



16. Sudden Death

The diseases that progress so rapidly that they kill animals with few signs of illness preceding death are relatively few in number. These diseases are important, however, because in many cases they are either infections or result from a deficiency that may also affect the rest of the herd.

These diseases almost all have the potential to be flock problems. They include:

- Anthrax
- Clostridial diseases
- Lightning
- Grass Staggers

Anthrax

Cause

This is caused by the bacterium *Bacillus anthracis*. These bacteria are able to form spores. (Spores are like bacterial eggs; they are inactive and can survive for many years in the correct environment.)

Anthrax spores survive best deep in alkaline, organic soils. They can be brought to the surface following flooding or disturbance of the soil, for example, trenching. If eaten by an animal the spores reactivate and start to grow. Anthrax bacteria secrete three very potent toxins that can very quickly cause death.

Clinical Signs

The major sign of anthrax is simply death. In some cases there may be evidence of blood draining from the nose, anus or vagina of dead

animals. Carcasses may also appear bloated and appear to decompose quickly.

Diagnosis

Anthrax is a reportable disease in Canada. If you suspect an animal has died of anthrax you must contact your veterinarian or the Canadian Food Inspection Agency (CFIA). They will take a blood sample from the dead animal and send it off to the lab. Results should be available in about 48 hours. In the meantime, you will be asked to protect the carcass from predators, pending the results of the test.

Treatment

Although anthrax is a frightening disease, it does actually respond to most antibiotics. Penicillin and oxytetracycline are both very effective. The difficulty is in identifying sick animals before they die. Animals with anthrax appear very sick and separate themselves from the flock. They will also have a very elevated body temperature.

Prevention

Preventing anthrax requires two strategies:

- 1.** Vaccination. There is a very effective anthrax vaccine available.
- 2.** Preventing environmental contamination. When an animal dies of anthrax the body tissues are riddled with anthrax bacteria. If the carcass remains closed, the tissues will rapidly putrify. During the rotting process, the natural rotting bacteria will destroy the anthrax bacteria.

If, however, the carcass is opened and the anthrax bacteria are exposed to oxygen, they will form spores. The spores will then be washed or dropped onto the soil where they can survive for years.

The goal is to prevent this environmental landmark by ensuring that the carcass is disposed of correctly.

- Ensure that carcass predation does not occur. Cover the carcass with a tarpaulin and spray with formalin.
- CFIA will direct you on how to dispose of the carcass by either deep burial or incineration. Both techniques are very effective and the choice is based on the environmental conditions.

Because anthrax is a reportable disease there is an indemnity paid by the federal government to cover the expenses associated with carcass disposal.

Vaccination

If anthrax is identified on your farm, the CFIA will arrange to have your animals vaccinated.

Vaccination Verses Prophylaxis

The decision to vaccinate your flock against anthrax on a routine basis is complex. The disease is rare but can be devastating. It is best to discuss your farm's particular risk factors with your veterinarian.

Note: Anthrax can affect a wide variety of species. Sheep and goats are not particularly susceptible, while cattle and bison are much more likely to develop the disease. Anthrax can also affect humans; however, the risk is extremely small. Anthrax bacteria from the tissues of a dead animal getting into cuts on your hands constitutes the greatest risk. You can avoid this risk completely if you do not handle suspect anthrax carcasses.

Clostridial Diseases

Enterotoxemia (Pulpy Kidney)

Cause

This is caused by the bacterium *Clostridium perfringens* type D. These are also a spore-forming bacteria widely present in soil and feces. In certain circumstances, such as animals being fed a high grain diet, the environment in the intestines may allow the bacteria to start to grow. The bacteria release toxins which are absorbed into the bloodstream and result in rapid death.

Clinical Signs

Sudden death, typically of very well grown healthy animals on a rich diet (high in concentrates).

Diagnosis

A prompt post-mortem examination of an animal that has died of clostridial disease will reveal that the carcass appears to rot very quickly. The intestines will be reddened and the tissues often appear slightly "cooked." The kidneys will appear very soft and will dissolve if held under running water.

None of these signs is specific as all carcasses will look like this as they rot. The key is that animals that have died of pulpy kidney look like this **immediately**.

The urine of animals that have died of pulpy kidney normally contains glucose; this can be easily tested by the lab.

Treatment

Pulpy kidney occurs so fast that treatment is nearly always impossible. If an animal is identified in the early stages, penicillin should be effective.

Prevention

Vaccination for pulpy kidney is highly effective.

Animals should be vaccinated twice, approximately three weeks apart, and then vaccinated yearly. Ewes and does should be vaccinated in late pregnancy. This will ensure that high levels of antibodies are transferred to the lambs and kids in the colostrum.

The ewe's/doe's antibodies will last approximately two to three months. Lambs/kids should then receive their first dose, followed by the booster three weeks later.

Lambs/kids being fattened on high grain diets are at the most risk of pulpy kidney; this can be a serious disease in a feedlot situation.

Blackleg

Cause

This is caused by the bacterium *Clostridium chauveoi*. Like other clostridia, these bacteria are present in the soil. Bacteria are consumed and can migrate from the intestines to the muscle tissue, where they lie dormant.

It is thought that if the muscle becomes injured or bruised the local environment changes, and the bacteria start to grow. They release toxins into the bloodstream that result in rapid death.

Clinical Signs

Simply sudden death. Sometimes an animal may appear lame shortly before death.

Diagnosis

On post-mortem examination a small, very dark lesion may be found in the affected muscle. Samples sent to the lab can finalize the diagnosis.

Treatment

None.

Control

This disease seems to be much more uncommon in sheep and goats than in cattle.

All 8-way vaccines available contain blackleg. Vaccination should be used as outlined for other clostridial diseases.

Tetanus

Cause

Clostridium tetani bacteria. These bacteria are commonly found in soil and feces where they exist as spores. If spores get into a wound where there is damaged tissue and low levels of oxygen, they hatch and start to grow. Castration and tail docking sites are prime locations for infection.

The bacterium produces a toxin that causes the extensor muscles to constrict and become rigid.

Clinical Signs

Animals may initially appear lame with stiff legs. As the disease progresses, the animals will be unable to stand. They have straight legs and arched backs and their faces may have a leering expression. The disease eventually paralyzes the muscles required for breathing, resulting in death.

Diagnosis

Samples collected at post mortem can be submitted to the lab for a final diagnosis.

Treatment

If the disease is identified early, anti-toxin may be administered. Other at-risk animals in the flock may be given high doses of penicillin before clinical signs develop.

Prevention

There is a highly effective vaccine for tetanus. All sheep and goats should be vaccinated.

Lambs and kids are at especially high risk as the wounds left by tail docking and castration are very prone to tetanus infection. Therefore, it is vital that ewes and does are vaccinated in late pregnancy with a clostridial vaccine that contains tetanus antigen. Also, all lambs/kids should receive sufficient colostrum.

Other Clostridial Diseases

There are a number of other clostridial diseases that occasionally cause clinical disease in unvaccinated flocks. All these diseases kill very quickly, so the most common clinical sign is death.

Lamb Dysentery – *Cl. perfringens* Type B – causes intense abdominal pain and bloody diarrhea in young lambs. Very rare in Canada.

Braxy – *Cl. septicum* – occasionally eating rich frosted forage can result in bacterial overgrowth in the abomasum (true stomach), resulting in bloating and death. Rare.

Struck – *Cl. perfringens* Type C – similar to pulpy kidney but very rare.

Black Disease – *Cl. oedematiens* – similar to blackleg, but the bacterial spores settle in the liver and can be activated following a liver fluke infection. Particularly important in areas where liver flukes are prevalent.

Bacillary Haemoglobinuria – *Cl. haemolyticum* – bacterial spores settle in the liver but when activated produce toxins that destroy red blood cells. This results in red/black urine and death. Occasionally seen in western Canada.

Malignant Edema (Gangrene) – caused by a variety of clostridial organisms. Following a severe injury, tissues may be infected with clostridial organisms, resulting in rapid rotting of the tissues; may be seen following birthing, in bad mastitis, or in head injuries in fighting rams or bucks.

Lightning Strike

Cause

Lightning.

Clinical Signs

None. Animals are found dead after a thunderstorm.

Diagnosis

A full post-mortem undertaken for insurance purposes may find signs consistent with lightning strike.

Prevention

None.

Hypomagnesemia (Grass Staggers)

Cause

The cause is a deficiency of magnesium in the consumed forages. This is typically seen in adult animals turned out to lush green pasture in the spring. The animals are typically low in magnesium after the winter; lush spring grass is low in magnesium but rich in potassium and nitrogen, which makes the situation worse.

Clinical Signs

Animals are typically found dead. If alive, animals are often convulsing.

Diagnosis

Samples can be submitted to the laboratory after a full post-mortem.

Treatment

If an animal is found convulsing, treatment with magnesium solutions administered intravenously is possible. However, finding an animal in this stage of disease is unusual.

Prevention

Ensure animals have access to a mineral supplement containing magnesium.

Other Less Common Causes of Sudden Death

Acute Pneumonia

Occasionally, animals may become infected with a large dose of a virulent respiratory pathogen causing very severe pneumonia that kills before clinical signs are observed. Refer to *Chapter 12, Sudden Onset (Acute) Pneumonia*.

Bloat

Occasionally animals that develop bloat will be found dead. A full post-mortem can determine if bloat was the cause of death, rather than a result of death, since all animals will bloat after death. Refer to *Chapter 19, Bloat*.

Coccidiosis

Especially in young goats, exposed to a high dose of coccidia—the first sign of disease may be sudden death. Refer to *Chapter 11, Coccidiosis*.

Predation

Animals dying at pasture are prone to predation by scavengers. It is important to be mindful that scavenged carcasses may represent natural deaths rather than healthy animals taken by predators. The wound patterns on the carcass will help distinguish between the two.

Casting

Heavily pregnant ewes occasionally roll onto their backs and are unable to right themselves. They may bloat while cast, and casting also makes them easy prey for scavengers, especially carrion birds.



17. Important Diseases of Sheep and Goats

A number of diseases of sheep and goats can be considered especially important because they are either highly infectious to other sheep and goats, transmissible to humans, or important economically. Some of these diseases are controlled by governments while others are not. The details of many of these diseases can be found in other chapters of this manual.

Highly Transmissible	Zoonotic	Economic
Foot and Mouth Disease (FMD) <i>(this chapter)</i>	Anthrax <i>(Chapter 16)</i> Toxoplasmosis <i>(Chapter 6)</i> Chlamydial Abortion <i>(Chapter 6)</i>	Scrapie <i>(Chapter 10)</i> Foot Rot <i>(Chapter 9)</i> Cysticercus <i>(Chapter 20)</i> Maedi-Visna(MV) <i>(Chapter 12)</i> CAE <i>(Chapter 9)</i> Johne's Disease <i>(Chapter 13)</i> Infectious Abortion <i>(Chapter 6)</i>

Zoonosis
 – a disease of animals that may secondarily be transmitted to man.

Foot and Mouth Disease

Cause

Foot and mouth is perhaps the most important animal disease in the world. The virus affects all cloven footed animals and pigs. The disease causes small blisters to form on the mouth, lips and feet. The blisters cause pain and animals often lose a lot of weight. If pregnant, the animals may abort. The virus will also affect the hearts of newborn lambs and kids, killing them. The virus is very tough and highly contagious. It has been eradicated from North America. Because foot and mouth disease has been eradicated from most developed nations, there are very strict import restrictions to keep it out. If foot and mouth were to be diagnosed in Canada, we would immediately lose all export of agricultural products.

Clinical Signs

The small vesicles (blisters) on the mouth and feet are very distinctive, but are small and easily missed. Animals appear sick, off feed and are commonly lame. You have to look very closely to see the blisters.

Diagnosis

If you ever suspect foot and mouth disease **you must** contact your veterinarian or the local Canadian Food Inspection Agency (CFIA) office. It is important that you stay where you are and prevent all animal movement to limit the spread of disease. The CFIA will take samples for the laboratory to rule out FMD as the cause of disease.

Treatment

There is no treatment for this viral disease. Due to the highly infectious nature of the disease and the need to eradicate it, the CFIA's policy has been to slaughter all affected and in contact animals. Producers are paid compensation to cover the losses associated with the disease.

Control

The key to controlling foot and mouth disease in Canada is to keep the disease out of the country. Ensure that if you travel abroad, you never bring any animal products back with you. If you have such products or if have been in contact with livestock you should speak to the custom officials at the airport or port authorities.



18. Deficiency and Toxicity Syndromes

Deficiency

Much of the discussion on nutrient deficiencies has been done in other parts of this document. These can be found as follows:

Iodine – *Chapter 5, Goitre*

Vitamin E and Selenium – *Chapter 5, White Muscle Disease*

Copper – *Chapter 5, Swayback*

Thiamine – *Chapter 10, Polioencephalomalacia*

Magnesium – *Chapter 16, Grass Staggers*

Calcium – *Chapter 6, Hypocalcemia*

Refer to the *Nutrition Module* for additional information on nutrient deficiencies.

Toxicity

There are several situations in which materials might be consumed by sheep or goats which present a toxicity problem for the animal. These are discussed on the following pages.

Copper Toxicity

Cause

Sheep and goats differ in their tolerance to copper. In fact, different sheep and goat breeds have different susceptibilities to copper toxicity. Sheep generally are very susceptible to copper poisoning. Rations developed for cattle, goats, or horses should **never** be fed to sheep as they could cause disease. Other sources of copper include land fertilized with hog manure (which contains high levels of copper). Changes in the molybdenum content of the diet can also change copper absorption. Since copper is stored in the liver, at times of stress there may be a massive release of copper, which causes the acute disease.

Clinical Signs

Affected animals are depressed, off feed, weak and may have diarrhea. Many animals are simply found dead. Others may have red urine or may show yellowing of the mouth and eyes (jaundice).

Diagnosis

You need to have blood samples from affected animals or a post-mortem examination to confirm a diagnosis

Treatment

Treating affected animals is usually unrewarding; affected animals should probably be euthanized. If the animal is valuable, contact your veterinarian about treatment options.

Prevention

Never feed a ration or mineral mix to sheep that is not designed specifically for that species. When in doubt, have your feed analyzed and contact your veterinarian or a nutritionist.

Lead

Cause

Lead is mainly found in old batteries or old lead paint. An inquisitive animal may eat these products; young sheep and goats are particularly at risk.

Clinical Signs

In sheep the disease looks almost exactly like polio (refer to *Chapter 10, Polioencephalomalacia*). In goats it generally causes anorexia, weight loss, diarrhea and death.

Diagnosis

A diagnosis can be made using blood tests or a post-mortem examination.

Treatment

Although treatment is possible, it is rarely attempted.

Prevention

Ensure old car batteries are disposed of appropriately and that grazing animals do not have access to areas where lead based products are stored.

Bracken Fern

If there is little else available, sheep and goats will graze on bracken fern. This plant contains a number of different toxic elements which can cause polio (refer to *Chapter 10, Polioencephalomalacia*), retinal degeneration leading to blindness and suppression of bone marrow. It is best avoided.

Yew

The needles from yew trees are highly toxic. Animals should be kept away from yew trees and any clippings. Animals found dead from yew poisoning typically have the needles still in their mouths.

Sulphates

Cause

Sulphates are naturally present in soil. In low amounts they can interfere with copper absorption. In high concentrations they have a direct effect on the brain, causing a syndrome which is identical to polio (refer to *Chapter 10, Polioencephalomalacia*). The most common sources of sulphates are water and certain plants (for example, kochia weed and thistles). Surface water is problematic as the sulphate concentration may change, not only throughout the year but from year to year depending on the precipitation level. Well water should be analyzed to determine if the sulphate level is safe for livestock. The worst problems occur when there are very high concentrations of sulphate in the water and the forage is also high in sulphates.

Clinical Signs

Identical to polio. (Refer to *Chapter 10, Polioencephalomalacia*.)

Diagnosis

The only difference between sulphate poisoning and polio is that classical polio responds to thiamine treatment while sulphate poisoning does not. To finalize the diagnosis you must determine the sulphate content of the water and feed.

Treatment

None.

Prevention

Consider testing water sources annually. Water with more than 4,000 ppm sulphates should not be used for livestock. Water with sulphate levels of 200 to 4,000 ppm can be used with caution. If your water is not suitable for your livestock you may have to haul water from another source.

Sweet Clover

Cause

If hay made from sweet clover becomes mouldy, natural plant chemicals may be converted to dicoumerol which acts as an anti-coagulant, preventing the blood from clotting.

Clinical Signs

Animals may develop swellings associated with bleeding under the skin. Most affected animals die.

Diagnosis

Diets may be analyzed for dicoumerol content.

Treatment

Stop feeding contaminated feed and treat animals with vitamin K at a level of 1.3 mg/kg body weight, intramuscularly (im).

Prevention

Avoid sweet clover hay if there is evidence of mould.

Nitrate Poisoning

Cause

Nitrate exposure may come from fertilizer, contaminated water or plants (depending on the stage of their growth). Nitrates irritate the gastrointestinal tract, which may result in diarrhea. More importantly, nitrates are converted to nitrites by the rumen; nitrites react with hemoglobin in the red blood cells, preventing it from carrying oxygen.

Clinical Signs

Animals appear to be in respiratory distress, weak and stumbling. The mucous membranes of the mouth and eyes may be blue and blood may be brown in colour. Many affected animals will die; if animals are to be saved treatment must be initiated early and aggressively.

Treatment

If animals are to live, aggressive and immediate treatment must be initiated by your veterinarian. Contact your veterinarian at the first suspicion of nitrate exposure.

Prevention

Prevent animals from accessing fertilizer, contaminated feedstuffs or contaminated water sources. By timing the harvesting of high risk feeds, plant exposure can be controlled.

Alga Blooms (Cyanobacteria)

Cause

Stagnant sloughs occasionally develop “blooms” of blue-algae. Certain species of algae produce potent toxins, which are ingested when animals drink the water.

Clinical Signs

Animals are simply found dead.

Diagnosis

Immediately collect water samples for analysis.

Prevention

Prevent animals from drinking potentially contaminated water. Provide alternative water sources.