TEXTBOOK OF

CLINICAL VETERINARY MEDICINE

By

Dr. Mumtaz Ahmad Khan

DVM, MSc(CMS), MSc(TVM), PhD(MIPM)

Assist. Professor,
Department of Clinical Medicine & Surgery,
Faculty of Veterinary Science,
University of Agriculture, Faisalabad.

A Textbook of Veterinary Clinical Medicine

First edition, 1998
(1000 copies)

Designed and published by the author.

Dedicated

To My

Late Beloved Parents

History.

CONTENTS

	Page
Chapter-1: General medicine	1
Definitions	1
Chapter-2: General Systemic States	6
Fever	6
Types Of Fever	11
A Truth About Fever	11
Hyperthermia	13
Hypothermia	15
Toxemia	16
Septicemia	20
Bacteremia	22
Hypersensitivity	23
Anaphylaxis	24
Disseminated Intravascular Coagulopathy	27
Chapter-3: Diseases Of Alimentary Tract	29
Stomatitis	30
Pharyngitis	33
Esophagitis	35
Esophageal Obstruction/Choke	37
Gastritis	39
Acute Vomiting	42
Equine Colic	52
Impactive Colic	56
Flatulent Colic	58
Spasmodic Colic	60
Diarrhea	62
Acute Intestinal Obstruction	68
Simple Indigestion	71
Bloat	74
Diseases Associated With Hardwares	78
Traumatic Reticulo-peritonitis	78
Acute Rumen Impaction	83
Rumen Alkalosis	87
Vagal Indigestion	88
Left-sided Displacement of Abomasum	91
Peritonitis	93
Ascites	96
Chapter-4: Diseases Of Liver	98
Hepatitis	98
Jaundice	100
Chapter-5: Diseases Of Cardiovascular System	104
Definitions	104
Congestive Heart Failure	108

Urticaria

History & Scope of medicine

Pharyngeal obstruction

Cholelithiasis, Pancreatitis, Diabetes Mellitus.

Enteritis

Acute Heart Failure 111
Peripheral Circulatory Failure 112
Traumatic Pericarditis 114
Chapter-6: Diseases Of Blood And Blood Forming Organs 117
Anemia 117
Edema (Oedema) 120
Hemorrhage 123
Chapter-7: Diseases Of Respiratory System 127
Major Clinical Signs Of Respiratory Diseases 127
Respiratory Noises 131
Principles of treatment of respiratory tract diseases 133
Rhinitis 136
Epistaxis 138
Laryngitis, Tracheitis, And Bronchitis 141
Pulmonary Congestion And Edema 143
Pneumonia 146
Pulmonary Emphysema 151
Pulmonary Abscess 155
Hydrothorax, Hemothorax, And Chylothorax 157
Pneumothorax 159
Pleurisy 161
Chapter-8: Diseases Of Musculo-skeletal System 164
Myositis 164
Myopathy 166
Arthritis 170
Arthropathy 173
Osteomyelitis 176
Osteodystrophy 178
Bursitis 181
Tendinitis 181
Sprains, Sub-luxation, And Luxation 182
Ankylosis 182
Spondylitis, Spondylosis 183
Muscle Spasm And Myoclonus 183
Paresis, Weakness Of Muscle 183
Chapter-9: Diseases Of Nervous System 184
Common Manifestations Of Diseases Of The Nervous Syst. 185
Common Signs Of Cranial Nerve Dysfunction 188
Common Drugs Used In Diseases Of Nervous System 189
Encephalitis 190
Encephalomalacia 193
Meningitis 196
Traumatic Injury To Brain 200
Traumatic Injury To The Spinal Cord 202
Peripheral Nerve Disorder 205
Cerebrospinal Fluid And Its Analysis 207
Chapter-10: Diseases Of Skin 209
Definitions 209
Dermatitis 211
Bacterial Dermatitis 211
Viral Dermatitis 214
Mycotic Dermatitis 215
Parasitic Dermatitis 216
Physical Dermatitis 219
Chemical Dermatitis 220
Allergic Dermatitis 220
Nutritional Dermatitis 220
Pityriasis 221
Hyperkeratosis 221
Parakeratosis 222
Pachydermia 223
Seborrhea 223
Urticaria 224
Eczema 225
Angioneurotic Edema 226
Photosensitization 226
Pododermatitis 230
Tumors And Cysts Of The Skin 231
Diagnostic Techniques For Skin Diseases 232
Antiseptics Used For Skin Ailments 233
Chapter-11: Toxicology 235
Principles Of Therapy In Poisoning 236
Common Toxic Chemicals, Clinical Signs, And Antidotes 242
Common Toxic And Medicinal Plants Of Pakistan
Chapter-12: Common Antibacterial, Antiparasitic, And Anti- Inflammatory Drugs Used In Veterinary Medicine 248
Sulfonamides 248
Antibiotics 248
Antifungal Antibiotics 250
Antiprotozoans 250
Anthelmintics 250
Steroids 251
Non-Steroid Anti-Inflammatory Drugs 252
Chapter-13: Diseases Of Urinary System 255
Definitions 255
Urolithiasis 258
Pyelonephritis 266
Nephrosis 269
Renal Ischaemia 272
Cystitis 274

Chapter-14: Diseases Of The Eye 277
Introduction 277
Ophthalmic Manifestation Of Systemic Diseases 278
Antimicrobial and anti-inflammatory therapy in ocular diseases 280
Trauma To The Eye 282
Kerato-conjunctivitis 282
Cataract 284
Glaucoma 285
Ocular Parasites 285
Chapter-15: Diseases Of The Ear 287
Otitis 287
Ear Hematoma 287

INDEX 289

PREFACE

The textbook covers a comprehensive and tangible commentary on selective miscellany topics on disorders of organ systems and affiliated disciplines of Clinical Veterinary Medicine. The topics picked are mainly corresponding the needs of syllabus of clinical veterinary medicine for DVM studies and the directions of the Curriculum Advisory Committee, University Grants Commission, The Government of Pakistan. The book remains primarily a textbook for the under-graduate and post-graduate students of clinical veterinary medicine. At the same time it contains information of value to the practicing veterinarians in various departments, working with both large and small animals, who inquire for the current concepts of therapy and quest the Art Of Healing.

The diseases of major organs/systems have been presented in sequence and depicted under different headings in order to construct a substantial background knowledge. This allows the veterinary clinicians to employ a problem oriented approach in diagnosis and treatment of animal diseases. Even if a final diagnosis is not accomplished, the sick animals with any ailment can be medicated symptomatically, a practice that is customary exercised in the everyday world. This book also includes chapters with practical information on toxicology and antimicrobial, antiparasitic, and anti-inflammatory therapy. The author regrets for non-compliance in providing the list of authors or references of all books and journals which have been consulted and inspired for the completion of this textbook.

The author extends acknowledgements to his all loving teachers and professional colleagues and appreciates the commendations and support of his wife and daughters.

Mumtaz A. Khan
1998

CHAPTER-1

GENERAL INTRODUCTION

DEFINITIONS

Veterinarian (D.V.M); One who treats the diseases and injuries of animals. A veterinarian is to inspect or examine animals with carefully thoroughness and especially in the quality of an expert. Many veterinarian are not only directly engaged in treating sick animals but also they are doing a variety of kinds of jobs not fitting directly in the above definition.

Medicine; Very simply it can be defined as "THE ART OF HEALING". The theory of medicine lays down the principles on which the disease acts on the body and remedies produce their effects. The practice of medicine consists in the adaptastion of these principles to the treatment and prevention of diseases.

Scientific Medicine; It is the art of treating sick individuals and is based upon the sound anatomical, physiological, and chemical investigations. Beside this, Empirical medicine is an art of treating sick individuals and is based upon the observations and experiences as a mere source of knowledge, but it is closely allied to quackery.

Quack; The person who pretend to skill and knowledge.

Veterinary Medicine; It deals with the diagnosis, treatment or control of diseases of animals. It can be classified into farm (food) animal medicine and pet (companion) animal medicine.
Veterinary medicine can further be arranged into two branches.

i. Clinical Medicine; (Syn: Therapeutic Medicine, Internal medicine, or Curative medicine); It is the branch of medicine which covers the art of achieving correct diagnosis and treatment of sick animals (i.e: following the occurrence of a disease).

ii. Preventive Medicine; It is the art of maintaining health of animals by adopting preventive or control measures against diseases well before their occurrence.

Disease; It is the condition in which an individual shows an anatomical, chemical, or physiological deviation from the normal, even though nutrition and environmental requirements are provided at adequate level.

Health; a) A state of an individual living in complete harmony with its environment.

In human medicine; health is a state of complete physical, mental, d social well-being and not barely the absence of a disease.

In farm animals it is a state of maximum economical livestock oduction or efficient livestock maintenance.

gn; It is the objective evidence of a disease. It is the disease idences that are observed by a veterinarian, livestock owner, endant, or others. Most of the authors used (rightly or wrongly) ns and symptoms as synonyms.

mptoms; Any functional manifestation of a disease, often tricted to subjective evidences of disease, i.e. as experienced and rated by the patient, and which is out of question in veterinary dicine. Veterinary medicine scout this definition to use symptoms l signs synonymously. In contrast, a Lesion is the pathological or actural changes in organs which could be detectable either by ed eye or microscopically. The symptoms could be either;

jective symptoms; Those symptoms which are discovered by a ician at examination of a patient.

bjective symptoms; Example, flank watching in horses is icative of colic, simulating the identification of the site of pain by rrogation in human medicine. (Feature of Disease which can be readily examined.)

monitory or precursory symptoms; Those symptoms which evidenced during the early phase of the disease. Example include ering of the body before the development of fever.

irect or idiopathic symptoms; Those symptoms which directly ect of the primary disease, as presence of mucous and tenesmus indicative of dysentery.

ndirect or sympathetic symptoms; Those symptoms which are ociated with diseases of any remote organ. Examples, vomiting in ociation with pyelonephritis, or dyspnea in myocardial asthenia.

ical symptoms; Those symptoms which are considered acteristic of a disease and which are of diagnostic value, e.g. icaemia with crepitation in muscle is sign of Black-leg.

ical symptoms; The symptoms which do not bear any conformity sease, as development of nervous signs in coccidiosis.

Periodical symptoms; Those symptoms which disappear and reappear with due coarse of time in a disease process, as behavior of fever in Trypanosomiasis.

Pathognomonic symptoms; The symptoms which definitely and directly point to a particular disease, as lock-jaw in Tetanus or tarry blood from natural orifices in Anthrax. or

Prognostic symptoms; The symptoms which frame the basis for a clinician to determine the outcome of a disease, e.g. High blood urea nitrogen contents in acute nephritis and grave prognosis.

Prognosis; It is an opinion (forecast) as to the probable course and outcome of a disease. The animal may recover completely following a disease or not possible. Prognosis may be graded as favorable, unfavorable, doubtful, poor, hopeless, or grave. This is the basis for economical treatment, decision of keeping the animal for fattening, or its immediate and proper disposal.

Complication; A morbid condition arising from during the course of the disease and not caused by the primary factors, e.g. congestion of lungs or decubitus after prolonged recumbency.

Sequelae; It is a pathological change which occurs after recovery or apparent recovery and is usually in the nature or a defect resulting from the original disease, e.g. Roaring following Strangles.

Convalescence; It is that state through which a patient passes after a prolonged illness till it regain normal health and optimum power of production.

Diagnosis; (Dia = thorough, Gnosis = knowledge). It is an art to recognize and determine the seat and nature of the disease and its distinction from other morbid conditions. This is the most difficult and important factor in veterinary medicine. The diagnosis may be classified as under;

i. **Clinical diagnosis;** in which diagnosis of a disease is made on the basis of history as narrated by the owner and observations of the signs/symptoms of made by the clinician.

ii. **Differential diagnosis;** it is the comparison made on the basis of symptoms of different diseases and to rule-out the presence of others similar diseases.

iii. Laboratory diagnosis; based upon the information obtained through the application of common laboratory procedures on pathological materials. It may also include newly developed techniques, i.e. ultrasonography, endoscopy, radiography, and nuclear imaging.

iv. Postmortem diagnosis; diagnosis based upon the observations of post-mortem lesions in an animal died due to any disease.

v. Therapeutic diagnosis; based upon the clinical recovery from any disease following treatment with drugs, pharmaceuticals, or biologics, e.g. A positive response of calcium therapy in milk-fever is diagnostic for this condition.

vi. Tentative diagnosis; is based on the clinical observations, not supported by any solid evidence, and may have some doubts of disease confirmation.

vii. Confirmatory diagnosis; it denotes the actual disease condition of an animal in which diagnosis is supported by laboratory analysis, therapeutic responses, and/or postmortem findings.

viii. Symptomatic diagnosis; is based upon some leading symptoms or principal pathological changes only.

For making a correct diagnosis, complete information obtained or supplied by the client (history), complete and systematic examination of the patient for observation of symptoms/signs, and application of certain laboratory tests/analysis are important and necessary components. All are done to obtain in-depth information about the etiology and patho-physiology of a disease with ultimate aim of accurate diagnosis and which allows effective treatment. The diagnosis should be made before the commencement of treatment, unless otherwise necessary (as use of sedatives in acute colic in horses before making specific diagnosis).

There are many obstacles in identification of a disease in animals compared to medical clinicians. Animals lacks the subjective expression of their feelings and so the veterinarians have to depend upon their own observations. Most of the times, the animal owners/attendants try their own remedies for treating their sick animals and give second preference to any qualified person, which may mask the actual status of the disease. Also, they deposit wrong statements about the health status of their animals.

In addition, veterinarians have to deal with a variety of kind of animals (pretty likely all living-beings except the human-beings) with lot of variations in their normal behavior and physiology, which make the task of diagnosis more cumbersome than medical clinicians. Animal patients do not cooperate with the operators while being examined which enlarges the inconvenience in the process of diagnosis. Above all, most veterinary hospitals lack the laboratory facilities in support of identification of diseases and therefore so, the veterinarians have to depend upon their own personal skills and experiences in making diagnosis.

Treatment or therapy; In veterinary medicine, particularly in farm animal medicine, the economic aspect of treatment is very important. The cost of treatment should not exceed the value of animal or the loss due to disease aspects. The aim of treatment of sick animal is to bring it back to its normal state of health as quickly and completely as possible. The protocol of treatment should include the followings;

1. Specific treatment; The treatment is directed toward the nature of the cause (etiological agent) of a disease if known. The aim of treatment is to remove or overcome the etiological agent(s) by the use of specific drugs or therapeutics. Examples, administration of penicillin in Strangles, anthelmintics in Helminthiasis, or adrenaline in Anaphylaxis.

2. Symptomatic treatment; In veterinary medicine this is the basis for most of the therapeutic protocols. In this the treatment is diverted towards the clinical abnormalities observed due to any disease condition. Example, use of astringents in diarrhea, expectorants in bronchitis, antipyretic in fever.

3. Supportive treatment; particularly adopted during the convalescence period, and

4. Treatment of complications; if present following recovery from a primary disease.

Other terms in the Treatment of animal diseases are; Palliative treatment are adopted for incurable diseases, just to prolong the life span, Empirical treatment based upon mere experience of clinician without rationale, and Prophylactic treatment or measures to prevent the spread of a disease when it is likely to develop in animals.

CHAPTER-2

GENERAL SYSTEMIC STATES

Many general systemic states are encountered in domestic animals. The knowledge of development and line of treatment are important because of their contribution in the outcome of many specific diseases. In addition, many of them are closely associated for their impact on an individual. The notable systemic states experienced in our domestic animals are described.

FEVER
Syn: (PYREXIA, FEBRIS)

"Humanity has but three great foes, i.e. Fever, Famine, and Fight (wars), of these the most terrible by far is Fever" (William Osler).

Fever is an elevation of the body temperature caused by stimulation of the thermoregulatory center (lies within hypothalamus) under the influence of "pyrogens" circulating in blood. Clinically, the fever is characterized by an increase in body temperature (>2 °F), anorexia, depression, and loss of milk production. It is one of the most common disease problems facing the practicing veterinarians.

Pyrexia or Fever of Unknown Origin (PUO or FUO); Most of the times, in large animal practice, the fever is caused by some infectious agents, easily diagnosed, run a shorter course, and progress to complete recovery (within 2 weeks). If a febrile condition continue for weeks or months accompanied by non specific signs of depression, variable anorexia, weight loss, and above all, its diagnosis remains obscure, it is known as PUO or FUO.

ETIOLOGY
The most common etiology of fever are different infections with either bacteria, virus, protozoa, or fungi. Some of the common diseases of animals and birds associated with fever are;

Bacterial Diseases; Hemorrhagic septicemia, black quarter, anthrax, listeriosis, mycoplasmosis, brucellosis, tuberculosis, salmonellosis, strangles, glanders, and tetanus.

Viral Diseases; Examples are malignant catarrhal fever, bovine viral diarrhea, rinderpest, blue-tongue, African horse sickness, rabies, canine distemper, canine hepatitis, infectious coryza, and Newcastle disease..

Parasitic Diseases; Examples are babesiosis, toxoplasmosis, anaplasmosis, trypanosomiasis, theileriosis, and coccidiosis.

Fungal Diseases; Examples are aspergillosis and other mycotoxicosis.

Immune mediated disorders; Examples are anaphylaxis, drug allergies, purpura hemorrhagica, thrombocytopenia, and rheumatoid arthritis.

Septicemic and toxemic conditions; Examples are mastitis, metritis, localized abscess formation anywhere in the body, endocarditis, pericarditis, arthritis, pyelonephritis, peritonitis, pneumonitis, and hepatitis.

Tumors; Examples include any fast growing tumors such as lymphosarcoma, squamous cell carcinomas, fibrosarcomas, and melanomas.

Plant, Chemical, and Animal Poisonings; Examples include poisoning with ergot, mustard, bracken-fern, castor beans, gossypol, thorn apple (datura). Many drugs, (e.g. quinidine, levamisole, and furazolidone), chemicals (e.g. iodides, arsenic, and mercury), kerosine oil, many insecticides, and bites of poisonous snakes, scorpions, and spiders can also cause fever.

Miscellaneous conditions; Examples are massive burns, trauma, surgery, intravascular hemolysis, infarctions, and post-parturient hemoglobinuria.

PATHOGENESIS
Infectious agents, any inflammatory lesions, massive tissue damage, or immune complexes release certain chemicals (toxins), called "Exogenous toxins or Pyrogens". The most common exogenous toxin of Gram negative bacteria is the Lipid-A moiety of their lipopolysaccharides. These exogenous toxins (pyrogens), if present in blood can cause the release of certain low molecular weight proteins from polymorphonuclear leukocytes, lymphocytes, monocytes, macrophages, histeocytes, kupffer cells, and vascular endothelium. These proteins are collectively called as "Endogenous Pyrogens" or "Leukocytic pyrogens". Some of the known endogenous pyrogens are interleukin-I, tumor necrosis factor (a & b), cachectin, and interferons.

These endogenous pyrogens don't directly cross blood-brain barrier but bind to surface receptors on endothelial cells of blood vessels close to the neurons of the "temperature regulatory center", i.e.

hypothalamus. This binding of pyrogens activates the calcium channels, leading to the influx of calcium ions into the cells which initiates the activation of phospholipase-A_2. It along with cyclo-oxygenase initiates the production of arachidonic acids, e.g. prostaglandin E_2 (PGE_2). This PGE_2 stimulates the release of certain neuro-transmitters which raise the body temperature to a new "set point" or thermostatic level. Endogenous pyrogens also can directly stimulate the production of prostaglandins in the microvessels of brain.

The response on the organ involved in heat regulation is the conservation of heat loss and increased heat production by vasoconstriction and shivering of muscles respectively, both result in production of fever. A feed-back mechanism of prostaglandin is the stimulation of cAMP which blocks the synthesis of interleukin-1 and other endogenous products, to keep control the body temperature.

In case of prolong fever in an animal, due to constant increase in metabolic rate and decrease feed intake, there is rise in catabolism of body proteins. This can result in wasting and atrophy of muscles and general body weakness. A prolonged fever coupled with general debility can also result in cardiovascular failure, early embryonic death, or abortion.

CLINICAL FINDINGS

A rise in body temperature (>2 °F), depression, loss of appetite, constipation or diarrhea, decrease in milk production, and in chronic cases, emaciation, and muscle wasting are some of the common signs of fever. These clinical signs can be distinguished with each stage of fever. These include;

Stage-I. Period of Increment Temperature or Chill; The duration of this stage fever is variable and is characterized by cutaneous vasoconstriction, coldness and dryness of skin, absence of sweating, muscle shivering, piloerection, curling, and gathering. There is reduction in respiration rate and urine production but heart rate and core body temperature are at increase.

Stage-II. Period of Constant Temperature or Fastigium; In this stage of fever the body temperature remains constant but at a higher set point, so the heat production and loss remain constant. The duration of this stage is also variable.

Stage-III. Period of Decrement Temperature or Defervescence; This stage of fever is characterized by vasodilatation, sweating, muscle flaccidity, polyuria, and decrease in body temperature than of stage-I.

CLINICAL PATHOLOGY

For the diagnosis of fever perform complete blood count for the recognition of cellular response of an animal. Leukopenia in early period, leukocytosis with or without shift to left in later stages, and increased serum protein (fibrinogen) are common evidences in most cases of fever. Other laboratory tests may be recommended for the diagnosis of the etiology. These include blood culture (for the identification of the causative bacteria), direct and stained blood smears (for hemoparasites), serum liver enzymes (to evaluate liver function), and abdominocentesis (for the evaluation of peritoneal fluid). Serological tests are recommended for identification of viral causes of fever.

NECROPSY FINDINGS

In animals dying of a disease associated with high fever, the general necropsy changes include vasodilatation, rapid rigor mortis, and early putrefaction of the carcass. The additional postmortem changes associated with any specific disease will also be present.

DIFFERENTIAL DIAGNOSIS

A rise in core body temperature, anorexia, depression, and constipation are some typical sings of fever in animals. Identification of the specific etiology of fever requires physical examination of animal coupled with laboratory tests (and necropsy changes).

TREATMENT

For treating any animal with fever the followings steps should be considered.

1. Specific Treatment; This include the use of antibacterial, antiprotozoan, or antifungal drugs corresponding to the nature of the etiological agents. Specific antitoxins or hyper-immune serum (e.g. Hyper-immune H.S. serum, anti-enterotoxemia serum, anti-tetanus toxin, and anti-foot and mouth disease serum) may be also be used, as and if required.

2. Symptomatic Treatment; This include the use of antipyretics, adrenocortical hormones, and diuretics.

It is suggested that a mild rise in body temperature (up to 1-2 °F) may be allowed to occur and antipyretics should only be used when fever is injurious. It is also suggested that to maintain a slight rise in temperature, the body may be covered with blankets, so as the animal should use less of its own energy stores for metabolic heat production. The common antipyretics used in the veterinary practice lower the

ody temperature by blocking at different steps of prostaglandins nthesis.

Some of the antipyretics used in veterinary medicine are sodium alicylate, @ dose of 30-50 mg/kg, large animals (LA) & small animals SA), per oral (PO). Horses may be given by intravenously (IV), @ dose f 100 mg/kg. Acetyl salicylic acid (Aspirin), @ dose of 100 mg for cats nd 500 mg of dogs, PO. Phenylbutazone, @ does of 10-20 mg/kg, for A, PO, and in horses @ dose of 4-8 mg/kg, PO, twice a day (b.i.d.), and -4 mg/kg, IV. Other antipyretic drugs used are oxyphenylbutazon Butazolidine), Dipyrone, and Novalgin, @ dose of 25-30 ml/LA, IV or M (intramuscular). These drugs have antipyretic effects for longer uration than salicylates.

IB. A high rise of body temperature can be lowered by application of old water or ice-packs (see also hyperthermia).

Adrenocortical hormones are used particularly in cases of collapse or hock out of fever. Examples include, cortisone, dexamethasone Decadron), flumethasone, hydrocortisone, methylprednisolone, and prednisolone.

Diuretics drugs help in lowering the body temperature as well as emoval of toxins from the body through excessive urine production. Examples are ammonium chloride; potassium nitrate, sodium acid itrate, fursemide (Lasix), chlorthiazide (Diuril), nd mersalyl (Salyran),

A common recipe of a "Fever Mixture" contains the followings ngredients (dose for large animals). Soda salicylate (30 gm), Ammonium chloride (20 gm), Potassium nitras (20 gm), Magnesium ulfate (200 gm), and Water QS (quantity suffient) to drench.

Chiretta, a common herbal medicine used for gastric disturbaces, is lso known to have antipyretic effect).

3.) Supportive Treatment; This include the use of multi-vitamins and minerals preparations. High protein, high caloric, and palatable diet should be used as supportive therapy during the convalescent period. The use of preparations containing iron salt are not recommended in febrile animals.

4.) Treatment of Complications Associated with Fever or Specific Disease; For examples, treatment of dehydration, anorexia, hypoproteinemia, or anemia, if and as required, should also be administered.

TYPES OF FEVER

a. Simple Fever; It is the type of fever where temperature does not touch to normal limit in 24 hours and during this the temperature variations does not exceed 2 °F. Example is of fever associated with Enteric fever.

b. Remittent Fever; In this type of fever the temperature does not touch to normal limit during 24 hours but the variation in temperature during this stretch is more than 2 °F. Examples of this type are of fever due to septicemia, broncho-pneumonia, urinary infection, brucellosis, babesiosis.

c. Intermittent (Relapsing) Fever; In this type of fever the rise in body temperature is for a few hours of the day. There are brief attacks of fever for several hours following by afebrile period and again there is rise in temperature. Examples include human malaria, chronic trypanosomiasis, canine distemper, and fever caused by neoplasia and certain pyrogens.

d. Atypical (Irregular) Fever; In this type of fever there is daily fluctuation of body temperature which does not follow any pattern. This type of fever is most commonly observed in Louping ill and strangles, etc.

e. Transient (Ephemeral) Fever; In this type of fever the body temperature subsides within about 24 hours after its development, as seen in ephemeral fever in cattle.

f. Continuous Fever; In this type of fever the temperature of the body remain elevated for a longer period than in the simple fever, as seen in Q-fever.

g. Periodic Fever; In this type of fever the body temperature rises with an alternate afebrile periods.

h. Sustained Fever; This type of fever is characterized by a consistently high rise in body temperature without any variation.

A TRUTH ABOUT FEVER

i. A rise in body temperature is attributed due to the release of endogenous pyrogens from pathogen activated leukocytes, including lymphocytes (both B and T lymphocytes) activating factors (e.g. interleukin-I). This raise the thermoregulatory set-point (like

thermostat) at higher level. Therefore, a new thermo-equilibrium is produced which means that animals feel cold at the previous normal temperature. In response to this there is an increase in general body metabolism, shivering of body, vasoconstriction in peripheral tissues, and attempts by the animals to seek warm places to insulate form cold.

ii. A short rise in body temperature, particularly at the face of a viral infection, is beneficial for the animals because;

a) A mild increase in temperature enhances and accelerate lymphocyte proliferation responses to antigens or mitogens and antibody production. b) There is increase in hepatic synthesis of acute phase plasma proteins, indirectly by interleukin-I. c) A direct suppression effect of high temperature on growth of pathogenic organisms has also been observed. d) Iron is considered an essential elements for the growth of bacteria. In fever a reduction in iron plasma concentration is observed which so inhibits the growth of certain bacteria utilizing iron. e) Certain neoplastic cells division is known to be inhibited in animals having a considerable febrile period but the exact mechanism is not yet clear.

iii. Anorexia is an important sign of fever and develops under the influence of interleukin-I. With infectious fever there is much increase in tissue catabolism, nitrogen loss, break down of muscle proteins, and much loss of muscle amino acids than simple starvation. Apart from this, anorexia has two roles in reducing fever. a). By cutting food intake an animal lessen the chances of raising plasma iron concentration and thus the growth of certain bacteria is inhibited. b) An anoretic animal don't has the desire to move in search of food which may help to maintain reduced muscular activity and decrease body heat production by staying at one place.

iv. General depression is an other important sign of fever and is also under the influence of interleukin-I and its analogues. Depression reduces the muscular action that may go into increase body temperature. Thus the depression may not be the result of debilitating effects of acute infection but an adaptive component of acute phase response for the survival of animals.

HYPERTHERMIA

Syn: HEAT-STROKE, HEAT PROSTRATION, and MALIGNANT HYPERTHERMIA

It is a remarkable elevation in body temperature either due to excessive heat production or absorption, or deficient heat loss. The causes of this condition are purely physical, i.e. high environmental temperature and humidity (and not infectious).

ETIOLOGY

High environmental temperature and humidity which is commonly observed during summer in the tropics and sub-tropics, are the important factors for developing this condition. Severe muscular exertion, dehydration, and electrolytes imbalance are predisposing factors for hyperthermia. Sweating is an important way of loss of absorbed heat. A decrease in heat loss through sweating begins when humidity is >60 per cent. Ruminant being unable to sweat, so in the presence of mentioned factors, may suffer more with this condition. Similarly, animals of exotic breeds are less heat tolerant and thus are more prone to develop heat-stroke than of indigenous breeds.

In addition, fat animals with long hair coat or lactating animals are attacked more by this condition than their counter-parts. Poisoning with strychnine, mycotoxins, iodide, and damage to hypothalamus (neurological hyperthermia) are other factors associated in the development of hyperthermia.

PATHOGENESIS

When heat load or production increases and homeostatic mechanism of temperature regulation fails to dissipate heat, the body temperature increases. The associated changes within the body due to high temperature are an increase in metabolic rate and non protein nitrogen contents in the blood, a decrease in liver glycogen, and hypoglycemia. Heart and respiration rates are also increased under direct influence of high body temperature and metabolic rate. When body temperature reaches the critical point i.e. 108 °F or above, there is depression of nervous activity and respiratory center which may lead to sudden death of the individual.

CLINICAL FINDINGS

A sudden rise in body temperature (107-108 °F), increase in heart and respiration rates, dyspnea, absence of sweating, increase in thirst, and general depression, particularly during the months of high environmental temperature and humidity, are indicative of heat-stroke. If the period of hyperthermia is prolonged, complications such as abortion, early embryonic death, disseminated intravascular

coagulation (DIC), renal or liver dysfunction, and myocardial necrosis may be noted. In untreated animals convulsion, collapse, and terminal coma leading to death are common with hyperthermia.

LABORATORY FINDINGS

No specific laboratory test is available for the diagnosis of hyperthermia. However, complications such as DIC, renal and liver dysfunctions may be observed in survival cases.

NECROPSY FINDINGS

Incomplete and slow clotting of blood and early rigor-mortis and putrefaction may be noted in animal died of hyperthermia. Non specific microscopic necrosis may be noted in kidneys, liver, and heart in long standing cases of hyperthermia.

DIFFERENTIAL DIAGNOSIS

High environmental temperature, humidity, muscular exertion, followed by high rise in body temperature are diagnostic of hyperthermia. Septicemic and toxemic conditions, e.g. hemorrhagic septicemia, anthrax, and black-leg are also characterized by high temperature. The presence of other specific clinical features associated with each of the disease, widespread mucosal hemorrhages, changes in blood parameters, and presence of the causative organisms in the blood may ease in rule-out.

TREATMENT

Following lines of treatment should be adopted immediately in order to lower the body temperature.

A. i) Continuous application of ice-cold water at the head, neck, and whole body (by immersion, spraying, or bath), till the body temperature falls to 1-2 °F above the normal temperature of that species.

ii) Ice-cold water enemata may helps in lowering the body temperature.

iii) Fluid therapy containing dextrose (5 per cent) and/or normal saline (0.85 per cent), at lower temperature, may serve combating dehydration and lowering the body temperature.

iv) Adequate drinking of ice cold water with common salt and ice blocks to lick also good in lowering body temperature. A well ventilated shelter with good air movement or provision of fan could be of great assistance in decreasing the temperature.

B. (i) Antipyretics can be used next to step A. For this purpose one can use novalgin or dipyrone, @ dose of 25-30 ml/LA, IM or IV. Sodium salicylate, aspirin, or other non-steroid anti-inflammatory drugs may also be used.

(ii) Tranquilizer drugs can be given to sedate the animal and the nervous center in animals with signs of convulsions.

C. After the reversion of initial attack of hyperthermia, supportive therapy including dextrose (5-10 per cent), multi-vitamins, minerals, and sources of quality protein and amino-acids should be advised.

D. Use of broad spectrum antibiotics, particularly during the later stages or following recovery from initial attack of heat-stroke, may be needed to combat secondary bacterial infection.

E. For future prevention of re-occurrence, avoid any kind of exercise, hard work, or even grazing during hot sunny days with high humidity. An ad-libitum excess to drinking water also helps to decrease the incidence of hyperthermia during the season of high environmental temperature.

HYPOTHERMIA

This condition is common in temperate areas of the world compared to tropics and sub-tropics. It is lowering of the body temperature than normal. This condition develops either due excessive heat loss or its insufficient production. It is particularly seen in cold, wet, and windy weather if animal can't regulates its body temperature by metabolic activity, muscle tone, and peripheral vasoconstriction.

ETIOLOGY

In animals the hypothermia may develops due to followings causes. i) Decrease muscular activity in new-borns or very old animals, particularly during the winter months. ii) Metabolic conditions such as hypoglycemia, hypocalcemia, acidosis, and electrolytes (potassium) imbalances. iii) Damage to thermo-regulatory center of an individual. iv) Diseases causing hypoxia, peripheral vasodilatation, cardiac dysfunction, coma, or collapse. v) Development of perfused diarrhea particularly in winter. vi) Over sedation or anesthesia and other drugs which lower the blood pressure and body temperature. vii) Exposure to cool and humid wind during winter months. viii) Emaciation an malnutrition predisposes more for over cooling of animals.

CLINICAL FINDINGS

Sub-normal body temperature, cold skin and extremities, weak pulse, shallow breathing, depression, and lethargy are common signs of hypothermia. In terminal stages of hyperthermia a low cardiac output leading to hypoxemia, decrease reflexes, prostration, and coma may lead to death of the animal.

NB: Development of hypothermia following a period of hyperpyrexia or in the terminal stages of a fatal disease is a bad prognosis.

TREATMENT

Attempts should be made to raise the body temperature and protect the animal from further exposure to cold and direct exposure to wind-chill. For this purpose following measures may be adopted.

a. Keep the animal in pre-heated room or shed. Apply thermal blanket or heat pad (if available), or keep the animal body warm using other means, i.e. gas or electric heater.

b. Administer pre-warmed dextrose solution (10, 25, or 50 per cent), through IV, or other readily utilizable energy sources.

c. Corticosteroids may be used for the prevention or treatment of shock, if demonstrated.

d. In cases of hypoxia, a warm humidified oxygen therapy if given, may makes differences in recovery process.

e. Rectal enemata with luke-warm water can also help in raising core body temperature.

f. The temperature of animal should be raised slowly over a long period of time with careful monitoring the body temperature and cardiovascular system. A quick increase in body temperature through artificial means may be detrimental.

TOXEMIA

It is the presence of toxins in blood, clinically characterized by anorexia, depression, lethargy, and decrease in production.
(It does not include poisoning or toxicity caused by toxic plants, organic or inorganic salts, and poisonous animals).

ETIOLOGY

Toxemia may be classified into two groups, i.e. antigenic toxemia (bacterial & sometime parasitic origin) and metabolic toxemia (due to absorption of toxic body metabolites).

The antigenic toxemia may further be divided into two types, based on the source of toxins. a) Exotoxic toxemia and b) Endotoxic toxemia.

a) **Exotoxic toxemia;** In this type the toxins produced by the bacterial growth outside the body environment is absorbed in the blood to cause the toxemia. Examples include are i) black-quarter disease of cattle, in which the toxin produced by the growing organisms in muscles is absorbed to cause the disease, ii) enterotoxemia of sheep in which the toxins are produced in the intestine by the growing organisms and are absorbed in blood to cause the toxemia, iii) botulism, in this the toxins are produced outside the body, on feed items, by the growing bacteria on them, and are ingested with the infected feed to cause the disease.

b) **Endotoxic toxemia;** The common toxin of most toxigenic Gram negative bacteria is Lipid-A moiety of LPS, which is present in the bacterial cell wall. This is released by the bacteria at bacteriolysis (not by the intact bacteria) and if liberated in large quantity in blood, will cause toxemia.

The metabolic toxemia develops due to accumulation of metabolic toxins produced in the alimentary tract or body tissues. These toxins are normally eliminated from the body through urine and faeces or detoxified by the liver and plasma. Any abnormality with these organs or systems can result in the development of metabolic toxemia. Examples include the followings;

i) In intestinal impaction if faeces are retained for longer time, auto-intoxication will develop. This is because of re-absorption of phenol, creosol, and toxic amines from the intestine. ii) In hepatitis the metabolic end products, i.e. glycine, glucuronic acid, cysteine, and sulfuric acid, are not detoxified by the liver and so they accumulate to a point causing metabolic toxemia. iii) Histamine and histamine like substances are produced whenever there is body tissue damage. In cases of any necrotizing disease these toxic amines may accumulate to the point to cause toxemia. iv) Ketosis develops due to accumulation of ketone bodies in cases of abnormal fat metabolism in some animals. v) Lactic acidosis develops due to the absorption of lactic acid produced from rumen due to over eating of carbohydrate rich feeds.

PATHOGENESIS

Toxins have specific effects on various body organs or systems. Some of those consist of the followings;

Effects on defence and immune systems; Most of the endotoxins exert their effects on body defenses, i.e. coagulation, immune responses, and acute phase reaction. Toxins have the affinity to bind with the surface receptors on macrophages, PMN, and platelets, lymphocytes. As a result various products (such as tumor necrosis factor, cachectin, prostaglandins, interferons, and interleukins) are released from them which may be the main causes of clinical disease.

Effects on hormones and enzymes systems; Toxins have damaging effects on enzyme systems and endocrine glands of the body particularly, anterior pituitary, adrenal gland, liver, and kidney parenchymas and interfere their normal functioning. Disturbance in enzymes and co-enzymes may also interfere with various biochemical processes within the body.

Effects on carbohydrate metabolism; Toxins may effects the utilization of glucose and metabolization of liver glycogen. Therefore, glucose tolerance of tissues is decreased and administration of glucose is not readily utilized by the body.

Effects on protein metabolism; Toxins may interfere the metabolism of protein and increase in muscles breakdown. This may result in muscle wasting and increase in blood urea nitrogen level.

Effects on mineral metabolism; A hypoferremia, hypozincemia, and hypercupremia has been noted with toxemia, which may interfere different metabolic processes utilizing these minerals as essential components.

Effects on blood cells; Platelets-toxin interaction may result in the release of serotonin and histamine, formation of intravascular occlusive coagulates, and disseminated intravascular coagulation (thrombus formation). DIC

Toxins as an antigen; Toxins can also act as an antigen so there may be development of either hypersensitivity or allergic reactions (e.g. purpura hemorrhagica in horses).

Others effects of toxins; The cumulative effects of toxins may also be noted in the form of depression effects on cardio-vascular, nervous, gastro-intestinal, urinary, musculo-skeletal, and hemopoietic systems.

CLINICAL FINDINGS

The general clinical signs of toxemia are anorexia, general body depression, lethargy, emaciation, constipation (occasionally diarrhea), and decrease in milk production. In antigenic toxemia, there will be development of fever but with metabolic toxemia, sub-normal body temperature is a common sign. Other signs of toxemia may include mucosal hyperemia, anemia (in later stages), increase in capillary refill time, and altered bleeding time. In terminal stages of severe toxemia prostration, toxic shock, coma, and death of animal due to the development of peripheral circulatory failure. The specific signs produced by individual toxin will be dealt elsewhere.

CLINICAL PATHOLOGY

Hypoglycemia, increase blood urea nitrogen (BUN) level, decrease glucose tolerance, leukopenia (initially), leukocytosis (in later stages) with or without shift to left, and aplastic anemia (chronic cases) are common features of toxemia. Presence of endotoxins in blood can be observed utilizing "Horseshoe crab amebocytes" or "Limulus amebocyte lysate" tests. In these formation of a gel is an indication of the presence of toxemia.

NECROPSY FINDINGS

There are no common gross lesions specific with different types of toxemia but microscopic changes can be noted in liver, kidneys, myocardium, and endocrine glands.

DIFFERENTIAL DIAGNOSIS

Toxemia requires rule-out from metal (arsenic) and other poisoning, which also alter the body enzyme system and develop similar clinical signs. A chemical assay on feed and gut contents may be required for their rule-out from toxemia.

TREATMENT

The treatment protocol of toxemia can be stepped as under;

1. Removal of the source of toxin by the use of specific antitoxin or hyper-immune antisera (if and as available) and/or antibiotics (orally or parenterally), depending upon the type and source of toxin. In antigenic toxemia, bactericidal antibiotics should be avoided particularly if signs of shock are present. The common bacteriostatic antibiotic for veterinary use are chloramphenicol, neomycin, kanamycin, gentamicin, penicillin, streptomycin, polymyxin-B, and terramycin.

2. a) Treatment of shock by parenteral use steroid therapy. For example, dexamethasone, @ dose of 1 mg/kg, IV, repeated 24 hourly,

or cortisone, @ dose of 1-2 mg/kg, repeated 12 hourly. (With long term steroid therapy broad spectrum antibiotics in higher dosage should also be recommended).

b) Use of antipyretics or non-steroid anti-inflammatory drugs, such as sodium salicylate, aspirin, or phenylbutazone, may be used to reduce fever and coagulopathic changes associated with toxemia.

3. a) Fluid and electrolytes therapy, in massive doses, should be used for the removal of toxin from the blood, combating dehydration, electrolyte or acid-base imbalances, and maintaining of the circulating blood volume. Examples include; Normal saline, Ringer's or Lactate ringer's solutions and IV fluids containing sodium bicarbonate and calcium. Fluid therapy may consistently be given till the animal starts eating and drinking. A flow of urine following fluid therapy is an evidence of good prognosis.

In precious animals the use of whole blood, plasma, or plasma expanders (e.g. Dextran 6 per cent, a high molecular weight solution of glucose), particularly in septic shock may be given to hasten recovery process.

b) Use of multi-vitamins and minerals may be recommended during the convalescent period of toxemic states. Use of vitamins help to repair the enzyme systems and so the appropriate utilization of glucose by the body tissues.

4. Treatment of complications, if and as necessitated during the course of the disease.

SEPTICEMIA

[redacted]

It is the presence of pathogenic organisms and their toxins in blood, characterized by the development of leukocytosis, fever, sub-mucosal and sub-cutaneous hemorrhages, and other signs of toxemia.

ETIOLOGY

Septicemia may develop either with some specific diseases of animals or as a complication associated with any suppurative process within the body. Some of the specific diseases causing septicemia are anthrax, septicemic (and pneumonic) pasteurellosis, salmonellosis, leptospirosis, coli-bacillosis, and listeriosis. The common suppurative processes known to cause (secondary) septicemia are metritis, mastitis, arthritis, endocarditis, pericarditis, pneumonitis, and internal rupture of abscesses.

The predisposing factors which affects the body defence or lower the immune mechanism can indirectly be involved in development of septicemia. These are diabetes mellitus, uremia, severe burns, viral infections, and immuno-deficiency diseases.

PATHOGENESIS

Most of the pathogenic organisms produce endotoxins when multiply in the blood. The development of clinical signs are not only because of the different kinds of toxins produced by invading bacteria but also due to body cells and tissues. In addition, the organisms may invade directly to certain body tissues producing focal or diffuse type hemorrhages. Others may impair the coagulation system, causing intravascular clotting of blood or severe hemorrhages, which may lead to death of the animal. Presence of septicemia for a long time may lead to depression of hemopoietic system which may lead to severe anemia and other cardiovascular defects.

CLINICAL FINDINGS

The primary clinical signs of septicemia are the development of high fever (106-108 °F), sub-mucosal and sub-epidermal hemorrhages, which can best be seen at the conjunctiva and oral mucosa. Other signs are anorexia, constipation or diarrhea, lethargy, general depression, and decrease in milk production. In chronic cases, severe emaciation, cachexia, anemia, and evidences of cardiovascular or respiratory failure may be evident.

CLINICAL PATHOLOGY

The causative bacteria can be isolated from blood culture specially before the use of any antibacterial. Complete blood count may reveal leukopenia (in per-acute cases), leukocytosis with shift to left (in acute or sub-acute cases), intravascular coagulopathic defects, and thrombocytopenia. In chronic cases, an increase in blood urea nitrogen concentration, hypoglycemia, and anemia can be also be noted.

NECROPSY FINDINGS

Sub-mucosal, sub-epidermal, and sub-endocardial hemorrhages are some of the common postmortem evidences of acute septicemia. General hyperemia, variable degree of inflammatory response, congestion of spleen, lungs, liver, and lymph nodes can also be identified. In addition, distinctive necropsy changes may be present in specific diseases mentioned in the etiology.

DIFFERENTIAL DIAGNOSIS

Isolation of bacteria from blood, presence of sun-mucosal and sub-epidermal hemorrhages, and other acute signs of antigenic toxemia

are diagnostic for septicemia. Hyperthermia (heat-stroke) is characterized by high rise of body temperature when the environmental temperature and humidity is high and without the presence of any bacteria in blood.

TREATMENT

Following protocol may be adopted for the treatment of septicemia. When the body temperature exceeds 107 °F, consider it an emergency, and start treatment as in step 2 first.

1. Use of broad spectrum antibacterials (bacteriostatic types), through IV or IM route and in heavy doses. Hyper-immune antisera or antitoxins, if available, can also be used in some specific diseases.

2. Lower the body temperature to nearly normal limits, by using cold application on the body and/or antipyretics. (See fever and hyperthermia).

3. Supportive drugs and nourishing diet should be used during the convalescent period. It may include normal fluid and electrolytes, multi-vitamins and minerals, and amino acids. Hematinics or blood transfusion may also be used in severe chronic cases.

BACTEREMIA

Bacteremia is just the presence of microorganisms in blood for a transient period without production of any toxin and clinical illness.

Various factors determine the occurrence of bacteremia. These include the exposure dose, virulence of the organisms, route of entry, and host's resistance. Generally, systemic illness do not occur in bacteremia, because, the bacteria enter the circulation accidently, usually small in numbers, from some local infection elsewhere in the body. Most of the bacteria entering into the circulation are rapidly cleared off by the body defence mechanism and it varies with the type of organisms. For example, Staphylococcus spp. are cleared off more rapidly than E. coli.

The liver, lungs, and spleen are the major organs and macrophages are the major cells of bacterial clearance. Listeria, Brucella, and Mycobacterium are virulent organisms that multiple within the macrophages, so their clearance is related to the impaired immune system, such as lack of macrophage activation, absence of specific antibodies against those bacteria, and some deficiency in activation of complement system.

A transient bacteremia has been reported in cattle following the use of endoscopy, rectal, vaginal, or mucosal manipulations, and it is not clinically important. Bacteremia has also been noted following IV catheterization, peritoneal paracentesis, CSF harvest, trocarization, or other minor surgical procedures where antibacterials were not used. Occasionally, persistence of bacteremia may cause complications such as endocarditis, meningitis, arthritis, osteomyelitis, liver or lung abscesses, or may leads to generalized septicemia.

The laboratory diagnosis is mainly made by culture of blood, which gives persistent and uniform growth of a single type of organisms. There is neither any clinical evidence of illness nor any change in blood picture.

TREATMENT.

Broad spectrum antibacterials may be recommended in known cases of bacteremia in order to avoid complications. Neonates should be provided with colostrum during early hours of their life to introduce good passive transfer (immunity) against common pathogens.

HYPERSENSITIVITY

It is a state of increased body reaction to any foreign substances (allergens) resulting in injurious aftermath to self.

The hypersensitivity can be divided into 4 types based on the clinical signs. These includes:

Type-I hypersensitivity (Immediate or anaphylactic type reaction); This type of hypersensitivity reaction is produced through the production of reagins, i.e. immunoglobulin-E (Ig-E) which have high affinity for mast cells, basophils, and to some extent neutrophils. It is the most spectacular hypersensitivity type reaction of an individual to an allergen (antigen), characterized clinically by sudden development of respiratory, cardiovascular, skin, or alimentary tract signs. Which in some cases may lead to anaphylactic shock and death.

Type-II hypersensitivity (Cytotoxic reaction); This type of reaction is produced due to immunoglobulins G and M (Ig-G and Ig-M) against antigenic components attached to cell surfaces. The development of hemolysis due to mismatch blood transfusion is an example of this type of hypersensitivity reaction.

Type-III hypersensitivity (Arthus or Toxic complex reaction); This type of reaction is produced by Ig-G type antibody and is due to the formation of antibody-antigen complexes in and around small blood

vessels. This may lead to local damage of tissues particularly of skin, lungs, joints, and kidneys or systemic reactions, e.g. serum sickness and systemic lupus.

Type-IV hypersensitivity (Cell mediated or delayed type hypersensitivity reaction); This type of reaction develops slowly, due to the sensitized T-lymphocytes (involved in cellular immunity), and not by humoral antibodies. Sensitivity responses in Tuberculin and Mallein tests are examples.

TYPE-I HYPERSENSITIVITY REACTION OR ANAPHYLAXIS

ETIOLOGY

The most important cause of hypersensitivity reaction of this type is the introduction of any foreign protein (sometimes carbohydrates) into the some individuals which makes them sensitized and at re-exposure of it causes type-I hypersensitivity or anaphylaxis reaction. The severity of clinical disease will depends upon the chemical nature and amount of foreign materials, route of exposure, species of animals involved, and familial predisposition. In farm animals anaphylaxis is recorded by a number of materials. Some of them are;

i) Injection of certain vaccines (bacterins), such as foot & mouth disease vaccine, hemorrhagic septicemia vaccine, rabies vaccine, and Brucella abortus strain-19 vaccine.

ii) Injection of heterologous antiseras and biologicals containing foreign serum, such as anti-tetanus toxin and foot & mouth disease antiserum.

iii) Injection of certain drugs, such as penicillin and acaprine.

iv) As a part of disease response as seen in malignant catarrh fever, glanders, and brucellosis.

v) Certain parasites may also cause this type of reaction. Example is of rupture of Hypoderma ovis larvae within the skin or Fasciola spp. infestation.

vi) This type of reaction can be caused with certain toxins, endotoxins, toxoids, and moulds introduced in the body.

vii) Other types of foreign proteins, such as glandular extract injection, pollens inhalation, blood transfusion may also cause anaphylaxis reaction.

PATHOGENESIS

Anaphylactic reaction develops when reaginic antibody (Ig-E) molecules present on tissue mast cells and basophils (to some extant neutrophils) cross link with some specific foreign materials (allergens). This antibody-antigen union trigger the release of certain chemical mediators. Some of these are histamine, leukotrienes, serotonin, bradykinin, kinin, dopamine, prostaglandins, eosinophil chemotactic factors, catecholamines, anaphylatoxins (complement 3a, complement 5a), slow releasing substances of anaphylaxis (SRS-A), and many others from those cells which are responsible for anaphylactic reaction.

The clinical response of individual depends upon the chemical mediators, species of animals, distribution of mast cells and development of smooth muscles in various organs. For example, in cattle, there is more involvement of gastrointestinal, skin, reproductive, and cardiovascular systems. In different organs the impact of anaphylaxis are;

Lungs; Constriction of bronchi, bronchioles, and pulmonary arteries, resulting in difficulty in respiration and edema. The chemical mediators responsible for these are SRS-A, leukotriene-C, and prostaglandins.

Gastrointestinal and Cardiovascular Systems; Constriction of hepatic blood vessels, vasodilatation of portal, mesenteric, and intestinal vessels which results in pooling of blood in them. This results in intestinal and ruminal dysfunctions, shock, and vascular collapse.

Reproductive System and Udder; Decrease fetal survival and milk secretion.

Skin; Edematous swelling due to increase in vascular permeability under the effect of histamine at the dermis and subcutaneous tissues, especially around eyes, nose, vulva, and anus.

CLINICAL FINDINGS

Hypersensitivity (anaphylaxis) develops within few minutes following exposure to inciting substances. The initial signs include fast and labored breathing, slow heart rate, hyper-salivation, muscle shivering, frequent urination and defecation, and patchy urticarial type swellings on the skin. Body temperature may fall but later on, it may rise high to few degrees due to muscular activity. Udder edema and decrease production is common in milch animals. Additional signs of anaphylaxis are bloat in ruminants, sweating, laminitis, and colic in horses. Severe cases may end up with anaphylactic shock and death

due to respiratory and/or cardiovascular failure, within 30 minutes of the onset of severe reaction.

CLINICAL PATHOLOGY

Not useful in routine veterinary practice, however, a eosinophilia, increased hematocrit value, high plasma proteins, and neutropenia may be diagnostic.

NECROPSY FINDINGS

Emphysema, congestion, and edema in lungs, congestion and edema in abomasum, subcutaneous tissues, intestine, lymph nodes, and endocardium (and laminies in horses) are common in animals died of anaphylaxis.

DIFFERENTIAL DIAGNOSIS

Development of clinical signs soon after the exposure to an allergens and response to specific treatment are good evidences of anaphylaxis. The diseases which need rule-out are viral pneumonia, other causes of pulmonary congestion and edema, bloat, diarrhea, and subcutaneous edema due to other etiology. In pneumonia, signs of toxemia will also be present. Other diseases mentioned are not accompanied by generalized signs as described in anaphylaxis.

TREATMENT

A prompt and specific treatment can make the different between complete recovery or death. The line of treatment should include;

A. Adrenaline, a well known sympathomimetic drug, is the first drug of choice for the treatment of anaphylactic shock. It can be used @ dose rate of 5-8 ml, by IM or SC (subcutaneously) route, in large animals. One third of the dose can be given by slow IV route for prompt response. If the clinical signs of anaphylaxis persist the drug can be repeated at 30 minutes intervals. Ephedrine, is a commonly used sympathomimetic drug having bronchodilator, vasoconstrictor, and heart stimulant effects, so can be used in asthma like anaphylactic attack.

B. Corticosteroids are the second choice drugs if they are given as a sole therapeutic agent. However, best therapeutic effects can be achieved if they are given with adrenaline. Prednisolone, or similar drugs (Methylprednisolone, Cortisone, Hydrocortisone, Deltacortil, Meticortilone) can be given @ dose rate of 100-200 mg/LA, or 2-20 mg/SA, by IM route. Dexamethasone (and similar drugs as Cortisol, Decadron, Dexasone) can be given @ dose rate of 10-30 mg/LA, and 0.5-2 mg/SA, by IM route.

C. Antihistaminics are the third choice drugs because they are less effective as compared to adrenaline or corticosteroids. It is due the presence of other chemical mediators of anaphylaxis than histamine. These are of two types antihistaminics, (a) H_1 blockers, e.g. Antazoline HCl (Antistin), Diphenhydramine HCl (Benadryl), Chlorpheniramine maleate, Mepyramine maleate (Mepyrasone, Anthisan), and Promethazine HCl) and (b) H_2 blockers e.g. Metiamide, Cimetidine, and Burimamide. Chlorpromazine HCl (Largectil) can be used for similar action.

D. Non-steroid anti-inflammatory drugs, i.e. Sodium salicylate, acetylsalicylic acid (Aspirin), and phenacetin have little value in treating anaphylaxis both in cattle and horses.

DISSEMINATED INTRAVASCULAR COAGULOPATHY (DIC)
Syn: INTRAVASCULAR COAGULOPATHY

This includes either intravascular coagulation of blood (thrombus formation) or fibrinolysis (coagulation defects) which may end up with diffuse hemorrhage.

ETIOLOGY

It is not a primary condition but is associated with a number of conditions leading either to the activation of coagulation cascade or stimulation of fibrinolysis. This condition (DIC) may develop in a number of diseases, such as hyperthermia, toxemia, septicemia, canine heart worm disease, infectious canine hepatitis, feline infectious peritonitis, feline panleukopenia, gastric dilatation, acidosis, leukemia, malignancies (hemangioma and lymphosarcoma), tissue necrosis, and many other inflammatory conditions.

PATHOGENESIS AND CLINICAL FINDINGS

The balance of hemostasis is affected by two types of proteases. "thrombin and plasmin". If thrombin is dominating, thrombus formation will develop, and if plasmin dominates then internal hemorrhages will occur. Which of the above mechanisms will operate depend upon the underlying disease process.

In response to inflammation there may be release of procoagulants and thromboplastin like substances necessary to initiate coagulation sequence. For example, activation of platelets in RBCs lysis causes the release of adenosine diphosphate and thromboglobulin (thrombin) which can initiate intravascular coagulation of blood. Antigen-antibody complexes (in many viraemias) and lipo-polysaccharide can also initiate coagulation of blood through the production of thrombin. Activation of coagulation mechanism through this pathway may also

stimulate complement activation processes. Plasmin (a protease) can degrade fibrin, fibrinogen, and other coagulation proteins through producing fibrin degradation products or fibrin split products, which can result into coagulation defects and diffuse hemorrhage.

CLINICAL PATHOLOGY

Buccal mucosal bleeding time test is a rapid and relatively simple test to evaluate components of hemostasis. The test measures the period required for bleeding to stop from a standardized wound and depends on proper vascular and platelet functions and the number of platelets. Bleeding times between 1.5 to 4.5 minutes are usually considered normal in dogs. If bleeding time is abnormal then platelet counts should be determined to identify the thrombocytopenia. Normal platelet counts are >200,000/ul. Other laboratory tests can also be used for the determination of thrombin time or different coagulation factors.

TREATMENT

Specific treatment should be recommended according to the nature of the etiology (if known), through the use of antibiotics, antitoxins, antipyretics (as required), and surgical removal of necrosed tissues. The inherited disorders are incurable but can temporarily be treated by the use of specific deficient blood component.

In cases of perfuse hemorrhage whole blood transfusion may be necessary for the replacement of lost blood volume and pressure. Whole blood transfusion has many advantages over plasma or plasma expanders but it carries the risk of hemolysis that can exacerbates the DIC. Intravenous fluids therapy using normal saline or Ringer's solutions may be considered for the replacement of lost fluid volume.

In cases of poisoning with rodenticides, administration of vitamin K1 is preferred therapy. In case of thrombus formation the coagulation inhibitors, e.g. Heparin (@ dose of 5-10 units/kg, IV) should be used. This can be repeated at 12 hour intervals and the dose can be adjusted with clinical response.

The use of plasma or plasma expanders along with heparin may have some advantages. Normalization of prothrombin time and fibrinogen contents indicates the success of the treatment. Side effects of heparin are bleeding and thrombocytopenia.

Aspirin is under investigation for its platelet inhibition effects in human medicine and it may be tried for the same in animal practice.

CHAPTER-3

DISEASES OF ALIMENTARY TRACT

The disorders involving gastro-intestinal (G.I.) tract are among the most common disease problems and have great leverage particularly in intensive livestock production. The digestive systems and processes in most ruminant species are quite similar. It includes the oral cavity and associated organs such as the lips, teeth, tongue, and salivary glands; the esophagus; the rumen, reticulum, omasum, and abomasum; the small intestine; and the large intestine, rectum, anus; liver; and pancreas. The peritoneum covers the abdominal viscera and is involved in many diseases of the digestive tract. The abomasum is true glandular stomach similar to monogastric animals.

The primary function of the digestive tract include prehension of feed and water; mastication, ensalivation, and swallowing of feed; digestion of feed and absorption of nutrients; maintenance of fluid and electrolyte intake; and evacuation of waste products of digestion. The primary functions can be divided into 4 major modes and, correspondingly, 4 major modes of dysfunction: motility, evacuation, digestion, and absorption.

Therapeutic objectives of diseases of gastrointestinal tract are to remove the cause and to promote the return of normal digestive function as quickly as possible. However, the major part of treatment is symptomatic (e.g. correction of abnormal motility, or relief of distension, pain, or tenesmus); supportive (use of vitamins); and treatment of complication, if any (dehydration, electrolyte and acid-base imbalances, and reconstitution of ruminal flora).

The normal microbial fermentation process can be achieved, particularly during convalescent period, by providing a palatable and balanced ration and encouraging the animal to eat. Optimum use of molasses can provide a readily available energy source to stimulate ruminal digestion. A source of nitrogen for microbial protein synthesis and a number of co-factors (including minerals) are necessary for normal fermentative processes. Other factors for normal functioning of G.I. tract are the removal of soluble end-products, undigested materials, and gas, optimal intraruminal pH, a fluid medium essential for normal rumino-reticular fermentation; and active rumino-reticular motility. Some of the common ailments of alimentary tract are characterized.

STOMATITIS

It is a non-specific inflammation of the oral cavity including tongue (glossitis), palate (palatitis or lampas), and gums (gingivitis). Clinically, it is characterized by partial or complete anorexia and excessive salivation (ptyalism, sialosis, or polysialia). The oral lesions may vary from simple wound to the appearance of papules, vesicles, erosions, ulcers, heavy deposits, or abnormal growths.

ETIOLOGY

The causative agents of stomatitis may be either of physical, chemical, or infectious in nature.

A. The physical agents inflicting trauma at oral examination, medicine dosing with sharp edged instruments, accidental excess to hard metallic objects like nail or needles with feed, feeding on spiny plants, dental abnormalities, or drinking of hot water.

B. Chemical agents may include the oral administration of irritant drugs (chloral hydrate), licking of counter-irritants (mercury blister), accidental administration of acids or alkalies, systemic poisoning (furazolidone), prolonged medication with chemicals (arsenic, mecurials, and iodides), and chronic development of uremia.

C. Infectious diseases characterized by stomatitis include the followings;

i) Bacterial diseases, e.g. actinobacillosis, actinomycosis, or secondary infection caused by Sphaerophorus sp. and other bacteria.

ii) Viral diseases, e.g. vesicular stomatitis, viral diarrhoea mucosal disease complex, foot and mouth disease, rinderpest, bovine papular stomatitis, blue-tongue (sheep), contagious ecthyma (sheep), malignant catarrhal fever, rhinosporidiosis, and papillomatosis.

iii) Mycotic infection either caused by monilia spp., aspergillus spp., or candida spp. may also cause stomatitis.

iv) Parasites infestation such as plant lice and mites and larvae of many insects particularly during flowering in common fodders may cause acute (allergic type) stomatitis.

PATHOGENESIS

The local inflammatory response due to any of the causative agents described above varies from simple erythema or wound to the development of papules, vesicles, erosions, ulcerations, papilloma, phlegmon, necrosis, granulomas, or to the deposition of heavy velvet deposits in the oral cavity. Some of the lesions may cause pain and difficulty in either prehension, mastication, or swallowing of feed and hence may result in dysphagia or reluctance to eat. Local painful lesions also stimulate salivation. Excessive salivation for long period may lead to electrolytes and acid-base imbalances, and dehydration.

CLINICAL FINDINGS

The first clinical evidence of stomatitis is moderate to excessive salivation with or without frothiness. The saliva is usually clear, but may contain pus (bacterial infection), shreds of mucosa, or some blood (oral wound). The affected animals will stand with open mouth accompanied champing or chewing movements. Partial or complete anorexia due to dysphagia is an other common sign of oral lesions. Fever and signs of toxaemia, if present, is an indications of systemic infectious disease. Other signs of any specific disease listed above should be looked for their diagnosis.

Various types of oral lesions could be seen at physical examination. These may include the development of vesicles in the form of thin walled elevated areas, 1-2 cm in diameter, filled with clear serous fluid, which at their rupture leave sharp edged, shallow ulcers. Erosions are shallow, usually discrete, areas of necrosis, readily seen in the early stages of infections. They tend to occur most commonly on the lingual mucosa. Necrotic tissues may remain in-situ but are usually shed off leaving a very shallow discontinuity of the mucosa with a dark red base. Ulcerative lesions penetrate more deeply to the lamina propria. Catarrhal lesions are in the form of diffuse inflammation of the mucosa and are usually caused by chemical or physical agents.

Lesions in the mycotic infections are in the form of heavy, white, velvety deposit with little obvious inflammation or damage to mucosa. Gangrenous lesions are characterized by rapid and massive destruction of the tissue with foul odor from breath.

CLINICAL PATHOLOGY AND NECROPSY FINDINGS

Swaps from oral mucosa may be taken for the isolation of causative bacteria or transmission experiment, or serological investigations in viral infections are recommended. Oral lesions in stomatitis are not usually fatal however, other lesions should be looked for the identification of any systemic involvement.

DIFFERENTIAL DIAGNOSIS

Presence of oral lesions and ptyalism is a good indication of stomatitis. However, each etiological agent listed above, needs to be differentiated

by careful examination of lesions, clinical pathology, and postmortem findings. Systemic poisoning with insecticides, chemicals, or toxic plants are also associated with hyper-salivation. But a history of accidental excess, careful examination for the source of poisoning, or gut assay may help to identify the cause of illness.

TREATMENT

Following line of treatment should be adopted for stomatitis.

1. Atropine sulphate, @ dose of 0.1-1 mg/kg, IM or SC route is a standard treatment for hypersalivation.

2. Apply mild, non-irritant antiseptics dressings on the oral mucosa. Examples include 2 per cent boric acid in glycerine, 1-2 per cent sulfonamide in glycerine, 1-2 per cent iodine-glycerine, weak solution of potassium permeganate (1:10,000), or 2 per cent solution of copper sulfate.

3. Indolent ulcers require curettage or cauterization with either 4 per cent silver nitrate solution or Tr. iodine. Hydrogen peroxide 2 per cent helps cleaning of wounds and early recovery.

4. Gangrenous and proliferative type stomatitis require debridement and surgical excision of the lesions followed by routine treatment.

5. Use of systemic antibiotics or sulfonamides is necessary for primary or secondary bacterial infection. Antihistaminic drugs in cases of allergic causes and antifungal antibiotic in fungal stomatitis should be used in combination of symptomatic treatment.

6. High doses of vitamins-C and B-complex especially Niacin, seem to hasten recovery.

7. Dextrose-Normal saline or Ringer's solution are also recommended to prevent dehydration and electrolyte imbalances in long standing cases of ptyalism.

8. Fluids or semi-solid palatable feed (barley, rice gruel) should be offered during and following recovery period. It may be fed by stomach tube if oral feeding is not possible.

9. Animals with any infectious diseases should be handled with proper care because of danger of their transmission to other susceptible animals.

PHARYNGITIS

It is the inflammation of pharyngeal mucosa and submucosa, caused by a variety of agents, and characterized by painful swallowing, cough, salivation, and occasional regurgitation of food or water through nostrils while eating or drinking.

ETIOLOGY

Pharyngitis is a common condition in horses and dogs, associated with many viral and bacterial diseases. In other animals it is usually seen as an extension of lesions from stomatitis or esophagitis. The etiological agents of pharyngitis may include physical injuries while faulty drenching, passing of stomach tube or probang having rough edges, accidental eating of metallic objects or bones, and intake of hot or very cold water or food, and sometime excessive barking (in dogs).

Among the infectious diseases actinobacillosis, strangles, infectious bovine rhinotracheitis, pasteurellosis, tuberculosis, Kennel cough (dogs), para-influenza virus and rhino virus infections (horse), and feline rhinotracheitis virus infection are some examples.

PATHOGENESIS

Depending upon the causative agents the inflammatory response may vary from catarrhal, diphtheric, phlegmonous, ulcerative, to suppurative type. The resultant response to inflammation will be painful swallowing (dysphagia) and/or disinclination to eat. There could be physical obstruction in passing of feed and/or attempts to eat are usually followed coughing and regurgitation through nostrils, especially if swelling is very severe. Other patho-physiological mechanisms are the same as in cases of stomatitis and esophagitis.

CLINICAL FINDINGS

Partial or complete anorexia, excessive salivation, pain and coughing at palpation of throat, and regurgitation of feed through nostrils are diagnostic features of pharyngitis. Fever accompanied by signs of toxaemia may be seen if there is systemic involvement. Inflamed lymph nodes (parotid, retropharyngeal) can be seen or palpated at throat region in some specific diseases (strangles). Aspiration pneumonia is a common complication of pharyngitis.

CLINICAL PATHOLOGY AND NECROPSY FINDINGS

Nasal or throat swabs may be helpful in identify the causative agent. Varying degree of inflammatory response in other organs and swollen lymph nodes at throat region can be seen at post-mortem examination.

)IAGNOSIS

haryngitis should be differentiated from pharyngeal paralysis and ostruction) where former is characterized by absence of local pain nd inability to swallow food, and later by complete inability to wallow feed and water, regurgitation through nostrils, and coughing. egurgitated material from rumen (in acute impaction) will contain artially digested food particles mixed with fluid containing ruminal ora, compared with undigested whole food particles and absence of uminal flora in regurgitate due to pharyngitis.

REATMENT

he line of treatment should include the following steps;

Specific treatment directed toward the primary disease if known. or example, antibiotics or sulfonamides by parenteral routes for the eatment of primary or secondary bacterial infection.

Local use of antiseptics (see stomatitis), applied with a long swab at ıe throat region. Local application of Mandel's paint is a common all me therapy.

)rmula of Mandel's paint; Iodine (1 gm), Potassium Iodide (2 gm), eppermint oil (0.5 ml), and Glycerine (60 ml).

A 10 per cent sodium iodide, @ dose of 100-300 ml/LA, IV, or same se of potassium iodide, PO, can reduce inflammatory response rticularly of the lymph nodes in the chronic cases (e.g. tinobacillosis). Lugol's iodine solution, @ dose of 300 ml/LA, through drip may be used for the same purpose.

Soft palatable or semi-solid feed is recommended during the period painful swallowing. Intravenous fluids may be given in cases where al route is completely blocked and dehydration is evident.

Use of sedatives (Dipyrone, IM, or 6 per cent chloral hydrate rough IV) may be recommended for reducing pain in severe cases.

Expectorants may be used for reducing cough if present. For dogs a mbination of the following drugs may be used as an expectorant rup. These are ammonium chloride (1 gm), ammonium carbonate .5 gm), camphor (200 mg), Ext. Belladonna (1 ml), and syrup (30 ml). ake 2 doses to be given in a day.

r large animals (Horse, cattle, and buffalo) use the following drugs the same purpose. Ammonium chloride (15 gm), ammonium rbonate (10 gm), potassium iodide (10 gm), pulv. glycyrrhiza (30 gm), linseed (30 gm), and treacle (300 gm). Make 2 doses to be given i a day.

7. Antihistaminic or anti-inflammatory drugs can be given in order to reduce acute inflammation and edema.

ESOPHAGITIS

It is the inflammation of esophagus, characterized by signs of esophageal spasm and obstruction, pain at swallowing and palpation, and regurgitation of feed and water.

ETIOLOGY

It is a common condition in horses and is mainly caused by exposure of animals to irritant chemicals (e.g. acids, alkalies, red iodide of mercury). Physical causes include traumatic injury to esophagus while passing of stomach tube or probang and intake of metallic objects. Common infectious diseases associated with esophagus are bovine viral diarrhea, malignant head catarrh, infectious bovine rhinotracheitis, feline calici-virus infection, moniliasis, and candidiasis. Death of Hypoderma lineatum larvae in the submucosa of esophagus in cattle has been reported to cause acute local swelling and subsequent gangrene.

PATHOGENESIS

Acute local inflammation and edema developing in response to any irritating etiology may result in increase in muscle tone and functional disturbances in the part of organ involved. Systemic involvement may not be evident in mild and local cases, but in long standing cases, signs of general body involvement will be due to lack of feed and water intake and aspiration of regurgitate in the lungs.

CLINICAL FINDINGS

The incidence of esophagitis is grossly underestimated because of the relatively inaccessibility of esophagus externally for clinical examination. Dysphagia (difficult swallowing), salivation, spasms of esophageal and cervical musculature, extension of head and neck, and hematemesis (hemorrhagic vomitus) are some clinical evidences of acute esophagitis. Affected animals may refuse to eat or drink, and if they do so, may regurgitate (through mouth or nostrils) soon after swallowing attempts. Palpation at the jugular groove will elicit pain. Sharp foreign bodies may lacerate or puncture the esophageal wall leaving a fistulous tract. Perforation of the thoracic wall may lead to fatal pleurisy. Stenosis of the lumen can occur due to part of the healing process following extensive lesions.

NECROPSY FINDINGS

The development of lesions within the esophageal lumen may be from patchy local to diffuse type. The lesions could be in the form of hyperemia, edema, hemorrhages, catarrhal, ulcerative, erosive, and/or necrotic types, according to the etiology.

DIFFERENTIAL DIAGNOSIS

Pharyngitis is usually confused with esophagitis, where regurgitation is a common sign, but is usually associated with coughing. Complete esophageal obstruction is characterized by complete inability to swallow feed and even water, presence of visible swelling at a particular site (in cervical obstruction), and development of bloat (in ruminants). Passing of stomach tube or probang can help to differentiate esophageal obstruction.

TREATMENT

1. Complete esophageal rest in the form of withholding feed and water for 2-3 days and IV administration of physiological fluids often relieves the condition.

2. Administration of sedative, analgesics, and anti-inflammatory drugs to control pain and reducing swelling during acute stage of disease. These may include IM or IV use of meperidine, dipyrone, salicylates, or chloral hydrate.

3. Corticosteroids and atropine sulphate may be used for reducing acute phase inflammatory response and spasm, respectively.

4. Physiological solutions, such as normal saline, dextrose-saline, or Ringer's solutions need to be given to combat dehydration and electrolyte imbalance, particularly in prolonged cases. In case of excessive salivation a 7.5 per cent solution of sodium bicarbonate may be recommended to combat secondary acidosis.

5. Use of antibacterials are recommended in cases of infectious diseases or for the control of secondary bacterial infections.

6. A soft palatable nutritious feed will keep the animal healthy and ensure early recovery.

X ESOPHAGEAL OBSTRUCTION
Syn: CHOKE

It is the obstruction of esophagus due to any solid food masses or foreign objects and is characterized clinically by inability to swallow, regurgitation of food and water, severe distress, and bloat (in ruminants).

ETIOLOGY

Choke is the most common esophageal disorder seen particularly in horses. It may develop by eating of material such as ears of corn, whole potatoes, turnips, apples, onions, or dry hay boluses. This condition is more seen in greedy or hungry animals fed after a long period of work. The predisposing factors are narrowing of the lumen diameter due to previous lesion in the esophagus, anchoring of foreign material such as nail or pieces wire in the esophageal wall, or by external pressure of inflamed structures (papilloma or neoplasia) surrounding the esophagus. This condition also develop due to inflammation of lymph nodes in mediastinum or at the base of lung (as in tuberculosis). The condition has been reported to develop in chronic esophagitis, thymoma, or carcinoma at cardiac region of stomach in horses.

PATHOGENESIS

There is inability to swallow or regurgitate food due to physical blockage of esophagus. At the site of obstruction the esophagus wall become distended leading to spasm (especially in horses). In ruminants, bloat usually develop due to inability to belch. Persistence of obstructing material in the esophageal lumen may cause ischemic necrosis and later stenosis of lumen diameter following healing. Excessive salivation may end up acid-base imbalance (acidosis) and dehydration. Attempts to drink or eat may end up with aspiration pneumonia.

CLINICAL FINDINGS

Signs of choke are not specific, however, affected animals suddenly stop eating and show anxiety, neck stretching, retching, and restlessness related to pain due to mucosal distension or disruption. There is forceful attempt to swallow of feed or water followed by regurgitation through nostrils (horses) or mouth (cattle). Ptyalism, chewing movement, and regurgitation of (whole) feed tinged saliva through nostrils (or mouth) are common. In horses coughing, swallowing movements, and signs of acute spasmodic type colic are also present. In ruminants, varying in degree of bloat is an additional sign, depending upon the duration and completeness of obstruction. Dilatation at cervical obstruction may be visible or palpable

externally. Obstruction at the thoracic region can be located by passing of probang or stomach tube. Persistence of obstruction for a long time may end up with death of the individual, either due to severe dehydration and/or aspiration pneumonia.

In chronic and partial oesophageal obstruction, the clinical signs are of moderate in nature. The persistence of mild tympany, difficult swallowing, and frequent expulsion of feed from nostrils are their indications. There is usually no obstruction in the passage of water.

CLINICAL PATHOLOGY

Laboratory tests are of no value in diagnosing stenosis, however, radiographic examination after a barium swallow is helpful to outline the site of stenosis, or obstruction. Impacted feed mass can be detected in the cervical region of esophagus by its dense granular pattern.

DIAGNOSIS

Esophageal obstruction can be mistaken for esophagitis in which local pain at palpation is more apparent and there is often an accompanying lesions of stomatitis and pharyngitis. Inflamed lymph nodes in tuberculosis and lymphomatosis may cause obstruction which usually accompanied by other signs of the diseases. Primary tympany can be differentiated from choke by easy passage of stomach tube in the former case. Chronic tympany needs to be differentiated from diaphragmatic hernia, traumatic reticulitis, vagus indigestion by the history and presence of other signs in later diseases. Botulism and rabies also have associated signs of swallowing difficulties but and need careful examination of patients.

TREATMENT

1. Ingestion of feed, water, or oral medication should be prohibited initially to reduce the risk of aspiration pneumonia.

2. Sedatives, analgesics, and muscle relaxant such as IV chloral hydrate (6 per cent), xylazine hydrochloride (Rompun), pentazocine, acepromazine (Sedastress), dipyrone, or use of atropine sulfate is necessary to relieve acute spasm particularly before handling of the animal.

3. Obstructing material at lower cervical and thoracic regions may be tried to push gently down into the stomach with probang or stomach tube following lubrication of the material with warm water. Objects at upper cervical regions may be pushed (milked) up and tried to be pulled out through mouth. An object near the diaphragm can be removed by rumenotomy.

4. In ruminants, evidences of bloat need urgent attention treatment with the use of trocar and canula or with rou antizymotics (see ruminal tympany).

5. Esophagotomy may be elected in cervical choke in either species should be resorted to only when usual methods of treatment which often is followed by complication of esophageal fistula, that cause further problem of healing, or if it does, may result esophageal stricture.

GASTRITIS
Syn: GASTRIC CATARRH

It is an inflammation of the wall of stomach, generating disorder motility and secretion, and is characterized clinically by signs dyspepsia and vomiting.

ETIOLOGY

Gastritis is a common problem in dogs and rarely in horses. ruminants abomasitis is equivalent to this condition. The kn causative agents of gastritis may be grouped as physical, chemi bacterial, viral, and metazoan agents.

a) Physical agents causing gastritis include the ingestion overheated or frosted feed, old spoiled feed, indigestible feed, non-f items such as bone, pieces of cloth, hairs, plastic toys, tennis b polythene bags, and large quantity of sand intake.

b) The chemical materials which are accidently ingested or given excessive doses and are known to cause gastritis are caustics irritant chemicals, arsenic, mercury, copper, phosphorus, lead, phe irritant drugs (aspirin in dogs), mycotoxins, and lactic acid (ruminants).

c) Infectious agents causing gastritis include canine distemper, can viral hepatitis, leptospirosis, and gastrointestinal parasiti Gastritis can also develop as a complication in some diseases such chronic pyelonephritis, and chronic renal failure.

PATHOGENESIS

Gastritis may develops as an independent condition, but is usua seen together with enteritis and is called gastro-enteritis. response of stomach to inflammation caused by any etiological ag includes i) an increased motility and ii) secretion. In acute gastr the major effect is on motility, whereas, in chronic gastritis

secretion. Moreover, in acute cases there is increase in peristalsis causing abdominal pain and rapid emptying of the stomach either by vomiting or via the pylorus in animals unable to vomit. In chronic cases there is increase secretion of mucus which does protect the mucosa but also delays digestion and permits putrefactive breakdown of the ingesta which may favor inflammatory process. In these cases motility is not reduced, rather stomach contractions equivalent to hunger pain result in depraved appetite.

CLINICAL FINDINGS

Vomiting, abdominal pain, and depression are the cardinal signs in acute gastritis. Vomiting may develops soon after eating or drinking and is in the form of repeated forceful retching movements. The vomitus may contains much mucus, sometime blood, and occasionally shreds of gastric mucosa. Pain is manifested in the form of restlessness, violent movements, and sweating (horses). Animals usually object to palpation of the anterior abdomen. Appetite is reduced but thirst may be present. Faeces is loose or severe diarrhea if accompanying enteritis. A dark and tarry feces is an indication of gastric hemorrhage. Dehydration, electrolyte imbalances, alkalosis, and progressive weight loss are some common complications of gastritis.

Chronic gastritis is characterized by periodic anorexia, depraved appetite (licking concrete, chewing dirt, eating non-food items), and sporadic vomiting. The vomitus contain much mucus. Signs of acute abdominal pain and dehydration are absent.

TREATMENT

Specific treatment should be recommended according to the cause. A general line of treatment includes the followings;

i) Nothing per oral. In acute gastritis withhold food for at-least 12-24 hours. This allows restoration of mucosal integrity and a more rapid return of G.I. tract homeostasis. It also helps minimizing fluid loss from vomiting, reduce gastric irritation, and remove the major stimulus to acid secretion.

ii) Vomiting and gastric secretions needed to be controlled by the using chlorpromazine hydrochloride (Largectil) or prochlorperazine.

iii) Fluid and electrolytes such as normal saline solution or Ringer's solution should be given to combat dehydration, and electrolytes and acid-base imbalances.

iv) Sedatives or antispasmodics drugs, such as meperidine, atropine sulphate, or Tr. belladonna may be recommended in case of acute abdominal pain.

v) If the poisonous material has recently been ingested, vomiting is encouraged and can be induced by using emetics (apomorphine). Emetics should not be given if corrosive materials, such as strong alkali or acids, has been ingested. Gastric lavage using 2 per cent sodium bicarbonate solution needs to be performed in order to remove irritant foreign material from stomach using a stomach tube.

vi) Gastric sedative, antacid, and protective drugs, such as kaolin, pectin, bismuth carbonate, magnesium carbonate or hydroxide, sodium bicarbonate, calcium carbonate, aluminum salts, may be of value for protecting gastric mucosa and reducing secretions. Aluminum salts bind selectively to injured mucosa and form a local protective layer. It also bind to pepsin and bile and prevent them to cause further damage.

vii) Antibiotics and anthelmintics therapy needs to be given if the cause identified necessitated.

viii) Soft, laxative, non fibrous, low-fat, and highly nutritious diet should be utilized at post-fasting and during convalescence period for easy passage and less damaging to the inflamed mucosa. These include rice gruel and bran mashes. Mild laxative feed and water may be started soon after recovery from initial signs.

A routine formula of gastric sedative powder consists of calcium carbonate (1 part), sodium bicarbonate (1 part), magnesium carbonate (1 part), and bismuth carbonate (1 part).

(For dogs, @ dose of 2-4 gm/SA, PO, t.i.d., and for horses, 30-60 gm/LA, PO, b.i.d. in rice gruel).

In ruminants (and horses) rumenitoric drugs may be given during the convalescent period. These include magnesium (sodium) sulfate (200 gm), pulv. Nux vomica (20 gm), and pulv. Gentian (20 gm).

NB: Oral administration of certain drugs containing salicylates, phenylbutazone, and corticosteroids are contraindicated in gastritis.

ix) Supportive therapy should include the use of vitamins particularly of B-complex.

Calcium carbonate = 1 part
Sodium carbonate = 1 part
magnesium " = 1 part
magnesium carbonate = 1 part
bismuth carbonate = 1 part

For chronic gastritis treatment is mainly directed toward the predominating symptoms. Rumen transfaunation should be recommended in all long standing anorexic cases for the normalization of digestion processes.

ACUTE VOMITING

Vomiting may be the outcome of primary gastrointestinal diseases or secondary G.I. effects due to systemic diseases. Isolated incidence of vomiting is rarely considered significant in dogs and cats, however, if protracted vomiting occurs, alterations in the patient's fluid, electrolyte, and acid base balance can cause a life-threatening situation. The initial management of vomiting should be standardized regardless of the etiology. It is important to have knowledge of the pathophysiology, metabolic abnormalities, and pharmacological control of vomiting.

Regurgitation; It is the reflux ejection of previously swallowed ruminal material into the mouth. It is a normal phenomenon in ruminants. Any blockage of rumino-reticular outflow by foreign body or diaphragmatic hernia can cause ruminal distension and excessive regurgitation following eating. An esophageal foreign body can cause irritation and also result in regurgitation. Vomiting or forced regurgitation in ruminants (and horses) is rare and is commonly associated with many toxic conditions, choke, peritonitis, acute abomasal obstruction, traumatic reticulitis, etc. A detail portrait of vomiting in small animals is described.

ETIOLOGY

There are variety of kinds of disorders and stimuli causing vomiting in animals. Some of them include; sudden change in diet, ingestion of foreign material (garbage, grass or plant leaves, food allergy, intolerance to many drugs (cardiac glycosides, arsenic compound, non-steroid anti-inflammatory drugs, anticholinergic), toxins (lead, ethylene glycol, fertilizers, herbicides), metabolic disorders (diabetes mellitus, renal or hepatic diseases, acidosis), mineral imbalances, gastric disorders (including inflammation, parasites, hypermotility, ulcers, polyps, neoplasia, dilatation), enteritis, intestinal obstruction, paralytic ileus, pyelonephritis, prostatitis, pyometra, hernias, and heartworms.

PATHOPHYSIOLOGY

Vomiting Center (VC); The medullary VC is the final pathway from which efferent impulses arise to initiate vomiting. Vomiting cannot occur without an intact vomiting center. Thus, drugs which act at this site, such as phenothiazine, may be used to symptomatically treat refractory vomiting from any cause. There are 4 sources of afferent input into the VC. These are;

a) The chemoreceptor trigger zone (CRTZ),
b) Higher brain (CNS) centers,
c) The vestibular apparatus (VA) receptors, and
d) Peripheral sensory receptors.

a. Chemoreceptor Trigger Zone; The CRTZ is connected to the VC by neural pathways and is functionally devoid of the blood brain barrier. This allows blood-born substances, such as bacterial or uremic toxins, cardiac glycosides and apomorphine, to initiate vomiting. Toxins in the CSF also stimulate the CRTZ in a similar manner. Stimulation of the CRTZ results in the release of dopamine from neurons that serve as an intermediate afferent station for stimulation from the VA. Antiemetics which act at the CRTZ will reduce vomiting induced by this mechanism.

b. Higher Brain (CNS) Center; Areas of the cerebral cortex and limbic system also stimulate the VC. The mechanism is unclear, but it is known that inflammation within the CNS, increases in intracranial pressure, and head trauma can induce vomiting. In humans, psychogenic factors such as fear, stress, emotional excitement, visual and olfactory stimulation result in stimulation of the vomiting center. The role of psychologic factors in animals in unknown.

c. Vestibular Apparatus Receptors; Stimulation of receptors in the semicircular canals of the vestibular system results in vomiting by stimulation of VC. The receptors in the vestibular system are stimulated by inflammation in the semicircular canals or rotation and unequal stimulation of the labyrinth. Afferent impulses travel from the vestibular system down the acoustic nerve (CN VII) and the stimulation of the CRTZ via the histamine H1 receptors. The CRTZ then stimulates the VC. Some of the efferent impulses from the vestibular apparatus bypass the CRTZ and pass directly to the VC. Cholinergic and histaminic (H1) neurotransmitters are involved in these pathways. Antihistaminic drugs and scopolamine appear to have a direct effect on neural pathways arising in the VA and are effective therapy for vomiting caused by these disorders.

d. Peripheral Sensory Receptors (PR); Peripheral sensory receptors with afferent pathways to the VC are located throughout the body. Most are located in the abdominal viscera (G.I. system, urogenital system, peritoneum) and impulses are conveyed via the sympathetic and vagal nerves. Chemical stimulation, distension of abdominal

organs, and inflammation initiate vomiting through this pathway. Acetylcholine is the major afferent neurotransmitter. Receptors in the pharynx transmit their impulses through cranial nerve IX.

Once stimulate the VC initiates the stages of vomiting by efferent impulses that pass along somatic pathways of the vagus and sympathetic nerves. Acetylcholine is the major neurotransmitter involved in efferent impulses from the VC.

THE ACT OF VOMITING

The vomiting process consists of three active stages, nausea, retching, and vomiting.

Nausea; It is often described by the owner as restlessness, licking of the lips, changing posture frequently, and excessive salivation or swallowing. These signs are suggestive of widespread autonomic discharge. During nausea gastric tone is reduced. Reverse peristalsis in the duodenum occurs in dogs and cats during nausea stage which allows the passage of intestinal contents into the stomach. Thus some animals will have bile stained vomitus. These promontory events may exist for several seconds to several minutes.

Retching; The next phase of vomiting is the retching stage. During this stage the patient attempts to overcome anti-reflux mechanisms. As the retching begins the animal takes deep inspirations. The glottis closes, the intrathoracic pressure decreases, and the abdominal muscles contract. This results in an increase in intra-abdominal pressure and decrease in intra-thoracic pressure. As the pyloric end of the stomach contracts and the fundic area relaxes, gastric contents enter the relaxed esophagus. At this time, however, the upper esophageal sphincter remains constricted and the abdominal contractions are slow. As the abdominal muscles then relax the ingesta re-enters the stomach from the esophagus. This cycle repeats itself every few seconds to minutes. The patient during this phase usually has its mouth either opened or closed, its head is lowered, and becomes oblivious to nearby persons, or events. This stage last from seconds to minutes.

Active Vomiting; The final stage is active vomiting which requires the proceeding two phases and occurs during an active retching episode while the esophagus is full of gastric or duodenal contents. As a strong contraction of the abdominal musculature occurs a rise in intrathoracic pressure results and forces the contents through the upper esophageal sphincter into the oral cavity and out of the mouth. After passage of material the muscles relax and a period of deep rapid respiration occurs. This sequence of events may be repeated at

intervals of minutes to hours if the inciting cause remains. During vomiting the most distal part of the small intestine is not active. The colon, however, often stimulated allowing defecation to occur during vomiting.

METABOLIC CONSEQUENCES OF VOMITING

Vomiting results in loss of water and electrolytes, containing sodium, chloride, hydrogen, and potassium ions.

Hydration; Dehydration is a frequent problem in the vomiting patient. It results from fluid loss via vomiting and the inability to adequately take in sufficient oral maintenance fluid requirements. Hydration is clinically estimated through skin turgor assessment and mucous membrane moisture. In per-acute fluid loss the patient may clinically appear normal until fluid shifts from the peripheral tissues to the intravascular fluid compartment. However, even though the patients appear clinically normal the PCV is elevated in patients with per-acute fluid loss from vomiting. Dehydration associated with adequate perfusion is often due to slow fluid loss with adequate cardiovascular compensation.

Potassium; Potassium (K) deficiency is the most frequent electrolyte abnormality observed in the vomiting patient. It occurs both through minor losses in vomiting and more importantly from urinary losses as the kidney responds to maintain volume expansion. The inability to adequately consume maintenance water needs will complicate K deficiencies. A total K deficit can cause muscle weakness, neurologic alterations, and cardiac arrhythmias.

Sodium; Serum sodium (Na) concentrations are normal in the majority of patients with vomiting as both sodium and water are lost in the vomitus. Hyponatremia is occasionally found indicating a disproportionate loss of Na compared to water or water retention. Recent studies indicate that nausea and vomiting stimulate release of antidiuretic hormone (ADH), presumably via a reflex mechanism. Excessive ADH release could contribute to hyponatremia and oliguria in some patients. Hypoadrenocorticism or chronic renal failure should also be considered. Hyponatremia reflects a loss of solute free water and is found in diabetes mellitus, severe hyperthermia, or chronic renal failure.

Acid-base; Patients with mild vomiting episodes usually have normal blood pH. Dehydration or poor perfusion may cause metabolic acidosis. Alkalosis is a rare finding and suggests pyloric outflow obstruction.

DIFFERENTIAL DIAGNOSIS

History is important to rule out different types and origin of vomiting.

Regurgitation is a passive expulsion of food without nausea, with passive abdominal efforts, and mainly associated with pharyngeal or esophageal diseases. X-ray examination may help in diagnosis of esophageal dilatation, foreign body in stomach, neoplasia, inflammatory conditions, or intestinal obstruction.

Other medical problems (gastritis due to excessive use of aspiring, corticosteroids, digoxin, antibiotics, or access to garbage, bones, and mouldy food) should be ruled-out to understand the cause.

Vomiting that occur short after eating suggests inflammation of stomach. Passing out of large quantity of undigested food 4-6 hours after eating is suggestive of gastric retention (motility disorders). Projectile vomiting is an indication of pyloric outflow obstruction. Blood in the vomitus should always be taken seriously and implies esophageal, gastric, or duodenal erosive or ulcerative diseases. Fetid green or brown fluid is usually duodenal in origin. Yellow or green vomitus indicates biliary reflux. The presence of diarrhea implies more generalized gastro-intestinal involvement.

TREATMENT

1. Nothing per os. Animal with acute vomiting should be fasted for at least 12-24 hours. If no vomiting during the time, small quantity of fluid may be offered. If it is tolerated, then soft liquid diet may be given in small quantity. Diet high in fat or protein should be avoided.

2. Maintenance of hydration status. Vomiting animal may require a larger volume of fluids for rehydration and maintenance than expected. Physical parameters, PCV, and total protein may be determined to evaluate hydration status. A balance electrolyte solution such as Ringer's solution is given initially. Dehydration associated with poor perfusion requires volume replacement in 1-3 hours. Estimated volume of administration fluid to correct fluid deficit is calculated by the following;

percentage dehydration X killo gram of body weight = litters of fluid (to be administered)

Normal hourly maintenance (estimated at 3 ml/kg) and ongoing fluid losses are added to the fluid deficit for the total volume administer. The fluid losses into G.I. tract can continue during rehydration. Packed cell volume, total protein, and central venous pressure should be assessed frequently to assure that fluid administration is adequate.

3. Electrolytes to be administered are sodium, potassium, chloride and bicarbonates. Serum potassium values <3.0 mEq/L call for potassium supplementation in the initial therapy, especially in acidosis. Hyperkalemia normally responds to proper fluid therapy and specific therapy is not needed.

4. The acid-base imbalance. The fluid replacement and prevention of ongoing losses through the G.I. tract are the primary means of treating acid-base imbalances due to vomiting. Rarely, IV administration of sodium bicarbonate may be required to treat severe metabolic acidosis. This is done slowly using the following formula;

0.3 X kg body weight X (18 - measured plasma bicarbonate value) = mEq/L of sodium bicarbonate to be administer over a minimum of 20-30 minutes.

When alkalosis is present, it is treated by volume replacement with chloride containing electrolyte solutions, such as normal saline.

PHARMACOLOGIC CONTROL OF VOMITING

Antiemetics are regularly used as primary or symptomatic therapeutics in veterinary patients. The use of antiemetics for primary therapy involves the prevention of vomiting that is induced by vestibular system (motion sickness in human) or CRTZ (in cancer therapy) stimulation. The use of antiemetics as symptomatic therapy is used for a wide variety of conditions to mask the clinical sign of vomiting. Their use is indicated when vomiting is so protracted that hydration status, electrolyte and acid-base balances is difficult to maintain. As mentioned before, since vomiting is a clinical sign and not a diagnosis, it is important to always attempt to identify the underlying cause. Potential problems with the use of antiemetics for symptomatic therapy can develop when their use may delay the identification and treatment of serious disorders, such as renal failure, liver failure, pancreatitis, or intestinal obstruction. Therefore, the therapeutic goal in the management of vomiting animals should be to identify the primary disorder.

Central acting antiemetics are those that inhibit vomiting by blocking impulses within the CNS. These drugs work as the VA, CRTZ, VC, or at a combination of these sites.

Receptors in the VA are stimulated by local inflammation and motion. Efferent from the vestibular system synapse primarily at the vomiting center, however, some report that a portion of the afferent nerves travel to the CRTZ which then sends input to the vomiting center. The

neurotransmitters involved in this pathway are muscarinic cholinergics and histamine (H1 receptor). The drugs used to inhibit vomiting induced by the VA are antihistamines and anticholinergics.

Antihistamines; The antihistamines block transmission of afferent impulses at the VA. They only block vomiting that is associated with motion sickness or vestibulitis. The antihistamines also have anticholinergic properties. It is thought that the anticholinergic activity of these drugs contribute to their effectiveness as antiemetics. Examples are; Diphenhydramine (Benadryl), @ dose of 2-4 mg/kg, PO, SA, 8 hourly. Dimenhydrinate (Dramamine), @ dose of 8.0 mg/kg, PO, SA, 8 hourly. Meclinzine (Bonnie), @ dose of 12-25 mg/kg, PO, SA, 24 hourly.

These can also be used as prevention of motion sickness associated vomiting. Adverse effects associated with this group are drowsiness, dryness of the mouth and respiratory tract, and headaches (man). Anorexia, diarrhea, or constipation have also been reported.

Anticholinergics; Anticholinergics have been used in the past as broad spectrum antiemetics. The effect of these drugs appears to be predominantly peripheral where they decrease peripheral receptor afferent stimulation. Centrally, their action appears to have an action at the VA and the vomiting center. Scopolamine has been shown to prevent motion sickness in dogs through its effect on the VA.

Phenothiazine; Phenothiazine are broad spectrum antiemetics that suppress both CRTZ (at lower doses) and the vomiting center (at higher doses). These drugs are generally thought to be the most effective antiemetics available for suppressing vomiting in a wide variety of disease conditions. The phenothiazine are generally thought of as antipsychotic drugs that are dopamine antagonists, but also have weak to mild antihistamine and anticholinergic properties. The major antiemetic effect of the phenothiazine is thought to be the direct blocking of dopamine release by the CRTZ thus inhibiting stimulation of the vomiting center. The most effective and commonly used phenothiazine in veterinary medicine as antiemetics are; Chlorpromazine (Largectil), @ dose of 0.1 mg/kg, IV, 4 hourly, or 0.5 mg/kg, IM, 8 hourly. Prochlorperazine (Compazine), @ dose of 0.5 mg/kg, IM or IV, 6-8 hourly. Perphenazine (Trilafon), @ dose of 0.5 mg/kg, IM, 6 hourly.

The first two drugs are also available in parenteral and oral forms. The adverse effect associated with phenothiazine include hypotension due to the peripheral alpha-adrenergic blocking activity that results in vasodilation. In addition, these drugs are negative inotropes at

therapeutic dosages. With IV use they result in hypotension and reflex tachycardia. Therefore, these drugs should be used with extreme caution in hypovolemic patients. It is recommended that a IV fluid by initiated prior to the IV administration of phenothiazine. These drugs may also interfere with thermoregulation in some patients resulting in hypothermia. Xerostomia may occur due to their mild anticholinergic properties.

Trimethobenzamide; Trimethobenzamide is a central acting antiemetic that is effective in blocking the vomiting reflex at the CRTZ. The mechanism of action is unknown but the drug directly inhibits the CRTZ. The drug does not inhibit impulses to the vomiting center and does not act peripherally to decrease the input from the peripheral visceral receptors. Structurally, it is related to antihistamines, but exhibits only weak antihistaminic activity. Since it is only effective at one site (CRTZ) it is less effective than the phenothiazine, but is generally associated with fewer adverse effects than phenothiazine. Therefore, it is the drug of choice for vomiting that is mediated through the CRTZ. The side effects associated with trimethobenzamide are infrequently reported. Occasionally, the human patients may exhibit depression, drowsiness, and disorientation. The suggested dosage is 3 mg/kg, IM, and can be repeated at 8 hours intervals.

Metoclopramide; Metoclopramide is frequently used drug in veterinary medicine that has both central and peripheral effects on the G.I system. The drug is derivative of procainamide. Centrally, the drug inhibits vomiting by its antidopaminergic activity at the CRTZ. Peripherally, it appears to sensitize the smooth muscle of the distal esophagus, stomach, and proximal small intestine to acetylcholine. Within the esophagus the actions of this drug result in increased lower esophageal pressure and an increase in esophageal contraction strength. These actions result in an increase competence of the lower esophageal sphincter decreasing esophageal reflex. Within the stomach the drug increases gastric antral contractions, promotes relaxation of the pylorus and increases smooth muscle contractions in the proximal small intestine. These actions accelerate gastric emptying and small intestine transit time which counter the events that precede vomiting. Therefore, they are said to have a peripheral antiemetic effects.

Because of these actions metoclopramide has been advocated to control persistent vomiting associated with functional gastric retention, gastro-esophageal reflex, or inflammatory gastrointestinal disorders, as well as situations where the vomiting is due to CRTZ stimulation. The half life of this drug is short which means that

therapeutic blood levels are not maintained with IM or IV injections. Therefore, it needs to be administered orally, SC, or as a continuous IV infusion (and protected from light). The dose is 0.2-0.5 mg/kg, PO, SC, repeated 8 hourly, or 1.0-2.0 mg/kg, IV infusion over 24 hours. Due to effects on CNS and as anticholinergic, this drug should not be used in combination with phenothiazine and with anticholinergics and intestinal blockade.

NB: The following drugs used in for treating vomiting may be used in the treatment protocol.

Atropine (0.04 mg/kg, SC),
Chlorpromazine (0.2-0.4 mg/kg, SC),
Dimenhydrinate (Dramamine, 8 mg/kg, PO),
Diphenhydramine (Benadryl, 2-4 mg/kg, PO,IM),
Isopropamide (Darbazine, Darbid, 0.2-0.4 mg/kg, PO), Metoclopramide (0.2-0.4 mg/kg, PO, SC, IM),
Prochlorperazine (Darbazine, Compazine, 0.5 mg/kg, SC, IM),
Propantheline (Pro-Banthine, 0.5-1 mg/kg, PO), and
Trimethobenzamide (3 mg/kg, IM).

EQUINE COLIC
Syn: ABDOMINAL PAIN

Equine colic include a group of conditions manifested by signs of visceral pain. Horses can exhibit signs of colic frequently since this species appears to have a low threshold to pain. In terms of intensity, the pain could be per-acute (violent and severe), acute (severe), sub-acute (moderate), or chronic (mild) in nature. The alimentary tract pain produces a wide diversity of signs which also reflect their underlying pathogenesis. Most horses will recover from an episode of (non obstructive) colic with conventional medical treatment, but this condition is still the largest single cause of death in horses. The equine colic can broadly be classified into 5 types;

1) Spasmodic colic; This type of colic develops due to increase in bowel motor activity and the signs of pain are acute to per-acute and intermittent type, i.e. wax and wane at frequent intervals.

2) Impactive colic; This type of colic develops when bowel lumen is much distended and its motility is reduced due to impaction of bowel with dry, and (partial) digested feed contents. The signs of pain are sub-acute and continuous type.

3) Obstructive colic; In this type of colic the passage of ingesta obstructed by any major injuries to the bowel (e.g. torsi intussusception, strangulation, or paralytic ileus). This condition also results from obstruction in the vascular supply to any bo segment due to a variety of causes. The pain is continuous and usu per-acute to acute in nature.

4) Flatulent colic (Intestinal tympany); This type of colic devel because of over distention of bowel lumen due to accumulation gases, yielding pain which is of acute and (intermittent initia continuous type.

5) Idiopathic colic; In this type of colic there may be no obvious ca or lesions even established at laparotomy to account for development of pain experienced by the horse.

ETIOLOGY
The etiology of colic can be classified into either (a) colic primarily G.I. tract origin. Examples are intestinal, cecal, or colonic tympa (flatulent colic or intestinal tympany); Intestinal hyper-motility a muscle spasm (Spasmodic colic); Impaction of ileum, cecum, colon, ileocecal valve with feed (Impactive colic); Acute intestinal or pylo obstruction (Obstructive colic); Gastric dilation; Gastric ulcers a retention of meconium (foals); Intestinal foreign bodies (sand col enterolith, phytobezoar); Enteritis caused by bacterial, viral, parasitic (strongyle) infestation; and Diaphragmatic, umbilical, inguinal hernias.

(b) Causes of colic originating from other organs (false colic Examples are peritonitis, parturition, urolithiasis or other causes retention of urine, cholelithiasis, verminous mesenteric arteriti (thrombo-embolism), mesenteric neoplasia or abscess, acute hepatitis use of parasympathomimetic drugs, botulism, tetanus, anaphylaxis African horse sickness, and chemical or plant poisoning.

PATHOGENESIS
Generally speaking the pain receptors (nociceptors) are present in the superficial layers of skin, joint surfaces, periosteum, muscles, tooth pulp, and visceras. Stimulation of these nociceptors results in transmission of impulses through the spinal cord, thalamus, brain-stem, to cerebral cortex where the signals are processed. The end result is perception of pain which is also mediated by autonomic nervous signs i.e. alteration of heart rate, blood pressure, sweating, ventilation, and pupil size.

The primary causes of visceral pain include: i) Over distension of the wall of the stomach or intestines with fluid, gas, or ingesta, ii) Increased tension or pulling on the root of mesentery or supporting ligaments, iii) Local ischemia or infarction, and iv) Spasm of the visceral smooth muscles.

Intestinal distension with gas or ingesta is the most common cause of over stretching. This distension may be static (as in cases of impactive, obstructive, and flatulent colics), or transient, when local periodic distension occurs as a result of spasm or increased peristalsis of intestinal segment (spasmodic colic). Shock, dehydration, and electrolytes imbalance, especially following acute intestinal obstruction are other basis of clinical signs (and sometimes the fatal outcome).

CLINICAL FINDINGS

The clinical signs of each type of colic are variable. The pain may be intermittent (in the form of bouts of pain at frequent intervals followed by periods of relaxation) or continuous types. The general clinical signs of colic include tail swishing, teeth grinding, pawing, stamping, restlessness, body stretching, looking at the abdomen (flank watching), frequent urination, protrusion of penis without urination, kicking at the belly (abdomen), frequent lying down and soon getting up, grunting, rolling, sweating, constant sitting in lateral recumbency, dog sitting posture, or sitting on haunches, and suddenly dropping to the ground in pain.

36-41 Normal

Horses with pain also develop evidences of an increase in heart rate with weak pulse and prolong capillary refill time. The mucous membranes are initially bright red (vasodilatory phase) followed by development of dark coloration (vasoconstrictive phase). If the problem is severe there may alter the cardiovascular integrity. Evidences of shock and dehydration are common in acute cases of flatulent colic or intestinal obstruction. In all visceral types colic; external palpation or pushing in the abdomen doest not elicit pain. Anorexia and depression often accompany these signs.

Causes of Death in Colic; Death may occur in colic is either due to rupture of stomach or intestinal wall resulting in deposition of large quantity of toxic ingesta into peritoneal cavity causing shock and death within few hours. In lingering cases of colic the cause of death may be a combination of exhaustion, autointoxication, anorexia, and reflex inhibition of cardiovascular functions.

Saw Horse Appearance

Hypokalemia in chronic

DIAGNOSIS

Hyperkalemia in Acute.

The primary aim of examination of animals suffering from colic should be to distinguish mild or uncomplicated cases of colic than those of potentially life threatening which require further surgery or intensive care. A rapid physical examination is demanded before commencing treatment of any kind. This includes:

a) History; A complete history of the nature and duration of clinical signs, feed management, defecation or urination, pregnancy, and premedication are important for the diagnosis and management of colic. For example, intermittent pain for the last 48 hours with minimum physiological abnormalities is less life threatening than continuous pain of 4 hours with deterioration of physiological parameters.

b) Physical examination; Observation about general outlook, palpation of abdominal pressure (over distension), auscultation of the presence or absence of intestinal movements (borborygmi), temperature, pulse, and respiration rates, color of mucous membrane, capillary refill time, passage of naso-gastric tube, rectal palpation of abdominal organs, and absence or presence of the faeces may help in establishing the etiology and possible prognosis. A gross examination of the faeces i.e. presence of blood, mucous, sand (checked by mixing the faeces in water), or odor may assist in diagnose.

c) Laboratory findings; It includes the estimation of packed cell volume and plasma proteins which can help to assess the hydration statu.. and vascular integrity. Paracentesis of abdominal fluid and its gross and microscopic examinations are necessary to differentiate peritonitis from other causes of colic (visceral or urogenital origin). If peritoneal fluid is grossly contaminated with faeces materials, rupture of viscus should be considered.

d) Exploratory surgery; It is sometime necessary to classify the cause of acute or persistent colic and extent of damage done. The technique will be discussed in surgery classes.

TREATMENT

The treatment of colic depends upon the nature of the cause, types of lesions, and severity of pain. A general line of treatment may include the followings;

A. Medical Therapy;

1. Use of narcotic-analgesics drugs. Examples are morphine or pethidine (@ dose of 2 mg/kg, IV or IM), or methadone (@ dose of 0.1

mg/kg, IV or IM). These drugs are under narcotic regulation so are not available in open market for routine use.

2. Use of sedatives-analgesics drugs. These types of drugs may normally be given even before proceeding for clinical examination to establish the cause (for diagnosis) in order to prevent self inflicting injuries. The common examples of sedative-analgesic drugs used in horses are; Opioids (act through CNS, and increase the tolerance to pain, but promotes constipation), such as pentazocine (lactate), @ dose of 0.5-1 mg/kg, IM, meperidine (HCl), @ dose of 1-2 mg/kg, IM, and butorphenol tartrate (Torbutrol). Other sedative-analgesic drugs commonly used are chloral hydrate (a drug of all time use, it truly has analgesic effects, but in larger doses, it has hypnotic effects), @ dose of 30-60 gm/LA, PO, or same dose as 6-10 per cent solution by IV route. Acepromazine (Sedastress- at present a drug of choice), may be given @ dose of 0.05-0.1 mg/kg, IV or IM. Hyoscine compound (Buscopan), used @ dose of 30-60 ml/LA, IV. Xylazine HCl (Rompun, most popular being sedative, analgesic, and muscle relaxant), to be used @ dose of 1.0 mg/kg, IM, and Promazine (in mild cases of colic).

3. Use of non-steroid anti-inflammatory drugs. These drugs may be used in mild cases of colic. Examples are phenylbutazone and flunixin meglumide, @ dose of 0.5-1 mg/kg, IM, (induce good analgesia for 6-8 hours).

4. Use of antispasmodic drugs. These drugs are used for the treatment of spasmodic colic, i.e. Atropine sulphate, @ dose of 15-30 mg/LA, IM or IV and Dipyrone (for mild cases of colic), @ dose of 10 mg/kg, IM or IV. (Both of these drugs should not be given together). Chlorodyne, @ dose of 6-8 ml/LA, PO, or Lignocaine 2 per cent, @ dose of 15-30 ml/LA, PO, can also be used for the same purpose.

5. Use of posmomimetic drugs, such as carbachol, @ dose of 2-4 ml/LA, SC, or neostigmine. These drugs are recommended 'only' in mild cases of intestinal impaction and following the administration of mineral or vegetable oil. These drugs should not be given in other types of colic, pregnancy, and peritonitis.

6. Use of lubricants and purgatives. These types of drugs are particularly recommended in cases of impactive colic. Examples of lubricants are mineral oil (liquid paraffin) or vegetable oil, @ dose of 2-4 liters/LA, PO. Examples of purgative are anthraquinone or dioctyl sodium sulfosuccinate, @ dose of 8-30 gm/LA, PO. Magnesium sulphate, @ dose of 200-400 gm/LA, PO, is good in treating sand colic.

7. Use of antizymotic drugs, such as turpentine oil, @ dose of 30-60 ml, PO, spirit ether nitrosi, @ dose of 30 ml/LA, PO, and formalin (10 per cent), @ dose of 15-30 ml/, PO, may be use in cases of intestinal tympany (flatulent colic).

8. In per acute cases of flatulent colic "Trocar and Canula" needed to be applied at the point of maximum distension, either at right, left, or both sides of abdomen.

9. Use of fluid and electrolyte therapy. For the treatment of dehydration, electrolyte imbalances, and metabolic acidosis large volume of fluid therapy (10-15 L) is necessary for early recovery. It may include the use of normal saline, lactate Ringer's solutions, or normal sodium bicarbonate solutions. Plasma volume expanders (Dextran 6 per cent, @ dose of 2-3 liters/LA, IV), or Gelatin polymer (Haemaccel, @ dose of 2-3 liters/LA, IV) may be used in debilitating and exhausted animals. In cases of dehydration and shock, NSAIDs may be added in the prescription.

10. Steroid therapy. In acute cases of endotoxic shock large doses of corticosteroids, eg. Dexamethasone @ dose of 1-2 mg/kg, IV., or Betamethasone, @ dose of 1-2 mg/kg, IV, should be recommended.

11. Use of specific therapy. Antibiotics may be used in cases of acute bowel obstruction, following surgical manoeuver, or in other cases where necessary. Anthelmintics (Ivermectin @ dose of 1 mg/5 kg, SC or IM), is recommended for the treatment of verminous colic.

B. Surgical intervention; It is indicative when ever there is,
i) Severe pain giving no response to analgesics,
ii) Shock as evident by weak and rising pulse rate over 80 beats per minutes,
iii) Increased CRT,
iv. Congested and dark mucous membrane,
v) Cold extremities,
vi) Absence of gut sounds,
vii. Marked distension of bowel, and
viii) High protein contents, RBCs and WBCs counts in the peritoneal fluid.

Surgery is contraindicated when there is;
i) High fever,
ii) Neutropenia or marked neutrophilia,
iii) Sever icterus,
iv) Evidence of enteritis, and
v) Evidence of extra-intestinal cause of colic not restored by surgery.

Commonly used colic drafts for horses include the followings;
For spasmodic colic; i) Atropine sulphate, 2 dose of 15-30 mg/animal, IM or SC. ii) Chloral hydrate (60 gm) and simple oil (300 ml), and given orally.

For flatulent colic; i) Chloral hydrate (60 gm), turpentine oil (30 ml), and simple oil (300 ml), and given orally.

For impactive colic; i) Chloral hydrate (60 gm), simple oil (1000 ml), given orally.

IMPACTIVE COLIC
Syn: IMPACTION OF LARGE INTESTINE

It is the most important colic type of horses. It develops due to impaction of large intestine with ingesta and characterized clinically by mild to moderate abdominal pain, depression, and constipation.

ETIOLOGY
A. Primary causes of impactive colic include feeding of low grade and indigestible roughage (old hay, sorghum, or bedding), molar defects resulting in poor mastication, greedy feeders, or feeding animal after longer intervals. Consumption of sand with hay or deliberate eating by foals (sand colic) may also cause this condition.

B. Predisposing or secondary causes of this colic are general debility leading to decreases intestinal motility, dehydration, poor or worn dentition, verminous mesenteric arteritis causing occlusion of blood supply to any segment of intestine, enterolith or fiber ball causing partial obstruction in the passage of ingesta. Foreign bodies such as pieces of rope, plastic bags, etc. finding their way to small colon and causing its severe impaction. Rectal paralysis developing occasionally at time of parturition or encephalitis (rabies) may also cause impaction/ constipation. Retention of meconium in foals is a common occurrence of impaction in large intestine.

PATHOGENESIS
Continued overloading of the colon and/or caecum with any of the undigested feed along with any of the predisposing factor can cause prolongation of intestinal contents and excessive inspissation of fecal material. With time a severe distension of the colon wall causes its insensitivity to stimuli which otherwise in normal cases provoke defecation. In horses the distension of large intestine is more serious than in other animals and often incite pain. Auto-intoxication (re-

absorption of toxic substances from gut) due to retention or delayed passage of ingesta can results in metabolic toxemia.

CLINICAL FINDINGS
A mild to moderate abdominal pain of a longer duration (3-4 days, even sometimes longer) with general depression, progressive anorexia, constipation, and almost normal pulse and respiration rates are indicative of impaction of large intestine. The intestine sounds are either decreases or absent, and there is passage of firm, dry, and scanty faeces sometimes coated with mucous. Impacted faecal mass can be palpated occasionally as a firm mass with doughy consistency in the colon or caecum, by rectal examination. The caecum can be palpated at exterior in the upper right flank and small colon to the right of the midline. Impaction of the caecum is more serious and have bad prognosis than of colon, as some time it may cause caecal rupture and death.

Retention of meconium in foals will be associated with signs of constant straining with elevation of tail, humping of the back with feet under the belly, and even sometime walking backward. Hard faecal balls can be palpated with the finger in the rectum.

Enterolith or similar foreign bodies are usually located in the entrance to the small colon and may or may not be palpable per rectum. The caecum or colon are distended with gas and ingesta, and a reflux distension of the stomach with fluid is detectable in some cases by passing a nasal tube. In sand colic diarrhea may sometime be present.

CLINICAL PATHOLOGY and POSTMORTEM FINDINGS
Laboratory tests are of no value for diagnosis of impactive colic. Radiographic examination in foals may help to identify the impacted mass. Sand particles may be found at the bottom of faecal mass if it is mixed with water in sand colic. Faecal examination may be undertaken for the diagnosis of nematode infestation. The large intestine may be found full of firm faecal material or its rupture with escape of faecal material in peritoneal cavity. Enterolith may be located at the junction of the right dorsal or small colon.

DIFFERENTIAL DIAGNOSIS
Impaction of large intestine needs to be differentiated from other causes of colic, e.g. enteritis, spasmodic colic, flatulent colic, peritonitis, and dehydration on the basis of detailed clinical examination, severity of pain, rectal examination, and therapeutic response to drugs. Moderate straining, gradual distension of lower abdomen, absence of urine, and uremic smell are indicative of urinary bladder rupture.

TREATMENT

The line of treatment in impactive colic includes the following steps;

1. Use of lubricant and laxative drugs. Examples is vegetable or mineral oil, @ dose of 2-4 liters/LA, PO, repeated 12 hourly, if required.

2. Sedative and analgesic drugs are used for the reduction of pain, but some of these may reduce the intestinal motility. So repeated doses of such drugs (e.g. Xylazine or Acepromazine) may be avoided.

3. The parasympathetic stimulant drugs (e.g. carbachol, @ dose of 2-4 ml/LA, SC) may be recommended in this type of colic in horses. These are only used after the administration of 1-2 liters of mineral oil, to soften the faecal mass, but not in other types of colic and pregnant animals.

4. Purgative drugs are some time used to remove the impacted mass. These include anthraquinone (Istizen, @ dose of 10-30 gm/LA, PO, only after the use of mineral oil), magnesium sulfate, @ dose of 500-800 gm/LA, PO, dioctyl sodium sulfosuccinate (an anionic surfactants) used @ dose of 8-30 gm/LA, PO, following the use of mineral oil.

5. Luke-warm soap water enemata may help to soften the impacted fecal mass and its easy passage out.

6. Retention of meconium (in neonates) may be treated with vegetable, mineral, or castor oils or glycerine. These drugs may be administered into the rectum as enemata with a long rubber tubing.

7. Oral and/or parenteral fluid therapy particularly in cases of dehydration.

NB: If medical treatment fails to remove impacted material, surgical laparotomy and removal of impacted mass is advised. Surgery is recommended when ever there is unmanageable pain, signs of displacement of large colon, increase in nucleated cells in the peritoneal cavity, and deteriorated condition of the animal.

FLATULENT COLIC
Syn: INTESTINAL TYMPANY, TYMPANITES

It is distension of the abdomen with gas collected in large intestine, characterized clinically by signs of acute abdominal pain, respiratory distress, and passage of much flatus.

ETIOLOGY

Intestinal tympany is a common condition of horses and can be classified as primary or secondary based on their etiology. The primary intestinal tympany is usually caused by ingestion of large quantity of highly fermentable green fodder. Whereas, the secondary intestinal tympany is more common and usually associated with partial or complete obstruction of caecum or colon (see also impaction of large intestine). It can also develop following castration due to fibrous adhesions of intestine or verminous aneurysm.

PATHOGENESIS

In equine most of the fermentation and digestion of feed takes place in the large intestine. The excessive production of gas or its retention in any segment of large intestine causes its distension, especially on the right side, and due to so abdominal pain. In the primary intestinal tympany the intestinal distension is periodically reduced because of evacuation of some gas via flatus, and this results in intermittent type of colic. If the causes of intestinal tympany are secondary ones the intestinal lumen distension is severe which could result in acute and continuous type of pain. Intestinal distension also causes pressure on the thoracic cavity which could result in breathing and circulatory problems. Rupture of intestine while rolling, in severe cases of tympany, may also lead to death.

CLINICAL FINDINGS

Acute abdominal pain with obvious abdominal distension are present in all cases of flatulent colic. The abdominal distension is more visible at the right flank, but whole abdomen could be distended. Intestinal motility may be absent but moving fluid sounds can be auscultated in gas filled loops of intestine, producing tinkling metallic type sounds. Pinging sounds can be auscultated mainly over the right flank. Rectal examination can reveal a large gas filled caecum on the right side of the abdomen. In primary intestinal tympany, there is more passage of flatus and anus may be in a constant state of dilatation.

NECROPSY FINDINGS

Animals dying of primary intestinal tympany, the caecum and colon will much distended with gas and may contain pasty and loose type intestinal contents. In secondary intestinal tympany one could search for evidences of obstruction and ruptured parts of intestine.

DIAGNOSIS

History of feeding on highly fermentable fodder, intermittent type colic, and passage of much flatus are some indications of primary intestinal tympany. Gas filled loop of intestine can be palpated at

rectal examination. It is however, difficult to locate the obstruction site in secondary tympany as the gas filled loop may interfere with the palpation. Presence of acute abdominal colic, passage of no faeces, presence of blood and mucous in the rectum, and no response to routine treatment are indications of acute intestinal obstruction.

TREATMENT

Following line of treatment should be complied with impactive colic.

1. Use of analgesic and sedative drugs (see colic). The administration of Xylazine is limited as it will also reduce intestinal motility.

2. Use of antizymotic or antifermenting drugs.

3. Use of trocar and canula, mainly through the right flank (or anywhere at the abdomen with maximum distention or even through rectum.

4. Use of antibacterials to control secondary bacterial infection particularly following trocarization.

A tympany draft to be used for intestinal tympany in adult horse contains the following drugs.

Chloral hydrate (60 gm), Turpentine oil (30 ml) or asafetida (20 gm), and Vegetable or mineral oil (500 ml).

NB: Dissolve chloral hydrate with approximately 200 ml of water and then mixed in vegetable oil. The mixture may be given via stomach tube or drenching bottle.

SPASMODIC COLIC

It is a spasmodic type abdominal pain occurring in horses with excitable temperament and characterized by the development of intermittent boots of pain followed by periods of relaxation and auscultation of loud peristaltic sounds (borborygmi) during the attacks.

ETIOLOGY

This type of colic occurs frequently in horses with excitable temperament. The inducing causes may include drinking of cold water soon after work, thunderstorm, preparation of animals for show or race, or loud drum beats. This type of colic has been reported to occur due to penetration in submucosal layers by migrating larvae of Strongylus vulgaris.

PATHOGENESIS

It is hypothesized that there is an increase in parasympathetic tone resulting in much increase in peristaltic movements in animals having excitable temperament due to any of the mentioned causes. This could result in periodic distension of various loops of intestine with either gas or ingesta. This increase in motility and distension are the consequence of varying in degree of intermittent type abdominal pain.

CLINICAL FINDINGS

The spasmodic colic is clinically manifested by bouts of intermittent type abdominal pain, i.e. rolling, pawing, kicking at the belly, flank watching, and patchy sweating for a few minutes, followed by periods of relaxation with normal posture and behavior until the next bout of pain. Initially the periods of relaxation are long (hours) but during later stages, intervals of bouts become short and attacks of pain are so frequent that it resembles a continuous type colic. Loud intestinal sounds (called borborygmi) can be auscultated or even heard without stethoscope during the periods of pain. This type of colic may disappear spontaneously without any treatment or it may leads to serious complications such as intestinal rupture or acute intestinal obstruction.

DIAGNOSIS

Spasmodic colic need rule out from enteritis, since both diseases are characterized by abdominal pain and increased intestinal sounds and motility. Diarrhea and/ or dysentery are common in the later condition along with other specific signs of enteritis. Acute intestinal obstruction is characterized by peracute type abdominal pain with absence of faeces and presence of blood and mucous in the rectum. Other causes of colic can be differentiated by history of pain and careful clinical examination.

TREATMENT

The treatment of spasmodic colic can be achieved by treating the animals according to the following steps.

1. Use of antispasmodic (spasmolytic) drugs such as Atropine sulphate, @ dose of 15-30 mg/LA, SC, following the PO use of 1-2 litters of mineral or vegetable oil.

2. Use of sedative and analgesic drugs (see colic).

3. Luke warm soap water enemata.

4. Catheterization for the relief of urine.

A common colic draft used in veterinary clinics include chloral hydrate (60 gm) and vegetable oil (400 ml).

NB: Dissolve chloral hydrate first in water and then mix with vegetable oil.

DIARRHEA
Syn: ENTERITIS, DYSENTERY, INTESTINAL CATARRH

Diarrhea is derived from Greek words Dia (through) and rhein (to flow). It is an increase in the frequency and fluidity of faeces and bowel movements.

Enteritis is an inflammation of the intestinal mucosa, characterized by diarrhea (sometime dysentery), abdominal pain, dehydration, and electrolyte imbalance.

Dysentery (Bloody diarrhea; When faeces contains blood, mucous, and/or fibrin, it is called as dysentery. Dysentery is an indication of severe inflammatory bowel disease.

The normal frequency of bowel motions (defecation) varies with the species of animal. For examples, cattle defecate 11-16 times, horses 8-10 times, and dogs 1-2 times in a day. The defecation may be affected by the breed, age, and body weight of an animal, and type and amount of feed intake.

ETIOLOGY
There are several specific and non specific diseases of animals in which diarrhea (dysentery) is a major clinical findings. Some of the diseases causing diarrhea are as under;

A) Bacterial diseases; Examples are coli-bacillosis (young animals) salmonellosis, enterotoxemia caused by Cl. perfringens types A, B, and C infections, and Johne's disease or paratuberculosis (a chronic diarrhea of adult animals).

B) Viral diseases; Examples are rinderpest, bovine malignant catarrh, bovine viral diarrhea, blue tongue disease (in sheep), pest des petits ruminant (in goats), rotavirus and coronavirus infections (in young animals of many species), and canine distemper and parvo infections (in dogs).

C) Parasitic diseases; Examples are paramphistomiosis, ostertagi haemonchosis, strongylosis, trichostrongylosis, moniezi fascioliasis, giardiosis, toxoplasmosis, cryptosporidiosis, coccidi balantidiosis, and whipworm infestations (dogs only).

D) Fungal disease; Examples are aspergillosis (aflatoxicos candidiosis, and histoplasmosis.

E) Miscellaneous diseases; Examples are chemical and pla poisoning, lactic acidosis, copper deficiency, congestive heart failu certain toxemia, eosinophilic colitis (dogs), colitis-X disease (horse anaphylaxis reactions, disaccharidase deficiency (foals), and ma neoplasias developing in the vicinity of gastrointestinal tract, e lymphosarcomas.

PATHOGENESIS
Diarrhea develops when ever the small intestinal absorption proces decreases and secretion increases. The pathophysiology of diarrhe can be divided as under;

1) Osmotic effects; An increase in osmotic pressure within the gu lumen will result in the withdrawal of body fluid from plasma into the intestinal lumen. This fluid, if not reabsorbed and accumulate in excessive quantity, will result in diarrhea. This mechanism of diarrhea development operates in non invasive bacterial diseases (e.g. enterotoxaemias), disaccharidase deficiency, with use of saline purgatives or laxatives and certain surfactants (e.g. Dioctyl sodium sulfosuccinate).

2) Exudation or Inflammation (acute or chronic) of the intestinal mucosa; It effects both motility and secretion, this causes increase in peristalsis and fluid production beyond the absorptive capacity, which outcome in diarrhea. It may also accompany by an increase in other products of inflammation and loss of serum proteins. Persistent loss of fluid and electrolytes will result in dehydration and electrolyte imbalances. This mechanism of diarrhea operates in most of the invasive bacteria, virus, and parasites.

3) Malabsorption; Malabsorption of the fluid due to defects, destruction, or atrophy of villous absorptive cells, and is as seen in Rota and Coronavirus infections in young animals.

4) Others mechanisms; Increase in blood-to-lumen hydraulic pressure (as seen in congestive heart failure), hypoproteinemia (as seen in chronic inflammatory bowel disease), and decrease lymphatic drainage (e.g. lymphadenitis) may also result in an net increase in faecal water contents and diarrhea.

METABOLIC ABNORMALITIES WITH DIARRHEA

The effects of enteritis are losses of water (dehydration), electrolytes, i.e. sodium, chloride, and potassium (electrolytes imbalance), bi-carbonates (acid-base-imbalance), and varying quantity of protein (hypoproteinemia) from the body. The fluid moves out first from intravascular compartments (vasculature), then but of extravascular compartments (intercellular spaces), followed lastly from the intracellular spaces. The location and types of lesions also effect the severity of illness. In cattle much of the fluid (80 per cent) is absorbed through small intestine, so inflammatory lesions at small intestine will cause severe dehydration compared to similar lesions at large intestine, but opposite is true with horses. The acute and severe diarrhea may lead to sudden shock and subsequent death.

Acidosis may occur by various means in acute diarrhea. One of these is intestinal bicarbonate loss. Water and carbon dioxide combine forming carbonic acid. Carbonic acid deionizes forming hydrogen and bicarbonate. As the bicarbonate loss in diarrheic feces, more bicarbonate and hydrogen is produced. Since bicarbonate is lost in the feces and hydrogen remains, the hydrogen concentration increases resulting in metabolic acidosis. Dehydration also influences the hydrogen ions concentration by its decrease glomerular excretion.

In chronic enteritis there is decrease in digestion and malabsorption of food, resulting in body wasting without clinical evidences of dehydration.

CLINICAL FINDINGS

The major clinical findings of enteritis are the frequent passage of soft and fluid faeces, evidences of dehydration, occasional abdominal pain, and toxemia. In most cases of acute enteritis, fluid faeces may contains blood, mucous, and/or fibrinous casts (dysentery) with unpleasant odor. In this type of enteritis and particularly of large intestine inflammation, the faeces may be passed with tenesmus (straining).

Fever may be present initially, but during later stages of perfuse diarrhea (and circulatory collapse) there may be subnormal body temperature. Dehydration may develops within few hours of acute diarrhea, though clinical dehydration will be obvious only when fluid

loss is more than 10 per cent of the body weight. This can be assessed by tenting the skin of neck or eyelids (skin taking longer time, i.e. >2 seconds, to come to the original position) and recession of eyeballs in their sockets. Auscultation of abdomen may reveals fluid rush sounds in early stages due to hypermotility.

In chronic diarrhea (enteritis), the faeces may be soft and homogenous with no odor. In addition, a progressive weight loss and emaciation without evidences of clinical dehydration or systemic involvement are common occurrence.

A foul smelling faeces is indicative of abnormal fermentation and decomposition of epithelium, blood, fibrin, or due to pus and necrotic tissues. A fishy smell is indicative of decomposition of protein. A pungent smell is diagnostic for salmonellosis or biliary colic. Sour or acidic smell is due to fermentation of glucose or lactose as seen in lactic acidosis. Offensive odor is noticed with incomplete digestion and diagnostic for pancreatic insufficiency.

CLINICAL PATHOLOGY

Macroscopic examination of color, consistency, constituents (mucus, fibrin, blood), odor, and presence of any segments of cestodes or intact roundworms may help in identification of the etiology.

Microscopic examination of faeces may be necessary for the identification of ova (larvae or oocysts) for parasitic cause, and cultural examination for the bacterial cause. Complete blood count, biochemical profile, and urinalysis may help in evaluation of hemoconcentration, metabolic acidosis, electrolytes, fibrinogen, and protein (albumen) contents to assess the severity of disease or its complication. An increase in blood urea nitrogen is an indication of inadequate renal perfusion associated with severe dehydration.

Serological tests may be of use for the recognition of viral diseases. Rectal or intestinal biopsy for cultural examination is recommended for the diagnosis of Johne's disease. Colonoscopy may help in diagnosis of large bowel diseases particularly in small animals.

NECROPSY FINDINGS

A gross evidences of acute enteritis may include the presence of edema, hyperemia, hemorrhages, catarrhal to fibrinous inflammation, ulceration, and/or necrosis of the intestinal mucosa, and foul smelling intestinal contents. A general thickness and edematous type swelling without other visible gross lesions are indications of chronic enteritis.

DIFFERENTIAL DIAGNOSIS

The gross appearance of faeces can provides first clue of the cause of diarrhea. In general, the lesions in the small intestine of cattle cause perfuse and watery diarrhea, whereas, the lesions in large intestine are associated with mild diarrhea with excessive mucous. Salmonellosis is characterized by the development of dysentery with fibrinous casts, toxaemia, and fever. Johne's disease is characterized by chronic diarrhea in mature animals with progressive weight loss and little or no response to medical therapy. Helminthiasis is characterized by mild diarrhea, progressive weight loss, and without obvious systemic reaction. In dietary diarrhea the faeces are soft and voluminous without systemic illness and clinical dehydration.

Diarrhea	Dysentery
1. Voluminous fluid faeces	Scanty sticky faeces
2. No blood in the faeces	Presence of blood in the faeces
3. No mucous or fibrin in in the faeces	Presence of mucous and fibrin in the faeces
4. Less pus cells	Abundant pus cells
5. No straining (tenesmus) during defecation	Straining during defecation

TREATMENT

Control of diet is recommended first even before making diagnosis. The general lines of treatment of diarrhea should incorporate the followings steps;

1. Specific treatment if cause of diarrhea is known. Example, antibacterials or anthelmintics used via parenteral and/or oral routes. Antibiotics may be started when ever there are evidences of inflammation, leukocytosis, fever, or systemic response.

2. Symptomatic therapy; (a) Replacement of lost fluid and electrolytes, both via oral and parenteral routes, to combat dehydration, acidosis, and electrolyte loss, preferably within 18-24 hours. In severe cases of dehydration, i.e. >10 per cent of body weight, the dose of fluid therapy should be approximate to 100 ml/kg B.Wt, per 24 hours. The amount of fluid can roughly be calculated by the formula;

Body weight (kg) X Per cent dehydration = L of fluids required in 24 hours.

The total amount of fluid to be administered may be increased if there is ongoing loss through vomiting and diarrhea. The fluid is administer either IV, SC, PO, or IO (intraosseous) depending upon the condition of the animal.

(b) In cases of severe acidosis a hypertonic sodium bicarb solution of 7.5 per cent, should initially be given, followed normal solution (1.3 per cent), as maintenance dose for the ne hours. The common IV fluids available includes dextrose 5 per dextrose with normal saline, normal saline 0.9 per cent, Ri solution, lactate Ringer's solution and sodium bicarbonate solutio

Formula for oral rehydration salt (ORS) commonly availabl human and veterinary use contains the followings:

i) Sodium chloride (3.5 gm), sodium bicarbonate (2.5 gm), pota chloride (1.5 gm), glucose (20 gm), and distilled water (1 liter).

ii) Glucose (67.5 per cent), sodium chloride (14.3 per cent), g (10.3 per cent), citric acid (0.8 per cent), potassium chloride (0.2 cent), and potassium dihydrogen phosphate (6.8 per cent).

(c) Alteration and withholding diet for 24 hours is nece particularly in acute enteritis to avoid fermentation and putrefa of feed which may further damage the intestinal mucosa. During period fluid containing ORS or parenteral normal solutions desirable to minimize harmful effects of dehydration.

(d) For the protection of intestinal mucosa, inhibition of secretion, intestinal hypermotility the common drugs for veterinary use chlorpromazine, kaolin, calcium carbonate, aluminum hydrox pectin, chalk, catechu, bismuth subnitras (or subsalicylate), charc and opiate derivatives. These are administered PO either alone or combination of few of them (if and as required). Chlorodyne, @ dose 30 ml/LA, PO, may be given for reducing diarrhea but not in hor suffering from hepatitis.

NB: Per oral antibiotics should not be used in conjunction wi intestinal protectants.

Anticholinergic drugs such as atropine sulphate, probanthelin (ProBanthine), and hyoscine compound (Buscopan) may be used reduce intestinal hypermotility and secretions. Salicylates may also b beneficial in secretory diarrhea and their antisecretary effects are d to inhibiting prostaglandins activity. There are evidences that pect and salicylates can absorb and inactivate enterotoxins produced by E coli.

Formula of astringent powder commonly used in veterinary clinics fo large animal contains the followings;

Bismuth subnitras (10 gm), chalk (15 gm), catechu (15 gm), kaolin (15 gm), pectin (10 gm), and rice gruel (QS).

NB: The rice gruel contains amylose and amylopectin, which themselves can prevent secretory type diarrhea.

(e) For the management of abdominal pain narcotic analgesic drugs are needed to be administered. Examples are meperidine, diphenoxylate, and loperamide.

ACUTE INTESTINAL OBSTRUCTION
Syn: INTESTINAL ACCIDENTS

It is the blockade in the passage of ingesta caused by complete intestinal obstruction, characterized clinically by violent to severe abdominal pain, severe shock, absence of defecation and faeces, instead the presence of blood and/or mucous in the rectum.

ETIOLOGY

The major causes of acute intestinal obstruction can be grouped into 3 types. These are;

1) Volvulus (Torsion or Twisting) of any intestinal segment on its mesenteric axis,

2) Intussusception (Telescoping or invagination) of one part of intestine into the adjacent segment, and

3) Strangulation (Incarceration or entrapment of intestinal segment), e.g. umbilical and ventral hernias.

Either of these types can be seen in all animal species, in any age group, and at any part of intestine. The exact cause of these conditions is not clear, however, increase in peristalsis as seen in spasmodic colic, intestinal parasitism, enteritis, severe exercise, jumping, rolling, and truck journey are considered contributing factors for their development.

Enteroliths, bowel tumors, caecal impaction, fibrous adhesions, mesenteric fat necrosis, atresia ani-cum-recti, etc, are some minor causes of intestinal obstruction.

PATHOGENESIS

The effects of intestinal obstruction on an individual differ between species, site of intestine involved, and type of obstruction. For example; a complete obstruction of small intestine in horses is more severe and could be lethal within 24 hours, compared to a similar obstruction in cattle, which may take days to kill the animal. The development of clinical disease is associated with many factors. There is complete obstruction in the passage ingesta, fluid, and gasses which are the cause of over-distention of intestinal lumen and visceral pain. Complete occlusion of the blood supply to the affected segment may result in ischemia, edema, devitalization, and later necrosis of the part(s).

In addition, there is also development of toxemia either due to infection with toxigenic bacteria within the intestine or auto-intoxication which aggravate the clinical disease. Other factors which contributes the disease are acute shock (both endotoxic and circulatory origin), dehydration, electrolytes, and acid-base imbalances.

CLINICAL FINDINGS

In horses the obstruction of small intestine is characterized by sudden onset of per-acute or acute abdominal pain, evidences of severe shock, and toxemia, progressive abdominal distention, and multiple areas of pinging sound. A progressively rising pulse (>80-100 per minute) and respiration (>70-80 per minute) indicate the severity and acuteness of the condition. Faeces may pass initially but later on, it may completely be absent, instead, there may be presence of blood and/or sticky mucous in the rectum. The obstruction site in the form of distended loop of intestine can be palpated per rectum. A reflex filling of stomach with fluid and its evacuation via vomiting (also if stomach tube is passed) is not uncommon in horses. Terminal rupture of gut, severe endotoxic shock, and acute diffuse peritonitis can kill the animal within 24 hours. Obstruction at the large intestine have similar clinical features but can run a longer course compared to small intestine obstruction.

Similar condition in cattle is characterized by an acute attack of spasmodic type colic lasting for few hours (8-10 hr), followed by days of depression accompanying with variable signs of ill-health. A slow development of toxemia and vascular damage escalate the condition. Distention of abdomen, progressive increase in heart and respiration rates, and recumbency are the signs of grave consequences. In untreated cases, intestinal rupture will ultimately occur with ensuing shock which could lead to death within 1-2 weeks.

CLINICAL PATHOLOGY

Hemoconcentration (PCV >50 per cent), acid-base imbalance, increased BUN, leukopenia and neutropenia initially followed by leukocytosis are common laboratory findings of acute intestinal obstruction. Examination on peritoneal fluid could reveal the presence of red blood cells and other inflammatory cells,. contamination with variety of kinds of bacteria and intestinal flora, and faecal materials (in gut rupture).

NECROPSY FINDINGS

Physical lesions described can readily be identified with or without evidence of gut rupture. Presence of faecal matters in the peritoneal cavity is a good evidence of intestinal rupture. The involved segment will have varying degree of congestion, discoloration, edema, necrosis, and/or gangrene. In cases of intact intestinal wall, the oral segment will contain fluid, gas, and/or ingesta but the aboral segment empty.

DIFFERENTIAL DIAGNOSIS

Enteritis is associated with frequent passage of fluid faeces mixed with blood and/or mucous, dehydration, and hyper-peristalsis. In flatulent colic there is passage of faeces with much flatus and less severe signs of pain and shock as compared to acute intestinal obstruction. Spasmodic colic is characterized by acute visceral pain of intermittent type and which respond well to antispasmodic drugs. Gastric dilatation (in horses only) is characterized by periodic sub-acute attacks of colic and frequent vomiting. There is passage of large quantity of fluid and gas in vomitus followed with relief of pain. Traumatic reticulitis (in cattle and buffaloes) is characterized by abdominal pain of short duration, mild fever, ruminal stasis, constipation, recurrent attacks of tympany, tenderness of abdomen, and a chronic course.

TREATMENT

The effective treatment of this condition is surgical, therefore, as soon as the condition is diagnosed the case should immediately be referred to qualified veterinary surgeon.

In cases of severe and violent type pain medical therapy should be started to relief the pain and shock well before the surgical intervention. The response of medical therapy could be monitored after every 3-4 hours till 24 hours. In cases of no response to therapy. then exploratory surgery is required to find out the underlying cause and its correction.

Antibiotics therapy is indicated to control the secondary bacteria infections following intestinal accidents or surgery. Any complication

associated with these conditions should be treated accordingly. T prognosis of these conditions is not favorable but one should alw remember "early the surgery better the prognosis".

SIMPLE INDIGESTION

It is a common but mild and self-limiting condition of ruminan developing mainly due to dietary abnormalities, particularly in sta fed animals. Clinically the disease is characterized by anorexi decrease in rumination and ruminal motility, drop in milk productio and mild constipation or diarrhea.

ETIOLOGY

The common causes of simple indigestion include dietar abnormalities of minor degree. It includes feeding animals o indigestible roughages (straw or bedding particularly during dry periods), mouldy or over fermented silage, over-heated or frosted feed moderate excess to grains (wheat, oat, barley, corn, rice) and carbohydrate rich diet to animals unaccustomed to them. Other causes includes, sudden change in feed, i.e. from one type to an other type, lack of salt intake or water intake (especially in summer), and improper exercise specifically in stall fed animals. Prolonged and heavy oral dosing with sulfonamides or antibiotics can also inhibits normal ruminal flora and thus can cause indigestion.

PATHOGENESIS

Diet or feed presented to ruminal flora in which they are not metabolically adopted or less adopted, or diet containing inhibitory substances will result in imbalance of ruminal micro flora and its fermentation. A second mechanism operating in indigestion is the decrease in ruminal motility either because of change in ruminal pH, as seen in mild cases of acidosis due to high fermenting or carbohydrate rich diet or feeds, or alkalosis due to feeding on urea or protein rich diet. A physical impeding of ruminal activity by over eating of indigestible roughages is an other cause of decrease ruminal motility. In addition, a number of toxic amines and histamine are known to cause ruminal atony when produced locally by highly fermenting or spoiled feed. The resultant effects are decrease in micro floral fermentation and digestion of feed, ruminal atony, and delayed in passage of ingesta. There is sharp fall in volatile fatty acid production in atonic rumen which results in decrease of milk production.

CLINICAL FINDINGS

Generally, it is a mild and self-limiting condition of ruminants. A reduction in feed intake (anorexia), decrease or absence of rumination, and slight drop in milk production (in milch animals) are some of the first appearing signs. A slight depression, decrease or absence of ruminal motility (in frequency and amplitude), firm to doughy rumen, and mild tympany are other related signs. The faeces is usually firm and drier initially, but later (after 24-48 hours) it may become loose and diarrheic. The duration of illness is usually short unless there is destruction of ruminal micro-flora where it may run a longer course. Signs of systemic involvement, as evident by an increase in temperature, pulse, and respiration and pain are not present in simple indigestion.

CLINICAL PATHOLOGY

A change in rumen pH is also an indicator of digestive abnormality. A pH 6.5-7 in green fed animals and 5.5-6 in grain fed animals is considered normal. Tests to assess the activity of ruminal micro-flora may also be used. These include;

1. Sedimentation activity test; The ruminal fluid is aspirated out and strained to remove course particles. The fluid is allowed to stand in a glass vessel at room temperature and time required for floatation of particulate material recorded. The time in normal animals is between 3 minutes (in recently fed animal) to 9 minutes (if fed previously). Setting of particulate material indicates gross inactivity and less severe degree is manifested by prolongation of the time for floatation.

2. Cellulose digestion test; A plastic bead tied to the end of a cotton thread is put in a vessel containing freshly removed ruminal fluid. The time required to digest the cotton thread (separation of bead from thread) is noted. Digestion time more than 30 hours indicate abnormality.

DIFFERENTIAL DIAGNOSIS

Simple indigestion needs to be differentiated from all diseases of the forestomach and abomasum in which ruminal atony is a common clinical findings. Acetonemia is seen in heavy milk producers 1-2 months following parturition. Clinically it is characterized by anorexia, acute fall in milk, and ketonuria. Traumatic reticulitis is associated with sudden onset of anorexia, agalactia, mild fever, and evidence of pain at palpation of reticulum. Acute rumen acidosis is an acute disease developing following over eating of grains, characterized by depression, dehydration, acute ruminal stasis, and much fall in rumen pH. Left sided abomasal displacement (LDA) is a chronic condition having similar clinical signs as in indigestion and which do

not respond to medical therapy. A pinging sound over the lower left flank is diagnostic of LDA. Vagus indigestion is also a chronic condition, associated with hyper- or hypomotility of rumen, dehydration, passage of scanty faeces, and give poor response to medical therapy. (A secondary anorexia is associated with many systemic illnesses where treatment of primary condition also alleviate indigestion).

TREATMENT

The objective of treatment should be restoration of normal ruminal flora, motility, and pH.

1. The treatment of indigestion adopted is usually symptomatic consisting of the use of rumenitoric drugs. The most common and available drug used is magnesium sulphate (@ dose of 500 gm/LA, PO). Use of nux vomica, pulv gentian, or pulv ginger (each @ dose of 15-20 gm/LA, PO), is good to restore normal ruminal motility and secretion. Parasympathetic stimulants such as carbamylchloline (carbachol), physostigmine, and neostigmine may given in low doses to increase ruminal motility but there use in simple indigestion is controversial.

2. More rational therapy include the use of alkalizer as magnesium hydroxide (@ dose of 400 gm/LA, PO) suffering from mild acidosis or acetic acid (10 per cent) or vinegar (@ dose of 10-15 liters/LA, PO) when rumen pH is alkaline.

3. In cases of lost ruminal flora with prolonged cases of indigestion, reconstitution of normal flora by rumen transfaunation (cud transfer or rumen transplant) is the best treatment.

4. Use of vitamins especially B-complex may help in early recovery and improve health in anorexic animals.

Common prescriptions for indigestion contain the following;

a. Tonic or stimulant powder; It consists of magnesium sulphate (500 gm), pulv nux-vomica (10 gm), pulv gentian (10 gm), pulv ginger (10 gm), and molasses or water (QS to be given orally).

b. Stomachic or digestive power; It consists of aniseed (15 gm), osmum seed (15 gm), chiretta (15 gm), mustard (15 gm), Indian rhubarb (15 gm), ginger (15 gm), black salt (20 gm), and common salt (30 gm). Make 2 doses (boluses in molasses) and give orally twice daily.

c. Carminative or digestive mixture; It consists of spirit ammonia aromatic (8 ml), spirit chloroform (8 ml), spirit ether nitrosi (8 ml), Tr.

cardamum co (8 ml), Tr. ginger (8 ml), Tr. nux vomica (8 ml), sodium bicarbonate (30 gm), and water (QS) to make mixture for drench.

NB: Nux vomica should not be given in pregnant animals.

BLOAT
Syn: RUMINAL TYMPANY

It is an over-distension of the rumen and reticulum with gases of fermentation, characterized clinically by signs of colic, left abdominal distension, dyspnea, and sudden death.

ETIOLOGY

The disease is common in all ruminants but more cases have been reported in cattle (buffaloes). Based upon the etiology, the tympany may be classified as primary or secondary. The primary ruminal tympany or frothy bloat is caused by intake of certain types of diet that lead to formation of stable foam within the rumen. It can further be classified into leguminous bloat and feed-lot bloat. The secondary ruminal tympany or free gas bloat is caused either by certain types of diet that may lead to excessive gas production and low intraruminal pH, or by failure to eructate normally produced gases from extra-ruminal causes.

The causes of frothy leguminous bloat are the excessive consumption of rapidly growing, lush green, leguminous plants, i.e. alfalfa (Medicago sativa or lucern), cereal crops as wheat, clovers (Trifolium sp.), rape, leguminous vegetables (peas, beans, etc). The plants contain high contents of certain foaming agents, such as soluble leaf cytoplasmic proteins, saponins, pectins, and hemicellulose. These agents are particularly increased during the rainy season and/or when heavy fertilizers (urea) is used. The foaming agents cause an increase in surface tension of ruminal contents which inhibit the coalescence of the small gas bubbles, and so there is formation of a stable foam in the rumen. The frothiness of ruminal content causes obstruction of the cardia, inhibits the eructation reflex, and closure of caudal esophageal sphincter which result in increase intraruminal pressure.

In feed-lot bloat the intake of high grain diet, especially of finely grounded with particle size <4 mm, promotes frothiness of rumen contents by entrapment of the gases of fermentation by those fine particles. In addition, the type of ruminal flora i.e. encapsulated insoluble slime producing bacteria, also helps in increasing viscosity and production of insoluble stable foam.

Susceptibility of disease increases when animals are not accustomed to bloat producing diet. The specific condition of the rumen prior to feeding or rate of physical breakdown of feed are other factors in the production of bloat. The pH of the rumen effect the stability of foam, (maximum stability at pH 6), ruminal motility, and interference in eructation. The rate of flow and composition of saliva (having buffering effect) also determine the tendency of bloat.

The free gas bloat or secondary ruminal tympany may develops due to ruminal or extra ruminal causes. In ruminal causes tympany may develops either secondary to the acute onset of ruminal atony that may occur due to histamine production (in anaphylaxis), increase or decrease in ruminal pH (in alkalosis or acidosis), rumenitis, or prolonged lateral or dorsal recumbency which may interfere with eructation and evacuation of normally produced gases. The extra ruminal cause of secondary tympany are physical obstruction in eructation, as seen in choke, interference of nerve pathway as in vagus indigestion, diaphragmatic hernia, and tetanus. Any lesion in the wall of reticulum (which contains tension receptors and receptors that discriminate between gas, foam, and liquid) may interrupt the normal reflex that is essential for escape of gas from the rumen.

PATHOGENESIS

Either with primary or secondary ruminal tympany there is over distension of rumen with gases. With mild cases, the rumen movement are stimulated by rumen distension which may acerbate the frothiness of the ruminal contents. In severe cases there is much distension to the point where there is loss of muscle tone and ruminal motility. The mechanical pressure exerted on chest cavity by distended rumen causes reflexely depression of cardiovascular and respiratory systems. Rupture of diaphragm may occur which may cause sudden death due to respiratory and cardiac arrest. In addition, absorption of toxic gases (hydrogen sulfide), or toxic amines (histamine) are contributing factors in causing clinical disease and death.

CLINICAL FINDINGS

There is sudden development of distension of entire abdomen but more obvious at upper left flank, usually soon after grazing on bloat producing paster. Distension of the left flank may be so severe that the contour of the paralumber fossa protrudes above the vertebral column. Animals show the signs of colic, including lying down and getting up frequently, kick at the belly, treading, and some times, rolling on the ground. Dyspnea and grunting are marked in the form of open mouth breathing, with protrusion of the tongue and extension of the head. The ruminal movements initially are increased, but late

they are decreased or totally absent. Respiration and heart rates are increased (60/ min and 120/ min respectively), with heart murmurs at auscultation. Palpation of left flank in primary bloat may reveal a feeling of fluid and gas under pressure, whereas, in secondary bloat, a free gas cap at the top of the solid. High pitched tympanic sound (ping) can be heard at percussion at the left paralumber fossa. Vomiting occasionally may occur and soft faeces type material may be expelled out in a stream. Severe cases may end up with much distress, cyanosis of mucous membranes, sudden death following collapse, and asphyxia.

CLINICAL PATHOLOGY

Clinical pathology is usually not required for diagnosis of bloat. However, estimation of ruminal pressure, examination of ruminal flora, physical and chemical nature of ruminal fluid may help understanding the cause and prescribing treatment and control measures of the condition.

NECROPSY FINDINGS

Animals died of bloat may show protrusion and congestion on the tongue, marked congestion and hemorrhage of lymph nodes of head, neck, epicardium and upper respiratory tract, friable kidneys and hyperemia of intestinal mucosa. The lungs are compressed and intrabronchial hemorrhage may be present. The rumen is distended, and contents much less frothy at postmortem than before death. There may be pale liver due to expulsion of blood and rupture of diaphragm.

DIAGNOSIS

The primary bloat can be differentiated by secondary bloat by history of feed intake. The use of trocar or canula with complete evacuation of gas from rumen is indication of secondary bloat. Passage of stomach tube can also help in diagnosis of esophageal obstruction or stenosis. Vagus indigestion, traumatic reticulitis, and diaphragmatic hernia have a chronic history of illness with recurrent tympany. Tetanus is characterized by prolapse of 3rd eyelid, rigidity of whole body, and hyperaesthesia. Blackleg, lightning stroke, anthrax, and snake bite are characterized by sudden death which need careful examination.

TREATMENT (Acute cases should be considered as emergency).

1. In life threatening cases use of trocar and canula or performing rumenotomy can help to remove gases and marked relief to the animal. Gases can also be removed by passing of stomach tube. In case of secondary tympany gases are evacuated in a single attempt and pressure is released easily by this method, however, multiple attempts are required to evacuate foamy nature of gases in primary bloat.

2. In less severe cases, induction of salivation (which is alkaline) by tying a bit in the mouth may help in denaturing of stable foam. Use of antacids (Sodium bicarbonate, @ dose of 100-200 gm/LA in one liter of water, PO, Magnesium oxide or hydroxide, @ dose of 200-400 gm/LA, in water) may help in reducing feed-lot bloat.

3. Antizymotics and antifoaming agents, administration by oral or through stomach tube or trocar and canula help to reduce gas formation and tympany. Examples are; vegetable oil (corn, rape seed, sunflower, linseed or soybean), @ dose of 200-400 ml/LA, mineral oil, @ dose of 200-400 ml/LA, turpentine oil, @ dose of 15-30 ml in 250 ml of vegetable oil/LA, formalin 4 ml in 300 ml of water (for secondary bloat), methyl silicone (2-5 per cent solution, 50-100 ml), dioctyl sodium sulfosuccinate (a surfactant) with vegetable oil, polaxalene (a polymer of Polyoxypropylene-polyoxyethylene series) 25-50 gm/LA, PO, or directly in intraruminal injection.

Injection of Neometeoryl, Tympaveex, and Poloxalene, @ dose of 10 ml/LA, intra-rumen injection (or PO) are available for primary leguminous (frothy) bloat). For primary (feed-lot) bloat dimethyl dialkyl quaternary ammonium compound is more good than poloxalene.

A common "Tympany draft" used for large animals consists of turpentine oil (15-30 ml), pulv asafoetida (15-20 gm), vegetable oil (250-500 ml), and chloral hydrate (in pain only, 30-60 gm).

NB: Chloral hydrate should first be mixed with water before adding to the oil.

Carminative mixture or rumenitoric drugs, i.e. nux vomica, ginger, gentian (and as in simple indigestion) and antacids i.e. magnesium oxide and hydroxide, calcium and ammonium carbonate, sodium bicarbonate, during the convalescent period for maintenance of ruminal motility and digestion. Transfaunation fluid containing viable ruminal flora, @ dose of 4-6 liters/day for 4-5 days is recommended to re-establish normal floral population.

CONTROL

1. Feeding of hay or fibrous feed before grazing on bloat producing pasture, or all available fodders (clovers and grasses together) may be cut, mixed, and chopped before feeding to animals.

2. Administration of vegetable oil or emulsified fat, @ dose of 100-200 ml/LA, PO, before grazing on bloat producing pasture help to reduce

incidence of bloat. Vegetable oil or emulsified fat may be sprayed on pasture of feed to be offered during specific time of the year when occurrence of bloat is common.

3. Antifoaming/antizymotic drugs can be used as prophylaxis for reducing the incidence of bloat during peak bloat season.

4. Incidence of feed-lot bloat can be reduced by feeding grains mixed with about 10 per cent of cut and chopped roughages.

5. Avoid overfeeding following a prolonged period of starvation.

DISEASES ASSOCIATED WITH HARDWARES

The possible outcomes (sequelae) of accidental ingestion of metallic foreign objects such as nails, needles, wires, or any sharp pointed object by the ruminants, are as under;

* Traumatic stomatitis (with signs of sudden stoppage of feed intake during feeding, complete anorexia, and hyper-salivation).
* Traumatic pharyngitis (same signs as above).
* Traumatic esophagitis (sudden anorexia, salivation, regurgitation, and other signs of choke).
* Traumatic rumenitis (rare in animals, signs similar to TR)
* Traumatic reticulitis (TR) (It is the most important condition among the diseases caused by hardware).
* Traumatic rupture of left gastroepiploic artery (a rare sequelae which may leads to fatal hemorrhage).
* Acute local peritonitis (a common complication of TR).
* Acute diffuse peritonitis (a rare outcome of TR).
* Chronic local peritonitis (a common outcome of TR).
* Vagus indigestion (a rare outcome of TR).
* Diaphragmatic hernia (a rare outcome of TR).
* Traumatic pericarditis (a common sequelae of TR).
* Cardiac tamponade due to rupture of coronary artery.
* Septicaemia and related complications such as splenic abscess, hepatic abscess, diaphragmatic abscess, mediastinal abscess, endocarditis, arthritis, pneumonitis, & nephritis.

TRAUMATIC RETICULO-PERITONITIS

Syn: Traumatic Reticulitis, Hardware Disease.

Accidental ingestion of any sharp metallic object along with feed may result into traumatic perforation of reticular wall resulting in reticulo-peritonitis. Clinically, the acute disease is characterized by sudden

onset of complete anorexia, cranial abdominal pain, ruminal stasis, sharp fall in milk, and mild temperature.

ETIOLOGY

This is a common disease particularly in confined dairy animals which are given premixed feed. Cattle and buffaloes usually can not discriminate between food and non-food items if mixed together with their feed. Metallic objects such as nails, pieces of wire, large sewing needles, etc, if swallowed fall first into rumen and are subsequently carried over the rumino-reticular fold into the lower anterior part of the reticulum.

The metallic objects with blunt ends may remain in rumen and/ or reticulum without causing harm, but some with pointed/ piercing ends may cause problems. The honey comb like reticular mucosa acts as a trap for sharp metallic objects. Contraction of reticular wall promotes penetration of the wall by the swallowed sharp metallic object, resulting in reticulitis. The penetrated metallic object subsequently may cause local or systemic peritonitis (if it is contaminated). Many cases of the disease are seen soon after parturition, heavy exercise, or truck journey, as these conditions can contribute in penetration of any sharp metallic bodies into the reticulum.

PATHOGENESIS

The metallic objects following ingestion first settled down in the rumen due to their gravity. Subsequent contraction cycles of the forestomach dump those objects from the rumen into the reticulum. Injury to the reticular wall with mere penetration to the serous surface may not cause any complication and foreign bodies are either corroded away or remain embedded for rest of their life. In such cases, the related intramural inflammation may result sometime in dysfunction of the rumino-reticulum, because of interference with chemoreceptors.

Perforation of the wall of the reticulum followed by development of infection, initially cause acute local peritonitis, which may turn in either complete recovery (if foreign body falls back into the reticulum), chronic local peritonitis (part of healing process), or acute diffuse peritonitis. Most of the perforations occur in the lower parts of anterior reticular wall but other may pass through in any direction and can result in traumatic injuries and infections, particularly in diaphragm, liver, abomasum, spleen, pericardium, lung, or pleura. It is also possible that the metallic body may be directed out through exterior wall in the form of a local abscess and followed by mostly, complete recovery.

From initial local peritonitis infection may spreads through blood, to other organs and may cause to develop endocarditis, myocarditis, arthritis, nephritis, generalized septicaemia, or pulmonary or liver abscesses. Sudden death in animals has been recorded and may be because of cardiac arrest due to traumatic rupture of either coronary artery, ventricular wall, or severe hemorrhage due to rupture of left gastro-epiploic artery.

CLINICAL FINDINGS

The clinical disease can be divided into 3 forms.
i) Acute local reticulo-peritonitis, ii) Chronic local reticulo-peritonitis, and iii) Acute diffuse peritonitis.

i) In acute local reticulo-peritonitis, there is sudden development of complete anorexia, mild fever (103-104 °F), increase in pulse (>80/ min), decrease in ruminal contraction, and evidence of cranial abdominal pain lasting for 2-3 days. The animal shows reluctance to move, prefer to remain standing for long periods, or lie down with great care. Pain at the anterior abdomen is manifested by abduction of elbows and arching of or rigidity of the back (tucked up abdomen). Walking (particularly) downhill and acts of defecation and urination often accompanied with grunting sounds (due to pain). Occasionally affected animals may the show signs of acute abdominal pain in the form of kicking at belly, stretching of the body, rolling on the ground, or habitual recumbency with inability to rise.

The pain can be induced by either pinching of the withers, sharp elevation of tail, deep palpation of abdominal wall just behind the xiphoid process of sternum with the help of sharp jerk with a closed fist or knee, or lifting up with a poll (bamboo) and simultaneous auscultation of grunt at the trachea. Lactating animals show a sudden decrease in milk production which never return to normal even after recovery from acute attack.

ii) The signs of chronic local peritonitis are not characteristics. There is development of chronic anorexia, persistent decrease in milk than before, rough hair coat, chronic diarrhea or constipation with firm, dry, and scanty feces, cranial local abdominal pain at palpation, recurrent attacks of mild tympany (a cap of gas with doughy or impacted ruminal contents beneath), absence of severe signs of pain and fever, and no response to medical treatment are some signs of the disease. The pain can be elicited by strong wither pinch, xiphoid pressure with fist or pole, down hill walk, and percussion by hammer.

iii) Occasionally, there may be development of acute diffuse peritonitis which is manifested by profound toxaemia, severe depression, absence

of ruminal motility, and distension of lower abdomen. High fever (occasionally subnormal body temperature if condition develops soon after calving), presence or absence of abdominal pain, and sudden development of circulatory collapse are some evidences of diffuse peritonitis.

CLINICAL PATHOLOGY

In acute local peritonitis, there is mild leukocytosis with mild neutrophilia (with shift to left), during first 2-3 days are diagnostic. Later on, this change in blood count returns down but never to normal level through out the course of chronic disease. A severe leukocytosis with shift to left in indicative of acute diffuse peritonitis. Urine may contain protein or albumen due to passive venous congestion.

Abdominocentesis may be performed for obtaining peritoneal fluid for analysis of its chemical nature and identification of the type of pathogenic organism. Radiographic examination of the reticulum with animal at its dorsal recumbency may be of value in diagnosis. Use of metal detector is an other method for detection of ferrous metallic objects in the rumen or reticulum in animals with clinical disease. Non ferrous metallic objects can not be detected by this method.

NECROPSY FINDINGS

Animal dying due to peracute disease may have a lacerated myocardium with resulting hemorrhage or cardiac tamponade. Diffuse peritonitis is characterized by copious foul smelling peritoneal exudate, with fibrinous and exudative inflammation, and presence of causative organisms. Chronic cases may have extensive pericardial effusion with a thick layer of fibrin at the pericardium. The penetrating foreign body is generally still present within the wall of the reticulum (or may have fallen back), or embedded within a fibrous tract originating from reticulum to either pericardium or anywhere else.

DIFFERENTIAL DIAGNOSIS

In acute local traumatic reticulo-peritonitis, a sudden onset of anorexia and pain, ruminal stasis, sharp fali in milk, slight fever, and leukocytosis are diagnostic. Ephemeral fever has similar clinical picture but abdominal pain is absent, and it occurs in the form of an outbreak. Acute intestinal obstruction, simple indigestion, abomasal displacement or torsion, acute grain overload, tympany, pyelonephritis, postpartum metritis, mild toxaemia, hepatitis, vagus indigestion, pleurisy, and ketosis have lot of similarities in their clinical signs and need careful and thorough examination to differentiate them from reticulo-peritonitis.

TREATMENT

A. Conservative treatment;

a) Immobilization of the animal (for 10-15 days) by tying to one spot and performing all activities of feeding, watering, or milking at the site.

b) Standing the animal on an inclined plane keeping the front feet at least one foot higher than hind feet, may help to relieve pain and sometime pulling foreign body backward.

c) Feeding the animal with less roughage and more concentrate diet may ease the animal by lesser pressure toward its lower cranial parts.

d) Feeding a cylindrical or bar magnet, 7.5 cm long by 1-2.5 cm in diameter with round ends, to animal may help for embedding and immobilization of foreign body at situ.

e) Use of sulfonamides (Sulfamezathine @ dose of 100-150 mg/kg, IV or PO) and/or antibiotics (Penicillin or tetracycline), intraperitoneal and/or parenteral may help to reduce infection both local and systemic.

f) Mild laxative (magnesium sulphate, magnesium hydroxide) or other rumenitoric drugs may be used for improving motility and digestion.

g) Symptomatic treatment.

B. Surgical rumenotomy; It is surgical removal of the foreign body through rumenotomy incision with manual removal of foreign body from reticulum, if it can be reached. This techniques has the advantages of complete recovery in large per cent of cases, in places where such facility exists, and especially in chronic reticulitis.

PREVENTION

Prophylaxis administration of a bar magnet, at the age of 1-2 years, by oral route, preferably after fasting for 24 hours, is an effective and economical method for prevention of the disease particular in areas near big industrial cities. Use of magnetic platform for screening prepared-feed to remove metallic objects is an other attempt for prevention of this condition.

ACUTE RUMEN IMPACTION

Syn: RUMEN LACTIC ACIDOSIS, GRAIN ENGORGEMENT, GRAIN OVERLOAD, ACUTE CARBOHYDRATE ENGORGEMENT, TOXIC INDIGESTION, RUMEN OVERLOAD, ACUTE RUMENITIS.

It is an acute metabolic disorder of ruminants, caused by ingestion of large quantity of readily fermentable carbohydrate rich feeds, and resultant production of lactic acid in the rumen. Clinically the disease is characterized by complete anorexia, severe depression, rumin stasis, dehydration, incoordination, recumbency, coma, and customarily, death.

ETIOLOGY

This condition is commonly seen in dairy cattle, feed-lots, and sma ruminants (newly purchased for Eid-ul-Adha or slaughters) wh were recently shifted from green to concentrate feeds. The sudd ingestion of excessive quantity or accidental excess to read fermentable carbohydrates (starch or sugars) rich feed, such as grai (wheat, barley, corn, oats, rice, and sorghum), and less common potatoes, turnips, bread, sugar beets, grapes, flour dough, or excess molasses are among the disease triggering factors.

The amount and type of feed required to produce illness depends the type of grain, previous adaptation, nutritional status a condition of animal, and the nature of ruminal flora. This disea usually develops in animals suddenly exposed to grain feeds witho prior adaptation, consuming excessive grains because of accidental unrestricted access, or returning to grains following a long period off-feeding. For example; Animals that are accustomed to heavy gra diets may consume larger quantity of grain which may be toxic a lethal to unaccustomed animals. All the kinds of grains are consider more toxic when finely ground or even crushed than whole grains being readily exposed to micro-flora.

PATHOGENESIS

Normally, the rumen environment is anaerobic, having a pH of 6.5 and microbial population mainly of protozoa and bacteria, primarily Gram negative types. Microbial digestion convert carbohydrate volatile fatty acids, carbon dioxide, and methane. The volatile fat acids include acetic acid, propionic acid, and butyric acid. Within 2- hours of ingestion of excessive quantities of highly fermentable feed there is a marked change in number and species of the microbi population in the rumen. There is increase in number of fast growin Gram positive bacteria, such as Streptococcus bovis, which utilizes th

carbohydrate or starch to produce large quantities of lactic acid and other organic acids (including formic, valeric, and succinic acids).

Production of large quantity of lactic acid decreases the rumen pH to <5 at which the most of the normal cellulolytic Gram negative bacteria and protozoa are destroyed. As the rumen pH decreases (<4.5) the growth of St. bovis is also inhibited and the growth of Gram positive Lactobacillus spp. is promoted. These acid producing bacterial spp. contribute not only to the falling of pH levels but also to increase histamine levels by decarboxylation of histidine within the rumen.

The ruminal motility is decreased with a fall in pH, and at pH 5 or below it may totally be absent. The decrease in ruminal motility is mainly attributed to the increase in the production of volatile fatty acids (particularly butyric acid) and histamine rather than increased lactic acid or hydrogen ion concentration. The production of large quantity of lactic acid (both L and D type) and its salts (lactate), causes substantial increase in osmotic pressure in the rumen, which draw fluid from systemic circulation into the rumen and causes hemoconcentration and dehydration. Some of the lactic acid is buffered by ruminal buffers, if absorbed through rumen and intestine, by plasma bicarbonate buffering system, unless a toxic amount is accumulated which results in acidemia. With the progress of acidemia there is depression of central respiratory centers.

The overall pathophysiology will be anuria (due to decrease in renal blood flow), muscle weakness, cardiovascular collapse (hemoconcentration, dehydration, and decrease cardiac output), and death within 24 hours in acute cases. Several toxic substances (histamine, ethanol, methanol, butyrate, lactate, other endotoxins) are produced which also contributes to metabolic toxemia. Laminitis is common sign and is considered to be due to the absorption of histamine.

In chronic cases the high concentration of lactic acid for longer period may end up with chemical rumenitis, which predisposes to the development of mycotic rumenitis, liver abscesses, and pulmonary thrombo-embolism. Damage to ruminal mucosa in the form of necrosis and gangrene (due to necrosis of pillars at the dependent part) may lead severe consequences (acute peritonitis) which may be sufficient to kill the animal. Bacteria and fungi can also invade damage ruminal epithelium causing infectious rumenitis.

CLINICAL FINDINGS

The speed of onset and severity of clinical disease depends upon amount and type of feed, and amount and type of acids produced. The

clinical signs may range from simple indigestion to rapidly developing fatal acidemia. In mild cases, the animal may exhibit evidences of abdominal pain (kicking at the belly), reduced ruminal motility, anorexia, but remains alert, and pass soft faeces. These animal may recover with or without treatment within 3-4 days.

In severe cases animals are usually found down, staggering, or in a state of somnolence. There is complete anorexia, (no feeding or drinking) but some may engorge themselves soon after grain over feeding. Body temperature may be subnormal, but some may show fever (106 °F) especially during summer. The heart and respiration rates are increased, depending upon the amount of acidosis. Diarrhea is perfuse and the faeces is light colored containing shreds of mucosa, with sweet-sour odor, and may contain grains. Anuria and absence of faeces is considered as a grave prognosis. Dehydration is evident by sunken eyes and dry skin which further progresses as the case advances. The rumen fluctuates as if containing fluid at palpation and a gurgling and tinkling sound at auscultation. In some animals there may be impairment in the eyesight, and animal stagger and may bump into objects. Recumbency may follow within few hours, with absence or decrease response to external stimuli in a posture similar to parturient paresis. Rapid development of recumbency is suggestive of unfavorable prognosis (and require emergent and radical treatment).

A reduction in heart rate, mild increase in temperature, return of normal ruminal motility, passage of large amount of soft faeces, and urination are favorable signs. A sudden development of acute sign following apparent recovery from an initial attack of grain overload is an indication of fungal rumenitis (and peritonitis), which may end up with death within few days.

CLINICAL PATHOLOGY

A decrease in rumen pH (<5) with history of grain feeding is suggestive of grain overload. Microscopic examination of ruminal fluid reveals absence of normal Gram negative micro-flora and protozoa and replacement with Gram positive bacteria is common. A rise in hematocrit value (from normal 30-32 per cent to 50-60 per cent), and fall in blood pressure is indicative of dehydration. A fall in blood pH, calcium contents, and bicarbonates but increase in lactate and inorganic phosphate are other evidence of acidosis.

NECROPSY FINDINGS

In acute cases with sudden death, contents in rumen (also reticulum) are thin, and porridge like with fermenting odor. The cornified epithelium at the dependent areas can easily be wiped off, leaving a

dark, hemorrhagic surface beneath. The blood is thick and dark in color and visceral veins look prominent. Long standing cases, may show evidence of hemorrhages, necrosis, gangrene of rumen wall, and peritonitis. A terminal ischemic nephrosis, fungal hepatitis, abomasitis, enteritis may also be evident.

DIFFERENTIAL DIAGNOSIS

A low rumen pH, absence of rumen protozoa, ruminal stasis, gurgling fluid sound, diarrhea, dehydration, staggering gait, sub-normal temperature, and history of grain feeding are diagnostic. Parturient paresis is characterized by recumbency, hard faeces, without dehydration, history of parturition, and response good to calcium therapy. Acute antigenic toxaemia in coliform mastitis and in peritonitis have similar signs but careful examination may help to identify the cause of toxaemia.

TREATMENT

(A) Peracute cases should be considered for an emergency slaughter or vigorous therapy should be provided. It include;

i. Rumenotomy with washing of the contents and replacement with chopped hay or normal ruminal contents from other healthy animals if available, and fluid containing magnesium oxide or hydroxide.

ii. A 5 per cent solution of sodium bicarbonate >10-15 L for adult cattle by IV injection completed within 30-45 minutes followed by 10-15 L of 1.3 per cent solution of sodium bicarbonate in saline during next 6-12 hours for systemic acidosis.

iii. Withhold water and feed for next 10-20 hours, to avoid further engorgement.

(B) For acute cases of rumen acidosis;

i. Rumen lavage, by irrigation of rumen by pumping warm water followed by emptying out with a stomach tube of about 1 inch diameter (if facilities exists).

ii. Fluid therapy as described above. No oral water or grain feed.

(C) Moderate cases may be dealt utilizing the following steps;

i. Use of alkalizers such as magnesium oxide or hydroxide salt, @ dose of 500 gm in 10 L of water, PO, by stomach tube.

ii. Isotonic solution of sodium bicarbonate and electrolytes for systemic acidosis, dehydration, and restoration of renal function.

All animals should be monitored very closely for re-establishment of normal body functions and if required treat them accordingly.

A course of antibiotic (penicillin, tetracycline, or chloramphenicol) may be given to check the growth of Gram positive bacteria and fungal rumenitis. Recovered animals usually are deficient of normal micro-flora, which may be replaced through rumen transfaunation from some healthy animals.

Ancillary treatment may include; Antihistaminics (Meprasone or Avil, @ dose of 20-30 ml/LA, IM) for laminitis. Corticosteroids (Dexamethasone or Cortisone, @ dose of 10-15 ml/LA, IM) for shock. Vitamins especially thiamin (Vitamin B-compound, @ dose of 10-20 ml/LA, IM) to promote metabolism of lactic acid. Rumenitoric drugs (see simple indigestion) to promote ruminal motility. Injection of calcium borogluconate, @ dose of 300 ml/LA, IV, particularly in cases of calcium deficiency.

NB: Animal should be given only hay unless recovered completely and if desired should be progressively adapted to grain diet.

RUMEN ALKALOSIS

Alkalosis can be causes by an increased absorption of alkali, excessive loss of acid or a deficit of carbon dioxide. In ruminant alkalosis can occur when excessive amount of protein, or nonprotein sources are fed to animals, resulting in production of excessive ammonia, and so increase in rumen pH. A pH >7.5 is sufficient to cause clinical alkalosis. This condition may develop when feeding animal with urea, ammonium phosphate, or accidental ingestion of some common fertilizers that contain ammonium salts. Clinically the disease is not characteristic enough, but usually one can observe the signs of rumen dysfunction (as hypomotility, tympany, and diarrhea), slow and shallow breathing, muscle tremor, tetany, occasional vomiting, incoordination, weakness, tachypnea, tonic or clonic convulsions, and CNS excitation, and affected animals can die rapidly. The rumen fluid show alkaline pH between 7.5-8.5 and has a strong odor of ammonia.

Mild cases of alkalosis may develop when microbial fermentation is reduced while the animal continues to ingest saliva. It may also be seen in prolonged cases of indigestion, impaction or torsion of abomasum, decreased microbial activity due poorly digestible roughages, and absorption of volatile fatty acids (acetate particularly), causes increase in bicarbonate production, and during anorexic condition this mechanism is over compensated, resulting in alkalosis.

These mild cases seldom diagnosed clinically, and recovered by normal fermentation processes.

Balanced isotonic fluids containing an excess of chloride (as bellow) and no bicarbonate should be administered. Severe cases may be treated with acetic acid or vinegar, @ dose of 4-8 L, PO, depending upon amount of urea feeding or alkalosis.

Alkalosis solution containing the followings may also be used for treatment of this condition. It contains; sodium chloride (34 gm), potassium chloride (1.2 gm), calcium chloride (1.3 gm), and Distilled water (to make a total volume of 1000 ml).

VAGAL INDIGESTION
Syn: VAGUS INDIGESTION

It is a chronic indigestion caused by any lesions at vagus innervation of the forestomach and abomasum, causing varying degree of paralysis of the organs, and characterized commonly by delayed passage of ingesta, distension of abdomen, anorexia, and the passage of scanty and pasty faeces.

ETIOLOGY

The most common cause of vagus indigestion in cattle is traumatic reticulo-peritonitis. Less common causes vagus indigestion are those diseases having some lesion at the vicinity of vagal nerve supply. These may include actinobacillosis abscess, lymph nodes enlargement in tuberculosis, and lymphomatosis. In some cases vagal nerve injury cannot be demonstrated. Damage to the tension receptors situated in the right wall of the reticulum, which reflexly control vagal activity, may explain such cases. Typical signs of vagus indigestion has been produced (by Hoflund) by transection of various branches of abdominal vagus nerve.

PATHOGENESIS

The pathogenesis of the disease has been debated for years and still remains to be fully clarified because numerous investigations have yielded conflicting information. A number of syndromes develop depending upon the branches of the nerve which are involved and possibly, upon the degree of immobilization caused by adhesion of the reticulum to the diaphragm. On the basis of experimental results, four types of functional disturbances, with obstruction of ingesta at two sites have been reported.

1) Omasal transport failure (anterior functional stenosis) impairs flow of ingesta through the reticulo-omasal orifice (sphincter) and occurs with a). atony of the reticulo-rumen, often associated with chronic recurrent bloat, or b). normal to increased ruminal motility.

2) Pyloric outflow failure (posterior functional stenosis) impairs flow through the pylorus and occurs either continuously or in an intermittent and recurrent pattern (incompletely).

Depending upon the location and severity of the functional obstruction, distension, or impaction there will be varying degree of dehydration and a tendency towards a hypochloremic, hypokalemic alkalosis and uremia.

CLINICAL FINDINGS

The clinical signs may be grouped into 1. Ruminal distention with hyper-motility, 2. Ruminal distention with atony, and 3. Pyloric obstruction and abomasal impaction. In addition, a general clinical picture present in all types of disease includes, chronic anorexia, pasty or firm scanty faeces, right sided abdominal enlargement with or without bloat, normal temperature, pulse, and respiration, and no response to treatment.

1. Ruminal distension with hyper-motility is characterized by mild ruminal tympany which can be well palpated at rectal examination, distension of abdomen at left as well as lower right flank, pasty scanty faeces, normal to increased and forceful ruminal motility, normal temperature, and no response to standard treatment.

2. Ruminal distension with atony is characterized by mild ruminal tympany with reduced or absent of ruminal motility, absence of pain and other evidences of systemic involvement.

3. Pyloric obstruction with abomasal impaction is characterized by pasty scanty faeces, a firm distension of abomasum which can only be palpated at lower abdominal floor at rectal examination, decrease to absent of ruminal motility, chronic anorexia and death either suddenly due to abomasal rupture or following chronic illness due to inanition.

CLINICAL PATHOLOGY

Laboratory tests are of little use in identification of the disease. However, one can find mild leukocytosis, with monocytosis due to chronic traumatic reticulo-peritonitis. A varying in degree of hypochloremia, hypokalemic alkalosis, and hemoconcentration in abomasal distension and impaction may be found in vagus indigestion.

NECROPSY FINDINGS

Commonly, an impaction of the abomasum and empty or partly digested fiber in other organs is seen in animals died of primary vagus indigestion with distended abomasum. In ruminal impaction the contents are in advanced state to digestion and putrefaction. Mostly intestines are empty and if contain faeces, it is pasty and dark green in color. Lesions of traumatic reticulo-peritonitis in the form of adhesion or fistulous tract and some time containing metallic objects may be present.

DIFFERENTIAL DIAGNOSIS

The common clinical picture of chronic anorexia, ruminal distention with hyper-motility or atony, pasty scanty faeces, persistent loss of body condition and failure to respond to standard medical treatment is suggestive of vagus indigestion. Traumatic reticulo-peritonitis, abomasal and omasal impaction need to be differentiated from vagus indigestion.

TREATMENT

Treatment of vagus indigestion is purely symptomatic, though prognosis is not favorable in most cases.

1. In ruminal distension with fluid, the evacuation of ruminal fluid by passing a stomach tube in rumen and withdraw of fluid by gravity flow can provide relief to the animal. The use of trocar and canula or temporary rumen fistulation for removal of the gas from the rumen for the same purpose. Rumenotomy to remove the contents if chronic impaction, or foreign body from reticulum, provide comfort and recovery (and also to identify the cause). Drainage of abscess if present in the vicinity of rumen or reticulum can help in permanent recovery.

2. Rumenitorics (see indigestion), purgatives (magnesium sulfate, @ dose of 500 gm/LA, PO, Istizen, @ dose of 30 gm/LA, mixed in oil, PO), lubricants (mineral oil or vegetable oil 1-2 liters, PO), or rumen stimulants (see indigestion) may be tried.

3. Fluid therapy by IV route is recommended in cases of dehydration and electrolyte imbalance and should be continued until oral fluid is allowed, i.e. after re-establishment of reticulo-ruminal motility.

4. Use of antibiotics and anti-inflammatory if causative agent is known and inflammatory response present.

5. Rumen fluid transfaunation (cud transfer or rumen transplant) from healthy animal if required assist in recovery from ruminal distension.

6. Other symptomatic or supportive therapy if and as required.

LEFT-SIDED DISPLACEMENT OF ABOMASUM (LDA)

Displacement of abomasum from its normal position on the abdominal floor (from the midline to the right) to the left side of the abdomen between the rumen and the left abdominal wall.

ETIOLOGY

Left-sided displacement of abomasum occur more commonly in large, high-producing, adult dairy animals immediately after parturition, or occasional weeks before parturition. Atony of the abomasum and distension with gas or ingesta caused by either abnormally high volatile fatty acids concentration with heavy grain feeding or hypocalcemia, causes floatation of abomasum up along the abdominal wall on left (or right) and displacement. In addition, in late pregnancy, rumen is lifted from the abdominal floor by the expanding uterus and abomasum is pushed forward and to the left under the rumen. Following parturition the rumen is drops, trapping the abomasum, especially if it is atonic or distended with feed as is likely if the animal is fed heavily on grain. Occasional cases are also seen in calves, bulls, and beef animals with unexplained etiology.

PATHOGENESIS

Atony is considered to be the primary dysfunction of LDA. The atonic and distended abomasum becomes displaced upward along the left abdominal wall, usually lateral to the spleen and the dorsal sac of the rumen. It is primarily the fundus and greater curvature of the abomasum that becomes displaced which, in turn, causes displacement of the pylorus and duodenum. The omasum, reticulum, and liver are also rotated to varying degrees. The displacement invariably results in rupture of the attachment of the greater omentum to the abomasum. Compression by the rumen of the impounded part of the abomasum causes a great decrease in its volume and interference with its normal movements. There is some interference with the function of the esophageal groove due to light clockwise rotation of all the stomachs, and this impedes, though incompletely, the passage of digesta, which may leads to chronic inanition. A mild metabolic alkalosis due to mainly atony cont...

flow of hydrochloric acid into the abomasum and impairment of flow in to duodenum. A secondary ketosis may be a common complication in fat animals.

CLINICAL FINDINGS

A moderate to complete anorexia, decrease faecal output, decrease in ruminal motility, decrease in rumination, marked dropped in milk production, and ketosis are common signs usually developing soon after parturition. Moderate to severe dehydration, mild pain and little increase in pulse are also evident in early cases. The last 1-2 ribs are sprung, but the abdomen is sunken in the paralumbar fossa. A gurgling or tinkling, of more fluid rather than normal scratching sounds of rumen, may be heard on auscultation in the left paralumbar fossa. Simultaneous auscultation and percussion reveal a ping over the gas filled portion of the abomasum. The area of ping is between lower third of the abdomen in the 8th intercostal space to the paralumbar fossa. Faeces may be drier than normal or scant or watery. Rectal examination may reveal abomasum to the left of the caudo-dorsal blind sac of the rumen or at least perceive that the rumen is displaced medially. A chronic mild ruminal tympany (secondary type) and ketosis are common complication of LDA. The disease will run a chronic course with low level of production, thin body condition, and reduced sized of abdomen (gauntness) unless treated otherwise.

CLINICAL PATHOLOGY

There is no marked change in blood picture except increased level of PCV. A moderate to severe ketonuria is commonly present. Paracentesis of the LDA through 10-11 intercostal space in the middle third of abdomen will reveal the presence of fluid with no normal protozoa and very low pH. Ruminal fluid will contain normal motile protozoa and a pH 6-7.

DIFFERENTIAL DIAGNOSIS

Ruminal tympany, pneumoperitoneum, physometra, left displacement of caecum, and collapsed rumen may all produce pings on the left side of the animal. An assisted blow in rumen with a stomach tube and auscultation at left side may help to differentiate rumen from other structures. Rectal examination may help to differentiate displaced caecum and physometra. Paracentesis at left lower abdomen may help to differentiate rumen from abomasum. Acute traumatic reticulo-peritonitis may be differentiated by the presence of mild temperature, ruminal stasis, abdominal pain at palpation, and slight neutrophilia. Ruminal tympany have a wider area of low pitched pinging sound. Vagus indigestion is characterized by abdominal distension with or without an enlarged abomasum. Diaphragmatic hernia is

characterized by chronic ruminal tympany and abdominal enlargement.

TREATMENT

The treatment for LDA involves returning the abomasum to its normal anatomical location, occasionally treating the coincident electrolyte abnormalities and providing therapy for other complications such as ketosis and tympany.

The non surgical approaches involve casting the animal on right side, rolling her into dorsal recumbency, rocking back and forth, and then rolling the legs over the torso so she is on her left side. The animal is then allowed to stand. The gas within the abomasum causes it to float to a ventral location when the animal is in dorsal recumbency.

The surgical approaches include the blind-stitch abomasopexy that don't involve celiotomy. Other techniques may include left flank or right paramedian abomasopexy and right flank omentopexy.

PERITONITIS

It is an inflammation of the mesothelial lining of the peritoneal cavity, characterized clinically by acute abdominal pain, tenderness and rigidity of abdomen, constipation, and high fever.

Peritonitis can be classified according to its origin (as primary or secondary), onset (as peracute, acute, or chronic), region affected (as local or diffuse), and presence of infection (as septic or non-septic).

The peritoneum reflects off of the abdominal wall parietal and onto the major viscera. The dorsal aspect of the abdomen associated with kidneys and the pelvic region are considered retroperitoneal.

ETIOLOGY

The cause of peritonitis is multi-factional. Due to the primary etiology variance, all animals are considered at risk. Traumatic perforation of gastro-intestinal tract (traumatic reticulitis), punctured wounds (gunshot), horn-gore, falling on posts, abdominal surgery or laparotomy, and use of trocar and canula are some of the common causes of peritonitis in animals.

Other causes includes rupture of abomasum or intestine (in torsion), rupture of vagina (during parturition, correction of uterine prolapse, handling of dystokia, coitus), rupture of rectum (at its examination or parturition), rupture of urinary bladder (in urolithiasis), rupture of

ernal abscesses, abomasal ulcers, intraperitoneal injection of itant chemicals, migrating larvae (Gastrophilus sp., Strongylus lgaris (horses), Habronema sp., Oesophagostomum sp. (sheep), etc.

ATHOGENESIS

least six factors operates in the genesis of clinical signs. These ludes;

Toxemia and septicemia; Toxins are produced either by the usative bacteria (antigenic toxins) or the breakdown of tissues etabolic toxins) are readily absorbed through the peritoneum using severe toxemia. The severity of clinical signs produced pends upon the type of bacteria, area of peritoneum involved, and ount of toxins absorbed. (See also class notes on toxemia for other tails).

Shock and hemorrhage; The development of shock due to severe ection, sudden deposition of gut or uterine contents in the itoneal cavity, and extensive hemorrhage may have fatal outcome e to acute shock and peripheral circulatory failure.

Paralytic ileus; In acute peritonitis, paralytic ileum may arises as a ult of reflex inhibition of alimentary tract tone and movement. The effect will be functional obstruction of intestine as indicated ically by absence of defecation.

Accumulation of exudate; Accumulation of large quantity of lammatory exudate (fluid) in the abdominal cavity can cause lominal distension, interfere with diaphragmatic, respiratory, and estinal movements.

Abdominal pain; In peracute and diffuse type peritonitis, the emia, if severe enough, may inhibits the animal response to pain nuli. But in less severe cases, there will be acute pain as indicated ically by rigidity of abdomen, arching of back, and grunting at ing, standing, defecating, urinating, or palpation and percussion.

Development of fibrous adhesions; In chronic cases the development ibrous adhesions (a normal healing process) to any part of intestine abdominal wall) may physically inhibit intestinal motility, ulting in delayed passage of ingesta. These adhesions may break h severe exercise and thus may lead to acute peritonitis, norrhage, and pain.

INICAL FINDINGS

The clinical signs of peritonitis are variable and depend upon the nature of peritonitis. In per-acute and diffuse peritonitis caused by rupture of intestine, gravid uterus or bladder, the signs of acute toxemia, peripheral circulatory failure, and shock, in the form of severe depression, recumbency, coma, subnormal temperature, and weak pulse will be predominant. The signs of abdominal pain will be masked by the presence of severe toxemia and shock. In this type of peritonitis death is common outcome within 48-72 hours.

The acute and sub-acute peritonitis is characterized by complete anorexia, high fever (104-107 °F), constipation (no faeces or passage of dry, firm, mucous coated faeces), and abdominal distension. The affected animals are disinclined to sit or stand, move, and, if do so, with great care and grunting. Grunting can also be evident at urination, defecation, and deep palpation or percussion particularly at lower abdomen.

In chronic peritonitis, the acute signs are absent but variable degree of chronic anorexia, recurrent tympany and constipation, emaciation, poor growth and performance may be evident. Fibrous adhesions can be palpated at rectal examination.

CLINICAL PATHOLOGY

In per-acute diffuse peritonitis, examination of blood may reveals leukopenia, and degenerative shift to left. A marked leukocytosis with regenerative left shift and increase PCV will be present in acute diffuse peritonitis. In chronic form leukocytosis with lymphocytosis and monocytosis will be present. Abdominocentesis is a valuable aid in diagnosis of peritonitis. For this purpose a 2-3 inch long 16-18 gauge needle may be applied at the rumino-reticular recess at the floor of the peritoneal cavity, 3-7 cm medial to left milk vein. The peritoneal fluid will contain the causative bacteria, high protein contents, Pus cells, RBCs, fibrin clumps, and also can clot. Presence of fecal contents and green colored fluid in an indication of gut rupture.

NECROPSY FINDINGS

Gross hemorrhages, serosanguineous to purulent exudation, thick cheesy coating on abdominal viscera and peritoneum of depending parts, fibrinous to fibrotic adhesions, and foul smelling odor are some of the evidences of suppurative peritonitis. Presence of gut contents and greenish fluid in the peritoneal cavity are indicative of gut rupture which can also be identified. Tight fibrous adhesions among omentum, parts of intestine, mesentery, or abdominal wall are some evidences of chronic peritonitis.

DIFFERENTIAL DIAGNOSIS

The knowledge of history of any of the above mentioned cause and presence of clinical signs are necessary for diagnosis of peritonitis. However, it requires to differentiate from Traumatic reticulitis, Vagus indigestion, Acute intestinal obstruction, Abomasal torsion, Left or right abomasal displacement, Ketosis, Parturient paresis, Grain overload, Urolithiasis, Ascites, and suppurative processes in the abdominal cavity.

TREATMENT

i) Specific treatment according to the cause (if known).

ii) Use of antibacterials, PO, IM, IV, or via intraperitoneal (IP) routes, based upon the culture and sensitivity tests. For use of IP injection, only non irritant antibacterials diluted with normal saline should be recommended.

iii) Treat toxemia and fever if present.

iv) In cases of severe exudation, peritoneal lavage may be adopted using 10 gauge 4-6 inch long needle or canula to drain maximum exudate containing toxic metabolites. For this purpose, iodine solution diluted with normal saline (1:20) as a wash solution may be used.

v) Symptomatic treatment should also be focussed on use of fluid replacement therapy for the correction of dehydration, electrolyte imbalances, and acid-base abnormalities.

vi) Corticosteroids can be advised for endotoxic shock and stabilization of cell membranes along with broad spectrum antibacterials. Non-steroid anti-inflammatory drugs (phenylbutazone or sodium salicylate) may be considered as second alternative.

vii) Therapy for pain, anorexia, and constipation may also be considered if signs of these conditions are present.

viii) Supportive therapy as and if required.

ASCITES

It is the accumulation of fluid (exudate, transudate, blood, chyle, or bile) in the peritoneal cavity and is indicates an underlying disease process. The clinical signs of ascites may simulate peritonitis (except toxemia) as discussed elsewhere.

Peritoneal fluid analysis for differential diagnosis of ascites;
The peritoneal fluid may be withdrawn by peritoneocentesis. The nature of fluid may be of;

a) Exudate; An exudate is an opaque or serosanguinous type fluid, containing >2.5 g/dl total protein, >5,000 nucleated cell counts/ microliter, mainly of neutrophils, macrophages, and mesothelial or neoplastic cell types. This is mainly present in peritonitis, urinary tract rupture, pancreatitis, and Feline infectious peritonitis.

b) Pure transudate; It is a clear watery type fluid, containing <2.5 g/dl of total protein and <1000/microliter of nucleated cells count, occasionally neutrophils, macrophages, or mesothelial type. It may be seen in protein loosing nephropathy, protein loosing enteropathy, liver diseases, and congestive heart failure.

c) Modified transudate; Which is serosanguinous fluid containing 2.5 to 6.0 g/dl of protein and 250 to 20,000/microliter of nucleated cell count, mainly of macrophages, mesothelial cells, neoplastic cells, and neutrophils. This type of fluid may be seen in heart and liver diseases.

d) Chyle; It is a milky fluid, containing 2.5 to 6.0 g/dl of protein, and 250 to 20,000 cells/microliter, of lymphocyte, neutrophil, and macrophage types. It can be seen in lymphangiectasia, congenital anomalies, heart diseases, and thoracic duct diseases. or

e) Hemorrhagic; It is a clear red (blood) colored fluid containing 3.5 to 7.5 g/dl of protein and 1,000 to 20,000 nucleated cell/microliter of fluid, mainly erythrocytes, neutrophils, macrophages, mesothelial cells, or neoplastic cells. It can be seen in traumatic hemoperitoneum, bleeding neoplasia, and coagulopathy.

ASSAD

CHAPTER-4

DISEASES OF THE LIVER

Liver is an important visceral organ and perform a variety of metabolic functions for the body. It is affected, directly and indirectly, by a number of etiological agents, both infectious and non-infectious. In the primary hepatic diseases the clinical manifestation are mainly by intra-hepatic lesions, while in secondary diseases, the clinical signs are not associated with hepatic lesions. In this chapter the primary hepatic diseases are described.

HEPATITIS

It includes all types of degenerative, diffuse, and inflammatory diseases which effect the liver. Hepatitis is commonly characterized by the evidence of jaundice, abdominal pain, edema at various body parts, G.I. tract disturbances, and occasionally, nervous signs.

ETIOLOGY

A number of diseases are involved in hepatitis, some are not even identified. The etiological agents associated with hepatitis includes;

Toxicity with copper, phosphorus, arsenic, carbon tetrachloride, hexachloroethane, gossypol, chloroform, aflatoxicosis, and certain plant poisonings such as clovers, and water damaged alfalfa hay.

Infectious diseases/agents causing hepatitis are equine viral hepatitis, equine viral arteritis, infectious necrotic hepatitis, infectious canine hepatitis, leptospirosis, Cl. novyi type-B infection (black disease), bacillary hemoglobinuria, systemic histoplasmosis (mycotoxicosis), massive Liver fluke infestation, and migration of larvae of Ascaris spp.

Miscellaneous conditions causing hepatitis are congestive heart failure, fat cow syndrome, hepatic neoplasms and abscesses, post-immunization hepatitis, and Pyrrolizidine alkaloid poisoning.

PATHOGENESIS

In hepatitis the effect of toxigenic causes varies form focal or diffuse cloudy swelling to necrosis of liver parenchyma. With parasitic causes the changes depend upon the damage done by migrating parasites. If the cause is removed, because of regeneration capability of liver, there may be complete recovery.

With any inflammatory lesion, the liver cannot perform its normal detoxifying functions. Hence so, there is accumulation of excess amino acids and ammonia. With fall in blood sugar and metabolic toxemia there is development of nervous signs. Failure of anabolizing of protein (in chronic cases) will results in hypoproteinemia and edema. Photosensitization is an other complication develops due to retention of bilirubin in the tissues. In acute hepatitis there is increased tension to the capsule or its damage stimulate pain endorgans under the capsule and so the signs of pain.

CLINICAL FINDINGS

The signs of acute hepatitis includes anorexia, mental depression, muscular weakness, terminal somnolence, recumbency, coma with occasional convulsion, and jaundice. The color of the faeces changes and either constipation or diarrhea may develop. Sub-acute hepatitis may develop nervous signs like convulsion, hyperexcitability, aggressive behavior, and evidence of pain at palpation of liver. In chronic hepatitis, there is development of local (bottle jaw) or general edema (ascites, anasarca), and photosensitization with loss of hair/wool, along with much local itching.

CLINICAL PATHOLOGY

An increase in serum level of liver enzymes are diagnostic in detection of hepatic lesions. Sorbitol dehydrogenase (SDH) is a selective test for liver damage and its increase in serum is indication of liver damage so is very specific indicator in cattle, horses, and sheep. Aspartate aminotransferase (AST, previously called as SGOT) and L-alanine aminotransferase (ALT, previously called as SGPT) are also increased with liver damage, but are non specific. Glutamate dehydrogenase is an other specific enzyme increase in liver damage in ruminants. Measurement of icteric index of plasma used to measure the degree of jaundice.

NECROPSY FINDINGS

In hepatitis the liver is usually large with edges rounded and swollen. The color of liver may change from dark red in acute cases to pallor in chronic hepatitis. Histological lesions varies from cloudy swelling, degeneration, or necrosis of parenchyma to development of fibrous tissue (fibrosis). Yellow coloration of all body tissues and fat is an indication of jaundice.

DIFFERENTIAL DIAGNOSIS

Presence of nervous signs with evidence of jaundice and photosensitization are diagnostic of hepatitis instead of encephalomyelitis. Congestive heart failure is characterized by heart murmurs, brisket edema, and anasarca without the signs of nervous

derangement. Other diseases characterized by nervous signs, jaundice, and signs of toxemia require careful clinical and laboratory examinations for differentiation from hepatitis.

TREATMENT

The principles of treatment in hepatitis includes;

i) Specific treatment according to the cause; i.e. Removal of the source of toxin; Treatment of bacterial infections by using antibiotics (e.g., for leptospirosis use of streptomycin or chlortetracycline); For protozoan parasites use antiprotozoan (e.g. Diminazine, Acaprine); and for trematodes use of Zanil or Carbon tetrachloride.

ii) Administration of glucose (5-10 per cent, @ dose of 1-2 ml/kg/h, IV) helps to treat hypoglycemia. Peroral route may also be used to treat hypoglycemia.

iii) Administration of calcium (IV or PO) help to decrease toxicity caused by different metabolites, i.e. calcium borogluconate 10-20 per cent solution.

iv) Feed/diet containing high level of calcium, glucose, and protein have a protective effect against hepatotoxic agents.

v) Mild purgation with mineral oil, or magnesium sulfate, and enemas may be good for removal of toxic material from GI tract and release of constipation.

vi) Administration of water soluble vitamins (K) supplement (also B1) should be given as supportive therapy.

vii) Symptomatic treatment, e.g. control of nervous signs with sedatives or tranquilizers.

*Drugs which are known to cause hepatic injury in dogs and cats are acetaminophen, acetylsalicylic acid, androgenic anabolic steroids, chloroform, chlortetracycline, erythromycin; glucocorticoids, halothane; mebendazole, oxibendazole, phenobarbital, phenylbutazone, phenytoin, and trimethoprim-sulfonamide drugs.

JAUNDICE
Syn: ICTERUS

Jaundice is a clinical sign and is referred to the expression of a yellow discoloration of the serum and staining of soft tissue e.g. sclera,

mucous membrane, and skin, resulting from hyper-bilirubinemia (total plasma bilirubin <2mg/dl). It occurs when the rate of production of bilirubin exceeds the rate of elimination.

The intensity of tissue bile pigmentation depends on 4 important factors: i) The total serum bilirubin concentration, ii) The form of bilirubin (unconjugated or conjugated), iii) Capillary perfusion, and iv) Tissue composition (i.e. unconjugated bilirubin which is fat soluble is more deposited in fat tissues compared to conjugated water soluble bilirubin form which is high in elastic fibers).

ETIOLOGY

Based upon the origin, jaundice can be classified into;
A. Pre-hepatic (hemolytic or over-production) jaundice.
B. Hepatic (intra-hepatic, toxic, or infectious) jaundice.
C. Post-hepatic (extra-hepatic, obstructive, or biliary) jaundice.

A. Prehepatic jaundice; Caused by diseases which produce intravascular hemolysis. These include; Bacillary hemoglobinuria (toxin), leptospirosis (toxin), babesioses, anaplasmosis, theileriosis, trypanosomiasis (horse), equine infectious anemia, chronic copper and selenium poisoning (sheep), phenothiazine toxicity (horse), post-parturient hemoglobinuria (phosphorus deficiency), isoimmunization hemolytic anemia (young animals), immune mediated hemolytic anemia (foal), certain snake bite, blood transfusion reaction, and onion toxicity.

B. Hepatic jaundice; It is associated with diseases causing damage to hepatic cells (hepatitis). The etiological agents includes certain toxins e.g. copper, phosphorus, arsenic, carbon tetrachloride, gossypol, chloroform, aflatoxicosis, and certain plant poisoning. Infectious agents including equine viral hepatitis, infectious necrotic hepatitis, infectious canine hepatitis, and mycotoxicosis.

C. Obstructive jaundice; It is common with biliary obstruction as seen in cholelithiasis, nematode (Ascaris) larvae, trematodes (Fasciola spp.), or any tumor mass developing in the vicinity of bile duct. Escape of bile into surrounding tissues and its reabsorption may also occur with rupture of gallbladder and cholangitis.

PATHOGENESIS and CLINICAL SIGNS

Normally, the bilirubin is derived from the metabolism of various hemoproteins including hemoglobin, myoglobin, cytochromes, catalase, and peroxidase. About 80 per cent of bilirubin that is synthesized from hemoprotein comes from hemoglobin. The hemoglobin is released into blood form the breakdown of RBCs and is

phagocytized by the mononuclear cells in the liver, spleen, and bone marrow, where the heme is converted to biliverdin by heme oxygenase and then to bilirubin by biliverdin reductase. Bilirubin is then released into the circulatory system where it circulates bound to the albumin molecule. This type of bilirubin is referred as unconjugated, indirect-reacting, or lipid-soluble bilirubin. This unconjugated bilirubin is insoluble at physiologic pH and is only deposited in soft tissues that is high in fat.

The albumin bound bilirubin is carried through vascular system to hepatocytes. At the hepatic sinusoidal membrane the unconjugated bilirubin is dissociated from albumin and is taken up by liver cells and transported to smooth endoplasmic reticulum, where it is conjugated with glucuronic acid and glucose via glucuronosyl-transferase to form bilirubin glucuronide or bilirubin digluconide (also called as conjugated, direct reacting, or water soluble bilirubin). The conjugated bilirubin is secreted out into the bile canaliculus by an active transport process and stored into gall bladder until feeding which initiates it emptying into duodenum.

In the small intestine bacteria reduce most of the conjugated bilirubin to a group of colorless compounds known as urobilinogens, which can readily be reabsorbed by the intestines and re-secreted in bile, but a small amount (20 per cent) is excreted in the urine. Most of the urobilinogens (90 per cent) is oxidized to urobilin (stercobilin) and eliminated in the faeces. The stercobilins impart color to the faeces.

In hemolytic type of jaundice there is excessive production of heme molecule which leads to the excessive formation of bilirubin beyond the hepatic reserve capacity to remove the unconjugated bilirubin. This type of jaundice is characterized by moderate degree of yellowing of mucosae, anemia, and by the presence of hemoglobinuria in severe cases. There is also presence of excessive urobilinogens in the urine and presence of indirect bilirubin in the serum. The faeces and urine color give a yellow tinge. The treatment of this type should be focussed to compensate the lost blood along with specific and symptomatic treatment of jaundice. (If the hemolysis occurred prior to 24 hours, then direct bilirubin will also be present in serum)

In hepatic jaundice there is impairment in hepatic uptake, storage, conjugation and excretion of bilirubin, and so the changes in urine and faeces are in between hemolytic and obstructive jaundice. There is rise in both urobilinogens and bilirubin in the serum and urine. There should be significant damage to hepatic cells before the animal develops hyper-bilirubinemia. For treatment of hepatitis the antibiotic of choice includes tetracycline and neomycin.

In obstructive jaundice, there is blockage in hepatic ducts, gall bladder, cystic duct, common bile duct, or major duodenal papilla interfering the release of bile release into the intestine. The majority of bilirubin may be conjugated form but both forms could be detected in blood. The color of sclera and mucosa become intense yellow. There is complete absence of bile pigments in the faeces (complete obstruction) giving it a clay color and the presence of high level of direct bilirubin in the serum. Because of failure of bilirubin to pass in the GI tract, there is absence of urobilinogens in urine and only bilirubin is present in large quantity giving it an intense yellow color. If there is partial obstruction of bile flow the faeces will contain some bile pigments and urine may have urobilinogens.

Differential basis of jaundice:	Type A	Type B	Type C
Indirect bilirubin*	+++	-	-
Direct bilirubin (intense color)	-	+	+++
Impairment of bile flow	-	+	+++
Intense yellow color of sclera	+	++	+++
Clay colored faeces (no bile)	-	±	++
Yellow colored faeces (bile)	+++	+	-
Urobilinogens in the urine	+++	±	-
Anemia	++	-	-
Hemoglobinuria	++	-	-
Yellow coloration of M.M.	+	++	+++
Van den Berg's test	indirect	bi-phasic	direct
Alkaline phosphatase (E')	-	+	++
Prothrombin time increase	-	+	++
Cholesterol	-	±	++
Amount of serum bilirubin	± 3 mg/dl	± 20 mg/dl	>20 mg/dl

*Indirect bilirubin, which has not passed through the hepatic cells and is not excreted through kidneys e.g. hemolytic jaundice. Staining of tissues is much more pronounce with direct than with indirect bilirubin.

TREATMENT

1. Pre-hepatic jaundice; For excessive hemolysis, blood transfusion will make the difference between health and disease.

2. Hepatic jaundice; The general principles of treatment of jaundice is the same as for hepatitis.

3. In obstructive jaundice surgical removal of the obstruction (i.e. gall stones or tumor mass) may be the only choice.

CHAPTER-5

DISEASES OF CARDIOVASCULAR SYSTEM

Cardiovascular system plays a significant role in blood circulation, exchange of oxygen and electrolytes, and excretion of body waste. The diseases of cardiovascular system are infrequently treated in large animals practice as compared to dogs and cats but they often present diagnostic problems. Those conditions that are of primary interest to veterinary clinicians are discussed in this chapter.

DEFINITIONS

Cardiac Dysrhythmia and Arrhythmias; Cardiac dysrhythmia and arrhythmias are abnormalities in the normal heart rate, rhythm, or conduction pattern. This may result from abnormalities of impulse generation, impulse conduction, or a combination of both. In the normal heart, the impulse is generated in the sinus node. There is variability in normal range for heart rates in large adult animals. For example;

The normal pulse rate in horses is 30-50 beats/minutes, cattle and buffaloes, 50-70 beats/minutes, sheep and goats, 70-90 beats/minutes, dogs, 70-80 beats/minutes, and cats, 90-110 beats/minutes.

Dysrhythmia are more common in horses than in other domestic animals. In horses (also goats) dysrhythmia at rest is considered benign or functional and is due increase in vagus tone which should disappear following atropine injection. Cattle don't have benign dysrhythmia. The pathological dysrhythmia are termed as atrial or ventricular fibrillation, tachycardia, and bradycardia. The primary dysrhythmia are caused by pathological conditions of the heart, e.g. myocarditis, pericarditis, valvular diseases, and cardiomyopathy. The secondary dysrhythmia are caused by non-cardiogenic conditions such as excitement, fever, electrolyte imbalances, G.I. tract disturbances, toxemia, and pulmonary hypertension.

Fibrillation; This is an increase but irregular cardiac rhythm (non coordinated contractions of atrium and ventricle). In the ventricular fibrillation the ventricle contracts faster than atrium resulting in decrease cardiac output. This condition may occur in terminal stages of most sudden fatal diseases, i.e. lightening stroke, electrocution, certain acute plant poisoning, quick overdose of general anesthesia, severe toxemia, and cardiac diseases. In atrial fibrillation the atrium contracts faster than ventricle due to lack of coordinated atrial electrical activity, and is usually not identified clinically but through

its effects on ventricular functions. This condition is seen secondary to diseases of myocardium, autonomic nervous system imbalance, G.I. tract disturbances, electrolyte or acid-base imbalances, anesthetics, or tranquilizers use. The clinical signs of atrial fibrillation are exercise intolerance, exercise induced epistaxis, respiratory dyspnea, syncope, and signs of congestive heart failure.

Tachycardia; An increase in heart rate which is influenced by increased discharge of impulses from sino-atrial node which has its intrinsic rate of discharge but can also be modified by vagus nerve. It is commonly seen in pain, excitement, exercise, hyperthermia, fall in blood pressure, or administration of adrenergic drugs.

Bradycardia; This is a slow heart rate due to decrease rate of discharge from sino-atrial node. It may be normal in athletic animals at rest but can occurs in hypocalcemia, anesthesia, increase in intracranial pressure (space occupying lesions), digitalis intoxication, hypoxemia, hyperkalemia, and adrenocortical insufficiency.

Cardiac murmurs; It is the turbulent flow of blood through heart/blood vessels causing an increase of resonance in adjacent structures. An increase in flow velocity or reduced blood viscosity (as in anemia) predisposes to murmur development. The characteristics of the murmur depend on the velocity of blood flow and the nature of the structures that cause it to vibrate. The murmur could be systolic (occurring anytime between the 1st and 2nd heart sounds), diastolic (occurring anytime between 2nd and 1st heart sounds), or continuous (occurring throughout the cardiac cycle).

The location of cardiac murmur correspond to the location of one of the heart valve. Murmur associated with mitral valve be heard in the left 5th intercostal space just dorsal to the level of the elbow, pulmonic valve at the 3rd intercostal space 4-6 cm below the level of the shoulder, aortic valve murmur are located just dorsal to pulmonic valve in the 4th intercostal space, and with tricuspid valve in the right 3-4 intercostal space between the shoulder and the elbow. These murmurs are classified in different grades, i.e. from I to VI, grade I being the lowest and grade VI of the highest intensity murmur (loudest).

Grade-I. A soft murmur, heard only after minutes of cardiac listening.

Grade-II. A soft murmur that is heard immediately on auscultation.

Grade-III. A murmur of moderate intensity.

Grade-IV. A loud murmur associated with a palpable thrill.

Grade-V. A loud murmur, but is not heard when the stethoscope is removed from the chest wall.

Grade-VI. A loud murmur, which is audible with the entire stethoscope chest piece and even it held away from chest wall.

Muffled heart sounds; Auscultation of the heart sounds requires that the vibrations generated by the heart be transmitted through the tissues of the thorax to the outer chest wall with sufficient amplitude to be heard. Factors such as enlarged lungs, large and thick chest, obesity, and even faulty stethoscope attenuate heart sounds and cause heart sounds to be muffled. Primarily the heart sounds are muffled because of displacement of heart from its normal position, tumor or abscess in the thoracic cavity, congestive heart failure, pericardial effusion, traumatic pericarditis, pulmonary emphysema, pneumothorax, or weak cardiac contractions as seen in hypocalcemia.

Cardiovascular exercise intolerance; Exercise intolerance is a clinical sign associated with diseases of many body system. Dyspnea, cough, fatigue, sudden stopping, poor performance, and a prolonged elevation in heart rate following mild exercise are signs suggestive of exercise intolerance. It can also be noted if there is sudden change in the level of performance and production, lowered enthusiasm for work, or excessive sweating. Weakness can be manifested as recumbency, difficulty in rising from recumbency, muscle tremors, reluctance to move, or toe-dragging.

The diseases associated with exercise intolerance are myocardial diseases, cardiac dysrhythmia, aortic or pulmonary artery rupture, vascular thrombosis, congenital cardiac defects, congestive heart failure, pericarditis, pulmonary emphysema, Pulmonary congestion and edema, CNS disturbances resulting in loss of consciousness, and hyperkalemia.

Syncope; It is a sudden collapse and loss of consciousness or fainting.

Venous distention and Pulsations; The jugular venous pulsations observed in the neck are primarily a reflection or right atrial and right ventricular activity, which is influence by blood volume, right ventricular cardiac output, and right atrial contractility. Jugular venous pulse can be observed in normal animals, but it seldom radiates more than one third of the distance from thoracic inlet to the ramus of the mandible when the head is held in a normal position.

Abnormal pulsation occur with increased resistance to ventricular filling, regardless of the cause. Distention and pulsation jugular vein are usually associated with an elevated right ventricular pressure. The common diseases having jugular pulsation in animals are congestive heart failure, cardiomyopathy, chronic atrial fibrillation, pericarditis, myocarditis, pulmonary hypertension, heart based tumors (lymphosarcoma, fibrosarcoma, squamous cell carcinoma), abscess, jugular venous thrombosis, brisket disease, and over-hydration.

Cardiac enlargement; It is a compensatory response of heart to persistent increased workloads associated with either physiological (athletics) or pathological (cardio-vascular diseases) conditions. The components of cardiac enlargement are either dilatation, hypertrophy of myocardium, or combination of both. Hypertrophy of myocardium is a usual response to an increased pressure load, and there is hypertrophy of an individual fibers with an increase in total muscle mass. Dilatation is a usual response to an increase volume load and probably results from fiber rearrangement.

The clinical significance of cardiac enlargement is that it indicates the presence of a significant volume or flow load on the heart, or the presence of myocardial diseases and a reduction of cardiac reserve. Cardiac enlargement can be detected by careful auscultation of large area of audible heart beat, radiograph, and ECG examinations.

ESSENTIAL DRUGS USED IN THE CARDIO-VASCULAR ARREST

Vasoactive and cardio-stimulatory agents used are epinephrine HCl (adrenaline), isoproterenol HCl (Isuprel), dopamine HCl (Dobutrex), and methoxamine HCl (Vasoxyl).

Drugs used to specifically increase contractibility are calcium chloride and digoxin (Lanoxin)

Drugs used to combat acidosis are sodium bicarbonate and trihydroxy-methylamino-methane (THAM)

Drugs used to treat acute cardiac arrhythmias are atropine (Atropine sulphate), isoproterenol (Isuper HCl), digitoxin (Lanoxin), diltiazem (Carizem), glycopyrrolate (Rubinul-V), propranolol HCl (Inderal), lidocaine HCl (Xylocaine without adrenaline), quinidine sulfate (Quinaglute, Cardioguin), procainamide (Pronestyl), brethlium tolysolate (Bretylol), acetylcholine KCl, and Verapamil (Calan).

Drugs used to stimulate ventilation are soxapram HCl (Dopram) and methetharimide (Mikedimide).

Drugs used to combat cerebral edema are oxygen, mannitol (Osmitrol 20 per cent), and dexamethasone.

Common steroids used in cardiac patient are prednisolone sod. succinate and dexamethasone.

Non-steroidal anti-inflammatory drugs are aspirin, ibuprofen, phenylbutazone, flunixin meglumine, naproxen, and dipyrone.

CONGESTIVE HEART FAILURE

In congestive heart failure (CHF) the heart due to some intrinsic or extrinsic defects is unable to maintain circulatory equilibrium even at rest, resulting in dilatation and congestion in the venous circuit, edema (lungs and/or periphery), and cardiac enlargement.

ETIOLOGY

Congestive heart failure is seen with diseases which interfere with the flow of blood away from heart. These could be either intrinsic or extrinsic cardiac defects.

a) Examples of intrinsic cardiac defects are endocarditis, myocarditis, pericarditis (septic or traumatic), myocardial asthenia, hydropericardium, valvular defects, neoplasias, and congenital cardiac defects.

b) Examples of extrinsic cardiac defects are pulmonary hypertension (Cor-pulmonale) and systemic hypertension. In these conditions due to increased resistance the heart is required to perform strenuous work to eject an equivalent amount of blood, and thus resultant cardiac enlargement and CHF.

PATHOGENESIS

With either of the etiology there is reduction in contractile power and increased blood load toward the heart. The compensatory mechanism of heart operates in response to increased blood load. This includes increase in heart rate and ventricular filling, and with time, dilatation and hypertrophy of myocardium. If this compensatory mechanism reaches it physiological limits, the heart will be unable to cope with circulatory emergencies, i.e. exercise, resulting in CHF and cardiac tamponade. (In advanced cases, the heart may be unable to cope with

the circulatory requirement even at rest). As a consequence of CHF, there will be increase in venous hydrostatic pressure and decrease in blood flow from heart toward the tissues. The outcome will be edema development in different body organs and tissue hypoxia.

In right-sided CHF, the congestion and edema will be in greater circulation involving liver, kidneys, subcutaneous tissues, and G.I. tract, with resultant edema and impairment of their normal functions. For example, Anuria and hypoproteinemia will develop due to reduction of blood flow through kidneys followed by anoxic damage to the glomeruli, respectively. Whereas, in left-sided CHF, the congestion and edema will be in pulmonary circuit (pulmonary edema), which may interfere with normal oxygenation process.

CLINICAL FINDINGS

A general body weakness, jugular venous distension and pulsation, weak arterial pulse, muffled heart sounds, edema at different body parts, exercise intolerance (indicated by respiratory distress, fast pulse, and prolonged time to return to normal limits even with mild exercise), and sudden collapse (syncope) are some common signs of CHF.

With right-sided CHF, there may be development of generalized edema (anasarca), ascites, hydrothorax, and/or hydropericardium. The Left-sided CHF may be manifested by respiratory distress (even at rest), soft productive cough, moist rales especially at the ventral side, and in severe cases, cyanosis with acute dyspnea. Other signs include anuria, albuminuria, diarrhea, epistaxis (in horses), anorexia, severe depression with shuffling gait are common in long standing cases of CHF.

CLINICAL PATHOLOGY

There will be increase in venous pressure than normal which may be noted as blood passage with pressure at venepuncture. Albuminuria can be identified by urine analysis and is indicative of hypoxic damage to glomeruli. The edematous fluid obtained by needle puncture will be transudate in nature, that may contain protein in later stages, and is an indication of anoxic damage to capillary endothelium. Evaluation of pericardial fluid (through pericardiocentesis) may be required for differentiation between infectious and non-infectious causes of pericarditis.

X-rays examination may reveal cardiac enlargement (loss of chamber contour) and pulmonary edema. A low amplitudes QRS may also be noted in ECG examination.

NECROPSY FINDINGS

Cardiac enlargement with hypertrophy of ventricle(s) and presence of causative lesion of CHF may be seen at postmortem. Pulmonary congestion and edema is present in left-sided CHF, and anasarca, ascites, hydrothorax, hydropericardium, liver enlargement and congestion, and edema of G.I. tract may be present in right-sided CHF.

DIFFERENTIAL DIAGNOSIS

Following diseases may require to be ruled out.

The peritonitis is characterized by the presence of exudative fluid containing causative bacteria and pus cells, without any evidence of cardiac involvement. Rupture of urinary bladder is associated with evidences of anuria/dysuria, abdominal pain, increased BUN, and urine mixed peritoneal fluid. Hepatic fibrosis is characterized by edema, jaundice, and photosensitization. In hypoproteinemia due to parasitism, an edema at submandibular space along with signs of anemia are common. In anaphylactic shock there will be pulmonary congestion and edema along with other signs of hypersensitivity reaction.

TREATMENT

i. Specific treatment according to the cause.

ii. Use of drugs which improve the contractility of heart, and includes digitalis glucosides (Digitoxin and Digoxin) and theophylline derivatives (Theophylline and Aminophylline) by IM use, but contraindicated when infection is the primary cause. Digitalis is used in large initial doses for a short time followed by small doses as maintenance for longer time.

iii. Use of diuretics (mercurial, acetazolamide, chlorothiazide, or fursemide) and restriction of sodium salt may assist in reducing edema.

iv. Avoid violent exercise in early stages and no exercise in later stages of CHF.

v. Venesection in severe cases of pulmonary edema may reduce venous pressure on heart. About 4-8 ml/Kg of blood can be withdrawn at one time. The process may be repeated at one week interval, if desired.

vi. Drainage of the serous cavities by paracentesis in order to remove edema fluid, as much as possible but dehydration should be avoided.

ACUTE HEART FAILURE

It is an acute cardiac arrest characterized clinically by sudden syncope, severe pallor of mucosae, falling, followed by either death or recovery.

Actually severe CHF.

ETIOLOGY

The causes of acute heart failure (AHF) may be grouped into the followings;

1. Severe disorders of cardiac filling and is mostly seen when there is traumatic rupture of either atria, ventricle, pericardium, aorta, pulmonary, or coronary arteries.

2. Failure of heart to act as a pump and it may be seen in (a) ventricular fibrillation. and severe tachycardia (associated with bacterial myocarditis, nutritional myopathy with selenium or copper deficiency, plant poisoning, electrocution, and lightening stroke), (b) Severe bradycardia (associated with overdose of any sedatives/anesthetics, e.g. xylazine, barbiturates, halothane, or IV injection of concentrated potassium salt.

3. Sudden increase in workload usually associated with acute anaphylaxis or rupture of aortic valve.

PATHOGENESIS

Under the influence of any etiological factors mentioned above, the cardiac output will be seriously reduced to the point where there is development of severe degree of tissue anoxia. Brain is the most sensitive organ to be first affected as indicated by nervous signs (unconsciousness and clonic convulsions). It has been observed that a complete anoxia for 15 seconds may lead to irreparable damage in brain tissues. An acute reduction in arterial blood flow will lead to the development of severe pallor of mucosae and absence of pulse. In less severe cases, the signs of acute congestive heart failure, such as pulmonary congestion and edema, may be noted.

CLINICAL FINDINGS

The clinical signs of AHF are sudden development of dyspnea, staggering, falling, unconsciousness, which may be followed by death accompanied by deep asphyxial gasps with or without clonic convulsion or recovery. A marked pallor of mucosae, absence of palpable pulse, or heart sounds may be observed in severe cases. In less severe cases, signs of tachycardia, arrhythmias, distress and fast breathing, and pulmonary edema may be noted. If animal survive for

long, the clinical sings will be similar to that of pulmonary edema, ascites, and hydrothorax.

CLINICAL PATHOLOGY and POSTMORTEM FINDINGS

There is no laboratory or postmortem changes diagnostic for AHF. In subacute cases there may be engorgement of pulmonary vessels and development of lung edema. Macro or microscopic lesions of primary myocardial diseases could be identified.

DIFFERENTIAL DIAGNOSIS

Diseases of nervous system have similar clinical features and pattern of development as of AHF. Epilepsy is characterized by repetitive and transient attacks of unconsciousness with tonic convulsions. Many diseases of animals are characterized by sudden death, but sudden development of severe tachycardia or bradycardia, pallor of mucosae, absence of pulse, falling, unconsciousness, and clonic convulsions are indicative of AHF.

TREATMENT

At the diagnosis of AHF emergent and symptomatic treatment should be tried. For example, in cases of excessive bradycardia, use of Adrenaline by both IV and IM routes may do some good (but not in severe tachycardia or fibrillation). In cases of tachycardia or fibrillation, the use of Lignocaine without adrenaline (IV) should be advised. In cases of cardiac arrest cardio-pulmonary resuscitation (CPR) can, some times, makes difference between death and recovery.

PERIPHERAL CIRCULATORY FAILURE

It is a condition of reduced cardiac output due to failure of venous return toward the heart. This disease is clinically characterized by muscle weakness, subnormal body temperature, tachycardia with low pulse pressure, coma, mild clonic convulsion (occasionally), and death (in severe cases).

ETIOLOGY

The etiology of peripheral circulatory failure (PCF) can be classified into two groups.

1. Vasogenic failure: In this the total blood volume is normal but because of pooling of blood in dilated greater vessels there is reduction in total circulating blood volume. This condition is commonly seen in shock either septic (e.g. Acute diffuse peritonitis and gangrenous mastitis), endotoxic (Coli-form mastitis and Acute intestinal obstruction), or metabolic deficiency (Parturient paresis).

2. Hematogenic or hypovolemic failure: In this form there is reduction in total circulating blood volume either due to severe hemorrhage, massive burns, or severe dehydration.

PATHOGENESIS

In PCF the cardiac output is reduced owing to the loss of total circulating blood volume which result in tissue hypoxia and reduction in cellular/organ functions. In acute hypoxia there may be development of irreversible damage, particularly in central nervous system and myocardium, leading to sudden death. The advancement of hypoxic damage to tissues is directly proportional to the speed of loss of circulatory blood volume. It is also influenced by the speed of replacement of blood constituents by the compensatory mechanism (including evacuation of blood from stores, vasoconstriction, and hemopoiesis).

CLINICAL SIGNS

The severity of clinical signs depends upon the amount and speed of circulatory blood loss. In mild cases of PCF there is general body depression and muscle weakness. Sub-normal body temperature, cold extremities, severe pallor of mucosae, increased capillary refill time, tachycardia, low pulse pressure, weak heart sounds, and dyspnea with shallow breathing are signs of acute PCF. A listlessness, severe depression, falling, coma, unconsciousness, and clonic convulsions are indicative of nervous hypoxia.

CLINICAL PATHOLOGY

Laboratory tests for the determination of anemia, degree of dehydration, acid-base imbalance, and hypoproteinemia are of diagnostic significant for primary etiology. A decrease in RBCs, WBCs, and thrombocytes counts, and low hemoglobin percentage are significant in acute hemorrhage. An increased level of PCV and hypoproteinemia is indicative of dehydration and protein loss, respectively.

DIFFERENTIAL DIAGNOSIS

A history of road accident, surgery, or other evidences of blood loss, dehydration, severe burn followed by above mentioned clinical signs are diagnostic for PCF. Other causes of shock and convulsions require careful identification to rule-out them.

TREATMENT

1. The treatment of PCF should be immediate and directed toward restoration of total blood volume (to avoid tissue anoxia). This could be achieved by whole blood transfusion in hemorrhage, plasma or plasma

expanders in shock, and infusions of isotonic fluids in cases of dehydration.

An animals with hemorrhage showing PCV less than 20 per cent require immediate blood transfusion. Animals with evidence of shock having total protein contents less than 3.5 g/dl require plasma).

2. For acid-base and electrolyte imbalances, use of normal sodium bicarbonate solution (1.3 per cent) particularly if blood pH is <7.1 and multi-salt solutions, e.g Ringer's solution, is recommended.

3. The signs of shock may be treated with corticosteroids (e.g. Dexamethasone, @ dose of 5-10 mg/kg, or methylprednisolone, @ dose of 30 mg/kg), antihistaminics, or non-steroid anti-inflammatory drugs. In case of septic or endotoxic shock, heavy doses of broad-spectrum antibiotics, particularly of bacteriostatic type, should also be used.

4. Symptomatic and supportive therapy, if and as required.

TRAUMATIC PERICARDITIS

It is the traumatic perforation of the pericardial sac by any foreign metallic object originating from the reticulum followed by the development of congestive heart failure with toxemia.

ETIOLOGY

Perforation of the pericardial sac can occur as a sequelae to traumatic reticulitis caused by perforation of reticular wall by any sharp metallic object. There is greater tendency for any sharp metallic object to be directed cranially toward the pericardium causing pericarditis following initial perforation of reticular wall. There are more chances of this condition to occur during the last trimester of gestation, soon after parturition, or following (truck) journey. (See also traumatic reticulo-peritonitis).

PATHOGENESIS

A metallic object may be directed toward thoracic cavity and perforate pericardial sac causing traumatic pericarditis (TP) following perforation of the reticular wall. This may also be accompanied by the introduction of mixed bacterial infection into the pericardium, originating from the reticulum, causing severe local inflammatory response. The development of trauma and infection initiate early clinical signs, but their persistence is not essential for further progress of the disease. The collection of inflammatory exudate in the pericardium is sufficient enough to interfere cardiac activity to cause congestive heart failure and development of antigenic toxemia.

CLINICAL FINDINGS

The TP may be acute or chronic. Rapid the development of generalized edema - bad is the prognosis. In acute or subacute cases of TP the common clinical signs are complete anorexia, diarrhea with scanty faeces, habitual recumbency, arching of the back, and abduction of elbows. There is grunting while sitting, urination, and defecation which can also be induced by pinching of the wither (back), xiphoid pressure, palpation of cardiac area, or down-hill walking (slope walking test). There may be high fever, increased pulse, and respiration rate. The engorgement and pulsation in the jugular vein and development of gross edema particularly at the brisket and ventral abdomen are diagnostic signs of pericarditis.

Auscultation of cardiac area may reveals a friction (rub) heart sound which wax and wane with respiration in early stages and later, gurgling, splashing, and tinkling sounds, due to hydropericardium. The area of cardiac impulses may be increased (can also be heard on right side) due to cardiac enlargement.

The disease may run a chronic course where the obvious clinical signs of toxemia and congestive heart failure as above may be absent. The animal will be in poor health condition, with variable appetite, emaciation, and may never perform well. The cardiac sounds are muffled due to pericardial effusion but fluid sounds may not be auscultated. The presence of jugular pulse and tachycardia are usually present.

CLINICAL PATHOLOGY

A pronounced leukocytosis with neutrophilia (and shift to left) in acute stages and mild leukocytosis with lymphocytosis in chronic stage are indicative of persistent infection. Paracentesis of pericardial fluid can assist in identify the nature of pericardial fluid and type of infection. The infection is usually of mixed type (caused by both Gram +ive and -ive aerobic and anaerobic bacteria). The pericardial fluid is usually straw color, slightly blood tinged, contains high protein content, and has a foul odor. There may be an elevation of myocardial isoenzymes such as aspartate transaminase, creatine phosphokinase, and L-alanine dehydrogenase.

NECROPSY FINDINGS

Evidences of edema at the subcutaneous tissues, brisket, abdominal cavity, inter-mandibular space, pleural cavity (pleurisy), G.I. tract, liver, and lungs may be present. Cardiac enlargement, gross

distension of pericardial sac, and pericardial cavity containing serosanguineous yellowish colored exudate containing flakes of fibrin (and fibrosis) with foul odor can be noted. In chronic cases gross thickening and fibrous adhesions of pericardium to surrounding tissues and epicardium may also be present.

A cord like fibrous sinus tract, connecting the reticulum and pericardium, and usually presence of the causative (metallic) object may be located at situ (but not essential). Histopathological examination may reveals pericardial, epicardial, and myocardial fibrosis with infiltration of eosinophils, neutrophils, lymphocytes, and plasma cells.

DIFFERENTIAL DIAGNOSIS

Endocarditis is usually accompanied by abnormal heart sounds except friction (rub) sound as noted in TP. Lymphomatosis is characterized by abnormal heart sounds, lymphocytosis, leukocytosis, and enlargement of lymph nodes. Congenital cardiac defects are usually identified in early ages. Diaphragmatic hernia, and thoracic tumors or abscesses require careful rule-out.

TREATMENT

The treatment of TP is usually not very successful, the prognosis is usually guarded and so early slaughter is recommended at confirmation of the condition.

For symptomatic and supportive treatment look for topics on congestive heart failure, edema, and toxemia.

A short term survival treatment may includes repeated pericardial drainage by means of pericardiocentesis or pericardiectomy.

Prevention and control (see traumatic reticulitis).

CHAPTER-6

DISEASES OF BLOOD AND BLOOD FORMING ORGANS

ANEMIA

Anemia is decrease in oxygen carrying capacity of blood and is identified by pallor of conjunctiva/mucosae, increased capillary refill time, exercise intolerance, and decreased packed cell volume.

ETIOLOGY

The etiology of anemia may be grouped into i) Depression anemia caused by inefficient RBCs production, ii) Hemolytic anemia caused by increased RBCs destruction, and iii) Hemorrhagic anemia caused by excessive blood loss. The former is also called as aplastic anemia and both the later are called as responsive and regenerative type anemia.

i) Depression anemia; This type of anemia develops due to decrease production of RBCs or Hb, mostly associated with deficiency of different minerals and vitamins. Examples include, deficiency of copper and cobalt (ruminant), iron (mostly young animals), pyridoxine (calves), and vitamins E, B-6, B-12, and folic acid (all animals). Depression anemia may also develop in any chronic systemic suppurative conditions, massive bone fractures, radiation injuries, parasitism (Trichostrongylosis), certain plant (Bracken fern) or chemical poisoning, stem cell disorder (aplastic anemia), and neoplasia of bone marrow.

ii) Hemolytic anemia; It develops due to increase destruction of RBCs resultant decrease in total red blood cell mass. It may be seen in certain protozoan diseases (e.g. babesiosis, anaplasmosis, theileriosis), bacterial diseases (e.g. leptospirosis, bacillary hemoglobinuria), viral diseases (e.g. equine infectious anemia, feline infectious anemia), and rickettsial diseases (e.g. ehrlichiosis). Hemolysis of RBCs may also occur in mineral deficiencies (e.g. post-parturient hemoglobinuria causes by phosphorus deficiency), plant poisoning (e.g. rape and other cruciferous plants and onions), chemical poisoning (e.g. phenothiazine in horses, copper in sheep), animal poisoning (e.g. snake poisoning), immunological disorders (e.g. autoimmune hemolytic anemia), and in blood transfusion reaction.

iii) Hemorrhagic anemia; (To see details read chapters on hemorrhage, and peripheral circulatory failure).

PATHOGENESIS

The exact pathogenesis of anemia is diverse with each disease referred in the etiology. The net effect of acute anemia is the loss in total oxygen carrying capacity of blood and tissue anoxia, resulting in depression of various body functions and peripheral circulatory failure. In sub-acute cases the cardiac output is reduced so there is increase in heart rate and stroke volume. In chronic anemia the effects are slow and sub-clinical.

CLINICAL FINDINGS

Pallor of mucosae, increase capillary refill time, decrease performance, exercise intolerance, tachycardia, tachypnea, are some of the important clinical signs of anemia. Additional findings include weakness, lethargy, anorexia, increase intensity of heart sounds, and systolic murmurs (due to reduced blood viscosity). In terminal stages there may be tachycardia and dyspnea with increase in depth of respiration without increased rate. In hemolytic anemia the pallor mucosae may be overshadowed by over production of bilirubin, and staining of mucosae a yellow color. In most protozoan diseases hemoglobinuria, fever, and secondary renal failure can also be noted.

CLINICAL PATHOLOGY

A decrease in total RBCs, Hb, and PCV are good indications of anemia. In both hemorrhagic and hemolytic anemia, there is increase in number of immature RBCs (macrocytosis) in blood which appear granular and stain darker with Methylene blue stain. There may also be an increase in mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH). A decrease in hemoglobin concentration to <50 per cent of normal is required to develop clinical anemia. In hemolytic anemia the serum color is pink to red but a clear serum with low total protein is associated with hemorrhagic anemia. In deficiency anemia, the RBCs count may be nearly normal with low Hb and a decrease in MCV and MCH (microcytosis). In this type the RBCs stain lighter in color without any granular look. Melena is an indication of G.I. tract hemorrhage. Black vomit or dark, bloody stained feaces.

Erythrocytic parasites can be identified in stained blood films. Anaplasma marginale are round basophilic inclusion bodies at the edges of the RBCs and are present in their highest number before a hemolytic crisis. Babesia trophozoites can also be seen in erythrocytes as round bizarre rods or typical piriform formation. Babesia bigemina of cattle are large piroplasms, whereas, B. bovis are small and the organisms are connected at their narrow ends by filaments. Babesia bovis are seen in sheep and goats and B. equi and B. cabali in horses. Theileria spp. infect mainly lymphocytes but can also be seen in RBCs

of ruminants. Common Theileria spp. are T. parva (East-coast fever), T. anulata, T. mutans, and T. ovi.

MCV = Hematocrit / Erythrocyte count (million).

MCV is a reflection of mean erythrocyte size. An increase MCV indicates a regenerative type anemia, since immature erythrocytes are larger than mature ones, called macrocytosis. Iron deficiency can result in decrease MCV because cells undergo an extra division as a result of inadequate Hb concentration.

MCH = Hb (gm/100 ml) x 10 / RBC count (million).

It is an estimation of amount of Hb per RBC. An increase of MCH is an indication of either 1. presence of immature RBCs in the peripheral blood, or 2. hemolysis of RBCs. Iron deficiency may also cause decrease in MCH.

MCHC (per cent) = Hb (gm/100 ml) x 100 / Hematocrit (per cent).

This is the most accurate erythrocyte indices. Erythroid regeneration and iron deficiency will cause a decrease MCHC, and hemolysis cause increase MCHC.

NECROPSY FINDINGS

A pallor of tissue, thin watery blood, and contracted spleen are indicative of anemia. Other signs of primary disease should be looked for their diagnosis.

DIFFERENTIAL DIAGNOSIS

The difference among different types of anemia can be made as under.

	Hemorrhagic	Hemolytic	Aplastic
History of hemorrhage	+	-	-
Hemoglobinuria	-	+	-
Pallor of mucosae	+	-	-
Jaundice	-	+	+
Fever	-	+	-
Reduction in PCV	+	+	-
Pink plasma	-	+	+
Polychromasia	+	+	-
Macrocytosis	+	+	-
Microcytosis	-	-	-
Erythrocytic parasites	-	+	+

TREATMENT

1. Specific treatment according to the primary cause.

2. Non-specific treatment includes whole blood transfusion in acute hemorrhage, and hematinics and vitamins in hemorrhagic or hemolytic anemia (see hemorrhage).

3. Application of symptomatic or supportive treatment and treatment of complications as and if required.

EDEMA (Oedema)

Edema is an abnormal accumulation of fluid in the body cavities or interstitial spaces of tissues where connective tissue structure is relatively loose, caused by an alteration in the equilibrium between capillary permeability, tissue spaces, and lymphatic vessels or damage to capillary endothelium. The edematous swelling is painless, soft, pits on pressure, and tends to gravitate at the dependent body parts.

ETIOLOGY/PATHOGENESIS

The development of edema can be classified as under;

i) Increase hydrostatic pressure; In this case edema develops due to local or generalized, congestion and hypertension of blood within the venous circulation at capillary level, resulting in escapes of fluid through endothelial pores into the surrounding tissues (i.e. intercellular spaces). Examples include Congestive heart failure (generalized edema), local venous thrombosis (local edema), local portal hypertension due to cirrhosis of liver (ascites), and local mammary vein compression by enlarged fetus (udder/teat and ventral body edema, particularly seen mares, goats, and cows).

ii) Decrease plasma colloid osmotic pressure; Generalized edema can also develop due to decrease in plasma colloidal osmotic pressure due to decrease level of plasma protein (<5 gm/100 ml of blood), which let the fluid part to escapes into the surrounding tissues. The loss of plasma protein may be noted either in heavy parasitic infestation, particularly of Fasciola spp. Strongylus spp. Haemonchus spp. or Bunostomum spp., or Chronic nephrosis. Hypoproteinemia may also be seen in cases of chronic hepatitis, John's disease, chronic starvation and malnutrition, and is due to decrease anabolizing of proteins.

iii) Obstruction of lymphatic; Localized or generalized edema may be noted due to obstruction in lymphatic flow, caused either by local neoplastic growths (e.g. Lymphosarcoma), inflammation (e.g.

Lymphangitis or lymphadenitis), or elevated central venous pressure (e.g. heart failure).

iv) Small vessel endothelial damage; It may be due to the following reasons;

a). Local liberation of vasoactive amines (histamine) form platelets, mast cells sensitized to Ig-E, basophils, may cause vasodilation and the leakage of vascular fluid (and proteins) across endothelial barrier, e.g. Angioneurotic edema, Urticaria, Equine infectious anemia, Purpura hemorrhagica, and equine viral arteritis.

b). Toxins damage to capillary endothelium may cause the leakage of vascular fluid into surrounding tissue spaces, e.g. Gas gangrene, Malignant edema, Anthrax in horses, Gut edema, Equine viral arteritis.

c). Hypoxic damage to capillary endothelium caused by anoxic anoxia may also cause the release of vascular fluid into surrounding tissues, e.g. Congestive heart failure, Nitrite, and Carbon monoxide poisonings.

CLINICAL FINDINGS

The edema can be local or general. The edematous swelling is typically cool, soft, painless, without any sign of acute inflammation, and pits on pressure leaving indentation following deep palpation. Anasarca is the accumulation of edematous fluid in subcutaneous tissue of ventral abdomen, thorax, brisket, and inter-mandibular space. Ascites referred to edema of peritoneal cavity. Hydrothorax is edema of pleural cavity.

The edematous swelling have variable effects on body functions and is depending upon its severity. In cerebral edema, there will be derangement of nervous activities, edema of chest cavity will be associated with respiratory problems, and edema of intestine may cause diarrhea.

CLINICAL PATHOLOGY

The edematous fluid is transudate in nature, i.e. straw color, clear, low specific gravity, do not clot, and is free from protein, bacteria, and inflammatory cells. It some times may contain high protein contents, due to severe capillary damage, so the fluid may clot, have high specific gravity, and may also contain free blood. Laboratory tests are required for the identification hepatitis, nephrosis, peritonitis, congestive heart failure, and other diseases characterized by clinical edema.

DIFFERENTIAL DIAGNOSIS

edema requires rule-out form urolithiasis, peritonitis, emphysema, and other diseases with signs of abdominal distension. Subcutaneous and peritoneal accumulation of urine may be noted in urethral obstruction or its rupture and urinary bladder rupture. Anuria, dysuria, increases BUN, and uriniferous smell from breath are characteristic of bladder/urethral rupture. Peritonitis is characterized by accumulation of exudative type fluid containing inflammatory cell, causative bacteria, and with signs of toxemia. Emphysema is a puffy type swelling which crackle under pressure, as seen in Gas gangrene.

TREATMENT

1. Correction of the primary disease, e.g. cardiotonics in congestive heart failure, anthelmintics in heavy parasitism, and antibiotics in bacterial infections.

2. Restriction of water and salt (particularly sodium) intake in diet may aid in reducing edematous swelling.

3. Use of diuretics by oral, parenteral, or both routes, e.g. Fursemide (lasix, @ dose of 2-3 ml/SA, and 20-25 ml/ LA, IM). The use of ammonium chloride, potassium nitrate, and potassium citras can also be used to produce diuresis.

4. Antihistaminics, such as mepyramine, promethazine, diphenhydramine, or corticosteroids can be used in reducing edematous type swellings.

5. Colloidal solutions, i.e. whole plasma or plasma expander (6 per cent Gelatin or Dextran), or whole blood needed to be used to correct hypoproteinemia.

6. Aspiration of fluid, by paracentesis with a wide bore needle, may be carried out (slowly) in severe cases of edema, but one should avoid excessive fluid loss. It may cause complications such as peripheral circulatory failure or development of dehydration.

7. Supportive therapy includes the use of vitamin-B complex and vitamin-A, mineral mixture containing iron, calcium, copper, and cobalt, and diet rich in protein and carbohydrates.

A "diuretic mixture" commonly used for large animals consists of magnesium sulphate (200-300 gm), ammonium chloride (15-20 gm), potassium nitras (15-20 gm), sodium citras (15-20 gm), and water (QS) to make a drench.

HEMORRHAGE

It is the loss of whole blood from vascular system, externally or internally, resulting in development of signs of peripheral circulatory failure and anemia.

ETIOLOGY

Spontaneous laceration or traumatic rupture of any of the major blood vessels (or organs), resulting in escape of blood, in or out side of the body, is the major cause of acute hemorrhage. Other (minor) causes of hemorrhage are abomasal ulcers, rupture of coronary artery or ventricular wall in traumatic pericarditis, rupture of middle uterine artery, surgical endeavors, broken horn, open castration, rupture of spleen, massive infestation of blood sucking parasites, coccidiosis, bleeding neoplasms, erosion of the carotid artery by guttural pouch mycosis (horse), and disseminated intravascular coagulopathy.

PATHOGENESIS

The effects of hemorrhage on an individual are due to total loss of circulating blood volume and its constituents, i.e. erythrocytes, plasma protein, and electrolytes. Therefore, in severe hemorrhage, there will be development of peripheral circulatory failure, hypovolemic shock, and anemia, all could lead to fatal outcome. However, in sub-acute hemorrhage, the clinical signs may not be evident, owing to the compensatory mechanisms operation. It include vasoconstriction (operates within few sec of hemorrhage), evacuation of stored blood from spleen and liver (operates within few min), withdrawal of fluid from tissue spaces (operates within 12-24 hours), and increase hemopoiesis (operates after 96 hours).

CLINICAL FINDINGS

The clinical signs of acute hemorrhage are muscles weakness, staggering, recumbency, (later coma), along with rapid heart and respiration rates, weak pulse, sub-normal body temperature, cold extremities, pallor of mucosae (hypovolemic shock), and occasionally, cardiovascular collapse (acute peripheral circulatory failure). Listlessness, dullness, and low performance are signs of sub-acute hemorrhage. An inspiratory dyspnea, pallor of mucosa, and depression following thoracic injury or accident are signs of thoracic hemorrhage.

CLINICAL PATHOLOGY

A history or external evidences of hemorrhage may be necessary for confirmatory diagnosis. For the suspicion of internal hemorrhage, abdominocentesis or thoracocentesis is required to identify hemoperitoneum and hemothorax, respectively. Examination of blood

for PCV, RBCs, hemoglobin, or total plasma protein may appear normal, if checked with 12 hours of acute hemorrhage which was controlled. Because, changes in these parameters are masked by compensatory mechanism. Therefore, a history of an accident or recent surgery along with clinical evidence of peripheral circulatory failure is suggestive of internal hemorrhage. A decrease in RBC count, hematocrit, total plasma protein, and hemoglobin level are suggestive of persistent sub-acute hemorrhage. A constant lowering of PCV (from say 40 per cent to 20 per cent within 24 hours) is indicative of acute loss which needs immediate blood transfusion, compared to low but stable PCV (20 per cent), in slow loss of blood, which does not need immediate blood transfusion. In ruminants, polychromasia, Howell-jolly bodies, and occasionally, nucleated erythrocytes may be present following 4th day of hemorrhage.

NECROPSY FINDINGS

A thin watery blood and extreme pallor of body tissues/organs are seen in animals died of severe hemorrhage. If hemorrhage is internal (hemoperitoneum or hemothorax), un-clotted blood may be found at those sites.

TREATMENT

Acute hemorrhage should always be considered an emergency.

1. The first-aid of acute external hemorrhage should be aimed to control hemorrhage (by pressure wraps, ligatures, artery forceps, and/or used of styptics (i.e. Alum, Tr. Benzoinco, or Tr. ferri-perchloride). In cases of internal bleeding, parenteral use of blood coagulants, such as vitamin-K, Thrombin, Prothrombin (e.g. Coagulin, Adrenochrome, Dicynone, Kapilin, Hemostop, Anaroxyl, or Carbazochrome), or other coagulation factors (if deficient) should be advised to control hemorrhage.

2. Acute hypovolemic shock should be treated by prompt I/V administration of crystalloid sodium solutions, @ dose of 20-40 ml/kg. In dogs the use of hypertonic (7.2 per cent) saline, @ dose of 5-7 ml/kg, initially, followed by normal saline may reverse the sequelae of severe hemorrhagic shock.

3. In cases of severe anemia due to hemorrhage, whole blood transfusion is the most satisfactory treatment. A PCV less than 20 per cent in animal with blood loss, suggests a depletion of RBCs reserve, and a persistent reduction of PCV over a period of 24 hours to 12 per cent indicates the need for blood transfusion. A low but stable PCV (12-20 per cent) does not necessitate transfusion.

Blood can be collected from a healthy donor (large animal) at the rate of 4 liters in 10 minutes, in a sterile container containing anticoagulant solution (@ dose of 10 ml of 3.85 per cent sodium citrate solution/100 ml of blood). A total of 10-15 ml of blood/Kg body weight can be drawn off at one time without any danger of health from a healthy donor and whose blood is not be sensitized by previous blood transfusion or multiple pregnancies. Clotted blood inevitably block the needle, so it has to be discarded.

Horses and cattle display a high degree of blood type polymorphism. It is estimated that the number of possible blood types is 400,000 in horses and well over 2 trillion in cattle. So identification of complete compatible individual is nearly impossible. The cross-matching of blood to determine compatibility is not often practiced in animal, except in horses where risk of isoimmunization hemolytic anemia is high. Repeated transfusion after more than a week interval from one donor may provoke anaphylactic shock. The sensitization to a particular donor may persists for years. Therefore, to avoid any risk of mismatching, a matching test can be carried out by mixing the serum of the recipient with the cells of the donor.

Two drops of donor's blood are mixed in 2 ml of a 3.85 per cent sodium citrate solution and 2 drops of the mixture are added to 2 drops of the recipient's serum on a glass slide and gently rocked. Agglutination between incompatible samples is readily seen. Alternatively, inject a small amount of blood (100-200 ml) into the recipient and wait at least 10-15 minutes for any adverse reaction (i.e. hiccough, dyspnea, muscle tremor, salivation, frequent coughing, piloerection, lacrimation, fever, sometimes ruminal tympany, and sudden collapse). If there is no immediate reaction then blood can be transfused safely. However, sometimes, in later stages, hemoglobinuria and abortion may occur if the blood is not compatible.

In case of anaphylaxis, stop immediately the blood transfusion and administer normal saline solution instead. Treatment with Adrenaline hydrochloride, 1:1000, @ dose of 0.5-1 ml, IV, and 4-5 ml, IM, in large animals, immediately after observing anaphylactic reaction. Antihistaminics or corticosteroids can be given in combination with adrenaline or alone as a second alternative.

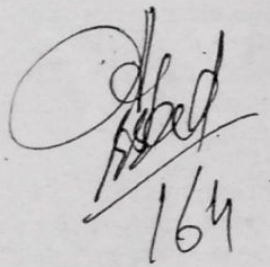
Blood should be administered very slowly in order to avoid overloading of the circulation and acute heart failure, i.e. at dose rate of 4.5 liters of blood in one hour in adult animals. The survival of blood cells in the recipient cow is limited, i.e. up to 72 hours following the first transfusion and less than 1-2 hours following the second

transfusion. Even then the blood transfusion is useful to reverse acute anemic anoxia and so for survival of the individual.

4. Plasma and plasma extenders, such as 6 per cent Dextran or 6 per cent Gelatin, can be administered as an alternative to whole blood, specially to compensate the loss of plasma protein contents, however, their therapeutic value in anemia is questionable.

5. Chronic blood loss may be treated with iron preparations (ferrous sulfate or ferrous fumarate). Folic acid and vitamin-B complex should also be given for improving hemopoiesis.

Note: Cardiac stimulant or vasoconstrictor drugs are of no value in hypovolemic shock.



CHAPTER-7

DISEASES OF RESPIRATORY SYSTEM

The respiratory system comprises of nares, larynx, trachea, bronchi, bronchiole, and pulmonary parenchyma. Gaseous transfer occurs in the alveoli of the lungs. Failure of gas transfer can result in severe consequences. Anoxia (hypoxia) is responsible for most of the clinical signs of respiratory diseases. Any foreign body entering into the respiratory system, through blood or airborne, is taken care by the respiratory defence. Marked anatomical and physiological variations exist in lungs of various animal species, and it implies to variations of normal lung functions and response in diseases. In this chapter disease problems of respiratory system are dealt with.

RESPIRATORY DEFENCE

the respiratory defence mechanism is multifactorial and include sneezing, local nasal antibodies, cough, sero-mucous blanket, mucociliary transport clearance mechanism, alveolar macrophages and other mononuclear cells, local secretory antibody (Ig-A), glycoproteins (lysozyme) and lactoferin, systemic antibodies (Ig-A, Ig-G, Ig-M), interferon, complement system, and cellular defense.

MAJOR CLINICAL SIGNS OF RESPIRATORY DISEASES

COUGH; Cough is a sudden noisy expulsion of air through the glottis to clear mucus and other material from the tracheo-bronchial tree. It is an involuntary reflex but can also voluntarily be initiated or suppressed. The reflex pathway involves sensory (irritant) receptors of nerve fibers that extend between epithelial cells and ramify throughout the tracheo-bronchial tree from the level of the larynx to the respiratory bronchiole. These irritant receptors can be stimulated either mechanically (pinching of trachea), chemically (Carbon or irritant gasses, as Ammonia), inflammatory conditions (Tracheitis or Bronchitis), or by chemical mediators (Histamine) produced in an individual.

The cough could be productive or moist (passing out sputum), non productive or dry (without expulsion of sputum), and painful or painless. A PAROXYSMAL cough is a dry, painful, and hacking type cough which is episodic in occurrence. Cough originating from upper respiratory tract is usually loud, harsh, coarse, hacking, dry, and of non productive type. A lower airway cough is usually soft, productive and tends to persist more chronically than of upper respiratory tract. The productive cough is difficult to asses in animals as the sputum at the pharynx region is usually swallowed in. Therefore, a swallowing reflex following cough is indicative of productive cough. Occasionally

the coughed out sputum, in large animals, may be found sticking at front wall.

Common causes/diseases associated with cough in animals are; Equine influenza, equine rhinovirus infection, bacterial pneumonia, bacterial pleuritis, pharyngitis, strangles, glanders, pasteurella pneumonia, hemophilus pneumonia, lungworm infestation, interstitial pneumonia, mycoplasmal pneumonia, aspiration pneumonia, pharyngitis, choke, aspiration or inhalation pneumonia, pharyngeal paralysis, guttural pouch infection, pulmonary abscess, congestive heart failure, equine viral arteritis, fungal pneumonia, pulmonary tumor, anaphylaxis, African horse sickness, pulmonary anaphylaxis, pneumothorax, congestive heart failure, diaphragmatic hernia, tuberculosis, pulmonary aspergillosis, virulent sheep and goat pox, Peste des petits ruminants, and contagious caprin pleuropneumonia.

NASAL DISCHARGE; It is inflammatory secretions (non gaseous material) that escapes from the respiratory tract through the nostrils. Nasal discharge can be classified as serous, mucoid, purulent, hemorrhagic (sanguinous), or a combination of above. The discharge could be perfuse (copious) or scanty, continuous or intermittent, and unilateral or bilateral. It can sometime be mixed with ingesta.

Mostly normal cattle keep their external nares clean by licking them with their tongue, but sick animals often neglect to do so, as a result, discharge may be seen accumulated at the nares. Any condition causing inflammation of respiratory tract may result in increase in nasal secretions. The secretions are initially serous, become mucoid, and then purulent as the disease progresses, particularly with bacterial infections.

FOUL ODOR (OZENA); The ozena associated with nasal discharge is generally an indication of anaerobic bacterial infection, local necrotizing conditions (fungal rhinitis), nasal tumor, turbinate necrosis, or necrotizing (gangrenous) pneumonia. Maxillary sinusitis or dental diseases may also be associated with ozena.

Common causes of nasal discharge in animals are; Equine influenza, pharyngitis, laryngitis, tracheitis, nasal polyps, bacterial pneumonia, strangles, glanders, Guttural pouch mycosis, aspiration or inhalation pneumonia, lung abscess, choke, pasteurellosis; nose bots, lungworm infestation, interstitial pneumonia, parainfluenza 3 virus, mycoplasma pneumonia, infectious bovine rhinotracheitis, aspergillosis, tuberculosis, ascarid migration, African horse sickness, nasal tumors, nasal actinobacillosis, pulmonary aspergillosis,

theileriosis, virulent sheep and goat pox, nasal schistosomiasis, pulmonary embolism, and immune deficiency states.

Common causes of ingesta in nasal discharge in animals are; Cleft palate, choke, pharyngitis, strangles, guttural pouch infection, glossopharyngeal nerve damage, botulism, retropharyngeal abscess, pharyngeal abscess or trauma, tetanus, pharyngeal fistula, esophageal stricture, gastric dilation, rabies, gastric tumor, persistent right aortic arch, plant or chemical poisoning, diaphragmatic hernia, and acute intestinal obstruction.

EPISTAXIS; Epistaxis is actual hemorrhage originating from the nostrils, either primarily because of vessel damage in nasal cavity, nasopharynx, guttural pouches, and lungs or secondary to some systemic diseases. The volume of hemorrhage may be as small blood stained nasal discharge to a perfused blood flow form nostril(s) and mouth. -Hemorrhage originating form the nasal septum is usually unilateral and often occurs spontaneously, but of nasopharynx or areas below is usually bilateral.

HEMOPTYSIS; It is the expectoration of pure blood or sanguinous sputum from mouth.
Common causes of epistaxis in animals are; Guttural pouch mycosis, nasal trauma, nasal polyps, pharyngeal trauma, purpura hemorrhagica, thrombocytopenia, pharyngeal trauma, nasal bots, dehorning, nasal adenoma, exercise induce pulmonary hemorrhage, pulmonary neoplasia, hepatitis, pasteurellosis, multiple clotting defects, equine infectious anemia, skull fracture, gunshot wounds of respiratory tract, snake bite, acute renal diseases, congestive heart failure, plant and chemical poisoning, fungal granuloma, anthrax, bacillary hemoglobinuria, and disseminated intravascular coagulopathy.

TACHYPNEA AND DYSPNEA; Tachypnea (polypnea) is an increase in respiratory rate than normal. It can be either physiological (due to pain, exercise, fear, high environmental heat, and anxiety) or pathological associated with diseases of lungs, chest cavity, heart, nervous systems, and general systemic states.

Dyspnea or respiratory distress is a clinical sign that implies labored or difficult breathing. It indicates an inappropriate degree of effort to breath, based on assessment of its rate, rhythm, and character. Dyspnea (shortness of breath) describes the subjective feeling of difficult, uncomfortable, or unpleasant breathing in human patients. In animal, the manifestations of dyspnea include increased respiration rate, extension of head and neck, mouth breathing

(ruminants), nostrils flaring (horse, sheep and goat), extra abdominal efforts, elbows abduction, stridor, anxious expression, and inactivity.

Common causes of tachypnea or respiratory distress in animals are; All types of pneumonia, hyperthermia, pain, acidosis, anaphylaxis, anemia, cardiac diseases, smoke inhalation, strangles, septicaemia, toxaemia, pulmonary embolism, neoplasia, inhalation of irritant foams, hyperthermia, hypocalcemia, hypomagnesemia, anemia, hypovolemia, bloat, electrocution, burns, systemic toxoplasmosis, snake bite, photosensitization, botulism brisket disease, nasal septal abnormalities, guttural pouch infection, tracheal stenosis, diaphragmatic hernia, pneumothorax, thoracic trauma, tetanus, electrocution, cleft palate, purpura hemorrhagica, pulmonary edema and emphysema, white muscle disease, nasal tumor, choke, laryngitis, tracheitis, thoracic trauma, and pneumothorax

ANOXIA (Hypoxia); It is failure of the tissue to receive an adequate supply of oxygen. It can be classified according the nature of etiology.

i. Anoxic anoxia; It can occur when there is defective oxygenation of the blood in the pulmonary circuit and is primarily associated with diseases of respiratory system (pneumonia, pulmonary emphysema, hydrothorax, pulmonary edema), heart, large vessel, poisoning (botulism and strychnine), and specific diseases (tetanus).

ii. Anemic anoxia; It occurs when there is deficiency of hemoglobin per unit volume of blood or reduced oxygen carrying capacity of blood. This type of anoxia is seen in all types of anemias and poisoning caused by nitrite and carbon monoxide.

iii. Stagnant anoxia; This type of anoxia develops due to decrease oxygen exchange in tissues because of reduced capillary blood flow, even though the total oxygen load and oxygen tension in arterial blood is normal. Examples include congestive heart failure, peripheral circulatory failure, and local ischaemia.

iv. Histotoxic anoxia; It occurs when tissue oxygenation system fails to take up oxygen from blood, as seen in Cyanide poisoning.

The compensatory mechanism as a result of anoxia include; a) Increase in depth and respiratory movements mediated by the chemoreceptors of carotid and aortic bodies, b) stimulation of splenic contraction to evacuate blood stored and increase in production through erythropoiesis in the bone marrow that may result in polycythemia, and c) increase in heart rate and stroke volume that outcome in an increase in minute volume.

If this compensatory mechanism fails to supply adequate quantity of oxygen to tissues, signs of dysfunction of various organs occur. Nervous system is the most susceptible to anoxemia resulting in cerebral anoxia. Myocardial asthenia, renal and hepatic dysfunction and reduction in motility and secretion of alimentary tract are other consequences developing in terminal stages of anoxemia (respiratory insufficiency).

RESPIRATORY NOISES

SNEEZING; It is initiated by stimulation of cilia of nasal mucosa and not a common condition in animals, but forceful expulsion of air through nostrils as a snort is commonly seen in rhinitis, foreign body in the nasal mucosa, allergy, or presence of larvae (Estrous ovis) in the nares.

WHEEZES (RHONCHUS, DRY RALES, STRIDORS); These are continuous, high pitched, whistling type sounds, primarily listen at expiration (also at inspiration). These are produced by vibration of airways when ever the upper air-passage becomes narrow with inflammation, e.g laryngitis.

CRACKLES (POPPING, BUBBLING, MOIST RALES, COARSE OR FINE CRACKLES, CREPITATION SOUNDS); These are discontinuous sizzling, clicking, popping, or bubbling type moist sounds mostly heard at inspiration, suggestive of the presence of swelling at the airways which are being forced to open. It may also be due to exudate and edematous secretions blocking in the airways, as seen in broncho-pneumonia or pulmonary edema.

PLEURITIC FRICTION (RUB) SOUNDS; It is a sandpaper or dry grating sounds, may be continuous or discontinuous type, produced by the rubbing of inflamed parietal and visceral pleura. The sound is loud, coarse, and usually is not influenced by dry coughing, as seen in pleuritis and pulmonary emphysema.

SNORE (SNORTING); It is a more deeper guttural sound originating from pharynx and larynx and is usually intermittent, seen in chronic obstructive lesion in the pharynx.

ROARING; It is a more continuous sound caused when air passed through a narrow larynx, as seen in laryngeal hemiplegia in horses.

GRUNT; It is a sound coming along expiration through a partially closed larynx (glottis) with sudden release of air and may be audible

at auscultation of the thorax and trachea with or without stethoscope. It is an evidence of pain anywhere in the body.

NB: An absence of lung sounds may be observed due to the presence of some medium between the lungs and the thoracic wall, e.g. space occupying masses, diaphragmatic hernia, large pulmonary abscess, or pleural effusion.

Common causes of respiratory noises (stridors) in animals are; Epiglottiditis, retropharyngeal abscess, strangles, guttural pouch infection, laxity of alar cartilage, laryngitis, pharyngitis, nose bots, actinobacillosis, anaphylaxis, trauma, foreign body, nasal polyps, pharyngeal paralysis, botulism, nasal trauma or foreign body, sinusitis, equine influenza, laryngeal trauma, granuloma, tracheal rupture and collapse, stenotic nares, purpura hemorrhagica, inhalation pneumonia, congestive heart failure, glanders, pasteurellosis, tracheitis, tracheal collapse, interstitial pneumonia, pulmonary embolism, and nasal schistosomiasis.

CYANOSIS; It is blue coloration of skin, conjunctiva, and visible mucous membrane, developing due to incomplete oxygenation of blood. It is usually seen in terminal stages of all forms of anoxia, except anemic anoxia, i.e. because of insufficient amount of hemoglobin.

In cyanosis, the oxygen saturation of hemoglobin is usually <80 per cent and is seen only if the hemoglobin concentration in blood is normal and not with severe anemia. At thumb pressure blue discoloration disappears due to temporary stoppage of blood. This condition may also be seen in congenital cardiac and greater vessels defects. In nitrite poisoning (methemoglobinemia) there is brown coloration of skin and mucosa.

Common causes of cyanosis in animals are; All types of pneumonias, methemoglobinemia, anaphylaxis, cardiac, septic, or hypovolemic shock, bloat, pulmonary edema, collapse, pneumothorax, smoke inhalation pneumonia, pulmonary neoplasia, multiple cardiac anomalies, post-parturient hemoglobinuria, and drowning

EXERCISE INTOLERANCE AND POOR PERFORMANCE; Exercise intolerance is either a suboptimum capacity to perform the expected level or a gradual/sudden decrease in the level of performance (exercise) which is previously attained by some individual. Sudden slowing or stopping while exercise, coughing, signs of respiratory distress precipitated by exercise, or excessive sweating (in horses) are some of the evidences of intolerance. It may be

associated with diseases of lungs, heart, musculoskeletal system, and debilitating systemic illnesses.

Respiratory causes of exercise intolerance in animals are; Obstruction of upper respiratory tract, roaring, epiglottic entrapment, guttural pouch infection, tracheitis, stenotic nares, nasal amyloidosis, nasal polyps, influenza, viral pneumonia, bacterial pneumonia, pleuropneumonia, pulmonary abscessation, interstitial pneumonia, and diaphragmatic hernia.

Cardiovascular causes of exercise intolerance in animals are; Atrial fibrillation, heart blocks, congestive cardiac failure, valvular defects, septal defects, endocarditis, pericarditis, myocarditis, congenital cardiac defects,

Musculoskeletal causes of exercise intolerance in animals are; Exertional rhabdomyolysis, degenerative joint disease, fracture, osteochondrosis, tendinitis, hoof imbalance, back problems, laminitis, and lameness.

Systemic causes of exercise intolerance in animals are; Febrile illness, anemia, fluid and electrolyte deficiencies, anhidrosis, heat stroke, dermatomycosis, hyperparathyroidism, liver diseases, diarrheal diseases, systemic neoplasia, obesity, poorly trained animals, plant/chemical poisoning, and debilitating diseases.

PRINCIPLES AND LINE OF TREATMENT OF RESPIRATORY TRACT DISEASES

A. Specific Treatment; Which includes the use of anti-bacterials, antifungals, and anthelminitics for bacterial, fungal, and parasitic causes, respectively.

The antibiotic selected should cover both Gram's positive and negative type bacteria and for either primary or secondary bacterial infection. The common antibacterial used in animals are oxytetracycline, chloramphenicol, kanamycin, lincomycin, gentamicin, erythromycin, penicillin-streptomycin, ampicillin, sulfadimidine, sulfadiazine-trimethoprim, etc. Amphotericin-B, thiabendazole, and griseofulvin are used for fungal pneumonia. Levamisole, benzimidazole group, and ivermectin are some commonly used drugs for respiratory tract parasites.

B. Symptomatic treatment; 1) Use of respiratory stimulants. Examples of (i) parenteral respiratory stimulants are, picrotoxin,

nikethamide, amphetamine sulfate, leptazol, and adrenaline. (ii) Inhalant respiratory stimulants, ammonium carbonate.

NB: The inhalant stimulants can improve respiration by causing reflex stimulation of respiratory as well as vasomotor centers, but has no effect when respiration is completely arrested.

ii) Oxygen therapy; About 40-60 per cent oxygen, @ dose of 4-8 liter/minute is usually recommended, if facility for oxygen therapy exists.

iii) Expectorants; These are used for reducing cough and expulsion of respiratory tract secretions. The expectorant drugs may be grouped into saline or sedative expectorants, stimulant expectorants, anodyne expectorants, and inhalant expectorants.

The saline expectorants decrease the viscosity of bronchial secretion that help their removal and are used in painful type cough where the secretions are tenacious (sticky). It include glycyrrhiza, ammonium chloride, potassium nitrate, ammonium carbonate, camphor, belladonna, potassium chlorate, etc. For chronic type of cough use of potassium iodide to increase the expulsion of secretions.

The stimulant expectorants are used where is soft, productive, and voluminous exudate exudes out. Examples include turpentine oil, eucalyptus oil, guaiacol, creoline, terebene, etc.

The anodyne (antitussive) expectorants are used in painful and exhaustive type of cough where bronchial secretions are absent. This type of cough interfere with normal activity of the individual and may cause damage to the lungs. Examples are codeine (methylmorphine), dihydrocodeine, chlorpheniramine maleate, benzonatate, belladonna, etc.

NB: Anodyne expectorants are not used for a long time for management of cough, except early use in painful bronchitis. Atropine sulfate is also contra-indicated as it increases the viscosity of bronchial secretions.

Inhalant expectorants (with steam inhalation) help to remove bronchial and nasal secretions and reduce congestion. These are commonly used in the form steam inhalation. The drugs are turpentine oil, Tr. benzoin-co, eucalyptus oil, terebene, and cresol.

Simple steam inhalation, for the removal of tenacious secretion and ease in breathing, is considered as good as the use of any saline expectorants.

2. Bronchodilators; These are used in cases of bronchial spasm (asthmatic syndrome) to improve ventilation and maintenance of oxygen exchange. Examples are atropine sulphate (particularly for pulmonary emphysema in horses), adrenaline, aminophylline, theophylline, silbephylline, ephedrine hydrochloride, etc.

3. Antihistaminics; Drugs such as mepyramine maleate, anthisan, antazoline, diphenhydramine maleate, etc. can be used in acute cases of pneumonia.

4. Steroids or non steroid anti-inflammatory drugs; These drugs are used in acute cases of inflammatory responses in the lungs and for the reduction of edema of the bronchioles. These may include cortisone, hydrocortisone, dexamethasone, prednisolone, phenylbutazone, and sodium salicylate.

5. Diuretics; These are indicated in cases of pulmonary congestion and edema, but may be harmful with other forms of pneumonia, as it may enhance stickiness of the secretions. Examples of parenteral diuretics are aminophylline, theophylline, furosemide, and mercurial.

C. Supportive therapy; This type of therapy only recommended if and as required. The use of isotonic solutions may be good to improve hydration status of an individual for adequate functioning of muco-ciliary clearance mechanism through oral and parenteral routes, but over hydration should be avoided particularly in acute cases of acute pneumonia, pulmonary congestion and edema. Multi-vitamins, minerals, and good high energy diet should be used in the convalescent period particularly following long standing illnesses. Provision of warmth on the chest and avoidance of direct wet and windy winter weather are necessary for pneumonic patients. A room with fresh air and appropriate humidity is required for animals suffering from respiratory diseases.

D. Enzymes; These have mucolytic effect in liquefying exudates and breakdown of fibrin proliferation and are useful particularly in cases of chronic pneumonia. Examples include trypsin and chymotrypsin with or without antibiotics.

RHINITIS

Syn: NASAL CATARRH, CORYZA, NASAL GLEET, RHINO-SINUSITIS

It is the inflammation of nasal mucosa, characterized clinically by periodic sneezing, noisy breathing, and nasal discharge.

ETIOLOGY

Rhinitis may develop either due to local infection in nares or in alliance with primary diseases of respiratory and other systems. Rhinitis may also be classified based on the lesion types as catarrhal, ulcerative, granulomatous, and proliferative rhinitis. Some of the diseases known to cause rhinitis are;

Bacterial diseases; The bacterial diseases characterized by rhinitis in animals are strangles (catarrhal rhinitis), glanders and melioidosis (ulcerative rhinitis) of horses; septicemic and pneumonic pasteurellosis (catarrhal rhinitis) of cattle, and mycoplasmosis in all animals.

Viral diseases; Viral diseases causing rhinitis are bovine malignant catarrh, mucosal disease complex, and rinderpest (causing erosive or ulcerative rhinitis). Infectious bovine (equine) rhinotracheitis, equine viral arteritis, equine influenza, African horse sickness, blue tongue (sheep), canine distemper, feline panleukopenia, infectious coryza (poultry) are characterized by catarrhal rhinitis. Contagious ecthyma and poxes lesions extending to the nasal cavity. Bovine nasal granuloma is characterized by the development of granulomatous rhinitis.

Fungal diseases; Epizootic lymphangitis causes ulcerative rhinitis, aspergillosis, and rhinosporidiosis may cause the development of proliferative rhinitis (polyps). This type of lesion may also be seen in cryptococcoses and histoplasmosis in small animals.

Parasitic diseases; Schistosoma nasalis (cattle), larvae of sheep nasal botfly, Oestrus ovis, Habronema sp. and other myiasis producing flies can cause local destructive and obstructive type lesions in the nasal cavity. Linguatula serrata is a parasite which infest the nasal passage of dogs.

Allergic rhinitis; Summer-snuffles is an allergic condition of cattle and sheep causing granulomatous rhinitis. Inhalation of irritant fumes (e.g. ammonia) or chemical dust may also cause the development of rhinitis.

Physical causes; Trauma to nasal cavity externally or while passing of stomach tube or probang, irritant vomitus materials (e.g. in choke), penetrate of spiny awns of grasses in the nostril(s) while grazing, and crossing of feed/water through cleft palate into nostrils are some examples.

Neoplasia; Squamous cell carcinomas, papillomatosis, fibrosarcomas, osteosarcomas, chondrosarcomas, and benign polyps, have been reported to cause proliferative rhinitis in animals.

PATHOGENESIS

Rhinitis may be primary or secondary to diseases of respiratory system. The inflammatory response developing in the nostrils following infection varies with the etiology. It may vary from simple erythema to catarrhal, erosive, ulcerative, granulomatous, proliferative, or necrotic type lesions. The inflammation in the nasal mucosa will cause the sneezing and exudation. Acute bilateral inflammation and exudation may cause obstruction in the air-passage leading to dyspnea and noisy breathing. The infection may extend further to sinuses, resulting in rhino-sinusitis.

CLINICAL SIGNS

The early clinical signs include periodic sneezing of varying intensity, dyspnea and wheezing both at inspiration and expiration, and nasal discharge of varying consistency. Shaking of the head and rubbing of nose against posts in effort to reduce irritation may be noted in some animals. Nasal discharge may be unilateral or bilateral, which initially be of serous but later may become mucoid, purulent, or sero-sanguineous type, depending upon the etiology.

Nasal lesions, form simple erythema to the development of erosion, ulceration, granulomas, polyps, or degeneration of the turbinate bones may be inspected. Signs of systemic involvement may also be demonstrated in some diseases mentioned in the etiology.

CLINICAL PATHOLOGY

Nasal swabs should be taken for the culture of causative bacteria or fungi, cytological examination of neoplastic growth, and confirmation of parasitic ova. Complete blood count may be helpful to identify the nature and severity of cellular reaction. X-rays examination and rhinoscopy may be useful for the identification and demarkation of intranasal neoplastic growths and traumatic lesions. Specific serologic tests may be required to identify the viral diseases.

DIFFERENTIAL DIAGNOSIS

Fascial sinusitis and guttural pouch infections in horses need rule-out from rhinitis. Both the conditions are characterized by persistent, purulent, often unilateral nasal discharge without the presence of sneezing or lesions in the nasal cavity. Pharyngitis and tracheo-bronchitis are associated with bilateral nasal discharge and cough without sneezing and nasal lesions. Epistaxis is mostly associated with known history of trauma and characterized by passage of frank blood from the nostrils. Choke is characterized by complete inability to swallow and greenish nasal discharge due to regurgitated feed.

TREATMENT

Specific treatment include the use of broad spectrum antibiotics, anthelmintics, or antihistaminics, according to the etiology.

Physical removal of the sticky exudates from nostrils by flushing with normal saline may benefits to relief dyspnea. In addition, application or washing with non irritant antiseptics followed by coating with antiseptic creams or ointments have beneficial effects.

Steam inhalation alone or in combination with decongestants (Eucalyptus oil, Turpentine oil, Tr. benzoin co., or Camphor) may be of great value in clearing the air-passage. Chlorbutol (1 per cent in liquid paraffin) may be used as nasal drops for the same purpose. Humidification with vaporizer or nebulizer improve clearing of nasal secretions.

4. Anti-inflammatory and antihistaminic drugs using parenterally can be of great value in acute rhinitis.

5. Trephining or surgical removal of proliferative type lesions of nasal cavity should be attempted if possible and required.

Recipe for Sheep Nasal Fly; (i) Lysol (3 per cent in water) to be used as nasal douche, and (ii) liquid paraffin and carbon disulfide, in equal parts, or tetrachloroethane, @ dose of 3 ml per nostril (instillation).

EPISTAXIS
Syn: NOSE BLEED, HEMOPTYSIS

Epistaxis is the spontaneous hemorrhage caused by trauma originating from the nostrils. It could be unilateral or bilateral. The later may originate either from nasopharynx or areas below (guttural pouches, lungs) or secondary to systemic diseases. The volume may be as small flecks mixed with nasal discharge to a large volume of pure blood flowing rapidly form nostrils and mouth.

Hemoptysis is the hemorrhage mixed exudate (sanguinous sputum) originating from the lungs.

ETIOLOGY

This is common condition of horses and cattle. The possible causes of epistaxis are listed below;

Physical trauma by blow or by passing of probang or gastric tube, ulcerative tumor or polyps, foreign bodies damage at the nasal, trauma to pharyngeal regions and skull, dehorning injuries, and gunshot wounds of respiratory tract are some of the causes of nose bleed.

Specific disease associated with nose bleed are glanders, aspergillosis, pasteurellosis, equine infectious anemia, blue- tongue disease, bovine virus diarrhea, malignant catarrhal fever, anthrax, bacillary hemoglobinuria, sheep nasal bots, and many granulomas of fungal origin.

Non specific disease causing nose bleed are guttural pouch mycosis, purpura hemorrhagica, thrombocytopenia, exercise induce pulmonary hemorrhage, hepatitis associated clotting defects, acute renal diseases, congestive heart failure, acute renal failure, endocarditis, and disseminated intravascular coagulopathies.

Snake bites, poisoning by certain plants and chemicals may also cause hemorrhage from nostrils.

CLINICAL FINDINGS

Hemorrhage of varying degree may occur either from one or both nostrils. Passing of perfuse and pure blood from one nostril is usually an indication of lesion in the nasal cavity. Hemorrhage originating form lower respiratory tract is usually bilateral, of foamy texture, and may be mixed with serous or muco-purulent exudate. Severe hemorrhage may cause circulatory disturbances and even death. Except in animal with clotting defects the blood will not clot, otherwise, clotted blood may be seen at the external nostrils.

CLINICAL PATHOLOGY

Bleeding tendency, complete blood count, culture, and cytological examination may be necessary to find the underlying defects and cause. Radiograph examination may be necessary to diagnose the fractured bone or space occupying lesions (tumors, polyps) in the nostrils.

DIFFERENTIAL DIAGNOSIS

Epistaxis can be differentiated from hemoptysis by the physical examination of the exudate and blood. History, clinical examination, and laboratory findings may be necessary if the primary lesion is also present other than the nostrils.

TREATMENT

Specific treatment depends upon the nature and location of the cause. Space occupying lesions require their surgical removal. Hemorrhage from the guttural pouches need direct ligation of the bleeding vessels.

Symptomatic treatment should include the following steps.

a). Complete rest and no exercise of any kind when epistaxis.

b). Cold water immersion or ice packs in and on the nostrils (both nostrils should not be packed at one time).

c). Local use of vasoconstrictor drugs, e.g. Adrenaline with xylocain local spray (Adrenaline should never be used by parenteral routes in cases of epistaxis).

d). Use of local coagulants (styptics) if bleeding spot is located. For this purpose one can use Alum (10 per cent), Tr. ferric per chloride, or Tr. benzoin-co.

e). Parenteral use of coagulants/hemostatic preparations that enhance coagulation process. Examples, conjugated estrogen, adrenochrome isonicotinic acid, carbazochrome salicylate, oxalic and malonic acids, and vitamin-K.

In cases of systemic hemorrhage, one can also use parenteral calcium borogluconate and diuretics (furosemide).

In order to compensate the acute and severe loss of blood, blood transfusion may be advised (see hemorrhage for blood transfusion).

In cases of chronic (mild) and repeated episodes of epistaxis, hematinics (Ferrous sulfate or chloride) and vitamin B complex may be used.

(Aspirin and live virus vaccine should not be used if known history of coagulation defects in cases of nose bleed).

LARYNGITIS, TRACHEITIS, AND BRONCHITIS

Inflammation of the upper respiratory tract (larynx, trachea, and bronchi), characterized clinically by cough, dyspnea, and noisy breathing.

ETIOLOGY

In cattle the common causes of upper respiratory tract (URT) inflammation are infectious bovine rhinotracheitis, malignant catarrhal fever, calf diphtheria, haemophillosis, pasteurellosis, tuberculosis, actinobacillosis, mycoplasmosis, rhinosporidiosis, and lung-worm infestation.

In sheep, infections with Corynebacterium pyogenes, Spherophorus necrophorus, Mycoplasma ovipneumoniae and lung-worm (Dictyocaulus spp) infestation are common causes.

Horses suffer with URT infection with equine viral rhino-pneumonitis, equine viral arteritis, infectious bronchitis, equine influenza, strangles, glanders, and lungworm (Dictyocaulus arnifieldi).

In dogs, canine distemper and Bordetella bronchiseptica (kennel cough) infection are common causes of URT.

PATHOGENESIS

The inflammatory response in URT varies with the types of infection. Varying degree cough and exudation are common outcome of URT inflammation. Profuse and thick exudate may obstruct the air passages. Signs of systemic involvement will depend upon the nature of the causative agent(s) and response of the hosts.

CLINICAL SIGNS

Presence of cough, dyspnea, nasal discharge, wheezes are common signs of URT infection. In acute diseases, the cough will be painful, dry, and non productive (but later may become moist and productive). The nasal discharge will be frothy and serous to mucoid initially, which later may become sticky and purulent. Occasionally, the discharge may contain flacks of fibrin and blood. Respiratory dyspnea with loud wheezes (audible at the trachea and base of the lungs) are common in acute inflammation.

In parasitic bronchitis the cough is usually dry and non- productive. Evidence of toxemia including anorexia, fever, and depression, and other signs of specific diseases could also be noted.

CLINICAL PATHOLOGY

Sputum or nasal swabs may be examined for the identification of causative bacteria, parasite, or fungi. Complete blood count may reveals the type and severity of cellular response. Serological tests may be of value for identification of viral diseases. Presence of larvae in the faeces is the confirmation of lung-worm infestation.

NECROPSY FINDINGS

Postmortem lesions of URT disease varies with type and severity of infection. In most bacterial infections a diphtheritic (pseudo) membrane may be present with exudate. Uncomplicated viral infection may cause the accumulation of sero-mucoid foamy exudate filling the URT.

DIFFERENTIAL DIAGNOSIS

Pneumonia and pulmonary edema are characterized by dyspnea at each respiration and presence of crackles originating from the lower lungs. Pulmonary emphysema is characterized by expiratory dyspnea and poor exercise tolerance.

TREATMENT

The line of treatment of URT infection should include;

1. Specific therapy by using specific antibacterials or anthelmintics, according to the nature of etiology.

2. Symptomatic therapy include the use of; (a) Oral expectorants for reducing cough and exudation, (b) Analgesics (if cough is painful and distressing), (c) Inhalant expectorant or simple steam inhalation may be used to clear the respiratory passage, (d) Antipyretic may be advised if fever, and (e) Corticosteroids and antihistaminics may be used for reducing acute inflammatory response.

3. In chronic cases, the use of mucolytic agents (e.g. Bisolvon) can be of help in liquefying the thick exudate and breaking fibrin deposits.

4. Multi-vitamins and minerals or fluid therapy may be advised if and as required.

5. In severe dyspnea due to blockage of upper air passage with exudate and inflammation, tracheotomy may be performed.

NB: Avoid cold and dusty atmosphere. Liquid drugs or feeding in form of drench should be avoided, as it may cause further complication in case of faulty administration.

CONGESTION AND PULMONARY EDEMA

Pulmonary congestion is an increased amount of blood in pulmonary vascular bed. Pulmonary edema is the collection of intravascular fluid into the pulmonary parenchyma and alveoli. Both of these conditions are associated with respiratory abnormalities.

ETIOLOGY

Pulmonary congestion could be either primary (if basic lesions are in the lungs) or secondary to diseases of other organs than lungs. The primary pulmonary congestion is commonly seen in early stages of pneumonia (of infectious, inhalation, or aspiration types), anaphylaxis, and even with prolonged lateral recumbency. The secondary pulmonary congestion may be seen in congestive heart failure (particularly left-sided) and many septicaemic conditions (e.g. acute mastitis and metritis).

Pulmonary edema usually develops following long standing cases of pulmonary congestion. The specific diseases causing pulmonary edema are African horse sickness, infectious canine hepatitis, bovine malignant edema, bovine pleuropneumonia, poisoning by gossypol or organophosphorus compounds, acute renal failure, and local lymphatic obstruction. Pulmonary edema can also develop with over dosing of IV fluid therapy in acute pneumonia.

PATHOGENESIS

In pulmonary congestion, there is engorgement of capillary vascular bed with blood and so the effective air space is lost. Because of reduction in oxygen diffusion of blood, there is development of anoxic anoxia and reduction of vital capacity. The intensity of clinical signs will dependent upon the severity of anoxic anoxia.

In pulmonary edema, because of increase hydrostatic pressure within pulmonary capillaries or anoxic or toxic damage to capillary endothelium, intravascular fluid escape into peribronchial spaces and pulmonary parenchyma. In advanced cases there may be filling of alveoli, interstitial tissues, bronchioles, and bronchi with transudate, and so there may be interference with normal gaseous exchange. The aftermaths are hypoxia, hypercapnia, acidosis, and other respiratory abnormalities. Pulmonary edema may also develop in local lymphatic obstruction or as a consequence of generalized edema associated with hypoproteinemia.

CLINICAL FINDINGS

The respiratory signs including tachypnea and dyspnea, both at inspiration and expiration are common with both pulmonary congestion and edema. In pulmonary congestion, there may be no cough and exudation and auscultation of lungs may reveal harsh vesicular murmurs without crackles or wheezes. In pulmonary edema, crackles (fluid rales, particularly at depending parts of the lungs), soft productive cough, and copious frothy mucoid type nasal discharge (sometime blood tinged) are commonly noted. Signs of cyanosis and exercise intolerance may also be evident in later stages of severe pulmonary edema.

Other signs like restlessness, difficulties in sitting and standing, tachycardia, and elbow abduction could also be noted in severe cases of pulmonary congestion and edema.

CLINICAL PATHOLOGY

Nasal swab and complete blood count needed to be carried out for identification of the causative organisms and cellular response, respectively. X-rays examination may be useful in demarkation the areas of increased density due to congestion and edema.

NECROPSY FINDINGS

Death may occur due to asphyxial respiratory failure particularly in severe pulmonary edema. In pulmonary congestion the lungs are darker in color and excessive venous blood may exudes out at cut. In pulmonary edema the lungs are pale, heavy, doughy in consistency, do not collapse, and excessive quantity of transudate may exude out from the cut areas.

DIFFERENTIAL DIAGNOSIS

Pulmonary congestion and edema need to be differentiated from early cases of acute pneumonia, anaphylaxis, congestive heart failure, and other causes of respiratory dyspnea and generalized edema (see specific topics).

TREATMENT

The line of treatment of pulmonary congestion and edema is mostly directed toward the primary cause (if known). For example, in case of anaphylaxis, use of adrenaline and corticosteroids (to maintain vascular integrity) or antihistaminics are recommended. Drugs like phenylbutazone, aspirin, and salicylic acid (@ dose of 100 mg/kg, PO, b.i.d), may also be used for the same purpose. (see specific diseases causing edema).

Symptomatic treatment mainly aimed at reversing hypoxemia, decrease plasma volume and left atrial pressure, increase colloid osmotic pressure, and removal of transudate from the lungs.

a). Use of bronchodilators reduce the bronchospasm and reversing hypoxemia. Examples are, theophylline or aminophylline, @ dose of 5: 10 ml/adult dog, IV.

b). Diuretics, i.e. furosemide, @ dose of 1-2 mg/kg, IV or IM, may be repeated at 2 hours intervals, may also be used for reducing pulmonary edema.

c). Venesection, @ dose of 4 ml of blood/kg body weight (10 per cent of total volume) at a time, may be adopted in severe cases of pulmonary congestion.

d). Oxygen therapy may be necessary if acute signs of anoxic anoxia are evident.

e). Respiratory stimulants, such as nikethamide or ammonium carbonate (inhalation) in cases of respiratory depression.

f). Saline and/or stimulant type expectorants are necessary for the removal of copious exudate from the trachea/lungs. For this purpose ammonium chloride or carbonate, potassium nitras, eucalyptus oil, terebene, camphor, or benzoin may be used in the form of electuary.

g). Colloid solution as Dextran may be used to increase osmotic pressure and so reduction of edema.

3. Supportive treatment if and as desired.

4. Treatment of complications if any present or expected.

Antibiotics should be used for the treatment of primary or secondary bacterial invaders. This is particularly important because of the common observations that the edema of lung can impair pulmonary bacterial defense mechanisms, thus increasing the chances of infection.

NB: Complete rest and no exercise of any kind will help to recover from stages of severe pulmonary distress. Clean and dry atmosphere and dust free palatable fodder can make a considerable difference in early recovery.

Do not use excessively the IV fluids therapy particularly in pulmonary edema. Excessive use of IV fluid may add to the severity of the pulmonary congestion and edema which is not desirable anyway.

PNEUMONIA
Syn: PNEUMONITIS

Pneumonia is an inflammation of pulmonary parenchyma, characterized clinically by the presence of cough, nasal discharge, abnormal respiration and lung sounds, and toxaemia.

Pneumonia can be classified, based on the etiology or lesion types as;

Broncho-pneumonia (irritant migrating down the bronchial tree),
Interstitial pneumonia (inflammation of alveolar wall),
Fibrinous pneumonia (excessive fibrin deposits on the pleura),
Catarrhal pneumonia (excessive mucous in the airways),
Granulomatous pneumonia (discrete granulomas in lung),
Gangrenous pneumonia (devitalization and necrosis of lung),
Hypostatic pneumonia (prolonged congestion and edema of lung),
Embolic pneumonia (metastasis emboli in the lung),
Verminous pneumonia (presence of parasites in the lung),
Pneumoconiosis (pneumonia due to inhaled mineral contamination),
Aspiration/ inspiration or drenching pneumonia (pneumonia caused by aspiration of chemicals, drugs, or vomitus).

ETIOLOGY

The etiology of pneumonia may includes viruses, bacteria, fungi, parasites, physical, or chemical agents. Infections may enter the body either through lungs (bronchogenic) or via blood (hematogenic). Most of the infectious agents can cause the disease alone, in association with other infections, or in alliance of the predisposing factors. The predisposing factors commonly associated with pneumonia include inclement weather (severe winter and summer), various stresses, closed confinements, inadequate ventilation, poor hygiene, malnutrition, and many debilitating diseases. The common diseases causing pneumonia in animals are;

Virus (Interstitial pneumonia): Virus pneumonia of calves and lambs, parainfluenza virus-3, adenovirus 1, 2, and 3; rhinovirus, reovirus, enzootic pneumonia, and bovine herpes virus of cattle. Progressive interstitial pneumonia (Maedi), and pulmonary adenomatosis (Jaagsiekte) of sheep, equine influenza, equine viral rhinopneumonitis, infectious equine pneumonia, canine distemper, infectious tracheobronchitis of dogs are some of the examples.

Bacteria (Broncho-pneumonia); Examples includes contagious bovine (ovine, caprine) pleuropneumonia, hemorrhagic septicaemia, shipping fever, glanders, strangles. Pneumonia can also be caused by infection with Corynebacterium pyogenese (calves), Hemophilus somnus, Streptococcus and Staphylococcus spp, Mycobacterium bovis (cattle), Streptococcus zooepidermicus, Corynebacterium equuli (foals), and Bordetella bronchiseptica (dogs).

Fungi; Common diseases of fungal origin causing pneumonia in animals are aspergillosis, blastomycosis, histoplasmosis, nocardiosis, and cryptococcus.

Metazoan Parasites (Verminous pneumonia); Pneumonia can be caused by infestation with Dictyocaulus viviparous and D. filaria in cattle, sheep, and goats, and D. arnifieldi in horses and donkeys. Parasitic pneumonia may also develop by migrating larvae of Ascaris lumbricoides, Filaroides osleri, Paragonimus spp. and lung-flukes (dog).

Physical or chemical agents; Pneumonia may also be caused by physical or irritant chemicals through faulty drenching, aspiration of vomitus, and inhalation of toxic gases or fumes. Pneumonia caused by faulty drenching or inhalation is called as aspiration pneumonia, drenching pneumonia, or Inhalation pneumonia. Pneumonia can also occur due to aspiration of pus from rupture of pharyngeal abscess. A common complication of paralysis or obstruction of larynx, pharynx, or esophagus is the inspiration pneumonia and is due to aspiration of water or feed into lungs.

PATHOGENESIS

Pulmonary defence plays important role in preventing the entry and neutralization of the infectious agents entering through lungs. Pneumonia will occur if the pulmonary defence is compromised or overwhelmed by the disease producing agent(s).

Most of the bacteria enter the lungs through inhalation and cause initially bronchitis and bronchiolitis followed by spread of infection to the adjacent parenchyma. The tissue reaction in the form of lesions alters with the causative agent It may be in the form of simple congestion to catarrhal, fibrinous (H.S.), necrosis (necrobacillosis), granulomatous, caseous type lesions (mycoplasma and fungal infections), or consolidation. The spread of infection to adjacent structures occurs either by extension, bronchioles, or lymphatics. This spread of infection is facilitated by lung movement and acute coughing.

Viruses are introduced in the body chiefly through inhalation and initially cause bronchiolitis followed by spread to the lung parenchyma causing enlargement and proliferation of alveolar epithelium, alveolar edema, and thickening of interstitial tissues without much tissue reaction, necrosis, or toxemia (except with secondary bacterial infection).

Anoxia and hypercapnia are common outcome of pneumonia which are due to interference of normal gaseous exchange between alveolar air and blood. The abnormal lung sounds of varying intensity and nature, exudation in the airways, and toxemia are common in most bacterial pneumonia.

CLINICAL FINDINGS

The primary signs of pneumonia are respiratory abnormalities, cough, nasal discharge, abnormal lung sounds, and evidences of toxemia. The respiratory abnormalities are indicated in the form of fast and shallow breathing in early pneumonia, dyspnea both at inspiration and expiration, in latter stages of most pneumonias. Cough of various types and intensity is an important sign of inflammatory lesion of lungs. In most bacterial pneumonia, the cough is moist, painful, and productive type, whereas in viral infection, it is usually dry, paroxysmal, and non productive type. Cough can be induced (voluntarily) by pressing the trachea or pharynx.

In most bacterial pneumonia the nasal discharge is muco-purulent and some times hemorrhagic, but with most viral infections it is of sero-mucous type (which may become purulent if secondary bacterial infection). Presence of ozena (decay or putrid odor) is an indication of necrosis, gangrene, or anaerobic bacterial infection.

Auscultation of lungs is an important aid for the diagnosis of affected parts. The respiratory sounds varies from simple vesicular murmurs (in early cases of bacterial and viral infections), to wheezes or crackles both at inspiration and expiration, particularly at the depending parts of lungs and in later stages. Pleuritic rubbing (friction) sounds may be auscultated in cases of pleuritis.

Additional signs include, fever (particularly in bacterial pneumonia), exercise intolerance, anorexia, depression, weight loss, tachycardia, constant standing, and cyanosis (in terminal stages) may also be present in pneumonia.

The chronic type of pneumonia is usually characterized by chronic moderate cough, mild toxemia, emaciation, and gaunt appearance.

Nasal discharge if present is usually of thick purulent type. Auscultation may reveal areas of increased bronchial tones particularly at ventral half or absence of any sound due to consolidation of the lung.

CLINICAL PATHOLOGY

Nasal or tracheal swabs are usually taken for Gram's staining or culture of bacterial for their identification and antibiogram analysis. Hematological examination may be important to identify the type and severity of cellular response. Serological tests are useful aid for the diagnosis of viral diseases. X-rays examination may be helpful to denote areas of consolidation or abscessation. An increase opacity is indicative of areas of lost normal aeration space.

NECROPSY FINDINGS

Gross lesions varies from mere congestion to consolidation (hepatization) of various parts of lung. In most bacterial diseases lesions are mainly seen at the anterior lobes and at depending parts of middle and caudal lobes, and a sero-fibrinous to purulent exudate in the bronchioles. In viral pneumonia the lesions may involve whole lung in the form of general areas of congestion and granular appearance.

Histological examination in viral pneumonia may reveal enlarged alveoli, thickening of interstitial tissues, peribronchiolar and perivascular lymphocytic cuffing. Fibrinous exudation, fibrosis, and severe and generalized cellular reaction is seen in most bacterial pneumonia. Lymphoid hyperplasia, hyperplasia of smooth muscles around bronchioles, and diffuse lymphocytic infiltration are suggestive of chronic pneumonia (e.g Mycoplasma or fungal infection).

DIFFERENTIAL DIAGNOSIS

Pneumonia needs to be ruled-out from the followings diseases.
Congestive heart failure, hyperthermia, acidosis, pulmonary congestion and edema, pleurisy, hydrothorax, pneumothorax, anaphylaxis, histotoxic anoxia (cyanide poisoning), and anemia.

TREATMENT

1. Specific treatment according to the nature of cause, e.g. antibiotics, antifungals, antiparasitics, or antihistaminics. Vigorous use of broad spectrum antibiotics are usually recommended in (mixed) bacterial (or viral) infection, parenterally, for at-least 7 days or till recovery.. Hyper-immune (specific) antiserum is used for specific viral pneumonia.

2. Symptomatic treatment include; a) Steam inhalation (inhalant expectorant) for the reduction of congestion and viscosity of secretions present in the air-passages. Eucalyptus oil, turpentine oil, Tr. benzoin co, camphor, or menthol can be used as decongestants along with steam inhalation.

b) In winter, warm application in the form of heat pads or blankets or message with turpentine/camphor liniments on the chest may reduce adverse cold effects and reduce pain.

c) Respiratory stimulants (e.g. nikethamide) or oxygen therapy in young animals may be required if signs of anoxic anoxia and cyanosis are evident.

d) Expectorant, mucolytic, and antitussive drugs if and as required. For example, sedative and anodyne expectorants (containing morphine, codeine) are used in cases of acute, non- productive, and painful type cough. Saline expectorants (containing ammonium chloride or carbonate, potassium or sodium nitrate, sodium or potassium iodide) are used in cases of soft and productive cough. Mucolytic expectorants (acetylcysteine 10-20 per cent solution administered via aerosol, bromhexine @ dose of 1 mg/kg, PO, in dogs and cats) may be used for reducing viscosity of the secretions. This type of expectorants have irritant effects and so should not be used in acute and painful type cough. Proteolytic enzymes e.g. streptokinase, streptodornase, or trypsin can be used to liquify bronchial secretions and for deep penetration of antibacterials particularly in chronic cough.

Recipe for saline electuary (Expectorant) used for animals especially horses. The ingredients are ammonium chloride (8-12 gm), potassium nitrate (8-12 gm), sodium citrate (8-12 gm), pulv glycyrrhiza (8-12 gm), pulv linseed (8-12 gm), pulv aniseed (8-12 gm), and molasses (QS to make electuary).

Potassium iodide, @ dose of 10-20 gm/LA, PO, can be added in electuary in cases of chronic cough).

e. The use of acetylsalicylic acid (aspirin), sodium salicylate, phenylbutazone, or ibuprofen is better than steroid therapy in reducing fever and acute inflammation. (Sod. salicylate and aspirin @ dose of 1 gm/10 kg, 12 hourly, are only the non-steroid anti-inflammatory drugs which are quite safe to use in cattle). Diphinhydramine HCl may also be used in cases of acute cough.

f. Corticosteroids are indicated in cases of severe pneumonia for reducing acute inflammatory response but should always be accompanied with broad spectrum antibiotics.

g. Bronchodilators (aminophylline or theophylline, @ dose of 1-3 mg/kg, b.i.d. in horses and ephedrine @ dose of 1-2 mg/kg, b.i.d. in dogs) may be used for the dilation of bronchioles (easy breathing) and increase ciliary beats.

3. Supportive therapy and good nursing care as and if required. This includes the provision of well ventilated, clean, and dry rooms. Avoidance of the sick animals from rain, direct cold wind or hot sun. Good palatable and dust free feed, luke warm water to drink should be used during the course of the acute disease.

During the convalescent period prescriptions containing multivitamins and minerals (oral or parenteral) may be required particularly in long standing cases of pneumonia and following optimum success of antibiotic treatment.

4. Treatment of complications as and if required.

NB: Levamisole an anthelmintic for nematodes has also been used in attempts to stimulate the bovine immune system with equivocal success but not as supportive treatment.

PULMONARY EMPHYSEMA

Syn: a. (ALVEOLAR EMPHYSEMA) BROKEN WIND, HEAVES, OR CHRONIC OBSTRUCTIVE PULMONARY DISEASE OF HORSES.

Syn: b. (INTERSTITIAL EMPHYSEMA) BOVINE ASTHMA, OR PANTING SYNDROME OF CATTLE.

Pulmonary emphysema is an abnormal distension of pulmonary parenchyma (alveolar duct, alveoli, or interstitial tissues) with air. Clinically, it is characterized by expiratory dyspnea, exercise intolerance, weak, chronic cough sporadically, and unthriftiness.

ETIOLOGY

Alveolar emphysema is a chronic condition which is seen absolutely in horses, whereas, interstitial emphysema is usually acute and seen in cattle. The exact cause of pulmonary emphysema is not well understood. This condition has been recorded as a quandary to chronic atypical pneumonia, acute bronchitis, aspiration pneumonia

pneumonia, mycotic pneumonia, allergic and anaphylactic reactions, pulmonary edema, inhalation pneumonia due to irritant fumes or gases, pasteurellosis (pneumonic type), pulmonary abscesses, and septicemia due to other causes.

The commonly observed predisposing factors associated with the disease are prolonged feeding on low grade roughage, long-term feeding in indoor dirty barns during winter, mouldy or dusty feed, sudden and over-exertion in animals not accustomed to exercise.

PATHOGENESIS

There are various hypothesis for the pathogenesis of pulmonary emphysema. One proposes that there is primary deficiency in strength of supporting tissues of lung which are unable to coop with emergencies such as coughing or over-exertion. Hence so, they become over-stretched and remain dilated even after expiration. Second suggests that due to inflammatory conditions as mentioned in the etiology, there is obstruction in air passages, but air still can enter the alveoli through the communications (canals of Lambert's and pores of Kahn's). So if the air becomes entrapped within the alveolar spaces that can cause over-distension and/or rupture of alveoli under increased air pressure of coughing or over-exertion so the air will escape into interstitial tissues. The over-distension of connective tissues with air can cause compression of alveoli. A current hypothesis suggests an excessive release of proteolytic enzymes by inflamed cells, which cause breakdown of fibrin (in disease state) but can also cause damage to normal connective tissue fibers and so there may be remodeling of the airspaces.

In either of the case, there is air retention (incomplete evacuation of lungs) at each expiration and failure of normal gaseous exchange. There is increase in residual volume, and to maintain the normal gaseous exchange as compensation, there is increase in tidal volume. The increased retention of carbon dioxide will cause an increase in depth of respiration and acidosis. In addition, because of pressure of trapped air over the surround parenchyma, there is decrease in capillary bed and increase intrathoracic blood pressure which can leads to myocardial hypertrophy (of right ventricle) and in later stages, congestive heart failure. The anoxia, if severe, may result in depression of general body metabolism and functions.

Incomplete evacuation of lungs as result of pulmonary emphysema will lead to a continuous and increased efforts of diaphragm and external abdominal obliques muscles to expel the air from lungs. This will result in hypertrophy of those muscles. The hypertrophied muscles may be visible externally and is called as "Heaves line".

Occasionally, the entrapped air in the interstitial tissues may find an escape through pleura into the mediastinum and via the fascial planes to beneath the skin of the back to cause the subcutaneous emphysema.

CLINICAL FINDINGS

The clinical signs in interstitial emphysema are usually acute (in cattle) and can be observed even at rest, where as of alveolar emphysema (in horses) are chronic in nature and are mostly observed following exercise. These include;

1. Expiratory dyspnea is a cardinal sign of pulmonary emphysema and usually associated with double uplift of abdominal wall at each expiration. In time the shape of chest becomes barrel-like and heaves line on either side is distinctly visible.

2. Cough is short, weak, and more pronounced and wheezing following exercise. Also the cough is common during cold temperature, at excitement or dusty environment.

3. Nasal discharge if present will be intermittent, sero-mucous to purulent, or mixed with blood. Occasionally epistaxis may develop following severe exercise, particularly in horses.

4. Auscultation of lungs may reveals wheezes which may accompanied by pleuritic friction-rub sounds during the early stages of the disease. In later stages crackles may be observed due to exudation. Heart sounds are usually muffled due to expansion of lung over the cardiac areas.

5. Other signs include severe exercise intolerance, generalized edema and other signs of congestive heart failure, and emphysematous swelling at the upper cranial half may also be noted. There may be no evidence of toxemia or fever.

CLINICAL PATHOLOGY

There is no typical hematological changes of pulmonary emphysema, however, neutrophilia or eosinophilia may be indicative of primary bacterial bronchitis or allergy (or parasitic bronchitis), respectively. An increase in alkaline reserve due to carbon dioxide retention and compensatory polycythemia due to anoxic anoxia may be present in advanced stages of the disease.

NECROPSY FINDINGS

Lungs appears pale, enlarged, bears ribs imprints, and have thick and rounded margins at macroscopic examination. Histologically, a

distended interlobular septa and collapsed alveoli are indicative of interstitial emphysema. Large distended empty spaces (multiple alveoli) with common opening due to rupture of alveolar septa may be seen in alveolar emphysema. Cardiac enlargement and other evidences of congestive heart failure may also be present.

DIFFERENTIAL DIAGNOSIS

Pulmonary emphysema both horses and cattle need differentiation from primary 1) pulmonary congestion and edema, 2) anaphylaxis, 3) pneumonia of different etiology, and 4) pneumothorax.

TREATMENT

Complete recovery from pulmonary emphysema is difficult. The treatment is mainly directed toward reducing the severity of the disease and keeping the animals alive only for meat (cattle) or breeding (horse) purposes. The lines of treatment should include;

1. Complete rest (no exercise or if demanded for only a short period), a dust-free feed and allergen-free atmosphere should be provided throughout the rest of life.

2. Treatment of the primary disease if known thoroughly till complete recovery (see etiology).

3. Symptomatic, supportive, and treatment of complication if and as required. Good nutritive diet, multi-vitamins and minerals should be used to improve health of the animals. Injection of arsenic salt (Aricyl, mericyl, @ dose of 10-20 ml/LA, IM, on alternative days) is in common use by large animal practitioners.

4. Corticosteroids, antihistaminics, atropine sulphate, epinephrine, acetylsalicylic acid, and/or diuretics (in cattle particularly) in early stages of disease are supposed to be of palliative value.

5. Anodyne and saline expectorants may be recommended for suppressing cough and removal of bronchial secretions.

6. Antibacterials should be used for treatment of primary/ secondary bacterial infection, if evident.

Prognosis: Unfavorable (particularly in advanced cases).

PULMONARY ABSCESS
Syn: SUPPURATIVE PNEUMONIA

It is the development of single or multiple abscesses in lungs, characterized clinically by signs of chronic toxemia, short chronic cough, and progressive emaciation.

ETIOLOGY

Pulmonary abscesses develops either from inhalation of septic emboli (e.g. Strangles or guttural pouch infection) or metastasis of embolic foci originating from suppurative process elsewhere in the body (e.g. endocarditis, myocarditis, metritis, mastitis, navel ill, actinobacillosis, or systemic septicemia). Pulmonary abscesses may also develop as a sequelae to primary pulmonary infections, e.g. broncho-pneumonia, contagious bovine (ovine, or caprine) pleuropneumonia, mycotic pneumonia (Aspergillosis), aspiration pneumonia, or traumatic reticulitis. Abscess in the lungs can also be seen as an independent disease of lung (tuberculosis).

PATHOGENESIS

Small sized single abscess in the lung may remain un-noticed during the life of an animal. If an abscess reaches a tremendous size or there is development of multiple abscesses, the clinical signs will develop. The clinical signs are mainly because of toxemia rather than respiratory interference. However, a large sized abscess in the lung may cause respiratory abnormalities due to obliteration of air-passages. Tachypnea may be observed because of the stimulation of stretch receptors in the alveoli rather than anoxia. Occasionally, erosive lesions involving blood vessels may result in hemoptysis. Encapsulated abscesses may remain dormant for rest of the life. Its rupture may cause extension of infection to adjacent areas or to other organs and thus may ends up with acute suppuration in those organs, e.g., suppurative bronchopneumonia, pleurisy, or pulmonary emphysema.

CLINICAL FINDINGS

The signs of mild toxemia, including anorexia, depression, emaciation, decrease production, moderate fever, and chronic, short, painless cough are common if multiple lung abscesses. Occasionally, hemoptysis and purulent nasal discharge with fetid odor may be present. Auscultation of chest cavity may reveals audible crackles (at the periphery of a large abscess). Affected animals remain emaciated and discarded. Signs of acute broncho-pneumonia with severe respiratory involvement may reappear suddenly following rupture of an abscess, leading to death of the individual.

CLINICAL PATHOLOGY

Nasal, tracheal, or bronchial swabs are needed for the isolation of the causative organisms and their antibiotic sensitivity. Radiograph may be help for detection of any large sized abscess. Presence of a mild leukocytosis (neutrophilia) without shift to left, monocytosis, and lymphocytosis may indicate chronic active suppurative process in the body.

FINDINGS NECROPSY

Thick walled hard masses can be palpated at physical examination of the lung. At cut of an abscess, one can find thick fibrous connective tissues covering containing pus of varying quality and quantity in the center. Histopathology of surrounding lung sections may reveal obliterating air-passages and cellular reaction of varying degree and nature depending upon the type of the causative organisms and duration of illness.

DIFFERENTIAL DIAGNOSIS

There are many diseases of chronic nature need rule-out from suppurative pneumonia. Tuberculosis run a similar clinical course but can be diagnosed by performing "Tuberculin" test. Hydatid cysts in the lungs may have clinical picture similar to pulmonary abscesses but the evidence of toxemia and cellular changes typical of bacterial infection may be absent. Pulmonary neoplasia is difficult to differentiate from a large abscess at clinical examination and radiographic analysis. Traumatic reticulo-peritonitis is associated with chronic recurrent tympany, arching of back, positive grunt tests, mild fever, and lack of respiratory system involvement.

TREATMENT

1. Administration of large doses of broad-spectrum antibacterial for several days should be attempted for active abscesses.

2. Proteolytic enzymes, e.g. chymotrypsin (25000 IU), streptokinase or streptodornase (10000 IU), in combination with antibacterial help in lysis of fibrin and pus and deep penetration of antibiotics.

3. Symptomatic treatment for dyspnea, hemoptysis, bronchial exudation, should be applied as and if required.

4. Supportive treatment aimed to improve appetite and health may be adopted in long standing survival animals.

Prognosis: Because of encapsulation of abscesses, the antibacterial treatment is usually not very successful, so prognosis, particularly, in cases of multiple abscesses is not favorable. Therefore, if treatment recommended fails to give any clinical response, slaughter should be advised.

HYDROTHORAX, HEMOTHORAX, AND CHYLOTHORAX

It is the accumulation of either edematous fluid (Hydrothorax), whole blood (Hemothorax), or lymph (Chylothorax) in the thoracic cavity. Clinically, either of the disease, is characterized by signs of respiratory inadequacy due to collapse of ventral parts of lungs.

ETIOLOGY

Hydrothorax may occurs as a part of generalized edema, associated with either congestive heart failure (CHF), hypoproteinemia, or as a sequelae of specific diseases (e.g. bovine viral leukosis, African horse sickness).

Hemothorax occurs due to hemorrhage originating from lungs or damage to visceral or parietal pleural vessels. Hemothorax may also occur due to rupture of pleuritic adhesions following severe exercise or severe coughing, rib fracture, thoracic surgery or punctured wounds, neoplastic growths (e.g. hemangiosarcoma) involving the pleural surfaces.

Chylothorax may occur due to erosion or accidental rupture of any thoracic lymphatics (a rare condition in animals). This may also been recorded as iatrogenic, in heart diseases, fungal infection, heartworms, or in malignancies. This condition is common in dogs (Afghan hounds) than in other animals

PATHOGENESIS

The accumulated of fluid (transudate, blood, or chyle) in the pleural cavity, if in enormous quantity, may result in hinderance of normal gaseous exchange and dyspnea, because of compression at ventral part of lungs. The accumulated fluid may also cause mechanical pressure on the atria and thus increase in venous pressure in greater veins. Rupture of pulmonary parenchyma may also result in pneumothorax.

CLINICAL SIGNS

A slow developing inspiratory dyspnea without the presence of lung sounds (i.e. dull instead of resonant sounds at auscultation), rib-cage movements (at each respiration), pain (at palpation), or fever are the cardinal signs of fluid accumulation in thoracic cavity. A history of trauma followed by appearance of the above signs increases the suspicion of hemothorax. The condition is bilateral and severe in

equine, whereas, it is usually mild and unilateral in ruminants (because of having complete mediastinum). Sudden death may occur in large volume of fluid due to respiratory arrest or damage to major blood vessel and excessive hemorrhage.

Additional signs of hydrothorax, due to congestive heart failure, are the presence of generalized edema, jugular engorgement and pulsation, anemia, and general body weakness. Auscultation may reveal decrease or absence of lung sounds and muffled heart sounds due to fluid accumulation.

CLINICAL PATHOLOGY

Thoracocentesis is required for the identification of the nature of fluid. The fluid drawn can be utilized for its cytological and cultural examinations. The edematous fluid, whole blood, or lymph can be differentiated based on their physical and chemical characteristics. Generally the chyle is whitish or pinkish in color, if it left to stand, a cream layer is formed, and it remains opaque even at centrifugation. The predominant cell type found in chyle is the lymphocyte. The triglycerides are also elevated in chylous effusions compared to serum.

Radiograph may reveal a denser than normal area of lungs with loss of other details due to overshadowing by accumulated fluid.

NECROPSY FINDINGS

The pleural cavity will contain either edematous fluid (in hydrothorax), un-clotted blood (in hemothorax), or lymph (in chylothorax). The evidences involving other organs (in specific diseases) may also be noticed.

DIFFERENTIAL DIAGNOSIS

The diseases need rule-out are; i) Sub-acute pleurisy which is characterized by severe pain, pleuritic friction sounds, and signs of toxemia; and ii) Diaphragmatic hernia, which is associated by the presence of peristalsis sounds, at the vicinity of chest wall, and signs of digestive disturbances.

TREATMENT

1. Immobilize the animal and treat accordingly if the primary disease is known (see congestive heart failure, shock, hemorrhage, anaemia, and edema).

2. Dyspnea due to accumulated fluid may be relieved by thoracocentesis. This will provide relief, may be temporarily, as fluid can accumulate again. Other conservative measures should be applied to control the primary cause and signs of respiratory disturbances.

One may include dietary fat control, IV hyper-alimentation in chylothorax, and continuous pleural drainage.

3. Antibiotics (broad-spectrum) may be used for the control of secondary bacterial infection.

4. Supportive therapy, if and as needed, particularly in long standing cases.

4. Surgical correction of the cause may sometime be required depending upon its severity with instillation of pleuroperitoneal fenestrated drain.

PNEUMOTHORAX

It is the presence of free air in the thoracic cavity, originating either internally from ruptured lung or from external punctured wound at the chest wall. The air in thoracic cavity if in sufficient volume, may cause acute respiratory distress or collapse of lungs, particularly in horses.

ETIOLOGY

It is an uncommon condition both in ruminants and horses: Rupture of chest wall by external trauma, leading to pneumothorax, can occur either by sharp metallic objects, gun-shot injuries, horn gore, broken ribs, or as complication of thoracic surgery. This condition can also occur when rupture of lung occur either due to spontaneous coughing, traumatic reticulitis, tracheotomies, or other causes of tracheal puncture.

PATHOGENESIS

Pneumothorax can be either close, when air is trapped within the pleural space, or open, when air can go in or out through the wound. It can be unilateral or bilateral. Depending upon the volume of air escaped in the thoracic cavity there is interference in normal respiration may followed by respiratory collapse. The outcome of the disease varies, depending upon the volume of air escaped in the thoracic cavity. Small volume of air can be reabsorbed without the development of any ill-health. If volume of air is sufficient, there is development of varying degree of dyspnea , anoxemia, and cyanosis. Simultaneous presence of hemothorax may aggravate the condition.

CLINICAL FINDINGS

A history of external trauma at the chest cavity followed by sudden development of tachypnea, inspiratory dyspnea, and cyanosis in

advanced cases are some common signs of pneumothorax. If the air in the chest cavity is sufficient, and bilateral, there will be development of sudden and fatal respiratory collapse. Auscultation may reveals absence of normal lung sounds in the dorsal thorax when the lung is particularly collapsed. The lung of the normal side will reveal a high pitched resonant sounds. Percussion may reveal hyper-resonance sounds (PING) over the area of pneumothorax.

CLINICAL PATHOLOGY

Thoracocentesis may shows air and no fluid. Radiographs are diagnostic; an area of increased darkness and loss of other details, unilateral (cattle or other animal species) or bilateral (horses) with collapsed lungs are indicative of pneumothorax. Other evidences such as fractured ribs or punctured wounds may also assist in diagnosis.

NECROPSY EXAMINATION

Collapsed lung of effected side with air in the pleural cavity are suggestive of pneumothorax. Un-clotted blood may be present in the thoracic cavity.

DIFFERENTIAL DIAGNOSIS

The condition requires differentiation from bronchopneumonia, viral pneumonia, diaphragmatic hernia, space occupying lesions (large abscesses or tumors), or Clostridial infections.

TREATMENT

Treatment consists of relieving the pneumothorax and treating the cause, which may be attempted by;

1. An open sucking wound should be occluded promptly. Simultaneously, aspiration of escaped air by negative pressure (by syringe or sucking pump), and side by side inflation of lung by positive pressure.

2. Broad spectrum antibiotics as prophylaxis use, during the recovery period.

3. Any fractured rib should be stabilized so as to prevent further damage.

4. Drainage of chest by thoracocentesis, if hemothorax present.

PLEURISY
Syn: PLEURITIS

It is an inflammation of the pleura (both parietal and visceral layers). Acute cases are characterized by shallow and rapid breathing, evidence of pain at respiration, and toxemia. Subacute pleurisy is associated with signs of respiratory arrest due to collapse of lungs with empyema and toxemia. In chronic pleurisy there may be minor degree of respiratory arrest related with restriction in lung movement due to fibrinous adhesions.

ETIOLOGY

The primary pleurisy is association with direct traumatic perforation of thoracic wall from external injuries (bullet wound, fractured rib, horn gore, traumatic reticulitis, etc.) followed by bacterial infection.

The secondary pleurisy may develop as a part of some specific diseases of the lungs in which the causative bacteria gain excess to the pleura through process of extension, e.g. Contagious bovine (caprine, ovine) pleuropneumonia, pasteurellosis (both pneumonic and septicemic types), tuberculosis, sporadic bovine encephalomyelitis, equine infectious anemia, strangles, pulmonary abscessation, severe lungworm infestation, lymphomatosis, and coccidiomycosis. Infection to pleura can also develop through blood or lymphatics from other organs, e.g. peritonitis, pericarditis, or metastating tumors (lymphosarcomas).

PATHOGENESIS

In per-acute and acute pleuritis there is stimulation of pain end-organs at each lung movement due friction of inflamed parietal and visceral layers which result in much pain. Because of this the respiratory movements are restricted in the form of shallow and fast breathing. At this stage exudation is minimal but signs of toxaemia may be evident.

At sub-acute inflammation (which usually follows about 48 hours after infection) there is much exudate collection in the pleural sac, which may either cause collapse of the ventral parts of lungs or interfere with normal gaseous exchange and thus cause reduction in vital capacity. A hinderance in normal heart action and reduction in blood flow may occur if the fluid is too much. At this stage the toxemia is intense because of absorption of both antigenic and metabolic toxins (produce due to bacterial proliferation and tissue damage, respectively).

In chronic pleuritis the exudate is reabsorbed and there is organization of fibroblast on the fibrin resulting in the development of strong fibrous adhesions between the both layers as well as visceral layer and lungs. This may cause mechanical restriction in lung movement and chest wall. At this stage the signs of toxemia is usually absent.

CLINICAL SIGNS

A rapid and shallow breathing with absence of chest movements, disinclined to move, elbows abduction, pleuritic friction (rub) sound which continuous to and fro with each respiration, and manifestations of pain at palpation and percussion of chest wall are substantial evidences of acute pleurisy. Horses may exhibits the signs of colic at this stage.

In sub-acute cases the pain at respiration and friction sounds may be absent, but inspiratory dyspnea with abdominal respiration, high body temperature, severe depression, and complete anorexia are noticeable. Auscultation and palpation may reveal the signs of pain and fluid sounds from the chest cavity. A concurrent pneumonia with painful cough may also be present.

The chronic pleuritis is characterized by moderate respiratory arrest particularly evident at exercise, poor exercise tolerance, progressive weight loss, and emaciation. Adhesions may break following severe exercise or acute cough and outcome in fatal hemorrhage. Some animals may remain healthy without apparent clinical signs.

CLINICAL PATHOLOGY

Complete blood count in per-acute cases may reveal leukopenia, but alarming leukocytosis with left shift may be noted in acute and sub-acute stages of the disease. Thoracocentesis at later stages may reveal exudative fluid containing much cellular contents (neutrophils, lymphocytes, macrophages, mesothelial cells, eosinophils), and causative bacteria. X-rays examination may point out the areas with loss of details and increased density indicating fluid.

NECROPSY FINDINGS

A marked edema, thickening, and hyperemia of pleura with engorgement of adjacent vessels may be noted in per-acute pleuritis. In later stages the pleural cavity may contains highly turbid exudate contain flakes and clots of fibrin. The adjacent lungs may be collapsed and congested. A strong fibrous adhesions between the two layers and/or the lungs is indicative of chronic pleuritis.

DIFFERENTIAL DIAGNOSIS

Pneumonia with pleurisy is difficult to rule-out as clinical and laboratory evidences are similar in both the conditions. Pulmonary emphysema is characterized by loud crepitant lung sounds and expiratory dyspnea rather than inspiratory one as noted in pleurisy. Hydrothorax, hemothorax, and chylothorax are characterized by inspiratory dyspnea with fluid sounds at auscultation but signs of toxemia, pain at palpation, and friction sounds at auscultation are absent. Thoracocentesis can be a testimony of the underlying etiology. Pericarditis is characterized by brisket edema, jugular engorgement and pulsation, and friction sounds which wax and wan with each respiration, contrast to continuous to and fro type friction sound in acute pleuritis.

TREATMENT

1. Specific antibacterials, should be selected based on the sensitivity analysis, be used in heavy doses, and by parenteral route.

2. Drainage of fluid by thoracocentesis is suggested to relieve respiratory distress. Selected antibacterials, diluted in normal saline, can also be administered right away through this route. Enzymes therapy using chymotrypsin, streptokinase, or streptodornase with antibiotic, intrapleurally, can be used in sub-acute cases in order to liquify fibrin, break down fibrinous adhesions, or deep infiltration of antibiotics.

3. Corticosteroids (dexamethasone, cortisone) may be used to minimize inflammatory response in per-acute and acute stage of pleuritis along with antibiotics.

4. Non-steroid anti-inflammatory drugs such as sod. salicylate or phenylbutazone and analgesics (in severe pain) should be used as indicated.

5. To reduce pain and swelling, warm application with blankets, mustard plaster, poultices, or antiphlogestin ointments may be applied particularly in acute stages of the disease and in winter.

6. Anodyne type of expectorant should be used in acute stages of pleurisy accompanying painful coughing.

7. Supportive therapy if and as needed.

CHAPTER-8

DISEASES OF MUSCULO-SKELETAL SYSTEM

The musculo-skeletal system is composed of the striated muscles, joints, bones, tendon, and ligaments. The primary function is to support the body, provide a means of motion, and protection of some vital organs (brain, eye) of the body. The diseases of musculo-skeletal system are primarily manifested by dysfunctions in body movements. The disease of musculo-skeletal system are classified simply of inflammatory or degenerative types. Some of them are described.

MYOSITIS

It is an inflammation of muscles which may be caused by either physical trauma or some specific diseases involving muscles. Acute myositis is characterized by severe lameness, swelling, and signs of toxemia. In chronic myositis only muscle wasting is the major finding.

ETIOLOGY

The specific diseases causing myositis are as under;

i) Bacterial diseases; Black-leg (black quarter), malignant edema, actinobacillosis, and tetanus are some common examples.

ii) Viral diseases; Foot and mouth disease, blue tongue, ephemeral fever, bovine viral diarrhea, equine influenza, and coxsackie disease may cause myositis.

iii) Parasitic infestation; Larvae of certain parasites, e.g. Toxoplasma gondi, Taenia solium or Taenia saginata, and Cysticercosis, if present in the muscles of animals, may cause myositis.

iv) Fungal diseases; Blastomycosis, coccidioidomycosis, and nocardiosis, can be associated with myositis.

v) Miscellaneous causes; Eosinophilic myositis in beef calves and dogs, Atrophic myositis in dogs, Myositis ossificans (an inherited condition), chemical myositis due to IM injection of irritant chemical or drugs (Iron preparations, chloral hydrate, antibiotics), and neoplasia of muscles (lymphoma, hemangioma) are some examples.

vi) Physical causes; Trauma to muscles caused by wither kick, blow, bite, road accident, sprain, or fire-arm injuries, which sometime may followed by secondary bacterial infection, are common causes of myositis.

PATHOGENESIS

The initial response to infection or trauma are the cellular infiltration (leukocytes) and inflammatory changes (varying degree and types) in the muscles. Commonly, the degenerative changes varies from cloudy swelling, fatty degeneration, to hyaline necrosis. Some of the specific changes are seen in specific diseases, e.g. emphysematous swelling in black-leg, eosinophilic infiltration in eosinophilic myositis, and calcification in myositis ossificans. The damage to muscle cell wall will cause the release of certain muscle enzymes and myoglobin into blood circulation.

CLINICAL FINDINGS

The signs of inflammation i.e. swelling, heat, pain, and loss of function can be seen in the muscle(s) involved. If leg muscles are involved, there will be lameness, stiff gait, difficulty in sitting and standing, or persistent recumbency. If neck muscles are affected, the neck may be turned to one side with difficulty in grazing, and so on. The signs of toxemia and systemic reaction may also be present in acute cases. Urine color is changed from red to chocolate due to the presence of myoglobin. In chronic cases, wasting and atrophy of affected muscles are common signs.

CLINICAL PATHOLOGY

Complete blood count may reveals leukocytosis with shift to left in acute cases of myositis (but eosinophilia in eosinophilic myositis). The serum muscle enzymes, particularly serum creatinine phosphokinase (SCPK), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH) will be increased. The SCPK is a specific enzyme, having half-life of 2-4 hours, so its elevation in serum is a good indication of active muscle damage. The AST is also a specific enzyme, having half-life of about 7 days. Its high level in blood along with high SCPK level is diagnostic for acute myositis. Muscle biopsy may be obtained for histological and immuno-histochemical examinations.

NB: These enzymes can also be increased with fatiguing exercise, long journey, race, damage to heart muscles (myocarditis), hepatitis, or RBCs destruction. Therefore, these conditions should also be considered while making conclusion.

NECROPSY FINDINGS

The affected muscles will appear dark red to black at cut due to congestion and inflammatory changes in acute myositis. Fibrosis and calcification are common outcome of chronic cases. Histological changes may include form cloudy swelling, fatty degeneration, to

selenium deficiency in animals consuming fodders growing on that soil.

viii) Congenital myopathy: Occasionally congenital myopathy may be seen in new-born animals, and it is suspected to be caused by congenital infections (e.g. Akabani virus infection in uterus).

PATHOGENESIS

An important inter-relationship and synergism exist between vitamin-E and selenium in preventing vitamin-E deficiency. Selenium (an essential trace element) and vitamin-E play an important role in cell protection by destroying cell oxidizing agents (e.g. Hydrogen-peroxide, lipid-peroxides, superoxide, singlet oxygen radical, etc). Selenium dependant glutathione peroxidase destroys these toxic oxygen radicals. Deficiency of vitamin-E or selenium can cause deficiency of this enzyme and thus result in lipid peroxidation of cell membrane and cell necrosis.

In exertional myopathy, there is increased (anaerobic) glycolysis with depletion of muscle glycogen and increase production of lactate and other metabolites in the muscles, causing hyaline degeneration to coagulative necrosis of muscle fibers. At any muscle damage there is increase in release of muscle enzymes (e.g. creatine phosphokinase, aspartate aminotransferase, alkaline phosphatase, and lactate dehydrogenase) and myoglobin in serum.

CLINICAL FINDINGS

In acute myopathy, animal appear bright but there is sudden onset of stiffness of gait and inability to move which may be accompanied by respiratory or circulatory disturbances. Other signs includes sweating (horse), firmness and pain at palpation of muscles, and myoglobinuria. The animals may die suddenly due to cardiac muscle involvement. In less acute cases the signs may vary from stiffness to paresis, pseudo-paralysis, and prolonged recumbency.

Sub-acute cases are characterized by slow developing lameness, short shuffling gait, lethargy, upward arching of back, difficulty in weight bearing, recumbency, and passing of chocolate colored urine. Temperature may remain within normal limits. Occasionally, there may be hyper-salivation and desquamation of lingual epithelium.

CLINICAL PATHOLOGY

Increase in serum muscle enzymes level, particularly SCPK and AST, and presence of myoglobin in urine are diagnostic of active muscle damage. Muscle biopsy may be helpful to identify to cause and resultant changes in the muscle fibers.

NECROPSY FINDINGS

In nutritional myopathy, the affected muscles appears white, waxy and swollen (like fish flesh) in the form of linear strips of large muscle masses and the distribution of lesions are characteristically bilaterally symmetrical. Microscopically, the degenerative lesions include from hyaline to coagulative and from mild to severe necrosis. Sometimes there may be disappearance of groups of muscle fibers and replacement with connective tissue and occasionally, by mild degree of calcification. The biopsy material of 1/4 x 1/4 x 1/2 inches in dimension is sufficient for histopathological examination.

DIFFERENTIAL DIAGNOSIS

This condition needs to be differentiated from myositis, arthropathy, osteodystrophy, local nerve damage, and paralysis.

TREATMENT

The treatment should be started as early as possible with adequate supportive therapy, well before the development of irreversible degenerative changes in the affected muscles. It include;

1. Specific treatment comprising of the use of vitamin-E and selenium (alone or in combination). For this purpose, a combination of Alpha tocopherol acetate and Sodium selenite, at a rate of 150 mg and 3 mg per ml respectively. It can be given @ dose of 2 ml/45 kg, IM, for 4-5 days.

2. Fluid and electrolytes balance should be maintained by using Ringer's or Saline dextrose solutions. In cases of acidosis the use of Sodium bicarbonate, 6 per cent solution, @ dose of 500-1000, IV, is recommended to keep the urine alkaline and avoid the precipitation of myoglobin in renal tubules, which otherwise can cause myoglobinuric acid nephrosis.

3. Sedatives and tranquilizers, i.e. promazine and chlorpromazine (@ dose of 2-5 mg/kg), acetyl promazine (Sedastress), xylazine (Rompun), Diazepam, mepredine hydrochloride (@ dose of 5 mg/kg), or chloral hydrate (@ dose of 40-60 gm/LA, PO) may be used in cases of severe pain.

4. The use of corticosteroids (to improve tissue perfusion), non steroid anti-inflammatory drugs (Sodium salicylate), thiamine hydrochloride, @ dose of 500 mg/LA, IM, or calcium borogluconate, @ dose of 300-450 ml/LA, IV, can also help to improve the condition.

CLINICAL PATHOLOGY

Aspiration of joint fluid with a sterile needle may be obtained for its biochemical and cultural examinations. Blood examination may reveal leukocytic changes (and left shift), depending upon the severity of infection. X-rays examination may be helpful, particularly in chronic cases, to identify the severity of lesion development at articular surface and adjacent bones. Serological tests may assist in diagnosis of specific diseases, particularly, brucellosis, tuberculosis, salmonellosis, and mycoplasmosis.

NECROPSY FINDINGS

Varying in degree of pathological lesions depending upon severity and duration of the disease. A thickening and roughening of synovial membrane, erosions at articular cartilage, and presence of fibrino-purulent exudate are suggestive of acute arthritis. Chronic arthritis is characterized by ossification at articular cartilage and surrounding surfaces. The synovial fluid is changed both in quality and quantity and may contain the causative bacteria.

Characteristics of synovial fluid:

	Normal	Suppurative arthritis	Degenerative arthropathy
Color	Clear	Yellow/ green	Yellow
Clarity	Transparent	Turbid	Transparent
Volume	Low	Increased	Low
viscosity	High	Low	Variable
Fibrin clots	Absent	Present	Absent
WBCs	<200	30,000-100,000	200-2,000
Gram's stain	No bacteria	Bacteria present	No bacteria

TREATMENT

1. Specific antibacterials.

Acute infectious arthritis should be treated as soon as possible with specific antibiotics particularly following sensitivity test, in order to avoid irreversible changes in the joint. The antibiotics should be administered for several days and even after clinical recovery. The antibiotics used should be of lower molecular weight for better penetration into synovial membrane. e.g. Penicillin-G (@ dose of 20,000 i.u./kg) and Streptomycin (@ dose of 10-20 mg/kg) alone or in combination, Terramycin, Kanamycin (@ dose of 6 mg/kg), Gentamicin (@ dose of 2 mg/kg), Cloxacillin, Methicillin, Cephalosporin, Erythromycin, Tylosin (for mycoplasmosis).

2. Cold applications (ice packs or cold water) locally in peracute stages, and warm application or rubifacients (warm water or heat pads, liniment turpentine, iodine ointments, muster poultices) in acute or subacute stages of the disease. In chronic arthritis, the use of counter irritants (red iodide of mercury) or hot-iron firing (pin point or linear type), or combination of both, may be adopted to turn chronic inflammation to acute one for the promotion of healing processes.

3. Analgesics as sodium salicylate, @ dose of 25-50 gm/LA, PO, or phenylbutazone, @ dose of 2 gm/300 kg, PO, may be advised to reduce pain in acute stages of the disease.

4. In chronic suppurative arthritis joint lavage may be recommended. This is achieved by aspiration of exudate form joint capsule by using a sterile syringe and needle followed by irrigation with solution of antibiotics in sterile normal saline at pH 7.0. Surgical drainage, debridement of membrane, and articular surface may some time be required for removal the necrosed structures.

5. In chronic arthritis, intra-articular injection of antibiotics combined with proteolytic enzymes may be useful.

6. In non suppurative arthritis, the use of long acting corticosteroid with antibiotics by IA route, to reduce local swellings and pain. For this purpose, Methylprednisolone acetate, @ dose of 20-40 mg/joint or Prednisolone, @ dose of 2-5 ml/joint are in common (in human medicine).

ARTHROPATHY

Syn: DEGENERATIVE JOINT DISEASE, ARTHROSIS, POLYARTHROSIS, AND OSTEOARTHROSIS.

It is the non-inflammatory lesions at the articular surface of joint(s), including degenerative and erosive changes in articular cartilage, eburnation of sub-chondral bones, and hypertrophy of bone surrounding the articular cartilage, characterized clinically by stiffness of joint(s) and chronic progressive lameness.

ETIOLOGY

A) The primary arthropathy is the result of wear and tear as a part normal aging process. It may start quite early in some individuals than others, perhaps inheritance may be the factor.

B) The etiology of secondary arthropathy may be grouped into:

i. Conformational or angular limb deformities caused by either congenital or acquired factors.

ii. Acute and repeated traumatic injuries at the joint that increase the rate of cartilage loss.

iii. Nutritional deficiencies such as calcium, phosphorus, copper, manganese, and magnesium. Examples includes rickets (in all species of animals), osteodystrophia fibrosa (horse), and osteomalacia (ruminants).

iv. Chronic chemical poisoning, such as zinc, fluorine, and selenium.

v. Metabolic disorders, hormonal imbalances, obesity, and inherited spasticity have also been reported to be the causes of arthropathy.

PATHOGENESIS

The exact mechanism of arthropathy development is not well understood. The primary arthropathy is a part of normal aging processes and ordinary joint usage. With increasing age there is loss of normal resilience of cartilage, because of lowering of chondroitin sulphate and reduction in permeability of the cartilaginous matrix, which can result in progressive degeneration of articular cartilage.

The etiology factors associated with secondary arthropathy result in greater shearing stress on some particular points, causing erosions of the cartilage, increase density of sub-chondral bone, and proliferation of bone and cartilage at articular margins. There may be no remarkable change in synovial fluid. Cartilage damage can also occur through indirect pathway, i.e. damage to chondrocytes and the release of lysosomal enzymes that may cause the loss of intercellular proteoglycans, thus more wearing off the cartilage.

CLINICAL FINDINGS

The severity of signs will depend upon the joint involved, amount of cartilage degenerated, and periarticular remodeling. A slow but progressively (usually bilateral) developing lameness and stiffness of joint which do not respond to medical therapy are characteristic of arthropathy. The limitation in movement of a joint usually results in disuse muscle atrophy and prominence of bony points of the joint. A constant shifting of weight due to pain, prolonged recumbency, difficulty in sitting and standing, progressive loss of body condition, poor appetite, and loss of production are additional signs.

Crepitus can be produced in larger joint due to detached pieces of cartilage, bone, or osteophytes surrounding the articular cartilage. There may be no visible distension of joint capsule or pain at palpation (pain can be elicited from manipulation and flexion of a

joint). Signs will also vary with the joint involved, e.g. arthropat coxofemoral joint may interfere with breeding act in male.

CLINICAL PATHOLOGY

The quality and quantity of synovial fluid remain norm Radiographic examination of affected joint may reveals varying deg of proliferative and degenerative changes. There may be imbalance serum calcium and phosphorus level in advanced stages of disease.

NECROPSY FINDINGS

The joint cartilage become discolored, thin, patchily eroded ulcerated, and sometimes folded. The non-articular surface contai osteophytes deposits. There could be calcification of joint capsule ar chip fracture of the head of the tibia. The head of the long bon becomes flattened and smaller than normal. The synovial lini becomes thick, red, and proliferative.

DIFFERENTIAL DIAGNOSIS

	Arthritis	Arthropathy
1. Lameness	Acute	Chronic
2. Speed of development	Quick	Slow
3. Treatment response	Good	No response
4. Visible swelling at joint	Yes	No
5. Signs of acute inflammation	Present	Absent
6. Synovial fluid	Increased	Almost normal
7. Contain RBCs, WBCs, pus cells and bacteria	Yes	No, may contain few RBCs & WBCs
8. Fluid clots after collection	Yes	No

TREATMENT

There is no specific treatment for arthropathy. The goals of treatment are to maintain joint motility and slowing the process of degeneration. For this purpose following lines of treatment should be adopted.

1. Rest and restrict strenuous exercise and abnormal weight bearing activities. Normal exercise may be helpful.

2. Use of analgesics and anti-inflammatory drugs such as salicylates, phenylbutazone, meclofenamic acid can be used for reducing pain and swelling.

3. Intra-articular injection of long acting corticosteroids or hyaluronic acid can provide temporary relief from pain and discomfort. Corticosteroids don't promote healing of joint, instead their long term

use in arthropathy may actually accelerate erosion, loss of joint sensation, and so may lead to the development of steroid arthropathy.

4. Surgical treatment includes curettage of necrosed articular cartilage, removal of osteophytes, and reduction of degenerative process by re-establishing alignments of supporting tissues.

5. Soft flat floor, warm and dry quarters, and diet containing balanced calcium, phosphorus, and other essential minerals can delay the process of degeneration.

NB: In cases of irreversible, troublesome, multiple, and chronic joint lesions, slaughter or euthanasia should be considered.

OSTEOMYELITIS

It is inflammation of bone and central marrow cavity, usually caused by pyogenic organisms that are mostly introduced through trauma, and characterized by erosion of bone, chronic lameness, and continuous discharge of exudate.

ETIOLOGY

The causative organisms (commonly pyogenic, less commonly non-pyogenic bacteria, and occasionally fungi) can be introduced into the bone tissues either through trauma to bone as in compound fracture, penetrating wounds, non-sterile orthopedic surgery, or tail docking (sheep). Infection may also spread through lodging of septic emboli in the bone marrow via blood originating from other organs. The common organisms isolated from osteomyelitis belong to species of Streptococci, Staphylococci, Salmonella, Corynebacterium, Brucella, Mycobacterium, Actinomycetes, and Escherichia coli. The common fungi reported to be isolated from osteomyelitis belong to species of Coccidioides, Blastomyces, and Nocardia.

PATHOGENESIS

Following introduction and establishment of infection in bone(s) and marrow cavity there is massive accumulation of leukocytes at the site of infection. Lysosomal enzymes and other products are released from activated leukocytes which thus result in tissue necrosis, suppuration, vascular thrombosis, and bone ischaemia.

CLINICAL FINDINGS

The clinical signs will depend upon the bone(s) involved. In acute cases, a persistent lameness, postural abnormalities, swelling, warmth, and pain at palpation are common signs. The patients are reluctant to move or found in a state of constant recumbency. In

chronic cases lameness and discharge of pus into surrounding tissues causing cellulitis or phlegmon, or to the surface as a sinus are common. The affected bone becomes weak and so fracture easily. When bones of jaw are involved there will be difficulty in chewing and mastication. Involvement of vertebral column may lead to the spread of infection to the meninges, which can cause paralysis of the caudal area. Multiple lesions may be seen in young animals, which are deficient of maternal immunity. In these cases, the affected animals remain recumbent and unable to stand because of severe pain. Elevation of body temperature, anorexia, and depression, in acute cases, and progressive weakness and weight loss in chronic cases are additional signs.

CLINICAL PATHOLOGY

Pus samples should be obtained for their staining reaction, culture, or sensitivity tests. Blood examination may reveal leukocytosis with left shift, higher plasma fibrinogen level, and increases ESR. Radiographic examination particularly in chronic cases, may display loss of bone density, bone lysis, and new bone formation around the affected area.

POSTMORTEM FINDINGS

The typical lesions of osteomyelitis may not be obvious, unless the bone is opened longitudinally. The cut surface of the metaphysis and epiphysis may expose inflammatory changes of varying degree.

TREATMENT

1. Use of specific antibiotics, both systemic and local, opting for bacteriocidal effects, are recommended. These include ampicillin, cephaloridine, chloramphenicol, gentamicin, kanamycin, and amphotericin-B (for fungal infection). The chemotherapy should be continued at least for a week following clinical recovery.

2. Surgical debridement and furnishing drainage at the infected site may sometime be adopted. Dead and necrosed tissues should be removed and the area cleaned of pus using specific antibiotic diluted in normal saline. To achieve desired objectives, all sequestra should be removed, fibrous tissues be explored, and bone curetted till bleeding. The superficial wound should be left opened for drainage and healing by granulation.

3. Fenestrating tubing can be implanted to allow flushing of the infected area utilizing a large volume of sterile saline solution mixed with antibiotic and enzyme (streptokinase, streptodornase).

4. Proper rigid fixation of bone is required for healing of compound fracture.

5. In cases of failure of response even with intensive long term antibiotic therapy, amputation of affected appendages may be the solution as a last resort.

OSTEODYSTROPHY

It includes those diseases of bones in which there is failure of normal bone development (Rickets) or abnormal metabolism of bone which is already mature (Osteomalacia in cattle, Osteodystrophia fibrosa in horses, and exostoses and osteoporosis in adult animals). Clinically these conditions are characterized by distortion and enlargement of bones, liable to fracture, and hindrance in posture and gait.

ETIOLOGY

1. Absolute dietary deficiency or imbalance of calcium, phosphorus, or vitamin-D, for a long period, is the main cause of osteodystrophy. Deficiency of copper (cause of osteomalacia in ruminants), protein (cause of osteoporosis in cattle and sheep), and other vitamin and minerals (Vitamin-A, Mg, Zn, and Mn) can also cause this condition.

Chronic poisoning with either lead (causing osteoporosis), fluorine (causing exostosis), or eating of certain toxic plants (e.g. turnips, rape, setaria and solanum spp.) may result in osteodystrophy. Hyperthyroidism (a cause of osteodystrophia fibrosa), inherited predisposition (e.g. Achondrosis), physical injuries to bone, and malconfirmation can also cause bone deformities.

PATHOGENESIS

A low level of vitamin-D in the diet (or serum) for a longer time -> decrease absorption of Ca -> low serum calcium -> stimulate parathormone production and release -> increase urinary excretion of Po_4 and increase serum Ca -> low serum phosphate -> abnormal Ca:P ratio -> failure of calcification and ossification.

Rickets is a disease of young and fast growing animals in which there is failure of provisional calcification of the osteoid tissues and defective mineralization of developing bones. The underlying sequence of events includes;

a). Failure of cartilage to mineralize properly,
b). Failure of growing cartilage to degenerate,
c). Irregular persistence of cartilage,

d). Formation of osteoid on persistent cartilage that forms an irregularity of osteochondral junctions,
e). Over growth of fibrous tissues in the metaphyses, and
f). Bone deformity.

All of the above may result in increase in depth and width of the epiphyseal plates particularly of long bones. The uncalcified soft tissues of the metaphyses and epiphyses become distorted under the pressure of weight bearing which may also cause deviation of the shaft of long bones. The decrease rate of longitudinal growth of bones and enlargement of the ends of long bones due to affect of weight, cause flaring of the diaphysis adjacent to the epiphyses plates. The articular cartilage remains normal or there may be sub-articular collapse which may result in grooving and folding of articular cartilage and ultimately degenerative changes.

In osteomalacia, there is softening of mature bone due to extensive resorption of mineral deposits in bone and failure of mineralization of newly formed matrix. There is no enlargement of ends of long bones but spontaneous fracture is common.

Osteodystrophia fibrosa occurs in horses receiving diet low in calcium and high in phosphorus (particular grains) or secondary to hyperparathyroidism. There is extensive resorption of bone and replacement with connective tissue. There is swelling of mandibles, maxillae, and frontal bones. Spontaneous fracture of long bones is common.

Osteoporosis develops due to failure or inadequacy of formation of the organic matrix of bone thus bone becomes porous, light, fragile, and fracture easily.

Exostoses may develops due to periosteal hyperostosis. The articular surface remains normal but periosteum may be involved. There is encroachment of osteophytes on the tendons, ligaments, and shaft of the long bones called exostoses.

CLINICAL FINDINGS

Slow developing lameness, reluctance of walking, habitual recumbency, stiffness in joint action, and deformities of bone and joints are the cardinal signs of osteodystrophy.

In rickets, engagement of the ends of long bones and osteochondral junction, deformities of axis and bending of long bones, delayed and irregular eruption of teeth are common signs. Upward arching of back,

poor appetite, poor growth, and poor body condition are some additional signs.

Bone fracture with minimum or no apparent stress along with signs of lameness are evidences of osteomalacia.

In osteodystrophia fibrosa, there is facial distortion due to thickening and enlargement of mandible, maxilla, and frontal bones, which initially are plastic soft but later grows hard. Spontaneous bone fracture with minimum stress are also common.

CLINICAL PATHOLOGY

Serum calcium and phosphorus levels may remain normal for a long period until the lesions are well advanced. Radiography is good mean of diagnosis. In rickets, it may reveal cortical thinning and lack of density of bones, bowing of long bones, ends of long bone appear wooly and moth-eaten, enlargement and widening of the epiphyseal plates, an irregular radiolucent band at the junction of the metaphysis with the epiphysis, and appears concave or flat instead of convex contours. In osteomalacia, a decrease in density of bone shadow and in osteodystrophia fibrosa, an increase of translucency of bones may be noted.

The organic matter to ash ratio is decreased from 3:2 to 1:2 or 1:3 in osteodystrophy.

NECROPSY FINDINGS

In rickets the ends of the long bones are enlarged and epiphyses prominent, circumscribed by periosteal and fibrous tissue thickening. At sectioning, the cortices may appear thinner with enlarged marrow cavity. In addition, erosion of articular cartilage may be noted in osteomalacia. In osteodystrophia fibrosa, the hard bones of mandibles, maxilla, nasal bones, and red bone marrow replaced with fibrous tissues may be noted.

TREATMENT

1. Provision of suitable source of calcium and phosphorus in ratios of 2:1, 1:1, to 1:3, combined with parenteral injection of vitamin-D. To achieve this one may use di-calcium phosphate (containing 23 per cent Ca, 18.5 per cent P), calcium gluconate, monosodium (or ammonium) phosphate, bone meal, or ground limestone (which is a cheap and good source of calcium). Injections of vitamin-D (@ dose of 10,000 to 30,000 i.u./kg. IM) may be necessary for the effective utilization of Ca and P, particularly in rickets.

2. Good quality sun dried hay (alfalfa or clover) is a better and natural source of calcium and vitamin-D.

3. Copper deficiency may be treated with copper supplements.

4. Other than sun-light, the sources of vitamin-D are cod-liver oil or other fish oils which can be supplemented in needs.

NB: A slight acidic pH tends to promote absorption of Ca from G.I. tract, so grain diet, acetic acid, or vinegar may enhance calcium absorption.

BURSITIS

It is inflammation of bursa, mostly those located at the bony prominences or between tendons, and are named according to the structure involved. For example, inflammation of bursa over the olecranon process is called as capped elbow, of knee joint as capped knee, and of tuber calcis as capped hock.

Acute serous bursitis is usually caused by repeated trauma and is characterized by rapid swelling, local heat, pain at palpation, and lameness. The signs of pain may be absent if only subcutaneous tissues are involved. Hematogenous spread of infections to bursa may be possible in some specific diseases, e.g. brucellosis. In chronic bursitis, the signs of acute inflammation are usually absent except the presence of pain at joint movement, occasionally.

TREATMENT

1. Symptomatic treatment is advised according the nature of inflammatory response. For example, local use of cold application along with parenteral or oral use of analgesics in acute cases and warm application in chronic cases.

2. Aspiration or drainage of inflammatory fluid, using sterile instruments.

3. Surgical debridement if and as required.

TENDINITIS (Bowed tendon)

It is the inflammation of tendon with partial rupture of tendon fibers. This condition is mostly seen in horses used for fast work (race horses). Flexor tendon (and of fore limbs) are more frequently involved than extensor tendons (and of hind limbs). This condition is commonly

seen in animal which are forced to exercise, over exertion without proper conditioning, particularly over any hard and rough tract. Improper shoeing and poor confirmation may also lead to tendinitis.

Evidences of acute lameness, abnormal standing posture with heal upward, visible swelling, and pain at palpation the structure are common clinical signs. Chronic cases may end up in fibrosis and fibrotic adhesions with shortening of tendon.

TREATMENT
The lines of treatment should include;
1. Complete rest.

2. Cold application (to reduce swelling and pain) in acute cases and warm application or use of counter irritant, in chronic stages of the condition.

3. Surgical correction.

SPRAINS, SUB-LUXATION, AND LUXATION

Sprain is the abnormal over stretching of periarticular ligaments, characterized by mild to severe non weight bearing lameness, visible swelling, and tenderness of joint.

Sub-luxation is the partial dislocation (destabilization) of a joint. Luxation is the complete dislocation of a joint with or without ligament damage. Both the conditions are characterized by acute lameness and postural abnormalities.

TREATMENT
1. Attempts to reduce local pain and swelling.

2. Immobilization of the joint for a variable time period.

3. Manual or surgical correction of luxated joint.

ANKYLOSIS

It is obliteration of a movable articulation which become rigid due to the formation of new bones, bridging between opposing periosteal callous. The condition is characterized by loss of joint movement, lameness, postural abnormalities, stiff gait, and bony enlargement at the joint. The common cause of ankylosis is repeated trauma to joint

and inflammatory or non-inflammatory joint diseases. Counter irritants or hot iron firing may used as treatment, however, the treatment of chronic condition is not affective.

SPONDYLITIS, SPONDYLOSIS

It is an aseptic degenerative disease of vertebral bodies particularly of thoracic and lumber vertebra, leading to ankylosis of affected vertebra. The cause of this condition is not clear, but could be related to the normal aging processes. Reluctance to move, still gait, dragging of hind feet, and difficulty in standing are some of the initial signs. In the later stages, ataxia, paresis, paralysis, and loss of spinal reflexes could be noted. Radiographic examination may be helpful to delineate the affected parts.

Analgesics may be used in treating the acute cases, however, in advanced cases the prognosis is usually guarded.

MUSCLE SPASM AND MYOCLONUS

Muscle spasm is a sudden transient and involuntary contraction of a muscle or a group of muscles, attended by signs of acute pain and loss of function of the affected muscle(s).

Myoclonus is a series of shock like contractions of a muscle or a group of muscles, and which could be localized or generalized. The condition could be either due to dysfunction or brain, spinal cord, peripheral nerve, neuro-muscular junction, or muscle itself. Examples are electrolyte imbalances, certain poisoning (strychnine). Analgesics and muscle relaxant may used along with specific treatment according to the cause.

PARESIS, WEAKNESS OF MUSCLE

Paresis refers to the loss or decrease in normal motor function from a neurologic or musculo-skeletal defect. Muscle weakness refers to a lack of physical strength of muscles, usually caused by generalized diseases that affect the musculo-skeletal system. This condition is not similar to depression, ataxia, or incoordination. Multivitamins and neurotonics may be used in the treatment.

CHAPTER-9

DISEASES OF NERVOUS SYSTEM

The nervous system comprises of central nervous systems (CNS) and peripheral nervous systems (PNS). The CNS comprises the brain and spinal cord, and PNS comprises the cranial and spinal nerves, and the peripheral nervous system. Most of the diseases of nervous system are either due to any defect in the transmission of impulses through the nervous system or local physical damage to the nervous cells or discrete lesion of the organ. However, the physical changes may not be detectable even by electron microscopy. For the normal functioning of the nervous cells, there is requirement for electrolytes, glucose, oxygen, and other nutrients. Only oxygen, carbon dioxide, and glucose can readily cross blood brain barrier. The major objective here is to recognize the common diseases of nervous system and assess whether treatable or non-treatable (for proper salvage).

NERVOUS SIGNS

The patterns of nervous signs depends upon either enhancement or depression in transmission of impulses along the nerve fibers. The clinical signs which should rouse suspicion of neurological disturbance include abnormalities of posture, gait, movements, sensory perceptivity, mental state, and sphincter activity.

The clinical signs of nervous system involvement can broadly be grouped into 2 types.

Type 1. Signs of irritation, excitation, or exaggerated nervous activity; These type of signs develop due to increase in number of nerve impulses received because of excitation or inflammation of neurons or by facilitation of passage of stimuli. Examples includes the use of stimulant drugs, inflammatory diseases of nervous tissues, or mild degree of anoxia.

Type 2. Signs of paralysis, loss of function, or depression of nervous activity; These signs develop due to decrease in number of nerve impulses because of depression, degeneration, or death of nerve cells. Examples include complete failure of oxygen supply, death of nervous cell in the terminal stages of many diseases of nervous system, toxins, or lack of essential nutrients.

Type 1. The exaggerated, excitation, or irritation signs originating from different origin and include;

a. Sensory system; The major signs of sensory system involvement are hyperaesthesia (over sensitivity to physical touch, light, or noise) and paresthesia (abnormalities of sensation).

b. Motor system; The major clinical signs of motor system involvement are convulsion (involuntary contraction of limbs) and tremor (continuous involuntary trembling of muscles). The release signs are in the form of combined limb action in rate, range, force, and direction.

Type 2. Signs of paralysis, depressed activity, or loss of function originating from different origin include;

a. Sensory system; The major clinical signs of depression of sensory system are hypoesthesia (diminished sensitiveness) or anaesthesia (complete loss of sensation which could be local or general/systemic).

b. Motor system; The major signs of loss of motor activity are paresis (a state of temporary paralysis in which there is difficulty in rising, staggering of gait, and easily falling) and complete paralysis.

COMMON MANIFESTATIONS OF DISEASES OF THE NERVOUS SYSTEM

A. Mental State: (i) The irritation signs originating from cerebral cortex are;

Mania; Abnormal behavior which is not influenced by external environment, as we see them, such as licking, chewing (gum fits), abnormal voice, panicky behavior, constant bellowing, apparent blindness, walking aimlessly, drunken gait, depraved appetite, and aggressive behavior. The common diseases characterized by mania are encephalitis (rabies), metabolic diseases (hypoglycemia, acetonemia), poisoning (lead, carbon tetrachloride), etc.

Frenzy; Violent uncontrolled activity without regard to surroundings which could be dangerous in the form of physical attacks to others even to inert objects in the form of biting, butting, or charging. The diseases associated with frenzy are encephalomyelitis (rabies, Aujeszky's disease), metabolic diseases (hypomagnesemic tetany), poisoning, acute colic, or acute photosensitization.

(ii) The signs of loss of function mental state originating from cerebral cortex are;

Somnolence (sleepy or drowsiness); These may be seen in many poisonous conditions, e.g. bromide, Amitraz, or methyl alcohol poisoning.

Lassitude; It is the disinclination to move or un-interested oneself.

Narcolepsy; It is the episodic and uncontrollable sleep in which the cause may be inherited in some breeds of animals.

Syncope; It is the sudden development of fainting. It may occur from acute heart failure, cerebral hemorrhage, cerebral anoxia, electrocution, or traumatic injury to brain.

Coma; It is the complete loss of consciousness with no response to external stimuli along with dilation of pupils, e.g. encephalomyelitis, encephalomalacia, uremia, hypoglycemia, hypocalcemia, acute toxemia, hypoxemia, heat stroke, concussion or contusion of brain.

Head pressing; It is the pushing head down against fixed objects. Compulsive walking is an other form of the same syndrome where affected animal put their head down and walk, often appear blind. The condition may be seen in toxic or metabolic brain diseases, space occupying lesions, and encephalomyelitides.

Aimless wandering; It is similar to compulsive walking with evidences of depression, apparent blindness, constant chewing, and protrusion of tongue. This condition may be seen with space occupying lesions, chemical or plant poisoning, and degenerative brain diseases.

Nervous shock or unconsciousness; It is the temporary cessation of function of part or whole nervous system present in immediate vicinity of the primary acute traumatic lesions of the brain.

B. Involuntary Movements:

Muscle tremor; It is continuous and repeated twitching of skeletal muscles. This condition may be seen in many degenerative, toxic, or metabolic diseases involving nervous system.

Tics; These are spasmodic twitching made at longer intervals than tremor, usually of many seconds, and is caused by injury to a spinal nerve.

Convulsions; It is violent muscular contractions of a part or whole body occurring either for short periods in which there is alternate periods of convulsion and relaxation or a long period of convulsion without intervening periods of relaxation. The convulsions may be clonic or atonic in which there is repeated muscle spasms alternate with periods of relaxation, and tetanic or tonic which is prolonged muscle spasm without intervening periods of relaxation. Convulsions

are mostly seen in meningitis, encephalomalacia, cerebral edema and ischemia, space occupying lesions in the brain, hypoglycemia, poisoning, or congenital defects in the brain.

C. Posture and Gait: These are controlled by sensorimotor system, and depends upon the tone of skeletal muscles.

Posture; Abnormal posture is adopted either due to pain, blindness, or diseases of peripheral or central nervous system causing disturbance of balance. Deviation or rotation of head, drooping of lips, eyelids, cheek, and ears, head pressing, sternal or lateral recumbency, and dog sitting posture are some examples.

Gait; Abnormality of gait in the form of ataxia and incoordination of muscle action may be seen in many diseases including of peripheral or central nervous system involvement.

Paresis; is difficulty in rising, staggering gait, easily falling, persistent recumbency, decrease muscle tone and reflexes are some evidences of paresis. It develops due to depression in neuromuscular transmission due to either defects in spinal cord and motor neuron or metabolic and toxic diseases.

Paralysis; It can be flaccid or spastic. The flaccid or complete paralysis occurs because of nerve cells death, characterized by absence of voluntary movement and muscle tone, no tendon jerking, no anal reflex, and muscle wasting with time. (The muscle wasting or atrophy of limbs or neck indicates damaged to the particular nerve that innervates that muscle and can be helpful in localizing a lesion to that site).

The spastic or partial paralysis characterized by absence of voluntary movements, increase in muscle tone and reflexes, extension of all limbs, opisthotonos, and increase tendon reflexes (jerking). It can occur due to damage to medulla, pons, or midbrain caused by abscess or listeriosis.

Paraplegia; It is the paralysis of hind limbs along with bladder and rectum (sphincter), usually caused by paralytic rabies or local spinal cord meningitis.

Hemiplegia; It is the paralysis of either right or left side of the body.

Vestibular disease; It may be due to affection of vestibular nerve (otitis media, listeriosis) may cause rotation of head, imbalance, facial

paralysis, and falling on normal side. Nystagmus and forced circling are some additional signs.

COMMON SIGNS OF CRANIAL NERVE DYSFUNCTION

Head pressing (Butting);
Propulsive walking;

Circling; Circling with head rotation or deviation (tilt) to the side. It is seen when lesion in the vestibular nucleus, cranial/facial nerve, or cerebrum.

Blindness; (With or without pupillary reflex, animal bumps into objects). It is usually seen with damage to optic nerve (avitaminosis-A), cerebral cortex (lead poisoning, polio-encephalomalacia), or retina (avitaminosis-A).

Mydriasis; It is abnormal dilatation of pupils and seen in oculomotor nerve damage or retinal defects.

Miosis; It is abnormal constriction of pupils, and seen when diffuse lesions as polio-encephalomalacia and acute lead poisoning.

Paralysis of pharynx, larynx, tongue, and coma; With or without convulsion (clonic, tonic or tetanic).

Twitching of muzzle;

Nystagmus; It is the involuntary jerky repetitive eyeball movement. If it is associated with rotation of head then lesion will be located in the vestibular apparatus (otitis media or interna). Nystagmus with impaired consciousness, abnormal pupils, opisthotonos, and facial palsy is seen if damage to midbrain. Examples, space occupying lesions, listeriosis, encephalomalacia, etc.

Tenesmus (straining); It may be with or without passage of faeces and is an indication of paralysis of anus or tail head. It is usually seen in rabies (sometime in local meningitis).

Opisthotonos or star gazing; It is the extreme extension of head.

Mydriasis; It is the abnormal dilation of pupil.

Inability to prehend, and chewing feed; It is commonly seen when sensory branch of Vth trigeminal, VII hypoglossal, or VII facial nerve is damaged by trauma, poisoning, or infection. Inability of swallowing (in the absence of physical obstruction) is seen when there is damage

to IX glossopharyngeal nerve as seen in poisoning, listeriosis, or abscess in medulla.

Smooth muscles and endocrine glands; These are controlled by autonomous nervous system.

Dysuria; It is frequent attempts to urinate in the form of dribbling and is seen due to lesion in the sacral, spinal cord, or pelvic nerve and bladder wall.

Urine Incontinence; It is the loss of control on urination and an indication of sphincter paralysis.

Inappropriate sexuality or hypersexuality; It is the protrusion or erection of penis in male and signs of estrus in female.

Hyperphagia or Hypophagia; It is an increase or decrease in hunger.

Anisocoria; It is the unequal diameter of the pupils.

Strabismus (squint) convergent; When eyes turn toward the medial line.

Strabismus divergent; When eyes turn outward. Both of these are seen in trochlear or abducent nerve damage.

Salivation, Without obvious oral lesion.

Drop jaw; It may be seen when lesion is present in the trigeminal nerve or trigeminal motor nucleus.

Facial palsy; It is the facial paralysis and is seen when damage to the medulla, facial nerve, or facial muscles.

Roaring/ snoring; It is seen in laryngeal hemiplegia.

COMMON DRUGS USED IN DISEASES OF NERVOUS SYSTEM

1. Sedatives and Hypnotics Drugs in common use;
Meperidine hydrochloride, @ dose of 1-2 mg/kg, IV.
Morphine hydrochloride, @ dose of 30-60 mg/SA, SC
Pethidine hydrochloride, @ dose of 3-5 mg/kg, SC, IM.
Chloral hydrate, @ dose of 30 gm/LA, IV (6-10 per cent solution), or 30-60 gm/LA, PO.
Phenobarbitone sodium, @ dose of 2-5 mg/kg, IV.

Chlorpromazine hydrochloride (Largectil), @ dose of 0.5 mg/kg, LA, IV, or 1-2 mg/kg, SA, IV.
Phenazocine, @ dose of 1-2 mg/SA, PO.
Bromides (potassium or sodium), @ dose of 10-15 mg/kg, PO. Xylazine (Rompun^{R}), @ dose of 0.1-0.2 mg/kg, IM.
Diazepam (Valium^{R}), @ dose of 0.01-0.4 mg/kg, IM. IV.

2. Stimulants Drugs in common use;
Nalorphine hydrobromide, @ dose of 2-3 mg/kg, SA, IV.
Bemegride sodium, @ dose of 20 mg/kg, IV.
Adrenaline, @ dose of 2-4 mg/LA, IM, SC.
Methyl-amphetamine, @ dose of 100-300 mg/LA and 5-10 mg/SA, SC.
Neostigmine, @ dose of 0.5-1 ml/SA, IV.
Picrotoxin, @ dose of 60 mg/LA, IV. IM.
Nikethamide, @ dose of 10-25 ml/LA, IM. IV and 1-3 ml/SA, IM. IV.

3. Anti-inflammatory or Anti-edema drugs in common use;
Methylprednisolone, @ dose of 1-20 mg/kg, IV.
Dexamethasone, @ dose of 1-4 mg/kg, IM. IV.
Acetylsalicylic acid (Aspirin), @ dose of 10-15 gm/LA, PO.
Phenylbutazone, @ dose of 2-4 mg/kg, IV. PO.
Mannitol (20 per cent solution), @ dose of 0.2-0.5 mg/kg, IV.
Furosemide (Lasix^{R}), @ dose of 1 mg/kg, IM. IV. SC.
Dimethyl sulfoxide (DMSO), @ dose of 1-2 mg/kg, IV.
(see also colic)

NB. Animals suffering from zoonotic diseases (e.g. Rabies) should be handled with great care.

ENCEPHALITIS

Encephalitis is an inflammation of brain (tissues and vessel wall), mainly caused by viruses (sometime bacteria), clinically characterized by starting signs of irritation of nervous activity followed by signs of loss of function of neurons. Encephalomyelitis is an inflammation of tissues and vessels of both brain and spinal cord.

ETIOLOGY

Viral diseases; The most common viral disease causing encephalitis in all animals is the rabies (in Pakistan). Other diseases characterized by encephalitis reported from many countries of the world are pseudorabies and Japanese-B encephalitis (all animals); Equine alphavirus and flavivirus encephalitis (e.g. Western, Eastern, and Venezuelan equine encephalomyelitis), semliki forest virus infection, equine herpes virus-1 infection, and Borna disease (equine); sporadic

bovine encephalomyelitis, bovine malignant catarrh (cattle); ovine encephalomyelitis (louping-ill), Scrapie, and Visna (sheep); caprine viral arthritis-encephalitis; avian encephalomyelitis (epidemic tremor); canine distemper, and infectious canine hepatitis.

Bacterial diseases; The bacterial diseases characterized encephalitis are listeriosis, hemophillosis, and toxoplasmosis.

Parasitic diseases; Babesiosis, occasionally may cause encephalitis small proportion of cattle.

Mycotic infection; Extension of mycotic infection from guttural pouch lesion via internal carotid artery has been reported to cause encephalitis.

Allergic causes; The vaccines containing brain cell suspension (e.g. Anti-rabic vaccine) may occasionally cause encephalitis due to development of antibodies against brain tissues.

PATHOGENESIS

The disease organisms can enter the brain through several ways. For example (i) most viruses have the ability to cross the blood brain barrier directly or indirectly (by causing damage to first vascular endothelium and then carrying over to brain tissues). A synergistic relationship exists between the rickettsiae of tick born fever and virus of louping-ill, as the rickettsia causes damage to vascular endothelium and then the virus of louping-ill disease invade the brain cells.

(ii) Some infectious agents reach the brain through progression up of infection through peripheral nerve trunk, e.g. rabies and pseudo-rabies viruses, and listeriosis organisms.

(iii) Spread of infection from middle or inner ear to brain cells can occur via olfactory nerve or from initial local sinusitis to nearby brain cells by extension of lesion.

Most of the viruses exert their direct effects on cellular elements of the brain causing initially inflammation and edema and later lethal effects (death) on neuron cells. But the virus of bovine malignant catarrh and equine herpes virus-1, and most of the bacterial diseases cause their effects first on vascular endothelium and then on nervous cells, resulting in diffuse type encephalitis. The clinical signs in most diseases are generalized, except in listeriosis in which signs are restricted to head and neck only and it is because of the development of micro-abscesses in pones and medulla.

CLINICAL FINDINGS

There is initial response in the form of irritation, excitement, or hyperactivity of neurons as the expansion of inflammation and edema of brain cells. The clinical signs may include viciousness, mania, frenzy, blind charging, hyperaesthesia, convulsion, and muscle tremor. Followed then the signs of loss of function of nervous activity or paralysis occur due to depression of function or death of neurons. These sings include paresis, knuckling, with spasticity, ataxia, incoordination, abnormal posture and gait, head deviation, walking in circle, and partial or complete paralysis. Depending upon the nature of the causative organisms either of the above signs may predominate. Other signs than nervous system including the signs of toxemia, cardiac, or respiratory involvement may also be present, particularly in bacterial infections.

CLINICAL PATHOLOGY

Serological tests (e.g. CFT, HA, HAI, SNT, and ELISA) may be helpful for the diagnosis of most of the viral diseases mentioned in the etiology. Biopsy material may be taken for isolation and identification of the causative agent, immuno-histochemistry, and histopathology conducive for diagnosis of some of the diseases. Hematological profile, i.e. lymphopenia and neutropenia in early virus diseases, leukocytosis with neutrophilia and left shift in acute bacterial infections may be of diagnostic value.

NECROPSY FINDINGS

Macroscopic appearance of the brain of affected animals is altered, i.e. from generalized congestion to discoloration. The histopathological findings may reveal the infiltration of mononuclear cells and neutrophils, perivascular lymphocytic cuffing, neuronal inflammatory in acute stages of many diseases, or degenerative changes particularly, when paralytic signs predominate.

DIFFERENTIAL DIAGNOSIS

Diseases need rule-out from encephalitis because of having some clinical analogy are: Acute cerebral edema, space occupying lesions in the cranium (i.e. tumor, abscess, Cysts, or hematoma), encephalomalacia, meningitis, avitaminosis-A, hypoglycemia, hypocalcemia, and poisoning with lead, arsenic, mercury, rotenone, or chlorinated hydrocarbons.

A careful monitoring of disease history, clinical signs, and laboratory/ postmortem findings, and therapeutic trials are required for their differentiation from primary encephalitis.

TREATMENT

A general line of treatment of encephalitis may include;

1. Specific treatment should be given for diseases where the cause is known. For example, antibacterials in listeriosis and specific hyper-immune serum for most viral diseases.

2. Symptomatic treatment should includes i). sedative, hypnotic, and anti-inflammatory drugs in cases where irritation signs of nervous involvement predominate, in order to prevent convulsions and hurting to self or to others, and ii). use of stimulant drugs in cases where paralytic signs due to depression nervous activity predominate.

3. Supportive therapy consists on the use of vitamins, minerals, electrolytes. A good plane of nutrition and nursing care of sick animals should be carried out during the course of disease or convalescent period.

NB: Medical treatment should not be recommended for diseases of zoonotic significance. For other diseases associated with nervous signs, a treatment trials may be carried out for not more than 6 weeks and if there is no clinical response during this period, slaughter or euthanasia may be suggested.

ENCEPHALOMALACIA

It is the softening of brain tissues resulting from degenerative changes (demyelination and necrosis) in brain tissues, and characterized clinically by signs of loss of function.
Leuko-encephalomalacia is the softening of white matter and
Polio-encephalomalacia is the softening of grey matter.

ETIOLOGY

A variety of agents are known to be associated with the etiology of encephalomalacia. Some of those includes;

a). Deficiency of vitamins; Thiamine deficiency and vitamin-E are important known causes of polio-encephalomalacia in ruminants and poultry, respectively.

b). Bacterial/ Fungal toxins; Examples are toxins of Clostridium perfringens type-D (focal symmetrical encephalomalacia), Clostridium botulinum, and fungus Fusarium moniliforme (moldy corn disease in horses) are known to cause encephalomalacia.

). Chemical poisoning; Examples include chronic intake of nanganese, arsenic, alcohols, mercurial salts, lead, repeated low losages of cyanide salts, and excessive sulfate or urea feeding with nolasses in ruminants (which can inhibit the use of various enzymes nd co-enzymes in the body). A high level of ammonia (in horses) is nown to cause severe liver damage and encephalomalacia. Lactic cidemia (grain over-load) in sheep and goats causes deficiency of itamin-B1 and thus may lead to the development of the disease.

). Pants poisoning; Examples are Yellow star thistle, (causing hewing disease in horses), Bracken-fern, and Horse-tail poisoning an cause encephalomalacia indirectly by causing thiamine deficiency.

). Deficiency of minerals; Examples include copper and cobalt leficiencies can cause enzootic ataxia (encephalomalacia) in young ambs and cattle, respectively.

). Local ischaemia; Ischaemia at any part of brain (e.g. by space ccupying lesions) may lead to degenerative changes at the site and ience development of signs of loss of nervous function.

). Certain drugs; Repeated and large doses of certain drugs in eterinary use, e.g. piperazine, levamisole, thiabendazole, and icepromazine, produce certain substrates for thiaminase during their netabolism which breakdown thiamine and thus cause its deficiency vhich may lead to encephalomalacia.

). Viral diseases; Certain viral diseases are known to cause legenerative changes in the brain tissues of animals, these include Scrapie in sheep and Bovine spongiform encephalopathy (BSE) in certain breeds of cattle.

PATHOGENESIS

The pathogenesis of encephalomalacia is not very clear. One ypothesis illustrates the development of disease as under;

Thiamine acts as a co-enzyme for transketolase, pyruvate and a-etoglutarate dehydrogenases, which are utilized in carbohydrate CHO-) metabolism. Its direct or indirect deficiency will cause lecrease activity of these enzymes and thus will result in increase in lood concentration of pyruvic, lactic, and a-ketoglutaric acids. Transketolase is found in erythrocytes and brain cells and its lepletion will limit the rate of glucose metabolism and utilization by he brain tissues. Thus the CHO- metabolism dependent neurons and glial cells cannot utilize CHO- for their energy requirements. This can ead to first depression of neuronal functions and later on

development of degenerative changes. As the degree of neuronal degeneration (necrosis) expands, this may lead to the swelling and proliferation of capillary endothelium and infiltration of macrophages in cerebral cortex. The combined effects of these changes can cause increase in intracranial pressure, and intra-neuronal edema, laminar cortical necrosis, and polio-encephalomalacia.

The polio-encephalomalacia also appears to develop (in some cases) as a complication of acute edematous swelling of brain and cortical ischaemia. Defective myelination taking place in some diseases due to interference with phospholipid formation may be the other reason of development of encephalomalacia. The predominant signs in all cases of encephalomalacia are depression of nervous function, except in polio-encephalomalacia where the initial signs may be of irritation types.

CLINICAL FINDINGS

The predominant signs of encephalomalacia are of loss of nervous function, developing slowly over a long period, and includes depression, dullness, somnolence, blindness, incoordination, ataxia, repetitive chewing, head pressing, circling, and terminally coma. If untreated the animal may die due to respiratory failure.

In polio-encephalomalacia, the early specific signs may be (of irritation type) muscle tremor, opisthotonos, nystagmus, hyperaesthesia, tetanic convulsion, followed by the signs of loss of nervous activity as mentioned above. The pupillary eye reflex may be normal. The development of paralytic signs once develop usually remain lasting.

CLINICAL PATHOLOGY

In poisoning (plants or chemical) a careful examination of gut contents may be necessary for their identification. There is increase in concentration of blood pyruvate and lactate along with above mentioned clinical signs are suggestive of encephalomalacia. A high level of thiaminase can be detected in rumen and faeces of affected animals is indicative of thiamin deficiency.

NECROPSY FINDINGS

The affected parts of brain will be soft to touch and easy to break. Histopathologic lesions varies from cortical swelling, softening, cavitation, to laminar necrosis of varying degree at various parts of brain. Evidences of intra and intercellular edema and neuronal necrosis can also be noted.

DIFFERENTIAL DIAGNOSIS

The diseases require rule-out from encephalomalacia are;
i. Space occupying lesions of brain i.e. abscesses, tumors, cysts, or hematoma in the vicinity of brain and ii. Hydrocephalus. Both of these conditions cause pressure ischaemia and necrosis to underlying nervous tissues. A sudden development of permanent signs of loss of function are characteristic of these diseases. In addition, these diseases can be identified by radiographic examination.

TREATMENT

There is no effective and specific treatment available particularly for paralytic signs seen in advanced stages of the disease. The treatment commonly recommended includes the use of;

1. Injection of thiamine, @ dose of 10-20 mg/kg, IM or SC, per day in three divided doses, for 7-10 days, or vitamin-B complex with equivalent quantity of thiamin (in ruminants) and vitamin-E (in poultry) for rapid activation of deficient enzyme complexes.

Early administration of thiamine may ensure rapid activation and utilization of enzyme complexes for carbohydrate utilization. Also, since carbohydrate metabolism is impaired during the disease, the use of dextrose is contra-indicated, except during convalescent period of the disease.

2. A good nutritive value diet contain Brewer's yeast may be used for animals suffering from encephalomalacia and kept long for slaughter purpose.

3. Symptomatic treatment includes the use of i. Tranquilizers in convulsion during early stages of disease, ii. Diuretics in cerebral edema, and iii. Stimulants in animals exhibiting paralytic type signs.

4. Supplementation of essential minerals particularly of copper and cobalt in feed and stoppage the use of feed containing toxic chemicals may serve to prevent the condition.

MENINGITIS
Syn: MENINGOENCEPHALITIS or SUPPURATIVE MENINGITIS

Meningitis is an inflammation of meninges, mostly caused by bacteria, and characterized clinically by fever, cutaneous hyperaesthesia, and rigidity of muscles.

Meninges are membranes covering the brain and spinal cord. It caries blood vessels which nourishes the brain tissues and inner layer of the skull. The outer most layer is called as dura-matter, middle one as arachnoid, and inner most as piamatter. Inflammation of dura-matter and piamatter are called as pachymeningitis and leptomeningitis, respectively.

ETIOLOGY

The causative organisms invade the meninges either through blood or extension of lesions from adjacent structures/infection (e.g. compound skull fracture, penetrating skull wounds, improper trephination holes, osteomyelitis, thermal cauterization of horns, frontal sinusitis, horn infection, otitis media or interna, etc.). The common diseases/ organisms associated with meningitis are;

Bacteria; The bacterial diseases reported to cause meningitis are strangles and glanders (horses), listeriosis (all animals), pasteurellosis, both pneumonic and septicemic types (cattle), colibacillosis (young animals), and tuberculosis, leptospirosis, and septicemic conditions originating in other organs (e.g. arthritis). The other bacteria isolated from meningitis are Staph. aureus, St. epidermidis, and St. uberis. Ehrlichiosis (a rickettsial disease) can also cause meningitis in dogs.

Virus; The viral diseases associated with meningitis are bovine malignant catarrh, sporadic bovine encephalomyelitis, scrapie (sheep), rabies, canine distemper, and feline infectious peritonitis.

Fungus; The fungi Cryptococcoses neoformis is a common isolate from granulomatous type lesions associated with meningitis.

Parasites; Meningitis may develop by migrating larvae of certain nematodes and warble flies through skull and spinal cord. Larvae of Strongyle spp. (horses), Hypoderma bovis, bladder stage of Taenia coenurosis (sheep, goats, and cattle) are few examples.

Sporadic cases of meningitis like disease have been seen in acute lead or copper poisoning in cattle, whether causing true meningitis or not, is not clear.

PATHOGENESIS

Inflammation of meninges will initially cause local inflammatory response and interference of blood supply to brain tissues and spinal cord. In later stages the inflammatory response may reach up to nervous tissues resulting in superficial encephalitis. Inflammation around nerve trunk can also occurs as it passes across the

subarachnoid space. Advanced stages of meningitis may end up with fatal choroiditis and ependymitis with exudation into cerebro-spinal fluid. The irritation signs produced are thus due to combined affect of inflammation at central and peripheral nervous systems. In bacterial meningitis, additional signs of systemic involvement and toxemia can also be noted.

CLINICAL FINDINGS

In acute cases of cerebral meningitis, a sudden development of fever, anorexia, stiff neck, hyperaesthesia to light or touch, and other nervous signs including trismus, convulsion, opisthotonos, rigidity of back, with muscle tremor, and stumbling are common. Slight percussion of skin may result in spasmodic contractions of the body or limbs. Disturbance of consciousness, in the form of excitement and mania in early stages, followed by drowsiness, propulsive walking, facial paralysis, blindness, nystagmus, coma, and other signs of loss of function may be noted in later stages of the disease.

In spinal meningitis, localized signs such as, local muscle spasm, tremor, hyperaesthesia, or rigidity of affected muscles, followed by paralysis may be present. Abnormalities in heart beat, respiration, urination, and defecation can also be present.

CLINICAL PATHOLOGY

Cerebrospinal fluid (C.S.F.) collected from atlanto-occipital or from lumbo-sacral junction, may be used for an aid in diagnosis. The C.S.F. when withdrawn may indicates a high pressure, from turbid to reddish or opaque in color, can clot, contains high protein contents (>200 mg/dl) and cell counts (neutrophils in bacterial or lymphocytes in viral infections), and will also contain causative bacteria that can easily be checked by Gram's staining or cultural examination.

NECROPSY FINDINGS

Hyperaemia, hemorrhages, and thickening of meninges at macroscopic and infiltration of neutrophils and lymphocytes in nervous tissues at microscopic examination are important evidences of meningitis. The C.S.F. will be changed both in quality and quantity. The underlying nervous tissues will also have inflammatory changes. Evidences of suppurative lesions in organs other than brain or clinical signs of a primary disease may be identified.

DIFFERENTIAL DIAGNOSIS

Hyperaesthesia, rigidity of muscles, and fever followed by blindness and severe depression in later stages are common clinical evidences of meningitis. However, it requires to be ruled-out from cerebral edema,

encephalitis, spinal cord compression, hypomagnesemia, and hypoglycemia. Examination of C.S.F. may be a useful tool for differentiation meningitis from other disease of nervous system.

TREATMENT

Early recognition of the disease and administration of specific treatment is essential for acceptable recovery from meningitis.

1. Parenteral execution of specific antibacterials, particularly following sensitivity test should be recommended. However, one may select an antibiotic having broad spectrum action for use in routine cases. Some antibiotics may attain higher plasma concentration, but may not cross the blood-brain barrier. To ensure the antibacterial efficacy, antibiotic concentration should range from 10-30 times the minimum inhibitory concentration of the infecting bacteria.

Chloramphenicol, @ dose of 4-10 mg/kg, IM, is the antibiotic of choice as it can readily be diffused in C.S.F., but its efficacy against Gram negative bacteria is limited. Penicillin, @ dose of 250,000 i.u./kg daily, Oxytetracycline, @ dose of 5-15 mg/kg, IM or IV, Gentamicin, @ dose of 1 mg/kg, SA, and 0.05 mg/kg, LA, Cephalosporins, @ dose of 50 mg/kg and Trimathoprim plus sulfonamide, @ dose of 15-20 mg/kg, IV, can also be used. The treatment needs to be given at least for 10-14 days or to be continued for 4-5 days post clinical recovery.

2. Direct intra-thecal or Intra-cerebromedullary cistern injection using chloramphenicol, kanamycin, or gentamicin, diluted in normal saline, can increase the rate of recovery. For this purpose CSF should be withdrawn equal to the volume of fluid (drug) injected.

3. Symptomatic treatment includes the use of tranquilizers, sedatives, or hypnotics if the irritation signs predominate and stimulants, in cases having paralytic type signs.

4. Supportive therapy, using large doses of IV fluids with electrolytes, particularly in long standing cases, and in those animals unable to drink, should be selected. A good nutritive value diet/feed may also have beneficial effects.

Prognosis: In long standing cases, usually grave.

TRAUMATIC INJURY TO BRAIN
Syn: CEREBRAL or CRANIAL TRAUMA

Traumatic injury to brain occurs by many ways and has variable effects, depending upon the extent of injury and area of brain damaged, clinically characterized by initial nervous shock, followed by death, or complete recovery or with or without perpetual residual signs.

ETIOLOGY

Traumatic injury to brain is relatively common in urban areas. The common causes of injury to brain are as under;

i) Direct trauma to brain tissue may occur either by straight head collusion usually result from automobile accidents, severe blow to the skull, falling over backward while jumping, or over-burning of tissues with hot iron during dehorning operation.

ii) Nervous tissues damage can also develop by migrating nematode larvae of Setaria sp. & Parelaphostronylus tenuis (sheep & goats), Strongylus vulgaris (horses), and occasionally larvae of Toxocara canis, Hypoderma bovis, & Oestrus ovis.

iii) Space occupying lesions in the skull or embolic obstruction in blood supply will cause to develop local ischemia and brain tissue damage.

PATHOGENESIS

The initial response to any trauma or blow is the development of nervous shock with or without structural damage and hemorrhage. There could be either inflammation or herniation of the brain tissues which is associated with serials of dysfunctions of cranial nerves and disturbance of consciousness. There is development of increased intra cranial pressure due to hematoma, vasogenic edema, brain tissue swelling, or depression fracture of cranium following head injury causing local pressure on underlying nervous tissues and leading to local ischemia, hypoxemia, followed by degenerative and necrotic changes in the underlying brain tissues.

Nematode larval migration through nervous tissue can result in local tissue damage at any site of brain and thus the clinical signs will depend upon the part of brain area involved. In addition, severe cellular and/or allergic reaction can occur against the toxins produced by the larvae or tissue damage and death of larvae within the body that may behave like a foreign protein (allergen).

CLINICAL FINDINGS

The manifestation of head trauma are diverse and may be complicated by injury to other organ systems. The initial signs of brain injury are the development of nervous shock which will be evident by sudden falling, unconsciousness, with or without clonic convulsion, dilation of pupil, absence of eye preservation and pupillary light reflexes, slow and irregular respiration, occasionally epistaxis or ear bleeding, followed by sudden death or recovery from initial nervous shock. Recovered animals may show residual nervous signs, such as hemiplegia (mid-brain injury) or blindness (optic cortex injury) which could be temporary or permanent depending upon the extent of damage to these structures.

In equine the common nervous signs (reported) due to migrating nematode larvae are incoordination, head-pressing, clonic convulsion, blindness, and gradual or sudden onset of paralysis of cranial nerve(s).

CLINICAL PATHOLOGY

Presence of RBCs or heme-pigments in C.S.F. without evidence of bacterial infection is an good indication of pre-existing trauma and hemorrhage. The presence of eosinophilia at CBC studies can be supportive of parasitic invasion. Radiographic examination may be useful for the identification of skull fracture.

NECROPSY FINDINGS

Evidences of fractured skull, gross hemorrhages, or hematoma along with history of accident may be diagnostic of brain injury. Damage to brain tissues caused by migrating larvae can only be identified by histopathology.

DIFFERENTIAL DIAGNOSIS

A history of trauma with clinical signs mentioned above are diagnostic of brain injury. However, space occupying lesions and acute chemical poisoning (Lead, arsenic, chlorinated hydrocarbon, etc.) need careful clinical and laboratory tests to rule-out them. Clinical diagnosis of nematodiasis is difficult.

TREATMENT

Early recognition and treatment directed toward the increased CSF pressure, depressed fracture, edema, and hematoma as quickly as possible, are important for complete recovery. General principles for cerebral trauma should include the establishment of proper respiration, use of osmotic diuretics, control of irritation type signs particularly causing self inflicting injuries, and protection from decubitus. Symptomatic and supportive treatment inclu[illegible]e,

1. Administration of corticosteroids (dexamethasone, @ dose of 1-2 ng/kg, repeated 6 hourly, and 20 per cent solution of mannitol (@ dose of 0.25 gm/gm, over 30 minutes, IV) may be recommended for reducing nflammation and vasogenic edema. A 40 per cent solution of DMSO n normal saline may be useful in reducing acute inflammatory eaction in the brain tissues.

2. Nervous shock should be considered as an emergency and may be reated using stimulant type drugs. Parenteral coagulants needs to be mployed in suspected cases of internal hemorrhage (see lemorrhage).

. For the correction of fractured bone or pressure inducing parts of roken skull bone(s), surgical manoeuver may be required. imultaneously, pressure inducing hematoma within the cranial ıvity should be removed, if possible.

Good nursing care in the form of frequent turning in paralysed ıimals and good layer of bedding should be employed particularly ıring early period of nervous shock, in order to avoid severe mplications as decubitus.

In cases of animals which remain unconscious for >3-4 hours, ognosis is usually unfavorable, so slaughter or euthanasia may be nsidered to reduce the miseries.

Provide complete rest and avoid exercise of all kinds in cases of any ad injury unless emerge uncomplicated.

TRAUMATIC INJURY TO THE SPINAL CORD

auma to the spinal cord occurs commonly due to accidental fracture the vertebral column and is characterized clinically by sudden velopment of spinal shock and signs of loss of function of various dy parts.

'IOLOGY

slocation or fracture of vertebral column and intervertebral disc otrusion are common cause of spinal injury in animals. These may ur while falling on slippery floor, roadway accidents, butting or king by other animals, jumping or galloping (horses), casting, unting on other animals especially with nutritional osteodystrophy.

Spinal injuries can also occur due to penetrating spinal ... parasitic nematodiasis, or development of space occupying lesion... abscess, hematoma, or neoplasia in the vicinity of the spinal cord.

PATHOGENESIS

Either the direct local trauma to the spinal cord, compression displaced bone, hemorrhage, hematoma, or local edema, all responsible of the development of the clinical disease. At first, ... development of spinal shock, affecting different nerves, at one or ... sides of the body, resulting in reduction or complete termination ... neuronal transmission. Following then there may or may not be ... persistence of residual signs which are dependent upon the resorp... of edema and hematoma, myelin degradation, and demyelination. ... cases of structural damage to the spinal cord, the clinical signs will ... of permanent nature owing to the poor regeneration capacity ... nervous cells. The common sites of spinal fracture are C1, T12, and ... in horses and C2-C4, T10-T13, and L3-L6 vertebrae in ruminants.

CLINICAL SIGNS

There will be development of initial spinal shock in the form flac... paralysis in area caudal to the injured site with concurrent local fal... blood pressure because of vasodilation. These signs may disappear ... the effect of spinal shock passes away, followed by retreating ... persistent of residual signs. These signs may includes flaccid paral... at the injured area, spastic paralysis caudal to the trauma, ... hyperaesthesia in a girdle like zone at the cranial edge of the lesi... due to irritation of sensory fibers by local inflammation and edema. The variation in clinical signs may depend upon the site of trauma... injury, severity of spinal cord compression, and involvement of specific anatomic tracts. For example;

If cervical spine are involved, there will be stiffness at neck ... recumbency due to spastic paralysis of all limbs, and pain at palpation ... in acute cases. Animals with mild lesions may develop ataxia, crossing over, knuckling, or excessive body sway. Muscle wasting or atrophy ... long standing cases are common signs.

In thoracic spines involvement, the gait abnormalities are limited to the caudal limbs, while forelimbs remain normal. Affected animals ... on their side or assume dog sitting posture with hind-limbs ... extended. In severe cases, there could be hypertonicity of forelimbs and flaccidity of hind limbs.

In cases of damage to lumber spines, the defects in posture and ... are similar to thoracic spinal cord lesions. The forelimbs are usually normal, but hind limbs ...

sensitivity and withdrawal reflexes, or complete flaccidity. The urinary bladder may be distended but the sphincter tone may be normal.

Involvement of sacrococcygeal spine have clinical picture similar to that of lumber spine, but there may be bladder distension with hypotonicity and decrease or absent of anal tone. A persistence of tenesmus and dysuria are the indications of paralysis of rectum and bladder, respectively.

CLINICAL PATHOLOGY

Plain radiographs are the most conclusive methods of diagnosis for spinal fracture. Examination of spinal fluid (S.F.) collected through lumbo-sacral puncture may be useful for making diagnosis. In acute cases a diffuse blood contamination with high R.B.Cs, normal to high W.B.Cs., and a high protein concentration in the S.F. may be diagnostic. In chronic cases S.F. may contains normal W.B.Cs., normal to increase R.B.Cs., but high concentration of protein.

DIFFERENTIAL DIAGNOSIS

A history of trauma to the vertebral column, sudden development of spinal shock, and other features of spinal cord injury are diagnostic. But it needs rule-out from meningo-encephalitis, azoturia, acute ruminal impaction, parturient paresis, downer's cow syndrome, severe toxemia, and acute Coli-form mastitis.

TREATMENT

The treatment of spinal injury may include the followings;

1. Surgical correction of spinal fracture (if possible).

2. A good nursing care, use of periodic slings (horses) or frequent turning after every 3 hours periods, thick layer of bedding in recumbent animals, use of catheter for bladder evacuation, and manual removal of faeces if rectal paralysis are recommended.

3. The use of anti-inflammatory drugs particularly DMSO 40 per cent solution in saline, @ dose of 0.25-1 gm/kg, IV, or dexamethasone, @ dose of 0.1-0.2 mg/kg, 4 times a day, for 4 days, is recommended.

4. Use of tranquilizers, analgesics, or non-steroid anti-inflammatory drugs may be good to reduce pain, except in animals with ataxia.

5. Use of nervous tissue tonics, particularly vitamin-B1 and other vitamins may aid in early recovery.

6. Local message with turpentine liniment, iodine ointment, or warm application may help to hasten recovery in chronic cases.

7. Recumbency beyond 48 hours period is likely to result in wide spreading necrosis of posterior muscles of the thigh and other related complications. The recovery in those cases is rare.

Prognosis: In long standing cases with nervous signs of loss of functions the prognosis may be considered as "Guarded".

PERIPHERAL NERVE DISORDER (PND)

The PND are usually traumatically induced, but faulty IM injections, abscesses, tumors, or parasitic invasion of the nerve may also occur. Examples are;

a) Supra-scapular Nerve; Damage to supra-scapular nerve usually results in paralysis of the infra-spinatus and supra-spinatus muscles. The early clinical signs are slight outward bowing of the scapulo-humoral joint as weight bearing. Neurogenic atrophy develops after several months and the scapular spine becomes prominent (and it is called as Sweeny).

b) Brachial Plexus; Damage to brachial nerve results in combination of dysfunction of biceps, coracobrachialis, pectoral, sub-scapularis, and triceps muscles.

Motor Deficits; Lesions in brachial plexus results in complete flaccidity of the fore limb; i. Loss of pectoral nerve function results in abduction of elbow, ii. Sub-scapularis muscle paralysis results in dropped shoulder, and iii. Avulsion of brachial plexus results in complete desensitization of the entire fore limbs.

c) Radial Nerve; It is a motor nerve to extensor muscles of fore limbs. High radial nerve paralysis is characterized by dropped elbow, failure of limb protraction with scuffing of toe. The foot is knuckled over at rest. The animal is unable to bear weight on the affected leg. Distal radial nerve lesion is usually associated with signs of knuckling of the carpus, fetlock, and pastern joints. The animal can support some weight.

d) Femoral Nerve; The femoral nerve is distributed to the quadriceps femoris muscle and skin of the rear limb (medial thigh to coronet). The signs of femoral nerve damage are related to inability to extend and fix stifle (and hock). This results in collapse of limb at weight bearing and constant flexion of distal digital joints. Chronic lesions of

femoral nerve results in atrophy of quadriceps femoris and posterior gluteal muscles.

e) Sciatic Nerve; The sciatic nerve innervates most of the muscles of near limb, i.e. Extensor muscles of hip, flexor muscles of stifle. At rest the affected limb hangs behind the animal. The stifle is dropped and extended. The affected foot is constantly knuckled. Chronic cases ends up atrophy of caudal thigh muscles, distal stifle, and of entire limb.

f) Peroneal Nerve; It is a branch of sciatic never, distributed to flexor muscles of the hock joint and extensor muscles of the digits. Neurologic defects result in hyper extended hock joint and flexion of fetlock and pastern. Many animals knuckle at rest but the toe does not drag on the ground.

g) Obturator Nerve; Damage to obturator never is rare. Knuckling and inability to support weight is common in obturator nerve paralysis. Clinical evidences are hopping gait at running (common) and sternal recumbency (in few cases).

) Fascial Nerve; Fascial nerve paralysis affects masseter muscles unctioning and fascial paralysis, analgesia, or facial palsy.

) Hypoglossal Nerve; Paralysis of this never will lead to flaccidity of ongue and later on, tongue atrophy.

Optic Nerve; Paralysis of it will lead to permanent blindness.

Occulo-motor Nerve; Paralysis of this nerve will lead to mydriasis d sluggish pupillary response.

Trochlear Nerve; Paralysis of trochlear nerve will cause dorso-edial strabismus.

) Trigeminal Nerve; Paralysis of this nerve will cause dropped jaw.

Auditory Nerve; Paralysis of this will cause head tilt and stagmus.

Caudal Rectal Nerve; Paralysis of caudal rectal nerve will cause s of control of anal sphincter, bladder, and rectum.

CEREBROSPINAL (CSF) FLUID AND ITS ANALYSIS

The CSF is analyzed in patients which have signs of central nervous system disorder for which the type of disease process cannot be otherwise determined. Physical and radiological examinations and laboratory analysis of other body fluids should be studied first. The CSF analysis may reveal the nature of the basic processes causing the clinical signs.

The CSF is produced by the choroid plexus within the ventricular system of the brain and flows through the ventricles and subarachnoid space of the brain surface to exit into the dorsal longitudinal sinus. Therefore, the CSF reflects brain surface events.

The CSF may be obstructed in its route to the venous system by space occupying masses or cellular infiltrates in the ventricular system or meninges. Such an obstruction may cause local or generalized increase in CSF pressure.

The CSF is usually collected from the cerebro-medullary cistern subjacent to the foramen magnum. Under general anesthesia a sterile draped field is prepared. A point on the midline of the neck, half the distance between the external occipital protuberance and a line connecting the cranial edges of the wings of the atlas is identified for cisternal puncture. A 20 gauge short bevel spinal needle (with a stilette) is advanced until the dura mater is punctured. The stilette is removed and if CSF flows a standpipe Manometer is attached to the needle hub to measure the CSF pressure. One to 2 ml of fluid are then collected with a syringe.

For routine CSF analysis the following measurements should be made: cisternal pressure, total protein content, total white cell count, red cell count, and cytology.

1) The CSF should have a pressure of 60-180 mm water. Elevation of CSF pressure indicates brain swelling and/or CSF outflow obstruction which is usually due to brain tumors. Contrary to prevalent teaching, dogs with congenital hydrocephalus almost always have normal CSF pressure.

2) The total protein should be less than 25 mg/dl. The protein content in CSF may be increased by almost any process including tumors, inflammatory processes, or trauma. It is normal in toxic or metabolic nervous disorders.

3) There should be fewer than 5 WBCs. An increase WBC count in the CSF indicates inflammatory lesions of or near the brain surface. The nucleated cells should be lymphocytes or arachnoidal macrophages, PMN leukocytes are abnormal. Some infectious diseases have characteristic features such as FIP meningitis which often has an abundance of PMN leukocytes, protein concentrations over 300 mg/dl, and an abundance of fibrin. Contrarily, Canine distemper encephalitis may produce mild increase in protein and WBC count.

4) There should be no erythrocyte in the normal CSF. Presence of red blood cells in the CSF are usually due to trauma associated with cisternal puncture, cranial trauma, or bleeding tumors. Macrophages containing hemosiderin indicate that bleeding occurred before the tap.

Specific studies of CSF can be made for diagnosis of diseases. Examples, creatine kinase concentration which may parallel the degree of brain necrosis in head injury, quantitative determination of CSF immunoglobulin concentrations as an aid in the diagnosis of canine distemper encephalitis, and the use of special centrifuge to examine malignant cells in CSF cytology even when the cell count is normal. Infectious organisms may be found in inflammatory cells or free in the fluid.

CHAPTER-10

DISEASES OF SKIN

Skin is the largest body organ and principal structure of communication between the animal and its environment. It consists of a variety of cellular and tissue components. The skin perform diversity of functions and among those the major is the protection of body from absorption and penetration of noxious agents. Diseases of skin may be either primary or secondary in origin. For the identification of the common skin disease a list of common manifestations is described.

Papule (Pimble); A papule is a circumscribed, small, solid, elevated lesion of skin, up to 1 cm in diameter, which do not extend beneath dermis.

Nodule; Nodule is a circumscribed, large, solid elevation, greater than 1 cm in diameter on skin which does not deform at palpation. It involves deeper layers of the skin. It could be either inflammatory or neoplastic origin.

Pustule; It is a small fluctuating, circumscribed, elevated, accumulation of pus (containing inflammatory cells and exudate, causative bacteria, and their necrotic debris), up to the diameter of 1 cm. and involve only superficial layers of skin. It is mostly associated with infectious diseases, e.g. Contagious acne and Pox(es), etc. A pustule could be a sterile elevation containing pus.

Vesicle; A vesicle is a small, fluid filled, acellular, circumscribed, elevated lesion of skin, up to 1 cm in diameter, and which contains serum or lymph.

Blister (Bleb or Bulla); A blister is like a vesicle but of greater diameter than 1 cm.

Cellulitis; It is a severe, deep, and discrete suppurative process of skin in which a poorly defined area of infection tends to dissect through tissue planes.

Folliculitis; It is an inflammation of hair follicles usually involving secondary bacterial infection.

Furunculosis; When inflammation process of hair follicle ruptures and extends into the surrounding dermis and subcutis, it will be called as furunculosis.

Impetigo; It is less severe inflammation of superficial layers of skin, without involving the hair follicles.

Wheel; It is a circumscribed area of swelling caused by local edema and erythema as seen in urticaria/allergies.

Spongiosis; It is an intercellular edema of epidermis.

Scab; It is dried form of exudate and other inflammatory products collected at latter stages on any skin wound/lesion.

Crust; Crust is dried exudate that accumulate and adhere to the skin surface and hair.

Hyperkeratosis; It is dry excessive exfoliation and accumulation of fragments of the horny layer of skin (stratum corneum).

Parakeratosis; It is an excessive cornification of superficial layer of skin in which the nucleus of cells are retained.

Orthokeratosis; It is excessive cornification of cells of stratum corneum without nucleus retention.

Excoriation; It is superficial discontinuity on skin surface usually caused by mechanical trauma.

Fissure; Fissure is a linear crack on skin surfaces which penetrate more deeply usually up to the subcutaneous tissues.

Ulcer; A break in the continuity of epidermis, i.e. an area with complete loss of epidermis and sometime part of underlying dermis with exposure of underlying dermis tissues. An ulcer often heals with scarring that is caused by destruction of dermal collagen.

Erosion; is a an cutaneous defect with irregular boundary, resulting from a partial loss of epidermis and which does not penetrate the dermis. Because an erosion does not involve the dermis, it usually heals without leaving a scar.

Alopecia; It is the loss of hair from the area where hair are normally present. It could be cicatricial or non-cicatricial. The cicatricial alopecia is associated with loss of hair with their follicles and is usually permanent. In the non-cicatricial hair loss, the hair follicles are retained with potential of re-growth of hair.

Hyper-pigmentation; It is an excessive tissue deposition of melanin pigment. Whereas, Hypo-pigmentation is less than normal pigmentation, which could be either congenital or acquired.

Leukoderma (acroderma or hypomelanosis); It is a total and acquired loss of melanin pigment from the skin. Albinism is a congenital lack of pigment in skin and other tissues.

Leukotrichia (achromotrichia); It is an acquired loss of pigment form the hair.

Pediculosis; It is presence of lices or other ectoparasites that are generally host specific.

Pruritus (itching); It is an unpleasant sensation that provokes the desire to scratch. It could be either physiological or pathological. The physiologic itch refers to a sharp, local, and well defined pruritic sensation. Pathological itch is a less well defined, intense, cutaneous discomfort that may elicit vigorous desire of scratching. A variety of primary or secondary local skin disorders or many systemic diseases are associated with pruritus.

DERMATITIS

It is simply an inflammation of skin (dermis and epidermis), caused by direct or indirect causes involving a wide variety of infectious or non-infectious agents. The infectious causes of dermatitis are as under.

A. BACTERIAL DERMATITIS
Syn: PYODERMA, ACNE, OR PYOGENIC DERMATITIS

A variety of bacteria have been known to cause pyoderma, which may be primary or secondary, and superficial or deep. Some of them are;

1. Dermatophilus congolenses causing dermatitis commonly known as Streptothricosis. It is a common disease of cattle particularly in Africa, and characterized by exudation and curst formation on the skin.

2. Staphylococcus aureus (coagulase positive) and Staph. epidermis (coagulase negative) are known to cause Contagious acne, which is characterized by the development of a pustular type dermatitis. Other species of Staph. and Streptococci (both hemolytic and non hemolytic

types), and Proteus vulgaris have also been isolated from pyoderma lesions.

2. Impetigo in cattle is also a Staphylococcal skin infection. It is characterized by the development of first vesicles, later pustules, and finally scab formation on the superficial layer of skin without involving hair follicle.

4. Corynebacterium pseudotuberculosis causes nodular and pustular type skin lesions, particularly in horses.

5. Skin form tuberculosis may cause single or multiple nodular type skin lesions, from a pea to tennis ball size, developing at the limbs, neck, or trunk of all animals.

6. Glanders (equine farcy) is a specific contagious disease of equine which is characterized by the development of nodular and/or ulcerative type chronic skin lesions, particularly on the hind legs.

DIAGNOSIS

The cytological examination of skin is of great value in diagnosis, particularly when dealing with pustules, vesicles, nodules, tumors, or other types of swellings. It can provide evidences for the presence or absence of infectious organisms and associated cell types, either inflammatory or neoplastic. Bacterial culture should be carried out for the identification of the organisms and their antibiotic sensitivity. Histopathology of biopsy specimen helps to identify the type of tissue response and so may support the diagnosis.

TREATMENT

1. First remove or scrub the dust, loose scabs, or crusts, clip all hair, and wash to clean the infected area using any mild non irritant antiseptic.

2. Apply any formulation containing antiseptics/antibiotics. The choice of formulation depends upon the lesion type. For acute inflammatory skin reaction use wet and astringent type antiseptics. For chronic skin inflammation and dry wounds use ointment based antiseptics. Powders formulations are use only in weeping (wet) wounds.

3. Do not use irritant drugs particularly at fresh skin wounds, which helps to avoid further self-inflicting injuries.

4. For superficial skin infection, topical antiseptics may be good enough, but for deep seated skin infections, parenteral administration of antibacterials should also be used

A formulation of an astringent antiseptic (lotion) contains the followings;

Salicylic acid, 8 parts, Tannic acid, 8 parts, Alcohol (70 per cent), 100 parts.

A list of common antiseptics and their concentration used in most veterinary hospitals is provided.

Solutions/lotions;
Gentian violet 0.01-0.05 per cent alcoholic solution,
Acriflavine 0.01-0.001 per cent (alcoholic or water solution),
Mercurochrome 0.05 per cent solution (water based),
Tincture of Iodine,
Hexachlorophene 3 per cent solution,
Boric acid 4 per cent solution,
Potassium permeganate 0.001-0.004 per cent solution, and
Antibiotic bases solutions.

Ointments; Antiseptic ointments mostly contain either single or combination of antibiotics and/or sulfonamides. Some common ointments used in veterinary hospitals are;

Ointments of Zinc oxide (12 per cent),
Salicylic acid (5 per cent),
Acriflavine (0.1 per cent),
Sulfur (10 per cent),
Zinc sulfate or gluconate ointment (4-8 per cent),
Iodine, and
Copper sulfate (4 per cent), and
Ointment containing a variety of combination of antibiotics or sulfonamides.

NB: Most of the moisturizers and emollients contain either glycerol esters, glycerine, lanolin, oils, fatty alcohols, propylene glycol, lactic acid, or urea. Refined Coal tar, sulfur, and salicylic acid (also Benzoyl peroxide are commonly used in many antiseborrheic shampoos.

BIPP (is a very good antiseptic paste and fly repellent). It contains Bismuth subnitras, 2 parts, Iodoform, 1 part, Liq. paraffin Q.S. to make paste.

Powders; Antiseptic powders

boric acid. Some dusting powder formulation may contain any of the available antibiotics.

A formula for dusting Powder contains copper sulfate, boric acid, zinc oxide, sulfanilamide, and salicylic acid in equal parts. Iodoform may also be added as fly replant.

A formula for keratolytic gel containing salicylic acid (6.6 gm), urea 5.0 gm), sodium lactate (5.0 gm), sulfur(5.0 gm), propylene glycol gel QS to make up to 100 gm of gel.

B. VIRAL DERMATITIS

Some of the common viral diseases of farm animals associated with skin lesion are as under;

1. Pox(es), in all animals is characterized by the development of first erythema (roseola stage), followed by papules, vesicles, pustules, and scab formation.

2. Pseudo-cowpox (pseudo-pyoderma, bovine papular dermatitis) is characterized by formation of larger skin lesions than true pox, but are less acute (less painful).

3. Contagious ecthyma (Orf or Sore-mouth) of sheep is characterized by initially vesicular followed by pustular type lesions particularly in mouth.

4. Bovine herpes mammillitis which is characterized by initially edematous swelling followed by the development of vesicles and ulcers.

5. Lumpy skin disease is characterized by the formation of nodular type skin lesions in cattle.

6. Papillomatosis (warts) occurring in all species of animals, in which there is development of firm, mostly harmless, protruding masses in the form of warts, sometimes, with a dry horny surface.

7. Other viral diseases, e.g. rinderpest, ulcerative dermatitis (sheep), food and mouth disease, vesicular exanthema, blue-tongue (sheep), and mucosal disease (cattle) can also cause skin lesions.

8. Many kinds of neoplastic growths (oncogenic virus) may involve skin and related structures.

TREATMENT

In all types viral dermatitis, local application of antiseptics (solution, ointment, or powder forms) is used to control secondary bacterial infection.

Warts, protruding masses, and like lesions may be removed by surgery. The post-surgical lesion can be treated with routine antiseptic dressing. Autogenous wart vaccine (from the same animal) may sometimes be effective in prevention of new warts development caused by the same virus.

C. MYCOTIC DERMATITIS
Syn: DERMATOPHYTOSIS

Dermatophytosis refers to infection of the keratin bearing tissues (horny cells of epidermis, hairs, hoof or nails, and horns) by fungal infections.

Anthropophilic dermatophytes primarily infect man, zoophilic dermatophytes infect animals, whereas, geophilic dermatophytes are free living soil fungi (all can infect man).

A number of factors influence the susceptibility of animals to dermatophytes infection. Probably the most important is the age, with young animals being far more susceptible to infection. Some of the fungal skin diseases of animal are;

1. The major fungi causing dermatitis in animals belongs to genus Microsporum or Trichophyton, causing well known disease called "Ringworm". It is characterized by superficial, dry, crusty, raised, and circular lesions formation on skin (and hair).

The infection starts at the stratum corneum where thread like hyphae develop from spores. The hyphae grow about half way down the hair follicles and then enter the hairs. The hyphae may produce spores within or outside the surface of hairs. The lesions usually spread in circular manner from the original point of infection giving rise to the term ringworm.

2. Sporotrichosis is a contagious disease of horses, caused by Trichophyton equinum, and is characterized by development of nodular and ulcerative type skin lesions, particularly on legs.

DIAGNOSIS
The identification of dermatophytes can be made by;
i) The use of Wood's lamp (cobalt filter UV light which fluoresce yellow green light in contact with fungal lesions),

ii) Skin scrapings taken superficially from marginal areas of the lesion, treated with 10-15 per cent solution of potassium hydroxide (KOH), and then examining the presence of branching mycelia or spores under microscope; and

iii) Culture of sample on Sabouraud's medium may sometime be necessary for the diagnosis and identification of dermatophytes, by examining their typical morphology and colony/growth characteristics.

TREATMENT
. Clean thoroughly the skin lesions preferably with either 10 per cent Carbon-tetrachloride, Caustic potash, Benzene, or mild soap using a scrubbing brush.

. Topical application of any antifungal preparation, daily, in the form f solution, cream, or ointment, respond well in weeks of treatment. or examples salicylic acid (5-20 per cent solution or cream), benzoic id (5-10 per cent solution or cream), Resorcin (1-10 per cent) cream), ioform (3 per cent cream), gentian violet (0.1 per cent solution), ypochlorite solution (1:10), thiabendazole 15 per cent solution, ugol's iodine, Tr. iodine and Glycerine in equal parts. Ointment or eam containing Amphotericin-B, or Griseofulvin (not in food imals) can also be used.

Antifungal antibiotics (either fungistatic or fungicidal), i.e. mphotericin-B, with a total dose of 150 mg/LA, IV, diluted in normal line, or Nystatin or Griseofulvin, @ dose of 10-20 mg/kg, LA, or 50 g/kg, SA, PO, for 10-12 days.

Use of 10-20 per cent solution of sodium iodide, @ dose of 100-200 /LA, IV, may also be recommended for use in large animals. This n be continued for 7-10 days or until the development of iodism.

: Systemic use of iodide solution may cause abortion in pregnant imals.

D. PARASITIC DERMATITIS

EDICULOSIS (Lice infestation); Several species of both biting and animals. The associated clinical

signs are mainly pruritus and self inflicting trauma, dry coat, patchy alopecia, and crusting due to excoriation of superficial layer of skin. Anemia and weight loss can develop with heavy infestation of suckling lice. In young animals, a secondary complication in the form of hairball impaction (physical impaction in G.I. tract due to hair ball) may develop because of self grooming associated with lice infestation.

TREATMENT
Topical application of appropriate insecticide, repeated (at 2 weeks interval) for 3 consecutive treatments is effective in curing the condition. Repetition of treatment is necessary to break the louse life cycle, since eggs are not killed by most of the insecticides available for use in animals.

Some of the insecticides used in the field contain Coumaphos, Malathion (and other organophosphate compound), Pyrethrin, Lindane, BHC (and other chlorinated hydrocarbons), and Ivermectin (Ivomec® injection for use only in suckling lice infestation).

ii. MANGE MITES; The most important metazoan parasites causing dermatitis in almost all domestic animals are various species of mange mites. The include; Psoroptic, Chorioptic, Sarcoptic, Psorergatic, and Demodectic mange.

a) Psoroptic mange (mainly Sheep scab); It is caused by various species of Psoroptes mites, mostly in sheep and goats, sometimes in horses, cattle, and rabbits. It is characterized by the development of papules and nodules formation on skin along with oozing of serum, shedding of wool, and itching at various parts of the body depending upon the species of animal involved. The mites are relatively host specific and contagious among members of the same species (but don't affect people). The mites live on the surface of the epidermis and don't burrow.

Diagnosis of Psoroptic mange is mainly based upon the demonstration of mites in skin scrapings. The mites can be identified by their round bodies with long segmented pedicles.

b) Chorioptic mange (Foot and leg mange); It is a common mange in cattle (at tail), horses (at legs), and sheep, but uncommon in goats and horses. Chorioptic mange is characterized by the formation of swollen scabby, scaling, cracked, ulceration, and alopecia. Occasionally, greasy skin and extreme itching can also be noted in this mange.

Mites are usually numerous and can readily be demonstrated in skin scrapings seen under light microscope. These mites can be

differentiated with other mites by their round body, long legs, and short unsegmented pedicles. These mite may also be demonstrated in routine skin scrapings without apparent clinical signs.

c) Sarcoptic mange (or Barn itch); Barn itch is caused by various sub-species of Sarcoptic mange in various animal hosts. These mites are considered host specific sub-species but can be transmitted to human. The disease is characterized by initial development of erythema, small red papules, and much itching, followed by loss of hair, scaling, ulceration, thickening, and wrinkling of skin. The mite burrows in the epidermis where the egg is deposited and its life cycle is completed within 10-17 days.

In the skin scrapings the mites can be seen of having round body, terminal anus, short leges, and long unsegmented pedicles. These are usually present in small numbers so a negative skin scrapings do not rule-out the disease.

d) Psorergatic mange (sheep itch mites); It is mainly the mange of sheep and occasionally of cattle. The mites live in the epidermis and is usually transmitted by direct contact. Clinical signs include chronic pruritus and secondary alopecia at the affected sites. Multiple skin scrapings may be required to produce the mite. The mite is very small with radially arranged legs.

e) Demodectic mange (Follicular mange); Follicular mange is caused by the mites of Demodex spp. in almost all species of domestic animals. This mange is characterized by the development of nodules, pustules, alopecia, thickening of skin, and mild itching. Secondary pyoderma is common complication with this disease. The mites live in the hair follicles and sweat glands and are host specific. The mites are transmitted from mother to offsprings and not from one to other members of the same species. The mites are elongated and have short stubby legs. This mange is difficult to treat.

TREATMENT

Following line of treatment should be adopted for mange mite infestation in animals.

1. Clipping and burning of hair.

2. Use of insecticide in the form of either dust, spray, or dip. Examples are Asuntol, Gamaxane, Gamatox, (0.1 per cent solution), Coumaphos (0.25-0.5 per cent solution), Diazenon (0.025 per cent solution), Dichlorovas (0.5 per cent solution), Lindane (0.05 per cent solution),

Malathion (0.5 per cent solution), and Rotenone (0.5 per cent solution). All of the above are effective against most the species of mites.

3. Lime-Sulfur (ointment or dip), Sulfur ointment (1:4), Tarra-mirra oil, Benzyl benzoate (10-20 per cent solution), and Odylene (10-20 per cent solution) can also be used for the treatment of most types of mange mites.

4. Injection of Mericyle (acetyl arsenic), @ dose of 10-20 ml/LA, IM, can be used as supportive treatment in mange (and other skin ailments).

5. Injection of ivermectin (Ivomec^{R}) has been in use for the treatment of different mange mites with good results.

iii. OTHER FLYING INSECTS INFESTATION (e.g. stable flies, black flies, horn flies, horse flies, deer flies, and mosquitos); These are considered important as being associated in causing irritation and allergic skin diseases as well as vectors of many parasitic and viral diseases.

iv. MYIASIS OR MAGGOTS; The myiasis producing flies are also considered important as they can cause not only delay in healing of wounds but much nuisance to the victim. Some of the important myiasis producing flies are Blow flies, Screw worm flies, and Hypoderma (warbles) flies.

v. OTHER WORM INFESTATION; Some diseases like cutaneous habronemiasis (a nematode larvae infestation through sand fly particular in horses) and stephanofilariasis (a filarial worm infestation in cattle) infest skin to cause local lesions.

E. PHYSICAL DERMATITIS

Dermatitis can also be caused by physical agents. Examples include, excessive heat or sun-burns, excessive cold (Frostbite), physical trauma, photosensitization, X-rays irradiations, decubitus, harness injuries, and formation of callus (dogs).

TREATMENT

1. For burns, immediately immerse the burned part in cold water, and apply antiseptic containing analgesics in the form of ointment or cream or solution; e.g. Acriflavine ointment, Flamazine ointment, Cetrimide cream, and Calamine lotion.

2. Use of IV isotonic fluids as supportive therapy, if excessive skin damage or fluid loss is present.

E. CHEMICAL DERMATITIS

Dermatitis may also be caused by either local application of strong chemicals (acids and alkalies) or systemic use of certain chemicals such as Iodide or Arsenic.

F. ALLERGIC DERMATITIS

Skin lesions can also develop by skin allergies caused by allergens acting either directly on the skin (contact allergens), or indirectly by inhalation or ingestion of allergens.

TREATMENT

1. Local and/or parenteral use of anti-allergic preparations, with or without antibiotics, e.g. Corticosteroid (ointment, cream, or injection), Anthisan cream, Dexamethasone with neomycin ointment, Hydrocortisone cream, etc. (See Hypersensitivity)

NB. Corticosteroids are contra-indicated in Diabetes mellitus, Heart diseases, Uremia, during healing of wounds, and active latent infections. It should be remembered that corticosteroids do not cure but only give temporary relief. Serious side-effects can develop with prolonged systemic use of this group of drugs.

G. NUTRITIONAL DERMATITIS

Long term deficiency of certain macro or micro-nutrients and vitamins in diet of animals can cause both systemic as well as skin lesions in the form of dermatitis. Examples include the deficiency of zinc, essential fatty acids, proteins, other minerals deficiencies, avitaminosis-B, and avitaminosis-A may cause skin lesions. Deficiency disorders of the skin should be treated accordingly.

Supportive therapy in skin diseases; For supportive treatment in skin diseases, following drugs are considered significant in human medicine and can also be recommended for animals.

1. Source of vitamins particularly A and B complex,

2. Source of amino acids (aa) particularly sulfur containing aa,

3. Preparations containing essential minerals particularly calcium,
4. Source of trace minerals such as gold, antimony, manganese, and arsenic, and

5. Feed of animal should be rich in protein and mainly consists of green fodders.

PITYRIASIS
Syn: DANDRUFF

It is the presence of bran like scales on the skin surface.

ETIOLOGY

In farm animal pityriasis is usually associated with either dietary deficiency of vitamin-A, riboflavin, nicotinic acid, linolenic acid, or other essential fatty acids. Poisoning with iodine which causes conditional deficiency of fatty acid, is an important cause of pityriasis in animals. Other causes may include parasitic infestation with flea, lice, and mange mites and fungal (ringworm) infection.

PATHOGENESIS AND CLINICAL SIGNS

There is either over production (in avitaminosis-A) or excessive desquamation of keratinized epithelial cells (in parasitic infestation) leading to the accumulation of excessive scales on skin surface. Initially pityriasis begins around orifices of the hair follicles and then spreads to surrounding stratum corneum. The scales are superficial in nature and does not cause pruritus.

TREATMENT

1. Salicylic acid in the form of ointment or lotion may be applied locally as treatment of pityriasis following a thorough washing of the affected area.

2. Addition of selenium sulfide as 2.5 per cent suspension, in any skin preparation may has good effect.

3. Use of vitamins A and B, Mericyl, calcium borogluconate, and fish oil as supportive therapy can have early recovery.

HYPERKERATOSIS

It is an excessive keratinization of epithelial cells and inter cellular bridges, which accumulate at skin surface in the form of large scales.

ETIOLOGY

Hyperkeratosis may be seen when there is (i) excessive body pressure particularly at bony prominence, e.g. elbow; (ii) Poisoning with highly chlorinated naphthalene compound, which causes conditional deficiency of vitamin-A and chronic arsenic .poisoning; or (iii) Congenital ichthyosis (or fish-scale disease) in cattle.

PATHOGENESIS AND CLINICAL SIGNS

There is hyperkeratinization of epithelial cells (stratum corneum) and interference of normal cell division in granular layer of epidermis. This can result in excessive dryness, scaliness, thickening, and the development of fissures in the skin. The hyper-keratinized area if removed leaves the underlying skin intact.

TREATMENT

For the treatment hyperkeratosis, first try to remove the source of poisoning as mentioned above, Then one can use salicylic acid or other keratolytic ointments to improve the condition.

PARAKERATOSIS

It is an incomplete keratinization of epithelial cells of skin.

ETIOLOGY

The condition may be caused by either by chronic non-specific inflammation of cellular epidermis resulting in faulty keratinization of horny cells, dietary deficiency of zinc, or due to Inherited dermatosis vegetans.

PATHOGENESIS AND CLINICAL SIGNS

Due to any of the mentioned cause, there is development of edema of prickle cell layer, dilatation of intercellular lymphatics, and leukocytic infiltration. Imperfect keratinization of epithelial cells at the granular layer of epidermis follows. The horny cells thus produced are sticky, soft, and retain their nuclei. The cells either stay fixed to the underlying tissues or fall off as large scales.

Initially the lesions are red, followed by thickening, and gray coloration at the skin. The scales remain intact and also are held in place by hairs. The removal of the scales leave a raw and red underlying surface.

TREATMENT

The treatment of parakeratosis include the use of deficient nutrient (e.g. zinc sulfate or carbonate), vigorous washing with soap and water removal of the crusts, and topical application of keratolytic ointment (containing salicylic acid) or astringent lotion.

Mallenders/ Sallenders; Mallenders is a condition of horses in which there is parakeratosis at the flexor of carpal joint whereas, Sallenders is the parakeratosis of hock joint.

PACHYDERMIA

It is thickening of all layers of skin even including the subcutaneous tissues.

ETIOLOGY AND CLINICAL SIGNS

Non specific or recurrent inflammation of the skin is considered the main cause of this condition. The cells of all layers of skin may be normal but there is thickening of individual layers of skin. The affected part of skin becomes thicker and tougher than usual, and can not be picked into folds. There is no accumulation of cell debris or discontinuities of the skin surface.

TREATMENT

The treatment particularly in later stages of the disease may be difficult. Local use of ointment containing cortisone during early stages of the disease may help in recovery. Surgical removal may be attempted if small area is affected.

SEBORRHEA

It is an excessive secretion of sebum at the skin surface. This condition is rare in animals as compared to human. The common seborrhea encountered in animals is greasy heel in horses and flexural seborrhea in cattle.

ETIOLOGY

The condition can develops due to increase in blood supply in response to any inflammatory condition of skin. Excessive greasiness of skin in the form of film of oil may be associated with or without any skin lesion.

The treatment of seborrhea may be attempted through a thorough cleaning with soap and warm water followed by drying of skin by using astringent lotion. Acute cases of seborrhea may be treated with the following ingredients;

Salicylic acid (5 parts), Boric acid (3 parts), Phenol (2 parts), and Mineral oil (2 parts), or Petroleum jelly (2 parts).

URTICARIA

Urticaria is an allergic condition characterized by the appearance of transient swelling in the form of wheels on the skin and mucous membrane. It is a common condition of horses.

Primary urticaria; The primary urticaria may develops either due to local skin contact, ingestion, or parenteral administration of some allergens. Some of the known allergens are insect bites or their stings, stinging plants, ingestion of certain food items (mostly containing protein which can act as an allergen), body contact with pollens, molds, administration of drugs (penicillin), or skin contact with certain chemicals such as carbolic acid, turpentine, and crude oil.

Secondary urticaria; It may be seen occasionally secondary to some respiratory tract infections such as strangles and viral pneumonia of horses, in which urticarial type skin lesions may also be seen.

PATHOGENESIS AND CLINICAL SIGNS

Urticarial lesions are typical of allergic reaction mainly on skin. A primary dilatation of capillaries due to histamine release in response to allergens may cause local fluid escape, the development of erythema, and edema of skin (dermis and sometimes epidermis). Urticarial reaction is mainly of type-I hypersensitivity reaction, however, type-II, and type-III hypersensitivity reactions may also be involved (see hypersensitivity reaction).

Urticarial lesions are in the form of flat topped elevation in the form of wheels, from 0.5-10 cm in diameter, slightly tense to touch, pits on pressure, pruritus, and without evidence of exudation. Pitting is less apparent in later stages of the disease when there is cellular infiltration into the dermis. These lesions appear red on unpigmented (white) skin. Other evidences of allergic reaction may also be present (see allergy).

TREATMENT

1. Parenteral use of epinephrine (adrenaline), antihistaminics, or corticosteroids provide the best and most rational treatment.

2. Oral use diuretics can help in early recovery.

3. Topical application of ointment containing steroids, white lotion, vinegar, alcohol (70 per cent), or even cold water may provide ease by reducing pruritus.

4. Avoiding the contact with the known allergens is the best control.

ECZEMA

It is an inflammatory reaction of the localized epidermal cells to substances (allergens) present in external or internal environment to which they are sensitized. It is a rare condition of farm animals.

ETIOLOGY

The allergens could be endogenous or exogenous. The endogenous allergens are usually protein which are ingested as or with food. The exogenous allergens could be either external parasites and application of/or contact with medicines or other allergic agents. Inherited susceptibility of an animal to an allergen is an important predisposing factor.

CLINICAL SIGNS

he primary lesion eczema is in the form of erythema, followed by the development of intercellular and intracellular edema and usually followed by the formation of vesicles (spongiosis). Later on these vesicles rupture resulting in constant weeping of the surface, exfoliation of epidermis, and finally scab formation. Itching and irritation are intense, which could exacerbate the condition.

Chronic lesions may be in the form of much itching, scaliness, and hypertrophy of all skin layers which could result in pachydermia without discontinuity of the skin.

TREATMENT

The treatment of eczema includes (i) prevention of subsequent exposure to sensitizing substances by change of diet, clean bedding, and even the existing animal environment.

(ii) Parenteral use of antihistaminics and diuretics and local use of astringent drugs extensively may be helpful in treating eczema.

ANGIONEUROTIC EDEMA

It is the sudden development of edema in subcutaneous tissues due to both exogenous and endogenous allergens..

CLINICAL SIGNS

There is diffuse subcutaneous tissue edema at different parts of the body, particularly at head, perineum, and udder. There is no pain on touch, but mild itching may be present. General signs including bloat, diarrhea, dyspnea, salivation may also be present.

TREATMENT

The treatment of this condition tried with antihistaminics and oral use of purgatives often results in spontaneous recovery.

PHOTOSENSITIZATION
Syn: LIGHT SENSITIZATION

It is the hypersensitization of lightly pigmented (white) skin, in the presence of photodynamic agents to ultraviolet sun-light (of certain wave-length. The disease is characterized by initially sudden development of erythema, itching, edema, and later, necrosis of the skin (epidermis).

ETIOLOGY

Exposure of photodynamic agents/ substances may be systemic or by skin contact. Two basic types of photosensitization occur in animals. These are i) Photo-allergic photosensitization, and ii) Photo-toxic photosensitization.

. The Photo-allergic photosensitization; This condition occurs in those animals which are already sensitized to some photosensitizing photodynamic) compound(s). Photosensitization will occur if the animals are exposed to certain wavelength of ultraviolet (UV) light only with the presence of those compound in the body. It is a rare situation and so it occurs sporadically.

i. Photo-toxic type of photosensitization; This can further be grouped nto 4 types based on their etiological differences and includes; A. Exogenous or primary photosensitivity, B. Aberrant pigmentation ynthesis, C. Hepatogenous photosensitivity, and
). Photosensitivity of uncertain etiology.

A. In the primary photosensitivity; In this the photodynamic substances of certain plant origin reach the skin through absorption from G.I. tract. These include Hypericin (present in red pigmented leaves of plants of Hypericum spp., e.g. St. John warts), ii. Fagopyrin (present, in seeds and dried mature leaves of buck wheat i.e. Polygonum fagopyrum), iii. Perolin (present in perineal rye grass), iv. Un-identified photodynamic substances are also present in Indian wheat, wild carrot, rabbit bush, clovers, alfalfa, medicago, rape, and other members of genus Brassica.

Phenothiazine sulfoxide is a metabolic end-product of phenothiazine (a common anthelmintic) which can act as a photodynamic agent. Some compounds such as acriflavine, methylene blue, rose bengal, tetracycline, etc., can also behave similarly to cause primary photosensitization.

B. Photosensitization due to aberrant pigment synthesis; Examples are inherited/ congenital porphyria (a pink tooth disease seen only in cattle and sometimes in cats) is which there is excessive endogenous metabolism in the nuclei of developing normoblasts. This result in release of certain porphyrin pigments such as uroporphyrin, protoporphyrin, and coproporphyrin in tissue and fluid of the body. These can result in abnormal coloration of teeth, bones, body tissues, and urine. Some animals suffering from congenital porphyria may develop photosensitization.

C. Hepatogenous photosensitization; It is the most important cause of photosensitization in farm animals. In this type, the phyloerythrin, which is a porphyrin, derived from the normal microbial metabolism of chlorophyll in the alimentary tract of ruminants, is absorbed and either detoxified by the liver or excreted out in the bile. Anything that interfere with normal elimination of phyloerythrin from the body (i.e. calculi in the bile duct, hepatitis due to toxic, viral, or bacterial origin) can result in its accumulation in the peripheral circulation and thus leads to the development of photosensitization.

D. Photosensitization due to uncertain ETIOLOGY; Photosensitive dermatitis has also observed in animals fed on certain plants (kale, trefoil, lucern, etc.), grasses, fungus, or drugs in which the photodynamic substances could not be classified, but photosensitivity may occur by eating them.

PATHOGENESIS

Three factors are important in the development of photosensitization. These include i. Presence of photodynamic agents in the skin, ii. Exposure and absorption of UV light of sufficient wave-length, and iii

Lack of melanin pigment in skin (white skin) and hair coat on the skin.

The photodynamic agents or fluorescent pigment absorb UV light of certain wavelength and transform into light of a longer wave-length, which becomes activated and so passes extra energy to surrounding tissues resulting in their damage. There is liberation of histamine and other substances in those tissues which result in the development of edema and necrosis of those tissues.

CLINICAL FINDINGS

The skin lesions are limited to unpigmented or lightly pigmented areas which are under direct sun light or more exposed to sun light. These include muzzle, nostril, ears, eyelids, head, face, dorsal parts of body, with diminishing in degree down the sides, and absent from the ventral surface. Initially there is development of erythema, followed by edema and much pruritus. Affected animal seeks cool and shady places when exposed to sun light. The desire of itching and rubbing against rough surface may lead to laceration on skin. Severe edema may cause dropping of ears, and semi closure of eyes and nostrils. Exudation at the affected parts may lead to wetting of hair, and later on, exfoliation and removal of scales. More severe cases may end up with necrosis and gangrene with sloughing of affected parts (ears).

Other signs of photosensitization include anorexia, weakness, dyspnea, elevation of temperature, blindness, depression, or excitement. Shock (anaphylactic type) may be seen occasionally if much of the area is involved.

NECROPSY FINDINGS

Dermatitis of varying degree as mentioned above may be noted at unpigmented areas of skin. Occasionally, necrosis or gangrene at the skin or ears may be noted. Hepatitis can be recognized by distended gall bladder and evidences of jaundice. In congenital bovine porphyria there will be typical pink brown pigmentation on the teeth, bones, and other body tissues.

DIFFERENTIAL DIAGNOSIS

Photosensitization is limited to unpigmented or lightly pigmented areas of skin which are exposed to direct sun light. In other causes of dermatitis, e.g. mycotic or parasitic, the lesions could be seen anywhere on the body surfaces and even on the pigmented parts.

TREATMENT

The line of treatment to be adopted for photosensitization should includes the following;

1. Immediately remove the animals from direct sun-light and prevent further exposure or ingestion of toxic (photodynamic) materials.

2. Removal of toxic materials from the body by the use of purgatives/laxatives and diuretics.

3. Parenteral injection of antihistaminic and corticosteroids drugs should be used particularly in severe cases of pruritus.

4. Antibiotics may be recommended in severe cases of dermatitis to prevent secondary bacterial infection.

5. Local application of protective, anti-inflammatory, and antiseptic ointments should be recommended during the acute stages of the disease.

6. Sodium thiosulfate, 30 per cent solution, @ dose of 30 gm/LA, has been reported to reduce mortality.

7. Grazing of animals during early morning or late afternoon hours may decrease the incidence during severe outbreaks.

8. Application of blind or sun-screens on unpigmented parts during grazing hours may also reduce the development of the disease. A common hair coloring herbal powder, Henna (Mandhi) or other protectives if used during work or grazing may reduce further damage of skin by the sun light.

9. Phenothiazine use if necessary should be administered during evening hours followed by rest for grazing the next day. This practice can reduce the chances of hypersensitivity to light.

10. Symptomatic treatment may be directed to treat diarrhea, jaundice, fever, vomiting, dehydration, nervous signs, or shock if present.

PODODERMATITIS
Syn: FOOT ROT, INTER-DIGITAL NECROBACILLOSIS

It is necrotizing disease affecting the epidermis of inter-digital skin and hoof matrix. It is considered the major cause of lameness in sheep and occasionally in cattle.

ETIOLOGY

Fusobacterium necrophorum is the major cause of foot rot in cattle and sheep. Other bacteria such as Bacteroides nodosus, B. melaninogenicus, and occasionally, Corynebacterium pyogenes have also been isolated from foot lesions. Presence of these organisms together are known to cause synergistic effects and so contribute to the disease processes. These organisms can enter the epidermis through the foot injuries or bruises, usually developing by stone, straw, or hardened ground surface, particularly in warm damp/wet weather. This condition may also be seen even after long indoor housing during winter.

CLINICAL FINDINGS

The first clinical sign is development of mild to severe lameness, usually of one or both hind limbs, or even recumbency (if all foot are involved), anorexia, and progressive weight loss. Infected skin (dermal and subdermal) tissues of the inter-digital, coronet, or of bulbs become inflamed and found red initially, followed the development of necrosis, erosions, and sloughing of the affected tissues or even hoof. Purulent exudate with ozena are usually present. Occasionally, infection may reach to a wider area involving pastern or fetlock or deeper structures developing suppurative arthritis, and separation of hoof wall, basal epithelium, and dermis.

The disease can clinically be diagnosed by the presence of odor, characteristic signs, and lesion. The disease can be confirmed by identification of the causative bacteria by stain smears from the infected area.

TREATMENT

1. Removal of dead and necrosed tissues, followed by thorough washing with mild antiseptics.

2. Local use of antibacterials/ antiseptics in the form of spray, foot bath, or dusting powder often leads to early recovery. Systemic antibacterials (good against G +ive anaerobes) should also be given in acute cases. The common antiseptics used for foot rot are Tr. of iodine, copper sulfate (20 per cent), Zinc sulfate (10 per cent), and formalin (5 per cent).

TUMORS AND CYSTS OF THE SKIN

SQUAMOUS CELL CARCINOMA; The squamous cell carcinoma (SCC) are tumors that composed of squamous epithelial cells and can occur in all domestic animals. The most common SCC seen in cattle are bovine ocular tumors. The SCC can develop at the penile sheath, any where at the skin, lips, nose, or ears.

EQUINE SARCOID; This type of tumors are usually locally aggressive, fibroblastic tumors and represent the most common skin tumor of the horse. The usual sites are head, legs, and ventral abdomen. Some Virus (Oncornavirus) has been suspected as the etiology.

MASTOCYTOMA; Cutaneous mastocytoma (mast cell tumor) is a uncommon tumor of horses and cattle. The tumor mass commonly developing at the head, legs, of abdomen, in the form of single cutaneous nodule. There is multifocal regions of soft tissue mineralization as seen by radiographic examinations. The tumor is considered self limiting and metastasis has not been reported. Surgical excision often result in very low recurrence rate, even with incomplete excision.

MELANOMA; This type of tumor can occur in all type of animals, but more common in gray or white horses and cattle. The incidence of occurrence in horses increases with age, but in bovine, this type of tumor may be seen in young cattle. A disturbance in melanin metabolism associated with graying may act to stimulate formation of new melanoblasts or to stimulate their activity, resulting in focal areas of over production in the dermis and epidermis with subsequent tumor formation. Melanoma are firm, dome-shaped, hairless, and epidermal or dermal in location. They are usually multiple. Initially the overlying skin is intact, but with larger, rapidly growing tumors surface ulceration may occur. The tumor mass is typically gray to black with gross evidences of melanin pigments. The common sites are root of tail, head, udder, scrotum, prepuce, or limbs.

CUTANEOUS LYMPHOSARCOMA; It is uncommon tumor in horses and extremely rare in cattle. A number of etiological agents have been associated with these tumors, particularly bovine leukemia virus infection. The tumor may develop anywhere at the body along with bilateral enlargement of adjacent lymph nodes. The lesions tend to be raised and ulcerated, which may regress and recur any time later. Metastasis may be noted any where to the visceral organs.

CYSTS; Cutaneous cysts are benign lesions characterized by a epithelial wall with keratinous contents. These may divided into several types based on their histopathology.

Epidermal Cysts; They are of single or multiple, congenital or acquired, variable size and diameter. The cysts are covered with intact epithelium and generally do not attach to overlying epidermis. Microscopically, epidermal cysts consist of a wall of stratified squamous epithelium surrounding a keratin-filled lumen. Epidermal appendages are not associated with the cyst wall, a feature that distinguishes epidermal from dermoid cysts. Epidermal cysts are thought to originate from occlusion of a hair follicle or by traumatic implantation of the epidermis.

Dermoid Cysts; These cysts are very similar clinically to epidermal cysts but are much less common and appeared to be congenital of origin. They are believed to result from displacement of embryonic cells into the subcutaneous tissues. They can be differentiated from epidermal cysts by the presence of epidermal appendages within the wall of the cysts, and the lumen frequently contains hair and secretions from sebaceous and sweat glands in addition to keratin.

DIAGNOSTIC TECHNIQUES FOR SKIN DISEASES

Skin scrapings; Skin scrapings are used primarily for the demonstration of microscopic ectoparasites, specifically mange mites. It is quick, simple, inexpensive diagnostic technique and is particularly valuable in ruminants. The materials required for use in skin scrapings include a sterile container, mineral oil, a No. 10 scalpel blade, 2 glass slides and coverslips.

First clip the hair before taking any skin scraping. Multiple superficial scrapings that cover large surface areas, as well as several scrapings covering a small area that are deep enough to create capillary oozing should be performed. The collected material can be stored in a container until it can be examined microscopically.

Place the material on a glass slide and finely dispersed on enough mineral oil to provide a confluent layer without air bubbles beneath the coverslip. The slide so made should be scanned systematically with the 10-X objective. If something of significance is noted the 40-X objective can be used for detail examination of the specimen.

Dermatophyte culture; The material necessary to perform a dermatophyte culture include dermatophyte test medium, 70 per cent isopropyl alcohol, mosquito forceps, a No. 10 scalpel blade, and sterile empty containers such as evacuated blood collecting tubes. The forceps and the lesions site to be sample should be wiped with isopropyl alcohol to remove as many bacterial and saprophytic contaminants as possible and allowed to dry.

ANTISEPTICS USED FOR SKIN AILMENTS

Ingredients for dusting powders formulations; a) Iodoform (1 part), Boric acid (2 parts), and Zinc oxide (2 parts).

b) Sulfanilamide (1 part), Boric acid (1 part), Alum (1 part), and Copper sulfate (1 part).

c) Zinc oxide (1 part), Sulphur (1 part), and Salicylic acid (1 part).

Ingredients for lotions and solutions for topical use; a) Boric acid (1-2 gm) and distilled water (100 ml) to be used for eye wash.

b) Boric acid (0.5 gm), Alum (0.5 gm), and Zinc sulfate (0.5 gm) distilled water up to 100 ml to be used for eye wash.

c) Lead acetate (5 gm), Zinc sulfate (4 gm), and distilled water up to 100 ml (White lotion).

d) Iodine (1.5 gm), Potassium iodide (3.0 gm), Peppermint (1.0 ml), Glycerine (100 ml), and water (50 ml) to be used as throat paint.

e) Iodine (100 gm), Potassium iodide (50 gm), water (100 ml), and methylated spirit (1000 ml).

f) Salicylic acid (4 gm), Tannic acid (4 gm), and alcohol (100 ml), a formula of astringent lotion

Ingredients for skin ointment formulations; a) Benzoic acid (6 gm), Salicylic acid (3 gm), Paraffin jelly (91 gm), a formula for Whitfield ointment.

b) Iodine (1 part), Potassium iodide (1 part), and vaseline (20 parts), a formula for iodine ointment.

c) Sulphur (1 part) and vaseline (8 parts), a formula for sulphur ointment.

d) Zinc oxide (1 part) and vaseline (8 parts), a formula for zinc oxide ointment.

e) Zinc oxide (5 parts), Boric acid (1 part), Sulfanilamide (2 parts), and vaseline (20 parts).

f) Ichthamnol (5 gm), Salicylic acid (5 gm), Zinc oxide (5 gm), and vaseline (50 gm).

g) Salicylic acid (5 gm), Olive oil (50 ml), a formula for salicylic ointment.

h) Sulfanilamide or Sulfathiazole (5 gm) and vaseline (95 gm).
i) Zinc oxide (10 gm), Calcium carbonate (10 gm), Oleic acid (1 gm), Linseed oil (10 ml), and Lime water (10 ml), an ointment good for skin burn.

Ingredients of antiseptic pasts and counter irritants; a) Bismuth subnitras (1 part), Iodoform (2 parts), Liquid paraffine QS to make a paste; a formula for BIPP.

b Zinc oxide (1 part), Iodoform (1 part), Liquid paraffine QS to make a paste; a formula for ZIPP.

c) Red iodide of mercury and vaseline at the ratio of (1:8) in winter and (1:12) in Summer to be used as counter irritant.

CHAPTER-11

TOXICOLOGY

Toxicology is the study of harmful effects of chemical compounds on biological systems, including the properties, actions, and effects of the compounds. The toxic agents are called as toxicant or poison which include chemical toxins, biotoxins, or plant poisons. Accumulation (depot) and/or biomagnification can occur in the body with some toxic compounds. Acute toxicosis refers to effects of toxin during the first 24 hours periods, and the effects produced by prolonged exposure (3 months or more) are referred to as chronic toxicosis. Absorption of toxic material can occur by way of the alimentary tract, skin, lungs, or via the eyes, mammary glands, or uterus, as well as from the sites of injection.

Diagnosis of the toxicosis is based on the history of the intake or excess to the source of toxic materials, clinical signs (which may variable depending upon the type of toxic material), circumstantial evidences, observable lesions produced, laboratory examination of gut contents, blood, urine and faeces, and bioassays (animal inoculation) procedures. The following information should always be recorded for clinical investigation and despatched with the biopsy materials in suspected cases;

1. Number of animals exposed, sick, or dead, their ages, individual body weights, and chronology of morbidity and mortality.

2. Complete location and description of the facility, including other circumstantial evidences for the possible source of poisoning.

3. Complete clinical signs and course of the disease.

4. Any prior disease condition.

5. Lesions observed at necropsy with careful examination of ingesta.

6. Medication used and response of treatment if any.

7. Other related events, such as change of feed, source of water, any treatment, or application of any pesticides, herbicides or insecticides.

PRINCIPLES OF THERAPY IN POISONING

Prevention of Further Absorption; Topical applied toxicants can be removed by thorough washing with soap and water. Emesis is of value

in dogs and cats if the toxicant was ingested within a few hours. It is contraindicated when there is loss of swallowing reflex, coma, or convulsions, or if corrosive agents, volatile hydrocarbons, or petroleum distillates are involved. Oral emetics includes Ipecac (syrup) @ 10-20 ml in dog, PO, sodium chloride @ 10-15 gm in warm cup of water, and hydrogen-peroxide @ 5-20 ml, PO. Apomorphine can be used in dogs @ 0.05-0.1 mg/ kg.

Gastric lavage using an endotracheal tube and any largest bore stomach tube, should be done on unconscious or anesthetized animal. Approximately 10 ml/ kg of lavage fluid (water or saline) is gently flushed into the stomach and then removed several times until the return fluid is clear.

Laxatives or purgatives may be indicated in some instances for rapid elimination of toxicants from the gastro-intestinal tract. When poison can not be removed physically, certain agents which can either adsorb or prevent its absorption from the alimentary tract should be used. Activated charcoal is effective in adsorbing a wide variety of toxic compounds.

Supportive and Symptomatic Therapy; Various form of supportive/ symptomatic therapy is available, depending upon clinical condition of the animal. It may include i. control of convulsions, ii. maintenance of respiration, iii. treatment of shock, iv. correction of electrolytes and fluid loss, v. control of cardiac dysfunction, and vi. alleviation of pain.

Specific Antidotes; Antidotes are available for some of the known toxic materials and a list is provided with each toxicant given below. Some of them form inert complexes with toxicants, others block or compete for receptor sites, and some affect metabolism of toxicants.

COMMON TOXIC CHEMICALS, CLINICAL SIGNS, AND ANTIDOTES

Acids (Sulfuric, nitric, and hydrochloric acids); Signs of vomiting, abdominal pain, diarrhea, stomatitis, and death by acute shock. Treat with very weak alkali, Magnesium hydroxide, Calcium hydroxide, Egg-white & milk, mucilaginous substances. (Sod. bicarbonate, emetics, or use of stomach tube are not recommended).

Alcohols (Propyl, amyl, ethyl or methyl alcohols); Cause excitement, contraction of pupils, cold extremities, collapse, coma, and death. Treat by using stimulants, purgative, strong tea or coffee, or use strychnine injection when collapse.

Alkali (caustic soda or caustic potash); Symptoms as acute acids. Treat with weak acids like, 1 per cent Acetic acid, vinegar, or lemon juice. (Don't use emetics or stomach tube). Purgation with fatty oils, sedatives or stimulants.

Amphetamine SO_4; Chlorpromazine, diuretics.

Ammonia (as free ammonia and ammonium carbonate); Signs of cauterization of m.m. of eyes, cornea, and nostrils. Blood stained discharge from nostril, photophobia, dyspnea, and painful cough. Treat with diluted vinegar, demulcents, and stimulants.

Anticoagulants; Vitamin K, blood transfusion.

Antidepressants; Symptomatic, convulsions are controlled by phenobarbitone sodium, diazepam.

Antimony compound (Tartar emetic, antimosan); Cause vomiting, diarrhea, depression, and other signs similar to arsenic. Treat with Dimercaprol, @ dose of 5 mg/kg, IM, magnesium oxide, calcium hydroxide, oily purgatives, stimulants, and fluid therapy.

Arsenic salts; (Arsenic trioxide, Arsenic pentoxide, Sodium and potassium arsenate/ and arsenite, Calcium and lead arsenate, etc.); Signs including are of acute gastro-enteritis, dehydration, depression, colic, and death in acute cases. In chronic cases dermatitis with skin crackling. Treat with sodium thiosulfate 10 per cent, @ dose of 2 mg/kg, PO, dimercaprol, @ dose of 4 mg/kg, IM, purgatives, and symptomatic.

Atropine; Neostigmine, barbiturate, physostigmine.

Barbiturates; Alkaline diuretics.

Bleaches; Magnesium hydroxide, sodium thiosulfate.

Carbon-tetrachloride; High dose of this drug will cause toxicity in the form of anorexia, depression, staggering, blood stained faeces, jaundice, photosensitization, convulsions, collapse, and death. Treatment include gastric lavage, saline purgative, high protein & high carbohydrate diet, and fluid therapy in the form of calcium borogluconate, dextrose, or normal saline, IV. (Use no alcohol, oil, or fat).

Carbon-monoxide; 90 per cent oxygen, mannitol 40 per cent if cerebral edema.

Carbachol; Atropine sulphate, SC.

Carbamate; Signs similar to organophosphate poisoning. Treat with Atropine sulfate and alkaline diuretics.

Carbon monoxide; Signs are asphyxia, difficult breathing, depression, cardiac arrhythmia, m.m. bright red, muscle tremor, loose sphincter, coma, and death. Treat by artificial respiration, oxygen therapy, nikethamide, and leptazol.

Caster beans; Sedatives, arecoline hydrobromide, saline cathartics.

Chlorinated hydrocarbons; These include DDT, Rotenone, lindane, benzene hexachloride (BHC), chlordane, toxaphene, dieldrin, aldrin, and heptachlor. Clinical signs are of CNS stimulation, including muscle twitching, shivering, trembling, convulsions, and death. Treat symptomatically, Use barbiturates for convulsions, calcium gluconate and other IV fluid therapy, Gastric lavage and saline purgatives.

Chlorinated naphthalenes; (common uses in lubricant oils, insulating materials, and wood preservatives). It has depression action of vitamin-A absorption so causes 1. Hyperkeratosis (X-disease), lachrymation, corneal ulcers, salivation, depression, anorexia, diarrhea, and polyuria. 2. Decrease collagen in the skin (dermatitis), lesser hair, and poor horn growth. Treat symptomatically and with high doses of vitamin-A.

Coal-tar (Clay pigeon poisoning); Signs include severe depression, ataxia, recumbency, coma, and death. Treat symptomatically and with high protein diet.

Copper (Copper chloride, copper sulphate, copper oxide, etc.); Signs include gastro-enteritis, vomiting, abdominal pain, convulsions, and death in acute cases. In chronic poisoning, anorexia, depression, hemoglobinuria, and icterus. Treat with magnesium oxide, calcium versenate, penicillamine, and symptomatic.

Cresol, phenol; Gastric lavage with 50 per cent alcohol in water.

Cyanide; (Livestock get hydrocyanic (prussic) acid poisoning from cyanogenetic plants such as johnson grass, Sudan grass, sorghum, wild black cherry). Cyanide inhibits oxidative enzyme system and cause death from anoxia. Other signs include convulsions, salivation, dyspnea, opisthotonos, bright red mucous membranes, which later

become cyanotic. Treat with sodium thiosulfate & sodium nitrite, alone or together IV, as an antidote.

Digitalis; Potassium chloride, lignocaine.

Dinitro compounds (Herbicide); Can penetrate through skin, cause fever, dyspnea, acidosis, tachycardia, convulsions, coma, death with early rigor mortis. Treat symptomatically, IV dextrose, and sources of vitamin-A.

Fluoride; Signs include dryness of hide, staining and excessive wearing of permanent teeth, exostoses, abnormality of hooves and skeleton, anorexia, and decrease production. Treatment with calcium carbonate, and aluminum oxide or sulfate control fluorosis.

Iron salt; Desferrioxamine 2 gm in one liter of water.

Lead (paints, storage batteries, and other sources of lead);Signs; severe depression, diarrhea, blindness, walking in circle, teeth grinding, bellowing, muscle twitching, staggering, and convulsion in acute cases. Treat with sodium or magnesium sulphate, Ca-EDTA, isotonic saline and calcium solutions, and symptomatic.

Methyl alcohol; Gastric lavage.

Mercury chloride (calomel); Signs due to chronic poisoning include vomiting diarrhea due to alimentary tract necrosis, anuria or polyuria. Treat with Dimercaprol, sodium formaldehyde 5 per cent, sodium thiosulfate 20 per cent, Dextrose saline 5 per cent, egg white and milk.

Molybdenum (From Mo rich soil); Signs include diarrhea, anemia, emaciation, joint stiff and painful, unthriftiness, and fading of hair coat. Treat with 1-2 gm of (1-5 per cent) copper sulfate and sources of phosphorus as supportive therapy. One gram of copper sulphate and one milligram of cobalt, at weekly intervals for control.

Nicotine; From tobacco leaves, nicotine sulfate has been in use as insecticide, may cause toxicity in the form of dyspnea, incoordination, muscle tremor, diarrhea, salivation, cold extremities, death due to respiratory failure. Treat with respiratory stimulants, tannic acid, PO, and artificial respiration if possible.

Nitrate and Nitrite (sources are Fertilizer, Forage plants);

Signs include dyspnea, hypothermia, ataxia, cyanosis, and acute death. Blood color is chocolate brown. Treat with 2 per cent methylene blue, IV, and mineral oil, PO.

Opium alkaloid (Morphine, etc); Initial excitement, restlessness, later depression, coma, hypothermia, nausea, tympany, dilated pupil, etc. Treat with purgatives, emetic, Nalorphine hydrochloride, 2 mg/kg, IV, Atropine in small doses, stomach lavage with potassium permaganate solution, and respiratory stimulant as required.

Organophosphorus compounds; These may include Tetraethyl pyrophosphate, parathion, EPN, Malathion, Ronnel, coumaphos, diazinon, trichlorfon, dichlorvos, dioxathion, etc. The signs of toxicity are due to their cholinesterase inhibition activity, and include salivation, dyspnea, abdominal pain, ataxia, diarrhea, and occasionally convulsions. Treat with Atropine sulphate, oily purgatives, activated charcoal, and artificial respiration.

Oxalate; Calcium borogluconate.

Petroleum; Liquid paraffin.

Phenothiazine; A commonly used anthelmintic, have toxicity with high doses in the form of sensitivity to light, photosensitization, hemoglobinuria, hemolytic anemia, colic, diarrhea, depression prostration, corneal ulceration, abortion, etc. Treat symptomatically, also use purgative, stimulants, blood transfusion in anemia, and avoid direct sunlight.

Phosphorus compounds; The onset of signs is sudden, including vomiting, acute diarrhea, pain, and peculiar garlic odor from breath. Death in acute cases with exuding of blood which does not clot. Treat with Copper sulphate 0.2-4 per cent solution for emesis, potassium permeganate 0.1-0.2 per cent for gastric lavage, and IV 5 per cent dextrose.

Phytotoxin; Diphenhydramine hydrochloride.

Red squill (a common rodenticide obtained from see onion); Toxicity in the form of convulsions, paralysis, vomiting, diarrhea, depression, ataxia, dyspnea, and cyanosis. Death due to cardiac arrest. Treat with atropine sulphate, SC, and symptomatically.

Salicylates; Give alkaline diuretics and gastric lavage in treatment.

Salt (Common salt); Excessive use may cause abdominal pain, muscle twitching, diarrhea, dehydration, convulsions, incoordination, blindness, recumbency, coma, and death. Treat by salt free fresh water access, at frequent intervals with limited quantity, calcium borogluconate, and symptomatic.

Selenium; Cause alkali disease, which is characterized by lameness, deformities of hooves, stiff joints due to erosion of joints, dullness, emaciation, and loss of hair. Treatment with arsanilic acid 0.005 per cent in ration, and symptomatic. Blind stagger is an other syndrome associated with this poisoning, in cattle due to development of muscular dystrophy and vision impairment. No antidote.

Silver (Silver nitrate); Vomiting, convulsions, paralysis, and death in acute cases. In chronic cases blackening of skin, anorexia, emaciation, and anemia may be noted. Treat symptomatic along with use of sodium chloride.

Strychnine (Nux vomica); Signs; panting, muscle tremor, hyperaesthesia, tonic convulsion, and death due to respiratory paralysis. Treat with apomorphine, phenobarbital sodium, potassium permeganate (1:1000), strong tea or tannic acid 2 per cent, charcoal, diuretics and isotonic solution, and gastric lavage.

Sulfur and Lime sulfur (oldest insecticide); Lime sulfur may cause irritation and blistering at skin, rarely cause death. Treat symptomatically.

Thallium sulfate and acetate; These chemicals have been uses as rodenticide. Toxicity by accidental or deliberate access to the chemical in the form of abdominal pain, vomiting, salivation, dyspnea, convulsions, and death. Treat with potassium chloride, vitamin-B complex, and normal dextrose saline.

Warfarin; Have potent antithrombin activity, resulting in faulty clotting of blood, frequent formation of hematoma, epistaxis, and G.I. tract bleeding. Treat with vitamin K, blood transfusion, and dextrose 5 per cent, IV.

Zinc (Zinc phosphate, Zinc chloride, Zinc carbonate, etc.);
Cause abdominal pain, vomiting, acute diarrhea, severe depression, paresis, collapse, and death in acute cases. Treatment is symptomatic along with calcium and magnesium salt during the stages of paresis.

COMMON TOXIC AND MEDICINAL PLANTS OF PAKISTAN

Calotropis procera (AK or MADAAR); It is a potent toxic plant and have tow varieties. Milk of Ak is called as latex which is highly toxic and irritant. Young leaves, flowers, and bark contain toxic components. These are also used in indigenous (country) medicine.

Aloe vera (ALOE, BOLE-SIAH, ALUA); Medicinal plant commonly used in indigenous medicine for gastrointestinal disturbances, healing of wounds, making soft skin surface, and in muscle pain. It contains an alkaloid called Aloin which acts as an irritant cathartic.

Cassia fistula (AMMALTAS); Dried fruit (without seeds) of this tree is known to have laxative effect. These are commonly used with other local medicines for the treatment of constipation.

Tribulus terristris (BAKHRRA, Puncture vine, Caltrop); A forage weed, commonly grow at sandy soil, used in local medicine for colic and chronic cough. Also known to have phototoxic and hepatotoxic contents, called Phylloerythrin.

Sorghum halepense (BARU grass, Johnson grass); This grass under drought conditions can have high contents of Hydrocyanic acid, can cause acute poisonous similar to Sorghum and Sudan grass.

Chenopodium album (BATHU, Lamb's quarter); A weed, have laxative effects, also used for headache as poultices, and as a vegetable. Under circumstances may contain high contents of Nitrates, and Saponin. May cause nitrate toxicity when leaves are eaten raw, but not cooked leaves (see also Lucern).

Atropa belladonna (Belladonna, BABROOJ); The most important of belladonna alkaloids are Atropine, Hyoscyamine, and Hyoscine. Toxic signs similar to Henbane and Datura.

Cynodon dactylon (Bermuda grass or cough grass); It contains high contents of Hydrocyanic (prussic) acid under drought conditions. For toxicity see Sorghum.

Cannabis indica (BHANG); The leaves of this plant are well known to have analgesic, sedative, & narcotic effects. When in excess use can cause constipation.

Bitter cucumber (TUMMA); A medicinal plant, commonly used as rumenitoric and carminative. Also known to enhance T-cell response.

Pteridium aquilinum (FERN, Bracken fern); This plant contains Thiaminase which causes thiamine deficiency and a Radiomimetic factor causing bone marrow suppression and aplastic anemia. In addition a glucoside, b-glucopyranoside have histamine like activity. Toxicity signs include incoordination, bradycardia, and aplastic anemia. Blood transfusion along with other measures for treatment.

Ricinus communis (Castor oil bean, RIND); The seeds contain a phytotoxin, Ricin which acts as a proteolytic enzyme. Also have primary photosensitizing substances. Seeds can cause severe purgation, photosensitization, straining, salivation, depression, trembling, and incoordination. Specific antisera raised against Ricin may be used as antidote. Gastric sedatives, i.e. Arecoline hydrobromide may also be used as treatment.

Trifolium repens (White clover), T. pratense (Red clover); These contain a natural substance called Coumarin, and Phytoestrogen which is known to cause infertility, dystokia from uterine inertia, and vaginal prolapses.

Zea maize (Corn, Maize, MAKKI); Under circumstances of drought the plant may contain higher contents of Hydrocyanic acid. See also sorghum.

Cotton seed cake, KHAL BANOLA; Contain Gossypol, which is a phenolic substance present in cotton seed cake. It is a cardiotoxic and hepatotoxic may cause heart failure. Other signs are anorexia, dyspnea, and death. Cooking of the cake and addition of 1% calcium hydroxide or 0.1% ferrous sulfate is efficient method of detoxification.

Croton tiglium (Croton oil, JAMAAL-GHOTA); Many medicinal uses because of its highly irritant effects on gastric and intestinal mucosa. Used as drastic purgatives, and may cause super-purgation and severe colic.

Datura indica (Thorn-apple), D. stramonium (Jimpson weed), (DATURA); These plants contain many active alkaloids, i.e. Atropine, Scopolamine, Hyoscyamine, and hyoscine in all parts, seed in particular. Also a Nitrate accumulating plants, high contents during drought conditions. Signs of toxicity are tachycardia, dilated pupil, dry mouth, incoordination, convulsions, and coma. Symptomatic treatment and use physostigmine, pilocarpine, or arecoline.

Digitalis purpurea (Foxglove); This plant is not palatable for animals, but some may develop its taste, or accidental access may cause [illegible] and sudden death due to heart failure

Dysphania lepidota (Dysphiniya, MALATHI); Medicinal plants, roots are commonly used as cough sedative and expectorant. Also known to have anti-inflammatory effects.

Hyoscyamus niger (Henbane, AJWAEN KHURASANI); Medicinal plant, used as carminative, to control hemorrhage, for reducing abdominal pain, asthma, and anti-cestodal effects. It contain a tropan alkaloid, Hyoscyamine and Hyoscine. Toxicity may appear in the form of tachycardia, dry mouth, ataxia, convulsion, subnormal temperature, and death. Cholinergic drugs, like pilocarpine or physostigmine may be used in cases of respiratory depression.

Leucaena leucocephala (Ipla ipla); The tree is commonly uses as fodder because of high contents of protein, but it contain a toxic amino acid, called Mimosine. Mimosine is found in all parts but particularly, in young growing leaves and seeds. Toxicity may be seen in the form of alopecia, cataract, oral ulceration, goiter, infertility, low birth weight, jaundice, and nervous signs.

Plantago ovata (Isaghula, ISBAGOLE, Plantago seeds); Many medicinal uses, seeds are commonly used as simple bulk laxative, for coating of gastric and intestinal mucosa in inflammatory conditions. Commonly used in diarrhea, as the seeds absorb water and swell up and form a emollient gel. Also used in gastric ulcers in human.

Gelsemium sempervirens (Jessamine, Evening trumpet flowers); Alkaloids are Gelsemine and other related to Strychnine in all parts. Can cause incoordination, dilated pupils, convulsions, coma, and death.

Sorghum halepense (Johnson grass); See BARU grass.

Chenopodium murale (KRUNDI); A weed, have laxative effect, and bitter taste.

Lantana camara (LANTANA); A ornamental bush/hedge. Leaves and green berries are toxic due to Lantadene A & B. Also has photosensitizing substances. It can cause anorexia, jaundice, watery faeces, and photosensitization.

Delphinium spp (Larkspurs, GUL-E-NAFARMAN); Tall and small larkspur varieties. Alkaloid called Delphinine present in all parts in early growths. Can cause constipation, bloat, salivation, vomiting, respiratory or cardiac failure. Flowers may be used for lice infestation.

Linseeds and linseed cake, KHAL ALSI; Contain a Hydrocyanic acid glucoside called as Linamarin, toxicity occur when large quantity is feed to hungry sheep. Lamb may develop goiter. Cakes can be detoxified by boiling for 10 minutes before feeding.

Medicago sativa (Lucern or Alfalfa); Nitrate accumulating plants (higher contents in drought), contain Phytoestrogen (which can cause infertility), Nitrate contents, and also contain some Photosensitizing agents, so can cause photosensitization, dyspnea, brown mucosa due to formation of methemoglobin, and death. Treatment with 1-2% methylene blue, @ dose of 5-10 mg/kg, is recommended for nitrate poisoning.

Panicum miliaceum and other species (Millet); Young and fast growing plants contain high contents of Nitrate and some hepatotoxic substances (Phylloerythrin) which can cause liver and bile duct damage, and signs of jaundice and photosensitization. See also Sorghum.

Mentha pepprita (Mint, PODEENA); Many medicinal uses, particularly used for digestive disturbances. Contain high contents of Nitrate. For nitrate poisoning see Sorghum.

Thuja orientalis (MOORPUNKH); Seeds of this plant are known to cause toxicity.

Mustard seeds; Known to induce vomiting (emetic effect) through stimulation of peripheral receptors, very similar to copper sulfate, sodium chloride, and hydrogen peroxide. Can also cause photosensitization in animal fed on seeds and even green leaves.

Melia azedarach (Chinaberry, NEEM); Many medicinal uses, fruits and leaves are known to have bacteriostatic effects. Large quantity of fruit may cause restlessness, vomiting, constipation, cyanosis, dyspnea, and death. Contain many alkaloids and saponin in all parts.

Quercus spp (Oaks, Oak buds or Acorns); Contain a tannin, Gallotanin, in young leaves and acorns. Can cause anorexia, rumen stasis, constipation, followed by tarry diarrhea, abdominal pain, polyuria, dehydration, and rapid pulse. Oral rumenitoric and calcium hydroxide 15% in ration may be given as treatment.

Avena sativa (Oats); Contain high Nitrate contents particularly in drought. Can cause brownish color of mucosa due to methemoglobin, dyspnea, death. Treat with 1-2% methylene blue, @ dose of 5-10 mg/kg.

Nerium oleander (Oleander, KANAIR); Digitoxin like glycosides, Oleandroside, Nerioside, and others. Can cause gastroenteritis, vomiting, tachycardia, depression, and death. 30-40 dried leaves may be lethal to cows and horses. Atropine and propranolol may be used as treatment.

Allium sp. (Onion, PIAZ, GANDHA); Both cultivated and wild varieties of onion. Can cause Heinz body anemia when eaten in large quantity by horses, sheep, and cattle. Toxic agent is a disulfide, n-Propyl-disulfide. Signs include hemolytic anemia, hemoglobinuria, pale mucosa, and jaundice.

Oxalis europea (PATTKAL); A toxic weed containing lot of Oxalic acid and taste like sour. Oxalic acid form insoluble complex with calcium. Signs may be depression, dyspnea, recumbency, and unconsciousness. Treat with fluid therapy, diuretics, and calcium salts.

Solanum nigrum (PEELAK, MAKO, Garden nightshade); Contain glyco-alkaloids, Solanine and Hyoscine in all parts, and some Alkamine fractions. Can cause acute pain, hemorrhagic gastroenteritis, salivation, dyspnea, trembling, progressive paralysis, prostration, and death. Treat with pilocarpine, physostigmine, atropine, and G.I. tract protectants.

Papaver somniferum (Poppy plant, POSTE); Opium, a well known narcotic, is derived from the air dried juice obtained from unripe capsule of the white poppy. There are more than 2 dozens of active alkaloids, the principals of them are Morphine, Codeine, Heroin, and Narcotine. Toxicity appears when excess of poppy stems and whole capsule is ingested. Signs include severe depression, bradycardia, anorexia, bloat, and pupil dilated.

Solanum tuberosum (Potato); Potatoes are toxic if are green and sprouted due to toxic alkaloid, Solanin. Signs include dullness, diarrhea, subnormal temperature, prostration, and coma. Toxic effects can be reduced by boiling.

Brassica napus (Rape), B. oleracea (Kale), and also Turnips; Many chemicals such as Glucosinolates (Isothiocyanate, Thiocyanate, Dimethyl disulfide, Nitrites) are present in seeds and vegetative parts. Also contain primary photosensitizing substances. Can cause anorexia, gastro-enteritis, salivation, diarrhea, paralysis, photosensitization, hemolytic anemia, and hemoglobinuria. Turnips contain Thiaminase so can also cause thiamine deficiency signs.

Rape seed meal (cake), KHAL-TORIA or SARSON-KI-KHAL; Contain high goitrogenic substance, Goitrin and Glucosinolate (produced by the rumen bacteria within body). Abdominal pain and diarrhea may occasionally be seen as toxicity reaction.

Abrus percatorius (RATTI, GUNCHI); Active alkaloid is Arbin, having similar action as Ricin. Powder of ratti seeds has been used for poisoning cattle by culprits. Signs of poisoning include severe local swelling at the injection sites, salivation, incoordination, clonic spasms, and sudden death.

Atriplex sp (Salt bush, KALAR grass); A Selenium containing plant grow in selenium rich soils. Toxicity is associated with its predilection for reacting with sulphur containing amino-acids such as cysteine. Signs include pyrexia, dyspnea, myosis, abnormal posture/gait, frothy nasal discharge, recumbency, diarrhea, and death. Methionine, linseed oil, sodium sulfate or arsanilic acid may be used as treatment.

Sorghum vulgare, Sorghum spp (Sorghum, Sudan grass); High contents of Hydrocyanic (Prussic) acid, (particularly in drought and at second growth), and Nitrate (if heavy use of fertilizer). Can cause acute dyspnea, bloat, staggering, convulsions, and death. Blood bright red (cyanide poisoning) or chocolate brown (nitrate poisoning). Treatment include the use of 20% sodium thiosulfate, @ dose of 100 mg/kg, and 20% sodium nitrite, @ dose of 6 mg/kg. To treat high nitrate poisoning see Oats toxin.

Other plants containing high Hydrocyanic acid contents are Millet (Sorghum vulgare), White clover, Bitter almond (Prunus virginiana), Wild black cherry, Arrow grass, etc.

Beta vulgaris (Sugar beets, and fodder beets); Beets are occasionally used as fodder, may cause acute ruminal impaction with lactic acidemia. Green part contain Oxalic acid and high Nitrate contents. Toxicity may be in the form of hypocalcemia, hypomagnesemia, blindness, and hemolytic anemia (similar to Brassica sp).

Nicotiana tobacum (Tobacco) and N. glauca (wild tobacco); Plant contain Nicotine, Nicotimine which are ganglionic blockers, in leaves and bark. Can cause nausea, vomiting, salivation, diarrhea, shivering, staggering, progressive prostration, and cardiac abnormalities. Teratogenic effects in pregnant animals due to Anabasine present in wild variety. Nicotine sulfate may used occasionally as anthelmintic and insecticide.

CHAPTER-12

COMMON ANTIBACTERIALS, ANTIPARASITICS, AND ANTI-INFLAMMATORY DRUGS USED IN VETERINARY MEDICINE

SULFONAMIDES

All sulfonamides have bacteriostatic (BS) effect, inhibit folic acid formation of bacteria, and may be used against both Gram +ive & -ive bacteria. Followings are some of the common sulfonamides;

Sulfadimidine (Sulfadiadine), Sulfathiazole, Sulfaphenazole, Sulfadiazine (@ dose of 30-50 mg/kg, SA), Sulfamerazine, Sulfapyridine (@ dose of 60 mg/kg, LA), Sulfamethoxazole (@ dose of 25-55 mg/kg, LA and SA), Sulfaquinoxaline sodium,
Sulfaguanidine, Phthalylsulfathiazole (Gut acting sulfonamide).
Trimethoprim (is commonly used in combination with sulfa drugs and have synergistic effects (e.g. Tribrissen, Septran), @ dose of 20-30 mg/kg, for both LA and SA.

ANTIBIOTICS

There are many groups/classes of antibiotics. Examples are;
Tetracycline; These are BS, interfere ribosomal functions, and are used for both Gram -ive & Gram +ive bacteria. e.g. Oxytetracycline (Terramycin), @ dose of 10-15 mg/kg, LA and SA. Chlortetracycline (Aureomycin), @ dose of 20 mg/kg, SA., and
Doxycycline and Minocycline are more lipid soluble, better able to penetrate bacterial cell wall and CSF.

Penicillin; All are bactericidal (BC), inhibit the cell wall synthesis of bacteria. Examples include; Penicillin (Benzyl, Procaine, G, and V), used against Gram +ive bacteria, @ dose of 10,000 to 20,000 i.u./kg, for LA, and 20,000 to 40,000 i.u./kg for SA. Ampicillin (Penbritin), for both Gram -ive & +ive bacteria, @ dose of 10-15 mg/kg for LA, and 15-20 mg/kg for SA.
Amoxicillin, Cephlexin, Cefaclor, Carbenicillin (Pyopen), for Gram -ive bacteria, @ dose of 15-20 mg/kg for both LA and SA. Cloxacillin, for Gram +ive bacteria, @ dose of 5-10 mg/kg for both LA and SA, and Oxacillin, for Gram +ive bacteria.

Beta Lactam are antibiotics effective against G -ive bacteria. These include Penicillin, Cephalosporin, and the combination of beta lactam antibiotics/ beta lactamase inhibitors.

Cephalosporin; All are BC, inhibit cell wall synthesis, and are used for both Gram -ive & +ive bacteria. Examples are; Cephalothin (Keflin), @

dose of 10-20 mg/kg, LA, and 20-30 mg/kg, SA. Cephlexine (Keflex), Cefazolin, and Cephaloridine (Keflodin).

Cephamandole, Cefaclor, Cefixitin, and others are second generation cephalosporin.

Cefotaxime, Ceftizoxime, and oxa-beta-lactam moxalactam are third generation cephalosporin.

Note: These are very costly.

Aminoglycosides; All are BC, interfere ribosomal functions and disrupt respiratory function of bacteria. These are used for both Gram -ive (& +ive) bacteria, and effective at anaerobic environment. These are closely related chemical and tend to be toxic (nephrotoxic, ototoxic) to the host. Examples are; Streptomycin, @ dose of 10-20 mg/kg, LA and SA. Dihydrostreptomycin, Kanamycin, @ dose of 5-10 mg/kg, LA and SA.
Neomycin, @ dose of 10-20 mg/kg, LA and SA. Gentamicin (Gentocin), @ dose of 2-5 mg/kg, LA and SA. Amikacin, Toberamycin (new), and Netilmicin (new).

Polypeptides; All are BS, inhibit cell wall and impair membrane functions, for mainly Gram -ive bacteria. Examples are; Bacitracin, Polymyxin-B, @ dose of 40-50 mg/kg, SA, and Colistin, @ dose of 1 mg/kg, SA.

Nitrofurans; Interfere DNA polymerase functions?, used for both Gram -ive & +ive bacteria. Examples are; Furazolidone, (BC), Nitrofurantoin, (BC), @ dose of 2-3 mg/kg, SA, and Nalidixic acid (BS).

Chloramphenicol (Chloromycetin); BS antibiotic, inhibits ribosomal functions, used for both Gram -ive & +ive bacteria, as well as rickettsial organisms. Normal dose rate is 20-40 mg/kg, LA, and 40-50 mg/kg, SA.

Quinolones; These are synthetic BC antibiotics, effective against G -ive bacteria, and have minimal toxicity. These include; Nalidixic acid, Norfloxacin (Noroxin), Ciprofloxacin, and Enrofloxacin (Baytril).

Macrolides; (All are BS, inhibit ribosomal functions, and are used for mainly Gram +ive bacteria, mycoplasmas, and Actinomyces. Examples are; Erythromycin (Erythrocin), @ dose of 4-5 mg/kg, LA, and 10-12 mg/kg, SA. Oleandomycin, and Tylosin (Tylan, Tylocine), @ dose of 4-5 mg/kg, LA and SA.

Lincosamides; Antibiotics are either BC or BS, inhibit ribosomal function, mainly used for bone infection caused by G +ive bacteria. Example include; Lincomycin, Clindamycin.

Vancomycin: All are BC, used for Gram +ive bacteria.

Note: (Antibiotics can be used in combination, keeping in mind the rule of thumb that bacteriostatic should be used with bacteriostatic and bactericidal with bactericidal antibiotics. Two bacteriostatic antibiotics will have additive effect, whereas, two bactericidal antibiotics will have synergistic effects.

ANTIFUNGAL ANTIBIOTICS

Amphotericin-B, has both fungicidal & fungistatic effects, through binding with ergosterol. Nystatin, Ketoconazole (has fungistatic effect through inhibiting fungal cell membrane erosterol), Flucytosine (is a antimitotic antifungal drug), Griseofulvin, Thiabendazole, Crystal violet, Benzoic acid,
Salicylic acid, and Copper sulfate also have some local antifungal activity.

ANTIPROTZOANS

a). Babesicidal drugs; Examples are; Diminazene aceturate (Berenil), Imidocarb (Imizol), and Quinuronium sulfate (Acaprine, Pirevan).

b). Coccidiostates; Amprolium, Diaveridine, Furazolidone, Ionophores group, Qunolone group, Sulfadimidine and Sulfapyridine.

c). Trypanocidal drugs; Diminazene aceturate (Berenil), Homidium bromide, Pyrithidium bromide, Quinapyramine (Antrycide), and Suramin (Naganol).

ANTHELMINTICS

For Ascaris (Nematodes, Roundworms); Piperazine citrate, adipate, or hyxahydrate, @ dose of 100-200 mg/kg, Mebendazole preparation, @ dose of 100 mg/SA, b.i.d. for 3 days, Diethylcarbamazine citrate, @ dose of 6-12 mg/kg. Thiabendazole, @ dose of 50-60 mg/kg, PO. Parbendazole, @ dose of 30 mg/kg, as 4 per cent premix, PO. Levamisole, @ dose of 25 mg/kg, PO. Tetramisole, @ dose of 15-30 mg/kg, PO. Mebendazole, @ dose of 5-10 mg/kg, PO. Fenbendazole, @ dose of 7-10 mg/kg, PO. Carbendazole, @ dose of 20-40 mg/kg, PO. Albendazole, @ dose of 5-10 mg/kg, PO. Pyrantel, @ dose of 25 mg/kg,

PO. Morantel tartrate, @ dose of 10 mg/kg, PO. Methyridine, @ dose of 200 mg/kg, PO. Coumaphos, @ 2 dose of mg/kg, as premix.

For Hook-worms in dogs; Bephenium hydroxynapthoate, @ dose of 200 mg/kg, PO. Disophenol (Ancylol, Cyanamid), @ dose of 1 mg/kg b.w., S/C injection.

For Flukes (Trematodes); Niclosamide, @ dose of 50-100 mg/kg, PO. Hexachlorophene, @ dose of 1-2 mg/kg, PO. Hexachloroethane, @ dose of 200 mg/kg, PO. Resorantel, @ dose of 60 mg/kg, PO. Clioxanide, @ dose of 50 mg/kg, PO.

For Tape-worms (Cestodes); Copper sulphate, @ dose of 100-200 mg, sheep/goats, PO. Dichlorophen, @ dose of 200-400 mg/kg, PO. Albendazole, @ dose of 10-15 mg/kg, PO. Fenbendazole, @ dose of 200 mg/kg, PO. Niclosamide (Yomesan), @ dose of 75 mg/kg, PO. Bithinol, @ dose of 200 mg/kg, PO.

PHARMACOLOGICAL MODULATORS OF INFLAMMATION

STEROIDS THERAPY

Adrenocortical steroids (glucocorticoids) and their synthetic analogues are powerful anti-inflammatory agents, but they must be used with a fair degree of caution. The mechanism of action of these products are not entirely understood but are though to result in part from inhibition of arachidonic acid (aa) released from damage cell walls and subsequent metabolism of aa to potent mediators of inflammation. Glucocorticoids are effective only in altering tissue response and clinical signs and are useful both in early and later stages of inflammation. Other pharmacological effects include suppression of T-lymphocytes and macrophage activity, release of lysosomes from neutrophils, decreased neutrophilic chemotaxis (in large doses), decrease antibody production, and some inhibition of plasma mediators of inflammation. The net effects of glucocorticoids thus include inhibition of edema, decrease capillary permeability, decline leukocytic migration and phagocytosis in early inflammation, and cut capillary and fibroblast proliferation in later stages.

Glucocorticoid products are available for intravenous (IV), intramuscular (IM), intra-articular (IA), and topical administration. Glucocorticoids as a family tend to be widely distributed and can readily penetrate most tissues. Significant systemic absorption can result from IA and topical administration. While the plasma half life tends to be short, the biological half life vary and result in classification of these agents.

Dose Regimens Of Commonly Used Anti-inflammatory Drugs
Hydrocortisone, @ dose of 5 mg/kg, IM, IV, or PO, daily.
Prednisolone, @ dose of 0.5-2 mg/kg, IM, repeated 12 hourly.
Prednisolone, @ dose of 5.5-11 mg/kg, IV.
Methylprednisone, @ dose of 1 mg/kg, PO, repeated 8 hourly.
Methylprednisone, @ dose of 1 mg/kg, IM, at 14 days intervals.
Dexamethasone, 0.15 IM, IV, PO 24 hours
Dexamethasone, @ dose of 5 mg/kg, IV.
Betamethasone, @ dose of 0.17-0.35 mg/kg, IM, repeated 6 hourly.
Triamcinolone, @ dose of 0.11-0.22 mg/kg, IM, SC, PO, daily.
Flumethasone, @ dose of 0.05-0.25 mg/kg, IM, IV, PO, daily.
Aspirin, @ dose of 10 mg/kg, PO, repeated 8-12 hourly.
Orgotein, @ dose of 5 mg/SA, SC, repeated 24-48 hourly.
Phenylbutazone, @ dose of 15 mg/kg, PO, repeated 8 hourly.
Banamine, @ dose of 0.25 mg/kg, IV, daily.

NON-STEROID ANTI-INFLAMMATORY DRUGS (NSAIDs)

The NSAIDs can be divided into the following groups according to their chemical structure;

1. Salicylates (e.g. Aspirin),
2. Indoles (e.g. Indomethacin),
3. Propionic acid (e.g. Ibuprofen, Naproxen),
4. Fenamates (e.g. Meclofenamine, Flunixin meglumine), and
5. Pyrazolones (e.g. Phenylbutazone, Dipyrone)..

The common therapeutic benefits of NSAIDs result from their antipyretic, analgesic, and anti-inflammatory properties and these are through inhibition of prostaglandins (PGs) formation.

The PGs are eicosanoids which are formed within the body cells. Damage to cell membrane (by any way) activates phospholipidases (enzymes). These enzymes hydrolyses cell membrane and converts phospholipids to glycerol and fatty acids. The most important fatty acid is the arachidonic acid. Once arachidonic acid releases into the cells, it serve as substrate for other enzymes. Cyclo-oxygenases catalyses the formation of cyclic endoperoxides (i.e. PGG_2 & PGH_2), which rapidly metabolized to the final PG products by isomerases into PGE, PGF_2a or by synthetases into PGI_2 or prostacyclin and thromboxane-A_2.

As a group PGs have a variety of physiological effects. The process of inflammation is mediated perpetuated by PG by induction of changes in capillary permeability, and chemotaxis potentiate the effects of other

mediators, as histamine and bradykinin. In addition, there is formation of some unstable cyclic endoperoxide intermediates (toxic oxygen radicals) which are responsible for tissue destruction.

The NSAIDs inhibit cyclo-oxygenase activity and thus prevent the formation of all PG intermediates and end products. Some drugs are irreversible inhibitor (e.g. Aspirin) and other are reversible (e.g. Ibuprofen). The effect of aspirin is through inhibition on isomerases and synthetases, while salicylates inhibit other mediators (e.g. Kinin).

The toxicity associated with the prolong use of NSAIDs is through inhibition of PGs formation. Some of the toxic effects are gastro-intestinal ulceration, bleeding dyscrasias, and nephropathy.

The gastro-intestinal ulceration develops due to either direct irritation (e.g. Aspirin) or through inhibition of PGs formation. The PGs not only inhibits gastrin and hydrochloric acid secretions but also stimulate mucous and bicarbonate secretions. This help in maintenance of mucous barrier and diffusion of hydrogen ions into gastric mucosa. Inhibition of PGs reverse the situation.

The bleeding dyscrasia develops either due to impaired platelet function (deficiency of Adhesion protein) through synthesis of thromboxane synthesis or by depression effects on bone marrow hematopoiesis (e.g. phenylbutazone).

Renal nephropathy may develop either through impaired blood flow or tubular secretion of proteins.

Aspirin

Aspirin is used as analgesic, antipyretic, and anti-inflammatory effects, in degenerative joint diseases and thromboembolism. Aspirin is converted into an active ingredient called salicylic acid. 50-70 per cent of the salicylic acid is bond to serum albumen and rest is distributed in tissues. In phase-II, it is conjugated with glucuronide and then reutilized. The therapeutic dose of aspirin is 10-20 mg/kg (for 24-48 hours).

Phenylbutazone

Phenylbutazone is used as anti-inflammation, in degenerative joint disease, and musculo-skeletal and post operative pains. It has more toxic effects in human and cat than dogs. The dose is 10 mg/kg, PO, IM, BID, maximum dose up to 500 mg per day.

Flunixin meglumine

It is used as visceral analgesic, anti-inflammation, in endotoxic shock, and post operative analgesia. The dose is 1 mg/kg, IV or PO.

Naproxen
It is used to reduce inflammation and musculoskeletal pain. The recommended dose is 1-2 mg/kg, single dose therapy.

Ibuprofen
Ibuprofen may cause gastro-intestinal tract upset (vomiting in dogs) and diarrhea. The dose is 10 mg/kg, PO.

Acetaminophen
It is a coal-tar derivative and has analgesic and antipyretic but not as anti-inflammatory effects. These effects are through inhibition of endoperoxidase intermediates. This drug is toxic in cats.
its toxicity for this species.
Dipyrone (Novalgin)
It is used as antipyretic and analgesic in dogs and other animals. In cats is may not be used owing to

Chapter-13

DISEASES OF URINARY SYSTEM

DEFINITIONS

PROTEINURIA (ALBUMINURIA)
It is the passing of protein (mainly albumin) with the urine. This condition is commonly seen not only in diseases of urinary system but also of other systems. Examples are nephrosis, nephritis, renal infarction, congestive heart failure, toxemic fever, hematuria, hemoglobinuria, and myoglobinuria. In neonates, proteinuria may normally be present due to high intake of colostrum. A chronic persistence of proteinuria may end up with severe muscle wasting and emaciation. An increase quantity of protein in urine without leukocytes, red cells, bacteria, or cell casts suggest glomerular protein loss, as seen in nephrosis. Presence of protein with bacterial and leukocytes suggest active development of sepsis in the urinary tract (nephritis).

HEMATURIA
It is the passing of (whole) red blood cells in the urine. Hematuria indicates traumatic injury in the urinary tract. This condition is commonly seen in purpura hemorrhagica, acute glomerulonephritis, pyelonephritis, cystitis, renal infarction, renal ischaemia, urolithiasis any where in urinary tract, and enzootic hematuria of cattle.

The indication of hematuria may be in the form of gross blood clots passed at beginning or end of urination, or passing of uniform red colored (deep red to brown color) urine with or without any clot. In mild cases of hematuria there may be mere cloudiness of urine and if urine is left to settle red deposits may be noted at the bottom. Occasionally, hematuria may be so slight that intact RBCs are only visible at microscopic examination of the sediments. A horse having hematuria after exercise should be suspected of having cystic uroliths. Injury to the bladder may be identified by the presence of hematuria at the end of the urination.

HEMOGLOBINURIA
It is the presence of hemoglobin in the urine, making the color of urine from deep red (in mild cases), dark brown (moderate cases), to black in color (in severe cases). Presence of hemoglobin in the urine will also give positive (laboratory) biochemical reactions for blood as well as protein, without the presence of intact RBCs. Hemoglobinuria is commonly seen in babesiosis, anaplasmosis, bacillary hemoglobinuria.

leptospirosis, post-parturient hemoglobinuria, poisoning with rape and kale, copper intoxication, and autoimmune hemolytic anemia (particularly in foals).

MYOGLOBINURIA

It is the presence of myoglobin (myohemoglobin) in the urine. Its presence in the urine is a good indication of severe muscular dystrophy, e.g. azoturia. Myoglobin in urine can turn urine color dark brown, which is also seen in hemoglobinuria. So, if urine sample is vigorously shaken in a test tube, myoglobin tends to produce a brown foam, whereas hemoglobin produces a reddish foam. In addition, ammonium sulphate (80% saturated solution) will cause precipitation of hemoglobin but not the myoglobin. These tests are though crude means of differentiation myoglobinuria from hemoglobinuria.

PYURIA

It is the presence of purulent debris (pus cells) in the urine and is a good indication of active inflammatory process within the urinary tract (renal pelvis or bladder). A large clot of pus can be visible with naked eyes or it can be detected by microscopic examination, with or without prior centrifugation of urine sample. Pyuria is usually associated with the presence of bacteria in the urine and is an indication of development of sepsis. A fever may or may not be present with pyuria.

GLYCOSURIA AND KETONURIA

Glycosuria with ketonuria only occurs in diabetes mellitus, which is a rare condition in large animals. Presence of glycosuria without hyperglycemia strongly suggests renal tubular damage resulting from toxic or ischemic insults. Glycosuria is also commonly associated with enterotoxemia caused by *Clostridium perfringens* type-D infection (pulpy kidney in sheep), administration of high doses of glucose, catecholamine or glucocorticoid hormones release, stress, and also failure of tubular resorption in ischemia. Ketonuria is commonly seen in starvation, pregnancy toxemia, and acetonemia.

INDICANURIA

It is the presence of indicans (i.e. potassium indoxy sulfonate) in excessive quantity in urine. Presence of indicans in urine indicates an increase absorption of this detoxification product of indole from the large intestine as seen in severe constipation.

CREATININURIA

It is the presence of creatinine in urine, which is usually seen when there is excessive endogenous muscle breakdown, e.g. muscular dystrophy.

POLYURIA

It is the passage of abnormally large quantity of urine, either due to excessive intake of water and/or diet low in sodium chloride. A continued polyuria is the result of decrease tubular resorption. This condition can be seen when;

i) In the absence of antidiuretic hormone (ADH), e.g. central diabetes insipidus,

ii) Decrease effects of ADH on receptors in the kidneys, e.g. nephrogenic diabetes insipidus,

iii) Increase in solutes in glomeruli filtrate beyond the resorptive capacity of tubular epithelium,

iv) Due to damage to tubules (in nephritis).

Other conditions associated with polyuria are hyperglycemia, steroids and diuretic administration, or deficiencies of chloride, potassium, or urea. Persistence of polyuria for a long time can result in severe dehydration particularly, if water intake is reduced.

OLIGURIA AND ANURIA

Oliguria is the reduction in daily output of urine. A complete absence or stoppage of urine is called as anuria. Both the conditions are causes by almost similar etiology. Oliguria is seen in terminal stages of all forms of nephritis, congestive heart failure, and peripheral circulatory failure. Anuria usually occurs with complete urinary obstruction as seen in urolithiasis or acute tubular necrosis (e.g. acute mercury poisoning).

DYSURIA AND STRANGURIA

Dysuria is painful or difficult urination act, whereas, a slow and painful urination is called as stranguria. Clinically these conditions are characterized by frequent or constant efforts to pass the urine, dribbling of urine particularly in male sheep and goats, absence or presence of straining, increase in frequency of urination (pollakiuria), and occasionally, hematuria. Both conditions are difficult to differentiate clinically from each others. The clinical signs of acute abdominal pain due to diseases of urinary tract are rarely seen in animals as noted in human. But if they occur, are usually associated with over-distension of renal pelvis or ureters, infarction of kidneys, pyelonephritis, cystitis, and urethritis. Dysuria is a common sign of urolithiasis in animals. In bucks (He-goats), vocalization is commonly associated with urethral obstruction.

URAEMIA

It is the presence of excessive urinary constituents in the blood and their systemic toxic effects. This term is used to described the clinical syndrome, when there is complete loss of function of most nephrons, which occur in the terminal stages of acute or chronic renal insufficiency. Several uremic toxins have been associated with uremic process, these may include urea, parathyroid hormones, and phosphorus. The predominant clinical signs in large animals are depression, anorexia, seizures, encephalopathies, progressive weight loss, oral lesions, melena, gastrointestinal ulcers, and accumulation of excessive dental tartar.

UROLITHIASIS
Syn: URINARY CALCULI

Urolithiasis is the formation of calculi (urolith) in urinary tract (kidneys, ureters, bladder, or urethra), caused by inorganic or organic solutes, and characterized clinically by abnormalities in urination.

Renal calculi or nephroliths: Kidneys are the most common sites of calculi formation and lodging. It is characterized by signs of abdominal pain, occasionally hematuria, other signs of nephritis, but in animals, it is rarely diagnosed at antemortem examination.

Ureteral calculi: Calculi after formation in the kidneys if dislodged may reside any where in the ureter(s). It could be either unilateral or bilateral. Unilateral ureteral calculi is characterized by signs of hydronephrosis. Bilateral ureteral calculi is characterized by signs of anuria, acute renal failure, and usually ends up with death of the animal.

Vesicular or cystic calculi: Urinary bladder is the 2nd most common site of calculi formation and lodging. This site of calculi lodging is characterized by anuria or dysuria, hematuria, and distention of bladder at rectal palpation.

Urethral calculi: Urethra is the most common site for calculi lodgement in animals. It is characterized by partial or complete obstruction of urine, dysuria, abdominal pain, and if rupture of bladder, uremia, peritonitis, and death within 2-3 days.

ETIOLOGY

This condition is seen in all species, with all age groups, and animals of both sexes, but clinical disease is exclusive seen in males, particularly if castrated in young age, and those fed on concentrate rations (feedlots). Certain soil types and pasture plants containing high concentration of oxalate, estrogen, or silica are important in increasing incidence of urolithiasis. Mostly, inorganic and occasionally organic solutes precipitated out of solution on a nidus, and over a long period form a hard mass as crystals/stones or may remain as an amorphous deposits. The calculi are found in all shapes or sizes (i.e. from large to small or smoothly rounded to rough surfaced).

The etiology of urolithiasis may be grouped as under:
1. Factors which favor the development of nidus.
2. Factors which facilitate precipitation of solutes on a nidus.
3. Factors which favor concretion by cementing the precipitated salts to develop a calculus.

Nidus (nucleus): It is usually formed by a group of desquamated epithelial cells, necrotic tissues, leukocytes, erythrocytes, albumen, bacteria, or other organic substances collected in urinary tract in response to inflammation that favor the deposition of crystals around itself.

PATHOGENESIS

The pathogenic mechanisms operating in the formation of urolithiasis may be classified as under;

1. Factors favoring nidus formation; i) Local infection in the urinary tract, ii) Deficiency of vitamin-A, causing excessive desquamation of epithelial cells), and iii) Administration of estrogen which also causes excessive desquamation of epithelial cells.

2. Precipitation of solutes on a nidus; Even in highly saturated urine the solutes remain in solution form because of a number of factors, particularly, protective colloids, which remain efficient up to certain levels until the limits are increased, and then follows their precipitation. For examples;

i. The pH of urine effects stability of urinary colloids, and the solubility of some solutes. For example, the mixed phosphate and carbonate calculi being more easily formed in an alkaline rather than an acidic medium.

ii. A high level of silica in grasses can cause precipitation of silicic acid to form silicate calculi (intake of sodium chloride prevent its formation).

iii. Citrates act as buffer in urine which maintain calcium in solution form by the formation of calcium-citrate complex, but depression of citrate contents favors the precipitation of calcium salts.

iv. Continued depression of water intake or dehydration, drinking of heavy water for long period, or diet containing high phosphate contents (cotton seed meal, alfalfa hay, and sorghum) can cause concentration of urine which favors the precipitation of salts.

v. Hypovitaminosis-D with dehydration, imbalance of Ca:P ratio in diet, and ingestion of large quantity of oxalic acid are other causes of urine concentration of calcium and the formation of calculi.

3. **Factors favoring concretion;** Certain mucoproteins (mucopolysaccharides) act as a cementing agent for the solutes and thus favor the formation of calculi. These quantity of mucoproteins increases when, i) Feeding heavy concentrate feed and low roughages in feed-lot animals, ii) Implantation of diethylstilbestrol (estrogen) for fattening, and iii) High dietary intake of phosphate.

CHEMICAL COMPOSITION OF ANIMAL CALCULI

The chemical composition of calculi varies with species and sex of animal, different geological localities, and types of fodder used. There are at least 20 different crystalline substances of various minerals, reported of forming calculi, in different animals.

A. Calculi containing carbonates or phosphate of calcium, ammonium, and magnesium are common in cattle, sheep, and goats fed on heavy concentrate feed, sorghum, and clover rich pastures.

B. Silicate calculi are common in certain grass lands containing high silica contents (as high as 6%) without clover pasture.

C. Xanthin calculi are found in certain localities and is associated with certain genetic factors.

D. Calculi containing phosphates, cysteine, oxalate, and urates are common in dogs (xanthin and carbonate can also occur).

E. Phosphate and carbonate of calcium are common in calculi of horses.

PROPERTIES OF CERTAIN CALCULI

a). Phosphate calculi are white or gray in color but have a brown center.

b). Cysteine calculi are small, smooth, round, yellow color, and have a waxy appearance.

c). Oxalate calculi are white or light yellow color, brittle to hard, and elliptical in shape.

d). Urate calculi are small, brittle to hard, laminated, and yellow or tan in color.

A preformed calculi anywhere in the urinary tract can cause obstruction in the passage of urine along with many other complications. A unilateral but complete obstruction of ureter will cause hydronephrosis, and if it is bilateral, will cause uremia and death within few days. Cystic or urethral calculi can cause partial or complete obstruction in flow of urine and hence are clinically important. A complete obstruction in urine flow may end up with rupture of bladder or urethra, with escape of urine into abdominal cavity, followed with many complications (as chemical peritonitis and uremia), which may be acute enough to cause sudden death. Other complications, such as pyelonephritis, cystitis, and urethritis are also common with urolithiasis.

COMMON SITES FOR CALCULI LODGEMENT IN ANIMALS

	1st	2nd	3rd	4th
Male;				
Rams	Sigmoid flexor	Vermiform appendage	Bladder	Kidneys
Bucks	Sigmoid flexor	Vermiform appendage	Bladder	Kidneys
Bulls	Sigmoid flexor	Bladder	Kidneys	----
Stallions	Urethra/pelvic	Bladder	Kidneys	----
Dogs	Urethra (os-penis)	Bladder	Kidneys	----
Females;				
All spp.	Bladder	Kidneys	----	----

CLINICAL FINDINGS

Renal and ureteral calculi in farm animals are rarely diagnosed clinically. Abdominal pain, stiff gait, arching of back, presence of R.B.Cs. and pus cells in urine are suggestive of ureteral or renal calculi.

In cystic calculi, the evidences of dysuria, hematuria, abdominal pain, and other signs of cystitis are common. A large calculi, both in cattle or horses, can be palpated by rectal examination. Signs of abdominal pain (kicking of abdomen, treading, swishing of tail, grunting, and grinding of teeth), with complete or partial obstruction with dribbling

of blood stained urine are suggestive of urethral calculi. There may be frequent attempts to urinate with out success in passing the urine. Distended bladder can be palpated at rectal examination. The swelling resulting from calculus lodgement can some time be detected at the sigmoid flexure.

In cases of complete urinary obstruction, the sheath is usually dry, but occasionally small deposits or crystals can be found at preputial hairs. Rupture of bladder can occur, if the obstruction is not removed, which follows by the escape of urine in connective tissues and ventral abdominal wall leading to the development of chemical peritonitis. Subcutaneous swelling at umbilicus due to bladder rupture causes typical lesion called "water belly". Just after bladder rupture, the animal may look normal with absence of signs of pain or discomfort, but after some time, severe depression, subnormal temperature, and low pulse develop due to uraemia. Death may occur either soon after bladder rupture due to shock and hemorrhage, or 2-3 days later, with uraemia following a state of coma. Ruptured bladder with collapsed wall, floating or submerged in urine can be palpated at rectal examination.

In dogs, additional signs of diarrhea and vomiting (due to uraemia) may also be present. In horses clinical signs are similar to cattle. The additional signs in this species are relaxation of penis during urination and a grating while catheterization can be noted at the site of urethral calculi.

CLINICAL PATHOLOGY

In early stages of urolithiasis, there may be an increase in erythrocytes, epithelial casts, and crystals in urine. An increase in protein contents and pus cell may also be present if it accompanied with bacterial infection. An increase in BUN and creatinine is an indication of uraemia. Radiographs may help to identify the location of calculi in rams, bucks, and dogs. Chemical analysis, optical or X-Rays crystallography of a calculi may be necessary for the identification of its chemical elements (also for its future prevention and control).

NECROPSY FINDINGS

Calculi can be found in kidneys or bladder in normal animals dying due to other diseases. Uni or bilateral hydronephrosis may be an outcome of ureteral calculi. Rupture of bladder wall or urethra with escape of urine in the surrounding tissues can be identified if cause of death is obstructive uroliths. Evidences of chemical peritonitis can be seen in prolonged cases of bladder or urethral rupture.

DIFFERENTIAL DIAGNOSIS

Non-obstructive calculi can be confused with pyelonephritis or cystitis, but differentiation can be made by rectal examination of cystic calculi in large animals, or radiographs in small animals. Other causes of colic (see equine colic) are not associated with urination abnormalities. Tenesmus is often present in coccidiosis, salmonellosis, and rabies along with predominant enteric and neurologic signs in these diseases. Acute ruminal impaction with dehydration may be associated with signs of anuria, but physical and clinical examination can help to differentiate it from urolithiasis. The presence of crystals on the preputial hairs with other signs of dysuria are suggestive of obstructive urethral uroliths.

TREATMENT AND PREVENTION

The treatment of obstructive calculi is primarily surgical, primarily aimed to relieve obstruction and re-establishment the flow of urine. In dogs, the main aim in urolithiasis is to relieve obstruction. This can be achieved by passing a catheter and pushing the calculi gently down in the bladder followed by flushing the urethra with saline solution. If this procedure fails to dislodge urethral calculi then surgical correction is indicated.

It is not practicable to expect that calculi can be dissolved by medical treatment. Medical therapy is mainly aimed at normalization of metabolic status of animals with obstructive urolithiasis. Further increase in size of existing calculi and formation of new stones can be prevented by adopting some general measures. Following steps should be kept in mind while prescribing medical treatment.

i. Aminopromazine have been reported to have good effects in cases of obstructive calculi in feedlots.

ii. Administration of antispasmodic drugs in early urethral calculi will provide relief of pain and may allow sufficient relaxation of urethra for a small calculus to pass out.

iii. Elimination of infection by the use of antibiotics may be necessary for prevention of further progression of the disease.

iv. Change of urine pH (acidic to alkaline and vice versa) may help to avoid precipitation of certain type of solutes and future calculi formation.

v. Alteration of the body metabolism through use of certain drugs may be necessary for disruption of suitable environment for calculi formation.

vi. Identification of chemical nature of calculi is essential in order to avoid future intake of those chemicals in feed containing them.

vii. Feed should be balanced of calcium and phosphorus to avoid precipitation of excess phosphorus.

viii. Supplement the ration with sodium or ammonium chloride decreases the rate of magnesium and phosphorus deposition, respectively.

ix. The intake of water and subsequent urine output can be enhanced by oral administration of common salt in the feed of animal.

x. Administration of vitamin-A particularly in dry season may decrease the excessive desquamation of epithelial cells, which favors the deposition of solutes.

xi. Castrate animals at proper age, i.e. rams/bucks, at the age of 5-6 months, and bulls, at the age of 1.5-2 years (i.e. after sexual maturity) may reduce the incidence of urolithiasis. This postponement of castration till proper age allows greater development of urethra which may allow small sized calculus to pass out easily, but do not stop the process of calculi formation.

Further development of preformed calculus can be prevented by adopting certain measures. These includes;

A. FOR PHOSPHATE CALCULI

i. Control of infection by the use of antibacterials,

ii. For alkaline urine use urinary acidifiers to maintain urine pH 6.5, e.g. methionine, ascorbic acid, ammonium chloride, or phosphoric acid,

iii. Addition of sodium chloride salt in the animal feed may increase water intake and urine output,

iv. Addition of calcium chloride in diet can partially reduce the incidence of phosphate calculi, and

v. Vegetables and feed producing alkaline pH should be avoided in known cases of phosphate calculi.

B. FOR CYSTEINE CALCULI

i. Give feed to animals which having low animal protein,

ii. Raise pH to 7.8 by adding sodium bicarbonate in diet,

iii. Promote water intake to dilute saturated cysteine, and

iv. Cuprimine (penicillamine), @ dose of 30 mg/kg, decreases the solubility of cysteine and increases its excretion by forming a penicillamine-cysteine-disulfide complex.

C. FOR URATE CALCULI

i. Use of Allopurinol (xyloprim), @ dose of 5-10 mg/kg, can lower the urinary uric acid level through reducing the serum uric acid concentration,

ii. Diet should be low in animal protein and high in vegetables,

iii. Use sodium bicarbonate or sodium citrate to increase urine pH, and

iv. Decrease ammonium ions concentration and also avoid the use of urinary acidifiers.

D. FOR OXALATE CALCULI

i. Oxalate calculi should be removed surgically at first preference, and

ii. Avoid the use of pasture containing high oxalate contents.

E. FOR SILICEOUS CALCULI

i. These calculi can be prevented by adding sodium chloride in diet which reduces silicic acid concentration in urine,

ii. Feed rich in silica should not be given to animals prone to high incidence of urolithiasis.

F. FOR CALCULI CONTAINING CALCIUM SALTS

i. Calculi containing calcium can be prevented by adding sodium citrate in the feed,

ii. Feeding the animals at regular intervals and water supply a at libitum can prevent calculi containing calcium salts,

iii. Saline water should not be given for long periods to animals.

PYELONEPHRITIS
Syn: NEPHRITIS, PYELITIS

It is the inflammation of renal pelvis and parenchyma, usually caused by ascending bacterial infection, and characterized by pyuria, hematuria, dysuria, and bacteriuria.

ETIOLOGY

A variety of bacteria have been isolated from pyelonephritis. Among those, *Corynebacterium renale, C. pyogenes, Escherichia coli,* species of Hemolytic *streptococci, Staphylococcus aureus, Pseudomonas aeruginosa, Proteus vulgaris,* and some un-identified diphtheroid bacilli, were common isolates. Most of the organisms reach the kidneys through ascending infection from lower urinary tract (urethra or urinary bladder). Stagnation of urine plays important role in ascending of infection.. Infection may also reach to the kidneys through blood from primary septicemia.

PATHOGENESIS

Presence of bacteria and stagnation of urine are important factors for ascending and colonization of infection in the renal pelvis/parenchyma. Stagnation of urine may be seen if urethra or ureters are blocked with cell debris produced by local inflammatory process, physical pressure of gravid uterus or tumor, and uroliths anywhere in the lower urinary tract. Stagnation of urine for long time may result in ascending of infection to the renal pelvis, collecting ducts, calyx, medulla and cortex.

Inflammatory processes develop following colonization of bacteria, that depend upon the type of organisms involved. In acute purulent type inflammation there will be development of pyuria, hematuria, toxemia, and bacteriuria. Chronic lesions are associated with necrosis and fibrosis in the renal parenchyma and the evidences of chronic renal failure.

CLINICAL FINDINGS

The disease may be either acute or chronic, focal or diffuse, and unilateral or bilateral. In acute pyelonephritis presence of hematuria, pyuria, dysuria, renal colic, fluctuating body temperature, depression, anorexia, (diarrhea and vomiting in dogs), are some common findings. Chronic cases are usually sub-clinical and are characterized by gradual loss of body condition, poor appetite, emaciation, mild temperature, and development of pitting edema resulting from hypoproteinemia. Rectal examination may reveals thick walled bladder (cystitis), cord like ureters (ureteritis), inflamed kidneys with absence of lobulation (in cattle), and pain at palpation.

CLINICAL PATHOLOGY

In acute pyelonephritis, microscopic examination of urine may reveal the presence bacteria, leukocytes, RBCs, and other cell debris. In chronic cases, increase in pus cells, high protein contents and bacterial count without the presence of clinical signs are common. Complete blood count may reveal leukocytosis (in acute stages), hypoproteinemia, increase blood urea nitrogen and creatine contents (in chronic stages). Plain X-Rays may not be diagnostic, but contrast radiograph can help in identification of renal lesions. Fine needle biopsy technique may be used for the identification of the causative bacteria and associated histological changes.

NECROPSY FINDINGS

Thickening of bladder wall, inflamed cord like ureters, and enlarged kidneys with absence of lobulation (cattle) can be identified at gross examination. Affected renal parenchyma may reveal the evidences of congestion, hemorrhages, suppuration, ulceration, or necrotic changes. In chronic cases, the kidney(s) appears smaller than normal, may have fibrous adhesions, and infiltration of connective tissues instead of interstitial tissues at microscopic examination. Healed focal lesions in the kidneys may appear as contracted scar tissues.

DIFFERENTIAL DIAGNOSIS

Presence of pyuria, hematuria, dysuria, and bacteriuria are indicative of urinary tract infection. Dysuria and presence of traces of RBCs in the last portion of urine are diagnostic for lower urinary tract infection. If blood is mixed with whole urine, then it is difficult to set apart upper or lower urinary tract infection. Urethral calculi is characterized by anuria, dysuria, presence of RBCs, without presence of much pus cells and bacteria. Plain radiograph may help to diagnose uroliths in small animals.

TREATMENT

Specific antibacterials should be selected through the prior knowledge of the followings;

i. Sensitivity of the causative organisms,
ii. Required concentration of antimicrobial in urine and renal parenchyma,
iii. Activity of antimicrobials at different pH values,
iv) Its toxicity or contra-indications,
v) Total expenses for treatment, and
vi) Compatibility with other antimicrobials or drugs.

abolic steroids may be administered on routine basis in cases of
ronic nephrosis.

RENAL ISCHAEMIA
Syn: ACUTE TUBULAR INSUFFICIENCY

is the reduction in blood flow through kidneys resulting from
neralized circulatory failure, characterized by initially oliguria,
llowed by anuria and uraemia.

TIOLOGY

cute tubular insufficiency or renal ischaemia can be caused by
neral circulatory emergencies as seen in severe cases of
pticaemia, shock, dehydration, acute heart failure, and
moconcentration. This condition may also develop in coagulopathies
sociated with adverse drug reactions, acute hemorrhagic anemia,
ver burns, and major surgeries. Renal ischaemia can also be caused
thrombosis or embolism, in renal vessels, causing sudden stoppage
blood supply to localized or generalized renal parenchyma. Chronic
nal ischaemia has been seen in chronic circulatory insufficiency (e.g.
ngestive heart failure).

ATHOGENESIS

e pathogenic mechanisms operation for the development of renal
chaemia, in the most of the above mentioned etiology, have been
scussed previously. A sudden reduction in flow of blood and anoxia
the outcome of many of the conditions mentioned above. This may
sult in sudden vascular collapse and hypotension within the kidney.
acute reduction in glomerular filtration and tubular resorption will
ad to disruption in cellular metabolism and ischemic nephropathy.

e development of pathological changes (due to severe anoxia) varies
m local tubular or glomerular degeneration to diffuse necrosis of
nal cortices and medulla which end up in renal insufficiency.
sruption of tubular resorptive function will lead to the accumulation
body metabolites within the blood (e.g. BUN) and severe uraemia.

LINICAL FINDINGS

e clinical findings include oliguria, anuria, and other signs of
aemia. Among those anorexia, tucked up abdomen, depression,
scle weakness, hypothermia, vomiting (dogs), and in later stage
al ulcers are common. Mild attacks of colic and laminitis are
ditional signs in horses. Diarrhea, hematuria, and/or
moglobinuria are often found concurrent in acute cases. An

ammoniacal or uriniferous smell from breath can also be observed from effected animals suffering from acute renal insufficiency.

CLINICAL PATHOLOGY

An increase of BUN and creatinine levels and low urine specific gravity are good indices of degree of renal insufficiency. Metabolic acidosis can also be identified at laboratory examination.

NECROPSY FINDINGS

Macroscopically, renal cortex appears pale and swollen, and have distinct lines of necrosis at cortico-medullary junction. Histological examination may reveal degenerative and necrotic changes in tubular epithelium and glomeruli. The basement membranes of tubules can also be found necrosed and disrupted.

DIFFERENTIAL DIAGNOSIS

Evidences of oliguria and anuria in the presence of diseases characterized by circulatory failure are suggestive of renal ischaemia. Glomerulonephritis and nephrosis may have similar clinical picture without the evidences of circulatory failure.

TREATMENT

1. Treatment of the primary cause of circulatory failure as mentioned in the etiology.

2. Immediate attention should be paid to restore deficits of blood volume, extracellular fluid, and electrolytes, as required according to the cause, and maintained till complete recovery. Fluid replacement must be done with caution in animals with oliguria and anuria. Blood chemistry and urine flow should be monitored for several days for information of normal kidney function. In advanced cases of renal insufficiency, blood transfusion or isotonic fluid therapy may not have any effect.

3. Use of diuretics (in oliguria only), such as, mannitol (20%) @ dose of 200-500 mg/kg, IV, or furosemide @ dose of 1-2 mg/kg, IV, may be given only after fluid replacement therapy. Fluid therapy in oliguric animal should be monitored by simply monitoring fluid input and output.

4. Metabolic acidosis can be treated with isotonic sodium bicarbonate solution along with treatment of predisposing causes of renal ischemia.

Symptomatic and supportive treatment should be provided which ıy include the use of sedatives and tranquilizer in cases of anxiety d colic, and adequate caloric diet to reduce endogenous catabolism.

Hemodialysis in acute renal insufficiency (only if required and ssible).

ote: Use of calcium salt should be restricted in cases of acute renal haemia.

ognosis: If the renal function can not be restored within 48-72 urs (indicating acute renal failure), prognosis is unfavorable.

CYSTITIS

is the inflammation of the urinary bladder, usually caused by cterial infection, and characterized by dysuria, hematuria, pyuria, d bacteriuria.

TIOLOGY

rious causative organisms have been isolated from cystitis in imals (see pyelonephritis). These bacteria may enter the bladder her though urethra (ascending infection) or primary infection of dneys (descending infection). The predisposing factors associated th the introduction and colonization of infection are stagnation of ine and injury to the bladder wall. These may be associated with ethral or cystic calculi, advanced pregnancy, dystocia, vaginal olapse, paralysis of the bladder, and use of (contaminated) catheter d obstetric instruments.

stitis can also be associated with non-infectious causes, including ıg term feeding on Sudan grass and hybrid Sudan grass, and dicity with different fungi and Bracken-fern.

ATHOGENESIS

ormally, any bacteria entering in bladder is evacuated out by ysical emptying of bladder before colonization process. Predisposing ctors, such as trauma to bladder wall and stagnation of urine, cilitate the colonization and development of infection. The flammatory response varies from fibrinous to catarrhal, purulent, morrhagic, proliferative, or hyperplastic types, with or without velopment of toxemia or septicemia.

CLINICAL FINDINGS

Dysuria is an important sign of cystitis. It is characterized by frequent desire to urinate with passing of small quantity of urine often accompanied by grunting. The affected animal keep-on urination posture for some time after urine flow has ceased and with additional expulsive efforts to pass urine. Anuria may develop if urethra is completely blocked with pus or blood cells. The urine color may be changed form reddish (due to blood) to turbid (due to pus cells), and may has strong odor. Rectal examination may expose uniform or irregular thickening of bladder wall along with evidence of pain while palpation.

Supplementary signs of acute cystitis are moderate fever, anorexia, emaciation, and gradual loss of body condition. In dogs, additional signs of vomiting and diarrhea could also be noted. A moderate abdominal pain as evident by treading with hind feet, kicking at the belly, and swishing of tail, may also be noted particularly in horses.

CLINICAL PATHOLOGY

Microscopic examination of urine during acute stages may indicate the presence of increased number of pus cells, RBCs, transitional epithelial cells, and the causative bacteria. The urine may have a strong odor. Urine color may be red or turbid in acute cystitis but no physical abnormality of urine could be noted during chronic stages of cystitis.

NECROPSY FINDINGS

Hyperemia, hemorrhage, and edema of bladder wall may be seen in animals dying of acute cystitis. The inflammatory response may vary, depending upon the nature of the etiology, from fibrinous, purulent, catarrhal, phlegmonous, granulomatous, or hypertrophy of bladder wall. An irregular thickening of sub-epithelial connective tissues with rough and coarsely granular bladder surface may be seen in chronic cystitis. In neoplastic growth of bladder, the lesions may be of nodular, papillomata, smooth hyperplastic, or of proliferative type. In cystitis caused by Sudan grass or hybrid Sudan grass, a soft mass of calcium carbonate can be found deposited at the depending part of bladder.

DIFFERENTIAL DIAGNOSIS

Clinically, cystitis is difficult to differentiate from pyelonephritis because both the condition can develop together and have similar clinical picture. The inflamed kidneys can be palpated at rectal examination in pyelonephritis. Cystic calculi can be identified by rectal palpation in large animals, and by X-Rays or ultrasonography in small animals. Urethral calculi may cause complete or incomplete obstruction in flow of urine. In addition, distension of bladder and

obstruction in passing catheter in dogs and horses are diagnostic of urethral calculi. Prostatitis (in dogs) is characterized by dysuria, hematuria, and pyuria. The inflamed prostate glands can be identified either by digital palpation or by X-Rays examination.

TREATMENT

1. Specific treatment should be given preferably following culture and antibiogram analysis of the causative bacteria (see pyelonephritis).

2. The bladder may be irrigated by using sterile saline and antibiotics, using catheter and a syringe.

3. Change of urine pH, by using urinary acidifier or alkalizers, can decrease the growth of bacteria growing at their optimum urine pH. Diuretics should also be used for their bladder flushing effects.

4. Sedatives are recommended only if there are signs of pain in the urinary tract (Tr. hyoscyamus, or belladonna).

5. Symptomatic and supportive therapy, as and if required.

Note: Relapses after discontinuation of antibiotic therapy is common in urinary tract infection unless the treatment is continued for a minimum of 7 days and preferably for 14 days post clinical recovery. The persistence of infection is due to failure to destroy small foci of infection in the accessory glands and the bladder wall.

Chapter-14

DISEASES OF THE EYE

INTRODUCTION

The primary complaints of the owners and clinical signs of diseases of eye(s) of animals can be grouped into a) abnormal appearance of one or both eyes, b) the presence of ocular discharge, c) the presence of ocular pain, or d) total or partial blindness.

The abnormal appearance of the eye(s) or periocular region may be explained by facial or ocular asymmetry, variations in eye color, or presence of masses at the ocular surface. These can be seen in facial palsy, ocular tumors, space occupying lesions in the eye, glaucoma, strabismus, and congenital or hereditary defects of the eye(s). The variation in ocular color is observed in opacities, conjunctivitis, hemorrhage, keratitis, cataracts, anemia, poisoning, and abnormal eye pigmentation. The ocular asymmetry may be seen with orbital swellings, mass lesions of the orbit, facial nerve paralysis, or malposition of the eye(s).

Exophthalmos is the forward displacement of eye(s). It is commonly associated with a space occupying orbital mass lesion. Enophthalmos is the posterior malposition of the globe and is seen with ocular pain or loss of retrobulbar soft tissues. Microphthalmia is the smaller sized globe(s) and is mostly genetic origin.

The abnormal ocular discharge may be serous or muco-purulent and is suggestive of severity of ocular diseases. Examples are ocular trauma, misdirected cilia, foreign body, uveitis, sinusitis, and bacterial or viral infections in eye(s). Epiphora is facial wetting that results from spillage of tears onto the face. It may be due to excessive secretion of tears or obstruction in excretory duct system.

The evidence of ocular pain may be identified by the observation of blepharospasm, photophobia, periocular hyperesthesia, epiphora, and resistance to physical examination of eye(s). Ocular pain may be noted in ocular trauma, corneal ulceration, trichiasis, entropion, foreign body, traumatic uveitis, uveal prolapse (staphyloma), and ocular infections.

The signs of complete blindness are bumping into objects in the path, inability to respond to visual stimuli (light or hand movements), and other behavioral changes. Partial loss of vision in animals is difficult to determine. Tilting of head, searching nystagmus, and difficulty

maneuvering in dim light are findings of partial blindness. The blindness may be noted in corneal opacity, glaucoma, severe trauma, optic nerve dysfunction, neoplasia, and advanced cases of meningo-encephalitis.

OPHTHALMIC MANIFESTATION OF SYSTEMIC DISEASES

Infectious diseases, neoplastic, autoimmune, metabolic, toxic, and other miscellaneous disorders can blemish the eye(s). Treatment without accurate diagnosis may result in multiple adverse ocular sequelae including complete blindness and even death of animal. Different systemic diseases may have similar ocular findings, because the eye and adnexa can respond in a limited number of ways. General physical examination and laboratory examinations must be correlated with other findings to diagnose primary illness. The common findings of ocular diseases are conjunctivitis,. keratitis, uveitis, cataract, hemorrhage, opacities, and orbital changes usually in one or both the eyes. Some of the systemic diseases having ocular lesions are as under.

Borreliosis (Lyme disease); In this disease the development of uveitis, conjunctivitis, and polyarthritis have been reported in different species of animals.

Brucellosis; This may cause recurrent uveitis, corneal opacity, optic neuritis, and retinal detachment along with its typical signs. The disease may be treated with the use of antibiotics (tetracycline or doxycycline) through topical and systemic routes.

Leptospirosis; In this disease a non specific conjunctivitis and recurrent uveitis may develop along with primary lesions of the disease, including icterus, petechiation, and sub-conjunctival hemorrhages. Treatment with broad spectrum antibiotics is useful.

Miscellaneous septicemia; Ocular lesions may also develop with septicemia of known or un-known origin. Uveitis is the most common sign and usually due to direct bacterial colonization, toxins, or immunologic responses to primary infection. Use of broad spectrum antibiotic is recommended for primary septicemia.

Toxoplasmosis; Toxoplasma gondii infection may involve uvea, retina, or optic nerve, and cause granulomatous or non granulomatous type ocular lesions. Sulfadiazine and Trimethoprim combination gives good response in this disease.

Mycoplasmosis; Mycoplasmal infection particularly in goats may cause lesions in the eye also. These include conjunctivitis, keratitis, uveitis, and exudation in the anterior chamber. Tylosin and tetracycline may be used for mycoplasma infection.

Infectious bovine kerato-conjunctivitis; This disease is also called as pink eye disease and caused by Moraxella bovis infection. Kerato-conjunctivitis, uveitis, and corneal opacity and ulcers are important clinical signs of this disease.

Chlamydiosis; Conjunctivitis, rhinitis, pneumonia, and fever are common signs of chlamydial infection. The ocular discharge may be serous to mucopurulent. The causative organism respond well to tetracycline, chloramphenicol, and erythromycin treatment.

Ehrlichiosis; Ocular findings with this disease are conjunctivitis, corneal opacity, uveitis, retinitis, and optic neuritis. This disease can be treated with tetracycline. Corticosteroids needed to be used for uveitis.

Canine distemper; In this disease conjunctivitis, keratitis, optic neuritis, and blindness are common ocular lesions. The presence of viral antigens can be identified in ocular cytology examination. Un-vaccinated young dogs are of high risk in developing the ocular lesions.

Infectious canine hepatitis; In this disease uveitis is common complication in dogs. Other ocular lesions, i.e. keratitis, ocular edema, and glaucoma may also be present.

Feline infectious peritonitis; The primary ocular lesions with this disease are uveitis, keratitis, and necrotizing vasculitis. Symptomatic treatment is applied for ocular lesions in this disease.

Diabetes mellitus; In this metabolic disease bilateral progressive cataract is typical ocular lesion. Diabetic retinopathy is common ocular lesion in man. A blood glucose determination is use as screening test for the primary disease.

Neoplastic diseases; Various types of neoplastic diseases may involve different structures of eye and compromise its normal functioning. Some of them are localized masses but others may be metastatic in nature, affecting other organs as well. Some of the metastatic tumors in domestic animals are lymphosarcoma, hemangiosarcoma, squamous cell carcinoma, fibrosarcoma, adenocarcinoma, sarcoid, and transmissible venereal tumor.

A. If urine pH is acidic then sulfisoxazole, tetracycline, cephalothin, nitrofurantoin, and sulfadiazine may enhance the interstitial-medullary diffusion. In alkaline urine, gentamicin and trimethoprim have the similar effects. For ampicillin, cephaloridine, and cephalexin, pH of urine is immaterial.

Combination of sulfadiazine and trimethoprim, (Bactrim, Septran, Biotran, Tribrissin) are excreted through glomerular filtration and tubular secretion. Both of these drugs act on bacteria by interfering with folate metabolism and have synergistic action.

2. Diuretics have flushing effects in the urinary tract infection and urinary acidifiers or alkalizers change the urine pH and thus change the environment suitable for the growth of bacteria. Therefore, these drugs are considered to having bacteriostatic activity for urinary tract infections and are commonly used as ancillary therapy along with antibiotics.

Acetazolamide (Diamox), @ dose of 10 mg/kg, or Furosemide (Lasix), @ dose of 4-8 mg/kg are used as diuretics.

Monobasic sodium acid phosphate, @ dose of 60-120 gm/LA/day, in 3 divided doses, Ammonium chloride, @ dose of 10-15 gm/LA/day, Mandelic acid, @ dose of 500 mg/SA, Ascorbic acid, @ dose of 100 mg/SA, Methionine, @ dose of 200 mg/SA, or high protein diet help to decrease the urine pH. Use of Methenamine mandelate (Mandelamine), @ dose of 0.25-1 gm/SA, 4 times a day and Methenamine hippurate (Hiprex), @ dose of 1 gm/SA may help to increase the urine pH.

3. Use of enzymes, such as Streptokinase, Streptodornase, along with antibiotics are helpful in lysis of fibrin tags, deep penetration of antibiotic, and so good for treatment of chronic pyelonephritis.

4. A good plan of supportive therapy should be adopted in order to prevent further loss of body condition. Vitamin-B complex and anabolic steroids can be administered as a routine supportive therapy when there is progressive weight loss and emaciation. Any stressful activity to the animal should be avoided.

NB: Chronic cases of pyelonephritis are usually associated with polyuria hence diuretics are contraindicated in such cases. Common salt should be provided as long as there is no edema.

A urinary acidifier or antiseptic recipe contains the followings

Ammonium chloride	15-20 gm
Potassium nitrous	15-20 gm
Tr. Hyoscyamus	20-30 ml
Hexamine (methenamine)	15-20 gm
water	Q S

Sig. Mft haust, single dose for large animals.

Note: A. Cephaloridine, cephalothin, colistin, and kanamycin may cause nephrotoxicity if used with furosemide.

B. Use of acidifiers may tend to halt precipitation of crystalline sediments (in bovine). But don't use urinary acidifiers when metabolic acidosis exists.

C. In infection with urea splitting organisms, use antibiotics which are effective in alkaline pH (gentamicin, kanamycin, streptomycin).

D. Antibiotics should be used continuously at least for 7-10 days post clinical recovery.

E. Restriction of water may enhance urinary concentration of antibiotics.

NEPHROSIS
Syn: ACUTE RENAL FAILURE

Nephrosis includes degenerative changes in renal parenchyma, characterized by oliguria and uraemia in acute cases, and polyuria, dehydration, and progressive loss of body weight in chronic cases.

ETIOLOGY

The common causes of nephrosis in animals are either intake of toxic substances (called nephrotoxins), in large dosages or for a considerable length of time or development of renal ischaemia. Following are some of the nephrotoxins included in the etiology of nephrosis.

1. Accidental intake of heavy metals or inorganic salts are the main potential cause of nephrosis in all domestic animals. These include different compounds or formulation of mercury, cadmium, arsenicalis, thallium, selenium, copper compound, lead, sodium, nitrate, phosphorus, highly chlorinated hydrocarbons, ethylene glycol, and oxalate.

2. Use of certain drugs; Like antibiotics of aminoglucoside group, including neomycin (the most common cause), gentamicin, kanamycin, amikacin, and streptomycin. Other antibacterials like amphotericin-B, oxytetracycline, cephaloridine, monensin, and sulfonamides are also known to have nephrotoxic effects. Anthelmintics like benzimidazole compound (thiabendazole), phenothiazine, tetrachloroethylene, carbon tetrachloride, have nephrotoxic effects. Over dose of calcium salt may cause nephrosis in animals having dehydration. Non-steroid anti-inflammatory drugs, if used in higher dosage for long time, in horses, can cause nephrosis.

3. Certain poisonous plants such as Acorn (Oak-buds) or plants containing high oxalate contents can cause nephrotoxicity.

4. Fungal toxins (Ochratoxins and certain other mycotoxins) are also known causes of nephrotoxicity in animals and poultry.

5. Endogenous and exogenous toxaemias, by-products of excessive degradation of protein, myoglobin, and hemoglobin are known to cause degenerative changes in the renal tubules, particularly if dehydration is also present. In certain other diseases causing severe dehydration and icterus nephrosis may develop as a secondary complications.

6. Renal ischaemia caused by acute mastitis, septic metritis, abomasal torsion, toxemia, other septicemic conditions, major surgery, acute heart failure may indirectly cause degenerative changes in renal parenchyma and nephrosis.

7. Venoms of certain snakes causing slow poisoning may also end up in nephrosis.

PATHOGENESIS

Renal tubules have high metabolic rate and are very susceptible to the direct effects of toxins that cause inactivation of cellular enzyme systems. Most of nephrotoxins have degenerative effects on cells of renal tubules resulting in obstruction in flow of glomerular filtrate through them. This may result in the development of oliguria, uraemia, or acute renal failure. Aminoglucosides exert their effects by accumulation within tubular epithelial cells through urine. When their toxic amount is sequestered within the cells, cellular metabolism is disrupted, tubular swelling and their sloughing will occur. Nephrotoxicity mostly occurs in patients with acute dehydration and septicemia that are not given adequate fluid therapy. Intracellular accumulation of calcium salt can play a major role in cellular death.

In cases of renal ischemia, there is destruction of the tubul basement membrane, making the epithelial regeneration impossib (Renal cortical necrosis is a widespread tubular necrosis). In chror cases of nephrosis, there is impairment in tubular resorption solutes which may result in polyuria and albuminuria.

CLINICAL FINDINGS

In severe poisoning the clinical signs of acute nephrosis are masked other signs of primary disease (acute toxicity). In acute nephro oliguria, proteinuria, edema, and signs of uremia (i.e. anorex diarrhea, hypothermia, depression, weak pulse, and bleedi diathesis). Ulceration in oral cavity and vomiting are additional sig in dogs. Chronic nephrosis is characterized by the development polyuria, progressive dehydration, weight loss, and emaciation.

CLINICAL PATHOLOGY

In acute nephrosis proteinuria, increased serum blood urea nitrog and creatinine levels are diagnostic. In chronic cases a low in uri specific gravity with or without proteinuria are common.

NECROPSY FINDINGS

In acute cases the kidneys appear slightly swollen, pale, a edematous. The capsule can be peeled off easily. Histological presence of cloudy swelling, fatty degeneration, and tubular necro are common findings.

DIFFERENTIAL DIAGNOSIS

History of intake of nephrotoxins, characteristic clinical findings, a absence of pus cells, RBCs, and bacteria in urine are helpful findin to differentiate it with pyelonephritis.

TREATMENT

The lines of treatment adopted for nephrosis may include;

1. Specific treatment, if the cause is established.

2. Symptomatic treatment includes, the use of diuretics following wi administration of rehydration solutions, particularly in oliguria. Acu dehydration, electrolytes imbalances, and acid-base abnormaliti should be corrected first with proper isotonic fluids, particularly wh polyuria is the predominant sign. Fluid therapy is restricted in cas of oliguria when there is pitting type edematous swelling developi at the subcutaneous tissues.

3. Supportive therapy should include the use of high caloric diet order to minimis endogenous catabolism. Vitamin-B complex a

Autoimmune diseases; Ocular lesions may develop with foreign antigens or autoimmune disorders similar to allergic lesions in other organs but their occurrence in animals are rare. Examples are allergic blepharo-conjunctivitis, uveo-dermatologic syndrome, periodic ophthalmia, and ocular pemphigus.

ANTIMICROBIAL AND ANTI-INFLAMMATORY THERAPY IN OCULAR DISEASES

The corneal epithelium is the main barrier for penetration of topically applied drugs. Topically administered drugs do not concentrate in posterior segment of the eye and if it is required systemic therapy should be administered. Sub-conjunctival injection of injectable preparations only, may allow higher concentration of drugs in the cornea and anterior segment of eye.

ANTIMICROBIAL THERAPY

The selection of antimicrobial agent is based upon the nature of the organisms and their sensitivity. Therefore, bacterial culture and antibiogram should be considered before selection of antibiotic (particularly in chronic ocular lesions).

Aminoglycosides; These are bactericidal, broad spectrum antibiotics of which neomycin, gentamicin, and tobramycin are in common use. These are available for topical ophthalmic use in the form of ointments or solutions. These can be used in combination with other antibiotics.

Chloramphenicol; It is bacteriostatic, broad spectrum antibiotic, effective against a variety of bacteria, mycoplasma, rickettsia, and spirochetes. However, its use is in man is limited due to public health concerns about aplastic anemia.

Fluoroquinolones; These are bactericidal, broad spectrum antibiotics. Ciprofloxacin is commonly used in topical ophthalmic ointments.

Penicillins; They are bactericidal antibiotics and are only available for systemic use (and not for ophthalmic use). Some of antibiotics of this group are ampicillin, amoxicillin, carbenicillin, methicillin, and penicillin-G.

Tetracycline; These are broad spectrum, bacteriostatic antibiotics, and effective against a variety of bacteria, chlamydia, mycoplasma, and rickettsia. Ophthalmic preparations containing this groups of antibiotics are an excellent first choice in therapy in infections kerato-conjunctivitis.

Macrolide antibiotics; This group includes erythromycin and clindamycin. These are effective against many bacteria, chlamydia, mycoplasma, and rickettsia, and are also available for topical ophthalmic use.

Sulfonamides; These are relatively broad spectrum, bacteriostatic antimicrobial agents. However, they have been replaced by newer antibiotics for topical ophthalmic use. Combination of sulfonamide and trimethoprim may be used systemically for routine purpose. Prolong use of sulfonamides in the eye may cause toxicity on lacrimal gland and resultant development of dry eye.

Antifungal agents; Antifungal therapy is uncommonly indicated in animal practice. However, if required dermatological cream or spray (without ethyl alcohol) can safely be applied to the eye.

ANTI-INFLAMMATORY AGENTS

Corticosteroids; These are the primary therapy for most of the inflammatory conditions of the eye. Their anti-inflammatory effects are partly mediated via inhibition of phospholipase-A and subsequent reduction in prostaglandin formation. These may also be used for reducing cellular and fibrinous exudation, neovascularization, and reducing scar formation by inhibition of fibroblast proliferation. The adverse effects are delayed corneal healing, predisposition of infection, and enhance activity of proteolytic enzymes. Therefore, topical application of corticosteroids are contra-indicated in corneal ulcers.

Acetate and alcoholic preparation penetrate the cornea best and are preferred for topical ophthalmic therapy. Ophthalmic topical ointments containing both antibiotics and corticosteroids are favored over in the infectious ocular conditions. Systemic use of corticosteroids may also be indicated when there is severe anterior uveitis and posterior segment orbital disease. Some of the common corticosteroid preparations in ointment or suspension formulation contain prednisolone (1%), dexamethasone (0.1%), dexamethasone sodium phosphate (0.05%), hydrocortisone, and betamethasone.

Non-steroidal anti-inflammatory drugs (NSAIDs); The NSAIDs inhibit prostaglandin-mediated inflammation via cyclo-oxygenase inhibition and also provide analgesia. They are primarily indicated both for systemic and topical use when steroid therapy is contra-indicated, e.g. cataract surgery. Most of the preparations are for

human use and their use in domestic animals is limited (see NSAIDs). Among the three common NSAIDs drugs available, i.e. flunixin meglumine, phenylbutazone, and acetyl salicylic acid, the former is preferred particularly for uveitis.
Antihistaminic drugs are also in use in allergic conditions and therefore these can be used in veterinary ophthalmology, e.g. allergic conjunctivitis. Miscellaneous anti-inflammatory agents, e.g. Megesrol acetate, azathioprine, cromalyn sodium, and cyclosporine, if available, can be used in animal practice for the same purpose.

TRAUMA TO THE EYE(S)

Traumatic injuries to the eye(s) are common in domestic animals. This includes trauma to soft tissue of the eye as well as orbital rim and supra orbital process of the frontal bone. Trauma to the eyelid and nictitating membrane may range from simple swelling (blepharo-edema) with or without hemorrhage to extensive laceration and avulsion. Orbital cellulitis and exophthalmos may result from traumatic puncture of eyelids, nictitating membrane, and conjunctiva by hooking on bolts, nails, chains, wires, fixed objects, or locks.

The onset of swelling may be sudden. Animal may be off feed and feel much discomfort. In untreated cases complications such as severe chemosis, keratitis, cicatricial entropion or ectropion, and conjunctivitis may follow. For therapy the basic principles of wound treatment should are followed. It includes suturing, local use of ointments containing antiseptics, antibiotics, and anti-inflammatory (DMSO) drugs, ice packs, and systemic use of NSAIDs.

Trauma or laceration to the conjunctiva may result in chemosis and hemorrhage in the eye. These may resolve without medical therapy, however, antibacterial eye ointments may be used to prevent secondary infections. Corneal injuries, lacerations, trauma to the lens, vitreous humor, retina, and optic nerve may be treated accordingly, keeping in mind the nature and extent of the insult, complications encountered, intended use of animal, its economic value, and financial considerations.

07/06/2009

KERATO-CONJUNCTIVITIS

It is the inflammation of conjunctiva (both bulbar and palpebral mucosa) and cornea, caused by a variety of agents, both infectious and non-infectious origin, and characterized by hyperemia, swelling, blepharospasm, photophobia, and ocular discharge.

ETIOLOGY

A number of etiological agents are known to cause kerato-conjunctivitis in different species of animals. Some of them are as under.

Viral causes; Infectious bovine rhinotracheitis, Malignant catarrhal fever, Bovine viral diarrhea, Blue tongue, Equine viral rhino-pneumonitis, Canine distemper, Infectious canine hepatitis, and Feline infectious peritonitis.

Bacterial and Rickettsial causes; Infections with various Mycoplasma species, Moraxella bovis, Leptospira spp., Listeria monocytogenes, Streptococcus equi, and Rickettsia conjunctivae.

Mycotic infections; Mycotic infection with Aspergillus, Candida spp. and other fungi are known to cause kerato-conjunctivitis in different species of animals.

Parasites; Different eye worms, such as Onchocerca spp., Thelazia spp. Filarid worm, Trypanosoma spp., etc. may produce kerato-conjunctivitis and other ocular defects in different animals.

Physical or chemical agents; Kerato-conjunctivitis may also be caused by physical trauma associated with accidental blow or introduction of dust, smoke, awns, thorns, other foreign bodies, and irritant chemicals in the eye(s).

Allergic conjunctivitis; It may be noted when introduction of foreign protein, pollens, or drugs locally into the sensitize mucosa.

CLINICAL FINDINGS

This disease may be unilateral or bilateral, local or systemic of origin. The clinical signs of acute conjunctivitis are variable, and include excessive lacrimation (weeping eyes), injection of conjunctival vessels, chemosis, keratitis, photophobia, cloudiness of cornea, and blepharospasm. The discharge may be lacrimal, serous, to mucopurulent type depending upon the cause. There may be systemic involvement along with inflammation of regional lymph nodes in acute cases. In chronic cases there may be the presence of epiphora, hemorrhages, formation of follicles, or masses at the conjunctiva without much hyperemia and systemic reaction.

Blepharitis inflamation of eyelids

CLINICAL PATHOLOGY

In conjunctival scraping or swabs many neutrophils along with the causative bacteria may be observed in acute cases and lymphocytes and necrotic cells may be seen in chronic cases.

TREATMENT

The line of treatment includes the use of topical ophthalmic ointment based broad spectrum antibiotics or non-irritant antiseptics, particularly in kerato-conjunctivitis of infectious origin. Infected eye may be washed initially with non-irritant antiseptics or using normal saline before instillation of antibiotic. Antibiotics or steroids may also be used by sub-conjunctival injections. Foreign body should be removed with care before medical treatment. Blepharospasm can be relieved by instillation of 1% atropine sulfate. In allergic cases (and acute conjunctivitis) eye ointment containing steroids (and antibiotic) may be recommended. Surgical intervention is required in removing maldirected hair, ocular masses, and parasites. Vitamin-A may be recommended as supportive therapy.

CATARACT

It is the opacity the iris and lens mostly due to inflammatory origin. It may be primary or secondary and unilateral or bilateral in occurrence. In ruminants the cataract is seen in specific diseases of infectious origin, e.g. Bovine viral diarrhea and blue tongue. The other causes are traumatic injury to lens, instillation of irritant chemicals or drugs, and as complication of diabetes mellitus. It may also be seen due to senility. A genetic or congenital origin opacity of the lens (complete or incomplete) may be seen at birth in different species of animals.

The cataract may be observed as white spot of milky appearance of the lens by visual examination of the eye. It may be classified according to the extent to which a fundus reflex is present, either partial or complete. Cataractous changes are observed as dark areas seen within the field of reflected light. The affected lens may appear bluish, white, or gray. There may also be luxation of the lens. Depending upon the extent of lesion varying in degree of blindness will be the outcome. In congenital cataract, complete or incomplete opacity of lens may be seen at birth. In food animals practice the treatment of this condition is considered wasteful.

GLAUCOMA

It is the increase in intraocular pressure beyond that compatible with vision. It may be caused by either excessive production of aqueous fluid or blockage in its drainage. The glaucoma may cause damage to optic disc and retinal epithelium, which may result in blindness. In the affected eye the eyeball appears enlarged, injected conjunctiva, opacity of cornea, irreversible blindness, dilatation of pupil with loss of pupillary reflex, serous to seromucoid discharge, and pain at palpation. The intraocular pressure can be checked by tonometer or digital pressure.

The treatment endeavors for this condition include local use of miotic drugs (pilocarpine), carbonic anhydrase inhibitors (acetazolamide), sympathomimetics (epinephrin), use of hyperosmotic agents (mannitol and glycerine), and provision of aqueous drainage. For the later, an[illegible] bypass surgery has been shown to be of value in both dogs and man.

OCULAR PARASITES

A number of parasites (adult or intermediate stage) can invade eye(e) of animals, causing vision damage of varying degree, and reducing their economic value. Some of the major parasites of eye are as under.

Ocular Onchocerciasis; Onchocerca microfilaria, a non infective intermediate stage, during accidental migration can infect cornea and conjunctiva (of horses). It can incite an acute inflammatory response within the infected eye. The clinical disease is characterized by kerato-conjunctivitis particularly at temporal limbus, chemosis, hyperemia, lacrimation, and blepharospasm. The line of treatment is directed toward the control of inflammation and elimination of parasite. For conjunctivitis, local, sub-conjunctival, and system use of corticosteroids may be used. Antibiotics may be recommended for the control secondary infection.

Ocular Habronemiasis; Larvae of different spp. of Habronema are deposited on eye while feeding of fly on ocular discharge. These migrate to conjunctiva and other deeper tissues and produce a local granulomatous inflammatory reaction. The lesions are usually at the medial canthus in the form of proliferative non-healing wounds. Demonstration of the larvae in the granulomatous lesions and fistulous tract is diagnostic. Treatment should include local use of anti-inflammatory drugs and systemic of anthelmintics (Ivermectin). Fly control during their active season is also recommended

Ocular Thelaziasis; Infestation of different species of Thelazia (nematode) in the conjunctival sac of different animals is a commensal in occurrence. The characteristic signs are kerato-conjunctivitis, edema, and ulceration of cornea. The motile parasite (8-19 mm long of milky white color) can directly be seen in conjunctival sac. The parasite can be remove manually with saline flushing or forceps by using local anesthesia.

Ocular manifestation of Nasal bots; Larvae of Oestrus ovis can migrate through nasolacrimal duct and enter conjunctival sac and cause local inflammatory reaction, including erythema, chemosis, and epiphora. Treatment consist of mechanical removal of larvae and systemic and topical use of insecticide (organophosphate).

Ocular manifestation of Trypanosomiasis; Many species of Trypanosoma can penetrate conjunctiva causing local inflammation. Demonstration of organism in blood smear is necessary to suspect their ocular involvement. Treatment consists of systemic use of trypansomicidals (diminazene salts) and local use of anti-inflammatory drugs.

Chapter-15

DISEASES OF THE EAR

OTITIS

Inflammation of external ear is called as otitis externa, middle ear as otitis media, and internal ear as otitis interna. Otitis media-interna is a common condition seen in animals.

Otitis may develop due to infection either with bacteria (Streptococcus and Staphylococcus spp., Pseudomonas spp., and E. coli), fungi (Otomycosis), or parasites (Otodectes cynotis in dog and Psoroptes cuniculi in cattle). The bacterial infection may spread from otitis externa or naso-pharyngitis to deeper structures to cause otitis media-interna. In ruminants otitis media-interna can develop as complication to pneumonia caused by infection with Pasteurella and Hemophilus spp.

Head shaking, ear rubbing, head tilting, constant horizontal nystagmus, and uneasiness are common signs of ear infection. Exudation and foul smelling may be observed from the ear. Lesion may unilateral or bilateral, acute or chronic. Suppurative otitis is characterized by thickened mucosa of vestibular membrane and accumulation of thick fluid in the labyrinths. Complications such as ear hematoma or facial nerve paralysis may also develop. In acute cases signs of systemic involvement as fever, anorexia, decrease production may also be noted. In chronic cases ear canal could be block by inflammatory or granular mass and may end up with partial or complete deafness. Rule-out of this condition is required from Listeriosis, meningo-encephalitis, and space occupying lesion (parasite, abscess, tumor) in the brain.

Line of treatment include the provision of proper drainage of exudate, local use of specific antibiotics, antifungals, and acaricidal drugs. Antibiotics and analgesics should also be used systemically in acute otitis media-interna. Chronic cases may need surgical drainage.

EAR HEMATOMA

It is a common condition of dog, sheep, goats, and those species and of animals having pendulous ears. The trauma to the ear is the common cause. It may be associated with blow, biting, and repeated shaking or