made measurement of proximal esophageal segment, the thickened esophageal segment and normal esophagus of a foal of similar age. It revealed that thin proximal esophageal segment was less than one half of the

thickness of the normal oesophagus when measured at comparable site. There was a reduction in thickness of the mucosa, inner muscular layer (circular layer) and outer muscular layer (longitudinal layer). The inner muscular layer contained numerous, scattered skeletal muscle bundles that were homogenous, brightly eosinophilic surrounded by a clear space and had loss of normal cross striation.

Frappier (2001) stated that the normal thickness of thoracic esophagus in dog comprises of mucosa (stratified squamous epithelium, lamina propria and muscularis), and submucosa (loose connective tissue, large longitudinal arteries, vein and lymph vessels). The tunica muscularis contains inner circular and outer longitudinal muscle fibres arranged in a bundle form. The specific thickness of each layer is not mentioned in the present literature.

2.4.11. Transmission Electron Microscopy (TEM) Examination of Megaesophagus

Randelia *et al* (1990) examined the ultra structural study of muscle coat of the thoracic and abdominal oesophagus in two mice with megaesophagus and normal esophagus. The normal oesophagus showed striated muscle cells with distinct Z, A, I, H and M bands with mitochondria, glycogen and T-tubules. Each cell was surrounded by a distinct basal lamina. Myenteric plexus were observed in the connective tissue between the two layers of the muscle; well developed motor end plates were not observed. The small nerve endings containing dense-cored granules and some empty vesicles were seen close to some of these muscle cells. In other mice with megaesophagus, they found that, few apparently normal smooth muscle cells, closely intermingling with striated muscle cells. Myenteric plexus tissue consisting of groups of unmyelinated axons was present between two layers of the muscle coat and collagen fibres. However, they concluded that, 3 month old mice with abdominal

megaesophagus showed several abnormalities of smooth muscle fibres and paucity of plexus tissue, accompanied by interstitial collagen deposition and suggested that the primary cause of megaesophagus in mice is neurogenic and not myogenic.

Ross *et al* (1995) viewed in the transmission electron microscopic examination (TEM) and revealed that the nerve terminals with empty appearing synaptic vesicles are interpreted to be cholinergic, secreting the neurotransmitter acetylcholine; those nerve terminals with dense granular material in the synaptic vesicles are interpreted to be adrenergic, secreting neurotransmitter nor epinephrine and they are likely to be contained in large vesicles with an opaque content.

Rang *et al* (2007) stated that the neuromuscular junction is a robust structure that very rarely fails, myasthenia gravis being one of the very few disorders that specifically affects it. They also stated that, this disease affects about one in 2000 individuals, who show muscle weakness and increased fatigability resulting from a failure of neuromuscular transmission. Functionally, it results in the inability of muscles to produce sustained contraction, of which drooping eyelids one characteristic signs of myasthenia gravis patient.

Lemasters (2009) stated that cellular changes leading to onset of necrotic cell death include formation of plasma membrane protrusion called blebs, mitochondrial swelling, and dilatation of cisternae of the endoplasmic reticulum and nuclear membrane. The cellular swelling leading to rupture, release intracellular contents, leading to finally necrotic cell death, which is characterised by loss of cellular architecture, vacuolization, karyolysis and increased eosinophilia soon become evident.

2.5 MEDICAL TREATMENT

Roudebush and Delivorias (1985) treated the case of mixed gastritis in pointer dog with Metronidazole @ 25 mg/kg orally twice and faeces regained normal consistency after 5 days treatment.

Hoenig *et al* (1990) suggested the changes in dietary management such as feeding with the animal positioned vertically, and changes in the consistency of the food. The dietary management was successful in one cat with megaesophagus for six years.

Strauss-Ayali and Simpson (1999) treated the dogs and cats with gastritis and helicobacter infection. It showed that 90% of 63 dogs and cats responded to treatment with combination of Metronidazole and Amoxicillin @ 20 mg/kg body weight orally twice for 14 days and Famotidine @ 0.5 mg/kg body weight orally twice for 14 days and 19 animals re-examined endoscopically had no evidence of Helicobacter in gastric biopsies.

Simpson (2005) treated the chronic gastritis with amoxicillin (20mg/kg orally Bid), Metronidazole (30 mg/kg body weight orally for twice) and ranitidine (10 mg/kg body weight orally twice) for one week and all dogs were showed complete recovery and clinical symptom disappeared after therapy. Similarly also suggested that, the gastric erosion or ulcers can be treated with fluid therapy (0.9% NaCl, LRS), acid inhibitors (ranitidine 0.5mg/kg body weight) and mucosal protective agent like sucralfate (1gm/kg body weight orally for one week).

Jergens (2005) stated that severe reflux esophagitis will require drug therapy and dietary management. Sucralfate suspension at the rate of 1gm/kg body weight orally twice for one week followed by Metochlopramide at the rate of 0.5mg/kg body weight twice daily for one week and ranitidine (0.2mg/kg body weight orally twice a

day) as well as cisapride (0.2mg/kg body weight twice a day) for one week are recommended drug therapy in esophagitis.

Fossum and Hedlund (2007) elucidated that drugs used to treat gastric ulcers and erosions include those that lessen the gastric acidity and those that protect the gastric mucosa from damaging includes cimetamidine (@5-10mg/kg), ranitidine (@2.2mg/kg PO, bid), famotidine (@0.5-1.0mg/kg, PO, bid) are H_2 receptor blockers that reduce gastric acid secretion, a key step in treating the gastric ulcers and erosions. They also suggested antacids (Magnesium hydroxide) that stimulate endogenous prostaglandin release, neutralize acids and bind bile salts. Sucralfate (@1gm/kg, PO, qid) forms a protective coating over the ulcers or erosions.

Glazer and Walters (2008) reported that esophageal healing is promoted by decreasing mechanical trauma to the oesophagus through feeding soft or blenderized food or administration of the sucralfate (healing of mucosal surface by releasing the prostaglandins) as slurry 3-4 times per day. Further injury to the oesophagus can prevent by reducing the acidity of gastric secretion and increasing LES tone. Antacids (calcium carbonate), H2-blockers (cimetidine, famotidine and ranitidine), proton pump inhibitors (Omperazole) are all used to lower gastric acidity and to prevent the ulceration of gastric mucosa and prokinetics (Metochlopramide and cisapride) are used to increase LES tone and increase gastric emptying.

Johnson *et al* (2009) recommended the use of prokinetics therapy to improve the esophageal motility in cases of megaesophagus such as Metochlopramide or cisapride. The observation have been questioned because Metochlopramide and cisapride both increase motility by binding $5HT_4$ (serotonin) receptors on enteric cholinergic neurons resulting in depolarization and contraction of GI smooth muscles. They also noted that, the prognosis of megaesophagus is variable and difficult to

predict. Successful outcome depends upon early diagnosis and aggressive dietary management.

Neiger (2009) stated that as ulcerated mucosa is vulnerable to further damage, mucosal protection with sucralfate (1gm/kg body weight) is indicated. Sucralfate is sulphate disaccharide-aluminium hydroxide complex that adheres to ulcerated tissue and provides a barrier to acid and pepsin penetration. It also stimulates endogenous prostaglandin synthesis in the gastric mucosa since sucralfate is effective at acidic to almost neutral pH. They further noted that, H_2 receptor antagonist (Cimetidine, ranitidine, Famotidine and Nizatidine) acts via competitive inhibition at the histamine receptor on parietal cells while proton pump inhibitors (Omperazole, Lansoprazole, Esomeoprazole, Pantoprazole and Robeprazole) inhibit the final step of acid secretion, the gastric pump and thus prevent histamine, acetylcholine and gastrin induced acid secretion.

Willard and Carsten (2009) stated that inflamed esophageal mucosa usually heals if it is protected from further damage because esophageal mucosa is more easily damaged by acid than gastric mucosa because the oesophagus does not have a mucous carbonate pre epithelial barrier and does not heal by epithelial restitution. They treated with cisapride, particularly effective in preventing recurrence after esophagitis.H_2 receptor antagonist (Cimetidine, ranitidine, Famotidine and Nizatidine) inhibits gastric acid secretion, thus diminishing the volume than can be refluxed into the esophagus. Proton pump inhibitors (Omperazole and Lansoprazole) are non-competitive and diminish the gastric acid secretion to a much greater degree for a much longer time do the H_2 receptor antagonist.

2.6 ENDOSCOPIC RETREIVAL OF ESOPHAGEAL AND GASTRIC FOREIGN BODIES

Guilford (1990) suggested that endoscopic removal of esophageal or gastric foreign bodies depends upon type of foreign bodies, anatomical location, the clinical appearance of the animal and attentiveness with which the pet is being observed by owner. Careful survey radiograph is essential to rule out location of foreign bodies and detection of perforation. Also he suggested that all esophageal foreign bodies should be rapidly removed, because they cause pain and dysphagia and may result in esophageal stricture.

Michels *et al* (1995) studied various techniques for removal of fishhook from the gastrointestinal tract of dogs and cats. The authors recommended the endoscopic retrieval if the equipment is available and if perforation of oesophagus or stomach is not present. Endoscopic retrieval was attempted in 62 of the 72 dogs with gastric or esophageal fishhook and was successfully removed from 17 out of 25 dogs (68%) dogs with gastric fishhook. Out of 36, 32 (34%) dogs with esophageal fish hook were successfully removed endoscopically.

Fossum and Hedlund (1999) stated that gastroduodenoscopy is more sensitive than radiographs in finding foreign bodies and it offers the chance to remove it at the same time. In addition, one can find other gastric lesion and take a biopsy from them. They also suggested that, endoscopic removal of linear foreign bodies should be probably only be attempted when the linear foreign body has only been present for a relatively short time (i.e. less than 3-4dyas).

Gualtieri (2001) reported that esophagoscopy confirm and remove the foreign bodies and help to assess the mucosal damage for prognostic purpose. Endoscopic removal should be considered as first approach to esophageal foreign bodies, depending upon shape and size. Flexible grasping forceps are inserted through the working channel of the endoscope, whereas the laparoscopic rigid grasping foreign

body forceps inserted alongside the endoscope help very well to firmly grasp large proximal objects. Once the objects grasped, if no resistance felt, the endoscope and forcep are withdrawn simultaneously, paying particular attention to the passage through the upper esophageal sphincter.

Zoran (2001) stated that endoscopic examination of upper gastrointestinal tract (GIT) in dogs and cats is a relatively recent development. It was just over 20 years ago; in 1978 that the first series report of GI endoscopy in dogs and cats was presented. In those initial days endoscopy was used to retrieve foreign bodies and for visualization of oesophagus and stomach. They also reported that, the gastric foreign bodies in dog i.e. surgical gloves in the pylorus were successfully removed by using foreign retrieval forcep through the accessory channel of the endoscope. Also he observed the thread like worm *(physaloptera)* on the surface of the gastric mucosa which caused chronic vomiting in one case. The worm was retrieved with endoscopic biopsy forceps and its removal was curative.

Fossum and Hedlund (2002) reported that endoscopy generally is of greater value than radiographic contrast studies in diagnosing the esophageal foreign bodies because the foreign body can potentially be removed during the endoscopic procedure.

Guilford (2005) suggested that the timely endoscopic retrieval of foreign bodies of oesophagus and stomach via endoscope is recommended because of the risk of perforation of the esophagus or stomach or if an object leaves the stomach perforate the intestine. Factors that influence this decision include the type of foreign objects, its anatomic orientation, location and clinical appearance of the animal. Basket retrieval instrument, a snare, a three or four pronged grasper and rat tooth grasper are the endoscopic grasping instruments for retrieval of esophagus and gastric

foreign bodies in dogs. Endoscopic entrapment with suture material can be used to remove large foreign bodies, such as choke chains, from the stomach. He also stated that, pushing obstinate esophageal foreign bodies into the stomach where they may be digested, better manipulation to an appropriate orientation for removal, or more easily removed surgically.

Baron (2008) stated that removal of foreign bodies has become the purview of gastroenterologist and almost all foreign bodies within the reach of endoscope can be removed endoscopically. Further he concluded that endoscopic extraction of foreign body (endotracheal tube) is highly effective method.

Chen *et al* (2008) stated that gentle pushing of the food bolus of foreign body into the stomach using the tip of the endoscope can be easily performed if there is no distal obstruction. The "Push technique" has been found to have success rates greater than 95% and complications approaching 0% in the treatment of food impaction. They also successfully removed the goose liver by application of this easily homemade instrument by attaching a modified nasogastric tube to the endoscope.

Meyer and Levine (2008) removed esophageal foreign body (endotracheal tube) in 41 year old man with esophagogastroduodenoscopy (EGD) technique and examined the periphery of the esophageal lumen which was characterized by erythematic and friability surrounding the foreign body, but no perforation or ulceration was appreciated. The tube was successfully removed using a rat tooth grasping forceps.

Weissberg (2008) stated that extraction of esophageal foreign bodies with the flexible fiber optic endoscope is often inadequate and may be dangerous. This is particularly so in the presence of sharp foreign bodies and those with protruding parts.

Under such circumstances as the danger of perforation is increased and the rigid (open tube) instrument should be used for their extraction.

Ponnuswamy *et al* (2009) reported that successful endoscopic retrieval of nasopharyngeal foreign body (bone fragment) in a nondescript 2 months old pup by using a rat toothed-alligator endoscopic forceps under general anaesthesia.

2.7 SURGICAL TREATMENT

Richard *et al* (1967) suggested that the best treatment for the dog with achalasia is to feed fluids two or three times each day with the dog in a vertical position; if fails they recommended a stomach tube to be passed for intragastric feeding.

Michels *et al* (1995) suggested that surgical removal is recommended if endoscopic retrieval is unsuccessful or if perforation is evident. In his study, twenty of 21 dogs in which endoscopic retrieval was not successful, underwent surgery. Surgical retrieval was not pursued in one dog with an esophageal fishhook, and the dog was euthanized at the owners' request. Gastrotomy was performed in 8 dogs with fish hook in the stomach.

Fossum and Hedlund (1999) suggested that if endoscopy fails to retrieve the foreign bodies, then surgical removal of foreign bodies (gastrotomy) is indicated. They also sated that the most common indication of gastrotomy in dos and cats is removal of foreign bodies.

Griffiths *et al* (2000) in a retrospective study on oropharyngeal penetrating injuries in 50 dogs, they carried surgical exploration in 48 dogs. The remaining 2 dogs had non wooden foreign bodies that were retrieved via an oral approach. In 85% of the dogs, sinus tract were identified to assess the in-depth by using probe. Wooden foreign bodies were removed in 18 dogs. No attempt was made to close the deep tissue, although dependent Penrose drains were placed in all wounds.

Kumar and Ameerjan (2001) studied eight clinical cases of esophago-gastric foreign bodies and pyloric stenosis and treated with gastrotomy and pyloroplasty respectively.

Eggertsdottir *et al* (2001) reported that the recurrence of GDV is 42% to 71% in dogs that have survived an acute episode of GDV, without a gastropexy being performed. Also they noted that there are several surgical techniques for fixation of stomach which have been recommended to reduce the risk of recurrence.

Fossum and Hedlund (2002) suggested that in cases of esophageal diverticulum, feeding of a soft, bland diet with the animal in an upright position to prevent accumulation of food in the pouch and large diverticulum should be surgically excised.

Johnson *et al* (2009) suggested that the management of idiopathic megaesophagus as well as most cases of megaesophagus resulting from neurological disease is entirely symptomatic and centres on special feeding techniques. A diet should be formulated using a high calorie food to provide adequate nutritional intake. Meals should be fed in small portions several times daily and given to the dogs in an upright position. This can be accomplished by placing food on elevated feeding platform or by simple holding the dogs in a vertical position for several minutes after

feeding. Upright feeding provides surprisingly effective symptomatic control of regurgitation in many dogs.

CHAPTER-III

MATERIALS AND METHODS

3.1 MATERIALS

The present study was undertaken in 42 cases out of the 120 clinical cases of dogs presented with diseases of esophagus and stomach with the history of persistent vomiting or regurgitation to the Department of Veterinary Surgery and Radiology, College of Veterinary Science, Rajendranagar, Hyderabad and Teaching Veterinary Clinical Complex, Bhoiguda, Secunderabad, from March-2008 to May -2010.

The dogs with upper gastrointestinal disorders were screened by physical, clinical, haematological and relevant biochemical examinations to rule out the presence of systemic, metabolic, parasitic and other extra GI causes of vomiting. Besides, radiography and ultrasonography were used in conjunction with endoscopy, whenever the condition of the animal permitted for further confirmation of diseases of esophagus and stomach, especially in cases where the medical treatment failed. Depending upon the diagnosis, the cases selected were treated by medical management or by surgery. In the present study, Flexible fiberoptic endoscope[1] with an outer diameter of 10 mm and 120 cm length were used (Fig.1). The angulations of endoscope were 120^{o} field view, 210^{o} upward, 90^{o} downward and 100^{o} right and left direction. Other ancillary instrumentation for flexible endoscope included biopsy forceps with serrated cups and central banoyet spike, sheathed cytology brushes and

foreign body retrieval forceps (Fig.2). In addition, electrically operated suction apparatus was used as an ancillary aid to the endoscope.

1- Karl Stroz Endoskope, Germany.

PHOTO PLATE 1, 2

Logiq α 100 ultrasound scanner[1] was also used for examination of the esophagus and stomach, wherever feasible. For routine contrast radiographic studies of upper gastrointestinal tract, barium sulphate was used as the contrast agent.

3.2 DESIGN OF STUDY

A total of 120 dogs were presented with the chief complaint of recurrent vomiting or regurgitation. These cases were examined and attempts were made to treat them symptomatically by medical means. The cases that responded to routine medical treatment and those cases of severe haemorrhagic gastroenteritis like Canine Parvovirus gastroenteritis were excluded and did not form a part of the present clinical study. Out of the total of 120 cases selected for closer examination, 78 dogs (65%) responded to routine medical treatment with antiemetics, antibiotics and fluid therapy and hence, they were excluded from the purview of the present clinical study. The remaining 42 dogs (35%), which continued to vomit or regurgitate for more than one week and showed signs consistent with the primary diseases of the esophagus and stomach formed the patient material for the present clinical study.

3.3 EXAMINATION OF ESOPHAGEAL AND STOMACH DISEASES

3.3.1 Physical Examination

The cervical part of the esophagus and stomach were palpated thoroughly for the detection of foreign bodies, swelling, pain, inflammation, distension and for evidence of any other structural abnormalities at cervical esophagus and abdomen.

1-Wipro GE Logiq α $100V_4$

3.3.2 Clinical Examination

The history and the patient signalment (age, breed, sex, size and body weight), duration of the symptoms, nutritional status, current deworming and vaccination status, past disease history and treatment were inquired to rule out concurrent systemic, parasitic and metabolic diseases prior to consideration of the cases for present study. All the dogs were subjected to routine clinical examination.

3.3.3 Haematological Studies

Blood samples were collected in a vial containing anticoagulant to perform complete blood picture (CBP) from all the 42 dogs that were included in the present study. The blood samples were analyzed with aid of the automatic whole blood analyzer[1].

3.3.4 Biochemical Studies

Five ml of serum samples were collected for the estimation blood urea nitrogen (BUN, mg/dl), Serum creatinine (mg/dl), ALT (U/L), AST (U/L) in all the dogs as part of the standard laboratory investigations. All these parameters were estimated by standard kits and methods supplied by Span Diagnostic Laboratories[2].

1- Humacount, Med Source Ozone, Biomedical, and Pvt.Ltd

2- Span Diagnostic Ltd. Surat.

3.4 DIAGNOSIS OF ESOPHAGEAL AND STOMACH DISEASES

3.4.1 Plain Radiographic Examination

Left lateral survey radiographs of the neck and thorax were obtained to rule out the structural or pathological abnormalities of esophagus and stomach, with a 500 mA Heliophos Multix D X-ray machine[1].

3.4.2 Contrast Radiographic Examination

Esophagrams and gastrograms were obtained to either confirm or rule out pathological conditions affecting the esophagus and stomach using barium sulphate[2] as the positive contrast medium. Prior to contrast studies of esophagus and stomach, all animals were kept off feed for 12 hours.

Esophagography was performed by oral administration of barium sulphate paste. The barium paste was prepared by mixing barium sulphate powder to raw eggs to a pasty consistency to encourage voluntary consumption of the same. Some of the dogs that refused to voluntarily swallow the paste, the same was orally administered by using a 50 ml syringe. The dogs were positioned for radiography and lateral and ventrodorsal radiographs of the neck, thorax and abdomen were obtained immediately after administration of barium paste and after 30 minutes.

1- Heliophos, Made in India

2- Eskay Fine Chemicals, Mumbai.

For Gastrography, a 25% barium sulphate solution was prepared in water and orally administered with a 50 ml syringe at the rate of 6 ml of the 25% solution per kg body weight. Lateral and ventrodorsal radiographs were taken immediately and after 10 and 30 minutes of administration.

3.4.3 Ultrasonographic Examination

All dogs kept on fasting for 12 hours but water was allowed free choices. The hair over the neck and the ventral abdomen wall was shaved, cleaned and an ultrasound gel was applied. In the present study, ultrasonography was used as a complimentary diagnostic procedure with radiography and endoscopy. Esophagus was scanned with a 7.5 MHz transducer while a 5 MHz transducer was used for the stomach.

3.4.4 Endoscopic Examination

3.4.4.1 Patient Preparation

All dogs were prepared by withholding food from them for 12 hours prior to endoscopy and necessary symptomatic therapy was instituted to address any fluid or electrolyte abnormalities to ensure anaesthetic safety.

3.4.4.2 Anaesthesia and Patient position

All the dogs about to be subjected to endoscopy and surgical procedures wherever necessary were premedicated with atropine sulphate[1] at the rate of 0.04 mg/kg body weight intramuscularly. The dogs were anaesthetized with xylazine hydrochloride[2] at the rate of 1 mg/kg body weight and ketamine hydrochloride[3] at the

---

1- Unison Drugs Pvt. Ltd. Chandigarh-160002.; 2- Indian Immunological Ltd. Gachibowli, Hyderabad-500032.

3- Themis Medicare Ltd.Haridwar-249403, Uttarkhand.

rate of 7.5 mg/kg body weight intramuscularly. As soon as the animals were induced with anaesthesia, an intravenous life line of ringer's lactate was started in all the dogs. Whenever required, anaesthesia was maintained by intravenous incremental doses of xylazine and ketamine in the same ratio.

All the dogs in the present study subjected to endoscopy for examination of esophagus and stomach were positioned in left lateral recumbency (Gualtieri 2001 and Guilford 2005).

Inj. Meloxicam[1] was administered intramuscularly at the rate of 0.4 mg/kg body weight, pre and post operatively for pain relief in cases where surgical treatment was contemplated. Inj. Ampicillin and Cloxacillin[2] were intramuscularly administered at the rate of 15 mg/kg body weight twice a day before surgical procedure and after surgery for seven days in dogs that were surgically treated.

3.4.4.3 Esophagoscopy

With the animal under deep surgical anaesthesia, a reliable mouth gag was placed to protect the endoscope during the procedure. The flexible fibre optic endoscope was introduced into the esophagus as it was insufflated with air until the lumen was clearly visualized. After mid cervical esophagus was examined, the tip of endoscope was advanced slowly to the thoracic part of esophagus until the lower gastroesophageal sphincter was visualized.

---

1 & 2- Intas Pharmaceutical Ltd. Matoda, Ahmedabad.

3.4.4.4 Gastroscopy

Following examination of the esophagus, the endoscope was gently advanced through the gastroesophageal sphincter (GES) into stomach. The stomach was distended with air by insufflation and the gastric mucosa and the contents were examined. Simultaneously, whenever necessary the retained gastric fluid was

aspirated with the help of electrically operated suction apparatus to visualize the mucosa and pathological lesions.

3.4.4.5 Collection of Cytological Samples

Cytological smears were obtained from all the dogs undergoing endoscopic examination of the stomach. Cytological smears were prepared, using brush cytology technique. Exfoliated cells were obtained by rubbing the brush inserted through the working channel of the endoscope against the gastric mucosa. The brush was then retracted and withdrawn from the endoscope. The brush was then rolled across the surface of glass slides to prepare cytological smears (Valentine, 2007; Stromar *et al*, 2008 and Rogers, 2009). The smears were air dried and stained with a Giemsa stain for cytological evaluation (Stromar *et al*, 2008).

3.5 DISEASE CONDITIONS OF THE ESOPHAGUS AND STOMACH ENCOUNTERED

Following the use of one or more of the examinations and/or diagnostic techniques described above, the following disease conditions of the esophagus and stomach were diagnosed. The details of the 42 cases selected for the present clinical study were presented in table 1.

Table 1: Age, breed and sex wise occurrence of diseases of esophagus and stomach in dogs (n=42)

S. N	Disease Condition	Age	Breed	Sex	No. of cases
1	Megaesophagus	4 years	Labrador	Male	
		2 years & 7 months	Golden Retriever	Male	
		7 months	German Shepherd Dogs	Female	

		8 years	Doberman	Male	18
		1years & 5 months	German Shepherd Dogs	Female	
		11 years	Mongrel	Male	
		6 years & 8 months	Pomeranian	Female	
		4 years	Cocker Spaniels	Male	
		9 years	Mongrel	Male	
		13 years	German Shepherd Dogs	Male	
		10 years	Pomeranian	Female	
		4 years & 4 months	Boxer	Female	
		3 years	German Shepherd Dogs	Male	
		9 years	Mongrel	Female	
		8 years	Doberman	Female	
		10 years	Labrador	Male	
		11montshs	Golden Retriever	Male	
		7 years	German Shepherd Dogs	Male	
2	Reflux Esophagitis	3 years & 5 months	Labrador	Male	2
		1years & 5 months	German Shepherd Dogs	Female	
3	Esophageal Nodule	9 years	Pomeranian	Male	1
4	Esophageal Foreign Body	2 years	Pomeranian	Male	5
		8 years	Labrador	Female	
		3 years	Labrador	Male	
		4 years & 5 months	Mongrel	Male	

		1years	German Shepherd Dogs	Male	
5	Vascular Ring Anomalies	2 years and 5 months	German Shepherd Dogs	Male	2
		3 months	English Bulldog	Male	
6	Esophageal Diverticula	3 years	German Shepherd Dogs	Female	1
7	Esophageal Erosions/Ulcers	7 years	Labrador	Male	2
		4 years	German Shepherd Dogs	Male	
8	Chronic Gastritis	3 years	Pomeranian	Female	3
		9 years	Mongrel	Male	
		8 years	Lhassa Apso	Female	
9	Gastric Erosions/ Ulcers	4years	Labrador	Male	2
		5 years & 5 months	German shepherd Dogs	Male	
10	Gastric Foreign Body	2 years	Labrador	Male	4
		3 years & 7 months	Mongrel	Male	
		5 years	Labrador	Male	
		7 years & 8 months	Pomeranian	Female	
11	Gastric Dilation & Volvulus	8 years	Great Dane	Male	2
		4 years	Labrador	Male	
Total number of cases encountered (n=42)					**42**

3.5.1 TREATMENT OF MEGAESOPHAGUS

A total of 18 cases (42.85%) of megaesophagus were encountered in the present study. The symptoms exhibited by these cases were recorded. These 18 cases of megaesophagus were divided into three groups by randomly assigning 6 dogs to each group. The medical treatment protocols adopted were as follows:

3.5.1.1 Cisapride Group

Out of 18 cases, six dogs of megaesophagus were subjected to medical treatment include Cisapride[1] at the rate of 0.5mg/kg body weight orally, twice a day. The owners were advised that medication will be needed permanently. The owners were also advised to feed the dogs from and elevated platform while the dogs were in an upright position. They were advised to maintain the dogs in an upright position for some time after feeding.

3.5.1.2 Metochlopramide Group

Out of 18 cases, six dogs of megaesophagus were treated with Metochlopramide[2] at the dose rate of 0.4mg/kg body weight, orally, twice daily. The owners were advised that medication will be needed permanently. The owners were also advised to feed the dogs from and elevated platform while the dogs were in an upright position. They were advised to maintain the dogs in an upright position for some time after feeding.

1- Kopran Pharmaceuticals Ltd. Worli Mumbai-400018.

2- Ipca Laboratories limited, Gujarat.

3.5.1.3 Feeding in Upright Position Alone

The owners were advised to feed the dogs from and elevated platform while the dogs were in an upright position. They were advised to maintain the dogs in an upright position for some time after feeding. Apart from this, no other medication was advised.

3.5.2 TREATMENT OF REFLUX ESOPHAGITIS

Out of the 42 dogs, two dogs (4.87 %) with reflux esophagitis were treated with sucralfate suspension[1] at the rate of 1 gm/kg body weight orally three times a day, Metoclopramide (0.2 mg/ kg orally) three times a day and Ranitidine[2] at the rate of 0.5mg/kg body weight, orally twice a day were used and treatment were continued for one week (Jergens, 2005).

3.5.3 ESOPHAGEAL NODULE

In the present study, one dog (2.43%) was reported with Spirocerci nodule in the thoracic part of esophagus just cranial to the base of heart. No treatment could be undertaken in this case.

3.5.4 ESOPHAGEAL AND GASTRIC FOREIGN BODIES

Out of the 42 dogs, nine cases (21.42%) of esophageal and stomach foreign bodies were reported. Among the nine dogs, five dogs had esophageal obstruction and four dogs had gastric foreign bodies. Esophageal foreign bodies included chicken bone, pork bone and socks whereas stomach foreign bodies were synthetic dog collar in two dogs, sewing needle and cool drink bottle metal caps (two) entrapped to each other in one case each.

1- Fourrts Laboratories Pvt. Ltd Chennai-6000020.

2- GlaxoSmithKline ltd, Worli Mumbai-4000025.

3.5.4.1 Endoscopic Retrieval of Esophageal Foreign Bodies

Five cases of esophageal obstruction due to foreign bodies were encountered in the present study. The type and location of the foreign bodies were recorded. In four out of the five dogs, they were found to be osseous foreign bodies. In one dog, socks caused esophageal obstruction just caudal to the pharynx. Out of the four dogs with osseous foreign bodies in the esophagus, in one dog, the obstruction was found to be just caudal to the pharynx and in the other three dogs; they were located in the mid thoracic portion of the esophagus.

Under deep surgical anaesthesia, the post pharyngeal foreign body was retrieved orally by endoscope, while simultaneously passing a long curved cheatle forcep along the endoscope. Endoscopic illumination helped locate and retrieve the foreign body. In the other three dogs, the osseous foreign body was dislodged and pushed back into the stomach.

3.5.4.2 Endoscopic Retrieval of Gastric Foreign Body

Under deep surgical anaesthesia, the synthetic dog collar encountered in the stomach of one dog was retrieved by rat tooth graspers through the endoscope.

3.5.4.3 Surgical Removal of Gastric Foreign Bodies

The two cases of gastric foreign bodies were retrieved through standard gastrotomy procedures. The linear gastric foreign body i.e. synthetic dog collar in one case detected on gastroscopy which could not be retrieved by the endoscope technique, was removed by performing gastrotomy and also enterotomy as the foreign body passed through stomach into the intestine.

3.5.4.4 Post operative care

Routinely the operated dogs were administered Ampicillin and Cloxacillin at the dose rate of 20 mg/kg body weight intramuscularly twice a day for seven days and Inj. Meloxicam at the dose rate of 0.5mg/kg body weight four days. The surgical wounds were dressed daily in a routine manner. The skin sutures were removed on 10^{th} postoperative day.

3.5.5 VASCULAR RING ANOMALIES

Out of the 42 dogs, two cases (4.76%) of vascular ring anomalies i.e. right persistent aortic arch (PRAA) were reported with severe debility and malnutrition in English bulldog and German shepherd dogs aged three months and two and half years respectively. These cases could not be treated.

3.5.6 ESOPHAGEAL DIVERTICULUM

In the present study, one three years old female German shepherd dog was reported with history of recurrent regurgitation, weakness and dehydration. No treatment could be initiated in this case since owner was not willing to do the surgical treatment.

3.5.7 TREATMENT OF ESOPHAGEAL AND GASTRIC EROSIONS /ULCERS

Out of the 42 dogs, four cases (9.72%) of esophageal and/or gastric erosion and ulceration were recorded endoscopically. They were treated with fluid therapy to restore electrolyte and acid-base balance.

Additionally all the four cases were treated supportively with sucralfate suspension at the dose rate of 1gm/kg body weight orally twice a day for 7 days and Omeprazole[1] at the dose rate of 0.7 mg/kg body weight, orally thrice a day (Jergens, 2005). All four dogs were kept under observation for one month to assess the response.

3.5.8 TREATMENT OF CHRONIC GASTRITIS

In the present study, three cases (7.14%) of chronic gastritis were recorded and treated with combination of Amoxicillin[2] (20mg/kg body weight orally twice a day); Clarithromycin[3] (7.5 mg/kg body weight orally twice a day) and Metronidazole[4] (10mg/kg body weight orally twice a day) for two weeks. Ranitidine was given at the rate of 0.5 mg/kg body weight, orally for 14 days as described by Simpson (2005).

3.5.9 GASTRIC DILATATION AND VOLVULUS (GD-V)

The clinical and radiographic features of GD-V were recorded. The dogs were surgically treated by performing laparotomy, derotation of the stomach and gastropexy as per the standard procedure described by Fossum and Hedlund (2007).

3.6 NECROPSY EXAMINATION

In the present clinical study, out of total 42 cases, nine (21.42%) of dogs were died and necropsy examination were conducted. Among nine cases, seven dogs were died from megaesophagus during the course of the study were examined for necropsy, histopathological, scanning and electron microscopic examinations. The remaining

two cases of dogs were died due to vascular ring anomalies during conservative treatment; the gross and histopathological changes were recorded.

1- Alkem Laboratories Pvt. Ltd Lower Parel, Mumbai-400013

2- Zydus Cadila health care Ltd Ahmedabad-380015.

3- Alembic Chemicals Works Co. ltd.Vodadara-390003.

4- Alkem Laboratories Pvt. Ltd Lower Parel, Mumbai-400013

The tissues from the relevant areas, i.e., esophagus and stomach were collected and fixed in 10% formalin and processed for histopathologal, scanning and transmission electron microscopic examination. Remaining two dogs were died due to right persistent aortic arch during course of treatment. Necropsy and Histopathological findings were recorded.

3.6.1 Histopathological Examination

After proper fixation of tissues in 10% formalin, tissues samples were trimmed properly and kept for washing under running tap water for over night. The tissues were dehydrated in ascending grades of alcohols and finally cleared in xylol. The tissue specimens were embedded in paraffin blocks. Sections of 3-5 µ thickness were cut by rotary microtome. Sections were stained with routine Haematoxylin and Eosin staining method (Culling, 1957) and examined under a microscope to study the histopathological changes.

3.6.2 Scanning electron microscopic (SEM) examination

The normal and diseased wall of esophagus was subjected to scanning electron microscopic (SEM) examination to study the morphological or structural changes and tissues sample were collected during the post-mortem examination study. The normal esophageal tissue samples were obtained from a dog that was euthanized due to

irreparable trauma in an automobile accident. This dog was not related to the present study.

For microscopic studies, the samples were transferred to vials and fixed in 2.5% glutaraldehyde in 0.05 M phosphate buffer (ph 7.2) for 24 hrs at 4^0 and post fixed 2% aqueous Osmium tetraoxide in the same buffer for 2 hrs. After the post fixation samples were dehydrated in a series of graded alcohol and dried to critical point with electron microscopy science CPD unit. Then the dried samples were mounted over the tubes with double sided conductivity tape. Finally, a thin layer of gold metal was applied over the sample using an automated sputter coater (JEOL JFC-1600) for about three minutes. Then the samples were scanned in the scanning electron microscope (Model: JOEL-JSM 5600, JAPAN) at various magnifications at RUSKA LAB, College of Veterinary Science, SVVU, Rajendranagar, Hyderabad, India.

3.6.3 Transmission electron microscopic (TEM) examination

The normal and diseased wall of esophagus was subjected to transmission electron microscopic (TEM) examination to study the cellular changes and tissues sample were collected during the post-mortem examination study. The normal esophageal tissue samples were obtained from a dog that was euthanized due to irreparable trauma in an automobile accident. This dog was not related to the present study.

Samples were fixed in 2.5% glutaraldehyde in 0.05 M phosphate buffer (pH 7.2) for 24 hrs at 4^0 and post fixed 2% aqueous Osmium tetraoxide in the same buffer for 2 hours. Dehydrated in series of graded alcohols, infiltrated and embedded in araldite 6005 resin or spur resin (Spurr 1969). Ultra thin (50-70nm), sections were made with a glass knife on ultra microtome (Leice ultra cut UCT-GA-D/E-1/00),

mounted on cooper grids and stained with saturated aqueous uranyl acetate and counter stained with Reynolds lead citrate. Viewed under TEM (Model: Hitachi, H-7500 from JAPAN) at required magnifications as per the standard procedures at RUSKA Lab, College of Veterinary Science, SVVU, Rajendranagar, Hyderabad, India.

CHAPTER-IV

RESULTS

In the present clinical study, 42 cases out of 120 clinical cases of dogs were presented with chief complaint of vomiting, recurrent regurgitation, anorexia, and generalised weakness with severe to moderate dehydration. All dogs were screened and medically treated with antibiotics, antiemetics and fluid therapy. Out of the 120 cases, 78 (65%) dogs recovered. These dogs were considered to be cases of gastroenteritis of viral aetiology like canine parvovirus or other infections and hence were excluded from the present study as they fell beyond the purview of the present study. The remaining forty-two (35%) dogs that did not respond to medical treatment and were thought to be suffering from diseases of the esophagus and/or stomach. Therefore, they were included in the present study for further detailed investigation. These dogs continued to vomit, had recurrent regurgitation, ptyalism, coughing, hematemesis, and cachexia of more than two weeks duration despite medical treatment.

4.1 OCCURENCE OF ESOPHAGEAL AND STOMACH DISEASES

The results of the present study revealed that out of the total 42 cases encountered, 31dogs (73.81%) cases accounted for the diseases of the esophagus while the rest (11dogs; 26.19%) were diseases of the stomach. Out of these 42 cases,

megaesophagus was diagnosed in 18 (42.86%) dogs. Similarly, the other diseases diagnosed during the present clinical study were reflux esophagitis (2 dogs; 4.76%), vascular ring anomalies (2 dogs; 4.76%), esophageal diverticula (1 dog; 2.38%), esophageal nodule (1 dog; 2.38%), esophageal foreign bodies (5 dogs; 11.90%), esophageal erosion or ulcers (2 dogs; 4.76%), chronic gastritis (3 dogs; 7.14%), gastric foreign bodies (4 dogs; 9.52%), gastric erosion/ ulcer (2 dogs; 4.76%) and in 2 dogs (4.76%), gastric dilatation and volvulus (Fig.3). The results also showed that the occurrences of these diseases were seen in dogs ranging from two and a half months to thirteen years of age. The occurrence of these diseases was found to be more in male dogs (29 dogs; 69.05%) than in females (13 dogs; 30.95%).

Irrespective of the disease entity encountered, German Shepherd Dogs were found to be the most commonly affected with the diseases of the esophagus and/or stomach (11 dogs; 26.19%), followed by Labrador (10 dogs; 23.80%), Pomeranian (6 dogs; 14.28%), Mongrels (6 dogs; 14.28%), Golden retriever and Doberman (2 dogs each; 4.76% each). Solitary cases (2.38% each) of the diseases of the esophagus and/or stomach were encountered in Lhassa Apso, English Bull dog, Cocker Spaniel, Boxer and Great Dane (Fig. 4).

4.2 EXAMINATION OF ESOPHAGEAL AND STOMACH DISEASES

4.2.1 Physical Observations

All the 42 dogs underwent routine physical examination as an essential part of clinical investigation. Out of the 42 cases, 18 (42.86%) dogs with megaesophagus showed emaciation, hide bound condition, shrunken abdomen, prominent rib cage (Fig.5) and slight to moderate pain and discomfort at cervical and abdominal area on palpation. Abdominal palpation revealed variable degree of abdominal tympany or distension in two dogs (4.76 %) with GD-V. It was found that in the Labrador dog,

abdominal distension was difficult to perceive. In four dogs (9.52 %) palpation revealed slight to moderate pain and swelling at cervical area, depending on the diseases encountered.

PHOTO PLATE 3,4,5

History revealed chronic weight loss in most of the dogs that had diseases of esophagus and/or stomach, except for the dogs with acute onset of disease like esophageal/gastric foreign bodies, gastric dilatation and volvulus (GD-V). The physical findings were prominent in dogs that had primary esophageal diseases like megaesophagus, vascular ring anomalies, esophageal foreign bodies etc., while dogs with gastric abnormalities except gastric dilatation and volvulus exhibited non specific symptoms.

4.2.2 Clinical Observations

Clinical signs exhibited by all the dogs suffering from the diseases of the upper gastrointestinal tract were recorded in all 42 dogs. Mean duration of the clinical signs prior to presentation was approximately three months (range 2-4 months). Vomiting or regurgitation and weight loss were the most commonly recorded clinical signs in thirty (71.42%) dogs. Persistent regurgitation of variable frequency and timing of regurgitation after feed was consistently observed in all the dogs with primary esophageal disease. The regurgitated material was reported to be undigested food particles. Weight loss, stiff gait, generalised weakness and emaciation secondary to malnutrition were seen in 24 dogs (57.14%) including two puppies aged between 3 months to two years and five months, suffering with vascular ring anomaly. One dog showed postprandial regurgitation, odynophagia and retching.

Out of the 42 cases, six (14; 29%) dogs had coffee coloured vomiting, severe to moderate dehydration, weakness, pale mucous membrane, inappetence, hypersalivation and abdominal pain. These symptoms were noticed in the dogs afflicted with primary diseases of the stomach.

4.3 MEGAESOPHAGUS

In the present clinical study, 18 clinical cases of megaesophagus were recorded. The disease was encountered in Labrador (2 dogs), Golden Retriever (2 Dogs), German Shepherd (5 Dogs), Doberman (2 Dogs), Mongrel (3 Dogs), Pomeranian (2 Dogs), Cocker Spaniel (1 Dog) and Boxer (1 Dog) breeds of dogs. The mean age of occurrence was found to be 6.25 ± 0.88 years. Out of these 18 dogs, 11were found to be males (61.12 %) and the rest were females (7 dogs; 38.88%).

4.3.1 Physical and Clinical Observations

The eighteen dogs with megaesophagus showed symptoms of emaciation hide bound condition, shrunken abdomen, prominent rib cage and slight to moderate pain and discomfort at cervical area on palpation. History revealed chronic weight loss in most of the dogs that had megaesophagus with the owners reporting a weight loss of about 3 to 5 kg over the past 2 to 3 months.

Physical examination also revealed that these dogs consumed food very eagerly when offered and appeared extremely hungry. However, all these 18 dogs showed the signs of regurgitation of the food soon after food consumption. Regurgitation was reported to have occurred immediately after to several hours after feeding. The regurgitated material was reported to be undigested food. Stiff gait due to generalised weakness and emaciation secondary to malnutrition were seen in all the cases. Postprandial regurgitation, odynophagia and retching were also reported in 10 out of the 18 dogs. In the rest of the dogs, regurgitation was reported to be effortless.

4.3.2 Haematological and Biochemical Observations

The dogs with megaesophagus had a mean total erythrocyte counts of 4.29 ± 0.08 millions per cu. mm, haemoglobin level of 8.74 ± 0.12 g/dl and the mean total leucocyte count of 15.63 ± 0.12 thousands per cu mm. The differential leucocyte counts were with in normal limits and are depicted in table.2.

The mean biochemical values in the present study in dogs with megaesophagus were presented in table.3. The mean Blood Urea Nitrogen levels were found to be 20.94 ± 0.20 mg/dl, the mean Serum creatinine was found to be 1.04 ± 0.02mg/dl. The mean serum AST levels of 60.19 ± 0.66 U/I and serum ALT levels of 48.12 ± 0.61 U/I were recorded at the time of presentation for diagnosis and treatment.

4.3.3 Plain Radiographic Findings

The present study showed that megaesophagus could be readily diagnosed in seven out of the 18 dogs by plain radiography. Radiographic features of megaesophagus in these seven dogs were recorded to be air filled dilated esophagus, extending throughout the length of the esophagus (Fig.6). Tracheoesophageal stripe sign was consistently noticed in these seven cases.

In four out of the remaining 11 dogs, megaesophagus could still be diagnosed because of discernability of gas filled esophagus in a segment that stood out against the trachea. In these dogs, however, the entire esophagus could not be delineated (Fig.7). These dogs later turned out to be affected with megaesophagus on further examination by other means. In the remaining seven dogs, the radiographs did not reveal any signs suggestive of esophageal dilatation or megaesophagus.

PHOTO PLATE 6,7

Table.2: Mean values of haematological parameters in dogs with esophageal and stomach diseases (n=42).

S.N	Condition	TEC (millions per	Hb (g/dl)	TLC (thousands per	Differential Leukocyte Count (%)				
					G	L	M	N	E

		cumm)		cum m)	**(%)**	**(%)**	**(%)**	**(%)**	**(%)**
1	Megaesophagus (n=18)	4.2± 0.08	8.74± 0.12	15.63 ±0.12	80.72 ±0.66	17.83 ±0.21	5.22± 0.10	78.72 ±0.64	1.39 ±0.5
2	Reflux Esophagitis (n=2)	5.25	11.57	16.94	84.00	16.00	6.00	78.00	1.50
3	Vascular Ring Anomalies(n=2)	5.35	13.85	18.05	93.00	21.52	5.50	69.50	0.50
4	Esophageal Diverticula(n=1)	5.83	15.40	16.70	67.00	24.00	7.00	84.00	2.00
5	Esophageal Tumour(n=1)	6.41	12.97	17.50	81.00	18.00	3.00	68.00	1.00
6	Esophageal Foreign Bodies(n=5)	5.55	12.75	17.15	85.54	16.75	4.25	73.25	1.25
7	Esophageal Ulcers(n=2)	3.00	10.14	17.95	82.55	18.00	4.00	78.00	0.50
8	Chronic Gastritis(n=3)	4.90	9.83	17.07	77.00	16.67	4.00	84.33	1.33
9	Gastric Foreign bodies(n=4)	4.78	12.98	16.33	84.75	18.00	5.00	78.00	0.75
10	Gastric erosion(n=2)	4.57	12.15	16.95	83.50	21.50	4.00	80.00	1.50
11	GDV(n=2)	5.85	14.50	17.25	80.50	19.00	3.50	75.50	1.00

Table.3: Mean values of biochemical parameters in dogs with esophageal and stomach diseases (n=42).

S. N	**Condition**	**BUN(**mg/dl**)**	**Creatinine** (mg/dl)	**ALT(**I/U**)**	**AST(**I/U)
1	Megaesophagus (n=18)	20.94±0.20	1.04±0.02	60.19±0.66	48.12±0.61

2	Reflux Esophagitis (n=2)	19.51	1.05	68.10	40.50
3	Vascular Ring Anomalies(n=2)	22.00	1.00	67.95	66.35
4	Esophageal Diverticula(n=1)	16.00	1.40	71.50	56.80
5	Esophageal Tumour(n=1)	19.00	0.70	56.10	68.40
6	Esophageal Foreign Bodies(n=5)	18.75	1.07	48.85	43.55
7	Esophageal Ulcers(n=2)	19.00	0.85	57.21	38.32
8	Chronic Gastritis(n=3)	18.33	1.27	67.57	37.00
9	Gastric Foreign bodies(n=4)	18.50	0.88	44.78	36.98
10	Gastric erosion(n=2)	20.00	1.05	43.55	38.15
11	GDV(n=2)	16.50	1.05	41.70	44.00

4.3.4 Contrast Radiographic findings

Contrast radiographic examination (Esophagography) with barium paste helped confirm the tentative diagnosis of megaesophagus in all the 18 dogs. This could be confirmed even in the seven dogs that did not show any radiographic signs of megaesophagus on plain radiography. Esophagography clearly revealed generalized distention of esophagus in all the dogs (Fig.8.), confirming

megaesophagus. Esophagography also revealed the extent of dilation of the esophagus (Fig.9) and whether the dilation was involving the entire length of the esophagus or was confined to some segments of the esophagus. Esophageal dilatations of only segments of esophagus were not considered as megaesophagus in the present study.

4.3.5 Ultrasonographic Observations

Ultrasonography of the cervical esophagus failed to reveal any indication of any sort of esophageal pathology. In most of the cases, the esophagus could not be ultrasonographically delineated.

4.3.6 Endoscopic Findings

Endoscopic examination of the upper gastrointestinal tract in the present study enabled complete visual examination of the larynx, esophagus and the stomach. The epiglottis could be clearly visualized in all the dogs (Video.1) and this helped to rule out oropharyngeal diseases. A few normal dogs selected which did not form patient material in the present clinical study, were subjected to endoscopic examination of normal esophagus with a view to study the, normal endoscopic appearance of mucosa of esophagus. The normal esophagus appeared healthy pinkish in colour, with longitudinal folds indicating its ability to expand when needed. The normal esophagus was noticed to

PLATE 8 & 9

be a closed tube that always remained collapsed until it was dilated by insufflation during endoscopy (Video.2).

The eighteen dogs examined by endoscopy revealed markedly dilated, flaccid esophagus extending from the cranial cervical region to the gastroesophageal sphincter and pooling of retained fluid, saliva and fermenting fluid in the lumen, in the most dependent segment of the esophagus (Fig.10 and Video.3). The most

dependent region was found to be the cranial to the mid thoracic portion of the esophagus. The impression of the ribs was consistently found at the cranial to the mid thoracic region (Fig.11) in all the dogs confirming the endoscopic diagnosis of megaesophagus (Video.4). In the caudal thoracic esophagus, however, on endoscopic examination did not reveal the impressions of the ribs onto the esophageal lumen in cases of megaesophagus. Esophagoscopy also revealed the level of the base of the heart, thereby enabling the location of the endoscope tip within the thoracic esophageal expanse.

4.3.7 Treatment of Megaesophagus

The 18 cases of megaesophagus were randomly assigned to three groups of six dogs each. Medical management of these cases was attempted by the use of cisapride, Metoclopramide and feeding in an upright position in the three groups mentioned above.

4.3.7.1 Cisapride Group

Out of the six cases that received cisapride at the rate of 0.5 mg/ kg body weight orally, twice a day combined with feeding the dog while in an upright position from an elevated platform, four dogs showed good improvement in body weight by three months of medical treatment. The frequency of regurgitation gradually reduced starting from one week of treatment. Gradually, no regurgitation was reported during the six months of

PLATE 10 & 11

follow-up period. These dogs continue to be appear normal till the present study was closed, although occasional regurgitation is reported by the owners. In two dogs, recurrent regurgitation, loss of body weight and frequent episodes of aspiration continued despite treatment. These two dogs died after 4 months of treatment. No improvement could be noticed in these two dogs due to the treatment.

4.3.7.2 Metochlopramide Group

Five dogs showed good response to metochlopramide therapy (0.4mg/kg body weight orally) along with feeding the dog in an upright position from an elevated platform. The clinical symptoms of regurgitation gradually reduced in frequency and disappeared three months later. During the follow up visits, all dogs showed gain in considerable body weight. The sixth dog showed continued regurgitation of the food material inspite of the medical treatment. Two months later, this dog died due to generalised weakness, malnutrition and aspiration pneumonia.

4.3.7.3 Feeding in an Upright Position Group

Out of six cases, two dogs showed some response to feeding the dog in an upright position from an elevated platform. Regurgitation and other clinical symptoms did not improve during the course of the follow up period. The remaining four dogs continued to regurgitate during the dietary management period without any improvement in the clinical signs. As per the request of the owner all four dogs were euthanized as the body condition was worst or irreparable.

4.3.7.4 Necropsy Findings

In the present study, out of 18, three dogs died and four dogs were euthanized during the course of the treatment due to malnutrition, generalised weakness and dehydration. The dilated part of esophagus contained ingesta and the esophageal wall was noticed to be thin (Fig.12) with congestion of the mucosal surface. The lungs were congested with ecchymotic or patchy haemorrhagic spots over, suggestive of aspiration pneumonia (Fig.13).

4.3.7.5 Histopathological Findings

In present study, the esophageal tissues from the seven dogs that either died or were euthanized were subjected to histopathological examination. The esophageal samples showed infiltration of polymorphonuclear cells with submucosal congestion (Fig.14) and enlargement of submucosal glandular pattern with epithelial irregularity (Fig.15).

4.3.7.6 Scanning Electron Microscopic Findings

In the present study, scanning electron microscopic examination was conducted on the mid portion of the normal esophagus to enable comparison with those of megaesophagus. Scanning electron microscopy of the normal esophagus showed blood vessels and mixed mucous glands. The thickness of the various layers of the esophageal wall in theses normal dogs were measured. The thickness of the esophageal mucosa was found to be 669 µm while the submucosa was found to be 227 µm thick. The inner circular muscularis and the outer longitudinal muscularis layers measured 376 µm and 336 µm respectively (Fig.16).

PLATE-12 to 15

PLATE-16

On the contrary, scanning electron microscopy of the esophageal tissues from dogs affected with megaesophagus revealed destructed blood vessels, loss of normal architecture and direction of inner circular as well as outer longitudinal muscle fibers. The mucosal layer contained frothy deposition with damaged ductile pattern and stratified squamous epithelial layer. The layer wise thickness the different layers of the megaesophagus tissues was measured. The mucosal layer measured 269.14 ± 9.84 µm, submucosal layer measured 315.42 ± 5.15µm, the inner circular muscularis layer measured 214.28 ± 5.50 µm and outer longitudinal muscularis layer was found to be 247.42 ± 4.06 µm thick (Fig.17 and table.4).

4.3.7.7 Transmission Electron Microscopic Findings

In the present study, the transmission electron microscopic examination was conducted on mid portion of the normal esophagus of dog which was died due to accidental trauma to enable comparison with those of megaesophagus.

The transmission electron microscopic examination of normal esophagus showed intermyofibrillar junction characterised by loose connectivity between the myofibril bundles, nucleus contains dark and scanty chromatin material distributed at periphery of the nuclear wall (Fig.18). Neuromuscular junction (myoneuronal plate) located between the two muscle fibers with synaptic cleft and vesicles (Fig.19).

In contrast, the transmission electron microscopic examination of dogs with megaesophagus showed dilated myofibrillar bundles, condensed nucleus, complete loss of cellular architecture and vacuolization between muscle fiber bundles, karyolysis (Fig.20). All dogs showed the complete loss of normal shape of mitochondria characterised by swollen and condensed mitochondria with thick electron dense material

PLATE-17,18

Table.4. Mean values of scanning electron microscopic (SEM) measurements of esophageal wall thickness (µm) of the megaesophagus dogs (n=7).

Tissue layers	**Megaesophagus dogs**							**Mean ± S.E**
	Case-1	Case-2	Case-3	Case-4	Case-5	Case-6	Case-7	
Mucosa (µm)	240	257	249	273	298	257	310	269.14 ± 9.84
Submucosa (µm)	308	315	324	307	342	310	302	315.42 ± 5.15
Inner circular muscularis (µm)	216	210	198	241	221	211	203	214.28 ± 5.30
Outer longitudinal muscularis (µm)	252	235	268	246	238	245	248	247.42 ± 4.06

PLATE-19, 20

(Fig.21). Neuromuscular junction showed complete loss of architecture of myoneural plate with thick synaptic cleft and vesicular degeneration (Fig.22).

4.4 REFLUX ESOPHAGITIS

In present study, two clinical cases of dogs were presented with history of anorexia, dysphagia and hypersalivation since one month. Physical examination did not point to any tentative diagnosis. However, loss of body weight was reported in both the cases. Clinically, intermittent regurgitation, anorexia, frothy and ropy blood tinged salivation was noticed in both the dogs. The haematological and biochemical parameters were within normal limits and are presented in table 2 and 3 respectively. Plain and contrast radiography in these dogs did not reveal any abnormality.

Endoscopic examination showed the evidence of reflux esophagitis in these two dogs. Endoscopic examination was characterized by mucosal erythema, haemorrhage, mucosal irregularity, erosion with thickened mucosal folds especially at the lower esophageal area (Fig. 23 and Video.5).

Both the dogs recovered uneventfully following the treatment adopted as mentioned in the chapter III. Endoscopic examination after two weeks showed resolution of all the esophageal changes with the esophagus appearing normal on endoscopy.

4.5 ESOPHAGEAL NODULE

In the present study, a nine year old male Pomeranian dog presented with history of recurrent cough and retching was subjected to endoscopy. Esophagoscopy revealed normal a lemon sized nodular growth located just caudal to the base of the heart. A small lesion resembling an opening was also noticed (Fig.24).

Plate 21,22

PLATE-23, 24

Esophagoscopy helped locate the position of the nodule, since it was found just caudal to the area where the movements of the base of the heart could be seen (Video.6).The owner refused any surgical exploration since the symptoms were mild and the dog was not available for further evaluation.

4.6 ESOPHAGEAL FOREIGN BODIES

In the present study, out of 42 cases, five dogs (11.90%) were reported with history of sudden onset of regurgitation, dysphagia, anorexia, odonyphagia, and retching, gagging, excessive salivation, cough reflexes and uneasiness after a meal.

Plain lateral radiographs of the neck and thorax revealed the presence of osseous foreign bodies in the esophagus of four dogs. In one dog, the osseous foreign body was located just caudal to the pharynx (Fig.25) and in the other three, in the mid thoracic region (Fig.26). In another dog, the lateral cervical radiograph revealed the presence of a radiolucent foreign body, which later turned out to be socks (Fig.27).

Treatment of these five dogs with esophageal obstruction due to foreign bodies was treated by various means. The dog with socks in the cervical esophagus vomited the socks out and was relieved of obstruction due to the emetic effect of xylazine even before the dog could be anaesthetized and an endoscopic examination could be performed. In the dog where the osseous foreign body was located just caudal to the pharynx, the same was retrieved orally using a long cheatle forceps passed along the endoscope and was removed under endoscopic illumination (Fig.28). In the three dogs where the osseous foreign bodies were located in the thoracic region, by endoscopy, the bone pieces could be pushed back into the stomach (Fig.29) by the endoscope itself. Unfortunately, video recordings in these cases are not available.

PLATE-25, 26, 27

PLATE-28, 29

However, following pushing of the osseous foreign body into the stomach, the recording of the same is presented in video.7. In one case, however, an endotracheal tube was first introduced into the esophagus and the endoscope was passed through its

bore. Under endoscopic illumination, the tip of the endotracheal tube was used to push the foreign body into the stomach. Once the foreign bodies in these five dogs were removed as described above, all the five dogs were immediately resolved of their symptoms. The osseous foreign bodies probably got digested in the stomach and hence, the dogs were reported to be normal subsequently.

After two week all the dogs were again underwent routine endoscopic examination of esophagus as well as stomach revealed normal mucosal orientation.

4.7 VASCULAR RING ANOMALIES

In the present study, two dogs aged between two and a half months to three months were presented with history of regurgitation, debility and generalised weakness. History revealed that both the puppies started regurgitation from the time of weaning. The haematological and biochemical values are presented in table 2 and 3.

Positive contrast radiography with barium sulphate paste revealed narrowing of esophageal lumen just at cranial to the base of heart and the portion of esophagus cranial to it was severely and widely dilated (Fig.30). Endoscopic examination could not be carried out in these cases because their physical condition rendered them unfit for anaesthesia. Both the dogs died soon after despite intravenous nutrition in a attempt to make them fit for anaesthesia and surgery. Post mortem examination of these two carcasses was carried out.

Plate-30

Necropsy revealed severe and flaccid distension of the esophagus cranial to the constrictive vascular band. Closer examination of the esophagus also clearly showed the area where the vascular ring had obstructed the esophagus (Fig. 31). The distended esophagus contained recently consumed food. In one of the dogs, in addition to the changes in the esophagus, there were minute nodules in the gastric wall, hypertrophied rugae with generalised congestion of mucosa (Fig.32).

Histopathological examination of the esophageal wall revealed enlarged mucous gland, congestion of submucosa and cellular infiltration (Fig.33) whereas the stomach wall showed patchy foci fibrosis with infiltrates of lymphocytes, plasma cells and neutrophils (Fig.34), suggestive of concurrent mild diffuse sub acute gastritis in addition to esophageal distension.

4.8 ESOPHAGEAL DIVERTICULUM

Three year old female German shepherd dog presented with chronic and postprandial regurgitation. Physical and clinical examination revealed moderate dehydration, weakness and anaemia. Haematological and biochemical values were within normal limits and depicted in table 2 and 3.

Lateral plain radiography revealed sac like out pouching of cranial thoracic oesophagus that rested on the sternum (Fig.35). Endoscopy confirmed the diagnosis of esophageal diverticulum with sac like out pouching of esophageal wall and presence of partially digested food particles (Fig.36). No treatment could be initiated in this case since the owner was reluctant for surgery.

Plate-31, 32

Plate-33, 34

Plate-35, 36

4.9 ESOPHAGEAL EROSIONS/ULCERS

Two dogs with vomiting of about three week's duration that did not respond to routine medication were closely examined for any primary diseases of esophagus. The haematological and biochemical values were within the normal range and depicted in table 2 and 3.

The contrast Esophagram showed remnants of barium sulphate still to the mucosa of the cervical part of esophagus (Fig.37). Endoscopically, both the dogs showed friable mucous membrane, petechial haemorrhage and erosion of the esophageal mucosa (Fig.38 and video.8).

Both the dogs responded well to the treatment adopted. By one week after initiation of treatment, the dogs stopped vomiting and became normal. They were monitored for two months for signs of relapse of clinical symptoms. Relapse of clinical signs was not observed in these dogs.

4.10 CHRONIC GASTRITIS

In present study, three (7.14%) dogs with chief complaint of chronic vomiting, anorexia and loss of body weight were presented. The haematological and biochemical findings in these dogs are presented in table 2 and 3.

During the course of the present clinical study gastroscopy was also carried out in normal dogs which did not form the patient material of the present study with a purpose to visualise the normal endoscopic appearance of gastric mucosa. The features of the normal gastroscopic appearance was characterised by healthy glistening pink mucosa, with sufficient gastric secretions and normally appearing smooth rugal folds.

Plate-37, 38

The normal gastroscopic appearance of the stomach was presented in video.9**.** The presence of a few strands of hair in the stomach as seen in the video was considered normal. Gastroscopy of all three dogs revealed generalised congestion as well as petechial hemorrhagic spots on gastric mucosa with minor areas of mucosal erosion (Fig.39).

Cytological examination of stomach fluid collected during endoscopy showed large cocci and rod shaped bacteria along with the presence of lymphocytes indicating gastritis (Fig.40). Cytological examination also showed colonization of non-specific bacteria in the stomach contents (Fig.41). No Helicobacter Like Organisms (HLO) was found in any case during the present study. These three dogs recovered uneventfully in response to the treatment adopted.

4.11 GASTRIC EROSIONS/ULCERS

In the present study, two dogs with history of vomiting and weakness since one and half months, were diagnosed to be affected with gastric erosions/ulcers. The haematological and biochemical findings in these dogs are presented in table 2 and 3.

Endoscopic examination revealed gastric ulcerative patches with marked thickening and irregular edges. Erosion or ulcers were characterised slightly by depressed areas with focal streaks of haemorrhage (Fig.42 and video.10). Treatment of these dogs as mentioned in chapter III led to uneventful recovery by one month of medication.

Plate-39, 40

Plate-41, 42

4.12 GASTRIC FOREIGN BODIES

4.12.1 Clinical, Haematological and Biochemical Findings

Four dogs were reported with chief complaint of persistent vomiting, dehydration and inappetence since five to seven days. Physical examination of abdominal area revealed intense pain, discomfort, uneasiness and loss of appetite. Clinical parameters were within the normal limits. Haematological and biochemical parameters were in the normal range and are presented in table 2 and 3.

4.12.2 Radiographic and Endoscopic Findings

Plain radiographic examination revealed presence of sewing needle in the stomach of one dog and a pair of cool drink bottle caps together and a safety pin (Fig.43) in the stomach of the other dog. In the remaining two dogs that had swallowed radiolucent foreign bodies, plain radiographs did not show any abnormality. Endoscopic examination in both the dogs revealed presence of dog collar made up of nylon (Fig.44 and Video.11).

4.12.3 Endoscopic and Surgical Retrieval of Gastric Foreign Bodies

Endoscopic retrieval of the gastric foreign bodies was done in one dog under surgical anaesthesia induced as described in chapter III. The endoscope was introduced into the stomach while it was being insufflated until its tip was flush with the nylon dog collar that the dog has swallowed. Through the instrument channel of

the endoscope, the foreign body retrieval forceps was introduced and the jaws were opened (Fig.45). The nylon collar was grasped with the above forceps (Fig.46) and it was pulled out until its tip was at the level of the tip of the endoscope. The endoscope was slowly retracted from the stomach through the esophagus and the oral cavity while the foreign body was still being

Plate-43, 44

Plate-45, 46

held in the retrieval forceps. This ensured endoscopic retrieval of the gastric foreign body (Video.12).

In the second case of radiolucent foreign body, it turned out to be a linear foreign body. This was found to be attached to a long piece of nylon thread (Fig.47) that was causing damage to the intestinal loops at several places. The abdominal cavity was opened through standard method. This foreign body was surgically removed by performing gastrotomy and enterotomy at three places in the intestines to completely remove the foreign body. Gastrotomy and enterotomy were performed following the standard procedures using aseptic technique (Fig. 48 and 49). This led to uneventful recovery of this dog. In the other two dogs, standard gastrotomy was performed through a cranial mid ventral approach and the foreign bodies were surgically removed. The gastrotomy and the abdominal incisions were closed in a routine manner. The dogs recovered uneventfully.

4.13 GASTRIC DILATATION AND VOLVULUS (GD-V)

In the present study, a eight year Great Dane and a four year old Labrador were diagnosed to be suffering from Gastric Dilatation and Volvulus (GD-V). Physical examination revealed abdominal distention and tympany, tachycardia and hypothermia. The haematological and biochemical values were within normal limits and are presented in table 2 and 3. Plain right lateral radiograph of the abdomen showed distention of stomach with air in the fundus. The fundus was severely distended with gas with smaller gas filled pylorus located dorsally and separated from fundus by a band of soft tissue forming the double gas bubble appearance or

reverse "C" shaped fundus (Fig.50). Both the dogs recovered uneventfully following surgical decompression derotation and gastropexy.

Plate-47, 48 late-49,50

CHAPTER V

DISCUSSION

The diseases of the gastrointestinal system are most commonly encountered in canine medical practice. Vomiting, among others is a cardinal sign of the diseases affecting of the upper gastrointestinal system. In addition, there are so many other organ systems, whose diseases also cause vomiting (Guilford 1990; Lobetti 2000; Jergens 2005 and Glazer and Walters 2008). In effect, what this means is that dogs presented with vomiting as one of the complaints may actually be afflicted with disease of esophagus, stomach, kidney, liver etc., among several other diseases.

In addition to the complexity of making a definitive diagnosis of the cause of vomiting, the occurrence of regurgitation in some dogs makes pinpointing the diagnosis of the cause of vomiting all the more perplexing (Gualtieri 2001; Tams 2003 and Johnson *et al* 2009). To make the matters worse, many owners will be unable to make a distinction between vomiting and regurgitation. Both these signs will be described by the owner to the veterinarian as vomiting. A busy general practitioner may not have the ample time or the suitable means to further investigate the case.

Also, the fact that small animal hospitals are swarmed by vomiting and purging dogs at the time of outbreaks of diseases like canine Parvoviral gastroenteritis, puts tremendous pressure on the practicing veterinarians and consequently they will be unable to single out or differentiate dogs suffering from the primary esophageal or gastric diseases. Therefore, all the vomiting dogs are treated symptomatically for a few days and when they do not improve, and remain refractory efforts are initiated to investigate the underlying cause. The causes may be found to be the diseases of the gastrointestinal system, renal system etc.

Diagnosis of the primary diseases of the esophagus and stomach may require the use of plain or contrast radiography, endoscopy etc. (Johnson *et al* 1976; Crow 1985; Happe 1985; Davies 1986; Twedt 1993; Moore 2003; Sherding *et al* 1999; Michael and Goldstein 2004). However, none of the diagnostic procedures by themselves led to a definitive diagnosis. Often, a combination of the procedures may be required to conclude that a primary esophageal or gastric disease exists.

Consequent upon the above mentioned considerations, a search of the published literature was made. A perusal of the published literature revealed scanty information on the primary diseases of the esophagus and stomach. This published literature was found all the more lacking in the Indian context. Very little germane information is available on the occurrence of the primary esophageal or stomach diseases. Therefore, the present clinical study on the diseases of the esophagus and stomach in dogs with special reference to endoscopy was contemplated.

5.1 OCCURENCE OF ESOPHAGEAL AND STOMACH DISEASES

Out of many dogs that were presented to the hospitals mentioned elsewhere, in the present study, a total of 120 cases that did not respond to routine medical treatment for control of vomiting. These 120 cases were closely followed up, examined in detail and as a result of these efforts, a total of 42 cases were diagnosed to be suffering from the diseases

primarily affecting the esophagus or the stomach. The results of the present study revealed that out of the total 42 cases selected for the present study, 31dogs (73.81%) had diseases of the esophagus while the rest (11dogs; 26.19%) had diseases of the stomach. During the present study, megaesophagus was diagnosed in 18 (42.86%) dogs, reflux esophagitis in 2 dogs (4.76%), vascular ring anomalies in 2 dogs (4.76%), esophageal diverticula and esophageal nodule in one dog each (2.38% each), esophageal foreign bodies in 5 dogs (11.90%), esophageal erosion or ulcers in 2 dogs (4.76%), chronic gastritis in 3 dogs (7.14%), gastric foreign bodies in 4 dogs (9.52%), gastric erosion/ ulcer in 2 dogs (4.76%) and gastric dilatation and volvulus in 2 dogs (4.76%). The results of the present study are in agreement with the published reports of Jergens *et al* (1992); Michels *et al* (1995); Berry (2000) and Rousseau *et al* (2007). The results also showed that the occurrences of these diseases were seen in dogs ranging from two and a half months to thirteen years of age. Koc *et al* (2004) and Michael and Goldstein (2004) also observed the primary diseases of the esophagus and stomach in dogs of varying age groups. No sex wise occurrence of the esophageal or stomach diseases was reported in the literature. The present study showed that the esophageal and gastric diseases occurred in several breeds of dogs. In accordance with the reports of Jergens *et al* (1992) and Simpson (2005) who also reported these diseases which were common in several breeds of dogs.

5.2 PHYSICAL AND CLINICAL EXAMINATION

Barring certain diseases like esophageal obstruction and GD-V, all the dogs afflicted with the various diseases encountered in the present study exhibited similar symptoms. Signs like emaciation, shrunken abdomen, chronic weight loss, prominent rib cage, regurgitation, vomiting or both, etc. were also uniformly reported by several authors (Richard *et al* 1967; Gualtieri 2001; Rousseau *et al* 2007 and Glazer and Walters 2008).

5.3 MEGAESOPHAGUS

In the present clinical study, megaesophagus was recorded in 18 dogs, out of which 11 were males and 7 were females. The condition of megaesophagus was also encountered across eight different breeds of dogs indicating that the condition had no breed predisposition. While Richard *et al* (1967); Michael and Goldstein (2004) and Guilford (2005) stated that German shepherd dogs, breeds were more commonly affected with megaesophagus, Gualtieri (2001) and Michael and Goldstein (2004) reported that megaesophagus had no breed predisposition, as in the present study. The average age at which megaesophagus was diagnosed was found to be 6.25 ± 0.88 years in the present study with a range of 7 months to 13 years. Similar observations were made by Richard *et al*, (1967) and Michael and Goldstein (2004). This indicated that some of the dogs had congenital megaesophagus, although it was diagnosed at the age of 7 months in the present study. Since it is a slowly developing disease (Clifford *et al* 1971; Sherding *et al* 1999; and Johnson *et al* 2009), it was assumed that it took some months for the changes to occur and hence, by the time it was examined and treated, the pup was seven months old. No specific cause could be identified with these cases and hence, it was concluded that the disease was idiopathic. Similar observations were also made by Sherding *et al* (1999); Gualtieri (2001); Keles *et al* (2007) and Johnson *et al* (2009) although, Ross *et al* (1995) and Sherding *et al* (1999) mentioned that it is a disease related to myasthenia gravis. It is also mentioned by several authors (Barber *et al* 1983; Ross *et al* 1995; Gualtieri 2001 and Johnson *et al* 2009) that megaesophagus is a neuromuscular disorder.

The symptoms of megaesophagus recorded in the present clinical study were in concurrence with the reports of Richard *et al* (1967); Sherding *et al* (1999); Gualtieri (2001) and Johnson *et al* (2009).

Physical examination also revealed that these dogs consumed food very eagerly when offered were extremely hungry. In the present study, the time of regurgitation of undigested food was found to be immediately after to several hours after feeding and

Sherding *et al* (1999); Gualtieri (2001) and Michael and Goldstein (2004) also reported that regurgitation occurred at varying times after consumption of food. One distinguishing feature associated with regurgitation in dogs with megaesophagus and choking with a foreign body is that in the latter, regurgitation is of acute origin. This observation is in agreement with those made by Michael and Goldstein (2004).

The hematological and biochemical studies showed that all the parameters studied were with in the normal range, except for haemoglobin. Gualtieri (2001) and Jergens (2005) also reported normal levels of all the hematological and biochemical parameters. Reduced levels of hemoglobin found in the present study were assumed to be due to chronic malnutrition associated with inability to swallow the food.

The present study showed that megaesophagus could be readily diagnosed in seven out of the 18 dogs by plain radiography. Radiographic features of megaesophagus in these seven dogs were recorded to be air filled dilated esophagus, extending throughout the length of the esophagus tracheoesophageal stripe sign was consistently noticed in these seven cases. Watrous (2002) and Guilford (2005) also reported that plain radiography is not always diagnostic method of megaesophagus, but may, in some cases prompt further examination is required (Sherding *et al* 1999 and Michael and Goldstein 2004). In some cases, plain radiography may be completely misleading and these animals may appear completely normal, or in some normal healthy cases, the esophagus may appear as air filled esophagus because of aerophagia at the time of making radiographic exposure. In four dogs of the present study, megaesophagus could still be diagnosed because of discernability of gas filled esophagus in a segment that stood out against the trachea. In these dogs, however, the entire esophagus could not be delineated. This prompted further investigation of these cases that helped confirm a diagnosis of megaesophagus in the present study. Therefore, the results of the present study were in conformity with the reports of Sherding *et al* (1999) and Michael and Goldstein (2004).

Contrast radiographic examination confirmed the tentative diagnosis of megaesophagus in all the 18 dogs. This could be confirmed even in the seven dogs that did not show any radiographic signs of megaesophagus on plain radiography. These observations are in corroboration with the findings of Hoenig *et al* (1990); Sherding *et al* (1999); Gualtieri (2001); Lavin (2003); Keles *et al* (2007) and Rousseau *et al* (2007).

Ultrasonography of the cervical esophagus failed to reveal any indication of any sort of esophageal pathology in any of the dogs. Hence, it was concluded that ultrasonography was an unreliable diagnostic tool for diagnosis of primary diseases of esophagus in animals. Similarly Washababu (1996) and Kealy and McAllister (2005) stated that ultrasonography does not have a traditional role in the diagnosis of esophageal diseases, but it has proved to e useful in diagnosis of periesophageal mass or other mediastium diseases. Unlike human beings, dogs cannot be made to drink large quantities of water that helped in scanning the stomach for arriving at ultrasonic diagnosis. This is possibly the reason for unreliability of this tool in diagnosis of esophageal or gastric diseases.

The eighteen dogs examined by endoscopy revealed markedly dilated, flaccid esophagus extending from the cranial cervical region to the gastroesophageal sphincter and pooling of retained fluid, saliva and fermenting fluid in the lumen, in the most dependent segment of the esophagus. Although it may appear as if direct visualization of the esophageal of lumen by endoscopy makes it the most reliable method in megaesophagus, in reality it is not so. One problem that was always puzzling during the endoscopy is the fact that it is difficult to determine whether the esophagus appears dilated due to megaesophagus or due to its insufflation with air. Gualtieri (2001) and Jergens (2005) and also encountered some difficulty in confirming megaesophagus by endoscopy. In addition, Tams (2003) also reported that some anesthetic drugs might alter esophageal motility and mimic megaesophagus. In the present study, however, this was not found since all the normal esophagi had to be insufflated, to enable visualization. The dogs with megaesophagus, however, revealed some degree of esophageal dilation even in the mild

cases. In the severely affected dogs, the condition was confirmed since the impressions of the ribs could be seen from within the esophageal lumen at endoscopy.

To sum up, the results of the present study clearly indicated that contrast radiography of the esophagus (esophagraphy) was the most accurate and reliable method in diagnosing the presence and extent of megaesophagus. Similar observations were also made by Hoenig *et al* (1990); Sherding *et al* (1999); Gualtieri (2001); Keles *et al* (2007) and Rousseau *et al* (2007). The results of the present study are in agreement with the general impression (Michael and Goldstein 2004 and Jergens 2005) that the megaesophagus is an in curable disease. However, treatment is always aimed at alleviating suffering and attempts to improve esophageal motility and/or ability to swallow. One standard method of improving entry of the food contents from esophagus to stomach is by feeding the dogs in an upright position from an elevated platform (Hoenig *et al* 1990 and Johnson *et al* 2009).This ensures passage of food material into the stomach by gravity. The same procedure was also adopted in the present study in one of the groups and as an adjunct to medication in the other two groups (Cisapride and Metoclopramide).

The results of the present study indicated that among the three treatment regimens tested, Metoclopramide combined with feeding the dogs in an upright position from an elevated platform improved the esophageal function to the maximum extent. The number of times of regurgitation was lesser in these dogs. Improvement in the physical condition was also reported by the owners in five of the six dogs treated with Metoclopramide combined with feeding the dogs in an upright position from an elevated platform. Hoenig *et al* (1990); Michael and Goldstein (2004) and Johnson *et al* (2009) also reported similarly. However, the observations of Jergens (2005) are not in agreement with the present study, who reported cisapride to be better for this purpose in cats. This could be due to some species related variations and need further investigations. In spite of treatment, a total of

seven out of the 18 dogs either died or had to be euthanized because there was no improvement.

Post mortem examination of these dogs showed severe dilatation and thinning of the esophageal wall in al the dogs. Similar observations were also made by Richard *et al* (1967) Barber *et al* (1983) and Keles *et al* (2007). Histopathological examination of the dogs with megaesophagus showed scanty muscle bundles, infiltration of polymorphonuclear cells with submucosal congestion and enlargement of submucosal glandular pattern with epithelial irregularity. Similar observations were also made by Richard *et al* (1967) and Hoenig *et al* (1990).

In the present study, scanning electron microscopic examination of the normal esophagus showed comparatively more muscle mass and intactness of all the concerned layers of the esophagus, as was also not reported in the present literature. On the contrary, scanning electron microscopy of the esophageal tissues from dogs affected with megaesophagus revealed destruction of blood vessels, loss of normal architecture and direction of inner circular as well as outer longitudinal muscle fibers. In all the dogs, in cases of megaesophagus showed considerable destruction of the muscle mass of the esophagus. Similar findings also reported by Barber *et al* (1983) were a reduction in thickness of the mucosa, inner muscular layer (circular layer) and outer muscular layer (longitudinal layer) of colt esophagus affected with megaesophagus.

The transmission electron microscopic examination of normal esophagus showed intermyofibrillar junctions characterized by loose connectivity between the myofibril bundles. The nucleus contained dark and scanty chromatin material distributed at the periphery towards the nuclear wall whereas neuromuscular junction (myoneuronal plate) was located between the two muscle fibres with synaptic cleft and vesicles. In contrast, the transmission electron microscopic examination of dogs with megaesophagus showed dilated myofibrillar bundles, condensed nucleus, complete loss of cellular architecture, vacuolization

between muscle fiber bundles and karyolysis. Mitochondrial destruction and destruction of the neuromuscular junction when compared to the normal esophagus established the fact that megaesophagus is a neuromuscular disorder. While and Richard *et al* (1967) and Johnson *et al* (2009) also stated that megaesophagus is a neuromuscular disorder; there are very few electron microscopic studies on megaesophagus. The available literature supports the present findings (Randelia *et al* 1990; Ross *et al* 1995 and Lemasters 2009).

5.4 REFLUX ESOPHAGITIS

In present study, two cases of reflux esophagitis were diagnosed with history of anorexia, dysphagia and hyper salivation since one month. These cases resembled megaesophagus in the sense that these cases exhibited symptoms like intermittent regurgitation, and ropy blood tinged salivation. The only difference in the symptoms of megaesophagus and reflux esophagitis is the presence of extreme hunger in the former. The dogs with megaesophagus ate the food very greedily when offered, as seen during contrast radiography. All the dogs lapped up the egg mixed contrast agent readily. In contrast, both the dogs with reflux esophagitis were anorectic and showed no inclination to eat. There are no records to this effect in the published literature.

The results of the present study also revealed that the two diseases mentioned could not be differentiated from each other by clinical and laboratory examinations. Plain and contrast radiographic examination of esophagus in both the cases in this study did not evince any prominent signs suggestive of reflux esophagitis. The finding is in agreement with the observation made by Jergens (2005) and Glazer and Walters (2008).

Reflux esophagitis could only be diagnosed on endoscopy. In reflux esophagitis, endoscopic examination showed that the changes in the esophagus were limited to the caudal esophagus, in contrast to megaesophagus. Endoscopic observations of mucosal erythema, haemorrhage, mucosal irregularity, erosion with thickened mucosal folds especially limited to the lower esophageal area helped confirm reflux esophagitis. Similar

observations were also made by Sherding *et al* (1999); Guilford (2005); Gualtieri and Olivero (2006); Glazer and Walters (2008) and Meineri *et al* (2008). Shaheen and Ransohoff (2009); the results also showed that both the dogs recovered due to the treatment adopted. Jergens (2005) and Shaheen and Ransohoof (2009) also suggested similar treatment.

5.5 ESOPHAGEAL NODULE

In the present study, a nine year old male Pomeranian dog presented with history of recurrent cough and retching and esophagoscopy revealed lemon sized nodular growth appearing as broad based protuberance with nipple like orifice located just caudal to the base of the heart. The typical intraluminal esophageal nodule with nipple like orifice could be due to the Spirocerci lupi infestation. Similar clinical and endoscopic findings were observed by Gualtieri (2001); Fossum and Hedlund (2002); Head *et al* (2002); Tams (2003); Yildirin *et al* (2007) and Mylongkis *et al* (2008). In the present study the owner was refused any surgical exploration and biopsy were not collected for accurate confirmation disease condition as dog was not available or further investigation.

5.6 ESOPHAGEAL FOREIGN BODIES

In the present study, out of 42 cases, five dogs (11.90%) were reported with history of sudden onset of dysphagia, salivation, anorexia, gagging, excessive salivation, cough reflexes and uneasiness after a meal. Similar clinical signs were also observed by Tams (2003) and Jergens (2005) and Weissberg (2008) in the dogs with esophageal foreign body.

In the present clinical study, the esophageal foreign bodies were one pork bone, three chicken bones (radiopaque) and one socks (radiolucent) lodged into the post pharyngeal esophagus, caudal thoracic and cervical part of the esophagus, respectively. While Michels *et al* (1995); Jergens (2005) and Weissberg (2008) reported fishhook, chicken bone, pork bone, leather piece and needle as common esophageal foreign bodies in dogs. The most common sites of obstruction of esophageal foreign bodies were thoracic inlet, the base of

heart and the hiatus of the diaphragm (Sherding *et al*, 1999; Griffiths *et al*, 2000; Gualtieri, 2001; Fossum and Hedlund, 2002 and Watrous, 2002).

Plain radiography was useful in confirmation of esophageal obstruction in all the cases. Michels *et al* (1995); Fossum and Hedlund (1999); Lamb (1999); Fossum and Hedlund (2002); Meyer and Levine (2008) and Ponnuswamy *et al* (2009) also reported that plain radiography was useful in diagnosis of esophageal foreign bodies.

Treatment of these five dogs with esophageal obstruction due to foreign bodies was treated by various means. The dog with socks in the cervical esophagus vomited the socks out and was relieved of obstruction due to the emetic effect of xylazine even before the dog could be anaesthetized. It is not uncommon for the dogs to vomit out certain foreign bodies. In the dog with the osseous foreign body located just caudal to the pharynx, it was retrieved orally using a long cheatle forceps passed along the endoscope and was removed under endoscopic illumination. Meyer and Levine (2008) and Ponnuswamy *et al* (2009) also recommended similar maneuvers for retrieval of esophageal foreign bodies. In three dogs, the osseous foreign bodies were pushed into the stomach and once this was done, the dogs were symptom free and recovered uneventfully. Similarly Gualtieri (2001) and Chen *et al* (2008) also used push technique for caudal esophageal foreign bodies into the stomach with success rate greater than 95%. It is assumed from the present study that once the osseous material is dislodged into the stomach, it is decalcified by the gastric acid and is digested. All three dogs were verified by abdominal radiograph for assessment of dissolution of esophageal foreign bodies into the stomach. This could be the cause of immediate recovery in these dogs**.** Gualtieri (2001) also made similar observations.

5.7 VASCULAR RING ANOMALIES

In the present study, two dogs were presented with history of regurgitation, debility and generalized weakness. However, history revealed that both the dogs started

regurgitation from the time of weaning. Similar clinical observations were made by Fossum and Hedlund (2002); Koc *et al* (2004) and Saini *et al* (2009).

A barium swallow revealed stricture of esophagus over the heart base and cranial esophageal dilatation. While Clifford et al (1971); Nelson and Coute (1998); Sherding *et al* (1999); Gualtieri (2001); Lavin (2003); Jergens 2005; Keles *et al* (2007) and Saini *et al* (2009) reported the similar contrast radiographic signs of vascular ring anomalies in dogs.

In the present study both the dogs consumed food very eagerly when offered and appeared extremely hungry because of good health. Similarly Haverly (2003) also reported that inspite of good health shorthaired cat regurgitated since weaning. In the present study, esophagoscopy was not done because the dogs were extremely weak and not fit for even mild anesthesia and eventually died after some period. Although as part of medical management the both dogs were administered semisolid to liquid food in upright position, they eventually died after some time.

Post mortem findings in both the dogs showed severe and flaccid distension of the esophagus cranial to the constrictive vascular band with recently consumed food. Closer examination of the esophagus also clearly showed the area of constriction caused by vascular band. In one of the dogs, minute nodules in the gastric wall were observed and hypertrophied rugae with generalized congestion of gastric mucosa were observed which are in accordance with the published literature (Clifford *et al* 1971; Tams 2003). Histopathological examination of the esophageal wall revealed enlarged mucous glands, congestion of submucosa and cellular infiltration whereas the stomach wall also showed patchy foci fibrosis with infiltrates of lymphocytes, plasma cells and neutrophils indicating concurrent mild diffuse sub acute gastritis in addition to esophageal distension.

5.8 ESOPHAGEAL DIVERTICULUM

Three year old female German shepherd dog presented with chronic and postprandial regurgitation. Physical and clinical examination revealed moderate dehydration, weakness and anemia. Similarly Sherding *et al* (1999) and Jergens (2005) reported the same clinical symptoms in esophageal diverticula. Lateral plain radiography revealed sac like out pouching of cranial thoracic esophagus that rested on the sternum. Similar observations were also made by Sherding *et al* (1999); Gualtieri (2001); Fossum and Hedlund (2002); Jergens (2005) and Meyer and Levine (2008).

Endoscopy confirmed the diagnosis of esophageal diverticulum with sac like out pouching of esophageal wall and presence of partially digested food particles. Similar endoscopic findings were also made by Gualtieri (2001); Fossum and Hedlund (2002) and Jergens (2005). No treatment could be initiated in this case since the owner was reluctant for surgery.

5.9 ESOPHAGEAL EROSION/ULCERS

The two dogs with vomiting of about three weeks duration that did not respond to routine medication were diagnosed to be suffering from esophageal ulcers, both by endoscopy and by positive contrast radiography. The contrast esophagram showed remnants of barium sulphate still to the mucosa of the cervical part of esophagus suggestive of loss of mucosal integrity and filling defects. Similar observation also were made by Crow (1985); Gualtieri *et al* (1999); Hall and Washabau (1999) and Lamb (1999).

Endoscopically, both the dogs showed friable mucous membrane, petechial haemorrhage and erosion of the esophageal mucosa. While Jergens *et al* (1992); Sherding *et al* (1999); Gualtieri (2001) and Jergens (2005) also observed similar endoscopic lesions in case of esophageal erosion and ulcers. Both the dogs responded well to the treatment

adopted. By one week after initiation of treatment, the dogs stopped vomiting and became normal mentioned in chapter-II. Similarly Jergens (2005) and Fossum and Hedlund (2007) were treated with same line of treatment.

5.10 CHRONIC GASTRITIS

In present study, three (7.14%) dogs with chief complaint of chronic vomiting, anorexia and loss of body weight since one month were recorded. Similar clinical observation also made by Dennis *et al* (1992); Baez *et al* (1999) and Simpson (2005).

Gastroscopy of all three dogs revealed generalized congestion as well as petechial hemorrhagic spots on gastric mucosa with minor areas of mucosal erosion suggestive of chronic gastritis. However, Tams (1999); Gualtieri (2001) and Simpson (2005) and Fossum and Hedlund (2007) reported same endoscopic observations.

Cytological examination of stomach fluid collected during endoscopy also showed large cocci and rod shaped bacteria along with the presence of lymphocytes indicating gastritis. Colonization of non-specific bacteria in the stomach contents further confirmed the diagnosis of chronic gastritis. Such non specific organisms are also implicated to be the cause of chronic gastritis by several authors (Guilford 1990; Guilford 2005 and Akhtardanesh *et al* 2007 and Stromar *et al* 2008). On the other hand, Akhtardanesh *et al* 2007 and Stromar *et al* 2008 stated that either *Helicobacter or Helicobacter* Like Organisms (HLO) was generally found to be causing chronic gastritis in their studies. In the present study, no *Helicobacter or Helicobacter* like organisms was encountered.

These three dogs recovered uneventfully to the response of combination of amoxicillin @ 20mg/kg body weight twice daily with Clarithromycin[1] (7.5 mg/kg body weight orally twice a day) and Metronidazole[2] (10mg/kg body weight orally twice a day) for two weeks. Ranitidine was given at the rate of 0.5 mg/kg body weight, orally for 14 days.

However, Roudebush and Delivorias (1985); Strauss-Ayali and Simpson (1999) and Simpson (2005) also treated with similar medical treatment in cases of chronic gastritis.

5.11 GASTRIC EROSION/ULCERS

In the present study, two dogs with history of vomiting and weakness since one and half months were diagnosed to be affected with gastric erosion/ulcers. Esophagoscopy revealed gastric ulcerative patches with marked thickening and irregular edges. Erosion or ulcers were characterized by slightly depressed areas with focal streaks of haemorrhage. Similar clinical observations also were made by Gualtieri *et al* (1999); Boston *et al* (2003); Simpson (2005) and Neiger (2009). However, the endoscopic observation were made by Dowdle (2005); Guilford (2005); Fossum and Hedlund (2007) and Luna *et al* (2007).

The results also showed that both the dogs responded well to complete recovery due to treatment protocol adopted. Similarly, Simpson (2005) and Fossum and Hedlund (2007) also used same treatment protocol for management of gastric erosion or ulcers in dogs.

5.12 GASTRIC FOREIGN BODIES

In present clinical study, four dogs were reported with chief complaint of persistent vomiting, dehydration and inappetence since five to seven days. Physical examination of abdominal area revealed intense pain, discomfort, uneasiness and loss of appetite. Clinical and haematological parameters were within the normal limits. Similar observations also were made by Michels *et al* (1995) and Lamb (1999) in cases of dogs with gastric foreign bodies.

In the present study, two dogs that underwent plain radiographic examination revealed presence of sewing needle in the stomach of one dog and a pair of metal caps of cool drink bottle and a safety pin in another dog. Similarly Michels *et al* (1995); Fossum and Hedlund (1999) and Dilip Kumar and Ameerjan (2001) also found various gastric foreign

bodies such as fish hook, plastic toys, cooking bags, tile pieces, black stones, chicken bones, sewing needle and mango turnips. Hence plain radiography in the present study for diagnosis of gastric foreign bodies becomes excellent and confirmative tool. Removal of these foreign bodies was considered to be too risky by endoscopy due to the possibility of esophageal tearing. Fossum and Hedlund (1999) and Simpson (2005) also stated that endoscopic retrieval of gastric foreign bodies was not always possible due to similar reason. There fore gastrotomy was considered as a safer mean of removal of these foreign bodies in two out of four cases in present study.

In the remaining two dogs that swallowed dog nylon belt, plain radiograph did not confirm their presence. Endoscopic examination in these dogs revealed presence of dog collar made up of nylon in the stomach. Similarly, Johnson *et al* (1976); Crow (1985); Happe (1985); Davies (1986) and Twedt (1993) and Moore (2003) also reported that, gastroscopy is a useful, safe, direct, accurate and confirmative diagnostic tool for identifying the gastric foreign bodies in dogs.

Endoscopic retrieval of the nylon belt in the stomach was done in one dog under surgical anesthesia. In the second case of radiolucent foreign body (another nylon belt), it turned out later on to be a linear foreign body. This was found to be attached to a long piece of nylon thread traversed in to the small intestine loops. Hence, the linear foreign body was removed by standard gastrotomy and enterotomy operation. Similarly Michels *et al* (1995); Fossum and Hedlund (1999) and Dilip Kumar and Ameerjan (2001) also advised removal of the gastric foreign bodies those were not possible to be retrieved through endoscopic method, by following standard gastrotomy and enterotomy procedures. All the dogs recovered uneventfully.

5.13 GASTRIC DILATATION AND VOLVULUS (GD-V)

In the two dogs with gastric dilatation and volvulus (GD-V) in the present study, the clinical and radiographic signs and the clinical out come of their treatment with surgical

derotation and gastropexy compared well with the published reports (Fossum and Hedlund 1999; Eggertsdottir *et al* 2001 and Simpson 2005).

5.14 CONCLUSIONS

The following conclusions were drawn from the results of the present clinical study of the diseases of esophagus and stomach with special reference to endoscopy in dogs:

1. The present study revealed that 35% of dogs (42 out of 120 dogs) refractory to general medical treatment for vomiting or regurgitation were appeared are to be affected with primary diseases of esophagus and stomach.

2. The most common malady affecting the esophagus in dogs was megaesophagus (18), and it was well managed by using metoclopramide and feeding the dogs in an upright position from an elevated platform. The disease is considered to be a neuromuscular disease as seen during scanning and transmission electron microscopy. The neuromuscular junctions as seen by transmission electron microscopy appeared to be destroyed in cases of megaesophagus.

3. Esophagography was found to be more useful procedure in confirming the cases of megaesophagus than endoscopy.

4. On endoscopy examination, a large cavernous lumen extending the length of the esophagus is vividly suggestive of megaesophagus

5. The other diseases encountered in the present study in their order frequency of occurrence are esophageal foreign bodies (5), gastric foreign bodies (4), chronic gastritis (3), reflux esophagitis (2), esophageal erosion/ulcer (2), gastric erosion/ ulcer (2), vascular ring anomalies (2), gastric dilatation and volvulus (2), esophageal nodule (1) and esophageal diverticulum (1).

6. Endoscopy proved to be a valuable and useful aid in diagnosis of esophageal and gastric ulcers/ erosions, esophageal nodule and esophageal diverticulum.

7. Endoscopy is not always useful in treatment of esophageal or gastric foreign bodies in dogs. For the shape and size of foreign body, duration of obstruction and nature and location of foreign body in the esophagus and stomach determine the use and success of endoscopy for therapeutic purpose.

8. Albeit veterinarians are still in the early phase of adoption of minimally invasive procedures, endoscopies prove to be vital diagnostic and therapeutic tools of the future veterinarian's armamentarium.

CHAPTER VI

SUMMARY

The present study was undertaken in 42 cases out of the 120 clinical cases of dogs presented with diseases of esophagus and stomach with the history of persistent vomiting or regurgitation to the Department of Veterinary Surgery and Radiology, College of Veterinary Science, Rajendranagar, Hyderabad and Teaching Veterinary Clinical Complex, Bhoiguda, Secunderabad.

A total of 120 dogs were presented with the chief complaint of recurrent vomiting or regurgitation. These cases were examined and attempts were made to treat them symptomatically by medical means. The cases that responded to routine medical treatment

and those cases of severe haemorrhagic gastroenteritis like Canine Parvovirus gastroenteritis were excluded and did not form a part of the present clinical study. Out of the total of 120 cases selected for closer examination, 78 dogs (65%) responded to routine medical treatment with antiemetics, antibiotics and fluid therapy and hence, they were excluded from the purview of the present clinical study. The remaining 42 dogs (35%), which continued to vomit or regurgitate for more than one week and showed signs consistent with the primary diseases of the esophagus and stomach formed the patient material for the present clinical study.

All the 42 clinical cases of dogs underwent routine physical, clinical, hematological and biochemical examinations as well as plain and contrast radiographic, Ultrasonographic and endoscopic assessment to rule out the concurrent diseases prior to the consideration in the present study. The results of the present study revealed that out of the total 42 cases selected for the present study, 31dogs (73.81%) had diseases of the esophagus while the rest (11dogs; 26.19%) had diseases of the stomach. During the present study, megaesophagus was diagnosed in 18 (42.86%) dogs, reflux esophagitis in 2 dogs (4.76%), vascular ring anomalies in 2 dogs (4.76%), esophageal diverticula and esophageal nodule in one dog each (2.38% each), esophageal foreign bodies in 5 dogs (11.90%), esophageal erosion or ulcers in 2 dogs (4.76%), chronic gastritis in 3 dogs (7.14%), gastric foreign bodies in 4 dogs (9.52%), gastric erosion/ ulcer in 2 dogs (4.76%) and gastric dilatation and Volvulus in 2 dogs (4.76%). The results also showed that the occurrences of these diseases were seen in dogs ranging from two and a half months to thirteen years of age. No sex wise occurrence of the esophageal or stomach diseases was reported. All the dogs afflicted with the various diseases exhibited similar symptoms like emaciation, shrunken abdomen, chronic weight loss, prominent rib cage, regurgitation, vomiting or both.

In the present clinical study, 18 clinical cases of megaesophagus were recorded. The disease was encountered in Labrador (2 dogs), Golden Retriever (2 Dogs), German Shepherd

(5 Dogs), Doberman (2 Dogs), Mongrel (3 Dogs), Pomeranian (2 Dogs), Cocker Spaniel (1 Dog) and Boxer (1 Dog) breeds of dogs. The mean age of occurrence was found to be 6.25 ± 0.88 years. Out of these 18 dogs, 11were found to be males (61.12 %) and the rest were females (7 dogs; 38.88%).

The eighteen dogs with megaesophagus showed symptoms of emaciation hide bound condition, shrunken abdomen, prominent rib cage and slight to moderate pain and discomfort at cervical area on palpation. Physical examination also revealed that these dogs consumed food very eagerly when offered and appeared extremely hungry. However, all these 18 dogs showed the signs of regurgitation of the food soon after food consumption. Regurgitation was reported to have occurred immediately after to several hours after feeding. The regurgitated material was reported to be undigested food. The hematological and biochemical studies showed that all the parameters studied were with in the normal range, except for haemoglobin.

Ultrasonography of the cervical esophagus failed to reveal any indication of any sort of esophageal pathology in any of the dogs. Radiographic features of megaesophagus in these seven dogs were recorded to be air filled dilated esophagus, extending throughout the length of the esophagus. Tracheoesophageal stripe sign was consistently noticed in these seven cases. Esophagography clearly revealed generalized distention of esophagus in all the 18 dogs. The eighteen dogs examined by endoscopy revealed markedly dilated, flaccid esophagus extending from the cranial cervical region to the gastroesophageal sphincter and pooling of retained fluid, saliva and fermenting fluid in the lumen, in the most dependent segment of the esophagus.

The results of the present study indicated that among the three treatment regimens tested, Metoclopramide combined with feeding the dogs in an upright position from an elevated platform improved the esophageal function to the maximum extent. The number of times of regurgitation was lesser in these dogs. Improvement in the physical condition

was also reported by the owners in five of the six dogs treated with Metoclopramide combined with feeding the dogs in an upright position from an elevated platform.

In spite of treatment, a total of seven out of the 18 dogs either died or had to be euthanized because there was no improvement. Post mortem examination of these dogs showed severe dilatation and thinning of the esophageal wall in al the dogs. Histopathological examination of the dogs with megaesophagus showed scanty muscle bundles, infiltration of polymorphonuclear cells with submucosal congestion and enlargement of submucosal glandular pattern with epithelial irregularity. Scanning electron microscopy of the esophageal tissues from dogs affected with megaesophagus revealed destruction of blood vessels, loss of normal architecture and direction of inner circular as well as outer longitudinal muscle fibers. In all the cases of megaesophagus showed considerable destruction of the muscle mass of the esophagus. The transmission electron microscopic examination of dogs with megaesophagus showed dilated myofibrillar bundles, condensed nucleus, complete loss of cellular architecture, vacuolization between muscle fiber bundles and karyolysis. Mitochondrial destruction and destruction of the neuromuscular junction when compared to the normal esophagus established the fact that megaesophagus is a neuromuscular disorder.

Two clinical cases of dogs were presented with history of anorexia, dysphagia and hypersalivation since one month. Physical examination did not point to any tentative diagnosis. Clinically, intermittent regurgitation, anorexia, frothy and ropy blood tinged salivation was noticed in both the dogs. The haematological and biochemical parameters were within normal limits. Plain and contrast radiography in these dogs did not reveal any abnormality. Endoscopic examination was characterized by mucosal erythema, haemorrhage, mucosal irregularity, erosion with thickened mucosal folds especially at the lower esophageal area. Endoscopic examination showed the evidence of reflux esophagitis in these two dogs. Both the dogs recovered uneventfully following the treatment adopted.

A nine year old male Pomeranian dog presented with history of recurrent cough and retching. Esophagoscopy revealed a lemon sized nodular growth appearing as broad based protuberance with nipple like orifice located just caudal to the base of the heart. A small lesion resembling an opening also noticed. Esophagoscopy helped locate the position of the nodule, since it was found just caudal to the area where the movements of the base of the heart could be seen. The owner refused any surgical exploration since the symptoms were mild and the dog was not available for further evaluation.

In the present study, out of 42 cases, five dogs (11.90%) were reported with history of sudden onset of regurgitation, dysphagia, anorexia, odonyphagia, retching, gagging, excessive salivation, cough reflexes and uneasiness after a meal. Plain lateral radiographs of the neck and thorax revealed the presence of osseous foreign bodies in the esophagus of four dogs. In one dog, the osseous foreign body was located just caudal to the pharynx and in the other three, in the mid thoracic region. In another dog, the lateral cervical radiograph revealed the presence of a radiolucent foreign body, which later turned out to be socks.

Treatment of these five dogs with esophageal obstruction due to foreign bodies was treated by various means. The dog with socks in the cervical esophagus vomited the socks out and was relieved of obstruction due to the emetic effect of xylazine even before the dog could be anaesthetized. In the dog where the osseous foreign body was located just caudal to the pharynx, the same was retrieved orally using a long cheatle forceps passed along the endoscope and was removed under endoscopic illumination. In the three dogs where the osseous foreign bodies were located in the thoracic region, by endoscopy, the bone pieces could be pushed back into the stomach by the endoscope itself. Once this was done, the dogs were symptoms free and recovered uneventfully.

The present study, two dogs aged between two and a half months to three months were presented with history of regurgitation, debility and generalized weakness. Positive contrast radiography with barium sulphate paste revealed stricture of esophagus over the

heart base and cranial esophageal dilatation. Esophagoscopy was not done because the dogs were extremely weak and not fit for even mild anaesthesia. Both the dogs died soon after despite intravenous nutrition in an attempt to make them fit for anaesthesia and surgery. Necropsy revealed severe and flaccid distension of the esophagus cranial to the constrictive vascular band with recently consumed food. Closer examination of the esophagus also clearly showed the area of constriction caused by vascular band. Histopathological examination of the esophageal wall revealed enlarged mucous gland, congestion of submucosa and cellular infiltration whereas the stomach wall showed patchy foci fibrosis with infiltrates of lymphocytes, plasma cells and neutrophils indicating concurrent mild diffuse subacute gastritis in addition to esophageal dilation.

Three year old female German shepherd dog presented with chronic and postprandial regurgitation. Physical and clinical examination revealed moderate dehydration, weakness and anaemia. Haematological and biochemical values were within normal limits. Lateral plain radiography revealed sac like out pouching of cranial thoracic esophagus that rested on the sternum. Endoscopy confirmed the diagnosis of esophageal diverticulum with sac like out pouching of esophageal wall and presence of partially digested food particles. No treatment could be initiated in this case since the owner was reluctant for surgery.

The two dogs with vomiting of about three weeks duration that did not respond to routine medication were diagnosed to be suffering from esophageal ulcers, both by endoscopy and by positive contrast radiography. The contrast esophagram showed remnants of barium sulphate still to the mucosa of the cervical part of esophagus suggestive of loss of mucosal integrity and filling defects. Endoscopically, both the dogs showed friable mucous membrane, petechial haemorrhage and erosion of the esophageal mucosa. By one week after initiation of treatment, the dogs stopped vomiting and became normal.

Gastroscopy of all three dogs revealed generalized congestion as well as petechial hemorrhagic spots on gastric mucosa with minor areas of mucosal erosion suggestive of

chronic gastritis. Cytological examination of stomach fluid collected during endoscopy also showed large cocci and rod shaped bacteria along with the presence of lymphocytes indicating gastritis. Colonization of non-specific bacteria in the stomach contents further confirmed the diagnosis of chronic gastritis. No *Helicobacter* Like Organisms (HLO) was encountered. These three dogs recovered uneventfully to the treatment adopted in the present study.

Two dogs were diagnosed to be affected with gastric erosion/ulcers. Esophagoscopy revealed gastric ulcerative patches with marked thickening and irregular edges. The results showed that both the dogs responded well to complete recovery due to treatment protocol adopted.

In present clinical study, four dogs were diagnosed to be affected with gastric foreign bodies. Out of four cases, two dogs that underwent plain radiographic examination revealed presence of sewing needle in the stomach of one dog and a pair of metal caps of cool drink bottle and a safety pin in another dog. Hence plain radiography in the present study for diagnosis of gastric foreign bodies becomes excellent and confirmative tool. Removal of these foreign bodies was considered to be too risky by endoscopy due to the possibility of esophageal tearing. There fore gastrotomy was considered as a safer mean of removal of these foreign bodies in two out of four cases in present study. In the remaining two dogs that swallowed dog nylon belt, plain radiograph did not confirm their presence. Endoscopic examination in these dogs revealed presence of dog collar made up of nylon in the stomach. Endoscopic retrieval of the nylon belt in the stomach was done in one dog under surgical anesthesia. In the second case of radiolucent foreign body (another nylon belt), it turned out later on to be a linear foreign body. This was found to be attached to a long piece of nylon thread traversed in to the small intestine loops. Hence, the linear foreign body was removed by standard gastrotomy and enterotomy operation. All the dogs recovered uneventfully.

In the two dogs with gastric dilatation and volvulus (GD-V) in the present study, the clinical and radiographic signs helped in confirmation of the disease and the clinical out come of their treatment with surgical derotation and gastropexy was uneventful.

The following conclusions were drawn from the results of the present clinical study of the diseases of esophagus and stomach with special reference to endoscopy in dogs:

9. The present study revealed that about 35% of dogs (42 out of 120 dogs) that were refractory to general medical treatment for vomiting or regurgitation were appeared to be affected with primary diseases of esophagus and stomach.

10. The most common malady affecting the esophagus in dogs was found to be megaesophagus (18), and this can be reasonably well managed by using metoclopramide and feeding the dogs in an upright position from an elevated platform. The disease is considered to be a neuromuscular disease as seen during scanning and transmission electron microscopy. The neuromuscular junctions as seen by transmission electron microscopy appeared to be destroyed in cases of megaesophagus.

11. Esophagography was found to be more useful procedure in confirming the cases of megaesophagus than endoscopy. This is because it is sometime difficult in mild cases to be sure whether the esophagus was really dilated or it appeared so due to insufflation during endoscopy.

12. On endoscopy examination, a large cavernous lumen extending the length of the esophagus is vividly suggestive of megaesophagus. But a normal esophagus can appears flaccid and distended under anesthesia.

13. The other diseases encountered in the present study in their order frequency of occurrence are esophageal foreign bodies (5), gastric foreign bodies (4), chronic gastritis (3), reflux esophagitis (2), esophageal erosion/ulcer (2), gastric erosion/

ulcer (2), vascular ring anomalies (2), gastric dilatation and volvulus (2), esophageal nodule (1) and esophageal diverticulum (1).

14. Endoscopy is not always useful in treatment of esophageal or gastric foreign bodies in dogs. The shape and size of foreign body, duration of obstruction and nature and location of foreign body in the esophagus and stomach determine the use and success of endoscopy for therapeutic purpose.

15. Endoscopy proved to be a valuable and useful aid in the diagnosis of esophageal and gastric ulcers/ erosions, esophageal nodule and esophageal diverticulum.

16. Albeit veterinarians are still in the early phase of adoption of minimally invasive procedures, endoscopy proves to be vital diagnostic and therapeutic tool of the future veterinarian's armamentarium.

LITERATURE CITED

Akhtardanesh B, Jamshidi S, Sasani F, Mohammadi A, Bokaee S and Saheli T Z 2006 Helicobacter spp. Infection and gastric lesions in domestic and stray cats. Veterinarski Arhiv 76 (6): 479-488.

Baez J L, Hendrick M J, Walker L M and Washabau R J 1999 Radiographic, ultrasonographic and endoscopic findings in cats with inflammatory bowel disease of the stomach and small intestine: 33 cases (1990-1997). Journal of American Veterinary Medical Association 215 (3):349-354.

Barber S M, McLaughlin B G and Fretz P B 1983 Esophageal Ectasia in a Quarter horse Colt. Canadian Veterinary Journal 24:46-49.

Baron T H 2008 Clinical recognition and endoscopic retrieval of misplaced endotracheal tubes. Gastroenterology and Hepatology 4(4): 282.

Beitzel C and Brinker S 1956 Removal of esophageal foreign body which had penetrated the trachea. Journal of American Veterinary Medical Association 1:241-242.

Berry W L 2000 Spirocerci lupi esophageal granulomas in 7 dogs: resolution after treatment with doramectin. Journal of Veterinary Internal Medicine 14(6): 609-612.

Boothe D M 1999 Gastrointestinal pharmacology. Veterinary Clinics of North America Small Animal Practice 29 (2):343-376.

Borer L R, Peel J E, Seewald W, Schawalder P and Sperng D E 2003 Effect of carprofen, etodolac, meloxicam or butorphenol in dogs with induced acute synovitis. Australian Journal of Veterinary Research 64 (11):1429-1437.

Boston S E, Moens N M, Kruth S A and Southorn E P 2003 endoscopic evaluation of the gastroduodenal mucosa to determine the safety of the concurrent administration of meloxicam and dexamethasone in healthy dogs. American Journal of Veterinary Research 64(11):1369-75.

Brunnet S R, Dee L A, Herron A J J and Altman N H 1992 Gastric extramedulary plasmcytoa in a dog. Journal of American Veterinary Medical Association 200:1501-1502.

Chen Y H, Liao W C, Hou M C, Lin H C and Lee S D 2008 Esophageal food impaction: A homemade suction tube attached to esophagogastroduodenoscopy for food bolus removal. Journal of China Medical Association 71(12): 635-638.

Chiang K H and Chou A S B 2005 Imaging of a Gastrointestinal Foreign body in a Feline-A Case Report. Tzu. Chi Medical Journal 17(3):187-189.

Clifford D H, Soifer F K, Wilson C F, Waddell E D and Guilloud G L 1971 Congenital Achalasia of the Esophagus in Four Cats of Common Ancestry. Journal of American Veterinary Medical Association 158 (9):1554-1560.

Crow S E 1985 Tumours of the Alimentary Tract. Veterinary Clinics of North America Small Animal Practice 15 (3):577-596.

Culling C F A 1957 Handbook of Histopathological Technique. Landon Butterworth and Co (Publishers) Ltd.

Davies J V 1986 Small Animal Endoscopy British Medical Bulletin 42: 330-332.

Day M J, Bilzer T, Mansell, Wilcock B, Hall E J, Jergens A, Minami T, Willard M and Washabau R. 2008 Histopathological standards for the diagnosis of gastrointestinal inflammation in endoscopic biopsy samples from the dog and cat: a report from the World Small Animal Veterinary Association Gastrointestinal Standardization Group. Journal of Comparative Pathology 138 (1): 51-43.

Den H E 2003 Endoscopic removal of foreign bodies from cats or dogs. Tijdschr Diergeneeskd 128 (14 - 15):434 – 439.

Dennis F, Megraud F and Brassens R 1992 Histology of gastritis and Helicobacter Pylori infection in Dog. Journal of Medical Association Thai. 75: 386-392.

Kumar D and Ameerjan K 2001 Foreign bodies in caudal oesophago-gastric region and pyloric stenosis in dogs. Indian Veterinary Journal 78:426-427.

Dowdle S M 2005 The prevalence of subclinical gastroduodenal ulceration in dachshunds with intervertebral disc prolapse. Thesis submitted to University of Pretoria, Onderstepoort.

Dutta S K, Arora M, Kireet A, Bashandy H and Gandsas A 2008 Upper gastrointestinal symptoms and Associated Disorders in morbidly obese patients: A prospective study. Digestive Disease Science 31.

Eggertsdottir A V, Stigen O, Lonaas L, Langeland M, Devor M, Vibe-Petersen G and Eriksen T 2001 Comparison of the Recurrence Rate of Gastric Dilatation with or Without in Dogs After Circumcostal Gastropexy Verses Gastrocolopexy. Veterinary Surgery 30: 546-551.

Ettinger S E and Feldman E 2005 Text Book of Small Animal Veterinary Medicine, sixth edition, Elsevier Saunders. St. Louis, Missouri.Pp.1321-1326.

Fossum T W and Hedlund C S 1999 Surgery of the digestive system Pp 339-530. *In* Small Animal Surgery (Edi. Fossum T W) Mosby Elsevier St. Louis, Missouri, USA.

Fossum T W and Hedlund C S 2002 Surgery of the digestive system Pp 339-530. *In* Small Animal Surgery (Edi. Fossum T W) Mosby Elsevier St. Louis, Missouri, USA.

Fossum T W and Hedlund C S 2005 Surgery of the digestive system Pp 339-530. *In* Small Animal Surgery (Edi. Fossum T W) Mosby Elsevier St. Louis, Missouri, USA.

Fossum T W and Hedlund C S 2007 Surgery of the digestive system Pp 339-530. *In* Small Animal Surgery (Edi. Fossum T W) Mosby Elsevier St. Louis, Missouri, USA.

Frappier B L 2001 Digestive System pp: 170-211. In Dellmanns Textbook of Veterinary Histology (6^{th} edition) Blackwell Publishing.

Glazer A and Walters P 2008 Esophagitis and Esophageal Strictures. Compendium, Pp 281-291.

Gomez N A, Alvarez LR, Mite R, Andrade J P, Alvaraz J R and Vargas P L 2000 Percutaneous endoscopic gastrostomy (GEP) designed reusable (NAGEP) for one time endoscopic time: experimental study in dogs. Acta Gastroenterology Latinoam 30(4):233-5.

Griffiths L G, Tiruneh R, Sullivan M and Reid S W J 2000 Oropharyngeal Penetrating Injuries in 50 dogs: A Retrospective Study. Veterinary Surgery 29: 383-388.

Gualtieri M 2001 Esophagoscopy: In Endoscopy, Veterinary Clinics of North America Small Animal Practice 31 (4):605-630.

Gualtieri M and Olivero D 2006 Reflux Esophagitis in Three Cats Associated With Metaplastic Columnar Esophageal Epithelium. Journal of the American Animal Hospital Association 42: 65-70.

Gualtieri M, Monzeglio M G and Scanziani E 1999 Gastric neoplasia *In* Progress in Gastroenterology. Veterinary Clinics of North America Small Animal Practice 29(2):415-439.

Guilford W G 1990 Upper Gastrointestinal Endoscopy. *In* Veterinary Endoscopy. Veterinary Clinics North America Small Animal Practice 20 (5):1209-1227.

Guilford W G 1999 Idiopathic Inflammatory Bowel disease. Veterinary Clinics North America Small Animal Practice 29:501-521.

Guilford W G 2005 Upper Gastrointestinal Endoscopy. *In* Veterinary Endoscopy for the Small Animal Practitioner Elsevier Saunders Pp.279-321.

Guilford W G and Stromback G R 1996 Small Animal Gastroenterology: Third Edition, W.B.Saunders Philadelphia, Pp.286.

Hall J A and Washabau R J 1999 Diagnosis and treatment of gastric motility disorders. Veterinary Clinics of North America Small Animal Practice 29 (2):377-395.

Hall J E 2008 Flexible endoscopy: Upper gastrointestinal tract Pp:42-47 *In* BSAVA manual of canine and feline endoscopy and endosurgery (Edited by Lhertte and Sebel) British Small Animal Association, England.

Hamir A N 1986 Oesophageal perforation and pyothorax associated with *Spirocerci lupi* infestation in a dog. Veterinary Record 119:276.

Happe R P 1985 Gastrointestinal endoscopy in the dog Veterinary Q 7(3): 231:234.

Happe R P, Vanden Brom W E and Vander Gaag I 1982 Duodenogastric reflux in the dog a clinicopathological study. Research in Veterinary Science 33(3):280-286.

Harvey H J 2008 Pyloric stenosis in a female boxer puppy. Senior Seminar Paper. Cornell University College of Veterinary Medicine Pp: 1-16.

Haverly C P 2003 vascular Ring Anomaly Resulting in Esophageal Stricture in a Cat. Senior Seminar Paper, Cornell University College of Veterinary Medicine.

Head K W, Else R W and Dubielzig R R 2002 Tumours of the alimentary tract 401-482. In Tumours in Domestic Animals (4th Edition, Meuten D L) Iowa State, Blackwell Publishing Company.

Hoenig M, Mahaffey M B, Parnell P G and Styles M E 1990 Megaesophagus in two cats. Journal of American Veterinary Medical Association 196 (5): 663-665.

Jergens A E 2005 Diseases of the Esophagus Pp:1298-1310 In Textbook of Veterinary Internal Medicine (6th Edition by Ettinger and Feldman) Elsevier Saunders St Louis Missouri.

Jergens A E, Moore F M, Haynes J S and Miles K G 1992 Idiopathic inflammatory bowel disease in dogs and cats: 84 cases (1987-1990).Journal of American Veterinary Medical Association 201 (10): 1603-1608.

Jergens A E, Andreasen C B, Hagemoser W A, Ridgeway J and Campbell K L 1998 Cytological examination of exfoliative specimens obtained during endoscopy for diagnosis of gastrointestinal tract diseases in dogs and cats. Journal of American Veterinary medical Association 213 (12): 1755-1759.

Johnson B M, Denovo R C and Mears E A 2009 Canine Megaesophagus 486-492 *In* Kirk's Current Veterinary Therapy XIV (Edited Bonaguru J D and Twedt D C), Saunder Elsevier, USA.

Johnson G F, Jones B D and Twedt D C 1976 Esophagogastric endoscopy in Small Animal Medicine. Gastrointestinal Endoscopy 22:226.

Jones B D 1986 Canine and feline Gastroenterology Pp: 518-519 *In* Clinical Procedure. Philadelphia, W.B.Saunders Company.

Jones J C, Streppa D , Harmon B G and Budsberg S C 2002 In vivo effects of meloxicam and aspirin on blood, gastric mucosal and synovial fluid prostanoid synthesis in dogs. Australian Journal of Veterinary Research 63 (11):1527-1531.

Kapatkin A S, Mullen H S, Matthiesen D T and Patnaik A. K 1992 Leiomyosarcoma in dogs: 44 cases (1983-1988). Journal of American Veterinary Medical Association 201 (7):1077-1079.

Katerina K A, Rallis T S, Prassinos N N and Galatos A.D 2002. Benign esophageal stricture in the dog and cat: A retrospective study of 20 cases. The Canadian Journal of Veterinary Research 66: 55-59.

Kealy J K and McAllister 2005 Diagnostic Radiology and Ultrasonography of the Dogs and Cats (4th edition) Elsevier Saunders.Pp.67.

Keles I, Altug N, Kaya A, Yuksek C O N and Aslan L 2007 Atropine sulphate test can be an aetiological indicator of vagal bradycardia development in a megaesophagus case. Bulletin of Veterinary Institute Pulawy 51:181-184.

Koc Y, Turgut K, Sen I, Alkan F and Birdane F M 2004 Persistent right Aortic Arch and Its Surgical Correction in a Dog Turkish Journal of Veterinary and Animal Science 28: 441-446.

Kovacs T, Nemeth T, Orosz Z and Koves I 2001 Endoscopy and autopsy follow-up of biodegradable esophageal anastomosis in dogs. Acta Veterinaria Hungarian 49 (4):451-463.

Lamb C R 1999 Recent development in diagnostic imaging of the gastrointestinal tract of the dog and cat. Veterinary Clinics of North America Small Animal Practice 29 (2):307-342.

Lavin L M 2003 Special procedure 235-252 *In* Radiography in Veterinary Technology 3rd edition Elsevier Publication, W.B. Saunders.

Leib M S and Duncan 2009 Gastric Helicobacter spp. And chronic vomiting in Dogs 492-497 *In* Kirk's Current Veterinary Therapy XIV (Edited Bonaguru J D and Twedt D C), Saunder Elsevier, USA.

Leib M S and Sartor L L 2008 Esophageal foreign body obstruction caused by a dental chew treat in 31 dogs (2000-2006) Journal of American Veterinary Medical Association 232(7): 1021-1025.

Lemasters J J (2009) Molecular mechanism of cell death pp-3-24 In Molecular pathology-The molecular basis of human disease (edn by Coleman W.B and Tsongatis G J) Elsevier Publication, California, USA.

Lobetti R G 2000 Survey of the incidence, diagnosis, clinical manifestation and a treatment of Spirocerci in South Africa Journal of South African Veterinary Association 7 (1) 43-46.

Luna S P L , Basilio A C, Steagall P V M, Machado L P, Moutinho F Q, Takahira R K and Brandao C V S 2007 Evaluation of adverse effects of long-term oral administration of carprofen, etodolac, flunixin meglumine, ketoprofen and meloxicam in dogs. Australian Journal of Veterinary Research 68 (3):258-264.

Mahaffey M B and Barber D L 2002 The stomach Pp-615-633 In Text book of Veterinary Diagnostic Radiology (4^{th} edition by Thrall) W B Saunders, Philadelphia, London.

Mansell J and Willard M D 2003 Biopsy of the gastrointestinal tract. Veterinary Clinics of North America Small Animal Practice 33(5):1099-116.

Meddings J B, Kirk D and Olson M E 1995 Non-invasive detection of Non-steroidal anti-inflammatory drug induced gastropathy in dogs. American Journal of Veterinary Research 56(8):977-81.

Meineri G, Peiretti P G, Vitale N, Vercelli A and Ferraris R 2008 Evaluation of Gastroesophageal Reflux Disease and related Alimentary factors in Dogs and Their Owners Journal of Animal and Veterinary Advances 7(10): 1292-96.

Meyer M M and Levine E J 2008 Iatrogenic esophageal foreign body after motor vehicle accident. Gastroenterology and Hepatology 4(4):279-282.

Michael A K and Goldstein R 2004 Canine Idiopathic Megaesophagus Senior Seminar paper, Cornell University College of Veterinary Medicine.

Michels G M, Jones B D, Huss B T and Wagner-Mann C 1995 Endoscopic and surgical retrieval of fishhooks from the stomach and oesophagus in dogs and cats: 75 cases (1977-1993). Journal of American Veterinary Medical Association 207 (9):1194-1197.

Miwa M, Senoue I and Miwa T 1981 Magnified endoscopic observation on the healing process of experimental ulcers Tokai Journal of Experimental Clinical Science 6(2):147-149.

Moore L E 2003 The advantages and disadvantages of endoscopy. Clinical techniques in Small Animal Practice 18 (4): 250-253.

Mylonakis M E, Rallis T and Koutinas A F 2008 Canine Spirocercosis. Veterinary Compendium Pp.111-116.

Mylonakis M E, Rallis T and Koutinas A F 2004.Clinical signs and clinicopathological abnormalities in dog with clinical Spirocercosis: 39 cases (1996-2004).Journal of American Veterinary Medical Association 228 (7):1063-1067.

Neiger R 2009 Gastric Ulceration 497-501 *In* Kirk's Current Veterinary Therapy XIV (Edited Bonaguru J D and Twedt D C), Saunder Elsevier, USA.

Neilson R W and Couto C G 1998 Common congenital cardiac anomalies. Pp-145-161. In Small Animal Internal Medicine. Mosby Inc St. Louis USA.

Paddleford R R 1999 Analgesia and pain management Pp 227-245 *In* Manual of Small Animal Anaesthesia (2nd edition) Philadelphia, Saunders Company.

Ponnuswamy K K, Jeyaraja K, Madhvan U N and Subramanian M 2009 Gastric erosive ulcerative disease in a dog. Indian Veterinary Journal 86:729.

Ponnuswamy K K, Vijayakumar G, Venkatesakumar E E and Subramanian M 2009 Endoscopic retrieval of nasopharyngeal foreign body in a pup. Indian Veterinary Journal 86:189-190.

Raekallio M R, Hielm-Bjorkman A K, Kejonen J, Salonen H M and Sankari S M 2006 Evaluation of adverse effects of long term orally administered carprofen in dogs. Journal of American Veterinary Medical Association 288 (6):876-880.

Ramprabhu R A, Prathaban S, Nambi A P, Nagarajan B and Dhanapalan P 2001 Endoscopic evaluation of gastric mucosa after oral administration of Ibuprofen in dogs. Veterinarski Arhiv 71 (1):47-51.

Randelia H P, Panicker K N S and Lalitha V S 1990 Megaesophagus in the mouse, histochemical and ultra structural studies. Laboratory Animals 24:78-86.

Rang H P, Dale M M, Ritter J M and Flower R J 2007 Cholinergic Transmission Pp.144-167 In Rang and Dales Pharmacology (sixth edition) Churchill Livingstone Elsevier.

Richard J E, Paul E Z and Ellis E H Jr 1967 Congenital oesophageal achalasia in the dog. Thorax 22: 466.

Rogers K S 2009 Collection of Specimens for Cytology 301-304. *In* Kirk's Current Veterinary Therapy XIV (Edited Bonaguru J D and Twedt D C), Saunder Elsevier, USA.

Rohrer C R, Hill R C, Fischer A, Fax L E, Schaer M, Ginn P E, Casanova J M and Burrows C F 1999 Gastric haemorrhages in dogs given high doses of methylprednisolone sodium succinate. American Journal of Veterinary Research 60(8): 977-981.

Ross M H, Romrell L J and Kaye G I 1995 Muscle Tissue pp214 *In* Histology A text and Atlas 3rd edition Lippincott Williams and Wilkins, Philadelphia Pennsylvania, USA.

Roth L, Leib M S, Davenport D J and Monroe W E 1990 Comparison between endoscopic and histologic evaluation of the gastrointestinal tract in dogs and cats: 75 cases (1984-1987). Journal of American Veterinary Medical Association 196 (4):635-638.

Roudebush P and Delivorias M H 1985 Duodenal aspiration via flexible endoscope for diagnosis of giardiasis in a dog. Journal of American Veterinary Medical Association 187 (2):162-163.

Rousseau A, Prittie J, Broussard J D, Fox P R and James H 2007 Incidences and characterization of esophagitis following esophageal foreign body removal in dog: 60 cases (1999-2003) Journal of Veterinary Emergency and Critical Care 17(2): 159-63.

Rychlik A, Nieradka R, Kander M, Depta A, Nowicki M and Sartik 2007 Usefulness of endoscopic examination for the diagnosis of inflammatory bowel disease in the dog. Poland Journal of Veterinary Sciences 10 (2): 113-118.

Saini N S, Singh S S, Toor A S, Kumar A and Kaur A 2009 Surgical management of persistent right aortic arch and patent ductus arteriosus in a Labrador pup. Indian Journal of Veterinary Surgery 30 (1):53-54.

Shabestari A A S L, Mohammadi M, Jamshidi S, Sasani F, Bahadori A and Oghalaie A 2008 Assessment of chronic Gastritis in Pet Dogs and its relation with Helicobacter-Like Organisms. Pakistan Journal of Biological Sciences 11(11): 1443-1448.

Shaheen N and Ransohoff D F 2002 Gastroesophageal Reflux, Barret esophagus and Esophageal Cancer. Journal of American Medical Association 287 (15):1972-1981.

Sherding R G, Johnson S E, and Tams T R 1999 Esophagoscopy pp 39-96 *In* Small Animal Endoscopy (IInd Edition Tams T R) Mosby Publishing St. Louis, Missouri.

Simpson K W 2005 Diseases of the Stomach Pp:1310-1331 In Textbook of Veterinary Internal Medicine (6th Edition by Ettinger and Feldman) Elsevier Saunders St Louis Missouri.

Sorjonen D C, Dillon A R, Powers R D and Spano J S 1983 Effects of dexamethasone and surgical hypotension on the gastric mucosa of dogs: clinical, endoscopic and pathological evaluation. American Journal of Veterinary Research 44(7):1233-7.

Spurr A R 1969 Journal of Ultra structural Research 26:311.

Strauss-Ayali D and Simpson K W 1999 Gastric Helicobacter infection in Dogs. Veterinary Clinics of North America Small Animal Practices 29(2):379-414.

Stromar I K, Razumovic J J and Obad A K 2008 Imprint cytology of gastric mucosa biopsy–fast and reliable method for detection of Helicobacter Pylori. Coll. Anthropology 32(1): 171-175.

Tams T R 2003 Gastrointestinal symptoms Pp 1-50. *In* Hand Book of Small Animal Gastroenterology (2nd Edition Tams E R) St. Louis W.B.Saunders. Philadelphia.

Tams T R 1996 Disease of the Esophagus Pp163-216 *In* Handbook of Small Animal Gastroenterology. W.B. Saunders, Philadelphia.

Tams T R 1999 Gastroscopy Pp 97-172 *In* Small Animal Endoscopy. 2nd edition Mosby Publication St Louis, Missouri.

Twedt D C 1993 Perspectives on gastrointestinal endoscopy. Veterinary Clinics of North America Small Animal Practice 23(3):481-95.

Valentine B A 2007 Endoscopic biopsy Handling and Histopathology Pp 31-47. *In* Veterinary Endoscopy for the Small Animal Practitioner (Editor McCarthy T C) Elsevier Saunders St. Louis Missouri, USA.

Vander Gaag I 1988 The histological appearance of peroral gastric biopsies in clinically healthy and vomiting dogs. Canadian Journal of Veterinary Research 52 (1): 67-74.

Vlasin M, Husnik R, Fichtel T and Rauserova L 2004 Acquired Oesophageal Stricture in the Dog: A case report. Veterinary Medical Czech 49(4): 143-147.

Wandera J G 1976 Further observation on canine Spirocercosis in Kenya. The Veterinary Record (30):348-351.

Warren J R 1983 Unidentified curved bacilli on gastric epithelium in active chronic gastritis. The Lancet 4:1273-1275.

Washabau R J 1996 Swallowing Disorders Pp-67-89 In BSAVA Manual of canine and Feline Gastroenterology (1st edition by Thomas, Simpson and Hall) British Small Animal Veterinary Association, UK.

Watrous B J 2002 The esophagus PP: 329-348 *In* Text book of Veterinary Diagnostic Radiology (4th edition by Thrall) W.B. Saunders Company, Philadelphia, London.

Weissberg D 2008 Esophageal Foreign Bodies-How will we extract them in the future? Advances in Clinical Experimental Medicine 17 (6): 595-97.

Willard M D 1984 Some newer approaches to the treatment of vomiting. Journal of American Veterinary Medical Association 184 (5):590-592.

Willard M D and Carsten E W 2009 Esophagitis 482-486 *In* Kirk's Current Veterinary Therapy XIV (Edited Bonaguru J D and Twedt D C), Saunder Elsevier, USA.

Yildirim M Z, Kutsal O and Avcioglu H 2007 Spirocercosis in a dog. Ankara University of Veterinary Fak Derg 54: 61-64.

Zoran D L 2001Gastroduodenoscopy in the dog and cat. *In* Endoscopy. Veterinary Clinics North America Small Animal Practice 31 (4):631-656.

Table 1: Age, breed and sex wise occurrence of diseases of esophagus and stomach in dogs (n=42)

S. N	Disease Condition	Age	Breed	Sex	No. of cases
1	Megaesophagus	4 year	Labrador	Male	18
		2 year & 7 months	Golden Retriever	Male	
		7 months	German Shepherd	Female	
		8 year	Doberman	Male	
		1year & 5 months	German Shepherd	Female	
		11 year	Mongrel	Male	
		6 year & 8 months	Pomeranian	Female	
		4 year	Cocker Spaniels	Male	
		9 year	Mongrel	Male	
		13 year	German Shepherd	Male	
		10 year	Pomeranian	Female	
		4 year & 4 months	Boxer	Female	
		3 year	German Shepherd	Male	
		9 year	Mongrel	Female	
		8 year	Doberman	Female	
		10 year	Labrador	Male	
		11 months	Golden Retriever	Male	
		7 year	German Shepherd	Male	
2	Reflux Esophagitis	3 year & 5 months	Labrador	Male	2
		1year & 5 months	German Shepherd	Female	

3	Esophageal Nodule	9 year	Pomeranian	Male	1
4	Esophageal Foreign Body	2 year	Pomeranian	Male	5
		8 year	Labrador	Female	
		3 year	Labrador	Male	
		4 year & 5 months	Mongrel	Male	
		1year	German Shepherd	Male	
5	Vascular Ring Anomalies	3 months	German Shepherd	Male	2
		2 year and 5 months	English Bulldog	Male	
6	Esophageal Diverticula	3 year	German Shepherd	Female	1
7	Esophageal Erosion/Ulcer	7 year	Labrador	Male	2
		4 year	German Shepherd	Male	
8	Chronic Gastritis	3 year	Pomeranian	Female	3
		9 year	Mongrel	Male	
		8 year	Lhasa Apso	Female	
9	Gastric Erosion/ Ulcer	4year	Labrador	Male	2
		5 year & 5 months	German shepherd	Male	
10	Gastric Foreign Body	2 year	Labrador	Male	4
		3 year & 7 months	Mongrel	Male	
		5year	Labrador	Male	
		7 year & 8 months	Pomeranian	Female	
11	Gastric Dilation & Volvulus	8 year	Great Dane	Male	2
		4 year	Labrador	Male	
Total Number of cases encountered(n=42)					**42**

Table.2: Mean values of haematological parameters in dogs with esophageal and stomach diseases (n=42).

S.N	Condition	TEC (millions per cumm)	Hb (g/dl)	TLC (thousands per cumm)	Differential Leukocyte Count (%)				
					G (%)	L (%)	M (%)	N (%)	E (%)
1	Megaesophagus (n=18)	4.2± 0.08	8.74± 0.12	15.63 ±0.12	80.72 ±0.66	17.83 ±0.21	5.22± 0.10	78.72 ±0.64	1.39 ±0.5
2	Reflux Esophagitis (n=2)	5.25	11.57	16.94	84.00	16.00	6.00	78.00	1.50
3	Vascular Ring Anomalies(n=2)	5.35	13.85	18.05	93.00	21.52	5.50	69.50	0.50
4	Esophageal Diverticula(n=1)	5.83	15.40	16.70	67.00	24.00	7.00	84.00	2.00
5	Esophageal Tumour(n=1)	6.41	12.97	17.50	81.00	18.00	3.00	68.00	1.00
6	Esophageal Foreign Bodies(n=5)	5.55	12.75	17.15	85.54	16.75	4.25	73.25	1.25
7	Esophageal Ulcers(n=2)	3.00	10.14	17.95	82.55	18.00	4.00	78.00	0.50
8	Chronic Gastritis(n=3)	4.90	9.83	17.07	77.00	16.67	4.00	84.33	1.33
9	Gastric Foreign bodies(n=4)	4.78	12.98	16.33	84.75	18.00	5.00	78.00	0.75
10	Gastric erosion(n=2)	4.57	12.15	16.95	83.50	21.50	4.00	80.00	1.50

11	GDV(n=2)	5.85	14.50	17.25	80.50	19.00	3.50	75.50	1.00

Table.3: Mean values of biochemical parameters in dogs with esophageal and stomach diseases (n=42).

S. N	**Condition**	**BUN(**mg/dl**)**	**Creatinine** (mg/dl)	**ALT(**I/U**)**	**AST(**I/U)
1	Megaesophagus (n=18)	20.94±0.20	1.04±0.02	60.19±0.66	48.12±0.61
2	Reflux Esophagitis (n=2)	19.51	1.05	68.10	40.50
3	Vascular Ring Anomalies(n=2)	22.00	1.00	67.95	66.35
4	Esophageal Diverticula(n=1)	16.00	1.40	71.50	56.80
5	Esophageal Tumour(n=1)	19.00	0.70	56.10	68.40
6	Esophageal Foreign Bodies(n=5)	18.75	1.07	48.85	43.55
7	Esophageal Ulcers(n=2)	19.00	0.85	57.21	38.32
8	Chronic Gastritis(n=3)	18.33	1.27	67.57	37.00
9	Gastric Foreign bodies(n=4)	18.50	0.88	44.78	36.98
10	Gastric erosion(n=2)	20.00	1.05	43.55	38.15
11	GDV(n=2)	16.50	1.05	41.70	44.00

Table.4. Mean values of scanning electron microscopic (SEM) measurements of esophageal wall thickness (µm) of the megaesophagus dogs (n=7).

Tissue layers	**Megaesophagus dogs**							**Mean ± S.E**
	Case-1	Case-2	Case-3	Case-4	Case-5	Case-6	Case-7	
Mucosa (µm)	240	257	249	273	298	257	310	269.14 ± 9.84
Submucosa (µm)	308	315	324	307	342	310	302	315.42 ± 5.15
Inner circular muscularis (µm)	216	210	198	241	221	211	203	214.28 ± 5.30
Outer longitudinal muscularis (µm)	252	235	268	246	238	245	248	247.42 ± 4.06

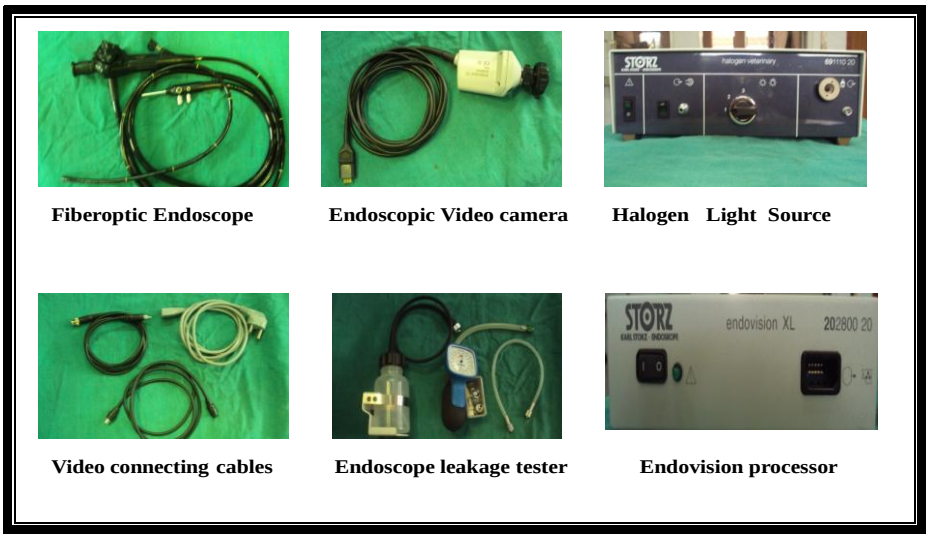


Fig.1 Flexible Fiberoptic Endoscope with required accessories

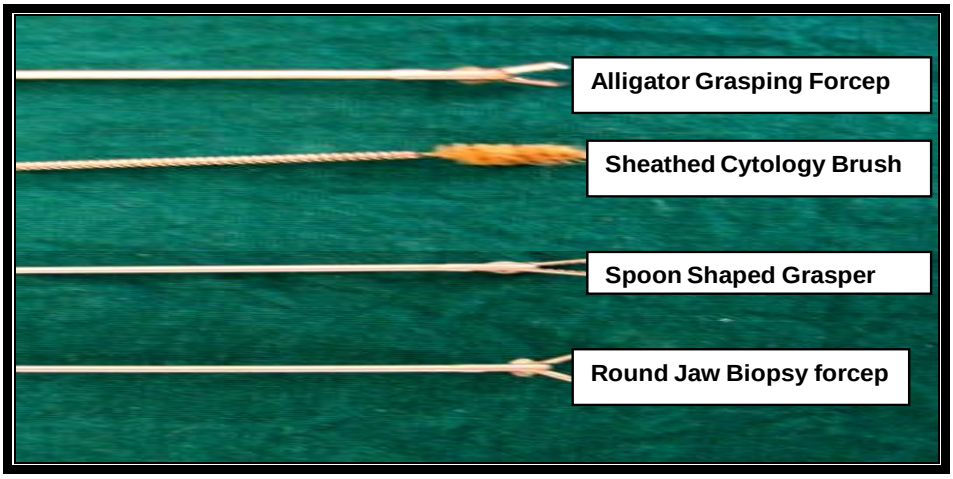


Fig.2 Ancillary instrumentation for use with flexible fiberoptic endoscope

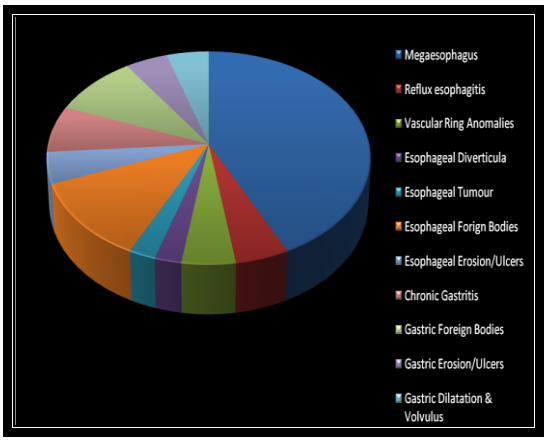


Fig.3 Occurrence of esophageal and stomach diseases in dogs

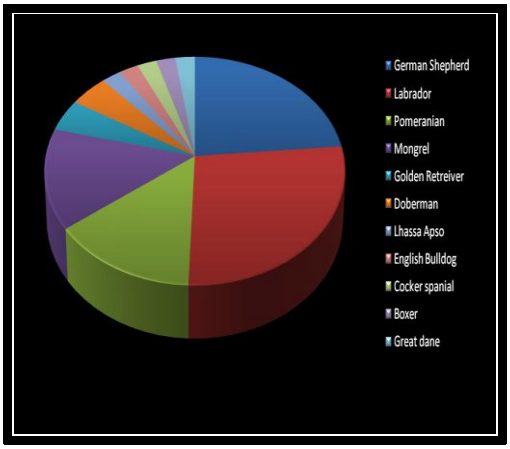


Fig.4 Breed wise occurrence of esophageal and stomach diseases in dogs

Fig.5 English bulldog with persistent regurgitation showing emaciation, hide bound condition, shrunken abdomen, prominent rib cage.

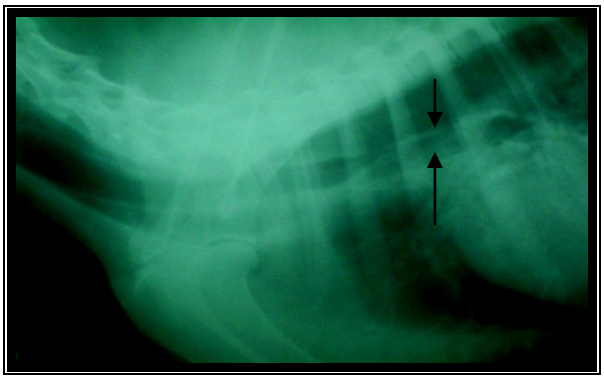


Fig.6 Plain radiograph of the neck and thorax showing severely distended air filled esophagus extending the entire course of the esophagus

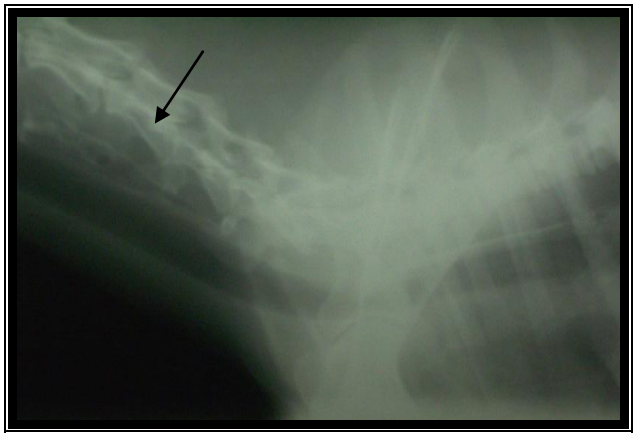


Fig.7 Plain radiograph of the lateral cervical region showing air filled local dilatation of the esophagus (arrows).Esophagus was not discernible in the rest of the radiograph.

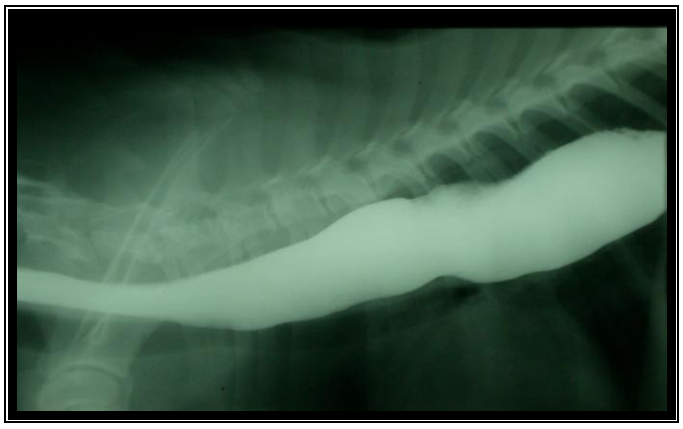


Fig.8 Esophagram with barium sulphate showing severe dilatation of the entire esophagus. The contour of the esophagus indicated its flaccidity

Fig.9 Esophagram with barium sulphate showing severe dilatation of the entire esophagus. Note the filling defects in the cranial to mid thoracic esophagus due to accumulation of retained food material.

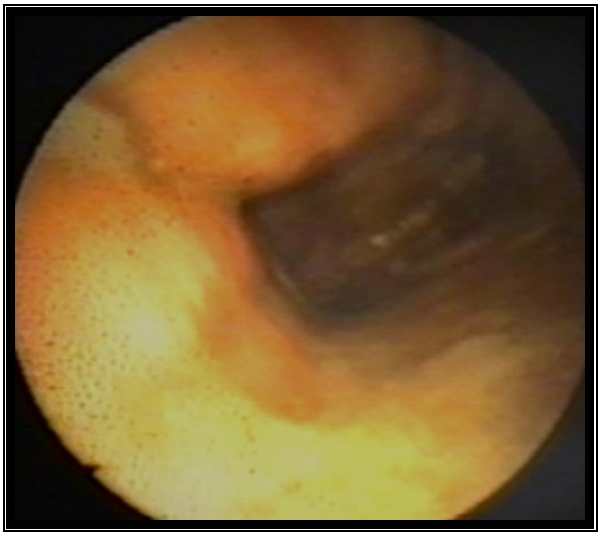


Fig.10 Esophagoscopy showing dilated esophagus with fermented fluid in the caudal esophagus.

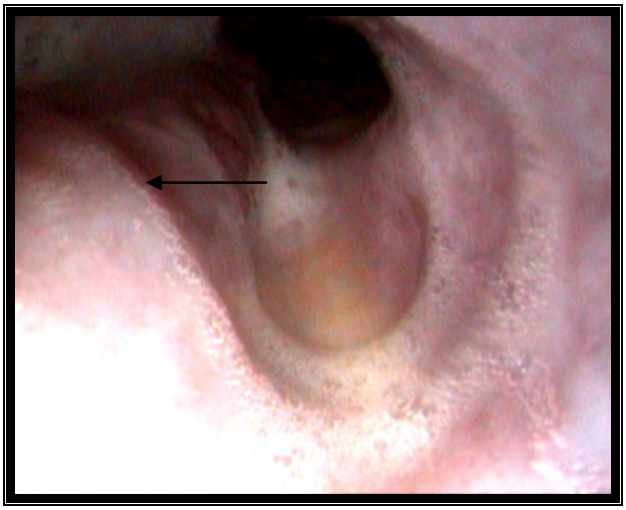


Fig.11 Esophagoscopy showing impressions of the ribs in the cranial to mid thoracic esophageal lumen.

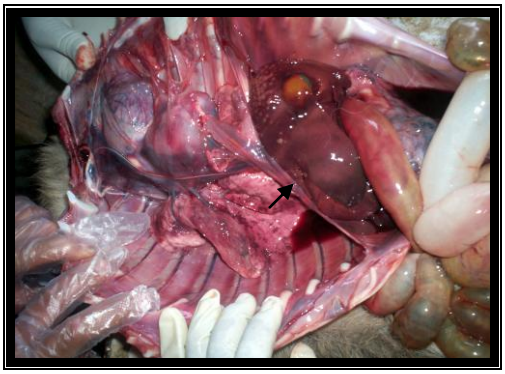


Fig.12 Necropsy examination-Dilated thoracic esophagus with partially digested food and osseous material.

Fig. 13 Necropsy examination - aspiration pneumonia (arrow) due to megaesophagus

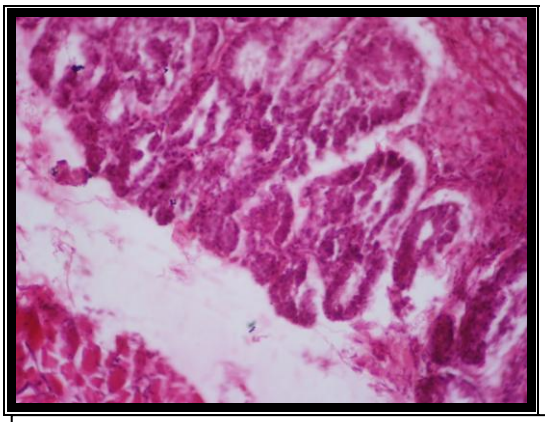


Fig.14 Photomicrograph of the megaesophagus sample showing infiltration of polymorphonuclear cells with submucosal congestion (H & E X 200)

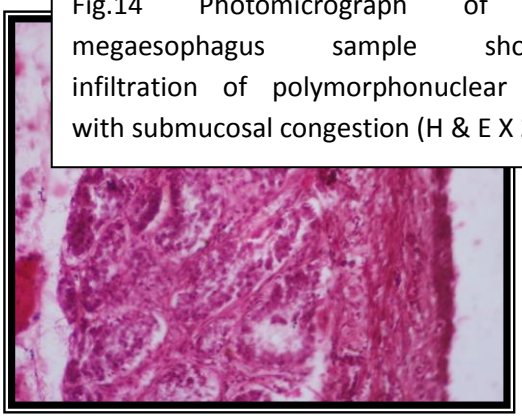


Fig.15 Photomicrograph of the megaesophagus sample showing submucosal congestion and epithelial irregularity (H & E X 200)

cxc

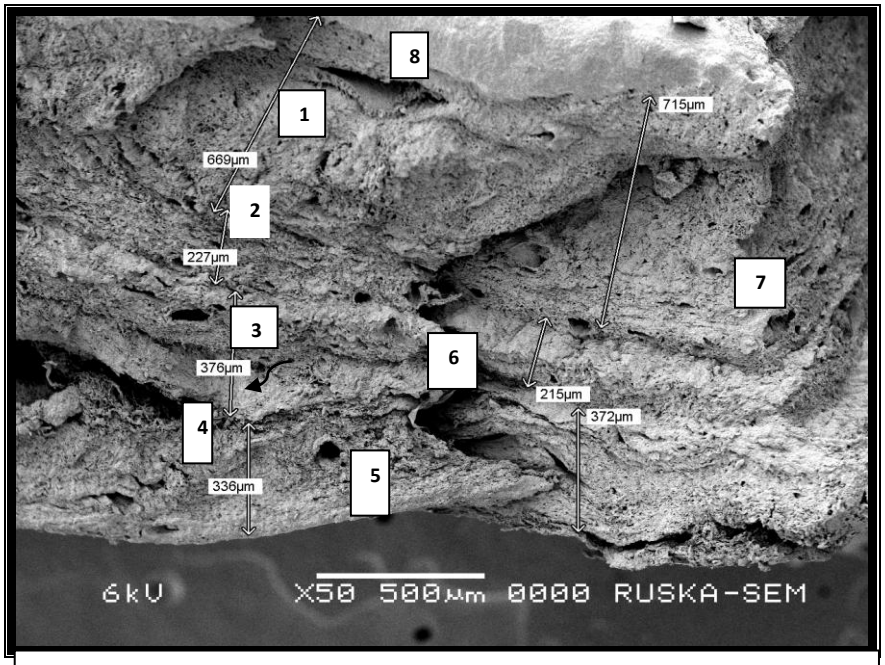


Fig.16 Photomicrograph showing the scanning electron microscopic (SEM) structure of normal thoracic esophagus in dog (6kVx50µm).

1-Mucosa

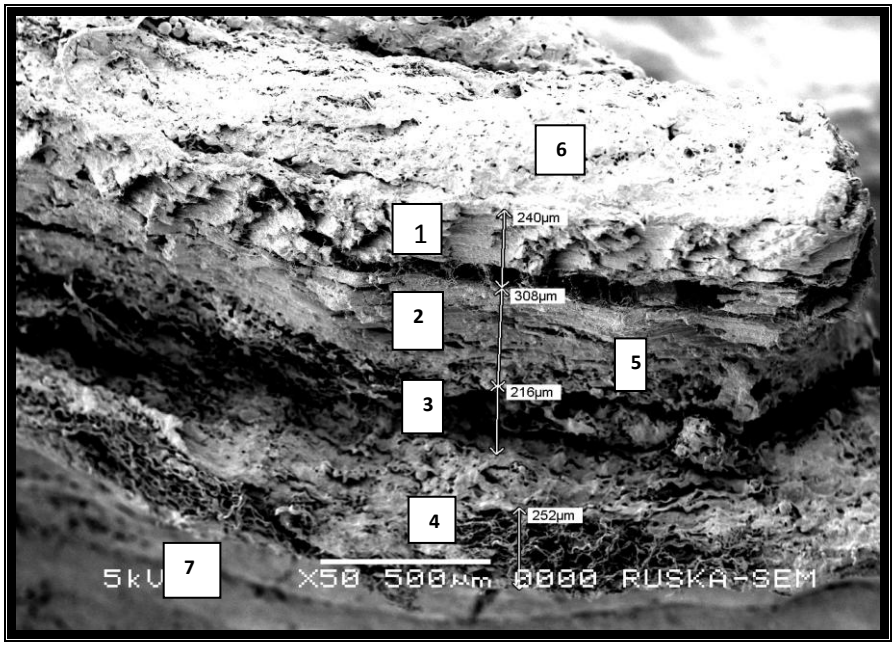
2-Submucosa

3-Inner circular Muscularis layer

4-Outer Longitudinal Muscularis layer

5-Blood vessel

6- Dilated blood



1-Mucosa

2-submucosa

3-Inner Circular Muscularis

4 Longitudina

Fig.17 Photomicrograph showing the scanning electron microscopic (SEM) structure of dilated thoracic esophagus in a dog with megaesophagus (5kVx50µm)

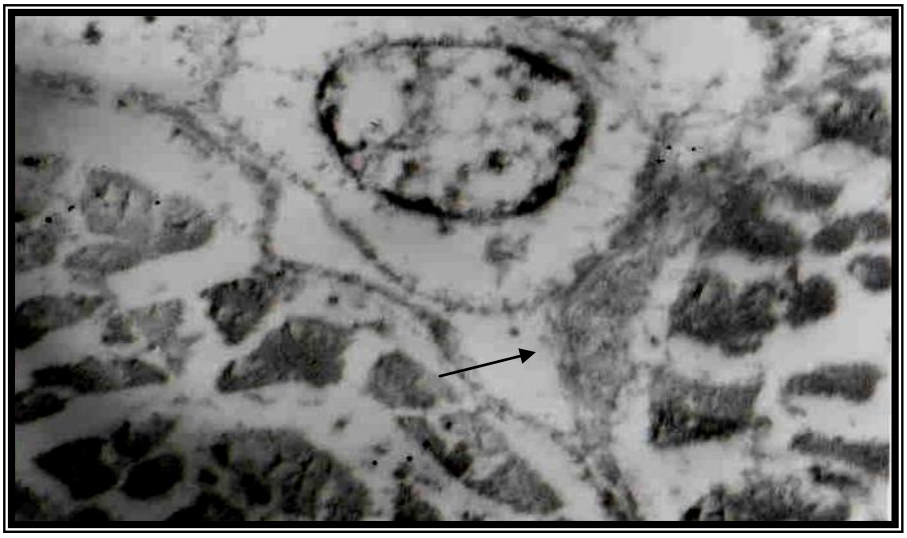


Fig.18 Transmission Electron microscopic examination of normal esophageal wall characterized by loose connectivity between the myofibril bundles (arrow); nucleus contains dark and scanty chromatin material (12530X52)

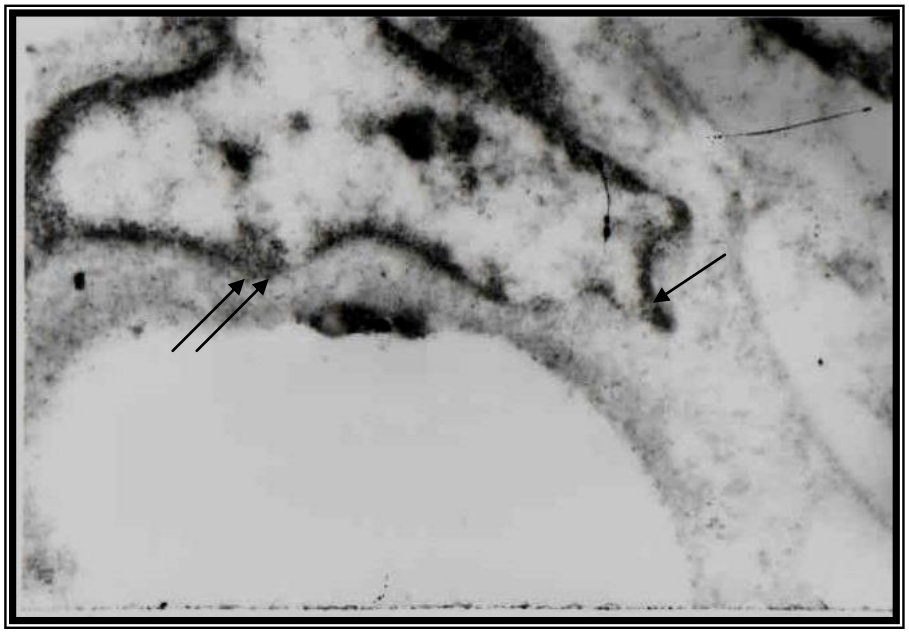


Fig. 19 Electron microscopic examination normal neuro-muscular junction (NMJ) showing with synaptic cleft (single arrow) and vesicles with myoneuronal plate (double arrow) (21480X52).

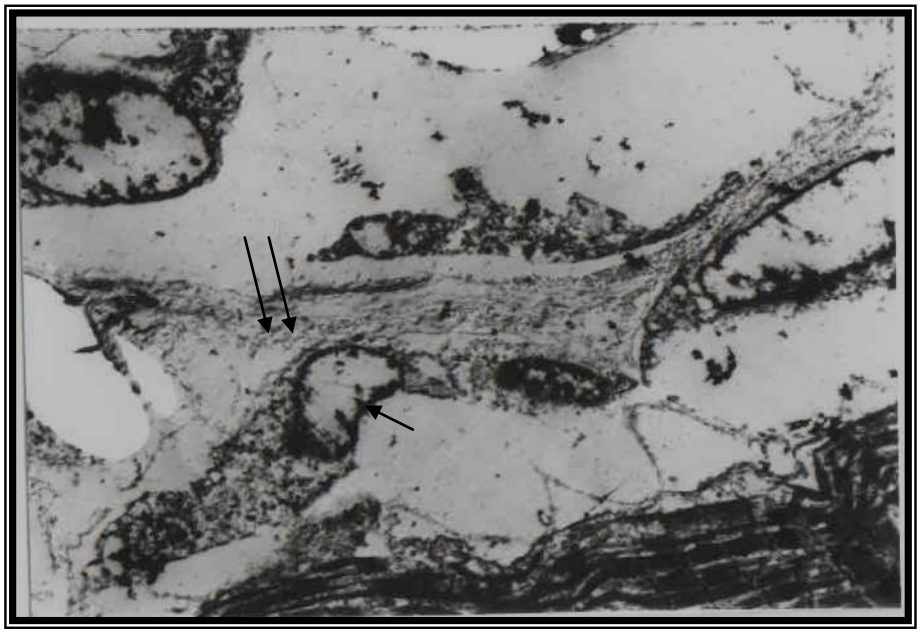


Fig.20 Electron microscopic examination of megaesophagus showing dilated myofibrillar bundles (double arrow), condensed nucleus (single arrow), complete of loss of cellular architecture and vacuolization (4475X52)

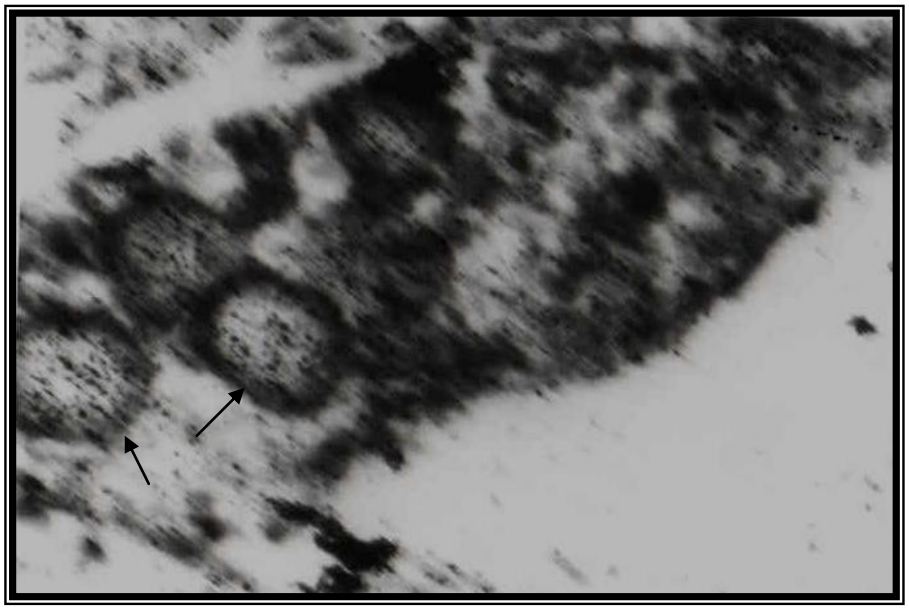


Fig.21 Electron microscopic examination of megaesophagus showing swollen mitochondria (arrows) (44750X52).

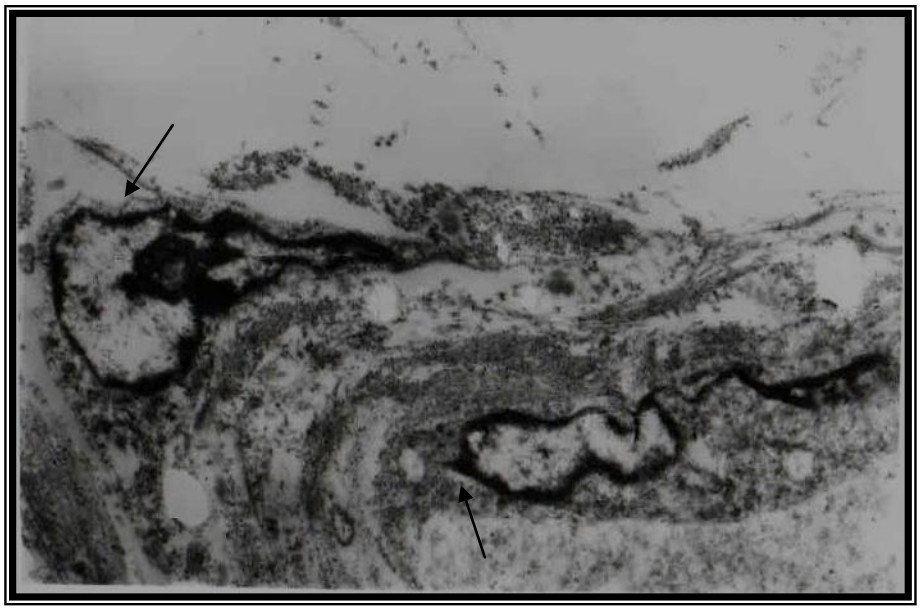


Fig.22 Electron microscopic examination of megaesophagus showing completely destructed neuromuscular junction (arrow) (12530X52).

Fig.23 Esophagoscopy in a dog showing congested and thickened mucosa in the caudal esophagus indicative of reflux esophagitis

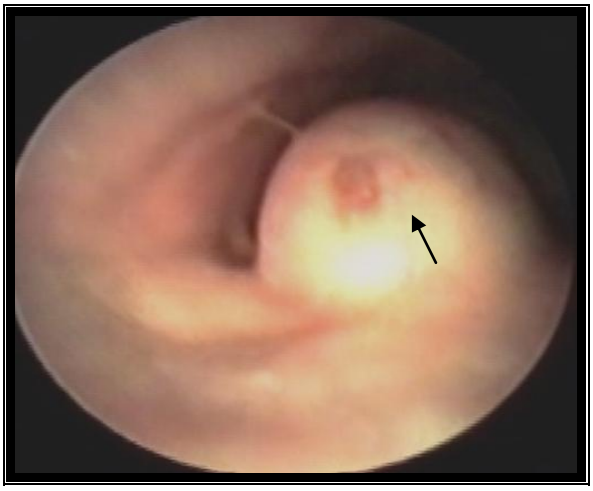


Fig.24 Esophagoscopy showing a lemon sized nodule with an opening on it (arrow).

Fig.25 Plain radiograph of the cervical and thoracic region showing an osseous foreign body in the esophagus just caudal to the pharynx.

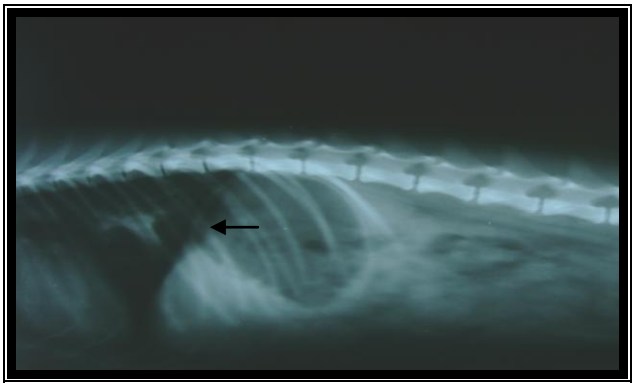


Fig.26 Plain radiograph of the thoracic region showing an osseous foreign body in the esophagus caudal (arrow) to the cardiac silhouette

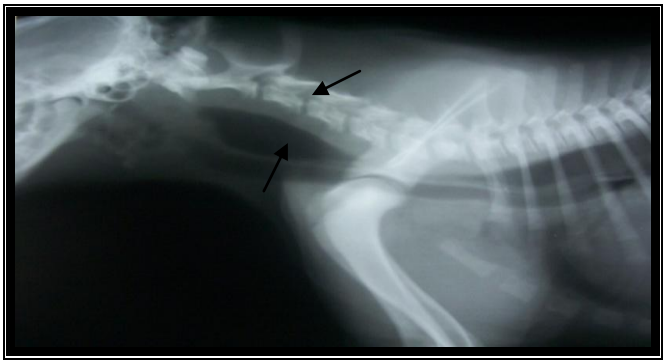


Fig. 27 Plain radiograph of the cervical and thoracic region showing a radiolucent foreign body (socks) in the esophagus just caudal to the pharynx.

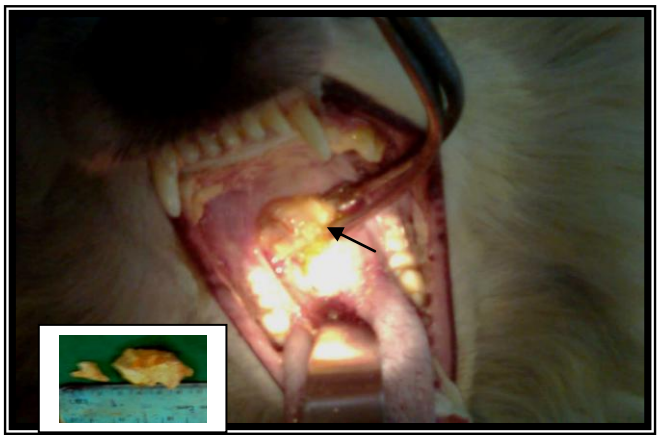


Fig.28 Retrieval of a pharyngeal foreign body under endoscopic illumination using a curved cheatle forcep.After removal of FB (inset).

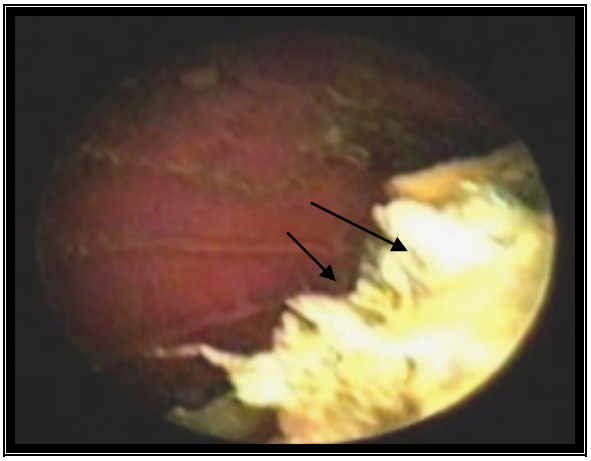


Fig.29 An osseous foreign body pushed into the stomach by using the endoscope tip. Note the osseous foreign body lying in the gastric lumen

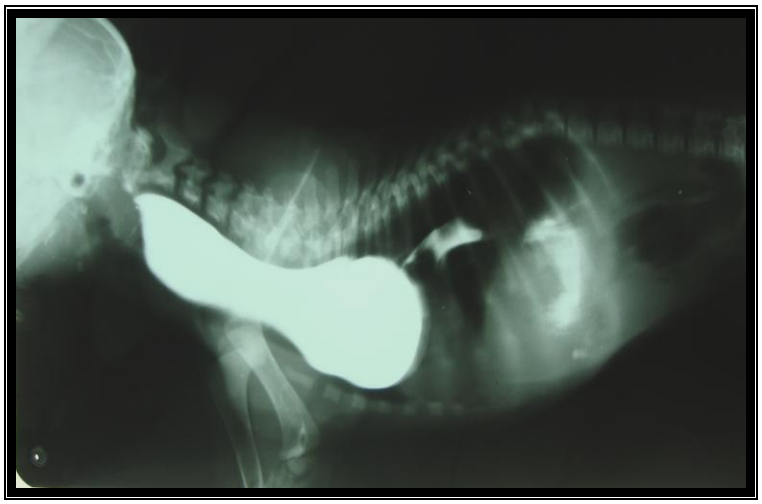


Fig.30 Positive contrast radiograph with barium paste – Lateral view of the neck and thorax showing severe distention of the esophagus cranial to the base of the heart

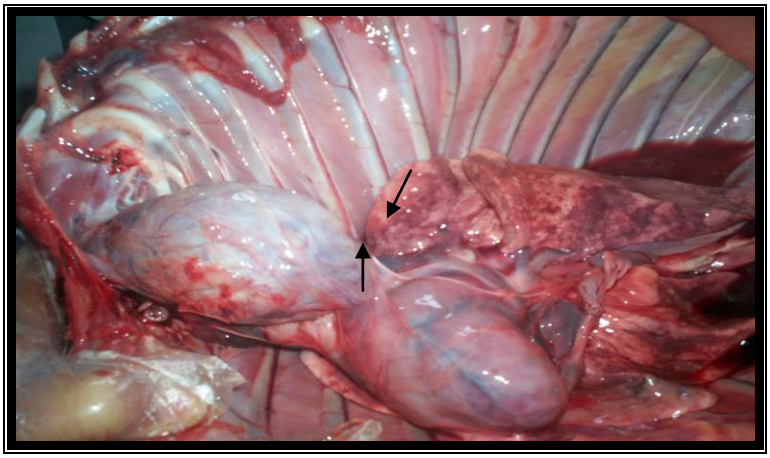


Fig.31 Necropsy examination revealed severe and flaccid distension of the esophagus cranial to the constrictive vascular band

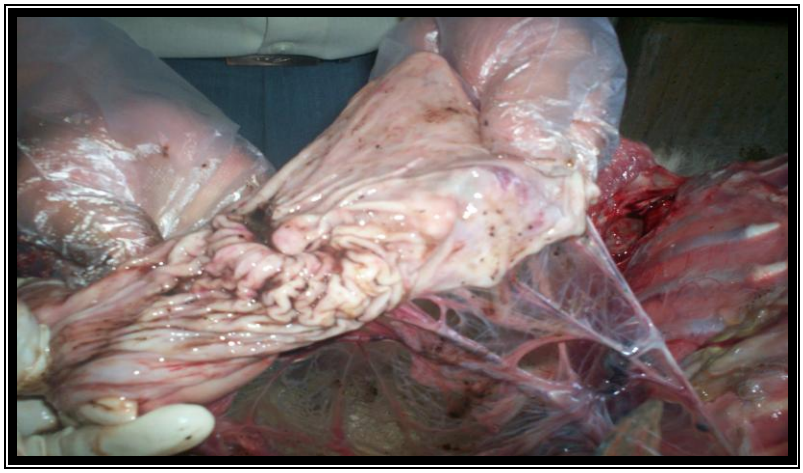


Fig.32 Necropsy examination of stomach showed hypertrophied rugae with generalized congestion of mucosa

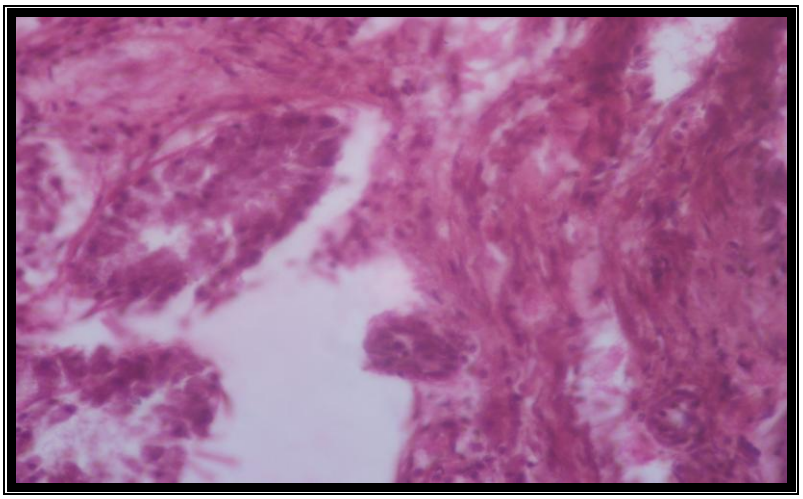


Fig.33 Photomicrograph of esophageal wall showing enlarged mucous gland with submucosal congestion. (H & Ex200)

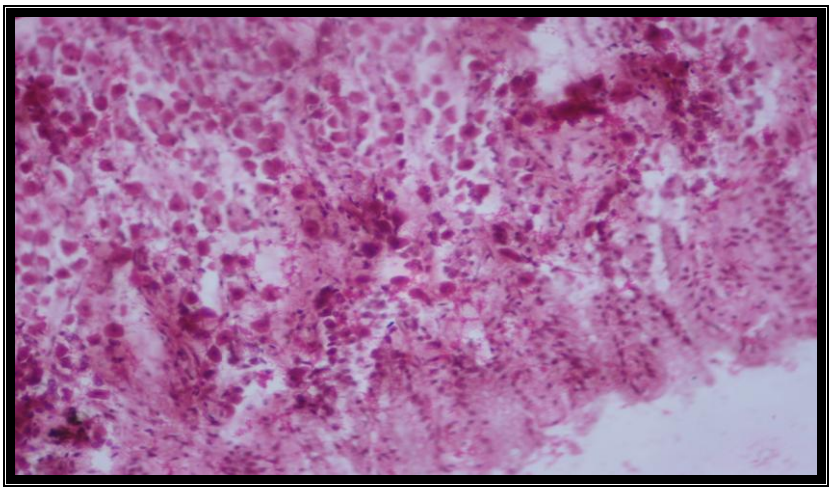


Fig.34 Photomicrograph of stomach wall showing mucosal congestion infiltrates of lymphocytes, plasma cells and neutrophils (H & Ex200)

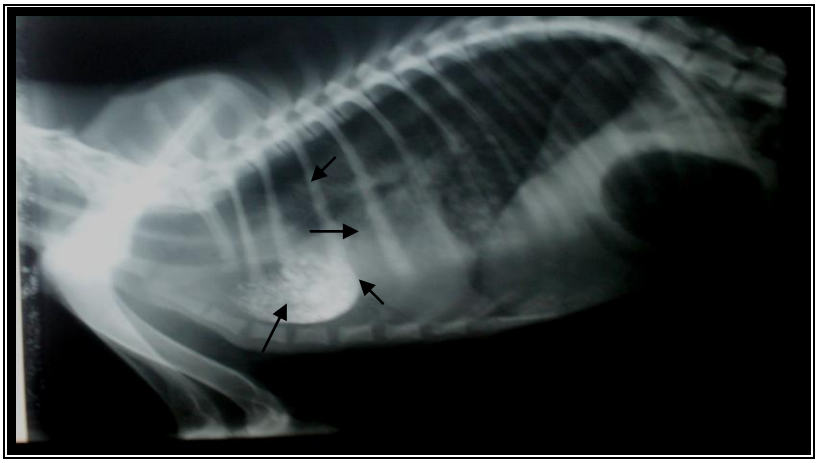


Fig.35 Plain lateral radiograph of the cervical and thoracic region showing sac like out pouching of cranial thoracic esophagus resting on the sternum

Fig.36 Esophagoscopy showing esophageal diverticulum. Note the food material (arrow) on the lower portion of the picture.

Fig. 37 Esophagram with barium sulphate showing remnants of barium still sticking to the inflamed esophageal mucosa (arrow).

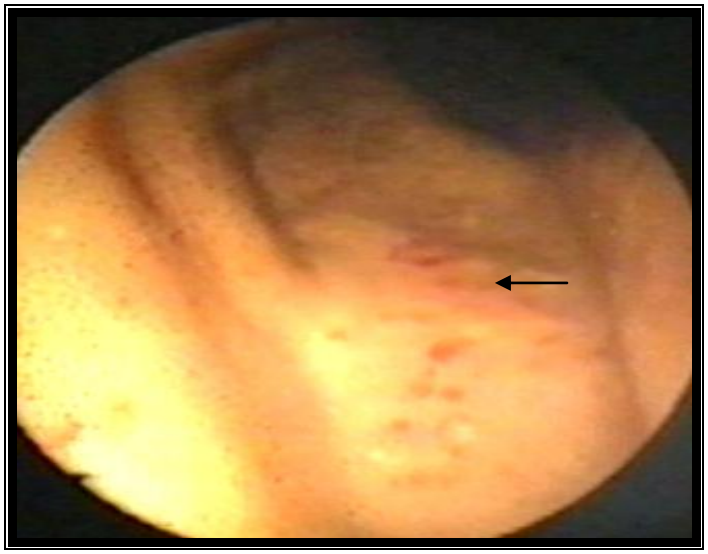


Fig. 38 Esophagoscopy showing esophageal erosions/ulcers.

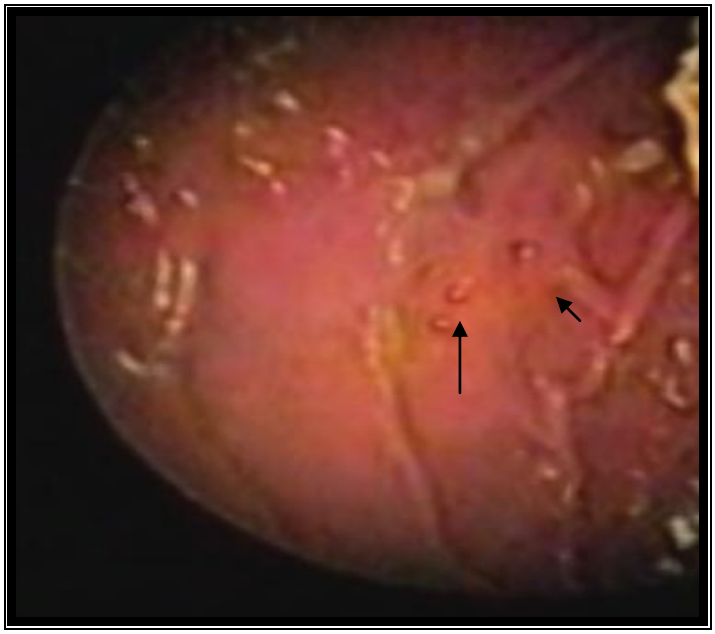


Fig.39 Gastroscopy showing generalized congestion and small areas of mucosal erosion (arrow).

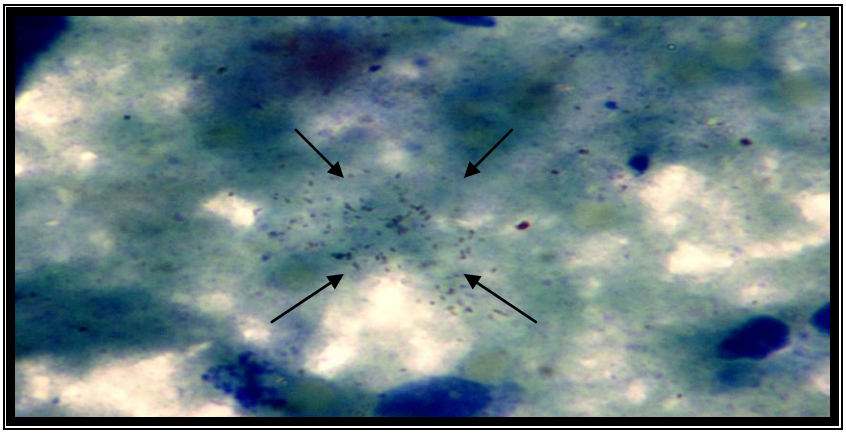


Fig.40 Non-specific cocci colonization (arrows) and lymphocytes in the gastric cytology smears in a dog with chronic gastritis (Giemsa X1000).

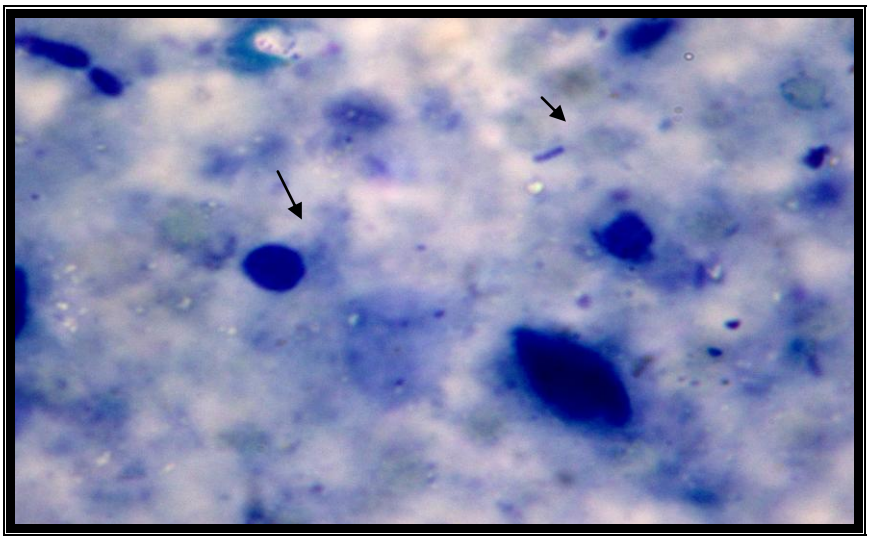


Fig.41 Non-specific bacilli and lymphocytes in the gastric cytology smears in a dog with chronic gastritis (Giemsa X1000)

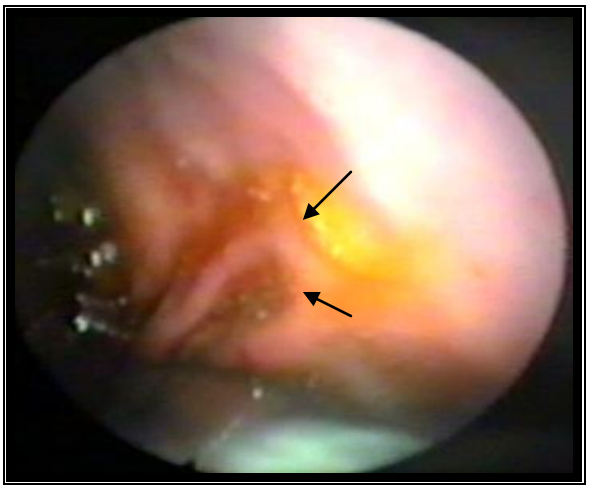


Fig.42 Gastroscopy showing a gastric erosion/ulcer (arrow).

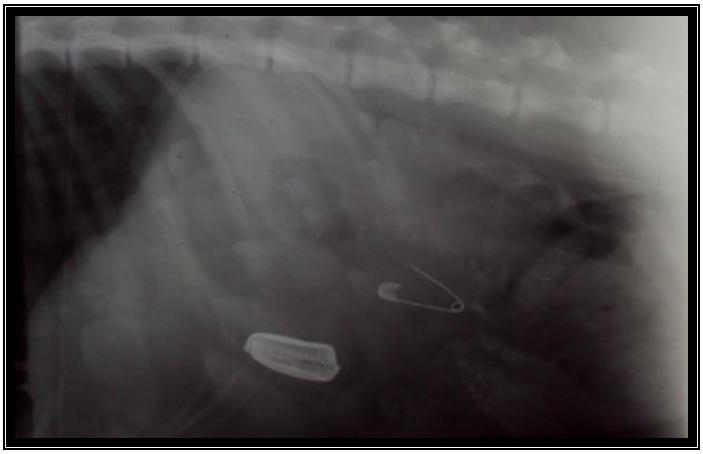


Fig.43 Plain lateral radiograph of abdomen showing pair of cool drink bottle caps and a safety pin

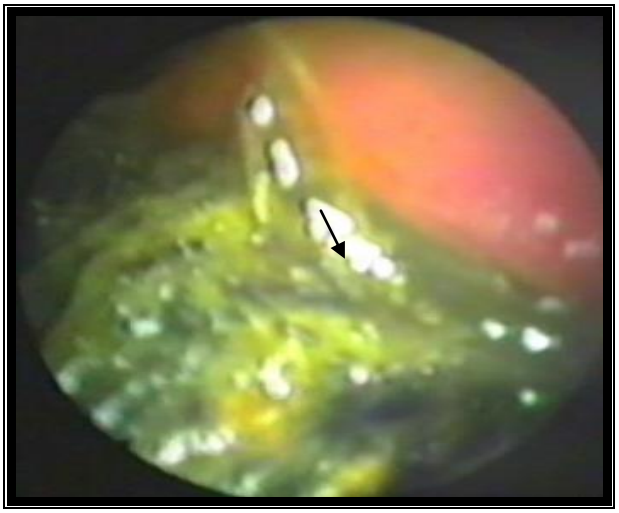


Fig 44 Gastroscopy revealed presence of dog collar made up of nylon

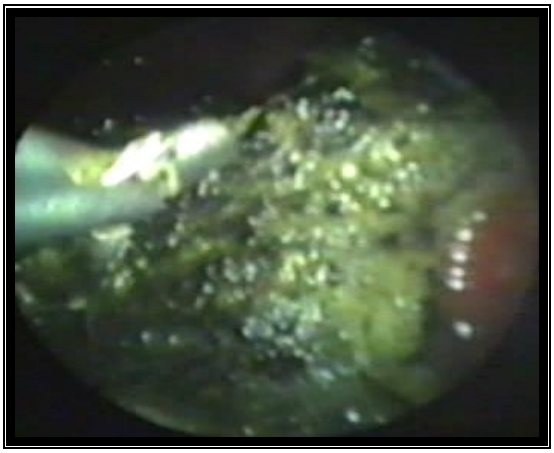


Fig.45 Gastroscopic retrieval of the gastric foreign body. Note the opened jaws of the foreign body retrieval forceps

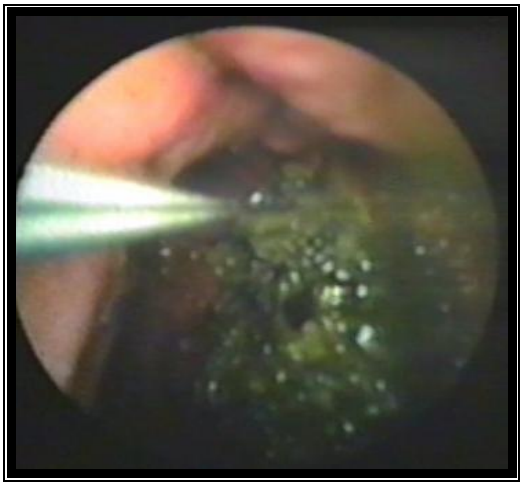


Fig.46 Gastroscopic retrieval of the gastric foreign body. Note that the foreign body in held with retrieval forceps

Fig 47 Radiolucent foreign body, it turned out to be a linear foreign body attached to thread material

Fig.48 Linear foreign bodies removed through gastrotomy incision

Fig.49 Linear foreign bodies removed through enterotomy incision

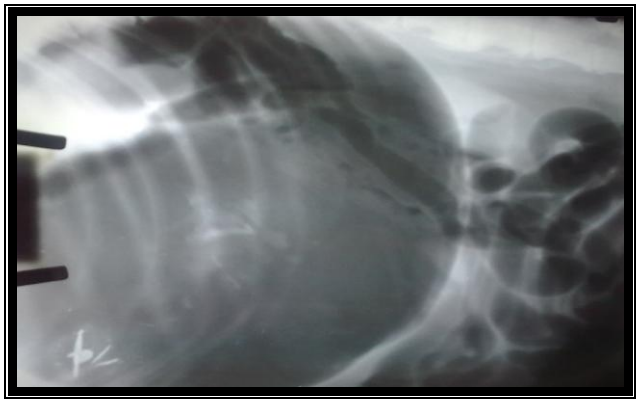


Fig.50 Right lateral radiograph of the abdomen in a Great Dane dog showing severe distension of the stomach with reverse 'C' sign suggestive of GD-V.