

# Botulism

*A fatal neurologic disease of horses mostly caused by C. botulinum type B, for which there is a vaccine*

## Overview

Botulism is a fatal neurologic disease of horses caused by toxins produced by the anaerobic, spore-forming, soil-dwelling bacterium *Clostridium botulinum*. Eight different toxins are produced by *C. botulinum*, but type B is associated with the majority (85%) of botulism cases in North America.<sup>1</sup> Type B toxin is found in forage and soil and is inadvertently ingested by the horse (or it contaminates wounds). Types A and C also affect horses. Type A is associated with consumption of contaminated forage and type C is caused by rotting carcasses that contaminate horses' food or water.<sup>2</sup>

The botulinum toxin enters the bloodstream, circulates throughout the body, and enters various motor nerve cells (neurons). The toxins then travel to the end of these neurons (where they innervate muscle tissues) and prevent the transmission of the nerve impulses to the muscle cells. As a result, the horse becomes weak and potentially paralyzed.<sup>1</sup>

There are three different ways horses are exposed to botulinum toxins:<sup>1,2,3</sup>

**Ingesting the toxins directly** This is also called forage poisoning and is the primary form of botulism in adult horses

**Ingesting spores and absorbing the toxins from the gastrointestinal tract** This is also referred to as toxicoinfectious botulism or shaker foal syndrome. Toxicoinfectious botulism is the second-most-common form of botulism in horses. Foals ingest the bacterium from soil while grazing. *C. botulinum* colonizes the gastrointestinal tract and subsequently produces toxin which is absorbed into the bloodstream.

**Contamination of wounds** *C. botulinum* spores invade wounds such as castration sites, umbilical hernia repairs, and deep puncture wounds. The bacterium produces toxins within the wounds which are absorbed systemically causing disease.

Botulinum toxins are very potent, and



Silage, seen here, is made by packaging forage while it's still moist in airtight plastic. If something goes wrong in the fermentation process and the acidity does not increase fast enough to inhibit bacterial growth, such as that of *C. botulinum*, the silage can become a toxin factory.

horses are extremely sensitive to these toxins (it takes more toxin to kill a mouse than a horse).<sup>2</sup> Thus, clinical signs of disease or even death can occur very rapidly post-exposure. For example, ingestion of large amounts of contaminated feed can lead to clinical signs of disease within 12-24 hours. In contrast, it can take up to seven to 10 days before botulism is noted in horses that ingested a small amount of toxin.<sup>1</sup>

## Clinical Signs

The earliest clinical signs of botulism in adult horses usually include drooling, dropping food, dysphagia (inability to swallow), and inappetence/anorexia. Pacing, weakness, exercise intolerance, muscle tremors, depression, lying down more frequently or for longer periods of time, and recumbency (inability to rise) are also noted. A characteristic sign of botulism prior to onset of recumbency is reduced tongue strength and slow or absent tongue retraction.

In foals common clinical signs include an increased amount of time lying down. When forced to stand, they rapidly develop muscle tremors (hence the term "shaker foal") and flop to the ground in lateral recumbency. Additional signs include drooling, weak eyelid tone, a tongue that is easily pulled from the mouth and only slowly retracted, constipation, and ileus (lack of intestinal contractions). Affected foals are otherwise bright and alert. Foals are typically between two and five weeks old, but cases up to eight months of age have been reported.

If untreated the toxin blocks nerve transmission to major muscle groups throughout the body—including the diaphragm—resulting in respiratory arrest.

## Diagnosis

Botulism can be challenging to diagnose, and a large number of diseases can look like botulism including rabies—a potentially fatal neurologic disease capable of infecting humans. Other diseases to consider are equine protozoal myeloencephalitis (EPM), the viral encephalitides (e.g., Eastern, Western, and Venezuelan equine encephalitis), the neurologic form of equine herpesvirus-1 (EHV-1), West Nile virus, wobbler syndrome (cervical vertebral myelopathy), white muscle disease, and hyperkalemic period paralysis, among others. Call your veterinarian immediately to examine any horse exhibiting neurologic signs.

A definitive diagnosis of botulism can only be achieved by demonstrating the presence of the botulinum toxin in the bloodstream, demonstrating *C. botulinum* spores in the gastrointestinal tract and/or feedstuffs, or identification of an antibody response to *C. botulinum* in recovered patients. Since each of these tests is challenging and time consuming, botulism is generally diagnosed by excluding other potential diseases. Complete blood counts

(CBCs) and serum biochemistry generally show few or no changes early in the course of disease. Response to treatment is also a common method of “confirming” a diagnosis of botulism.<sup>1</sup>

### Treatment

While some horses with a slow onset and mild course of disease can survive without treatment, botulism is usually fatal if left untreated. The treatment of choice is the intravenous administration of a single dose of botulism antiserum. The antiserum contains neutralizing antibodies that bind to the botulinum toxins that are circulating in the horse’s bloodstream. The antiserum has no effect on the toxin that has already blocked the junction between the neuron and the muscle cell. Thus, intensive nursing care is required for affected horses until new neuromuscular junctions are created (approximately 10 days). Confine affected horses to a padded stall and provide food and water (or milk in foals) via nasogastric intubation. Muzzle horses to minimize the chances of an aspiration pneumonia, administer mineral oil as a cathartic to prevent a large colon impaction,

catheterize the urinary bladder for horses unable to urinate, maintain recumbent horses in a sternal position, and minimize the development of pressure sores.<sup>1,2</sup>

### Prognosis

Prognosis depends on the dose of botulinum toxin and whether or not the horse is recumbent. Horses that remain standing often regain the ability to swallow within three to seven days after antitoxin administration. A full recovery for these horses typically occurs in about one month.

Once a horse has been recumbent for 24 hours the prognosis is very poor. Recumbent foals have a better prognosis than adult horses and many affected foals can stand again seven to 10 days after treatment assuming intensive nursing care is provided. Amazingly, horses and foals that do recover tend to have a full recovery and show no residual neurologic deficits.<sup>1</sup>

### Prevention

One vaccine is available against *C. botulinum* type B. The American Association of Equine Practitioners recommends vaccinating broodmares annually four to six

weeks prior to foaling. The antibody levels in a vaccinated mare’s colostrum are thought to protect the foals against shaker foal syndrome for up to eight weeks postpartum. High-risk foals can be vaccinated. Three doses administered four weeks apart can be administered as early as two weeks of age. Work with your veterinarian to develop the most appropriate vaccine schedule for your horse(s).<sup>3</sup> Other ways to avoid botulism are to provide high-quality feeds and to remove carcasses (e.g., dead water fowl) as soon as possible. 🐾

### REFERENCES

1. Whitlock, R.H. Botulism (Shaker Foals; Forage Poisoning). Smith, B.P., ed. In: Large Animal Internal Medicine. Mosby, 2002. 1304-1306.
2. King M. Beat Botulism. March 2010 edition of The Horse.
3. American Association of Equine Practitioners. Botulism. <http://www.aaep.org/botulism.htm>.

More information on botulism is also available on the botulism Horse Course by Robert H Whitlock, DVM, PhD, [www.TheHorse.com/Videos/Horse-Courses.aspx](http://www.TheHorse.com/Videos/Horse-Courses.aspx).

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