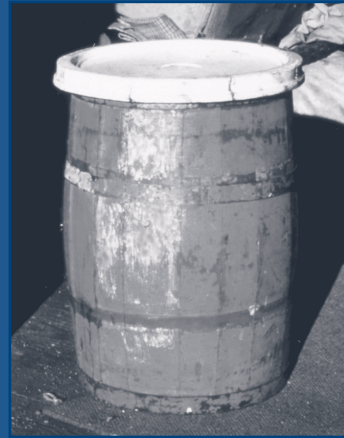


# Botulism

in *Alaska*

A guide for physicians and healthcare providers  
*2005 update*



State of Alaska  
Department of Health & Social Services  
Division of Public Health  
Section of Epidemiology

# **Botulism in Alaska**

## **A guide for physicians and healthcare providers**

### **2005 update**



Frank H. Murkowski, *Governor*  
*State of Alaska*

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*Department of Health & Social Services*

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## 2005 Update:

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## 1993 Edition:

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# Introduction

The Alaska Division of Public Health, the Arctic Investigations Program of the U.S. Centers for Disease Control and Prevention and the Alaska Area Native Health Service of the U.S. Indian Health Service first produced this monograph in 1993 to give Alaska healthcare providers an up-to-date summary of botulism in Alaska. In 1998 and now in 2005, Alaska Division of Public Health staff updated the original monograph.

Botulism can result from several different circumstances, the most common of which is from consumption of preformed toxin in food. Cases of foodborne botulism in the United States have been associated with consumption of home-canned products, rarely with consumption of commercially available products, and in Alaska, with consumption of Alaska Native traditional foods.

Other potential routes of exposure to botulinum toxin include: through wounds, as previously documented among injection drug users; or the as-yet undocumented exposure to toxin as a result of a bioterror attack. Although all routes of intoxication will be mentioned briefly, this monograph will focus on the epidemiology of Alaska botulism cases, which have all occurred among Alaska Natives who had a history of consuming traditional foods.

Information was derived from the references listed on pages 22 to 23, and from surveillance data collected by the Alaska Division of Public Health.

The 2005 update includes the following changes:

1. Clarification of the use of the botulism diagnostic pentad (see page 7–8).
2. Changes to the protocol for administration of botulism antitoxin (see page 10 and inside back cover).
3. Epidemiologic data for foodborne botulism cases in Alaska from 1998 to 2004 (see page 12–16).
4. Steps for safe food preparation (see page 17).
5. Details on treatment of infant botulism with botulism immune globulin. (see page 20).
6. Information about the role of botulinum toxin in wounds and as a possible bioterror agent (see page 21).

Botulism is relatively uncommon and healthcare providers unfamiliar with its epidemiology and presentation in Alaska may not consider botulism in their differential diagnoses. It is critical that healthcare providers in Alaska are able to accurately diagnose botulism for several important reasons:

1. Botulism is a life-threatening disease.
2. Early administration of antitoxin appears to be beneficial, especially with type E botulism, the most common type in Alaska.
3. Botulism is a public health emergency. The occurrence of a single case implies that other persons may also be at risk.
4. Current methods for detecting botulinum toxin in clinical specimens or food samples require at least 3 to 5 days. Therefore, early intervention and epidemiologic investigation depends upon accurate and rapid clinical assessment.

After reading this monograph, a healthcare provider should have an understanding of the following:

1. The signs and symptoms of botulism.
2. The importance of rapid diagnosis, evaluation and treatment.
3. The types of foods that have been associated with botulism in Alaska.
4. The need for immediate reporting of suspect cases to the Alaska Division of Public Health, Section of Epidemiology.

**Report cases of botulism  
to the Alaska Section of  
Epidemiology:**

**(907) 269-8000**

**(800) 478-0084 after-hours**

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# General Review

## Early descriptions

Botulism, or sausage poisoning as it was originally termed, was first seriously studied following an outbreak in Wildbad, Germany, in 1793. The outbreak involved 13 people, six of whom died, and was associated with consumption of a locally produced blood sausage. Following this outbreak, the number of reported cases of sausage poisoning rapidly increased, prompting a study of the disease by the local health officer, Justinius Kerner (1829). He described 230 cases, most of which were attributed to the consumption of sausage. The illness became known as “botulism” after “botulus,” the Latin word for sausage.

Many years later, Van Ermengem (1897) investigated an outbreak of botulism involving 34 individuals who had consumed raw, salted ham served at a gathering of amateur musicians in Ellezelles, Belgium. In the investigation of this outbreak, Van Ermengem established that botulism was an intoxication, not an infection, and that the toxin was produced by a spore-forming obligate anaerobic bacterium, *Clostridium botulinum*. He also found that toxin was rapidly inactivated by heating and was only toxic to certain animal species.

A later outbreak in Darmstadt, Germany, associated with canned white beans established that there was a second type of botulism. The new strain was type A and the Van Ermengem strain was probably type B (Landmann 1904).

In 1922, type C botulism was identified as causing disease in chickens (Bengston 1922) and cattle (Seddon 1922). Robinson (1929) identified type D in cattle and type E was identified by Gunnison (1936–1937) as causing botulism in people after consuming fish. Type F was first described by Moller and Scheibel (1960) from a Danish outbreak of botulism involving homemade liver paste. Finally, type G botulism was identified in soil from cornfields in Argentina by Gimenez and Ciccarelli (1978). The principal types of botulism involved in human disease are types A, B, and E.

The toxin of *C. botulinum* acts at cholinergic neuromuscular junctions by blocking the release of acetylcholine. The action only affects peripheral sites and is believed to be irreversible. Both autonomic and voluntary motor activities are affected and molecular

differences in toxin types may result in somewhat different signs and symptoms for the three toxin types responsible for most human disease.

## Foodborne botulism in the Arctic

### a. Epidemiology

For many years, outbreaks of illness associated with traditionally prepared and preserved food have been described. Early explorers reported clusters of deaths in villages among groups of northern Natives that the explorers attributed to “ptomaine” poisoning or trichinosis (Stefansson 1914). However, descriptions of many of these outbreaks resemble foodborne botulism. Later, ethnographers described food preparation and storage practices that could support the production of botulinum toxin (Nelson 1971).

The first reported outbreaks of foodborne botulism in the Arctic occurred in the early 1900s and over 200 outbreaks have been recorded since. In Canada, the first reported outbreak was in 1919 and since then, over 100 outbreaks involving over 230 individuals have occurred (Dolman 1960 and 1974). The first reported outbreaks in Greenland (Muller and Thomsen 1968) occurred in 1967 and over 20 additional outbreaks have been reported since. Rabeau (1959) recorded the first Alaska outbreak that involved beluga whale flipper consumed in the village of Kotzebue and occurred in 1947. Outbreaks among Siberian Eskimos in Russia are not well reported.

The overall case fatality rate in past arctic outbreaks was about 20%. Because not all northern Native groups consume the same traditionally prepared foods, it is difficult to determine true incidence rates of disease. However, using total population as the denominator, Canadian Inuit and Alaska Native residents had annual incidence rates of 30 cases per 100,000 (Smith 1977) and 8.5 cases per 100,000 (MacDonald et al. 1986), respectively. These rates compare to 0.43 cases per 100,000 in Washington state, the highest rate in the United States other than Alaska (MacDonald et al. 1986). Most foodborne botulism in the Arctic is type E.

### b. The foods, their preparation and storage

There is an important difference between foodborne botulism in Alaska compared with the rest of the United States (CDC 1998). All cases in Alaska have been associated with the consumption of traditional Alaska

Native foods. These include fermented foods, dried foods and traditionally prepared condiments, such as seal oil. In other parts of the United States, foodborne botulism is usually associated with improperly canned foods or with improperly stored unrefrigerated foods.

Foods involved in Native botulism outbreaks are usually putrefied (fermented). These foods are either intentionally subjected to putrefaction or are unintentionally putrefied due to inadequate storage and preservation. In either case, most outbreaks involve traditional foods, such as sea mammals or fish. Whale and seal are the most frequently involved sea mammals. Salmon, including salmon eggs, is the most frequently involved fish. Land mammals (beaver and caribou) contribute to a small proportion of outbreaks.

Conditions allowing inadvertent putrefaction of food were described by the ethnographer Nelson (1971) when he observed:

*Meat is frequently kept for a considerable length of time and sometimes until it becomes semiputrid. At Point Barrow, in the middle of August 1881, the people still had the carcasses of deer which had been killed the preceding winter and spring. This meat was kept in small underground pits, which the frozen subsoil rendered cold, but not cold enough to prevent the bluish fungus growth which completely covered the carcasses of the animals and the walls of the storerooms.*

Similar conditions and practices are still found in many areas of the Arctic. In Canada, First Nations persons living outside the Arctic, e.g., British Columbia, also ferment traditional foods and cases of botulism are well documented (Dawar et al. 2002).

Dried foods, particularly dried fish, have also been implicated in foodborne botulism outbreaks. Fish are dried either with or without a brine stage. However, even if fish are put in brine prior to drying, the salt concentration is rarely high enough to inhibit botulinum toxin formation (Zottola and Zoltai 1981).

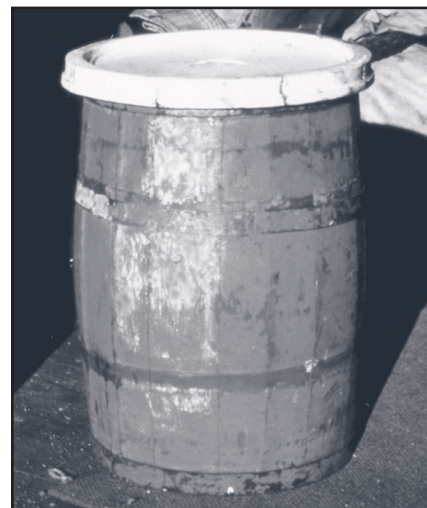
In addition to inadvertent spoilage, many traditional methods of food preparation lend themselves to botulinum toxin formation. Traditional “stink” foods such as fermented salmon eggs (stink eggs) or salmon heads (stink heads) are prepared by burial in moss-lined

pits or barrels in the ground. Nelson (1971) described the process he observed during a visit to the coastal villages of northwest Alaska in 1878–1881:

*In the district between the Yukon and Kuskokwim, the heads of king salmon, taken in the summer, are placed in small pits in the ground surrounded by straw and covered with turf. They are kept there during the summer and in the autumn have decayed until even the bones have become the same consistency as the general mass. They are taken out and kneaded in a wooden tray until they form a pasty compound and are eaten as a favorite dish by some of the people.*

The process described by Nelson has changed somewhat. Now, fermentation is usually carried out in either a barrel, a plastic or glass jar, or a plastic bag. These containers may increase the risk of botulinum toxin formation because most can be easily sealed, thereby increasing the likelihood of anaerobic conditions. Some foods are fermented in a seal skin or fish skin bag or “poke,” which is either buried or hung up. If salmon eggs are fermented in this manner, they can be left until they dry out somewhat and form a “cheese” that is firm on the outside and soft in the center.

Toxin production is also temperature dependent, and is less likely to occur at the lower temperatures that were usually attained during traditional fermentation. Fermentation now may be done indoors, or in a container above ground and in the sun, which produces warmer temperatures that make fermentation more rapid and production of botulinum toxin more likely.



A wooden barrel with a plastic lid that was used to ferment salmon heads.

# Clinical Considerations

## Initial presentation and evaluation

Because laboratory testing for botulinum toxin takes at least several days, the initial diagnosis depends on accurate and rapid clinical assessment. A careful history often reveals recent consumption of traditional Alaska Native foods, particularly fermented foods. The incubation period, or interval for consumption of contaminated food to illness onset, varies but is usually 12 to 36 hours. Severely affected patients may have a more rapid onset (as short as 6 hours) and although unusual, incubation periods as long as 10 days have been described.

The salient clinical features of botulism can be grouped into three major areas: gastrointestinal/urinary, neurologic and muscular (Table 1).

**Table 1. Signs and symptoms of botulism.**

### Gastrointestinal / Urinary

- Nausea
- Vomiting
- Diarrhea
- Abdominal pain
- Intestinal ileus
- Urinary retention

### Neurologic

- Dry mouth
- Blurry vision
- Diplopia
- Dilated or unreactive pupils
- Dysphagia
- Decreased gag reflex

### Muscular

- Symmetrical skeletal muscle weakness
- Respiratory muscle paralysis
- Fatigue
- Dyspnea (without typical signs, such as gasping)

## a. Gastrointestinal / Urinary

Gastrointestinal symptoms are usually the initial manifestations of botulism; however, they lack diagnostic specificity unless associated with other findings. Nausea, vomiting, diarrhea and abdominal pain may be present initially or appear within 2–3 days

of illness onset. The origin of these symptoms is not completely clear, but may be secondary to toxin induced intestinal ileus. Ileus is sometimes severe and relatively long lasting (i.e., more than a week). Urinary retention, presumably caused by detrusor weakness, is often present; however early in the course of the illness, it is frequently asymptomatic.

## b. Neurologic

When the effects of the cholinergic blockade are observed, the diagnosis of botulism must be seriously considered, especially in an Alaska Native patient with gastrointestinal symptoms. Dryness of oral mucous membranes may be extreme and can lead to fissuring of the tongue and severe pharyngeal pain. The pharyngeal presentation of botulism has been confused with diphtheria. Ocular findings are classic: diplopia, blurry vision, fixed or dilated pupils. Ptosis is commonly present. The absence of ocular findings does not rule out the diagnosis of botulism. However, the absence of any objective signs of cranial nerve weakness makes botulism extremely unlikely. The progressive paralysis typically descends, affecting the cranial nerves first, then the neck, upper arms, trunk and diaphragm, and finally the hands and legs. The fingers may be the last to be affected.

## c. Muscular

Skeletal muscle weakness, manifested by fatigue, shoulder, neck or truncal weakness, or dyspnea, is ominous. Because the muscles of respiration are weakened, typical signs of dyspnea such as gasping, vigorous chest motions or use of accessory muscles of respiration are usually absent. Precipitous deterioration of respiratory reserve with concomitant respiratory arrest has caused almost all of the early deaths from botulism and is not necessarily preceded by other complaints. Often a patient's paralysis prevents demonstration of agitation or restlessness, so they may appear to be resting comfortably. It is imperative that respiratory reserve be assessed and followed diligently. Measurement of forced vital capacity (FVC) should be sufficient to indicate the degree of respiratory compromise and is a convenient index to follow for signs of deterioration.

Assessing changes to FVC is an objective method of documenting diminishing respiratory capacity and muscular weakness. Other quick and more subjective



tests can also provide evidence of muscular impairment. For example, serial counts of how many times a patient can successively stand up and sit down, or the number of stairs they can walk up and down, before becoming fatigued can provide evidence of progression of muscular weakness. This information can be used together with other signs and symptoms to support a diagnosis of botulism.

Knowledge of findings that should be normal in botulism may be helpful in establishing or ruling out the diagnosis. Body temperature, orientation to person, place and time, sensory examination and deep tendon reflexes (if the patient is not completely paralyzed) should all be normal. Rare exceptions have occurred. Even if signs or symptoms not usually associated with botulism are present, clinicians may still need to consider botulism in the differential diagnosis, especially if other findings are suggestive.

The differential diagnosis of botulism (Table 2) generally involves consideration of rare conditions or unusual presentations of common problems, such as stroke. It is often best to pursue a diagnosis of botulism, perhaps in parallel with others, until the diagnosis is clear, particularly if the patient is an Alaska Native who has consumed fermented food during the week before onset of symptoms.

Laboratory data from electromyography, nerve conduction studies, cerebrospinal fluid analysis, or Tensilon® testing are more helpful for diagnosing other conditions than for establishing the diagnosis of botulism. Occasionally an electromyogram will show convincing post-tetanic potentiation, which is almost specific for botulism. Cerebrospinal fluid and nerve conduction studies should be normal in patients with botulism.

Past reports suggested that if a patient has three or more signs or symptoms in a “diagnostic pentad” and a history of consuming traditional Alaska Native food, botulism should be strongly suspected (Table 3) (Eisenberg and Bender 1976; Wainwright et al. 1988). The term “diagnostic pentad,” however, can be misleading because when pentad symptoms are present, they are *suggestive*, but not necessarily diagnostic, of botulism.

Also listed in Table 3 is a botulism “clinical paradigm” that focuses on body systems (i.e., gastrointestinal,

<b>Condition</b>	<b>Points of Differentiation from Botulism</b>
<b>Diphtheria</b>	<ul style="list-style-type: none"> <li>• Fever</li> <li>• Typical pharyngeal or nasal mucosal lesions</li> <li>• Culture</li> <li>• Cervical adenopathy</li> <li>• Cardiac conduction abnormalities</li> </ul>
<b>Drug ingestion / Poisoning</b>	<ul style="list-style-type: none"> <li>• History</li> <li>• Central nervous system abnormalities</li> <li>• Drug levels</li> </ul>
<b>Gastroenteritis</b>	<ul style="list-style-type: none"> <li>• Lack of autonomic, ocular or muscular findings</li> </ul>
<b>Guillain-Barré syndrome</b>	<ul style="list-style-type: none"> <li>• Cerebrospinal fluid protein elevated</li> <li>• Abnormal nerve conduction</li> <li>• Absent deep tendon reflexes</li> <li>• Sensory findings</li> </ul>
<b>Myasthenia gravis</b>	<ul style="list-style-type: none"> <li>• Response to Tensilon® testing</li> </ul>
<b>Paralytic shellfish poisoning</b>	<ul style="list-style-type: none"> <li>• History</li> <li>• Presence of sensory findings</li> </ul>
<b>Poliomyelitis</b>	<ul style="list-style-type: none"> <li>• Abnormal cerebrospinal fluid</li> <li>• Muscle denervation findings</li> </ul>
<b>Stroke</b>	<ul style="list-style-type: none"> <li>• Asymmetric findings</li> <li>• Absence of gastrointestinal and autonomic findings</li> </ul>

neurologic, muscular) and may be a more useful tool in assessing suspected cases of botulism compared with the pentad. Similarly, the pentad may be more meaningful when signs or symptoms are considered with respect to body systems, e.g., dry mouth from cholinergic blockade, as opposed to resulting from repeated vomiting and subsequent dehydration.

Neither of these approaches have been rigorously tested, but both have been found useful by clinicians experienced in the diagnosis of botulism in Alaska.

Either approach may help trigger suspicion of botulism. Regardless of approach, all relevant clinical and exposure information should be considered when assessing whether a patient might have an illness compatible with a diagnosis of botulism.

**Table 3. Signs and symptoms profiles suggestive of botulism.**

Botulism should be considered in any patient having a history of consumption of traditional Alaska Native food with either of the following symptom profiles:

**At least three of the five following signs or symptoms of a botulism diagnostic pentad:**

- Nausea or vomiting
- Dysphagia
- Diplopia
- Dilated or fixed pupils
- Dry throat

**Any of the following elements of a botulism clinical paradigm:**

- Gastrointestinal symptoms with autonomic or neurologic abnormality<sup>1</sup>
- Descending symmetrical paralysis or weakness with no apparent cause
- Cranial nerve deficit with no apparent cause<sup>2</sup>

<sup>1</sup>Autonomic involvement includes evidence of hypotension

<sup>2</sup>Cranial nerve deficits include lack of a gag reflex, nonreactive pupils or ptosis.

## Laboratory evaluation

Since the laboratory bioassay for botulinum toxin usually takes at least 72 hours, the results are not useful for the immediate management of patients, but can be very helpful in corroborating the diagnosis. Samples of serum, vomitus or gastric aspirate, suspect food(s) and stool should be collected for analysis (Table 4). Serum should be obtained prior to the administration of antitoxin, and again at least an hour following the completion of antitoxin administration. All specimens are evaluated for presence of botulinum toxin; non-sera samples are further evaluated for presence of *Clostridium botulinum* organism by culture. Rarely has toxin or organism been detected from specimens from asymptomatic persons. Because ileus resulting from botulism intoxication slows transit of potentially infected food products, gastric aspirates and

**Table 4. Botulism specimen collection.**

**Patient specimens**

- At least 10 cc of serum collected in red top or serum separator tubes  
BEFORE administration of antitoxin  
AND  
AT LEAST 1 HOUR AFTER administration of antitoxin.  
Note: If antitoxin is not administered, send at least 10 cc of serum.
- At least 10 to 50 grams of bulk stool placed in a sterile unbreakable container.
- At least 20 ml of gastric aspirate placed in a sterile unbreakable container.

**Food specimens**

- Any food item from the suspected meal should be collected.
- Foods should be left in their original containers if possible.
- If not already in a container (e.g., dried fish), food items should be placed in separate sterile unbreakable containers.

All specimens must be carefully labeled, placed in leakproof containers or plastic bags, and shipped according to current guidelines. Contact the Section of Epidemiology with specific questions or concerns about additional types of specimens not listed here. As well, laboratory staff who package specimens should contact Epidemiology with details of shipments so packages can be tracked in transit.

stool specimens often yield positive results among persons with laboratory-confirmed botulism. However, sufficient quantities of sample must be collected, i.e., rectal swabs with minimal stool have an extremely low yield.

More detailed information regarding specimen collection and handling procedures is available in the CDC Handbook for Epidemiologists, Clinicians, and Laboratory Workers (1998).

Arrangements for testing are handled by the Alaska Division of Public Health, Section of Epidemiology. All specimens must be shipped to the Alaska State Public Health Laboratory (ASPHL) in Anchorage. The mailing address is Division of Public Health, P.O. Box

196093, Anchorage, Alaska, 99519-6093. The physical address is 4500 S. Boniface Parkway, Anchorage, Alaska. For consultation and more information about specimen collection and handling, call the Section of Epidemiology at (907) 269-8000, or after-hours (800) 478-0084.

## Hospital course and treatment

Clinical caution: All information regarding clinical course and treatment has been based on the experience of healthcare providers in Alaska treating Alaska Natives with botulism acquired from consumption of fermented traditional foods. Providers should be aware that the clinical course and recovery may be quite different for persons with botulism acquired from consumption of home-canned products in areas outside of Alaska. Convalescence after foodborne botulism can be prolonged. Few data are available on long term sequelae among botulism survivors.

The clinical courses for infant, wound or botulism possibly as a result of a bioterror attack, may also be quite different.

The most urgent clinical concern for the patient suspected of having botulism is assessment of respiratory reserve. Most patients will require frequent (at least hourly initially) determination of FVC or an equivalent measure. Any significant decline in respiratory function should prompt consideration of endotracheal intubation and assisted ventilation. For patients in an outlying hospital requiring transfer for management of respiratory insufficiency, placement of endotracheal and nasogastric tubes should be strongly considered *before* transfer.

## Reporting and outbreak response

Botulism is both a medical and public health emergency. If a healthcare provider suspects botulism, he or she should immediately notify the Alaska Division of Public Health, Section of Epidemiology so that any possible associated cases can be identified and treated. Reporting should never await laboratory confirmation. Delayed reporting may result in additional persons consuming toxin-containing food and additional cases of botulism. The Section of Epidemiology, assisted by local or regional public health nurses, environmental health

officers and community health aides, leads investigations of all botulism cases in the state (Table 5).

Possible cases should be reported immediately by telephone to the Section of Epidemiology in Anchorage at (907) 269-8000 or after-hours at (800) 478-0084. Medical epidemiologists from the Section are available 24 hours a day to provide clinical consultation and advice regarding diagnosis, specimen collection, and treatment.

**Table 5. Steps in a botulism outbreak investigation.**

1. A healthcare provider reports suspected botulism to the Section of Epidemiology.
2. After discussing the clinical presentation, if botulism is considered possible, an investigation is immediately started.
3. The Section of Epidemiology contacts the patient (or patient's family) to determine possible source(s) of exposure to botulinum toxin.
4. Other persons who ate suspect food(s) are asked their food consumption history, the method of food preparation, and recent symptoms, in order to determine the extent of the outbreak and to more precisely define a likely source.
5. Symptomatic persons are immediately evaluated at the nearest healthcare facility. Asymptomatic exposed persons are warned that they may become ill and are told to immediately seek care if symptoms of botulism develop. Community health aides and local physicians are alerted and active surveillance is maintained for 10 days, ensuring that asymptomatic persons are contacted daily so that they do not become ill without others being aware.
6. The Section of Epidemiology recommends not consuming any suspect food(s) until laboratory testing has been completed.
7. Appropriate food and clinical specimens are collected and shipped to the Alaska State Public Health Laboratory in Anchorage for testing.
8. When laboratory results are received, this information is transmitted to healthcare providers and persons in possession of suspect food(s).
9. If any part of the investigation cannot be completed quickly and reliably by telephone, a site investigation is conducted.

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## Antitoxin use and clinical management

Although the primary treatment for botulism patients is supportive care, antitoxin is available and indicated for persons suspected of having botulism intoxication. Before 1999, an equine trivalent antitoxin was used. However since 1999, botulinum antitoxin has been available in two formulations: bivalent type A and B, and type E. Antitoxin type E is still considered an investigational drug and must be administered according to specific guidelines and protocols that are included with each antitoxin kit.

Antitoxin kits are packaged by the Alaska Section of Epidemiology and supplied to 10 hospital pharmacies located throughout the state. Epidemiology has additional kits stocked in Anchorage to be sent as needed to other locations. The kits consist of one vial of each antitoxin (type AB and type E) formulation and materials needed for sensitivity testing. In the past, sensitivity testing involved instillation of drops into the eye; currently, sensitivity to antitoxin products is assessed using skin testing with dilutions of antitoxin. *See the Botulism Antitoxin Administration Flowchart located on the inside back cover.*

Before 1999, there was only one documented case of a hypersensitivity reaction following administration of antitoxin in Alaska. Serum sickness and anaphylaxis, although reported elsewhere, have not been reported in Alaska following administration of antitoxin. No adverse reactions have been reported since the change in antitoxin administration protocols in 1999.



Fermented whale skin with blubber.

Even though most botulism outbreaks in Alaska have resulted from type E intoxication, there have been outbreaks caused by type B and rarely type A. Because there are no good predictors (e.g., geographic location or food source) of toxin type prior to laboratory confirmation, persons suspected of having botulism should always receive BOTH vials of antitoxin.

Healthcare providers should carefully read through the administration guidelines to ensure that sensitivity testing is performed correctly, and that sera specimens are collected prior to administration of antitoxin and at least an hour following administration. Section of Epidemiology staff are available 24 hours a day for assistance in interpreting protocols and collecting specimens.

Botulism antitoxin acts by blocking attachment of circulating toxin to presynaptic acetylcholine release sites. At sites where toxin has already bound, antitoxin will not “neutralize” or reverse the effect of bound toxin. The effects of toxin resolve only as presynaptic end plates regenerate with time. Patients who receive antitoxin should not be expected to have immediate reversal or improvement of clinical signs and symptoms. Approximately one third of patients in one series (Barrett 1991) had continuing neurologic and muscular deterioration after receiving antitoxin. Close observation of all patients must be maintained after treatment.

Because antitoxin can stall the toxin binding process, healthcare providers should administer antitoxin immediately upon diagnosis in all but the mildest cases of foodborne botulism.

Descriptions of foodborne botulism often emphasize the long duration of toxin effect (St. Louis 1991). Alaska Natives with botulism have had a remarkably more benign course, generally with rapid and complete recovery (Barrett 1991). Most patients requiring intubation and mechanical ventilation can be successfully weaned within days. Tracheostomy should be considered rarely as the duration of paralysis is usually short. Even so, each patient’s respiratory capacity must be individually assessed. For example, in 2001, one patient required over 3 weeks of mechanical ventilation, and subsequently had a tracheostomy. This patient had a history of underlying respiratory

dysfunction, and had also experienced a case of laboratory-confirmed type E botulism in 1997.

Patients with moderate to severe symptoms are prone to develop intestinal ileus and urinary retention. Ileus is of concern because retained gastric secretions may be aspirated and decreased intestinal motility may allow continued absorption of toxin. Nasogastric tube drainage is often useful to decompress the stomach.

Controversy exists around attempting to remove potentially unabsorbed toxin from the gut (Daris 2003). However, if profound ileus exists, cathartic agents or enemas may be useful (Shapiro et al. 1998). Urinary retention is also of concern in every case of botulism and, if present, is best managed by catheterization.

Nosocomial infection complicates the recovery of many severely affected patients; fever is the cardinal sign because botulinum toxin itself does not provoke fever. Pneumonia is the most frequent complication and appears to be due to a variety of factors including reduced gag reflex, highly inspissated respiratory secretions, atelectasis associated with low tidal volumes and aspiration of pharyngeal or gastric secretions due to paralysis or weakness. Protection of the airway, high environmental humidity, adequate lung expansion and use of mucolytic agents may all help to reduce pulmonary infection. Urinary infections also occur and are probably related to catheter use.

## Recovery

It is important for healthcare providers of a completely paralyzed patient to remember that the person is fully awake. The illness, procedures and medical routines should be explained with recognition that the patient is conscious. Physicians should provide appropriate pain control and sedation for intubated patients who are otherwise alert. Patients can have excellent recall for events and conversations heard during total paralysis.

Alaska patients generally have rapid recovery of respiratory function but may have lingering ocular or intestinal symptoms. Persistent ileus has delayed oral feeding in some patients for several weeks and necessitated total parenteral nutrition. The risk of aspiration of gastric contents exists until the gag reflex has clearly returned and ileus resolved. If any concern about motility is present, it is reasonable to conduct a radiologic evaluation of swallowing prior to beginning oral feeding. Complete resolution of all effects of botulinum toxin is expected within 1 or 2 months.

Several previous botulism patients in Alaska have experienced another episode of illness following consumption of traditional Native foods at a subsequent occasion (CDC 2001). This has also been documented among persons in Canada (Proulx et al. 1997). There is no evidence to suggest that having a history of botulism intoxication mitigates the course of a subsequent illness. Based on evidence from a survey of botulism knowledge among Alaska Natives, almost half the respondents believed that there was some form of immunity to botulism (Chiou et al. 2002). Healthcare providers should ensure that current patients are aware they may contract botulism in the future if they are exposed again and should educate them about how to avoid contracting botulism.



*Beaver paws, like those pictured here, are traditional Alaska Native foods.*

# Botulism in Alaska

## Surveillance

As already mentioned, botulism is a public health emergency and healthcare providers should report all suspected cases to the Alaska Division of Public Health, Section of Epidemiology. In the past, the Alaska Area Native Health Service of the Indian Health Service, the Arctic Investigations Program of the U.S. Centers for Disease Control and Prevention (CDC) and the Alaska Division of Public Health conducted epidemiologic investigations of all patients with possible botulism. Although early records contain less detail, results of the investigations have been collected and analyzed. Currently all cases are investigated by the Alaska Section of Epidemiology.

## Cases

For this monograph, the definitions of confirmed and possible botulism explained in Table 6 were used. Botulism cases may go undiagnosed and therefore unreported if a person either does not seek medical care or the diagnosis is not considered. The botulism cases summarized here likely represent all **confirmed** cases in Alaska from 1947 through 2004.

From 1950 to 2004, 124 confirmed outbreaks of foodborne botulism involving 251 persons occurred in Alaska (there were no confirmed cases during 1947–1949) (Figure 1). Almost half (58 of 124 or 47%) of the outbreaks were associated with more than one case, and eight outbreaks were associated with five or more cases. The largest outbreak, with nine cases, occurred in 1973; the next largest, with eight cases, occurred in 2002.

All cases occurred in Alaska Natives. The average annual incidence among Alaska Natives increased from 3.5 cases per 100,000 population during 1950–1954 to a peak rate of 12.6 cases per 100,000 during 1985–1989 (Table 7). Reasons for the increase are unclear but may relate to changes in food preparation practices or improved recognition of mild cases. Since 1989, rates have slowly declined to reach 4.9 cases per 100,000 in 2000–2004. The reasons for this declining trend are also not clear.

The median age of persons at onset of foodborne botulism has not significantly changed (Table 8). Most cases were in their 40s when they became ill. In general, more than 50% of cases were between 40 and 70 years, which is not surprising as previous research showed that the proportion of persons who reported eating traditional foods increased with increasing age (Chiou et al. 2002).

There have been a total of 19 deaths for an overall case fatality rate of 8%. The case fatality rate declined from 31% during 1950–1959 to 0% for the past 10 year period, 1995–2004.

**Table 6. Definitions of confirmed and possible botulism.**

A **confirmed** case of botulism was any person in Alaska with a compatible illness having one or more of the symptoms listed in Table 1, and who met at least one of the following conditions:

1. The identification of botulinum toxin in an implicated food or in serum, stool, gastric aspirate or vomitus collected from the person.
2. The isolation of *C. botulinum* organism from the person's stool.
3. A history of eating the same incriminated food as a person meeting one of the first two conditions.

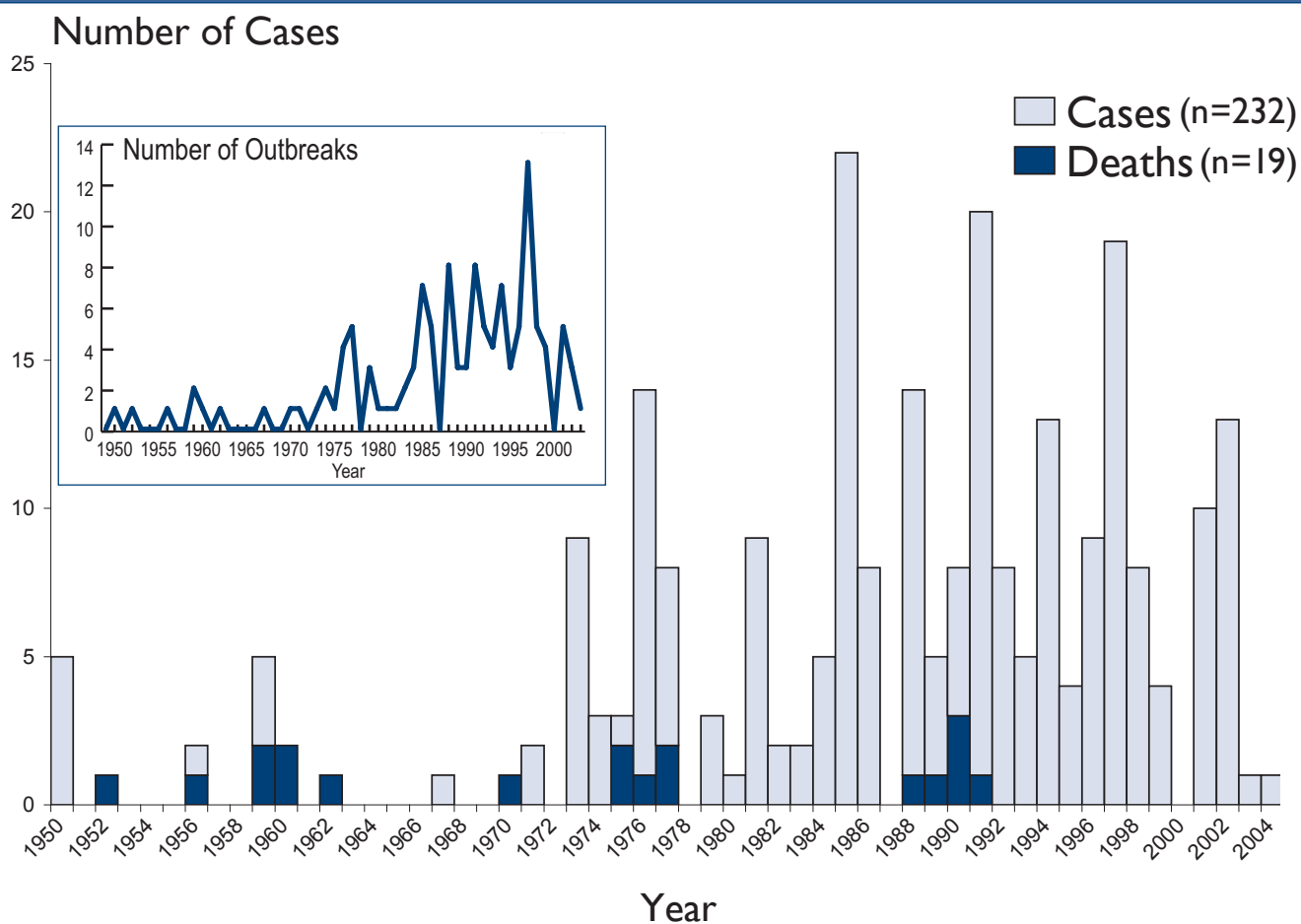
A **possible** case of botulism was a person with a compatible illness following consumption of food frequently associated with botulism, but who did not meet any of the three above conditions.

Laboratory testing of clinical specimens and food samples was conducted at the CDC, Atlanta, Georgia, using the mouse bioassay method described by the Center for Disease Control (1979).

An outbreak was the occurrence of botulism (whether confirmed or possible) among one or more persons who had eaten a common food.

Forty-six possible cases of botulism (from 27 outbreaks), which occurred between 1947 and 2004, were excluded from the analysis presented here. Information on cases from 1947-1985 can be found in a publication by Wainwright et al. (1988).

**Figure 1. Botulism outbreaks, cases and deaths, by year — Alaska, 1950 to 2004.**



**Table 7. Incidence, deaths, and case fatality rates by 5-year intervals of confirmed botulism cases — Alaska, 1950 to 2004.**

Intervals (Years)	Number	Incidence* of Cases	Number of Deaths	Case Fatality Rate
1950 – 1954	6	3.5	1	0.16
1955 – 1959	7	3.6	3	0.43
1960 – 1964	3	1.3	3	1.00
1965 – 1969	1	0.4	0	0.00
1970 – 1974	15	5.6	1	0.07
1975 – 1979	28	9.4	5	0.18
1980 – 1984	19	5.6	0	0.00
1985 – 1989	49	12.6	2	0.04
1990 – 1994	54	11.7	4	0.09
1995 – 1999	44	8.6	0	0.00
2000 – 2004	25	4.9	0	0.00
<b>1950 – 2004</b>	<b>251</b>	<b>6.1</b>	<b>19</b>	<b>0.08</b>

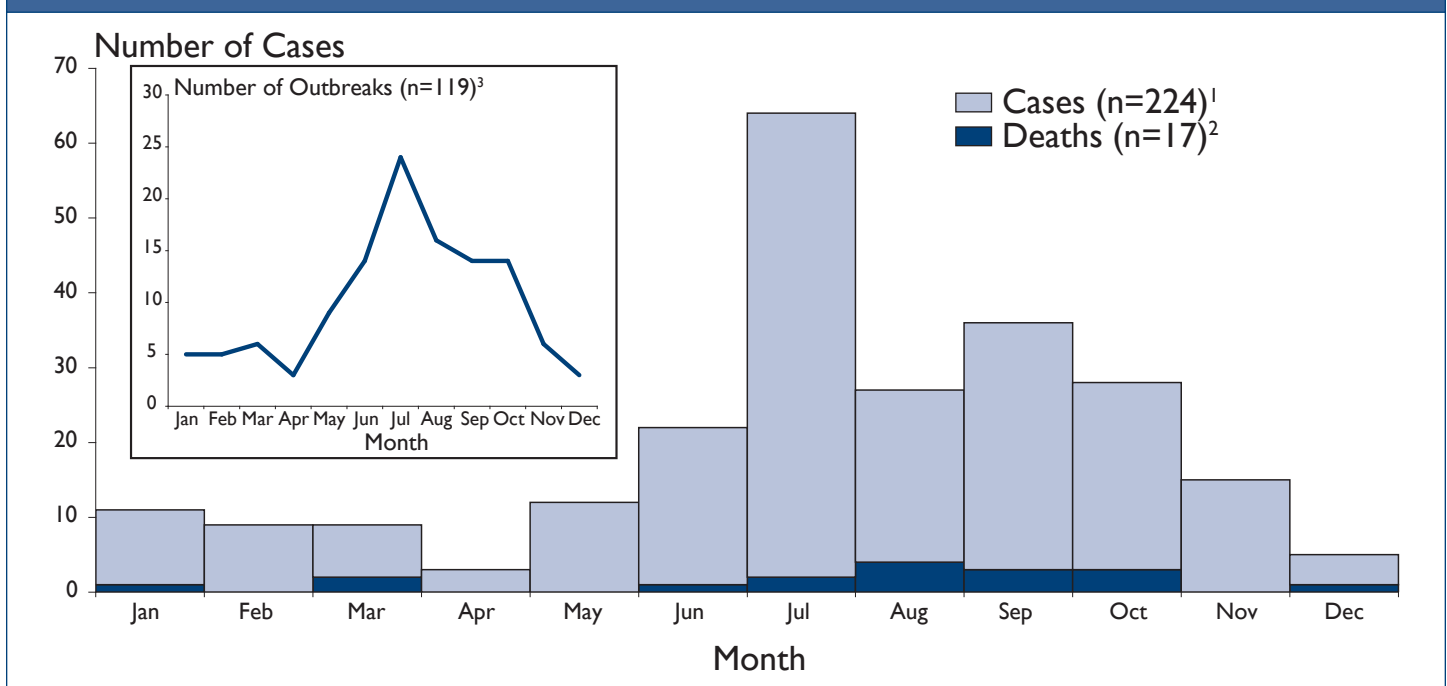
\*Annual incidence per 100,000 Alaska Natives. Mid-period population was used for each interval except 1950–2004, which was calculated by averaging the individual intervals.

**Table 8. Characteristics of age of botulism cases — Alaska, 1950 to 2004.**

	Number of cases	Median age (years)	Mean age (years)	Age range
1950-1954	6*	62	47.7	18-63
1955-1959	7	29	29.6	8-52
1960-1964	3	51	53.3	39-70
1965-1969	1	40	40	40
1970-1974	15	29	28.3	6-63
1975-1979	28	45.5	41.4	13-66
1980-1984	19	42.5	43.4	22-77
1985-1989	49	45	43.8	5-74
1990-1994	52	49	45.4	4-79
1995-1999	44	42	46.3	8-78
2000-2004	25	48	54.6	13-83

\*Age not reported for three of the cases.

**Figure 2. Botulism outbreaks, cases and deaths, by month of onset — Alaska, 1950 to 2004.**



<sup>1</sup>Data missing for 8 cases.

<sup>2</sup>Data missing for 2 deaths.

<sup>3</sup>Data missing for 5 outbreaks.



## Seasonality, types and location

Outbreaks occurred in every month of the year (Figure 2). Over half of the outbreaks (69 of 119 or 58% with a known date) occurred between June and September.

Type E botulism was by far the most frequent type, accounting for 81% (101 of 124) of the outbreaks, 81% (204 of 251) of the cases and 84% (16 of 19) of the deaths. Types A and B caused 9 and 34 cases, respectively. In 1998, both types B and E toxin were isolated from the emesis of one patient who had consumed stinkheads with a large group of persons. Three other individuals became ill in that particular outbreak but did not themselves have laboratory evidence of botulinum toxin.

Botulism occurred predominately in coastal villages in the western and southeastern parts of the state (Figure 3).

## Foods

In 56% (69 of 124) of the outbreaks corresponding to 55% (139 of 251) of the cases, an implicated food sample tested positive for botulinum toxin (Table 9). The remaining outbreaks were confirmed based on results from a patient's serum, stool or gastric contents; or of a positive culture of a patient's stool specimen for *C. botulinum* organism. When food samples did not contain botulinum toxin, the results of the investigation were used to identify the food most likely responsible for the outbreak.

**Table 9. Foods implicated in confirmed botulism outbreaks — Alaska, 1950 to 2004.**

Type of Food Implicated	Number of Outbreaks	Number of Outbreaks with Positive Food
<b>Sea mammal</b>		
Seal (including seal oil)	47	31
Whale	12	6
<b>Fish</b>		
Salmon eggs	26	15
Salmon heads	12	5
Salmon, other	2	1
Whitefish	8	4
Herring	1	0
<b>Land mammal</b>		
Beaver tail or paw	6	5
<b>Other</b>	5	2
<b>Unknown</b>	5	0
<b>Total</b>	<b>124</b>	<b>69</b>

A variety of traditional Alaska Native foods including seal, whale and fish have been implicated (Table 9). The most common method of preparation was fermentation — a process in which fresh food was allowed to putrefy for 1 to 2 weeks either in a pit in the ground or a closed or air-tight container (Table 10). No cases of foodborne botulism in Alaska have been associated with home-canned food.

**Table 10. Method of preservation for implicated foods in confirmed botulism outbreaks — Alaska, 1950 to 2004.**

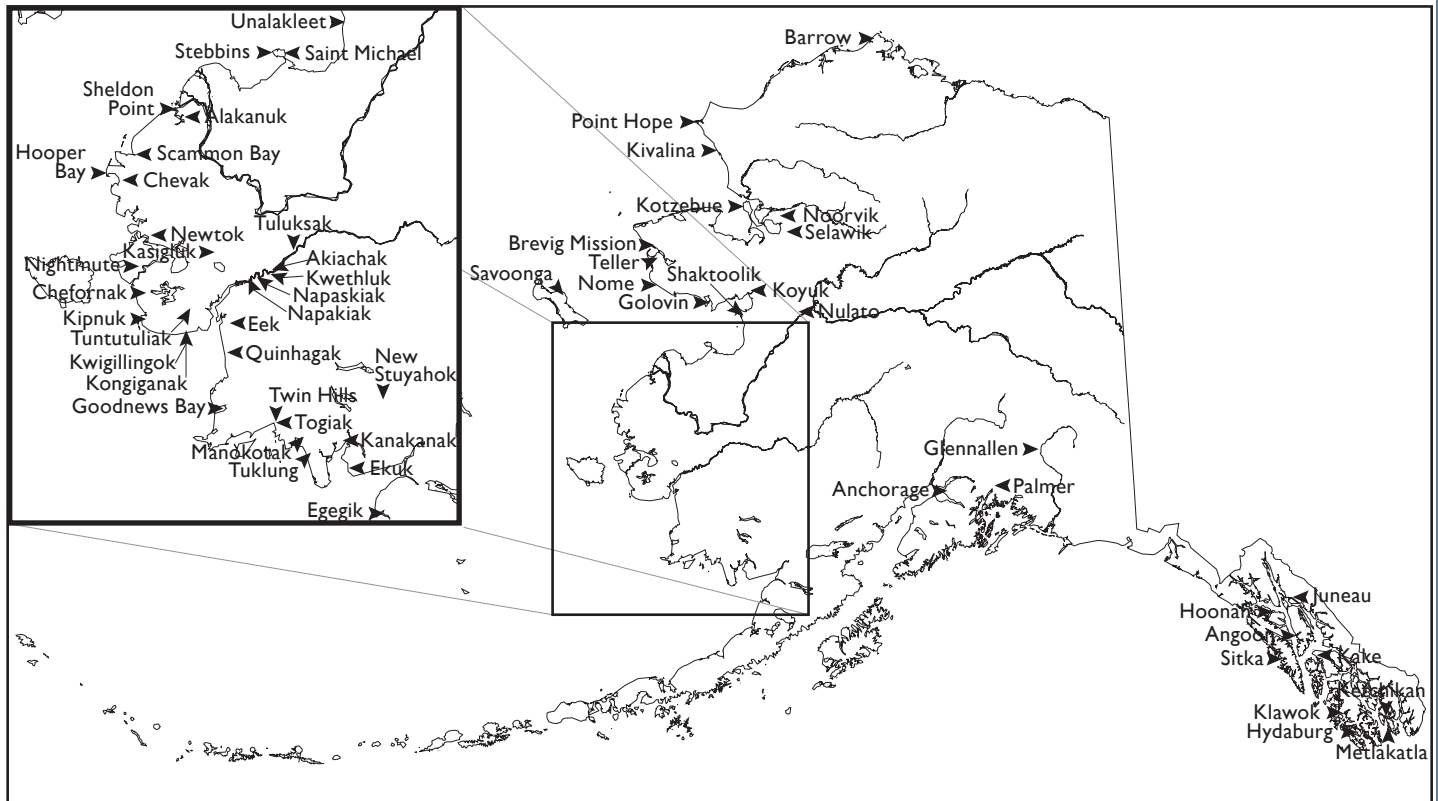
Method of Preservation	Number Implicated
Fermentation	75
Drying	7
Rendering (oil)	24
Salting	1
Other / Unknown	17
<b>Total</b>	<b>124</b>

## Prevention

Prevention and control of foodborne botulism in Alaska remains problematic. Although a multivalent toxoid, which includes type A, B and E, is available, immunization of the entire population is neither practical nor indicated. Strategies for controlling foodborne botulism fall largely into two approaches: (1) reducing contamination of food with *C. botulinum* spores and preventing toxin production in food; and (2) early identification of botulism cases. Reducing contamination and preventing toxin formation are difficult to achieve. Subsistence hunting and fishing remain an integral part of Alaska Native life and botulism spores, particularly type E, are ubiquitous in Alaska.

Shaffer et al. (1990) surveyed Alaska Native food consumption patterns and preparation practices in the Bristol Bay region in 1987. In four Yupik villages, they found that fermented foods were regularly prepared by 15% of high school students, 71% of the students' parents, and 80% of their grandparents. Fermentation practices appeared to have changed from the traditional method of using a clay pit in the ground. Only 13% of the preparers reported that they used the traditional method to ferment fish heads while 42% used a wooden barrel above ground, 38% used a wooden barrel in the ground, and 8% used a plastic bucket above ground.

**Figure 3. Location of botulism cases — Alaska, 1950 to 2004.**



Location	No. of cases	Location	No. of cases	Location	No. of cases	Location	No. of cases
Akiachak	3	Hooper Bay	2	Manokotak	9	Selawik	1
Alakanuk	3	Hydaburg	1	Metlakatla	1	Shaktoolik	1
Anchorage	6	Juneau	7	Napakiak	7	Sheldon Point <sup>2</sup>	1
Angoon	4	Kake	2	Napaskiak	2	Sitka	5
Barrow	4	Kanakanak <sup>1</sup>	8	New Stuyahok	3	Stebbins	14
Brevig Mission	9	Kasigluk	5	Newtok	1	St. Michael	2
Cheforanak	10	Ketchikan	10	Nightmute	1	Teller	5
Chevak	5	Kipnuk	3	Nome	3	Togiak	13
Eek	1	Kivalina	1	Noorvik	1	Tuklung <sup>1</sup>	1
Egegik	2	Klawok	4	Nulato	1	Tuluksak	2
Ekuk	7	Kongiganak	1	Palmer	3	Tuntutuliak	4
Glennallen	8	Kotzebue	8	Point Hope	7	Twin Hills	4
Golovin	4	Koyuk	7	Quinhagak	1	Unalakleet	1
Goodnews Bay	5	Kwethluk	3	Savoonga	3		
Hoonah	3	Kwigillingok	10	Scammon Bay	8		

<sup>1</sup>No longer incorporated.

<sup>2</sup>The community of Sheldon Point became Nunam Iqua in 2000.

**Table 11. How to protect your family from botulism: five food safety steps.**

The following five food safety steps are recommended for persons who prepare or eat fermented foods:

1. Before preparing food, wash your hands, your containers, and your food.
2. Try to use traditional methods for preparing fermented Native foods as these may decrease the presence of botulism bacteria in food. Plastic, glass, or sealed plastic bags do not allow air to reach the food and can promote the growth of *C. botulinum* bacteria. Use salt to preserve dried fish and to also discourage growth of *C. botulinum* bacteria.
3. Ferment food at a cold temperature, ideally below 37° Fahrenheit (or 2° Celsius). This will also discourage the growth of *C. botulinum* bacteria.
4. Consider boiling your food before eating it. Heat destroys botulinum toxin and may be the best way to reduce the risk of getting botulism after eating fermented foods.
5. **When in doubt, throw it out!** Don't take the risk of getting botulism if you don't know how the food was prepared. Botulinum toxin is so deadly, even a small taste can make you ill.

For more information about preventing botulism, or to order an educational video or brochure, visit the Public Health Training Network website: [www.phppo.cdc.gov/phtn/botulism/default/default.asp](http://www.phppo.cdc.gov/phtn/botulism/default/default.asp).

[Table adapted from CDC's Arctic Investigation Program Botulism website: [www.cdc.gov/ncidod/aip/research/bot.html](http://www.cdc.gov/ncidod/aip/research/bot.html).]

Another survey was performed in 1999 by the U.S. Centers for Disease Control and Prevention's Arctic Investigations Program (AIP) based in Anchorage, Alaska, to assess whether educational messages could be tailored to decrease the risk of botulism from consuming traditional foods. The survey examined the knowledge, attitudes and practices of a sample of Alaska Natives in the Bristol Bay region, and found that knowledge and awareness of botulism was relatively high. Almost half the respondents (total of 140) indicated they would consider eating traditional foods that had been boiled to reduce toxin, or consider not eating foods that had been prepared without the use of a refrigerator or by methods that allow for an anaerobic environment (Chiou et al. 2002).

The results from the 1999 survey were used to create an educational video in 2000 (Arctic Investigations Program 2000). In conjunction with the video, safe food preparation steps were developed (Table 11). This video was then distributed to all rural schools and medical facilities in Alaska in the spring of 2000. Approximately a decade earlier, a similar video was produced by the Inuvik Regional Health Board in the Northwest Territories, Canada (Inuvialuit Communications Society 1990). A follow-up survey for the Canadian video was not done.

In 2001, AIP conducted a follow-up survey to evaluate the effectiveness of the Alaska video (Dentinger et al. 2002). Approximately 40% of the 254 adults interviewed had watched the video. Most had seen the video at home or in a healthcare facility. No changes were documented between consumption and preparation practices between the pre- and post-viewing surveys. Because of the relatively small number of persons who had watched the video, assessing the effect of the video was difficult. However, the video could be a valuable education tool. It is available in English and Yupik languages. See the website in the table above for ordering information.

In contrast to the lack of success in controlling foodborne botulism by educating the preparers, educating healthcare providers to recognize botulism early in its clinical course and to report cases promptly has proved effective in limiting adverse outcomes. Educational efforts directed toward eliciting a careful food consumption history, having a high level of suspicion when confronted with an illness with gastrointestinal and neurologic symptoms, and using the "diagnostic pentad" to prompt suspicion of botulism as a possible differential diagnosis, have been the mainstay of control efforts in Alaska. These educational efforts combined with rapid epidemiologic investigation of suspected cases, prompt supportive care and the availability of botulism antitoxin may be responsible for the reduction in the number of fatalities.

Table 12. Summary of environmental testing for <i>Clostridium botulinum</i> spores in Alaska, 1975. <sup>1</sup>			
Type of Sample	Number Collected	Number Positive <sup>2</sup>	Comments
<b>Beach Soil</b>			
Ocean	88	36	Positives from Kotzebue, Kotzebue Sound, Southeast. Negatives from north coast, Aleutians, Kenai Peninsula, Prince William Sound.
River or lake	115	7	Positives from Yukon-Kuskokwim Delta, Interior, Kenai Peninsula, Prince William Sound. <sup>3</sup>
<b>Ocean</b>			
Sediment	66	32	91% of samples from Kotzebue were positive. Other positives from Kodiak and Southeast.
Water	14	8	All samples were from the Kotzebue area.
<b>Animals</b>			
Salmon	41	2	Positives were gill specimens from Kotzebue and Bethel. Negatives were flesh, viscera, and roe from Bethel.
Marine mammals	44	1	The positive was a beluga whale. Negatives were walrus, sea otter, and harbor, ringed and bearded seals. <sup>4</sup>

<sup>1</sup>All data from Miller (1975).

<sup>2</sup>Toxin detected in all positive samples was type E.

<sup>3</sup>One positive sample was from a pond approximately 50 miles south of Prudhoe Bay. Five of the remaining six positive samples were collected from areas directly associated with salmon or salmon processing.

<sup>4</sup>Animals were harvested in the Chukchi Sea, Bering Straits, and Kotzebue Sound. Colonic contents were anaerobically cultured and tested for botulism toxicity.

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## Distribution of botulism spores in the environment

*C. botulinum* spores are widely distributed in the Alaska environment. Miller, Clark, and Kunkel (1972) demonstrated type E botulism toxicity in enrichment cultures in 17 of 23 beach soil samples collected in the Kotzebue region. Other investigators (Houghtby and Kaysner 1969) detected low-level intrinsic contamination of Alaska salmon with type E spores. Among 589 pink, sockeye, chinook and chum salmon collected from Bristol Bay, Southeastern Alaska, Kodiak and the Yukon River, six (1%) had gill specimens yielding positive cultures for type E toxin. None of the 494 viscera specimens were positive.

Probably the most extensive environmental survey for *C. botulinum* in Alaska was conducted by Miller (1975). Samples of beach soil, ocean water and sediments, salmon and marine mammals were collected from 23 sites in both interior and coastal areas. As shown by the results summarized in Table 12, type E spores were widely distributed. No other type of *C. botulinum* was identified and, with one exception, no specimens from north of Point Hope were positive.

Die-offs from botulism among bird populations in the U.S. and Canada have occurred sporadically during the summer months for many years. Birds ingest botulinum toxin present in decomposing vegetation or invertebrates, which have already accumulated toxin. *C. botulinum* can be found in many different natural environments. However, the majority of birds affected by botulism are waterbirds or waterfowl. Avian botulism is usually associated with toxin type C and sometimes type E. Humans appear to be relatively resistant to type C (NWHC 2001). Since 1980, only one outbreak in 1999 has been recorded among Alaska birds (personal communication 2004, Dr. Kimberlee Beckmen, Alaska Department of Fish and Game). The die-off occurred in Haines and involved more than a dozen birds of several different species, including five trumpeter swans and two golden eyes. Blood samples from two of the swans and a goldeneye tested positive for botulinum toxin type E. No human illness was associated with this incident.

## Unanswered questions

Many questions concerning botulism in Alaska remain unanswered. Future studies may improve our understanding of botulism pathophysiology, treatment and ecology. Important concerns include the following questions:

- Although repeated episodes of botulism suggest that illness provides no or short-lived immunity against future disease, what are possible immunologic effects of ingesting small quantities of toxin over time?
- Why does the clinical course of botulism in Alaska patients appear milder than that of patients with foodborne botulism in other states?
- What is the prevalence of botulinum toxin in traditional Alaska Native foods?
- Can *C. botulinum* spores be isolated from the feces of asymptomatic persons who regularly consume traditional Alaska Native foods? What would be the implications of such a result with regard to screening and case investigation?
- What are determinants of illness severity: is it dose-dependent or influenced more by individual factors?
- Does illness severity correlate with circulating antitoxin levels found after treatment?
- As food consumption patterns and preferences change with new generations, will the demographics of botulism cases change?
- Has botulism incidence really changed over time? If so, are changes in food preparation practices responsible?
- Do culturally acceptable interventions exist that could decrease the occurrence of botulism?

Botulism remains an illness that challenges the clinician to make a diagnosis using the classic elements of medical practice — history and physical examination. Practitioners who care for Alaska Natives have a reasonably high likelihood of encountering cases of botulism. They should be alert to this possibility and act decisively if the diagnosis is entertained.

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# Infant Botulism

Infant botulism is the most commonly reported form of botulism in the United States. In Alaska, three infants have been diagnosed with this disease. In contrast to foodborne botulism where the toxin is ingested, infant botulism results from ingestion of *C. botulinum* spores with subsequent intestinal colonization and toxin production. Most infants affected by botulism are between 3 and 20 weeks of age. In the United States, infant botulism is usually toxin type A or B. There are currently other clostridial species, *C. baratti* and *butyricum*, which elaborate botulinum toxins, that are also recognized to be causative agents for infant botulism (Fox et al. 2005).

The first symptom is often constipation, followed in several days by progressive muscular weakness, poor suck, weak cry, and difficulty swallowing. Respiratory arrest occurs in half of affected infants. Numerous examples exist of infants presenting with apnea, or becoming apneic, during a diagnostic procedure.

Examination may show a decreased gag reflex; cranial nerve involvement including ptosis, ophthalmoplegia, and facial nerve palsy; mydriasis; and areflexia and generalized hypotonia. Patients are usually afebrile and have normal cerebrospinal fluid. Electromyography may be helpful in differentiating botulism from other causes of neuromuscular disease.

Diagnosis is made by demonstration of botulinum toxin in stool and supported by a positive stool culture. It is unusual to find toxin in serum.

Therapy for infant botulism is primarily supportive; mechanical ventilation can be lifesaving. Antimicrobials, particularly aminoglycosides, have been reported to increase the incidence of respiratory paralysis (L'Hommedieu et al. 1979). However, complications such as respiratory infections, may require antimicrobial therapy.

Prompt diagnosis and treatment of infant botulism with human-derived Botulism Immune Globulin Intravenous (BIG-IV) might reduce the length of time needed for recovery. BIG-IV can be obtained from the California Department of Health Services, Infant Botulism Treatment and Prevention Program (IBTPP, 510-540-2646; see also [www.infantbotulism.com](http://www.infantbotulism.com)). Use of BIG-IV, a Food and Drug Administration (FDA)-approved Investigational New Drug, requires informed parental consent and coordination with the treating hospital's institutional review board. BIG-IV should be requested from the pediatrician on-call without awaiting laboratory confirmation. BIG-IV has been shown to be most effective if given within 7 days of hospital admission (unpublished data, California IBTPP). FDA approval of BIG-IV was based upon studies that its use produced a statistically significant reduction in the durations of hospital stay, mechanical ventilation, and tube feeding (Fox et al. 2005).

Antitoxin, such as is used in cases of foodborne botulism, has not been shown to affect the outcome of infant botulism.

None of the reported cases of infant botulism in Alaska have occurred in Alaska Natives. In a study conducted outside of Alaska, affected infants had higher birthweights, their mothers tended to be Caucasian, and they were more commonly breast-fed (Spika et al. 1989).

Approximately 20% of infant botulism cases reported to the CDC have been associated with the ingestion of honey. The sources for the other cases are unknown, but hypotheses include soil, household dust, and other foods. Honey should not be fed to infants less than 1 year of age. No other specific prevention measures exist.

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# Wound Botulism

Wound botulism occurs after *Clostridium botulinum* spores have been introduced into a wound and begin to elaborate toxin which in turn causes signs and symptoms of a symmetric descending paralysis. Case reports of wound botulism were quite rare until 1982. From 1943 to 1982, 27 cases of wound botulism were reported to the U.S. Centers for Disease Control and Prevention (CDC 1982). In 1982, New York reported the first case of wound botulism associated with injection drug use. Since then, other clusters of wound botulism have been described among injection drug users, primarily in California (CDC 1995).

Among injecting drug users, *C. botulinum* spores are introduced subcutaneously either directly via

contaminated drugs or indirectly by injecting through insufficiently disinfected skin. Spores, unlike botulinum toxin, are not inactivated by heat. Therefore heating heroin mixtures does not guard against wound botulism. Wound botulism has also been documented among intranasal cocaine users, who may have wounds or skin breaks that allow for spore germination in the paranasal sinuses or nasal septums.

Wound botulism has never been documented in Alaska. Even so, healthcare providers who see patients with clinical signs, such as a descending paralysis or severe weakness and a history of injection drugs or infected wounds, should consider a diagnosis of botulism.

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# Bioterror Considerations

Botulinum toxin, produced by *C. botulinum* organisms, poses a major bioweapons threat because of its extreme potency and lethality, its ease of production, transport and misuse, and the potential need for prolonged intensive care in affected persons. A number of nations or states named by the U.S. State Department as “state sponsors of terrorism” have developed or are developing botulinum toxin as a biological weapon.

A deliberate aerosol or foodborne release of botulinum toxin could be detected by several features which include a large number of acute cases presenting all at

once; cases involving an uncommon toxin type (C, D, F, G, or non-aquatic food associated E); patients with a common geographic factor without a common dietary exposure; or multiple simultaneous outbreaks without a common source.

As for all cases of suspected botulism, healthcare providers who suspect an intentional aerosol or foodborne release of toxin as the source of a patient's symptoms should contact the Section of Epidemiology immediately. Call (907) 269-8000 Monday–Friday 8am–5pm, or after-hours (800) 478-0084.

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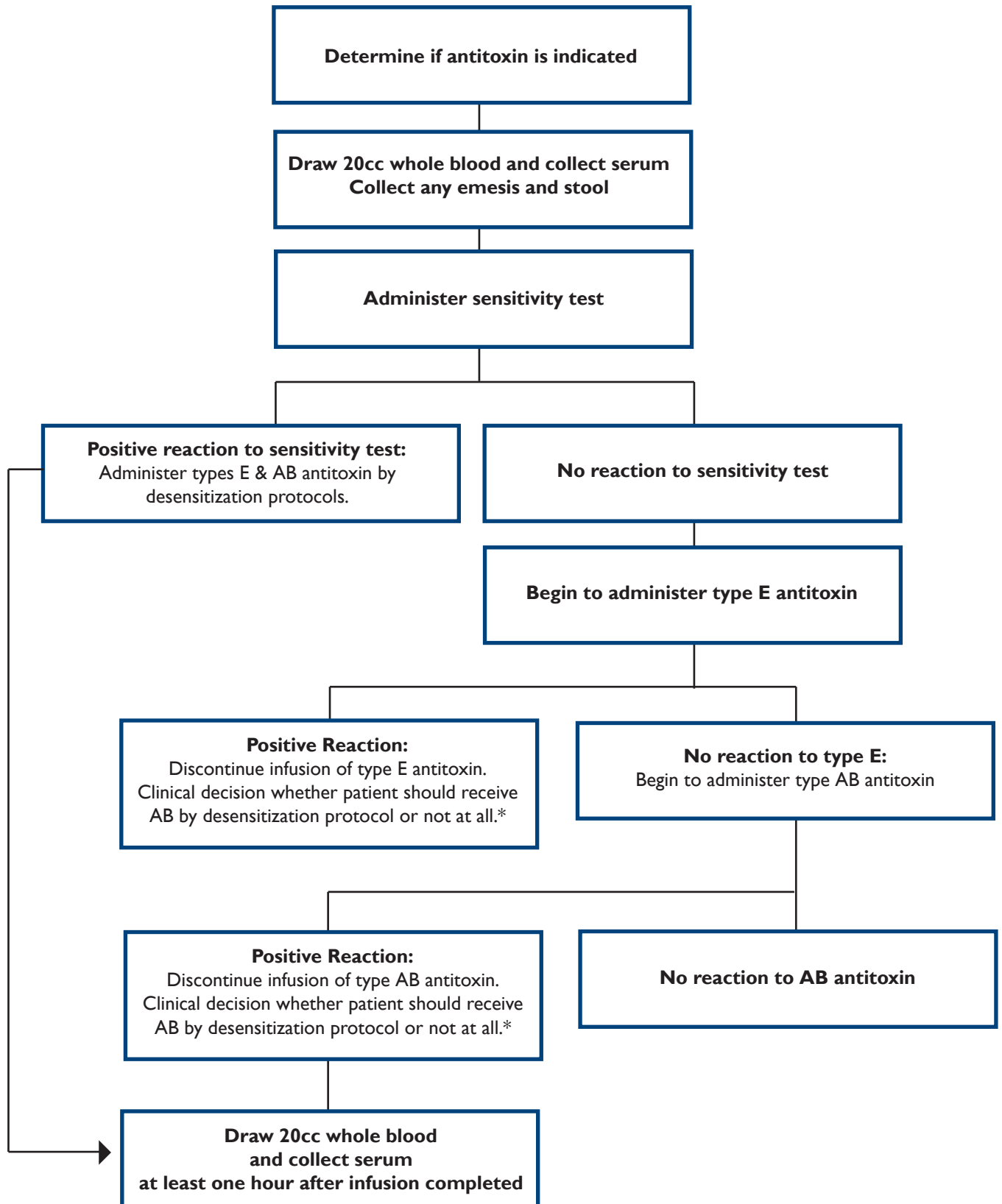
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