Interim Guidelines for Action in the Event of a Deliberate Release: Botulism

INTERIM PHLS GUIDELINES FOR ACTION IN THE EVENT OF A DELIBERATE RELEASE

Contents:

1 Background 2
   1.1 Introduction 2
   1.2 Epidemiology 2
   1.3 Clinical features 3
   1.4 Mortality 4
   1.5 Organism survival 4
   1.6 Antimicrobial susceptibilities 4

2 Clinical procedures 5
   2.1 Diagnosis and collection of samples 5
   2.2 Treatment 6
   2.3 Infection control procedures 7
   2.4 Prophylaxis 7
   2.5 Environmental decontamination 7
   2.6 Protection of frontline workers 8
   2.7 Patient, visitor and public information 8

3 Laboratory procedures 9
   3.1 Risk assessment 9
   3.2 Isolation and identification 9
   3.3 Confirmation 9
   3.4 Waste disposal 9
   3.5 Reference laboratories 9
   3.6 Transport of samples 9

4 Public Health procedures 11
   4.1 Surveillance and detection 11
   4.2 Case definition 11
   4.3 Public health action 12
   4.4 Epidemiological investigations 13

List of national specialists 14
References 15

Note: these are interim guidelines. Comments are welcome from healthcare, laboratory and public health professionals, and should be sent to DRcomments@phls.org.uk. Since these are interim guidelines, they may be subject to changes as comments are received, so please ensure that you have the latest version, which is available through the PHLS website.
http://www.phls.org.uk/topics_az/deliberate_release/menu.htm
1 BACKGROUND
These guidelines are intended for healthcare, laboratory and public health professionals to guide clinical and public health action in the event of a deliberate release of botulinum toxin.

1.1 Introduction
Botulinum neurotoxins are produced by the anaerobic spore forming bacterium Clostridium botulinum and, rarely, by Clostridium baratii and Clostridium butyricum. There are seven neurotoxins (A-G). Illness in humans is usually caused by types A or B or E, or rarely F. Types C, D and E cause illness in mammals, birds and fish.

All toxins block the release of acetylcholine at the neuromuscular junction, which results in flaccid paralysis.

There are three naturally occurring forms of illness:
- Food-borne botulism, caused by ingestion of pre-formed toxin
- Wound botulism, caused by growth of cells and production of toxin in vivo
- Intestinal colonisation botulism, usually seen in infants, but also very rarely in adults, caused by growth of cells and production of toxin in vivo.

1.1.1 Deliberate release of botulinum toxin
A deliberate release may involve airborne dissemination of toxin, producing botulism through inhalation. Alternatively, it may involve contamination of food and water supplies either with toxin or with C. botulinum bacteria.

1.2 Epidemiology

1.2.1 Transmission
C. botulinum spores are found throughout the world in soil samples and marine sediments.

Food borne botulism is caused by ingestion of preformed toxin. A normal healthy adult can consume small numbers of spores with no ill effect, for example when consuming freshly picked raw vegetables such as mushrooms or garlic. Most cases of food borne botulism are associated with home preserved meats, fish and vegetables. Food is contaminated before preservation, and the bacteria germinate and reproduce in anaerobic conditions, producing toxin, which is then ingested. The disease is rare in the UK, but more common in the rest of Europe where the practice of home preservation is more widespread. In 1989 the largest ever outbreak of food borne botulism in the UK affected 27 people who had consumed hazelnut yoghurt. The illness was caused by type B toxin produced by bacteria growing in canned hazelnut conserve that had been inadequately heat-treated and was used to flavour the yoghurt. Since then there have only been 2 further cases in the UK, in 1998.

Water borne botulism may also be caused by ingestion of preformed toxin. This route will only pose a risk to humans in some deliberate release scenarios because the toxin is inactivated by normal treatment of mains water supplies. There have been no reported cases of illness in humans worldwide due to contaminated water supplies.

Wound botulism follows infection of wounds caused by penetrating injuries. C. botulinum spores, which are present in soil, then germinate and produce toxin in vivo.
has also been caused by injecting or sniffing drugs that are contaminated by spores. Wound botulism is now the most common form of botulism in the UK and Eire. There have been 24 clinically diagnosed cases of wound botulism between March 2000 and September 2002. All cases have been injecting drug users.

**Accidental botulism** may follow mis-injection of pharmaceutical preparations of botulinum neurotoxin. There have been no reported cases in the UK.

**Intestinal colonisation botulism** occurs in infants of less than two years of age, and most are under 6 months of age. A small number of cases in adults have been reported worldwide. Illness in infants and adults results from ingestion of C. botulinum spores in contaminated food, followed by germination and colonisation of the gut and production of toxin. The illness has been associated in infants with the ingestion of honey, and environmental sources of spores (dust, soil) have also been implicated. It is rare, with approximately 1 case in four years in the UK.

**Inhalation botulism** does not occur naturally, but may result from a deliberate release of toxin in the form of an aerosol.

1.2.2 **Incubation period**
The duration before onset of symptoms depends on the time taken for ingested toxin to reach the target site. The onset of symptoms in naturally occurring cases occurs between 2 hours and 8 days after ingestion, depending on the type and dose of toxin. Following aerosol exposure onset of symptoms may be more rapid, possibly less than one hour after exposure, but onset was 3-4 days after exposure in three cases of accidental inhalation botulism.

1.2.3 **Period of communicability**
Person to person transmission does **not** occur. Toxin can be detected in the faeces of cases, but normal infection control precautions will prevent ingestion. (see reference 8).

1.3 **Clinical features**
Clinicians should be aware of the possibility of cases of botulism.

Any previously healthy patient with the classic triad of:

- **symmetrical descending flaccid paralysis** with prominent bulbar palsies including diplopia, dysarthria, dysphonia and dysphagia
- **afebrile**
- **no change in sensory awareness**

should be immediately reported to the Consultant in Communicable Disease Control.

**Gastrointestinal symptoms** occur only in food borne and intestinal colonisation botulism. Nausea, vomiting and diarrhoea followed by constipation arise in food borne botulism, however ingestion of large amounts of toxin may lead directly to neurological symptoms, and the diagnosis should be considered in the absence of gastrointestinal symptoms. In intestinal botulism in infants, several days of profound constipation may precede neurological symptoms.

**Neurological symptoms** are the same irrespective of the route of entry of toxin, but may develop more quickly in the event of inhalation, depending on the dose. Symptoms are of a descending, symmetrical flaccid paralysis:
• Cranial nerve palsies produce diplopia, ptosis, facial weakness, dysphagia and dysarthria.
• This is followed by weakness in the neck and arms, after which respiratory muscles and muscles of the lower body are affected.
• In some cases, weakness in the neck and arms may be followed by respiratory paralysis or respiratory arrest and then by ptosis.
• In some cases, marked respiratory compromise or respiratory arrest may occur before typical oculobulbar weakness and weakness in limbs.
• There is no fever and no loss of sensory awareness.

Autonomic signs may be present, with dry mouth, fixed or dilated pupils, and cardiovascular, gastrointestinal and urinary autonomic dysfunction. Respiratory paralysis may be fatal.

Onset of gastrointestinal and neurological symptoms may be between 2 hours and 8 days. If onset is very rapid, there may be no symptoms before sudden respiratory paralysis occurs.

1.4 Mortality
Without treatment mortality can reach 100%, but this can be considerably reduced with supportive treatment and the use of antitoxin.

The lethal dose of C. botulinum toxin for an adult can be less than 1 microgram, depending on toxin type and route of administration. Exposure of rhesus monkeys to aerosols of botulinum toxin has shown that type F is about 60 times more toxic than type B and the order of toxicity is F>C>A>D>B.

1.5 Organism survival
C. botulinum is a spore-forming organism. Spores survive well in the environment, and may also survive heat and cooking. Spores do not produce toxin, but in anaerobic conditions they will germinate, and toxin is produced by the vegetative organisms. The toxin undergoes natural inactivation in surface and drinking water over several days and is destroyed by chlorine. The toxin is inactivated by heat and normal cooking but can be more stable in some foods and drinks.

1.6 Antimicrobial susceptibilities
• In cases of botulism which result from ingestion (in food or water) or inhalation of toxin, antibiotic therapy is not appropriate.
• In cases of wound infection, C. botulinum is susceptible to benzyl penicillin and metronidazole, which should be used together with surgical debridement.
• Intestinal colonisation botulism results from colonisation of the gut by vegetative cells. Risk factors are not completely understood, but changes in gut flora around the time of weaning may permit colonisation. Another factor may be decreased gut motility which results in increased anaerobicity in the gut. Antibiotic therapy is not recommended, because there is a risk of further reducing the normal gut flora and therefore increasing susceptibility to colonisation, and because lysis of vegetative cells killed by antibiotics may result in release of toxin.
2 CLINICAL PROCEDURES

2.1 Diagnosis and collection of samples

2.1.1 Misdiagnosis and differential diagnoses
Misdiagnosis of botulism is not uncommon and botulism is often low on the list of differential diagnoses at presentation in real cases of botulism. The most frequent misdiagnoses, and their distinguishing features are:

- Polyradiculoneuropathy (Guillain-Barre or Miller-Fisher syndrome): antecedent febrile illness, paresthesias, paralysis is often ascending, early loss of reflexes, increased protein in CSF (may not be seen early in illness), EMG findings.
- Myasthenia gravis: recurrent paralysis, sustained response to anti-cholinesterase therapy, EMG findings.
- Stroke: often asymmetric paralysis, abnormal CNS image.
- Intoxication (e.g. carbon monoxide, organophosphates, mushrooms): drug detected in body fluids.

Other misdiagnoses and their distinguishing features include:

- Tick paralysis: parasthesias, ascending paralysis, tick attached to skin.
- Poliomyelitis: antecedent febrile illness, asymmetric paralysis, CSF changes.
- CNS infections: changed mental status, changed CSF and EEG.
- Viral syndrome: no bulbar palsies and no flaccid paralysis.
- Psychiatric illness: EMG findings.
- Paralytic shellfish poisoning: onset of <1hr, parasthesia, food history.

Several clinical tests are useful for distinguishing botulism from other diseases, but they may give misleading results:

- Deep tendon reflexes: may be present initially in botulism but decrease or disappear over the following days.
- Tensilon tests: negative in botulism but can be transiently positive.
- EMG: can be normal early in botulism.

Confirmation of the clinical diagnosis is by identifying botulinum toxin or the organism itself in the patients’ faeces, stomach contents, or specimens from wounds, and the detection of toxin in blood samples. Routine laboratory tests are not helpful and specimens should therefore be sent immediately to the reference laboratory (see section 3.5).

2.1.2 Precautions for sampling
The samples outlined below should be taken to confirm the diagnosis. Standard Universal Precautions (gloves, gowns and hand washing) provide sufficient protection for healthcare workers attending patients and laboratory staff handling specimens. Procedures for transporting samples to the laboratory are outlined in section 3.6. The receiving laboratory should be telephoned to expect arrival.

2.1.3 Samples to be taken from acutely ill humans

- Serum. At least 10ml. Serum samples must be collected before antitoxin is administered. Do not send clotted blood to the reference laboratory.
- Faeces. At least 10g in a sterile container. Rectal washout may be required, since patients with food borne botulism may have diarrhoea in the early stages, but this is followed by constipation.
- Vomitus, gastric washings or gut contents. At least 10g in a sterile container.
• **Bronchiolar lavage** or similar in a sterile container.

• **Wound.** Pus. As much as possible in a sterile container, transferred as soon as possible to an anaerobic transport medium. If pus is not available, a swab of the lesion should be taken and put immediately into a transport medium for anaerobic culture.

• If surgical debridement is performed, **biopsy tissues**, placed immediately into a sterile container, then transferred as soon as possible to an anaerobic transport medium.

### 2.1.4. Post mortem specimens
Heart blood (10ml), if not haemolysed, should be separated into serum before dispatch to the reference laboratory (see section 3.5). Specimens of faeces, stomach contents and from infected wounds may also be useful.

### 2.1.5. Samples to be taken from the environment

• **Food samples** associated with suspect cases must be obtained as a matter of extreme urgency in order to prevent further cases. The local Consultant in Communicable Disease Control, or the CDSC on call duty doctor should be contacted to arrange collection and transport to the Reference Laboratory by Environmental Health Departments or other agencies.

• If **water** is suspected as the source of illness, the CDSC on call duty doctor should be contacted to activate the drinking water inspectorate emergency plans and arrange for testing of **water samples**.

### 2.1.6 Samples to be taken from others who have or may have been exposed
In the event of deliberate release, those who have been exposed but have not developed any symptoms within the first few hours should be identified. Instructions should be given to the exposed cohort to seek immediate medical attention should symptoms develop later.

### 2.1.7 Transport of samples
All samples must be kept **refrigerated** after collection. Procedures for the transport of specimens, both from the clinical environment to the laboratory, and from local laboratories onto the reference laboratory are outlined in section 3.6.

### 2.2 Treatment
Specialist advice should be obtained from an Infectious Disease Physician.

#### 2.2.1 Adults (food borne, wound, aerosol)
Specific treatment is with trivalent equine antitoxin (see below for source). It must be given as early as possible after a clinical diagnosis has been made, and not delayed for the results of confirmatory laboratory tests. A test dose is necessary in view of the risk of serum sickness or anaphylaxis.

Detailed instructions on administration are provided with each dose. Antitoxin is held at different sites across the UK, and must be accessed by contacting the duty doctor at CDSC (020 8200 6868; 24 hour service). Repeat doses are not necessary. Antimicrobial therapy is appropriate only in cases of wound botulism; use penicillin and metronidazole according to standard dosing regimens.

#### 2.2.2 Intestinal colonisation, (infant botulism).
Because of the risk of serum sickness or anaphylaxis, treatment with antitoxin must be considered on a case by case basis. Antitoxin is not usually recommended for treatment of
infant botulism as toxin continues to be released into the gut at a low level for some time, in contrast to food borne botulism which results from ingestion of toxin on a single occasion. **Infants recover because they grow new nerve endings.** Advice should be obtained from an expert paediatrician, ideally from a centre that has managed such cases.

2.2.3 **Antitoxin supplies**
Antitoxin is held at sites around the UK - details are in the PHLS handbook, (pages 132-135; 2000/01 edition). Details are also available from the duty doctor at CDSC.

2.3 **Infection Control Procedures**

2.3.1 **Decontamination of exposed persons**
Botulinum toxin naturally loses activity over a few days, and the toxin does not enter the body through intact skin. The risk of infection from contaminated clothing is low. However, in the event of release of large amounts of toxin, clothing and other fomites may be sufficiently contaminated to pose a risk from hand to mouth ingestion. In such situations, decontamination may require:

- Removal of contaminated clothing and possessions – these should be stored in labelled double plastic bags until they can be washed with soap and water.
- Minimal handling of clothing and fomites to avoid agitation.
- Instructing exposed persons to shower thoroughly with soap and water-appropriate facilities will be provided at the scene as necessary.
- Instructing attending personnel to wear appropriate barrier protection – Universal Precautions - when handling contaminated clothing and other fomites.

2.3.2 **Isolation of patients**
Patient to patient transmission of botulism does not occur. Patient room selection should be consistent with availability, but single room placement is not necessary. Universal Precautions are sufficient for the nursing of patients.

2.3.3 **Cleaning, disinfection & waste disposal**
Standard hospital procedures.

2.3.4 **Post-Mortem**
No specific precautions required. Universal Precautions apply

2.4 **Prophylactic treatment for people exposed to botulinum toxin**
- The use of antibiotics post-exposure is **not** indicated.
- A toxoid vaccine is available, but it has no effect in post-exposure treatment. Certain individuals who work with the organism or toxin (such as Reference Laboratory staff) can be given pre-exposure immunisation. However, following exposure, people must be observed for symptoms for a minimum period (see section 4.3.1).

2.5 **Environmental decontamination**
Following a known release, re-aerosolisation of toxin is not thought to pose a serious risk, and the toxin naturally loses activity over a few days. The contaminated area will remain out of bounds for at least this period, and subsequently environmental decontamination is
not necessary. In situations where surfaces have been grossly contaminated and cannot be avoided for these few days, they should be cleaned with a 0.5% solution (5,000ppm) of hypochlorite.

2.6 Protection of frontline workers

2.6.1 Protective clothing

The release of a botulinum toxin aerosol will create an exposed zone that presents a high risk of inhaling toxin. Anyone entering this zone should wear full protective equipment such as Type 3 high efficacy air filter masks with Class A suits, conferring full biological protection.

Healthcare workers will not normally be asked to enter this zone, but may be called into it to treat casualties, for example if an explosive device has accompanied the release of biological agent. In this case the appropriate protective clothing should be worn.

Exposed persons will normally be moved from the exposed zone, through decontamination if necessary, and into a place of safety (see section 4.3.1) for medical assessment. Frontline workers involved in decontamination, and others who have who have any contact with contaminated clothing and fomites need only observe standard Universal Precautions (gloves, gowns and hand washing) for adequate protection.

Again, for healthcare workers involved in the management of hospitalised patients with all forms of botulism Universal Precautions provide sufficient protection.

No antibiotic prophylaxis or immunisations are necessary for frontline workers.

2.7 Other Considerations – patient, visitor and public information

Fact sheets have been prepared for distribution in the event of an incident.
3 LABORATORY PROCEDURES
A key objective is to maximise the potential for laboratory confirmation of the clinical
diagnosis. Blood should be separated and sent as serum. Samples should not be tested at
the receiving laboratory but must be sent immediately to the Reference Laboratory.

3.1 Risk Assessment
C. botulinum is a Hazard Group 2 organism and normal laboratory precautions are
sufficient to provide protection. Specimens can be handled on the open bench to prepare
and package them for onward transportation to the reference laboratory.

3.2 Isolation and identification
Laboratory diagnosis is by detection and identification of neurotoxins from sera or other
samples.

3.3 Confirmation
By neutralisation and typing of toxin.

3.4 Waste disposal
Normal procedures.

3.5 Reference Laboratory
All specimens should be sent directly to the reference laboratory. The sender’s name and
address should be clearly marked. The reference laboratory should be telephoned prior to
sending to expect the sample. Samples should be forwarded urgently to:

Dr Moira Brett
Food Safety Microbiology Laboratory.
Central Public Health Laboratory
61 Colindale Avenue
London
NW9 5HT
Tel: (+44) 020 8200 4400 ext 4933/4116
E-mail: mbrett@phls.org.uk

3.6 Transportation of samples with suspicion of C. botulinum
The following procedures must be adopted for the transport of all specimens. These apply
within hospitals and laboratories as well as for specimens sent to the reference laboratory:

• Every effort should be made to avoid external contamination of specimen containers
during specimen collection.
• The primary container should be screwed tight, labelled and placed in an intact plastic
bag.
• A ‘High Risk’ label should be affixed to both specimen and request form. The latter
should include any other relevant information and include adequate clinical details to
indicate level of suspicion.
• Under no circumstances should the request form be placed in the same bag as the
specimen.
• The bag should be sealed, using tape or heat sealer. Pins, staples and metal clips
should not be used. A separate bag should be used for each specimen.
• Each specimen must then be placed in a leak-proof secondary container with sufficient
absorbent material to absorb all the contents should leakage occur.
• Each specimen must be packaged individually - ie. three specimens, three separate
packages.
• The secondary container should be externally disinfected - eg. by wiping with 0.1% hypochlorite (1,000ppm).

3.6.1 Samples sent to the reference laboratory
• Secondary containers should be placed within a final outer tertiary packaging.
• This packaging must comply to the UN 602 standard packaging for the transport of infectious substances by air, road or rail.
• The package should be certified to this standard and carry the appropriate UN certification numbers on the tertiary packaging along with the following information:
  1 BIOHAZARD – danger of infection symbol Class UN 6.2.
  2 Instructions not to open if found.
  3 Telephone number of a responsible person - eg. Consultant Microbiologist, Laboratory Manager.
• The container should be transported by an approved courier, without delay, directly to the reference laboratory.

3.6.2 Samples sent within hospitals and laboratories
• Secondary containers should be placed in a good quality box, which is well taped up and clearly labelled “Pathological Specimen – Open only in a Containment Level 2 Laboratory”.
• Specimens should be transported by hand by a responsible person using the above packaging. Vacuum-tube systems should not be used for transportation of specimens within hospitals or laboratories.
• Extra care should be taken to ensure that laboratory records are kept to a high standard.
4 PUBLIC HEALTH PROCEDURES

4.1 Surveillance and detection of deliberate releases of C. botulinum

Regardless of the circumstances, it must be remembered that a single case of botulism constitutes a public health emergency and requires immediate public health action day or night.

A deliberate release may be overt with an announcement and/or confirmation by environmental sampling. However, it is also possible that a deliberate release may be covert and will not be identified until the first cases of disease arise.

Naturally occurring botulism is very rare in the UK (see section 1.2.1); it is more common in the USA but even there the disease is not widespread. An average of 110 cases of botulism are reported each year in the USA. Of these, approximately 25% are food borne, 72% are infant botulism, and the rest are wound botulism. Outbreaks of food borne botulism involving two or more persons occur most years in the USA, and are usually caused by eating contaminated home-canned foods. The number of cases of food borne and infant botulism has changed little in recent years, but wound botulism has increased because of the use of black-tar heroin.

Deliberate release should be considered in the event of:
- Outbreaks of two or more cases of acute flaccid paralysis, especially where there are common geographic factors between cases (eg. airport, work location) but no common dietary exposures (ie. features suggestive of an aerosol attack).
- Multiple simultaneous outbreaks with no obvious common source.
- Cases of botulism with an unusual toxin type (ie, type C, D, F, or G, or type E toxin not acquired from an aquatic food).

Many animal species, including farm and domestic animals, are highly susceptible to botulinum toxin. Close coordination with veterinary colleagues is thus essential, as confirmed and suspected cases of botulism in animals may provide an early warning system. However, humans are not at risk of acquiring botulism from affected animals.

4.2 Case Definition

4.2.1 Suspected case
Clinicians should be alert to the possibility of cases of botulism. Any previously healthy patient with afebrile, descending, flaccid paralysis with an onset of 2 hours - 8 days should be immediately reported to the Consultant in Communicable Disease Control.

In the event of a suspected deliberate release of botulism, a higher index of clinical suspicion should be maintained and the diagnosis considered if the symptoms outlined below present at medical services, especially if they arise in persons who have been within or in close proximity to the exposed zone. Obviously the level of suspicion of botulism depends on clinical symptoms and the circumstances, but if a case is suspected, microbiological specimens should be sent to the reference laboratory to eliminate or confirm the diagnosis, and empirical treatment should be considered in the interim.
The symptoms of botulism are due to muscle paralysis caused by the toxin:

- Symptoms in adults include:
  - Blurred or double vision, drooping eyelids, enlarged or sluggishly reactive pupils, slurred speech, difficulty swallowing and dry mouth, loss of head control, generalised hypotonia and weakness. Loss of the protective gag reflex may necessitate intubation and mechanical ventilation.
  - Deep tendon reflexes may be present initially but diminish or disappear in the ensuing days.
  - Diarrhoea, nausea and vomiting, followed by constipation, may occur in food borne botulism.
- Infants with intestinal colonisation botulism appear lethargic, feed poorly, are constipated, and have a weak cry and poor muscle tone as well as symptoms in adults described above.
- In untreated persons, death results from airway obstruction (pharyngeal and upper airway muscle paralysis) and inadequate tidal volume (diaphragmatic and accessory respiratory muscle paralysis).
- Clinical tests that may help distinguish between other differential diagnoses include brain scan, CSF examination, nerve conduction tests (EMG), and a Tensilon test for myasthenia gravis (see section 2.1).

4.2.3 Confirmed case
A case that clinically fits the criteria for suspected botulism, and in addition the results of one or more pathological specimens fit one or more of the laboratory criteria for diagnosis (see below).

4.2.4 Laboratory criteria for diagnosis
Detection of botulinum toxin from serum or other pathological specimens. It may take 4 to 5 days for toxin to become apparent, so it takes up to 5 days for a definitive negative result. A presumptive diagnosis based on detection of toxin is followed by confirmation by neutralisation and typing.

4.3 Public Health Action

4.3.1 Procedure for handling exposed persons at the scene of an overt release
Some of them will still be at the scene when emergency services respond to the incident. This group will be decontaminated and then referred to health workers at a nearby place of safety for assessment (this is a clinical area just outside the exposed zone and within the cordon that will be established at the scene of the incident). Others will have left the scene before emergency services arrive and will be identified later when they approach GPs and A+E departments after details of the incident have been made public. Procedures need to ensure that these individuals are identified for monitoring.

4.3.2 Follow up of exposed persons
After an overt release, all exposed persons will be moved to a place of safety. It is possible that by the time this has occurred, the first casualties may be developing symptoms and will be transferred directly to hospital.
Follow up monitoring arrangements will be made for those who remain free of symptoms for the first few hours after release. Instructions will be given to seek immediate medical attention should symptoms develop later.

4.3.3 Case finding
If cases of botulism arise and a covert release is suspected, health services should be contacted to raise awareness of the possibility of further cases, and determine whether any others might already have presented.

4.3.4 Preventing secondary spread
As previously mentioned, person to person spread of botulism is negligible, and therefore there is no specific treatment or advice required for secondary contacts. There is no requirement for quarantine of infected patients.

4.4 Epidemiological investigation
In the event of strongly suspected or confirmed naturally occurring cases, the PHLS-CDSC should be notified immediately. If a deliberate release is suspected, follow the Deliberate Release Guidance.

A single case of botulism constitutes a public health emergency, and it is important to obtain a clear food history to enable identification and testing of high-risk foods. The usual response to a case normally also involves tracing others exposed to high risk foods and contacting local hospitals to determine whether there are other cases.

The principal use of epidemiological investigations in the event of a deliberate release is likely to be following the covert release of organisms into food or water supplies in order to identify and eliminate the source. Where cases arise in the absence of a known or obvious source such as a particular food item or a water quality zone, it is important to collect comprehensive details relating to diet and use of water supplies.

4.4.1 Epidemiological sampling
Where a food or water borne source of botulinum toxin is suspected, relevant samples are essential to aid identification of the source of infection.
LIST OF NATIONAL SPECIALISTS

Laboratory diagnosis and treatment
Advice can be obtained from:

- Dr Moira Brett
  Food Safety Microbiology Laboratory,
  Central Public Health Laboratory,
  61 Colindale Avenue,
  London.
  NW9 5HT
  Tel: (+44) 020 8200 4400 ext 4933/4116
  E-mail: mbrett@phls.org.uk

and

- Dr Nigel F Lightfoot
  Group Director
  Public Health Laboratory Services (North)
  Directorate Office, E Floor, Milburn House
  Dean Street
  Newcastle upon Tyne
  NE1 1LF
  Tel: (+44) 0191 261 2577
  Fax: (+44) 0191 261 2578
  E-mail: grpnligh@north.phls.nhs.uk

Out of hours contact details are held by the 24 hr CDSC on call duty doctor;
Tel: (+44) 020 8200 6868

Public Health

Contact details are held by the 24 hr CDSC on call duty doctor;
Tel: (+44) 020 8200 6868
REFERENCES


Food borne botulism

Intestinal colonisation (Infant botulism)

Wound botulism