Case 1.
A 27 year old male active injection drug user presents to the ED with facial palsy and diplopia. He has injection scars on his arms and legs and neck. His vital signs are normal. He has a small dried laceration above his right eyebrow from passing out after injecting into his neck 3 days ago. Despite being unkempt, no wound on his body appears grossly infected, nor cellulitis.

Make a differential diagnosis.

What toxins induce cranial neuropathy?

What is your treatment plan?

How can this patient expire despite managing his airway and respiratory status?

Case 2. A neonate presents 9 days after an unremarkable birth at home for fever, tachypnea, stridor and poor feeding. The infant is has an acidemia with a pH of 7.25 pCO₂ 30

What are common causes for this presentation?

Is infant botulism a possibility? How would that present similarly or differently?

Case 3.
A 27 year old female arrived from London on a Thursday morning. Due to her exhaustion, she decided to take 2 zolpidem tablets and eat a few brownies that her roommate left cooling on the counter.

She awoke later that afternoon with diffuse facial pain which she initially attributed to sinus pressure from the flight.

If you have time in advance, make a differential diagnosis from this first part.

She left her apartment to get some food, realizing her roommate had been in the apartment and left a note saying “Don’t eat so many brownies, they contain hash”. She felt immediately anxious and tried to get herself a cup of coffee. She checked her phone and was horrified to realize it was now Friday afternoon and she had lost track of an entire day.

As she paid for her coffee she discovered a receipt for $1200 from her dermatologist’s office from the previous day with the last four digits corresponding to her credit card. When she called the office, they confirmed that she was there and that she insisted on getting “Botox” injections to which she was completely amnestic. She touched her
tender face and spent the rest of the night on the internet, exhausted and reading about the weakness and fatigue that can accompany injection of botulinum toxin given in excess. She thinks she is poisoned. She arrives to the emergency department Saturday afternoon.

How do you decide if she has internet-induced anxiety disorder or if she could be correct?

What if this was a child with cerebral palsy who received a therapeutic dose of botulinum toxin a few days ago and who mother complains of weakness, would that change your concern?
<table>
<thead>
<tr>
<th>Citations</th>
<th>Presentation</th>
<th>Serotypes</th>
<th>Risks</th>
<th>Differential</th>
<th>Work Up</th>
<th>Rx</th>
<th>Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chertow 1996 Francisco 2007 Long 2007 Ammon 2006 Nevas 2005 Mitchell 2005 Hurst 1993 Infantbotulism.org</td>
<td>Infant neonate to 1 y Floppy Ptosis ± constipation weak cry poor suck sluggish pupils SIDS ± drooling Findings ± Hyponatremia (excess ADH) ± Fever Fatigable pupillary response (see infantbotulism.org) low amplitude EMG</td>
<td>A, B, F</td>
<td>Spore ingestion, inhalation? Household dust</td>
<td>Sepsis Meningitis Electrolytes Hypoglycemia Reye's Congenital myopathy Spinal muscular atrophy Subacute necrotizing encephalomyopathy See also adult differential for other possible differentials</td>
<td>r/o sepsis Serum and stool cultures (see infantbotulism.org under &quot;For Lab Scientists : Specimen Collection) for detailed description of the proper way to obtain stool cultures.</td>
<td>HBIG or human botulism immune globulin “BabyBig”. 100mg/kg</td>
<td>California infant Botulism Program for HBIG 510 231 7600 day or night dhs.ca.gov/ps/dcdc/infantbot/ibtindex.htm CDC Botulism Hotline 770 488 7100</td>
</tr>
</tbody>
</table>

If not other infections, then avoid clostridia-cidal antibiotics as these bacteria may lyse and release more toxin. If co-infections must be treated then avoid aminoglycosides as these may impair neurotransmission at the neuromuscular junction unless HBIG given. Place infant in reverse trendelenberg without flexion at diaphragm. Transcutaneous PCO₂ monitoring, esp if not intubated Delay vaccine schedule for live vaccines by 5 months. Maintain vigilance for co-infections esp. c. difficile Whenever possible, perform manual decompression of bladder instead of foley placement. Continue nutritional support. Infantbotulism.org

For “Toxicology of Tetanus and Botulism” break Out Session Rama B. Rao, MD ACMT 2012 Spring Conference Document Title “INFANT BOTULISMFINAL.doc” Last Updated 28 Feb 2012
## WOUND BOTULISM

<table>
<thead>
<tr>
<th>Citations</th>
<th>Presentation</th>
<th>Serotypes</th>
<th>Risks</th>
<th>Differential*</th>
<th>Work Up</th>
<th>Rx</th>
<th>Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sandrock 2001</td>
<td>Ophthalmoplegia Weakness Ptosis Dysphagia Dysarthria</td>
<td>Mostly A, occasionally B, rarely mixed A, B</td>
<td>Intramuscular or subcutaneous injection of black tar heroin Hx of abscesses High frequency of injection Clean needles and skin cleaning do not appear to reduce risk</td>
<td>Myasthenia gravis Guillan Barre (Miller Fisher Variant) Lyme disease Neurosyphilis CNS TB Lambert Eaton Vasculitis Infectious Vertebral artery Dissection Cavernous sinus disease Venoms: Elapidae, tetrodotoxin Tic paralysis Hypokalemia Endocrine emergencies</td>
<td>Sensitivity of blood, wound and stool cultures are limited but can be performed. Stool will generally be negative. For blood, require 15 mL of sera in anaerobic cultures. CSF cultures, gram stain, chemistries Can consider double blind Edrophonium (Tensilon™) test. Neuroimaging EMG/Nerve conduction studies (Classically:Nerve conduction velocities normal, normal sensory and EMG reduced amplitude and incremental response to repetitive stimulation at 50Hz but this can be variable with wound botulism)</td>
<td>- Heptavalent equine derived botulinum antitoxin (HBAT) &lt;2% IgG &gt;90% Fab fragments, mostly despecticated HBAT replaced antitoxin AB and investigational E HBAT contains: 7500 U anti A 5500 U anti B 5000 U anti C 1000 U anti D 8500 U anti E 5000 U anti F 1000 U anti G Available thru CDC as an investigational new drug Early administration for best outcome Dosing 1 vial (will rarely require more) -Debridement of wound Antibiotic administration with any of these: Penicillin, metronidazole, tetracycline and less desirable, chloramphenicol Update tetanus status</td>
<td>Early antitoxin may avoid intubation Place patient in reverse trendelenberg to reduce abdominal pressure on diaphragm Avoid aminoglycosides if possible as these may impair neurotransmission at the neuromuscular junction. Greater than 90% of wound botulism cases are in USA, mostly on West coast.</td>
</tr>
<tr>
<td>Passaro 1998</td>
<td></td>
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<td></td>
<td>Antidote Contact local PCC/DOH Or CDC Hotline 770-488-7100 bt.cdc.gov/agent/botulism/clinicians</td>
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<tr>
<td>Werner 2000</td>
<td></td>
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<td></td>
<td>* For an extensive review of cranial neuropathies see Carroll 2009</td>
</tr>
<tr>
<td>Chang GY 2003</td>
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<td>Horowitz BZ 1998</td>
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<tr>
<td>Kudrow DB 1988</td>
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<td>CDC website MMWR 2010</td>
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</tbody>
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For “Toxicology of Tetanus and Botulism” Break Out Session
Rama B. Rao, MD
ACMT 2012 Spring Conference
Document Title “WOUND BOTULISMFINAL.doc”
Last Updated 28 Feb 2012
* For an extensive review of cranial neuropathies see Carroll 2009
## Food Borne Botulism

<table>
<thead>
<tr>
<th>Citations</th>
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<th>Differential*</th>
<th>Work Up</th>
<th>Rx</th>
<th>Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chertow 2006</td>
<td>± GI distress</td>
<td>A, B, E</td>
<td>Ingestion of preformed toxin</td>
<td>Myasthenia gravis</td>
<td>Sensitivity of blood, wound and stool cultures are limited but can be</td>
<td>Heptavalent equine derived botulinum antitoxin (HBAT) &lt;2% IgG</td>
<td>Can consider activated charcoal as it may bind, highest concentration in the first 24 hours, but can be present for up to 3 weeks. Must weigh against the risk of aspiration or reduced GI activity.</td>
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<tr>
<td>Tacket 1984</td>
<td>± Throat pain</td>
<td></td>
<td>Canning or pickling inadequately heat treated or improperly packaged</td>
<td>Guillan Barre (Miller Fisher Variant)</td>
<td>Stool will generally be negative.</td>
<td>&gt;90% Fab fragments, mostly de-specified HBAT replaced antitoxin AB and investigational E</td>
<td>Higher dose generally inverse relation to incubation period.</td>
</tr>
<tr>
<td>Amon 2001</td>
<td>Cranial neuropathy</td>
<td></td>
<td></td>
<td>Lyme disease</td>
<td>For blood, require 15 mL of sera in anaerobic cultures.</td>
<td>HBAT contains: 7500 U anti A 5500 U anti B 5000 U anti C 1000 U anti D 8500 U anti E 5000 U anti F 1000 U anti G</td>
<td>Early treatment best outcome and may avoid intubation.</td>
</tr>
<tr>
<td></td>
<td>Descending weakness</td>
<td></td>
<td></td>
<td>Neurosyphilis</td>
<td>CSF cultures, gram stain, chemistries</td>
<td>Available thru CDC as an investigational new drug</td>
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<td></td>
<td>Onset between 2 and 36 hours up to</td>
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<td></td>
<td>CNS TB</td>
<td>Can consider double blind Edrophonium (tensilon) test.</td>
<td>Early administration for best outcome</td>
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<td></td>
<td>10 days</td>
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<td>Lambert Eaton</td>
<td>(Usually: Nerve conduction velocities normal, normal sensory and EMG reduced amplitude and incremental response to repetitive stimulation at 50Hz)</td>
<td>Dosing 1 vial (will rarely require more)</td>
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<td></td>
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<td>Vasculitis</td>
<td>EMG/Nerve conduction studies:</td>
<td>Antibiotic administration with any of these: Penicillin, metronidazole, tetracycline and less desirable, chloramphenicol</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td>Inflammationary</td>
<td>(Usually: Nerve conduction velocities normal, normal sensory and EMG reduced amplitude and incremental response to repetitive stimulation at 50Hz)</td>
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<td>Vertebral artery Dissection</td>
<td>Neuroimaging</td>
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<td>Cavernous sinus disease</td>
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<td></td>
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<td>Venoms: Elapidae, tetrodotoxin</td>
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<td>Tic paralysis</td>
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<td></td>
<td></td>
<td>Hypokalemia</td>
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<td></td>
<td></td>
<td>Endocrine emergencies</td>
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</tbody>
</table>

For “Toxicology of Tetanus and Botulism” Break Out Session
Rama B. Rao, MD
ACMT 2012 Spring Conference
Document Title “FOOD BOTULISM FINAL.doc”
Last Updated 28 Feb 2012 *For well describe differential of multiple cranial neuropathies see Carroll 2009
# IATROGENIC BOTULISM
Includes Illicit Botulism

<table>
<thead>
<tr>
<th>Citation</th>
<th>Presentation</th>
<th>Serotype</th>
<th>Risks</th>
<th>Differential</th>
<th>Work Up</th>
<th>Rx</th>
<th>Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Derry 2008</td>
<td>Drooling, dysphagia, bulbar weakness, apnea, incontinence, ptosis, dysphagia, flaccid paraplegia</td>
<td>Mostly A, some B</td>
<td>Death in patients receiving therapeutic administration as opposed to cosmetic administration. As many as 16 reported in US to FDA</td>
<td>See food borne</td>
<td>See food borne</td>
<td>Supportive care No indication for antidote at this time.</td>
<td>Allergan™ has a special label on each bottle that can be held under a fluorescent light and rotated to reveal a rainbow color and the company name under the print. A product without this may not be true pharmaceutical grade for Botox™ One patient responded to pyridostigmine, a caution when doing the edrophonium test. Units vary between products and could potentially cause dosing errors: 3U Dysport™= 1U Botox™ Response to prior injections is not predictive Classic presentation may be absent</td>
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<tr>
<td>Naidu 2010</td>
<td>Classic presentation may be absent</td>
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<tr>
<td>Goldstein 2006</td>
<td>Overwhelmingly these are patients with underlying neurological diseases such as spasticity, MS, who are receiving therapeutic administration of botulinum toxin as regional treatments</td>
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<td>Naumann 2006</td>
<td>Onset between 2-10 days lasting a few weeks up to 3 months</td>
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<td>Bakheit 2001</td>
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<td>Crown 2007</td>
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<td>Crown 2010</td>
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<td>Berweck 2007</td>
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<td>O’Flaherty 2006</td>
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<td>Heinen 2006</td>
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<tr>
<td>Narayanan 2011</td>
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<td>Video 2006: See refs</td>
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</tbody>
</table>

**ILLICIT USE**

| Marcus SM 2009            | Rapid weakness, cranial neuropathy, dyspnea, pain, tonic movements No GI symptoms | A, lab grade Far too potent | Despite antitoxin administration, days on vent between 36 and 171 days | Less applicable as 4 patents shared the vial | All received antitoxin but the doses of botulinum each received were massive | See Allergan™ markings on bottle described above. This case was not pharmaceutical grade. |
| Chertow 2006              |                                                                                |                |                                                                      |              |         |                             |                                                                                                  |

For “Toxicology of Tetanus and Botulism” Break Out Session
Rama B. Rao, MD
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Document Title “IATROGENIC BOTULISMFINAL.doc”
Last Updated 28 Feb 2012 *For well describe differential of multiple cranial neuropathies see Carroll 2009
### INHALATIONAL BOTULISM

<table>
<thead>
<tr>
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<th>Presentation</th>
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<th>Risks</th>
<th>Differential</th>
<th>Work Up</th>
<th>Rx</th>
<th>Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arnon 2001</td>
<td>Unwell day 2 Weakness day 3 Only 3 described cases, all lab workers</td>
<td>Type A Re-aerosolization of botulinum toxin? on fur of guinea pigs and rabbits inhaled by vets Original citation in German</td>
<td>Rare Lab workers Bioterrorism</td>
<td>See food and wound botulism</td>
<td>See food and wound botulism</td>
<td>See food and wound botulism</td>
<td>Difficult to aerosolize botulism Even concentrated botulinum toxin does not come packaged in sufficient quantities to pose a terrorist risk Most sources state decontamination is not necessary, but aerosolized botulism may warrant decontamination as suggested by the veterinarians who suffered, details of these cases are limited. There is a botulinum pentavalent toxoid vaccine used to protect labs workers from acquiring botulism toxicity. Interestingly, the serum from these vaccinated lab workers is used to make Human botulinum immunoglobulin (BabyBIG)</td>
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<tr>
<td>Nevas 2005</td>
<td>Some suggestion that infant botulism may be transmitted via inhalation/swallowing of airborne spores found in household dust as +GI spores identical to vacuum bag dust</td>
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For “Toxicology of Tetanus and Botulism” Break Out Session  
Rama B. Rao, MD  
ACMT 2012 Spring Conference  
Document Title “INHALATIONAL BOTULISMfinal.doc”  
Last Updated 28 Feb 2012  
For an extensive differential of multiple cranial neuropathies see Carroll 2009
BOTULINUM REFERENCES

BOTULINUM REFS
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Francisco AMO, Pediatrics 2007;119:826
Goldstein EM. J Child Neurol 2006;21:189-192
Horowitz BZ JAMA 1998;280:1479 (letter)
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Hurst DL. J Pediatr 1993;122:909
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MMWR 2010;59(10):399
Naidu K. Dev Med Child Neurol 2010;52:139-144
Narayanan UG. Dev Med Child Neurol 2011;53:101-102
O’ Flaherty F. Dev Med and Child Neurol 2011;53:125
Passaro DJ. JAMA 1998;279:859-863
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Werner SB Clin Inf Dis 2000;31:1018-1024

Video: Excellent graphics, case discussion.
Advanced Topics on Medical Defense Against Biological Agents- Botulinum Toxin 2006.
Available by request thru the following link:
www.usamriid.army/mil/education/distancelearning.cfm

IMPORTANT INFORMATION, each available 24/7 year round.
CDC.gov/laboratory/drugservice/formulary/html
770-488-7100

California Infant Botulism Program
Infantbotulism.org
510-231-7600

For “Toxicology of Tetanus and Botulism” Break Out Session
Rama B. Rao, MD
ACMT 2012 Spring Conference
Document Title “BOTULINUM REFERNCESFINAL.doc”
Last Updated 28 Feb 2012
TETANUS GENERAL SUMMARY

Toxins elaborated by *Clostridium tetanus*
  Tetanospasmin affects clinical manifestations
  Tetanolysin may increase bacterial growth

Inoculation via:
Bites, puncture or other traumatic wounds, surgical wounds, injection drug use
subcutaneous and intramuscular
In neonates:
poorly immunized mother, ± maternal immunosuppression: HIV, combined with
contamination of umbilicus or during circumcisions or other injury. Days 2-14, usually
day 7
Acidemia facilitates entry

20% of all affected patients with no obvious source

Disease is described in 2 Phases
  **Incubation:** time from inoculation to first clinical manifestation. Can be 1-60
days. An incubation of less than 7 days is associated with a poor prognosis.

  **Period of Onset:** Time from initial clinical manifestation to first reflex spasm.
  Usually 1-7 days. A period of less than 2 days also has a poor prognosis

Clinical findings: spectrum disorder with varying degrees of involvement
Types of Manifestations
  **Localized** This is increased tone localized to the area around the wound,
  has the potential to become generalized
  **Generalized:** Aside from neonatal, this is a life threatening form, with
  hyperreflexia, tetanic spasm and opisthotonos, may start as trismus
  **Cephalic:** A unique and uncommon form of localized tetanus where the
  wound is in the ear or on the head or neck. The manifestation is
  cranial neuropathies and trismus. It can potentially progress and
  may be hard to distinguish from botulism in early presentations
  **Neonatal:** fever, malaise, stiff neck, cranial neuropathy, poor
  feeding, acidemia, then tetanic spasm, opisthotonos

There are 2 major life threatening aspects:
  **Neuromuscular rigidity** and spasm/seizures/opisthotonos can cause hyperthermia
  and rhabdomyolysis, fluid and electrolye disorders

  **Autonomic instability:** labile hypertension, diaphoresis, with
  catecholamine surges can occur in second week and can precipitate hypertensive
  crises, or cardiovascular collapse
TETANUS GENERAL SUMMARY

Differential of initial presentation (see Ataro 2011): Strychnine poisoning, dystonia, illicit botulinum injections, other causes of cranial neuropathy (see botulism handouts), bismuth toxicity, hypocalcemia, and for simple trimus: alveolar abscess or orpharyngeal infection, serotonin syndrome, NMS, black widow envenomation, rabies

Diagnosis: Wound culture can be performed but poor sensitivity and specificity, Abdomen may be tonically contracted in tetanus, rather than sporadically in strychnine poisoning Spatula test: stimulate the posterior pharynx to cause the masseters to spasm. 100% specific, 94% sensitive (Apte 2009)

Treatment pearls:
- Rx with antibiotics but AVOID or CAUTION with Penicillin (seizure induction at high doses)
- Rx motor tone potentially with paralytics but CAUTION with pancuronium as it can block catecholamine re-uptake which may be more consequential in the second week. Caution with succinylcholine –risk hyperkalemia (a few deaths reported)
- Rx Autonomic instability: Consider centrally acting alpha blockade: clonidine or dexmetomidine and AVOID pure beta blockade as episodic release of adrenergic tone can result in unopposed alpha, sudden death reported
- Rx Tetanus IG (TIG) intramuscularly. TIG has be administered intrathecally, off label and may limit progression to severe disease, but one case of reversible paraplegia has been reported. Manufacturer advises against its administration intrathecally. Data however suggests it may be protective for length of stay and hospitalization even in neonates.
  - Wound debridement
  - Good supportive care
  - Tetanus toxoid to initiate or complete vaccination for future events once stable
  - Dark, quiet, non-stimulating area

Other
Vaccine can cause a diffuse localized swelling of upper arm, an Arthus like reaction usually 2-8 hours post injection in persons who have received multiple vaccine doses. These patients can be treated supportively and they usually have high protective antibody titers and require q10 year dosing. Generalized allergic reactions can occur, and very rarely an association with GBS.
TETANUS REFERENCES

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