attributable to consumption of home-preserved fish or vegetable products such as bean curd and bamboo shoots. In parts of Europe, including Poland, France, and Germany, illness is often associated with home-preserved meat such as ham or sausage. Since 1950, commercial products have rarely been implicated in botulism in the United States, and botulism from commercial products is most often attributed to consumer error in storage or cooking. However, manufacturer deficiencies do occur. In 2007, botulism developed in eight persons in the United States who consumed a commercially canned hot-dog chili sauce. Significant deficiencies discovered by regulatory authorities involved 91 different products and resulted in the recall of 111 million cans of food.

Wound Botulism This form of disease was first recognized in 1951 as a result of a review of the clinical records on an accidental injury in 1943. Between 1943 and 2011, 491 cases of wound botulism were reported in the United States; 97% of cases reported after 1990 were associated with injection drug use. The typical patient was a 30- to 50-year-old resident of the western United States with a long history of black-tar heroin injection. In the early 2000s, wound botulism associated with drug use emerged in Europe, and at least two case clusters have been reported.

Infant Botulism More than 3900 infant botulism cases have been reported worldwide (84% in the United States alone) since this form of the disease was first recognized in 1976; ~80–100 cases (commonly caused by serotypes A and B) are reported annually in the United States.

Adult Intestinal Colonization Botulism This form of botulism is difficult to confirm because it is poorly understood and because no clear guidelines are available to help differentiate it from other adult botulism cases. Often these cases are caused by *C. baratii* type F, but the involvement of both *C. botulinum* type A and *C. butyricum* type E has been reported.

Iatrogenic Botulism Paralysis of variable severity has followed injection of licensed botulinum toxin products for treatment of conditions involving hypertonicity of large muscle groups. The U.S. Food and Drug Administration received 658 reports of adverse events related to botulinum toxin use—some very serious—between 1997 and 2006. Although some patients had symptoms consistent with botulism, no cases were laboratory confirmed. Injection of approved doses of licensed products for cosmetic purposes has not been associated with botulism. However, four cases of laboratory-confirmed botulism resulted from illegal injection of research-grade toxin for cosmetic purposes in a U.S. medical facility in 2004.

Inhalational Botulism Inhalational botulism does not occur naturally. One report from Germany has described botulism resulting from possible inhalational exposure to botulinum toxin in a laboratory incident.

Intentional Botulism Botulinum toxin has been "weaponized" by governments and terrorist organizations. An attack might use aerosolization of toxin or contamination of foods or beverages ranging in scope from small-scale tampering to contamination of a widely distributed food item. An unnatural event may be suggested by unusual relationships between patients (e.g., a visit to the same building), atypical exposure vehicles, or atypical toxin serotypes.

CLINICAL MANIFESTATIONS

The distinctive clinical syndrome of botulism consists of symmetric cranial-nerve palsies followed by bilateral descending flaccid paralysis that may progress to respiratory failure and death. The incubation period from ingestion of contaminated food to onset of symptoms in food-borne botulism is usually 8–36 h but can be as long as 10 days and is dose dependent. Incubation periods of 4–17 days have been documented in wound botulism associated with accidental injury. However, estimation is difficult in cases involving injection drug users because most of these patients inject drugs several times daily. Similarly, the incubation period for infant botulism has not been established, but the fact that the illness has affected infants <3 days old suggests that this interval may be very short.

Cranial nerve deficits may include some of the following: diplopia, dysarthria, dysphonia, ptosis, ophthalmoplegia, facial paralysis, and impaired gag reflex. Pupillary reflexes may be depressed, and fixed or dilated pupils are sometimes noted. Autonomic symptoms such as dizziness, dry mouth, and very dry, occasionally sore throat are common. Constipation due to paralytic ileus is nearly universal, and urinary retention is also common. Patients are afebrile and remain alert and oriented. Respiratory failure may occur due to either paralysis of the diaphragm and accessory breathing muscles or pharyngeal collapse secondary to cranial nerve paralysis. Weakness descends, often rapidly, from the head to involve the neck, arms, thorax, and legs; occasionally, weakness is asymmetric. Deep tendon reflexes may be normal or may progressively disappear. Paresthesias, while rare, have been reported and may represent secondary nerve compression from immobility due to paralysis. Absence of cranial nerve palsies or their onset after the appearance of other true neurologic symptoms makes botulism highly unlikely. Nausea, vomiting, and abdominal pain may precede or follow the onset of paralysis in food-borne botulism. Infants with botulism typically present with reduced ability to suck and swallow, constipation, weakened voice, ptosis, sluggish pupils, hypotonia, and floppy neck; as in adults, illness can progress to generalized flaccidity and respiratory compromise.

Even when intubated, patients can respond to questions by moving their fingers or toes unless paralysis has affected the digits. In some instances, unfortunately, the severe ptosis, expressionless face, and weak phonation of patients with botulism have been interpreted as signs of mental status changes due to alcohol intoxication, drug overdose, encephalitis, or meningitis—a conclusion that delays an accurate diagnosis. Because of skeletal muscle paralysis, patients experiencing respiratory distress may appear placid and detached even as they near respiratory arrest. Death in untreated botulism is usually due to airway obstruction from pharyngeal muscle paralysis and inadequate tidal volume resulting from paralysis of diaphragmatic and accessory respiratory muscles. Death can also result from hospital-associated infections and other sequelae of long-term paralysis, hospitalization, and mechanical ventilation.

A history of preparing home-canned foods may assist with the diagnosis. Patients with wound botulism may or may not have a discernible wound or abscess. A history of injection drug use or the presence of track marks may raise suspicion of the diagnosis. Clinical improvement follows sprouting of new nerve terminals and may take weeks to months. Patients often require outpatient rehabilitation therapy and may experience residual deficits.

DIAGNOSIS

Botulism is diagnosed primarily on clinical grounds, with laboratory confirmation by specific tests that identify botulinum toxin in clinical and food samples. In the setting of an outbreak with multiple patients presenting to the same treatment facility, the diagnosis is apparent as long as physicians recognize that cases within a cluster may have varied signs and symptoms. The temporal occurrence of two or more cases with symptoms compatible with botulism is essentially pathognomonic because other illnesses that resemble botulism do not usually occur in clusters. In lone (sporadic) cases, the diagnosis is often missed. The rarity of this disease prevents many physicians from gaining experience with its clinical diagnosis, and some patients present with signs and symptoms that do not fit the classic pattern. Assessing clinical characteristics of other paralytic illnesses in single cases is sometimes critical to rule in or rule out the diagnosis of botulism.

In adults, a food history and the names of contacts who may have shared foods should be obtained before illness progresses to respiratory failure; specific questions should include information about the consumption of home-preserved and/or exotic foods and of products requiring refrigeration that have been left at room temperature in sealed plastic containers or bags. A history of recent consumption of home-canned food substantially enhances the probability of food-borne botulism.

Ascertainment of the patient's behavioral history related to injection drug use is critical to the diagnosis of wound botulism unless an accidental wound is evident. Caretakers' observations up to and including the onset of symptoms are vital to the diagnosis of infant