Advanced Pathology and Treatment of Diseases of Domestic Animals

C.D.N. Singh
B.V.sc. &A.H., M.sc. (Vet.), Gold Medalist, Ph. D., F.R.V.C.S. (Sweden)

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(With Special Reference to Etiology, Signs, Pathology and Management)

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Dedicated to

Lord Pawan Putr Hanuman Ji and to the memory of Late Veena singh, wife of the author

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राजभवन, पटना
RAJ bHAVAN
PATNA, 800 022

23 February, 2007

MESSAGE

I am glad to note that the book "Advanced Pathology and Treatment of Diseases of Domestic Animals" written by Shri C.D.N. Singh will be usefull for veterinary students. The user will find the book useful.

I wish success of the publication.

(R.S. Gavai)

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संदेश

'Advanced Pathology and Treatment of Diseases of Domestic of Animals' के लेखक भूतपूर्व विश्वविद्यालय प्राध्यापक, डा० चन्द्रदेव नारायण सिंह, व्याधि विभाग बिहार भेटनरी कॉलेज पटना के इस पुस्तक लेखन प्रयास को जानकर अत्यन्त खुशी हुई। मुझे पूर्ण विश्वास है कि इस पुस्तक से पशुचिकित्सा विज्ञान के छात्र, शिक्षक एवं शोधकर्त्ता लाभान्वित होंगे। इन शिक्षकों का यह कार्य प्रशंसनीय है और राजेन्द्र कृषि विश्वविद्यालय के कुलपति शिक्षकों के पुस्तक लेखण कार्य के उत्साहबर्धन के लिए धन्यवाद के पात्र हैं।

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(नरेन्द्र सिंह)
कृषि मंत्री
बिहार सरकार,
पटना

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MESSAGE

Advanced pathology and treatment of disease of domestic animals has been written mainly for the under - and post graduate students but it is also intended for use of teachers and diagnosticians of diseases in the animals. Dr. C. D. N. Singh has kept in mind the needs and interests of the students and veterinary practitioners by giving a relevant coverage of symptoms, lesions, diagnosis and management of diseases of the animals.

K. R. Maurya
(Vice- Chancellor)
Rajendra Agricultural University,

Pusa (Samastipur) - 848 125
10-9-2005

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Preface

The book grew out of more than three decades of teaching and the schematic structure of the text has been designed to cater to the needs and problems of the students in both undergraduate and postgraduate courses in the veterinary colleges, diagnosticians and veterinary practitioners engaged in the diagnosis and treatment of animal diseases. The VCI syllabi followed in different agricultural universities have been kept in view in preparing the manuscripts of this book for inserting pertinent pathological changes noticed in different animal diseases and relevant information on the management and treatment needed with the object of making this book of high value to the students, teachers and veterinary practitioners. The book has been written in a simple easy and lucid language with more emphasis on the symptoms, lesions and treatment of diseases. The material in the advanced pathology and treatment of diseases of domestic animals is organized in a manner so that the students are introduced to symptoms and lesions of diseases and their relevance to diagnosis, chemotherapeutic and Prophylactic measures.

Different tables on symptoms and lesions of animal diseases, veterinary drugs and vaccination schedules have been incorporated in the book for quick diagnosis, treatment on time and prophylactic steps against such diseases.Molecular imaging is an important adjunct to today's pathology and medicine helping the detection of disease processes much earlier with information on the origins of disease realated pathways, targets and adoption of proper methods for treatment and management of the root cause of diseases. It, thus, revolutionises the usual practice of treatment through symptoms by adopting technology driven techniques like PET, CT, SPECT, ultrasound, MRI and optical imaging etc. in patients.

My indebtedness lies to various scholars and pathologists whose views have been included in this book. I am grateful to Dr. P.B. Kupuswamy, former Principal, Bihar Veterinary College, Patna for help and encouragement in writing this book. I lay no claim to any originalities and welcome suggestions from users to improve its qualities. I wish to thank Dr. S.P.Verma, Principal BVC, Patna. Bihar, Dr. S.R.P Sinha, University Professor, B.V.C Patna and Dr. S.V Singh College of veterinary science, NDUAT, Kumar Ganj, Faizabad, U.P in preparing the manuscript of the book. My thanks are also due to ***Somya Raj***, Shalini Raj, K-3, P.C. Colony, Kankarbagh, Patna, ***Neha***, West Ashok Nagar Road No-1, Kankarbagh Patna-20 and ***Amit kumar***, R.M.S Colony, Kankarbagh, Patna-20 for its meticulous composing. Pathology, an observational science, is combined with a live demanding subject of medicine for making this book as stimulating and exciting for the veterinarians.

C.D.N.Singh

CONTENTS

Chapter 1
Diseases

These are reactions to injurious agents in the bodies of man and animals. Various diseases are caused by different animate factors like bacteria, viruses, fungi and protozoa etc. The pathogens (microbes) exist in the bodies of susceptible animals solely as obligate parasites. The existence of the organisms for a short period outside the body of animals is noticed in some cases as seen in *Mycobacterium tuberculosis* infection. Both saprophytic and parasitic existence can be noticed in certain bacteria e.g., *Erysipelothrix rhusiopathiae* in pigs. Saprophytes are such bacteria, which can live upon dead organic material of both the plant and animal origins. Infection (from the Latin infiecere) is that condition in which living agents enter the animal body and a disturbance of function, with or without tissue abnormalities, in any part is set up. A disturbance of function is recognized in the form of symptoms. Facultative pathogens are those organisms which are capable to produce diseases in the state of lowered vitality. The organisms existing on the external surfaces of the body and in the mouth, anus, vagina etc., in usually harmless state are called commensals. An infectious disease results from presence of living organisms in or on the animals' body with the capability to produce a state of disturbed functions (i.e., symptoms). When an infectious disease spreads from one individual to another by direct or indirect contact, it is said to be a contagious disease. All contagious diseases are said to be infectious, but at the same time, all infectious diseases are not considered to be contagious. Some infections may be highly contagious, others may be slightly contagious and a few others may not be at all contagious. The facultative pathogenic bacteria behave as secondary

invaders in some diseases i.e., *Brucella bronchiseptica* in canine distemper and *Pasteurella suiseptica (P. multocida)* in swine fever. When there is a departure from a state of health, the condition is called a disease. The disease spreads by two basic routes. The first route is the horizontal spread of disease, which means a spread of disease between infected animals and others. This involves contact of an infected animal with the healthy ones through the contaminated litter, sand and airborne dust particles. The second route of spread is a vertical one which means the passage of the disease producing agents from parents to offsprings through the eggs etc. Pullorum disease, lymphoid leucosis and mycoplasmosis are examples of vertical spread. Vertical spread of a disease is prevented by elimination of the infected parents.

The conditions of toxaemia and septicaemia developing in the infectious diseases are given below:

1. Toxaemia

It means the presence of toxins (derived from bacteria or produced by the body cells) in the blood stream of animals. This definition does not cover the toxic substances produced by plants and insects or ingested organic or inorganic poisons. Toxins may be exotoxins, endotoxins and metabolic ones. In toxic shock, there is a peripheral vasodilation, fall in blood pressure, pallor of mucosa, hypothermia, tachycardia, pulse of small amplitude and muscle weakness. It is commonly associated with bacteraemia or septicaemia. Microscopically, degenerative changes are noticed in the organs like liver, kidneys (glomeruli and tubules), heart and adrenal glands.

2. Septicaemia

It is a condition characterized by toxaemia, hyperthermia and presence of bacteria, viruses and protozoa in the blood stream. Bacteriaemia refers to the state of presence of bacteria in the circulation for transitory periods. But in septicaemia, the causative agents i.e., bacteria and

viruses etc., are present in the blood throughout the course of the disease. Anthrax, pasteurellosis, salmonellosis, leptospirosis, neonatal septicaemia and hogcholera are examples of septicaemia.

Special septicaemic states arise from radiation (because of injury to bone) and congenital defects in the immune system in the body. Immunological suppression due to corticosteroid therapy can also cause septicaemia.

Toxaemia, high fever and causative agents (like bacteria or viruses) are seen in septicaemia in the body. The infectious agents damage endothelial cells and cause haemorrhages in the organs. Toxins are not produced by viruses but they damage the tissue cells themselves or through the products of tissues killed by the viruses. In septicaemia , the causative bacteria can be isolated from the blood stream. Leucopenia or leucocytosis can be found in the patients of septicaemia. Postmortem examination of the septicaemic cases shows subserous and submucosal haemorrhages. Emboli of the infectious agents can be found in different organs. Subepicardial and subendocardial petechiae are also seen in the septicaemic state.

Pathological lesions

These are important bases to identify and establish a disease and also to correlate it to the suspected biological agents. The lesions or pathological abnormalities caused in the body of animals by different biological factors may be different in different diseases. The lesions may be quite characteristic specific and pathognomonic in certain diseases as seen in cases of (1) rabies, (e.g., Negri bodies in neurons) and (2) Gumboro disease (e.g., reddened, haemorrhagic or enlarged bursa of Fabricius) or may be of a general character. These could be detectable in different diseases and can be caused by different biological factors. Some of the lesions in bacterial and viral diseases are as follows:

(i) Bacterial Diseases

Lesions in animals are caused by both simple and higher bacteria and can be narrated separately in the following manner :

(a) Lesions caused by simple bacteria (order *Eubacteriales*)

Pasteurella multocida type I causes exudation, petechiae or ecchymosis, oedema, congestion and haemorrhagic or purulent inflammation.

Some effects of certain bacteria are as under-

(i) Haemolysis of red blood cells due to haemolysin (a toxin produced by many bacteria). For example, *Clostridium perfringens* type A and *Cl. haemolyticum* cause haemolysis of red cells.

(ii) Destruction of leucocytes by leucocidin released from certain bacteria.

(iii) Tissue destruction by localization of organisms in certain organs, for example *Corynebacterium renale* in kidneys in cattle and *Erysipelothrix rhusiopathiae* in the valves of the heart in pigs.

(b) Lesions caused by higher bacteria (other than *Eubacteriales)*

These bacteria grow slowly and progressively in the tissues of susceptible animals over a long period and produce chronic inflammatory changes as found in TB, glanders, brucellosis, Johne's disease, actinomycosis and actinobacillosis etc. There is a massive destruction of tissues affected by many of these agents producing necrotising toxins. Different inflammatory cells such as monocytes, lymphocytes, eipthelioid cells, reticuloendothelial cells and giant cells are noticed in affected tissues. These lesions are usually encapsulated by proliferated fibroblasts and lesions look like granulomas or granulomata (characterized by nodules or granules). Such granulomas arising from infectious processes are called infective granulomata (for

example, nodules in tuberculosis etc.). In Johne's disease caused by *Mycobacterium paratuberculosis*, there is an accumulation of plasma cells and epithelioid cells etc., in the mucosa and these processes do not reach the stage of necrosis and caseation. In other words, they do not go beyond the stage known as symplasma stage.

In the diseases caused by higher bacteria, marked debility, anaemia and emaciation are noticed in the affected animals.

(ii) Viral Diseases

Different viruses show affinity for different tissues in body. Such affinity is called tropism. FMD virus shows affinity for epithelial tissue, rabies for nervous tissue and rinderpest for different kinds of tissues and these viruses are called as epitheliotropic, neurotropic and pantropic respectively. Viruses may produce degenerative or necrotic changes in the cells of affected animals or may stimulate the cells to proliferate in uncontrolled manner to give rise to tumour formation (neoplasia) as noticed in cases of bovine viral papilloma, Shope's papilloma and avian leucosis complex. Ulceration, necrotic, exudative, congestive and oedematous changes are produced by viruses in several species of animals. The viruses of equine encephalomalacia and rabies produce degenerative changes in nervous tissues. Characteristic changes in such tissues like neuronophagia, satellitosis and perivascular cuffing (accumulation of lymphocytes around blood vessels) are the main microscopic changes. Tissue tropism is also influenced by certain deficiency, for example, vitamin B deficiency enhances tropism in human beings to the virus of poliomyelitis. In table 1 the positions of different inclusion bodies in the cells or tissues of affected animals in some diseases are as follows :

Table 1 : Diseases and Locations of Inclusions in the Cells

	Diseases	Positions of Inclusions in the Cells
1.	Rabies	Intracytoplasmic inclusions
2.	Canine destemper	Intranuclear and intracytoplasmic inclusions
3.	Pox	Intracytoplasmic (e.g. Bollinger bodies in fowl pox and Guarnieri bodies in small pox)
4.	Infectious laryngotracheitis	Intranuclear bodies
5.	Infection canine hepatitis	Intranuclear inclusions
6.	Rinderpest	Intranuclear and intracytoplasmic inclusions
7.	Swine fever	Intranuclear inclusions. Not considered to be specific

(iii) Fungal Diseases

Fungal infections can occur in the skin, lungs and mucous membrane of the body of the susceptible animals. Toxic substances are produced by the fungi in the tissues or cells in the body as seen in cases of aspergillosis, coccidioidomycosis and histoplasmosis.

The mycoses are diseases caused by fungi which infect both man and animals. Some fungal diseases are contagious (i.e., dermatophytoses - ring worm). Dermatophytoses are found in the skin of the individuals. Systemic mycosis (resulting from inhalation of spores etc.) may cause pulmonary lesions. Diseases affecting the skin or mucous membrane of the body may be chronic.

Fungi can be demonstrated in pus etc., by adding a drop of 10 per cent KOH or NaOH to a little amount of pus or sputum on a slide. These two are mixed together and examined under low or high power objectives of the

microscope at once after a gentle heating. Fractions of tissues of the diseased organs may be stained by the periodic acid Schiff (PAS) or methenamine silver stains to detect the fungi in organs. Inhalation of spores from a culture due to improper disposal of fungal plates, debris, stale bread or food etc., is extremely hazardous for man.

The mycetomas are fungal infections in the subcutis of the body in man and animals, but the infections may spread to the adjacent bones as seen in the cases of maduromycosis and rhinosporidiosis.

Systemic mycoses may affect different systems of the body and are exemplified by blastomycosis, cryptococcosis and histoplasmosis etc. Nearly identical tissue reactions are seen in hosts affected by very closely and biologically related fungi or higher bacteria. Toxins are produced in the disease like aspergillosis, coccidioidomycosis and histoplasmosis in the affected individuals. Granulomas seen in aspergillosis in chickens are characterized by necrotic centre surrounded with giant cells of the foreign body type, lymphocytes and connective tissue in the affected tissues. Septate branching of fungi are seen in such lesions. In blastomycosis, nodules containing purulent exudates are found in the lungs and there is an infiltration of epithelioid cells in the nodular lesions. Neutrophiles and lymphocytes are also found in such lesions. Caseation necrosis and proliferation of fibroblasts can be seen in the infected tissues.

Lesions in Metazoal Parasitic Infections

Different types of tissues reactions are produced by the metazoan parasites which produce injuries or damages etc., in the body of the hosts. These are as follows :

(i) Mechanical injuries (invasions, displacement and destruction etc.) in the infected organs or tissues are seen in the cases of kidney worm infection in swine or lung worm infection in cattle. Intermediate stages of

Trichinella spiralis produce mechanical damages in the affected muscular tissues.

(ii) Utilization of the nutrients or food by the metazoan parasites in the affected hosts. Such harmful effect is seen in cases of infections of tape worms and ascarids.

(iii) Development of neoplasms – *Spirocerca* lupi produces tumours in the oesophagus of dogs. Sarcomas develop in the livers of rats affected by *Cysticercus fasciolaris. Schistosoma haematobium* produces tumors in the urinary bladder of human beings.

(iv) Obstructions in the ducts or passages can arise from liver flukes in bile ducts, *Spirocerca lupi* in the oesophagus, ascarids and tape worms in intestines and *Dioctophyma renale* in kidneys.

(v) Attachment to or utilization or destruction of the vital tissues. This is seen in abomasal mucosae in cattle and in small intestines due to *Trichostrongylus axei* and hookworm infection respectively.

(vi) Ova of schistosomes (flukes) or dead larvae of nematodes like *Toxocara canis* and *Dirofilaria immitis* behave like foreign bodies in the tissues. Toxins like haemolysin, histolysin and anticoagulants are produced in infections due to hook worms, stomach worms and strongyles. Ascarids and stomach worms are known to devour the tissue of the hosts.

Lesions in the Protozoal Diseases

Affinity to different types of tissues is noticed in certain protozoal infections. *Babesia* spp. are known to haemolyse red cells and produce haemolytic jaundice in animals. Haemorrhagic or necrotic lesions are produced in epithelial cells of the intestinal mucosa in coccidial infection. *Eimeria steidae* are known to cause papillary ingrowths of cells of the biliary passages in the liver of rabbits.

Summing up, the somatic tissues react to animal or plant

pathogens in different ways. When the individuals succumb to the injurious agents, the different kinds of the lesions are noticed in the body and these can be correlated to various kinds of symptoms in the patients during life. The bodies can show degenerative, infiltrative, proliferative and regenerative changes in the tissues affected by different injurious agents. Some cells show hyperplastic reactions (e.g., viral neoplasia). Rise in the eosinophilic count is seen in heliminthiasis. Granulomatous lesions are seen around migrating parasites and infiltration of eosinophiles, lymphocytes and reticoloendothelial cell is seen in such lesions. There can be granulomatous encepsulation of the parasites in the intestinal nodular disease (Oesophagostomiasis) and necrotic changes are seen in the centres of such nodules.

Encysted parasites or trichinae undergo degenerative and calcareous changes. Haemorrhagic enteritis is seen in hook worm infection. In toxoplasmosis, there can be presence of mononuclear and giant cells etc., in lesions. In many bacterial diseases, production of antibodies in the body of the infected individuals is noticed. The bodies react to bacteria and fungi through manifestation of purulent or proliferative reactions leading to encapsulation or fibrosis. Lymphocytes or giant cells can be seen in such lesions. Animals react to viruses or simple bacteria by exhibiting fever, exudative or infiltrative changes in the tissue or organs. In many viral diseases, lymphocytic infiltration is seen in the tissue of the brain (e.g., equine encephalomyelitis). In bacterial infection, agglutinins, precipitins, opsonins and antitoxins etc., are produced in individuals' bodies.

Chapter 2
Bacterial Diseases

Anthrax

It is a febrile and highly fatal septicaemic disease of mammals (especially herbivores like cattle and sheep). The infectious organisms are noticed in the blood films singly or arranged in chains of twos or threes in the affected animals. These organisms exist as an obligate parasites but they can also exist in the soil which may remain infective for several months. Humans handling the diseased animals or animal products may be infected through cuts in the body with development of malignant pustules in the skin or through inhalation of spores into the lungs (woolsorter's disease). Ingestion of the contaminated materials also leads to the occurrence of anthrax in the animals. A few hours before death, the state of bacteraemia is caused by these organisms in the infected animals to be followed by the state of septicaemia. A fulminant septicaemia is seen in cattle, sheep and horses but swines, dogs and cats are resistant to anthrax infection. In swines, the disease is localized and confined to the regional lymph nodes e.g., cervical lymphnodes. The anthrax bacilli fail to grow in the body due to lack of oxygen. Organisms like *Clostridium septique, Cl. sporogenes* and *Cl.welchii* grow rapidly in the dead bodies of animals after entering from the gut and anthrax bacilli undergo degenerative changes and disappear from the carcasses within one to three days. While diagnosing anthrax in animals, the anthrax bacilli must be differentiated from putrefactive or other postmortem invaders which may form very long chains in the stained films. *Bacillus anthracis* fails to grow in long chains in the blood of the infected hosts before death.

These infected animals may die without any overt sign of this disease. Incubation period of anthrax infection varies from 1 to 3 days and death may occur within a few hours or a day. Peracute cases may die even within an hour or two. Anthrax may last over 2 to 4 days in the subacute cases. Subcutaneous oedemas are found in the neck, breast and abdomen. These swellings are hot and pit on pressure and yellow serous gelatinous fluid may escape from such swellings. In the abortive cases of anthrax, afebrile reaction can be seen in the animals. The main changes in the dead cases of sudden death are tarry blood at the natural orifices, lack of rigor mortis and blood clots in the blood vessels.

PATHOLOGY

Gross Appearance

Cattle

(i) A yellow gelatinous oedema in the abdominal or throat region at the point of entrance of bacteria.

(ii) Haemorrhagic gastroenteritis, oedematous and haemorrhagic serous membranes.

(iii) Haemorrhagic lymph nodes may have dark colour like spleen.

(iv) Spleen is greatly enlarged (splenomegaly) with dark and black pulp containing unclotted blood. It may be friable or its capsule may rupture. There is a massive haemorrage in the spleen. Spleen may be normal in rare cases.

(v) Liver, kidneys and intestinal tract are congested, swollen and haemorrhagic.

(vi) Trachea and bronchi are filled with frothy blood stained material.

(vii) Blood is dark in colour but does not clot firmly and rarely a string or blood clot can be found in the case of an anthrax.

(viii) Tarry blood discharge at the mouth and other body openings.

(ix) Haemorrhages on mucous membranes of the stomach and intestines.

(x) Throat lesions are not common in cattle but there is a strong tendency of the carcass to undergo putrefaction.

Sheep

The main changes are :

(i) Splenic enlargement is not a characteristic change.

(ii) Presence of gestroenteritis.

(iii) Rapid putrefaction in the carcass.

(iv) No throat lesions are found in the sheep

Equines

One can notice the following :

(i) Presence of throat swellings.

(ii) Oedematous fluid in the subcutaneous and intermuscular tissue.

(iii) Swollen and congested lymph nodes.

Pigs

The main changes seen in pigs are as under :

(i) Splenic enlargement is not seen.

(ii) Oedema and haemorrhage in the pharynx and cervical lymph nodes. In old cases, lymph nodes may be enlarged and firm in consistency.

Microscopic Appearance

Anthrax bacilli are large rod shaped organisms which can be found in the blood and affected tissues. Splenic architecture is not visible due to numerous red cells. Trabeculae are visible amidst red cells and nuclear debris. Splenic sinuses and cords of Bilroth are filled with red cells and pulmonary oedema is noticed. Toxins produced by anthrax bacilli are known to produce increased permeability of the blood vessels. In pigs, there is focal necrosis surrounded

by granulation tissue in the lymph nodes. The toxins produced by anthrax bacilli are lethal and produce oedema, tissue damage, acute renal failure, terminal anoxia mediated by the central nervous system and death from shock.

Diagnosis

It is based on symptoms, lesions and examination of the blood films of sick animals and identification of *Bacillus anthracis.* Death of several animals (all of sudden) in a herd or farm makes one to suspect of anthrax infection. Monoclonal antibody fluorescent conjugates, cultural examination and Ascolis's test provide strong bases to confirm anthrax infections in animals. The incidence of sudden death in the cases of haemorrhagic septicaemia and black quarter may be confused with the death pattern seen in the outbreak of anthrax in animals.

The main methods of diagnosing the anthrax infection are as under :

(1) Direct Smears

The blood films are made with the oozing blood from a small cut made in the ear vein. It is stained with 1% methylene blue solution and another film by Gram's method. The film is examined for Gram positive square ended rod with distinct capsule. A granular purple ground work arising from distntegration of imperfectly fixed capsules is recognizable in the stained organism of anthrax by 1% methylene blue solution. Anthrax bacilli in short chains surrounded by a common capsule are noticed in the blood films of anthrax patients.

(2) Cultural Examination

Inoculation of heart blood, liver etc., is made on to blood agar to see the colonies of *Anthrax bacilli.*

(3) Animal Inoculation

0.5 ml of 24 hour broth culture is injected subcutaneously

into a mouse or guinea pigs. These animals die in about 48 hours in positive cases. Capsulated organisms are noticed in the blood films of the sick animals.

(4) Examination of Decomposed or Putrified Material

When the carcass of the dead animal is putrified, Ascoli's test is done to diagnose the anthrax cases.

Treatment/Management

In case of either suspected or confirmed diagnosed cases of anthrax, antibiotics and anthrax antiserum can be used. It is better to use Procaine penicillin and broad spectrum antibiotics such as oxytetracycline. However, Penicillin is a drug of choice in the cases of anthrax. Since anthrax is a septicaemic disease with high fever, so antipyretics such as Bolin inj./Novalgin and corticosteroids such as Coradex or Dexavet may be used in view of severity of the infection. For convalescent animals after fall of temperature to normal, supportive treatment (5% Dextrose and B. Complex inj.) is also given for quickly restoring the animals to health. Anthrax cases detected in an early febrile stage prior to development of other clinical signs of disease respond very well to treatment with Penicillin (10,000 units / kg b.wt) twice daily. Streptomycin 8-10 gm / kg b.wt in two doses i/m and oxytetracycline (5 mg/kg b.wt/day) are very effective in the affected cases. Antianthrax serum (100-250 ml i/v) can be given daily to the patients of anthrax. One may also utilize choice of ampicillin, tilmicosin and erythromycin etc., in anthrax cases.

Control

The authorities (e.g.Animal husbandry department) are to be informed for adopting regulatory steps to check the spread of the disease. The infected areas or premises should be quarantined. The dead animals are buried deep (6 ft under ground) between layers of lime (about 1 ft thick) and bleaching powder and the ditch is covered with soil in a perfect way in order to check the removal of dead bodies by

wild animals and anti-social elements.

The manure, feedings and other contaminated materials should be thoroughly burnt. Sick animals should be removed from healthy animals and put under observation to see rise of temperature etc. Cattle sheds, pen and milking equipments should be thoroughly disinfected. Scavengers like jackals etc., should not be allowed to feed on dead animals. See table 37,38 and 39 for information on different drugs or vaccines used for treating animal diseases.

Vaccination

All animals in enzootic areas should be vaccinated annually. When there is an outbreak of anthrax, all in-contact animals, should be administered anthrax vaccine.

Anthrax spore vaccine (1 ml s/c) should be administered and immunity is provided for one year. Annual vaccination in enzootic areas is highly recommended to keep the disease under control. More importantly, it is worthwhile to note that the suspected or confirmed cases of anthrax should not be opened. However, the post-mortem examination of anthrax cases is allowed in well equipped laboratory with all facilities to check the spread of the disease to the autopsists or animals living in the areas surrounding the laboratory.

Haemmorrhagic Septicaemia (Pasteurellosis)

It is an acute septicaemic bacterial disease caused by *Pasteurella multocida* type I (or B) and its Hindi synonym is Galaghotu. Pasteur (1880) discovered the organisms of cholera in fowls. This disease appears in cattle and buffaloes as a very serious febrile disease. With the onset of monsoon rains, it appears in animals exposed to chills, exhaustion and strenuous work in an acute or subacute form of an infectious disease recognized by subcutaneous oedema, and acute gastroentritis with high fever (107°F). The pasteurella organisms are present in the blood of the infected animals (i.e., organisms present in all the body fluids and tissues as

well as tonsillar and pharyngeal mucosae). Young animals (cattle and buffaloes) are usually affected. When the infection in the livestock takes place by ingestion, injuries are produced in the nasopharyngeal region. Incubation period ranges from a few hours to fortyeight hours. Animals whose resistance has been lowered by cold rains or wet conditions are usually affected by pasteurellosis. Occasionally type 4(D) and type E also cause pasteurellosis in cattle. Rabbit is the most susceptible animal to HS Affected animals my die within a few hours (i.e., even upto 6 hours) or it may last over 3 to 6 days. A less rapid course may be seen in the infected animals and pasteurellae may be found in the nasopharynx and healthy animals may remain as carriers. The pasteurellae are gram negative bipolar organisms as seen in the blood films stained by Leishman's or Gram's stain.

Signs

There is presence of profuse salivation and nasal discharge is rich in the pasteurellae. Dullness, reluctance to movement and fever (upto 107°F) are noticed. Hot and doughy swellings around the laryngeal region or along the neck or brisket upto the head region or around head is noticed in the infected animals. The tongue may be swollen or protruding and the mucous membranes are congested and haemorrhagic. Respiratory distress is seen in the affected animals. Recovery is rare in the infected buffaloes due to the severity of infection. Septicaemic effects are most pronounced in the respiratory tract, heart and gastrointestinal tract.

Pathology

The main lesions are as follows :

(1) Oedematous swelling due to subcutaneous infiltration of gelatinous fluid in the head, throat and brisket region in the majority of the cases. Clear straw coloured fluid escapes from the incised surfaces of the swellings.

(2) Scattered or generalised petechiae are noticed under the serous membranes throughout the body. Subserosal, subcutaneous and submucosal haemorrhages or

(petechiae) noticed in the dead bodies of bovines are very characteristic lesions.

(3) Pharyngeal and cervical lymph nodes are swollen and haemorrhagic. Hot and painful swellings about the throat, dewlap, brisket and perineom are noticed in the sick animals.

(4) Presence of blood tinged fluid in the thoracic, abdominal and pericardial cavities. Oedema is noticed in the lungs and lymph nodes of the affected bovines.

(5) Occurrence of pulmonary consolidation or some pneumonic changes by *Pasteurella haemolytica*. Other serotypes (Type-IV) can cause changes of bronchopneumonia. No marked change is seen in the spleen of patients.

(6) There will be presence of haemorrhagic gastritis or gastroenteritis. Only enteritis may be seen in some cases. Enlarged oedematous and haemorrhagic lymph nodes, haemorrhagic endocarditis and serofibrinous pericarditis in the fatal case of HS are the marked lesions.

(7) Little or no swellings in the atypical cases.

Diagnosis

It is based on the symptoms and the lesions like throat swellings with detection of Gram negative bipolar organisms in the saliva, blood films or tissue smears of the infected animals or recently dead cases. Blood films or tissue smears are stained by Gram's method or Leishman's technique. The pasteurellae may be in abundance (about 10 to each red cell) or may be scanty or undetectable and are usually recovered from heart blood and spleen. Swabs or sealed pipettes with blood or oedema fluid, pieces of internal organs, a long bone from animals (within a few hours of death) and even a portion of ear in a suitable container are required for cultural or isolation work of the organism in the laboratory. Pasteurella types like type 4 or type E may also cause this disease. A rapid enzyme- linked immunoassay may be carried

out to identify the specific serotypes of *Pasteurella multocida* in the infected animals.

The following table 2 will help in its differential diagnosis from some other diseases of domestic animals.

In short, pasteurellosis is diagnosed by examining thin blood films stained by Gram's and Leishman's method, Pasteurellae can be found in the red cells in the smears like short Gram negative bipolar rods. Heart blood or liver etc., is

Table-2. Causes of Diseases in Bovines and their main Features

Diseases	Causes	Distinguishing features in cattle
H.S (Pasteurellosis)	*P. multocida* Type-1	1. Presence of neck or throat swellings and high fever and profuse salivation with respiratory distress in the tropical countries like India. 2. No characteristic change in the spleen. 3. Isolation of Gram negative bipolar organisms from the infected tissues.
Anthrax	*Bacillus anthracis*	1. High fever, restlessness and abdominal pain noticed in the highly infectious and septicaemic disease. Disease may last over 2 to 4 days or animals may die within an hour. 2. Throat swelling is not common lesion in cattle. 3. No firm clotting of blood. Strings of blood clots in blood vessels are not noticed in anthrax. 4. Blood at mouth, nostrils. Spleen severely enlarged (splenomegaly) and may even rupture in cattle. 5. Presence of haemorrhagic gastroentritis. Lymphnodes may be dark like spleen pulp. 6. Tarry blood and lack of rigor mortis in the head and limbs are important features in the fatal cases.

Black quarter (BQ)	*Clostridium chauvoei*	1. Local lesions in the muscles and subcutaneous tissues of the shoulders or hindquarters as swollen and emphysematous wounds. Crepitating sounds are produced when the lesions are pressed between fingers. The swellings may not be painful and may be cold or insensitive to touch (due to necrosis). The muscles may be riddled with gas bubbles or may contain black or blackish red exudate. 2. Young cattle between the age of 6th and 24 months in a very good nutritional state are usually victims.
Rinder-pest	A filterable virus (i.e. a virus of the alimentary tract of the genus *Molillivirus* of the family *Paramyxovi vidae.*	1. Presence of fever, erosion or ulcers on the mucous membranes 2. Indigenous cattle resistant to its infection. Crossbred or pure foreign imported cattle are very susceptible to this disease (mortality may exceed 90%). 3. Rectal mucous membrane is swollen and congested and the anus is soiled with blood stained foetid faeces. Zebra markings (i.e., areas of haemorrhage and hyperaemia) are present in the rectal mucosae as very characteristic lesions of RP. 4. Greyish membranous deposits on the mucous membrane of the mouth and pharynx give rise to ulcers or erosions after their removal from the underlying tissues. The grayish white lesions on the mucous membrane of the tongue or mouth look like bran deposits on these tissues.

inoculated on to the blood agar and MacConkey's plates to recognize and isolate pasteurella colonies. A portion of ear or a long bone after tirturition can be injected into rabbits which show typical haemorrhagic tracheitis in their bodies. A quick septicaemic state is produced in infected rabbits.

Treatment/Management

The patients of pasteurellosis can be either given parenterally or orally sodium sulphadimidine or sulphamezathine (33.3% soln). The affected animals in the cases of relapse should be treated for three successive days with the same dose rate. Anyone of the routes i.e., i.v. or i.p. can be selected for drug administration. Streptomycin may also be given to sick animals. Penicillin is not so much effective in treating the cases of HS. Use of more than one kind of antibiotics is not usually advisable in treating bacterial infections. Broad spectrum antibiotics like tetracycline and chloramphenicol should be used for three days. Since HS is also a septicaemic disease with high fever, it is worthwhile to administer corticosteroids, and antipyretics with supportive treatment. In short, sulphadimidine and oxytetracycline are very effective in bovine and swine pasteurellosis respectively. Early detection of pasteurellosis in animals coupled with timely treatment minimises the risk of mortality. The dose shedule in affected animals is as follows

1. Oxytetracycline @10 mg/kg b.wt i/m
2. Procaine penicillin G @ 30,000 to 45,000/kg b.wt i/v
3. Trimethoprin-sulphadoxine @ 3 ml /45 kg b wt i/m
4. Tilmicosin @10 mg/kg b.wt

Control

Prophylactic vaccination in HS infection is advisable in

enzootic areas and the vaccination should be done before the onset of monsoon.

In an area of outbreak, healthy animals should be separated from sick ones and kept under observation. Immunity imparted by HS vaccine lasts for about 10 months.

Steps should be taken to prevent the contamination of the pastures and feeding troughs etc. It is always better to isolate the infected animals from the healthy ones. Animals should not come in contact with discharges of sick animals. The dead animals should also be burried underground with all precautions to avoid removal by wild animals. The vaccination schedule is as follows :

Table 3. Vacciens and their dosages

Vaccines	Dosages	Remarks
1. HS vaccine-monovac (inactivated culture of *P. multocida*)	Cattle, buffaloes and calves 2 ml s/c	Primary vaccination at the age of six month or above with annual revaccination.
2. HS+BQ vaccine-a biovac	2. Cattle buffaloes and calves 3 ml s/c	DO

Colibacillosis

Escherichia coli which are gram negative rod shaped organisms colonise the intestinal tract of newly born animals very shortly after birth. These harmless organisms become pathogenic under reduced state of resistance to produce the condition of colibacillosis. Young calves upto 2 month of age suffer from colibacillosis or white scour. These organisms are also non-sporing and non-capsulating and the most of the strains are motile.

Some of the types of colibacillosis are as follows :

(1) Enterotoxic colibacillosis

It is also called *E. coli* diarrhoea which is seen in young piglets, lambs and calves. These organisms adhere to the mucosa of young animals (under 3 month of age) and produce an enterotoxin in the lumen. This toxin causes excessive production of fluid from the intestinal mucosa resulting in diarrhoea. Dehydration in the animals arises from diarrhoea. The goblet cells discharge their secretion and there is no other important change in the intestinal mucosae as revealed by light microscopy.

(2) Enterotoxaemic colibacillosis

The virulent *E.coli* produce toxins in the intestinal lumen and the toxins absorbed into the body are considered to be a neurotoxin (i.e., oedema disease principle). Oedema is produced in gastric, colonic, palpebral, subcutaneous and central nervous tissues. Vascular damage, vasculitis and hyaline necrosis of arterial and arteriolar walls are produced. Some strains of *E.coli* may produce an enterotoxin as well.

(3) Septicaemic colibacillosis

The *E.coli* even invade the buccal cavity, pharynx, umbilicus and respiratory system. In this disease, lesions are produced by an endotoxin. Calves deficient in colostoral immunoglobulins are very much susceptible to this strain of *E.coli.* Colostrum fed calves, piglets, lambs and foals are very resistant to *E. coli* infection.

Bacterial arthritis, polyserositis, meningitis, opthalmitis and pyelonephritis are found in the sick calves. The lesions are necrotic with purulent or fibrinous exudation. 50 per cent of the infected calves may die and morbidity is noticed in survivors.

Treatment

Colibacillosis caused by *E.coli* is a severe disease of calves (neonates). It is quite safe to make choice of antibiotics after having report about sensitivity of *E.coli* strains affecting the animals. The most important antibiotics are chloramphenicol, neomycin, tetracycline, and streptomycin etc. There is a severe development of dehydration in sick animals. Fluid therapy can be given to dehydrated patients. In neonates, the fluid should be administered in divided doses for a period ending over 2 to 3 days. Streptomycin and chloramphenicol can be given to patients with good results.

In order to have quick effect of drugs, parenteral administration with oral preparation is advisable. Since toxaemia is seen in white scour, administration of fluid therapy is greatly helpful. Corticosteroids can also be given to animal patients for quick response.

Sanitation and feeding management have got to be improved and calves suffering from diarrhoea should be given cold (previously boiled) milk.

In order to control colibacillosis, the calf feeding pan should be thoroughly disinfected. Overcrowding of calves should be avoided. There should be proper housing and hygienic conditions. The calves must be given fresh water. The calves during their first 7 to 10 days of life should be given prophylactic medication with sulphonamides, antibiotics or deworming agents. Newly born calves must be fed colostrum within first twelve hours of life. The calves should have milk at the rate of 10% of body weight till the attainment of the age of three months. The calves may be given 2 kg of colostrum depending upon their weights. This practice imparts passive immunity to the newly born calves. For successful treatment of colibacillosis, important precautions to be kept in one's mind are alteration in the diet of the calves, fluids, and intestinal protectants antimicrobial therapy and proper management of the infected

animals. It is better to reduce milk intake until observation of clinical improvment in the patients. Glucose electrolyte mixtures are given orally to the infected calves. Parentral and oral administration of balanced electrolytes solutions is very vital step to prevent dehydration, acidosis and electrolyte imbalance. Solutions containing sodium bicarbonate are preferably given to patients. An equal mixture of isotonic saline (0.85%), isotonic sodiumbicarbonate 1.3% and isotonic dextrose 5% is given parenterally for obtaining quick improvement in the patients.

MASTITIS (Garget)

Mastitis refers to an inflammation of the mammary glands caused by several factors like bacteria, *Mycoplasma* spp. and fungi etc. This term has been derived from the Greek word mastos, meaning mammary gland. Trauma to mammary gland by wires or pointed objects can lead to transient inflammation of the mammary glands but the infection of the mammary glands is usually caused by living agents i.e., animate factors like bacteria and fungi etc.It is usually seen in the mammary glands of lactating dairy animals like cattle and bufalloes etc.

Causes

The main causes of mastitis are as under :

Streptococcus agalactiae, Streptococcus dysgalactiae, Str zooepidemicus, Str faecalis, Str pyogenes, Corynebacterium pyogenes, Mycobacterium bovis, Pasteurella multocida, Mycoplasma bovis, Nacardia asteroides, Trichosporon spp. Aspergillus fumigatus, Candida spp., Cryptococcus neoformans and Torulopsis spp. etc. *Leptospira spp. Staphylococcus, Mycoplasma spp., Eischerichia coli and Pseudomones aeruginosa* are also known to cause mastitis in animals. *Str. dysgalactiae* can cause acute mastitis in the udder and udder may be permanently non-functional in such affected animals.

Strep. pyogenes produces an acute mastitis with fever and malaise in the lactating animals. The udder can become reddish purple in staphylococcal infection and later, it becomes blackish green or cold. Gangrenous changes may set in the udder. *Staph. aureus* produces chronic mastitis. Granules can be found in pus of the abscesses in the udder (e.g., equine mammary gland showing botryomycosis). The major contagious pathogens in bovines are *Str. agalactiae, Staph. aureus* and Mycoplasma spp.

Gangrenous type of mastitis can be caused by *Corynebacterium pyogenes* in sheep. In black garget, the disease runs an acute rapid course and is frequently fatal in the mastitis cases. The mammary gland in the sheep may be hot, tense and painful and death can occur in 24 to 40 hours.

PATHOLOGY

Gross Changes

1. The cardinal signs of inflammation i.e., heat, redness, swelling, pain and loss of function (i.e., reduced milk secretion) etc. can be seen in the inflamed udder. Clots, flakes, blood, discolouration of milk and an excess of leucocytes are important findings in the milk of mastitis cases.
2. Cessation or reduction in the milk secretion. Exudative fluid of abnormal colour or consistency can be milked from the teat in place of usual milk. Warmth, diffuse swelling and gangrene may be noticed in the mammary glands in acute cases.
3. Firmness or shrinking in the quarters. When two or more quarters are involved, the udder atrophy (marked by a small quarter or small teat) is a more marked change. The reason of the firmness in the udder is diffuse proliferation of the fibrous tissue (fibrosis).
4. Old inspissated abscesses or lumps of firm tissue are evident in the affected quarter and these can be palpated.

5. A dark pink or light red colour can be marked on the cut surface of the acutely inflamed udder. The cut surface of gland is moistened and lobulations are less distinct on the cut surfaces. Even blood can be noticed on the incised surfaces of the gland. Yellowish milk or pus can be seen on the cut surfaces of the udder. In chronically inflamed udder, abscesses or somewhat shiny white fibrous tissue can be detected on its cut surfaces.

Microscopic appearances

Acute exudative inflammatory changes can be noticed in the udder except the mucous inflammation (because of udder being a non-mucus forming organ). Serous, purulent and fibrinous changes occur in the lactating dairy animals like cattle and buffaloes in the mammary glands. Haemorrhagic inflammatory changes can be found rarely in the udder. Fibrous proliferative changes are more marked in chronic mastitis. (Fig. 1; p. 199) The exudative changes are marked in the alveolar lumina and the intercellular spaces. Dead or dying leucocytes (pus cells) can be found in the alveoli of the udder. Scanty albuminous precipitate can be seen in the alveoli indicating distension of the alveoli with fluid. In such alveoli, the epithelial lining shows compressed or vacuolated cells. Inflammatory cells like leucocytes, neutrophiles, monocytes and lymphocytes can be noticed in the adjoining alveolar stroma. The alveolar capillaries can be found distended with blood. Clots of fibrin and leucocytes are seen in the smaller ducts. Occlusion to the drainage of the exudate leads to growth and spread of the infectious organisms. In *Streptococcus agalactiae* infection of the udder, there is hyperplastic thickening and cornification of the epithelial lining of the lactiferous ducts and sinuses with infiltration of the inflammatory cells beneath the hyperplastic epithelial cells. The infection spreads to supporting stroma. In virulent infections, the alveolar Epithelium is destroyed and the alveolar walls may collapse against each other with

replacement by fibrous tissue growth at later stages. The affected parts of the udder become smaller than normal due to fibrosis and destruction of the mammary tissues and such quarters have shrunken appearance.

Diagnosis

It is based on the symptoms, lesions and isolation of the aetiological factors from the milk or udder lesions. The secretion from the udder will be yellowish or watery in nature. In severe chronic cases, the milk will be stringy, ropy or curdy. The coagulated fibrin gives rise to stringy milk. Coagulated casein is seen in the milk. The milk may be acidic in reaction and strip up test shows solidified material.

The following steps can be taken to diagnose mastitis :

1. The leucocyte count and direct bacterial count: For this, a measured quantity of milk is spread on a slide and then it is stained and all kinds of body cells and bacteria are counted.
2. Bromothymol blue test : It is carried out to detect increased alkalinity in the milk.
3. The white side test: A drop of 5% NaOH is added to 4 to 5 drops of milk on a glass slide. It is mixed with a palatinum loop. Discrete clumps or flecks can be found in milk after half a minute.
4. The coagulase test : A colony of staphylococci (i.e., from culture of milk sample) is mixed with normal saline. A drop of 1/10 dilution of the rabbit plasma is added to it and clumping of bacteria is seen after a few seconds in staphylococcal mastitis.

Bacteriological Examination

A loopful of well shaken milk sample is inoculated on to an Edwards plate and an another loopful on to a blood agar plate and the plates are incubated at 37^0C. The bacterial sediments of the centrifused milk are used to make smears

which are stained by Gram's method and acid fast method to detect the acid and non-acid test organisms respectively.

Treatment/Management

Treatment of mastitis is carried out either locally or in general ways. Steps are taken to protect the animals from toxaemic condition. The pus, clots, debris and fibrinous materials are removed from udder to maintain the milk flow and restore the milk yield.

The systemic general treatment includes parentral administration of drugs to check septicaemia or bacteraemia. Antibiotics like penicillin, streptomycin and ampicillin can be given. Oxytetracycline can also be given by i/m or i/v route. Use of antihistaminics by i/m route (e.g., avil) is also advisable. Cortico-steroids are given parenterally in cases of toxaemia. Use of electrolytes solutions is also recommended in cases of toxaemic condition.

The local treatment of mastitis includes udder infusion. For this, disposable intrammary infusion tubes containing antibiotics are utilized. Such intramammary tubes contain ampicillin and strepto-penicillin with cortisone etc. If the bacteria are susceptible to such antibiotics, recovery takes place. Administration of antibiotics maintains high level in plasma and milk and it is always advisable to adopt local treatment alwongwith parenteral administration of antibiotics. It is usually helpful to have application of similar kind of antibiotics in the udder and in the body by parenteral route.

For controlling mastitis in herd, it is proper to adopt hygienic measures and avoid contamination or infection of udder with yeast or fungi. Utensils must be thoroughly sterilized before their coming in contact with udder of healthy animals. The use of cortisone (steroid) in acute mastitis reduces inflammation. The chronic cases of mastitis can be treated satisfactorily when the cows are dry. When

the cows are under dry state, the infusion of the udder is done as a preventive measure at the end of the last milking and also at the beginning of the first milking. The presence of antibiotic residues in milk causes sensitivity in human beings. It is better not to use the milk having residues of the antibiotics.

There should be lapse of atleast ninty-six hours before consumption of milk from infected quarters. The quarters of lactating animal can be dried by using infusion of the following preparation like 30 to 60 ml (3% $AgNO_3$ soln or 20 ml of 5% copper sulphate soln) or 100 to 300 ml of 1:500 acriflavin solution. In case of visibility of reactions in other tissues, the quarters should be milked out. The quarter of the animals can be stripped after 10 -14 days of infusion. Such two infusions may be required for rendering the udder dry. If there is a suspicion of resistant strains of bacteria, treatment of mastitis can be done only by obtaining report on the antibiotics sensitivity test for organisms present in the milk.

Mastitis due to fungi and yeast do not respond satisfactorily to antibiotics and drugs nystatin may be used.

In order to control mastitis, it is advisable to remove the sources of infection, reduce the susceptibility of mammary glands and also prevent the spread of infection between lactating quarters.

The milk from affected quarter is examined bacteriologically and such infected cow should be separated from healthy ones. As a general procedure, careful milking, udder washing, disinfection of teats and cup etc., should be followed strictly. The quarters of the udder, if fibrosed, can not be restored to normalcy and any surgical interference in such udder is not helpful. Cows and buffaloes etc., can go on giving milk inspite of one or two functioning quarters for whole of their life spans. Subclinical mastitis due to contagious pathogens in cows can be treated by intramammary injection

of long acting anti-microbials at drying off.

Strangles

It is noticed as an acute contagious disease mainly in young horses which is caused by *Streptococcus equi* (sub species equi). There is a catarrhal inflammation of the upper respiratory tract and formation of abscesses in the submaxillary and pharynagal lymph nodes. The causative agent of strangles is *Streptococcus equi* which is gram positive. The recovered horses exist as carriers which can spread infection to other young horses. Organisms like *Str. equi* can enter the blood stream produces fever and set up metastatic lesions like abscesses. Horses over 5 years of age are resistant to *Str. equi* infection. Contaminated utensil, bedding, food and drinking water etc., spread the infection to healthy young horses. Contact with diseased horses also spreads infection to healthy ones. Infection can enter the body by ingestion, inhalation and wound infection with discharges etc., of the animal patients. The incubation period varies form 3 to 8 days in the horses. The important clinical signs are fever, anorexia, depression, submandibular as pharyngeal lymphadenopathy marked by abscessation, rupture of the abscesses and purulent nasal discharge.

Pathology

The main lesions are:

(1) Catarrhal inflammation of the nasal and pharyngeal mucous membrane. This results in formation of a thick yellow discharge from the nostrils.

(2) Submaxillary, pharyngeal, parotid and cervical lymph nodes are swollen and can undergo suppuration which may burst into the pharynx.

(3) Rise of temperature (pyrexia).

(4) Catarrhal bronchitis, pneumonia, pleurisy and pericarditis.

(5) Pyaemia and abscesses in the different internal organs. Septic peritonitis may arise from bursting of the abscesses in the mesenteric lymph nodes. Abscesses may occur in the lymphnodes like mediastinal, axillary, inguinal or popliteal lymph nodes etc. Abscesses of the retropharyngeal lymph nodes drain into the guttural pouches. Empyema in such structures can be noticed. In short, caseous lymphadenopathy with rhinitis and pharyngitis, pneumonia and metastatic infection are main changes in equine strangles.

Diagnosis

It is based on the symptoms, lesions and isolation of *Str. equi* from the nasal discharge and abscesses.

Treatment/Management

For treating stangles in horses, antibiotics like dicrysticin and sulpha-mezathine sodium can be used. Tetracycline can also be effective.

In order to control strangles in equines, it is very important to isolate the affected horses and keep them separately. The horses should be given warm shelter, good food, etc. The nostrils and muzzles should be kept clean. They should be given steam inhalation. The house, bedding, broom, brush and blankets should be disinfected. An autogenous vaccine consisting of culture of *Streptococcus equi* can also be given to control the cases of strangles. The vaccine can be given in two or three doses at an interval of 10 to 15 days. The dose of vaccine in the beginning is 1 cc by s/c route . Penicillin (Procaine penicillin G) 22,000 iu/kg b.wt. twice daily is injected intramuscularly for five days.

Navel-Ill

This disease is also called pyosepticaemia neonatorum which means a septicaemic state in the affected young animals (e.g., calves and foals etc.). Lesions in the joints of

the foals are not constant accompanying facts. Death occurs in foals within 3 or 4 days of birth due to acute septicaemic changes in bodies. In health, amminiotic membrane of the umbilical cord is torn at birth with closure of the umbilical vein and the umbilicus dries up within a week after birth in calves.

Causes

The main causes are as under :

Table 4. Animals and causative factors

Animals	Organisms
Equines (foals)	*Shigella equirulis (Actinobacillus equuli)* and other organisms like *E.coli,S. (abortus equi* and *Corynebacteria* etc.)
Bovines (calves)	*E. Coli, Br. abortus, C. pyoguenes,* and *Pasteurella* etc. can be isolated alone or in mixed infections.
Ovines (lambs)	*Streptococci and Staphylococci.*
Swines (piglets)	*E.coli,* pyogenic coceci and *Haemophilus influenzae* etc.

Signs

The affected animals (foals) are weak, unable to stand and show swollen, hot and painful joints. Fever and depression are seen in the foals which die within a few days of birth. When the infection is of prenatal origin, the foals are found dead at birth. These foals get infected from the infected udder through the mouth or infected umbilicus. Even contaminated vagina can spread the disease to the young animals. The carrier mares of infection of navel-ill are known to exist and pose problems before horse rearing.

Pathology

At autopsy, joints are found to be enlarged and contain an excess of synovial fluid (with sanguinous or purulent material) in the foals. Grey foci are found in the renal cortex

and haemorrhages are seen in the renal medulla. The grey foci in the cortex are areas of abscesses arising from lodgement of the bacteria in the glomeruli. The kidneys are swollen and their capsules can be easily detached. In pigs, the navel-ill occurs as a septicaemic disease. Other lesions in the foals include enlargement and oedema of the lymph nodes, petechiae or congestion in the serous membranes and degenerative changes in the skeletal and cardiac muscles. Inflammation of the umbilical vein (omphalophlebitis) and purulent material can be found in the navel. The cartilages or bones in the joints can show necrotic changes. The mucosae of the intestine may be thickened and congested and oedematous changes are found in the lungs. There is no blood in the normal umbilical vein of calves and foals etc., but in navel-ill, imperfectly coagulated blood can be found in the umbilical vein.

Diagnosis

It is based on the symptoms, lesions and isolation of the organisms like *Actinobacillus equuli, Streptococci,* and *E. coli* etc.

Treatment/Management

Navel-ill can be treated by injection of penicillin for the period of three days (10,000 iu/lb body wt.). Antibiotics can be given for longer period in case of suppurration in navel. Cortisone can be used parenterally and can also be applied locally. Calves should be fed Vitamins (B-complex) orally. Infected animals should be prevented to come in contact with pregnant or newly born animals. It is very much advisable to disinfect the navel of calf at birth and for this, betadine lotion can be used. Exploratory laprotomy and surgical removal of abscesses are also advisable in some cases of navel-ill.

GLANDERS

It is a contagious bacterial disease primarily affecting the solipeds like horses and now-a-days only sporadic incidence of this disease is noticed. Man and members of the cat family are also be affected. The obligate causative organism is *Pseudomonas mallei* which produces an acute or more usually a chronic disease in horses, mules and asses. This disease ends fatally and fibro-caseous nodules are formed in the upper respiratory tract, lungs and skin etc. Pigs and cattle are immune to this disease. Ingestion of food or water contaminated with discharges of excretion of the infected animals produces it in healthy animals. Skin infection through contamination of open wounds in the body may occur in some cases. Infection by inhalation is unusual. Incubation period ranges from 2 weeks to 2 or 3 months or longer. Guinea pig is very susceptible to the glanders organisms and nodules develop in their lymph nodes, spleen, lungs and liver etc. The male guinea pig which is infected with the organisms given intraperitoneally, develops pus formation in the tunica vaginalis and orchitis is seen in 2 to 3 days. This is called Strauss test for glanders. Organisms are found in the guinea pig's lesions and these can be isolated in pure form from such lesions. The organisms are rodshaped, Gram negative, non-sporulating and non-motile and are isolated on glycerine-agar and potato etc.

Glanders can be divided into the following types :

(i) Nasal glanders

(ii) Pulmonary glanders

(iii) Cutaneous glanders

It may develop in either acute, sub acute or chronic form in animals. The characteristic lesions in the glandered animals are pneumonia, nodules or ulcers in the respiratory tract (e.g., larynx and trachea etc.), skin and subcutaneous tissues along lymphatics. Vari-sized nodules in the subcutaneous tissues

of hind limbs, neck, face and liver etc. are called farcy buds.

(i) Nasal Glanders

Submucosal nodules are found on the nasal septum and these nodules break down to give rise to shallow, crater like ulcers or erosions which liberate a thick, sticky or oily yellowish brown discharge. Later, these ulcers turn into stellate shaped scars. Swelling is noticed in regional lymph nodes. The ulcers on the nasal septum may have characteristic punched out appearance. There is an irregular contour of these lesions with raised or eroded borders. Ulcers can penetrate into cartilage to cause perforations. The discharge from the ulcers may be often blood tinged in nature. Usually isolated ulcers are found in the larynx and trachea. Ulcers can also be found on the turbinate bones and in the guttural pouches and Eustachean tubes.

(ii) Pulmonary Glanders

Greyish, firm round encapsulated nodules like tubercles of *Mycobacterium tuberculosis* infection are found in the lungs. Diffuse pneumonic changes may be present in such lungs. The nodules can be found subpleurally like small shots in the lung parenchyma. The nodules are red at the earliest stages but later show yellowish centres projecting above the surrounding red zone of hyperaemia. Later, yellowish grey nodules are formed in the lungs from such lesions. Capsules are found in the old lesions. Pleurisy is found with a covering of fibrinous material. Bronchial and thoracic lymph nodes may show degenerative changes or abscesses. Microscopically, the lesions in the lungs show alveoli filled with leucocytes and the alveolar walls can be seen disappearing or disintegrating at places. The degenerative necrotic changes occur in the affected tissues with appearance of nuclear chromatin as fragmented or scattered particles (phenomenon of karyorrhexis). At peripheral areas of such changes, red cells and fibrinous material are found

and the surrounding lung tissue is hyperaemic. Endothelioid cells are seen around these central degenerated areas in the older nodular lesions. Necrotic centre with surrounding endothelioid cells can be found marginal to fibrous capsule. Giant cells are found in such areas and calcification may be present occasionally. Highly virulent strains of *P.mallei* produce acute lesions marked by necrosis and disintegration of the cellular elements where as endothelioid cells, fibrous tissue and giant cells are noticed in the less acute lesions i.e., subactue or chronic ones.

Conversion of lesions of chronic glanders into acute ones is seen in cases of acute glanders arising from chronic glanders. Catarrhal or croupous pneumonia and haemorrhagic infarcts are found in the pulmonary glanders in addition to nodular changes. Such nodular lesions are also found in the organs like liver and spleen. These nodules may be firm and grey with central softening in these organs. Submaxillary lymph nodes are most commonly infected and show enlargement, oedema and one or two yellowish-grey centres. Later, these become hard, fibrous and fixed to the jaw of horses.

(iii) Cutaneous glanders

It is also called farcy. Nodules appear along the lymphatic channels, particularly often on limbs and break through the skin discharging a thick, sticky and yellowish grey pus. Such ulcers heal very slowly. Farcy is characterized by chronic lymphangitis and lymphadenitis affecting one or more limbs(especially the hind limbs). Small round nodules (farcy buds) appear in chains in subcutaneous tissues along the course of lymph channels. These buds turn into abscesses which later give rise to ulcers. Such lesions can be found on face, neck and body of the infected horses.

Diagnosis

It is based on the following :

(i) Symptoms, lesions and detection and isolation of *P. mallei* from glanders lesions, nasal discharge, ulceration, chronic cough, submaxillary lymphadenitis. Cutaneous lesions help diagnosis of glanders in the affected animals.

(ii) Cultures from the lesions of glanders and performance of Strauss test in the guinea pigs. These tests help its diagnosis.

Mallein Test

It is done by subcutaneous, intradermo-palpebral, ophthalmic and cutaneous methods. Intradermo-palpebral mallein test is the most reliable one. 0.1 ml of concentrated mallein is injected into the dermis of the lower eyelid about a one- fourth of inch below the lashes. A voluminous oedema develops in the positive cases. The oedema is very hot and painful one.

Direct smears are made from a farcy bud and stained by Gram's method after fixation over heat. Films are examined for the presence of slender Gram negative rods. For cultural examination, pus is inoculated on to blood agar plates and the plates are examined after 3 - 4 days for the growth of organisms. Animal inoculation is done to diagnose glanders. Pus or contaminated material or culture is inoculated into two male guinea pigs subcutanceously and examination of guinea pigs is done for the development of orchitis daily after 3 days. Complement fixation test confirms its diagnosis.

Treatment/Management

The treatment of glanders is very costly one and in the case of confirmation of glanders cases, the authorities should be informed immediately, in fact, there is no effective

treatment. Sodium sulphadiadine has got good effect in treating experimental glanders, in hamsters. Sulfonamides are effective in treating glanders in man and laboratory animals. Penicillin and streptomycin are not effective. Horses can be treated for atleast 20 days with sulpha drugs. The treatment of glanders is not advisable in view of threat to public health. In order to control glanders in horses, the best procedure is to test horses for glanders and destroy the positive cases. The mallein test and complement fixation are done in horses to diagnose glanders.

Leptospirosis

A bacterial disease of man and animals caused by organisms of the genus *Leptospira icterohaemorrhagiae. L. canicola* are the organisms affecting dogs. *L. pomona and L. grippotyphosa* (bovis) infect cattle and swine etc. Several other serotypes of *Leptospira spp.* have also been identified. The organisms may be single, helical, flexuous or hooked. They may have also curved ends.

The important types of leptospirosis are canine leptospirosis, bovine leptospirosis and procine leptospirosis.

Canine Leptospirosis

Leptospira interrogans and serotypes as *L. canicola and L. icterohaemorrhagiae* infect dogs. The important signs in these animals are fever, vomiting, icterus, dehydration, bloody diarrhoea, increase in E.S.R., albuminuria and debility etc. The dogs develop nephritis with uraemia. Leptospira can be found in the kidneys of dogs. The dogs show dehydration and uraemic breath is recognized in them by smell. Ureamic smell can be marked in the stomach contents or mucosae of the mouth at autopsy of fatal cases.

Pathology

Leptospirosis occurs in acute and subacute phases in dogs.

The main lesions in the acute phases are :

(i) Severe dehydration, icterus, petechiae on the pleura, peritoneum and nasal and oral mucosae.

(ii) There is shrinkage of the liver cells alongwith dissociation from each other (disorganization of the hepatic cells). The liver cells in the hepatic cords break into individual ones and disruption of the cells in the hepatic cords occurs (individualisation). The liver cells are granular with eosinophilic cytoplasm and hyperchromatic nuclei. Regeneration is recognized owning to presence of binucleate cells, hyperchromatic nuclei and mitotic figures. There are areas of focal necrosis and plugging of the bile canaliculi due to bile. Portal vessels are congested and haemosiderin is noticed in the Kupffer cells. Leptospirae can be found within sinusoids of liver cells by silver impregnation technique. There is almost no change in the glomeruli of the kidneys but the epithelial cells of the convoluted tubules are swollen, granular and highly eosinophilic. These epithelial cells may be vacuolated or desquamated into the tubular lumens. The basement membrane of the tubules can be seen in some cases and the tubular lumina can be found packed with eosinophilic epithelial cytoplasmic debris which may show nuclei with occasional presence of some red cells. The tubular epithelial cells may show regenerative changes which are evidenced by the presence of mitoses, hyperchromatic nuclei or multinucleated giant cells (due to fusion or coalescence of the cells). Leptospirae can be found in the affected tubules by following silver impregnation methods for staining the tissues. The tubules can be seen surrounded by red cells, plasma cells, lymphocytes and occasional some red cells. Such cells also diffusely infiltrate into interstitium of the tubules.

Spleen and lymph nodes are enlarged and may show oedema and haemorrhages. Microscopically, paucity of mature lymphocytes with an apparent increase in vesicular

cells can be seen in the stained sections of these organs. Red cells can be found free or inside the macrophages in the medullary sinuses. There are areas of diffuse haemorrhages in the fundic portion of the gastric mucosa. Necrosis, neutrophilic infiltration and desquamation of the gastric mucosa can be noticed. Haemorrhages in the submucosa and less frequently in the muscularis are noticed. Small petechiae in the intestinal serosae are seen. Haemorrhages and oedema are also noticed in the heart, submucosa and muscularis of the urinary bladder, and organs like adrenal glands, pancreas and gall bladder. Tiny spherical haemorrhages are seen in the pleural lesions in subacute phase.

Animals recovering from acute phase die later from development of uraemia. The animals in the subacute phase of the disease show dehydration, emaciation and uraemic odour can be marked in the oral mucosae. Icterus and haemorrhages can be found. The kidneys are grossly enlarged with usually smooth surface and capsule may be tense, white or greyish in colour with sometimes haemorrhages in the renal parenchyma. The kidneys are somewhat firm and are cut into slices with some resistance and the cut surfaces of the kidneys are moist and turgid and somewhat bulging. Petechiae amy be present on the cut renal surfaces. Greyish masses of firm turgid tissue can be seen at the corticomedullary junction of the kidneys and these lesions may more or less obliterate the cortex. Microscopically, convoluted tubules may show degenerative changes or may be replaced by large dense masses of cells such as lymphocytes, plasma cells, macrophages, occasional neutrophiles and some red cells. Glomeruli are often spared or involved only secondarily. Leptospirae can be demonstrated in the luminae of tubules or epithelial cells lining the tubules by silver impregnation techniques. Lesions in the state of the uremia noticed in the affected dogs include gastric haemorrhages, deposits (i.e., calcium in the gastric mucosa) and calcareous deposits in the wall of the aorta and larger arteries.

Bovine Leptospirosis

Leptospiral organisms have been noted in bovines showing signs of mastitis, fever, icterus, emaciation, haemoglobinuria, abortion (usually late abortions i.e. abortion after 6 months), occasional anaemia, transient leucopenia and death. The principal serotype of leptospira in cattle is *L. interrogans* serovar pomana. 30 % of the pregnant infected cows abort dead foetuses with yellowish brown discolouration of the placenta. Gelatinous oedema may be seen between allantois.

Pathology

Lesions

Acute septicaemic or chronic nephritic form of leptospirosis has been noticed in cattle. The lesions are similar to those of dogs. Icterus and swollen and yellowish liver with petechiae are found in acute septicaemic form. Haemolytic anaemia is seen in the diseased cattle. Microscopically, portal lymphocytic infiltration with splenic haemosiderosis and centrilobular necrosis are noticed in the livers of the affected cattle. Hepatic cells dissociation, cholangitis, congestion and haemosiderosis of the spleen are marked in cattle affected with the *Grippotyphosa* serotype. Swelling and disorganization of the convoluted tubular epithelium are associated with the presence of bile and haemoglobin in the tubular lumina. Greyish white focal lesions are seen in the kidneys and these foci are discrete and scattered through out the cortex. Microscopically, the epithelial cells of the tubules are granular and swollen with vacuolated cytoplasm. Fragmentation of the cytoplasm and detatchment of the cells can be seen in the tubules. Leucocytes like lymphocytes and plasma cells can be found around the tubules. Syncytial giant cells of the Langhan's type can also be found. Leptospirae can be demonstrated in the epithelial cells of tubular lumina by silver impregnation method.

Porcine Leptospirosis

Several serotypes of leptospira affect the pigs. Serotype *Pomona* is a common type producing disease in the pigs. These organisms can produce subclinical infection or acute lesions or changes like hepatitis and icterus. Subacute chronic nephritis, abortion, still birth or birth of the weak piglets can occur in the leptospiral infections. Tubular degeneration and intense focal lymphocytic infiltration can be found in kidneys. Tubular epithelium and lamina also show leptospirae. The organisms can be found in the nodules of the lymphocytic infiltrations. Leptospirae are observed in the urine for some time. Still born pigs and aborted foetuses are found in the last third of gestation. The foetuses may be macerated. Focal necrosis is found in the livers of still born or dead foetuses.

Diagnosis

Fresh material is used for diagnosis. Blood examination is made in early febrile or septicaemic stage of the disease. Dark ground microscopy is adopted to identify the organisms. The main methods of diagnosis are as follows :

1. **Direct Examination** - Deposits of blood collected at height of the fever or deposits of fresh urine is examined by dark ground microscopy to detect leptospirae.
2. **Fresh Kidney or Liver Tissue is examined** by (i) Dark ground microscopy and (ii) by Levaditi's methods. Smears of liver of kidneys can be fixed and stained by Levaditi method to identify the leptospirae.
3. **Biological Examination** White guinea pigs are inoculated intraperitoneally with preparations from urine, kidney or liver tissue. They are observed for 3 weeks. These animals usually die within 8 to 12 days. Jaundice and haemorrhage in the lungs, serous membranes and muscles are found in the dead guinea pigs. Direct examination of liver and kidneys reveals leptospirae.
4. **Cultural Examination** For this, fresh kidney or liver

material and blood at the height of fever is used for culture on special media like Noguchis or Fletcher's media. *Leptospira* spp are isolated from samples like blood, urine cervico vaginal mucus, body fluids and lesions in the affected tissue.

5. **Elisa, DNA Probes and Microscopic Agglutination Tests**: These tests are performed to confirm the diagnosis of leptospirosis in the infected animals.

Treatment/Management

Leptospirosis in animals like cattle and dogs etc. is caused by several species of leptospirae. It responds well to treatment with antibiotics such as Streptomycin and Tetracycline etc. Dihydrostreptomycin in single dose of 25 mg/kg body can be used to eliminate the infection from shaders. The treatment with tetracycline can be carried out for 7 to 10 days. For controlling leptospirosis, vaccination can be done in large animals.

In order to treat haemolytic anaemia in acute leptospirosis, blood transfussion @5-10 lt/450kg b.wt and @50-100ml/5kg b.wt gives good results in the infected cattle and dogs respectively. A combination therapy of dihydrorestptimycin @ 10-11 mg/kg b.wt twice daily with a single daily dose of penecillin G @ 10,000/kg b.wt or tetracycline @ 20mg/kg bwt by oral route twice daily is very effective in dogs suffering from acute leptospirosis.

Calves from immune mothers get passive immunity through colostrum which lasts 2 to 6 months. The immunity produced by vaccination may last six months and the susceptible animals are vaccinated to control this disease. In short, antimicrobials are used to eliminate infection in the carriers. Vaccines containing serovars are beneficial for control of leptospirosis in a particular zone.

Nocardiosis

It is a bacterial infection caused by *Nocardia asteroides* in man and animals. Bovine farcy is produced by it. In cattle, bovine mastitis is also caused by Nocardia spp . Infection of the bovine foetus and placenta causes abortions in bovines.

Pathology

Lungs, pleura and skin are affected in the dogs and the organisms are found to localize at places like peritoneal and pleural cavities, brain and different visceral organs. Tangled indistinct colonies of the organisms surrounded by necrotic cellular debris, purulent exudate and granulation tissue are seen in the lesions caused by *Nocardia* spp. The organisms are Gram positive and have also acid fast staining properties. In bovine farcy, there is a chronic suppurative granulomatous inflammation in skin lymphatics draining lymph nodes in the infected limbs. Infection can metastasise in the organs like lungs, liver, spleen and internal lymph nodes. Extensive granulomatous lesions are noticed in nocardial mastitis in cattle.

Diagnosis

Symptoms, lesions and isolation of the organisms from the lesions establish the infection in animals. The nocardial organisms are also detectable in the stained sections of the lesions in the tissues like mammary glands.

Treatment/Management

Sodium iodide can be used parenterally. The affected limbs of cattle may be disinfected with proper drugs to control this disease.

Tuberculosis

It is a bacterial disease of man and animals caused by *Mycobacterium tuberculosis.* Koch (1882) demonstrated the

casual organisms of tuberculosis. Different types of tubercle bacteria produce lesions in man, animals and birds. Human and bovine types of tubercle bacteria infect human beings wherease fowls are affected by *M. avium*. *Mycobacterium bovis* produces a progressive and fatal infection in cattle, guinea pigs and rabbits of all ages but in cattle, a progressive type of infection is not produced by human types. Tuberculosis is the most frequent disease in cattle but is also noticed in buffaloes, sheep, goat, pig, dog, cat and horse etc. The organisms are acid fast and stained by Ziehl-Neelsen method. *Mycobacterium tuberculosis* is also Gram positive.

Contaminated excretions and secretions from tuberculous animals infect man and animals either by ingestion or by inhalation. Urine, faeces and milk from tuberculous animals contaminate water or food which are eaten by healthy animals and tuberculosis subsequently develops in them. Milk in tuberculous mastitis is a dangerous source of infection to man and calves or young ones fed on such milk. The tubercle bacilli penetrate through the pharyngeal mucosae and produce tuberculous lesions in the pharyngeal and submaxillary lymph nodes. The organisms may infect the intestinal mucosae and reach mesenteric lymph nodes to produce tuberculous lesions. No macroscopic lesions can be seen at the site of penetration of tubercle bacilli in the mucosae, but the lesions of tuberculosis develop in the neighbouring lymph nodes due to spread through lymph channels (called metastasis). Cattle get infected through inhalation of infected material (droplet infection) with tubercle bacilli. These droplets containing TB organisms are present as a fine spray expelled by tuberculous patients during coughing and on being inhaled, these droplet nuclei teaming with tubercle bacilli give rise to pulmonary tuberculosis in the infected animals. Dried dusts rich in tubercle bacilli produce TB on being inspired by healthy animals. The organisms enter the alveoli and finally reach the neighbouring lymph nodes draining lymph from such

organs and tuberculous nodules are produced in the affected organs. Congenital tuberculosis is found in calves produced by cows infected with tuberculous metritis. Bulls suffering from TB of penis, testicle or epididymis may infect the healthy cows during service. Infected cows may also spread TB to bulls. Cutaneous infection of TB rarely occurs. Generalisation of tuberculosis occurs due to spread of tubercle bacilli by blood to different sites or organs in the body. TB lesions can develop in the different organs like lungs, liver, kidneys and spleen to produce what is termed as generalized tuberculosis. When the lungs are invaded by innumerable tubercle bacilli released in the blood, several small tuberculous lesions develop in the lungs to produce what is called miliary tuberculosis. Progressive emaciation, capricious appetite and fluctuating temperature are some important signs of TB in animals.

Pathology

A tubercle is a characteristic lesion of tuberculosis in the body of an organism. It starts as a cluster of neutrophiles surrounding the tubercle bacilli. In a few hours, they are replaced by more powerful phagocytic cells like macrophages or cells like epithelioid, endothelioid and reticulo-endothelial cells. The encircling epithelioid cells engulf the bacteria. Such cells die and undergo caseous necrosis and more epithelioid granulation tissue is formed. The epithelioid cells have abundant foamy, pale acidophitic cytoplasm and their nuclei are eccentrically placed. When epithelioid cells fuse, Langhan's types of giant cells are formed. These giant cells measure about 50 μ in diameter and their nuclei are arranged like a wreath or crescent at the periphery of pale acidophilic cytoplasm. (Fig.) Other inflammatory cells like lymphocytes also appear at the scene of infections called infectious granulomata and the whole lesion, later, gets encapsulated by connective tissue. Such tubercles show calcification in cattle and buffaloes etc., but no calcification is seen in birds

like chickens. A tubercle may exist as a mass of fibrous or hyaline scar tissue in the case of destruction of the invading tubercle bacilli. In meningeal tuberculosis, there is a scanty or fibrinous or fibrinopurulent material on the surface of the piameter. Firm hard white and grey or yellow nodules are formed in the organs like liver and lungs. The cut surfaces of such lesions are yellowish, caseous, dry or solid in consistency. Chronic proliferative lesions with rare calcification or caseation are noticed in equine tuberculosis. In dogs, calcification and caseation are not prominent lesions but there is diffuse infiltration of epithelioid cells. Tubercle bacilli produce local cell proliferations to cause formation of microscopic tubercles, which on fusion with each other, give rise to macroscopic tubercles (1 mm to 2 cm in diameter) in an organ. Microscopic tubercles are noticed in miliary tuberculosis. These nodules grow to the size of millet seeds and are translucent or grey in colour and are almost of the same sizes. In short, a T B nodule consists mass of endothelioid or epithelioid cells and some giant cells of Langhan's types surrounded by several lymphoid cells. (Fig. 2; p. 199) This nodular structure is supported by a fibrous recticulum or fibrous tissue capsule. The enothelioid cells are derived from reticuloendothelial cells and have a large pale staining vesicular nuclei and irregular or ill defined bodies. The Langhan's type of giant cells are produced due to division of the nuclei of the endothelioid cells. The nuclei in these giant cells have got a peripheral arrangement and the cytoplasm is granular or hyaline. The lymphocytes are migrated cells from the blood vessels invading the tubercles. T B nodules are non-vascular. Later, necrotic changes with karyorrhexis of nuclei are noticed at the centre of such nodules. The dead tissue undergoes caseation i.e., becomes cheese like in consistency. Lime salts are precipitated into the dead caseated areas (a kind of dystrophic calcification) to form a substance of a gritty calcified nature. Such changes at the centre of the tubercle are accompanied by proliferation of the fibroblasts

at the periphery of the lesions in order to encapsulate the TB lesions. Encapsulation tends to isolate the disease processes and limits their spread into healthy tissues or other organs.

Diagnosis

It is based on the following :

(i) Symptoms, lesions and detection and isolation of *M. tuberculosis* from tuberculous lesions. TB bacilli are found in the smears made from the coughed up mucus, milk sediment, vaginal discharge, urine and faeces etc. Ziehl-Neelsen's technique is followed to stain the organisms in these smears. The tubercle bacilli are acid fast rods which stain red by this technique. The non-acid fast organisms stain blue. The affected animals show mild cough and also some rise of temperature (say, 103°F).

(ii) Subcutaneous inoculation of the suspected material is made inside the thigh of the guinea pigs which are usually killed in 5 to 6 weeks for postmortem examination. TB bacilli are found in the smears of the lesions in the body of guinea pigs. Cultures are also done to isolate TB bacteria from such lesions.

(iii) Tuberculin Test : Intradermal tuberculin test is done to detect TB in the affected herds of cattle and buffaloes. Tuberculin or purified protein derivative (P.P.D.) is injected intradermally into the skin of neck. A hot, diffuse and painful swelling develops at the site of injection of tuberculin in positive cases.

In short, tuberculosis is diagnosed on the basis of examination of stained direct smears from tissues (for example, lymph nodes and lungs showing nodules) by Ziehl-Neelson's method, cultural examination (inoculation of suitable preparation of the deposit, say 1 ml, into left leg of two guinea pigs). Gross lesions of TB are noticed in the related or regional lymph nodes i.e., lymph nodes draining the inoculation site. Milk sediment from suspected tuberculous

udder can be used as an inoculum to be injected into the left legs of two guinea pigs by the intramuscular route.

Treatment/Management

Tuberculosis in human beings is treated effectively by using drugs like Isoniazid and streptomycin etc. But in cases of animals, the treatment of tuberculosis cannot be safely advocated in view of public health and economical factors. In short, it is risky for human beings to remain in contact with tuberculous animals. Cattle with lesions in the lungs kidneys and other organs do not show quick response to treatment and usually die.

Tuberculosis can be controlled in a herd or farms maintained in public or private sector by following rigid eradication programme :

1. Regular testing work in all animals.
2. Segregation of positive rectors from non-reactors.
3. But the positive animals to TB can be sent to concentration camps meant for such purpose in this country (India) on account of various factors.
4. Slaughter of positive cases is not considered in view of different factors (social or religious) in India.

Cases of open tuberculosis which show the organisms of tuberculosis in their discharges and secretions like urine, milk etc., must be immediately separated from herd and owners of such milch cattle must be informed about this fact. Such animals can also be considered for transport to segregation camps. Now a days, a lot of several cattle herds are being maintained in increasing numbers at many places in small and big cities in india, so a vigorous programme of tuberculin test should be strictly followed in them. Tuberculin is used to diagnose tuberculosis in animals and it is injected into the body intradermally for diagnosis of TB. The vaccination with B.C.G is costly in young animals but even the vaccination can be too costly matter for the owner. In

short, B.C.G vaccination giving premunity is the only choice for field use. However, tuberculin test and segregation or slaughter of positive reactors is the most satisfactory method of control of tuberculosis. Animals sensitized to some mycobacterial allergens or allergens as found in *Nocardia* spp, advanced tuberculosis, early cases of infected animals, desensitized animals, following tuberculin test, old age and tuberculin of low potency etc., are factors for giving false reactions in execution of tuberculin test in animals.

Johne's Disease (Paratuberculosis)

It is a bacterial disease caused by an acid-fast organism known as *Mycobactrium paratuberculosis* (also called *M.johnei*) Cattle, sheep, goats and deer are affected by this disease. The organisms seem almost entirely confined to the lesions in organs like mesenteric lymph nodes and intestinal mucous membrane of the infected animals. These are usually found in the faecal material of the diseased animals or mucosal tissue pinched from the rectum. But these can also be found in other organs (e.g., mesenteric lymph nodes or other parts of small intestine); or also some different lymphoid tissues. Evidence of Johne's disease is found in the animals of at least 18 months of age and it is usually seen between the age of 3 to 6 years . Ingestion of food and water contaminated with the faeces or infected material produces this disease in healthy animals. Young cattle are more susceptible to infection than old ones. Parturition, lactation and other factors lowering resistance of animals frequently accelerate the disease process in the infected aminals. In short, infection is transmitted by ingestion of contaminated food and water and JD is marked by a long incubation period in the affected animals.

Signs

Faeces in the infected animals is thin, watery, foetid and mixed with gas bubbles. Affected animals are emaciated, dull

and their bony eminences or ribs are very prominent with a little subcutaneous fat or adipose tissue in the gelatinized form. All affected animals suffer from this disease for months or years and usually die of it. Progressive emaciation and persistent diarrhoea are important features of paratuberculosis.

Pathology

Gross Appearances

The main lesions are as follows :

1. Emaciated carcasses with prominent ribs or bony eminences (hide bound condition of the skin).
2. The lesions are more common in the ileum in the region of Peyer's patches or near the ileo-caecal valves. Caecum, colon and rectum also show lesions. The small intestinal mucous membrane may be severely thickened (4 to 5 times of its normal thickness) and is also corrugated. The corrugations in the mucosae, run into longitudinal or transverse directions like the convolutions of the brain. The crests of the folds may show hyperaemia. The intestinal mucous membrane is smooth to touch, shiny in appearance or covered with grey green slime. The mucous membrane of the intestine is intact with no necrosis or ulceration in animals (e.g., cattle). The mesenteric and colic lymph nodes are swollen and oedamatous. The characteristic observation of JD cases is chronic enteritis with persistent diarrhoea in the affected animals.

Microscopic Appearance

Proliferation of plasma cells and endothelioid cells is noticed in the mucosae of the intestinal lesions. There are no changes in the muscular coat of the intestine. There is a disappearance of the normal cells and glands of the intestine with increasing number of proliferated plasma cells etc., in

the mucosae causing swelling or distortion of the villi. The changes like necrosis and caseation are not noticed in the intestine affected with Johne's disease in cattle. The disease process in JD stops at a stage called symplasma stage which is marked by a partial fusion of the cytoplasm in the endothelioid calls and appearance of clefts or channels. The nuclei of the cells are found free in such clefts. The microscopic sections of the small and large intestines reveal lamina propria of the mucosa to be packed with epithelioid cells. These epithelioid cells have foamy cytoplasm and are often multinucleated. Such cells also cause thickening of the submucosae but the muscularis mucosae and the muscularis are left intact. Nests of epithelioid cells are also found in the mesenteric lymph nodes. Acid-fast rods indistinguishable from *M. paratuberculosis* are seen in the epithelioid cells of the sections of intestines and mesenteric lymph nodes stained by Ziehl-Neelsen's method. Necrosis and calcification are noticed in the nodular lesions in the sheep and goats.

Diagnosis

It is based on the following facts :

1. Symptoms and lesions with detection of acid-fast organisms in clumps in the smears from the affected tissues (say, intestinal mucosae mesentric lymph nodes or faecal material stained by Ziehl-Neelsen's stain).

Smears are prepared from mucosal material pinched from rectum. Films of moist faeces are made on the clean glass slides and stained by Z.N. method to look for acid-fast bacteria.

2. Performance of Johnin Test

The process is similar to double intradermal test with tuberculin to detect tuberculosis. 0.2 ml of Johnin (or avian tuberculin) is injected into the dermis on the side of the neck. A diffuse hot and painful swelling appears in 72 hours after the injection of Johnin. The test is very useful to eradicate JD

from herds of cattle.

3. Cultural Examination

Suitable preparations of faecal material are sown on to tubes of serum Dubos agar and reading is taken after 8 - 12 weeks.

4. JD in the infected animals is confirmed by ELISA, AGID and CF tests.

Trematment/Management

Johne's disease is caused by a bacterium which resists treatment by antibiotics etc. However, streptomycin at the rate of 25 mg/lb body weight can be given for long period to have some recovery. Since there is no absolute recovery from this disease, and as such, an advocacy of Johne's disease treatment is not economical for farmers. However, symptomatic treatment can be given to check diarrhoea by giving drugs like streptomycin and furazolidone etc. In short, there is no significant and effective drug to treat JD in the infected animals for the sake of recovery.

Treatment is not an encouraging suggestion because other animals may get infection from the sick infected patients. This disease rapidly spreads through contaminated water and feeds etc., with the faeces of infected animals.

The best way to control Johne's disease is to test the animals for its diagnosis and all the animals found positive for the test should be segregated from non-reactors and the positive animals for JD can be disposed of by sending them into concentration camps. The method followed in Bihar is to send the positive reactors into segregation camps. The excreta or faecal material of sick animal is preferably incinerated. The animals can be protected from Johne's disease by adopting vaccination programme. Proper hygienic conditions must be maintained in farms to prevent spread of the condition from animals to animals. Identification and segregation of JD cases and rigorous vaccination programme

are important steps to control the disease in dairy farms.

Salmoneliosis (Paratyphoid Fever)

Salmonella spp.are Gram negative rod shaped organisms which are incapable to form spores or ferment lactose (non-lactose fermenters). Many of the organisms produce host specific diseases in animals. Some salmonellae like *S. cholerae suis* act like secondary invaders as seen in hog cholera due to a virus. These organisms produce a disease called salmonellosis in different species of animals.

Some of the diseases caused by them are as follows :

Table 5. Organisms, hosts and disease casused

Organisms	Hosts	Diseases caused
1. *Salmonella cholerae suis*	Swine	Enteritis and speticaemia
2. *S. typhi*	Man	Typhoid fever
3. *S. gallinarum*	Poultry	Enteritis, septicaemia and fowl typhoid
4. *S. typhimurium*	Cattle	Enteritis (Salmonellasis)
5. *S. pullorum*	Chicks	Entritis, septicaemia, pullorum disease
6. *S. abortus equi*	Mares	Abortion (Salmonellasis)
7. *S. dublin*	Cattle, swine and sheep	Abortion, enteritis and meningitis
8. *S. typhimurium and S.dublin*	Sheep, goat	
9. *S. anatum*	Ducks Monkeys	Enteritis and septicaemia
10. *S. abortus ovis*	Ewes	Abortion

Food poisoning is caused by salmonellae in man. These organisms produce a powerful, toxin within 18-24 hrs after intake of infected food and the disease is marked by gastroentritis, nausea, vomiting, cramps and diarrhoea in man. It is an important zoonosis.

Signs

These are as follows :

1. Septicaemia and enterocolitis in neonatal ruminants, fowls and pigs upto 4 months of age and high case fatality rate.
2. Toxaemia, dehydration, acute diarrhoea, dysentery fibrinous faecal casts and chronic enteritis.
3. Abortion, dry gangrene of extremities, arthritis and foci of osteomyelitis.
4. Acute enteritis is noticed in almost all species of animals. In short, the usual autopsy findings in acute fatal cases of salmonellosis are enteritis and septicaemia in the affected species of animals and the clinical signs in the infected animals are recumbency, weakness, temperature, abortion in pregnant animals, diarrhoea (an usual sign) and convulsions.

Pathology

The main lesions are :

(1) Septicaemia and enterocolitis

(2) Presence of mucoenteritis in ileum and colon. The mucosae of the intestine is hyperaemic or haemorrhagic. Enlarged mesenteric lymph nodes and haemorrhagic necrotic enteritis are also seen in the infected animals.

(3) The musocae are covered with red yellow or grey exudate and even ulcers are formed in the intestines. Microscopically, haemorrhage, oedema, necrosis and leucocytic infiltration (mainly macrophages) are seen in the mucosae of intestinal wall. In the livers, there are foci of

necrosis and formation of paratyphoid nodules which consist of aggregates of reticuloendothelial cells like histiocytes or macrophages etc. The Kupffer cells are prominent and leucocytes are present in the sinusoids. There is reticuloendothelial hyperplasia in lymphnodes and spleen. Haemorrhage and necrosis are seen in mesenteric lymph nodes. Septicaemia in the animals is marked by petechiae, ecchymoses on the pleura, peritoneum, endocardium, kidneys and meninges. Microscopically, there is fibrinoid necrosis of the vessel walls and hyaline material is deposited in the glomerular capillaries and small vessels of the dermis. Petechial haemorrhages in kidneys, congestion and hepatisation of lungs, skin discolouration (e.g., as seen in pigs), haemorrhagic enteritis, putrid odour of the intestinal contents, diphtheritic pseudomembrane, enlarged mesenteric lymph nodes and thickened wall of gall-bladder are some important changes in salmonellosis.

S. Cholerae suis causes villous atrophy in the ileum of pigs. *S. typhimurium* causes ulcerative proctitis in swine. Rectal stricture in pigs is caused due to fibrous thickening of the submucosa and muscularis anterior to the rectum. *S.abortus equi* causes abortion in mares between the 6th and 9th months of pregnancy. The placenta is oedematous and contains focal haemorrhages and shows necrosis in a few days. Oedema and haemorrhage are found in the foetus. Infected young ones are born weak and die, later, within a few days.

Carrier animals shed salmonellae in the faeces and transmit the Salmonella spp. to healthy stock of animals.

Diagnosis

Symptoms, gross and microscopic lesions alongwith isolation and identification of the organisms help the diagnosis of the salmonellosis in the animals. An immunoflourescence

method is followed for its diagnosis.Antigen-capure ELISA,Polymerase chain reaction (PCR) and DNA probes are important tests to diagnose the cases of salmonellossis, Serum ELISA idenfies *S.dublin* carriers Leucopenia, neutropenia, hyponitraemia and faecal leucocyte count are indirect tests of salmonellosis. Important signs and lesions etc., of salmonellosis in some domestic animals are given for making differential dignoses (table 6)

Table 6. Some Signs, Lesions and Features in Salmonellosis of Animals

Sl No.	Animals	Signs, Lesions and Features etc.
1.	Cattle (bovines)	1. Septicaemia in neonatal ruminants. Fever,depression, toxaemia dyspnoea, weakness, diarrhoea and dysentery etc., are seen in calves. 2. Intermittent diarrhoea in calves affected with chronic enteritis owing to salmonellosis. 3. Outbreak in cattle caused by *S.typhimurium.* 4. Spread by direct or indirect contact. 5. Milk may be contaminated with faeces of infected cows. 6. Salmonellae invade the blood of bovines and cause the persistence of carrier state subsequent to invasion of blood by salmonellae. 7. Abortion in cows due to *S.dublin* infection. 8. Dry gangrene (due to end arteritis) in the extremities of ears, limbs and tails etc., in calves. 9. Septicaemia, abortion and enteritis in cattle and sheep infected with *S.cholerae suis* associated with deficiency of some mineral or vitamin in certain geographical zones.

2.	Pigs (swines)	1. Septicaemia in pigs upto 4 months of age and outbreaks of salmonillosis in suckling pigs. 2. Enteritis, meningitis, encephalitis, colitis and pneumonia etc., in infected pigs. 3. Erosions, necrosis and oedema in caecal mucosa and slough of necrotic intestinal mucous membrane. 4. Dark to purple discolouration of skin and subcutaneous tissue in *S. cholerae suis* infection.
3.	Goats (caprines)	1. Peracute septicaemia and acute enteritis in goats.

Treatment/Management :

Before administration of antimicrobial antibiotics, it is better to find out the drug sensitivity of the organisms in question.

Antibiotics like ampicillin, oriprim and chloramphenicol are found to be quite useful. In severe cases, one can administer intravenously chloramphenicol at the dose rate of 20 mg per kg body weight six hourly in the animals like cattle, horse, sheep and pigs.

Nitrofurazone at the rate of 20 mg per kg body weight can be given daily for 5 days by oral route.

Demulcent and astringent preparation and fluid therapy can be given in view of factors like dehydration and imbalances of electrolytes.

Vit. B complex can also be given to patients for quick recovery. It is very important to have strict hygienic precautions. The houses should be well ventilated and there should not be overcrowding in animal sheds. As recovered cases may exist as carriers, steps should be taken to eliminate such animals. It is advisable to vaccinate the cows in their late pregnancy for giving passive immunity to the calves.

The passive immunity lasts about six weeks. The calves must be allowed adequate amount of colostrum from their mother to control salmonellosis in animals. Identification of carrier animals, restricted movement of animals, clear water supply hygenic disinfection of buildings, disposal of contaminated material and vaccination of livestock are important preventive measures to control salmonellosis

Actinomycosis

It is also called lumpy jaw in cattle which is caused by *Actinomyces bovis*. Cattle and pigs are commonly affected by these organisms but its infection is rare in sheep and goats. The organisms seem to enter inside the body of the host through injuries in the mucosae of the alimentary tract. Colonies formed by *A. bovis* are gritty to the touch on the cut surfaces of the lesions in bones or other tissues in the infected animals. The most marked feature of lumpy jaw is a rarefying osteomyelitis of jaw bones. *A.bovis* lives in the mouth of cattle and can also invade and grow in the visceral organs of the affected hosts.

Pathology

The lesions in the tissues of animals infected with *A. bovis* are of a granulomatous kind and the organisms are found embedded in the granulation tissue or found as granules in the pus of suppurative lesions. The swollen lesions may discharge sticky honey like exudates containing minute hard yellowish white granules called sulphur granules.

In cattle, the jaw bones and its surrounding structures show the granulomatous lesion or a painless bony swelling on the mandible and maxillae. Involvement of the tongue is an occasional incidence. The bones, particularly, the maxillae and soft structures like gums, palate and tongue show actinomycotic lesions characterized by granulomatous reaction. The swellings on the bones are hard and immovable. Lymph nodes in the vicinity get infected by direct extension. There is no metastasis via the lymphatic channels as seen in

the cases of actinobacillosis. Suppurative osteitis is found in the maxillae. A mass of granulation tissue with soft purulent centres is found in the medullary cavity of the maxillae. Several sulphur granules are seen in such purulent centres. The bone adjacent to these growths becomes absorbed and rarefied and the formation of a new bone on its outside forms-what is called lumpy jaw. Sinuses leading to the surface of the skin with discharge of pus develop in such affected areas (maxillary region). Actinomycosis is found in the mammary gland of sows. Chronic mastitis is found in such sows. Firmness or induration and fibrosis of the nodular type with suppurative changes are found in such affected mammary glands. Gums and thorax show lesions of *A. bovis* in the dogs and cats. Impaired digestion arises from the lesions in the oesophageal groove. Orchitis and abscesses in the lungs and brain are noticed in infected cattle.

Microscopically, the stained sections of the lesions of actinomycosis show a granular centre as a mass of Gram positive filaments matted together to form a mycelium. This mycelium is surrounded by a zone of clubs. The filamentous centre in the old colonies becomes degenerated and has a structureless granular appearance. (Fig. 3; p. 199) Dichtomous branching of the filaments in the colonies can be seen. The clubs appear as a homogenous structure with swollen ends. Formation of clubs is also known in botryomycosis (a staphylococcosis). The clubs are usually Gram negative and mycelial centre is Gram positive in the sections of the lesions stained by Gram's method.

Diagnosis

It is based on the following :

(i) Appearance of lesions (lumpy jaw in cattle), nodular or firm lesions in the mammary gland in sows and presence of Gram negative clubs in the Gram positive mycelia in the granules in the lesions helps one to diagnose actinomycosis.

(ii) Isolation and identification of *A.bovis* is done to confirm its diagnosis.

(ii) Animal inoculation

Two male guinea pigs are inoculated intraperitoneally with culture or fresh material. Multiple peritoneal nodules develop in the infected guinea pigs. These experimental animals inoculated with *Actinomyces bovis*. do not die due its infection.

The following table 7 gives the main differences between actionmycosis and actionbacillosis.

Table 7. Difference between actinomycosis and actinobacillosis

Actinomycosis	Actionbacillosis
1. The centers of the colonies containing short rods or branching filaments are gram positive with gram negative clubbed periphery in the stained sections of lesions in the tissues. In short, actinomyces are gram positive but the peripheral clubs are usually gram negative.	1. Gram negative mycelia center surrounded by gram negative clubs in the colonies of *A. lignieresi* in the stained sections of lesions in the tissues. The organisms are small and gram negative.
2. Bones (chiefly jaw-bones) and adjacent soft tissues are affected.	2. There is a tendency of the organisms to attack soft tissues and lymph nodes.
3. Spread of infection by blood channels.	3. Spread of infection by lymph channels.
4. Calcification.	4. No calcification
5. Lymph nodes are not affected.	5. Lymph nodes affected.
6. Minute yellowish granules in pus of the abscesses in organs.	6. Minute white flecks or granules.
7. Animal inoculation. There are multiple peritoneal nodules but no death in the inoculated guinea pigs intraperitoneally with actinomyces is seen.	7. Animal inoculation. Male guinea pige inoculated intraperitoneally with fresh material shows Strauss reaction and death in 7 days.

8. Granular mycelial centre and zone of somewhat less distinct clubs surrounded by leucocytes and fibroblasts in the stained section from the lesions in jawbones are some main observations.	8. Colonies of *A. lignieresi* surrounded by a zone of leucocytes, epitheliod cells and fibroblasts in stained sectinos of lesions are main findings. A few giant cells may be seen around the finger like clubs of the rosettes (rose like structures) in tissues sections.

Actinobacillosis

It is a chronic infectious disease caused by *Actinobacillus lignieresi* (an inhabitant of alimentary tract) occurring mainly in cattle. The characteristic lesion in this disease is a granulomatous one which occurs in tongue or soft tissues of head and internal organs. The organism *A. lignieresi* invades the injured areas in the mouth, pharynx or intestines etc., to enter different kinds of tissues (especially soft tissues) to produce the lesions. The granulomatous growth of the tissue marked by inflammation and abscessation in this infection contains foci of pus having clubs radiating from the centres of the masses called rosettes. Microscopically, these rosettes are surrounded by giant cells, epithelioid cells and neutrophiles. The tumour like mass in the tongue produces serious disability and leads to emaciation and inanition in the affected animals. Lingual actinobacillosis is noticed in solipeds like horses. Granulomatous lesions develop in the lips and cheeks of sheep infected with this organism and may extend into the mucous membrane of turbinates and soft tissues of head and neck.

Pathology

The lesions occur in the tongue, pharynx, gums, palate and neighbouring lymph nodes of the affected animals. Other organs like stomach, intestines, lungs, liver and skin etc., also show its lesions. There is an occasional involvement of the

jaw bone. Metastatic lesions develop by lymphatic stream and there is a frequent involvement of the lymphatic lymp h nodes. The granulomas developing in these organs contain several colonies of the organisms. Grossly, these are firm, hard and the colonies of the organisms look like yellowish white specks on the cut surfaces of the granulomas. Finger like clubs or projections abutting the bacterial colonies are surrounded by cellular granulation tissue consisting of epithelioid cells, a few giant cells and leucocytes. Fibroblasts form masses of fibrous tissue beyond these infiltrating inflammatory cells. The lesions may be soft and suppurative in nature. Bacterial colonies form granules in the pus present inside the granulomas. The granulomas in the submucosa or muscular tissue or tongue may be equal to peas or hazel nuts. The affected tongues may be hard or indurated due to fibrous tissue formation and ulcers may be found in the submucosae of these organs. Such hard and firm tongues are called wooden tongues in animals. The enlarged or distorted tongues may protrude from the mouth. These lesions may assume the shape of a wart or of a polypoid to fungoid growth. Tongue lesions usually do not exist in form of abscesses. Mucosal ulcers or diffuse thickenings of the submucosae due to granulation tissue can also be found on the gums or palates and characteristic bacterial colonies are seen in such lesions. Ulcers of polypoid lesions are also seen in the pharynx and cirrhotic lesions containing bacterial colonies are noticed in the internal organs. The lungs contain fibrous growth or sponge like cavities. Chronic pleuritis with extension of fibrosis along the interlobular septa occur in the lungs. Lymphatic nodes draining such areas also become infected and show nodules on their cut surfaces.

In short, the typical granulomatous lesions of the actinobacillosis consists of the following facts :

(i) Small Gram negative colonies at the centers of the granules surrounded by a zone of Gram negative clubs (like Indian clubs). These clubs are radially arranged with

their ends outwards to form a palisade around the colonies of *A. lignieresi.* The organisms can be found between these clubs and stain blue in the sections staind by Ziehl-Neelsen's method. The clubs stain red with carbol fuchsin used in this method.

(ii) There is a discharge of pus to the exterior from granulomatous lesions in the soft tissues like tongue.

Diagnosis

Its diagnosis is based on the following :

1. Appearance of the granulomas in organs like tongue (wooden tongue) and presence of granules in the pus of the suppurative lesions. Inability to eat properly and excessive salivation is noticed.
2. Detection of Gram negative bacilli in the smears prepared from the crushed granules. A small sample of pus is examined for the presence of granules. Presence of lymphadenitis, snoring and enlarged retropharyngeal lymph nodes helps its diagnosis.
3. Isolation and identification of *A. lignieresi* by cultural methods.
4. Presence of ulcers on the surface of the palate and thickened interlobular septa in the lung or pleurae.
5. Animal Inoculation.

Two male guinea pigs are inoculated intraperitoneally with culture or fresh pus. Strauss reaction is given by *Actinobacillus spp.* and infected guinea pigs die in about 7 days.

Treatment of Actinomycosis/Actinobacillosis

For treating actinomycosis/actinobacillosis, the use of iodides is a standard procedure. Iodide reduces the severity of fibrous tissue reaction. Potassium iodide (6 to 10 gms/ with Sodabicarb 10 gm) are given per day for the period of 7 to 10 days orally in the form of drench to cattle. This is

continued till iodisim develops and this state of iodisim can be recoganised by lacrimation, anorexia and development of dandruff. Sodium iodide as 5 to 20% solution (1gm/12kg body weight) can be injected I/V through slow drips. Such administration of drug can be repeated and it can not be given in the advanced stage of pregnancy. Antibiotics like streptomycin can be given in high doses for 6 to 8 days. There can be adoption of one course of Potassium Iodide or one injection of Sodium Iodide for curing lesions in soft tissues. Sulphonamides are of greater value in the treatment of actinobacillosis.

The treatment can be repeated once or twice after 10 to 14 days of interval in cases of bony lesions. Bony lesions rarely disappear and exist in a subsided state. Animals receiving Sodium Iodide may show reaction like restlessness, dyspnoea, tachychardia and staggering. Sodium Iodide can also be given by s/c route and is suitable for sheep. In cattle, it causes severe irritation and swelling at the site of injection. Triple sulph can also be given @ 1 gm per 15 lbs body wt. per day for 4 to 5 days. Other drugs like isoniazid (3 to 5 mgms per lb body wt.) can be given orally for 2 to 3 weeks. Treatment is discontinued with onset of iodism. Avoid contamination of feed, troughs and pasture to control this disease.

Contagious Bovine Pleuropneumona (CBPP)

It is a highly contagious disease of cattle caused by pleumorphic *Mycoplasma mycoides* (sub sp. mycoides). The causative agent *Mycoplasma mycoides* sub sp. *mycoides* exists in acute, sub-acute or chronic cases of CBPP in cattle. Pneumonia, serofibrinous pleurisy and oedema of the interlobular septae of the lungs are found in the affected animals. The organism (a mycoplasma) is isolated from the blood and tissues like lungs and thoracic exudates and some forms of mycoplasma are also filterable. An infection of healthy animals can occur due to inhalation of the organisms expelled by the infected animals and the carrier animals are

called lungers. Such animals have some infected foci in their lungs. A focus can exist as a sequestrum equal to the size of a pea or a large orange. Such focus also possesses a fibrous capsule but the capsule breaks down or bursts and viable organisms escape into the bronchi and get expelled with the expectorate. Contaminated discharge into air may infect in contact animals. The incubation period ranges from 3 to 6 weeks and mortality may vary from 10 to 15 per cent. Direct contact of the diseased animals with healthy ones spreads the infection. Adult cattle are more susceptible than calves under 12 months of age. An attack of the natural infection of CBPP is followed by a stong immunity in cattle and vaccination has an important role in its control. CBPP has been reported in Asiatic countries.

Signs

Animals affected with acute form of the disease show a rise in temperature, rapid respiration, a rough coat, anorexia and a dejected appearance. There is a laboured breathing in the affected animals which may also show grunting. Symptoms are less marked in sub-acute or chronic cases and recovered animals are potential carriers. In the acute disease, there is a dry and painful cough, laboured breathing and dyspnoea in the sick cattle. The young calves show swelling of the joints, polyarthritis. The infection spreading to foetus and placenta in the pregnant animals can cause abortion. Rare natural infection of CBPP is noticed in buffaloes and antelopes.

Pathology

The main lesions are as follows :

(i) Lymphatics bring the infection to the lungs which show lesions of bronchopneumonia or pleuropneumonia. The lesions which may be firm or prominent may occur in one or both lungs. Yellowish white thickened septae around various coloured lobules of lungs impart a marbled

appearance to the pulmonary parenchyma. The interlobular septae contain clear straw coloured exudate (i.e., lymph). Later, the fluid may be gelatinized and fibrotic changes (called organization) causes thickening of the interlobular septae. The lymphatics in the septae may be dilated and prominent enough to be visible to the naked eyes imparting a beaded appearance to the septae. Fibrinous pneumonia develops in the lungs. Red, gray or orange coloured lobules can be found in the lungs causing marbled appearance of the pulmonary tissue. Thrombosis causes deep red haemorrhagic infarcts in the lobules of the lungs. Later, pleurisy of serofibrinous nature marked by a clear yellow brown fluid containing fibrinous pieces develops in the thorax and pleurae may be thickened or firm with development of adhesive lesions in the thorax. Necrotic pulmonary tissue is surrounded by fibrous capsule to form what is called a sequestrum. Small sequestra get absorbed but bigger ones communicate with bronchioles leading to expulsion of infected droplets during expiration on coughing. When the thorax is opened, the lungs do not collapse. The lobules may reveal both patent alveoli, and the interlobular septae are distended with amber coloured fluid but there may be complete consolidation in others. Infiltration of lymphocytes and plasma cells etc., can be seen around the bronchi and blood vessels. Round cell infiltration can be seen in the portal triads and the liver cells near the central veins may show necrotic changes. In the spleen, the germinal centres are enlarged with decrease in the number of mature lymphocytes. The plasma cells may be found in increased numbers. Red blood cells and blood pigments are found in excess in the sections of spleen.

Diagnosis

It is based on the symptoms or lesions in the diseased animals and detection of the mycoplasma (i.e., *M. mycodies)* in the smears from the pleural fluid by dark ground illumination. Organisms are to be isolated for the sake of

confirmatory diagnosis. A histopathological examination also aids the diagnosis and complement fixation test and FAT are also useful in detecting infection in the suspected cases. The polymerase chain reaction (PCR) is useful in identifying the specific organism in the infected patients.

Tremtment/Management

For treating contagious bovine pleuropnuemonia, the drugs like streptomycin, chloramphenicol and terramycin are quite useful ones. Tylosin tartarate 2 to 5 ml per pound body wt. should be given to animals i/m every six hours.

For controlling CBPP tail tip vaccination with natural lymph or culture can be done. A live attenuated vaccine is also available which can be given in a dose of 0.2 ml at the ear tip. Immunity conferred lasts about six months. The animals recovered from the disease are in immune state. For effective control free movements of such cases to healthy areas should be discouraged. Animals can also be tested for CBPP and the positive ones can be slaughtered or disposed of. Infected animals are not usually treated and more stress is given on vaccination, establishment of disease free herds and controlled movement of the infected case to control the spread of this disease.

Enterotoxaemia

It is a pathological toxaemic condition caused by different kinds of *Clostridium perfringens* types (A, B, C and D etc.) in domestic animals. Pulpy kidney disease, lamb dysentery, struck and enterotoxic haemorrhagic enteritis are some of the diseases caused by these organisms. *Cl. perfringens* type A has been isolated from the cases of diarrhoea in horses and pigs. The details of some diseases are as follows :

(i) Pulpy Kidney Disease (PKD)

It is a disease of fattening lambs, adult sheep, calves and goats caused by *Clostridium perfringens* type D. The

organisms which proliferate in the intestine, liberate epsilon toxin and damage vascular channels and nervous tissues. Nervous symptoms and sudden death happen in the affected animals. This bacterial infection may last over half to few hours. Heads are pushed against the solid objects and convulsions, depression , opisthotonus, respiratory distress, diarrhoea, abdnominal pain and comma are seen in the sick animals. The organisms are found in the intestine and liberate strong toxins to cause death. Lambs (3-10 weeks of age) and goats of all ages are affected by it. Animals in good health or on a rising plane of nutrition are easy targets of its infection *Cl. perfringens* type D produces a toxin called epsilon. Table 8 gives important information about toxins released by clostridial species and their pathologic effects produced in different animals.

Table 8. Diseases Produced by Clostridial Species, the Toxins Released and Pathologic Effect in the Infected Animals

Species	Diseases Produced	Toxins Released	Pathological Effects
1.*Clostridium chauvoei*	Black quarter (black leg) Animals susceptible cattle and sheep	1. Alpha toxin (a Dnase) 2. Beta toxin 3. Gamma toxin (a hyaluronidase) 4. Delta toxin (a haemolysin) 5. A lethal toxin	1. Necrotic and haemolytic changes by alpha toxin 2. Cardiac myositis and death by lethal toxin produced by *Cl. chauvoei*
2. *Clotridium prefrigens* (type A)	Enterotoxaemia in lambs, cattle, goats, horses and pigs etc.	1. Alpha toxin produced by *Cl. perfringens* type A	1. Gas, gangrene, food poisoning and enterotoxaemia in lambs cattle, goats and horses. 2. Necrosis, haemolysis and increased vascular permeability caused by alpha toxin

Cl. haemolyticum	Bovine bacillary haemoglobinuria.	1. Beta toxin.	1. Icterus, hepatic infarcts, venous thrombosis and haemoglobiluria.
Cl. novyi	Black disease is caused by *Cl. novyi* type B insheep and wound infections in many species.	1. Alpha toxin produced by *Cl. novyi* type A. 2. Alpha and beta toxins. produced by *Cl. novyi* type B. 3. *Cl. novyi* type C.	1. Necrotising and lethal toxins produced by *Cl. novyi* 2. Alpha and beta toxins have necrotising haemolytic and lethal toxic effects 1. *Cl. novyi* type C is non-toxigenic 2. Anaerobiasis is created by migrating flukes like *Fasciola* spp. and *Dicrocoelium* spp. favouring growth of *Cl. novyi* and release of necrotizing toxins for liver cells by this organism.
Cl. sordelli	Wound infections and gangrene in many species.		
Cl. tetani	Tetanus in many species.	1. Tetano-spasmin. 2. Tetanolysin (a haemolysin). 3. Nonspasmodic toxin.	1. Tetanospasmin travelling along the peripheral nerve trunks reaches the central nervous system to produce spasms. Haemolysis is caused by tetanolysin. Non-spasmogenic toxin interferes with motor nerve functions.

Cl. perfringens type B	Lamb dysentery.	Alpha, beta and epsilon-toxins produced.	1. Lamb dysentery enterotoxaemia in calves and foals etc. **1.** Necrosis and vascular permeability produced by beta and epsilon toxin.
Cl. perfringens type C	Struck.	Alpha and beta toxins produced.	1. Enterotoxaemia (necrotic enteritis) in lambs, goats, cattle, pigs, struck in adult sheep.
Cl. perfringens type D	Pulpy kidney disease.	Alpha and epsilon toxins produced.	1. Enterotoxaemia (pulpy kidney disease) in sheep, goats and cattle. 2. Epsilon toxin causes vascular permeability and tissue necrosis.
Cl. perfringens type E	No significant infection.	Alpha and iota toxins produced.	1. Enterotoxaemia in calves and lambs. 2. Iota toxin increases vascular permeability and necrotic changes caused.
Cl. septicum	Malignant oedema and braxy in sheep.	1. Alpha toxin.	1. Haemolysis and necrosis caused by alpha toxin
Cl. haemolyticum	Bovine bacillary haemoglobinuria.	1. Beta toxin.	1. Icterus, hepatic infarcts, venous thrombosis and haemoglobiluria
Cl. botulinum *Cl. botulinum* type A *Cl. botulinum* type B *Cl. botulinum* type C *Cl. botulinum* type D	Botulism in many species.	1. Type A toxin. 2. Type B toxin. 3. Type C toxin. 4. Type D toxin.	Botulism in man Limber neck in fowls Lame sickness (loin disease) in cattle due to chewing of decomposed bones (called pica) in phosphorus deficiency.

Pathology

The following are the main lesions produced by *Cl. perfringens* type D :

(i) Petechial and ecchymotic haemorrhages are seen sub-epicardially and sub-endocardially. Such haemorrhages are also present on the serosal surfaces of the intestine, abdominal muscles, diaphragm and thymus.

(ii) Hydropericardium.

(iii) Hyperglycemia, glycosuria, distension of the rumen, retilculum, abomasum and lower intestine by gas.

(iv) Mild catarrhal gastroenteritis and presence of bloody intestinal contents.

(v) Distended gall bladder as a sign of digestive dysfuncition.

(vi) Pulpy kidneys. The kidneys of dead animals examined soon after death show a blotchy appearance and congested cortex. The kidneys, later, disintegrate and break down into a soft pulpy mass (called pulpy kidneys). This characteristic feature is valuable in the autopsy of dead animals conducted a few hours after death. But a cautious opinion is required on a carcass of a long dead animal.

(vii) Nervous lesions i.e., lesions in the central nervous system like malacia of the basal ganglia, substantia nigra, thalamus and demyelination in subcortical white matter and cerebral peuduncles are found.

Diagnosis

(1) Examination of the stained thin smears of intestinal contents, any inflamed or ulcerated areas by Gram's method to detect presence of clostridial organisms.

(2) Mouse toxin test and demonstration of epsilon toxin.

Mice are injected I/V 0.03 ml of the supernatant material or mixture of the suspected contents of the intestine. These

experimental animals are observed for 15 minutes and again after 24 hours. Deaths in the mice within 3 minutes are due to nonspecific factors, shock or air emboli etc.

(3) An ELISA is quite specific to confirm its diagnosis.

Treatment/Management

PKD is a toxaemic condition caused by *Clostrudium perfringens* type D growing in the intestine of the animals and is marked by diarrhoea, convulsion etc., and as such, the treatment is adopted in light of the aforesaid facts. Anti-epsilon anti-toxin is very effective in such enterotoxaemic cases.

Hyperimmune serum is quite beneficial for the sick animals. Sulphadimidine and penicillin can be given orally to animals. Absorbent can also be given to produce chelating effects against toxins. It is better to give kaolin and calcium for having such effects.

The ewes should be vaccinated prior to 6 weeks and 2 weeks before lambing in order to have two vaccinations. The revaccination can be done in the subsequent year. Colostrum provides passive immunity to the newly born ones (e.g., calves). After vaccination, no immunity can be found in calves up to the period of 10 days.

The vaccine used is an activated alum precipitated toxoid. A multiple vaccine consisting toxoid of enorotoxaemia, tetanus, black leg and braxy can be used.

Clostridium Perfringens Type-A Enterotoxaemia

It is an enterotoxaemia of short duration in the lambs and calves with a very high rate of mortality.

Signs

Icterus, haemolytic anaemia and haemoglobinuria are the main signs in the affected animals in the haemolytic form

of *Cl. perfringens* type A infection. Some other important signs are severe depression, collapse and dyspnoea in sick animals.

Pathology

The main lesions are :

(i) Exessive pericardial fluid

(ii) Dark kidneys

(iii) Enlarged pale and friable liver, hydropericardium and pulmonary oedema

(iii) Extensive necrotic changes in the intestine

Lamb Dysentery

(*Cl.perfringens* type B enterotoxaemia)

It is an enterotoxaemia caused by *Cl. perfringens* type B. Lambs less then two weeks of age are affected. Foals and calves also suffer from it. Lambs are reluctant to suckle, lie down and show signs of abdominal pain. Faeces are semifluid, brownish and may have blood. Recumbency, coma and death within 24 hours after the onset of symptoms are noticed in the diseased animals.

Pathology

Presence of haemorrhagic enteritis with ulceration, petechiae and ecchymoses are seen on serous surface of epicardium and endocardium. Excessive amount of pericardial fluid is also found. In acute disease, the contents are blood stained. In short, the main lesions are haemorrhagic enteritis and ulceration.

Cl. perfringens type C Enterotoxaemia

It is a type of entrotoxaemia caused by *Cl. perfringens* type C in adult sheep and is known as struck marked by sudden death. Haemorrhagic entritis and ulcerative changes are found in the mucosae of the duodenum and jejunum. Presence of peritonitis with a large volume of clear fluid is

noticed in the peritoneal cavity. Type C enterotoxaemia is also noticed in lambs and goats. Both alpha and beta toxins are produced by *Cl. perfringens type C.*

Enterotoxic Haemorrhagic Entritis

It is also a type of enterotoxaemia caused by *Cl.perfringens* type C in calves and lambs. Enterotoxaemia is caused in suckling piglets, usually during the first week of life. Diarrhoea and haemorrhagic enteritis with ulceration are noticed in calves and lambs.

Haemorrhages also occur in epicardium and thymus. Piglets die within 12 to 48 hours. Depression, dehydration, diarrhoea (blood often present) haemorrhagic lymphadenitis of draining lymph nodes, serosanguinous fluid in the peritoneal, pleural and pericardial cavities are main pathological changes. Haemorrhage is seen in the epicardium, endocardium and kidneys of the sick piglets.

Another type of *Cl. perfringens* type E is found in lambs and calves. This organism is of no significance.

Treatment/Management

Enterotoxaemia is a toxic disease in animals like sheep, calf and goat etc. The patients can be given hyperimmune antiserum. Calf can be given 25 ml of antiserum s/c. Annual vaccination can be done by using vaccine and immunity conferred lasts over 9 months.

The patients can be given broad-spectrum antibiotics like chlortetracycline hydrochloride and terramycin in drinking water. Absorbent can also be given for having chelating effects in the alimentary tract of sick animals.

The enterotoxaemia in cattle can be controlled by regular vaccination of toxoid in time. The enterotoxaemia vaccine is polyvalent one and can be given in doses of 1 cc s/c. Supportive treatment such as fluid therapy, haematinics and Vit B. complex may be used.

Black Leg

Synonyms, Black quarter, *Langari* (Hindi)

It is a highly fatal and febrile bacterial disease caused by *Clostridium chauvoei* in ruminants (particularly cattle) and characterized by swollen and emphysematous or crepitating lesions of the muscles and subcutaneous tissues in some region in the bodies such as the shoulder or the hind quarter of the affected animals. The infectious myositis is the most marked lesion. The organisms exist in the soils and are also found in large numbers in the local muscle lesions and exudates around such lesions. Young cattle on high plane of nutrition between the age of six months to two years are chiefly affected by this disease. Grazing infected animals suffer from lameness, depression, anorexia and ruminal stasis etc. Outbreaks of black quarter have been found in sheep of all ages after lambing, docking and castration etc., and this disease occurs as an wound infection in the sheep. Horses are refractory. Ruminants in good nutritional state fall victims to this infection. *Cl. chauvoei* are Gram positive and may have central or subterminal spores. BQ patients die of severe toxaemia and myonecrosis of skeletal or cardiac muscles.

Signs

It occurs as an acute disease usually with a fatal ending. Animals are found dead without showing signs of illness. There is a characteristic extension of the limbs a short time after death of the affected animals. Lameness is noticed in the sick animals. High temperature (106^{0}F), increased pulse rate (100-120/min.) are seen in the sick animals. Sheep reveal myositis, lameness and depression in the cases of *Cl. chauvoei* infection.

Pathology

The local lesions occur mainly in some muscular parts of the body and these lesions are found in the shoulders, hind quarters, neck, back and loins etc. Lesions of black

quarter do not occur below the hock or knee in the ruminants. These lesions in the muscles are hot, painful insensitive (cold) and (painless). Blackened, soft, emphysematous and necrotic muscles infiltrated with gas bubbles produce a crackling sound as heard from application of pressure on them. Affected animals die within 24 to 40 hours. A large amount of blood stained fluid or dirty red exudate escapes from the oedematous tissues. Bubbles of gas are found in the exudate. The affected muscles are dry, black or of dark colour with dissection of the muscle by infiltrating gases because of bacterial growth. An odour of rancid or sweetish butter is emitted by the muscular lesions and the adjoining lymph nodes are swollen. Serous cavities contain blood tinged fluid and internal organs show degenerative changes. Microscopically, the affected muscles show spherical spots (gases) separating muscle bundles and fascia. The muscles show areas of necrosis and collections of neutrophiles and lymphocytes along the muscle septae. Gram-positive bacteria are seen singly or in small irregular clumps in the muscles. The skin over the affected muscles shows dryness, cracks and discolouration etc.

Diagnosis

It is based on the clinical symptoms and the characteristic crepitating lesions in the muscle of the black quarter cases. Culture from needle biopsy is done to isolate the causative bacteria. The organisms of black quarter can be seen in the smears of the exudate and can also be isolated in cultures of the infected materials. Guinea pig inoculation is done to confirm diagnosis of black quarter. Impression smears from peritoneum or liver capsules show the bacteria in ones, twos or threes but not in long chains. There is no tendency to form long chains as seen in the case of *Cl.septique* infection. The organisms of black quarter have rounded ends and occasional spores near the ends of the bacterial bodies. Fluorescent antibody test identifies the causative bacteria in the muscular lesions.

Treatment/ Management

Animals are given parenterally penicillin by i/v route to have quick effects. Tetracycline can also be tried by i/v or intraperitonealy or by i/m route. Since it is a bad toxaemic condition with high fever, antiallergic and antipyretic drugs should be given. In the cases of abscesses in gluteal muscles, the abscesses should be opened and properly drained and washed antiseptically with betadine lotion. Penicillin inj. may be given around the abscesses to facilitate quick healing of the wounds.

Control

In enzootic areas, all cattle between 6 months and 2 years of age should be vaccinated with BQ vaccine before onset of monsoon. Monsoon rains and inclement weather increase the susceptibility of cattle. Animals can be given vaccines by s/c and the immunity lasts for about 6 months to one year. The movements of animals from infected areas should be controlled and there should be constant surveillance to check the disease. Vaccines should also be given to sheep about 2 to 3 weeks before shearing.

The dead bodies of the infected animals are buried deeply in order to prevent spread of infection. Practice of following annual revaccination programme in enzootic areas is highly recommended to combat with the black quarter in animals in time.

The vaccination schedule of BQ vaccine is as follows:

BQ vaccine- a monovac (inactivated culture of *Cl. chauvoei*)	Dosage Cattle, buffaloes and calves 2ml s/c	Remarks 1st vaccination at 6 months or above with annual revaccinaiton

Tetanus (Lock Jaw)

It is an infectious fatal disease of all domestic animals

caused by *Clostridium tetani.* The organisms are Gram positive and have terminal spores. The toxin produced by *Cl. tetani* is absorbed from the site of its production, travels along the peripheral nerves and exerts action on the motor nerve cells of the cord to cause spasmodic contractions of muscles in the body. State of hyperaesthesia, opisthotonus and enhanced reflex excitability are produced in cases of tetanus. Tetanospasmin and tetanolysin, two toxins produced by the tetanus bacilli which induce the toxaemic state and haemolytic changes. Red cell are haemolysed by tetanolysin. The organisms are frequently found in the alimentary tract of horses. *Cl.tetani* is found confined to the wounds and toxins produced therein cause symptoms i.e., tetany or convulsions are noticed in the infected animals. Horses are most often affected and the disease lasts 3-10 days in them. Sheep (on being castrated or docked) and cattle with the wounds in their bodies suffer from this disease. Fowls are resistant to it. Contaminated nails or instruments which have injured the animals, may cause tetanus in them. Dirty contaminated objects infect wounds in the body with appearance of symptoms like tetanic spasms and arrested respiration. A wound is found in the cases of tetanus but in an idiopathic tetanus, no wound or no history of injury is found in the body of the patient. Tetanus is found in animals during and after parturition and in young animals with unhealed infected umbilicus and its incubation period varies four days to 3 weeks. Death is noticed in 3-5 days from the onset of symptoms. Carnivorous animals rarely suffer from tetanus.

Signs

These are as follows :

Animals	Signs
Horses	Erection of the ears and the tails, rigidity or spasms of the muscles, visibility of the membrana nictitans, lock jaw and presence of umbilical wounds in new born foals.

Pigs	Tetanic spasms and presence of castration wounds, if any.
Cattle	Tetanic spasms and presence of wounds of castration or nose ringing, umbilical wounds and infected genital tract after calving.
Sheep	Tetanic spasms, presence of wounds following castration and docking and opisthotonus in lambs.

Pathology

There are no specific lesions in cases of tetanus. Patches of hyperaemia are noticed in the spinal cord, medulla and along the peripheral nerves. Only local wounds as generation sites of tetanus toxins can be found in patients.

Diagnosis

It is based on the following :

(i) Symptoms (almost similar symptoms noticed in all animals) and detection of Gram positive rods with terminal spherical spores in the smears prepared from the wounds.

(ii) Isolation of the organisms in culture from pus or wound discharge and identification of the isolate.

(iii) Inoculation of mice or guinea pigs with material from the wound or culture of the organisms. In this test, the control is one which has been given a prophylatic dose of antitoxin.

Treatment/Management

The tetanus is a toxaemic condition. It is very difficult to treat it after the appearance of the symptoms. Horses and sheep respond poorly to treatment but treatment leads to recovery in cattle.

For treating tetanus, the steps adopted are the following:

(i) Elimination of causative agents by using suitable antibiotics.

(ii) Neutralization of the residual toxin in the body.

(iii) Relaxation of muscle spasms and avoidance of asphyxia.

(iv) Maintenance of hydration and nutrition.

It is proper to find out the site of wound or infection in the body which is thoroughly cleaned and dressed with antiseptic solution (for this, hydrogen peroxide is preferred). The antitetanus serum is used to check tetanus intoxication. If the ATS is given with the onset of tetanus, it has got laudable effects. The ATS can be given by s/c or i/v route. 3,00000 I.U. of ATS can be given 12 hourly to horses upto a maximum of three injections. Antibiotics like penicillin and terramycin can destroy the organisms in the body. Chloralhydrate can be given orally (i.e., 30 gm) and Magsulph (20 to 25%) can be given s/c to tetanus patients. Chloropromazine can be used i/m @ 1 mg/Kg body wt at the interval of 8 - 12 hours till the disappearance of tetany. Animals should be kept in the house comfortably in a quite place. Sound, noise and sunlight should be avoided to lessen irritability in tetanus in the animal patients. Use of enema and catheterization reduces animal discomfort. Animals can be fed by stomach tube or by i/v injection.

In order to control tetanus, strong aseptic conditions during operation or in treatment of wound should be adopted. Protective vaccination (toxoid injection) of costly animals can be done in the cases of presence of wound on the body caused by trauma etc.

The immunity in tetanus lasts a year and it can be maintained for an other year by next injection of toxoid. Use of antitoxic immune serum confers passive immunity in the animals and such immunity lasts a few weeks.

Castration and dockings etc., should be done very carefully to avoid contamination of the wounds. In short,

aseptic precautions ward off tetanus infection in animals.

Malignant Oedema

It is bacterial disease of horses, sheep and cattle caused by *Clostridium septique* which is quite ubiquitous in nature with its presence in the cultivated soil or intestinal tract of herbivores. These organisms are Gram positive and may produce infection in animals by entering the tissues of the body through the skin or mucosal wounds. Organisms like *Cl. chauvoei, Cl. perfringens, Cl. sordelli and Cl. novyi* have been isolated from the cases of malignant oedema.

Lowered resistance of the mucosae favours the penetration of the organisms in the body. The organisms are common postmortem invaders. Human beings are vary susceptible to *Cl.septique* infection and the organisms are found in gas gangrene associated with war wounds. Shearing, docking or faulty parturition may be followed by malignant oedema. Animals of all ages and species are affected by *Cl. septique.*

Pathology

This disease has a short duration. The lesions are hot and painful at site of infection. The wounds, later, become oedematous, less painful and cooler. Gas bubbles or haemorrhages are seen inside the lesions. There is often, development of septicaemia. The lungs may be congested or oedematous. Oedema of subcutaneous and intermuscular tissues around the site of infection or gangrenous areas in the skin are important lesions in the cases of gas gangrene.

Diagnosis

It is based on the symptoms and lesions. The organisms are readily demonstrable in affected tissues and found to form long coiling filaments on the peritoneal surface of the liver of the guinea pigs inoculated with *Cl .septique.* In *Cl. chauvoei* infection, single or organisms in short chains are seen in the

liver impression smears of the infected experimental cases. FAT is conducted to confirm its diagnosis.

Treatment/Management

Malignant oedema is a toxaemic disease of cattle, horse, sheep, goats and swines etc. The disease responds well to therapy to penicillin or broad spectrum antibiotics. Antitoxin, if available, can also be used. The wounds in the body of the animals should be treated with H_2O_2 (hydrogen peroxide). Cold pack is used over the site of wound to minimize the absorption of toxins.

In order to control the disease, the following steps are to be adopted:

(1) Antiseptics like acridine dyes and betadine lotion etc. can be used for dressing the wounds.

(2) Lambing, shearing, castration and docking should take place under hygienic conditions.

(3) Vaccination can be done in the calves.

(4) Penicillin must be given in adequate doses to reduce the infection.

(5) When there is a development of high fever in malignant oedema with toxaemia, antipyretics and corticosteroids can be administered.

Botulism

It is a toxaemia called food poisoning in man and animals characterized by the symptom of paralysis. This disease in poultry is known as limberneck. A soluble exotoxin (neurotoxin) is produced by it and the disease produced is not an infection but a state of intoxication. Botulism in man has been caused by eating sausages or decomposed animals products. This organism exists in the soil or nature as a saprophyte *Cl. botulinum* B, C and D are associated food poisoning in animals.

The main types of *Cl. botulinum* are as follows :

Types

A

B

Ca

Cb

D

E

F

The aforesaid types are serologically distinct and the toxin of any one is not neutralized by antitoxin of the other. The toxins produced by different Clostridial species are given in the table 8. No signs are seen in the peracute cases of botulism.

Signs

The course of the disease depends on the amount of toxin ingested and incubation period ranges from a few hours to 10 days. Affected animals die in a few hours. Muscular weakeness, paralysis of the jaw is noticed. No fever is seen but the prostration of the body occurs. In birds, extreme weakness, sluggish movement, drooping wings, beak and head resting on the floor (limber neck) are the main signs of the disease. Wild ducks ingest contaminated material in shallow ponds and lakes etc. Spoiled canned contaminated food given to chicks produces limber-neck in them. Nutritionally deficient cattle ingest decaying decomposed bits of meat clinging to the bones of dead animals in aphosphorosis and get intoxicated with *Cl. botulinum.*

Pathology

No specific lesions are found in the dead animals. Punctiform haemorrhages can be found in the brain, spinal cord and lungs. The toxin produced by *Cl.botulinum* does

not act on the nervous system but act at the myoneural junction of the peripheral nervous system. The symptom of paralysis is mediated through the prevention of release of acetylcholine. In limberneck, the affected birds show sleepiness, a flaccid paralysis of neck muscles and usually the birds die.

Diagnosis

It is based on the symptoms, isolation and identification of the organism. Filtrated extract of the saline suspension of the contaminated food is injected into mice or guinea pigs to produce the disease. Preformed toxin of *Cl.botulinum* is demonstrated in food material by injecting filterate of the suspected food material subcutaneously into guinea pigs. The inoculated pigs die within 12 hours in positive cases. Preformed toxins of *Cl. botulinum* may be present in the contaminated feeds.

Treatment/Management

Specific or polyvalent antitoxic serum is useful in early stages of the disease. Purgatives are used to remove toxins from the body.

Vaccinate the animals with type specific precipitated toxoid in the enzootic areas. Prevention of intake of preformed toxins in the feeds and improvement of the feed management are very important measures to control the food poisoning in animals.

Braxy

It is an acute infections bacterial disease of sheep caused by *Clostridium septicum* and the characteristic primary lesion is an acute abomasitis. Predisposing factor like ingestion of frosted foods may lower vitality of the mucous membrane and facilitate the penetration of the abomosal mucosae by the organisms which may, later, enter the blood stream. Young sheep or the best conditioned animals are very

susceptible to this disease. The incubation period is very short and the sheep found sick in the late evening may die during the night hours. Death may be seen even all of a sudden in *Cl. septicum* infection.

Pathology

The abomasal mucosa is thickened, oedematous and haemorrhagic. Such congested, oedematous and necrotic or ulcerated lesions are also seen in the intestinal mucous membrane. The mucosae will be necrotic and the bacteria can be found in the tissue sections of the affected animals.

Diagnosis

It is based on the symptoms, lesions, isolation and identification of the bacilli obtained from the lesions. FAT confirms its diagnosis.

Treatment/Management

There is no use of doing any treatment in the case of braxy because of its very high mortality due to toxaemia, high fever and depression etc. The animals affected with *Cl. septicum* die within a few hours with the prominent lesions of abomasitis. However, this disease can be controlled by proper management of the herds. The sheep should be kept in yard at night and not allowed to graze forsted pasture. It is better to feed the sheep before driving these animals for grazing during morning hours. The vaccine consisting of formaline killed *Cl. septicum* is quite useful.

Gas Gangrene

It is caused by *Clostridium* spp. and associated with war wounds in human beings. The counterpart of the malignant oedema (a disease of animals) occurring in humans is called gas gangrene. In gas gangrene, there is an excessive destruction of tissue leading to necrosis and gangrene. Oedema and gas production are quite marked changes in

the affected tissues. Experimental inoculation of *Cl. welchii, Cl. septique, Cl. oedematiens or Cl. chauvoei* in animals produces gas gangrene in them. Advanced techniques of treatment of the wounds have very much lessened incidence of this condition. The organisms found in clostrodial wound infection are as follows :

(1) *Cl. perfringens*
(2) *Cl. novyi*
(3) *Cl. sordelli*
(4) *Cl. feseri*
(5) *Cl. hitolyticum* and
(6) *Cl carnis.*

Treatment/Management

The wounds should be properly dressed with antiseptic solution. Penicillin at the higher dose rate can be used to treat the condition.

Bovine Baciliary Haemoglobinuria (Infectious Ictero Haemoglobinuria)

It is a bacterial disease of bovines. Evidence is seen in favour of *Cl. haemolyticum* which produces this disease in cattle and sheep. It produces the powerful haemolytic toxin in the infected body.

Signs

The disease is sudden with onset of toxaemia and haemoglobinuria and is marked by high fever, collapse and death within a day or two following infection in the animals.

Pathology

Large areas of infarcts surrounded by hyperaemic zone are found in the livers of affected animals. Hepatic damages caused by *Fusobacterium necroforum* and liver flukes favour rapid growth of *Cl. haemolyticum* in the liver parenchyma.

The toxins produced by the organisms produce the characteristic constant lesions like liver infarcts in this disease. Venous thrombi are found in the liver. Haemorrhages and haematuria are noticed in the body of affected animals dying of anoxaemia.

Diagnosis

It is based on the constant lesions like hepatic infarcts, haemoglobinuria and isolation of *Cl. haemolyticum* from the hepatic lesions. FAT is helpful in confirming its diagnosis.

Treatment/Management

The animal patients of this disease should be given immediate treatment. The affected animals are administered penicillin or tetracycline in high doses. Antitoxic serum (500 to 1000 ml) can be given to sick animals. Supportive treatment in form of blood transfusion, parenteral fluid therapy and electrolyte solution can be given.

Animals should be given mineral supplement containing iron, copper and cobalt. The patients may have sufficient rest at least for three weeks after recovery in view of damage done to liver and destruction of red cells.

Formalin killed whole culture absorbed on aluminium hydroxide provides immunity for a year to cattle. All the susceptible stock should be vaccinated before the expected period of disease occurrence. Annual vaccination can also be done.

Abortion

It refers to the expulsion of a dead embryo or foetus previous to the end of full term of pregnancy. Sometimes, the expelled embryo is too small to be observed. It can occur at any stage before the full term of pregnancy in animals. Abortions occur at the 7 month of pregnancy in brucellosis. It is frequent in the farm animals but it is quite rare in dogs and cats. In short, it is an immature end of pregnancy with

expulsion of dead or living embryo or foetus.

Causes

1. When the foetus dies in the uterus, it becomes like a foreign body and its very death leads to abortion. The dead foetus is like a foreign substance in the uterus which attempts to get rid of it. Injuries to the tissues of placental union occur with the onset of inflammation, necrosis, hyalinization and degenerative changes. Injured placenta fails to supply nutrients and oxygen to the foetus which dies and is then expelled by the uterus. In cases of intoxication, the foetus gets poisoned and dies. In equine viral abortion, the foetus dies and is expelled like a foreign object.

2. Erogot Poisoning

Erogot causes violet abnormal contractions of uterine muscles to cause foetal expulsion. In such poisoning, no death of the foetus occurs.

3. Hormonal Imbalances

When progestational effect of the corpus luteum is eliminated or neutralized early, an abortion can occur. Injection of large amout of oestrogen or manual removal of corpus luteum can bring an end to pregnancy owing to disturbances in the action of progesterone.

4. Infections

Infections injuring the placenta and factors preventing implantation of the embryos (for example, placentitis etc.) can cause abortion. An acute or septicaemic infection produces anoxia because of generalized venous congestion and the foetus dies with its ultimate fate of abortion.

5. Deficiencies

Certain deficiencies lead to a state of deprivation in the mother or its foetus. Deficiencies of minerals, oxygen and vitamins can even prevent conception rather than abortion in animals.

(6) Trauma

Traumatic injury can lead to death of the foetus which is expelled later.

(7) Mummified Foetus

A dead foetus is an irritating foreign substance in the uterus and is soon expelled. Putrefactive or pathogenic bacteria invade the dead foetus and the state of maternal infection and septicaemia is produced. When the cervix is closed, the uterine contents are sterile and the foetus undergoes postmortem autolysis and may not get expelled. The soft tissues are liquified and the liquid formed is absorbed by the maternal blood or lymph. The foetus is, then, expelled as a mass of bones with the shrunken or wrinkled skin. The foetus looks like a dried or shrivelled mummy.

Retained Foetal Membranes

Placentitis (inflammation of placenta) leads to retention of the foetal membranes. Chorion is separated from maternal structures owning to acute swelling of chorionic laminations. Post-mortem autolysis and putrefaction take place in the retained placenta. A retained placenta is life-less, devoid of blood supply and is also exposed to external contamination with bacteria through the cervix. The disintegration of chorion loosens its attachment to the endometrium and the placenta is expelled in fragments. Toxic products produced by putrefactive or pathogenic bacteria enter the body of the animals to produce condition of toxaemia. Infectious organisms can also enter the uterus through the vagina *Clostridum tetani* can grow in the placenta and produce a disease called tetanus.

Hydatiform Mole

Small bits of chorion remain even attached to the endometrium and derive nutrients. The cells of the chorionic villi proliferate to produce an irregular mass of cystic

structures called hydatiform mole. A malignant epithelial neoplasm called chorinoepithelioma arises from tissues of the foetal villi or hydatiform mole. Multinucleated giant cells are found in such tumours.

Hydrops Amnii

It means an oedema of the amnion i.e., presence of two much fluid in an amniotic cavity (upto 3 to 6 litres in mare and cow). 6 to 15 litres of fluid can be found in the allantoic cavity. Rotation of uterus or twist of the umbilical cord can give rise to the state of hydrops amnii. Oedema arises on account of the compression of the veins. This condition may last in cow until parturition. The foetus may die and is then expelled from the body.

Diseases or disorders of the uterus causing abortions and placentitis (inflammation of placentitis) in the animals.

Placentitis is caused by several infectious agents. It ends in abortion (expulsion of the foetus). Granulomatous inflammation in the chorionic villi is caused by tuberculous bacilli. The foetus dies before the development of the tubercles on the chorion and it gets expelled by the uterus.

The following are the important diseases causing **abortions** in the animals :

(1) Brucellosis

Brucella spp. causes placentitis. Bang's diseases is caused by *Br. abortus* in cattle. In acute brucellosis, there is an extensive formation of seropurulent exudate between the chorion and endometrium in the interplacental areas (chorionic leaves). This leads to separation of the surfaces with subsequent expulsion or abortion. Chorion shows inflammatory oedema and there is also infiltration of reticuloendotheial cells, lymphocytes, plasma cells and neutrophiles. Chorionic epithelium is rich in bacterium. Necrosis extends in the allantochorion which undergoes

hyalinization and has also brownish colour with leathery consistency. Weakening of the placental connection between foetus and mother ends in abortion. The foetus may survive to be born alive. A chronic proliferative form of placentitis occurs and chorion is tied to the endometrium due to diffuse or sparse fibrosis and, thus, the placenta is retained in animals like cows. A very few cows abort more than thrice and abortions in the cows are noticed mainly towards the end of pregnancy. Unvaccinated heifers suffer from abortion due to *Br. abortus* infection usually after 5th month of pregnancy.

Vibriosis

In this disease, there is placentitis caused by *Vibrioi* spp. Necrosis and cellular infiltration are noticed in the placental structures and there is formation of serofibrinous exudate in the inter-placental areas. The smears prepared from placentomes show Gram negative curved organisms. Foetus is killed in vibriosis in ewes about one month before full term and no permanent injuries are found in the foetus. The foetal liver is discoloured and changes like necrosis and perivascular infiltration of neutrophiles and eosinophiles are found. Vaginitis, metritis, infertility and abortions are some important findings in vibriosis.

Trichomoniasis

It is an important disease of cattle caused by *Trichomonas foetus*. *T. foetus* infection spreads from infected vagina to uterus which show endometritis and placentitis. Copious pyometra and a mild purulent reaction in the endometrium is produced. In chronic cases of trichomoniasis, the foetal membranes are retained usually following abortion during the 1st half of gestation. The organisms are noticed in foetal membranes.

Paratyphoid Abortion of Mares

The main lesions of this disease caused by *Salmonella abortus equi* are purulent haemorrhagic placentitis and necrosis of chorionic villi. Abortion occurs between the 4th and 8th month of pregnancy. The organisms are present in the foetal membranes, uterine exudate, blood and also internal organs like stomach and intestines of the foetus.

Equine Virus Abortion

The main lesions of this disease include oedema of foetal membranes and necrotic foci in the livers of the aborted foetuses. The liver cells around these necrotic foci show intranuclear inclusions. The foetal lung is also oedematous. Viral infection is present in the foetus which is also icteric. Foetus is infected between 8th to 11th month of pregnancy and abortions take place between the 9th and 10th month of pregnancy. The virus kills the foetus, which is, later expelled. Ninety per cent of the infected mares abort due to this viral infection.

Treatment/Management

In order to adopt logical treatment of abortion, it is very important to find out the cause of abortion. When causes of abortion have been established, specific treatment can be adopted. In case of abortion due to *Salmonella abortus equi,* drugs used for salmonellosis are quite beneficial. There should be proper management of housing condition. Drugs used in treating the cases of salmonellosis can be safely given to treat abortion in view of symptoms shown by the animals. In brucellosis, treatment is not successful due to intracellular sequestration of *Br. abortus* in the lymph nodes, mammary glands and reproductive organs. This organism is known to multiply in the **macrophages** of the body of infected animals.

Brucellosis (Bang's Disease in Cattle)

It is a very impotant zoonotic bacterial disease of cattle caused *Brucella abortus* and leads to abortion or birth of very weak off-springs or still born ones. Metritis, infertility, interruption in breeding programmes and loss of milk in the dairy stock are important features of the disease. Bang (1896) discovered this organism in Denmark in cattle. Acute catarrhal inflammation and degenerative changes are produced in the cotyledons of the gravid uterus with the subsequent death of the foetus, abortion and its expulsion from the uterus. *Br. abortus* forms abscesses in the testicles. It does not lead a saprophytic existence outside the body of the infected animals and grows in the pregnant uterus between the wall of the uterus and chorion and also in the chorionic epithelium. It shows tissue predilection e.g., pregnant uterus, udder, testicle, lymph nodes and joints etc. Organisms are present in the catarrhal discharge of the uterus and in pure culture form in the stomach contents of the foetus. The calves show congenital form of the disease. Lungs and other tissues also contain the organisms of *Br. abortus* and the organisms can be found in the discharge from the uterus and vagina within 3 weeks of calving or abortion. Tissues like udder, supra mammary and iliac lymph nodes also contain *Br. abortus* in infected animals even for months or years. All affected cattle secrete organisms in their milk and the elimination of the organisms may be confined to one or more quarters. Abortion in cattle occurs within the last three months of pregnancy in the most cases. The infection in the cattle occurs by the mouth or by ingestion of food or water contaminated by uterine discharge or aborted foetuses. Infection can also occur through the vagina, conjunctiva and skin (even intact one). Infected bulls also transmit the infection to healthy cows through the contaminated seminal fluid. Testicles show inflammatory liquefactive or degenerative changes due to *Br. abortus* infection. Painful swellings are noticed in the scrotum of infected bulls. Eighty

per cent of the infected cows abort only once. Affected bulls show enlarged seminal vesicles, become sterile, and scrotum may be swollen. *Br. abrotus* is a Gram negative, non acid fast and non sporulating rod like organism.

Pathology

The main lesions are :

(1) Placentitis leading to abortion of the foetus and necrotic granulomatous changes in the placenta.

(2) Discharge of yellowish brown slimy flocculent aborted odourless discharge from the vagina in the aborted cases.

(3) A yellow necrotic appearance of the foetal membranes or uterine cotyledous. The chorion becomes thick and the intercotyledonary areas present a leather like wrinkling. *Br. abrotus* is found in the scrappings of such tissues. The placenta may be dull or granular.

(4) Endometritis and retention of the foetal membrane.

(5) Presence of subacute or chronic inflammatory changes in foci in the alveoli, inter-alveolar tissue and lactiferous ducts of the infected mammary glands in the cattle

(6) Presence of orchitis, epididymitis and vesicullitis in the infected bulls. Scrotal lymph nodes may show inflammatory changes.

(7) Synovitis and hygroma are also seen in the cattle.

(8) Phagocytic cells are attracted towards the *Brucella* organisms which are engulfed by them. The organisms grow and multiply in the cytoplasm of these cells. Later, epithelioid cells accumulate at these sites. The early microscopic lesion in the tissues infected with such organisms is a tiny nodule of epithelioid cells surrounded by a narrow zone of lymphocytes. Later, caseous necrosis can occur at the centres of such lesions which may be surrounded by a fibrous capsule formed at the periphery. Neutrophiles and lymphocytes get attracted towards such necrosed cells. Frank abscesses and nodules or frank

granulomas are rarely formed.

The aborted foetus shows oedema and serosanguinous fluid is present in the body cavities. The mammary glands and supramammary lymph nodes show diffuse inflammation with presence of lymphocytes, neutrophiles, epithelioid cells and occasional Langhan's giant cells. The scrotum in the bulls becomes enlarged and indurated. The tunica vagina may be thickened, compressing or replacing the testicle or epididymis. There can be rare suppuration in the scrotum.

Diagnosis

It is based on the symptoms, lesions and isolation of the organisms by cultural methods or by the inoculation of guinea pigs with suspected material. Guinea pigs are destroyed after a month and cultures are made from spleen to isolate the *Burcella* spp. Agglutination test is quite useful in the diagnosis of *Brucells* spp. infection and a positive reaction is one in which there is complete agglutination in serum dilution of 1 in 40 or higher. Agglutination in the suspected sera at one in 20 but incomplete at one in 40 gives a suspicious reaction. Other tests to diagnose *Br. abortus* infection in animals are complement fixation and abortion or brucellin tests.

In case of abortion, stomach contents of the foetus are inoculated on to blood agar and MacConkey's agar aerobically and under 10 per cent Co_2. The plates are examined for growth of brucella, vibrio etc. Placenta is examined for necrosis on cotyledons or in intercotyledonary areas. Smears from such lesions are stained by Gram's method and examined under oil immersion objective for *Brucella* spp. and *Vibrio* spp. etc. Bacteriological examination of milk is done by inoculating the mixture of the cream and deposit intramuscularly into the left leg of guinea pigs. The animal is bled and killed after a week and the blood is examined by the agglutination test and the body is examined for the lesions of brucellosis.

Treatment/Management

The attempt for treating brucellosis in animals is usually not made. But, however, sulpha drugs and antibiotics can be used. Streptomycin and chloramphenicol can be given to the cases of brucellosis. There can be intraperitoneal injection of teteracycline in a single dose of 10 mg and it can be repeated after 3 days.

For control of brucellosis, specific programme of testings like serological agglutination test can be adopted and the reactors can be segregated from healthy stock. Hygienic methods are very much useful in controlling brucellosis. This includes isolation and segregation of infected animals, proper disposal of aborted foetuses, placenta and uterine discharges etc. Cattle sheds and premises are thoroughly disinfected. The infected animals can be isolated for thirty days and retested.

For controlling brucellosis, vaccination of the animals with *Brucella abortus* strain 19 living vaccine is quite useful. This vaccine protects the healthy animal from contacting infection in contaminated environment. For any control programme, the method of isolating and disposing of infected ones is very useful one. Calves getting infection after birth remain infected till they are adult.

The vaccination of female calf can be done between 4 to 8 months of age. If adult cattle are vaccinated, abortion rate comes down but this is not advisable. Bulls and pregnant animals are not vaccinated. Cows vaccinated in the advanced stage of pregnancy may abort. Reactions of vaccination are seen in the form of high fever, anorexia, decrease in milk yield and local reaction.

There is a single 5 ml dose of *Brucella abortus* strain 19 which is given s/c at the age of six months in calves. The immunity conferred in calves is adequate one for five or more lactations.

The followings methods of eradication can be followed:

(1) During an outbreak of abortion, it is proper to vaccinate all reactors.

(2) Disposal of all reactors is not satisfactory.

(3) Herds free of abortion should be tested and vaccinated immediately and positive reactors should be culled out or disposed of.

(4) In lightly infected herds, all calves are to be vaccinated and positive reactors should be culled.

(5) An eradication programme envisages attempts to reduce susceptibility of cattle population till the incidence becomes very low and the eradication programme by test and then disposal becomes quite economical. For controlling brucellosis in sheep, vaccination can be done and it is also better to isolate the infected ones.

Swine Erysipelas

It is a bacterial disease caused by *Erysipelothrix rhusiopathiae* (*E. insidosa*) in pigs. The organisms are Gram positive, non-sporing and non-motile. In less severe form of the disease, rhomboid - shaped areas of erythema are seen in the skin and the disease is so called as diamond skin disease. Acute and chronic forms of the diseases in animals are noticed. Septicaemia, skin discolouration, high fever (upto 108^0F) and gastroentritis are seen in the acute infections. The chronic form is recognized by unthrifitines, endocarditis and lameness due to arthritis. Human beings handling the infected pigs show cutaneous lesions (erysipeloids) on the hands, face or over the body. Pigs between 3 to 9 months of age are affected and those over one year of age are resistant to it. Ingestion of contaminated food or water produces infection of the disease in pigs. Carrier stage is also seen in the pigs. Incubation period varies from 1 to 5 days. This organism also infects birds like turkeys, chickens, ducks and geese etc.

There are four forms of this disease :

(1) Acute form

(2) Urticarial form (Diamond skin disease)

(3) Chronic form

(4) Arthritic form

1. Acute or Septicaemic Form

Signs

Pyrexia, inappetence, thirst, vomiting, conjunctivitis and prostration are seen in the affected pigs. Bright or dark red patches are present over the ears, neck, abdomen and inside the thigh and forelegs. These patches get covered with scabs. Tips of ears and tail are necrosed and sloughs of dead tissues are cast off. Eighty per cent of the affected pigs die within 2 to 3 days.

2. Urticarial or Diamond Forms

This form of the disease is mild in nature. Loss of appetite, dullness, stiffness, characteristic quadrangular, deep red or purple patches or blotches with a paler centre on the sides, back and buttock are noticed in the affected pigs. The skin lesions vary from an inch to 2 inches in size. These patches become swollen and covered with crusts which are later thrown off. The skin lesions like diamond shaped plaques in infected pigs are considered pathognomonic.

(3) Chronic form (Cardiac form)

It is usually sequel to acute form or may arise independently. Cardiac insufficiency from chronic valvular endocarditis marked by vegetations on the cardiac valves in pigs may cause death. Dyspnoea, stunted growth, coughing and dark red discolouration of skin and extremities are quite marked signs. Chronic venous congestion may be seen in the lungs and livers of infected pigs. The lungs may be odematous in such cases.

(4) Arthritic or joint form

It may arise independently or be sequel to urticarial form. Some important signs in the patients are stunted growth, difficult movement, back arched and enlarged joints of the limbs.

Pathology

Reddish purple, patchy or diffuse discoloration of skin is seen. Scabs are also formed over such areas. Tips of ear and tail show necrosis. Septicaemic lesions are in the form of haemorrhages on serous membranes, heart muscles and joints.

The lesions in different types of swine erysipelas have been given in tabular form (table 9).

Table 9. Pathological changes in different forms of swine erysipelas.

Acute Form	Joint Form	Chronic Form	Urticarial Form
1. Diamond shaped skin lesions.	Enlarged limb-joints and difficult movement.	Non-proliferative arthritis in limbs and intervertebral joints.	Rhomboid congested areas in the skin with sharp margins of lesions.
2. Diffuse purplish discolouration of the belly and extremities.	Oedema and congestion of synoival tissues and joint capsules.	Synovitis marked by serous or serofibrinous or amber coloured exudates.	Nearly bright red tissues turning into purpulish or dark blue ones.

Acute form	Joint form	Chronic form	Urticarial form
3. Petechial and ecchymotic haemorrhages on the pleura, peritoneum and beneath the renal capsules.	Presence of chronic non-suppurative arthritis.	Thickening of the joint capsules and patches of vascular granulation tissue on the articular surfaces of the joints.	Dry epidermal tissues peel off and their removal leaves behind raw bleeding surfaces.
4. Infarcts in spleen and kidneys and intracellular presence of organisms in the affected tissues.		Large friable vegetations of the heart valves and presence of Gram positive organisms in such lesions. Nodular masses on the mitral or bicuspid valves of the affected ventricles. Hypertrophy of the ventricles due to valvular stenosis.	

Diagnosis

It is based on the lesions and demonstration and isolation of organisms from cardiac and valvular or joint lesions. Acute form of the disease is marked by early leucocytosis. Later, leucopenia and monocytosis are noticed.

Treatment / Management

In infected cases, treatment is not much effective. Long acting penicillin (Penidure) can also be given.

Use of cortisone is also suggested in cases of arthritis. In skin lesions on the bodies of pigs, topical applications (Himax or betadine lotion) can be given. Since this disease may appear as an acute septicaemic form, use of an antipyretic and supportive treatment may be applied. For the sake of controlling swine erysipelas, it is advisable to adopt general hygienic conditions and affected pigs should be isolated and disposed of at the earliest. Eradication becomes a difficult problem on account of existence of *Erysipelothrix rhusiopathiae* as soil borne bacterium. Animals with arthritis and endocarditis must not be allowed to mix with healthy ones. Steps should be taken to immunize animals against swine erysipelas. Immune serum and virulent culture live vaccine using a virulent stain of *E. rhusiopathiae and* inactivated bacterium can also be given. The method followed in U.S.A. for control of swine erysipelas is as follows :

(i) Use of the autogenous killed vaccine.
(ii) Use of immune serum simultaneous with virulent culture.
(iii) Avirulent culture alone.
(iv) Immune serum alone.

Immunity is quite durable in pigs (aged 5 to 8 weeks). The infected animals and new or fresh animals should be kept in quarantine. New or fresh animals should be only acceptable after, keeping them in the quarantine for about fortnight and they should be vaccinated before being added to the herds. Yearly vaccination is followed. There should a thorough disinfection of the premises, utensil etc. Caustic soda, carbolic acid or copper sulphate solution or even hot house soap should be used as disinfectant.

The main symptoms and lesions of different bacterial diseases in animals are given in table 10.

Table 10. Signs and Lesions in Some Important Bacterial Diseases of Domestic Animals

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
1. Anthrax	**Bacillus anthracis** *(Isolated for the first time by Robert Koch in 1876).*	Marked effects of the lethal toxin produced by *Anthrax bacilli* are oedema, tissue damage, acute renal failure, terminal anoxia and death from shock. Oedema factor (EF), protective antigen (PL), lethal factors like I, II, III and EF (an adenylate cyclase) are some exotoxins produced by *Anthrax bacilli.* EF causes increases in intracellular c AMP (cyclic adenosine monophosphate).	A disease of worldwide occurrence. Human beings and all domestic animals are affected by anthrax bacilli. Anthrax bacilli are capsulated organisms which are noticed singly, in twos or threes in the blood smears of the infected cattle and buffaloes etc. 1% methelene blue and Wright or Leishman's stains the organisms in the blood films. The bacilli in short chains (but never in long filaments) appear in the blood of anthrax cases. The blood films give a purple ground work between the bacilli after staining with methelene blue. Heat, moisture and shade favour existence of the anthrax organisms in the soil of tropical countries. Septicaemia is seen in infected animals and organisms degenerate or disintegrate in the	

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
			blood or tissues soon after death of the affected animals. Peracute or acute forms of infection in cattle, buffaloes and sheep etc., is noticed. Death of bovines (cattle and buffaloes) within an hour or so after the onset of symptoms i.e. detection of a sudden onset and death in peracute cases. In acute form, the sick animals die in 10 to 24 hours. It is marked by fever and bleeding from the body openings (mouth and nose etc.). Malignant carbuncle is seen in cutaneous anthrax in man. The important postmortem findings in bovines like cattle and buffaloes are haemorrhagic gastritis and enteritis, an excessive enlargement of the spleen with black pulp (splenomegaly). The blood fails to form clots (i. e., absence of blood clot in the blood vessels). Dark tarry.	1. Based on the symptoms and lesions 2. Staining feature (i.e., M'Fadyean's methylene blue reaction of the organism in the blood or tissues stained by 1% methylene blue. 3. Ascoli's test (precipitin reaction). It is quite dependable in cattle and can be performed using extract of blood, spleen and hide etc. from the suspected cases, Even extract of infected tissues from advanced cases of putrefaction is used to do Ascolis`s test.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
			blood and haemorrhages on the serous membranes. Gelatinous yellow oedema at the mucosae is seen due to entry of the anthrax organisms. Rapid putrefaction is noticed in the dead animals marked by growth of the putrefactive organisms e.g., *Clostridium septique, Cl. welchii* and *Cl. sporogenes.* Marked absence of rigor mortis and lack of blood clotting in the carcasses of dead animals are important features of anthrax infection in animals. More rapid growth of anthrax bacilli in lymph nodes than in other tissues of the patients.	
2. Haemorrhagic septicaemia (HS or Barbone) cattle, buffaloes, sheep, goat and swine affected by *P. multocida.* Dogs are immune to HS.	***Pasteurella multocida* type I (or B) *P. multocida* serotypes B and E are noticed in Asia and Africa.**		A septicaemic pasteurellosis (galaghotu) in cattle and buffaloes in India. Peracute form of disease with high mortality. European cattle die of pneumonic form of pasturellosis. *Pasteurella multocida* produces a fibrino purulent broncho pneumonia in cattle	1. Based on the symptoms and lesions in the carcass 2. Isolation of pasteurellae from blood and spleen of infected dead animals 3. Detection of gram nega-tive or bipolar organism in the

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
			A pneunomonic form of pasteurellosis by *Pasteurella multocida* in swines, sheep and goats is noticed. Mortality rate in cattle, buffaloes and camels by *Pasteurella multocida* type I lies between 50 to 100%. Pasteurellae isolated form tonsillar and nasopharyneal . mucosae of the carriers of this disease. The main signs are acute septicaemia, sudden onset of disease, high fever (106^0-107^0), salivation, submucosal petechiation, depression, restlessness, subcutaneous swelling in intermandibular space,neck, throat and brisket and severe dyspnoea. Generalised petechiation under the serosae, oedema of the lungs, haemorrhagic gastroenteritis and pneumonic lesions are noticed in acute cases of pasterurellosis.	stained film with Wright's or Giemsa's stain. 4. An enzyme linked immu-noassay test (ELISA test) is done to identify pasteurellae. 5. Death of rabbits within 24 hours following inoculation of the isolates from the infected animals.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
3. Blackquarter (BQ) Hindi (langari)	*Clostridium chauvoei Affected animals are cattle and buffaloes Sheep and horses also suffer from it.*	Alphatoxins, betatoxins (a DNase) gammatoxin (a hyaluronidase) and delatatoxin (a haemolysin)	An acute infectious febrile disease of cattle and buffaloes and marked by swollen, painful emphysematous and crepitating lesions in the muscles of young animals (4 months to 2 years of age) or animals in good condition The muscular lesions are dry and infiltrated with gas bubbles. The affected muscles emit rancid odour and produce crackling sound on being pressed with fingers. The characteristic signs are fever, lameness and depression. Cardiac muscles are pale and friable and show myositis in the dead calves. Shearing docking, castration and lambing may cause out breaks of BQ in sheep. Quick development of bloating and putrefaction are also noticed. In the cases of bovines, body cavities reveal the excess of fluid.	1. Based on symptoms lesions and isolation of *Cl. chauvoci* 2. Increase in the levels of SGPT, SGOT and lactic dehydrogenase myositis
4. Botulism (food intoxication or food poisioning)	*Clostridium botulinum Animals suceptable: Cattle, sheep and horses and man.*	1. A powerful neurotoxin	A serious intoxication in animals and birds following ingestion of infected, spoiled or decomposed or spoiled food.	1. Based on the symptoms and isolation of neurotoxins, the causative agent from gut contents of the

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
	Botulism in fowls is called limberneck (paralysis of respiratory muscles) Type C toxin of *Cl. botulinum* causes limberneck in fowls and ducks. Types C and D affect cattle.		Aphosphorosis in cattle is marked by craving for phosphorus (pica). Cattle which chew decomposed bones contaminated with *Cl. botulinum* shows signs of botulism. The neurotoxin produced by *Cl. botulinum* acts at the neuromuscular junction or synapitic clefts and block release of actyl choline (a neurotransmitter) at the motor end plates. As a result, paralysis of the related muscles innervated by cholinergic nerves is noticed. There are no characteristic lesions of botulism in animals and birds etc., as comparable to the negative lesions seen in tetanus.	affected animals. 2. Demonstration of preformed toxin in the food ingested or by injecting saline suspension of gut contents in mice or guinea pigs.
5. Tuberculosis (TB)	*Mycobacterium bovis* Animals affected: Cattle, buffalo, goats and pigs etc.		Tuberculosis is marked by formation of tubercles in the different organs of diseased animals. Tuberculous organisms are noticed in the exhaled air, faeces, sputum, milk, urine and discharges from the infected uterus and peripheral lymph nodes. Animals pick up	1. Based on the symptoms and lesions. 2. Isolation of *M. bovis* from the lesions or discharges. 3. Tuberculin test. 4. Detection of acidfast bacilli in the smears of the lesions of the organs, milk, urine and

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
			infections by ingestion or inhalation of. *M. bovis* in the contaminated milk. Children consuming such milk.becomes infected. In pulmonary tuberculosis, bacilli are present in the expectorated material as fine or large droplets in the air. Hardening , enlargement, caseation and offensive odour from the cut surfaces of the lesions are found in the lymph nodes like pharyngeal, bronchial, medistinal lymph nodes. Caseation calcification and encapsulation by fibrous tissue are noticed in the lesions. A nodule reveals caseation, cheesy consistency and calcified zone due to deposition of calcium salts in the necrosed tissues which are surrounded by cells like epithelioid cells, giant cells of Langhan`s types, lymphocytes and fibrous capsules at its margins of such lesions.	sputum etc., of infected animals.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
6. Jhone's disease (Paratu-berculosis)	*Mycobacterium paratuberculosis. Animals susceptible Cattle, sheep, goats and deer.*		An infectious disease of cattle, sheep and goats etc., marked by emaciation, persistent diarrhoea thin, watery, foetid, faeces mixed with air bubbles. The mucosae of small intestine, caecum colon and rectum show lesions like corrugations in the intestinal mucosae. No necrosis is seen in the intestinal mucosae. Swollen and oedematous mesenteric lymph nodes are often seen in the affected animals.	1. Based on the symptoms and lesions. 2. Isolation of *M. paratuberculosis.* 3. Acid fast organisms in clumps in the smears of intestinal mucosa. 4. Johnin test.
7. Tetanus	*Clostridium tetani All domestic animals are affected but horses are most susceptible*	1. Neurotoxin (tetanospasmin). 2. Haemolysin (tetanolysin). 3. Non-spasamogenic toxin.	A total infectious toxaemic disease of domestic animals and death of almost all affected animals with tetanus. The	1. Based on the symptoms like muscular spasm, prolapse of the 3rd eyelid membrana nictitans and history of some

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
	animals.		important signs are tetanus, hyperaesthesia due to exotoxins of the organisms. No wound is seen in idiopathic tetanus. Punctured wound leads to development of this disease. Castration, shearing and docking parturition in sheep and pigs. lead to tetanus. Neurotoxins travel along the peripheral nerves to reach the central nervous system and cause muscular spasm. Incubation period varies from 1 to 3 weeks. Restricted jaw movements (lock jaw), stiffness of the hind limbs, stiffly held out tails during backing or turning.	injuries or surgical wounds. 2. Isolation of *Cl. tetani* from the sites of wounds or injuries detected in the patients.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
			Prolapse of 3rd eyelid, constipation and retention of urine are important signs of tatanus. Normal range of temperature and pulse noticed in the sick animals. No characteristic lesions are noticed in the animals died of tetanus except some congestion in the spinal cord, medulla and peripheral nerve trunks chosen by the toxins to reach CNS (central nervous system).	
8. Brucellosis (Bang`s disese).	*Brucella abortus A gram-negative organism. Animals susceptible cattle*		An infectious disease marked by abortion towards the end of pregnancy and placentitis causing premature expulsion of the foetus (abortion). A yellowish brown pasty slimy odourless floccule -nt discharge in the case of abortion. A yellow appearance of the necrotic cotyledons in the cases of late abortion, synovitis, hygroma and orchitis, epididymitis and vesiculitis in the infected bulls. The uterus of infected cows usually gets rid of *Br. abortus*	1. Based on the symptoms, lesions and isolation of *Br. abortus* from the lesions in the endometrium or foetal membranes 2. Customary agglutination test (of one in forty dilution of suspected serum) is noticed in positive cases.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
9. Actinomycosis (Lumpy jaw)	*Actinomyces bovis* *Animals susceptible* *Cattle and rare cases in sheep and goats*		*An infectious disease of cattle marked by chronic granulomatous lesions in the jaw bones and surrounding soft tissue. Lesions in the internal organs e.g., lungs are also noticed.* *Suppurative osteitis is seen in the maxillae and affected bones become absorved and rarefied and the sinuses releasing pus open on the skin surface. Granules in the lesions of cows are gritty to the touch. In stained sections of tissue, the microscopic appearance reveals gram positive filaments forming a mycelium, gram-negative clubs in the peripheral zone. Granules in the bony lesions are gritty due to calcification and the causative organisms spread to other organs or parts by blood stream.*	1. Based on the microscopic appearance of the granulomatous lesions. 2. Dectection of gram positive organisms in the stained crushed granules. 3. Isolation and identification of the causative agent from the lesions.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
10. Actionbacillusis (Wooden tongue)	*Actinobacillus ligniersii* Animals susceptible Cattle and occasional cases in sheep		An infectious disease of cattle marked by wooden tongue. Inflammatory lesions are noticed in tongues, gums, palate, pharyngeal lymph nodes and oesophageal groove etc., Granules (called sulphur bodies) are noticed in tissues, purulent discharge, necrotic and granulamatous lesions. Microscopically gram negative organisms are seen in the granules which reveal club like rosettes with a central mass of bacteria. Microscopically, the granulamatous lesions in organs reveal a central zone of bacterial colonies delimiting finger like clubs lecocytes, epithelioid cells and peripheral location of	1. Based on the symptoms, granulomatous appearance of lesions in organs of tissues. Gram negative granules in the pus smears or stained films on the glass slides and isolation of the organisms.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
			fiborblasts. The causative organisms may enter by lymphatics.	
12. Glanders	*Pseudomonas mallei* Solipeds like horses, mules and donkeys affected. Man also suffer from its infection. Cattle and pigs are immune to it.		A contagious disease of solipeds marked by chronic cough, high fever, sticky or oily nasal discharge, ulcerative nodules on the skin and death from septicaemia. A lymphatic infection in equines and organisms present in different organs. Acute and chronic glanders are two forms of the disease. Chronic forms are marked by nodules in the lung parenchyma. Foci of catarrhal and croupous pneumonia and pulmonary haemorrhagic infarcts present. Haemorrhages on	1. Based on the lesions in different organs and isolation of *Pseudomonas mallei.* 2. Strauss test in male guinea pigs characterised by orchitis and pus in the tunica vaginalis following intra peritoneal inoculation of the isolate 3.ELISA, mallein and complement fixation tests confirm the diagnosis of glanders.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
			the serous membranes, congested respiratory mucosae with ulcerated surfaces, nodules in the spleen, hyperaemic and enlarged lymph nodes are some other lesions noticed in acute form. The lesions noticed in the chronic forms are chronic lyphangitis and lymphadenitis (farcy) in affecting fore or hind limbs. Small round nodules (called farcy buds) along the lymphatics in subcutaneous tissue in the affected animals. The farcy buds rupture and leave ulcers with ragged borders. Ulcers also arise from rupture of nodules in the nasal, pharyngeal and tracheal mucosae with punched out appearance. Nasal discharge is yellowish and oily in nature.	

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
13. Mastitis Animals affected Lactating animals, like cattle, buffaloes, sheep and goats etc.	The organisms causing mastitis are as follows: (1) *Streptococcus agalactiae* (2) *Staphylococcus aureus* (3) *Escherichia coli* (4) *Str. zooepidemicus* (5) *Str. pyogenes* (6) *Corynebacterium pyogenes* (7) *Mycobacterium bovis* (9) *Pasteurella multocida* (10) *Mycoplasma bovis* (11) *Fungi like Trichosporon, Aspergillus fumigatus* and yeasts		An infectious disease of lactating animals. Peracute, acute, subacute and chronic mastitis are some different forms of udder infection. A disease of zoonotic potential. Acute mastitis is marked by cardinal signs of inflammation e.g., redness (rubor), swelling (tumor), heat (colour) and pain (dolor) etc. Peracute or acute from of mastitis may cause death of the affected cows. There is no visible abnormality in the milk or udder of subclinical form of mastitis. But a high cell count in the milk may be an evidence of mastitis. Indurated udder and nodular lesions are seen in chronic mastits. Toxaemia, fever, depression, tachycardia,	1. Based on the symptoms and lesions in the lactating mammary glands 2. Staining of the centrifuged milk sediment by Gram's and acidfast staining technique to detect organisms in the milk 3. Isolation and identification of the bacteria obtained from the milk samples 4. Strip cup, cell count, direct capture ELISA test and California mastitis test (CMT), white side test and chloride content are useful in diagnosing cases of mastitis.

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
	(e.g *Candida* and *Troulopsis* spp.etc) (12) *Leptospira interrogans serovar* Pomona and Interrogans (13) Some viruses etc.		anorexia and recumbency are signs of peracute mastitis. Wateriness is marked in the milk of mastitis cases, serous, fibrinous and purulent exudate in acute mastitis. Dead neutrophils (pus cells), vacuolation or compression of the epithelial cell in the alveolar lining, mono nuclear cell or neutrophiles in the alveolar septa are noticed in the cases of mastitis. Capillaries in the alveolar walls are distended with blood in acute mastitis. In chronic contagious mastitis caused *Str. agalactiae*, thickening and cornification of the epithelial lining are present in the lactiferous ducts. Lack of regeneration in the alveoli affected with epithelial destruction and collapse of the alveoli followed by replacement with a mass of proliferated fibrous tissues are marked in	

Diseases	Causes	Toxins Produced	Signs and Pathological Changes	Diagnosis
			chronic mastitis. Hardening and induration are noticed in such cases of chronic contagious mastitis.	
14. Navel-ill (Septic omphalophlebitis)	*E. coli Pasteurella spp., C. pyogenes,* streptococci and staphylococci etc. Animals susceptible: Calves, foals, lambs and piglets etc.		Pre-natal or post natal infection may cause navel ill in animals. Purulent discharge from the open inflamed umbilical veins in the affected calves. No changes are noticed in the normal naval cord (umbilicus). Blood is seen in the umbilical vein in navel ill. Imperfectly coagulated blood is noticed in the infected umbilical vein and bacteria are isolated from such lesions which may show pus or dead tissues on their cut surfaces	1. Based on the symptoms, lesions and isolation of the causative organisms.

Chapter 3
Viral Diseases

Foot and Mouth Disease (FMD)

FMD is a highly contagious viral disease of ruminants i.e., cloven footed animals like cattle, sheep and goats and pigs. It is characterized by fever and formation of vesicles especially in the foot and mouth. Foot and mouth disease virus is an aphthovirus causing aphthous fever in domestic and some wild animals and possesses epidermotropic character (that is, affinity for epithelial cells).

The horse is not susceptible to FMD and reindeers suffer from it. Susceptible animals are infected through ingestion or by droplet infection from in-contact infected FMD cases. This virus may enter respiratory or alimentary mucous membrane to produce infection. There is a high fever (104^0 - 106^0F) with fall in milk in cattle. Contaminated food, water and utensils etc., spread infection to healthy stock. Incubation period varies from 2 to 7 days and vesicles (called aphthae) are seen in the mouth and the foot of the diseased animals. This viral disease has low mortality but it has a great deal of economic impact. FMD virus occasionally occurs in milk or urine of infected animals

Types of the FMD viruses

These are as follows :

1. Vallee O	4. S.A.T. 1	7. Asia-1
2. Vallee A	5. S.A.T. 2	
3. Waldman C	6. S.A.T. 3	

These types are immunologically distinct from each other

and animals recovered from the attack of one type are fully susceptible to infection with another types. Cattle, pigs, sheep and goats are susceptible to it. The disease is transmitted by direct contact between healthy and diseased animals. Tissues and discharges have high infectivity. Airborne droplets of saliva or discharges from infected cases of FMD lead to spread of infection. Animals in good fat conditions are more severely affected than lean animals. Immune status of the population affected also influences the severity of FMD outbreak among cattle and other susceptible animals. In enzootic areas, animals are partially immune. A malignant from of FMD occurs in the young stock of cattle with severe heart damage because of degenerative changes in the heart muscles. Several calves die of cardiac lesions marked by subendocardial yellowish streaks and foci with a high rate of mortality. 50% glycerine in normal sline is a good preservative of this virus in vesicle fluid or epithelium.

Signs

Inappetence, loss of condition, ulcers in the mouth and feet and severe lameness are important symptoms of the disease in animals. Superficial erosions of the feet can be seen with loss of horny covering of the hoof. Degenerative lesions develop in the myocardium of the infected young animals (like calves).

Pathology

Necrotic lesions are found in the stratified epithelium of the tongue, buccal mucosae, rumen and coronary bands or teats. Vesiculation in such tissues occurs with loss of the epithelium covering at a later stage. Ulcers may heal with presence of very little scarring. There is an extensive denudation of epithelial cells because of rupture of the vesicles in the mucosa of tongue. The virus multiples in the stratum germinativum of the epithelium which develops degenerative changes (ballooning of the cells with swelling and pyknosis

of the nuclei). The remnants of epithelial cells get separated from more superficial layers e.g., cornified layers. The space created in this way gets filled with fluid to form vesicles. The vesicles appearing on the mucous membranes of the mouth tongue, dental pads, cheeks and gums and also on the skin of muzzle, of the interdigital space on the teats or on udder rupture to form ulcers. The roof of the vesicle is formed by the stratum lucidum and some cells of the stratum germinativum and the floor by the remnants of the stratum germinativum. The cells of the vesicles are dead and white in color like the skin over blisters. When these dead cells get denuded or sloughed off, a red and painful base is seen in the ulcers. Punctate haemorrhages or diffuse oedema of the mucosae in the abomasum or small intestine can be found. The mucosae of the small intestine may be hyperaemic or blue red. Subpleural, subepithelial or subepicardial haemorrhages can also be seen. Balloon degeneration is seen in the cells in the middle of stratum spinosum of the epidermis. Such cells are loose in the cellular prickles (stratum spinosum), the cells become round or detached from one another.

Diagnosis

The mouth lesions in FMD are differentiated from those of R.P. in view of the depth of the involvement of the epithelium. In FMD, there is a tendency to form an ulcer (dip even upto dermis) with under run edges. The characteristic lesion is a vesicle (an aphthus) containing straw coloured fluid.

Distinguishing features between the FMD, vesicular exanthema and vesicular stomatitis are given in the following table 11. New techniques for identifying different strains are enzyme linked immunosorbent assay (ELISA), reverse transcriptase polymerase chain reaction (RT-PCR) and nucleotide sequence analysis.

Table 11. Some features of FMD, VS and VE

FMD	Vesicular Stomatitis (VS)	Vesicular Exanthema (VE)
1. Cattle and ruminants are affected. Sudden death in young ruminants.	Horse and cattle are affected, occasional incidence in pigs.	Pigs are affected with vesicles in the mouth or snouts and teats etc.
2. Presence of both foot and mouth lesions, fever and profuse salivation.	Generally there are no foot lesions. Vesicular lesions are seen on the oral mucosae, teats and prepuce.	Signs like fever or lesions are not distinguishable from FMD and vesicular stomatitis in pigs is seen.
3. FMD virus (an aphthovirus i.e., a picorna virus) is serologically distinct one.	This virus is serologically distinct one. Vesiculovirus is a member in the family Rhabdoviridae.	The causative virus (a *Calcivirus*) is serologically distinct one.

Isolation and identification of the virus along with tests to detect complement fixing bodies, or virus neutralizing bodies can also be done. The cells become also highly eosinophilic and their nuclei get pyknosed. Liquefaction necrosis or accumulation of serum or leucocytes can extend down to the cells of the basal layer. The vesicles and bullae are seen over the highly congested dermis. Bullae are formed from fusion of the vesicles. When the vesicles rupture, a red raw surface with oozing blood is exposed to exterior. Anorexia arises from pain in the vesicles. In calves or lambs, the cardiac lesions occur in the form of hyaline degeneration and necrosis in the myocardial muscle fibres. The skeletal muscles may also show such lesions. Necrotic areas look like grey foci or spots in the muscles. Features of DNA and RNA viruses are given in Table 12.

Treatment/Management

There is a no specific treatment of foot and mouth

Table 12. DNA and RNA viruses their genetic and some other features

Viruses	Families	Sites of Viral replication	Species/virus types	Genome
A. DNA Viruses				
(i) Double stranded (ds) DNA				
Enveloped (ds) DNA (+/-)	i. Poxviridae (enveloped)	Cytoplasm	Cow pox, small pox, swine pox and fowl fox ' viruses	A linear double stranded DNA molecule (dsDNA)
	ii. Herpesviridae (enveloped)	Nucles	Pseudorabies and Mareks diseases viruses	do
Nonenvelope d	i. Adenoviridae (non-enveloped)	do	Bovine adenovirus and canine adenovirus-1 (Rubarth disease virus)	do
	ii. Papovaviridae (non-enveloped)	do	Bovine, canine and Shope papilloma viruses (rabbits)	Circular super double standed DNA molecule
Single stranded (ss) DNA Nonenvelope d (ss) DNA (+/-)	Parvoviride	do	Feline panleucopenia virus and canine parvovirus-2	A linear single DNA molecule (ssDNA)

B. RNA virsues Double strnaded (ds) RNA Nonenvelope d (ds) RNA (+/-)	Reoviridae (non-enveloped)	Cytoplasm	Bluetongue virsues I-25 and African horse sickness viruses 1-9	10 or 11 molecules of linear double stranded RNA (segmented genome) dsRNA
Bisegmented double stranded (ds) RNA nonenvelope d	Birnaviridae (non enveloped)	do	BNirnavirus	Two molecules of double stranded (segmented genome
Single stranded (ss) RNA ssRNA (-) Nonsegmente d	(i) Paramixovirida e (enveloped)	do	Rinderpest virus and canine distemper virus	A linea single stranded RNA molecule (-ssRNA)
	(ii) Rhaboviridae (enveloped)	do	Rabies virus and bovine ephemero virus	do
	(iii) Bornaviridae	Nucleus	Born disease virus	Three molecules of linear single stranded RNA
Negative sense, segmented	(i) Orthonyxovirid ae (enveloped)	Nucleus	Influenza viruses	8, 7 and 6 molecules of linear single stranded RNA (-ssRNA)
	(ii) Bunyaviridi (enveloped)	Cytoplasm	Rift valley fever virus	3 molecules of linear stranded RNA
(iii) Single stranded (ss) RNA viruses	(i) Coronaviridae (enveloped)	Cytoplasm	Canine Corona virus	A linear Single stranded molecule of RNA (+ss RNA)

	(ii) Picornaviridae (nonenveloped)	do	FMD virus and swine vesicular diseases virus	A linear single stranded RNA molecule (+ssRNA)
	(iii) Astroviridae	do	Astroviruses	do
	(iv) Togaviridae (enveloped)	do	Equine encephalitis virus	do
	(v) Flaviviridae	do	Hogcholera virus and Bovine and viral diarrhoea virus	do
	Calciviridae (non-enveloped)	do	Vesicular exanthema virus of swine	do
c. DNA and RNA reversetranscri bing virsues partly double stranded (ds) DNA/partly single stranded (ss) DNA DNA (nonenveloped)	Hepadnaviridae (non enveloped)	Nucleus	Hepatitis B virsus (Orthohepad na virus) Duck hepatitis virus (Avihepadna virus)	A circular double stranded DNA molecule (ss/ds DNA)
Single stranded (ss) RNA or ss RNA (+)	Retroviridae (enveloped)	do	Bovine leukaemia	Diploid linear single stranded
DNA step in replication (non enveloped)			virus, feline leukaemia virus and avian leucosis virus	RNA molecule
Note :- Subviral agents These are self replicating proteins with no known nuclic acid and are known as prions (PrP)-a protein associated with scrapie.				

disease. However, antibiotics can be used for controlling secondary bacterial infections. Only symptomatic treatment is advisable.

Tissue culture inactivated vaccine containing all the four important strains (O, A, C, and Asia-1) of foot and mouth disease can be used. Calves above four months of age can be given FMD vaccine. There is a need of booster dose after three to four months and revaccination is done every year. When a locality has incidence of FMD, it is better to have revaccination programme after every six months. Use of mild disinfectants and protective dressings on the inflamed parts checks the spread of secondary bacterial infections.

The lesions in mouth are treated with antiseptic electuary of mouth wash (2% alum. soln., boric acid in glycerine; 1 : 2000 soln. of potassium permanganate). Antiseptic ointment (Furacin oint., Himax lotion etc.) may be used on ulcers or the cleft of the hooves. Immunity in the vaccinated animals develops after 15 days and cannot be absolute in vaccinated animals due to involvement of different immunologically distinct strains. It short, mass vaccination with killed vaccines is advisable in the endemic areas of a country. The vaccines against FMD are as follows :

FMD vaccines	Dosage	Remarks
1.FMD vaccines (inactivated strains like O, A,C and ASIA-1) 2. FMD+HS+BQ oil adjuvant vaccine-triovac. 3. FMD+HS oil adjuvant vaccine-biovac.	Cattle, buffaloes and calves 3ml s/c in the mid neck region. Sheep and goats 1ml. s/c ---------do-- ------ ---------do-------	Primary vaccination at 4 months of age Booster to primovaccinate after 2-4 weeks Revaccination 6 months thereafter 1st vaccination at 4 months of age and the revaccination after 9 months followed by annual revaccination.

See tables-37,38 and 39 for different drugs or vaccines used in the treatment of viral diseases.

Rinderpest

It is an acute contagious febrile disease caused by a filterable virus known as rinderpest virus of the genus *Morbillivirus.* Cattle and buffaloes are very susceptible to it and the virus of cattle plague (RP) is present in all the secretions and the excretions of the body. The viral position is intraleucocytic in the blood and the virus is not available in the serum of the patients. Many other ruminants (like cattle and buffaloes) and pigs are also susceptible to rinderpest. Food, water, utensils, hides etc., contaminated with the virus of RP infect the healthy animals. The virus penetrates through the alimentary tract to enter inside the body. There is a natural transmission of disease by direct contact between infected and susceptible animals. Incubation period of RP varies from 3 to 9 days. R.P. runs a milder course in sheep, goat and camels than in cattle but causes an explosive out break in Asiatic countries.

Signs

Affected animals show fever (104^0 to 105^{0}F), profuse diarrhoea and inflammation of mucous membranes in the alimentary tract and small ulcers or erosions are found in its mucosae. In India, RP is an enzootic disease and indigenous cattle suffer from a mild course and the mortality in them is also low, but RP is a serious disease in imported exotic cattle (i.e., foreign breeds or crossbred animals) and mortality in them may exceed over 90 per cent. The disease in the infected animals lasts over 2 to 9 days and these animals usually die. Marked leucopenia is found is RP cases. The body temperature rises to 105^{0}F or higher and remains high for 3 to 10 days before becoming normal. The peak of the fever in RP reaches on the 3rd to 5th day but there is an abrupt drop with the onset of diarrhoea. Lesions within the oral mucosa

are seen on the 2nd or 3rd day of fever. The RP cases show prostration, subnormal temperature and death after a course of 6 to 12 days. High temperature, congested mucous membranes, watering of the eyes, erosions in the mouth, nasal discharge and diarrhoea are important signs to suspect RP in a herd of cattle and buffaloes. The muzzle and mouth are hot. Diarrhoea and arching of back are seen at the later stages. Red spots can be seen on the inner aspects of the lips, cheeks, gums, hard palate and the sides of the tongue. Inflammatory changes are present in the papillae of the cheek and the commissures of the mouth. Greyish yellow necrotic areas replace the red spots on the mouth and nostrils and on being sloughed off, these spots give rise to red ulcers which may be deep or bleeding. The discharges from the mouth are offensive and the breath is foetid. Weakness, exhaustion, sunken eyes, fever and dehydration due to diarrhoea are very important signs of RP patients. In epizootics of R.P., numerous cases of rinderpest are seen all at once in many places but in enzootic cases, the symptoms are less intense and are not so violent as seen in the epizootics of this disease. Restlessness, dryness of the muzzle and constipation can be seen in cattle with the onset of rinderpest, but after a day or two, nasal discharge, larcrimation, photophobia, depression, excessive thirst, retarded rumination, anorexia and excessive salivation (drooling of saliva) can be noticed in the rinderpest cases. Lesions in the oral mucosa occur on the third day of infection and severe diarrhoea develops at later stages.

Pathology

The bodies of the RP patients are dehydrated and emaciated due to rapid loss of condition. Haemorrhages, small papules or pustules can be found in the skin of udder or scrotum etc. The mucous membrane in the rectum is swollen and congested and blood stained foetid faeces can be found around the anal region. Conjunctivitis may be present. Crusts of discoloured mucopurulent discharge are found in the areas around eyes, nostrils and mouth and

carcass is soiled with faeces.

There is congestion in the oral and pharyngeal mucous membranes and red spots and greyish white nodular elevations are found in these mucosae. Later, ulcers or erosions are formed in these red spots or elevations after removal of greyish membranous deposits on surfaces of these nodules or elevations. Erosions and ulcers also extend to pharynx, oesophagus and pyloric region in the abomasum.

The R.P. virus has strong affinity for lymphoid tissues and epithelium of the gastrointestinal tract. Necrosis of the lymphoid tissue is seen in lymph nodes, spleen and Peyer's patches. There is a fragmentation of the nuclei in the germinal centers and disappearance of mature lymphoid cells in the lymphoid follicles. Eosinophilic cytoplasmic inclusion bodies are found inside the multinucleated giant cells and there is also presence of intranuclear inclusions in RP The lymphoid cells are destroyed to leave behind eosinophilic and a cellular matrix, in place of lymphoid cells.

Necrosis of a few epithelial cells occurs in the deep layers of the stratum Malpighi in the oral mucosae. Nuclei show pyknosis, fragmentation and multinucleated giant cells are found in the stratum spinosum. Mucosal epithelial cells and giant cells contain eosinophilic cytoplasmic inclusions. Intranuclear and intracytoplasmic viral inclusions may be present in organs like tonsils. Basal layer of the squamous epithelium is rarely penetrated or destroyed and the ulcers are rarely found in RP in the oral mucosae. The erosions which are shallow with a red raw floor are found in thr oral mucosae. Rarely, lesions are found in the rumen, reticulum and omasum. Congestion and haemorrhages are found in the lamina propria of the abomasal mucosae. Abomasal folds are thickened and oedematous and erosions with red raw floor are seen. There may be deep ulceration in occasional cases. Numerous petechiae are found in the large intestines (e.g., caecum). Chronic passive congestion in the liver can

arise from cardiac or pulmonary complications. At caecocolic junctions, infundibuli are formed due to invagination of necrotic epithelium through muscularis mucosae. A characteristic striped appearance called zebra marking is noticed in the colonic mucosa arising from transversely running zones (stripes) of haemorrhages and congestion in the caecum and rectum etc.

In the respiratory system, petechiae are found on the turbibnates and erosions or petechiae can occur in the larynx. There are sub-endocardial haemorrhages in the heart. A mucopurulent tenacious exudate forms a coating on the nasal turbinates epithelial lining. Removal of such exudates causes erosions or ulcers in the septa.

Diagnosis

It is based on the following facts :

(1) Symptoms, gross and microscopic lesions. These help in making a presumptive diagnosis on a herd basis and not at all on an individual basis.

(2) A challenge of immune and normal control animals is made with splenic suspensions from suspected cases of RP This is done to confirm the diagnosis.

(3) Serum virus neutralization test

(4) Gel diffusion preciptin test is an important aid to differential diagnosis. Agar gel immunodiffusion test using needle biopsy specimens and ELISA test also confirm RP in the outbreaks affected animals.

Some important features of diseases marked by diarrhoea or intestinal lesions are given in table 13.

Table 13. Diseases characterized by Diarrhoea or Intestinal Lesions etc., in Cattle.

Diseases	Common Signs or Lesions	Pathologic Signs/Features
(1)	(2)	(3)
1. Rinder pest (cattle plague).	1. Erosions in the mouth. 2. Zebra markings in the rectum.	Presence of erosive stomatitis, blood stained saliva, high fever, 105^0 F, severe diarrhoea and dysentery. Marked leucopenia, and lymphopenia with karyorrhexis of the lymph nodes, rapid outbreaks, commonly affecting the young and mature cattle and death of animals may be upto 90%. Resistant animals may show subacute or chronic form of the disease.
2. Foot and Mouth disease.	1. Vesicles in the mouth. 2. Vesicles on the udder and cornet. 3. Mycoardial degenerative changes in the calves.	Ropy saliva, high fever, lameness, painful stomatitis. Morbidity of the FMD patients may rise to 100%. Rapid spread of the disease in the enzootic areas. Serological and animal transmission tests confirm its incidence.
3. Bovine malignant catarrh.	1. Erosions in the mouth 2. Skin lesions 3. Diarrhoea and dysentery.	Marked by persistent high fever. Hyperaemic buccal mucosae, severe conjunctivitis, enlarged swollen lymph nodes and shedding of the horn coverings are noticed in the affected animals which are mostly young or immature. Haematuria, vasculitis and corneoseleral opacity are marked. Presence of leucopenia and neutropenia at the early stages of the disease in the animals and leucocysts noticed at a later stage. Disease is confirmed by transmission test.

(1)	(2)	(3)
4.Salmonellosis (paratyphoid fever).	1. Leucopenia 2. Diarrhoea 3. Dysentery 4. Fibrinohaemorrhagic enteritis 5. Septicaemia and enterocolitis 6. Focal hepatic necrosis and permeation of paratyphoid nodules consisting of histiocytes and macrophages etc.	Presence of high fever, foul smell in the faeces, fibrous casts and abdominal pain are marked in this disease. Death within 24-48 hours. Cattle of all ages affected by its outbreak. Acute disease followed by subcute or chronic form.
5.Johne's disease *Mycobacterium paratuberculosis* infection.	1. Emaciation 2. Chronic diarrhoea 3. Enteritis.	Watery homogenous faeces inspite of almost normal appetite and temperature. Loss of weight is a marked sign. Folds or corrugations of the mucosae of the intestine. Older or animals of 2 years of age show low morbidity. Disease is confirmed by serological test or cultural examination of faeces. Almost normal hydration in the body.
6. Ostertagiasis.	1. Persitent diarrhoea 2. Bottle jaw (subcutaneous oedema in the mandibular space).	No presence of mucus, blood or smell in the faecal matter. Decreased appetite, Cattle (6 months to 2 years of age) are mostly affected. Presence of parasitic eggs in the faeces, several treatments with fenbendazole give good results.
7. Coccidiosis.	1. Presence of dysentery 2. Haemorrhagic cecitis and colitis.	Presence of enteritis in the young cattle. Nervous signs are noted in 20% of the patients with deaths. Presence of mild fever, oocysts are noticed in the faeces. A self limiting disease in the cattle and respond very well on treatment with amprolium and sulfona-mides.

(1)	(2)	(3)
8. Arsenic poisoning.	1. Sudden death 2. Acute abdominal pain.	Diarrhoea, muscular tremors and convulsions are noticed in the poisoning cases in cattle. Affected animals die within 4-8 hours of the appearance of symptoms. Belowing and regurgitation are also noticed in the cases of arsenical poisonings. Abomasal oedema is noticed. Feeds, faeces and organs are collected for the sake of chemical analysis.

Treatment/Management

As applicable for all viral diseases, there is no specific treatment of rinderpest in animals. However, symptomatic treatment is given and in order to check diarrhoea and superimposed infections (secondary complications), administration of sulpha drugs and antibiotics is advisable. In early cases of rinderpest, hyper-immune serum can be given for the first few days (200 to 500 cc. of the immune serum can be given s/c or i/v). To check the acute dehydration in the suffering animals, fluid therapy (5% dextrose saline) at the dose rate of 50 to 100 ml/kg body weight can be given. In convalescent animals vit. B-complex may be given for 5 to 7 days. Antipyretics can be given in the early stage to lower down the high temperature in developing rinderpest infection. There is no specific therapy for rinderpest but surveillance and annual vaccination with tissue culture or other vaccine is advisable in the endemic areas.

In order to control rinderpest in cattle, the steps followed are as under :-

(i) There should be compulsory vaccination of all susceptible animals. Legislation can be passed for compulsory vaccination.

(ii) Movements of cattle and other susceptible animals should be restricted from one place to another place. No infected animals should be allowed to enter outbreak affected states or into the states free from this disease. There should be a strict quarantine of all infected imported livestock.

(iii) The utensils should be thoroughly disinfected to prevent spread of infection to healthy stock.

Different available vaccines can be used. Freeze dried goat tissue vaccine is quite useful in the indigenous stock and immunity conferred lasts for whole life (say, about 14 years).

It is better to administer tissue culture vaccine (TCRP) to exotic and crossbred animals and immunity in them lasts two years.

The application of freeze dried GT virus (GTV) vaccine in exotic cattle is not advisable because of development of severe RP like symptoms leading to several deaths in such animals. The cattle (aged upto 6 months) should be vaccinated with suitable choice of vaccine. R.P. vaccine protects goats and sheep against Peste-des-petits-ruminants (PRP) infection.

BOVINE EPHEMERAL FEVER (Three day sickness)

A vesiculovirus (family Rhabdoviridae) has features similar to those of vesicular stomatitis and rabies and causes an acute disease in bovines. Very few sick animals die of three day sickness. Biting arthopods (flies) are believed to transmit this infection to healthy animals. The incubation period varies from 2 to 3 days and there is quick spontaneous recovery in the affected animals. The causative type species is an Ephemerovirus of the family Rhabdoviridae.

Signs

The main signs of this disease in bovines are as follows :

1. Transitory high fever, enlargement of peripheral lymph nodes, muscular shivering inappetence and dyspnoea or respiratory distress of short duration.
2. Mucoid or purulent discharge from nose, shivering and shifting lameness. Presence of running eyes and nose.
3. There may be occurrence of cessation of rumination, diarrhoea, ruminal stasis or constipation.
4. Stiffness and lameness (helpful in diagnosis) are very important signs of this disease. Swollen eyelids and drooping ears are seen in the sick animals. There is hyperfibrirogenemia, elevated creatin kinase, hypocalcaemia, leucocytosis and fall in milk production.

Pathology

The main lesions in the animals are generalized vascular engorgement, oedematous lymph nodes, hydropericardium, hydroperitoneum, oedema of the lymph nodes, rhinitis, tracheitis and pulmonary emphysema. Fasciculitis, tendovaginitis, cellulitis and focal necrosis in the muscles are also found. The blood vessels are seen surrounded by oedema and leucocytic infiltration and endothelial proliferation are also present. Blood vessels can be thrombosed and necrotic changes are found in their walls. Haemosiderosis of spleen and lymph nodes can be found. Haemorrhages in the trachea and heart muscles are seen. When the throat muscles are involved, drenching can produce inhalation pneumonia due to improper entry into trachea.

Diagnosis

It is based on the symptoms, lesions and isolation and identification of the viral agent i.e., a vesiculovirus. Sudden onset of illness (fever) and short course are characteristic signs of the disease. Other diagnostic tests are blocking ELISA, agargel immunodiffusion (AGID), serum neutralization and fluorescent anti-body test etc.

Treatment/Management

Ephemeral fever in cattle is a viral infection and no specific treatment is given for this disease. The principle of symptomatic palliative treatment is followed. Animals suffering from constipation are given purgative or an enema. Saline and diuretics may also be given. The sick animals should have an access to plenty of water. The body of animal is protected from cold and heat.

Analgesic (Novalgin) 10-20 ml (i/m B.I.D.) can be given to animals to reduce temperature and stiffness of the body. Antibiotics can also be given to check secondary infection in the lungs and other organs. Non steroidal anti-inflammatory drugs and calcium borogluconate in the cases of hypocalcaemia are beneficial in the sick animals.

Pox Diseases (Variolae)

These diseases refer to an acute viral diseases affecting man, animals and birds. The viruses of pock or pox diseases are basically similar and belong to the family Poxviridae but under natural conditions, different species of animals are attacked by the pox viruses. All strains in man, animal or birds produce cross immunity against each other but the fowl pox and swine pox viruses are quite distinct from each other. Typical lesions in the skin and mucous membranes are found in these diseases.

The lesions are as follows :

1. Macules : These are small red spots like flea bites which are formed in the roseola stage. This stage in noted after an incubation period of 3-6 days in cattle.
2. Papules : These small spots (macules)are converted into small red, swollen areas or nodules due to proliferation of cells. These lesions are found in papular stage. Raised papules have a zone of hyperaemia around their bases.

3. Vesicles : These are formed in the vesicular stage in which clear lymph accumulates in the papules to be converted into vesicles on the 7th or 8th day. In short, a vesicle is a yellow coloured blister with a pitted centre.
4. Pustules : In the pustular stage, these are formed when the lymph turns turbid and purulent. Pustules, later, undergo umbilication.
5. Desquamative stage : In this, exudate or lymph dries into a tenacious scabs and crusts. These scabs fall in about 3 weeks after the beginning of the eruptions in the bodies.

In short, the typical skin eruptions in the skin and mucous membranes of the infected animals progress by stages i.e., from macules to papules to vesicles and sometimes pustules etc. Incubation period varies from 3 to 5 days. Paschen bodies which are elementary bodies are formed inside the epithelial cells and these bodies give rise to large cytoplasmic inclusion bodies called Guarnieri bodies. Paschen granules are the viruses of vaccinia (cow pox).

The pox diseases are transmitted by direct contact between infected and healthy individuals. Droplet infection also occurs through the inhalation of the droplets contaminated with the viral particles. The viruses of the pox diseases primarily involve the epithelial tissues. The parasitized epithelial cells contain inclusion bodies which consist of smaller elementary bodies (i.e., the viruses themselves). Guarinieri bodies are found in all poxes. These are noticed in the cytoplasm in the state of encroachment upon the nuclei of the epithelial cells.

The inclusion bodies are specific for fowl pox and are called Bollinger bodies. These bodies are found in the cytoplasm of the affected epithelial cells in the skin and have an accumulation of granules within a limiting membrane. The granules involved in the formation of Bollinger bodies are called Borrel bodies.

Cow Pox

It is caused by a species called cow pox virus or variola vaccinia (vaccinia virus) of the genus Orthopox. There is a basic relationship between the viruses of small pox, cow pox and horse pox. Adults and children infected with cow pox virus attain immunity against the small pox. During milking, the virus is transmitted to the dairy man or milkers through the injured skin. Buffalo pox is closely related to cow pox.

The main lesions of cow pox are found on the teats and the udders and these heal without leaving any scar at the sites of the lesions. Such cows suffer from superimposed infection of mastitis. Cows recovered from pox get lifelong immunity. The cow pox is a self-liming disease with characteristic lesions on the teats and udder.

Sheep Pox

It is a very serious contagious damaging generalized disease in sheep. The causative virus is a sheep pox virus of the genus Capri pox virus. This disease is spread by Stomoxys *calcitrans* and has an incubation period of 12-14 days. A malignant form of sheep pox is noticed in lambs.

Signs

Fever and eruptions in the skin and mucous membrane are found in the infected sheep. Mortality varies from 5 per cent to 50 per cent.

Pathology

Lesions like nodules on skin devoid of wool and mucous membrane are found in the diseased sheep. These animals also suffer from pneumonia. Cheeks, lips and nostrils show the lesions. Haemorrhages are also seen. Later, pustules develop. There is a subcutaneous gelatinous oedema.

Goat Pox

It resembles sheep pox and is a very contagious disease. A viral dermatitis resembling goat pox occurs as an acute fatal disease in the goats in India. Pneumonia developing in goats is usually of a fatal type. The lesions in the skin develop in the hairless parts like axillae, inside the thigh, nose, chick and lips. In goat, the pox is less severe than in sheep.

Horse Pox

The virus of horse pox is similar to that of cow pox. There is a cross immunity between the viruses of horse and cow pox which transmit immunity to non-vaccinated human beings. This fact led to development of vaccines for use in the human beings.

Two kinds of horse pox are :

1. Skin Type :The flexor surfaces of joints in the lower part of the legs are attacked by the virus. Very greasy painful crusts are formed. Nodules, vesicles, pustules and scabs are noticed on the back of the pastern. Sick horses suffer from pain and lameness with formation of nodules, vesicles and pustules etc.
2. Mouth Type : The lesions develop in the buccal mucous membranes, lips, gums, tongue, nose and cheek etc. Fever is present in the sick horses. Drooling of saliva from the mouth and loss of appetite are seen in the affected horses. Death may occur in foals. Recovered horses get lifelong immunity. Healing of lesions occur in 2-4 weeks.

Buffalo Pox

It is a contagious disease of water buffaloes caused by a virus very similar to vaccinia virus. Lesions develop on the teats and udders of milking buffaloes. Necrotic nodules are formed in an experimental infection in mice.

Swine Pox

1. Swine pox caused by swine pox virus of the genus Suipoxvirus. Typical lesions appear on the ventral surface of the abdomen of the affected pigs. A solid immunity is a sequel to viral infection in swines. It can occur as a sporadic or epizootic disease in pigs.

The virus affects usually the young pigs and the lesions like eruptions of pox are found on skin, mouth and pharynx. The lesions in the lungs cause a lot of mortalities. Black pustular crusts are found on the abdomen, inside the thighs and arms. *Hamatopinus suis* transmits the virus to pigs. Lesions like vesicles and scabs heal and drop off at the end. Young pigs are very susceptible to this virus which may be transmitted by mosquitoes and flies etc. There is no cross immunity with either cow pox vaccine or with other types of swine pox.

Fowl Pox

A virus infection of birds characterized by development of wart like nodules on the skin, a watery or mucopurulent discharge from the eyes and nose and presence of diphtheritic growths in the oral cavities. The poxes in all birds are closely related. The causative agent is a fowl pox virus of the genus Avipoxvirus. Skin lesions are noticed on combs, eyelids and wattles of chickens. The lesions are marked as hyperplastic epidermis, thick layers of keratin, oedema in dermis and inclusions (called Boolinger bodies) in epithelial cells.

Treatment/Management

Since pock (pox) diseases are caused by viruses, only palliative treatment is advisable. Antiseptic cream or a stringent lotion can be used before milking. Control of the pock disease is a very difficult task. However, milking machine and udder cloth should be disinfected to avoid spread of infection to healthy animals. For washing teats,

any detergent (weak soln. of tincture iodine) can be used. Ointment like terramycin, savlon cream, Furacin etc. can be used.

Chickens from 6 weeks of age can be administered fowl pox vaccine. Birds develop immunity in about 14 days. Birds recovered from natural infection are immune.

No specific treatment for sheep and goat pox is available. Only palliative treatment is available. In order to check secondary bacterial infection, broad spectrum antibiotics or sulpha drugs are used.

For controlling sheep and goat pox, the following steps are taken :

1. No animals from infected source should be imported.
2. Affected flock of sheep and goats should be destroyed and animals in the affected areas must be quarantined.
3. Tissue culture vaccines absorbed on aluminium hydroxide can be used. Immunity develops in two weeks and persists for about 9 to 12 months. Sheep and goat pox vaccines are available for prophylaxis. Sheep pox vaccine in doses of 0.5 ml can be given s/c about an inch inside the tip of ear using 21 guage needle. Precaution should be taken to check the entry of vaccine inside bloods vessels. Immunity produced lasts two years.

Sheep pox cell culture vaccine (Indian immunological Ltd.) can even be given at the dose rate of one ml i/m at midneck or thigh region of the sheep at the age of three months usually after lambing season or during onset of breeding season.

Contagious Pustular Dermatitis (CPD) Or (Contagious Ecthyma) Of Sheep And Goat

This virus affects the sheep(usually young lambs) and goats which show eruptions around the mouth and the buccal mucosae. Such eruptions like papules or vesicles of

pox like lesions can be seen on cornea,nostrils and eyes, vulva,udder and teats of the sheep.The lesions are proliferative and lead to formation of scabs.The viruses of goat pox and sheep pox are immunologically distinct ones, i.e., lack of cross immunization but the goat pox virus is related to virus of contagious pustular dermatitis.Goat pox virus offers immunity against CPD but the virus of CPD does not immunise against goat pox.

Pathological Changes

The important signs are sore mouth, emaciation and scabs.These scabs fall and heal in about a month .The main lesions are papules, vesicles,pustules and scabs on the skin of lips, around nostrils and eyes.

The signs and lesions are helpful in making its diagnosis.

Vaccines (e.g., live strain CPD virus in the dried scabs) are used to protect the sheep against this disease.

African Horse Sickness

African horse-sickness is an infectious but not contagious febrile seasonal disease of horses which is caused by a filterable virus (an Orbivirus of the family Reoviridae). It is characterized by fever, oedema of the lungs and subcutaneous tissues in the affected animals. Culicoides are responsible for its transmission to healthy horses from diseased ones. Standing or stagnant water and warm humid conditions give a suitable environment for the multiplication of these vectors. Antigenically, different types of viruses (1-9-main immunological types) have been identified and the virus is present in the blood, other affected tissues and even in the foetus of the affected animals. Horses are highly susceptible to infection and donkeys can also be affected.

Signs

Incubation period varies from 2 to 21 days. An average one is 6 days. Fever is seen in the sick horses.

Two types of African horse sickness are as follows :

1. Dikkop horse sickness (marked by hydropericardium).
2. Dunkop horse sickness (marked by respiratory distress).

Dikkop (thick head) horse sickness

Subcutaneous oedema of head and neck, shoulders and chest, fever (reaching its height after about 2 weeks of its infection) are noticed. Such symptoms arise from abnormality of heart and circulation. The affected animals show drowsiness, arched back, restlessness, hanging of the head, loss of natural wrinklings in the mandibular space and jugular furrows.

Dunkop (thin head) Horse Sickness

This is an acute form of the disease affecting primarily the lungs of the animals. These animals show pulmonary oedema and death at a later stage. Dyspnoea, paroxymal coughing and escape of frothy fluid from the nose and struggle, severe breathing problems are noticed in the sick animals.

Pathology

In Dikkop horse sickness (thick head form), the main pathological lesions are the following :

1. Oedema of the subcutaneous and serous tissues.
2. Oedema in the head, neck and region of temporal fossa is seen and it can extend to shoulders, sternum and abdomen. Ascites is a marked lesion in this viral infection.
3. Excess of pericardial fluid (hydropericardium and subendocardial and subepicardial haemorrhages), myocardial degeneration and anasarca.
4. In Dunkop (thin head horse sickness), there is no subcutaneous oedema but the pulmonary oedema is massive and the interlobular tissue is infiltrated with fluid of gelatinous consistency. The trachea and bronchi are

filled with forthy material. Hydrothorax is also seen in such cases.

Diagnosis

Its diagnosis is based on the symptoms, leucopenia and lesions. The immunological type of the virus infecting the horse is also identified. Other tests are detection of the virus by culture and reverse transcriptase - polymerase chain reaction (RTPCR) in blood or tissues.

Oedema of head and neck (especially supraorbital fossae) along with fever, hydrothorax and pulmonary oedema are important signs of the disease. In cases, horses are found dead, the diseases like piroplasmosis and anthrax should be eliminated before diagnosing it as a cause of death. Agar gel immunodiffusion (AGID), indirect fluorescent antibody (IFA), complement fixation (CF), virus neutralization (VN) and ELISA test are advisable to confirm its diagnosis.

Treatment/Management

African horse sickness is a viral disease with fever respiratory and cardiac trouble. Symptomatic treatment in view of the lesions can be given to horses. Antibiotics can be administered to check bacterial infection. Cough electuary and expectorants can also be given. Diruetics and cardiac tonics are also given in cardiac form of African horse sickness.

Proper managemental care should be taken in stable and freeze dried polyvalent attenuated vaccine can be used in doses of 5ml by subcutaneous route. The horses are given rest for three weeks and immunity caused by this vaccine lasts a year. The horses should be protected from the bites of insects and flies by providing fly proof stables. Vaccination and reduction of exposure of horses to bites of insects are very important steps to control African horse-sickness. Foals (aged 3-4 months) are also vaccinated. An immunity following vaccination lasts a year in the vaccinated horses.

Equine Encephalomyelitis

It is a viral disease caused by the arthropod borne alpha viruses affecting the central nervous system of the horses. The virus belongs to the genus Alphavirus of the family Togaviridae. The three immunologically distinct strains of equine encephalomyelitis are :

1. Western strain or Western equine encephalitis virus.
2. Eastern strain or Eastern equine encephalitis virus.
3. Venezuelan strain or Venezuelan equine encephalitis virus.

Mosquitoes are the main vectors of the virus of equine encephalomyelitis. Human beings and other animals like pigs are also affected by these viruses. Incubation period varies from strain to strain as given below :

EEE - 1 to 3 days

WEE - 2 to 4 days

VEE- 1 to 6 days

Signs

Loss of awarness of the surroundings, tremors of shoulders, facial muscles, aimless wandering, continuous walking in circles, unresponsive to commands, mental depression and collision with different objects are some of the main signs of equine encephalomyelitis. Fever occurs at the outset of the disease and paralysis of the various groups of muscles occurs. The sick horses lie down and are unable to stand and soon die or relapse into a state of somnolence.

Pathology

The neurons are attacked by the virus of equine encephalomyelitis and are greatly damaged. Neurons show degenerative or necrotic changes. There is a dissolution and loss of tigroid substance (tigrolysis) and chromatin (chromatolysis) in the neurons. The neurons, later, undergo

fragmentation and are removed by phagocytes (neuronophagia). The leucocytes and glial cells form nodules around the damaged neurons. The grey matter around the affected neurons may become oedematous and diffusely infiltrated with lymphocytes, neutrophiles and red cells etc. Lymphocytes escaping from the nearby arterioles form a collar of densely packed cells around the blood vessels (perivascular cuffing). Small intranuclear acidophilic inclusions are noticed in neurons in western equine encephalomyelitis of horses. In the infection with the eastern strain, the grey matter is diffusely infiltrated with neutrophiles. Lesions of the equine encephalomyelitis are numerous in thalamus, hypothalamus and brain stem etc.

Diagnosis

It is based on the symptoms, lesions and isolation and identification of the virus. Complement fixing antibodies are noticed in the sera of the affected or recovered cases. Indirect immunofluorescence, virus neutralisation and complement fixation are helpful in diagnosis of equine viral encephalitis.

Treatment/Management

As equine encephalomyelitis is a viral disease with high fever and nervous symptoms, symptomatic or supportive treatment is advisable. Hyperimmune serum in doses of 500 ml can be given in the beginning of infection. Horses should be given good nutrition. Nervine tonics such as thiamine, vit. B-12, neurobion etc., should be given. Horses should also be given laxative. There should be proper care and management of the sick horses. Bedding should be turned frequently and the horses are to be protected from bites of the mosquitoes, flies, ticks and bugs etc. It is very difficult to control flies and for this purpose, fly repellants should be used. Attenuated virus vaccine can be used and it gives immunity for a year. There can also be annual vaccination within eight months of age and revaccination is done

annually. Vaccination of all horses against viral encephalomyelitis and quarantine of infected horses are important steps to protect the equine population.

Blue Tongue (Synonym. Catarrhal Fever of Sheep)

It is an infectious febrile viral disease (an Orbivirus infection of the family Reoviridae) of sheep. It is a disease of non-contagious nature and characterized by fever and catarrhal inflammatory changes in the buccal and nasal mucosae in the affected sheep. The inflamed buccal and nasal mucosae have a tendency to excoriation and ulceration. Congestion and capillary haemorrhages are noticed in the coronary tissues of the hoof and the muscle fibres show degenerative changes. Blood tissue fluids and organs of the infected animals contain the blue tongue virus (1-25). Blood is found to be the most virulent material. The sheep is the most susceptible animal but the cattle and goats may harbour the virus. The disease is not transmitted directly from one animal to another in the closest contact but natural transmission occurs by infected vectors like mosquitoes or culicoides.

Signs

It has an incubation period of less than a week. Rise of temperature (between 105^0 to 106^0F), unwillingness to feed, rolling movements of the tongue, licking of the lips and nasal discharge of mucoid or watery consistency and salivation are noticed in the affected animals. Incrustations of the dried up discharges are formed on the nostrils. Ulcerations and congestion are noticed in the nasal mucosae. The lips are swollen, tender and blood escapes readily when handled. The buccal mucous membrane is congested and cyanotic and has a tendency to excoriate to form superficial ulcers on the inside of the lips. Deep ulcers are found at the sides of the tongue. Oedema is seen in the face and ears and submaxillary regions of the body.

Lameness or stiffness is marked in the affected sheep. There is an intense congestion and haemorrhage in the coronary band or horny tissues and bands may be red or purplish. The skin in the interdigital cleft has a purplish discolouration. The losses of sheep may be upto 90 per cent. Death may occur within 6 days after the first appearance of the symptoms. The nasal or buccal discharges are foetid and diarrhoea can be seen in the sick animals. Periople may be pink and it later, turns red.

Pathology

The main pathological lesions are as follows :

(1) Blood vessels are congested or engorged with blood in general. There is a presence of arteritis with the hyperplasia of endothelium and there is also infiltration of neutrophiles and lymphocytes in the adventia. Such changes are found in the oral mucosae, brain and placenta.

(2) Hyperaemia, oedema, cyanosis and multiple haemorrhages are present in the tongue and cheeks. There is an erosion or ulceration of the epithelium. Lips and tongue are cyanotic.

(3) The skin is hyperaemic and subcutis around head and neck are oedematous. There is intensive hyperaemia of the vascular corium of the hoof especially marked at the dermal papillae and oedematous changes with neutrophilic infiltration are present. Red cells and neutrophiles accumulate in the medullary canals of the horny walls and form red streaks which continue as channels from dermal papillae.

(4) There are focal haemorrhages in the muscles with hyalinasation and loss of striations in the muscle bundles. There is coagulation, irregular swelling and fragmentation of sarcolemmal nuclei.

(5) Presence of haemorrhages in the abomasal and duode-

nal mucosae and fatty changes at periphery of the hepatic lobules are noticed.

(6) There is an extravasation of blood in the endocardium and blood tinged fluid in the pericardial sac. The spleen is congested, enlarged with haemosiderosis and neutrophilic infiltration of the red pulp. Degenerative changes in the cardiac muscles lead to collapse of the heart.

(7) Presence of gelatinous infiltration in the subcutaneous tissues and intermuscular fasciae is found.

Diagnosis

It is based on the symptoms and lesions. Fever, lameness, swollen and hyperaemic conditions of the lips, cyanosis of the tongue, ulceration in the mouth, foot lesions, stiffness and nasal discharge etc. helps in its diagnosis. The petechiae in the trapezius muscle is quite suggestive of the disease. Suspected blood collected during febrile stage in sick sheep is inoculated into healthy and immune sheep to confirm its diagnosis. Virus isolation, detection of viral nucleic acid complement fixation, AGID, ELISA and virus neutralization (VN) tests are carried out to confirm the blue tongue infection in sheep.

Treatment/Management

No specific treatment is available for blue tongue. Antibiotics are used to control secondary bacterial infections. Vitamin A (prepalin) or Vit.C (Redoxone) can be used as supportive drugs. Egg-attenuated polyvalent virus vaccine is used in South Africa and the immunity lasts in the sheep for a year. Use fly repellants in the sheep dwellings and control insect population by spraying. Avoid the risk of exposure to control the insect vectors. Timely annual vaccination in sheep is recommended and immunity develops in 10-15 days following vaccination in sheep. Import of sheep from infected countries or zones is prohibited to prevent spread of blue tongue.

Scrapie (Ovine Spongiform Encephalopathy)

It is a non-febrile prion disease of sheep which is an infection caused by a self-replicating infectious protein (PrP) with no known nucleic acid in its structure. Sheep of 2 to 3 years of age are usually affected. The ewes suffer from it at about their first pregnancy. The disease spreads to other sheep from even ewes or lambs belonging to an infected flock. The most characteristic sign of the disease is continuous scraping of the skin. Incubation period may be upto several months to several years in nature. The causative factors are prions that cause a group of diseases in man and animals known as spongiform encephalopathy.

Signs

Two varieties of scrapie are :

(i) Itchy variety.

(ii) Trotting variety.

Itchy Variety

There is an intense pruritus (itchiness) or scraping of the skin with loss of flesh and abnormalities of gait. The sheep get emaciated and show extreme debility and loss in weight. There is an increasing weakness with fixed stare or listening attitude and twitching of the ears and eye lids. Sheep are restless and affected cases may live from weeks to months. 5 to 20 per cent of the affected sheep die. Sheep show restlessness and a startled look and the pupils are dilated in the sick sheep. The skin irritation commences in the lumbar region and the affected sheep scratch the back with grinding of the teeth.

Trotting Variety

Itchiness and scratching or scraping of the skin is seen. Sheep trot with flopping ears. There is an emaciation in the body. Nervous twitchings are seen in the sheep. Grinding of

teeth and twitching of the lips and muscles of the shoulders and thighs are seen in the affected sheep. The sheep are unable to stand and show incoordination and paralysis. The case fatality rate is 100 per cent.

Pathology

There are no characteristic lesions in scrapie. But the most characteristic change is the presence of large vacuoles in the cytoplasm of neurons, diffuse astrogliosis and occasional accumulation of lymphocytes in the Virchow-Robin spaces. Such lesions can be found in the medulla oblongata, pons, mid-brain and spinal cord etc.

Diagnosis

It is based on the symptoms (scraping or restlessness) lesions and detection of scrapie prion protein in the affected sheep. Neurons with vacuoles in the medulla oblongata or pons are very helpful in the diagnosis of scrapie. Scrapie defies all sorts of treatment.

Treatment/Management

Scrapie is a viral disease and its treatment should be done symptomatically but the treatment is not so effective and advisable on account of mortality rising upto 100%. Methods for its control and eradication are to be adopted.

The sheds and barns should be disinfected. When sheep are found to be suffering from the disease, it is better to destroy them. There should be complete restriction on movement of sheep and destruction of all clinical cases is adopted. Selective breeding to evolve resistant sheep is a reasonable approach to control this disease.

Rabies (Hydrophobia)

It is an infectious viral disease which is transmitted usually by the saliva of a rabies infected dog's bite. Incubation period ranges from 2 weeks to 6 months or may be longer in

rare cases. Rabid animals show symptoms of paralysis, death and encephalomyelitis. The causative virus is a rabies virus of the genus Lyssavirus of the family Rhabdoviridae.

The virus of rabies is a neurotropic virus and the greatest concentration of the virus is found in the brain (Pasteur, 1881) and salivary glands of the infected cases. It may be either a street or a fixed virus. It is the street virus, which usually produces the natural disease in animals by bites of the rabid animals and this virus can be isolated from naturally infected warm blooded animals like cattle, dogs and sheep etc. The incubation period of the street virus can be shortened by several intracerebral passages (say, 50 passages) in rabbits or in the same species of animals. After a certain stage, the incubation period becomes constant or fixed for that species. In short, the fixed virus is a strain adapted to the secondary hosts for experimental use. Fixed virus does not produce Negri bodies in the brain as done by the street virus and does not normally produce infection in the animals. The commonest affected species of the domestic animals is the dogs. Poultry also gets infected with rabies. In short, all the warm blooded animals can be infected by rabies virus. Wolves, foxes, jackals, wild, felidae and vampire bats etc., get naturally infected with rabies virus and they can transmit this disease to man and animals by their bites. The saliva from submaxillary and parotid glands of the infected dogs may be infective due to the excreted rabies virus 3-8 days prior to appearance of any symptoms in these animals. The foetus of the infected bitch reveals virus in the brain. The milk of the milch cattle and buffaloes with history of bites of the rabid canines also contains rabies virus. There is a possibility of infection to humans drinking such milk. The rabies virus enters the body of the victims through some wounds or abrasions. The rabies virus to the extent of 53 per cent has been reported in rabid cattle. The virus has been reported to be present in the mammary glands and milk of the infected cases. It is worthwhile to note that the attempts

to communicate rabies by consumption of the contaminated milk have not been successful.

Rabies is almost invariably transmitted in healthy animals by the bites of rabid animals and this disease occurs all over the world. Contact of infected material (say milk or saliva) with the abraded skin or mucous surfaces of healthy animals or individuals can produce rabies in them. Rabies virus reaches the spinal cord and brain by travelling through the nerve fibres. There is a centrifugal spread throughout the peripheral central nervous system and the virus may reach salivary glands and the saliva (Zinke, 1804) and it becomes a potent source of rabies infection even before the onset of symptoms of rabies in animals. When one animal is suspected for rabies, it is very important to trace all human and animal contacts during the 3 to 5 days period before the onset of symptoms for appropriate action of safety and protection against rabies. The incubation period ranges from a few days to many months depending upon the distance of the bite in the body from the brain i.e., the central nervous system.

Signs

The main signs of rabies are :

(i) A sudden change in normal behaviour of the animals. There may be ferocious look of the dogs. Rarely sudden death of the infected animals with no revealation of any symptoms may also occur.

(ii) A normal taciturn dog may suddenly become friendly or normally docile dog may become short tempered. Hydrophobia (fear of water) is not a symptom to be noticed in rabid animals.

(iii) Resentment of interference or tendency to hide in the corners of houses. Foam may adhere to the lips and face.

(iv) Restlessness, hyperaesthesis to noise and light. Animal may stare or bark at nothing or at something and may bite themselves and snap at imaginary flies or objects.

(v) Apprehensive expression of the eyes tenesmus and strains as if to defaecate are seen in rabid cases.

(vi) The rabid domestic animals are not afraid of human beings, may enter into the human houses and attack them without provocations.

(vii) Cats attack humans or other animals. Cattle paw the ground or make short turns and can toss their heads. Horses are excited. Infected poultry and crows are aggressive. Dogs howl and may become unfriendly to their masters.

(viii) Paralysis of the tongue and lower jaw (dropped jaw) and profuse salivation are very common in cases of rabies in dogs. In furious form of rabies, dogs are very aggressive, bite at their manger or try to tear up an object. Such dogs, if loose, walk aimlessly for miles before collapsing in the state of exhaustion or fatigue. Dogs have decreased appetite and swallowing of food becomes very difficult.

(ix) Sudden change in behaviour of any animal (with or without aggressiveness and irritability) followed by paralysis gives one a strong suspicion of rabies in such cases.

(x) In all species, death usually takes place in 3 to 7 days after the onset of first symptoms. All suspected rabid dogs should be quarantined or kept under observation for 10 days and if alive after the end of this period, the case is considered not to be one of rabies. If once symptoms of rabies have appeared in animals, treatment is of no use and death is inevitable in them. Post vaccinal paralysis has been reported in both man and animals inoculated with antirabic vaccine.

Pathology

The main pathological changes found in the central nervous system is non-suppurative encephalomyelitis. The

meninges of the brain are usually hyperaemic. Cerebral oedema may be present. Foreign bodies like sticks, stones and pieces of metal etc., are found in the stomach of dog at autopsy table.

Microscopically, there is a diffuse encephalitis and cytoplasmic inclusion bodies in neurons known as Negri bodies are pathognomonic findings for rabies in animals. Negri (1903) demonstrated inclusions in the brain of rabid dogs. These inclusions are found in the hippocampus major in the brain of dog Presence of Negri bodies is very well expected in those rabid dogs which are allowed to die of their own accord. Perivascular cuffing and neuronophagic nodules or degenerative changes in the neurons are found throughout the brain. Glial cells proliferate and replace neurons to form nodules known as Babes's nodules. Capsule cells around the ganglion cells also proliferate in the brain in the rabies infection. Necrosis is found in the acinar epithelium of the salivary glands. Negri bodies are always intracytoplasmic and can be found in the Purkinje cells of the cerebellum in the cattle. All neurons in the brain or neurons of the gangalia may show inclusion bodies. Inclusion bodies possess a distinct limiting membrane and may be encircled by a clear halo as seen microscopically. Rabies virus is seldom observed in the blood or tissues of organs of the infected animals because of the neurotropic character of the virus.

Diagnosis

(1) The dogs suspected to be cases of rabies should be kept in cages. In case of rabies infection, the dogs usually die within a week or may linger upto 9 days.

(2) Part of the brain of suspected animal is preserved in 50% glycerine saline solution. Two rabbits or mice are inoculated intracranially with suspected material (brain). A hole is bored into the cranium with a gimlet and the inoculum is introduced through a find needle

subdurally. On about 11th day, the rabbit shows head nodding and paralysis in them is seen on the 14th day. If the inoculum is contaminated, dogs die even earlier.

(3) Impression smears are made from the hippo-campus major of the brain of the suspected cases and stained by Seller's staining method. Intracytoplasmic inclusion bodies (Negri bodies) are searched out in the stained films under the oil immersion objective of the microscope.

(4) Even sections of brain are stained by specific methods to look for Negri bodies in them. For this, part of the brain is preserved in Zenker's fixative.

(5) When the animals are destroyed in the early stages, no Negri bodies could be seen in the impression smears. These inclusions require enough of time for the formation in neurons. Negri bodies are not even formed in all cases of rabies. If there are no Negri bodies in the impression smears, it is not fair to conclude that the disease is not a case of rabies. This inference is a guiding factor in making decision about prophylactic measures.

If symptoms of rabies develop in animals, the prognosis is grave and the affected animals showing the symptoms of rabies invariably die. Rabies virus can be present in the mammary tissue or in milk, but it is difficult to experimentally transmit the infection to other animals or human beings by cunsumption of infected milk. Attempts to transmit the disease by ingestion of milk are unsuccessful. If the facility for diagnosis of rabies is not available, it is advisable to send the whole head after proper preservation between layers of ice slab with proper packing in the wooden boxes through a courier.

(6) Certain other test done to confirm rabies in animals are Dot ELISA, fluorescent antibody test and histological examination. Results of histological examination of stained section may be available within 24 hrs in labo-

ratories.

Treatment/Management

Rabies is a disease which defies treatment, in case, symptoms of rabies are evident in the animals. And as such, preventive or timely steps are essential to ward off danger of rabies infection. The sites of bite-wounds in the body of animal should be washed with dettol solution or ordinary soap solution within a few minutes i.e., just after the dog bite. Wound can be very well washed with ordinary cloth soap (i.e., 20% soft soap solution) without producing deep scratches at the bite site. The site of dog bite must be washed immediately. A solution of zephiran may be used to irrigate the site of the wounds.

Vaccination against rabies cases showing symptoms is useless, because the animal usually dies before onset of immunity. If the human beings had been bitten by rabid animals like dogs, these animals should not be destroyed and rather advised to be kept under observation in cages for 7 to 10 days. In positive case of rabies infection, animals invariable die within 7 days. In animals, alike human beings, post vaccination paralysis develops after vaccination with rabies vaccine. The rabid cases may show unsteady gait and hypersensitiveness. The symptoms may be treated with cortisones, and sedatives. Immunity does not develop effectively in the animals, which are under three months of age (and in the case of cat under six month of age).However there are vaccines which can be innoculated under three months of age.

In order to protect the pet dogs and cats, it is always advisable to keep them in chains. All unprotected street dogs and cats (without owners) should be destroyed or sterilised for reproduction, if possible. Such canines or felines pose threat to human beings under the spell of rabies. To eradicate rabies, practice of keeping animals in chains is highly

advisable and in no case, domestic animals should be allowed to come in contact with wild carnivores and bats etc. The vampire bats are well known to spread rabies to domestic animals in certain parts of the world.

In case of bites by suspected rabid animals, the post bite treatment envisages timely and promptly vaccination at an earliest in the affected animals. Application of antibiotics and bandages can be resorted to have quick recovery from bite wounds.

Inactive rabies vaccines are quite satisfactory for domestic animals. In short, it is quite safe to resort to vaccination immediately after bite of dog or animals in order to ward off the danger of rabies infection in man and animals.

A vaccination schedule of rabies veterinary vaccine (BP) is as follows:

Table 14. Vaccine and its dosage

Vaccine	Dosage	Immunity
Rabies vaccine (inactivated culture).	Dogs, cattle and sheep 1 ml s/c or i/m	lasting for a period of three years and annual revaccination recommended.

Rabies vaccine can be given at the age of three months for prophylaxis. If pups are vaccinated below three months of age, a booster dose is also given to them at three months of age. Only healthy pups or pups free of parasitic infestation are chosen for the sake of vaccination.

Post exposure therapy of dogs following bites by rabid or wild animals is as follows (Table 14, 14a):

Table 14a. Dosage schedule

Dosages	Days / Dates
1st	On "0" day i.e. day of bite
2nd	3rd day
3rd	7th day
4th	14th day
5th	28th day
6th booster	90th day

Canine Parvovirus Infection (Haemorrhagic Gasteroentitis)

A parvovirus which is related to that of feline panleukopenia has been found to cause a disease entity in several parts of world. A more severe attack of this disease appears in young pups and imported breeds of dogs. Cases of this disease have been reported in India.

Pathology

Two forms of haemorrhagic gastroentritis are as under:

(1) Intestinal Form

It occurs in dogs of all ages and is seen in more severe form in young pups. Vomiting, diarrhoea, dehydration, fever and rapid loss of condition are noticed in dogs. Leucopenia is also present. Lymphopenia and neutropenia arise from death of their percursor cells e.g., lymphoblasts etc.

The intestine shows haemorrhagic and necrotic enteritis. The mucosae are reddened and covered with blood tinged mucoid material. Microscopically, crypts are found dilated and regenerative changes are seen in the epithelium. Intranuclear inclusions can be found in the intestinal epithelium. The cardiac lesions may be seen associated with lesions of the intestinal form.

(2) Cardiac Form

It is noticed in puppies (aged 2 to 8 weeks). The lesions in the intestine can be found in this form. Dyspnoea and respiratory distress are found in the sick animals. Microscopically, the foci of necrosis in the myocardium are associated with infiltration of mononuclear cells. Intranuclear inclusions can be found in the muscle fibres. Interstitial oedema is also present in the mycocardium. Later, fibrosis in the heart can be seen in the old cases.

Diagnosis

It is based on the symptoms, isolation of the virus, lesions and detection of intranuclear inclusions in the intestinal epithelial cells. Virus neutralization test can be done to confirm its diagnosis.

Treatment/Management

There is no specific treatment for this viral disease and symptomatic and supportive treatment is usually required for this purpose.

The steps taken for treatment of this parvoviral infection are as follows :

(1) Broad spectrum antibiotics like ampicillin and chloramphenicol etc., can be given to check bacterial infection.

(2) In order to check dehydration from diarrhoea and vomiting and maintain acid base balance in the body, the fluid and electrolyte like 5% Dextrose, Ringer lactate solution etc., can be used.

(3) To control intestinal haemorrhages, ascorbic acid, Vit. K, chromostat etc., can be given to the patients.

(4) To check hyperperistaltic movement and diarrhoea, intestinal sedatives such Buscopan inj. Baralgan etc., can be used. Antiemetics like perinorm and Stemetil inj. may be used.

(5) Anti-inflammatory drugs like Dexamethazone and gamma globulin can also be given to sick animals.

(6) Supportive drugs such as B-complex inj. can be used.

For the purpose of prophylaxis, vaccines against parvovirus infection, distemper and hepatitis can be used in susceptible dogs.Some details of canine vaccine are given in table 15.

The gudelines for preparing vaccination schedule with respect to 1st, 2nd and yearly vaccinations in canines are are as follows:

Basal Immunization Against	Revaccination Against
Distemper, canine viral hepatitis, rabies and leptospirosis 1. First vaccination 7-9 weeks of age 2. Second vaccination 12-14 weeks of age.	1. Distemper, Rubarth disease (canine viral hepatitis) at intervals of one to two years 2. Rabies and leptospirosis at an interval of twelve months.

Table 15. Vaccination Schedule of Some Canine Vaccines

First vaccination (age 6 weeks)	First vaccination (age 8-9 weeks)	Second vaccination (age 12 weeks)	Annual vaccination (age 12 months and above)	Dosage	Route	Remarks
Megavac-D Megavac-P Biocan-DHPPi Nobi vac puppy DP.	Megavac-P Megavac-DHL Megavac-DHRL Megavac-6 (Distemper,* hepatitis - CAV2,* parvovirus,* Hepatitis-CAV1** *L. canidola*** *L. icterohaemor*	Megavac-DHL Megavac-DHRL Megavac-6 Biocan-L inj. ---------	Megavac-D Megavac-P(live) Megavac-P(inactivated) Megavac-DHL Megavac-DHRL Megavac-6 Biocan-DHPPi Biocan-L inj. Nobi vac puppy DP	1 ml 1 ml 1 ml 1 ml 1 ml 1 ml 1 ml 1 ml	i/m or s/c i/m or s/c i/m or s/c i/m or s/c i/m or s/c i/m or s/c s/c s/c	Pups should be dewormed at the age of 4-5 weeks before vaccination Pups should be healthy

	*rhagiaae***) NB Live attenuated viruses* Inactivated antigens** Biocan-L inj.(primovaccinated dogs at the age of 8/9 weeks) revaccinated after 14 to 28 days Nobi vac puppy DP					with normal tempera t-ure and free from parasitic inf-estation Megava c-product s of Indian immuno l-ogicals

Infectious Canine Hepatitis

(Rubarth Disease; Hepatitis contagiosa canis)

Etiology, clinical features and even actual existence of this disease was established by Rubarth in 1947. It occurs in peracute form and good dogs fall victim to this disease which is a viral infection caused by the species called Canine adenovirus I of the genus Mastadenovirus. Symptoms may be seen a few hours before death. The viral genome consists of a single molecule of double stranded DNA. A characteristic intranuclear inclusion body develops in the affected cells.

Signs

Anorexia, intense thirst, oedema of head and neck, ventral aspect of the trunk, vomiting and diarrhoea are seen in the sick animals. Moaning sounds due to abdominal pain are heard. Haemorrhages, clonic spasms of extremities and neck, paralysis of hind quarters and extreme agitation are also present. There is a rise of temperature and subnormal temperature is seen just before death. Mucous membranes are anaemic or slightly icteric. Petechiae in the mucous membrane (particularly gingival mucosa) are also present. Tonsils are reddened and swollen. There is a copious lacrimation with hyperaemic conjunctivae. The cornea is

opaque and shows cloudiness. Urine contains albumin.

Pathology

The adenovirus causing Rubarth disease has strong affinity for parenchymal and Kupffer cells of the liver and endothelial cells (i.e., in general sense). Specific intranuclear inclusions in the hepatocytes, Kupffer cells and endothelial cells (for example, endothelial cells of the glomeruli) are noticed. The hepatocytes show necrotic changes and liver cells with intranuclear inclusions show margination of the chromatin in the liver cells. Lacunose dilation of hepatic sinusoids are found. Congestion is usually noticed in the liver and spleen. The gall bladder is oedematous and thickened. Liver is engorged with increased amount of blood and adjacent liver cells are compressed due to lacunose dilation of the sinusoids. There are focal areas of necrosis in liver with disappearance of the cell and their nuclei. Intranuclear inclusions have distinct outline with basophilic tint. Hepatic lymph nodes are oedematous with petechiae. Intra-nuclear inclusions are also noticed in the endothelial cells of the spleen. Petechiae are seen around the small capillaries with intranuclear inclusions in the endothelial cells. Endothelial cells are increased in the brain and also contain intranuclear inclusions. Haemorrhages are also present in the thalamus, midbrain, pons and medulla oblongata. Loss of myelin and collection of glial cells are seen adjacent to the affected blood vessels. Proliferation of endothelial cells and increased vascular permeability are important features of this disease.

Diagnosis

It is based on the symptoms, lesions of focal necrosis and presence of intranuclear inclusions. As this disease is seen associated with leptospirosis and canine distemper, this fact is borne in mind while diagnosing cases of infectious canine hepatitis.Liver biopsy specimens are examined to confirm the presumptive clinical diagnosis.

Treatment/Management

There is no specific treatment of canine hepatitis. Steps for prophylaxis and control are adopted. Recovered dogs develop solid immunity. Symptomatic treatment can be given. It is better to vaccinate the susceptible dogs. Supportive treatment must be given in view of degenerative changes in livers of the patients. Corneal opacity should be treated with ointment containing corticosteroids. Combined distemper hepatitis vaccine is available for controlling the canine distemper and Rubarth disease. Important guide lines are given for preparing vaccination schedule of canines. (table 14). Rabies and leptospirosis are very serious zoonosis for humans. Even the parainflunza and influnza viruses are also pathogenic for humans. And as such, immunisation against these viral infections is also included in vaccination regimen. Multicomponent vaccines for providing simultaneous immunisation against four or five viral diseases like canine distemper, rabies, canine leptospirosis (its two types), and viral hepatitis (Rubarth disease) are administered to dogs

Canine Distemper (Abbreviation CD)

It is a viral disease caused by the species of the genus *Morbillivirus* which affects dogs, ferret, fox and mink etc. Most of the dogs may get infected with it before the attainment of the age of one year. The virus of canine distemper is present in the blood (in the initial febrile stage), secretions and excretions etc.

Disease is transmitted from infected animals to healthy ones through contaminated discharges, secretions, food or air droplets etc. The secondary bacterial invader in canine distemper is *Brucella bronchiseptica*- an organism usually recovered from pneumonic lesions in CD cases.

Signs

Incubation period of canine distemper is about four

days. A typical diphasic fever is noticed in canine distemper. In this disease, there is an acute fever. Whithin 96 hours, temperature drops to almost normal, then it again attains the second peak (i.e., a marked diphasic character). Other symptoms include excessive salivation, epileptiform fits, chewing and abnormal movements etc. Hyperkeratosis of the digital pads is also seen. Dehydration due to diarrhoea, coryza, purulent conjunctivitis, bronchitis, bronchopneumonia and vesicular pustular lesions may be seen in dogs infected with this virus.

Pathology

Conjunctivitis, inflammation of the respiratory tract, nervous disturbances, skin eruptions and gastroenteritis are found in the sick animals. There is a purulent or catarrhal exudate over the nasal and pharyngeal mucosae. Intranuclear and intracytoplasmic inclusion bodies are found in the cells associated with exudate on the mucosal surfaces of urinary bladder, trachea and bronchi etc. Schorr S-3 stain is used for staining the inclusions which are also eosinophilic in character. Inclusions are seen in the epithelial cells of the urinary bladder and gastric mucosae. Purulent bronchopneumonia is seen in the sick animals. Neutrophiles, mucin and tissue debris are seen in the alveoli and smaller bronchi. The exudate contain mononuclear cells. Multinucleated gaint cells containing inclusions are found free in the alveoli and also seen in the bronchial lining of alveolar septa. Vesicular and pustular dermatitis is seen in the skin of abdomen Formation of thick keratin layer on the foot (hard pad disease) is also present. The lymphocytic infiltrations are noticed in the gastric mucosa. Spleen is enlarged, congested and lymphoid follicles show necrotic changes. Myelinated tract in the cerebrum is destroyed and delimited holes of irregular sizes are formed (leading to status spongiosa). Gitter cells, astrocytes and microglia are seen around areas of destruction. There are necrotic changes in the white matter. Neurons show degenerative changes.

pyknosis, chromatolysis, gliosis and neuronophagia are also present. There is an oedema and congestion in the retina. Perivascular cuffing with lymphocytes is seen in the brain. Ganglia show degenerative changes. There is also neuritis of optic nerves with demyelination.

Diagnosis

It is based on the symptoms, lesions and detection of inclusion bodies in lining cells of the urinary bladder or bronchial mucosae.

A ferret is kept in contact with a suspected dog or is inoculated with suspected material in form of blood, spleen or mesenteric lymph nodes obtained from a dead infected dog. An immune ferret is also infected with suspected material. Immunised ferret survives whereas healthy or non-immunised one dies in positive cases of infection.

Complement fixation test using dog's spleen as an antigen is also done to diagnose canine distemper. Immunological staining techniques and viral isolation or identification are required to confirm its diagnosis.

Treatment/Management

There is no specific treatment of canine distemper. However, use of antibiotics, electrolyte solution, protein hydrolysate, anti-emetics, antipyretics, anticonvulsants and supportive treatment is advisable. For controlling canine distemper, anti-canine distemper serum (10-15 ml s/c) can be given. All steps should be taken to check secondary bacterial infections.

There should be good nursing of dogs and they must have plenty of comfort.

The vaccine can be used to prevent the occurrence of the disease in the age groups of 6 or upto 8-10 weeks . See table 14 for primary, booster and annual vaccinations of pet animals.

Swine Fever (hog cholera)

It is a highly contagious febrile disease of swines of all ages caused by a *Pestivirus* of the family *Flaviviridae*. This virus has an RNA genome. *Salmonella cholerae suis* exists as a secondary bacterial invader in this viral infection. In the infected body, all the fluids, tissues and secretions contain this virus which is principally discharged in the urine. The blood becomes infective on the first day of infection whereas urine is found infected on the 4th and 5th day. Persons, premises, litters and utensils contaminated by the infected urine spreads the disease to healthy pigs. The principal way of spread of infection to the healthy one is by direct contact between the diseased and healthy pigs. Swines of all ages are susceptible to this disease and the natural way of infection is by ingestion of contaminated food.

Signs

The main signs are as under :

(i) The pigs are depressed with high fever (106^0F) after experimental exposure to infection. The primary infection of the virus occurs in the tonsils of infected pigs. Symptoms of swine fever appear in 3 to 5 days after parenteral administration of blood or serum etc.

(ii) Presence of severe leucopenia. The white cells may become less than 4000 per cmm of blood. The swine fever virus concentrates in the organs like spleen, peripheral and visceral lymph nodes, bone marrow and Peyer's patches of small intestine of infected pigs.

(iii) Weakness, inappetence, lethargy, convulsions, grinding of teeth and difficult locomotion are seen in the sick animals. Erythematous lesions can be seen on the skin of the abdomen, axillae and inner surfaces of the legs. In peracute cases, the infected pigs die in 3 days but in the average acute case, death may occur in about one to two weeks. The recovered pigs act like carriers of the virus.

(iv) Symptoms arising from intestinal and pulmonary lesions in the infected pigs.

Pathology

The main lesions are :

(1) Petechial haemorrhages in the various parts of the body like pericardium and endocardium etc. Haemorrhages can be found in the skin and subcutaneous tissues. Hydropic degeneration and proliferation of the vascular endothelium and splenic infarcts are important lesions of swine fever.

(2) Turkey's egg markings are seen in the kidneys and arise from several varisized subcapsular haemorrhages which could be seen after removal of the renal capsule with finger tips.

(3) Congestive or inflammatory changes in mucosae of the stomach and intestines. The mucosae of the small intestine is studded with numerous haemorrhages and intestinal serosae also reveal haemorrhages.

(4) Ulcers develop in the lymphoid follicles of the small intestines. The necrotic areas which give rise to ulcers are seen as grey, yellow or black areas. In the beginning, the ulcers have more or less flat surfaces but they, later, project above the level of surrounding mucosae. Such ulcers are called button ulcers and the ulcers are covered with epithelium. Only scars can be noticed at such places. In chronic cases, the ulcers can be found on the ileocaecal valves and mucosae of the first portion of the large intestine. The epiglottis, tongue, pharynx, larynx and stomach also reveal ulcers. The infected endothelial cells show degenerative and hyaline changes owing to viral affinity for endothelial cells. The vascular changes give rise to conditions of haemorrhages, thrombosis and infarction in the infected organs like intestines and spleen. Swelling and degeneration of endothelial cells and muscle cells can be seen in the central arteriole of

the spleen. The embryos in the pregnant swines are destroyed by the virus to produce the state of still birth. Precapillaries and capillaries may be dilated or closed in the infected organs. In the central nervous system, perivascular accumulation of lymphocytes, mononuclear cells and plasma cells can be found in the Virchow Robin spaces. Haemorrhages are seen in the brain and spinal cord. Proliferated microglia can be present in the white matter of the brain. The neurons show degenerative or necrotic changes (i.e., tigrolysis of the cytoplasm and fragmentation of the nuclei). Neuronophagia and satellitosis can be seen in the grey masses of the brain. Intranuclear inclusions have been noticed in the neurons. These inclusions are found to be homogenious and acidophilic in appearance.

Diagnosis

It is based on the symptoms, lesions and isolation of the virus. Fluorescent antibody staining is done to confirm the diagnosis. Diagnosis can also be made by inoculation of infected food or tissue filtrate into both healthy susceptible pigs and pigs (controls) which have received a large dose of anticholera immune serum. The unprotected pigs die but the immunized ones show no change in their health. Antigen-capture ELISA and agar gel precipitation tests are helpful in confirming the diagnosis of the cases of swine fever.

Treatment/Management

Since it is an acute septicaemic disease, symptomatic treatment can be given. Analgesics should be used to lower down the temperature. Antibiotics are not effective because of the viral origin of the disease. To prevent secondary bacterial complications, antibiotics can, however, be used.

In order to control swine fever, hyperimmune sera and vaccines are used. 50 to 150 ml serum can be given to sick pigs. It is better to eradicate swine fever by slaughter of all in

contacts and infected pigs. In enzootic areas with the incidence of hog-cholera, suitable vaccine should be selected. If swine fever spreads in a herd, steps should be taken to prevent infection from spreading to others. The sources of infection or infected pigs should be removed and vaccination should be carried out. It is also proper to maintain hygienic precautions.

Vaccination should be followed strictly and there should be adoption of garbage cooking regulation. A carrier animal poses problems before the steps of eradication.

For immunization, killed virus vaccine or crystal violet vaccine can be given thrice after interval of a month. Immunity lasts 12 months. Serum can be given only after lapse of five days of vaccination. The dose of crystal violet vaccine is 5 ml (s/c). Serum virus vaccination is done to give immediate immunity. Piglets are passively immunized by getting antibiotics in colostrum from immune pigs and immunity lasts 4 weeks. Breakdown in vaccination programme occurs when immune serum fails to counteract the administered virus. Use of fully virulent virus can also cause out-break of the disease. It is difficult to control swine fever in baby pigs. If baby pigs (between one and seven days) after giving passive immunization with serum is given vaccine, there can be failure of vaccination programme. It is not safe to do vaccination of sows during first 3rd of pregnancy because born piglets show congenital defects. Congenital defects are very common in pigs vaccinated between 15th to 25th days of pregnancy. Freeze-dried swine fever vaccine should be better transported on ice and 1 ml of reconstituted vaccine is given s/c inside the thigh and immunity conferred in pigs lasts a year.

Pseudorabies (Aujeszky's Disease or Mad Itch)

Pseudorabies is primarily a viral disease of pigs, cattle, sheep and dogs etc, of worldwide occurance and is caused

by porcine herpesvirus-1 or pseudorabies virus, a member of family *Herpesviridae*. It occurs in the infected herds after a period of 1-2 weeks post infection and its outbreak lasts over 1-2 months. The morbidity and mortality rates reach 100% in dogs, cattle and young and piglets. Introduction of infected animals transmit the disease to healthy herds. Recovered pigs are latent cariers of the virus for whole life. Mad itch is mildly infectious for adult pigs but this disease is naturally transmitted to pigs.

Signs

The main signs are :

(i) Coughing, nasal discharge, sneezing and dyspnoea in pigs

(ii) Intense pruritus at the sites of bites in cattle and sheep.

(iii) Infected piglets show fever (upto 107^{0}F), inco-ordination, recumbency, convulsions and death.

(iv) Excitement, pruritus of the skin, circling, convulsions, fever, recumbency, paralysis and death are noticed in cattle and sheep within 48 hours. Cattle show grinding of teeth, mania and frenzied behaviour.

Lesions

These are as follows:

(i) No typical gross lesions in the dead animals.

(ii) Extensive damage to local areas of skin and extensive subcutaneous oedema in infected animals with a history of pruritus.

(iii) Reddened and abraded skin is seen in animals like cattle. Excess fluid with the pericardial sac and subendocardial haemorrhages in the carcasses.

(iv) Some splenomegaly, meningitis and excess pericardial fluid in pigs. Foci of necrosis in livers of the aborted foetuses.

(v) Extensive neuronal damage in the spinal cord and brain in all species of animals.

(vi) Perivascular cuffing and focal necrosis in the grey matter (particularly in cerebral cortex), and presence of intranuclear inclusions in degenerating neurons or astroglial cells. Necrotic lesions with inclusions in the upper respiratory tract are features of porcine pseudorabies.

Diagnosis

It is based on symptoms, lesions and serological test etc. Detection of virus in tissues and inclusion bodies in nervous tissue and respiratory tract are carried out for diagnostic confirmation. Serum- neutralization (SN) and ELISA are done to confirm preudorobies in infected animals.

Treatment/Management

There is no specific treatment for pseudorabies. The reproduction ratio is kept in view in control and eradication of pseudorabies. Control and eradication of pseudorabies depend on reproduction ratio (i.e., Ro-to average number of new infections caused by one typical infection in animals. A ratio greater than one (Ro>1) indicates the potentiality of spread of pseudorabies. A ratio less than 1 indicates the end of infection. Depopulation and repopulation of the herd is practiced when the herd infection is above 50%. But depopulation is a very extensive method. Subunit vaccines which distinguish between infected and vaccinated pigs are also used to control this disease. Infected swines are segregated from healthy cattle herds.

Some important details about viral diseases and their viral genomes are given in Table 16.

Table 16. Viruses, their genetic features, signs and pathological changes (lesions) etc.

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
A DNA viruses Double stranded (ds) DNA viruses 1. Poxviruses Pox viruses (poc= pustule) marked by a single linear molecule of double stranded (ds) DNA, replication and assembly within the cytoplasm, releasing of enveloped virions by budding and non-enveloped virions released by cell lysis and formation of intracytoplasmic inclusions (called	*Poxviridae*	*i. Orhtopox virus*	*a. Vaccinia virus*	Cow Humans Buffaloes Pigs	Cow pox or Variola vaccina (vaccinia)	**Pock Diseases** Pock diseases are called variolae. Pox is spread by contact, abrasions etc. Dogs and cats do not suffer from it. Human beings, cattle and sheep are affected by it. Cow pox lymph protects humans against small pox. The important stages of pox are as follows (1) Roseola stage - Marked by small red spots and measles (2) Papular stage - Small red areas formed in the skin due to proliferation of	1. Based on the symptoms and lesions in the affected animals. 2. Detection of inclusion bodies in the epithelial cells. 3. Eruptions on the skin in the affected animals are considered characteristic lesions.
			b. Cow pox virus	Cattle humans and elephants	Cow pox		
			c. Camel pox virus	Camels	Camel pox		
		ii. Capri pox virus	*a. Sheep pox virus*	Sheep & goat	Sheep pox		
		Do	*b. Goat* pox virus	Goat & sheep	Goat pox		
		iii. Suipox virus	*c. Swine pox virus*	Pigs	Swine pox		
		iv. Avipox virus	*Fowl pox virus*	Fowls	Fowl pox		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
Guarnieri bodies and Bollinger bodies in cattle and fowls respectively). An example of enveloped DNA virus.						epithelial cells. (3) Vesicular stage - Accumulation of fluid in papules is the main change. (4) Pustular stage - Conversion of lymph in vesicles into a purulent material in about 10 days. (5) Desquamative stage - Dessication of the exudate in the pustule, formation of scabs and final desquamation in about 21 days Fever, fall in the milk yield and pox like lesions are seen on the teats and udders of cows affected with cow pox. Lesions of pox are noticed on the	

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
						lips, muzzle and around the nostrils in calves. Cow pox virus is distinguishable from the variola and vaccinia virus. Variola causes small pox in man but vaccinia (a created laboratory virus) is used for immunising human beings.	
2.Herpesviruses A single molecule of linear double stranded (ds) DNA i.e. a monopartite genome. An example of ds DNA enveloped virus	*Herpesviridae Alpha herps-virinae (a sub-family) Gamma her pesvirinae (a sub family)*		*Suidherpes or Porcine herpes-virus 1 (SHV-1)*	Pigs and cattle	A generalized disease and abortion in pigs and pseudorabies in second-dary hosts	**Pseudorabies** Pseudorabies is a highly fatal disease in swines. Pharyngeal paralysis, salivation mania, bellowing and convulsions are noticed in cattle. Cattle die within 24 hours. Intense itching,	1. It is based on the symptoms of pruritus, lacerations, torn areas in the skin of the sick animals and intranuclear inclusions in the capillary

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
						local irritation, necrosis, hyperaemia and oedema in the skin. 2. Neuronal degeneration, proliferation of capsule and glial cells 3. Intranuclear inclusions in the nerve cells, glial cells, capillary endothelium and sarcolemmal cells, etc. Lesions are noticed on flank or hind limbs of affected cattle. Pigs do not show pruritus, become prostrate and die in 12 to 24 hours.	endothelium and nerve cells. 2. Experimental production of mad itch in the susceptible animals helps its diagnosis in the patients. It is based on signs and lesions and acidophilic cytoplasmic inclusions 0.3/0.5 micron in diameter in the affected neurons
			Bovine herpes-virus 1.	Cattle	Infectious pustular vulvo vagi-nitis and	Subcutaneous, oedema, haemorrhages on the epicardium and	

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
					abortion	oedema of the lungs seen in the animals showing symptoms of pruritus. **Malignant catarrhal fever (MCF)** Malignant catarrhal fever (malignant catarrh) is marked by fever, depression, loss of condition, leucopenia, neutropenia and weakness. Three clinical types are (1) head and eye (2) abdominal and (3) cataneous forms. Catarrhal inflammation of the mucosae of the respiratory and digestive tracts leading to congestion	
			Bovine herpes-virus 2.	Cattle & sheep	Bovine mammalitis		
			Gallid herpes viurs 1	Fowls	Infectious larynggo tracheitis of fowls		
			Galid herpes virus 2	Fowls	Marek's disease		
			(a) Alcela phine herpes virus 1- AHV-1	Cattle	AHV-1 associated with bovine malignant catarrhal fever		
			(b) Ovine herpesvirus 2 (a sheep associated MCF virus)	Sheep	Catarrhal fever(MCF)of sheep		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
						of the mucous membranes and formation of catarrhal exudate, presence of ulcers on the gums, checks and dental pad. Nodules of pea size turning into pustules and ulcerated areas. Swollen and closed eyes with catarrhal conjunctivitis.	
Adenoviruses Double stranded (ds) DNA viruses linear A single molecule of linear double stranded (ds) genome. An example of non-enveloped DNA	*Adenoviridae*	*(a)Aviadenovirus*	*(i) (Fowl adenovirus-1)*	Fowls	Egg drop syndrome	**Bovine adenoviral infection** Symptoms in bovines arising from infection of respiratory and gastrointestinal tract. Presence of oedema, haemorrhage around the joints,	It is based on the symptoms, lesions inclusions in the enterocytes and isolation and identification of the adenovirus.

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
virus.						keratoconjunctivitis, lymphangitis, colic and diarrhoea etc. are seen in the infected bovines. Both the mobidity and mortality are low in the patients. Haemorrhage, oedema congestion, and consolidation in the lungs and inclusions in the enterocytes are other changes in diseased animals. **Rubarth disease** This viral hepatitis is noticed in young canines.The main signs are	

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
						anorexia,intense thirst,vomiting diarrhoea abodominal pain high temperature with the onset of illness. The main lesions are congested and larged oedematous and thickened gall bladder,haemorrhages in the lungs and serosal surfaces, characteristic basophilic inclusion bodies in hepatocytes and endothelial cells.	It is based on signs lesions and basophilic intranucler inclusions in the hepatocytes and endothelial cells.
Papovaviruses (Papova-Pa= papilloma, Po= polyoma and Va= vacuolating) A	*Papovaviridae*	*1. Polyoma virus*	*(a) Murine polyoma virus*	Murines	Murine polyoma	The tumours look like warts or papillary growths and reveals a fibrous core with a layer of stratified	

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
single molecule of circular super coiled ds DNA. An example of non-enveloped DNA virus.Transcription to form m RNA(+)						squamous epithelium with hyper keratinisation in the outer epithelial layer in the stained sections. Spontaneous regression noticed in tumours after persistence of 4-6 months.	
		2. Papilloma virus	*(b) Papilloma viruses*		Papillomas caused in canines, ovines and bovines		
			(c) Bovine papilloma virus 1-2	Bovines	Cutaneous fibro papilloma		
			(d) Ovine papill-oma virus	Sheep	Do		
			(e) Canine papilloma virus	Dogs	Oral Papilloma		
			(f) Cotton tail	Rabbits	Cutaneous		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
			rabbit papilloma virus		papilloma		
Single stranded DNA viruses Parvoviridae A linear single stranded DNA, non-enveloped virus of negative or positive sense.	*Parvoviridae*	*Parvovirus*	1. *Canine parvovirus2* 2. *Bovine parvovirus* 3. *Porcine parvovirus*	Dogs Calves	Haemorrhagic gastroenteritis (Canine parvovirus infection) Diarrhoea Still birth, abortion and foetal death	**Canine parvovirus** infection (related to feline pan aeukopenia) Two forms of haemorrhagic gastroententis are as under. (i) Intestinal form A very rare infection in pups and dogs of all ages affected and marked by vomiting, diarrhoea, dehydration, fever and loss of condition. Intestinal mucosae congested. Nuclear inclusions are found in the epithelium of	(1) Based on the symptoms and lesions and intra nuclear inclusions in the intestinal epithelial cells.

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
				Pigs		intestinal mucosae. (ii) Cardiac form seen in puppies in the age group of 2 to 6 weeks Dyspnoea and respiratory distress are the main signs. Necrotic changes along with infiltration of mononuclear cells are noticed in the myocardium of the dead pups.	
			4. Feline panleukopenia virus	*Members of Felidae*	*Panleukopenia and enteritiss*		
B. DNA and RNA reverse transcribing viruses DNA reverse transcribing virus 1.It is marked by DNA reverse transcribing features and a single molecule of circular double stranded	*(a)Hepadnaviridae*	*1.Orthohepadna virus*	*Hepatitis B virus*	Human beings	Hepatitis		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
DNA with a region of a single stranded DNA An example of non-enveloped virus 2 Single stranded RNA of positive sense							
		2.Avihepa dna virus	*Duck*	Ducks	Duck hepatitis		
	(b) Retroviridae	*i. Alpha retrovirus*	*Avian leucosis virus*	Fowls Cattle	Avian leucosis and Rous sarcoma		
		ii. Delta retrovirus	*Bovine leucosis virus*		Bovine leukemia		
C. RNA viruses Single standed (ss) RNA virus with negative sense genome	*Paramyxovirid ae*					**Rinderpest** Cattle and buffaloes of all ages and swines are affected by RP virus. A	1. It is based on the symptoms and lesions 2. Challenge or Inoculation of immune and healthy animals

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
Paramyxoviruses						contagious highly febrile disease (108° F) with mortality upto 90%. Related to the virus causing peste des petitis ruminants. Hill cattle more susceptible to RP virus than the plains cattle. Excessive salivation with nasal discharge. A generalised disease with greyish white elevations like deposits of bran (small discrete necrotic areas on the lips, gums and dental pad of the sick animals). Erosions or ulcers seen on the buccal mucosae.	with suspected material from RP cases. ELISA and AGID agar gel immunodiffusion tests are performed to confirm the diagnosis of RP. Canine parainfluenza is marked by cough, nasal discharge, retch, rhinitis, tracheitis, bronchitis and bronchopneumonia. These changes and viral identification help the
	Paramoyxoviruses	*Morbilli virus*	*i Rinderpest virus*	Buffalo of all ages, cattle and wild ruminants.	Rinderpest		
			ii. Peste des petitis ruminants virus (related to R P and CD virus)	Goats and less often sheep	Peste des petitis ruminants (a disease alike RP)		
Single standed RNA with non-segmented negative (-) sense genome.	*Paramyxovirinae(sub family)*	*Rubula virus*	*iii. Canine distemper virus i. New castle disease virus or Avian paramyxo virus 1 i.e*	Dogs Fowls	Canine distemper Ranikhet disease		

Genetic Features	Family/ Sub ' Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
			PMV-1			Abomasal and intestinal mucosae congested and ulcerated or eroded (erosions or ulcers formed). Linear brightened stripes in the colonic and rectal mucosae called Zerba markings are the characteristic lesions of RP. Necroses or ulcerated areas are noticed in the Peyer's patches of the intestinal mucosae. High fever seen round about 3rd day of infection followed by fall in temperature, diarrhoea and faecal matter coated with mucus or blood are	diagnosis of this disease.
			u. Canine parainfluenza virus-2 SV-5	Dogs	Parainfluensis (Kennel cough syndrome)		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
						strong indications of RP infection.	
Rhabdoviruses(Rhabdo=rod) Enveloped negative (-), non segmented single stranded RNA genome	*Rhabdoviridae*	*The main Genus types are*		All warm blooded animals		**Rabies** A disease marked by lesions in the nervous system of all warm blooded animals and caused by the bites of infected rabid animals. Reservoirs of rabies are wolves, mongooses, bats (vampire) and squirills etc. The furious and dumb forms are its two kinds of rabies. Tendency to attack, mania, paralysis of the tongue and limbs, excessive salivation are important signs of	1. It is based on the symptoms and presence of Negri bodies in the neurons of hippocampus major in carnivores and in the neurons of cerebellum of bovines. 2. Seller's stain is used to stain the inclusion bodies in to neurons of infected brains. 3. A suspected animal of rabies dies within a week in positive
		1.Lyssa virus	*Rabies virus*	e.g. Cattle and horses etc	Rabies		
		2.Vesiculovirus	*Vesiculostomatitis virus*	Horse, cattle and swines	Vesiculo stomatitis		
		3.Ephemerovirus	*Bovine ephemeral fever virus*	Cattle	Bovine ephemeral fever (an Arthopod borne enzootic disease in tropical countries.		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
						furious rabies. Some 50 passages of the rabies virus through rabbits turns it into a fixed virus capable to produce symptoms of rabies and paralysis as early as 30 days in the rabbits consequent to inoculation of such fixed virus. Street virus is noticed in the wild carnivores and street dogs etc. Rabies virus is present in the milk, fetus and many organs of the infected cases. The dumb form of rabies is marked by paralysis of the muscles of head and neck and mandible causing inability to	cases, when it is kept in cages Inclusion bodies are usually noticed in such cases when sufficient time is given for formation of inclusions. Immunofluorescent antibody testing on the brain material to confirm its diagnosis

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
						swallow water or chew food given to the patients. Broken teeth, foreign bodies in the stomach, encephalitis, proliferation of capsule cells around the ganglion or glial cells and inclusion bodies (called Negri bodies) in the neurons are pathognomonic lesions of rabies infection in animals.	
Double stranded (ds) non-enveloped RNA viruses Reoviruses Marked by segmented genome, linear double stranded	*1. Reoviridae*	*Orbivirus*	*(a) Blue tongue virus (BTV) 1-25 serotypes of BTV recorded)*	Sheep, cattle and deer		**Bluetongue** It is a highly febrile viral disease of sheep transmitted by Culicoides. Inflammatory changes are noticed in the	1. It is based on symptoms and lesions 2. Isolation and identification of Blue tongue virus (BTV) 3.
			(b) African horse sickness	Horse, donkey mules	African horse sickness (an		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
RNA Birnaviridae a. Non-enevloped bisegmented ds RNA virus Single stranded (ss) RNA enveloped viruses Coronaviruses Marked by a single molecule of positive (+) sense RNA genome. An enveloped virus Togaviruses (Toga=cloac) Linear single stranded (ss) RNA (+) sense genome marked by direct transcription.			*virus (AHSV)*	and zebras	Arthopod borne infectious disease)	mucous membrane of lips, tongue, cheeks and gums i.e. occurrence of chelitis, stomatitis, rhinitis and enteritis etc. Swollen oedematous and cyanotic tongue, sloughing of tissues noticed in the oral mucosae. Congested patches in the mucosae of stomach and intestine and inflammatory changes in the coronary band of the foot. **African horse** sickness It is a febrile (105° F) viral disease of equines transmitted by culicoides or biting	ELISA and Agar gel immunodiffusion test (AGID) It is based on the signs and lesions and isolation of immunilogically distinguished viral types.
			(c) Rota virus	Almost all animals	Enteritis		
		Orthoreo virus	*Avian reoviruses-1-11*	Chickens, turkeys and geese	Viral arthritis of fowls,CRD and myocarditis		
	2.Birnaviridae	*Avibirna virus*	*Infectious bursal disease virus*	Fowls	Infectious bursal disease		
	1.Coronaviridae	*Coronavirus*	*Avian infectious bronchitis virus (at least eight serotypes)*	Fowls	Avian infectious bronchitis virus		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
		Alphavirus	*Canine coronavirus*	Dogs	Enteritis (a)	midges etc. Mortality upto 90%. Dikkop and Dunkop (pulmonary) are the two forms of african horse sickness. Dikkop (i.e. thick or cardiac form) is marked by oedema of the head, neck and face, hydropericardium and degenerative changes in the myocardium In Dunkop (pulmonary form), hydrothorax, oedema of lungs, dyspnoea and discharges from the nose are the main changes.	
		Alphavirus	*Canine coronavirus*	Dogs	Enteritis (a)		
	2.*Togaviridae*		*(a) Eastern equine encephalo myelitis virus*	Horse	Eastern equine encephalo myelitis		
			(b) Western equine encephalomyeli tis virus		(b) Western equine encephalomyel itis		
			(c) Venezuelan equine encephalomyeli tis virus		(c) Venezuelan equine encephalomyel itis		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
Astroviridae A single molecule of linear single stranded RNA virus having positive (+) sense genome	*Astroviridae*	*Astrovirus*		Several animal species	Self limiting gastro enteritis		
Picornaviruses Direct transcription A single molecule of linear single stranded (ss) RNA, positive sense and a non-enveloped virus	*Picornaviridae*	*1.Enterovirus*	*1.Swine vesicular disease virus*	Pigs	Swine vesicular disease	**Foot and mouth disease** It is a febrile viral disease of many cloven footed animals. Excessive salivation, anorexia due to mouth sore, lameness. Severe depression and sudden death in calves arising from mycardial degenerative changes The characteristic lesion is an aphthous or vesicle formation.	(1) It is Based on the symptoms and lesions (2) Injection of the suscepted material in to guinea pigs (3) Performance of the compliment fixation test.
			2.Avian entroviruses	Chicken s Ducks and turkeys	Avian encephalomyel itis		
			3.Poliovirus 1,II and III	Man	Hepatatis Poliomyelitis		
		Aphthovirus	*Foot and mouth disease virus The viral types are O, A, C, SAT-1, SAT-2,*	Cattle, sheep, goats pigs and wild rumints	Foot- and - mouth disease		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
Calciviruses Direct transcription A single molecule of linear single stranded (ss) RNA. Non enveloped Flaviviruses Direct transcription A single molecule of linear single stranded RNA, positive sense genome			*SAT-3 and Asia-1*			A vesicle is a circumscribed cavity containing fluid. A Aphthae are noticed on lips, dorsum of tongue, palate and in the skin near the coronary band, teat, vulva and udder. Ballooning degenerative changes in epithelial cells in the form of pyknosis, fragmentation leading to appearance of vesicles (aphthae) on the lips and tongue etc. Degenerative changes in the sensitive laminae of the foot causes shedding of the hoof in cattle or claws in	
		Hepato virus	*Hepatatis A virus*	Man	Hepatatis		
	Calcviridae	*Calcivirus*		Pigs	Enteritis		
			Vesicular exanthema virus	Pigs	Vesicular lesions in epithelial cells of lips, tongue, nostrils and snouts. Mucosal disease		
	Flaviviridae	*i.Pestivirus*	*Bovine viral diarrhea virus*	Cattle and calves			
			Swine fever/ Hog cholera virus	Pig	Hog cholera (Swine fever)		
		ii.Flavivirus	*Japanese encephalitis*	Swine man and birds	Abortion and encephalitis		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
			virus			sheep and goat.	
			Mosquitoes act as its vectors				
Bronaviridae Single stranded RNA with negative (-) sense genome.	*Bornaviridae*	*Bornavirus*	*Borna disease virus*	Horses	Borna disease		
Orthomyxovir idae Single stranded negative (-) sense RNA, segmented genome.	*Orthomy xoviridae*		*a.Influenza virus A*	Man, birds e.g. fowls and horses	Human influenza caused by influenza A and B		
			(b) Influenza virus B	Influenza virus- a patho gen of only man			
			(c) Influenza	Horses,	Bird flu (called		

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
			virus C	swines and fowls.	fowl plague)		
Bunyaviruses Single stranded negative (-) gense RNA segmented genome	*Bunyaviridae*	*Bunya virus*	*Bunyawera virus*	Sheep and cattle	Arthrogryposis and hydranencephaly		
		Nairovirus	*Nairobi sheep disease virus*	Sheep and goats	Nairobi sheep disease	Marked by acute haemorrhagic gastroentritis	
		Phlebovirus	*Rift valley fever virus*	Sheep cattle and buffaloes	Rift valley fever		
NB Prions (self replicating infectious or rogue proteins with no nucleic acid) Aminoacid sequences of many prions or variants are known but information about				Cattle Sheep and occasionally goat	(i) Bovine spongy form encaphalopathy (ii) Scrapie	a.Marked by neuronal degeneration b.Spongiform degeneration in the grey matter of brain, astrological proliferation and hypertrophy are the marked changes in bovine spongiform	

Genetic Features	Family/ Sub Family	Genus	Species/ Virus Types	Animal Affected Humans	Diseases caused	Signs and Pathological Changes etc.	Diagnosis
1	2	3	4	5	6	7	8
their families, genetics and species is lacking.						encphlopathy. a. Neuronal vacuolation and degeneration with astrocytic hypertrophy, hyperplasia, neuronophagia and perivascular cuffing are the marked lesions in the cases of scrapie.	

Note : Influenza viruses are classified in the light of the virul hanagglutinin and nuraminidase i.e. H and N genes.

Viral Types of Influenza Viruses	Animls Affected	Diseases
i. H7 N7 and H3 N8	Horse	Respiratory disease
ii. H1 N1and H3 N2	Pigs	Do
iii. H3N2 and H3 N1	Man	Do
iv. H5 N1 and H7 N1	Fowls	Bird flu

Canine parainfluenza virus and canine adenovirus 2 (CAV-2) are part of a complex pathogenesis leading to infectious laryngotracheitis in dogs. The CAV-2 vaccine protects dogs against ILT and also against CAV-1 infection (Viral hepatitis) in dogs due to cross immunity.

Fig. 1. Widening of the interductal and interalveolar interstitium with marked fibrosis and infiltration of round cells in chronic mastitis in buffalo (H & E x 100.)

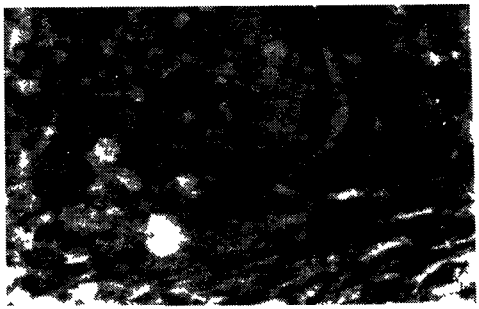

Fig. 2. Epithelioid granulation tissue in tuberculous mammary gland of a buffalo. Note the epithelioid cells, Langhan's giant cells lyphocytes and fibroblasts (H & E x 400).

Fig.3. Actionomycos is (pharynx, cattle) A colony of *Actinomyces bovis* in a tissue surrounded by epithelioid cells and neutrophiles (H & E x 400)

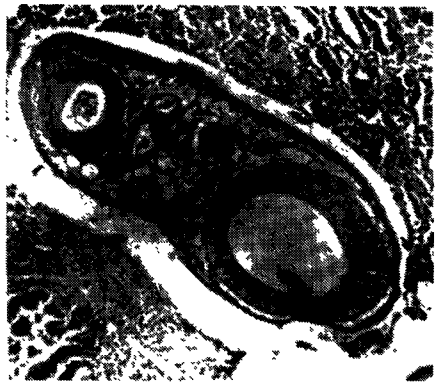

Fig. 4. Section of duodenum of a cow showing a migrating immature fluek in the submecosa and the Empty tract left behind the parasite H & E x 100

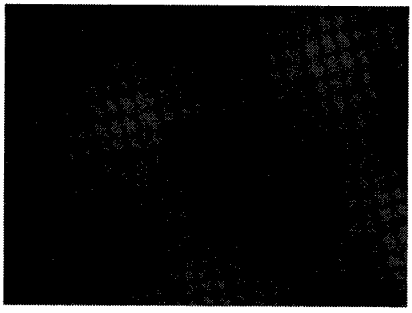

Fig. 5. Colloid goiter, foal, Note the deposits of the colloid distending the thyroid follicles. H & E x 400

Fig. 6. Section of liver of a Tharparker cow, showing a fluke in the severely thickned bite duct. Note the proliferated connective tissue cells in the wall of the bil duct. H&E; Stain;x100

Pathologic effects of viral infections

Both reversible and irreversible changes are produced by different viruses in bodies of the hosts. The most nonenveloped viruses grow either in the nucleus or in the cytoplasm of the infected cells. Viruses are released from the cells by cytolysis. Retroviruses and togaviruses derive cellular membranes and proteins from the cells before their release as enveloped virions. Retroviruses are noncytolytic because they do a little damage to the infected cells. Examples of cytolytic viruses are paramyxoviruses, rhabdoviruses and togaviruses which destroy the infected cells. Pox viruses fuse with the cellular membranes of the infected cells and are, then, released due to rupture of such cells. Herpesviruses, picornaviruses, parvoviruses, adenoviruses and flaviviruses also cause cell lysis or necrotic changes in the infected cells. No visible change is seen in latent, persistent and chronic infections of the cells by viruses. Infectious carriers arise from chronic cases of viral infections of animals as seen in the cases of FMD and canine distemper. Slow viruses e.g., HIV or Maedi virus produce persistent infection in the host body. Inclusion bodies produce cytopathic effects in the infected cells by destroying cytoplasm and nuclei of the infected cells. Neurotropic viruses produce necrotic changes in the neurons of the brain. FMD virus destroys epithelial cells of the skin or mucosae by producing balloon degeneration and vesicle formation which lead to appearance of erosions or ulcers etc. Birds affected with leucosis are emaciated but enlarged neoplastic growths are seen in the internal organs like liver, heart and lungs etc.

In short, the main effects are as follows :

1. Cell lysis (cytolysis) and degenerative changes in th nuclei of the cells infected with viruses. For examples, FMD virus causes directly hyaline degeneration and necrosis of myocardial fibres in young calves.
2. Destruction of cells by toxic capsid proteins or some other

viral proteins. Viruses do not produce toxins like bacteria.

3. Viruses shutdown the host DNA and RNA synthesis in the cells because of the takeover of the cellular ribosomes. Such a situation is incompatible with survival of cells in the host body.
4. Latent, persistent and chronic infections of cells by viruses cause deterioration or degeneration in bodies of the hosts.
5. Cell mediated immune response causes cytolysis in the infected cells of the hosts. Sensitized lymphocytes (T lymphocytes) and natural killer (NK) cells destroy the cells infected with viruses.
6. Viral inclusions lead to destruction of cytoplasm or nuclei of the cells.
7. Certain viruses (e.g., retroviruses) produce irreversible neoplastic transformation of the normal cells. This leads to formation of tumours in the bodies of the hosts which are useless structures.
8. Certain viruses like herpes viruses and paramyxoviruses produce syncytial cells which are multinucleated cellular structures. Such structures only help diagnosis of infections of herpesvirus or paramyxovirus in the animals.

Chapter 4
Protozoan Diseases

Babesiosis (Piroplasmosis)

It is a febrile protozoan disease caused by intraerthrocytic organisms of the genus *Babesia spp.* or piroplasms capable to multiply into two or four by means of binary fusion inside the red blood cells of the hosts like cattle and buffaloes etc. Pear shaped structures are formed by the causative prototozoa inside the erythrocytes. These forms are pathogenic asexual structures and are transmitted by blood sucking ticks. A piroplasm or a typical babesia consists of three parts :

(i) A thin envelope containing protoplasm.

(ii) A dot of chromatin.

(iii) A vacuole.

The newly divided forms of piroplasms are held together be means of stalk and the affected red cells, later, disintegrate releasing haemoglobin into circulation. Excessive amount of haemoglobin so formed passes into urine through glomeruli to give rise to a condition of haemolytic jaundice and haemoglobinuria. These protozoa attack healthy red cells and cause them to disintegrate. In *B. canis* infection, the division or subdivisions of parasites give rise to more than two piroplasms inside the red cells with the result of crowding inside their bodies. Such red cells, later, disintegrate or get haemolysed to produce anaemia, haemoglobinuria and jaundice etc. The reticuloendothelial cells in the liver, spleen and bone narrow dispose of the excess of released haemoglobin from lysed red cells. But in the case of massive destruction of red cells, excessive haemoglobin formed in the

blood creates the state of haemoglobinaemia or bilirubinaemia. The hosts, then, suffer from haemolytic jaundice or icterus along with haemoglobinuria. The RE cells in the liver etc., fail to dispose of the massively formed haemobilirubin in the blood and give rise to symptoms of haematogenous jaundice. Haemolytic anaemia is an important feature of piroplasmosis. The Babesia organisms are found inside the red cells during the appearance of acute symptoms. The Leishman's or Giemsa stain is quite suitable to stain these organisms in the red cells. In the stained piroplasms, there is blue cytoplasm and red chromatin mass attached with strings of chromatin granules and an arrangement in couples of pear shaped structures which may be seen in the infected cells. This disease is called red water in cattle.

Transmission

Ticks transmit this disease from one vertebrate host (cattle) to another one. The hard ticks of the family Ixodidae are important vectors of this disease and in India, *Hyalomma anatolicum* is the common tick to transmit the infection to healthy stock of cattle. Ticks infect healthy animals during different stages in their life cycle (i.e., transmission of diseases from stage to stage). The ticks have the following four stages in their life :

1. Eggs : These are very small equal to the size of a pin's head and can be found in crevices or holes on the ground or walls of houses.
2. Larvae : These have six legs and bury their mouth into skin of the host. They get attached to these animals during the period of feeding, then drop off and moult into nymphs.
3. Nymphs : These forms possess 8 legs and are actually immature forms of ticks with no appearance of sexual organs. These may be oval in shape and attach to the body of host for feeding. Later, they drop off on the

ground to moult into adult ticks.

4. Adult ticks : They are found as males or females. While attached to the verebrate host, the female ticks may be fertilized by male ticks and when engorged with blood, they drop off to the lay eggs in crevices or cracks on the ground. Two to three thousand eggs can be laid by a single female tick. An *Ixodex ricinus* is known to shrivel during the process of egg laying and, later, it dies or disintegrates.

Ticks may be one-host tick, two-host-tick or even three-host-tick. *Ixodex ricinus* is a three host tick i.e., each stage (larval, nymphal or adult) is passed on different hosts. In a two-host ticks, the adult stage is passed on a second host. This is noticed in *Rhipicephalus evertsi.* In one-host tick, the three stages (namely, larval, nymphal or adult ones) are passed on a single host and all moultings (i.e., from larva to nymph to adult) occur on the same host. While attached to the susceptible host, infected ticks spread the infection to healthy animals through their bites but transmission of the disease may be hereditary i.e., adult infected ticks may carry on the infection to the next stage of the life cycle i.e. their infected eggs with protozoa may carry the infection to larval stage and so on and so forth. Infection, thus, passes through the eggs of the ticks to other stages. In short, transmission of babesiosis may be hereditary or may occur from stage to stage.It means that infection taken in by larvae of ticks may be transmitted by the emerging nymphs or infection taken in by the growing nymphs will be transmitted by the adult. It is worthwhile to note that in the 2nd generation, only the adult ticks may be infective or the infectivity may be shown by nymphs and adults or by all the 3 stages like larvae, nymphs and adults of ticks. The important types of piroplasms are as follows :

Table 17. Piroplasms, their hosts and vectors

Species	Hosts	Vectors
Babesis bigemina	Cattle	*Boophilus microplus* in India
B. bovis	Cattle	*Isodex ricinus* in Europe
B. canis	Dog	*Rhipicephalus sanguineus* in India
B. gibsoni	Dog	*Haemaphysalis bispinosa* in India
B. caballi	Horse	*Dermacentor* and *Hyolomma* etc.
B equi	Horse	*Rhipicephalus evrtsi*
B. motasi and B. ovis	Sheep	*Haemaphysalis* and *Dermacentor* spp.
B. felis	Cat (in India)	----

B. major, B. divergens, B.bovata are reported in cattle. *B. trautmanni and, B. perroncitos* are piroplasms of pigs.

Signs

The important signs of babesiosis are as follows :

(i) Fever.

(ii) Anaemia marked by thin watery blood.

(iii) Jaundice or icterus.

(iv) Haemoglobinuria (red water).

(v) Ascites or debility during later stages.

This febrile disease is called red water in cattle, biliary fever in horse and tick fever in dogs.

The excreted urine is red or black in colour. Heart beat is increased in cattle. In hyperacute cases, the cerebrum is attacked and involved with appearance of nervous signs. The incubation period of the babesiosis varies from 5 to 10 days and the infected animals may die within 2 to 3 days. The temperature may become subnormal and the animal may collapse.

In chronic cases, there is a marked anaemia, emaciation, debility and constipation and diarrhoea etc. The disease in chronic form may last over several weeks and the animals may eventually die.

Pathology

The red cell count drops to 25 per cent of the normal and haemoglobin percentage is less than 50 per cent of the normal. Anisocytes (red cells of different sizes) are seen in the stained films of the blood of the affected animals. The spleen is enlarged with red cells (four to five times its normal size) and may even rupture. The spleen is softer than normal and its Malpighian bodies and trabeculae present a blurred appearance. The splenic pulp is dark and the liver is yellow brown in colour as revealed by the incised surfaces and it is enlarged with parenchymatous or fatty degeneration. The gall bladder is distended with bile owing to hepatic disturbances. The renal tubules are blocked and tubular degeneration is noticed. There is presence of catarrhal gastroentritis. The blood is thin, pale and watery Haemorrhages are seen on the heart, seruous membranes and mucosae of the stomach and intestines. There is presence of red coloured urine in the urinary bladder. Oedematous changes are present in the lungs. The subcutaneous, subserous and intramuscular connective tissue is oedematous and yellow in colour. The capillaries in the brain and optic choroids contain several *Babesia* organisms. Signs and lesions of diseases in bovines marked by red urine are given in the table 18.

Table 18. Signs and lesions in bovine diseases marked by red urine

Diseases	Common Signs	Pathological Signs / Main Features
(1)	(2)	(3)
1. Babesiosis (an infection caused by the organisms of the genus *Babesia.*	Red Urine	High fever, pallor, thin and watery blood and severe terminal icterus. Urine is red in colour with haemolobinuria. *Babesia spp* present in the red cells. Bodies of animals like calves infected with ticks. Morbidity and mortality upto 90%. Incubation period varies from 2 to 3 weeks. Enlarged liver, spleen enlarged 4 times the normal and oedematous lungs and red urine in the urinary bladder.
2. Theileriasis (an infection caused by the parasites of the genus *Theileria* spp.	Red Urine	Anorexia, enlarged lymph nodes, fever, haemoglobinuria, theileria in the red cells. Animal's bodies infested with disease transmitting ticks neveal oedematous lymph nodes, pulmonary oedema and enlarged liver. Patients suffer or die of asphyxia.
3. Bacillary haemoglobinuria (*Clostridium haemolyticum* infection).	Red urine	Fever upto 104°F, haemolytic anaemia, toxaemia, diarrhoea, shallow rapid respiration, abdominal and diaphragmatic pain. Acute onset of the disease with often death in the animals. Large areas of necrosis in liver, hamoglobinuria, leucocytosis and Gram positive organisms in the sections of necrosed tissues in livers are some important findings.
4. Leptospirosis (*Leptospira interrogans pomona* infection).	Red urine	High mortality (upto 50%) in calves. Urine red, septicaemia, toxaemia and haemolytic condition. Fever varies from 104°F to 106°F. Presence of haemoglobinuria. Pallor, icterus and haemorrhages in the mucosae, Leptospiruria (i.e., *Leptospira* spp. in urine) initially for 3 days in the patients. Leptospira antibodies in the guinea pigs given intraperitoneal injection of *Leptospira* spp.
5. Post parturient haemoglobinuria.	Red urine	Occurrence post calving 2-4 weeks. Tremor, pallor, tachycardia, audible heart sounds. Weakness, tremor. Acute onset with 50% mortality. Death from anaemia. Deep brown to frothy urine. Cells present in the urine on standing. Presence of severe haemolytic anaemia.

6. Chronic copper poisoning.	Red urine	Weakness, pallor, icterus, death in 24-48 hours. Sudden onset of the disease. Presence of haemoglobinuria. 2000 ppm copper in the dry matter, icterus, yellow coloured friable liver, hepatic degenerative and necrotic change (acute toxic hepatitis).
7. Enzootic haematuria (a desease marked by persistent haematuria in cattle and water baffalo due to braken fern poisoning).	Red urine	Persistent intermittent haematuria, acute or chronic anaemia (haemorrhagic type). Death from anaemia. Animals older than 1 year are affected. Hyperplastic inflammatory changes Hyperaemia and haemorrhages may be noticed in the urinary bladder or other parts of urinary tract.

Diagnosis

It is based on the symptoms, parasites in the blood or smears of spleen, vector present in environment and lesions in the body. In enzootic areas, the blood is examined to detect protozoa during the height of temperature. Thin films of peripheral blood are good for such purposes. If there is a history of high temperature with anaemia in cattle in enzootic areas, it is better to treat them for babesiosis. The protozoa can be demonstrated in the blood of the susceptible or splenectomised animals after inoculating them with infected blood from chronic cases of babesiosis or healthy carriers. Haemagglutination or complement fixation test is done to diagnose it. Fluorescent antibody and agglutination tests are also helpful to confirm its diagnosis.

Treatment/Management

In treating cases of babesiosis, two principles are to be kept in mind, that is,

(i) Babesiosis is an acute disease in the initial stages and the animals may die due to delay in starting the treatment on account of anaemia and

(ii) Development of pre-immunity. There should be avoidance of complete sterilization of the patients. Pre-im-

munity is dependent on the fact of existence of protozoa (similar to pre-immunity as seen in B.C.C. vaccination) in the body of patients.

The drugs to be tried in cases of babesiosis are the following :-

(i) Berenil (Diminazene acturate) is given at the rate of 0.8 to 1.6 gm per 100 kg of body weight by deep intramuscular route. There should be administration of antihistamnic drugs prior to administration of Berenil (e.g., Avil, Zeet etc.).

(ii) One per cent soln. of trypan blue can also be given to horses and bovines. Administration of diminazene and imidocarb in infected animals gives good results.

Blood transfusion and haematinics can also be given to patients. Supportive therapy in form of liver extract (Belamyl, Livogen inj.) are given for quick recovery in animals. Inferon can be given i/m twice weekly to sick animals. Liv-52 can also be given to animals. (see tables 37 and 39 for different drugs or patent preparations used in treating animals

Theileriasis

It is a tick borne protozoan disease of animals (mammals) caused by the parasites (*Theileria* spp.) of the family *Theileridae* and transmitted from infected animals to healthy animals by the bites of ticks (for example, ticks of the family *Ixodidae*). Ruminants suffer drom these protozoal parasites of the genus *Theileria.* Damage to hosts is mainly caused by schizonts arising from the process of schizogony in the affected cells i.e., the causative agents.

Theileria spp.

These organisms multiply in the cells of the lymphocytic series of the mammalian hosts by a process called schizogony. This process leads to formation of schizonts known as Koch's blue bodies in such cells. The sexual stage of the life cycle i.e.,

of gametocytes in the forms of males and females is found in the red cells of the hosts. These male and female forms show sexual life cycle in the gut of the infected ticks after a blood meal on the animals.

Important species of theleria are given below :

Table 19. Theileria their hosts and diseases caused

Species	Hosts	Disease	Main Vectors
T. parva	Bovines (cattle)	East coast fever	*Rhipicephalus appendiculatus*
T. bovis	- do -	Corridor disease	
T. annulata	do & water buffalo	Tropical theileriasis	*Hyalomma anatolicum*
T.mutans	- do -	Benign bovine theileriasis	*Hyalomma* spp and *Haemaphysalis bispinosa*
T. hirci	Sheep and goats	Malignant ovine or Caprine theileriasis	*Hyalomma* spp.
T. ovis	- do -	Benign ovine or caprine theileriasis	*Rhipicephalus bursa*

Schizonts or agamonts or Koch's blue bodies are found either in the cytoplasm of the lymphoid cells or as free bodies. These bodies are masses of blue cytoplasm containing 1 to 80 small pale or pink dots in the stained films of the blood or tissue smears with Giemsa stain and their diameters vary from 1 to 15 μ in measurements, (an average of 8μ). Presence of other forms like gamonts are less frequent. Gamonts appear as small dense, chromatin dots and are commonly noticed in reticular cells. When these cells rupture in smears, individual theilerial forms of organisms are scattered around the cell and appear as comma shaped structures with small round nuclei. In the red cells, they appear as dots, commas, short rods, round or oval forms.

T.annulata schizonts occur in the lymphoid tissues and are more common in the liver, spleen and peripheral lymph nodes. Theilerial organisms in red cells are found in large number in severe infections. The organisms are round, oval, rod shaped or anaplasmoid in structures with predominance of round forms. These forms measure 1 to 2 μ and infect bovines in India.

Transmission

Cattle and buffaloes are important mammalian hosts. The Theileria organisms develop in the ticks of the genus *Hyalomma. Hyalomma anatolicum* is the variety common in Indian cattle and buffaloes. Infection occurs in larval or nymphal stages of 2 host or 3 host ticks etc., (but not the eggs) and is transmitted to the adult stage. That is why transovarian transmission does not occur in theileriasis. In other words, infection does not pass through the egg to the larval stage as seen in babesiosis. Infected ticks bite the healthy animals and transmit the disease to them. Engorged larvae or nymphs hide themselves in crevices of walls and floors and moult there in the spring or summer. When these ticks bite healthy livestock, seasonal incidence of theileriasis occurs in summer or spring.

Signs

Incubation period ranges from 9 to 25 days (an average of 15 days) after a tick bite. The disease occurs in the acute form and the temperature of the hosts rises to 104^0 to 107^0F. During febrile stage, Kock's bodies are found in the smears prepared from the lymph nodes (e.g., prescapular nymph nodes). The affected animals show loss of appetite, swelling or enlargement of superficial lymph nodes, and nasal and ocular discharges. Animals are anaemic and emaciated with loss of condition and mucous membranes are pale. Constipation is seen in the early febrile peri d but diarrhoea and blood stained faeces are noticed commonly in the later stages. In short, fever, enlarged superficial lymph nodes,

dyspnoea, wasting and terminal diarrhoea are the main signs of theileriasis.

The course of the disease varies in animals from 8 to 15 days. Death occurs in 3 or 4 days in the peracute cases. Indigenous stock is resistant to theilerial infection. Respiratory distress or asphyxia may arise from pulmonary oedema. Heavy mortality is noticed in the exotic or cross bred cattle. Icterus, haemoglobinuria and severe anaemia as seen in the cases of red water are not the symptoms of theileriasis.

Pathology

The mucous and serious membranes are pale and show numerous petechiae. The liver is enlarged and friable and may be pale brown or yellow in colour. In the spleen, the Malpighian corpuscles are conspicuous with no enlargement in its size. The lymph nodes are enlarged, oedematous and often congested. The kidneys are pale. A very common lesion is haemorrhgic ulceration of the abomasums of the infected bovines. Ulcers can be seen in the tonsils of sick animals. Oedema and congestion are present in the lungs. There is presence of subepi-and subendocordial haemorrhages or petechiae in the heart. Hydrothorax and hydropericardium are other lesions in the dead animals.

Diagnosis

It is based on the following :

(1) Symptoms like fever, anaemia, emaciation, dyspnoea, wasting and terminal diarrhoea and lesions of lymphoproliferative character in the dead animals.

(2) Detection of the parasites (*Theileria spp.*) in the smears made from the peripheral lymph nodes, spleen and liver etc., by the process of puncture. During life, the smears are prepared from enlarged lymph nodes particularly (prescapular lymph nodes) to detect the theilerial organisms in the red cells and schizonts in the lymphoblasts of lymphoid organs.

Treatment/Management

There is no suitable or reliable drug for absolute treatment of theileriasis. However, drugs like Berenil, steclin and supportive medicine can be given to sick animals. Berenil is to be given @ 0.8 to 1.6 gm/100 kg. body weight I/M to cattle. Recently, a drug called Butalex has been found to be a specific drug of choice for treating theileriasis. Tetracyclines show moderate efficacy in sick cattle.

Supportive therapy by way of administration of haematinics, B-complex and inferon etc., may be used. Blood transfusion can also be done. 10 ml of 3.8% soln. of sodium citrate is added to 100 ml of blood for the sake of transfusion through i/v routes. 5% dextrose saline may also be given through i/v in sick animals. Halofuginonelactate and parvaquone are found to give good results in the cases of theileriasis.

The disease can be controlled by destroying the ticks by hand picking or application of acricide externally. Recovery of animals is followed by a solid immunity in them.

Surra (Hindi for Rotten)/Trypanosomiasis

It is a protozoan disease of cattle, buffaloes, horses, elephants, donkeys, mules and dogs etc., and is caused by *Trypanosoma evansi*. Tabinidae (Tabnid files) spread *T.evansi* infection mechanically to healthy animals from diseased ones by their bites. Ruminants like cattle exist mainly as reservoirs. Surra is an endemic disease of cattle, horses and camels etc. in tropical countries like India.

Signs

There is a loss of glycogen from the liver and muscles in the affected animals due to its rapid utilization by the *T.evansi* and the animals become weak and emaciated inspite of good appetite. In horses, emaciation and oedema are important signs of the disease. Urticarial plaques are seen on the neck

and flanks in these animals. Oedema of the muzzle, chest wall, sheath, scrotum and legs up to the knees and hocks can also be found. Ulceration and necrosis may also occur in these oedematous areas and abscesses may develop requiring three or more months for healing. Chocolate coloured ecchymoses are found at skin mucosa junctions in the diseased animals. Such haemorrhages can be found on membrana nictitans and anus. Temperature may shoot upto 106^0F and death may occur in a few days or months. The main clinical highs are fever, progressive emaciation, anaemia, subcutaneous oedema, nervous changes and death etc.

In dogs, oedema can be noticed in the scrotum, ears and neck etc., there is frequent cloudiness or opacity of the cornea. A rapid emaciation in the body with change in the voice is seen in dogs which may die within two weeks.

In camels, the disease is also called tibarsa which is its possible period of duration. Affected animals show loss of condition, weakness and debility.

Cattle and buffaloes are main reservoirs, but surra can occur in acute form in these animals but one often sees a subclinical infection. Buffaloes suffering from frank cases of surra die suddenly giving a suspicion of anthrax in these animals.

The elephants show muscular weakness and the back bones become prominent. There is a shrinkage in the back muscles and normal bagginess of the skin due to loss of muscle glycogen. The infected cats with surra show pyrexia and loss of hair. The disease takes a semi-chronic course and extend over 1 to 2 months in these animals.

Pathology

Oedema extending subdermally can be seen in the affected animals. Necrosis can be seen in the skin on the thoracic and abdominal walls. Anaemia is present and

petechiae can be seen on mucous surfaces and also in the liver and renal parenchyma. Sugar, red cells, protein and haemoglobin may be found in the urine of affected animals. Spleen may be enlarged with serous fluid in the body cavities. Stomach and intestines show congested areas. Acidosis, anoxia and elevated level of potassium, glutamic oxaloacetic and glutamic pyruvic transminase can be found in the cases of surra. There are no specific lesions in surra cases but emaciation and jaundice indicate *T. evansi* infection in animals.

Diagnosis

It is based on the symptoms, progressive anaemia, emaciation and icterus lesions and detection of *T. evansi* in the stained blood films of affected animals. It is quite helpful to await a paroxysm before the protozoa can be detected in the stained blood films either by Leishman's or Giemsa's stain. Mercuric chloride test is done in the camels to diagnose the disease. This is performed by adding a drop of serum to 1 ml of a 1: 25,000 aqueous solution of mercuric chloride. A white precipitate is formed in the mixture within a few minutes in cases of positive infections. There is no such reaction with serum obtained from healthy camels. It is of no use in other animals (for example, cattle). Formal gel or stilbamidine test is based on the increased level of serum proteins in the patients of surra.

Treatment/Management

In case of surra, the followings steps are to be adopted :

(i) Treat patients with effective drugs.

(ii) Use of fly proof sheds and spraying of the sheds with insecticides. This procedure lessens the changes of mechanical transmission.

(iii) Check up of surra cases during hot and humid climate i.e., especially during the heavy population of flies.

There are many drugs which were used in the past but now-a-days triquine, triboxine and trypnil etc. are found to be more effective. Since surra is a disease of cattle, buffalo and horse etc., it is very important to recommend supportive therapy in such cases. Haematinics, B-complex inj. and 5% dextrose solution can be given to sick animals.

In severely anaemic patients, blood transfusion can be done. Blood transfusion can be done after having compatibility report or necessary precautions in view about the blood groups of the donors and recipients to avoid transfusion reactions. Berenil (diminazene and quinapyramine sulphate) are also effective in treating surra cases.

Anaplasmosis (Gall- Sickness)

It is a rickettsial disease of cattle and sheep caused by minute obligate intra-erythrocytic parasites of the genus of the order Rickettsiales i.e. the genus *Anaplasma.* The two species of this genus are *Anaplasma marginale* and *Anaplasma centrale.* These organisms occur as spherical cocci like particles in the erythrocytes. *A. marginale* is a round or oval particle of chromatin (0.15μ in diameter) which is situated in or on the red cells but *A. centrale* is more centrally placed in the red cells. One form of the organism is usually found in the red cells but the red cells may contain two or four of these forms. These protozoan parasites multiply by simple fission and are transmitted by tabnid vectors and ticks like species of the Genus *Boonhilus, Rhipicephalus, Hyalomma, Ixodes and Dermacentor.* In this disease, there is only stage to stage transmission but hereditary transmission may occur. Contaminated surgical instruments can also transmit it mechanically.

Signs

Incubation period varies from 14 to 40 days after

infection and from six weeks to 3 months after the bite of the infected ticks. There is dullness, emaciation, pallor of the tissues depression and loss of condition in the affected animals. Anaemia is a very important feature of this disease and red cells count drop to one to 2 millions per cu. mm. and the blood becomes thin and watery Anisocytosis, punctate basophilia, polychromasia and nucleated red cells are noticed in the blood films. There is usually no haemoglobinuria and jaundice may be noticed in the later stages. Death in the imported cattle rises to 80%. The affected animals show rise in temperature upto 107^{0}F. The temperature may become normal or subnormal before the death of the animals. Breathing is accelerated and animals show depraved appetite, exhaustion and lack of rumination or appetite. The skin and mucous membranes become yellow and anaemic. There is a presence of stiff and unsteady gait in the sick animals. There is also no change in the colour of urine. Constipation can be noticed in them and the animals pass blood stained faeces covered with mucus. Death occurs within 24 hours in the acute cases. Chronic cases are marked by anaemia. There is very slow recovery and also practically no mortality in enzootic areas.

Pathology

There is an emaciation and icterus in the body. The lymph nodes are enlarged and oedematous. Enlargement of heart with marked petechial haemorrhages is noticed. The blood is thin and watery and does not stain the fingers. There is presence of gastroenteritis and the lungs are anaemic with changes like emphysema. The liver is enlarged, brownish yellow or deep orange in colour and saturated with bile and the gall-bladder is distended with dark green bile. The spleen is enlarged and has a soft pulp. A yellowish discolouration may be found throughout the viscera. Excess of fluid is found in the body cavities. The kidneys are congested and myocardial haemorrhages are seen in the dead animals.

Chronic cases of anaplasmosis is marked by serous atrophy of bone marrow.

Diagnosis

The disease can be diagnosed on the basis of symptoms and lesions. It is confirmed by detecting the organisms in the red cells of the stained blood films of the sick animals. A complement fixation test is also done to diagnose anaplasmosis. A serological test like polymerase chain reaction (PCR) confirms its diagnosis.

Treatment/Management

Antibiotics like tetracycline and terramycin can be given to sick animals. Symptomatic treatment is also done by administration of antipyretics in cases of high fever. Haematinics, blood transfusion, and fluid therapy are also given to protect the animals from severe anaemia and exhaustion. Supportive therapy may also be given by injecting B-complex with liver extract. Liv-52 tab. can be given orally.

For effective control of anaplasmosis, the vectors i.e., ticks must be destroyed. Ticks can be picked up from the host's body and burnt. Acricides such as Butox solution etc., may be used externally to destroy the ticks. The sick or carrier animals should be separated from healthy ones to check spread of infection. Proper sanitation is to be maintained in cattle shed.

Prophylactic immunization by administration of vaccine can also be done. Vaccination with killed *A. marginale* vaccine or live *A. centrale vaccine* is carried out in susceptible livestock of endemic zones.

Coccidiosis

It is a protozoan disease affecting several domestic animals which is caused by many different *Eimeria or Isospora*

spp. The coccidia are intracellular parasites in the epithelium of the alimentary tract. Serious damage is caused by coccidian in young animals like calves and lambs by growing in the intestinal mucosae during asexual cycle. Asexual life cycles (one, two or more) are followed by a sexual life cycle resulting information of oocysts which have to reach the ground infaecal matter for sporulation (i.e., formation of spurulated oocysts). That is why the coccidiosis is a self limiting disease provided the hosts are protected from further infection. Several species of Eimeria or Isosopa cause coccidiosis in animals. *Eimeria tenella and Eimeria necatrix* are very pathogenic in fowls. *Eimeria zurnii* is pathogenic in cattle. *Eimeria stiedae* causes hepatitis in rabbits and papilliform ingrowths are formed in the bile ducts of rabbit. Young animals like calves, lambs, piglets, kids (rarely foals) are very susceptible to coccidial infection. Coccidiosis is marked by high morbidity with low case fatality in the affected animals. Schizogony in the epithelial cells is influenced by developing immunity in hosts.

Pathology

In cattle the lesions in the large intestine show thickening, congestion and haemorrhages in its mucous membranes. Mucous membrane is covered with shreds of mucus or epithelium. Erosions are also formed in mucosae. The infected animals become carriers and symptomless infection persists in such apparently healthy looking animals.

In acute form, the faeces are found to be thin, foetid and mixed with mucus and blood. Animals show straining, tenesmus and emaciation and may die within a week or 10 days and mortality may reach upto 50 per cent.

In subacute coccidiosis, diarrhoea and debility can be found in animals. In coccidiosis in sheep and goat, there is diarrhoea and dysentery and death may occur. Enteritis is found in the small intestine and yellowish white patches

about ½" diameter in mucosae represent areas of sexual stages. (i.e., gametocytes etc.). Gametogenesis is marked by entry of merozoites in sexual phase of the life cycle. Sporogony is marked by development of the fertilized macrogamet within the oocysts outside the body of the hosts.

In schizogony in fowls, coccidiosis causes death due to schizonts being formed following entry in large numbers of merozoites in the subepithelial tissue of caeca. In short, destruction and sloughing of mucous membrane and submucosa are found in caeca because of schizogony. Caeca show multiple haemorrhages and may be enlarged and distended with haemorrhagic contents.

In the debris of inflamed caeca, caecal plugs of caseous material are found. The aforesaid lesions are found in cases of *Emeria tenella* infection in chickens. The birds after recovery from the disease become resistant. Two to three generation of schizogony (i.e., formation of schizonts due to repeated division of trophozoites arising from the conversion of a sporozoite in the intestinal epithelium) may be seen in the infected chickens.

In *Eimeria necatrix* infection in birds, the lesions in the form of haemorrhage or white spotted areas discernible to the naked eyes are found in the small intestines. White spots may indicate masses of schizonts. The lumen contains blood which stains the mucus.

Diagnosis

The disease is diagnosed on the basis of symptoms, lesions and detection of oocysts in the faecal matter and merozoites or developmental stages in the epithelial cells of the infected intestines. Stained sections of tissues reveal the coccidial parasites.

Treatment/Management

Since coccidiosis is a selflimiting disease, proper administration of drugs successfully controls outbreaks. The infection of birds with coccidia needs to be checked by all means. The drugs to be used to treat coccidiosis, are sulphadimidine, thalazole and nitrofuran etc., in domestic animals and birds e.g., cattle, calves, sheep and goats and poultry. It is worthwhile to mention that sulpha drug is a drug of choice to treat coccidiosis in the animals. Amprosol 20% @ 30 gm in 100 litre water is very useful in coccidiosis and can be given to minimize dehydration and anaemia etc. Blood transfusion in young stock may also be advisable. The severe straining in coccidiosis can be reduced by giving intestinal sedatives or epidural anaesthesia. Injection of prepaline forte and B-complex with liver extract can be recommended in large animals.

Toxoplasmosis

Toxoplasmosis is an infectious disease of domesticated and wild animals, which is caused by *Toxoplasma gondi.* It is a universal systemic coccidian of the suborder *Eimeriina.* T. gondi is a specific parasite of the members of the family *Felidae,.* which are defiinitive hosts of *T. gondi.*

Three infective stages are :

Infective stages	Intermediate hosts (farm animals)
1. Tachyzoites	Multiplying forms in the body of intermediate hosts.
2. Bradyzoites	Forms present in the tissue cysts in intermediate hosts
3. Oocysts (containing sporozoites)	Forms present in the faeces of intermediate hosts.

Oocysts are infective stages in farm animals and livestocks (intermediate hosts of *T.gondi*) which ingest the oocysts excreted by rats. The oocysts invade the hosts and form tissue cysts. Tissue cysts damage the neurosystem, myocardium, pulmonary tissue and placenta and even foetus of pregnant animals. Human toxoplasmosis arises from tissue cysts in animals. Ingestion of tissue cysts in carrion infect the swines. Abortion and stillbirths are the main signs in ewes. Encephalitis, pneumonia and neonatal mortality can occur in other species of animals but these pathological effects are minor manifestations of toxoplasmosis. It is a serious zoonosis and the parasites in the meat are sources of infection to humans. Seropositive prevalence of toxoplasmosis has been noticed in goats, horses and swines. Infected cats shed oocysts in their faces. These oocysts grow in infected intermediate hosts. Humans are intermediate hosts of *T. gondi* and are infected from ingestion of bradyzoites and tachyzoites in meat tissues. Raw or uncooked meat is a disease risk to man.

Pathology

T. gondi is an intracellular parasite and grows in the cells of reticuloendothelial cells and central nervous system. Sporozoites from oocysts or bradyzoites multiply in the intestinal epithelium which is ultimately destroyed. Released toxoplasma reach other organs, blood stream and set up the stages of parasitaemia (i.e., toxoplasma in blood). The effects of toxoplasmosis are congenital encephalitis, febrile exanthema, pneumonitis and enterocolitis in infected animals. A weak immunity causes the tissue cysts to rupture and inflammatory cells are noticed in the infected tissues and the lesions appear in the granulomatous form. Pregnant sheep and goat suffer from abortion. Neonatal mortality is seen in sheep, Fever, dyspnoea, nervous signs, anorexia and early

hyperexcitability and lethargy at later stages are the signs of toxoplasmosis in cattle. Pregnant pigs abort and the piglets are premature or stillborn. Diffuse necrotising and nonsupporative infiltration of brain parenchyma, accumulation of red cells in the Virchow- Robin space, delimited areas of coagulation necrosis in the hepatic lobules, cuboidal or columnar lining of the alveolar walls (foetalisation of lungs), coagulation necrosis in the enlarged lymphnodes, ulcers in the intestine, lymphocytic infiltration and presence of Toxoplasma in the cardiac muscles and focal necrosis in the infected placenta are some important lesions of toxoplasmosis.

Diagnosis

It is based on the symptoms, lesions and detection of bradyzoites in the infected animals. PCR and detection of parasites in tissues confirm its diagnosis. Immunohisto chemical staining techniques confirm the diagnosis of toxoplasmosis in the infected individuals

Treatment/Management

Use of sulphamethazine and pyrimathamine as a combination proves effective in pregnant ewes. Drugs are effective against proliferating parasites in the tissues of hosts but fail to eradicate the disease in the patients. Treatment of infected ewe is usually not indicated.

Some main signs and lesions etc., of protozoal diseases are given in Table 20.

Table 20. Some signs and lesions in protozoal diseases

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
1. Coccidiosis	Species Involved *Eimeria Zurnii, E.bovis, E.arloingi,* and *E.intricata, E.canis and Isosproa bigemina, Isosproa suis E.arloingi* and *E.fauri*	Cattle, Sheep, Dogs, Pigs, Goats.	An acute disease in young animals (cattle, buffaloes,sheep and goats etc).Intestinal epithelial cells are parasitized by coccidia.Both asexual and asexual generation i.e. Schizogony and gametogenesis occurs in the same host but sporogony (involving development of oocysts) occurs outside the body on the proper availability of oxygen and some other conditions.Schizogony is influenced to a great extent by developing immunity of the host.Thin and foetid faeces mixed with blood or mucus is noticed in acute coccidiosis in cattle and buffaloes.Mortality up to 50% in cattle.Tenesmus, straining and progressive emaciation in the affected cattle.A mixed infection of *E.bovis* and *E.zurnii is* noticed in cattle. Localization of	1.It is based on the symptoms and the lesions of haemorrhagic mucoid enteritis. 2.Detection of oocysts, schizonts and merozoites in direct smears of faeces and intestinal scrapings. 4.Sporocysts with two sporozoites in each sporocyst and two sporocysts with four sporozoites in each sporocysts are noticed in *Eimeria* sp. and *Isospora* sp. respectively.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			different coccidia in different parts of the intestine.Sporozotes released from the oocysts and merozoites emerging from the schizonts cause death or destruction of the intestinal epithelial cells.Merozoites differenciate in macrogamets for the sexual life cycle of coccidia.Oocysts are formed following fertilisation of macrogamets by microgamets for starting the stage of sporogony.The oocysts develop outside the body of the hosts on the ground under suitable conditions and a fresh life cycle of the coccidia starts following ingestion of these cysts by suitable hosts.A check of reinfection converts coccidiosis in to a self limiting disease owing to elimination of all coccidia from hosts'bodies.	

Disease	Causes	AnimalsAffected	Signs and Lesions	Diagnosis
2. Trypanosomiasis (surra)	*Trypanosoma evansi* Vectors Biting flies (*Tabanus* spp.) transmit this disease to healthy animals.	Cattle, buffaloes, horses and camels etc., cattle and buffaloes act like reservoirs of *T.evansi.*	A protozoan host specific disease marked by fever, anaemia, emaciation, nervous signs and death in the diseased animals and increased incidence of surra during rainy seasons favouring breeding of biting files. In sick animals,there are intermittent fever,oedema of the dependent parts of body and loss of weight despite good appetite. Nasal discharge and terminal nervous symptoms e.g., paraplegia, paralysis, delirium and presence of trypanosomes in the blood of cattle and buffaloes during acute phase. Pale mucosae of stomach and small intestines, enlargement of spleen infiltrative changes of leucocytes in the muscles are important changes in surra. Cases of irregular	1.It is based on the symptoms,lesions and detection of trypanosomes in the blood. 2.Death of rodent inoculated with suspected blood of sick animals. 3.Mercuric chloride formal gel or stilbamidine test for increased proteins levels are carried out to diagnosis cases of surra.. 4.Enzyme like immunoabsorbent assay (ELISA),complement fixation and indirect fluorescent antibody test are done to confirm its diagnosis. Appearance of white precipitate following addition of one drop of serum from

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			oestrus,still birth and abortion may be noticed in bovines.There are no characteristic lesions in the animals died of surra.	suspected cases to 1ml of 25000 dilute solution of mercuric chloride in water is noticed in to the cases of surra in camels.
3.Babesiosis (red water)	*Bibesia bigemina* in cattle and buffalo,B.ovis and *B.motasi* in sheep and *B.equi* in horses and *B.gibsoni* and *B.canis* in dogs are causes of this disease. Transovarian transmission by *Boophilus annlatus* or *Boophilus* microplus in Indian cattle.Hereditary and stage to stage transmission is shown by the infected ticks.	Cattle, Buffaloes, horses, sheep, swines and dogs etc.	A febrile disease (temperature upto 106° F) of mammals (cattle and buffaloes). High susceptibility is seen in the age group of 6-12 months. The parasites (i.e., *Babesia* spp.) inhabit the red cells and cause the release of the haemoglobin from the red cells in the plasma. The reticuloedothelial cells of bone marrow and spleen convert the iron free moiety in to bile pigments. Later, the bile pigments in the circulation cause haemolytic jaundice (icterus) in the affected animals amd haemoglobin in the urine causes red urine in the patient e.g., cattle. In short, the effects are jaundice,	1. It is based on the symptoms and the lesions. 2.Detection of *Babesia bigemina* in the red cells of cattle. 3.ELISA and complement fixation test confirm its diagnosis.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			haemoglobenaemia. Haemoglobinuria, obligocythaemia, anisocytosis, poikilocytosis and nucleated red cells (erythroblasts) etc. Transmission of babesia by ticks like *Boophilus* spp and *Hyalomma anatolicum* etc. The main lesions include ecchymoses in the subcutaneous tissues on the heart, mucosae of the stomach and intestine, enlarged spleen (increased upto 2 to 4 times the normal size), indistinct Malpighian corpuscles and trabeculae, enlarged liver with fatty changes and swollen kidneys with degenerative changes and casts in the tubules. The urine in the bladder is red or tinged with blood. Gastroenteritis and icteric discolouration or pallor of mucosae, all visceral organs and yellow intima of the	

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			arteries etc. are some important changes of babesiosis in cattle. Babesia is also found in the capillaries of the brain and carriers or recovered animals from babesiosis are immune due to premunition (or residual parasitism) and the immunity in them lasts a year or so. Relapse of the disease may occur due to reduced premunition from some causes.	
4. Theileriasis (theileriosis)	*Theileria annulata* (the cause of tropical theileriasis) The vectors are ticks like *Hyalomma* anatolicum In cattle a three host tick marked by stage to stage transmission i.e., both larvae and nymphs turn to be infective.	Cattle, sheep, goat and ungulates. *Theleria annulata* is highly virulent for crossbred or imported exotic European cattle in India with great mortality.	A febrile blood protozoal infection marked by anorexia, diarrhoea, loss of weight, dysentery, respiratory distress and iceteric mucous membrane. Thelerial forms in red cells and schizony in the lymphocytes are noticed in the affected animals. Schizonts are seen as faint blue masses of cytoplasm containing several red chromatin dots. Chromatin dots surrounded by some cytoplasm are called	1. It is based on the symptoms and the lesions like enlarged superficial lympha (commonly pescapular lymph nodes) in the infected animals. 2. Lymph node or liver biopsy is done to detect schizonts called (Koch's blue bodies) in stained smears of the lymph nodes. 3. Indirect fluorescent antibody test.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			merozoites. Merozoites enter red cells and exist in them possibly as gametocytes. The thelerial forms are seen in red cells as minute oval or coccoid bodies or as commas or red shaped structures. Blood containing schizonts is infective for healthy animals. Survivors or recovered animals are solidly immune due to premunition(i.e., occurance of no relapse). The main pathological lesions are oedema of the tongues and subcutaneous and intramuscular tissues, serous exudates in the body cavities, petechiae or haemorrhages on serous and mucous membranes, enlarged and oedematous lymph nodes, haemorrhagic or ulcerative gastritis in the abomasums, excess of pericardial and	

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			pleural fluids and enlarged liver with mottled appearance. Affected animals die of asphyxia. Almost no change is seen in spleen.	
5. Anaplasmosis (gall sickness)	*Anaplasma marginale* and *A.centrale* of the order Rickettsials Vectors, *Hyalomma spp. Boophillus, microplus, and Dermacenter andersoni* transmit its healthy live stock.	Cattle, sheep and goats	A febrile disease (temperature upto 105°F) of ruminants like cattle, sheep and goats marked by severe debility, anaemia, emaciation, jaundice (i.e., pale mucosae), depression, constipation, dyspnoea and death of the affected animals. Incubation period varies from 3 to 4 weeks. Death within 24 hours in paracute cases. A. subclinical form of anaplasmosis is seen in sheep and goats. Red cell count reduced to 1.5 million, immature red cells in the blood films, anisocytosis, poiikilocytosis, puctate basophilia, abortion in pregnant animals and	1.Based on the symptoms and the lesions. 2.Detection of *Anaplasm* spp. in red cells in the stained films. 3.Complement fixation and dot ELISA tests etc. 4.Presence of tick on the body of affected animals supports a positive or a suspected diagnosis.

Disease	Causes	Animals Affected	Signs and Lsions	Diagnosis
			hyperexciatability etc. some other signs. The main pathological lesions are as under: 1. Thin watery blood, excess fluids in the serous cavities and deep orange liver, gall bladder distended with dark green bile, congested kidneys, a severe enlargement of spleen with soft pulp are some important changes seen in dead animals. Antierythrocyte antibodies exacerbate anaemic changes. 2. Subepicardial haemorrhages, extramedullary haematopoiesis in the spleen and other organs, dark red faeces covered with blood or mucus, swollen and oedematous lymph nodes are important postmortem findings. Parasitised red cells are destroyed by reticuloendothelial cells of the spleen. 3. Anaplasmosis is marked by absence of haemoglobinuria. Survivors are carriers of anaplasmosis and a state of preimmunity is seen in the recovered animal patients or carriers of residual parasitism.	

Chapter 5
Parasitic Diseases

FASCIOLASIS (Distomiasis)

Distomiasis is caused by species of the families like *Fasciolidae* and *Dicrocoelidae*. The important species of these families are :

(i) *Fasciola gigantica* (common liver fluke in India).

(ii) *Fasciola hepatica* (common liver fluke).

(iii) *Fascioloides magna* (large liver fluke).

(iv) *Dicrocoelium dendriticum* (lancet fluke).

Fasciola hepatica is noticed in the liver, bile ducts and gall bladder in cattle and sheep and animals like, goat, horse, dog, deer and pig etc., may also be parasitised. Ova are laid by this parasite in the biliary passages and they reach the intestine with the biliary secretions to be finally expelled with the faeces. The micracidium emerges from these ova and the cercariae (motile forms) escape after many developmental stages of the miracidium in the body of snails. These cercariae encyst on the tips or blades of the grasses as metacercariae which on being grazed with grass or ingested by the suitable hosts like cattle, reach mature stages finally in the biliary passages of the liver.

Signs

The main signs of fascioliasis are :

(i) Weakness, oedema, anaemia (paleness of the mucous membrane), icterus and emaciation.

(ii) Diarrhoea, constipation and oedema in the intermandibular spaces (called bottle jaw in parasitic diseases).

(iii) Abortion in the pregnant animals. In cattle, the faeces

may be hard or brittle due to constipation.

(iv) Prostration and death in the final stages

The parasites, on being lodged in the biliary channels, cause icterus (i.e., obstructive icterus).

Pathology

The main lesions of this disease are :

(i) Abscesses in the lungs by the wandering infected parasites.

(ii) Destruction or damage is caused to the hepatic cells by the migrating flukes i.e., a kind of traumatic hepatitis. The flukes, after passing through the intestinal walls, wander in the peritoneal cavity and penetrate into liver substance. These parasites in the liver lie in a mass containing blood, fibrin and cellular detritus. Cells like neutrophiles, eosinophiles and lymphocytes may also be added to the infiltrate or exudate. Macrophages or epithelioid cells can also be found around the dead larvae or flukes. The liver capsule reveals small perforations and hepatic parenchyma is swollen and severely damaged or destroyed by migrating young flukes.

(iii) Occurrence of anaemia, eosinophilia and peritonitis. Such signs with sudden death are noticed in the sheep. Eggs are laid by the flukes in the biliary passages on 10th day postinfection. These flukes cause proliferation of the biliary epithelium to form papillary and glandular hyperplasia. Occlusion of the bile ducts may follow such changes in the biliary passages. The mucosae of the gall bladder can also undergo hyperplasia. Fibrous proliferation occurs in the walls of bile ducts which may also get calcified. The fibrosis or scarring around the bile ducts may spread into the surrounding liver lobules to produce marked proliferation of connective tissue (i.e., perilobular fibrosis or cirrhosis). Destruction of the liver parenchyma leads to weight loss of the patients.

In such animals with liver damage, hypoproteinaemia, eosinophilia and anaemia (normocytic normochromic) etc. are found. Albumin (plasma) leakage through the bile ducts causes hypoproteinaemia. Anisocytes, poikilocytes and basophilic stippling can be noticed in the liver fluke disease. *Clostridium oedematiens novi* can grow in the yellowish white necrotic lesions produced by roaming or migrating immature flukes in the liver of sheep infected with *F.hepatica*. Acute hepatitis develops and a pathological state of black disease occurs in the sheep (aged 2 to 4 years). The acute, subacute and chronic inflammatory lesions are noticed in the liver in the affected animals of all the age groups. In fascioliasis, pericellular fibrosis (fibrous tissue growth around single liver cells) and monolobular fibrosis (i.e., proliferation of fibrous tissue around individual lobules) are noticed after 12-30 weeks of infection. Pipestem liver is noticed in fascioliasis in cattle and arises from changes like fibrosis and calcification in the wall of bileducts invaded with the flukes. In cattle, the flukes can be found in the lung parenchyma in which cysts containing brownish purulent gelatinous material with living or dead flukes are noticed. G. *explanatum* causes hepatic trematodiasis (e.g., biliary fibrosis) in the parasitised liver of cattle (fig.-).

In *Dicrocolium dendriticum* infection, there is a thickening of the walls of the bileducts and occurrence of periportal fibrosis. The epithelium in the bileduct of the liver shows hyperplastic changes.

Diagnosis

It is based on the following :

(i) Symptoms and lesions.

(ii) Detection of eggs or ova of the flukes in the faeces of the affected animals. ELISA is also helpful in diagnosis of the cases of fascioliasis.

Treatment/Management

The same steps as given under amphistomiasis are followed. Certain details about patent preparation or dosage schedule of different diseases (including parasitic diseases are given in table 37 and 39.

Amphistomiasis (Paramphistomiasis)

The trematodes of the family *Paramphistomidae* set up a pathological state known as amphistomiasis (stomach fluke disease) in domestic animals like cattle, sheep and goats etc. Paramphistomes are found in the rumen, reticulum and intestines of the ruminants and immature forms of these trematodes are very pathogenic in many kinds of ruminants. The trematodes of the family are also found in pigs, equidae and human beings. *Paramphistomum cervi* is a conical pear shaped fluke and is red in colour. This fluke is found in cattle, sheep, goats and buffalo etc., *Cotylophoron cotylophorom* is noticed in the rumen and reticulum of sheep, goat and cattle. *Calicophron calicophorum* is a parasite of sheep and cattle and found in their rumen and reticulum. *Gigantacotyle explanatum* is noticed in the bile ducts, gall bladder and duodenum of buffalo and cattle. The cattle are less commonly infected with the flukes.

Pathology

Liberated micracidia from the eggs enter the bodies of suitable snails in which they undergo different developmental stages i.e., formation of sporocysts and rediae inside the body of the snails. The rediae release the cercariae inside the body of the snails in which they remain for some time for the sake of maturation. After they have undergone maturation inside the snails, the mature cercariae which are dark brown are released and the liberated cercariae are immature amphistomes (highly pathogenic forms for animals). These amphistomes undergo encystment on plants in the form of metacercariae which are dark or black in colour. The

metacercariae ingested by the ruminants along with green vegetables are immature amphistomes. On being released in the gut from the state of encystment, these pathogenic immature forms attack the intestinal mucosae (i.e., in the first three meter of small intestine) and after a period of 6 to 8 weeks, they migrate to the rumen through the reticulum where they reach the stage of maturity. In the forestomachs, they live in the non- pathogenic form. But the immature paramphistomes penetrate into the mucosae of the duodenum of the upper ileum. (Fig. 4; p. 200) The parasites engulf plugs of the mucosae into their suckers. Haemorrhages and necrotic changes are producaed in the mucosae (i.e., haemorrhagic duodenitis). Catarrhal and haemorrhagic enteritis are noticed in the duodenum and jejunum etc. The affected animals show anaemia, hypop-roteinaemia, oedema and emaciation. Profuse, watery, foetid diarrhoea, acute enteritis and marked weakness are noticed in the affected animals which frequently die. Immature amphistomiasis is commonly seen on the river sides or on plains in India during rainy season when the population of the snails is very high in waters. A lot of calves and kids die of immature amphistomiasis.

Diagnosis

It is based on the symptoms (foetid watery diarrhoea), lesions (intestinal ulcers and erosions) and detection of immature amphistomes in the fluid faeces. The paramphistomes can be found lying over or adhering to the mucosae of the duoenum and in the intestinal contents during the postmortem examination of the fatal cases of immature amphistomiasis.

Treatment/Management

These are important parasitic diseases in cattle, sheep and goats etc., which are marked by diarrhoea and debility etc. Before the specific drug is given to the animals, the presence of this infection should be confirmed by faecal

examinations.

The drugs used for this disease are :

(i) Carbon tetrachloride can be used to treat such cases. One should be careful in avoiding its use in debilitated animals.

(ii) Distodin tab., fasinex tab, Terramaffin tab. Talzone F. etc., can be used.

(iii) Trodex inj. can be used s/c in positive cases.

(iv) Fluid therapy 5 to 10% Dextrose liver extract. Some of the drugs acting on the guts can be used.

(v) To improve the general debilitating conditions, calboral or mifex can be used.

Oxyclozanide, hexachlorophene, niclosamide are also used to treat the cases of amphistomiasis in domestic animals.

Ancylostomiasis (Hook Worm Disease)

Hookworms are parasites of cosmopolitan distribution and produce this disease in man and animals. However, horses do not suffer from hookworms. The important hookworms are :

Table 21. Hookworms and their hosts

Hookworms	Definitive hosts
Ancylostoma dnodenale	Man
Necator americanus	Man and rarely dogs
Ancylostoma caninum	Dog and cat
Ancylostoma braziliense	- do -
Bunostomum phlebotomum	Cattle
Bunostomum trigonocephalum	Sheep and goat
Globocephalus urosubulatus	Swine

These worms have buccal cavities containing teeth and suck blood from the hosts. Adults inhabit the intestinal lumen. The infective larvae penetrate through the skin or enter the body through ingestion. They migrate to the lungs and, then,

get access to the tracheal exudate. They are swallowed and reach the gut where they grow as adults. When the hook worms penetrate into the skin of aberrant host, a specific type of dermatitis (creeping eruption) is produced.

Signs

The infective larvae (the 3rd stage larvae) produce ancylostome dermatitis in man (water itch). *A.braziliense* produces reddish papules and linear tunnels (creeping eruption). The larvae, on reaching the lungs, produce cough and pneumonia. The adult worms in the intestine draw plugs of mucous membrane in their mouth, abrade the mucose with their teeth and the juices produced by them digest the mucous membrane. The anticoagulants produced by the worms prevent clotting. These worms leave bleeding points at the point of attachments and the blood escapes from these points for sometime after these parasites have moved on to other fresh spots. An *Ancylostomum caninum* can cause loss of 0.01 to 0.09 ml of blood in a day. Enough blood is lost from the body of the host to produce the state of anaemia. The affected animals become pale and anaemia. Emaciation hypoproteinemia, weakness and diarrhoea containing blood and mucus are seen in the affected animals. The hook worm infection may lead to cardiac failure or death of the patients. The anaemia produced in patients is usually microcytic and hypochromic. Eggs and occult blood are found in the faecal matter.

Pathology

Skin dermatitis is produced due to penetration of the infective larvae into the skin. Exudate rich in lymphocytes, eosinophiles and macrophages is seen in the dermis of skin. Prenatal infection occurs through the placenta caused by hookworm larvae when the larvae break into the alveoli from the alveolar capillaries, haemorrhage occurs in the alveolar spaces and leucocytes also infiltrate into the alveoli and

lobular pneumonia is produced in the affected animals. Fibrotic changes occur in the lung parenchyma. Bleeding continues from the denuded epithelium of the mucosae and the worms change their positions, leave bleeding points, cause also loss of blood and the worms produce enteritis. The red cells count may drop to 25 per cent of the normal. Hyperplasia of the haematopoietic tissue occurs in the bone marrow. Myeloid metaplasia is seen in the spleen.

Diagnosis

It depends upon the detection of hookworm ova in the faeces of the affected animals. Adult worms can be found attached to the intestinal mucous membrane in the dead cases. Larvae are found in the stained sections of the affected tissues. The symptoms and lesions also help the diagnosis of this disease in the animals.

Treatment/Management

Hookworm infection in dogs is characterized by diarrhoea, dysentery, anaemia and itching in the anal portion etc. At first, this disease is confirmed by stool examination.

The following line of treatment can be adopted for treating hookworm infections :

(i) Ancylol inj. (0.2 mg/kg body wt.) can be given s/c. Drugs can be repeated after 21 days.

(ii) Mebendazol or other broadspectrum anthelmentics can be used at the dose rate 50 mg per kg body wt. for three days in dogs.

(iii) Supportive treatment in the form of Livogen, Belymyl at the rate of 1 to 2 ml i/m on alternate days can be followed.

(iv) Iron preparation such as Inferon inj. 1 to 2 ml i/m twice weekly can be also given to raise haemoglobin level.

(v) Liv-53 tab. or sharcoferrol syrup can also be fed to animals.

Trichostrongylosis

Trichostrongyles infect the domestic animals like cattle and sheep etc. Affected animals show anaemia, cachexia, diarrhoea and debility. Lambs and calves die of this disease. The important trichostrongyles are as follows :

Table 22. Parasites and their hosts

Parasites	Hosts
Haemonchus contortus	*Sheep, goat and cattle*
Ostertagia ostertaqi	*-Do-*
Trichostronglylus axei	*-Do-*
Cooperia spp. e.g. *C. punctata* and *C. spatulata*	Sheep and cattle
Nematodirus spp. e.g. *N. spathiger*	*-Do-*
Mecistocirrus digitatus	*Sheep, cattle, goats and buffaloes*

H. contortus ingests large qualities of blood in the host body and lacerations in the mucosae and gastritis are produced. The infective larvae of *T. axei* burrow into the mucosa of the abomasum but other species of the genera *Trichostrongylus and Cooperia* invade the mucosa of the small intestine. Larvae of *Ostertagia spp.* penetrate into the abomasal mucosa and burrow deeper to cause severe damages to the gastric wall. These parasites develop in small nodules and the adults may emerge from the nodules in the mucosa.

Signs

Infection with *Haemonchus contortus* even causes death from loss of blood. There are oedematous swellings under the jaw and ventral abdomen. Mucoid degeneration takes place in the body fat (e.g., fat in the coronary groove of heart or subcutaneous tissue) to produce gelatinous tissue (serous atrophy of fat). Affected animals become weak, show staggering gait and become moribund and later die. Diarrhoea with thin, foeted, dark coloured faces and anaemia are important signs of infection with trichostrongyles.

Pathology

In acute cases, dead animals are severely anaemic. The mucous membranes are pale and the blood is thin and watery. Oedematous swelling in the subcutis under the jaw and abdomen and excessive amount of fluid in pericardial, thoracic and peritoneal cavities are important pathological changes. The body fat is highly gelatinized. The liver is friable, light brown and may show fatty changes. Large number of worms are present in the abomasum. Mucosa of the abomasum may show ulcers or red bite marks by the worms. Small round nodules are found in the abomasal mucosae and local lymph nodes are swollen. There is myeloid hyperplasia in the bone marrow and liver shows fatty changes and mucoid atrophy of fat is seen in the body. The term bottle jaw refers to submandibular oedematous tissues because of gastro intestinal parasitism.

Diagnosis

It is based on the detection of ova in the faeces and helminths in the abomasum and small intestine of animals. Anaemia and gelatinisation of fat (serous atrophy of fat) etc., aid the diagnosis of trichostrongylosis.

Treatment/Management

The round worms include different species or nematodes affecting various domestic animals. Diarrhoea, potbelly, urithriftiness, bottle jaw and fits etc., are important signs of disease in cattle and young calves.

The drugs for treating round worms infection are as follows :

(i) Piperazin adipate, vermex liquid dewormer, Helmacid and helatac, etc.

(ii) Broad spectrum antihelminthic drugs like ivermectins, benzimidazoles and imidazothiazoles etc., can be used to treat round worm infection. Other drugs such as panacure, albomar, zodex, banminth forte, zenil and

albedol etc. are used to treat trichostrongylosis affecting mainly ruminants and some other animals like pigs and rabbits etc.

Schistosomiasis

The disease produced by sachistosomes in animals is called schistosomiasis. These flukes inhabit the blood vessels and the eggs laid by them circulate as emboli in the circulation. On being lodged in the tissues, these eggs behave like foreign irritants to them and invoke pathological changes (mainly proliferative or of a granulomatous inflammatory type). The important blood flukes found in India are as follows :

Table 23. Schistosomes and their hosts

Species	Hosts	Anatomic sites
Schistisoma indicum	Cattle, sheep, goat, horse and camel.	Mesenteric, portal and pelvic veins.
S.incognitus	Dogs and pigs.	Mesenteric and portal veins.
S. nasalis	Cattle, goats and horse.	Nasal veins.
S. spindale	Cattle, sheep, goats antelopes and water buffaloes.	Mesenteric and portal veins.

Pathology

The male and female blood flukes copulate within the lumina of the blood vessels and move against the blood stream to lay eggs inside the small venules in the different organs or tissue of the hosts. The ova with the help of cytolytic fluid produced by them penetrate into the walls of the capillaries and move throughout the tissues to reach in the lumen of the intestine or the urinary bladder etc. The ova then leave the body of the host with the faeces. The ova which fail to be excreted with urine or faeces etc., reach at unusual sites in the body and provoke tissue reactions at such sites. A miracidium escapes from the fertile mature ovum in proper

environment and attacks a suitable host (for example, a snail) to complete further stages of their life cycle like formation of sporocysts etc. From the secondary sporocysts, the cercariae escape into the surrounding, search a right kind of definitive host and after penetrating into the skin of such host, they turn into matacercariae for progression to other stages in their life cycle. Metacarcarie enter small peripheral veins in the skin of the hosts, reach the lungs, then the liver and finally they lodge at certain anatomic sites of predilection like adult portal, mesenteric and nasal veins depending upon the species in definitive hosts involved. The lesions produced by eggs or migrating ova in the tissues of the hosts are small haemorrhagic ulcers or chronic inflammatory growths.

The lesions in the hosts result from the presence of adults or ova etc., in the veins of the body. The adults in the veins, ova in veins or tissues, cercariae in the skin and metacercariae during migration through the tissues elicit tissue reactions of lesions.

Phlebitis and intimal proliferation are the changes in the veins. The macrophages which have engulfed blood pigments discharged by the flukes can be found in the organs like liver and spleen etc. The ova in the organs like liver and spleen and in the capillaries of intestines etc., escape from them due to action of lytic enzymes (produced by them) and finally reach the lumen of the intestine etc. Small haemorrhagic ulcers are produced by migrating eggs and these eggs cause marked chronic inflammatory reaction in the tissues of the hosts. A microabscess containing neutrophiles and eosinophiles can be found around the eggs and giant cells of the foreign body types appear at the later stages. In sections of these granulomatous lesions, foreign body type of giant cells can be seen around the ingested ova. The spine is noticed at one terminal pole in case of *S. indicum, S.nasalis* and *S. spindale*. Eggs of *S. nasalis* are non-acid fast. Marked fibrosis is noticed around these fluke eggs. The emboli of the eggs of the flukes can reach organs like liver, spleen,

lungs, lymphnodes, skin, testes and brain etc. *S. nasalis* grows in the veins of the nasal mucosa in cattle, buffaloes, goats, sheep and horses. Rhinitis, mucopurulent discharge, sneezing, coryza, dyspnoea and snoring (snoring disease) are seen in the affected animals. Nasal veins may dilate and these are seen in the nasal mucosae. Nasal mucosae show chronic fibrosis and proliferation of the epithelial tissue. Pin head size eruptions and congestion of the nasal mucosae are noticed in the affected buffaloes.

Diagnosis

It is based on the detection of eggs in the faeces of the host or in the sections of the granulomatous lesions in the body of the affected animals.

Treatment of Nasal Granuloma

This disease is characterized by snoring sound produced by the affected animals. *S.nasalis* produces granuloma in nasal mucosae on the nasal septum. The diagnosis of the disease should be confirmed first by examining the nasal discharge.

The drugs used for treating nasal granuloma are as follows :

(i) Anthiomaline in doses of 20 ml i/m alternatively (maximum five injections) can be given.

(ii) Sodium antimony tartarate (Tartartmetics) 2 to 4% can be used twice weekly (maximum 5 injections). Tartartemetics may be dissolved in 5% dextrose saline and given i/v into the body. The drug is found very efficacious in treating nasal granulomas.

(iii) Ivomac inj. 10 ml s/c in case of nasal granuloma is claimed to have definite effect on this disease. Ivomac inj. can be repeated after 2 weeks and, then, on 25th day of first injection.

Ascariasis (Common Round Worms Infection)

The common round worms called ascarids inhabitate the gastrointestinal tract of mammals and birds all the world over. The ascarids are large worms in the small intestine of mammals but small ones e.g., caecal worms are seen in caeca of fowls. Unsegmented thick shelled or pitted eggs are laid by ascarids. Some of the ascarids of the family *Ascaridae* found in animals are as under :

Table 24. Ascarids and their hosts

Species	Definitive Hosts
Toxocara vitulorum	Cattle and buffalo
Parascaris equorum	Horses
Toxoxara canis	Man
Toxocara cati	Dog and cat etc.
Toxocaris leonina	Dog and cat etc.
Ascaris lumbricoides	Pigs.

Certain ascarids like *T.canis* and *T.catis* etc., may use an intermediate host before completing their life cycle in the definitive host. The intermediate hosts may be insect larvae, amphibia or insectivora. These parasites can show migration through the liver, lungs and many organs of the definitive hosts during their life cycle. They even may not show migration and return to the lumina of the gut after passing some time in the wall of the gut to attain the adulthood therein. The maximum damage is done to the organs (say, liver and lungs) owing to migratory activity during the prepatent period (a period elapsing between ingestion of the infective larvae and the appearance of the eggs in the faeces of definitive hosts) like man, dog, cattle, and horse etc.

Pathology

The main pathological changes are as follows :

(i) Depriving the final host of food and nutrients. The hosts starve and undergo emaciation and debilities but the parasites multiply and thrive well in hosts's body. One may find ascariasis as a febrile state in the patients.

(ii) Peritonitis following perforation of the intestinal wall by the round worms.

(iii) Injuries to mucosae owing to abrasions caused by the worms. The animal patients suffer form diarrhoea.

(iv) Icterus due to worm obstruction in the bile ducts. The worms may even enter the stomach or pancreatic ducts from the small intestine.

(v) Presence of neutrophiles, eosinophiles and lymphocytes etc., in the liver due to inflammatory reaction. Epithelioid cells, eosinophiles, lymphocytes and neutrophiles are seen around areas of caseous necrosis in the hepatic tissue.

(vi) Haemorrhages due to damages caused by migrating larvae in the pulmonary capillaries and inflammatory changes in the lungs. Bronchopneumonic changes in the lung parenchyma are noticed.

(vii) White spots in the liver due to fibrosis at the necrosed sites caused by the migrating larvae. Severe form of enzooting pneumonia of pigs and swine influenza are noticed in the pigs affected with ascariasis.

(viii) Presence of congestion, enlargement and subcapsular haemorrhages in the livers of the affected animals. Migrating larvae leave behind necrotic tracts during their movements in the liver parenchyma.

(ix) Oedema, subpleural haemorrhages and cyanosis are

noticed in the lungs. Tracheal scrapings may reveal larvae on microscopic examination. Presence of blood stained fluid is noticed in the thorax.

Diagnosis

It is based on the following :

(i) Detection of eggs in the faeces or larvae in the tracheal exudate at autopsy of the fatal cases.

(ii) Symptoms and lesions in the liver and lungs. In general, the worm infested animals are debilitated and suffer from diarrhoea.

Treatment/Management

1. Avoid exposure of young pigs and goats to infested adults and contaminated soil.
2. There should be periodical treatment of young animals.
3. Rearing of pigs in concrete pans to avoid the ascarid infestation.
4. Piperazine adipate, vermox liquid dewormer, helmacid, belatac etc., can be used. Pyrental tantrate and levamisole are found quite effective in worm infected pigs. Pyrental 250 mg/kg or levamisole 7.5 mg/ kg body weight gives good results in the buffalo calves.

Other suitable anthelmentics given to patients of ascariasis are ivermectin (0.3mg/kg), doramectin, flubendazole (5 mg/kg), oxibendazole (.15 mg/kg) and levamisole etc.

Some important features of parasitic diseases in animals are summarized is Table 25.

Table 25. Some important features of parasite diseases

Disease	Causes		Animals Affected	Signs and Lesions	Diagnosis
Fluke disease 1. Fascioliasis (a disease arising from ingestion of the metacercariae encysted on the blades of grasses).	***Fasciola gigantica in India,and F.hepatica in the western countries.Intermediate*** hosts are snails.These are as under;		Cattle and buffaloes in India. Horses, pigs and rabbits are occasionally affected.	A disease caused by liver flukes with marked characteristic chronic inflammatory changes of the bile ducts and liver in animals like cattle and sheep etc.The main symptoms are weakness,anaemia and iceterus in some cases and emaciation, constipation and diarrhoea are some other changes of the disease. In liver fluke infestation,the liver is enlarged, congested, soft and friable with numerous haemorrhages or opaque spots on the peritoneal covering. The haemorrhagic spots are caused by liver fluke larvae penetrating into the hepatic parenchyma from the abdominal cavity.One notices cavities in the liver containing liver flukes and such flukes are also found in the bile ducts.Microscopically,haemorrhagic tracts are noticed and these lead the liver flukes to enter the bile ducts etc.In chronic stages (i.e., cirrhosis),the livers are enlarged and bear nodulated appearance.The liver is yellow in colour firm in consistency and hard or difficult to be cut with knife.The bile ducts are enlarged, rigid and look like white tubes and the livers with calcified and fibrosed bile ducts are called pipe stem livers.The ducts possess thickened walls and contain	1.Based on the symptoms and lesions and detection of ova in the faecal matter. The ova are recognised by yellow shell with an indistinct operculum and embryonic cells.
	Trematodes 1.Fasciola gigantica 2.Fasciola hepatica	Intermediate hosts *(a)Limnea species e.g., Limnea luteola and L.truncatula (b)Limnea species*			

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			greenish brown mucoid fluid or inspissated bile,flakes,calcareous deposits and flukes etc.The thickened bile ducts are dilated and some-what calcified in the livers of cattle. Microscopically,the important changes are fibrous connective tissues around one or more lobules of hepatic parenchyma.The tracts in the liver used for migration contain neutrophiles,eosinophiles and lymphocytes.Macrophages,epithelioid cells and multinucleated giant cells are found around the dead larvae.Healing of the wounds in the liver is marked by ingrowth of granulation tissue and scarring.	

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
(b) *Gigantocotyle explanatum infestation* (hepatic amphistomiasis) Animals affected Buffaloes and cattle 3. Nasal granuloma (a disease caused by *S. nasalis* in the veins of cattle,buffalo goat and horses in India.	*Gigatocotyle explanatum* Its intermediate host is the snail called *Gyraulus convexiusculus* *Schistosoma nasalis* of the family *Schistosomatidae* *Indolanorbis exustus* (a snail) is its intermediate host(I.H)		The amphistomes inhabit in the bile ducts,gall bladder duodenum etc.of the herbivores like buffalo and cattle. It is commonly seen in the livers of affected buffalo. Intrahepatic biliary cirrhosis is caused by *G. explantum.* The flukes obstruct the bile ducts and are found in large numbers in the gall bladders of the buffaloes. A marked fibrosis is seen around the obstructed bile ducts. *Nasal Granuloma* The blood flukes live in the veins of nasal cavity of the patient and produce respiratory disturbances. A spine is located at one terminal pole of the ovum of *S. nasalis.* A granulamatous inflammatory lesion develops around the eggs of the schistosomes in the nasal mucosae and produce respiratory distress and abnormal the sound. Section of the stained ova reveal parts of miracidia.	1.Based on the symptoms and the lesions and detection of the parasite *G.explanatum* in the gall bladder. 2. Eggs of *G. explanatum* in faecal matters of the patients. Based on the symptoms and the granulomatous lesions containing ova of *S. nasalis* in the affected tissues. Its ova are acidfast in character as revealed by Ziehl-neelsen staining method.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
4. *Ancylostomiasis* (hook worm disease).	*(1) Ancylostoma caninum* in dog and cat *(2) Ancylostoma duodenale* and *Necatar Americanus* in man *(3) Bunostomum phlebotomum* in cattle *(4) Ancylostoma braziliense* in dogs and cats *(5) Bunostomum trigonocephalum* in sheep and goats.	Dogs, cats, humans, sheep, goats and cattle etc.	Hook worms have well developed buccal cavities and are blood suckers of man and animals. When these worms penetrate into the skin, dermatitis is produced in the affected patients. They reach the lungs and gain access to the intestine via the tracheal route. Creeping eruption (specific dermatitis) is produced in the aberrant hosts. Ingestion also causes infection of the hosts. The main signs are cough, pneumonia, anaemia (microcytic anaemia), weakness, diarrhea, progreesive emaciation and death. The other changes are : (1) Inflammatory reaction or skin sensitization in the viscinity of the migratory tract, infiltration of lymphocytes, eosinophiles, macrophases etc. in the dermis of the affected skin. (2) Haemorrahages in the alveoli of the lungs following rupture of the capillaries owing to larval movements. Lobularpneumonia, leucocytic infiltration in the lung parenchyma and secondary bacterial pneumonia in the devitalized or damaged parenchyma are other changes. 3.Hook worms damage the intestinal mucosae, cause bleeding, release anticogulants, feed	1. Based on the symptoms and lesions. 2. Detection of eggs in the faeces. 3. Hook worms attached to the intestinal mucosa. The worms are also noticed in the faeces or intestinal contents at autopsy. 4. Presence of the hook worm larvae in the injured tissues like skin and lungs etc.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			on the flowing blood and change the sites to attack fresh areas in the mucosae with the development of anaemia in the affected animals. 4. The larvae of these hook worm breach the placental layer and produce prenatal infection in young animals.	
5. *Trichostrongylosis* (hair worm or scour worm infestation)	A. *Trichostrongylus spp.* 1. *Trichostrongylus axei* in the stomach of the horse and also in the small intestines of sheep and goats. 2. *T.colubriformis* in cattle, sheep and goat. 3. *T.capricola* in goat B. *Ostertagia ostertagi* in the abomusum of cattle *C.nematodirus spp.* 1. *C.puncta* and *C.oncophom* in the abomusum of cattle *E. Haemonchus spp. 1. Haemonchus contortus* in the abomasums of cattle and sheep.	Cattle, sheep and goats Lambs and calves are severaly affected.	The hair worms belonging to several genera like Trichostrongylus, Cooperia, Ostertagia and Nematodirus predominantly attack young animals like lambs and yearlings. The worms are noticed in the alimentary tract (e.g., abomasums). Over crowding and malnutrition are predominant indirect predisposing causes. The hair worms have direct life cycle i.e., they reach the abamasums, attack the abomasal mucosae and then burrow in to the abomasal mucosae and feed on hosts blood. The larvae of the genera like *Trichostrongylus* and *Coopria* project partially from these inflammatory growths in the mucosae. Anaemia, presence of oedematous fluid under the jaw (called bottle jaw) and ventral abdomen particularly in sheep affected with such intestinal parasitism. Serous atrophy mucoid	1.Based on the signs and the lesions in the bodies of the dead animals. 2. Eggs in the faeces of infected animals. 3. Worms in the abomasal contents are easily seen following sieving of the contents.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			degeneration of body subcutaneous fat, and fat between calices of kidneys, weakness, staggering and cachexia are important signs of hair worm infection. The main findings are pale mucous membrane, pale viscera, thin and watery blood, and excess fluid in the thorax abdomen and pericardium, light brown liver with fatty changes and several motile worms in the abdomasal contents. The worms e.g., *Haemonclus contorius* are called stomach worms or barber pole worms. The larvae of the genus *Ostertagia* burrow deep into the abomasal mucosae to form nodules and project partially over these nodules.	
6.*Ascariasis*	*Ascaris spp.in* dogs *Toxocara cati, Toxocara canis* and *Toxocara leonina* in cats and dogs.*Ascaris lumbricoides* in man and *A.suum in pigs Toxocara vitulorum* in cattle and buffalo *Parascaris equorum* in horses and *Ascaradia* galli in fowls and turkeys.	Man, swine, dogs, cats, lions, tigers, cattle, equines and poultry.	Ascariasis in animals is caused by parasites called ascarids. The lesions are produced in the intestines and the parasites migrate through organs like liver and lungs etc., of the affected hosts. The worms from the normal usual habitat of the intestine can migrate in to the bile ducts or may cause perforation in the intestines. The main signs of ascariasis are poor growth, diarrhoea, pneumonia in young pigs, convulsions, intestinal perforation and	1.Symptoms , lesions and presence of ascarid in the guts and bile ducts. 2.Ova or adults in the faeces of infected hosts.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			obstructions in foals, anaemia, steatorrhoea and loss in weight of calves. Migratory activities by the ascarids cause the damage or destruction in the organs of the hosts. Subcapsular white spots in pigs are seen in the cases of chronic ascariasis. Oedema, haemorrhages and cyanosis are noticed in the infected lungs of animals. Infection in pigs causes emphysema, alveolar wall thickening with fibrin and eosinophills. Even haemorrhages are seen in the alveoli of the infected pigs. Anaemia and jaundiced caracasses are seen in fatal cases of ascariasis. Larval migration through lungs and livers and other somatic tissue results in prenatal infection in foetus. In *Toxocara canis* infection, ascarids gnaw on the intestinal mucosae and consume the nutrients needed by the hosts.	
2. Amphistomiasis (a) Immature amphistiomiasis (called cheraha in Bihar).	Immature amphistomes of the species like *Cotylophoron Cotylophorum and Calicophoron calicophorum*	Cattle, buffaloes, sheep and goats etc.	Severe diarrhoea, haemorrhagic duodenitis and high mortality in the affected animals i.e., particularly in young bovines, goat and sheep etc. Mature amphistomes are harmless and reside in the rumen, reticulum etc. Metacercariae encyst in the small	1.Based on the symptoms lesions and the presence of immature amphistomes on the duodenal or intestinal mucosae. 2.Presence of flukes in the loose faecal matter. 3.Eggs with transparent wall and distinct operculum in the faecal matter.

Disease	Causes		Animals Affected	Signs and Lesions	Diagnosis
				intestines,produce severe enteritis (duodentis),attain maturity in the intestines and then, migrate to four stomachs which reveal several mature adult amphistomes. Some important signs are anorexia dyspepsia, emaciation, exhaustion, increase in thirst, foetid loose faeces, anal rim or hind legs soiled with faecal matter, persistent straining, anaemia and pale mucosae.One can notice immature amphistomes attached to the duodenal mucosae and histopathologically, plugs of the intestinal mucosae are seen in the oral suckers of the flukes.The flukes migrate to the mucosae of the duodenum or some part of ileum etc. Necrosis, erosions, hyperaemia, petechiation in the intestinal mucosae, excess mucus and slough of mucosal epithelium are important findings of this acute disease. The flukes damage or destroy the intestinal mucosae, Burner's glands but do not appear to perforate the duodenal wall to enter the peritoneum.	
	Trematodoes *1.Cotylophoron cotylophorum* *2.Paramphistomum cervi* *3.Gartrothyla*	*Intermediate hosts* *1.Indoplanorbis exustu*			

Disease	Causes		Animals Affected	Signs and Lesions	Diagnosis
	x crumenife r 4.Fishaed erus enlongatu s.	*s 2.Iex ustus 3.Gyr a ulus conve xiuscu lus 4.Lim anaea luteol a.*			
7. *Echinococ cosis (hydatid disease.*	1.*Echinococcus granulosus* Its intermediate hosts are cattle sheep, pigs and horse etc. Definitive hosts are dogs, fox, wolf, jackal and man. 2.*E.multilocularis* Intermediate hosts are cattle, sheep, swines and human beings. The definitive hosts are dogs, jackal, wolf and man etc.			A serious disease of cattle, sheep, goats, horses, deer and man due to very harmful effects in organs like lungs, liver and other viscera of affected hosts by developmental stages of larval forms (bladder worms) of the tapeworms called *E.granulosus*. Larval forms are detected in the lungs, liver etc., of intermediate hosts like cattle, man, pigs and sheep etc., The cysts of *E.granulosus* grow slowly, develop fibrous capsules around it and also produce daughter cysts. The larvae enclose germinal layers which give origin to several spherical brood capsules. The brood capsules also possess germinal layers and several scolices (even upto 40) are noticed in such brood capsules. The daughter cysts (i.e., endogenous cysts) grow inside the parent cyst or outside the parent cyst as exogenous daughter cysts. *E.multilocularis* produces an alveolar form of hydatid disease marked by development of several exogenous	1. Based on the detection of ova or segment of *Echinococc us* spp. in the definitive hosts. 2. Histopath ology of the hydatid cysts in lungs and the liver etc. of the intermedi ate hosts. 3. Performan ce of compleme nt fixation test to confirm its diagnosis.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			daughter cysts in organs e.g. lungs of the intermediate hosts like cattle, cattle, sheep, horse and human beings. Distension, displacement and replacement of the pulmonary or hepatic parenchyma by bladder forms produce severe effects upon the intermediate hosts.	
Gid (Sturdy)	Larval stages of the tapeworm called *Coenurus cerebrealis* developing in the brain and spinal cord of sheep and goats. Intermediate hosts are sheep and goats Definitive host like dogs harbour the tapeworm *Multiceps multiceps.*		A disease marked by growth of the larval form called *Coenurus cerebralis* in the brain and spinal cord of sheep and goats. These animals suffer from incoordination and paralysis. The larval forms migrate through different tissues of the body and finally localise in the brain and spinal cord of the intermediate hosts. The cyst in the brain has scolices (upto 500) and contains clear fluid. It is also lined by a germinal layer capable to form scolices. The symptoms of this disease in the nervous tissue depend upon the damages done to pressed nervous tissue or destruction of the nervous tissue by the developing *Coenurus cerebralis.* In short, the tape worms consume the nutrients or food required by the definitive hosts like dogs and but their larval forms involve nervous tissues. Cause necroses and loss of function and	1. Detection of ova and segment of adult forms in the faeces of the definitive hosts 2. Histopatholo gy of *Coenurus cerebralis* in the affected brains of the intermediate hosts like sheep and goats.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			displacements of cells and tissue in organs like brain and spiral cord etc. in the intermediate hosts.	
Cerebrospinal nematodiasis (lumber paralysis or kumri) .	Filarid worms of the genus *Setaria* e.g., *S.digitata* *S.digitata* is frequently noticed as an adult worm in the abdominal cavity of cattle. The adult worms and their microfilariae do not cause serious ailment in the usual host like cattle.	Sheep, goats, horses as its aberrant hosts.	This disease called kumri is caused by larvae of *S.digitata* in animals like horses, sheep, goats and man etc. Blood sucking mosquitoes are its intermediate hosts. They suck blood of horses, sheep and goats etc., and spread infection to them. The main signs are paresis, weakness or blindness, incoordination and paralysis of the hind limbs. Endophthalmitis, acute focal encephalomalicia haemorrhage, axonal swelling, loss of myelin and degenerative changes are caused by larval forms in the central nervous tissue of the unusual or aberrant hosts. Adult worms may even be found in the eyes of bovines.	1. It is based on the symptoms and the detection of immature forms of *S.digitata* in the stained sections of the brain of animals like horses and sheep etc.
Pulmonary nematodiasis (Dictyocaulosis) It is also called lungworm disease (a kind of verminous pneumonia). *D.viviparus* is frequently noticed in the trachial exudate of affected cattle in India.	Three important species are :- 1. *Dictyocaulus viviparus* 2. *D.filaria* 3. *D.arnfieldi*	1. Cattle, buffaloes, deer and camels. 2. Sheep and goats 3. Horses and donkeys.	Lungworms lay the eggs in the bronchi in which hatching may also take place. The 4th stage larvae after their development enter the pulmonary artery or lymphatics and ultimately reach the bronchioles and bronchi to be matured. The main signs are laboured respiration, anorexia, diarrhoea and stunted growth. Pulmonary consolidation, occlusion	Lungworm larvae or eggs in the faeces of patients ova or larvae in the bronchi or bronchioles exudate of and larvae in the lung parenchyma are the bases to diagnose the lungworm disease in the animals.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			of the bronchioles and alveoli and secondary bacterial infections are seen in the lungs. The larvae damage the alveolar walls and capillaries and cause haemorrhage and necrosis in the lung parenchyma. Granulomatous reaction occurs around the dead larvae behaving like foreign bodies in pulmonary parenchyma. The bronchial epithelium proliferates and lymphocytes and eosinophils infiltrate the bronchial walls and peribronchial tissues.	
Spirocercosis Dogs, fox, cat and wolf are its susceptible hosts. It is often seen in dogs in India	*Spirocaca lupi*		Spirocercosis - It is caused by ingestion of coprophagous beetles harbouring the infection i.e., the 3rd stage larvae of *S.lupi*. The beetles ingest embryonated eggs in the faeces of the final hosts (like dogs) and get infected. The main signs of spirocercosis are as follows :- 1.Vomiting owing to obstruction arising from nodules in the oesophageal wall. 2. Haemorrhages due to rupture of nodules in the aorta or alimentary tracts. The main lesions are as follows :- 1. Nodules in the walls of aorta, oesophagus and stomach etc. 2. Saccular dilatation of the affected aortic walls. Aortic rupture may lead to fatal haemorrhages in the	It is based on the symptoms, lesions and detection of embryonated eggs in the faeces of the infected hosts.

Disease	Causes	Animals Affected	Signs and Lesions	Diagnosis
			affected dogs. 3. Nodules in the oesophageal wall with partially projecting *S.lupi* in its lumen. 4. Development of malig neonplasms like oesteosarcoma or fibosarcoma in the oesophagus affected dog with *S.lupi*.	

Chapter 6
Deficiency Diseases

Various deficiency diseases in animals arise from wants or deprivations of several vitamins or mineral substances. Even improper supply of certain amino-acids in the animal feeds produce diseases in bodies of individuals. If an animal does not ingest a single nutritional factor, it can suffer from particular disease or deficiency e.g., goiter due to deficiency of iodine or anaemia arising from the lack of iron in animal feeds. A disease due to single factor in the animal is noticed in animals e.g., anthrax due to *Bacillus anthracis* infection but complex or mixed diseases are always noticed in animals due to deficiency of nutritional factors. For example, vitamin A deficiency causes coryza superimposed with infection of organisms. An excess of vitamins e.g., A and D in feeds cause toxic conditions in animals known as hypervitaminosis A and hypervitaminsis D respectively. The feeds given to birds and animals should be completely balanced in respect of minerals, vitamins, proteins, fats and water etc. A diet low in phosphorus causes hypophosphetaemia in animals. Pica (depraved appetite) is an example of this condition. In short, deficiency of one or more nutrients, interference with intake and absorption, capacity of storage or utilisation of nutrients and increased excretion or proper retention of nutrients are responsible for producing diseases in animals.

Below are the main pathological signs and the lines of treatment to be followed by the veterinarians and practitioners are given in table 26. Curative and preventive dosage in avitaminoses and mineral deficiences affecting animals are presented in table 26b.

Table 26 : Deficiency Diseases of Animals, their Pathological Science and Treatment

Deficiency Diseases	Pathological Signs	Treatment
1.Hypovitaminosis A Vitamin A_1 (retinol[1]) and vitamin A_2 (retinol[2]) are its two forms. Vitamin A occurs in the form of carotene (a yellow pigment with chlorophyll in green plants).	The main signs are : 1. Night blindness (nyctalopia). Inability of the animal patients to see in the partial darkness. The light bleached rhodopsin (visual purple) is not restored to its previous status due to its deficiency. Presence of dryness of the eye due to deficient lacrimal secretion. Thickening and sqamous metaplasia in the lacrimal ducts lead to ductal blockage. Conjunctivitis and keratitis present in the cattle and horses and Opacity, infection and ulcers occur in the eyes of the patients. Loss of reproductive efficiency. Deficiency signs usually appear in animals. Other signs in animals include failure of growth in the young animals, improper development of the bones and nervous system, degenerative changes in the various glands and kidneys. Sterility may supervene in male and females affected with its deficiency. Atrophy, metaplasia and necrosis affect the respiratory and digestive tract mucosae. Urinary obstruction is present in cattle, sheep and goats suffering from vitamin A deficiency which also precipitates urolithiasis (renal culculi or stones etc.). Presence of the cornified epithelium in the vaginal wall. Foetal malformations may occur. Piglets are born without eyes. In hypovitaminosis A. Diets deficient in viatmin A or defective absorption of this vitamin from the gut causes its deficiency. In short, the main signs of vitamin A deficiency are as under : (nyctolopia) 1. Night blindness due to interference with regeneration of visual purple. Vitamin A is a must for preventing dim-light vision. It is needed for proper function of both rods and cons. 2. Corneal keratinisation, and dryness of eyes (xerophthalmia) and atrophy of retina, defects in the hooves and loss of weight.	Since the tissue changes are reversible, the symptoms or lesions disappear with normal supply of vitamin A at the dose rate given below. See table-26b

Deficiency Diseases	Pathological Signs	Treatment
	3. Infertility and congenital defects in offsprings of the deficient dams. 4. Increased susceptibility to many infectious diseases. Depressed immune system. 5. An increase in cerebrospinal fluid pressure in calves causing syncope, incoordination and convulsions in them 6. Poor bone growth. Normal position and activity of osteoblasts and osteocysts is maintained by vitamin A. Incoordination of bone growth or defective moulding of bones present. Presence of distortion in the animal bone, constriction of optic nerve, ataxia and weakness are also seen. 7. Atrophy of the epithelial tissues, Secretory epithelial cells fail to divide and there is an excessive presence of stratified keratinised epithelial cells. Secretory epithelium is replaced by keratinized epithelium in the salivary glands and placenta etc. 8. Defective embryonal development. Congential defects in pigs and rats are seen. Congenital hypovitaminosis is seen in calves. Weakness, incoordination and blindness with dilated pupils are the changes noticed. Retinal dysplasia and constriction of the optic canal may also be present. 9. Focal necrotic hepatitis, squamous metaplasia of the epithelium in interlobular ducts of glands e.g., salivary glands is a marked feature of this deficiency. The pathognomonic lesions of vitamin A deficiency include sqamous metaplasia and hyperkeratosis of mucous glands opening into the oesophagous and pharynx and nodule formations in their mucosae. Hyperkeratinisation of the epithelium of prefuce, rumen and reticulum are some other important changes. Defective formation of dentin and enamel.	

Deficiency Diseases	Pathological Signs	Treatment
2. Hypovitaminosis B. Vitamin B includes chemical compounds as given below : 1. Thiamin (vitamin B_1) 2. Riboflavin (vitamin B_2). 3. Nicotinic acid (Niacin) 4. Pantothenic acid 5. Pyridoxine Vitamin B_6 6. Vitamin B_{12} (cyanocobalamin) Vitamin B_6 Cobalt is needed for bacterial synthesis of vitamin B_{12}. Vitamin B_{12} requires an intrinsic factor (a protein produced by the gastric parietal cells) for its absorption in the gut.	Diseases like avian curly toe-paralysis, canine pellagra and chronic arthritis are caused by vitamin B deficiency. Paralysis and myelin degeneration are noticed in birds. Cyanosis of the distal portion of tongue, petechiae or haemorrhages in the digestive canal mucosae, haemorrhagic gastritis and enteritis, ulcers in the mouth, myelin degeneration, diarrhoea and ulceration of the buccal mucosa are the signs of canine pellagra (or blue tongue). Pigs suffer from chronic enteritis, (diarrhoea), anorexia, stunted growth and necrotic patchy mucous colitis. Bovines do not suffer from its deficiency because of its manufacture by ruminal bacteria. Horses do not suffer from its deficiency. In experimental cases of thiamin deficiency, pigs show focal necrosis of atrial myocardium with flabbiness and dilatation of the heart. Microbes in the presence of adequate amount of cobalt play a role in its synthesis in intestines of herbivores. Deficiency of vitamin B1 and B2 produce severe aliments in birds. Adult ruminants and horses suffers from thiamine deficiency because of consumption of plants containing thiaminase.	See table. 26b
3. Hypovitaminosis C. Man, monkey and guinea pigs need vitamin C in their diets. It is present in large quantities in animal and plant tissues. Birds and several types of animals synthesize it in their livers.	In animals, only monkeys or guinea pigs suffer from its deficiency. Lesions like subperiosteal haemorrhages, failure of the proper ossification of long bones, defective dentin formation and hyperkeratotic dermatitis are noticed in the animals. Ordinarily, the domestic animals do not suffer from its deficiency. Poultry does not need vitamin C in its feeds.	See table. 26b
4. Hypovitaminosis D Its important components are : 1. Vitamin D_3 (cholecalciferol) It is produced by sunlight in the skin. Vitamin D_2 is an ergocalciferol.	Leg weakness in chickens. Rickets in the young animals.,decrease in appetite, poor and bending of bones and enlargement of joints are seen in vitamin D deficient animals. Bones become fragile and are prone to multiple fractures. Vitamin D needed for mineralization of osteoid and epiphysel cartilage and its deficiency leads to resorption of bones.	See table. 26b

Deficiency Diseases	Pathological Signs	Treatment
5. Hypovitaminosis E This Vitamin (alphatocophevrol) occurs in the wheat germ oil. Tocopherols have antioxidant activity and prevent autooxidation of unsaturated fatty acids.	This deficiency produces white muscle disease in lambs. White muscle disease is a degenerative in nature causing appearance of white streaks in the muscles. A weakness of the limbs develops in patients. Retarded development or death of the embryo in the rodents, necrotic changes in the seminiferous tubules, aspermatogenesis, Zenker`s necrosis and degenerative changes in the muscles are the main findings in cases of experimental vitamin E deficiency in rodents. Yellow fat called ceroid which stains red by acid fist technique, is noticed at the interstices of the adipose tissue (cells) and in phagocytes and Kupffer cells of the liver etc. Such pathological state develops in pigs feeding with fish offal and seems to be connected with vitamin E deficiency.	See table 26b
6. Hypovitaminosis K Its two forms are vitamin K2 (active form) and vitamin K3. Vitamin K is required for normal blood cloting Excessive haemorrhages are seen in vitamin K deficiency.	As this vitamin is produced by bacteria in the normal animals they do not suffer from its deficiency. It is required for proper formation of prothobin and clotting factors VII, IX and X Anorexia, weakness, marked decrease in prothrombin are signs of vitamin K Deficiency. Vitamin K deficiency is associated with a haemorrhagic disease in pigs which fail to grow, become pale and also show subcutaneous haemorrhages. Vitamin K is a powerful anti dote to sweet clover poisoning in the animals.	See table 26b
7. Iron deficiency Iron is a cofactor in cytochromes, catalase and ferredoxin and forms an essential part of haemoglobin.	There is a need of iron for proper formation of haemoglobin and the body gets it by absorption through the intestine. Iron deficiency leads to anaemia and failure to thrive properly in the animals. In young pigs, diarrhoea, lack of hydrochloric acid (achlorhydria) and destruction of red cells lead to the state of anaemia. When it combines with calcium phosphate in bone, the disease produced is called osteomalacia or rickets. A little amount of copper and cobalt is required for normal haematopoiesis. Suckling pigs (1 to 8 weeks of age) affected with piglet anaemia are pale, emaciated, unthrifty anaemic and stunted in growth. At autopsy, oedematous lungs, dilated heart, enlarged liver, pericarditis, pneumonia, haemorrhagic gastritis and enteritis are noticed in bodies of the dead pigs. Diarrhoea is a common sign in piglet anaemia. Affected piglets also show dyspnoea, lethargy, pale mucosae and pigs deficient in iron show a high incidence of still births. Mottling of liver, fatty infiltration and decrease in the parietal cells are some other changes in the iron deficient persons.	See table 26b

Deficiency Diseases	Pathological Signs	Treatment
	There is a fall in values of red cell count and haemoglobin percentage (1 to 2 gm. per 100 ml of blood) in the affected piglets. In general, the pathologic changes in the anaemia are as follows: 1) Fall in red cell count (less than a million per cubic millimeter of blood), and presence of anasarca in the body. 2) Pale red cells due to haemoglobin deficiency 3) Presence of anisosytes, poikilocytes, polychromasia, reticulocytes and nucleated red cells in the blood smear 4) Oedematous and haemorrhagic mucous membrane. 5) Pale organs. 6) Haematopoiesis (regenerative changes) in the bone narrow seen postmortem. Replacement of fatty marrow by red marrow. 7) Punctate basophilia is present in chronic lead poisoning and anaemia of toxic origin. 8) Presence of megaloblasts in man affected with pernicious anaema due to deficiency of liver factor or anti-anaemic factor i.e., vitamin B_{12} (cyanocobalamin).	See table 26b
7. Copper deficiency Copper is a part of ceruplasmin and enzymes like superoxide dismutase cytochrome, oxidase and tyrosinase.	Most of the tissues contain copper, 20 to 30 mg of copper is present in one kg of the dry matter of the central nervous system. Its deficiency leads to anaemia and unthriftiness in the animals.For example, steely wool is noticed in the copper deficiency affecting the sheep. Enzootic ataxia (sway back) is an other disease seen in sheep due to copper deficiency. The lesions or signs noticed in the lambs i.e., in the patients of enzootic ataxia are the following : 1) Demyelinization of the central nervous system in neonates and necrosis, softening and cavities in the cerebral white matter in the brain containing translucent, greyish white material. 2) In coordination and weakness of the posterior limbs, following lesions due to copper deficiency is noticed in cows which fall and die all of a sudden. Diffuse fibrous scarring of heart, haemosiderosis of the liver, spleen and kidneys. Unthriftiness and aneamia and osteoporosis are some effects of copper deficiency. Diarrhoea, emaciation, lameness and changes in the hair are some other signs of copper deficiency in animals. Villous atrophy of the mucosae of the small intestines causing diarrhoea in patients is noticed. Flabby heart, pale colour, sudden death and atrophied muscle fibres with replacement by fibrous tissue are seen in falling disease noticed in animal suffering from copper deficiency.	See table 26b

Deficiency Diseases	Pathological Signs	Treatment
9. Cobalt deficiency Cobalt present in the soil and herbage etc. and 4 percent of Vitamin B_{12} is constituted by this substance.	Cobalt is required for proper activity of the ruminal bacteria and normal production of vitamin B_{12}. Its deficiency in the feeds causes deficiency of Vitamin B_{12} Its deficiency produces a cachectic syndrome (e.g. an enzootic marasmus) in sheep and cattle (bulls and calves) which show gradual loss of appetite, wasting away, death, anaemia and haemosiderosis in the organs like kidneys, liver and spleen etc. Fatty changes are present in livers of dead sheep and cattle. When the pasteur contains less than 0.0 + 7 mg/kg dry matter, cattle suffer from cobalt deficiency. Sheep consuming pasteur with cobalt less than 0.07 mg/kg dry matter also show signs of cobalt deficiency and show pallor of the mucosae. Animals are emaciated and easily fatigued. Growth, milk and wool production are badly affected. Decreased lambing percentage, increased rate of still births and increased neonatal mortality are noticed in the calves. Reduced concentraction of immunoglobulins and lower vitamin B_{12} level are found in the cobalt deficient dams. Lambs of such dams are slow to start sucking. Excess of haemosiderin in the spleen and liver with high levels of iron in the liver and spleen. Cobalt level is low in the liver of the deficient ruminants (e.g., cobalt content less than 0.07 mg/kg dry matter of the liver).	See table 26b
10. Manganese deficiency	It is required in small amounts for proper functioning of the body tissues. Its deficiency in the rats produce aspermatogenesis, retarded growth, weakness and defective offsprings. Perosis (i.e., the condition of slipped tendon) occurs in chickens and turkeys. In slipped tendon, there is a medial displacement of the gastrocnemius tendon. Deformed chicks with globular heads, short and thick legs and wings hatch from the eggs laid by the manganese deficient hens. Infertility, congenital and acquired skeletal deformities are some effects of this deficiency. Infertility problem in the animals (e.g., cattle) rise, in case, the soil contains less than 3 mg/kg of manganese. Manganese occurs as a cofactor in enzymes like arginase and phosphotransferase in animals bodies. It plays its role in bone matrix formation and also in synthesis of chordroitin sulphate which maintains the rigidity of the connective tissue.	See table 26b

Deficiency Diseases	Pathological Signs	Treatment
10 Zinc deficiency : Zinc is a component of the enzyme carbonic anhydrase.	Zinc in small amounts is an essential requirement for animals like pigs, sheep, cattle and goats etc. Thickening of the stratified squamous epithelium in the skin and oesophagus is produced by its deficiency in animals body. Zinc deficiency results in parakeratosis (i.e., retention of nuclei in stratum cornium in the pigs). One notices thickening in the stratum spinosum as well as the cornified layer. Hairs become sparse in the rats affected with experimental zinc deficiency. Alopecia, wool eating, abnormal hoof growth, lameness and unthriftiness are some other signs of zinc deficiency. Skin biopsy of the zinc deficient patients reveals a prominent increase in the thickness of all the elements of epidermis.	See table 26b
11 Iodine deficiency : It occurs in soil and water etc.	Man and animals require iodine in small amounts. Its deficiency causes goiter (i.e., enlargement of the thyroid gland) (Fig. 5; p. 200) Hyperplastic changes are noticed in the thyroid glands of cretins. Goiters are seen in the animals like lambs, pigs, calves and newly born colts etc. Iodine deficiency results in the birth of hairless pigs. If animals are not treated in time, they succumb to its deficiency. In the endemic zones of goiter, both the soil and water are deficient in iodine. An excessive intake of iodine leads to the state of iodism marked by lacrimationa and exfoliation of the epidermal scales like dandruff. Alopecia, enlarged thyroid and myxoedema are the common changes seen in iodine deficiency in animals. Hypoplastic hair follicles, delayed osseous maturation, absence of centres of ossifications, retarded foetal brain development, lack of wool growth and delayed skeletal maturation are some other signs of iodine deficiency.	See table 26b

Deficiency Diseases	Pathological Signs	Treatment
12. Phosphorus deficiency (aphosphorosis)	Inorganic phosphorus 4 to 8 mg per 100 ml of blood is found in the normal animals. Its imbalance or deficiency with calcium and vitamin D may lead to rickets or osteomalosia in animals (for example, cattle). A depraved or an abnormal appetite called pica develops in cattle due to aphosphorosis. Dirt is eaten by the affected animals. Phosphorus deficient cattle chew bones of dead animals (osteophagia). Rickets in sheep is mostly produced by phosphorus deficiency. In short, pica, poor growth, oestodystrophy and infertility are the main signs of phosphours deficiency. Weight loss, rough hair cat, lameness, fractures in the vertebrae, pelvis, ribs and porous, chalky white, soft and fragile bones are seen in the experimental cases of phosphorus deficiency in cattle. Such deficiency in cattle produces retarded growth, reduced fertility and low milk yield in cattle. Rise in cases of bolulism in cattle due to osteophagia is noticed. Pregnant cows become recumbent. Poor stature and perverted appetite are present in phosphorus deficient sheep and horses. Some other pathological changes in the phosphorus deficient animals are as follows. 1. Lighter bones showing a low ratio of ash to organic matter as seen in oesteomalacia. 2. Excessive deposits of osteoid in the adult bones of animals 3. Softer and large bone shafts due to subperiosteal osteoid deposits, enlarged joints, epiphyseal cartilage thicker than usual and the ash to organic matter ratio as 1:2 to 1:3 are noticed in the cases of rickets. In health, the ratio of ash to organic matter in bones is 3:2 in animals .	See table 26b
13. Sodium chloride deficiency	Deprivation of common salt in the feeds of animals causes anorexia, loss in body weight. Dairy cattle on a sodium deficient diet show polydipsia, polyurea, salt hunger, pica, drinking urine, licking dirt, a fall in milk production and loss of appetite and weight. Salt in the diet at al level of 0.5% is quite adequate for all farm animal species. Consuming inadequate amount of common salt causes poor growth, rough coat and unthriftiness in such animals. Men and horses loose sodium chloride through sweating. A cow can eat common salt upto 2 lbs. per day for an indefinite period without much ill effects. Animals may collapse and die due to salt deficiency. Excess of common salt in feeds causes salt poisoning in pigs and chickens.	See table 26b

Deficiency Diseases	Pathological Signs	Treatment
14. Magnesium deficiency : It is a cofactor in phosphotransferases and phosphohydrolases. Bones of animals contain about 7% magnesium. It is an activator of the enzyme alkaline phosphatase and many enzymes utilizing adenosinetriphosphatase (ATP).	Bovines contain about 2 mg of magnesium per 100 ml of blood. It plays an important role in the transmission of impulses at the neuromuscular junctions. An excessive formation of acetyl choline is favoured by low concentration of magnesium and the low ratio of magnesium to calcium. When the level of magnesium goes below 0.7 mg per 100 ml of blood, hypomagnesemia is noticed causing grass tetany in animals. The main changes of experimental hypomagnesemia in calves are as under : 1. Opisthotonus, muscle tremor and reluctance to move, nervous hyper irritability and apprenhensiveness. 2. Sudden convulsive seizures, scratching and kicking at the belly. Light stimuli produce twitching in the skin. Continual shifting of weight from limb to limb and incoordination. 3. Agonal haemorrhages in the heart, intestinal and mesentric serosae. Presence of thrombosis of the venules in the heart 4. Deposition of calcium in the internal layer of heart and large blood vessels. Hypocalcaemia and hypomagnesemia are noticed in cattle and sheep affected with hypomagnesemia. Agonal haemorrhages or convulsive death may be seen in the patients of hypomagnesemia. Deficiencies of copper cobalt, selenium, zinc, iodine and magnaese constitute important trace elements deficiencies.	See table 26b
15. Slenium and /or Vitamin E deficiencies. It is an essential element but highly toxic in large doses. It exists in the soil and certain seleniferous plants. It is a component of the peroxidase. Selenium requirement in the diet for most species of animals is 0.1-0.3 ppm of diet (mg/Kg) Toxic level of selenium varies from 2-10 ppm in the feed.	Either selenium or vitamin E or both may cause or be associated with development of many diseases in animals like pigs, sheep, cattle and horses etc. Role of selenium and vitamin E is interlinked in either causing a disease in an animal or in the treatment of certain ailments produced. When these nutrients are given to animals, they recover from the maladies. An inter-relationship between selenium, vitamin E and sulphur containing amino-acids has been marked in preventing many nutritional diseases in animals. The main diseases associated with the deficiencies of either selenium or vitamin E or both are given below in Table- 26a	See table 26b

Table 26A. Pathological signs and lesions in animals

Deficiency diseases	Animals affected	Pathological signs
1. Nutritional muscular dystrophy.	Cattle, sheep, pigs and horses.	Grey or white localized focal areas in the skeletal muscle and diaphragm due to degenerative changes in the patients of nutritional muscular dystrophy. The affected muscle, looks like fish flesh. Some grayish streaks may involve group of muscle fibers. The affected muscles are oedematous, friable and calcified. Sub-endocardial white areas of degeneration are noticed in the heart of the affected sheep and cattle. An extension of such lesions may lead to cardiac hypertrophy, pulmonary congestion and oedema, calcification of the dystrophic tissues and coagulation necrosis and hyaline degeneration in the affected muscle fibers. Yellowish brown fat deposits causing discolouration in the muscles is noticed in older animals.
2. Retained foetal membranes.	Cattle	The role of selenium deficiency as a cause of retained foetal membranes is doubtful. The findings on the inferior reproductive performance are also inconsistent. There is need of further research work in this direction.
3.Hepatosis diaetetica : Note:- Resistence to infectious diseases (e.g., mastitis) increases with maintenance of normal level of vitamin E and selenium in the diet of the animals. Animals fed with diet low in vitamin E and selenium show the following phenomena : 1. Immune response of animals is badly affected 2. Development of immunosuppression 3. Increased susceptibility to bacterial and viral infections 4. Poor antibody production or poor humoral immune responses	Pigs	The VESD syndrome (vitamin E and selenium deficiency) affects the growing pigs (3 weeks to 4 months of age). The pathological state of hepatosis dietetica arises from consuming a diet low in vitamin E and selenium. In Scandinavian countries (Sweden or Denmark), the disease occurs even naturally in pigs. Degenerative and necrotic changes are noticed in the organs like liver, heart, skeletal muscle and blood vessels etc. There is a degenerative myopathy of cardiac and skeletal muscles. Oedema, microangiopathy and

Deficiency Diseases	Pathological Signs	Treatment
5. Suppression of proliferation of T and B lymphocytes. One also finds cytodestruction of T lymphocytes and natural killer lymphocytes. 6. The performance of neutrophils is rendered poor. In other words, phagocytic role of neutrophiles markedly declines. Decrease in the phagocytic function of neutrophils is also noticed in the mammary glands. Administration of vitamin E and selenium to the animals maintained on deficient diets greatly improves the condition of these animal patients.		yellowish discolouration of adipose tissue are also seen in this condition. The affected pigs die suddenly.
Potassium (a part of sodium potassium pump for maintenance of proper cellular osmotic pressure).	Almost all domestic animals	The main signs or changes are as follows 1. Dehydration, chronic diarrhoea, hyperadrenalism, loss of appetite, rough hair coat, muscular weakness and paralysis in the deficient animals 2. Development of cellular oedema (the mildest and earliest degenerative change in parenchymatous organs due to failure of sodium potassium pump and poor milk yield by lactating cattle

Table: 26b. Curative and Preventive Doses used in Avitaminoses and Mineral Deficiencies in Animals

Deficiency Diseases	Minerals	Treatment	Prevention
1. Cobalt deficiency	Cobalt	Sheep : 1 mg/kg body weight per day infected orally Lambs : 300 mg per lamb at an interval of a month Cattle : 3 to 10 mg feed to each animal.	Sheep :0.1 mg/day to a sheep in feed. Cattle : 0.3 to 1.0 mg/day individually in feed.
2. Copper deficiency	Copper	Calves : 4 gms per calf per week (2-6 months) Cattle : 8 to 10 gms per animal at one interval for 3 to 5 weeks.	Calves : 1.5 gms at weekly interval in (2-6 months) feed. Cattle : 4 gms orally at weekly interval in feeds.
3. Iodine deficiency	Iodine	Therapeutic doses not recommended owing to its toxicity with body.	Lactating and pregnant Cows : 0.8 to 1.0 mg per kg dry matter of the feed. Dry cows & calves : 0.1 to 0.3 mg/kg dry matter of the feed.
4. Iron deficiency	Iron	Elemental Iron : 0.5 to 1 gm one injection once each week in large animals like horse, Vitamin B_{12} (cyanocobalamin) 5000 ug per week in a single dose to large animals like horse. It is better to follow the manufacture's guide.	Ferrous sulphate : 1.8 per cent 4 ml daily given to animals.
5. Sodium chloride deficiency	Sodium chloride	Farm animals : 0.5 of sodium chloride in the feeds per day.	
6. Magnesium deficiency	Magnesium	Pigs : 400-500 mg/kg of the total per day.	
7. Zinc deficiency	Zinc	Goat : Zinc sulphate 250 mg/kg daily for 4 weeks in the feeds Sheep : Zinc Oxide 200 mg given i/m in olive oil Lambs : 50 mg given I/M in olive oil Pigs : Zinc Inj. 2 to 4 mg/kg body weight to be given parentereally daily for 10 days.	Zinc sulphate : 50 mg/kg of feed daily for pigs.

Deficiency Diseases	Minerals	Treatment	Prevention
8. Potassium deficiency	Potassium	Pigs, piglets and ruminants : 65 mg/kg body weight to be given in the diet.	
9. Vitamin E selenium deficiency	Selenium	Calves, goats & lambs : 3 mg selenium and 150 I.U. per ml of DL alphatocopherol @ 2 ml/45 kg body weight intramuscularly. Only one injection sufficient. E-Care-Se. Horse, cattle and Dogs : 1 ml per 25 to 50 kg body weight 1 ml per 25 kg body weight by intramuscular route.	
10. Calcium deficiency	Calcium	Parenteral administration of calcium preparations like calboral 25%, calcium borogluconate (25%) are used. H + C - 200 - 400 m by i/v and s/c route to animals. Calcium sandoz (10%) Dog : 25ml to be given by i/v route. Note : Daily requirements in gms of calcium, Phosphorus and vitamin D for cattle are given as below : Growing cattle Calcium Phosphorus Vit-D 15-30 12-23 500 i/v kg dry matter intake Pregnant and 17-34 13-26 - do - Lactating cows.	
		For each kg of milk, 2 to 3 gms of calcium and 1.7 to 2.4 gms of phosphorus are to be given as milk production requirement over and above maintenance rate. Ewes 0.3-0.5-2 0.28 - 0.37 250-300I.U. Lambs 0.40 0.27 150 I.U. Horse (adults) 0.3-0.8 0.20 - 0.51 6-8 I.U. per Kg body wt.	
11. Hypovitaminosis A		Calf : 440 IU/kg body wt. I/M in a quar solution. The main daily requirement of Vitamin A for all species is 40 IU by parenteral route.	Calf : 40 I.U. per kg body weight daily I/M.

Deficiency Diseases	Minerals	Treatment	Prevention
12. Hypovitaminosis B	Vitamin B	For all species Thiamine hydrochloride @ 5 mg/kg body wt. If needed, it may be repeated after 3 hours. Initial dose is given by I/V route followed by I/M injection for 2 to 4 days. Berin For large animals : 10 ml of Berin is given I/M for 5 to 7 days on alternate days to treat Vitamin B deficiencies in all species, the following treatment rate is recommended 1. Vibelan Injection : 10 ml to large animals 2 to 5 ml to small animals 2. Conciplex Inj. : 10 ml to large animals 2 to 5 ml to small animals 3. Neuroxin B_{12} : 10 ml to large animals 2 to 5 ml to small animals. All the above three products are given on alternate days for 5 to 7 days. Vitamin B_{12} (cyanocobalamin) is required at the dose rate of 20 to 40 ug daily be calves and goats. For pigs the dose is 10 to 50 mg of	For monogastric animals. Thiamine hydrochloride @ 30 to 60 ug per kg body weight parenterally with additional supply of yeast, cereals and grain etc.
		Vitamin K is required per tone of its feeds. In case of a ruemia, monogastric animals at the dose rate of 2 ug vitamin B_{12} parenterally.	
13. Hypovitaminosis K	Vitamin K	Treatment For all animals Dose : 3 mg/kg body weight parenterally as a single injection restores deficiency or prevents coagulation defects etc. As a preventive dose, 25 mg/kg of feed is given to all aniamls for 4 days 300-500 mg/vitamin K is given subcutaneously every 4 to 6 hours as anti dose to counter poisoning in horses.	
		Note : Different proportions of Vitamins and minerals are available in the market. It is better to follow the inoculation details for their use to avoid reactions and complications.	

Chapter 7

Pathologic Changes and Treatment of the Cases of Poisoning in Animals

Poisons are substances producing pathological changes in the body of the animals. Such substances enter the body through the digestive tract, by inhalation, through the skin or also by some other routes. Some poisons or toxins do not leave any detectable signs or lesions in the body of animals. Acute or chronic effects are produced in different organs in the patients of poisonings. The kinds of the poisons are as under.

(i) Poisons or toxins produced by the pathogenic organisms.

(ii) Poisons produced by abnormal metabolism in the body.

(iii) Extraneous poisons like phosphorus or carbon tetrachloride etc.

(iv) Poisons existing in the body of the plants.

Pathology

The main effects of poisonings are as follows :

(i) Necrosis or death of the cells or tissue coming in contact with the poisons e.g., acids or alkalies. Concentrated acids or alkalies kill the cells out right on their immedi-

ate contact. Poisons in mild concentritious produce degenerative changes in the cells.

(ii) Acute inflammatory changes in the affected organs are seen due to various poisonous substances.

(iii) Lesions in the organs excreting the poisonous substances e.g., the existence of inflammatory lesions during the elimination of the mercury through the colonic mucous membrane.

(iv) Degenerative changes in the organs like heart and kidneys etc.

(v) Bacterial toxins or plant poisons exert degenerative effects.

(vi) Interference with some nervous function as seen in cases of strychnine poisoning.

(vii) Petechiae or ecchymoses due to injuries to the endothelium of the capillaries. Poisons, toxins and viral proteins (capsids) increase permeability causing petechiae and haemorrhages in organs.

(viii) Haemolysis of red cells.

(ix) Depressing or destroying the haematopoietic tissue.

(x) Blocking of the enzyme systems in the body.

Pathologic changes in some cases or poisonings and their treatment of such cases are given in table 27.

The following facts are to be borne in mind while treating the cases of poisoning in animals :

(1) Stop any more ingestion of poison and its absorption through the skin.

(2) The residual poison of alimentary tracts should be removed. Emetics, purgatives and gastric lavage can be used.

(3) Plenty of water is used to wash the skin and animals thus, get protected from further absorption of the poisons.

(4) A teaspoonful of salt can be given in water orally to produce vomiting. Apomorphine 20 mg can be used to produce vomiting.

(5) It is also proper to neutralize the residual toxins in the body.

(6) Tannic acid can be used to precipitate the alkaloids. If irritants and corrosive poisons have been ingested, milk or egg may be used.

(7) In the cases of poisonings, principle of using chemical antidotes is very beneficial. This drug makes the poison half-life.

(8) Calcium preparation (calboral) can be used as universal antidotes.

(9) Activated charcoals which absorb poisons, can be used as slurry by dissolving 5 tea spoonfuls of it in 100 ml of water and this can be given by stomach tube. Sodium sulphate can also given by stomach tube.

Important details about poisons, their pathologic signs and lesions and the line of the treatment adopted are given in Table 27.

Table 27. Pathological signs and Lesions in some poisoning cases

Poisons	Pathologic Signs and Lesions etc	Treatment
1. Organic phosphates or phosphorus compounds (including insecticides, and defoliants e.g. Tetraethyl pyrophosphate (TEPP), parathion, malathion, carbaryl, demetron, dichlorvos and endosulphan etc.)	These poisons are anticholinesterases and the harmful effects are dependent on the ability to prevent or inhibit the action of the enzyme called cholinesterase. There is a marked decrease of cholinesterase in the red cells. Acetylcholine is left free to act continuously as there is no enzyme to destroy this substance. The main signs are : 1. Excessive salivation. 2. Twitching and fasciculation of muscles. 3. Ataxia and asphyxia. 4. Haemorrhages in the heart, lungs and gastrointestinal tract. 5. Swelling and degenerative changes in the axons and pulmonary congestion.	This compound is greatly used as insecticide and pesticide. It, thus, enhances the chances of poisoning. Atropine sulphate 0.25 mg/kg. B.wt. and 1 mg/kg body weight can be given respectively to cattle and sheep. Average total dose in cattle is 50 mg and half of these is given by i/v or through i/m route. This can be repeated at an interval of 4-5 hours. Calboral 200 ml can be given by i/v route. For dogs, Atropine sulphate @ 0.02 mg/lb body (average dose 1 ml) can be given by i/v route. Fluid therapy (5% dextrose) can be used by i/v route. Calcium (Sandoz) 4-10 ml can be given by i/v route in dogs. Sedatives such as largactil can be used in case of convulsion and excitement in the patients.
1. Chlorinated hydrocarbons as insecticides e.g., dichloro, diphenyl, trichloro ethane (DDT), chlordane, lindane (gamma isomer of benzene hexachloride), toxaphene, endrin, eldrin and dieldrin etc.	Poisonings are caused due to errors in mixing and application of the insecticides. Soft egg shells and loss in hatchability are seen in poisoned birds. The main signs are : 1. Nervous hyperirritability, spasmodic twitching and quiverings of the muscles (including those of eye lids). 2. Belligerency or frenzy or convulsions and stopping of the heart beat in systole. 3. Pressing the heads against hard objects.	In order to treat cases of poisoning from chlorinated hydrocarbons, skin should be washed with soap water. If the dogs have ingested such poisons, emetics (concentrated solution of the common salt) and gastric lavage can be used. For sedation, pentobarbitone may be given. Calboral 200 ml i/v is quite useful. Calcium sandoz 4-5 ml can be given through i/v route. Cold water pack should be used to reduce the body temperature. Coramine 1 ml i/v. or i/m can be used to improve respiratory circulation in dogs. 10 ml of

Poisons	Pathologic Signs and Lesions etc	Treatment
	4. Augumentation of symptoms due to sharp noise. Temperature upto 115°F. 5. Dyspnoea and cyanosis and grinding of the teeth. 1. Pulmonary congestion, oedema Nissl's degeneration and necrosis of neurons. Necrotic changes in the ganglia of medulla, cerebellum and brain stem. Increased cerebrospinal fluid. 2. Acute toxic hepatitis and acute tubular nephritis.	coramine can be given to improve the respiratory circulation in dogs. 10 ml of coramine can be given to large animals at the dose rate of 5ml/kg b.wt
3. Mercury or mercury compounds e.g., mercuric chloride, mercuric iodide, mercury vapour, methyl mercury and arylmercury etc.	1. Haemorrhagic gastro-entritis, and coagulation necrosis in the gastric mucosa. 2. Ulcerative colitis due to elimination of the mercury through the colonic mucosae. 3. Destruction of renal tubules resulting in uraemia and anuria. 4. Necrosis in jaw bone, anaemia, oedema, cachexia and paralysis of the limbs. 5. Hydropic degeneration in the salivary glands and renal tubules. 6. In the nervous system, degeneration, necrosis, increase of microglia, demyelination of the nerve tracts, encephalomalacia and myelomalacia are seen. Neuronal necrosis is seen in cerebral cortex. 7. Hyaline degeneration of Purkinje fibres and cardiac muscle fibers.	In order to treat the mercury toxiciation, animals should be given large amount of coagulable protein such as egg orally to be immediately followed by mild purgatives. Sodium thiosulphate 15-30 gms dissolved in 100-200 ml of distilled water can be given i.v. and this should be followed by oral dosing (30-60 gm at 6 hour intervals). Treatment should continue till recovery. BAL (6.5 mg/kg b.wt.) should be given every 4 hours. Supportive treatment such as use of astringent. It can be given orally to control gastroenteritis and fluid therapy should be given i/v to correct the dehydration in animals.

Poisons	Pathologic Signs and Lesions etc.	Treatment
4. Lead When animals lick or ingest paints containing lead, they get poisoned. Lead shots or fumes from burning storage battery, smelting works etc., can lead to a state of poisoning.	1. Anaemia in affected animals. Nucleated red cells and basophilic stippling (i.e., red cells studded with purple stained granules). 2. Increased fragility of erythrocytes, head pressing, hyperaesthesia, colic, ataxia, convulsions and distension of arterioles, venules and capillaries. Swelling, hyperplasia and pyknosis in the endothelial cells. Astrocytosis and demyelinating encephalopathy are seen.	In order to treat lead poisoning cases, magnesium sulphate can be given orally. Calcium versenate 10 mg/kg b.wt. as 12.5% solution can be used i/v along with Dextrose solution. Dogs can be given this drug at the rate of 25 mg/kg b/wt. i/v Dose should be repeated for 4 to 5 days. Siquil or largactil can be used for sedation of animals.
5. Strychnine	1. Intermittent tonic spasms initated by sound or noises etc. This poison gets bound to synaptic membrane and acts antagonistically to the action of glycine to produce this state of hyperirritability in the spinal part of reflex arc. 1. Failure of respiration due to spasms. No postmortem lesions except petechiae in organs due to anoxia. Urine collected from fatal cases of poisonings in dogs, shows toxic spasms in frogs after intraperitoneal administration.	Emetics like ampomorphine 20 ml i/v can be used and the stomach can be evacuated. The animals can be sedated using pentobarbitone largactil and siquil etc., gastric lavage can be given after anesthetization. Drugs like caffeine or narcotics should not be used.
6. Lathyrus (*L. sativus*). The poisoning due to this plant species is called lathyrism in animals and human beings. It develops from ingestion of the seeds over several weeks or months. A neurotoxin, β-N	1. A gradual increase in paralysis of the posterior limbs degeneration and disappearance of nervous are found in the spinal cord. 2. Paralysis of recurrent laryngeal nerve produces roaring in horses. 3. Blindness, torticollis and anaesthesia (skin) in cattle. 4. Rapid weak pulse and death occurring from	In order to treat the animals suffering from *Lathyrus satwus* poisoning, calcium salts. (Calboral) 200-250 ml i/v can also be given to animals.

Poisons	Pathologic Signs and Lesions etc.	Treatment
Oxalyl-L-α,β-diaminopropionic acid is found in the seeds of *L. sativus*.	respiratory paralysis. Pulmonary congestion and subepicardial haemorrhages. 5. Abortions and intrauterine death of foeus in pregnant animals. Contracted tendons and aplasia of the lower jaw in the offsprings. 6. Dissecting aneurysms of the aorta in turkeys.	
7. Fluorine It produces acute and chronic poisoning (fluorosis) in the animals on account of ingestion over a long period. 1-2 mg/kg of fluorine/day in feeds can lead to fluorosis. Pastures and feeds contaminated with airborne residues from aluminium factories or phosphate refineries can lead to the state of poisoning. Calcium fluoride and sodium fluoride can produce fluorois.	**Chronic fluorosis** The signs are : 1. Mottling and attrition of teeth. 2. Intermittent lameness and periosteal hyperostosis. 3. Demonstration of more than 6 ppm of fluorine in the urine of affected animals. 4. Hyperplastic pitting of the enamel and chalky areas in the teeth. 5. The bones in the patients are heavier and thicker than normal with reduction in the size of the marrow space. Metacarpals, metatarsals and mandibles show such changes. 6. Osteoporosis in seen in sodium fluoride poisoning. Fluorine to the extent of 1.5% can be found in the affected bones. 7. Lameness, anorexia, loss of weight, fall in milk yield unthriftness and intermittent diarrohea are found in chronic fluorosis. In acute cases of fluorosis, abdominal pain, convulsions, weakness,	Fluorine intoxicated animals can be treated successfully by using gastrointestinal sedative. Treatment to neutralize residual fluorine in alimentary tract is given Calcium salts can also be used I.V. Balargan (inj) or Baladona inj. Can be used as sedative. Doses of 30 gm of aluminium sulphate daily should be used as neutralizing agent. Calboral 200-250 ml I.V.can be given fluid therapy (5%) Dextrose i.v. should be used.

Poisons	Pathologic Signs and Lesions etc	Treatment
	lethargy and acute gastroentritis are seen. Death occurs from respiratory and cardiac failure.	
8. Cyanides Animals suffer from cyanides due to ingestion of cyanogenic plants. The cyanides prevent the intracellular oxidative processes.	1. Death may occur instantaneously. Convulsion, forthing and mouth gasping, respiration, dilated pupils, involuntary defaecation and micturition are seen in the patient of cyanide poisoning. 2. Bright red colour of the arterial blood (an important diagnostic sign). 3. No lesions in the organs in acute poisoning. 4. Ataxia, vomiting involuntary urination and defaecation, incoordination and unconsciousness are noticed in patients. 5. Bright cherry red colour in the blood and tissues due to formation of carboxyhaemoglobin. 6. Bilateral necrosis of cerebral white matter and cortical grey matter. 7. Almost no lesions in acute cases of poisoning. 8. Arrest of respiration but continuation of heart beat in cyanide poisoning is a marked feature of cyanides poisoning.	Cattle suffer from HCN poisoning due to ingestion of shorgum plant at certain stages of its growth (Hiwar pods and cyanogenic plant). If animals ingest such poisons, they can be given the following preparation by i/v route. i) Sodium nitrite-3 gm. ii) Sodium thiosulphate-15 gm in 250 ml (distilled water). If necessary, use of this drug may be repeated. Animal can be fed sodium thiosulphate at the rate of 30-60 gm orally every hour four to five times. Calboral or Dextrose saline can be used. Antihistaminics can be given. Supportive treatment may also be used. Purgatives can be given immediately. Exposure to sunlight can be prevented by providing shade to animals.

Poisons	Pathologic Signs and Lesions etc.	Treatment
9. Onions Cattle and sheep are sometimes fed unusable onions (i.e. even in the decomposed state) and a state of poisoning develops in them. The toxic substance (haemolysin) in onions is nprophyl disulphide which causes precipitation of proteins.	1. Haemolytic anaemia, haemolytic icterus and haemoglobinuria are found in animal patients. 2. Odour of onion can be marked in the breath, milk, urine and tissues etc.	Further access to the onion can be prevented. Haemostatics agents may be used parenterally. Haematinic drugs can be used to improve the haematopoiesis. Liver tonics and anti-allergic drugs can be used.
10. Selenium The soil may be rich in selenium content and the plants (seleniferous) growing in such soil have sulphurous odour and also contain selenium. Sodium selenite in dose of 10 mg can cause death in the lambs. Ingestion of seleniferous plants causes selenium toxicity in cattle and sheep etc.	Two types of poisoning are 1. Acute selenium poisoning. 2. Chronic selenium poisoning. Acute selenium poisoning 3. Halmorrhagic enteritis and proctitis and passive congestion in lungs and abdominal visera are noticed. 4. Acute toxic hepatitis and toxic tubular nephrosis. 5. Mucosae of the urinary bladder are inflamed and subepicardial haemorrhages are present. **Chronic selenium poisoning** 1. Staggering and pressing forward their heads are signs in equine patients. Great weakness, paralysis, dyspnoea and cyanosis are noticed in them. Death occurs form respiratory failure, impairment of hatchability in the hens and foetal malforations are noticed. 2. Falling hair in the region of mane and tail and hoof lesions are found in the affected animals like horses and cattle etc. 3. Encircling grooves parallel to the coronary band and laminitis. Cracks or inordinate growth of the toes and erosion of the joints.	Selenium in toxicated patients can be treated by giving ascorbic acid and copper sulphate orally at the dose rate of 1-2 gm orally.

Poisons	Pathologic Signs and Lesions etc.	Treatment
	4. Chronic passive congestion in the lungs and focal necrosis in the heart are noticed in the patients. 5. Oedema present in pericardial, thoracic and peritoneal cavities. Hydropic degeneration or necrosis in the liver, hyaline casts in the convoluted tubules, hyaline degeneration and fibrinoid necrosis of arterioles are found.	
11. Lantana poisoning *L.camara* is a toxic plant for domestic animals like cattle and buffaloes etc., The animals ingesting suffer from poisoning. The toxic components of this plant are lantadene A and lantadene B.	The main pathological changes are: 1. Icterus, hepatotoxic photosensitivitis (photosensitivity, dermatitis and pink nose) and inflamed reddened muzzles are found in cattle. 2.Cracks or ulcers in the skin, haemorrhagic gastroentritis, subcutaneous oedema and subepicardial haemorrhages are important changes in this plant poisoning. 3. Gall bladder may be distended. Proliferation of bile ductule in the liver and necrosis of hepatocytes more severely in the peripheral portions of the lobules.	In order to treat the cases of Lantana intoxicated animals, the following steps should be taken. Purgatives can be given immediately. Exposure to sunlight can be prevented by providing shade to animals prevented by providing sheds to animals. Liver tonics and calcium borol-gluconate (250 ml to 300m i/v) and antihistaminic drugs like Avil inj. or candistin inj. can be used 5% Dextrose saline is also beneficial on i/v administration.
12. Phenothiazine It has severe toxic effecs in domestic animals like horse .	The signs and the lesions are : 1. Haemolytic anaemia, haemoglobinuria, acute toxic hepatitis and nephrosis. 2. Dullness, depression and weakness in the animals and primary photosensivitity of skin can be found.	Use purgatives, liver tonics, fluid therapy and haematinics to treat the cases of phenothiazine poisoning.
13. Carbontetrachloride : It is an anthelmintic drug and causes sometimes acute poisoning.	1. Presence of acute or catarrhal haemorrhagic gastroentritis, diarrhoea, abdominal pain and loss of appetite. 2. Icterus and blood stained faeces may be present. Acute toxic hepatitis. Fatty changes and necrosis in the epithelium of tubules in the kidneys. Petechiae in different organs are also seen.	Carbontetrachloride intoxication leads to liver damage. In such cases, the treatment includes calcium and glucose therapy by I.V. route. Antiallergic drugs can be given

Poisons	Pathologic Signs and Lesions etc.	Treatment
	1. In chronic cases, cirrhosis develops in the liver.	by parenteral route. Supportive treatment such as liver tonics should be used. Purgatives may be also tried.
14. Phosphorus Accidential ingestion of fire works of phosphrous matches causes poisoning in the animals.	1. In per acute cases, coma, convulsions and central nervous depression are noticed. 2. Presence of nausea, vomiting, abdominal pain, icterus, fever, polydipsia and polyurea in the patients of phosphorus poisoning. 3. Degenerative changes in the liver and kidneys. In the liver, the hepatocytes are filled with fat. Fatty changes are seen in the heart and kidneys. Hydropic degeneration is found in the proximal convoluted tubules. Other changes are small atrophic spleen, hydrothorax, generalized oedema, haemorrhagic enteritis and ecchymotic haemorrhages in heart etc. In chronic cases of phosphorus poisoning, mandible and maxillae show necrotising purulent osteomyelitis or deformities.	An emetic or purgative should be given immediately. Copper sulphate (1%) solution can be given orally as an emetic in small animals. Supportive treatment includes the administration of astringents to soothe the gastroenteritis and parenteral electrolyte solution is given to relieve the dehydration in the bodies of patients.
15. Urea: Urea as a substitute for protein nitrogen is fed to cattle and cases of poisoning occur in animal due to ingestion of excessive amount of urea.	Ammonia released from urea due to action of enzyme urease causes symptoms of poisoning. The signs are ; 1. Twitching of the eye lids, tails and ataxia. Salivation and frothing seen at mouth. Slow pulse and respiration are noticed. 2. Heamorrhagic enteritis, acute toxic hepatitis and toxic tubular nephrosis are seen in the affected animals. 3. Congestion and neuronal degeneration in the central nervous system. Haemorrhages may be found in the lungs.	In case of urea poisoning, animals should be given 2.4% acetic acid solution or 2 - 4 liter or vinegar given orally. Antiallergic inj. cab be given. Fluid therapy or electrolyte solution can be used I.V. B-complex orally may also be tried.

Poisons	Pathologic Signs and Lesions etc.	Treatment
16. Salt : Salt poisoning is seen in pigs and poultry due to excessive ingestion of sodium chloride. Cattle tolerate larger doses of salt,in case,the water is available in plenty to these animals.	1. 0.9% Nacl kills the baby chicks. 2. Presence of hydrothorax and hydroperitoneum in the chickens. Nephritis is also found in them. 3. Blindness, hyperirritability and convulsive seizures are seen in salt poisoning in the animals. 4. Oedema in the tissues and body cavities. 5. Eosinophilic meningoencephatitis is quite pathognomonic for swines. Accumulation of eosinophiles can be found in the perivascular spaces of the brain.	In order to treat the case of salt poisoning, the toxic feed and water must be removed immediately Symptomatic treatment includes use of alimentary tract sedatives in gastro-enteritis, and provision of isotonic fluids when dehydration has occurred. In case of cerebral oedema, diuretics or hypertonic solution are injected parenterally. A balanced ration (0.5% sodium chloride) should be given to poultry and the feeding of old feed should be immediately replaced by fresh balanced poultry feed.

Chapter 8
Some Miscellaneous Pathological Conditions/ Diseases

Pneumonia

Inflammation of the lungs in man and animals is called pneumonia. There are several causes (e.g., viruses and bacteria etc.) of pneumonia. An acute inflammatory state of the lungs is called pneumonia whereas pneumonitis refers to an inflammatory disease of the lungs characterised by a chronic reaction in the form of proliferative changes in the fibroblasts and lining cells of the alveoli. Reticuloendothelial cells, epithelioid cells and septal cells take active proliferative or defensive role in the lungs.

In short, pneumonia refers to acute infectious inflammation of the lungs with **exudative changes** in the alveoli. Two main kinds of pneumonia are:

(i) Lobar pneumonia (croupous pneumonia).

(ii) Bronchopneumonia (lobular pneumonia).

In man, lobar pneumonia is found and characterised by inflammatory reaction at one point which spreads by direct continuity to other parts of the lungs till the whole or most of the lobes of lungs get involved in the extending inflammatory reaction. In bronchopneumonia, the infection spreads through the air passages i.e., there is an inflammation of the bronchi or bronchioles (bronchiolitis). Croupous pneumonia or lobar pneumonia occurs in the human beings and is caused by a pneumococcus called *Diplococcus*

pneumoniae. There is a characteristic accumulation of the exudate in the air passage and the affected part gets consolidated and the consolidation is more or less in uniform manner and is clearly separated from the surrounding healthy tissue by a line of demarcation as marked at autopsy. Croupous inflammatory changes are quite uncommon or rare in animals. The involvement of the whole lobe results from the fusion of lesions in the different foci. In large number of pneunmonic cases in animals, the alveolar exudate consists of cells like epithelioid cells or large cells with rounder central nuclei and extensive amount of cytoplasm. Lymphocytes are also found in such exudate.

There are four important stages in the lobar or croupous pneumonia in man and these are as follows:

(1) Congestion

The lungs are red, congested and swollen and the pieces of the lungs float in water. When cut, a blood tinged fluid escapes from the cut surfaces of the lungs. Microscopically, the alveolar walls have distended capillaries with red blood cells. Alveolar exudate may reveal a few red cells in some cases.

(2) Red Hepatization

The lungs look prominent and healthy and the inflamed parts in the lungs are well demarcated. The diseased pieces of the lungs are firm like liver in consistency with red colouration. The cut pieces of the consolidated lungs sink in water. Pleura in the neighbourhood of the inflamed part of the lung is also congested. Microscopically, the alveoli contain fibrinous exudate with numerous red cells, neutrophiles and desquamated cells. These inflammatory cells lie between the fibrinous strands. The alveolar capillaries are also congested and the alveoli are full of exudate. Such stage of pneumonia occurs in about 2 days.

(3) Grey Hepatization

The consistency of the diseased lungs is firm or solid and the lungs are grey in colour. The cut pieces of the lungs also sink in water and have dull appearance. A greyish purulent fluid escapes from the cut pieces on being compressed between fingers. Microscopically, the alveoli are not full of exudate and a little space between the exudate and alveolar walls is noticeable in light microscopy. The fibrin in the exudate contracts and forms a free mass in the alveolar spaces. The alveolar capillaries are less congested and the red cells disappear from the exudate with predominance of fibrinous meterial in the alveoli. In purulent pneumonia, neutrophiles are largely noticed in the alveolar exudate. In humans, the alveolar exudate is exclusively fibrinous in the infection of *Diploccus pneumoniae*.

(4) Resolution

In this stage, the solution of the fibrinous material is a well marked change in the alveoli and the cells in the alveolar spaces also disintegrate. The liquified fibrinous exudate is either expectorated or absorbed into the circulation. Repair of the damaged alveolar walls occurs and the lungs revert to the state of normalcy.

When the lungs fail to revert to normal state, diffuse suppurative softening, gangrene and organisation of the exudate (fibrosis) at later stages can be seen in the lungs.

Lobular Pneumonia (Bronchopneumonia)

When there is spread of the acute inflammatory reaction from air passage to the air sacs (alveoli) of the lungs, the state of bronchopneumonia (lobular pneumonia) is produced. It starts as a capillary bronchitis or bronchioilitis with involvement of the alveoli served by these air passages. In lobular pneumonia, the inflammatory changes are noticed within individual lobules and their associated bronchioles.

The inflammatory areas in the lobules assume a patchy appearance in bronchopneumonia. In equine rhinopneumonitis (a viral disease), monocytes and neutrophiles are noticed in the alveoli, bronchioles and interstitial tissue of the affected lungs.

Causative Factors

(1) Organisms of the genra *Pasteurella, Streptococcus, Haemophilus* and *Croynebacterium* etc.

(2) Inhalation of the irritant gases lowers the resistence or vitalitis of the lungs and the invasion of the lungs with many organisms is facilitated. Bronchopneumonia is seen in the aspiration pneumonia due to faulty drenching etc.

(3) Invasion of the lungs or bronchioles by parasites i.e., *Muellerius capillaris.* Larval stages of the nematodes like *Ascaris* spp. cause bronchopneumonia in animals. A fungus known as *Aspergillus fumigatus* also produces bronchopneumonia in poultry.

Pathology

The disease (i.e., bronchopneumonia) starts as capillary bronchitis or bronchiolitis and then it spreads to associated alveoli. Alveoli get consolidated and the plugging of the broncholes with exudate causes collapse of the alveoli in the lobules (lobular collapse). Later, catarrhal inflammatory changes take place in these collapsed alveoli. The infection can also spread from the bronchi to the alveoli of the closely related lobules.

Gross Changes

There is an irregular or patchy distribution of large or small areas of consolidation, collapse and emphysema in the parenchyma of the lungs. When the pieces of the lungs are squeezed, exudate escapes from the bronchioles. The feel of the lungs is not found uniform throughout the parenchyma.

The colour of the lesions varies from red to pale or grey and these may be solid to the touch and look more prominent (i.e. raised) than the adjacent normal areas of the lungs. Large areas of the lesions in the lobules are presented as confluent bronchopneumonia which resembles lobular pneumonia. The areas of the collapsed lungs are dark red, sunken in appearance and are surrounded by small zones of the raised emphysematous tissue. Such changes are observed in the lungs of pneumonic cases.

Microscopic changes

Catarrhal inflammatory changes are found in the smaller bronchial and associated air spaces. These bronchioles show denudation of lining epithelium and may be filled with catarrhal exudate which consists of red cells, leucocytes and desquamated epithelium. A little fibrinous material may be found in the exudate. Areas of collapse and emphysema are found in the stained sections of the lungs. In the collapsed areas, the alveolar walls are found approximated (i.e., very close to each other) and the capillaries are congested and more apparent. Ephysematous areas bordering collapsed alveoli are marked by over distended air spaces (alveoli) whose walls may be thin and look like delicate strands of tissue. Ruptures in the alveolar walls of such tissues can be noticed and ruptured alveoli intercommunicate with each other.

When the exudate is removed by absorption or expectoration, the lungs revert to the state normalcy. Bronchopneumonia may pass on to chronic stage characterised by fibrosis. Even suppurative or gangrenous changes can occur in the lungs due to pyogenic or saprophytic organisms in the dead and devitalised tissues.

The changes noticed in some types of pneumonia are given in table 28.

Table 28. Signs and Lesions in some Main Types of Pneumonia

Pneumonia	Signs and Lesions
1. Aspiration pneumonia (gangrenous pneumonia) It is caused by faulty drenching, regurgitation and aspiration of drugs or irritating, stomach contents into the trachea and bronchi etc.	1. loud breath sound or crackles into ventral portions of lungs (usually apical or lower portions of the lobes) the pulmonary lesions are white or black and emit foul odour 2. lesions of acute broncho pneumonia and toxaemic changes in organs of the affected animals. The pulmonary lesions are soft, dark green or brown colour in the dead animals 3. Cavitation due to growth of putrefactive organisms or saprophytes in the dead pulmonary tissues because of aspirated drugs or stomach contents. 4. Developments of gangrenos pneumonia in the lungs is marked by hyperaemic, haemorrhagic and exudative changes around the necrosed tissues in the lung parenchyma.
2. Interstitial pneumonia the causative factors are viruses, mycoplasma or members of psittacosis lymphogranuloma group.	1. Alveolar septa thickened by infiltrating lymphocytes, plasma cells, macrophages, serum or fibrin and increase of connective tissue elements 2. Hyperplasia of alveolar lining cells is a frequent finding 3. Macrophages and some free lining cells may be noticed in the alveolar spaces but neutrophiles are not usual components of the alveolar exudate.
3. Viral pneumonia	1. A serous and mononuclear reaction is a feature noticed in viral pneumonia. Nature of the exudates in alveoli (that is the kind of leucocytes or fluid material) is dependent on the species of the animals and the causative factors involved.
4. Hypostatic pneumonia It is usually seen in recumbent animal patients frequently as a kind of terminal pneumonia.	1. Oedema, congestion and pneumonic lesions of the lower parts of the lungs of dying animals (i.e., a long delayed death on the beds and the ground) gravity and porous nature of the lungs favour an accumulation of blood or fluid in the dependent portions of the lungs.
5. Verminous pneumonia (lung worm disease).	1. Chronic pneumonic areas noticed in the diaphragmatic portions of the lungs due to chronic inflammatory cellular reaction involvinc the giant cells of foreign body types etc. around the dead or degenerating larvae in the

Pneumonia	Signs and Lesions
	parenchyma with fibrous capsule around the lesions in the lungs. Areas of collapse as dark red sunken areas are surrounded by raised, pale emphysematous areas. 2. Worms or strongyles and eggs are noticed in the mucopurulent exudate in the bronchi. Devitalized portions of the lungs caused by parasitic invasions are favourable targets of pathogenic bacteria 3. Oedema, interstitial pneumonia, foreign body types of giant cells, emphysema and fibrin, mononuclear cells and granulocytes in the affected alveoli are some important changes of verminous pneumonia.

Diagnosis

It is based on the symptoms, lesions and isolation of the organisms from the diseased tissues in the affected animals.

Treatment/Management

After the diagnosis of pneumonia (bronchopneumonia) has been confirmed in animals by clinical examination, a systematic line of treatment is followed.

Pneumonia or pulmonary infections are caused different kinds of bacteria, viruses, fungi and parasites etc. The aetiology of pneumonia (i.e., viral or bacterial) should be first established in making choices of drugs. When the aetilogy of pneumonia gets established, a proper selection of drug can be made for quick recovery. However, treatment can also be done on the basis of presumptive diagnosis i.e., a diagnosis without knowing exact cause of disease by means of laboratory tests.

The steps of treatment are given below:

1. Administration of antibiotics and sulpha drugs such as terramycin or gentamycin or ampicillin or Vesadin (33.3%). Any of these can be used for 7 days.
2. Use of antipyretics or analgesics, such as Bolin inj. and Novalgin inj. etc.

3. Anti-histaminics (avil) or cortico-steroids may be used.
4. Antiseptic inhalation with Tr. Benzoin and oil of turpentine etc., can be used to clear the congestion in the respiratory tract. Add 2 to 3 ml of these drugs in 10 kg of water (hot) and cover the head of animal with blanket and make it to inhale the rising vapour. In young pups, vicks vapour can be used. When the animals recover from fever after a few days, supportive treatment is given to the animals. For this, B-complex injection, Liv. 52 or Belamyl injection can be used.

In the case of respiratory faliure and shock etc., coramine can be given to animals in the doses of 10 to 25 ml in horses and cattle and in doses of one to two ml in dogs. Artificial respiration should be given to small animals. Camphor oil (1:20) in doses (10-20 ml by s/c) can be given to animals. Sometimes, very good response has been seen from camphor therapy.

Hepatitis

Hepatitis is an inflammation of liver in the body of an animal or individual.

Hepatitis can be divided into the following types:

(1) Suppurative or infectious hepatitis.

(2) Non-infectious or toxic hepatitis.

Infectious Hepatitis

It can be caused by various etilogical factors (i.e., bacterial and viral infections). *Actinobacillus lignieresi* and *Mycobacterium tuberculosis* infections lead to hepatitis. In pygonic infections (for example, strangles, abscesses, septic metritis and ulcerative endocarditis etc.), the infectious or septic emboli reach the livers through the blood vessels and produce abscesses in their parenchyma. Pyaemic infections can reach the liver by portal vein from the intestines. If there

is a pyaemic phlebitis, the infection can reach the liver by direct extension. (e.g., inflammation of the unbilical vein). Infective biliary passages can also lead to pus formation in the liver. If there is a traumatic injury to the liver with contaminated objects from rumen or reticulum, abscesses are noticed in the liver. There is an infectious hepatitis in Rubarth disease, leptospirosis, viral infection of ducks (viral hepatitis). Infection reaches the liver by various routes which are as follows:

(1) Portal vein
(2) Hepatic artery
(3) Umbilical vein in the newly born animals
(4) Bile ducts
(5) Hepatic vein
(6) By direct extension or continuity from the adjoining infected tissues.

Toxic Hepatitis

It is caused by different kinds of toxins or poisons and can be divided into acute toxic hepatitis and chronic toxic hepatitis. The main causes are:

(1) Chemical Poisons

Copper, arsenic, phosphorus, mercury, chloroform and carbon tetrachloride etc., are examples of chemical poisons.

(2) Plant Toxins

Plants of the genus *Senecio* cause hepatitis in animals. Toxins produced by fungi (aflatoxin) set up toxic hepatitis.

(3) Metabolic Poisons

Toxic substances are produced in cases of gastroenteritis. Toxic injury to liver happens in many acute infectious diseases. Toxic substances enter the body in cases of pregnancy disease of ewes. Hepatic damages are also

produced by vitamin E and selenium deficiencies and excessive administration of follicular hormones.

Pathology

Gross Changes

The livers due to toxic effects are swollen or somewhat smaller than normal, lighter in colour (pale or yellow) and more red due to an increased amount of blood in the organ. There is an accentuation of the lobular markings in the liver. The centers of the liver lobules are congested (red) and the peripheries of the lobules are more yellow or pale due to degenerative changes like cellular swelling, fatty changes and necrosis. The coagulated necrotic cells may form a pale part at the centers of the lobules. In fascioliasis, the bile ducts are enlarged and look like prominent macroscopic rigid tubes to the naked eyes.

Coagulative necrosis is found in the cells of the liver lobules. The hepatocytes show pyknosis and acidophilic cytoplasm. Based on the position of necrosis, diffuse necrosis, focal necrosis, peripheral necrosis, midzonal necrosis and centrilobular necrosis and paracentral necrosis are certain types of the necrosis found in livers. In paracentral necrosis, the necrotic areas adjoining the central veins are found on one side of the lobules.

The animals affected with acute toxic hepatitis may die or the disease in them may pass on to chronic toxic hepatitis (cirrhosis). The term cirrhosis is derived from the Greek, Kirros, which means tawny or orange coloured. The main characteristic change in the liver is formation of tissue growth seen arising either from the portal connective tissue or from that around the venous outflow or both.

In cirrhosis, the liver cells are injured or destroyed and disappear from the lobules. It is followed by progressive proliferative growth of fibrous tissue as a kind of replacement

step. In short, it can be said that the toxins destroy the liver cells and induce proliferative changes in the connective tissue elements.

Cirrhosis can be classified into different types on the basis of distribution of the connective tissue elements and the main types are as follows:

(1) Multilobular Cirrhosis (older synonyms: atrophic cirrhosis, postnecrotic cirrhosis and Laennec's cirrhosis).

In this cirrhosis, the newly formed connective tissue is distributed along the interlobular branches of the portal vein. In short, there is interlobular proliferation of the connective tissue.

(2) Pericellular Cirrhosis

It is marked by arrangement of the penetrating connective tissue elements around the individual liver cells.

In cirrhosis of the liver, the connective tissue elements may be in the coarse and dense form or in more cellular or fine form. The connective tissue elements may also be seen encircling the bile ducts in the liver.

(3) Monolobular cirrhosis

In this, increased connective tissue elements are usually seen around individual lobules.

Causes

The main causes are:

(1) Toxins of metabolic, bacterial or plant origin and chemical poisons like carbon tetrachloride and manganese chloride etc. Toxins of plant origin also produce cirrhosis as caused by plants like lupin or ragwort in horses. Toxins in moldy feed or toxins absorbed in gastritis and entritis also lead to cirrhosis of the liver.

(2) Irritants in the biliary passages or concentrated bile can act like an irritant.

(3) Parasites

Liver flukes, migrating ascaris and ecchinoccus cysts are known to produce parasitic cirrhosis in the liver.

The monolobular cirrhosis and pericellular cirrhosis are associated with toxins of plant origin.

Gross Appearance

The liver is enlarged in the early stages of multilobular cirrhosis. Later, it is reduced in size owning to contraction of the connective tissue elements.The liver becomes firm in consistency and is difficult to be cut into pieces with knife and its surface bears a granular or nodulated appearance. The liver possessing nodules is called hobnail liver. Such cirrhosis causes reduction in size of the liver and is known as atrophic cirrhosis. The monolobular cirrhosis is hypertrophic in nature because the liver is enlarged and bears a fine granularity on its surface. Yellow brown colour due to fatty changes or green colour due to bile staining of the tissues can be seen in the liver parenchyma. Whole liver or only parts of it can show cirrhosis and many newly formed bile ducts in the portal spaces can be found in the stained sections. Large bile ducts may be prominent, thickened and tortuous in appearance.

Microscopic appearance

Course dense bands of fibrous tissue containing newly formed bile ducts are noticed in the stained sections of liver and these bands can encircle one to several lobules. The newly formed connective tissue elements are found along the interlobular branches of the portal veins. When these fibrous bands contract, depressed areas are formed on the surface of the liver. Degenerative changes like fatty changes and necrosis are found in the liver cells. Hyperplasia of the liver cells can be found with loss of normal architecture of the liver lobules. The interalobular vein (central vein) may not

have central position or may be peripherally located in the lobule. In monolobular cirrhosis, the fibrous tissue can be found either around small areas of the liver tissue or around individual lobules and contain newly formed bile ducts. Fine strands of fibrous tissue can be found encircling the individual liver cells in the pericellular cirrhosis.

Parasitic Cirrhosis

Now-a-days, the older terms are not in use and the classification based on etiology, morphology and functional state of the liver is quite satisfactory. The term cirrhosis can be considered synonymous with chronic toxic hepatitis.

Under the heading cirrhosis, the types of cirrhosis like parasitic cirrhosis, biliary cirrhosis, Glissonian cirrhosis and central cirrhosis can be considered. A brief description of these types is as follows:

Parasitic Cirrhosis

In fascioliasis, fibrous connective tissue elements are found around the bile ducts. Such fibrous tissue can also be noticed around one or more lobules of the liver. In the early stages of fascioliasis, the liver lobules show hemorrhagic tracts which are the paths followed by the migrating flukes. The effects of cirrhosis in the individuals are jaundice, ascites, chronic venous congestion and enlargement of the spleen. Haemorrhages can be found in the veins in stomach and intestines.

Porphyrinaemia and photosensitisation may be seen in the patients of cirrhosis. Jaundice arises from biliary cirrhosis in animals.

The migrating larvae of *Stephanurus dentatus* and *Ascaris lumbricoides* cause necrotic changes in the hepatic lobules where the latter show growth of fibrous tissue (cirrhosis).

Biliary Cirrhosis

This is characterized by chronic inflammation of the intrahepatic bile ducts. In animals, liver flukes produce just an encircling fibrosis in the immediate vicinity of the invaded bile ducts. In the sections of the affected liver, fibrous tissue is found encircling the various bile ducts lodged with parasites etc. (see Fig. 6; p. 200) Fibrosis spreads through the portal areas and around the lobules and several newly formed nonfunctional bile ducts are found in the biliary cirrhosis. Infiltration of inflammatory cells, like lymphocytes and monocytes can be noticed. Jaundice is almost always present in this kind of cirrhosis. The liver is found enlarged with a greenish hue and the surface of the liver is nearly smooth.

Glissonian Cirrhosis

In this, there is spread of the chronic fibrosing inflammation of Glisson's capsule. The fibrosis extends over a short distance beneath the capsule and is not considered to be a cirrhosis.

Central Cirrhosis

In this, fibrosis occurs around the central veins due to chronic valvular stenosis or incompetence. The fibrous tissue around the central vein does not show extensive spreading growth.

Treatment / Management

Hepatitis in domestic animals is caused by different animate and inanimate factors like bacteria, viruses, parasites and poisons etc. These factors can induce either acute or chronic hepatitis. After the cause and the kind of hepatitis have been diagnosed in animals, rational selection of drugs can be made.

Hepatitis in animals can show changes like enlargement of liver, chronic indigestion, dullness, clay coloured faeces,

icterus and anaemia etc. Such pathological signs are to be kept fully in consideration in treating hepatitis cases.

The drugs used for treating hepatitis are as follows:

(i) Administer specific drugs for treating diseases like fascioliasis, leptospirosis, babesiosis, anaplasmosis, theileriasis and poisinings etc.

(ii) B-complex with liver extracts such as livogen, Belmyl, Pepsid, Liverzet etc., can be used.

(iii) Fluid therapy (10 to 20% dextrose saline) can be used.

(iv) In presence of toxicity, calcium therapy can also be used such as calboral and mifex.

(v) Anti-allergic drugs such as Cortisone and antihistaminics etc., can be used and antibiotics may also be injected in febrile and diffuse condition.

(vi) In case of constipation, purgatives or laxatives can be used in animals.

(vii) A balanced diet with adequate amount of protein can be given to animals for lessening the danger of hypoproteinemia.

Last but by no means least, a complete rest is very essential for the patients of hepatitis in animals.

Gastritis

It means inflammation of the stomach or abomasum in the body of animals. Dogs and pigs commonly suffer from it. This condition is noticed in the diseases like braxy, canine distemper, rinderpest, haemorrhagic septicaemia and swine erysipelas etc.

Two types of gastritis are as under:

(1) Acute gastritis

(2) Chronic gastritis

Acute Gastritis

It may be of catarrhal or haemorrhagic type.

Causes

The main causes are:

(1) Arsenic, phosphorus and carbontetrachloride in cattle and sheep etc.

(2) Hot fluids, frosted foods and irritant vegetables.

(3) Bacteria, viruses and parasites.

Pasteurella multocida, rinderpest virus, *Erysipelothrix rhusiopathiae* and trichostrongyles cause gastritis in the animals.

Signs

Anorexia, vomiting and pain etc., are the main signs of the gastritis.

PATHOLOGY

Gross Changes

The gastric mucosae may be swollen, reddened and thrown into irregular folds. The mucosae can be found covered with mucopurulent exudate. There can be reddening and covering of the gastric mucosa with bloody exudate in acute haemorrhagic gastritis. The presence of extravascular blood on the gastric mucosa is an important sign of haemorrhagic gastritis.

Microscopic changes

The mucosa of the stomach is found congested and also shows numerous haemorrhages. The epithelium of the abomasum or stomach may be found desquamated. Some neutrophilic infiltration into mucosa and submucosa can be found. Lymphoid hyperplasia in the mucosae may form some nodules.

Chronic Gastritis

Acute gastritis can be followed by chronic gastritis or it may be chronic from the very outset. Parasites cause chronic gastritis in animals.

The main parasites are:

	Parasites	Animals
1.	Species of Ostertagia, *Trichostrongylus* and *Haemonchus*	Cattle
2.	*Gasterophilus* and *Trichostrongylus* spp.	Horse
3.	Ascarides	Puppies

Signs

Anorexia, vomiting, lethargy and anaemia are found in the affected animals.

Pathology

The mucous membrane is prominently thickened near the pylorus and elevation and folding of the mucosae can arise from epithelial hyperplasia in the fundic region of the stomach. Microscopically, the increase in the connective tissue elements is noticed in the sections of the stomach. Inflammatory cells are present in the lamina propria. The mucosa of the stomach is covered with viscid, adherent and mucopurulent material. The mucosa has reddish brown colour due to haemorrhages and the surface of the mucosa is irregular and granular in appearance. The surface epithelium is desquamated and the glands may be atrophied or cystic in appearance. In atrophic chronic gastritis, the mucosa of the stomach is thinner and smooth but in the hypertrophic form, the mucosa is thickened and prominent.

Ulceration of the Stomach

Ulceration in the mucosae of stomach is frequently found in calves and pigs. In many acute infective and septicaemic infection, small ulcers are found in the stomach. The covering of the mucosa is eroded and followed by haemorrhages. In chronic gastric catarrh, ulcers are found in the stomach. Ulcers are found in the stomach damaged by irritants and foreign bodies and, thus, get predisposed to the action of gastric juice and ulcers are finally formed in the mucosae.

The ulcers developing due to actions of gastric juice are called peptic ulcers which are round or oval and possess punched out edges. They are found towards the pyloric end of the stomach. In chronic ulcers, the fibrous tissue changes are seen around the ulcers. The ulcers are shallow or deep or may penetrate through the wall to form perforating ulcers. The ulcers assume a funnel shaped appearance and fibrous adhesions and peritonitis develop in the vicinity of the perforating ulcers. Excessive haemorrhages from the ulcers end fatally.

Treatment / Management

Gastritis in animals is also caused by different factors like bacteria, viruses, parasites and poisons etc. Abomastitis with many ulcers is found in theileriasis. In dogs, it is a common condition marked by vomiting.

The line of treatment is designated in a specific or non specific way. In absence of diagnosis about specific factors, a general symptomatic non-specific way of treatment is adopted. The drugs used for checking vomiting and gastritis are as under:

(i) Suitable antibiotics such Ampicillin, Gentamycin, Amikasin etc., can be used.

(ii) To check vomiting in dogs and cats etc., antiemetics can be used such as avomin, perinorm, stemetil and largactil etc.

(iii) Antacids can also be given in dogs such as diagene tab. gelusil, diagene suspension and triple carb etc.

(iv) In case of gastritis with fever, antipyretics can be used.

(v) To protect the life of animals from dehydration, the fluid therapy can be used.

(vi) To lessen the gastric irritation, emulsion of egg albumin and milk with ice can be fed to dogs.

(vii) As supportive measures, electral powder, B-complex with liver extract, enzyme etc., can be used.

In other domestic animals, it is better to find out the specific cause of gastritis and then rational specific drugs can be given to the patients.

Enteritis

It means inflammation of the intestinal mucosa caused by several factors. The main factors for causing enteritis are:

(i) Bacteria

Eischerichia coli, Salmonelle spp. Clostridum perfringens types B and C, *Mycobacterium tuberculosis, Proteus* and *Pseudomonas* spp. are important bacteria to cause enteritis in the animals. *E. coli* is known to produce a powerful enterotoxin to cause enteritis.

(ii) Fungi

Candida spp. is known to cause diarrhoea in young calves.

(iii) Viruses

Rotavirus, coranavirus, virus causing mucosal disease, viruses of rinderpest and bovine malignant catarrah are known to cause enteritis and diarrhoea in the domestic

animals like cattle. Villous absorptive cells are damaged or destroyed by rota virus and coronavirus in new born calves and transmissable gastroenteritis virus in piglets produces state of maladsorption which results in diarrhoea. These infections cause villous atrophy and loss of digestive and absorptive abilities.

(iv) Protozoa

Eimeria spp. and *Cryptosoporidium* infections are marked by enteritis or diarrhoea in cattle.

(v) Chemical Agents

Enteritis, diarrhoea, dysentery and abdominal pain are seen in the poisoning caused by various factors like arsenic, fluorine, copper, sodium chloride, mercury nitrates and molybdemum etc.

In many cases, gastritis and enteritis may occur together in animals.

Signs

The main signs of enteritis are as under:

(a) Diarrhoea, dysentery and abdominal pain.

(b) Dehydration and disturbed acid-base imbalance.

(c) Fever, septicaemia and toxaemia.

(d) Faeces are soft or like fluid in consistency.

There may be unpleasent smell in it. Blood, fibrinous material and mucus etc., may be present in the faeces. The faecal matter may be pale yellow or red or dark (melaena) and blood may be present in the faeces in such cases.

(e) The eye ball may show recession. Dehydration can be evident after 10-12 hours from the onset of enteritis.

(f) Presence of mucus in faeces in chronic enteritis. Emaciation and loss of weight can be seen in patients suffering from chronic enteritis.

Diagnosis

It is based on the symptoms, lesions and identification of the etiological factors in the faeces. Bacteria like *E. coli and Salmonella* spp. can be isolated from the faeces. Parasites can be found in faecal matter. There is severe diarrohea, serous atrophy of fat in the chronary grooves of the heart and bone marrow and loss of weight with normal temprature in oestertagiasis.

Treatment / Management

The condition of enteritis is recognised by diarrhoea (loose motion) and dysentery (loose faecal matter mixed with blood). There are diverse causes of enteritis and treatment of enteritis can be successful if causes of enteritis like rinderpest, H,S parasitic infection and Jhone's disease etc., are known.

Use of drugs acting selectively against the organisms or parasite ensures success of treatment.

The specific treatment of enteritis has already been dealt with under the heading of specific diseases.

However, the following general line of treatment can be given to treat cases of enteritis in the absence of diagnosis of causes.

(i) Use of Astringent powder such as Neblon powder, Didisco powder - 50 gm BID for 5 days.

(ii) Sulpha drugs or antibiotics can be used such as sulpha boulus or diadine bolus (3 boluses BID in large animals for 5 days). Steclin bolus or Terramycin tab. or Gastina tab. 4-6 tab. twice daily for 3-5 days can be used.

(iii) In case of dehydration, fluid therapy should be given.

(iv) In case of enteritis with fever, antipyretics are recommended.

If there is no recovery in the affected animals with enteritis, it indicates turning of acute enteritis into chronic

enteritis. In such situation, it is safe to findout exact cause of enteritis for deciding further line of treatment. In such cases, it becomes very important to rule out the chances of Johne's disease by examining smear of rectal pinch for the presence of *Mycobacterium paratuberculosis.*

Table 29. Astringent mixtures for enteritis (diarrhoea and dysentery)

R/

(1) Pulv. Creta prep.	Adult cattle	Calf.
	30 gm	10 gm
Pulv. catechu	30 gm	15 gm
	or	
(2) Acid tannic	15 gm	--
Kaolin	30 gm	15 gm
Pulv ginger	15 gm	5 gm

Give in gruel once in 12 hours.

Nephritis

Inflammation of kidneys in the body of animal or an individual is called nephritis. Several suppurative factors cause nephritis in animals. Nephritis has been classified in various ways by different authors but the types given below serve its purpose to a great extent.

Nephritis

A. Suppurative (infectious) nephritis
(a) Pyaemic or embolic nephritis
(b) Pyelonephritis

B. Non-suppurative(non infectious) nephritis
a. Acute Nephritis

I. Glomerulonephritis
II. Tubular nephritis
III. Interstitial nephritis
b. Subacute nephritis
c. Chronic nephritis
d. Nephrosclerosis

Inflammatory changes are seen in the glomeruli, tubules and interstitium of the renal tissue. Bacteria cause inflammatory reaction in the glomeruli and tubules etc. and several toxins extert their toxic effects on the kidneys during their excretion through the glomerular filter. The factors like intensity and duration of the lesions are used to classify it into acute, subacute and chronic types.

The details of the nephritis are as under:

Pyaemic Nephritis (Embolic Nephritis)

In the kidneys, the pyaemic nephritis is noticed as a sort of secondary infection i.e., the primary infection metastasing from the kidneys can be found in some other distant organs like uterus, heart or navel etc., in the body of the animals.

Causes

The main causes are:

(1) Bacteria such as streptococci, staphylococci and *Bact. equirulis* etc.

(2) Septic emboli, thrombi or vegetatious from the heart valves. When these septic materials reach the kidneys, the organisms transported in these infected tissues start growing in such organs. Such infected objects (e.g., bacteria in the stained sections of the kidneys) are arrested in the glomerular capillaries. Emboli can also be lodged in the intertubular capillaries and arteriolae rectae. Lesions in the medulla develop from impaction of the emboli in the arteriolae rectae.

Pathology

The lesions can be found in both kidneys which are found enlarged, congested and haemorrhagic. The renal capsules are easly stripped of from the parenchyma and numerous small abscesses are seen scattered in the renal parenchyma. The infectious agents are carried into the

kidneys through the circulation of blood and there is a heavy showering of the pyogenic bacteria throughout the renal tissue. The renal cortex shows the rich growth of the abscesses but the abscesses can also be found in the medulla. The abscesses are small, numerous and may be invisible to the naked eyes. The abscesses developing in the glomeruli are somewhat round in shape whereas those developing in the intertubular capillaries show grayish white enlongated appearance along the medullary rays. The lesions are usually of the same size and age and also originate from the same source due to raining of bacterial emboli.

The lesions consist of microorganisms and leucocytes, obliterating or replacing the normal renal tissue. In the stained sections of kidneys, the organisms look like solid mass or plug surrounded by a zone of neutrophiles.

Pyaemic nephritis ends fatally in the animals.

Pyelonephritis and Pyelitis

Inflammation of the pelvis or calices of the kidneys in animals is called pyelitis. When the inflammatory changes are present in both pelvis and renal parenchyma, it is called pyelonephritis.

Types and its causes

(A) Pyelitis

Its main causes are :

(1) Tuberculous infection and other bacterial infections like infections of *E. coli*, staphylococci, streptococci and corynebacteria. Irritation caused by calculi in the kidneys, ureter and urinary bladder predisposes the tissues to bacterial growth.

(B) Pyelonephritis

The causes of pyelonephritis are :

(1) *Corynebacterium renale*

This organism reaches the kidneys through the circulation (haematogenous or descending types of infection) and set up inflammatory changes in the pelvis. Pyelonephritis is common in cattle (mainly cows) and pigs and may develop as a primary infection. In cows, the infection can be of ascending types from genital tract following metritis or renention of the foetal membranes in the uterus. In short, infection to the pelvis of kidneys can either extend from the infected genital tract or reach the kidneys through the blood circulation (i.e., a descending type of infection). Use of contaminated surgical instruments also causes pyelonephritis and such pyelonephritis is called surgical kidney. A surgical kidney is an example of ascending infection.

Pathology

Gross Changes

In cattle, one or both kidneys are usually affected.

The main lesions are :

(1) Enlargement of the kidneys.

(2) Thick capsule adherent to the underlying renal tissue.

(3) Mottled surface of the kidneys (appearance of greyish white patches against the normal reddish brown colour of the kidneys).

(4) Abscesses and areas of haemorrhages.

(5) Dilatation of the calices with dirty greyish or brownish exudate. The exudate may be blood stained, slimy or mucoid fluid containing pus, necrotic material and conceretions etc.

(6) Greyish white streaks owing to extension of the suppurative processess.

(7) Destruction of the renal tissues bordering the calices. Pus can even replace much of the renal parenchyma.

The ureters in such cases are found thickened.

Microscopic Changes

In infectious of the kidneys, the uriniferous tubules are found dilated and filled with desquamated epithelium, leucocytes, cast and bacteria etc. A prominent leucocytic infiltration is found around the tubules and glomeruli. Necrotic changes are found in the renal tissues around the renal pelvis. There is also presence of haemorrhages and infarcts in the kidneys. There is a severe destruction of the renal parenchyma and interstitial changes are seen in the kidneys.

Pus, albumin, bacteria, blood and catarrhal cells are found in the urine of such patients of pyelonephritis.

Perinephric Abscesses

In this condition, abscesses are found around the kidneys. The suppurative processes may extend into kidneys. It differs from pyonephrosis in which abscess is found in the parenchyma of the kidneys.

Non-suppurative Nephritis

As the term implies, non-suppurative nephritis is caused by different factors other than the pyogenic organisms or non infectious agents and is commonly found in domestic animals and its occurrence is not common in carnivores.

The details of the different kinds of non-suppurative nephritis are as follows:

1. Acute Nephritis

In this, acute inflammatory changes are noticed in the kidneys. Any injury to the glomeruli results into tubular destruction as the blood supply to the tubules is dependent upon the normal functioning of the associated glomeruli.

Causes

The main causes are:

(1) Bacterial Toxins

Toxins are produced in pyaemia and strangles in the body of animals.

(2) Chemical Poisons

Turpentine, cantharides and carbolic acid are examples of such poisons.

(3) Harmful products of faulty metabolism.

(4) Extensive damage to the skin as seen in burns, mange and eczema etc.

(5) Injuries or Cold

Nephritis can be caused by such aforesaid factors and can assert itself in the exacerbated form with development of acute inflammatory changes in the kidneys.

Pathology

Gross Changes

Little or no change can be seen in the kidneys. The kidneys may be swollen and paler than normal. The kidneys are soft to the feel or touch and the capsules are very much distended and can be easily be striped off leaving behind a smooth renal surface.

Small haemorrhages are found in the renal parenchyma and the enlarged glomeruli are visible to the naked eyes.

Microscopic Changes

The histopathologic changes can be found in the glomeruli, tubules and interstitial tissue and the conditions produced are known as (1) glomerulonephritis, (2) tubular nephritis and (3) interstitial nephritis respectively.

The glomerual tufts are increased in acute glomerulonephritis and also show increased cellularity. The capsules of Bowman are distended due to enlarged glomeruli. Proliferative changes are seen in the endothelium lining the glomeruli and epithelium investing the tufts from the outside. Leucocytic infiltration can be seen in the glomerular tuft. Hyaline changes occur in the proliferated endothelium to form hyaline intracapillary thrombi. There is a little presence of blood in the glomeruli. The capsules of Bowman contain red cells, desquamated cells, fibrin and some leucocytes.

In acute glomerulonephritis, some changes are present in the tubules and interstitial tissue because of the dependance of the tubules for blood supply upon the normal functioning of the glomerular circulation. The efferent glomural vessels nourish tubular walls.

In the tubular nephritis, the main causes are poisons like phosphorus. Cellular swelling and necrosis are found in the tubular epithelum of the kidneys. Cell debris, desquamated and disintegrated cells choke the tubules and these substances can be moulded in the shapes of the tubules to form structures called casts. For example, epithelial cells form epithelial casts which can lead to formation of fatty, hyaline or granular casts due to degenerative changes. Haemorrhages may be present in the kidneys.

The pathologic changes in some important types of glomerulonephritis are given in Table 30.

Table30. Main Pathologic Changes in Some Types of Glomerulonephritis

Acute Proliferative Glomerulonephritis	Membrano Proliferative Glomerulonephritis	Chronic Glomerulonephritis	Focal Embolic Nephritis	Membranous Glomerulonephritis
1. An important condition noticed in children and some animals affected with streptococcal infections. 2. Enlarged pale kidneys with petechiae and occluded glomeruli 3. Enlarged glomerular tufts due to proliferated endothelial and mesangial cells of the glomeruli 4. Neutrophiles and monocytes infilterated into glomeruli and compressed glomerular capillaries lacking red cells. 5. Proliferated parietal epithelial cells (capsular epithelial cells of Bowman`s capsules). Presence of fat in the swollen tubular epithelial cells of the affected kidneys.	1. It is seen as a naturally occurring disease of animals. Increase of mesangial cells (a third kind of cells associated with the endothelium and epithelium of glomerular tufts). 2. Thickened mesangial basement membrane and splitting of the basement membrane. 3. Presence of immunoglobulin deposits (humps) on the subendothelial side of the basement membrane of the glomeruli. 4. Humpy and bumpy appearance of the disrupted basement membrane of the glomeruli seen in stained sections of the kidneys by silver staining method.	1. Kidneys smaller than normal with pitted surfaces 2. Occlusion of the lumens of glomerular capillaries and increased numbers of endothelial, mesangial and epithelial cells. 2. Proliferated epithelial cells forming crescents in the Bowman`s capsules. 3. Bowman`s capsules obliterated or adherent to occluded glomerular tuft. 4. Degenerative changes in the epithelium of nephrons, ischaemia, tubular atrophy and interstitial fibrosis.	1. it is noticed in the cases of bacterial endocarditis in man, cattle, swine and dogs etc. 2. Abscesses and petechuation in the kidneys 3. Proliferated epithelial cells (both parietal and visceral) forming crescents in the Bowman`s capsules. 4. Neutrophilic infiltration in the necrosed glomeruli and bacterial colonies lodged in the glomeruli.	1. it is noticed in man and animals 2. Thickening, splitting and reduplication of the glomerular basement membrane 3. Loss of foot processes of the podocytes (i.e., the epithelial cells of the glomeruli) 4. Irregular thickening of the glomerular basement membrane and its humpy-bumpy appearance.

In acute interstitial nephritis, the causes can be bacteria like leptospira. Interstitial tissue between the tubules undergoes proliferation and shows infiltration with nongranular leucocytes. The round cell infiltration can be found around the convoluted tubules and the Malpighian bodies.

Acute nephritis may lead to uraemia and the affected animals may die. There can be progression of acute nephritis into subacute or chronic nephritis.

Subacute Nephritis

It can occur as a primary condition. The affected kidneys are paler than normal and are also enlarged and their capsules can be easily taken off. The cut surfaces of the convoluted tubules (due to compression of the capillaries) also impart paleness to the cortical surface. The glomeruli show lobulation and shruken apperance. Bowman's capsules have proliferated cells and may also be hyalinised or thickened. The epithelial lining of the Bowman's capsule proliferates to form a cresentic mass in the capsular space. Cellular swelling, fatty changes and necrosis are found in the epithelial cells lining the convoluted tubules. Casts are formed in the tubules by the desquamated cells and debris. Interstitial tissues may show proliferated changes.

The animals may die of subacute nephritis and the disease may progress to chronic nephritis.

Granular Contracted Kidney

In this disease, there are marked proliferative changes of the connective tissue cells with destruction and disappearance of renal parenchyma. It may develop insidiously or may be sequel to acute or subacute nephritis. Nephritis developing insidiously is called primary contracted kidney and the other one sequel to acute type is called secondary contracted kidney.

Pathalogy

The kidneys are small, fibrous and pale in colour. The renal surface may be rough or granular and the capsule may be thickened and adherent to underlying renal tissue. The stripped capsule shows fragments of the kidneys leaving behind a rough surface. The cortex is very much narrow due to destruction and can be seen as a mere rind i.e., hard skin of the fruits. The medullary and boundary zones are wide and show cysts due to dilatation of the tubules. In dogs, proliferation of fibrous tissue is found in the medulla and boundary zones. In a form of chronic nephritis of dogs, the capsular adhesions are not the main changes and the medulla and its boundary zone shows fibrous tissue proliferation. The glomeruli can be found destroyed and there are crescents showing hyaline changes or may be replaced by the fibrous tissue in case of chronic nephritis. The increase of connective tissue between the tubules is a conspicuous change. The tubules may be dilated and form cysts like spaces due to compression of the tubules by the growing fibrous tissue.

The animals suffering from chronic nephritis ultimately die.

Nephrosclerosis

In this, there is a renal fibrosis due to an arterial disease (i.e., an arterioselerotic condition). Hypertension is found in man suffering from nephrosclerosis. Narrowing or constriction renal of vessels causes necrosis and subsequent fibrosis of the glomeruli. Necrosis follows obliteration of the vessels. Tubules show degenerative changes. Atrophy, fibrosis and necrosis are found alternating with areas showing little change owing to existance of some normal glomeruli and tubules. The kidneys present numerous infarcts in their parenchyma. The depressions on the surface of the kidneys are found due to contraction of fibrous tissue in the kidneys. The nephrosclerosis may occur with or without hypertension

in areteriosclerotic and senile contracted kidney respectively. An obstructed blood flow through the kidneys causes an increase in blood pressure. Ischemia in renal parenchyma causes increased formation of the enzyme called rennin. Rennin combines with hypertensinogen (a blood globulin) to form hypertensin leading to vasoconstriction and increase in blood pressure. Increased blood pressure is the cause of arteriosclerosis of intrarenal blood vassiles giving rise to nephrosclerosis – a condition usually noticed in human beings.

Pathological changes or abnormalities in urine:

Acute Nephritis

The main changes in the urine are:

(1) Oliguria (a diminished volume of urine).

(2) A high specific gravity.

(3) Presence of albumin.

(4) Presence of casts and blood in the urine.

(5) Reduction in the amount of urea and chlorides in urine.

Subacute Nephritis

The main changes in the urine are:

(1) Deficient urine i.e. less production of urine.

(2) Presence of albumin.

(3) Epithelial casts and blood cells in the sediments.

(4) Slight rise in the specific gravity or it may be normal.

Chronic Nephritis

The main changes in the urine are:

(1) Polyuria (increased quantities of urine).

(2) Decrease in the specific gravity.

(3) Albumin may be seen in the urine.

(4) There may be occasional presence of casts.

Treatment / Management

Nephritis in animal is caused by different factors, like bacteria, parasites and poisons etc. The signs of pain, dysuria, albumin or blood in urine, oedema, and uraemia warrant immediate attention of the clinician for the sake of treatment. For treating nephritis, cultural examination of urine is done to find out antibiotic sensitivity of organisms in question.

The treatment of nephritis is continued over a long period, say 10 to 15 days, for eradicating infection. Antibiotics such as streptomycin, gentamycin, nitrofuran and ciprofloxacin etc., can be used. Leptospirosis causes nephritis in dogs whereas pylonephritis (pyelitis and nephritis) are caused by *Corynebacterium renale* in cattle. These infections in such animals respond well to treatment in time.

The other drugs used in nephritis are as follows:

(i) Use of diuretics: Diuretics should be used with caution and also with object of eliminating toxic and waste metabolic products from the body. The patients of nephritis are marked by signs of ascites and oedema in their bodies. In cattle and dogs, lasix can be used to treat the complications like ascites or oedema.

Phenacetin in doses of 4 gm mixed with treacle can be given twice to horse and cattle as diuretics.

(iii) Fluid therapy and antiemetics can be given to animals (specially dogs) in case of vomiting and dehydration.

Oedema

It denotes the excessive accumulation of fluid in the tissue spaces, body cavities and cells etc., due to disturbance in the fluid interchange machanism between tissues spaces, capillaries and lymphatic vessels.

The main types of oedema are :

(i) Non - inflammatory oedema.

(ii) Inflammatory oedema.

The inflammatory and non inflammatory fluids are called exudate and transudate respectively.

Causes

These are as follows:

1. A decreased osmotic pressure of the blood.
2. Increased hydrostatic pressure in the capillaries.
3. Obstruction to lymphatic flow.
4. Damage to the capillary walls.

Decreased Osmotic Pressure (Hypoproteinaemia):

It is noticed in the following conditions:

1. Congestive heart failure
2. Portal hypertension due to hepatic fibrosis. It causes ascites in animals.

Compression of mammary veins due to an enlarged foetus. It causes ventral oedema in cows and mares in late pregnancy.

Decreased osmotic pressure also arises from the continued loss of blood.

It is seen in infestations with blood sucking parasites like *Strongylus* spp. in horse, *Fasciola* spp. in cattle, *Bunostomum* spp. in calves and, *Haemonchus* spp. in ruminants. Heavy nematode infestations also cause loss of proteins as seen in ostertagiasis.

Renal diseases cause the loss of proteins as seen in cases of nephritis in animals.

Gastroenteropathy is marked by loss of proteins as noticed in Johne's disease.

Liver damage results in failure of plasma proteins synthesis. Diets low in proteins cause oedema.

3. Obstruction to Lymphatic Flow

Tumours (e.g., lymphosarcoma in buffaloes), or inflammatory swellings, and granulomatous lesions lead to oedematous states like ascites and hydrothrax.

Inherited (congenital) lymphatic obstruction e.g. oedema of cross bred calves.

4. Vascular Damage to Small Vessels

The causes are as given below.

1. Allergic Factors

Oedema due to allergic factors (vasodilators) is noticed in urticaria or purpura haemorrhagica.

2. Toxic injures

Toxins damage the capillaries and increase their permeabilities leading to oedema in the diseases like anthrax, gas gangrene, malignant oedema, hepatosis dietetica and gut oedema etc.

Pathology

Oedema in tissue spaces or serous cavities does not arise, when the existing balance of pressure between the hydrostatic pressure and the plasma osmotic pressure at the venous end of capillaries permits the return of the fluid to the capillaries from the tissue spaces. In other words, the plasma osmotic pressure(inward attractive force) is greater than capillary hydrostatic pressure(outward expulsive force) at venous end of capillaries.

The main pathological conditions seen in animals are as follows:

Table 31. Oedema in different tissues etc.

1. Anasarca	-	An accumulation of fluid in the subcutaneous tissues
2. Ascites	-	An accumulation of fluid in the peritoneal cavity
3. Hydrothorax	-	An accumulation of fluid in the thorax
4. Hydropericardium	-	An accumulation of fluid in the pericardium
5. Bottle jaw	-	An accumulation of fluid in the intermandibular space.

It is a marked lesion seen in cases of parasitic intestinal infestations in animals.

6. Brisket disease It is marked by an accumulation of fluid in the brisket region of animals.
7. Oedema of limbs due to obstruction to venous blood as seen in case of pregnancy.
8. Local oedema of head.

It is seen in causes of African horse sickness and purpura haemorrhagica.

An oedematous swelling in the tissue is soft, painless and pits on pressure. Oedema in the thorax and pericardium affects cardiac movements, respiration and may even cause collapse. The pulmonary oedema which is a local oedema, may produce localised signs. e.g., respiratory distress and an outpouring of frothy fluid from the nose etc., Nervous symptoms are caused by cerebral oedema.

Diagnosis

It is based on the symptoms, lesions and an examination of the oedematous fluid etc. In non-inflammatory oedema, the fluid (transudate) does not contain inflammatory cells as noticed in the cases of oedema arising from lymphatic obstruction or hypoproteinaemia. The pathological changes are the same as noticed in specific diseases with important sign of oedema. When there is a rupture of urinary bladder or urethra, one notices subcutaneous accumulation of urine,

peritomitis, pleuritis (pleurisy) and pericarditis etc. In oedema due to hypoproteinaemia, one notices fall in the level of serum proteins.

Treatment / Management

The primary cause of oedema is the guiding principle of treating the cases of oedema.

1. Use cardiac tonics digitoxin or digitalin to treat oedema in congestive heart failure as seen in the cases of stenosis or incompetence of heart.
2. Oedema due to parasitic gastroentritis is treated by using anthelmintics.
3. Oedema due to hyproproteinaemia in animals needs the administration plasma substitutes. Such animals may be given diets rich in protiens.
4. Water and salt intakes should be restricted.
5. Restrict the administration of diuretics and aspiration of fluid. Diruretics like laxis or diamos may be used to remove oedema fluid from the body.
6. Oedematous fluid from the cavities should be aspirated slowly to avoid the peripheral circulatory failure or acute dilatation of splanchnic vessels in the abdominal cavity. One-third of the fluid from a cavity (say, abdominal cavity) is to be removed by paracentesis abdominis.
7. As a supportive treatment, liver extract, nervine tonic and vitamin B complex (oral) and iron preparation are given to the animal patients.

Table 32. Heart Tonics

Tr. Digitalis	-	8 ml
Tr. Nux Vom	-	10 ml
Tr. Zingiberis	-	30 ml
Aqua add	-	125 ml
Mft. hust. sig. once daily for eight days.	-	
Digitalin	Horse and cattle	15-60 ml s/c or i/m
	Dog	1-10 ml s/c or i/m
Digitoxin	Dog	0.1 to 1 mg orally or i/m

Preparation to treat pulmonary oedema

Atropine sulphate - 0.25 mg/kg body weight subcutaneously.

The average dose for large animals is about 75 to 100 mg of Atropine sulphate. This is dissolved in distilled water for the sake of subcutaneous injection.

Respiratory stimulants for use in respiratory failure, coma and anaphylactic shock etc.

1. Caffeine citrate

   H & C : 4 gm, Dog . 50 - 250 mg.

2. Coramine (Nikethamide)

   H & C 10 - 20 ml Intramuscularly Dog - 1-3 ml I/M or subcutaneously

3. Camphor in oil (1 in 20)

   10 - 20 ml is injected subcutaneously.

Diuretics

For Horse and cattle

R/

Phenacetin 4 gm

Treacle Q.S.

Mft. Elect. sig. B.D.

For dogs

R/

Sod. Salicylas 0.5 gm

Pot. Acetes 0.5 gm

Spt. Amm. Arom 1 - 2 ml

Syrup 5 ml

Aqua Add 15 ml

Aft. Haust. sig. T.D.S.

For ascites in small animals. Use injection Laxis 3-6 ml inj. Neptal 1 ml - intramuscularly.

Ruminal Tympany (Bloat)

Ruminal tympany refers to an abnormal distension of the rumen and reticulum with gas. Gas arises from ingesta or fermentation of food in the rumen and exists in the form of foam in the rumen and reticulum. Interference with eructation or ingestion of bloating forages causes bloat in ruminants. Legumes cause primary frothy bloat. Physical obstruction (e.g., recticular adhesions) in the oesophagus interferes with eructation.

Signs

The main signs are :

(i) Detection of dead animals on the farm with mild to severe distension of left abdomen.

(ii) Distension of right abdomen in the cases of severe tympany.

(iii) Distress, dyspnoea and protrusion of tongue.

(iv) Release of froth following passage of stomach tube in ruminal frothy bloat.

A lot of gas is released in secondary free gas bloat.

(v) Death of bloat cases occurs within a few hours if treatment measures are not attempted to.

Pathology

1. Marked hyperaemia and haemorrhages in tissues of cranial part of body like tongue, nasal sinuses, lymph nodes and proximal part of the oesophagus due to ruminal tympany. Such changes are not apparent in the caudal part of the body.
2. Distended abdomen and frothy contents seen at autopsy conducted soon after death.
3. Compressed state of the lungs, pale liver due to expulsion of blood from the liver because of tympany.

Treatment / Management

Treatment measures depend upon the kind of tympary and differ in both the frothy bloat or free gas bloat. The trocar and canula is used for emergence or release of the ruminal contents and gas in the bloat. If animal's life is not threatened, the passage of the stomach tube is recommended for immediate relief.

Viral Diarrhoea

Rota viruses, coronaviruses, toroviruses and parvoviruses cause viral diarrhoea in new born farm animals like lambs, kids, piglets, foals. Viral infections spreads to healthy young animals by faeces. Specific antibody in colostrum offers resistance to young suckling farm animals. Bovine coronaviruses (being pneumonic tropic viruses) cause respiratory diseases in young calves.

The main signs and age groups of different young farm animals are as follows :

Table 33. Viral diarrhoea and the age group affected.

Farm Animals	Signs	Age Group
1. Calves	Out break of diarrhoea.	5-14 days and older upto 3-4 weeks.
2. Piglets	Out break of diarrhoea (porcine).	1-4 weeks of age epidemic diarrhoea type I 4-5 weeks of age.
	Porcine epidemic diarrhoea type II.	Pigs of all ages.
3. Foals	Profuse diarrhoea, fever and dehydration.	

The main lesions in the affected young farm animals are dehydration and fluid filled intestines. Intestinal villi and crypts reveal atrophic changes.

The main viruses causing viral diarrhoea belong to families like Reoviridae, Coronaviridae, Toroviridae and Parvoviridae.

1. Rotaviruses

These viruses cause diarrhoea in calves. The factors influencing susceptibility to rota virus **infection are as follows :**

1. Age of animals.
2. Degree of viral exposure.
3. Immune status of dam and absorption of colostral antibody.
4. Practice of weaning.
5. Presence of other enteropathogens.

Youngest animals show highest mortality. Infected calves spread infection to incontact calves.

2. Coronavirueses

Bovine corona viruses infect calves all over the world and are pneumotropic viruses in bovines.

3. Parvoviruses

These have been associated in outbreak of postweaning diarrhoea.

4. Toroviruses

These are also associated with diarrhoea in young calves.

Group A rotaviruses cause diarrhoea in foals.

Pathology

Calves, lambs, pigs and foals show similar viral pathogenesis, infected epithelial cells become detatched and epithelial regeneration is noticed within 4-6 days following the onset of diarrhoea. Lactose malabsorption is noticed in young patients of virus diarrhoea. Parvovirus infection causes lymphopenia and damages the small intestinal crypt epithelium with villous atrophy. The faeces are voluminous mucoid and slimy, dark green or light brown in colour in calves infected with coronaviral enteritis.

Diagnosis

It is based on symptoms, lesions and detection of virus in faeces by electron microscopy. Immunofluorescence tests using faecal smear and fluorescent antibody technique are performed to confirm viral infection. ELISA test is done to detect virus in the faecal matter.

The most marked pathological lesions of viral diarrhoea are dehydration, fluid filled intestinal tract and distension of abomasum. Villus fusion or atrophy is noticed in the intestinal mucosae.

Treatment/ Management

The treatment procedure is the same as done in the causes of undifferitiated diarrhoea of calves. The withholding of milk for 24-48 hour is quite useful. Oral and parenteral fluid therapy is quite essential in treating the diarrhoeic cases.

Affected foals are given fluid electrolyte therapy for 72 hours. Infected calves should be isolated from the calving grounds. New born calves must get adequate amount of colostrum. Dams are vaccinated to induce specific immunity. Colostrum intake imparts resistant against several diseases..

Indigestion

Indigestion is noticed in animals following excessive ingestion of feeds like grain, silage and indigestible roughage. The main signs of indigestion are inappetence, drop in milk production, lack of rumination, usually full rumen and decreased or lack of reticulorumen contractions. Recovery in such patients occurs in 12-24 hours and is spontaneous in nature. There are no fatal lesions of indigestion. Primary atony (e.g., atony of rumen and reticulum etc.) is followed by indigestion.

Pathology

High protein diets increase alkalinity or changes in pH of ruminal contents and cause atony of rumen. Damaged or putrefied feeds also produce ruminal atony. Putrefaction of proteins produces amines i.e., histamine causes atony of rumen. Grossly, the affected rumen is firm or doughy without distension. Indigestion is commonly followed by diarrhoea at later stages.

Sediment activity test and cellulose digestion test are carried to asses the activity of ruminal microflora.

Treatment/Management

Animal patients of indigestion spontaneously recover from it. Rumenotonics, parasympathomimetics, alkalinizing and acidifyings materials are administered to animals to treat indigestion. Neostigmine is very effective at a dose of 2.5 mg/ 45 kg bwt. Magnesium hydroxide at the rate of 400g per adult is given to cattle in case of acidic ruminal contents. If the ruminal contents are alkaline 5 - 10 lt. of acetic acid or vinegar is recommended to treat indigestion.

Anaemia

Anaemia is a condition which is marked by either deficiency of red cells or reduced haemoglobin content of the blood. Red cells, incompletely charged with haemoglobin may cause anaemia, despite the normal red cell count in the patients. It can arise from defective blood formation or from loss of blood (i.e. as a posthaemorrhagic phenomenon). Excessive blood destruction causes haemolytic anaemia. Babsia, anaplasma and bacterial haemolysins cause haemolytic anaemia. Anaemia may be specific due to specific causes. The main clinical signs of anaemia are pallor to mucosae, thin and watery blood, petechial and ecchymotic haemorrhages, haemoglobinuria and bleeding tendencies, Postmortem findings are also specific to specific causes. Decreased values of red cells, or PCV indicate anaemia. Anaema may be classified as haemorrhagic or haemolytic anaemia or anaemia due to decreased production of red cells. In short, anaemia is marked by a decrease of red cells or of haemoglobin or of both in the patients.

1. Haemorrhagic Anaemia

The causes of haemorrhagic anaemia are as follows :

1. Spontaneous rupture or traumatic injury to blood vessels is seen in the cases of castration and dehorning.
2. Severe parasitic infestation e.g., Strongyles, hookworms, *Haemonchus* spp. infestation, *Fascicla hepatica* and coccidia cause severe loss of blood.
3. Clotting defects.

Anaemia is caused by coagulation defects as seen in some acute haemolytic anaemia.

2. Haemolytic Anaemia

The causes of haemolytic anaemia are :

1. Babesiosis, anaplasmosis, trypanosomiasis and theileriasis.

2. Bacillary haemoglobinuria.
3. Leptospirosis.
4. Postparturient haemglobinuria.
5. Ingestion of *Brassica* sp and rape etc and excessive feeding of culled onions.
6. Transfusion reaction.
7. Autoimmune haemolytic anaemia.
8. Congenital anaemia with jaundice.

Anaemia is also caused due to decreased production of red cells or haemoglobin.

The main causes are :

1. Nutritional Deficiency

Deficiency of copper, cobalt and iron produces anaemia in animals. Iron deficiency causes piglet anaemia in baby pigs. Anaemia develops in calves due to potassium deficiency. Pyridoxine deficiency can contribute to anaemia in calves. Folic acid deficiency causes anaemia in pregnant mares.

2. Chronic Disease

Erythropoiesis is depressed by chronic supportive diseases. Radiation injuries and intestinal parasitism (trichostrongylosis in calves and sheep) induce anaemia in them. Some unknown causes also lead to anaemia in animals.

3. Myelophthistic Anaemia

It is a rare kind of anaemia in animals which is marked by neoplastic growth in marrow cavities, fractures of skeletal bones, porosis and cavitation of bones. Anaemia in affected animals is macrocytic and normochromic. It is noticed in lymphosarcoma and plasma cell myelomatosis in calves.

Autoimmune Haemolytic Anaemia

It is marked by production of antibody targeted against

surface antigens of red cells in animals. Intravascular haemolysis destroys the red cell, and macrophages of the reticuloendothelial system which removes the dead and destroyed cells. The antibodies produced in such patients are of the IgG or IgM types.

Signs

The main signs are :

1. Pallor of the mucosae, muscular weakness, increased rate of pulse and tachycardia.
2. Weak heart sounds and weak pulse.
3. Labored breathing or dyspnoea with terminal stages of anaemia.

Pathology

The main changes are :

1. Increase in number of immature red cells in the blood and normal total protein in haemolyitc anaemia.
2. Presence of bilirubinemia and bilirubinuria and discoloured plasma. There is an increased red cell fragility and a positive antiglobulin test in immune mediated haemolytic anaemia.
3. Haemorrhages due to thrombocytopenia, thin watery blood and contracted spleen, lcterus in haemolytic anaemia and decreased haemoglobin percentage.
4. Other changes affecting red cells are anisocytosis, (variations in sizes) poikilocytosis (variations in shapes), presence of nucleated red cells, reticulocytes and variations in staining reaction (punctate basophilia)
5. Regeneration of red cells, replacement of the fatty marrow by red marrow and haemosiderosis in some organs.

Treatment/Management

The primary cause of anaemia determines the future line of treatment in patients. Blood transfusion and

haematinic preparations or administration of blood are useful steps to treat anaemic patients.

Laminitis

Laminitis refers to degeneration of the sensitive laminae of the hoof in animals like cattle and horses. The acute degenerative changes are noticed in the sensitive primary and secondary laminae of the hoof. Acute vascular exudative changes in the hoof are rated as secondary changes to degenerative changes. The exact cause of laminitis is not known. The clinical signs of laminitis in horses are colic, diarrhoea, metritis and grain engorgement. Laminitis in cattle is associated with ruminal acidosis (clinical or subclinical). Lameness and recumbency are noticed in sick horses. Inapparent or severe lameness is noticed in cattle. Physical examination and radiography confirm diagnosis of laminitis. Over weight ponies are frequent victims of laminitis. Trauma and other physical factors (i.e., excessive worms) contribute to laminitis in horses. Laminitis is also associated with systemic illness in ponies. Subclinical laminitis is noticed in cattle. In jersey heifer, inheritance autosomal recessive gene conditions development of laminitis. Laminitis develops in heifer soon after calving. Laminitis is marked by separation of sensitive laminae from the laminae lining the internal surface of the hoof. The 3rd phalanx drops through the hoof and rests on the sole. Degeneration of the sensitive laminae is secondary to ischaemia due to formation of microthrombi within the capillaries of the sensitive laminae. Rotation of the 3rd phalanx leads to the state of dropped sole. Chronic traumatic aminitis is common in heifers. The pedal bone shows rarefaction in cattle by radiography. In horse, autopsy reveals excessive amounts of grains in stomach. Microscopic examination of the affected tissue sections shows loss of some of the keratogenic structures of the epidermal laminae.

Treatment/Management

Acute laminitis is an emergency disease which needs immediate attention of surgeons or doctors.

The main steps of treatment of the horse are as follows:

1. Removal of causative agents.
2. Relief from pains.
3. Vasodilation of blood vessel in foot.
4. Prevention of formation of microthrombi in dermal capillaries.
5. Avoiding the rotation of the pedal bone.
6. Promotion of keratinisation and hoof growth. Nonsteroidal anti-inflammatory drugs (NSAIDS) are administered to sick horses. Phenyl betazone at doses of 2.2-4 mg/kg is given intravenously to horses. NSAID is also administered to cattle suffering from laminitis.

Aspirin 0.3 grain/kg orally every 12 hours or phenyl butazone 4.4 mg/kg orally every 48 hours is administered to bovine cases of laminitis. Antibiotics are used to prevent infection, Vasodilatory drugs are used to prevent infection. These drugs are also used to retard progression of acute laminitis in animals like solipeds and cattle. Anticoagulants are used to prevent capillary microthrombi in the hoof of the patients.

Diseases Caused by Zootoxins

Zootoxins introduced into the body of farm animals by bites of snakes, stings of bees or tick bites etc., are very harmful to these animals. Snake bites cause even death in large animals. Fang marks are located or traced at the bitten sites. Some details about diseases caused by zootoxins are as follows :

Snake Bites

Snake bites inject venom in to the body of the victims

with fangs and such bites are especially noticed during summer months. The clinical signs of the disease are muscle weakness, stumbling gait, recumbency, pupillary dilation, swallowing paralysis, salivation and muscle tremor. Inflammatory swelling and necrosis are noticed at bite sites. The clinical examination reveals venom in blood, urine, body tissues and fluids. Autopsy findings include necrosis and local swelling at the bitten sites of snakes.

The toxic effects of snake venoms are as under :

1. Flaccid paralysis, pupillary dilation and paralytic respiratory failure caused by neurotoxins in snake venoms.
2. Tissue necrosis and necrosis of platelets are caused by cytolysins. Such changes are followed by intravascular coagulation.
3. Haemolysis of red cells by haemolysin in the snake venom. Coagulant fraction of snake venom causes intravascular coagulation.
4. Haemorrhagic tendency due to thrombosed and anticoagulants in snake venom.
5. Muscle necrosis and myoglobinuria are caused by myotoxins. The size and species of the snake, the size of the bitten animal and the location of bite, thickness of hair coat subcutaneous fat and the actual amount of toxin injected influence the effects of snake bites in the animals.

The clinical signs of snake bites are severe pain, excitement, anxiety, swellings, dilatation of the pupils, salivation, hyperaesthesia, tetany, depression, recumbency and terminal paralysis. Asphyxia is seen as factor causing death in small animals. Local sloughing of the swelling at bite site is noticed in recovered cases. An ELISA is used to identify snake venom in blood, urine or other body tissues. Autopsy findings include several local swellings containing serous or inflammatory exudate. Fang marks are noticed on the under surface of the reflected skin.

Treatment/Management

The principles behind treatment of snake bites in man animals are almost identical.

A tourniquet is applied above the bitten site to restrict the blood circulation. If possible, suction is soon carried to remove the snake venom from the bite site. Tourniquet is released at 20-minute intervals. The bitten site is incised to reach the site of deposition of snake venom i.e., up, to a depth of 0.5 cm. Excision at the bite site of snakes reduces local tissue reaction. A firm pressure bandage is used in humans to restrict distribution of snake venom by lymphatics. Snake venom retained at bitten sites prevents systemic effects. Application of firm pressure over the bitten areas delays absorption of snake venom. A broad firm bandage around the limbs above bitten areas occludes the superficial venous and lymphatic outflow. Mechanical suction removes blood or fluid from the bitten zones. A cross incision (about one cm. long and ½ cm deep) over fang marks causes an escape of blood or fluid containing the snake venom. The wound is cleaned with sterile water or normal saline.

Antivenin, antibiotics and antitoxins are used Antivenin contains antibodies against the venoms of all snakes in the areas of snake bite incidence. The i/v route is preferred for quick effects. One unit of antivenin is sufficient for animals weighing 70 kg or more but smaller animals of 9-18 kg b.w require about 5 units of antivenin. Broad spectrum antibiotics are administered into the victims to control the local infection. Sedatives may be given to control pain and excitement. ACTH, cortisone and antihistaminics are used to protect the victims from anaphylaxis.

Bee Stings/Stings of Scorpions and Wasps

Severe local swellings (up to 6 cm. diameter) are caused by multiple stings of bees. The swellings in lips, eyelids, tongue and in vulva are painful. Diarrhoea, haemoglobinuria

jaundice, tachycardia, cardiac arrythimia, rapid breathing, sweating, moderate colic and prostration are noticed in horses having multiple stings of bees. Victims of bee stings may die in some cases. Haemorrhages and oedema of connective tissues are noticed as autopsy lesions in fatal cases. Application of a weak solution of ammonia or sodium bicarbonate or tincture of iodine at sites of bee stings has beneficial effects. Nervous stimulants are administered to check prostration. Tracheotomy is performed to save the victims of bee stings from asphyxia. It is also better to incise the areas of bee stings or stings of scorpions to allow escape of blood having zootoxins. Even mechanical suction can be applied over the bitten zone to remove blood or fluid containing toxins.

Impaction (Impaction of Omasum and Abomasum)

Impaction of Omasum

Impaction of the omasum and abomasum (forestomachs) is noticed in ruminants. When the omasum is excessively enlarged and firm with ingesta, it is called impaction of the omasum. Tough and fibrous feed or even soil cause impaction of the omasum. This condition is noticed in sheep. Bouts of indigestion, decreased rumen motility, infrequent and scanty faeces and negative ketone tests are signs of impaction in animals like sheep. In fatal cases, omasum reveals distension and patches of necrosis on omasal leaves with even peritonitis. Such necrotic changes may also be noticed in the ruminal mucoase. In short, the important clinical signs of impaction are complete anorexia, cessation of defaecation, and empty rectum, abdominal pain and disinclination to move or lie down.

Impaction of the Abomasum

Impaction of abomasum is noticed in cattle ingesting excess quantity of poor or low grade roughage. The important signs of impaction are anorexia, scant faeces, distension of

abdomen, loss of body weight. In abomasal impaction, rumen in patients is full and atonic. Distension is noticed in right lower flank which can be palpated. Affected animals become weak and recumbent. The clinical observations include metabolic ketosis, hypochloremia and hypokalemia. Autopsy of fatal cases reveals greatly distended abomasum, Laprotomy may be made for diagnostic confirmation. When animals (e.g., pregnant cows) ingest large quantities of sand, the ingested sand causes impaction of omasum, abomasum, large intestine and caeca etc. Chronic dilatation and atony of abomasum ensues from gradual accumulation of sand. Dehydration is noticed in cases of abomasal impaction. Hypokalemia follows sequestration of potassium iones in abomasum. This condition can turn into cases of permanent abomasal atony. The important clinical findings are akalosis, hypochloremia, haemoconcentration and dehydration in animal patients. There is an increased respiration and static rumen with full of contents. Over distension of abomasum causes stretching of its serosa.

Pathology

At autopsy, the abomasum is abnormally enlarged up to twice the normal size and impacted with dry contents. Such impaction may also be seen in omasum. Beyond the pylorus, the intestinal tract is dry and empty. Dehydration and emaciation are the marked changes in the carcasses. Acute diffuse peritonitis follows rupture of over distended abomasum. Abomasal tears or necrosis or ulcers also may be present in rumen, omasum and abomasum.

Treatment/Management

Measures to correct metabolic alkalosis, hypochloremia and dehydration in affected animals are taken to save the patients. Balanced electrolyte solutions are given i/v to animal patients for up to 72 hours @ of 100-150 ml/kg body weight over a 24 hour period. Dioctyl sodium sulpho succinate is

administered into rumen by stomach tube at a dose rate of 120-180 ml of a 25% solution for 450kg animals. It is repeated daily for 3-5 days. This drug can be mixed with 10 L of warm water and 10L of mineral oil while administering into the body. Dioctyl sodium sulfosuccinate is given by stomach tube. Chlolinergics like neostigmine, physostigmine and carbamylcholine can also be used effectively. An abomasotomy is a step for surgical correction of abomasal impaction.

Shock

Shock (Fr. = to shake or jolt) refers to an acute peripheral circulatory failure. Infact, it is any entity with multiple causes and marked clinically in patients by changes like functional depression, elevated heart rate with weak pulse pallor, sweating, poor venous return and feeling of cold, lack of strength and subnormal body temperature. Shock is divided into two kinds, namely (1.) Primary shock and (2.) Secondary (haematogenous shock).

Peripheral circulatory failure (shock) arises from decrease in effective circulatory volume (hypovolaemia) due to haemorrhages, fluid loss, fluid sequestration or septic or toxic effects on blood vessels. Haemoconcentration, multiple organ dysfunction, low arterial blood pressure, low central venous pressure and elevated blood lactate are important changes.

Primary shock or neurogenic shock occurs in immediate conjunction to trauma and is marked by unconsciousness due to vasodilatory effects of histamine.

The main causes of peripheral circulatory failure are as follows :

1. Hypovolaemic Failure

It arises from reduction in circulationg blood volume due to blood or fluid loss.

2. Vasogenic Failure

It is caused by peripheral vasodilatation, pooling of blood in vessels and lack of blood into tissues.

3. Septic or toxic shock

Toxic or septic factors cause vasogenic failure because of loss of vascular integrity or increased vascular permeability. Impairment of oxygen uptake, anaerobic metabolism in tissues and widespread cellular dysfunction cause shock to pass into an irreversible shock or death.

Severe burn injuries, extensive surgery, prolapse of uterus, sudden reduction of pressure in body (for example, quick withdrawl of ascitic fluid), severe pain (as seen in colic) and trauma with local sequestration of blood and fluid can induce shock in animals. Potent endotoxins of Gram negative organisms cause toxic (septic) shock in animals. Shock is seen in animals affected with septicaemic diseases or Gram-negative bacterial infections and toxic infections (e.g., acute gangrenous mastitis, acute mastitis and paracute coliform mastitis). An absorption of toxin from intestines and infarction in intestines induce septic or toxic shock in animals. Endotoxins or other bacterial toxins damage the endothelial cells of the capillaries which turn into leaky vessels.

The main clinical findings are depression, weakness and restlessness with a fall in body temperature.

Pathology

There are no specific autopsy findings in the cases of shock. Congestion in the capillaries and small vessels of the splanchnic area and pulmonary oedema are noticed in toxic shock. Traumatic injury is noticed in the animals died of

shock. The carcasses of dead animals bear a dehydrated appearance. Severe congestion is noticed in smaller vessels of the liver intestines and lungs. Spleen of dead animals shows an empty bloodless appearance. The peripheral parts of the dead bodies reveal ischaemic appearance. The lungs of such cases show oedema fluid and red cells in the alveoli. Blood tinged fluid is noticed in peritoneal, pericardial, and pleural cavities of animals died of shock.

Treatment/Management

The line of treatment in the cases of shock depends upon the identification of the causes. Quick surgical intervention in the injured animals with fluid therapy is a must in the case of shock to save the animals life. Early treatment of burn or accident cases with extensive tissue damage is needed for treating the patients of shock. Fluid therapy and use of antibiotics is resorted to improve the patients condition.

Treatment of some diverse pathological conditions faced in day to day veterinary practice.

I. How to Treat Ectoparasitic Infection

To control ticks, lice, fleas and mange in animals, the animals can be dusted with Gammexine (BHC 5%) powder. The houses and cattle sheds should be disinfected with 5% DDT solution. Malathion in strength of 0.1% solution may be used for spraying on cattle bodies. 0.2% malathion solution can be used as spray on animal sheds. In no case, grass, hey and feeding trough etc., should be sprayed with malathion solution in order to check poisoning. Sumithion is also used as spray in poultry houses. Butox and Pestoban can also be used to control the ticks.

II. How to Treat Infertility (failure to conceive and give birth) in Cows and Buffaloes

It arises from diseases in gonads like ovary and testicles and also other parts of reproductive system like oviduct and uterus etc., Ovarian cyst, lack of balanced diet, malnutrition,

managemental errors, congenital defects and persistent corpus luteum are very important factors causing infertility in cattle and buffaloes. Thus, hormonal imbalance is an important cause of infertility. The animals can be rendered infertile either due to anoestrus or repeat breeding. The following line of treatment can be adopted for treating anoestrus after confirmation of non-pregnancy state in an animal :

(i) Prajana capsule can be given to cows and buffaloes at the rate of 3 capsules on the first day and if there is no heat, this can be repeated after 24 hours.

(ii) Tonophosphane inj. 5 ml. i/m on alternate day (6 inj.). Prepalin forte 2 ml i /m on alternate day (6 inj.) can be used.

(iii) Mineral mixture (30 gms) and Vitamin supplement such Vitablend at dose rate of 5 gm. should be given for 15 days. There should be massage of ovary and uterus. Hormonal therapy can be given. The uterus should be tampooned with Lugol's iodine using speculm and swab holders. Other heat producing drugs in place of Prajana such as Fertivet @ 300 mg can be given orally for 5 days.

III. How to Check Repeat Breedings in Bovines

Animals suffering from repeat breeding are noticed to come in heat regularly without conceiving after natural or artificial service. Ovarian cysts produce nymphomania (abnormal heat) in cows. Such animals with history of repeat breeding should be thoroughly examined for ovarian and uterine abnormalities. After the cause of repeat breeding has been ascertained, the following line of treatment is followed:

(i) There should be intra-uterine application of antibiotics before and after artificial insemination. Gentamycin, hostacyclin and terramycin liquid can be used. One should always do insemination in late oestrus which lasts over more than 30 hours in crossbred and exotic animals. Two

inseminations are usually done at intervals of 12 hours. Intra-uterine application of gentamycin should be done during oestrus (that is the sexual or oestrus cycle for 18-24 hrs for both cows and buffalloes) at 12 hours from the onset of oestrus and this should be repeated after lapse of 24 hours from the moment of insemination. It is better to start breeding within four months after delivery to achive one calf every year.

IV. How to Enhance Lactation after Parturition

Cows and buffaloes some times show poor milk production due to various factors. In order to increase production, the following preparations can be used:

(i) Ostocalcium B_{12} 500 ml and Vimeral syrup 30 ml in mixed state can be used in doses of 100 ml daily for 2 weeks.

(ii) Leptadine tablets - 100 tab. Ten tab. daily for 10 days can be given orally.

(iii) Glactagouge - 50 gm daily for 2 weeks. This drug is recommended to enhance the milk production.

V. How to Treat Retention of Placenta

There should be expulsion of placenta normally after six to eight hours of parturition in cattle. If there is a retention of placenta, the following drugs can be used.

(i) Erbolin tab. 20 tab. should be given twice daily for two days.

(ii) Replanta 50-100 gm can be given per day for 3-4 days.

(iii) Oxytocin inj. 10 units per ml 5 ml of this drug is given i/m after parturition.

In case of persistence of retention of placenta, the placenta should be removed manually with hand after using hand gloves smeared with cream of antibiotics. This precaution is very important to protect the physicians from brucellosis. *Brucella abortus* can even penetrate into intact skin to produce infection. The infection due to *Brucella abortus* in

the uterus may lead to retention of placenta in cattle.

After removal of placenta, the following pessaries can be used at least for three days. Fureabolus, Steclin bolus or Sulcoprime bolus etc., can be used.

How to Treat Pyometera

Pyometera in cattle arises from vibriosis, trichomoniasis and brucellosis. Fever and toxaemia can be found and the uterine discharge has foul smell. The treatment suggested is as follows:

(i) Uterine discharge is removed by gentle uterine massage per rectum.

(ii) Injection of the post pituitary hormone (Oxytocin) should be given for 2 to 3 days. Even Vetestrol 10-20 ml can be given.

How to Treat Prolapse of Vagina in Cattle

Prolapse of vagina and uterus occurs due to severe irritation and excessive straining. In such cases, it is advisable to keep the hind quarters of animals on a raised ground level or platform and prolapsed mass in uterus and vagina should rest on clean cloth. The protruding mass should be irrigated with cold water containing 1% Savlon or 0.1% solution of Potassium permangnate. Use adrenaline topically to check bleeding. Anasthetic ointment like xylocin should be smeared on prolapsed organs. It is also proper to have gentle manoeuver for reduction. Posterior pituitary hormone can be injected at a dose rate of 5 ml i/m. In such cases, it is better to remove urine with help of catheter.

Antihistaminics and sedatives such as Chloral hydrate can be used. Mifex can be advised to be given parenterally.

How to Treat General Cases of Poisonings

Symptoms like vomiting, bloating, frothing from mouth, diarrhoea, loss of consciousness, coma, convulsions, clamping of jaw, arrhythmia, heart beat, dilatation of pupils and subnormal or a febrile conditions are found in case of poisoning in animals. The following principle of treatment can be adopted: (i) Further ingestion or absorption of poisons should be checked (ii) Residual poison from intestine can be removed by giving emetics. Gastric lavage or purgative etc., is used. (iii) Skin can be washed in cases of poisons detected over it. (iv) Residual poisons can be neutralized by the followings drugs :

(a) Oxidizing agents like tannic acid, milk, eggs, are very useful in cases of poisonings (particularly corrosives).

(b) Chemical antidotes.

(c) Calcium B-12 acts as antidote.

(d) Use of activated charcoal.

(e) Fluid therapy to check dehydration and use of sedatives to check excitement.

The antidotes applicable against particular or specific poisons should also be tried.

How to Treat Snake Bites

In cases of snake bites, lower extremities, head and muzzle should be examined for swelling and pain etc. Death in snake bites occurs due to asphyxia. In 1 to 10 hours, dogs may die but cattle and horses may die within 48 hours. The treatment is as follows:

(i) Use tourniquet above the bitten site i.e., in another words, there should be tight tying or ligation above the site of bite.

(ii) Give an incision at the site of bite and allow it to bleed profusely and wound should be washed with sterile water.

(iii) Antivenin is administered into the bodies of victims of snake bites.

The basic principles in the treatment of snake bites in man and animal are almost the same ones. The main steps are given below for the sake of information:

(i) Apply firm pressure over the bitten area in order to delay the absorption of snake venom (the first basic step). Or a broad firm bandage around the limb above the bitten site should be used. The purpose of using bandage is to occlude the superficial venous and lymphatic outflow.

(ii) Apply firm pressure over the bitten area on the head, trunk or neck. It is easy to use bandage on the extremities or limbs. Bandage can be loosened for 90 seconds every 10 minutes. Immobilize the limbs to check spread of the snake venom through the out flowing blood or limb from the bitten site .

How to Treat Drowning Cases of Animals

Drowning of an animal means the submersion of its body in water. If this occurs for a period of about three to four minutes (say, man), it is sufficient to cause asphyxia and death. It may produce collapse from which recovery is not possible.

In such cases, the management includes the following steps:

1. As soon as the animal has been rescued from water, it should be placed in a position which will allow water taken into the lungs to run out by the mouth and nostrils.
2. Small animals may be held up by the hind legs and swung from side to side and the large ones should be laid on their sides with the hind quarters elevated at a higher level than their heads.

3. Pressure should be brought to bear on the chest by one throwing all his weight on to the upper part of the chest wall or by kneeling on this part. When no more fluid runs from the mouth, the animal should be turned over on to opposite side and the process is repeated.
4. Then, the animal should be removed to warm surrounding as soon as possible and dried by wiping or by rigorous rubbing with rough towel. Clothing should be used and the smaller animals may be provided with one or more hot water bottles. Steps should be taken to prevent the development of pneumonia in such patients.

How to Treat Burns in Animals

Burns may defined as a destruction of tissues caused by the application of heat or chemical substances to the external or internal surfaces of the body.

Burns includes all lesions, whether produced by fire, hot water or chemical substances.

In cases of burns, the parts most affected are face (especially the eye lids), conjunctivae, lips, surface of the body especially udder, teats, perineum, and the coronets. Badly burnt skin will be dry and ready to sloughed off in a week. Burns of all degrees (1st, 2nd and 3rd degree etc.) may be found in the bodies of the animals.

Symptomatic treatment may be adopted in case of burns. Cooling astringent lotions containing calamine and antihistaminics (Avil inj.) should be used with excellent results. In view of having quick recovery, antibiotic, therapy should be given to patients. Painkillers or analgesics may also be used. Sloughing in skin should be removed by surgical interference. Fly repellent should also be used. Fluid therapy may also be given.

Fluid therapy may be also given to suffering animals in order to prevent the risk of shock.

Antiserum can be given at the rate of 2 ampoules. i/v as initial dose and this should be repeated till recovery is noticed.

How to Treat a wound or a swelling in animals

The veterinary practioners or doctors usually come across cases of swellings without any opening or even open wounds.

It is very important to ascertain at first the nature of the swelling or wounds which may be tumours, chronic inflammatory growths or enlarged lymphatic glands. Then, the wounds should be treated with all precautions to prevent unnecessary complications or spread of malignancy. In case of establishment of lesions as an inflammatory growth or simply as absceses, the wounds should be opened to drain the pus with sterilized instruments . There should be proper draining and irrigation of the wound with antiseptic lotions and packing of the wound with magnesium sulphate powder. In order to effect the quick recovery, there can be parenteral use of antibiotics and daily dressing with antibiotic lotions. In case of malignant lesions or growth, these should be operated with all surgical precautions to prevent metastasis in the body. Enlarged organs e.g., lymphoid organs, do not require to be surgically removed and after the cause of lymphatic swelling is known, the specific treatment should be followed as done as in cases of theilariasis etc. Swellings or wounds on the limbs of pets (say, dogs) are also caused by malignant osteosarcomas and other growths.

Chapter 9
Some Metabolic Disorders and Others

Milk Fever

The term milk fever is a misnomer as there is no fever or no milk in this disease of animals. It does not indicate what its term implies. and occurs in the dairy cows and some times also in sheep. Hypocalcaemia and defects in the parathyroid glands (poor level of parathormone) cause this condition in the animals. It is characterized by hypocalcaemia (calcium level may be as low as 3.0 mg per 100 ml of blood). Hyposphosphatemia and slight hypermagnesemia are also present. Certain information as regards the diseases in bovines marked by recumbency are given in table 34.

Table 34. Pathological Signs and Lesions in the Diseases of Animals (bovines) Marked by Recumbency

Diseases Diagnosed	Common Signs or Lesions	Pathologic Signs/Features Expected
(1)	(2)	(3)
1. Milk fever (parturient paresis) characterized by no fever and no milk. An example of metabolic disease often noticed in cows.	Recumbency	Mature cows usually affected within 48 hours of calving. Early excitement and tetany followed by coma, hypothermia, flaccidity, dilated pupil, absence of ruminal movements, soft heart sounds and impalpable pulse. Presence of hypocalcaemia (calcium less than 5 mg/dl (1.25m mol/L). High level of magnesium in serum (over 3 mg/dl) 1.25 m mol/L). inorganic phosphate low i.e. less than 3 mg/dl (0.9 m mol/L). Quick response following injection of soluble calcium salt. Absence of gross and microscopic lesions in fatal cases.

Diseases Diagnosed	Common Signs or Lesions	Pathologic Signs/Features Expected
2. Downer cows (Downer syndrome) This syndrome is a metabolic disease	Recumbency	Marked by inability to rise in spite of efforts to do so (creepers). Animal patients are bright and active and also eat the feeds given. Slight rise of temperature. Downer cows may be dull or depressed and show lack of alertness (non-alert downers). The course may range from 1 to 2 weeks. Presence of ketonuria and proteinuria. There may be low level of calcium, potasssium and inorganic phosphate. Usually occurring in cattle following milk fever or lactation tetany. Presence of fatty changes in the liver and myocarditis. Traumatic injuries to muscles or nerve in dead bodies at autopsy.
3. Ephemeral Fever	Recumbency	Presence of myositis, thin nasal discharge, lymphadenitis and transitory fever. Marked by high count of white cells. The patients recover spontaneously within 3 days. Epidemics caused by insect vectors.
4. Hypomagnesemia (also called grass tetany). A metabolic disorder seen in cows with in few hours of calving	Recumbency	Occurfence of disease in cattle (mostly calved females) Magnesium level is less than 1.2 mg/dl (05 m mol/L)/. Presence of excitement, hypersensitivity, muscle tremor and tetany. The animals patients go down with tetanic convulsions, presence haemorrhages in the peritoneum, intestinal mucosa. Subcutaneous, subencocardial haemorrhages.

Signs

The main signs of the disease are :

(i) Recumbency, somnolence. Often cows are found in comatose condition. The sick animals are seen lying on their sternum with their heads turned towards their flanks.

(ii) Inability to stand on their feet and death within a day or two due to lack of treatment.

Pathology

There are no significant gross and microscopic lesions in milk fever in the sick animals.

Downer Cow Syndrome

It refers to those cases of milk fever in cows which do not respond to calcium therapy and patients fail to stand on their feet. This syndrome incorporates complicated lingering cases of milk fever. The animal may be alert with usual appetite but attempts to stand on their legs are unsuccessful.

Treatment/Management

Since milk fever is a disease developing due to low blood calcium level, treatment is directed towards correcting the level of calcium in the body after detection of symptoms in the animals.

The following drugs can be used :

1. Calcium borogluconate preparation is a drug of choice and can be given in the doses of 400 - 500 ml of a 20% solution. Calcium borogluconate is given slowly by i/v route. It is better to warm the bottle equal to body temperature and i/v injection should be interrupted for 20 - 30 seconds twice during administration. The administration may be completed within 20 - 30 minutes and there should be auscultation of heart during i/v administration of the drug. If there is any irregularity in heartbeats, its administration is stopped immediately.
2. Antihistaminic drugs or cortisone can be given.
3. Mifex may also be used in place of calcium borogluconate.
4. In order to maintain blood calcium, drugs like Caldi - 12 in dose rate of 15 - 20 ml i/m daily for 3 days is advocated.

Supportive treatment should also be given in the form

of mineral mixture and calcium B_{12} syrup etc.

Underdosing with calcium preparation (calcium borogluconate) is not safe because the cows may fail to rise or a relapse of milk fever may occur in them.

Cows suffering from hypocalcaemia respond well to a definite pattern of calcium therapy.

Apart from discussion on treatment aspect of milk fever, it is very important to put stress on the control aspect of milk fever. The milk fever can be very well controlled by following steps :

1. Calcium intake : It is better to keep appetite of the pregnant cow at normal level and steps should be taken to avoid alimentary tract stasis. It is well known that cows kept on a diet high in calcium content before calving have increased chances of milk fever. If cow is dependent on gastro-intestinal absorption of calcium and not on skeletal release, the chance of milk fever is very much increased. And as such, usual balanced diet should be given to pregnant cows.
2. Avoid excessive intake of calcium during pregnancy.
3. Use of calcium, gel-calcium (in the form of gel) can be given to pregnant cows by way of drench or in the feed to provide 100 gm calcium daily.
4. Administration of Vitamin D_3 (Cholecalciferol) can be given to the cow as a prophylatic measure against milk fever.

Treatment/Management of Downer Cow Syndrome

This condition which is characterized by the inability of the cow to stand on their feet usually after parturition requires prompt curative measures. In view of its existence as complication of milk fever or a condition associated with various factors like metabolic factors, toxaemic or deficiency states of vitamin and selenium, treatment measures are taken

accordingly. The measures are as follows :

(i) Inject tonophosphate 20 ml by i/v route.

(ii) Injection of berin or Neurobion at the rate of 10 ml i/m for a period of 3 to 5 days.

(iii) Calcium borogluconate 450 ml i/v can be given slowly.

(iv) Potassium acetate 10% solution in dose of 100 ml by i/v route can be given daily for eight days and along with this, dianabol (100mg) i/v should be given for three days and betamethasone 200 mg epidurally for 2 days.

(v) Animals should be supported with ceiling. The body should be massaged with camphor liniment etc.

Steps should be taken to prevent bedsores by providing them very good bedding with frequent turns to the animals from side to side. Such steps check development of ischaemic necrosis due to prolonged pressure or recumbency. The main objectives of treatment are directed towards helping the animals to stand on their own feet.

Ketosis (Acetonaemia)

When the acetone or ketone bodies appear in the blood of animals, it is called ketosis. The ketone bodies appear in the urine of such diseased animals to produce (ketonuria). The chemical substances like acetone, betahydroxybutyric acid and aceto-acetic acids are called ketone bodies. In all species of animals, ketosis arises from decrease in the availability of blood glucose or from hypoglycaemia or from inability to utilize glucose as seen in case of diabetes mellitus. Lack or want of glucose is compensated by oxidation of fatty acids to produce energy for different kinds of cellular activities. This abnormal phenomenon results in the appearance of ketone bodies in the blood of animals. If the production of ketone bodies is within the physiologic limit of utilizing fat in the case of non-availability of glucose, the situation is an usual phenomenon in the body. When there is very poor availability or lack of glucose in the body, the

situation reaches the pathological level of excessive ketone bodies in the body, that is, in true sense, the state of ketosis exists in the body of the animals. Unavailability of oxaloacetate allows incorporation of fatty acids in tricarbolic acid cycle which causes abnormal production of ketone bodies. A sickly sweet smell due to ketone bodies is imparted to the animals. This is metabolic disease is often seen in ruminants.

The causes of ketosis are as follows :

(i) Starvation : The animal derives energy by oxidation of its stored fat in body. Proteins of muscles are also used later by the animals for energy. Emaciation is noticed in such cases.

(ii) Loss of appetite or failure to eat, ruminal indigestion or atony of the rumen.

(iii) Use of too much coarse roughages such as straw also causes development of ketosis.

(iv) Digestive disturbances.

Simple starvation causes primary ketosis whereas secondary ketosis arises from other diseases.

When there is an interference with appetite or oxidation, primary spontaneous ketosis is produced in the bovines. Diabetes mellitus and toxic damage to the liver as found in phosphorus or carbon tetrachloride poisoning also cause ketosis in the animals. Metabolism and storage of glycogen are very much disturbed in such states. Ketogenic diet (i.e., diet containing too much fat and little carbohydrate) can also lead to ketosis in the body of the animals.

Pathology

High level of ketone bodies is found in the blood serum. Acetone is also found in the urine. Milk can also be tested for the presence of acetone. There is development of hyperlipemia and presence of excessive amount of fatty acid which appears

as neutral fat in the liver cells. Depletion of the body's reserve of alkaline ions (i.e., $NaCHO_3$) causes acidosis in the body. Ketones (i.e. acetone, acetoacetic acid and beta hydroxy butyric acid) neutralize alkaline ions in the blood to produce such state. Reduction in the storage of glycogen in the liver causes hypoglycaemia in ketosis.

Pregnancy Disease of Ewes

It is characterized by ketosis i.e., a toxaemic stage of pregnancy. The affected ewes show depression, somnolence and coma. Hypoglycaemia is also seen in such animals. This pregnancy disease is seen only in the last week of pregnancy in the ewes. Fatty changes are seen in the heart, liver and kidneys. Subepicardial petechiae and ecchymoses arise from toxaemia in the ewes. This condition in the ewes arises from toxic products of pregnant mother and foetus, starvation, maintainance on non-nutritious diet and lack of exercise etc.

Diagnosis

It is based on the detection of abnormal level of ketones in the blood, urine and milk etc. Symptoms and lesions in the sick animals also aid the diagnosis of ketosis.

Treatment/Management

Ketosis in the animals is caused by hypoglycaemia within 6 to 8 days of parturition. The treatment is directed towards lowering the level of ketones in blood with measures to raise the blood glucose level. Hypocalcaemia can also occur with ketosis and indicates the steps to be taken to raise the level of blood calcium.

The measures suggested are the following :

(i) Inject Dextrose 25-50% solution in doses of 500 -1000 ml by I/V route. This parenteral administration of Dextrose is a specific treatment.

(ii) Mifex can be given in doses 450 ml (i/v route) with ob-

ject of treating hypocalcaemia.

(iii) Corticisteriods can also be given.

(iv) In order to control nervous symptoms, animals can be given chloral hydrate 30 gm orally as haust (drench) followed by 7 gm of this drug twice daily for several days. Supportive treatment by way of injecting 5 to 10 ml Vibelon i/m for 3 to 5 days can be given.

(v) Lastly, it is also advisable to give cobalt salt and a mixture of crushed maize @ 2 kg with ½ kg of molasses per day to make adequate supply of carbohydrate to sick animals.

Magnesium Deficiency

Magnesium is an important intracellular bivalent cation in the body of an individual. It is involved in many enzymic reactions or biologic activities. The different kinds of reactions in which the magnesium is involved are as follows :

(i) Membrane transport.

(ii) Activation of aminoacid, acetate or succinate.

(iii) Synthesis of protein, nucleic acid, fat or co-enzymes.

(iv) Generation and transmission of nerve impulses and contraction of muscles.

(v) Oxidative phosphorylation.

(vi) Activatior of alkaline phosphatase and adenosine triphosphatase (ATP) Normal level of magnesium in blood is 2.0 mg/100 ml.

Dietary deficiency causes low level of magnesium.

The main signs of magnesium deficiency are as follows:

There is a nervous hyperirritability. But high levels of magnesium cause depression, coma and death.

Grass tetany of adult cattle and hypomagnaesema of calves are its important clinical syndromes.

Hypomagesemia in Calves

A diet low in magnesium (like milk) causes this condition in calves. The level of magnesium is 0.7 mg/100 ml of blood and magnesium falls to one third of its normal value.

Signs

There are hyperirritability, tetany, scratching, fixed depression of the ears and opisthotonos, kicking of the belly, salivation and exophthalmos and tonic and clonic convulsions.

Pathology

Necrosis of myocardial fibres takes place. Later, calcification occurs at such sites. Metastatic calcification is seen in calves maintained on a diet low of magnesium. Calcification can also occur in the kidneys and muscles as well. Due to hypomagnesemia, calcification in the internal layer of heart and large blood vessels is noticed. Grossly, these lesions occur as slightly raised, light coloured plaques on the internal surfaces of heart etc. Renal tubules show toxic degenerative changes. Agonal haemorrhages are noticed in heart, intestinal and mesenteric serosae in calves.

Grass Tetany

Affected animals like cattle, sheep and rarely horses show tetanic convulsive seizures and incoordination of limbs. Asphyxia and agonal haemorrhages occur in the affected animals.

In winter tetany in cows, hypocalcaemia and hypomagnesemia are noticed. Nutritional deficiency of magnesium causes hypomagnesemia. A ration deficient in magnesium also produces metastatic calcification in foals.

Treatment/Management

Lactation tetany or hypomagnesemia arises from

deficiency of magnesium. The principle of treatment is to correct the magnesium level in the blood.

The following steps give the line of treatment :

(i) Inject mifex to sick animals at the doses of 450 ml to adult and 50- 150 ml to young ones.

(ii) Inject 100-200 ml of sterilized magnesium sulphate (25 to 50%) S/C.

(iii) Magnesium oxide @ 100 to 200 gm can be given to animals every day for a week and a combination for 12 per cent magnesium adipate and 5 per cent calcium gluconate at dose rate of 500 ml is also recommended. In making choice of drugs for administration in any diseases, the fact of avoiding overdosing with particular antibiotics or chemotherapeutic agents must be kept in mind.

This will always save the animals from toxic effects of particular drugs.

Azoturia (Monday Morning Disease)

If the working horses are given diet rich in carbohydrate and put to work after rest for a few days, a pathological state called azoturia characterized by sudden stiffness and lameness develops in them. The muscles of the croup and hind quarters become hard and the horses become reluctant to move and fall down. Degenerative changes like Zenker's degeneration develops in the muscles and myoglobin appears in the urine. The mortality can rise upto 40% and the sick animals require months to recover from muscular or renal changes or damages etc.

Signs

The main signs are as under :

(i) Appearance of myoglobin (a muscle pigment) in the urine.

(ii) Lameness or stiffness in the body. There is a sudden inability to move after first making movement by a few steps and the horses tremble, get transfixed in pain and, then, sweat due to pain.

(iii) The affected muscles may be hard in consistency as wood and are slightly swollen.

Such muscles in the body are in extreme cramp or tonic spasm. The painful state of muscles can be comparable to muscular cramps in human beings. Increase in the level of serum glutamic oraloacetic transminase and creatine phosphokinase occurs in azoturia.

Pathology

The cause of azoturia is not finally unsderstood. An accumulation of sarcolactic acid in the muscles which have stored sufficient glycogen due to intake of excessive carbohydrate during the period of rest is seen in the muscles. This acid causes swelling of the muscle fibres and compression of the capillaries. The state of oxygen deficiency is produced in the muscles.As a result, Zenker's necrosis occurs in the muscles due to excess of lactic acid and oxygen deficiency.

The muscles like sublumbar, gluteal, thigh or pectoral have pale yellowish tint and increased fragility or friability. They look like muscles of the fish. Haemorrhages are seen in the affected muscles.

Microscopically, the affected muscles show swelling of the sarcoplasm and loss of striations. The muscle fibres become hyaline or dark red in colour and also break into small fragments. Later, the leucocytes infiltrate into such affected muscles. When the lesions become chronic, there is an atrophy of the muscles and replacement of the muscle fibres by newly proliferated fibrous tissue. Degenerative changes are found in the heart muscles. The lungs show hypostatic congestion and renal tubules get plugged with muscle

pigments (myoglobins).

Diagnosis

It is based on the symptoms and lesions in the affected muscles and presence of myoglobin in the urine of the patients.

Treatment/Management

This disease affects the horses with characteristic degenerative change in muscles and kidneys etc. The treatment is directed as ameliorative measures. The measures are as follows:

(i) The horses should be given complete rest and they should never be put to exercise.

(ii) Give chloral hydrate 30 gm as a drench.

(iii) Alternatively inject largactil 5% soln. in dose of 4 to 6 ml (i/m).

(iv) Thiamine hydrochloride can be given in dose of 500 mg by i/m or i/m route.

(v) Antihistaminics like Avil or Zeet in doses of 10 to 20 ml can be given i/m.

(vi) Vitamin E can be given interamuscularly.

(vii) Fluid therapy (5% or 10% Dextrose solution) can also be given.

Post Parturient Haemoglobinuria

It is a disease of dairy cows within several weeks after calving and is characterized by anaemia, haemoglobinuria and intravascular haemolysis.

The causes are as under :

1. Diets low in phosphorus.
2. Copper and selenium nutritional imbalances.
3. Some other factors not fully understood.

Signs

1. Appearance of symptoms in the period 2-4 weeks after calving. Sudden onset of haemoglobinuria, inappetence and weakness in the calved animals
2. Fall in milk yield and discolouration of urine. Development of dehydration, pale mucous membrane, augumented pulse, rise of temperature upto 103.5°F, dry and firm faeces are noticed in the affected dams.
3. Other signs include dyspnoea, weakness, staggering and recumbency, ketosis and sloughing of the tail.
4. Inorganic phosphorus level in the affected animal may be as low as 0.4-1.5 mg/dl (0.13-0,400 m mol/L). Marked fall in the red cell count and haemoglobin value are noticed.
5. The urine in the affected animals is dark brown or black in colour.

Pathology

The main changes are :

1. Jaundiced carcass due to haemolytic icterus.
2. Thin and watery blood. Red cell count less than 2 million per cubic millimeter of blood (i.e., presence of severe anaemia).
3. Swollen liver with fatty changes (fatty degeneration and fatty infiltration) Centrilobular necrosis and midzonal fatty changes are noticed. In short, there is a presence of acute toxic hepatitis.

Diagnosis

It is based on the symptoms and changes in the blood and urine etc. Occurrence of haemolytic anaemia within 4 weeks of calving is a characteristic indication of postparturient haemoglobinuria. Haemolytic jaundice, haemoglobinuria, and acute toxic hepatitis marked by phosphataemia help diagnosis of postparturient

haemoglobinuria.

Treatment/Management

The steps are as given below :

1. Transfuse 5 litres of blood to severely affected cows. If required, transfusion may be repeated after 48 hours.
2. Fluid therapy (i.e., 5% dextrose) is also adopted to compensate the fluid loss.
3. Administer sodium acid phosphate (60 gms in 300 ml of distilled water by i/v route. The same dose in distilled water is also to be given to the patients by s/c route at the interval of 12 hours. A similar dose of sodium acid phosphate is also given orally.

Photosensitisation

It denotes the condition characterised by sentisisation of the skin (i.e., the superficial layers of lightly pigmented skin), mucosa and cornea following exposure to sunlight in animals like cattle and sheep. Photosensitivity in the skin arises from the presence of photodynamic substances in sufficient concentration in the skin. Porphyrins of both the animal and plant origin are known to originate from the decomposition of the pigments like haemoglobin and chlorophyll. These substances are sensitive to light. Presence of porphyrins in the blood is called porphyrinemia. If these are present in the urine, the condition is called porphyrinuria. Skin sensitisation to light arises from porphyrinemia.

The photodynamic substances may be exogenous or endogenous in nature.

Types of photosensitisation

These are :

(i) Congenital porphyria or pink tooth.
(ii) Primary photosensitisation.
(iii) Secondary photosensitization.

The condition of pink tooth occurs in cattle whose bones and teeth possess reddish or brown colour due to deposition of porphyrins in these tissues. Such pigments can also be found in some internal organs. Porphyrinuria is also noticed in the lead poisoning in dogs.

Aetiopatholgy

The causes and pathological changes are given in the table 35:

Table 35. Types Causes and Pathological Changes in Different types of Photosensitisation

Types of Photosensitisation	Causes	Pathological Changes
(1)	(2)	(3)
1. Congenital porphyria	1. Porphyrins formed in excess due to aberrant pigment synthesis.	1. Presence of porphyrins in the urine, bones and teeth. 2. Reddish or brown colour of the teeth etc.
2. Primary photosensitisation	1. Ingestion of exogenous photodynamic agents present in certain plants. The plants and the pigments are as under. Plants Photodynamic agents *Hypericum perforatum* Hypericin *Polygonum esculentum* *Fagopyrum esculentum* Fagopyrin	1. Presence of dermatitis in unpigmented skin. Animal patients are very much sensitive to light and prefer sheds. The liberated histamine from the damaged skin due to sun light leads to tissue oedema and cell death. Loss of skin is noticed. Subcutaneous oedema and enlargement of local lymph nodes are found. 2. Chemicals e.g., phenothiazine and sulfoxide acridine dyes. The metabolic product phenothiazine sulfoxide is a photodynamic substance in calves.
3. Hepatogenous photosensitisation	1. Phylloerythrin. It is an end product of chlorophyl metabolism in the body and is excreted in the bile. Hepatitis or biliary obstruction causes the accumulation in the skin	The damaged liver fails to remove phylloerythrine from the portal blood and this pigment is carried to the skin by circulating blood. When poorly pigmented skin is exposed to sun light,

Types of Photosensitisation	Causes	Pathological Changes
	which is reddened and sensitive to sunlight. The animals grazing green pasture develop a type of sensitivity called hepatogenous photosensitisation. A toxic injury to the livers is noticed in nearly all types of hepatogenous photosensitivity. The plants containing hepato-toxins are : 1. *Agave lechuguilla* 2. *Lantana camara and Lippia rehamanni* 3. Certain chemicals like carbon tetrachloride and phenanthridium. Water contaminated by pathogenic fungi may also lead to photosensitivity. 4. Toxic tubular nephritis and toxic hepatitis, oedema of the ears and face and lymphocytic infiltration in the portal spaces are noticed in *Agave lechuguilla* toxicity. The changes noticed in *Lantana camara* toxicity are bloody faeces, icterus, haemorrhagic gastroentritis, pseudomembranes in the intestine, hepatitis, dermatitis and itching. Note: Congenital photosensitivity owing to a defect in the excretion of bile pigments is noticed in lambs.	photosenstivity and development of skin lesions are noticed The skin lesions are as follows : 1. Erythema and oedema. 2. The skin presents a weeping surface and gangrenous changes mostly on the dorsum of the body. Ears, conjunctiva, eyelids, muzzle face, and perineum are the predilection seats for the skin photosensitivity lesions. Rubbing the affected skin against bushes lead to lacerated skin. 1. Drooping of the ears due to oedema, dyspnoea due to nasal obstruction and dysphagia due to swelling of the lips are some signs of such photosensitivity. 2. Swelling of the eyelids, redness of the muzzle, fissuring and sloughing of the large patches of the skin due to necrosis or gangrene are also seen.

Diagnosis

It is based on the followings :

1. Skin lesions on the unpigmented areas.

2. Presence of porphyrinemia and porphyrinuria.

Treatment/Management

Some steps of treatment are as follows :

1. Remove the animals from direct sunlight.
2. Prevent the animals, from taking the toxic water, soaked hay or grass contaminated with fungi.
3. Administer laxatives to remove toxic materials from the gut.
4. The animals are given anti-histaminics immediately in adequate doses.
5. Use antibiotics to check the development of septicaemia in animal patients.
6. Administer 5% Dextrose as fluid therapy to dilute and eliminate the absorbed toxins.
7. Calcium borogluconate (25%) at the dose rate of 250 ml intravenously and the same dose by subcutaneous route should also be given.
8. Liver extract (e.g., livogen, Belamyl) at the rate of 10 ml intramuscularly is given on the alternate days.

External application of drugs on skin lesions include use of Himax and Herbex.

Equine Colic

It is a disease of horses recognized by the presence of abdominal pain. Colic arises from distension of the stomach or intestines due to accumulation of ingesta, gas and fluid. Transient distension also occurs due to spasm or increased peristalsis in the intestine.

The chief causes of colic in horses are the following :

1. Low grade roughage, lush green feed, exhaustion, excessive perspiration and. retained meconium etc. Distension results from accumulation of ingesta as noticed

in the impaction of the large intestine. Accumulation of gas can cause intestinal colic or flatulent colic. Gastric distension results from accumulation of food in its cavity.

2. Engorgement due to pyloric obstruction. Gastric dilatation can arise from accumulation of fluid.
3. Fibrin balls, enteroliths, foreign bodies, volvulus, torsion, intussusception, diaphragmatic hernia and pedunculated lipomas. Impaction of ieocaeal valve can be also noticed. Fluid accumulation leads to acute intestinal obstruction. Verminous mesenteric arteritis can also cause distension of intestine from accumulation of fluid.
4. Bacterial infection, strongylosis, viral infection (equine viral arteritis), chemical poisons and excitement etc. The infections or poisons cause enteritis and spasm and increased peristaisis is noticed in the affected horses.

Signs

The main lesions are :

1. Restlessness, pawing or stamping or kicking at the belly and frequent getting up and lying down. The horses look at the flanks, can roll and lie on the back and can attempt to sit like dogs. Protrusion of penis without urination is seen in geldings. A horse may go down and roll on the ground.
2. Playing with water without actual drinking. Bouts of pain lasting for over 10 minutes are noticed. Profuse sweating and sobbing respiration in the horses are present. There can be increase in the respiratory or the pulse rate. Pain is severely increased in colic and failure to respond to analgesics is considered to be a grave prognosis. Vomiting in the horses indicates a very serious sign of this disease.

Pathology

Rupture of the stomach or intestine can lead to death of the horse. Large quantities of toxic ingesta or faecal contents in the peritoneal cavity can cause choking or death within a few hours. The animals get exhausted due to pain or autointoxication. There is a marked degenerative change in the mucosae and this change is more evident in the obstructive colic. The damage is more marked in the intestine. Distension of the intestine is seen in cases of colic due to peritonitis and verminous measenteric arteritis. Verminous arteritis causes an infarction of the gut wall.

Diagnosis

It is based on the symptoms like severe pain, absence of gut sounds and defaecation (acute obstruction of the small intestine), recurrent attacks of pain (verminous mesenteric arteritis), fever and toxaemia (infected verminous arteritis), flank watching and lying down frequently (as seen in subacute or chronic obstruction) Rupture of the gut or deposits of faecal contents in the peritoneal cavity can be seen postmortem.

Treatment

In cases of flatulent colic, gases from the stomach should be removed by passing stomach tube. A funnel can be attached to stomach tube and the following drugs can be used

Oil terebinth	20 ml
Acid carbolic	20 ml
Oil line	300 ml

Timpol 100 ml is given as drench with lukewarm water. Anti-allergic drug lke Avil-5 to 10 ml can be given by intramuscular route.

In colic due to impaction of large intestine, the following drug can be used

Aloes barb - 15 gm, Aqua - Q.S., Oil terebinth - 60 ml Chloral hydras - 30 gm,

Oleum lini - 500ml.

Mft. Haust. Sig. Dissolve aloes and chloral hydras in hot water and shake well with oil of terebinth and linseed oil and the mixture should be given by drench. In case of spasmodic colic, drugs like Baralgan can be used. The drugs are repeated after three hours if it is so needed.

Preparation to Treat Impaction of Colon with Colic

Aloes barb	**15 gm**
Aqua ferv (warm water)	**A.S.**
Ol. Terebinth	**60 ml**
Chlonal Hydras	**30 gm**
Ol. lini	**500 ml**

R/

Mft. haust. sig. Dissolve aloes and chloral hydras in hot water and shake well with oil of Turpentine and linseed oil. Administrator as a dresh slowly.

Preparation to Treat Spasmodic Colic.

R/

Choloral Hydras	30 gm
Ol. Terebinth	30 ml
Ol. Lini	500 ml
Mft. Haust. sig. Administrater by stomach tube or	
inj. Novalgin (Hoechst)	
inj. Beralgan (Hoechst)	15 - 20 ml intramuscularly

Table36. Physiological Values of Domestic Animals

Parameters	Cow	Buffalo	Mare	Ewe	Bitch
Temperature °C °F	 38.5 101.5	 38.5 101.5	 38 100.5	 39 102.5	 38.5 101.5
Pulse/minute	60-70	50-70	30-40	70-90	90-120
Respiration/minute	10-30	20-30	10-12	10-20	22
Age of puberty	14-24 months	14-24 months	15-24 months	7-12 months	6-12 months
Age of sexual maturity	30 months	30 months	36 months	12-18 months	9-12 months
Length of oestrus Cycle	21 days (18-24 days)	21 days (21-23 days)	21 days (seasonally polyestrus)	17 days (seasonally polyestrus)	21 days (16-23 days)
Length of oestrus	18 hours (12-28 hours)	20 hours	5 days (4-7 days)	18-24 hours after onset of oestrus	2-3 days after onset of oestrus
Optimum service time	10-16 hrs after onset of oestrus	10-16 hrs after onset of oestrus	2-3 days after onset of oestrus	18-24 hrs after onset of oestrus	2-3 days after onset of oestrus
Gestation period	282 days (278-293 days)	310 days	336 days (330-345 days)	148 days (144-151 days)	63 days
Time for breeding (for health, prevention of infertility and regular supply of milk)	45-90 days post partum	60-90 days post partum	25-30 days or second oestrus	First oestrus (Sept-Nov)	First oestrus (Feb-Apr and Aug-Oct)

Appendix

Common Chemotherapeutic Agents and Vaccines

A tabular statement or box pattern of some common drugs and vaccines has been given in view of the requests of the students and veterinarians to check the accuracy of the treatment adopted as per history of animal patients and fatal cases. Such routine practice to correlate the symptoms, lesions and diagnosis made in animal patients with the line of treatment followed will make one a successful veterinary practitioner. Efforts have been made to include medicines, prophylactic agents and patent preparations needed by a clinician in every day practice in Tables 37, 38 and 39.

Table 37. Some information about Chemotherapeutic agents

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
Antibiotics 1. Benzathine Penicillin and derivatives	Longacillin, 6,12,24,48 lakh units injection ii. Pronapen 20 lakh units iii. Pronapem-40 lakh units iv. Bestripen-40 lakh units Procillin- vet. 20 and 40 lakh units vi. Frotifed Procaine penicillin (inj).	All species of animals. Dogs/Cats	i. 4000-10000 units per kg. body weight or 5mg/kg.bwt every week. Young animals and small animals are given higher doses. 2-4 Lakh units.	In case of Benzyl penicillin 6-12 hours and for procaine penicillin 24 hourly.	Parenteral (I/M)	Anthrax, B.Q. tetanus. malignant oedema, bacillary haemoglobinuna, braxy, F.M.D., entero toxaemia, F.M.D., foot rot, joint ill or navel ill.
2. Cloxacillin & ampicillin	i. Inclox 500mg. 1000 mg. and 2000 mg.injection ii. Binocin-1000 & 2000 mg. injection iii. Catlox inj. iv. Betalactin inj.	All species -do-	i. Large animals 4-7 mg/kg Body weight ii. Small animals 4-10 mg/kg iii. 5mg/kg.b. wt.	12 hourly	I/M	Anthrax,B.Q.,H.S. , tetanus, braxy etc.

Chemotherapeutic Agents	Pharmaceutical products (drugs) specialities	Species	Dose rate	Repetition	Route of Administration	Indications
3. Ampicillin	i. Marcocillin inj. ii. Albercillin inj. iii Conampi inj. iv. Eskycillin inj. v. Dynacil-Vet.inj. vi. Catcillin inj. vii. Vetampin inj. viii. Stancillin inj.	All species.	2-7 mg/kg Body weight.	12-24 hourly 3-5days.	I/M	Respiratory, urinary, and genital tract infections. Anthrax, B.Q., & tetanus etc.
4. Streptomycin and penicillin	i. Combiotic Large and small vial inj. ii. Munomycin forte inj. iii. Dicrysticin-s Large vial iv. Vetopen inj. 0.5 gm, 1.0 gm, 2.5 gm	Horse/foal/cow /calf /sheep/ goats/ Dogs and cats Large animals Small animals	10 mg/kg Body weight 10-20 mg/kg Body weight 0.5gm/50kg bwt 0.25mg/5kg bwt	24 hourly	I/M	Urinary, respiratory, gastrointestinal, tract infections. T.B., Johne's disease, actinomycosis and actinobacillosis etc.
5. Kanamycin	Kanacin inj. 1 gm	All species	7-10 mg/kg Body weight	12 hourly	I/M	Urinary tract infection. mastitis, respiratory tract infection, metritis cervicitis etc.
6. Gentamycin sulphate I.P.	i. Gentamycin inj. ii. Genticin inj. iii. Gentim inj.	All species Small animals Large animals	 2-4 mg/kg bwt. 1-2 mg/kg bwt.	12 hourly, for 7-9 days	I/M	Respiratory tract, urinary tract, genital tract infections, mastitis etc.

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
1	2	3	4	5	6	7
	vi.Catlogenta	Do	Do	Do	Do	Do
7. Amoxycillin + Cloxacillin	I. Moxel inj. 2000 mg.	All species.	5-10 mg/kg. Body weight.	24 hourly	I/M	Respiratory problem, H.S urinary tract infection etc.
8.Oxytetracyclin	i. Terramycin inj. ii. Tetra inj. iii. Oxy. Steclin inj. iv.Terramycin-LA v.Oxyvet-LA vi. Telon-LA vii.Veterinary oxytetracyclin inj.	All species Do Cattle/Horses/ Buffaloes.	5-10 mg/kg Body weight. 10-20 mg./kg. Body weight. 5-10 mg./kg. bwt.	24 hourly At 72 hrs. interval	I/M Do	H.S., B.Q.,tetanus, respiratory, urinary and genital tract infections etc.actinomycosis, anaplasmosis, leptospirosis etc.
9. Tetracyclin hydrochloride	Hostacyclin water soluble powder Tetracycline hydrochloride bolus Steclin bolus.	Horse, cattle, sheep and pig C/H/S/G pigs Do	5 to 20 gm. or 2.5 mg/30 kg bwt. depending on weight of the animal 4-6 boluses daily. Do	24 hourly 24 hourly /48 hourly. Do Do	Oral use restricted /intra-uterine. Do Do	Pasteurellosis, vibriosis, anaplasmosis, feline in fluenza and goat pneumonia etc.

Chemotherapeutic Agents	Pharmaceutical products (drugs) specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
10. Chlortetracyclin hydrochlor	Aureomycin	Small animals	20-25 mg/kg bwt.	12 hourly in two divided doses.	Orally	Mastitis, strangles, pasteurellosis anthrax, leptospirosis, listeriosis, glanders, samonella infection.
1	2	3	4	5	6	7
Do	Do	Large animals	7-10mg/kg bwt.	12 hourly	Do	Do
11. Chloramphenicol	Neochlor inj.	Large animals S/G/P/F/Calves Do	2-4 mg/kg. bwt 4-10 mg/kg. Body weight Do	24 hourly -do-	I/M	Calf diphtheria, pasteurellosis, footrot, virus pneumonia, coliform or salmonella infection. metritis, kennel cough, mastitis, infectious bovine keratitis, otitis, eye infections etc.
12. Norfloxacin	Neonox-200 Dispersible tab.	C/B D/cats	1-2 tab in distilled water 1 tab in distilled water	48 hourly 24 hourly	Intraterine Orally after meals	All bacterial infections.

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
13. Cephalosporins	Keflong inj. (0.5 gm., 1gm.)	All species	10-20 mg/kg Body weight	24 hourly	I/M	Pneumonia, H.S., metritis, nephritis, cystitis, septicaemia and otitis.
14. Pefloxacin methene sulphonate dihydrate	Pelwin inj. Peflon inj.	All species	As directed by physician	-----	I/V	All microbial infections (e.g., H S)
15. Enorofloxacin 10%	Meriquin inj. Enrosin inj.10% Floxidin inj.	All species cattle / Buffloes.	Iml/40kg.bwt. Do Do	24 hourly 24 hourly Do	I/M Do Do	Do
16. Tylosin	Tylosin inj.	Cattle Pigs,dogs & cats.	4-10 mg/kg bwt 2-10 mg/kg bwt.	Do	I/M	Do
1	**2**	**3**	**4**	**5**	**6**	**7**
17. Plymyxin B	Plymyxin B	Large animals	40,000 units/kg. body weight 10,000 units/kg. body weight	24 hourly	Orally I/M	Mastitis, septicaemia & otitis externa.
18. Lyncomycin	Lyncomycin inj.	Dogs and cats Do Pigs	20 mg/kg.bwt. 20 mg/kg.bwt. 4-10mg/kg.bwt.	12 hourly 24 hourly 24 hourly	Orally I/M I/M	Mycoplasma infection erisipelothrix infection, tetanus, infectious arthritis, enterotoxaemia etc.

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
19. Sulphadimidine or Sulphamezathine	Sulpha drugs 1. Diadin inj. (33.3%) ii. Vesadine inj. (33.3%) iii. Suphadimidine sodium inj. (33.3%) iv. Diadin tablet (5 gm) v. Sulpha bolus.	All species H/C/S/G/P etc. „ „ D/C/Rabbits/ Other small animals H/C/S/G/P 1 gm./7.5 kg bwt.	1 gm./7.5 kg bwt. 30 ml/50 kg. Body weight as initial dose followed by half of this dose for 2 days 3 ml/5 kg. B. wt. initially followed by 1.5 ml/5kg. B. weight daily. 0.2gm/kg.B.wt.f ollwed by half dose. -do-	24 hourly 24 hourly 24 hourly -do-	Orally S/C, I/V and I/periton eal. „ Orally in divided doses -do-	H/S, foot rot, calf pneumonia, calf scours, pneumonia, strangles, joints ill in foals, speticaemia, metritis, necrotic enteritis, coccidiosis etc. Enteritis, coccidiosis
Do	-do-	Dogs/Cats/Rabbits and other small animals	1 gm/5 kg.bwt. followed by 0.5 gm. kg. bwt.	-do-	Orally	Strangles metritis, pneumonia, H.S. etc.

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
20. Sulphathalazole Phthalylsulph	vi. Thalazole Tab. (5gm)	Calves/Foals	7.5 g.m/50 kg. b. wt. for 3-4 days	24 hourly	Orally	Indicated in bacterial infections of alimentary tract.
		Pigs		24 hourly	Orally	
		Dogs	1 gm/9 kg. B. weight daily upto 6 days	24 hourly	Orally	
		Cats	1 gm/7 kg. B. weight daily upto 6 days	24 hourly	Orally	
			0.5 gm/3 kg. B. wt. daily upto 6 days			
21. Sulphonamides and Trimethoprim	i. Atrima bolus (1.2 gm, 2.5gm)	Cows/Mares/Foals/ Ewes/	30 mg/kg Bwt. in divided doses	12 hourly for 3-5 days	Orally	Respiratory and urinary alimentary tract disease, etc.
				-do-		
	ii. Sulcoprim bolus	Dogs C/H	15 mg/ kg. B. weight 2-4 bolus daily	Morning-evening for 5 days	-do-	
	iii. Oriprim bolus	S/G/Calves Horse and cattle	0.5- 1 bolus daily Two boluses	-do-	-do-	
	iv. Oriprim inj. I/M	Small animals	1/2-1 bolus	injectable 24 hourly		
	v. Sulprim 24 inj.	All species	1 ml/20 kg body weight daily	Do	I/M I/M	Actinobacillosis
		Do	1 ml/ 16 kg bwt.			

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
1	2	3	4	5	6	7
	vi. Oriprim I/V	Small animals Large animals	2-5 ml daily 15-30 ml daily	24 hourly 24 hourly	I/V I/V	Actinomycosis, colibacillosis, infectious polyarthritis. Toxoplasmosis, Coccidiosis, G.I. tract infection, urogenital tract infection
22. Antipyretics, analgesics and antiflammatory drugs Diclofenac Sodium Do Do	1.Novalgin inj. 2. Bolin inj. 3. Bovalgin inj. 4. Analgin inj. 5. Paracetol inj. 6.Catlofenac inj 7.Zobid inj. 8. Nufenac inj. 9. Esgipyrin inj.	All species Horse Cattle Foals/Calves Pigs Sheep/goat Dog/cat Large Animals Small Animals C/H Foal/calve Large animals Small animals	8-10 ml/100 kg bwt./ 20-60 ml 20-40 ml 5-15 ml 30 ml 2-8 ml 1-5 ml 1 ml/25 kg bwt. 0.5 to 2 ml 15-20 ml 5-10 ml 1-15 ml 1-5 ml	12-24 hourly	I/M	In high fever, myositis and painful conditions.

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
23. B.complex with liver extract inj.	i. Liverjet inj. ii. Livogen inj. iii. Belamyl inj. + iv. Bivinal forte inj. v. Pepsid inj. vi. Vibelan inj.	Large animals + Small animals	5-10 ml daily + 1-2 ml daily	 24 hourly	 I/M	Anorexia, liver disorder, hepatitis, Jaundice. debility and exhaustion, to maintain normal erythropoiesis and check anaemia.
24.B.complex inj. **$B_1B_6B_{12}$** and other water soluble vitamins	i. Polybion inj. ii. M.V.I iii. Conciplex iv. Neurobion inj. v. Vibelon inj. vi. Triradisol. inj. H-500 vii. Triradisol inj. H-1000 viii. Neuroxin -B12 Inj.	Large animals. Small animals Large animals Do Do C/B/H Dog	5-10 ml daily. 2-3 ml daily. 5-10 ml Do Do 5-10 ml 2-3 ml	24 hourly or alternate day. 24 hourly or alternate day Do Do Do Do	I/M I/M Do Do	Neurological disorder and paralysis Do Do Do

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
25. Anti allergic/ anti histaminics **Pheniramine maleate** Chlorphenriamine maleate Promethazine Hcl Promethazine Hcl Anti-inflammatory/anti-anaphylactic shock/Anti toxic.	i. Avil inj. ii. Zeet inj. iii. Cadistin inj. iv. Catlan v. Promethazine inj. Phenergan inj.	All species Large animals Small animals.	5-10 ml daily 1/2-1 ml daily.	24 hourly.	I/M	Allergic reaction allergic pulmonary emphysema, asthma uticaria, insect bite, taileczema, dermatitis. ruminanl atony. tympany and bloat in ruminants, acute septicmetritis, pregnancy toxaemia, shock etc.
Dexamethasone Sodium phosphate Betamethasone	i. Dexona inj. ii. Dexavet inj. iii. Curadex inj. iv. Cadex inj. v. Dexamethasone inj.	All species Cattle/Buffaloes/Horses Calves/Pigs/Sheep/ Goat/ Dogs and Cats C/ H /B S/G/Calves C/ B/H Dog/Cat	4-20 mg daily 2-4 mg daily 0.5 to 2 mg. daily 8-20 mg daily 2-4 mg daily 4-6 ml 0.25-1 ml.	24 hourly in severe conditions it may be repeated in 12 hour.	I/M	Ketosis, inflammation of respiratory tract, urogenital tract, local inflammatory condition arthritis, surgica shocks, anaphylactic shock, traumatic shock pregnancy toxaemia, pneumonic pasteurellosis, H.S etc.

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
			As given above			
Prednisolone	vi. Betanesol inj vii. Betacotril inj. viii. Hostacortin- H	All species Do Cattle/horses Calves/Pigs Dogs/Cats	Do 50-200 mg daily 25-30 mg daily 10-30 daily.			
26. Antiemetics/ Anti-convulsants Chlorpromazine Hcl Trifluopromazine Hcl. Trimeprazine tart Prochlorperazine mesylate Metoclopramide Hcl	i. Largactil ii. Siquil iii.Vallergan iv. Stemitil inj. v. Perinorm inj.	All species Small animals Dog, Cattle, Horse Small animals (Dog & cats).	2.5 mg/kg.bwt. 1-2 mg/Ib.bwt. 10 mg/100 Ib bwt. 10-15 mg/100 Ibs Body weight 1 amp.daily or twice daily depending upon the condition.	12 or 24 hours Do Do do Do Do	I/M or I/V I/M I/M I/M or I/V I/M Do	Vomiting & restlessness.

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
27. Antispasmodic	i. Pethidine Hydrochloride	Horse/cattle	150 mg to 200mg/50 kg bwt.	Repitition depends upon condition	**I/M**	Painful condition, colic, difficult parturition etc.
		Pigs, Dogs, Cats	3-5 mg/kg.B.wt.		**I/M**	
			10 mg/kg.B. wt.	**-Do-**	**I/M**	
		Large animals	25 mg/kg.B.wt.	**-Do-**	**Orally**	
	ii. Polygesic inj.	D/C	5-10		**S/C, I/V**	**-do-**
		Foal/Calf	mg/kg.B.wt.		**S/C/ or**	**-do-**
		Large animals	20-60 ml	**-Do-**	**I/M**	
	iii. Ridalpin inj.	Small animals	1-2 ml		**I/M**	
		Calf/foal	5-15 ml	Repeated after 3 to 4 weeks	-do-	
			20-60 ml		-do-	
	iv. Trigan-D inj.	Large animals.	2-4 ml	,,	-do-	
			2-15 ml	-Do-		
			10 ml			
28. Anti trematodal (Fluke remedies)	i. Corbon tetrachloride	Cattle, Sheep and goats	5 ml	Repeated after 2 weeks	Orally	Liver flukes , paramphistomiasis etc.
			1 ml	Repeated after	,,	
	ii. Distodin (1gm) Tab.	Adult animals	1 gm tab/100 kg.b.wt.	2-3 weeks	,,	
	Distodin 100 mg. tab	Small animals	100 mg tab(1-2 tab.)		Orally	**-do-**
	iii. Trodex inj.,					

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
Nitroxyl Oxyclozanide Rafoxanide	Oxyclozanide 3. Tolzan-F susp. 4.Amfanide	Large animals Cattle Cattle & buffaloes Sheep & goats.	 10 ml. 10 ml/kg.b.wt. 033 ml/kg.b.wt. 7.5 mg/kg b.wt.	As desired by physician.	S/C Orally Orally	-do-
Oxyclozanide Oxyclozanide + Tetramisole hel	5. Hexanide 6. Nilzan 7. Fasinex	C/B Swines Cattle + sheep C/B/S/G	10 mg/kg bwt. 0.33 ml/bwt. kg. do (60-90 ml) as total dose 1-4 tablets.	As desired by physician. do do	Orally Do Do Do Do	Do do Flukes.& G.I.tract nematodes. Do
Lithium antimony thiomalate	Anthiomaline inj.	Adult Cattle	20 ml.	Repeat twice a week Maximum 8 Inj.	I/M deep	Nasal Granuloma (NG)
Sodium antimony tartrate	Tartarematic	Do	1 ml (4%) / 40 kg b.wt. or 20 ml. 2-3 % as total dose	Do	I/V	Do

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
29. **Antinematodal (Round worm remedies).** **Piperazine salt** **Fenbendazole**	1. Vermex 2. Piperazine Adipate 44.4 % 3. Helmacid liquid 4. Peperazine 45 % liquid 5. Panacur bolus (2.5mg) 6. Panacur tab. (150mg)	Cattle & buffaloes Horses/swines Calves/ Foals Dogs & Cats Cattle, buffaloes /horse Sheep/ Goats/ Pigs. Dog & cats	15-30 ml/30 kg. B.wt. 15 ml/30 kg. B.wt. 15 ml/25 kg. B.wt. 2.5 ml/10 kg. B.wt. 5-10 mg/kg B.wt. 5 mg/kg B.wt. 15-25 mg./kg.bwt	Repeat after 3 weeks Do	Orally Orally	Round worms of poultry, cattle, buffaloes horses, swine, dogs, cats etc.
Mebendazole **Fenbendazole**	7. Wormin 500mg. bolus 8. Curaminth 9. Zodex	C/B C/H do Calves/sheep/	5-10 mg/kg.b.wt.upto 5-10	Do	Orally	All round worms i.e., Round worms, Whipworms,

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
Morental citrate		pigs/goats	mg/kg.b.wt.			hookworms etc.
	10. Banminth II forte (1gm. bolus) Tablet		4-8 boluses	For 3 days	Orally	Do
		All species	2-4 boluses	continuously		
Levamisole hcl						
	11. Kalmisole vet tablet		1 bolus/200 kg b.wt.	Do	Do	Do
Albendazole		Livestock				Round worms & Flukes
	12. Albomar suspension /powder		1 tablet/20 kg but.	Do	Orally	
		Do				Tapeworm infestation
	13. Analog suspension		One tablet/20 kg b.wt.	Do Repitition	Orally	
30. Anticestodal drug		C/B/H/S/G/P				
Ivermectin	14. Dicestal					
		Large animals	0.33ml/kg or 5-7.5 mg/kg b.wt.	After 2-3 weeks		All endo and ecto parasitic infections
	15. (a) Ivomec inj.	Horses D/C/S		After weekly		
		All species		inetervals 2-3	S/C	
	(b) Mectin inj.	C/H/D/S/	1 ml/5 kg./bwt.	inj.		Babesiosis + theileriosis etc.
31. Anti-haemo-protozoal				After 48 to 72		Do
4, 4'-diamidino-			0.5gm/4kg/bwt	hours.	I/M deep	
	i. Berenil	All species				
	ii. Pronil-H.	(cattle)	0.5 gm/3kg/bwt.			
		Small animals	1 ml/50 kg/bwt. or 0.2 mg/kg bwt.			

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
			0.8 to 1.6 gm/100 kg. B.W 3 to 4 mg/kg bwt			
diazoaminobenzne diaceturate Sodium antimony tartarate Quinapyramine sulphate and Q.P.chloride	iii. Tartaremitic	Horse/Cattle/ Camels	1ml 4%/40kg bwt.or 20 ml. (2-3%sol)	Twice week for 2 weeks	I/V	Surra
	iv. Trypnil inj.			After 3 months	S/C	Surra
	v. Triqin inj.	Horse/Cattle	3mg./kg body wt.			
	vi. Tribexine inj	Camels	or 0.025 ml/kg			
32. Bupravaquone	vii. Butalex	Cattle	Bwt.or (10-12 ml as total douse)	Within 48-72 hours of the initial injection	I./M	Theileriasis
33. Antianemic drugs				Twice weekly		Anaemia, Degnala disease and debilitating conditions
	i. Acetylarsan (23.6%)	Large animals	1 ml/20kg.body wt.	Do	I/M	
	ii. Cal-D rubra	Dogs and Cats		Thrice daily	I/M	
	iii.Sharcopherol	Small animals		Twice daily	Orally	Do
	iv.Neuroxin-B12	Large animals		Thrice daily	Orally	Do
	v. Imferon Inj.	Small animals	5 to 10 ml	Alternate day	Orally	Do
			1to 2 ml	Alternate day daily	I/M	Do
		Large animals	10 ml			Do
	vi. Fesol cap.	Small animals	50 ml		I/M	Do
		Dogs and Cats	5-10 ml		Orally	
			10 ml			

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
			1 to 2 ml 1 cap.			
34. Intramammary infusions Pencillin Streptomycin + Hydrocortisone Ampicillin + Cloxacillin + Dexamethasone	i. Pendistrin-SH ii. Vetclox plus iii. Penicur-D iv. Alciclox v. Campidex vi. Vetmas	Large animals Cattle and buffaloes (Acute mastitis) Chronic Mastitis.	1 tube every 12 hrs. after milking 1 tube every 12 hours after milking. Do	5-7 days Do 3-5 days	I/mammary Do Do "	Mastitis(acute and chronic) Do Do " "
35. Antiectoparasites	1. Ascabiol (emulsion) 2. Malathion 3. Lorexane cream 4. Butox 5. Pestoban	-do- Dogs All species All species All species	Apply on affected surface with a soft brush 0.5-1% suspension 1% solution and 0.6% dust . 2ml/litre of water for spray or dip.	Apply on alternate day Spray or apply on the dogs. Applied as a spray & powder should be sprinkled on body. May be repeated after 2 weeks.	" Topical application -do- Used as spray as toping up	Sarcoptic mange,parasitic otitis,removal of lice. Ectoparasites of livestock poultry and pets. Ectoparasites Effective at gainsticks, mites, lice and flies, These drugs stimulate, increase and maintain milk yield in the

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
36. Galactogauge	Leptaden tab.	Large animals	3ml/litre of water for toping up.	Twice daily for 2 to3 weeks or more	Orally	animals. Do
	ii. Dudhdan tabs	Cattle Buffaloes		Twice daily for 2	Orally	-do-
	iii. Galog tabs.	Goat and ewes	10 tabs.	Weeks	Orally	-do-
	iv. Calsus Plus liquid.	Large animals	3-5 tabs.	Do		-do-
37. Rumenotoric drugs	1.Bovirum bolus	Cattle / Buffalo	1 tab		-do-	
	2.Rumenton		Do		-do-	
	3.Bio-spur bolus		5-10 ml	For a week	-do-	
			3-4 boluses per day	-do-		
			-do-			
			2-4 boluses			
38. Anti-convu-Isants(chorea)	i. Mysoline tab.	Small animals	250 mg/5kg.B.wt.	Total dose divided in to 2 parts given at intervals of 12 hours.	Orally	Epilepsy, epileptic form of convulsions, and hysteria Nervous disorders convulsion, cholrea.eclampsia,tetanty, poisoning by strychnine and cocaine.
	ii. Gardenal tab.	Dog and Cats	30-300 mg according to bwt.	30 mg. tab-Thrice daily	Do	
	iii. Lardopa (500mg) tab.	Dogs		once daily	Do	

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
	iv. Pacitane (2mg)	Dogs	1/2 tablet	followed Twice daily	Do	
	v. Amantrel (100mg) tab.	Dogs	1 tab.	Thrice daily		
			1 tab.	Thrice daily		
39. Drugs used in calcium therapy	i. Calborol Calcium (borogluconate)	Cattle & Horse	350 ml to 450 ml (Warmed to body tem)	Repeat after 12 hours if necessary	S/C or I/Vslow	Hypocalcaemia, (milk fever) Lactation tetanty Acute reaction in cattle and sheep due to anthelmintics.(e.g., CTC)
	ii. Calcium boro - gluconate inj.	Sheep/Pigs/Goat	60 ml.	24 hours		
	iii. Mifex	Pig/Goats/Sheep	20 to 50 ml		-do-	-do-
	iv. Thiacal	C/B	300-450 ml	-do-	-do-	-do-
		Cattle & buffaloes	200 to 350 ml	-do-	-do-	-do-
	Osto calcium	C/B	250-450 ml			-do-
	B12 liquid(500ml)		100 ml	Daily for 15 to 30 days	Orally	
			25 to 75 ml		-do-	-do-
		Cattle & buffaloes	100 ml		-do-	-do-
	Cobacal-D Inj.	Calves	10 to 20 ml	Thrice a week for 1 to 2 weeks	Orally I/M	-do-
		Small animals	10 to 15 ml.			-do-
		Cattle/Buffalo				

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
40. Fluid therapy	i. 5% Dextrose	All species	25 ml to 50 ml/kg Body wt.	12 or 24 hours depending upon severity	I/V	1. In cases of dehydration, from vomiting, diarrhoea &
	ii. 5 to 10% Dextrose saline or (DNS)	Do	Do	of deydration Depending upon the severity of the	Do	enteritis. 2. Jaundice, hepatitis, ketosis loss of appetite and impaired
	iii. Rintose solution	C/B/H	500-1000 ml	disease (e.g., jaundice)	Do	digestion etc. Do
	iv. Ringer's lactate	S/G Do	100-200 ml Do	Do 24 hrly.3 to 4 days	Do	Do
	v. Dextrose (5%,10%) (20%,25%)	Large animals Sheep and goat	450 ml. 1000 ml. 100-150 ml	Do	Do	Do
	vi. Electrovet	Small animals	25 to 50 ml.	Do	Do	
	vii. Lecrivet	C/B/H Large animals	500-2000 ml. 450 ml	Do	Do	Do
				Do Do		Do

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
41. Antihaemorrhagic drugs	i. Styptobion	Dogs/Cats	1-2 ml.	Twice daily	Do	Epistaxis, haemorrage
	ii. Styptochrome (inj.)	Large animals	10 ml.	Twice daily	I/M	etc.
	iii. Revice (inj.)				I/M	Do
	iv. Styptocid	Do	Do	Do		Do
	v. Chromostat	Do	Do	Do	Do	Do
	vi. Kalpin (inj.)	Do	Do	Do	Do	
42. Antibloat						
	i. Bloatosil	Cattle buffaloes horse donkey sheep & goat	100 ml. as a drench	Repeated after 6-12 hrs or 24 hours if needed	Do Orally	Do Do Tympany, frothy bloat
	ii. Blotinox			Do		Do
		Cattle & buffaloes	100 ml.		Do	
		Horse & Donkey		Do	Orally	Do
			50-100 ml.	Do		Do
		Sheep & Goat			Do	
			Do	Do		Do
	iii. Tympol	Cattle & buffaloes	50 mg.	Do	Orally	Do
	iv. Blatal	Sheep & goat		Do	Do	Do
		Large animals	20-30 ml.		Do	
			110 ml.			

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
43. Anti diarrhoeal suspension	1. Chlorostrep	Calves/Colts	40-55 ml	Do	Orally	Gastroenteritis,
		Dogs & Cats	1-2 teaspoon ful.	Thrice daily for	Orally	diarrhoea dysentery
	2. Diarmycin-n	Neonates	5-10 ml.	5 to 7 days	Do	etc.
	suspension	,,	,,	Twice daily	Do	
	3. Enterostrep	,,	,,	Do	Orally	
	suspension		1-2 tablets	Do	Orally	
	4. Kaltin	,,	Do	In morning &	Do	
	suspension	,,	Do	evening	Do	
	5. Pesulin	Dogs & cats	1-2 teaspoon ful	3-4times daily	Do	
	suspension	neonates	1-2 teaspoon ful	As per	Orally	
	6. Furoxone	,,		requirement	Do	Orally
	suspension	,,				Do
	7. Streptomag-	Dogs & cats	Do	Do	Do	Do
	ma suspension	Do	Do	Do	Do	
	8. Drivet					Do
	suspension					
	9. Gramogyl	Do	Do	Do	Do	
	10. Unimycin	Do	Do	Do	Do	Do
	bolus	Cattle + Buffalo	One bolus/45	Given for 3		Do
	11. Diarosil		kg bwt.	days Twice		
	12. Fazole bolus	Do		Daily	Do	
		Large animals	Two boluses	Do	Orally	Diarrhoea+ dysentery
			1 bolus/50 kg bwt.			

Chemotherapeutic Agents	Pharmaceutical Products (drugs) Specialities	Species	Dose Rate	Repetition	Route of Administration	Indications
44. Anti constipative drugs	1. Dulcolax tabs.	Do	Do	As required		Constipation
	2. Vaculax	Do	Do	Do	Do	Do
	Glaxenna tab.	Do	Do	Do	Do	Do
	3. Cremaffin suspension	Do	Do	Do	Do	Do
	4. Agarol suspension	Do	Do	Do	Do	Do
	5. Liquid paraffin.	Do	Do	Do	Do	Do
45. Supportive drugs	1. Tonophosphan	All species Large animals/small animals.	5- 20 ml. /1-3 ml.	Do	I/M	Debility + Exhaustion

Some Common Ointments, Liminents and Antiseptic lotions etc.

1. Iodine ointment (1:25)

Iodum	- 4 gm.
Pot. Iodide	- 4 gm.
Glycerine	- 12 gm.
Vaseline	- 80 gm.

Use-Painful swellings and sprains.

2. Tr. iodine

Iodine	- 5 gm.
Pot. iodide	- 3 gm.
Dist. water	- 5 ml.
Rectified sprit	- 200 ml.

Use- Cut or Injuries

5. Sulphur ointment

Sulphur sublimated	- 1 part
Vaseline	- 10 parts

Use in case of mange

6. Embee Veterinary Cream for wound dressing Or Dressing oil

Creosote	- 10 ml.
Oli. Turpentine	- 125 ml.
Oli. Lini	- 500 ml.

7. Carron Oil

Liquor calcis (Lime water)	- 1 part
Linseed oil	- 8 parts

To be mixed by shaking

3. Lugol's iodine

Iodum	- 2 gm.
Pot. Iodide	- 3 gm.
Dist. water	- 80 gm.

Use – Uterine paint

4. Red ointment

Hydragyri Iod. Rubr.	- 1 part
Vaseline	- 8 parts

Use - Blister

Use - Burns

8. B.I.P.P.

Bismuth subnitrate	- 1 gm.
Iodoform	- 2 gm.
Liquid paraffin	- q.s.

Use - Sinus and fistulae

9. Salicylic ointment

Acid salicylic	- 2 gm.
Acid carbolic	- 2 gm.
Vasiline	- 30 gm.

Use - Ringworm

10. In case of moist eczema, the following can be used

Acid salicylic	- 2 gm.
Acid tannic	- 2 gm.
Sprint	- 30 ml.

11. Liniment Ammonia

Liq. Ammonia fortis	- 30 ml.
Oil. Turpentine	- 30 ml.
Camphoor	- 30 ml.
Aqua	- 30 ml.
Simple oil	- 250 ml.

13. Betadine solution & Povidone – Iodine (U.S.P)

50% w/v. for dressing of wounds.

14. Himax ointment

Use – Skin or foot wounds

15. Mouthwashes

Alum	**- (1%)**
Borax	**- (2-3%)**
Boric acid	**- (2-3%)**
Copper sulphate	**- (0.5%)**
Potassium permagnate	**- (1:2000)**
Tannic acid	**- (0.5-2%)**
Sulphanilamide	**- (1%)**

16. Common antiseptics

To be mixed by shaking
Use - Sprain and chest pain

12. Antiseptic powder and solution

Boric acid	- 2 parts
Iodoform	- 1 part
Zinc oxide	- 1 part

Use - Dressing of unhealthy wounds and ulcerated surfaces.

17. Febrifuge

For cattle, buffaloes and horses.

Ammon, carbonate	- 8 gm.
Quinine sulphate	- 4 gm.
Sodium salicylate	- 16 gm.
Powder liquorice	- 16 gm.
Treacle	- Q.S.

One such electuary thrice daily.

18. Diuretics

i. Ridema inj. Large animals (C/B/H) 5-50 (I/M) ml. (I/M)
ii. Lasix inj. Dogs/Cats 0.25 - 0.5 ml.

Acriflavin	- (1:1000 to 1:10,000)
Boric Acid	- (1-2%)
Cetavlon	- (1:500 - 1:5000)
Iodine	- (2.5%)
Hydrogen peroxide	- (1:5 to 1:10)
Pot. permagnate	- (1:1000 to 1:5000)

20. Antibloat preparation
For cattle & buffaloes

R1 Oil turpentine (Tarpin)	- 60 ml.
Carbolic acid	- 4 gm.
Extract nuxvom	- 4 ml.
Linseed oil (Tisi tel)	- 1000 ml.

Give as haust.

21. Carminative mixture For cattle, buffaloes and horses

Ajwain powder	- 15 gm.
Anisi powder (sunf)	- 15 gm.
Black pepper (Golmirch)	- 0.5 gm.
Black salt (Kala mirch)	- 50 gm.
Ginger powder	- 15 gm.

Mix with water and drench thrice daily.

19. Stomachic

For horse and cattle.

Copper sulphate (Tutia)	- 2 gm.
Cobalt chloride	- 100 gm.
Ginger powder (Adrakh)	- 30 gm.
Nux vomica powder (Kochila)	- 30 gm.
Sodium bicarbonate (Khane ka soda)	- 20 gm.

Prepare electuary and give such thrice daily.

22. Ecbolic

For cows, buffaloes and mares

R1

1.	Ergot extract	- 20 ml.
	Magnesium sulphate	- 180 gm.
	Pulv ginger	- 30 gm.
	Pulv anisi	- 30 gm.
	Treacle	- Q.S.

One electuary daily.

ii. Replanta @ 50 gm. per day for 3-4 days in cows & Buffaloes.

23. Laxative/Purgative For cattle and buffaloes

Rx

Magnesium sulphate	- 150 gm.
Sodium sulphate	- 60 gm.
Sodium sulphate	- 60 gm.
Ginger powder	- 16 gm.
Water	- 750 ml.

Drench at once.

24. Expectorant

For cattle, buffaloes and horses

Anise powder	- 15 gm.
Ajwan powder	- 8 gm.
Camphoor powder	- 15 gm.
Extract belladona	- 3 gm.
Potassium chloride	- 12 gm.

26. Heart tonics

Horse and cattle

Tr. digitalis	- 8 ml.
Tr. Nux Vom	- 10 ml.
Tr. Zingiberis	- 30 ml.
Aqua add	- 125 ml.

Mft. Haust. Sig. once daily for eight days.

Digitalin - Horse and cattle - 15-60 mg S/C

Dog. - 1-10 mg.- S/C or I/M

Digitoxin Dog - 0.1 to 1 mg. orally or I/M

27. Astringent mixture for enteritis, diarrhoea and dysentery

Rx

(1)	Adult cattle	Calf.
Pulv. Creta	30 gm.	10 gm.
Pulv. Kaolin	30 gm.	10 gm.

Treacle - Q.S.
Prepare electuary and feed thrice daily

25. Tonics -

For cattle, buffaloes and horses
Rx

Cobalt sulphate	- 50 mg.
Copper sulphate	- 100 mg.
Ferrous sulphate	- 5 gm.
Gentian powder	- 6 gm.
Nuxvom powder	- 6 gm.
Treacle	- Q.S.

Such one dose thrice daily.

28. (2) Acid tannic 15 gm.

Kaolin	30 gm.
Pulv. ginger	15 gm.

Give in gruel once in 12 hours.

29. Preparation to treat impaction of colon with colic in horse

R/x

Aloes barb	15 gm
Aqua ferv (warm water)	Q.S.
Oi. Terebinth	60 ml.
Chloral Hydras	30 gm.
Oi. lini	500 ml.

Pulv. Catechu	30 gm.	10 gm.

2. Inj. Novalgin (Hoecht)

3. Inj. Polygesic (Hoecht)

Large animals	20-60 ml.
Calves/Foals	5-15 ml.
Sheep/Goat	2-8 ml.
Dog/Cat	1-2 ml I/M

4. Trigan - D

31. Feed supplements

A. For dogs

1. Vita pet
2. Calmin

B. For cattle & buffaloes

1. Milkmin
2. Gwala
3. Supplevite

Mft. Sig. Dissolve aloes and chloral hydras in hot water and shake well with oil of Turpentine and linseed oil. Administer as a drench slowly.

Preparation to treat spasmodic colic

Rx

30. 1. Chloral Hydras 30 gm.
Oil. Terebinth 30 ml.
Oil. Lini 500 ml.
Mft. Haust. Sig. Administer by stomach tube

4. Supplimin
5. Hiyield
6. Provimin Forte
7. Biotone fs. Powder
8. Kalmin forte

Mft. Elect. Sig. B.D.

32. Triple Carb (Gastric sedative for dogs)

Rx.
Mag. Carb. Lev.
Bismath Carb.
Sodi Bicarb aa 2.0 gm,
Mft. Pulv 6. Sig. Give 4 hourly

33. Mistura alba (Laxative mixture for dogs)

Rx.
Mag. Sulph 15-25 gm.
Mag. Carb levis 4 gms.
Aqua mentha pip ad 180 ml.
Mft. Mist. Sig. 2.4 teaspoonfuls according to weights

36. Diuretic for Dogs.

Rx.
Sod. Salicylas 0.5 gms.
Pot. Aceats 0.5 gms.
Spt. Amm-Arom- 1-2 ml.
Syrup 5 ml.
Aqua ad 15 ml.
Mft. Haust Sig T.D.

37. CNS depressants for cattle or horse

Rx.
Chloral hydras 20 gms.
Aqua 125 ml.
Oil Lini 500 ml.

34. Antiemetic (for dog)

Rx.

Tr. opii.	8 ml.
Spt. Chloroform	30 ml.
Aqua. Mentha pip ad	180 ml.

Mft. Mist. Sig. 2-4 teaspoonfuls 3 hourly

35. Diuretic (for horse and cattle)

Rx.

Phenacetin	4 gms.
Treacle	Q.S.

Mft. Haust Sig. Stat

38. Zinc iodoform paste (Z. I. P. P)

Rx.
Zinc Oxide 1 Part
Iodoform 2 Parts
Liquid Paraffin q. s. to make a paste
Use As an antiseptic, germicide and stimulant to granulation tissue.

TABLE 38. IMMUNIZATION PROGRAMME IN ANIMALS

Disease	Vaccines	Animals to be Vaccinated	Dose & Route of Administration	Immunity	Remarks
1. Anthrax	Anthrax spore vaccine.	Cattle/buffaloes/sheep/goats/ camels/elephants.	1 ml S/C (In elephant second dose of 3 ml after an interval of 3 months).	1 year	Vaccination (annually).
			5-10 ml S/C		
2. Black Quarter	Black quarter vaccine	Cattle and buffaloes	1-2 ml S/C	1 year	Do before
	(a) Monovalent	sheep and goats	Do	1 year	monsoon.
	(b) Polyvalent	Do		1 year	
					-
	Brucella vaccine		2-5 year		
3. Brucellosis	(Strain- 19)	Cattle 6 to 9 months and the adult except pregnant animals.		2-5 year	Do
		Lambs & sheep.			
4. Enterotoxaemia (P. K. D. and Enteroxaemia in sheep).	Enterotoxaemia vaccine.	(above 3 months)	2-2.5 ml S/C Two injection at an interval of 2-3 weeks.	1 year	Do
			5-10 ml S/C		
5. Haemorrhagic septicaemia (H.S.).	(a) H.S. borth vaccine	Cattle and buffaloes.		3-6 months	Vaccination before
	(b) H.S. agar wash vaccine	Do	Do		monsoon (6
			3 to 5 ml I/M	Do	months).
	(c) H.S. Oil adjuvant vaccine.	Do		1 year	Do

Disease	Vaccines	Animals to be Vaccinated	Dose & Route of Administration	Immunity	Remarks
6. Rinderpest	(a) Freeze dried goat tissue vaccine (FDGTV).	Do	1ml S/C	Life long immunity (above 14 years).	Annually
	(b) TCRP (Tissue culture vaccine)	Do	1ml S/C		
7. Foot and Mouth Disease	Hoechst FMD vaccine Quadrivalent	Do	10 ml S/C for cattle buffaloes and calves above 4 months.	2 years immunity.	
	I.V.R.I. Banglore, B.A.I.F. (Vaccine (All the four straines of the F.M.D. virus in to the vaccine) Types O,A,C & Asia 1.	Do			Revaccination after every six months in animals (e.g., exotic & cross bred animals).

VACCINATION PROGRAMME IN DOGS					
SL. No	Name of Vaccines	Age 1st Vaccination	Route of Administration	Booster	Repeat	Diseases
1.	Rabisin	6 to 8 weeks	I/M	14 weeks	1 to 2 years 3 years	Rabies. Rabies.
2.	ARV (20%)	3 months	I/M			
3.	Candur DHL	2 months	I/M or S/C	9 months 12-14 weeks	3 years 1 year	Distemper, hepatitis and leptospirosis.
4.	Caniffa	2 months	I/M	12-14 weeks	1 to 2 years	Distemper
5.	Pentadog	6-8 months	I/M	12-14 weeks 14 weeks		Distemper, hepatitis, leptospirosis parvo virus infection . etc.
6.	Candur-P	2 months	I/M	12-14 weeks	1 year	Distemper infectious
7.	Parvodog	2 months	I/M or S/C	12-14 weeks	1 year	hepatitis, parvo virus
8.	Commander-7 Annumune	6 weeks	I/M	Annually	1 year	infection and leptospirosis.

ARV	Anti rabies vaccine Disease Age for vaccination.
DHL	Distemper, hepatitis and leptospirosis Distemper 12 weeks.
Diseases	Age for vaccination
Distemper	12 weeks
Rabies	3 months of age
DH	Distemper and hepatitis Rabies 3 month of age.

Precautions

1. Animals in advanced pregnancy are not to be vaccinated.
2. Only healthy animals with normal morning and evening temperature are chosen for vaccination.
3. Bacterial and viral vaccines can be repeated after 2 and 3 weeks respectively.
4. During an outbreak of a disease in a particular area, clinically healthy animals are required to be vaccinated.
5. Reconstituted vaccine should be used within two hours of its preparation.
6. Vaccination against H.S., B.Q. & Anthrax may be avoided during an outbreak of such diseases owing to suspectibility of the animals.
7. Bacterial vaccinations are preferred to be given before onset of rainy season.
8. Vaccination against viral diseases e.g., rinderpest is usually given during winter months.
9. The animals should be dewormed at 4 to 6 month intervals and underdose anthelmintic therapy should be avoided.
10. Postbite vaccination in the cases of dog bites is preferred.

Table 39. PATENT PREPARATIONS USED IN ANIMALS

ANTIBIOTICS

Name	Presentation	Dose	Manufacturer
AMIKACIN			
Inj Akacin	Inj. 2ml amp (100mg)	10 mg/kg TID	**Morvel**
Inj Vetacin	Inj. 2ml, 10ml, 30ml vial (250mg/ml)		**IBC**
FORTIFIED PROCAINE PENICILLIN INJ			
Fortified Procaine Penicillin inj 20 lac	Inj. 20 lac vial (Procaine penicillin G 15 lac, penicillin G Sodium 5 Lac)/vial	Cattle, horse, sheep, goat, pig: 4000-10,000 IU/Kg BW Dog: 10,000-20,000IU/Kg BW	Alembic
Fortified Procaine Penicillin inj 40 lac	Inj. 40 lac vial (Procaine penicillin G 30 lac, penicillin G Sodium 10 Lac)/vial		Alembic
Fortified Procaine Penicillin 20 lac	Inj. 20 lac vial (Procaine penicillin 15 lac, Benzyl Penicillin Sodium 5 Lac)/vial		IBC
Fortified Procaine Penicillin 40 lac	Inj. 40 lac vial (Procaine penicillin 30 lac, Penicillin G Sodium 10 Lac)/vial		IBC
Fortified Procaine Penicillin 20 lac	Inj. 20 lac vial (Procaine penicillin 15 lac, Benzyl Penicillin Sodium 5 Lac)/vial		Sarabhai Zydus
Fortified Procaine Penicillin 40 lac	Inj. 40 lac vial (Procaine penicillin 30 lac, Penicillin G Sodium 10 Lac)/vial		Sarabhai Zydus
AMPICILLIN			
Ampilin Vet	Inj. 2gm & 2.5 gm vial	Cattle, Sheep, Goat, horse, Pig : 5-10mg/kg BWQID Dog, Cat: 20mg/kg BW QID orally; 10mg/kg BW QID parentrally	Lyka
Bacipen	Inj. 2& 2.5 gm vial		Alembic
Catcillin inj. vet	Inj. 2.5gm vial		Cattle remedies
Conampi	Inj. 1gm, 2gm & 2.5gm vials		Concept

Name	Presentation	Dose	Manufacturer
Dynacil vet	Inj. 1gm & 2.5gm vial		HAL
Roscillin	Inj. 250, 500 mg& 1gm vials Tab 125mg & 250mg Syrup 125mg/5ml & 250mg/5ml		Ranbaxy
Stancillin	Inj. 2gm vial		Sarabhai zydus
Vetampin	Inj. 2gm vial		Wockhardt
AMPICILLIN AND CLOXACILLIN			
AC -VET	Inj. 2gm vial (Ampicillin sodium 1gm & Cloxacillin sodium 1 gm)		Intas
AC -VET forte	Inj. 3gm vial (Ampicillin sodium 1.5gm & Cloxacillin sodium 1.5 gm)		Intas
Ampicillin and Cloxacillin	Inj. 2gm vial		Wockhardt
Baxivet inj	Inj. 500mg, 1 gm & 2 gm vials	4-10 mg / kg BID	Lyka
Binocin	Inj. 1 gm & 2 gm vials		Concept
Biolox	Inj. 2 gm vial		Jeps
Biolox forte	Inj. 2.5 gm vial		Jeps
Catlox inj. vet	Inj. 2gm vial		Cattle remedies
RC forte	Inj. 4gm vial		Ranbaxy
Vetclox forte	Inj. 2gm vial		Sarabhai zydus

Name	Presentation	Dose	Manufacturer
AMOXYCILIN			
Amoxyrum bolus	1.5 gm bolus(2's)	10 mg/kg BW BID (LA); 10-20 mg/kg BW (SA)	Glaxo
Comoxyl powder500mg/5g	100gm; 500gm	Cattle, horse : 20g for 3 days; sheep, goat, calf: 5gm for 3-5days	Concept
Hipen	Tab: 125 mg & 250 mg; inj. 250mg and 500 mg vial	10 mg/kg BW BID (LA); 10-20 mg/kg BW (SA)	Cadila Health Care
AMOXYCILIN + CLAVULANATE POTASSIUM			
Augmentin Dry syrup	30ml (Amoxycilin 200mg + Clavulanic acid28.75mg)/5ml	Dog & cat: 12.5-25 mg/Kg BW BID	Smith Kline Beecham
Augmentin 1000DUO	Tab 1000mg (Amoxycilin 875mg + Clavulanic acid125mg)	-do-	Smith Kline Beecham
Temobax	Dry susp 6.6gm (30 ml)	-do-	Ranbaxy
AMOXYCILIN + CLOXACILLIN			
Amclox	Inj. 2g (Amoxycillin sodium 1g and cloxacillin sodium1gm)/vial		Novartis
Centamox	Inj. 2g (Amoxycillin sodium 1g and cloxacillin sodium1gm)/vial inj. 500mg (Amoxycillin sodium 250mg and cloxacillin sodium 250mg)/vial		Century
Comaxvet	Inj. 500mg & 2gm vial		Vetindia
Conmox	Inj. 2gm vial		Concept

Name	Presentation	Dose	Manufacturer
Crilmox inj. vet	Inj. 2gm vial		Cattle Remedies
Hipenox	Inj. 1gm & 2 gm vial		Sarabhai zydus
Inimox	Inj. 500mg, 2gm & 4 gm vial		Indian Immunologicals
Intamox	Inj. 500mg, 2gm, 2.5gm & 4 gm vial		Intas
Klomivet	Inj. 2gm vial		Lyka
Megamycin	Inj. 2gm vial		Pfizer
Megamycin forte	Inj. 3gm vial		Pfizer
Moxel	Inj. 500mg, 2gm & 3 gm vial		Alembic
Moxyclox	Inj. 3gm vial		Wockhardt
CEPHALOSPORINS			
CEPHALEXIN			
Sporidex	Drops: 100 mg/ml, 10 ml Granules: 125 mg/5ml, 30 ml Granules: 250 mg/5ml, 30 ml DT-tab; 125 & 250 mg, 10's Cap: 250& 500 mg, 10's	Small animals 10-25 mg/kg BW/ 8-12 h PO	Ranbaxy

Name	Presentation	Dose	Manufacturer
Alcephin	Tab: 125 mg, 10's Syrup: 125mg/5ml, 40 ml	-do-	Alembic
Phexin	Drops: 100 mg/ml, 10 ml Syrup: 250 mg/5ml, 30 ml DT-tab; 125 mg, 10's Cap: 250& 500 mg, 10's	-do-	Glaxo pharma
Sepexin	Kid-Tab 125mg, 10's Disp-Tab 250mg, 10's Syrup: 125 mg/5ml, 30 ml		Lyka
CEFTRIOXONE			
Cefaxone	Inj. 250mg, 500mg, 1 g vial	15-50 mg/kg BW/day	Lupin
Monotax	Inj. 125mg , 250mg, 500mg, 1 g vial	-do-	Biochem
Zefone	Inj. 250mg, 1 g vial	-do-	Cadila healthcare
CEFOTAXIME			
Biotax	Inj. 125mg, 250mg, 500mg, 1 g vial	Dog: 10-50 mg/kg BW/ 6-8h Goat: 50 mg/kg BW/ 12 h	Biochem
Taxim	Inj. 125mg, 250mg, 500mg, 1 g vial		Alkem
Britax	Inj. 250mg, 500mg, 1 g vials		Brihans
Lyfovet	Inj. 2gm vial		Oriental

Name	Presentation	Dose	Manufacturer
CEFADROXIL			
Cefadrox	Kid-Tab 125mg, 10's Syrup: 125 mg/5ml, 30 ml	Dog, Cat: 10-30mg/kg BW TID	Aristo
Droxibid	Kid-Tab 250mg, 4's Tab 500mg, 4's		HAL
Droxyl	Kid-Tab 250mg, 4's Tab 500mg, 4's Susp 250mg/5ml, 30ml		Torrent
Lydroxil	Dis-Tab 250mg, 4's Syrup 125mg/5ml, 40ml		Lyka
STREPTOMYCIN			
Ambistryn-S	Inj. 1 gm vial	All animals : 10 mg / kg BW	Sarabhai
KANAMYCIN			
Kancin inj	Inj. 500 mg & 1g vial	Cattle: 5-10 mg / Kg BW IM, IV	Alembic
Kanamycin	Inj. 500 mg & 1g vial	Dog, Cat: 5 mg / Kg BW IM, IV	Biochem
Indomycin	Inj. 2gm vial		IBC
GENTAMICIN (40 mg in each ml)			
Catlogenta Inj Vet	Inj. 10ml & 30ml vials	Cattle, Sheep, Goat, Pig, Dog, Cat: 4mg/kg BW BID Poultry: 3-5mg/kg BW	Cattle remedies

Name	Presentation	Dose	Manufacturer
G-Cin	Inj. 30ml & 90ml vials		Indian Immunologicals
Gentamicin	Inj. 30ml & 100ml vials		Alembic, IBC, TTK, Vetcare, Wockhardt, Ranbaxy
Gentamicin	Inj. 10ml & 30ml vials		Vetindia
Marcogenta	Inj. 2ml, 5ml , 10 ml vial& 30ml vials		Marc
Gentim inj.	Inj. 10 & 30 ml vial		Merind
ERYTHROMYCIN			
Althrocin Tab	Tab 250 mg, 500 mg Liq 25mg/ml	Cattle, sheep, goat, horse: 2.2-4.4 mg/kg BW Pig: 2.2-6mg/kg BW Dog 10-40 Mg/kg BW	Alembic
Anithrocin FS	Powder 20mg/g		IBC
Anithrocin SP	Powder 50mg/g		IBC
CHLORAMPHENICOL			
Chloramphenicol sodium Succinate	Inj. 1 gm vial	LA- 2-4 mg /kg I/V 20-30 mg I/M	KAPL
Chloramphenicol sodium Succinate	Inj. 1gm & 2gm vials		Lyka
Chlorophen	Inj. 30 ml vial (100 mg/ml)		Vetindia
Chlorovet	30 ml vial (100 mg/ml)	LA-4 mg/kg SA-5-15 mg/kg	G. Loucatos
Neochlor	Inj. 10 & 30 ml vials (each ml contains 100mg)	4-11 mg/ kg I/m	Vet care

Name	Presentation	Dose	Manufacturer
Neochlor forte powder 20% w/w	50gm pack	1gm/Lt of water on day 1; 1gm/10 Lt. of water for next 3-5 days.	Vet care
Lykacetin	Inj. 1gm, 2gm, 3gm vials		Lyka
Vetnicol bolus	Bolus 500 mg (4's)		Vets farma
CIPROFLOXACIN			
C-Flox	Inj. 40mg/ml (50ml vial)	Dog, Cat: 5-15mg/kg BW Cattle, sheep 4-5 mg/kg BW	Intas
Ciprovet	Inj. 10ml, 30ml vial		Vet India
Ciplox	Tab 250mg (10's) INF IV 20mg/10ml (100ml)		Cipla
Ciprobid	Tab 250mg (10's) Inj. IV 2mg/ml (50ml & 100 ml)		Zydus Cadila
Ciprowin	Tab 250 mg & 500 mg (10's)		Alembic
CIPROFLOXACIN COMBINATIONS			
AV-Floxin 2000	Powder (Ciprofloxacin 20g, Norfloxacin 20 g)/kg: 500 gm pack	Broilers: 500mg/M ton of feed Layers, breeders: 1000mg/M ton of feed	AVR
C-Flox-TZ	Tab (Ciprofloxacin HCl 250mg, Tinidazole 300mg): 10's Bolus: (Ciprofloxacin HCl 1500mg, Tinidazole 1800mg): 2's	Large animal: 1-2 boli OD for 3-5 days Small animals: 1tab/15-25 Kg BW	Intas
ENROFLOXACIN			
Conflox-vet inj 5%	Inj. 30ml (50mg/ml)		Concept
Conflox Vet Oral Solution 10% Soln. (100mg/ml)	Soln 100ml & 500ml	Poultry 10mg/kg BW	Concept
Enrocare	Inj. 20ml vial (100mg/ml)		Vetcare

Name	Presentation	Dose	Manufacturer
Enrocin	Inj. 15ml, 50ml, 100ml vials		Ranbaxy
Enrodac-10	Inj. 15ml vial		Sarabhai zydus
Enrofloxacin	Inj. 15ml vial		Vetindia
Enrox (10% w/v)	Inj. 15ml & 100ml vial		Alembic
Floxidin	Oral Soln. (5% w/v) 100ml & 250ml Oral Soln. (10% w/v) 100ml & 250ml Inj. (10% w/v) 15ml & 50ml vial	Poultry 10mg/kg BW 5% soln. 2ml/Lt. of water 10% soln. 1ml/Lt. of water	Intervet
Meriquin	Tab 50mg (10's) Inj. (100mg/ml): 1ml & 50ml vial Liq (100mg/ml): 100ml & 500ml	Poultry 1ml/10kg BW	Wockhardt
Quin Intas	Inj. (100mg/ml): 15ml, 30ml & 15ml vial Liq (100mg/ml): 100ml & 1Lt	Poultry Prophylactic: 1ml/4-8 Lt. of water Curative: 1ml/2-4 Lt. of water	Intas
Roflox	Inj. (10%): 15ml & 100ml		Novartis
OXYTETRACYCLINES			
Alcyclin-O	Inj. 30ml vial (50mg/ml)	Cattle, sheep, goat, swine, horse: 5-10mg/kg BW/day Dog & Cat: 10-20mg/kg BW/day	Alembic
Intamycin	Inj. 30ml & 100ml vial (50mg/ml)		Intas
Intamycin LA	Inj. 30ml & 100ml vial (200mg/ml)		Intas
Oxy-100	Inj. 30ml & 100ml vial (100mg/ml)		Vetindia
Oxytetracycline	Inj. 30ml, 50ml & 100ml (50mg/ml)		Alembic, IBC
Oxytetracycline	Inj. 30ml & 100ml vial (50mg/ml)		Concept, Indian Immunologicals

Name	Presentation	Dose	Manufacturer
Oxytetracycline	Inj. 30ml, 50ml & 100ml (50mg/ml) Bolus 500mg		Vetindia
Oxytetra-LA	Inj. 30ml (200mg/ml)		Vetindia
Oxyvet	Inj. 30ml, 50ml & 100ml (50mg/ml)		Sarabhai zydus
Oxyvet-LA	Inj. 30ml (200mg/ml)		Sarabhai zydus
Terramycin	Inj. 30ml & 100ml vial (50mg/ml) Tab 500mg (4's) Soln 60 ml (50mg/ml)		Pfizer
Terramycin-LA	Inj. 30ml & 50ml vial (200mg/ml)		Pfizer
Wolicycline	Inj. 30ml (50mg/ml)		Wockhardt
Wolicycline DS	Inj. 30ml (100mg/ml)		Wockhardt
Wolicycline LA	Inj. 30ml (200mg/ml)		Wockhardt
TETRACYCLINE			
Neocyclin bolus	500mg (4's)		Intas
LINCOMYCIN (300 mg/ml)			
Alincomycin - vet	Inj. 5ml vials	Bovine:10mg/kg BW	Alved
Lincocin inj.	Inj. 2ml vials	Dog : 15-25mg/kg BW BID Pig: 11mg/kg BW BID	Max
STREPTOMYCIN + PENICILLIN			
Dicrysticin inj.	Inj. 2.5 gm vial(Procaine penicillin G 15Lac, Penicillin G sodium 5 Lac, streptomycin sulphate 2.5 gm)/vial	Large Animal: 2ml/50kg BW Small Animal; 1ml/5kg BW	Sarabhai Zydus

Name	Presentation	Dose	Manufacturer
Munomycin forte inj.	Inj. 2.5gm vial	Large animal: 1-2 vials daily	Glaxo Smithkline
Vetopen inj.	Inj. 0.5,1.0, 2.5 gm vial		HAL
Bistrepen-V	Inj. 2.5gm vial		Alembic
SULPHONAMIDES			
Sulphadimidine			
Aldine	Bolus 5gm	All animals: 100mg/kg BW oral, IV	Alembic
Diadin	Bolus 5gm		Pfizer
Pabadine bolus	Bolus 5gm (4's)		Intas
Sulfam	Inj. 100ml vial (1gm/3ml)		Pranav
Sulfamin	Bolus 5gm(4's) Inj. 100ml & 450ml (333mg/ml)		Indian Immunologicals
Sulpha	Bolus 5gm		Sarabhai zydus
SULPHONAMIDES and TRIMETHOPRIM COMBINATION			
Atrima bolus	Bolus 1.2gm (Sulphadiazine 1gm, trimethoprim 0.2gm) Bolus 2.4gm (Sulphadiazine 2gm, trimethoprim 0.4gm)	Large animals: 15-30mg/kg BW Dog : 15mg/kg BW	Prima Vet Care

Name	Presentation	Dose	Manufacturer
Bactridox	Inj. 30 ml vial (sulphadoxine 20mg, trimethoprim4mg)/ml	-do-	Alved
Bactrisol bolus	Bolus 1.2gm(Sulphadiazine 1gm, trimethoprim 0.2gm)	-do-	Alved
Bactrisol Inj	Inj 30 ml vial (Sulphadiazine 400 mg, trimethoprim 80mg)ml		Alved
Biotrim IM	Inj. 10 & 30 ml vial(Sulphadiazine 400 mg, trimethoprim 80mg)ml		Ranbaxy
Biotrim IV	Inj. 30 ml vial(Sulphadiazine 200 mg, trimethoprim 40mg)ml		Ranbaxy
Biotrim DS	Bolus 2.4 gm(Sulphadiazine 2gm, trimethoprim 0.4gm)		Ranbaxy
Cotrimal bolus	Bolus (sulphamethoxazole 1.25gm, trimethoprim 0.25gm)		Alembic
Cotrimal DS bolus	Bolus (sulphamethoxazole 2.5 gm, trimethoprim 0.5gm)		Alembic
Intrim	Bolus (sulphamethoxazole 2 gm, trimethoprim 400 mg)		Indian Immunologicals
Intrim bolus	Bolus (sulphamethoxazole 1.25 gm, trimethoprim 250 mg)		Intas

Name	Presentation	Dose	Manufacturer
Intrim Forte Bolus	Bolus (sulphamethoxazole 2 gm, trimethoprim 400 mg)		Intas
Oriprim Bolus	Bolus (sulphamethoxazole 2 gm, trimethoprim 400 mg)		Sarabhai zydus
Oriprim IM	Inj. 5ml amp		Sarabhai zydus
Oriprim V	Inj. 30ml vial		Sarabhai zydus
Sulcoprim	Bolus (Sulphadiazine 2gm, trimethoprim 200mg)		Concept
Sulprim -24	Inj. 30ml vial (Sulphadiazine 200mg, trimethoprim 40mg)/ml		Unichem
Oriprim powder	Powder 100gm (Sulphamethoxazole 500mg, trimethoprim 100mg)/g	Animals: 2g/40kg BW BID Poultry: Upto 6 weeks: 2gm/100birds 6-12 weeks : 4gm/100birds 12-18 weeks: 8gm/100 birds	Sarabhai zydus
Biotrim oral Liq.	Liq. 100ml (Sulphadiazine 200 mg, trimethoprim 40mg)ml	Poultry: 1ml in 2-4 Lt. Of drinking water	Ranbaxy
Bactrisol Dispersible powder	Powder 100gm, 500gm & 1kg	Chicks: 1gm/ 2Lt of water Adults: 1gm/ Lt of water	Alved

Name	Presentation	Dose	Manufacturer
NORFLOXACIN			
Anquin	Tab 400mg (4's)	Dog, Cat: 22mg/kg BW BID	Lyka
Negaflox	Tab 400ng (8's)		Cadila Health Care
Norflox	Distab 100mg, 200mg, 400mg (10's)		Cipla
Uroflox	Tab 400mg (10's)		Torrent
METRONIDAZOLE			
Anarobin	Inj. 50ml vial (5mg/ml)	Dog: 25-50mg/kg BW/day in divided doses, Oral 20mg/kg BW/day in divided doses, IV Cattle: 20mg/kg BW/day in divided doses, IV	Unichem
Flagyl	Tab 200mg, 400mg (10's)		Rhone-Poulenc
Flagyl I.V	Inj. 100ml (100mg/ml)		Rhone-Poulenc
Metrogyl	Tab 200mg, 400mg (10's) Susp 30ml, 60ml (200mg/5ml) Inj. I.V 100ml (500mg)		Unique
Metronidazole	Inj. 100ml (5mg/ml)		Prima Vetcare
Unimezol	Tab 200mg (10's)		Unichem
METRONIDAZOLE COMBINATIONS			
Amedol-F	Susp. 60ml; (Metronidazole 100mg, Diloxanide furoate 125mg, Furazolidone 50mg)/5ml	Small animals 3-5 ml	Vetchem

Name	Presentation	Dose	Manufacturer
Centrogyl LM	Bolus (Metronidazole 1gm, Furazolidone 500mg, Loperamide hydrochloride 75mg)	Large animals 4-6 boli / day Small animals 1-2 boli / day	Century
Dependal M	Tab (Metronidazole 300mg, Furazolidone 100mg)	Dog, Cat : 1 tab TID	Smithkline Beecham
Diamet	Bolus (Metronidazole 1g, Furazolidone 200mg)	Animals: 1 bolus/50kg BW BID	Novartis
Dirolin	Bolus (Metronidazole 1g, Furazolidone 200mg)	Animals: 1 bolus/50kg BW BID	Vetindia
Fazole	Bolus (Metronidazole 1g, Furazolidone 200mg)	Animals: 1 bolus/50kg BW BID	Unichem
Metrofural	Bolus (Metronidazole 1gm, Furazolidone 500mg, Loperamide hydrochloride 75mg)	Large animals 4-6 boli / day Small animals 1-2 boli / day	Alembic
PEFLOXACIN			
Pelwin inj. **(Infusion)**	100 ml bottle	100 m.l/ 200 kg	Wockhardt

Antidiarrhoeals

Name of Drug	Presentation	Dosage	Firm
Diphenoxylate			
Lomotil	Tab 2.5mg (10's0	Dogs: 2-5mg (total dose) Cats: 0.5mg/kg BW	RPG
Loperamide			
Lopamide	Tab 2mg (10's)	Dogs, cats: 100mg/Kg BW	Torrent
Indigenous Preparations			
Becknor Bolus	Bolus	Large Animals: 1bolus BID PO Small animals: ½ bolus BID	Natural remedies
Catorrhoea	Powder 100gm, 1 kg Tab 8X 6's	Cattle buffalo: 25-50 g BID/ 3-4 Tabs TID Horse, mule: 20-30 g BID/ 2-3 Tabs TID Sheep, Goat: 10-20 g BID/ 1-2 Tabs TID	Cattle remedies
Diamukt	Dry susp. 15 gm (add water upto the mark on label)	Dogs oral: (upto 10kg): 5ml TID (more than 10 kg): 10-15 ml TID	Dabur
Diarex Vet	Bolus	Large animals: 2 boli BID PO Small animals: ½ -1 bolus BID PO	Himalaya
Diaroak	Dry susp. 30gm, 400gm, 1kg, 2.5 kg	Large animals: 30gm Small animals 10-15gm Poultry (mixing rate): 2.5 kg/ton of feed	Dabur
Neblon Powder	Powder 100 gm & 1 kg pack	Large animals:30-50gmBID/QID Small animals: 6-10gm BID/QID Dogs, piglets: 2-3 gm BID/QID	Indian herbs

HEPATOBILIARY DRUGS			
Name of Drug	**Presentation**	**Dosage**	**Firm**
Beekom-L	Inj. 30 ml vial	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Wockhardt
Beekom-L	Inj. 30 ml vial	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Wockhardt
Belamyl Inj.	Inj. 10 ml, 30ml & 50ml vials	Large Animals 5-10 ml , IM Small Animals 0.25-0.5ml, IM	Srabhai Zydus
Bivinal Forte	Inj. 10 ml, 30ml vials	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Alembic
Bovoplex-CC	Inj. 10 ml, 30ml vials	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Indian Immunologicals
Brotone	Liq 120ml & 500ml	Cattle: 40ml OD for 3 days Dog 5ml BID Poultry: Broliers: 5-10ml/100 birds Growers & layers: 20ml/100 birds	Glaxo
Hepaplex	Inj. 10 ml, 30ml vials	Large Animals 2.5-5 ml, IM Small Animals 0.5-1 ml , IM	Unichem
Livamyl Inj. Vet	Inj. 10 ml, 30ml vials	Large Animals 4ml , IM Small Animals 0.25-0.5ml, IM	Cattle Remedies

Name of Drug	Presentation	Dosage	Firm
Livaplex	Inj. 30ml Vial	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Pranav
Liverjet	Inj. 10 ml, 30ml vials	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Cadila Pharma
Livogen	Inj. 10 ml, 30ml vials	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Glaxo
Nutriliv injection	Inj. 10 ml, 30ml vials	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Vetcare
Nutriliv forte	Liq 500ml, 5Lt., 30 Lt.	Broilers: 5-10ml/100 birds PO Layers: 20ml/100 birds	Vetcare
Pepsid	Inj. 10 ml, 30ml vials	Large Animals 3-5 ml, IM Small Animals 0.5-1 ml , IM	Concept
Stronic	Inj. 10 ml, 30ml vials	Large Animals 5-10 ml, IM Small Animals 0.5-2 ml , IM	Ranbaxy
Enliv	Powder 1kg, 5kg pack Liq.: 1Lt, 5 Lt	Chicks: 3ml/1000 chicks/day Growers: 7ml/12000 birds/day Layers/brolers: 10ml/1000 birds/day	Alembic
Liv 52 vet	Powder 10g, 100g, 1kg, 5kg Liq.: 11oml, 1Lt., 5Lt		Himalaya
Liver Up	Powder 1kg	Large animals: 5gm BID PO Small animals: 2gm BID PO	Sarabhai Zydus

Name of Drug	Presentation	Dosage	Firm
Livol	Powder 100g & 1kg	Cow, Buffalo: 40-60gm OD/BID PO Calf, heifer, colt, pig: 15-20g OD/BID PO Sheep, Goat: 8-12 gm OD/BID PO Dog, Piglet: 3-5 gm OD/BID PO	Indian Herbs
Yakrifit	Bolus Liq. 125ml, 250ml & 500ml	Cow, buffalo, horse: 50ml or 2 bolus BID Sheep, Goat: 15-20 mlor ½ bolus BID	Dabur

Anthelmintics in Veterinary Practice

A) Benzimidazole (BZD) Anthelmintics:

Dosage

Name of the Anthelmintic	Species				
	Cattle (mg/kg)	Sheep (mg/kg)	Horse (mg/kg)	Pigs (mg/kg)	Dogs (mg/kg)
Albendazole	7.5	5	5	-	-
Fenbendazole	7.5	5	5-10	5	100
Mebendazole	-	10-15	5-10	30 ppm for 10 days	25-50 twice daily for 5 days
Thiabendazole	100	50-75	50	50-100	
Triclabendazole	12	10	-	-	

Route of Administration: Per Os

B) Imidazothiazole

Dosage:

Name of the Anthelmintic	Species				
	Cattle (mg/kg)	Sheep (mg/kg)	Pigs (mg/kg)	Dogs (mg/kg)	Poultry (mg/kg)
Levamisole	7.5	7.5	7.5	5	25-50

Route of administration: Per Os or Subcutaneous

C) Salicylanilides:

Dosage:

Name of the Anthelmintic	Species		
	Cattle (mg/kg)	Sheep (mg/kg)	Dogs & Cats (mg/kg)
Closantel	5 SC, 10 PO	5 SC, 10 PO	5 PO
Niclosamide	-	-	150 PO
Oxyclozanide	10-15 PO	10-15 PO	-
Rafoxanide	7.5 PO	7.5 SC; 3 SC	-

SC: Subcutaneous; PO: Per Os

D) Piperazine Derivatives:

Dosage:

Species Anthelmintic	Horses (mg/kg)	Pigs (mg/kg)	Dogs & cats (mg/kg)
Diethyl carbamazine	-	-	6.6
Piperazine	200	250-300	110-220

Route of administration: Per Os

E) Tetrahydropyrimidines:

Dosage:

Morantel: Cattle & sheep-10mg/kg, orally

Pyrantel: Horse - 20 mg/kg (6.6 mg/kg base) orally; Dogs - 15mg/kg (5 mg/kg base), orally.

F) Macrocyclic Lactones (Macrolide Endectocides):

Dosage:

Ivermectin: Cattle, sheep & horses: 0.2 mg/kg SC; Pigs: 0.3 mg/kg SC; Dogs: 0.006 Mg/kg (monthly, for heart-worm)

Milbemycin D: Dogs: 1mg/kg SC once monthly

Milbemycin oxime: Dogs: 0.5 mg/kg SC once monthly

Postmortem Examination

It consists of systematic anatomical examination (i.e., first with the naked-eye and, then, with the aid of a microscope) Bacteriological and chemical examinations etc., are carried out to find out the definite diagnosis as the cause of death. It gives an insight into the nature, extent and evolution of the disease in an organism Obduction, cosmetic postmortem complete and incomplete postmortems are some of the kinds of postmortem examinations which are carried out according to the object or purpose of the investigation. For example, an examination of only brain may serve the purpose as done usually in the cases of rabies in animals (canines). Pre autopsy data (e.g., age, sex and symptoms etc enables the autopsists to adopt a more logical approach in discussing the priorities or severities of different pathoanatomical diagnoses (i.e., placement of different pathoanatomical diagnoses in order of severities of the lesions) and the art of writing the epicrises guides the autopsist to the definite cause of death after looking into details of merits of several primary secondary and many other co-existing lesions in the dead animals. Postmortem protocol should be thoroughly prepared without missing to note any lesions and pathoanatomical diagnoses are made for different systems of the body in view of the lesions therein. The most important or severe pathoanatomical diagnosis is given the first priority in declaring the cause of death. Deaths in animals may be either due to biological or aetiological factor. A rupture of heart liver and thrombosis in cerebral artery may be responsible for biological death or somatic death of the

animal but an aetiological diagnosis refers to a definite aetiological factor for bringing about an end to life of an animal. For example, *Mycobacterium tuberculosis, Bacillus anthracis and Clostridium chauvoei* are considered as aetiological causes of death. The fact that an aetiological factor ultimately produces fatal biological or somatic changes in the body refers to another aspect of discussion.

Obduction refers to a medicolegal postmortem examination. The findings are used in the court for deciding the cause of death or accident or duration of some pathologic condition.

Cosmetic postmortem is a postmortem which is done with least disfigurement to the cadaver and the parts are replaced in the body . The cuttings are sewed together and the body Is washed and made to appear as nearly intact as possible.

Complete postmortem refers to an autopsy in which organ or part is carefully examined to make possible a correct diagnosis.

Incomplete postmortem refers to an autopsy in which a part of body (brain or limb etc.) is removed and examined for diagnosis of the disease. This is a sort of postmortem done to corroborate a postitive laboratory diagnosis e.g., rabies in animals.

Authority should be in a written form to do postmortem examination from the police officer , or the farm management or the owner of the dead animal to avoid unnecessary complications.

Clinical history (anamnesis) must be procured before autopsy of the dead body. Details of clinical diagnosis, treatment or other concerning facts are very much needed to arrive at a correct diagnosis postmortem.

Destruction

The dead animals after autopsy must be either burnt or suitably buried between layers of lime sufficiently deep (about 6 feet in depth) in the ditches dug for such purposes.

Care of the Instruments

These must be sharp and properly sterilized to protect the autopsists during postmortem examination. The autopsists must be properly dressed i.e., there should be utilization of aprons, white coats, gumboots during autopsy.

Recording the Lesions

Postmortem findings must be recorded immediately after autopsy in the postmortem hall by another person. There is usually a need of three persons (including the autopsist) for postmortem work.

A uniform and careful method should be followed for opening the dead body and examining the organs of the cadaver. To maintain anatomical continuity of the organs of a system during postmortem examination is very helpful to make conclusions about the cause of death. This helps in an easy detection of an obstruction (e.g., stone or calculus) in the tubal structures (say, bile duct, urethera and parts of the gut etc.).

Postmortem protocol refers to a detailed written description of the postmortem findings. The protocol includes all information in history of the case or in relation to preautospy data (e.g., antemortem changes, age, breed, etc.), findings of external and internal examination along with pathoanatomical diagnosis and epicrisis.

Postmortem report refers to a brief report of the protocol and usually consists of pathoanatomical diagnoses as cause of death. A vetromedical report must be brief to prevent confusion in the court.

Pathoanatomical diagnosis refers to specific diagnostic terms in the listed form after external examinations of different organs of various systems in the dead body. The terms like pneumonia, cirrhosis and nephritis are pathoanatomical diagnoses inferred from observations of changes like degenerations,haemorrhages or inflammations in various organs.

Description of lesions

In describing a lesion in a correct manner, descriptive terms such as colour, shape, consistency, odour, appearance of cut surface and relationship of the lesions to the surrounding structures are cautiously taken into consideration. A regular practice of this kind makes one a keen observer. Patholohgy is an observational science. Preautopsy data refers to certain informations like time, place, position of the cadaver, weather, description of the agonal state, position of cadaver after death, owner, species, breed, sex, size and weight etc. It is very important to know the position of the animal before death and also after its death for correct interpretations of conditions like hypostasis or lividity etc.

Hypostasis or cadaveric lividity (livor mortis) refers to bluish red spots in the subcutis of the side upon which the animal has been lying down before death. This condition arises from excessive accumulation of blood as seen in veins of the most dependent parts of the body due to gravitation.

Epicrisis refers to the personal written opinion of the autopsist about the entire case. The following facts are included in the epicrisis:

1. Pathogenesis of the disease.
2. Cause of death.
3. Opinion about the primary lesions regarding its cause or development.
4. Secondary and tertiary lesions regarding their occur-

rence and relationship to the primary lesions.

5. Statement concerning the importance of certain possibly co-existing lesions.

Rigormortis

It begins soon after death of the animal (say, after 4 to24 hours). In some cases, it lasts usually 24 hours. Sometimes, it may continue for 48 hours or longer and disappears with onset of decomposition or putrefaction. It appears first in the eyelids, than the masseter and disappears in the same manner. (i. e. disappearance seen first in the eyelids) It causes and reaches at maximum level after 20 to 24 hours and then it declines quickly.

It is characterised in dead animal by hardening and contraction of all the voluntary and involuntary muscles arising from coagulation of the myosin of muscles by lactic acid produced from muscle glycogen due to lack of oxygen. Rigormortis is related to breakdown of the muscle enzyme adenosine triphosphate and the energy so released is used for the muscular contraction during rigormortis. It disappears due to the softening of the coagulated myosin by autolytic enzymes.

Rigormortis which takes place in heart earlier than its development in skeletal musculature is powerful enough in the left ventricle to express the blood from it. Some clotted blood occurs in normal heart but presence in the left vertricle indicates incomplete rigor because of myocardial degeneration. Unclotted blood in the left ventricle indicates hypoxia or disapprearance of rigormortis from the left ventricle, currant jelly clot or chicken fat in heart arises from the processes of sedimentation and blood coagulation. Red cells are present in the currant jelly clot but absent in the chicken fat clot.

Algormortis (L=coldness+of death) refers to a gradual

drop in body temperature to that of the surrounding atmosphere in dead animals.

In vetrolegal cases, the veterinarians are confronted with the problems of differentiating antemortem wounds from postmortem wounds. Actually, there are no postmortem wounds or injuries. Wounds can only be produced during life of an animal.

Wounds or injuries like changes are made into the dead body of organs with bad motives. Organs also tear or rupture due to putrefaction or gas postmortem and postmortem wounds like changes are given below:

Antemortem Wounds

1. Presence of arterial haemorrhage.
2. Inflammatory and reparative processes are seen in the body.
3. Presence of gaping wounds due to stretched condition of the skin.
4. Presence of blood at the site of wound.
5. Spouting (i.e., flow of blood with great force from arteries).

Postmortem changes like Wounds

1. No haemorrhage and no blood flow after death of an animal. Slight venous escape of blood present
2. Absence of inflammatory or reparative processes
3. No gaping of the edges is seen
4. No clotting of the blood at the wound like sites
5. No spouting of blood

Sometimes, the necessisty for deciding age of injuries arises and the following facts help the veterinarians in arriving at a conclusion:-

i. Inflammatory changes around the site of injury and

changes in colour indicate the occurrence of injuries before death or probably 24 hours before death.

ii. Haemorrhages and arterial spouting do not occur at the so called injuries site after death of the animal.

iii. Coagulation of blood occurs during life or within 10 minutes after death.

iv. Edges of the wound may be everted or retracted and these changes occur during life or not within more than three hours after death.

(a) Healing of superficial cut in 10 to 24 hours.

(b) Presence of inflammatory swelling within 36 hours.

(c) Union of the edges of the incised wounds in 48 hours.

(d) Presence of complete healing in 4 to 7 days, leaving behind a tender scar.

(e) Growth of the granulation tissue to fill in the gaps of the wound in about 7 days.

Direct causes of death from wounds are haemorrhage, injury to vital organs like heart, lungs, brain etc., and shock. A blow on the heart or abdominal region can cause shock in an organism and necrosis, septicaemia, payemia, inflammation of internal organs, diseases, (e.g., tetanus,) are indirect causes of death. Cessation of respiration and circulation, cooling of the body, primary flaccidity, rigormortis, secondary flaccidity, putrefaction and mummification are the main signs of death in the animals. After a few hours following death, the body temperature falls and becomes equal to that of the atmospheric temperature, (i.e., cooling of body). Primary flaccidity (relaxation of the muscles) occurs immediately after death and lasts a few hours. Secondary flaccidity (relaxation of the muscles) becomes apparent with the onset of putrefation and decomposition. The putrefaction is very rapid at 100^0 F Air and moisture promote putrefaction.

The signs of putrefaction are as follows :

1. Abdomen distended with gas.
2. Blood stained fluid from mouth and nostrils.
3. Liquefaction of eye balls.
4. Presence of obnoxious odour or smell.
5. Bursting of the abdomen and thorax with protrusion of stomach and intestine through it or eversion of the rectum through the anus.
6. Semifluid consistency of the tissue.

A putrefied body is not suitable for postmortem examination but putrefied tissues can be used to perform some tests to ascertain the cause of death as done in the suspected cases of anthrax e.g., Ascolis test to diagnose the anthrax cases.

A veterinarian is frequently requested to give wound certificate in medico or vetrolegal cases. The following form can be used for writing a wound certificate:

No. Date

This is certify that at the request of

(1) .....
I have this day examined.

(2) .....

Having the following identification marks

.....

(3) .....

The said animal has got the following injuries on its body

(4) .....

I am of opinon .....

Place : Signature

Qualification

Designation

Autopsy Techniques of Different Animals and Birds

The postmortem techniques as followed by veterinarians in countries like Sweden are very satisfactory, systematic and can lead to valuable findings in animal disease investigation. These methods definitely merit the acceptance and adoption in the veterinary colleges in India. A mere observation of the lesions in the organs of body exposed by untrained persons is not a healthy practice.

The techniques to be adopted for postmortem examination in animals like cattle, horse, sheep, pigs and dogs etc., are given below:

Autopsy technique in large ruminants (Rubarth, 1964).

The technique in vogue in Sweden is as below :

The body is supported on its back and inclined towards its left side. Evisceration of abdominal and thoracic cavities is performed from the left side.

The **hind legs** are abducted by cutting through the medial thigh muscles, opening the hip joints and cutting through the teres the accessory ligaments.

The udder is removed from females. In the case of males, the penis and prepuce are drawn backwards to avoid being damaged when the abdominal wall is incised. This is done in the following manner. First, the parietal tunica vaginalis is incised and opened as far as the external inguinal ring and, then, the penis prepuce are dissected free as far back as the ischialarch. The abdomen is, then, opened by an incision running along the linea alba from the xiphoid cartilage backwards to the pelvis (do not extend the incision too far forwards otherwise the disphragm will be damaged). The abdominal wall is reflected by incising along the costal arch and, in females, along the anterior border of the pelvis. In males, the abdominal wall is incised from the midline towards the inguinal canal so that spermatic cord, the testicles can be freed.

After the abdomen has been opened and the abdominal walls reflected, the omentum is freed along its insertion to the lateral grooves of the rumen and from the duodenum, but the spleen is left attached to the rumen.

The **fore stomachs and abomasum** are removed. The duodenum is sectioned between two ligatures at the pylorus. Then the rumen is freed from its attachments to the dorsal abdominal wall while the assistant pulls it over to the left side of the body. The oesophagus is then cut through(be careful of the diaphragm), the forestomachs and abomasum are freed from their remaining attachments, and removed to the left side of the body.

The **intestinal tract** except for the duodenum is removed. After ligating the duodenum at the junction between its second and third parts, the duodenum and the pancreas are exposed and freed as much as is possible at this stage. The rectum is then freed, ligated and divided. The intestines are removed in one piece by cutting the mesentery along its attachments from the rectum and far forwards as the root of the mesentery. The duodenum and pancreas are allowed to remain in the abdomen for subsequent removal together with the liver. When the mesenteric root is cut through, the intestinal tract can be lifted out of the abdomen.

The urogenital organs, adrenals and rectum are removed. The floor of the pelvis is removed by sawing from the anterior and posterior borders through the obturator foramen on each side (do not saw too far laterally).

The penis has meanwhile been freed from the ischial areh. The kidneys and adrenals are freed together and by carefully pulling them backwards, the ureters will be freed from the dorsal abdominal wall as far backwards as the urinary bladder in females. The broad ligament is incised along its insertion on the abdominal wall so that the ovaries fallopian tubes and uterus accompany the kidneys and ureters. All these organs are gathered into the left hand and

then are drawn backwards at the same time as the attachments to the pelvic walls are cut through by cautious use of the knife and incision around the anus completely frees these organs.

The abdominal aorta is opened in situ.

The dispharagm is cut through along its insertion beginning at the xiphoid cartilage (avoid incising the pericardial sac).

Any pleural contents are collected and the amount is measured.

The appearance of the pleura is inspected.

The **pericardial sac** is freed from the sternum by inserting the edge of the hand anterior to the pericardial sac and drawing the hand backwards, keeping it as close to the sternum as far as possible. If any fluid is present in the pericardial sac, it should be collected and the amount is measured.

The structures of the **oral cavity and neck** are then removed. An incision is made on each side of the tongue along the borders of the mandibles as far forwards as the symphysis. The tongue is, then, pushed upwards between the mandibles, grasped and drawn backwards at the same time as the first incisions are extended backwards. The soft palate is cut free by incisions from each side which run forwards and medially to meet in the midline. The hyoid bones are divided at the joint between the main branch and the thyroid branch.The knife is placed at this junction with the sharp side upwards so that the great branch is lateral to the knife and the thyroid branch medial to it. If the knife has been correctly placed, a single sharp jerk will separate the bones. The pharyngeal structures are, then, dissected free laterally and dorsally. The trachea and oesophagus are freed to half way down the neck and then cut through.

Inserting two fingers through a short transverse incision between two of the cartilage rings close to the free stump are then drawn backwards and freed as far as the thoracic aperture. After opening the aperture, the trachea and oesophagus are pushed into the thoracic cavity. The left hand is then inserted into the thoracic cavity from the abdomen and the trachea is again picked up through the transverse incision. The trachea is then drawn backwards and the thoracic organs freed from the dorsal thoracic wall by cutting through the mediastinum.

The **thoracic aorta,** is removed together with the other thoracic organs.

The **head,** with the salivary glands attached, is removed by cutting the atlanto-occipital joint.

The body **lymph nodes** are then examined. On the **fore limbs** (after these have been cut down), the prescapular, and axillary, are examined. On the **hindlimbs,** the popliteal lymph nodes as well as the deep inguinal and the internal iliac lymph nodes should be checked for lesions.

The **joints** are opened from the medial surface beginning distally on the limbs.

The **skeletal musculature** is inspected by means of several incisions, not forgetting the back and neck muscles.

The spinal cord is exposed by removing the dorsal arches of the vertebrae if examination is warranted.

Autopsy of Horse

The autopsy technique is much the same as in the cattle and buffalo except for the abdominal organs. The body is placed on its back and inclined towards its right.

Evisceration of the abdominal and thoracic cavities should be performed from the right side.

Inspect the contents of the abdominal cavity. The

position of the organs is inspected, particulary the position of the left ventral and dorsal colon and the pelvic flexure. The diaphragm should be normally arched and tense, if it is not, ascertain the cause.

Winslow's foramen is located by following the visceral surface of the caudate lode of the liver mediocranially. The foramen is a narrow slit which will admit two fingers.

The great colons are lifted out of the abdomen to the right side of the body.

The small intestine is freed along its mesenteric attachment beginning with the duodenum at the site of the duodenocolic ligament. This ligament connects the third part of the duodenum and the large intestines at the termination of the right dorsal great colon and the beginning of the small colon. The small intestine is divided between two ligatures and then, is freed along the mesenteric attachment as far back as the ileum where it is divided again between two ligatures. During removal of the small Intestine, the assistant should stand on the left side of the body and help by drawing out the intestine and tensing the mesentery.

The rectum, small and great colon, and caecum are removed together. The rectum is divided in the pelvis and the intestines are freed along their mesenteric attachment as far forward as the right dorsal great colon. In this region, the pancreas is to be left in the abdominal cavity (it is removed later together with the liver and duodenum), it is necessary to free the great colon from the pancreas and the dorsal abdominal wall by blunt dissection or by careful use of the knife in places. At this point only, the branches of the mesenteric blood vessels connect the great and small colon, caecum and rectum to the body. These blood vessels are freed as far out as possible and then cut close to the intestinal wall. The large intestinal are now completely freed and be lifted out to the right side of the body.

The spleen and omentum are removed.

The stomach is removed. The duodenum is divided between two ligatures immediately posterior to the pylorus. Then, a short longitudinal incision is made through the serosa covering the portion of the oesophagus which extends into the abdominal cavity and the stomach is, then, freed of its ligaments and removed from the body.

The liver, duodenum and pancreas are removed together. The pancreas is first freed from the root of the mesentery and the dorsal abdominal wall and then the lateral and falcifrom ligaments of the liver are cut through. The incision through the wall of the vena cava is then slit by the insertion of the finger while the liver is held towards the right side of the body. The liver is now attached to the body by only one wall of the vena cava and when this is cut through, the liver can be removed together with the pancreas and duodenum. Removing the liver in this manner avoids damage to the diaphragm.

The **mesentery and the abdominal aorta** are removed. The aorta is divided immediately behind the diaphragm and in front of the root of the mesentry and by drawing gently backwards, it can be freed from the dorsal abdominal wall. The aorta, with mesentery attached, is then removed by cutting the iliac branches as far distally as possible (about10 cm).

The remaining steps in the horses are similar to those followed during autopsy in cattle and buffaloes.

Autopsy of Small Ruminants

The technique is similar to that for larger ruminants except that the sternum is removed by cutting through the costochondrial junctions. The neck and thoracic organs are, then, removed together, i.e., without cutting of the trachea and oesophagus anterior to the thoracic aperture.

Autopsy of Carnivores

On the whole, the autopsy technique for these species follows the description described above but with the following differences.

Examination and removal of the abdominal organs.

The incision along the linea alba should not extend farther forwards than the xiphoid cartilage, otherwise, the diaphragm can easily be damaged. The omentum with the spleen is lifted up and folded over the thoracic wall so that position of the abdominal organs can be inspected.

The intestinal tract is removed. Divide the rectum in the pelvis, after ligatation, if necessary, and then work forwards by cutting along the mesenteric attachments. Continue until the tip of the duodenal branch of the pancreas is reached. This represents the junction between the duodenum and jejunum. Ligate the small intestine at this point and divide it.

The omentum and spleen are removed along the insertion of the omentum, taking care to avoid the splenic branch of the pancreas.

The liver stomach pancreas and duodenum are removed together.

Begin by freeing the pancreas from the root of the mesentery and the dorsal abdominal wall. The ligaments of the liver are, then, freed to avoid damaging the diaphragm. Follow the contour of the liver very closely. The stomach is, then, cut free through the cardiac region, the incision should not be made too far otherwise the plieural cavities will be opened. Then, cut through the vena cava to free the liver completely. After breaking down or cutting through any remaining ligaments, the organs can be removed in one piece.

The autopsy is, then, continued as for the cattle with the exception that the sternum is removed by cutting through

the costochondral junctions before taking out the neck and thoracic organs in one piece.

Autopsy of Pigs

The technique is similar to that applied to carnivores except for removal of the intestinal tract.

To remove the intestinal tract, begin at the tip of the colonic spiral and free the entire intestinal tract along the mesenteric attachments orally and aborally.

In large pigs, the stomach is removed separately. In smaller animals, the stomach is removed together with the liver, duodenum and pancreas.

Autopsy of Birds

1. The legs are abducted by cutting or breaking open the hip joints.
2. The abdomen is opened and the sternum is freed by lateral incision through the ribs.
3. The crop is then freed by blunt dissection from its attachment along the thoracic aperture to avoid being damaged when the heavy anterior osseous attachments of the sternum (the coracoid and clavicle) are clipped through with bone tongs on each side to join the lateral incision through the ribs.
4. The spleen is removed separately. The stomachs and intestinal canal are removed in one piece after cutting through the oesophagus just anterior to be proventriculus.
5. In sexually mature females, the ovary is removed at its base and the oviduct is first extended by cutting through its dorsal and ventral mesenteric attachments and then removed by cutting through the cloaca.
6. The pericardial sac is incised and the chambers of the heart is opened in situ by incising the wall or the right

ventricle near the apex and continuing the incision anteriorly up through the pulmonary artery and laterally up through the right atrium. The procedure is repeated for the left ventricle extending an incision at the apex up through the aorta and up through the left atrium, the heart is then removed by cutting through its base.

7. The lungs are freed by blunt dissection from the thoracic walls cutting through the dorsal attachment (dorsal to the thoracicoesophagus and aorta), and, then, removed by cutting through the trachea immediately anterior to the syrinx.
8. The upper beak is cut transversely at its base to expose the nasal cavities, and, then, the mouth is opened by cutting throught one corner (the right is most convenient) and the incision continued throught the pharynx and down the oesophagus to open the crop. The trachea is, then, opened along its whole length.
9. The brachial plexus and the sciatic nerve are exposed on both sides.
10. The major joints are opened.

Precautions

When a case of a dead animal is presented for postmortem examination, there are certain extremely important precautions to be remembered and followed during autopsy work. Postmortem work done in a haphazard manner will lead one to no conclusion with waste of the material and time.

The precautions are as follows:

1. Consideration of a postmortem case should be made as a serious bit of research work and anamnesis (history) of the case should be read carefully. If necessary more information about the dead animal can be obtained from either the attending veterinarian or owner of the animal.

2. Special attention is given to the organs in the dead body of the animal which have shown clinical signs of abnormality.
3. Lesions in the bodies should be noted as encountered during postmortem work. Postmortem findings should never be written from one's memory after the work is over or even after lapse of a day or two. There is always a risk of forgetting very valuable findings after passage of sometime from the moments of postmortem examination.
4. Materials for bacteriological, parasitological and chemical examination etc. should be removed and properly preserved for further laboratory investigation.
5. The skin of the dead animal should be carefully examined on both sides of the body and distinguishing marks or any abnormalities e.g., discharges from naturalorifices should be recorded .
6. The dead body should be examined to ascertain the state of nutrition, inanition and anaemia etc.
7. The autopsist must obtain the **authority** to do postmortem examination from police, attending doctor or the owner of the animal and he should be properly dressed for this kind of work. Gum boots, white coat, apron and rubber gloves should be used invariably as a routine measure before commencement of the autopsy work.
8. Nutritional state (obesity vs. emaciation) and wasting conditions affect the relative weights of the organs in dead animals.

The formula for calculating relative weight in dead animals is as follows :

$$\text{Relative weight} = \frac{\text{Weight of organ}}{\text{Weight of the body}} \times 100$$

9. Descriptive terms likes size, shape, weight, colour, odour, consistency, incision, contents, appearance, of cut surface and relationship of the lesions to the surrounding are to be used in depicting changes or lesions in the organs of the bodies in a careful and objective manner as they are exposed or encountered while examining the dead bodies' postmortem.
10. A first aid kit should be readily available in the postmortem room to treat minor cuts or pricks incurred during autopsy.
11. Gastrointestinal tract should be left to the last for the sake of examination during autopsy.
12. For collecting the fluid or blood for cultural examination, heart wall should be first seared with red hot spatula and the fluid is drawn into sterilised capillary pipettes.
13. During the postmortem examination, films and cultures are made from the different fluids and tissues. The surface of the organ should be seared with red hot spatula in order to be picked up with sterilised scalpel and the material is picked up with sterilised platinum loop in the hot flame for bacteriological examination using suitable media. Culture can be made from bone marrow in the animals which have been dead for a long time. The bodies of such animals are usually in very advanced stage of decomposition.
14. The various inoculation routes for administering the extract of lesions in the animals are subcutaneous, intracerebral, intravenous, intraperitoneal, intradermal. intratesticular and intravenous etc. The inoculation site is clipped or shaved and cleared with cotton soaked in ether or spirit. For collecting blood, the needle is inserted into the vein in the direction of blood flow.
15. All the contaminated instruments and discarded cultures etc., should be placed in Big jars containing disin-

fectant solution like 5 per cent carbolic acid or 1 per cent Lysol solution in water.

16. A long dead animal is useless for bacteriological examination.
17. Blood films for microscopical examination are made thin ones as far as possible. Dry the blood films immediately by waving them in the air. Prepare thick blood films in cases of anthrax with the help of a platinum loop. Interacellular parasites *(say. Babesia sp.)* and leucocytes are found easily in large numbers at the edges or termination of the blood films.
18. Sputum for detecting tuberculosis organisms may be collected from the wall etc., in front of suspected TB cases. The animal can be made to cough on a piece of a large sheet placed over the wall. Thick or turbid mucus from TB patients is quite satisfactory.
19. Nematodes should be collected in a test tube containing normal saline (0. 85 per cent sodium chloride in distilled water) and the tube is shaken well to clean the parasites. Hot 70 per cent alcohlol containing 3 to 5 percent glycerine is poured over the worms to kill and fix them. The worms get cleared after sometime in this solution.
20. Materials from animals showing symptoms or **representative lesions** of bacterial, viral or parasitic diseases etc., are collected in the conditions as given below:
    (1) Acute stage of the disease.
    (2) Immediately after death of the animal.
    (3) After sacrificing the diseased animal.

Disposal of the dead bodies of the animals is done by burial or burning the dead body alongwith dungs, bedding, blood stained soil or discharges in order to prevent the spread of outbreak of diseases (e.g., anthrax). Burning of the dead bodies may be disadvantageous owing to high cost of the

fuel. But one should be extremely careful in burying the cadavers. At first, a trench of the adequate size with a depth of atleast 6 feet is prepared by digging the earth. A foot of lime is layered over the trench and the carcass is laid over it. The whole dead body must have a covering of a foot of lime from all sides (i.e., top, sides and base etc.). Burial sites must be located far from human dwellings or drinking water resources (e.g., well). If possible, one can burn the cadaver using an incinerator. Spores of anthrax are known to be spread by **earth worms** from trenches having improperly buried dead bodies of anthrax cases.

Some Common Specimens for Laboratory Tests

Blood Collection

There are suitable sites in different animals for taking blood from veins for haematological and cultural examination as given below:

Table 40. Sites of blood collection

Sites	Animals
Jugular vien	Cows, buffaloes and horses
Ear veins may also be used for this Purpose	Sheep and goats
Anterior vena cava or by cutting the end of tail or tip of the ear	Pig
Saphenous vein	Dogs and cats
Brachial vein (on the ventral surface of the wing)	Fowls.

Hairs or feathers are removed from the sites of blood collection. The sites should be disinfected with methylatyed spirit or 70% alcohol. Aseptic precautions are taken while collecting the blood for cultural and bacteriological examination.

1. Serum

It is prepared by allowing the blood to clot by keeping the glass tubes in slanting positions at a cool place. Separated serum can be preserved on ice or one drop of 1% merthiolate (Aq. Sol.) is added to 10 ml of the serum as a preservative. 1 ml of 5 per cent solution of phenol can be mixed with 10 ml of serum for preservation.

1. Blood

It is collected from the patients for investigational purposes and can be preserved with suitable anticoagulants.

Anticoagulants for hematological studies.

There are different kinds of anticoagulants used for this purpose and some of these are as under:

1. EDATA: 1% (1ml for 10 ml of blood)
2. 4% Sodium citrate (1 ml for 10 ml of blood)
3. 1 ml of 10% solution of potassium oxalate for 3 oz of blood is used for chemical analysis. No oxalated blood is used for determination of calcum, magnesium or acetone in the blood.
4. Mixture of 6 parts (say, gms) of ammonium oxalate and and 4 parts of potassium oxalate in 100 ml of distilled water is prepared and kept in a reagent bottle. 0.3 ml of this solution is taken in small specimen bottles and kept in the incubator overnight to allow the evaporation of water. The sedimented salt is sufficient for 5 ml of blood.
5. Heparin 1% solution - Rinse the vial or syringe with 0.1-2 ml solution for 10 ml of blood for glucose estimation.

2. Faeces

It is collected directly from the rectum at the time of defaecation. 4- 10% of formalin is used for collecting faeces to detect heminthic eggs. Postassium dichromate is a suitable

fixative for coccidial oocysts. Faeces is suitably emulsified in the preservative.

3. Urine

It is collected during micturition or with the help of a sterilized catheter. It is preserved on ice in sterilized tubes for bacteriological examination. 2 drops of 40% formalin or toluene sufficient to form a thin layer over the surface of the urine in glass container is used in different investigations or chemical analysis etc.

4. Milk

It is collected in the sterile glass containers for cultural work in cases of mastitis. The first few drops should be discarded and, then, it is collected in sterilized glass containers to be preserved on ice. 0.1% boric acid is added for examining milk for acid fast organisms like ***Mycobacterium tuerculosis.***

5. Pus

For its collection, sterile swabs are used. Pus may be collected directly from an abscess in sterile glass vials for bacteriological examination. Usually thin smears are prepared from pus for microscopical examination.

6. Tissues

There are different purposes for the collection of tissues from the organs of dead animals.

Three important objects are as under:

1. Bacteriological virological and parasitological examinations etc.
2. Histopathological examination.
3. Chemical examination.

I. Bacteriological Examination

Specimens like liver, spleen, lungs and lymph nodes etc.

are collected with aseptic precautions just after the postmortem examination in sterilized containers. At first, the surface of the heart is seared with hot spatula, sterilised capillary pipettes or syringe or needles are used for blood collection. Fluid from thorax, abdomen and pericardium is collected for cultural work with the help of sterile capillary pipettes.

II. Histopathological Examination

Small pieces of tissue (about 0.5 cm thick or 2 cm cubed) showing typical representative lesions are removed from the different organs and placed in glasswares containing 10% formal in saline solution. Collection of some adjoining normal tissues with the representative lesions is preferred for the sake of comparison with normal features during the microscopical examination of the stained tissue sections. There is a demerit of liquefaction or autolysis in big chunks of tissues collected for preservation and so the tissue pieces selected should be small in size and placed in a preservative which is 10-20 times more than the volume of the tissue pieces taken together for the sake of proper fixation of tissue elements.

III. Chemical Examination

Sometimes, animals are willfully poisoned with chemicals under the influence of ulterior or criminal motives and the different organs in such cases are required to be examined in the forensic laboratory to identify the poison used to kill the animal. The duplicate samples or materials from different organs and stomach or intestinal contents are preserved in saturated solution of sodium chloride (common salt) or rectified or methylated spirit for onward transmission to the forensic laboratories to detect the poisons. Suspected plants or feeds are sent to the laboratory in the unpreserved state.

Collection of Materials in Different Diseases

Smears from tissues or blood are prepared for microscopical examination to identify the aetiological factor for producing the diseases. Different organs in different diseases are needed for bacteriological and histopathological examinations.

There are many animal diseases which require laboratory examinations to arrive at a definite diagnosis. Bacteriological, virological, parasitological or chemical examinations of these materials are done in the laboratories. The types of specimens as noted in table 41 against the different diseases for ready reference are as under:

TABLE 41. Diseases and speciments required for innestigation in some bacterial viral and protozoan diseases etc.

Diseases	Required Specimens/Materials Collected
1. Anthrax	Blood smears, smears from swellings in case of horse, pigs, dog and cat. Pieces of skin and spleen (even from putrefied organs for Ascoli`s test).
2. Actinomycosis and actinobacillosis	Pus smears from the different parts of the lesions, Affected organs both on ice and in 10% formalin.
3. Black quarter and malignant oedema	Smears of haemorrhagic muscle exudate from freshly dead animals (air dried on ice) and in formalin soaked cloth separately.
4. Botulism	Suspected food and intestinal contents on ice.
5. Brucellosis	Blood and serum samples, quarter milk samples, in case of abortion,. Foetal stomach and heart on ice.
6. Chronic respiratory disease (CRD) Enterotoxaemia	Affected live birds, or dead birds packed on ice. Serum samples. Smears from bowel mucous membrances. Samples of ingesta from duodenum, jejunum and ileum. Ingesta preserved with 2 or 3 drops of chloroform.
7. Fowl cholera	Affected birds, heart, blood, liver and spleen on ice.
8. Fowl spirochaetosis	Sick birds and blood smears from such birds.
9. Glanders	Swabs from nasal and skin lesions (farcy cords or buds etc.) Lungs and other organs showing lesions on ice and in 10% formal saline separately.
10. Johne's disease	Smears from rectal mucosal scrapings. Portions of intestines and associated lymph nodes (mesenteric lymph nodes) on ice and in 10% formal saline separately.

Diseases	Required Specimens/Materials Collected
11. Leptospirosis	Portions of kidney and liver on ice and in 10% formal saline. Blood (collected) during febrile stage on ice. Freshly voided urine on ice. Serum samples.
12. Listeriosis	Aborted foetus or foetal stomach and heart on ice. When there are symptoms of circling disease in the patients, portions of brain, liver, spleen and kidney on ice and in formal saline are preserved separately.
13. Haemorrhagic septicaemia	Smears of blood and exudate obtained form oedematus swellings with help of sterile syringe and needle. Blood from heart, portions of liver, spleen, kidneys and lungs showing pneumonic lesions. Long bones packed on charcoal.
14. Contagious bovine or carpine pleuropneumonia	Bovine or caprine blood serum. Portions of affected lungs and associated lymphnodes on ice and also in 10% formal saline.
15. Pneumonia due to other infections agents	Portions of diseased lungs and associated lymphnodes on ice, portion of lungs in glycerine saline and portions of diseased lungs in formal saline.
16. Tuberculosis	Portions of lesions unpreserved on ice or in 25% glycerine saline and in 10% formal saline.
17. Swine erysipelas	In acute cases, heart, blood, portions of liver, kidneys and spleen on ice. In chronic cases, affected joints on ice. Organs showing lesions in formal saline. Serum samples.
18. Ulcerative lymphangitis and Caseous lymphadenitis	Swabs and smears from the lesions. In cases of caseous lymphadenitis pieces of affected organs or lymph nodes.
19. Vibriosis	Foetus or foetal stomach over ice. Portion of placenta and uterine discharge on ice.
SOME VIRAL DISEASES	
DISEASES	**SPECIMENTS REQUIRED TO BE COLLECTED**
1. African horse sickness	Blood, spleen and lungs in 50% sodium citrate and carbolic acid.
2. Foot and mouth disease, Vesicular stomatitis and vesicular exanthema Infectious bronchitis	Lungs including trachea, spleen and heart blood in buffered glycerine and on ice. Serum samples from old cases. Lungs and trachea in formal saline.
3. Infectious laryngotracheitis	Live birds in early stages of disease or tracheal exudate in glycerine saline. Portion of trachea in formal saline.

Diseases	Required Specimens/Materials Collected
4. Pox in sheep, goats, pig, cattle, contagious pustular dermatitis in sheep and goats.	Swabs in 50% glycerine saline or unpreserved.
5. Fowl pox	Scabs in 50% glycerine saline or unpreserved. Sick birds or animals, Brain and spinal cord in formal
6. Avian encephalomyelitis and infectious porcine encephalomyelitis	Saline and buffered glycerine respectively. Serum from convalescent animals.
7. Avian leucosis complex (ALC)	Affected birds or portions of liver, spleen, nerve and other organs showing lesions in 10% formalin saline.
8. Canine distemper	Pieces of liver and spleen on ice. Portions of urinary bladder, kidneys, liver, lungs and trachea in formal saline.
9. Louping ill	Serum from acute and convalescent cases. Defibrinted or oxalated blood, portions of brain cord from freshly dead carcasses or buffered glycerine and in 10% formalin saline.
10. Equine abortion	Portions of placenta, foetal organs in glycerin saline. Foetal stomach and heart blood on ice. Pieces of liver, spleen, lungs and trachea of foetus in 10% formalin saline. Serum of the mare.
11. Infectious bursal disease (Gumboro disease)	Serum, Bursa of fabricious and kidneys in charcoal medium and in 10% formal saline.
12. Infectious canine hepatitis (Rubarth disease)	Liver, gall bladder and kidney in 10% formal saline.
13. Marek's disease	Live birds for isolation of virus and histopathology, Serum and suspected material in 10% formal saline or charcoal medium.
14. Pseudorabies	Brain and spinal cord in glycerine saline and in formal saline.
15. Psittacosis and ornithosis	Serum from acute and convalescent cases. Whole blood on ice. Dead or sacrified bird wrapped with lysol soaked cloth and packed over ice.
16. Rabies	Head packed over ice. Brain (half of it divided longitudinally in formal saline and the other half in glycerine saline.)
17. Ranikhet disease	Freshly dead birds packed over ice and brain, spleen and liver in 50% glycerine saline

Diseases	Required Specimens/Materials Collected
18. Rinderpest and mucosal Disease complex	Defibrinated blood prepared during height of temperature, pieces of lymph nodes and spleen on ice or in buffered glycerin.
19. Swine fever	Defibirinated blood from living animals, heart blood, liver and spleen on ice. Organs (including brain) showing lesions in 10% formal saline.
20. Swine influenza and virus pneumonia of pigs.	Portions of lungs showing pneumonic lesions on ice, in buffered glycerin and in formal saline separately.
Some important protoxoan and fungal diseases etc.	
1. Anaplasmosis	Thin blood films. Citrated or oxalated blood from sick animals.
2. Coccidiosis	Faeces in 10% formalin or in a 2.5 per cent pot-dichromate solution. Portion of intestine showing lesions in formal saline.
3. Piroplasmosis (Babesiosis)	Thin blood smears. Heart blood, spleen and kidney smears from dead animals, citrated or oxalated blood.
4. Theileriasis	Thin blood films. Smears or biopsy tissue specimens from prescapular lymph node, liver and kidneys on ice and in 10% formalin saline.
5. Trichomoniasis	Uterine discharges in sterilised glasswares (collected within 24 hours of abortion or during heat period) on ice.
6. Trypanosomiasis	Citrated blood films. Serum to be collected in camel, cattle or buffaloes.
7. Parasitic and fungal infections	Faeces samples in formalin.
8. Mange and parasites	Skin and deep skin scrapings. The parasites collected from the gut or heart or body cavities etc., of animals during autopsy are first kept in hot solution of 5% formalin for 2 minutes. Then, these are transferred into a glass container containing 70% alcohol to which a little glycerin is added for clearing the worms.
9. Fungal diseases	Portions of organs/tissues showing lesions, on ice and in formal saline separately.
10. Aspergillosis	Affected birds. Lungs along with caseous masses from the air sacs over ice and in 10% formalin saline.
11. Ring worm	Skin scrapings from the lesions, including some hair roots unpreserved in a tightly stopped container.
12. Neoplastic conditions (Tumours)	Entire tumour or small pieces 2 cm cubed or pieces less than 1/ 2 inch in diameter in 10% formal saline solution (20 times the volume of the tissue pieces)

Despatch of Materials to the Pathological Laboratory

The materials obtained from live animals either through biopsy or after their death should be sent by post with adequate precaution to prevent the materials from being spoiled during the transit. The materials can be carried in large containers like thermoflask containing ice. The laboratory should be provided with a copy of letter giving complete history or anamnesis of the case for enabling the pathologist to chalk out the proper kind of tests to ascertain the cause of death.

The history sheet of the case must include the following information :

1. Clinical symptoms of the disease with complete anamnesis.
2. Animals involved (species).
3. Breed, sex and age.
4. Number of animals attacked and total population of the susceptible animals.
5. Duration of the sickness or outbreak.
6. Number of animals dead.
7. Nature of feed, sources of water supply, management, houses and pens etc.
8. Postmortem changes.
9. Time elapsed between death of animal and collection of the material.
10. Type of material collected and despatched. Specify container or specimens, source material, giving case number and name of the owner.
11. Type of the preservative used.

Despatch of Live Animals and Birds

Small animals or birds like poultry, suckling pigs, lambs, kids and rabbits etc., are sent to the laboratory. It is better to

select only those cases with typical symptoms of the disease for laboratory investigation. These sick cases are sent through courier with proper precaution to prevent spread of the outbreak to healthy animals during transporation from place of occurrence of the disease to the laboratory.

Materials can be sent to the laboratory for histopathological examination and to Forensic Laboratory for identifying the chemicals etc. used to kill the animal.

The methods followed are as under :

1. Despatch of the materials for histopathological examination.
2. Despatch of the materials for chemical examination in a Forensic Laboratory.

Despatch of the Materials for Histopathological Examination

Requirements

Glass container, cotton, fixative (10% formal saline) and pieces of organs.

Procedure

1. Pieces of tissues (about 2 cm cubed) are removed from the organs immediately after death of the animal. Decomposed, autolysed or puterefied organs are useless for histopathology work.
2. A tissue piece is placed into atleast 10-20 times its volume of preservative in a screw capped jar (with wide mouth).
3. Place a pad of cotton on the bottom and, then, the tissue is place in the jar on it and pour enough solution of 10% formal solution (unless otherwise specified). Another pad of cotton is placed over the tissue.
4. After that, a label showing particulars of specimens (number, animals, organs, name of the owner and disease suspected etc., written with a hard pencil) should

be placed inside the jar which is then nicely stoppered. Paste another label on the outside of the jar or container.

5. Seal the mouth with molten paraffin and pack well. It is better to keep the bottle in a small cloth bag which should be protected in a wooden box of the proper size filled with wood dust or any soft packing material. Give the address of the sender on the wooden box with instructions to the post office i.e., **to handle it with care.**

Despatch of Materials in Vetrolegal or Medicolegal Cases of Poisoning in Animals

Police report and a sample of sealing wax and an impression of the seal used on a piece of paper along with forwarding letter are required to be sent to the forensic Laboratory in the packing box containing the specimens.

Each specimen bottle should contain sufficient quantity of preservative (about 4-5 times more than volume of the specimen used) and should be tightly stoppered and sealed before sending through a courier or by post. There is a risk of material being spoiled during transit when it is sent by post.

Process of Packing

Materials from organs of dead animals suspected to have been poisoned are first kept in a glass container (with wide mouth) containing preservative. (say, supersaturated solution of salt i.e., namak) a label giving details of the case number etc., is placed inside the glass bottle. The bottle is tightly stoppered and mouth is properly sealed with paraffin, sealing wax and is then kept inside a suitable cloth bag which is also suitably sealed and labelled. All the organs are, thus packed separately. The specimens are, then, kept in a larger wooded box which is also sealed in a cloth bag. Details of the addresses of the senders and of the laboratory for investigation are written on outside of the box and sent to the forensic laboratory through a courier. While doing postmortem examinations in police cases, the following facts are to be kept in view as precautions:

1. Obtain a letter from police **authority** for postmortem examination. Any postmortem examination should be done after having the letter of authorisation to do so.
2. It may be done in presence of a police representative and no unauthorised person should be allowed to witness the autopsy.
3. The postmortem examination should be done as soon as possible after death of the animal.
4. Examine all organs thoroughly following the autopsy techniques as described earlier for all important domestic animals.
5. A sample of preservative (about 2 oz) should be sent to the Forensic laboratory along with other specimens in a separate container in all medicolegal cases.
6. Measuring tape for accurately measuring the injury or lesions is used to avoid false guess of wound dimensions.
7. Glass bottles (with wide mouths) are needed to collect viscera. Have enough glass slides, pipettes and spirit lamps to fix the smears on the slides.
8. Postmortem findings should be recorded on the spot of postmortem examination in a notebook with the help of an assistant and nothing should be recorded from one's memory after leaving the place of autopsy. Help of at least 3 person is needed to complete the postmortem work.

Preservatives used in Cases of Poisonings

1. Use saturated solutions of common salt (sodium chloride) or rectified spirit for preserving specimens like stomach or intestinal contents and pieces of organs like liver, heart spleen and loops of intestine etc. Use sufficient preservative (about 5-10 times the total volume of the pieces of organs) in order to facilitate examinations. In cases of suspected HCN poisoning, 1% mercuric chloride is a good

preservative. The blood or muscle can be sent on ice in suitable containers. The weights of the organs and stomach contents etc., to be preserved for chemical analysis in large animals are as under :

Liver	:	1/2 kg.
Kidney	:	1/2 kg.
Ruminal contents	:	1/2 kg.

The different organs should be packed separately in sterile airtight containers.

Veterolegal or Medico-legal Examination on a Live Case

The animals suffer from accidental or wilful injuries, weapon injuries and other offences and these are brought to veterinary hospitals for the sake of injury reports.

Precaution

1. Prepare a correct and thorough description of the animal.
2. Describe the injuries in details (age, depth, size, direction, situation or relation to the surroundings).
3. Record the probable cause of injuries and indicate prognosis of the injuries.
4. Give your accurate opinion about nature and outcome of the injuries done to the animal. Avoid use of vague or ambiguous words leading to risks in the court-enquiry. Injury reports prepared in a slipshod style warrants punishments to the investigating doctors.

Common Diagnostic Tests

Mastitis

There are many methods to diagnose mastitis in lactating livestock. Some of these techniques are as follows :

1. Bromothymol Blue Test

In mastitis the reaction of milk changes to alkaline pH (early 7.4). This can be detected by bromothymol-blue test.

Bromothymol-blue stock solution.

Dissolve 1 gm. of bromothymol-blue in 500 ml. of 47% alchohol.

Procedure

1. Take 1 ml. of Bromthymol-blue solution in a graduated 10 ml. cylinder.
2. Add to this 5 ml.of fresh milk and mix both by inversion once or twice. If the milk contains inflammatory exudates, mastitis is indicated by development of green, dark green or blue colour. In normal case, the colour of mixture is yellow or slightly yellowish green.

Strip Cup Test

While milking, milk the first 2 or 3 streams from the teats in the strip cups from the four quarters (i.e., left fore, lef-thind, right hind and right fore teats). The strip cup base has a black surface and any clots or flakes are easily detected. If clots are seen in the milk drawn from the teats of the cows or buffaloes, the case is positive for mastitis and segregate them immediately.

White Side Test

The principle of this test is based on an increased leucocyte count of mastitis milk.

Requirement

A glass plate with underside painted black, 2 droppers, broom stick and 4% sodium hydroxide solution.

Procedure

Place 5 drops of suspected milk on the glass plate and

add to it 2 drops of 4% sodium hydroxide solution. Stir rapidly with the broom stick for 20 to 25 seconds. If the mixture becomes thick and viscid, then the case is positive for acute mastitis.

Note : Milk collected in sterilised class vials is sent to district or provincial investigation laboratory for bacteriological investigation in thermoflask containing ice.

Urine Examination

Use a clean container for collecting fresh urine. Test reaction with litmus paper. Record colour, odour and transparency of the urine sample.

Specific Gravity

Place the urinometer in urine and record the sp. gr. In decimals as 1.050. There should be no bubbles in the urine to confuse recordings on the scale.

Albumin Determination

1. Take 2 ml. of Robert's reagent or Nitric acid (concentrated) in test tuble.
2. Add 2 ml. of urine on the reagent slowly with a dropper.
3. A white or grey ring at the zone of contact of two fluids indicates albumin.

Report as given below:

Trace	+
Moderate	++
Heavy	+++

N.B

Boil about 5 ml. of urine in the upper part of the test tube for 3 minutes. A cloudy precipitate appears in the presence of albumin in the heated upper part of urine in the

test tube. The precipitate does not disappear on addition of dilute acetic acid solution to it.

Acetone Determination

Rothera's Test:

1. Take 3 ml. of urine in a test tube.
2. Add 1 ml. Rothera's reagent and mix.
3. Layer 1 ml. liquor amm fortis over the solution in a tube. Development of a permanganate colour at the zone of contact with in a minute indicates a positive reaction.

Sugar Determination (Benedict's test)

1. Take 0.5 ml. (7-10 drops) of urine in test tube.
2. Add 5 ml. of Benedict's reagent to the urine.
3. Mix well and boil the mixture on a spirit lamp for 5 minutes.
4. Allow to cool.

Record reading as given below:

Green colour with slight yellow sediment traces

Green colour with heavy yellow sediment +(1.0%)

Yellow colour with heavy sediment ++ (1.5%)

Yellow colour with heavy reddish sediment +++ (2.0%)

Occult Blood Examination (Haemoglobin)

1. Prepare a saturated solution of Benzidine in 2 ml. glacial acetic acid.
2. Add 2 ml fresh 3% sol. of hydrogen peroxide.
3. Add equal part of urine to the above solution and formation of a blue colour indicates blood or haemoglobin.

Examination of Urinary Sediment

Take 5 ml of urine in a centrifuge tube and centrifuge

it for 20 mts at 1000 rpm. Put a small amount of sediment on a glassslide and after placing a cover slip over it, examine under the low and high power objectives of the microscope for presence of red cells, leucocytes and casts etc.

Faecal Examination

Two important methods are as follows :

I. Direct Method

Take a small quantity of faeces and make a uniform suspension with water. Place a Cover slip on a drop of the emulsion on a glass slide and examine first under low power and then high under power objective for eggs of parasites etc.

II. Concentration Methods

(A) Floating Method

A faecal suspension is made in sugar solution because floating of the ova occurs on the top of this solution.

Procedure

Take 2 gm of faeces in a beaker and make a uniform suspension using 20 ml. of sugar solution. Remove the coarse particles by using a strainer. Pour the suspension in centrifuge tube and centrifuge at 600-1000 rpm for 15 to 20 minutes. Transfer a drop of the supernatant on a glassslide. Examine under microscope after putting a cover slip over it.

(B) Sedimentation Method

Prepare the faecal suspension of 2 gm faeces in 20 ml. water. Strain and centrifuge it at 1000 rpm for 2-3 minutes. Discard the supernatant and examine the sediment for egg or ova after putting a cover slip over it. It is quite good for trematode or operculated ova.

White Cell Count

The steps are as under :

Fresh blood is sucked upto the mark (11) in a clean white cell pipette containing a white kead. A piece of blotting paper is used to clean the tip of the pipette. The WBC diluting fluid (Turk's fluid) is sucked up to 11 mark and the pipette is shaken well to mix the blood and, then, the tip of the white cell pipette is applied to the haemocytometer at an angle of 45° and the dilution is sucked between the cover slip and ruled surface of the counting grid by the capilliary force.

A haemocytometer has two ruled platforms with a distance of 0.1 mm between the coverslip and the ruled platform. There are nine primary squares in each of these platforms. Each primary square spreads over an area of 1 sq. mm. The four corner primary squares are, further. subdivided into 16 small squares each and are used for counting of the white cells.

The central primary square possesses 25 small squares each of which is further subdivided into 16 tertiary squares. All the white cells touching the top and the left hand boundaries of the ruled square millimeters are included for the sake of counting. The sum of white cells in these 4 corner squares is multiplied by 50 (if blood is diluted 20 times) to estimate the number of white cells per cubic millimeter of the undiluted blood obtained form an indiviusal.

Red Cell Count

For calculating this value ,the oxalated blood is sucked upto 0.5 mark in the red cell pipette and its tip is cleaned with blotting paper . The diluent (Hayem's fluid) is sucked upto 101 mark and both the diluent and blood are shaken well to give a dilution factor of 1 in 200.

Fill the haemocytometer as done in case of white cell count and all the four small corners and one central square

of the counting grid are counted (i.e., 80 tertiary squares). In all, one finds 400 small tertiary squares for the red cell count in an area of 1 sq. mm.

The sum of the red cell in five small squares multiplied by 10,000 gives the red cell count per cubic millimeter of the blood, in case, the blood from the patient has been diluted 200 times in the red cell pipette.

Note: As in case of white cells, include all the cells touching the top and left hand boundaries for the sake of counting and discard all the red cells touching the rest sides of the small squares within the 16 tertiary squares.

Sedimentation rate (Westergren method)

1. Put half ml. of 3.8 per cent sodium citrate solution in the tube.
2. Collect 4.5 ml. of blood directly into the citrated solution and shake.
3. Draw this mixture into the special pippete upto the zero mark. Fix the stand.

Record the level of sedimentation value in mm.

Calculate the average sedimentation (ESR) in minutes per hour as given below:

$$\frac{\frac{\text{Level at 2 hours}}{2} + \text{Level at one hour}}{2}$$

The ESR rates are given :

Normal range :

Men :	3-7 mm in one hour.
Women :	4-11 mm in one hour.
Cattle :	0 mm in 30 min.

Horse :	2-12 mm in 10 min.
Dog :	0-6 mm in 30 min.

Haemoglobin Estimation

Sahli's Haemometer Method

It consists of following steps :

1. A fresh or citrated whole blood from the body is used.
2. The blood is drawn up to 20 cmm. mark into the special diluting Sahli's pipette.
3. The tip of the pipette is cleared and its content is discharged into a special haemoglobinometer tube containing a few drops of N/10 or 10% Hcl.
4. The Hcl treated blood is further diluted drop by drop with distilled water or N/10 Hcl till the colour matches the two standard coloured rods of the haemometer.
5. The reading of the haemoglobin value in grams per 100 ml of blood is recorded.

Seller's Staining Method for Rabies

Requirements

1. Saturated solution of basic fuchsin in acetone free methyl alcohol.
2. Saturated solution of methylene blue in acetone free methyl alcohol.
3. Working solution.

Methylene blue (in methyl alcohol) 2 parts

Basic fuchsin (in methyl alcohol) 1 part

Mix the two solutions for immediate use.

Procedure

1. Make impression smears from the hippocampus major in the brain of the suspected case. Moist films are used without fixation over flame.

2. Put the mixture of the stain over the film just for a few seconds (2-3 seconds.)
3. Wash the smear in running water. A properly stained film will show a violet colour. In case, the staining solution is not properly balanced, a trial should be made by adjusting the proportions of basic fuchin and methylene blue solution in order to obtain the violet colouration of the film.

Results

Negri bodies are stained bright red or cherry red with a halo around them and other structures are stained blue. The presence of Negri bodies is pathognomic for diagnosis of rabies. Staining of distemper inclusions is done with Shorr s3 differential stain. Lyssa bodies are small granular bodies in the neurons of animals infected with the fixed virus.

Staining of Acid Fast Organisms by Ziehl Neelsen's Method

Requirements

1. Ziehl-Neelsen carbol fachsin

   Basic fuchsin 1 gm

   Absolute alcohol 10 cc

   Aqueous solution of 100 cc

   Carbolic acid (Phenol 1 to 20)

   Dissolve the stain in alcohol and add aqueous phenol solution to it.

2. Acid alcohol (1 to 2%)

   Concentrated hydrochloric acid -2 cc

   Absolute alcohol : 98 cc

3. 1% aqueous methylene blue solution make a working dilute solution of the stock solution by adding 1 cc of 1% metheylene blue to 9 ml of distilled water for using as a counter stain.

Procedure

1. Fix the smear above the flame of the sprit lamp or gas burner.
2. Pour Z.N. carbol fuchsin on the smear and warm the slide till the stain steams. Maintain the temperature and let it cool slowly for 3 to 5 minutes.
3. Wash it in running water.
4. Decolourise the smear with acid alcohol (2%) till the thickest part remains a faint pink.
5. Wash in water to remove traces of acid alcohol.
6. Counterstin the film with working aqueous solution of methylene blue for ½ to 1 minute. Non acid fast bacteria are stained blue with methylene blue.
7. Wash with water, dry it and examine under oil immersion objective after putting a drop of cedar wood oil over the smear. Acid fast bacteria stain red with carbolfuchsin.

Staining of Bacterial Spores

1. Prepare a thin film of the spore forming bacteria.
2. Stain with steaming Z.N. carbol fuchsin.
3. Decolourise with 5% acetic acid until the film is light pink and, then , wash with water.
4. Counterstain 3 minutes with 1% methylene blue (aqueous solution).

Results

Spores stain bright red.

The Leishman's method

Place a drop of blood on a clean grease free slide. Lower another slide at an angle of 45^0 just in front of the drop and allow the drop to spread along its edge.

Drawing the blood should be done in only one direction

and only once from the starting point for a uniform film. Dry the smear in the air. Make atleast three such smears and save the slides from house flies which destroy the film by licking.

Leishman's stain

Leishman's powder 0.15 gm

Acetone free, methyl alcohol 100 ml.

Procedure

1. Cover the blood film with Leishman's stain for 1 minute.
2. Apply double quantity of freshly prepared distilled water (or buffer sol, pH 6.6) and mix by blowing with a pipette.
3. Leave the stain for 10 minutes on the film. This time limit may vary according to the quality of the stain. Overstaining is to be avoided for smooth counting of the leucocytes.
4. Wash the smear with fresh or neutral distilled water. Dry and examine under the oil immersion objective after placing a drop of cedar wood oil over the stained film. Acidic water is not used for washing the blook smears to avoid red colouration of the blood films.

Gram's Method of Staining

Reagents required

a. 1% crystal violet solution. Crystal violet 1 gm in distilled water 100 ml.

b. Gram's iodine Iodine crystals 1 gm Potassium iodide 2 gm Distilled water 300 ml.

c. 95 percent alcohol.

d. Dilute carbol fuchsin Z.N. carbol fuchsin ½ to 1 ml. Distilled water 9 ml.

Procedure

1. Fix the smear by passing glass slide (quite above) over the flame of the spirit lamp or gas burner for a few times. Avoid the burning of the smear. The opposite surface of the slide should be just tolerably hot to the touch.
2. Cover the slide for a half minute with 1% solution of crystal violet solution.
3. Pour off the stain and put Gram's iodine over it and allow it to remain for 1 to 2 minutes.
4. Wash it with alcohol (95%) till purple colour ceases to come off.
5. Wash in water and counterstain with 1% dilute carbol fuchsin (1 to 10) for ½ minute.
6. Wash in water, dry an examine under oil immersion objective after placing a drop of cedar wood oil over the smear.

Preparation of Stained Tissue Sections

It includes the steps like fixing, embedding, section cutting, staining and mounting of the stained sections. A short description of the different processing steps are given below:

1. Fixing

It is done to stop the tissue autolysis and also to obtain the hardening of the pieces of the organs. Pieces of the tissues (not more than 0.5 to 1 mm thick) are immersed for the sake of preservation in the following reagents:

(a) 10% formalin (i.e., 40% formaldehyde gas in solution). Tissues are preserved in aqueous solution of formalin (10%) for 12 to 24 hours.

(b) Absolute alcohol or methylated spirit.

The solid tissues can be preserved in absolute alcohol. To strain the glycogen in tissues, the tissues are preserved in

absolute alcohol soon after death of the animal. Contact of water with tissues washes out glycogen in the cells.

(c) Zenker's fluid

It is good for fixing the nervous tissue in 12 to 24 hours.

Zenker's fluid.

Postassium bichromate	2,5 g
Sodium sulphate	1.0 g
Mercuric chloride	5 g
Glacial acetic acid	5.0 ml

(to be added just before use)

Water	100 ml

(d) Decalcifying fluid

It can be used for preserving the bone pieces and is prepared by adding 1 part of the Nitric acid to 9 parts of the formal in solution.

(e) The tissues can be preserved in 70% alcohol or in methylated spirit for long period or till required.

2. Washing

Tissues are washed in the running tap water for 12 to 24 hours depending upon the thickness of the pieces of the organs.

Dehydration and Processing in Xylol

The fixed pieces of tissues are placed into the following solutions of alcohol to replace the water in the tissues with alcohol for approximate periods as given below:

Solution	Timing
1. 80% Alcohol	1 to 2 hours
2. 80% Alcohol	1 to 2 hours
3. 95% Alcohol	1 to 2 hours
4. 95% Alcohol	1 to 2 hours
5. Absolute alcohol or Acetone	2 to 3 hours
6. Absolute alcohol or Acetone	1 to 2 hours
7 Absolute alcohol or Acetone	1 to 2 hours
8. Benzene or chloroform	1 to 2 hours
9. Benzene or chloroform	1 to 2 hours

3. Processing in paraffin

The pieces of the tissues are given 3 changes of one hour duration each in the melted paraffin (m.p.52°c) and finally the tissues are placed in the melted paraffin at 58°c overnight.

Embed the tissues in the molten paraffin in moulds and cool rapidly. When set, cut and trim the paraffin blocks.

4. Section Cutting

Paraffin sections (5 to 6 microns thick) are cut with th microtome. Float the sections on to the slides smeared with egg albumin and the sections are, then, left in the incubator for 1 to 2 hours for attaching the sections to the glass surfaces.

5. Processing of the Paraffin Sections for Staining

Paraffin sections are treated with two changes of xylol to remove the paraffin from the sections for 1 to 2 minutes. Two changes for 1 to 2 minutes in absolute alcohol are given to the sections to remove the xylol. The sections are passed through all descending grades of alcohol (say, 95% and 70% alcohol) and are then washed in water to remove the alcohol. The washed tissue sections are, thus, ready for staining.

6. Staining

The different types of the staining solutions are used for staining cells, fungi,bacteria and spirochaetes etc. The tissues

washed with tap water are at first stained with haematoxylin solution for 5 to 10 minutes. As a routine staining procedure, it is prepared as follows :

Harris's Haematoxylin

Haematoxylin crystals	5.0 gm
Alcohol 95%	50.00 cc
Ammonium or potassium alum	100.00 gm
Distilled water	1000.00 cc
Mercuric oxide	2.5 gm

1. Use heat to dissolve haematoxylin in absolute alcohol. The alum is also dissolved in water with the application of heat. The two solutions are mixed and boiled quickly. Then, mercuric oxide is added to it. The solution is re-heated until it turns a dark purple. Then, the prepared stain is plunged into cold water in a basin for rapid cooling. After cooling, the stain is ready for use. If desired, 2 to 4 cc of glacial acetic acid may be added to 100 cc of the staining solution.
2. The stained section is rinsed in tap water and differentiated in 1% acid alcohol by giving 3 to 10 quick dips. The nuclei will be distinct in the well differentiated sections under the microscope.
3. The sections are washed in tap water for a brief period and, then, dipped in ammonia water or saturated solution of lithium carbonate until sections turn bright blue.
4. Then, wash the sections stained with Haematoxylin solution in running tap water for 10 to 20.
5. Finally, the sections are counterstained with eosin solution of the stain to complete the H & E staining method. The sections stained with haematoxylin and eosin solution are given one change in 95% alcohol and two changes in absolute alcohol. These are also given two changes in xyline and then the sections are mounted in

permount or any mounting medium. The eosin solution is prepared as follows.

1% Alcoholic eosin solution

Eosin-Y water soluble	1 gm.
Distilled water	20cc.
Dissolve and add:	
Alcohol 95%	80 cc

At first, Eosin Y is dissolved in the distilled water and then, the 95% alcohol is added to it. A drop of acetic acid may be mixed with 100 cc of eosin solution to obtain a deeper shade of staining.

Results

The nuclei are stained blue against the pink background of eosin.

NB : Good staining of the sections depends upon the quality of the stain used and the experience of an individual.

Examination of Skin Scrapings to find out Ectoparasites (mange)

Requirements

1. 10% solution of KOH (Potassium hydroxide) 10 gm
   Distilled water 100ml
2. Slides

Procedure

1. Scrap the skin against the direction of the hairs until it bleeds slightly.
2. Boil the material collected in a few ml of 10% KOH solution in a test tube until the hairs are broken up.
3. Cool and centrifuse it for about 3 mts at 1000 rpm.
4. Take a loopful of sediments on a clean side and exam-

ine it under low and high power objectives after putting a cover slip over the sediments for detecting the mange.

Examination of Skin Scrapings for Ring Worm

Requirements

1. 10% KOH solution.
2. Slides.

Procedure

1. Scrap the epidermis from the outer edge of the lesions with a sterile scalpel. Scraping must have a few hairs from the margin of the lesions and heat it for a few minutes in 10% KOH solution in a test tube and examine it after taking a loopful on a clean slide after putting a cover slip over it under low and high power objectives.

Micrometery

In order to measure a microscopic object (say, a bacterium), the process adopted is called micrometery which envisages the use of a stage micrometer, an occular micrometer and a microscope. The one hundredth division of the scale measures 1/100 (or 0.01 mm). A micron is equal to 1/1000 mm (or 0.001 mm). One small division of the stage micrometer measures 10μ or microns (i.e., 1/.001 x 0.01= 1000 x 1/100= 10μ). The objective, eye piece and the tube length of the microscope influence the millimeter value of a division of the ocular micrometer (i.e., the eye piece scale). The value of a division of the occular micrometer is determined separately for the different objectives, eye piece and tube lengths. The tube length is kept constant to avoid errors. The scale of the occular micrometer (0.1 mm in length) is divided into small divisions. The geometric relation between a division of stage and occular micrometer is 1 to 10.

Procedure

(1) Insert an ocular micrometer inside the eye-piece of the microscope keeping its tube length (usually 160 mm.) constant.

(2) Place a stage micrometer on the microscopic stage.

(3) Find out the number divisions of the ocular micrometer which are in complete coincidence or just covering the divisions of the micrometer scale.

Standardisation With 90 x Objective

50 divisions of the ocular micrometer = 5 divisions of the stage micrometer

1 division of the ocular micrometer = 5/50 = 5/50 x 10 μ = 1 μ (micron)

(4) A stained film of a bacterial culture is mounted on the stage of the microscope. The divisions on the stage of the micrometer covering a bacterium are noted and, say, two divisions are in exact coincidence with the length of an organism. So with the given objective, eye piece and tube length, the bacterium in question measures 2 μ (microns).

Note : With other two objectives (8 x or 40 x), the value of the ocular micrometer division is to be standardised, while keeping the tube length constant.

Estimation of Age of Animals

An autopsist's task is to estimate the age of the animals during postmortem examination. Determination of the age of an animal is of great importance in veterolegal cases.

All the teeth in the mouth of the animals erupt at different times which roughly guide to estimate the ages of animals. Wear of the teeth also guides in determining the age of the animals.

Longevity of cattle and buffalo is 20 years and 15 years respectively but these animals can live up to 28 years.

The longevity of some animals and birds is as follows :

Table 42. Longevity of different animal

Animals	Years	Birds	Years
Horse	20	Parrot	30
Sheep	12	Dove	15
Goat	15		
Dog	10		
Coat	12		
Dog	10		
Cat	12		
Elephant	60		
Camel	45		

The dental formulae of cattle and buffaloes are given below :

Dental formula 1/1, C/C, P/P, M/M (1= Incisor C= Canine, P= Premolar M= Molar).

Temporary dentition 0/4 0/0 3/3 0/0 = Total teeth : 20

Permanent dentition 0/4, 0/0 3/3 3/3 = Total teeth : 32

Breed, individual factors, feeding and nutritional conditions etc., affect the eruption of teeth in animals. The following table incorporates the eruption of teeth in cattle and buffaloes.

Table 43. Age groups and teeth exeption in animals

Type of teeth	*Age at erupton (months) Cattle*	*Age at eruption (Months) Buffalo*
Central incisors	24-30	30
2nd pair incisors	36	42
3rd pair incisors	48	54
Corner incisors	54-60	70-72
1st pair molars	24	
2nd pair molars	24	
3rd pair molars	36	
4th pair molars	6	
5th pair molars	18	
6th pair molars	24	

The wear and general appearance of the teeth help in the estimation of age of animals after eruption of all the permanent teeth in the mouth. The arrangement of the teeth in the jaw forms an arc or a horse shoe pattern in the adult mouth but it becomes straight with advancement in the age of the animals. Cattle do not have canine teeth and there are no incisors in the upper jaw. They have dental pad in place of upper incisors quite suitable for grazing purpose.

Some details of teeth eruption times of important domestic animals with dental formulae in age estimation are given below :

Cattle, buffalo, sheep and goat.

Deciduous Teeth

2 (Di 0/4 D/c 0/0 Dp 3/3) = 20

Permanent teeth 2 (1 0/4 C 0/0 P 3/3 M 3/3) = 32

Eruption of the deciduous teeth in cattle before birth

Di 1 : before birth

Di 2 : before birth

Di 3 : birth to one week

Di 4 : birth to 2 weeks.

Dp 1 : birth to 3 weeks.

Dp 2 : birth to 3 weeks.

Dp 3 : birth to 3 weeks.

Eruption of the permanent teeth in cattle

11 1.1/2 to 2 years

12 2 to 2.1/2 years

13 3 years

14 3.1/2 to 4 years

P1 2 to 2.1/2 years

P2 1.1/2 to 2.1/2 years

P3 2.1/2 to 3 years
Mi 5 to 6 months
M2 1 to 1.1/2 years
M3 2 to 2.1/2 years

Table 44. Appearance of Teeth at Various Ages

At birth	All the temporary incisors cut through the jaws.
At 1 month	The temporary incisors are prominent and well defined
At 1 month	The temporary molars are in wear.
At 6 month	The 4th molar will cut through the gums. No over lapping of the teeth.
At 1 Year	The temporary incisors are in wear.
At 1 Year	The fifth molar will cut through the jaw.
At 1 Year 3 months	The fifth molar is well up and in wear. The incisors show more signs of wear.
At 1 Year 9 months	Eruption of the first pair of permanent incisors. The sixth molar will cut through the gums.
At two Years	The first and 2nd permanent molars cut through the gums. The first pair of permanent incisors are well up and in full wear.
At 2.1/2 Years	All the four permanent incisors are well up and in full wear.
At 3 years	All the six permanent incisors are in full wear. The molars present a uniform ridge.
At 3.1/2 years	All the eight permanent incisors are well up in Wear.
At 3.1/2 years	The corners are obviously younger and less worn than the others.
At 4 to 5 years	The teeth are slightly worn along their cutting edges.
At 6 years	The surface of the wear reaches half way across the upper surface of the teeth. A portion of the root is exposed.
At 10 years	The greater part of the crown is lost from the teeth. A portion of the root is exposed.
At 10 to 14 years	Only stumps of the teeth remain. The teeth are widely separated from each other.
At 16 years	The teeth become close together again.

Age Estimation on the Basis of Rings in the Horns of Cattle

The rough estimate of age can be made by counting the number of rings around the base of the horn.

The first ring appears at the age of 2 years and then one ring is added annually. The age of cattle can be calculated by the following formulae :

Age of the cattle in year = The number of rings in the horn + 1.

Age Estimation in Pigs

The approximate age estimation can be done on the basis of period of teeth eruption in the pigs. Factors like breeds, individuality etc., very well affect the dentition periods. The dental formulae are as under :

Deciduous

2 (D1 3/3 DC 1/1 DP 3/3) = 28

Permanent 2(1 3/3 C 1/1 P 4/4 M 3/3) = 44

Eruption Chart of the Deciduous Teeth

Di 1	1 to 4 weeks
Di 2	6 to 12 weeks
Di 3	before birth
DC	before birth
DP 1	5 to 7 weeks
DP 2	5 to 7 weeks
DP 3	1 to 4 weeks

Eruption Chart of the Permanent Teeth

11	1st Year
12	16 to 24 months
13	8 to 10 months
C	6 to 10 months
P1	5 months
P2	5 months
P3	12 to 15 months
P4	12 to 15 months
M1	4 to 6 months
M2	8 to 12 months
M3	10 to 20 months

Appearance of the Teeth at Various Ages

Above 6 months	: The permanent incisors cut through the gums.
At 9 months	: The permanent tusks reach more than half up.
At 12 months	: The central permanent incisors are well up. Eruption of any of the first three permanent molars.

Dental formula of horse

Horse Deciduous 2(Di 3/3 DC 0/0 DP 3/3= 24

Permanent 2 (1 3/3 C 1/1 P3 - 4/3 M 3/3) = 40/42

Dental formula of dog

Deciduous : 2(Di 3/3 DC 1/1 DP 3/3) = 28

Permanent : 2(1 3/3 C 1/1 P 4/4 M 2/3) = 42

Teething

Deciduous	Horse	Dog
Di 1	day - 1 weeks	3-5 weeks
Di 2	4-6 weeks	4-5 weeks
Di 3	6-9 weeks	5-6 weeks
DC	-	3-4 weeks
DP 1	day-2 weeks	4-6 weeks
DP 2	day-2 weeks	4-6 weeks
DP 3	day-2 weeks	6-8 weeks

Permanent	Horse	Dog
1 1	2.1/2 years	2-5 months
1 2	2.1/2 years	2-5 months
1 3	4.1/2 years	4-5 months
1 4	-	-
C	3.1/2-5 years	5-6 months
P 1	5-6 months	4-5 months
P 2	2.1/2 years	5-6 months
P 3	3 years	5-6 months
P 4	4 years	4-5 months
M 1	9-12 months	5-6 months
M 2	2 years	6-7 months
M 3	3.1/2-4 years	6-7 months

Horse

7 Years 1_1 Infundiblum

6 Years 1_3 (Upper) 7 year hook

6 Years 1_2 Infundiblum disappears

9 Years 1_3 -do- -do-

10 Years $1_{3(upper)}$ -do- Galvayne's groove appears

15 Years 1_3 -do- -do- ½ way down

21 Years 1_3 -do- -do- reaches tip

21 Years 1_3 -do- -do- gradually grows out

Table 45. Normal Physiological Values and Some Reagents

Normal Range of Blood Values :

	Cow	Sheep	Goat	Horse	Pig	Dog	Poultry	Human
R.B.C (millions)	5.4-9	8.5-13.5	12.5-22.0	6.5-9.4	5.0-9.4	6.4-8.0		4-6
W.B.C (thousands)	4.5-13.0	4.0-12.0	5-13	5-11	8.6-20	6-20	20-40	5-10
Neutrophiles (percent)	30 15-55	40 20-50	36	56 50-65	39 30-50	69 60-75	31 20-40	55-70
Eosinophiles (percent)	8 1-15	6 0-15	3.5	4 1-5	4.5 1-10	5. 2-10	6 2-10	1-4
Basophiles (percent)	0.5 0-1	0.2 0-2	0	0.5 0-1	1 1-4	0.5 0-2	2.5 1-4	0-1
Lymphocytes (percent)	52 40-70	52 40-70	58	30 20-40	52 40-60	20 10-30	78 55-96	25-70
Monocytes (percent)	9 3-15	4 1-12	2	8 2-12	3 1-10	6 2-12	1 0-3	3-7
Haemoglobin (gms/100ml)	8.0-14.5	9.0-14.5	9.4-14.0	9.0-14	9.0-16.8	12.0-17.8	8.0-13.0	13-17
Sedimentation rate (E.S.R) mm	0 (30 min)	0	0	2-12 (10 min)	1-6 (30 min)	0-6 (30 min)	Men (3-7mm/hr.)	Women (0-15mm/hr)
Coagulation time(min)	6	----	----	11	3	4	----	----

Normal Values of Specific Gravity of Urine

	Range	Average
Horse	1020 to 1050	1035
Cattle	1025 to 2045	1035
Sheep	1015 to 1045	1030
Goats	1015 to 1045	1030
Dogs	1015 to 1045	1030
Cats	1020 to 1045	1030
Pigs	1010 to 1030	1015

Normal life span of red cells

Horse	140-150 days
Cattle	150 days
Sheep	70-153 days
Goats	125 days
Dogs	107-115 days
Cats	125 dats

Laboratory reagents

1.Physiological saline :

Sodium chloride : 0.85 gm

Distilled water : 100 ml

2. Buffer, pH 6.6 (for use in staining blood films)

Disodium phosphate : 3.80 gms

Monopotassium dihydrogen phosphate : 5.47 gms

Distilled water : 100 ml

Mix them together by shaking the container for making a uniform solution.

3. Leishman's stain

Dissolve 0.15 gm of Leishman's powder in 100 ml of absolute (acetone free) methyl alcohol. Take 0.15 gm of Leishman's stain in a glass mortar. Add 10 ml of methyl alcohol (acetone free). Triturate with the pestle and decant it into a glass bottle. Repeat it till the complete dissolution of the stain in the mixture. Coloured bottle protect the stain during storage. The volume of the stain is made up to 100 ml in a coloured bottle by taking more methyl alcohol.

4. Giemsa Stain

Dissolve 0.85 gm of the Giemsa stain in 100 ml of a mixture of equal parts of glycerol and acetone free methyl alcohol by shaking and keep for 4 or 5 days.

5. Seller's Stain

Stock solution

I. Methylene blue : 10 gm

Acetone free, methyl alcohol to make 1000 ml.

II. Basic fuchsin : 5 gm

Acetone free methyl alcohol to make 500 ml keep the stock solutions in blue ground glass stoppered bottles in a refrigerator.

Staining solution is made at the time of staining the fresh impression smears. Smears are made from hippocampus major of the brain in dog or cerebellum in cattle. Methylene blue solution 2 parts and basic fuchsin solution 1 part are mixed well in a test tube before use to stain Negri bodies.

6. Benedict's Qualitative Reagent for Sugar

Copper sulphate	:	17.3 gm
Sodium citrate	:	173.0 gm
Sodium carbonate	:	100 mg

Distilled water to make : 1000 ml

7. Sugar Solution for Faecal Examination

Sugar : 1280 gm

Water : 1000 ml

Phenol (as preservative) : 20 ml

8. Hayem's Solution (for total red cell count)

Mercuric chloride : 0.5 gm

Sodium sulphate : 0.5 gm

Sodium chloride : 1.0 gm

Distilled water : 200 ml

9. Hcl Solution (for haemaglobin estimation and total leucocytes count)

Conc. Hcl : 1.0 ml

Distilled water : 100 ml

10. Rothera's Reagent

Ammonium nitrate : 30 gm

Sodium nitroprusside : 2 gm

Distilled water : 80 ml

11 Neutral Buffered Formalin

40%formal dehyde : 100 cc

Distilled water : 900 cc

Sodium phosphate monofolic : 4 gm

Sodium phosphate difolic anhydrous : 6.5 gm

12. Cavony's Fluid

Absolute alcohol : 60 ml

Chloform : 30 ml

Clear

The proforma for postmortem report is given below:

Postmortem No-.....

Species .....

Colour.....Breed.....

Owner .....Sex.....

Residence.....Age.....

Weight.....

By whom sent for examination and reasons if any

Date and hour of death / sacrifice Autopsy findings

Date and hour of postmortem examination 1.

History 2.

Cadaverous changes 3.

Rigor mortis

External appearance on removal of

skin (describe contusions and wounds etc.)

Mouth and pharynx

Nasal cavities

Larynx and trachea

Oesophagus

Pleural cavity and lungs

Pericardium and heart (absolute wt..... relative wt.....%

Peritoneum

Liver (absolute wt.....relative wt.....%

Gall bladder

Spleen (absolute wt.....relative wt.....%

Stomach and intestine

Urinary organs

Kidneys (absolute wt .....relative wt.....%

Generative organs

Brain and spinal cord

Lymph glands in general

Blood or tissue smears sent for microscopical examination

Diagnosis on the basis of above examination

Remarks (state if viscera and tissue specimens etc. sent for chemical, histopathological and parasitological examinations)

Microscopical examinationof blood etc.

Results of chemical, bacteriological parasitological and histopathological examinations.....

Place where postmortem examination was done .....

Dated :

Signature of the autopsist

Designation :

Fixation

When neoplastic lesions are suspected in the organs of an organism during postmortem examination, small pieces of tissues (sbout 2 cm.cubed) are removed and placed in fixatives like formalin saline or Zenker's solution which is 10 to 15 times more in volume than the total volume of the tissue pieces selected with representative lesions for histopathological examination. Fixatives preserve tissue elements as they were visible during life and also prevent further autolytic changes in them.

The following are 4 important fixatives :

1. 10% formalin solution

Formaldehyde 40% solution	: 10 cc
Water	: 90 cc

2. Formalin saline solution

Formaldehyde 40% solution	: 10 cc
Sodium chloride	: 0.9 gm
Water	: 90 cc

3. Even absolute alcohol is used for fixation of tissues. What kind of fixative is to be choosen for histopathology depends upon a particular process to be adopted for staining the tissues.

4. Romahny's fluid

Formalin (40%)	:	120 ml
Pyridine	:	10 ml

Nicotin (50% solution in water) crude 10 ml.

$Na_2 S_2 O_4$	:	20 gms
Water add	:	1000 ml.

Note : Air should not be allowed to come in contact with the fluid.

Wash the tissues to be fixed in saline water. The fluid can be filtered and reused. It protects the live colour of the lesions in tissues preserved, making it quite good for demonstration.

Postmortem Lesions

How to write postmortem lesions in the dead animals.

While describing the pathological lesions in the dead animals, the abnormalities (i.e., diviations from normalcy) are described in view of certain features like size, shape, colour, consistency, appearance of the cut surface of the

organs and lesions in the organs are exactly noted in view of the anatomical locations. One should take care of separating the interpretations from the observations of the lesions. The terms like nutmeg appearance and chronic passive congestion in livers are observation and interpretation respectively. It is better for students to note only observations during postmortem examination of dead animals.

Below are some clinical signs and gross lesions and the corresponding diagnoses in some important pathological states :

Table 46. Diagnosis and related observations in different diseases.

Diagnoses or Interpretations	Observations on Organs in the Dead Bodies
1. Necrosis	Grey or white areas or spots with some what firm consistency in an organ (say, liver). A necrosed tissue may be red to the naked eye due to infiltration with blood. Such tissues may be liquefied like water as exemplified by pus in an organ or skin (e.g. abscess).
2. Fatty Changes	Grey or yellow discolouration in the affected organs which may be also larger than normal. Loss of strength or toughness can be marked in the organs. The friability is also noticed in such organs and one can easily thrust the fingers into such tissues. Fat droplets are seen on the edges of the cutting instruments like knives.
3. Amyloid infiltration	The organs like livers in the affected animals may be larger, paler and firmer than normal. The organs or parts affected with anyloid infiltration may be white or opaque with waxy cut surfaces. The amyloid material in the tissues stains mohogony brown following application of a solution of iodine on the cut surfaces.
4. Mucoid degeneration (Serous Atrophy of Fat)	(I) Presence in excess of the slimy grayish white slippery material on the mucous membrane (II) The affected tissue becomes translucent and watery beneath the serosal covering of the tissues or organs. Mucoid degeneration in the tissue converts it into a structure resembling Wharton's jelly or watery fat or adipose tissue. Usually seen in the coronary groove of heart of cattle suffering from chronic diseases or parasitic infections (bottle jaw).

Diagnoses or Interpretations	Observations on Organs in the Dead Bodies
5. Pathological calcification	The calcium salts removed from the tissues are white or grey, irregular and sometimes honeycombed. When the affected tissue is sliced with knife, a gritty sound is heard. When touched, the feel of the cut surface is also gritty.
6. Pathological ossification	1. Pieces of the bone in the nonosseous tissue like lungs, legs of turkeys (tendon) and lateral cartilages in the horses.
7. Atrophy	Organs are smaller than normal as found by weighing or measurement and comparing with the normal.
8. Hypertrophy	Organs are larger than normal in the thickness or size. Increase is also seen in their weights.
9. Neoplasia	An enlargement or swelling anywhere in the body. Swelling may not be detectable in some cases (occult neoplasms). Presence of thickness is seen in the intestinal wall. There may be bulging of the organ or part involved in the body. The cut surfaces of the swelling may be grey or shining or red like currant jelly and may be completely black in colour as seen in melanoma.
10. Thrombus	Friable growth (dull in colour) with roughened or stringy surface in the blood vessels. Such mass may be shining and red like currant jelly and may completely block a vessel. An attachment of mass of clot with the intimal lining of the vessel can be marked. An attempt to dislodge the thrombotic mass may fragment it into pieces, tearing also the underlying intimal layer.
11. Emboli	Pieces of tissues (red or grayish masses), fragments of parasites, air or even fatty material or pieces of the tumour are seen as blockings in the vessels.
12. Hyperaemia /congestion	The affected part may be red and swollen and heavier than normal. Bluish red swollen areas in the organs. When cut, the blood escapes from such incised tissues. When there is rush of arterial blood to a part, the part may have bright red colour imparted by the accumulated blood.
13. Oedema	When a part is swollen (e.g., ventral wall of the abdomen), the affected part may be thick , firm or doughy due to accumulation of fluid in the cells or intercellular spaces. It can pit on pressure causing pits in the skin. When cut, a pale yellowish fluid escapes or drips from the cut surfaces. During life, such parts in the body are cold, puffy and swollen.

Diagnoses or Interpretations	Observations on Organs in the Dead Bodies
14. Shock	Severest state of depression, weakness, subnormal temperature, lack of warmth on the body surface, fall in blood pressure, shallow and irregular respiration and rapid or irregular pulse in the living animal. At autopsy, excessive serum in the bruised tissues, pulmonary oedema, excess of blood in the dilated blood vessels of the internal organs (e.g., abdominal organs like liver and intestines etc.) and haemorrhages in the intestines, ischaemic mucosae and ischaemic heart or brain following extensive haemorrhage from stabbing or gun-shot wounds are some important changes of haemorrhagic shock.
15. Tissues in acute inflammation	Cardinal signs of inflammation i.e., redness (rubor) swelling (tumour), heat (colour), pain (dolor) and loss of function are seen in the affected part during life. When the parts showing changes are incised or examined postmortem, an exudate (serous, fibrinous, mucoid, purulent, mucopurulent or haemorrhagic) escapes from the cut surface. The layer of fibrin on a mucous membrane may form a pseudomembrane or diphtheritic membrane. The pseudomembrane is detachable whereas a diphtheritic membrane is firmly attached or adherent to the underlying tissue.
16. Tissues in chronic inflammation	Granulomas, nodules or granules are seen in the tissues. Granulomas can be found in TB, aspergillosis, brucellosis and colibacillois (in chickens). In chronic inflammation, granulation tissue is also formed as seen in the walls of tissues, abscesses or sinuses. Affected tissues have a rough granular surface and may be hard, tough, white, oedematous (swollen) and congested (reddened).
17. Abscess	A collection of pus in the tissue with or without a wall or capsule. The pus may be a viscous cream coloured fluid with obnoxious smell. The pus may be red due to blood or blue green due to pigments.
18. Pulmonary congestion	Lungs are swollen and reddened. The cut pieces do not sink in water. Presence of blood on the cut surfaces.

Diagnoses or Interpretations	Observations on Organs in the Dead Bodies
19. Pneumonia	Swollen lungs of liver like consistency and also red in colour. The cut pieces sink in water as seen on testing durng autopsy and present a dry or somewhat bulging surface. The pneumonic tissue is slightly raised above the level of the surrounding normal tissue. Droplets of exudate on the cut surfaces of the lungs.
20. Pulmonary collapse or Atelectosis	Portions of lungs reduced in size and depressed below the level of the surrounding normal parenchyma with red colouration and firm in consistency. The cut pieces sink in water and no blood flows on surfaces of the incised slices of lungs, thus, differentiating it from pulmonary congestion.
21. Pulmonary emphysema	Pale bloodless areas in the lungs. Marking of the ribs present on the pleural surface. The lungs pit on pressure and also do not collapse after the thorax has been opened. Somewhat raised pale areas are found close to the red and sunken areas (collapse) in the lungs affected with bronchopneumonia.
22. Nutmeg liver	Lobules of the livers showing congested central areas surrounded by pale or yellowish peripheral areas. A mottled appearance of the liver is effected as a resulting change.
23. Acute cardiac failure	Right ventricle severely dilated with formation of a groove between the left and right ventricle and the line from the atrioventricular level to the apex is not a straight one. The right ventricular wall is flexible or yielding in nature.
24. Congestive heart failure (chronic)	The lungs are firm in consistency and possess brownish discolouration due to chronic passive congestion. The livers are mottled (nutmeg liver). The wall of the left ventricle is thicker than normal (i.e. hypertrophied). Mitral stenosis or incompetence is present. Congestion in the spleen, kidneys and intestines.
25. Congestive heart failure (acute)	The vena cava and large veins of the abdominal and thoracic cavities are large dark and also filled with blood. The vessels in the mesentery are prominent and dark. The liver and lungs are swollen, reddened and the blood escapes from the cut surfaces of these organs. Degenaerative lesions in the heart or ventricles.

Diagnoses or Interpretations	Observations on Organs in the Dead Bodies
26. Hepatic infarction	Red areas seen in the liver due to blockage of a branch of the portal vein from embolism or thrombosis. In infarction, necrotic changes occur but in the hepatic infarcts, necrosis is prevented by branches of hepatic artery supplying good oxygenated blood.
27. Pyaemic nephritis	The kidneys are larger than normal, congested (reddened) and show petechiae. Several abscesses are seen subcapsularly but abscesses in the intertubular capillaries are elongated in shape. The renal capsule easily strips in such kidneys.
28. Pyelonephritis	Greyish white patches subcapsulary against the normal reddish brown kidneys. Dirty grayish brown blood tinged material present in the dilated calices or pelvis. Grayish white streaks seen on the cut surfaces of kidneys. In short, the kidney is mottled and capsules may be attached with renal parenchyma.
29. Chronic nephritis	The kidneys bear granular surface and are contracted and smaller than normal. Capsule is removed with fragments of the kidneys. A narrow cortex reduced to a mere rind is seen in chronic nephritis. Small cysts cortaining a little fluid are visible owing to dilation of the uriniferous tubules.
30. Goitre	An enlarged or somewhat firm thyroid gland.
31. Osteomalacia	Lameness and fracture of the ribs or jawa. Maxillae, frontal and nasal bones are swollen. Reduced intermandibular space. Reduction in the nasal cavity with difficult breathing. Tumour like swellings in the maxillae.
32. Rickets	Epiphyseal cartilage is irregular and thickened. Extremities of the bones and costochondral junctions enlarged in size. Bending of the bones and pendulous pot belly are seen. The bones are too soft to be cut with a knife. Birds show crooked or twisted sternum.
33. Hydronephrosis	When the kidneys are incised into two halves, several cysts (multilocular) of different sizes are seen on the cut surfaces. Medulla almost disappears and the pelvis is severely dialated.
34. Hepatic cirrhosis	There is a firm liver which is difficult to be cut into pieces. Hepatic surface is granular or nodular. The cut surfaces may be yellowish brown or green in colour. Bile ducts are thickened, hardened and appear as rigid tubes.

Diagnoses or Interpretations	Observations on Organs in the Dead Bodies
35. Postmortem imbibition	It is a sign of postmortem decomposition. Red areas of uniform colour with no elevated surfaces are seen. This occurs due to a pigment (haemoglobin) which is released from the red cells. These pigments stain the tissues along the margins of the congested veins. Such areas may be red or pink.

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