ANIMAL HEALTH

Level – III

Based on March, 2022 Version -4 Occupational Standard



Module Title: Identifying and Handling Non-Infectious Disease of Animals

LG Code: AGR ANH3 M03LO (1-4) LG (8-11)

TTLM Code: AGR ANH3 M03TTLM 0523v1

May 2023

Addis Ababa, Ethiopia

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Introduction to the Module

This module covers the knowledge, skills and attitude required to identify and treat different categories of non-infectious causes of animal disease. It also covers the ability to participate in prevention and minimization of its effect through community education and providing consultancy to other stack holders in the line of livestock industry.

LG #8	LO1. Develop general understanding on non-infectious diseases of animals

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following content coverage and topics:

- Introduction to non-infectious diseases
- Current status and economic implication of non-infectious animal disease
- Categories of non-infectious disease of animals
- Policies, legislations, regulations and directives in handling non-infectious disease
- Hazard identification and risk minimization during case handling

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, you will be able to:

- Define non-infectious animal disease and introduce clinical as well as economic impacts of non-infectious diseases.
- Explain Statistical information on the current condition and implication of the problem in Ethiopian context.
- Mention and explain the causes of different categories of non-infectious disease of animals
- Follow and implement working area policies, legislations, regulations and directives.
- Identify and minimize work place hazards and risk.

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the information Sheets
4. Accomplish the Self-checks

Information Sheet 1

1.1.Introduction to non-infectious diseases

Non-infectious animal diseases refer to any health conditions suffered by animals that are not caused by infectious agents such as viruses, bacteria, fungi or parasites. These diseases are usually related to the environment, nutrition, age, heredity, trauma, and other factors that can affect an animal's wellbeing. While infectious diseases can be caused by bacteria, viruses, fungi, or parasites that enter an animal's body and cause harm by replicating and damaging cells, non-infectious diseases are typically chronic conditions that develop slowly over time and may have a variety of contributing factors.

One of the most common examples of non-infectious diseases in animals is diabetes, which is caused by a lack of insulin production or an inability to properly use insulin. This can be genetic or caused by lifestyle factors such as obesity or poor diet. Controlling diabetes in animals requires monitoring blood sugar levels and providing insulin injections or other medications as needed, as well as making dietary changes to balance carbohydrate intake. Another example of a non-infectious disease is toxicosis, which occurs when animals ingest or are exposed to toxins such as pesticides, heavy metals, or poisonous plants. Symptoms can range from mild symptoms like lethargy or vomiting to severe organ damage or death. Preventive measures such as proper storage of chemicals and limiting access to potentially harmful areas can help reduce the risk of toxicosis.

Physical trauma is another common cause of non-infectious animal diseases. Fractures or other injuries can occur due to accidents or rough handling, and proper care such as immobilization or surgery is necessary for recovery. Preventive measures such as safe handling procedures and appropriately designed housing can minimize the risk of physical trauma.

Nutritional imbalances can also lead to non-infectious diseases. For example, energy deficiency can cause milk fever in cattle or hypoglycemia in dogs. Nutritional deficiencies such as vitamin or mineral deficiencies can cause a range of health problems. Proper feeding practices and balanced diets can help prevent nutritional imbalances and improve animal health. Inadequate nutrient intake may cause

nutritional deficiencies resulting in various health issues like anemia, growth retardation, reproductive problems, and weakening of bones among others.

Animals can also be affected by non-infectious respiratory diseases such as asthma caused by environmental pollutants or infectious diseases. Liver conditions affecting the liver such as cirrhosis and Hepatitis can occur from ingestion of toxins over long periods of time. Kidney disease, which often occurs in older dogs and cats, may be caused by various factors including age-related degeneration or exposure to toxins. Environmental factors such as temperature, humidity, and air quality can affect an animal's health. For instance, extreme heat or cold exposure may lead to heat exhaustion, hypothermia, dehydration or frostbite.

In conclusion, non-infectious animal diseases are a significant concern to veterinarians and pet owners alike. These diseases can affect any animal at any age or stage in life thus the importance of effective prevention measures including regular check-ups, maintaining optimal body condition, and providing a balanced diet. A proper diagnosis of the disease is also key in designing effective treatment plans which may involve surgery, medication, or lifestyle changes to alleviate symptoms and prevent their recurrence.

1.2.Current status and economic implication of non-infectious animal disease

- **Current status of non-infectious animal disease**

Non-infectious diseases continue to pose a significant risk to animal health and productivity in Ethiopia. Some of the most common ones include photosensitization, thiamine deficiency, and ingestion of teratogenic plants. These conditions can cause a wide range of symptoms depending on the type of animal and the severity of the illness. In some cases, the symptoms may be mild such as hair loss or skin lesions, while in other cases they may be much more serious and potentially life-threatening. These diseases also can cause economic losses for farmers and ranchers due to reduced productivity, increased mortality rates, and decreased reproductive rates. It's important to work with veterinary professionals for proper diagnosis and treatment of non-infectious diseases in animals. Additionally, preventative measures should be taken to avoid the economic losses associated with these conditions in the agricultural sector. Preventative measures such as proper feed management, weed control, and vaccination programs can help to reduce the incidence of these diseases.

- **Clinical impact of non-infectious Animal diseases**

Non-infectious animal diseases can have a significant clinical impact on animal health. These diseases can result in a wide range of clinical symptoms, depending on the specific condition and the severity of the disease. Some common clinical impacts of non-infectious animal diseases include:

- ✓ Reduced productivity: Non-infectious diseases can negatively impact animal performance, leading to reduced productivity in terms of growth rate, milk production, reproductive efficiency, and overall output.
- ✓ Nutritional imbalances: Some non-infectious diseases can lead to nutritional imbalances, which can result in a variety of clinical symptoms such as muscle weakness, poor growth, and decreased appetite.
- ✓ Metabolic disorders: Non-infectious diseases such as metabolic disorders can lead to significant disruptions in the normal metabolic processes of the animal, resulting in symptoms such as weight loss, muscle weakness, and neurological abnormalities.
- ✓ Toxicoses: Exposure to toxic substances can cause a range of clinical symptoms, including gastrointestinal distress, neurological issues, respiratory problems, and even death.
- ✓ Autoimmune disorders: Non-infectious autoimmune disorders can lead to chronic inflammation and tissue damage, resulting in a range of clinical symptoms such as joint pain, fever, and organ failure.

Effective prevention and control measures for non-infectious diseases can help to minimize their clinical impact on animal health and wellbeing.

- **Economic impact of non-infectious Animal diseases**

Non-infectious animal diseases can have significant economic implications on the agricultural sector, including livestock production and animal health.

- ✓ Reduced productivity: Non-infectious diseases can reduce the productivity and efficiency of animals, resulting in decreased production of meat, milk, wool, and other products. This can lead to significant losses for farmers and producers.
- ✓ Increased treatment costs*:* Non-infectious diseases often require specialized veterinary care and treatment, which can be expensive. In addition, prolonged illness can result in increased labor costs for farmers as they care for sick animals.
- ✓ Decreased market value: Animals that are affected by non-infectious diseases may have reduced market value due to lower quality or inability to produce desired products. This can further impact the profitability of farmers and producers.

- ✓ Risk of spreading disease: Non-infectious diseases can also increase the risk of spreading infectious diseases among herds or flocks, which can lead to significant losses and expenses associated with quarantine, treatment, and prevention measures.

It is therefore important for farmers and producers to implement preventative measures and work with veterinary professionals to minimize the impact of non-infectious diseases on their herds or flocks.

1.2.1. Worldwide information

Non-infectious diseases in animals are caused by a variety of factors such as genetics, environment, nutrition, or physical trauma, rather than by infectious agents. These diseases affect animals of all ages and species and can result in significant economic losses for livestock producers.

According to the Food and Agriculture Organization of the United Nations (FAO), some common non-infectious animal diseases include metabolic disorders such as milk fever and ketosis in dairy cattle, bloat in ruminants, abomasal displacement in dairy cows, and obesity in companion animals like cats and dogs. Other non-infectious diseases include nutritional deficiencies, such as copper deficiency in sheep and goats; toxicosis caused by exposure to toxins like mycotoxins or heavy metals; physical trauma leading to fractures or injuries; and endocrine disorders like diabetes that affect animals' ability to regulate glucose levels. Proper diagnosis and treatment by trained veterinary professionals is crucial for managing these conditions and improving animal health. Preventive measures such as minimizing exposure to toxins and providing a balanced diet that meets an animal's nutritional requirements can also help minimize the risk of non-infectious diseases.

1.2.2. Statistical information in Ethiopian

In Ethiopia, the Ministry of Agriculture provides information on non-infectious animal diseases and their prevalence. According to the Ministry's 2019 Annual Report, some of the most common non-infectious diseases in Ethiopia include:

- Nutritional deficiencies: Inadequate intake of nutrients such as minerals, vitamins, and protein can lead to deficiencies in animals, which can cause various health problems.
- Poisoning: Exposure to toxic substances such as pesticides, industrial chemicals, and contaminated water or feed can cause poisoning in animals.
- Reproductive problems: Various reproductive issues such as infertility, abortion, stillbirths, and birth defects can occur due to factors such as poor nutrition and genetics.

- Metabolic disorders: Some of the common metabolic disorders include ruminal acidosis, ketosis, and milk fever, which are caused by a disruption in normal metabolism in animals.

While specific statistics on the prevalence of these non-infectious animal diseases are not readily available, the Ministry of Agriculture has been working to prevent and control these diseases through various programs aimed at improving animal nutrition, promoting safer farming practices, and providing education and resources to livestock owners.

1.3.Categories of non-infectious disease of animals

Some categories of non-infectious diseases in animals include

- **Allergies:** in animals occur when their immune system overreacts to certain allergens, which can be anything from pollen and dust mites to certain foods, insect bites, or flea bites. The most common types of animal allergies are environmental allergies, which are caused by exposure to airborne allergens such as pollen, dust, and mold.

  Symptoms of environmental allergies in animals can include itchy skin, excessive scratching or licking, sneezing, coughing, and watery eyes.

  - ✓ Flea allergies are another common type of animal allergy, which is caused by an animal's hypersensitivity to flea saliva. Animals with flea allergies may experience intense itching and hair loss around the base of the tail and back legs.
  - ✓ Food allergies are also possible in animals, although they are less common than environmental or flea allergies. Symptoms of food allergies can include skin irritation, gastrointestinal upset, and ear infections. If an animal is suspected of having an allergy, it is important to consult with a veterinarian for proper diagnosis and treatment. Treatment options may include medications to relieve symptoms, allergy shots to desensitize the animal to certain allergens, and avoiding exposure to the allergen altogether.
- **Nutritional imbalance** (such as protein deficiency, carbohydrate engorgement, vitamin deficiency and mineral deficiency). Nutritional imbalances in animals occur when the animal's diet lacks certain nutrients necessary for their growth, health, and wellbeing, or contains excess amounts of others. The severity of the consequences of a nutritional imbalance can range from mild to severe depending on the duration of the deficiency and the nutrient involved. For example, a lack of protein can lead to reduced growth rates, while a lack of calcium can cause weak bones and muscle

spasms. Overconsumption of certain nutrients can also have negative effects on an animal's health, such as obesity or mineral imbalances. Common solutions for correcting nutritional imbalances in animals include dietary adjustments, feed fortification with vitamin and mineral supplements, and providing animals with access to a variety of high-quality feed.

- **Metabolic diseases**: Metabolic diseases are a group of conditions that affect the normal metabolic processes in animals, which can lead to an imbalance in their physiological state. Depending on the specific disease and animal species affected, the causes, symptoms, and treatments vary widely. Common metabolic diseases in animals include diabetes, hypoglycemia, hypocalcaemia, ketosis, and fatty liver disease. Preventative measures for metabolic diseases in animals include appropriate nutrition, exercise, and regular veterinary checkups. Proper diagnosis and treatment often require working closely with veterinary professionals who can provide customized recommendations based on the animal's specific circumstances.
- **Dietary indiscretion**: Dietary indiscretion refers to the consumption of inappropriate or "forbidden" foods by animals, which can lead to different gastrointestinal problems such as vomiting, diarrhea, and intestinal blockage. Eating garbage, toxic plants, chocolate, or bones are common examples of dietary indiscretion in pets. Preventative measures for dietary indiscretion include avoiding or limiting the animal's access to inappropriate foods, providing a balanced and nutritionally complete diet, and keeping garbage and other potentially harmful substances out of reach. If an animal exhibits symptoms of dietary indiscretion, it is important to seek veterinary care as soon as possible to avoid complications such as dehydration or intestinal obstruction. Your veterinarian may recommend hydration therapy, medication, or dietary adjustments to manage symptoms and promote recovery.
- **Chemical poisoning**: Chemical poisoning in animals can occur when they ingest, inhale, or come into contact with toxins or chemicals that are harmful to their health. These toxic substances can include common household cleaners, pesticides, plant toxins, and other chemicals. The symptoms of chemical poisoning in animals can vary widely depending on the type of chemical and the severity of exposure, but may include vomiting, diarrhea, seizures, difficulty breathing, weakness, and even death. Treatment for chemical poisoning may involve inducing vomiting, activated charcoal administration, or supportive care such as oxygen therapy or fluid therapy. It is essential to contact a veterinarian immediately if you suspect that your animal has been exposed to a toxic substance. Preventative measures for chemical poisoning include keeping all hazardous substances out of reach of animals, storing chemicals safely and securely, and following proper usage

instructions for any products that could be hazardous. Regular veterinary check-ups can also help detect early signs of toxicity and prevent more serious consequences.

- **Plant poisoning:** Plant poisoning in animals can occur when they ingest, come into contact with, or even inhale certain toxic plants. There are many potentially poisonous plants for animals, including common houseplants and outdoor vegetation. Phytotoxic plants include bracken fern, crotalaria, cyanide, gossypol, lantana camara, sweet clover.etc.) .Symptoms of plant poisoning in animals can include vomiting, diarrhea, and loss of appetite, weakness, respiratory distress, seizures, and even death. Treatment may involve inducing vomiting or administering activated charcoal to prevent further absorption of the toxins. In severe cases, hospitalization for supportive care may be necessary. Preventative measures for plant poisoning in animals include identifying and removing any toxic plants from the animal's environment. If you are unsure about a specific plant's toxicity, consult with a veterinarian or a qualified botanist who can help you identify the plant and evaluate its potential dangers. It is also important to ensure that your animal has access to safe and nutritious food and that you supervise them at all times when outdoors.
- **Physical trauma:** Physical trauma in animals can occur as a result of many different incidents, such as car accidents, animal fights, falls, or rough handling. It can result in a wide range of injuries, from mild cuts and bruises to more severe fractures, organ damage, or spinal cord injuries. Symptoms of physical trauma in animals may include limping, difficulty breathing, loss of consciousness, bleeding, or changes in behavior. Treatment for physical trauma will depend on the severity of the injury and may involve interventions such as wound care, pain management, or surgical procedures. It is essential to seek veterinary care immediately if you suspect that your animal has suffered any type of physical trauma. Preventative measures for physical trauma include keeping animals in safe and secure environments, providing appropriate supervision when outdoors or around other animals, and using appropriate protective equipment when engaging in activities such as riding or working with large animals.
- **Neoplastic disease:** Neoplastic disease in animals refers to the abnormal and uncontrolled growth of cells that leads to the formation of tumors. These tumors can be benign or malignant. Benign tumors do not spread to other parts of the body and are generally less concerning than malignant ones, which have the potential to spread to nearby tissues and organs. Neoplastic diseases can occur in any animal species, including dogs, cats, horses, and livestock, among others. The causes of neoplastic diseases are not entirely understood but can include genetic predispositions, exposure to environmental toxins, and chronic irritations or infections. Symptoms of neoplastic diseases in

animals can vary depending on the type and location of the tumor. Some common symptoms include changes in appetite or weight loss, lethargy, difficulty breathing, and abnormal growths or swellings on the body. Treatment for neoplastic disease in animals may involve surgical removal of the tumor, radiation therapy, chemotherapy, or a combination of these approaches. Prevention measures include regular check-ups with a veterinary professional, prompt treatment of any injuries or infections, and avoiding exposure to environmental toxins when possible.

1.4. Policies, legislations, regulations and directives in handling non-infectious disease

In Ethiopia, the government has established policies, legislations, regulations, and directives to handle non-infectious diseases in animals. These include:

- **Animal Health Directive** - This directive aims to prevent and control animal diseases, including non-infectious diseases. It provides guidelines for animal health management practices, disease diagnosis, treatment, and prevention.
- **Veterinary Drug Administration Directive** - This directive regulates the distribution and use of veterinary drugs, including those used to treat non-infectious diseases in animals.
- **Animal Welfare Proclamation -** This proclamation provides guidelines for the humane handling and treatment of animals, including those affected by non-infectious diseases.
- **Animal Feed Proclamation** - This proclamation regulates the manufacture and distribution of animal feed to ensure that animals receive proper nutrition and prevent the development of non-infectious diseases caused by malnutrition or dietary imbalances.
- **Meat Inspection Regulation -** This regulation is aimed at ensuring that meat sold in Ethiopia is safe for human consumption. It includes guidelines for the inspection and monitoring of livestock health, including non-infectious diseases.
- **Ethiopian Society of Animal Production (ESAP)** – The ESAP is an organization that promotes the sustainable use of animal resources in Ethiopia through research and education. They aim to improve animal health management practices and prevent non-infectious diseases through various training and education programs.

Overall, these policies, legislations, regulations, and directives aim to promote animal welfare, protect public health, and prevent the spread of animal diseases in Ethiopia, including non-infectious diseases. Veterinary professionals and animal owners are required to comply with these laws to ensure the proper management and treatment of animal health issues in the country.

1.5.Hazard identification and risk minimisation during case handling

Definition of Terminologies

- **Safety:** The protection from physical injury and mental well-being of the individual.
- **Health:** The protection of the body and mind of people from illness
- Welfare: The provision of facilities to maintain the health and well-being of the individual in the work place.
- **Hazard:** The way in which an object or a situation may cause harm.
- **Risk**: The chance that such effects will occur.
- **Exposure**: To the extent to which the likely recipient of the harm is exposed to or can be influence by the hazard
- **Occupational health and safety**: a state of complete physical, mental and social well-being of a worker.

✓ **Hazard identifying in the workplaces are**

- By carrying out a workplace risk assessment
- Determining how employees might be at risk
- Evaluating the risks
- Recording and review hazards at least annually, or earlier if something changes

Purposes of hazard identifications are:

To ensure there is a formal process for hazard identification and risk assessment which will effectively manage the hazard that may occur within the workplace.

✓ **Types of hazards which are encountered when handling non-infectious animal disease:**

Introduction: The most common definition of hazard: types it is something that has the potential to cause harm to: People, Property and, and the environment. Risk: refers to the chance or probability of that hazard causing harm to: People, Property and The environment.

- **Physical hazard**: any hazard that comes from environmental factors. Examples; vibration, loud noise, electrical frayed cord
- **Biological hazard**: a living or once living organism that have a potential to poses a threat to human health. Examples; blood or other body fluids, fungi, bacteria etc

- **Chemical hazard:** any hazard that comes from a solid, liquid, or gas elements, compound, or mixture that could cause health problem or pollution. Examples; cleaning products, pesticides, etc.
- **Ergonomic hazard:** physical conditions that may pose a risk of injury to the musculoskeletal system. Examples; awkward postures, static postures, high forces, repetitive motion, etc.
- **Psychological hazard**: aspects of the work environment and the way that work is organized that are associated with psychiatric, psychological and /or physical injury or illness. Example; stress, bullying, drug addiction, sexual harassment etc.

- **Procedures to remove or minimize hazards**
  - ✓ **Design or re-organize to eliminate hazards**

It is often cheaper and more practical to eliminate hazards at the design or planning stage of a product, process or place used for work. In these early phases, there is greater scope to design out hazards or incorporate risk control measures that are compatible with the original design and functional requirements. For example, remove trip hazards on the floor or dispose of unwanted chemicals.

- ✓ **Substitute the hazard with something safer**

If it is not reasonably practical to eliminate the hazards and associated risks, you should minimize the risk. For example, today the dangers associated with asbestos are well known and there are numerous alternatives to asbestos products currently on the market including cellulose fiber, thermoset plastic flour or polyurethane foams. Replacing solvent- based paints with water-based ones is also a better alternative.

- ✓ **Isolate the hazard from people**

This involves physically separating the source of harm from people by distance or using barriers. For example, introducing a strict work area, using guard rails/fence around exposed edges and holes in the floors, using remote control systems to operate machinery, enclosing a noisy process from a person and storing chemicals in a fume cabinet.

- ✓ **Use engineering controls**

An engineering control is a control measure that is physical in nature, including a mechanical device or process. For example this can be done through the use of machine guards, effective ventilation systems and setting work rates on a roster to reduce fatigue.

- ✓ **Use administrative controls**

Administrative controls are work methods or procedures that are designed to minimize exposure to a hazard. Establish appropriate procedures and safe work practices such as; limit exposure time to a hazardous task so that fewer employees are exposed, routine maintenance and housekeeping procedures, training on hazards and correct work methods and use signs to warn people of a hazard.

- ✓ **Using Required PPE and Safety Equipment**

As an employer, you are in charge of a safe and healthy working environment for your employees. You are familiar with the safety risks within your sector, as well as the measures you can take to counteract this risk.

Self-check-1	

Name…… ID…… Date…….

Directions: Answer all the questions listed below.

Part I. Write true if the statement is correct and write false if the statement incorrect false (2pts.)

1. Non-infectious diseases are caused by agents such as viruses, bacteria or parasites
2. Wearing appropriate personal protective equipment's can minimize work place hazard.

Part II. Choose the best answer (5pts each)

1. Which one of the following is a clinical impact of non-infectious animal diseases?
   A. Nutritional imbalance B. Toxicosis
   B. Reduced productivity C. All
2. One of the following is different from the other
   A. Chemical poisoning
   B. metabolic disease
   C. Physical trauma
   D. Zoonosis
   E. All

Part III. Give short answer.

1. Define non-infectious disease (3 pts.)
2. Discuss economic impact of non-infectious disease(5pts)
3. Describe categories of non-infectious animal disease (10 pts.)

Note: **Satisfactory rating – 32 points Unsatisfactory - below 32 points**

LG #9	L02. Identify and handle causes of animal poisoning

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following content coverage and topics:

- Aetiology, clinical sign, diagnosis, treatment and control of chemical toxins
- Aetiology, clinical sign, diagnosis, treatment and prevention of phyto-toxines

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, you will be able to:

- Identify and implement Aetiology, clinical sign, diagnosis, treatment and prevention/control methods of animal diseases caused by chemical toxins
- Identify and implement Aetiology, clinical sign, diagnosis, treatment and prevention/control methods of animal diseases caused by phyto-toxines (toxic plants).

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the information Sheets
4. Accomplish the Self-checks

Information Sheet 2

2.1.Aetiology, clinical sign, diagnosis, treatment and control of chemical toxins

Definition of terms

- **Poison:** refers to any substance that can cause harm or death when ingested, inhaled, or absorbed through the skin. This can include a wide range of substances, such as toxic plants, insecticides, rodenticides, and household chemicals.
- **Poisoning**: is injury or death due to swallowing, inhaling, touching or injecting various drugs, chemicals, venoms or gases.
- **Toxin**: is a poisonous substance produced by living cells or organisms that can cause harm or death when ingested, inhaled, or absorbed through the skin. Examples of toxins include botulinum toxin, which is one of the most poisonous biological substances
- **Toxicosis:** is a condition caused by exposure to a toxin or poisonous substance. It occurs when an animal ingests, inhales, or absorbs through the skin a toxic substance that can cause harm or death.
- **Toxicant:** is a chemical or substance that can cause harm or death to living organisms, including animals, plants, and humans. Toxicants may come from natural sources, such as certain plants or minerals, or may be man-made, such as chemicals used in agriculture or industry. Examples of toxicants include pesticides, heavy metals, and air pollutants.

2.1.1. Pesticide poisoning

Pesticide poisoning in animals transpires as a result of consuming or inhaling various forms of pesticides. Pesticides are either chemical or natural substances that are produced naturally or scientifically to kill pests. Pesticides are used to specifically repel or kill a group of undesired organisms.

These biocides are used to kill a variety of unwanted bugs, plants, fungi, rodents, larvae, and bacteria. Unfortunately, pesticides are prevalent; they are found in a variety of places such as in homes, garages, on farms, in sheds, in the yard, and on plants. Pesticides should not be used outdoors when an animal is present; as this is one way poisoning can occur.

Etiology

Accidental ingestion of contaminated feed or water, exposure to pesticides during application or storage, and inadequate protective measures for workers or animals in the vicinity of pesticide use can cause pesticide poisoning in animals.

Clinical sign

Pesticide poisoning in animals can cause a wide range of clinical signs, which can vary depending on the type and dose of pesticide involved. The following are some of the most common clinical signs associated with pesticide poisoning in animals:

Pesticide Poisoning Can Cause

- Vomiting
- Diarrhea
- Anorexia
- Tremors
- Skin Irritation
- Eye irritation
- Difficulty breathing
- Even Blindness
- Ataxia
- Muscle Weakness
- Seizures
- Coma, or
- Death due to Respiratory failure

Diagnosis

Diagnosing pesticide poisoning in animals can be challenging because the symptoms can mimic other conditions. A veterinarian will typically perform a physical examination, along with obtaining a detailed history including the type and timing of exposure, and may run diagnostic tests to confirm the presence of toxins.

Treatment

Some of the treatment options may include:

- **Dermal decontamination**: In some cases, decontamination may be needed to remove the pesticide from the animal's fur or skin. This may involve giving the animal a bath or washing their skin with water.
- **Gastrointestinal** decontamination: Administer activated charcoal and cathartic agent to prevent further absorption from the intestine.
- **Supportive care:** Providing supportive care such as intravenous fluids, medications to control vomiting and seizures, and oxygen therapy can help manage the clinical signs associated with pesticide poisoning.
- **Specific antidotes:** Atropine to control muscarinic parasympathetic signs (salivation and others)

Prevention and control

Prevention and control of pesticide poisoning in animals involve several measures. The first step is to minimize exposure to pesticides through safe handling and storage practices. This includes wearing appropriate protective clothing and equipment, such as gloves, goggles, and masks, when working with pesticides, as well as storing pesticides in secure areas away from animals. Additionally, using safer alternatives to pesticides, such as biological controls or integrated pest management techniques, can reduce exposure and risks.

Another important aspect is proper pesticide application. This includes following label instructions for dosages, timing of application, and safe use. It is also critical to avoid applying pesticides near water sources or in areas where animals may graze.

Overall, prevention and control of pesticide poisoning require careful attention to safe handling practices, proper application, and early intervention if poisoning is suspected.

2.1.2. Acaricide poisoning

Acaricides are pesticides used to kill ticks and mites.

Acaricides include chlorinated hydrocarbons (e.g., dichlorodiphenyltrichloroethane; DDT),

Organophosphorous compounds (e.g., Diazinon), carbamates (e.g., carbaryl), pyrethroids (e.g., permethrin, flumethrin), formamidines, and avermectins.

Etiology

Poisoning by organic insecticides and acaricides may be caused by direct application, by ingestion of contaminated feed or forage treated for control of plant parasites, or by accidental exposure.

Clinical sign

Some common symptoms include vomiting, diarrhea, difficulty breathing, tremors, seizures, and coma. If you suspect that your animal has been poisoned by an insecticide or acaricide, contact your veterinarian immediately.

Diagnosis

The diagnosis of acaricide poisoning is based on clinical signs and history of exposure.

Treatment

The treatment of acaricide poisoning may include

- **Decontamination**: decontamination may involve washing the animal with soap and water or activated charcoal
- **Supportive care**: may include intravenous fluids, oxygen therapy, and medications to control seizures or other symptoms.
- **Antidotes if available:** Antidotes are available for some types of acaricides and may be administered if appropriate.

Prevention and control

To prevent acaricide poisoning in animals, it is important to follow the instructions on the label of the product being used. The label will provide information on how to use the product safely and effectively. It is also important to store the product in a safe place that is out of reach of animals and children. When using acaricides, it is recommended to wear protective clothing such as gloves and a mask.

2.1.3. Herbicide poisoning

An herbicide is a substance used to kill unwanted plants.

Etiology

Animals seldom ingest sufficient sprayed plant material to produce clinical illness or Herbicide poisoning occurs when a person or animal is exposed to an excessive amount of herbicide.

Clinical Signs

Common symptoms include, Nausea, vomiting, Diarrhea, loss of appetite, lethargy, weakness, seizures, and difficulty breathing. In severe cases, herbicide poisoning can lead to coma and death.

Diagnosis

History of exposure to a concentrated source of herbicides

Clinical signs

Exclusion of other possible causes

Treatment

- Decontamination
- Activated charcoal
- Ocular decontamination
- Dermal decontamination
- Supportive and symptomatic
- Fluid therapy
- Antioxidant therapy

Control

Feed the animal high-quality diet.

Activated carbon exhibits a great adsorptive capacity and acts as a physical barrier to herbicide uptake.

2.1.4. Rodenticides poisoning

Many poisons have been used against rodent pests. Farm animals, pets, and wildlife often gain access to these poisons via the baits or the poisoned rodents or by malicious intent. This discussion covers the most commonly used rodenticides.

Etiology

Ingestion of anticoagulant containing rodenticides. When an animal ingests a rodenticide, it interferes with their blood clotting ability, leading to hemorrhage and other bleeding issues. Pets and wildlife may be poisoned directly from baits or indirectly by consumption of poisoned rodents.

Clinical signs

Clinical signs of rodenticide poisoning in animals can includes pale mucosa, weakness, anorexia, dyspnea, tachycardia, hematuria, epistaxis, melena, hematoma formation, recumbency, and death.

Diagnosis

Anticoagulant rodenticide toxicosis is usually diagnosed based on history of ingestion of the substance.

Anticoagulants or their metabolites can be detected in blood or urine in live animals and in the liver of animals that have died.

Treatment

Vitamin K1 (phytonadione); blood transfusion in critical cases.

Control

Prevent access to rodenticides.

Use safer alternative strategies.

2.1.5. Industrial chemical poisoning

- **Lead Poisoning (Plumbism)**

Eitology

In veterinary medicine, lead poisoning is most common in dogs and cattle. Lead poisoning in other species is limited by reduced accessibility, more selective eating habits, or lower susceptibility. In cattle, many cases are associated with seeding and harvesting activities when used oil and battery disposal from machinery is handled improperly. Other sources of lead include paint, linoleum, grease, lead weights, lead shot, and contaminated foliage growing near smelters or along roadsides. Lead poisoning is also encountered in urban environments, and renovation of old houses that have been painted with lead-based paint has been associated with lead poisoning in small animals and children.

Clinical sign

Acute lead poisoning is more common in young animals. The prominent clinical signs are associated with the GI and nervous systems. In cattle, signs that appear within 24-48 hr of exposure include ataxia, blindness, and salivation, spastic twitching of eyelids, jaw champing, bruxism, muscle tremors, and convulsions.

Sub-acute lead poisoning, usually seen in sheep or older cattle, is characterized by anorexia, rumen stasis, colic, dullness, and transient constipation, frequently followed by diarrhea, blindness, head pressing, bruxism, hyperesthesia, and incoordination.

Chronic lead poisoning, which is occasionally seen in cattle, may produce a syndrome that has many features in common with acute or sub-acute lead poisoning. GI abnormalities, including anorexia, colic, emesis, and diarrhea or constipation, may be seen in dogs. Anxiety, hysterical barking, and jaw champing, salivation, blindness, ataxia, muscle spasms, opisthotonos and convulsions may develop. CNS depression rather than CNS excitation may be evident in some dogs. In horses, lead poisoning usually produces a chronic syndrome characterized by weight loss, depression, weakness, colic, diarrhea, laryngeal or pharyngeal paralysis (roaring), and

dysphagia that frequently results in aspiration pneumonia. In avian species, anorexia, ataxia, loss of condition, wing and leg weakness, and anemia are the most notable signs.

Diagnosis

Lead levels in various tissues may be useful to evaluate excessive accumulation and to reflect the level or duration of exposure, severity, and prognosis and the success of treatment. Concentrations of lead in the blood at 0.35 ppm, liver at 10 ppm, or kidney cortex at 10 ppm are consistent with a diagnosis of lead poisoning in most species.

Radiologic examination may be useful to determine the magnitude of lead exposure. Lead poisoning may be confused with other diseases that cause nervous or GI abnormalities. In cattle, such diseases may include polioencephalomalacia, nervous coccidiosis, tetanus, hypovitaminosis A, hypomagnesemic tetany, nervous acetonemia, arsenic or mercury poisoning, brain abscess or neoplasia, rabies, listeriosis, and Haemophilus infections. In dogs, rabies, distemper, and hepatitis may appear similar to lead poisoning.

Treatment

If tissue damage is extensive, particularly to the nervous system, treatment may not be successful. The following treatments are used singly or in combination:

- ✓ Immediate relief of acute nervous signs in calves by IV pentobarbital sodium
- ✓ Emptying of rumen and reticulum completely, supplemented by wash-out
- ✓ Calcium versenate 150mg/kg BW
- ✓ Thiamine (2-4 mg/kg/day SC) alleviates clinical manifestations and reduces tissue deposition of lead.

Control

Remove herbivores from contaminated pastures if possible.

- **Common salt poisoning (sodium chloride poisoning)**

An excess of salt intake can lead to the condition known by various names including salt poisoning, hypernatremia, sodium ion toxicosis and water deprivation-sodium ion intoxication. The last name in this list is the most descriptive, giving both the result (sodium ion intoxication) and the most common predisposing condition (water deprivation). Lactating females are most susceptible.

Etiology

Is directly related to water consumption.

- ✓ Thirsty cattle get access to saline bore water
- ✓ Animals on low salt diet allowed access to ad lib salt
- ✓ Animals on normal salt intake (2% of ration) temporarily deprived of water
- ✓ Prepared feeds to housed cattle contain excess salt

Clinical sign

In cattle poisoning may develop gastroenteritis, weakness, dehydration, tremors, ataxia, blindness, seizure-like activity or partial paralysis including knuckling over at the fetlocks, lateral recumbency with paddling and opisthotonus. Cattle can die within 24 h following the appearance of severe clinical signs. In in swine and include loss of appetite, thirst, restlessness, pruritus, constipation head pressing, circling or pivoting around a limb. The animal may display seizure-like activity and assume a dog-sitting position, draw its head back in a jerking motion and fall over on its side. Terminally, the animal will be in lateral recumbency with paddling and opisthotonus.

Diagnosis

Based on history, clinical sing and excess sodium concentrations in serum,
Analysis of food, water, or suspect material for sodium content

Treatment:

- The toxic feed or water must be removed immediately In advanced cases animals may be unable to drink and water may have to be administered by stomach tube.
- Supportive care: In some cases, supportive care may be necessary to stabilize the animal's condition. This may include intravenous fluids, electrolyte replacement therapy, and the administration of oxygen.

Prevention and control

Preventing salt poisoning in animals involves keeping all salt cellars, salt containers and rock salt out of your pet's reach

2.1.6. Arsenic poisoning

Arsenic poisoning in animals is caused by several different types of inorganic and organic arsenical compounds. Toxicity varies with factors such as oxidation state of the arsenic, solubility, species of animal involved, and duration of exposure.

Etiology

Inorganic arsenic is often incorporated into pesticides, which are the most common sources of arsenic poisoning in cattle. Inorganic arsenicals are also used as herbicides and cattle sometimes are exposed by eating recently sprayed forage. Another common source is when cattle lick ashes of burned fence posts.

Clinical sign

Poisoning is usually acute with major effects on the GI tract and cardiovascular system. Arsenic has a direct effect on the capillaries, causing damage to micro vascular integrity, transudation of plasma, loss of blood, and hypovolemic shock. Profuse watery diarrhea, sometimes tinged with blood, is characteristic, as are severe colic, dehydration, weakness, depression, weak pulse, and cardiovascular collapse. The onset is rapid, and signs are usually seen within a few hours (or up

to 24 hr.). The course may run from hours to several weeks depending on the quantity ingested. In per acute poisoning, animals may simply be found dead.

Diagnosis

Chemical determination of arsenic in tissues (liver or kidney) or stomach contents provides confirmation. Liver and kidneys of normal animals rarely contain >1 ppm arsenic (wet wt.); toxicity is associated with a concentration >3 ppm. The determination of arsenic in stomach contents is of value usually within the first 24-48 hr. after ingestion. The concentration of arsenic in urine can be high for several days after ingestion. Drinking water containing >0.25% arsenic is considered potentially toxic, especially for large animals.

Treatment

In animals with recent exposure and no clinical signs, emesis should be induced (in capable species), followed by activated charcoal with a cathartic (efficacy of charcoal in arsenic toxicosis remains to be determined) and then oral administration of GI protectants (small animals, 1-2 hr after charcoal) such as kaolin-pectin, and fluid therapy as needed. In animals have clinical sign, aggressive fluid therapy, and blood transfusion (if needed).

Sodium thiosulfate has also been used, PO, at 20-30 g in 300 mL of water in horses and cattle, one-fourth this dose in sheep and goats, and 0.5-3 g in small animals or as a 20% solution, IV, at 30-40 mg/kg, 2-3 times/day for 3-4 days or until recovery.

Prevention and control

Preventing and controlling arsenic poisoning in animals involves several strategies, including:

- Regular monitoring: Regular testing of feed, water sources, and soil for arsenic levels can help to identify potential sources of contamination before they cause harm to animals.
- Proper disposal of waste: Proper disposal of waste such as batteries, pesticides, and industrial chemicals can help prevent contamination of the environment, which can ultimately lead to arsenic buildup in animals.
- Avoiding contaminated sources: Arsenic can accumulate in plants such as rice and apple trees that are grown in contaminated soil, so avoiding these sources or limiting their consumption is important.

- Mineral supplementation: Providing animals with minerals such as calcium and zinc can help limit the absorption of arsenic in their bodies.
- Early detection and treatment: Early detection and treatment of arsenic poisoning is important for a successful outcome.

2.2. Aetiology, clinical sign, diagnosis, treatment and prevention of phyto-toxines

Introduction

Phytotoxins are toxins produced by plants that can have harmful effects on animals. These toxins are naturally occurring compounds that are synthesized by plants as a defense mechanism against herbivores and other potential predators. When ingested, phytotoxins can cause a range of harmful effects in animals, including neurological symptoms, gastrointestinal distress, and even death. There are many types of phytotoxins that can affect animals, and they can have different mechanisms of action depending on the specific compound. Some phytotoxins act as neurotoxins, affecting the function of the nervous system and causing symptoms such as muscle weakness, paralysis, and convulsions. Others can cause digestive problems such as vomiting and diarrhea, or damage internal organs like the liver or kidneys.

Animals can be exposed to phytotoxins in a variety of ways, depending on the specific toxin and the animal species involved. Grazing animals may consume plants containing phytotoxins as a significant portion of their diet, while predators may be exposed through consumption of prey that has ingested toxic plants. In some cases, animals may come into contact with phytotoxins through skin contact or inhalation. Due to the potential risks posed by phytotoxin exposure, it is important for animal owners and veterinary professionals to be aware of the types of plants that may contain toxic compounds and take appropriate measures to minimize the risk of exposure. This may include restricting access to potentially toxic plants, monitoring animals for signs of toxicity, and seeking prompt veterinary care if toxicity is suspected.

2.2.1. Oxalate Poisoning

Oxalate poisoning is a type of phytotoxin poisoning that can occur when animals ingest plants containing high levels of oxalic acid. Oxalic acid is a naturally occurring compound found in many plants, including some that are commonly consumed by domestic animals such as horses, cattle, and goats. When ingested, oxalic acid can bind with calcium in the body to form crystals that can accumulate in the kidneys, leading to kidney damage and potentially fatal kidney failure. Oxalate ingestion produces several syndromes depending on the type of oxalate found in the plant. Oxalates can be found as water soluble oxalates, or can combine with calcium to form water insoluble calcium oxalate.

Eitology

Intoxication is more common when hungry, naive animals rapidly consume oxalate-containing plants. Plant species: many different species contain varying amounts of oxalate. Some of the plants commonly known to have high levels of oxalate include:

- Pigweed (Portulaca)
- Setaria (Setaria sphacelata)



Figure.2.1 Pigweed (Portulaca)



Figure 2.2 Setaria (Setaria sphacelata

Relative oxalate concentration: leaves > seeds > stems

Species

Sheep more commonly affected

Cattle are susceptible

Clinical sign

Clinical sign is Similar to milk fever in cattle include; tremors, swelling, depression, tetany, coma, convulsions , colic, dehydration, restlessness, anorexia, depression, weight loss, difficulty breathing due to swelling of the throat and lungs, and seizures and polyuria or anuria.



Fiure2.3 Big head in horse due to oxalate poisoning



Figure 2.4 oxalate poisoning in cattle

https://www.google.com/url?sa=i&url=https%3A%2F%2Fwww.youtube.com%2Fwatch%3Fv%3DYgObLSr7V18&psig=AOvVaw3sGRLFdtQPvs0R_9_xi0t&ust=1684871663795000&source=images&cd=vfe&ved=0CBAQjhxqFwoTCJjFsa_aif8CFQAAAAAdAAAAABAD

Diagnosis

Diagnosis of oxalate poisoning is based on clinical sign, history of exposure to oxalate containing plant.

Treatment

Intravenous administration of calcium borogluconate to correct hypocalcemia.

Supportive nursing care

Prevention

Limit access to pastures containing the plants.

Do not introduce hungry animals to a new pasture.

Use herbicides on dense stands of oxalate-containing plants.

2.2.2. Cyanide poisoning

Cyanide poisoning in animals is a condition that occurs when animals consume toxic forage containing cyanide. These include Triglochin maritima (arrow grass), Hoecus lunatus (velve grass), Sorghum spp (Johnson grass, Sudan grass, common sorghum), Prunus spp (apricot, peach, chokecherry, pincherry, wild black cherry),

In Sorghum spp forage grasses, leaves usually produce 2-25 times more HCN than do stems; seeds contain none. New shoots from young, rapidly growing plants often contain high concentrations of prussic acid glycosides. Ruminants are more susceptible than monogastric animals, and cattle slightly more so than sheep.



Figure 2.5.Sorghum plant

Aetiology

In livestock, the most frequent cause is ingestion of plants that contain cyanogenic glycosides.

Clinical sign

Signs can occur within 15-20 min to a few hours after animals consume toxic forage. Excitement can be displayed initially, accompanied by rapid respiration rate. Dyspnea follows shortly, with tachycardia. Salivation, excess lacrimation, and voiding of urine and feces may occur. Vomiting may occur, especially in pigs. Muscle fasciculation is common and progresses to generalized spasms before death. Animals stagger and struggle before collapse.

Mucous membranes are bright red but may become cyanotic terminally. Death occurs during severe asphyxial convulsions. The heart may continue to beat for several minutes after struggling and breathing stops. The whole syndrome usually does not exceed 30-45 min. Most animals that live ≥2 hr after onset



Figure2.6. Cyanide poisoning in cattle



Figure2.7. post mortem findings due to cyanide poisoning

Diagnosis

Appropriate history, clinical signs, postmortem findings, and demonstration of HCN in rumen (stomach) contents or other diagnostic specimens support a diagnosis of cyanide poisoning.

Specimens recommended for cyanide analyses include the suspected source (plant or otherwise), rumen or stomach contents, heparinized whole blood, liver, and muscle. Ante mortem whole blood is preferred; other specimens should be collected as soon as possible after death, preferably within 4 hr. Specimens should be sealed in an airtight container, refrigerated or frozen, and submitted to the laboratory without delay. When cold storage is unavailable, immersion of specimens in 1-3% mercuric chloride has been satisfactory.

Hay, green chop, silage, or growing plants containing >220 ppm cyanide as HCN on a wet-weight (as is) basis are very dangerous as animal feed.

Treatment

Immediate treatment is necessary. Sodium nitrite (10 g/100mL of distilled water or isotonic saline) should be given IV at 20 mg/kg body wt., followed by sodium thiosulfate (20%), IV, at ≥500 mg/kg; the latter may be repeated as needed with little hazard. Sodium nitrite therapy may be carefully repeated at 10 mg/kg, every 2-4 hr. or as needed. Many clinical signs of nitrate and prussic acid poisoning are similar, and injecting sodium nitrite induces methemoglobinemia identical to that produced by nitrate poisoning. If in doubt of the diagnosis, methylene blue, IV, at 4-22 mg/kg, may be used to induce methemoglobin. Because methylene blue can serve as both a donor and acceptor of electrons, it can reduce methemoglobin in the presence of excess methemoglobin or induce methemoglobin when only hemoglobin is present (but sodium nitrate is the more effective treatment for cyanide poisoning if the diagnosis is certain).

Prevention and control

Pasture grasses (e.g., Sudan grass and sorghum-Sudan grass hybrids) should not be grazed until they are 15-18 in. tall to reduce danger from prussic acid poisoning. Forage sorghums should be several feet tall. Animals should be fed before first turning out to pasture; hungry animals may consume forage too rapidly to detoxify HCN released in the rumen. Animals should be turned

out to new pasture later in the day; prussic acid release potential is reported to be highest during early morning hours. Free-choice salt and mineral with added sulfur may help protect against prussic acid toxicity. Grazing should be monitored closely during periods of environmental stress, e.g., drought or frost. Abundant regrowth of sorghum can be dangerous; these shoots should be frozen and wilted before grazing.

Green chop forces livestock to eat both stems and leaves, thereby reducing problems caused by selective grazing. Cutting height can be raised to minimize inclusion of regrowth. Hay has been dried at oven temperatures for up to 4 days with no significant loss of cyanide potential.

2.2.3. Nitrate Poisoning

What is nitrate poisoning?

Nitrate in itself is not toxic to animals, but at elevated levels it causes a disease called nitrate poisoning. Nitrates are normally found in forages and are converted by the digestion process to nitrite, and in turn the nitrite is converted to ammonia. The ammonia is then converted to protein by bacteria in the rumen. If cattle rapidly ingest large quantities of plants that contain high levels of nitrate, nitrite will accumulate in the rumen. Nitrite is ten times as toxic to cattle as nitrate. Nitrite is absorbed into red blood cells and combines with hemoglobin (oxygen-carrying molecule) to form methemoglobin. Methemoglobin cannot transport oxygen as efficiently as hemoglobin, so the animal's heart rate and respiration increases, the blood and tissues of the animal take on a blue to chocolate brown tinge, muscle tremors can develop, staggering occurs, and the animal eventually suffocates. Nitrate poisoning can occur commonly in animals.

Aetiology

Poisoning is usually associated with animals ingesting forage or feed with a high nitrate content. Sheep and cattle are more susceptible to poisoning than non-ruminant species, because microbes in their digestive tracts favor the conversion of nitrate to nitrite.

What plant factors favour nitrate poisoning?

The majority of nitrate poisoning cases occur with drought-stressed oats, corn and barley. However, a number of other plants can also accumulate nitrate, including Sudan grass, sorghum-sudan hybrids, and pearl millet. Table 2.1 lists common plants known to accumulate nitrate if conditions are favorable. Plants that have been fertilized have higher nitrate levels than non-fertilized plants. The abnormal accumulation of nitrate in plants is influenced by various factors such as moisture conditions, soil conditions and type of plant. Plant stresses such as drought are associated with increased levels of nitrate in plants. Soils high in nitrogen readily supply nitrate to plants. Acidity, sulfur or phosphorus deficiencies, low molybdenum, and low temperatures are known to increase nitrate uptake by plants. Plant parts closest to the ground (stalks) contain the highest concentrations of nitrates. Leaves contain less than stalks or stems, and the seed (grain) and flower usually contain little or no nitrate. Most of the plant nitrate is in the bottom third of the stalk. Research from Oklahoma has shown that the lower 6 inches of the stem in pearl millet contains three times more nitrate than the top part of the plant. While difficult to do with drought-stressed forages, raising the cutter bar above 6 inches can reduce nitrate content of forages. Nitrate decreases as plants mature. Young plants have higher nitrate concentrations than mature plants. However, mature plants can still have excessive nitrate concentrations if environmental and soil conditions are favorable.

Table 2.1 Common plants known to accumulate nitrate

Crop	Weeds
Barley	Canada thistle
Sweet clover	Wild sunflower
Flax	Jimsonweed
Oats	Kochia
Rape	Lambs quarter
Rye	Nightshade
Soybean	Pigweed
Sudan grass	Russian thistle



Figure 2.8 Sweet clover



Figure2.9 Nightshade

Clinical sings

Signs of nitrite poisoning usually appear suddenly due to tissue hypoxia and low blood pressure as a consequence of vasodilation. Rapid, weak heartbeat with subnormal body temperature, muscular tremors, weakness, and ataxia are early signs of toxicosis when methemoglobinemia reaches 30-40%. Brown, cyanotic mucous membranes develop rapidly as methemoglobinemia exceeds 50%. Dyspnea, tachypnea, anxiety, and frequent urination are common. Blood that contains methemoglobin usually has a chocolate-brown color, although dark red hues may also be seen. There may be pinpoint or larger hemorrhages on serosal surfaces. Dark brown discoloration evident in moribund or recently dead animals is not pathognomonic, however, and other methemoglobin inducers must be considered. If necropsy is postponed too long, the brown discoloration may disappear with conversion of methemoglobin

Some monogastric animals, usually because of excess nitrate exposure from nonplant sources, exhibit salivation, vomiting, diarrhea, abdominal pain, and gastric hemorrhage. Affected animals may die suddenly without appearing ill, in terminal anoxic convulsions within 1 hrs. Or after a clinical course 'of 12-24 hr or longer. Acute lethal toxicoses almost always are due to development of 380% methemoglobinemia. Under certain conditions, adverse effects may not be apparent until animals have been eating nitrate-containing forages for days to weeks. 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 be seen in some cattle 5-14 days after excessive nitrate/nitrite exposure, but likely only in cows that have survived a 350% methemo-globinemia for 6-12 hrs. or longer. Prolonged exposure to excess nitrate coupled with cold stress and inadequate nutrition may lead to the alert downer cow syndrome in pregnant beef cattle; sudden collapse and death can result.



Figure2.10 nitrite poisoning in cattle



Figure2.11. cyanotic mucus membrane due to nitrate poisoning in cattle

Diagnosis

Diagnosis of nitrate intoxication is based on observed clinical signs and the possibility of exposure to toxic plants or water. Laboratory analysis can be performed on suspected plants, water, stomach contents, blood, urine, and aqueous humor of the eye of dead cattle to confirm the diagnosis.

Postmortem specimens of rumen contents are of little value for nitrate determination because most nitrate in the rumen is reduced by anaerobic fermentation to ammonia. Samples from fresh grass or dry forages need to be representative of the field or bales in question. Package these samples in a clean plastic bag and ship them to the laboratory for analysis.

Collect water samples in a sterile bottle. When collecting from a water system, let the water flow for a couple of minutes before collecting the sample.

Treatment

Slow IV injection of 1% methylene blue in distilled water or isotonic saline should be given at 4-22 mg/kg body wt., or more, depending on severity of exposure. Lower dosages may be repeated in 20-30 min if the initial response is not satisfactory. Lower dosages of methylene blue can be used in all species, but only ruminants can safely tolerate higher dosages. If additional exposure or absorption occurs during therapy, retreating with methylene blue every 6-8 hr should be considered.

Note that methylene blue is not approved by the FDA for use in food-producing animals. Consult your veterinarian before using this treatment. Rumen lavage with cold water and antibiotics may stop the continuing microbial production of nitrite.

Control

Animals may adapt to higher nitrate content in feeds, especially when grazing summer annuals such as sorghum-Sudan hybrids. Multiple, small feedings help animals adapt. Trace mineral supplements and a balanced diet may help prevent nutritional or metabolic disorders associated with long term excess dietary nitrate consumption. Feeding grain with high-nitrate forages may reduce nitrite production.

Heating may assist bacterial conversion of nitrate to nitrite; feeding high nitrate hay, straw, or fodder that has been damp or wet for several days or stockpiled, green-chopped forage should be avoided. Contact your veterinarian or extension personnel if you need assistance in determining the correct ratios of high and low nitrate forages to blend to develop a ration for a particular class of livestock.

2.2.4. Gossypol Poisoning

Gossypol is produced naturally by the glands of the cotton plant. Although all animals are susceptible to gossypol toxicity, monogastrics, preruminants, immature ruminants, and poultry are affected most frequently. Gossypol toxicosis—usually chronic, cumulative, and sometimes insidious—follows consumption of cottonseed or cottonseed products containing excess free gossypol. It is of most concern in domestic production animals, especially preruminants or immature ruminants and pigs; mature ruminants are more resistant to gossypol's toxic effects. However, gossypol toxicity can affect high-producing dairy cows with high feed intake, dairy goats, and other mature ruminants fed excess gossypol for long periods. It has also been reported in dogs fed diets containing cottonseed meal or housed on cottonseed bedding.

Etiology

Gossypol, the predominant pigment and probably the major toxic ingredient in the cotton plant (*Gossypium* spp), and other polyphenolic pigments are contained within small, discrete structures called pigment glands found in various parts of the cotton plant. Gossypol is found in cottonseed as both protein-bound and free forms; only the free form is toxic.

All animals are susceptible to gossypol toxicity; however, monogastrics, preruminants, immature ruminants, and poultry appear to be affected most frequently. Toxic effects usually occur only after long-term exposure to gossypol, often weeks to months. Adult ruminants are able to detoxify gossypol by formation of stable complexes with soluble proteins in the rumen, thus preventing absorption, something lacking in swine, preruminants, and young ruminants with only a partially functioning rumen. Pigs, guinea pigs, and rabbits are reported to be sensitive to

gossypol; dogs and cats appear to have intermediate sensitivity. Goats may be more sensitive to gossypol than are cattle. Horses appear relatively resistant to gossypol, but caution is still advised.



Figure 2.12 A young cotton plant (Gossypium spp). And flower

Clinical Findings

Clinical signs of gossypol toxicosis may relate to effects on the cardiac, hepatic, renal, reproductive, or other systems. Prolonged exposure can cause acute heart failure resulting from cardiac necrosis. Also, a form of cardiac conduction failure similar to hyperkalemic heart failure can result in sudden death. Pulmonary effects, labored breathing, and chronic dyspnea are most likely secondary to cardiotoxicosis from congestive heart failure.

Hepatotoxicosis can be a primary effect from direct damage to hepatocytes or metabolism of phenolic compounds to reactive intermediates, or liver necrosis may be secondary to congestive heart failure.

Hematologic effects include anemia with reduced numbers of RBCs and increased RBC fragility, decreased oxygen release from oxyhemoglobin, and reduced oxygen-carrying capacity of blood with lowered Hgb and PCV values due to complexing of iron by gossypol.

Reproductive effects include reduced libido with decreased spermatogenesis and sperm motility, as well as sperm abnormalities (which are reversible) resulting from enzyme inhibition of steroid synthesis in testicular Leydig cells. Specific mitochondrial damage in spermatozoa appears to cause immobility and depressed sperm counts. Extensive damage to germinal epithelium in both rams and bulls fed excessive gossypol may be responsible for depressed spermatogenesis.

Effects in females may include irregular cycling, luteolytic disruption of pregnancy, and direct embryotoxicosis; probable mechanisms include an endocrine effect on the ovary as well as a direct cytotoxic effect on the uterus or embryo. Green discoloration of egg yolks and decreased egg hatchability have been reported in poultry. However, antifertility and reproductive effects in many nonruminant species are secondary to the more toxic effects, particularly in females.
Clinical signs of prolonged excess exposure to gossypol in many animals are reduced growth rate, 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. Adult dairy cattle may show weakness, depression, anorexia, edema of the brisket, and dyspnea, and also have gastroenteritis, hemoglobinuria, and reproductive problems. In monogastric animals, acute exposure may result in sudden circulatory failure, whereas subacute exposure may result in pulmonary edema secondary to congestive heart failure; anemia may be another common sequela. Violent dyspnea ("thumping") is the predominant clinical sign in pigs. In dogs, gossypol has primarily cardiotoxic effects; deterioration is progressive, and ascites may be marked. Affected dogs may show polydipsia and have serum electrolyte imbalances, most notably hyperkalemia; with pronounced ECG abnormalities.

Diagnosis

- A history of dietary exposure to cottonseed meal or cottonseed products over a relatively long period
- Clinical signs, especially sudden death or chronic dyspnea, affecting multiple animals within a group
- No response to antimicrobial treatment
- Lesions consistent with gossypol toxicosis at postmortem (e.g., cardiomyopathy and hepatopathy, with increased fluids in various body cavities)
- The presence of notably high concentrations of free gossypol in the diet

Treatment

There is no effective treatment for gossypol toxicosis. Adsorbents such as activated charcoal and saline cathartics are of little value because of the chronic exposure and cumulative nature of gossypol. If gossypol toxicosis is suspected, all cottonseed products should be removed from the diet immediately. However, severely affected animals may still die up to 4 weeks after withdrawal of gossypol from the diet

A high-quality diet supplemented with lysine, methionine, and fat-soluble vitamins should be included in supportive therapy. Selenium or copper deficiencies may potentiate gossypol toxicosis.

Prevention and Control

- Removal of suspected feed
- Provision of feed without cottonseed or cottonseed meal
- Avoiding transport of affected animals, or transporting them only very slowly
- A high intake of protein, calcium hydroxide, or iron salts appears to be protective in cattle.

Mature cattle should also be given ≥40% of dry-matter intake from a forage source, and dietary gossypol concentrations should be limited to ≤1,000 ppm, because 1,500 ppm may cause anemia, poor growth, or decreased milk production.

Swine and young ruminants are affected by dietary gossypol concentrations >100 ppm.

Added iron of up to 400 ppm in swine diets and up to 600 ppm in poultry diets was reported to be effective in preventing clinical signs and tissue residues of dietary gossypol exposure when used in ratios of 1:1 to 4:1 of iron to free gossypol. Poultry are affected by dietary gossypol concentrations >200 ppm.

2.2.5. Sweet clover poisoning

Etiology

Sweet clover poisoning is seen when animals eat spoiled sweet clover hay or silage. Any method of hay storage that allows sweet clover to mold increases the chance of a toxin forming in the hay. Weathered, large round bales, particularly the outer portions, usually contain the highest

levels of toxin. When toxic hay or silage is eaten, blood clotting may be delayed and spontaneous bleeding may. Instances of poisoning have involved mainly cattle and some horses.

Clinical sign

Signs are caused by faulty clotting of blood and loss of blood from ruptured blood vessels. The amount of time between eating toxic sweet clover and appearance of signs varies greatly. If the sweet clover has a low level of toxin, animals may eat it for months before disease appears.

The first signs may be stiffness and lameness, caused by bleeding into the muscles and joints. The animal may bleed from the nose or into the gastrointestinal tract. Death from massive bleeding after injury, surgery, or giving birth can occur suddenly without other signs.

Diagnosis

Diagnosis is based on a history of eating sweet clover hay or silage over relatively long periods, signs, and a prolonged blood clotting time. Sweet clover poisoning is usually a problem in herd animals. It is unlikely if bleeding or slow blood clotting are seen in a single animal from a group.

Treatment

Blood transfusions are usually needed and can be repeated if necessary. Severely affected animals are also treated with vitamin K1, which reverses the effects of the toxin. Several hours are needed for significant improvement, and more than 24 hours is needed for clotting to return to normal.

Prevention

The only certain method of prevention is to avoid feeding sweet clover hay or silage. However, variations of sweet clover, low in the toxin and safe to feed (for example, Polara), have been developed.

A simple management technique is to alternate feeding (every 7 to 10 days) sweet clover hay suspected of containing the toxin with other roughage such as alfalfa or a grass-legume hay mixture. Pregnant animals should not receive sweet clover hay for at least 2 to 3 weeks, and

preferably 4 weeks, before giving birth. Similarly, animals should not receive sweet clover hay for 3 to 4 weeks before castration or other surgery.

2.2.6. Plant causing photosensitization

Photosensitization is the disease caused by the sensitization of the superficial layers of lightly pigmented skin and mucus to light of certain wavelength. Dermatitis develops when the sensitized skin is exposed to the strong light.

Aetiology

- **Primary photosensitization:** is due to ingestion of exogenous photodynamic agents. Usually occurs when the plant is in lush green stage.

Important plants causing primary photosensitization in animals

- ✓ St. John's wort
- ✓ Spring parsley
- ✓ Bishop's weed
- ✓ Dutchman's britches
- ✓ Rain lily
- ✓ Alfalfa



Figure 2.13 St. John's wort plant



Figure2.14. Spring parsley



Figure 2.15 Dutchman's britches



Figure 2.16 Rain lily



Figure 2.17 alfalfa (medicago sativa)

- **Secondary photosensitization:** is primarily caused by a variety of compounds toxic to the liver that are found in plants, the most important of which are the pyrrolizidine alkaloids (PAs). Is due to insufficient excretion of photosensitizing substances In hepatic

or biliary insufficiency excretion of these substances is retarded and photosensitization occurs.

Important plants causing secondary photosensitization in animals

- ✓ Lantana camara
- ✓ Agave leche guilla
- ✓ Groundsels, Senecio and
- ✓ Panicum



Figure 2.18 Lantana camara



Figure 2.19 Groundsels, Senecio



Figure 2.20 Agave leche guilla



Figure 2.21 Panicum

Clinical signs

Clinical signs of photosensitization can include:

- Skin redness, swelling, or irritation
- Scabbing or crusting of the skin
- Lesions or blisters on areas of the skin that are exposed to sunlight such as the ears, nose, lips, udder, or teats
- Itching or pain in affected areas
- Loss of hair or wool where skin damage has occurred
- In severe cases of photosensitization, animals may also experience secondary infections, fever, anorexia, depression, and lethargy.



Figure2.22 Photosensitization, causing Scabbing or crusting of the skin cattle



Figure 2.22 Photosensitization, causing cracking and peeling of the skin, in an adult horse.

Diagnosis

To diagnose photosensitization in animals, a veterinarian will typically perform a physical examination and conduct a medical history review. The veterinarian may observe and evaluate the animal's skin lesions to determine the severity and location of the condition.

Treatment:

Immediate removal from direct sunlight

Topical medications - In some cases, applying a topical medication to the affected area can help to reduce swelling and inflammation.

Prevention from ingestion of photodynamic agent

Prevention and control

Preventing photosensitization in animals involves controlling their exposure to sunlight and also the ingestion of plants that can cause this condition. Here are some measures that you can take to prevent or control photosensitization in animals:

- Use dark skinned breeds in endemic areas

- Avoid exposing your animals to sunlight during peak hours when the sun's UV radiation is most intense, typically from about 10 am to 4 pm.
- Provide shade and shelter for your animals during the hottest parts of the day.
- Avoid feeding your animals plants that are known to cause photosensitivity.

2.2.7. Plants Causing Thiamine Deficiency

- **Bracken fern (Pteridium aquilinum)**

Bracken fern is a common plant found in many parts of the world. Several toxic principles are recognized and poisoning in non-ruminants is due to presence of thiaminase that leads to thiamine deficiency. Most common species affected are horses and cattle.



Figure2.23 Panicum(Bracken fern)

Etiology

Consumption of large amounts of bracken fern over time.

Clinical sign

In cattle different clinical manifestations of the poisoning by bracken fern are reported. When large amount of plants are consumed in a short period of time aplasia of the bone marrow develops and results in mortality. Thrombocytopenia, neutropenia and anemia develop and early myeloid cells are destroyed. Those animals consuming less quantity for longer period develop hematuria and are called enzootic hematuria. All these lead to death due to anemia and chronic

wasting. The pathological findings include widespread petechial and ecchymotic hemorrhage in intestines, serosal surfaces of several internal organs including urinary bladder, gall bladder, heart, subcutaneous tissue and muscles.

Diagnosis

Diagnosis is by evidence of consumption of the plant material with appropriate clinical signs and pathological findings.

Treatment

The treatment of bracken fern poisoning in animals involves administering thiamine (Vitamin B1) supplements to counteract the effects of thiaminase, thus restoring normal levels of thiamine in the body.

2.2.8. Teratogenic Plants

Teratogenic plants are those that have the potential to cause birth defects or fetal malformations in animals or humans. These plants contain naturally occurring toxins or chemicals that can harm the developing fetus. Some examples of teratogenic plants in animals include:

- Bracken Fern: Bracken Fern contains a compound called ptaquiloside, which is known for its teratogenic effects in animals.
- Lupine: Some species of lupine contain alkaloids that can cause skeletal deformities and other birth defects in livestock when ingested by pregnant animals.
- Poison hemlock: This plant contains a toxin called coniine, which can cause teratogenic effects when consumed by pregnant animals.
- Locoweed: Locoweed is known to cause birth defects and other reproductive problems in livestock, especially when consumed by pregnant animals.
- Tall Fescue: The fungus that infects tall fescue called "Neotyphodium coenophialum" produces an alkaloid toxin that can cause fetal development problems in the livestock that consume it.

It is important to note that these plants may not always cause teratogenic effects in all animals or humans who come into contact with them.

Etiology

Caused by accidental ingestion of plant by animals or humans.

Clinical signs

The clinical signs of an animal affected by teratogenic plants can vary depending on the specific plant and the severity of the condition. However, some common signs include:

- ✓ Abnormal growth of fetuses or newborns
- ✓ Deformities in fetuses or newborns
- ✓ Failure of pregnancy
- ✓ Reduced fertility in reproductive animals
- ✓ Weakness, lethargy, and lack of appetite
- ✓ Digestive issues such as vomiting and diarrhea
- ✓ Photosensitivity (in some cases)

It's important to note that not all animals who consume teratogenic plants will show clinical signs, and in some cases, the effects may not be immediately visible. Therefore, it's crucial for animal owners to take preventative measures to avoid their animals consuming teratogenic plants in the first place.



Figure 2.24. Lupine Induced "Crooked Calf Disease.

Diagnosis

Diagnosing an animal with teratogenic plant poisoning can be challenging, as the clinical signs may also be associated with other conditions. However, a veterinarian may suspect teratogenic plant poisoning if the animal is showing any of the clinical signs mentioned earlier and has access to plants known to be teratogenic. To make a definitive diagnosis, the veterinarian may perform a thorough physical examination and run various diagnostic tests such as blood tests, urinalysis, and radiographs to evaluate the animal's overall health and check for any abnormalities. Additionally, if fetal abnormalities or failures have occurred, postmortem examinations of dead fetuses or newborn animals can provide evidence of teratogenic plant poisoning.

Treatment

The treatment for animals affected by teratogenic plants largely depends on the severity of the condition. Mild cases may only require supportive care and removal from the source of the plant, while more severe cases may require hospitalization and symptomatic treatment such as administration of fluids and electrolytes.

Control

Controlling animals from teratogenic plants involves several measures, including identifying the presence of such plants in grazing areas, preventing access to pastures or areas where these plants are present, and implementing measures to remove or control such plants' growth.

Here are some ways to control animals from teratogenic plants:

Identification of Teratogenic Plants

Physical barriers

Grazing Management

Removal of Teratogenic Plants

Providing Nutritious Forages

Self-Check – 2	

Name…… ID…… Date…….

Directions: Answer all the questions listed below.

Part I. Choose the best answer (5pts each)

1. Toxin produced by plants is ?
   A. Chemical toxin C. Plant toxin
   B. Viral toxin D. Bacterial toxin
2. One of the following is plant causing thiamine deficeiency
   A. Alfalfa C. Lantana camara
   B. Bracken fern D. None
3. _____ is the disease caused by the sensitization of the superficial layers of lightly pigmented skin and mucus to light of certain wavelength.
   A. Neoplasia C. Bloat
   B. Milk fever D. Photosensitization

Part II GIVE SHORT ANSWER.

1. Define the following terms (5pts.)
   a) Poisin
   b) Poisonin
2. Discuss aetiology, clinical sign, diagnosis, treatment and prevention of cyanide Poisoning (5pts)

Note: **Satisfactory rating – 25 points** **Unsatisfactory - below 25 points**

LG #10	LO3. Identify and handle metabolic and nutritional causes of animal diseases

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following content coverage and topics:

- Aetiology, clinical sign, diagnosis, treatment and prevention of nutritional imbalances
- Aetiology, clinical sign, diagnosis, treatment and prevention of metabolic disorders

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, you will be able to:

- Identify and implement aetiology, clinical sign, diagnosis, treatment and prevention/control methods of diseases caused by nutritional imbalances.
- Identify and implement aetiology, clinical sign, diagnosis, treatment and prevention/control methods of animal diseases caused by metabolic disorders.

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the information Sheets
4. Accomplish the Self-checks
5. Perform Operation Sheets
6. Do the “LAP test”

Information Sheet 3

3.1. Aetiology, clinical sign, diagnosis, treatment and prevention of nutritional imbalances

Nutritional imbalances in animals can occur when an animal's diet does not contain the proper balance of nutrients that the animal requires to maintain good health. These imbalances can be caused by a variety of factors, such as feeding a diet that is deficient in a particular nutrient, feeding too much or too little of a particular nutrient, feeding a diet that has an insufficient level of protein, or feeding too much of a particular kind of feedstuff.

3.1.1. Protein and energy source deficiency

- **Deficiency of energy**

A deficiency of energy is the most common production-limiting nutrient deficiency of farm animals. There may be inadequate amounts of feed available, or the feed may be of low quality (low digestibility). The availability of pasture may be inadequate because of overgrazing, drought, or snow covering. Alternatively, it may be too expensive to provide enough supplementary feed of the required quality, or the available feed may be of such low quality and poor digestibility that animals cannot consume enough to meet energy requirements. In some cases, forage may contain a high concentration of water, which limits total energy intake.

Aetiology

Insufficient quantity or quality of feed is a common nutritional deficiency and practical problem of feeding livestock. The term protein-energy malnutrition is used to describe a form of incomplete starvation in which a suboptimal amount of energy and protein is present in the diet. Such deficiencies typically occur when livestock are underfed, and often the two scenarios cannot be separated.

Clinical signs

The clinical findings of an energy deficiency depend on the age of the animal, whether or not it is pregnant or lactating, concurrent deficiencies of other nutrients, and environmental factors. In general, an insufficient supply of energy in young livestock causes decreased growth and delayed onset of puberty. In mature animals, there is reduced milk production and a shortened lactation. A prolonged energy deficiency in pregnant beef heifers will result in a failure to produce adequate quantities of colostrum at parturition.

In mature animals, there is also a marked loss of body weight, especially when demand for energy increases in late pregnancy and early lactation. There are prolonged periods of anestrus, which reduces the reproductive performance of the herd.

A deficiency of energy during late gestation can produce undersized, weak neonates with a high mortality rate, whereas abomasal impaction is associated with energy deficiency during prolonged cold weather, especially in pregnant beef cattle and ewes being wintered on poor-quality roughage.

Diagnosis

Diagnosing energy deficiency in animals typically involves a combination of clinical evaluation and laboratory tests.

Treatment

The treatment of energy deficiency in animals depends on the underlying cause and severity of the deficiency. In mild cases, adjusting the animal's diet to provide more energy, such as increasing the amount of feed or providing a more nutrient-dense diet, may be sufficient to address the problem. In more severe cases, or when the animal is unable to eat, other interventions may be necessary.

If the animal is not eating, it may be necessary to provide enteral nutrition, which involves feeding the animal through a tube that is placed in the stomach or intestines. This allows for delivery of nutrients directly to the digestive tract and can help improve the animal's energy levels and overall health.

Prevention and control

Prevention and control of energy deficiency in animals involves a combination of management practices, proper feeding practices, and regular veterinary care. Here are some steps that can help prevent and control energy deficiency:

Provide a balanced diet: Ensure that the animal's diet provides all the essential nutrients, including adequate energy intake. Work with a veterinarian or animal nutritionist to develop a feeding plan that meets the specific nutritional needs of the animal.

Monitor feed intake: Regularly monitor the amount of feed that the animal is consuming to ensure that it is meeting its energy requirements.

Avoid sudden changes in feed: Sudden changes in feed can lead to digestive upset and decrease feed intake, which can contribute to energy deficiency. Gradual changes to the animal's diet are recommended.

Proper storage of feed: Ensure that feed is stored in a dry, cool, and well-ventilated area to prevent spoilage and mold growth, which can decrease the nutritional value of the feed.

Regular veterinary care: Schedule regular check-ups with a veterinarian to monitor the animal's health and nutritional status. Early detection and management of any health problems can help prevent and control energy deficiency.

Proper housing: Ensure that the animal's housing environment is appropriate for its species and age. Cleanliness and good ventilation are important factors in maintaining animal health. By following these best practices, you can help prevent and control energy deficiency in animals, ensuring that they remain healthy and productive.

- **Deficiency of protein**

A deficiency of protein commonly accompanies a deficiency of energy. However, the effects of the protein deficiency, at least in the early stages, are usually not as severe as those of energy deficiency.

Etiology

Protein deficiency in animals can have several causes, including inadequate protein intake, poor digestibility of protein in the diet, altered protein metabolism due to illness or disease, and

increased protein requirements that may exceed the animal's capacity for synthesis. In some cases, protein deficiency can be compounded by other nutrient deficiencies or imbalances, such as inadequate intake of essential amino acids or certain vitamins and minerals that are required for protein synthesis.

Clinical sings

The clinical findings of a protein deficiency are similar to those of an energy deficiency, and the clinical findings of both resemble those of many other specific nutrient deficiencies and subclinical diseases. Protein malnutrition in beef cattle occurs most commonly in late gestation and is characterized clinically by weakness, clinical recumbency, marked loss of body weight, a normal mental attitude, and a desire to eat.

Diagnosis

The diagnosis will depend on an estimation of the concentration of energy and protein in the feed, or a feed analysis, and comparing the results with the estimated nutrient requirements of the class of affected animals. In some cases, a sample of feed used several weeks earlier may no longer be available, or the daily feed intake may not be known. Marginal deficiencies of energy and protein may be detectable with the aid of a metabolic profile test.

Treatment

The treatment of protein deficiency in animals involves providing them with protein-rich foods and supplements. The following are some methods commonly used to increase protein intake in animals:

Increasing dietary protein: Feeding animals with protein-rich food sources such as meat, eggs, dairy products, beans, and grains can help treat protein deficiency.

Protein supplements: Adding protein supplements such as soybean meal, fishmeal, or whey protein powder to the animal's diet can help increase their protein intake.

Antibiotics: In cases where a bacterial infection is causing the deficiency of protein, antibiotics may be prescribed to treat the infection and restore the animal's health.

It is important to note that the underlying cause of the protein deficiency should be identified and addressed in addition to providing adequate dietary protein.

Prevention

Proper nutrition and monitoring are crucial for preventing and treating protein deficiencies in animals.

3.1.2. Vitamin and mineral deficiency

- **Deficiencies of Vitamins**
  - ✓ **Vitamin A Deficiency (hypovitaminosis A)**

Vitamin A is essential for the regeneration of the visual purple necessary for dim-light vision, for normal bone growth and for maintenance of normal epithelial tissues. Deprivation of the vitamin produces effects largely attributable to disturbance of these functions. Major sources of this vitamin are preformed vitamin A from animal sources (fish oil, egg yolk, liver) and provitamin carotene from plants. Dark green leafy vegetables are the good sources of carotenes.

Etiology:

Inadequate intake of this vitamin or its carotenoid precursors in the diet, or

Dietary supply of the vitamin or its precursor is adequate, but their digestion, absorption or metabolism is interfered with to produce a deficiency at tissue level.

Clinical signs

Clinical signs of deficiency may include poor coat quality, night blindness, xerophthalmia (dry eyes), reproductive issues, and overall poor growth and development.

Diagnosis: History and clinical signs (night blindness, dry eye, poor growth)

Treatment: High dose of vitamin parenterally, even ten times of it may be warranted in some vulnerable sick animals. Doses: Calf 1,000,000 to 1,500,000 IU, cow 250,000 to 2,500,000 IU, lamb 125,000 to 250,000 IU, horse 200,000 to 300,000 IU.

- ✓ **Vitamin K Deficiency**

This vitamin is adequately available in the plants and synthesized by herbivorous animals in the gut. Therefore, deficiency of it is rarely seen in animals. This vitamin helps the formation of prothrombin and takes part in the normal coagulation process. Thus deficiency of vitamin K causes prothrombinemia and hemorrhage.

Etiology

Prolonged use of oral antibiotics can retard the synthesis of this vitamin due to reduction or destruction of beneficial bacteria. High dosing with vitamin A can also interfere with bacterial synthesis of this vitamin.

Clinical signs

Vitamin K deficiency in animals can result in a variety of clinical signs, including increased bleeding and bruising, anemia, poor growth and development, and skeletal abnormalities. In severe cases, it may also lead to hemorrhagic disease of the newborn in young animals, which can be fatal if left untreated. Additionally, animals with vitamin K deficiency may have reduced clotting factors, which can result in prolonged bleeding times and hemorrhaging even from minor injuries.

Diagnosis

Diagnosis of vitamin K deficiency in animals can be made through a combination of physical examination, medical history, and laboratory testing.

Treatment

Treatment for vitamin K deficiency typically involves dietary supplementation with vitamin K.

Depending on the severity of the deficiency and the animal's condition, different routes of administration may be used, including oral administration or injection.

The treatment for vitamin K deficiency involves administration of vitamin K supplements. Vitamin K1 is the preferred supplement for animals, as it is readily absorbed and utilized. In cases of severe deficiency or when animals are unable to absorb vitamin K1, vitamin K3 may be administered instead.

- ✓ **Vitamin B Complex Deficiency**

Vitamin B complex deficiency seldom occurs in ruminants as they can synthesize adequate amount of these vitamins in the rumen through the bacteria. But, prolonged use of antibiotics and sulphonamides through oral route cause reduction of bacterial population and thus impede the synthesis of this vitamin. High carbohydrate diet may also reduce the bacterial population and synthesis of this vitamin.

Clinical signs

Clinical signs of vitamin B complex deficiency include lethargy, weakness, anorexia, weight loss, and dermatological issues. In advanced cases, neurological symptoms such as seizures and ataxia may occur.

Diagnosis

To diagnose vitamin B complex deficiency in animals, veterinary professionals may perform blood tests to check for low levels of specific B vitamins.

Treatment

Treatment typically involves administration of B vitamin supplements, including thiamine, riboflavin, niacin, pantothenic acid, pyridoxine, folic acid, and cobalamin. Supplements may be given orally or via injection, depending on the severity of the deficiency.

Prevention

Prevention of vitamin B complex deficiency in animals involves providing a well-balanced diet that contains sufficient amounts of all necessary vitamins and minerals.

- ✓ **Vitamin D Deficiency (hypovitaminosis D)**

Vitamin D deficiency is usually caused by insufficient solar irradiation of animals or their feed and is manifested by poor appetite and growth, and in advance cases by osteodystrophy.

Etiology

A lack of ultraviolet solar irradiation of the skin, coupled with a deficiency of calcium and phosphorous.

Clinical findings

Reduced productivity, poor weight gains, in appetence, and infertility. In late stages there is rickets in young and osteomalacia in adults.



Figure3.1. Vitamin D deficiency in calves

Diagnosis:

Based on history of feeding vitamin D deficient diets and less exposure to sunlight, clinical signs, response to vitamin D therapy.

Treatment and control:

- Arrange exposure to solar irradiation
- Include sun-dried hay in diet

✓ **Selenium And/ Or Vitamin E Deficiency**

Selenium is an essential nutrient for animals. Selenium and/or Vitamin E deficiency is a common issue in animals, especially in those who are unable to obtain sufficient amounts of these nutrients from their diet.

Etiology

Selenium deficiency occurs in animals fed plant material grown on soils poor in the element. Vitamin E deficiency may be caused primarily by deficiency in the feed

Clinical findings*:* The clinical effects are manifested principally in skeletal muscles but occur also in most other organs.

a) **Acute enzootic muscular dystrophy:** Sudden death, without prior illness, often after exercise and excitement.

b) Sub-acute enzootic muscular dystrophy (white muscle or stiff lamb disease)

- ✓ Sternal recumbency
- ✓ Patient anxious to stand but most unable to do so
- ✓ Standing patients have rigidity, tremor, stumbling gait, easy falling
- ✓ Weakness and stiffness followed by paralysis
- ✓ Large, upper limb muscle masses symmetrically swollen, firm
- ✓ Myopathy as muscular degeneration or "white muscle disease"
- ✓ Retention of placenta in cattle
- ✓ Dyspnea, transient fever in some
- ✓ Heart rate moderately elevated, no irregularity

Diagnosis: history of diets, clinical signs, lesions in muscles and estimation of vitamin E. Respond to vitamin E therapy may confirm diagnosis.

Treatment: administration of vitamin E and selenium-containing medicine.

- **Deficiency of Minerals**

At least 15 mineral elements are nutritionally essential for animals. The macrominerals are calcium, phosphorus, potassium, chlorine, magnesium, and sulfur. The trace elements, or microminerals, are copper, selenium, zinc, cobalt, iron, iodine, manganese and molybdenum. The trace elements are involved as component parts of many tissues and one or more enzyme activities and their deficiency leads to a wide variety of pathological consequences and metabolic defects.

- ✓ **Iodine Deficiency**

Goiter (thyroid hyperplasia) is the cardinal sign of iodine deficiency. The major clinical manifestation is neonatal mortality, with alopecia and visible and palpable enlargement of the thyroid gland.

Etiology

Primarily due to deficient iodine intake or secondarily conditioned by a high calcium intake.

Iodine deficiency reduces the ability of thyroid gland to produce thyroxin hormone.

It stimulates the secretion of TSH by pituitary gland.

It causes hyperplasia of the thyroid gland and goiter.

Clinical findings

Thyroid hyperplasia, loss of libido in the bull, failure to express estrus in cow, hair or wool loss (alopecia), high incidence of abortion, stillbirth and weak newborn animals.

Diagnosis:

Easily diagnosed if goiter is present but the occurrence of stillbirths without goiter may be confusing. Estimation of iodine levels in the blood and milk is needed.

Treatment:

Potassium iodine 200mg/kg dry feed or provision of iodine salt preparations.

Recommended intake of iodine for lactating and pregnant cows is 0.8-1mg/kg dry feeds; for dry cow are 0.1-0.3mg/kg dry matters.

Treatment must be undertaken with care, as over dosage will cause toxicity.

- ✓ **Iron Deficiency**

Functionally iron is necessary for hemoglobin formation. A deficiency of iron in the diet causes anemia and failure to flourish. It is the most common problem of piglets kept under artificial conditions.

Etiology:

Primary: is most likely to occur in newborn animals whose sole source of iron is the milk of the dam, milk being a poor source of iron. Deposits of iron in the liver of the newborn are insufficient to maintain normal hemopoiesis for more than 2-3 weeks, and are particularly low in piglets. Continued blood loss by hemorrhage in any animal may bring about a sub clinical anemia and an associated iron deficiency.

Secondary: due to chronic bleeding, high infestation of cattle with sucking lice, high burden of blood-sucking strongylid parasites in horses.

Clinical findings:

The highest incidence occurs at 3 weeks of age although the disease can occur in pigs up to 10 weeks of age. Significantly lower growth rate of anemic pigs than normal pigs. There is reduced food intake. Diarrhea is very common, but the feces are usually normal in color. Severe dyspnea, lethargy, and marked increase of the apex beat with exercise. The mucosa and skin are pale, edema of the head and forequarters and death usually occurs suddenly.

Diagnosis:

Is based on clinical findings, age (neonates are highly affected), laboratory test to determine Hb level and treatment response (IM injection of iron-dextran preparations or other iron preparations).

Treatment:

The treatment is usually parenteral and consists of organic iron preparations such as iron-dextran, iron-sorbitol-citric acid complex, iron saccharate or gluconate.

The dose rate is 0.5-1g elemental iron in one injection once each week.

Prevention and control

Preventing iron deficiency in animals requires an adequate dietary intake of iron, as well as appropriate management practices to minimize the risk of iron deficiency.

- ✓ **Calcium Deficiency (hypocalcicosis)**

Calcium deficiency may be primary or secondary, but in both cases the end result is an osteodystrophy, the specific disease depending largely on the species and age of the animals affected.

Etiology:

Primary calcium deficiency is due to an absolute deficiency in the diet. This occurs rarely.

Secondary calcium deficiency is due to marginal calcium intake aggravated by high phosphorus in the diet. Such a diet depresses intestinal absorption and retention of calcium in the body, and

the absorption of calcium from bones is increased. This can also occurs accompanied by a vitamin D deficiency in animals confined indoors.

Clinical findings

The clinical sign include; decreased rate or cessation of growth, dental mal-development, soft bones, bendable ribs, easily fracture, reduced fertility, difficult parturition and tetany in pigs and young cattle.

Diagnosis

Diagnosis is based on clinical signs and diagnostic response to diet supplementation with calcium.

Treatment

Calcium borogluconate solution injection (as in milk fever) for tetany

Supplement diet with calcium (ground limestone, bone meal)

Reduce excessive phosphorus intake

Aim at calcium: phosphorus ratio of 2:1 (optimum) up to 1:1; if urolithiasis is a problem ratio should be 2.5:1.

- ✓ **Phosphorus Deficiency (hypophosphatosis)**

Phosphorus deficiency is usually primary and is characterized by pica, poor growth, infertility and, in the later stages, osteodystrophy.

Etiology

Phosphorus deficiency is usually primary under field conditions but may be exacerbated by a deficiency of vitamin D and possibly by an excess of calcium. Unlike calcium, a dietary deficiency of phosphorus is widespread under natural conditions.

Clinical findings

Primary phosphorus deficiency is common only in cattle.

Young animals grow slowly and develop rickets. In adults there is an initial sub clinical stage followed by osteomalacia. Retarded growth, low milk yield and reduced fertility are the earliest signs of phosphorus deficiency. Osteophagia is common and may be accompanied by a high incidence of botulism. Cows in late pregnancy often become recumbent and although they continue to eat are unable to rise.

Diagnosis

Diagnosis is based on clinical findings.

Treatment and control

Urgent treatment is 30g sodium dihydrogen phosphate in 300ml water IV for cattle. Daily supplementation for cattle of 15g phosphorus minimal, 40-50g optimal, as bone meal, rock phosphate, soft (colloidal clay) phosphate in form of free-access lick, mineral mix fed in concentrate or hay, pasture, in drinking water.

3.2.Etiology, clinical sign, diagnosis, treatment and prevention of metabolic disorders

Metabolic disease

Metabolic diseases are very important in dairy cows and pregnant ewes. In the other livestock species, metabolic diseases occur only sporadically. The high-producing dairy cow always verges on abnormal homeostasis, and the breeding and feeding of dairy cattle for high milk yields is etiologically related to metabolic disease so common in these animals. The term production disease includes those diseases previously known as metabolic diseases, such as parturient paresis (milk fever), hypokalemia, hypomagnesaemia, hyperketonemia and ketosis, hyperlipemia, and other conditions that are attributable to an imbalance between the rates of input of dietary nutrients and the output of production.

3.2.1. Carbohydrate engorgement (Ruminal lactic acidosis, Rumen overload)

It is a condition in which large amounts of highly fermentable carbohydrate rich feed causes an acute disease due to the excessive production of lactic acid in the rumen.

Etiology

Sudden ingestion of toxic doses of carbohydrate rich feeds especially grain.

This results in production of lactic acid that affect the microbial population of the rumen (marked increase in the number of gram positive bacteria) that further increases production of large quantity of lactic acid.

Clinical findings

The speed of onset of the illness varies with the nature of the feed.

The severity increases with the amount of feed eaten.

In the mild form; affected cattle are anorexic and still fairly bright and alert, and the feces may be softer than normal, Rumen movements are reduced but not entirely absent, affected cattle do not ruminate for a few days but usually begin to eat on the third or fourth day without any specific treatment.

The temperature is usually below normal, 36.5°C to 38.5°C but animals exposed to the sun may have temperatures up to 41°C (106°F).

In sheep and goats, the rectal temperatures may be slightly higher than normal.

Diagnosis: History of sudden access to toxic doses of grain and characteristic clinical signs.

Treatment: Correction of ruminal and systemic acidosis by using alkalinizing agents through stomach tube and /or IV by sodium bicarbonates. Cud transfer (10-20lit) from healthy animals;

Restore ruminal motility by providing high fiber content feeds. In animals still standing, rumenotomy is preferred to rumen lavage, because animals may aspirate during the lavage procedure and only rumenotomy ensures that all ingested grain has been removed. Rumen lavage may be accomplished with a large stomach tube if sufficient water is available.

Prevention and control

To prevent grain overload, accidental access to concentrates for which cattle have developed an appetite, in quantities to which they are unaccustomed, should be avoided. Feedlot cattle should be introduced gradually to concentrate rations over a period of 2–3 weeks, beginning with a mixture of ≤50% concentrate in the milled feed containing roughage.

3.2.2. Ketosis /Acetonemia of cattle

Acetonaemia or ketosis is a metabolic disorder of high yielding lactating cows characterized by reduced milk yield, loss of body weight, inappetance and, occasionally, nervous signs.

Ketone bodies, e.g. acetoacetate, bhydroxybutyrate or acetone, are present in all body fluids. Hypoglycaemia together with increased plasma free fatty acids and liver fat and decreased liver glycogen are also a feature of this disease. These changes are associated with an inadequate supply of the energy that is necessary to sustain high levels of milk production in early lactation. Pregnancy toxaemia, a common disease of pregnant sheep and characterized by hypoglycaemia and hyperketonaemia, can also occasionally affect pregnant cows particularly when carrying twins

Aetiology

To understand the aetiology of acetonaemia one must realize the precarious metabolic balance that exists in all cows in early lactation. To satisfy the requirements of milk production the cow can draw on two sources of nutrients, food intake and her body reserves. In the first two months of lactation a cow producing up to 45kg of milk daily will use up to 2kg of body fat and up to 350 g of body protein per day. As far as the dietary supply of nutrients is concerned 80 per cent of the ingested carbohydrates are fermented by the rumen microflora into the volatile fatty acids, acetic, propionic and butyric acids, which are themselves absorbed. Acetate may be oxidized by various tissues or incorporated into milk fat by the mammary gland

Clinical Signs

Hypoglycaemia is the major factor involved in the onset and development of the clinical signs of acetonaemia. There will have been a gradual loss of body condition over several days or even weeks. There is also a moderate decline in milk yield over two to four days before the onset of the obvious clinical signs, which are refusal to eat grain and concentrate feeds and a more sudden drop in milk output. At this stage a sweet smell (as in pear drops) of acetone is apparent on the breath and the discerning stock worker will even detect the same acetone smell in the milk. Once appetite is decreased weight loss is accelerated due to utilization of body stores. Rectal temperature, pulse rates and respiratory rates are normal in the early stages of the disease, as are ruminal movements. Faeces will usually be firm with a dark 'waxy' appearance. A small number of cows with acute acetonaemia exhibit nervous signs, which include; excessive salivation, abnormal chewing movements and licking walls, gates or metal bars. In coordination with apparent blindness will also be a feature. Some cows will even show a degree of aggression and will sometimes charge into walls, occasionally injuring themselves. The other signs observed above are also present. The nervous signs often only last for a few hours with the animals showing more normal behaviour in between.

Diagnosis

The diagnosis is made on the history of a cow in early lactation with a sudden fall in milk yield, some weight loss, refusing to eat concentrates, with normal temperature, pulse and respiratory rates and normal rumen movements. Many astute stock workers will recognize the acetone odor on the breath or in the milk and report this to the attending veterinarian. The diagnosis is confirmed by a positive Rothera's reaction on milk and urine and, if this is not conclusive, a blood sample can be analysed for glucose and ketone levels. It is important to differentiate between primary and secondary ketosis so a complete clinical examination must be performed. Many cases presented by the farmer as acetonaemia are in fact suffering from displaced abomasum (p. 839). Some cows with hypocalcaemia (p. 783) may also show acetonaemia

Treatment

An intravenous infusion of 500ml of 40 per cent glucose will cause a transient rise in blood glucose levels that lasts approximately two hours. This should be accompanied by oral administration of glucose precursors such as propylene glycol (150ml, twice daily). Glucocorticoid drugs are the most commonly used therapy for acetonaemia, either used alone or in combination with glucose therapy or when followed by oral administration of glucose precursors.

Prevention

The prevention of acetonaemia starts before calving. Cows should not be too fat at calving, a condition score of 2.5–3.0 would be optimum and anything higher would be considered too fat. Access to a plentiful supply of long coarse fiber to promote good rumen digestion is also important during the dry period. Concentrates used during lactation should be introduced in small quantities (1–2kg/day) two weeks before calving to allow adjustments in the rumen micro flora. Changes to diet in early lactation should be made gradually.

3.2.3. Parturient paresis/milk fever

Aetiology

It is caused by a sudden drop in calcium levels in the blood due to the high demand for milk production and insufficient calcium intake.

Clinical findings

Three stages of milk fever in cattle are commonly recognized and described.

Stage 1 In the first stage, the cow is still standing. This is also the brief stage of nervousness, excitement, and tetany with hypersensitivity and muscle tremor of the head and limbs. The animal is disinclined to move and often has a decreased or no feed intake. There may be a slight shaking of the head, protrusion of the tongue, and grinding of the teeth. The rectal temperature is usually normal to slightly above normal; the skin may feel cool to the touch. The animal appears

ataxic, with a stiff and insecure gait, and falls easily. Close examination reveals agalactia, rumen stasis, and scant feces. Heart rate and respirations may be within normal limits or slightly elevated.

Stage 2 The second stage is characterized by sternal recumbency with depressed consciousness; the cow has a drowsy appearance in sternal recumbency, usually with a lateral kink in the neck or the head turned into the flank. When approached, some of these cows will open their mouths, extend the head and neck, and protrude their tongues, which may be an expression of apprehension and fear in an animal unable to stand. The tetany of the limbs present in the first stage is not present, and the cow is unable to stand. The muzzle is dry, the skin and extremities cool, and the rectal temperature subnormal (36 to 38°C, 97 to 101°F). There is a marked decrease in the absolute intensity of the heart sounds, whereas the heart rate is increased (about 80 bpm). The arterial pulse is weak and the venous pressure is also low, making it difficult to raise the jugular veins. The respirations are not markedly affected, although a mild forced expiratory grunt or groan is sometimes audible. Ruminal stasis and secondary bloat are common, and constipation is characteristic. There is also relaxation of the anus and loss of the anal reflex. The eyes are usually dry and staring. The pupillary light reflex is incomplete or absent, and the diameter of the pupil varies from normal to maximum dilatation. A detailed examination of the pupils of cows with parturient paresis, nonparetic disorders, and nonparturient paresis found that the mean sizes of the pupils were not significantly different from one another. Rather, disparity of the size of the pupils was common. In cows that develop hypocalcemia a few hours before or at the time of parturition, the second stage of parturition may be delayed. Vaginal examination usually reveals a fully dilated cervix and normal presentation of the fetus. The cow may be in any stage of milk fever, and administration of Ca-salts IV will usually result in a rapid beneficial response and normal parturition. Prolapse of the uterus is a common complication of milk fever, and often the Ca levels are lower than in parturient cows without uterine prolapse. Thus it is standard practice to treat cases of uterine prolapse with IV calcium salts.

Stage 3 The third stage is characterized by a severely obtunded or even comatose cow in lateral recumbency. There is complete flaccidity on passive movement, and the cow cannot assume

sternal recumbency on its own. In general, the depression of temperature and the cardiovascular system are more marked. The heart sounds are almost inaudible, and the rate is increased up to 120 bpm; the pulse is almost impalpable, and it may be impossible to raise the jugular veins. Bloat is common because of prolonged rumen stasis and lateral recumbency. Without treatment, a few animals remain unchanged for several hours, but most become progressively worse during a period of several hours and die quietly from shock in a state of complete collapse.

Diagnosis

A diagnosis of milk fever is based on the occurrence of paresis and depression of consciousness in animals following parturition. The diagnosis is supported by a favorable response to treatment with parenteral injections of calcium solutions and by biochemical examination of the blood. In ewes, the history usually contains some reference to recent physical stress, and the disease is more common in the period preceding lambing

Treatment

Milk fever cases should be treated with 500 milliliters of 23 percent calcium gluconate IV and followed by the administration of two oral calcium bolus given12 hours apart. It is important to emphasize that oral calcium bolus should not be administered if cows do not respond to the calcium IV treatment.

In milk fever cows, failing to rise after treatment with IV calcium is a signal that normal muscular function has not been reestablished. Cows may choke on the calcium bolus if treatment is given while they are still down. A veterinarian should be consulted and further treatment should be evaluated when milk fever cows do not respond to IV administration of calcium.

Do NOT give calcium IV to cows with no signs of milk fever

Control

Reduce dietary calcium intake 2 to 3 weeks before calving to less than 20 g Ca/cow/ day (R-1) Reduce dietary potassium content as much as possible in late gestation (in any case, below 2% in feed dry matter) confirmation.

3.2.4. Pregnancy toxemia (twin lamb disease) in sheep

Etiology

A multi factorial disorder of energy metabolism, with hypoglycemia and ketonemia (the accumulation in blood of acetoacetate, β-hydroxybutyrate, and their decarboxylation products acetone and isopropanol).

Clinical findings

Encephalopathy with blindness, muscle tremor, convulsions, metabolic acidosis, and a clinical course of 2 to 8 days, usually terminating fatally unless treated early.

The earliest signs of ovine ketosis are separation from the group, altered mental state, and apparent blindness, manifested by an alert bearing but a disinclination to move. Sheep at pasture may fail to come up for supplementary feeding, and housed sheep may stand near the feed trough with other sheep but not eat. The ewe will stand still when approached by attendants or dogs and will turn and face them, but it will make no attempt to escape. If it is forced to move, it blunders into objects; when an obstacle is encountered, it presses against it with its head. Many affected ewes stand in water troughs all day and lap the water. Constipation with dry, scanty feces is common, and there is grinding of the teeth.

Diagnosis

Diagnostic confirmation of Ketonemia, ketonuria, or elevated ketones in milk. Elevated β-hydroxybutyrate (BHBA) in aqueous humor of dead sheep.

Treatment

Parenteral glucose with corticosteroid and oral glucose precursors such as propylene glycol, occasionally insulin, or oral glucose and electrolyte therapy. Cesarean section or induction of parturition. Case fatality high.

Control

Monitoring of condition score, pasture availability, feeding, and biochemical indicators of ketosis. Correction of energy imbalance if detected

3.2.5. Bloat (ruminal tympany)

Bloat refers to an excessive accumulation of gas in the rumen and, because of a failure to eructate, rumen distension occurs, frequently resulting in death. It is a major cause of death in cattle in all intensive livestock areas of the world.

There are two types of bloat, free gas bloat or secondary rumen tympany and frothy bloat or primary rumen tympany.

i. **Free Gas bloat (secondary rumen tympany):** Any condition that causes an esophageal obstruction or that interferes with eructation will produce gaseous bloat. Physical obstruction of the oesophagus with potatoes or other root vegetables causing choke will prevent eructation. The condition is generally sporadic in occurrence and is less common than frothy bloat. The following conditions will lead to gaseous bloat. Lesions of the esophageal groove, e.g. vagus indigestion, abscessation or infection with A. lignieresii obstruct the groove and prevent eructation. Inability to eructate is also a feature of tetanus and milk fever and gaseous bloat is a frequent feature of these diseases. Prolonged lateral recumbency as a result of disease or animals that are cast for prolonged surgery will frequently lead to gaseous bloat because of the inability of the rumen gas to escape.

ii. **Frothy bloat (primary rumen tympany):** Frothy bloat is much more common than gaseous bloat and usually affects several animals in the group at the same time. Although frothy bloat does occur in feedlot cattle it is more generally associated with pasture feeding. Pastures that are most commonly incriminated in the cause of frothy bloat usually contain high levels of leguminous plants, particularly clover or alfalfa.

Clinical signs

Rumen tympany, as evidenced by distension of the left sub lumbar fossa, is well recognized by most stock workers and should present no problems. Dyspnea and grunting are marked and are accompanied by mouth breathing, protrusion of the tongue, extension of the head, and frequent urination. Rumen motility does not decrease until bloat is severe. If the tympany continues to worsen, the animal will collapse and die. In acute frothy bloat the disease progresses rapidly and the animal soon becomes recumbent and can die in 30–60 minutes from the onset of tympany.

Diagnosis

The preliminary diagnosis presents no problems and is based on distension of the left sub lumbar fossa. If only one animal is affected in the herd the bloat is probably a gaseous bloat but if several animals are affected to varying degrees and they are at pasture the diagnosis will certainly be frothy bloat. However, if there is any doubt the passage of a stomach tube will provide the answer. If the problem is one of gaseous bloat and the stomach tube reaches the rumen and possibly removes an obstruction on the way, the gas will escape through the stomach tube and the rumen will rapidly revert to its normal size. If gaseous bloat is confirmed and the bloat relieved, a full clinical examination should be performed to ascertain the cause of the failure to eructate.

If the bloat is due to froth little or no gas will escape via the stomach tube, which will itself become blocked with froth.

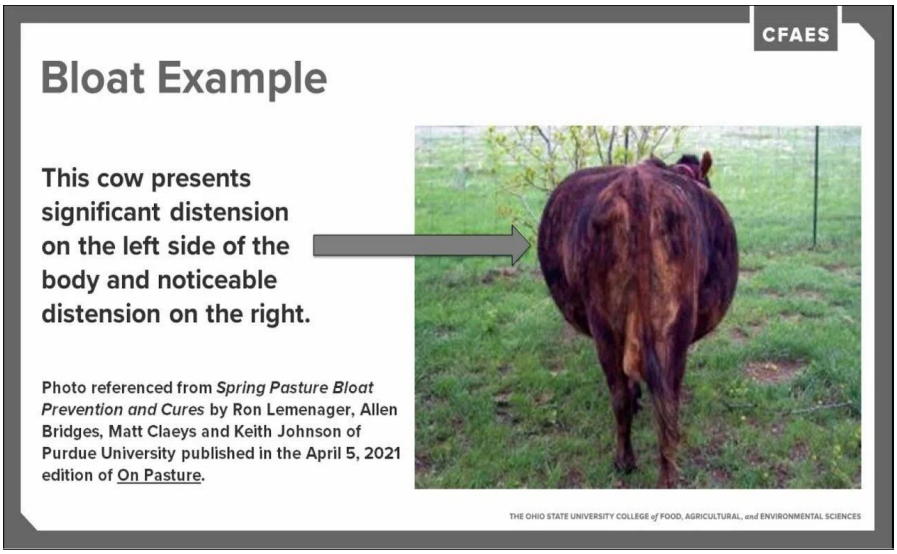


Figure 3.2 bloat in cattle

Treatment

Frothy bloat: antifoaming agents, administered ororuminally, Indigestion powder/$MgSO_4$

Free-gas bloat: placement of stomach tube or removal of an esophageal obstruction.

A trocar and cannula may be used for emergency relief of free-gas bloat, although the standard-sized instrument is not large enough to allow the viscous, stable foam in per acute cases to escape quickly enough.



Figure 3.3 trocar and cannula



Figure3.4. Stomach tube



Figure 3.5 Insertion of stomach tube

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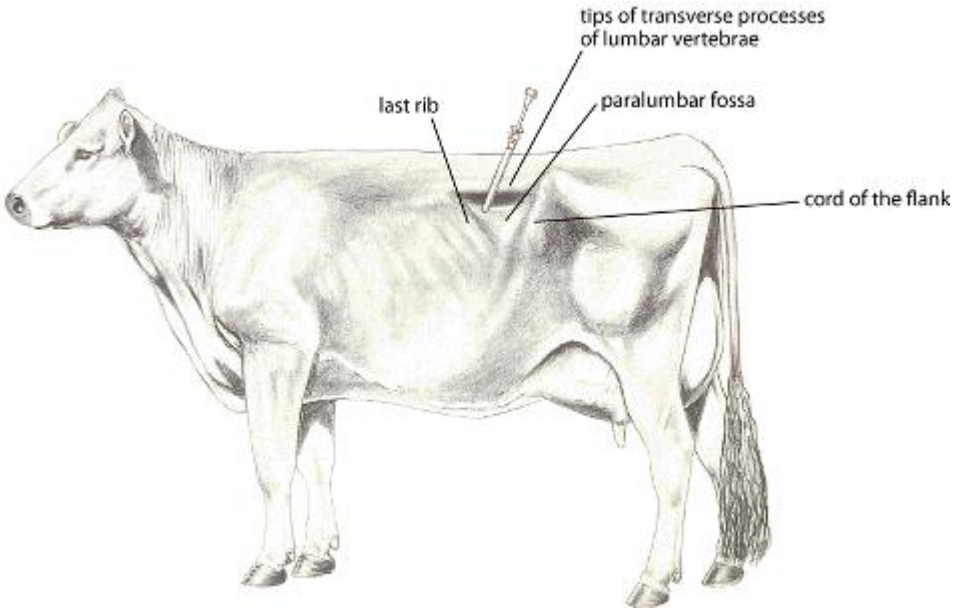


Figure 3.6 Illustration of cannulation of a cow rumen. The ideal location to ensure optimal free-gas release is midway in the Para lumbar fossa and 5–10 cm below the transverse processes of the lumbar vertebrae. Illustration by Dr. Gheorghe Constantinescu.

Prevention

To prevent further cases of frothy bloat occurring when a sudden acute outbreak is encountered at pasture the cattle should he removed immediately, provided with dry food such as hay or straw and all cows showing any degree of rumen tympany drenched with an antifoaming agent. The pasture should not be used for grazing for at least 10 days.

Where risk pastures exist, e.g. those containing high proportions of legumes, gradual access to the pasture should be practiced, starting with 10 minutes a day and increasing by 10 minutes each day. Long fiber should be fed before access is allowed to the pasture.

Free gas bloat in animals can be prevented by, providing adequate and consistent access to fresh water, feeding smaller, more frequent meals to avoid overeating and ruminal distention, avoiding sudden changes in feed or introducing new feedstuffs abruptly, ensuring that animals are not grazing on legumes or cereal crops that are bloating-prone, manage grass tetany, as treatment can cause ruminal stasis leading to bloat.

Self-Check –3	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below.

Part I. Write true if the statement is correct and write false if the statement is incorrect (2pts each.)

1. A deficiency of energy is the most common production-limiting nutrient deficiency of farm animals
2. Any condition that causes an esophageal obstruction or that interferes with eructation will produce gaseous bloat..

Test III. GIVE SHORT ANSWER.

1. Discuss bloat(5pts
2. Discuss etiology, clinical sign, diagnosis, treatment and prevention of metabolic disorders(10pts)
3. Discuss Protein and energy source deficiency(5pts)

Note: **Satisfactory rating – 24 points** **Unsatisfactory - below 24points**

Operation Sheet -3

Procedures of trocarization for free gas bloat in cattle

A. Tools and equipments

The following tools and equipment are typically required for trocarization in cattle:

i. Trocar and cannula:
ii. Scalpel or large-gauge needle:
iii. PPE:
iv. Sterilized dressing materials: E.g. Sterile gauze pads, adhesive bandages, sterile adhesive strips, sterile wound dressings, and sterile cotton balls.
v. Antiseptic solution:
vi. Restraint equipment:.
vii. Stethoscope:

B. Steps for trocarization

1. Assess the animals to see how severe bloat is.
2. Restrain the animal that needs treatment.
3. Disinfect and clean the area where trocar insertion will take place.
4. Make a small incision into the skin in same area as the previous step. The incision should only be 1 centimeter (0.39 in) long so that the trocar and cannula can be placed into the rumen. The hole also needs to be small enough so that the cannula will stay in.
5. Insert the trocar (cannula attached) through the abdominal muscles into the rumen through the hole just made with your knife. Grasp the top of the handle of the trocar and with one move inward, punch through the abdominal muscles into the rumen. This will take a concerted effort (done right and only once) because the abdominal muscles are not soft like cookie dough. It will hurt the animal, but remember this is done not for the purpose of hurting the animal, but rather to save its life.

6. Remove the trocar and leave the cannula in the hole. Doing so allows the froth and gases to escape from the rumen.
7. Leave the cannula in place for several hours, or a few days if the animal keeps having bloat issues.
8. Check the animal and the cannula regularly for any blockage that can occur when the froth solidifies via drying when exposed to air.
9. Check with the vet as well, as you may need to inject an antibiotic like penicillin to ward off infection. Infection is inevitable with having a cannula in the animal for several days.
10. Have the vet out to remove the cannula and suture up the damaged tissues.

LAP TEST-3	Performance Test

Name_____ ID_____ Signature_____-Date_____

Time started: 2:30 Time finished: 3:30 local time

Instructions: Given necessary templates, tools and materials you are required to perform the following tasks within 1 hour. The project is expected from each student to do it.

Task1. Perform trocarization in cattle

LG #11	LO4. Identify and handle miscellaneous causes of animal diseases

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following content coverage and topics:

- Aetiology, clinical sign, diagnosis, treatment and prevention of
  - ✓ Colic
  - ✓ Abomasal displacement
  - ✓ Diabetics
  - ✓ Physical trauma
  - ✓ Traumatic reticulo pericarditis (TRP)
  - ✓ Neoplasia

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, you will be able to:

- Identify and implement aetiology, clinical sign, diagnosis, treatment and prevention/control methods of diseases caused by hormonal deficiencies and colic.
- Identify and implement aetiology, clinical sign, diagnosis, treatment and prevention/control methods of diseases caused by physical trauma
- Identify and implement aetiology, clinical sign, diagnosis, treatment and prevention/control methods of diseases caused neoplastic causes of animal disease

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the information Sheets
4. Accomplish the Self-checks

Information Sheet 4

4.1.Aetiology, clinical sign, diagnosis, treatment and control of colic

Colic: - is a complex symptom exhibited by animals when there is painful condition of any abdominal organs. It is common in equine specie; however, it may occur in other animals as well.

Etiology

Feed related: -coarse roughage may predispose the horse to improper digestion of feedstuffs with a resultant impaction.

Water related-insufficient amounts of water create dry ingesta prone to impaction;

Poor teeth- similar to poorly digestible feeds, improper mastication cause some impactions.

Parasitic agents: migrating larval forms of S.vulgaris interfere with circulation and innervations of various parts of the large intestine, which affects gut motility leads to impaction.

Clinical sign

Clinical sign of colic includes, pawing, rolling, sweating, kicking at the abdomen, getting up and down frequently, flank-watching, hyper motility of intestine, increased respiratory and heart rate, absence of faeces.

Diagnosis: clinical findings and history of the animal related to management of feed.

Treatment: treatment should aim to relief pain targeting to the predisposing factors.

- ✓ Use of analgesics like flunixinmeglumine, xylazine, phenaylbutazole
- ✓ Use of laxatives in case of impactive colic for softening the mass
- ✓ Surgical exploration or laparotomy
- ✓ Regular deworming of horses

Control

The ideal management that prevents colic includes the following recommendations:

- Feed at least 60 percent of the daily ration as forage (hay or pasture)
- When possible, pasture in non-irrigated fields and/or use a grazing muzzle to control weight and intake of rich forage
- Limit grain to as little as possible – none is preferable

- Substitute high-fat feeds and high-fiber feed for grain supplements when more calories are needed
- Provide feeding systems that limit the intake of sand and dirt
- Provide plenty of turnout and exercise each day
- Provide clean, ice-free drinking water
- Implement regular and frequent deworming programs for the herd
- Implement a herd health program of preventive care
- Minimize stress (transport, herd dynamics, housing, illness, injury) as much as possible

4.2. Etiology, clinical sign, diagnosis, treatment and prevention of abomasal displacement

Abomasal displacement is a condition in which the abomasum (the fourth compartment of the stomach) of an animal moves out of its normal position. Most abomasal displacements are left-sided and occur within the first 2 months after calving in grain-fed dairy cattle, particularly Holsteins.

Etiology

There are two forms of abomasal displacement in cattle:

- **Left displaced abomasum (LDA)** is displacement of the gas-filled, distended abomasum to the left side of the abomasum, trapping it between the rumen and the abdominal wall
- **Right displaced abomasum (RDA)** is displacement of the gas-filled, distended abomasum from the ventral abdominal wall into the craniodorsal right abdominal cavity.

The etiology of displaced abomasum is multifactorial, although decreased abomasal emptying by abomasa hypo motility and/or dysfunction of the intrinsic nervous system are thought to play an important role in development of displacement or volvulus.

Clinical sign

The typical history of abomasal displacement includes anorexia (most commonly a lack of appetite for grain with a decreased or normal appetite for roughage) and decreased milk production (usually notable but not as extensive as with traumatic reticuloperitonitis or other causes of peritonitis). In abomasal displacement, temperature, heart rate, and respiratory rate are usually normal. The caudal part of the rib cage on the side of the displacement may appear "sprung." Hydration appears subjectively normal with LDA except in some chronic cases. Rumen motility may be normal but often is reduced in frequency and strength of contraction. Feces are usually reduced in quantity and more fluid than normal; however, they may be shed with normal consistency.

The most important diagnostic physical finding is a ping on simultaneous auscultation and percussion of the abdomen, which should be performed in the area marked by a line from the tuber coxae to the point of the elbow, and from the elbow toward the stifle on both sides of the patient. The ping (detected during simultaneous percussion and auscultation) characteristic of an LDA is most commonly located in an area between ribs 9 and 13 in the middle to upper third of the left abdomen; however, the ping can be more ventral or more caudal, or both.

Diagnosis

For abomasal displacement or volvulus, diagnosis is based on the presence of the characteristic ping on simultaneous auscultation and percussion and by exclusion of other causes of left- or right-side pings.

The characteristic rectal examination findings with LDA include a medially displaced rumen and left kidney. The abomasum is rarely palpable in LDA and only occasionally palpable in RDA. Recent parturition, partial anorexia, and decreased milk production suggest displacement.

Treatment

Both conservative (medical) and surgical therapy options are available for patients with LDA. A patient with LDA can be medically treated with administration of spasmolytic and analgesic drugs and corrected surgically.

Prevention

Maintaining optimal dry cow and calving management, avoiding rapid dietary changes, maintaining adequate roughage in the diet, avoiding post parturient hypocalcemia, avoiding endometritis/metritis, and minimizing and promptly treating concurrent disease and ketosis contribute to effective prophylaxis of LDA and RDA. The incidence of abomasal displacements can be decreased by ensuring a rapid increase in rumen volume after calving and by feeding a total mixed ration rather than feeding grain twice daily.

4.3. Aetioloy, Cause, clinical sing, diagnosis, treatment control of diabetics

Diabetes in animals is a metabolic disorder that results from an absolute or relative insulin deficiency, leading to hyperglycemia (high blood sugar). Insulin is a hormone produced by the pancreas and its role is to regulate blood glucose levels. When the body does not produce enough insulin or becomes resistant to insulin, it cannot use glucose properly, leading to an elevated concentration of glucose in the bloodstream. The condition is most commonly seen in middle-aged dogs and older cats.

- **Diabetes Mellitus**

Diabetes Mellitus is chronic disorder related to glucose metabolism due to insulin insufficiency. Old age group and obese animals frequently suffer from this problem.

Etiology:

- ✓ Primary diabetes: Is characterized by destruction of the beta cells and eventual complete loss of insulin secretion. These animals are hypoinsulinemic, show little insulin response to glucose administration, and require treatment with exogenous insulin. “Juvenile diabetes”
- ✓ Secondary diabetes: secondary diabetes is caused by other diseases or drugs that affect insulin production or action. In dogs, secondary diabetes is often associated with hyperadrenocorticism (Cushing’s disease), pancreatitis, and administration of glucocorticoids or progestins.

Clinical sign

Common clinical signs include:

- Polyuria

- Polydipsia
- Polyphagia
- weight loss
- cataracts (dog)
- weakness

Diagnosis: Diabetes may be suspected based on the signs a pet is showing, but the diagnosis is confirmed by finding consistent hyperglycemia and glucosuria.

Treatment: is not always possible as prolonged therapy is required. Diet-low carbohydrate diet should be provided; Insulin therapy once daily to control hyperglycemia following meals.

Control

Controlling diabetes in animals typically involves careful management of diet, exercise, and medication. Regular veterinary monitoring and testing of blood glucose levels may also be necessary to ensure that the treatment plan is effective.

- **Diabetes Insipidus**

Etiology

Diabetes insipidus is caused by a lack of antidiuretic hormone (ADH) or an inability of the kidneys to respond to ADH.

Clinical sign

Affected animals excrete large volumes of hypotonic urine and drink equally large amounts of water.

Diagnosis:

Diagnosis of diabetes insipidus is based on chronic polyuria that does not respond to dehydration and is not due to primary renal disease.

Based on laboratory test

Treatment: ADH should be given as needed to control the polyuria.

4.4.Aetiology, clinical sign, diagnosis, treatments and prevention of allergies

- **Flea allergy dermatitis**

Definition: it is a pruritic skin disease that arises as a result of hypersensitivity to the bite of fleas.

Etiology: when fleas bite, their mouthparts release a small amount of saliva, which contains anticoagulants (thus facilitating taking of the blood meal), and pruritogenic enzymes.

Clinical findings:

Pruritis, partial alopecia, and a popular eruption.

The skin lesion is aggravated by licking and scratching (self-trauma). Secondary bacterial infection with pyoderma usually occurs.



Figure 4.1 Flea Allergy Dermatitis

Diagnosis: history, physical findings, and fleas.

Treatment: eradication of fleas along with other appropriate measures to control secondary infection. Corticosteroid creams to relieve symptomatic skin lesions.

Prevention

Preventing flea allergies in animals involves eliminating fleas and their eggs through regular flea control measures such as flea baths, flea collars, and flea medication. Regular grooming and cleaning of bedding and living areas can also help reduce the risk of flea infestations. Here are some sources on the prevention of flea allergies in animals:

4.5. Aetiology, clinical sign, diagnosis, treatments and prevention of physical trauma

Physical trauma in animals refers to any kind of physical injury that an animal sustains due to an accident, attack, or other harmful event. Physical traumas may affect the animal's body and its physical systems, causing injuries to bones, muscles, organs, or tissues. For example, physical trauma can include injuries like fractures, sprains, burns, and lacerations.

a) Fractures

Fracture in animals is a break in the continuity of a bone.

Aetiology

Bone fractures are often caused by car accidents, firearms, fights, or falls.

Clinical sign

Signs of fracture usually include lameness, pain, and swelling. X-rays are useful in determining the type and extent of the fracture.

Diagnosis

The diagnosis of fractures in animals typically involves a physical examination by a veterinarian followed by imaging tests such as X-rays or ultrasounds.

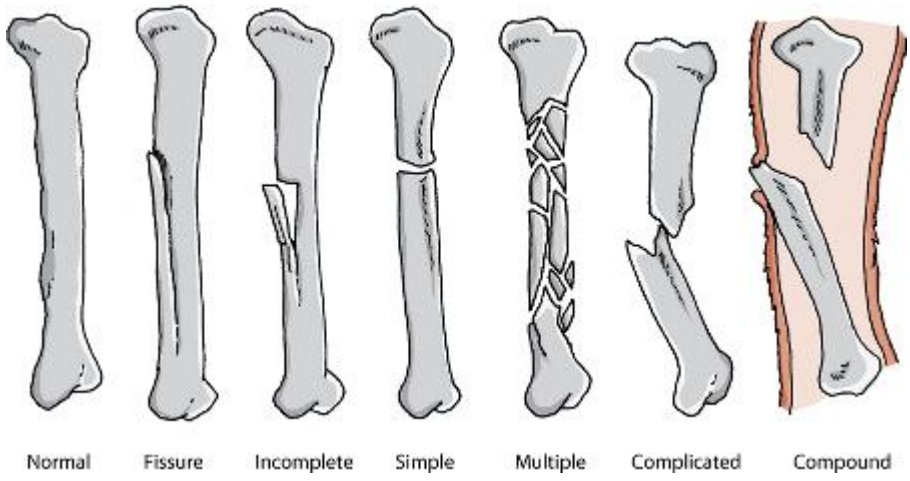


Figure 4.2. Forms of fracture

Treatment

The treatment of a fracture in an animal depends on the severity of the injury and the animal's age, weight, and overall health. For minor fractures, the primary treatment may be rest and

immobilization of the affected limb using a splint or cast. In more serious cases, surgical intervention may be required to align the fractured bone and secure it in place with pins, screws, or plates. Pain management and physical therapy may also be prescribed to aid in healing and recovery.

Prevention

There are several preventative measures that can be taken to minimize the risk of fractures in animals:

Proper nutrition: Feeding animals a well-balanced diet rich in calcium and other essential nutrients can help maintain strong and healthy bones.

Exercise and activity: Regular exercise and mental stimulation can help strengthen an animal's muscles, bones, and joints, reducing the risk of fractures.

Safe housing and living conditions: Providing animals with a safe and secure living environment can help prevent falls, accidents, and other injuries that can result in fractures.

Regular check-ups: Routine veterinary check-ups can help detect any underlying medical conditions that may increase an animal's risk of developing fractures, such as osteoporosis or bone cancer.

Supervision: Close supervision of animals during playtime or outdoor activities can help prevent them from engaging in potentially dangerous behavior that could lead to injury.

It is important to note that prevention is always better than treatment, and that it's always best to consult with a veterinarian on preventative measures specific to your animal's needs.

b) Burn

Aetiology

Burns in animals can be caused by a variety of factors such as exposure to flames, hot objects or liquids, electrical currents, and extreme cold. It is important to identify the cause of the burn to ensure proper treatment.

Clinical signs:

The clinical signs of burns in animals can vary depending on the severity and extent of the burn. Symptoms may include redness, swelling, blisters, pain, hair loss, and skin discoloration. In severe cases, shock or collapse may also occur.

Diagnosis:

Diagnosis of burns in animals can be based on clinical signs and history of exposure. However, in some cases, additional tests such as blood work or radiographs might be necessary.

Treatment:

Treatment of burns in animals depends on the severity and location of the burn. Basic first aid should be administered immediately by flushing the affected area with cool water or saline solution if possible. For more serious burns, veterinary treatment may include medications for pain and inflammation, wound care such as dressing changes or debridement, and possibly surgery.

Prevention

Preventative measures can include keeping hot objects and liquids out of reach of animals, providing adequate shelter and protection from extreme weather conditions, ensuring electrical wiring is safely installed, and monitoring animals during outdoor activities.

c) Sprains

Aetiology:

Sprains in animals occur when a ligament is stretched or torn, typically due to overexertion or trauma. Common causes of sprains in animals include running or jumping excessively, tripping or falling, and sudden stops or turns.

Clinical signs:

The clinical signs of a sprain can depend on the severity of the injury. Signs can include limping, reluctance to bear weight on the affected limb, swelling, pain, and difficulty walking or standing.

Diagnosis:

Physical examination is often the first step in diagnosing a sprain in an animal. Radiographs (X-rays) or other diagnostic imaging may also be used to rule out other injuries and determine the extent of the sprain.

Treatment:

Treatment for a sprain in an animal may involve rest, pain management, and physical therapy. In more severe cases, surgery may be necessary to repair torn ligaments.

Prevention:

Preventative measures can include monitoring animals during physical activity and ensuring proper conditioning through exercise and training. Additionally, keeping areas free of hazards and providing safe footing can help prevent accidents that could lead to sprains.

d) Lacerations

Lacerations in animals can be caused by a variety of factors including accidents, fights with other animals, and sharp objects.

Aetiology

The etiology of lacerations is the specific cause or factors behind the injury.

Clinical signs

The clinical signs of lacerations in animals can include bleeding, pain, swelling, and visible cuts or wounds.

Diagnosis

The diagnosis of lacerations in animals typically involves a physical examination, including checking for any underlying damage to muscles, tendons, or bones.

Treatment

Treatment for lacerations can vary depending on the severity of the injury, but may include cleaning the wound, suturing or stapling the wound shut, using antibiotics to prevent infection, and administering pain medication.

Prevention

Preventative measures for lacerations in animals can include keeping animals in safe environments and away from sharp objects, as well as training animals to avoid dangerous situations. Providing regular veterinary care can also help ensure that any underlying health conditions are identified early and treated appropriately.

4.6. Aetiology,clinical sign, diagnosis treatments and prevention of Traumatic reticulo pericarditis (TRP)

Definition

Traumatic reticuloperitonitis (TRP), also called hardware disease, is a common disease of cattle but is rarely seen in small ruminants. It is the most common cause of anterior abdominal pain in cattle.

Etiology

The ingestive behavior of cattle predisposes them to the accidental swallowing of metal foreign objects that settle in the reticulum. Ingestion of a foreign body may also be associated with diseases that cause pica, such as phosphorus deficiency.

Subsequently, the foreign object may enter the reticulum and attach to a magnet and prevent any clinical disease; penetrate the reticulum wall only with intramural inflammation; perforate the reticulum wall, penetrate into the peritoneal cavity, and create a localized peritonitis; or Migrate into the peritoneal and thoracic cavities.

Clinical Signs

TRP in the most severe, acute form is characterized by fever, anorexia, decreased or absent ruminal contractions, and evidence of cranial abdominal pain.

Pinching of the withers or upward pressure on the xiphoid region may elicit a grunt on expiration. Affected cattle may stand with an arched back and resist ventral flexion of the back when pinched over the withers (normal cattle flex ventrally). Tachycardia, reluctance to move or lie down, mild bloat, constipation, or abducted elbows may also be seen.

Fever may be absent. Weight loss, rough hair coat, diarrhea, or generalized lameness, along with cranial abdominal pain that is difficult to localize, may be the only signs.

Diagnosis

Clinical findings provide a preliminary diagnosis, but may be present in several pathologies, requiring additional tests to confirm the disease. Foreign body tests such as withers clamping, painful percussion on the reticulum with a rubber hammer and pole test are important when foreign bodies are suspected of being ingested, being more common in cattle when compared to buffaloes. Eventually, characteristic lesions of traumatic reticulopericarditis observed in on postmortem examination supply the diagnosis.



Figure 4.3 A Holstein Friesian cow with an arched back, a clinical sign associated with traumatic reticuloperitonitis.

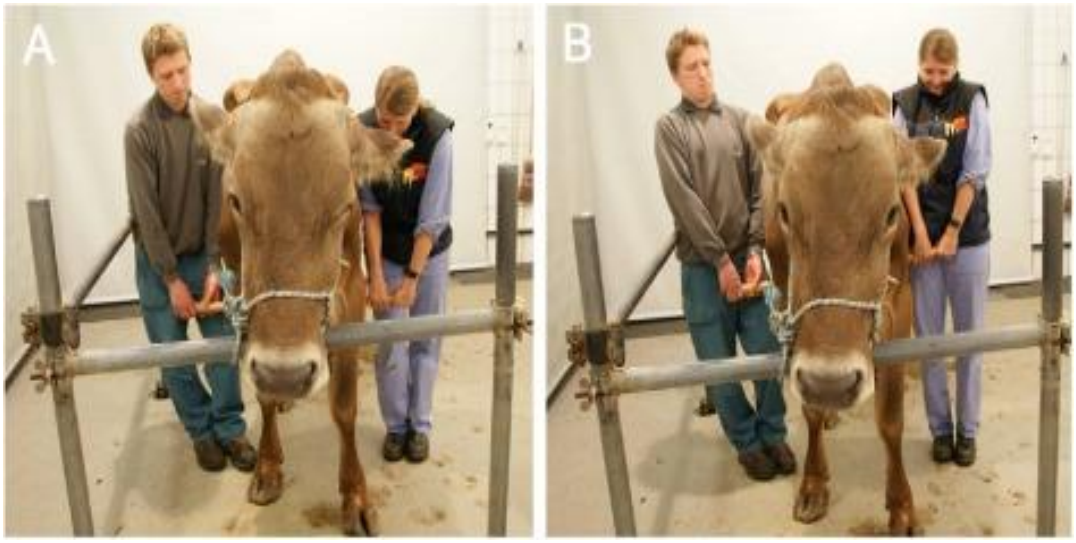


Figure 4.4 pole test



Figure 4.5 The veterinarian in this photograph is using a rubber hammer to elicit a pain response by means of percussion in the region of the reticulum in a Holstein Friesian cow suspected of having traumatic reticuloperitonitis.

Treatment

Treatment for the disease is generally ineffective and used for the animals' short-term survival.

- Conservative therapeutic
- Manage using diuretics to reduce oedema, venous return and cardiac preload.
- Broad-spectrum systemic antibiotics and drainage of the pericardial sac can be used, but are rarely effective.
- The administrations of non-steroidal anti-inflammatory drugs
- Rumenotomy if medical (conservative) treatment fails

Prevention

Measures to prevent traumatic reticuloperitonitis include avoiding the use of baling wire, keeping cattle away from sites of new construction, and completely removing old buildings and fences

4.7.Aetiology clinical sign, diagnosis, treatments and prevention of neoplasia

Neoplasia is the uncontrolled, abnormal growth of cells or tissues in the body, and the abnormal growth itself is called a neoplasm or tumor. It can be benign or malignant. Benign neoplasms tend to grow slowly; displace, but do not tend to invade, the surrounding body tissues; and do not spread throughout the body. Neoplasia is a complex condition that can have a significant impact on an animal's health. In order to effectively diagnose and treat neoplasia, it's important to understand the underlying causes, clinical signs, and treatment options available.

Etiology:

The etiology of neoplasia in animals is complex and multifactorial. While there are many known risk factors for this condition (including exposure to certain environmental toxins or carcinogens), the exact underlying cause of neoplasia is not fully understood. Some potential contributing factors may include genetic predisposition, chronic inflammation or infection, hormonal imbalances, or immune system dysfunction.

Clinical Signs:

The clinical signs of neoplasia can vary depending on the location and severity of the growth. Some common signs include swelling or lumps, changes in appetite or digestion, weight loss, difficulty breathing or swallowing, lethargy, and changes in behavior or personality. In some cases, animals may not show any specific symptoms until the growth has become advanced. It is important to note that clinical signs of neoplasia may mimic other health conditions, so a thorough veterinary evaluation is necessary to make an accurate diagnosis.



Figure 4.4 Common Skin Tumors in Dogs

Diagnosis:

Diagnosis of neoplasia typically involves a combination of physical examination, imaging tests (such as X-rays or ultrasounds), and biopsy or other laboratory testing to confirm the presence of abnormal cells. Blood tests may also be performed to detect any abnormal levels of specific substances that may indicate the presence of neoplasia.

Treatment:

Treatment options for neoplasia depend on the type and location of the growth, as well as the animal's overall health status. Common treatments for neoplasia include surgical removal of the growth, chemotherapy or radiation therapy to slow or stop the growth of cancer cells, and supportive care (such as pain management or nutritional support) to improve the animal's quality of life. In some cases, a combination of these approaches may be necessary to effectively manage the condition.

Prevention:

Preventative measures for neoplasia in animals focus on minimizing exposure to known environmental toxins or carcinogens, as well as promoting overall health and wellness. This may include regular vaccination, proper nutrition, exercise, and routine veterinary check-ups to ensure any underlying health conditions are identified and treated early.

Self-Check –4	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below.

Part I. Write true if the statement is correct and write false if the statement is incorrect (2pts. each)

1. TRP is pruritic skin disease that arises as a result of hypersensitivity to the bite of fleas.
2. Fracture is a break in the continuity of a bone

Part II Choose the best Answer (3pts each)t

1. Which one of the following disease condition characterized by uncontrolled, abnormal growth of cells or tissues in the body, and the abnormal growth itself?
   A. Fracture C. Neoplasia
   B. Bloat D. Allergy
2. Which one of the following is technique is used for diagnosis TRP?
   A. Withers clamping, C. Pole test
   B. Pain percussion on the reticulum with a rubber hammers D. All

Test III. GIVE SHORT ANSWER (5 pts. each

1. Discuss aetiology, clinical sign, diagnosis, treatments and prevention of physical trauma.
2. Discuss aetioloy, Cause, symptoms, diagnosis and treatment of diabetics.
3. Discuss aetiology clinical sign, diagnosis, treatments and prevention of neoplasia.

Note: **Satisfactory rating – 25 points Unsatisfactory - below 25points**

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AKNOWLEDGEMENT

Ministry of Labor and Skills wish to extend thanks and appreciation to the many representatives of TVET instructors and respective industry experts who donated their time and expertise to the development of this Teaching, Training and Learning Materials (TTLM).

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