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Common Nutritional and Metabolic Diseases of Goats

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Introduction

Goats, like any other living animal, must consume feed containing essential nutrients to support body functions (maintenance and activity) as well as various productive functions (growth, pregnancy, lactation). Essential nutrients include a wide array of chemical elements and molecules, which can not be sufficiently synthesized by the body to support daily functions. Essential nutrients include water, energy, amino acids (protein), minerals and vitamins. Minerals include macrominerals (calcium, phosphorus, magnesium, potassium, sodium, chloride, sulfur) and micro- (trace) minerals (cobalt, copper, iodine, iron, manganese, selenium, zinc). Similarly, vitamins include the fat-soluble (A, D, E, K) and water-soluble (B-vitamin complex, C) groupings. Most mammals, including goats, can synthesize their own vitamin C and thereby do not require additional dietary supplementation. Also ruminant animals can obtain needed K and B-complex vitamins from bacterial synthesis in the rumen or intestinal tract. The science of nutrition studies essential nutrient requirements for various animal species, including humans, to maintain proper health, support productive activities and prevent disease.

All essential nutrients are required within some optimum amount on a daily or semi-regular basis to support body functions. If intake of any given nutrient is significantly below daily needs for a sufficient period of time then nutritional deficiency disease can occur. Toxicity disease results when a nutrient is in excess of requirements. Clinical signs resulting from a nutritional deficiency or toxicity disease will depend upon the nutrient in question. Many nutrients are associated with specific disease processes, but there is much overlap across nutrients. The bottom line is all essential nutrients can cause disease associated with either deficient or excessive intake. Essential nutrients vary considerably in their inherent propensity to induce a disease state. Detailed information relative to specific nutrient deficiency or toxicity disease can be found in any basic animal nutrition textbook. This presentation will address specific nutritional diseases commonly encountered in raising and managing goats.

Pregnancy Toxaemia / Ketosis / Fatty Liver Disease

Commonly known as pregnancy disease, pregnancy ketosis or twin lamb/kid disease. Pregnancy toxemia is a metabolic disease of goats and sheep commonly occurring in the last six weeks of gestation in does with multiple fetuses. A similar syndrome occurs in early lactation in heavily lactating does (see next section). Factors important in the development of the disease are: (1) presence of two or more fetuses; (2) undernourishment during late pregnancy when the fetuses have the most rapid growth and (3) addition of stress such as severe weather, sudden changes in feed or other disease or transportation upon the previous factors. The mortality rate is high in affected animals. Most information available comes from studies in sheep. Through recognition of early signs and symptoms and avoidance of predisposing factors, pregnancy toxemia can be reduced to a sporadic condition.

Clinical Signs. Disease course varies but generally develops over three to ten days. A more sudden onset is usually associated with a sudden stress or poor producer observation. Appetite is poor or absent, with decreased consumption of grain observed first. Does separate from the herd, lag behind and become depressed and gaunt. Other signs of predisposing disease may also be present. Producers vigilant when hand feeding does will easily recognize these animals.

Clinical signs are those observed with involvement of the central nervous system. Initially, the animal tends to separate from others. There is mild depression. Evidence of blindness develops, the animal runs into objects, shows little or no reaction when approached, and wanders aimlessly. Dullness and depression become progressively severe. There is reluctance to move. Eventually they go down in sternal or lateral recumbency and show little or no response to their environment. The does become comatose and eventually die. Occasionally, animals may show a short period or intermittent periods of hypersensitivity, due possibly to ketone-induced magnesium deficiency. There may be quivering, twitching of the ears, muzzle or eyelids or spasms of certain muscles. Incoordination may be evident. Recumbent animals may have convulsive paddling movements.

Chewing, teeth grinding or vigorous licking movements may be seen. Mild scouring may be present. A snuffling respiration due to excessive nasal secretion may be common. Drooling of saliva is also seen. Temperature and pulse are within normal limits. Respiration is usually normal until the later stages when it may become labored. Ketones may be detected in the urine using diagnostic strips or smelled on the breath (sweet acetone smell). Diseases that may look similar to pregnancy toxemia include polioencephalomalacia, hypocalcaemia, toxic mastitis (if near or after kidding), grain overload, listeriosis and lead poisoning.

Causes. The primary cause of pregnancy toxemia is a lack of glucose as an energy source. This results from either poor nutrition, excessive demand from multiple fetuses or some combination. As pregnancy progresses, an increasing demand is on the available blood glucose supply of the doe due to rapid fetal development. The principal source of energy to the fetus is glucose and utilization by the fetus occurs at the detriment of the mother. Glucose requirements during late pregnancy are increased 70-80% over nonpregnant state since more than 60% of fetal growth occurs during the last 40 days of pregnancy. Blood sugar levels decrease as pregnancy progresses (hypoglycemia) from a normal 35-45 mg per 100 ml blood to 20-25 mg per 100 ml blood in late pregnancy. Pregnancy toxemia may develop when levels decrease to about 18 mg per 100 ml blood. The severity of hypoglycemia will be directly affected by undernourishment of the mother or by increased requirements of the fetus(es).

As the glucose supply diminishes from increasing fetal demands and decreased glucose production due to undernourishment, energy requirements must be derived from alternative sources. The pregnant does is primarily using body fat for an energy source. Fats can not cross the placenta, and thereby do not provide energy for the developing fetus. Amino acids from proteins can be made into glucose and are a primary source when doe nutrition is inadequate. As the doe breaks down more fat as an energy source, the resultant fatty acids can overwhelm the liver's capacity to metabolize them. This results in excessive ketone bodies, acetone, acetoacetate and beta-hydroxybutyrate, and fatty infiltration of the liver (fatty liver disease).

Fatty infiltration of the liver is recognized on post mortem findings of an enlarged pale yellow to orange colored liver.

As ketosis increases, bicarbonate level in the blood decreases and acidosis may result. When the bicarbonate level declines sufficiently, the animal will become comatose. During the later stages of pregnancy toxemia, water consumption decreases, urine output is decreased and kidney function is impaired. The blood sugar level may increase severely (hyperglycemia) during the late stages of the disease as a result of the response of the adrenal glands to stress.

Circumstances causing severe hypoglycemia will usually result in pregnancy toxemia. Under-nourishment of the doe may not meet the demands for glucose production. Level of nutrition should be increasing as pregnancy progresses so that the doe will be able to provide fetal requirements. The doe should be gaining weight during pregnancy. A gradual onset of undernourishment, as would be seen if the feed intake was not increased during pregnancy, may be tolerated by the doe and toxemia may not develop. However, if the animal is starved for several days, pregnancy toxemia may develop readily. Sudden changes in weather, infections or transport may result in periods of inappetence and may trigger pregnancy toxemia.

Treatment. The following are the important areas to address in treating pregnancy toxemia.

Glucose Replacement. Does in the very early stages of pregnancy toxemia may respond to oral administration of propylene glycol (60 ml twice daily) for at least 3 days. In addition to oral glucose precursors, especially for all does beyond the very early stages of the disease, intravenous glucose administration for 1 or more treatments, depending upon response, will be required. Insulin may be used with these treatments for better utilization of glucose (consult your veterinarian). During later stages of the disease, glucose administration may be ineffective or detrimental because the blood glucose levels may be very high.

Address Dehydration, Acid-Base Balance. During the later stages of the disease, acidosis and dehydration may be important factors. Intravenous administration of large volumes of electrolyte solutions with sodium bicarbonate may be important. Corticosteroids may not be effective in the later stages unless given at dosages utilized to combat endotoxic shock.

Reducing Glucose Demand. Developing fetuses are inducing the glucose drain on the doe. A decision will need to be made regarding the relative importance of kid versus doe survivability. Abortion is the preferred method as it is more affordable and less stressful to the doe. If the kids are more than 2 to 3 days premature, they will be unlikely to survive but are already at great risk of death in a severely ill doe. Consult your veterinarian about this procedure. Before a caesarian section is performed, the doe should be stabilized using appropriate fluid therapy.

This is a disease that needs to be prevented rather than treated. It is generally a management disease and should be initially investigated as a herd level problem rather than an individual sick goat. If one doe is clinically ill, many more in the herd are likely at risk.

Prevention. An adequate nutritional level throughout the pregnancy will prevent pregnancy toxemia. Protein and energy levels during the last 30-40 days of pregnancy should meet the doe's maintenance requirements as well as the growth requirements of the fetuses. Dietary protein content for late pregnancy should be between 12 and 15% of dry matter. Dietary energy content can be increased by feeding 1-2 lbs of a cereal grain-based supplement. Management during late pregnancy should be directed at avoiding appetite problems in the animals. Avoid sudden feed changes, diminish stresses of severe weather, delay or avoid transportation and prevent concurrent disease problems.

Doe body condition score entering into late gestation is important. Does that are very thin (< 2.5) have little fat or muscle reserves to draw upon and are then at increased risk despite a good ration. Very fat does (> 4.0) will readily use body fat reserves in late gestation but also experience decreased voluntary intakes, thus predisposing them to formation of ketone bodies that further suppress appetite.

Lactational Ketosis / Fatty Liver Disease

Similar to the disease process described for pregnancy toxemia, dietary glucose deficiency occurring during peak milk production can result in a ketotic state in heavily lactating dairy goats. This is generally not a disease problem in sheep or non-dairy breed goats.

Clinical Signs. Lactating does will initially reduce milk production. Refusal of grain and further reductions in milk production will ensue. Does will rapidly lose body condition during early lactation. Body temperature, pulse rate and respiratory pattern will be within normal limits. Rumen activity may diminish. In severe cases, neurologic signs (nervous ketosis) similar to those described for pregnancy toxemia will be observed.

Causes. Glucose demand to support lactose (milk sugar) production by the mammary gland tremendously increases the does requirement for dietary glucose precursors. Sugars and starches primarily in cereal grains are the predominate sources of glucose precursors. As lactation is initiated, dry matter intake starts at it’s lowest level on the day of kidding and then slowly increases. However, milk production by the mammary gland increases glucose demand more rapidly than accounted for by dietary intake. This results in a period of negative energy balance, resulting in body weight loss to support lactation. If grains are increased in the diet too rapidly, a condition of acidosis might result (see lactic acidosis).

To compensate for the reduced glucose availability, the doe will mobilize body protein and reserve fat to meet increased energy and glucose needs. Excessive body fat mobilization results in large amounts of fatty acids being delivered to the liver for processing. The liver can only metabolize a fraction of the fat delivered and in the face of low blood glucose concentrations, will generate excessive amounts of ketone bodies. Fatty acids not metabolized to ketone bodies will be synthesized back into fat and stored in liver cells. Excessive liver fat storage will result in associated fatty liver disease.

Treatment. Similar to pregnancy toxemia, glucose supplementation in the form of intravenous dosing followed by 3 days of oral propylene glycol is needed. Repeat treatments

may be necessary for full recovery. Corticosteroid therapy is also used to stimulate the doe's ability to generate glucose from amino acids. In refractory cases, insulin therapy in conjunction with glucose infusions may be necessary. Supportive therapy to stimulate intake and dietary modification to increase glucose availability are also warranted.

Prevention. Good supportive care following kidding and appropriate dietary management of the early lactation doe are important. Recently kidded does should be managed carefully to ensure adequate opportunity to eat a well-balanced diet without any obstacles. Recently kidded does may be more timid and reluctant to compete for food. Observe does carefully for any indications of other postparturient disease problems that may negatively impact appetite. Ensure the early lactation diet has higher protein and energy content, but is not excessive in grain. Gradually increase grain over the first 2 weeks of lactation.

Body condition score of the late pregnant doe is again critical. Does should enter lactation with some body reserve, but not excessive. Heavy body condition does will not only have more fat reserves to mobilize and be more susceptible to fatty liver disease, but their intake will be reduced. Excessively thin does will not have the nutrient reserves to support good lactational production.

Periparturient Hypocalcemia (Milk Fever)

Hypocalcemia (low blood calcium concentration) is a disease commonly seen in dairy cattle on or immediately following calving. Sheep can experience hypocalcemia during late pregnancy associated with rapid calcium loss to the developing fetus for bone mineralization. Other species can experience hypocalcemia at or near the time of peak lactation (lactational eclampsia). Based on limited information available regarding goats, it seems dairy breed goats are potentially prone to all three manifestations of hypocalcemia.

Clinical Signs. Initially the doe is ataxic, nervous and hyperactive. The doe is hyperirritable and may show muscle twitching of the lips, eyelids and ears. Trembling or twitching of other muscles of the body may also occur. Convulsions may develop. The doe quickly becomes sternally recumbent and laterally recumbent in the final stages. The head may be turned back to the flank. Less severely affected does (subclinical hypocalcemia) show lethargy, poor appetite and poor milk production.

Affected does stop eating and their ears and skin are cold to the touch. Body temperature will initially be slightly elevated, but will decline to subnormal in a short period of time. Pupils are dilated and respond very slowly or not at all to a flashlight being shone directly at them. Sometimes the hind legs are splayed out behind the doe. The heart is very hard to hear or feel and beats rapidly and weakly. Death follows bloat, regurgitation of rumen contents and aspiration.

The disease course can be as short as a few hours or occur over a couple of days. Occasionally it may occur as "sudden death"; the doe is found dead in the morning. Serum calcium levels are decreased, usually less than 6 mg/dl (normal 8 - 12 mg/dl). To help in diagnosing hypocalcemia in a sudden death case, fluid from eye chambers obtained during a

postmortem examination can be analyzed for calcium concentration up to 48 hours after death. Hypocalcemia may look like other diseases and the doe must be examined by a veterinarian in order to differentiate from polioencephalomalacia, advanced grain overload, toxic mastitis, lead poisoning and listeriosis. Hypocalcemia is often a secondary complicating factor with pregnancy toxemia and ketosis.

Causes. Much research has been done on hypocalcemia in dairy cows and this information is often extrapolated to dairy goats. Dairy cows experience a decline in blood calcium concentration on the day of calving, associated with colostrum production and reduced intake. If the normal homeostatic system is dysfunctional, blood calcium concentration will remain low following calving and result in milk fever syndrome. It is thought that either high calcium intake during pregnancy or high cationic diets (high in potassium, sodium, calcium) suppress the homeostatic system. In contrast, sheep experience hypocalcemia during late pregnancy as a result of insufficient dietary calcium to meet fetal needs. Insufficient calcium intake during late pregnancy or early lactation will require the body to mobilize calcium from bones to meet the need. If the hormonal mechanisms are not properly prepared, mobilization will be delayed and low blood calcium concentration will result. The dairy goat capable of heavy lactation and pregnant with multiple fetuses seems a prime candidate for hypocalcemia at any of the time periods seen with other species.

Treatment. Clinical cases of hypocalcaemia are usually treated with careful intravenous calcium borogluconate solution infusions. This may be followed by subcutaneous injections of calcium solutions as well as oral supplements. Less severely affected does can be treated with just subcutaneous solutions. Response to intravenous treatment should be dramatic. The doe usually starts to shiver and brightens up by the time treatment is finished. If she does not, it may be the diagnosis is incorrect or is complicated by another disease. It is important that intravenous treatment only be given in the face of strong clinical evidence of disease. Calcium can easily cause death if given intravenously to an animal with normal calcium levels. Following intravenous or subcutaneous injections, dietary intake of calcium should be increased with use of alfalfa hay or calcium-based mineral supplements.

Prevention. Without good scientific evidence describing the mechanism responsible for hypocalcemia in dairy goats, it is difficult to define specific feeding recommendations. It would seem prudent to maintain appropriate dietary calcium and phosphorus content in late pregnancy to support fetal bone development, but not to supplement to excess. Dietary potassium should be monitored in an attempt to maintain a level below 2%. Dietary magnesium should also be monitored and maintained according to dietary potassium (see next section on hypomagnesemia). Once into lactation, dietary calcium and phosphorus content should be increased to a level to support milk production capacity. Alfalfa hay can provide this. Cereal crop forages such as wheat or oat hay are very low in calcium (0.15% and 0.24% dry matter (DM) basis respectively) as opposed to alfalfa hay (1.4% DM) and should be avoided unless the ration is balanced with other calcium sources.

Use of anionic salts (minerals high in chloride and sulfur) has been advocated for dairy cattle in preventing milk fever. This requires specialized feed ingredients to be fed and close monitoring as well as controlling dietary potassium content. In using anionic salts, the goal is to induce a state of compensated metabolic acidosis, which stimulates calcium absorption and

mobilization. To achieve the desired effect, one must ensure the animal is appropriately acidified. Urine pH measurements are used for this purpose. In general, anionic salt feeds are not very palatable and can reduce feed intake. This is an undesirable effect. Also these products are only to be fed for a brief period of 10-14 days immediately prior to calving. At present, there seems to be little information to support use of such a control method in goats. In selected situations where hypocalcemia is a serious problem, then with veterinary guidance, this approach may be warranted.

Hypomagnesemia (Grass Tetany, Lactation Tetany, Milk Tetany)

Hypomagnesemia is a common problem in beef cattle on spring pasture, but sporadically seen is dairy cattle and small ruminants. Many clinical syndromes have been identified relative to disease circumstances, but all have hypomagnesemia in common. Lactating does on spring pasture are susceptible (Grass tetany or Lactation tetany) as well as growing kids on milk replacer (Milk tetany).

Clinical Signs. Hypomagnesemia (low blood magnesium concentration) usually occurs in early lactation and results in a life threatening disease process characterized by severe tetanic muscle spasms. Affected animals initially show ataxia, stiffness and hyperexcitability. This rapidly progresses into recumbency and paddling. All muscles are overstimulated resulting in extreme leg stiffness and observed muscle spasms. This is very different from the paralytic muscle weakness of hypocalcemia. Convulsions may be triggered by some stimuli including predator attacks, severe weather changes, transportation and other stressors.

Causes. Magnesium is inefficiently absorbed from the rumen. Dietary levels of potassium and excessive calcium can interfere with magnesium absorption. Potassium is especially of concern relative to magnesium absorption. Magnesium also plays a role in maintenance of blood calcium concentrations and hypomagnesemia can induce hypocalcemia. Besides mineral interactions, differences exist between grasses and legumes as to magnesium content. Grasses contain less magnesium than legumes and when growing rapidly in cooler conditions (lush spring pasture), magnesium availability is greatly reduced. Goats like other ruminants, have little ability to manage blood magnesium concentrations if dietary levels or absorption are depressed. The combination of low intake coupled with greater losses during early lactation result in the clinical syndrome.

Treatment. Like hypocalcemia, hypomagnesemia must be treated as an emergency situation. Intravenous administration of combined magnesium and calcium solutions is necessary. This may be followed by subcutaneous injections of magnesium sulfate solutions as well as oral magnesium supplementation. Response to intravenous therapy is rapid, but may be short-lived. Repeat treatments may be necessary. Subcutaneous and oral supplements are useful in preventing relapses.

Prevention. Appropriate dietary supplementation of magnesium from late pregnancy through early lactation is needed. Dietary magnesium should be increased to account for high dietary potassium, up to a point. Dietary magnesium should not exceed 0.4% of dry matter. A suggested ratio of dietary potassium to magnesium of 4:1 is suggested. Magnesium can be

supplemented in mineral mixes, but it is unpalatable. Mixing 1 part magnesium oxide, 1 part trace mineral salt and 1 part soybean meal or other palatable feed has been shown to be effective in maintaining good magnesium intakes and preventing disease problems.

Lactic Acidosis (Grain Overload)

Clinical Signs. Simple indigestion may be the first indication of a lactic acidosis problem. Any goat eating solid feed containing concentrate is potentially susceptible to ruminal acidosis. In mild to moderate cases the goat will back off feed, especially grain, usually only for one feeding. One may observe a slug feeding behavior where does eat well one day, then back off and repeat the cycle. More chronic acidosis problems will manifest as variable appetite, depressed milk fat and chronic laminitis. Toes grow abnormally fast with "rings". Acute laminitis shows up as painful feet. The quality of the horn is poor and flaky. Goats may be lame and prone to foot abscesses. In some cases, diarrhea will be present, smelling acidic and yellow in color. With more severe acute lactic acidosis, protozoa and bacteria die, rumen becomes static and the goat becomes depressed and dehydrated. The rumen is fluid filled and "sloshy". Acute lactic acidosis may result in death within hours or days. In very severe cases, there is no diarrhea because of total gut stasis. The goat may appear "drunk" and ataxic. Acute acidotic goats will be recumbent and look very similar to milk fever, i.e. cold with dilated pupils. Rumen examination (pH and examination of flora) need to be done to confirm a diagnosis.

Causes. The rumen ecosystem is comprised of more than 200 species of bacteria, protozoa, fungi and viruses. Bacterial species span the range of substrate fermented and end product formation. Lactic acid is one of many potential fermentation end products generated by sugar and starch fermenting bacteria. In normal rumen conditions, production of lactic acid is counterbalanced by its consumption by lactate fermenting bacteria. Lactic acid is a potentially deleterious product in the rumen as it will reduce pH to a point of suppressing bacteria responsible for fiber fermentation. Most bacteria in the rumen are pH sensitive, not being able to survive below pH of 6.0. In situations where excessive sugar or starch is consumed, lactic acid will be overproduced and accumulate, thus inducing a severe decline in rumen pH. As the rumen pH declines, Lactobacillus bacteria will start to proliferate generating more lactic acid. The animal becomes acidotic and loses fluids to the rumen, becoming dehydrated.

The rumen system in best maintained on a consistent dietary regimen. Dramatic changes in dietary forage to concentrate ratio, total amount of concentrate fed and concentrate fermentability will be conducive to lactic acidosis situation. Fiber (e.g. hay or silage) is important in the diet as it stimulates the goat to chew, thus producing alkaline saliva serving to buffer the rumen. Diets with minimal fiber or chopped too finely are more at risk of lactic acidosis.

Treatment. With mild to moderate cases, symptomatic therapy along with a reduction in grain feeding may suffice. In severe acute cases treatment is heroic, intensive supportive care and surgical emptying of the rumen (rumenotomy). Supportive therapy includes intravenous and oral fluids, rumen transfaunation (rumen juice from a healthy animal), alkalinizing solutions for the rumen (only done with caution), antibiotics and nursing care. Even if one recovers an affected goat, secondary problems related to bacterial and fungal infections of the liver and

rumen wall need to be addressed.

Prevention. Inadvertent accessability to grain storage must be prevented. Grain feeding within a diet must be controlled and managed appropriately. Processed grains need to be carefully managed to prevent acidosis as they are more readily available for fermentation in the rumen. Grains should never be fed prior to a forage meal as forage consumption initiates salivary buffering. If grain accessability is separate from forage feeding, grain feedings should be divided into 3 or more separate feedings depending upon the amount needed to support production level. Problems with grain feeding can be minimized by feeding a total mixed ration (TMR). Feed changes all need to be made gradually over several days so the flora have time to adapt.

Low Milk Fat Syndrome

Clinical Signs. Milk composition is an important aspect of dairy goat production as it influences income and quality of product produced. Low milk fat syndrome is a commonly encountered problem in the dairy cattle industry and one frequently observed in dairy goat production. Low milk fat is defined as a milk fat content well below standards for a breed with possible inversions of milk protein and fat content. For breeds where milk fat is typically between 4 and 5%, low milk fat may result in fat percentage at 3% or lower.

Causes. The phenomenon of low milk fat syndrome has be well studied with a number of hypotheses proposed as to the cause. Recent research with dairy cattle has unified two observations as to dietary causes. Diets low in fiber and high in grain can produce milk with low fat content. Similarly, diets with higher polyunsaturated fats can cause the same problem. Currently research suggests an intermediate compound resultant from rumen biohydrogenation of dietary polyunsaturated fatty acids produces a family of compounds identified as conjugated linoleic acids (CLA). Many isomers of CLA have been identified, but one, trans-10, cis-12 CLA has been shown to suppress fat synthesis in the mammary gland. The suppression in milk fat production is dose-dependent to the amount of trans-10 CLA.

Prevention. There is no treatment other than to prevent the formation of trans-10 CLA in the rumen. Low fiber diets with high grain intake seem to initiate the right rumen conditions to generate more trans-10 CLA. Feeding diets higher vegetable fats will also increase trans-10 CLA production. Preventive practices should focus on appropriate fiber to concentrate levels in the diet and minimizing additional vegetable fat supplemention (whole soybeans, whole cottonseed).

Urolithiasis (Urinary Stones)

Also known as urinary calculi, urolithiasis, kidney/bladder stones or waterbelly. Urolithiasis is a metabolic disease of bucks and wethers characterized by formation of concretions within the urinary tract with obstruction to outflow of urine. The male urethra is narrow and long. At the end of the penis is the urethra process (vermiform appendage). Sand may become blocked anywhere but most frequently is at the urethral process, sigmoid flexure (about the level of the testicles) and ischial arch as the urethra travels out of the pelvis.

Clinical Signs. Signs do not develop until there is partial or complete obstruction of the urethra. Uneasiness, frequent attempts to urinate and straining are seen early. Often the presenting complaint is constipation. If observed carefully, the producer may notice frequent dribbling of small amounts of urine, which may be blood tinged. Crystal deposits may collect on the preputial hairs. The animals may refuse food, isolate from the group and kick at the abdomen. If the bladder ruptures, the abdomen may enlarge. If the urethra ruptures, the lower abdominal wall may become thickened from urine infiltration. If the bladder or urethra rupture, the animals may show temporary improvement. However, as time progresses, the animal becomes depressed and death eventually results.

Causes. The disease occurs in animals on a high concentrate diet with a mineral imbalance resulting in excessive phosphorus intake. A high phosphorus level develops in the blood and in the urine. Magnesium and ammonium phosphate precipitate to form a concretion or calculus. The size may vary from sand-like particles to as much as 5-10 mm. The most common type in uroliths are calcium phosphate and struvite (magnesium ammonium phosphate) from high grain diets rich in phosphorus but deficient in calcium. The calculi often have the appearance of sand. Alkaline urine resulting from high potassium intake in forages predisposes to the formation of these crystals in urine. Vitamin A deficiency alters the lining of the urethra predisposing it to cell exfoliation. These cells can act as a nidus to promote crystal formation. Urinary calculi problems are seen most frequently during the winter or periods of very warm weather when water consumption may be reduced.

Treatment. If the blockage is at the urethral process, then it can be snipped off. If urine is voided after this "surgery" then the prognosis, while not good, has some hope. Oral therapy with ammonium chloride or anionic salt products, which dissolves remaining stones is highly recommended. If the blockage is higher, then there are two options, both with major problems. A perineal urethrostomy, in which the penis is exposed and cut in its location below the rectum and the urethra exposed, often results in failure if the blockage is higher. In addition, even if immediately successful, it is a salvage procedure as the hole heals up in a few weeks and the goat re-blocks. Therefore it is not a suitable option for breeding bucks or pets. The second option is very expensive and few practitioners will undertake the surgery. The abdomen is opened and the bladder opened. A catheter is introduced from the bladder into the urethra and the stones flushed down the penis. If successful, all stones are removed and the buck is still capable of breeding. Often the stones are firmly lodged, there is tissue damage from the stones and recovery is very prolonged. Only valuable bucks or valued pets are recommended for this option.

Prevention. As usual, this is a condition better prevented than treated. The diet should have a calcium:phosphorus ratio of 1.5 to 2:1. Salt should be included at 1% of total dry matter intake. Plenty of fresh, palatable water should always be available. Diets high in potassium should be avoided. Vitamin A requirements should be met (good quality green hay and pasture will do this). For herds with previous problems, it is sometimes recommended to include ammonium chloride in the ration at ½ % of dry matter intake. This is particularly true with kids on creep grain. Other diseases such as coccidiosis, pneumonia, etc. which might cause decreased water consumption or increased needs may spark an "outbreak" of urolithiasis so these diseases should be managed as well.

White Muscle Disease (Nutritional Myodegeneration, Stiff Lamb Disease)

Clinical Signs. Selenium deficiency may result in various syndromes including abortion, stillbirth, weak kids, poor growth and muscular lesions. Selenium and vitamin E deficiency can cause acute muscle necrosis known as white muscle disease. Usually young fast growing kids are affected anywhere from birth to full grown. Kids are acutely painful, reluctant to move but may still eat. Sometimes it manifests itself as sudden death as the heart muscle is affected.

Causes. Most soils around the Northeast are very deficient in selenium, resulting in low selenium content of forages. Selenium functions as an antioxidant, generally in concert with another antioxidant, vitamin E. Selenium and vitamin E have been shown to be equally or mutually protective against a wide range of disease processes. Both vitamin E and selenium have been associated with maintaining normal immune function. Degeneration of muscle fibers associated with white muscle disease is believed to be related to a lack of antioxidants to protect cells against prooxidant reactants generated during normal metabolic reactions.

Treatment. In acute cases of white muscle disease, injection of selenium with vitamin E may result in clinical recovery. Multiple injection products are available with varying selenium concentrations. One must be careful in proper dosage as an excessive dosing can result in an immediately fatal reaction.

Prevention. Pregnant does can be injected two to four weeks prior to kidding to protect the newborns if the doe was not supplemented in the diet. Kids should be re-injected at one month of age if no feed supplementation. Appropriate feed supplementation is the preferred method of prevention. Selenium is considered a feed additive and regulated by the Food and Drug Administration. There are specific regulations for dietary selenium supplementation. Free choice trace mineral salts can have a maximum of 90 ppm selenium. At a typical consumption rate, this would provide the maximum of 0.7 mg selenium per day. Selenium premixes can be added to a maximum of 0.3 ppm supplemental selenium in the total diet.

Polioencephalomalacia

Clinical Signs. Polioencephalomalacia (PEM) is a disease of ruminant animals characterized by derangement of the central nervous system due to necrosis of the cerebral cortex of the brain. Young animals on high grain diets are affected more often. Older animals and pastured animals may be occasionally involved.

The onset is often sudden with blindness and disorientation. The head may be elevated. Excitement may be seen but is usually replaced with dullness. The animal may go down on its side with its head thrown back. The legs may be rigidly extended. Convulsions may occur. If untreated, death usually occurs within a few days. Appetite is lost and the animal does not drink. Temperature and respiratory rate are usually normal, but heart rate may be depressed.

Cause. Specific pathogenesis of this disease is not understood. Thiamine is an essential B-vitamin needed for many metabolic functions of the body. In a normal feeding situation,

rumen microbes generate sufficient thiamine to meet animal needs. In PEM, thiaminase, an enzyme that destroys thiamine, is thought to be produced by certain starch fermenting bacteria within the rumen and thiamine deficiency develops. A thiamine - analogue is also produced within the rumen, which may replace thiamine in important metabolic reactions in the brain. Selected necrosis of the brain then occurs, resulting in the clinical signs. Another nutritional situation, excess sulfur intake from either feed, water or both can produce clinical signs identical to thiamine-dependent PEM.

Treatment. Administration of large doses of thiamine intravenously, intramuscularly or both methods early in the disease will usually produce a dramatic improvement within a few hours. In the later stages of the disease, the brain necrosis may be too severe for the animal to recover.

Prevention. Gradual adaptation of animals to initial grain feeding. Until further elucidation of the cause and development of the disease, little can be done to economically prevent the disease. If a case of PEM is diagnosed in a group of animals, it is advisable to inject the remaining animals with thiamine to prevent further cases.

Resources

The first two papers were cited extensively in preparing this material. Other resources offer additional information on goat nutrition.

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