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.

Perioperative Implications

Monitoring
- Temp
- A-line
- CVP +/– PAC
- UOP
- ABG

Airway
- Lung-protective ventilation: TV 6-8 mL/kg of ideal body weight; PEEP 8-10 cm H_2O
- Judicious IV fluid (CVP <10)

Maintenance
- Correct metabolic derangements (acidosis, hypoxemia, and hypercarbia) and electrolyte abnormalities (hypernatremia and hyperglycemia).
- Evidence suggests volatile anesthetics are best for long-term organ outcome.
- Restore intravascular volume, replacing DI urinary losses and evaporative losses.
- Consider vasopressin to support hemodynamics and control polyuria (Vasopressin 1 U IV bolus, 2.4 U/h IV infusion).
- Use other vasopressors as necessary to maintain adequate organ profusion (norepinephrine and dopamine).
- "Lung-protective" ventilatory strategy: TV 6 to 8 mL/kg of predicted body weight; PEEP 8 to 10 cm H_2O.
- Maintain SBP >100 mm Hg, MAP >70, HR 60 to 120 bpm, and CVP 4 to 8 (<10) mm Hg.
- Insulin infusion to maintain serum glucose <180 mg/dL.
- Consider hormone replacement with thyroxine or T3 infusion (thyroid hormone (tetraiodothyronine) 20 lg IV bolus, 10 mcg/h IV infusion) and corticosteroids (methylprednisolone 15 mg/kg IV q24h).
- Transfuse for Hgb <7 or 8 g/dL.
- Correct coagulopathy with clotting factors or platelets if evidence of ongoing bleeding.
- Maintain skeletal muscle paralysis.
- Keep normothermic.

Extubation
- Not done
- Ventilation discontinued when aorta cross-clamped

Adjuvants
- Heparin per procurement team

Anticipated Problems/Concerns
- Increased HR and BP on incision do not obviate the criteria for brain death.
- Removal of CVC or PAC during heart procurement may be requested.
- Lung recruitment maneuver may be requested with the lungs held open with 10 cm H_2O continuous airway pressure before lung procurement.

Brain Injury, Traumatic

Mitchell L. Weinstein

Risk
- Incidence in USA: 1.7 million TBIs per year as of 2010, resulting in more than 280,000 hospitalizations and over 50,000 deaths.
- TBI is responsible for about 30% of all deaths due to injury.
- TBI, primarily from falls, has increased more than 50% in geriatrics from 2001 to 2010.

Perioperative Risks
- Brain herniation
- Coagulopathy, DIC
- Metabolic derangement

Worry About
- Occult cervical spine injury
- Other preexisting medical conditions
- Neurogenic pulm edema
- Subclinical seizures

Overview
- TBI is a major cause of death and disability with increasing rates among senior citizens.
- Care is focused on avoiding secondary injury to the brain.
- Normal saline without glucose should be used instead of colloid or albumin. Hypertonic saline can be used with appropriate caution.
- Brief moments of hypocapnia may occur to urgently lower ICP, otherwise normocapnia.
- Avoid hyperthermia. There is no consensus on therapeutic hypothermia.
- Antiseizure prophylaxis with phenytoin to levetiracetam for high-risk pts.

Etiology
- External trauma causing brain contusion, laceration, diffuse axonal, injury, or hematoma.
- Spontaneous bleeding from cerebral vessels may occur, subarachnoid or intracerebral.
- GCS ≤8 is severe TBI; 9 ≤GCS ≤12 is moderate TBI.

Usual Treatment
- Emergent decompressive craniectomy usually occurs in TBI from a stroke.
- Keep ICP <20 by elevating HOB and extraventricular draining of CSF.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Facial fractures; cervical spine injury, oropharyngeal injury	Mechanism of injury	Periorbital or mastoid ecchymosis; epistaxis; blood or vomitus in mouth	CT head; skull X-ray
RESP	Neurogenic pulm edema	Sudden onset of dyspnea	Tachypnea, tachycardia, pink frothy sputum	ABG, CXR
CV	Neurogenic stunned myocardium	ECG changes	Cardiac dysrhythmias, decreased CO	Cardiac enzymes, TEE
METAB	Hyponatremia/hypernatremia Hypoglycemia/hyperglycemia	Mental status changes Changes in urine output		Blood lytes, glucose
CNS	Seizures	Decreased mental status, failure to improve with treatment	Seizure activity may be subclinical	EEG

Key References: Wijayatilake DS, Jigajinni SV, Sherren PB: Traumatic brain injury: physiological targets for clinical practice in the prehospital setting and on the neuro-ICU, *Curr Opin Anaesthesiol* 28(5):517–524, 2015; Sharma D, Vavilala MS: Perioperative management of adult traumatic brain injury, *Anesthesiol Clin* 30(2):333–346, 2012.

Perioperative Implications

Preoperative Preparation
- Early intubation if GCS <8 or cannot maintain airway.
- Evaluate other injuries.
- Mannitol (0.25–1 g/kg body weight) if ICP >20 mm Hg and no severe hypovolemia.
- FFP, platelets, and 2 units PRBC typed and crossed.

Monitoring
- Arterial line is mandatory.
- Consider CVP.
- ICP monitor.
- Urine output.
- Consider monitoring cerebral oxygenation, blood flow, or metabolism if available.

Airway
- Manual in-line stabilization.
- Avoid nasal intubation.

Preinduction/Induction
- Aspiration risk; use a rapid sequence induction; use succinylcholine if concerned about difficult airway and sugammadex is not available.
- Avoid hypoxia (PaO_2 <60 mm Hg or SpO_2 <90) and hypercarbia.
- Avoid hypotension (SBP < 90 mm Hg).

Maintenance
- Keep pt normocarbic, normoglycemic, and normothermic.
- Maintain cerebral perfusion pressure between 50–70 mm Hg by either lowering ICP, raising MAP, or both. Keeping CPP >70 can increase risk of ARDS.
- Low-dose inhaled agents or propofol for maintenance.

Extubation
- Consider extubation if airway reflexes are intact and can maintain $PaCO_2$ 35–45 mm Hg.
- Avoid coughing or agitation.

Postoperative Period
- Avoid significant hypertension to prevent rebleeding.
- Keep head of bead elevated at 30 degrees and set ICP monitor appropriately.
- Continue to follow blood chemistry and coagulation.
- Deep sedation to reduce cerebral metabolism, if needed.