TVET CERTIFICATE IV in ANIMAL HEALTH

ANHNM401

INTOXICATIONS, NUTRITIONAL AND METABOLIC DISEASES TREATMENT

Treat intoxications, nutritional and metabolic disease

Competence

Credits: 9

Learning hours: 90

Sector: Agriculture and Food processing

Sub-sector: Animal Health

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Purpose statement

This core module describes the skills, knowledge and attitude required by the learner to treat intoxications, nutritional and metabolic diseases. Any veterinary has to treat intoxications, nutritional and metabolic diseases during his professional work. So, this competence is very important for the Veterinary assistant training. Upon completion of this module, the trainee will be able to:

- ✓ Describe intoxications , nutritional and metabolic diseases
- ✓ Perform clinical diagnosis of intoxications, nutritional and metabolic diseases
- ✓ Apply treatment of intoxications, nutritional and metabolic diseases

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Learning Unit 1 – Describe intoxications and toxi-infections

LO 1.1 – Identify intoxications and toxi-infections

- Content/Topic 1:Identification of intoxications and toxi-infections

A. Definition of some concepts

Veterinary toxicology involves the evaluation of toxicosis and deficiencies, identification and characterization of toxins and determination of their rate in the body, and treatment of toxicosis. Veterinary toxicology can be challenging because of the low frequency of cases observed in a practice setting. When a toxicosis occurs, it often involves a large number of animals and may also involve litigation.

- **Intoxications**

Food intoxication is a form of food-borne illness caused by ingesting exotoxins made by organisms such as bacteria.

- **Toxigenesis**

Toxigenesis or the ability to produce toxins, is an underlying mechanism by which many bacterial pathogens produce disease.

- **Toxicosis**

Toxicosis is the term used to describe the syndrome of adverse health effects that result from exposure to a toxicant.

- **Poison**

Poison is any substance that can impair function, cause structural damage, or otherwise injure the body.

- **Lethal Dose_50 (LD50)**

This is the dose of a toxic substance that kills 50% of the population at risk.

- **Antidote**

Remedy or other agent used to neutralize or counteract the effects of a poison.

- **Endotoxins**

Endotoxins are cell-associated substances that are structural components of bacteria. Most endotoxins are located in the cell envelope.

- **Exotoxins**

Exotoxins are usually secreted by bacteria and act at a site removed from bacterial growth. However, in some cases, exotoxins are only released by lysis of the bacterial cell

- Content/Topic 2: Types and source of intoxication

1. Types and sources of intoxication

Types and sources of intoxications vary greatly in the environment. All toxic effects are dose dependent. A dose may cause undetectable, therapeutic, toxic, or lethal effects. A dose is expressed as the amount of

compound per unit of body weight, and toxicant concentration as part per million or part per billion. These quantitative expressions are also used for feedstuffs, water, and air as well as tissue levels.

A. **Intoxications by metals**

- Aluminium, Arsenic ,Cadmium, Copper, Fluoride, Iron, Lead
- Mercury, Molybdenum, Selenium, Sodium chloride, Sulphur,
- Zinc, Chromium, iodine and phosphorus

B. **Intoxications by pesticides**

- herbicides, Fungicides, molluscicides, insecticides, Rodenticides, acaricides

C. **Intoxications by environmental pollutants**

- Human activities (Industrial Toxicants, livestock)

D. **Intoxications by drugs use and abuse**

- Amphetamines and related drugs, Marijuana

E. **Intoxications by poisonous plants**

- beet, fern, lantana, sorghum, cottonseed, datura, solanacea, cassava,
- clover, oxalates containing plants, nitrate and nitrite containing plants

F. **Intoxications by Mycotoxicoses**

- Aflatoxines, clavacine

G. **Intoxications by poisonous and venomous organisms**

- snakes, bees, wasp

H. **Bacterial Toxins**

- *Staphylococcus aureus*, *Clostridium botulinum*

I. **Feed and Water Contaminants**

- Chemicals accumulated in water become contaminants

LO 1.2 – Identify nutritional and metabolic diseases

- Content/Topic 1:Identification of nutritional and metabolic diseases

A. Nutritional disorders due to energetic and proteins deficiency or excess

- Growth retardation, Cachexia and Lactic indigestions

B. Metabolic diseases

- Acetonemia, Rumen acidose, and Alkalosis

C. Mineral nutritional disorders

- Milk fiver , (hypocalcemia), Grass tetany (hypomagnesemia), Rachitism, Urolithiasis,
- Osteomalacia, Osteofibrose, Hyperkeratose, Parakeratose and Goiter

D. Vitamins nutritional disorders

- Hypovitaminose and Hypervitaminoses

E. Drinking and feeding errors

- Dehydration and Water hematuria

F. Digestives disorders

- Constipation, Diarrhea, Indigestions, Bloating, Dehydration,
- Alcalose, Acidose, Enterotoxemia and Vomiting

LO 1.3 – Characterize Toxicoses and toxi-infections

- Content/Topic 1: Intoxications by metals

Lead and **cadmium** are ubiquitous heavy metals and have both been associated with testicular toxicity and impaired fertility in a number of species. Excessive **cobalt** can potentially interfere with normal spermatogenesis, and severe cobalt intoxications have actually resulted in generalized hypoxia related to increased blood viscosity which affects the testes. **Chromium** and **vanadium** have also been associated with adverse reproductive effects. The following minerals will be discussed for their toxic effects: **Lead, Mercury, Zinc, Selenium, Cadmium, Sodium Chloride and Arsenic.**

A. Lead Poisoning

Lead poisoning in animals and people is a major concern worldwide. 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.

A.1. Clinical signs

- **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 hour 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**, occasionally seen in cattle, may produce a syndrome that has many features in common with acute or sub-acute lead poisoning. Impairment of the swallowing reflexes frequently contributes to the development of aspiration pneumonia.

A.2. Treatment

If tissue damage is extensive, particularly to the nervous system, treatment may not be successful. In livestock, calcium disodium edetate (Ca-EDTA) is given IV or SC (110 mg/kg/day) divided bid for 3 days; this

treatment should be repeated 2 days later. In dogs, a similar dose divided is administered SC in 5% dextrose for 2–5 days. After a 1-week rest period, an additional 5-day treatment may be required if clinical signs persist. No approved veterinary product containing Ca-EDTA is currently commercially available.

A.3. Prevention and control

Lead poisoning of cattle can be avoided if a farmer practises good waste management on the farm. Prevention is easier, cheaper and more effective than treatment by a veterinarian. The following practices greatly reduce the risk of lead poisoning.

To prevent lead poisoning or lead residues in farm animals, take the following steps:

- Identify all the potential sources of lead on your farm. For example, if you have painted feeders, test the paint for lead content.
- Develop a plan of how to prevent farm animals from accessing the lead sources identified. In the case of a lead-painted shearing shed, for example, an unpainted barrier could be placed around the shed to above stock head height. In the case of the farm rubbish dump, securely fencing it will minimize the risk.
- Carry out the plan.
- Document the plan and the actions you have taken in order to satisfy food safety and quality assurance program requirements.

B. **Mercury Poisoning**

Historically, mercury poisoning was a common occurrence in both human and animal populations. The replacement of mercury products used for medicinal, agricultural, or industrial purposes has resulted in a decline in acute and chronic poisoning, although many wildlife species remain at risk.

Mercury exists in a variety of chemical forms, including elemental mercury (e.g., thermometers, light bulbs), inorganic mercurial (mercuric or mercurous) salts (e.g., batteries, latex paints), and organic mercury (aryl, methyl, or ethyl). Fossil fuels represent an important environmental source of mercury. In the environment, inorganic forms of mercury are converted to methyl mercury under anaerobic conditions in the sediment of most water bodies. Similar conversions may also occur in the body.

B.1. Clinical Findings

The inhalation of corrosive elemental mercury vapors that produces severe dyspnea and compromised respiratory function is usually fatal at high levels of exposure. Neurologic manifestations may eventually develop if exposure is not excessive. Inorganic mercury, related to its corrosive nature, produces primarily GI manifestations, including colic, anorexia, stomatitis, pharyngitis, vomiting, diarrhea, shock, dyspnea, and dehydration. Death often occurs within hours at high levels of exposure.

B.2. Treatment and Control

Because the neurologic and renal damage is irreversible, treatment alternatives may be ineffective. Consequently, the prognosis for a complete recovery is very poor. In food-producing animals, significant mercury accumulation in edible tissues and profound effects on reproduction limit treatment options.

Oral administration of activated charcoal (1–3 g/kg) and sodium thiosulfate (0.5–1 g/kg) will bind mercury and limit absorption. Vitamin E and selenium, which are antioxidants, may limit oxidative damage. If the GI tract has been decontaminated for mercury, administration of Penicillamine (50–100 mg/kg/day, PO, for 2 week) may reduce clinical signs.

C. Zinc toxicosis

Zinc is an essential trace metal that plays an important role in many of the body's enzymatic processes. It is ubiquitous in nature and exists in many forms. The ingestion of some forms of zinc causes the creation of toxic zinc salts in the acidic environment of the stomach.

C.1. Clinical Findings

Clinical signs vary based on the duration and degree of exposure. Signs progress from anorexia, vomiting, diarrhea, and lethargy to more advanced signs such as intravascular haemolysis, icterus, hemoglobinuria, cardiac arrhythmias, and seizures. Large animals often show decreases in weight gain and milk production, and lameness has been reported in foals secondary to epiphyseal swelling.

C.2. Treatment and Prevention

After stabilizing the animal with fluids, oxygen, and blood products as necessary, removal of the source OF zinc as early as possible is paramount. This often requires surgery or endoscopy. Inducing emesis to remove chronic gastric zinc foreign bodies may be tried within the first hour or two of exposure but may not be rewarding in advanced cases because zinc objects can adhere to the gastric mucosa.

With early diagnosis and treatment, the outcome is usually favourable for animals with zinc toxicosis. Of course, eliminating exposure to zinc in the environment is essential to prevent recurrence.

D. Selenium Toxicosis

Selenium is an essential element that has a narrow margin of safety, with the difference between adequate and potentially toxic concentrations in the diet being approximately 10 to 20 fold. Feed supplements, resulting in final selenium content of 0.2–0.3 ppm, are added to diets to prevent deficiency and resultant diseases such as white muscle disease in cattle and sheep, exertional myopathy in horses, hepatosis dietetica in pigs, and exudative diathesis in chickens. The maximum tolerable concentrations for selenium in most livestock feed is considered to be 2–5 ppm, although some believe 4–5 ppm can inhibit growth.

D.1. Etiology

All animal species are susceptible to selenium toxicosis. However, poisoning is more common in forage-eating animals such as cattle, sheep, horses, and other herbivores that may graze selenium-containing

plants. Plants may accumulate selenium when the element is found at high concentrations in the soil, but pH and moisture content of the soil play roles in the relative bioavailability of selenium to plants.

Poisoning may also occur in swine, poultry, and other species consuming grain raised on seleniferous soils or, more commonly, due to errors in feed formulation. Selenium toxicosis after ingestion of selenium-containing shampoos or selenium supplement tablets is rare in small animal pets but can occur. Several factors are known to alter selenium toxicity; however, in general, a single acute oral dose of selenium in the range of 1–10 mg/kg may be lethal in most animals. Parenteral selenium products are also quite toxic, especially to young animals, and have caused deaths in piglets, calves, lambs, and dogs at dosages as low as 1 mg/kg.

D.2. Diagnosis

Severity of clinical signs of selenium toxicosis depends on the quantity and duration of exposure. Poisoning in animals is characterized as acute, sub chronic, or chronic. Diagnosis is based on clinical signs, necropsy findings, and laboratory confirmation of the presence of high selenium content in an animal's diet (feed, forage, grains, or water), serum, blood, or tissues (e.g., kidney, liver). Environmental exposure potential should be based on forage, feed, or water content, not on soil selenium content, because some chemical forms in soil are not available for uptake by plants and would not result in high exposure potential.

D.3. Treatment and Control

There is no specific treatment for selenium toxicosis. Eliminating the source and exposure, as well as symptomatic and supportive care of the animal, should be started as soon as possible. Addition of substances that antagonize or inhibit the toxic effects of selenium in the diet may help reduce the risk of selenium toxicosis. A high-protein diet, linseed oil meal, sulphur, arsenic, silver, copper, cadmium, and mercury have reduced selenium toxicity in laboratory animals, but their use under field conditions is limited. However, some of the poor reproductive performance associated with selenium poisoning can be decreased by copper supplementation. Addition of arsenic salt at 0.00375% to enhance biliary excretion of selenium or a high-protein diet to bind free selenium has historically been used to reduce incidence of selenium poisoning in cattle. However, this has minimal to poor overall efficacy. Chronically selenium-poisoned animals are less likely to thrive than herd mates, even after exposure has been stopped. Forages should be tested regularly in high-selenium areas to evaluate year-to-year risk.

E. Cadmium Poisoning

E.1. Introduction

Cadmium (Cd) has no known biological function in either animals or humans but mimics the actions of other divalent metals that are essential to diverse biological functions. Bioavailability, retention and consequently toxicity of Cd are affected by several factors such as nutritional status low body iron (Fe) stores) and multiple pregnancies, pre-existing health conditions or diseases.

Sources

Most of the Cd is produced as a by-product during the production of other metals, such as copper, lead and zinc. The major source of Cd exposure to animals can be from industrial pollution and environmental contamination, especially water.

E.2. Pathogenesis and clinical signs

Cadmium (Cd) works by disrupting the normal state of a cell. Its similar structure to zinc and calcium allows it to interfere with the absorption of these elements, and it similarly inhibits copper and iron. Like most heavy metals, when absorbed into the body cadmium is stored in the liver and kidney of the animal. Cadmium is an accumulative poison, meaning that it builds up slowly over time in the bodies of exposed animals. Cadmium poisoning affects nearly all major organ systems and has noted reproductive effects, including decreased testes size and infertility. Additionally, because of this element's interference with the absorption of the mineral calcium, cadmium poisoning can also severely impact the healthy function of the bones.

F. Sodium chloride

F.1. Introduction

Excessive salt (sodium chloride, NaCl) intake can lead to the condition known as salt poisoning, salt toxicity, hypernatremia, or water deprivation-sodium ion intoxication. The last title is the most descriptive, giving the result (sodium ion intoxication) as well as the most common predisposing factor (water deprivation.) Salt poisoning is unlikely to occur as long as sodium-regulating mechanisms are intact and fresh drinking water is available.

F.2.Etiology

Salt poisoning has been reported in virtually all species of animals all over the world. Although salt poisoning has historically been more common in swine (the most sensitive species), cattle, and poultry, there are increasing reports of adverse effects in dogs from acute excess salt consumption. The acute oral lethal dose of salt in swine, horses, and cattle is ~2.2 g/kg; in dogs, it is ~4 g/kg. Sheep appear to be the most resistant species with an acute oral lethal dose of 6.0 g/kg. Chickens can tolerate up to 0.25% salt in drinking water but are susceptible to salt poisoning when water intake is restricted.

F.3. Clinical Findings

- In pigs, early signs (rarely seen) may be increased thirst, pruritus, and constipation. Affected pigs may be blind, deaf, and oblivious to their surroundings; they will not eat, drink, or respond to external stimuli.
- In cattle, signs of acute salt poisoning involve the GI tract and CNS. Salivation, increased thirst, vomiting (regurgitation), abdominal pain, and diarrhea are followed by ataxia, circling, blindness, seizures, and partial paralysis.

- In poultry and other birds, clinical signs include increased thirst, dyspnea, fluid discharge from the beak, weakness, diarrhea, and leg paralysis.

F.4. Treatment

There is no specific treatment for salt poisoning. Immediate removal of offending feed or water is imperative. Fresh water must be provided to all animals, initially in small amounts at frequent intervals to avoid exacerbation of clinical signs. On a herd basis with large animals, water intake should be limited to 0.5% of body weight at hourly intervals until normal hydration is accomplished, usually taking several days. Severely affected animals can be given water via stomach tube. For all affected animals, the treatment should slowly return the animal to normal water and electrolyte balance over 2–3 days.

G. Arsenic Poisoning

G.1. Introduction

- Arsenic constitutes one of the most important toxicological hazards to farm animals.
- Toxicity variety with factors such as oxidation state of arsenic, solubility, species of animal and duration of exposure.
- Arsenic poisoning is caused by different types of inorganic and organic arsenical compounds. Arsenical gas (lewistee) used in World War I treated by Dimercaprol (BAL-British Anti Lewistee).

G.2. Sources of poisoning

Poisoning occur due to arsenic trioxide, arsenic pent-oxide, sodium and potassium arsenate. Also occur because of using of arsenic salts as insecticides for plants and fruit tree or from using of it's in sheep baths for mosquito's resistance. Drinking water containing more than 0.25% arsenic is consider potentially toxic especially in large animals. Cats are poisoned because they ingest syrup baits intended for insects.

G.3. Toxicokinetics

Arsenic especially in the form of inorganic salts can be easily absorbed from intestine, site of injection and from wounds. A part of arsenic could be reserved in the liver and kidney. In domestic animals, arsenic does not stay in the soft tissues for long period. It is rapidly excreted in bile, milk, saliva, sweat, urine and faeces. After continuous intake arsenic tends to accumulate in bones.

G.4. Clinical signs

Poisoning due to arsenic is usually acute with major effects on GIT and cardiovascular system. In acute cases, profuse watery diarrhea (rice watery) sometimes tinged with blood is characteristic along with sever colic, dehydration, weakness, depression, weak pulse and cardiovascular collapse. The onset is rapid and signs are usually seen within few hours (up to 24 h). In peracute poisoning animals may simply dead. Chronic cases are rare and are characterized by wasting, poor condition, thirst, brick-red mucous membrane and irregular pulse.

On admission, the affected beef cow was very weak, anorexic, depressed, and dehydrated. Her body temperature, heart and respiratory rate were within normal limits; however her mucous membranes were hyperaemic and dry.

G.5. Treatment

There is no specific treatment, but the neurotoxic effects are usually reversible if the offending feed is withdrawn within 2–3 days of onset of ataxia. Once paralysis occurs, the nerve damage is irreversible. Blindness is usually irreversible, but animals retain their appetite, and weight gain is good if competition for food is eliminated. Recovery may be doubtful when the exposure is long and the onset of intoxication slow.

- **The following points should be considered in case of Arsenic toxicity:**
  - ✓ Making gastric lavage by warm water to remove the poison.
  - ✓ Giving laxatives to remove poison from the intestine before absorption.
  - ✓ In animals with recent exposure and no clinical signs emesis.
  - ✓ Giving oral administration of GI protectants such as kaolin-pectin.

- Content/Topic 2: Intoxications by pesticides

A. Introduction

Livestock and pet animal exposure to pesticides is usually accidental. The most common source of dermal toxicity induced with insecticides in both large and small animals is the miscalculation of concentrations for spraying and dipping procedures. A common source of non-recommended exposure to animals is the inadvertent mixing of powdered pesticides, mistaken for salt or mineral preparations, into animal feeds. A low-level contamination of animals by insecticides can also occur with the use of persistent chlorinated hydrocarbons such as dichlorodiphenyltrichloroethane (DDT).

B. Herbicide Poisoning

Herbicides are used routinely to control noxious plants. Most of these chemicals, particularly the more recently developed synthetic organic herbicides, are quite selective for specific plants and have low toxicity for mammals; other, less-selective compounds (e.g., sodium arsenite, arsenic trioxide, sodium chlorate, ammonium sulfamate, borax, and many others) were formerly used on a large scale and are more toxic to animals. Most health problems in animals result from exposure to excessive quantities of herbicides because of improper or careless use or disposal of containers. When used properly, problems of herbicide poisoning in veterinary practice are rare. With few exceptions, it is only when animals gain direct access to the product that acute poisoning occurs.

Chronic disease caused by herbicides is even more difficult to diagnose. It may include a history of herbicide use in proximity to the animals or animal feed or water source, or a gradual change in the animals' performance or behavior over a period of weeks, months, or even years.

C. Rodenticide Poisoning

Many poisons have been used against rodent pests. If baits are not well secured, they may be ingested directly by non-target animal species (farm animals, pets, and wildlife). Sometimes, no target species may ingest recently poisoned rodent pests and develop relay or secondary poisoning. Occasionally, baits may be used maliciously or intentionally to kill either domestic animals or wildlife.

Whenever a rodenticide exposure is suspected, owners should be asked history questions to determine the day and time of exposure, brand name and manufacturer of rodenticide, active ingredients and their concentration, package size, and the potential amount missing.

Anticoagulant rodenticides inhibit the enzyme vitamin K epoxide reductase, which normally reactivates vitamin K, a crucial component in a number of normal clotting factors, after those factors are consumed in normal maintenance. Potentially dangerous to all mammals and birds, anticoagulant rodenticides are a common cause of poisoning in pets and wildlife. Intoxications in domestic animals have resulted from contamination of feed with anticoagulant concentrate, malicious use of these chemicals, and feed mixed in equipment used to prepare rodent bait.

- Content/Topic 3: Intoxications by insecticides and environmental pollution

A. Introduction

Depending upon the duration, frequency and level of exposure, the insecticides of various classes (**organophosphates, carbamates, organochlorines and pyrethroids**) can adversely affect one or all three components of the maternal/placental/fetal unit. Numerous studies demonstrate that the insecticide residue is present in the exposed mother, placenta, cord blood, embryo and fetus, suggesting that the placenta and fetus are potentially exposed to these compounds.

A.1 Intoxications by environmental pollutants

Environmental pollutants of great importance in the 21st century include industrial chemicals, combustion products, pesticides, flame retardants, salts, acids, nutrients, and heavy metals. Combined with aggressive and careless actions that cause habitat loss, overharvest of native species, introductions of exotic species, climate change, and increasing exposures to pathogens, contaminants are contributing to an accelerating sixth extinction. Problems related to environmental contamination persist in much of the developed world, and they are burgeoning in areas of the developing world where mineral extraction, industrialization, and urbanization are proceeding at unprecedented rates and environmental monitoring and regulation are weak and often corrupt.

Having a contaminant in the environment does not mean that all nearby organisms will have contact with it. For example, surface-dwelling animals may not have direct access to a contaminant buried under many centimetres of topsoil. However, certain organisms may be exposed by consuming earthworms or plants that bring the contaminants to the surface. In ecotoxicology, receptor generally refers to intact organisms. When pollutants and receptors co-occur, pathways of exposure, including dermal, ocular, oral (via water, diet, and grooming), inhalation, and/or gills, may be important.

- Content/Topic 4: Intoxications by drugs use and abuse

A. Introduction

Exposures to illicit or abused drugs in pet animals can be accidental, intentional, or malicious. Occasionally, drug-sniffing dogs also ingest these substances. Because of the illegal nature of illicit or abused drugs, owners may provide inaccurate, incomplete, or misleading exposure histories. Illicit drugs are often adulterated with other pharmacologically active substances, making the diagnosis even more difficult. In suspected cases of exposure to illicit or abused drugs, an attempt should be made to gather information about the animal's environment; amount of exposure; and time of onset of clinical signs and their type, severity, and duration.

A.1. Amphetamines and related drugs

Amphetamines and their derivatives are CNS and cardiovascular system stimulants commonly used in people for suppression of appetite, narcolepsy, attention deficit disorder, Parkinsonism, and some behaviour disorders. Amphetamines are rapidly absorbed in the GI tract, reaching peak plasma concentrations in 1–2 hr.

- **Clinical signs** of amphetamine and cocaine toxicosis are similar and difficult to differentiate clinically. The only difference may be the longer duration of clinical signs of amphetamine toxicosis because the half-life of amphetamine is longer than that of cocaine. The most commonly reported signs are hyperactivity, aggression, hyperthermia, tremors, ataxia, tachycardia, hypertension, mydriasis, circling, head bobbing, and death.

A.2. Marijuana (*Cannabis*)

Marijuana refers to a mixture of cut, dried, and ground flowers, leaves, and stems of the leafy green hemp plant *Cannabis sativa*. Several cannabinoids are present in the plant resin, but delta-9-tetrahydrocannabinol (THC) is considered the most active and main psychoactive agent.

The most common route of exposure is oral. After ingestion, THC goes through a substantial first-pass effect. It is metabolized by liver microsomal hydroxylation and non-microsomal oxidation. In dogs, clinical signs begin within 30–90 min and can last up to 72 hr. THC is highly lipophilic and readily distributes to the brain and other fatty tissues after absorption. The oral LD_{50} of pure THC is 666 mg/kg in rats and 482 mg/kg in mice. However, clinical effects of marijuana are seen at much lower doses than this.

- **The most common signs of marijuana toxicosis are** :
    - ✓ Depression,
    - ✓ ataxia,
    - ✓ Brady cardia,
    - ✓ hypothermia,
    - ✓ vocalization,
    - ✓ hyper salivation,
    - ✓ vomiting,
    - ✓ diarrhea,
    - ✓ urinary incontinence
    - ✓ , seizures, and coma.
- **Treatment**: It.consists of supportive care. If the exposure is recent and there are no contraindications, emesis should be induced and activated charcoal administered.

A.3. <u>Opiates</u>

- **Introduction**

The term opiate initially referred to all naturally occurring alkaloids obtained from the sap of the opium poppy (*Papaversomniferum*). Opium sap contains morphine, codeine, and several other alkaloids. Currently, opioid refers to all drugs, natural or synthetic, that have morphine-like actions or actions mediated through opioid receptors.

- **Commonly reported clinical signs of toxicosis**

This includes:

- ✓ CNS depression,
- ✓ Drowsiness,
- ✓ Ataxia, vomiting, seizures,
- ✓ Miosis, coma, respiratory depression,
- ✓ Hypotension, constipation/defecation, and death.
- ✓ Some animals—especially cats, horses, cattle, and swine—can show CNS excitation instead of CNS depression.

- **Clinical signs** can be reversed with the opiate antagonist naloxone. The dosages in different species are:
    - ✓ Dog and Cat, 0.04–0.16 mg/kg, IV, IM, or SC;
    - ✓ Rabbit and rodent, 0.01–0.1 mg/kg, SC or IP;
    - ✓ Horse, 0.01–0.02 mg/kg, IV.

- ✓ Administration of naloxone should be repeated as needed (hourly), because its duration of action may be shorter than that of the opioid toxicity being treated.

- Content/Topic 5: Intoxications by poisonous plants

A. Introduction

Livestock are accidentally poisoned from eating toxic plants in hay and pasture forage crops. What is a poisonous plant? It is one that causes such problems as animal sickness, skin irritation, loss of appetite, loss of weight, reduced milk production or death. All poisonous plants do not contain the same toxin. There are at least six different classes of poisons within plants. The two largest groups are alkaloids and glycosides. Within each of the six classes are several different poisonous compounds.

Some plants are likely to be a greater hazard to animal health than others because they

(**1**) Are so abundant in an area,

(**2**) Contain a more deadly poison or

(**3**) Because animals seek them out for selective grazing.

B. Severity of Poisoning

Some of the factors that influence the degree of hazard associated with poisonous plants are as follows:

- **Plant Species** –

All plants absorb nitrates, but plants such as the sorghums, small grains, corn, turnips, rape, kochia, orchard grass, pigweed, lambs quarter and soybeans are more likely than other plants to accumulate nitrates in toxic levels.

- **Plant Parts**

The entire plant (as in the case of Johnson grass) or only certain parts of plants (as in the case of acorns and buds of oak trees) may accumulate poisons to a lethal level.

- **Environment**

Reduced light caused by shade or cloudy weather can encourage nitrate accumulation in plants; droughts may also encourage nitrate accumulation; and frost or freezing weather may release deadly levels of prussic acid.

- **Plant Age**

Poisons that occur in plants such as white snakeroot and Johnson grass are more likely to be hazardous in younger plants.

C. Poisoning control

Livestock losses due to poisonous plants may be reduced or eliminated by weed control, by grazing practices, by keeping time forage stands healthy and thick, by using caution during drought periods and by diluting contaminated feed with forage known to be free of poisonous materials. Fortunately, most (but not all) poisonous plants must be consumed in large quantities to be lethal. Also, many have an

undesirable taste, and animals do not consume them in toxic levels unless they are forced to do so by a shortage of forage that occurs during drought or long winter seasons.

D. Types of poisonous plants found in Rwanda

D.1.Sorghum Poisoning

- Sorghum species are drought-tolerant plants that may produce neuropathic and teratogenic manifestations. The syndrome is reported almost exclusively in horses, although a similar disease has been reported in sheep and cattle.
- Sorghum poisoning is characterized by posterior ataxia or incoordination, cystitis, urinary incontinence (which predisposes both male and female horses to cystitis), and alopecia on the hind legs due to urine scald. The loss of urinary bladder function is related to degeneration of spinal cord neurons. The incoordination may progress to irreversible flaccid paralysis.
- Consumption of sorghum hybrids with low cyanogenic potential or restriction of access to sorghum grasses may limit the incidence. Dietary supplementation with sulphur may be beneficial. Affected horses often die from pyelonephritis. Treatment with antibiotics may be helpful, but a full recovery is rare if ataxia has developed. Consumption of pastures containing sorghum plant species is not recommended for horses

Figure 1: Sorghum plant

D.2.Sweet

Clover Poisoning

Sweet clover poisoning, an insidious haemorrhagic disease, is seen in animals that consume toxic quantities of spoiled sweet clover (*Melilotus officinalis* and *M. alba*) hay or silage.

- **Etiology**

During the process of spoiling, the coumarins in sweet clover are converted to toxic dicumarol, a potent vitamin K antagonist and anticoagulant. Any method of hay storage that allows molding of sweet clover promotes the likelihood of formation of dicumarol in the hay. When toxic hay or silage is consumed for several weeks, dicumarol alters pro enzymes required for synthesis of prothrombin, resulting in hypo prothrombinemia (by preventing formation of the active enzyme). Dicumarol concentrations of 20–30 mg/kg of hay ingested throughout several weeks are usually required to cause poisoning in cattle. The toxic agent crosses the placenta in pregnant animals, and new born animals may be affected at birth. All species of animals studied are susceptible, but instances of poisoning involve cattle and, to a limited extent, sheep, pigs, and horses.

- **Clinical signs**

Clinical signs are preferable to haemorrhages that result from faulty blood coagulation. The first indication of dicumarol poisoning may be the death of one or more animals. In affected animals, the first signs may be stiffness and lameness, due to bleeding into the muscles and joints. Hematomas, epistaxis, or GI bleeding may also be seen. Death is generally caused by massive haemorrhage or bleeding after injury, surgery, or parturition. Neonatal deaths rarely occur without signs in the dam.

- **Treatment**

The hypo prothrombinemia haemorrhages, and anaemia can be immediately corrected, to a degree, by IV administration of whole blood. This may be difficult in large animals, because effective dosages range from 2 to 10 L of fresh blood per 1,000 lb. (450 kg) body weight. In addition, all severely affected animals should receive parenteral administration of synthetic vitamin K1 (phytonadione). SC or IM injection is recommended to avoid the substantial risk of anaphylaxis; SC vitamin K1 may not be as effective as IM treatment.

- **Prevention**

Cultivars of sweet clover low in coumarins and safe to feed (e.g., Polara) have been developed. If one of these is not available, the only certain method of prevention is to avoid feeding sweet clover hay or silage. Although well-cured sweet clover is not dangerous, the absence of visible spoilage is insufficient evidence of safety.

Alternating sweet clover hay suspected of containing dicumarol with other roughage such as alfalfa or a grass-legume hay mixture can be used to avoid severe poisoning. From 7- to 10-day period on the sweet clover hay, followed by an equal time on the alternative hay, can prevent poisoning, but it will not

completely prevent prolonged bleeding times. Because some animals have higher risks of serious hemorrhage (surgical candidates or pending parturition), they should not receive sweet clover hay for a minimum of 2–3 week, and preferably ≥4 week, before surgery or parturition.

Figure 2: Sweet clover

D.3. Nitrate and Nitrite Poisoning

Many species are susceptible to nitrate and nitrite poisoning, but cattle are affected most frequently. Ruminants are especially vulnerable because the ruminal flora reduces nitrate to ammonia, with nitrite (~10 times more toxic than nitrate) as an intermediate product. Nitrate reduction (and nitrite production) occurs in the cecum of equids but not to the same extent as in ruminants. Young pigs also have GI micro-flora capable of reducing nitrate to nitrite, but mature monogastric animals (except equids) are more resistant to nitrate toxicosis because this pathway is age-limited.

- **Etiology**

Nitrates and nitrites are used in pickling and curing brines for preserving meats, certain machine oils and anti-rust tablets, gunpowder and explosives, and fertilizers. They may also serve as therapeutic agents for certain non-infectious diseases, e.g., cyanide poisoning. Toxicoses occur in unacclimated domestic animals most commonly from ingestion of plants that contain excess nitrate, especially by hungry animals engorging themselves and taking in an enormous body burden of nitrate.

Nitrate toxicosis can also result from accidental ingestion of fertilizer or other chemicals. Nitrate concentrations may be hazardous in ponds that receive extensive feedlot or fertilizer runoff; these types of nitrate sources may also contaminate shallow, poorly cased wells. Crops that readily concentrate nitrate include cereal grasses (especially oats, millet, and rye), corn (maize), sunflower, and sorghums.

Nitrates and nitrites are closely linked as causes of poisoning. Nitrate is not always toxic to animals. When feed containing nitrate is eaten by ruminant animals, nitrate is converted to nitrite, and then to ammonia, by rumen microbes. Non-ruminant animals are unable to do this.

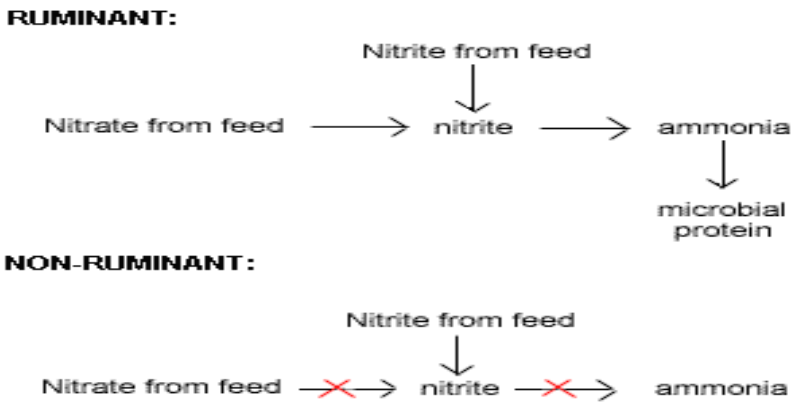

Figure 3: diagram of nitrate poisoning

- **Clinical Findings**

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%.

Signs of nitrate poisoning are:

- ✓ diarrhoea and vomiting;
- ✓ salivation;
- ✓ Abdominal pain.

Signs of nitrite poisoning usually appear 6–24 hours after the toxic material is consumed. These include:

- ✓ rapid, noisy and difficult breathing;
- ✓ blue/chocolate-coloured mucous membranes;
- ✓ rapid pulse;
- ✓ salivation, bloat, tremors, staggering;
- ✓ dark, chocolate-coloured blood;
- ✓ abortions – pregnant females that survive nitrate/nitrite poisoning may abort due to a lack of oxygen to the foetus; abortions usually occur 10–14 days after exposure to nitrates;
- ✓ Weakness, coma, terminal convulsions, death.

- **Treatment and Prevention**

- ✓ Slow IV injection of 1% methylene blue in distilled water or isotonic saline should be given at 4–22 mg/kg 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 hour should be considered.

- ✓ Rumen lavage with cold water and antibiotics may stop the continuing microbial production of nitrite.
- ✓ 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.
- ✓ However, caution is advised when combining other feed additives/components, including non-protein nitrogen, Ionophores (such as monensin) and other growth and performance enhancers, with high-nitrate diets in ruminants.

D.4. **Gossypol Poisoning (Cottonseed)**

- **Introduction**

Gossypol poisoning, which is usually sub-acute to chronic, cumulative, and sometimes insidious, follows consumption of cottonseed or cottonseed products that contain excess free gossypol. It is of most concern in domestic livestock, especially preruminants or immature ruminants and pigs; mature ruminants are somewhat more resistant to gossypol's toxic effects. However, gossypol toxicosis can affect high-producing dairy cows with high feed intake, dairy goats, and other mature ruminants fed excess gossypol for long periods of time. It has also been reported in dogs fed cottonseed meal in diets or housed on cottonseed bedding.

Figure 4:Cotton seed

Cottonseed

- **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.

- ✓ All animals are susceptible, but monogastric, preruminants, immature ruminants, and poultry appear to be affected most frequently. Adult ruminants are able to detoxify gossypol by formation of stable complexes with soluble proteins in the rumen, thus preventing absorption, something that is lacking in swine, pre ruminants, and young ruminants with only a partially functioning rumen.
- ✓ Pigs, guinea pigs, and rabbits are reported to be sensitive. Dogs and cats appear to have intermediate sensitivity. Holstein calves seem to be the most sensitive of cattle breeds. Goats may be more sensitive to gossypol than are cattle. Horses appear relatively resistant but caution is still advised. Toxic effects usually only occur after long term exposure to gossypol, often after weeks to months.

- **Clinical Findings**

Signs 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 with no visible cardiac lesions. Pulmonary effects, labored breathing, and chronic dyspnea are most likely secondary to cardio toxicity from congestive heart failure.

Signs of prolonged excess gossypol exposure 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.

- **Prevention, Treatment, and Control**

There is no effective treatment. Adsorbents such as activated charcoal and saline cathartics are of little value due to the chronic exposure and cumulative nature of gossypol. If gossypol toxicity is suspected, all cottonseed products should be removed from the diet immediately.

Caution is advised when incorporating gossypol-containing components in any animal diet. 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.

D.5.<u>Cyanide/Cassava Poisoning</u>

- **Etiology**

Various chemical forms of cyanides are found in plants, fumigants, soil sterilizers, fertilizers (e.g., Cyanamid), pesticides/rodenticides (e.g., calcium cyanomide) and salts used in industrial processes, such as gold mining, metal cleaning and electroplating, photographic processes, and others. Hydrogen cyanide is

also known as prussic acid, and cyanide salts liberate cyanide gas in the presence of acids (eg, in the stomach).

Toxicity can result from accidental, improper, or malicious use or exposure. However, in livestock species, the most frequent cause of acute and chronic cyanide poisoning is ingestion of plants that either constitutively contains cyanogenic glycosides or is induced to produce cyanogenic glycosides and cyanolipids as a protective response to environmental conditions (plant cyanogenesis).

Plant species of notable veterinary importance include *Sorghum spp* (Johnson grass, Sudan grass, the common cereal grain crop referred to as "sorghum" or the synonyms durra, jowari, milo), *Acacia greggii* (guajillo), *Amelanchieralnifolia* (western service berry), *Linumspp* (linseeds and flaxes), *Sambucusnigra* (elderberry), *Suckleysuckleyana* (poison suckleya), *Triglochinmaritima* and *T. palustris* (marsh arrow grasses).

- **Clinical Findings**

Acute cyanide poisoning: Signs generally occur within 15–20 min to a few hours after animals consume toxic forage, and survival after onset of clinical signs is rarely >2 hr. Excitement can be displayed initially, accompanied by rapid respiration rate. Dyspnea follows shortly, with tachycardia.

Chronic cyanide poisoning syndromes: Chronic cyanogenic glycoside hypothyroidism will present as hypothyroidism with or without goiter. Cystitis ataxia toxidromes are typically associated with posterior ataxia or incoordination that may progress to irreversible flaccid paralysis, cystitis secondary to urinary incontinence, and hind limb urine scalding and alopecia. Death, although uncommon, is often associated with pyelonephritis. Late-term abortion and musculoskeletal teratogenesis may also occur.

- **Treatment, Control, and Prevention**

Immediate treatment is necessary. The goal of treatment is to break the cyanide-cytochrome c oxidase bond and re-establish the mitochondrial electron transport chain. One way to accomplish this is by using inducing Fe3+ in hemoglobin (i.e., inducing methemoglobinemia), which then acts as a high-affinity decoy chemical receptor for cyanide and forms cyanmethemoglobin. Sodium thiosulfate alone is also an effective antidotal therapy at ≥500 mg/kg, IV, plus 30 g/cow, PO, to detoxify any remaining HCN in the rumen.

The best preventive step is to test suspect feed and/or pastures before allowing consumption. Pasture and forage sorghums (e.g., Sudan grass and sorghum-Sudan grass hybrids) should not be grazed until they are >60 cm tall or have been proved by testing to have acceptable cyanide levels, to reduce danger from prussic acid poisoning.

Figure 5: Cassava leaves and roots

D.6.Oxalates containing plants

Oxalate poisoning in animals generally occurs when quantities of oxalate-containing plants are grazed by livestock that are not accustomed to eating the plants. The most important oxalate-containing plants include halogeton (*H. glomeratus*), and greasewood (*S. vermiculatus*). Oxalate poisoning most often occurs when unadapted sheep or cattle are allowed to graze large amounts of *Halogeton* or *Sarcobatus* as they pass through or are pastured overnight on rangeland containing large stands of these plants.

Figure 6: Oxalate poisoning

- **Etiology**

Under normal range conditions sheep are most frequently poisoned by oxalate-containing plants. Ruminants in general tolerate relatively more oxalate in their diet than other animals because they are able to detoxify oxalate in the rumen thereby preventing the absorption of the soluble oxalates. When large quantities of soluble potassium and sodium oxalates are eaten that overwhelm the rumen's ability to metabolize the oxalates, they are absorbed into the bloodstream and form insoluble calcium and magnesium oxalates. It is these insoluble salts that precipitate in the kidneys and cause kidney failure

- **Toxic Effects of Oxalates**

Once absorbed from the gastrointestinal tract, soluble oxalates rapidly combine with serum calcium and magnesium, causing a sudden decrease in available serum calcium and magnesium. In the acute phase of oxalate poisoning the sudden decrease in soluble serum calcium (hypocalcemia) impairs normal cell membrane function, causing animals to develop muscle tremors and weakness, leading to collapse and eventually death.

In chronic oxalate poisoning, insoluble calcium oxalate filtered by the kidneys causes severe damage to the kidney tubules (oxalate nephrosis). If animals do not die from the acute effects of the low blood calcium levels and impaired cellular energy metabolism, death results from kidney failure.

- **Clinical Signs**

Within a few hours of consuming toxic levels of oxalate, sheep and cattle develop muscle tremors, tetany, weakness, reluctance to move, depression, and recumbency resulting from hypocalcemia and hypomagnesemia. Coma and death may result within 12 hours. Animals that survive the acute effects of oxalate poisoning frequently succumb to kidney failure. As animals become uremic (increased serum creatinine and urea nitrogen levels), they develop severe depression, stop eating, and after a few days become comatose and die.

- **Treatment**

Treatment with intravenous calcium gluconate, although theoretically appropriate for correcting hypocalcemia, is not effective in reversing the effects of the oxalate on cellular energy metabolism. Irreversible oxalate nephrosis and the effects of oxalates on cellular energy metabolism are more detrimental to the animal than hypocalcemia. A theoretical approach to treating acute oxalate poisoning would be to administer intravenous calcium gluconate, magnesium sulphate, glucose, and a balanced electrolyte solution to maintain kidney perfusion.

- **Prevention of Oxalate Poisoning**

Livestock should not be grazed on rangeland on which oxalate-containing plants predominate without precaution, especially if the animals are hungry and have not been adapted to oxalate in their diet. Livestock should be introduced to oxalate plants for at least 4 days by incrementally increasing the time they are allowed to graze the plants. Overstocking and overgrazing will potentiate oxalate poisoning if there is no other vegetation for animals to eat. Cattle and sheep driven through or held overnight in pastures rich in oxalate-containing plants are prone to poisoning, and such circumstances should be avoided. Supplementary dicalcium phosphate in the diet before and during high-risk oxalate exposure is an effective means of reducing losses. High levels of dietary calcium bind oxalate in the rumen as insoluble, non-absorbable calcium oxalate.

D.7. <u>Bracken Fern Poisoning</u>

Bracken fern (*Pteridium aquilinum*) is found throughout the world and is among the five most numerous vascular plants. The species includes numerous subspecies and varieties, and plant size varies with frond lengths ranging from 0.5 to 4.5 m.

Figure 7: Bracken fern

- **Syndromes**

A variety of syndromes have been associated with bracken fern poisoning. These syndromes are largely determined by the dose and duration, and also by the species of the poisoned animal.

- ✓ **Enzootic hematuria** is the most common result of bracken fern poisoning. It primarily affects cattle and, less frequently, sheep. It is characterized by intermittent hematuria and anemia.
- ✓ **Acute Brackenism or Haemorrhagic Disease:** Acute Brackenism occurs when animals ingest high doses over relatively short durations of weeks or months. It is characterized by bleeding.
- ✓ **Bright Blindness:** A less common manifestation of ptaquiloside toxicity is called bright blindness.
- ✓ **Bracken Staggers:** Bracken fern poisoning in monogastric animals was first recognized as a neurologic disease when horses consumed contaminated hay. Ingestion at a rate of 20%–25% bracken fern for ≥3 month may result in bracken staggers. Clinical signs in horses include anorexia, weight loss, incoordination, and a crouching stance while arching the back and neck with the feet placed wide apart.

- **Treatment**

Initial treatment for all species is to discontinue exposure to bracken fern; however, disease can appear weeks after livestock are removed from the fern-infested area. In acutely affected cattle, mortality is usually >90%. Measurement of the platelet count is recommended, because it is the best prognostic indicator for poisoned animals. Antibiotics may be useful to prevent secondary infections.

- **Prevention**

Poisoning, apart from thiamine deficiency, is essentially untreatable; however, it is most easily controlled by preventing exposure. Bracken fern is usually grazed for want of alternative forages. Most commonly, animals are forced to eat bracken fern when other forage is exhausted in late summer, although some animals may develop a taste for the young tender shoots and leaves. Poisoning can be avoided by improving pasture management to increase production of alternative forage. It has been suggested that alternating bracken fern–contaminated and non-contaminated pastures at 3-wk intervals can minimize poisoning.

- Content/Topic 6: Intoxications by Mycotoxicoses

A. Introduction

A **mycotoxicosis** is a disease caused by a natural toxin produced by a fungus. Acute or chronic Toxicoses can result from exposure to feed or bedding contaminated with toxins produced during growth of various saprophytic or phytopathogenic fungi or molds on cereals, hay, straw, pastures, or any other fodder.

A.1. A few principles characterize mycotoxic diseases:

- The cause may not be immediately identified;
- They are not transmissible from one animal to another;
- Treatment with drugs or antibiotics has little effect on the course of the disease;
- Outbreaks are usually seasonal because particular climatic sequences may favor fungal growth and toxin production;
- Study indicates specific association with a particular feed; and
- Although large numbers of fungi found on examination of feedstuff does not necessarily indicate that toxin production has occurred.

A.2.Aflatoxicosis

Aflatoxins are produced by toxigenic strains of Aspergillus flavus and *A. parasiticus* on peanuts, soybeans, corn (maize), and other cereals either in the field or during storage when moisture content and temperatures are sufficiently high for molds growth. The toxic response and disease in mammals and poultry varies in relation to species, sex, age, nutritional status, and the duration of intake and level of Aflatoxins in the ration.

- **Clinical Findings**

In acute outbreaks, deaths occur after a short period of inappetence. Sub-acute outbreaks are more usual, and unthriftiness, weakness, anorexia, and sudden deaths can occur. Generally, aflatoxin concentrations in feed >1,000 ppb are associated with acute aflatoxicosis. Frequently, there is a high incidence of concurrent infectious disease, often respiratory, that responds poorly to the usual chemotherapy. Dairy cattle experience inappetence, and ruminants may have decreased ruminal contractions at high concentrations

(>1 ppm) of aflatoxins. Liver damage can lead to reduced clotting factor synthesis with acute to chronic haemorrhage.

- **Control**

Contaminated feeds can be avoided by monitoring batches for aflatoxin content. Local crop conditions (drought, insect infestation) should be monitored as predictors of aflatoxin formation. Young, newly weaned, pregnant, and lactating animals require special protection from suspected toxic feeds. Dilution with non-contaminated feedstuff is one possibility. Ammoniation of grain reduces contamination but is not currently approved for use in food animals because of uncertainty about by products produced.

Hydrated sodium calcium aluminosilicates (HSCAS) reduce the effects of aflatoxin when fed to pigs or poultry; at 10 lb. /ton (5 kg/tonne), they provided substantial protection against dietary aflatoxin. HSCAS reduces aflatoxin M1 by ~50% but does not eliminate residues of aflatoxin M1 in milk from dairy cows fed aflatoxin B1. Other adsorbents (sodium bentonites, polymeric glucomannans) have shown variable but partial efficacy in reducing low-level aflatoxin residues in poultry and dairy cattle.

A.3. <u>Ergotism</u>

This worldwide disease of farm animals results from continued ingestion of sclerotia of the parasitic fungus ***Claviceps purpurea***, which replaces the grain or seed of rye and other small grains or forage plants, such as the bromes, bluegrasses, and ryegrasses. The hard, black, elongated sclerotia may contain varying quantities of ergot alkaloids, of which ergotamine and ergonovine (ergometrine) are pharmacologically most important. Cattle, pigs, sheep, and poultry are involved in sporadic outbreaks, and most species are susceptible.

- **Etiology**

Ergot causes vasoconstriction by direct action on the muscles of the arterioles, and repeated dosages injure the vascular endothelium. These actions initially reduce blood flow and eventually lead to complete stasis with terminal necrosis of the extremities due to thrombosis.

- **Clinical Findings and Lesions**

✓ **Cattle** may be affected by eating ergotized hay or grain or occasionally by grazing seeded pastures that are infested with ergot. Lameness, the first sign, may appear 2–6 week or more after initial ingestion, depending on the concentration of alkaloids in the ergot and the quantity of ergot in the feed. Hind limbs are affected before forelimbs, but the extent of involvement of a limb and the number of limbs affected depends on the daily intake of ergot. Body temperature and pulse and respiration rates are increased.

✓ In **pigs**, ingestion of ergot-infested grains may result in reduced feed intake and reduced weight gain. Occasionally, swine may show necrosis of the tips of ears or tail. If fed to pregnant sows, ergotized grains result in lack of udder development with agalactia at parturition, and the piglets

born may be smaller than normal. Most of the litter die within a few days due to starvation. No other clinical signs or lesions are seen.

- ✓ Clinical signs in **sheep** are similar to those in cattle. Additionally, the mouth may be ulcerated, and marked intestinal inflammation may be seen at necropsy. A convulsive syndrome has been associated with Ergotism in sheep.

- **Control**

Ergotism can be controlled by an immediate change to an ergot-free diet. In pregnant sows, however, removal of ergot in late gestation (<1 week before parturition) may not correct the agalactia syndrome. Under pasture feeding conditions, frequent grazing or topping of pastures prone to ergot infestation during the summer months reduces flower-head production and helps control the disease. Grain that contains even small amounts of ergot should not be fed to pregnant or lactating sows.

- Content/Topic 7: Intoxications by poisonous and venomous organisms

Fatal snakebites are more common in dogs than in other domestic animals. Because of the relatively small size of some dogs in proportion to the amount of venom injected, the bite of even a small snake may be fatal.

A. Snakebite

A.1.Introduction

Fatal snakebites are more common in dogs than in other domestic animals. Because of the relatively small size of some dogs in proportion to the amount of venom injected, the bite of even a small snake may be fatal. In dogs and cats, mortality is generally higher in bites to the thorax or abdomen than bites to the head or extremities.

Because of their larger sizes, horses and cattle seldom die as a direct result of snakebite, but deaths may follow bites on the muzzle, head, or neck when dyspnea results from excessive swelling. Serious secondary damage sometimes occurs; livestock bitten near the coronary band may slough a hoof.

A.2. Treatment

Intensive therapy should be instituted as soon as possible, because irreversible effects of venom begin immediately after envenomation. The bite site(s) should be shaved, and the wounds cleansed thoroughly with germicidal soap. For animals bitten by crotalids, the leading edge of tissue swelling should be marked on the skin with a magic marker at frequent intervals to monitor the spread of tissue injury. All snakebite victims should be monitored closely for a minimum of 24 (crotalids) to 48 (elapid) hour for the development of clinical signs. Anti-venom is the only direct and specific means of neutralizing snake venom.

Animals bitten by elapids may be treated with supportive care as needed (IV fluids, ventilatory support, anticonvulsants, etc.) and anti-venom/antivenin, if available.

A.2. Prognosis

The prognosis of snakebite depends on the type and species of snake, location of the bite, size of the victim, degree of envenomation, and the time interval between the bite and the institution of treatment. Animals that survive elapid bites generally make full recoveries, but crotalids bites can result in long term sequelae due to tissue necrosis (amputation, loss of function, etc.), depending on the severity of the bite and the promptness and aggressiveness of treatment instituted.

B. Venomous Arthropods

Envenomation of animals by poisonous arthropods is relatively uncommon and difficult to recognize. It may be suspected relative to presenting clinical signs, but confirmatory evidence is rare because of the inability to capture the offending arthropod.

C. Spiders and Scorpions

Spiders of medical importance do not inflict particularly painful bites, so it is unusual for a spider bite to be suspected until clinical signs appear. It is also unlikely that the spider will remain in close proximity to the victim for the time required for signs to develop (30 min to 6 hour). Almost all spiders are venomous, but few possess the attributes necessary to cause clinical envenomation in mammals—mouth parts of sufficient size to allow penetration of the skin and toxin of sufficient quantity or potency to result in morbidity.

Most of the scorpions found throughout the USA possess posterior abdominal stingers that connect to venom glands. The stinger and its associated venom can be used both as mechanisms of self-defence and of predation. For the most part, the stings of these scorpions are considered to be innocuous in most domesticated mammalian species, because the amount of venom is too minute or the venom has very little pharmacologic potency. The sting of these arthropods is analogous to an insect sting/bite, with pain and swelling at the site of the injury.

D. Hymenoptera

There are many venomous hymenopterans (e.g., honey bees, wasps, hornet, and yellow jackets), in which the female drone possesses a barbed ovipositor on the tip of the abdomen that connects to paired venom glands. A single bee sting will produce pain, swelling, erythema, edema, and local induration, which can be followed by pruritus at the injection site.

In dogs, bee and wasp stings cause only local redness, erythema, and transient pain. Dogs may vocalize when stung and may rub their mouth and eyes on the ground. Usually cutaneous reactions appear quickly and regress spontaneously. With repeated stings, anaphylaxis, with salivation, vomiting, diarrhea, circulatory collapse, pallor, or cyanosis, may result.

In cases of anaphylaxis, epinephrine should be immediately administered, SC, at a dosage of 1:1,000 (0.1–0.5 mL) for dogs or cats. This dosage can be repeated every 10–20 min. When given IV, it must be diluted to 1:10,000, and 0.5–1.0 mL is administered with vigilant monitoring of heart rate, heart rhythm, and blood pressure. IV fluids are indicated to prevent vascular collapse. Antihistamines and corticosteroids should also be given.

E. Africanized Honeybees ("Killer Bees")

Africanized bees are difficult to distinguish morphologically from their European counterparts. Their stinging behavior is primarily defensive, e.g., in response to a perceived threat to the colony.

Animals that receive massive envenomations are visibly depressed and usually febrile. They may exhibit facial paralysis, ataxia, seizures, and neurologic signs. The urine may be dark brown or red, and the feces are bloody. Bloody or dark brown vomitus may be seen.

The animal may develop acute renal failure caused by acute tubular necrosis or direct toxic effect of massive envenomation. Dogs may develop a secondary immune-mediated hemolytic anemia. Access to cardiac monitoring, supplemental oxygen, "crash cart" drugs, and airway intubation must be readily available. Any animal that receives massive; multiple stings must be hospitalized, treated aggressively, and hospitalized for 24 hour after cessation of clinical signs.

- Content/Topic 8:Bacterial Toxins and Salmonellosis

A. Bacterial toxins

Bacterial foodborne infections occur when food that is contaminated with bacteria is eaten and the bacteria continue to grow in the intestines, setting up an infection which causes illness. *Salmonella*, *Campylobacter*, hemorrhagic *E. coli* and *Listeria* all cause infections.

Food intoxication results from consumption of toxins (or poisons) produced in food by bacterial growth. Toxins, not bacteria, cause illness. Toxins may not alter the appearance, odor or flavor of food. Common kinds of bacteria that produce toxins include *Staphylococcus aureus* and *Clostridium botulinum*. In the case of *Clostridium perfringens*, illness is caused by toxins released in the gut when large numbers of vegetative cells are eaten.

A.1.Botulism

Botulism is a rapidly fatal motor paralysis caused by ingestion of the toxin produced by *Clostridium botulinum* types A-G. The spore-forming anaerobic organism proliferates in decomposing animal tissue and sometimes in plant material

- **Etiology**

Botulism is in most cases intoxication, not an infection, and results from ingestion of toxin in food. There are seven types of *C botulinum*, differentiated on the antigenic specificity of the toxins: A, B, C_1, D, E, F, and G. Types A, B, and E are most important in people; C_1 in most animal species, notably wild ducks, chickens, cattle, and horses; and D in cattle.

- ✓ The exact incidence of botulism in animals is not known, but it is relatively low in cattle and horses, probably more frequent in chickens, and high in wild waterfowl.
- ✓ Dogs, cats, and pigs are comparatively resistant to all types of botulinum toxin when challenged orally; however, there are recent individual case reports mentioning botulism in dogs.

- **Clinical Findings**

The signs of botulism are caused by flaccid muscle paralysis and include progressive motor paralysis, disturbed vision, difficulty in chewing and swallowing, and generalized progressive paresis. Death is usually due to respiratory or cardiac paralysis.

- **Treatment and Control**

Any dietary deficiencies in range animals should be corrected and carcasses disposed of, if possible. Decaying grass or spoiled silage should be removed from the diet. Botulinum antitoxin has been used for treatment with varying degrees of success, depending on the type of toxin involved and the species of host. Treatment of ducks and mink with type C antitoxin is often successful; however, such treatment is rarely used in cattle.

Note: *Clostridium perfringens* belong to the same genus as the botulinum organism. However, the disease produced by *C. perfringens* is not as severe as botulism. *Clostridium perfringens* type A is believed to cause diarrhea by elaboration of an enterotoxin, which is released during sporulation and stimulates intestinal epithelial cells to secrete excess fluid into the lumen.

A.2.Salmonellosis

Salmonella, a rod-shaped gram-negative bacterium belonging to the family Enterobacteriaceae, is the causative agent of salmonellosis. Salmonellosis in warm-blooded vertebrates is in most cases associated with serovars of *Salmonella* subsp *enterica*.

- **Etiology and Pathogenesis**

Salmonellosis has been recognized in all parts of the world but is most prevalent in regions with intensive animal husbandry. Although this facultative intracellular pathogen is primarily an intestinal bacterium, it is commonly found in an environment subject to fecal contamination. Feces of infected animals can contaminate feed and water, milk, fresh and processed meats from abattoirs, plant and animal products used as fertilizers or feedstuffs, pasture and rangeland, and many inert materials.

Although many other *Salmonella* spp may cause enteric disease, the more common ones (to some extent varying according to geographic location) in each species are as follows: cattle—*S typhimurium*,*S dublin*, and *S.newport*; sheep and goats—*S typhimurium*, *S dublin*, *S abortusovis*, *S anatum*, and *S montevideo*; pigs—*S typhimurium* and *S choleraesuis*; horses—*S typhimurium*, *S anatum*, *S newport*,*S enteritidis*, and poultry—*S enteritidis*, *S typhimurium*, *S gallinarum*, and *S pullorum*.

- **Clinical Findings**

Infection with localization of the pathogen in tonsils or the GI tract that is not associated with clinical disease is a common form of salmonellosis termed as the carrier state.

Enteritis with septicemia is the usual syndrome in new born calves, lambs, foals, fowl, and piglets, and outbreaks may occur in pigs up to 6 months old. When systemic disease occurs with enteritis as a result of insufficient immunity, illness may be acute, with depression, fever (40.5°–41.5°C), and death in 24–48 hr. Nervous signs and pneumonia may be seen in calves and pigs. Mortality may reach 100%, depending on the host genetic background and strain virulence. Acute enteritis without extensive systemic involvement is more common in adults as well as in young animal's ≥1 week old.

- **Treatment**

Early treatment is essential for septicemic salmonellosis, but there is controversy regarding the use of antimicrobial agents for intestinal salmonellosis. Oral antibiotics may be ineffective and may deleteriously alter the intestinal micro flora. If oral medication is chosen, it should be given in drinking water and not mixed into solid feed, because affected animals are thirsty due to dehydration and their appetite is generally poor. Fluid therapy to correct acid-base imbalance and dehydration may be necessary.

- **Control and Prevention**

Carrier animals and contaminated feedstuffs and environment are major problems. Drain swabs or milk filters may be cultured to monitor the salmonellae status of a herd. The principles of control include prevention of introduction and limitation of spread within a herd.

- **Zoonotic Risk**

Infections with *Salmonella* in food-producing animals present a serious public health concern, because food products of animal origin are considered to be a significant source of human infection. Most common sources of infection are eggs and related products, and meat from poultry and other food animal species. Milk and dairy products have also been associated with outbreaks of salmonellosis in people.

- Content/Topic 9: Feed and Water Contaminants

Groundwater will normally look clear and clean because the ground naturally filters out particulate matter. But, natural and human-induced chemicals can be found in ground water. As groundwater flows through the ground, metals such as iron and manganese are dissolved and may later be found in high

concentrations in the water. Industrial discharges, urban activities, agriculture, ground-water pumpage, and disposal of waste all can affect ground-water quality.

Contaminants can be human-induced, as from leaking fuel tanks or toxic chemical spills. Pesticides and fertilizers applied to lawns and crops can accumulate and migrate to the water table. Leakage from septic tanks and/or waste-disposal sites also can introduce bacteria to the water, and pesticides and fertilizers that seep into farmed soil can eventually end up in water drawn from a well.

Or, a well might have been placed in land that was once used for something like a garbage or chemical dump site. In any case, if you use that water for animal feeding, intoxication m happen after a long use of that contaminated water. Different signs, some of them similar to the previous ones for other types of intoxication (Vomiting, diarrhea…) will be seen.

LO 1.4 – Characterize nutritional and metabolic diseases

- Content/Topic 1: Differentiation of nutritional and metabolic diseases and their Causes

A. Difference between nutritional and metabolic diseases

Most production-induced metabolic diseases result from a negative balance of a particular nutrient. In some cases, dietary intake of the nutrient is rapidly reduced because of an on-going, high metabolic requirement for that nutrient. Examples include pregnancy toxemia in ewes, protein-energy malnutrition in beef cattle, and fat cow syndrome in dairy and hyper lipemia in ponies. Furthermore, some diseases may be precipitated when producers, primarily because of economic concerns, are compelled to not supplement the diet of animals that already have a substandard nutritional plane.

Diseases such as hypocalcemia, hypomagnesemia, and hypoglycemia are augmented by management practices directed toward improving and increasing production. They are therefore correctly considered production diseases. However, they are also metabolic diseases because management of the animal is directed at production, which at its peak is beyond the capacity of that animal's metabolic reserves to sustain a particular nutrient at physiologic concentrations. For example, parturient paresis of cows occurs when the mass of calcium in the mammary secretion is greater than the cow's diet or its skeletal reserves can supply. Comparable situations occur with magnesium and glucose metabolism, and with phosphorus in relation to post parturient hemoglobinuria.

The difference between production-related metabolic diseases and nutritional deficiencies is often subtle. Typically, nutritional deficiencies are long term, steady-state conditions that can be corrected through dietary supplementation. Metabolic diseases are generally acute states that dramatically respond to the systemic administration of the deficient nutrient or metabolite, although affected animals may require subsequent dietary supplementation to avoid recurrence. An important aspect of dealing with production-

induced metabolic diseases is accurate and rapid diagnosis. Ideally, diagnostic tests can be used to predict the occurrence of disease before its clinical onset.

B. Causes of nutritional and metabolic diseases

- Inadequate nutrients provision (deficiency, excess, intoxications)
- Metabolic routes alteration
- Unbalanced feed ration
- Inert or actives feed contaminants
- Drinking and feeding errors
- Storage and conservation conditions of feeds

- Content/Topic 2: Nutritional disorders due to energetic and proteins deficiency or excess

A. Growth retardation

A.1.Malnutrition

One of the most common causes of retarded growth is malnutrition resulting from dietary deficiency or severe gastrointestinal parasitism. When these problems are severe or prolonged, reduced growth may never be totally made up during the catch-up growth phase that occurs once normal nutrition is established. Deficiencies of vitamin A almost always cause growth retardation. Initially, deficiency of vitamin D does not slow growth; however, the resulting abnormalities in bone metabolism and bone growth may cause severe growth suppression. The mechanism by which zinc deficiency causes growth suppression is unknown.

Disease of the digestive system that results in mal digestion or mal absorption of nutrients may cause severe growth retardation. Pancreatic insufficiency in dogs may occur prior to puberty and may result in severe growth retardation.

A.2. Cachexia

Cachexia is a common condition in companion animals, occurring in a variety of chronic and acute diseases, such as CHF, cancer, CKD, chronic respiratory disease, and acute illness or injury. As in humans, the weight loss that occurs in cachexia is unlike that seen in a healthy animal that loses weight, and the primary tissue lost in cachexia is muscle and lean body mass (LBM). Although there are some differences among forms of cachexia in different diseases (e.g., cardiac cachexia versus cancer cachexia), a quantitative and qualitative loss of muscle is a hallmark of cachexia in dogs and cats. Fat and bone also are lost to a lesser degree, although in advanced cases of cachexia, all body compartments are depleted.

- **Clinical implications of cachexia**

In dogs and cats, although there are studies associating thin body condition with decreased survival in general populations of pet cats and in a variety of diseases. Many of the effects of cachexia that have been documented in humans also are identified in dogs and cats with cachexia (e.g., weakness, anorexia, weight loss, and poor quality of life.

- **Diagnosis of cachexia**

In veterinary medicine, the diagnosis of cachexia has focused on muscle loss, rather than weight loss in an effort to diagnose these conditions at an earlier stage. Weight loss can be an additional indicator of cachexia but is not required for a diagnosis of either of these conditions.

Moderate to severe cachexia is not difficult to identify in a person or dog with advanced CHF or cancer. However, identification of cachexia in its earlier and more subtle stages can be more challenging, but is one of the keys to successful management of these common conditions. Body composition of companion animals can be assessed by a variety of methods (e.g., dual X-ray absorptiometry [DEXA], computed tomography [CT]), and both have been used to identify cachexia in dogs and cats The most commonly used clinical assessment of body composition in veterinary medicine is the body condition score (BCS). This is a validated score classified on a 1-9 scale where 1=emaciated, 9=obese, and 4-5 is considered ideal. Body condition score is a semi-quantitative assessment of body composition focusing primarily on stores of adipose tissue

Note: Lactic indigestion will be discussed in metabolic disease

- Content/Topic 3: Metabolic disease

A. Acetonemia (Ketosis in ruminants, Acetonemia in cattle, Pregnancy Toxemia in Sheep)

A.1 Etiology

- A multi factorial disorder of energy metabolism.
- Negative energy to hypoglycemia and ketonemia (the accumulation in blood of **acetoacetate**, beta-**hydroxybutyrate** and their decarboxylation products **acetone** and isopropanol).
- The diseases in cattle and sheep occur in different parts of the pregnancy-lactation cycle.

It is not unreasonable to view clinical ketosis as the top end of a spectrum of a metabolic state that is **common in heavily producing cows** in the **post calving period**. This is because high yielding cows in early lactation are in negative energy balance and are sub clinically ketotic as a result. Ruminants are particularly vulnerable to ketosis because, although very little carbohydrate is absorbed as such, a direct supply of glucose is essential to tissue metabolism, particularly the formation of lactose. The utilization of volatile

fatty-acids for energy purposes is also dependent upon a supply of available glucose. This vulnerability is further exacerbated, particularly in the cow, by the tremendous rate of turnover of glucose.

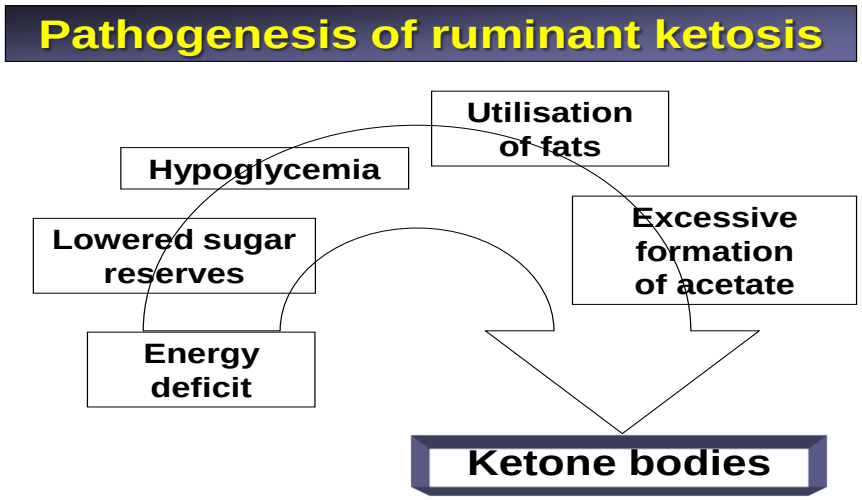

Figure 8: Pathogenesis of ruminant ketosis

A.2. Epidemiology

Primary ketosis occurs in well-conditioned cows with high lactation potential, principally in the first month of lactation with a higher prevalence in cows with a higher lactation number. Secondary ketosis occurs where other disease reduces feed intake. The disease in sheep is associated with a falling plane of nutrition, principally in the last month of pregnancy, in ewes bearing twins and triplets but can be induced by other stress at this time.

A.3. Clinical findings

Cattle show wasting with decrease in appetite, fall in body condition and milk production. Some have short periods of bizarre neurological and behavioural abnormality. Response to treatment is good. Sheep have encephalopathy with blindness, muscle tremor, convulsions, metabolic acidosis and a clinical course of **2-8** days, usually terminating fatally unless treated early.

A.4. Diagnostic confirmation

- Ketonemia,
- Ketonuria or elevated ketones in milk.
- Elevated beta-hydroxybutyrate (BHBA) in aqueous humour of dead sheep.

A.5. Treatment

- In cattle, parenteral glucose with corticosteroid and oral glucose precursors such as propylene glycol, occasionally insulin.
- Similar treatment in sheep, or oral glucose and electrolyte therapy.
- Cesarian section or induction of parturition in sheep. In cattle, the disease responds readily to treatment and is self-limiting, but in sheep the disease is highly fatal.

A.6. Control

- Correction of energy imbalance.
- Herd and flock biochemical monitoring coupled with condition scoring.

B. Rumen acidose(lactic indigestion)

B.1. Introduction

Acidosis is the most common nutritional disorder in the feedlot. A large amount of highly fermentable feeds, such as cereal grains, consumed in a short amount of time can result in the production of **more lactic acid than can buffered by the rumen.** This results in water from the circulatory system being drawn into the rumen (body becomes dehydrated) and pronounced changes in the blood Ph. Signs will usually be acute or sub-acute. Survivors of acute acidosis may have chronic problems such as fungal rumenitis, liver abscesses, bloat, and founder or laminitis.

B.2. Acute acidosis

Animals that are not adapted to readily fermentable feeds are more susceptible to acidosis (sometimes called grain overload) than animals that have been carefully adjusted. However, even animals conditioned to full feed can be susceptible under some conditions such as feed changes and temporary restrictions in feed availability. Acutely affected animals will usually develop signs within 12-24 hours of overeating. They will be completely off feed, depressed and unwilling to move, weak, and dehydrated. They may appear blind, grind their teeth, grunt, and occasionally kick at their belly. Fullness and distension of the abdomen (rumen) may be observed. A foul smelling diarrhea may be observed unless the condition is so acute that the animal dies before it can develop.

In severe cases animals will lie down, unable to rise. They generally lie quietly with their head tucked to the side. Body temperature may be subnormal and the pulse is weak. Death usually occurs within a few hours after the animals go down.

Animals that survive may suffer from damaged ruminal lining and destruction of rumen microflora leading to a fungal overgrowth of the rumen and death. Some deaths may occur as long as 3 weeks after a herd episode of overeating and acidosis. Less severe rumen lining damage may lead to liver abscesses and growth impairment. Laminitis, or founder, may follow acute acidosis, and evidence of subacute laminitis in the form of overgrown and deformed hooves may be present 30-60 days later.

B.3. Sub-acute acidosis

Animals with less acute and severe signs may still eat but may not consume as much as normal or be off feed for only a short time. The only overt signs of sub-acute acidosis may be reduced gains and the presence of diarrhea in the form of flat gray stools. Because rumen lining damage may still occur in the absence of severe signs, these animals may develop chronic rumen damage and liver abscesses.

Weather conditions can cause fluctuations of intake of an otherwise acceptable ration. Storm conditions can cause cattle to consume a greater amount of feed before and after the storm. Muddy conditions which can alter feed intake. A drop in barometric pressure can indicate oncoming storm conditions. Conditions that promote intake of the regular ration in a shorter amount of time can cause acidosis. Hot, humid weather will cause cattle to eat a greater proportion of their feed at night, rather than during the day.

Improper mixing of feed can cause acidosis. As previously discussed improper bunk management can be a cause of acidosis. Only occasional cleaning of water troughs will also affect intake. Inclusion of an ionophore may help reduce intake fluctuations.

C. Alkalosis:

The physiology of the digestive tract of ruminants is based on relatively low-energy plant foods (grass, hay) and therefore administration of high energy protein feed frequently leads to dietary diseases such as ruminal acidosis and alkalosis. In high performance animals an adequate supply of nutrients can only be achieved with additional high energy protein feed. Rumen acidosis and rumen alkalosis are among the most frequent diseases caused by a faulty diet in dairy cattle husbandry. The economic losses are caused more by subclinical or chronic cases than by acute acidosis.

- Content/Topic4: Mineral nutritional disorders

A. Milk fiver (hypocalcemia)

- **Causes**

Milk fever (**parturient paresis or hypoca+lcemia**) is generally associated with older, high producing dairy cattle. However, incidences of milk fever may also occur with beef cattle. Milk fever occurs **shortly after calving and the onset of milk production**. Milk fever occurs when the lactating cow is not capable of absorbing enough calcium from the diet or has not started mobilizing bone calcium to meet the increased calcium demand of lactation. Calcium losses from lactation coupled with inadequate supply results in a drop in blood calcium level. Since calcium is needed for muscle contraction, cows suffering from milk fever often lose their ability to stand.

- **Clinical Signs**

There are three progressive stages of parturient paresis.

- ✓ **During stage one**; cows are able to stand but show signs of hypersensitivity and excitability.Cows may appear restless and bellowing. If calcium therapy is not instituted, cows will progress to stage two.

- ✓ **In stage two**, cows are unable to stand but can maintain sternal recumbency. Depression, anorexia, dry muzzle, subnormal body temperature, and cold extremities are seen. Cows often tuck their heads into their flanks or, if the head is extended, an S-shaped curve to the neck may be noted.
- ✓ **In stage three**, cows lose consciousness progressively to the point of coma. They are unable to maintain sternal recumbency, unresponsive to stimuli, and can suffer severe bloat. Cardiac output worsens, heart rate can approach 120 beats /min, and pulse may be undetectable. Cows in stage three may survive only a few hours.

Figure 9: Clinical Presentation of milk fever in stage one

- **Treatment**

The most common treatment is slowly applying an intravenous injection of a calcium gluconate solution. Single dose bottles are typical and are available at local veterinary clinics and supply stores. Calcium may also be provided orally as calcium propionate in a gel form. Retreatment is necessary in some cases.

Figure 10: Intra venous injection of calcium borogluconate

- **Prevention**

Numerous steps can be taken to prevent milk fever. The first is to evaluate the calcium and phosphorus levels of the diet. Excessive dietary calcium during late pregnancy could leave the cow unprepared to absorb or mobilize (resorb from bone) enough calcium to meet elevated requirements when lactation starts.

B. <u>Grass tetany (hypo magnesia)</u>

- **Causes and clinical signs**

Grass tetany is associated with low levels of magnesium or calcium in cattle grazing ryegrass, small grains (e.g., oats, rye, wheat) and cool season perennial grasses (e.g., tall fescue, orchard grass). Grass tetany most commonly affects lactating cattle, particularly the highest producing animals in the herd. Magnesium and calcium requirements of lactating cattle are far greater than those of dry cattle. This predisposes cattle to grass tetany during lactation. Grass tetany results when magnesium and calcium levels in forages are too low to meet the requirements of cattle and cattle do not receive adequate magnesium and calcium supplementation.

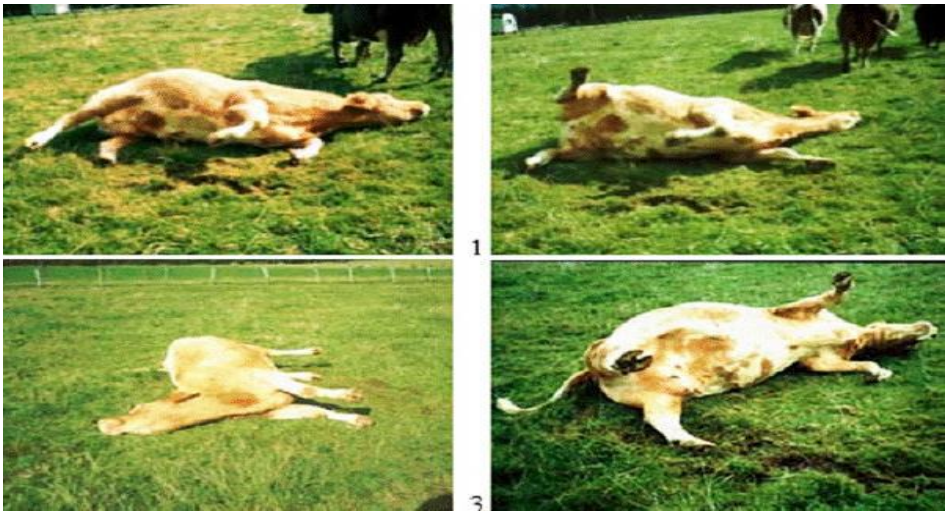

Figure 11: Cattles with Grass Tetany

- **Clinical signs of Grass tetany in cattle**

Clinical signs of grass tetany include nervousness, muscle twitching and staggering during walking. An affected animal may go down on its side, experience muscle spasms and convulsions and die if not treated.

- **Prevention**

Magnesium deficient pastures should be limed with dolomitic lime, which contains magnesium. This may not be effective in preventing grass tetany on waterlogged soils, since plants may not be able to take up sufficient magnesium under wet conditions. Phosphorus fertilization may also be useful for improving forage magnesium levels. However, environmental concerns associated with excessive soil phosphorus

levels should be considered. Legumes (e.g., clovers, alfalfa, lespedezas) are often high in magnesium and may help reduce the risk of grass tetany when included in the forage program.

The most reliable method of grass tetany prevention is supplemental feeding of magnesium and calcium during the grass tetany season**.** Both can be included in a mineral mix as part of a mineral supplementation program. Start feeding high magnesium mineral one month prior to grass tetany season.

- **Treatment**

Early treatment of grass tetany is important. Collapsed cattle that have been down more than 12 to 24 hours will seldom recover. Blood magnesium levels can be increased within 15 minutes by intravenously administering 500 ml of calcium borogluconate solution with 5 percent magnesium hypophosphate. The solution must be administered slowly, and heart and respiratory rates should be monitored closely during administration.

C. Rachitism

C.1.Introduction

Rickets is a disease of young animals—calves, foals, pigs, lambs, kids, pups, and chicks—which are characterized by a failure of growing bone to calcify, or harden. Bones that are rapidly increasing in length, such as the long bones of the legs and the ribs, are the most likely to be visibly affected. At the epiphysis (the region near the ends of bones where growth in length takes place) cartilage continues to be produced, but normal bone is not formed. The epiphyseal region thus becomes wider than it is normally, and because it is soft from lack of ossification (bone formation) it becomes more or less curved and bulges out, causing irregular enlargements, due presumably to the weight of the animal body and the normal tension or strain of the muscles. The result is a noticeable enlargement of the joints, particularly the knees and hocks, with an abnormal straightening or curving of the pasterns which interferes with normal walking. In addition the joint surfaces (articular cartilages) may become eroded and roughened so that movement of the joint is painful. This results in a typical stilted gait, and the animal lies down often. These malformed bones may cause paralysis or pain by pressing on nerves, and they are very easily fractured because of their fragility. Bones also become softened because of partial resorption of salts from previously formed bone when there is a deficiency of the normal quantity of mineral salts to be deposited in the newly forming bone. The shafts of the leg bones may bend, presumably owing to the weight of the body and to muscle tension. The spinal column also may bend sidewise, a hump may form, or the back may sway. The bones of the head may become distorted, with a tendency towards shortening and an increase in width. Teeth that grow in during a rachitic period may be malformed. The skeletal abnormalities described are symptoms of the advanced stage of the disease, but they may be the first ones a stockman notices or recognizes. Symptoms that often precede the more severe clinical manifestations are loss of appetite, slowing down of growth or even loss of weight, digestive disturbances, and tetany (convulsions, rigid muscles, stiff legs). A decline in

blood calcium or inorganic phosphorus or both usually precedes all other clinical symptoms. Where laboratory facilities are available to make such determinations, the loss of these minerals provides an excellent means of detecting vitamin D deficiency or other abnormalities affecting calcium and phosphorus metabolism.

C.2.Cause prevention and treatment

Swine and poultry that are normally fed heavily on grains may not receive an adequate supply of calcium unless a mineral supplement or a feed source of calcium is provided. The supply of phosphorus is usually ample in a heavy grain ration. The normal calcium-phosphorus ratio has been defined as lying between 1: 1 and 1:2, but adequate nutrition is possible outside of these limits." The general statement may be made that the harmful effects of vitamin D deficiency do not develop so soon when the calcium phosphorus ratio is between 1:2 and 2:1. Calves and lambs rarely suffer from calcium under nutrition on ordinary rations and are not so sensitive as colts to an abnormal calcium-phosphorus ratio, but they are affected if the amount of phosphorus is inadequate. Rickets may or may not be due to a simple vitamin D deficiency. An adequate supply of vitamin D cannot compensate for a faulty proportion or inadequate amounts of calcium and phosphorus in the diet. How vitamin D acts is not entirely clear, but in cases of rickets where vitamin D deficiency is the limiting factor the results of administering it are very specific and dramatic. Almost immediately there is an incidence case in the retention of calcium and phosphorus- in the body, probably involving both increased absorption and reduced excretion. Blood phosphorus and calcium return to normal, and new deposits of bone salts can be demonstrated within a few hours. Direct exposure of the body to sunlight has the same effect on calcium and phosphorus metabolism as vitamin D in the ration, but the effectiveness of sunlight in curing and preventing.

D. Urolithiasis

D.1. Introduction

Obstructive urolithiasis means the formation of calculi in the urinary tract with subsequent urinary blockage by uroliths. It appears to affect equally both sexes, but urinary blockage is an important problem only in males. Steers are most commonly affected by the obstructive form of the disease because of the anatomical conformation of their urinary tract. Urinary calculi formation usually results from a combination of physiologic, nutritional and management factors. It is mainly attributed to excessive or imbalanced intake of minerals. These circumstances occur especially in feedlots with fattening cattle receiving rations high in cereal grains and oil meals. These feedstuffs contain phosphorus and magnesium in excess, but relatively low levels of calcium and potassium, predisposing to occurrence of the disease. A calcium-phosphorus imbalance results in high urinary phosphate excretion which is an important factor in the genesis of phosphate calculi. Numerous additional factors have been incriminated as contributing

causes for the development of phosphate calculi with resultant obstructive urolithiasis in cattle. These include heavy concentrate-low roughage diets, limited water intake, deprivation of water or dehydration, urine alkalinity, mineralized artesian water, alkaline water supplies, excess of sodium bicarbonate in the diet, vitamin imbalances e. g. hypovitaminosis A and Hypervitaminosis D, and high-protein rations. Less frequent types of uroliths include those composed of silica, carbonates or oxalates. Livestock grazing in areas in which pasture plants contain large quantities of oxalate, estrogens or silica are prone to develop these types of calculi. Urolithiasis in castrated beef cattle associated with diethylstilbestrol implants was reported in only one single occasion.

Fatality rate of urolithiasis in male cattle due to rupture of the urethra or urinary bladder and the economic impact of this condition has been extensively reported in field and slaughterhouse studies developed in many countries. Occurrence of calculi has been associated with lower weight gain and lighter carcasses. Similar information is not available for Brazilian feedlot cattle herds. Obstructive urolithiasis has been infrequently reported in beef cattle in south-eastern and central Brazil. Relevant data concerning composition of basal diet, mineral constitution of urinary calculi and influence of the disease on weight gain and carcass characteristics at slaughter are not available. Effective prevention measures of this metabolic disorder in these areas rely on knowledge of the major causative factors of the disorder and the circumstances in which they occur.

D.2. Prevention

Recurrence is a major problem with calcium oxalate uroliths. An “ideal” diet is considered to be low oxalate, low protein, and low sodium and would maintain urine pH at 6.5–7.5 and urine specific gravity <1.020. A few commercially available canned foods achieve these goals and may minimize the risk of recurrence. Potassium citrate may be added as needed to assure the urine pH is within the desired range; water may be used to provide appropriate reduction in urine concentration. If these urine conditions are achieved and calcium oxalate crystals are still seen in warm, fresh urine, then vitamin B_6 and/or thiazide diuretics can be considered. Effectiveness of therapy should be reevaluated at 1- to 4-mo intervals by urinalysis.

E. . Osteomalacia and Osteofibrosis in Relation to Phosphorus and Calcium Deficiency

Three abnormal bone conditions are characterized by rather definite alterations in the conformation of the skeleton. Several factors, among them phosphorus deficiency, calcium deficiency, an abnormal proportion of these two minerals in the ration, vitamin D deficiency, and an altered rate of secretion of certain endocrine glands such as the parathyroid, may be involved in the production of these changes, A coexisting protein and vitamin A deficiency may also be involved in some cases.

E.1. Osteomalacia

Osteomalacia is a disease of adult animals named from one of its characteristic symptoms—softening and replacement of bone with osteoid tissue, which resembles uncalcified bone. In some respects this condition is similar to rickets and has been referred to as adult rickets, but it occurs after growth in the length of the bones has largely ceased. When the condition is caused by a deficiency of phosphorus in the animal's ration, it is usually called phosphorus deficiency. The disease is common in pregnant or lactating cows, particularly on phosphorus-deficient range or during periods of drought. It is also seen in sheep, goats, swine, horses, and mules. Calcium and vitamin D deficiency may also be involved. Osteoporosis is a similar condition resulting from faulty bone metabolism, except that the changes in the bone are those of atrophy, producing a thin porous structure and a failure of normal bone regeneration, but without the osteoid tissue seen in Osteomalacia.

E.2. Osteofibrosis

Osteofibrosis is characterized by enlargement and partial replacement of the bones with soft, poorly calcified fibrous tissue which may also occupy the marrow cavity. The bones of the face and jaws especially become enlarged, and the condition in horses is commonly known as bighead. Goats and swine are similarly affected. The animals are easily fatigued and may have a snuffling respiration, and they often have enlargements of the leg bones. The supply of phosphorus and protein was low on the natural range where they had been grazing, and phosphorus deficient animals often chew hones and other non-food materials apparently in an effort to remedy the deficiency. Lameness, stiffness of gait, fragility of bone, and enlargement of the bones of the face and jaw may occur in horses, swine, and goats. Bony enlargements are frequently due to osteofibrosis. Animals receiving an insufficient supply of calcium may also develop fragile bones, and fail to reproduce and lactate normally. This condition is very rare, however, and does not usually occur if good hay or pasture forage of normal calcium content is available.

- **Cause, prevention, and treatment**

Diseases caused by abnormal phosphorus and calcium metabolism due to errors in diet can be prevented or treated in any one of three ways:

- ✓ By using natural feeds that contain sufficient quantities of calcium and phosphorus;
- ✓ By increasing the calcium and phosphorus content or the yield of pasturage or hay by fertilizing the soil on which the crops are grown; and By feeding a specific mineral supplement.
- ✓ Feeds low in phosphorus and requiring supplementation if they make up a large proportion of the ration are beet pulp, molasses, corn fodder, prairie hay, sorgo hay and fodder, and legumes and grasses grown on phosphorus-deficient soils, especially during dry seasons.

- ✓ Feeds rich in phosphorus are wheat bran; whole cereal grains; tankage; cottonseed, linseed, peanut, and soybean meals; and hays or other herbage from phosphorus-rich soils.
- ✓ Feeds rich in both calcium and phosphorus are steamed bone meal, skim milk, and buttermilk.
- ✓ When they contain bone, tankage, meat scrap, and fish meal also supply considerable calcium as well as phosphorus.
- ✓ High-quality ground limestone (calcium carbonate) and oyster-shell are excellent sources of calcium.

F. <u>Hyperkeratosis</u>

Epithelial cells accumulate on the skin as a result of excessive keratinization of epithelial cells and intercellular bridges, interference with normal cell division in the granular layer of the epidermis, and hypertrophy of the stratum corneum.

- **Local hyperkeratosis may be caused by the following**:

- ✓ Mechanical stress on pressure points (e.g., elbows, hocks, or brisket) when animals lie habitually on hard surfaces
- ✓ Mechanical and/or chemical stress (e.g., **teat-end keratosis** of dairy cows that can be caused by improper milking machine settings, over milking, improper use of teat sanitizers or cold weather)
- ✓ Parasitism (e.g., hyperkeratotic form of sarcoptes mange of pigs and small ruminants)

- **Generalized hyperkeratosis may be caused by the following**:

- ✓ Poisoning with highly chlorinated naphthalene compounds
- ✓ Chronic arsenic poisoning
- ✓ Inherited congenital ichthyosis
- ✓ Inherited dyserythropoiesis–dyskeratosis
- ✓ Infection with *Scopulariopsis brevicaulis*, a fungus, was recently associated with generalized hyperkeratosis in a calf and a goat kid.
- ✓ The skin is dry, scaly, and thicker than normal, usually corrugated, hairless, and fissured in a grid like pattern.
- ✓ Secondary infection of deep fissures may occur if the area is continually wet.
- ✓ However, the lesion is usually dry, and the plugs of hyperkeratosis material can be removed, leaving the underlying skin intact.

Confirmation of the diagnosis is by the demonstration of the characteristically thickened stratum corneum in a biopsy section, which also serves to differentiate the condition from parakeratosis and inherited ichthyosis.

Primary treatment depends on correction of the cause. Supportive treatment is by the application of a keratolytic agent (e.g., salicylic acid ointment)

G. Parakeratose

G.1. Definition

Parakeratosis is a zinc-responsive dermatosis usually observed in 2- to 4-month-old swine. Pigs not allowed access to soil or not supplemented with zinc are more likely to have parakeratosis.

G.2. Cause

The disease is caused by a relative deficiency of zinc. The deficiency is usually caused by feeding an unbalanced diet that has one or more of the following features: excessive calcium; excessive phytic acid (sometimes present in soybean protein); or a low concentration of essential fatty acids. These features all adversely affect availability of dietary zinc. In addition, enteric pathogens or changes in intestinal flora can adversely influence zinc absorption. Parakeratosis most often is caused by consumption of excessive calcium.

G.3. Signs

Affected pigs show few signs of illness other than skin lesions and reduced growth rate. Initial lesions appear as reddened macules and papules on the ventrolateral abdomen and medial surface of the thighs; these lesions often go unobserved. The lesions are slowly covered by thick, roughened scales and crusts. More obvious lesions soon become apparent on the lower legs and on the dorsum. Lesions sometimes can be seen around the eyes, ears, snout and tail and eventually may become generalized. Affected areas of the skin are hyperkeratotic and there may be fissuring of the epidermis with secondary infection of the fissures. A unique feature occasionally seen is a focal or diffuse hyperkeratosis on the tongue. Parakeratosis is a microscopic feature of affected epidermis and gives this dermatosis its name.

G.4. Treatment

Pigs with parakeratosis will recover if excessive calcium is removed from the ration and it is properly supplemented with zinc. Most good commercial or carefully compounded rations now are supplemented with adequate levels of zinc salts. Although once common, parakeratosis seldom occurs today unless a feed mixing error occurs.

H. Goiter

A. Introduction

Enlargement of the thyroid gland is a common manifestation of iodine deficiency in domestic animals. It is an advanced symptom, however, and the chief loss from iodine deficiency is from interference with reproductive processes and the birth of weak, deformed offspring which often fail to survive. The gestation period of mares receiving insufficient iodine is frequently longer than normal, and the foal is either still-born or so weak that it is unable to get up and nurse normally. Most such colts die within a few days, although some survive and make a complete recovery.

Most such calves are alive at birth, although a few may be still-born; some are weak and die within a few days; others have approximately normal vigour and are not noticeably affected, except for enlargement of the thyroids, which may or may not cause difficult breathing because of pressure on the trachea. If the calf is able to take nourishment, the goitre frequently diminishes in size until it is no longer noticeable, but sometimes it remains throughout adult life. In severe cases the hair of the calf may be thinner than normal or the animal may be almost completely hairless. Sows may give birth to weak pigs which are often more or less hairless and may be still-born or die within a few hours. Some of the pigs in a litter may be more seriously affected than others. Thyroid enlargement may be present but is often overlooked unless a dissection is made. The skin of the abnormal pigs is often thick and pulpy, especially over the shoulder and neck region, owing to a watery swelling (oedema) similar to that seen in human beings suffering from hypothyroidism. Ewes receiving insufficient iodine also give birth to weak lambs which often show thyroid enlargement and may be partially wool less. The death rate among such lambs is very high. A lamb with the skin removed to show an enlarged thyroid gland (goitre), a typical result of iodine deficiency.

B. <u>Prevention and treatment of goiter</u>

The effectiveness of supplementary feeding of iodine to prevent goiter and associated symptoms due to iodine deficiency has been conclusively demonstrated for all classes of farm animals, but the exact minimum requirement of iodine has not been established. Salt of this iodine content can be purchased from feed dealers, or salt containing approximately the same iodine content can be prepared by thoroughly mixing 23.8g of powdered potassium iodide with 135kg of granulated stock salt. The iodized salt should be made available to sows during at least 3 months of the pregnancy period, to ewes for 3 to 4 months. If goiter appears in animals born during midsummer or fall, iodized salt in place of ordinary salt should be fed at least to breeding animals, throughout the year. The need of feeding iodine to other than pregnant animals in iodine-deficient areas is questionable. Iodine compounds are expensive, however, and they have toxic effects on animals if fed in large amounts. Where extra iodine is needed, the amounts required are so small that the increase in feed costs is not appreciable. Control of goiter and related conditions in animals—at one time a serious threat to the livestock industry in some areas by feeding

iodine compounds is so successful that economic losses from this ;condition should be very small if proper precautions are taken.

- Content/Topic 5: Vitamins nutritional disorders

A. Vitamins

Vitamins, a group of complex organic compounds that are present in minute amounts in natural foodstuffs, are essential for normal metabolism, and a lack of these compounds in the diet causes deficiency diseases. Some vitamins are synthesized by intestinal tract bacteria in quantities that are often adequate for bodily needs and others are food origin.

Classical deficiency symptoms and non-specific parameters (e.g. lower production and reproduction rates) are associated with vitamin deficiencies or excesses. Vitamin nutrition should no longer be considered important solely for preventing deficiency signs, as vitamins can play a role in optimizing animal health (e.g. immune function), productivity and product quality.

A.1.Vitamin Poisoning or Hyper vitaminosis

Hypervitaminosis is a pathological condition resulting from excessive intake of vitamins which can lead to toxicosis. Under natural condition it is unlikely to receive excessive doses of vitamins. Generally, toxic levels of vitamins are achieved through high supplement intake. Water soluble vitamins rarely deliver dangerous levels, with few exceptions like vitamin C and some of B complex vitamins. Hyper vitaminosis usually occur with fat soluble vitamins, which are stored in the liver and fatty tissue of the body. These vitamins build up and remain for longer time in the body than water soluble vitamins. Toxicities of fat soluble vitamins can occur due to large intake of fortified foods. Most commonly the vitamins associated with toxicity are vitamin A, D, E, K and vitamin C.

B.2.Vitamin deficiency or Hypo vitaminosis

Vitamin deficiencies are most commonly due to inadvertent omission of a vitamin premix from the birds' diet. Multiple signs are therefore seen, although in general, problems with deficiencies of the B vitamins appear first. Because there are some stores of fat-soluble vitamins in the body, it often takes longer for these deficiencies to affect the bird. In ruminant diseases, only fat soluble vitamins A, D and K have real importance.

- **Vitamin A deficiency**

This is available in most green plants, and if the animals graze on well managed pastures and forage, deficiencies will not occur. However, cattle fed on poor quality roughage, such as poor quality hay and straw, require supplementation.

- ✓ **Clinical Signs of Vitamin A Deficiency**
  - Decreased appetite leading to reduced growth.
  - Impaired night vision.
  - Increased still births in pregnant animals due to affected reproductive function, especially in cases where dry cows are offered poor diets.
  - Fainting fits in calves: the calf collapses as if in a deep sleep then gets up and walks away quite normally.
  - In latter stages of deficiency, bone growth is affected and this may cause pressure on nerves to the eye, which may lead to total blindness.
- ✓ **Diagnosis of Vitamin A Deficiency**

This can be done by investigating the history of animals and their diets, and by analysis of blood and liver samples in a laboratory.

- **Vitamin D deficiency**

There is little vitamin D in plants. Animals obtain most of it from the sun. Vitamin D is necessary for the absorption of calcium and phosphorous from the intestines and the deposition of the minerals in bone as well as in the maintenance of normal blood levels. Vitamin D deficiency in young calves is likely to occur when they are housed in dim lights and offered poor quality diets.

- ✓ **Clinical Signs of Vitamin D Deficiency**
  - Reduced Growth Rates.
  - The legs may be bent and there is abnormal swelling, with stiffness and lameness occurring in a number of animals.
  - The teeth may be out of line and the jaw bone deformed.
- ✓ **Treatment**

Treatment is by injecting vitamin D and by correcting the ration, including oral supplementation with vitamin D.

- **Vitamin K deficiency**

This is available in plenty in leafy forages. Primary deficiency does not occur. Deficiency can be induced by dicoumarol poisoning such as warfarin rat poison and mouldy clover hay, which inhibit the action of vitamin K. Vitamin K is involved in blood-clotting mechanisms.

- ✓ **Clinical Signs of Vitamin K Deficiency:**
  - Failure of blood clotting, including excessive bleeding from cuts
  - Appearance of large red haemorrhagic areas in the membranes of the mouth, eyes and nose.
  - Abdominal pain and lameness.

- ✓ **Treatment and Prevention:**
  - Identify and remove the source of poison.
  - Give Vitamin K by mouth or through injection.

- **B and C Vitamins deficiency**

This group of vitamins is formed by micro-organisms in the rumen and any excess is absorbed by the cow. They are also present in ample quantities in milk and therefore primary dietary deficiency is never seen.

- ✓ **Vitamin C**

This is produced in tissues of all farm livestock (cattle, sheep and goats) and dietary supply is unnecessary.

- **Treatment and prevention**

Treatment and prevention of vitamin deficiencies rely on an adequate dietary supply, usually microencapsulated in gelatine or starch along with an antioxidant. Vitamin destruction in feeds is a factor of time, temperature, and humidity. For most feeds, vitamin efficiency is little affected over 2-mo storage within mixed feed.

- Content/Topic 6: Drinking and feeding errors

A. Introduction

In all animals, the body water pool must remain reasonably constant in the long term, although livestock adapted to arid areas may be able to tolerate fairly large short-term fluctuations. As already suggested, animals drink primarily to replace lost fluid, rather than in anticipation of future needs. Thus water loss largely dictates water gain, and will therefore be dealt with first.

A.1. Dehydration

Dehydration is an imbalance of water and electrolytes (minerals) in the body and can cause serious complications for livestock animals. Water is essential to animals, which depend on proper daily fluid intake to maintain appropriate health and replace fluids that are routinely lost through urine, feces and respiration.

Under African ranching conditions, livestock use 5 to 30% of their total body water pool per day. This loss is reduced to as little as 1.5% in dehydrated, arid-adapted animals such as the camel.

A.2. Water hematuria

- **What you need to know**:

Drinking water for calves should be availed throughout the day as calves deprived of water tend to consume more than required resulting in bloody urine.

- **Blood in cow's urine**

What causes a cow to urinate blood?

Red urine or hematuria in cows may be brought about by several reasons. It may be caused by tick-borne disease called babesiosis.

It could also be as are result of ingestion of poisonous plants such as Bracken fern, or bacterial infections of the kidneys and bladder.

Drinking water for calves should be availed throughout the day as calves deprived of water tend to consume more than required resulting in bloody urine. It is important that you call a qualified veterinarian to examine your cow and determine the cause of bloody urine. The vet would then manage the condition. Tick borne diseases are treatable but are best controlled by spraying your animal's weekly using acaricides which are available in agro vets.

- Content/Topic 7:Digestives disorders

Determination of the cause of intestinal disease in cattle is based on clinical, epidemiologic, and laboratory findings. Nonspecific therapy includes oral and parenteral fluid therapy to restore the fluid, electrolyte, and acid-base homeostasis. Specific therapy and prevention are detailed under the individual disease headings.

A. Constipation

Constipation is the infrequent or difficult evacuation of faeces, which are typically dry and hard. Constipation is a common clinical problem in small animals. As faeces remain in the colon longer, they become drier, harder, and more difficult to pass. **Obstipation** is intractable constipation, characterized by an inability to evacuate the mass of dry, hard faeces; impaction extending from the rectum to the ileocolic valve can result. Mega colon is a pathologic condition of hypo motility and dilation of the large intestine that results in constipation and obstipation.

A.1. Etiology and Pathophysiology

Peristaltic waves are responsible for the aboral movement of fecal material in the colon. Giant migrating waves that occur intermittently throughout the day move this matter farther and more rapidly. These waves constitute the "gastrocolic reflex" and are common after ingestion of a meal. A reduction or loss of these waves may contribute to constipation. Similarly, an increase in segmentation wave activity may predispose to constipation. However, diet is the most important local factor affecting colonic function.

A.2. Clinical Findings

The classic clinical signs of constipation are tenesmus and the passage of firm, dry feces. If the passage of feces is hindered by an enlarged prostate or sub lumbar lymph nodes, the feces may appear thin or "ribbon-like" in appearance. Abdominal palpation and rectal examination can confirm the presence of large volumes of retained fecal matter. Passed feces are often putrid. Some animals are quite ill and also have lethargy, depression, anorexia, vomiting (especially in cats), and abdominal discomfort.

A.3. Treatment and Control

Affected animals should be adequately hydrated. Mild constipation can often be treated by dietary adjustment consisting of avoidance of dietary indiscretion, ready access to water and high-fiber diets, and the use of suppository laxatives. Continued or long term use of laxatives should be discouraged unless absolutely necessary to avoid constipation.

- Laxatives are classified as bulk-forming, lubricant, emollient, osmotic, or stimulant types. Most act on fluid transport mechanisms and colonic motor stimulation. They should be avoided in the presence of dehydration.
- Mineral oil and white petroleum are lubricant laxatives that impede colonic water absorption and permit greater ease of fecal passage. These effects are moderate, and lubricant laxatives are beneficial only in mild cases of constipation. Mineral oil use should be limited to rectal administration because of the risk of aspiration pneumonia with oral administration.

B. Diarrhea

Diarrhea is caused by one of four major mechanisms—secretion, increased osmotic load, inflammation, or hyper motility of intestines. Increased secretion of fluid beyond the absorptive ability of the intestine is best exemplified by the diarrhea seen in neonates infected with enterotoxigenic *E. coli*.

Chronic diarrhea and wasting often in combination with good appetite, occurring as a sporadic disease in adult cattle is typical for paratuberculosis. Chronic diarrhea and wasting also occurring in younger animals may be caused by chronic salmonellosis and chronic BVD infection. Other possible causes of chronic diarrhea include congestive heart failure, uremia, and chronic peritonitis. Diarrhea may also accompany selenium-responsive ill-thrift syndromes in growing cattle.

Diarrhea may follow cases of simple indigestion and is common in grain overload. It also follows ingestion of toxic amounts of chemicals (e.g., arsenic, copper, zinc, and molybdenum) or certain poisonous plants and Mycotoxicoses; dipyridyl and organophosphate poisoning can also cause diarrhea.

C. Indigestions

Indigestion in ruminants can be classified as: Simple indigestion and vagal indigestion.

C.1. Simple Indigestion in Ruminants

Simple indigestion is a minor disturbance in ruminant GI function that occurs most commonly in cattle and rarely in sheep and goats. Simple indigestion is a diagnosis of exclusion and is typically related to an abrupt change in the quality or quantity of the diet.

- **Etiology**

Almost any dietary factor that can alter the intraruminal environment can cause simple indigestion. The disease is common in hand-fed dairy and beef cattle because of variability in the quality and quantity of

their feed. Dairy cattle may suddenly eat excessive quantities of highly palatable feeds such as corn or grass silage; beef cattle may eat excessive quantities of relatively indigestible, poor-quality roughage. Simple indigestion is usually associated with a sudden change in the pH of the ruminal contents, such as a decrease in ruminal pH due to excessive fermentation or an increase in ruminal pH due to putrefaction of ingested feed. It can also result from the accumulation of excessive quantities of relatively indigestible feed that may physically impair rumen function.

- **Clinical Findings**

The clinical signs depend on the type of animal affected and cause of the disorder.

- ✓ Silage overfeeding causes anorexia and a moderate drop in milk production in dairy cattle.
- ✓ The rumen is usually full, firm, and doughy; primary contractions are decreased in rate or absent, but secondary contractions may be present although usually decreased in strength.
- ✓ Temperature, pulse, and respiration are normal.
- ✓ The feces are normal to firm in consistency but reduced in amount.
- ✓ Recovery usually is spontaneous within 24–48 hr.

- **Treatment**

The diagnosis is confirmed by collection and examination of ruminal fluid, which may have an abnormal pH (<6 or >7), decrease in the numbers and size of protozoa, or prolonged methylene blue reduction time (a measure of bacterial activity).

Treatment is aimed at correcting the suspected dietary factors. Spontaneous recovery is usual when animals are fed a typical ruminant diet. Administration of ~20 L of warm water or saline via a stomach tube, followed by vigorous kneading of the rumen, may help restore rumen function in adult cattle. Magnesium hydroxide PO may be useful when excessive amounts of grain have been ingested, but magnesium hydroxide should only be administered to cattle with low ruminal pH (<6), otherwise excessive fore stomach and systemic alkalinisation can result.

C.2. <u>Vagus (Vagal) Indigestion</u>

Cattle affected with vagus indigestion develop progressive intermittent, then chronic abdominal distention. Improper forestomach emptying, due to a functional outflow problem, can be caused by damage to the ventral vagal trunk. Mechanical inhibition of motility from adhesions or abscesses can also decrease forestomach emptying.

- **Etiology and Pathogenesis**

Diseases that result in injury, inflammation, or pressure on the vagus nerve can result in clinical signs of vagal indigestion syndrome. However, vagal nerve damage is not present in most cases of vagus indigestion, and the most common cause is traumatic reticuloperitonitis. Conditions resulting in mechanical obstruction of the cardia or reticulo-omasal orifice (e.g., papilloma or ingested placenta) can

also result in vagal indigestion if ruminoreticular distention is present and the condition is sub-acute to chronic.

D. Bloating

Bloat is an overdistention of the rumenoreticulum with the gases of fermentation, either in the form of persistent foam mixed with the ruminal contents, called primary or frothy bloat, or in the form of free gas separated from the ingesta, called secondary or free-gas bloat. It is predominantly a disorder of cattle but may also be seen in sheep.

- **Etiology and Pathogenesis**

In **primary ruminal tympany**, or **frothy bloat**, the cause is entrapment of the normal gases of fermentation in stable foam. Coalescence of the small gas bubbles is inhibited, and intraruminal pressure increases because eructation cannot occur. Several factors, both animal and plant, influence the formation of a stable foam. Soluble leaf proteins, saponins, and hemicelluloses are believed to be the primary foaming agents and to form a monomolecular layer around gas rumen bubbles that has its greatest stability at about pH 6.0. Salivary mucin is antifoaming, but saliva production is reduced with succulent forages. Bloat is most common in animals grazing legume or legume-dominant pastures, particularly alfalfa, ladino, and red and white clovers, but also is seen with grazing of young green cereal crops, rape, kale, turnips, and legume vegetable crops.

In **secondary ruminal tympany**, or **free-gas bloat**, physical obstruction of eructation is caused by esophageal obstruction due to a foreign body (e.g., potatoes, apples, turnips, and kiwi fruit), stenosis, or pressure from enlargement outside the esophagus (as from lymphadenopathy or sporadic juvenile thymic lymphoma).

- **Clinical Findings**

Bloat is a common cause of sudden death. Cattle not observed closely, such as pastured and feedlot cattle and dry dairy cattle usually are found dead. In lactating dairy cattle, which are observed regularly, bloat commonly begins within 1 hour after being turned onto a bloat-producing pasture. Bloat may develop on the first day after being placed on the pasture but more commonly develops on the second or third day.

- ✓ In primary pasture bloat, the rumen becomes obviously distended suddenly, and the left flank may be so distended that the contour of the Para lumbar fossa protrudes above the vertebral column; the entire abdomen is enlarged. Death may occur within 1 hour after grazing began but is more common ~3–4 hour after onset of clinical signs.
- ✓ In secondary bloat, the excess gas is usually free on top of the solid and fluid ruminal contents, although frothy bloat may be seen in vagal indigestion when there is increased ruminal activity. Secondary bloat is seen sporadically. There is tympanic resonance over the dorsal abdomen left of

the midline. Free gas produces a higher pitched ping on percussion than frothy bloat. The distension of the rumen can be detected on rectal examination. In free-gas bloat, the passage of a stomach tube or trocarization releases large quantities of gas and alleviates distention.

- **Treatment**

In life-threatening cases, an emergency rumenotomy may be necessary; it is accompanied by an explosive release of ruminal contents and, thus, marked relief for the cow. Recovery is usually uneventful with only occasional minor complications. A trocar and cannula may be used for emergency relief, although the standard-sized instrument is not large enough to allow the viscous, stable foam in per acute cases to escape quickly enough.

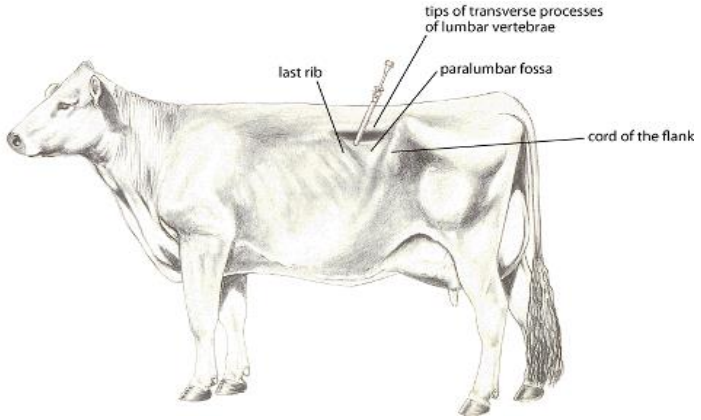

Figure 12: Treatment of bloat with trocar

- **Control and Prevention**

Prevention of pasture bloat can be difficult. Management practices that have been used to reduce the risk of bloat include feeding hay, particularly orchard grass, before turning cattle on pasture, maintaining grass dominance in the sward, or using strip grazing to restrict intake, with movement of animals to a new strip in the afternoon, not the early morning. Hay must constitute at least one-third of the diet to effectively reduce risk of bloat. Feeding hay or strip grazing may be reliable when the pasture is only moderately dangerous, but these methods are less reliable when the pasture is in the pre-bloom stage and the bloat potential is high. Mature pastures are less likely to cause bloat than immature or rapidly growing pastures.

Available antifoaming agents include oils and fats and synthetic non-ionic surfactants. Oils and fats are given at 60–120 mL/head/day; doses up to 240 mL are indicated during dangerous periods. The ultimate aim in control is development of a pasture that permits high production, while keeping incidence of bloat low. The use of pastures of clover and grasses in equal amounts comes closest to achieving this goal. To prevent feedlot bloat, rations should contain ≥10–15% cut or chopped roughage mixed into the complete feed. Preferably, the roughage should be a cereal, grain straw, grass hay, or equivalent.

E. Dehydration

E.1. Introduction

Water is the second-most important nutrient, behind oxygen for livestock. It accounts for 98 percent of all molecules in the body and between 50 and 81 per cent of an animal's total body weight at maturity. Mammals can only lose about 10 per cent of their water weight without risking extreme illness or the potential of death.

So, it's vital to ensure livestock and poultry have access to high quality and high volumes of water to ensure their wellness and productivity.

Water is critical to many body functions, including the following:

- Eliminate waste products of digestion and metabolism
- Regulate blood osmotic pressure
- Produce milk and saliva
- Transport nutrients, hormone and other chemical messages within the body

Aid in temperature regulation affected by evaporation of water from the skin and respiratory tract

Water also plays a role with tissue maintenance, eye development, thermal regulation, mineral homeostasis and body pressures.

E.2. Causes

Dehydration can be caused by poor management practices, such as not ensuring an adequate volume of water is flowing to water troughs and water nipples, or that water lines are plugged and not flowing properly, especially during warmer weather. Dehydration can also be caused by poor water quality, or animals not being trained on a new water source.

In the late part of the summer, water sources prone to runoff can become contaminated with blue-green algae. The algae impacts the taste of the water and animals will be less willing to drink. In the case of swine, it can actually make piglets feel sick, especially nursery pigs.

E.3. Dehydration Causes Production Challenges

Dehydration can have a negative effect on many production factors on a livestock operation. Dehydration can weaken tight junctions, gut integrity and reduce the strength of an animal's epithelial tissue. When this happens, inflammation issues may arise for the animal as they become more susceptible to disease and infections.

Animals that become dehydrated also suffer from poor production, including lower growth rates in livestock, and reduced milk production in dairy cattle. Dehydration usually leads to a decrease in feed intake, thus reducing growth rates in cattle, swine and poultry.

Water is a critical component of milk production, since water comprises 87 percent of milk. As a result, lactating dairy cows that become dehydrated will experience near cessation of milk production.

Dehydration in swine can result in salt poisoning, which can be fatal. A normal salt concentration in swine nutrition becomes toxic in the absence of water. Pigs affected by salt poisoning will be uncoordinated and have intermittent convulsions. The worst clinical signs and fatalities occur if water is given to pigs after a period of restricted water intake or dehydration. It's important to provide access to water sparingly until they are fully rehydrated.

Dehydration can especially be a problem for endurance animals, like racehorses, during warmer weather. When you put an animal through long endurance training or racing tactics, they can become dehydrated if they're not drinking enough water before or after those events. Just like in human athletes, this increases gut leakage and the breakdown of tight junctions on the epithelial barrier of the gut. This allows harmful bacteria, pathogens and breakdown products of specific bacteria called endotoxins into the bloodstream, which then causes inflammation and sets off another cascade of problematic events.

E.4. Visual Dehydration Symptoms

There are both physical signs and behavioral signs that can indicate if an animal is dehydrated. Common signs include lethargy, tightening of the skin, weight loss and drying of mucous membranes and eyes.

When dairy and beef cattle become dehydrated, the eyes will appear sunken and dull. As mentioned earlier, dehydration in lactating dairy cows results in a near cessation of milk production.

In horses, dehydration results in a reduction in skin elasticity. You can check this by pulling the skin over the shoulder and holding it for a moment. Release the skin and count the seconds until the fold disappears. If the horse is dehydrated, the skin will stand for several seconds.

In swine, signs of dehydration include thirst, constipation, skin irritation and lack of appetite, often followed by nervousness, apparent deafness and blindness.

E.5. Prevention

There are several things that livestock and poultry producers can do to ensure their animals stay hydrated, including the following:

- Clean water lines. This includes using bleach or hydrogen peroxide to clean bacteria in water lines and to help prevent clogging. Many livestock producers do not check their water lines until they aren't functioning properly. A diligent cleaning process is needed every year to make sure water lines are functioning and will continue to function properly.
- Check water flow. It is recommended that you have half a gallon (about 2 liters) per minute for lactating a sow. Flow rate and quantity can be less for young pigs, as low as a half a liter to 1 litter per minute. Make sure enough water is flowing to the troughs and to the water nipples. For example, sows in gestation need about four liters of water per day, so if you're getting a flow of at

least a half a liter to one liter per minute, that is adequate. On the other hand, sows in lactation can need as much as 10-15 liters per day for milk production. Be sure to increase water flow appropriately when needed.

- Ensure ventilation fans are working properly. By making sure your ventilation fans are working and effective at cooling your animals, you are improving the animals' utilization of water.
- Test water for palatability. Animals will drink more water when it is clean and tastes good.

F. Enterotoxemia

Enterotoxemia is a frequently severe disease of sheep and goats of all ages. It is caused by two strains of bacteria called *Clostridium perfringens* – the strains are termed types **C and D.** These bacteria are normally found in low numbers in the gastrointestinal tract of all sheep and goats. If that is so, when and why do they cause disease?

These organisms are normally "laying low" in the small and large intestine – that is, they are present in relatively low numbers and appear to be in a relatively quiescent state in the normal, healthy animal. What appears to trigger them to cause disease is a *change in the diet* of the animal. Most commonly, the change that triggers disease is an increase in the amount of grain, protein supplement, milk or milk replacer (for lambs and kids), and/or grass that the sheep or goat is ingesting. Collectively, these feeds are rich in starch, sugar, and/or protein. When

Unusually high levels of these nutrients reach the intestine; *Clostridium perfringens* undergoes explosive growth, increasing its numbers rapidly within the intestine. As the organism grows in number, it releases very potent toxins (bacterial poisons) that harm the animal. These toxins can cause damage to the intestine as well as numerous other organs. This can result in fatalities, particularly in the non-vaccinated animal or in the newborn lamb or kid whose dam has not been vaccinated.

Figure 13: Sheep with enterotoxemia

- **The signs of enterotoxemia in sheep and goats include**:

✓ The animals may abruptly go off of feed and become lethargic.

✓ Affected animals may show signs of stomach pain, such as kicking at their belly, repeatedly lying down and getting up, lying on their sides, panting, and crying out.

✓ Diarrhea may develop; in some cases, there is blood visible in the loose stool.

- ✓ Animals may lose the ability to stand, lie on their sides, and extend their legs, with their head and neck extended back over their withers. This posture is caused by the effects of the toxins on the brain. Death commonly occurs within minutes to hours after this sign is seen.
- ✓ Because enterotoxemia can progress so quickly, animals may be found dead with no previous signs of disease.

- **Treatment**

Treatment of enterotoxemia may not be successful in severe cases. Many veterinarians treat mild cases with analgesics, probiotics (gels or pastes with "good bacteria), oral electrolyte solutions, and antisera, which is a solution of concentrated antibodies that neutralize the toxins that these bacteria produce. More severe cases may require intravenous fluids, antibiotic therapy, and other types of supportive care, such as supplemental oxygen.

- **Prevention**

Prevention of enterotoxemia is far more likely to be successful than trying to treat the disease.

- **Vaccination**

Vaccination is the cornerstone to prevention of this disease. For sheep and goats, there are multiple vaccines available that induce immunity to the toxins generated by *Clostridium perfringens* types C and D.

G. <u>Vomiting</u>

Vomiting is the reflexive, forceful expulsion of part or all of the contents of the stomach and proximal small intestines through the mouth. It involves a series of involuntary spasmic movements requiring visceral, diaphragmatic, and abdominal muscular contractions. The act of vomiting is usually preceded by a number of stereotypical prodromal clinical signs, including excessive salivation, repeated swallowing, retching, and marked contractions of the abdominal musculature.

Vomiting must be differentiated from regurgitation, dysphagia (difficulty swallowing), and various forms of esophageal dysfunction. **Regurgitation** is a passive process whereby partially or completely undigested food or liquid is released from the esophagus and/or stomach through the mouth without effort or muscular contractions (i.e., through gravity and body position).

- **Etiology, Pathophysiology, and Clinical Findings**

The reflex act of vomiting is initiated by the vomiting (or emetic) center, located in the reticular formation of the medulla. The vomiting center is responsive to input from 4 major sources:

- ✓ Afferent receptors from a variety of peripheral structures (the GI tract, pancreas, heart, liver, urogenital tract, and peritoneum) carried by the vagus and sympathetic nerves;
- ✓ The chemoreceptor trigger zone (CRTZ) of the area postrema of the medulla;
- ✓ The cerebral cortex and limbic system; and

- ✓ The vestibular apparatus. In this way, the vomiting centre is responsive to neural, humoral, and chemical input.

Anxiety, depression, hyper salivation, and repeated swallowing accompanied by relaxation of the gastro oesophageal sphincter are followed by retching. Forceful contractions of the abdominal muscles and diaphragm against a closed glottis combined with increases in intra-abdominal pressure force expulsion of food, fluid, or debris.

- **Diagnosis**

Diagnosis begins with accurate and complete identification of the problem. This includes differentiating vomiting from regurgitation or dysphagia, and then characterizing the duration and character of the vomiting. Following a complete history and physical examination, primary and secondary GI differential diagnoses should be considered.

- **Treatment and Control**

The primary goal of treatment in a vomiting patient is to identify and treat the underlying cause of the vomiting. Antiemetic therapy is an essential component of the plan for many vomiting patients, but with administration of an antiemetic, the clinician losses the ability to correlate the cessation of vomiting to any other specific parameter (i.e., rehydration, resolution of a partial obstruction, effective treatment of an underlying disease process, the natural progression of simple gastritis).

Drugs that act directly on the vomiting centre include the phenothiazine tranquilizers such as prochlorperazine and chlorpromazine.

Learning Unit 2 – Perform diagnosis of intoxications, nutritional and metabolic diseases

LO 2.1 – Perform clinical diagnosis for intoxications

- Content/Topic 1: Anamnesis and specific examination

A. Anamnesis

Diagnosis of poisoning is based on history, signs, tissue changes, and laboratory examinations. Giving your veterinarian a complete history is important to help him or her makes an accurate diagnosis and begins appropriate treatment. You should have the following information available:

- Sex, age, weight, and number of exposed or sick animals;
- A list of signs of illness in the order they appeared;
- Any prior disease conditions;
- Any medications the animal is receiving;
- Possible related events, for example, change in diet or water source, other medications, feed additives, or pesticide applications;

- Description of the environment, including access to garbage, machinery, or vehicles; and
- Recent past locations and when moved (if applicable)

B. **Specific examination**

B.1. Acute toxicity (LD50) test

Acute systemic toxicity evaluates the adverse effects that occur following exposure of organisms to a single or multiple doses of a test substance within 24 hours by a known route (oral, dermal or inhalation) . After administration, the test substance is absorbed and distributed to various parts of the body before it elicits systemic adverse effect.

The LD50 (median lethal dose) test was introduced in 1927 by J. W. Trevan to estimate the dose of a test substance that produces 50% death in a given species of animals. It is usually the first test conducted for every chemical before further toxicity tests are carried out. Although its major endpoint is death, non-lethal acute effect may occur as signs of toxicity depending on the chemical being tested.

Assessment of the acute toxic potential of substances is required to determine their adverse effects that might occur due to accidental or deliberate short-term exposure

From the result of an acute toxicity test, a conclusion can be made on the toxicity status of the test substance. As depicted substances with LD50 below 5 mg/ kg are classified to be highly toxic while substances with LD50

- 5,000-15,000mg/kg
- Practical non toxic
- Above 15,000mg/kg are termed relatively harmless

LO 2.2 – Perform diagnosis for nutritional and metabolic diseases

- Content/Topic 1:Anamnesis and Specific examination

A. Introduction

The difference between production--related metabolic diseases and nutritional deficiencies is often subtle. Typically, nutritional deficiencies are long term conditions that develop gradually and can be corrected through dietary supplementation. Metabolic diseases usually begin suddenly and respond dramatically to administration of the deficient nutrient (although affected animals may need dietary supplements to avoid recurrence). Because production-related metabolic disorders are serious and develop suddenly, accurate and rapid diagnosis is essential. Ideally, diagnostic tests can be used to predict the chance of disease occurring so that either it can be prevented or preparations can be made for rapid treatment.

B. Anamnesis

Anamnesis is the medical or developmental history of a patient. In case of metabolic and nutritional diseases, a collection of information on occurrence, previous cases, feeding system and so on, should be performed. This collected information will differ from the type of the disease to another.

C. Specific examination

For each metabolic disease, specific examination will be performed. The following are some examples of specific examinations in metabolic diseases.

C.1. Bovine Ketosis

The clinical diagnosis of ketosis is based on presence of risk factors (early lactation), clinical signs, and ketone bodies in urine or milk. When a diagnosis of ketosis is made, a thorough physical examination should be performed, because ketosis frequently occurs concurrently with other peripartum diseases. Especially common concurrent diseases include displaced abomasum, retained fetal membranes, and metritis. Rabies and other CNS diseases are important differential diagnoses in cases exhibiting neurologic signs. Cow-side tests for the presence of ketone bodies in urine or milk are critical for diagnosis.

In a given animal, urine ketone body concentrations are always higher than milk ketone body concentrations. Trace to mildly positive results for the presence of ketone bodies in urine does not signify clinical ketosis. Without clinical signs, such as partial anorexia, these results indicate subclinical ketosis. Milk tests for acetone and acetoacetate are more specific than urine tests.

C.2. Milk fever, Hypocalcemia

There are 3 discernible stages of parturient paresis.

- During stage 1, animals are ambulatory but show signs of hypersensitivity and excitability.
- Cows in stage 2 are unable to stand but can maintain sternal recumbency. Auscultation reveals tachycardia and decreased intensity of heart sounds.
- In stage 3, cows lose consciousness progressively to the point of coma. They are unable to maintain sternal recumbency, have complete muscle flaccidity, are unresponsive to stimuli, and can suffer severe bloat.

Differential diagnoses include toxic mastitis, toxic metritis, other systemic toxic conditions, traumatic injury (e.g., stifle injury, coxofemoral luxation, fractured pelvis, spinal compression), calving paralysis syndrome (damage to the L6 lumbar roots of sciatic and obturator nerves), or compartment syndrome. Some of these diseases, in addition to aspiration pneumonia, may also occur concurrently with parturient paresis or as complications.

LO 2.3 – Perform prognosis

- Content/Topic 1: Favorable and Unfavorable prognosis

A. Prognosis

Prognosis is defined as an outcome of a given disease. It can also be defined as the prospect of survival and recovery from a disease as anticipated from the usual course of that disease or indicated by special features of the case.

A.1.**Favourable prognosis**

The prognosis may be **favourable**; when there are many chances to recover after the treatment.

A.2.Unfavouble prognosis

The prognosis may be unfavourable, when the treatment will not succeed.

As it has been described above, the prognosis for metabolic diseases varies also depending on the case.

A.3.Example:

- Hypocalcemic cows typically respond to IV calcium therapy immediately. Tremors are seen as neuromuscular function returns. Improved cardiac output results in stronger heart sounds and decreased heart rate. Return of smooth muscle function results in eructation, defecation, and urination once the cow rises. Approximately 75% of cows stand within 2 hour of treatment. Animals not responding by 4–8 hour should be re-evaluated and retreated if necessary. Of cows that respond initially, 25–30% relapses within 24–48 hour and require additional therapy.
- Ketosis cases occurring within the first 1–2 week after calving (type II ketosis) frequently are more refractory to therapy than cases occurring nearer to peak lactation (type I).

Learning Unit 3 – Apply treatment of intoxications, nutritional and metabolic diseases

LO 3.1 – Select appropriate treatment

- Content/Topic 1: Manage the acutely poisoned animals

A. Assess the condition of the animal.

- Is the animal seizing?
- Is the animal breathing?
- What is the animal's heart rate?
- What color are the animal's mucous membranes?
- Is the animal in shock?

- What is the core body temperature?
- Is there any evidence of hemorrhaging?
- What is the basic history of the exposure?
- What is the toxicant?
- How long ago was the exposure?
- How old is the animal?
- How much does it weigh?
- Find out this general triage information to be sure that adequate measures can be taken for stabilization and treatment. Once animal is stable, a more thorough medical history should be obtained including complete medical background of the animal, exact toxicant information (including brand name, generic name (especially if it is a medication), and active ingredients.

✓ **A more complete history of the exposure should also be obtained including**

- Exposure time,
- Amount of toxicant the animal was exposed to, and
- By what route was the animal exposed (orally, dermally, etc.) Ideally, a support staff member could be taking the history from the owner and phoning the APCC as soon as possible while the animal is being treated.

B. **Stabilization**

- Stabilization is a priority – treat the patient, not the poison!
- Be prepared to intubate the animal upon presentation.
- Not only will this secure an airway, but also it will help prevent aspiration in the event that gastric lavage is necessary.
- This may or may not be necessary in every case, but preparation is the key. Have oxygen and an AMBU bag standing by in case they are needed.
- It is best to try to insert an I.V. catheter upon presentation to allow access for administration of medications and fluids.
- Once the catheter is placed, draw blood (at least one 3cc EDTA tube and two serum tubes are ideal) for any diagnostic tests to be performed later.
- If possible these samples should be taken before any other meds are administered.
- Monitor the animal for any cardiovascular abnormality. Atropine at a dose of 0.02-0.04 mg/kg I.V. May be recommended for correction of bradycardia.

- Propranolol is the drug of choice for treating tachycardia, administered slow I.V. at a dose of 0.02-0.06 mg/kg in dogs and 0.04 mg/kg in cat.
- These drugs as well as detailed protocols for their administration (in case a veterinarian is not readily available) should be easily accessible in a crash cart.
- Control seizures. If an animal is seizing when it presents, controlling the seizures is a top priority.
- Drugs to have on hand for this purpose are diazepam and inhalant anesthetics such as Isoflurane, or halothane.
- It may be necessary to mask an animal down to get the seizures under control.

C. Principles of Therapy of Toxicosis

At initial examination, certain immediate, life-saving measures may be needed. Beyond this, treatment for toxicosis includes three basic principles:

1) Prevention of further absorption,

2) Supportive/symptomatic treatment, and

3) Specific antidotes.

C.1. Prevention of Further Absorption:

- **Topically**

Topically applied toxicants usually can be removed by thorough washing with soap and water; clipping of the hair or wool may be necessary. Emesis is of value in dogs, cats, and pigs if done within a few hours of ingestion. Emesis is contraindicated when the swallowing reflex is absent; the animal is convulsing; corrosive agents, volatile hydrocarbons, or petroleum distillates are involved; or risk of aspiration pneumonia is imminent. Oral emetics include syrup of ipecac (10–20 mL, PO in dogs) and hydrogen peroxide (2 mL/kg, PO). Apomorphine can be used in dogs parenterally at a dosage of 0.05–0.1 mg/kg.

- **Gastric lavage**

Gastric lavage, using an endotracheal tube and the largest bore stomach tube possible, is done on the unconscious or anesthetized animal. The head is lowered to a 30° angle, and 10 mL of lavage fluid (water or saline) per kg of body weight is gently flushed into the stomach and then removed. This process is repeated until returned fluid is clear. Cathartics and laxatives may be indicated in some instances for more rapid elimination of the toxicant from the GI tract. A gastrotomy or rumenotomy may be necessary when lavage techniques are insufficient (or too slow in ruminants).

When the toxicant cannot be physically removed, certain agents administered orally can adsorb it and prevent its absorption from the alimentary tract. Activated charcoal (1–2 g/kg) effectively adsorbs a wide

variety of compounds and usually is the adsorbent and detoxicant of choice when toxicosis is suspected. The maximum amount of a drug adsorbed by activated charcoal is ~100–1,000 mg/g of charcoal. Sorbitol is sometimes added to activated charcoal to increase its palatability (in people) and to increase the GI transit time and flush out charcoal-bound toxins more rapidly. Activated charcoal should not be used in animals with known hypersensitivity or allergy to the drug. With administration of high doses, vomiting, constipation, or diarrhea may occur, and feces will appear black.

- **Supportive Therapy**:

Supportive therapy is often necessary until the toxicant can be metabolized and eliminated. The type of support required depends on the animal's clinical condition. Supportive efforts may include control of convulsive seizures, maintenance of respiration, treatment for shock, correction of electrolyte imbalance and fluid loss, and control of cardiac dysfunction, as well as alleviation of pain.

- **Fluid Therapy**

Fluid therapy is one of the cornerstone therapies of emergency management for the poisoned patient. Fluid therapy is warranted in the poisoned patient to help with the following

- ✓ To maintain perfusion at a cellular level
- ✓ To correct or prevent dehydration
- ✓ To aid in detoxification by increasing renal excretion of toxicants by forced diuresis
- ✓ To vasodilate the renal vessels (particularly with nephrotoxicants such as nonsteroidal anti-inflammatory drugs, lilies, grapes, raisins)
- ✓ To correct electrolyte imbalances
- ✓ To treat hypotension (particularly with toxicants like β-blockers, calcium channel blockers, angiotensin-converting enzyme (ACE) inhibitors that may result in profound decreases in cardiac output.
- ✓ To treat hypoproteinemia secondary to protein loss (e.g., long-acting anticoagulants) with the use of synthetic colloids (e.g., hydroxyethyl starch) (Please see the article "Fluid therapy: Crystalloids, Colloids and Albumin Products" elsewhere in this issue for more information.)
- ✓ To treat decreased oxygen delivery with blood or plasma transfusions if indicated (e.g., anemia secondary to GI blood loss from NSAIDs or coagulopathy from long-acting anticoagulants)
- ✓ In general, a balanced, maintenance, isotonic crystalloid (e.g., Lactated Ringers Solution, Normosol-R) can be used.
- ✓ In a healthy patient, fluid rates of 4 to 8 mL/kg/h can be used to force renal clearance of the toxicant.
- ✓ Neonates have a higher maintenance fluid rate (80–100 mL/kg/d), and fluid rates should be adjusted accordingly.

- ✓ Patients with cardiac disease, respiratory disease, or those who have ingested toxins that may increase the patient's risk toward pulmonary edema (e.g., tricyclic antidepressants [TCA], phosphide rodenticides) should have judicious fluid administration tailored to the patient's condition.
- ✓ Patients treated with fluid therapy should be appropriately monitored for hydration status by assessing for weight gain (or loss), hemodilution (PCV/TS), azotemia (for evidence of prerenal azotemia), or urine specific gravity while on IV fluid therapy.
- ✓ Evidence of hypersthenuria (cat >1.040; dog >1.025) in the hospitalized patient on fluid therapy is consistent with continued dehydration, and aggressive fluid therapy is warranted.
- ✓ The use of artificial colloids (e.g., hydroxyethyl starch) should be considered for those patients that have a low colloid osmotic pressure (normal reference range 18–20 mm Hg).
- ✓ Colloids are large molecules that stay in the intravascular space for a long time (i.e., they do not easily sieve across the vascular membrane).
- ✓ In general, patients that are hypoproteinemic (TS <6 g/dL) or persistently hypotensive may benefit from the addition of a colloid (e.g., hydroxyethyl starch boluses at 5 mL/kg, followed by a constant rate infusion (CRI) of 1 mL/kg/h).

- **Specific Antidotes:**

- ✓ Specific antidotes for various toxicants work by various mechanisms.
- ✓ Some complex with the toxicant (e.g., the oximes bind with organophosphorous insecticides, and EDTA chelates lead).
- ✓ Others block or compete for receptor sites (e.g., vitamin K competes with the receptor for coumarin anticoagulants).
- ✓ A few affect metabolism of the toxicant (e.g., nitrite and thiosulfate ions release and bind cyanide)

LO 3.2 – Prescribe medicines

- Content/Topic 1: Antidotes

A. **Antidotes**

An antidote is a medicine or other remedy for counteracting the effects of poison, disease, etc., which is very broad and would cover all therapeutic agents used to treat poisoning.

A.1. **An antidote can act in a number of ways. Examples include**:

- Limiting absorption
- Sequestering the poison
- Inhibiting metabolism to a toxic metabolite
- Promoting distribution from tissues

- Displacing the poison from a receptor or competing for the receptor
- Counteracting the toxic effect
- Enhancing detoxification.

A.2. **Important facts to bear in mind about antidotes include the following:**

- Administration of an antidote does not obviate the need for supportive and symptomatic treatment. On the contrary, supportive treatment may still mean the difference between survival and death.
- Poisonings for which there are antidotes are not necessarily less serious than those for which there is no antidote, and do not necessarily carry a better prognosis. If anything, the opposite is true; antidotes have been developed for some poisons because those poisons carry a particularly high mortality rate.
- A number of antidotes are toxic in their own right and may cause serious toxicosis if used when the animal is misdiagnosed, or if the antidote is given for too long a course. For example, **atropine** in the absence of anticholinesterase poisoning can cause life-threatening atropine poisoning, and the course of calcium disodium EDTA in lead poisoning must be limited because of the toxicity of the antidote.

A.3.Poisons for which there are specific antidotes

Table 1: Poisons with specific antidotes

Poison	Antidote	Dose and Comments
Amitraz	Atipamezole OR Yohimbine	Dogs: intramuscular injection of 50 µg Atipamezole/kg bodyweight (BW) OR intravenous injection of 0.2 mg Atipamezole/kg BW OR intravenous injection of Yohimbine at 0.1 mg/kg BW
Anticoagulant rodenticides	Dimercaptosuccinic acid (Succimer),	Succimer: 10 mg/kg PO, three times daily for 10 days
Arsenic	Dimercaprol	Dimercaprol: 3–6 mg/kg IM, 3 to 4 times daily, or 2–5 mg/kg IM every 4 h for 2 days, then every 8 h for 1 day, and every 12 h thereafter
Carbamates insecticides	Atropine	Atropine 0.25 to 0.5 mg/kg BW. Give 1/4 of dose immediately, IV if possible. Remainder is administered IM or SC if a response is seen to the IV bolus

Copper	d-Penicillamine	d-Penicillamine, 50 mg/kg orally for up to 6 days
Cyanide, HCN orally or by inhalation	Sodium nitrite followed by sodium thiosulfate	Sodium nitrite IV, 16–22 mg/kg, as a 3% solution followed by IV sodium thiosulfate, 1.65 mL of a 25% solution. Treatment may be repeated at half the initial doses after 30 min
Cyanogenic glycosides	Sodium nitrite + sodium thiosulfate	10–20 mg/kg sodium nitrite administered IV as 20% solution, up to 600 mg/kg of sodium thiosulfate as 20% solution
Lead	Calcium disodium EDTA	d-Penicillamine and Ca-EDTA can be cycled Cats: d-Penicillamine PO 125 mg/cat every 12 h for 5 days Cage birds: d-Penicillamine 55 mg/kg PO every 12 h, for up to 2 weeks before giving a 5 day rest
Molybdenum	Copper glycinate	Copper glycinate SC; 120 mg/adult cow; 60 mg/calf
Nonprotein nitrogen (urea, ammonia	5% acetic acid (vinegar)	Cattle is 2–8 L; sheep or goats, 0.5–2 L
Organophosphate insecticides	Pralidoxime (2-PAM) chloride	Atropine or glycopyrrolate, see carbamates toxicity, above 2-PAM chloride 20 mg/kg IM, twice daily

- Content/Topic 2: Drugs, feed additives for intoxication, food poisoning, nutritional and metabolic diseases

A. Food additives in toxicology

Table 2: Food additives in toxicology

Food additives	**Comments**
sodium nitrate	sodium nitrate resulted in testicular toxicity as evidenced by decreased sperm count and motility, daily sperm production and testis weight, inhibited activity of enzyme markers of spermatogenesis and induction of histopathological changes
Sulphites	Sulphites are widely used as preservative and antioxidant additives in the food and pharmaceutical industries. Exposure to sulphites has been reported to induce a range of adverse clinical effects in sensitive individuals, ranging from dermatitis, urticaria, flushing, hypotension, abdominal pain and diarrhoea to life threatening anaphylactic and asthmatic reactions.

potassium bromate	Potassium bromate (KBrO3) is widely used as a food additive and is a major water disinfection by-product
propylene glycol	Propylene glycol (PG) is a commonly used solvent for oral, intravenous, and topical pharmaceutical agents. It also thickens dairy products and salad dressing. toxicity cause seizures, agitation, and lactic acidosis
monosodium glutamate	Beside its flavour enhancing effects, MSG has been associated with various forms of toxicity. It has been linked with obesity, metabolic disorders….
recombinant bovine growth hormone	Is a synthetic hormone that is marketed to dairy farmers to increase milk production in cow?
Saccharin	Saccharin is an artificial sweetener. Sodium saccharin produced urothelial bladder tumors in rats by a non-DNA reactive mechanism that involves the formation of a urinary calcium phosphate containing precipitate, cytotoxicity and enhanced cell proliferation

B. **Drugs commonly used in toxicology**

Table 3: Drugs used in toxicology

Drugs used for decontamination	**Miscellaneous Drugs**	**Drugs used to control tremors or seizures**
• Hydrogen peroxide • Syrup of Ipecac • Apomorphine (in dogs only) • Xylazine • Activated charcoal	• Hydrogen peroxide • Syrup of Ipecac • Apomorphine (in dogs only) • Xylazine (Rompun ®) • Activated charcoal (Liquid Char Vet ®)	• Diazepam (Valium ®) • Barbiturates (Pentobarbital/ • Phenobarbital) Methocarbamol (Robaxin ®) • Inhalant anesthetics • Sodium Bicarbonate • Calcium gluconate • Ammonium Chloride

LO 3.3 – Administer prescribed medicine

- Content/Topic 1: Administration routes, techniques and dosage

A. Administration route, techniques and dosage of prescribed medecine

Table 4: Technique, route and dosage of prescribed medecine administration

Drugs	Administration route	Administration technique	Dosage
Atropine	Dermal	I.V	0.02-0.06 mg/kg in dogs and 0.04 mg/kg in cats
Xylazine	Dermal	I.M	0.44 mg/kg
Yohimbine	Dermal	I.V	0.1 mg/kg
Activated charcoal	Oral	Insert by stomach tube	1-3 mg/kg body weight
LD_{50}	Oral		666 mg/kg in rats
Dimercaprol	Dermal	IM	3–6 mg/kg body weight

LO 3.4 – Follow up the patient

- Content/Topic 1: Regular monitoring for evolution, possible complications and Post treatment check-up

A. Monitoring of the poisoned patient

Appropriate monitoring of the critically ill poisoned patient may necessitate monitoring of continuous electrocardiogram (ECG), blood pressure (BP), central venous pressure (CVP), urine output (UOP), pulse oximetry, end-tidal carbon dioxide ($ETco_2$), and venous blood gas (VBG) or arterial (ABG) blood gas analysis.

Table 5: Monitoring of poison patient

Monitoring Parameter	Important with Key Toxicants	Parameters for Concern
ECG	Amphetamines Beta-blockers	Brady cardiac (dog, HR <50 bpm; cat HR <120 bpm)Presence of R-on-T phenomenon
BP	Cardiac glycoside-containing plants	Hypotension (systolic <90 mm Hg)

CVP	Cardiopulmonary disease predisposing toward volume overload	CVP <0 or >10 cm H_20 (normal reference range 0–5 cm H_20)
UOP	Cardiac medications (eg, calcium channel blockers, β-blockers) potentially resulting in decreased cardiac output and therefore decreased renal flow	Anuric: <0.5 mL/kg/h
Pulse oximetry	Toxicants affecting pulmonary function (eg, long-acting anticoagulants, zinc phosphide rodenticides, essential oils, hydrocarbons)	Pulse oximetry reading <92%, indicating moderate to severe hypoxemia
$ETco_2$	Toxicants resulting in severe hypercapnea (hypoventilation) such as macrocyclic lactones (e.g., ivermectin, moxidectin), muscle relaxants (e.g., baclofen), anticonvulsants (e.g., phenobarbital, gabapentin, pentobarbital), opioids (e.g., fentanyl), and benzodiazepines or nonbenzodiazepines (e.g., sleep aids)	Gradient between $ETco_2$ and Pco_2 is typically <5 mm Hg $ETco_2$ >40–50 mm Hg suggests severe hypercapnea and hypoventilation
VBG	Toxicants resulting in the presence of a metabolic acidosis (e.g., ethylene glycol, amphetamines, salicylates)	BE <−5 mm Hg
ABG	Toxicants affecting pulmonary function (eg, long-acting anticoagulants, zinc phosphide rodenticides, essential oils, hydrocarbons)	Pco_2 <25 or >50 mm Hg

Abbreviations: ABG, arterial blood gas; BP, blood pressure; BE, base excess; bpm, beats per minute; CVP, central venous pressure; ECG, electrocardiogram; $ETco_2$, end-tidal carbon dioxide; HR, heart rate; UOP, urine output; VBG, venous blood gas; SSRI, selective serotonin reuptake inhibitor.

- Content/Topic 2: Preventive measures: preventing continued absorption of the Toxicant and dosage

A. External Exposures

A.1. Ocular Irrigation

With any ocular exposure, the eyes should be flush repeatedly with water or saline solutions for a minimum of 20-30 minutes. Only normal saline or tepid distilled water should be used – not contact solutions as they can have cleaning agents in them.

A.2. Bathing

The animal should be bathed in a mild hand dishwashing detergent. Baths may need to be repeated to completely remove the toxicant. Afterwards the animal should be rinsed well with warm water. The animal should be towel dried, not blow-dried, to prevent chilling.

B. Oral Ingestion

B.1. Dilution

Dilution with milk or water in combination with demulcents is recommended in cases of corrosive ingestion. A demulcent is an agent that coats or soothes the stomach. Milk of magnesia. A dosage of 1-3mg/lb. is a suggested dose.

B.2. Emesis

Emesis (vomiting) is most productive if performed within 2-3 hours post ingestion. Feeding the animal a small moist meal before inducing vomiting can increase chances of an adequate emesis. Emetics generally empty 40-60% of the stomach contents and are assumed to be more beneficial than gastric lavages. Some drugs can have anti-emetic effects. Examples of such drugs include phenothiazines, antihistamines, barbiturates, narcotics, antidepressants, and marijuana. It is important when taking the history to find out if the animal has been taking these or any other medications.

B.3. Emetic Agents

- **Three-per cent hydrogen peroxide** is an effective emetic for the dog, pig, ferret, and cat. Do not induce emesis in rodents, rabbits, birds, horses, or ruminants. The dosage is 1 teaspoon per 5 lbs., not to exceed 3 tablespoons. It should be administered undiluted – not mixed into water or food.
- **Syrup of ipecac** acts by causing gastric irritation and also stimulates the central nervous system to induce vomiting. In dogs, 2.2 ml/kg (not to exceed 30 ml) is administered by mouth. 3.3ml/kg should be diluted 1:1 and administered to cats via nasogastric tube.
- ***Apomorphine Hydrochloride*** should be used cautiously in cats. It is considered to be the emetic of choice in dogs by many clinicians. Apomorphine can be administered parentally or topically to the eye. The recommended dose is 0.04mg/kg IV or conjunctivally. When given intravenously in dogs, emesis occurs very rapidly.
- **Xylazine** is an alpha 2-adrenergic agonist, which can cause emesis in dogs and cats. Xylazine can cause bradycardia, hypotension, reduced respiratory rate, and CNS depress. The dosage for cats is 0.44 mg/kg IM while the dosage for dogs is 1.1mg/kg SQ or IM. Xylazine can be reversed with Yohimbine at a dose of 0.1 mg/kg IV.
- **Activated Charcoal:**Activated charcoal adsorbs a chemical or toxicant and facilitates its excretion via the feces. It basically acts like a magnet, attracting and holding the toxicant to its surface so that it passes through the gastrointestinal tract without being absorbed by the body. It is administered when an animal ingests organic poisons, chemicals or bacterial toxins or if enterohepatic

recirculation of metabolized toxicants can occur. The recommended dose of activated charcoal for all species of animals is 1-3 mg/kg body weight. Repeated doses of activated charcoal every 4-8 hours at half the original dose may be indicated when enterohepatic recirculation occurs.

Activated charcoal can be given orally with a large syringe or with a stomach tube. In symptomatic or uncooperative animals, anesthesia may be needed.

B.4. Enemas

Enemas are helpful when elimination of toxicants from the lower gastrointestinal tract is desired. Activated charcoal can be used in enema solution to help adsorb toxicant. Premixed enema solutions for humans are contraindicated in small animals due to potential electrolyte/acid-base imbalance.

B.5. Gastric Lavage

Gastric lavage should not be performed in cases of caustic or petroleum distillate ingestion. General anesthesia should be performed when performing a lavage. The type of toxicant involved in the exposure should always be considered when choosing an anesthetic agent. Isoflurane is the optimal anesthetic agent, but diazepam or a short-acting barbiturate may be appropriate.

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