Horses are highly susceptible to botulism, which causes neurologic disease and death.

Clostridium botulinum neurotoxin (BoNT), the underlying cause of botulism, tops the world’s list of potent toxins, which means BoNT is more poisonous than snake and spider toxins, arsenic, and mercury. Compared to other, smaller mammals, horses are much more sensitive to BoNT. For example, it takes less BoNT to kill a horse than it does to kill a mouse.

**WHAT IS BOTULISM?**

Botulism in horses results following ingestion or exposure to BoNT produced by the bacterium *C. botulinum*. This ubiquitous, soil-dwelling, spore-forming bacterium produces several distinct forms of the highly potent BoNT (labeled A through H), as well as several subtypes. Of those, type B most commonly affects horses and foals. Types A and C also occur in the United States, but far less frequently.

Once absorbed by its victim, BoNT blocks the transmission of impulses, or signals, between nerves and muscles. Due to this “radio silence,” muscles throughout the body become progressively weak, especially those in the head and neck. Paralysis promptly ensues, which all too frequently results in the rapid demise of affected individuals.

**EXPOSURE IN HORSES**

Regardless of the exact “type” of toxin, horses and foals typically become victims of BoNT through one of the following routes:

1. **Forage Poisoning**
   - Horses (and sometimes foals) ingest BoNT directly, usually when offered hay or haylage contaminated with *C. botulinum*. Forages become contaminated with the soil-dwelling bacterium either during raking and baling or due to improper storage. Deceased animals trapped in forage during feed processing or where baleage or hay bales are stored allows BoNT to enter feed (e.g., through packaging punctures). Unfortunately, offending feeds don’t necessarily look or smell spoiled, making it almost impossible to pre-emptively screen for spoiled sustenance. In herd situations, outbreaks can occur, with several adult horses falling ill simultaneously.

2. **Ingesting C. botulinum Spores**
   - Foals, being naturally inquisitive, ingest bacterial spores from the soil while exploring their environment and grazing. The spores then produce BoNT, which is absorbed from the gastrointestinal tract into the bloodstream and subsequently spread throughout the foal’s body. This form of botulism is usually referred to as toxicoinfectious botulism or “shaker foal syndrome,” so named because of the violent muscle tremors affected foals exhibit.

3. **Wound Contamination**
   - *C. botulinum* spores can invade wounds, such as castration sites, umbilical hernia repairs, and deep punctures. The spores then produce toxins within the wounds that, once absorbed systemically, cause clinical signs of disease.

**CLINICAL SIGNS**

In adult horses, the head and neck muscles typically become affected first. Characteristic signs of botulism prior to recumbency (down and unable to rise) include reduced tongue strength and slow or absent tongue retraction. Other classic signs of botulism include:

- Drooling, dropping food, dysphagia (inability to swallow), and inappetence/anorexia;
- Pacing;
- Weakness, exercise intolerance;
- Muscle tremors;
- Depression; and
- Lying down more frequently or for longer periods of time and, ultimately, recumbency.

Although the botulism disease process is the same as in adult horses, foals with botulism remain bright and responsive and show slightly different signs of disease, such as:

- An increased amount of time lying down;
- Rapidly developing muscle tremors when encouraged to stand and a tendency to flop easily to the ground in lateral recumbency;
- Drooling;
- Weak eyelid tone;
- A tongue that is easily pulled from the mouth and retracts slowly; and
- Constipation and ileus (lack of intestinal contractions).

**DIAGNOSIS**

Veterinarians can find botulism challenging to diagnose, especially when only one horse in a herd is affected. Diagnosing botulism using laboratory tests can be time-consuming, expensive, and worst of all, incorrect.

Due to the continued lack of a sensitive “gold standard” test for botulism, diagnosing botulism remains clinical, meaning the veterinarian has made every attempt to rule out all other possible causes of disease, leaving botulism highly suspected. Differential diseases include rabies, the viral encephalitides (e.g., Eastern, Western, and Venezuelan equine encephalitis), equine protozoal myeloencephalitis, the neurologic form of equine herpesvirus-1, West Nile virus, wobbler syndrome.
Cervical vertebral compressive myelopathy, white muscle disease, hyperkalemic period paralysis, among others. Many early botulism cases are thought to be horses with esophageal obstruction (choke) or colic due to the difficulty eating/dysphagia or muscle tremors and frequent recumbency.

**Treatment and Outcomes**

After exposure to BoNT, clinical signs of botulism and death can strike swiftly. Once suspected, affected horses require immediate treatment with the botulism antitoxin. The antitoxin binds to free BoNT in the bloodstream but doesn’t bind to the toxin already bound to nerve cells. Therefore, most affected horses require hospitalization and round-the-clock nursing. Intensive supportive care involves feeding, manually expressing/evacuating the urinary bladder, assistance standing (when possible), and altering recumbent horses’ positions to minimize the development of pressure sores.

Survival rates of horses with botulism vary markedly depending on when they receive antitoxin and supportive care. Whether an affected horse (or foal) will recover usually depends on the dose of toxin the animal was exposed to and if the animal remains standing. In a recent study, the overall survival rate for adult horses in a hospital setting was 48%, but survival rates as meager as 20-30% have also been reported. Overall, horses that remain standing have better outcomes than recumbent horses.

After antitoxin administration, horses often regain the ability to swallow within seven to 10 days. A full recovery for surviving horses typically occurs in about one month, but even with aggressive treatment, some horses take 30-90 days. Generally, treated foals have a better chance of survival than adult horses. Even when mechanical ventilation is necessary during treatment, foal survival rates exceed 87%.

**Prevention**

The vaccine against *C. botulinum* type B, the most common type of botulism in horses, effectively prevents disease. This vaccine doesn’t protect horses against other toxin types, but recall that type B accounts for most botulism cases in horses. Detailed recommendations for administering the vaccine to pregnant mares and foals are available from the American Association of Equine Practitioners’ website.

Providing high-quality feeds, feeding hay off the ground, ensuring animals don’t have access contaminated feed, and removing dead animals from a horse’s environment as soon as possible can minimize botulism risk.

**References**

2. Yao G, Lam KH, Perry K, et al. Crystal structure of the receptor-binding domain of botulinum neurotoxin type HA, also known as type FA or H. Toxins (Basel) 2017;9(3).

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**Botulism Exposed**

Just one teaspoon of toxin is enough to kill 5,000 horses.

For protection against *Clostridium botulinum* type B, the #1 cause of botulism poisoning, ask your veterinarian for BotVax® B.