STANDARD VETERINARY TREATMENT GUIDELINES FOR ETHIOPIA

2020
SECOND EDITION

Veterinary Drug and Animal Feed Administration and Control Authority

Food and Agriculture Organization of the United Nations

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Picture on the cover page is Dr. Hanna Zewdu, a veterinary doctor treating a horse at the Teaching Hospital Addis Ababa University, Bishoftu.

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EDITORS AND CONTRIBUTORS FOR THE 2ND EDITION SVTG

Editors
Prof. Alemayehu Lemma (Chairman)
Dr. Solomon Kebede (Coordinator)
Dr. Kassaw Amsalu (Member)
Dr. Joseph Othieno (Scientific Editor)

Focal person from IFC/WBG
Mr. Isaac Njoro Thendiu

Focal person from FAO
Mr. Tenaw Andualem

Contributors: Members of the review committee

Name	Profession	Organization
Dr. Abebe Fromsa	DVM, MVSc, PhD Candidate,	AAU, ALIPB
Prof. Alemayehu Lemma	DVM, PhD	AAU, CVMA
Dr. Anwar Nuru	DVM, MSc, PhD, Assoc. Prof	UoG, CVM
Dr. Bewket Siraw	DVM, MVSc	AKLDP, Tuft University
Dr. Solomon kebede	DVM, MVSc	VDFACA (Coordinator)
Dr. Tadesse Eguale	DVM, PhD, Assoc. Prof	AAU, ALIPB
Mr. Tenaw Andualem	Pharmacist / Pharmacologist	FAO
Dr. Tiruneh Zerihun	DVM, Private	Vet drug importer

Members of the technical working group

Name	Profession	Organization
Dr. Kassaw Amssalu	DVM, MVSc	MOA
Dr. Biniam Tadesse	DVM, MVSc	NAHDIC
Dr. Asmamaw Kasaye	DVM	Donkey sanctuary
Dr. Dawit Ketema	DVM	Pet animal veterinarian
Dr. Solomon Tarekegn	DVM	Poultry farm practitioner
Dr. Abera Gemeda	DVM	VDFACA (Coordinator)
Dr Belayneh Getachew	DVM, PhD	National Veterinary Institute

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We acknowledge the efforts of the editors who steered this process under the chairmanship of Prof. Alemayehu Lemma from Addis Ababa University (AAU), assisted by Dr. Solomon Kebede of Veterinary Drugs and Animal Feed Administration and Control Authority (VDFACA) and Dr. Kassaw Amsalu of Ministry of Agriculture. We are greatly indebted to the contributors who also served in the review committee namely: Dr. Abebe Fromsa (AAU), Dr. Anwar Nuru (UoG), Dr. Bewket Siraw (Tuft University), Prof. Tadesse Eguale (AAU), Dr. Tirune Zerihun (Private Vet.). We appreciate the practical experience brought in by members of the Technical Working Group who included Dr. Biniam Tadesse (NAHDIC), Dr. Asmamaw Kasaye (Donkey Sanctuary), Dr. Dawit Ketema (Small Animal practitioner), Dr. Solomon Tarekegn (poultry expert), Dr. Abera Gemeda (VDFACA) and Dr. Belayneh Getachew (National Veterinary Institute).

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PREFACE

The objective of this Standard Veterinary Treatment Guideline (SVTG) is to provide information on epidemiology, clinical symptoms, diagnosis and treatment of terrestrial and aquatic animals namely cattle, equine, pigs, shoats, poultry, honeybee and fish. In writing this manual, we have made utmost effort to review available literature both locally and internationally. We have compiled knowledge which has been acquired during long experience in clinical practice, diagnostic and research laboratories, teaching and in veterinary public health.

This SVTG is an update from a previous version (SVTG for Veterinary Clinics; First Edition, 2006). The SVTG is organized as a compendium so that clinicians or those involved in the veterinary profession will use it as a quick reference. Despite the fact that many diseases affect different species of animals, an account of each disease has been described separately. However, a full account of the disease in each animal species is given only when there is a significant difference in the epidemiology or clinical symptoms. Although there is no sufficient data on the status of fish diseases in Ethiopia, a comprehensive review of diseases affecting fresh water fish and aquaculture is included.

The SVTG document is structured into ten sections referring to diseases of each species of animals (Sections I – IX) and Poisoning in animal (Section X). In as much as possible, the most important descriptions on the epidemiology, clinical symptoms, diagnosis and treatment are included. However, descriptions have been truncated and made concise compared to the first edition. Much emphasis is given to the treatment part to select the most effective and economical drugs, that are acceptable to practicing veterinarians and farmers. The types of drugs included are based on the updated Ethiopian Veterinary Drug List – EVDL (Second Edition, 2018) issued by VDFACA. However, some drugs which were found essential but not found in the EVDL were also included. Drugs are listed under "Treatment and Prevention" for each disease and are arranged in numerical order based on their priority of choice (e.g. number one on the list means it is the first drug of choice). Combinations of drugs are separated by **"plus"** while alternative usages are presented as **"or"** on the list.

Since prevention in most instances is more economical than clinical treatment of sick animals; appropriate preventive measures are briefly described in each section. In addition, a summary of vaccines and their management, normal body parameters of domestic animals and anesthetics, analgesics and tranquilizers are described in a separate section to make the guideline more comprehensive for clinicians and professionals involved in the animal health practice.

FOREWORD

The Ethiopian Veterinary Drug and Feed Administration and Control Authority (VDFACA) has developed and updated a number of guidelines and directives since its establishment in 2011 GC. The guidelines have greatly strengthened the regulatory capacity of the Authority and promoted rational use of veterinary medicines in general and antimicrobials in particular. This has helped in the containment of antimicrobials resistance and strengthened animal health services and systems. One of the most important guidelines is the Standard Veterinary Treatment Guidelines (SVTG) which were first developed in 1998 GC.

Through financial and technical assistance from International Finance Corporation -IFC/World Bank Group and the Food and Agricultural Organization (FAO) of the United Nations, VDFACA in collaboration with stakeholders, has recently updated the STVG. The guidelines will help animal health professionals to administer veterinary drugs and vaccines rationally and enhance the quality of veterinary clinical services. This will contribute to a decrease in economic losses and public health hazards due to livestock diseases. Professionals who are handling and using veterinary drugs and vaccines are encouraged to read and utilize this guidelines for further information and knowledge.

Finally, I wish to express my gratitude to the development partners and professional experts who have directly or indirectly supported the updating of this guidelines. Moreover, I want to encourage all practicing veterinary professionals to pay attention to prevention of diseases and rational utilization of veterinary drugs to minimize development of drug resistance which negatively affect animal health, productivity and food safety. I also call upon all animal health professionals and other interested parties to forward their inputs for continuous development and revision of the guideline.

Terzu Daya Degaga (Dr.)
Director General,
Veterinary Drugs and Animal Feed Administration and Control Authority

GENERAL GUIDANCE

Rational Use of Drugs

The Standard Veterinary Treatment Guidelines (SVTG) is important to rationalize treatment of animal diseases, utilization of drugs properly and protection of the public from exposure to unnecessary drug residues. Drugs should be used only when required and in the right dosage and combination. Improper usage of drugs may result in ineffective treatment, wastage of resources and may harm the patient. In using veterinary drugs, certain steps should be followed before deciding on the procedures to be carried out. One way of promoting such a practice is developing SVTG.

Rational approach to therapeutics requires careful evaluation of the health problem in each species of animal and selection of appropriate therapeutic strategies. Proper diagnosis of animal diseases requires extensive discussions with owners, clinical examinations and empirical confirmation by appropriate laboratory procedures. The efficacy of treatment largely depend on correct diagnosis. Whenever alternatives exist, priority should be given to non-pharmacological treatment than chemical treatment. The issue of microbial resistance is getting more attention where increasing number of diseases are becoming untreatable because of drug resistance. In veterinary medicine, preventive measures are better than treatment. Thus possible preventive measures should be given attention to minimize the spread of animal diseases and reduce antimicrobial resistance.

Selection of treatment requires cost/benefit analysis particularly in food animals. Uneconomical treatment should be avoided unless the animals are genetically valuable deserving priority for conservation or have special aesthetic attachment with the owner (e.g. pets like dogs and cats). Apart from the cost of a particular drug, its efficacy and safety with minimal adverse effects and residues in food animals should be given due attention. A drug of choice depends on individual patient and disease condition; whenever written it should clearly indicate the species of animal, age and sometimes breed, the dose of the drug and the formulations available locally, frequency of use and the duration of treatment. In food animals, considerations must be given to the withdrawal period of drugs in case an emergency slaughter is recommended (included in prescription writing).

Prescription Writing

A prescription is an instruction from a prescriber to a dispenser. The prescription is the link between a prescriber, drug dispenser and patient. A good prescription should provide relevant information, instructions and warnings to the patient. Currently, veterinary doctors and other animal health professionals can prescribe to veterinary drugs dispensary and pharmacies. Prescriptions should be clear, legible and indicate precisely what should be given. It should include the following:

- Name, address, telephone of prescriber
- Date of the prescription
- Name, form and strength of the drug. Generic name of the drug should be used
- Formulation of the drug (e.g. tablet, oral solutions, feed additive or ointment) should also be stated
- Strength of the drug should be stated in standard units
- Dose, route of administration and frequency should be clear and explicit. Use of vague phrases such as "take as directed" or "take as before" should be avoided
- Quantity of the medical product to be applied should be stated. Alternatively, the length of treatment course should be stated
- Signature or initials of prescriber.

Adherence (Compliance) to Drug Treatment

Once drugs are dispensed according to the prescription, the owners or those who administer it should stick to the dosage and frequency of treatment. Poor adherence (compliance) with the treatment plan is one of the most important reasons for treatment failure. There may be mistakes in prescriptions, which might result in errors in calculations or where different formulations have been quoted. It is not uncommon to find counterfeit veterinary drugs in Ethiopia whose active ingredient is less than the concentrations recommended in the British Pharmacopeia, World Organization for Animal Health (OIE) or other standards while the size or quantity remains the same. In such circumstances, professionals may prescribe a size or quantity of a drug whose active ingredient is low.

Non-compliance in veterinary medicine is more common than in humans and particularly over the counter drugs where livestock owners have access. The owners usually share drugs with other animal's owners in order to save money. In this case the owners should be sensitized on the hazards and disadvantages arising from such practices.

Veterinary Considerations in Drugs Use

The types of drugs used in veterinary medicine are chemically similar with those used in human medicine. However, certain conditions should be considered before deciding to use a certain drug. These include: whether treatment is economical, if there are regulatory and public health concerns, compatibility, stability and compounding process and pharmacokinetics of the active ingredient.

As there is great variation in body weight in different species of animals, setting a standard dose applicable to all is difficult. It is thus imperative that a veterinary professional with sufficient experience should be involved in prescription of drugs. On the other hand some drugs may be administered to certain species but not to others requiring good knowledge of adverse effects in particular species of animals. The type of feed is also another consideration. Drugs formulated as feed additives are preferred; but these drugs should only be given to certain species of animals or age groups if they don't adversely affect the microflora. For example, tetracycline in feed is more often prescribed to calves and not to adults.

Adverse Drug Reactions and Drug Interactions

An adverse drug reaction may be defined as any unwanted response to a drug or combination of drugs which is noxious, unintended and occurs at doses normally used for prophylaxis, diagnosis or therapy. These reactions are mainly individual based and thus closer attention should be given to drugs with reported adverse reactions. Adverse reactions may also arise from old age.

Drug interactions (drug-drug interactions) on the other hand are reactions that occur between two or more drugs when they are used to treat pathophysiologically distinct illnesses or a single illness in a patient at the same time. Interactions can occur between drugs competing for the same receptor or acting on the same physiological system. They may also occur indirectly when a drug-induced disease or a change in fluid or electrolyte balance alters the response to another drug. Interactions may also occur when one drug alters the absorption, distribution or elimination of another drug; such that the amount which reaches the site of action is increased or decreased.

When two drugs are administered to an animal simultaneously, they may either act independently of each other, or interact with each other. Interactions may either decrease or increase the action of the interacting drugs. In general, the combination of bactericidal and bacteriostatic drugs is not recommended; as it results in antagonistic effects.

Drug Residues and Withdrawal Period

By consuming animals and animal products; humans are at risk of consuming whatever chemicals the animal had ingested or was exposed to. Such chemicals include veterinary drugs, insecticides or herbicides. The residue limits and the withdrawal periods of these chemicals and drugs have been established; though controversy still surrounds this issue. Withdrawal period is the time between the application of the drug and clearance to a level of its residue limit. Thus, withdrawal period should be considered during treatment of food animals including livestock, honeybee and fish.

The use of drugs in food-producing animals has the potential to generate residues in meat, milk, eggs and honey and poses a health hazard to consumers. Factors that influence the occurrence of residues in foods of animal origin are: drug's properties and their pharmacokinetic characteristics, physicochemical or biological processes of animals and their products. The most likely reason for drug residues in foods of animal origin are due to improper drug usage and failure to observe withdrawal period. The major public health risks associated with drug residues in food are antimicrobial drug resistance, hypersensitivity reaction, carcinogenicity, mutagenicity, teratogenicity and disruption of gastrointestinal normal flora. Since there is little data available, there is a need to study the magnitude and impacts of residues and to create awareness to animal health professionals, farmers and the general public. The knowledge of pharmacokinetics, pharmacodynamics and toxicological effects of veterinary drugs to minimize the potential health hazards due to residues in foods of animal origin should be documented. Withdrawal period should be considered during treatment of food animals including livestock, honeybee and fish.

A. Prudent Use and Stewardship of Antimicrobials

Prudent use of antimicrobials will result in more rational and targeted use, thereby maximizing the therapeutic effect and minimizing the development of antimicrobial resistance (AMR). Resistance occurs when micro-organisms: bacteria, fungi, viruses and parasites evolve in ways that render medications used to cure the infection they cause ineffective. As a result of AMR, medicines that were once effective in treatments of certain diseases become less effective or not effective at all. Effects of AMR include reduced ability to successfully treat infections, increased mortality; more severe or prolonged illnesses; high production loses in agriculture and reduced household incomes and a threat to food security. AMR micro-organisms can develop in human food chains and environment and move between animals and humans by direct exposure, consumption or contact with the environment.

However, antimicrobials are often misused for therapeutic and non-therapeutic uses. About 75-90 % of antimicrobials used by humans and animals are excreted unchanged. Release of waste from health care facilities and farms and effluent from pharmaceutical plants enter into the environment all of which contribute to selection pressure and act as reservoir to AMR. These have health and economic consequences.

Globally, the bulk of antimicrobials are used in animals to treat diseases, prophylactically to prevent diseases in animals at a higher risk and controversially as growth promoters. Use of antimicrobials as growth promoters in intensive animal production systems is influenced by increasing global demand for meat, milk, fish, eggs and other foods of animal origin. In addition to their use in animal husbandry, antimicrobials used in companion animals and agriculture also contaminate the environment.

AMR is a major global threat to animal and human health. The health consequences and economic costs of AMR are estimated at 10 million human fatalities a year, an 11% decrease in livestock and production and productivity and between 2 to 3.5 % decrease in global Gross Domestic Product (GDP). Cumulatively this is estimated to reach US$ 100 trillion by 2050. However, the full impact remains hard to estimate. Before prescribing antimicrobials, it is essential to consider cross- and co-resistance, where any exposure to antimicrobials can increase the chances of AMR.
The benefits of SVTG includes;

- Provision of standardized guidance to practitioners and options
- Enables animal health providers to concentrate on making the correct diagnosis
- Promotion of high quality of care by directing practitioners to the most appropriate drugs for specific conditions
- Provision of best quality of care since patients are receiving optimal therapy
- Provision of information for forecasting and ordering according to the morbidity
- Provision information for purchase of pre-packed drugs; fixed dose combinations, and of various relevant formulations
- Will ensure patients receive optimal drug therapy
- Enable consistent and predictable treatment from all levels of providers and at all locations
- Improve the quality of health care and minimize the use of unnecessary or harmful interventions.

In order to rationalize the use of antimicrobials, the following principles should be taken into account:

- The Antimicrobial should be prescribed by a licensed veterinarian
- The continued need for antimicrobials should be reassessed on a regular basis to avoid unnecessary use
- Perioperative prophylaxis antimicrobials use should be minimized through use of aseptic techniques; if they must be used then they should be administered prior to surgery and not for more than 24 hours
- Other alternative options for controlling disease (e.g. vaccines) should be considered over antimicrobials
- As much as possible antimicrobial susceptibility tests should be used in zoonotic and commensal microorganisms and target pathogens should be established
- Antimicrobials categorized as critically important and reserve should not be used in animals since they are also used in treating life-threatening infections in humans and should only be used under strict supervision to minimize the development of resistance
- Antimicrobial metaphylaxis (mass administration of antimicrobials to a group of animals to eliminate or minimize disease outbreak) should not be used in place of good management practices
- Routine antimicrobials use for prophylaxis must be avoided and reserved for exceptional case-specific indications

- Administering antimicrobials to an entire herd or flock should be avoided whenever possible
- The cause and nature of infections in animals and range of available antimicrobial options should be taken into account when making decision regarding antimicrobial treatment
- Sick animals should be isolated and treated individually
- Narrow-spectrum antimicrobial should always be the first choice unless prior susceptibility testing has been carried out
- As much as possible use of broad-spectrum antimicrobials and antimicrobial combinations should be avoided with few exceptions of combinations
- In case of recurrent infection(s) in animals requiring antimicrobials, efforts should be made to identify and eradicate the strains of the microorganisms and changing the production conditions, animal husbandry and/or management
- Antimicrobials should not be used for treatment of self-limiting infections in immunocompetent animals.

B. Oral Administration of Antimicrobials to Groups of Animals via Feed and Drinking Water

Where possible other options of antimicrobial use to groups of animals through feed or drinking water or feed on the farm should be preferred e.g. individual treatment of the animal, administration through injection etc. However when a group or mass administration is preferred, the following should be considered:

- It should be prescribed by a veterinarian
- It should be based on evidence of bacterial infection, requiring treatment
- This route should not be used for prophylaxis
- The quantities of antimicrobials administered in feed or water should be monitored and documented on a continuous basis, especially in intensive food-animal production systems
- The instructions given in the product information and by the veterinarian must be complied with, both in terms of dosage and duration of treatment
- Where an antimicrobial is administered through feed, it is important to ensure the homogeneity of distribution of the drug so that each animal obtains the required therapeutic dose for treating the disease in accordance with the prescription
- Adequate, clean storage facilities should be available in the farm to ensure proper storage of the antimicrobial.

C. Extra label use of veterinary drugs in food producing animals

- Extra or off-label use should be limited to the bare minimum and to exceptional occasions where no other authorized treatment options are available
- The off-label use of antimicrobials for non-food-producing animals should be avoided and strictly limited to very exceptional cases
- Extra label use is only allowed if there is no approved animal drug that is labeled for such use, or that contains the same active ingredient in the required dosage form and concentration
- Use of a human drug, or an animal drug that is only approved for use in nonfood-producing animals, has further restrictions
- When using drugs in an extra label, requires extended withdrawal intervals be established using appropriate scientific information.

The extra label use of certain drugs is prohibited in food animals. The following list are examples:

- **Prohibited therapy in food animals:** chloramphenicol, clenbuterol, diethylstilbestrol, dimetridazole, ipronidazole, other nitromidazoles, furazolidone, nitrofurazone, glycopeptides and fluoroquinolones
- Prohibited therapy in lactating dairy cows: any sulfonamide except for approved uses of sulfadimethoxine, sulfabromethazine and sulfaethoxypyridazine
- **Prohibited therapy in chickens, turkeys and ducks:** adamantane and neuraminidase inhibitor classes of drugs that are approved for treating or preventing influenza
- Prohibited cephalosporin (excluding cephapirin) use in cattle, swine, chickens and turkeys
- Using cephalosporin drugs at unapproved dose levels, frequencies, durations or routes of administration is prohibited;
- Using cephalosporin drugs in cattle, swine, chickens or turkeys that are not approved for use in that species (e.g. cephaloporin drugs intended for humans or companion animals) or production class;
- Using cephalosporin drugs for disease prevention.

D. Vaccines Use and Storage

Vaccines are products intended to initiate protective immune responses and prepare the immune system to fight future infections from disease-causing agents. Vaccines stimulate the immune system to produce antibodies that identify and destroy disease-causing organisms that enter the body. Vaccines provide immunity against one or several diseases that can lessen the severity or prevent certain diseases altogether.

The goals of veterinary vaccines is to improve the health and welfare of companion animals, increase production of livestock in a cost-effective manner and increase safe food supplies. They prevent animal-to-human disease transmission from both domestic animals and wild animals. Successful veterinary vaccines have been produced against viral, bacterial, protozoal and multicellular pathogens, which in many ways have led the field in the application and adaptation of novel technologies.

The success of efforts against vaccine-preventable diseases is attributable in part to proper storage and handling of vaccines. Vaccines exposed to temperatures outside the recommended ranges can have reduced potency and protection. Storage and handling errors can cost a lot in wasted vaccines and revaccinations. Errors can also result in the loss of animal owners' confidence when repeat doses are required. It is better to not vaccinate than to administer a dose of vaccine that has been mishandled. Vaccines must be stored properly from the time they are manufactured until they are administered. Cold chain refers to a system of storing and transporting vaccines at the recommended temperatures from the point of manufacture to the point of use (administration).

A proper cold chain therefore is a temperature-controlled supply chain that includes all equipment and procedures used in the transportation, storage and handling of vaccines from the time of manufacturer to administration of the vaccine. The cold chain begins with the cold storage unit at the manufacturing plant, extends through transport of vaccines to the distributor and delivery to and storage at the provider facility and ends with administration of vaccine to the patient.

When the cold chain fails for example when there is exposure to excess heat, cold or light at any step in the cold chain can damage vaccines, resulting in loss of vaccine viability and potency; these are irreversible. Every time vaccines are exposed to improper conditions, potency is reduced further. Eventually, if the cold chain is not properly maintained, potency will be lost completely and vaccines will be useless.

Most live vaccines tolerate freezing temperatures, but deteriorate rapidly after they are removed from storage. Inactivated vaccines can be damaged by exposure to temperature fluctuations (for example: extreme heat or freezing temperatures). Potency can be adversely affected if vaccines are left out too long or exposed to multiple temperature excursions (out-of-range temperatures) that can have a cumulative negative effect. It is a good idea to post a sign on the front of the storage unit(s) indicating which vaccines should be stored in the freezer and which should be stored in the refrigerator. A freezer is device or a part of a device that maintains a temperature below the freezing point of water (below -20°c to -18°C).

- Most live vaccines such as PPR, LSD, Sheep and goat pox, AHS, Camel Pox, CBPP, Newcastle, and Fowl pox) should be stored between -15°C and -20°C.
- Anthrax and IBD can be stored either in the freezer or the refrigerator. However, storing these vaccines in the freezer will confer longer shelf-life.
- Refrigerator is the device that maintains a temperature a few degrees above the freezing
- point of water (0° to 4°C).
- All inactivated vaccines such as Blackleg, Bovine pasteurella, Ovine pasteurella, Fowl Cholera, CCPP, Inactivated Newcastle), foot and mouth disease, rabies and diluents require refrigerator storage temperatures between 2°C and 8°C, with a desired average temperature of 5°C.
- Before reconstitution with a diluent, all vaccines can be stored at refrigerator temperature between 2°C and 8°C for up to 72 hours.
- It is necessary to read the manufacturers' instructions about the specific storage temperatures of the vaccine. All personnel who handle, deliver, accept, administer or have access to vaccines should be familiar with the storage and handling procedures. These procedures should be available in writing as a reference for all staff members. It is highly recommended to refer and follow catalogue product guide.

INTRODUCTION

Modern veterinary service in Ethiopia started in early nineteen hundreds with the aim of improving productivity and trade of animals and animal products as well as protecting the public from zoonotic diseases. The current animal health services is organized at federal and regional levels, each acting independently and in cooperation. The main functions of the Federal Veterinary Services (FVS) are: Formulation of polices and strategies, collection and collation of animal health data for distribution to those who need it, coordination of disease surveillance and outbreak investigation, formulation of projects to collect baseline data on animal diseases and involvement in the control of transboundary diseases. In addition to this FVS is also responsible for enforcement of animal health regulations, meat inspection at export slaughterhouses, issuance of certificates for export purpose, preparation of work plans and budgets for its activities and provision of technical inputs to the regional governments.

The functions of the Regional Veterinary Services (RVS) office are; provision of clinical services, annual vaccinations, collection of animal health data and reporting to the FVS. The RVS is also involved in infrastructure development, training animal health technicians and Community Animals Health Workers (CAHW), performing laboratory diagnostic activities, procurement of veterinary drugs, licensing private practices and monitoring veterinary public health services including inspection of meat and other foods of animal origin.

Veterinary manpower in the public sector: Qualified veterinarians and animal health assistants work in the federal, regional and state veterinary establishments, the federal and regional state veterinary laboratories, the research institutes and the universities and other training institutions **(Table 1 below).**

Table 1: Summary of veterinary professionals in Ethiopia in 2017

Category of veterinary staff Sector	No. of vets employed at federal level(DVM And BVS or BVLT)	No. of AHAs employed at federal level	No. of vets employed at regional level(DVM And BVS or BVLT)	No. of AHAs employed at regional level
Veterinary services	315	102	2391	11572
Research	18	142	11	-
Laboratory	62	5	122	37
Faculties/Colleges	363	36	-	-
Total	**696**	**285**	**2524**	**11,609**

Laboratory capacity: Veterinary diagnostics in Ethiopia is a function of the federal (NAHDIC) and 15 regional veterinary laboratories following the segregation of mandates at the two hierarchies of governance. The federal government is mandated with the control and prevention of trans-boundary diseases and quarantine operations. The regional states are mandated with all the remaining diseases of both economic and public health importance. (Ethiopian Animal Health Year book, 2015).

The NAHDIC was established in 1995, as the national referral and reference laboratory of Ethiopia and works on animal disease surveillance, outbreak investigation, testing of animals to be exported or imported, research and capacity building. NAHDIC implements a quality management system ISO/IEC 17025/2005 that enables the centre to generate quality diagnostic results and information on animal health. NAHDIC has strong linkages with national, sub-regional and international organizations, OIE reference laboratories and universities working on animal health. NAHDIC is also engaged in a three year program of OIE FMD twinning with the World Reference Laboratory, the Pirbright Institute (WRLFMD-TPI).

It consists of ten laboratories that actively function in various disciplines. Four of the laboratories are engaged in the field of microbiology (viral and bacterial serology, mycology, bacteriology and virus isolation), two in the field of parasitology (protozoology and acarology-entomology-helminthology), one pathology laboratory with autopsy facilities, one laboratory for molecular diagnosis and one biosecurity level three laboratory (BSL-3) for the diagnosis of zoonotic diseases.NAHDIC has also experimental and laboratory animal facilities to conduct animal health experiments.

National surveillance system: The primary objective of surveillance is to understand epidemiological situation of diseases in the country and to identify the level of threat. Surveillance thus assist in preparing for control and eradication. Surveillance systems also contribute to the identification of priority diseases. Currently both active and passive surveillance systems are being implemented nationally.

Animal health information system plays an important role in surveillance and provision of information for economic assessment of diseases and fulfillment of international reporting obligations. The passive surveillance system mainly depends on a paper based system from woreda to the regional veterinary laboratory covering the respective woreda. The monthly disease outbreak reports received from woreda level are entered in a web-based Disease Outbreak and Vaccination Reporting (DOVAR-II) system. Syndrome surveillance is practiced in some selected areas (as a pilot) using a mobile phone based animal disease notification system known as ADNIS. This helps in immediate notification and early detection of some selected animal diseases.

Active surveillance is an essential tool for early detection of diseases and introduction of exotic diseases. It also provides information for declaring a country or a zone as disease free. Annual sero-surveillance is being conducted for top priority diseases and trade sensitive diseases.

Regarding introduction of exotic diseases, mainly for the assessment of ECF, RVF and Highly Pathogenic Avian Influenza annual risk based sero-surveillance is conducted around border areas of the country. Risk assessment is done for RVF by studying the serological status of live animals to be slaughtered using ELISA. Ten percent of every batch of animals to be slaughtered for meat export are tested. For other endemic diseases annual surveillance is conducted to know their status while gathering information for national control programmes. In order to detect areas with active disease transmission, Participatory Disease Surveillance (PDS) is conducted.

The Ethiopian Veterinary Drugs and Animal Feed Administration and Control Authority (VDFACA) was established in 2013 as a government institution with a mandate to oversee the standards for safety, efficiency and quality of veterinary drugs used in Ethiopia. It is primarily responsible for preparing a national veterinary drug list, evaluating and registering veterinary products, formulation of regulations and legislations for their use and application. A number of guidelines, regulations and legislative documents have been published in Ethiopia on animal diseases control, meat inspection, drug importation and standardized use of veterinary drugs.

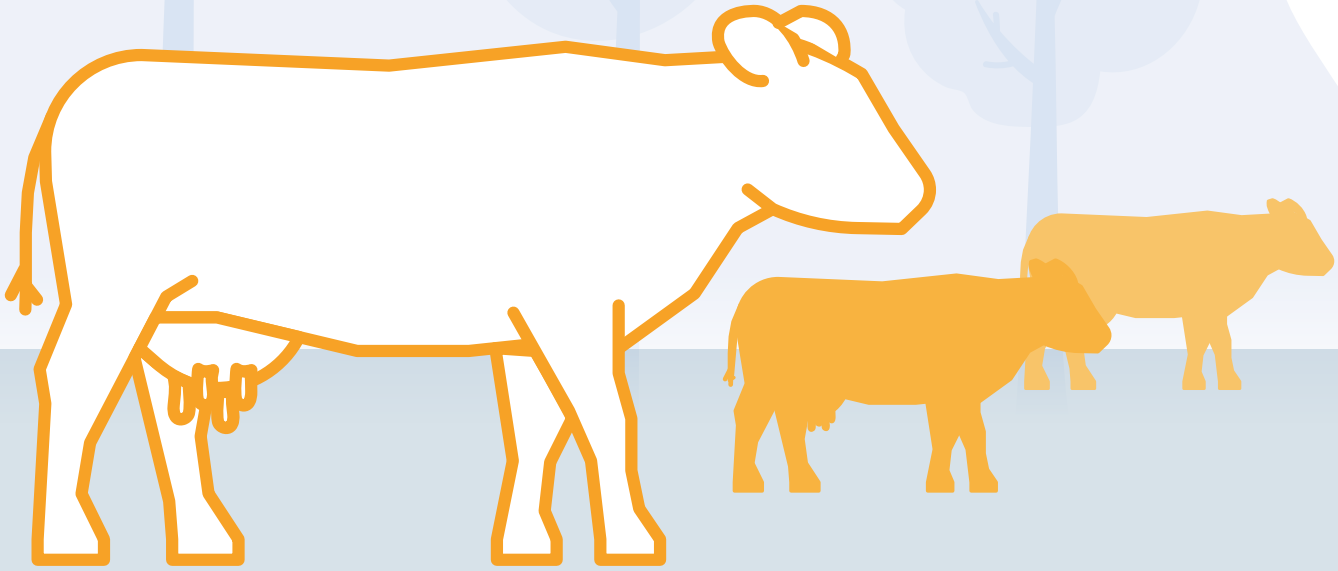
However, the century long experience on modern animal health service has not significantly improved their implementation for various reasons. The main reason is accessibility of such documents to the end users. The lack of updated standardized treatment guidelines at all levels of the veterinary practices has resulted in irrational use of drugs. It has become a common practice for veterinary drug importers to trade in drugs primarily on the basis of lowest price quotations and consequently practitioners use drugs that are locally available rather than prescribing effective drugs. Others, especially midlevel animal health professionals prescribe a combination of drugs that probably have antagonistic action and do not consider withdrawal periods of drugs in food animals, a precaution that should be taken to protect the public from drug residues. Prescriptions are mostly based on tentative diagnosis from clinical signs rather than using a laboratory based confirmatory diagnosis. This leads to incorrect prescription which is the common cause of drug resistance. Therefore VDFACA which is mandated to regulate and manage the overall use of veterinary drugs in Ethiopia, considers improving the accessibility of an updated version of the Standard Veterinary Treatment Guideline (SVTG) a priority to circumvent such irrational use of drugs in Ethiopia.

This SVTG comprises guides to general and special prescription of veterinary drugs for common diseases and syndromes in different species of livestock, pets, honeybees and fish. It also consist relevant annexes on drug information, normal physiological values, anesthetics, tranquilizers and sedatives used in animals.

Diseases are classified based on causative agents and body systems affected as: infectious diseases, non-infectious diseases, reproductive diseases, respiratory diseases and syndromes and chemicals and plant poisoning. Accordingly, description of each disease, epidemiology, clinical symptoms (plus lesions in poultry and fish) and diagnosis, treatment and preventive measures are presented. The descriptions on each section were relatively detailed so that professionals will use it as a compendium for diagnosis and treatment of animal diseases. Drugs are listed according to their priority of use and important information is included for each drug on side effects and contraindications, drug interactions, drug formulations and withdrawal periods (particularly for food animals) in the Annexes.

All the drugs listed in this STVG are those listed in the updated National Veterinary Drug List (NVDL). They are either already registered by VDFACA or awaiting registration to be available on Ethiopian market. It is hoped that this STVG will be updated whenever change in the scientific knowledge demands a modification or improvement. Comments on the general guideline by veterinary professionals, veterinary pharmacists and others will greatly improve its quality.

SECTION I



DISEASES OF CATTLE[1]

1 *Guide Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.*

NON INFECTIOUS DISEASES OF CATTLE

Bloat

It is an over distention of the rumen and also reticulum with retained gases of fermentation. These gases can either be in the form of persistent foam mixed with rumen content (primary bloat or frothy bloat) or free gas separated from ingesta (secondary bloat, free gas bloat)

Clinical Symptoms

Mild to marked distension of left flank and in severe cases right abdomen may also be distended. Protrusion of the paralumbar fossa above the ventral column and enlarged abdomen, dyspnea, granting and mouth breathing, protrusion of the tongue and extension of the head and occasionally vomiting may be observed.

Diagnosis

It is easy to diagnose frothy bloat as passage of stomach tube relieves or releases only small amount of gas. The causes of secondary bloat must be ascertained by clinical examination to determine cause of failure to eructate.

Treatment and Prevention

Non Drug Treatment

Remove free-gas by passing a stomach tube in free gas bloat.

Drug Treatment

Vegetable or mineral oil at a dosage of 250–500 mL/animal PO via stomach tube once. Or
Poloxalene (25–50g, PO) is effective in treating frothy bloat caused by legume but not feedlot bloat.

Surgical Treatment:

Emergency rumenotomy in life threatening cases or use of a trocar and cannula with a bore of 2.5cm to puncture the rumen and administer antifoaming agents e.g. vegetable or mineral oils 80-250 ml/animal through the hole into the rumen.

Prevention

Gradual adaptation of animals to high grain rations
Wilt highly leguminous plants before feeding to cattle
Feed cattle on hay before leguminous pasture
Give oils and fats (in dangerous situations) at 60-120 ml/head/day; may be increased up to 240 ml if monoionic surfactants like poloxalene are used
Monensin, at 300mg daily protects against pasture bloat.

Grain Overload /Carbohydrate Engorgement/Lactic Acidosis

Grain overload (Rumen Overload; Rumen Acidosis) is an acute disease of ruminants characterized by indigestion, rumen stasis, dehydration, acidosis, toxemia, incoordination, collapse and in severe cases death. It is caused by unrestricted access to highly fermentable feedstuff by unaccustomed animal or in larger volume than the normal amount.

Clinical Symptoms

Splashy rumen, profuse diarrhea which is fetid and may contain undigested grain, dehydration, reduced or absence of rumen motility, subnormal body temperature (36.5-38.5 °C), shallow and rapid respirations and increased heart rate.Prognosis is poor for cattle with heart rates of 120-140 bpm. Recumbency follows after about 48 hours but may occur earlier.

Diagnosis

Diagnosis is based on history of unlimited exposure to grains, clinical findings and a low ruminal pH (5.5-6). Examination of rumen microflora. A pH of <5 indicates severe acidosis.

Treatment and Prevention

Non Drug Treatment

Surgical intervention (Cud transfer): Remove the rumen contents and replace with ingesta taken from healthy animals e.g. animals slaughtered in the abattoir.

Drug Treatment

Magnesium carbonate or magnesium hydroxide 1g/kg, PO mixed in 8 to 12 liters of warm water, repeated every 6 to 12 hours; if the rumen is evacuated, do not exceed 225g/ 450kg bodyweight
Activated charcoal 2g/kg to inactivate endotoxemia
Sodium bicarbonate 5% solution, 5 L/450 kg IV within ~30 min.

Plus

Balanced Electrolyte Solution (BES)/Normal Saline, or a 1.3% solution of sodium bicarbonate in saline, up to 60 L/450 kg, IV for the next 6-12 hours for maintenance.
Flunixin meglumine: To treat endotoxomeia, 1.1-2.2 mg/kg IV, IM once daily for three days. Avoid rapid intravenous administration of the drug.
Procaine penicillin G (22,000 U/kg/day) should be administered IM to all affected animals for at least 5 days to minimize development of bacterial rumenitis and liver abscesses.

Prevention

Avoid sudden and drastic ration changes.
Restrict animals from accessing grains and feeds should contain at least 10% roughage.
In feedlot, give ionophore compounds such as monensin to maintain the rumen pH at a higher level.

Note: The principles of treatment are to correct the ruminal and systemic acidosis and prevent further production of lactic acid, restore fluid and electrolyte losses and maintain circulating blood volume and restore stomach and intestinal motility to normal.
Treatment should commence only if animals are not in serious condition; otherwise slaughter is recommended.

Ketosis

Ketosis (Acetonemia) is a metabolic disease of lactating dairy cows that occur as result of negative energy balance. This results from loss of glucose through milk in highly productive cows leading to low blood glucose level. When blood glucose level is too low, the cow mobilizes body reserves, especially body fat. Part of the mobilized body fat will be converted to ketones in the liver (e.g. acetone) which results in elevated ketone levels in the cow's blood.

Clinical Symptoms

Lack of appetite, constipation, mucus covered feces, depression, staring expression, drop in milk production, arched back posture and loss of weight. CNS signs like circling, staggering, licking, chewing and bellowing, hyperaesthesia, compulsive walking and head pressing. The breath has acetone odor.

Diagnosis

History on the length of dry period, level of nutrition during the dry period and after parturition, daily milk production records if available. Blood glucose levels drop from the normal level of 400-600 mg/L to 250 mg/L in clinical ketosis. Ketone bodies in urine increase from 500 mg/L up to 12000 mg/L.

Treatment and Prevention

Drug Treatment

Glucose (dextrose 50%) solution, 500 mL, IV and Propylene glycol (glucose precursor) 125-250 g/dose mixed with an equal volume of water, PO q 12h for 2 days as initial dose

Propylene glycol 100 g PO, daily for 2 days for maintenance

Plus

Dexamethasone 5-20 mg/dose, IM once, in combination with glucose or glucose precursors.

Prevention

Cows at calving should not be too fat or in very poor condition

Avoid sudden change of feed

Add sufficient protein to the ration.

Precautions: Following adequate fluid volume replacement, steroids should be given as a single IV injection over a 13 minute period.

Parturient Paresis /Hypocalcemia, Milk Fever

Parturient paresis (Milk fever) is a metabolic disease of mature high producing dairy cows predominantly during the first 48 hours of parturition. The disease is associated with low level of ionized calcium in plasma. It is characterized by general muscle weakness, circulatory collapse and depression and predisposes cows to dystocia, uterine prolapse and retained fetal membranes.

Clinical Symptoms

There are three clinical stages related to the severity of hypocalcaemia.

Stage 1: Lack of appetite, lethargy (weakness), dullness, cold ears and dilated pupil.

Stage 2: Muscle tremor particularly of the head and limbs, grinding of teeth and incoordination may occur. Hyperexcitability and hypersensitivity may occur.

Stage 3: This is the final stage, the cow becomes recumbent, drowsy (lethargic), flaccid paralysis. At first the animal lies on the sternum with curvature of the neck and may struggle to stand, then lies on the lateral side. Body temperature becomes subnormal, heart rate increases and irregular breathing.

Diagnosis

Hypocalcaemia can be diagnosed based on history, clinical signs, quick response to calcium borogluconate and by measuring the blood levels of calcium.

Differential diagnosis includes metabolic and nutritional diseases like: Hypophosphatemia, hypomagnesaemia, fat cow syndrome, carbohydrate engorgement.

Treatment and Prevention

Drug Treatment

Calcium borogluconate single IV dose of between 8 and 10 g Ca; 400 ml of 40% solution will give 12 g of available Ca. The drug is infused within 5 to 10 minutes. It may also be concurrently administered Propylene glycol 125 – 250 g/dose PO q 12 h mixed with an equal volume of water prevents relapse.

Plus

Calcium borogluconate as above for maintenance.

Prevention

Maintain low level calcium in feed (<50g Ca/day), supply Mg (>50 g per day) and limit phosphorus intake to less than 45 gram per day (at 0.35 % of the diet).

General rule for fluid therapy is to give 1 g calcium /45 kg body wt. The amount of fluid to be supplied as initial treatment is given below.

Precautions: Following adequate fluid volume replacement, steroids should be given as a single IV injection over a 13 minute period.

NOTE: Quick response to treatment with calcium borogluconate intravenous injection is confirmatory diagnosis for hypocalcaemia.

Pregnancy Toxemia (Fat Cow Syndrome)

Pregnancy toxemia results from fetal carbohydrate or energy demand exceeding maternal supply during the last trimester of pregnancy. It is precipitated by large or multiple fetuses, low energy or protein feeds and health conditions that increase energy demand or decreased ability to take in feed.

Clinical Symptoms
Mild cases: Decreased appetite, rumination, fecal production and nose licking.
Severe cases: Depression, weakness, ataxia and recumbency. Opisthotonos, seizures or coma may be seen terminally.

Diagnosis
The history, stage of pregnancy and nutritional status give tentative diagnosis. Elevated ketone and decreased calcium levels in blood are confirmatory.
Differential diagnosis includes milk fever, ketosis, and abomasal displacement.

Treatment and Prevention
Supportive treatment
Glucose (dextrose 5%) solution, 500 ml, IV
Calcium borogluconate + Magnesium Hypophosphite Hexahydrate and 20% glucose 500 ml, IV.
Propylene glycol 125 – 250 g PO q 12 h in severe cases
Drug treatment
Anabolic steroids like dihydrotestesterone undecyclenate, 200-300 mg/animal, IM, stat.

Prevention
Pregnancy toxemia can be prevented by management strategies that maintain a good appetite and supply of adequate feed to meet this demand of energy during the late stages of pregnancy. Avoid over conditioning of dairy cow.

Note: Early treatment becomes successful.

Simple Indigestion

Simple indigestion or ruminal atony is characterized by accumulation of indigestible feed in the rumen. It is caused by a sudden change of the feed, such as addition of urea to a ration, turning cattle onto a lush cereal grain pasture or introducing feedlot cattle to a high-level grain ration. This may lead to excessive fermentation or putrefaction and impaired rumen function for 24-48 hours

Clinical Symptoms
Silage overfeeding: anorexia, rumen becomes full, firm and doughy, primary ruminal contractions are absent, but secondary contractions may be present.
Excessive feeding of grain: anorexia and ruminal stasis; the rumen is not necessarily full and may contain excessive fluid; the feces are usually soft and with a foul smell.

Diagnosis
This is based largely on elimination of other possibilities and a history of a change in nature or amount of the diet.

Treatment and Prevention

Non-drug treatment

Warm water or saline, 20-40 L, PO, followed by vigorous kneading of the rumen.

Drug Treatment

For Indigestion due to excessive high-energy feeds intake: Magnesium hydroxide 0.5 mg/kg in a 10% aqueous suspension, PO or by stomach tube.

For Indigestion due to too much urea or protein intake: Acetic acid 5% or vinegar, PO by stomach tube.

4-8 L of ruminal fluid from a healthy cow in case of reduced ruminal microbes.

Prevention

Gradual adaptation of feed change and avoid accidental access to highly fermentable feedstuff.

Traumatic Reticuloperitonitis/Hardware disease

Traumatic reticuloperitonitis occurs as a consequence of perforation of the reticulum by sharp foreign objects such as nails or pieces of wire. Perforation of the wall of the reticulum by a sharp foreign body initially produces an acute local peritonitis, which may spread to cause acute diffuse peritonitis. The penetration of the foreign body may proceed beyond the peritoneum into other organs resulting in pericarditis, pleuritis and pneumonia.

Clinical Symptoms

Sudden anorexia and fall in milk yield, ruminal stasis and local pain in the abdomen. The animal will be reluctant to walk and does so slowly, particularly downhill and is often accompanied by grunting. Painful and infrequent defecation and urination usually accompanied with grunting. Muffled heart sounds, jugular vein distention with a pronounced jugular pulsation, marked submandibular and brisket edemas.

Diagnosis

Based on clinical signs. Pinching the back or holding the animal by a wood at the sternum to detect pain reaction.

Treatment and Prevention

Non-surgical or conservative treatment

Confine cows for 1-2 weeks, placing them on an inclined plane (forequarters raised by 25 cm) to limit further advance of the sharp foreign body to the thoracic cavity

Administer antibacterial drugs for 3-5 days; Oxytetracycline (10%) 5–10 mg/kg/day, IM or slow IV. **Or** Procaine penicillin G 22,000 IU/Kg, aqueous suspension, IM or SC, q 24 hr. for 3 to 5 days or repository preparations q 48-72 h.

Surgical treatment

Left side laparotomy, exploratory rumenotomy with manual removal of the foreign body from the reticulum. Abscess should be aspirated and drained.

Flexible magnetic metal retrievers, PO or via an incision in the flank.

Supportive therapy

Oral or occasionally IV fluids and SC calcium borogluconate or Calcium gluconate should be administered as needed.As a post-operative management, administer opioid or NSAIDs

Clean the wound daily with mild antiseptic solution such as 0.05% chlorhexidine. Remove paralumbar skin sutures in 14 to 21 days.

Urolithiasis

Urolithiasis is a common urinary tract disease in cattle caused by obstructive uroliths. Uroliths most frequently lodge in the distal sigmoid flexure near the insertion of the retractor penis muscle where the urethral diameter is narrowed in male ruminants. Risk factors include high dietary phosphorus/calcium ratio, high dietary magnesium, low fiber content of rations, low urine output and an alkaline urine pH. Early castration also predisposes to urolithiasis due to removal of sex hormones necessary for development and maturation of the penis and urethra.

Clinical symptoms

In partial obstruction, animals dribble blood-tinged urine, pain on urination and on complete obstruction tenesmus, tail twitching, weight shifting and signs consistent with colic are observed. Inappetence, depression and rectal prolapse may also be seen. Affected steers may elevate the tail and show urethral pulsations just ventral to the rectum. Complete occlusion results in urethral perforation or urinary bladder rupture and animals die due to uremia.

Diagnosis

The history, clinical signs, and physical examination are helpful, but definitive diagnosis requires using diagnostic imaging such as ultrasonography and/or radiography. Urinalysis may reveal proteinuria and occult hematuria.

Treatment and Prevention

Drug Treatment

Conservative therapy involving administration of antispasmodics (Diazepam 0.1 mg/kg, slow IV) and tranquilizers (Xylazine 0.05-0.1 mg/kg, IV or IM) to relax urethra and straighten the sigmoid flexure. Or In severe uremic and dehydrated animals, fluid therapy for correction of acid-base and electrolyte abnormalities. 0.9% NaCl IV is a good choice. Urine acidification using ammonium chloride (200-300 mg/kg PO SID or 2-4 g PO SID) administered in feed or syrup to acidify urine and aid in struvite stone dissolution. NSAIDS (e.g. flunixin meglumine at 1-2 mg/kg IV or IM) are useful to reduce swelling and irritation of the urethra. Antimicrobial therapy: If an active UTI is present, a minimum of 3 weeks of antimicrobial treatment is recommended. Penicillin, Ampicillin, sulphadimidine sodium (107 mg/kg, IV or PO q 24 hr.).

Prevention

Increasing water intake and urine volume
Adjust calcium to phosphorus ratios of the feed at 2.5:1 or 2:1
Feeding of urine acidifier (anionic salts containing chlorides) such as sodium chloride (3-5%), calcium chloride (1%–2%) and ammonium chloride (0.5%–2%) (45 g/steer/day) added to the dietary dry matter intake and supplement with Vitamin A.

Note: Surgical treatment of urolithiasis is usually done as a short-term salvage procedure in cattle destined for slaughter.
During urine acidification urine pH should ideally be between 6.0pH and 6.8pH and should be monitored once to twice a week. Excessive urine acidification can lead to metabolic acidosis and the formation of calcium oxalate containing uroliths.

VIRAL DISEASES OF CATTLE

Ephemeral Fever

Bovine ephemeral fever is a non-contagious vector-borne viral disease of cattle characterized by biphasic fever, stiffness or lameness and drop in milk production (in dairy).

Clinical Symptoms
Biphasic fever that occurs for 3-4 days, stiffness or lameness of more than one leg, sudden drop in milk production are observed. Other signs include shivering, nasal and ocular discharge, edema affecting the area around joints and subcutaneous emphysema.

Diagnosis
Clinical findings, demonstration of viraemia or of high levels of neutralizing antibody and isolations are confirmatory.

Treatment and Prevention
No specific treatment however to reduce pain Acetylsalicylic acid 60 to 120 g/head, PO, q 12 h, or Phenylbutazone120 mg/ml plus isopyrin 240 mg/ml), 20 to 30 ml, IV slowly or IM can be administered.

Foot and Mouth Disease

Foot-and-Mouth Disease (FMD) is a highly communicable viral infection of cattle, pigs, sheep, goats, and buffalo. It is caused by an **Aphthovirus** that is transmitted by contact. Recovered animals remain carriers for up to 2 and a half years.

Clinical Symptoms
The disease is characterized by fever, anorexia, vesicles in the mouth on the muzzle, gums, pharynx, teats and interdigital cleft. Dullness, inappetence, fever and shivering followed by smacking of lips, drooling saliva and shaking or kicking of the feet are primary signs. Pregnant animals may abort.

Diagnosis
Clinical signs are indicative and confirmed by serology and isolation of the virus.

Treatment and Prevention
No specific treatment however, supportive treatments against secondary bacterial infection are necessary.

Prevention
Vaccination, test and quarantine of infected herds and movement control.

Infectious Bovine Rhinotracheitis

IBR is a viral disease of cattle caused by Bovine herpesvirus 1 (BHV 1 IBR virus) which is also Causative agent of respiratory and genital infection causing abortion. Transmission is usually by body contact (such as licking, nuzzling and contact with body fluid and aerosol over a short distance) is vital, primarily via respiratory route, but also venereal, by contaminated semen from virus shedding bulls (intermittent shedding). Bulls may shed virus in the semen both during clinical and subclinical infection. After genital infection and seroconversion, BHV-1, localizes and persists latently in sacral ganglia. Shedding of the virus reoccurs during periodic reactivation of viral replication. Viral reactivation from latent state is generally thought to be stress-induced but can also be induced by injection of corticosteroids (dexamethasone).

Clinical Symptoms
A major feature of the diseases is that after infection the virus invariably remains in the nervous system for the rest of the animal's life (latent infection) and sporadic shedding of the virus. IPV and IPB are features of venereally-transmitted form of the diseases

Diagnosis
Viral isolation from semen in tissue culture. Dilution of the semen with tissue culture medium to reduce the cytotoxicity of semen is one disadvantage reducing sensitivity to detect low titer. Also may take 1-3 weeks. Nested PCR assay, serology, and histopathology.

Treatment and Prevention
There is no effective treatment

Prevention
Both inactivated and live attenuated vaccines are available. Immunity usually lasts 6-12 months. Marker vaccines or DIVA (differentiation of infected from vaccinated animals) are widely used to both protect cattle clinically in the case of infection and significantly reduce the shedding of the virus.
Vaccination prevents diseases but does not eradicate virus from the herd. So once vaccination is started in an infected herd all introduced cattle (purchased or homebred) should be vaccinated before they contact the infected herd.

Dedicated clothing and equipment and Isolation pens are needed for control of the diseases.
Note: Eradication is a time consuming process, is a difficult goal, and requires diligent testing. In a small, vaccinated herd BoHV-1 can be eradicated within one or two decades, whereas in a large herds eradication is unlikely. Any cattle over 8 months of age with antibody to BHV-1 are almost certainly latently infected for the rest of their lives and can excrete infectious virus at any time.

Lumpy Skin Disease

Lumpy Skin Disease (LSD) is a highly infectious viral disease of cattle characterized by pox like intracutaneous firm nodules, edema of the limbs, superficial lymph nodes swelling and lymphangitis. The disease is widely distributed in Ethiopia and causes severe economic losses.

Clinical Symptoms
Painful swelling, fever, lacrimation, nasal discharge and hyper salivation followed by characteristic nodules on the skin and other parts of the body. The nodules are circumscribed, round, slightly raised, firm and painful and involve the entire cutis and mucosa of the gastrointestinal, respiratory and genital tract. The regional lymph nodes are swollen and edema develops in the udder, brisket and legs.

Diagnosis
The widespread nodular lesions of the skin and mucous membranes and biphasic fever are indicative.

Treatment and Prevention
There is no effective treatment but secondary bacterial infections are prevented by administration of broad-spectrum antibiotics.

Prevention
Vaccination with sheep/goat poxvirus or LSD strain.

Malignant Catarrhal Fever

Malignant Catarrhal Fever (MCF) is an acute, highly fatal viral disease of cattle and other ruminants caused by a lymphotropic herpes virus. Wildebeests are natural reservoirs of infection to cattle. MCF has two forms namely: Peracute and catarrh form.

Clinical Symptoms
The most common types of the disease is the head and eye form. Typical symptoms of this form include fever, depression, occulonasal discharge, lessions in the oral cavity and muzzle, lymphadenopathy, corneal opacity leading to blindness, innappetance and diarrhea. Some animals have neurological signs, such as ataxia, nystagmus and head pressing.

Diagnosis
Characteristic pathologic findings, serologic conversion and histopathologic evidence of a necrotizing vasculitis.

Treatment and Prevention
No specific treatment but antibiotic therapy to prevent secondary bacterial infections. Corticosteroids are contraindicated.

Rabies

Rabies is a fatal viral infection of all warm-blooded animals and transmitted by a bite of infected animal. It is caused by **Lyssavirus,** family **Rhabdoviridae** and manifested by motor irritation with clinical signs of mania and an attack complex and by an ascending paralysis.

Clinical Symptoms
The initial clinical signs are often nonspecific and may include fearfulness, restlessness, anorexia or an increased appetite, vomiting, diarrhoea, dilation of the pupils, hyperreactivity to stimuli, excessive salivation, wandering, attacks on other animals, people or inanimate objects. The eyes and ears follow sounds and movement and show characteristic abnormal bellowing, which may continue intermittently until shortly before death.

Diagnosis
Clinical diagnosis is difficult and confusing with other diseases. Immunofluorescence microscopy on fresh brain tissue smears stained with sellers stain is the test of choice. The mouse inoculation test or tissue culture techniques using mouse neuroblastoma cells or both are also used.
Any suspected mammalian encephalitis and neurological disorder must be considered in the differential diagnosis.

Treatment and Prevention
There is no effective treatment, however it can be prevented by vaccination of domestic dogs and stray dog control.

Public health significance: Rabies is the most fatal infectious zoonotic disease transmitted by bite of infected animals.

Rift Valley Fever

Rift Valley Fever (RVF) is a peracute or acute zoonotic disease of domestic ruminants caused by **phlebovirus**. Many different species of mosquitoes are vectors for RVF virus. The disease is more severe in sheep than in other animals. Though less severe, the epidemiology of RVF in cattle is similar to sheep.

Clinical Symptoms
Depends on the species of animal affected, age and pregnancy status. The diseases is characterized by fever, severe illness, nasal discharge, abortion, high morbidity and mortality rate.

Diagnosis
Clinical signs in endemic areas. Laboratory tests are required to confirm the case (OIE Terrestrial Animal Health Code and OIE Manual Diagnostic Test and vaccine for Terrestrial Animals).

Treatment and Prevention
There is no specific treatment

Prevention
Vaccination in endemic areas. A modified live vaccine is available that requires only one dose and produce long-lived immunity.
Control of vectors (Mosquitoes)

Public health significance: Rift valley fever is a fatal, zoonotic disease transmissible to humans.

Rinderpest

Rinderpest or cattle plague, is a highly contagious, usually fatal, acute or subacute viral disease of ruminants caused by morbilli virus. Ethiopia is declared free of infection since 2005.

Clinical Symptoms
Fever, lacrimation, nasal discharge, profuse diarrhoea and necrotic erosions of the epithelium of the mouth and other mucosa of the digestive tract.

Diagnosis
Provisional diagnosis is based on history, clinical signs and pathological lesions. Infection is confirmed by serological tests and culture.

Treatment and Prevention
There is no specific treatment available. However it can be prevented by maintaining disease free status by importation of animals from Rinderpest -free countries only; ring vaccination if reintroduced and proper disposal and hygiene of infected animals.

BACTERIAL DISEASES OF CATTLE

Actinobacillosis

Actinobacillosis also known as wooden tongue is a sporadic disease of cattle and sheep commonly caused by the bacteria **A. lignieresii**. It is characterized by nodular abscession of soft tissues.

Clinical Symptoms
Excessive salivation, gentle chewing of the tongue and upon palpation the tongue is swollen and hard, particularly at the base, the tip often appearing to be normal. Nodules, ulcers and abscess are present on the side of the tongue. In the later stages the acute inflammation is replaced by fibrous tissue and tongue becomes shrunken, hard and immobile.

Diagnosis
Clinical manifestations are fairly distinctive; Gram stained preparations from crushed exudates, cultural examination and response to treatment with iodine preparations.

Treatment and Prevention
Drug Treatment
Sodium iodide 8g/100kg, 10% solution, IV once or local injection at the tumorous masses
Potassium iodide 6-10g/day/animal, PO, for 7-10 days
Procaine penicillin G 22,000 IU/kg, aqueous suspension, IM or SC, q 24 hr. for 3 to 5 days. **Or**
Alternative
Streptomycin or dihydrostreptomycin sulfate 5.5 mg/kg, IM, q 24 hr. for 5 days. **Or**
Sulfadimidine sodium 107 mg/kg, IV or PO q 24 hr. **Or**
Oxytetracycline 10-20 mg/kg (adult) and 20-40/ kg (calves) for 3-5 days.

Prevention:
No vaccines are available
Control of actinobacillosis is best achieved by early recognition and prompt treatment of cases; isolation or disposal of animals with disease is recommended.

Note: The appearance of signs like lacrimation, anorexia, coughing and the appearance of dandruff indicate that maximum systemic levels of iodine have been reached.
Iodides should not be given to milking cows whose milk will enter the human food chain.

Actinomycosis/ Lumpy Jaw

Actinomycosis or lumpy jaw is a subacute or chronic bacterial disease of cattle caused by **Actinomyces bovis**. Injury or wound in the oral cavity is the predisposing factor. It is characterized by swelling of the mandible, maxillae or other bony tissues. In young animals the infection can also occur through dental alveoli when the teeth are erupting.

Clinical Symptoms
Initially painless, hard, immovable bony swelling on mandible or maxilla. Eventually discharge small amounts of pus through one or more openings in skin.

Diagnosis
Clinical signs in conjunction with the demonstration of Gram-positive rod shaped bacteria in yellowish "sulfur granules" from aspirated purulent material will help confirm the diagnosis.

Treatment and Prevention

Non drug Treatment

Surgical debridement of the granules with antibiotic therapy for 2-4 weeks.

Drug treatment

Sodium iodide 1 g/12kg, 10% solution in sterile distilled water IV or injected locally into the tumorous masses, stat. For effective treatment of bony lesion, at least one or preferably two further treatments at 10- to 14-day interval is required **Or**

Potassium iodide 6-10g/day/animal, PO, for 7-10 days

Procaine penicillin G 22,000 IU/kg, aqueous suspension, IM or SC, q 24 h for 3 to 5 days.

Alternative

Oxytetracycline 10 mg/kg for 3-5 days. **Or**

Erythromycin base 2-4 mg/kg, IM q 24 hr. for 3-5 days.

Isoniazid 10-20 mg/kg, PO, for 30 days.

Prevention

Avoiding coarse, stemmy feeds that might damage the mucosal epithelium.

Anthrax

Anthrax is a zoonotic bacterial disease caused by **Bacillus anthracis**. Anthrax is most common in wild and domestic herbivores (e.g. cattle, sheep, goats, camels, antelopes). It is primarily a disease of herbivores. However, it can also affect carnivores and scavengers but large outbreaks in these groups are very rare. Cattle are highly susceptible.

Clinical Symptoms

Peracute and acute forms are the common clinical forms of anthrax in cattle. Peracute form is characterized by sudden death. Acute illness is characterized by fever and excitement followed by depression, stupor, anorexia muscle tremors and dyspnea. Pregnant cows may abort. All natural orifices usually exude dark blood that does not clot.

Diagnosis

Anthrax should be suspected if an animal dies suddenly and further investigated by methylene blue stained blood smears.

Differential diagnosis of anthrax in cattle includes lightning strike, accidental electrocution, pasteurellosis, piroplasmosis, blackleg, malignant edema, intoxications, botulism, chemical poisoning and snake bite.

Treatment and Prevention

Drug Treatment

Penicillin 22,000 IU/kg, IM, q 12hr. for 2 days, then daily for 3 days or Benzathine penicillin or other repository preparations, q 48-72hr. the initial dose should be administered IV. **Or**

Dihydrostreptomycin or streptomycin, 10 mg/kg, q 12hr. IM, SC. **Or**

Oxytetracycline 6-11 mg/kg, IM or IV, q 12-24 hr. Initially, divide the daily dose into two doses. **Or**

Amoxicillin 5-10 mg/kg q 24 hr. for 3-5 days. **Or**

Doxycycline 20 mg/kg, IV.

Prevention

Vaccination using Stern avirulent live spore vaccine and proper burial and burning of carcass. Attention should be given to selection of an appropriate site for carcass burial.

Necropsy of affected carcasses is not advised. Care should be taken not to contaminate water sources, residential areas, livestock facilities, pastures and other establishments in the area. Take all the necessary precaution in handling a case suspected of anthrax and take all necessary measures not to contaminate the environment while taking samples. Animals should not be vaccinated within 2 months of anticipated slaughter; antibiotics should not be administered within 1 week of vaccination.

Bacillary Hemoglobinuria

Bacillary hemoglobinuria is an acute and highly fatal clostridial disease that affects primarily cattle and is caused by **Clostridium haemolyticum**. The highest incidence of bacillary hemoglobinuria occurs in irrigated or poorly drained pasture, especially in alkaline soils. It is rare in calves less than 1 year old and cattle with poor body condition. It causes severe hepatic necrosis and local thrombosis.

Clinical Symptoms

Depression, arched back, the animal grunts when walking, fever of 40-41°C, red dark urine, jaundice and anemia. Other clinical signs include abdominal pain, dyspnea, dysentery, hemoglobinuria and edema of the brisket. Pregnant cows often abort.

Diagnosis

Clinical signs and postmortem findings usually permit a tentative diagnosis. Diagnosis can be confirmed by isolating **C. haemolyticum** from the liver infarct, but the organism is difficult to culture. Rapid and accurate diagnosis can be made by demonstrating the organism in the liver tissue by a fluorescent antibody or immunohistochemical test and PCR.

Differential diagnosis includes babesiosis, leptospirosis, postparturient haemoglobinuria and haemolytic anemia caused by poisonous plants.

Treatment and Prevention

Non Drug Treatment

Blood transfusions, parenteral fluid and electrolytes may help to control hemolytic anemia and the dehydration

Antitoxin 500-1000 ml per adult animal.

Drug Treatment

Procaine penicillin G, 22,000 IU/kg, IM or SC q 24 hr. for 3 to 5 days or Benzathine penicillin or similar repository preparations, q 48-72 hr. **Or**

Oxytetracycline 10 mg/kg IV or IM q 24 hr.

Prevention

Proper disposal of carcass is mandatory

Vaccination with C. haemolyticum bacterin

Note: Early treatment with penicillin or tetracyclines at high doses is essential; bulls should not be allowed to mate within 3 weeks to avoid liver rupture

Blackleg

Black leg is an acute, febrile disease of cattle caused by **Clostridium chauvoei** characterized by emphysematous swelling of the heavy muscles and severe toxemia. Black leg is common in Ethiopia during dry periods of the year.

Clinical Symptoms

Depression, anorexia, rumen stasis, high fever (41-42°C) and tachycardia, marked lameness with pronounced muscle swelling of the upper limb with crepitation sound.

Diagnosis

The clinical signs, epidemiology and postmortem findings are indicative and bacterial isolation is confirmatory.

Differential diagnosis of blackleg includes Lightning strike, accidental electrocution, pasteurellosis, piroplasmosis, malignant edema, intoxications, botulism, chemical poisoning and snake bite.

Treatment and Prevention

Drug Treatment

Procaine penicillin G, 22,000 IU/kg, IM or SC q 24 h for 3 to 5 days or Benzathine penicillin q 48-72 h.

Prevention

Annual vaccination of all cattle with **C. chauvoei** bacterin is used to prevent the disease
In an outbreak; all susceptible cattle should be vaccinated and treated prophylactically with penicillin (10,000 IU/kg, IM) to prevent new cases.

Botulism

Botulism is rapidly fatal motor paralysis caused by ingestion of preformed neurotoxin produced by **Clostridium botulinum** during vegetative growth from vegetable materials such as decaying grass, hay, grain or spoiled silage. Ingestion of botulism toxin from decaying tortoise carcasses has been reported in Bale zone, southern Ethiopia.

Clinical Symptoms

Decreased tongue tone that protrudes out and problems associated with prehension and deglutition (swallowing) of food, followed by progressive muscular weakness until animal becomes recumbent in a parturient paresis-like posture, ataxia and stumbling gait affecting the hind legs are commonly observed.

Diagnosis

Commonly, the diagnosis is made by eliminating other causes of motor paralysis and search for potential sources of toxin.
Differential diagnosis of botulism includes Tick paralysis, Paralytic rabies Organophosphate/carbamate poisoning.

Treatment and Prevention

Drug treatment

Ruminal lavage, followed by 50-80 ml lactic acid in 5-10 L of water through a stomach tube in adult cattle.
Botulinum antitoxin (Polyvalent anti-C and anti-D antisera (if available).

Prevention

Correction of dietary deficiencies, proper disposal of carcass and removal of decaying grass or spoiled silage from cattle feed is indicated for prevention.

Bovine Farcy

It is a chronic infectious disease of zebu cattle caused by **Mycobacterium farcinogenes** and **M. senegalense.** It is characterized by purulent lymphangitis and lymphadenitis.

Clinical Symptoms

Chronic, painless, localized subcutaneous cellulitis, which spreads along lymphatics to involve local lymph nodes. The lesions are sub-cutaneous swellings, enlargement and thickening of local lymphatics and lymph nodes that may rupture and ooze an odorless thick gray or yellow, often granular or cheesy pus.

Diagnosis

It is based on microscopic examination of smears from pus or isolation of the agent and CFT.

Treatment and Prevention

Sodium iodide1 g/14 kg, 10%, IV, every 7 days, for more than 5 weeks.

Prevention

Early disinfection of cutaneous abrasions in cattle on affected farms is recommended for prevention.

Calf Diphtheria

Calf diphtheria (Necrotic laryngitis, Laryngeal necrobacillosis) is a disease of young cattle caused by invasion of **Fusobacterium necrophorum** into laryngeal mucosa and cartilage. Calf diphtheria primarily affects cattle between 3 and 18 months of age; however, it may sometimes occur in adults.

Clinical Symptoms

Severe inspiratory dyspnea, salivation, painful swallowing movements, complete anorexia and severe depression are the characteristic signs. High temperature (41°C; 106°F), the pharyngeal region may be swollen and painful on external palpation and there is salivation and nasal discharge. The breath has a foul rancid smell. Untreated calves may develop necrotized pneumonia and die within 2-7 days from toxemia and upper air way obstruction.

Diagnosis

Clinical signs are usually sufficient to establish a diagnosis.

Treatment and Prevention

Non Drug Treatment

Debridement of the ulcers and application of a solution of tincture of iodine.

Drug Treatment

Sulfadimidine, initial dose: 140 mg/kg, IV; maintenance dose: 70 mg/kg, IV, q 24 hr. for 5-7 days. **Or**
Procaine penicillin, G, 22,000 IU/kg, IM or SC q 24 hr. for 3 to 5 days or Benzathine penicillin or similar repository preparations, q 48-72 hr.

Supportive treatment

A single dose of dexamethasone (0.2–0.5 mg/kg, IV or IM) may be used to decrease laryngeal edema in animals with severe respiratory distress. **Or**
Flunixin Meglumine. **Or**
Acetylsalicylic acid (Aspirin) 100 mg/kg, PO, q 12 hr. to decrease the degree of laryngeal inflammation and edema.

Prevention

Good hygiene in calf pens or feeding and drinking places together with avoidance of rough feed should prevent the spread of the disease. When the incidence is high prophylactic antibiotic can prevent the disease occurrence.

Bovine Brucellosis

Bovine Brucellosis is a highly contagious disease mainly affecting cattle caused by **Brucella abortus.** It is characterized by abortion late in pregnancy and subsequent high rate of infertility in female and varying degree of sterility in the male. It is an important zoonosis, causing undulant fever in humans.

Clinical Symptoms

Abortion (after 6 month of pregnancy) is the cardinal feature of the disease in cows and retention of placenta and metritis are the common sequels to abortion. In subsequent pregnancies the fetus is usually carried to full term. In bulls clinical signs include orchitis, epididymitis and enlargement of seminal vesicles. Such bulls are potential carriers of the disease.

Diagnosis

The presence of clinical history like abortion, retention of placenta and orchitis in bull are suggestive. Serological examination using Rose Bengal plate test and CFT are used for serological detection of infected animals.

Differential diagnosis includes Trichomoniasis, neosporosis, leptospirosis, infectious bovine rhinotracheitis, listeriosis and epizootic viral abortion.

Treatment and Prevention

Treatment is unsuccessful because of the intracellular sequestration of the organisms.

Prevention

Careful selection of replacement animal
Good herd health management and application of biosecurity measures
Separate calving pen
Culling of infected animals
Vaccination of calves with B. abortus Strain 19 or RB51.

Campylobacteriosis

Campylobacteriosis is caused by **Campylobacter** species of bacteria. The disease is either intestinal presenting as diarrhea or genital; causing infertility or abortion. In livestock; **C. jejuni**, subspecies **venerealis**, **C. fetus** subspecies **fetus** and **C. fetus** subspecies **venerealis** are most important species. The organisms are transmitted via the fecal-oral route and via feed or water.

Clinical Symptoms

Clinical manifestations may be more severe in younger animals. Diarrhea is mucoid and occasionally with blood flecks in the mucus. Abortion may occur.

Diagnosis

Culture and using dark-field or phase-contrast microscopy.

Treatment and Prevention

Drug Treatment

For uterine and bull infections:

Streptomycin or dihydrostreptomycin, 1-2 g + 1 million IU Penicillin infusion, per animal for 3-5 days and locally it can be applied intrauterine. In bulls, it can be directly applied onto the prepuce. Or
Tylosin 5-10 mg/kg, IM or slow IV injection for not more than 5 days or in combination with sulfonamides.

Or

Erythromycin15 mg/kg q 12-24 hr. IM. For 3-5 days. **Or**
Ampicillin or Ampicillin + Clavulanic acid adult cattle: 15 mg/kg q 12 hr. IM; calves, 25 mg/kg q 8-12 hr. PO for 5 days.

For Enteritis

Erythromycin15 mg/kg q 12-24 hr. IM. For 3-5 days.
Doxycycline20 mg/kg, IV for 3 days.
Gentamicin5 mg/kg q 8 hr. IV or IM for 3-5 days.
Tilmicosin 10mg/kg SC or IM once.

Prevention

Vaccination with bacterins in an oil emulsion adjuvant.

Public health significance: The disease is transmitted to human by consumption of animal products resulting is gastroenteritis and diarrhea.

Colisepticemia

Colisepticemia is caused by **Escherichia coli**. It is a common disease of calves characterized by acute septicemia (2-5 weeks age) or of a chronic bacteremia (calves less than 2 weeks age) with localization. Initial infection can occur from a contaminated environment followed by direct nose-to-nose contact, respiratory aerosols or as the result of navel-sucking or fecal-oral contact. Failure of sufficient passive transfer of immunoglobulin predisposes calves to infection.

Clinical Symptoms

Affected animals are depressed and weak, commonly recumbent and dehydrated; tachycardia is present. Although the temperature may be high initially it falls rapidly to subnormal levels. The suckling reflex in claves is weak or absent, diarrhea and dysentery are common.

Diagnosis

History and clinical findings, demonstration of a severe deficiency of circulating IgG and the organism in the blood or tissues.

Treatment and Prevention

Non Drug Treatment

Fluid therapy for endotoxic shock as supportive treatment, **See Annex 5** for choices

Drug Treatment

Gentamicin 5 mg/kg q 8 h IV or IM for 3-5 days. **Or**
Trimethoprim-sulfadiazine 30mg/kg IM, q 24 h for 3-5 days. **Or**
Norfloxacin 20% oral liquid given twice daily at a rate of 10ml/75-150kg PO for 3-5 days
Lincomycin Hydrochloride: 5 mg /kg intramuscularly twice daily for the first day followed by once daily for 2-4 days
Enrofloxacin (5%) 5 mg of Enrofloxacin/kg IV or SC, once daily for 3-5 days.

Prevention

Prevention can be achieved by giving adequate colostrum to calves during the first 24 hours of life and keeping the calf in separate calf pen.

Caution: Animals should have a good water supply.

Dermatophilosis

Dermatophilosis is a skin disease of cattle and other domestic livestock caused by the bacteria **Dermtophilus congolensis**. It is clinically characterized by superficial, pustular, crusting, and/or ulcerative dermatitis.

Clinical Symptoms

Papules, serous exudates causing matting of hair to form a tufted appearance, scab formation may be severe and generalized.

Diagnosis

Clinical appearance, microscopic examination of geimsa or gram stained smear and culture.

Treatment and Prevention

Drug Treatment

Procaine penicillinG 70,000 IU/kg **plus** streptomycin 70 mg/kg, IM, single dose; if this fails continue treatment with respective dose of 5000IU and 5 mg/kg q 24 h for 5 days.
Oxytetracycline (LA) 20 mg/kg, IM, once; if required repeat after 3 to 5 days. **Or**
Amoxicillin 10 –15 mg/kg q24h IM.
The following Topical drugs can be used
Iodophores: 2-5% lime sulfur, 0.5% zinc sulfate 0.2% copper sulfate and 1% potassium aluminum sulfate (alum) as sprays or wash for 3 to 5 days, then weekly until the lesion heals.

Hemorrhagic Septicemia

Hemorrhagic septicemia is a highly fatal bacterial disease seen mainly in cattle and water buffalo. Hemorrhagic septicemia results from infection by **Pasteurella multocida** subsp. **multocida**. Only **P. multocida** serotypes B: 2 and E classically cause hemorrhagic septicemia.

Clinical Symptoms

Initial fever and inappetence followed by respiratory distress with profuse salivation and nasal discharge. A characteristic swelling of the head, throat and brisket, swollen hemorrhagic lymph nodes and numerous mucosal and subserosal petechial hemorrhages.

Diagnosis

Hemorrhagic septicemia should be suspected in animals with a rapid course of infection, fever and edematous swellings in the throat, neck and brisket. A high herd incidence, high case fatality rate and season of the year are also suggestive of this disease. Definitive diagnosis depends on identifying **Pasteurella multocida** serotypes. Hemagglutination could be confirmatory**.**
The differential diagnosis includes other causes of sudden death such as lightning strikes, blackleg (**Clostridium chauveoi** infection), rinderpest and anthrax.

Treatment and Prevention

Drug Treatment

Sulphadimidine 33%, IV, q 24 h for 3-5 days. **Or**
Oxytetracyline 5-10 mg/kg IM or IV, q 12-24 hr. Long-acting 20 mg/kg SC, IM or IV, q 2-4 days **Or**
Penicillin-streptomycin; 200,000 IU + 250 mg, 1ml/25kg, IM.
Amoxacillin + Clavulanic Acid 8.75 mg/kg bodyweight [7 mg/kg bodyweight Amoxicillin and 1.75 mg/kg/ bodyweight of Clavulanic acid] IM, once daily for 3-5 days.
Oxytetracycline + Flunixin Meglumine (30%,2%) 2 mg/kg Flunixin and 30 mg/kg Oxytetracycline, IM, once.

Infectious Keratoconjunctivitis

Infectious keratoconjunctivitis (pinkeye) is a disease of the eyes of cattle caused by the bacteria **Moraxella bovis**. In Ethiopia, the disease is more serious in exotic cattle breeds and particularly calves are more susceptible.

Clinical Symptoms

Generally, it is characterized by photophobia (increased sensitivity to light) blepharospasm, conjunctivitis with or without keratitis, lacrimation and varying degrees of focal corneal opacity, central corneal ulceration, mucopurulent ocular discharge and extensive corneal necrosis, corneal neovascularization, dense granulation tissue and corneal fibrosis.

Diagnosis

Seasonality of the disease, characteristic clinical signs and high incidence of ocular lesions are indicative of infectious keratoconjunctivitis. Microbial culture is confirmatory.

Treatment and Prevention

Drug Treatment

Gentamicin 0.3%, triple antibiotic (neomycin, bacitracin, and polymyxin B) applied topically
Oxytetracycline 20 mg/kg, IM, q 24 hr. for 2 days; long-acting formulation, stat. **Or**
Sulphadimidine 100 mg/kg, IM; other sulfonamides can be used. **Or**
Penicillin + streptomycin; 200,000 IU + 250 mg, 1ml/25kg, IM. O **Or**
Tulathromycin: 2.5 mg/kg (1ml/40 kg BW) into the neck SC stat. For treatment of cattle over 300 kg bodyweight, divide the dose so that no more than 7.5 ml are injected at one site. Plus
Dexamethasone 1mg, IM.

Prevention

For prevention and control minimize exposure to Moraxella, and maintaining irritant-free environment as much as possible. Active cases of pinkeye with excessive tearing attract flies that widely spread the bacteria. Topical application of a fly repellant on the face will also help reduce the spread.

Leptospirosis

Leptospirosis is a contagious disease of animals, including man, caused by **Leptospira interrogans** serovars. Serological evidence shows wide distribution of infection in irrigated areas of Ethiopia. It is transmitted by skin contact or mucous membrane and intake of urine contaminated feed and water.

Clinical Symptoms

Acute form occurs in calves, signs include fever, hemolytic anemia, hemoglobinuria, jaundice, pulmonary congestion and occasionally meningitis. High mortality agalactiae and blood tinged milk may occur in milking cows. In chronic form abortion, stillbirth or premature birth and weak calves with increased neonatal mortality and renal failure are the major signs.

Diagnosis

Dark field microscopy of fetal fluids, Microscopic Agglutination Test (MAT) ELISA and demonstration of leptospirosis in urine or tissues are commonly used. Isolation is difficult.

Treatment and Prevention

Drug Treatment

Tetracycline; 10-15 mg/kg, IM, q 12 hr. for 3-5 days **Or**
Streptomycin; 12.5 mg/kg, IM q 12 hr. for 3 days. **Or**
Penicillin-streptomycin 200,000 IU + 250 mg, 1ml/25kg, IM.

Prevention

Avoid direct contact with carriers or rodents and vaccination for the most endemic serovar.

Listeriosis

Listeriosis is a sporadic bacterial infection that affects a wide range of animals, including human and birds. It is principally caused by **Listeria monocytogenes**, and occasionally by **L. ivanovii**. Humans acquire infection by consuming not well cooked animal products

Clinical Symptoms

The characteristic symptoms are encephalitis or meningoencephalitis and abortion in adult cattle. Perinatal mortality, septicemia and fever in neonatal ruminants. Animals with encephalitis propel themselves into corners, lean against stationary objects or circle toward the affected side. Facial paralysis and inability to control balance are also observed.

Diagnosis

Isolation of **L. monocytogenes** from brain and aborted placenta and fetus; occasionally from the spinal fluid, nasal discharge, urine, feces and milk of clinically ill ruminants.
Differential Diagnosis includes nervous ketosis in cattle, rabies and brucellosis are the list of differential diagnosis.

Treatment and Prevention

Drug Treatment

Procaine penicillin G, initially 44,000 IU/kg, IM or SC q 12 hr. for 7-14 days, then 22,000 IU/kg for 7 to 14 days
Ampicillin 5-10 mg/kg, IM, IV, or SC, q 8-12 hr. **Or**
Amoxicillin 4-7 mg/kg, IM q 12-24 hr. **Or**
Erythromycin2.2-4.4mg/kg, IM, **Or**
Trimethoprim-sulfadoxine 2.7mg/kg + 13.3mg/kg q 24 hr. for 5 days.

High doses are required because of the difficulty in achieving minimum bactericidal concentrations in the brain.
Public health significance: All suspected material should be handled with caution because of its high zoonotic risk.

Neonatal Diarrhea

Diarrhea (Calf Scours) is common in newborn calves characterized by progressive dehydration and death, sometimes as early as 12 hours after birth. It can be caused by bacteria, viruses and protozoa as summarized in **Table 2. Below.** It is the major cause of calf mortality in Ethiopia.

Clinical Symptoms

Subacute form: Diarrhea may persist for several days and result in malnutrition and emaciation. This form is common in dairy calves.

Acute form: The major clinical signs are diarrhea, dehydration, profound weakness and death within one to several days of onset. Clinical signs depend on the etiology.

Table 2: Differential diagnosis of diseases of calves characterized by diarrhea

Aetiology	Age	Clinical signs	Prognosis
Enterotoxigenic **E.coli**	<3-5 months old, rarely up to 3 weeks.	Sudden onset, profuse amount of liquid faeces (pale yellow to white), cold clammy skin, pale mucosa, wet mouth.	Poor.
Salmonella spp.	At least 4 days old.	Foul smelling feces that contain blood, fibrin and copious amounts of feces, septicemia, high fever (40.5-41.5°C), depression and coma.	
Hemorrhagic enterotoxemia due to **C. perfringens** type B and C	Few days old that have voracious appetite.	Acute onset of depression, weakness, bloody diarrhea and abdominal pain.	The calf dies within hours if not detected early.
Rota & corona virus and other viral infection	5-15 days old and even several months old calves.	Large volume of feces containing mucus.	
	5-35 d old calves, commonly 2nd week.	Persistent diarrhea that does not respond to therapy.	
Dietary diarrhea	<3 weeks old.	Voluminous feces of pasty gelatinous consistency, emaciation at later stages.	

Diagnosis

Differential diagnosis of calf scours caused by infectious agents are given in **Table 2 above.**

Treatment and Prevention

Non Drug treatment

The amount of electrolytes needed depends on the extent of dehydration.

For less severe cases (6% body weight loss), electrolyte therapy with fluid containing sodium, glucose, glycine or alanine, potassium and either bicarbonate or citrate or acetate as a bicarbonate precursor by stomach tube or nipple. Milk should not be withheld for >24 hours.

For more severe cases (≥8% of the body weight loss), IV fluid and electrolyte therapy given as follows:-

Initial

Sodium bicarbonate 13% in isotonic solution, at 100 ml/kg over 4-6 hr. 25-50g of dextrose may be added to the solution

Maintenance

A combination of sodium bicarbonate solution and physiological BES, 5-8 ml/kg/hr. IV for the next 20 hr

Plus alterations of the diet.

Drug Treatment

Erythromycin,2.2-4.4mg/kg, IM **Plus** Dexamethasone 20-200mcg/kg, IM, or 10-30mg/animal, PO. **Or**
Trimethoprim-sulfadoxine 2.7mg/kg + 13.3mg/kg q 24 h for 5 days plus Dexamethansone 20-200mcg/kg, IM, or 10-30mg/animal, PO
Enrofloxacin 5mg/kg, SC, q 24 h for 3-5 days

Prevention

Isolate sick animals or maintain good hygiene of the farm/barn
Newborn calves should consume ≥5% of their body wt. of high-quality colostrum within 6 hr. of birth, followed by equivalent amounts q 12 h for 2 day
Provide the dam and neonate with good nutrition
Vaccinate the dam 6 and 3 weeks before parturition.

Antibiotic treatment of diarrhoea caused by E.coli is not advisable but should be indicated if there is bacteremia, navel infections or infectious arthritis. Combined treatment with antimicrobials plus immunoglobulin and antidiarrheal adsorbent drugs such as activated charcoal, combination of Kaolin and Pectin.

Salmonellosis

Salmonellosisis a bacterial disease caused by many serotypes of salmonellae and characterized clinically by one or more of three major syndromes septicemia, acute enteritis and chronic enteritis in young calves. Stress factors precipitate clinical disease.

Clinical Symptoms

The clinical signs include acute enteritis without extensive systemic involvement which is more common in adults as well as in young animal's ≥1 wk old. Initially, there is fever (105°–107°F [40.5°–41.5°C]), followed by severe watery diarrhoea, sometimes dysentery and often tenesmus. The feces may have a putrid odor. Persistent diarrhoea and unthriftiness characterize chronic cases.

Diagnosis

The clinical syndromes usually are characteristic. Serological tests and isolation are confirmatory.

Treatment and Prevention

Drug Treatment

Trimethoprim-sulfadiazine 30 mg/kg, PO for 3-5 days. **Or**
Enrofloxacin 5mg/kg, SC, q 24 hr. for 3-5 days **Or**
Gentamicin3.5 mg/kg, IM, q 8 hr. for 3-5days. **Or**
Ampicillin15 mg/kg q12 h, IM or 25 mg/kg PO (calves) for 3-5 days.
Lincomycin Hydrochloride: 5mg/kg intramuscularly twice daily for the first day followed by once daily for 2-4 days.

Plus

Good nursing care, good hygiene and if possible, separation of the sick from healthy animals.

Prevention

As a preventive measure, carrier animals should be isolated and culled or treated vigorously. Clean contaminated buildings, dispose contaminated materials and minimize stress in outbreaks. A strict farm management program should be introduced.

Tetanus

Tetanus toxemia is caused by a specific neurotoxin produced by **Clostridium tetani** growing in necrotic tissue.

Clinical Symptoms

The signs progress from stiff gait, prolapse of the third eyelid and trismus (lockjaw) extending to the head, neck and all four extremities, and the tail. Other signs include exaggerated response to external stimuli, erection of the ears and drooling of saliva. As the disease progresses, tetanic convulsions, accompanied by opisthotonus occur following external stimuli.

Diagnosis

It is usually made by its typical clinical signs.

Treatment and Prevention

Non Drug Treatment

Wound debridement, keep animals in the dark, quiet place, observe if bloat occurs and treat accordingly and fluid and electrolyte therapy.

Drug Treatment

Procaine penicillin G 40,000 IU/kg, q 24 hr. IM or 25,0001/kg q 12 hr. for 3-5 days followed by q 24 hr. for another 5 days.

Plus

Acetylpromazine 0.05 mg/kg, IM, q 12 hr. **Or**

Xylazine 0.05-1 mg/kg, IM or 0.016 to 0.034 mg/k IV q 12 hr. until severe signs subside (10-12 days)

Plus

Tetanus antitoxin 1500 IU, SC, q 24 hr. for 3-5 days

Prevention

As preventive measures clean wound with antiseptics; if contamination is minimal, open wound healing is preferred.

FUNGAL DISEASES OF CATTLE

Aspergillosis

Aspergillosis is caused by a number of **Aspergillus** spp, especially **A. fumigatus** and affects almost all domestic animals and birds. It causes abortion. Systemic mycotic diseases are a result of overgrowth of fungi in hay, grain or silage feeds.

Clinical Symptoms

Aspergillosis can have different clinical presentations in cattle and most infections are asymptomatic. However, it can cause mycotic pneumonia, mastitis or cause placentitis and abortion. Mycotic pneumonia is rapidly fatal and signs include pyrexia, rapid, shallow, stertorous respiration, nasal discharge and a moist cough. Abortion in pregnant cow is the predominant outcome of infection.

Diagnosis

Culture plus agar gel double diffusion test.

Treatment and Prevention

Drug Treatment

Natamycine 0.01% solution, topical, repeat after 4-5 days and again after 14 days if required. **Or**
Fluconazole 10-14mg/kg PO or IV with two weeks between treatments **Or**
Potassium iodide 10%, PO for 1-2 weeks.

Prevention

To prevent Aspergillosis hay should be prepared to ensure dry conditions throughout storage time. Precautions should also be given to silage making and storage.
Note: Fluconazole is less irritating and has a poor tissue penetration. Do not expose animals treated with Fluconazole to direct sunlight.

Candidiasis

Candida species particularly **C. albicans** is an opportunistic pathogen resulting in systemic or local candidiasis infections in cattle and calves secondary to prolonged antibiotic or corticosteroid therapy. Mastitis and abortion also occur in cattle.

Clinical Symptoms

Calves with fore-stomach candidiasis have watery diarrhea, anorexia and dehydration, with gradual progression to prostration and death.

Diagnosis

Examination of scraping or biopsy from mucocutaneous lesions and culture.

Treatment and Prevention

Nystatin 10% ointment
Topical application of 1% iodine solution may be useful in the treatment of oral or cutaneous candidiasis,
Or

Ketoconazole 40 mg/kg q 8 hr. for two weeks
Fluconazole; Oral suspension10mg/ml.

Prevention

Prevention of candidiasis involves avoidance of excessive immunosuppression as well as discriminate use of antibiotics, which should be based on the results of culture and susceptibility whenever possible.

Dermatophytosis

Dermatophytosis is a disease of cornified epidermis, hair, horn and nails. It is caused most frequently by fungal genera **Microsporum** and **Trichophyton.** Transmission occurs through contact between animals or by contact with soil. High humidity and temperature, trauma, poor nutrition and especially crowding are the predisposing factors.

Clinical Symptoms
Infected hairs become brittle, dry and lusterless and break off. Ring-shaped lesions develop which becomes alopecic.

Diagnosis
Clinical signs are indicative; direct microscopic examination and culture of skin scrapings and hairs from the periphery of lesions are necessary to confirm diagnosis.

Treatment and Prevention
Iodophores 3% spray q 24 hr. for several weeks depending on its response. **Or**
Sodium hypochlorite 0.5% irrigation q 24 hr. for several weeks. **Or**
Sodium iodide1 g/14 kg, as 10% IV, every 7days for several weeks depending on response
Fluconazole 10mg/kg /day for 3-4 weks PO.

Mycoses

Most agents of systemic mycoses exist as saprophytes in soil, in decaying vegetation and dung, and on keratinized animal tissues. Infection is acquired by inhalation, ingestion or traumatic introduction. Mycotic diseases such as histoplasmosis, coccidioidomycosis and blastomycosis are regarded as primary systemic mycoses. Opportunistic fungi usually require a host that is debilitated or immunosuppressed. Predisposing factors include stress, metabolic acidosis and long term antibiotic treatment among others.

Diagnosis
Direct microscopic examination and/or culture from exudates and biopsy material, serological tests for mycotic diseases such as histoplasmosis, blastomycosis, cryptococcosis and coccidioidomycosis.

Treatment and Prevention
Ketoconazole 40 mg/kg, q 8 h, for 7-14 days. **Or**
Itraconazole 10-20 mg/kg, q 24-48 h for 7-14 days.
Public health significance: Fungi affecting cattle might be transmitted to humans and cause local or systemic infections.

PARASITIC DISEASES OF CATTLE

HELMINTH PARASITES

Echinococcosis

Echinococcus granulosus is a tapeworm found in the small intestine of the canid definitive host. The cyst in the intermediate host, for example in cattle, localizes in various organs (the liver and lungs) and occupies a large portion of functional tissue.

Clinical Symptoms
Clinical signs depend on the organs involved but usually no visible clinical symptoms are observed.

Treatment and Prevention
There is no specific treatment of hydatid cyst
Deworm dogs regularly.
Public health significance: It can be transmitted to humans and cause serious problem.

Eye Worms

Eye worms or Theilezia worms affect eyes of cattle and causes keratitis, including opacity, ulceration, perforation and permanent fibrosis in severe cases, particularly with **T. rhodesii** infections. The worms localize in the conjunctival sac.

Clinical Symptoms
Conjunctivitis, photophobia and keratitis are common signs. Characteristically there is chronic conjunctivitis with lymphoid hyperplasia and seromucoid exudates.

Diagnosis
Clinically, thelaziasis tends to cause chronic conjunctivitis which does not spread.

Treatment and Prevention
Non drug Treatment
Apply local anesthesia and remove the worms with forceps
Drug Treatment
Irrigation of the eyes with 50-75 ml aqueous solution of 0.5% iodine and 0.75% potassium iodide has been recommended for **T. gulosa** and **T. skrjabini**
Levamisole 15 mg/kg, PO stat, maximum dose 4.5 g **Or**
Ivermectin at 0.2 mg/kg SC stat.

Prevention
Fly control and use of insect repellants.

Fasciolosis

Fasiolosis is a parasitic disease of cattle caused by the liver parasites **Fasciola hepatica** and **F. gigantica.**

Clinical Symptoms
Anaemia, hypoalbuminaemia and submandibular edema are characteristic. The acute form is common in sheep but can sometimes occur in calves. Diarrhea may occur if complicated by the presence of **Ostertagia** species.

Diagnosis
Diagnosis can be based on clinical findings, seasonality and weather conditions and previous history of the presence of the parasite in the area. Laboratory demonstration of the egg with fecal examination technique is confirmatory.

Treatment and Prevention

Triclabendazole 9-12 mg/kg, PO stat (all stages of Fasciola). **Or**
Rafoxanide; 7.5 mg/kg, PO, stat against flukes above 4 weeks old & most nematodes. **Or**
Albendazole 10 mg/kg, PO, stat repeat after two weeks. **Or**
Oxyclozanide; 15-mg/kg PO or 30 mg/kg in feed stat. **Or**
Closantel 10 mg/kg PO, stat. **Or**
Clorsulon 1 ml/50kg (200 mcg ivermectin plus 2 mg clorsulon per kg), stat, S/C under the loose skin in front of, or behind the shoulder. Divide doses greater than 10ml between two injection sites.
Nitroxynil 10mg/ kg, SC, repeat treatment as necessary throughout the period when infestation is occurring, at intervals of not less than one month.

Prevention

Bovine Fasciolosis can be prevented by strategic deworming of animal, draining of habitat for the intermediate host snail and grazing management.

Gastrointestinal Parasitism

These are infestations of the gastrointestinal tract with nematodes, cestodes and trematodes. The common stomach worms of cattle are **Haemonchus placei, Ostertagia ostertagi** and **Trichostrongylus axeii.**

Clinical Symptoms

Ostertagia and **Trichostronglyus** infections are characterized by profuse, watery diarrhea that is usually persistent. Signs of anemia, hypoproteinemia and edema, particularly the lower jaw and sometimes along the ventral abdomen manifest these infections together with **Haemonchus** infection.

Diagnosis

In animals with poor body condition anemia and diarrhea are suggestive; confirmed by fecal examination.

Table 3: Treatment of gastrointestinal parasitism in cattle

Parasite genera	Treatment		Remark
	Type of drug	Dose	
Trichostrongylus spp **Bunostomum plebotomum** **Chabertia ovina** **Oesophagostomum spp** **Haemonchus spp** **Nematodiurus spp** **Cooperia spp**	Fenbendazole	Cattle 7.5 mg/kg; sheep and other animals 5 mg/kg	○ **Trichostronglyus axei** is not eliminated by these compounds ○ Migrating and inhibiting larvae are affected only by fenbendazole, albendazole and oxfendazole ○ All anthelmtics are administered only once
	Oxfenbendazole	Cattle 4.5 mg/kg; sheep 5 mg/kg; goats 7.5 mg/kg	
	Albendazole	Cattle and sheep: 7.5 mg/kg and for adult liver fluke 15 mg/kg	
	Tetramisole	15 mg/kg PO or SC, but should not exceed 4.5 g for cattle in a single SC or oral dose	
	Levamisole	8 mg/kg SC for cattle, sheep and goat; 10 mg/kg pour on for cattle	
	Pyrantel pamoate	25 mg/kg	
	Ivermectin	200mcg/kg, SC	
	Eprinomectin	1ml/10 kg BW (0.5 mg/kg BW), Pour-On, stat for cattle	

PROTOZOAL AND RICKETTSIAL DISEASES

Anaplasmosis

Anaplasmosis is a tick-borne protozoal disease of ruminants. Clinical bovine anaplasmosis is usually caused by **Anaplasma marginale**. Cattle are also infected with **Anaplasma caudatum** and **Anaplasma centrale**. The tick vectors of anaplasmosis include Boophilus, Dermacentor, Rhipicephalus, Ixodes, Hyalomma, Argas and Ornithodoros genera.

Clinical Symptoms

Clinical signs of Anaplasmosis include fever, progressive anemia and inappetence. Loss of coordination, breathlessness when exerted and a rapid bounding pulse are usually evident in the late stages. Pregnant cows may abort. In animals <1 yr. old anaplasmosis is usually subclinical, in yearlings and 2-yr-olds it is moderately severe and in older cattle it is severe and often fatal.

Diagnosis

Microscopic examination of Giemsa-stained thin and thick blood films. Other tests include complement fixation or card agglutination tests.

Treatment and Prevention

Non-drug treatment

Supportive therapy may be necessary in dehydrated or anemic animals.

Drug Treatment

Oxytetracycline 20 mg/kg, IM, once, to eliminate carrier state two doses with a 1-wk interval. **Or**
Imidocarb dihydrochloride 1.5mg/kg SC once. **Or**
Imidocarb dipropionate at 3.0 mg/kg and high dose 5 mg/kg, IM or SC is required for elimination of the carrier state at 2 weeks interval.

Prevention

Anaplasmosis can be prevented by tick control

Note: Prompt administration of tetracycline drugs (tetracycline, oxytetracycline, doxycycline,) in the early stages of acute disease (e.g. PCV >15%) improves survival.

Babesiosis

Babesiosis is a tick-borne disease of cattle caused by intraerythrocytic protozoan parasites of the genus **Babesia**. The two important species in cattle **B. bigemina** and **B. bovis** are widespread in Ethiopia. The major vectors for **B. bigemina** and **B. bovis** are **Rhipicephalus microplus** (formerly **Boophilus microplus**) and in some areas, **R. annulatus** (formerly **Boophilus annulatus**).

Clinical Symptoms

Clinical signs usually appear 2-3 weeks after a bite from an infected tick. High fever (41°C) that lasts 4-12 days, icterus and sunken eyes, accompanied by anorexia, weakness, trembling, dyspnea and tachycardia are the major symptoms of babesiosis. In the terminal stages, severe jaundice and dark red urine that produce stable froth is observed.

Diagnosis

History and clinical signs of jaundice with hemoglobinuria and fever are suggestive; thick and thin Giemsa-stained blood smears is confirmatory. Serological diagnosis (CFT, indirect haemagglutination test, Agar gel diffusion test, rapid card or tube or latex agglutination tests)

Table 4: Drug therapies for treatment and prophylaxis of Babesiosis in cattle

Drug	Route	Toxic level mg/kg	Dosage (mg/kg)		Precautions
			B. bigemina	B. bovis, B. divergens	
Quinuronium (sulfate), 50%	SC	15	0.5-0.75	1	Low safety index; antidote in case of toxicity: adrenalin and Ca-borogluconate.
Amicarbalide 50%	IM	60 mg/kg		10-15	
Treatment			4-8	Not possible	
Sterilization			8-12	10	
Premunition			4		
Phenamidine, 40%,	IM	22.5mg/kg	10-15	-	
Diminazene					Phenazone is commonly added to reduce side effects.
Treatment	IM	25	2-4	5-6	
Sterilization	IV	10	7-10	Not possible	
Premonition			2	5	
Imidocarb					The drug should be given at least 2 months before slaughter.
Treatment	IM/SC	30	0.5-1	1-2	
Prophylaxis and premonition	2	2; q 12 wks	2; q 6 wks		
Sterilization			2	2-5	

Supportive treatment

Blood transfusions may be lifesaving in very anemic animals. Anti-inflammatory drugs, such as phenylbutazone, help relieve the inflammatory processes that occur, particularly with **B. bovis** infections, Vit. B 12 can be given.

Note: Imidocarb is the drug of choice for bovine babesiosis caused by **B. bigemina, B. bovis**, **B. divergens** and **B. caballi.** At a dosage of 3 mg/kg, imidocarb provides protection from babesiosis for 4 weeks and will also eliminate B. bovis and B. bigemina from carrier animals.

Precaution: Use the solution within 5 days of preparation (up to 14 days if refrigerated).

Besnoitiosis

Besnoitiosis is a protozoan disease of the skin, subcutis, blood vessels, mucous membranes and other tissues. Cutaneous disease in cattle is caused by **Besnoitia besnoiti**. Severely affected bulls can become permanently sterile. Affected animals remain carriers for life.

Clinical Symptoms
Infected cattle often show no clinical signs other than a few cysts in the scleral conjunctiva. Illness begins with fever followed by warm, painful swellings ventrally (anasarca). Swollen lymph nodes, diarrhea, inappetence, photophobia, rhinitis and orchitis also are seen. The skin becomes hard, thick, wrinkled and develops cracks.

Diagnosis
Cysts in the scleral conjunctiva and nasal mucosa are diagnostic.

Treatment and Prevention
Drug Treatment
Oxytetracycline 10 mg/kg IV or IM q 24 hr.

Prevention
Affected animals should be isolated and treated symptomatically.
Reduction of biting insects and ticks may also reduce transmission.

Coccidiosis

Coccidiosis is a protozoan disease of cattle caused by the genus **Eimeria**. The most common species affecting cattle include **E. bovis**, **E zuernii**, and **E auburnensis**. The disease is common in young cattle up to two years old.

Clinical Symptoms
Watery diarrhea, with little or no blood and shreds of epithelium and mucus. Calves may appear unthrifty and have soiled rear quarters. Animals that develop fever; become anorectic, depressed and dehydrated and lose weight. **Eimeria zuernii** is highly pathogenic and cause bloody diarrhea. Animals may die from secondary bacterial complications or concurrent infections (e.g. corona virus infection).

Diagnosis
Demonstration of the parasite in feces of clinically affected animals

Treatment and Prevention
Drug Treatment
Amprolium10 mg/kg, q 24 hr. PO for 5 days. **Or**
Sulfamethazine 50-110 mg/kg, q 24 hr. PO for 4 days. **Or**
Decoquinate;167g/10kg of feed for 28 days

Prevention
Diclazuril, 1 mg/kg (1 ml/2.5 kg) PO, stat, at 14 days after moving into a potentially high risk environment.
Or
Amprolium 5-10 mg/kg q 24 hr. for 21 days, PO, Monensin,1 mg/kg in feed for 30 days, PO. **Or**
Monensin 1 mg/kg in feed for 30 days
Good housing and ventilation, avoid mixing different age groups of calves and avoid fecal contamination of feeds.

Heartwater

Heartwater (cowdriosis) is an infectious, noncontagious tick-borne disease of ruminants caused by the **rickettsiae** spp. **Ehrlichia ruminantium** formerly called **Cowdria ruminantum.** Heavy losses reported in exotic cattle breeds in Ethiopia. Acute form occurs mainly in cattle between 3 and 18 months old.

Clinical Symptoms
The symptoms for acute cases of Heartwater include fever (40°C), cessation of rumination, inappetence, petechiae on the mucous membrane of the conjunctiva, lacrimation, convulsion, sudden death and/or nervous symptoms such as depression, a high-stepping stiff gait, exaggerated blinking of eyes and chewing movement and terminate in convulsions and prostration. The signs in subacute cases are less marked and CNS involvement is inconsistent.

Diagnosis
History or epidemiology, presence of Amblyoma ticks and clinical signs are suggestive. Giemsa stained brain impression smears are confirmatory.

Treatment and Prevention
Oxytetracycline 10 mg/kg IV, q 12-24 h; long acting formulation, q 48-72 h, 2 times; **Or**
Doxycycline 2 mg/kg q 24 h, IV.

Prevention
Tick control but maintain enzootic stability.

Note: Treatment is effective only in early febrile stages before neurological signs develop. Affected animals must be kept quiet in a cool area with soft bedding totally undisturbed; any stimulation can lead to a convulsive episode and subsequent death.

Theileriosis (East Coast Fever)

Theileriosis is a devastating protozoan disease of cattle prevalent in East Africa caused by **Theileria parva**. The parasite is transmitted by **Rhipicephalus appendiculatus**. The disease and its vector do not exist in Ethiopia; however since it is prevalent in other East African nations such as Kenya and Uganda similar clinical manifestations have to be investigated thoroughly.

Clinical Symptoms
High fever (41-42°C) for up to 3 weeks, generalized adenitis, epistaxis and lacrimation, leukopenia, anemia are common. Other signs include pulmonary edema, ruminal atony and alternate constipation and diarrhoea. Abortion and agalactiae may appear early in pregnancy or in lactating cows.

Diagnosis
History and clinical symptoms are indicative. Lymph node and blood smear examination are needed as confirmatory

Treatment and Prevention
Drug Treatment
Imidocarb2.5 mg/kg, 12% IM or SC, stat. **Or**
Parvaquone10 mg/kg, IM, q 48 h, two treatments **Or**
Buparvaquone 2.5 mg/kg, IM, single dose.

Prevention
Strategic tick control.
Note: These drugs do not sterilize infection and recovered animals remain carriers.

Trypanosomosis

Trypanosomosis is a chronic disease of cattle caused by protozoa of the genus **Trypanosoma.** Depending on the species of parasite, the organisms are transmitted cyclically by Tsetse flies of the genus **Glossina** or mechanically by tsetse or other biting flies. The disease is the most economically damaging and widely distributed in most parts of Ethiopia.

Clinical Symptoms
Intermittent fever, anemia and weight loss; high mortality, especially if there is poor nutrition or other stress factors.

Diagnosis
In an endemic area, anemic animal in poor condition is suggestive. Confirmation depends on demonstration of trypanosomes in stained blood smears or wet mount

Treatment and Prevention

Table 5: Chemotherapy of trypanosomosis

Drug	Preparation	Trypanosome	Main Action
Diminazene aceturate	1.05 g sachet dissolved in 10 ml of sterile distilled water.	**T.vivax, T.congolense, T.brucei.**	Curative (with the possible exception of brucei).
Homidium bromide (Ethidium bromide)	250 mg tablets.	**T.vivax, T.congolense, T. brucei.**	Curative.
Homidium chloride (Novidium chloride)	250 mg tablets.	As for the bromide salt.	
Isometamidium (samorin)	1 g or 125mg sachet.	**Vivax, congolense.**	Curative and prophylactic.
Prothidium Amicarbalide		**Vivax, congolense.**	Curative and prophylactic.

Prevention/Control
Control of tsetse flies includes frequent spraying and dipping of animals (Spray mobile targets) (e.g. Pour-on on cattle)
Spraying insecticides on fly-breeding areas, bush clearing and other methods
Insecticides-impregnated screens (fixed targets).
Precautions: The problem of drug resistance must be carefully monitored by frequent blood examinations for trypanosomes in treated animals.

ECTOPARASITES IN CATTLE

Demodex

In bovine, demodex mange causes pea-sized nodules on the skin each containing caseous material and several thousand mites. It damages the skin extensively. Transmission occurs during suckling. The muzzle, neck, withers and back are common sites.

Treatment and Prevention

Drug Treatment

Pour-on organophosphates acaricide.

Ivermectin 200 mcg/kg, stat.

Leech

Leech is an external parasite characterized by a cylindrical or slightly flattened body with suckers at either end for attaching to prey. Some are permanent parasites of man, horses, cattle, fish and mollusks, but most are merely predatory. The salivary secretions of the leech contain hirudin, an anticoagulant. Leeches may lodge as parasites in animals and humans while drinking water.

Treatment and Prevention

Manual removal.

Precaution: Rubber gloves and protective clothing should be worn.

Lice infestation / Pediculosis

Pediculosis in cattle infests the head, neck, shoulders, back and rump and occasionally the tail switch. **Damalina** species are biting lice; **Linognathus** and **Solenopotes** are sucking lice. In heavier infestations, pruritis, with rubbing and licking of the body, anemia and weakness occur. Pediculosis in cattle can decrease weight gain and milk production, result in weight loss and cause hide and hair damage.

Treatment and Prevention

Organophosphate insecticides

Ivermectin 200 mcg/kg, SC

Moxidectin 5.0 mg + Triclabendazole 200.0 mg per ml: 0.5 mg moxidectin/kg BW and 20 mg triclabendazole/kg BW (1 ml of solution for 10 kg) and as a single topical application.

Sarcoptes

Sarcoptes mange is caused by **Sarcoptes scabiei var. bovis** and it is a highly contagious disease spread by direct contact between infested and naive animals or by contaminated fomites. It is common in medium altitudes of Ethiopia.

Clinical Symptoms

Early lesions are characterized by the presence of small red papules and general erythema of the skin. The affected area is intensely itchy and frequently excoriated by scratching and biting. Loss of hair, thick brown scabs overlying a surface, this is followed by thickening and wrinkling of surrounding skin. In cattle the lesion commences on the inner surface of the thighs, the underside of the neck and brisket and around root of the tail. The whole body may be involved in six weeks.

Diagnosis

Skin scrapings examined under stereomicroscope.

Treatment and Prevention

Drug Treatment

Ivermectin1%, 200 mcg/kg, SC. **Or**

Doramectin 1%, 200 mcg/kg, SC, stat. **Or**

Organophosphates acarcides such as phosmet which is repeated after 14 days, if needed.

Macrocyclic lactone (ivermectin, eprinomectin, moxidectin and doramectin) are the preferred products for treatment of sarcoptic mange.

Psoroptes

Psoroptic mange in cattle is caused by infestation with **Psoroptes ovis. P ovis** is a nonburrowing mite that lives on the skin surface. All stages of the mite are found on the host and transmission is through direct contact of infested and susceptible hosts.

Clinical Symptoms

Infestations are intensely pruritic, with papules, crusts, excoriations, and lichenification on the shoulders and rump initially, spreading to cover almost the entire body. Secondary bacterial infections are common in severe cases.

Diagnosis

Skin scrapings examined under stereomicroscope.

Treatment and Prevention

Pour-on organophosphates acaricide. **Or**

Ivermectin 200 mcg/kg, stat.

Ticks

Ticks are obligatory parasites that feed on blood. They transmit a large number of pathogens while others may directly cause disease due to the salivary toxins and fluids (tick paralysis). More than 40 species of ticks are found in Ethiopia.

Table 6: Some important tick genera and the main livestock diseases they transmit

Tick genus	Disease transmitted by the ticks
Amblyoma	Heart water.
Boophilus	Babesia, **Anaplasma marginale.**
Hyalomma	Tick toxicosis, many other babesial, theilerial and rickettsial infections.
Rhipicephalus	Babesia infections in domestic animals; East Coast Fever in cattle; Nairobi Sheep Disease, Tick toxicosis and many other babesial, theilerial and rickettsial infections of domestic animals.
Dermacentor	Babesia infections in horses, cattle and sheep; **Anaplasma marginale** in cattle; tick paralysis.
Ornithodoros	African Swine Fever, Relapsing fever.

Treatment and Prevention

Drug Treatment

Chlorfenvinphos spray 10% w/v Emulcifiable Concentrate (EC). Or

Diazinon spray or dip or ear tag. 15% w/v, 16.2%, 20% and 60% w/v. Or

Cypermethrin spray or dip. EC, 5%, 10% w/v; Powder, 25%; Pour-on solution, 1%, 1.25%, 2%. Or

Deltamethrin can be applied as spray or dip, pour on , ear tag, concentration and dosage as manufacturers' instruction. **Or**

Fenvalerate spray (20%) 2ml/Liter. **Or**

Permethrin as directed by the manufacturer. **Or**

Flumethrine spray as directed by the manufacturer. **Or**

Amitraz as recommended by the manufacturer.

Prevention

As a control option, all niches and crevices in affected building should be sprayed with acaricides in case of soft ticks

For hard ticks, use acaricide pour-ons or sprays or deep and combined with other control methods. Acaricide spray or dip every 21 days; however, to control all nymphs, treat every 12 days during tick season

Control of two-host and three host-ticks is by weekly dipping during tick season.

DISEASES OF THE RESPIRATORY SYSTEM

Aspiration Pneumonia

Aspiration pneumonia is a common type of pneumonia caused by aspiration of foreign material into the lungs. The most common causes of aspiration pneumonia are faulty administration of medicines or other supplement and especially if the tongue is pulled out, and the head is held high or during coughing or bellowing. Other predisposing causes include aspiration of vomitus, anaesthetized or comatose animals, vagal paralysis, acute pharyngitis, abscesses or tumors of pharyngeal region and cleft palate. It is common in Ethiopia because most cattle owners attempt to drench traditional medicines before consulting an animal health professional.

Clinical Symptoms

A purulent nasal discharge which sometimes is tinged reddish brown or green. Occasionally, evidence of aspirated material, e.g. oil droplets, green herbs can be seen in the nasal discharge or expectorated material. Fluid sounds over one or both sides of the chest, followed by wheezing sounds, pleuritic friction rubs and sometimes crackling sounds of subcutaneous emphysema; toxemia may occur if ruminal fluid is aspirated.

Diagnosis

History of the animal within the last 2-3 days, auscultation and clinical signs are indicative.

Treatment and Prevention

Non- Drug Treatment

Saline nebulization and coupage may assist with generating a productive cough to facilitate clearance of the aspirated material.

Drug Treatment

Ampicillin 15 mg/kg q12 h, IM or 25 mg/kg PO (calves) for 3-5 days. **Or**

Procaine penicillin G, initially 44,000 IU/kg, IM or SC q 12 h for 7-14 days, then 22,000 IU/kg for 7 to 14 days. **Or**

Tetracycline; 10-15 mg/kg, IM, q 12 h, for 3-5 days. **Or**

Florfenicol 40 mg/kg and flunixin 2.2 mg/kg (2 mL/15 kg), SC, Stat. The dose volume given at any one injection site should not exceed 10mL

Flunixin Meglumine 1.1mg/Kg q 8-12 h IV or IM.

Bovine Respiratory Disease Complex

Bovine Respiratory Disease Complex (BRD) is a multifactorial disease. The most common viruses involved in BRD include Bovine Viral Diarrhea (BVD), Infectious Bovine Rhinotracheitis (IBR), Bovine Respiratory Syncytial Virus (BRSV) and Parainfluenza Type-3 Virus (PI-3). The common bacteria found in the lungs of cattle with BRD include **Mannheimia haemolytica** and **Pasteurella multocida.**

Clinical Symptoms

High fever (42.2°C), nasal discharge and as the disease progresses inappetence, dyspnea with extended head and open mouth breathing are observed. The distribution of the lesion is usually anteroventral.

Treatment and Prevention

Drug Treatment

Oxytetracycline hydrochloride 11 mg/kg, SC q 24hr. for 3 days; LA formulations, 20 mg/kg, IM, q 48 hr. continued for at least 2 days after the rectal temperature has returned to the normal range. **Or**
Procaine penicillin G, 22,000 IU/kg, IM or SC q 24 hr. for 3 to 5 days or benzathine penicillin or other repository preparations q 48-72 hr. **Or**
Amoxicillin trihydrate11 mg/kg, IM, SC q 24 hr. for 5 days. **Or**
Sulfadimethoxine: **Initial dose:** 60 mg/kg; **maintenance dose:** 30 mg/kg, IM, q 24 hr. for 3-4 days. **Or**
Spectinomycin dihydrochloride pentahydrate, 33 mg/kg, SC, q 8 h for 5 days. **Or**
Tylosin44 mg/kg, IM, q 24 hr. for 5-7 days. **Or**
Ceftiofur sodium 2.2 mg/kg IM, SC q 12 hr. **Or**
Gamithromycin: 6mg/kg (1 ml/25 kg) into the neck SC stat. cattle over 250 kg divide the dose so that no more than 10 ml at single site. **Or**
Tildipirosin: 4 mg/kg into the neck SC stat. Do not inject more than 10 ml per injection site. **Or**
Tulathromycin: 2.5 mg/kg (1ml/40 kg BW) into the neck SC stat. For treatment of cattle over 300 kg bodyweight, divide the dose so that no more than 7.5 ml are injected at one site.

Contagious Bovine Pleuropneumonia (CBPP)

Contagious Bovine Pleuropneumonia (CBPP) is a highly contagious pneumonia of cattle is caused by **Mycoplasma mycoides** subspecies **mycoides** (small colony type) and transmitted to susceptible cattle by aerosol. The disease is prevalent mainly in pastoralist areas of Ethiopia with up to 10% prevalence.

Clinical Symptoms

Fever (41.5°C), anorexia, difficult breathing, grunt at expiration, cough when forced to move, standing with the elbows apart, arched back and extended head. A quarter of the animals may recover but remain carriers and 50% die of infection. Subclinical cases occur and may be important sources of infection.

Diagnosis

Clinical signs, complement fixation test and characteristic marble appearance of the lung in dead animals.

Treatment and Prevention

Tylosin10 mg/kg, IM, q 12 hr. for 3-5 day **Or**
Oxytetracycline 10 mg/kg, IM, for 5 days.

Treatment is not recommended because animals remain carriers after treatment; however treatment could be attempted in valuable animals.

Enzootic Pneumonia

Enzootic pneumonia is primarily a problem in calves <6 months old but may occur in calves up to 1 year of age. It is more common in housed dairy calves than those raised outside. Peak incidence of disease may coincide with decline of passively acquired immunity. Morbidity rates may approach 100%; case fatality rates are variable but can reach 20%. The etiology is similar to Bovine Respiratory Disease complex (**See** Diseases of Cattle: Bovine Respiratory Disease Complex above).

Clinical Symptoms and Diagnosis
Depends on individual viral and bacterial etiologies.

Treatment and Prevention
There is no effective drug treatment

Prevention
Vaccinate cows against specific respiratory viruses and bacteria 3-4 weeks prepartum to improve the quality of colostral antibodies
Calves should receive quality colostrum at 8-10% of body weight in the first 12 hours after birth
Group calves of similar age with all-in all-out management system
Proper housing, adequate ventilation, and good nursing care are also important.

Pneumonic Pasteurellosis (Shipping Fever)

Shipping Fever is a respiratory disease of cattle of multifactorial etiology. It is associated with infection by **Mannheimia [Pasteurella] haemolytica**, **Pasteurella trehalosi** and, less commonly, **P. multocida** or **Haemophilus somnus** and in chronic pneumonia with pulmonary abscessation, **Arcanobacterium pyogenes** is frequently isolated.

Clinical Symptoms
Depression, toxemia, pyrexia (40-41°C), serous to mucopurulent nasal discharge, moist cough, a rapid shallow respiratory rate, increased bronchial sounds, crackles and wheezes. In severe cases, pleurisy may develop, which is characterized by an irregular breathing pattern and grunting on expiration, mucoid or purulent nasal discharge. The animal may become unthrifty in chronic cases.

Diagnosis
Microbiological culture from the lower respiratory tract by tracheal swab, transtracheal wash or bronchoalveolar lavage.

Treatment and Prevention
Treatment is similar to other bovine respiratory diseases.

Prevention
Mass prophylactic medication with antibiotics before stress with Oxytetracyline (LA) 20 mg/kg, IM q 72 h, Or Tilmicosin 10 mg/kg, SC q 72 h for 3 days.
Avoid or minimize stress, provide proper housing with good ventilation.

Note: Early recognition and treatment with antibiotics is essential for successful therapy. If one treatment fails continue with other drugs.

Tuberculosis

Tuberculosis (TB) is an infectious, granulomatous disease of animals and man caused by acid-fast bacilli of the genus **Mycobacterium**. The main tubercle bacillus in cattle is **M. bovis,** though **M. tuberculosis** and **M. avium** are also involved. **Mycobacterium bovis** can cause progressive disease in most warm-blooded vertebrates including man.

Clinical Symptoms

Tuberculosis has two clinical forms: pulmonary or extrapulmonary. In the latter form, generalized signs including progressive emaciation, lethargy, weakness, anorexia and a low-grade, fluctuating fever are observed. The respiratory form of the disease causes a chronic, intermittent, moist cough with later signs of dyspnea and tachypnea.

Diagnosis

Single or comparative intradermal tuberculin test; confirmation of diagnosis requires isolation and identification of the organism, which may take 4 to 8 weeks.

Treatment and Prevention

Treatment of bovine tuberculosis is not recommended because it is not economical. However, in valuable animals: Isonicotinic acid hydrazine/isoniazid (INH), 20 mg/kg, PO q 24 h for 8 weeks, maximum is 12g for bulls and 10g/day for cows.

Prevention

For prevention keep herds free by test and slaughter of reactors.

Verminous Pneumonia

Verminous pneumonia (lungworm infection, parasitic pneumonia) is an infestation of the lower respiratory tract of cattle by a nematode parasite, **Dictyocaulus viviparous.** It is characterized by bronchitis or pneumonia, which may be aggravated by secondary bacterial pneumonia.

Clinical Symptoms

In acute verminous pneumonia, frequent bronchial cough that may be persistent, slight nasal discharge, fever (40-41°C), increased vesicular murmur and bronchial tones, dyspnea, expiratory grunt, cyanosis and death are common signs. In adult dairy cattle, milk yield drops severely and abnormal lung sounds are heard over the caudal lobes.

Diagnosis

Clinical signs, epidemiology, presence of first-stage larvae in feces using the Baerman technique.

Treatment and Prevention

Drug Treatment

Levamisole15mg/kg, PO, stat. **Or**
Fenbendazole5-15 mg/kg q 24 hr. PO for 3 days **Or**
Oxfendazole 4.5 mg/kg, PO, stat **Or**
Albendazole7.5 mg/kg, PO, stat. **Or**
Ivermectin 200 mcg/kg, SC, stat acts against all stages of D. viviparous.

Prevention

Strategic deworming and grazing management for prevention of Verminous Pneumonia.

DISEASES OF THE REPRODUCTIVE SYSTEM

Abortion

Abortion is the termination of pregnancy after organogenesis is complete but before the expelled fetus can survive. If pregnancy ends before organogenesis, it may be called early embryonic death. The causes of abortion could be infectious or non-infectious. The most important infectious causes of abortion are listed in **Table 7 below.** In additions, any disease causing high fever may also cause abortion.

Diagnosis

Diagnosis requires isolation and identification of the causative agent from uterine swab, biopsy, examination of fetus and placenta, urine and serological tests.

Treatment and Prevention

No drug treatment

A balanced nutritional program
Genetic selection and a functional record keeping system
Adequate facilities for housing, handling, and environmental control with focus on hygiene
A positive working condition between cattlemen and the veterinarian need to be established
Regular immunizations against diseases that cause abortion.

Table 7: Infectious causes of abortion in cow

Disease	Period of abortion	Clinical signs or lesions	Diagnosis	Control
Bovine Viral Diarrhea (BVD-Mucosal disease)	42 - 125 days of gestation.	Fetal death and abortion or resorption or fetal immunotolerance and persistent infection, fetal mummification or deformity.	Diagnosis is difficult because BVD does not cause specific fetal lesions. Samples: from placenta, fetal spleen or dam serum Isolation, immunologic staining, PCR or detection of precolostral antibodies in aborted calves.	Vaccination (Booster vaccination may be desirable).
Infectious Bovine Rhinotracheitis (IBR, Bovine Herpes virus 1)	≥4 months (Second half of gestation)	Infected fetus dies within 24 hr., placentitis with blanched, necrotic cotyledons and edematous, yellow intercotyledonary areas; autolysed fetus.	Fluorescent antibody on kidney, histopathology of liver and adrenal, and serology, PCR.	Vaccination (Booster vaccination may be desirable).
Leptospirosis	Last trimester, 2-6 wk. after an outbreak occurred or 7-10 days after onset of the disease.	Infertility, weak calves, fever and bloody urine; Placentitis with avascular, light tan cotyledons and edematous, yellowish intercotyledonary areas, autolysed fetus	Isolation and identification; Dark field or phase-contrast microscopy and indirect fluorescent antibody, Serum agglutination test using tube or plate procedure; Microbiological culture of the dam's Urine; PCR.	Sanitation, Vaccination, Ceftiofur Sodium 1ml/50kg (1mg/kg), IM q 24 h for 3-5 days in acute cases.

Disease	Period of abortion	Clinical signs or lesions	Diagnosis	Control
Brucellosis	Last trimester (usually around 6-9thmonth), premature birth or weak calves.	Cotyledons may be normal to necrotic and red or yellow: The intercotyledonary area is focally thickened with a wet, leathery fetus may be autolysed with broncho-pneumonia; retention of fetal membrane.	Maternal serology (serum agglutination test plus CFT); Brucellosis ring test (BRT) used as screening for dairy herd.	AI and maternity hygiene to prevent exposure, calf hood vaccination; test and slaughter.
Mycotic Abortion	≥ 4 months	Cotyledons are enlarged and necrotic with turned-in margins. The intercotyledonary area is thickened and leathery. Adventitious placentation fetus seldom is autolysed.	Hyphae associated with fetal dermatitis (especially eyelids), bronchopneumonia, abomasal contents and placental lesions.	Moldy feed should be avoided.
Campylobacteriosis	Embryonic mortality and early abortion (2-3months)	Venereal transmission and repeat breeding, mild fibrinous pleuritis and peritonitis , bronchopneumonia, mild placentitis with hemorrhagic cotyledons and an edematous intercotyledonary area, Infertility.	Dark field microscopy of abomasal contents or culture of placenta or abomasal contents, histopathology is also helpful. Abortion is sporadic.	AI and vaccination 2 months before breeding; 60-90. Days sexual rest; uterine infusion with 0.5-1gm of streptomycin 24 hrs. after AI to restore fertility.
Listeriosis	Late abortion (4-7th month)	Sporadic abortion. RFM, metritis, keratoconjunctivitis and encephalitis. The fetal liver is shrunken and gray and contains pinpoint micro abscesses. Necrosis of the cotyledons and intercotyledonary area.	Culture of **Listeria** from fetus or placenta.	Sanitation, antibiotic therapy.
Chlamydiosis	Sporadic abortion ≥ 4 months	Placentitis, fetal pneumonia and hepatitis	Stained smears of cotyledons tissues, culture in embryonating chicken eggs.	Ovine chlamydial vaccine.
Toxoplasmosis	Late abortion, still birth	RFM, premature birth, weak young; Necrotic areas of the placentomes	Isolation of the organism or serologic test, Sabin-Feldman dye test for serology.	Prevent ingestion of contaminated placenta (break the infection cycle).
Neosporosis	All stages of gestation	Congenital infection, paralysis, stunting, infected calves may be symptom free.	Histologic and immunohistochemical tests on the brain of aborted fetus, IFAT and ELISA.	Test aborting cows and cull; eliminate exposure.

Disease	Period of abortion	Clinical signs or lesions	Diagnosis	Control
Trichomoniasis	Early abortion	Symptoms similar to Campylobacteriosis, Pyometra, sterility discharge containing pus, placentitis, with hemorrhagic intercotyledonary areas.	Direct microscopy of vaginal/uterine discharge, detection in abomasal content.	Sexual rest (90days), breed through AI, slaughter infected bulls, vaccination in herds using natural mating.

Bovine Mastitis

Mastitis is an inflammation of the mammary glands. It is caused by bacterial or mycotic pathogens. **Staphylococcus aureus, Streptococcus agalactiae, Str. uberis, Str. dysgalactiae,** other streptococci, **Arcanobacterium pyogenes, Mycoplasma spp, Nocardia asteroidesasteroids** and coliforms are the most common agents.

Clinical Symptoms

Clinical mastitis is manifested by inflammation of the udder and often accompanied by abnormal milk secretions. The signs depend on the organisms involved. Systemic signs could also be observed. Subclinical mastitis is the most common form.

The clinical signs are usually non-specific, but the following gives a clue for the type of agent involved.

Table 8: Causes of bovine mastitis

Microorganism	Clinical findings
Staphylococcus aureus	Severe swelling, purulent milk with clots.
Mycoplasma species	Drop in milk production, infection of all quarters simultaneously.
Arcanobacterium pyogenes	Profuse foul-smelling, purulent discharge.
Mycoplasma bovis	Rapid onset.

Diagnosis

Is based on clinical signs, isolation and identification of the causative pathogen. Tests to detect subclinical mastitis include California Mastitis Test (CMT) or direct somatic cell count.

Treatment and Prevention

Drug Treatment

Intramammary infusion, q 48 hr. repeated 3 times, applied separately into every quarter. Each tube of intramammary infusion should contain one of the following:

Benzathine Cloxacillin, 500mg/quarter for 3 days; **Or**

Erythromycin 300 mg/quarter for 3-5 days. **Or**

Streptomycin plus penicillin (1g + 100,000 IU per quarter for 3-5 days. **Or**

Neomycin 500 mg per quarter for 3 days

Lincomycin: 330 mg + Neomycin: 100 mg per 10 ml Intramammary Solution syringe/ quarter for 3 days

Amoxicillin: 200 mg + Clavulanic acid: 50 mg +Prednisolone: 10 mg/ 3 g suspension syringe.

Procaine benzylpenicillin (100mg) + Streptomycin Sulphate (100mg) + Neomycin Sulphate (100mg) + Prednisolone (10mg) syringe/quarter once daily for 3 day

Systemic treatment for peracute or acute mastitis

Procaine penicillin G 22,000 IU/kg, aqueous suspension, IM or SC q 24 h for 3 to 5 days. **Or**

Amoxicillin or Ampicillin 10 mg/kg, q 24 hr. IM. **Or**

Benzathine penicillin G or a similar repository preparation, q 48-72 hr. **Or**

Long acting penicillin preparation before drying off **Or**

Erythromycin 12.5 mg/kg, q 24 hr. IM. **Or**

Oxytetracycline 10 mg/kg, q 24 hr. IV. **Or**

Sulfonamides 200 mg/kg, q 24 hr. PO. **Or**

Potentiated Sulfonamides 48 mg/kg, q 48 hr. IM. **Or**

Penethamate: 15 mg /kg (5.5ml/100 kg BW) for 3 consecutive days.

Prevention
Disinfection of the teat before and after milking
Proper house hygiene
Routine checkups of the udder and dry cow therapy with long acting penicillin preparation or single dose combination of 500mg cloxacillin and 250mg ampicillin or Cefalonium 250 mg Intramammary Suspension /3g syringe should be applied as a preventive measure.

Dystocia

Dystocia refers to difficult birth. It may result from maternal or fetal causes. Maternal causes include myometrial defects such as uterine atony, metabolic abnormalities such as hypocalcemia, inadequate pelvic diameter, insufficient dilation of the birth canal, while fetal causes include fetal hormone (corticosteroid) deficiency, fetal oversize, fetal death or abnormal fetal presentation and posture. Dystocia is considered if the animal had previous history of the condition and parturition does not occur within 24 hours after onset of labor and drop in rectal temperature (to <37.7°C) or if gestation is prolonged.

Treatment and Prevention
Determine the cause of dystocia and the condition of the animal before treatment commences.
Manual correction of abnormal position of the fetus
Surgical removal (Caesarian operation)
Denaverine hydrochloride 40.0 mg /ml: 10.0 ml/animal (400 mg / animal).

Prevention
Cull heifers with very small pelvic area
Follow proper feeding and maintain good nutritional condition of pregnant animals

Follicular Cystic Ovary Disease

Cystic ovary disease (Follicular cysts, Cystic follicles, Nymphomania "Bulling") primarily affects dairy cattle. It commonly occurs within 3-8 weeks of parturition. In animals developing cystic ovary disease, ovulation fails to occur and the dominant follicle continues to enlarge.

Clinical Symptoms
Cows may show signs ranging from constant estrus activity to nearly complete anoestrus. Behavioral change ranges from frequent, intermittent estrus with exaggerated monosexual drives to bull like behavior, including mounting, pawing the ground and bellowing. Relaxation of the vulva, perineum and the large pelvic ligaments, which causes the tail head to be elevated is common in chronic cases.

Diagnosis
Rectal palpation and history, conformation and uterine changes, when present ultrasound per rectum or transrectal ultrasonography could be beneficial.

Treatment and Prevention
Non Drug Treatment
Manual rupture with moderate pressure on the ovary with the finger pads against the palm of the hand; but the potential danger of traumatizing the ovary and causing hemorrhage with subsequent local adhesions should not be overlooked. Estrus is expected after 4-7 or 15-25 days.

Drug Treatment
Gonadotropin-Releasing Hormone (Gn-RH) 100mcg, IM; Cows come in estrus within 15-30 days (peak 19-23 days); few may require second or even third treatment. To hasten the onset of the first estrus after treatment, PGF2α products can be given 7 days after GnRH or hCG **Or**

Luteinizing hormone (LH) 500 mcg/animal, IM **Or**
Human chorionic gonadotropic (hCG), 10,000 units IM or 2,500 to 5,000 units IV or by intrafollicular injection of 500 to 2500 units; may be repeated in 14 days if necessary
Prostaglandins (cloprostenol, dinoprost) can be administered 9-11 days after hCG or GnRH to hasten onset of estrus.
Ablation using OPU apparatus

Note: Human chorionic gonadotropic (hCG) must be kept at 15-30°C prior to reconstitution and later on refrigerated for not more than 30 days.
Give epinephrine hydrochloride and parenteral antihistamine as antidote.

Luteal Cystic Ovary Disease

Luteal Cystic Ovary Disease (luteal cysts) is characterized by enlarged ovaries with one or more cysts, the walls of which are thicker than those of follicular cysts. Luteal cysts are accompanied by normal conformation and anestrous behavior. They are recognized as smooth, fluctuant domes protruding above the surface of the ovary. Usually, they are single structures.

Clinical Symptoms
It is accompanied by normal conformation and anestrous behavior. Follicular cysts explode under minimal pressure while luteal cysts can be ruptured with considerable pressure though it is not recommended.

Diagnosis
Signs and Ultrasonography.

Treatment and Prevention
Luteolytic doses of PGF2α like synthetic Cloprostenol, 500 mcg per animal, IM. **Or**
HCG and GnRH, 10,000 units IM or 2,500 to 5,000 units IV or by intrafollicular injection of 500 to 2500 units but the next estrus will occur from 5 to 21 days after treatment.
Note: Manual rupture of luteal cysts is not recommended.
The same procedures applied for follicular cystic ovary are also effective.

Metritis and Endometritis

It is the inflammation of the muscular and endometrial layers of the uterus. Retained fetal membranes, dystocia, trauma to the reproductive tract, abortion, concurrent systemic disease and emphysematous fetus. Unsanitary conditions at parturition can all predispose to metritis. Metritis is a much more severe disease than endometritis, requiring a different therapeutic approach.

Clinical Symptoms
Fetid uterine discharge is common; systemic signs include fever, anorexia, depression and swollen and friable uterus. The mucus character (0=clear translucent; 1= mucus containing flecks of white pus; 2= exudate containing 50% white or cream pus; 3= exudate containing >50% white, cream or bloody pus) and odour scores are summed to give an endometritis clinical score ranging from 0 to 6 (vaginal mucus odour: 0 for no odor; 6 for a fetid odor).

Diagnosis

Examination of the uterus with a speculum combined with history, visual inspection of the perineum, examination of the tail for evidence of vaginal discharge and manual or ultrasonic transrectal examination of the reproductive tract. The examination of the contents of the vagina for the presence of pus is the most useful procedure for diagnosis of uterine infection.

Treatment and Prevention

Drug Treatment

Procaine penicillin G 22,000 IU/kg, aqueous suspension, IM or SC q 24 h for 3 to 5 days or benzathine penicillin G or a similar repository preparation, q 48-72 h. **Or**
Sodium Penicillin G, 10 million IU, intrauterine infusion stat during the postovulatory period. **Or**
Ceftiofur Sodium 1ml/50kg (1mg/kg), IM q 24 h for 3-5 days. **Or**
Oxytetracycline 11 mg/kg, IV q 12 h for 3-5 days. **Or**
Tetracycline 2 to 6 g, intrauterine pessaries daily for 3 days. **Or**
Prostaglandin PGF2α or its analogs like cloprostenol, at usual luteolytic doses, for the management of endometritis
If the disease progresses to puerperal metritis, with the presence of systemic signs such as dehydration and pyrexia, fluid therapy (4-5ml/4-5minute) to restore normal hydration level and antipyretic (flunixin meglumine at a rate of 2.2mg/kg) drugs for 1 to 3 days may be beneficial
Infusion of the uterus with warm water or saline (1 L) to which 1gm streptomycin is added at early postpartum; siphon it and repeat the procedure 2-3 times. This procedure is best applied with two way Foley catheter
Uterine lavage (Douching) with lugol's solution.
Note: Antiseptics and many antibiotics inhibit phagocytosis, thus microorganisms that are resistant to antibiotics could get favourable condition.

Pyometra

Pyometra is the accumulation of purulent exudates in the uterus accompanied by the persistence of an active corpus luteum and a closed cervix. Pyometra is also commonly accompanied by anoestrus. It is common 15-60 days postpartum and commonly a sequel to dystocia, retained fetal membranes or acute septic metritis.

Clinical Symptoms

The uterus is distended with fluid, persistent corpus luteum occurs on one of the ovaries, vulval discharge may be yellow, creamy white, grayish white, greenish gray or reddish brown in open cervix or the cervix may be closed with no visible discharge.

Diagnosis

Absence of any positive signs of pregnancy i.e. amniotic vesicle, fetal membranes, placentomes and fetus in fluid filled uterus are diagnostic.

Treatment and Prevention

Drug Treatment

PGF2α or its analogs like cloprostenol at normal luteolytic doses. No intrauterine treatment is recommended in conjunction with the prostaglandin.

Non Drug Treatment

Lavage of the uterus using large volumes of fluid is recommended, but the condition frequently recurs
Hysterectomy (see procedure for C-section)
Manual removal of the corpus luteum.
Precautions: Excessive hemorrhage and adhesion of the ovary may occur.

Retained Fetal Membranes (RFM)

Retention of fetal membranes usually is defined as failure to expel fetal membranes within 24 hours after parturition. The incidence is increased by abortion, dystocia, hypocalcaemia, twin birth, high environmental temperature, advancing age of the cow, premature birth or induction of parturition, placentitis, ducts infection and nutritional disturbances.

Cows with retained fetal membranes are at a higher risk of developing metritis, ketosis, mastitis and even abortion in a subsequent pregnancy. Cows that have once had retained fetal membranes are at increased risk of recurrence at a subsequent parturition.

Clinical Symptoms

Discolored membranes are seen hanging from the vulva after 24 hours. Occasionally there is foul smelling discharge, inappetence and decreased milk production. Systemic infection is not common. When systemic signs are seen, they are related to toxemia.

Diagnosis

Degenerating, discolored, ultimately fetid membranes are seen hanging from the vulva >24 hours after parturition.

Treatment and Prevention

Drug Treatment

Procaine penicillin G 22,000 IU/kg, aqueous suspension, IM or SC q 24 hr. for 3 to 5 days

Prostaglandin at luteolytic dose and Oxytocin at a rate of 100IU IM

Intrauterine antimicrobials (see Metritis and endometritis); however, the cost of treatment and withdrawal period should be considered

Ceftiofur Sodium 1ml/50kg (1mg/kg), IM q 24 h for 3-5 days and flunixin meglumine at a rate of 2.2mg/kg if systemic signs are observed, treat with systemic antimicrobials.

Precautions: Manual removal of the retained membranes is no longer recommended and is potentially harmful.

Trichomoniasis

Trichomoniasis is a venereal disease of cattle caused by a protozoa **Trichomonas fetus** characterized primarily by early embryonic death, prolonged breeding and infertility. The estrus cycle becomes irregular resulting in extended calving interval. Pyometra occasionally develops after breeding.

Clinical Symptoms

Symptoms similar to Campylobacteriosis and Pyometra include:-

Sterility, vaginal discharge containing pus, placentitis, with hemorrhagic intercotyledonary areas, infertility caused by death of the fetus, usually 50-100 days after conception and marked by repeat breeding and irregular estrous cycle.

Diagnosis

History and clinical signs are indicative. Isolation of **T. fetus** from the preputial fornix or dark field microscopic examination of aspirates with saline or Ringer's douching or from vaginal/uterine discharge, vaginal pus or vaginal mucus are diagnostic. Detection in abomasal content is also possible.

Treatment and Prevention

No effective treatment is available for cattle. Therefore, animals with genital abnormalities should be culled from the herd.

Use artificial insemination

For heifers, use young bulls for breeding.

Uterine Prolapse and Uterine Eversion

Prolapse of the uterus is common in dairy cows. Recumbency with the hindquarters lower than the forequarters, invagination of the tip of the uterus, excessive straining due to trauma during parturition, excessive traction to relieve dystocia or retained fetal membranes, uterine atony, hypocalcaemia, have all been incriminated as contributory causes. Prolapse of the uterus invariably occurs immediately after or within several hours of parturition.

Clinical Symptom

Eversion of the uterine horns, swelling, redness and traumatic damage to the endometrium. It must be considered an emergency case.

Treatment and Prevention

Apply epidural analgesia; Lidocaine 5-8 ml of 8-15 ml of 2% procaine
Remove the placenta (if still attached), clean the endometrial surface thoroughly, and repair any lacerations
Rubbing the surface of the uterus with glycerol and return to its normal position
Instillation of warm, sterile saline solution
Apply antibiotics
Meloxicam 0.5mg/kg IV (96hr milk withdrawal) to reduce pain and straining after replacement
Use Ketoprofen 10% at a rate of 3mg/kg injection to reduce swelling
Caslick operation to prevent relapse of prolapse.

Vaginal and Cervical Prolapse

Eversion and prolapse of the vagina, with or without prolapse of the cervix, occurs most commonly in mature cows during the last trimester of pregnancy. Predisposing factors include increased intra-abdominal pressure associated with increased size of the pregnant uterus, intra-abdominal fat or rumen distention superimposed upon relaxation and softening of the pelvic girdle and associated soft-tissue structures in the pelvic canal and perineum mediated by increased circulating concentrations of estrogens and relaxin during late pregnancy. Intra-abdominal pressure is increased in the recumbent animal.

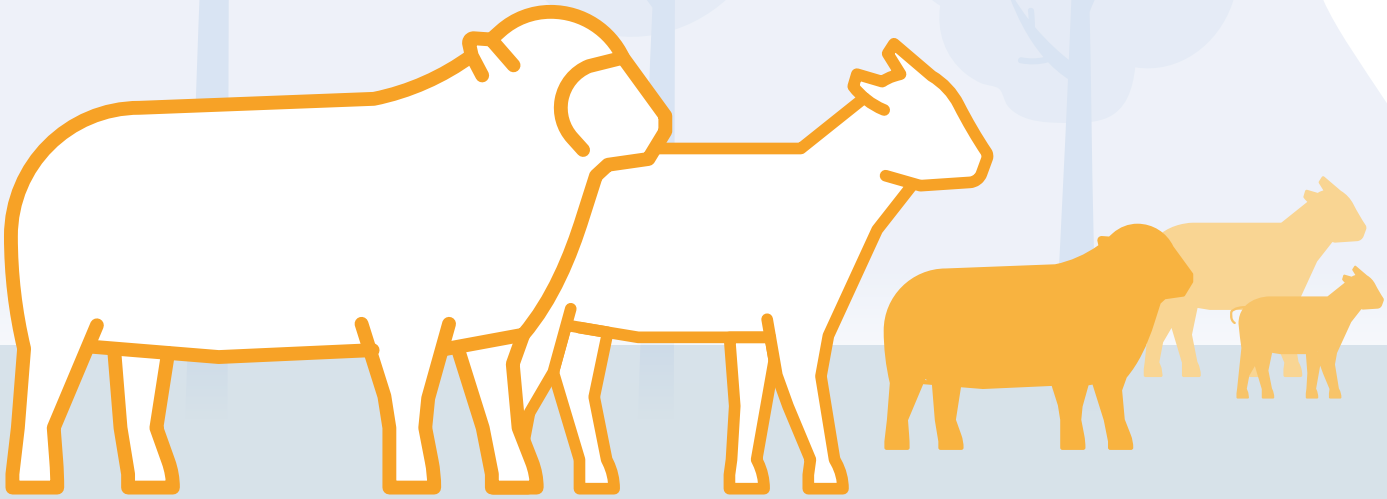
Clinical Symptoms and Diagnosis

The floor of the vagina prolapses first and repeated eversions may result in a diverticulum of one or both sides of the vagina. The cervix occasionally prolapses through the vulva.

Treatment and Prevention

Apply epidural anesthesia (see Diseases of cattle: Uterine prolapse)
Wash the vagina, empty the bladder, lubricate the vagina with glycerol and replace it to its original position
Retention is achieved by insertion of a Buhner suture or doing Caslick operation.

SECTION II



DISEASES OF SHEEP & GOATS (SHOATS)[2]

2 *Guide*

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.

NON-INFECTIOUS DISEASES OF SHEEP AND GOATS

Bloat

Bloat is an over distention of the rumenoreticulum with the gases of fermentation, either in the form of a persistent foam mixed with the ruminal contents, called primary or frothy bloat, or in the form of free gas separated from the ingesta, called secondary or free-gas bloat.

Clinical Symptoms
Distension of the left flank, protrusion of the paralumbar fossa above the ventral column and enlarged abdomen, dyspnea and grunting, mouth breathing, protrusion of the tongue and extension of the head.

Diagnosis
In frothy bloat, clinical diagnosis can be done based on history and clinical sign. The causes of secondary bloat must be ascertained by clinical examination to determine the cause of the failure of eructation.

Treatment and Prevention

Non-Drug Treatment

Remove free-gas by passing a stomach tube in case of free gas bloat
Vegetable or mineral oil at doses of 250–500 mL/animal PO via stomach tube, stat. or Poloxalene (25–50 g, PO) is effective in treating Frothy bloat (legume bloat) but not feedlot bloat.

Drug Treatment

Dioctyl sodium sulfosuccinate, 15-30 ml/head
Surgical intervention in life-threatening cases: Emergency rumenotomy or use of trocar and cannula with a bore of 2.5cm to puncture the rumen and administration of antifoaming agents e.g. vegetable or mineral oils 80-250 ml/ animal.

Prevention
Poloxalene 2-3g/animal PO
Animals should be gradually adapted to lush pastures
Grain feeding should be supplied with sufficient hay
Concentrate feeds should not be too finely ground.

Note: When drenching the oils care should be taken to avoid Aspiration Pneumonia.

Enzootic Ataxia

Enzootic ataxia (swayback) in lambs and kids is caused by copper deficiency either primary by inadequate copper intake or secondary as result of high molybdenum, sulfur and zinc in the diet which interferes with absorption of copper from intestine. In Ethiopia this condition has been reported in sheep in the rift valley region.

Clinical Symptoms
The congenital form (present at birth) is called swayback. These kids may be unable to rise or may walk with severe incoordination. They may be depressed and have muscle tremors, most die soon after birth. True enzootic ataxia is the second form of this condition, it has a delayed onset of signs. Affected kids actually appear normal at birth but start showing problems between one week and six months of age. It is characterized by paraparesis (ataxia of the forelegs), excessive flexion of the joint, atrophy of the hind limb muscle, uncoordinated movement of the hind limb.

Diagnosis
Diagnosis is based on clinical findings, history and Low serum and hepatic copper level.

Treatment and Prevention

Drug Treatment

Treatment is effective in mild cases. Copper glycinate, 60 mg/ head, SC, q 24 h until signs subside
Copper sulphate 4g weekly PO for 3-4 weeks
Copper supplement, 5 ppm in feed for sheep especially in the last half of gestation
Copper methionate: 20mg/50kg BW I/M, stat.

Prevention

Copper sulphate solution 20mg/kg for sheep and 35mg/kg for goats PO during the first and second week of life. The same treatment can also be used for emergency control of the disease.

Grain Overload/Lactic Acidosis/ Carbohydrate Engorgement

Grain overload (Lactic acidosis, Carbohydrate engorgement), is an acute disease of ruminants when they gain access to large quantities of readily digestible carbohydrates, particularly grain.

Clinical Symptoms

The clinical symptom includes:-
Splashy rumen, profuse diarrhea which is fetid and may contain undigested grain, dehydration, reduced or absence of rumen motility. In severe cases subnormal body temperature (36.5-38.5°C) shallow and rapid respirations and heart rate is increased in accordance with severity of the acidosis.

Diagnosis

The diagnosis is based on the history of available, clinical findings and a low ruminal pH (5.5-6). Examining the microflora of the rumen using Gram stain of ruminal fluid will reveal a change from predominantly gram-negative bacteria (normal) to gram-positive bacteria. A pH of <5 indicates severe acidosis.

Treatment and Prevention

Treatment and Prevention is similar with cattle. **See** Grain Over Load under The Diseases of Cattle.

Parturient Paresis

Parturient paresis in pregnant and lactating ewes and does is a disturbance of metabolism characterized by acute-onset of hypocalcemia and rapid development of hyperexcitability and ataxia, progressing to depression, recumbency, coma and death. Unlike parturient paresis in dairy cattle, which primarily occurs within a few days of calving, the condition in ewes and does usually occurs before and less commonly after parturition.

Clinical Symptoms

The earliest signs are slight hyperexcitability, muscle tremors and a stilted gait. These are soon followed by dullness, sternal decubitus (often with the hind legs extended backward), mild ruminal tympany and regurgitation of food through the nostrils, staring eyes, shallow respiration, coma and death within 6-36 hours.

Diagnosis

Diagnosis is based on history and clinical signs. Total serum calcium levels will be <2 mmol/L. Dramatic responses to calcium therapy are confirmatory. It must be differentiated from pregnancy toxemia

Treatment and Prevention

Drug treatment

Calcium borogluconate 25%: 50-100 ml IV or SC. Administer the mentioned volume slowly for IV injection within 10-20 minute

Calcium Borogluconate + Magnesium Hypophosphate (40% w/v + 5% w/v) 50 to 100 ml.

Prevention

Dietary modifications to increase the calcium: phosphorus ratio (>1.5:1) and

Ensure total calcium in the diet meets NRC requirements and avoid sudden dietary changes or other stressors.

Pregnancy Toxemia

Pregnancy toxemia affects ewes and does during late gestation. It is characterized by partial anorexia and depression, often with neurologic signs, progressing to recumbency and death. It is seen more often in animals carrying multiple fetuses. The primary predisposing cause of pregnancy toxemia is inadequate nutrition during late gestation, usually because of insufficient energy density of the ration and decreased rumen capacity as a result of fetal growth.

Clinical Symptoms

In the early stages of the disease, the ewe/doe may show decreased aggressiveness at feeding, particularly with grain consumption and animals will spend more time laying down. As the disease advances, ewes or does may also show signs of listlessness, aimless walking, muscle twitching or fine muscle tremors, opisthotonus and grinding of teeth. This progresses (generally over 2–4 days) to blindness, ataxia and finally sternal recumbency, coma and eventually death.

Diagnosis

Clinical signs associated with under nutrition or error of management and ewes are affected during the last weeks of pregnancy.

Treatment and Prevention

Drug Treatment

Dextrose 50%, 60–100 mL, IV, followed by balanced electrolyte solution with 5% dextrose

Calcium gluconate or borogluconate solution 50–100 mL, SC

Glycerol (50%), 120ml, PO, q 12 h from 1-3 times depending on its response. **Or**

Propylene glycol (50%), 60 ml, q 12, for 3 days or 100ml/ day PO.

Plus

Dexamethasone 2mg/ml, 1-2.5mg IM or IV once. To induce parturition/abortion if the ewe or doe is also thin or fat and cannot manage fetal demands in late pregnancy

Ewes or does in the early stages (i.e. are ambulatory, have a decreased appetite for grain and show few nervous signs) can often be treated successfully with oral propylene glycol (60 mL, bid, for 3 days, or 100 mL/day).

Prevention

Proper feeding schedule during pregnancy feed obese sheep and goats with roughage and 500 g concentrate per head

Propylene glycol 60 ml/adult animal as prophylactic

Note: Although aggressive therapy and intensive nursing care may be successful it is common to see case fatality rates >40%.Aggressive therapy should be directed against the ketoacidosis and hypoglycemia.

VIRAL DISEASES OF SHEEP AND GOAT

Bluetongue

Bluetongue is an arthropod-borne, noncontagious viral disease of sheep, wild ruminants and rarely cattle, goats and carnivores. Serological surveys have shown that blue tongue is prevalent in central and North West Ethiopia.

Clinical Symptoms

Dyspnea with panting hyperemia of the lips, muzzle and ears, pyrexia [42°C], depression, and inflammation, erosions and ulceration of the oral mucous membranes, particularly the dental pad are observed, other signs include swollen cyanotic tongue, lameness due to coronitis and widespread muscle necrosis, torticollis, vomiting, pneumonia, conjunctivitis, and alopecia.

Diagnosis

Clinical signs are presumptive. Confirmation is based on identification of the virus by isolation in embryonated chicken eggs, susceptible sheep or cell cultures. Serologic tests include ELISA, agar gel immunodiffusion, complement fixation and virus neutralization tests.

Treatment and Prevention

There is no specific treatment. Bluetongue is difficult to handle because of a wide variety of culicoid vectors of the disease. Monovalent vaccine is available in the USA only.

Contagious Ecthyma (Orf)

Contagious ecthyma (orf, contagious pustular dermatitis, sore mouth) is an extremely infectious dermatitis of sheep and goats. Young animals are more susceptible to the disease. The disease is usually more severe in goats than in sheep.

Clinical Symptoms

The lesions develop as papules and progress through vesicular and pustular stages before encrusting. Discrete large scabs, a verrucose mass underneath which are distributed primarily on the skin of lips extending to the mouth, inter-digital region, around the coronet and udder. Lambs fail to eat normally and lose condition, shows lameness and in ewes mastitis may occur.

Diagnosis

A diagnosis is based on characteristics and location of lesions, as well as flock history of previous outbreaks. A definitive diagnosis is based on viral isolation and an immunologic test.

Differential diagnosis includes dermatophilosis, bluetongue, sheep and goat pox and ulcerative dermatosis.

Treatment and Prevention

Animals are cured spontaneously in most cases with supportive care

Lesions can be treated with a single application of 3 % iodine solution

Antibiotics may help combat secondary infection

In endemic areas, appropriate repellents and larvicides should be applied to the lesions.

Public health significance: The virus is zoonotic; use protective gloves during handling of clinical cases.

Foot and Mouth Disease

Foot-and-Mouth Disease (FMD) is a highly contagious viral infection transmitted by direct or indirect contact. The epidemiology is similar to that of cattle (See Diseases of cattle - FMD).

Clinical Symptoms
In small ruminants, clinical disease is milder than in cattle, thus it is frequently overlooked. The characteristic clinical signs include fever (41-43°C), which persists for 3-4 days; lassitude and inappetence followed by vesicles in the mouth and foot. Other signs include lameness, increased breathing, prostration that may end up in death.

Diagnosis
On the bases of clinical signs and laboratory findings (ELISA, CFT) and isolation of the virus.

Treatment and Prevention
There is no specific treatment except supportive therapy including avoiding confinement, provision of easy medication, high quality feed and water and broad-spectrum antibiotics to prevent secondary bacterial infections (see FMD in cattle).

Prevention
Quarantine of farms and vaccination.

Nairobi Sheep Disease

Nairobi Sheep Disease (NSD) is a noncontagious, tick-borne, viral infection of sheep and goats. It is caused by NSD virus of the genus Nairovirus, family Bunyaviridae. The virus is transmitted primarily by the brown tick **Rhipicephalus appendiculatus**.

Clinical Symptoms
Acute hemorrhagic gastroenteritis, fever (40-41°C), followed by a temperature decline and diarrhea; mucopurulent nasal discharge. Breathing may become rapid and painful, mucoid and fetid diarrhea or fetid dysentery that causes painful straining. Deaths may occur within 2–7 days after the onset of acute disease but may be as long as 11 days in less acute cases. The clinical signs are less severe in goats.

Diagnosis
Clinical signs with high mortality accompanied by a tick infestation and history of movement of susceptible animals into an endemic area where **R. appendiculatus** is abundant are presumptive. Serological tests like CFT, virus neutralization and ELISA are confirmatory.
Differential diagnosis include Peste des Petits Ruminants, Rift Valley Fever and heartwater.

Treatment and Prevention
There is no specific treatment for NSD
Electrolyte supplement is recommended in diarrheic or dysenteric cases. Sick animals should be isolated and nursed well.

Prevention
Control of ticks by dipping or spraying of susceptible flocks can reduce the transmission and incidence of the disease
Movement of animals into endemic areas must be controlled.

Peste des Petits Ruminants

Peste des Petits Ruminants (PPR) is an acute or subacute highly contagious viral disease of goats and sheep. The disease is caused Peste des petits ruminants' virus (PPRV) which is closely related to rinderpest virus. The virus is transmitted by contact with nasal discharge, tears and secretions from coughing of infected animals.

Clinical Symptoms
Fever (40-41°C), depressed appetite, dull coat, dry muzzle, congested mucous membrane, congested conjunctiva, necrotic stomatitis, gastroenteritis and pneumonia. Diarrhea maybe profuse and is accompanied by dehydration and emaciation. Hypothermia and death follows, usually after 5-10 days. In goats the major clinical signs are stomatitis, enteritis and pneumonia.

Diagnosis
A presumptive diagnosis is based on epidemiology, clinical signs and pathologic features. Laboratory confirmation is by isolation of the virus, serological tests and molecular diagnostic tests.
The disease may resemble many common diseases including Foot and Mouth disease, contagious ecthyma, sheep and goat pox or Bluetongue.

Treatment and Prevention
No specific treatment
Fluid therapy is recommended to alleviate the effects of diarrhea/dysentery
Good nursing of sick animals facilitates recovery.

Prevention
Vaccination with an attenuated PPR vaccine.

Rabies

Rabies is an acute and often fatal viral disease of the central nervous system that principally affects carnivores and insectivorous bats, although it can affect any mammal. It is caused by a neurotropic rabies virus of the genus **Lyssavirus** and family **Rhabdoviridae**. Animals or human are infected through bites of rabid animals, principally dogs.

Clinical Signs
The incubation period of rabies is about 14-90 days. The clinical signs of rabies in different animal species are similar with slight variations. Two forms of clinical syndrome occur; namely: - furious and paralytic rabies. The furious syndrome is more common in goats and characterized by aggressiveness and continuous bleating in sheep. The paralytic form of the disease is characterized by inability to swallow, excessive salivation, posterior paralyses, convulsions and respiratory arrest. In terminal stages the animal become comatose and death occurs within 5-7 days after the onset of clinical signs.

Diagnosis
History of bite by a rabid animal and clinical signs. An animal suspected to be infected with rabies should be confined and clinical course monitored for at least 10 days. If the animal survives for more than 10 days then the presence of rabies is ruled out. Presence of intracytoplasmic inclusion bodies (Negri bodies) in neurons of formalin fixed and paraffin-embedded brain tissue sections particularly in cerebellum and brain stem is pathognomonic. Fluorescent antibody test is confirmatory.

Treatment and Prevention
There is no effective treatment.
Control of rabies in carnivores.

Public health significance: Rabies is the most fatal infectious zoonotic disease transmitted by contact.

Rift Valley Fever

Rift Valley Fever (RVF) is a peracute or acute, mosquito -borne zoonotic disease of domestic and wild ruminants in Africa caused by a Rift Valley fever virus of the genus **phlebovirus.** Susceptibility of different breeds to RVF varies considerably. Lambs and kids are the most susceptible. Clinical disease has not been reported in Ethiopia.

Clinical Symptoms

In peracute form, sudden death with no appreciable signs may occur. Acute form of the disease is more often in adult sheep. Biphasic fever (40-42°C)that subsides just prior to death, anorexia, weakness, listlessness, depression, increased respiratory rate, vomiting, bloody/fetid diarrhea, mucopurulent nasal discharge and icterus may be evident in a few animals, in pregnant ewes, 'Abortion storms' with rates approaching 100%.

Diagnosis

Diagnosis is based on history of heavy rains and flooding followed by widespread occurrence of abortions and mortality. Other clinical signs and epidemiology are suggestive. Virus isolation from aborted fetus of infected animals, serological tests and detection of viral nucleic acid by PCR are confirmatory.

Treatment and Prevention

There is no effective treatment

Control of mosquitoes by applying phosphoric acid esters powder in sheep during the rainy season

Movement of stock from infected area to non-infected area

Prevention

Vaccination of newly introduced animals is recommended with the modified live Smithburn vaccine is cheap and stimulates a long lasting immunity

The vaccination should be based on a country's RVF control policy.

Note: Rift Valley Fever is zoonotic and transmitted through contact with infected animals and their tissues and aerosols.

Sheep Pox and Goat Pox

Sheep and goat pox are serious, often fatal diseases characterized by widespread skin eruptions. Both forms are widely distributed in Ethiopia. The incubation period of sheep pox is 4–8 days and that of goat pox is 5–14 days. The clinical picture is similar in sheep and goat but is generally less severe in goats. Transmission is usually by close contact with severely affected animals containing ulcerated papules on the mucous membranes.

Clinical Symptoms

Fever (above 40°C), conjunctivitis with swollen eyelids and mucopurulent discharge crusts on the nostrils labored and noisy breathing. Widespread skin nodules are easily observed on the muzzle, ears and areas free of wool or long hair. Pregnant ewes may abort. In severe cases, lesions can occur in the lungs. In some sheep and in certain breeds, the disease may be mild or the infection is unapparent.

Diagnosis

Clinical signs are suggestive. Virus isolation and virus neutralization, serological and molecular tests are employed for confirmation.

Differential diagnosis include contagious ecthyma, Bluetongue, Peste des petits ruminants, Photosensitization and Dermatophilosis.

Treatment and Prevention

Antibiotics to prevent secondary bacterial infections.

Prevention

Vaccination of animals.

BACTERIAL DISEASES IN SHEEP AND GOATS

Actinobacillosis

Actinobacillosis is caused by gram-negative **coccobacilli** in the genus **Actinobacillus**. **A lignieresii** causes granulomatous swelling of the tongue, usually referred to as wooden tongue. The organism may also cause pyogranulomatous lesions in soft tissues associated with the head, neck, limbs, and occasionally the lungs, pleura, udder and subcutaneous tissue.

Clinical Symptoms

The primary lesion associated with **A. lignieresii** infection is a very hard, diffusely swollen, painful tongue. This leads to excessive salivation, the inability to prehend feed normally and sometimes a visibly enlarged tongue that protrudes from the mouth. On palpation, the tongue will feel very hard. Pyogranulomatous lesions are found in soft tissues associated with the head, neck and limbs.

Diagnosis

Clinical manifestations are fairly distinctive; **Gram stained** preparations from crushed exudates, cultural examination and response to treatment with iodine preparations. Differential diagnosis includes Actinomycosis and corynebacterium infections.

Treatment and Prevention

Similar with actinobacillosis in cattle. Refer Actinobacillosis under Disease of Cattle.

Anthrax

Anthrax is peracute or an acute, often fatal disease of animals caused by spore forming bacteria, **Bacillus anthracis.** In small ruminants it is characterized by septicemia and splenomegaly.

Clinical Symptoms

Peracute form is characterized by sudden death without premonitory sign. The course of the acute disease takes about 2 hours and it is initially characterized by depression. Fever (42°c), anorexia, labored breathing, congested and hemorrhagic mucosa, increased heart rate and ruminal stasis are common features. There may be blood discharges from natural orifices.

Diagnosis

Clinical signs are highly suggestive. Demonstration of square- ended rods in thin blood smear stained with polychrome methylene blue.

Differential diagnosis includes lightning strike, acute bloat, peracute lead poisoning and other clostridial infection should be considered as a differential diagnosis.

Treatment and Prevention

Drug Treatment

Penicillin G 20000 to 40000 IU/kg, IM, q 24 hr. for 3-5days; for severely affected animals, administer sodium penicillin, IV. **Or**

Oxytetracycline 20%, 20mg/kg bodyweight, IM, single dose; if required repeat after 3 to 5 days. **Or**

Oxytetracycline 10%, 5-10 mg/kg, IM, q 24 hr. for 5-7 days. **Or**

Doxycycline, 20 mg/kg, IV. **Or**

Erythromycin 12.5 mg/kg (adult), IM, IV or SC; 15-30 mg/kg (calves), IM, IV, or SC.

Prevention

Annual vaccination with Stern avirulent live spore vaccine. Animals should not be vaccinated within 2 months of anticipated slaughter; antibiotics should not be administered within 1 week of vaccination

Proper burial and burning of carcass: Due attention should be given to Select an appropriate site for carcass burial

Bacillary Haemoglobinuria

It is an acute, toxemic and highly fatal clostridial disease, with the liver being the main target organ. It is caused by **C. haemolyticum (C.novyi type D**). The incubation period is extremely variable and onset depends on the presence of a locus of anaerobiosis in the liver. Such a nidus for germination is most often caused by liver fluke (**Fasciola hepatica**) infection.

Clinical Symptoms

Animals may be found dead without premonitory signs. Usually, there is sudden onset of severe depression, fever, abdominal pain, dyspnea, dysentery and haemoglobinuria (red urine). Anemia and jaundice are present in varying degrees. Edema of the brisket may occur. Hemoglobin and RBC levels are quite low.

Diagnosis

The tentative diagnosis can be done by presence of typical port-wine-colored urine, which foams freely when voided or on agitation. Diagnosis can be confirmed by isolating **C. haemolyticum** from the liver infarct. Rapid and accurate diagnosis can be made by demonstrating the organism in the liver tissue by a fluorescent antibody or immunohistochemical test.

Differential diagnosis includes Bracken fern poisoning, leptospirosis, babesiosis, and postparturient haemoglobinuria.

Treatment and Prevention

See Diseases of Cattle: Bacillary Haemoglobinuria.

Blackleg/ Black Quarter

Blackleg is an acute infectious disease of ruminants which is characterized by emphysematous swelling, usually in the heavy muscles, sever toxemia and high mortality. The disease is caused by **Clostridium chauvoei** which is gram positive, spore forming and rod shaped bacterium. In Ethiopia, it is has been reported in cattle and sheep during the dry season.

Clinical Symptoms

Affected animals exhibit fever, lameness and severe depression, stiff gait and hot painful swelling of the affected muscles. Muscles of the shoulder, loin and buttock are mostly affected. The muscle become edematous, spongy and with crepitation sound. The skin over the affected area becomes dark and in later stages the swelling becomes cold and painless.

Diagnosis

Clinical and pathological features can aid in arriving at a tentative diagnosis of black quarter. The disease is confirmed by the demonstration of large gram positive single rod or chains with oval sub terminal or central spores in smears from tissue or exudates.

Differential diagnosis include anthrax, lightning strike, snake bite, malignant edema and other clostridial diseases.

Treatment and Prevention

See Disease of Cattle: Blackleg

Brucellosis

This is a disease caused by infection with bacteria of the genus **Brucella** and it is characterized by abortion in late pregnancy and subsequent high rate of infertility. Brucellosis in Goat and sheep normally caused by **B. melitensis** although **B. abortus** may also cause clinical brucellosis. The disease is zoonotic and occupational causing undulant or Malta fever in man. **Brucella ovis** causes epididymitis and orchitis that impair fertility in rams.

Clinical Symptoms
Abortion, retained placenta and orchitis. Abortions usually occur in late pregnancy in sheep and during fourth month of pregnancy in goats. In goats, mastitis and lameness may be seen. Arthritis is rare in sheep.

Diagnosis
Abortion storms in ewes and does and swelling of the scrotum in rams are indicative. Confirmation is by isolation and identification of bacteria and serological tests like complement fixation test, ELISA.
Differential diagnosis includes enzootic abortion, leptospirosis and other diseases that cause abortion.

Treatment and Prevention
Treatment is not economical except in especially valuable rams and even if infection is eliminated, fertility may remain impaired
Oxytetracycline 10%, 5-10 mg/kg, q 24 h, for 4-5 days. Or Oxytetracycline 20% 200mg/ml, 1ml per 10kg bodyweight, IM, single dose; if required repeat after 3 to 5 days

Prevention
Test animals before introduction to a flock and vaccinate against the disease, proper disposal of aborted materials, culling of infected sheep/goat.

Campylobacteriosis

Gastrointestinal campylobacteriosis is caused by **Campylobacter jejuni** or **C. coli.** The disease causes diarrhea in various large and small animals, pets and humans. Transmission is through fecal-oral spread. Food or waterborne transmission appears to be the principal avenues for infection.

Clinical Symptoms
Clinical manifestations of campylobacteriosis in young sheep may be more severe. In goats there are only occasional reports. Signs and diagnosis are almost similar to cattle. In adults it causes abortion (See Diseases of Sheep and Goats: Abortion).

Diagnosis
Round, yellow-white areas on the liver are often seen in foetuses aborted due to campylobacter infection. To confirm diagnosis at a laboratory, veterinarians will need to provide samples of the foetal liver for selective campylobacter culture or PCR testing, placenta or maternal vaginal discharges or formalin-fixed specimens of foetal liver for histopathology. Blood samples can be used to test for antibodies from a flock suspected to have been exposed to Campylobacter.

Treatment and Prevention
Drug Treatment
Enteric form is self-limiting, thus treatment is not usually indicated.
Outbreaks of abortion Streptomycin 70 mg/kg, IM, 2-5 days **Or**
Tetracycline in feed for 5 days followed by 100 mg/head q 24 h for the rest of the lambing period.

Prevention
For prevention we can use vaccination of replacement ewes only.
Public health significance: transmitted to humans through consumption of contaminated meat.

Caseous Lymphadenitis

Caseous Lymphadenitis (CL) is a contagious chronic bacterial disease of small ruminants caused by gram positive, facultative anaerobic and pleomorphic bacterium, **Corynebacterium pseudotuberculosis**. The disease is found throughout the world and is a major concern in sheep and goat. It causes economic loss from wool and hides loss, carcass condemnation and death. Caseous Lymphadenitis (CL) affects animals of all ages although it is commonly encountered in adult animals

Clinical Symptoms

The lymph nodes around the head and neck region are most commonly affected. Enlargement and abscessation of the peripheral lymph nodes which may form white, yellowish or greenish pus. In chronic case lungs are also involved. Other clinical signs include: dyspnea, exercise intolerance and weight loss.

Diagnosis

Provisional diagnosis of the disease can be based on clinical sign and pathological features. Laboratory diagnosis is achieved by demonstration of **Corynebacterium pseudotuberculosis** in smear made from pus. Further confirmation is through isolation and identification of the agent.

Differential diagnosis includes as **S. aureus, C. pyogenes and Actinomyces pyogenes** which cause similar abscess in or close to lymph nodes.

Treatment and Prevention

Draining or surgically removing the abscessed nodes at the ventral position and the cavity should be drained with iodine solution. However, this procedure must be done carefully to prevent environmental contamination

Erythromycin 100 mg/ml, 10mg/kg body weight, IM, q 24 hr. for 4 to 6 weeks. **Or**

Gentamicin Sulfate, 100mg/ml, 10mg/kg body weight IM, q 24 hr. for 4 to 6 weeks. **Or**

Penicillin G Procaine + Streptomycin sulfate, 200000 IU + 200 mg, 1 ml/10 kg bodyweight, IM, q 24 hr. for 3 – 4 days.

Prevention

Clean any environment contaminated with pus. Dilute bleach and chlorhexidine solutions are effective disinfectants of hard surfaces and fomites.

Dermatophilosis

Dermatophilosis in sheep and goats is a skin disease caused by the dimorphic gram positive bacterium **Dermtophilus congolensis.** The source of infection is sick or carrier animal and the disease is spread by contact. Prolonged wetting and mechanical damage to skin are predisposing factors. Arthropod vectors such as ticks, flies, lice and sheep ked may be involved in the transmission of dermatophilosis.

Clinical Symptoms

Goats are more susceptible to dermatophilosis than sheep. Clinical signs include pustules, ulcerative dermatitis and scab formation on muzzle, face, nose, ears, dorsal midline, scrotum and feet. The under surface of the scab is covered with a yellow, creamy or hemorrhagic and hair-matting exudates.

Diagnosis

Presumptive diagnosis depends largely on the clinical features and demonstration of gram positive **mycelia** organisms in stained smears made from the under surface of the scabs. A definitive diagnosis is made by culture and identification of **D. congolensis** bacterium.

Differential diagnosis include mange, contagious ecthyma, fungal dermatitis, fleece rot and photosensitization.

Treatment and Prevention

Drug Treatment

Penicillin G Procaine + Streptomycin sulfate, 200000 IU + 200 mg, 1 ml per10 kg bodyweight, IM, q 24 hr. for 3 – 4 days. **Or**
Ampicillin;4-10 mg/kg, body weight, IM, q 12 hr. for 4-5 days **Or**
Oxytetracycline 20%, 20 mg/kg, IM, single dose; or two doses one day apart **Or**
Iodophores; 2-5% lime sulfur; 0.5% zinc sulfate 0.2% copper sulfate and 1% potassium aluminum sulfate (alum) as sprays or wash for 3 to 5 days, then weekly until the lesion heals.

Prevention

Potash alum and aluminum sulfate have been used as wool dusts in sheep to prevent dermatophilosis. Minimizing moist conditions is helpful in control and prevention. Control of external parasites or other factors that cause skin lesions is important.

Foot Rot

Foot rot is a contagious disease of sheep and goats caused mainly by the coexistence of two gram-negative, anaerobic bacteria, **Fusobacterium necrophorum** and **Bacteroides nodosus.** The bacteria affect the interdigital epidermis extending to the horny and laminar structures. Wet and warm weather conditions favor the proliferation of the bacteria and soften the animal skin thus making it easily breakable and penetrable.

Clinical Symptoms

Initially, moist inflammation of the skin-horn junction and macerated interdigital skin and later foul smelling discharge from the lesion is observed, ulceration and necrosis of the sensitive laminae of the foot and severe lameness. Affected animals often carry the affected leg or lie down for extended periods, rubbing off the wool/hair on their flanks, brisket and knees.

Diagnosis

Clinical signs are highly suggestive of the disease. Demonstration of **B. nodosus** in smears taken from pus and scraping from the lesion.
Foot rot should be differentiated from other causes of lameness such as traumatic injury, necrobacillosis, strawberry foot rot and FMD.

Treatment and Prevention

Drug Treatment

Foot-baths containing 5% copper sulphate, 10% Zinc sulphate, 5-10% Formalin. **Or**
Topical antimicrobial sprays including oxytetracyline sprays. **Or**
Chlortetracycline hydrochloride + sulfanilamide Spray 2.850gm + 50gm. **Or**
Erythromycin Injectable; 10mg/kg body weight, IM, q 24 hr. 3-5 days **Or**
Gamithromycin: 6mg/kg (1 ml/25 kg) into the anterior to the shoulder SC stat. Sheep over 125 kg divide the dose so that no more than 5 ml at single site. **Or**
Tulathromycin: 2.5 mg/kg (1ml/40 kg BW) into the neck SC stat. For treatment of cattle over 300 kg bodyweight, divide the dose so that no more than 7.5 ml are injected at one site.
Thiamphenicol, Cutaneous use. Spray the solution on the affected area for 3 seconds once a day. Treatment can be repeated depending on the healing process, up to 3 consecutive days.

Prevention

Inspection of newly introduced animals for lesions of the foot and run them on footbath as above. Regular hoof trimming and foot bath and keep the floor dry.

Infectious Keratoconjunctivitis

Infectious keratoconjunctivitis in sheep and goats is characterized by blepharospasm, conjunctivitis, lacrimation, varying degrees of corneal opacity and ulceration. In sheep and goats, keratoconjunctivitis can be associated with **Chlamydia psittaci**, **Chlamydia pecorum**, **Mycoplasma** spp (notably **M. conjunctivae**) **and Moraxella ovis**. In sheep, infection with **Chlamydia psittaci** is the most common cause. In goats, it is mainly caused by **M. conjunctivae.**

Clinical Symptoms

The earliest clinical signs are photophobia, blepharospasm and epiphora, later, ocular discharge may become mucopurulent. Conjunctivitis, with or without varying degrees of keratitis, is usually present. In sheep and goats, concurrent polyarthritis may be present in association with **C. pecorum** infections.

Diagnosis

Presumptive diagnosis is based on ocular signs and concurrent systemic disease. Microbial culture of eye swabs may be beneficial in confirming though **Chlamydia** and **Mycoplasma** spp require special media.

Treatment and Prevention

Drug Treatment

Tetracycline ointment 10 mg/g, qid, for 3-4 days. **Or**
Oxytetracycline HCL-Polymixin B sulphate, 5 mg-10000 U/g, qid, 3- 4 days. plus
If animals have substantial uveitis, Atropine 1% ointment topically or IM, 1-3 times daily for 3-5days.
If topical therapy is not practical
Oxytetracycline 20%, 20mg/kg bodyweight, IM, single dose; if required repeat after 3 to 5 days. **Or**
Oxytetracycline 10%, 5-10 mg/kg, IM, q 24 h for 5-7 days.

Prevention

Good management practices are of paramount importance to reduce or prevent spread of infectious keratoconjunctivitis infection
Ultraviolet radiation from sunlight may enhance disease; therefore, affected animals should be provided with shade
Dust bags or insecticide-impregnated ear tags can be used to reduce the number of face flies (Musca autumnalis), an important vector for Moraxella.

Leptospirosis

Leptospirosis is a zoonotic disease with a worldwide distribution caused by several pathogenic serovars of **Leptospira**. The most common serovar of **Leptospira interrogans** involved in caprine abortion is **grippotyphosa**. Sheep are relatively resistant to leptospirosis and **Leptospira interrogans hardjo** is the major serovar. Localization and persistence of the organism in the uterus may result in fetal infection, with subsequent abortion, stillbirth, birth of weak neonates or birth of healthy but infected offspring.

Clinical Signs

Abortion, stillbirth, birth of weak neonate often febrile illness, hemolytic anemia, icterus and hemoglobinemia.

Diagnosis

History and clinical signs may indicate leptospirosis. Bacterial isolation of leptospirosis from blood, cerebrospinal fluid and milk can be attempted in acute cases. Serological tests are useful for screening herds with a history of abortions or reproductive problems.
Differential diagnosis include Enzootic abortion, Toxoplasmosis and Brucellosis

Treatment and Prevention

Penicillin G Procaine + Streptomycin sulfate, 200000 IU + 200 mg, 1 ml per10 kg bodyweight, IM, q 24 h, for 3 – 4 days. **Or**

Streptomycin or dihydrostreptomycin sulfate 200 mg/ml, 0.5ml/10kg body weight, IM, q 12 h, for 3-5 days. **Or**
Oxytetracycline 20%, 200mg/ml, 1ml per 10kg bodyweight, IM, single dose; if required repeat after 3 to 5 days or Oxytetracycline 10%, 5-10 mg/kg, IM, q 24 h for 5-7 days.

Prevention
For Prevention avoid direct or indirect contact with carriers and rodents and vaccination with the most endemic serovars.

Public health significance: Leptosirosis is zoonotic disease and thus care should be taken to avoid infection of humans.

Listeriosis

Listeriosis is caused by **L. monocytogenes.** It is characterized by encephalitis or abortion in adults and by septicaemia in fetuses and neonates. The clinical signs and diagnosis are similar to cattle (see Diseases of Cattle: Listeriosis).

Treatment and Prevention
Similar drugs that are used in cattle are also applied here; however, treatment of listeriosis in sheep is less effective.

Malignant Edema

Malignant edema is a sporadic often acute, febrile and fatal soil-borne wound infection characterized by acute gangrenous inflammation at the site of infection, edema and toxemia. Infection occurs by contamination of wounds with spores of bacteria. Deep puncture wound and severe trauma to tissue create anaerobic conditions that are favorable for the proliferation of the organism.

Clinical Symptoms
The clinical signs may be observed within 4-5 days after infections. Swelling and inflammation occurs initially at the site of infection. The swelling expands rapidly, becomes tense and the skin over it becomes dark. Emphysema and marked frothy exudation from the wound may occur. These muscle infections are extremely painful. Fever (41-42°C), weakness, depression, muscle stiffness and tremors and lameness occur.

Diagnosis
Diagnosis is usually based on history of surgery, obstetric complications, injection and clinical signs. Diagnosis can be confirmed rapidly on the basis of fluorescent-antibody staining of **C. septicum** from a tissue smear.
Differential diagnosis include Black leg.

Treatment and Prevention
Penicillin G Procaine, 20000- 40000 IU/ kg bodyweight, IM, q 24 h, for 3 – 5days. **Or**
Benzathine Penicillin G + Procaine Penicillin G, 200,000IU + 112,500IU/ml, 1 ml per10 kg bodyweight, IM, q 24 h, for 3 – 4 days. **Or**
Amoxicillin 6-10mg/ kg bodyweight, q 24 h, IM, for 3 days.

Salmonellosis

Salmonellosis is characterized clinically by one or more of three major syndromes: septicemia, acute enteritis and chronic enteritis. Transmission occurs after ingestion of contaminated feed. The most common **Salmonella** serotypes in sheep and goats are **S. typhimurium, S. dublin, S. abortus ovis**, **S. anatum, and S. montevideo.**

Clinical Symptoms

Acute enteritis with septicemia is the common form in adults as well as in lambs and kids, usually ≥1 week old. Initially, there is fever (40.5-41.5°C), followed by severe watery foul-smelling profuse diarrhea, sometimes dysentery, often tenesmus and death occurs in 24-48 hours. Subacute enteritis may occur in adult sheep on farms where the disease is endemic. The signs include mild fever (39-40°C), soft feces, inappetence and some dehydration. There may be a high mortality rate due to enteritis in lambs under a few weeks of age.

Diagnosis

Diagnosis of Salmonellosis depends on clinical signs and isolation of the pathogen from feces, blood or tissues of affected animals. Serological tests are also available. The disease must be differentiated from several diseases such as colibacillosis and coccidiosis.

Treatment and Prevention

Sulfadimidine sodium107 mg/kg body weight, IM, q 24 h, for 3-5 days. **Or**
Amoxicillin 6-10mg/ kg bodyweight, q 24 h, IM, for 3 days. **Or**
Penicillin G Procaine + Streptomycin sulfate, 200000 IU + 200 mg, 1 ml per10 kg bodyweight, IM, q 24 h, for 3 – 4 days. plus
Dexamethasone 2mg/ml, 1-2.5mg/kg, IM or IV once as a supportive therapy.

Prevention

Carrier animals should be isolated and culled or treated. Clean contaminated buildings, dispose contaminated materials and minimize stress in outbreaks. A strict farm management program should be introduced.

Public health significance: Except few species-specific Salmonella serovars, most serovars are zoonotic.
Note: Oral antibiotics may deleteriously alter the intestinal microflora, interfere with competitive antagonism and prolong shedding of the organism.

Septicemic Pasteurellosis

Septicemic or systemic pasteurellosis is caused by **P. haemolytica** biotypes A and T which were reclassified as Mannheimia **haemolytica** (biotype A) and **P. trehalosi** (biotype T). More recently, **P. trehalosi** has been reclassified as **Bibersteinia trehalosi**. It is most common in young, weaned sheep (~6 months old) after transport or a sudden feed change, but it can occur in sheep of any age throughout the year.

Clinical Symptoms

Acute onset of illness, very high fevers, dyspnea, anorexia and recumbency, dullness or coma, frothy discharge from the mouth, often death. Septicemia or localization of the infection in one or more tissue such as the joints, udder, meninges or lungs may occur.

Diagnosis

The clinical signs or sudden death, gross pathology and histopathology may be indicative. Isolation of **B. trehalosi** from a range of tissues and gross findings are confirmatory.

Treatment and Prevention
Oxytetracycline 20%, 20mg/kg bodyweight, IM, single dose; if required repeat after 3 to 5 days. **Or**
Oxytetracycline 10%, 5-10 mg/kg, IM, q 24 h for 5-7 days. **Or**
Sulfamethoxazole-Trimethoprim, 200mg/ml-40mg/ml, 1ml/10 kg, q 12h, IM, 3-5 days. **Or**
Amoxicillin 6-10mg/ kg bodyweight, q 24 h, IM, for 3 days.

Prevention
Environmental and husbandry risk factors that may predispose the animal should be minimized and preventive vaccination.

Tetanus

Tetanus is a sporadic disease of domestic animals characterized by hyperesthesia, tetany and convulsion of muscles. Tetanus toxemia is caused by a specific neurotoxin produced by **Clostridium tetani** in necrotic tissue. **C tetani** in most cases is introduced into the tissues through wounds, particularly deep puncture wounds that provide a suitable anaerobic environment and during docking or castration.

Clinical Signs
The earliest signs include muscle stiffness, tremors and prolapse of third eyelid. This is followed by trismus, unsteady gait and inability to move. Spasms of head muscles cause difficulty in prehension and mastication of food hence the common name lockjaw. Tetany of the masseter muscles causes drooling of saliva from the mouth and regurgitation of food through the nostril. The disease is highly fatal and death occurs 3-4 days after the onset of clinical signs.

Diagnosis
The clinical signs and history of recent trauma are usually adequate for a clinical diagnosis of tetanus. It may be possible to confirm the diagnosis by demonstrating presence of tetanus toxin in serum from affected animal.
Differential diagnosis includes strychnine poisoning, heartwater and cerebral meningitis.

Treatment and Prevention
Procaine penicillin G 40,000 IU/kg, q 24 hr. IM or 25,000IU/kg q 12 hr. for 3-5 days followed by q 24 hr. for another 5 days
Tetanus antitoxin 1500 IU, SC, q 24 hr. for 3-5 days
Acepromazine 0.05- 0.2mg/kg, IM, q 12 hr. until the signs subside
Clean wound with hydrogen peroxide
Keep animals in the dark, quiet place and Provide IV fluids.

Prevention
Prevention can be achieved by cleaning wound with antiseptics; and a tetanus anti toxin should be administered before mass surgical operations are carried out.

Tuberculosis

Tuberculosis is a chronic infectious granulomatous disease caused by acid-fast bacilli of the genus **Mycobacterium.** Sheep and goats are quite resistant to **M tuberculosis** infection. The disease is characterized by the development of tubercles and by abscess formation with resultant caseation and calcification.

Clinical Symptoms

Tuberculosis has two clinical forms: pulmonary or extrapulmonary. In the latter form; generalized signs including progressive emaciation, lethargy, weakness, anorexia and a low-grade and fluctuating fever. The respiratory form of the disease causes a chronic, intermittent, moist cough with later signs of dyspnea and tachypnea.

Diagnosis

Single or comparative intradermal tuberculin test using biologically balanced purified protein derivative tuberculin of **M bovis** and **M avium**. Confirmation of diagnosis requires isolation and identification of the organism, which may take 4 to 8 weeks.
Differential diagnosis includes Contagious Caprine Plueroneumonia (CCPP) and Maedi visna.

Treatment and Prevention

Treatment of tuberculosis in animals is not recommended because it is not economical. See disease of cattle under TB.
For prevention keep herds free by test and slaughter of reactors.

Public health significance: The disease is transmitted to humans mainly via consumption of raw milk. Thus Goat milk should be boiled or pasteurized before consumption.

PARASITIC DISEASES OF SHEEP AND GOATS

HELMINTHES

Coenurosis

Coenurosis is a parasitic disease caused by the larval stage (**Coenurus cerebralis**) **Taenia multiceps. Taenia multiceps** is is an intestinal parasite of canids and occasionally man. Taenia eggs expelled in the feces of infected dogs or other canids are the source of infestation for intermediate hosts, like sheep. **C. cerebralis**, affects the CNS, particularly the brain of sheep and gives rise to neurological signs of coenurosis (gid or stagger).

Clinical Symptoms
Dullness, circling, torticollis, loss of appetite, frequent bleating, separation from the flock, visual impairment, muscle tremors and pain response on pressure over the cystic area. Sometimes unilateral partial blindness correlated to cystic presence in cerebrum with depression, tilting of the head either towards right or left, head pressing, in coordination, irregular gait and paralysis.

Diagnosis
The disease can be diagnosed on the basis of history, clinical signs and on postmortem examinations. The differential diagnosis includes Listeriosis, louping ill, scrapie and brain abscessation and tumors.

Treatment and Prevention
Treatment is based on surgical removal of the coenurus cyst after general anesthesia of the animal. The approach has a very good success rate, especially after accurate localization of the lesion
Deworm the Dogs with anthelmintic at 6–8 week intervals and careful disposal of the carcasses/brain of affected sheep.

Fasciolosis

Fasciolosis is a parasitic disease of sheep and cattle caused by the ingestion of metacercariae of **Fasciola hepatica** or **F. gigantica**. It is common in water logged areas which favor the breeding of the snail which is the intermediate host.

Clinical Symptoms
In the acute form; sudden death is common. In subacute form rapid and severe hemorrhagic anemia, rapid loss of condition, a marked paleness of the mucous membranes and an enlarged and palpable liver, submandibular edema or facial edema and ascites. In chronic form; there is progressive loss of condition, anemia, resulting in submandibular edema and ascites.

Diagnosis
The diagnosis of fasciolosis is based on clinical signs, seasonal occurrence, prevailing weather patterns and a previous history of fasciolosis on the farm or identification of snail habitats. Fecal microscopic examination reveals the oval, operculated, golden brown eggs.

Treatment and Prevention
Triclabendazole (10 mg/kg PO once after meals or, for severe infections, twice 12 to 24 h apart). **Or**
Rafoxanide see Treatment of Fasciolosis in cattle **Or**
Albendazole 300 mg/bolus, 7.5mg/kg body weight, PO
Closantel (for flukes above 8 weeks old) 10 mg/kg, PO **Or**
Oxyclozanide 1 mg/kg, PO **Or**
Nitroxynil 10mg/kg, SC, repeating treatment as necessary throughout the period when infestation is occurring, at intervals of not less than one month.

Prevention
Strategic deworming of animals
Draining of habitat for the intermediate host snail control and grazing management.

Gastrointestinal Parasitism

Helminthosis is wide spread infections in small ruminants in the sub-Saharan region. Gastro intestinal parasites cause disease when they are present in large numbers or when the host animal is weakened by another disease or by poor nutrition. The epidemiology of gastrointestinal infections is influenced by environmental factors, managements system, host factors and parasitic factors. The common stomach worms with the appropriate treatment is described in the **Table 9 below.**

Clinical Symptoms

Gastrointestinal worm infestations are characterized by anorexia, loss in body weight, dehydration, profuse and watery diarrhea which is usually persistent. However, **Haemonchus** infections are characterized by severe anemia accompanied by generalized edema and progressive weight loss, which is caused by chronic infection and low burden, otherwise, it is fatal and acute. In the case of **Oesophagostomum** infections feces may have excessive mucus and streaks of blood.

Diagnosis

The clinical signs associated with GI parasitisms are shared by many diseases and conditions; however, a presumptive diagnosis based on signs, grazing history and season is often justified. Infestation usually can be confirmed by demonstrating nematode eggs or tapeworm segments on fecal examination.

Table 9: Choice of drug for gastrointestinal parasite of Sheep and Goat

Parasite genera	Treatment		Remark
	Type of drug	**Dose**	
• **Bunostomum plebotomum** • **Chabertia ovina** • **Oesophagostomum spp.** • **Haemonchus spp.** • **Nematodiurus spp.** • **Cooperia spp.** • **Trichostrongylus spp.**	Fenbendazole	Sheep and Goat 5 mg/kg	• **Trichostronglyus axei** is not eliminated by these compounds. • Migrating and inhibiting larvae are affected only by Fenbendazole; Albendazole and Oxfendazole. • All anthelmintics are administered only once.
	Oxfenbendazole	Sheep 5 mg/kg; Goats 7.5 mg/kg	
	Albendazole	Sheep: 7.5 mg/kg and for adult liver fluke 15 mg/kg	
	Tetramisole	15 mg/kg PO or SC, but should not exceed 4.5 g for cattle in a single SC or oral dose	
	Levamisole	8 mg/kg SC for Sheep & goat	
	Pyrantel pamoate	25 mg/kg	
	Ivermectin	200mcg/kg, SC	
	Monepantel	2.5 mg/kg, PO,	
	Moxidectin	200 µg /kg, PO, Stat	

PROTOZOAL AND RICKETTSIAEL DISEASES

Babesiosis

Babesiosis is a tick-borne protozoal disease affecting domestic animals. The disease is characterized by high fever, haemoglobinuria, anemia and prostration. In sheep and goats it is caused by an intra- erythrocyte protozoan parasite **B. motasi** and **B. ovis**, the former being the primary cause in goats.

Clinical Symptoms

The disease is manifested by anorexia, fever (41.7°C), weakness, anemia, dyspnea and tachycardia, pale mucous membrane, icterus and haemoglobinuria with coffee colored urine.

Diagnosis

Clinical signs are indicative and blood smear stained with giemsa and serological tests are confirmatory. Differential Diagnosis includes bacillary haemoglobinuria and Bracken fern poisoning.

Treatment and Prevention

Drug Treatment

Diminazene aceturate, 3 mg/kg, IM for 3 days **Or**
Diminazene aceturate + Phenazone, 70mg/ml+375mg/ml, IM, for 3 days **Or**
Imidocarb dipropionate 120 mg/ml, 1-2 mg/kg, IM **Or**
Amicarbalide.

Prevention

Strategic tick control

Caution: It should be used within 5 days from date of solution preparation and 14 days if kept in refrigerator.

Coccidiosis

Coccidial infection is universal in sheep and goats and can be a significant problem in the young ones of both species. Coccidiosis is an acute invasion and destruction of intestinal mucosa by protozoa of the genera **Eimeria or Isospora.** Coccidia can invade and destroy intestinal cells of the hosts, causing anemia, electrolyte loss and poor absorption of nutrients.

Clinical Symptoms

The most common clinical sign of infection is mucoid or bloody diarrhea. Infected sheep and goats will show inappetence, dehydration, a rough hair coat, poor weight gain and weakness emaciation. Death may occur.

Diagnosis

The diagnosis of coccidiosis depends on the clinical findings (diarrhea, dehydration, and progressive emaciation) and the presence of large numbers of pathogenic species oocysts in the feces usually at tens of thousands to millions per gram of feces.
Differential diagnosis: Salmonellosis, colibacillosis and cryptosporidiosis.

Treatment and Prevention

Decoquinate, 0.5 mg/ kg body weight in feed mixture is safe and very effective coccidiostat. **Or**
Amprolium 200 mg/ 1g, 5g /25kg BW, PO, for 5 days **Or**
Sulfadimidine sodium 107 mg/kg body weight, IM or SC, q 24 hr. for 3-5 days **Or**
Sulfamethoxazole-Trimethoprim, 200mg/ml-40mg/ml, 1ml/10 kg, q 12hr. IM, 3-5 days.

Prevention

As a prophylactic measure, Amprolium 200 mg/ 1g, 1g /50kg Body weight, PO, for 21 days Or
Monensin, 20g/ton of feed for 28 days, after lambs are introduced to the pasture/grazing Or
Diclazuril 1 mg/kg (1 ml /2.5 kg BW), PO, stat at about 4-6 weeks of age at the time that coccidiosis can normally be expected on the farm. Under conditions of high infection pressure, a second treatment may be indicated about 3 weeks after the first dosing.

Heartwater

Heartwater (Cowdriosis) is an infectious, non-contagious, tick-borne rickettsial disease of ruminants caused by **Cowdria ruminantum** and transmitted by tick belonging to the genus Amblyomma. Epidemiology depends on interaction of tick vector, causative agent, and vertebrate hosts. (For clinical sign, diagnosis, treatment and prevention see Disease of cattle: Heart water).

Toxoplasmosis

Toxoplasmosis is a disease caused by a protozoan parasite called **Toxoplasma gondii**. Sheep and goats are infected by ingesting sporulated oocysts from contaminated pasture with cat feces.

Clinical Symptoms

The fetus may be mummified, macerated, aborted or stillborn or may be born weak and fail to suckle properly, often succumb to disease and die within a week. Adult sheep do not show clinical signs but goats may die.

Diagnosis

Clinical signs and pathological features are indicative of infection. Available tests include the Sabin-Feldman dye test, complement fixation, direct and indirect hemagglutination, latex agglutination, modified agglutination, ELISA and indirect fluorescent antibody testing.

Treatment and Prevention

Monensin at 30mg/ewe q 24hr.

Sulfadiazine (15–25 mg/kg) and pyrimethamine (0.44 mg/kg) act synergistically and are widely used for treatment of toxoplasmosis.

Public health significance: Toxoplamosis is zoonotic and transmitted by consumption of raw meat and contact with infected cat feces. Thus pregnant women should particularly be protected from sources of infection to avoid abortion.

ECTOPARASITES IN SHEEP AND GOATS

MITES

Chorioptes

Chorioptic sheep mites (also called "leg mites", or "foot scab") are found worldwide, but less harmful than psoroptic or sarcoptic mites. Preferential sites for chorioptic mites are the hooves and lower part of the legs. Occasionally they also affect the scrotum, face and lips. **Chorioptes caprae** is fairly common in goats.

Clinical Symptoms
Affected parts show formation of scales and crusts, non-nodular papules, alopecia, erythema and ulcerative mainly in limbs, udder, scrotum and peri-anal region. Pruritis is common.

Diagnosis
Based on clinical signs and microscopic examination of deep skin scrapings.

Treatment and Prevention
See Treatment for Sarcoptes in cattle.

Demodex

Demodex ovis infests sheep, and **Demodex caprae** infests goats. Demodectic mange in sheep is not common, whereas **D. caprae** are relatively common in goats. Demodectic mange in goats occurs most commonly in kids, pregnant does and dairy goats.

Clinical Symptoms
In goats, nonpruritic papules and nodules develop, especially over the face, neck, shoulders and sides. The nodules contain a thick, waxy, grayish material that can be easily expressed; mites can be found in this exudate. The disease can become chronic.

Treatment and Prevention:
Non-drug treatment
Incise nodules and infuse with Lugol's iodine or rotenone in alcohol (1:3)
Drug Treatment
Ronnel in propylene glycol, 180 mL of 33% ronnel in 1 L of propylene glycol, applied to one-third of the body daily until cured for generalized cases in goats **Or**
Rotenone in alcohol (1:3) applied to one- fourth of the body daily until cured. **Or**
Trichlorfon 2% spray or dipping, in sheep.

Psoroptes (Sheep Scab)

Sheep scab is a serious and very harmful sheep disease. Lesions often affect the back, the flanks and the shoulders. Infestations remain often unnoticed until wool loss becomes evident, which mostly means that the whole flock is probably already infested. The sheep mite **Psoroptes ovis** causes large, scaly, crusted lesions almost exclusively on wooly parts of the body.

Clinical Symptoms

Clinically animals show intense pruritus manifested by vigorously scratching, biting and rubbing against objects, large, scaly, crusted lesions almost exclusively on wooly parts of the body. Untreated sheep often become emaciated and anaemic. Mites are sometimes found in the ears.

Diagnosis

Diagnosis is based on clinical signs and examining skin scrapping of affected parts under the microscope for visualization of the mites.

Treatment and prevention

Diazinon 0.03-0.1% every 7 days for 3 times. **Or**
Coumaphos 0.3% spray q 7 days. **Or**
Phosmet 0.15-0.25% every 7 days. For 3 times **Or**
Doramectin or Ivermectin 0.3mg/ kg body weight SC, every 7 days for 3 times.

Psoroptes (in goats)

Psoroptic mange (ear mange) in goats is caused by **P cuniculi**, which is likely a variant of **P ovis**. **P. cuniculi** typically infests the ears of goats but can spread to the head, neck and body.

Clinical Symptoms

It usually affects the ears but can spread to the head, neck and body and cause severe irritation resulting in crusting, inflammation, alopecia, ear scratching, head shaking and rubbing of ears and head to alleviate irritation.

Diagnosis

Diagnosis is based on clinical signs and examining skin scrapings of affected parts under the microscope for visualization of the mites**.**

Treatment and Prevention

Chrotoxyphos or Coumaphos, 0.25%, spray q 7 days. **Or**
Trichlorphon 0.2%, spray q 7 days. **Or**
Diazinon spray or dip. **Or**
Amitraz 12.5% solution, 1liter Amitraz/ 250 liter of water, spray animals 2 times at 10-14 days interval.

Note: Lactating dairy goats should be treated only with lime sulfur solution. Although the course is chronic, the prognosis is good with appropriate treatment.

Sarcoptes

The sarcoptic mange that causes scabies in sheep and goat are **Sarcoptes scabiei** var **ovis** and **Sscabiei** var **caprae** respectively. Sarcoptic mites of sheep are a species-specific strain of **Sarcoptes scabiei**, a mite species that infests also cattle, pigs, other livestock and also humans. They are less abundant on sheep than psoroptic mites.

Clinical Symptoms

The affected skin develops pimples and papules that become crusty and shows hardening, thickening and building of folds. Infestations often affect non-wooly skin and frequently start on the head and later spread along the neck and forelegs.

Diagnosis

Based on clinical sign and microscopic examination of deep skin scrapings.

Treatment and Prevention

Lime Sulfur solution or 0.05% Amitraz 5-10 applications, every 5 to 7 days.

Doramectin or Ivermectin 0.3mg/kg body weight SC, single dose.

Public health significance: Sarcoptic manges are zoonotic and care should be exercised to avoid human infection.

Tick Infestation

Ticks are important vectors of infectious diseases of small ruminants. The species of ticks which are commonly recovered from small ruminants include **R. evertsi**, **R. appendiculatus, R. lunulatus**, **H. truncatuma**, **A. varigatum**, **A. hebraema, Ixodes ribicundus** and **Boophilus decoloratus**. Different species of ticks have different predilection sites which include the ears, perianal area, scrotum and lower parts of the limbs. Direct contact is the principal mode of transmission but animals can be infested by various stages of ticks which have dropped on pasture.

Clinical Symptoms

Heavy infestation with ticks can cause anaemia. Some species of ticks causes tick paralyses while others cause tick toxicosis. Intense lameness has been noted in goats where ticks are attached around the coronary band.

Treatment and Prevention

All niches and crevices in affected building should be sprayed with acaricides to control soft ticks. For choice of acaricides, see in Cattle part

Acaricide spray or dip every 21 days; however, to control all nymphs, treat every 12 days during tick season for the control of one host-ticks

Weekly dipping/spraying during the tick season for the control of two-host and three-host ticks

Other control options include: Cultivation of land, improved drainage and vaccines (recently developed).

Cutaneous Myasis

It is caused by infestation of the skin by blowfly maggots of **Lucilia, Phormia, Calliphora, Chrysomya** etc. species. Infestation causes serious wool loss. The incidence of fly strike is common during humid months of the year and usually associated with fleece rot, mycotic dermatitis, diarrhea, urine staining and foot rot. Once the larva strike decomposing tissue or fleece, it may spread to normal skin and give way to other secondary flies that may invade and extend the lesion.

Clinical Signs

The sheep become restless, tend to bite or kick at the struck area, wriggle their tails, the wool might be lifted off, ulceration may occur and serous fluid oozes out followed by pus.

Diagnosis

Predisposing diseases such as foot rot, wound infections and diarrhea due to parasitic gastroenteritis are usually easily detected and fleece rot is indicated by matting of the wool and discoloration.

Treatment and Prevention

Application of larvicidals, BTH dressing (Boric acid, tar oil, bentonite mixture). **Or**
Organophosphate insecticides.

Prevention

Cyromazine 10 per cent w/v was used at an application rate of 50 to 100 mg/kg pour-on
Dicyclanil, 7.5–25 mg/kg (0.7–2 ml/kg), Pour-on, best applied before an anticipated blowfly challenge.

RESPIRATORY DISEASES OF SHEEP AND GOAT

Aspiration Pneumonia

Aspiration pneumonia (inhalation pneumonia) is characterized by pulmonary inflammation and necrosis due to inhalation of foreign material mostly due to inappropriate oral drenching. The severity of the inflammatory response depends on the material aspirated and the distribution of aspirated material in the lungs. In sheep, poor dipping technique with repeated immersion of the animal's head may cause aspiration of fluid. Bacteria in aspirated material may initiate acute infection or secondary infection.

Clinical Signs
Affected animals separate from the rest of the group and present with pyrexia (40°C–40.5°C), a painful expression, arched back, inappetence, depression and increased respiratory rate (>40–60 breaths/min) with a shallow abdominal component. This is often associated with a purulent nasal discharge that sometimes is tinged reddish brown or green. Thoracic auscultation reveals reduced lung sounds over affected lung.

Diagnosis
A history suggesting recent foreign body aspiration within the past 1–2 days. Auscultation and clinical signs are indicative.

Treatment and Prevention
See under the Diseases of cattle

Contagious Caprine Pleuropneumonia (CCPP)

Contagious Caprine Pleuropneumonia (CCPP) is one of the most severe diseases of goats caused by **Mycoplasma capricolum** sub species **capripneumoniae**. This disease, which affects the respiratory tract, is extremely contagious and frequently fatal; in some naive flocks, the morbidity rate may reach 100% and the mortality rate can be as high as 80%.

Clinical Symptoms
Peracute, acute and chronic forms may be seen in endemic areas. Peracutely affected goats can die within 1 to 3 days with minimal clinical signs. In acute disease, the initial signs are a very high fever (41-43°C; 106-109°F), lethargy and anorexia. This is followed by coughing and labored respiration and grunting. Pregnant goats may abort. Subacute or chronic cases tend to be milder with coughing mainly following activity. Chronic CCPP is characterized by a chronic cough, nasal discharge and debilitation.

Diagnosis
The clinical signs, epidemiology and necropsy findings are used to establish a diagnosis. Definitive diagnosis requires culture of the causative organism from lung tissue samples and/or pleural fluid taken at post-mortem. Serologic tests such as complement fixation, passive hemagglutination, ELISA and latex slide agglutination may be used.
Differential diagnosis includes PPR, pasteurollosis and contagious agalactia.

Treatment and Prevention
Tylosin 11mg/kg body weight, q 24 h, for 3-5 days **Or**
Oxytetracycline 10%, 5-10mg/kg, IM, q 24 h, for 3-5 days
Animal must be vaccinated annually for prevention of the disease.

Enzootic Pneumonia

Enzootic pneumonia is an acute infectious disease of sheep characterized by fever, nasal discharge, pneumonitis and pleuritis. The aetiology is complex. The main causes of the disease are **Chlamydia psittaci**, **Parainfluenza 3 virus** and **Pasteurella Spp.** Thus 'enzootic pneumonia' is more of a clinical term describing a range of respiratory diseases in sheep. Enzootic pneumonia is transmitted by direct contact between infected and susceptible sheep. During crowding the micro-organisms contaminate the air, feed, water and equipment and are transmitted from carrier sheep to susceptible sheep by inhalation and ingestion.

Clinical Symptoms
Fever, Refusal to eat, depression, loss of condition, mucopurulent nasal discharge, ocular discharge, dyspnea often accompanied by dry hacking coughing.

Diagnosis
Given the multifactorial nature of pneumonia in sheep, a single cause may not be found. Isolation and identification of the causative agents from affected tissues. It may be supported by ELISA from acute and convalescent sera samples.
Differential diagnosis includes CCPP, PPR and Pasteurollosis.

Treatment and Prevention
Oxytetracycline 20%, 20mg/kg bodyweight, IM, single dose; if required repeat after 3 to 5 days. **Or**
Oxytetracycline 10%, 5-10 mg/kg, IM, q 24 hr. for 5-7 days. **Or**
Tylosin 11mg/kg body weight, IM, q 24 hr. for 3-5 days. **Or**
Penicillin G Procaine + Streptomycin sulfate, 200000 IU + 200 mg, 1 ml per10 kg bodyweight, IM, q 24 hr. for 3 – 4 days. **Or**
Amoxicillin 150 mg/ml, 1ml/10 kg bodyweight, q 24 hr. IM, for 3 days.

Maedi-Visna

Maedi-visna is a chronic and economically important viral disease of sheep that occasionally affects goats. Maedi-visna results from infection by the maedi-visna virus, a member of the genus **Lentivirus** in the family **Retroviridae**. The disease is most common in animals >4 years old. Housing and close confinement facilitate transmission of infective droplets. The virus can also be transmitted mechanically by biting insects, fleas and lice.

Clinical Symptoms
Most infections are asymptomatic. In animals with clinical signs, coughing, bronchial exudates, depression, fever, mastitis and progressive wasting with increasing respiratory distress are common. The affected animals lag behind the flock. In the encephalitic form (visna); ataxia, muscle tremors or circling progresses to paresis and eventually complete paralysis. The disease has been recently introduced to Ethiopia possibly through importation of exotic sheep from Europe.

Diagnosis
Maedi-visna should be suspected in animals that are at least 2 years old and have a wasting disease with slowly progressive respiratory distress and neurologic signs. The diagnosis should be confirmed by histopathology, serology or isolation of the virus.
Differential diagnosis includes CCPP and Coenerus cerebralis.

Treatment and Prevention
There is no effective treatment for maedi visna. Supportive therapy may be helpful in individual animals.

Prevention
All replacement stock should come from Maedi Visna virus negative herds
Separate all sero-positive animals from sero-negative ones
Separate newborn lambs from their mothers and rear with milk from healthy ewes.

Pneumonic Pasteurellosis (Shipping Fever)

Pneumonic pasteurellosis is a cranioventral fibrous bronchopneumonia caused by **Pasteurella** and **Mannheimia spp** particularly **Pasteurella multocida, Mannheimia haemolytica** and **P. trehalosi.** The disease affects sheep and goats of all ages. It can be particularly devastating in lambs and kids around weaning. Predisposing factors that expose to pasteurella infection include inadequate colostrum intake, stress due to transportation weaning, infections with **parainfluenza type 3**, **adenovirus,** respiratory syncytial virus, **Bordetella parapertussis** or in particular **Mycoplasma ovipneumoniae.**

Clinical Symptoms
Fever of 40-41.1°C, increased respiratory rate, serous to mucopurulent ocular and nasal discharges, congested mucous membranes, anorexia, coughing, dyspnea and lethargy are noted. There may be evidence of dehydration with sunken eyes. Morbidity and mortality rates are variable. Frothy fluid may be noted around the mouth in terminal stages.

Diagnosis
In acute cases, diagnostic cultures may be obtained from tracheal washes or from necropsy specimens. In chronic cases, bacterial cultures may be less rewarding.
Differential diagnosis includes CCPP and maedi visna.

Treatment and Prevention
It is similar to Bovine Respiratory Diseases

Prevention
Mass prophylactic medication with antibiotics before stress with Oxytetracycline 20%, 20mg/kg bodyweight, IM, single dose; if required repeat after 3 to 5 days. Or Tilmicosin 10mg.kg; SC q 72 h for 3 days.
Avoid or minimize stress.

Nasal Bot

Oestrus ovis is the cause of the disease and it is a cosmopolitan parasite whose larval stages, inhabits the nasal passages and sinuses of sheep and goats and migrates to the nasal cavity. Individual sheep may show signs of nasal irritation or (rarely) breathing difficulties and flocks may show signs of disturbance due to bot fly attacks.

Clinical Symptoms
Profuse discharge which is at first clear and mucoid but later mucopurulent and frequently tinged with fine streaks of blood emanating from minute hemorrhages produced by the hooks and spines of the larvae. Thickening of the nasal mucosa, impairment of respiration, paroxysms of sneezing. To avoid the fly's attempts at larval deposition, a sheep may run from place to place, stamping the front feet, running in short bursts and burying their noses into the fleeces of other sheep.

Diagnosis
It is based on clinical signs and isolation of the larvae.

Treatment and Prevention
Rafoxanide, 7.5 mg/kg, PO, **Or**
Closantel, 10 mg/kg, PO, **Or**
Doramectin or Ivermectin 0.2 mg/kg body weight SC, single dose is effective against all stages of the larval development,

Verminous Pneumonia

Verminous or parasitic pneumonia of sheep and goats most commonly are caused by infection with **Dictyocaulus filaria**, **Muellerius capillaris** or **Protostrongylus rufescens**. **Muellerius capillaris** is the most common lungworm in goats**.** Affected goats have diffuse pneumonia without nodular lesions. **M. capillaris** may predispose animals to secondary infections and compromise health in general. The pathogenic effect of lungworms depends on their location within the respiratory tract, the number of infective larvae ingested and the animal's immune status.

Clinical Signs
Chronic fever, coughs, thick nasal discharge, increased respiratory rate, poor appetite and weight loss. Respiratory difficulty may ensue and heavily infected animals stand with their heads stretched forward and mouths open and drool.

Diagnosis
Diagnosis is based on clinical signs, epidemiology, presence of first-stage larvae in feces and necropsy of animals in the same herd or flock. The first-stage larvae can be isolated and identified in fecal samples using the Baermann Floatation technique to provide a definitive diagnosis.
Differential diagnosis include CCPP and Maedi visna.

Treatment and Prevention
Levamisole 8 mg/kg, PO or
Doramectin or Ivermectin 0.2mg/kg body weight SC, single dose. **Or**
Oxbendazole or Oxfenbendazole, sheep 5 mg/kg; goats 7.5 mg/kg **Or**
Fenbendazole15 mg/kg, PO, q 12 h 3 weeks apart. **Or**
Albendazole300 mg/bolus, 10mg/kg body weight, PO

DISEASES OF REPRODUCTIVE SYSTEM

Abortion

Abortion in the ewe is caused by infectious or non-infectious causes. The major infectious agents causing abortions are **Campylobacter, Chlamydia, Toxoplasma, Listeria, Brucella** and **Salmonella**. See Diseases of Cattles for Abortion.

Enzootic Abortion

Enzootic Abortion (EA) or Chlamydial abortion is a disease of sheep caused by **Chlamydophila abortus** (formerly known as **Chlamydia psittaci).** Exposure to the bacteria is usually as a result of direct contact with aborted materials. The infectious organism enters the body via ingestion or inhalation. Following an abortion the ewes will often continue to have normal lambing in subsequent years. However, these ewes are carriers of the disease and may shed organisms every year even if they produce normal looking lambs. This shedding infects their own lambs but also any other in contact sheep.

Clinical Symptoms
Characterized by late abortions, stillbirths or weak lambs. Abortions can occur at any stage of pregnancy, but most are in the last month. Aborted lambs are usually fresh with no gross pathology. Placentitis is usually present and consists of reddish brown exudate covering cotyledons and intercotyledonary areas.

Diagnosis
Diagnosis is based on examination of aborted materials. In infected ewes gross examination of the placenta reveals thickened, edematous intercotyledonary areas and necrotic cotyledons. Demonstration of the organism as elementary bodies in intercotyledonary areas on modified Ziehl Neelsen smears stain. ELISA or the fluorescent antibody test can be used as well.
Differential diagnosis includes Brucellosis, Leptospirosis, campylobacter and Toxoplasmosis

Treatment and Prevention

Sulfamethoxazole-Trimethoprim, 25mg/ kg bodyweight, q 24h, IM, 3-5 days **Or**
Penicillin G Procaine, 20000-40000 IU per10 kg bodyweight, IM, q 24 hr. for 5 – 7days,
Ceftiofur sodium, 2.2 mg/kg, IM, SC, q 12 hr. 5-7 days. **Or**
Penicillin G Procaine + Streptomycin sulfate, 200000 IU + 200 mg, 1 ml per10 kg bodyweight, IM, q 24 hr. for 3 – 4 days. **Or**
Oxytetracycline 10%, 5-10 mg/kg, q 24 hr. for 4-5 days. Or Oxytetracycline 20%, 200mg/ml, 1ml per 10kg bodyweight, IM, single dose; if required repeat after 3 to 5 days.

Mastitis in Goats

The organisms infecting the udder of goats are similar to those in cows. It's a multi factorial disease caused by a range of etiological agents. Coagulase-negative **staphylococci** are prevalent and appear to cause persistent infections and cause subclinical mastitis. The level of infection and incidence of mastitis due to **Staphylococcus aureus** and **Streptococcus agalactiae** and other streptococci tend to be low.

Clinical Symptoms

Clinical mastitis is manifested by inflammation of the udder and often accompanied by severe swelling, drop in milk production and abnormal milk secretions with clots. The signs depend on the organisms involved. Systemic signs could also be observed. Subclinical mastitis is the most common.

Diagnosis

It is based on clinical signs and identification of the pathogen. Tests to detect subclinical mastitis include California Mastitis Test or direct somatic cell count.

Treatment and Prevention

For subclinical mastitis

Erythromycin sodium intramammary infusion, 300 mg, per quarter, q 12h, for 3 days
Lincomycin + Neomycine Intramammary Solution 330mg + 100 mg, per quarter, q 12 h, for 3 days
For per acute and acute mastitis
Penicillin G Procaine, 30000 IU per10 kg bodyweight, IM, q 24 h, for 3 – 5days. Dry period therapy can be applied with long acting penicillin preparation
Amoxicillin 6-10mg/ kg bodyweight, q 24 h, IM, for 3 days
Cloxacillin sodium intramammary infusion, 200 mg per quarter, q 12h, for 3 days.

Puerperal Metritis

In all species, acute puerperal metritis occurs within the first 10–14 days postpartum. It results from contamination of reproductive tract at parturition and often, but not invariably, follows complicated parturition. Predisposing factors include prolonged delivery, dystocia and retained fetuses or placentas. It is characterized by fetid, watery uterine discharge.

Clinical Symptoms

Fever, purulent vaginal discharge, depression, lethargy, inappetence and may neglect their offspring. A large, flaccid uterus may be palpable.

Diagnosis

Examination of the uterus with a speculum combined with history, visual inspection of the perineum, examination of the tail for evidence of vaginal discharge and transrectal examination of the reproductive tract.

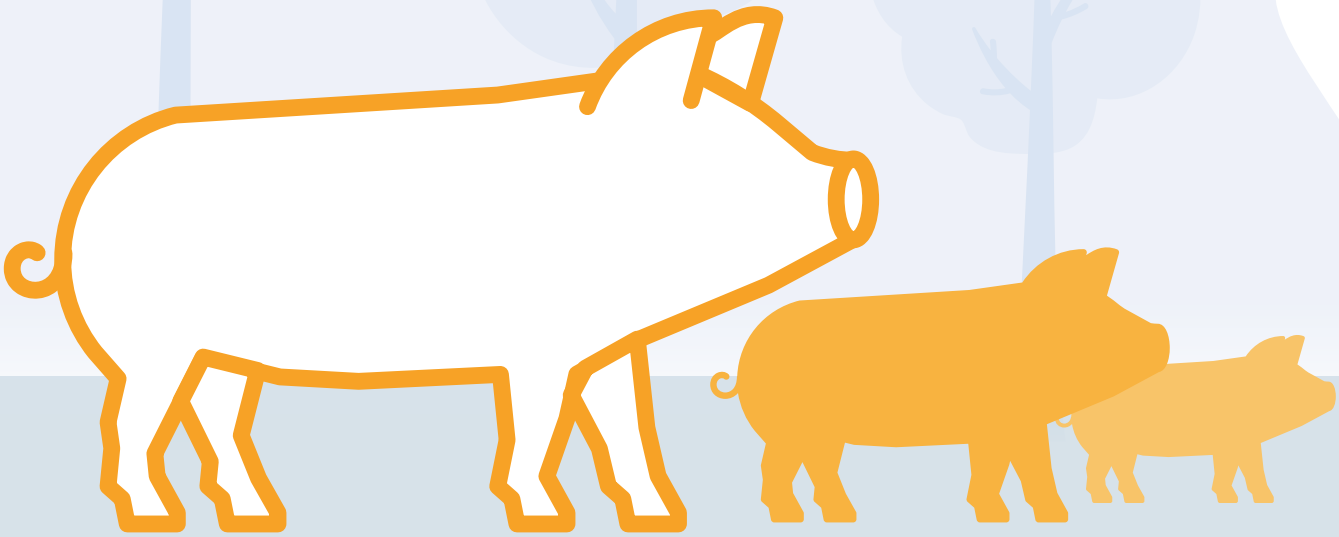
Treatment and Prevention

Penicillin G Procaine + Streptomycin sulfate, 200000 IU + 200 mg, 1 ml per10 kg for 3-5 days.
Oxytetracycline 20%, 20mg/kg bodyweight, IM, single dose; if required repeat after 3 to 5 or Oxytetracycline 10%, 5-10 mg/kg, IM, q 24 h for 5-7 days,
Sulfamethoxazole-Trimethoprim, 25mg/ kg bodyweight, q 24h, IM, 3-5 days.
Maintain good hygiene in delivery pens and assistance during delivery for prevention and control.

Retained Fetal Membranes

Retention of fetal membranes usually is defined as failure to expel fetal membranes within 24 hours after parturition. Most field veterinarians however, agree that if retention is over 12 hours, it must be considered as pathologic. The incidence increases with abortion, dystocia, hypocalcemia, twin birth and high environmental temperature, advancing age of the cow, premature birth or induction of parturition, placentitis, ducts infection and nutritional disturbances. (For details see under Disease of Cattle).

SECTION III



DISEASES OF PIGS[3]

3 *Guide*

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.

NON INFECTIOUS DISEASES

Iron Deficiency in Piglets

Iron deficiency usually develops in nursing piglets reared in confinement mainly as a result of low iron content of sow's colostrum and milk due to elimination of contact with iron from soil.

Clinical Symptoms
In absence of iron supplementation, pigs rapidly become anemic and retarded. The common clinical signs are identified with reduced growth rates, severe dyspnea, contracting jerks of the diaphragm muscle, lethargy with exercise, pale skin and mucosae and diarrhea.

Diagnosis
Clinical sign are suggestive.

Treatment and Prevention
Iron dextran; 100-200mg, IM, at 1-3 days of age and may be repeated in 10-14 days.
For prevention, Iron dextran, 100-150mg, IM, at 1 - 3 days of age.

VIRAL DISEASES OF PIGS

African Swine Fever

It is a peracute highly contagious and highly fatal disease of pig caused by African swine fever virus. The transmission occurs by Argasidae ticks, once established, the disease spreads rapidly by direct contact or ingestion of contaminated feed.

Clinical Symptoms
Fever that subsides after four days and development of cyanotic skin, anorexia, huddling together, disinclination to move, incoordination of hind quarter and move on front legs dragging the hind legs, naso-occular discharge, dyspnea and cough.

Diagnosis
Clinical sign and serological tests are indicated for diagnosis.

Treatment and prevention
No specific treatment
For secondary bacterial infection.
Lincomycin hydrochloride 10-15 mg/kg, IM, q 12 hr. and with hydrogen peroxide 8.3 mg/kg q 24 h, PO for 3-5 days **Or**
Oxytetracycline 0.5g/kg of feed for 7 days, and 10-20 mg/kg IV or IM q 6 h for 7 days. **Or**
Trimethoprim-sulphadiazine, 15 mg/kg, PO, q 12 h for 3-5days Or 48 mg/kg, q 24 hr. IM.

Prevention
Maintain disease free status by restriction of pig movement and serological monitoring and prevention of contact between domestic pigs and warthogs. Prohibition of importation of pigs and pig products and tick control

Foot and Mouth Disease

It is a highly contagious infectious viral disease. There are seven immunologically distinct serotypes: A, O, C, Asia 1 and SAT (Southern African Territories) 1, 2 and 3. There are a number of vaccine strains for each serotype, particularly O and A are required to cover the antigenic diversity. Transmission occurs by contact through oral or respiratory routes.

Clinical Symptoms
The clinical signs and lesions are similar to cattle (**see** Diseases of Cattle: Foot and Mouth Disease). Vesicles may also appear on the teats and udder and on areas of skin subject to pressure and trauma, such as the legs. Piglets may die before showing any vesicles.

Diagnosis
Clinical signs and confirmed by serology, ELISA or virus culture.

Treatment and Prevention
There is no specific treatment for FMD. But antibiotic treatment is indicated for secondary infection.

Swine Influenza

An acute, highly contagious, respiratory disease that results from infection with type A influenza virus.

Clinical Symptoms
Depression, fever 42°C, anorexia, coughing, dyspnea, weakness, prostration and a mucous discharge from the eyes and nose. Mortality is generally 1-4%. Animals recover from 3-7 days in uncomplicated infections.

Diagnosis
Clinical sign are indicative and confirmed by isolation of the virus or demonstration of virus-specific antibody.

Treatment and Prevention
There is no specific treatment, although antimicrobials may reduce secondary bacterial infections.

Prevention
Vaccination and strict import controls
Good management practices and freedom from stress, particularly due to crowding and dust, help to reduce losses.
Public health significance: Swine influenza virus might revert its antigenicity and infect humans.

BACTERIAL DISEASES OF PIGS

Brucellosis

Brucellosis in pigs is caused by **B. suis**. The disease often self-limiting, however, it may persist for years in some herds. It is usually spread mainly by ingestion of infected tissues or wastes. Infected boars may transmit the disease during mating. Suckling pigs may become infected from sows, but most reach weaning age without becoming infected.

Clinical Symptoms
Common manifestations are abortion, temporary or permanent sterility, orchitis which may be unilateral, lameness, posterior paralysis, spondylitis and occasionally metritis and abscess formation in extremities or other areas of the body and sterility.

Diagnosis
The Rose Bengal agglutination test and ELISA

Treatment and Prevention
No practical recommendation for pig treatment
Segregation and slaughter of infected breeding stock
Test and replace with healthy animals
Public health significance: Brucellosis is transmitted to humans via contact with infected materials and consumption of raw infected milk.

Infectious Polyarthritis

Porcine infectious polyarthritis is an infectious disease of swine caused by **Hemophilus suis**. The transmission is not well established.

Clinical Symptoms
Fever (40-42°C), complete anorexia, rapid shallow mouth breathing, anxious expression, extension of head, animals stand on toes and move with shuffling gate. All joints are swollen and painful on palpation. Chronic arthritis is more common in weaning pigs.

Diagnosis
Clinical signs and isolation of the organism.

Treatment and Prevention
Trimethoprim-sulphadiazine 15 mg/kg of feed, PO, q 12 h for days.

Prevention
See Diseases of Pigs: Pleuropneumonia.

Salmonellosis

It is a bacterial disease of pig caused by **Salmonella typhimurium** and **Salmonella choleraesuis**. Outbreaks may occur in pigs up to 6 month old.

Clinical Symptoms
Septicemia, anorexia, depression, fever (40.5-41.5°C), severe watery diarrhea, sometimes dysentery and often tenesmus and death occur in 24-48 hours. A dark red to purple discoloration of the skin is common, especially at the ears and ventral abdomen. Nervous signs and pneumonia may occur. Mortality may reach 100%.

Diagnosis
Clinical signs and isolation of salmonella from feces, tissues animals, feed or water supplies.

Treatment and Prevention
Trimethoprim-sulphadiazine15mg/kg, PO, q 12 h. **Or**
Ampicillin10-20mg/kg of feed, PO, q 12-24 h for 3-5 days.
Plus
Dexamethasone; 20-200mcg/kg, IM, q 6-12 h.

Note: Oral medication should be given in drinking water because affected animals are thirsty due to dehydration and their appetite is generally poor. Public health significance: Salmonella typhimurium is zoonotic.

Streptococcal Lymphadenitis

It is contagious disease of pigs transmitted by ingestion of **streptococcus** contaminated feed.

Clinical Symptoms
Abscessation of cervical and cephalic lymph nodes. Scattered miliary abscesses may also develop in other lymph nodes; within 7 days of infection.

Diagnosis
Isolation of the organism from abscess exudates and agglutination test are indicated for diagnosis.

Treatment and Prevention
Neomycin 11 mg/kg, q 24 h for 5 days. **Or**
Tetracycline; 200-400 mg/kg of feed, **Or**
Trimethoprim-sulphadiazine (80:400) 15 mg/kg, PO, q 12 h or 48 mg/kg, q 24 h, IM for 3-5days.

Prevention
In affected herds, piglets should be weaned at 21 days and reared in an environment free of older pigs.

Streptococcosis

It is a common cause of meningitis and arthritis on large, intensively managed pig farms. It is also associated with pneumonia, endocarditis, myocarditis and diseases of the genital tract in sows. Transmission occurs by close contact with clinical cases or subclinical carriers and the environment.

Clinical Symptoms
Meningitis manifested by depression, fever, tremors, incoordination, opisthotonus, convulsions, blindness and deafness. Polyarthritis and lameness are common. The skin may be reddened in patches. Lymph nodes are often enlarged and congested, fibrinous polyserositis is common. Joint capsules may be thickened and joints may contain excess clear or cloudy fluid. Often, the meninges and brain appear normal, but there may be congestion, edema and excess clear or cloudy CSF.

Diagnosis
History, clinical signs, histology and fluorescent antibody (FA) tests are important. Definitive diagnosis depends on isolation and identification of the causative organism.

Treatment and Prevention
Penicillin 10,000-20,000 IU/kg, IM, q 12 h for 3 days. **Or**
Trimethoprim-sulphadiazine; 15 mg/kg, PO, q 12 h for days.

Swine Dysentery

A contagious disease of pigs caused by **Treponema** characterized by mucohemorrhagic diarrhea affecting the large intestine. The transmission occurs by ingestion.

Clinical Symptoms
Fever, depression, inappetence, soft feces, progressive dehydration with gaunt and sunken abdomen.

Diagnosis
Clinical signs are indicative and confirmed by isolation of the organism.

Treatment and Prevention

Lincomycin hydrochloride 10-15 mg/kg, IM, q 12 hr. for 3 days.
Tylosin 8.8 mg/kg, IM, q 24 hr. for 3 days.
Tylvalosin 4.25 mg /kg BW per day in-feed for 10 consecutive days. For treatment and metaphylaxis of swine dysentery.

Swine Erysipelas

An acute septicaemic disease of swine caused by **Erysipelothrix rhsiopathiae**. Recovered pigs and those chronically infected remain carriers.

Clinical Symptoms

Acute form of the disease, septicemia, discoloration of the skin, arthritis. Vegetative endocarditis may occur in sequence or separately. Pigs with acute septicemia may die suddenly without signs. Acutely infected pigs are febrile (40-42°C), walk stiffly on their toes and lie on their sternum separately rather than piling in groups. Skin discoloration may vary from widespread erythema and purplish discoloration of the ears, snout and abdomen to diamond-shaped skin lesions almost anywhere on the body.
Chronic arthritis, the most common form of chronic infection, produces mild to severe lameness; the affected joints may be difficult to detect but tend to become visibly enlarged and firm.

Diagnosis

Response to penicillin within 24 hours supports the diagnosis. The typical diamond-shaped skin lesions, organism in stained smears or cultures confirms the diagnosis.

Treatment and Prevention:

Penicillin 10,000-20,000 IU/kg, IM, q 6 h for 3 days for acute cases. Or Tetracyclines 5-6 mg/kg, IM, q 12 h for 3 days. For Chronically ill pigs culling is better

PARASITIC DISEASES OF PIGS

HELMINTHES PARASITES

Ascariasis

Pigs are infected by **Ascaris suum.** The parasites are found principally in the small intestine but may migrate into the stomach or bile ducts. The migration of larvae in the body damages tissues.

Clinical Symptoms
Poor growth rate of young pigs, mechanical obstruction of the intestine, migration into liver may occlude the bile ducts, producing icterus. Affected pigs show abdominal breathing, unthriftiness and weight loss are common.

Diagnosis
History, clinical signs and microscopic examination of fecal smears.

Treatment and Prevention
Piperazine250-300mg/kg, PO **Or**
Ivermectin 0.3 mg/kg, PO, or SC **Or**
Fenbendazole; 5-7 mg/kg, PO for 3 days
Flubendazole 5mg/kg twice per year can be used for prevention

Prevention
Strategic deworming of the flock, rotational grazing, zero grazing (if possible) and managing the stock density.

Cysticercosis

Taenia solium is a tapeworm found in the small intestine of man. Its metacestode (larval) stage, a cysticercus is a large fluid-filled cavity or vesicle or bladder found in the musculature of pigs.

Clinical Symptoms
Infection causes pain, paralysis, epileptiform seizures, locomotor disturbances and possibly death. The coenuri commonly localize on the meninges and in the neuropil.

Treatment and Prevention
It has no specific treatment in pigs.

Prevention
Prevention of access to human feces by pigs, total confinement of pigs if integrated with other management practices such as housing and feeding with locally available materials and feedstuffs and strict meat inspection and control and cooking of pork.

Public Health significance: Humans are final hosts and could acquire the condition by eating raw pork.

Strongyloides Species

It is intestinal threadworm caused by **Strogyloides ransomi.** Transmission occurs by ingestion of larvae via the colostrum in neonatal pigs. The adult worms (only females in the parasitic cycle) burrow into the wall of the small intestine.

Clinical Symptoms
In heavy infections, diarrhea, anemia and emaciation develop and death may result.

Diagnosis
Demonstration of the characteristic small, thin-shelled, embryonated eggs in the feces or of the adults in intestinal mucosa scrapings is diagnostic.

Treatment and Prevention
Fenbendazole 5 mg/kg, PO **Or**
Ivermectin 0.3 mg/kg, PO
Plus
Treat with antibiotics to control secondary infection

Prevention
Flubendazole, 5mg/kg body weight twice per year. Ivermectin is effective against adults and treating the sow 1-2 weeks before farrowing, controls transmission to the piglets.

Stomach Worms

Three types of stomach worms occur in pigs. A thin worm, **Hyostrongylus rubidus** (the red stomach worm) and thick stomach worms, **Ascarops strongylina** and **Physocephalus sexalatus.**

Clinical Symptoms
Poor body condition, anemia, diarrhea and weight loss. In sows, inhibited larvae resume development near parturition and may cause severe gastritis and contaminate the environment of the young pigs.

Diagnosis
Fecal examinations and necropsy.

Treatment and Prevention
See treatment of Strongyloides sp.

Trichuris spp

Trichuris suis is 5-8 cm long and has a slender anterior portion and a thickened posterior third**.** Infection is by ingestion of embryonated ova.

Clinical Symptoms
Heavy infections may cause inflammatory lesions in the caecum and adjacent large intestine and be accompanied by diarrhea and unthriftiness.

Diagnosis
Demonstration of double-operculated eggs are diagnostic.

Treatment and Prevention
Levamisole10 mg/kg, PO. **Or**
Fenbendazole 5 mg/kg, PO.

Swine Kidney Worm

A worm infestation of kidney and uterine wall of pigs caused by **Stephenurus dentatus**. Transmission occurs by ingestion of earth worm which are the intermediate host.

Clinical Symptoms
Pleuritis, peritonitis and cirrhosis.

Diagnosis
Detection of eggs in urine is definitive.

Treatment and Prevention
Ivermectin, 300 mcg/kg, SC, PO, q 24 h for 3 days. **Or**
Fenbendazole 3 mg/kg, PO, q 24 h for 3 days.

Prevention
Sanitation of the area by provision of a concrete pad under the feed.

PROTOZOAL PARASITES

Coccidiosis

Eight species of **Eimeria** and one of **Isospora** are responsible to infect pigs. **Isospora suis** is prevalent in neonatal pigs.

Clinical Symptoms
Watery or greasy diarrhea, usually yellowish to white and foul smelling. Piglets may appear weak, dehydrated, weight gains are depressed and may die.

Diagnosis
Isolation of the parasite from impression smear or histological section of small intestine. In severely affected piglets, histological lesions are confined to the jejunum.

Treatment and Prevention
Amprolium 0.012-0.024%, q 24 h for 5 days in drinking water
Sulfamethazine 0.1%, q 24 h for 2-4 days in drinking water.

Prevention
Provide Decoquinate 60 gm/kg feed and 200mg/kg feed throughout the risk period and removal of feces and disinfection of farrowing facilities between litters greatly decreases infection.

EXTERNAL PARASITES OF PIGS

Sarcoptes

Mange mites infest pigs and cause high morbidity but no mortality.

Clinical Symptoms
Appearance of small nodules and pustules that forms large abscess which is cheesy in consistency loss of hair and thickened skin in that commences on the face and spread down the ventral surface of the neck and belly. Intense pruritis, erythematous spots with scales and minor exudation are common. Chronic infestation may result in to thickening and wrinkling of skin.

Diagnosis
Clinical sign and confirmed by detection of mite in skin scrape.

Treatment and Prevention
Vigorous therapy with acaricides. **Or**
Ivermectin, 300 mcg/kg, SC, PO
Doramectin 3 mg/10kg body weight IM, single dose.

Prevention
Avoid the contact of sick animals with healthy ones and disinfect house with acaricides.

RESPIRATORY DISEASES OF PIGS

Pleuropneumonia

It is a disease of pig caused by **Haemophilus pleuropneumoniae**. It is transmitted by inhalation. Overcrowding and poor ventilation facilitate the spread of the disease.

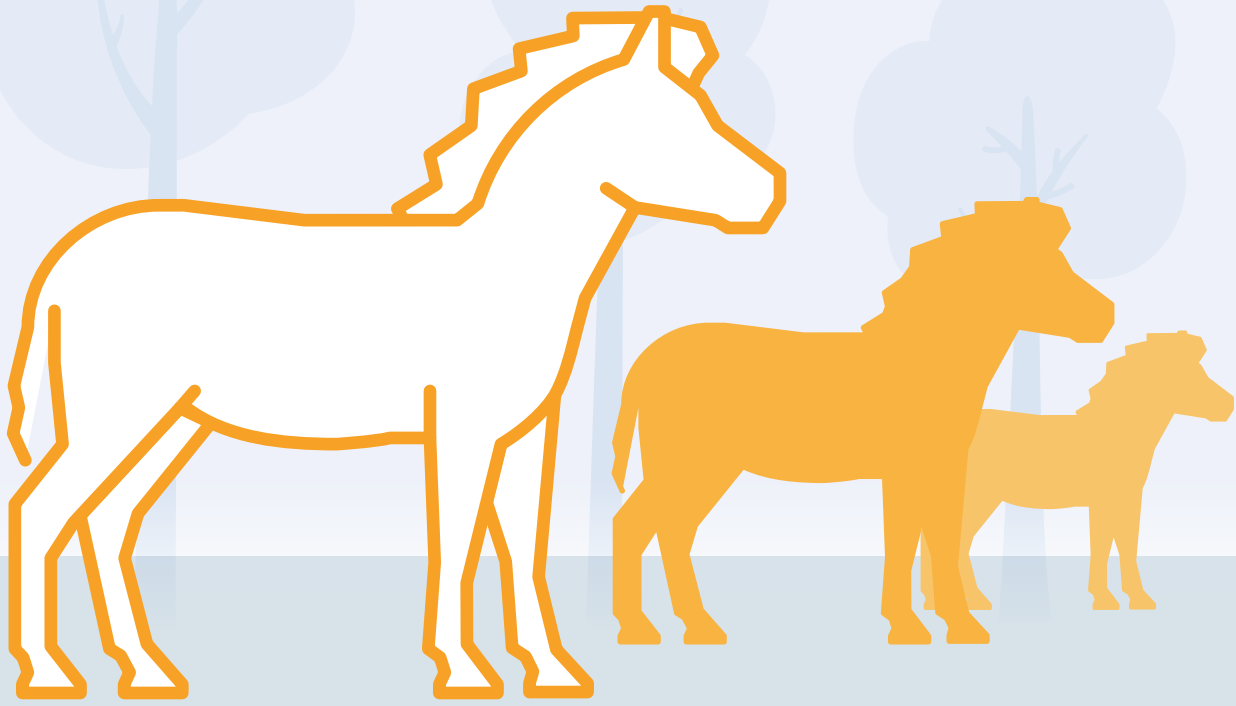
Clinical Symptom
Rapid onset, usually several death and others show respiratory problems, fever and anorexia, severe dyspnea and blood stained frothy discharge through nose and mouth.

Diagnosis
Clinical signs are suggestive isolation is confirmatory.

Treatment and Prevention
Lincomycin hydrochloride 10mg/kg, IM, q 12 h for 3-5 days. **Or**
Oxytetracycline, 500 mg/kg of feed for 7 days. **Or**
Trimethoprim-sulphadiazine (80: 400)15 mg/kg, PO, q 12 h for days.

Prevention
Provide Chlortetracycline or Oxytetracycline mixed with feed 20mg/kg of feed, q 24 h for 7 days.

SECTION IV



DISEASES OF EQUIDAE[4]

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index

NON INFECTIOUS DISEASES OF EQUIDAE

Colic in Equines

Colic or abdominal pain occurs when the wall of the intestine is stretched excessively either by gas, fluid or ingesta. Excessive tension on the mesentery, as a result of incarceration or severe twisting of the intestine or inflammation that may involve either the entire intestinal wall (enteritis) or the covering of the intestine (peritonitis) can cause it.
The common causes of colic include the following:
In foals: atresia coli, meconium retention, uroperitoneum and gastroduodenal ulcers
In yearlings: ascarid impaction
In the young horses: small-intestinal intussusception, nonstrangulating infarction and foreign body obstruction
In the middle-aged horses: epiploic foramen, cecal impaction, enteroliths and large-colon volvulus; and
In the aged horses: pedunculated lipoma and mesocolic rupture. Migratory larvae of blood worms of **Strongylus vulgaris,** are common causes of colic.

Clinical Symptoms
Pawing repeatedly with a front foot, looking back at the flank region, curling the upper lip and arching the neck, repeatedly raising a rear leg or kicking at the abdomen, lying down, rolling from side to side, sweating, stretching out as if to urinate, straining to defecate, distention of the abdomen, loss of appetite, and decreased number of bowel movements. Donkeys do not show overt clinical signs at early stage, may present as inappetence and dullness.

Diagnosis
For presumptive diagnosis use history of any previous problems or treatments, determining which part of the intestinal tract is involved. Rectal examination, nasogastric intubation and fecal sample test for parasite and bacteria are useful techniques.

Treatment and Prevention
Flunixin meglumine;1.1 mg/kg, IM, or IV or PO stat, **Or**
Phenylbutazone 4.4 mg/kg PO or IV q24 hr. for 10 days. In Donkeys use 2.2 mg/kg PO or IV q12 hr. **Or**
Ketoprofen 2.2 mg/Kg PO or IV once or repeat after 24hr. for abdominal pain. **Or**
Xylazine 1.1 mg/kg, IV or 2.2 mg/kg IM.
For parasitic coli
Ivermectin 0.2 mg/kg, PO stat. for SE, CI, D/I, D/F, see Annex 2 or
Fenbendozole;10 mg/kg, PO q 24h for 5 days or 20 mg/kg, PO q 24hr. for 3 days.

Prevention
Control and prevention depends on etiology and for parasitic colic, routine deworming of horses every 8 weeks with ivermectin 0.2 mg/kg, SC or PO Or Fenbendazole 5 mg/kg, PO or Pyrantel pamoate 6.6 mg/kg every 4 weeks.
In addition prevention also requires regular access to water and feed; avoid access to moldy feed and foreign body such as polythene bags, avoid overworking.

Note: Almost all require some form of medical treatment but only those with certain mechanical obstructions of the intestine need surgery.

Lameness in Equines

Lameness is a deviation from a normal stance or gait. It is an indicator of a structural or functional disorder of the musculoskeletal system that is noted while the animal is either moving or stationary. Lameness is not a disease but an indication of pain, weakness or other impediment in the musculoskeletal system. Other factors unrelated to the musculo-skeletal system, such as metabolic, circulatory and nervous system abnormalities can cause lameness.

Clinical Symptoms

Clinical symptoms depend on the type/cause of lameness. Include pain, increased digital pulse, abnormal gait, incoordination, shifting of weight from lame foot, nodding of head, exhaustion, sweating, tendency to lie down, abnormal wear of hoof, discharge or bad smell swellings in joints or tendons, atrophy or swelling of muscles.

Diagnosis

In all cases of musculoskeletal pain and lameness, diagnostic procedures must be performed to determine the nature, extent and exact location of the injury. Diagnostic imaging techniques such as radiography, ultrasonography, MRI, CT, nuclear imaging and thermography can be used to evaluate the soft-tissue structures and bones. When joint sepsis is suspected, analysis of synovial fluid of the affected joints is necessary. Refer to grade set by the American Association of Equine Practitioner's (AAEP) for further classification of the lameness.

Treatment and Prevention

Flunixin Meglumine 1.1mg/Kg q 8-12 h I/V or I/M. **Or**
Butrophanol (Turbogesic tartarate) 0.1mk/kg IV, repeated at 3-4 hrs interval. **Or**
Ceftiofur 20ml of the 50mg/ml q 24 h, Regional perfusion in synovial infection
Phenylbuthazone 2.2-4.4 mg / kg q 8-12h PO or IV.
Carprofen 0.7-1.3 mg / Kg q 24h IV or PO.
Dexamethasone 0.05-2.0 mg/ kg q 24h I/V, I/M or PO.

Non drug Treatment

Conservative therapy – Provide rest as much as possible, restrict activity, immobilization of diseased or injured structures in splints and casts

Removal of the underlying cause: As it has diverse causes, identifying and eliminating the underlying cause is very important. E.g. If the cause is solar abscess – drain pus and treat foot, if overgrown hoof – trim hoof, if bad ferrying – correct the shoe

Surgical Repair - if there are fracture and tendon problems

Cold Therapy [Compression or hosing] - to treat inflammatory problems such as acute tendonitis and desmitis. Once a day for three to five days

Extracorporeal Shock Wave Therapy (ESWT), to treat chronic conditions like arthritis

Joint injection with various regenerative and anti-arthritic compounds such as **Hyaluronic Acid (HA), Interleukin-1 receptor antagonist protein (IRAP®) or** autologous conditioned serum (ACS), **Platelet-rich plasma (PRP)**

Educate owners how to recognize the early signs of lameness. Daily checking and cleaning of hooves, keeping dry and hygienic stables. Regular hoof trimming and proper shoeing. Limit work load and maintain good and balanced load. Use properly designed carts with symmetrical and inflated tyre and good harness. Provide good nutrition and avoid loading of immature equids.

Note: Treatment for any lameness will vary depending on the exact nature of the lameness and type work the animal does.

Lightning Stroke and Electrocution

Injury or death of an animal due to high-voltage electrical currents may be the result of lightning, fallen transmission wires, faulty electrical circuits or chewing of an electrical cord.

Clinical Symptoms and Lesions
Occasionally, the animal becomes unconscious but may recover in a few minutes to several hours; residual nervous signs (e.g. depression, paraplegia, cutaneous hyperesthesia) may persist for days or weeks or be permanent. Burn-marks on the carcass are more commonly found on the medial sides of the legs, although rarely much of the body may be affected; damage to the immediate environment may be seen.

Diagnosis
History of a recent storm together with finding a dead or injured animal under a tree or near a fence. Rigormortis develops and passes quickly. The mucosae of the upper respiratory tract including the turbinates and sinuses are congested and hemorrhagic, large blood clots are occasionally found in the trachea.

Treatment and Prevention
Diphenhydramine hydrochloride, 100-200mg, IM or IV or SC
Dopamine hydrochloride 4.4-11.1 µg/Kg/minute, IV in case of electric shock or cardiac problems
Lactated ringer's solution 35 to 40ml/kg, q 24 h, IV and oxygen therapy for surviving animals

Note: Intubation may be required, cardiorespiratory resuscitation while monitoring vital signs. Antibiotic treatments with similar dose as for other infections may help.

VIRAL DISEASES OF EQUIDAE

African Horse Sickness

African Horse Sickness (AHS) is an acute or subacute, arthropod-borne viral disease of **Equidae** that is endemic in Ethiopia. Mortality can reach up to 95% in horses. It is caused by virus of the genus **Orbivirus** and outbreaks occur after heavy rains. Transmission is by culicoides biting midges. Other potential vectors include mosquitoes, flies and ticks.

Clinical Symptoms
Four forms exist: Respiratory form, cardiac form, mixed form and "horse sickness fever". Incubation period is 3-5 days [acute] up to 2 week in cardiac form, interlobular edema, fever (40-42°C) dyspnea, spasmodic coughing and dilated nostrils, frothy nasal discharge, the animal stands with its legs apart and head extended. The conjunctiva is congested and the supraorbital fossa swollen. Swelling usually extends to the eyelids, facial tissues, neck, thorax, brisket and shoulders in cardiac form. Recovery is rare and death is due to anoxia. African horse sickness is usually sub-clinical in donkeys ["Horse sickness fever"], but mild clinical signs that include fever, weakness, dyspnea and extensive swelling of supraorbital fossa and eyelids may be observed.

Diagnosis
In endemic areas, clinical signs and lesions may lead to a tentative diagnosis. Serology and culture are confirmatory. Differential diagnosis include Bacterial pneumonia, Strangles, Equine herpes virus, Anthrax and Piroplasmosis.

Treatment and Control
No specific treatment.

Prevention
Vaccination with polyvalent or specific serotype based vaccine before outbreak season,
Reduce exposure to biting insects using repellents.

Equine Infectious Anaemia

It is a mild viral disease caused by a Lentivirus affecting equine species. The virus is transmitted by hematophagous insects.

Clinical Symptoms

Usually mild and related to the host immune response rather than to direct viral damage. These include fever, depression and petechiae on mucous membranes and conjunctiva. Recovered animals may show chronic form of the disease characterized by weight loss, anaemia, ventral oedema and debilitation leading to death.

Diagnosis

Agar gel diffusion test and ELISA.

Treatment and Prevention

There is no specific treatment

Prevention and control include Test and cull of animals before introduction to an EIA free farm and Insect control.

Rabies

Rabies is an acute viral encephalomyelitis that principally affects carnivores and insectivorous bats, although it can affect any mammal. It is almost invariably fatal once clinical signs appear. It is endemic in Ethiopia. For its epidemiology **see** Disease of dogs and cats -Rabies.

Clinical Symptoms

Most signs are similar to those in pets. Horses and mules frequently show evidence of distress, abnormal vocalization and extreme agitation. These signs, especially when accompanied by rolling, may be interpreted as evidence of colic. As with other species, horses and donkeys may bite or strike viciously and because of size and strength, become unmanageable in a few hours. Such animals frequently suffer self-inflicted wounds. Owners mostly complain of lameness or colic. Self-mutilation at extremities and scrotum is common.

Diagnosis

Clinical diagnosis is difficult. Therefore, when rabies is suspected and definitive diagnosis is required, laboratory confirmation is indicated.

Treatment and Prevention

No specific treatment for rabies.

Dog rabies control

Public health significance: Rabies is highly fatal zoonotic disease transmitted through bite of infected animals.

BACTERIAL DISEASES OF EQUIDAE

Actinobacillosis

Actinobacillosis in horses is caused by **Actinobacillus equuli**. It almost always involves soft tissue, including lymph nodes through which the organisms frequently spread, but also may involve adjacent bony tissues. Infection occurs through a contaminated umbilicus, inhalation or ingestion.

Clinical Symptoms
In young foals; diarrhea, which may be followed by meningitis, pneumonia, purulent nephritis, or septic polyarthritis (sleepy foal disease or joint ill) are common symptoms. Clinical features in adult horses include abortion, septicemia, nephritis and endocarditis.

Diagnosis
Clinical findings are suggestive and confirmed by microscopic examination from the exudates and by culture.

Treatment and Prevention
Non-drug treatment
Surgical debridement and 10% potassium iodide PO.
Drug Treatment
Oxytetracycline 5%, 5 mg/kg, q 12 h, IV or IM for 3-7 days. **Or**
Procaine Penicillin G, 20,000 IU/kg, q 12h, IM for 3-7days. **Or**
Potassium iodide 10% PO With Iodine tincture (Iodine 2%) applied topically

Anthrax

Anthrax is an acute, febrile disease caused by **B. anthracis.** Most commonly, it is a septicemic disease characterized principally by a rapidly fatal course. It is reported in all domestic animals and humans all over Ethiopia. The epidemiology is similar to that in cattle.

Clinical Symptoms
The disease in horse is acute. Signs may include fever, chills, severe colic, anorexia, depression, weakness, bloody diarrhea and swellings in neck, sternum, lower abdomen and external genitalia regions. Death usually occurs within 2-3 days of onset.

Diagnosis
Clinical signs, epidemiology, methylene blue stained blood smear, Western blot and ELISA. Culture requires extreme care since aerosol transmission may occur.
Differential diagnosis: Acute infectious anemia, Lead poisoning, African Horse Sickness, Lightning strike, sunstroke and Colic.

Treatment and Prevention
Penicillin G Procaine, 22,000 IU/kg, q 12 h, IM or 44,000 IU/Kg, q 24 h, IM for 3-5 days in the early stages of the disease.
Alternative
Penicillin and streptomycin sulphate (200,000IU/250mg), 1ml/20 Kg, IM for 3-5 days. **Or**
Oxytetracycline hydrochloride 5%, 11 mg/kg, q 12 h for 3-5days.

Prevention
Annual vaccination of animals and proper disposal of carcasses.
Precautions: Antibiotics should not be administered within 1 week of vaccination.
The carcass of animal that suddenly died should not be opened. It should be burned or buried deep.

Conjunctivitis (Pink Eye)

Conjuctivitis is a very common condition affecting horses' eyes, caused by bacterial, viral and irritants. Moraxella is the common cause of conjunctivitis. **Pseudomonas aeruginosa** and **Sterptococcus equi subsp. zooepidermicus** are also known to infect and cause corneal ulcers and lead to keratitis.

Clinical Symptoms

There are variable degrees of swelling of the eyelids, painful discomfort, purulent exudate, inflammation and hyeraemia of the conjuctiva and photophobia.

Diagnosis

Clinical signs: Corneal ulceration and recurrent uveitis (periodic ophthalmia) and biopsy.

Treatment and Prevention

Non drug treatment

Mechanical removal of the worms

Drug treatment

Gentamycin eye drops, 10 drops q 6h for 5 days. Prolonged use is prohibited. **Or**

Chlortetracycline hydrochloride eye oint. Apply 2-3 cm of ointment in the conjunctival sac 4 times a day for 5 days.

Alternative (If no improvement after 3 days of treatment)

Fusidic acid eye drops. 2-3 drops q12 h for 5 days. **Or**

Cloxacillin benzathine 16.7%, ointment.

Ivermectin in case of thelaziasis,

2% Boric Acid drop

Glanders

Glanders is caused by **Burkholderia mallei** formerly **Pseudomonas mallei**. It is a contagious disease of Equidae characterized by the formation of nodules and ulcers in the respiratory tract or on the skin. Transmission occurs following ingestion of food or water contaminated by nasal discharges of infected animals; less commonly through inhalation and skin abrasions.

Clinical Symptoms

Acute form: fever, mucopurulent nasal discharge and respiratory signs. Chronic form- nasal ulcerative nodules in the nasal septum and lower turbinates, blood stained nasal discharge and regional lymphadenopathy and ulcers with star-shaped scars. Pulmonary form - respiratory distress and nodular lesions in the lung. Cutaneous form-nodules along the lymphatic vessels of the legs.

Diagnosis

Clinical manifestations are indicative. Culture examination, complement fixation test, agglutination test and mullein test.

Treatment and Prevention

No completely satisfactory treatment is known.

If applied early, sodium iodide and potassium iodide, 10%, IV for at least a week. Good results are obtained if combined with surgical incision of wounds. **Or**

Surgical excision of lesions combined with Amphotericin B ointment q 6-12 h.

Note: Glanders is zoonotic disease handle with care.

Salmonellosis

Salmonellosis is caused by many serotypes of **Salmonellae** and characterized by septicemia and acute and chronic enteritis. Young foals usually develop the septicemic form. Adult horses and donkeys commonly develop acute enteritis. The main causes are **S. typhimurium, S. anatum, S. newport, S. enteritidis** and **S. arizonae**. The common route of infection is oral. The disease occurs after stress of any cause.

Clinical Symptoms

Septicemic; usual syndrome in newborn foals is acute, depression is marked, fever (40.5-41.5°C) is usual, and death occurs in 24-48 hr. Acute enteritis; abdominal pain and severe in horses, they are severely dehydrated and may die within 24 hours from the onset of diarrhea. Subacute enteritis; occurs in adult horses. The signs include mild fever (39-40°C), soft feces, inappetence and some dehydration.

Diagnosis

Clinical signs are indicative but confirmation is based on the culture examination.

Treatment and Prevention

Sulfadiazine Trimethoprim 200/40 mg/ml, 25+5mg/kg q 24 hr. IM for 6 days. **Or**
Ampicillin; 6.6 mg/kg q 12h, IM or IV for 6 days **Or**
Cephalothin, 20-35 mg/kg, IM or IV q 6 to 8hr. for six days.
For acute intestinal salmonellosis, sodium bicarbonate with sodium and potassium chloride, PO, 100gm/450 kg body wt.

Strangles

"Strangles" is contagious bacterial respiratory disease of horses caused by obligate intracellular bacteria, **Streptococcus equi var. equi**. It is characterized by inflammation of nasal and pharyngeal epithelium, followed by lymphadenitis and abscessation of the lymphnodes. Species affected are horses, donkeys, mules and camels. Young (3 months to three years) are more affected. Infection is by inhalation or ingestion of the organism with subsequent localization in the mandibular and pharyngeal lymph nodes.

Clinical Symptoms

Submandibular swelling, inappetence, depression, dysphagia, prominent cough, progressive difficulty in swallowing, purulent nasal discharge, enlarged submandibular/parotid lateral and medial retropharyngeal lymph nodes. The lymphnodes, later rupture and discharge purulent material. There will be salivation, choke and fever (38-39.5°C).

Diagnosis

Clinical signs are highly indicative and confirmed by culture.

Treatment and Prevention

Non Drug Treatment.

Nursing with soft feeds, hot compress or topical ichtamol 20% on immature abscesses
Lance/drain mature abscesses Bath conjunctivae/abscess sinus tracts and Tracheotomy in case of severe dyspnea

Drug Treatment

Procaine penicillin 20,000 – 44,000 IU/kg q 12h, IM for 7-10 days. **Or**
Penicillin 20,000IU and dihydrostreptomycine sulphate 250mg 1ml/25kg, q 24 h, IM for 3-5 days/ **Or**
Trimethoprim-Sulphamethoxazole 30 mg/kg q 12h , PO fro 3-5 days
Ceftiofur 2.2 – 4/4 mg/kg IV or IM q 12 h for 3-5 days
Plus
Flunixin Meglumine 1.1mg/Kg q 8-12 h IV or IM , **Or**
Phenylbuthazone 2.2-4.4 mg / kg q 8-12h PO or IV, **Or**
Carprofen 0.7-1.3 mg / Kg q 24h IV or PO,
For Purpura haemorrhagica requires aggressive long-term treatment with penicillin 20,000 IU/kg daily for more than 6 weeks and dexamethasone 0.1-0.2 mg/Kg IV or IM q12-24

Prevention

Avoid direct contact of infected animals with others, apply strict hand hygiene, cleaning and disinfection practices at clinics to reduce risk of transmission.

Note: Guttural pouch empyema needs surgical drainage of both pouches.

Tetanus

Tetanus toxemia is caused by a specific neurotoxin produced by **Clostridium tetani**. In Ethiopia, the incidence of tetanus in horses and donkeys is more common than in any other species. Infection most commonly occurs after an injury and puncture wounds but may also develop after surgical operation. The epidemiology is similar to tetanus in cattle.

Clinical Symptoms

Stiffness, often involving masseter muscles and muscles of neck, hind limbs and ears are erect, tail gets stiff and extended, anterior nares are dilated, walking and turning back are difficult, prolapse of the third eyelid. Sweating is common. General spasms interfere with circulation and respiration, which results in increased heart rate, rapid breathing and congestion of mucous membranes. Usually there is little or no fever except in severe cases and just before death. Recumbency is indicative of poor prognosis.

Diagnosis

The clinical signs and history of recent trauma are usually adequate for a diagnosis of tetanus. Confirm the diagnosis by demonstrating the presence of tetanus toxin in the serum, and gram-stained smears and anaerobic culture from suspected wounds. Differential diagnosis: Colic, Laminitis, Hepatic encephalopathy, Meningitis, Electrolyte imbalance and Organophosphate poisoning.

Treatment and Prevention

Non-drug treatment

The horse should be placed in a quiet, darkened box stall with feeding and watering devices high enough to allow their use without lowering the head

Sufficient bedding to allow the animal to rest comfortably and prevent self-inflicted injuries

If the horse is dysphagic, hydration and feeding should be done by nasogastric intubation and/or intravenous infusion

Urinary catheterization for bladder emptying, enemas and manual rectal evacuation of feces.

Drug treatment:

Tetanus antitoxin, 15,000 IU (15ml), IV and 8,000 IU (8ml), IM to an average (120kg) weighing Ethiopian donkey and 25,000 IU, IV or 12,000 IU, IM to an average (260kg) horse.

Acepromazine, 0.05-0.09 mg/kg, IV or IM **Or**

Xylazine;1.1-2.2 mg/kg, IV or IM in conjunction with 300,000 IU of tetanus antitoxin q 12 h, SC, IV or IP.

Cautions: Don't use for horses with pulmonary diseases.

Plus

Debriding and cleaning the wounds with Hydrogen peroxide.

Injection of maximum dose of short acting penicillin, 4,000,000 IU suspension of fortified procaine penicillin G, q 24h for 5days. Or Phenylbutazone 4.4 mg/kg PO or IV q24 h, for 10 days. In donkeys use 2.2 mg/kg PO or IV q12 h.

Prevention

Management of contaminated or necrotic wounds should include thorough debridement, large-volume flushing and comprehensive cleansing.

Rational antibiotic therapy should be instituted for all contaminated and at-risk wounds.

Toxoid should be given simultaneously with the antitoxin and repeated in 30 days. Yearly booster injections of toxoid are advisable.

Mares should be vaccinated during the last 6 weeks of pregnancy and the foals at 5-8 weeks of age. In high-risk areas, foals may be given tetanus antitoxin immediately after birth and every 2-3 weeks until they are 3 months old, at which time they can be given toxoid.

PARASITIC DISEASES OF EQUIDAE

HELMINTHES PARASITES

Lungworm Infection (Verminous Pneumonia)

Is an infection of lower respiratory tract of horses and donkeys with the parasite nematode **Dictyocaulus arnfieldi** which causes pneumonia or bronchitis. The larva of the parasite may be demonstrated in faeces of the animal.

Clinical Symptoms
Coughing may be moderate to severe and persistent. Coughing, tachypnea and unthriftiness in older horses but few if any signs in foals or donkeys.

Diagnosis
Clinical signs, epidemiology and presence of first-stage larvae in feces. **D. arnfieldi** infections in horses from bronchial larvae.

Treatment and Prevention
For Treatment see under disease of cattle.

Prevention
Strategic deworming before and after the rainy season and pasture management or rotational grazing.

Onchocerciasis

Onchocerca cervicalis localizes in the ligamentum nuchae and possibly other sites in Equidae. The microfilariae are found in the dermis and rarely in circulating peripheral blood.

Clinical Symptoms
In horses it has been associated with fistulous withers, poll evil, dermatitis and uveitis. Adults in the ligamentum nuchae induce inflammatory reactions resulting in marked fibrosis and mineralization common in older horses. Larger numbers of microfilariae may be found in the skin without causing dermatitis. In some animals skin lesions such as scales, crusts, ulceration, alopecia and depigmentation and pruritis may be observed.

Diagnosis
Microscopic examination of filarial worms from skin biopsy.

Treatment and Prevention
Ivermectin 0.2-0.3mg/kg I/M or S/C. **Or**
Moxidectin 0.4 mg/kg kills the larvae.Plus
Dexamethasone 2.5 to 5 mg/450kg, I/M.
Most cases resolve with one treatment. Except for emergency therapy, do not use dexamethasone in animals with chronic nephritis and hypercorticalism (Cushing's syndrome)
Do not use dexamethasone in viral infections during the viremic stage

Gastrointestinal Helminthiasis

Gastrointestinal parasites affecting horses include **oxyuris** species, **Parascaris equorum**, large strongyles including **S. vulgaris, S. edentatus** and **S.equinus**; small strongyles like **S. tenuicolis**, **Strongyloides** species and **Trichostrogylus** species. The migratory larval stages of most of these parasites cause severe damage in vital organs of thoracic and abdominal cavity.

Clinical Symptoms
Large strongyles cause anemia, weakness, emaciation and diarrhea. **Strongylus vulgaris** causes colic, gangrenous enteritis or intestinal stasis, torsion or intussusception and possibly rupture. In heavy **P. equorum** infestation, unthriftiness, loss of energy and occasionally colic may be observed**. T. tenuicollis** produces severe ulcers in the wall of the colon. Others disturb digestive and absorptive function, resulting in catarrhal enteritis of the large intestine.

Diagnosis
Microscopic examination of feces for egg and there are also serological tests.

Treatment and Prevention
Ivermectin 200µg/kg, PO or SC stat. **Or**
Moxidectin 0.4 mg/kg, PO or SC stat. **Or**
Albendazole 7.5mg/kg PO stat. **Or**
Fenbendazole at 10 mg/kg q 24h, PO for 5 days stat. **Or**
Pyrantel pamoate 6.6 mg/kg, PO stat

Prevention
Strategic treatment; at the beginning of rainy season and at the end of rainy season. Pasture management or rotational grazing. Ascaris prophylaxis at age of ~8 weeks old and repeated at 6- to 8-weeks interval until they are yearlings.

ARTHROPOD PARASITES

Gastrophilus spp. (Bot Fly)

Eggs of **G. intestinalis, G. nasalis** and **hemorrhoidalis** are deposited mainly between the knee and hoof, intermandibular space and hairs of lips and around the mouth. Larvae are found on cardiac portion of the stomach, pylorus region of the stomach and duodenum. Larvae can also be found attached to the rectum. **Rhinoestrus** spp. affects donkeys and is found in the nasal cavity.

Clinical Symptoms
Distress, dermatitis, inflammation of the pharynx, esophagus, stomach, rectum or rectal prolapse and problems of pyloric sphincter are among the signs. In donkeys the larvae causes dysphagia.

Diagnosis
Observation of cream-white bot eggs, dysphagia in donkeys and examination of rectal mucosa for larvae is necessary for diagnosis.

Treatment and Prevention
Ivermectin, 0.2mg/kg Oral powder, or 1% S/C for larvae treatment
Adult fly control
Fenvalerate spray, dilute before use as per manufacturer's recommendation on the container.
Diazinon spray, dilute before use as per manufacturer's recommendation on the container.
Cautions: Provide adequate ventilation for operator during spraying.
Avoid direct contact with milk and milking machine.

PROTOZOAL PARASITES

Equine Piroplasmosis

It is caused by **Babesia caballi** and **Theileria equi**. The pathogens are transmitted by ixodid tick species.

Clinical Symptoms
Fever, petechial hemorrhages of mucus membrane, anemia, hemoglobinuria, icterus and ecchymosis of the third eye lid are common. Other clinical signs include extensive subcutaneous oedema of the eyelids, ventral abdomen, genital organs and legs. Often high case-fatality rate for **T. equi** but not for **B. caballi.**

Diagnosis
Microscopic examination of Giemsa stained blood smears and presence of vector in the environment.
Differential diagnosis: Equine monocytic ehrlichiosis, Equine infectious anemia, Liver failure and other causes of hemolytic anemia.

Treatment and Prevention
Non Drug Treatment
IV Fluid as supportive
Drug Treatment
Diminazene aceturate **B. cabali** 5mg/kg IM, twice q 24 hr. interval and for **B. equi** 6-12 mg/kg IM, twice q 24 hr. interval; inject at multiple sites, **Or**
Imidocarb diproprionate 2.2 mg/Kg q 24 x 2 for **B. caballi** and 4 mg/Kg q 72hr. x 4 for **B. equi** IM
Indoxacarb 200mg Spot-on Or Dichlorovos (0.2%) + Fenitrotione (0.8%) spray topically for the tick infestation
Flunixin Meglumine 1.1mg/Kg q 8-12 hr. IV or IM.

Prevention and control
Tick control.

Trypanosoma evansi Infection (Surra)

Disease is often acute in horses but tends to be more chronic in donkeys and mules.

Clinical Symptoms
Animals become emaciated, anemic and oedematous plaques appear on the ventral surface of the body. Invasion of the central nervous system leads to meningitis. Paralysis of the hind limb occurs and the animal experiences difficulty in standing.

Diagnosis
Thin blood film for **T. evansi.**
Differential diagnosis: chronic parasitism, African Horse Sickness, Dourine, Equine viral arteritis, Equine infectious anaemia.

Treatment and Prevention
See, treatments in **Trypanosoma equiperdum** infection below.
Prevention: Control measures are aimed at the host rather than vector including detection and treatment of infected animals, prophylactic treatment of susceptible animals and protection of animals from biting flies.

Trypanosoma equiperdum Infection (Dourine)

T. equiperdum is transmitted venereally. Trypanosomes penetrate the genital mucosa and develop at the site of entry for up to 3 months.

Clinical Symptoms

Within a month of infection there is oedema of the vulval labia in the mare and of the scrotum and penis sheath in the stallion. In the stallion, persistent oedema can enlarge the scrotum and sheath 2-3 times normal size. Ventral oedema can persist for long periods and may fluctuate throughout infection.

Skin plaques, often containing trypanosomes are considered to be a pathognomonic feature of dourine. Pyrexia is intermittent and development of nervous signs may be observed. Animals with nervous involvement become emaciated and paralysis of the hind limb and muscles of the head region.

Diagnosis

It depends on clinical signs and confirmation is by complement fixation test.

Treatment and Prevention

Suramin 10 mg/kg IM stat. **Or**

Diminazene aceturate 3.5 mg/kg IM stat. **Or**

Isometamidium 0.5 mg/kg, IM stat (curative treatment), (See also Diseases of cattle; Trypanosomosis), **Or**

Quinapyramine sulphate 7.5mg/kg, SC, stat

Control and prophylaxis:

Isometamidium 0.5 mg/kg, IM stat is also used for prophylaxis and castration of infected male is effective.

Prevention of dourine is based on the establishment of freedom from infection (testing and culling). Identification of chronic carrier by CFT and castration of positive stallions is the most successful prevention and eradication method. All equids in an area where dourine is found should be quarantined and breeding should be stopped for 1 to 2 months while testing continues.

ECTOPARASITES

Sarcoptes

Sarcoptes scabiei equi is the parasite that affect horses.

Clinical Symptoms

Intense pruritis due to hypersensitivity to mite products that appear on the head, neck and shoulders. Regions protected by long hair and lower parts of the extremities are usually not involved. Lesions start as small papules and vesicles that later develop into crusts. Alopecia and crusting spread and the skin becomes lichenified, forming folds. If untreated, the lesions may extend over the whole body, leading to emaciation, general weakness and anorexia.

Diagnosis

Biopsy may establish a diagnosis. Negative skin scrapings do not rule out the disease.

Treatment and Prevention

1. Diazinon 0.06% sponging q 12-14 days for at least 3-4 days. **Or**
2. Ivermectin at 0.2mg/kg, q 2-3 weeks.

Note: Amitraz is contraindicated in horses because it can cause severe colic and death

It is important to treat all in contact animals.

Psoroptic Mange

Psoroptes equi produces lesions on thickly haired regions of the body, such as under the forelock and mane, at the base of the tail, under the chin, between the hind legs and in the axillae. **Psoroptes cuniculi** can sometimes cause otitis externa in horses and may cause head shaking.

Clinical Symptoms
Pruritus is characteristic. Lesions start as papules and alopecia and develop into thick, hemorrhagic crusts.

Diagnosis
Mites are more easily recovered from skin scrapings compared with sarcoptic mange.

Treatment
It is similar to sarcoptic mange.

Lice Infestation

Common lice of equine are **Haematopinus asini** and **Damanilia equi**. Lice are host-specific. They have a particular adverse effect on young animals and those in poor condition or subjected to stress.

Clinical Symptoms
Unthriftiness and anemia.

Diagnosis
Examination of the skin.

Treatment
Similar to sarcoptic mange. Treat all in-contact animals
Spray on rugs, harness and saddler.

Tick infestation

Important tick species to equidae are **R. evertsi, Boophilus, Amblyoma,** and **Hyalomma**. Ticks can act as a vector for pathogenic organisms.

Clinical Symptoms

Ticks can cause deterioration of condition in equines resulting from loss of blood, tick worry and tick toxicosis. Abscesses or deep-seated suppurating wounds may form which may also be infested with maggots.

Diagnosis
Clinical Symptoms and the presence of ticks on the body.

Treatment and Prevention
Diazinon spray or sponging q 12-14 days for at least 3-4 days. **Or**
Chlorfenvinphos spray and sponging every 2 weeks. Spraying of animals with organophosphates before season of high tick infestation as a control measure.
Cautions: Not approved for sheep scab control.

FUNGAL DISEASES OF EQUIDAE

Aspergillosis

Aspergillosis is caused by a number of **Aspergillus** spp, especially **A. fumigatus**. In horses, epistaxis and dysphagia are common complications of gutturomycosis.

Clinical Symptoms
The infected guttural pouch is characterized by a necrotizing inflammation which is thickened, hemorrhagic and covered by a friable pseudomembrane. Mycotic rhinitis is characterized by dyspnea and nasal discharge that could be rapidly fatal associated with diffuse pulmonary invasion. Locomotor and visual disturbances, including blindness, may occur when the infection spreads to the brain and optic nerve.

Diagnosis
The agar-gel double-diffusion test is indicative and ELISA is confirmatory.

Treatment and Prevention
Natamycin 10% in affected eye q1-2 h then 1drop q 3-4 h after 3-4days. **Or**
Potassium iodide; 10%, q 24 h, PO, 1-2 weeks. S/E and D/F **see** Annex 5. **Or**
Itraconazole; 3 mg/kg, q 12 h for 84-120 days and D/F: 10% ointment.

Prevention
Hay should be sufficiently dry and care has to be given to silage making and storage.

Candidiasis

Candidiasis is a localized mucocutaneous disease caused by fungus, **Candida albicans**. It is an opportunistic pathogen. Factors associated with candidal infections are disruption of mucosal integrity, indwelling intravenous or urinary catheters, long term administration of antibiotics and immunosuppressive drugs or diseases.

Clinical Symptoms
Lesion may be single or multiple, raised, circular, white masses covered with scabs. Marked keratinous thickening of the mucosae of the tongue, esophagus and rumen may be observed. Candida species also causes arthritis in horses.

Diagnosis
Microscopic examination or culture of scrapings or biopsy specimens from mucocutaneous lesions.

Treatment and Prevention
Ketoconazole 10 mg/kg, q 24 h, PO for 5-7days. **Or**
Itraconazole, 3 mg/kg, q 12 h, PO for 84-120 days. **Or**
Fluconazole , 4mg/ kg , q 12h, PO for 4-8 Weeks,
Natamycin ointment 10%

Dermatophytosis

Trichophyton equinum and **T. mentagrophytes** are the primary causes of ringworm in horses. Transmission is by direct contact or by grooming implements. Most lesions are seen in the saddle and girth areas known as "girth itch."

Clinical Symptoms
Patches of alopecia and erythema, scaling and crusting; early lesions may resemble papular urticaria.

Diagnosis
Examination of skin scraping and culture is confirmatory.

Treatment and Prevention
Miconazole 1% cream or lotion q 24 h for 2-4weeks topically **Or**
Chlorhexidine solution, 0.5% rinse **Or**
Chlorhexidine Gluconate (0.2%)+ Miconazole nitrate (0.2%) shampoo
Povidine-iodine, 0.5% rinse

Prevention
Disinfecting grooming or harnessing equipment, isolation of infected animals is important for the control and prevention.

Epizootic Lymphangitis

It is chronic granulomatous disease of the skin, lymph vessels and lymph nodes of the limbs and neck of Equidae caused by the dimorphic fungus **Histoplasma farciminosum**. In Ethiopia it is widely distributed and is most common in cart horses. Infection is acquired by wound infection or by blood-sucking insects. Harness sharing may transmit the disease.

Clinical Symptoms
It is characterized by freely movable cutaneous nodules, which originate from infected superficial lymph vessels and nodes and tend to ulcerate and undergo alternating periods of discharge and closure. Affected lymph nodes are enlarged and hard. The skin covering the nodules may become thick, indurated and fused to the underlying tissues.

Diagnosis
The clinical features are highly suggestive. Microscopic examination of exudates using giemsa or gram stain is important.

Treatment and Prevention
If applied early, sodium iodide 10% and potassium iodide 10%, IV for at least one week. Good results are obtained if combined with surgical incision of wounds. or
Surgical excision of lesions combined with Amphotericin B ointment q 6-12 h.

Prevention
Prevent harness wounds
Avoiding harness sharing
Euthanasia of severe cases with Thiopental Sodium 10g dissolved in 60 ML of water administered by 14 G cathether followed by 100mg Succinylcholine Or Pentobarbital Sodium 200mg/ml 200ml followed by 100 mg Succinylcholine IV Or Secobarbital and Cinchocaine hydrochloride (Somulose) at 5ml/50Kg IV.
No completely satisfactory treatment is known. Surgical excision of nodules and removal of pus.

RESPIRATORY DISEASES OF EQUIDAE

Acute Bronchointerstitial Pneumonia in Foals

Acute bronchointerstitial pneumonia is a sporadic, rapidly progressive, disease of foals aged between 1 week and 8 months characterized by acute respiratory distress and high mortality. The etiology is unknown, but autodestructive inflammation, endotoxins from gram negative bacteria or infection with **Rhodococcus equi** have been suspected.

Clinical Symptoms

Acute or peracute onset accompanied by a marked fever. Foals are unable or reluctant to eat or move and are usually cyanotic. Severe respiratory distress is the most marked clinical sign.

Diagnosis

Clinical examination

Treatment and Prevention

Dexamethasone 0.1-0.2 mg/kg, q 12 h for 1-2 days initially (maximum for the two days ~80 mg), then once daily for another 3-5 days followed by a gradual tapering of the dose over the next 5 days) as early as possible.

Plus

Erythromycine, 20-25mg/kg PO q 8h

Penicillin and streptomycin sulphate 20,000IU/250mg, 1 ml/20-25kg, q 24h for 3-7days. **Or**

Oxytetracycline 5%, 3-5 mg/kg q 12h, IV or IM. **Or**

Sulfadiazine-Trimethoprim, 1 ml/30kg, q 12h IV or IM.

Caution: Prolonged steroid use may predispose foals to the development of gastric ulcers. The treatment should be immediate and aggressive.

Aspiration Pneumonia

Aspiration pneumonia is characterized by pulmonary necrosis due to inhalation of foreign materials. Faulty administration of medicines is the most common cause. The epidemiology is similar as that of the condition in cattle.

Clinical Symptoms

Fever (40-40.5°C), accelerated pulse, rapid and labored respiration, sweetish, fetid breath characteristic of gangrene may be observed. A purulent nasal discharge which sometimes is tinged reddish brown or green. Occasionally, evidence of aspirated material, e.g. oil droplets, green herbs can be seen in the nasal discharge or expectorated material. On auscultation, fluid sounds over one or both sides of the chest are heard early in the condition followed by wheezing sounds, pleuritic friction rubs and sometimes crackling sounds of subcutaneous emphysema.

Diagnosis

Clinical signs and history suggesting recent foreign-body aspiration is of great diagnostic value. Auscultation is also of great value.

Differentiatial diagnosis: Acute bacterial bronchopneumonia and Septicemia

Treatment and Prevention

Non Drug Treatment:

The animal should be rested and kept quiet.

Drug Treatment

Penicillin G Procaine, 20,000 IU/kg, q 12 hr. IM for 5-7 days. **Or**

Penicillin and streptomycin sulphate, 1ml/kg, IM for 5-7 days. **Or**

Trimethoprim-Sulphamethoxazole 30mg/Kg q 12hr. for 5-7 days PO. **Or**

Ampicillin 7.5-10 mg/Kg q 12h for 5-7 days IM. **Or**

Oxytetracycline hydrochloride 5%, 11 mg/kg, q 12 hr. for 5-7 days. **Or**
Gentamicin 5%, mg/Kg, q 8h IV or IM for 3-5days.
Plus
Flunixin Meglumine 1.1mg/Kg q 8-12 hr. IV or IM. **Or**
Phenylbuthazone 2.2-4.4 mg / kg q 8-12 hr. PO or IV.
Carprofen 0.7-1.3 mg / Kg q 24 hr. IV or PO.

Prevention
As prevention and control measure inject atropine sulfate to control salivation stimulated by the anesthetics (e.g. thiobarbiturates) or use of an endotracheal tube with an inflatable cuff prevents fluid aspiration during surgery and care must be taken when drenching fluid.

Pleuropneumonia

Pleuropneumonia is an acute or chronic inflammation of the pleural membranes. Typically isolated organisms include **Streptococcus equi subspecies zooepidemicus**, **Escherichia coli, Pasteurella spp, Klebsiella spp,** anaerobes such as Bacteroides and **Clostridium spp** and **Mycoplasma felis** and other **Mycoplasma spp.**

Clinical Symptoms
Fever, inappetence, depression, dyspnea, standing with abducted elbows and reluctance to move, subcutaneous edema of the ventral thorax and limbs and colic or myositis are typical. In chronic cases, there is often anorexia, weight loss, intermittent fever and cough, abnormal respiratory effort and in horses with sterile or neoplastic effusion, reduced exercise tolerance.

Diagnosis
Clinical examination, septic fluid from pleural space, Gram's stain and culture.

Treatment and Prevention
Penicillin G procaine, 22,000IU/kg, q 12h, IM or 40,000units/Kg q 24 h, IM for 3-5 days. **Or**
Gentamicin Sulphate 5%, 6.6 mg/Kg, IM, q 12 h for 3-5 days. **Or**
Oxytetracycline hydrochloride 5%, 10 mg/Kg, q 12 h, IM is indicated for Mycoplasma spp infection for 5 days. **Or**
Ceftiofur sodium inj., 2.0 mg/ Kg, q 24h, IM for 4-5 days and re-evaluate. Safety in breeding animals and suckling foals (under 6 months of age) has not been established.
Plus
Flunixin Meglumine 1.1mg/Kg q 8-12 h IV or IM. **Or**
Phenylbuthazone 2.2-4.4 mg / kg q 8-12h PO or IV. **Or**
Carprofen 0.7-1.3 mg / Kg q 24h IV or PO

REPRODUCTIVE DISEASES OF EQUIDAE

ABORTION

Brucellosis

Brucellosis is a disease caused by several species of Brucella bacterium. It is chronic and contagious. It can affect multiple species of mammals, but is seen particularly in cattle, sheep, goats, bison, pigs, and the horse.

Clinical Symptoms

The clinical symptoms due to brucellosis are mostly noticed in the musculoskeletal system mainly as the organism localizes in the bursae (causing septic bursitis), joints (causing septic arthritis) and tendon sheaths (causing septic tenosynovitis). Few reports regarding abortion, vertebral osteomyelitis and infertility in male horses have also been documented.

The gold standard test for diagnosis of Brucellosis is isolation and identification of the organism which needs 5-10% carbon dioxide for its growth.

Diagnosis

Serological tests like Rose Bengal Plate Test (RBPT), Standard Tube Agglutination Test (STAT) and ELISA are commonly employed.

Treatment and Prevention

Non drug treatment:

Periodical drainage, cleaning the region with antiseptics will aid to control further complications of poll evil and fistulous withers.

Drug treatment

Oxytetracycline 5%, 3-5 mg/kg q 12h, for 5-7 days for secondary infections.

Prevention and Control

Test and quarantine or euthanize positives since brucellosis is zoonotic (Test and cull)

House or graze equines away from cattle suspected for brucellosis

Use proper fitting saddle to prevent trauma, parasite control (Onchocerca spp) and vaccination.

Other Bacterial Causes of Abortion

Potomac horse fever caused by **Ehrlichia risticii** causing abortion in mid to late gestation. The organism has been isolated from fetal lymphoid tissues after abortion. Abortion due to **Streptococcus zooepidemicus**, other **Streptococcus** spp, **Escherichia coli**, **Pseudomonas**, **Klebsiella** or other bacteria is normally caused by an ascending infection through the cervix that results in placentitis. **Salmonella** and **Leptospira spp** have been implicated in equine abortions probably involving stress and systemic endotoxemia on the fetus.

Clinical Symptoms

The placenta is edematous with brown fibrinonecrotic exudate near the cervical star. Chronic placentitis results in retardation of fetal growth. The fetus may be severely autolyzed when expelled. Organisms can be recovered from aseptically obtained stomach contents.

Treatment and Preventions

Dihydrostreptomycine sulphate, 12-25 mg/kg, q 12 h, IM, for 3 days. **Or**

Gentamicin Sulphate, 2 mg/kg, IM, q 12 h for 3 days as first line drugs. **Or**

Oxytetracycline hydrochloride 5%, 10 mg/kg, q 12h, IM is indicated for **Mycoplasma spp** infection for a week as an alternative.

Note: The objective of treatment is to control systemic infection of the dam.

Acute Puerperal Metritis

Acute puerperal metritis occurs within the first few postpartum weeks (10–14 days postpartum). It results from contamination of the reproductive tract at parturition and often, but not invariably, follows complicated parturition. Important causative organisms include **Escherichia coli** and **Trueperella (Arcanobacterium) pyogenes**. The condition is usually acute in onset.

Clinical Symptoms
Affected mares are depressed, febrile and lose appetite. Milk production is diminished and nursing young may show signs of food deprivation.

Diagnosis
It is based on its clinical symptoms and history of recent parturition.

Treatment and Prevention

Non-drug treatment

Drainage of the uterine content may be advantageous.
Uterine lavage with Lugol's solution or saline to which 1gm/L streptomycin is added and treatment is repeated 2-3 times

Drug treatment

Procaine penicillin G at 20,000-25,000 IU/kg, IM, q 24 h. or Sodium Penicillin q 12 h for 3-5days for secondary infection. **Or**
Oxytetracycline 50 mg/ml a dose of 11 mg/kg, q 12 h for 3-5days as a first line option. **Or**
Gentamicin 50 mg/ml at a dose of 5 mg/kg q 8h IV or IM for 3-5days as a second line option.
Note: Drainage of uterine content should be done very carefully because the inflamed uterus may be friable and manipulation of the uterus may result in bacteremia.

Equine Herpes Virus Abortion

EHV-1 is recognized as the most common cause of virus-induced abortion in pregnant mares.

Clinical Symptoms
Pregnant mares usually abort without prior clinical symptoms, most infections occurs late in gestation (8-11 months) and appear refractory to abortion if the virus is encountered earlier (less than 120 days) in gestation.

Diagnosis
Virus isolation, immunofluorescence, Polymerase Chain Reaction (PCR)-based assays and serologic analyses. Virus isolation remains the "gold standard" for laboratory diagnosis of EHV infections.

Treatment and Prevention
No specific treatment but it is possible to use antibiotics to prevent and treat secondary infections for 4-6 days.

Prevention
Is based on vaccinating at 5, 7, and 9 months of gestation as well as preventing exposure of pregnant mares to exposed horses or use of AI.

Dystocia

Dystocia in mare is very rare and occurs occasionally and may result from myometrial defects, metabolic abnormalities such as hypocalcaemia, inadequate pelvic diameter, insufficient dilation of the birth canal, fetal hormone (corticosteroid) deficiency, fetal oversize, fetal death or abnormal fetal presentation and posture.

Clinical Symptoms

Dystocia should be considered in any of the following situations: 1) an animal has a history of previous dystocia or reproductive tract obstruction; 2) parturition does not occur within 24 hours after the drop in rectal temperature (to <37.7°C); 3) the resting period during active labor exceeds 4-6 hr.; 4) there is a black, purulent or hemorrhagic vaginal discharge; 5) there are signs of systemic illness or 6) gestation is prolonged.

Treatment and Prevention

Non-drug treatment

Remove dead fetuses or carefully facilitate delivery of malpresented or partially delivered fetuses. Gentle manipulation and adequate lubrication must be used to prevent injury or death to living fetuses. Episiotomy may be helpful

Surgery is indicated for obstructive dystocia, if dystocia is accompanied by shock or systemic illness, for primary uterine inertia, when active labor is prolonged, and/or if medical management has failed

Assistance is necessary in dystocia where a delay in assisting may mean the loss of the fetus. Breeding management is important.

Drug treatment

Oxytocin, 40-120 IU, I/M once

Or 50 IU slow intravenously in 2 liters of Dextrose 5% solution over 20 minutes

Or I/V, I/M or S/C injection of 5 to 20 units at every 15 to 20 minutes intervals and I/V drip of 60 to 120 units in 1 L saline solution at 1 unit/min until the second stage of labor ensues to induce parturition as the uterus of the term mare is very sensitive to the effects of oxytocin. Response is dose dependent

Note: Medical management may be considered when the condition of the dam and fetuses are stable, when there is proper fetal position and presentation, and when there is no obstruction.

Precautions: It is imperative to determine the cause of dystocia (.i.e. obstructive vs non-obstructive.)

Endometritis

Although profound endometritis accompanies contagious equine metritis in mares, most breeding problems are related to endometritis caused by nonspecific infections. In mares, the most common etiologic agent is **Streptococcus zooepidemicus**, but several other organisms may be involved, including **Escherichia coli**, **Pseudomonas aeruginosa** and **Klebsiella pneumoniae**. Yeasts and fungi are incriminated in some cases, particularly in mares with reduced resistance or as a sequel of exuberant antimicrobial therapy.

Clinical Symptoms

Visible exudate is rarely a feature of endometritis in mares.

Diagnosis

Isolation of potentially pathogenic bacteria from appropriately guarded swabs of the endometrium. Ultrasonic demonstration of exudates may also help in diagnosis.

Treatment and Prevention

For bacterial infections

Doxcycline 10mg q 12 h PO **Or**

Amphotericine B 0.3-0.9mg/kg IV q 24-48 h plus 100 – 200mg Intrauterine q 24 h. **Or**

Oxytetracycline 1000mg, Intrauterine **Or**

Infusion with 250ml saline to which 3gm Crystalline Penicillin is added.

Infusion 250ml saline with 1gm streptomycin.

Alternative
Penicillin and streptomycin, 1ml/20kg, I/M for 3-5 days. **Or**
Oxytetracycline 50 mg/ml a dose of 11 mg/kg, I/M, q 12 h for 3-5days. **Or**
Gentamicin 50 mg/ml at a dose of 5 mg/kg q 8h IV or IM for 3-5days.
For fungal or yeast infections
Amphotericin B, 0.1 mg/ml at a dose of 0.1-0.5 mg/kg slow IV 3 times/week.
Lugol's Iodine solution 2.5%, 5-10ml of in each uterine horn.
Oxytocin 100 units within several days of parturition as a control and prophylaxis measure.
Note: Treatment should be continued for several consecutive days, preferably during estrus.

Equine Mycotic Placentitis

Mycotic placentitis in horses is also due to an ascending infection that causes a thickened chorioallantois with variable exudate. Causative agents include **Aspergillus**, **Mucor** and **Candida**. A pale, enlarged liver or dermatitis may be found. Hyphae are found in the placenta, liver, lung or stomach contents.

Clinical findings, diagnosis and treatment,
See Diseases of Equidae, Aspergillosis or Candidiasis.

Equine Viral Arteritis (EVA)

Abortion may follow clinical cases of EVA by 6 to 29 days. Arteritis may be found in the fetal myocardium or there may be no fetal lesions. EVA can spread venereally or by aerosol.

Treatment and Prevention
It has no specific treatment
Use antibiotics to prevent and treat secondary infections for 4-6 days
Prevention is similar to EVR.

Mastitis in Mares

Acute mastitis occurs occasionally in lactating mares, most commonly in the drying-off period, in one or both glands. **Streptococcus zooepidemicus** is the most frequent pathogen, but **S. equi**, **S**. **equisimilis**, **S. agalactiae**, and **S**. **viridans** are also found.

Clinical Symptoms
Marked painful swelling of the affected gland and adjacent tissues and the secretion is often seroflocculent. Fever and depression may be present. The mare may walk stiffly or stand with hind legs.

Diagnosis
Clinical symptoms and rapid tests similar to cattle.

Treatment and Prevention
Non-drug treatment: constitute massaging with warm water
Drug treatment, control and prophylaxis: **See** treatment for bovine mastitis.
Note: Little is known about subclinical infections.

Retained Fetal Membranes in Mares

The equine fetal membranes are normally expelled within 3 hours after parturition, but expulsion may be delayed for 8-12 hours or even longer without signs of illness. The cause of retention of fetal membranes often is not known but the condition is associated with infection, abortion, short or prolonged gestation and uterine atony. Systemic signs of dehydration, septicemia, toxemia, laminitis and colic may accompany retained fetal membranes for 24 to 36 hours. Intervention should be sought as early as possible.

Treatment and Prevention

Non-drug treatment

Manual removal of retained membranes carries the risk of uterine damage or prolapse therefore it should be gentle tugging to remove already loosen membranes.

Drug treatment

Oxytocin, 10 to 20 IU, IM or SC every 2-3 hr.
Flunixin meglumine 1.1 mg/kg, IM, or IV or PO stat
Trimethoprim-sulfadiazine 15 to 30 mg/kg, orally twice daily if infection occur **Or**
Penicillin G 22,000 mg/kg, IV 4 times daily plus Gentamycin 6.6 mg/kg, IV once daily.
Oxytetracyline, 1000 mg/kg, intrauterine for prolonged cases.
Uterine lavage with isotonic saline solution.

Prevention

Oxytetracyline 1000 mg/kg, intrauterine should be administered prophylactically along with 2.5-5mg/kg, IM post-partum.
Use of Tetanus Antitoxin is advisable to prevent complication with tetanus.

WOUND MANAGEMENT IN EQUINES

The types of wounds in working equine are abrasions, bruising, hematoma, puncture wound, laceration, burns, proud flesh and contaminated wounds. Wound healing is commonly delayed if infection or infestation occurs, there is restriction of blood supply, there is foreign material in the wound, excessive movement or self-trauma, poor nutrition and health status, inappropriate treatment and exuberant granulation tissue.

Treatment and Prevention

Non drug Treatment

Intervene on time to avoid bacterial contamination [less than 4 hours]
Use a clean, manageable work site with minimal disruption
Cover the wound with gel or Vaseline to prevent entry of hair and debris
Wet and clip hair starting at the edges of the wound and move outward
Irrigate the wound with physiologically sound lavage solution at an oblique angle and pressure that removes adherence of bacteria while avoiding tissue damage
Debride the wound and use appropriate scrubbing techniques
Unlike acute wounds, chronic or heavily contaminated wounds may benefit from diluted antiseptic added to physiological saline
Serial wound cleansing may be needed in heavily contaminated wounds and those healing by second intention
Honey at 5-10 % v/v: can be applied to infected wounds for 7-10 days. Honey provides protective care, anti-inflammatory effect, antibacterial activity, moist healing environment, removes bad odor and promotes healing.

Plus

Education of owners on principles and practice of good harness
Advise on sound loading practices including balancing and flour cooling
Advise owner to rest the animal for the wound to heal

Drug treatment

Lavage solutions

Physiological Saline: teaspoonful of salt to 0.6 liters of warm (previously boiled) water

Povidone-iodine 0.1 to 0.5% solution for wound lavage, highly diluted as serum binds free iodine only. Repeat every 4-6 hours. For SE, CI, D/I, See Annex 5

Chlorhexidine 0.05% solution. Significant residual activity than iodine, binds to cells.

Pain Relief

Phenylbuthazone 2.2-4.4 mg / kg q 8-12h PO or IV.

Flunixin Meglumine 1.1mg/Kg q 8-12 h IV or IM

Topicals

Zinc oxide ointment 15%, apply every other day after cleaning the wound

Systemic Antibiotics:

Procaine Penicillin 20,000 units/kg, IM, q 24 hr. 3-7days depending on severity as a first line therapy

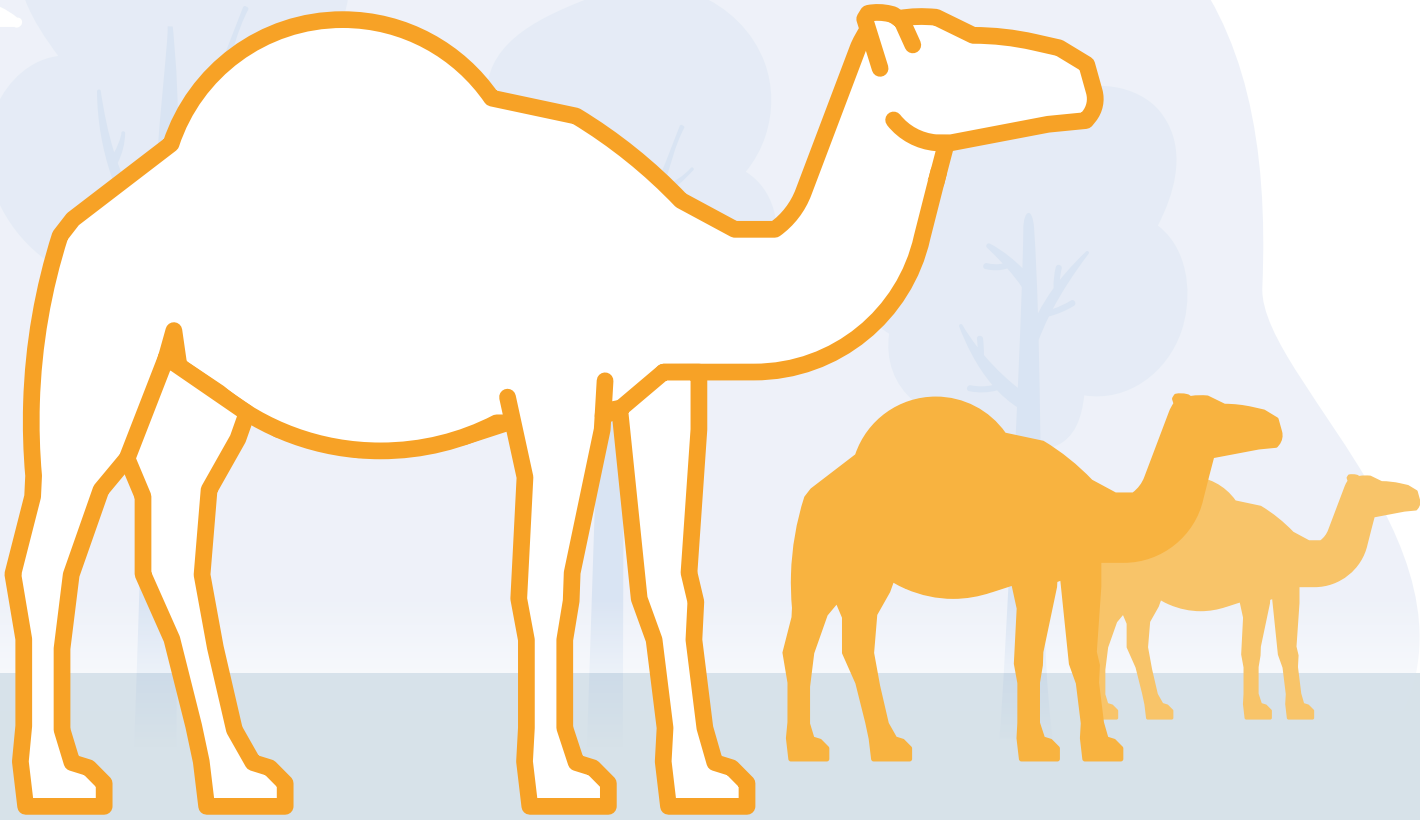
Streptomycin sulphate 10 mg/kg, IM, q 24 hr. 3-7days depending on severity as a second line therapy

Gentamicin, 1-2 mg/kg IM, s.i.d for 3-7days depending on severity.

Note: Make sure bleeding is controlled, no vital organs damaged beyond the wound. Use of antibiotics as additives to lavage fluid should be discouraged or only approached with due caution because of resistance.

Hydrogen peroxide at 3% is tissue toxic. Its use should be limited to circumstances when anaerobic conditions are likely (e.g. irrigation of a deep wound which might be infected with Clostridium tetani). If not vaccinated apply tetanus anti-toxin.

SECTION V



DISEASES OF CAMELS[5]

5 *Guide*

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.

VIRAL DISEASES OF CAMEL

Bluetongue

Bluetongue is a disease of ruminants caused by an **Orbivirus** belonging to the family **Reoviridae**. Camelids are less susceptible but are considered to be reservoirs of the infection to sheep and cattle. There are serological indications in Amhara and Oromia regional states in sheep; however, the causal virus is not yet isolated.

Diagnosis
Serology and virus isolation.

Treatment and Prevention
No specific treatment for bluetongue
Control of Culicoides, which are vectors of the disease and vaccination with the appropriate serotype; however, it is difficult to implement

Camel Pox

Camel pox is a highly contagious viral disease caused by **Orthopox cameli**. It occurs at the beginning and early rainy season and is characterized clinically by pox lesions.

Clinical Symptoms
Initially moderate depression, mild fever and anorexia and enlargement of mandibular lymph nodes followed by vesicles that rupture and become covered by thick brown scabs and scar.

Diagnosis
Clinical symptoms are presumptive and confirmed by serological tests.

Treatment and Prevention
There is no specific treatment
Antibiotic treatment is useful to control secondary bacterial infection, **see** Anthrax treatment above.

Prevention
Vaccination and improved management strategies to reduce prevalence.

Contagious Ecthyma

Contagious ecthyma is a highly contagious viral disease characterized clinically by pruritis and itching against objects followed by hemorrhages and extensive skin excoriations, which leads to impaired grazing ability. The lesions could be localized or generalized.

Treatment and Preventions
There is no specific treatment; however, secondary infection could be prevented by high doses of Procaine Penicillin G, 30,000IU/kg, IM, q 24 hr. for 3 days. **Or**
Amoxicillin, 10-20 mg/kg, IM, q 24 hr. for 3 days.

Prevention
The effect of vaccination is controversial
Isolation of infected animals from the herd is important
Public health significance: Contagious ecthyma is zoonotic thus care should be taken to avoid human infection.

Rabies

Rabies is a fatal disease for humans and other warm blood animals causing encephalitis. It is transmitted by the bite of infected animals. Camelids are susceptible to rabies.
Clinical symptoms, Treatment and prevention are similar to dogs and cats (**see** Diseases of Dogs and Cats: Rabies).

BACTERIAL DISEASES OF CAMEL

Anthrax

Anthrax in camel is caused by **Bacillus anthracis**. It is a peracute disease characterized by septicemia and sudden death. The spore can survive in contaminated soil for many years and continue to infect animals by inhalation or ingestion.

Clinical Symptoms
Dromedaries affected show difficult breathing, trembling and pronounced swelling of the throat and base of neck and groin region.

Diagnosis
It is based on blood smear and confirmed by fluorescent antibody test.

Treatment and Prevention
Procaine Penicillin G, 22,000 IU/kg, IM, q 24 hr. for 5 days; the dose should be divided into two parts for the first two days. **Or**
Oxytetracycline, 4.4 mg/kg, q 24 hr. for 5 days; the dose should be divided into two parts for the first two days.

Prevention
To prevent sporulation, carcass should not be opened; the cadaver should be burnt and equipment disinfected by either of the following solutions: 10% hot caustic soda solution, **Or** 4% formaldehyde solution, **Or** 7% hydrogen peroxide, **Or** 2% glutaraldehyde, **Or** calcium hypochloride with 5% active chlorine
Vaccination with Sterne vaccine annually; the dose should be similar to cattle.
Public health significance: Anthrax is zoonotic disease transmitted through consumption of contaminated meat or contact with wound.

Botulism

This is toxemia caused by ingestion of preformed toxin of **Clostridium botulinum** and characterized by progressive paralysis. The epidemiology of botulism in camels is similar to that in cattle (**see** Diseases of Cattle: Botulism). It is sporadically reported by pastoralists and nomads in Somali and Oromia regional states.

Diagnosis
Commonly, the diagnosis is made by eliminating other causes of motor paralysis and search for potential sources of toxin.

Treatment
It is the same as in cattle (**see** Diseases of Cattle: Botulism).

Brucellosis

Brucellosis is the most common disease of camelids caused by **Brucella melitensis**, **B. abortus** and **B. ovis** and characterized by abortion. Retained placenta has not been reported in camels. Serological tests conducted in Borena zone have shown relatively high prevalence. The prevalence of brucellosis is high in areas when camels are in contact with infected ruminants.

Clinical Symptoms
Abortion is the most common sign.

Diagnosis
Tube agglutination test using 5% NaCl phenolized solution, CFT, ELISA and culture of blood, milk or tissue such as the placenta and stomach and lung of aborted fetuses are recommended in camel.

Treatment and Prevention
Oxytetracycline, 25 mg/kg, q 48 h for 30 days I/M. **Or**
Streptomycin, 25 mg/kg, q 48h for 16 days, I/M for seropositive animals.

Prevention
Test and slaughter and vaccination of camels.

Dermatophilosis

Dermatophilosis is skin disease of cattle, sheep, goat, camels and other animals caused by the actinomycete bacteria, **Dermatophilus congolensis.** The hair underneath infected skin appears as paintbrush. When the hair is pulled off, the skin is wet, reddish brown.

Diagnosis
The bacterium is easily observed by direct smear stained with Giemsa or Gram's stains. Culture from the skin scrapping is confirmatory.

Treatment and Prevention
Oxytetracyline 20%, 20 mg/kg, IV, q 12 hr. **Or**
Disinfecting locally with 5% Povidone-iodine solution for 7 days.
Plus
Shear of long hairs along the lesions to reduce further development of the lesions and remove the scab.
Public Health significance: Dermatophilosis is zoonotic disease.

Hemorrhagic Septicemia

Hemorrhagic septicemia is caused by particular serotypes of **Pasteurella multocida.** It is characterized by high fever, increased pulse and respiratory rate and general depression. The disease is common in adult animals and precipitated by environmental stress factors such as high humidity and heavy **Hyalomma dromedarii** tick infestation. Infection is usually endogenous but also by ingestion of contaminated foodstuffs or by arthropods.

Clinical Symptoms
Acute onset, high fever (>40°C), increased respiratory and pulse rate and general depression. Swelling of the subcutaneous tissue results in hot, painful swellings around the neck and lymph nodes become enlarged. Hemorrhagic enteritis characterized by acute abdominal pain and excretion of tarry feces and discolored urine are observed.

Diagnosis
It can be diagnosed by its clinical symptoms and confirmed by microscopic examination of blood smears.

Treatment and Prevention
Sulphadiazine-trimethoprim, 15 mg/kg in drinking water, PO, for 5 days; remove all other sources of water before administration. **Or**
Sulphamethoxine as suspension, injection or bolus; Initial 55 mg/kg, maintenance: 27.5 mg/kg, q 24 h, PO or IV or SC, for 5 days. **Or**
Other antimicrobial used in bovine (**see** Diseases of Bovine, Hemorrhagic septicemia)
Caution: Store sulpha drugs at room temperature and protect from light. IM injection cause local pain and inflammation. Frequent watering, supplemental feeding and vaccination (in endemic areas) with specific bacterin are important.

Tetanus

Tetanus is a toxemia caused by infection with **Clostridium tetani** that affects all mammals. The camelids are rarely affected. Infection occurs via contaminated wound and/or frequent puncture wounds due to the long hard thorns of the acacia bush.

Clinical Symptoms
The clinical symptoms are similar to the classical tetanus with muscle spasms, neck stiffness and the characteristic disturbances of mastication, increased reflex activity and tetanic spasms.

Treatment and Prevention
Tetanus antitoxin (TAT) 100 ml, 1500 IU, IV q 12 h or SC, q 24 h for 3-5 days.
Plus
Debridement of the wound and feeding via gastric tube
Plus
Antibiotic treatment (similar to any systemic infection in camels like pneumonia)

Prevention
Wound should be treated with antiseptic.

Tuberculosis

Tuberculosis is caused primarily by **M. tuberculosis** and **M. bovis** and atypical mycobacteria. Transmission is mainly through aerogenic by inhalation and alimentary by ingestion of food contaminated with infected feces, urine or milk.

Diagnosis
The intradermal tuberculin test often gives non-specific reaction and the sensitivity has also been low.

Treatment and Prevention
Isoniazid 5-10 mg/kg, PO in feed SID. Treatment is sometimes recommended for zoo Camelidae.

Prevention
Disinfect contaminated utensils and surfaces with 3% formalin, 2% Lysol and 2.5% phenol.

OTHER BACTERIAL DISEASES OF CAMEL

Mastitis

Mastitis refers to inflammation of the udder. It is less common in camelids compared to bovine, ovine or caprine species. The etiologies are similar in other animals. In camels, it may be peracute, subacute and gangrenous. The lymph nodes are enlarged and the milk becomes watery, yellowish or blood-tinged.

Diagnosis

The California Mastitis Test or Somatic Cell Count could be used as a screening test. Culture is important to determine the causal agent.

Treatment and Prevention

Drug Treatment

Benzathine-Cloxacillin, 500mg, Intramammary infusion, single dose and may be repeated after 2-3 days; **Or**

Streptomycin plus penicillin G (1 gm. + 100,000 IU) I/M q 24 h for 3 days

Non Drug Treatment

The teat canal should be penetrated with catheter and left open. .

Note: Each teat in the camel has two streak canals, which are narrow; thus care should be taken not to damage it during intramammary infusion.

Pneumonia

Pneumonia is the most common syndrome in camels. It is caused by viruses, bacteria, fungus or aspiration, as well as by toxins arriving hematogenously or by inhalation. Handling, transport, mixing and overcrowding are often considered predisposing factors.

Clinical Symptoms

Acute onset of lower respiratory disease characterized by a change in respiratory rate and depth, wheezing, coughing, unilateral or bilateral nasal discharge, hyperthermia, anorexia and usually general depression with reluctance to move or work. Hyperlacrimation, abduction of the elbows, extended neck and deviated carriage of the head with apparent swelling of the temporal region or above the sinus frontalis.

Treatment and Prevention

Drug Treatment

Amoxicillin trihydrate 6.6-11 mg/kg I/M or S/C once a day for 5 days, Or long acting (150 mg/ml or 200 mg/ml) at 15 mg/kg or 1 ml/20 kg. **Or**

Procaine Penicillin G, 40,000IU/kg, IM, q 24 hr. for 3 days. **Or**

Oxytetracycline, 20 mg/kg, IV, q 24 hr. for 3-5 days. **Or**

Gentamycin sulphate, 5-8 mg/kg, IM, q 24 hr. for 3 days

Plus

Dexamethasone, 3 mg/kg, IV or IM, q 6 hr.

Prevention

Regular cleaning and flushing of the nasal cavity with saline plus improved management such as housing, hygiene and good nursing care.

Note: Treatment depends on the type of organism involved and the type of pneumonia.

Saddle Sore

Saddle sore is characterized by injuries of the skin and deeper soft and bony tissues of the back.

Clinical Symptoms
The clinical symptoms range from erythematous through papular, vesicular, pustular and finally necrotic lesions. Diagnosis is based on the site of the lesion location; specifically on the area where the saddle rests.

Treatment and Prevention
Lead acetate, 2% astringent packs for early stage
Povidone-iodine as 0.1 to 0.5% solutions for wound lavage in chronic lesions. **Or**
Systemic antibiotics (e.g. penicillin G + streptomycin) see Treatment of wound in cattle. **Plus**
Surgical removal of dead tissue and treating with astringent (500 ml of 0.1% Alcohol sublimate plus 30g Tannic acid, and 1g Gentian violet)
Give the animal complete rest.

FUNGAL DISEASES OF CAMELS

Dermatomycosis

Dermatomycosis is caused mainly by **Trychophyton spp**. of fungi. The predisposing factors are similar to dermatophytosis in cattle (**see** Diseases of Cattle: Dermatophytosis). The disease is mainly spread by direct contact with infected animals, but grooming tools, blankets, enclosures and man can also act as fomites. The importance of "carrier animals" without clinical symptoms in the spread of infection is not known.

Clinical Symptom
Non-pruriginous dry circumscribed discrete, scaly white encrustations or crusty hairless lesions distributed over the head, neck, shoulder, limbs and flanks. The lesions starts small (1-2 cm) and then enlarge or merge.

Diagnosis:
History and skin lesions, demonstration of the arthrospores in skin scraping.

Treatment and Prevention
Amphotericine B 0.2mg/kg/day IV infusion freshly prepared in 5% dextrose
Treatment and control options are similar to cattle (**see** Diseases of Cattle: Dermatomycosis). **Or**
Soap and water for washing to remove crusts, then apply 50:50 tincture of iodine: glycerin repeated every day until the patches disappear
Topical antimycotic agent such as Mycostatin.
Note: Spontaneous recovery is common.

PROTOZOAN DISEASES OF CAMEL

Trypanosomosis

Camel trypanosomiasis is a debilitating protozoal disease caused by **T. brucei evansi** infection. The parasite is transmitted mechanically by biting flies such as **Stomoxis spp**. and **Tabanus spp**. Disease outbreaks show seasonal pattern associated with increasing numbers of biting flies during the rainy season or shortly thereafter. All age groups are susceptible, but immature, stressed and lactating animals are extremely vulnerable.

Clinical Symptoms

Severe anemia, high fever, anorexia and marked generalized edema and rapid deterioration and death. In the chronic form of **T. evansi** progressive weight loss, intermittent high fever, marked generalized muscular atrophy especially the rear end, pale mucous membranes and occasionally abdominal edema are observed. They may also exhibit a characteristic sweet odor due to urinary ketones. Late term abortions and premature birth are common.

Diagnosis

Clinical symptoms are indicative. Decreased PCV between 18-20 %, a responsive anemia and demonstration of the parasite in stained blood smears.

Treatment and Prevention

Quinapyramine sulfate/chloride 4 - 5 mg/kg S/C as curative dose. **Or**

Suramin 5-10 mg/kg I/V. Max. 10 g/camel as a curative & 5 g/head for preventive dose. **Or**

Isometamidium chloride 0.5-1.0 mg/kg I/V, as a curative and preventive dose. **Or**

Diminazene aceturate 3.5-5 mg/kg S/C as curative dose.

Note: Use 5% Dextrose or lactated Ringer's solution to dissolve the drugs. Drug resistance should be frequently monitored. Treatment of non-sedentary camel herds is only advisable if it is performed regularly.

INTERNAL AND EXTERNAL PARASITIC DISEASES OF CAMEL

Camel Myiasis

Camel myiasis is caused by invasion of wound in the nasal or aural cavities by larvae of dipterous flies. In Ethiopia and Sudan the larvae of **Wohlfahrtia Nuba** causes myiasis particularly in camels. The larvae from **Lucilia cuprina** and **Cephalopina titillator** are also reported as pathogenic myiasis.

Clinical Symptoms
Larvae is found in wounds, nostrils, nasopharynx and nasal sinuses, folds of the skin e.g. perineal area, continues rubbing with objects and biting the body. Clinical Symptoms are observed only when the larvae mature. Apart from the local inflammation of the pharynx and congestion of the nasal cavity, the camel may show respiratory and nervous signs.

Treatment and Prevention
For Wohlfahrtia nuba
Ivermectin 1%, 1 ml/50 kg. For S/C stat. **Or**
Rafoxanide, 7.5 – 15 mg/kg, P/O as a drench or bolus; or 1 ml / 25 kg B.wt. S/C at the neck as injection (7.5%). Do not inject more than 10 ml at one site.
For Lucilia cuprina and **Cephalopina titillator**
Insecticides and the wound should be cleaned and dressed, **Or**
Ivermectin 1%, 1 ml/50 kg, S/C (as above), **Or**
Hydrogen peroxide 2 %, ether or chloroform may cause hidden larvae to crawl.

Hydatidosis

Hydatids cysts of tapeworms belonging to the genus **Echinococcus** are common also in camelid particularly affecting the lungs and the liver.

Diagnosis
It is difficult in live animal. At postmortem the cysts are clearly observed.

Treatment and Prevention
Treatment in the camel is rarely employed. However, treatment in the dogs with anticestodal drugs is recommended (refer treatment in **dogs**)
Give the camel complete rest.

Note: Rigorous meat inspection should be implemented and proper disposal of abattoir waste and offal. Contamination of food/feed with infected dog's feces could cause human hydatidosis.

Lice Infestation

Lice infestation can be caused by **Anoplurida** or the sucking lice and **Mallophagida** or the biting lice. Transmission is either by direct close contact of the host or indirectly by grooming equipment, blanket and saddles, scratching posts or dust bath areas.

Clinical Symptoms
Sucking lice infestation is characterized by licking, scratching and rubbing. Anemia may follow heavy infestations, particularly in young animals. They are usually found around the head, neck and withers. Infestation with biting lice is characterized by heavy invasion in matted wool and alopecia. It also has pruritis resulting in self-trauma. The predilection sites are at the base of the tail, the back, along the ventral column and sides of the neck and the body.

Treatment and Prevention
Coumaphos 0.05% on skin and wet the coat thoroughly. **Or**
Ivermectin, 0.2 mg/kg, SC or 0.5 mg/kg pour on
Imidacloprid + Moxidectin Spot-on solution.

Mites Sarcoptes

It is caused by **Sarcoptes scabiei** var **cameli** transmitted by contact with animals or fomites. Young, immature, stressed adult and otherwise debilitated animals are generally affected and usually develop chronic generalized form of mange. Healthy animals may be affected as well, but usually lesions remain localized.

Clinical Finding and Lesions
Intense pruritus, evolving small vesicles and inflammatory reaction of the skin followed by loss of hair and later becoming moist and exudative. Animals rub against any object. The head, axillary, inguinal and perineal areas are commonly affected. Affected animals show a general loss in condition, decrease in milk production and poor reproductive performance.

Diagnosis
Signs are indicative. Demonstration of mite from skin scrapings from affected area is confirmatory.

Treatment and Prevention
Ivermectin, 0.2mg/kg, SC and can be repeated after 15 days. For severe cases use topical application.
Or
Diazinon 60% EC, spraying or sponging, similar to application in cattle and repeat every 10 days, **see** Cattle Treatment ectoparasite
Amitraz pour-on, repeat after 15 days.

Prevention
Improve management practices and sanitation.

Tick Infestation

Ticks suck considerable blood and cause debility and anemia. Their role as vectors is much less important than in other animals, but could expose to bacterial infections by mechanical damage of the skin. The most important tick genus affecting camels is **Hyalomma** with the species **H. asiaticum, H. dromedarii, H. franchini** and **H. scupense.** Other genera are **Amblyomma, Rhipicephalus** and **Dermatcentur. Ornithodorus** and **Boophilus**. **Dermacentor** species can inject neurotoxins while ingesting a blood meal, which may cause paralysis and sudden death.

Clinical Symptoms

Listlessness, sudden death in some, paralysis and high infestation of ticks on the body are common signs.

Treatment and Prevention

Deltamethrin or Flumethrin 1% pour-on, 1-2ml/10kg applied along the back or

Organophophates, carbamates and synthetic pyrithroids could be applied (**see** External Parasites of cattle), Or

Ivermectin 1%, 10 mg/50kg

Routine application of acaricides is not recommended in camels, but can applied in highly infested animals

Gastrointestinal Disorders

The most common gastrointestinal tract disorders in adult camels are caused by endoparasites, sudden diet changes and some specific diseases such as chronic trypanosomiasis, hemorrhagic septicemia and plant poisoning. Neonatal calf diarrhea is mainly caused by bacterial infection including **Eschrichia coli,** enterotoxaemia and salmonellosis. Dietary diarrhea caused by ingestion of excessive quantities of milk is rarely seen in immature and young calves.

Clinical Symptoms

The disorder is mainly manifested by diarrhea and usually occurs at the beginning of the rainy season. Diarrhea may show alterations of odor, color and presence of parasites, blood, mucus and undigested feed. Persistent diarrhea may also result in continuous soiling of rear quarter and a progressive loss in condition. Signs of septicemia may also be observed in bacterial infections.

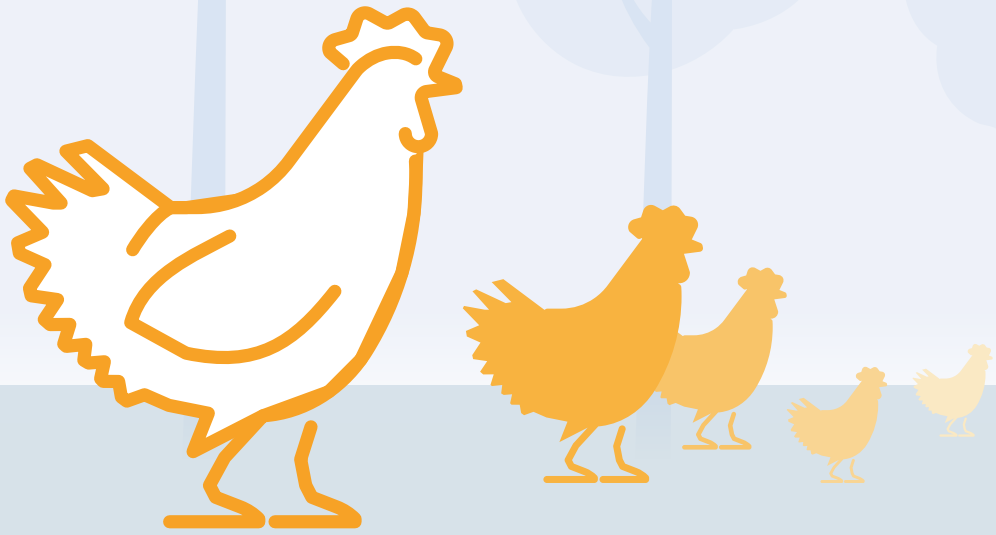
Treatment and Preventions

Endoparasitic diarrhea are treated with broad-spectrum anthelminthic (**see** Gastrointestinal disorder treatment for cattle)

Bacterial or viral infections are treated with long acting antibiotics (**see** Gastrointestinal disorder treatment for cattle)

Dietary diarrhea usually resolves by itself if the diet change is corrected.

SECTION VI



DISEASES OF POULTRY[6]

6 Guide

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.

NON-INFECTIOUS DISEASES OF POULTRY

Ammonia Toxicity

Ammonia is an invisible and irritant gas with a characteristic smell. The level of it in poultry house are dependent on the frequency of feces removal, ventilation, number and activity of chickens, type of bedding, temperature and pH of litter. Toxic level (>25ppm) will dissolve in liquid on mucous membranes and eyes to form ammonium hydroxide which cause irritation and damage to the eyes and mucociliary apparatus of the respiratory tract. The damage of mucosa predisposes to secondary infections like colibacillosis.

Clinical Symptoms and Lesions

50-75 ppm, decreased feed consumption, reduced body weight and egg production

75-100 ppm respiratory signs

>100 ppm ulceration of cornea, blindness, photophobia, swollen eyelids and stunting

Lesions include damage to the cornea or keratoconjunctivitis, excess mucus production and hemorrhages in respiratory tract.

Diagnosis

Based on history, clinical symptoms and examination of the eyes.

Treatment and Prevention

Non Drug Treatment

Ammonia levels should be reduced by adequate ventilation

Maintenance of adequate ventilation, provision of dry and clean bedding, avoidance of overcrowding and addition of natural zeolites to litter.

Ascites Syndrome

Ascites or pulmonary hypertension syndrome is metabolic disease in broiler chickens. It is an accumulation of non-inflammatory transudate in one or more of the peritoneal cavities or potential spaces. It develops due to hypoxemia. Hypoxemia leads to an increased cardiac activity and may subsequently result in pulmonary hypertension. Hypoxemia also leads to increase in red blood cells resulting in an increased blood viscosity which contributes to a further increase of pulmonary pressure.

Clinical Symptoms and Lesions

Cyanosis, redness of abdominal skin and congestion of peripheral vessels. Affected chicks are smaller than their penmates. Ruffled feathers, reduced weight, pale and shrunken comb, reluctance to move, distended abdomen, difficult breathing, sudden death, affected broilers frequently die lying on their back.

Postmortem lesions include yellowish clear fluid with or without fibrin clots, sometimes hydropericardium, right sided cardiac enlargement, left-side ventricular dilatation, mottled or shrunken liver, edematous and congested lungs and often no specific pathologic lesions.

Diagnosis

Based on lesions found at post-mortem examination.

Treatment and Prevention

No specific treatment.

Prevention

Control of early growth to reduce oxygen demand, feed restriction, feeding mesh feed, use of a diet containing less energy, light restriction or intermittent light programs, adequate ventilation, movement to lower areas, monitoring of sodium levels in feed and water to prevent salt intoxication and genetic selection.

Cannibalism

Cannibalism is a vice of chickens and turkeys most often manifested as vent-picking or picking at unfeathered skin on the head, comb, wattles or toes. No single cause has been identified, but overcrowding, excessive light and nutritional imbalances are directly correlated with its occurrence. Other factors that predispose to cannibalism are overly fat pullets where mucosa protrudes from the vent during and after egg laying, insufficient feeder space, mineral and vitamin deficiencies, skin injuries and failure to remove dead birds daily.

Treatment and Prevention
There is no specific treatment

Prevention
For control, trim the tip of the beak at one-day old and repeat between 6 and 12 weeks age. Cautery often is required to provide hemostasis.

Carbon Monoxide Toxicity

Carbon monoxidide (CO) is a colorless and odorless gas produced by not properly working brooders or furnaces. CO disrupts oxygen transport from lungs to tissues due to the greater binding affinity for blood hemoglobin than oxygen. CO is very dangerous both for chickens and humans and cause illness and mortality.

Clinical Symptoms
Lethargy, difficult breathing, incoordination, stunting, convulsions, death, sublethal levels are associated with an increased prevalence of ascites.
Post-mortem lesions include bright red-colored blood, bright pink-colored lungs, cyanotic beak and face.

Diagnosis
Based on history, clinical symptoms, post-mortem findings, measurement of air CO and carboxyhaemoglobin analysis in blood.

Treatment and Prevention
Removal of the source of Carbon monoxide production and adequate ventilation
Use of properly working brooders and furnaces
Adequate ventilation.

Calcium and Phosphorus Deficiency

Adequate phosphorus and calcium nutrition depends not only on sufficient total dietary supplies, but also on the chemical forms in which they occur in the diet and on the vitamin D status of the diet or the animal. The optimum Ca: P ratio for growing chicks and pigs lies between 1:1 and 2:1. For laying hens greater Ca is required.

Clinical Symptoms
Abnormalities in the bones, subnormal growth and reduced egg production, depressed appetite and efficiency of feed use and the development of pica or depraved appetite.

Treatment and Prevention
Direct supplement with 0.9% Ca and 0.7% P for starting chicks (0-8weeks) and for growing chicks (up to 18 weeks) with 0.6% Ca and 0.4% P.

Manganese Deficiency

Manganese functions as an important co-activator of enzyme involved in lipid and carbohydrate metabolism.
Manganese deficiency is relatively common condition in poultry because of poor absorption from feed.
A deficiency of manganese in the diet of young growing chicks is one of the causes of perosis, thin-shelled eggs and poor hatchability (see also Calcium and phosphorus imbalances, Vitamin D deficiency). It may also cause chondrodystrophy.

Clinical Symptoms
Perosis is a malformation of the hock joint in young chicks. The tibia and the tarsometatarsus of one or both legs may bend near the joint and rotate laterally. A shortening and thickening of the long bones of the legs and wings may be apparent. In adult chicken the shells of their eggs tend to become thinner and less resistant to breakage. There is a reduced egg production, smaller egg weight and lower hatchability.

Diagnosis
Based on clinical symptoms, physical examination and nutritional analysis of feed.

Treatment and Prevention
Diet that contains 20 and 60 mg/kg manganese in feed for laying hens and broilers, respectively
Requires a diet adequate in all necessary nutrients, especially manganese, choline, niacin, biotin and folic acid.
Note: Prevent excessive levels of calcium and phosphorous, as they decrease the absorption of manganese from feed.

Riboflavin Deficiency

If the ingredients of a poultry feed are not carefully selected, or if a special supplement is not included, a deficiency may result.

Clinical Symptoms
Young chicks, as early as 1-week-old, exhibit curling of the toes, inability to walk and sometimes diarrhea. Decreased egg production, increased embryonic mortality and an increase in size and fat content of the liver. Hatchability declines within 2 weeks.

Treatment and Prevention
Administering vitamin B preparations cures rapidly. A 100-mcg dose should be sufficient for treatment of riboflavin-deficient chicks, followed by incorporation of an adequate level in the diet
It is important to ensure adequate vitamin B levels not only in starter and grower diets, but also in the diet of parent breeders.

Vitamin D Deficiency

Vitamin D is required for the normal absorption and metabolism of calcium and phosphorus. Thus, a deficiency can result in rickets in young growing chickens and osteoporosis and poor eggshell quality in laying hens.
Rickets and osteoporosis can be seen in poultry due to a deficiency of vitamin D. Most poultry reared in strict confinement need a higher dietary level of vitamin D than those that have access to sunshine. Mycotoxins in the feed or litter may interfere with absorption of the vitamin (as well as of fat and the other fat-soluble vitamins).

Clinical Symptoms
Young chicken and turkeys: a tendency to rest frequently in a squatting position, disinclination to walk and a stiff gait. Other signs are retarded growth, enlarged hock joints, beading at the ends of the ribs, marked softening of the beak and the feathers become ruffled.

Laying chickens: thinning of their egg shells and if severe prompt reduction of egg production and hatchability, breast bones become noticeably less rigid and there may be beading at the rib ends. The lesions in young chickens and turkey are soft bones and enlarged parathyroid gland. In adult chickens, bones tend to become rarefied (osteoporotic) rather than soft.

Treatment and Prevention

Dry, stabilized forms of vitamin D3 is added to commercial diets to provide three times the normally recommended level for a period of ~3 wk.

Use of diet with adequate Ca, P and Vitamin D levels

Increase exposure to sunlight

Be aware large doses can be toxic.

In cases of severe mycotoxicosis, a water-miscible form of vitamin D is administered in the drinking water to provide about three times the amount normally supplied in the diet.

Vitamin E Deficiency

Vitamin E acts as an antioxidant by protecting cell membrane against oxidative damage. Oxidative damage due to vitamin E deficiency manifests itself as encephalomalacia, muscular dystrophy or exudative diathesis.

Clinical Symptoms

Growing chicks:

Encephalomalacia: Ataxia, paresis, imbalance, falling on the back, twisting of the head, repeated muscle contractions of the legs. Post-mortem lesions softened and swollen cerebellum, edema, hemorrhages and necrosis in the cerebellum.

Muscular dystrophy: Usually no signs but locomotors problems may appear. Postmortem lesions are light-colored linear streaks in the breast muscle.

Exudative diathesis: Subcutaneous edema in breast, abdomen and mandibular space and Cyanotic legs. Post-mortem lesions are subcutaneous edema in breast, abdomen and mandibular space.

Mature chickens: Reduced hatchability, increased embryonic mortality (cloudy spots in the eyes, blindness, hemorrhages, reduced growth, and abdominal vascular system).

Diagnosis

Involves recognition of major Clinical Symptoms particularly encephalomalacia and muscular dystrophy and examination and analysis of feedstuff to assess selenium and vitamin E levels.

Treatment and Control

Encephalomalacia can be prevented by adding synthetic antioxidants to the feed; adding selenium to the feed will prevent exudative diathesis and adding cysteine, a sulfur containing amino acid, to the feed will avert muscular dystrophy.

Recommended vitamin E levels are 30 to 150 mg/kg in the feed. Oral administration of a single dose of vitamin E (300 IU) per bird will often cure exudative diathesis or muscular dystrophy.

Birds with encephalomalacia do not usually respond well to treatment.

VIRAL DISEASES OF POULTRY

Avian Encephalomyelitis

Avian Encephalomyelitis (AE) (also known as "epidemic tremor" is an infectious viral disease caused by avian encephalomyelitis virus (AEV). After vertical infection incubation period ranges from 1-7 days. After horizontal infection, incubation period has minimum 11 days. Morbidity of young chickens ranges 40-60% and average mortality rate is 25% but may increase up to 50%. In laying hens a sudden drop in egg production and return to normal.

Clinical Symptoms
Young chicks: Nervous signs, dull expression of the eyes, ataxia, sitting on hocks, failing on its side, tremor of the head and neck and paralysis.
Laying hens: Sudden transient drop in egg production.

Diagnosis
Based on clinical symptoms and histopathologic lesions in brain, spleen and heart muscle. Confirmation is based on isolation and identification of virus from brain.
It must be differentiated from Newcastle disease, Nutritional disturbance (Vitamin E or A deficiency, Riboflavin, Rickets) and Marek's disease (although this affects usually older birds).

Treatment and Prevention
No treatment is available for AE

Prevention
Vaccination of breeder flock in the growing period prevent vertical transmission.
Vaccination must be done at least four weeks before the start of lay, because the vaccine virus can be transmitted vertically resulting in infected progeny.

Avian Influenza

Influenza in chicken is caused by an **orthomyxovirus type A** influenza viruses.

Clinical Symptoms and Lesions
Signs range from only a slight decrease in egg production or fertility to a fulminating infection with CNS involvement, but respiratory signs are most common. Other common signs in severely affected birds are greenish diarrhea, cyanosis and edema of the head, comb, and wattle, discoloration of the shanks and feet due to ecchymoses and blood-tinged oral and nasal discharges.

Diagnosis
Isolation of the virus in chicken embryo.

Treatment and Prevention
There is no specific treatment
Broad-spectrum antibiotics may be used to control secondary bacterial infections and increasing house temperatures may help reduce mortality.

Prevention
Vaccination with autogenous virus or a virus of the same hemagglutinin type
The virus could change its antigenic characteristic and infect humans.

Chronic Respiratory Disease

Chronic Respiratory Disease (CRD) also called airsacculitis is an infection of chicken and turkeys caused by **Mycoplasma gallisepticum.** It is responsible for mild to severe respiratory disease. Concurrent infections such as IB, NC, **E. coli**, **P. multocida** and **H. paragallinarum** or increased level of dust in the environment predispose to CRD.

Clinical Symptoms and Lesion

Signs of CRD in a flock develop slowly and are usually manifested by drop in egg of (10-20%) and meat production. They show varying degrees of respiratory distress, with slight to marked rales, difficulty in breathing, coughing, mild to severe catarrhal inflammation of conjunctivae and/or sneezing, airsacculitis, distended oviducts. The lesions include airsacculitis, fibrinous perihepatitis and adhesive pericarditis.

Diagnosis

Based on clinical expression of the disease and identification of the agent or specific antibodies. Positive plate or tube agglutination tests and ELISA.

Differential diagnosis: from **A. parragallinarum, P. multocida, M. synoviae,** Newcastle Disease, Low Pathogenic Avian Influenza, **Avian metapneumovirus,** Infectious laryngotracheitis, Infectious Bronchitis.

Treatment and Prevention

Tetracyclines 2.5 to 10 mg/kg, PO in drinking water q 24 hr. for 3-5days. **Or**

Tiamulin 10%, 100g per 200 litter of drinking water during 5 days or 1000g per 1000 kg of complete feed during 5 days. **Or**

Erythromycin 125 g per 100 liters of drinking water during 3-5 days (20mg per Kg body weight). **Or**

Tylosin, 0.5% in drinking water 3-5 days (50 mg/kg body weight). **Or**

Enrofloxacin 10 mg/kg in drinking water during 3-5 days or other fluoroquinolones can be given in the feed or water for 5-7 days; however, relapses are common. **Or**

Lincomycin + Spectinomycin, 16.65 mg lincomycin and 33.35 mg spectinomycin/kg bw Chickens /day, for 7 days.

Prevention and control

Obtaining chicks from **M. gallisepticum** free parent stock

Antimicrobial treatment is applied in diseased flocks in order to alleviate the severity of the course of the disease, this also contributes to reduction in shedding the agent and vertical transmission

Since treatment has short term effect, improve the management, husbandry or nutrition e.g. reduce dust in the house, remove accumulated litter and improve ventilation as a preventive measure.

Note: Treatment is expensive and thus marketing an infected flock with low incidence of disease may be more economical. Antibiotic treatment does not prevent occurrence of lifelong carrier status and the occurrence of residues.

In situation of high prevalence, antibiotic treatment and vaccination programs may contribute to the reduction of clinical and economic impact of the disease.

Egg Drop Syndrome

Egg Drop Syndrome (EDS) is a major cause of egg production losses mostly severe in broiler breeders and brown egg laying chickens caused by Egg Drop Syndrome Virus (EDSV) transmitted vertically through eggs or via feco-oral route. The virus remains silent until chickens approach peak egg production.

Clinical Symptoms

Loss of eggshell pigmentation, production of thin-shelled, soft-shelled and shell-less eggs, reduced egg production (up to 40%), rough or chalky eggshells and mild depression. Post-mortem lesion are inactive ovaries, atrophy of oviducts, edema and white exudates in the uterus and eggs in various stages in abdominal cavity.

Diagnosis

History and clinical symptoms. Definitive diagnosis needs laboratory testing. In non-vaccinated flocks, seroconversion can be tested using haemagglutination-inhibition test (fowl red blood cells), ELISAs or serum neutralization tests. Virus DNA can be detected by PCR. The disease must be differentiated from Newcastle Disease, Avian Influenza and Infectious Bronchitis.

Treatment and Prevention

No effective treatment is available. Molting may be induced in laying hens to restore egg production
Obtaining of chicks from an uninfected flock and prevention of horizontal transmission by implementation biosecurity.

Prevention

Inactivated vaccines can be given to birds 14-18 weeks of age and prevent clinical disease. However, virus shedding is not prevented but only reduced.

Fowl Pox

Fowl pox is a slow-spreading viral infection of chickens and other poultry characterized by proliferative skin lesions. The disease is transmitted by direct contact between infected and susceptible birds or by mosquitoes. In Ethiopia fowl pox is common in commercial and backyard chicken.

Clinical Symptoms

Fowl pox is manifested in two forms:
Dry form (cutaneous form) characterized by proliferative nodular wart like lesions on unfeathered areas such as comb, wattles, angle of the beak, feet, vent and other areas of the skin.
Wet form (diphtheritic form) yellowish lesions on mucosa of mouth, pharynx, larynx and trachea and causing respiratory distress. Cutaneous lesions on the eyelids may cause complete closure of one or both eyes and drop in egg production in laying hens.

Diagnosis

Cutaneous lesions are indicative; hematoxylin-eosin staining reveals eosinophilic cytoplasmic inclusion bodies; virus isolation is confirmatory.

Treatment and Prevention

There is no specific treatment
Vaccinate the remaining flocks with an attenuated fowl pox virus of high immunogenicity and low pathogenicity. Vaccination should be executed before expected exposure to FPV, but may also be effective in limiting the spread of the disease when already present but less than 20% of the birds show lesions
Regular cleaning and disinfection
Avoid multi-age sites, control of cannibalism, dust and mosquitos and other insects.
Note: Passive immunity may interfere with multiplication of vaccine virus.

Infectious Bronchitis

"Infectious bronchitis" (IBV) is an acute and a highly contagious viral disease caused by coronaviruses affecting the respiratory, urogenital and intestinal tracts of hybrid layer, meat type and fancy chickens of all age groups. It is a major cause of economic losses around the world. Virions leave the body with mucous secretions from the upper respiratory tract and with fecal droppings. The airborne spread of IBV is the most common and most significant mode of transmission in areas with a dense chicken population.

Clinical Symptoms and Lesions

Young chicks manifest respiratory form characterized by coughing, gasping, sneezing rales nasal discharge, exudate in the eyes, huddling near heat source, reduced weight gain, reduced feed efficiency. Nephrogenic form is characterized by depression, wet droppings and increased water intake, a lower level of egg production. Laying hens sometimes show respiratory signs, drop in egg production up to 70%, increase misshapen eggs, reduced egg size and unpigmented eggshell.

Postmortem lesions include serous to catarrhal tracheitis, airsacculitis and sinusitis of variable degree can occur. Lesion may become caseous. Nephropathogenic strains cause interstitial nephritis leading to pale, enlarged kidneys. In laying hens, fluid yolk material may be observed when infected at a young age.

Diagnosis

Based on identification viral genome, viral antigens or antibodies against the virus. Postmortem lesion are observed in the respiratory tract and urogenital tract but are often aspecific. Note that the most clearly visible respiratory lesions in the field cases are often associated with secondary bacterial infection.

Differential diagnosis: Avian Metapneumovirus mild form of Newcastle Disease, Low Pathogenic Avian Influenza and Infectious laryngotracheitis.

Treatment and Prevention

No specific treatment is available

Mortality can be reduced by supportive treatment and elimination of overcrowding, strict biosecurity and prevention of stress (cold stress)

Antibiotic treatment for secondary bacterial infection may also reduce mortality. For nephropathogenic strain a reduction of protein concentration on feed and provision of electrolytes in drinking water.

Prevention

Vaccination is commonly used worldwide, usually aimed at specific serotypes. First vaccination is administered in the hatchery. Broader protection can be achieved by combining different vaccine strains.

Live vaccines against different respiratory pathogens can interfere with live IBV vaccines and vice versa if applied at short intervals. Simultaneous application can be preferable.

Infectious Bursal Disease

Infectious Bursal Disease (IBD) also known as Gumboro disease is an acute, contagious, viral disease of young chickens (3-6 weeks age). The virus replicates in the bursa of Fabricius and destroys B lymphocytes. It is characterized by diarrhea, trembling and incoordination followed by atrophy of the bursa of Fabricius and by a variable degree of immune suppression. The virus is resistant to most disinfectants and environmental factors and persists for months in contaminated house for weeks in water, feed and droppings. It is shed in the feces and transferred by fomites (direct and indirect contact). Morbidity is generally high and mortality depends on pathogenicity of the strain and immunity of the birds.

Clinical Symptoms and Lesions

The clinical symptoms include vent picking, whitish or watery diarrhea, soiled vent fathers, anorexia, depression ruffled feathers, trembling and severe protrusion. At Necropsy, the bursa may be swollen, edematous, necrotic hemorrhagic and atrophied. Hemorrhages may occur in thigh and pectoral muscles, kidneys may be swollen.

Diagnosis

Presumptive diagnosis can be based on characteristic clinical symptoms such as rapid onset, high morbidity, spiking mortality curve and rapid recovery. A definitive diagnosis is based on isolation of virus from cloacal bursa and spleen and subsequent identification.

Treatment and Prevention

No specific treatment available only drugs to protect from secondary bacterial infections.

Prevention

Depopulation and rigorous disinfection of contaminated farms
Strict hygienic measures including control of personal and material movement
Application of all- in/all-out procedures.
Antibiotics may be used to prevent secondary infection
Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight). **Or**
Chlortetracycline, 3-4 kg/tone of feed, i.e. 20-60 mg/kg 5-7 days.
Vaccination is another means of prevention
Live vaccines of chick-embryo or cell-culture origin and of varying virulence can be administered by drinking water
Live vaccines mainly used in pullets and broiler to provide early protection in addition to maternal antibodies. Maternal antibodies interfere with the effect of (live) vaccination
Vaccines with Delaware variants are also used, alone or in combination with classical strains
A universal vaccination program for infectious bursal disease does not exist, due to variability in maternal antibody levels, management etc.
Vaccination programs for breeders are designated to ensure highest possible pre-point of low levels of antibodies, to provide progeny with high levels of maternal antibodies
In Breeders, Live vaccine are used for priming. Effective priming can also be accomplished by field infection
Inactivated vaccines are used to boost and prolong immunity. These vaccines can contain classical or variant strains. First with a live vaccine and again just before egg production with an oil-adjuvant, inactivated vaccine
Recently, in-ovo vaccination or day one is available. A live IBD vaccine is injected into the egg, alone or in combination with an anti-IBDV antibody.
Monitoring of maternal antibody levels in pullets (breeders flock) can help to determine the optimal vaccination moment and decrease the gap in IBDV immunity.
Vaccination of breeding flocks one or more times during the growing period.

Infectious Laryngotracheitis

Infectious Laryngotracheitis (ILT) is an acute, highly contagious, herpes virus infection of chickens and pheasants characterized by severe dyspnea, coughing, rattling, extension of the head and rales.

Clinical Symptoms and Lesions

Acute form blood, mucus, and yellow caseous exudates or hollow caseous cast in the trachea are observed.
Subacute form punctiform hemorrhagic areas in the trachea and larynx and conjunctivitis with lacrimation, tracheaitis, conjunctivitis, and mild rales. The mouth and beak may be bloodstained from the tracheal exudates and drop in egg production. Mortality may reach 50% in adults.

Diagnosis

Clinical symptoms are suggestive. Demonstration of intranuclear inclusion bodies in the tracheal epithelium early in the course of the disease and isolation and identification of the specific virus or animal inoculation are confirmatory.

Treatment and Prevention

No specific treatment

Prevention

Purchase of new chicks from ILT- free flocks
No mixing of infected or vaccinated birds with naïve birds
Strict biosecurity
Reduce dust level of the house to reduce severity of signs
In endemic areas, vaccination with modified strains of low virulence virus, by eye drop.

Marek's Disease

It is a viral induced-neoplastic and immunosuppressive disease of chickens caused by herpes virus (**Gallid herpesvirus 2** or GaHV-2, genus **Mardivirus**) characterized by infiltration of various nerve trunks and other organs by lymphoid cells. It is most commonly encountered in birds at the age of 8-9 weeks and onwards. The cases at the age of 16-20 weeks and 24-30 weeks are predominant. Transmission occurs by inhalation of virus laden feather follicle or from excretion.

Clinical Symptoms and Lesions

Clinical symptoms of MD usually appear at about 3 weeks of age and peak between 2 and 7 months. Multifocal lymphoid proliferation in a variety of tissues. Tumors are common in the liver, spleen, gonads, kidneys, heart, proventriculus and skeletal muscles and the skin. Cellular infiltration of peripheral nerves, leading to gross enlargement, loss of striation and paralysis. Wing paralysis, blindness, ataxia and wasting away.

Diagnosis

Presence of herpes virus does not always result in clinical disease. Therefore, it is important to diagnose clinical disease instead of detection of herpes virus. Case history, clinical symptoms, gross lesions and histopathology.

Should be differentiated from peripheral neuropathy of unknown etiology and lymphoid leucosis.

Treatment and Prevention

There is no specific treatment

Severely affected flocks should be culled regularly and sick birds euthanized.

Prevention

Strict biosecurity measures

Use of genetically resistant chicks

Avoiding immunosuppression

Proper application of an appropriate vaccine and good biosecurity can prevent clinical disease

Three most commonly used vaccines are 1) Rispens or CVI988, which is a serotype 1 MDV. 2) SB1, which belongs to serotype 2, and 3) HVT, which is a serotype 3 MDV. These vaccines are often combined for a broader protection.

Newcastle Disease

Newcastle Disease (ND) or pseudo-plague is a contagious viral disease of poultry caused by a **Paramyxovirus.** It is characterized by a high variability in morbidity, mortality, clinical symptoms and lesions. ND has huge economic impact. The disease was first reported in Ethiopia in 1974 from exotic chicken. Since then, ND has become cosmopolitan.

Clinical Symptoms and Lesions

PMV-1 are classified into five pathotypes.

Velogenic viscerotropic strains cause high mortality (up to 100%) associated with characteristic intestinal damage

Velogenic neurotropic stains also cause very high mortality (up to 100%) associated with respiratory and nervous disorders

Mesogenic strains are responsible for respiratory and nervous disorders associated with a low mortality rate in adults but a high mortality rate among young birds (up to 50%)

Lentogenic strains cause only respiratory disorders without mortality neither in young nor adult birds

Lentogenic asymptomatic strains cause no clinical symptoms. The clinical symptoms of ND may be neurotropic (drooping wings, dragging legs, twisting of the head and neck, circling, depression, inappetence and complete paralysis) or viscerotropic (respiratory signs gasping, coughing, sneezing and rales with peracute disease; watery-greenish diarrhea and swelling of the tissues of the head and neck). Onset is rapid, young chickens are more susceptible and show signs sooner than older ones, laying flocks may have partial or complete cessation of production and not recover.

Diagnosis
Tentative diagnosis is based on clinical symptoms, lesions and the general epidemic context of the disease diagnosis should be confirmed by isolation and identification of the virus. A rise in hemagglutination-inhibition antibodies in paired serum samples is confirmatory.
Differential diagnosis: acute fowl cholera, avian influenza, Infectious laryngotracheitis and Fowl pox (diphtheritic form).

Treatment and Prevention
No specific treatment. Only bacterial complications can be treated with antibiotics.

Prevention
Control of insects and rodents
Control of water and quality
Use of good litter quality
Use of adequate sanitation and disinfection protocols.
Vaccination - For a good protection level at least one live vaccination, in the absence of maternal antibodies and an inactivated/vector vaccination, should be applied. Live vaccines, individual application methods such as the use of eye drops and beak dipping. Inactivated vaccines can be used in birds after priming with a live virus. A vector vaccine is also an alternative.
Goal of vaccination is to reduce the number of infected birds and the amount of virus shed into the environment.
Control mycoplasma and other bacteria that may act synergistically with some vaccines to aggravate the vaccine reaction after spray administration.
Newcastle disease virus can produce a transitory conjunctivitis in human.

BACTERIAL DISEASES OF POULTRY

Avian Campylobacterisosis

Campylobacteriosis is caused by **Campylobacter jejuni** or **Campylobacter coli**. Environmental contamination is the source of infection. It is a food-borne infection of humans derived from poultry.

Clinical Symptoms and Lesions
Clinical symptoms of infection with **Campylobacter** species range from no clinical symptoms to severe diarrhea and death. This range in clinical symptoms is related to the strain of the organism, the infective dose and the age of the bird at the time of infection. Gross lesions seen with Campylobacter range from no lesions to distension of the intestinal tract, watery fluid in the intestine, and even hemorrhages in cases of infection with cytotoxic.

Diagnosis
Examination of culture.

Treatment and Prevention
Treatment of birds for Campylobacter infection is rare.

Prevention
Strict biosecurity measures for contaminated housing between successive flocks, exclusion of rodents and wild birds and insect eradication
Campylobacteriosis is zoonotic disease transmitted through ingestion of raw contaminated poultry meat and its susceptibility to antimicrobials is important in making a treatment decision.

Colibacillosis

Colibacillosis is a localized or systemic bacterial disease of poultry caused by **E. coli.** Systemic infection usually requires predisposing environmental or infectious causes; most outbreaks occur in chicken under low sanitary standards. Infection is acquired by ingestion or transovarian. Although all ages are susceptible to the disease, young birds are more often affected and usually show more severe infection.

Clinical Symptoms and Lesions

Colibacillosis is characterized by an acute fatal septicemia or subacute pericarditis, sinusitis, omphalitis, airsacculitis and sudden death. Young birds that survive septicemia develop subacute fibrinopurulent airsacculitis, pericarditis, perihepatitis and lymphocytic depletion of the bursa and thymus. Peritonitis, pericarditis and perihepatitis are the findings during postmortem.

Diagnosis

The presence of predisposing factors, typical visceral serosites and epicarditis, coupled with isolation of a pure culture of **E. coli** from heart blood, liver and spleen should be considered.

Treatment and Prevention

Chlorotetracycline HCl, 100-125 mg per kg body weight, or 100 g per 75-100 liters of drinking water during 4-5 days. **Or**

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight per day). **Or**

Sulfamethazine-trimethoprim suspension, 1ml/5 liters of drinking water daily for 5 days.

Or

Amoxycillin trihydrate (20-50%) powder, 200g per 600 ml of drinking water (for 2000 birds each 1kg), 20mg/kg, q 24 h for 3-5 (33.3%) days. **Or**

Tetracycline 13.3 gm/120 liters of drinking water (equivalent to 60 mg/kg feed q 24 h) for 3-5 days; individual birds could be treated at 1ml of 5% SC.

Prevention

Management measures are focused on a reduction of contamination

Obtaining newly hatched birds from disease-free breeder flocks, sanitation of drinking water, access to quality feed, adequate ventilation, avoidance of wet litter and vermin infestation and strict biosecurity measures

Autogenous vaccines have proven successful. Inactivated vaccines specific to some serogroups, such as O2:K1 and O78:K80, and live or recombinant vaccines are also effective against specific strains.

Note: It is best to perform a sensitivity test in order to select the proper antibiotic. However, the results of antibiotic treatments are often disappointing. Therefore, emphasis should be made on prevention. Earlier treatment is advised.

Chlamydiosis

Chlamydiosis is subclinical, acute, subacute or chronic disease of wild and domestic birds characterized by systemic, respiratory or digestive signs and lesions. It is caused by **Chlamydia psittaci**, an obligate intracellular bacterium. The organism is excreted via secretions or excretions and transmitted through inhalation.

Clinical Symptoms and Lesions

Nasal and ocular discharges, conjunctivitis, sinusitis, green to yellow-green droppings, inactivity, ruffled feathers, weakness, inappetence reduced feed consumption and weight loss. Air sacculitis, pericarditis, periohepatitis and peritonitis with serofibrinous exudates and hepatosplenomegaly are common in acute cases. In chronic cases enlarged spleen or an enlarged, discolored liver or both occur.

Diagnosis

Impression smears of affected tissues stained by Giemsa, Gimenez or Macchiavello's methods are sufficient to demonstrate intracellular organisms.

Treatment and Prevention

Chlortetracycline, 20-30 mg/kg or 400-750 g/ton for a minimum of 2 weeks in feed. **Or**
Doxycycline, 20 mg/kg, PO in drinking water, daily for 3-5 days.

Alternative

Flumequine 12 mg/kg body weight, diluted 1:1500-2000 in drinking water for 3-5 days.

Note: Require extended treatment for 2-6 weeks. Recovered birds remain carriers.

Respiratory discharges or feces are infective; air borne particles and dusts may harbor the organism.

The organism can be transmitted to humans and cause eye lesions.

Fowl Cholera

Fowl cholera, **(Avian Pasteurellosis** or **avian hemorrhagic septicemia)** is a contagious bacterial disease of poultry characterized by septicemia, sudden onset with high morbidity and mortality. Transmission occurs by secretions from carrier birds, infected droppings, cannibalism of dead birds and contaminated water, feed, equipment or clothing. The organism is primarily excreted from mouth, nose and conjunctiva of diseased birds that contaminate their environment.

Clinical Symptoms and Lesions

In acute: sudden death in the flock, fever, depression, anorexia, mucoid discharge from the mouth, ruffled feathers, diarrhea, bluish discoloration of the head and increased respiratory rate.

In chronic: swelling of the joints, footpad, wattles or tendon sheaths over the caudal portion of the hock joint could be observed. Conjunctivitis, rales and torticollis.

Postmortem lesions include localized infection of oviduct, peritoneal cavity.

Diagnosis

The disease is caused by **P. multocida** and commonly reported in commercial flock in Ethiopia. History, clinical symptoms and lesions are presumptive; bacterial culture and isolation of **P. multocida** are definitive.

It should be differentiated from HPAI, NCD, Collibacillosis, fowl typhoid, fowl pox.

Treatment and Prevention

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight per day). **Or**
Oxytetracycline Hcl 200 mg, 80 mg per Kg body weight daily, during 3-5 days, or 40 g per 100-150 liter of drinking water for 3-5 days. **Or**
Amoxicillin (as trihydrate) 150 mg, 16 mg/kg BW or 100 g per 100 liters of drinking water for 3-5 days. **Or**
Flumequine 10 mg per kg body weight, 250 ml per 1000 liters of drinking water during 3-5 days. **Or**
Enrofloxacin 10 mg/kg in drinking water for 3-5 days. For **Or**
Sulphonamides (Sulphadimethoxazole, Sulphaquinoxalene, Sulphamethazine and sulphaquinonxalene) 0.5-1% (500mg-1gm/kg) in feed or 0.1% (100mg/liter) in drinking water. **Or**
Chlortetracycline 10%, 20-60 mg/kg, daily in feed for 5 to 7 days or should be continued until signs of the disease are no longer apparent. **Or**
Amoxicillin trihydrate water-soluble crystal, 20 mg/kg in drinking water, once per day for 3-5 days.

Prevention

Vaccination of pullet is possible in areas with high prevalence based on serotype. Sanitation, rodent and predator control and proper disposal of dead birds is important as preventive measure.

Note: Early treatment and adequate dosages are important. Sensitivity testing often aids in drug selection.

Fowl Typhoid

Fowl typhoid is an acute or chronic septicemic disease caused by host-adapted salmonella serotype, **Salmonella gallinarum.** It is an egg-transmitted infection of particularly growing or mature flocks or transmitted by ingestion. Morbidity and mortality are highly variable depending on age, nutrition, flock management, concurrent diseases, route and dose of exposure. In chicks, death rate may vary from 10-93%.

Clinical Symptoms

Common signs in young chicks are severe depression, pumping respiration, decreased appetite, slime in faces, white urates on the cloaca and mortality up to 93%. Decrease hatchability (in case of vertically transmitted disease). Common signs in mature birds include misshapen and discolored cystic or nodular ova among normal ova, lowered feed intake, egg production, hatchability, inactivity, acute septicemia and mortality.

Lesions include enlarged liver, spleen and kidney, white nodules in liver, spleen, cardiac muscle, pancreas, lungs and caecal wall, salpingitis, peritonitis, caecal cores (Cheesy material in the cecum), swollen joints, containing yellow and viscous fluids.

Diagnosis

Isolation and identification of the causal agent.

The disease must be differentiated from **P. Multocida, E. coli,** Newcastle Disease and Highly Pathogenic Avian Influenza.

Treatment and Prevention

Oxytetracycline Hcl 200mg, 80mg per kg body weight daily, during 3-5 days, or 40 g per 100-150 liter of drinking water during 3-5 days. **Or**

Sulphachloropyrazine 300 mg, 100g per 100 liter of drinking water during 5 days for chicks and pullets, as well as for laying hens.

Prevention

Poults should be obtained from free stock and place them in a clean house. Culling of infected breeder flocks is preferable, proper maintenance of biosecurity, strict sanitation of the poultry houses and vaccination with a rough strain of **S. gallinarum** (9R) at 6-7 weeks of age helps in prevention.

Antibiotic treatment is not always useful because recovered birds can become carriers. The method of choice to control the disease is eradication.

Fowl Paratyphoid

Paratyphoid infections are caused by any one of the many non-host-adapted salmonellae serotypes such as **Salmonella typhimurium** (most common), followed by **S. enteritidis** and **S.arizonae.**

Infections are often subclinical and mortality is increased due to shipping, delayed feeding, chilling or overheating. Depression, poor growth, weakness, diarrhea and dehydration may be seen. The clinical symptoms are not distinctive. Post-mortem lesions are enlarged liver with necrosis, enlarged spleen, unabsorbed yolk sac and necrotic lesions in the intestinal mucosa and caeca filled with cheese-like exudate.

Diagnosis

Isolation and identification of the causal agent is essential.

Differentiatial diagnosis: Pullorum disease, Newcastle Disease and staphylococcosis.

Treatment and Prevention

Spectinomycin, 100 mg/kg q12h PO for 3-7 days in 1-2 gm/gallon of drinking water; Chicks 1-3 days old can receive 2.5 to 5 mg/bird, SC. **Or**

Trimethoprim-Sulphaquinoxalene sodium (1:5), 30 mg/kg PO in drinking water, q 24 h, for 5 days or until two days after symptoms have subsided or 50-150 g per 200 liter of drinking water during 3-5 days, but not exceeding 14 days altogether. **Or**

Oxytetracycline Hcl 500mg, 80mg per kg body weight daily, during 3-5 days, or 40 g per 100-150 liter

of drinking water during 3-5 days. **Or**
Flumequine 10 mg per kg body weight, 250 ml per 1000 litres of drinking water during 3-5 days. **Or**
Colistin (As sulphate) 4,800,000 IU, 100 g per 500 - 1000 liters of drinking water during 5 days.
Prophylaxis
Vaccines against **S. enteritidis** and **S. typhimurium** are available for prevention
Strict sanitation, strict biosecurity and early fumigation of hatching eggs and treatment of the feed with heat to kill the bacteria are advisable.
Public health significance: Paratyphoid salmonellae are often zoonotic, logistic slaughter and heat treatment may be used to reduce the exposure of people to Salmonella from poultry products. Obtain the chicks and eggs from salmonella free breeding stocks.

Infectious Coryza

Infectious Coryza (IC) is an acute respiratory disease of chickens caused by the bacterium **Avibacterium paragallinarum** (**A. gallinarum**). Chronically ill or healthy carrier birds are the reservoir of infection. Transmission is by direct contact, airborne droplets and by contamination of drinking water. Typically, the incubation period is 1-3 days and the duration of the disease is usually 2-3 weeks for a simple infection. Usually the disease shows high morbidity and a low mortality.

Clinical Symptoms and Lesions

Serous to mucoid nasal discharge sometimes even purulent sneezing, facial swelling, conjunctivitis, abscesses in the head and wattles, dyspnea, sinusitis, tracheitis, airsacculitis, hepatitis, endocarditis, salpingitis, oophoritis, peritonitis and synovitis. In layers, severe reduction in egg production (10-40%); in chronic cases, a foul odor.

Diagnosis

History, clinical expression of disease and identification of agent.
The disease must be differentiated from chronic fowl cholera, Newcastle Disease, Infectious Bronchitis, Avian Influenza, mycoplasmosis and infectious laryngotracheitis.

Treatment and Prevention

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight), Or
Oxytetracycline (20%), 200g/100 liters of drinking water for 3-5 days. **Or**
Enrofloxacin, one liter suspension /1500-4000 liters of water for 3-5 days. **Or**
Sulfamethazine-trimethoprim, 1000 mg/l in drinking water or 2000g/ton for 3 days. **Or**
Amoxicillin 16 mg/kg bw or 100 g per 100 liter for 3-5 consecutive days**.**

Prevention

After cleaning and disinfection poultry house should remain empty for 2-3 weeks before restocking.
Avoid multi-age farms
Vaccination of pullets in endemic area with serotype A, B and C may help to control the disease.
Note: Antibiotic treatment does not eliminate the bacteria. Antibiotics are applied to alleviate the severity of the course of the disease. Sulphonamides are contraindicated in layers.

Gangrenous Dermatitis

Gangrenous Dermatitis (GD) is a sporadic skin infection characterized by necrosis of the skin and subcutaneous tissue of breast of poultry between 4 and 6 weeks old for broilers and 6-20 weeks old for pullets caused by **Staphylococcus aureus.** Clostridial species such as **Clostridium septicum** may also be involved. The disease precipitates due to immune suppression associated with infectious bursal disease, poor environmental conditions such as high litter moisture and failing to remove dead birds and skin damage caused by scratching overcrowding or meal time feeding.

Clinical Symptoms and Lesions

Sudden onset, depression, lethargy, sudden death, sharp increase in mortality within 8-24 hours and gangrenous necrosis of the skin over the wings, abdomen, thighs and breast are common. At necropsy, cellulitis of subcutaneous tissue, hemorrhagic edemas beneath the skin. Emphysema under the skin and the underlying musculature has a cooked appearance.

Diagnosis

Tentative diagnosis can be based on history (rapid onset, high mortality, low morbidity) and characteristic skin lesion. Definitive diagnosis is based on isolation and identification of **C. septicum** or **S. aureus** (or both).

Treatment and Prevention

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight). **Or**

Tetracyclines and erythromycin. (See Treatment under "Diseases of Poultry", Mycoplasma synoviae infection). **Or**

Amoxicillin 16 mg/kg bw or 100 g per 100 liter for 3-5 consecutive days. **Or**

Tilmicosin (as phosphate) 25%, 300 ml per 1000 litres of drinking water during 3-5 days (20mg/kg body weight).

Prevention

Maintaining proper litter condition, avoiding overcrowding, strict sanitation and disinfection in the poultry house, minimizing mechanical injury and controlling cannibalism and removing dead birds regularly will help in prevention

Vaccinate breeders against Infectious Bursal Disease.

Earlier treatment is advisable. Oxytetracycline, 0.02% in the feed rapidly reduces mortality in field outbreaks of clostridial infections.

Mycoplasma Synoviae Infection

Infectious synovitis is an acute or chronic mycoplasmosis of chicken and turkeys caused by **Mycoplasma synoviae**, which is characterized by inflammation of synovial membranes exudates in the joints and tendon sheaths. Most outbreaks occur in young broiler chicken, though adults are also infected. Transovarian transmission is the most important route of infection. Infection can also spread via aerosols, bird to bird contact or ingestion of contaminated feed or water.

Clinical Symptoms and Lesions

Affected birds rest on the floor; head and hocks or foot pads are swollen. At post mortem, the joints contain sticky, viscid, gray to yellow, characteristic exudates. Parenchymatous organs (liver and spleen) become swollen, airsaculitis and diarrhea may occur.

Diagnosis

Signs and lesions are suggestive; agglutination reactions and ELISA are commonly used for diagnosis. **M. synoviae** should be confirmed by isolation and identification in embryonated eggs or by hemagglutination-inhibition.

Differential diagnosis: Diseases caused by **Avibacterium parragallinarum, M. gallisepticum,** Newcastle Disease, Low Pathogenic Avian Influenza, **Avian metapneumovirus,** Infectious laryngotracheitis, Infectious Brochitis, **E. coli, S. aureus, Enterococcus faecalis, Reovirus.**

Treatment and Prevention

Tylosin 100 g per 200 liters (0.05%) of drinking water for the first 3 days of life; repeated for 1 to 7 days every 5 weeks depending on the severity of the infection. **Or**

Erythromycin, 500mg/gallon of water, for 7 days. **Or**

Chlortetracycline, 20-60 mg/kg or 300-400 mg/ton of feed, for 5 days. **Or**

Tilmicosin, 15-20 mg/kg BW in chickens and 10-27 mg/kg BW in turkeys for 3 days, which may be achieved by the inclusion of 75 mg tilmicosin per litre (30 ml Tilmicosin per 100 litres).

Prevention

Maintain sero-negative stock, because the organism is transmitted via eggs

Antimicrobial treatment is applied in diseased flocks in order to alleviate the severity of the course, but also contributes to the reduction of shedding and vertical transmission. However treatment has short-term effect

Elimination of egg transmission by culling of the infected reproduction stock is not economically sustainable in a situation of high prevalence. In situation of high prevalence antibiotic treatment and vaccination programs may contribute to the reduction of the clinical impact of the disease.

Note: Treatment is most effective if given by injection.

Effective biosecurity measures are of importance for the reduction of horizontal transmission.

Necrotic Enteritis

Necrotic enteritis is an acute enterotoxemia characterized by sudden onset, explosive mortality and confluent necrosis of the mucosa of the small intestine associated with **Clostridium perfringens** Type A and less often type C. It particularly affects broiler chickens at 2-5 weeks of age, but also reported in pullets and laying hens near the start of egg laying and during peak of laying most frequently in houses with built-up litter. Presence of pathogenic **C. perfringens**, coccidiosis, alteration in nutrition and stress are predisposing factors.

Clinical Symptoms and Lesions

Severe depression, inappetence, ruffled feathers, dark-colored diarrhea, in laying hens decreased egg production and sudden increase in death rate. The breast becomes dehydrated and darkened and the liver is usually swollen and congested. The small intestine is ballooned and friable and contains foul-smelling, brown fluid; necrotic mucosa covered with a pseudomembrane. Subclinical infection signs include lower performance and reduced weight gain.

Diagnosis

Tentative diagnosis based on post-mortem examination with isolation and identification of bacterium.

Stained smear preparations are indicative and confirmed by anaerobic cultural examination.

Differential diagnosis: Coccidiosis and Ulcerative enteritis

Treatment and Prevention

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight). **Or**

Amoxicillin 16 mg/kg bw or 100 g per 100 liter for 3-5 consecutive days. **Or**

Erythromycin, 500 mg/gallon of water for 7 days.

Prevention

Use of coccidiostats or anticoccidial vaccines

Optimizing of feed composition by avoidance of changes and minimizing of fishmeal wheat, barley and rye in the diet

Minimizing stress factors, strict hygiene and disinfection measures

Administration of probiotic to reduce the prevalence and severity of necrotic enteritis

Due to the rapid course of the disease antibiotic treatment can be difficult. The control should therefore be focused on predisposing factors.

Pullorum Disease

Pullorum (Bacillary white diarrhea) is a bacterial disease caused by **S. pullorum** that usually causes high mortality in young chickens which have major economic impact. Infection is transmitted vertically via egg or horizontally in the hatchery usually results in death that occurs during the first few days of life and up to 2-3 weeks of age. The highest mortality rate may be observed during the second week after hatching in vertically infected flocks.

Clinical Symptoms and Lesions

Decreased hatchability (in case of vertical transmission), severe depression, weakness, decreased appetite, slime in faces, blindness, pumping respiration and whitish, chalky urates fecal pasting around the vent. Recovered animals remain carriers.

Lesions in young birds include unabsorbed yolk sacs, focal necrosis of the liver and spleen. Firm, cheesy material in ceca and raised plaques in the mucosa of the lower intestine.

Adult carriers usually have pericarditis, peritonitis or distorted ovarian follicles with coagulated cystic ova, sometimes salpingitis and peritonitis. However, sometimes no gross lesions are seen.

In mature chickens, acute infections produce lesions that are indistinguishable from those of fowl typhoid.

Diagnosis

A tentative diagnosis of PD can be made based on the flock history, clinical symptoms, mortality and lesions. Lesions may be highly suggestive, but diagnosis should be confirmed by isolation and identification of **S. pullorum**. Tube-agglutination or serum-plate test could be used as screening tests. Differential diagnosis: Colibacillosis, pasteurellosis, erysipelas, paratyphoid infections, Highly Pathogenic Avian Influenza (HPAI) and Newcastle Disease.

Treatment and Prevention

Trimethoprim-Sulphaquinoxalene sodium (1:5), 30 mg/kg PO in drinking water, q 24 h, for 5 days or until two days after symptoms have subsided or 50-150 g per 200 liter of drinking water during 3-5 days. **Or**

Oxytetracycline Hcl 500mg, 80mg per kg body weight daily, during 3-5 days, or 40 g per 100-150 liter of drinking water during 3-5 days. **Or**

Gentamicin 5-10mg per Kg body weight in drinking water for 4-5 days

Prevention

Breeding stock should be free from infection

Obtaining chicks from **S. Pullorum** free farms

Elimination of carriers and sound biosecurity program will go a long way in preventing PD.

Antibacterial treatment helps to reduce mortality and treated chicken remain carriers.

Every effort should be made to eradicate PD and treatment should be the last option. Various sulfonamides followed by nitrofurans and other antibiotics have been found to be effective in reducing mortality.

Salmonelloses

Salmonellosis in chicken and turkey is caused by host adapted serotypes including **Salmonella pullorum** and **S. gallinarum** causing fowl typhoid or other non-host adapted serotypes such as **S. typhimurium** and **S. arizonae** causing paratyphoid infections. The latter are the most important as zoonotic diseases.

Treatment and Prevention

See treatment for Pullorum disease.

Spirochetosis

Avian spirochetosis is an acute, febrile and septicemic disease of poultry which is transmitted by the tick spp. **Argas pericus.** The disease affects a wide variety of birds.

Clinical Symptoms and Lesions
Listlessness, depression, somnolence, moderate to marked shivering and increased thirst are observed. Young birds are affected more severely than older ones. During the initial stages of the disease, there is usually a greenish yellow diarrhea with increased urates.

Diagnosis
Dark field microscopic examination of blood and demonstration of **Borrelia,** the active motile organism or as stained spirochetes in Giemsa-stained blood smears. Anemia is common.

Treatment and Prevention
Amoxycillin trihydrate and other penicillin derivatives, 20%, 100g/10 L drinking water for 3-5 days. **Or**
Chlortetracyline, 10%, 20-30 mg/kg to individual birds for 5 to 7 days in feed.

Prevention
Control of tick species that serve as vector.

Staphylococcosis

Staphylococcus infections caused by **Staphylococcus aureus** is usually characterized by synovitis and septicemia. The organisms are normal flora of the skin. Most infections occur because of a wound, either accidental or intentional (e.g. debeaking or detoeing).

Clinical Symptoms and Lesions
Omphalitis, synovititis and lameness due to involvement of the proximal tibiotarsus and proximal femur (Osteomyelitis), the proximal tarsometatarsus, distal femur and tibiotarsus. Gangrenous dermatitis, bumble foot and septicemia.

Diagnosis
Isolation of **Staphylococcus aureus.**

Treatment and Prevention
Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight). **Or**
Oxytetracycline Hcl 500mg, 80mg per kg body weight daily, during 3-5 days, or 40 g per 100-150 liter of drinking water during 3-5 days. **Or**
Amoxicillin 16 mg/kg bw or 100 g per 100 liter for 3-5 consecutive days **Or**
Tilmicosin (as phosphate) 25%, 300 ml per 1000 litres of drinking water during 3-5 days (20mg/kg body weight). **Or**
Erythromycin, 500 mg/gallon of water for 7days. **Or**
Sulphadimethoxine, 1892 mg/gallon of water or 0.05% solution for 6 days.

Prevention
Prevention of specific immunosuppressive diseases
Avoid using sharp objects in poultry houses or during transportation
Proper sanitation of hatchery
Competitive gut exclusion using **Lactobacillus acidophilus** protects germ-free chickens.
Note: Sensitivity test should be performed because antibiotic resistance is common.
Proper precautions must be taken as Staphylococcus aureus can cause food poisoning and Enterotoxin-producing strains are found in poultry.

Streptococcosis

Streptococcosis in poultry is a systemic or local disease due to infection with the streptococci, which are opportunistic pathogens. **Streptococcus zooepidemicus** and **S. faecalis** are considered the most pathogenic in avian species; however, all streptococci are capable of causing severe disease. Transmission is via the oral or aerosol route.

Clinical Symptoms and Lesions

In acute form, birds show depression, lethargy, lassitude, pale combs and wattles and a decrease in or cessation of egg production. Necropsy lesions include an enlarged spleen, liver and kidney.

In chronic form, lameness, swollen hock and wing joints, conjunctivitis and depression with emaciation. Associated lesions include fibrinous arthritis and synovitis, salpingitis, pericarditis, perihepatitis, necrotic myocarditis and valvular (vegetative) endocarditis are observed.

Diagnosis

Isolation and identification of the bacteria.

Treatment and Prevention

See, Diseases of poultry - Staphylococcosis

Prevention

Reduce stress and prevent immuno-suppressive diseases and conditions. Cleaning and disinfection is also effective

Appropriate housing conditions and flock management

Egg sanitation to prevent eggshell contamination.

Note: Sensitivity test should be performed before antibiotic treatment; because antibiotic resistance is common.

Tuberculosis

Tuberculosis is a slow spreading, chronic, granulomatous bacterial infection, characterized by gradual weight loss. **Mycobacterium avium** serovars 1 and 2 are the usual causes. Disease is observed in birds above one year of age. Infected birds excrete the organism through feces. Cadavers and offal may infect predators and cannibalistic flock mates.

Clinical Symptoms and Lesions

Signs usually develop late in the infection when birds become depressed; progressive weight loss, breast muscle atrophy, diarrhea, in laying hens reduced egg production and lameness may be seen. In young chicks, yellow to grey granulomatous nodules of varying sizes are usually found in the liver, spleen, bone marrow and intestines.

Treatment and Prevention

No treatment exists

Prevention

No vaccines are available

The entire flock should be culled. Rapid turnover of chicken flocks

Contaminated equipment should be destroyed and several centimeters of the ground should be removed from the poultry house. The poultry house should be cleaned and disinfected

Implementation of strict biosecurity measures which include control of rodent and wild bird contact.

Mycobacterium avium may cause disease in human.

Ulcerative Enteritis

Ulcerative enteritis is an acute bacterial infection in young chickens, poultry and game birds characterized by sudden onset and rapidly increasing mortality. It is caused by **Clostridium colinum.** Outbreaks of the disease occur following coccidiosis, aplastic anemia and infectious bursal disease or stress conditions. It is transmitted through droppings and birds are infected by feeding or via drinking water.

Clinical Symptoms and Lesions
Infected birds become listless and humped up, with eyes partly closed and feathers dull and ruffled. The lesions include hemorrhagic enteritis in duodenum followed by necrosis and ulcerations. The livers vary from light yellow to large, irregular yellow areas along the edges.

Diagnosis
Gross postmortem lesions and isolation of the large Gram-positive sporulated bacteria are sufficient.

Treatment and Prevention
Streptomycin sulphate 60g/ton of feed or 1 g/gal in water. **Or**
Phenoxymethylpenicillin 13.5 –20 mg phenoxymethylpenicillin/kg BW per day for 5 days

Prevention
Streptomycin sulphate, 0.006% in feed as a prophylactic dose
Remove contaminated droppings and disinfect the house
Avoid stress.
Note: Earlier treatment is advisable.

FUNGAL DISEASES OF POULTRY

Aspergillosis

Aspergillosis is a mycotic disease caused by the fungus **Aspergillus fumigatus** that affects all species of birds. The organism is found in litter and feed. Infection occurs predominantly by inhalation of spore.

Clinical Symptoms
Acute Aspergillosis occurs in young birds usually characterized by outbreaks with high morbidity and mortality. It is manifested by respiratory distress and reduced feed intake. The chronic form, which occurs in mature birds, is manifested by reduced feed intake and is economically important. The most frequent clinical symptoms are dyspnea, gasping, hyperpnoea, cyanosis and usually without rales.
The mortality rate varies between 5 and 50% in the first one to three weeks of age. Other signs include diarrhea, anorexia, somnolence, progressive emaciation and increased thirst. Few animals show nervous signs. Pulmonary lesions are cream-colored plaques a few millimeters to several centimeters in diameter; which may occur in the larynx, air sacs, liver, intestines and occasionally the brain and mediastinal canthus of the eye.

Diagnosis
The signs and gross lesions are suggestive; culture or microscopic examination of the fungus are confirmatory.

Treatment and Prevention
Treatment of affected birds is considered useless
Strict adherence to sanitation procedures in the hatchery and fumigation of contaminated hatcheries with formaldehyde or thiabendazole (120-360 g/m3)
Avoid moldy litter or ranges; contaminated feed could be sprayed with nystatin.
Aspergillosis is zoonotic, it is transmitted via inhalation or ingestion.
Use of formaldehyde is prohibited in some countries. Human exposure should be avoided, and gas masks and protective clothing are essential.

Mycotoxicoses

Mycotoxicosis is a disease caused by toxic fungal metabolites that results from fungi growing in grains and feeds. Hundreds of mycotoxins have been identified, many of which have additive or even synergistic effects with other mycotoxins, infectious agents and nutritional deficiencies. Many are quite stable chemically and maintain toxicity over time. The following are some of the most important groups:

Aflatoxicosis

Aflatoxins are highly toxic, carcinogenic fungal metabolites produced by **Aspergillus flavus, A. parasiticus**, and others. Aflatoxicosis in poultry primarily affects the liver but immunologic, digestive and hematopoietic functions may be involved. Weight gain, feed intake, feed conversion efficiency, pigmentation, processing yield, egg production, male and female fertility and hatchability may be affected. Susceptibility varies, but in general, ducklings, turkeys and pheasants are susceptible Chickens, Japanese quail and guinea fowl are relatively resistant.

Clinical Symptoms and Lesions
Unthriftiness to high morbidity and mortality, the liver may be acutely reddened due to necrosis and congestion or yellow discoloration due to lipid accumulation; it may have hemorrhages of various sizes and patterns. In chronic aflatoxicosis, the liver may be discolored yellow to gray and atrophied. Although aflatoxins are carcinogenic, tumor formation is rare with the natural disease, probably because the birds do not live long enough for this to occur.

Ergotism

Claviceps spp are fungi that attack cereal grains, including wheat and especially rye. The mycotoxins that cause ergotism form in the sclerotium, a visible, hard, dark mass of mycelium that displaces the grain tissue. Within the sclerotium are the ergot alkaloids, which affect the nervous system, causing convulsive and sensory neurologic disorders. In the vascular system, vasoconstriction and gangrene of the extremities occur. In the endocrine system it influences neuroendocrine control of the anterior pituitary.

In poultry, the vasoconstrictive effect results in vesicles on the comb, wattles, face and eyelids that rupture to form crusts, which may be followed by permanent atrophy and disfigurement. Vesicles also develop on the shanks of the legs and on the tops and sides of the toes and rupture to form ulcers. In chicks, the toes may become darkly discolored due to vasoconstriction and ischemia. In laying hens, feed consumption and egg production are reduced.

Fusariotoxicosis

The genus **Fusarium** produces many mycotoxins injurious to poultry.

Clinical Symptoms and Lesions
Feed refusal, caustic injury of the oral mucosa and areas of the skin in contact with the mold, acute digestive disease and diminished function of the bone marrow and immune system. Lesions include necrosis and ulceration of the oral mucosa, reddening of the mucosa of the remainder of GI tract, mottling of the liver, atrophy of the spleen and other lymphoid organs and visceral hemorrhages. In laying hens, egg production may decrease, accompanied by depression, recumbency, feed refusal and cyanosis of the comb and wattles. Deformity of the bones may occur in others.

Ochratoxicosis

Ochratoxins are among the most toxic mycotoxins to poultry. These nephrotoxic metabolites are produced chiefly by **Penicillium viridicatum** and **Aspergillus ochraceous**, which commonly occur on numerous grains and feedstuffs. It is a renal disease but also affects the liver, immune system and bone marrow.

Clinical Symptoms and Lesions
Reduced spontaneous activity, huddling, hypothermia, diarrhea, rapid weight loss and death are common. Sublethal intoxication can seriously impair weight gain, feed conversion, pigmentation, carcass yield, egg production, fertility and hatchability.

Citrinin Mycotoxicosis

Citrinin is a natural contaminant of corn, rice and other cereal grains. It is produced by numerous species of **Penicillium** and **Aspergillus.** Spontaneous citrinin mycotoxicosis causes a diuresis that is manifested as watery fecal droppings and reductions in weight gain. At necropsy, lesions involve chiefly the kidney. Citrinin acts directly on the kidney to transiently alter several tubular transport processes.

Oosporein Mycotoxicosis

Oosporein is a mycotoxin produced by **Chaetomium** spp that can cause gout and high mortality in poultry. **Chaetomium** spp have been isolated from numerous feeds and grains, including peanuts, rice and corn. Oosporein mycotoxicosis is characterized by visceral and articular gout related to impaired renal function and increased plasma concentrations of uric acid. Chickens are more sensitive to oosporein than turkeys. Water consumption increases and fecal droppings may be fluid in nature.

Cyclopiazonic Acid

This is a metabolite of **Aspergillus flavus**, which is the predominant producer of aflatoxin in feeds and grains. In chickens, cyclopiazonic acid causes impaired feed conversions, decreased weight gain and mortality. Lesions are found in the proventriculus, gizzard, liver and spleen. The proventriculus is dilated and the mucosa is thickened due to hyperplasia and ulceration.

Sterigmatocystin

This is a biogenic precursor to aflatoxin and is also hepatotoxic and hepatocarcinogenic. It is less common than aflatoxin.

Diagnosis of Mycotoxicosis
Mycotoxicosis should be suspected when the history, signs and lesions are suggestive of feed intoxication. Definitive diagnosis depends on a complete diagnostic evaluation including necropsy, histopathology, accompanied by identification and quantification of the toxins in feed from multiple samples.

Treatment and Prevention
There is no specific treatment

Prevention
The toxic feed should be removed and replaced with uncontaminated feeds. Regarding aflatoxicosis, supplementation of vitamins and increase of crude protein and dietary energy may neutralize the toxic effects. Regarding T2 toxins and ochratoxins A, Vitamin E and C may neutralize the toxic effects

Concurrent diseases (parasitic, infectious and nutritional) should be treated to alleviate additive or synergistic disease interactions
In individual birds, give activated charcoal in the feed
Regular inspection of feed storage, keeping the storage time of feed as short as possible and adequate protection against rain
Inspect feed mills regularly and clean any feed residues
Poultry houses should have sufficient ventilation to avoid high relative humidity
Addition of Mycotoxin-binding agents
Antifungal agents such as organic acids (propionic acid 0.5-1.5 g/kg) are effective inhibitors.

PARASITIC DISEASES OF POULTRY

Coccidiosis

Avian coccidiosis is a protozoal disease caused by **Eimeria spp** affecting usually young growing birds which is frequently characterized by diarrhea and enteritis. Cross protection does not occur among the species. Coccidial oocysts are present in litter and can be transmitted by feed, water or soil ingestion. Severity of infection is dependent on Emeria species, number of ingested sporulated oocyst, age and immune status of the bird.

Clinical Symptoms and Lesions

Decreased growth rate, severe diarrhea and high mortality, feed and water consumption are depressed followed by weight loss, decreased egg production and increased mortality.
The location of the lesions in the intestine depend on the species of **Eimeria** involved. **Eimeria acervulina** is located in the upper small intestine; **E. bruneti** in the lower part of the intestine; **E. maxima** and **E. necatrix** in the middle intestine and **E. tenella** localizes in the caecum.

Diagnosis

The lesions are not characteristic. Demonstration of oocysts in feces or intestinal scrapings coupled with the presence of clinical symptoms should be considered during diagnosis.

Treatment and Prevention

Sulfachlorpyrazine sodium monohydrate, 300mg/kg (0.03%) or 100 gm. per 100 liters in drinking water for 3 days. **Or**
Trimethoprim-Sulphaquinoxalene sodium (1:5), 30 mg/kg PO in drinking water, q 24 hr. for 5 days or until two days after symptoms have subsided or 50-150 g per 200 liter of drinking water during 3-5 days, but not exceeding 14 days altogether. Adequate water should be given. **Or**
Amprolium HCl, 125mg/kg (0.0125%) in feed or 38.4 mg/ml in drinking water for 1-2 weeks or 1.2g/l/day for 3-5 days by continuous feeding. **Or**
Amprolium HCl 200 mg + Sulphaquinoxaline base 200 mg + Vitamin K3 2 mg, 100 g per 150 liter of drinking water for 5-7 days (for 2500 chickens of 5 weeks age or 1500 chickens of 10 weeks of age).Or
Sulphadimidine Sodium100mg/kg BW (1 g powder per 1-2litres of drinking water) In case of acute coccidiosis: Administration during 3 consecutive days; after 2 days of no treatment, another 3 day treatment. In case of subclinical coccidiosis: Administration for 3 consecutive days.**Or**
Toltrazuri 25 mg, 7mg per kg body weight or 100 ml per 100 liter of drinking water for continuous medication during 48 hours or 300 ml per 100 liters of drinking water for 8hours each day during 2 days. **Or**
Diclazuril 25 mg, 0.3 mg per kg body weight per day during 3 days.) **Or**
Monensin, 20 mg/kg, premix. **Or**
Clopiodol, 125 mg/kg (0.0125%) of feed, by continuous feeding. Note that infected birds remain carriers despite treatment.

Prevention

Amprolium HCl (25%) Plus Ethopabate (1.6%) combination, 500g/ton of feed, from day-old to 16-18 weeks of age. Or

Lasalocid sodium 75-125 mg/kg continuously feeding from day old to adult age **Or**

Clopidol for applications and formulations, see treatment above.

The main intervention against coccidiosis is the buildup of immunity after natural infection or vaccination. Treatment is recommended only if it is commenced at early stage of the disease. Other prevention methods are use of vaccines and strict hygiene measures

Plan a rest period for every product used. Avoid contact of Clopiodol with skin and eyes; if it occurs, rinse immediately with water. Resistance against anticoccidial drugs occurs. The rapidity and the severity of resistance differ between anticccidials.

Histomoniasis

Histomoniasis or Black head is a disease of chicken and occasionally other galliform birds caused by the protozoan parasite **Histomonas meleagridis**. It is transmitted most often in embryonated eggs of the cecal nematode **Heterakis gallinarum** and sometimes directly by ingestion of contaminated feces. These worms' eggs are extremely resistant to environmental condition and remain infective for the periods of 2-3 years.

Clinical Symptoms and Lesions

Listlessness, drooping wings and sulfur-colored droppings, cyanosis of head. It is more acute in young birds and birds die within a few days; older birds may become emaciated before death. Make differentiation from **Eimeria tenella** and Salmonella infections. In case of caseous or cheesy cores in the caeca and from **Tetratrichomonas gallinarum granulomas**, coligranulomas, infections with **staphylococcus, clostridium** or **Mycobacterium avium**, sulphonamide intoxications and liver tumors (including Marek's disease) in case of liver lesions.

Diagnosis

Diagnosis is based on pathognomonic ulceration of the ceca and necrotic lesions in the liver. A more accurate diagnosis is based on molecular diagnosis (PCR) of the agent is recommended.

Treatment and Prevention

Dimetridazole, 0.06-0.08% in feed, for 7 days. The same drug can be used for prophylaxis at a rate of 0.015-0.02% in feed, for 5 days.

Prevention

Strict biosecurity focusing on:

Avoidance of birds access to cecal worm ova

Control of H. gallinarum infection through deworming

Separate housing for turkey and chicken

Do not house turkeys and chickens on infected soil for at least one year

No effective vaccinations are available.

Note: Flocks with clinical disease should be treated as soon as possible. Immediately after the start of treatment, the bedding should be renewed. The treatment should be continued for a while after the clinical symptoms have disappeared. Antihistomonal drugs are banned from market.

Gastrointestinal Parasitism

Poultry could be infected with nematode and cestode parasites. Among them: round worms, **Heterakis gallinarum**, **Syngamus trachea**, **Ascarids** and **Capillarids** are economically important. **Raillietina** spp **Davainea proglottina** are important cestodes species of poultry. The worm **Syngamus trachea** inhabits the trachea and lungs of many domestic and various wild birds.

Clinical Symptoms

Reduced appetite, reduced growth rate, diarrhea, anemia, gasping, coughing (in case of **syngumus** infection), Loss of condition and anorexia.

Diagnosis

Based on clinical symptoms, post-mortem examination and identification of the parasite itself and eggs.

Differential diagnosis: necrotic enteritis, streptococcosis, enteroccosis, avian tuberculosis, rotavirus infection, coccidiosis, histomoniasis or mycotoxicosis.

Treatment and Prevention

Piperazine diHCL 582-970 mg per Kg body weight with drinking water or 195-390g per 100 liters of drinking water and deworm chicken at 4-6 weeks of age.

Plus

Phenothiazine (for cecal worms in chickens) 0.5 g/bird individual treatment or in dry or wet mash feed, once a month to control ascaris. **Or**

Phenothiazine 0.5 gm/head or combined with piperazine (0.5-0.51% & 0.11% in drinking water) as a 1-day treatment, removes both heterakids and ascarids. **Or**

Ivermectin 10mg, 0.25-0.32 mg per Kg body weight. C/I do not use in breeders in production or layers producing eggs for human consumption. **Or**

Coumaphos, 0.004% in feed for 10-14 days for replacements, or 0.003% in feed for 14 days for layers, is used against capillarids.

Prevention

Strict sanitation and disinfection measures of bedding as well as feeders and drinkers,
Control of secondary hosts such as beetles roaches, grasshoppers and earth worms,
Use of a deworming program, and Avoidance of multi age farming.

ECTOPARASITES OF POULTRY

Fowl Ticks

Argas persicus is the tick species particularly active in poultry houses during warm and dry weather. All stages may be found hiding in cracks and crevices during the day. Ticks cause anemia and are vectors of diseases such as spirochaetosis and aegyptianellosis.

Clinical Symptoms

Fowl ticks produce anemia (most important), weight loss, depression, toxemia and paralysis. Egg production decreases. Red spots can be seen on the skin where the ticks have fed. Birds may show some uneasiness when roosting because fowl ticks are nocturnal. Production may be severely depressed.

Treatment and Prevention

Coumaphos 20% E.C., at a concentrations of 250 ppm, to be sprayed on walls, ceilings, cracks and crevices.

Or

Permethrin 10% EC, spray, with 0.05-0.1% solution. The 0.1% solution is applied to ceilings, walls, and suspended objects using a pressurized or power sprayer; the 0.05% solution can be applied directly to the birds at the rate of 1 gallon per 75 adult chickens. The solution applied to the birds is effective for mites only.

Or

Carbaryl (Sevin) 80% WP, apply 0.5% solution. Apply to birds with a pressurized or power sprayer at the rate of 1 gallon per 75 adult hens. Do not repeat treatment before 4 weeks.

Acaricides are best applied using a high-pressure sprayer after poultry house has been thoroughly cleaned.

Lice

Heavy infestation of poultry and other avian species with **Mallophagia** lice. These insects are found in farms with poor hygiene conditions. They are common during cold seasons. Introduction into a flock occurs through infested equipment or by other poultry. Lice infestation results in reduced egg production (up 40%) and weight loss. The skin irritations are also sites for secondary bacterial infections.

Diagnosis

The eggs or adult lice observed on the skin or feathers.

Treatment and Prevention

Apply acaricides for treatment and control as for ticks. Treatment should be repeated after 10-14 days since eggs are not killed

Regular inspection for adult lice or egg cluster at the base of the feather on the abdomen or around the vent

Avoid contact with other poultry

Clean the premises and replace the litter, paying special attention to the nests and transport cages.

Chicken Mite

Dermanyssus gallinae (Red mite) infests chickens, turkeys, other birds in breeder and small farm flocks. They are rare in modern commercial cage-layer operations. Chicken mites are nocturnal feeders that hide during the day under manure, on roosts, and in cracks and crevices of the chicken house, where they deposit eggs. They are the most significant economically.

Clinical Symptoms and Lesions

This mite is hardly visible but the consequences of infestation are visible due to the change in bird behavior (nervousness, pecking, stress and aggression), decline in production (eggs, lower feed conversion ratio), signs of anemia observed in birds and bloodstains noted on the eggs. Anemia may be severe during a massive infestation and can cause death. Economic losses are associated with production losses and treatment costs.

Treatment and Prevention

Dichlorovos 23 EC, diluted 5oz/gallon, 1 gallon/100 birds or 1-2gallon/1000 sq. ft of litter.
Tetrachlorvinfos 50 WP, 6.5 gallon, 1 gallon/100 birds or 1-2gallon/1000 sq. ft of litter

Prevention

Mite are difficult to control because they can hide themselves, and are able to survive for a long period without feeding and can rapidly build up resistance against pesticide
Biosecurity measures are essential to prevent new infestations, especially during the downtime period by cleaning and disinfection with effective acaricides and biological control.
This parasite can also attack human causing pruritic dermatitis or an allergic reaction with eczema.
Accaricides for ticks can be used and miticide spray treatments must be applied with sufficient force to penetrate the feathers in the vent area.

Common Chigger

Trombicula alfreddugesi and other chigger species (harvest mites, red bugs) infest birds as well as human and other mammals.

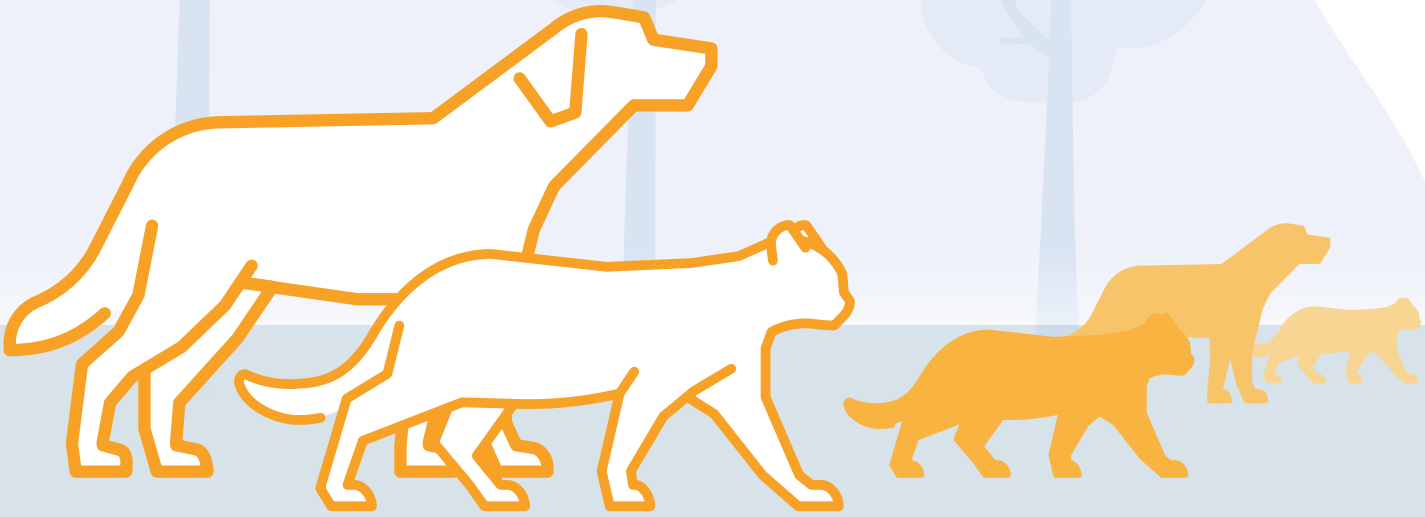
Clinical Symptoms and Lesions

Heavily parasitized birds become droopy, refuse to eat, and may die from starvation and exhaustion.
Larvae may be found either singly or in clusters on the ventral portion of the birds.

Treatment and Control

Use miticide sprays like chicken mite above.
Control on the range is aided by keeping the grass cut short and dusting with sulfur or Malathion.

SECTION VII



DISEASES OF DOGS & CATS[7]

7 *Guide*

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.

NON-INFECTIOUS DISEASES

Diabetes Mellitus

Diabetes mellitus is a chronic disorder of carbohydrate metabolism due to relative or absolute insulin deficiency. It is common in dogs and cats. Most cases of spontaneous diabetes occur in middle-aged dogs and older cats. Likely causes include infection with certain viruses, stress, obesity and administration of corticosteroids or progestogens may increase the severity of clinical symptoms.

Clinical Symptoms

The onset is often insidious and the clinical course is chronic. Common signs in dogs include polydipsia, polyuria and polyphagia with weight loss, bilateral cataracts and weakness. Diabetic animals have decreased resistance to bacterial and fungal infections and often develop chronic or recurrent infections, such as cystitis, prostatitis, bronchopneumonia and dermatitis. Cataracts develop frequently in dogs (not cats) and edema of the lens and disruption of normal light transmission.

Diagnosis

Diagnosis is based on persistent fasting hyperglycemia and glucosuria. Normal fasting blood glucose level for dogs and cats is 75-120mg/dl. A search should be made for drugs or diseases that predispose to diabetes.

Treatment and Prevention

In dogs the initial treatment is NPH Or lente insulin, 0.5 u/kg q 12 hr. As some dogs have poor glycemic control while on treatment it is preferable to do a blood glucose testing and determine the appropriate maintenance dose.

In cats 1-3 u, q 12 hr. of ultra-Lente insulin. **Or**

Glipizide initial dose 2.5 mg, q 12 h, PO, in conjunction with dietary management.

In complicated cases, Insulin 0.2 u/kg IM initial dose, followed by 0.1 u/kg, q 1 hr. once the serum glucose is <250 mg/dL, 0.25-0.5 u/kg SC, q 4 to 6 hr. Monitor the serum glucose at 1- to 2-hr intervals. If blood glucose levels fall rapidly, 2.5-5% dextrose IV may be required.

Note: Once the animal is on maintenance therapy and its condition is stable, it should be reassessed every 4-6 months.

Treatment involves a combination of weight reduction, diet (high fiber, high complex carbohydrate), and insulin injection or implant. Owner understanding and cooperation is vital to success of treatment. Intact females should be spayed.

Do not give Glipizide to dogs.

Epilepsy

Epilepsy is a sudden, involuntary change in behavior, muscle control, consciousness, and/or sensation occurs commonly in dogs. A seizure is often accompanied by an abnormal electrical discharge in the brain.

Clinical Symptoms

Stages of epileptic are:

The Prodrome – This stage can last from minutes to hours or even days before the manifestation of the actual seizure activity. This stage is typically characterized by changes in the dog's mood or behavior.

The Aura – Some dogs will begin pacing, licking, salivating, trembling, vomiting, wandering aimlessly, hiding, whining or urinating. Other dogs may exhibit stranger activities such as excessive barking and attempts to get an owner's attention.

The Ictus- It is a period of abnormal activity during which they lose consciousness, gnash their teeth or appear to be chewing gum, thrashing about with their head and legs, drooling excessively, crying, paddling their feet as if running as well as losing control of their bladders and bowels are observed. Others signs that may occur include, running in circles, chew gum, some suddenly go blank and stare

into space and then there are the ones that only have partial seizures in which the twitching is localized in one area. Such as in the face, leg, shoulder or over the hips.
Treatment and Prevention
Phenobarbital 65 mg/ml, starting dosage for dogs is 5 to 10 mg /Kg of body weight given twice daily. This can be increased up to 40 mg per kg of body weight per day based on the dog's response.
Potassium Bromide (in dogs with liver problem) 450 mg/kg dose is divided over 5 days (90 mg/kg/day) and added to a maintenance dose of 20 to 30 mg/kg (average of 25 mg/kg) per day. Thus, a new patient will receive 120 mg/kg of potassium bromide each day for 5 days and then back down to 25 mg/kg per day.
Note: If resistance to a drug occurs, change to another alternative drug or inject Diazepam IV.
It's important to continue giving the drug to your dog for the full duration of the prescription and not miss a dose as this can result in seizures. Overdose can result in nervous system depression.

Hip Dysplasia

Hip dysplasia is a multifactorial abnormal development of the coxofemoral joint in dogs that is characterized by joint laxity and subsequent degenerative joint disease. It is most common in large breeds. Excessive growth, exercise, nutrition and hereditary factors affect the occurrence of hip dysplasia. The pathophysiologic basis for hip dysplasia is a disparity between hip joint muscle mass and rapid bone development. As a result, coxofemoral joint laxity or instability develops and subsequently leads to degenerative joint changes, e.g. acetabular bone sclerosis, osteophytosis, thickened femoral neck, joint capsule fibrosis and subluxation or luxation of the femoral head.

Clinical Symptoms

Signs are variable and do not always correlate with radiographic abnormalities. Lameness may be mild, moderate or severe and is pronounced after exercise. A "bunny-hopping" gait is sometimes evident. Joint laxity (Ortolani sign), reduced range of motion, and crepitation and pain during full extension and flexion may be present.

Diagnosis

Physical inspection and radiography are useful in delineating the degree of arthritis and planning of medical and surgical treatments.

Treatment and Prevention

Aspirin 10-25mg/kg PO ,q 12 hr.,
Carprofen 2.2-4mg/kg PO ,q 12 hr.,
Meloxicam 0.2mg/kg PO initially and followed by 0.1mg/kg q 24 hr. (and other pain-relieving drugs) reduce pain and inflammation. Caution is advised with long-term NSAID usage in dogs.
Mild cases or nonsurgical candidates (because of health or owner constraints) may benefit from weight reduction, restriction of exercise on hard surfaces, controlled physical therapy to strengthen and maintain muscle tone.

Foreign Bodies in the Esophagus

Esophageal foreign bodies are common in dogs than cats. Bones are the most common foreign bodies, but needles, fishhooks, wood, metal objects, etc., may be found. Usually, the object lodges at the thoracic inlet, base of the heart, or in the caudal esophagus just before the diaphragm; occasionally, it may lodge at the upper esophageal sphincter.

Clinical Symptoms

Salivation, gagging, dysphagia and regurgitation are signs of esophageal foreign bodies. Complete obstruction causes regurgitation after food or water intake. In chronic obstruction, anorexia and weight loss may be predominant signs. Perforation of the thoracic oesophagus may result in pleuritis, mediastinitis, pyothorax, and pneumothorax or bronchoesophageal fistula. Aspiration pneumonia can be a serious complication of regurgitation.

Diagnosis

Esophagoscopy or contrast esophagogram, aqueous iodinated contrast medium and external palpation.

Treatment and Prevention

Tetracycline 10-20 mg/kg, q 8h to q 6 hr. PO for dogs and cats for 3 days. **Or**

Procaine Penicillin + Dihydrostreptomycin Sulfate 200,000IU: 200mg/ml, 1-2ml/20kg, IM, q 24 hr. for 3-5days.

Plus

Methylprednisolone 1 mg/kg, IM or SC, q 24 h for 3 days.

The object should be removed per os either with a flexible endoscope and forceps or with a rigid scope and alligator forceps. If it cannot be removed PO, it may be pushed into the stomach where it can be digested (e.g. smooth bones), passed or removed via a gastrostomy.

In severe cases surgery may be performed; however, the prognosis is poor.

Esophagitis should be managed medically after the foreign body is removed.

Fractures

A fracture is a complete or incomplete break in the continuity of bone or cartilage. A fracture is accompanied by various degrees of injury to surrounding soft tissues, including blood supply along with compromised function of locomotor system. The patient's local and overall conditions must be taken into consideration during evaluation. At least 75% to 80% of all fractures are caused by car accidents or motorized vehicles. Fractures are classified based on many factors including the cause, presence or absence of a communicating external wound; location, morphology and severity of the fracture and stability of the fracture after axial reduction of fragments. The type of fracture and forces acting on it are important determining factors for selection of appropriate management approach. Therefore a complete understanding of the five forces acting on bone fracture: bending, compression, shear, tension and torsion is very important for good fracture repair.

Symptoms

The visible signs associated with fracture including pain or localized tenderness, deformity, abnormal mobility, local swelling, loss of function, crepitus may be observed or detected.

Diagnosis

The history and clinical symptoms usually indicate the presence of a fracture. However, radiographs are essential for precise determination of its nature. Radiographs of at least two views at right angles to each other are essential for accurate diagnosis and selection of the best procedures for reduction and immobilization.

Treatment and Prevention

Non drug treatment

Reduction (replacing the fractured segments in their original anatomical position) and fixation (immobilizing the fractured bone fragments)

External fixation using splint, casts; best only for simple, transverse fractures of bones below stifle or elbow such as radius/ulna or tibia in younger animal, but not good for oblique or highly comminuted fractures

Internal fixation using bone plates, interlocking nail, intramedullary pinning together with screws and circlage wires. The best internal fixation method is the one that neutralizes all forces acting on the fracture. Selection of the method of internal fixation depends on the fracture location, type.

Drug treatment

Premedications

Midazolam (0.2 mg/kg IV, IM), **or** Diazepam (0.2 mg/kg IV) **plus**

Hydromorphone 0.05 – 0.2 mg/kg SC or Morphine (0.1-0.2 mg/kg IV or 0.2-0.4 IM)

Maintenance

Propofol (4-6 mg/kg IV) or (see Annex for use of protocols with other available drugs)
Cefazolin 22mg/kg every 90 minutes, first dose given while being clipped; 1 gram vials – mix with 9.6mL sterile water to make a 100mg/mL solution – date bottle and refrigerate after mixing, good for 3 days
Cephalosporin - 20 mg/kg IV q 2 hours until the completion of the operation
Animals with fracture caused by car accident will also have concurrent injuries of core body systems that requires timely therapy to eliminate or stabilize life-threatening injuries.
If the animal is in shock, administer crystalloid (Lactated Ringer's solution) I/V
Stop bleeding, if the fracture is open, clean, debride and put the animal on broad-spectrum antibiotic immediately.
Provide immediate relief for pain and discomfort
See lists of analgesic in the annex (it is good to combine opioid with NSAID for synergistic effect).

Motion Sickness

Motion sickness is characterized by nausea, excessive salivation and vomiting. It is caused by travel by plane or by car though fear from vehicle in its stationary state may also induce motion sickness.

Clinical Symptoms

Animals may yawn, whine and show signs of uneasiness or apprehension, diarrhea in severe cases. Motion sickness is usually seen during travel.

Treatment and Prevention

Promazine hydrochloride, Injection powder, 2.2-4.4 mg/kg, IV or IM.
Chlorpromazine hydrochloride injection or tablet, 3.3 mg/kg, q 6-24 hr. PO, IV, or IM.
Acepromazine maleate injection or tablet, 0.025 - 0.2mg/kg, I/V, I/M or S/C for canine and feline with a maximum dose not exceeding 3mg in dogs and 1mg in cats, 1-3 mg/kg, PO, for canine and 1.1-2.2 mg/kg, PO for feline.
Dimenhydrinate, 4-8mg/kg, PO. And in cats 12.5mg (total dose), PO.
For prevention: Maropitant Citrate, tablet can be used.
Conditioning of the animal before travel or use of Phenobarbital and diazepam to produce a general sedative effect. Oral administration of one of these drugs several hours before departure should reduce or eliminate the signs of motion sickness.

Tick Paralysis

Tick paralysis is a toxin-induced, febrile, ascending, symmetrical condition in which there is flaccid tetraplegia and functional impediment to the reflexes of the superficial and deep tendons of the limbs and abdomen. Dogs are affected most commonly, but losses can also occur in cats. In Rhipicephalus, **Haemaphysalis, Otobius**, and **Argas** have been associated with paralysis in dogs and cats.

Clinical Symptoms

Early signs include change or loss of voice, lack of coordination of the hind legs, change in breathing rate and effort, gagging or coughing, vomiting and dilated pupils. Signs occur 3-9 days after the tick attaches, depending on the type of tick involved. There are also nystagmus and difficulties in breathing, chewing and swallowing. Death can occur within several hours from respiratory paralysis. Temperature remains normal; blood and humoral values are unchanged.

Diagnosis

This is based on the presence of ticks, sudden appearance of paralysis, rapid course and quick clinical recovery after tick removal. Normal body temperature, blood and fluid values are observed. No specific laboratory diagnostic techniques are available. Other diseases and disorders (botulism, poisoning) have the same signs as tick paralysis, but in areas where ticks are prevalent, tick paralysis is a strong possibility.

Treatment and Prevention

Drug treatment

Diazinon as collar 15%

Amitraz to be diluted as per the label on its bottle

Ivermectin 400-600mg/kg, start with 100mcg/kg and increase by 100 every day until the maximum dose is reached.

(S)-Methoprene + Fipronil + Amitraz, Dog, 6.7 mg/kg b.w. for Fipronil, 6 mg/kg for (S)-Methoprene and 8 mg/kg for Amitraz. At monthly intervals throughout the tick and/or flea seasons.

Non drug treatment

Tick (s) removal is necessary and animals usually improve within the first 24 hours

Rug treatment and application of acaricides to kill the ticks.

Vomiting

Vomiting is the forceful ejection of the contents of the stomach and proximal small intestine. Vomiting can be due to primary GI disease, renal or hepatic failure, electrolyte abnormalities (e.g. hypoadrenocorticism), pancreatitis or CNS disorders (including toxin ingestion).

Clinical Symptoms

Anxiety, depression, hypersalivation and repeated swallowing accompanied by relaxation of the gastroesophageal sphincter are followed by retching. Forceful contractions of the abdominal muscles and diaphragm against a closed glottis and increases in intra-abdominal pressure force expulsion of food and fluid.

Diagnosis

Start with differentiating vomiting from regurgitation or dysphagia. Define the vomiting as acute or chronic, persistent or intermittent, static or progressive or recurrent.

When vomiting has been of a short duration, i.e. <3-4 days, it is limited to a detailed history, a physical examination, examination of the oropharynx and a rectal examination. Chronic vomiting, in addition to a detailed history and physical examination an initial database should include a complete blood count, biochemical profile (including serum electrolytes), urinalysis and abdominal radiographs (and abdominal ultrasound if available).

Treatment and Prevention

Lactated Ringer's solution (hypovolemic patients) for acute vomiting

Diphenhydramine 2-4 mg/kg, q 8-12 hr. PO for chronic vomiting. This must not be used in pregnant or nursing animals. **Or**

Prochlorperazine, 0.3 mg/kg, q 8 h, PO; 0.1 mg/kg, q 6 hr., I/M; Or 0.1-0.5 mg/kg, q 8 hr., S/C. **Or**

Chlorpromazine, 0.5 mg/kg, q 6 h, PO; 0.5 mg/kg, q 8 hr., IM; or 1 mg/kg, q 8 hr., rectally. **Or**

Metoclopramide 0.2-0.5 mg/kg, q 6 hr., PO or SC, or 1-2 mg/kg/day, slow IV or 1.3 µg/kg/min. **Or**

Cisapride 0.1-0.5 mg/kg, q 8 h, PO facilitates gastric emptying in dogs and cats and may be useful in the management of vomiting associated with delayed gastric emptying.

Maropitant Citrate, Dogs 2-4 Months of Age: 1 mg/kg equal to 0.1mL/kg BW,SC, once daily for up to 5 consecutive days. Dogs 4 months of Age and Older: 1 mg/kg equal to 0.1 mL/1 kg BW, IV over 1-2 minutes or SC at once daily for up to 5 consecutive days.

For Treatment of Vomiting in Cats 4 Months of Age and Older:1 mg/kg equal to 0.1 mL/kg BW, IV over 1-2 minutes or SC once daily for up to 5 consecutive days.

For Prevention of Vomiting in Dogs 4 months of Age and Older Caused by Emetogenic Medications or Chemotherapeutic Agents: 1 mg/kg BW IV over 1-2 minutes or SC one time, 45-60 minutes prior to use of emetogenic medications or chemotherapeutic agents.

The primary goal of treatment in a vomiting patient is to identify and treat the underlying cause but symptomatic treatment should be done as follows.

Fasting and withholding water can be done for 24 hours to rest the GI tract and water can be provided in the form of ice.

Systemic Anaphylaxis

Anaphylaxis occurs in sensitized animals after parenteral injection of vaccines or drugs, ingestion of foods or insect bites. Clinical symptoms occur within seconds to minutes after exposure to the allergen. This latent period is the time required for the allergen to bind to sensitized mast cells and for vasoactive mediators to be released. In cats, lungs are the primary target organ and the portal-mesenteric vasculature is secondary; this is reversed in dogs.

Clinical Symptoms

Mast cell degranulation in the portosystemic vasculature causes venous dilatation and pooling of blood in the intestines and liver, with resultant shock, agitation, colic, nausea, vomiting and diarrhea, hypersalivation, dyspnea, cyanosis and in severe cases death.

Treatment and Prevention

Adrenaline 0.05-0.5 mg, IV, SC or IM;
Dopamine 2-5mcg/kg/min, IV;
Diphenhydramine, 2-4 mg/kg, q 8 hr., PO;
Prochlorperazine, 0.1 mg/kg, q 6 hr., IM; or 0.1-0.5 mg/kg, q 8 hr., SC.
Intubation is important to maintain air way. Perform cardiorespiratory resuscitation, while monitoring vital signs.

VIRAL DISEASES OF DOGS AND CATS

Canine Distemper

Canine distemper is a highly contagious, systemic, viral disease of dogs prevalent in Ethiopia. Clinically canine distemper is characterized by a diphasic fever, leukopenia, GI and respiratory catarrh and frequently pneumonic and neurologic complications. The disease occurs in **Canidae** (dogs, foxes and wolves). Infection occurs via aerosol. Some infected dogs may shed virus for several months.

Clinical Symptoms

Transient fever, 3-6 days after infection and leukopenia (especially lymphopenia). The fever subsides several days and second fever accompanied by serous nasal discharge, mucopurulent ocular discharge and anorexia. GI and respiratory signs may follow, an acute encephalomyelitis with or without systemic disease and hyperkeratosis of the footpads ("hardpad" disease) and epithelium.

Neurologic signs include: localized involuntary twitching of a muscle or group of muscles, such as in the leg or facial muscles. Paresis or paralysis, often beginning in the hind limbs evident as ataxia, followed by ascending paresis and paralysis and convulsions characterized by salivation and chewing movements of the jaw (petit mal, "chewing-gum fits"). In Chronic distemper encephalitis (old dog encephalitis) ataxia, compulsive movements such as head pressing or continual pacing and uncoordinated hypermetria may occur.

Diagnosis

Clinical symptoms (febrile catarrhal illness with neurologic sequel) plus serologic demonstration of virus-specific IgM or an increased ratio of CSF to serum virus-specific IgG.

Treatment and Prevention

Procaine Penicillin + Dihydrostreptomycin Sulfate 200,000IU: 200mg/ml, 1-2ml/20kg, IM, q 24 h for 3-5days. Or
Sulfadiazine-Trimethoprim, 25:5 mg/ml, 1ml/10-20mg/kg, IM, q 24 h, for 3-5 days. **Or**
Oxytetracycline 10 mg/kg, IM, PO, q 24 h for 3-5 days; oral treatment is preferred;
Plus
Lactated Ringer's solution 40-50 ml/kg q 24 h SC, IV, IP.
Antipyretics e.g. Acepromazine maleate 0.05-0.2 mg/kg, PO, SC, IM or IV q 12 h for three days. **Or**
Metoclopramide, 0.2-0.5 mg/kg, q 6 h., PO or SC or 1-2 mg/kg/day, slow IV
Phenylbutazone 10-22 mg/kg PO, q 8 h for dogs and 6-8 mg/kg PO, IM q 1 2 h for cats every other week.

Prevention

Successful immunization of pups with canine distemper Modified Live Virus (MLV) depends on the absence of interference by maternal antibody. To overcome this barrier start vaccinations at 6 weeks old and repeat at 3 to 4 weeks interval until 16 weeks old

Cleaning of the nasal cavities; good nursing and dietary supplements are essential.

Note: Treatments are directed at limiting secondary bacterial invasion and supporting the fluid balance that include electrolyte solutions, antipyretics and anti convulsants.

Canine Parvovirus

Canine parvo virus is a highly contagious and a relatively common cause of acute, infectious GI illness in young dogs. Due to its resistance to many common detergents and disinfectants, as well as changes in temperature and pH the virus can persist for many months and possibly years in the environment.

Clinical Symptoms

Two clinical forms – myocarditis seen on pups especially early neonatal period and gastroenteritis on pups 6-20 weeks. Initial Clinical symptoms may be nonspecific (e.g. lethargy, anorexia fever) with progression to vomiting and bloody diarrhea, myocarditis, presenting as acute cardiopulmonary failure or delayed progressive cardiac failure can be seen with or without signs of enteritis. Physical examination findings can include depression, fever, dehydration and abdominal pain.

Diagnosis

Clinical symptoms are indicative and confirmed by ELISA test or hemagglutination test from fecal samples.

Treatment and Prevention

Procaine Penicillin and Dihydrostreptomycin Sulfate. **Or**

Sulfadiazine-Trimethoprim. **Or**

Oxytetracycline. **Or**

Ampicillin 10-25 mg/kg, q 8 h, PO, or 5-10mg/kg, IV, q 8 hr. for a maximum of 5 days. **Or**

Gentamicin 4-6 mg/kg, SC or IM, q 24 hr. for a maximum of 5 days.

Plus

Most dogs benefit from IV fluid therapy with a balanced electrolyte solutions.

Such as Lactated Ringer's and 5% dextrose with additional potassium chloride 10-20mEq/L for severe cases. **Plus**

Metoclopramide 0.2-0.5 mg/kg, q 6 hr. PO or SC or 1-2 mg/kg/day, slow IV

Food and water should be withheld until vomiting has subsided

Prevention

Vaccinate at 5-8 weeks age, and 16-20 weeks of age and annual vaccination thereafter is recommended.

The main goal of treatment includes restoration of fluids, electrolytes and metabolic abnormalities and prevention of secondary bacterial infections

In the absence of significant vomiting oral electrolyte solution can be offered but if vomiting is severe withhold oral food and solutions.

Feline Infectious Anemia

Feline infectious anemia is caused by **Haemobartonella felis**, belonging to the family **Anaplasmataceae**. The method of transmission is uncertain. Recovered animals may remain asymptomatic carriers.

Clinical Symptoms
Peracute form: anemia, immune suppression with high parasitaemia.
Acute form: fever, anemia, depression, weakness and occasionally jaundice.
Chronic form: anemia, lethargy and marked weight loss.

Diagnosis
The parasite may be demonstrated on the surface of erythrocytes in Giemsa stained blood smear. Reduced PCV.

Treatment and Control
Doxycycline hydrochloride, 5 mg/kg loading dose PO; then 2.5 mg/kg, then 2.5 mg/kg q 24 h for both dogs and cats acute cases.
Blood transfusion as a supportive treatment and flea control are important.

Feline Panleukopenia

It is highly contagious disease of cats caused by **Feline Panleukopenia** virus. It affects bone marrow cells, lymphoid tissue cells and intestinal crypt cells. The virus is shed through vomits and feces. Infected cats can shed the virus for one year after recovery.

Clinical Symptoms
It may be acute or peracute in its clinical course. Anorexia and lethargy followed by vomiting and yellow and blood tinged diarrhea. Infected cat will have swollen gastrointestinal system.

Diagnosis
Clinical symptoms are important. White blood cell counts are low.

Treatment and Prevention
Penicillin and streptomycin 200,000 IU/250mg, 1-2ml/20kg, IM, q 24 hr. for 3-5days. **Or**
Sulfamethazine-Trimethoprim 200 mg/ 40mg, 5- 30 mg/kg, IM, q 24 hr. or 1ml/10-20kg, IM, q 24 hr. for 3-5 days.
Antiemetics:
Acetylpromazine Maleate 0.05-0.2 mg/kg PO, S/C, I/M or I/V q 12 h for 3 days. **Or**
Metoclopramide, 0.2-0.5 mg/kg, q 6 h., PO or SC, or 1-2 mg/kg/day, slow IV.

Feline Infectious Peritonitis

Feline Infectious Peritonitis is a viral disease caused by some strains of corona virus. It is transmitted by inhalation or ingestion. Infected cats may remain carriers.

Clinical symptoms
Early signs include anorexia, weight loss, listlessness and dehydration. Pleural exudates, dyspnea and death may occur within 6 weeks.

Diagnosis
Histological examination of affected tissue is the only definitive diagnostic method. Pleural or peritoneal fluid, which may contain fibrin strands, clots on standing, neutrophilia, lymphopenia and in chronic cases, anemia; serum hyperproteinaemia are indicators.

Treatment and Prevention

There is no specific treatment of FIP.

Broad spectrum antibiotics (for animals in good condition); for choice of drugs, **see Antimicrobials in** Annex 1.

When breeding, animals should be selected from breed with known FIP negative bloodlines of cats.

Feline Respiratory Disease Complex

Feline respiratory disease complex is characterized by rhinitis, conjunctivitis, lacrimation, salivation and oral ulcerations. The principal diseases, Feline Viral Rhinotracheitis (FVR) and Feline Calicivirus Infections (FCV), affect exotic as well as domestic species. **Chlamydia psittaci** and mycoplasmal infections appear to be of lesser importance. Transmission occurs via aerosol droplets and fomites; convalescent cats may continue to harbor virus for many months. Calicivirus is shed continuously, while infectious FVR virus is released intermittently. Stress may precipitate a secondary course of illness.

Clinical Symptoms

Feline Rhinotracheitis: fever (40.5°C), frequent sneezing, conjunctivitis, rhinitis, serous to mucopurulent nasal discharge and often salivation which may be induced by excitement or movement. Severely debilitated cats may develop ulcerative stomatitis and ulcerative keratitis. Signs may persist for 5-10 days in milder cases and up to 6 weeks in severe cases.

Calicivirus: An acute febrile response, inappetence and depression are common signs. Serous rhinitis and conjunctivitis can also occur.

Chlamydia psittaci infections characteristically produce conjunctivitis and cats sneeze occasionally. Fever, serous lacrimal discharge to mucopurulent conjunctivitis, lymphoid infiltration and epithelial hyperplasia may occur. Convalescent cats may undergo relapses.

Diagnosis

Typical signs and cytologic examination of Giemsa-stained conjunctival scrapings give presumptive diagnosis. A definitive diagnosis is based on isolation and identification of the agent.

Treatment and Prevention

Tetracycline 1% eye drops, 5 to 6 times daily against **C. psittaci**.

Plus

Nebulization or saline nose drops to remove tenacious secretions.

Plus

Vasoconstrictor (e.g., two drops of ephedrine sulfate [0.25% solution] in each nostril, q 12 hr.

Plus

Lysine 250 mg, PO, q 12 hr. to q 8 hr. interferes with herpetic viral replication and may help reduce the severity of FVR infection

Plus

Oxygen supply (if dyspnea is severe). Fluids may be indicated to correct dehydration, and force-feeding may be necessary. Esophagostomy may be indicated in severe cases

Plus

Antihistamines e.g. chlorpheniramine maleate, 8 mg for adults, 4 mg for kittens, PO, q 12 hr. early in the course of the disease may be used.

Infectious Canine Hepatitis

Infectious Canine Hepatitis (ICH) is contagious disease of dogs (also foxes) caused by canine **Adenovirus**. Ingestion of urine, feces or saliva of infected dogs is the main route of infection. Recovered dogs shed virus in their urine for ≥6 months. The mortality rate is highest in very young dogs.

Clinical Symptoms

The incubation period is 4-9 days. Biphasic fever of 40°C which lasts 1-6 days, leukopenia one day after fever, acute illness develops, tachycardia, leukopenia persists throughout the febrile period. Apathy, anorexia, thirst, conjunctivitis, serous discharge from the eyes and nose and occasionally abdominal pain and vomiting are common. Intense hyperemia or petechiae of the oral mucosa, as well as enlarged tonsils, may be seen. There may be subcutaneous edema of the head, neck and trunk. Simultaneous infection with CAV-1 and distemper virus is sometimes seen.

Diagnosis

Usually, the abrupt onset of illness and bleeding suggest ICH. The gross changes in the liver and gallbladder are more conclusive. Diagnosis is confirmed by virus isolation, immunofluorescence or characteristic intranuclear inclusion bodies in the liver.

Treatment and Prevention

Dextrose 5% in isotonic saline should be given, preferably IV, **Or**
Iron sulfate or gluconate, 100-300mg/kg, PO.
Plus
Sulfamethazine-Trimethoprim, 200mg/ 40mg, 5- 30 mg/kg, IM, q 24 h, or 1ml/10-20kg, IM, q 24 h for 3-5days.
Plus
Atropine ophthalmic ointment one drop in affected eye q 24 h to q 12 h;
Blood transfusion in severely infected dogs
Vaccination with modified live virus vaccine after 9-12weeks of age
Treatment is symptomatic and supportive and to prevent secondary bacterial infections with broad spectrum antibiotics, IV electrolyte solutions supplemented with 5% Dextrose.

Infectious Tracheobronchitis

Infectious tracheobronchitis (Kernel cough) is a self-limiting mild disease of dogs that results from inflammation of the upper airways. The disease may progress to fatal bronchopneumonia in puppies or to chronic bronchitis in debilitated adult or aged dogs. The illness spreads rapidly among susceptible dogs housed in close confinement.
Infections involved include **Canine parainfluenza** virus, **canine adenovirus** 2 (CAV-2), or **canine distemper virus**, can be the primary or sole pathogen involved; **Bordetella bronchiseptica** especially in dogs <6 months old; other bacteria such as **Pseudomonas** spp, **E. coli**, and **Klebsiella pneumoniae** may also cause secondary infections after viral injury to the respiratory tract. Stress and extremes of ventilation, temperature and humidity apparently increase susceptibility to and severity of the disease.

Clinical Symptoms

Paroxysms of a harsh dry and easily induced cough, followed by retching and gagging to more severe signs such as fever, purulent nasal discharge, depression, anorexia and a productive cough, especially in puppies, indicates a complicating systemic infection such as distemper or bronchopneumonia. Disease relapses after stress, particularly due to adverse environmental conditions and improper nutrition. Severity diminishes during the first 5 days, but the disease persists for 10-20 days.

Diagnosis

History and clinical symptoms are indicators.

Treatment and Prevention

Codeine phosphate 0.25 mg/kg, q 6-12 hr. PO to control persistent nonproductive coughing.
In severe chronic cases:
Tetracycline 10-22 mg/kg, PO, q 8 h for 5 days in severe chronic cases. **Or**
Sulfamethazine -Trimethoprim (200:40mg) 5- 30 mg/kg, IM, q 24 hr. for 3-5days. **Or**
In more severe cases:
Gentamicin sulfate (50 mg) diluted in 3 mL of saline may be administered by aerosolization q 12 hr. for 3 days in more severe cases.
Plus
Phenylbutazone 20gm in 100ml, 1ml/15kg IM, q 24 hr. for 1-4 days
Immunize with modified live virus vaccines against distemper, parainfluenza, and CAV-2, which also provides protection against CAV-1
Good nutrition, hygiene, and nursing care and correct predisposing factors.

Rabies

Rabies is an acute viral encephalomyelitis caused by **Rhabdoviridae** that principally affects carnivores and insectivorous bats, although it can affect any mammal. It is almost invariably fatal once clinical symptoms appear. Rabies is endemic to Ethiopia. No cat-to-cat transmission of rabies has been recorded. Transmission is almost always by introduction of virus-laden saliva into the tissues, usually by the bite of a rabid animal. Contact with fresh wound or even intact mucous membrane may also transmit the disease.

Clinical Symptoms

Clinical symptoms of rabies are rarely definitive. The most reliable signs are behavioral changes and unexplained paralysis. Behavioral changes may include anorexia, signs of apprehension or nervousness, irritability and hyper excitability. The animal may seek solitude, ataxia, altered phonation and changes in temperament. A normally docile animal may suddenly become vicious and vice versa.
Prodromal Form: Lasts 1-3 days; animals show only vague CNS signs, which intensify rapidly.
Furious Form: This is the classical "mad-dog syndrome," the animal becomes irrational and with the slightest provocation, may viciously and aggressively use its teeth and claws. The posture and expression is one of alertness and anxiety with pupils dilated. Noise invites attack. Such animals lose all caution and fear of natural enemies. Carnivores with this form of rabies frequently roam extensively, attacking other animals and people. Rabid dogs chew wires and frame of their cages, breaking their teeth and will follow a hand moved in front of the cage, attempting to bite. Puppies usually become vicious in a few hours. Rabid domestic cats and bobcats attack suddenly, biting and scratching viciously. As the disease progresses, muscular incoordination and seizures are common.
Paralytic Form: Paralysis of the throat and masseter muscles, often with profuse salivation and inability to swallow. Dropping of the lower jaw is common in dogs. These animals are not vicious and rarely attempt to bite. The paralysis progresses rapidly to all parts of the body, coma and death follow in a few hours.

Diagnosis

Care must be taken as early stages of rabies can be easily confused with other diseases or normal aggressive tendencies.
A rabies diagnosis must be verified with laboratory tests. Immunofluorescence microscopy on fresh brain tissue culture techniques using mouse neuroblastoma cells or both are confirmatory tests.

Treatment and Control

No treatment for rabies.

Prevention

Notification of suspected cases and euthanasia of dogs with clinical symptoms and those bitten by a suspected rabid animal

Reduction of contact between susceptible dogs by leash laws, dog movement control, and quarantine

Mass immunization of dogs (and cats) by campaigns using modified live virus and inactivated types with continued boosters

Stray dog control and euthanasia of unvaccinated dogs that roam freely

Dog registration and continuous vaccination of dogs and cats.

Management of Suspected Rabies Cases and Exposed Pets

Any animal bitten or scratched by a wild, carnivorous mammal not available for testing should be regarded as having been exposed to rabies. Any unvaccinated dog or cat exposed to rabies be humanely destroyed immediately. If the owner is unwilling to do this, the animal should be placed in strict isolation for 6 months and vaccinated against rabies 1 month before release. If an exposed animal is currently vaccinated, it should be **revaccinated immediately** and closely observed for 45 days.

Exposure of Man and Human Immunization:

Any wild carnivore suspected of exposing a person to rabies should be considered rabid unless proved otherwise by laboratory testing. This also applies to "pet" wildlife. Any healthy dog or cat, whether vaccinated against rabies or not, that exposes (bites or deposits saliva in a fresh wound or on a mucous membrane) a person should be confined for 10 days; if the animal develops any signs of rabies during that period, it should be humanely destroyed and its brain promptly submitted for rabies diagnosis. If the dog or cat responsible for the exposure is stray or unwanted, it should be euthanized as soon as possible and submitted for rabies diagnosis. Pre-exposure immunization is strongly recommended for all people in high-risk groups.

Comprehensive guidelines for control in dogs have been prepared by the World Health Organization (WHO) and include the following:

- Notification of suspected cases and destruction of dogs with clinical signs and dogs bitten by a suspected rabid animal;
- Reduction of contact rates between susceptible dogs by leash laws, dog movement control, and quarantine;
- Mass immunization of dogs by campaigns and by continuing vaccination of young dogs;
- Stray dog control and destruction of unvaccinated dogs with low levels of dependency on or restriction by man; and Dog registration;
- Vaccination of dogs and cats with modified live virus and inactivated types

BACTERIAL DISEASES OF DOGS AND CATS

Brucellosis

Brucellosis in dogs is caused by **Brucella canis**.

Clinical symptoms
Abortions, decreased fertility, reduced litter sizes and neonatal mortality. In male dogs: orchitis and epididymitis.

Diagnosis
Tube agglutination test, ELISA and agar gel immunodiffusion test.
Treatment and Prevention
Treat animals that are not intended for breeding and during early course of infection. For treatment use broad spectrum antibiotics
No vaccine is available; serological detection and removal of infected animals is the only method of control.

Canine Monocytic Ehrlichiois

Ehrlichiois is a generalized disease of **Canidae** caused by the rickettsiae **Ehrlichia canis**. It is transmitted by ticks especially **Rhipicephalus sanguineus**.

Clinical Symptoms
The disease may progress through an acute, subclinical to chronic phases. The acute phase is characterized by fever, thrombocytopenia, leucopenia and anemia. Persistent bone marrow depression, along with hemorrhages, neurological disturbance, peripheral edema and emaciation are characteristic. The disease may progress to chronic phase.

Diagnosis
Clinical and hematological features; detection of **E. canis** in mononuclear cells in Giemsa stained smears of the buffy coat; indirect fluorescence and transmission to other dogs.

Treatment and Prevention
Doxycycline or any Tetracycline 2-10 mg/kg, I/M, PO, q 24 h for ten days. **Or**
Tetracyclines can be administered to susceptible dogs before they enter an endemic area as a prophylactic measure.

Campylobacteriosis

Gastrointestinal campylobacteriosis caused by **Campylobacterjejuni** or **C. coli**, is a common cause of diarrhea in dogs and cats. Transmission occurs via fecal-oral route in food or water. Uncooked or undercooked poultry, other raw meat products and asymptomatic carriers are sources of infection.

Clinical Symptoms
Diarrhea is common and most severe in kitten and puppies, which is mucus-laden, watery, and/or bile-streaked diarrhea or with blood that lasts 3-7 days; reduced appetite; and occasional vomiting. Fever and leukocytosis may also be present. In certain cases, intermittent diarrhea may persist for >2 weeks. Diarrhea with mucus and blood has also been seen in cats.

Diagnosis
Culture of feces and dark field or phase-contrast microscopy of fresh fecal samples are definitive.

Treatment and Prevention
Erythromycin 10-20 mg/kg, q 12 hr. PO for dogs cats for 3-5 d. **Or**
Gentamicin 4-6 mg/kg, q 12 h, IM, SC, IV for dogs and cats for 3-5 days. **Or**
Doxycycline hydrochloride, 5-10 mg/kg loading dose PO; then 2.5 mg/kg, then 2.5 mg/kg q 24 h for both dogs and cats.

Leptospirosis

Dogs are the primary host of **leptospira serovar canicola.** The most common serovars infecting dogs are **Leptospira canicola** and **L. icterohaemorrhagia.** New serovars isolated are **Pomona** and **grippotyphosa.** Dogs of all ages and both sexes are affected. German shepherd is more sensitive. The incubation period is 4-12 days. Acute renal failure occurs in 80-90% of clinically infected dogs.

Clinical Symptoms
Early findings are non-specific and include fever, depression, anorexia and generalized pain, vasculitis, thrombocytopenia and a coagulopathy may develop followed by uremia, dehydration, vomiting and oral ulceration. Icterus often reflects the severity of the disease, meningitis, uveitis and abortion have been reported in rare circumstances. Hematologic and serologic abnormalities include leukocytosis, lymphopenia, monocytosis and thrombocytopenia and azotemia and electrolyte disturbances secondary to the renal failure, including hyponatremia, hypochloremia and hyperphosphatemia. Muscle pain, stiffness weakness, trembling or reluctance to move may be observed.

Diagnosis
Serology is most useful and frequently used. Dark field microscopy, fluorescent antibody, PCR and culture. Demonstration of leptospira in tissues with silver staining or fluorescent antibody test.

Treatment and Prevention
Doxycycline 5mg/kg/day, IV or PO q 24 hr. for 2 weeks to eliminate renal carrier phase. For dogs that can't tolerate doxycycline, initial therapy with a penicillin is appropriate but this should be followed by a 2 week course of doxycycline to eliminate the renal carrier phase of infection. **Or**
Ampicillin 5-10mg/kg IV, IM, SC q 12 hr. or 10-25mg/kg PO, q 12 hr. **Or**
Sodium Penicillin G 10,000-20,000mg/kg q 6 hr. **Or**
Enrofloxacin 5 mg/kg, PO, IM, SC q 24 hr. for 3-5days.

Prevention
Recently infected dogs can be treated with Amoxicillin or Doxycycline with dose as above for 7-10days
Currently available vaccines provide good protection
Rodent control
Public health significance: Leptospira are pathogenic to humans and could be transmitted from dogs.

Listeriosis

Listeriosis is a sporadic bacterial infection that affects a wide range of animals, including man and birds. It is caused by **Listeria monocytogenes.** The organism is transmitted between host species and becomes a source of infection to dogs and cats.

Clinical Symptoms
Septicemic or visceral listeriosis is most common in dogs and cats. The septicemic form affects organs other than the brain, the principal lesion being focal hepatic necrosis.

Treatment and Prevention
Penicillin and streptomycin 200,000IU/250mg, IM, q 24 h for 3-5 days. **Or**
Oxytetracycline 10mg/kg, q 24 h, IM for 5 days. **Or**
Sulfamethazine-Trimethoprim (200 mg:40mg) 5- 30 mg/kg, IM, q 24 h, 0.1ml/10-20kg, IM, q 24 h for 3-5days.
Note: Listeriosis is zoonotic; thus handle suspected material with caution.

Colitis

Colitis is an inflammation of the colon, may be acute or chronic. In most cases, the inciting factor (s) is/are unknown. Bacterial (e.g. **Salmonella** spp, **Clostridium** spp and **Campylobacter** spp), parasitic, fungal, traumatic, uremic and allergic causes have been postulated. Inflammation may be a result of a defect in mucosal immunoregulation.

Clinical Symptoms
Tenesmus and frequent passage of mucus-laden feces, sometimes with frank blood on its surface; the feces is often of a small volume and of a more liquid consistency.

Diagnosis
A complete history, rectal palpation and evaluation of feces are suggestive. Fecal smears for giardia and fungal elements, fecal flotation for parasite identification and culture for bacteria is suggested in cases of chronic colitis.

Treatment and Prevention
Sulfamethazine-Trimethoprim 20 mg/kg, q 24 h, PO, IM for 3-5days.
Plus
Prednisolone, 2-4 mg/kg, q 24 h, for 2 weeks and then tapered over 6-10 wk. In cats, the prognosis is more guarded, and generally, high doses of prednisolone 3 mg/kg, q 12 h are required for maintenance. Withhold food for 24-48 hours in animals with acute colitis; the protein source should be one to which the animal has not previously been exposed.

Conjunctivitis

Different strains of **Chlamydia psittaci** cause significant eye infection in cats.
The disease in cats involves the eye and mucosa of the upper respiratory tract (rhinitis, sinusitis, pharyngitis). Chlamydial keratoconjunctivitis can also be seen in dogs.

Clinical Symptoms
Cats: Early signs are unilateral, reddened, slightly swollen conjunctivae. Bilateral conjunctivitis develops after a few days and the conjunctivae become hyperemic and chemotic, with prominent follicles on the inside of the third eyelid in more severe cases. The signs are most severe 9-13 days after onset and then subside over 2-3 weeks. Secondary bacterial infections with signs of keratitis; pannus and corneal scarring may follow. Gastric epithelial cells of cats are also infected. Ulcerative keratitis may also be observed.
Dogs: keratoconjunctivitis

Diagnosis
Diagnosis can be confirmed by demonstration of chlamydial inclusions in exfoliative cytologic preparations from scrapings or by isolation of the chlamydial organism in chicken embryos or cell culture.

Treatment and Prevention
Tetracycline eye drops 1%, 4 times/day for 5-7 days. **Or**
Penicillin & streptomycin 200,000IU: 250mg/ml, 1-2ml/20kg, IM, q 24 h for 3-5days.
To reduce recurrence, treatment in cats should be continued for 7-10 days after clinical symptoms disappear.
Chlamydia may be transmitted to human and cause disease.

Hemorrhagic Gastroenteritis

Hemorrhagic Gastroenteritis (HGE) is a disease of dogs characterized by an acute onset of bloody diarrhea in formerly healthy dogs. The etiology is unknown. It has been suggested that an abnormal response to bacteria, bacterial endotoxins or diet may be involved, but there is no evidence to support these suppositions. There is no sex predilection and dogs of any age may be affected.

Clinical Symptoms

Dogs 2-4 years old: vomiting and bloody diarrhea, anorexia and depression; do not become clinically dehydrated but hypovolemic shock may develop. The disease is not contagious and may occur without obvious changes in diet.

Diagnosis

The clinical sign of acute, bloody diarrhea accompanied by an increased PCV, which is often >60%. There is no change in biochemical profile.

Treatment and Prevention

For severe cases

Lactated Ringer's 40-50ml/kg, PO, IV, with Potassium chloride for severe cases

Plus

Gentamicin Sulfate 2-4 mg/kg, q 8 h, IV (with IV fluid), IM, or SC.

For mild cases:

Sulfamethazine-Trimethoprim 200/40mg, 5- 30 mg/kg, IM, q 24 h, for 3-5 days for mild cases.

Food and water should be withheld for 2-3 days.

For protein source, dogs should be given food which is unfamiliar.

Oral Inflammatory and Ulcerative Disease

Inflammation of the oral tissues may be a primary or secondary disease. Inflammation in the oral cavity may affect the gingival tissue (gingivitis), periodontium (periodontitis), oral mucosa (stomatitis), tongue (glossitis), glossopalatine arches (faucitis), palate (palatitis) or pharyngeal tissue (pharyngitis). The etiology includes chemicals, neoplasma, metabolic disorders, autoimmunity or immune deficiency, infectious diseases, trauma, burns, radiation therapy or idiopathic oral inflammatory diseases. Infectious agents include distemper virus, **Leptospira canicola** and **L. icterohaemorrhagiae**. Uremia can cause stomatitis and oral ulcers. Periodontal disease, including gingivitis and periodontitis is the most common oral problem in small animals.

Clinical Symptoms

Signs vary widely with the cause and extent of inflammation. Anorexia may occur, especially in cats. Halitosis and drooling are common with stomatitis, glossitis and faucitis and saliva may be blood tinged. The animal may paw at its mouth and resent any attempt to examine the oral cavity because of pain. Regional lymph nodes may be enlarged.

Diagnosis

Usually on clinical symptoms and physical examination.

Treatment and Prevention

Acetylsalicylic acid, 10-25 mg/kg PO, q 8 hr. for 3 days. Or other analgesics

Plus

Penicillin and streptomycin (200,000IU: 250mg/ml), 1ml/25 kg, IM, q 24 hr. for 3-5 days. **Or**

Oxytetracycline 10 mg/kg, q 24 hr. IM for 5 days.

Sedate and make a complete examination and treat the problem accordingly. Clean the area with Lugol's solution or mild iodine.

Public health significance: the signs may be similar to rabies; thus handle animal with precaution.

Otitis Media and Otitis Interna

Otitis media, inflammation of the middle ear structures, is usually due to extension of infection from the external ear canal or to penetration of the tympanic membrane by a foreign object. Otitis media can lead to otitis interna and inflammation of the inner ear structures and can result in loss of equilibrium and deafness.

Clinical Symptoms

Head shaking, rubbing the affected ear on the floor and rotating the head toward the affected side, pain with discharge and inflammatory changes, facial nerve paralysis or Horner's syndrome (miosis, ptosis, enophthalmos and protrusion of the nictitans), or both, may be present on the same side as the otitis media and fall toward the affected side. Nystagmus may also be seen.

Diagnosis

The diagnosis can be confirmed by bulging, discoloration or rupture of the tympanic membrane; radiologically, cytologic examination (Gram's stain and Wright's stain) and culture of the exudate may be beneficial, along with sensitivity testing of any microbial isolates.

Treatment and Prevention

Penicillin and streptomycin 200,000IU: 250mg/ml, 2ml/25kg, IM, q 24 h for 3-5 days. **Or**
Oxytetracycline 10mg/kg, q 24 h, IM for 5 days. **Or**
Sulfamethazine-Trimethoprim200:40mg, 5- 30 mg/kg, IM, q 24 h, for 3-5days.
Plus
Dexamethasone 0.5 mg/kg IM, PO, IV, q 24 h during the first 5-7 days of treatment.
Note: Otitis interna usually responds well to long-term antibiotic therapy, but some neurologic deficits (e.g. incoordination, head tilt and deafness) may persist for life.
If the eardrum is ruptured, the tympanic cavity should be carefully cleaned with visualization through an otoscope and the use of long alligator forceps, flushes of warm saline and low vacuum suction.

Pneumonia

Pneumonia is an acute or chronic inflammation of the lungs and bronchi characterized by disturbance in respiration and hypoxemia and complicated by the systemic effects of associated toxins. The primary etiologic agents include **canine distemper virus, adenovirus types 1 and 2, parainfluenza virus** and **feline calicivirus** that predispose to secondary bacterial invasion of the lungs. Others are parasites such as **Filaroides**, **Aelurostrongylus** or **Paragonimus** spp., protozoa e.g. **Toxoplasma gondii**, tuberculosis or mycotic pneumonia.
Aspiration pneumonia may result from persistent vomiting, abnormal esophageal motility, or improperly administered medications (e.g. oil or barium) or food (forced feeding); it may also follow suckling in a neonate with a cleft palate.

Clinical Symptoms

Lethargy and anorexia are common with deep cough. Progressive dyspnea and cyanosis may be evident, especially on exercise. Body temperature is increased moderately and there may be leukocytosis. Auscultation usually reveals consolidation, which may be patchy but more commonly is diffuse. In later stages of pneumonia, there is increased lung density and peribronchial consolidation caused by the inflammatory process can be visualized radiographically. Complications such as pleuritis, mediastinitis or invasion by opportunistic organisms may occur.

Diagnosis

Bacterial culture and sensitivity testing is required and may include anaerobe and mycoplasma culture, especially in refractory cases. A viral etiology generally results in an initial body temperature of 40-41°C. Leukopenia, often expected, may not be seen in many viral respiratory infections (e.g. canine infectious tracheobronchitis, feline calicivirus pneumonia, feline infectious peritonitis pneumonia). A history of recent anesthesia or severe vomiting indicates the possibility of aspiration pneumonia. Acutely affected animals may die within 24-48 hours of onset. Mycotic pneumonias are usually chronic in nature.

Treatment and Prevention

See, antibiotic treatment for Otitis media and interna.

Ketoconazole for Dogs: 15 mg/kg; for cats: 10 mg/kg, PO q 12 hr.

Oxygen therapy may be used if cyanosis is severe, administered by means of an oxygen cage with a concentration of 30-50%.

Plus

Aminophyline 10 mg/kg q 8 hr. until the clinical sign subsides. If no response is seen after 48-72 hr of therapy, the treatment plan should be reassessed.

Note: Animals should be placed in a warm, dry environment, re-examined frequently; atropine sulfate is contraindicated for severe respiratory diseases.

Pyoderma

Pyoderma is a pyogenic infection of the skin. Pyodermas are common in dogs but uncommon in cats. Primary pyoderma is usually due to infection with **Staphylococcus intermedius.** Secondary pyoderma occurs after infection of skin lesions with bacteria.

Clinical Symptoms

Canine: Alopecia, follicular papules or pustules, epidermal collarettes and serous crusts. The trunk, head and proximal extremities are most often affected and the hairs are easily removed. In severe cases, signs include: erythema, swelling, ulcerations, hemorrhagic crusts, alopecia and draining tracts with serohemorrhagic or purulent exudate. The bridge of the muzzle, chin, elbows, hocks, interdigital areas and lateral stifles are more prone to deep infections but any area may be involved.

Cats: alopecia, ulcerations, hemorrhagic crusts and draining tracts which often indicate other systemic disease, like feline immunodeficiency virus or feline leukemia virus or atypical mycobacteria may be present.

Diagnosis

Typical lesions are indicative. Tests for the underlying etiology include a complete blood count, chemistry profile, urinalysis, thyroid profile, intradermal or serologic allergy testing and a food elimination diet trial for up to 3 months, biopsies for dermatopathology, immunoglobulin quantitation, low-dose dexamethasone suppression testing, and Adrenocorticotropic Hormone (ACTH) stimulation testing.

Treatment and Prevention

Enrofloxacin 5 mg/kg, PO or SC for ≥21 days. **Or**

Amoxicillin trihydrate-clavulanic acid 7 mg/kg, IM, q 12 hr. for ≥21 days.

Chlorhexidine digluconate + Miconazole nitrate, Dog, shampoo twice weekly until the symptoms subside and weekly thereafter or as necessary.

Chlorhexidine digluconate + Miconazole nitrate, Cat, Shampoo twice weekly for a minimum period until coat brushings are negative for the culture of M. canis. The maximum length of the treatment period should not exceed 16 weeks. **Or**

Cefovecin, Dog, 8mg/kg, SC, Stat. If required, treatment may be repeated at 14 day intervals up to three times.

For skin and soft tissue abscesses and wounds in cats: Cefovecin, 8mg/kg, SC, Stat. If required, an additional dose may be administered 14 days after the first injection. **Or**

Pradofloxacin, Dog, 3 mg/kg, PO, once daily through duration of treatment depending on nature and severity of infection and on response to treatment.

For chronic, recurrent or deep pyoderma

Enrofloxacin or Amoxicillin trihydrateclavulanic acid for ≥8 wk to resolve completely for chronic, recurrent or deep pyoderma antibiotics; **Or**

Shampoo therapy containing Benzoyl peroxide, povidone-iodine, chlorhexidene, ethyl lactate and triclosan to remove bacteria, crusts and scales, as well as reduce the pruritus.

Public health significance: Most fleas are zoonotic.

Arthritis

Arthritis is a non-specific term denoting inflammation of a joint. The most common arthritis in dogs and cats is osteoarthritis (OA). Arthritis in pets can also be caused by infection (septic arthritis or bacterial arthritis in dogs) or by rheumatoid arthritis where the body is attacking itself in an immune system malfunction. Joint degeneration can be caused by trauma, infection, immune-mediated diseases or developmental malformations. Contributing factors to OA include genetics, age of dogs, bodyweight, obesity, gender, exercise and diet.

Clinical Symptoms

Activity impairment: reluctance to exercise, decrease in overall activity, stiffness, muscle atrophy lameness, inability to jump, changes in gait such as 'bunny-hoping'.
Pain on manipulation: behavioral changes such as aggression or signs of discomfort.

Diagnosis

Initially physical exam will orient towards the affected joint or joints. Painful response in the bones and joints upon palpation, thickening of joint capsule, accumulation of joint fluid (effusion) or sometimes osteophytes and muscle atrophy (wasting). Physical exam finding is combined with radiopgraphy. Arthrocentesis may have minor changes in color, turbidity or cell counts of synovial fluid.

Treatment and Prevention

Drug Treatment

Asprin 10-25mg/kg PO ,q 12 hr.,
Carprofen 2.2-4mg/kg PO ,q 12 hr.,
Meloxicam 0.2 mg/kg PO initially and followed by 0.1mg/kg q 24 h and other pain-relieving drugs) reduce pain and inflammation. Caution is advised with long-term NSAID usage in dogs.
Corticosteroids (prednisolone 2-4mg/kg q 24 hr., Or dexamethasone 0.1-0.5mg/kg IV, IM, PO q 24 hr.) also suppress prostaglandin synthesis and subsequent inflammation, but short-term use is advised to prevent iatrogenic hyperadrenocorticism, cartilage degeneration and intestinal perforation.
Joint-fluid modifiers such as glycosaminoglycans or sodium hyaluronate may prevent cartilage degradation.

Non drug treatment

Nonsurgical therapies include weight reduction, controlled exercise on soft surfaces, and therapeutic application of warm compresses to affected joints.
Surgical options may include joint fusion (arthrodesis), most frequently performed on the carpus and tarsus; joint replacement, such as total hip replacement; joint excision, femoral head and neck osteotomy; and amputation. Prognosis is variable and depends on the location and severity of the arthropathy.

FUNGAL DISEASES OF DOGS AND CATS

Aspergillosis

Aspergillosis is caused by a number of **Aspergillus** spp, especially **A**. **fumigatus**. It is primarily a respiratory infection that may become generalized. Nasal and paranasal tissues of dogs are most commonly infected. Pulmonary and intestinal forms have been described in domestic cats, with most of the intestinal cases associated with feline infectious enteritis.

Clinical Symptoms and Lesions
In dogs lethargy, nasal pain, ulceration of the nares, sneezing, unilateral or bilateral sanguinopurulent nasal discharge, frontal sinus osteomyelitis and epistaxis. Lesions may involve the eyes. A layer of gray-black necrotic material and fungal growth may cover the mucosa of the nasal and paranasal sinuses.

Diagnosis
Visualization of fungal plaques by rhinoscopy together with serologic and either mycologic or radiographic evidence of disease are indicated. Positive culture and ELISA are confirmatory.

Treatment and Prevention
Griseofulvin15-20 mg/kg, PO with fatty food, q 24 hr. for 7-14 days. **Or**
Ketoconazole 15 mg/kg, PO, q 12 hr. for dogs and 10 mg/kg, PO, q 12 hr. to cats for 6-8 wk.
In chronic cases of canine nasal aspergillosis, surgical exposure and curettage with 10% iodine flushes.

Dermatophytosis

Dermatophytosis is an infection of keratinized tissue (skin, hair and claws) by one of the three genera of fungi **Epidermophyton**, **Microsporum** and **Trichophyton**. **Microsporum canis** is the most common infection.

Clinical Symptoms
Kittens are most commonly affected. Lesions consist of focal alopecia, scaling and crusting; most infections occur around ears and face or on extremities. The hair becomes brittle. Feline miliary dermatitis with pruritis is occasionally observed. In dogs the lesions include alopecia, scaly patches with broken hairs and dogs may also develop regional or generalized folliculitis with papules and pustules.

Diagnosis
Direct smear, culture and clinical symptoms are sufficient; the Wood's lamp is useful in establishing a tentative diagnosis.

Treatment and Prevention
Miconazole 1% solution, q 1-2 hr. then q 3-4 hr. after 3-4 days. In chronic or severe cases.
In chronic or severe cases
Griseofulvin in dogs 25-100 mg/kg once daily or divided in two doses; in cats 25-50 mg/kg daily, also divided doses. Treatment should continue for 2-4 wk past clinical cure.
Alternative
Ketoconazole 10 mg/kg, continue for 2-4 wk. after clinical cure. **Or**
Itraconazole at 5 mg/kg, daily should be continued for 2-4 wk. past clinical cure.
Dermatophytosis in dogs and shorthaired cats is usually self-limiting, but resolution can be hastened by treatment.
It is zoonotic and one has to take care during handling of infected dogs. Avoid contact with infected pets.

PARASITIC DISEASES OF DOGS AND CATS

HELMINTHES PARASITES

Dirofilariasis

Dirofilariasis is infection of particularly the right ventricle and aorta of dogs by **Dirofilaria immitis**; cats are also infected occasionally. Mosquitoes are intermediate hosts.

Clinical Symptoms
Gradual weight loss, decreased exercise tolerance and cough aggravated by exercise and in advanced cases, dyspnea, increased temperatures, abdominal fluid, cyanotic mucous membranes and periodic collapse are observed.

Diagnosis
Identification of Dirofilaria worm in blood samples plus clinical symptoms and history; serological tests are also available.

Treatment and Prevention
Arsenamide. Steroids may be given to reduce anaphylaxis
Acetylsalicylic acid plus prednisolone prevents thromboembolism
Ivermectin 6 mcg/kg, I/M, every 30 days

Prevention
Diethylcarbamazine daily, during the mosquito season for prevention
Imidacloprid + Moxidectin, Cat, 10 mg/kg BW imidacloprid and 1.0 mg/kg BW moxidectin, equivalent to 0.1 ml/kg BW spot on ,q month during the time of the year when mosquitoes are present.
Selamectin, Dog, 6 mg/kg, Spot on, should be administered within one month of the animal's first exposure to mosquitoes and monthly thereafter until 1 month after the last exposure to mosquitoes.

Ascariasis

The large roundworms (ascaridoid nematodes) of dogs and cats are common, especially in puppies and kittens. **Toxocara canis** is the most important cause of fatal infections in young pups. **Toxascaris leonina** occurs in adult dogs and in cats. The life cycles of **T. cati** and **T. leonina** are similar except that, in the former, no prenatal infection occurs, while in the latter, migration is restricted to the intestinal wall so that neither prenatal nor transmammary transmission occurs.

Clinical Symptoms
Lack of growth and loss of condition, worms may be vomited and are often voided in the feces. Pulmonary damage due to migrating larvae may be complicated by bacterial pneumonia. Diarrhea with mucus may be evident. In severe infections of puppies, verminous pneumonia, ascites, fatty degeneration of the liver and mucoid enteritis are common. Cortical kidney granulomas containing larvae are frequent in young dogs.

Diagnosis
Microscopic examination of feces for eggs.

Treatment and Prevention

Piperazine 50-100 mg/kg, PO at ones for dogs and cats. **Or**
Mebendazole 22 mg/kg, q 24 hr. PO with food for 3 days. **Or**
Pyrantelpamoate, dogs: 5-10 mg/kg; cats 10 mg/kg, both PO, single dose. **Or**
Fenbendazole: bitches from 40 days pregnancy to day 14 after whelping plus nursing ones, 50 mg/kg; pups at 2 wk. after birth and repeated at 2- to 3-wk intervals to 3 month of age.
Emodepside + Praziquantel, Cat, 3 mg emodepside / kg and 12 mg praziquantel /kg, equivalent to 0.14 ml Profender / kg, Stat.
Milbemycin oxime + Praziquantel, Dog, 0.5 mg of milbemycin oxime and 5 mg of praziquantel per kg are given once orally.
Selamectin, Dog, 6 mg/kg, Spot on, Stat.
Nitroscanate, Dog, 50mg/kg, PO in the morning after overnight fasting with approximately one-fifth of the daily food ration.

Ancylostoma Caninum Infection

Ancylostoma caninum is the principal cause of canine hookworm disease in Ethiopia. Transmission may result from ingestion of infective larvae from the environment or, in **A. caninum**, ingestion of colostrum or milk of infected bitches or from larval invasion through the skin.

Clinical Symptoms

Acute normocytic, normochromic anemia followed by hypochromic, microcytic anemia in young puppies is the characteristic and often fatal. Dogs may be debilitated and malnourished. Diarrhea with dark, tarry feces accompanies severe infections. Anemia, anorexia, emaciation and weakness develop in chronic disease.

Diagnosis

Flotation of fresh feces from infected dogs and acute anemia and death may be seen in young pups before eggs are passed in their feces.

Treatment and Prevention

Mebendazole 22 mg/kg, q 24 hr. PO with food for 3 days. **Or**
Fenbendazole 50 mg/kg, q 24 hr. PO with food for 3 days.
Milbemycin oxime + Praziquantel, Dog, 0.5 mg of milbemycin oxime and 5 mg of praziquantel per kg are given once orally.

Plus, in severe cases

Iron dextran 10 mg/kg, q 24 h, IM in addition to anthelmintic to be followed by high protein diet in severe cases.

Prevention

Fenbendazole given to pregnant bitches from day 40 of pregnancy to day 14 after whelping greatly reduces transmammary transmission to the pups as prophylaxis.

Spirocerca Lupi

Spirocerca lupi infection is an esophageal worm of dogs. The adult worms localize within nodules in the esophageal, gastric or aortic walls. Infection is acquired when dogs eat dung beetles (the intermediate host) or other animals such as chicken or reptiles which are incidental hosts.

Clinical Findings

Clinical symptoms appear when the nodules become large enough to interfere with swallowing and frequently vomit when trying to eat. Other signs include spondylitis or enlargement of extremities characteristic of osteopathy. Dogs may die suddenly due to rupture of the aorta.

Diagnosis
Fecal examination, gastroscopic examination and radiologic examination.

Treatment and Prevention
Levamisole 5mg/kg PO stat. **Or**
Albendazole 5mg/kg PO stat.
Prevent dogs from eating dung beetles or raw chicken scraps, mice, lizard etc. that may harbor the parasite.

Strongyloidosis

Strongyloides stercoralis is a small, slender nematode that when fully mature is buried in the mucosa of the anterior half of the small intestine of dogs. Usually, infections are associated with warm, wet, crowded, unsanitary housing. The species found most often in dogs is identical to that found in man. Other species in dogs include **S. planiceps** and **S. fuelleborni**; **S. cati** and **S. tumefaciens** are found in cats. The parasitic worms are all females.

Clinical Symptoms
A blood-streaked, mucoid diarrhea, usually seen in young animals during hot humid weather, is characteristic. Early signs include emaciation and reduced growth rate are observed however, appetite usually is good. In the absence of concurrent infections, there is little or no fever. Usually in advanced stages, there is shallow, rapid breathing and pyrexia, and the prognosis is grave.

Diagnosis
Characteristic clinical symptoms and larvae identified by direct microscopical evaluation.

Treatment and Prevention
Mebendazole 22 mg/kg, q 24 hr. PO with food for 3 days. **Or**
Albendazole 25 mg/kg, q 12 hr. PO. **Or**
Ivermectin 200 mcg/kg, PO or SC stat.
Strongloides stercoralis infection causes severe disease in humans.

Tapeworms

Taenia species including **Dipylidium caninum** and **Taenia taeniaformis** are two important cestodes of pets acquired by ingestion of raw meat and offal. A number of cestodes can be expected in dogs. On sheep ranges and wherever wild ungulates and wild canids are common, dogs may acquire **Echinococcus granulosus** (the hydatid tapeworm).

Clinical Symptoms
Clinical symptoms vary from unthriftiness, malaise, irritability, capricious appetite and shaggy coat to colic and mild diarrhea; rarely, intussusception of the intestine, emaciation, and seizures are seen.

Diagnosis
Direct microscopic finding of proglottids or eggs in the feces. Fecal flotation may reveal the eggs of **E. granulosus** from other cestodes.

Treatment and Prevention
Mebendazole 22 mg/kg, q 24 hr. PO with food for 3 days. **Or**
Albendazole 25 mg/kg, q 12 hr. PO for 3 days. **Or**
Praziquantel at 7.5 mg/kg, PO for 2 consecutive days is effective against **Diphyllobothrium latum** and 20 mg/kg, PO as a single dose against **Spirometra mansonoide/ E. granulosus** same dosage for dogs and cats.

Emodepside + Praziquantel, Cat, 3 mg emodepside / kg and 12 mg praziquantel /kg, equivalent to 0.14 ml Profender / kg, Stat
Milbemycin oxime + Praziquantel, Dog, 0.5 mg of milbemycin oxime and 5 mg of praziquantel per kg are given once orally.
Nitroscanate, Dog, 50mg/kg, PO in the morning after overnight fasting with approximately one-fifth of the daily food ration.
Note: Cestodes of dogs and cats are of public health importance.

PROTOZOAL PARASITES

Amebiasis

Amebiasis is an acute or chronic colitis, characterized by persistent diarrhea or dysentery. The causative agent is **Entamoeba histolytica** where it infects dogs and cats. Transmission of cysts occurs by fecal-oral route. Cysts from human are likely sources of infection to dogs and cats.

Clinical Symptoms
Chronic diarrhea, dysentery, weight loss and anorexia are the usual signs in susceptible hosts.

Diagnosis
Demonstration of trophozoites or cysts in indirect saline smear of feces. Repeated examination may be necessary as it may pass in feces periodically.

Treatment and Prevention
Tinidazole 50 mg/kg, PO, q 24 h for 3 days in cats and dogs.
No zoonotic importance but humans are likeliest reservoirs of amoeba cyst.

Canine Babesiosis

Babesiosis is a tick transmitted hemoprotozoan infection caused in dogs by **B. gibsoni** and **B. canis** and **B. felis** in cats. Babesiosis is very common in dogs in Ethiopia. The parasite affects host erythrocytes. The disease is very severe in young dogs; though very young puppies that have maternal immunity are resistant. Canine babesiosis can range from chronic or subclinical to peracute and fatal, depending on the virulence of the species and susceptibility of the host.

Clinical Symptoms
Symptoms may come and go as the disease runs its course and can include lack of energy, lack of appetite, weakness, pale gums and tongue, orange or red colored urine, discolored stool, weight loss, enlarged spleen and jaundice. The severity of the disease depends on species of Babesia, presence of concurrent infections and age and immune status of the host.

Diagnosis
Clinical symptoms and apparent tick infestation are indicative. Demonstration of the Babesia parasite in erythrocytes of affected animals from blood film is confirmatory; serological tests are also available.

Treatment and Prevention
Diminazene aceturate 3-5 mg/kg, IM, stat. **Or**
Imidocarb dipropionate 5-6mg/kg IM or SC stat and repeated in 2 weeks. **Or**
Tetracyline10-20 mg/kg, PO, q 6-8 hr. for 3 days if infection occurs
Lactated Ringer's solution 40-50ml/kg, q 24 hr. I/V, I/P, or S/C can be given as a supportive treatment until rehydration is restored.

Prevention

Tick control.

In B. gibsoni infection Diminazene aceturate has been used for many years but this has been superseded in recent years by the anti-malarial drug Atovaquone 13,3mg/kg q 8 hr. in combination with Azithromycin 10mg/kg q 24 hr. for 10 consecutive days.

B. felis doesn't respond for normal babesiacide but for primaquine phosphate, 0.5mg/kg, I/M, with q 24 hr.

Provision of Vitamin B- complex is also helpful.

Cryptococcosis

Cryptococcosis is a fungal disease of dogs and cats caused by **Cryptococcus neoformans.** It may be systemic affecting the respiratory tract, Central Nervous System (CNS), eyes and skin (particularly the face and neck of cats). Transmission occurs by inhalation or wound contamination from the environment.

Clinical Symptoms and Lesions

In cats: sneezing, mucopurulent, serous or hemorrhagic unilateral or bilateral chronic nasal discharge, polyp-like mass in the nostril and a firm subcutaneous swelling over the bridge of the nose. Cutaneous papules and nodules that are fluctuant to firm, larger may ulcerate. Neurologic signs like depression, changes in temperament, seizures, circling, paresis and blindness. Dilated unresponsive pupils and blindness due to exudative retinal detachment, granulomatous chorioretinitis, panophthalmitis and optic neuritis.

In dogs: More severely disseminated disease and most have CNS or ocular involvement. Clinical symptoms are usually related to meningoencephalitis, optic neuritis and granulomatous chorioretinitis. About 50% of dogs have lesions in the respiratory tract, usually the lungs and most have granulomas throughout the body. Structures often involved in order of decreasing frequency are kidneys, lymph nodes, spleen, liver, thyroid, adrenals, pancreas, bone, GI tract, muscle, myocardium, prostate, heart valves and tonsils.

Diagnosis

The most rapid method of diagnosis is cytologic evaluation of nasal exudate, skin exudate, Cerebrospinal Fluid (CSF), or samples obtained by paracentesis of the aqueous or vitreous chambers of the eye or by impression smears of nasal or cutaneous masses. Gram's stain, Indian ink, new methylene blue and Periodic Acid-Schiff (PAS) stains are used. Culture from exudate, CSF, urine, joint fluid and tissue samples are useful.

Treatment and Prevention

Amphotericin B 0.1-0.5 mg/kg (cats) and 0.25-0.5 mg/kg (dogs), IV with 5-20ml of 5% dextrose solution 3 times a week until a cumulative dose of 4-11 mg/kg is reached. **Or**

Ketoconazole 5-20 mg/kg, q 12 h, PO has been used to successfully treat cats for a week. **Or**

Itraconazole 5-10mg/kg, q 12-24 h, PO a week for dogs. **Or**

Fluconazole 5-10 mg, PO, q 12-24 h, a week for cats.

Giardiasis

It is enteric disease of dogs and cats caused by parasitic protozoa of the genus **Giardia.** Transmission among hosts occurs by fecal-oral route. Cysts excreted in feces are immediately infective.

Clinical Symptoms

Chronic diarrhea either continuous or intermittent lasting for weeks, months or in unusual cases years or feces becomes pasty in consistency, pale, with foul odor and steatorrhea is likely to present.

Diagnosis

Microscopic examination feces is the definitive approach.

Treatment and Prevention
Tinidazole 50 mg/kg PO q 24 h for 3 days in cats and dogs.

Toxoplasmosis

Toxoplasma gondii is a protozoan parasite that infects most species of warm-blooded animals, including birds and man. Members of the cat family are the only known definitive hosts and thus, serve as the main reservoir. Transmission is by ingestion of raw meat, food, contaminated feces or congenitally. Infection clears due to immunity development.

Clinical Symptoms
Toxoplasmosis is usually a subclinical illness. However, in young animals, interstitial pneumonia, myocarditis, hepatic necrosis, meningoencephalomyelitis, chorioretinitis, lymphadenopathy and myositis are observed. The corresponding clinical symptoms include fever, diarrhea, cough, dyspnea, icterus, seizures and death. Finally, systemic signs are observed in immunocompromised adult animals.

Diagnosis
Available tests include the Sabin-Feldman dye test, complement fixation, direct and indirect hemagglutination, latex agglutination, modified agglutination, ELISA and indirect fluorescent antibody testing.

Treatment and Prevention
Clindamycin in dogs, 10-40 mg/kg q 12-24 hr. for 2-4 weeks; cats, 25-50 mg/kg, q 12-24 hr. for 2-4 weeks.
Or
Sulfadiazine 73 mg/kg and pyrimethamine 0.44 mg/kg act synergistically. Pyrimethamine related side effect relate to folate deficiency including agranulocytosis, megaloblastic anemia and thrombocytopenia; dehydration; gastrointestinal toxicity (diarrhea, occasionally bloody; vomiting); anorexia/decreased appetite; weakness; weight loss.
Toxoplasma gondii may cause abortion in pregnant women and may cause birth defects in the fetuses.

EXTERNAL PARASITES OF DOGS AND CATS

Canine Demodex

This common skin disease of dogs occurs only when large numbers of **Demodex canis** mites inhabit hair follicles, sebaceous glands or apocrine sweat glands. The mites are transmitted from dam to puppies during nursing within the first 72 hours after birth, but they are not contagious. **D. canis** and **D. cati** are normal inhabitants of dogs and cats, respectively but may cause clinical disease if animals suffer from immunosuppression, natural or iatrogenic. Other factors known to predispose to generalized demodicosis include systemic disease, estrus, and heartworm infection.

Clinical Symptoms
There are two clinical forms; localized demodicosis occurs in dogs <1 year old, and 90% of these cases are thought to resolve spontaneously. Lesions consist of areas of focal alopecia and erythema. A percentage of these cases will progress to the generalized form. In second form, generalized alopecia, papules, pustules and crusting are observed. Lesions are usually aggravated by secondary bacterial infections and pododermatitis is common. Dogs can have systemic illness with generalized lymphadenopathy, lethargy and fever when deep pyoderma, furunculosis and draining tracts are seen.

Diagnosis
Deep skin scrapings reveal mites, eggs and larval forms in high numbers. Dermatophyte cultures are essential, because dermatophytosis and demodicosis can be concurrent conditions.

Treatment and Prevention

Amitraz dips 0.025%, every 2 weeks after clipping the entire hair coat and a benzoyl peroxide shampoo among those applied for its follicular flushing activity before the dip is applied.

Ivermectin (400-600 mcg/kg PO, stat.

Sarolaner Dog, 2–4 mg/kg PO, at monthly intervals for three consecutive months. Treatment should be continued until skin scrapings are negative on at least two consecutive occasions one month apart.

Or

Afoxolaner, Dog 2.7–7 mg/kg, PO, Monthly administration of the product until two negative skin scrapings are obtained one month apart. **Or**

Fluralaner , Dog, 25 – 56 mg fluralaner/kg BW, PO, Stat.

The secondary bacterial infection must be treated with the appropriate antibiotic.

Fleas and Flea Allergy Dermatitis

Fleas cause flea allergy dermatitis. They are vectors of typhus-like rickettsiae and intermediate host for filarid and cestode parasites. Species that commonly infest dogs and cats are: **Ctenocephalides felis** (the cat flea), **C. canis** (the dog flea), **Pulexsimulans** (a flea of small mammals) and **Echidnophaga gallinacea** (the poultry sticktight flea). However by far the most prevalent flea on dogs and cats is **C. felis**.

Clinical Symptoms

In Flea Allergy Dermatitis (FAD) pruritus is observed. In dogs, papulocrustous lesions distributed on the lower back, tail head and posterior and inner thighs. Dogs may be particularly sensitive in the flanks, caudal and medial thighs, ventral abdomen, lower back, neck and ears. Affected dogs are restless and uncomfortable, scratching, licking, rubbing, chewing and even nibbling at the skin. Common secondary lesions include areas of alopecia, erythema, hyperpigmented skin, scaling, papules and broken papules covered with reddish brown crusts.

In cats, papule, which often becomes crusted is observed on the back, neck and face. Pruritus may be severe, evidenced by repeated licking, scratching and chewing. Cats with FAD can have alopecia, facial dermatitis, exfoliative dermatitis and "racing stripe" or dorsal dermatitis.

Diagnosis

History, clinical symptoms, presence of fleas or flea excrement, results of intradermal testing.

Treatment and Prevention

Microencapsulated chlorpyrifos (**see** accompanying insert for application). **Or**

Permethrin (see accompanying insert for application). **Or**

Non-encapsulated chlorpyrifos (**see** accompanying leaflet for application). **Or**

Organophosphates, carbamates, pyrethroids, for dosage, (**see** External Parasites of Cattle: Acaricides).

(S)-Methoprene + Fipronil + Amitraz, Dog, 6.7 mg/kg b.w. for fipronil, 6 mg/kg for (S)-methoprene and 8 mg/kg for amitraz. At monthly intervals throughout the tick and/or flea seasons. **Or**

Afoxolaner, Dog 2.7–7 mg/kg, PO, Monthly intervals throughout the flea and/or tick seasons. **Or**

Fluralaner , Dog, 25 – 56 mg fluralaner/kg BW within one weight band, q 12 weeks. **Or**

Imidacloprid + Moxidectin, Cat, 10 mg/kg BW imidacloprid and 1.0 mg/kg BW moxidectin, equivalent to 0.1 ml/kg BW spot on ,q month. **Or**

Indoxacarb + permethrin , Dog, 15 mg indoxacarb/kg BW and 48 mg/kg permethrin, equivalent to 0.1 ml spot-on solution per kg BW Stat. **Or**

Sarolaner, Dog, 2–4 mg/kg BW, PO, a single dose should be administered at monthly intervals and continue throughout the flea season.

For secondary infection-

Antibiotics could be given for secondary infection

Plus

Prednisolone 0.5-1.0 mg/kg daily, tapering the dosage and using alternate-day therapy until the lowest dose possible that still controls the pruritus.

Note: Anti-inflammatory therapy should never be used as a substitute for flea control. Fleas are vectors for zoonotic diseases and control should be implemented.

Control of fleas from the host and its environment is basic. Administration of topical or systemic residual insecticides or administration of topical, injectable or oral IGRs have become the preferred method to eliminate fleas.

Feline Demodex

Demodex cati is thought to be a normal inhabitant of feline skin follicles but causes disease in immunosuppressed animals. Another unnamed demodex species is found only in the stratum corneum of cats. Feline demodicosis is uncommon.

Clinical Symptoms

In localized demodicosis, there are one or several areas of focal alopecia on the head and neck. In generalized disease, alopecia, crusting and secondary pyoderma of the whole body are seen. The generalized form has also been associated with other systemic disease, especially diabetes mellitus. In some cases, ceruminous otitis externa has been the only clinical sign. Pruritus is variable.

Diagnosis

Examination of skin scraping and medical evaluation is indicated in cats with generalized disease.

Treatment and Prevention

Weekly lime sulfur dips (2%) are safe and usually effective;

Amitraz (0.0125%–0.025%) has been used but is not approved for use in cats and can cause anorexia, depression and diarrhea.

Prognosis of generalized demodicosis is unpredictable because of its potential relationship with systemic disease. Some cases spontaneously resolve.

Notoedres

This rare, highly contagious disease of cats and kittens is caused by **Notoedres cati**, which can opportunistically infest other animals, including man. The mite and its life cycle are similar to the sarcoptic mite.

Clinical Symptoms

Pruritus is severe. Crusts and alopecia are seen, particularly on the ears, head and neck and can become generalized.

Diagnosis

It is based on clinical symptoms and identification of mites in skin scraping.

Treatment and Prevention

Amitraz spray, 0.025% as sarcoptic mange

Imidacloprid + Moxidectin, Cat, 10 mg/kg BW imidacloprid and 1.0 mg/kg BW moxidectin, equivalent to 0.1 ml/kg BW spot on ,Stat.

Zoonotic importance: Notoedres cati is zoonotic and care should be exercised when handling infested cats.

Sarcoptes

Sarcoptes scabiei subspecies **canis** infestation is a highly contagious disease of dogs. The mites are fairly host-specific, but animals (including man) that come in contact with infested dogs can also be affected.

Clinical Symptoms

Asymptomatic carriers may exist. Intense pruritus is characteristic. Primary lesions consist of a papular eruption that, due to self-trauma, develops thick crusts with secondary bacterial infection. Typically, lesions start on the ventral abdomen, chest, ears, elbows and legs and if untreated, become generalized. Dogs with chronic, generalized disease develop severe thickening of the skin with fold formation and crust buildup, peripheral lymphadenopathy, and emaciation; dogs so affected may die.

Diagnosis

It is based on the history of severe pruritus of sudden onset, several extensive superficial scrapings at different sites and fecal flotation to reveal mites or eggs.

Treatment and Prevention

The hair should be clipped, the crusts and dirt removed by soaking with a good antiseborrheic shampoo. **Plus**
Amitraz, 0.025%. **Plus**
Prednisolone 0.5-1mg/kg, PO.
Sarolaner, Dog, 2–4 mg/kg BW, PO, a single dose should be administered at monthly intervals for two consecutive months. **Or**
Selamectin, Dog, 6 mg/kg, Spot on, a single dose should be administered for two consecutive months.
Or
Afoxolaner Dog 2.7–7 mg/kg, PO, Monthly administration of the product for two consecutive months.
Or
Fluralaner , Dog, 25 – 56 mg fluralaner/kg BW, PO, Stat.

Tick Infestation

For description, diagnosis, treatment and control **see** Ticks in cattle. For dosages, **see** the label on the container of acaricides or mite treatment above.

DISEASES OF THE REPRODUCTIVE SYSTEM

Acute Orchitis and Epididymitis

Acute inflammation or infection of the testis or epididymis may be caused by trauma, infection (fungal, bacterial or viral), or testicular torsion.

Clinical Symptoms
Pain and swelling of the testes, epididymides and/or scrotum are common signs. There may be wounds or draining tracts in the scrotal skin.

Diagnosis
Palpation of scrotal contents for evidence of torsions, foreign material or focal lesions of the testes or epididymis. Semen should be collected for cytology and for bacterial and mycoplasmal culture. Collection of semen may be difficult in an animal with acute orchiepididymitis. A fine-needle aspirate of the involved testis or epididymis provides material for cytology and culture. A rapid slide agglutination test for **Brucella canis** should be performed.

Treatment and Prevention
Broad-spectrum bactericidal antibiotics, if maintaining fertility is important. **Or**
Antifungal agents are indicated for fungal infections.
Plus
Prednisolone 0.5 mg/kg, IM, daily. **Or**
Acetylsalicylic acid 10 mg/kg, q 12 h and local hypothermia (i.e. cool water packs) may decrease testicular damage caused by local swelling and hyperthermia.
Note: The prognosis for maintaining fertility is guarded. There is no successful treatment for **B.canis** infection. All the antifungal agents interfere with spermatogenesis.

Balanoposthitis

Inflammation of the penile-preputial mucosa caused by bacteria normally present in the preputial cavity is common in dogs after trauma, lacerations, neoplasia, foreign bodies or mixed infections or phimosis may result in development of severe balanoposthitis. Balanoposthitis is rare in cats.

Clinical Symptoms and Diagnosis
Mucopurulent preputial discharge, swelling of the prepuce and possibly pain are seen. The penis and prepuce should be examined thoroughly for underlying predisposing factors. Bacterial cultures of the preputial cavity, although sometimes difficult to interpret because of the normal flora are helpful in identifying unusual organisms or antibiotic sensitivities for refractory cases.

Treatment and Prevention
Povidone-iodine douche or sterile saline solution
Infusing antibiotic ointment into the preputial cavity for 7-10 days.
Plus
Broad-spectrum systemic antibiotic 7-10 days if systemic illness is present.
Note: Recurrence of mild balanoposthitis is common despite therapy. Castration may be helpful.

Benign Prostatic Hyperplasia

Benign prostatic hyperplasia is the most common prostatic disorder and is found in most intact male dogs >6 year old. It is a result of androgenic stimulation or altered androgen /estrogen ratio.

Clinical Symptoms

There may be no clinical symptoms or tenesmus, persistent or intermittent hematuria, and bleeding may occur.

Diagnosis

History and physical examination confirmed by radiological examination and cytologic examination of massage or ejaculate specimens and confirmed by biopsy.

Treatment and Prevention

Finasteride 0.1mg/kg, PO q 24 h, for 16wks
Megestrol acetate (antiandrogens) 0.55 mg/kg, PO, q 24 h for 10 days
Medroxyprogesterone acetate, 3 to 4 mg/kg SC at intervals of 10 weeks or longer.
Note: Castration is the treatment of choice.

Dystocia

Difficult birth may result from myometrial defects, metabolic abnormalities such as hypocalcemia, inadequate pelvic diameter, insufficient dilation of the birth canal, fetal hormone (corticosteroid) deficiency, fetal oversize, fetal death or abnormal fetal presentation and posture.

Clinical Symptoms

Dystocia should be considered in any of the following situations:
Parturition does not occur within 24 hr. after the drop in rectal temperature 37.7°C;
Strong abdominal contractions or active labor for 1-2 hours without passage of a puppy or kitten;
The resting period during active labor exceeds 4-6 hr.
The bitch or queen is in obvious pain (crying, licking or biting the vulva);
There is a black, purulent or hemorrhagic vaginal discharge;
There are signs of systemic illness; or
Gestation is prolonged.

Diagnosis

History of pelvic trauma and breeding dates; a change in the normal dark green color of vaginal discharge at parturition; sterile vaginal examination and position of fetus and use of radiography and ultrasonography.

Treatment and Prevention

Forceps may be carefully used to remove dead fetuses or to facilitate delivery of malpresented or partially delivered fetuses. Gentle manipulation and adequate lubrication must be used to prevent injury or death to living fetuses. Episiotomy may help
Cesarean operation is indicated for obstructive dystocia, dystocia accompanied by shock or systemic illness, for primary uterine inertia, when active labor is prolonged, and/or if medical management has failed. The skin sutures should be removed in 8-10 days after operation
Oxytocin 3-20 IU in bitches, 2-5 IU in queens, I/M up to three times at 30- minutes intervals, with or without 10% calcium gluconate, 3-5 mL, IV slowly, to promote uterine contraction. This is indicated if there is proper fetal position and presentation, and in the absence of obstruction. Suggested initial doses are: 0.25 IU for dogs weighing less than 5 kg; 0.5 to 1.0 IU for dogs weighing 5 to 10 kg; 1 to 3 IU for dogs weighing 10 to 30 kg; and 3 to 5 IU for dogs weighing more than 30 kg.

Paraphimosis

It is an inability to completely retract the penis into the preputial cavity after erection and development of a functional phimosis. It is seen most often after semen collection or coitus or due to foreign objects around the penis, a constricting band of hair at the preputial orifice, trauma or chronic balanoposthitis.

Clinical Symptoms and Diagnosis

The exposed penis becomes edematous, desiccated and painful. If untreated, ulceration, ischemic necrosis or gangrene may develop. If recognized early, before severe edema and pain develop, paraphimosis is easily treated.

Treatment and Prevention

Cleansing and lubrication of the penis and replacement inside the prepuce by gently sliding the prepuce first in a posterior direction, extruding the penis further

Bathing the exposed penis in cold or hypertonic solutions may also help reduce swelling

If the urethra has been damaged, an indwelling urinary catheter may be needed

If necrosis or gangrene is severe, amputation of the penis and prepuce and castration may be necessary.

Phimosis

The inability to extrude the penis through an abnormally small preputial orifice may be congenital or develop because of inflammation, neoplasia, edema or fibrosis after trauma, irritation or infection.

Clinical Symptoms

Signs are variable. Usually, the problem is unnoticed until the dog attempts to mate and is unable to copulate.

Diagnosis

Physical examination of the prepuce and penis.

Treatment and Prevention

Phimosis caused by an inflammatory or infectious disease may be relieved by warm compresses, antibiotic therapy and urine diversion with a catheter. The prepuce should be lavaged daily with physiologic saline solution to reduce urine scalding.

If the dog is not used for breeding, therapy probably is not needed, although castration should be considered to prevent unexpected arousal.

Surgical enlargement of the preputial orifice is indicated if the animal is to be used for breeding, if the phimosis contributes to balanoposthitis, or in the unlikely event that phimosis interferes with normal micturition.

Pyometra

Pyometra is a hormonally mediated diestrual disorder characterized by an abnormal uterine endometrium with secondary bacterial infection. Factors associated with occurrence of pyometra include administration of long-lasting progestational compounds to delay or suppress estrus, administration of estrogens to mis-mated bitches and post-copulation infections. **Escherichia coli** is the most common bacterium isolated in cases of pyometra, although, **Staphylococcus, Streptococcus**, **Pseudomonas**, **Proteus** spp, and other bacteria have also been recovered. Pyometra is less common in queens than in bitches. Pyometra can develop in uterine tissue left after ovariohysterectomy (stump pyometra). Pyometra can also occur secondary to postpartum metritis.

Clinical Symptoms

Clinical symptoms are seen during diestrus, usually 4-8 weeks after estrus, or after administration of exogenous progestin. The signs are variable and include lethargy, anorexia, polyuria, polydipsia and vomiting. When the cervix is open, a purulent vulvar discharge, often containing blood, is present.

When the cervix is closed, there is no discharge and the large uterus may cause abdominal distention. Signs can progress rapidly to shock and death. Only 20% of affected animals have a fever. Shock may be present.

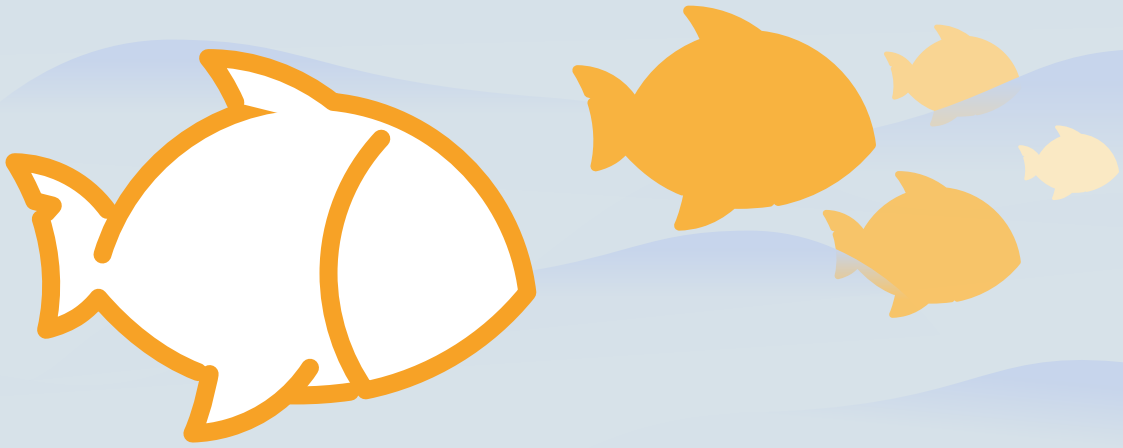
Diagnosis

Diagnosis can be established from the history, physical examination and abdominal radiography and ultrasonography. Vaginal cytology is often helpful in determining the nature of the vulvar discharge; complete blood count, biochemical profile and urinalysis is indicated.

Treatment and Prevention

Ovariohysterectomy is the treatment of choice for pyometra. Medical management could be considered if preserving the reproductive potential of the bitch or queen is desired. Fluids (IV) and broad-spectrum, bactericidal antibiotics should be administered. Fluid, electrolyte and acid-base imbalances should be corrected as quickly as possible, before ovariohysterectomy is performed. The bacterial infection is responsible for the illness and will not resolve until the uterine exudate is removed.
Oral broad spectrum antibiotics should be continued for 7–10 days after surgery.
Penicillin and streptomycin 200,000IU: 250mg per ml, 1ml/25 ml, IM, for 14 days. **Or**
$PGF_{2\alpha}$, 0.25 mg/kg, SC, q 24 h. for 5 days should be used in the bitch and queen. Synthetic analogs (e.g., cloprostenol, fluprostenol, and prostalene) are much more potent.
Broad-spectrum, bactericidal antibiotics, chosen on the basis of culture and sensitivity tests, should be given for ≥2 wk.
1% iodine solution, daily vaginal douches are beneficial in promoting vaginal drainage, cervical dilation, and uterine evacuation.
Note: Oral antibiotics after ovariohysterectomy. The animal should be re-examined 2 weeks after completion of medical therapy.
Prostaglandin therapy should not be used in bitches/queens with closed pyometra.

SECTION VIII



DISEASES OF FRESHWATER FISH[8]

8 Guide

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.

A wide variety of diseases and parasites have been recorded from fish both in the wild and in captivity. These include bacterial, fungal, viral, protozoan, trematode, nematode, copepod and mite species, as well as several nonparasitic diseases and syndromes. However, only a small number of these have caused fish health problems and mortalities in aquaculture facilities and some parasites have been recorded from wild fish only. The most common parasites and diseases that affect the health of freshwater fish in aquaculture facilities are described hereunder.

WATER QUALITY PARAMETERS

Heavy Metals

Fish are sensitive to dissolved metals. Metals are most toxic in low alkalinity waters in which high concentrations of metals remain dissolved. Metal contamination may occur from the following sources: Leaching from lead, copper or galvanized (containing zinc) plumbing fittings, groundwater (especially soft, acid water) that may have high concentrations of metals, Rainfall run-off from soils that are poorly buffered or are contaminated (e.g. mine wastes).

Symptoms
Metal poisoning varies and depends on the metal and fish species. Effects can be chronic or sub-lethal leading to a gradual decline in fish health and reduced immunity to other diseases. Most heavy metals primarily affect the gills.

Diagnosis
Water and fish samples must be submitted to a laboratory specializing in water analysis and diagnostic labs.

Treatment and Prevention
Avoid using metal fittings (especially those containing, lead, copper or zinc) from aquaculture systems reduces the risk of metal poisoning.

PH

A pH range of 6.5-9.0 is generally recommended for freshwater fish. Outside of this range is stressful to fish and levels less than 4.0 (acidic) and greater than 11.0 (alkaline) are usually lethal. Sources of low pH water include groundwater in contact with silicate minerals, and waters draining from or overlaying acid sulphate soils. The metabolic activity of fish and other aquatic organisms produces acid, which can gradually lower the pH in some systems. Some groundwater may also contain high pH levels. Both meters and test strips are used to measure pH levels. Extreme pH levels can be treated by:

- Dilution with fresh water and/or the addition of a buffering agent to lower/increase pH
- Monitoring pH levels regularly (daily in intensive recirculating systems, less frequently in ponds) and avoiding build-up of large algal blooms in ponds.

Salinity

Salinity is a measure of all ions in water and is most commonly expressed as parts per thousand (ppt). Freshwater generally has less than 0.5 ppt salinity while seawater is 30-40 ppt. Salinity tolerance of fish varies with species and length of exposure. The easy solution for correcting salinity is a dilution of the pond water using distilled water or similar.

Temperature

Temperature dramatically affects fish metabolism and each species has a preferred temperature range. Absolute temperature ranges do not exist because tolerance depends on several factors, including the acclamation history, salinity, life-stage and reproductive status. All fish are susceptible to rapid changes in temperature, but seem to tolerate a rapid decrease in temperature better than the reverse. At the extremes, fish may be stressed to the point where growth and survival are affected. At low temperatures, outside the fish's optimal range, the immune system is suppressed, which increases susceptibility to other fish diseases. Temperature stress depends not only on how low or high the temperature becomes, but how quickly it arrives at the temperature. Water temperature is readily measured by thermometer.

Water-borne Contaminants and other Problems

Organic compounds such as PCBs, detergents and hydrocarbons, pesticides, herbicides and molluscicides can be extremely toxic to fish and may reach aquaculture water supplies by accidental spillage or contamination and runoff from agricultural and industrial lands. Clinical symptoms vary with the type of compound but tend to include distress, respiratory failure, avoidance behavior and death.

WATER QUALITY AND ENVIRONMENTAL FACTORS RELATED TO FISH DISEASES

Fishes are poikilotherm aquatic animals and need continuous acclimatization to the environmental changes. Each species has a preferred range of water quality parameters and outside this range will suffer stress, which may lead to disease and even death.

Fish can be affected by external environment water contaminants including pollution from agriculture, sewage or industrial sources as well as natural variations in water quality caused by geology, soils and the climate. Internally, water quality may be influenced by farm management, fish husbandry (including stocking density and feeding) as well as excreted wastes of the fish. The physical action of the fish in stirring up sediments can also have an impact on some parameters. There are several key water quality parameters such as temperature, dissolved oxygen, pH, ammonia and nitrite that should be tested when any fish disease is suspected.

Ammonia Poisoning

Ammonia poisoning is one of the most common water quality problems in aquaculture. It is the primary nitrogenous waste product of fish, but also comes from the decay of organic matter such as waste feed and algae. Potential causes of elevated ammonia levels include overcrowding of fish, recent medication or other chemicals added, newly established recirculation systems, failure of biological filters, reduced water flow and accumulation of waste feed or other organic matter. Ammonia toxicity also varies with salinity, water hardness and other stressors.

Symptoms
Acute ammonia toxicity can cause behavioral abnormalities such as hyper-excitability and fish often stop feeding. Sub-lethal ammonia poisoning decreases growth and disease resistance.

Diagnosis
Chemical measurement of total ammonia, pH and temperature using appropriate test kits and meters are needed.

Treatment and Prevention
Dilution by addition of fresh water, stop feeding, decrease stocking density, reduce temperature and reduce PH are some of the immediate actions
Improve husbandry practices and take an active approach to managing the health of stock.

Carbon Dioxide Poisoning

Carbon dioxide (CO_2) is essential for plant growth and is usually present as a free gas or bound with other elements to form bicarbonates, carbonates and organic compounds. High levels of free CO_2 can cause problems at low pH levels (pH<7.0). The main sources of CO_2 are respiratory wastes of the fish, decomposition of organic matter, respiration by aerobic bacteria (such as within biological filters), groundwater and the atmosphere. Fish hemoglobin is highly sensitive to free CO_2. High levels of CO_2 interfere with oxygen uptake.

Symptoms

Symptoms include dyspnoea and chronic inflammation and calcium carbonate disposition in the kidneys and epaxial muscles. In severe cases there is granulomatous inflammation and cystic dilatation of tubules.

Diagnosis

Check content of CO_2 where >12 mg/L CO_2 is indicative.

Treatment and Prevention

Increasing aeration, reducing stocking density and in ponds, addition of calcium hydroxide.
Use of mechanical aeration and oxygenation systems as well as use of degassing devices to strip CO_2.

Chlorine/ Chloramine Poisoning

Chlorine is commonly used to treat town water to make it suitable for human consumption. Ammonia is sometimes also added to stabilize the chlorine and reacts producing chloramines. Both chlorine and chloramines are extremely toxic to fish and can cause acute or sub-acute toxicity.

Symptoms

Chlorine/chloramine poisoning includes breathing difficulty, dyspnoea and death. Gill tissue of affected fish becomes necrotic. Fish exposed to chlorine poisoning appear to have improved survival if the water is super-saturated with oxygen for several days and the temperature is lowered.

Diagnosis

A reliable water quality spectrophotometer-based test kit can measure chlorine. Chlorine can also be detected by smell. Any detectable amount of chlorine or chloramines is undesirable.

Treatment and Prevention

Chlorine can be removed from water by vigorous aeration, but chloramines are not so readily removed
Water may also be filtered through activated carbon/fresh charcoal or natural clays (zeolites)
Treat with a chemical neutralizer such as Sodium Thiosulphate.

Environmental Hypoxia

Environmental hypoxia is a low concentration of Dissolved Oxygen (DO) in the water and is perhaps the greatest risk in aquaculture. The minimum DO level of 5 mg/L is considered ideal for optimal growth and reproduction. Below this level food consumption decreases and growth slows. A DO of less than 2 mg/L is very stressful and may lead to opportunistic infections (bacteria, fungi, parasites).
It is caused by excessive plant respiration, increase water temperature, presence of decaying organic matter and use of industrial waste-water, breakdown of the aeration systems, overstocking and overfeeding.

Symptoms

Fish cease feeding and may "gasp" at the water surface or gather at water inlets. Death occurs with opercula flared and open mouth. Often large fish die and small fish may survive. The symptoms of hypoxia can also be symptomatic of gill damage (which impairs oxygen uptake) and anemia, which reduces oxygen bioavailability.

Diagnosis
Direct measurement of DO and temperature are required and should be taken in situ at the time of water sampling.

Treatment and Prevention
Non drug treatment
Increasing aeration, increasing water exchange, stopping feeding and reducing stocking density
Monitor ammonia and nitrite to ensure that biological filtration is functioning in Recirculating Aquaculture Systems (RAS)
Routine (daily) monitoring of DO in ponds (at dawn) and tanks are important for long-term management.

Prevention
In ponds, provide supplementary aeration that is automatically timed to operate during periods of low DO (especially early morning)
Install and maintain emergency back-up aeration and oxygenation systems
Do not overstock tanks and ponds, improve feed management and/or increase water exchange.

Gas Supersaturation (Gas Bubble Disease)

Gas super-saturation may be caused by excessive plant photosynthesis during daylight hours in ponds with dense algal blooms, inflow water saturated with gases as a result of heating under pressure, water inflow pipes sucking in air (prior to pumps) and ground waters that are supersaturated with nitrogen and/or carbon dioxide. Most gas emboli are produced by excess nitrogen. Oxygen rarely causes gas bubble disease because it is assimilated metabolically and hence is less likely to form persistent bubbles.

Symptoms
Fish become listless and float to the surface. Small bubbles can be seen forming in superficial blood vessels typically on gills and fins and behind the eyes.

Diagnosis
Measure water with a saturometer (an oxygen meter will give an indication of oxygen Supersaturation only). Small bubbles will immediately form on any object placed in the water.

Treatment and Prevention
Eliminate excess gases from the water source by aerating water in a reservoir to allow gases to equilibrate.
Install de-gassing devices on pipelines and systems before the water enters the culture vessels.

Hydrogen Sulfide Poisoning

Hydrogen sulfide (H_2S) forms from the reduction of sulphate under anaerobic conditions. It is more of a problem in brackish water/marine systems, but can occur in freshwater systems where there is an accumulation of organic matter. Disturbing pond sediments can also release this gas from the mud.

Symptom
H_2S interferes with respiration causing hypoxia. Concentrations of H_2S between 0.5-1.0 mg/L can cause acute mortality.

Diagnosis
It is detected by a characteristic "rotten egg" smell, and can be measured by a reliable water quality spectrophotometer.

Treatment and Prevention
Vigorous aeration or use of a degassing device.
Raising pH and lowering temperature also reduce toxicity.
Regular addition of fresh water also assists in reducing H_2S concentrations.

Nitrite Poisoning (Brown Blood Disease)

Nitrite is an intermediary product in the oxidation of ammonia to nitrate by bacteria. Many of the circumstances that lead to a buildup of ammonia can also lead to nitrite poisoning. In ponds, nitrite peaks can occur in autumn as the optimum temperature of the two bacteria (**Nitrosomonas** and **Nitrobacter**) are different leading to nitrite accumulation. Susceptibility to nitrite toxicity varies enormously between species.

Symptoms
Behavioral changes are similar to characteristics of hypoxia. In acute to chronic cases, dyspnoea may occur and gills become light chocolate to brown in color due to a change in blood coloration.

Diagnosis
Test for nitrite using a chemical test kit.

Treatment and Prevention
Nitrite toxicity is reduced by addition of salt (<50 mg/L salt is usually sufficient) as the chloride ion competitively inhibits nitrite uptake across the gills
Same as for Ammonia poisoning.

MAJOR BACTERIAL DISEASES OF FRESH WATER FISH

Bacterial Gill Disease (BGD)

One or more species of filamentous bacteria including **Flavobactrium** spp**. Flavobacterium branchiophilum** is the dominant species, which is Gram-negative and bacilli of varying length. Nearly all species of intensively cultured freshwater fish are potential hosts. Carrier fish in the rearing units or a contaminated water sources are sources of contamination.

Symptoms
The major diseases signs and lesions are loss of appetite; swimming at the surface and often upstream (against the current) to ensure efficient water flow over the gill surfaces.

Diagnosis
The diagnosis is usually based on clinical signs (obvious) and test for specific bacteria.

Treatment and Prevention
Treat fish using antibiotics such as Erythromycin given in feed at a rate of 100 mg/kg/day for 21 days
Chloramin-T dipping at 8.5-10 ppm for 1 hour daily for 3 days.
Non drug treatment
Improve environmental conditions (increase DO).

Columnaris Infection

It is caused by **Flavobacterium columnare,** which belongs to the family **Flavobacteriaceae.** It is Gram-negative, and slender flexible rods (0.5x4-12 µm). It inhabited mucus of both normal and diseased fish. The other possible causative agent is **Flexibacter columnaris**. Stressors such as crowding, high temperatures (> 18°C), and physical injury due to rough handling are key factor in outbreaks of the disease.
The **Flavobacterium columnare** distributed worldwide in freshwater sources and infect many different wild and cultured freshwater fish. Infected animals with gill or cutanueous lesions, dead fish and clinically healthy carrier fish are sources of infection.

Symptoms
A systemic form characterized by sudden death. Shallow erosions of the skin, and frayed fins and eroded gills are also manifested as disease signs. Gills lesions are more necrotic and death more rapid, skin lesions may develop hemorrhagic ulcers and lesions usually have yellow color.

Treatment and Prevention
External bath and antimicrobial feed additive therapy to combat both cutaneuous and systemic infections
Avoid exposure to the disease by using disease free water or UV disinfected water
Avoid temperature of water above 18°C, crowding and rough handling.

Edwardsellosis

The causative agent is **Edwardsiella tarda** which is G-negative, short rod–shaped and facultative anaerobe. The bacterium has broader host range but is predominantly enteric pathogen of both freshwater and marine fish. Organically polluted water from the intestine of domestic animals, rats, birds, frogs, turtles and healthy fish are the sources of infection. Carrier fish, aquatic invertebrates and human beings may serve as reservoir. The mode of transmission is via water by organisms shed through feces. Cannibalism of infected fish, feeding on dead or infected carcasses can also serve as mode of transmission. Birds, contaminated nets, equipment and carriers can also spread the disease from one pond to another.

Clinical Symptoms and Lesions
Abnormal swimming including spiral movement and floating near water surface are some of the behavioral changes observed. Loss of pigmentation, exophthalmia, opacity of eyes, swelling of the abdominal surface, petechial hemorrhage in the fin and skin and rectal hernia are some of the external signs. The internal signs are watery and bloody ascites in the abdominal space and congested liver, spleen and kidney.

Diagnosis
Clinical signs consistent with the disease are presumptive. However, isolation and identification of the etiological agent is confirmatory.

Treatment and Prevention
Oxytetracycline 50 mg/kg of body weight/day for 10 days.

Prevention
Removal of the stress factors,
Removal of sick and dead fish as soon as possible,
Good management of fish farm.
Note: The bacterium is responsible for several clinical conditions in humans such as diarrheal gastroenteritis, typhoid-like illness, peritonitis with sepsis and induced abscesses in liver.

Enteric Septicemia of Catfish (ESC)

It is caused by **Edwardsiella ictaluri** affecting mostly the channel catfish (**Ictalurus punctatus**). The disease also affects all ages of channel catfish. Transmission is via water by organisms shed with the feces, cannibalism of infected fish and feeding on dead and infected carcasses. The pathogen can enter the host through the gut, nasal openings and possibly the gills. Rough handling, close confinement, improper diet, low water chlorides, poor water quality and water temperature fluctuations lead to increased susceptibility to infection. Diseased or carrier fish are the main source of infection.

Symptoms and Lesions

The most common behavioral signs are: loss of appetite, swimming in tight circles and chasing their tails. Hang in the water column with the head up and tail down. The most common physical signs are red circular spots (2-3 mm diameter) over the entire body, white circular spots (2-3 mm diameter), rash-like areas on body, bloody areas on the base of fins, raised reddish area on top of head, ulcerated areas on top of the head (hole-in-the-head), protruding eyes, bloated fluid filled belly (yellowish or bloody), hemorrhages in internal organs and tissue and white pustules in the liver.

Diagnosis:

Isolation of the causative bacterium from the internal organs or brain tissue

Treatment and Prevention

Terramycin 2.5 - 3.75 g/45 kg of fish given daily in feed for 10-14 days and then followed by a 21-day withdrawal period before slaughter

Sulfadimethoxine-ormetoprim 50 mg/kg of fish body weight/day given in feeds for 5 days followed by a 3 days withdrawal period before slaughter

Florfenicol 10 mg/kg/day incorporated into floating catfish feed and administered daily for 10 consecutive days. The withdrawal period is12 days.

Prevention

The most common control measures are reduction of stress factors, proper nutrition and feeding practices and vaccination.

Epitheliocystis

Chlamydia organisms are the etiological agents, which are intracellular Gram-negative bacteria. Both freshwater and marine fish are affected. Mortalities usually associated in cultured fish and infection spreads very quickly in fingerlings held in overcrowded ponds.

Symptoms

Epithelial cells grow gradually into a grossly hypertrophic body. Severe erosion of the gill architecture, which interferes with the respiratory function is also involved.

Treatment and Prevention

Antibiotics such as Furazolidone given in feed 25-35 mg/kg/day for 20 days in all spp are effective.

Non drug treatment

Reduction of stocking densities.

Furunculosis

Aeromonas salmonicida is the causative agent which is an obligate fish pathogen, Gram-negative, facultative aerobe and non-motile rod. It causes disease both in freshwater and marine fish and both wild and cultivated fish, especially salmon. Contaminated waters and infected or carrier fish are sources of infection. Infected fish with open sores can spread the disease. The transmission is primarily horizontal via contaminated water, eggs, carriers, equipment, clothing and surface of aquatic birds. Due to the high density of fish in aquaculture, this disease can spread rapidly and with devastating results.

Symptoms
The major pathological changes observed in the affected fish are large boils on the side of the fish, external and internal hemorrhaging, swelling of the vents and kidneys, ulcers, gastroenteritis and fraying of fins (tail rot).

Diagnosis
Isolation and identification of the bacterium (easy from the kidney) are confirmatory diagnostic methods.

Treatment and Prevention
Sulfamerazine 10gm/45kg of fish/day for 3 weeks
Amoxicillin given in the diet at a dose rate of 80 mg per kg body weight/day for 10 days is effective against both a moderate and severe challenge.

Prevention
Use of clean water/fish, minimizing overcrowding
Vaccination has also reduced the incidence of Furunculosis outbreak.

Motile Aeromonad Infection (MAI)

The causative agents include Aeromonas **(A) hydrophila, A. sobria and A. cavia** which are Gram-negative, rod-shaped and facultative anaerobic bacteria. The disease common in freshwater aquatic environments in which all freshwater species are susceptible. Others such as frogs, snails, shrimp and humans are also affected by the disease. These bacteria are opportunistic pathogens transmitted horizontally from normal flora of healthy fish, diseased fish or frog and survivors which are carriers.

Symptoms
The symptoms are non-specific and easily confused with other diseases. Usually hemorraghic and necrosis or necrotic lesions on the skin/muscles, superficial circular or grayish-red ulcerated lesions around mouth similar to enteric red mouth disease, hemorrhaging of fins and exopthalmia are observed externally. Swollen and soft kidney, petechiae of musculature and intestines free of food are also observed as internal pathological changes.

Diagnosis
Diagnosis can be done by observing symptoms, lesion and usually by isolation of the bacterium from the kidney.

Treatment and Prevention
Provide feeds containing antibiotics such as Oxytetracyline 75mg/kg/day for 10days. Or
Amoxicillin and Ampicillin given in feed at a rate of 40-80mg/kg/day for 10days for systemic infections.

Non drug treatment
Good management
The disease causes gastroenteritis and occurs mostly in young children. People with compromised immune system are more vulnerable groups.

Mycobacteriosis in Fish/Atypical Mycobacteriosis

The disease is caused by Mycobacteriosis (M**.) marinum, M. fortuitum** and **M. chelonea**. It has a worldwide distribution and both freshwater and marine fish (many species) are equally affected. The disease is common, especially in intensive aquaculture systems. Infected fish are a constant source and reservoir of infection to other fish.

Clinical Symptoms and Lesions

Anorexia, weight loss accompanied by scale loss, ulcers or non-specific hemorrhagic lesions, spinal curvature, lethargy, loss of equilibrium, darker skin color, abdominal swelling, low level but chronic mortality, poor reproductive performance and milliary tubercle like lesions may be found in the liver, spleen and kidney.

Diagnosis

Chronic mortality, especially if accompanied by poor condition or emaciation and the existence of fish with chronic ulcers or unexplained reproductive problems can be used for presumptive diagnosis. Preliminary diagnosis is usually based on the presence of granulomatous lesions, either grossly visible or microscopic. Definitive diagnosis is based on culture using Lowenstein-Jensen medium.

Treatment and Prevention

Little scientific data on presence of approved drugs hence treatment is not recommended. Infected fish will spread the infection to their offspring which will be a chronic problem for the producer and a risk to employees on the farm and the wholesale facility. Ultimately, it will be a risk to the consumer
The infected system should be depopulated and all equipment disinfected with a mycobacteriocidal agent such as Lysol® or concentrated (50 to 70 percent) ethyl alcohol
Control is usually based on thorough cleaning and disinfection of all surfaces with a mycobacteriocidal agents (e.g. Nitric oxide).

Mycobacetrial infection of fish are zoonotic which means the organisms can cause diseases in human.

Yersiniosis/Enteric Red Mouth Disease

Yersiniosis is caused by **Yersinia ruckeri** affecting variety of fish species and age groups. It is acute in small fish up to fingerling size and chronic in larger fish. Fish most at risk are those subject to stress arising from poor management or environmental changes such as elevated temperatures or poor water quality (high ammonia and lots of organic matter). The bacterium is shed in the feces of infected fish and the quantity increases exponentially with stress. Carriers and diseased fish are the primary source and reservoir.

Clinical Symptoms and Lesions

There are no specific early signs of the disease. But behavioral changes like swimming near the surface, sluggish movement, dark color skin and inappetence are observed. The external signs are unilateral or bilateral exophthalmia often with patch of hemorrhagic congestion on the iris of the eye, subcutaneous hemorrhage in the mouth and throat (strongly indicative of the disease and hence the term enteric red mouth), hemorrhagic congestion at the base of the pectoral and pelvic fins, distended vent and a pallor to the gills arising from bacterial induced anemia. The internal signs are petechiation on the pyloric caecae, hypertrophy of the spleen, peritonitis and gastro-intestinal tract filled with clear to yellow mucus.

Diagnosis

Presumed from history at a site and clinical signs but these are not specific. Confirmation is most easily made by culture from tissue samples of the spleen, heart and kidney. Polymerase chain reaction technique is available but is best used to confirm the identity of ambiguous isolates.

Treatment and Prevention

Florfenicol 10 mg/kg/day incorporated into floating catfish feed and administered daily for 10 consecutive days

Amoxicillin given in the diet at a dose rate of 80 mg per kg body weight.

Non drug treatment

Good husbandry and water quality are key to minimize the stress that precipitates the disease.

MAJOR FUNGAL INFECTIONS OF FRESHWATER FISH

Branchiomycosis/Gill Rot

Two fungal organisms associated with the occurrence of gill rot are **Brachiomyces sauguins and Brachiomyces demigrans.** Infection occurs primarily on the gill filaments, lamellar capillaries and other tissues of the gills, both produce branched and non-septate hyphae. Most species of freshwater fish are susceptible. There is also a strong association between outbreaks of the disease and the presence of environmental stress such as low pH, low dissolved oxygen or a high algal bloom. Fungal spores carried in the water and debris on pond bottom are sources of infection for susceptible fish. Ponds with bottom sediments rich in organic matter increases sporulation.

Clinical Symptoms and Lesions

Acute course of the disease will be observed during warm months and disease signs include lethargy, bright-red gills due to impaired circulation and some areas of the gills become white to brown depending on the stage of necrosis. The subacute form develop slowly and the symptoms are marbling appearance of the gill on sectioning, raged and corroded gills. The chronic cases include signs like pale gills, swollen lamellae and slight to moderate necrosis on the gills.

Treatment and Prevention

Treatment with chemicals such as malachite green at a rate of 1.0 ppm at interval of 4-7 days and copper sulphate at 0.1-0.2 ppm are recommended.

Non drug treatment

Strict sanitation and disinfection. Dead fish should be collected regularly and burned or deeply buried

Ponds with enzootic branchiomycosis should be dried and treated with calcium oxide (quicklime)

Transport of the infected fish from place to place and stress factors should be avoided.

Saprolegniasis/Water Mold Disease/ Skin Fungus/ Cotton Wool Disease

Saprolegniasis is a fungal disease of fish and fish eggs. It is common in fresh or brackish water (salinity level of 28 parts per thousand). Each case of the disease becomes a source of zoospores for the surrounding water and an increased possibility of invasion on to other fish. Dead fish and fish eggs are a fertile medium for more fungal growth and production of zoospores.

The disease will attack an existing injury on the fish and can spread to healthy tissue. Saprolegniasis may be secondary to other infections (like bacterial gill disease) or damage to skin, fins or gills from external parasites. Poor water quality (for example, water with low circulation, low dissolved oxygen, or high ammonia) and high organic loads, including the presence of dead eggs are often associated with Saprolegnia infections.

Clinical Symptoms and Lesions

Fluffy, cotton-like, white to gray/gray to gray-brown growth manifested on the skin, fins, gills or eyes of fish or on fish eggs. Ulcerative mycoses progress to a deep necrotic muscular lesion. Death occurs within 36 hours of infection if the gills are involved.

Diagnosis
Presumptive diagnosis can be achieved based on disease signs. However, identification of the fungi will be used for definitive diagnosis.

Treatment and Prevention
Good management practices such as good water quality and circulation, avoidance of crowding to minimize injury, good nutrition and removal of dead eggs and fish at regular intervals
The disease can also be controlled using effective fungicides such as Malachite Green 1.0 ppm at interval of 4-7 days in fish and 2 ppm for 15 minutes every 24 hours for eggs.

Systemic Mycoses: Ichtyophonus

This diseases is caused by **Ichthyophonus hoferi.** The disease can affect both fresh and saltwater fish as well as wild and cultured fish. It is spread by fungal cysts which are released through feces (contact with fecal materials) and by cannibalism of infected fish or consumption of infected carcass.

Clinical Symptoms and Lesions
In severe cases-the following signs and symptoms will be observed: paper texture of the skin and lateral or dorsoventral curvature of vertebral column (scoliosis). Kidney, liver, spleen, spinal cord, ventricles of the heart and brain may be swollen with white or gray necrotic foci.

Diagnosis
Clinical signs, lesions observed at autopsy and presence of cyst in squash preparation of affected organs can be used for diagnosis. Isolation and identification of the fungal agent can give definitive diagnosis.

Treatment and Prevention
There is no cure. The infection will be carried for entire life. However, prevention is the only control option.

Prevention
Avoid feeding raw fish or raw fish products to culture fish
Complete disinfection of the raceway or aquaria and ponds with dirt bottoms need drying for months to totally eliminate the fungus.

FRESHWATER FISH PARASITOSIS

True worms can be either parasitic or commensal, internal or external. Parasitic worms act only in their best interest and at the expense of their hosts' resources, whereas commensal worms either benefit their hosts or have no effect on their host or environment.

Cestode Infection

Cestodes use fish as primary or intermediate host. They infest the alimentary tract, muscle or other internal organs. Larval cestodes called plerocercoids are some of the most damaging parasites to freshwater fish when affecting vital organs such as brain, eyes or heart.

Clinical Symptoms
More commonly, failure to thrive or gain weight is the most frequent sign of infection.

Diagnosis
It can be done by its signs and identifying its characteristic egg. Internal cestode parasites such as tapeworms are significantly harder to diagnose in fish. Active passing of cestode segments in feces can be very difficult to see. Positive diagnosis of cestode infection can only be made with a fresh fecal microscopic examination (identifying its characteristic egg).

Treatment and Prevention
Niclosamide dissolved in DMSO at a rate of 0.1 ppm as Immersion. This is also effective on immature parasites
Niclosamide given in feed at a rate of 40 mg/kg/day for 3 days. **See also Annex 6: Veterinary Drugs Administered for the Treatment of Fish Diseases**

Crustacea Infection

Parasitic crustacea are increasingly serious problems in cultured fish and can impact wild populations. Most parasitic crustacea of freshwater fish can be seen with the naked eye as they attach to the gills, body and fins of the host. Three most important genera are discussed below.

A. Ergasilus

Ergasilus are often incidental findings on wild or pond-raised fish and probably cause few problems in small numbers. However, their feeding activity causes severe focal damage and heavy infestations can be debilitating. Mostly affects the gills of freshwater fish, commonly seen in warm weather.

Clinical Symptoms
Debilitation and secondary bacterial or fungal infection.

B. Lernaea

Lernaea also known as anchor worm; is a common parasite of goldfish and koi fish. The copepod attaches to a fish, mates and the male dies. The female then penetrates under the skin of the fish and differentiates into an adult.

Clinical Symptoms
Debilitation and secondary bacterial or fungal infection may occur.

C. Argulus

Argulus or fish louse is a large parasite that attaches to the external surface of the host and is visible with naked eyes. **Argulus** is uncommon in freshwater aquarium fish but may occur if wild or pond-raised fish are introduced into the tank. It is especially common on goldfish and koi fish.

Clinical Symptoms
Debilitation and secondary bacterial or fungal infection.

Treatment and Prevention
Trichlorfom PO at a rate of 0.05 mg/kg every third day for 3 occasions. **See also Annex 6: Veterinary Drugs Administered for the Treatment of Fish Diseases**

Leech Infestation

Leeches are members of the annelid group occasionally seen in wild and pond-raised fish. Leeches have anterior and posterior suckers. These suckers will attach themselves to the side of a fish or to the inside of their mouths, where they secrete an anticoagulant (blood thinner) that can significantly affect fish health. They are found in both freshwater and marine systems.

Clinical Symptoms
Chronic anemia and secondary infection at the point of attachment are common.

Diagnosis
Although adult leeches are easy to see and manually remove, life cycle (i.e. potential offspring) considerations should be taken with any treatment protocol.

Nematode Infections

One of the largest group of worms, the nematodes contain a variety of parasitic, commensal, and zoonotic worms that can all potentially affect aquatic animals. This contains the genera **Ancylostoma, Uncinaria, Bunostomum** and **Toxocara.** Many aquatic invertebrates can be involved in the nematode life cycle. It is important to understand how these many components can be involved in parasite life cycles. Three common nematodes affecting fish are described.

A. Camillanus

Camillanus is easily recognized as a small thread-like worm protruding from the anus of the fish.

Clinical Symptoms
Signs of nematodiasis include anemia, emaciation, unthriftiness and reduced vitality.

Diagnosis
Based on clinical signs and identification of the parasite.

B. Capillaria

Capillaria is a large roundworm commonly found in the gut of angelfish, often recognized by its double operculated eggs in the female worm. Heavy infestations are associated with debilitated fish, but a few worms per fish may be benign.

Clinical Symptoms
See above (treatment for Camillanus)

Diagnosis
It is characterized by identifying its egg.

C. Eustrongylides

Eustrongylides is a nematode that uses fish as its intermediate host. The definitive host is a wading bird, a common visitor to ponds. The worm encysts in the peritoneum or muscle of fish and appears to cause little damage. Because of the large size of the worms infected fish may appear unsuitable for retail sales.

Clinical Symptoms
See above (symptoms for Capillaria).

Diagnosis
By identifying the parasite from the skin.

Treatment and Prevention
Triclabendazole immersion at a rate of 25 mg/L for 12 hours. **See also Annex 6: Veterinary Drugs Administered for the Treatment of Fish Diseases**

PROTOZOA INFECTIONS

A. CILIATES

Ciliates have a direct life cycle and many are common inhabitants of pond-reared fish. Most species do not seem to bother host fish until numbers become excessive. Many of the parasites proliferate in organic debris that accumulate at the bottom of a tank or vat. Nets, hoses or caretakers' wet hands easily transmit ciliates from tank to tank. Typical symptoms of ciliates include skin and gill irritation displayed by flashing, rubbing and rapid breathing. The most common ciliates are:

i. White Spot Disease

It is caused by **Ichthyophthirius multifiliis.** It is called 'Itch'.

Clinical Symptoms
Small blister-like raised lesions develop along the body wall and/or fins. If the infection is restricted to the gills, the gills will appear swollen and be covered with thick mucus without white spots.

Diagnosis
Identification of the parasite on the gills, skin and/or fins is definitive. Classically **I. multifiliis** is identified by its large horseshoe-shaped macronucleus.

ii. Chilodonella

Chilodonella is a ciliated protozoan that causes infected fish to secrete excessive mucus. Infected fish may flash and show similar signs of irritation. Many fish die when infestations become moderate (five to nine organisms per low power field on the microscope) to heavy (greater than ten organisms per low power field).

Clinical Symptoms
Common signs are excessive mucus secretion, irritation and many fish die.

Diagnosis
Examine scrapings of skin mucus or gill filaments microscopically.

iii. Tetrahymena

Tetrahymena is a protozoan commonly found living in organic debris at the bottom of an aquarium or vat. **Tetrahymena** is a teardrop-shaped ciliate that moves along the outside of the host.

Clinical Symptoms
Exophtalmia (markedly enlarged eyes) is common symptom.

Diagnosis
Identification of **Tetrahymena** internally is significant.

iv. Trichodina

Trichodina is one of the most common ciliates present on the skin and gills of pond-reared fish. Low numbers (less than five organisms per low power field) are not harmful, but when fish are crowded or stressed and water quality deteriorates, the parasite multiplies rapidly and causes serious damage.

Clinical Symptoms
Typically, heavily infested fish do not eat well and lose condition. Weakened fish become susceptible to opportunistic bacterial pathogens in the water.

Diagnosis
Trichodina can be observed on scrapings of skin mucus, fin or on gill filaments. Its erratic darting movement and the presence of a circular, toothed disc within its body easily identify it.

v. Ambiphyra

Ambiphyra, previously called **Scyphidia** is a sedentary ciliate that is found on the skin, fins or gills of host fish. It is common on pond-reared fish and when present in low numbers (less than five organisms per low power field) it is not a problem.

Clinical Symptoms
Debilitation associated with deteriorated water quality.

Diagnosis
Its cylindrical shape, row of oral cilia and middle bank of cilia identify **Ambiphyra**.

vi. Apiosoma

Apiosoma, formerly known as **Glossatella**, is another sedentary ciliate common on pond-reared fish. **Apiosoma** can cause disease if their numbers become excessive. The organism can be found on gills, skin or fins.

Diagnosis
The vase-like shape and oral cilia are characteristic.

vii. Epistylis

Epistylis is a stalked ciliate that attaches to the skin or fins of the host. **Epistylis** is of greater concern than many of the ciliates because it is believed to secrete proteolytic ("protein-eating") enzymes that create a wound, suitable for bacterial invasion at the attachment site.

Clinical Symptoms
Wounds dispersed on the skin and fins.

Diagnosis
It is similar in appearance to **Apiosoma** except for the non-contractile long stalk and its ability to form colonies.

viii. Capriniana

Capriniana, historically called **Trichophyra** is a sessile ciliate that attaches to the host's gills with a sucker.

Clinical Symptoms
In heavy infestations **Capriniana** can cause respiratory distress in the host.

Diagnosis
They have characteristic cilia attached to an amorphous-shaped body.

B. FLAGELLATES

Flagellated protozoans are small microscopic parasites that can infect fish externally and internally.

i. Hexamita / Spironucleus

Hexamita is an intestinal parasite commonly found in the intestinal tract of freshwater fish. Recent taxonomic studies have labeled the intestinal flagellate of freshwater angelfish as **Spironucleus**.

Clinical Symptoms
Sick fish are extremely thin and the abdomen may be distended. The intestines may contain a yellow mucoid (mucus-like) material.

Diagnosis
Make a squash preparation from the intestine and examine it at 200 or 400x magnification. The flagellates move by spiraling.

ii. Ichthyobodo

Ichthyobodo, formerly known as **Costia**, is a commonly encountered external flagellate. **Ichthyobodo** can be located on the gills, skin and fins; however, it is difficult to identify because of its small size.

Clinical Symptoms
Infected angelfish also produce excessive mucus that can give dark colored fish a gray or blue coloration along the dorsal body wall. Infected fish flash and lose condition, often characterized by a thin, unthrifty appearance.

Diagnosis
Microscopically identify **Ichthyobodo** by its corkscrew swimming pattern.

iii. Piscinoodinium

Piscinoodinium is a sedentary flagellate that attaches to the skin, fins and gills of fish. The common name for **Piscinoodinium** infection is "Gold Dust" or "Velvet" Disease. **Piscinoodinium** is most pathogenic to young fish. The life cycle of this parasite can be completed in 10-14 days at 73-77°F, but lower temperatures can slow the life cycle.

Clinical Symptoms
The parasite has an amber pigment, visible on heavily infected fish. Affected fish will flash, go off feed and die. There is amber pigment on the skin.

Diagnosis
Identification and signs on the skin.

iv. Cryptobia

Cryptobia is a flagellated protozoan common in cichlids. **Cryptobia** typically is associated with granulomas, in which the fish "walls off" the parasite. These parasites have been observed primarily in the stomach, but may be present in other organs.

Clinical Symptoms
Fish infested with **Cryptobia** may become thin, lethargic and develop dark skin pigmentation.

v. Myxozoa

Myxozoa are parasites that are widely dispersed in native and pond-reared fish populations. Most infections in fish create minimal problems, but heavy infestations can become serious, especially in young fish. Myxozoans are parasites affecting a wide range of tissues.

Clinical Symptoms
Clinical signs vary, depending on the target organ. For example, fish may have excess mucus production, observed with **Henneguya** infections. White or yellowish nodules may appear on target organs. Chronic wasting disease is common among intestinal myxozoans such as with **Chloromyxum**. "Whirling disease" caused by **Myxobolus cerebralis** has been a serious problem in salmonid culture.

Diagnosis
Spores can be observed in squash preparations of the affected area at 200 or 400 x magnifications or by histologic sections.

vi. Microsporidia

Microsporidians are intracellular parasites that require host tissue for reproduction. Fish acquire the parasite by ingesting infective spores from infected fish or food. Replication within spores (schizogony) causes enlargement of host cells (hypertrophy).

Clinical Symptoms
Infected fish may develop small tumor-like masses in various tissues. Its clinical signs can range from no visible lesions to mortalities. It has severe morbidity.

Diagnosis
It is confirmed by finding spores in affected tissues, either in wet mount preparations or in histologic sections.

vii. Coccidia

Coccidia are intracellular parasites described in a variety of wild-caught and cultured fish. Their role in the disease process is poorly understood, but there is increasing evidence that they are potential pathogens. The most common species encountered in fish are intestinal infections but also reproductive organs, liver, spleen and swim bladder.

Clinical Symptoms
It depends on target organ affected but may include general malaise, poor reproductive capacity and chronic weight loss.

Diagnosis
Histologic or electron microscopy are definitive for tissue coccidian.

Treatment and Prevention
Diclazuril 2.5%, mixed in food of the fish at a dose of 200 mg/kg, for 12 days
Lasalocid mixed in food of the fish at a dose of 150 mg/kg, for 12 days. **See also Annex 6: Veterinary Drugs Administered for the Treatment of Fish Diseases.**

TREMATODE (FLUKE) INFECTIONS

Flukes exist in almost all systems in very small numbers, but do not always induce clinical signs of disease. Only when an individual or system is stressed do their small numbers rapidly multiply and spread. The most basic of the worms are monogenean and digenean trematodes.

Monogenean Trematodes

Monogenean trematodes, also called flatworms or flukes, commonly invade the gills, skin and fins of fish. They have a direct life cycle and are host and site-specific. **Gyrodactylus** and **Dactylogyrus** are the two most common genera of monogeneans that infect freshwater fish.

Clinical Symptoms

Freshwater fish infested with skin-inhabiting flukes become lethargic, swim near the surface, seek the sides of the pool or pond and their appetite dwindle. Areas of attachment show scale loss and may ooze a pinkish fluid, swollen and pale gills, respiration rate may be increased and fish will be less tolerant of low oxygen conditions with piping and gulping air at the water surface. High mortality may occur if large numbers (>10 organisms per low power field) of monogeneans infest the skin or gills. Secondary infection by bacteria and fungus is common on tissue with monogenean damage.

Digenean Trematodes

Digenean trematodes have a complex life cycle involving a series of hosts. Fish can be primary or intermediate host depending on the digenean species. They are found externally or internally, in any organ. These digeneans can become encysted into gill tissue and can cause respiratory distress.

Clinical Symptoms

Mortalities in baitfish, but usually the only negative effect is reduced growth rate, even when the infection rate is high. In cases where mortalities occur, there are unusually high numbers in the eye, head and throughout the visceral organs.

Treatment and Prevention

Benzyl-ureas given in feed at a rate of 10 mg/kg/day especially for Monogenean flukes. **See also Annex 6: Veterinary Drugs Administered for the Treatment of Fish Diseases**

SECTION IX

DISEASES OF HONEYBEES[9]

9 *Guide*

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.

The honeybees can be affected by many infectious diseases and parasites, pests, poisons and predators. Both the brood and adult forms of the honeybees are affected by infectious agents specific to each stage of life. Brood diseases are easier to recognize. Generally bee diseases can be caused by bacteria, virus, protozoa, fungus and mites. The transmission of bee diseases from one colony to another can be by adult bee, re-use of contaminated comb, beekeepers, beekeeping equipment, feeding honey and pollen.

INFECTIOUS DISEASES OF HONEYBEE

American Foulbrood

American Foulbrood (AFB) is the most virulent infectious brood disease caused by the spore-forming bacterium **Paenibacillus larvae sub-spp. larvae [Bacillus larvae]**. The spore is resistant to heat and drought, capable of germinating in a favorable environment. The vegetative form of the bacteria is infectious. The spores germinate into vegetative form in the larval and early young bee pupae gut, which digests them with enzymes secreted by the bacterium.

Clinical Symptoms
Death typically occurs after the cell is capped, during the last two days of the larval stage or first two days of the pupal stage. The symptoms of AFB include: characteristic sour odor, perforated or sunken capping, darker in color than healthy brood capping and resultant black scales that are difficult to remove from the cell because of their stickiness.

Diagnosis
Inicative characteristic signs include glue-like consistency of dead larvae and are tested by Ropy test (Stir in the cell with a match stick. The affected larvae sticks tenaciously forming long string or rope when it is drawn) or Holst Milk Test (Swirl the affected individual with 1% skim milk; if the milk clears, it is indicative of AFB). Confirmatory laboratory tests include culture or transmission experiment.

Treatment and Prevention
Oxytetracycline 200 mg/colony in 1.6 g powdered sugar, apply at the top bars as dust, three times at 4-5 days interval; treat all colonies.
Precaution: commence one month before the first major nectar flow and again after the honey crop has been removed.

Chalkbrood

Chalkbrood, a fungal brood disease of honey bees, is caused by the spore-forming fungus, **Ascosphaera apis**. Worker, drone and queen larvae are all susceptible. Spores of the fungus are ingested with the larval food and germinate in the hindgut of the bee larva. A change in brood nest temperature can trigger chalkbrood disease by enhancing growth of **A. apis** in the honey bee. Common causes of stress are high and low temperatures, too moist or dry conditions, poor nutrition, failing queen, poor hive management and moving hives. Transmission occurs by contaminated pollen, infected foraging bees leaving spores, by queens, drifting bees and drones. The spores remain viable for up to 15 years or more in equipment and soil.

Clinical Symptoms
Dead larvae are usually chalky white and covered with fungus filaments (mycelia) that have a fluffy, cotton-like appearance. Sometimes the color may be mottled or blue-gray or black, which dries out and adheres to the cell wall. A slight non-objectionable odor may be present.

Diagnosis
Brood inspection and microscopic examination of samples.

Treatment and Prevention
No specific treatment is available
Keep strong colonies, requeen severely affected colonies and minimize colony dampness.

European Foulbrood (EFB)

European foulbrood is a bacterial brood disease caused by the bacterium **Melissococcus pluton [Streptococcus pluton].** It generally kills larvae that are two to four days old while they are still C-shaped in the bottom of cells. Unlike American foulbrood, most of the larvae die before their cells are capped. A spotty pattern of capped and uncapped cells develops only when EFB becomes serious. Occasionally, pupae die from the disease.

Clinical Symptoms and Lesions
The color changes are non-uniform ranging from a normal pearly-white to yellowish, then brown and finally grayish-black but can be blotchy or mottled. Infected larvae lose their plump appearance and look under-nourished. Their breathing tubes or trachea are visible as distinct white lines. The dead larvae form a thin, brown or blackish-brown scale which can be easily removed. EFB usually does not kill colonies, but a heavy infection will seriously affect population growth.

Diagnosis
The diagnosis of EFB is almost the same as that of AFB. The ropy test shows less stringiness or ropyness and the Holst Milk test does not result in a clearing of the solution.

Treatment and Prevention
Similar to American foulbrood
Requeening colonies with a young queen eliminates infection; it gives the colony a more prolific queen and it provides a time lag between brood cycles that allows the house bees to remove diseased larvae. Maintain young queen as a preventive method.

Nosema

Nosema is an adult bee disease caused by a spore-forming protozoan, **Nosema apis**. Infection occurs through the mouth, germinates in the gut and invades the hindgut of honeybee workers, queens and drones. Nosema increases during stress such as periods of long confinement, rapid brood buildup, nutritional imbalance and extreme weather; however, there is no specific season of the year where the disease is most prevalent.

Clinical Symptoms
Dysentery (diarrhea), bees may defecate in the hive or outside of the hive, decreased honey production, unhooked wings, distended abdomens and disoriented or paralyzed behavior are observed. If the queen is infected, loss of the colony may be observed; if the worker bees are infected, the production of royal jelly is inhibited subsequently retarding brood production and colony development.

Diagnosis
Remove the workers' head and examine the digestive tract microscopically. Numerous **Nosema apis** spores could also be microscopically observed.

Treatment and Prevention
Fumagillin 100 mg/gallon of sugar syrup (50% w/v with water), feed 2 gallons
Non drug treatment
Maintain sufficient honey during non-flowering season & vigorous queens.
Precaution: Protect the mix from exposure to direct sunlight.

Paralysis

Paralysis, a minor disease of adult honeybees, is usually associated with viruses. Two different viruses, Chronic bee Paralysis Virus (CPV) and Acute bee Paralysis Virus (APV), have been isolated from paralytic bees. Other suspected causes of paralysis include: pollen and nectar from such plants as buttercup, rhododendron, laurel and some species of basswood; pollen deficiencies during brood rearing in the early spring and consumption of fermented stored pollen.

Clinical Symptoms
Bees tremble uncontrollable and are unable to fly; lose the hair from their bodies and have a uniform dark, shiny or greasy appearance and are submissive to attack. Affected bees can be found at the top bars or frames next to the hive cover with wings extended or at the colony entrance, crawling up the sides of the hive and/or blades of grass around the hive and then tumbling to the ground. Healthy bees often tug at infected bees in an effort to drive them away from the hive.

Diagnosis
Clinical symptoms are indicative and usually one or two colonies in an apiary will show signs of the disease.

Treatment and Prevention
There is no specific treatment; however, a colony may recover from paralysis after a short time
If no recovery occurs, replace the colony completely with healthy colony.

Parasitic Mite Syndrome

Due to parasitic mites, honeybee colonies exhibit a variety of unusual and highly variable symptoms. Collectively these symptoms are called Parasitic Mite Syndrome (PMS). The disease is caused by several viruses at once. Although the circumstances under which the diseased brood appears are highly variable, all of the diseased colonies are infested with Varroa mites.

Clinical Symptoms and Diagnosis
Larvae affected with PMS die in the late larval or prepupal stage, stretched out in their cells with their heads slightly raised. The larvae appears dull, look deflated and later have gray or brownish spots. The cappings may be perforated or the cells may be uncapped completely by the bees. When the larval remains are stirred with a toothpick or small twig, they do not rope out but are globular (similar to EFB).

Treatment and Prevention
There is no specific drug treatment for the viruses but treat mites with miticides
To control mites, Fluvalinate 10%, one strip for each set of five frames of bees during low brood rearing, leave for at least 42 days and not more than 45 days.

Sacbrood

Sacbrood is a viral disease affecting the brood but of minor importance compared to the other brood diseases. It usually affects only a small percentage of the brood and the adult bees detect and remove infected larvae quickly. Both worker and drone larvae are infected by sacbrood. Death usually occurs after the cell is sealed and the larva has spun its cocoon. Pupae may be killed occasionally, but adult bees are immune to the disease.

Clinical Symptoms
Larvae that have died of sacbrood become like fluid filled sacs with heads pointing upwards. They change from pearly white to pale yellow and curls up as the body dries to a thin, dark brown scale lying along the bottom wall of the cell.

Diagnosis
Clinical symptoms: larvae with sacbrood disease are easily removed intact from the cells, the contents are watery and the scales are brittle and do not adhere to the wall.

Treatment and Prevention
There is no specific drug treatment
Maintain strong colonies and regular requeening

EXTERNAL PARASITES

Bee Louse (Braula coeca)

Braula coeca is a wingless bee louse that infests honeybees but cause little harm. The adults are small (slightly smaller than the head of straight pin) and reddish brown in color. When hungry they move to the bee's head near its mouthparts that stimulates the bees to regurgitate a drop of nectar. The bee louse feeds on it.

Clinical Symptoms
The louse lays its eggs on the capping of honey storage cells and upon hatching, the young burrow them. The adults can also burrow the comb. Braula adults are often found on queens but their damage to a colony of honey bees is limited.

Treatment and Prevention
Braula probably poses a minimal threat to the beekeeping industry because no economic loss can be attributed to its damage. There are very few recommendations for **Braula** control, largely because it is not considered a major honey bee pest.

Tracheal Mites

A second mite that infests honeybees is tracheal mite, **Acarapis woodi**. The adult mites penetrate the tracheal wall with their piercing mouthparts and feed on the bee blood. They are transmitted by drifting bees and swarms produced by infested colonies between apiaries.

Clinical Symptoms
The symptoms are not specific. The lives of affected adult bees become shortened; flight activity is affected and results in a large number of crawling bees that are unable to fly. They may exhibit dysentery and an excessive swarming tendency.

Diagnosis
Microscopic examination of honeybees' tracheae of infected bees is confirmatory. The samples are taken from bees crawling around the hive entrance and placed in 70% alcohol or methyl alcohol.

Treatment and Prevention
Menthol crystals, 50 g in a small screen packet (packets made of window screen or a similar mesh material, perforated plastic bags, coffee filters, cloth packets or other substitutes) per bee colony, placed on top bars of the frame of the brood nest for 4 to 6 weeks. It could be reused if kept at freezing temperature.
Note: All colonies in an apiary should receive mite treatment at the same time.

Varroa Mite

The Varroa mite **Varroa Jacobsoni**, is an external parasite that feeds on the hemolymph (blood) of adult bees, larvae and pupae. It is the most serious parasite of honeybees. Adult female mites are found on young drone and worker adults. They are mostly concentrated on the top of the thorax at the point where the wings attach, between the head and thorax, between the thorax and abdomen or between overlapping segments of the abdomen where the mites can easily penetrate the exoskeleton of the honeybees.
Varroa mites survive 2-3 months (5-8 months in fall) in the bees and only for five days outside bees.
Varroa mites have a definite preference for drone brood. The mites are spread by movement of honey bee colonies, i.e. migratory beekeeping, shipment of queens and package bees and movement of colonies for pollination or by beekeepers during normal apiary manipulations. Infestations are also spread as a result of drifting (especially drifting drones) and swarming bees.

Clinical Symptoms
Low-level varroa infestations: are difficult to detect. Medium to high infestations: spotty brood pattern and malformed worker and drone adults with deformed wings and small abdomens which often crawl and unable to fly. Colonies become severely debilitated as mite populations reach extremely high levels.

Diagnosis
Brush approximately 300 bees into a large empty clear plastic or glass jar. Close the lid and tap the jar to settle bees to the bottom and examine as follows. Add one- or two-second squirt of an ether-based aerosol starter fluid (the type used to start cars in cold weather) to kill the adult bees. Shake the jar of bees hard for 15-20 seconds and roll on its side. The mites stick to the sides. Another method is, uncap and examine sealed brood, especially drone brood preferably with uncapping fork. Examine with a small 10x hand lens to observe the mites.

Treatment and Prevention
Fluvalinate 10%, a synthetic pyrithroid strip, one strip for each set of five frames of bees during low brood rearing, leave for at least 42 days and not more than 45 days
Coumaphos strips are also applied as above.
Do not use if surplus honey is produced; the operator should wear gloves during handling, Or Do not treat more than twice a year.

Wax Moth

Larvae of the greater wax moth, **Galleria melonella** cause considerable damage to beeswax combs left unattended by bees particularly in weak or dead colonies and those placed in storage are subject to attack.
Wax moth larvae damage or destroy the combs by chewing through the beeswax cells as they feed on cocoons, cast skins and pollen. They spin silken galleries for protection from bees that will remove the wax moth larvae if they get the chance. Beeswax combs are often reduced.

Treatment and Prevention
Paradichlorobenzene crystals, in a small piece of paper between every fifth super in a stack repels wax moths, prohibit egg laying and kill young larvae
Maintain strong, healthy colonies, which can defend themselves against wax moths
Stored equipment and comb honey off colonies must be protected from this pest.
Caution: Mothballs and crystals made from naphthalene should not be used for wax moth control.

PREDATORS AND OTHER NON-INFECTIOUS DISEASES

Ants

Ants may enter bee colonies to search for food or establish nesting sites. Ants are typically found between the inner and outer covers of the hive and in pollen traps.

Control and Prevention
Once ants are established in a colony they are difficult to control
Maintain strong colonies and keep bottom boards raised off the ground and apply a fuel oil barrier
Remove brush rotten wood grass and weeds from around the colonies
Ant problem may also be reduced by allowing the bees access to the space between the inner and outer covers.

Beetle

The small hive beetle (**Athina tumida**) is a small (about one-third the size of a bee), black and hairy insect that could damage the bees comb and honey bee eggs.
The beetle lays its eggs on or near beeswax combs. The larvae consume pollen and comb but also will eat larval honeybees. This beetle is not considered a serious pest but in heavy infestations, it is blamed for the quick collapse of strong colonies. It also defecates in the honey and in some way alters the honey causing it to ferment and run out of the combs. Most vulnerable are weak hives with stored honey or full honey supers either in storage or above bee escapes.

Treatment and Prevention
There is no known treatment recommended except taking care of contamination with honey.

Mice

Adult mice move into bee colonies in the fall and usually nest in the corners of the lower hive body away from the winter cluster. Bee colonies located near fields or at the edge of wood lots where mice are common are especially vulnerable. Mice can successfully build a nest even in a strong colony. They move in and out of the colony while the bees are inactive and their nests furnish additional protection. Their activity may disturb the bees but the greater damage is to combs and equipment from their nest building.
Mice are a serious pest of stored combs and active honey bee colonies during the fall and rainy months. These rodents chew combs and frames to make room for building their nests. If the mouse urinates on combs and frames, the bees are reluctant to use the combs.

Control and Prevention
Protect the colony entrance from mice
Chase away any mice found inside a colony, then remove the nest and restrict the entrance
If comb chewing is extensive, replace the frames.

Skunks, Oppossums and Raccoons

In some localities, skunks are a serious threat to successful beekeeping, since they hamper the development of strong colonies. Being insectivorous (insect-eating), skunks will raid bee yards at night, consuming large numbers of bees.

To capture bees, skunks scratch at the hive entrance and guard bees come out to investigate the disturbance. A successful skunk will repeat the process several times and may feed at the hive entrance for an hour or more rapidly depleting bee population.

Diagnosis

Colonies visited by skunks may become defensive since skunks usually return night after night. Skunk predation can be detected by the front of the hive being scratched and muddy and the vegetation in front of the hive packed down or torn up. In addition skunks leave behind small piles of chewed-up bee parts. Skunk chews bees until all the juices are consumed and then spits out the remains. These remains resemble cuds of chewing tobacco. Opossums and raccoons sometimes attack an apiary in a similar manner as skunks do. The feces of these animals also contain large amount of honey bee exoskeletons since this material cannot be digested by animals.

Control and Prevention

Maintaining strong colonies is a partial deterrent to animal predation

Skunks and mice may be discouraged by screens or queen excluders attached to the hive entrance. These devices hamper the skunk's efforts to scratch at the front entrance and if it climbs up the screen over the entrance its belly becomes vulnerable to stings

Elevating the hives on stands (blocks bricks etc.) or fencing bee yard may also serve the same purpose.

Chilled Brood

This is caused by poor condition and cold weather, if larvae are underfed or if the brood covers a larger area than the bees can keep warm, some of the brood will die and turns gray. Such brood will be removed by bees from the cells as soon as the colony grows stronger and returns to normal.

Treatment and Prevention

There is no specific treatment

Work with the bees as little as possible when weather is cold

Replace combs in same order in which they were removed, especially if the colony is weak and it is early

Do not leave frames of brood standing outside the hive any longer than necessary. The frames of brood should not be left outside longer.

Poisoning in Honey Bees

Poisoning is a serious cause of morbidity and mortality. It is concerned with disease conditions caused by poisons or toxicants. A poison is a material that when it contacts or enters the body via ingestion, inhalation, dermal contact or injection, interferes with the normal biological processes and causes adverse health effects.

The nature of toxic responses depends not only on toxicant, but also route of exposure, duration and intensity of exposure and characteristics of the exposed individual, i.e. species, gender, age, pre-existing disease states, nutritional status and prior exposure to the agent or related compounds. The response may occur acutely soon after exposure or chronically much later and only after prolonged exposure. It is important to consider the individual poisons or classes of toxicants. This approach is used in organizing the various types.

Potential Factors for Honey Bee Damage

Many factors involving insecticide application affect the potential for honey bee losses. The more important ones are described below.

Plant Growth Stage: Severe bee poisoning most often results from spraying insecticides directly on flowering plants, either the crop itself or flowering weeds within its margins.

Relative Toxicity of the Chemical: Pesticides vary in their toxicity to honey bees. Most fungicides, herbicides and miticides (Kelthane, Comite) are relatively nontoxic to honey bees and can generally be used around them without serious harm.

Choice of Formulation: Different formulations, even of the same pesticide, often vary considerably in their toxicity to bees. Dust formulations are typically more hazardous than sprays because they are picked up on bee hairs. A wettable powder such as Sevin 80S would usually remain toxic in the field for a longer time than Sevin XLR Plus, an emulsifiable concentrate. However, granular insecticides are less hazardous to honey bees. Microencapsulated materials such as Penncap-M are particularly dangerous to use around bees because 1) the capsules have a special tendency to adhere to bees due to their size and electrostatic charge; and 2) when contaminated pollen collected by bees in treated fields is stored in the hive it remains toxic to bees for an extended period.
Residual Action: An insecticide which degrades within a few hours can generally be applied with minimum risk when bees are not actively foraging.
Drift: Sprays should not be applied if wind speed exceeds 10 mph and favors drift toward colonies.
Temperature: If temperatures following treatment are unusually low, insecticide residues can remain toxic to bees many times longer than if normal temperatures prevail.
Distance from Treated Fields: The most severely damaged colonies are usually those closest to fields where insecticides are being applied. However, during periods of pollen or nectar shortage, hives within five miles of the treated area can be affected.
Time of Application: Evening application of a short residual insecticide can greatly reduce any potential for bee damage.

Treatment and Prevention

Do not treat fields in bloom. Examine fields and field margins before spraying
Read the pesticide label. Choose short residual materials and low hazard formulations
Avoid spray drift. Adjust spray programs in relation to weather conditions
Apply insecticides when bees are not foraging. Contact local beekeepers and obtain locations of bee yards.

SECTION X

POISONING IN ANIMALS[10]

10 *Guide*

Numerical orders of the drugs (1, 2, 3 ...) in this document represent order of priority of choice. For S/E, D/I, D/F, C/I and W/P of drugs see Drug Index.

PLANT POISONING

Plant poisonings in farm animals have particular importance in countries like Ethiopia where extensive pastoral management is practiced.

Bracken Fern Poisoning

Bracken fern (Ptaquiloside) is a plant common in the humid tropics (and are common in open, acid woodlands, burned-over areas and open pastures in dry, sandy or gravelly soil). It is not palatable and only eaten when other forage is unavailable but some animals acquire an appetite for this plant. All parts, especially the roots cause acute poisoning related to thiamine deficiency in monogastric animals and to bone marrow depletion in ruminants. It has been reported in the highlands and rift valley areas of Ethiopia. The toxic effects appear to be cumulative and may require 1-3 months or even years to develop, depending on the species of animal, quantity consumed, time of year and other factors. The disease, also known as bovine enzootic hematuria, is usually fatal. Cattle over 3 years of age are most commonly affected and the disease has also been recorded in sheep and water buffalo exposed to infested pastures for periods exceeding 2 years.

Clinical Symptoms

Acute poisoning: In ruminants (especially cattle but sometimes sheep and goats): weakness, fever (41-43°C), reduced feed intake, digestive disorders, emaciation and haemoglobinuria followed by bleeding from nose and vagina, petechiae on the visible mucosae and edema along the intermadibular space may occur. From the excessive bleeding, cattle are anemic and can die within a week of showing signs. Young cattle may develop swelling in the larynx and have difficulty breathing.

The first sign in horses is weight loss after a few days on bracken. Later, weakness and gait abnormalities are present, which progress to staggering, hence "bracken staggers". Affected horses may stand with their legs widely placed and their back arched. Muscle tremors and weakness is apparent when the horses are forced to move.

Chronic poisoning: signs include intermittent hematuria, haemoglobinuria and animals try to urinate continuously.

Diagnosis

The acute hemorrhagic syndrome in cattle is distinctive but should be differentiated from other diseases showing hematuria.

Treatment and Prevention

Initial dose

Thiamine solution at 5 mg/kg, IV, q 3 h, followed by IM injection for several days.

Maintenance dose

Oral supplementation of thiamine for an additional 1-2 wk. or 100-200 mg, SC q 24 h for 6 days

Broad-spectrum antibiotics to prevent secondary bacterial infection

Blood transfusions 2-4 L blood, IV, from a donor not grazing bracken may be appropriate.

Crotalaria and Senecio Poisoning

Crotalaria and Senecio (**Pyrrolizidine alkaloides**) poisoning is mainly due to two pyrrolizidine producing plant species, namely: **Crotalaria** spp and **Senecio** spp. They are mainly found in damp grassland, especially in floodplains, depressions and along edges of swamps and rivers but also in deciduous bush land, roadsides and fields.

Outbreaks are in or enzootic to very large areas where toxic plants dominate pasture, mostly in cattle and horses. Pyrrolizidine alkaloid containing weed seeds in feed grains or dry plants in hay also lead to poisoning. Range of diseases associated with primary cumulative toxicosis and effect of metabolites.

The Crotalaria (rattlepods), the most common cause, is a nitrogen fixing leguminosa, which is normally avoided by livestock when grazing. However, cattle and horses may intentionally or unintentionally ingest the plant or seeds and succumb from poisoning. The seeds of **Crotalaria** species can also poison poultry.

Clinical Symptoms

Toxicity of **Senecio** is largely confined to the liver while **Crotalaria** will cause significant lung damage also. The signs may be acute or chronic. Chronic wasting, hepatic encephalopathy with blindness, head pressing, bouts of frenzy, jaundice, photosensitization and intravascular hemolysis, lusterless coat, weakness, icterus, ascites, diarrhea, tenesmus, prolapse of the rectum, lung emphysema, incoordination, aggressiveness, muscle spasm, refusal to eat and loss of weight are observed. Dry skin necrosis occurs in horses and the skin shrinks and pulls off. In the acute course, blood-tinged tar-like feces and tenesmus, bloating, blood-tinged nasal secretion, incoordination, colic and icterus with/ without haemoglobinuria are observed.

Diagnosis

Tentative diagnosis could be made by clinical symptoms.

Treatment and Prevention

Liver damage should be handled

Chronic cases: apply dextrose or fluid IV and Methionine

Do not allow livestock to graze on **Crotalaria and senecio plant** species.

Cyanide Poisoning

Cyanogenic glycosides accumulate in some plants (e.g. Johnson grass, Sudan grass, Sorghum etc.) whenever their cycle of vegetation is interrupted; for example when they wither after a sudden removal of water, after application of herbicides or under special climatic influences (Low soil moisture, high nitrogen and low phosphorus all favor HCN production). The glycosides are non-toxic but HCN may be liberated from them by β-glycosidase and lyase present in the plant tissue. Ruminal microorganisms also produce β-glycosidase. HCN interferes with the oxygen exchange from the lungs to the body tissues so that various tissues including the brain are starved-of oxygen and are consequently injured. Horses and pigs are much less susceptible to the glycosides because the acidity of the stomach in monogastric animals inactivates β-glycosidase and is not great enough to be associated with acid hydrolysis of the glycosides.

Clinical Symptoms

Acute poisoning - excitement followed by staggering, dyspnea with tachycardia, cattle stand with their mouths wide open; salivation and frothy foam, nystagmus, dilatation of the pupil, increased excitability, muscle tremors and finally tonic-clonic convulsions, vomiting and death.

Chronic poisoning - neonatal goiter or arthrogryposis, adult posterior ataxia, dysuria and frequent urination.

Diagnosis

Appropriate history, clinical symptoms, postmortem findings and demonstration of HCN in rumen (stomach) contents or other specimens support a diagnosis of cyanide poisoning.

Treatment and Prevention

Initial dose

Sodium nitrite 10% in distilled water or isotonic saline, 20 mg/kg, IV over 3-4min as an initial therapy followed by

Maintenance dose

Sodium thiosulfate 20%, ≥500 mg/kg, IV as a maintenance dose

Sodium nitrite therapy may be carefully repeated at 10 mg/kg, q 2-4 h or as needed.

Or

Sodium thiosulfate ≥500 mg/kg, IV **plus** 30 g/cow, PO, to detoxify any remaining HCN in the rumen; Methemoglobinemia may be induced in sodium nitrite injection identical to that produced by nitrate poisoning. Antidote: methylene blue, IV, at 4-22 mg/kg induces methaemoglobin.

Prevention

Pasture grasses (e.g., Sudan grass and sorghum-Sudan grass hybrids) should be large enough to before grazing.

Gossypol Poisoning

Gossypol poisoning, which is usually subacute to chronic, cumulative and sometimes insidious, follows consumption of cottonseed or cottonseed products that contain excess free gossypol, one of the several polyphenolic compounds found in the pigment glands of whole cottonseed. Gossypol is recognized for its toxic effect on cardiac, hepatic, pulmonary and reproductive systems. Whole cottonseed, cottonseed meal and cottonseed hulls are important feedstuffs. All animals are susceptible, but monogastrics, immature ruminants, and poultry appear to be affected most frequently. Pigs, guinea pigs and rabbits are reported to be sensitive. Dogs and cats appear to have intermediate sensitivity. Holstein calves are most sensitive of cattle breeds. Horses appear relatively unaffected.

Clinical Symptoms

It is mainly due to hepatotoxicity or secondary to congestive heart failure. Reproductive effects include reduced libido with decreased spermatogenesis in males and irregular cycling, luteolytic disruption of pregnancy and direct embryotoxicity in females. Other signs include weight loss, weakness, anorexia and increased susceptibility to stress. Young lambs, goats and calves may suffer cardiomyopathy and sudden death; if the course is more chronic, they may be depressed, anorectic and have pronounced dyspnea.

Diagnosis

History of dietary exposure to cottonseed meal or cottonseed products over a relatively long period. Clinical symptoms, absence of response to antibiotic therapy and presence of significant free gossypol in the diet.

Treatment and Prevention

There is no effective treatment

Prevention

If gossypol toxicity is suspect, all cottonseed products should be removed from the diet immediately.

Lantana Camara Poisoning

Lantana is a shrub that grows as scrambling and climbing vine. **Lantana camara** toxicosis can affect cattle, sheep, goats, horses and buffaloes. All lantana should be treated as poisonous to stock. Red flowered varieties are thought to be the most toxic but some white and pink flowered varieties can also be highly toxic. Significant lantana toxins are the triterpene acids, lantadene A (rehmannic acid), lantadene B and their reduced forms.

Clinical Symptoms

Symptoms of Lantana camara poisoning depend on the amount and type consumed and intensity of light to which animals are exposed. Signs can appear after one feed and in acute cases, within 24 hours. Early symptoms include, head swaying side to side, loss of appetite, constipation and frequent urination. After a day or two the eyes and the skin of the nose and mouth start yellowing with jaundice and the muzzle becomes dry and warm. The eyes may become inflamed and have a slight discharge. The animals become increasingly sensitive to light. Finally the muzzle becomes inflamed, moist and painful (pink nose).

Diagnosis

It is based on clinical symptoms.

Treatment and Prevention

Treating skin damage with antibiotics and sunscreens; other drugs can provide relief

Drenching with a slurry (2 - 2.5 kg activated charcoal in 20-30 liters of electrolyte replacement solution for cattle; 500 g in 4 liters for sheep and goats) stat and if required repeat after 24 hours.

Bentonite 5gm/kg can be substituted for activated charcoal but it will take longer days to respond.

Prevention
Cut and paint the plants with **Glyphosate** 360g/l or spray the entire plant with it
Biological control e.g. leaf-mining beetle feed on the upper leaf surfaces
Basal dark spraying and cut stump methods.

Sweet Clover Poisoning

Sweet clover **(Dicoumarol)** poisoning, is an insidious hemorrhagic disease, occurs in animals that consume toxic quantities of spoiled sweet clover (**Melilotus officinalis** and **M. alba**) hay or silage resulting from toxic dicoumarol that interferes with blood coagulation. Coumarin, ferulenol and melilotin, normal constituents of some plants are converted to dicoumarol in the process of infection by molds of the cut plant during the making or storage of hay or silage. In cattle, concentrations of 60 to 70 ppm results in clinical symptoms after 17 to 23 days.

Clinical Symptoms
Hemorrhages that result from faulty blood coagulation is common. These include stiffness and lameness, hematomas, epistaxis or gastrointestinal bleeding. Massive hemorrhage or bleeding after injury, surgery or parturition is observed. Pallor, weakness especially in newborn calves. Abortion or stillbirth may occur in pregnant animals.

Diagnosis
Determine blood-clotting time or demonstrate reduced prothrombin content of plasma. High dicoumarol content of feed.

Treatment and Prevention
There is no effective treatment
Avoid feeding moldy sweet clover hay or ensilage; dilute with other feed
Blood transfusion: 2-10L of whole blood per 450 kg, IV (from an animal not being fed sweet clover) as a first line therapy. Repeat it if necessary. **Plus**
Vitamin K1 (phytonadione), 1mg/kg, IM, q 12 h for 2days, with a small-bore needle.

Datura Poisoning

Datura appears to be the main (species) genus involved in poisoning by tropane alkaloids. Although **Datura** poisoning is possible in most animal species, it is particularly a practical problem in equines. The reason for this is that equines are particularly sensitive to atropine and related tropane alkaloids. Being annuals, **Datura** weeds invade and contaminate especially annual crops like corn (maize), sorghum, soybeans and linseed. The weeds ripen with crop and weed seed contaminates the finally harvested grain. With annual grass crops, such as teff, the equally toxic immature weeds are cut and dried with hay.

Clinical Symptoms
In the horse, additionally, the motility of the large intestine is seriously affected and either an acute paralytic ileus results with consequent death from acute complications like torsion, strangulation or tympany or in more chronic cases recalcitrant impaction colic develops. The signs recorded are anorexia, hyper excitability, staggers, muscular spasms, frequent urination and mydriasis and impaired vision progressing to convulsive seizures, rigor and coma preceding death. Cattle are probably susceptible to acute tropane alkaloid intoxication but it seems to be very dose-specific and no cumulative effects are evident. The signs noted in sheep and goat species include reduced water intake, ataxia, intermittent hyperesthesia, tremors, drowsiness and recumbence. Pigs are also susceptible.

Diagnosis
History of dietary exposure to **datura** spp or related plants over a relatively long period; clinical symptoms; and presence of the toxic plant in diet.

Treatment and Prevention
Therapy can include lavage by nasogastric tube, laxatives ($MgSO_4$ 1-2gm/kg as 5% solution PO as antidote or technical oil),
Cisapride PO, 2-8gm/kg or IV, 0.1mg/kg
Analgesic such as flunixin 1.1mg/kg when required.

Nitrate and Nitrite Poisoning

There is considerable variation between species in their susceptibility to nitrite intake poisoning, pigs being most susceptible, followed by cattle, sheep and horses in that order. But pigs and horses are not susceptible to nitrite poisoning from excessive intake of nitrates as they can't convert nitrite to ammonia.
The common sources of nitrate for farm animals include: certain weeds, cereal crops used as pasture, e.g. immature green oats, barley, wheat and rye or hay or green fodder e.g. Sudan grass, corn. Oat hay may contain 3-7% nitrate very heavy growths of rye-grass (**Lolium** spp.) in pastures freshly pulled mangels. Turnip tops may contain 8% nitrate and sugar beet tops and rape have been associated with nitrite poisoning.

Clinical Symptoms
In animals poisoned by nitrate there is salivation, abdominal pain, diarrhea and abdominal pain. It is also worthwhile submitting a sample of urine, as nitrite appears to pass unchanged into the urine.

Diagnosis
History of dietary exposure, clinical symptoms.

Treatment and Prevention
Methylene blue 1%, 4-22 mg/kg, slow IV, in distilled water or isotonic saline
Rumen lavage with cold water and antibiotics may stop the continuing microbial production of nitrite.

Prevention
Ruminants likely to be exposed to nitrites or nitrates should receive adequate carbohydrate in their diet and traveling or hungry animals should not be allowed access to dangerous plants.

MYCOTOXIN POISONING

A high moisture content of stored feeds is conducive to fungal growth.

Aflatoxin Poisoning

Aflatoxins are produced by toxigenic strains of **Aspergillus flavus** and **A. parasiticus** on peanuts, soybeans, corn (maize), and other cereals either in the field or during storage when moisture content and temperatures are sufficiently high for mold growth. They are related chemically to, and probably derived from, dicoumarin compounds. In cows, goat and camel fed with feed contaminated by aflatoxin B1 the metabolite aflatoxin M1 will be excreted. Aflatoxin M1 is toxic or carcinogenic to human.

Clinical Symptoms
In acute outbreaks, deaths occur after a short period of inappetence; other acute signs include vomiting, depression, hemorrhage and icterus. Subacute outbreaks are more usual, with unthriftiness, weakness, anorexia, reduced growth and feed efficiency and occasional sudden deaths. Affected animals usually die within 48 hours, calves in the 3-6 month group being most susceptible. Aflatoxicosis is also reputed to interfere with clotting of blood in cattle, leading to development of hematomas.

Diagnosis
History of dietary exposure and clinical symptoms; confirmatory diagnosis is based on the detection of aflatoxin in the feed and blood serum and the characteristic gross and histopathological findings in the liver and nervous tissue. Acutely affected animals have increases in liver enzymes (alkaline phosphatase, AST or ALT), bilirubin, serum bile acids and prothrombin time.

Treatment and Prevention

Symptomatic treatment of hepatic insufficiency is all that can be attempted

Contaminated feeds should be avoided by monitoring batches for aflatoxin content. Dietary levels of aflatoxin (in ppb) generally tolerated are ≤50 in young poultry, ≤100 in adult poultry, ≤50 in weaned pigs, ≤200 in finishing pigs, <50 in dogs, <100 in calves, and <300 in cattle

Acceptable regulatory values in milk may range from 0.05 ppb to 0.5 ppb in different countries

Hydrated sodium calcium aluminosilicates (HSCAS), which binds on it and reduce the effects of aflatoxin when fed to pigs or poultry at 10 lb/ton (5 kg/ton). HSCAS can reduce Aflatoxin M1 by 50% but it can't eliminate M1 fed with Aflatoxin B1.

Ergotism

Poisoning associated with the ingestion of large quantities of the naturally occurring ergots of **Claviceps purpurea**. Poisoning is most likely to occur during or after warm, wet seasons which favor the growth of the fungus. Ergotism occurs commonly only in cattle and usually in stall-fed animals feeding on heavily contaminated grain over a considerable period of time. Other species are not usually exposed to the infested grain. Ergot-infested pasture may be associated with the disease and the toxicity is preserved through the ensiling process.

Clinical Symptoms

The extremities, particularly the lower part of the hind limbs, the tail and ears are affected. There is reddening, swelling, coldness, loss of hair or wool and lack of sensation of the parts initially, followed by the development of a blue-black color, dryness of the skin and its separation from normal tissues. Affected cows have temperatures of 41-42°C, dyspnea and hypersalivation. Milk production and growth rate are depressed and morbidity is about 100%. The syndrome occurs in hot weather conditions when affected animals seek water or shade, but exposure to sunlight under normal conditions of air temperature and humidity can be enough to be associated with clinical symptoms.

Diagnosis

History of dietary exposure and clinical symptoms. Confirmatory diagnosis depends on a positive assay of ergot alkaloids (200–600 ppb) in feed or tissues.

Treatment and Prevention

The infested grain should be withdrawn from the ration immediately

In horses, parenteral use of the dopamine D2 antagonist domperidone (1.1 mg/kg, PO, q 12 h for 10–14 days) is effective in prevention of agalactia from ergot alkaloids.

Control and Prophylaxis

Heavily ergotized grain or pasture fields should not be used for animal feeding. They may be grazed if they are first mowed with mower blade set high to remove seed heads. Feed should not contain more than 0.1% of ergot-infested heads and concentrations of ergot alkaloids should be <100 ppm in the total diet. It is not safe to feed ergot-infested feeds to pregnant females.

HERBICIDE POISONING

Herbicides are plant growth inhibitor chemicals which, when applied in the field may accidentally poison all livestock species and small animals. Animals are exposed to poisoning when feed and water is contaminated, if animals are allowed to graze in recently sprayed fields or when treatment of poisonous plants with herbicide leads to accumulation of harmful amounts of nitrate, cyanogenic glycosides or other toxic plant constituents or if normally unpalatable toxic plants are rendered palatable. The most common herbicides are the following

Amides (Bensulide, Propanil)

A lethal dose of bensulide for dogs is ~200 mg/kg. The prominent clinical sign is anorexia; other signs and lesions are not definitive. Hemolysis, methemoglobinemia and immunotoxicity have occurred after experimental exposure to propanil. The half-life in catfish is ≥15 days.

Ammonium Sulfamate

Ruminants apparently can metabolize this chemical to some extent. Large doses (>1.5 g/kg b.w) induce ammonia poisoning in ruminants and the rumen becomes highly basic.

Treatment and Prevention
Weak acetic acid (vinegar), PO
Supply large amount of water.

Arsenicals

Arsenicals are used as desiccants or defoliants on cotton and residues of cotton harvest fed to cattle may contain toxic amounts of arsenic. Single toxic oral doses for cattle and sheep are 22-55 mg/kg b.w or toxicity may occur after consumption of smaller doses on successive days.

Treatment and Prevention
Large animals: Dimercaprol, 3 mg/kg, PO
Small animals: Dimercaprol Day 1-2: 10-12 mg/kg/day divided q6hr. deep IM for 2 days, Day 3: 5-6 mg/kg/day divided q12hr. deep IM for 1 days, Day 4-14: 2.5-3 mg/kg deep IM q Day for 11 days.

Bipyridyl Compounds (Diquat, Paraquat)

Skin irritation and corneal opacity occur on external exposure to these chemicals and inhalation is dangerous. Toxicity may occur if animals drink water from an old diquat containers.
Signs of diquat poisoning include anorexia, gastritis, GI distension and severe loss of water into GI tract lumen and in severe cases renal impairment, CNS excitement and convulsions. Additional signs in paraquat poisoning include incoordination and respiratory difficulty.

Treatment and Prevention
Vitamin E and selenium **plus** diuretics such as Mannitol 20% IV infusion at a maximum of 0.5 mg/kg/hr. and continue with 5% in Ringer's solution;
Furosemide, 1-5 mg/kg, IM, or IV.

Dinitro Compounds (dinoseb, 2, 4-dinitrophenol, dinitrocresol, binapacryl, DNOC)

The dinitrophenol and dinitrocresol compounds are highly toxic to all classes of animals; e.g., LD50 20-56 mg/kg b.wt. Poisoning occurs after feeding sprayed herbage or exposure of the skin or inhalation. Clinical symptoms include fever, dyspnea, acidosis, tachycardia and convulsions, followed by coma and death with a rapid onset of rigor mortis. Cataracts occur in chronic dinitrophenol intoxication. Skin, conjunctiva and hair may be stained yellow.

Treatment and Prevention
Effective antidote is not known.
Control and prophylaxis
Diazepam + Cool IV fluids + Vitamin A
Emetics or gastric lavage or activated charcoal.
Caution: Phenothiazine tranquilizers are contraindicated.
Atropine sulfate, aspirin, and antipyretics should not be used.

Triazine Compounds (atrazine, cyanazine, prometryn, metribuzin, simazine)

Although these herbicides are widely used, incidents of poisoning are uncommon. Toxic effects in sheep occur at doses of 500 mg of simazine/kg or 30 mg of atrazine/kg for 36-60 days. Generally, single doses >100-200 mg/kg body wt can be detrimental.

Treatment and Prevention
The same as treatment, control and prophylaxis for 2, 4-D toxicity.

INSECTICIDE AND ACARICIDE TOXICITY

Pesticide exposure, no matter how brief or small, results in some of the compound being absorbed and perhaps stored. Repeated short exposures may eventually result in intoxication. Thus every precaution should be taken to minimize human exposure.

CARBAMATE INSECTICIDES

Carbaryl

The oral LD50 in rats is 307 mg/kg body wt and >500 mg/kg, dermally. A 2% spray is nontoxic to calves; 4% is nontoxic to mature cattle when applied dermally.

Carbofuran

The oral LD50 is 8 mg/kg body wt in rats and 19 mg/kg in dogs. The minimum toxic dose in cattle and sheep is 4.5 mg/kg, becoming lethal at 18 and 9 mg/kg, respectively. Pigs have been poisoned after drinking water contaminated by this compound.

Methomyl

The oral LD50 in rats is 17 mg/kg body wt. Cattle have been reported to be poisoned after consumption of forage inadvertently sprayed with this compound.

Propoxur

The oral LD50 is 95 mg/kg body wt. in rats and >800 mg/kg in goats. Clinical signs include hypersalivation, gastrointestinal hypermotility, abdominal cramping, vomiting, diarrhea, sweating, dyspnea, cyanosis, miosis, muscle fasciculations (in extreme cases, tetany followed by weakness and paralysis) and convulsions. Death usually results from hypoxia due to bronchoconstriction. Diagnosis usually depends on history of exposure to a particular carbamate and response to atropine therapy.

Treatment and Prevention
Atropine sulfate for cattle and sheep: 0.61 mg/kg, ¼th given IV and the remainder dose given SC; repeat as needed. **Plus**
Alloxine, a cholinesterase reactivating compound, greatly improves recovery from carbamate poisoning if given with atropine.

Organophosphates

Organophosphates (OP) are insecticides used in animal and plant production. They vary greatly in toxicity, residue levels, and excretion. However, they produce little or no tissue and environmental residues.

Azinphos-methyl (or -ethyl)

The maximum nontoxic oral dose is 0.44 mg/kg body wt. for calves, 2.2 mg/kg for cattle and goats, and 4.8 mg/kg for sheep.

Carbophenothion

Dairy calves <2 wk. of age sprayed with water-based formulations show poisoning at 0.05% and higher concentrations. Adult cattle have been poisoned by concentrations of 1%.

Chlorfenvinphos

Adult cattle can be poisoned by ≥0.5% sprays, while young calves can be poisoned if the concentration is raised to 2%. The minimum oral toxic dose appears to be ~22 mg/kg for cattle of all ages.

Chlorpyrifos

The oral LD50 is 500 mg/kg b.w. in goats and 97 mg/kg in rats. In comparison with calves, steers and cows, bulls (particularly of the exotic breeds) are highly susceptible to a single dose of chlorpyrifos.

Coumaphos

Coumaphos is used against cattle grubs and a number of other ectoparasites and for treatment of premises. At 0.5% of spray, it may be safely used on adult cattle, horses and pigs; in young calves and all ages of sheep and goats the concentration must not exceed 0.25%; 0.5% concentrations may be lethal. Adult cattle may show mild signs at 1% concentrations.

Crotoxyphos

Crotoxyphos is of rather low toxicity; however, European breeds seem to be less susceptible; sheep, goats and pigs all tolerate sprays containing crotoxyphos at 0.5% levels or higher. The toxic dose appears to be in the 2% range, except for in Brahman cattle, in which 0.144%-0.3% may be toxic.

Diazinon

When sprayed, young calves appear to tolerate 0.05% but are poisoned by 0.1% concentrations. Toxic levels are as follows: calves 0.88 mg/kg, PO, adult cattle 22 mg/kg; Sheep 26 mg/kg PO.

Dichlorvos

Dichlorvos has many uses on both plants and animals. Because it is rapidly metabolized and excreted, residues in meat and milk are not a problem if label directions are followed. Toxic levels are as follows: young calves, 10 mg/kg; sheep and horses, 25 mg/kg PO. Flea collars that contain this compound may cause skin reactions in some pets.

Dimethoate

Toxic levels are as follows: calves 48 mg/kg b.w., cattle above 1 year 22 mg/kg b.w., horses 60-80 mg/kg, PO. When applied topically, 1% sprays have been tolerated by calves, cattle and adult sheep.

Dioxathion

Concentrations ≤0.15% are generally used on animals. Dioxathion has killed young calves at 8.8 mg/kg, PO, and produced intoxication at 4.4 mg/kg.

Disulfoton

The maximum nontoxic oral dose is 0.88 mg/kg body wt. for young calves, 2.2 mg/kg for cattle and goats, and 4.8 mg/kg for sheep. Intoxication has occurred in cattle after consumption of harvested forages previously sprayed with this insecticide.

Fenthion

The minimum toxic dose for cattle is 25 mg/kg body wt, PO; 50 mg/kg, PO, is lethal to sheep.

Malathion

Malathion is one of the safest OP. Young calves tolerate 0.5% sprays, but 1% sprays are toxic; adult cattle tolerate 2% sprays. Given PO, Malathion is toxic at 100 mg/kg; young calves tolerate 11 mg/kg but are poisoned by 22 mg/kg. Malathion is excreted in the milk of cattle. Thus the public health risk is high.

Methyl Parathion

The LD50 in rats from a single oral dose is 9-25 mg/kg body wt compared with 3-13 mg/kg for ethyl parathion. Microencapsulation of this compound decreases its toxicity and the lethal dose in cattle has thus been increased from a 0.5% spray to a 2% spray.

Parathion

Parathion is widely used for control of plant pests. Toxic levels in animals are as follows: sprays in young calves, at a 0.02% concentration and occasional transitory signs at 0.01%; sheep at 22 mg/kg b.w, PO, but not at 11 mg/kg; young dairy calves, 0.44 mg/kg, old cattle, 44 mg/kg b.w. Parathion is used extensively in the control of mosquitoes and

insects in orchards and on truck crops. Normal application on land does not present particular hazard to livestock. Care must be taken to prevent accidental exposure. Parathion is not stored in animal tissues in appreciable amounts.

Phorate

The minimum toxic dose is 0.25 mg/kg body wt in calves, 0.75 mg/kg in sheep, and 1 mg/kg in cattle. The oral LD50 in rats is 1-4 mg/kg.

Phosmet

Toxic levels in animals is as follows: adult cattle and calves, 25 mg/kg b.w., sheep, 50mg/kg b.w.

Ronnel (Fenchlorphos)

Toxic levels in animals are as follows: cattle, 132 mg/kg b.w. and mild signs are observed; severe signs occur at ~440 mg/kg; sheep, 400 mg/kg. Sprays at 2.5% do not produce poisoning of cattle, young dairy calves or sheep. Poisoning usually occurs in two stages. The animal first becomes rather weak and although moving about normally, may be placid. Diarrhea, often flecked with blood, may also be seen. Salivation and dyspnea appear if the dose is high but probably not at the lower dosages. Blood cholinesterase activity declines slowly over 5-7 days.

Public health risk: Ronnel produces residues in meat and milk; therefore, strict adherence to label restrictions is essential.

Treatment and prevention

Activated charcoal to remove residues already ingested.

OTHER INSECTICIDES

Ruelene

Ruelene is active both as a systemic and contact insecticide in livestock; it has some anthelmintic activity and is of rather low toxicity. Toxic levels in animals are as follows: dairy calves, ≥44 mg/kg b.w, PO; adult cattle 88 mg/kg; Sheep, 176 mg/kg show moderate toxicity; Angora goats are about twice as sensitive. Toxic levels in pigs is 11 mg/kg and horses 44 mg/kg. Most livestock tolerate a 2% topical spray.

Terbufos

This soil insecticide is used to control corn rootworms. Toxic level in cattle and sheep, is ~1.5 mg/kg b.w., PO.

Tetrachlorvinphos

Toxic level in pigs is 100 mg/kg.

Tetraethyl Pyrophosphate (TEPP)

TEPP is one of the most acutely toxic of all insecticides, 0.33% TEPP emulsion will kill cattle.

Trichlorfon

As a spray, trichlorfon at a 1% concentration is tolerated by adult cattle. When given PO, it is tolerated by young dairy calves at 4.4 mg/kg body wt. but produces poisoning at 8.8 mg/kg. Adult cattle, sheep and horses appear to tolerate 44 mg/kg. Toxic levels: 88 mg/kg produces poisoning. Dogs were unaffected when fed 1000 ppm of trichlorfon for 4 months. Trichlorfon is metabolized rapidly.

Clinical Symptoms of Organophosphate Poisoning

In general, organophosphate (OP) pesticides have a narrow margin of safety, and the dose-response curve is quite steep. The signs are mainly cholinergic overstimulation and are grouped as follows:

Muscarinic effects: hypersalivation, miosis, frequent urination, diarrhea, vomiting, colic and dyspnea due to increased bronchial secretions and bronchoconstriction. They appear first.

Nicotinic effects: muscle fasciculation and weakness.

The central effects: nervousness, ataxia, apprehension and seizure activity. Cattle and sheep commonly show severe depression. Stimulation in dogs and cats usually progresses to convulsions. Some OP (e.g. amidothioates) do not enter the brain easily, so that CNS signs are mild. Onset of signs after exposure is usually within hours but may be delayed for ≥2 days. Severity and course of intoxication is influenced principally by the dosage and route of exposure. In acute poisoning, the primary clinical symptoms may be respiratory distress and collapse followed by death due to respiratory muscle paralysis.

Diagnosis

Lowered cholinesterase activity in blood and brain, but does not correlate with severity. Results of analyses performed after exposure may be negative because OP do not remain long as the parent compound in tissues. Chlorinated OP compounds appear to have greater potential for tissue residue. Frozen stomach and rumen samples should be analyzed for the pesticide because OP is generally more stable in acids.

Treatment and Prevention

Atropine sulfate (**Muscarinic blocking agents)** Dogs and cats, 0.2-2 mg/kg q 3-6 dose IV, the remainder IM or SC, repeated as needed. Stop treatment when pupils are dilated. Horses and pigs, 0.1-0.2 mg/kg, IV, q 10 min; Cattle and sheep, 0.6-1 mg/kg, ⅓rd of the dose IV, the remainder IM or SC, repeated as needed. Stop treatment when pupils are dilated. **Plus**

Diazepam to reduce seizures

Atropine is combined with the cholinesterase reactivators

2-pyridine aldoxime methchloride (2-PAM, pralidoxime chloride) 10%, 20-50 mg/kg, IM or by very slow IV injection, repeated as needed followed by Oxime instituted within 24-48 hr because the response to 2-PAM decreases shortly after use.

Other treatments include

Emetics, cathartics and adsorbants to decrease further absorption

If the OP compound was applied dermally, wash the body with detergent and water; if through the mouth and within less than 2 hours, give emetic drugs (it is contraindicated for depressed animals);

Oral administration of mineral oil decreases absorption of pesticide from the GI tract.

Activated charcoal, 3-6 g/kg as water slurry, adsorbs OP and helps elimination in the feces; particularly recommended in cattle

Artificial respiration or administration of oxygen is advantageous.

Note: Phenothiazine tranquilizers should be avoided. Succinylcholine should not be used for at least 10 days after OP exposure.

RODENTICIDE POISONING

Anticoagulant Toxicants

Brodifacoum is the most commonly used and is the most toxic rodenticide, which has replaced the much safer warfarin-based coumarins.

Zinc Phosphide and Aluminum Phosphide

Both chemicals are rodenticides used in baits of bread, buns, soaked wheat, damp, rolled oats or sugar at concentrations of 5%. Aluminum phospide is also used as grain fumigant for insect and rodent control. Both are unstable in water or acid.

Clinical Symptoms

Zinc phosphide causes emesis, large doses cause death within 3-5 hours. Anorexia, lethargy, and rapid deep breathing are observed. In dogs, abdominal pain, ataxia, weakness and recumbency may follow. Acidosis, hyperesthesia, seizures, hepatic and renal damage and acetolene (garlic, rotten fish) odor of breath are observed. Abdominal pain, colic in horses, and bloat in cattle.

Diagnosis

It depends on clinical symptoms, an acetylene odor to the breath and opportunity for or better, evidence of exposure to zinc phosphide; frozen vomitus or gastric lavage fluid and other organs including stomach contents, liver and kidney should be examined.

Treatment and Prevention
Emesis followed by Aluminum plus Magnesium hydroxide gel, PO
Activated charcoal plus sorbitol, PO
Sodium bicarbonate IV.

Strychnine Poisoning

Strychnine is a bitter tasting alkaloid used as pesticides to control gophers, moles and rats. Its use is restricted in Ethiopia, but used for the same purpose under a veterinarian's supervision for the control of stray dogs. Accidental poisoning might occur to animals and humans. The onset occurs from 10 minutes to 3 hours.

Clinical Symptoms
The signs include anxiety, stiffness, violent tetanic seizures, saw- stance, opisthotonus, persistent rigid extension of all four limbs, non-muscular movement and anoxia due to rigidity of muscles.

Diagnosis
It depends on clinical symptoms and history of exposure and analysis of baits.

Treatment and Prevention
Non drug treatment
Artificial respiration.
Drug treatment
Pentobarbital intubation
Methocarbamol, 150 mg/kg, IV, repeat doses of 90 mg/kg as needed
Inhalation anesthetics for 48 hours. Ketamine and morphine are contraindicated and should not be given
Enterogastric lavage: activated charcoal and a saline or osmotic cathartic
Fluids: forced diuretics with 5% mannitol in 0.9% normal saline at 7 mg/kg/hr.
Ammonium chloride, 100 mg/kg, q12h, PO; contraindicated if the acidosis is from exertion or respiratory failure.
Prophylaxis
Extreme care should be taken when handling strychnine.

INDUSTRIAL CHEMICAL POISONING

Many industrial chemicals are toxic if ingested or even exposed to the skin. The most common known to cause toxicity in animals are listed below.

Lead Poisoning

In veterinary medicine, lead poisoning is most common in dogs and cattle but limited in other species due to reduced accessibility and selective feeding habits. The sources of lead include used oil and battery, paint, linoleum, grease, lead weights, lead shot and contaminated foliage growing near smelters or along roadsides.

Clinical Symptoms
Cattle: acute course characterized by ataxia, blindness, salivation, spastic twitching of eyelids, jaw champing, **bruxism,** muscle tremors and convulsions.
Sheep and old cattle: Subacute course characterized by anorexia, rumen stasis, colic, dullness and transient constipation, frequently followed by diarrhea, blindness, head pressing, bruxism, hyperesthesia and incoordination.

Horses: chronic course characterized by weight loss, depression, weakness, colic, diarrhea, laryngeal or pharyngeal paralysis (roaring) and dysphagia that frequently results in aspiration pneumonia.
Avian species: signs include anorexia, ataxia, loss of condition, wing and leg weakness and anemia.
Dogs: show gastrointestinal intestinal abnormalities, including anorexia, colic, emesis and diarrhea or constipation. Anxiety, hysterical barking, jaw champing, salivation, blindness, ataxia, muscle spasms, opisthotonus and convulsions may develop. CNS depression rather than CNS excitation may be evident in some dogs.

Diagnosis

Lead levels in various tissues and blood, hematologic abnormalities, δ-aminolevulinic acid and free erythrocyte protoporphyrin levels are confirmatory.

Treatment and Prevention

Non drug treatment

Rumenotomy may be useful to remove lead from the GI tract.

Drug treatment

Calcium disodium edetate (Ca EDTA), 110 mg/kg/day, IV or SC, divided into two treatments q 24h for 3 days; then repeated 2 days later
In dogs, similar treatment divided into four treatments; SC in 5% dextrose for 2-5 days; after one week rest, an additional 5-days treatment may be required if Clinical Symptoms persist
Thiamine, 2-4 mg/kg/day, SC, alleviates clinical manifestations and reduces tissue deposition of lead
Combined Ca-EDTA and thiamine treatment appears to produce the most beneficial response
D-penicillamine, 110 mg/kg/day, PO to dogs for 2 weeks; because of emesis and anorexia, it not recommended for livestock
Magnesium sulfate, 400 mg/kg, PO
Barbiturates or tranquilizers may be indicated to control convulsions.

Nitrate and Nitrite Poisoning

Nitrates and nitrites are used in pickling and curing brines for preserving meats, certain machine oils and antirust tablets, gunpowder and explosives and fertilizers.
Many species are susceptible to nitrate and nitrite poisoning, but cattle are affected most frequently. Ruminants are especially vulnerable because the ruminal flora reduces nitrate to ammonia, with nitrite, which is ~10 times more toxic than nitrate, as an intermediate product. Ingested nitrates may directly irritate the GI mucosa and produce abdominal pain and diarrhea.

Clinical Symptoms

Rapid, weak heartbeat, subnormal body temperature, muscular tremors, weakness and ataxia are early signs of toxicosis. Brown, cyanotic mucous membranes develop rapidly. Dyspnea, tachypnea, anxiety and frequent urination are common. Some monogastric animals, usually because of excess nitrate exposure from non-plant sources, exhibit salivation, vomiting, diarrhea, abdominal pain and gastric hemorrhage. Affected animals may die suddenly without appearing ill, in terminal anoxic convulsions within 1 hour or after a clinical course of 12-24 hours or longer. Some animals that develop marked dyspnea recover but then develop interstitial pulmonary emphysema and continue to suffer respiratory distress; most of these recover fully within 10-14 days. Abortion and stillbirths may occur in some cattle.

Diagnosis

Excess nitrate exposure can be assessed by laboratory analysis for nitrate in both antemortem and postmortem specimens.

Treatment and Prevention

Methylene blue 1%, 4-22 mg/kg, slow IV, in distilled water or isotonic saline
Rumen lavage with cold water and antibiotics may stop the continuing microbial production of nitrite
Feeding grain with high-nitrate forages may reduce nitrite production
High-nitrate forages may also be harvested and stored as ensilage rather than dried hay or green chop.

ABBREVIATIONS

Abbreviations in prescription writing			
ante	before	PO	by mouth
ad lib	as much as desired	Prn	as needed
amp	ampule	Qd	every day
aq	Water	q.i.d.	4 times per day
b.i.d.	twice a day	q.o.d.	every other day
c	With	q 4 h	every four hours
caps	capsules	Qs	as much as suffice
circ	about, approx.	Rep	repeat
et	And	S	without
ext	Extract	sid	once per day
		sig	instruction/label
gr	Grain	solv	dissolve
gtt	Drop	sol'n	solution
im	intramuscularly	sc	subcutaneously
iv	intravenously	ss	One half
id	the same	stat	medication given in one dose
m	mix	susp	suspension
m^2	square meter	tabs	tablets
mg	milligram	tbs	tablespoon
non rep	do not repeat	tid	three times a day
O.D.	right eye	tr	tincture
m	every morning	tsp	teaspoon
n	every evening	U	both eyes
O.S	left eye	mg	microgram
pc	after meals		

ACRONYM

AAU	Addis Ababa University
AHA	Animal Health Assistants
AHS	African Horse Sickness
ALIPB	Aklilu Lema Institute of Patho Biology
AMR	Antimicrobial Resistance
BES	Balanced Electrolyte Solution
BPM	Beats per Minute
BVD	Bovine Viral Diarrhea
C/I	Contraindication
CBPP	Contagious Bovine Pleuropneumonia
CCPP	Contagious Caprine Pleuropneumonia
D/F	Dosage Forms
D/I	Drug interaction
DACA	Drug Administration and Control Authority
FMD	Foot and Mouth Disease
IB	Infectious Bronchitis
IBD	Infectious Bursal Disease
LDA	Left Displaced Abomasum
LSD	Lumpy Skin Disease
NSAID	Non-Steroidal Anti-Inflammatory Drug
NAHDIC	National Animal Health Diagnostic and Investigation Center
MOA	Ministery of Agriculture
OIE	World Organization for Animal Health
P/O	Per os
PCR	Polymerase Chain Reaction
PPR	Pestes des Petits Ruminants
RAV	Right displaced abomasum with volvulus
RDA	Right Displaced Abomasum
RVF	Rift Valley Fever
S/E	Side effect
SVTG	Standard Veterinary Treatment Guideline
UoG	Universty of Gonder
VDFACA	Veterinary Drug and Feed Administration and Control Authority
W/P	Withdrawal period

Annex 1: Veterinary Drugs for the Treatment of Fish Diseases

Drugs	Spectrum of activity	Method of administration	Dosage
Antibacterial Drugs			
Amoxicillin and Ampicillin	• Pasteurollosis • Furunculosis • Edwardseillosis • Streptococossis • Aeromonasis Spp.	• Injection, IM • Feed	• 10mg/kg/day • 40-80mg/kg/day for 10days.
Chloraphenicol	• Carp dropsy caused by *Aeromonas liquefacience* • Trout ulcer disease caused by *Haemophilus piscium*	• Injection, IP • Feed/Injection	• 12-80 mg/kg, single dose • 50-75 mg/kg/day
Difloxacin	• Furunculosis	• Feed	• 2.5-10 mg/kg/day for 5 days
Enrofluxacin	• Bacterial Kidney Disease • Furunculosis	• Feed	• 2.5 mg/kg/day for 10 days • 10 mg/kg/day for 10 days
Erythromycin	• Bacterial Gill disease	• Injection, IP • Feed	• 10-20 mg/kg for 3 days • 100 mg/kg/day for 21 days
Florfenicol	• *Vibrio anguillarum*	• Injection/Orally	• 2.5 mg/kg
Flumequine	• Furunculosis	• Feed	• 100 mg/kg for 5-8 days
Furazolidone	• Gram-positive and Gram-negative fish bacteria • Protozoan parasites	Feed	25-35 mg/kg/day for 20 days in all spp.
Nifurpirinol	• Columnaris disease	Flush	2 ppm for 1 hour
Nalidixic acid	• Furunculosis • Vibriosis	• Immersion	• 20 mg/kg/day
Ormetoprim-Sulfadimethoxine	• Edwardseilla spp. (*E. tarda, E. ictaluri*)	• Feed	• 50 mg/kg daily for 7 days
Oxolinic acid	• Furunculosis	• Oral	• 10 mg/kg/day

Drugs	Spectrum of activity	Method of administration	Dosage
Oxytetracyline	• *Vebrio salmonicida* infection (Cold water vibriosis) • *Yersinia ruckeri* infection (Enteric Red Mouth • Flavobacteriosis in common carp • *Aeromonas salmonicida* infection (Furunculosis) • *Flexibacter columnaris* infection (Columnaris disease) • *Streptococcosis* in rainbow trout.	• Injection, IP • Feed • Immersion	• 20mg/kg • 75mg/kg/day for 10days • 5-120mg/L
Piromidic acid	• Furunculosis	• Feed	• 10 mg/kg/day for 5 days
Sarafloxacilin	• Furunculosis	• Feed	• 10 mg/kg/day for 5 days
Sulfamirazine	• All bacterial diseases of fish	• Feed	• 200 mg/kg/day for 14 days
Sulfadimidine	• All bacterial diseases of fish	• Feed • Immersion	• 110-220 mg/kg/day for 6 days • 650 mg/l for 96 weeks
Sulfadimethoxine	• All bacterial diseases of fish	• Orally	• 40 mg/kg
Sulfamonomethoxine	• All bacterial diseases of fish	• Orally	• 300 mg/kg/day
Trimethoprim-Sulfadiazine	• *Aeromonas spp.* (*A. salmonicida, A. hydrophila, A. liquefaciens, A. punctata*) • Vibrio spp. (*V. aguillarum, V. salmonicida*) • *Pastuerella piscicida* • *Yersinia ruckeri*	• Feed	• 30 mg/kg daily for 7 days
Ectoparasiticides (Organophosphorus Compounds)			
Benzyl-ureas	• Copepods (fish lice, Angulus, Lerneaa) • Monogenea flukes (*Gyrodactylus* spp., *Dactyogyrus* spp.)	• Feed	• 10 mg/kg/day
Cypermethrin	• Monogenea flukes (*Gyrodactylus* spp., *Dactyogyrus* spp.)	• Bath	• 0.5 ml/m^3
Trichlorfom	• Copepods (fish lice, Angulus, Lerneaa)	• Oral	• 0.05 mg/kg every third day for 3 occasions
Anthelmintics			
Fenbendazole	• Skin flukes	• Immersion	• 20 mg/l for 12 hours

Drugs	Spectrum of activity	Method of administration	Dosage
Mebendazole	• Gyrodactylus spp. • Skin flukes • Many tapeworms	• Immersion • Feed	• 25 mg/l for 12 hours • 150 mg/kg/day
Niclosamide dissolved in DMSO	• *Gyrodactylus* spp.	• Immersion	• 0.1 ppm • Effective on immature
Niclosamide	• Strobila stages of tapeworms	• Feed	• 40 mg/kg/day for 3 days
Parbendazole	• *Gyrodactylus* spp.	• Immersion	• 25 mg/l for 12 hours
Praziquantel dissolved in Dimethyl-Sulphoxide (DMSO)	• Eye flukes • Skin flukes	• Immersion	• 2 ppm for 2 or 4 weeks • 600 ppm and after 8 hours exposure at 120 ppm
Praziquantel	• Strobila stages of tapeworms	• Immersion	• 5 mg/kg/day for 3 days
Triclabendazole	• *Gyrodactylus* spp. • Skin flukes		• 25 mg/l for 12 hours
Systemic Anti-protozoal Drugs			
Fumagilline dicyclohexy-lamine	• Microsporae (*Enterocytozoan salmonis*)	Feed	0.1g/kg at 1.55 for 3 weeks
	• Whirling disease (*Myxobolus cerebralis*)	Feed	1% for 4 months (10 mg/kg/day)
Dimetridazole	• Ick or White spot disease (*Ichthyophthirius multifilis* infection)	Feed	28 mg/kg/day for 10 days
Externally Applied Antimicrobial Agents			
Formalin	• Many prozoan parasites of the skin or gills including *Chilodonella* spp., *Epistylis* spp., *Ichthyobodo necator*, *Ichthyophthirius multifili*, *Scyphidia* spp., *Trichodina* spp. • Bacterial Gill Disease due to *Flavobacterium branchiophilum* • *Dactyogrus* spp. • *Gyrodactylus* spp • *Cleidodiscuss* spp. • *Saprolegnia* spp.	Immersion	167 ppm or 1:6000 dilution for fish 1667 ppm or 1:600 dilution for fish eggs • Intermittent exposures of 15 minutes each

Drugs	Spectrum of activity	Method of administration	Dosage
Malachite Green (MG)	• The spectrum is similar to formalin. • BUT it's efficacy against fungi is greater than formalin	Immersion	1.0 ppm at interval of 4-7 days in fish 2 ppm for 15 minutes every 24 hours for eggs
Leteux-Meyer mixture	• *Ichthyobodo spp.* • *Trichodia spp.* • *Chilodonella spp.* • *Scyphidia spp.* • *Trichophyra spp.* • *Ichthyophthirius spp.*	Immersion	Single dose of 3.3 g MG in 1 liter formalin BUT 3 applications at interval of 2 days are recommended for Ichthyophthirius.
Chloramin-T	• Bacterial Gill Disease • Fin-rot	Dipping	8.5-10 ppm for 1 hour daily for 3 days
Copper sulfate	• Bacterial Gill Disease • Protozoal infections	Immersion	0.1-0.2 ppm

Annex 2: Anaesthetic Agents

Anesthetics, Sedatives, Analgesics, Anti-inflammatory and Emergency drugs

Note: Dose rates vary with species and health status of the animal. Combination of different anesthetic drugs is very common practice. It should be noted that including at least one sedative or tranquilizer combined with one opioid analgesic is highly recommended for neuroleptanalgesia. Since combining anesthetic drugs together produces an additive effect, the doses of individual drugs must be reduced. It should be noted that the safety margin of most anesthetic drugs is very narrow demanding very accurate measurement of the animal's weight and dose calculation.

$$\text{Dose in ml} = \frac{\text{Dose (mg/kg)} * \text{Body weight (kg)}}{\text{Concentration of the drug (mg/ml)}}$$

Example:

The following are givens:

- ✓ Weight = 40.5 kg
- ✓ Dose = 0.3 mg/kg and
- ✓ Drug concentration = 2% = 20 mg/ml

Solution:

- ✓ **Step 1 =** Dose (mg/kg) * Body weight (kg)=0.3 mg/kg*40.5 kg = 12.15 mg
- ✓ **Step 2 =** Divide the result to drug concentration=12.15 mg ÷ 20 mg/ml = **0.61 ml**

Anesthetics and Analgesics used in Cattle

Pre-anesthetic drugs used for premedication in cattle			
Drugs	**Dose and route of injection**	**Purpose of use and other comments**	**Withdrawal period**
Acepromazine	0.2 mg/kg IM or 0.03 - 0.06 mg/kg slow IV	Sedation and general anesthetic-sparing effects.	
Xylazine	0.05 to 0.1 mg/kg IM	Tranquilization, analgesia, muscle relaxation, caudal epidural analgesia and anesthetic-sparing effects.	Meat 5 days Milk 72 hrs
Detomidine	0.02-0.04 mg/kg IM/IV		Meat 3days Milk 72 hrs
Romifidine	0.02-0.05 mg/kg IM/IV		
Medetomidine	0.03 mg/kg IM		
Diazepam	0.02 – 0.1 mg/kg IV or	Anxiolytic and sedation.	Meat 10days Milk -
Morphine	0.1 to 0.3 mg/kg IM,	Analgesic	Meat 14 days Milk 48hrs
Butorphanol	0.2 to 0.5 mg/kg IM,	Analgesic	Meat 5days Milk 72hrs
Tramadol	4 mg/kg IV,	Analgesic in calves	
Buprenorphine	0.005-0.01 mg/kg IM or 0.005-0.01 mg/kg IV	Analgesic	
Induction and Maintenance Drugs			
Drugs	**Dose and route of injection**	**Purpose**	**Withdrawal period**
Diazepam	0.5 mg/kg IV	Combined for induction and maintenance.	Meat 10days Milk -
Ketamine	5 mg/kg IV		Meat 3 days Milk 48hrs
Diazepam	0.1 - 0.25 mg/kg IV	Combined for induction and maintenance	Meat 10days Milk -
Xylazine	0.2 mg/kg IV		Meat 5 days Milk 72 hrs

Xylazine	0.2 mg/kg IM or 0.1 mg/kg IV	Combined for induction and maintenance	Meat 5 days Milk 72 hrs
Ketamine	5 - 10 mg/kg IM or 2 mg/ kg IV		Meat 3 days Milk 48hrs
Butorphanol	0.01 mg/kg IV/ 0.02 mg/kg IM	**Ketamine Stun:** For standing sedation.	Meat 5days Milk 72hrs
Xylazine	0.02 mg/kg IV/ 0.02 mg/kg IM		Meat 5 days Milk 72 hrs
Ketamine	0.05 mg/kg IV/ 0.04 mg/kg IM		Meat 3 days Milk 48hrs

Local, regional and epidural analgesia used in cattle

Drugs	**Purpose and route of injection**	**Withdrawal period**
Lidocaine 2%	To produce local analgesia by directly infiltrating or injecting at the site of incision or by depositing around a specific major nerve trunks as a sole analgesic agent or as part of multimodal analgesia.	Meat 1day Milk 24hrs

Antagonists of anesthetics and other emergency drugs used to reverse overdose and treat anesthetic complications in cattle

Drugs	**Dose and route of injection**	**Purpose**	**Withdrawal period**
Yohimbine	0.125 - 0.2 mg/kg IV	alpha-2 antagonists used to reverse side effects of xylazine and other drugs of the same group.	
Tolazoline	0.5–1.5 mg/kg IV		Meat 8days Milk 48hrs
Atipamezole	20– 50 µg/kg IM (or slow IV)		
Idazoxan	0.05 mg/kg IV		
Flumazenil	0.02 mg/kg IV	Benzodiazepine reversal	
Nalorphine	0.0088 mg/kg IV	Opioid drug reversal	
Doxapram	1 mg/kg IV	Respiratory stimulant	

Commonly used analgesics and doses for postoperative pain management			
Drugs	**Dose and route of injection**	**Purpose**	**Withdrawal period**
Carprofen	1.4 mg/kg IV, SC	Opioids used for pain management both during surgery and postoperatively. Note: Opioids have short duration of action requiring frequent dosing for postoperative analgesia.	
Dipyrone	10-20 mg/kg		
Meloxicam	0.5 mg/kg once IV, SC		
Ketoprofen	3 mg/kg q 24 h IV, IM		
Flunixin meglumine	1.1–2.2 mg/kg q 24 h IV/I.M.		Meat 4days Milk 36hrs
Tolfenamic acid	2–4 mg/kg IV, SC		
Morphine	0.1 mg/kg q 4 h, IM/IV		Meat 14 days Milk 48hrs

Anesthetics and Analgesics used in Sheep

Pre-anesthetic drugs used for premedication in sheep:			
Drugs	**Dose and route of injection**	**Purpose of use and other comments**	**Withdrawal period**
Acepromazine	0.05 to 0.2 mg/kg IM/IV	Sedation	
Xylazine	0.1–0.4 mg/kg IV, used for same as purpose as in cattle	Tranquilization, analgesia, muscle relaxation, caudal epidural analgesia and anesthetic-sparing effects.	Meat 58days Milk 72hrs
Detomidine	0.02 mg/kg IV		Meat 3days Milk 72hrs
Medetomidine	10 to 20 µg/kg IM		
Diazepam	0.1 mg/kg IV	Anxiolytic and Sedative	Meat 10days Milk -
Midazolam	0.2 mg/kg IV		
Morphine	10 mg total dose per sheep, IM	Analgesic	Meat 14days Milk 48hrs

Induction and maintenance drugs used in sheep			
Drugs	**Dose and route of injection**	**Purpose of use and other comments**	**Withdrawal period**
Diazepam	0.5 mg/kg IV	Combined for induction and maintenance.	Meat 10days Milk -
Ketamine	5 mg/kg IV		Meat 3days Milk 48hrs
Diazepam	0.25 mg/kg IV	Combined for induction and maintenance.	Meat 10days Milk -
Ketamine	5 mg/kg IV		Meat 3days Milk 48hrs
Xylazine	0.2 mg/kg IM	Combined for induction and maintenance.	Meat 58days Milk 72hrs
Ketamine	22 mg/kg IM		Meat 3days Milk 48hrs
Atropine	0.2 mg/kg IM		
Local, regional and epidural analgesia used in sheep:			
Drugs	**Purpose and route of injection**		**Withdrawal period**
Lidocaine 2%	To produce local analgesia by directly infiltrating or injecting at the site of incision or by depositing around a specific major nerve trunks as a sole analgesic agent or as part of multimodal analgesia.		
Antagonists of anesthetics and other emergency drugs used to reverse overdose and treat anesthetic complications in sheep			**Withdrawal period**
Drugs	**Dose and route of injection**	**Purpose**	
Yohimbine	0.125 mg/Kg	To reverse alpha-2 adrenoceptor agonists like xylazine.	
Tolazoline	0.5–1.5 mg/kg IV		Meat 3days Milk 48hrs
Atipamezole	20– 50 µ g/kg IM (slow IV)		
Flumazenil	0.02 mg/kg IV	To reverse benzodiazepines	

Anesthetics and analgesics used in Goats

Pre-anesthetic drugs used for premedication in goats:			
Drugs	**Dose and route of injection**	**Purpose**	**Withdrawal period**
Acepromazine	0.05 to 0.2 mg/kg IM/IV used for sedation	Sedation.	

Xylazine	0.05–0.1 mg/kg IV mg/kg IM, used for same as purpose as in cattle	Tranquilization, analgesia, muscle relaxation, caudal epidural analgesia and anesthetic-sparing effects.	Meat 58days Milk 72hrs
Detomidine	0.04 mg/kg IV or 0.08 mg/kg IM		Meat 3days Milk 72hrs
Medetomidine	0.03–0.08 mg/kg IM		
Diazepam	0.1 to 0.2 mg/kg IV	Anxiolytic and sedation.	Meat 10days Milk -
Midazolam	0.6 mg/kg IM		
Morphine	10 mg total dose per goat, IM	Analgesic	Meat 14days Milk 48hrs

Induction and Maintenance Drugs used in Goats:

Drugs	**Dose and route of injection**	**Purpose**	**Withdrawal period**
Xylazine	0.05 – 0.1 mg/kg IV, IM	Combined for induction, first xylazine then Telazol.	Meat 58days Milk 72hrs
Telazol	2 – 4 mg/kg IV (IM).		
Xylazine	0.05-0.1 mg/kg IM	Combined for induction and maintenance.	Meat 58days Milk 72hrs
Ketamine	5-10 mg/kg IM		Meat 3days Milk 48hrs

Local, regional and epidural analgesia used in goats:

Drugs	**Purpose and route of injection**	**Withdrawal period**
Lidocaine 2%	To produce local analgesia by directly infiltrating or injecting at the site of incision or by depositing around a specific major nerve trunks as a sole analgesic agent or as part of multimodal analgesia.	

Antagonists of anesthetics and other emergency drugs used to reverse overdose and treat anesthetic complications in goats

Drugs	**Dose and route of injection**	**Purpose**	**Withdrawal period**
Yohimbine	0.125 mg/Kg	To reverse alpha-2 adrenoceptor agonists like xylazine.	
Tolazoline	0.5–1.5 mg/kg IV		
Atipamezole	20– 50 µg/kg IM (or slow IV)		

Flumazenil	0.02 mg/kg IV	Reversing benzodiazepines	

Anesthetics and Analgesics used in Pigs

Pre-anesthetic drugs used for premedication in Pigs

Drugs	Dose, Route	Purpose	Withdrawal period
Atropine	0.02–0.05 mg/kg IM	Used to prevent bradycardia, salivation and bronchial secretion. Note: Atropine is better for emergency treatment.	
Glycopyrolate	0.004–0.01 mg/kg IM		
Acepromazine	0.1 to 0.2 mg/kg.	Used for sedation. Note: Pigs are resistant to acepromazine. Total dose shouldn't exceed 15 mg).	
Azaperone	1-2.5 mg/kg deep IM,	Used for sedation	

Azaperone can be combined as follows for more deeper sedation in swine:

- Azaperone 1– 2 mg/kg, midazolam 0.3 mg/kg together deep IM.
- Azaperone 1 mg/kg, ketamine 5 mg/kg together deep IM.
- Azaperone 1 mg/kg, midazolam 0.1 mg/kg, ketamine 5 mg/kg all together deep IM.
- Azaperone 1– 2 mg/kg, +/ – butorphanol 0.1– 0.2 mg/kg, ketamine 5 mg/kg all together deep IM.

Xylazine	4 mg/kg IM or 2 mg/kg IV	Used for tranquilization, analgesia, muscle relaxation, caudal epidural analgesia & anesthetic-sparing effects. **Note:** Xylazine is less potent in pigs than in cattle or horses. Meat 2days Milk -	Meat 18days Milk -
Detomidine	0.04 mg/kg IV or 0.08 mg/kg IM		
Medetomidine	0.03–0.08 mg/kg IM		
Diazepam	0.44–2 mg/kg IV		
Midazolam	0.1–0.5 mg/kg IM/SC/IV		
Ketamine solo	20–30 mg/kg IM/SC		
Morphine	0.1 to 0.5 mg/kg IM	Opioids used for pain management both during surgery and postoperatively. **Note:** opioids have short duration of action requiring frequent dosing for postoperative analgesia.	
Butorphanol	0.1 to 0.5 mg/kg IM		
Pethidine	3.5 – 5 mg/kg IM.		
Tramadol	5 mg/kg IM		
Buprenorphine	0.006-0.02 mg/kg IM		

Induction and maintenance drugs used in pigs:			
Drugs	**Dose and route of injection**	**Purpose**	**Withdrawal period**
Diazepam	2 mg/kg IM/SC or 0.5 mg/kg IV	Combined for induction and maintenance. Meat 2days Milk -	
Ketamine	20 mg/kg IM/SC or 5 mg/kg IV		
Medetomidine	0.1 mg/kg IM/SC	Combined for induction and maintenance. Meat 2days Milk -	
Ketamine	1 mg/kg IM/SC		
Midazolam	0.5 mg/kg IM/SC	Combined for induction and maintenance. Meat 2days Milk -	
Ketamine	33 mg/kg IM/SC		
Ketamine	20 mg/kg IM/SC	Combined for induction and maintenance.	Meat 2days Milk -
Xylazine	2 mg/kg IM/SC		
Telazol	4.4 mg/kg IM	Combined for induction and maintenance. Meat 2days Milk -	Meat 4days Milk -
Ketamine	2.2 mg/kg IM		
Telazol	4.4 mg/kg IM	Combined for induction and maintenance.	Meat 4days Milk -
Xylazine	2.2 to 4.4 mg/kg IM		
Xylazine	1–3 mg/kg IM	All the three combined for induction and maintenance **Note:** Xylazine can be replaced with Detomidine 0.1 mg/kg or Medetomidine 30–80 µ g/kg or Romifidine 120 µ g/kg. Meat 2days Milk -	
Ketamine	5 mg/kg IM		
Butorphanol	0.1–0.2 mg /kg IM		

Local, regional and epidural analgesia			
Drugs	**Purpose and route of injection**		
Lidocaine 2%	Local infiltration at the incision site, regional nerve block and epidural as standalone or as part of multimodal analgesia.		
Analgesics for postoperative pain management			
Drugs	**Dose and route of injection**	**Purpose**	**Withdrawal period**
Meloxicam	0.4 mg/kg SC, SID	Opioid and NSAIDs used for analgesic treatment. Opioids are used for pain management both during surgery and postoperatively. **Note:** Opioids have short duration of action requiring frequent dosing for postoperative analgesia.	
Aspirin	10-20 mg/kg, PO, q 6 hr.		
Butorphanol	0.1–0.3 mg/kg IM/SC, QID		
Buprenorphine	0.01–0.05 mg/kg IM/SC, BID or TID		
Flunixine meglumine	1.1 - 2.2 mg/kg IM/SC, SID/BID		Meat 2days Milk -
Carprofen	2 mg/kg IM/SC, SID		
Ketoprofen	1-3 mg/kg IM/SC/PO, BID		
Tramadol	4 mg/kg, PO, TID		
Phenylbutazone	5–20 mg/kg, PO/BID		
Tolfenamic acid	2 mg/kg q 24 h IM		
Morphine	0.1–0.2 mg/kg q 4 h, IV, IM		

Anesthetics and Analgesics used in Camels

Pre-anesthetic drugs used for premedication in Camels:		
Drugs	**Dose and route of injection**	**Purpose**
Atropine	0.04 mg/kg IM or 0.01-0.02 mg/kg IV	Anticholinergic used to prevent vagal bradycardia, salivation and bronchial secretion.
Glycopyrolate	0.002-0.005 mg/kg IV	
Acepromazine	0.05 – 0.1 mg/kg IM	used for sedation
Xylazine	0.1 to 025 mg/kg IM	Used for tranquilization, analgesia and muscle relaxation.
Detomidine	0.03 – 0.06 mg/kg IM	
Medetomidine	10 - 30 µg/Kg IM	
Diazepam	0.2 to 0.4 mg/kg IV	Use for sedation or anxiolysis
Midazolam	0.1 to 0.2 mg/kg IV	

Morphine	0.05–0.1, IV, IM q 4 h	Used for analgesic treatment, good analgesic combined with 2% lidocaine line block.
Butorphanol	0.5 - 0.1 mg/Kg IM/SC	
Tramadol	11 mg/kg IM q 8 h	
Induction and Maintenance Drugs used in Camels:		
Xylazine	0.1 mg/kg IM	Combined for deep sedation and immobilization.
Ketamine	2.0 – 3.0 mg/kg IM	
Butorphanol	0.05 – 0.1 mg/kg IM	
Xylazine	0.4 mg/kg IM or 0.25 mg/kg IV	Deep sedation and Immobilization.
Ketamine	5 mg/kg IM or 3.0 mg/kg IV	
Diazepam	0.2–0.5 mg/kg IV	Combined for induction and maintenance.
Ketamine	5.0–8.0 mg/kg IM or 3.0 – 5.0 IV	
Xylazine	0.4 mg/kg IV	Combined for induction and maintenance. **Note:** ½ of the calculated dose is given initially and additional dose given as necessary.
Ketamine	0.4 mg/kg IV	
Drugs	**Purpose and route of injection**	
Lidocaine 2%	Regional nerve block and local infiltration Local injection at the incision site or around major nerve trunk as a sole method or part of multimodal analgesia.	

Antagonists of anesthetics and other emergency drugs used to reverse overdose and treat anesthetic complications in camels

Drugs	**Dose and route of injection**	**Purpose**
Yohimbine	0.125–0.25 mg/Kg IV	Alpha-2 antagonists To reverse side effects xylazine and other drugs of the same group.
Tolazoline	0.5–1.5 mg/kg IV	
Atipamezole	0.1–0.15 mg/kg IM (or slow IV)	
Flumazenil	1.0 –2mg/kg IV	Antagonist for benzodiazepines
Dexamethasone	2 mg/kg	For shock treatment. **Note:** Should not be used in case of shock caused by septicemia.
Lidocaine	0.5 mg/kg IV	For emergency treatment of ventricular arrhythmia.
Doxopram	0.1 mg/kg IV	For treatment of respiratory depression.
Epinephrine	0.01 mg/kg IV	For emergency treatment of cardiac arrest, anaphylaxis.
Atropine	0.04 mg/kg IV/IM	For emergency treatment of bradycardia.
Diazepam	0.1– 0.5 mg/kg IV	For emergency treatment of Seizures.
Calcium gluconate 23% 8 mEq/ml	0.7 mEq/ml	For emergency treatment of hypocalcaemia.

Bicarbonate, Na 1 mEq/ml	0.5 mEq/ml	For emergency treatment of metabolic acidosis.
Postoperative analgesic		
Drugs	**Dose and route of injection**	**Purpose**
Phenylbutazone	2-4 mg/kg IV q 24-48 h	**Note:** Perivascular injections may result in phlebitis D/F: 50 - and 100 - ml bottles (200 mg/ml)
Ketoprofen	1–2 mg/kg, IV q 24 h	
Meloxicam	1mg/kg, PO q 72 h	
Flunixin Meglumine	1.1 mg/kg, IV, q 24 h	
Morphine	0.05–0.1, IV, IM q 4 h	

Anesthetics and Analgesics used in Chicken

Pre-anesthetic drugs used for premedication in Chicken		
Drugs	**Dose and route of injection**	**Purpose**
Atropine	0.04 mg/kg	To prevent or treat bradycardia during anesthetic period.
Glycopyrrolate	0.01 mg/kg	
Xylazine	1.0-6.0 mg/kg 1M	
Diazepam	0.5-1.0 mg/kg 1M	For sedation and anxiolysis.
Midazolam	0.5-1.0 mg/kg 1M	
Butorphanol	1 to 3 mg/kg IM	Opioid analgesics
Buprenorphine	0.25 - 0.5 mg/kg IM	
Induction and maintenance drugs used in chicken		
Drugs	**Dose and route of injection**	**Purpose**
Pentobarbital	25 to 30 mg/kg IV	Slow acting (takes 15 minute) but prolonged duration (Several hours).
Local, regional and epidural analgesia used in chicken		
Drugs	**Dose and route of injection**	**Purpose**
Lidocaine	Local infiltration at 2 to 3 mg/kg	**Note:** the 2% lidocaine should be diluted 1:10 with sterile water for dosing accuracy.
Drugs	**Dose and route of injection**	**Purpose**
Carprofen	1.0-3.0 mg/kg SC, PO q 8-12 h	May cause muscle necrosis at injection site; hydration is essential; use only for short duration; potential Nephrotoxicity.
Flunixin meglumine	1.0-5.0 mg/kg IM, q 12-24 hours	

Antagonists of anesthetics and other emergency drugs used to reverse overdose and treat anesthetic complications in Chicken		
Drugs	**Dose and route of injection**	**Purpose of use and other comments**
Tolazoline	15 mg/kg I V	To reverse overdose or side effects of alpha-2 adrenoceptor agonists
yohimbine	0.1 mg/kg	

Anesthetics and Analgesics used in Equine

Pre-anesthetic drugs used for premedication in Equine		
Drugs	**Dose and route of injection**	**Purpose of use and other comments**
Atropine	0.002-0.01 mg/kg IV	To prevent or treat bradycardia during anesthetic period
Glycopyrolate	0.001 – 0.005 mg/kg IV	
Acepromazine	0.2 mg/kg IM or 0.03 - 0.06 mg/kg slow IV, used for sedation	Used for sedation. **Note:** can cause vasodilation and hypotension; need care in hypovolaemic patients
Triflupromazine HCl (Siquil)	0.2 – 0.3 mg/kg IM/IV	Maximum dose not to exceed 100 mg)
Xylazine	2 mg/kg IM or 1.1 mg/kg IV in **horses**, 2.6-3.0 mg/kg IM or 1.3-1.5 mg/kg IV in **donkeys and mules**	Used for tranquilization, analgesia muscle relaxation, caudal epidural analgesia & anesthetic-sparing effects. **Note**: care must be taken as animals may kick.
Detomidine	0.01 to 0.04 mg/kg IV or 0.02 to 0.05 mg/kg IM	
Romifidine	0.07 mg/kg IV	
Morphine	0.05-0.1 mg/kg, IM/Slow IV (maximum total of 60 mg)	Used as potent analgesic. Note: morphine may cause excitement in horses
Butorphanol	0.05-0.2 mg/kg IM or 0.05 -0.1 mg/kg IV	
Tramadol	2.4 mg/kg IV,	
Induction and maintenance drugs used in Equine:		
Drugs	**Dose and route of injection**	**Purpose of use and other comments**
Thiopental	10 mg/kg IV	Induction and maintenance surgical anesthesia. Use from 5% or 10% solution
Propofol	2 mg/kg IV in premedicated horse with **xylazine** 1.1 mg/kg	Induction and maintenance surgical anaesthesia
Telazol	1–2 mg/kg, IV	Induction and maintenance surgical anesthesia

Xylazine	1.1 mg/kg IV	Combined for induction and maintenance. Diazepam is given two minutes after xylazine & ketamine 2 minutes after diazepam
Diazepam	0.02 mg/kg IV	
Ketamine	2 - 2.25 mg/kg IV	
Antagonists of anesthetics and other emergency drugs used to reverse overdose and treat anesthetic complications in equines		
Drugs	**Dose and route of injection**	**Purpose of use and other comments**
Yohimbine	0.075 mg/kg IV	Alpha-2 antagonists used to reverse side effects xylazine and other drugs of the same group.
Tolazoline	2.0 mg/kg IV	
Atipamezole	0.1-0.2 mg/kg IV	
Flumazenil	0.01 to 0.04 mg/kg IV	Benzodiazepine antagonist
Naloxone	0.01-0.02 mg/kg IV	Opioids antagonist
Nalmefene	0.001-0.005 mg/kg IV	
Doxapram	0.5-1.0 mg/kg IV	Analeptic - respiratory stimulant
Local, regional and epidural analgesia used in equines:		
Drugs	**Purpose, dose and route of injection**	
Lidocaine 2%	4 mg/kg (the maximum safe dose). Local infiltration, peripheral nerve blocks and epidurals as part of multimodal analgesia. Short-acting (1.5 to 2 hours).	
Bupivacaine	1 to 2 mg/kg of a 0.25% solution (the maximum safe dose).	More commonly used for postoperative pain relief of musculoskeletal origin and intra-articular analgesia because of longer duration of action of 3 to 8 hours.
Xylazine	0.25 mg/kg diluted in 10 ml, Caudal epidural	Higher than recommended caudal epidural dose should be avoided as it will cause ataxia.
Detomidine	0.04 mg/kg diluted in 10 ml, Caudal epidural	
Morphine	0.1 to 0.2 mg/kg diluted in 20 mL 0.9% saline for Caudal epidural	
Commonly used drug and doses for postoperative pain management		
Drugs	**Dose, route & frequency of injection**	**Purpose**
Morphine	0.1–0.15 mg/kg q 4 h, IV, IM	Opioids used for intraoperative as well as postoperative pain management.
Buprenorphine	0.01–0.02 mg/kg q 6–8 h, IV, IM, SC	
Butorphanol	0.1 mg/kg, q 2–4 h, IV, IM	
Meperidine	0.2-1.0 mg/kg IV	
Oxymorphone	0.001-0.02 mg/kg IV	
Tramadol	2 mg/kg IM q 4 h	

Gabapentin	2.5–5 mg/kg PO q 8–24 h	NSAIDs better used for only postoperative pain and pain due to other inflammatory diseases. Note: don't give phenylbutazone above 4.4 mg/kg q 12 h, because of its higher toxicity risk
Carprofen	0.7 mg/kg q 24 h IV, PO	
Meloxicam	0.6 mg/kg q 24 h IV, PO	
Ketoprofen	2.2 mg/kg q 24 h IM/IV	
Phenylbutazone	4.4 mg/kg q 12 h for 4 days followed by 2.2 mg/kg q 24–48 h IV, PO	
Naproxen	5 mg/kg IV or 4 g PO BID per horse	
Flunixin	1.1-2.2 mg/kg mg/kg q 24 h IV/IM	
Firocoxib	0.09 mg/kg IV then 0.1 mg/kg PO q 24 h for up to 14 days	
Eltenac	0.5–1.0 mg/kg q 12 h IV	
Aspirin	17 mg/kg q 48 h PO	
Ibuprofen	10–25 mg/kg q 8–12 h IV or PO	
Dipyrone	5-22 mg/kg IV	
Pentazocine	0.5-1.0 mg/kg, IV/IM,	

Corticosteroids

Drugs	**Dose and route of injection**	**Purpose**
Dexamethasone	0.015-0.050 mg/kg IV	To treat life threatening conditions such as brain edema
Methylprednisolone	0.1-0.5 mg/kg IV	
Prednisolone	0.25-1.0 mg/kg IV/PO	
Hydrocortisone	1.0-4.0 mg/kg IV	

Drugs used in combination anesthetic protocols in dogs

Pre-anesthetic drugs used for premedication in dogs:

Drugs	**Dose and route of injection**	**Purpose**
Atropine	0.04 mg/kg IM, 0.02 mg/kg used to prevent bradycardia	Anticholinergics, used to prevent or treat vagal-induced bradycardia.
Glycopyrolate	0.01 mg/kg IM	
Acepromazine	0.05 mg/kg IM	**Note:** Total dose should not exceed 2 mg)

Xylazine	1.0 to 2.0 mg/kg IM	Used for tranquilization, analgesia & muscle relaxation
Dexmedetomidine	0.0015 - 0.0025 mg/kg IM	
Medetomidine	40 to 80 µg/kg IM/IV	
Romifidine	0.02-0.04 mg/kg IM or 0.01-0.02 mg/kg IV	
Ketamine solo	5 mg/kg IV or 10 mg/kg IM	**Caution**: May cause convulsion and muscle rigidity. To prevent this, combine with diazepam or xylazine.
Diazepam	0.2 to 1 mg/kg IV	For sedation and anxiolysis
Midazolam	0.2 mg/kg IV or 4 mg IM	
Morphine	0.25 to 0.5 mg/kg IM,	Opioid analgesics. **Note:** Best drugs for surgical pain management, but require frequent dosing per day for postoperative pain management due to short duration of action.
Hydromorphone	0.05 – 0.2 mg/kg SC	
Butorphanol	0.2 mg/kg IM	
Tramadol	2 mg/kg IM	
Buprenorphine	0.01-0.02 mg/kg IM	
Nalbuphine	0.2 – 0.4 mg/kg IM	

Induction and maintenance drugs used in dogs:

Drugs	**Dose and route of injection**	**Purpose**
Thiopentone	10 mg/kg IV in premedicated (or 15 to 20 mg/kg in unpremedicated)	Used alone
Propofol	4 mg/kg IV for premedicated (or 6.5 mg/kg IV for unpremedicated)	Used alone
Thiopentone	10.0 mg/kg IV	Combined
Diazepam	0.5 mg/kg IV	
Propofol	4.0 mg/kg IV for induction and 1-3 for maintenance	Induction dose combined with induction dose and maintenance dose with maintenance of dose of each drug.
Diazepam	0.5 mg/kg IV for induction and 0.25 mg/kg for maintenance	
Telazol	6-12 mg/kg IM, or 5 mg/kg IV for induction & 2-5 mg/kg IV for maintenance	Used alone
Diazepam	0.5 mg/kg IV	Combined
Ketamine	5 mg/kg IV	
Diazepam	0.25 mg/kg IV	Combined
Ketamine	5 mg/kg IV	

Xylazine	1 mg/kg IM	Combined for induction **Note**: Give IV fluids and avoid overdosing
Ketamine	10 mg/kg IM	
Atropine	0.04 mg/kg IM	
Etomidate	2.0 mg/kg IV	Combined for induction. **Note:** Induction quality could be poor that may need other anesthetic agent administration.
Diazepam	0.5 mg/kg IV	
Halothane	Induction - 2 to 4% at flow rate of 100 ml/kg/min in circle system & 200 ml/kg/min in Bain circuit. Maintenance - 1 to 2% at flow rate of 30-50 ml/kg/min in circle system and 200 ml/kg/min in Bain circuit.	**Route:** Inhalation via endotracheal tube connected to anesthetic machine. **Precaution:** Precision vaporizer, adequate ventilation and scavenging are essential.

Antagonists of anesthetics and other emergency drugs used to reverse overdose and treat anesthetic complications in dogs

Drugs	**Dose and route of injection**	**Purpose**
Yohimbine	0.1-0.125 mg/kg slow IV or 0.3-0.5 mg/kg IM	Alpha-2 antagonists used to reverse side effects xylazine and other α_2-adrenoceptor agonists.
Tolazoline	0.5-1.0 mg/kg IV or 2-5 mg/kg IM	
Atipamezole	0.05-0.2 mg/kg IV,IM	
Doxapram	0.2-0.5 mg/kg IV	Respiratory stimulant used to treat anesthetic induced respiratory depression.
Dexamethasone	4-6 mg/kg IV	To treat life-threatening airway swelling.
Diphenhydramine	2-4 mg/kg SQ, IM, IV, PO	To treat allergic reactions due to drugs, vaccines or snake bites.
Furosemide	2-4 mg/kg IV	To treat pulmonary and cerebral edema.
Atropine	0.02 mg/kg IV; 0.04 mg/kg IM	Used to treat bradycardia (HR < 60 bpm in dogs) caused by deep anesthesia.
Glycopyrolate	0.01 mg/kg IV	
Isoproterenol	0.5 µg/kg IV	
Epinephrine	0.02-0.2 mg/kg IV	
Ephedrine	0.1-0.5 mg/kg IM	To treat hypotension due to deep anesthesia.

Propranolol	0.02-0.06 mg/kg) slow IV	Beta-adrenergic blockers used to treat severe sinus tachycardia.
Esmolol	0.25-0.5 mg/kg slow IV	
Naloxone	0.04 mg/kg IM or 0.01 mg/kg IV	Opioid antagonist
Commonly used drugs and doses for postoperative pain management in Dogs		
Drugs	**Dose and route of injection**	**Purpose**
Carprofen	2–4 mg/kg IV, SC, PO q 24 h	**Note:** Most NSAIDs are not licensed for pre-operative pain medication due to side effects such as GIT ulceration renal failure. Their use is better limited for postoperative pain medication in well hydrated patients.
Meloxicam	0.2 mg/kg on day 1; 0.1 mg/kg q 24 h Thereafter IV, SC, PO	
Piroxicam	0.3 mg/kg q 48 h PO	
Ketoprofen	1-2 mg/kg q 24 h, IV, IM, SC, PO	
Firocoxib	5 mg/kg q 24 h PO	
Deracoxib	3-4 mg/kg q 24 h PO	
Aspirin	10 - 25 mg/kg q 12 h PO	
Acetaminophen	10 - 15 mg/kg q 8 h PO	
Mavacoxib	2 mg/kg q 1 month PO	
Robenacoxib	2 mg/kg SC or 1 mg/kg PO q 24 h	
Etodolac	10-15 mg/kg q 24 h PO	
Ketorolac	0.5 mg/kg q 12 h, IV, IM, for 1 to 2 treatments	
Tolfenamic acid	4 mg/kg q 24 h for 3 days, IM, SC, PO	
Tepoxalin	10 mg/kg, q 24 h PO	
Naproxen	2 mg/kg q48h PO	
Gabapentin	10 mg/kg q 12 h PO	
Phenylbutazone	10–14 mg/kg q 12 h PO	
Butorphanol	0.2–0.4 mg/kg, q 2–4 h, IM/IV	Opioid analgesics. Good for both pre-and postoperative pain medication
Morphine	0.2–0.5 mg/kg q 4 h, IV, IM	
Hydromorphone	0.05 – 0.2 mg/kg SC Q **q4-8h**	
Buprenorphine	0.01–0.02 mg/kg, q 4–6 h, IV, IM	
Tramadol	2-4 mg/kg IV or 5-10 mg/kg PO, q 6–12 h	

Local, regional and epidural analgesia used in dogs		
Drugs	**Dose and route of injection**	**Purpose**
Lidocaine 2%	4 mg/kg	
	2% 1 ml per 4.5 kg, duration 1-2 hr.	
Bupivacaine 0.5%	0.22 ml/kg, duration 2-5 hr.	Epidural.
Ropivacaine 0.5%	0.22 ml/kg, duration 2-5 hr	
Morphine (preservative free)	0.1 mg/kg, duration 10-24	Epidural
Buprenorphine	0.004 mg/kg, duration 12-18	Epidural

Drugs used in combination anesthetic protocols in cats

Pre-anesthetic drugs used for premedication in cats		
Drugs	**Dose and route of injection**	**Purpose**
Atropine	0.04 mg/kg IM, 0.02 mg/kg IV	Anticholinergics, used to prevent salivation and bronchial secretion and treat vagal bradycardia,
Glycopyrolate	0.01 mg/kg IM,	
Acepromazine	0.1 – 0.15 mg/kg IM	Sedatives
Xylazine	1.0 to 1.8 mg/kg IM	Tranquilization, analgesia and muscle relaxation.
Dexmedetomidine	0.0015 - 0.0025 mg/kg IM,	
Medetomidine	80 to 120 µg/kg IM	
Romifidine	0.03-0.06 mg/kg IM 0.015-0.03 mg/kg IV	
Diazepam	0.2 to 1 mg/kg IV	Anxiolytic, anticonvulsant and muscle relaxation.
Midazolam	0.2 mg/kg IV or 4 mg IM	
Morphine	0.1 mg/kg IM	Opioids – most potent analgesic drugs of choice for both pre and post-surgical pain. They are preferably used in combination with a sedative or tranquilizer.
Butorphanol	0.2 to 0.4 mg/kg IM	
Tramadol	2 mg/kg IM	
Buprenorphine	0.005-0.01 mg/kg IM or 0.005-0.01 mg/kg IV	
Nalbuphine	0.2 – 0.4 mg/kg IM	

Induction and maintenance drugs used in cats		
Drugs	**Dose and route of injection**	**Purpose**
Propofol	Induction - 4-6 mg/kg IV in premedicated (or 8 mg/kg IV in unpremedicated) Maintenance 2-3 mg/kg IV	Used for induction and maintenance.
Telazol	Induction 6-12 mg/kg IM, or 5 mg/kg IV & maintenance 2-5 mg/kg	Used for induction and maintenance.
Diazepam	0.5 mg/kg IV	Combined for induction.
Ketamine	5 mg/kg IV	
Diazepam	0.25 mg/kg IV	Combined for maintenance.
Ketamine	5 mg/kg IV	
Xylazine	0.26 mg/kg IM	Combined for induction and maintenance.
Ketamine	11 mg/kg IM	
Atropine	0.02 mg/kg IM	
Halothane	Induction & maintenance	**Note:** administer only using none rebreathing circuits.
Commonly used drug doses for postoperative pain management in cats		
Drugs	**Dose and route of injection**	**Purpose**
Carprofen	4 mg/kg ONCE IV, SC	Opioid and NSAIDs used for analgesic treatment. Opioids are used for pain management both during surgery and postoperatively. **Note:** opioids have short duration of action requiring frequent dosing for postoperative analgesia.
Meloxicam	0.3 mg/kg once SC OR 0.1 mg/kg on day 1; 0.05 mg/kg q 24 h thereafter SC, PO	
Ketoprofen	2 mg/kg q 24 h or followed by oral admin 1 mg/kg q 24 h for 5 days SC, PO	
Aspirin	1 - 25 mg/kg q 72 h PO	
Robenacoxib	2 mg/kg SC or 1 mg/kg PO q 24 h	
Tolfenamic acid	4 mg/kg q 24 h for 3 days SC, PO	
Butorphanol	0.1–0.5 mg/kg q 2–4 h, IM/IV	
Morphine	0.1–0.3 mg/kg q 4-6 h, IV, IM	
Hydromorphone	0.05 – 0.2 mg/kg SC Q q4-8h	
Butorphanol	0.2 to 0.4 mg/kg q 2–4 h, IM	
Buprenorphine	0.01–0.02 mg/kg q 6–8 h, IV, IM	
Tramadol	2 mg/kg IV, SC or 2–10 mg/kg PO	
Gabapentin	10 mg/kg PO q 8 h	

Antagonists of anesthetics and other emergency drugs used to reverse overdose and treat anesthetic complications in cats		
Drugs	**Dose and route of injection**	**Purpose**
Yohimbine	0.1 - 0.125 mg/Kg slow IV	Alpha-2 antagonists to reverse side effects xylazine and other drugs of the same group.
Tolazoline	1 - 2 mg/Kg	
Atipamezole	0.05-0.2 mg/kg IV,IM	
Naloxone	0.04 mg/kg IM or 0.01 mg/kg IV	Full Opioid antagonist.
Diazepam	0.5 – 1 mg/kg IV (If IV access gets difficult give rectally)	For treatment of seizures.
Epinephrine	0.02-0.2 mg/kg IV	For emergency treatment of cardiac arrest, anaphylaxis.
Atropine	0.02 mg/kg IV or 0.04 mg/kg IM	For prevention - or emergency treatment of bradycardia.
Glycopyrolate	0.01 mg/kg IV	
Doxapram	1-5 mg/kg IV	For treatment of respiratory depression.
Dexamethasone	4-6 mg/kg IV	To treat life-threatening shock and airway swelling.
Diphenhydramine	2-4 mg/kg SQ, IM, IV, PO	To treat allergic reactions due to drugs, vaccines or snake bites.
Furosemide	2-4 mg/kg IV	To treat pulmonary and cerebral edema.
Calcium gluconate	1-1.5 ml/kg of 10% solution IV over 10-20 minutes	For emergency treatment of hypocalcaemia.
Local, regional and epidural analgesia used in cats		
Drugs	**Purpose, dose and route of injection**	
Lidocaine 2%	2 ml (maximum dose) per cat, infiltration along incision line. **Note:** be aware of potential neurotoxicity in cats.	

Annex 3. Weight and Fluid Equivalents (British)

Conversions			
1000 mcg	1 mg	17 minims	1 ml
65 mg	1 grain(gr)	28.4 ml	1 once
1000 mg	1 gram	5 ml	Teaspoon
15	1 gram	15 ml	Tablespoon
1000	1 kilo gram	100 ml	305 ounce
30g	1 ounce	480 ml	1 pint
454 gm	1 lb pound	2 pints	1 quart
2.2 lb	1 kg	1.75 pints	1 liter
1000 kg	Ton (imperial)	32 ounce	1 quart
1% (w/v)	1gm/100 ml	1000ml	1 liter

Annex 4. Average Normal Vital signs (Pulse, Rectal Temperatures and Respiration rates)

Animal spp.	Age category	Pulse/minute	Temperature (±0.5 °C)	Respiration/minute
Horse	Adult (Stallion and Mare)	28-40	38	9-10
	Young (6-24month)	40-72		
	Foal (Newborn-6month)	64-128		
Ass/Mule,	Adult	45-60		
	Young	65-75		
Cattle	Adult (Cow and Ox)	35-70	38.5	12-16
	Yearling	90		27
	Calf (Newborn - 6month)	95-140		30-56
Sheep/Goat	Adult	70-80	39.5/39	12-15
	Lambs/Kids/Yearling	85-120		12-20
Pigs	Adult (Sow and Boar)	60-95	39 .0	10-12
	Piglets (Newborn -14weeks)	110-140		
Dogs	Adult	60-80	39	14-16
	Puppy	110-120		20-22
Cat	Adult	100-120	38.5	20-30
	Kitten	130-140		
	Rabbit	120-150	39.3	50-60
	Camel	30-50		5-12
	Chicken	180-440		15-30

DRUG INDEX

Annex 5. Antimicrobials

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Amoxicillin		Hypersensitivity reactions to penicillins.		Simultaneous administration of chloramphenicol, tetracycline or phenylbutazone. W/P: meat 14 days and milk 3 days.	14,20,22,38,43,6,3,64,65,76,79,116, 120,141,142,143, 145, 169,173, 197,199
Amoxicillin: 200 mg + Clavulanic acid: 50 mg +Prednisolone	Intramammary suspension.			W/P: Meat and offal: 7 days and Milk: 84 hours.	43
Amoxicillin (as trihydrate)	Oral suspension, Bolus, Injection 50 mg/ml, long acting suspension 150mg/kg, and 200mg/kg; power 50%.	Hypersensitivity. Not to be used in layers; To be used only in broilers;	Gastrointestinal effects such as lack of appetite, vomiting and diarrhea. Serious side effects include allergic reaction such as skin rash, fever, facial swelling, or difficulty breathing, in-coordination, or prolonged lack of appetite. Hypersensitivity following inhalation or ingestion or skin contact.	W/P: Meat 7 days, Poultry 7 days, Eggs 3 days.	139
Amphotericin B	Ointment	Animals with hepatic impairment, pregnant and lactating animals, Ketoconazole should not be used in animals with known hypersensitivity to the drug.	Hepatotoxicity, anorexia, nausea, vomiting, pruritis, alopecia, gynecomastia and sexual impotence/infertility. GI disturbances, changes in the hair coat, catarcats.	Drugs that reduce stomach acidity may reduce ketoconazole absorption, antimuscarinic drugs, cimetidine, ranitidine, phenytoin and warfarin. It has antagonistic effect with Ketoconazole, thus should not be used in combination.	97,106,112,180,

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Ampicillin	Powder; 0.1, 0.15, 0.2, 0.5, 20, 70 and 75%, Oral Suspension; 5, 10 and 15%.	Hypersensitivity reaction.		W/P: Meat 21 days, milk 7 days.	8,19,22,24,37,43,44,61,84,98,107, 162,169,197,
Benzathine Cloxacillin	Intramammary suspension, 200mg/dose.	Hypersensitivity to penicillin and cephalosporin.	Allergic reactions.	Withdrawal period during treatment period.	43
Cefalonium	Intramammary suspension.	Do not use in animals with known hypersensitivity to cephalosporins, and other ß-lactam antibiotics. Do not use in the lactating cow. Not intended for use within 54 days of calving.			44
Ceftiofur sodium	2.2 mg/kg.		Hypersensitivity reactions, potential nephrotoxicity.	W/P: meat nil; milk 72 hours.	38,41,46,47,79,108,
Cephalothin	Injection, 0.5gm, 1gm.	Hypersensitivity to penicillin, impaired renal function.	Painful when used IM.	Concurrent administration with Furosamide and ethacrynic acid.	98
Chlortetracycline, see also Doxycycline	10% or 20% feed mix powder.	Do not administer to animals with impaired kidney and liver function, hypersensitivity to tetracycline.	Diarrhea.	Concurrent administration with penicillin cephalosporin, quinolones and cycloserine. W/P: Meat 7 day, egg – 9 days.	61,90,135,139,143,
Cloxacillin and ampicillin	Intramammary suspension.				44,79
Chlortetracycline hydrochloride eye ointment	Eye ointment.	Do not use in cases of hypersensitivity to the active substance or to any of the excipients.	None known.	No data available. W/P: Meat and offal: 1 day Not authorized for use in horses producing milk intended for human consumption	97

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Cloxacillinbenzathine	Ointment, 16.7% in 5 gm syringe.	Hypersensitivity to penicillin and cephalosporins.	Swelling of face, difficulty in respiration.		
Clindamycin	25, 75 or 150 mg capsules.	Hepatic or renal function impairment.	Sensitivity to lincomycines; in cats, lip smacking in oral solution, salivation.	Anaesthetics such as enflurane, halothane, isoflurane, methoxyflurane, or neuromuscular blocking agents resulting in respiratory depression or paralysis; chloramphenicol or erythromycin.	187
Colistin (As sulphate)	2.5 mg/ml (equivalent to 50.000 IU of colistin).	Do not use for animals hypersensitive to polymyxins.	None.	W/P: Meat 21 days and Eggs 5 days.	141
Denaverine hydrochloride	Solution for injection.	Do not administer in cases of mechanical obstetrical disorders.		**Special warnings** The product is ineffective if no part of the foetus has entered the cervical canal and if abdominal pressing has not started. Before administering the product it is important to ensure there are no mechanical obstructions (e.g. oversized foetus). If present, obstructions must be removed prior to product administration.	44
Doxycycline	20% solution, 20% powder.		Hypersensitivity to tetracycline, birds with impaired renal/liver function.	Concurrent administration with penicillin cephalosporin, quinolones and cycloserine. W/P: Meat 2 days; eggs- nil.	14,19,30,33,5,7,139,163,16, 8,169

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Doxycycline hyclate	100 mg, 200mg, 300mg, 400mg tablet.	Do not administer to animals with hypersensitivity to tetracycline, impaired liver function, layers producing egg for human consumption.	GI upset, including nausea and vomiting. Overdose can result in acute sometimes fatal degeneration.	Do not combine with bactericidal agents like Penicillin and cephalosporin. Meat 8 days.	135,138,139,141,142,143,145
Enrofloxacin/Norfloxacin	Oral suspension, 2.5, 10 and 25% and Powder, 10%.	Impaired liver and/ or renal function, hypersensitivity.	Arthropathy.	Tetracycline, chloramphenicol, macrolides and lincosamides. W/P: Meat 12 days. Poultry during treatment and 8 days thereafter. Milk: 7 days.	19,24,132,139,141,169,173
Erythromycin	Powder, 5, 20 and 30%.	Animals with impaired liver function	Allergic reaction, diarrhea.	Theophylline, Warfarin and beta-adrenergic drug. W/P: Meat 6 days, Egg 6 days.	14,19,22,24,43 ,57,60,61,79, 107,132,142,143,145,169,194
Florfenicol	Solution for injection.	Do not administer to boars intended for breeding. Do not administer in cases of previous allergic reactions to florfenicol.	Commonly observed adverse effects are transient diarrhoea and/or peri-anal and rectal erythema/oedema which may affect 50% of the animals. These effects can be observed for one week. Transient swelling lasting up to 5 days may be observed at the site of injection. Inflammatory lesions at the injection site may be seen up to 28 days.	None known. W/P: Meat and offal*: 18 days.	37,196,199

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Flumequine	10% and 20% liquid in oil.	Animals with hypersensitivity to flumequine. Administration to animals with a seriously impaired hepatic and/or renal function; do not use for layers producing eggs for human consumption	Hypersensitivity to flumequine, impaired liver and/or renal function, concurrent administration with tetracycline, chloramphenicol, macrolides, and lincosamides.	Flumequine was found to have no effect on theophylline pharmacokinetics. The simultaneous administration with trimethoprim, sulphonamides, furazolidone, phenylbutazone, acetylsalicylic acid and hydrocortisone may accelerate the excretion of flumequin. Simultaneous administration with colistin sulphate decrease the bio-availability of orally administered flumequin. W/P Meat 3 days; poultry 3 days.	139,141
Fumagillin 100 mg/ gallon of sugar syrup			Infection could not be completely eliminated.	Fumagillin is less effective when fed with powdered sugar, extender patties, candy, or pollen supplements. **Precaution:** Protect the mix from exposure of direct sunlight.	211
Furazolidone	Powder, 20, 25, 98 and 99%.	Systemic administration.	Hyperestheisia and inappetence.	W/P: meat 7 days.	196

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Gamithromycin	Solution for injection.	Do not use in cases of hypersensitivity to macrolide antibiotics or to any of the excipients. Do not use this veterinary medicinal product simultaneously with other macrolides or lincosamides.	Visible injection site swellings associated with occasional slight pain may develop very commonly in cattle for one day. The swellings typically resolve within 3 to 14 days. Mild to moderate injection site swelling has been reported commonly in sheep and pigs, with occasional slight pain evident for one day in sheep.	Cross resistance may occur with other macrolides. Avoid simultaneous administration of antimicrobials with a similar mode of action such as other macrolides or lincosamides. Not authorized for use in lactating and pregnant animals within 2 months (cows, heifers) of expected parturition. W/P: Meat and offal: Cattle: 64 days Sheep: 29 days Pigs: 16 days	38,61
Gentamicin/ Gentamicin Sulfate	Injection; 40, 50 and 100 mg/ml. For large animals (cattle and pigs) 2.5 ml/100 kg q 12 hours. For dogs, 0.5 ml/10 kg twice daily.	Oral administration, in animals with renal failure, anaerobic infections and reserved for serious identified diseases.	Long-term application, as well as larger doses of gentamicin may cause damages to kidneys, hearing or balance.	Calcium gluconate, heparin sodium, sodium bicarbonate, IV and tylosin. W/P: meat and milk during treatment, as well as meat 72 days after the last medication.	19,21,24,60,108,109,110, 112,114,144,162,166,169,171
Isoniazid	20 mg/kg.		Hepatotoxic, cause peripheral neuropathy and is also both enzyme inducer and inhibitor.		14,40,119
Lasalocid sodium	15% Feed premix powder.	Should not to be given to layers that produce eggs for human consumption.	Overdose may cause inappetence.	W/P: Meat, 5 days	151
Lincomycin hydrochloride	Injection, 113.4 mg/ml.	Hepatic impairment.	May produce occasional vomiting and diarrhea.	Kaolin mixture, muscle relaxants, neostigmine	19,24,82,86,90
Lincomycin + Neomycin	Intramammary solution.	This product should not be used concomitantly with macrolides		W/P: Meat: 3 days. Milk: 84 hours.	43,79

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Lincomycin + Spectinomycin	White pale powder for use in drinking water	Do not use in case of hypersensitivity to the active substances or any of the excipients. Do not use in case of hepatic dysfunction Do not allow rabbits, rodents, horses or Ruminants, laying hens	Cases of diarrhoea or soft faeces and/or perianal region inflammation Rare cases of irritability / excitation, skin rash/ pruritus Allergic/ hypersensitive reactions were also observed.	The combination of lincosamides and macrolides is antagonistic, Combination with anaesthetics may lead to possible neuromuscular blocking, Do not administer with kaolin or pectine as they impair lincomycin absorption W/P: Pigs: Meat and offal: Zero days. Chickens: Meat and offal: 5 days.	132
Menthol crystals				Remove all surplus honey and empty supers. Honey for bee food may remain. **Note:** All colonies in an apiary receive mite treatment at the same time.	213
Neomycin (IMI)	Powder, 20, 30, 70 and 100%.	Myasthenia gravis.	Nephrotoxicity, ototoxicity, neuromuscular blockage, diarrhea, if dosage is higher.	Phenylmethyl penicillin and warfarin W/P: Pigs meat 14 days.	21,43,85
Neomycin sulphate + Penethamate hydriodide + Procaine benzylpenicillin	Intramammary suspension.			Milk may only be taken after 96 hours post calving in cows with a dry period of more than 50 days. Animals may not be slaughtered until 28 days from the last treatment.	43
Nystatin	ointment or cream	Nystatin should not be used in animals with known hypersensitivity or allergy to the drug.			26,147

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Oxytetracycline/ Tetracyclines	Powder: 5, 10, 20, 25 and 50%.	Renal impairment, last 2-3 weeks of gestation in pregnant animals and up to 4 weeks of age in neonates.	Diarrhea, gastric upset.	Antiacids, dairy products, calcium salts, iron salts, magnesium salts, zinc salts and warfarin, W/P: Meat 10 days.	7,13,14,15,20,21,30,32,33,38,43,46,57,59,61,62,63,65,75,76,77,79,80,82,90,96,107,109,110,111,112,117,118,120,141,142,161,162,169,171,172,195
Oxytetracycline Hcl	10mg/ml, 20mg/ml solutions.	Do not administer for animals with severe impaired renal or liver function. Not to be used in early gestation, in the last trimester of gestation, as well as very young animals not older than three weeks.	Have antianabolic effect and may cause azotemia. It may also cause metabolic acidosis and electrolyte imbalance. Prolonged application causes liver damage. In young animals, deposits in bones and discolouration of teeth may occur. Hypersensitivity reactions may be observed vitamin B and vitamin K deficiencies. In honey bees, treatment delays vegetative growth but does not eliminate completely.	Do not combine with other antibiotics such as penicillin, cephalosporin, Quinolones and cycloserine. Poultry 7 days, eggs 5 days. For honeybees, apply at least 4 weeks before the main honey flow. W/P: Meat during the treatment and 21 days thereafter.	38,62,96,108,109,139,140,144,145,
Oxytetracycline 200 mg/ colony	Soluble powder		Treatment delays vegetative growth but does not eliminate completely.	W/P: Apply at least 4 weeks before the main honey flow. **Precaution:** commence one month before the first major nectar flow and again after the honey crop has been removed.	210
Penethamate	Suspension	Do not administer intravenously.			43
Penicillin	Injection in 200,000 IU/ml to 400,000 IU/ ml.	Hypersensitivity.	Allergic reactions.	Chloramphenicol, tetracycline and phenylbutazone. W/P: meat 10 days.	8,14,16,17,38,43,63,85,86,99,111, 145,169

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Phenoxymethylpenicillin	Powder for oral solution	Do not combine with bacteriostatic antibiotics	penicillins may cause vomiting, diarrhoea and alter gut flora with selecting resistant bacteria	Meat and offal: 2 days. Eggs: zero days.	147
Potentiated Sulfonamides				W/P: milk 72 hr; meat 12 days.	43
Procaine Penicillin + Dihydrostreptomycin Sulfate	Injection, 200,000 IU and 200mg/ml respectively.	Hypersensitivity and should not be administered by intrathecal injection	Allergic reactions.	chloramphenicol, tetracycline, phenylbutazone, calcium gluconate, heparin sodium, sodium bicarbonate and tylosin	18,20,21,43,61,62,64,76,79,80,96,98, 107,112, 120,121,158,161,162,163, 169,170,171,172,188
Procaine Penicillin G	Injection 100ml, 250ml, 500ml.	Do not use in calves to be processed for veal.	Allergic or anaphylactic reactions.	Avoid giving penicillin in conjunction with bacteriostatic drugs such as tetracyclines. W/P: Meat: Cattle - 14 days, Sheep - 9 days, Swine - 7 days. Milk: 48 hours.	3,7,13,14,15,16,17,22,25,37,38,43,46,47,57,60,63,65,79,96,98,99,107,108,110,113,114, 116,117,120
Quinapyramine sulfate	1 g powder.		Overdosing can cause tremors, salivation, collapse and death		122
Spectinomycin		Birds remain carriers despite treatment			132,140
Spectinomycin dihydrochloride pentahydrate					38
Streptomycin sulfate/ Dihydrostreptomycine sulphate	Injection 200mg/ml, 250mg/ml, 72g/100g, Oral suspension 12.5%.	In animals suffering from myasthenia gravis.	Nephrotoxicity, ototoxicity, neuromuscular blockage. At higher dosage calves may develop diarrhea.	Calcium gluconate, heparin sodium, sodium bicarbonate, IV and tylosin W/P: meat 21 days; milk 2 days	13,14,18,21,42,46,59,63,109, 110,111,114,118,147

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Streptomycin plus penicillin	Intramammary suspension, 250mg +300001U/dose.	Hypersensitivity to penicillin and cephalosporin.	Allergic reactions.	Withdrawal period during treatment period.	21,42,43
Sulfachlorpyrazine sodium monohydrat		Should not be given to layers that produce eggs for human consumption.	Temporary egg drop, wind eggs and hypersensitivity reaction.	Do not combine with antacids as this may affect bioavailability. W/P: meat 7 days. eggs 3 days, Or	150
Sulfadiazine-Trimethoprim	Tablet , 100+ 20, 400+80 in mg; Injection, 200+40 and 400 + 80 in mg/ml; Powder, 500+50mg/g, 400+80 mg/g and 33.3g+6.67mg/100gm; Oral suspension, 50+10, and 400+80 mg/ml.		Crystallization in urinary tract, hypersensitivity and anaphylaxis for all species. In dogs: hemolytic anemia, anorexia, cutaneous drug eruption, diarrhea, facial swelling, hepatitis, hypothyroidism, keratoconjuctivitis, neurologic disorder, polyarthritis and polydipsia. *Cats:* salivation, thyroid function changes transient vomiting.	Detomidine and halothane. Withdrawal is 7 days for meat and don't use in lactating camel.	19,24,98,107,113,161,162
Sulfaquinoxaline	Granules, 50%+16.5%.	Intrauterine administration.	Crystallization in urinary tract, hypersensitivity and anaphylaxis.	Detomidine and halothane.	150
Sulfadiazine		Intrauterine administration.	Crystallization in urinary tract, hypersensitivity and anaphylaxis.	Detomidine and halothane.	70,181

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Sulfadiazine and pyrimethamine		Intrauterine administration.	Crystallization in urinary tract, hypersensitivity and anaphylaxis and the other relate to folate deficiency including agranulocytosis, megaloblastic anemia and thrombocytopenia; dehydration; gastrointestinal toxicity (diarrhea, occasionally bloody; vomiting); anorexia/ decreased appetite; weakness; weight loss.	Detomidine and halothane.	70,181
Sulfamethazine-trimethoprim		Intrauterine administration.	Crystallization in urinary tract, hypersensitivity and anaphylaxis.	Detomidine and halothane.	32,89, 138,148, 143,165,166,169,170,171172,
Sulphachloropyrazine	Powder, 10%.	Do not administer for animals with severe impaired renal or liver function. Should not be given to layers that produce eggs for human consumption.	Temporary egg drop, hypersensitivity reactions.	Do not combine with other antacids. W/P: Meat 7 days, eggs, 3 days.	140
Sulphadimethoxine	Injection 330, 333 and 160 mg/ml, Powder, 25 %.	In pregnant and lactating animals	IM injection cause local pain and inflammation.	Thiopentone sodium and warfarin.	38
Sulphadimethoxazole	Powder, 10%.	Should not be given to layers that produce eggs for human consumption.		W/P: Meat 4 days.	139
Sulphadimidine	Bolus, 5 g; injection 330, 333 and 160 mg/ml; powder, 8, 10, 16, 20, 25 and 30%.	Pregnant and lactating animals.	Crystallization in urinary tract, cutaneous eruption, hypothyroidism and idiosyncratic toxicosis.	Thiopentone sodium and warfarin.	20,21

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Sulfadimidine sodium	Bolus, 5 g; injection 330, 333 and 160 mg/ml; powder, 8, 10, 16, 20, 25 and 30%.	Pregnant and lactating animals.	Crystallization in urinary tract, cutaneous eruption, hypothyroidism and idiosyncratic toxicosis.	Thiopentone sodium and warfarin. W/P: Poultry meat and offal 14 days. Do not administer to laying hens	8,150
Sulphamethoxine	Bolus, 5 mg; Injection 330, 333 and 160 mg/ml; Powder, 8, 10, 16, 20, 25 and 30%.	Pregnant and lactating animals.	Crystallization in urinary tract, cutaneous eruption.	Thiopentone sodium and warfarin. W/P: Meat 5 days and milk 60 days.	119
Sulphonamides (Sulphadimethoxazole, Sulphaquinoxalene, Sulphamethazine and sulphaquinonxalene).		Should not be given to layers that produce eggs for human consumption.		W/P: Meat 4 days.	139
Tiamulin	10% and 20% Solution.	Not to be used in pregnant sows (during the early gestation period - the first four weeks after mating), lactating sows, breeding boars or egg laying hens	Irritation of skin and mucous membrane may occur after contact with powder.	Not to be used in conjunction with monensin, narasin and salinomycin, neither 7 days before nor after treating animals with listed medicine. W/P: Pigs meat, intestines and other edible tissues: 5 days for poultry: Meat, intestines and other edible tissues, 3 days.	132
Tilmicosin		Not authorised for use in laying birds producing eggs for human consumption. Do not use within 14 days of onset of the laying.		W/P: Pigs - 14 days Chickens - 12 days Turkeys – 19 days Calves - 42 days	19,39,77,142,143

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Tilmicosin (as phosphate)	Oral solution for use in drinking water	Do not administer in layers producing eggs for human consumption or to animals intended for breeding purpose, lactating dairy cattle or goats. Not for Iv injection.	In very rare cases, a decrease in water intake has been observed	Do not administer together with other macrolides or lincosamides. W/P: Meat 12 days.	145
Trimethoprim-Sulphaquinoxalene sodium	Granules or suspension.	Should not be given to animals with impaired liver, kidney and hematopoietic organs function, as well as in animals hypersensitive to sulfonamides and/or trimethoprim. Not to be administered for chickens producing eggs for human consumption.		Other sulfonamides or coccidiostats should not be administered concurrently; should not be used for long term treatment; W/P: Pigs, meat and intestines - 10 days. Calves meat and intestines- 14 days. Precautions: adequate water should be given	140,144,150
Trimethoprim-sulfadiazine	Bolus, 100+ 20, 250 + 50, 400+80 in mg; injection, 200+40 and 400 + 80 in mg/ml; powder, 10 + 2%; oral suspension, 50+10, and 400+80 mg/ml.		Crystallization in urinary tract, hypersensitivity and anaphylaxis for all species; local pain and swelling.	Detomidine and halothane. W/P: meat 10 days; milk 4 days Caution: Animals should have a good water supply.	19,24,113
Trimethoprim-sulfadoxine	Bolus, 5 g; injection 330, 333 and 160 mg/ml; powder, 8, 10, 16, 20, 25 and 30%.	Pregnant and lactating animals.	Crystallization in urinary tract, cutaneous eruption, hypothyroidism and idiosyncratic toxicosis.	W/P: Meat 10 days; milk 4 days.	22,24

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and Withdrawal period (W/P)	Page number
Tulathromycin	Solution for injection	Do not use in cases of hypersensitivity to macrolide antibiotics or to any of the excipients. Do not use simultaneously with other macrolides or lincosamides	Pathomorphological injection site reactions (including reversible changes of congestion, oedema, fibrosis and haemorrhage) are very common	Cattle meat and offal: 22 days. Pigs (meat and offal): 13 days. Sheep (meat and offal): 16 days	21,38,61
Tylvalosin	granular powder	None	None known	W/P: Meat and offal- 2 days.	86
Tylosin	Powder; 10, 20 and 30%. 200mg/ml solution.	Do not use in animals with impaired liver function. Not to be used in horses and small herbivores (rabbit, guinea pig, hamster, gerbil)	Allergic reaction. Anorexia, vomiting and diarrhea in dogs and cats may occur. In severe, and even life-threatening diarrhea in horses and cattle.	Theophylline, Warfarin and beta-adrenergic, **Caution:** store below 40ºC. W/P: Meat 2 days; egg 4 days	18,38,75,76,86,132,143
Thiamphenicol	Cutaneous spray, solution	Do not use in cases of hypersensitivity to the active substance or to any of the excipients Do not use on the udder of lactating animals if their milk is intended for human consumption.	None known	None known. Meat and offal: - horses, cattle, goats, sheep, rabbits: zero days. - pigs: 14 days. Milk: 0 hours.	61

Annex 6. Antiparasites

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Afoxolaner	Chewable tablets	Do not use in cases of hypersensitivity to the active substance or to any of the excipients.	Most reported adverse reactions were self-limiting and of short duration	None known, Not applicable.	152,182,184
Albendazole	Bolus, 150, 200, 250, 300, 600, 1000, 1125, 1500, 1875 and 2500mg; suspension, 1.5, 2.5, 5, 10, and 12.5%; powder, 20 and 30%; paste, 15gm.	Teratogenic and embryotoxic when administered at early gestation period.	Hypersensitivity.	W/P: Meat 14 days; milk 3 days.	29,40,67,68,78,101,178
Amicarbalide 50%					31
Amprolium / Amprolium HCl	Powder, 20, 30 and 60 % and Oral solution, 38.4 mg/ml/ powder in 73.4% cereal carrier.	Mixing with other medicinal products or substances having similar effect. Do not use for laying birds.	Interferes with egg quality and production.		32,69,89,150,151
Buparvaquone	Injection	Do not use subcutaneously or intravenously. Theileriosis has severe depressant effects on the immune system. Therefore, it is recommended that any vaccinations be delayed until the animal has recovered.	Localised, painless, oedematous swelling may occasionally be seen at the injection site.	W/P: Meat - 42 days Milk - 48 hours	33
Clindamycin	25, 75 or 150 mg capsules.	Should not be used in animals with hepatic or renal function impairment.	Sensitivity to lincomycines; in cats, lip smacking in oral solution, salivation.	Anesthetics such as enflurane, halothane, isoflurane, methoxyflurane, or neuromuscular blocking agents resulting in respiratory depression or paralysis; chloramphenicol or erythromycin.	181

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Clopiodol	Powder, 6%	Egg-laying chicken should not be treated with the drug; overdose might cause inappetence; concurrent administration with other anticoccidial drugs is contraindicated.		W/P: Meat for least 7 days.	150,151
Clorsulon	Solution for Injection.	This product is not to be used intramuscularly or intravenously. This product is registered for use in cattle only. Do not use in other species as severe adverse reactions, including fatalities in dogs may occur. Do not use in animals with known hypersensitivity to the active ingredient or any of the excipients.	Transitory discomfort has been observed in some cattle following subcutaneous administration. Soft tissue swellings may occur at the site of injection. These reactions resolve over time without treatment.	No interactions have been identified with other products W/P: Meat and offal- 66 days. Not permitted for use in animals producing milk for human consumption, including pregnant animals intended to produce milk for human consumption.	29
Closantel			May cause anorexia, labored breathing, recumbency, general weakness, decreased vision or blindness with prolonged use.		29,67,77
Coumaphos	Wettable powder, 50%.				7,72,124,152,153,214
Decoquinate				Do not mix with other anticoccidials W/P: Meat, at least one day; milk, avoid drinking milk from treated animals.	32,69,89
Diclazuril	oral suspension	Do not use in cases of hypersensitivity to the active substance or to any of the excipients	In very rare cases, adverse events causing diarrhea, with possiblenpresence of blood lethargy and/ or neurological troubles (agitation, recumbency, paresis) have been reported.	D/I: None known W/P: Meat and offal: Lambs: zero days Calves: zero days	32,69,150,207

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Dicyclanil	Pour-on suspension	Do not use in case of hypersensitivity to the active substance or to any of the excipients	None known	None known. Meat and offal: 7 days. Not authorised for use in animals producing milk for human consumption	74
Dimetridazole	Tablet 200 or 400mg, Injection 5mg/ml.		Central nerve system depression.	Phenobarbitone, phenitoin, warfarin, should not be used in poultry meant for human consumption.	151
Diminazene aceturate	Powder/ granule for injection, 1.1 gm., 1.05 gm., 496 mg and 444 mg; injection solution, 35 mg/ml.	Hypersensitivity to diminazene and should not be administered to animals with impaired renal and hepatic infections. Diminazene aceturate is toxic at >3.5 mg/kg, thus it is not used in camels.	Hypersensitivity reaction, sweating, salivation, tremors, sometimes nervous signs and fatty degenerative changes in organs with multiple therapeutic doses.		34,69,102,103,122,179,180
Doramectin	Solution for injection.	The product has been formulated specifically for pigs. It should not be administered to other species as severe adverse reactions, including fatalities in dogs, may occur. Do not use in case of hypersensitivity to the active substance or any of the excipients.	None have been observed.	None known W/P: Meat and offal 56 days.	36,72,73,77,78,90
Emodepside + Praziquantel	Spot-on solution.	Do not use in kittens under 8 weeks of age or weighing less than 0.5 kg. Do not use in cases of hypersensitivity to the active substances or to any of the excipients.	Salivation and vomiting may occur in very rare cases	Co-treatment with ivermectin and other antiparasitic macrocyclic lactones, erythromycin, prednisolone and cyclosporine) could give rise to pharmacokinetic drug interactions. Not applicable.	177,179

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Eprinomectin	0.5% w/v Pour-on Solution.	This product is formulated only for topical application for beef and dairy cattle,	None known.	None known, W/P: Meat and offal- 15 days, Milk - Zero hours/days	29,36
Fenbendazole	Bolus, 250 and 750 mg; Suspension, 2.5, 5, 10, and 12.5%.	Teratogenic and embryotoxic when administered at early gestation period	Hypersensitivity	W/P: Meat 30 days and milk 5 days.	29,40,68,78,87,88,89, 92,101, 177
Flubendazole		Early gestation.	Hypersensitivity.	Meat withdrawal is 14 days,	87,88
Fluralaner	Chewable tablet	Do not use in cases of hypersensitivity to the active substance or to any of the excipients	Mild and transient gastrointestinal effects such as diarrhoea, vomiting, inappetence and drooling are commonly observed in clinical trials.	Fluralaner is highly bound to plasma proteins and might compete non-steroidal anti-inflammatory drugs (NSAIDs) and warfarin. W/P: Not applicable.	182,184
Fumagillin				Fumagillin is less effective when fed with powdered sugar, extender patties, and candy or pollen supplements.	211
Homidium	Tablet for injection, 250mg.		Local swelling at injection site and transient lameness.		34
Imidocarb dipropionate		Decrease the dose in animals with hepatic dysfunction and cardiac disease.	Teratogenic and carcinogenic at high doses.		30,69,179
Isometamidium	Powder for injection, 125 mg and 1 gm.		Local transient reaction at injection site.		34,103
Isometamidium chloride	Powder for injection, 125 mg and 1 gm.		Local transient reaction at injection site. Most *T. evansi* can have innate resistance.		122

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Ivermectin	Bolus, 1.72 gm and 5 gm; Suspension, 800 mcg/ml; Powder or granule, 0.2%; Pour on, 5 mg/ml; Injection, 10 mg/ml; Paste, 1.876, 0.2 and 8%, Injection, 10 mg/ml.	Calves less than 12 weeks of age and lactating animals, tablet is contraindicated and Coli breeds are sensitive for more than 0.006mg/kg.	Ataxia, depression, tremors, mydriasis, listlessness, musculoskeletal pains, edema of face or extremities, itching and popular rash.	W/P: Meat 28 days and do not use in lactating animals.	28,29,35,36,40,68,72,73, 77,78,87,88,89,90,92,97, 100,101,103,123,124,125, 152,160,176,178,182
Lasalocid sodium	15% Feed premix powder.	Should not to be given to layers that produce eggs for human consumption.	Overdose may cause inappetence.	W/P: meat, 5 days.	151
Levamisole	Oral tablet, 75mg/ml S/C or I/M injection.	not to be used in animals hypersensitive to levamisole, lactating cows and sheep, whose milk is used for human consumption	Frothy nasal discharge, hypersalivation, excitedness or tremor, licking, and head jolting. transient excitedness, coughing or vomiting may occur.	Interacts with ganophosphorous, carbamic compounds or compounds similar to nicotine (in effect), such as: diethylcarbamazine citrate, pyrantel, and morantel. Meat and edible tissues: 28 days.	28,29,40,68,78,88,178
Mebendazole	Tablet, 100mg; suspension, 4 and 10%.	early gestation	Hypersensitivity.		177,178
Milbemycin oxime + Praziquantel	Tablet	Do not use in dogs weighing less than 5 kg. Do not use in case of hypersensitivity to the active substances or to any of the excipients.	In very rare occasions, hypersensitivity reactions, systemic signs (lethargy), neurological signs (muscle tremors and ataxia) and/or gastrointestinal signs (emesis, diarrhoea, anorexia and drooling) have been observed in dogs after administration.	caution should be taken in the case of concurrent use with other other macrocyclic lactones. Not applicable	177,179

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Monensin		High dose may result in reduced weight gain.	At high dose, inappetence, anorexia, ruminal atony, depression, weakness, ataxia, diarrhea, dyspnea, prominent jugular pulse, ventral edema, rear leg weakness, torticollis and death may occur.	Do not mix with drugs having similar action; don't treat animals with tiamulin for at least 7 days. **Precautions:** Avoid direct contact with skin, wash thoroughly after handling the product.	2,3,32,69,70,150
Monepantel	Oral solution	None	None	None known 7 days. Not authorised for use in animals producing milk for human consumption.	68
Moxidectin	Oral solution.	Do not use in cases of known hypersensitivity to the active substance or to any of the excipients.	None known.	The effects of GABA agonists are increased by moxidectin W/P: Meat and offal 14 days, Milk- 5 days.	35,36,68,100 ,101,124,176,182,183
Moxidectin + Triclabendazole	Pour-on solution.			W/P: Meat and offal- 143 days Do not use in cattle of any age intended to produce milk for human consumption.	35
Nitroscanate	Film coated table	Do not repeat treatment if vomiting occurs shortly after dosing. Do not administer to sick or convalescing animals. Do not use in puppies of less than 3 weeks of age. Do not use in cases of known hypersensitivity to the active substance.	When the product is not administered as recommended occasionally vomiting may occur	None known Not applicable	177,179

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Nitroxynil	Solution for injection.	Do not use in animals with known hypersensitivity to the active ingredient. Do not exceed the stated dose. Do not use in dogs as fatalities have been reported.	Small swellings are occasionally observed at the injection site in cattle. No systemic ill effects are to be expected when animals (including pregnant cows and ewes) are treated at normal dosage	No signs of incompatibility are to be expected if administered to cattle or sheep concurrently with therapeutic doses of levamisole and thiabendazole, or with clostridial vaccine.	29,67
Oxfenbendazole	Bolus 450, 1000 and 2000 mg, powder or granule 5%.	Early gestation	Hypersensitivity.	W/P: Meat 14 days; milk 48 hours.	29,68,78
Oxyclozanide	Bolus, 340, 450, 1000 and 2700 mg; Suspension 34mg/ml, 3% with 1.5% levamisole.		Although oxyclozanide has wider margin of safety, inappetence, loss of body weight and milk yield, dullness and loosening of feces and possibly diarrhea with increasing dose may occur.	W/P: Meat 28 days; do not use in lactating animals for human use.	29,67
Parvaquone					33
Piperazine	Tablets, 50 and 500 mg; powder, 65%; syrup, 100 mg/ml.	Do not use in animals with renal impairment.	Vomiting and diarrhea may occur at a higher dosage.		87,152,177
Piperazine diHCL	65% powder, 100mg/ml syrup.	Do not administer to animals with severe impaired liver and kidney function and hypersensitive to piperazine.	Overdosing may cause unrest, ataxia, tremors, apathy and paralysis.	Do not combine with organic phosphorous compounds like pyrantel and morantel.	152
Praziquantel	Tablets, 50 mg; Bolus, 3125mg.	Do not use in puppies under 4 weeks and kitten under 6 weeks of age.	Vomiting and transient pain.		177,178,179
Pyrantelpamoate	Bolus, 600mg; Liquid, 50 and 150 mg/ml.				177

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Quinapyramine sulfate / chloride	Powder for injection, 1 gm.		Overdosing can cause tremors, salivation, collapse and death.		103,122
Rafoxanide	Injection 7.5%, 25-100ml, drench or bolus.	Should not to be given to lactating animals by I/M or I/V injection.	Overdosed sheep and goats develop retinal lesions characterized by necrosis, loss of the photoreceptor layer and retinal separation.	Do not use in concurrent treatment with organophosphates and/or diethylcarbamazine. W/P Meat: Cattle-28 days, Sheep- 35 days. Milk: Not for lactating animals.	29,67,77,123
Sarolaner	Chewable tablets	Do not use in cases of hypersensitivity to the active substance or to any of the excipients.	Mild and transient gastrointestinal signs such as vomiting and diarrhoea, transient neurological disorders such as tremor, ataxia or convulsion and systemic disorders such as lethargy, anorexia/ inappetence may occur in very rare cases	None known. But Sarolaner is highly bound to plasma proteins and might compete with non-steroidal anti-inflammatory drugs (NSAIDs) and warfarin. W/P: Not applicable.	182,184
Suramin			Leakage of the drug into the tissues may cause phlebitis.		103,122
Tetramisole	Bolus, 150, 600, 700, 1000, 1200, 1500 and 2000 mg; powder or granule, 10, 20 and 30%; Injection, 30 and 100 mg/ ml.	Within 14 days of treatment of organophosphorus compound or diethylcarbamate. Don't exceed dose 4.5 gm per animal.	Frothing, salivation, tremor, transient head shaking, licks of lip, urination, defecation, vomiting, ataxia, collapse and death due to respiratory failure.		29,68
Tinidazole	Tablets, 200 and 400mg; Injection, 5mg/ml; Powder for injection, 400mg/ml.		Central nervous system depression.	Cimetidine, phenobarbitone, phenytoin and warfarin.	179,181

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Number
Toltrazuril	Solution for use in drinking water.	Do not use in birds with hypersensitivity to the active substance or to any of the excipients.	None known.	None W/P: Chickens: Meat and offal: 16 days Turkeys: Meat and offal: 16 days Not authorized for use in poultry producing eggs for human consumption. Do not use in pullets beyond the 15th week of life.	
Triclabendazole	Bolus, 200, 250, 300 900 and 1800 mg; suspension, 5, 10, and 20%.		Higher doses are associated with inappetence, increased blood urea nitrogen, transient weight loss, and slight effect on motor activities.	W/P: Meat 28 days; milk 7 days.	29,35,67,203

Annex 7. Acaricides

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Amitraz	12.5% solution	Do not use in horses, dogs or cats. Do not uses in sheep producing milk for human consumption.	None at the recommended treatment rates.	None, W/P: Meat 1 day, milk 2 days.	36,72,73,124,160 ,182,183,184
Carbaryl (Sevin) 80% WP		Do not repeat treatment before 4 weeks		W/P: Meat 7 days.	153
Chlorfenvinphos spray	Liquid, 10% w/v Emulcifiable Concentrate (EC).			Combination of two or more organophosphorus compounds or drugs with anticholinesterase activity.	36,104
Chrotoxyphos	0.25%, spray				72

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Coumaphos	Wetable powder, 50%.	Do not apply to sick,convalescent or stressed livestock or animals less than 3 months old. Do not spray in a confined area.		C/I: Do not apply in conjunction with oral drenches or other internal medication such as phenothiazine, or natural or synthetic pyrethroids or other organic phosphates	72,124,152,153,214
Cypermethrin spray	EC, 5%, 10% w/v; Powder, 25%; Pour-on solution.	Do not use in the treatment of lambs less than 1 week of age or during hot weather.		**Cautions:** Wash udder of sprayed animals before milking apply only on unbroken lesions.	36
Cyromazine	Pour on solution.		Do not use in case of known hypersensitivity to the active substance. Do not use in sheep producing milk for human consumption.	**Special precautions** For external use only. The product should be applied before an anticipated blowfly challenge. Established strikes should be treated with an authorised product according to label instructions. Dirty sheep or lambs should be dagged prior to treatment. Sheep or lambs which scour after treatment should be dagged.	74

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Deltamethrin	1% pour on, EC, 5%, 12.5%.	None	Minor signs and discomfort with some cattle up to 8 hrs. after treatment.	Some organo-phosphorous insecticides can reduce metabolism rate and thus enhance deltamethrin toxicity. Therefore, it is not advisable to proceed with such combinations. W/P: Meat 3 days; milk nil; mutton 7 days; pork 21 days. **Caution:** Wash udder of sprayed animals before milking and administration	36,125
Deltametrin spray	Pour-on solution, 1% ; EC, 5%, 12.5%.		Minor signs and discomfort with some cattle up to 8 hrs after treatment	W/P: Meat 3 days; milk nil; mutton 7 days; pork 21 days. **Caution:** Wash udder of sprayed animals before milking and administration.	36
Diazinon	250 mg/ml solution, Liquid 15% w/v, 16.2%, 20% and 60% EC w/v.	Calves younger than 8 weeks, +lambs and kid goats younger than 6 weeks, and piglets younger than 4 weeks. Puppies younger than 12 weeks also must not be treated. Do not use in sick, weak, thirsty and exhausted animals.	May cause hypersalivation, skeletal muscle tremor, labored breathing, hypersensitivity to external stimuli, coordination disorder and diarrhea.	Combination of two or more organophosphorus compounds or drugs with anticholinesterase activity. Meat 35 days. Milk from treated animals should not be used for human consumption.	36,72,103,160
Diazinon spray	Liquid 15% w/v, 16.2%, 20% and 60% w/v EC.			Combination of two or more organophosphorus compounds or drugs with anticholinestrase activity Meat 14 days. **Cautions:** Provide adequate ventilation for operator	36,72,101,104,124

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Dichlorovos/ Tetrachlorvinfos					102,154
Fenvalerate spray	Emulsified liquid, 10%, 20% EC.				36,101
Flumethrin	1% pour on				125
Flumethrine spray	EC, 1% and 6%			W/P: Mutton nil; milk nil **Caution:** Do not use for control of bowl fly larvae.	36
Indoxacarb + permethrin	Spot-on solution	Do not use in cats as adverse reactions and even death can occur. Do not use in cases of known hypersensitivity to the active substances or to any of the excipients.	Transitory erythema, hair loss or itching at the application site were commonly observed. Gastrointestinal signs (e.g. emesis, diarrhoea or anorexia), reversible neurological signs (e.g. tremor or ataxia) or lethargy have been observed in very rare cases.	None known. Not applicable.	102,182

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Imidacloprid + Moxidectin	Spot-on solution	Do not use in kittens under 9 weeks of age. Do not use in cases of hypersensitivity to the active substances or to any of the excipients	Use of the product may result in transient pruritus in cats. On rare occasions greasy fur, erythema and Vomiting and local hypersensitivity reactions can occur.	During treatment with this drug no other antiparasitic macrocyclic lactone should be administered. No interactions between this drug and routinely used veterinary medicinal products. Not applicable.	124,176,182,183
Iodophores 3% spray		Concurrent use of other antiseptics and detergents.	Irritation to tissues.		20,27,61
Permethrin	Dusting powder, 1.05%; Emulsified concentrate 0.5%; Pour-on solution, 4 %; Shampoo, 1.05%			W/P: Meat 3 days; milk nil.	36,153,182
Phosmet					36,72
(S)-Methoprene + Fipronil + Amitraz	Spot-on solution	Do not use on sick (e.g. systemic diseases, diabetes, fever) or convalescent animals. Do not use on rabbits and cats.	Transient skin reactions at the application site (skin discoloration, local hair loss, itching, and redness) and general itching or hair loss may occur on rare occasions.	No data available. Not applicable.	160,182

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Selamectin	Spot-on solution	Do not use in animals under 6 weeks of age. Do not use in cases of hypersensitivity to the active substance or to any of the excipients	On rare occasions may produce a local temporary clumping of the hair at the application site and/or an occasional. appearance of a small quantity of a white powder. Very rarely, reversible neurological signs.	No interactions between selamectin and routinely used veterinary medicinal products or medical or surgical procedures were observed.	176,177,184
Trichlorphon					72

Annex 8. Supportive drugs

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Activated charcoal					150,221,225,228, 229,230
Calcium borogluconate	Solution for infusion and injection.	Do not use in case of hypersensitivity to the active substances. Do not use in animals suffering from hypercalcaemia, hyperparathyroidism, acidosis, severe kidney damage.	Rapid intravenous infusion may result in transient cardiac distress. Excessive amount of calcium salts may lead to hypercalcemia. Symptoms include anorexia, abdominal pain, constipation, muscle weakness, mental disturbance, renal calculi, cardiac arrythmia and coma. Too rapid administration of calcium IV is also associated with many symptoms of hypercalcemia, particularly cardiac symptoms, thus administer slowly (at least over 10-20 min) and monitor heartbeat. Hypercalcemia is usually associated with the parenteral route of administration but also if there is renal insufficiency. Administration of oral calcium avoids these side effects.	The use of admixtures should be avoided because of possible incompatibility with other substances in solution. W/P: Meat and offal: zero days. Milk: zero days	5,6,7,52

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Calcium borogluconate + Magnesium Hypophosphite Hexahydrate + 20% glucose					6
Cisapride		Do not use in animals with decreased liver function, obstruction of the intestine, cardiac arrhythmia or conduction disorders, during pregnancy or lactation.	GI symptoms including abdominal pain and diarrhea.	Ketoconazole, itraconazole, IV miconazole, or troleandomycin, erythromycin, fluconazole, clarithromycin, cimetidine, and ranitidine, anticoagulants, benzodiazepine tranquilizers or alcohol.	160,223
Diphenhydramine	25 mg cap.	Do not use in pregnant or nursing animals.	Drowsiness, dry mouth and urinary retention; rarely, vomiting and diarrhea.		94,160,161
Glycerol	Liquid	Contraindicated in patients with known hypersensitivity to glycerol.	Glycerol when taken orally may cause headache, nausea and vomiting, and less frequently, diarrhoea, thirst, dizziness and mental confusion.	D/I: None known.	48,52
Iron dextran		Do not use in animals with hypersensitivity, anemia, acute renal infections and oral iron supplement.	High dose has teratorgenicity and embryotoxic, weakness, prostration and anaphylactoid reaction.		82,177

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Lactated Ringer's solution	Dextrose 20gm + Sodium chloride 0.60gm + Potassium chloride 0.04gm + Calcium chloride 0.027gm + Sodium chloride 0.312gm/100ml.	Do not use in animals with: - Alkalosis of any origin - Oedema (hepatic, renal, or cardiac) - Overhydration, - Hyperkalaemia, hypernatraemia, hyperlactataemia - Hepatic insufficiency.	This veterinary medicinal product contains calcium, thus an effect on the heart cannot be ruled out.	This veterinary medicinal product is incompatible with Chlortetracycline, Amphotericin B and Oxytetracycline. Meat and offal: zero days. Milk: zero days.	94,122,159,160,161,179
Lugol's Iodine solution	Solution, (5% Iodine and 10% KI); 2%, 2.5%, 5%, 10%.	Kidney disease, chronic inflammatory skin disease marked by blisters, pregnancy.	Allergic reaction, like rash, hives, itching, red, swollen, blistered or peeling skin.	Diazepam diclofenac doxycycline, lidocaine, meloxicam, multivitamin with minerals.	112
Magnesium carbonate					3
Magnesium hydroxide			May cause diarrhea and may interfere with absorption of drugs administered PO.		3,7,230
Metoclopramide		Do not use in animals with GI obstructions, phenothiazines or narcotic analgesics.	Increases seizure effects and extra pyramidal effect.		160,161,162,163
Phenylbutazone	Injection, 200 mg/ml; Bolus, 25, 100 and 200 mg.	Prolonged use may cause gastrointestinal lesions.	Cardiac, hepatic, or renal impairment, anemia.	Methotrexate, phenytoin, suphonylureas, thyroxine and warfarin	9,31,92,99,161,166
Poloxalene			It has poor absorption, thus toxicity is unlikely.	W/P: Meat, 3 days, milk, nil Or	2,50

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Prochlorperazine			Adverse effects include sedation, depression, hypotension, hepatoxicity and extrapyramidal reactions.	Phenothiazinether CNS depression, Quinidine - additive cardiac depression, antidiarrhoeal and Antacids - reduced gastro-intestinal absorption, Epinephrine – vasodilation and tachycardia, Propranol – increase blood levels of both drugs.	160,161

Annex 9. Other drugs

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Acepromazine maleate	Injection, tablet.				159,161
Acetylpromazine	Injection, 2 and 10 mg/ml; Tablet, 10 mg or 25 mg.	Contra-indicated in pregnant animals.	At higher dose, profound cardiovascular effects occur.	Anti-epileptic drugs, antimuscarinic drugs, metoclorpramide, combination with any other CNS depressant drugs, may potentiate toxicity of organophosphates.	25,163
Acetylsalicylic acid (Aspirin)	Injection, 100 mg/ml; Bolus, 15.6 gm or 1.4gm.	In pregnant animals, gastrointestinal ulceration and hemorrhage.	Prolonged use may cause gastrointestinal lesions, disturbance in acid-base balance, and alterations in platelet and renal functions.	Acetazolamide, antiacid, diuretics, heparin, methotrexate, metoclorpramide, phenytoin and warfarin.	9,17,171,176,185
Adrenaline	1 mg/ml		Nervousness, dizziness and cardiac arrythmias.		161
Aminophyline		Do not use in animals with digestive, hepatic or renal function impairment.	Nausea, vomiting, diarrhea, anxiety, nerveousness, seizure, unable to sleep, fast heart rate and irregular rhythm.	Amlodipine, cimetidine, ciprofloxacin, enrofloxacin, erythromycin, ketamine, ketoconazole, phenobarbital, thiabendazole	173

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Atropine ophthalmic ointment		Do not use in presence of glaucoma.	Salivation in cats.		62,108,165,173,226,229
Carprofen	Tablets, 25 mg, 75 mg, or 100 mg.	Carprofen should not be used in dogs exhibiting previous hypersensitivity to Carprofen.	Vomiting, diarrhea, black, tarry, or bloody stool, Constipation, fatigue, weakness, gastrointestinal ulcers, sores in the mouth, fluid retention and weight gain, muscle cramps, seizures.		93,98,108,157,174
Cefovecin	Powder and solvent for solution for injection	Do not use in cases of hypersensitivity to cephalosporin or penicillin Do not use in small herbivores. Do not use in dogs and cats less than 8 weeks old.	On very rare occasions gastrointestinal signs, including emesis and/or diarrhoea, have been observed. In very rare cases neurological signs and injection site reactions have been reported after the use of the product.	Concurrent use of other substances that have a high degree of protein binding (e.g. furosemide, ketoconazole, or NSAIDs) may compete with cefovecin binding and thus may cause adverse effects. Not applicable	173
Chlorhexidine	0.05% solution		Irritant to tissue, skin, eye and mucus membrane.		7,60,106,115
Chlorhexidine digluconate + Miconazole nitrate	Shampoo	Do not use in case of hypersensitivity to the active substances or to any of the excipients.	Exceptionally a dog with atopy or a cat with allergic skin disease may develop a pruritic and/or erythematous reaction after treatment.	None known Not applicable.	173

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Chlorpheniramine maleate	tablet, liquid syrup, or liquid drops	Chlorpheniramine maleate should not be used in pets that are allergic to it or other similar antihistamines.	The most common side effect is sleepiness. In cats, this medication can cause excitement rather than sedation. Less common side effects include diarrhea, vomiting, lack of appetite, dry mouth and mild straining to urinate. Serious side effects include seizures, abnormal breathing, incoordination, muscle tremors, and coma.	The following medications should be used with caution when given with chlorpheniramine maleate: anticoagulants, MAOIs, phenytoin, or central nervous system depressants.	164
Chlorpromazine hydrochloride	Injection, tablet	Chlorpromazine should not be used in pets that are allergic to it or in pets with low blood pressure. It should be used cautiously in pets that are generally debilitated or have liver disease, heart disease, seizures, or aggressive behavior. Use cautiously in pregnant or lactating animals	tiredness, low blood pressure, low heart rate, or a tendency to react or startle to noises. In cats, higher doses can cause tremors, shivering, rigidity, lethargy, diarrhea, loss of anal sphincter tone, and loss of certain reflexes. Serious side effects include coma, agitation, seizures, abnormal heart rhythms, or dangerously low blood pressure.	The following medications should be used with caution when given with chlorpromazine: acetaminophen, antacids, antidiarrheal mixtures, bromocriptine, buspirone, cabergoline, calcium channel blockers, CNS depressant agents, desmopressin, diazoxide, dipyrone, dopamine, doxorubicin, epinephrine, laxatives, loop diuretics, opiates, organophosphate agents, paraquat, phenytoin, physostigmine, propranolol, QT prolonging agents, quinidine, or tricyclic antidepressants.	159
Codeine phosphate	tablet, 30 mg	In respiratory insufficiency and liver disease.	Sedation		166

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Dexamethasone	Injection 1or 2 or 3mg/ml and tablet, 250 mg.	Pregnant cows	Metabolic effects (e.g. osteoporesis), delayed wound healing, increased liability to infection and adrenal suppression.		4,10,17,21,24,52,64, 84,93,98,100,107, 172,174
Dimenhydrinate	Injection, tablet				159
Diphenhydramine	Injection, 10 mg/ml	Pregnant animals.	Incoordination, xerostomia and blurred vision.		94,160,161
Dopamine	Powder for injection, 250mg in vial.		Tachyarrhythmea.		161,224
Dopamine hydrochloride	Powder for injection, 250mg in vial.		Tachyarrhythmea.		94
Diphenhydramine hydrochloride	Injection, 10 mg/ml	Pregnant animals	Incoordination, xerostomia and blurred vision		94
Fluconazole	Tablets, 200 mg.	Hepatic impairment, pregnancy.	Hepatotoxicity, anorexia, nausea, vomiting with chronic therapy pruritus, alopecia, gynecomastia and sexual impotence.	Antacid, antimuscarinic drugs, cimitidine, ranitidine, phenytoin and warfarin.	26,27,105,180
Flumequine	20% liquid in oil.		Hypersensitivity to flumequine, impaired liver and/or renal function.	Concurrent administration with tetracycline, chloramphenicol, macrolides and lincosamides. W/P: Meat 3 days.	139,141
Flunixin Meglumine	Injection, 50mg/ml.	In pregnant animals, gastrointestinal ulceration and hemorrhage.	Prolonged use may cause gastrointestinal lesions, CNS depression, listlessness and anorexia.	Acetazolamide, antiacid, diuretics, heparin, methotrexate, metoclopramide, phenytoin and warfarin, W/P: Meat 4 days, Not for milking animals.	3,8,17,20,37,46, 47,92,93,98, 102,108,113,114
Furazolidone	Powder, 20, 25, 98 and 99%	Systemic administration	Hyperesthesia and inappetence	W/P: Meat 7 days.	196
Furosemide 5%	Injection, 50 mg/ml.	Renal failure with anuria, acute gromeluronephritis.	Hypokalemia, ototoxicity, hyponatraemia.	Corticosteroids, acetazolamides, aminoglycosides, cephalothins and cephalosporin. W/P: Meat 48hrs, Milk 48hrs	225

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Fusidic acid eye drops	Eye drops, suspension	The product should not be used in conjunctivitis cases associated with Pseudomonas spp. Do not use in cases of known hypersensitivity to the active substance or any of the excipients.	Allergic reactions or hypersensitivity to the active substance or the excipients might occur. Discontinue use if hypersensitivity to the product develops.	D/I: None known. W/P: Not for use in rabbits intended for human consumption.	97
Gonadotropin-releasing hormone (Gn-RH)	Injection, 4 mcg/ml.			W/P: None	44
Griseofulvin	Tablets, 125 mg, 7.5% powder feed additive	Hepatic impairment, pregnant animals.	High doses may cause hepatotoxicity, particularly in cats, leucopenia and hypoplasia.	Phenobarbitone, phenylbutazone, prostogens and warfarin.	175
Human chorionic gonadotropic (HCG)	Powder for injection, 500 units, 1000 units.		Anaphylactic reaction may occur.	**Precaution:** Prior to reconstitution, keep at 15-30°C; after reconstitution, keep refrigerated for not more than 30 days Antidote: Epinephrine hydrochloride and parenteral antihistamine.	45
Imidocarb	Solution for injection.	Decrease the dose in animals with hepatic dysfunction and cardiac disease.	Teratogenic and carcinogenic at high doses.	W/P: Meat and offal: 213 days. Milk: 21 day	31,33
Imidocarb diproprionate		Decrease the dose in animals with hepatic dysfunction and cardiac disease.	Teratogenic and carcinogenic at high doses.		30,69,102,179
Iodine tincture		Concurrent use of other antiseptics and detergents.	Irritant and stains tissue.		17,26,,53,60,96, 121,171,175,188

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Iodophores	3% spray	Concurrent use of other antiseptics and detergents	S/E: Irritant to tissue		20,27
Itraconazole			Hepatotoxic causing anorexia, vasculitis causing ulcerative dermatitis and cardiotoxicity.		27,105,175,180
Ketoconazole	Tablets, 200 mg; ointment (topical use), 10%.	In hepatic impairment, pregnant and lactating animals, decreased appetite, GI disturbances, changes in the hair coat, catarcats, infertility. Use not recommended in pregnant and lactating animals. Ketoconazole should not be used in animals with known hypersensitivity to the drug.	Hepatotoxicity, anorexia, nausea, vomiting, pruritis, alopecia, gynecomastia and sexual impotence.	Drugs that reduce stomach acidity may reduce ketoconazole absorption, antimuscarinic drugs, cimitidine, ranitidine, phenytoin and warfarin. It Has antagonistic effect with amphotericin B, thus should not be used in combination. W/P: Meat 5 days; Milk 2 days	26,27,173,175,180
Fluconazole	Tablets, 200 mg	Hepatic impairment, pregnancy.	Hepatotoxicity, anorexia, nausea, vomiting, with chronic therapy pruritus, alopecia, gynecomastia and sexual impotence.	Antacid, antimuscarinic drugs, cimetidine, ranitidine, phenytoin and warfarin	26,27,105,180
Ketamine hydrochloride	Injection, 10, 50 and 100mg/ml.	Sole anesthetics in horses, donkeys and dogs; hepatic or renal impairment; later stages of pregnancy in animals.	Hypotension, increased cardiac output; tachycardia, muscle twitching.		230

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Ketoprofen	Solution for injection	Do not use in animals where there is the possibility of gastro-intestinal ulceration or bleeding, in animals suffering from cardiac, hepatic, or renal disease. in cases of known hypersensitivity to ketoprofen or acetyl-salcylic acid or to any of the excipients.	In very rare cases intramuscular injection of ketoprofen can cause mild. transient, necrotic subclinical muscular lesions In very rare cases, in horses, transient local reactions.	Do not administer other non-steroidal anti-inflammatory drugs (NSAIDs) corticosteroids, anticoagulants or diuretics concurrently or within 24 hours of each other W/P: Cattle: Meat and offal: 2 days Milk: zero hours Horses: Meat and offal: 1 day Milk: Not authorised for use in mares producing milk for human consumption Pigs: Meat and offal: 3 days.	48,92
Lidocaine	Injection, 600 mcg/ml or 1mg/ml.			Ketoconazole, metoclorpramide and phenothiazine derivatives.	48
Luteinizing hormone (LH)	Injection, 250mcg/ml, 0.075mg/ml.	Concurrent use with non-steroidal anti-inflammatory drugs; pregnant animals abort.	Gastrointestinal disturbances, transient cardiovascular symptoms and rarely convulsion.	Sequential administration with oxitoxic drugs causes marked hypertension, vomiting, and severe dyspnoea. W/P: Meat 1 day; milk nil	45
Maropitant Citrate	solution for injection				159,160
Meloxicam		In gastroenteritis, pregnancy and low dose for cats	Vomiting, diarrhea, mucosal erosion, ulceration, black and tarry stool.	Digoxin, gentamycin, corticosteroid, tetracycline, caprtopril, enalapril, furosemide, insulin and spironolactone.	147
Methylprednisolone	Injection, 10mg.ml, 25mg/ml			Barbiturates, phenylbutazone, phenytoin, diuretics	158
Metoclopramide		Do not use in animals with GI obstructions, phenothiazines or narcotic analgesics.	increases seizure effects and extra pyramidal effect		160,161,162,163

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Miconazole	1% cream or lotion.		Local irritation, burning, oedema, erythema.		106,173,175
Natamycin	0.01% solution.			**Warning:** Do not expose treated animals to direct sunlight.	26,105
Oxytocin	Injection, 10 units/ml.	In dystocia due to obstruction.	Occasionally swelling and sloughing at site of injection.	Clenbuterol.	47,111,112,113,186
Phenobarbital	Tablet, 1/4, 1/2, and 1 grain (1grain=60mg), and 100 mg. Injectable 65 mg/ml and 130 mg/ml.	Dogs with Addison's disease, kidney disease, respiratory problems or existing liver disease should not be given this drug.	Lethargy, sedation, anxiety, restlessness, loss of coordination, increased thirst or appetite, weight gain, increased urination, anemia, liver damage (long-term use).		157,159
Phenylbutazone	Injection, 200 mg/ml; Bolus, 25, 100 and 200 mg.	Prolonged use may cause gastrointestinal lesions.	Cardiac, hepatic or renal impairment and anemia.	Methotrexate, phenytoin, suphonylureas, thyroxine and warfarin.	9,31,92,99,161,166
Potassium iodide	Powder in aqueous solution prepared before use (10%).	Iodides should not be given to milking cows whose milk will enter the human food chain.			13,14,26,28,96,97, 105,106
Povidone-iodine	Liquid, 5 and 10%.	Concurrent use of other antiseptics and detergents.	Irritant to tissue, can stain, may damage exposed nerves.		114,118,121,173,185
Pradofloxacin	tablet	Do not use in cases of hypersensitivity to the active substance or to any of the excipients.	Mild transient gastro-intestinal disturbances including vomiting have been observed in rare cases in dogs and cats.	Should not be administered concurrently with antacids, sucralfate, multivitamins or dairy products.	173
Prednisolone	injection, 10mg/ml, 25mg/ml.	In conjunction with vaccine; systemic corticosteroids are generally contraindicated for treatment of corneal opacity associated with ICH.	Prolonged therapy may precipitate signs of adrenal insufficiency and cautious in pregnant pets.	Acetazolamide, antidiabetic drugs, barbiturates, phenylbutazone, phenytoin, diuretics.	43,158,170,174,176,183, 184,185

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Prochlorperazine			Adverse effects include sedation, depression, hypotension, hepatoxicity and extrapyramidal reactions.	Phenothiazine, Quinidine, Antidiarrhoeal and Antacids, Epinephrine, and Propranol.	160,161
Promazine hydrochloride	Injection powder				159
Propylene glycol	Glucose in 40 or 50% solution; Propylene glycol as aqueous solution.		Propylene glycol may cause hyperosmolality, lactic acidosis, central nervous system depression and ataxia if given parenterally.		4,5,6,52,71
Prostaglandin	Solution for injection	Do not use in pregnant animals where the induction of abortion or parturition is not intended. Do not use in case of bronchospasms or spastic diseases of the gastrointestinal tract	Localised post-injection bacterial infections, which may become generalised, are occasionally reported. When used for induction of parturition, the incidence of retained placenta may be increased, depending on the time of treatment.	Simultaneous use of oxytocin and cloprostenol increases the effect on contractility of the musculature of the uterus. Synthesis of endogenous prostaglandins is inhibited in animals treated with non-steroidal anti-inflammatory drugs. W/P: Meat and offal: 1 day Milk: zero hours.	45,46,47,174,188
Sodium bicarbonate	Powder, 12.8gm/100gm.		Excess dose may cause systemic alkalosis especially if renal function is impaired resulting in muscle weakness and shortness of breath. Excessive sodium may cause diarrhea, abdominal cramp, tachycardia and pulmonary edema.		3,23,98,230

Drug name	Dosage Form (D/F)	Contraindication (C/I)	Side effect (S/E)	Drug interaction (D/I) and W/P	Page Numbers
Sodium hypochlorite				Hard or alkaline water will precipitate the active ingredient necessary for disinfection	27
Sodium iodide	Powder in aqueous solution prepared before use (10%).	Iodides should not be given to milking cows whose milk will enter the human food chain.			13,14,17,27,97,106
Tetanus antitoxin	Injection, 1000 IU, 10000 IU	Do not administer to patients with known allergy to tetanus antiserum.	May cause: hypersensitivity reactions, anaphylactic shock, Quinke oedema; serum sickness up to 10 days after injection.	**Precautions** : Ensure that the injection does not enter a blood vessel Administer following Besredka's method	25,65,99,113,119
Xylazine	Injection, 20 and 100 mg/ml.	Late pregnancy in animals.	Bradycardia, arrhythmia and reduced respiratory rate. Use with caution with other CNS depressants.		8,25,92,99

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CONTRIBUTORS FOR THE 1ST EDITION OF SVTG

A. Editors

1. Dr Ademe Zerihun	Epidemiologist (Chairman)
2. Dr Ephrem Engdawork	Pharmacologist (Secretary)
3. Dr Tiruneh Zerihun	DVM (Coordinator)

B. Contributors

1. Dr Ademe Zerihun	Epidemiologist (Chairman)
2. Dr Ephrem Engdawork	Pharmacologist (Secretary)
3. Dr Tiruneh Zerihun	DVM (Coordinator)
4. Dr Eshetu Yimer	Fish Pathologist (Member)
5. Dr Abayneh Dagne	Microbiologist (Member)
6. Dr Dagnet Yimenu	Vet. Pharmacologist (Member)

List of Validation Workshop Participants

	Name	Organization
1	Abdulefeta Ahemed	Somali Region livestock office
2	Abebe Fromsa	AAU, CVMA
3	Abera Gemeda	VDFACA
4	Abera Terfasa	Oromiya livestock office
5	Alemayehu Lemma	AAU, CVMA
6	Amanuel Assefa	World bank group/IFC
7	Asmamaw Kasaye	Donkey Sanctuary
8	Assef Redahi	Tigray Region Agriculture office
9	Atakilti Hadush	VDFACA
10	Banteyegegn Tamrat	
11	Dawit Kassaye	Addis Ababa Agricultural Commission
12	Eyob Eticha	Addis Ababa Agricultural Commission
13	Feyessa Begna	Jimma University
14	Habtamu Yohannes	
15	Hayat Seid	VDFACA
16	Hilina Getachew	
17	Jemere Bekele	Hawassa University
18	Kalkidan Shemilis	SNNPR livestock office
19	Kenenet Atenafu	NVI
20	Kidest Jifar	SNNPR livestock office
21	Mesfin Negash	
22	Sadiya Haji	Oromiya livestock office
23	Sisay Guda	Private practitioner
24	Solomon Gebere	NAHDIC

25	Solomon Kebede	VDFACA
26	Solomon Tarekegn	ALEMA Farms
27	Tadesse Kebede	Addis Ababa urban agriculture office
28	Takele Beyene	AAU, CVMA
29	Tamru Tessema	Haramaya University
30	Tenaw Andualem	UN-FAO
31	Teshome Jagema	Oromiya livestock office
32	Tewodros Tesfaye	EVA
33	Tewodros Tsige	Ethio-chicken poultry farm
34	Yohannes Tsegaye	VDFACA
35	Yegnaneh Anley	VDFACA

NOTES

Comments or suggestions regarding this version of SVTG should be sent to:

The VDFACA of Ethiopia
P.O. Box 31303, Tel.251-115524045,
e-mail: vdrcd@vdfaca.gov.et
Addis Ababa, Ethiopia

Veterinary Drug and Animal Feed Administration and Control Authority

Food and Agriculture Organization of the United Nations

IFC International Finance Corporation WORLD BANK GROUP

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