

# Botulism: A Deadly Disease That Can Affect Your Horse

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**B**otulism is a deadly disease caused by the toxins produced by the bacterium *Clostridium botulinum*. The botulinum toxin is a potent neurotoxin that impairs nerve function, including those of the diaphragm, leading to paralysis. When the nerves to the diaphragm are paralyzed, the affected animal stops breathing and will die as a result.

*C. botulinum* is an anaerobic (lives without oxygen), gram-positive, spore-forming bacterium. The toxin produced by *C. botulinum* is one of the most potent toxins known to science. The *Clostridium* family is known for causing rapid, severe, and deadly diseases, including botulism, tetanus, blackleg, malignant edema, etc. Sometimes the onset of clostridial disease is so rapid that no clinical signs are noted, and the owner simply finds the animal dead.

The botulism toxin blocks the release of acetylcholine in the neuromuscular junction. Acetylcholine is a neurotransmitter, which transmits information from the nerve cell to the muscle cell. It is released in the neuromuscular junctions (the space where nerves communicate and stimulate muscles), and it enables muscle contraction; therefore, without acetylcholine, the muscles will not contract. A very important muscle in any mammal's survival is the diaphragm, which, when contracted, enables breathing. Ultimately, without acetylcholine, a horse infected with botulism will die of respiratory failure and asphyxia, due to the paralysis of the diaphragm.



**Figure 1:** Horses eating from round-baled hay are more susceptible to developing botulism, especially if the hay was not baled appropriately or if a dead animal was baled together with the hay.

There are seven types of botulism recognized (A, B, Ca, Cb, D, E, F, and G), based on the antigenic specificity of the toxin produced by each strain. Types A, B, E, and F cause human botulism. Types A, B, C, and D cause most cases of botulism in animals. In horses, type B botulism is responsible for more than 80% of the cases.

The bacterium and its spores are widely distributed in nature. They are found in soil, sediments of streams and lakes, and in the intestinal tracts of fish and mammals. The bacteria will produce toxins under conditions of decaying plants and animals.

A horse can get botulism in three ways:

- By consuming forage or feed containing the bacteria, which will then produce the toxins in the intestinal tract (more commonly found in foals, known as shaker foal syndrome, or toxicoinfectious botulism).

- By consuming feed or forage containing the pre-formed toxins of *C. botulinum* (known as forage poisoning).
- Through wounds contaminated with the bacteria, generally puncture wounds. Wound borders will close, providing an anaerobic environment, which is a favorable condition for the bacteria to produce the toxins.

Although the incidence of the disease is low, it is of considerable concern because of its high mortality rate if not addressed and treated immediately and properly. The mid-Atlantic region of the eastern United States and especially Kentucky is where botulism is most commonly found, although the disease is reported worldwide. The spores of *C. botulinum* Type B can be found in the soil of most regions of the United States, although they are more frequently found in the northeastern and Appalachian regions. The western region is more abundant with *C. botulinum* type A, and type C occurs mainly in Florida.

The frequency of occurrence of foodborne botulism in humans and in horses correlates with the distribution of the types of spores in the soil.

## The Different Faces of Botulism

Forage poisoning can occur when horses eat spoiled forage or any type of feed contaminated by a decaying animal. Spoilage is common in hay that was baled with a high moisture content. In cases associated with hay, horses eating round-baled hay are at higher risk of developing the disease. Also, horses consuming hay that is spilled on the ground and then allowed to be mixed with soil and feces are at increased risk for botulism. Moreover, botulism can occur when dead animals accidentally get baled in hay during harvest. The decomposing carcass is an excellent anaerobic incubator for botulism spores present in the intestinal tract of the dead animal or bird. Once toxin is formed in the carcass, it leaches out and contaminates the hay or other feed material. Improperly ensiled silage and haylage with a high moisture content and alkaline pH (pH >4.5) provide optimal conditions for the production of botulinum toxin. Improperly fermented silage (pH >4.5) should not be fed to horses because it predictably causes botulism. In contrast, cattle are more resistant to botulism and may not develop the disease as readily as horses when fed spoiled silage, although there have been several reports of cases of botulism in cattle.

Botulism in foals, also known as shaker foal syndrome, happens when the foal starts to nibble at hay and grass, between 2 and 12 weeks of age, and ingests the bacteria directly from the soil. The bacteria will form spores in the intestinal tract of the foal and produce toxins. One interesting fact is that the normal flora of adult horses inhibit the inraintestinal growth of botulinum spores, limiting the occurrence of toxicoinfectious botulism to neonates. The toxins are absorbed into



**Figure 2:** When a young foal, especially from an unvaccinated mare, starts to eat grass and hay, its chances of getting botulism increase.

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### **Botulism can be prevented for most cases with a vaccination protocol and good management practices.**

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the bloodstream and cause the blockage in the neuromuscular junction, which keeps the muscles from contracting. The foal will start to show signs of difficulty in standing, shaking (thus, the name of the syndrome), and then falling down over and over again. The foal will not be able to suckle and will, if not diagnosed and treated in time, eventually die of respiratory failure.

Wound botulism occurs when *C. botulinum* contaminates a wound, such as a deep puncture wound, where the wound borders close, creating an anaerobic environment. The bacteria then produce the deadly toxins anaerobically. It has been seen after castration and been associated with injection abscesses, puncture wounds, trauma, and surgery in adult horses and in foals with umbilical hernias treated with clamps. This mechanism is similar to tetanus infection.

Regardless of the port of entry, the sequence of events that follow is the same for every horse. Once the toxin enters the body, it reaches the bloodstream and is distributed to nerves throughout the body. The toxin then prevents the transmission of impulses from the nerve to the muscle, impeding muscle contraction and leading to paralysis. The course of the disease is very rapid, taking only one to two days to cause death, depending on the dose of toxin or bacteria ingested.

## Clinical Signs and Diagnosis

Clinical signs of botulism in horses are weakness; decreased muscle tone of the tail, eyelid, and tongue; trembling; dilated pupils; lying down; difficulty in swallowing; drooling; and green or milky nasal discharge. Foals will nurse for about one minute, start to shake, and collapse on the floor. They will not be able to swallow the milk, and the owner will notice the milk all around the muzzle of the foal or the foal drooling the milk. In the same way, adult horses may drop the grain, push the grain around the feed bucket, cover it in saliva, and not be able to actually eat it. Eventually, the horse will be lying down more often, be unable to stand, or stand with an “elephant-on-a-ball” posture, with all four feet placed close together under the body. As the horse gets weaker and cannot stand anymore, it may collapse instead of deliberately folding its legs under itself as a normal horse would do. Finally, as the chest muscles and diaphragm get involved, respiratory failure occurs, and the horse dies of asphyxia.

The disease can hit suddenly and result in the death of horses that were perfectly healthy the day before. Botulism causes a flaccid paralysis, unlike tetanus that causes a rigid paralysis.

One of the worst aspects of botulism is that only the motor nerves are affected, the ones that are responsible for muscle movement. As such, the sensory function is left unimpaired. This means that victims of botulism will continue to experience hunger, thirst, fear, distended bladder, pain, and all other sensations, but they simply cannot move in response.

Clinical signs, history, and environmental observation are used to make a tentative diagnosis. If one horse in a group shows signs of botulism, others may quickly follow, if they have been fed from the same source of contaminated hay or feed.

Botulism is clinically diagnosed and more challenging to diagnose with laboratory tests. It is difficult to detect botulinum toxin in animals or to isolate the toxin from feedstuffs and feces of an affected horse. Routine blood tests are found normal (suspect botulism if the blood work is normal, yet the horse is showing signs of weakness!). Therefore, there may be more actual cases of botulism than the ones that are diagnosed. If clinical signs are compatible with botulism and the hay being fed is spoiled and contains areas of moist, decomposing material or a dead animal, or if a foal is involved, one should suspect botulism.

## Treatment

Treating a horse with botulism can be very costly, difficult, and often too late. It is better to prevent the disease than to treat it. Recommended treatment for botulism includes early administration of hyperimmune plasma containing antitoxin. The antitoxin binds to the toxin

molecules that are free floating in the bloodstream and neutralizes them before they bind to nerve cells, but they cannot reverse the effects of bound toxin. The bond that forms between the toxin and the nerve cell is irreversible.

The horse's body can make new neuromuscular junctions to replace the ones that are affected by the toxins; however, this process requires 7 to 10 days. It is a challenge to keep a horse alive that is recumbent and cannot eat or drink. In adult horses, being recumbent for a few days poses a problem in itself. They can develop pressure sores, colic, muscle damage, etc. Moreover, the horse will need to be mechanically ventilated and administered supportive therapy. However, it is very difficult to keep an adult horse on a ventilator for days, as the available machines are not designed to support this workload. If the paralysis has extended to the breathing muscles of an adult horse, it is humane to euthanize it.

## Prevention

Horse owners should be cautious about feeding hay that has been rained on during the harvesting phases. Round-baled hay is particularly a risk factor when baled at excessive moisture content. Any hay with rotten or decaying material should not be fed to horses.

Since the spoiled material is most likely to be internal in round hay bales, it may be impossible to visually determine this condition unless the bales are opened. If the exterior of the bale is rotten with dark discoloration and moldy or if the bales feel warm, they should not be fed to horses. An unspoiled round bale, put out for a group of horses, is generally not a problem.

There is also a risk for botulism if horses are being fed silage or haylage, especially if the fermentation process was inadequate to lower the pH to inhibit the growth of the bacteria and toxin production. Haylage, silage, and high-moisture hay are more prone to spoilage. For people who own horses and cattle, and thus feed silage to all their animals, it is important to mention that cattle are not as sensitive to botulism as horses, but they do die from this disease.

## Vaccination

There is a USDA-approved vaccine available to prevent botulism. The vaccine can be purchased from your veterinarian. Talk with your veterinarian about the best vaccination schedule for your herd. The following vaccination schedule is the proposed schedule of the American Association of Equine Practitioners (AAEP).

<b>Vaccination Chart: Foals and Weanlings (&lt;12 months of age)</b>	
<b>Mares vaccinated in the prepartum period</b>	<b>Unvaccinated mares</b>
3-dose series: 1st dose: 2 to 3 months of age 2nd dose: 4 weeks after 1st dose 3rd dose: 4 weeks after 2nd dose	3-dose series: 1st dose: 1 to 3 months of age 2nd dose: 4 weeks after 1st dose 3rd dose: 4 weeks after 2nd dose
Maternal antibody does not interfere with vaccination; foals at high risk may be vaccinated as early as 2 weeks of age.	

<b>Vaccination Chart: Adult Horses</b>			
<b>Broodmares</b>		<b>Other Adult Horses (&gt; 1 year of age)</b>	
<b>Previously vaccinated</b>	<b>Previously unvaccinated or having unknown vaccination history</b>	<b>Previously vaccinated</b>	<b>Previously unvaccinated or having unknown vaccination history</b>
1 dose, annually, 4 to 6 weeks prepartum	3-dose series: 1st dose: at 8 months' gestation 2nd dose: 4 weeks after 1st dose 3rd dose: 4 weeks after 2nd dose	Annual revaccination	3-dose series: 2nd dose: 4 weeks after 1st dose 3rd dose: 4 weeks after 2nd dose  Annual revaccination

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