Etiology

- Conditions that can result in blindness following anesthesia include: Corneal abrasion, vitreous loss, hemorrhage, movement of pt while operating on or in the eye, chemical injury to the cornea or conjunctiva from cleaning materials on the anesthetic mask, spillage of prep solution into the eye, and direct trauma to the eye due to OR table padding, needle used in retrobulbar block, anesthetic mask pressure on the globe, or foreign body falling into the eye. Additionally, prone position, hypoxemia following cardiac arrest, prolonged hypotension, CRAO, increased intraocular pressure, and embolization, occlusion, thrombosis, or spasm of the retinal artery.
- Blindness may occur following absorption of glycine irrigating solution during TURP (glycine distribution similar to that of γ-aminobutyric acid, an inhibitory neurotransmitter; levels of glycine >143 mg/L associated with transient blindness).

Usual Treatment

- In the case of glycine, supportive treatment is indicated until plasma glycine levels <143 mg/L.
- ION: There is no effective treatment and most lost vision is not recovered.
- CRAO: Immediate lowering of intraocular pressure with acetazolamide and topical medications; hyperbaric O_2 therapy may be beneficial if begun within 2–12 h of symptom onset.
- Consider stated spine procedures on high-risk pts.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
GENERAL	Retinal artery occlusion	Migraines, coagulopathies, hemoglobinopathies, and oral contraceptives increase IOP	Pale ischemic retina with pathognomonic cherry-red spot and afferent papillary defect	
HEENT	Ischemic retinopathy	Hypotension Hypoxemia Shock	Funduscopic: Normal retina but optic nerve head is swollen and ischemic. Eventual optic nerve pallor	
	Orbital pressure		Funduscopic: Edematous retina with dilated arterioles and engorged veins	
GU	Transient blindness during or after TURP	TURP with glycine irrigating solution	Normal papillary response to light and accommodation; Fundus normal	Plasma glycine level (nml 13–17 mg/L)

Key References: Lee LA, Roth S, Posner KL, et al.: The American Society of Anesthesiologists Postoperative Visual Loss Registry: analysis of 93 spine surgery cases with postoperative visual loss. *Anesthesiology* 105:652–659, 2006; Shen Y, Drum M, Roth S: The prevalence of perioperative visual loss in the United States: a 10-year study from 1996 to 2005 of spinal, orthopedic, cardiac and general surgery. *Anesth Analg* 109:1534, 2009.

Perioperative Implications

Preinduction/Induction/Maintenance

- Proper positioning essential.
- If pt is prone, adequate padding so no pressure is transmitted to either globe or nasal bridge.
- When the face is completely draped, consider use of a metallic Fox shield to protect eye from inadvertent pressure.

Monitoring

- Eye checks frequently during the procedure to ensure no pressure on the globe
- Ensure adequate venous drainage without increased venous pressure or increased intracranial pressure, particularly when venous outflow may be compromised by position or procedure.

General Anesthesia

- Anesthetic masks may injure eye, either through inadequate drying and application of cleaning solution to eye or through direct pressure.
- Hypotension and hypoxemia implicated in cases of CRAO.
- Hypotension, anemia, and prolonged procedures are implicated in ION.

Regional Anesthesia

- In ophthalmic nerve blocks, needle does not enter globe or retinal artery, vein, or nerve. Avoid excessive volume of local anesthetic, which increases IOP and may compromise vascular supply of the globe.

Postoperative Period

- When pt is recovering in the prone position, ensure there is no pressure on orbit or globe.

Anticipated Problems/Concerns

- Absorption of glycine from 1.5% glycine irrigation fluid may be significant.
- ION usually occurs without any other evidence of vascular injury.
- Optic nerve may be very vulnerable to hemodynamic changes in the prone position.

Botulism

Debra E. Morrison

Risk

- Infant botulism.
- Wound botulism.
- Foodborne botulism.
- Adult intestinal toxemia.
- Injection botulism.
- Biological warfare/inhalational botulism (Category A biological threat).
- Incidence
  - In USA, approximately 145 cases are reported each year: infant botulism 65%, wound 20%, and foodborne 15%; adult intestinal colonization and iatrogenic botulism rare.
  - Foodborne outbreaks of two or more persons occur most years, and are usually caused by home-preserved foods with low-acid content (pH ≥4.6, although toxin will not be formed in acidic foods, low pH will not degrade any preformed toxin). Foods implicated differ between countries, reflecting local eating habits and food preservation procedures. Improper handling of commercially prepared foods has also been implicated (canned, fermented, salted, and smoked), including unrefrigerated infused cooking oils, baked potatoes wrapped in foil and left sitting out before eating, and ready-to-eat foods in low-oxygen packaging. Low temperature, high salt, and low pH prevent growth of bacteria and toxin formation. Food samples associated with suspect cases should be sealed, stored, and sent to labs.

Perioperative Risks

- Dx late, incorrect or missed
  - Differential Dx: For adults, myasthenia gravis, Eaton-Lambert, Guillain-Barre, virus attacking brain/spinal cord, CVA, organophosphate exposure, tick paralysis, other neurotoxin; may need brain scan, spinal fluid examination, and EMG, tensilon test to rule out other causes; for infants, sepsis, failure to thrive, dehydration, encephalitis, and metabolic disease
  - Nonspecific history and physical findings: classic adult symptoms include double vision, drooping eyelids, slurred speech, difficulty swallowing, dry mouth, muscle weakness; infants appear lethargic, feed poorly, are constipated, and have a weak cry and poor muscle tone—if untreated, symptoms may progress to cause paralysis of the respiratory muscles, arms, legs and trunk; fever and loss of consciousness are not associated symptoms
  - Onset of foodborne botulism: usually 12-18-36 h after eating contaminated food, but can be as early as 4 to 6 h or as late as 8 to 10 d
  - Laboratory result takes d to wk and should be used only as confirmation; treat before confirmation; tests are performed at some state health department labs and at CDC
  - Triad: Bulbar symptoms, resp compromise, and dilated pupils
- Prolonged weakness requiring prolonged support
- Enteral nutrition: Desired but problematic due to gastroparesis and bowel paralysis
- Aspiration risk
- Elevated potassium if immobile in ICU

Worry About

- Arrhythmias
- Hyperkalemia, arrhythmias, and then cardiac arrest
- Prolonged weakness necessitating prolonged intubation and leading to nosocomial infection
- Skin breakdown

Overview

- Botulism is a rare but serious neuroparalytic illness caused by a nerve toxin (BoNT) produced by the rod-shaped gram-positive bacterium *Clostridium botulinum* (and sometimes by strains of *Clostridium butyricum* and *Clostridium baratii*), commonly found in soil. *C. botulinum* grows best in low-oxygen conditions; spores survive in a dormant state until exposed to conditions that support growth. Seven types of toxins (A to G), but only A, B, E, and rarely, F cause illness in humans; three different intracellular protein targets; and different durations.
- In infant between 2 wk and 1 y old, occurs by ingestion of spores, which grow in intestine and release toxin, usually by honey ingestion, or associated with parent who works with soil or with living in rural areas.
- Occurs in wounds of IV/skin popping drug users (or any traumatized tissue contaminated with organisms) in which there is local infection and absorption of produced toxin. There is increased incidence over the last several years in IV drug users (black tar heroin), especially in California.
- Foodborne: Improperly preserved or cooked food, even properly cooked food left at improper temperature, allows germination and toxin production by contaminating spores; consumption of food with preformed toxin results in absorption of potent neurotoxin; with education and control of food industries, now uncommon in USA; ingestion of infected inadequately cooked wildlife poses at least potential risk. Foodborne botulism can be a public health emergency.
- Intestinal: Spore colonization possible in adults as well if normal gut flora has been altered by surgery or antibiotic therapy.
- Cosmetic injections (black market toxin, Botox overdose, or spread beyond injection site) or cerebral palsy (Botox overdose or spread beyond injection site) are a cause.
- Inhalational: Genetically engineered toxin, development of biological warfare (at-risk locations). Concern is inadequate stocking of antidotes worldwide and inadequate preparation and medical support. Biological warfare in Iraq has led to organization of task forces such as Scorpio at the national/regional level to stockpile antidotes. Median lethal dose for humans has been estimated at 2 nanograms/kg, approximately three-times greater than in foodborne cases. If inhalation exposure is suspected, additional exposure must be prevented by removal and storage of clothing in plastic until it can be washed, as well as immediate showering and decontamination of those exposed.
- Waterborne: Could theoretically result from ingestion of water contaminated with preformed toxin, but risk is low if common water treatment processes are used (boiling and disinfection with 0.1% hypochlorite bleach solution).

Etiology

- Botulinum toxin binds irreversibly to synaptic membrane of cholinergic nerves and prevents release of acetylcholine but not its synthesis and storage.

Usual Treatment

- Supportive; may be on ventilator for wk to mo and require intense medical and nursing care.
- Nutritional support; enteral preferred (basic maintenance plus need to keep bowels moving to eliminate spores), but parenteral also required.
- Early antitoxin treatment shows better outcome; antitoxin blocks action of circulating toxin and prevents patients from worsening, but recovery still takes many wk/up to several mo. Long-term effects may include fatigue and shortness of breath for y, requiring long-term therapy and with implications for later anesthetics.
- Efforts may be made to remove toxin from the gut by inducing vomiting and using enemas. Avoid cathartic agents containing magnesium because of the theoretical concern that increased magnesium levels may enhance the action of botulinum toxin.
- Equine-derived antitoxin for adults (risk of serum sickness/anaphylaxis); skin testing and desensitization instructions provided with antitoxin; more broad-spectrum antitoxins associated with increase in hypersensitivity; available from the CDC.
- Presently trivalent antitoxin preparation is available for adults (10 mL vial with 7500 IU type A, 5500 IU type B, and 8500 IU type E): available from the CDC.
- BabyBIG (human botulism immune globulin) used for infant botulism came out in 1990; more in use since 2003: available from state public health departments.
- Vaccine exists but rarely used because effectiveness has not been fully evaluated and negative side effects have been demonstrated.
- Botulism reportable to CDC or state health department and requires report to obtain antitoxin.
- Antibiotics for secondary infections.
- Avoid aminoglycosides and clindamycin, which may potentiate or exacerbate neuromuscular blockade.
- Guanidine increases the release of acetylcholine from nerve terminals and appears to be useful in mild cases.
- Modern clinical practice and early antitoxin treatment: mortality reduced from 50% to 60% to 3% to 5%; worldwide mortality cited as 5% to 10% by WHO.

Assessment Points

System	Effect	Assessment by Hx	PE	Tests
RESP	Pharyngeal constrictor and genioglossal hypotonia, paralysis of resp musculature Infection Atelectasis	Drooling Poor feeding Decreased resp effort Increased secretions Tracheal secretions Poor resp effort Poor color	Poor head control Absent gag Weak cough Fever Rhonchi Rales Cyanosis	Diagnosis of elimination; electrophysiology studies are fastest diagnostic tool to rule out other causes; EEG and neuroimaging are normal as long as there is no hypoxic insult; edrophonium test to rule out myasthenia gravis shows no improvement Blood, urine, CSF analysis and culture, and metabolic and hepatic profiles are generally within normal limits Stool samples are difficult to collect due to constipation; can use sterile water enema Serum testing possible if stool is unobtainable but has low sensitivity compared with stool testing (negative serum test does not exclude possibility of infant botulism) Samples injected into mice; look for signs of botulism Laboratory result takes d to wk and is used as confirmation Tests only performed at some state health department labs and CDC; samples must be collected sterilely, refrigerated, and shipped with cold packs
GI	Constipation	No bowel movement Irritability	Palpable stool Abdominal distention	
RENAL	UTI	Foul-smelling urine		
CNS	SIADH Seizures Cranial neuropathies	Infrequent urination Twitching Altered consciousness Ptosis Expressionless face Feeble cry	Diminished urine flow Seizure activity Fixed and dilated pupils Facial palsy Poor cough and gag	
PNS	Spinal neuropathies	Limp limbs	Hypotonia	

Key References: Centers for Disease Control and Prevention (CDC); National Center for Emerging and Zoonotic Infectious Diseases: Botulism: general information. <http://www.cdc.gov/nczved/divisions/dfbmd/diseases/botulism/>. (Accessed 24.02.16.); World Health Organization; Nantel AJ: *Clostridium botulinum*. International programme on chemical safety. Poisons Information Monograph 858 Bacteria. *Geneva*, 1999. <http://www.who.int/csr/delibepidemics/clostridiumbotulism.pdf/>. (Accessed 24.02.16.)

Perioperative Implications

- Early diagnosis, treatment, and optimization
- Continue supportive resp care
- Sepsis from secondary infections
- Avoid resp depressants and paralytics
- Aspiration risk
- Pts may require feeding tube (jejuna better than gastric to minimize aspiration risk) and/or parenteral nutrition
- Likely to OR/IR/GI for wound debridement, feeding tube, tracheostomy, and central line
- If possible, avoid airway manipulation, unnecessary medications, and those that are resp depressants
- Avoid narcotics because of their effect on bowel; consider alvimopan before narcotics are given if they are necessary

Preoperative Preparation
- Recommend pt receives antitoxin before wound debridement so additional toxin release does not cause further paralysis
- Low threshold for treatment if suspecting botulism
- Manage preop electrolytes
- Botulism does not affect endocrine, hematologic, hepatic, or renal function (except for neurogenic bladder)
- Continue antibiotics
- CXR to help assess status
- Aspiration prophylaxis
- If pregnant, parturient can safely be given as antitoxin (intrathecally in severe cases); consider early tracheostomy to avoid sequelae of resp depression; botulism is not known to cause direct fetal risks only those associated with mother's ventilatory compromise, because the molecule is too large to pass through placental barrier

Monitoring
- Standard ASA monitors.
- If pt unstable in ICU, consider arterial cannulation for management of autonomic dysfunction; infants may see motor function return before autonomic system function returns.

Airway
- Aspiration risk
- May already be intubated or have tracheostomy

Induction
- Avoid succinylcholine.
- May not require paralytic.

Maintenance
- May not require paralytic throughout treatment

Extubation
- Likely unable to extubate.
- Continue supportive care postop.

Adjuvants
- Avoid resp depressants if possible.
- Consider regional procedures and nonnarcotic pain medications rather than narcotics for pain control in wounds.

Postoperative Period
- Continued supportive care
- Manage electrolytes

Associated Problems/Concerns
- Aspiration pneumonia
- Sepsis from wound
- Missed diagnosis
- Malnutrition
- Biological warfare: Limited information on effectiveness of antitoxin success with inhalational botulism; amount of neutralizing antibody in presently available formulation may not be enough for treatment of genetically engineered toxin
- Travel: food preservation techniques vary according to local custom. WHO supports efforts to detect and respond to botulism, through INFOSAN, which links national authorities in charge of managing food safety events in member states; INFOSAN is managed jointly by FAO and WHO.

Brain Death

Jessica L. Shanahan | T. Anthony Anderson

Risk
- Number of pts awaiting organ transplantation is much greater than the number of available solid organs
- Medical management affects the viability of organs for transplant

Perioperative Risks
- Cardiovascular collapse
- Pulmonary edema
- Endocrine dysfunction
- Metabolic imbalance
- Coagulopathy
- Hypothermia

Worry About
- Cardiovascular collapse and metabolic derangement limiting organ viability

Overview
- Brain death is a clinical diagnosis in a comatose pt who has suffered terminal neurologic insult with confirmation of irreversibility and lack of confounding variables (e.g., hypothermia, severe electrolyte disturbances, endocrine disturbances, drug intoxication, acid-base abnormalities).
- Brainstem function is absent.
- Ancillary testing such as EEG, cerebral angiography, or transcranial Doppler may be used to support the diagnosis but is not required.
- An initial catecholamine surge occurs after brain death (initial increased HR with potential arrhythmias, increased SVR, and increased BP).
  - Associated myocardial injury may arise from increased SVR and result in LV failure and decreased CO.
  - Neurogenic pulmonary edema may result.
- After several h, loss of sympathetic tone may occur, causing hypotension and limiting organ viability if untreated.
- Endocrine dysfunction occurs due to pituitary infarction, causing DI, hypothyroidism, and hyperglycemia.
  - DI further exacerbates hypovolemia/hypotension.
- ICU care can affect the viability of organs for transplantation.

Etiology
- Elevated ICP, anoxic brain injury, and trauma

Usual Treatment
- Treatment protocols may improve organ viability, increasing the number of transplanted organs and the long-term function of the transplanted organs.
- Replete DI losses, maintain BP to allow adequate organ perfusion, use a lung-protective ventilatory strategy, control endocrine abnormalities with insulin and vasopressin (consider thyroxine/T3 and corticosteroids, especially if low ejection fraction or hemodynamic instability), transfuse to maintain oxygen delivery to organs, and correct coagulopathy if ongoing bleeding.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
RESP	Pulmonary edema ARDS/ALI	Low PaO_2 Increased peak airway pressures		ABG CXR
CV	Myocardial injury Loss of vascular tone Hemodynamic instability Hypovolemia	Hypotension		BP +/– CVC +/– PAC +/– Cardiac catheter +/– TEE
HEME	Coagulopathy, may progress to disseminated intravascular coagulation Anemia			Coagulation studies HCT
ENDO	DI Hypothyroid Hyperglycemia Hypernatremia			Lytes Low urine specific gravity Elevated UOP Glucose
CNS	Lack of cerebral and brainstem function Poikilothermic	Hx of drug ingestion, metabolic encephalopathy, and/or hypothermia excluded	Absent brainstem reflexes (apnea test)	Toxicology screen Temp monitor +/– EEG, cerebral angiography, brain imaging
MS	Reflex somatic movements mediated by spinal reflexes		Neurologic exam	

Key References: Anderson TA, Bekker P, Vagefi PA: Anesthetic considerations in organ procurement surgery: a narrative review, *Can J Anest* 62(5):529–539, 2015; Cross R: Brain death. In Fleisher LA, Roizen MF, editors: *Essence of anesthesia practice,* ed 3, Philadelphia, 2010, Elsevier, p 49.