



BOTULISM

ANIMAL GROUP AFFECTED	TRANSMISSION	CLINICAL SIGNS	FATAL DISEASE?	TREATMENT	PREVENTION & CONTROL
Mammals and birds (mainly ruminants, equids, primates, mustelids, waterfowl).	Spore or toxin ingestion. Wound contamination.	Progressive flaccid paralysis.	Yes.	Supportive care. Anti-toxin administration.	Good husbandry practices. Toxoid vaccination not available in EU).

<p>Fact sheet compiled by Antoine Leclerc, DVM, Réserve Africaine de Sigean, France.</p> <p>Contributor Manfred Brack, formerly German Primate Center, Göttingen / Germany (Botulism in primates).</p>	<p>Last update January 2009</p>
<p>Fact sheet reviewed by Michel-Robert Popoff, DVM, Chef de laboratoire, Centre de Référence des Bactéries Anaérobies et Botulisme, Institut Pasteur, Paris, France.</p>	
<p>Susceptible animal groups Many avian and mammalian hosts, including ruminants, equids, primates (<i>Papio hamadryas</i>, <i>Hylobates lar</i>, Callitrichidae), mustelids, game birds, fowl (particularly ducks), and other birds like loons, mergansers, geese, and gulls. Fish are also sensitive. Swine are reported to be resistant, whereas carnivores are rarely infected. Incidence of the infection, which is not precisely known, is relatively low in cattle and horses, but more frequent in chickens, and high in wild waterfowl.</p>	
<p>Causative organism <i>Clostridium botulinum</i> is a gram-positive, spore-forming, obligatory anaerobic rod belonging to the noninvasive clostridia. It produces seven protein neurotoxins (A to G) that have a similar pharmacologic action but differ in potency, antigenic properties, and distribution. Any of the neurotoxins can cause a neuromuscular intoxication, although type C is the most frequent in animals, notably in non-human primates, wild ducks, pheasants, chickens, mink, cattle and horse. Type D can be encountered in cattle (causing « lame disease », linked to phosphorus-deficient ranges, where grazing animals feed on carcasses and bones that often contain botulinum toxin), horse, and – sporadically – in dogs. Types A and E have occasionally caused botulism in minks. Type B toxin is generally associated with the « shaking foal » syndrome. Type F botulism only occurs in people, but type A, B, and E toxins also are important in human botulism.</p>	
<p>Zoonotic potential No : there is no direct transmission to humans. However, people can be indirectly infected by toxin ingestion, spore ingestion (important in human infant botulism), and wound contamination by spores from the environment.</p>	
<p>Distribution World-wide.</p>	
<p>Transmission Reservoirs of <i>Clostridium botulinum</i> are soil and aquatic sediments. Contaminated animals and plant materials can act as a vehicle of toxin-infection. Transmission occurs when spores or toxins are ingested (contaminated feed), or by wound contamination.</p>	
<p>Incubation period Varies with level of exposure : 12 hours to 10 days in cattle and horses.</p>	
<p>Clinical symptoms Progressive flaccid paralysis progressing cranially. Death occurs in 6 to 72 hours after the onset of clinical signs as a result of respiratory or cardiac paralysis. Motor paralysis can cause drowning of waterfowl. Other clinical signs are also caused by muscle paralysis and include disturbed vision, difficulty in chewing and swallowing, and generalized progressive weakness. <u>Ruminants</u> : drooling, inability to urinate, dysphagia, protrusion of the tongue, sternal then lateral recumbency. <u>Equids</u> : dysphagia, protrusion of the tongue, <u>Primates</u> : sudden death or lethargy with mild ataxia of the hindquarters progressing to severe ataxia,</p>	



drowsiness, flaccid paralysis, respiratory distress, collapse, and death.

Mustelidae : sudden death, dyspnea, flaccid paralysis.

Birds : typical flaccid paralysis of the neck, but also of the legs and wings (inability to fly). In waterfowl, neck paralysis can lead to drowning.

Post mortem findings

No characteristic lesions. Pulmonary edema and congestion can be noticed, as well as excessive pericardial fluid containing free-floating fibrin. Congestion of some intestinal loops can also be observed.

Diagnosis

A presumptive diagnosis is often made based on clinical signs and exclusion of other causes of motor paralysis. Demonstration of toxin in feedstuffs, fresh stomach contents, or vomitus supports the diagnosis.

Confirmation is based on toxin identification in serum, intestinal content, or tissue before death or from a fresh carcass. Isolation of the organism, especially from intestinal contents, and postmortem demonstration of toxin are not definitive.

Toxin is extracted from suspected material. Samples are homogenized in an appropriate buffer, centrifuged, and injected intraperitoneally to susceptible laboratory animals (mice). Clinical symptoms and death depend on the amount of toxin injected and generally occur in 3 hours to 3 days. *Clostridium botulinum* can be detected in enrichment cultures from suspected samples by toxin production and PCR analysis targeted on neurotoxin genes. Isolation of *Clostridium botulinum* on agar plates and further characterization require strict anaerobic conditions and are performed in specialized laboratories.

Material required for laboratory analysis

Serum, intestinal content or tissue samples.

Relevant diagnostic laboratories

Centre de Référence des Bactéries Anaérobies et Botulisme
Institut Pasteur
25-28 rue du Dr Roux 75724 Paris Cedex 15.
Tel : 00 33 1 45 68 80 00
<http://www.pasteur.fr>

Local medical laboratories.

Treatment

In case of suspected recent toxin ingestion, evacuation of the stomach and purging are helpful.

Clinical cases can be managed with supportive care and administration of specific anti-toxin. However anti-toxin treatment response and prognosis vary considerably with the type of toxin and the species of the host (often successful for mink and ducks). Some affected birds may also recover without treatment.

Antibiotics should be used in case of suspected aspiration pneumonia but are not reported to be effective against toxicoinfectious or wound botulism. *Clostridium* spp. are generally resistant to aminoglycosids and penicillins (ampicillin *per os*) should be preferred to attempt an antimicrobial therapy.

Guanidine hydrochloride (11 mg/kg body weight) and aminopyridine stimulate acetylcholine release and have some therapeutic efficacy against type A botulism, whereas germine intensifies neural impulses. Nonetheless, the use of these molecules has been reported only a few times and not extended enough to determine their value.

Prevention and control in zoos

Good husbandry practices (keeping vermin populations low, buying feed from reputable sources). Primates should not be kept in enclosures previously used for ungulates.

Removal of affected waterfowl to dry land.

Quick disposal of carcasses.

Removal of decaying grass or spoiled silage from the diet.

Immunization with toxoid vaccines (not available in Europe). Types A, B, C and D vaccines were developed for mink, types C and D toxoids were successfully used in cattle in South Africa and Australia while type B is available for horses in North America, and the type C mink vaccine was successfully used in waterfowl.

Suggested disinfectant for housing facilities

Chlorhydric acid, formaldehyde, glutaraldehyde, calcium hypochloride, sodium hypochloride, ethylene oxide, and hydrogen peroxide have a sporicidal activity depending on their concentration and duration of action.

Poultry litter should be treated (although not always effective) by sodium bisulfate at 1kg/200 square meters.

Notification

Guarantees required under EU Legislation

Guarantees required by EAZA Zoos



Measures required under the Animal Disease Surveillance Plan
Measures required for introducing animals from non-approved sources
Measures to be taken in case of disease outbreak or positive laboratory findings
Conditions for restoring disease-free status after an outbreak
Experts who may be consulted Michel-Robert Popoff, Chef de laboratoire, Centre de Référence des Bactéries Anaérobies et Botulisme Institut Pasteur 25-28 rue du Dr Roux 75724 Paris Cedex 15 FRANCE Tel : 00 33 1 45 68 83 07 mpopoff@pasteur.fr
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