



## BOTULISM

[Definition](#)  
[Clinical Signs](#)  
[Risk Factors](#)  
[Transmission](#)

[Diagnostic Sampling, Testing  
and Handling](#)  
[Post-mortem](#)  
[Specific Control Measures](#)

[Release of Animals from  
Isolation](#)  
[Biosecurity Issues for  
Receiving Animals](#)  
[Zoonotic Potential](#)

**Definition** Botulism is a progressive, flaccid paralysis resulting from *Clostridium botulinum* toxin production and absorption that can be rapidly fatal if not aggressively treated from the first signs of intoxication.

### Biology

*Clostridium botulinum* is an anaerobic, spore-forming bacterium, ubiquitous in soil.

There are four distinct groups of *C. botulinum* (groups I-IV), and 8 different toxins (A, B, C [C1, C2], D, E, F, and G). Type A is prevalent in the western US. Type B spores are found in the soils of the Northeast and Appalachian regions (mid-Atlantic states). Type C is present in the southeast (Florida). Clinically, Type B is the most commonly reported intoxication (responsible for greater than 85% of cases).

Botulism is a rapidly progressive neurologic disease with high mortality (100% in untreated animals).

**Clinical Signs** Regardless of the mechanism or route by which intoxication occurs, the clinical events are the same for every horse. The course of the disease can be very rapid, being 24 to 48 hours depending on the level of toxin or bacterial ingestion. Wound contamination and toxoinfectious cases may have variable and prolonged periods from inoculation of the organism until toxin release precipitating clinical signs.

Clinical signs include:

- Severe muscle weakness
- Flaccid paralysis with normal mentation
- Inability to swallow (foals will reflux milk from nostrils)
- Poor tail, tongue and eyelid tone
- Hypoventilation, respiratory arrest
- Paresis/inability to stand for extended periods
- Limb paralysis
- Progression to muscular weakness and recumbency
- Drooling



- Muscle trembling (particularly in foals)
- Unexplained Mydriasis with sluggish pupillary light reflexes
- Tachycardia (especially in foals)
- Colic and decreased gastrointestinal motility
- Sudden unexplained death
- Inability to rise after lying down

The interval between the onset of clinical signs and initiation of treatment is extremely important with respect to outcome. Immediate referral to a facility equipped to handle recumbent horses and access to antitoxin is imperative.

**Risk Factors**

- Decaying vegetable matter in food and water
- Soil contamination of feed (access to round bales)
- Animal carcass contamination (bird, fish, rodents, etc.) of feed, or standing water

**Transmission**

Three clinical scenarios are possible:

- Forage poisoning—consuming feed or forage containing pre-formed toxins of *C. botulinum*
- Wound contamination—allowing germination of spores and absorption of toxin from hypoxic areas
- Toxinfectious (“Shaker foals”)—consuming forage or feed containing the bacteria or spores by neonates suffering gastric ulceration may precipitate diseases. Necrotic/hypoxic foci allow germination of *C. botulinum* spores and development of the vegetative bacterial form, enabling production, liberation and local absorption of causative toxins

**Diagnostic Sampling, Testing, and Handling**

**Diagnosis**

Botulism should be the primary concern for horses suffering flaccid paralysis without any signs of precipitating conditions.

Diagnosis is by elimination of other causes of neurologic disease-causing weakness and recumbency: EPM, EHM, arboviral encephalitides, rabies, metabolic derangements, myopathies, and other intoxications. Blood work and evaluation for other neurological diseases is uniformly negative. Currently, there is no currently commercially available antemortem test.

Based on known type prevalence in a geographic region and suspected route of exposure, the most likely type affecting a horse with suspected botulism can be deduced. Definitive toxin type diagnosis requires laboratory testing.

<u>Sample</u>	<u>Test</u>	<u>Shipping</u>	<u>Handling</u>
20 grams fresh feces	Identification of <i>C. botulinum</i> spores	Leak-proof container	Chilled overnight

<u>Sample</u>	<u>Test</u>	<u>Shipping</u>	<u>Handling</u>
20 grams or 2 ounces of stomach contents or intestinal contents	Identification of <i>C. botulinum</i> spores	Leak-proof container	Chilled overnight
Dead animal from feed OR water source	Identification of <i>C. botulinum</i> spores or toxin	Leak-proof container	Chilled overnight
Soil from under affected areas	Identification of <i>C. botulinum</i> spores	Leak-proof container	Chilled overnight
Serum** *It is very difficult to isolate toxin from horses' sera. Send additional samples along with sera.	Minimum of 5cc. Provide as much as possible to increase detection of preformed toxin	Leak-proof container	Send sera off clot and frozen prior to shipping overnight

Definitive diagnosis is achieved by identification of the toxin in plasma, liver, or gastrointestinal tract contents.

- Tentative diagnosis is based on identification of *C. botulinum* spores in gastrointestinal contents or wounds
- Animals that recover from the disease do have antibody present but commercially available testing is limited

There is no demonstrable shedding of *C. botulinum* once clinical signs occur, particularly if the source of infection is a wound or an infected umbilicus.

Toxins are susceptible to sunlight, 1–3 hours.

**Post-Mortem**

There are no gross lesions associated with botulism. No pathologic findings are directly attributable to the effects of botulism toxin. Horses that have been recumbent may have pressure sores or self-trauma.

Recumbency and respiratory paralysis may cause nonspecific signs in the lungs. There may be evidence of aspiration pneumonia due to secondary to dysphagia.

Toxinfectious (shaker foal syndrome): the most consistent lesions are excess pericardial fluid with strands of fibrin, pulmonary edema and congestion.



<b>Specific Control Measures</b>	<p>Collection of intestinal contents at necropsy may be helpful for further testing; however, confirmatory diagnosis is difficult to achieve.</p> <p><b>Environmental management</b></p> <ul style="list-style-type: none"> <li>• Feed and water management</li> <li>• Remove old hay from around feeders; do not feed grass clippings</li> </ul> <p>Bleach is an effective disinfectant (after thorough removal of organic material) for toxins and/or vegetative cells.</p> <p>Clostridial spores are resistant to most environmental conditions and disinfectants.</p> <p><u><a href="#">Vaccination</a></u></p> <p>Understanding of regional variation in the prevailing antigenic type is helpful in determining utility of vaccination. Vaccine is for prevention of neurotoxin type B only.</p> <ul style="list-style-type: none"> <li>• Vaccination is recommended in endemic areas or if horse will travel to endemic area and be stabled in those areas</li> <li>• Adults: Broodmares and other adult horses, 3 dose series and annually.</li> <li>• Foals from vaccinated mares: Vaccinate starting at 1 month (see vaccine recommendations)</li> <li>• Foals from unvaccinated mares: Vaccinate as early as 2 weeks of age (see vaccine recommendations), plasma transfer from vaccinated horse</li> </ul>
<b>Release of Animals from Isolation</b>	There are no isolation requirements for horses with this disease.
<b>Biosecurity Issues for Receiving Animals</b>	There are no biosecurity issues over housing and/or handling of these animals.
<b>Zoonotic Potential</b>	None.

### Useful links

[Confirmatory testing: University of Pennsylvania Botulism Diagnostic Laboratory](#)

[Shipping and handling of the samples](#)