

Botulism

*Shaker Foal Syndrome,
Limberneck, Western
Duck Sickness,
Bulbar Paralysis,
Loin Disease, Lamziekte,*

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Etiology

Botulism is caused by botulinum toxin, a potent neurotoxin produced by *Clostridium botulinum* and a few strains of *C. baratii* and *C. butyricum*. *Clostridium botulinum* is an anaerobic, Gram-positive, spore-forming rod.

Botulism can result from the ingestion of preformed toxin or the growth of *C. botulinum* in anaerobic tissues. Seven types of botulinum toxin, designated A through G, have been identified. Types A, B, E and F cause illness in humans. Type C is the most common cause of botulism in animals. Type D is sometimes seen in cattle and dogs, and type B can occur in horses. Types A and E are found occasionally in mink and birds. Type G rarely causes disease, although a few cases have been seen in humans. All types of botulinum toxin produce the same disease; however, the toxin type is important if antiserum is used for treatment.

Geographic Distribution

C. botulinum is found worldwide and cases of botulism can be seen anywhere. In ruminants, botulism mainly occurs in areas where phosphorus or protein deficiencies are found. Botulism is seen regularly in cattle in South Africa and sheep in Australia. This disease is rare in ruminants in the United States, although a few cases have been reported in Texas and Montana.

Transmission

C. botulinum and its spores are widely distributed in soils, sediments in fresh and coastal waters, the intestinal tracts of fish and mammals, and the gills and viscera of shellfish. The bacteria can only grow under anaerobic conditions. Botulism occurs when animals ingest preformed toxins in food or *C. botulinum* spores germinate in anaerobic tissues and produce toxins as they grow.

Botulism in Humans

In humans, botulism is classified into three forms: foodborne, wound, and infant or intestinal botulism. Foodborne botulism is the most common form and occurs when humans ingest toxins in various foods. The foods associated with botulism are usually low acid (pH greater than 4.6) and may include home-canned foods, sausages, meat products, canned vegetables and seafood products. Commercial foods are occasionally implicated. Wound botulism occurs when an anaerobic wound is contaminated with *C. botulinum*, usually from the soil. Infant botulism is seen only in children less than a year of age. In this form, *C. botulinum* spores germinate in the intestinal tract and produce toxin. Honey has been associated with some cases of infant botulism but spores can also be found in many other sources. Adults with altered intestinal flora, secondary to gastrointestinal surgery or antibiotic therapy, may also be able to develop this form.

Botulism in Animals

Preformed toxins in a variety of sources, including decaying vegetable matter (grass, hay, grain, spoiled silage) and carcasses can cause botulism in animals. Carnivores, including mink and commercially raised foxes, usually ingest the toxins in contaminated meat such as chopped raw meat or fish. Cattle in phosphorus-deficient areas may chew bones and scraps of attached meat; a gram of dried flesh may have enough botulinum toxin to kill a cow. Similar cases occur in Australia, where protein-deficient sheep sometimes eat the carcasses of rabbits and other small animals. Ruminants may also be fed hay or silage contaminated by toxin-containing carcasses of birds or mammals. Horses usually ingest the toxin in contaminated forage. Birds can ingest the toxins in maggots that have fed on contaminated carcasses or in dead invertebrates from water with decaying vegetation. Cannibalism and contaminated feed can also result in cases in poultry.

The toxicoinfectious form of botulism corresponds to the wound and intestinal forms in humans. *C. botulinum* may grow in necrotic areas in the liver and GI tract, abscesses in the navel and lungs, or anaerobic wounds in the skin and muscles. This form of botulism appears to be responsible for shaker foal syndrome in horses. Toxicoinfectious botulism is also seen in chickens, when broilers are intensively reared on litter; the cause of this phenomenon is unknown.



**Institute for International
Cooperation in Animal Biologics**
*An OIE Collaborating Center
Iowa State University
College of Veterinary Medicine*



IOWA STATE UNIVERSITY®

**Center for Food
Security and Public Health**
College of Veterinary Medicine
Iowa State University
Ames, Iowa 50011
Phone: (515) 294-7189
FAX: (515) 294-8259
E-mail: cfsph@iastate.edu
Web: <http://www.cfsph.iastate.edu>

Botulinum and Bioterrorism

In a bioterrorist attack, botulinum toxin could be delivered by aerosols, as well as food or water. After aerosol transmission, the clinical disease is expected to be similar to foodborne botulism.

Disinfection/ Inactivation

Botulinum toxins are large, easily denatured proteins. Toxins exposed to sunlight are inactivated within 1 to 3 hours. Botulinum can also be inactivated by 0.1% sodium hypochlorite, 0.1 N NaOH, heating to 80°C for 30 minutes or 100°C for 10 minutes. Chlorine and other disinfectants can destroy the toxins in water.

The vegetative cells of *Clostridium botulinum* are susceptible to many disinfectants, including 1% sodium hypochlorite and 70% ethanol. The spores are resistant to environmental conditions but can be destroyed by moist heat (120°C for at least 15 min).

Infections in Humans

Incubation Period

The incubation period for foodborne infections is a few hours to 10 days; most cases become symptomatic after 18 to 36 hours. Wound infections may become evident within a few days to two weeks. The incubation period for intestinal or infant botulism is unknown. Inhalation botulism usually develops 12 to 36 hours after exposure, but in some cases the incubation period may be up to several days.

Clinical Signs

Foodborne Infections

In foodborne infections, gastrointestinal disturbances – including nausea, vomiting and abdominal pain – are often the first sign. Either diarrhea or constipation may occur. As the disease progresses, a symmetric, descending flaccid paralysis develops in the motor and autonomic nerves. The clinical signs may include blurred or double vision, photophobia, drooping eyelids, slurred speech, dysphagia, urine retention, a dry mouth and muscle weakness. In untreated progressive infections, descending paralysis of the respiratory muscles, arms and legs is seen. Fatal respiratory paralysis may occur within 24 hours in severe cases. Fever is not usually seen.

Wound Botulism

Wound botulism is very similar to foodborne infections; however, gastrointestinal signs are not usually present and patients may have a wound exudate or develop a fever.

Infant Botulism

Most cases of infant botulism occur in 2-week to 6-month-old babies. The first symptom is usually constipation. Other signs may include lethargy, weakness, excessively long sleep periods, diminished suck and gag reflexes and dyspha-

gia with drooling. Some babies develop a weak or altered cry. In progressive cases, the infant may develop flaccid paralysis; a “floppy head” is typical. In severe cases, there may be respiratory arrest and death. The symptoms and severity of this disease vary considerably in different babies.

Intestinal botulism in adults

The initial symptoms of intestinal botulism in adults may include lassitude, weakness and vertigo. As the disease progresses, patients may experience double vision and have progressive difficulty speaking and swallowing. Other symptoms may include dyspnea, general muscle weakness, abdominal distention and constipation.

Communicability

No person-to-person transmission has been seen.

Diagnostic Tests

Botulism can tentatively diagnosed by the clinical signs and the exclusion of other neurologic diseases. The definitive diagnosis relies on identifying the toxin in feces, blood, vomitus, gastric aspirates, respiratory secretions or food samples. Feces are usually the most reliable clinical sample in foodborne or infant botulism; the toxin may be found for days or weeks in foodborne cases. Botulinum toxin is rarely found in the blood in adults but is occasionally detected in infants. The toxin can be identified by mouse inoculation studies (the mouse neutralization test), ELISAs or electrochemiluminescent (ECL) tests. Botulinum toxins can be typed with neutralization tests in mice. Serology is not useful for diagnosis, as small amounts of toxin are involved and survivors rarely develop antibodies.

C. botulinum can often be cultured from the feces in infant botulism or the wound in wound botulism. In foodborne cases, the food is usually cultured as well as tested for the toxin. *C. botulinum* is an anaerobic, Gram positive, spore-forming rod. On egg yolk medium, toxin-producing colonies usually display surface iridescence that extends beyond the colony. The iridescent zone around the colony is usually larger for C, D and E toxins.

Treatment and Vaccination

Supportive treatment, with respiratory support if necessary, is the cornerstone of treatment. Botulinum antitoxin, given early, may prevent the disease from progressing and decrease the duration of symptoms. In foodborne illness, the amount of toxin in the gastrointestinal tract can be reduced with stomach lavage and enemas. Antibiotics and debridement are used in cases of wound botulism. Antibiotics are also used occasionally in foodborne cases, but are not generally recommended in infant botulism as they may change the intestinal flora. Investigational vaccines may be available for humans who have a high risk of exposure.

Morbidity and Mortality

Outbreaks of botulism can occur worldwide. Approximately 10 to 30 outbreaks are seen annually in the United States. In 1999, 107 cases of infant botulism, 26 cases of foodborne botulism and 41 cases of wound botulism were reported in the United States.

The death rate is high in untreated cases, but has been decreasing with improvements in supportive care. Before 1950, the mortality rate was 60%; currently, it is less than 5%. Recovery may be slow and can take several months or longer. In some cases, survivors report fatigue and shortness of breath for years.

Botulinum toxins are known to have been weaponized by several countries and terrorist groups.

Infections in Animals

Species Affected

Many species of mammals and birds, as well as some fish, can be affected by botulism. Clinical disease is seen most often in wildfowl, poultry, mink, cattle, sheep, horses and some species of fish. Dogs, cats and pigs are resistant; botulism is seen occasionally in dogs and pigs but has not been reported from cats.

Incubation Period

The incubation period can be 2 hours to 2 weeks; in most cases, the symptoms appear after 12 to 24 hours. Mink are often found dead within 24 hours of ingesting the toxin.

Clinical Signs

Botulism is characterized by progressive motor paralysis. Typical clinical signs may include muscle paralysis, difficulty chewing and swallowing, visual disturbances and generalized weakness. Death usually results from paralysis of the respiratory or cardiac muscles.

Ruminants

In cattle, the symptoms may include drooling, restlessness, incoordination, urine retention, dysphagia and sternal recumbency. Lateral recumbent animals are usually very close to death. In sheep, the symptoms may include drooling, a serous nasal discharge, stiffness and incoordination. Abdominal respiration may be observed and the tail may switch on the side. As the disease progresses, the limbs may become paralyzed and death may occur.

Horses

The clinical signs in horses are similar to cattle. The symptoms may include restlessness, knuckling, incoordination, paralysis of the tongue, drooling and sternal recumbency. The muscle paralysis is progressive; it usually begins at the hindquarters and gradually moves to the front limbs, head and neck.

The shaker foal syndrome is usually seen in animals less than 4 weeks old. The most characteristic signs are a stilted gait, muscle tremors and the inability to stand for more than 4 to 5 minutes. Other symptoms may include dysphagia, constipation, mydriasis and frequent urination. In the later stages, foals usually develop tachycardia and dyspnea. Death generally occurs 24 to 72 hours after the initial symptoms and results from respiratory paralysis. Some foals are found dead without other clinical signs.

Pigs

Pigs are relatively resistant to botulism. Reported symptoms include anorexia, refusal to drink, vomiting, pupillary dilation and muscle paralysis.

Foxes and Mink

During outbreaks of botulism, many animals are typically found dead, while others have various degrees of paralysis and dyspnea. The clinical picture is similar in commercially raised foxes.

Birds

In poultry and wild birds, flaccid paralysis is usually seen in the legs, wings, neck and eyelids. Wildfowl with paralyzed necks may drown. Broiler chickens with the toxicoinfectious form may also have diarrhea with excess urates.

Communicability

Botulism is not communicable by casual contact but, in some cases, tissues from dead animals can be toxic if ingested by other animals.

Diagnostic Tests

Botulism can be difficult to diagnose, as the toxin is not always found in clinical samples or the feed. Diagnosis is often a matter of excluding other diseases. A definitive diagnosis can be made if botulinum toxin is identified in the feed, stomach or intestinal contents, vomitus or feces. The toxin is occasionally found in the blood in peracute cases. Botulinum toxin can be detected by a variety of techniques, including enzyme-linked immunosorbent assays (ELISAs), electrochemiluminescent (ECL) tests and mouse inoculation or feeding trials. The toxins can be typed with neutralization tests in mice.

In toxicoinfectious botulism, the organism can be cultured from tissues. *C. botulinum* is an anaerobic, Gram positive, spore-forming rod. On egg yolk medium, toxin-producing colonies usually display surface iridescence that extends beyond the colony. The iridescent zone around the colony is usually larger for C, D and E toxins.

Treatment and Vaccination

The treatment is usually supportive and may include gastric lavage to remove some of the toxin. Botulinum antitoxin is sometimes used in animals; the success rate may depend on the type of toxin causing the disease and the species of ani-

mal. Type C antitoxins have been effective in some outbreaks in birds and mink. There are also some reports of success with guanidine hydrochloride. Antibiotics are used in the toxico-infectious form, but are not always successful in birds.

In endemic areas, vaccines can be used in horses, cattle, sheep, goats, mink and pheasants. In chickens, they may not be cost-effective.

Morbidity and Mortality

Botulism is common in wild waterfowl; an estimated 10 to 50 thousand wild birds are killed annually. In some large outbreaks, a million or more birds may die. Ducks appear to be affected most often. Botulism also affects commercially raised poultry. In chickens, the mortality rate varies from a few birds to 40% of the flock. Some affected birds may recover without treatment.

Botulism seems to be relatively uncommon in most domestic mammals; however, in some parts of the world, epidemics with up to 65% morbidity are seen in cattle. The prognosis is poor in recumbent large animals. In cattle, death generally occurs within 6 to 72 hours after sternal recumbency. Most dogs with botulism recover within 2 weeks.

Post-Mortem Lesions [Click to view images](#)

There are no pathognomonic lesions; the lesions are usually the result of general muscle paralysis. Respiratory paralysis may cause nonspecific signs in the lungs. In shaker foal syndrome, the most consist lesions are excess pericardial fluid with strands of fibrin, pulmonary edema and congestion. Foreign material in the fore-stomachs or stomach may suggest botulism.

Internet Resources

- Animal Health Australia. The National Animal Health Information System (NAHIS)
<http://www.brs.gov.au/usr-bin/aphb/ahsq?dislist=alpha>
- Bacteriological Analytical Manual Online
<http://www.cfsan.fda.gov/~ebam/bam-toc.html>
- Centers for Disease Control and Prevention (CDC)
http://www.cdc.gov/ncidod/dbmd/diseaseinfo/botulism_t.htm
- Manual on Meat Inspection for Developing Countries
<http://www.fao.org/docrep/003/t0756e/t0756e00.htm>
- Material Safety Data Sheets – Canadian Laboratory Center for Disease Control
<http://www.hc-sc.gc.ca/pphb-dgsp/msds-ftss/index.html#menu>
- Medical Microbiology
<http://www.gsbs.utmb.edu/microbook>
- The Merck Manual
<http://www.merck.com/pubs/mmanual/>
- The Merck Veterinary Manual
<http://www.merckvetmanual.com/mvm/index.jsp>

U.S. FDA Foodborne Pathogenic Microorganisms and Natural Toxins Handbook (Bad Bug Book)
<http://vm.cfsan.fda.gov/~mow/intro.html>

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