Diseases of Sheep

a. Respiratory Disease Sheep
   i. Viral Respiratory Diseases (PI-3, RSV, adenovirus, reovirus, enzootic nasal
      tumors, OPP)
   ii. Bacterial Respiratory Diseases (Mannheimia haemolytica biotype A, Pasteurella
      trehalosi biotype T, Mycoplasma ovipneumoniae)
   iii. Chronic Enzootic Pneumonia
   iv. Other Diseases of the Respiratory Tract (Oestrus ovis)

b. Enteric Disease Sheep
   i. Internal Parasitism (Nematodes, Protozoans-Coccidiosis, Cryptosporidiosis)
   ii. Neonatal Diarrhea (Clostridium perfringes type A,B,C,D,E, Escherichia coli K99
      F41, Salmonellosis, Cryptosporidiosis, Rota virus)
   iii. Johne’s disease (Mycobacterium avium subspecies paratuberculosis)

c. Reproductive Disease of Sheep
   i. Epididymitis (Brucella ovis, Actinobacillus seminis, Histophilus somni)
   ii. Campylobacteriosis (Campylobacter fetus var. fetus, Campylobacter jejuni)
   iii. EAE (Chlamydophila abortus previously Chlamydia Psittaci)
   iv. Coxiella burnitii
   v. Listeriosis (Listeria monocytogenes)
   vi. Toxoplasmosis (Toxoplasma gondii)
   vii. Pregnancy Toxemia

d. Other Diseases of Sheep
   i. Scrapie
   ii. Caceous Lymphadentitis: Corynebacterium pseudotuberculosis
   iii. Foot Rot
   iv. Ovine Posthitis (Corynebacterium renale)
   v. Mastitis (Maedi-Visna Virus/Small Ruminant Lentivirus, Corynebacterium
      pseudotuberculosis, Mannheimia hemolytica, Staphylococcus aureus ,
      Arcanobacterium pyogenes)
   vi. Copper toxicosis and other causes of anemia (A. ovis, E. ovis, toxic plants)
   vii. Rumenitis
   viii. Urolithiasis

Parasitic and Enteric Diseases of Sheep

I. Nematodes:
   a. Description: These nematodes infect the abomasum and small intestine. Refer to
      parasitology texts for specifics about the life cycle. The following are important
      concepts associated with infection. Eggs are shed in the feces and with appropriate
      moisture and temperature hatch and undergo two molts to third stage larvae. This is
      the infective form which is ingested by the host. Most undergo some tissue migration
      in the host and molt to the fourth stage larva, return to appropriate area and lumen of the
      gastrointestinal tract and mature to adults. Heavy infestations in an individual can lead
      to signs of anemia, diarrhea, weight loss, submandibular edema, poor performance and
in some cases death. Three important concepts are that of environmental resistance, hypobiosis and anthelmintic resistance. The shed eggs and third stage larvae are resistant to some extremes in the environment as they relate to desiccation and changes in temperature. Fourth stage larvae can arrest development in the mucosa in response to seasonal and environmental conditions until such conditions are favorable and appropriate for continued development. This concept is known as hypobiosis. Finally, anthelmintic resistance is wide spread and appropriate and judicious use is paramount in small ruminants. Discussion of treatment and use will follow in the treatment/control section.

b. Etiology: *Haemonchus contortus, Teladorsagia (Ostertagia) circumcincta, Trichostrongylus spp., Cooperia curticei, Oesophagostomum spp.* *(Other minor nematodes: Nematodirus spp., Trichuris ovis, Bunostomum trigonocephalus, Strongyloides papillosius).* Although each may have a slightly different lifecycle, mature in different areas of the small intestine or abomasum, clinical signs and loss of production and treatment will be similar. It is also important to note that sheep and goats share the same pathogenic nematodes.

c. Diagnosis: Detection of parasitism in individual flocks is based on examination of feces, postmortem examination, packed cell volume and FAMACHA score assessment. Routine fecal examination is commonly employed in small ruminant medicine. One important fact to note is this examination should be quantitative and be reported as eggs per gram of feces. Overt parasitism is often noted with post mortem examinations. Finally, packed cell volume and or FAMACHA scoring system can be a sensitive indicator of anemia causing nematodes especially *Haemonchus contortus*. FAMACHA scoring system compares the FAMACHA card with the inferior palpebral conjunctiva. This is a semi quantitative method of identifying animals showing various stages of anemia. The scoring system is from “1” (very anemic) to “5” (normal).

d. Treatment/Control: treatment of nematode infections should be based on clinical findings in individual animals using FAMACHA scoring and response to the anthelmintic using pre and post treatment quantitative fecal examinations. Treatment of the whole flock on a timed schedule has led to marked anthelmintic resistance. Treatment of only clinically affected animals using FAMACHA score or quantitative fecal examination helps to maintain parasites within the herd that are susceptible to available treatments. This approach is known as Targeted Selected Treatment (TST). Resistance as it relates to an individual host is also an important concept. Genetic selection of animals that do not require repeated anthelmintic treatments can also be a highly effective tool. An additional management approach is to institute rotational grazing with other species such as cattle or horses, all of which have parasites that do not infect the other species. Finally, pasture rotation can augment the control of parasite load. Unfortunately this method is not useful in many parts of the country.

II. Coccidiosis:

a. Coccidiosis is a parasitic disease causes by *Eimeria* and is a common cause of diarrhea in lambs and kids. Sheep and goats do not share the same infectious species. Lambs and kids less than 6 months of age are most commonly clinically affected. Signs associated with the disease include diarrhea, anemia, weakness, dehydration and anorexia.

b. Etiology: *Eimeria spp.*

c. Diagnosis: Fecal flotation looking for the oocyst in the feces. Post mortem examination and impression smears of the small intestine.
d. Treatment/Prophylaxis: Feeding of coccidiostats during periods of likely infection and shedding which are brought on during periods of stress and overcrowding. Events such as shipping, weaning and parturition are associated with clinical disease and warrant prophylactic administration of medication.

III. Neonatal diarrhea:
a. Neonatal diarrhea can be associated with inappropriate nutrition of the dam or lamb and infectious organisms. Clinical signs include diarrhea, dehydration, depression, anorexia, recumbency and death. Severity of clinical signs, correspond with the infectious organism(s), management of the flock and status of passive transfer.
b. Etiology: The four most common infectious organisms are enterotoxigenic *Escherichia coli* antigens K99 and F41, Rotavirus, *Cryptosporidium* spp. and Salmonellosis.
c. Diagnosis: History, Histopathology and specific immunologic testing and acid-fast staining of fecal smears for Cryptosporidiosis.
d. Treatment: Treatment is largely supportive. Prognosis and response to therapy is dependent on the clinician’s ability to provide fluid support, diagnose and correct hypokalemia, acidosis and possible hypoglycemia. Assessment of failure of passive transfer, acidosis, hypoglycemia and hypokalemia should be considered part of the minimum data base.

IV. Johne’s Disease:
a. Johne’s disease is characterized by chronic weight loss. Chronic diarrhea occurs in approximately 20% of cases. Signs are often exacerbated by stress. Mild anemia and hypoproteinemia are the only clinical pathologic signs. Presentation is similar to that of parasitism and often not easily ruled out due to the frequency of positive fecal findings. Small ruminants rarely are clinically affected before two years of age and most cases become evident between 2 and 7 years of age.
b. Etiology: *Mycobacterium avium* subspecies *paratuberculosis*
c. Diagnosis: Clinical signs (including submandibular edema/bottle jaw), herd history, post mortem examination and fecal culture or fecal PCR.
d. Treatment: There is no treatment for Johne’s disease. Prevention and control is based on sanitation and flock testing. Most tests have suboptimal sensitivity but a positive test is reason for culling an animal. Caution should be noted with serum ELISA as it may cross react with *Corynebacterium pseudotuberculosis*.

Reproductive Diseases of Sheep

I. Epididymitis of the Breeding Ram:
a. Description: Important disease of rams. Almost always located in the TAIL of the epididymis (vs. Granuloma). Two important modes of transmission hematogenous and ascending.
b. Etiology: *Brucella ovis* infection via hematogenous spread. Other bacteria (*Actinobacillus seminis*, *Histophilus somni* and *E. coli*) infection via ascending route.
c. Diagnosis: Physical exam finding via palpation. Frequently unilateral disease. Acute infection noted by soft swelling that is painful on palpation. Chronic infection enlargement is firm and frequently not painful.
d. Treatment/Control: R/O *Brucella ovis* then long acting tetracycline injections for a treatment length of 9-12 days. Cull ram unless very valuable.
II. Campylobacterosis
   a. Description: Clinical signs include late term abortion with little or no signs in affected ewes unless secondary complications associated with metritis. Some ewes may have mild diarrhea. Transmission via ingestion of contaminated feces or aborted fluid, membranes or fetus. C. jejuni zoonotic and can cause severe diarrhea in humans.
   b. Etiology: Campylobacter fetus var. fetus, Campylobacter jejuni.
   c. Diagnosis: Suspect with late term abortion or birth of live but sick lambs that die shortly after birth. Lesions consist of severe bacterial placentitis. One fourth of aborted fetuses have multifocal hepatitis in which the areas of necrosis are 1-2cm.
   d. Treatment/Control: Commercial and Autogenous vaccines. Some advocate adding tetracycline to the feed during lambing or kidding season or administering long acting tetracycline injections.

III. Enzootic Abortion of Ewes (EAE)
   a. Description: One of the most common causes of reproductive loss in the US. Naïve flocks may show abortion of 25% to 60%. Epizootic abortion rates between 1% to 15%. Transmission via oral-nasal contact with vaginal discharge or aborted material. Infection before 6 weeks of gestation causes late term abortion. Infection after 5-6 weeks of gestation generally causes abortion in the subsequent pregnancy.
   b. Etiology: Chlamydophila abortus.
   c. Diagnosis: Gross and microscopic lesions similar to Coxiella burnetii infection and results in intercotyledonary placentitis. Does may show mild and transient pneumonia and hepatitis leading to anorexia and pyrexia. Bloody vaginal discharge may occur 2-3 days before abortion. Demonstration of elementary bodies in cotyledonary trophoblasts and PCR of fresh placenta or fetal stomach content.
   d. Treatment/Control: Vaccination. Some promote feeding tetracycline in the face of an outbreak or administering long acting tetracycline injections.

IV. Q fever (Coxiella burnetti)
   a. Description: Late term abortion in ewes. Transmission via ingestion or inhalation of infected uterine fluids, colostrum, milk, urine, feces and possibly semen of infected ruminants. Cattle, sheep, goats, dogs and birds may serve as carriers. Multiple species of ticks are the primary natural reservoir and most likely responsible for spread to domestic animals. Zoonosis.
   b. Etiology: Coxiella burnetti.
   c. Diagnosis: Similar to EAE, one of the gross findings is intercotyledonary placentitis. Placental trophoblasts are often filled with Coxiella organisms. Detection of the organism by PCR on freshly aborted placenta is confirmatory.
   d. Treatment/Control: No effective treatment in the face of an outbreak. Current vaccines do not prevent abortion but may decrease shedding. Many infected animals carry the organism indefinitely.

V. Toxoplasmosis
   a. Description: Can be an important cause of abortion in ewes and does. Life cycle includes cats shedding sporocysts in the feces which later become infective; and infection often results in pregnant ewes/does when they are moved into barns used by these cats. Late term abortion and congenital infection of newborn lambs/kids also occurs. Potential for zoonosis exists.
   b. Etiology: Toxoplasma gondii.
   c. Diagnosis: In affected ewes, the placental cotyledons are bright red and contain whitish foci of necrosis.
Treatment/Control: Control of cat population and/or access of susceptible animals to areas commonly used by cats as a litter box. Feeding decoquinate or monensin throughout gestation may reduce the disease incidence.

VI. Listeriosis
   a. Description: Listeria abortions occur during the last trimester of pregnancy and may be associated with septicemia and endometritis in the dam. Often associated with feeding spoiled silage contaminated with rodent urine.
   b. Etiology: *Listeria monocytogenes*.
   c. Diagnosis: Signs of late abortion severe necrotizing placentitis as well as multifocal necrotizing hepatitis of the fetus. Lesions on the liver are often pin point and less than 1mm. Culture or PCR of placenta, liver fetal stomach contents.
   d. Treatment/Control: Addition of Chlortetracycline to the feed during an outbreak may limit or stop abortions. Do not feed poor quality silage.

VII. Pregnancy toxemia
   a. Description: Pregnancy toxemia (ketosis, hepatic lipidosis) usually develops late in gestation. Clinical signs occur in the last 2-4 weeks of gestation and are characterized by depression, incoordination, recumbency, circling, star gazing and bruxism.
   b. Etiology: Conditions that increase energy demands or lead to decreased intake predispose ewes to this condition. Pregnancies that are associated with multiple fetuses are a predisposing factor. Four categories of etiology are common. Primary pregnancy toxemia is associated with decreased intake associated with a period of fasting or poor quality feed. Fat ewe pregnancy toxemia is associated with excessive condition early in gestation and subsequent decrease in intake or quality of feed. Grossly under conditioned ewes also are predisposed to toxemia. Finally, secondary pregnancy toxemia is associated with concurrent disease.
   c. Diagnosis: Elevated urine ketone concentrations are a hallmark of an ante mortem exam. Supportive findings during the post mortem exam include lack of evidence of another disease and a swollen pale liver.
   d. Treatment: Correction of energy imbalance with administration of intravenous glucose and provision of highly palatable energy rich foods. Removal of the fetus is imperative via caesarian section or induction of parturition. Prevention is geared toward control of concurrent disease such as parasitism, maintaining proper body condition and monitoring nutrition.

Respiratory Diseases of Sheep

In general, respiratory disease in sheep results from a combination of appropriate risk factors, including management practices as well as disease agents. Clinical signs are not necessarily specific to the cause, and several agents may contribute. Furthermore, recognition of respiratory disease may be difficult in sheep, as they tend to hide illness. Particular attention to behavior, appetite, and potential risk factors are important in evaluating the flock and individual animals alike. Vaccination for ovine respiratory disease is limited; use of the bovine product Nalsagen may help lower incidence of PI3 pneumonias, but is unproven. Use of other bovine products is not advised.

I. Viral Respiratory Diseases
   a. Description: Like viral infections in other production livestock, sheep viral diseases typically don’t act alone, and likely cause clinical disease in concert with other infectious agents. Signs of
viral respiratory disease are typically mild, and include lethargy, anorexia, +/-pyrexia, +/- cough, tachypnea, dyspnea, tachycardia, sneezing, nasal discharge (typically serous).
b. Etiology: Mild or co-contaminant: PI3, adenovirus, RSV, herpesvirus; Enzootic nasal tumors and Ovine pulmonary carcinoma (retroviruses); Ovine progressive pneumonia (lentivirus);
c. Diagnosis: Suspicion with PE, radiographs, U/S, serum titers. Swabs, tracheal wash, thoracocentesis, biopsy or necropsy for virus isolation, PCR, ELISA, AGID depending on the disease in question
d. Treatment: As with most viral diseases, supportive therapy with antibiotic treatment to target secondary bacterial infection is typical. Surgery for nasal tumors is typically not performed. Management practices may include vaccination (limited), genetic selection (OPP), and culling of affected animals. Control of stress and adequate husbandry are essential for prevention.

II. Bacterial Respiratory Diseases
a. Description: signs are similar to viral infection, making it difficult to discern between these etiologies on physical exam. Additionally, a single etiology is rare. Nasal discharge may be thicker and more opaque compared to viral infections. Changes in lung sounds may be more evident in the cranioventral regions.
b. Etiology: Infections in upper and lower airways are commonly associated with opportunistic bacteria, such as Pasteurella multocida. In addition to lower airways infections, Pasteurella has been implicated in nasal infections causing turbinate destruction, as well as septicemia, arthritis, otitis media, and mastitis. Mannheimia haemolytica Type A2 (bovine form is type A1) and Bibersteinia (formerly Pasteurella) trehalosi biotype T are more common pathogen in the lower airways.
c. Diagnosis: along with diagnostics for viral pneumonias, culture and sensitivity before treatment is initiated.
d. Treatment: similar to viral treatment until pathogen can be identified. Antibiotics should be utilized if there is any concern of bacterial infection. Many antibiotics and NSAIDS will be off label in small ruminants-refer to FARAD for appropriate guidelines, and keep records.

III. Chronic Enzootic Pneumonia
a. Description: The mild clinical presentation of the disease lends itself to the chronic duration and damage. Co-infection with Pasteurella or other viral and bacterial pathogens may cause more significant signs, but may also complicate diagnosis. Management and husbandry practices are key in prevention, including good ventilation and minimizing stress.
b. Etiology: Mycoplasma ovipneumoniae, which is present in healthy respiratory tracts, encapsulates and invades the upper airways. Secondary invaders such as Pasteurella and Chlamydophilia may take advantage. The long, chronic progression of the disease allows for decreased detection, and high morbidity but low mortality in affected sheep may induce carriers and makes eradication challenging. Contagious in nature, Mycoplasma is spread through direct or aerosolized contact.
c. Diagnosis: usually based on necropsy findings, although changes in lungs may not be present. Culture can be confirmatory, but must specifically be requested, as initial culture will not yield Mycoplasmas. ELISA is available, but may cross react.
d. Treatment: In all species, Mycoplasma is challenging to treat, and protocols are based on studies in cattle. Florfenicol (Nuflor), tulathromycin (Draxxin), gamithromycin (Zactran) and enrofloxacin (Baytril) are labeled for Mycoplasma bovis infection in cattle. Long-acting oxytetracycline has been shown to have some effectiveness. No label exists for sheep, consult FARAD. Eradication involves an appropriate quarantine and testing strategy, and changes in management practices.
IV. Other respiratory odds and ends
   a. Verminous pneumonia-3 primary small ruminant lungworms- *D. filaria*, *M. capillaris*, *P. rufescens*. If routine deworming is not utilized, these may go higher on the differential list. The Baermann fecal float should be used for diagnosis; parasites are susceptible to most common anthelmintics.
   b. Aspiration pneumonia-Severe inflammatory response of the lower airways and lungs that results from inhalation of feed or liquids. Can be secondary to laryngeal paralysis, regurgitation during surgery, or iatrogenic introduction during fluid or medication administration. Anti-inflammatorises and antibiotics to reduce secondary bacterial infection are warranted, but prognosis is guarded, and death can occur rapidly.
   c. Coccidiodomycosis-Non-contagious fungal infection occurs in southwestern US. No treatment or vaccination available.
   d. Toxins-Perilla mint may cause a form of Atypical interstitial pneumonia; nitrate and hydrogen cyanide accumulating plants have a hypoxic component that will manifest as respiratory signs.

Other Diseases of Sheep

I. Scrapie
   a. Description: neurologic disease affecting the brain in sheep and less frequently in goats. One of the many transmissible spongiform encephalopathies in mammals that is progressive and always fatal.
   b. Etiology: Like other TSEs, neurodegeneration is instigated by prions that disrupt proper protein folding. Horizontal transmission is thought to be the primary route, with younger animals being more susceptible. Incubation and expression of clinical signs vary in onset and duration, with most affected becoming clinical at 2-5 years. In sheep, genetic susceptibility/resistance is well documented, although resistance to the atypical type of scrapie is not conferred.
   c. Diagnosis: Clinical signs of intense pruritus along with wasting and neurological symptoms may direct diagnostic testing. Antemortem collection of lymph tissues (tonsils, laryngeal lymph nodes, third eyelid) are used to detect the PrP protein. Post mortem changes in the grey matter of the brain can be confirmed by histopathology.
   d. Treatment: No treatment is available. Control relies on antemortem testing and culling, appropriate biosecurity, and genetic selection for resistant animals. Scrapie is a reportable disease, and certification of scrapie free herds is available for participating flocks.

II. Caseous Lymphadentis (*Corynebacterium pseudotuberculosis*)
   a. Description: Common infectious and contagious disease that causes abscess formation both internally (sheep) and externally (goats). It affects growth, performance, and longevity of those animals affects. While external abscesses are an annoyance and a viable source of environmental contamination, the internal abscesses are more of a concern for negative health effects. Additionally, treatment and prognosis of the two forms and varies greatly.
   b. Etiology: *Corynebacterium pseudotuberculosis*. Transmission is primarily horizontal, although there have been reports of transplacental infection. Environmental contamination from infected animals spreads to others through skin abrasions. Classically, infection during shearing occurs when and infected sheep is inadvertently cut by a blade, and the contaminated blade cuts a subsequent sheep. *C. pseudotuberculosis* likes moist environments and can remain viable in ideal conditions for some time.
   c. Diagnosis: Highly suspicious with external abscesses located in lymph node locations. Aspiration and culture of these lesions for confirmation. “Thin ewe” syndrome and poor
performance is associated with internal abscesses, and presence in the mediastinum may also elicit pneumonia-like symptoms. Diagnosis in these cases is made at slaughter or necropsy.

d. Treatment: For sheep exhibiting clinical signs and internal abscesses are suspected, treatment is mostly unsuccessful and culling is typical. For goats, sanitary drainage and flushing of the abscess with quarantine may resolve individual lesions, but more clinical animals should be expected. Depending on the goals of the operation, eradication of the disease is most ideal. Testing and culling, is the most rapid method, although testing does have limitations. Appropriate quarantine and biosecurity to prevent entrance is key. Vaccination is not recommended unless the disease is already present; available testing will be positive for vaccinated animals. Vaccine efficacy may depend on the location of vaccine development, and typically only works to reduce disease duration, not infection.

III. Foot Rot

a. Description: A contagious and infectious disease that can have huge economic and welfare impacts in small ruminants. Infections are difficult to control and can cause not only lameness, but decreases in production as well. Lesions occur in the interdigital space and can vary from mild to debilitating infections, with a range of soft tissue and hoof damage. Pastern swelling, if present, is bilateral, unlike claw abscesses, and an active infection can be malodorous.

b. Etiology: The primary bacterium of infectious concern is *Dichelobacter (Bacteroides nodosus. Fusobacterium necrophorum*, the causative agent in cattle foot rot, predisposes invasion by *D. nodosus*. Environmental conditions cause interdigital irritations and abrasions, leading to bacterial infection.

c. Diagnosis: Based on clinical signs. Confirmation can be made with culture, but can be confounded with other environmental contaminants.

d. Treatment: Treatment relies on good hoof care and proper environmental management. Using topical tetracycline powders and/or drying agents (copper or zinc sulfate) may be beneficial. In severe cases, parenteral administration of long-acting oxytetracycline or florfenicol has been shown to be effective. Footbaths can help control larger outbreaks; zinc sulfate dips are suggested, as copper sulfate carries a toxicity risk with ingestion. Vaccination is unavailable in the United States. Genetic resistance may play a future preventative role.

IV. Ovine Ulcerative Posthitis

a. Description: Also termed pizzle rot or sheath rot, it causes lesions on the external genitalia of males, and can leave lesions in females. Scabs and malodorous lesions occur typically on the end of the penis or sheath, and high protein diets are a risk factor.

b. Etiology: *Corynebacterium renale* is the common agent, and when NPN is excreted in the urine, these bacteria convert urea into ammonia at the level of the penis, leading to local necrosis of the tissues.

c. Diagnosis: Clinical signs and history is typically sufficient. Histopath, culture and PCR may be supportive.

d. Treatment: Treatment for mild cases consists of local wound management, anti-inflammatories, and antibiotics sensitive to the bacteria (penicillin). More severe cases and breeding stock may require surgical intervention to prevent scarring. Removal of high protein diet and sanitation are the primary preventative strategies.

V. Mastitis

a. Description: inflammation of the mammary tissues caused by numerous pathogens. Infection may result in temporary inflammation and discharge, up to intensive scarring of the tissues and systemic illness and death. The physical nature of the teats in small ruminants may predispose the udder to physical trauma.
b. Etiology: a vast amount of bacterial pathogens can be responsible for mastitis, but more common causes include Staph and Strep species, with *S. aureus* being the most frequently isolated. Gram negative bacteria tend to be less common. Mycoplasma species are rare in the US, but found throughout the world. OPP/CAE may cause “hard bag,” with reduced production, but normal appearing milk. Tumors and structural abnormalities are lower on the differential list.

c. Diagnosis: Clinical signs and history may narrow down causation, but culture and sensitivity of milk samples leads to a more appropriate treatment and preventative plan. *S. aureus* causes the more obvious heat, pain, and swelling, eventually leading to “blue bag,” or gangrenous mastitis. More mild signs are certainly less specific. Continued monitoring of bulk milk tanks and investigation when somatic cell counts (SCC) are elevated are applicable in a dairy operation. However, in sheep, mastitis may occur on a more sporadic basis, so individual cultures may be more appropriate.

d. Treatment: Supportive therapy in addition to appropriate systemic and local antibiotic treatment. The decision to treat systemically must be evaluated on a case-by-case situation. Anti-inflammatories may be very beneficial, and facilitate frequent stripping. If the condition of the udder does not permit suckling, grafting or bottle feeding becomes important.

VI. Copper Toxicosis and other causes of anemia

a. Description: As previously discussed, anemia due to parasitism is primary. Anemia due to chronic disease may occur, as well as mineral deficiencies. Sheep tend to be more sensitive to Cu toxicity.

b. Etiology: Nutritionally related, a history of inappropriate supplementation, feed mixing mistakes, or consumption of excess Cu from foot baths.

c. Diagnosis: Nutritional history, anemia, icterus, and death are suggestive, particularly if other causes of anemia have been ruled out (i.e. fecal exam to rule out parasites). Liver biopsy and ante-or post mortem histopathology is confirmatory.

d. Treatment: chelating agents administered orally. Supportive care to prevent kidney damage as a result of methemoglobinemia. Removal of inciting source and mass treatment of all exposed.

VII. White muscle disease

a. Description: Nutritional myodegeneration, or white muscle disease in lay terminology, affects young, rapidly growing animals, with symptoms evident in the first few weeks of life. Two forms, which can occur together, affect the cardiac muscle or skeletal muscle. Clinical signs result from a malfunction of these muscles, resulting in a multitude of symptoms. Death is rapid; those that survive have impaired growth and performance.

b. Etiology: A vitamin E and/or selenium deficiency. Typically, ewes have been overwintered on feeds that are deficient, and colostrum stores are insufficient.

c. Diagnosis: Necropsy is usually the first indication, although it may be anticipated based on previous experience. Characteristics white streaks on the myocardium and pale skeletal muscle may be evident grossly. Antemortem diagnostics: whole blood selenium and plasma vitamin E, liver biopsy to assess current stores.

d. Treatment: Prognosis with clinical signs is poor; injections of vitamin E/selenium combinations can be instituted and done prophylactically in neonates. Oral supplementation and/or injections in ewes may be helpful if done in advance of parturition, but ensuring that nutrition is appropriate during the last trimester is crucial to prevention.

VIII. Rumenitis/Acidosis
a. Description: Rumen inflammation and disturbance of the normal flora. Subsequent issues with acidosis include: worsening rumenitis, ulcers, liver abscesses, bacterial emboli of the lungs, endocarditis, peritonitis, laminitis, to name a few.
b. Etiology: Alterations in ruminal flora result in changes in rumen pH. Typically, this is the result of energy dense rations, especially when a ration change is made too rapidly. Progression of the disease whereby the pH drops (acidic) enough that lactic acid producing microbes take over leads to acidosis. Other causes of rumenitis are any source of irritation and inflammation.
c. Diagnosis: Clinical signs are vague, but decreased appetite, alterations in attitude, and decreased rumen function warrant further investigation. Additional history may give clues and lead to evaluation of rumen fluid, although results may be inconclusive (would expect fluid to be acidic, but anorexia causes a more alkaline pH). Response to therapy may give confirmation.
d. Treatment: In acute cases, removal of the inciting cause, and then supportive care of the affected animal(s). GI protectants (mineral oil, karmalax), forage diet, rumen buffers. In very valuable cases, rumenotomy with removal of feedstuffs, and/or tranfaunation. Chronic cases may result in other disease manifestations, and those must be managed accordingly, if possible.

IX. Urolithiasis
a. Description: Common disease presentation in castrated and intact male feedlot lambs and increasingly in pet small ruminants. Related to an improper Ca:P (2:1 is normal). As the name implies, urinary stones block the outflow of urine, either at some point in the urethra or the vermiform appendage.
b. Etiology: Diets high in phosphorus (concentrate diets) results in unbalanced Ca:P, and accumulation of struvite stones in the urinary bladder. Conversely, the increased feeding of legumes to pet males results in elevated Ca, and calcium carbonate stones can result. Other risks include early castration (urethral diameter) and alkaline urine pH.
c. Diagnosis: Emergency cases may be described as “water belly,” where the abdomen and prepuce may become swollen due to edema and/or urethral obstruction. Owners may note that the animal appears constipated, or attempts to urinate are incomplete. Rupture of the bladder results in temporary relief, but a very rapid decline followed by death. Clinical signs are characteristic of the disease, but confirmation with ultrasound can be performed.
d. Treatment: Temporary relief may be achieved by transection of the vermiform appendage, but re-obstruction is not uncommon. Infusion of acidifying solutions into the bladder may help dissolve stones, but surgical procedures are not uncommon. Tube cystostomy is the preferred choice for long term resolution; salvage procedures such as perineal urethrostomy and marsupialization involve less technical skill and maintenance, but long-term complications are more frequent. Prevention with appropriate nutrition and delaying castration of pets is advised.

X. Ovine Progressive Pneumonia
a. Description: As the name implies, a progressive disease of sheep resulting in severe diffuse interstitial pneumonia, reduced growth, mastitis, arthritis, encephalitis, and varying degree of mortality in younger lambs, with mortality at 100% once clinical signs are seen.
b. Etiology: Caused by a lentivirus; transmission is most commonly via colostrum, but horizontal transmission can also occur. The latency of the disease results in clinical signs not becoming apparent for several years. Close relative to the CAE virus in goats.
c. Diagnosis: History and clinical signs are typically vague; necropsy reveals swollen, heavy, wet, grey lungs, with enlarged tracheobronchial and mesenteric lymph nodes. Other findings coincide with other disease processes: arthritis, mastitis, etc. Ante mortem testing consists of ELISA, PCR, and AGID. Genetic susceptibility can be evaluated as well, and management decisions may benefit from this testing.
d. No treatment for the disease is available. Eradication depends on strict testing and culling methods. Removing newborns from dams prior to colostrum ingestion can be implemented to establish a clean flock.

The following resources were utilized in preparation of this document:


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