

Neonatal Care of Small Ruminants

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Neonatal care of small ruminants is an important aspect of production medicine. An estimated 90% of lamb mortality occurs perinatally (Hindson, *Manual*, 65). This can be significantly reduced with supervision. Hindson states that 10-12% of lambs die within the first 72 hours, but under strict supervision this statistic falls to 1-2%. Care must not only include the lamb or kid, but also the prenatal health of the dams. Annually, 4-6% of ewes die with three-quarters of those deaths occurring at or near lambing (Hindson, *Manual*, 65). Management protocols and skilled shepherds play a key role in decreasing mortality in the peripartum period. The following is an overview of prepartum care of ewes and does, care of neonatal small ruminants, and the common congenital and infectious diseases of small ruminants.

Late pregnancy nutrition is an essential part of prepartum care of ewes and does. Approximately 70% of a lamb's birth weight is gained during the last six weeks of gestation (Boden, 33). If the ewe is underfed at this time and is carrying multiple fetuses, pregnancy toxemia may develop. Pregnancy toxemia is a metabolic disorder where there is insufficient intake of energy to meet the increasing demands of pregnancy or lactation (Matthews, 107). Instead of carbohydrates being used to synthesize glucose, lipids must be mobilized to meet energy demands when demands exceed carbohydrate availability. This leads to accumulation of ketone bodies in the blood and may result in signs of hepatic encephalopathy if the liver becomes substantially compromised due to hepatic

lipid accumulation (Martin, 315). Pregnancy toxemia is more likely to occur in does that are obese with multiple fetuses.(Matthews, 107) Clinical signs are similar in sheep and goats and include inappetence, separation from the herd, unwillingness to move, aimless wandering, lethargy, jaw champing, salivation, star-gazing, head pressing, apparent blindness, tremors, recumbency, coma and death in a heavily pregnant animal (Martin, 315; Matthews, 108). Pathologically, severe hepatic lipidosis is the basis of these signs. Additionally, brain lesions have been observed that are similar to those observed in hypoglycemic human patients, therefore it is also suggested that cerebral hypoglycemia may play a role (Martin 316). Diagnosis is strongly supported by clinical signs. A positive ketostik analysis of milk or urine and a low blood glucose concentration (≤ 1.5 mmol/L) are both supportive of the diagnosis in addition to an elevated serum beta-hydroxybutyrate (> 3.0 mmol/L) concentration. Beta-hydroxybutyrate is also found in the aqueous humor and cerebrospinal fluid in similar amounts as the blood, and concentrations in these fluids remain unaltered up to 6 hours after death (Martin, 316). Demonstration of elevated liver enzyme activity may be useful in ewes, but is not usually significant in does (Matthews, 108). Hypocalcemia may also be noted on blood chemistry.

Pregnancy toxemia is difficult to treat and treatment is frequently unrewarding. Rapid recovery may follow removal of the fetus either through induction of parturition or Caesarean section. In ewes induction of parturition with an injection of 16 mg of dexamethasone can be beneficial but will take 36-48 hours (Martin, 317). In does, after day 144 of gestation 20 mg of Dinoprost Tromethamine (Lutalyse®) can be administered

intramuscularly and parturition will occur in 24-48 hours (Matthews, 15). The dam could also be treated solely with the following medical protocol by the shepherd. Administer 50-100 ml calcium, magnesium, phosphorus, dextrose solution subcutaneously and 160 ml electrolyte/dextrose solution orally, followed by 50 ml of propylene glycol orally 4 hours later. If this protocol does not work the veterinarian should treat with 100 ml of 40% dextrose IV plus 50 ml of 20% calcium IV. Martin states that it may take days for the plasma glucose to return to normal so the dextrose injections must be repeated every few hours even if the dam improves and starts eating again (Martin, 317). Treatment can be discontinued once the dam is eating normally.

Again, it is extremely difficult to treat pregnancy toxemia, especially in goats, without removing the fetus from the dam. It is wiser to prevent this disease than to treat it. Dams should be sorted based on body condition score two months prior to lambing and fed accordingly. Also fetal numbers can be obtained by ultrasound and dams can be sorted depending on the number of fetuses found (Martin, 317). Similar preventions can be undertaken for does keeping in mind that obese does have an increased risk of pregnancy toxemia. Does should be watched carefully in the dry period and not allowed to become over-conditioned (Matthews, 107).

Hypocalcemia is reported worldwide in sheep populations. It is uncommon, but outbreaks may result from errors in mixed rations, incorrect mineral supplementation, and stress-related activities (moving, mixing and dog worry). In non-milking sheep the highest demand for calcium occurs 3-4 weeks prior to parturition due to calcification of fetal bones. Ewes with clinical hypocalcemia will become isolated from the flock and

attain sternal recumbency (unable to raise themselves from their knees). They become weak, depressed and unable to stand even when supported within 2-6 hours. Bloat may develop due to rumen stasis and the rectum may be flaccid and may contain pellets of dried feces. Passive reflux of rumen contents may occur as well as stridor. The diagnosis is confirmed by demonstration of a serum calcium concentration <1.4 mmol/L. The rapid response of ewes to treatment with intravenous calcium differentiates hypocalcemia from pregnancy toxemia. Treatment consists of slow intravenous administration of 20-40 ml of 40% calcium borogluconate solution given over 30-60 seconds (Scott 282-283)

Vaginal prolapse during pregnancy is not a rare disease of small ruminants. Predisposing factors include overweight dams, multiple pregnancies, high fiber diets, coughing, straining and genetic predisposition. A red, round ball of tissue of varying size protruding from the vulva confirms the diagnosis. Treatments range from suturing the vulva closed with a horizontal mattress pattern or Buhner suture, or use of a prolapse harness, a bearing retainer or Minchev's technique (Bulgin). A bearing retainer is not commonly used in goats because it often causes a vaginitis, further straining and it is difficult to attach to short-coated dairy breeds. The Buhner suture is used more commonly used in goats, but must be removed before kidding (Matthews, 46). The cause of the prolapse must be addressed. If an overfull rumen is contributing, decreasing the fiber content in the ration can be helpful. Moistening or changing dusty feed and treating for lungworms may reduce coughing. Straining is a major cause of vaginal prolapse and the source usually remains unknown. *Coccidia* have been implicated in some cases and treatment with sulfa drugs may reduce straining in such cases. A lidocaine epidural may

be used to break the straining cycle (2 ml of 2% lidocaine solution). An injection of flunixin meglumine (150-250 mg IM) may also decrease straining. As a last resort a long acting epidural of lidocaine and isopropyl or ethyl alcohol are occasionally used but have detrimental side effects that include permanent paralysis of the tail, incontinence and permanent paralysis of the hind limbs. If the control of straining is not successful, the Minchev technique can be used. This is the same technique used in cattle, where the dorsal vaginal wall is sutured to the outside of the gluteal area. Since this condition usually recurs likely has a genetic basis, affected animals should be culled. Body weight should also be monitored, especially in dams that have not given birth, to avoid excessive condition (Bulgin).

Ewes and does should be vaccinated, dewormed, given a coccidiostat and administered selenium and vitamin E prior to lambing. There are various protocols and the following (Table 1) is just an example of one from Dr. Marie Bulgin at the Caine Veterinary Teaching Center.

Table 1

<p>30 days before lambing (ewe lambs and ewes)</p> <ul style="list-style-type: none">• Anthelmintic• Put out Deccox in salt for pregnant ewes• Booster vaccination for Clostridial diseases, with any of the following products<ul style="list-style-type: none">○ C&D, C,D&T, 7-way, 8-way, Covexin, Cavalry-9
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Many production ranches will shear the perineum of ewes, which is called 'crutching'. This aids shepherds in identifying parturient ewes and is thought to limit contamination during parturition.

When parturition is near the dam will select a birthing site. This is usually away from the rest of the flock. Isolation increases the mother-offspring relationship while minimizing the risk of interference by other pre-parturient ewes. Nowak states that twenty percent of pre-parturient Merino ewes may be attracted to lambs of other ewes. This may include brief inspection, grooming, suckling and even stealing of the newborn. This alien lamb is usually abandoned when the ewe delivers her own offspring (Norwak). The dam may also seek shelter for parturition. However this depends on weather, pre-lamb shearing and breed. Lastly, the exact site of birth is usually determined by the place where the fetal fluids are first expelled (Norwak). First stage labor includes the above birth site seeking behavior. It also includes dilation of the cervix which usually takes 3-6 hours, less in multiparous dams. Pawing at the ground and lying/standing alternately are also signs of first stage labor. These increased periods of activity occur every 15 minutes. There are also abdominal contractions at this time and the allantochorion appears at the vulva. The contractions and straining occur more frequently and this increased activity coincides with a change in fetal position, with the extension of the forelimbs (Scott, 37).

Second stage labor is approximately one hour and is represented by expulsion of the fetus(es). There is rupture of the allantochorion, with a rush of fluid. The amniotic sac appears at the vulva and may rupture at this stage or not until the ewe expels the fetus

and stands up. This is not uncommon in multiple births and may lead to asphyxia. The interval between offspring is 10-60 minutes. If longer than 60 minutes has passed and additional fetuses are suspected, then intervention should be considered (Scott, 37-38)

Third stage labor lasts 2-3 hours and is characterized by the expulsion of fetal membranes (Scott, 38).

The main focus of this paper is neonatal care, therefore the large topic of obstetrics can only be briefly mentioned here. Dystocia in small ruminants is very common and generally simple to correct. As in other species, a physical examination should be performed and the presentation, position and posture of the fetus determined. Good hygiene such as wearing arm-length disposable gloves and washing the vulva with a diluted surgical scrub is recommended. Also caudal anesthesia is essential for corrections and manipulations by a veterinarian (Scott, 40-41).

Once the offspring has been born it should stand and nurse in about 15-60 minutes (Scott, 87). The neonate finds the udder by nuzzling along the ventral side of the mother spending time in the axillary and inguinal areas of the udder until the teat is found (Nowak). The offspring should consume 50 ml/kg of colostrum within the first hour of life and 200 ml/kg during the first 24 hours of life (Scott, 87). This is a sufficient amount to avoid hypothermia in outdoor and indoor offspring and to protect offspring against gastrointestinal infections which cause 'watery mouth' or diarrhea (Melling, 49). If the dam lacks a sufficient amount or quality of colostrum, then spare ewe, goat or cow colostrum can be used. Only colostrum from goats that have tested negative for caprine-arthritis-encephalitis should be used because the virus associated with CAE is spread

readily in milk, and since this disease is similar to maedi-visna in sheep, it may pose a threat to lambs. Cow colostrum can be used but there are two important sequelae to note. First, cow colostrum contains fewer nutrients than ewe colostrum so the volume required should be increased by 20-40%. Secondly, colostrum from cows should only be fed to lambs or kids for the first day of life because prolonged feeding of cow colostrum may cause hemolytic anemia. If using any of the previously listed colostrum sources, if the dam has been vaccinated against clostridial disease these antibodies will transfer to the recipient (Melling, 55).

Hypothermia is a very real threat to newborn kids and lambs. Exposure, mis-mothering and failure to suckle all lead to hypothermia (Matthews, 60). During gestation, the offspring have accumulated brown adipose tissue and carbohydrate deposits in the muscle and liver. These stores will be used to maintain near-normal body temperature (around 39-40 C) for a period of hours after birth. The length of time these stores will last depends on the mothering ability of the dam (licking the lamb dry and finding shelter), the weather conditions and other factors. The offspring needs to consume colostrum before these reserves are exhausted or body temperature will fall. Once body temperature falls 2-3 degrees (to 37 C or below), the suckle reflex is no longer intact and the lamb will die of starvation if there is no intervention (Martin, 59). It is important to identify hypothermic lambs and kids and to treat them appropriately. In determining the treatment plan, the age of the offspring in hours should be estimated (either >5 h or <5 h). The rectal temperature should be noted as well as the level of consciousness. The reason for hypothermia should be assessed. The dam should be

examined for lack of milk or udder disease and the offspring should be towel dried if wet and examined for abnormalities. Neonates with rectal temperatures less than 37 C and under five hours of age will still have metabolizable energy to draw upon. Therefore these offspring should be warmed until their temperature reaches 37 C, then removed from the heat sources and fed. Neonates with rectal temperatures less than 37 C and over five hours of age will be hypoglycemic to some degree. It is important to supply these patients with energy before re-warming to prevent hypoglycemic seizures. If the lamb or kid can hold its head up than giving colostrum/milk by stomach tube is appropriate. If the neonate is semi-conscious or comatose then the swallow reflex will be absent and an intraperitoneal injection of glucose solution is warranted. It is best to inject 10 ml/kg of a 20% solution of glucose at body temperature. To give the injection, hold the neonate between the knees and using a 1 inch, 9 gauge needle give the injection 1 cm to the side and 2 cm below the umbilicus, directing the needle towards the lamb's tail-head (45 degrees). Hypothermia is treatable and treatment is usually very successful (Martin 60-61).

Below is a figure from Manual of Sheep Diseases that depicts the treatment plans for hypothermic lambs. This approach also applies to kids.

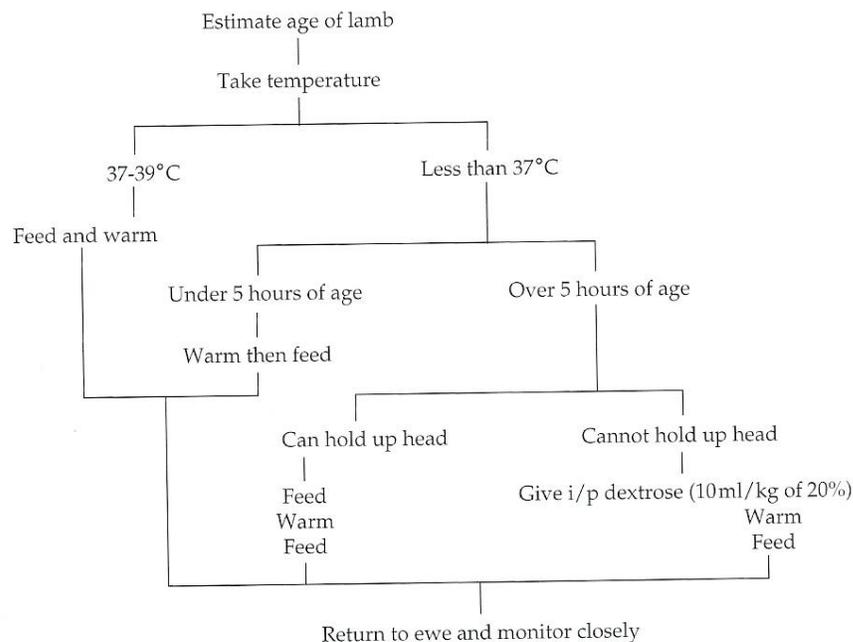


Figure 8.2 Treatment of hypothermia.

There are many instances when lambs and kids will need to be fed via a stomach tube. This is an essential skill that shepherds should master. Feeding lambs by stomach tube decreases the chance of inhalation of milk when weak lambs are bottle-fed and there is decreased risk of the lamb becoming “human oriented” and unwilling to suckle its mother. The tip of the tube is inserted into the rumen where the milk is deposited. Attempts to enter the abomasum are rarely successful and may cause the lamb discomfort. There are rubber and plastic feeding tubes, though the rubber tubes are less likely to cause trauma. The tubes typically come with a funnel-type holder. It is usually easier to use a syringe instead of waiting for fluid to flow through the funnel. Spillage

with the funnel is also common. Below are directions for placement of a stomach tube as contained in Sheep and Goat Practice (Boden, 51-54).

- 1) Sit comfortably with the lamb on your lap.
- 2) Insert a stomach tube (with no syringe attached) via the side of the mouth. All but a few centimeters can be easily introduced into a large lamb.
- 3) Watch the lamb for a few seconds. If intratracheal intubation has been performed the lamb will react extremely violently. Intratracheal intubation is extremely rare in conscious lambs.
- 4) Attach the syringe of feed and empty over about 20 seconds.
- 5) Remove the empty syringe and continue with further syringes until feeding is complete. Feeding is much easier if the whole meal is pre-syringed
- 6) Gently remove the tube with the final syringe still in place.

Stomach tubing very weak or unconscious lambs is contraindicated because protective reflexes which prevent intratracheal intubation are absent (Boden, 55).

The ewe-lamb bond is important in the prevention of hypothermia, exposure and failure of colostrum ingestion. This bond ensures that the dam dries, protects and feeds her offspring. These maternal behaviors are under physiological control. Rising estrogen and genital tract stimulation during birth are the cause of the dam's motivation to lick and to be generally receptive of any offspring. Licking the offspring introduces the dam to the unique odor of their young, dries the young off and stimulates respiration. It also elicits vocal and behavioral responses from the offspring including teat searching. This general receptiveness of the dam usually lasts 3-5 hours after birth. Once this time has past the dam will only be receptive to her own offspring (Martin, 46). In intensive lambing/kidding (indoor) situations it is best to pen the dam and offspring together at the place of parturition for at least one hour to solidify the maternal bond (Norwak).

When the maternal bonds fails to form or breaks down then fostering neonates becomes necessary. There are a variety of techniques for fostering lambs and kids. Boden supports using an Elizabethan collar on the dam. The collar prevents the dam from seeing or smelling the foster neonate. The plastic must be trimmed to the end of the nose of the dam so that she can graze comfortably (Boden 57-58). Other techniques involve dunking the lamb or kid in warm water and then letting the ewe lick it off. Salt can also be used. Using stocks to keep the ewe confined so that the lamb may nurse has also been used. Lastly fastening the skin of the dam's dead lamb over the neonate to be fostered may also be successful (West, 90).

When fostering fails then neonates are raised as orphans. The feeding schedule for these animals can be simplified by age. For the first 12 hours the newborn should be fed colostrum every 2 to 4 hours. After this time for up to 2-3 weeks the lamb or kid should be fed warm milk 3 to 4 times a day. Once they are 3 weeks of age, cold milk can be fed twice daily. Lastly, West notes that pasture and hay should be made available from one week of age to allow proper rumen development (West, 89).

Congenital Diseases of Neonates

Congenital diseases of lambs and kids are not rare and veterinarians should be able to readily diagnose them.

Enzootic ataxia or 'swayback disease' is a syndrome resulting from copper deficiency. Offspring born to copper deficient dams may have one of two forms of the disease. The congenital form is apparent at birth and is usually more severe in nature.

The delayed form, usually not clinically appreciable for several weeks after birth, may be inapparent unless the flock is driven. Affected lambs have a tendency to sway on their feet and are uncoordinated. They may also display muscle tremors and head shaking. The dams may have a band of uncrimped wool of low tensile strength. In colored breeds the band may lack pigment (Martin, 336). The diagnosis is made based on clinical signs, low plasma copper concentrations (>9 $\mu\text{mol/L}$ is normal), low liver copper concentrations (> 40 mg/kg DM is normal), normal cerebrospinal fluid characteristics and histological examination of the central nervous system (Matthews, 55). Treatment is unrewarding and does not reverse the clinical signs. Prevention is more appropriate and can be achieved using several methods. Fertilizers containing copper have been used with some efficacy in Australia. Oral dosing of 1g of copper 4-8 weeks before parturition has been used to prevent swayback. Copper can also be added to feed supplements. Lastly, chelated copper can be injected (SC or IM) but has drawbacks including local tissue inflammation leading to decreased carcass value and acute toxicity (Matthews, 337-338).

‘Border disease’ is a congenital disease of sheep and goats that is caused by a *Pestivirus* (family *Togaviridae*). Affected offspring may be small and weak, while others will show abnormal body conformation, tremor and hairy fleece with abnormal body pigmentation (“hairy shakers”). If normal newborn or adult sheep are infected with the virus the resulting disease is mild or inapparent and the virus is eliminated in approximately 11-14 days. In the pregnant ewe the virus crosses the placenta and infects the fetus within one week. The offspring are either born with abnormalities (as described above), or are aborted or still born. Pre-colostral blood samples will contain detectable

amounts of Border Disease Virus. Histopathology of the CNS shows myelin deficiency and increased hair follicles on histopathology of the skin. Virus isolation can be performed on tissue from the spleen, kidney, brain, lymph nodes and thyroid. Control requires identifying carries and removing them from the flock. In the face of an outbreak the entire lamb/kid crop and the dams suspected of introducing it should be removed from the farm (Boden, 101-110).

Entropion is a common occurrence in neonatal lambs but less so in kids. Usually the lower eyelids roll inwards and the hair rubs on the surface of the cornea resulting in secondary keratoconjunctivitis, epiphora and blepharospasm. Mild cases can be treated with an injection of long lasting procaine penicillin into the lower lid, effectively everting the lid away from the cornea. Michel clips can also be used as well as a vertical mattress sutures. In the most severe cases surgery must be performed to remove a strip of skin from the lower eyelid. Entropion is an inherited condition and rams that produce offspring with this condition should be culled (Martin, 304).

Infectious Diseases of Neonates

Diarrhea in neonatal lambs caused by enterotoxigenic stains of *Escherichia coli* is uncommon but may affect lambs and kids less than four days old. In intensive situations there is rapid spread of disease. The enterotoxigenic strains have fimbriae that assist attachment of the bacterium to the enterocytes, and they produce enterotoxin that interferes with normal water and electrolyte transport mechanisms. This causes a secretory diarrhea and rapid dehydration. Clinical signs include profuse yellow diarrhea,

and weakness, dehydration and death within 24 hours of onset of clinical signs. Diagnosis is made by fecal culture and serotyping for K99 and F41 fimbrial antigens. Treatment begins with isolation of the dam and offspring pair. Oral electrolytes are needed to combat dehydration. Oral antibiotics such as neomycin or spectinomycin are warranted. Systemic antibiotics are not necessary because the bacterium does not invade the gut wall and affected individuals do not develop a bacteremia. On farms that have a history with this disease, vaccination of the dams eight and four weeks prior to parturition will help control *E. coli* enteritis, however such infections rarely become endemic and there may be no infection seen in following years even if no preventive action is taken. The most economical plan may be to vaccinate all newly introduced breeding stock and to carefully monitor the disease situation (Scott, 91-92).

Salmonella infection is only a problem in neonates if there is an obvious source of infection. This problem can be from existing abortions in small ruminants or from cattle. In young kids and lambs the clinical signs appear rapidly and include dysentery, dehydration, septicemia and death. Affected neonates may appear gaunt and have abdominal pain with tenesmus. Fever may be seen initially, followed by subnormal body temperature as the severity of the disease increases. Diagnosis is made by culture or PCR on feces. Antibiotic treatment preferably directed by culture and sensitivity analysis forms the treatment for Salmonellosis. Prevention includes avoiding grazing of sheep and goats with endemic *Salmonella* infected cattle (Scott, 92-93).

‘Watery mouth’, ‘slavers’, and ‘rattle belly’ are all synonyms for an infection with *E. coli* in young lambs. Lambs are affected within 72 hours of birth with this rapidly

fatal disease. Clinical signs include profuse salivation, gut stasis, collapse and death. A swollen, tense abomasum may develop in some lambs as a result of gas formation. This can lead to respiratory distress. This disease is much more common in intensively managed flocks. There is high mortality in affected lambs, despite treatment attempts, thus prevention is the only logical approach. Failure to ingest adequate amounts of colostrum is a major risk factor for this disease. The neutral pH of the abomasum and the reduced mobility of the gut of the newborn lamb also allow the gram-negative bacteria to rapidly multiply and bacteremia results. The endotoxins released by the death of *E. coli* organisms produce the clinical signs seen with watery mouth. Diagnosis is based on the history of the flock, the clinical signs and often non-specific post-mortem lesions. Many illnesses in neonates may present with a similar appearance to watery mouth in their terminal stages. Treatment is disappointing even when cases are detected early. Preventing starvation and correcting dehydration should be the aim of treatment. Electrolyte fluids, fortified with glucose and administered via stomach tube are important. Avoid giving colostrum or milk by stomach tube because the lamb will not be able to digest the food. Antibiotics are often indicated though they should be used in conjunction with a non-steroidal anti-inflammatory to reduce the risk of endotoxic shock. The key to preventing this disease is to ensure adequate colostrum intake as early as possible in the lamb's life. Environmental cleanliness should also be a concern when preventing this disease (Martin, 62-63).

Lamb dysentery is caused by *Clostridium perfringens* type B and outbreaks usually occur at the end of the lambing and kidding season. The disease initially affects

neonates of 1-4 days of age. Older offspring can be affected but the severity of the disease is significantly decreased. In neonates, death usually occurs 2-12 hours after the onset of clinical signs. The first signs are sudden death of the strongest and fastest growing lambs. The lambs and kids may cease to suckle, vocalize continuously and have a tucked-up appearance with obvious abdominal pain. Prostration and death with or without central nervous signs can occur within hours. Dysentery may or may not be present. The causative bacteria are ingested from the soil and the udder contaminated with feces. The bacteria then proliferate and produce large quantities of beta and epsilon toxins. The beta toxin is inactivated by trypsin and colostrum is a potent trypsin inhibitor hence why the very young, well fed offspring likely succumb to this disease. Diagnosis is made on history, clinical signs and necropsy findings of hemorrhagic enteritis with ulceration of the intestinal mucosa. Lamb dysentery occurs in the UK, Europe and South Africa, but is rare in North America. Treatment is not warranted due to the rapid progression of the disease (Martin, 133-134) If caught early enough, administration of clostridial antitoxins may be beneficial.(Matthews, 209) Prevention is ideally achieved via vaccination of the dam. Limiting overcrowding and decreasing contamination of lambing yards is also helpful (Martin, 134).

Hemorrhagic enterotoxemia (or necrotic enteritis) caused by *Clostridium perfringens* type C is characterized by sudden deaths and hemorrhagic enterocolitis in lambs and kids. This is not a common problem among producers though some ranches may suffer substantial losses due to this disease. Neonates 12-72 hours of age are most susceptible and morbidity diminishes after three days of age. Hemorrhagic

enterotoxemia outbreaks are seen in late winter and early spring during cold, wet and windy periods. Lambs and kids will be anorexic, depressed, and develop tremors and abdominal pain. Some will have diarrhea that is dark in color due to slight staining of blood. The course of the disease is 6-12 hours ending in death for the majority of animals with clinical signs. Diagnosis is made by detecting the beta toxin in intestinal contents. Prevention is by vaccination of the pregnant ewes and does (Kimberling, 73-75).

Rotavirus is another cause of profuse diarrhea, weakness and dehydration in lambs and kids. Animals can become infected from birth to four weeks of age. The nature of the feces and the severity of depression are not helpful in diagnosing Rotavirus. Diagnosis is made by electron microscopy, ELISA, or immunofluorescence testing of a fecal sample (Hindson, Outline, 90).

'Floppy kid syndrome' or metabolic acidosis without dehydration occurs in kids and its cause is unknown. It is common in the United States and affected kids have a profound metabolic acidosis with a pH as low as 7.0 (normal 7.4 to 7.44), low serum bicarbonate and a base deficit of 20 mmol or more. Serum sodium and chloride concentrations are normal but serum potassium is increased due to the acidosis. Kids will be normal at birth but develop signs of sudden and profound muscular weakness or ataxia at 3-10 days of age. There is no diarrhea, respiratory disease or other signs specific for an organ system. The kids can swallow, but cannot use their tongues. Mortality can reach 30-50% in untreated cases, though spontaneous recovery may occur. Treatment consists of correcting the acidosis by administering sodium bicarbonate or liquid antacid such as Peptobismol or Gaviscon. Multiple treatments may be necessary to achieve response.

Supportive care including milk fed via stomach tube is also helpful. More severe cases require intravenous isotonic sodium bicarbonate to correct the electrolyte imbalance (Matthews, 59-60).

Cryptosporidium parvum is a protozoan coccidian parasite that primarily infects the small intestine. It is transmitted feco-orally via the sporulated oocyst. This disease affects neonates as young as 4 days old, but is more typically seen in 1-4 week old animals. Kids and lambs will have diarrhea which leads to dehydration, anorexia, abdominal tension and lethargy. Diagnosis is usually by microscopic examination of stained fecal smears. The stain is typically Ziehl-Neelsen which the oocysts stain rose pink to red against a blue-green background (Martin, 153-154). ELISA and IFA based detection methods also exist. There is no specific treatment and symptomatic care including correction of dehydration is helpful. *Cryptosporidium* is potentially zoonotic. It causes only mild or subclinical disease in adults, but can cause severe disease in children and the immunosuppressed (Matthews, 211).

The neonatal period is a crucial time during sheep and goat production. There are many obstacles and again, good shepherding can lead to lower morbidity and mortality. Understanding the concepts and diseases mentioned above can improve shepherding skills and profitability for the industry. Being able to relay this information to our small production clients is essential.