1 Small Ruminant, Respiratory Diseases

Rhinitis	Nasal myiasis	Laryngitis, Tracheitis, Bronchitis
Rhinitis means inflammation of nasal cavity which may be acute or chronic, unilateral or bilateral, characterized clinically by sneezing, wheezing and stridor during inspiration and nasal discharge which may be serous, mucoid purulent or blood-tinged in consistency depending on the cause.	It is a chronic rhinitis and sinusitis of sheep characterized by persistent annoyance and mucopurulent discharge and is caused by invading maggots.	Inflammation of air passages including larynx, trachea and bronchi. They are all characterized by coughing, noisy inspiration and some degree of inspiratory embarrassment.
Etiology		
1. Physical causes as: **a.** Sudden change of the environment. **b.** Exposure to air drafts, chilling and cold. **c.** Inhalation of vapors and dusts. **d.** Unhygienic stables. **2. Infectious as** **a.** *Oestrus ovis* **b.** Sheep pox **c.** Blue tongue **d.** Contagious ecthyma (ORF) **e.** Melioidosis (*Pseudomonas pseudomallei*) **3. Miscellaneous: as allergic rhinitis.**	***Oestrus ovis*** is the prevalent fly cause nasal myiasis. **Occurrence:** This disease occurs in all breeds, sexes and ages of sheep. In warm countries both adult and larval phases of the disease persist throughout all seasons, but in temperate zones, adult flies emerge, mate and oviposit only during summer and myasis continues throughout the cold seasons.	May result from **1.** Upper respiratory tract infection. **2.** Direct irritation from inhalation of dust, smoke, or irritating gas; foreign bodies; or the trauma of intubation, excess vocalization, **3.** Injury from roping or restraint devices. **4.** Laryngitis may accompany *Fusobacterium necrophorum* or *Corynebacterium pyogenes* infections in sheep.
Pathogenesis		
1- Rhinitis is of minor importance except when it led to obstruction of the nasal cavity leads to difficult in respiration. **2-** Inflamed nasal mucous membrane may be eroded and may help in secondary bacterial invasion. **3-** Rhinitis itself not considered as a disease but it may be considered as complication of other diseases as pox. contagious eczema.	**Complications** The movement of numerous maggots in the nasal cavities, paranasal sinuses annoy the hosts, and the larvae produce allergic and inflammatory reactions. These changes cause discomfort and interrupt the animal's feeding. Some larvae wander along the trachea into the lungs and there produce fatal reactions.	**1-** Irritation of mucosa causes frequent coughing. **2-** Swelling of mucosa that results in inspiratory dyspnea.
Clinical Signs		
1. In the early stage, there may be intense irritation causing the animal to shake its head, rub its nose along the ground. **2.** Stick and twigs may be pushed up into the nostrils causing laceration, bleeding and complete obstruction. **3.** Sneezing in acute stages. Later on, it is followed by snorting and expulsion of large amounts of mucopurulent discharge. **4.** Inflammation may be unilateral or bilateral **5.** Nasal discharge may be serous or mucoid or mucopurulent **6.** Later on, difficult respiration accompanied by mouth breathing when both nostrils are obstructed.	**1.** Sheep attacked by ovipositing flies stamp with their feet, shake their heads, sneeze, blow and rub their noses against the ground or other sheep. **2.** In some cases, these acts traumatize and infect the nostrils. **3.** The animals also seek cool areas such damp shade where the flies do not frequent. **4.** During the time the larvae occupy the nose and sinuses, affected sheep may hold their heads towards the ground and persistently discharge mucopurulent exudate from the nostrils. **5.** Breathing may be difficult due to swollen nasal membranes and plugged nostrils. **6.** The morbidity may reach 80% of a flock, but mortality is nil. **7.** The course of adult fly attack periodically extends through the summer and the rhinitis and sinusitis continue up to 10 months.	**1.** Coughing: dry non-productive and easily induced by grasping the trachea or larynx. The cough becomes exudative with much mucous after secondary bacterial infection. **2.** Inspiratory dyspnea manifested by roaring or stridors. **3.** Fever and toxemia in viral infection followed by secondary bacterial infection and affected animal cannot eat or drink. **4.** In acute laryngitis, the soft tissue around the larynx is enlarged and painful
Diagnosis		
I) History. **II)** Clinical signs. **III)** Lab. Diagnosis: Examination of nasal swabs of scabs of scraping for bacteria, inclusion bodies or fungi may aid in diagnosis.	**I)** History. **II)** Clinical signs. • Restlessness behavior of sheep during hot-mid – days and observation of the fly. • Chronic nasal discharge and finding of larvae in sinuses confirm myiasis.	**I)** History. **II)** Clinical signs. **III)** Endoscopic examination of upper respiratory tract
Treatment		
(A) Specific treatment to control the causative agent as described under the specific diseases. **(B)** Nonspecific symptomatic treatment **1-** Remove the thick tenacious exudates which causing nasal obstruction using warm boric acid, then irrigate the nasal cavity with physiologic saline or mixture of saline and antibiotics to provide symptomatic relief and minimize secondary bacterial rhinitis. **2-** Spray-up a nasal decongestant into the nostrils to provide some relief. E.g., Delta rhinol, Prosiline… etc. **3-** Culture and sensitivity testing to assist the selection of suitable antibacterial agents (antibiotic or Sulphonamides), which may be helpful in controlling secondary bacterial infection.	Sheep should be treated for nasal myiasis during autumn as larvae are small. Killing of mature larvae in the sinuses may result in severe reaction in the sinus membranes. Either of the following compounds and dosage rates are highly effective. **1.** Rafoxanide administered orally at dose rate of 7.5 mg/kg, Bwt **2.** Ivermectin at the rate of 50 ug/kg Bwt (Ivomic, Iveen, Ivomax, Uvemic, Avimic).	**1.** Most viral infection of the upper respiratory tract resolve spontaneously if the animal has rest away from the dusty food, or inclement weather. **2. Antibacterial:** **a.** Sulphadiazine: IV for 3-5 days. **b.** Broad spectrum antibiotics: penicillin-streptomycin or oxytetracycline. **3. Anti-inflammatory**: Dexamethasone to reduced inflammatory oedema at larynx. **4.** Administration of **diuretic** drugs, e.g., furosemide, may be indicated for resolution of laryngeal edema if present.

PNEUMONIA

Pneumonia

It is the inflammation of lung tissues, may be accompanied by inflammation of the bronchiole's bronchopneumonia. It may be associated with pleurisy pleuropneumonia.

Enzootic pneumonia

An acute type of pneumonia caused by mixed environmental and infectious factors affecting sheep of all ages with high prevalence in nursing and feedlot lambs

Characterized clinically by signs of respiratory distress, mucopurulent often blood tinged oculonasal discharge, depression, anorexia and pyrexia and pathologically by pneumonitis and pleuritis.

Etiology

Viral Pneumonia

1. *Adenovirus.*
2. *Parainfluenza virus type 3.*
3. *Respiratory syncytial virus*
4. *Reovirus.*

Bacterial

1. *Pasteurella haemolytica.*
2. *Mycoplasma pneumonia.*
3. *Chlamydia psittaci.*

Parasitic

1. *Dictycallus filaria.*
2. *Protostrongylus rufescence*
3. *Mullerius capillaris.*

Mixed environmental and infectious factors affecting sheep

Pathogenesis

The main disturbance induced by pneumonia related to interference with gaseous exchange between the alveolar air and the blood so anoxia and hypercapnia is one of the main signs associated with pneumonia beside the effect of toxin produced by the bacteria and necrotic tissues.

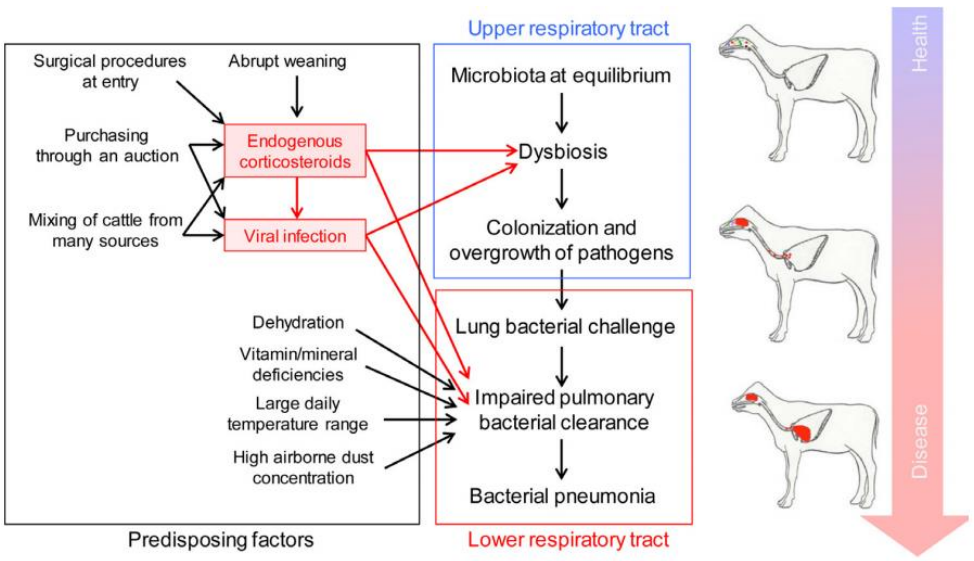
The process by which pneumonia develops varies with the causative agent and its virulence and with the portal way by which it is introduced into the lung

Bacterial

Respiratory air way	Hematogenous
↓	↓
Bronchiolitis	Form septic foci
↓	↓
Parenchyma	Lung abscess
↓	↓
Bronchopneumonia	Rupture of abscess in airways
	↓
	Bronchopneumonia

Viral

1. Viral infection introduced by inhalation in to interstitial tissue
2. There is absence of acute inflammatory reactions
3. The reaction of lung in this case is manifested by enlargement and proliferation of the alveolar epithelium, alveolar edema, thickening of interstitial tissues and lymphocytic aggregations around the alveoli, blood vessels ad bronchioles
4. Interstitial pneumonia results in consolidation of pulmonary parenchyma without involvement of bronchi
5. On auscultation, a loud bronchial sound heard. These are caused by passage of air in non-obstructed bronchioles and are made more audible by consolidation of the surrounding tissues.



1. Viral infection, stress and/or suboptimal environment.
2. Proliferation of bacteria in the nasopharynx.
3. Bacteria gain access to the lung via aerosolized droplets.
4. Adherence, colonization and replication in the lung.
5. Tissue destruction and inflammation.
6. Bacterial pleuro/broncho-pneumonia.

Clinical Signs

1. Shallow rapid respiration.
2. Nasal discharge may or may not present depending upon the amount of bacterial exudate and whether or not the upper respiratory passages are involved.
3. There is fever with anorexia, depression and reluctance to lie down.
4. The pulse rate is elevated.
5. Cough is an important sign that may be painful and moist in case of bronchopneumonia, while frequent, dry, and hocking and often in paroxysms in case of interstitial pneumonia.
6. The odor of the breath may be putrid in gangrenous pneumonia.
7. **Auscultation of the lung; Sounds Heard:**
   a. In the early stage of congestive stage of bronchopneumonia and interstitial pneumonia: there is increased bronchial sound [breath sound].
   b. **Crackles (moist rales):** develop in bronchopneumonia as exudation increases.
   c. **Presence of dry rales:** (Clear, harsh bronchial sounds) are audible in uncomplicated interstitial pneumonia (viral pneumonia).
   d. **Loud bronchial sound:** occurs when complete consolidation occurs (consolidation also causes increased audibility of heart sounds).
   e. **Frictional sound:** may occur if pleurisy is complicated with pneumonia (pleuropneumonia).
   f. **Muffling of bronchial sounds:** occurs in the late exudative stages i.e., Gray hepatization.

Sounds Heard according to disease stage:

1. **In the early congestive stages:** Harsh and exaggerated sound is heard.
2. **Consolidation:** There is dullness, while there is an increase of the audibility of the bronchial and heart sounds.
3. **In lobar pneumonia:** The sound heard over the affected lobe is the same.
4. **In lobular pneumonia:** Different sounds heard over different lobules according to the lesion formed.
5. **After resolution:** Harsh and exaggerated sound.

1. Fever (41-42°C), drooping ears and lowering head, the sick lambs refuse to eat and lose live weight.
2. Mucopurulent nasal discharge and lacrimation.
3. Respiration is accelerated, difficult and often accompanied by coughing.
4. Weakened lambs isolate from the flock.
5. Auscultation of thorax reveals consolidation of the anteroventral parts of lungs.
6. Morbidity reaches 50%, while mortality reaches 10%.

Diagnosis	
I) History. **II)** Clinical signs. **III)** Lab. Diagnosis: Laboratory examinations of nasal swab or tracheal sputum for microbiological examinations including staining and culturing on selective media. A sensitivity test is a valuable tool for effective treatment	**I)** History. **II)** Clinical signs. **III)** At necropsy, finding of consolidation in the ventral planes of lung accompanied by pleuritis and pericarditis.
Treatment	
Hygienic treatment Affected animals should be housed in warm, well ventilated, draught free, provided with fresh water and light and providing of easily digestible food (Barseem or Bran mash). **Medicated treatment** **1- Anti-inflammatory:** **a.** Meloxicam :2.5 ml/100 kg bodyweight. **b.** Flunixin Meglumine.1ml/25kg I/M or I/V **c.** Dexamethasone. **2- Antibiotics** **a.** Tulathromycin (Draxxin®). **b.** Tildipirosin (Zuprevo®). **c.** Ceftiofur ((Excede® 3.3ml/100 kg s/c under ear one shot for 3-7 days), (Ceftiocyl® 1ml/50 kg s/c under ear one shot for 3-7 days), (Eficur®). **d.** Marbofloxacin: (Marbocyl®, Marbox®, Forcyl®)1ml/50kg I/M or I/V every 24 hrs. **e.** Florfenicol: (Nuflur®, Aviflor®)1ml/15kg I/M every 48 hrs. or 1ml/10kg s/c every 96 hrs. **f.** Sulphonamides: 1ml/15 kg I/M OR I/V. **3- Bronchodilators**: (Minophylline-N®, Etaphylline®). **4- Antihistaminic**: (Histacure®, Allergamine®, Avil®).	

Ovine Progressive Pneumonia (OPP)	Pulmonary Adenomatosis or Pulmonary Carcinoma
It is a lymph proliferative pneumonia caused by a slow viral infection of adult sheep with signs of dyspnea, physical weakness and emaciation.	It is a contagious neoplastic disease of sheep characterized by insidious but continuous development of weakness and emaciation, dyspnea, nasal discharge and frequent cough caused by retrovirus.
Incidence and occurrence	
1. It is fatal disease with high morbidity in sheep producing countries. **2.** Economic losses may be related to death and unthriftiness during the long course of the disease and the cost of control programs. **3.** All sheep breeds are susceptible. **4.** The proportion of infected sheep increases with age.	**1.** Occurs in all sheep-producing countries, except Australia, all spp. and breeds, and all ages and sexes. **2.** The clinical signs appeared only in adult animals (Neoplastic disease). **3.** Goat are infrequently affected
Cause	
This disease caused by a non-oncogenic retrovirus of the subfamily *Lentivirinae*.	Retrovirus of the family *Retroviridae* (6 classes of the virus are isolated which are A, B, C, D, E and F).
Transmission & Pathogenesis	
Transmission The virus is transmitted in aerosol by inhalation from feces and colostrum (infected lymphocytes) by ingestion. **Pathogenesis** **1.** After entrance of virus by inhalation into the respiratory system the target cells are invaded and, in some animals, the virus spreads into the bronchial and mediastinal lymph nodes, blood, spleen and kidneys. **2.** The invaded pulmonary cells probably reticular cells and B-lymphocytes are not damaged but are stimulated to proliferate. **3.** The interalveolar septa thicken from the presence of numerous new histiocytes, some fibrocytes and collagenous fibers. **4.** As the septa enlarge, the squamous epithelial cells lining the alveoli morphologically transform into cuboidal cells. **5.** In addition, the peribronchiolar and perivascular lymphoreticular tissues may undergo hyperplasia and form active germinal center. **6.** The combined septal, bronchiolar and vascular changes reduce and obliterate alveoli and lower the efficiency of gaseous exchange and produce gradually fatal hypoxia. **7.** The apparent reduction in patency of septal capillaries probably causes pulmonary arterial hypertension and possibly right ventricular hypertrophy (congestive heart failure).	**Transmission** **1.** Respiratory secretions that contain all infected cell, which coughed by diseased animal during coughing or blowing in the environment then inhaled by healthy animal **2.** The infection occurs by the (virus) itself. **3.** Overcrowding of sheep in pens especially in closed buildings facilitates spread of infection from diseased to healthy animals. **Pathogenesis** **1.** The virus attacks the target cells which are: **a.** Alveolar type II epithelial cells **b.** Terminal non- ciliated bronchiolar epithelial cells At the initial stage, the virus-containing cells (infected cells) from disease animals proliferated forming a single cell layer lining the alveolar lumen. A papilliform mass develop from this layer and extend into the lumen leading to narrowing of lumen resulting in coughing, hypoxia, and dyspnea.	**2.** The papilliform mass continues to proliferate under the virus multiplication till occupy all the alveolar lumen. Then the proliferation transferred to the adjacent alveoli. **3.** The peripheral neoplastic cells contain secretory granules. These cells secrete osmophilic secretion that leads to increase the nasal discharge. **4.** With viral multiplication, protein synthesis and cell division increase which leads to expansion of the small foci or aggregation of multiple foci resulting in formation of large nodules that may extend to include the majority of lung tissue. **5.** Regional lymph nodes may be affected and enlarged after several months or years due to metastasis. **6.** The growth of extensive neoplastic tissue within the lung may increase the hindrance to pulmonary circulation resulting in right ventricular hypertrophy, cardiac failure and death. **7. Death occur due to:** **a.** Severe hypoxia and dyspnea. **b.** Ventricular hypertrophy and CHF. **c.** Secondary bacterial invasion may lead to death of premature animals.
Clinical Symptoms		
Incubation period: 1-2 years. **1.** Animals are unable to walk and separate from flock. **2.** Animals are emaciated with progressive loss of body weight. **3.** Respiration accelerates. As the disease worsens, breathing increases to 80- 120/ min. during exertion and remain fast during rest. **4.** Nostrils are dilated and head held high, the animal struggle, sometimes mouth breathing for adequate amount of oxygen. **5.** Animals remain standing because recumbency forces the diaphragm forward **6.** Auscultation and percussion of the thorax reveal aeration in dorsal parts of the lungs and consolidation in ventral parts. **7.** Body temperature is usually normal. **8.** Death results from hypoxia.	**Depends upon the severity and stage of disease** **(A) In early stage:** **1.** The animal appears in good health condition. **2.** Shallow rapid respiration appears after excitement or exercise with frequent coughing. **3.** Dilation of nostrils due to dyspnea. **4.** Extension of head and neck during inspiration. **5.** Auscultation reveals rales and consolidation particularly in the ventral plane.	**(B) In late stage:** **1.** Loss of weight despite normal appetite. **2.** Weakness and emaciation. **3.** Dyspnea. **4.** The animal remains in standing position because recumbency may exacerbate the dyspnea **5.** Nasal and ocular discharge. **6.** Death after a few months, usually associated with secondary pasteurellosis
Diagnosis		
I) History. **II)** Clinical signs. **III)** Laboratory confirmation by positive results from complement – fixation tests, agar gel immunodiffusion tests, enzyme –linked immunosorbent assay, virus isolation and histopathological findings. **IV)** Blood examination reveals mild hypochromic anemia hypogammaglobulinemia, and persisting leukocytosis.	**I)** History. **II) PM lesions:** **(A) In early stage:** the lung contains blue gray nodules 1-20 mm diameter. **(B) In late stage:** **1.** Lung contains small nodules with large neoplastic mass. **2.** Regional lymph nodes are enlarged from metastasis. **3.** Right ventricle is hypertrophied and dilated. **4.** Small number of exudates may excavate and accumulate in some body cavities.	**III) Histopathology of lung tissue** **1.** Hyperplasia and hypertrophy of alveolar and bronchiolar epithelium. **2.** Papillary masses are present that present in adjacent alveoli. **IV) Differential diagnosis** Parasitic pneumonia, Progressive pneumonia, Pasteurellosis. A simple test can be used to differentiate the Adenomatosis from these diseases. Elevation of the hindquarters of the affected sheep resulting in fluid run out of the nostril in severely affected animals

Prevention, Treatment and Eradication:

Prevention, treatment and eradication:

1. **Prevention** is accomplished by avoiding exposure of healthy sheep to diseased animals.
2. Addition to the flock should be considered only when the status of the originating flock is known to be free of opp.
3. Pop free lambs can be obtained by removing them from their dam at birth prior to receiving colostrum from the infected dam and rearing in a clean atmosphere away from infected sheep.
4. **Eradication:**
   - **a.** Animals from infected flocks cannot be sold live, taken to common pasture or to shows, thus restricting spread.
   - **b.** Testing of antibodies every 6 months (C.F.T and ELISA) and culling of all reactors.

1. There no treatment but control by culling of the affected sheep and goat.
2. Slaughter of affected and contact animals
3. Cleaning and disinfection of premises before replacing with sheep from clean flock.

Verminous Pneumonia

Definition

It is a chronic and prolonged type of pneumonia caused by nematode infestation of sheep and goats.

Characterized clinically by respiratory distress and pathologically by bronchitis and bronchopneumonia.

Incidence and Occurrence

1. This disease occurs in all breeds, sexes and ages.
2. The disease is common among animals 2-18 months old.
3. Most outbreaks of VP occur during cool seasons, especially autumn and early winter because development of these nematodes need low temperature.

Etiology:

1. *Dictycallus filaria.*	2. *Protostrongylus rufescence.*	3. *Mullerius capillaris.*

Pathogenesis

1. The prevalent parasite occupies the lumen of large bronchi mixed with exudates; they lay embryonated eggs.
2. Eggs expelled to the exterior by cough, they also escape occasionally through the nose, but usually through the alimentary tract.
3. Hatching occurs in intestine and the larvae pass to the exterior with feces.
4. Under favorable conditions, they molt twice and after 6-7days become infective.
5. Following ingestion by a susceptible sheep, the larvae invade the intestinal mucosa and move through the lymphatic and venous blood into the lungs
6. By penetrating the capillary walls, they enter the alveoli and moves into bronchiolar and bronchi lumen where they develop to the adult and this needs 5 weeks.
7. The bronchi, the irritating worms cause epithelial hyperplasia and muscular hypertrophy. Ova, larvae, bacteria and exudate are extruded from parasitized bronchi into alveoli provoke bronchopneumonia

1. Infection with *Protostrongylus rufescence* occurs through ingestion of intermediate hosts of the genus *helicello* containing third infective stage with forage.
2. From their definitive position in the bronchioles, the adult worms provoke chronic bronchiolitis and hyperplasia of peribronchiolar lymphoid tissues.
3. Exudate may descend into alveoli causing focal
4. lobular pneumonia.

1. *Mullerius capillaris* inhabits the lung in the sub pleural alveoli and stimulate around themselves granulomatous reactions that become nodules.
2. Hatching of eggs occurs in the lung as first larval stages are expelled to the exterior by cough where they need snails of genera Cepaea, Helicigona as intermediate hosts.
3. When infected intermediate hosts are consumed by sheep, the larvae penetrate the intestinal mucosa and move through lymphatics and venous blood back to the lungs where they develop into adults.

Clinical signs

The severity of clinical signs depends on the number of infecting worms and the age of affected animals. The signs include:

1. Fever.
2. Cough.
3. Nasal Discharge.
4. Rapid breathing.
5. Retarded growth.
6. Physical weakness.
7. Emaciation.
8. Some animals develop diarrhea.

Diagnosis

I) History.

II) Clinical signs, especially persistent cough and nasal discharge in young sheep.

III) P.M. lesions:

1. With *D.F.,* bronchi contain masses of worm mixed with exudate.
2. With *P. rufescence*, bronchioles are often closed with worms and exudate consequently affected lobules may be atelectasis and infected.
3. Lung infected with *Capillaris* contain red, grey or green nodules. These lesions located in the sub pleura of the diaphragmatic lobes vary in number, consistency and shape.
4. Demonstration of larvae in feces or adult in bronchi.

Treatment

1. **Albendazole** 10 mg/kg B. wt. PO is efficient for mature and immature *D. filaria* infections and efficient against *P. rufescence*.
2. **Fenbendazole** 15 mg/kg B.wt. PO is 10% efficiency against immature and adult *D. filaria* and efficient against *P. rufescence*.
3. **Oxfendazole** 5mg/kg B.wt. PO is 99% efficiency against 5th larval and adult *D. filaria* but less effective against 3rd and 4th stage larvae.
4. **Levamisole** 7.5mg/kg B. wt., 97-99% efficient against immature and mature *D.F.* it is also efficient against *P. rufescence* and *M. capillaris*.
5. **Ivermectin** 0.2 mg/kg B.wt. S/C or 0.3 mg PO is 80-100% efficient against *D. filaria* and *P. rufescence* but it is not efficient against *M. capillaris*.

2 Small Ruminant, Nutritional & Metabolic Diseases

Cobalt Deficiency

Synonyms: Pining disease, Enzootic marasmus, White liver disease, Coastal disease, Coat disease, Salt sickness.

- Chronic wasting disease in small ruminant.
- Occurs due to deficiency of cobalt.
- Result in failure to synthesize vitamin B 12 (cyanocobalamin).
- Ch.by anorexia, anemia, and loss of body weight.

Occurrence

a. Young animals are more susceptible than adults.
b. Most observed in weaned lambs at pasture during late summer/autumn.
c. Ruminant grazing on cobalt-deficient forages is more susceptible.
d. White liver disease mostly occurs in area of sandy soils that are deficient in cobalt.

Aetiology

1. Mainly due to deficiency of cobalt in ration resulting in deficiency of vit B12 synthesized in the rumen.
2. Absorption of Vit. B12 occurs primarily in the small intestine and is enhanced by slower rates of movement of material through the intestine.
3. Absorption is inhibited by damage to the walls of the small intestine, caused by worms, for example.

Pathogenesis

1. Cobalt is a component of vit. B12 which is synthesized in the rumen by ruminal microflora. Therefore, deficiency of cobalt results in inadequate synthesis of vit. B12.
2. Vit. B12 is used to metabolize propionate for energy and for making Sulphur-containing amino acids for wool growth.

Role of Vitamin B 12:

a. Energy Production
b. Cell Growth and Maturation
c. Coat Production

3. Cobalt plays an important role in carbohydrate metabolism so its deficiency may lead to ketosis (hypoglycemia).

Clinical Signs

Course of the disease is several weeks to few months.

In seriously deficient regions:

- The morbidity rate: 60%.
- The mortality rate: 80%.

Signs appear within few months after feeding on cobalt-deficient ration

1. Anorexia.
2. Anemia.
3. Pale color MM.
4. Loss of body weight.
5. Weakness and emaciation
6. Poor quality wool with an open fleece.
7. Profuse lacrimation (weepy eye) that stain the face.
8. Some animals may develop severe diarrhea and may die if not treated. This diarrhea may result from abnormal metabolism by the ruminal flora digestion associated with cobalt deficiency.

Cobalt deficiency is much less common in adults but is reported to cause:

1. Lack of thrift.
2. Infertility and abnormal estrus.
3. Decreased milk and wool production.
4. Neonatal mortality.
5. Reduced the resistance to infection because of the role of the cobalt in immune response (more susceptible to Clostridial diseases (for example pulpy kidney) and Pasteurellosis (pneumonia).
6. In severe cases of cobalt deficiency (referred to as Ovine White Liver Syndrome) lambs present with nervous signs including depression, head pressing, and aimless wandering.

In White Liver Disease, all mentioned signs are involved in about 10% of the cases with photosensitization.

Copper Deficiency (Hypocuprosis)

Synonyms: Enzootic Ataxia or Sway Back disease.

The minimum recommended dietary copper concentration (dry matter basis) is 5 ppm (mg/kg) for sheep.

-Ch. clinically by: -Unthriftiness. -Diarrhea. -Lameness. -Wool /hair abnormalities.

Occurrence

a. Copper deficiency occurs primarily in young ruminants.
b. Lambs up to 3 months of age due to copper deficiency in the last half of pregnancy for dam.
c. Young animals are more susceptible to copper deficiency than mature animals.

Aetiology

Primary Copper Deficiency

Occur when the copper intake in the ration is inadequate.

e.g., feeding plants grown on copper deficient soil.

Secondary Copper Deficiency

Occur when utilization of copper by tissues is impeded due to dietary excess of **Molybdenum** and **Inorganic Sulfate** alone or in combination.

Molybdenum, interfere with copper absorption, copper storage in liver, and copper utilization by tissues.

Pathogenesis

Copper plays an important role in "Tissue Oxidation "by formation of Copper-Containing Enzymes.

Enzyme/Cells	Function	Cu Def. Manifestation
Superoxide Dismutase (Antioxidant E.)	Immune response. Phagocytic activities. Growth rate.	Increase susceptibility for infection. Retardation of growth.
Cytochrome C oxidase	Production and maintenance of myelin sheath.	Nervous manifestations.
Tyrosinase	Neutralization of free thiol group. Essential for normal keratinization of skin & hair. Melanin production.	Abnormal keratinization and depigmentation of skin and hair.
Ceruloplasmin	Reutilization of iron for Hb synthesis.	Anemia.
Lysol oxidase	Maturation of elastin and collagen. Maintenance integrity of connective tissues.	Disease problems in ligaments and tendons. → Problems in locomotor system.
Osteoblast Cells	Bone growth and development.	Skeletal abnormalities. → Problems in locomotor system.

Clinical Signs

Morbidity up to 90% with high mortality rate.

The **Course of The Disease** varies form Few days to Several weeks.

General symptoms

1. General Unthriftiness.
2. Crimpless wool and steely appearance.
3. Diarrhea & Anemia.
4. Depigmentation of black wool.

Specific Symptoms

Sway Back

a. **Congenital Form** (cerebrospinal sway back): affected lambs born dead or weak and unable to stand or suck.
b. **Delayed Form** (progressive spinal sway back): usually 3-6 weeks age.
c. **Postnatal Form** (acute fatal sway back): Similar to delayed form but developing suddenly and lead to death within 1-2 days due to acute swelling of cerebrum.

Sings of three forms are incoordination, paralysis and blindness.

Enzootic Ataxia

1. Affect only unweaned lambs.
2. Most cases at 1-2 months of age.
3. Less incidence at birth.
4. Lambs affected at birth usually die within 3-4 days.
5. While older lambs for 3-4 weeks:
  a. There is incoordination of hind limbs and paresis.
  b. There are flexion joints.
  c. Knuckling at fetlock.
  d. Wobbling of hind quarters and falling.
  e. Recumbency when the fore limbs become involved.

Cobalt Deficiency	Copper Deficiency (Hypocuprosis)
PM lesions	
1. Emaciated carcasses. **2.** Fatty degeneration of liver (White Liver Disease). **3.** Atrophy of the bone marrow.	
Diagnosis	
I) Case history. **II)** Clinical signs. **III)** PM lesions. **IV)** Lab Diagnosis **1.** Haemoglobin: Low level; 8-9 gm/dl (N. 11-12 gm/dl). **2.** **Detection of vit-B12 level in feces** which is an indicator of cobalt status. Generally, each gram of feces contains 2.13 mg of Vit-B12 which indicates an intake of 0.09 mg of cobalt/kg of dry ration. Therefore, the measurement of VitB12 in the feces is an indicator of the cobalt intake in sheep. **3.** **Detection of plasma cobalt level** (N= 0.17-0.50 µmol/L) (1-3 ug/dl) which reduced to 0.03-0.04 µmol/L. **4.** **Detection of plasma vit-B12 level** (below 0.2 mg/ml). **5.** **Hypoglycemia** (below 60 mg % glucose), low level of alkaline phosphatase with high level of ketone bodies.	**I)** Case history. **II)** Clinical sign. **III)** PM lesions. **IV)** Lab Diagnosis **1.** Plasma copper is below 50 ug/100 ml (N= 70-120 ug/ 100ml). **2.** Liver copper level is below 80 mg/kg dry mater (N= 120-1000 mg). **3.** Copper content of hair (N = 6.6 –10.4 mg/kg dry matter). **4.** Estimation of Erythrocyte Superoxide Dismutase Enzyme 2-6 IU/mg haemoglobin in case of Hypocuprosis. **5.** Estimation of Ceruloplasmin (copper-protein complex) N= 40-100 mg/ dl.
Treatment & Prevention	
1- Supplementation of cobalt: **a.** On water with a range of 0.2-0.4 mg cobalt per litre of water. **b.** On feed: 2 mg cobalt/1 kg dry feed. **2-** Using of cobalt sulfate or cobalt chloride in salt mixture with average 2.5 gm cobalt/100 kg of salt mixture. **3-** The use of commercially available **Cobalt Pellets** will provide adequate cobalt levels up to 3 years that they stay in the rumen and not regurgitated. **4-** Application of 3-5 kg hydrated cobalt sulfate/hectare to deficient soil will increase the cobalt level for 3-5 years. **5-** Daily dietary supplement of 1 mg of cobalt / animal until improvement occurs. **6-** Administration of vit. B12.	**1.** Copper sulphate 1 gm weekly for lamb orally for 3-5 weeks. **2.** 2-3 gm copper sulphate weekly for 3-5 weeks for adult orally. **3.** Single parental injection of copper glycinate 150 mg I/M or S/C. **4.** Mineral mixture contains at least 3-5 % of copper sulphate. **Prevention** **1.** Supplementation of ration with copper sulphate, 5 mg copper per kg of matter by any of several different ways. **a-** Oral dosing or dietary supplementation 1.5 gm for sheep/week. **b-** Mineral mixture of salt licks containing ¼ – ½ % copper sulphate. **2.** Parenteral injection of copper glycinate as 150 mg I/M or s/c. **3.** Single injection of 45 mg copper glycinate in mid pregnancy is sufficient to prevent sway back in lambs. **4.** Copper oxide needles: Fragments of oxidized copper wire 8 mm in length and ½ mm in diameter for oral dosing considered as one of the most effective and safest methods for control of copper deficiency in ruminants. These needles retained in for stomach and abomasum for 100 days or more and copper slowly released, absorbed and stored in liver. **5.** Decrease the level of molybdenum, inorganic sulphate and cadmium in the ration.

Role of Vitamin B 12:

A. **Energy Production**

1. Propionic acid is a product of rumen fermentation.
2. Vit. B12 is essential in the conversion of propionic acid → succinate→ glucose for energy. The main site of this conversion is in the liver.
3. Therefore, vit. B12 def. failure of appetite and death from inanition (malnutrition).

B. Cell Growth and Maturation.

1. Young developing and pregnant or lactating animals, have high requirements for Vit. B12.
2. So, def. of vit B12 leads to failure of growth and ill thrift.

C. Coat Production

1. Vit. B12 is required for the metabolism of methionine. Methionine is necessary for optimum growth and coat production.
2. Vit. B12 plays an important role in synthesis of RBCs so its deficiency may lead to anemia.

Thyroid Hormones

Thyroid hormones are two hormones produced and released by the thyroid gland, namely triiodothyronine (T3) and thyroxine (T4). They are responsible for regulation of metabolism. T3 and T4 are partially composed of iodine. A deficiency of iodine leads to decreased production of T3 and T4, enlarges the thyroid tissue and will cause the disease known as goiter.

a. The thyroid hormones act on nearly every cell in the body.
b. They act to increase the basal metabolic rate, affect protein synthesis, help regulate long bone growth (synergy with growth hormone) and neural maturation.
c. The thyroid hormones are essential to proper development and differentiation of all cells of the body.
d. These hormones also regulate protein, fat, and carbohydrate metabolism, affecting how cells use energetic compounds. They also stimulate vitamin metabolism.
e. Thyroid hormone leads to heat generation.

Zinc Deficiency	Iodine Deficiency
Characterized clinically by: 1- Wool eating. 2- Alopecia. 3- Parakeratosis 4- Abnormal hoof growth. 5- Lameness. 6- Unthriftiness.	• The cardinal sign of iodine deficiency is Goiter. • The major clinical manifestation of iodine deficiency is neonatal mortality with alopecia and abnormal enlargement of the thyroid gland. **Occurrence** • All breeds, sexes, and ages • More frequent in ewes than rams • More frequent in fetuses and lambs than in yearling and adult

Aetiology

Primary Zinc Deficiency	Secondary Zinc Deficiency	Primary Iodine Deficiency	Secondary Iodine Deficiency
a. Feeding on zinc deficient ration. **b.** Plants from a soil of a pH above 6.5. **c.** Soil fertilized with high amount of phosphorus and nitrogenous fertilizers.	**a.** Feeding on late cure hay which may be poorly digestible and contain excessive Sulphur which hindrance absorption of zinc. **b.** Silage which contaminated with soil also affect the digestibility of zinc.	Iodine deficient in the ration or in drinking water (this related to geographical circumstances).	**a.** High dietary intake of calcium (decrease intestinal absorption of iodine). **b.** High dietary intake of rations containing goitrogenic substances e.g., cabbage, turnips, soya bean, linseed meal etc. **c.** Goitrogenic substances are thiocyanate, or nitrate (restrict iodine intake).

Pathogenesis

Deficiency in Zinc affect its role in:	
1. Development of somatic and sexual cells i.e. spermatogenesis. **2.** keratinization of skin. **3.** Calcification of bone. **4.** Protein and nucleic acids metabolism → Cell mediated immunity. **5.** Carbonic anhydrase enzyme in RBCs and glandular cells of stomach **6.** RNA function **7.** Secretion of glucagon and insulin → CHO metabolism. **8.** Wound healing.	**1.** Ionized iodine is absorbed from gastric and jejunal mucosa into the plasma, then distributed to all tissues and actively concentrated and stored in the thyroid glands (about 80% of the body iodine is stored in the thyroid gland). **2.** Iodine deficiency results in a decrease production of thyroxine hormone and subsequently stimulation of the secretion of thyrotrophic hormone by pituitary gland. **3.** Due to iodine def. in the ration, the thyroid gland tends to compensate this by increasing the activity with the resulting in hypertrophy and enlargement of thyroid gland (Goiter). **4.** The primary deficiency of thyroxine hormone is responsible for hair abnormalities (alopecia) by absence or diminution in the size of hair follicles.

Clinical Signs

1. Alopecia. **2.** Parakeratosis. **3.** Wool eating. **4.** Poor growth rate & Anorexia. **5.** Swollen hock. **6.** Arched back and abduction of legs. **7.** Increase susceptible to infection. **8.** Fissured hoof or sloughing of it. **9.** Decrease in fertility and cessation of spermatogenesis.	**1.** Low growth rate due to low basal metabolic rate (BMR). **2.** Enlargement in the laryngeal area. **3.** Dyspnea, noisy breathing, and dysphagia. **4.** Irregular estrus and abortion due to stillbirth and weak lambs. **5.** Dry skin and poor-quality wool. **6.** Reduced libido and low semen quality. **7.** Thick, scaly skin with sparse wool and thickened neck.

PM lesions

	Bilateral enlargement of thyroid gland. Body cavities may contain fluids.

Diagnosis

I) Case history. **II)** Clinical signs. **III)** Pm lesions. **IV)** Lab Diagnosis: A- Estimation of serum zinc level: - 1- Have a good diagnostic value. 2- Normal level in cattle and sheep: 80-120 ug/dl. B- Skin biopsy: - "Skin biopsy "will confirm the diagnosis of parakeratosis (marked increase in thickness of all elements of epidermis).	**I)** Case history. **II)** Clinical signs. **III)** Pm lesions. **IV)** Lab Diagnosis: 1- Estimation of iodine level in blood and milk. 2- Estimation of PBI in plasma (N = 2.4 – 14 ug%). 3- Estimation of iodine levels in soil and pasture.

Treatment & Prevention

1. Zinc sulphate or carbonate 200 mg/kg of food. **2.** Zinc oxide suspended in olive oil and injected I/M 200 mg of zinc oxide is sufficient to ewe and 50 mg for lamb. This supplementation gives result for 2 months. **3.** Injection of zinc at level of 2-4 mg/kg B. wt. for 10 days give good response. **4.** Oral supplementation of zinc sulphate 250 mg/day for 4 weeks give cure in goats in 12-14 weeks. **Prevention** 1. Intraruminal pellets give protection for 7 weeks. 2. S/c depots by injection of zinc oxide s/c. 3. Supplementation of zinc in ration. 4. Avoid feeding on plants from soil deficient in zinc. 5. Avoid excessive Sulphur or calcium in ration. 6. Feeding on good digestible feed.	**1.** Treatment must be undertaken with care as overdose will cause toxicity (Iodism). **2.** Specific iodine therapy is recommended for treatment e.g., strong iodine solution (Lugol's solution) 1 drop / 5 kg B. wt.in water daily for 2-3 days. **Prevention** **1.** Individual dosing of pregnant ewes, on two occasions during the fourth and fifty months of pregnancy, with 280 mg potassium iodide or 370 mg potassium iodate has found to be effective. **2.** The iodine can also be administered as an injection in poppy seed oil (containing 40 percent bound iodine). One ml given I/M 7 to 9 weeks before lambing is sufficient to prevent severe Goiter and neonatal mortality in the lambs. 3- 3. Salt mixture containing iodine added to the ration as 0.5%.

Vit, E (Selenium) Deficiency (Complex Syndrome of Young Lambs)

Synonyms

1. Enzootic muscular dystrophy
2. Nutritional muscular dystrophy.
3. Selenium responsive Unthriftiness
4. Stiff lamb disease
5. White muscle disease

- Selenium and vitamin E are essential in sheep rations.
- Selenium is a trace element which works with vitamin E to prevent and repair cell damage in the body.
- Selenium and vitamin E both play a role in immune function and are vital for growth, reproduction, and **preventing White Muscle Disease**.

Complex Syndrome of Young Lambs

- **Caused by** deficiency of vitamin E and selenium during gestation period or early life.
- Ch.by acute or subacute pathological degeneration and necrosis of skeletal and cardiac Ms.

Aetiology

Primary Def of Vitamin E and / or Se

Feeding rations which are deficient in vitamin E and / or selenium as feeding inferior quality hay or straw and a root crop.

Secondary Def of Vitamin E and / or Se

1. Feeding rations which are incorporated with excessive quantities of polyunsaturated fatty acids (myopathic agent).
2. Oxidation during rancidification of the oils causes destruction of vitamin E.

Occurrence

- All breeds, all sexes of lambs from birth to 3 months of age.
- Lambs whose dam heavily fed during pregnancy on poor feed, usually during spring.

Vit. A Deficiency

Characterized in

- **Young** animals by Nervous manifestations due to compression of the brain and spinal cord.
- **Adult** animals by Night blindness, Corneal keratinization, Pityriasis, Loss of weight and Infertility.

Aetiology

Primary Vitamin-A Deficiency

1. Deficiency of vit. A or its precursor carotene in the ration
2. Most commonly due to lack of green fodders or failure to add vit. A supplement to deficient rations

Secondary Vitamin-A Deficiency

1. Dietary supply of the vitamin or its precursor is adequate, but their digestion or metabolization is interfered
2. Mainly due to diseases of intestine and liver as most of carotene is converted in the intestinal epithelium and the liver is the main site of storage of the vitamin

Occurrence

1. Vitamin A present in two forms,
   a. **Alcoholic** form (carotene precursor of vit. A).
   b. **Ester** form (synthetic parenteral preparation).
2. **Alcoholic form** of vitamin A not pass through the placental barrier, while ester from has the ability to pass through the placental barrier so administration of such products in late pregnancy of the dam leads to increase of the hepatic storage of vitamin A in lambs or kids.
3. Vit. A is **Fat Soluble Vitamin**, so it is **rapidly oxidized** in the presence of unsaturated fatty acids or mineral oil as paraffin oil.
4. **High temperature, light or mineral mixture** also increase the destruction of vit. A in the commercial ration
5. **High Nitrate** decreases the conversion of carotene into vit. A.
6. Increase of the level of **Inorganic Phosphorus** cause decrease the storage of vitamin A in the liver while low level of phosphorus may lower the conversion of carotene into vit A.
7. **Vitamins E and C** prevent loss of vit. A from feed stuffs by their antioxidant action.

Vit. D deficiency

Vitamin D deficiency occurs in ruminants and result in:

- **Rickets** in young growing animals (lambs) Ch. by defective calcification of the growing bones. The poorly mineralized long bones are subjects to pressure distortions.
- **Osteomalecia** in mature animals affecting bones and Ch. by osteoporosis which manifested clinically by lameness and high susceptibility for bone fracture.

Aetiology

Primary Vit. D Deficiency

1. Deficiency of one or more of calcium, phosphorous and vitamin-D in ration.
2. Primary calcium deficiency due to lack of calcium in ration seldom occurs.
3. Phosphorous deficiency is usually "Primary "under field conditions, and may be exacerbated by a deficiency of vitamin D and possibly by an excess of calcium.

Secondary Vit. D Deficiency

1. Increase of carotene intake.
2. Insufficient solar irradiation of the animal.
3. Heavy coat is a predisposing factor as it insulates the skin from solar irradiation.
4. High levels of molybdenum or copper.

Vit, E (Selenium) Deficiency (Complex Syndrome of Young Lambs)

Pathogenesis

Ingestion/ Source of SE & Vit E

1. Selenium in the soil absorbed by plant and combined with sulphur containing amino acids as cysteine and methionine (selenium cysteine and selenium methionine) in the rumen, by the effect of microorganisms, these transformed to **Selenioprotein** (selenioamino acids) selenioprotein reach the blood, placenta and milk to give **Glutathione Peroxidase Enzyme** (**GSH.PX**).

Role of Selenium & Vit E:

2. Selenium and vitamin E are antioxidants therefore, deficiencies of these nutrients lead to oxidative damage to cells within the body. The muscle cells are the most vulnerable to damage in livestock species.
3. Main biological function of Vit. E is to control the rate of tissue oxidation and consequently prevention of muscular dystrophy.
4. Selenium appears to have a sparing effect on vitamin E also it has a prophylactic effect against enzootic muscular dystrophy as it enhances the transport and retention of vitamin E.
5. **GSH.PX** is **Antioxidant Enzyme**, his function is protection of all cells from oxidative damage through get rid of free radicals as peroxide (H2O2).

Deficiency:

1. With ration containing an increased amount poly unsaturated fatty acid and deficient of vit. E and selenium, the animal has low level of GSH-PX. This together with muscular exercise and provision of oxygen radical lead to excessive formation of H2O2, followed by:
   a. Hyaline degeneration.
   b. Coagulative necrosis.
   c. Swelling of muscle.
   d. Precipitation of calcium.
2. Acute muscular dystrophy results in the liberation of myoglobin into the blood stream and myoglobinuria.
3. **GSH-PX** play an important role in the regulation of **Glucose-6 Phosphate Dehydrogenase Enzyme (G.6.P-D**) which is very essential for maintaining of RBCs fragility.
4. So, deficiency of selenium causing deficiency of GSH-PX and subsequently deficiency of G.6.P-D enzyme causing haemolysis of RBCs and anemia.

Clinical Signs

A. Skeletal muscular dystrophy (subacute form)

1. May appear at birth or 2-4 weeks of age.
2. Dyspnea & Tachycardia.
3. Firm & swollen muscles.
4. Goose stepping gait.
5. Knuckling at fetlock.
6. Locomotor weakness & unsteadiness.
7. Reclining position.
8. Standing on the tip of toe.
9. Sternal recumbency.

B. Myocardial muscular dystrophy (Acute form)

1. Affected animals commonly die within 6-12 hours without previous signs.
2. Dyspnea and death.
3. Frothy nasal discharge.
4. Lateral recumbency.
5. Pulmonary edema.
6. Pulmonary edema.
7. Weak pulse.

Vit. A Deficiency

Pathogenesis

A. Night vision:

- Vit. A is essential for regeneration of the visual purple necessary for vision in dim-light.

B. Bone growth:

1. Vit. A is essential for normal bone growth as its responsible for maintain normal position and activity of osteoblasts.
2. Therefore, hypovitaminosis of vit. A causes incoordination of bone growth e.g.: facial paralysis and blindness due to construction of facial and optic cranial nerves.

C. CSF pressure:

1. Vit. A deficiency causes an increase in CSF pressure due impaired absorption of arachnoid villi and thickening of C.T. matrix of dura matter.
2. Increases CSF pressure causes convulsions and syncope.

D. Epith. Tissue:

1. Vit. A is essential for normal epith. Tissue and its deficiency led to atrophy of all epith. Cells particularly epith. Secretary cells, and these secretary cells are gradually replaced by stratified, keratinizing non-secretary cells.
2. Examples of organs and systems be affected are, eyes, salivary gland, thyroid gland, urogenital tract particularly placenta.

E. Immune mechanisms:

Vit. A deficiency associated with immunosuppression and susceptibility to infection.

F. Embryological development:

1. Vit. A is very essential for the development and growth of fetus.
2. Vit. A deficiency of pregnant dam may lead to some congenital abnormalities of the lamb or kid which Characterized By:
   a. Blindness due to constriction of the optic canal.
   b. Hydrocephalus.
   c. Dilated pupil.
   d. Nystagmus.
   e. Weakness and incoordination.

Clinical Signs

1. **Night blindness:** Inability to see in dim- light.
2. **Changes in skin:** Heavy deposits of bran-like scales on skin.
3. **Xerophthalmia:** Thickening and clouding of cornea.
4. **Reproductive efficiency:**
   a. **In Male** "Retained Libido" and degeneration of germinative epith. of seminiferous tubules causing reduction in number of motile, normal sperms.
   b. In **Female:** Abortion due to placental degeneration and birth of dead or weak young plus retention of placenta.
5. **Nervous sings:**
   a. Total blindness of both eyes due to construction of the optic nerve canal (manifested by **absence of menace reflex**).
   b. Encephalopathy, manifested by **Convulsive Seizures** due to increased C.S.F. pressure.
   c. Affected animals may **Collapse** (**Syncope**) and may **die** during episode.
6. **Congenital defects:**
   a. **Congenital blindness** due to construction of optic nerve.

Vit. D deficiency

Pathogenesis

1. Vitamin D is a complex of a substance with anti-rachitogenic activity and the important components of it include:
   a. **Vit. D2:** is present in sun-cured hay and is produced by ultraviolet irradiation of plants.
   b. **Vit. D4, D5** in fish oils.
   c. **Vit. D3**: (cholecalciferol) is produced from its precursor **7-dehydrocholestrol** in mammalian skin by natural irradiation with ultraviolet light.
2. Vitamin D which produced in the skin or ingested in the ration absorbed in the intestine is transported to the liver where it is transformed to 25-hydroxy cholecalciferol.
3. Which transport to the kidney in which at least two additional derivatives are formed by 1-hydroxylase enzyme including:
   a. 1, 25- dihydroxy cholecalciferol.
   b. 24, 25 dihydroxy cholecalciferol.

- In case of Calcium need or deprivation, the most predominant form that produced in the kidney is 1, 25-dihydoxy cholecalciferol (metabolic form of vit. D) which is important in mineralization of bones so deficiency of vit. D leads to disturbance of calcium and phosphorus absorption and metabolism.

In rickets

1- Deficiencies of Ca, ph. and vit. D result in defective mineralization of the osteoid and cartilaginous (calcification) matrix of the developing bones.

2- Poorly calcified spicules of diaphyseal bone and epiphyseal cartilage subjected to normal stresses resulting in bowing of long- bones and broadening of the epiphyses with apparent enlargement of the joints.

In osteomalecia

1- The drain of pregnancy and lactation caused resorption of bone minerals which leading to osteoporosis and bone weakness.

2- Bone fracture are commonly precipitated by exercise or any abnormal movement.

Clinical Signs

In Rickets

1. Stiffness in gait, lameness and tendency to lie down.
2. Abnormal curvature of long bones, usually forward and out word particularly at knee joint.
3. Enlargement of the limb joints, especially of the fore legs.
4. Enlargement of costochondral junctions (Rickets Rossetti).
5. Arching of the back.
6. Softness of jaw bones severely affected animals unable to close their mouth and tongue protrudes.

In Osteomalecia

1. In the early stages; the signs are those of phosphorus deficiency including:
   a. Licking and chewing of inanimate.
   b. Infertility and lower reproductivity.
2. In the latter stages the signs are specific to osteomalecia including:
   Painful condition of bones and joints manifested in animals by "moderate lameness".
3. Crackling sounds while walking.
4. Arched back.
5. Hind legs are most severely affected, and the hocks may be rotated inward.

Vit, E (Selenium) Deficiency (Complex Syndrome of Young Lambs)		Vit. A Deficiency	Vit. D deficiency	
		b. **Hydrocephalus** and **Encephalopathy**. **c.** **Dilated** pupil, **Nystagmus** and **Incoordination** and **Weakness**. **7.** **Other diseases:** Enteritis, Otitis media, Pneumonia.	**7.** Dental abnormalities delayed and irregular teeth eruption and teeth are poorly calcified with pitting, grooving and pigmentation. **8.** Dyspnea and chronic ruminal tympany may resulted from deformity of chest. **9.** Hypersensitivity, tetany and recumbency in final severe stages.	**6.** Fractures of bones and separation of tendon attachment without apparently precipitating stress. **7.** Dystocia when the pelvis bones is affected. **8.** Final weakness led to permanent recumbency and death from starvation.
PM lesions				
1. White to pale lesions of muscles including intercostal muscles. **2.** Presence of linear white streaks due to affected fiber bundles within the muscles. **3.** Petechial haemorrhage and muscular edema may occur. **4.** Same lesions present in cardiac muscle especially sub-endocardial muscles and may extend to septum. **5.** If death due to left ventricular failure, there is pulmonary edema.				
Diagnosis				
1. Case history and clinical signs. **2.** PM Lesions. **3.** Histopathological lesion: hyaline degeneration (coagulative necrosis). **4.** Laboratory diagnosis: **c.** Estimation of muscle specific enzymes **1)** Creatine phosphokinase (CPK) as high as 5000 – 10000 iu/L (N= 25- 65 iu/L). **2)** SGOT (AST) up to 300-900 iu/L (N up to 100 iu/L). **d.** Determination of vit. E status. **e.** Determination of selenium status (N- 0.05-0.06 ppm). **f.** Determination of Glutathione peroxidase (GSH-PX).		**1.** Case history and clinical signs. **2.** Laboratory diagnosis. **A- Estimation of plasma and liver vit. A and carotene:** **1-** Plasma carotene vary largely with ration. The level of about 150 mg/d1 is optimum. **2-** Plasma vit. A conc. Ranged from 25-60 mg/d1. **3-** Hepatic level of carotene 0.5 -4.0 mg/gm liver. **4-** Hepatic level of vit. A 2-60 mg/gm liver. **B- Estimation of C.S.F. pressure:** **1-** C.S.F. is a sensitive early indicator of vit. A status. **2-** Normal C.S.F pressure of less than 100mm of water but abnormal more than 200mm. **C- Conjunctival smears:** - Increased number of cornified epith. Cells.	**1. Case history and clinical signs.** **2. Laboratory diagnosis** **Estimation of serum Ca, ph and vit. D:** **a.** Serum calcium level usually lowered only in the final stages to 8.5 mg% (N = 10-12). **b.** Serum phosphorous if the cause, its level will be below 3 mg % (N = 5-7). **c.** Low level of vitamin D3. **d.** High level of alkaline phosphatase. **3. Radiographic exam**. Of bones and joints: Rachitic bones have a characteristic: **a.** Lack of density. **b.** Ends of long bones have a moth-eaten appearance. **c.** Long bones have a concave or flat, contour, instead of normal convex. **4. Histopathological exam.** Of a small piece of costochondral junction.	
Treatment & Prevention				
Sheep, Goats & Lambs: Viteselen 15® 0.5 ml / 10 kg B.wt. By I/M or S/C injection.		**Treatment** • Generally, in hypovitaminosis – A, animals should be treated with vit. A at dose rate equivalent to 10-20 times daily maintenance (requirement in all species is 40 I.U/kg B.W.). **Prevention** • Providing the daily maintenance requirement of vit. A which not less than 40 iu/kg B.wt. daily and must be provided during pregnancy. • I.M. Injection of 3000-6000 iu/kg B.wt. Vit. A at intervals of 2 months give optimum plasma and hepatic level of vit. A. **N.B.:** Ideally the last injection should be given one month before parturition.	Lesser deformities recover with suitable treatment, but gross deformities usually persist. **1.** Administration of vit. D (AD3E). **2.** Supplementation of calcium (from limestone, bone meal) and phosphorus (bran, sodium acid phosphate, disodium phosphate) in the ration. **3.** Injection of calcium borogluconate I/V, I/M, S/C or calcium gluconate I/V or S/C (0.5 – 1ml/kg B.wt.). **4.** Exposure to the sun light early morning. **5.** Sun –dried hay is a good source of vit. D2. **6.** Fish liver oil is a good source of vit. D4 and D5. **7.** Irradiated dry yeast is probably the simplest and cheapest source of vit. D and can be mixed with grain feeds.	

Ovine Pregnancy Toxaemia (OPT) **Ketosis - Lambing Sickness - Twin Lamb Disease – Hypoglycemia - Post-Parturient Dyspepsia**	Hypocalcaemia "HC" **Milk Fever – Transport Tetany – Parturient Paresis**	Hypomagnesaemia **Grass tetany/Staggers - Wheat Pasture Poisoning.**
Definition		
It's one of most important metabolic disease of ewe due to **disturbance in the Carbohydrate and Fatty acids metabolism** which leading to Hypoglycemia, Ketonemia and Ketonuria & ↓ in the level of the liver glycogen. **Occurrence (Incidence or History)** **1.** All breeds of sheep and goats. **2.** **Second** and sub-sequence pregnancies. **3.** More common in aged or senile animals (5-10 y old). **4.** Thin or over conditioned ewes. **5.** Ewes bearing twins or triplets develop the disease during the last months of pregnancy (i.e., 4th months). **6.** The disease usually associated with nutritional disturbance as low carbohydrates in diet **specially in the late stage of pregnancy**.	It's acute metabolic disease of sheep characterized by tetany, incoordination, paralysis and coma, caused by an inadequate supply of metabolizable calcium. **Occurrence** **1.** All breeds, sexes & ages beyond weaning age. **2.** Lactating and pregnant ewes, ram & feed lot lamb. **3.** More common in last 6weeks of pregnancy or 1st week of lactation. **4.** After fasting/transportation of ram or feed lot lambs. **5.** Females which have multiple foetuses are higher susceptible than that which have single foetuses. **6.** Senile or aged animal due to lack of **5-Hydroxy proline** is very important in skeletal metabolism **and bone resorption**.	It's an acute metabolic disease of pastured sheep and goats characterized by Hyperesthesia and spasmodic tetany (clinic convulsion) caused by a depressed supply of metabolizable magnesium from prolonged consumption of lush immature grasses. **Occurrence** **1.** All breeds of sheep and goats. **2.** Lactating ewes that grazing in early spring pastures. **3.** More common in the first 4 weeks of lactation. **4.** All classes of sheep and goats are susceptible i.e., ram, feed lot lambs, kids ……. etc.
Aetiology and Pathogenesis		
Predisposing Factors **1.** Rapid growth of twine or triple fetuses. **2.** Stress as transportation or fear. **3.** Inadequate supplementation of glucose. **4.** Nutritional disturbance as low carbohydrates. **5.** In the last 6 weeks of pregnancy **a.** **Fetus** need about **70-80gm Glucose/day.** **b.** **Dam** require about **85-100gm Glucose/day**. **6.** Disturbances in Glucose and Fatty acids metabolism → Hypoglycemia → OPT. **Compensatory roles of Ewe against Hypoglycaemia** **1.** ↑ Consumption of grains as exogenous source of glucose. **2.** Maternal tissue Reduces its consumption of Glucose to provide the Glucose requirement for fetuses. **3.** Maternal tissue hydrolyses the storage fat (i.e., increase the fat catabolism). Glycerol→ gluconeogenesis → glucose **4.** Oxidation of fatty acids produce large amount of Ketone bodies (Acetone, Aceto-acetic acid & B-hydroxybutyric acid) → ketonuria and ketonemia. **5.** Accumulation of lipid (Tri-acyl glycerol) Causing fatty liver (P.M.). → Renal disfunction and death. **Stress and Low Caloric Intake** lead to **1.** Elevate plasma Cortisol level (↑ size of Adrenal gland). **2.** Inhibits glucose utilization by the tissue → Nervous manifestations. **3.** That manifested PM by enlarged adrenal gland. **Acc. to the cause pregnancy toxaemia is classified into** **a.** Primary PT. **b.** Fat ewe PT (high volume of fat tissue is oxidized). **c.** Starvation PT (low food intake). **d.** Secondary PT (Side effect of diseases e.g., diarrhea). **e.** Stress-induced PT.	**Predisposing Factors of low Ca level** **1.** Prolonged consumption of calcium deficient diet → Lower the plasma calcium by 15-35%. **2.** Fasting from 2-6 days, especially when combined with transport → lower the plasma calcium by 17-25%. **3.** Late pregnancy (last 6ws) or early lactation (1st. 10days) → Lower the plasma calcium by 50-60%. **4.** Ca depressing factors as administration of epinephrine or consumption of **Oxalate Containing Feeds**…etc. **5.** Acute sever enteritis → impaired absorption of calcium from the intestine. **6.** Ration high in "Ca" and low in "ph" in late pregnancy → **Depression** the activity of Parathyroid gland. **7.** Stress factors (Transportation, fear, fasting…etc.) →Increase of Corticosteroid and Epinephrine secretion **a.** Antagonizing of vitamin D3. **b.** Decrease the Calcium renal resorption. **c.** Decrease the Calcium absorption. **d.** Decrease the mobilization of Calcium from bone. **Hypocalcaemia occurs as a result of** **1.** ↑ The rate of Ca movement out of plasma (excretion). **2.** Decrease in Ca resorption (mobilization) from bone. **3.** Decrease in calcium absorption in the gut. **Calcium level is controlled by:** **Vit D3 -Parathyroid hormone - Thyrocalcitonin - Mg** **a.** Resorption of calcium from bone. **b.** Absorption of calcium form intestine. **c.** Reabsorption of calcium from renal tubules. • Presence of **Calcium Depressing Factors** + **Inability** of the animal **to Mobilize the Calcium rapidly from Skeleton** (Calcium resorption) → **Serious and fatal "Hypocalcaemia".**	**Physiological point of view** **1.** The normal plasma level of magnesium and calcium is 2-3 mg%, and 8-12m% respectively. **2.** The Ca and Mg homeostasis are controlled by a complex mechanism under the influence of Parathyroid Hormone and vit. D3. **3.** Various factors that may contribute to a decrease in serum magnesium. Concentration as: **a.** Nor epinephrine and corticosteroid hormones (stress). **b.** Exposure to cold along with in adequate energy intake. **c.** Increased the dietary potassium intake. **Pathogenesis:** **1.** Lush grasses + Young wheat plants + Ammonium fertilizers + High concentration of protein, potassium and Ammonia → Absorption of ammonia by plants → Reduction of uptake of Ca and Mg. **2.** Eating of these plants by the animal causes **a.** High concentration of ammonia in the rumen. **b.** Reduction of availability of Ca and Mg. **c.** ↑ Level of ruminal PH **d.** Depletion of carbohydrate. **3.** **IF Mg level is below 1.8mg%;** Movement of Mg from bones to → Extracellular spaces. **4.** **Mg level is below 0.7mg%;** Irritability develops. **5.** **Mg level is below 0.5mg%** → Fatal tetany and convulsions → Death.
Clinical Signs		
1. Isolation from flock and declines feed (anorexia). **2.** In coordination in movement (forced to move, it blunders into objects). **3.** Elevation of head and pricked ears that give what so called "**Listening attitude**" or "**Star Gazing Position**". **4.** Accelerated respiration with mucous nasal discharge. **5.** Sternal recumbency and rises only by assistance. **6.** Neurological signs such as blindness, muscular tremors, convulsions, coma and finally death. **7.** Morbidity rate about 20% of flock and mortality rate may reach 80%. **8.** Uremic or acetone Adour of the mouth. **PM Lesions** **1.** Thin or over-Conditioned ewes. **2.** Uterus contains 2-3 fetuses. **3.** Fatty liver (friable liver and pale yellow in color). **4.** Enlarged adrenal gland by as much as 65%. **5.** Acetone odour of the carcase.	**Normal Ca level in nearly 8-12 mg.** When the level :3-6 mg clinical findings of hypocalcaemia recorded. **1.** Decrease in ruminal motility and uterine tone in case of pregnant ewe. **2.** Cervico-vaginal prolapse with complete or non-dilated cervix. **3.** In early stage, there is stiffness and in coordination. And abnormal stance, especially in the hind limbs. **4.** Muscular tremors, muscular weakness, apprehension and rapid breathing. **5.** Ewes in advanced stage fall to lateral recumbency and the hind limbs are extended to back "**Frog attitude**". **6.** Death occurs within 4-48 hours if not treated by Ca. **7.** **Normal or subnormal Body Temperature**. **8.** If the calcium level reached dangerous level near 3 – 6 mg%, Irritability develops, followed by Depression and Coma and most animals die.	**1.** Morbidity rate about 20%. **2.** Mortality rates up to 80% of untreated cases. **3.** Course about 2-24 hours. **4.** Clinical signs are of central origin and related to reduction in Mg of the cerebrospinal fluid. **5.** Hyperesthesia (hypersensitivity to external stimuli as noise or light…. etc). **6.** Accelerated breathing – Tremors and in coordinated movement. **7.** In advanced cases there are convulsion, recumbency and coma then finally **death due to Resp. failure.** **8.** Normal body temperature → Convulsions → High body temperature. **NB** Mg ions act as activator for cholinesterase responsible for muscle relaxation **9.** Elevation of head and pricked ears that give what so called "Listening attitude" or "**Star Gazing Position**". **10.** Foamy salivation, Spasm of the face **muscles**

Ovine Pregnancy Toxaemia (OPT) Ketosis - Lambing Sickness - Twin Lamb Disease – Hypoglycemia - Post-Parturient Dyspepsia	Hypocalcaemia "HC" Milk Fever – Transport Tetany – Parturient Paresis	Hypomagnesaemia Grass tetany/Staggers - Wheat Pasture Poisoning.
Diagnosis		
A. Case History of Occurrence, Incidence and Predisposing factors **B. Lab. Findings** **1.** Hypoglycemia the glucose level is below 25mg% (Normal = 40 – 60 mg%) **2.** **Ketonemia** 10-1000mg% (Normal = up to 10 mg%) **3.** **Ketonuria** 80-1300mg% (Normal = up to 50-70 mg%) **4.** **Milk ketones** 40mg% (Normal = 3 – 10 mg%) **5.** **High level of BUN** and **Creatinine** in the **terminal stag**e due to **decomposition of lambs** (**fetuses**). **6.** High level of cortisone is diagnostic in ewe. **7.** Iron, magnesium, potassium low level **8.** **Rother's test Used** for detection of ketonuria as the following: **a.** 5 ml of urine of suspected case. **b.** Ammonium sulphate until fully saturation of the urine sample. **c.** 1 ml of concentrated ammonium solution on the wall of tube. **d.** Few drops of Sodium Nitroprosside. **e.** Violet ring appears in positive case. **C. PM Lesion.** **D. Differential Diagnosis:** With Hypocalcaemia. In which **1.** The animal takes "**Frog Attitude**" lateral recumbence and head is reflected on the shoulder toward the flank region with extension of the hind limbs. **2.** Response to calcium administration. **3.** Low calcium level (N = 8-12mg%). **4.** Negative Rother ´s test.	**A.** Case history, predisposing factors and clinical signs **B.** Laboratory examination **1.** Calcium level lower than 8mg% (N = 8 -12mg%). **2.** Phosphorus may lower than 4mg% (N = 7-9mg%). **3.** Magnesium may lower than 2mg% (N = 2-5mg%) (Ca: Ph: Mg = 2:1:0.5) **4.** Eosinophilia, Neutrophilia and Lymphopenia due to the adrenal cortical hyperactivity.	**A.** History and clinical symptoms. **B.** Laboratory diagnosis (magnesium level about 0.50-0.25mg/dl) **C. Differential Diagnosis with** **1.** Hypocalcaemia in which **(1)** Calcium level below 4mg%. **(2)** Animal gives response to calcium administration **2.** Enterotoxaemia in which **(1)** Sudden death. **(2)** High body temperature. **3.** Presence of toxins in the intestine.
Treatment and Prevention		
1. Precursors of glucose **2. Correction Of Hypoglycemia** by injection of glucose preparation I.V. (R/Dextrose 5, 20, or 40% (250-500ml). **3. Correction Of Dehydration** and acid- base imbalance by injection of fluid therapy (R/-Ringer's lactate or normal saline or sod. Bic. I.V.) **4. Stimulation Of Depressed Appetite** by giving of stomachic (R/-Stamatone-Digestin-Uccamdigest. etc.) **5.** Administration of vit B-complex or Calcium to improve rumen motility "50125ml 20% Calcium borogluconate". **6.** Oral Propylene glycol or Corn syrup as source of energy (200ml 4 times daily along with 3-4 litres of fluid therapy.). **7.** Glycerol 4 oz. (120ml glycerol + 120ml worm water orally) i.e., 50% glycerol. **8. Good management**: Feeding on concentrate or hand feeding may be necessary. **9. Induction of abortion**/Surgical interference by caesarean section. **Control** Treatment is not usually very effective; therefore, prevention is the best way of dealing with ketosis. **1.** Avoiding over fattening or starvation **2.** Prophylactic dose of sodium propionate as it acts as Anti-ketogenic and it's glucogenic substances. **3.** Exercising of ewe specially in late pregnancy. **4.** Avoiding stress factors as transportation…etc. **5.** Feeding on concentrate contains 10% proteins 0.25 kg/day and increased to kg/ day at last 2 weeks giving good protection. **6.** Ground maize Due to it contains good amount of alpha-polymerized glucose which not fermented in the rumen and pass to the intestine and absorbed there. specially during late pregnancy.	**1.** Most sheep affected by Hypocalcaemia give good response to calcium treatment 50-100 cm Calcium gluconate I.V. or s/c. with vit D. **2.** Preparation of combination of Calcium, Phosphorous and Magnesium. **3.** Avoiding all mentioned etiological and predisposing factors. **4.** Massaging of hind limbs and avoid milking at least for 48 hr.	**1.** Successful treatment of mainly depends upon early diagnosis and prompt intravenous injection of one of indictable products of HM which contains: Calcium borogluconate, Dextrose and magnesium borogluconate as R1: Cal. D. Mag & Cal bor mag – Cal phormag…. etc. **2.** Vit D3 which helps in absorption and mobilization magnesium as R/ D-Verol injection **3.** Supportive treatment as fluid therapy and dextrose. Muscular relaxant for hyperesthesia as: R/Neurazine- or chlorpromazine……. etc. **Prevention** • Magnesium supplement especially in spring and autumn seasons, and the supplemental magnesium may be provided through animal mixture containing. **a.** 400 kg Calcium carbonate. **b.** 200kg trace mineral salt. **c.** 150kg Magnesium oxide **d.** 200kg ground corn or other concentrates. **NB.** 65kg lactating ewe requires from 43 to 96 mg/magnesium daily. i.e., 7-14gm of the above mineral mixture (i.e., 2.5gm/10kg b. wt.).

3 Small Ruminants, Skin Diseases

1. Photosensitization "Big head" swollen head" and clover disease	2. Eczema or False Mange	3. Dermatitis
Definition		
It is an acute dermatitis in non-pigmented sheep characterized by irritation, edema, sometimes anasarca (generalized edema) and necrosis of ears, eyelids, lips, face, vulva and cornet. It is caused by interaction of photodynamic pigments and sunlight. **Occurrence:** **a.** Occurs in all sheep-producing countries. **b.** All breeds, ages and sexes of non-pigmented sheep	Eczema means inflammatory reaction of the epidermal cells due to presence of substances which sensitize or stimulate the inflammatory process in the skin and such substances may be internal or external.	Dermatitis means inflammation of the skin including epidermis, dermis and may include blood vessels and lymphatics,
Aetiology		
1. Primary photosensitization: due to feeding on uncommonly plants contain photodynamic pigment as saponin or mycotoxin ...etc or some drugs as phenothiazine. **2.** Secondary photosensitization (hepatic) due to effect of Pyrrolizine, alkaloid containing plants (rare in sheep) or diseased liver.	**A. External** **A.** Using of strong or concentrated disinfectants as exogenous. **B.** Use of antiseptics on the skin. **C.** Administration of iodide, mercuric or arsenic drugs **D.** External parasites (mange) **E.** Prolonged wetting of the skin. **B. Internal (endogenous):** As absorption of toxic amines as in cases of toxic hepatitis, nephritis, constipation or ingestion of certain proteins. Allergens are absorbed and reach skin through blood circulation.	**A. Bacterial dermatitis: due to** **1.** *Staphylococcus aureus* **2.** *Corynebacterium pyogene* **3.** *Streptococcus spp.* **4.** Strawberry foot rot and *Dermatophilus pedis*. **B. Viral dermatitis: due to** **1.** Pox, and foot and mouth disease **2.** Ulcerative dermatosis **3.** Contagious ecthyma or blue tongue. **C. Mycotic dermatitis: AS** **1.** Ring worm. **2.** *Dermatophilus congolensis* **3.** Lumpy wool **D. Metazoan dermatitis: As** **1.** Sarcoptic and Psoroptic mange. **2.** Blowfly infestation (cutaneous myiasis). **E. Physical** dermatitis: as **1.** Exposure to so cold or hot preparations **2.** Photosensitization (primary or secondary) **F. Chemical dermatitis as** **1.** Due to strong acid as H_2SO_4 **2.** Arsenic preparation either local or parenteral with high concentration. **G. Allergic dermatitis** → as certain oil, soap or another antigen **1.** (External or internal antigens) as mentioned before in eczema. **H. Nutritional dermatitis** → as zinc deficiency, vit. A deficiency and vit. B deficiency.
Pathogenesis		
1. Normal metabolism of plant producing pigment in ruminant can be summarized as follow **2.** Plant (contains chlorophyll) (in rumen by normal/ microbial Action) → **Phylloerythrin** → Absorption by portal circulation then to liver then reach the bile for excretion and consequently the peripheral blood circulation is free from phylloerythrin. **3.** With liver disease → Generalized **Phylloerthrinemia**. **4.** Phylloerythrin in the skin absorb the sun light and activated (active form) in presence of protein molecules and amino acids and oxygen → it leads to the formation of toxic compounds and release of histamine → increase the permeability of capillaries to plasma proteins and consequently force the water into the affected tissue and increase the hydrostatic pressure of blood → edema and necrosis. **5.** Death due to secondary bacterial invasion, liver dysfunction, inability to feed and unthriftiness **6.** NB- sheep that possessing haemoglobin A or AB resist to such conditions while that possessing haemoglobin B. are highly susceptible.	• Allergic or inflammatory reaction between the skin and allergens (either external or internal allergens) leading to → erythema then papule (cellular infiltration). Vesicles **1. Dry eczema** → characterized by absorption of contents of vesicles, then ulceration and scar formation. **2. Moist or wet eczema** → characterized by rupture of vesicles, oozing of the contents of the vesicles on the surrounding skin. **3. Pustular eczema** → characterized by that vesicle are invaded by pyogenic microorganisms and formation of pustules. **4. Crust eczema** → characterized by dryness of the vesicles and crust formation. **5. Chronic eczema** → characterized by occurring when any forms of the mentioned before is persisted more than 3 weeks.	

1. Photosensitization "Big head" swollen head" and clover disease	2. Eczema or False Mange	3. Dermatitis
Clinical symptoms:		
1. The time from ingestion of plants contain photodynamic pigment and appearance of clinical sign vary from several hours to 10 days. **2.** The disease begins with **a.** Erythema **b.** Photophobia **c.** Irritation of the lips, vulva, face, cornea, cornet and eyelids. **3.** Rubbing and kicking of the affected parts. **4.** Facial edema and in this stage, there are: **a.** Thickening of lip and immobilization. **b.** Constricted and swollen nostrils. **c.** Dropping of heavy ears. **d.** Animal suffering from dyspnoea and anorexia **5.** The diseased animal is separated from the flock and depressed.	**1.** Erythema, itching and rubbing themselves as similar to manage so it is called "false mange" **2.** Symptoms vary according to the form of eczema that mentioned before in pathogenesis. **3.** Morbidity up to 50%.	**1.** The affected skin area firstly shows erythema and increase in the warmth **2.** The subsequent stages vary according to the type and severity of the causative agent. **3.** There may be development of discrete vesicular lesions or diffuse weeping. **4.** Edema of skin and S/C tissue may occur in severe cases. **5.** The next stage may be healing stage with scab formation or if the injuries are more severe there may be necrosis or even gangrene in the affected skin area. **6.** Spread of infection to S/C tissue may leads to cellulitis or phlegmonous lesion or pyoderma (suppurative lesions). **7.** Systemic reaction is likely to occur when the affected area is extensive. **8.** Shock with peripheral circulatory failure, may be present with excessive burns. **9.** Toxaemia due to absorption of tissue breakdown products or septicemia due to invasion via unprotected tissues may occur in later stage. **10. Ovine atopic dermatitis:** **a.** The skin is affected by symmetrical erythema and alopecia. **b.** Only occasional sheep in the flock are affected usually each summer with remission during the winter months. **11. Caprine Idiopathic Dermatitis:** **a.** Alopecia, exudative dermatitis of all ages and both sexes especially in pygmy goat **b.** Scaling and crusting around the lips and chin, ears, perineum and ventral abdomen.
P.M Lesions		
1. Facial, vulvar, and pedal skin commonly is edematous. **2.** Generalized icteric carcase as in case of secondary photosensitization. **3.** Liver lesions may present as cirrhosis or bile obliteration.		
Diagnosis		
1. Case history **a.** Feeding on uncommonly plants **b.** Non –pigmented sheep **c.** Administration of drugs as phenothiazine. **2.** Clinical findings and PM lesions. **3.** Detection of the level of photodynamic pigment as phylloerythrin. **4.** Differential diagnosis: either due to primary or secondary photosensitization. **a.** Liver disease as aflatoxicosis, and liver abscesses. **b.** Other skin diseases.		**1.** Clinical features and history. **2.** Skin biopsy and skin scraping to detect bacterial or parasitic dermatitis respectively.
Prevention and Treatment		
1. Avoid grazing on pasture that containing photosensitizing agent. **2.** Removing the plant containing photodynamic pigments from diet. **3.** Rectal enema of affected animal. **4.** Antihistamines as. Avil, Vetibenzamine and Allercure. **5.** Anti-inflammatory as Dexamethasone, Dexatimanol and Finadin **6.** Diuretic as Lasix and sod citrate …etc. **7.** Cold bath and palatable food. **8.** Sheep with chronic liver disease rarely recover.	**1. Hygienic treatment** avoiding the sources of allergen either internal or external (by gastric lavage, purgation, and eradication of external parasites……etc). **2. Local treatment:** • In case of moist or wet eczema we can use dust powder over skin as R/tannic acid 5 gm + salicylic acid 5gm. • R/zinc oxide 10gm + talc powder 10gm. • R/-Sulphur powder 5gm. ▪ Alcohol sol. 40ml used as skin application • - Formalin sol 5ml • - Olive oil up to 100ml **3. Anaesthetic skin ointments as** Lignocaine ointment. **4. Systemic treatment such as:** **a.** Antihistaminic, corticosteroids and antibiotics. **b.** Treatment of hepatitis, and nephritis if suspected.	**1.** Removal of the offending cause. **2.** Application of local antibiotics or antifungal. **3.** Antihistaminic. **4.** Anaesthetic ointment when pain or itching is severe. **5.** Providing of Vit. B in the food. **6.** Astringent in weeping stage. **7.** If shock present, fluid therapy should be administered. **8.** Protein supplementation in diet for enhancing repair of skin. **9.** Vaccination against viral causes as pox.

4. Acne and Folliculitis or Sycosis	5. Parakeratosis	6. Hyperkeratosis
Definition		
Acne means infection of the sebaceous gland or hair follicle caused by acne bacilli or by any other suppurative microorganism as staph (usually termed sycosis).	Parakeratosis means the inadequate keratinization of epithelial layer of skin which mainly due to zinc deficiency.	It is extensive keratinization of epithelial layer of the skin, which may be: **a.** Local or generalized as: in Arsenic or Naphtholphthalein poisoning. **b.** Deficiency of vitamin A.
Aetiology		
1. Blockage of sebaceous gland ducts due to scab or desquamated epithelium or dilatation of such ducts may lead to reaching of the microorganisms to the hair follicles. **2.** Infection usually occurs by acne bacilli or staphylococcus usually associated with mange as predisposing factors (demodectic mange).	**1.** First, there is red area converted into gray coloured area. **2.** The lesions appeared on the thigh especially at area of the joints in the form of alopecia and the skin appeared as raw skin. **3.** The lesions may be cracked in network form and secondary bacterial invasion may occur.	
Pathogenesis		
Clinical Symptoms:		
1. Presence of painful papules at the base of the hair. These papules may become pustules due to invasion with suppurative microorganisms. **2.** Under pressure the pustules may be ruptured and spread the infection to the surrounding tissues with itching. **3.** Alopecia at the affected area due to shedding of the hair.	**1.** Clinical symptoms **2.** Detection of serum zinc level (0.02-0.04mg%).	**1.** Area of alopecia and increase in the thickness of the skin. **2.** Scale formation and dryness of the skin. **3.** Cracks and fissures in network form and secondary invasion may occur.
P.M Lesions		
Diagnosis		
1. Skin symptoms. **2.** Swabbing for bacteriological and parasitological examinations. **3.** Differential diagnosis with impetigo.		Clinical symptoms and skin scraping and biopsy
Prevention and Treatment		
1. Isolation of diseased animal. **2.** Application of local antiseptics. R/H_2O_2, Betadine, Savlon…etc). **3.** Antibiotics either local or systemic. R/Terramycin skin oint. and R/Terracortil oint. and R/Gentamycin oint. **4.** Trimethoprim with Sulphathiazine give good results.	**1.** Washing the skin by soapy solution or white lotion (2mg zinc sulfate + lead acetate 3gm+ 50ml distilled water) or salicylic acid ointment as astringent. **2.** Zinc oxide ½ gm orally/day for 3 weeks or addition of zinc sulfate to the ration.	**1.** Washing of the skin by soap water. **2.** Removing of the excessive keratinized layer by astringent as white lotion (Zinc sulfate 2 gm + Lead acetate 3 gm + 50ml D.W.). **3.** Supplementation of vit. A.

7. Urticaria	8. Alopecia
Definition	
Urticaria means allergic condition of the skin characterized by formation of plaque like eruption of the skin due to hypersensitivity reaction and excessive released of histamine.	Alopecia means loss of hair in goat or wool in sheep and it may be localized or generalized.
Aetiology	
A. Primary causes: **1.** Feeding on feed rich in CHO and protein with inadequate exercise. **2.** Recent sudden change in diet. **3.** Injection of some drugs as penicillin or serum **4.** Feeding on unusual food. **5.** Milk allergy after drying of udder. **6.** Constipation with gastroenteritis. **7.** Insect bites or sting. **B. Secondary causes:** **1.** Mainly due to inhalation allergic vapour or dust or respiratory tract infection. **2.** Destruction of cells and releasing of histamine.	**1.** Long period of starvation as sheep begin to eat the wool of each other especially at flank region. **2.** Overeating may lead to shedding of wool or hair. **3.** Recovery from high fever may leads to hair loss. **4.** Ring worm or mite infestation (mange) or severe wounds. **5.** Nutritional as Vit. A, zinc, cobalt, Iodine deficiency. **6.** Viral infection of dam during pregnancy. **7.** Inability of hair follicles to produce hair either due to hypothyroidism or infection of hair follicle itself (Acne or folliculitis). **8.** Deep wounds, which may destroy the hair follicles. **9.** Severe rubbing or itching of skin. **10.** Metabolic dermatitis. **11.** Some physical or chemical agents.
Pathogenesis	
Allergen [antigen] + antibody → Release of histamine → Vasodilatation of the peripheral blood vessels → Erythema → Increase the permeability of capillaries to plasma → Edema.	
Clinical Symptoms	
1. Restlessness and the lesions appear as circumscribed elevated flat toped area of the skin ½ -5 cm in diameter in different areas of the body. **2.** Erythema and itching specially in insect bites. **3.** Diarrhoea or fever may occur. **4.** Edema of ears, muzzle and vulva. **5.** Symptoms may disappear within 3-4 days or after lowering of the level of histamine to normal level.	**1.** Absence of wool or hair and consequently the animal become more susceptible to the external environmental conditions. **2.** Alopecia may be associated with itching or pruritis as in mange. **3.** Crimp loss of the wool. **4.** Broken wool and hair may be observed at the seat of alopecia.
P. M Lesions	
Diagnosis	
1. History and symptoms. **2.** Blood examination to detect eosinophilia.	**1.** Clinical symptoms and history to differentiate between nutritional and other types of alopecia. **2.** Skin scraping as in mange and ring worm. **3.** Blood examination to detect the level of zinc, cobalt copper and Vit. A.
Prevention and Treatment	
1. Purgative or gastric lavage to remove noxious food by using of R/Magnesium sulfate or vegetable oil or mineral oil. **2.** Antihistaminic as Avil, Allercure and Tavagyl...etc. **3.** Adrenaline 1 % as vasoconstrictor up to 5 gm lead acetate + 50 ml D.W.). **4.** Calamine lotion. **5.** Sodium bicarbonate wash. **6.** Calcium has antihistaminic effect as it affect on mast cells. **7.** Has vasoconstrictor effect as it increase the viscosity of the blood.	Depends mainly upon diagnostic type of alopecia according to its cause.

4 SMALL RUMINANT, GIT

Definition

Dental "Attrition" or "Tooth wear"

Dental attrition means rapid and excessive wear of teeth ch. by inefficient prehension, mastication, and utilization of food and caused by abrasion of soft teeth by diets.

Economic losses due to:

1. Culling of affected animals.
2. Lower production of meat and wool.

Occurrence:

1. occurs in all types of sheep.
2. Ewes 5 years old and old aged animals are higher susceptible.
3. Geographically, this disease occurs in all sheep producing countries especially those that have sandy pasture or arid and semiarid soil.

Periodontal Disease, Periodontitis, Periodontosis, and Broken Mouth

It is chronic infection of the sockets of teeth characterized clinically by loosening, lengthening, wearing, spreading, malocclusion and expulsion of an individual tooth and pathologically characterized by destruction of the periodontal ligament and osteopathy.

Occurrence:

1. Periodontal diseases occur in all breeds, sexes and ages but more incidence in 3 years old or older ewes than do other age groups.
2. Occur in all seasons, and other sheep producing countries.

Economic Loss Due to: unthriftiness, inefficient use of food and low production of meat and wool and early culling of premature animals.

Stomatitis

Stomatitis is an inflammation of the oral mucosa and includes

- Glossitis (Inflammation of the lingual mucosa).
- Palatitis (inflammation of the mucosa of the hard palate).
- Gingivitis (inflammation of the mucosa of the gums).
- Clinically it is characterized by partial or complete loss of appetite, by smacking of lips and profuse salivation. It is commonly an accompaniment of systemic disease.

Aetiology & Pathogenesis

Dental "Attrition" or "Tooth wear"

Pathogenesis of dental attrition usually related to the animal and the diet as

- Mastication of sand and soil along with prehended forage causes dental attrition.
- Calcium deficiencies with softness of tooth enamel and dentine may accelerate the rate of wear.
    1. Especially in 1st 18 month susceptible to softness of tooth enamel and dentine.
    2. Ewes with annual pregnancies lose their dental calcium.
- The animals affected become malnourished and die from exhaustion or systemic disease.

Periodontal Disease, Periodontitis, Periodontosis, and Broken Mouth

1. Usually occur in association with nutritional deficiency especially calcium together with bacterial infection as diphtheroid or Bacteroides.
2. The initial pathological changes probably begin with degeneration of cementum, periodontal fibers and bone or possibly with gingivitis.
3. **Small calculus attaches of the enamel at the gingival sulcus**, so movement of the tooth and calculus leading to erosion and ulceration of epithelium that predispose and help the entrance of bacterial infection to the ligament leading to periodontitis
4. **Repetition of such process**, leads to formation of pocket 1-2 ml deep, and inflammation of the circumference of the affected teeth.
5. Pathological changes may reach the alveolus and bacterial infection leading to abscess formation and in advanced case expulsion of the tooth occurs and the socket is filled with granulation tissues and covered over by epithelium.
6. The disease usually does not spread to other teeth
7. Acute or chronic gingivitis.

Stomatitis

1. Stomatitis in small ruminants usually related to infectious origin, but some etiologic consideration regarded to accidental origin.
2. Accidental incidence: include trauma while dosing, mal-occluded teeth and sharp awns and spines.
3. Infectious causes:
    a. Bacterial stomatitis due to actinobacillosis.
    b. Viral stomatitis due to contagious ecthyma, blue tongue, food and mouth disease and rinderpest.
    c. Mycotic stomatitis due to *Monilia spp*.

Pathogenesis

The causative agents being applied directly to be mucosa or gaining entrance to it by way of minor abrasions (primary*or by localization in the mucosa from viraemia (secondary)*. the clinical signs of stomatitis are caused by inflammation or erosion of the mucosa and the signs vary in severity with the degree of inflammation.

Clinical Signs

Dental "Attrition" or "Tooth wear"

1. Abnormal prehension and mastication.
2. Salivation, weakness and unthriftiness.
3. Emaciation and anemia.
4. Death due to starvation or pregnancy diseases.

Post-mortem examinations:

Teared teeth or erosions on the gum.

Periodontal Disease, Periodontitis, Periodontosis, and Broken Mouth

1. Loss of incisors teeth usually between 3.5 to 6.5 years while healthy sheep retain its teeth beyond 7 years of age.
2. Teeth movement associated with eroding of gum and forming of pocket.
3. Gingivitis which characterized by redness and oedema of attached gingiva.
4. Incisors and molar teeth usually affected.
5. Lengthening of teeth, heamorrhage of gum, loosening of teeth and periodontitis are characteristic.
6. Unthriftiness and starvation due to inability to fed with loss of body weight.
7. Abscess formation may be detected due to bacterial infection.
8. Digital pressure may express exudate around the tooth.
9. Difficulty in prehension and mastication.
10. Salivation and dropping of fragments of food from mouth during the chewing movement.
11. Hypertrophy of the gum.
12. Incidence varies up to 10% and course extends through many months.

Stomatitis

1. Partial or complete anorexia.
2. Slow and painful mastication.
3. Chewing movements and smacking of the lips are accompanied by salivation either frothy and in small amounts, or profuse and drooling.
4. The saliva may contain pus to shreds of epithelial tissue.
5. A fetid odour is present on the breath if bacterial invasion occurs.
6. Swelling the face is observed only in cases where cellulitis has extended to involve the soft tissue.
7. Polydipsia and the animal resent examination of the mouth.
8. Toxaemia may be present when stomatitis is secondary to systemic disease or where tissue necrosis occurs. This is a feature of systemic viraemia.
9. Oral lesions differ according to the cause as with contagious ecthyma there is necrotic tissues on the tongue, there is haemorrhages in oral and buccal tissues and erosions on lips, dental pad, tongue and on papillae of buccal cavity.
10. In case of F.M.D, vesicles formed in the mouth in the dental pad and less commonly on the dorsum of the tongue. After rupture, the vesicated tissue is hyperaemic, swollen, raw and tender. With Rinderpest, the soft palate contains erosions.

Dental "Attrition" or "Tooth wear"

Diagnosis

1. History and clinical signs.
2. Differential diagnosis includes periodontal disease and chronic progressive pneumonia.

Prevention And Treatment

1. Calcium deficiencies should be avoided and treated by supplementation of balanced diet and injection of calcium borogluconate respectively.
2. 1% ground limestone in the ration supplies adequate for calcium.
3. In the autumn, all sheep should be examined or unsound teeth, and all animals showing excessive dental wear should be culled and marketed or maintained on pre-ground feed.
4. In winter, supply sufficient feed to avoid grazing and reduce the amount of masticated soil or sand.

Periodontal Disease, Periodontitis, Periodontosis, and Broken Mouth

Diagnosis

1. History and clinical signs.
2. Laboratory examination by isolation of spirochetes and fusobacterium.

Prevention And Treatment

1. Avoid nutritional deficiency.
2. Providing of mineral mixture.
3. Antibiotics to overcome the infection

Stomatitis

Diagnosis

1. Case history and clinical findings.
2. Careful clinical and necropsy exam. to define the type and extend of the lesions.
3. Laboratory examinations by taking a swab for microbiological examinations.

Prevention And Treatment

A. **Hygienic Treatment:**
   1. Affected animals should be isolated fed and watered from separate utensils if an infectious origin is suspected.
   2. Easily digestible feeds must be affected including bran mash.

B. **Supportive Treatment:**
   1. Fluid therapy must be indicated with animal with bad condition. Fluid therapy includes isotonic saline NaCl 0.9% or dextrose 5% or dextrose saline.

C. **Medicated Treatment:**

1. Flushing of oral cavity with mild antiseptic solutions including Clinitol, Sulphaboracyle, 2% Copper sulphate, 2% Potassium Chlorate and 2% Alum.
2. Ulcers require curettage or cauterization with iodine solutions.
3. With systemic disturbance we must give broad spectrum systemic antibiotic as Ampicillin, Amoxicillin Penicillin/ Streptomycin, Oxytetracycline and triple sulpha.

Oesophageal Obstruction / Oesophageal occlusion / Choke

Definition

Choke is the accidental lodgement of the Esophagus by ingesta or foreign body, and such obstruction may be partial or complete.

Occurrence:

Occurs in all breeds, sexes and more common in 4-6 months of age specially in feedlot lambs.

More common in late summer and early fall especially with weaned lambs grazing in harvested fields.

Aetiology & Pathogenesis

1. Eating globular food,
2. The foods similar to corn cobs, sugar beet, potatoes and carrots.
3. When the foreign body or globular food which has larger diameter than that of the Esophagus lodged in the Esophagus it leads to:
4. Pressure on the wall of Esophagus causing its necrosis.
5. Accumulation of saliva cranial to the obstruction.
6. Prevent the eructation so leading to accumulation of gases in the fore stomachs and bloat or tympany (free gas tympany)
7. Saliva is regurgitated from mouth and nostrils and some of it may enter the larynx and trachea which may lead to aspiration pneumonia.
8. The obstruction in sheep usually occurs in the cervical part of the Esophagus but it may be occurred in the thoracic part near to the cardia.
9. If the animal neglected it will be dying from pneumonia or bloat or asphyxia.
10. Oesophageal obstruction may occur due to external pressure on the Esophagus as in case of enlargement of mediastinal lymph node or carcinoma of stomach leading to obstruction of the cardia.
11. The onset of the disease is sudden and the animal is restless and alarmed.
12. Strong and repetitive movements of swallowing and mastication (dysphagia).
13. Extension of the head and neck.
14. Regurgitation of food immediately after swallowing and if food only is regurgitated this indicated incomplete oesophageal obstruction while if water and food is regurgitated this indicated complete oesophageal obstruction (bad prognosis).
15. If the obstruction close to the larynx it leads to cough and Dyspnea.
16. Bloat is developed rapidly after obstruction.
17. If the lodged material present in the cervical part of the Esophagus it can be palpated (gently) from left side. If the animal is neglected, it will die due to pneumonia, asphyxia and bloat.

Clinical Signs & Post-mortem Examinations

1. Lodged food particles or foreign body in the Esophagus.
2. Necrosis of oesophageal wall at the site of obstruction.
3. Trachea may contain aspirated fluid and pneumonia.
4. Tympanic animal.

Diagnosis

1. History, clinical signs and PM lesions.
2. Confirmatory diagnosis by
3. Passing of stomach tube (pass in partial obstruction –not pass in complete obstruction)
4. Palpation of Esophagus at left side.

Prevention And Treatment

A. **Hygienic treatment**: prevention of food intake completely and excessive water intake.

B. **Supportive treatment**: Parenteral feed as fluid therapy (Dextrose 5 or 25% and isotonic saline I.V).

C. **Medicated treatment:**

1. Give tranquilizer to calming the affected animal (R/-Rompun or Neurazine or Chloral Hydrate).
2. relieve the bloat by trocar and canula or large gauge needle.
3. In case of cervical obstruction, the lodged particle can be pushed forward and upward by gentle massage of Esophagus or taken up by long forceps.
4. In case of obstruction in the thoracic part of the Esophagus we can push it by stomach tube toward rumen.

D. **Surgically** by esophagectomy or Rumenotomy.

Ruminitis

Definition

Acute or chronic focal inflammation of the rumen caused by endogenous or exogenous irritant and microbial pathogens.

Occurrence: Rumenitis occurs in all breeds sexes and ages but more common in the feedlot lambs.

Aetiology & Pathogenesis

Pathogenesis

1. The diseases usually begin with the action of lactic acid (lactic acidosis) or virus or poisonous heavy metals on the mucosa.
2. Concentrated irritants on mucosa lead to its inflammation, ulceration and necrosis.
3. The continuous movement of the ruminal contents leads to erosion of the ulcerated or necrotic mucosa and may reach the lamina propria and stimulate formation of granulation tissues.
4. Bacteria of the rumen specially fusobacterium necrophorus can penetrate the wall of rumen and reach the blood circulation and may localized in the liver leading to abscesses formation in the liver.

Bloat or Tympany or Tympanitis

Definition

Bloat is the retention of gas in the rumen and characterized by increase the intra-abdominal and intra-thoracic pressures and caused by complex interactions of diet, animal and microbial factors.

Economic losses due to: Death, unthriftiness, high costs of therapeutic and prevention and incomplete utilization of bloat provoking feeds.

Types

A. Frothy bloat due to either:	**Occurrence**
1. Green Legumes Bloat (GLB)	High in lambs from 3 to 9 months old age during plant growing season.
2. Hay Legumes Bloat (HLB)	During all seasons.
3. Grain Concentrate Bloat (GCB)	In late summer and early fall following weaning for feedlot lambs.
B. Free gas tympany.	

Aetiology & Pathogenesis

- Gases normally produced in the rumen as a result of aerobic fermentation and neutralization of salivary bicarbonate. Gases are CO_2 (45-75%) Methane "CH_4" (20-30%), N_2, O_2, H_2 and H_2S with minor amounts.

N.B.: Bloat due to putrefaction of protein in rumen leads to production of amino acids as tryptophan, which are converted into serotonin, which has spasmodic action of the sphincter of the cardia.

A. GLB:

- Grazing on pastures containing clover or alfa alfa and green legumes, there is increased production of gases due to:

1. Acidification of dissolved bicarbonate.
2. Decarboxylation of organic acids.
3. Slime layer or surface-active material leads to prevention of the eruption of gases into the free layer.
4. The source of slime layer or surface-active material is dissolved proteins, saponins and pectin which concentrated in the leaves.

B. HLB

Nearly similar to Green Legumes Bloat, but it is due to feeding on whole chopped, ground or pelleted alfa alfa or clover hay which is conductive to bloat when highly leafy, recently cured and combined with barely and constituting more than 10% of the ration.

C. GCB

Resulted from feeding on bloat producing concentration especially corn, barely, soybean meal and ration containing less than 10% roughage also favor development of bloat.

Gas produced from

a. Acidification of saliva dissolved bicarbonate

b. Microbial fermentation. Slime layer formed by bacterial capsular slime composed of nucleoproteins and polysaccharides.

- Deficiency of mucin in the saliva may leads to bloat (mucin act as antifoaming agent).
- GLB, HLB and GCB lead to hindrance of the collection of gases in free layer due to the active surface slime layer so it leads to blocking of oesophageal sphincter.
- **Free Gas Tympany**: Occurs when there is hindrance to escaping of gas from the rumen as in case of ruminal atony. This also occurs as a result of pressure on the cardial sphincter of Esophagus due to enlarged lymph node, abscess and tumor. Choke prevent the escape of gases through the Esophagus.
- In severe cases of bloat there is an increase the intraruminal pressure leads to pressure on the diaphragm causing respiratory distress and increase the intrathoracic pressure. Increase of the intraruminal and intrathoracic pressure lead to pushing of the blood to the peripheral circulation away from viscera helping in increase of the dissolving CO_2 in the blood and acidosis or acidaemia.

Ruminal Parakeratosis

Definition

Nutritional degenerative change in the mucosa of rumen characterized grossly by browning, hardening, thickening and clumping of papillae and microscopically by an accumulation of excessive layers of keratinized squamous epithelial cells on the papillary surface, and caused by diet of finely ground feed.

Occurrence

1. In all breeds and sexes of fattening animals specially 3-9 months old age.
2. Usually more common in winter season because it is season of fattening.

Aetiology & Pathogenesis

Feeding on finely ground feed without pelleting or with less than 10% roughage for long period causes ruminal parakeratosis.

Pathogenesis

1. Ration with increased amounts of finely ground feed and decreased roughage leads to changes in the rumen as:
   - **a.** Lowering of ruminal pH → acidosis of rumen.
   - **b.** Alteration of the proportion of volatile fatty acids with increase of the production of butyrate and lactate.
   - **c.** Lactate converted into lactic and may lead to lactic acidosis. While butyrate leads to disturbances in normal ruminal keratinization and causes ruminal parakeratosis.
2. The normal physical abrasive action of roughage on the ruminal papillae plays an important role in prevention of abnormal keratinization.
3. The normal stratified keratinized epithelium of the ruminal mucosa act as protective barrier and play an important role in ruminal transportation and metabolism so abnormal absorptive efficiency and metabolism.

Ruminitis	Bloat or Tympany or Tympanitis	Ruminal Parakeratosis
Clinical Signs		
• Rumenitis of small or moderate extent may produce no sings. • Severe extent in lesions specially when associated with extra ruminal lesions as stomatitis or hepatic abscesses leads to: **1.** Loss of body weight. **2.** Ruminal immobility (stasis). **3.** Morbidity 10%. **4.** Course of the disease is about 10 days. **5.** Mortality of untreated animal up to 20%. **Post-mortem lesions:** • Acute inflammation and chronic ulcers in the anteroventral sac. • Acute or chronic or both lesions may present. **A. Acute from** **1.** Hyperaemia of papillae. **2.** Sloughing of papillae. **B. Chronic from** **1.** Granulation tissue formation and scare formation. **2.** Liver abscesses may be present.	**1.** In frothy bloat the signs are developed rapidly within few hours after eating of bloat producing foods so many cases escape clinical observation. **2.** Bulging of left paralumbar fossa due to increase of the intraruminal pressure. **3.** Discomfort of the affected animal and may kick the abdomen. **4.** Laying down and getting up again. **5.** Within few minutes the condition is exacerbated and breathing become difficult, shallow rapid and oral. **6.** Cyanosis of mucous membrane due to acidosis. **7.** Ruminal movement by auscultation is depressed. **8.** Morbidity 25% and mortality up to 50% of affected animals. **9.** Neglected animal die from acidosis or respiration distress. **10.** Free gas tympany may re-attack. **Post-mortem lesions:** **1.** Over distended abdominal of dead animal. **2.** Distension of the rumen with foam and viscous ingesta. **3.** Thorax and abdomen contain blood but rumen contains ecchymotic heamorrhage due to rupture of some blood vessels due to over pressures. **4.** Cyanosis of cervical part of Esophagus. **5.** Rupture of rumen, diaphragm or abdominal wall may occur.	**Clinical signs and PM:** • Post-mortem lesions include, browning, hardening, thickening and clumping of ruminal papillae. • histopathological, there is accumulation of excessive layers of keratinized squamous epithelial cells on the surface of the ruminal papillae.
Diagnosis		
Difficult to be diagnosed clinically but can be detected by PM lesions.	**1.** History of eating of bloat producing food, clinical signs and PM lesions. **2.** By using of stomach tube to differentiate between free gas tympany and frothy tympany as the pressure is completely relief by stomach tube only in free gas tympany. **3.** History of previous attack with free gas tympany. **4.** By percussion of abdomen - tympanic or drum sound. **5.** By auscultation - ruminal stasis. N.B.: Normal ruminal movements 3-6/2 min. sheep and 2-4/2min. Goat.	**1.** Difficult to be diagnosed clinically by veterinarian. **2.** PM lesions and histopathological change. **3.** Differential diagnosis with hypovitaminosis –A.
Prevention And Treatment		
1. Gradual change of ration of feedlot lambs from roughage to 905 concentrated rations. **2.** The control and lactic acidosis to decrease the amount of irritant lactic acid. **3.** Vaccination against contagious ecthyma. **4.** Oxytetracycline in ration (50mg/head/day; to prevent rumenitis and liver abscess (side effects).	**1.** In case of frothy tympany: Convert the frothy tympany into free gas tympany by using of antifoaming agent as simethicone containing drugs (R/Bloat Remedy, R/Gas Remedy, R/Dimethicone, R/Simethicone and R/Bloatzal). **2.** Also, we can use mineral oil as turpentine oil or vegetable oil as linseed oil and cotton seed oil. **3.** Using of trocar and canula to relief pressure or by surgical interference by rumenotomy. **4.** Evacuation of ruminal contents using purgative as MgSO4 or mineral oil as turpentine oil. **5.** Using of sod. bicarbonate 2.5 to 5% to overcome the acidosis. **6.** Combat the causes of bloat (bloat producing food) and providing of balanced ration (quantity and quality). **7.** **Ruminal tonics** as R/Digestin, R/-Vapcodigest R/Vetadigest, R/Hepavet, R/Rumostomaton and R/Bykahepar. **8.** **Antihistaminic**: as Avil.	**1.** Avoid feeding on complete ration of finely ground food without roughage (At least 10% of the ration). **2.** Monensin gives good result in the prevention of ruminal parakeratosis as it decreases the production of lactate and butyrate while increase the parakeratosis as it decreases the production of lactate and butyrate while increase the production of propionate.

Abomasal and Duodenal Ulcers

Definition

It is focal disruption of the membrane of the abomasum and duodenum, which usually recorded as a complication secondary to acute disease as salmonellosis, blue tongue and lactic acidosis.

Occurrence:

Affect all breeds, sexes and ages specially 3-6 months old age of feedlot lambs especially during late summer and fall season.

Aetiology & Pathogenesis

- Causes of primary ulcers are unknown but we can consider them as a result of hypersecretion acting on area of mucous of with lowered resistance.
- The factors that may contributed are:
  1. Haemorrhage in lamina propria.
  2. Penetration of nematode larvae.
  3. Some stress factors as sudden change from roughage to concentrates.
  4. Inadequate covering mucus.

Pathogenesis

1. The etiological agents that mentioned before leading to devitalization of the gastric mucosa and the gastric juice, affect on it leading to gradual digestion of the devitalized epithelial cells.
2. Just, the covering epithelium is destroyed, there is digestion to the deepest layer (mesodermal cells).
3. In advanced cases the ulcer may reach the lamina propria and may penetrate the gastric wall or duodenal wall causing fatal peritonitis.
4. If the erosion includes large blood vessel it leads to fatal heamorrhage.

Lactic Acidosis / Impaction

Definition

Grain Engorgement, Ruminal Acidosis, Acute Indigestion, Founder, And Grain Overload.

It is an acute metabolic disease of sheep and goats characterized clinically by inappetence, depression, lameness and coma.

Characterized biochemically by acidosis, acidaemia and haemoconcentration and caused by sudden engorgement with grains or other easily fermentable substances by the animals not accustomed to such foods with large amount.

Occurrence:

1. In all breeds, sexes and ages.
2. More common in feedlot lambs at 3-9 months old age than those in yard or pasture systems.
3. More common in farm flock than yard or pastured system.
4. More common after storms when the animals have been unable to eat for periods of time followed by feeding in unlimited quantities of high concentrates feeds.

Aetiology & Pathogenesis

1. Lactic acidosis is caused by engorgement of - Barely, Corn, Cane, wheat, Sugar cane.
2. The animals not accustomed to such food in large amounts.
3. Sudden change in diet from 100% roughage to 90% concentrates.
4. Miscalculating of ration constituents specially concentrates.
5. Feeding on large number of concentrates after period of anorexia and thirst or after accidental fats.
6. Adequate distribution of concentrates in the pans so the animal, separates the concentrates from roughage specially in poorly mixed ration.
7. Sudden change in the ration ingredients, to those high grains and low in roughage.

Pathogenesis:

1. Pathogenesis of lactic acidosis is a series of biochemical changes in the rumen and blood.
2. Excessive microbial production and fermentation in the rumen with subsequent production and absorption of lactic acid is responsible for lactic acid syndrome.
3. Normally the lactate is low in blood but it is increased with excessive production from fermentable feeds in the rumen.
4. When sheep fed on hay and grass (Cellulose food) the cellulytic microorganisms (Gram negative bacteria) act on such foods. But when there is engorgement with starch – rich foods (corn, wheat, barely), amylolytic gram positive streptococci and lactobacilli rapidly multiply and then replace the gram-negative bacilli and production of lactate.

Excessive production of lactic acid leads to:

1. Rumenitis, ruminal ulcers and parakeratosis.
2. Perforation of ruminal wall leading to peritonitis or liver abscess.
3. Absorbed through ruminal wall to blood leading to systemic acidosis or acidaemia.
4. Secreted in the urine leading to acidified urine.
5. Acidosis leads to histamine release causing laminitis and polioencephalomalacia (nervous signs).
6. Lowing of the ruminal PH leading to:
   a. Death of microflora.
   b. Decrease of the ruminal movement. When PH. Are 5 or 4 leads to complete ruminal stasis.
   c. Prevent production of fatty acids (milk fat) with decrease of the milk production.
7. Increase in the size of rumen leading to
   a. Pushing of the blood to the perishing circulation.
   b. Causing high solubility of CO_2 and acidaemia.
   c. Press on the diaphragm and lungs causing respiratory distress.
8. Death usually due to haemoconcentration, acidaemia and dehydration.

Enteritis & Dehydration

Definition

Inflammation of the intestinal mucosa resulting in malabsorption diarrhoea, dehydration and acid base imbalance.

Aetiology & Pathogenesis

Causes	Age & Predisposing Factors	Symptoms
E – Coli	In newly born lambs. Lack of colostrum. Usually in cold weather.	Acute yellow diarrhoea Toxaemia and death
Clostridium Perfringens type B – (lamb dysentery)	Not more than 10 days of age or up to 2 weeks.	Acute diarrhoea. Toxaemia, fever and death.
Salmonellosis	Young lambs. In adult specially in late pregnancy.	Acute diarrhoea. Septicemia and abortion.
Corona virus	Young lambs. Overcrowded area.	Acute diarrhoea. Fever.
Ostertagia spp	Lambs 10 weeks old. Older ewes.	Diarrhoea, weight loss. Inflammation of abomasum.
Trichostrongylus	4-9 months of age.	Chronic diarrhoea. Anorexia. Dullness.
Fascioliasis	All ages.	Acute or chronic diarrhoea. Icteric mucous membranes and bottle jaw.
Coccidiosis	After weaning or beginning of fattening. Bad hygiene or overcrowding.	Haemorrhagic diarrhoea. Dysentery. Death within 2-3 days. Fever – anorexia.
Cryptosporidiosis	7-10 days –lack of colostrum.	

Abomasal and Duodenal Ulcers	Lactic Acidosis / Impaction		Enteritis & Dehydration
Clinical Signs			
1. With ulcers that not accompanied with heamorrhage, the animal show anorexia and signs of pain as kicking **2.** With ulcer that associated by heamorrhage, the animals show anemia and weakness. **3.** Perforating ulcer leads to peritonitis with fever and abdominal distension. **PM lesions** **1.** Ulcer on gastric or duodenal mucosa up to 2.0 mm in diameter. **2.** Perforated abomasal or duodenal wall in case of perforated ulcer. **3.** Peritonitis and internal heamorrhage may be present.	**1.** Diseased animal separated from the flock. **2.** Depression, dullness, and weakness with incoordination in movement. **3.** Inappetence and may anorexia. **4.** Body temperature normal or may slightly elevated. **5.** Accelerated pulse and respiration due to systemic acidosis. **6.** Ruminal stasis with skin dehydration. **7.** Lameness and the animal reclined then coma and death (the animal present in lateral recumbency). **8.** Sunken eyes with lowering of the head due to dehydration. **9.** By Palpation - doughy material in the left flank region. **10.** Abdominal distension specially in left sub lumber fossa. **11.** By Auscultation complete ruminal stasis with some gurgling sound. **12.** In some advanced state diarrhoea may occur (good prognosis). **13.** In late stage, there are paddling, convulsions and death. **14.** Fluid splashing sound may be present by auscultation of the rumen		**1.** Diarrhoea or dysentery as faeces are soft and of foul odour and may be with normal color or dark green or black as a result of bleeding in the intestine or may streaked with blood due to haemorrhage in the lower portions. **2.** Fever if the cause is infectious. **3.** Anorexia, weakness and death may occur. **4.** Dehydration, which manifested in the form of sunken eyes, shrinkage of skin, loss of skin elasticity and increase of the capillary refill time. **5.** Submandibular edema in severe infestation with internal parasites.
Diagnosis			
A. Clinical signs. **B.** PM lesions. **C.** Prevention and treatment have not been developed	**A. History and clinical symptoms:** Palpation, auscultation and skin fold test (to detect degree of dehydration). **B. Laboratory diagnosis: -** **Ruminal juice:** **1.** Acidic 3.8-6 (N = 6.2-7.2). **2.** Disappearance of microflora. **3.** Increase of lactate contents.	**4.** Dark blackish coloured ruminal juice **5.** Gram positive cocci present. **Blood**: **1.** Increased P.C.V. up to 60% (N = 45%) **2.** Low PH. (N = 7-7.2) **3.** High lactate content. **4.** Urine - oliguria -proteinuria - lower pH (5)	**A.** Case history and clinical findings. **B.** Laboratory diagnosis **1.** Blood picture □Anaemia –Haemoconcentration- increase PCV□. **2.** Isolation of the virus or bacteria. **3.** Fecal analysis. **4.** Biochemical analysis for sodium, potassium, chloride, bicarbonate, plasma protein, blood pH … etc.
Prevention And Treatment			
	A. Hygienic treatment: **1.** Supplement the grain rations with large amount of hay. **2.** Withheld water supply to the limited amount. **3.** Nursing of recumbent animals by changing its position and adding good bedding of rice straw. **B. Supportive treatment:** Intravenous injection of physiological saline and dextrose **C. Medicated treatment:** It is planned to **1.** To evacuate rumen by: **2.** Oil purgative □0.25-0.5□ litre of mineral oil or vegetable oil orally□ N.B.: To avoid saline purgative as much as possible. **D. Surgically** by rumenotomy. Stop the harmful action of microorganisms by using antibiotics as oxytetracycline.	**E. Correct pH by:** **1.** Lime stone (50-100gm) then followed by 50gm after 12 hours and every 12 hours. **2.** Magnesium hydroxide or magnesium bicarbonate (50-100gm) then followed by 50gm after 12 hours and every 12 hours. **3.** Sodium bicarbonate orally 50-100gm and I/V as 2% solution (100ml). **4.** Calcium borogluconate 2% solution (100ml). **F. Treatment of dehydration:** **1.** In case of acidaemia, Sod. Bicarbonate solution or lactated **2.** Ringer's solution electrolyte therapy (250ml). **3.** Ringer's solution or isotonic saline 0.9% in case of alkalaemia. **G. Antihistaminic**: As Avil or Allercure or Pirafin amps. **Prevention** The case may be avoided by gradual changing of ration by beginning with 25% grains and 75% roughage. Gradually change this ratio over one month to be 75% grains and 25% roughage. Add grounded lime stone and sodium bicarbonate.	**A. Hygienic treatment.** **1.** Stop feeding for 24 hours then we offer easily digestible palatable food as bran mash. **2.** Thirst may be minimized by allowing animal to drink fresh clean water containing electrolytes. **3.** If toxic material is suspected evacuate the intestine using mild purgative. **4.** If infectious disease is suspected, isolation of animal must be applied. **B. Supportive treatment:** **1.** Isotonic saline solutions 0.9% NaCl. **2.** Dextrose saline solutions. **3.** Lactated ringer's solution. **4.** Sod. Bicarbonate 2% if there are signs of acidaemia **C. Medicated treatment:** According to the etiological factors **Parasitic - Anthelmintics** **1. Nematodes**: Mebendazole, Thiabendazole, Albendazole, Tetramisole and Levamisole **2. Trematodes**-(Fascioliasis): Rafoxanide, and Nitroxinil, and Albendazole. **3. Protozoal** - sulpha drugs and Sulphadimidine, Sulphaquinoxaline and Sulphamethazine and Amprolium compounds. **4. Bacterial** - antibiotics Poorly absorbed - Neomycin Partially absorbed → streptomycin, ampicillin and amoxicillin Kaolin and pectin mixture as intestinal coater and sedative.

5 SMALL RUMINANTS, URINARY DISEASES

Urolithiasis – Calculosis - Urinary Calculi

- It is a metabolic disease of male sheep characterized by the formation of Struvetic concentrations composed of matrix and minerals within the urinary tract that may be followed by urine retention and rupture of bladder or urethra.
- This disease has a high incidence among feedlot lambs (3-6 months) Urinary Diseases.

Economic Importance

Major economic importance for the lamb fattening industry.

Financial waste results from death of affected sheep and condemnation of carcasses at emergency slaughter.

Aetiology and Pathogenesis

This disease results from interaction of dietary, hormonal and environmental factors.

A. Dietary factors

1. Feedlot economics require fattening and marketing lambs in a minimum time, consequently fattening rations contain from 70 to 90% concentrates. The **Grains** especially sorghum or wheat often have **High Phosphorus Contents** and a **Calcium: Phosphorus Ratio Near 1.**
2. This **Dietary Imbalance** creates **High Phosphate & Magnesium level** (in serum and urine), High levels of low-molecular– weight peptides.
3. The increased urea plasma level → **Increase pH of urine** → Providing an environment in which magnesium and phosphate ions are less soluble and ammonium ions are more available.
4. **Mucoproteins, Cells cast or epithelial cells** may contribute to the initiation of Calculus formation by **forming the Nidus of calculus**.
5. The **High** concentration of **Phosphate** anions, **Magnesium** cations and **Peptides** in the urine **with increased pH, → Initiate precipitation** of Ca, Mg and Ammonium phosphates **around the nidus**. **Producing phosphatic** (**Struvitic Calculi**).
6. Another types of calculi may be formed depending upon the dietary components
   a. **Silicate calculi:** may be formed in sheep feed on grass or cereal hays.
   b. **Calcite calculi** ($CaCO_3$) formed in sheep feed on subterranean clover which is high in calcium.
   c. Plants rich in oxalates as Halogeton contribute to Calcite or Wedlite Calculi formation → ($CaC_2O_34H_2O$).

B. Environmental factors

1. Include water deprivation as this increase precipitation of salts.
2. Low atmospheric temperature (autumn and winter) → ↑ the excretion of Peptides and reflex contract the penis and urethra.

C. Hormonal factors:

1. Early **Castration predispose for calculi formation** as maturation of urethra process is dependent upon testosterone, and maturity.
2. The **Urethral process in <u>Immature</u> lambs is small** in calibre and adhered to the wall of prepuce.
3. Early castration removes the effect of testosterone on penis, urethra & urethral process causing these organs to remain hypoplastic during the entire growing & fattening period.
4. In ewe lambs, all calculi easily discharge through the expansive urethra. But in withers, descending calculi 1 to 2 mm diameter may traumatize the delicate tissue and lodge either at the sigmoid flexure or at the urethra process of the hormonally and occlude the urethra gradually accumulating urine following bladder rupture, urine spills into the peritoneal cavity and reabsorption lead to uraemia and finally death.
5. Rupture of urethra discharges urine into subcutaneous tissue of the abdomen and finally through sinuses to the exterior. Urine infiltrated tissue may sough or infected.

Clinical Signs and Post-Mortem Lesions

1. **The signs begin** with frequent, difficult painful urination with straining and rapid side to side movements of tail.
2. In some withers amounts of urine pass and after evaporation, leave deposits of crystals on preputial hairs.
3. **Later**, affected animals **Refuse** food and water(anorexia), **Isolate** from the flock (solitude) and **Kick** at the abdomen (distended).
4. **After Bladder Rupture**, abdomen is distended
5. **After Urethral Rupture**, subcutaneous tissue of abdomen thickens and devitalizes, eventually urine leaks through one or more sinuses of the skin.
6. Among feedlot withers, **Morbidity** reaches 20% while most affect animals die except successfully treated animals.
7. The **Course** of the disease varies from 1-2 weeks.

At necropsy

1. In cases of an animal with **Ruptured Bladder**, the abdominal cavity contains urine. Beginning hydronephrosis may be evident.
2. In cases of an animal with **Ruptured Urethral**, urine infiltrates the abdominal wall (abdomen & S/C are distended).
3. Dissection of bladder and urethra reveals one or more calculi in the urethra at the sigmoid flexure or at the urethral process.
4. Proximal to the occlusion, the urethra is necrotic and haemorrhagic.
5. Additional calculi may be found in the bladder. Uriniferous odor emanate from the tissues.

Diagnosis

1. Case history.
2. Clinical signs and post-mortem lesions.

Prevention

Knowledge of the type and composition of calculi establishing a preventive program.

1. Provide a 2: 1 Calcium to Phosphorus ratio in the total ration. Calcium Chloride should be added to the diet to adjust calcium to phosphorus ratio.
2. Add common salt (NaCl) to the ration, 1-4% is adequate.
3. Prevention is attributed to an ionic action than diuresis from increased water intake. In addition, presence of additional Chloride ions may lead to the formation of Mg Chloride in the urine which is soluble than Mg phosphate.
4. Provide an ample supply of fresh portable water.
5. The addition of ammonium chloride (2% of the ration) will aid in preventing urolithiasis as it induces acidification of urine.
6. Preventing precipitation of struvetic crystals.

   N.B.: Ammonium chloride is expectorant and can be used to initiate cough.
7. A well-balanced diet that includes adequate amount of vit. A.

Treatment

1. If the calculi present in the urethral process, simply cut of the process with scissors.
2. Calculi that lodge at the sigmoid flexure require exteriorization of the penis at the proximal part of the sigmoid flexure.
3. Acepromazine in early cases allow sufficient relaxation of the urethra for the calculus to passed.
4. Muscle Relaxants.