

HERD HEALTH MANAGEMENT PRACTICES FOR GOAT PRODUCTION

Seyedmehdi Mobini, DVM, MS

In any livestock production system, certain diseases and production constraints can be anticipated on the basis of accumulated experiences. Herd Health management and preventive medicine programs are designed to minimize potential adverse effects of these predictable constraints and to protect against unexpected ones. The goal of the program is to improve the goat herd's productivity through general husbandry, nutritional management, parasite control, vaccination, and environmental management. An understanding of some of these management practices and common disease problems is helpful to accomplish these goals.

In general, great emphasis would have to be placed on kid rearing techniques to reduce neonatal mortality and diseases that inhibit the rapid, efficient growth of young kids such as pneumonia, coccidiosis, and gastrointestinal parasites. A major obstacle to newborn kids survival is hypothermia/hypoglycemia and infectious diseases due to delayed or an inadequate intake of a colostrum. Failure of passive transfer of maternal antibodies to newborn kids leads to increased disease incidence and death throughout the postnatal period. The does in late pregnancy should receive their yearly vaccination booster for enterotoxemia and tetanus. The vaccine will both protect the doe and ensure high levels of antibodies in the colostrum, which will subsequently protect the newborn kid. Therefore, kidding should be a well-anticipated event, not an unexpected surprise. Being prepared for routine processing of kids at birth, such as dipping navels, colostrum feeding, and being ready to respond to emergencies will reduce neonatal mortality.

In goats surviving the neonatal period, diseases that inhibit rapid, efficient growth are pneumonia, coccidiosis, and gastrointestinal parasites. In finishing programs in which young goats are pushed on concentrate feeds, conditions such as bloat, urinary calculi, grain overload, and enterotoxemia are likely to be seen.

Conditions that could cause markdown or condemnation at slaughter, such as caseous lymphadenitis leading to lymph node and visceral abscesses, also need to be controlled. Finally, when therapeutic efficacy is not compromised, drugs and vaccines should be given subcutaneously rather than intramuscularly to minimize damage to muscle tissue. Subcutaneous injection is best made into the loose skin of the side of the neck or on the chest wall about 2 inches behind the shoulder. The skin of the injection site must be cleaned with 70% alcohol. A 20-gauge, one-inch needle can be used for an adult goat. Limit the volume of medication to 5 ml per site. If you have to inject a medication intramuscularly, inject it in the neck or thigh muscle. Gluteal muscle should not be used for IM injections in goats. Owners who find it necessary to administer drugs to goats should become familiar with the limitation of the medication. They should read the label and get advice from their veterinarian. Drug withdrawal times vary widely depending on the type used. Do not market or use meat and milk until the end of this withdrawal period. If meat and milk are tested and found to contain violative residues, the producer is identified and is subject to investigations.

Caprine arthritis-encephalitis, caused by a retrovirus, is a relatively new disease of goats first diagnosed in 1974. In spite of this, CAE is now considered as one of the most important disease affecting the goat industry in the United States. All breeds of goats are susceptible to CAE, however, serological surveys indicate that the disease is most common among the dairy goat breed. CAE virus is transmitted naturally in the neonatal period from an infected adult goat to the kid through consumption of colostrum and milk. There is evidence to suggest that CAE can also be transmitted directly from goat to goat possibly through saliva, nasal secretions, urine, or feces.

Under natural conditions, the CAE virus is associated with two disease syndromes. The encephalitis form is most commonly encountered in kids 2 to 4 months of age and is characterized by paralysis that may or may not progress to seizures or death. The arthritic form is most common, and is seen in adult goats 1 to 2 years of age. Affected goats gradually lose weight and develop a poor hair coat and enlarged joints, particularly the carpal, hocks, and stifle. Early in the course of the disease, affected animals may show a progressive and sometimes shifting, leg lameness, however, as the disease progresses, affected goats may walk on their knees and refuse to rise. A presumptive diagnosis can be made based on the history and clinical findings, taking into consideration the age of the animal and disease pattern. Serological tests are available for diagnosis and screening of herds.

There are no known treatments for any of the clinical forms of CAE. Animals with mild cases of the arthritic form can be made more comfortable by providing regular, correct hoof trimming, providing easily accessible feed and water, and by long-term use of oral nonsteroid antiinflammatory drugs to relieve pain (aspirin at the dose of 10 to 20 mg/kg every 8 to 2 hours and phenylbutazone at a dose of 10 mg/kg once a day). Goats with advanced cases of the arthritic form, unable to extend the legs and forced to walk on their flexed knees, should be humanely euthanized.

Before a control program can be instituted, the incidence of infection in the herd should be established using the serological test. If a herd is negative for CAE, it can be kept free of CAE by managing it as a closed herd and only introducing new genetic stock that has been tested free of CAE. Periodic herd test for CAE should be performed to monitor the herd's status. In an infected herd, culling should be considered. Kids should be removed from their dams before they are able to stand and suckle, and should be fed a pasteurized goat colostrum and raised on pasteurized milk or milk replacer. Kids should also be kept separate to avoid contact with

adults.

Caseous lymphadenitis is a bacterial infection of goats, caused by *Corynebacterium pseudo tuberculosis*. It is frequently referred as "abscesses" because swelling, rupture, and drainage of pus from affected lymph nodes is how it is expressed.

Transmission is by goat-to-goat contact through wounds, abraded or even unbroken skin, or by indirect means via brushes, clippers, or contaminated premises. Pus from ruptured abscesses can contaminate the environment, such as feed, fence posts, feeders, or other structures, which can in turn infect other goats.

Clinical signs include enlargement of one or more of the superficial lymph nodes. The most common ones in goats involved are lymph nodes of the parotid followed by prescapular. The abscesses commonly rupture and a thick, green pus is discharged. Internal abscesses, especially in the lungs, may develop and lead to respiratory disease.

Diagnosis of the condition is based on the presence of a firm to slightly fluctuant subcutaneous swelling in the anatomic location of a lymph node. In a herd with a history of caseous lymphadenitis, the clinical findings alone are considered presumptive evidence. A definitive diagnosis can be made by isolation and identification of the organism. Some serological tests have been developed and are used in the diagnosis and screening of goats for the disease.

Treatment with repeated injection of antibiotics does not resolve or eliminate the problem. The infected animal should be separated and isolated. Ripened abscesses can be lanced and flushed with diluted disinfectants. People performing this procedure should wear gloves, because the infection is potentially enzootic. The pus should be collected and destroyed and the goat should be isolated until the lesion is completely healed, typically 20 to 30 days later.

Eradication from a herd is difficult. The owners must be willing to cull animals with multiple abscesses and stop purchasing animals from infected herds. Control can be accomplished in part by removing clinically infected animals from the herd and avoiding contamination of the environment. A vaccine is available and should be considered as a last resort. The vaccine causes severe reaction in infected animals and interferes with serologic testing.

Floppy kid syndrome (metabolic acidosis without dehydration in kids) was first reported in the Spring of 1987. This unique condition was first recognized in herds on the west coast and in Canada. It has more recently been recognized throughout the United States. With the increase in popularity of Boer and other meat goats, there has been an apparent increase in reports of floppy kid syndrome in Texas and other states where meat goat number is increasing.

The affected kid is normal at birth and develops a sudden onset of muscular weakness (flaccid paresis or paralysis) or ataxia at 3 to 10 days of age. Cases tend to occur most commonly late in kidding seasons. Affected kids are depressed, cannot use their tongues to suckle but can swallow and have marked paradoxical metabolic acidosis (anion gap HCO_3 , normal chloride). There are no signs of diarrhea, respiratory disease, or other signs.

The clinical signs of paresis/paralysis/ataxia in 3 to 10-day old kids and supporting blood chemistry value are diagnostic features. The causative agents have not been identified. However, infection and endotoxemia could likely be the cause. Differential diagnoses include white muscle disease, abomasal bloat, colibacillosis, septicemia, or enterotoxemia.

Early detection and correction of a base deficit as well as good supportive care are critical. Since the etiologic agent is not known, no preventive or treatments, aside from correction of electrolyte imbalance and supportive care, is recommended. Less severe cases are most commonly treated by owners with oral bicarbonate or peptobismol at the onset of signs. Kids may need to be fed milk by stomach tube. More severe cases may require blood chemistry and intravenous fluid. A mixture of 2 teaspoons of baking soda, ½ teaspoon salt in a quart of water has been used successfully. Give 4 ounces of this mixture by mouth every 4 hours. Recovery has been seen in 12 hours.

Contagious ecthyma, also known as orf and sore mouth, is a contagious, zoonotic disease of sheep and goat caused by parapox virus. The virus forms scab or pus-like sores typically around the mouth and on the lips of goats. These sores can also appear on face, ears, feet, scrotum, teats, or vulva. The incidence of the disease may be increasing.

Infection spreads among animals by direct or indirect contact. Infected suckling kids contaminate the udder of dams and spread viruses among siblings. There is no specific treatment. One or a few lesions on the lips or nostrils cause little discomfort to the animal, however, lesions over both lower and upper lips cause intense pain, anorexia, and weight loss. Feeding softer concentrate feed may be beneficial to prevent severe weight loss. Diagnosis is usually by herd history and characteristic lesion.

The virus causing this disease is contagious to humans and any person handling goats with sores should wear rubber gloves. Affected goats should be isolated. When buying goats at a sale or direct from the farm, check for the history of a sore mouth. Some states require quarantine of the farm and no animal can be sold until symptoms are gone.

A live vaccine is available. Do not use vaccine in flocks exposed to the disease. The vaccine is recommended only if a sore mouth is a problem in the herd.

Urolithiasis -In goats, clinical obstructive urolithiasis is most frequently seen in young, castrated males fed grain. Urinary calculi can also cause obstructive disease in intact male and may result in their destruction as breeding animals. The tendency for urinary calculi to become lodged in the urethra derives from anatomic factors and castration practices in male ruminants. Increased urine concentration from a decreased water intake or increased water loss are other contributing factors. This condition is a well-recognized, highly prevalent, and costly disease of fattening steers and sheep wethers maintained in feedlot conditions and fed high

a concentrate ration. This could be a potential condition in meat goats raised on a high concentration ration.

Clinical signs include restlessness and anxiety. Tail twitching is an early sign. There may be excessive vocalization and animals will strain frequently and forcefully to urinate. Marked abdominal press may produce some degree of rectal prolapse. Inexperienced owners may assume that the animals are constipated and medicate goats inappropriately rather than seeking veterinary attention. Drops of bloody urine and(or) crystals may be seen attached to preputial hairs. Animals with partial obstruction may be able to void small intermittent streams of urine, but show discomfort.

When the obstruction goes uncorrected, rupture of the bladder or urethra usually results within 24 to 48 hours. Subcutaneous filling of the preputial or perineal region becomes noticeable when the urethra is ruptured. Advanced cases often are presented in a terminal stage and the condition is fatal if left untreated.

When conservative treatment does not alleviate the condition, or if urinary tract rupture has occurred, then some sort of surgical intervention is necessary. Dietary management is the key to control and prevention of obstructive urolithiasis. Providing a continuous supply of clean, fresh water, increasing the concentration of salt in the ration up to 4% to promote water consumption and diuresis are other management factors. Prophylactic uses of urinary acidifiers have also been advocated. Continuous administration of ammonium chloride along with grain at the level of 1 to 2% has been recommended in goats. Concentrations as low as 0.5% have been used successfully to control urolithiasis in Angora goats in Texas.

Polioencephalomalacia (thiamine deficiency) in goats is increasingly recognized under intensive management conditions when goats are fed more concentrated feed to encourage accelerated growth or increased production. The cause of this disease is either a thiamine deficiency or an inhibition of thiamine activity. In goats, the disease typically targets animals that are two months to three years of age. The condition has also been seen in young goats consuming thiamine- deficient milk replacers. Sudden changes in diet, the use of horse feed high in molasses, the feeding of moldy hay, the dietary stress of weaning, deworming with levamisole, and thiabendazole, some species of a fern, and overdosing of amprolium have all been associated with cases of caprine polioencephalomalacia.

Clinical signs may occur acutely or slowly over several days. The initial signs are depression, anorexia, and(or) diarrhea with gradual expression of a neurological dysfunction over a period of one to seven days. Early neurological signs include excitability, elevation of the head while standing, drowsiness, circling, ataxia, muscle tremors, and apparent blindness. As the disease progresses, rigidity, recumbency, nystagmus, and convulsions are observed. If there is no therapeutic intervention, goats will usually die between 24 to 72 hours after an onset of clinical signs. Diagnosis is primarily based on the history and observation of clinical signs under field condition. Other diseases with similar signs such as enterotoxemia and pregnancy toxemia should be ruled out. The critical nature of this disease demands swift intervention by a veterinarian. Goats that are diagnosed during the early stages of polioencephalomalacia respond well to parental administration of thiamine. The vitamin can be given at a dose of 10 to 20 mg/kg intramuscularly or subcutaneously three to four times, for 24 hours. Thiamine hydrochloride is more frequently used. If only multiple B vitamins are available, be sure to dose according to thiamine content. Some cases may require intravenous fluids and tranquilizers.

Common control measures include an increase in roughage feeding with a concomitant decrease in concentrate feeding, avoiding moldy feeds and feeds containing a large amount of molasses such as horse feed. Weaning procedures should be reviewed to ensure that kids are obtaining adequate roughage before weaning. In problem herds, supplementation of the grain ration with thiamine (50 to 60 mg per animal daily) or brewer's yeast may be initiated.

Pinkeye -Infectious keratoconjunctivitis (pinkeye) has been reported in goats as well as sheep and cattle. Mycoplasma and Chlamydia are currently believed to be the most common cause of keratoconjunctivitis in goats in the United States. Moraxella bovis, an important cause of pinkeye in cattle, is not involved in caprine keratoconjunctivitis. Cattle pinkeye vaccine has no place in goat medicine.

Early or mild cases show lacrimation and red and swollen conjunctiva. The cornea may be slightly hazy at the corner or entirely opaque. Some animals develop corneal ulcer, and the ulcer may perforate. The eye is painful and held partially closed. If both eyes are opaque or ulceration occurs, the goat will lose body condition due to inability to eat.

The intensity of treatment varies according to the number of infected goats and concern of the owner. Local treatments with antibiotic ophthalmic ointments or solutions, systemic antibiotics, and subconjunctival injections are the treatment options.

Ringworm-A variety of dermatophytes have been cultured from ringworms in goats. These include Microsporum and Trichophyton species. Lesions in goats consist of alopecia, scaling, erythema, and crust. They typically involve the face, ears, neck or limbs, as well as scrotum. Young animals or those living in a dark, damp, and dirty environment are most at risk for developing ringworm. Management changes may be required to control an outbreak in goats. People handling infected goats should wear gloves to avoid contracting the infection themselves. Lime sulfur (2 to 5%), iodophors as total body sprays, or shampoo daily for five days and then weekly are recommended for treatment. Captain (3%) is effective but not approved for food-producing animals in the United States. Topical iodine ointment and thiabendazole paste can be used on small lesions. All in contact animals should also be treated. Griseofulvin has been used in goats at the dose of 20 mg/kg daily for 1 to 2 weeks orally with good response. Griseofulvin is not approved for use in food animals.

Zinc deficiency in goats can cause weight loss, alopecia, itching, a thick crust on the back of the leg, face, and ears, dandruff, stiff joints, hoof deformities, and small testes that results in reduced libido. Skin lesions caused by zinc deficiency is very similar to ringworm and can only be differentiated by a skin biopsy.

Tetanus (lockjaw) is a well-known clostridial disease of man and animals that produces a characteristic syndrome of muscular rigidity, hyperesthesia, and convulsions. The disease largely arises out of wound becoming contaminated. Goats are susceptible to tetanus and routine vaccination against tetanus is recommended in goats.

Clinical disease has been seen in a one week-old kid within four days of disbudding, and in an adult doe several months after dystocia. Early signs of tetanus include stiffness, "sawhorse" stance, and the ears and tail become stiff. There is reluctance to move, and difficulty opening the mouth. Over time, animals become hyperesthetic and respond dramatically to touch and loud noise by stiffening, collapsing to the ground, followed by seizure. Eventually animals are permanently recumbent, with rigid extension of all limbs and opisthotonus. Affected animals will show convulsion periodically at the slightest disturbance. Once recumbent, death usually occurs within 24 to 36 hours.

In almost all cases, there is a history of recent injury, surgical procedure, dystocia, or vaccination. The disease is almost always diagnosed on the basis of history and symptoms. Treatment and management of the tetanus patients often become very expensive and involved. This includes high doses of antibiotics, high level doses of tetanus antitoxins, wound therapy, fluids, and parental feeding.

The prognosis is always guarded. Tetanus can readily be prevented by a combination of vaccination and good hygiene. When the immune status of young kids is unknown, routine procedures such as disbudding and castration should be accompanied by injection of 150 to 250 units of tetanus antitoxin. When the status of adult is unknown, 500 to 750 units of antitoxin can be administered when treating wounds and dystocias. It is recommended that routine vaccination for tetanus be incorporated into the herd health program for does, kids, and bucks.

Enterotoxemia or overeating disease (OD) is caused by *Clostridium perfringens*, which is found commonly in the environment and intestinal tract. OD is a highly fatal disease mostly affecting young kids. The typical history is a young healthy kid found dead. The affected kid has a history of nursing a heavy milking doe or being on full feed.

The causative organism produces toxins which damage the intestinal tract causing a fatal toxemia. Adult animals can also be affected by OD.

Diagnosis of OD is best made based on a history of the sudden death of a previously healthy animal that is on full feed. Animals have actually been seen to drop to the ground, convulse, and be dead within a matter of minutes. Final diagnosis would include necropsy findings and the identification of the causative bacteria or toxin.

There is usually no opportunity to treat these animals. Specific antitoxin is available and should be given according to label directions. Affected animals should be treated with a high level of penicillin (5 cc/100 lb) and treated with fluids and steroids.

All goat herds need to have a regular vaccination program for OD. This will consist of annual CD/T vaccination given 4 weeks before expected parturition which will protect the kids for 1 to 2 months. Kids are given a series of two vaccinations beginning at 4 weeks of age and repeated 3 to 4 weeks later.

References

American Association of Small Ruminant Practitioners. Avoiding drug residues in goat meat and milk. Ithaca, NY.

Braye, E.T. 1986. Prevention and control of infectious diseases in goats. Proceedings of Meat Goat Production Conference, Tallahassee, FL, pp. 84-88.

Braye, E.T. 1990. Some basic health considerations for goat producers. Proceedings of 1990 Goat Production Symposium, Fort Valley, GA, pp. 74-79.

Bretzlaff, K. 1993. Production medicine and health programs for goats, in Howard JL (ed): Current Veterinary Therapy-Food Animal Practice. Philadelphia, W.B. Saunders Company, pp. 162-167.

Council on Biologics and Therapeutic Agents, 1994. Vaccination guidelines for small ruminants (sheep, goats, llamas, domestic deer, and wapiti). J Am Vet Med Assoc. 205, 11: 1539-1544.

Gaskin, J.M. 1992. Controlling CAE and abscesses in goat herds can be a challenge. United Caprine News, October, pp. 17-20.

Harris, B. 1994. Polioencephalomalacia in goats. Veterinary Technician J. 15, 4: 172-179.

Hungerford, T. 1990. Diseases of Livestock, 9th edition, McGraw-Hill Company, Roseville, Australia.

Knight, A.P., Jokinen, M.P. 1982. Caprine arthritis-encephalitis. Comp. Cont. Ed. Pract. Vet. 4, 6: S263-S269.

Lawrence, L. 1996. Small Ruminant Medicine. Presented at the Ninth Annual Georgia Veterinary Food Animal Conference, Macon GA.

Mobini, S. 1995. Herd Health Management Program for Goats. Veterinary Technician J. 16, 4: 238-241.

- Mobini, S. 1998. Nursing Care of Goats. In Pratt (ed.). Principles and Practices of Veterinary Technology, pp. 549-553. Mosby Publishing Company.
- Mobini, S. 1996. Herd Health Management Practices for Meat Goat Production. Proceeding of the Southeast Regional Meat Goat production Symposium. Florida A&M University, pp. 83-86.
- Olcott, B.M. 1996. Caprine Herd Health. Proceeding of the Southeast Regional Meat Goat Production Symposium. Florida A&M University, pp. 69-79.
- Padmore, C.L. 1990. Infectious Diseases of goats. Proceeding of the International Goat Production Symposium. Tallahassee, FL, pp. 103-105.
- Paschal, J.C., Craddock, B.F., Wayne Hanselka, C. et al. Spanish goat management. Texas Agriculture Extension Service Bulletin B-5021.
- Radostit, O.M., Blood, D.C., and C.C. Gay. 1994. Veterinary medicine - A textbook of the diseases of cattle, sheep, pigs, goats, and horses. Bailliere Tendam Co. London, UK.
- Rowe, J.D., East N.E. 1995. Floppy kid syndrome, wool and wattles. American Association of Small Ruminant practitioners newsletter, Ithaca, NY.
- Scarf, A.D. 1990. Diagnosis, treatment and prevention of neonatal hypothermia/hypoglycemia. Proceeding of the international goat production symposium, Tallahassee, FL, P. 102.
- Shen, D.T., J.R. Gorham, and C.T. Sugihara. 1982. Caseous lymphadenitis (abscesses) of goats and a new diagnostic test. Dairy Goat, J. 60, 11: 841.
- Smith, M.C. 1990. Advances in Sheep and Goat Medicine. Veterinary Clinics of North America, Food Animal Practice, 6: 3. W.B. Saunders Company, Philadelphia.
- Smith, M.C., D.M. Sherman, 1994. Goat Medicine. Lea and Febiger, Philadelphia, Pennsylvania.