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Botulism Poisoning in Cattle in the Northern Territory

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Botulism is a significant cause of stock losses in the northern beef industry. It has been reported to be the most common animal health problem by producers in the Alice Springs, Barkly and Katherine regions of the Northern Territory[i].

Botulism outbreaks usually occur in unvaccinated herds, but can also occur in herds that are not properly vaccinated. Outbreaks have caused losses of up to 25% of the herd in some paddocks. The disease may also cause a persistent but undetected low level of mortality which may significantly increase the herd death rate by up to 10-20% annually.

With the introduction of vaccination as a standard management practice, the effect and frequency of this disease has been significantly reduced.

GEOGRAPHIC DISTRIBUTION

Botulism is an important disease in the northern regions of Australia, particularly where stock graze under range conditions and are subject to periods of protein and phosphorous deficiency. In southern Australia, botulism has been reported in feedlots and in dairy cattle under intensive feeding systems.

There are seven recognised types of botulism organisms but only two, types C and D, are important in cattle. These have been found to be widespread throughout the Northern Territory, northern Queensland and the Kimberley region.

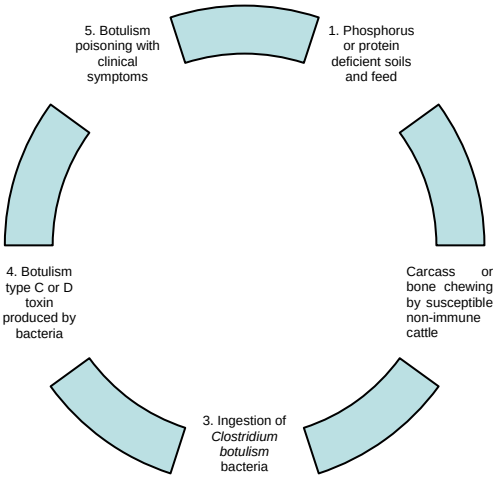
CAUSE

Botulism is caused by a toxin produced by the bacterium *Clostridium botulinum.* The bacteria belong to the clostridia group that includes the organisms that cause tetanus, malignant oedema (gas gangrene), enterotoxaemia (pulpy kidney), black leg and black disease. The bacterium is a spore forming an anaerobe that thrives in **decaying animal or plant material**. An anaerobic organism survives and multiplies in an oxygen-deficient or oxygen-free environment.

In anaerobic conditions, coupled with warmth and moisture, *C. botulinum* multiplies rapidly producing a highly lethal toxin. All warm-blooded animals can be paralysed by the botulism toxin. This toxin prevents nerve function resulting in paralysis. It is one of the most toxic poisons known, and cattle are one of the most susceptible species.

When conditions are not ideal, the bacterium can form highly resistant spores that can survive for years in the environment. The bacterium occurs commonly in the soil and is also found in the digestive tract of about 20% of normal cattle and other herbivores. This means that the organism can easily spread with cattle movements and then become established on a property in healthy cattle and soil.

The chain of events most commonly required for botulism poisoning to occur in NT cattle are:



Conditions are often most favourable for botulism to occur late in the dry season but the disease can occur at any time if conducive factors are prevalent.

Bones and carrion of decaying cattle and fly maggots are the best sources of toxin. Poisoning has also been known to occur by consuming water or feed that has been contaminated by animal carcasses and rotting feed **(mouldy hay or silage and decomposing grass tussocks)**. Another form of botulism, toxicoinfectious botulism, occurs when animals consume actively growing bacteria from the gastrointestinal tract of cattle where the toxin is subsequently produced.

Where unvaccinated cattle are kept on a protein and phosphorus deficient diet, and where carcass chewing is common, it is likely an outbreak of botulism will occur.

SUSCEPTIBILITY

The susceptibility of cattle in northern Australia to botulism poisoning is a complex issue that depends on the relative presence of the following six factors:

1. Phosphorous and protein deficiency. Lactating cows and growing cattle have a higher demand for phosphorus and protein. If such animals cannot meet these demands from the feed, they often develop a depraved appetite for carrion and bone chewing. The availability of phosphorus will vary with the soil type and seasonal conditions. Most of the NT is either phosphorous deficient or marginal. Even green pastures in the mid to late season are likely to be phosphorous-deficient. The protein content of native pastures is often insufficient through much of the dry season to maintain live-weight. Most NT stations now supplement their cattle. In a recent

survey, 93% of producers indicated that they fed supplement at some stage throughout the year[ii]. However, cost and practical problems mean that supplementation programs are not totally effective in satisfying need.

2. Carcass and bone chewing. This is commonly seen in the NT. It is habit forming and even when dietary phosphorus and protein are adequate, animals continue to chew bones and will be exposed to the risk of botulism poisoning. Adequate supplementation programs will not cure the problem in deficient areas.

3. Bacteria distribution. Bacteria producing type C and type D toxin are present in all pastoral regions of the NT.

4. Toxin. Cattle are very susceptible to botulism toxin. Toxin production occurs in an anaerobic environment, with moisture and an optimum temperature of around 23°C (15-35°C). All these conditions can be found in a rotting carcass. Toxin can last for a year at 30°C and is rapidly inactivated at 37°C, so the amount of toxin present is not constant. Not all carcasses are necessarily toxic but the proportion of toxic carcasses in tropical environments can be very high.

5. Any carcass. All decaying carcasses are potentially infective, including those of cattle, horses, donkeys, pigs, birds, wallabies and rodents.

6. Susceptible cattle. Unvaccinated and improperly vaccinated cattle can be assumed to be fully susceptible. Even properly vaccinated cattle can succumb if the amount of toxin is high enough to over power the body's immune system. Natural immunity can develop in cattle which have been exposed to the natural toxin and have recovered.

CLINICAL SYMPTOMS

Visual symptoms of botulism poisoning can vary dramatically depending on the amount of toxin ingested, pre-existing immunity and the stage of poisoning.

The period of time for the ingested toxin to show effect on the animal usually varies between three to seven days, depending on the quantity of toxin ingested. In very severe cases the animal can die in less than 24 hours without signs of illness. Animals may ingest low doses of toxin and not be affected, especially if they have antibodies to the toxin.

The **toxin binds to nerve endings and prevents nerve impulses to muscles. This leads to a floppy or flaccid paralysis, which usually progresses throughout the body.**..

Early symptoms result from the paralysis of the tongue, throat and the stomach. Often the earliest sign of botulism poisoning is refusal to drink and a lack of appetite, related to the paralysis of the throat. This leads to dehydration that is evident by sunken eyes, hollow paunch, unpliable skin and strange behaviour. The tongue may protrude and the animal may drop cud and drool saliva. The animal's voice may alter or fail.

The first observed signs may be depression, muscular weakness and incoordination which makes the animal appear lame. Muscle weakness and paralysis usually begins with the hind legs, and progresses forward to the forequarters, head and neck. The beast may be drowsy and stagger, stumble, knuckle over and possibly arch its back. The animal may become aggressive and often attempt to charge anyone who comes close. On falling down, the beast may appear very weak and have great difficulty in rising. It may not even be able to lift its head (limber neck). Initially the beast lies in a normal resting position with its head on the ground or turned towards the flank (sternal recumbency). As the paralysis worsens, the animal may go into a semiconscious state and when it goes onto its side (lateral recumbency) it is unlikely to get up again. Semicircular marks in the ground may be the only evident sign when a carcass is found, resulting from the uncontrolled paddling movement of the legs. Death usually results from respiratory failure or exposure between one to four days following the onset of clinical

symptoms, but can take up to 14 days. Most of the deaths occur within 1 km of water but carcasses can be scattered throughout the paddock.

DIAGNOSIS

Diagnosis of botulism is largely based on clinical signs suggestive of the disease. There are however, three field tests which provide supportive evidence for the disease. These tests are not definitive and include the following:

1. While the beast is still conscious, pull out the tongue. It is a natural reaction for cattle to retract the extended tongue. In a healthy beast this exercise can be very difficult. If paralysis has taken effect, the tongue can be easily grabbed and will remain extended. There may also be un-swallowed cud in the cheek pouches which is an indication of an inability to swallow. These signs are a very good indication of botulism in cattle, but are rarely seen.

An easy test for botulism – when the tongue is pulled out, it remains extended.

2. If it is possible to do a post-mortem, evidence of carrion consumption may be found in the rumen or reticulum (honeycomb) in the form of hide, bone or maggot pupae.

3. A post-mortem may also show the omasum (bible)) to be compacted and dry - known as "dry bible". This is probably due to dehydration and the effects may also be noticed elsewhere in the carcass.

Even if there are no visible symptoms in the carcass, a post-mortem is valuable to rule out other causes of death. Please consult a departmental veterinarians or Livestock Biosecurity Officer to investigate disease.

Field tests are often inconclusive as signs may not be seen. It is extremely difficult to prove death due to botulism even with the laboratory tests described below.

1. ELISA - Enzyme - Linked Immunosorbent Assay

This test is used to show that an animal has antibodies to the toxin in its blood serum. Antibodies may arise from natural exposure to a toxin or from vaccination.

The test can identify the type of toxin involved (type C or D) with natural infection, and the level of antibodies in the animal. Because of cross reactions following vaccination, it is not possible to differentiate between type C and D vaccination titres. This test is useful for assessing the success of a vaccination program and can be done on any group of animals where there is some doubt about their vaccination history. It is however, an expensive test.

In unvaccinated herds the ELISA test is very useful as a positive result shows natural exposure. It can be used together with the faecal culture test to confirm that animals have been exposed to botulism. A repeat sample taken from survivors two weeks following the outbreak should indicate rising levels of antibodies if botulism infection has occurred.

2. MPT - Mouse Protection test (Toxin neutralisation test)
This test relies on paralysing mice with an injection of a toxic bacterial growth or toxic serum from an affected beast, and then protecting them with specific type C or D botulism antiserum.

The test is good for identifying the presence of toxic botulism bacteria and is used with the ELISA test. However, it is not so useful in proving that a paralysed beast has botulism. This is because only very low doses of toxin are present for short periods in the bovine serum and the mouse is relatively resistant to the toxin compared to cattle.

3. Culture for the bacterium
The botulism organism can be grown from any gut contents or even carcass material. The best samples are from lower intestinal contents and maggots from carcasses. Once the organism is grown in the laboratory, tests are carried out to show that it is C. botulinum, and to identify the type. This test will show that a toxic bacterium may be present but it does not prove that it was the cause of death. It may have been present without ill effect.

TREATMENT

Treatment options are limited

Very early cases may respond to a purging of the intestinal tract to remove further toxin.

Once a beast has absorbed botulinum toxin and has become affected there is nothing that can be done to speed up recovery. Antiserum is very expensive to produce and only available in very small quantities. If nursing is attempted, the beast must not be drenched through the mouth as it cannot swallow. However, a tube may aide the delivery of fluids direct to the rumen. The kindest option may be euthanasia.

During an outbreak, vaccination may reduce potentially harmful effects if toxin is released by *C. botulinum* bacteria that have been consumed by cattle (toxicoinfectious form). Vaccination is not effective for cattle which have consumed toxin.

PREVENTION

Prevention is better than cure and the most practical way to prevent losses from botulism poisoning is by VACCINATION.

The three steps recommended for the prevention of botulism poisoning are:

1. Vaccination with bivalent botulism vaccine following a recommended program.

2. Supplementary feeding of cattle with phosphorus and protein.

3. Removal of all carcasses and bones.

Vaccination
Vaccination with a bivalent (type C and D) botulism vaccine is the most effective long-term prevention strategy. There is a range of botulism vaccines available. Conventional vaccines involve either an initial two shot program, one month apart, or a single shot followed by an annual booster shot. An alternative long-acting vaccine involves an initial single shot followed by a booster shot every three years. Both vaccines produce a similar level of protection and the decision on which vaccine to select will depend on cost and management practices. All vaccines require booster shots to maintain protective levels of immunity.

Calves

Vaccination of calves can occur from one month of age to produce effective immunity. Protective maternal antibody levels are depleted by six months of age. Properties conducting early weaning (60 kg+) programs should ensure that vaccination occurs at this early age.

Imported cattle

Often the vaccination history of imported cattle is overlooked. Botulism vaccination should take place along with other vaccinations before cattle are released into the herd.

Vaccine program failures do occur and it is important to store and administer the vaccine carefully. **Please read manufacturer instructions prior to use for storage, handling and vaccine program recommendations.** Station storage should be in a cool room at 4°C and at the yards in the shade in eskies with ice. Injection equipment including disposable items should be kept clean and well maintained or replaced.

Vaccine should be injected under the skin on the neck or behind the shoulder. Vaccine reactions can occur. This is often the result of injecting the vaccine into the skin or into the muscle. It is important to avoid sites close to the rumen, particularly the rumen fossa (triangular area under the hip) as the vaccine is destroyed if injected into the rumen.

Supplementation

The supplementation of non-protein nitrogen (e.g. urea) and phosphorous is a well recognised management practice. However, even the best supplementation programs will not completely prevent carcass or bone chewing.

Carcass removal

Removal of carcasses is not always an option under extensive range conditions where paddocks are large and the checking of stock is infrequent. However, the removal of all carcasses from areas of stock congregation (e.g. watering points) is important. Carcasses can be burnt, buried, locked up in the turkey nest enclosure or at least taken a considerable distance away.

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[i] Department of Primary Industry, Fisheries and Mines. Pastoral Industry Survey NT 2004. Northern Territory Government.
[ii] Department of Primary Industry, Fisheries and Mines. Pastoral Industry Survey NT 2004. Northern Territory Government.