**Enterotoxemia is one of the very important diseases and in some areas it is the most prevalent disease of goats. Despite the fact that it is also called ''Overeating Disease'' it is not caused by overeating. Actually, the cause (etiology) of the disease is the toxin (poison) produced by the bacterium Clostridium perfringens type C or type D.  
  
 The bacteria are normally present in the soil and the intestinal tract in relatively small numbers. Under certain conditions the organisms proliferate (reproduce billions of their own kind) in the intestine and produce toxin in lethal quantities. These conditions are those which (1) provide an ideal environment and food for bacterial proliferation and (2) slow down the normal movement of material through the intestinal tract; they are often satisfied by ingesting large amounts of starch when the intestinal tract is not accustomed to it. The disease is often associated with lush fast growing pasture or cereal crops, heavy grain feeding or access to a lot of milk. Illnesses which slow down the intestinal tract, may predispose to the accumulation of dangerous quantities of the toxin.  
  
Most of our knowledge about the disease comes from sheep. There are some important differences in purpose and manner of raising (management systems) that exist between sheep and goats that should be kept in mind when one reads and applies sheep information on goats. First, the disease usually occurs in single (rarely in twins) lambs of a high milk producing ewe; all the milk is consumed by that lamb. In contrast, most goat births are twins, triplets or quadruplets and in the best caprine management systems, the kids are removed from their mother soon after birth.**

**can give .15cc of**

**Thus, the type of birth is no factor in goats. Second, dairy kids are seldom fed large amounts of high energy diets for meat production and therefore do not have the same opportunity (unless accidentally or by mismanagement) of exposure to grain. Finally, goats  are natural browsers (eat from bushes and trees with their head reaching out or up); they do graze but do not consume as much lush feed (usually in a pasture) as rapidly as sheep. Summarizing, the well understood predisposing factors in the sheep disease are not strictly applicable to goats.**

**The predominant predisposing factors in goats have to do with sudden exposure to grain or large increases in quantity of milk consumed without gradually increasing the amount over several days. This leads to indigestion with slowing of the intestinal tract. This probably plays a large role in the disease in goats by allowing more time for toxin to accumulate within the intestinal tract.  
  
The Type D infection is probably far more common than Type C. The latter type produces a toxin called ''Beta Toxin'' which causes intestinal necrosis and severe intestinal hemorrhage. It occurs in adult goats.  
  
Epsilon toxin is produced by the Type D bacteria. It produces vascular damage and increases the permeability (openness) facilitating its own absorption. In the animals that die with neither signs nor  
tissue changes, an extremely large amount of toxin was absorbed very rapidly. When less toxin is produced, the animal lives longer and there is more time for clinical signs and pathological changes to develop.  
  
Signs  
In the Peracute disease course, a baby kid may be found dead with no signs or lesions. It may occur after consuming excess feed or after sudden access to highly palatable feed or after prolonged hunger and a  
normal quantity of feed.   
  
The Acute course of disease lasts 4-26 hours and usually ends in death. Initially the temperature may go to 105F with severe abdominal pain (the kid cries so loudly it is best described as screaming).  
Profuse slimy or water diarrhea will occur. Depression, wobbly gait, recumbancy (lying down on side often with head down) occur early. Convulsions often occur intermittently and may be accompanied by  
continuous or intermittent opisthotonos (head thrown straight over back). The animal may slip into a coma before death or die groaning or even crying. These signs occur in kids but can occur in adult  
milking goats from either Type C or D bacteria.  
  
The Subacute disease is more apt to occur in older kids and adults. They may be ill for several days or weeks and show anorexia (refusal to eat) and intermittent severe diarrhea occasionally with epithelial  
shreds in the feces. They will occasionally eat and with time and appropriate treatment, they will usually recover.  
  
The Chronic form is characterized by intermittent illness lasting several weeks. The goat (usually an adult) will have a dull, stary look, loose feces, an irregular appetite and, if a milker, drop in production.  
  
 Tissue Changes  
Type C is associated with acute hemorrhagic inflammation and necrosis of the mucosa of the omasum and small intestine.**

**Type D causes mild to moderate (occasionally severe) inflammation and even hemorrhage of the small intestinal mucosa. Petechial hemorrhages may be present anywhere in the body but especially on the  
epicardium and endocardium. The pericardial sac may contain slight excess of yellow fluid. Microscopic examination of the brain may reveal degeneration of the vascular endothelium with perivascular and  
intercellular edema with foci of necrosis in several subcortical areas.  
  
Diagnosis  
The diagnosis of ''enterotoxemia'' in goats is probably overdone and is sometimes used to lump any sudden death or acute intestinal disease. The peracute and acute signs are helpful but can also occur with acute  
salmonellosis or intestinal torsion. Individual or first cases of salmonellosis would probably**

**be diagnosed by post mortem bacteriological examination but if a herd problem exists the history, signs and lesions would justify a presumptive diagnosis. Intestinal  torsion is an individual and uncommon event and would rarely be  
diagnosed ante mortem.  
Subacute or chronic cases could resemble coccidiosis, salmonellosis, rumen impaction. Fecal examination, culture and smears would aid in diagnosis of the first two and abdominal palpation, the latter.  
The petechial hemorrhages, especially on the epicardium should make one think of enterotoxemia. However, one should look for at least two other signs which together give good presumptive evidence of  
enterotoxemia; these are, glucosuria and the presence of many short, plump gram positive rods on an intestinal smear.  
  
Ante mortem diagnosis is made early if one can demonstrate a distinct, though transient, improvement of signs after the intravenous injection of 40-100 ml of Type C and D Cl. perfringens antitoxin.  
  
Definitive diagnosis, however, can only be made in the laboratory. Intestinal contents should be preserved by adding 1 ml of chloroform to 10 ml of contents which have been collected in glass within 12 hours  
of death.  
  
Prevention and Treatment  
Vaccination, with Cl perfringens type C and D toxoid by the following schedule along with the good feeding practices of making changes and increases in feed and milk gradually, has provided excellent prevention of the disease. Vaccinate unvaccinated adults twice at 4 to 6 weeks intervals. Vaccinate again during the last month of each pregnancy in order to ''booster'' her immunity and provide colostral antibodies for the immediate protection of the newborn kids. Vaccinate kids at 2-3 weeks of age and 4-6 weeks later.  
  
The older literature suggested that goats produce poor immunity but the results obtained with the alum precipitated vaccine currently in use seem to contradict this idea.**

**Treatment is ineffective against the peracute and acute cases. However, if ante mortem diagnosis is made one should attempt the use of 50 ml of specific hyperimmune serum intravenously every 4 to 8 hours  
in the valuable animal. In the subacute and chronic case, antitoxin along with Tetracycline orally at the rate of 5-10 mg/lb (11 to 22 mg/kg) bodyweight will usually effect a cure.**

**breeder treatment**

**For a baby kid,  treat with 5cc CD antitoxin SQ, 5cc oral penicillin (the long acting) 5cc SQ penicillin, 15cc Pepto Bismol orally. If the kid is in acute pain, you can give .15cc of Banamine and this will also prevent  damage to the kidneys and Liver by toxins. Clostridial bacteria growing in the gut are killed topically by the penicillin, which is the drug of  choice for the species, Pepto is antacid, anti gas, and reduces pain. Usually give 500mg of thiamin orally also. You must treat the kid for  two days, and allow it to nurse if it wants to. On day three, give  only the penicillin, and CD antitoxin, and Penicillin SQ, and Calf Pac  the kid.**

**-----From Coni Ross of CR Ranch.--**

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