Neurosurgical Care – Case Examples from Bagram

The Deployed Experience: Oct 2018 – Apr 2019

Maj Patrick R. Maloney, M.D., USAF
Neurosurgeon
455th EMDG, Craig Joint Theater Hospital
Bagram Airfield, Afghanistan
No Disclosures

The views and opinions expressed are my own and do not reflect that of the USAF.
Objectives

- Overview of CJTH and the deployed environment
- History of Military Neurosurgery
- Pathophysiology of TBI
- Briefly summarize relevant JOINT TRAUMA SYSTEM CLINICAL PRACTICE GUIDELINE on Traumatic Brain Injury Management in Prolonged Field Care (see handouts)
- TBI outcomes in the deployed setting
- Highlight specific TBI topics through review neurosurgical cases
Most common question: Is it safe?

Incoming, incoming! IDF impact! TAKE COVER

Counter-rocket, artillery, mortar (C-RAM)

Mine-Resistant Ambush Protected (MRAP)

Individual Body Armor (IBA)
Consequences of Indirect Fire Impact (IDF)
Afghanistan is objectively and hauntingly beautiful
History

- Trephination documents pre-Incan surgeons in the area of the modern state of Peru performing the procedure as early as 3000 BCE.
- Military begins documentation of treating head wounds
  - Crimean War (1853–1886) and the American Civil War (1861–1865) with advent of ether inhalation and improved battlefield care → more opportunities to treat all injuries in controlled surgical setting → including cranial trauma
    - Operative mortality rates for cranial trauma reported 70-90%, mostly secondary to hemorrhage and infection
- Modern Neurosurgery begins as a specialty around 1901 with Harvey Cushing dedicating 100% of his surgical practice to invasive cranial surgery → just in time for WWI
History (continued)

- Cushing served as *the* Army neurosurgeon in France from 1917 to 1918.
  - 3-month period in 1917 he and his team operated on 133 soldiers with ‘brain’ wounds; 1.5 patients/day
  - Operative mortality steadily dropped throughout his deployment, achieving a mortality rate for his last 45 patients of ~29%, and an overall mortality rate of ~35% proving that forward deployment, meticulous debridement, hemostasis and closure lead to meaningful survival benefit
  - 1919 annual meeting of American College of Surgeons, William Mayo announces founding of neurosurgery as a distinct surgical subspecialty

- Modern Conflicts:
  - WWII utilizes improved technique, dropping mortality rate of all operative head traumas in theater to 14%; bigger strides made in treatment of SCI patients (paraplegics) dropping mortality from 90% at 1-year to 25% at 1–year (credit goes to physiatry>neurosurgery success story)
  - Korea and Vietnam Wars see development of two-tiered (rotary) triage system, widespread use of antibiotics and early evacuation see infection rate drop to 1% for neurosurgical patients
  - Israeli-Lebanese conflict utilize in theater CT, prompting shift to conservative management for some cranial injuries
  - Iraq and Afghanistan see higher incidence of blast induced TBI and aggressive use of early decompressive craniectomy

Injury Patterns of TBI

● Blunt (blast) Forces: IED, MVAs, Falls, etc.
  ○ Can result in mass lesions (EDH, SDH, ICH, etc.) requiring evacuation must be rapidly identified and treated surgically.
  ○ Decompression should be undertaken for all salvageable patients with mass lesions requiring evacuation following low-velocity and blunt mechanisms of injury.

● Penetrating intracranial injury: any projectile that can penetrate brain → shrapnel, gravel/rocks, bullets from GSW (high vs low velocity rounds)
  ○ Surgical decompression (liberal use of craniectomy) is recommended on patients with high velocity penetration injuries who do not have existing or impending signs of brain death
  ○ Appropriately debride devitalized brain tissue
  ○ Routine pursuit of individual foreign bodies within the brain is NOT advisable, but is left to the discretion of the neurosurgeon
Penetrating Brain Injury (PBI): GSWH

All Tissue Layers effected:

- **Injury to Soft Tissues:**
  - Scalp and Face burns
  - Soft tissue and bacteria dragged intracranially
  - Pressure waves of gas combustion can cause cavitation if close to head

- **Comminuted fracture of bone:**
  - Vessel injuries
  - Brain parenchymal injuries

- **Injuries amplified:**
  - Ricochet off bone
  - Bullet deviating from its original path:
    - (Pitch-forward rotation) (Yaw- Rotation on Vertical axis)
  - Bullet mushrooming from impact
Let’s Get Physical…..

- Primary Injury is related to bullet/projectile **IMPACT** Velocity

\[ v = \frac{d}{t} \]

- \( v \) = speed
- \( d \) = distance travelled
- \( t \) = time taken

Doesn’t take a genius to know that getting shot in the head is bad!
More Physical

- Primary Injury is related to bullet **IMPACT** Velocity (IV)
- \( IV > 100 \text{m/s} \rightarrow \) Explosive intracranial injury that is uniformly lethal.
Low Velocity PBI

- Non-Bullet Missiles: (Grenades, IED shrapnel)
- Low Muzzle Velocity (< 250m/s – Most handguns)
  - Tissue Injury caused primarily by laceration and maceration along path slightly wider than missile diameter

Don’t play with guns → even low muzzle velocity hand guns at close range can have devastating effects
High Velocity PBI

- High Muzzle Velocity (600-750m/s) from military weapons and hunting rifles:
  - Shock waves $\rightarrow$ cone of death in tissue
  - Low pressure areas that drag debris into wound
### TBI ‘Red Flags’

**Table 2. Features Indicative of Moderate to Severe Head Injury**

<table>
<thead>
<tr>
<th>Red Flags</th>
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<tbody>
<tr>
<td>Witnessed loss of consciousness</td>
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<tr>
<td>Two or more blast exposures within 72 hours</td>
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<tr>
<td>Unusual behavior or combative</td>
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<td>Unequal pupils</td>
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<tr>
<td>Seizures</td>
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<tr>
<td>Repeated vomiting</td>
</tr>
<tr>
<td>Double vision or loss of vision</td>
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<tr>
<td>Worsening headache</td>
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<tr>
<td>Weakness on one side of the body</td>
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<tr>
<td>Cannot recognize people or disoriented to place</td>
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<tr>
<td>Abnormal speech</td>
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</tbody>
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![Brain Diagram](image_url)
Herniation Syndrome
'Unequal’ Pupils in TBI

- Prognosis of pupillary dilation due to 3rd nerve compression from acute cerebral swelling (TBI, CVA, aSAH) →
  - Mortality 75%
  - 15% outcome was unfavorable (GOS 2 and 3)
  - 10% favorable (GOS 4, 5) at 24 month follow up
Avoid Hypotension:

- SBP >100 ages 50-69yrs,
- SBP > 110 for 15-49yrs, or over 70yrs
- ** SBP < 90 is the single greatest risk for poor outcome in the neurologically injured patient.**

Avoid hypoxemia and secure airway in obtunded patient:

- SaO2 > 90% and PaO2 >60

Perform serial neurological exams

- GCS, pupils, gross focal neurological signs
Primary survey → ABCDE → Perform a rapid trauma survey to assess all injuries according to ATLS guidelines
  ○ Determine and record the Glasgow Coma Scale (GCS) score
  ○ Assess pupils and motor function in all four extremities

Secondary survey: After stabilizing any immediate life-threatening injuries, assess for TBI red flags that may indicate moderate to severe head injury, and perform an initial detailed neurologic examination.

TBI Resuscitation and Medical Treatment:
● Blood products > albumin
● For mass transfusion and maintenance fluids:
  ○ Normal saline preferred crystalloid solution
    ■ Avoid hypotonic solutions and solutions with glucose → exacerbates brain swelling
  ○ Prophylactic Hyperventilation → do not perform!
  ○ Prophylactic Abx → No indication in closed, but recommend in open injuries
    ■ Rec: Ancef 2g q 8hrs, or clindamycin 600mg q 8hrs.
    ■ If penetrating injury and grossly contaminated with organic debris add metronidazole 500mg IV every 8-12hrs.
  ○ Monitor glucose q 6hrs (Goal < 180)
  ○ Steroids: Always bad in head trauma.
  ○ Normothermia
Transporting to Role 3:

- *Early* transport is shown to improve outcomes
- Sedation/Paralytics:
  - Vecuronium is preferred: readily available, no need for refrigeration
  - Propofol is preferred for sedation
    - Advantages: quick on and off, lowers ICPs, controls seizures, lowers cerebral metabolic rate of oxygen (CMRO2)
  - Pain control critically important (high pain/stress can → higher ICPs)
    - Use fentanyl over morphine or dilaudid in the neuro patient
      → histamine release

JTS CPG: Time is Brain
Combat-related brain injury sustained between 2005 and 2015 who underwent craniectomy at deployed surgical facilities were identified from the DOD Trauma Registry and JTS Role 2 Registry.

486 patients identified as having undergone craniectomy, 213 (44%) had complete date/time values

Postoperative mortality was significantly lower when craniectomy was initiated within 5.33 hours of injury.
Time from injury to craniectomy

- Follow-up for survival ended at death, hospital discharge, or hospital day 16, whichever occurred first.
- Follow-up extended to at least 1 day after hospital arrival for 95% of survivors, 2 days for 85%, 5 days for 50%, and 16 days for 15%. Survivors discharged before day 16 were considered alive.
Conclusions

● Increased mortality risk for delays greater than 5.33 hours was due mostly to *prehospital* rather than *in-hospital* delays.

● Mortality alone is a poor measure of success in severe TBI
  ○ Measuring stick for combat related injuries different than civilian trauma practices

● Focusing on functional outcomes with minimum follow-up time (at least 90 days)
ICP management:
- Head injury with GCS <8 or declining neuro exam, start hypertonic prior to transfer
- 3% saline
  - 250ml bolus, then 50-100cc/hr
  - Check q6hr sodium levels
- Mannitol: consider for hemodynamically stable patient in extreme stages of ICP elevation (posturing/herniating patients) or borderline ICPs with intracranial pressure monitor. Consider dose prior to flights.
  - Caution in hypotensive patients
  - Replace urine output with isotonic fluids
  - Mannitol: 1g/kg IV followed by 0.25g/kg IV push q 4hrs.
Other precautions in ICP management:

- Avoid hyperthermia because it increases metabolic demand and can worsen secondary injury.
- Elevate the head of the bed by 30-45 degrees.
  - With complete spine precautions, a flat bed in reverse Trendelenburg position is okay.
- Gastric (stress) ulcer prevention with PPI.
- Enteral nutrition when appropriate.
  - Avoid NG tubes with suspected head injury!
Risk of Venous Thrombosis:

- Starting DVT prophylaxis should be done in discussion with theatre neurosurgeon and trauma surgeon
  - Risk of spontaneous intracerebral hemorrhage expansion could theoretically be exacerbated with chemoprophylaxis
- Mod and Severe head injury: should get SCDs asap, and when appropriate chemical prophylaxis
- Lovenox 30mg bid, or 5000u sub q hep bid may be used without increased risk of hemorrhagic issues AFTER CLEARENCE BY NEUROSURGEON.
All Kinds of ICP Monitors

- ICP monitors:
  - ICP monitors for all salvageable patients with severe TBI and abnormal CT head.
  - ICP monitor for patients with severe TBI and normal CT if > 40 yrs of age, posturing, systolic blood pressure < 90 mmHg
  - Options:
    - External Ventricular Drain
    - Bolt → currently have no monitoring tower at CJTH 😞
  - Goal ICP is < 22 mmHg
Most Common/User Friendly ICP Monitor

Parenchymal ICP monitor “Bolt”
EVDs can measure $\text{IVP} \approx \text{ICP}$

External Ventricular Drain $\rightarrow$ serves both diagnostic and therapeutic purpose.
Surgical Considerations for in the field neurosurgery without power

Exploratory burr holes: useless → do not attempt and delay transport!

- Theoretically useful in the following situation when ALL criteria apply:
  - 1) no immediate neurosurgical capabilities
  - 2) no CT scan available
  - 3) deteriorating neurological exam that can be localized
  - 4) unilateral pupillary changes
"Every surgeon carries within himself a small cemetery, where from time to time he goes to pray- a place of bitterness and regret, where he must look for an explanation for his failures.” – Rene Leriche, La philosophie de la chirurgie, 1951

Long Term Outcomes

“Surgeons must always tell the truth but rarely, if ever, deprive patients of all hope. It can be very difficult to find the balance between optimism and realism.” – Henry Marsh, Do No Harm: Stories of Life, Death, and Brain Surgery
Long-term functional outcomes from combat-related penetrating brain injury (BPI)  
  ○ 24 months FU period!

Retrospective review of PBI cases of Role 3 Multinational Medical Unit at Kandahar Airfield, Afghanistan, from January 2010 to March 2013.

Primary outcome of interest: Glasgow Outcome Scale (GOS) score at 6, 12, and 24 months from date of injury.
80 patients
• 73% Blast
• 53% treated non-operatively
• Mortality 21% (n=17)
  • 11 presented with an admission GCS score ≤ 5, and in the remaining 6 cases, no admission GCS score was recorded.
• Stratifying patients into two admission GCS groups shows statistically different outcomes at all follow-up time points

![Graph showing mean GOS scores over time stratified by admission GCS score. Error bars indicate SDs. *p < 0.05.]

TABLE 1. Demographic and clinical characteristics and mortality data for the 80 patients in the study cohort

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years, mean (range)</td>
<td>24.9 (19–40)</td>
</tr>
<tr>
<td>Service, n (%)</td>
<td></td>
</tr>
<tr>
<td>Army</td>
<td>49 (61%)</td>
</tr>
<tr>
<td>Navy</td>
<td>2 (3%)</td>
</tr>
<tr>
<td>Air Force</td>
<td>6 (8%)</td>
</tr>
<tr>
<td>Marine Corps</td>
<td>23 (28%)</td>
</tr>
<tr>
<td>Mechanism of injury, n (%)</td>
<td></td>
</tr>
<tr>
<td>Blast</td>
<td>58 (73%)</td>
</tr>
<tr>
<td>GSWH</td>
<td>22 (28%)</td>
</tr>
<tr>
<td>Admission GCS score, n = 64, mean (SD)</td>
<td>8.5 (5.56)</td>
</tr>
<tr>
<td>6–15</td>
<td>33 (52%)</td>
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<tr>
<td>3–5</td>
<td>31 (48%)</td>
</tr>
<tr>
<td>Admission ISS, mean (SD)</td>
<td></td>
</tr>
<tr>
<td>Mild to moderate, ISS ≤ 25, n (%)</td>
<td>35 (44%)</td>
</tr>
<tr>
<td>Severe to critical, ISS ≥ 26, n (%)</td>
<td>45 (58%)</td>
</tr>
<tr>
<td>Injury characteristics, n (%)</td>
<td></td>
</tr>
<tr>
<td>Epidural hematoma</td>
<td>5 (6%)</td>
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<tr>
<td>Subdural hematoma</td>
<td>24 (30%)</td>
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<tr>
<td>Subarachnoid hemorrhage</td>
<td>35 (44%)</td>
</tr>
<tr>
<td>Intraparenchymal hemorrhage</td>
<td>60 (75%)</td>
</tr>
<tr>
<td>Intraventricular hemorrhage</td>
<td>5 (6%)</td>
</tr>
<tr>
<td>Vascular injury</td>
<td>7 (9%)</td>
</tr>
<tr>
<td>Retained fragments</td>
<td>36 (45%)</td>
</tr>
<tr>
<td>Hemiation</td>
<td>15 (19%)</td>
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<tr>
<td>Surgical intervention, n (%)</td>
<td></td>
</tr>
<tr>
<td>No intervention</td>
<td>42 (53%)</td>
</tr>
<tr>
<td>Intervention</td>
<td>38 (47%)</td>
</tr>
<tr>
<td>Craniotomy</td>
<td>8 (11)</td>
</tr>
<tr>
<td>Craniectomy</td>
<td>28 (37)</td>
</tr>
<tr>
<td>ICP monitor/EVD</td>
<td>30 (36)</td>
</tr>
<tr>
<td>Mortality</td>
<td></td>
</tr>
<tr>
<td>At KAF</td>
<td>13 (16%)</td>
</tr>
<tr>
<td>After evacuation</td>
<td>4 (5%)</td>
</tr>
</tbody>
</table>

GCS = Glasgow Coma Scale; GSWH = gunshot wound to the head; ISS = Injury Severity Score; KAF = Kandahar Airfield.
Results

- 908 total neurosurgical cases (over 3+ yr period), 156 US, and 80 of those involved PBI.
- Mean admission GCS score was 8.5 (SD 5.56), and mean admission ISS 26.6 (SD 10.2).
- GOS score for entire cohort trended (BUT NOT STATISTICALLY SIGNIFICANT) toward improvement at each time point (3.6 at 6 months, 3.96 at 24 months, p > 0.05)
  - Subgroup analysis: admission GCS score ≤ 5, gunshot wound as the injury mechanism, admission ISS ≥ 26, and brain herniation on admission CT head were all associated with worse GOS scores at all time points.
  - Excluding those who died, functional improvement occurred regardless of admission GCS score (p < 0.05)
- Overall mortality rate for the cohort was 21%.
  - Mortality rate with GSWH was 41% (9/22 patients) compared to 14% for those who suffered blast PBI
Cases...
- AAM s/p multiple whole body peppering injuries from Apache helicopter → Role2 showed polytrauma with complex scalp laceration → arrives in ED GCS 13, pupils 2-3 mm and reactive, right TM rupture
- Taken to OR for scalp washout/closure and inspection/debridement of skull fracture; small parenchymal contusion and IPH not explored; multiple other surgeries for extremity/torso wounds
- Discharges on HD#4, GCS 15 and GOS 5
Marco

- AAM s/p Suicide vest IED resulting in PBI and managed expectantly in field for unknown period of time → arrives to BAF ED as GCS 3T with reactive pupils b/l, hemodynamically stable and nonoperative abdominal injuries
- Taken immediately to OR for Right frontal EVD and posterior fossa decompression, and debridement of left posterior fossa skull injury evacuation large left cerebellar hematoma and metallic foreign body with expansile fascia lata duraplasty
- Discharged on HD#12 as GCS 15 with multiple lower cranial nerve injuries including dense left facial nerve palsy, left sided hearing loss, severe dysphagia, neurogenic bladder and ataxia and mild left hemiparesis, GOS 3
Marco:
- Multiple F/U appoints out to 3 mo shows continued improvement in all areas of neurologic function: neurogenic bladder resolves and Foley removed, complete eye closure, regains the ability to swallow (PEG removed), ataxia completely resolved. GOS 4
Toph:
- AAM s/p IED blast with multiple penetrating injuries to his head, neck, and extremities → intubated in ER and taken to the OR for craniectomy and neck exploration and globe repair
- PBI to the right hemisphere with posterior right frontal skull fracture, associated SDH and IPH with FB near M2 vessels, GCS 10T with left hemiparesis on admission, left pupil 2-3mm and reactive
- Receives decompressive hemicraniectomy (flap in abdomen) and multiple other surgeries over almost 1 month hospitalization, including cranioplasty prior to discharge with GCS 14 and GOS 4
- No F/U 😞
Long Term Complications of Decompressive Hemicraniectomy

- Syndrome of the trephined
  - Believed to be caused by exposure of the brain to atmospheric pressure → causes distortions of CSF flow, CBF, and changes local neural electrochemistry (seizures)
  - Exacerbated with positional changes → worse when sitting up, relieved when laying down
- Treatment → Cranioplasty
Progression of typical Afghan cranioplasty patient
Combined cases was the rule not the exception
CaptainAmerica:
- AAM s/p IED blast with multiple penetrating injuries to his head, extremities and abdomen → taken for exlap at Role1 with best GCS 3T → attempted to transfer to role 3, but patient ‘declined’ and taken to Role2 for second exlap w abdomen left open → arrives in ED GCS 3T, pupils 4-5 mm and nonreactive
  • Right frontal EVD placed in ICU with good CSF return, R pupil 3mm and reactive, L pupil 3-4 mm and nonreactive
  • ICPs climb to >30 mm Hg over the next 12 hrs despite aggressive CSF drainage and maximal medical therapy
  • Brain death on HD#2
The skull works....sometimes
Debridement...
Soldier:
- AM s/p mounted IED to MRAM suffering polyt trauma with femur fx → taken to Role2 where airway lost, cric attempted and patient coded briefly before ET tube secured, GCS 3T → arrived in ED GCS 3T with PERRL
- Taken to OR for femur fx and CT scanner goes down → reboot reveals concern for right MCA infarct and CTA confirms proximal R MCA occlusion
- Right decompressive hemicraniectomy done with intact brainstem reflexes postop and CCAT to Role4 within 12 hrs arrival at Role3
- At Role4, ICPs manageable x48 hrs, then become refractory → taken back to OR for more debridement and decompression of contralateral hemisphere
- Never regains much neurologic function and family withdraws (death) PID#5
Hank:
- AAM s/p IED Blast with significant polytrauma with operative femur fx → resuscitated at Role1
- Small right SDH (5 mm) and multiple sub-cortical white matter, including corpus callosum IPHs suggestive of diffuse axonal injury as well as traumatic SAH and IVH, 1-2 MLS → GCS 3T with intact brainstem reflexes
- R EVD placed with ICPs 12 mm Hg
- GCS improves to 6/7T (withdraws/localizes) with intact brainstem reflexes
- Has prolonged hospital course with cardiac arrest, renal failure, etc. and ultimately dies HD#18
PBI → High velocity round through face and middle fossa with supra and infratentorial involvement and transverse-sigmoid dural venous sinus injury

• Right decompressive hemicraniectomy with ligation of transverse sinus
Post op course...
Last case requesting transfer before I left......
7 Month service sign out and hand off completed!
Thank you for your attention.
References

2. JTS CPGs: Catastrophic Non-Survivable Brain Injury, Neurosurgery and Severe Head Injury, Traumatic Brain Injury Management in Prolonged Field Care
Infrascanner and Brainscope

Infrascanner – uses infrared technology to detect extravascular hemoglobin

https://infrascanner.com/

Brainscope – uses EEG to assess structural brain abnormalities

https://brainscope.com/

Sarah S. Sanjakdar, PhD
CPT, US Army
Assistant Product Manager, Neurotrauma
1430 Veterans Drive
Fort Detrick, MD 21702-5009
Office: 301-619-2937
Gov phone: 240-877-4208
Cell/WhatsApp: 703-967-9977
NIPR: sarah.s.sanjakdar.mil@mail.mil
SIPR:
sarah.s.sanjakdar.mil@mail.smil.mil
Vestibular/Ocular-Motor Screening (VOMS)

- *Smooth pursuits* – standard EOM exam in vertical and horizontal direction
- *Saccades* – hold fixed positions of fingers in the horizontal and vertical planes with patient instructed to look at each target in single plane as fast as possible
- *Convergence* – focus on finger move to tip of nose
- *Vestibular-ocular reflex (VOR) test* – focus on an object and rotate head in both vertical and horizontal directions at a standard rate
- *Visual motion sensitivity (VMS) test* – stare at thumb and rotate 80 degrees left and right
HEMODYNAMIC CONTROL NOTES

- Do not neglect scalp bleeding! → suture or staple simple scalp lacerations ASAP
- Hypotension is usually not caused by TBI except as a late finding due to herniation → always look for other causes (ongoing bleeding or tension pneumo).
- Urine output (UOP) assesses blood supply to the organs → goal UOP in a polytrauma patient is 30–50mL/h
  - Foley catheter is ideal.
  - If a Foley catheter is not available, monitor by a graduated cylinder.
- TXA in TBI → limited data suggest TXA limits ICH expansion and may improve outcomes in TBI patients. → TXA can be used in TBI patients.
- Colloids (e.g., albumin) have demonstrated a trend toward worsening outcomes in brain-injured patients.
  - Hetastarches are associated with coagulopathy and increased risk of kidney injury in trauma patients.
  - Avoid colloids and hetastarches in TBI patients; however, they can be used if no alternative is available.
  - Avoid hypotonic fluids (including lactated Ringer’s) whenever possible; they can make brain swelling worse.
AIRWAY, OXY/VENT\ MANAGEMENT

- Manually maintain or secure the patient’s airway and avoid hypoxia, hypocapnia, or hypercapnia to reduce the risk of secondary brain injury.
- GCS score is ≤8 or there is facial trauma with compromised airway, a definitive airway is most likely needed → cricothyroidotomy or endotracheal tube [ETT]) that they have the most confidence in placing, based on their training and practice.
  - Airway interventions may cause transient hypoxia during the procedure. Every effort should be made to optimize airway placement on the first attempt by preoxygenating with supplemental O2
  - Patients typically require less sedation after cricothyroidotomy than after ETT placement. This may help conserve resources if medications are limited.
- Gastric decompression with a nasogastric tube (NGT)** or oral gastric tube (OGT) will decrease the risk of aspiration in unconscious patients. If patients required bag-masking, they may have a distended stomach, which, in some patients, contributes to bradycardia.
  - NGT and OGT cannot be placed with a supraglottic airway.
INFECTION CONTROL

● Dress all wounds to prevent further exposure to environmental pathogens and administer antibiotic prophylaxis to all patients with **penetrating** TBI.
  
  ○ Dress all wounds to prevent further introduction of infectious materials. Optimize wound and patient hygiene to the extent possible given the environmental and situational conditions.
  ○ For penetrating head wounds, apply superficial dressings and seal the dressing to the extent possible.
    
    ■ Bleeding head injuries must be sutured or stapled to control bleeding.
    ■ DO NOT introduce any material into the wound cavity.
    ■ DO NOT attempt to flush the wound.

● Antibiotics are not necessary in TBI without open or penetrating trauma.
  
  ○ Ertapenem 1g IV/IO every 24 hours and moxifloxacin 400mg PO every 24 hours for 5 days.
  ○ Ceftriaxone 2 gm IV/IO every 24 hours or cefazolin 2g IV/IO every 8 hours for 5 days.
  ○ Add metronidazole 500mg IV/IO every 8 hours for 5 days for wounds that are grossly contaminated with organic debris (e.g., dirt, debris, clothing).
SEIZURE PROPHYLAXIS AND MANAGEMENT

- Witnessed *convulsive* seizure activity, place patient on side and clear the area of potentially harmful objects. Suction the mouth if possible, but DO NOT attempt to place anything inside a seizing patient’s mouth. Treat any witnessed or suspected seizures with a rapid-acting benzodiazepine.
  - Midazolam 5mg IV/IO/IM every 5 minutes until seizure stops.
  - An alternate benzodiazepine can be used if available (diazepam 5mg IV every 5 minutes until seizure stops; lorazepam 4mg IV every 5 minutes until seizure stops).
  - Add maintenance antiepilepsy drug. (IV levetiracetam (Keppra 2000mg IV/IO loading dose over 15 minutes) followed by maintenance dosing of 500mg IV/IO every 12 hours.
  - Alternate maintenance antiepilepsy drugs:
    - Phenytoin (loading dose: 1.5g IV over 1 hour, then 100mg PO/IV/IO every 8 hours)
    - Phenobarbital (loading dose: 1.5g IV/IO over 1 hour, then 100mg PO/IV/IO daily). Be ready to support ventilation if phenobarbital is used.

- Non-convulsive seizures (NCSs) should be considered in any TBI patient with a GCS score ≤8 and who does not improve with appropriate resuscitation and/or ICP management
FEVER CONTROL

● Maintain core temperature between 96°F and 99.5°F. Treat fever aggressively in TBI patients with a combination of medication, cold fluid boluses, and surface cooling techniques.
  ○ Ensure patient has been removed from heat or sun. Remove clothes to allow evaporative cooling. Use surface-cooling measures (e.g., evaporative heat loss by misting and fan cooling) to reduce core body temperature.
  ○ Apply cold packs to axillary regions, posterior cervical region, and the groin.
  ○ Acetaminophen 650mg every 4 hours orally (PO) or rectally as needed for rectal temperatures >99.5°F. Additionally, cold saline IV fluid bolus can be used for refractory fever, if available.

● Fever Notes
  ○ Fever will increase cerebral metabolism and may increase ICP.
  ○ Therapeutic hypothermia should not be attempted in Role 1 setting or in the field
Pharmacotherapy in transport of TBI with suspected increased ICPS

- Unconscious patients may experience pain and anxiety, manifested by hypertension (i.e., SBP >160mmHg) and/or agitation. Anxiety and agitation can increase ICP. In addition to all minimum measures, ensure adequate sedation and analgesia by targeting a Richmond Agitation and Sedation Score of −1 to −2.
  - Refer to Joint Trauma System CPG on PFC Analgesia and Sedation
    - Ketamine 20mg IV/IO
    - Hydromorphone 0.5–2mg IV/IO
    - Fentanyl 25–50μg IV/IO

- In addition to analgesics, consider administration of a rapid-onset, short-duration anxiolytic.
  - Midazolam 1–2mg IV/IO as needed for agitation or anxiety.
<table>
<thead>
<tr>
<th>Score</th>
<th>Term</th>
<th>Description</th>
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<tbody>
<tr>
<td>+4</td>
<td>Combative</td>
<td>Overtly combative, violent, immediate danger to staff</td>
</tr>
<tr>
<td>+3</td>
<td>Very agitated</td>
<td>Pulls or removes tube(s) or catheter(s), aggressive</td>
</tr>
<tr>
<td>+2</td>
<td>Agitated</td>
<td>Frequent nonpurposeful movement, fights ventilator</td>
</tr>
<tr>
<td>+1</td>
<td>Restless</td>
<td>Anxious but movements not aggressively vigorous</td>
</tr>
<tr>
<td>0</td>
<td>Alert and calm</td>
<td>Not fully alert but has sustained awakening (eye opening/eye contact to voice ≥10 seconds)</td>
</tr>
<tr>
<td>-1</td>
<td>Drowsy</td>
<td>Briefly wakes to voice with eye contact (&lt;10 seconds)</td>
</tr>
<tr>
<td>-2</td>
<td>Light sedation</td>
<td>Movement or eye opening to voice (but no eye contact)</td>
</tr>
<tr>
<td>-3</td>
<td>Moderate sedation</td>
<td>No response to voice but movement or eye opening to physical stimulation</td>
</tr>
<tr>
<td>-4</td>
<td>Deep sedation</td>
<td>No response to voice or physical stimulation</td>
</tr>
<tr>
<td>-5</td>
<td>Unarousable</td>
<td>No response to voice or physical stimulation</td>
</tr>
</tbody>
</table>
Aeromedical Evacuation of the Neurosurgical patient

Transportation away from Role 3

Aeromedical evacuation considerations
- Do not discontinue a post-operative drain in the immediate period prior to aeromedical evacuation
- Due to stresses of flight, patients with marginal ICP measurements require observation in theater or further decompression
- Avoid hyperthermia during transport; temperatures > 99F may increase risk for vasospasm
- Head of bed should be elevated 30-45 degrees unless T12-L5 fracture is present
  - Reverse trendelenberg for patients requiring spine immobility

ICP monitoring is recommended for the following:
- Patients that cannot be awakened hourly for neurologic evaluation
- Patients who have ongoing resuscitative requirements and an intracranial lesion or the potential for developing cerebral edema
- Pneumocephalus The effects of altitude will potentially result in expansion of air in the calvarium. Coordinate with CCATT, particularly those who have not undergone decompression prior to flight.

Begin DVT prophylaxis in all patients on POD #1 with enoxaparin 30mg bid UNLESS:
- Hemorrhagic complication (increased blood seen on follow-up CT scan)
- Prohibitive contraindication for bleeding risk (high-grade liver injury with ongoing coagulopathy
- Planned re-operation; hold enoxaparin 24-36 hours prior to surgery
SODIUM MANAGEMENT

● Avoid *hyponatremia*, which can worsen brain swelling. The target serum sodium level in patients with severe TBI is slightly above normal, between 145mmol/L and 160mmol/L.
  ○ Avoid the administration of any free water or hypotonic fluids that will lower serum sodium levels.
  ○ Monitor serum sodium level via blood sampling. In a stable patient, check sodium level every 6 hours. In an unstable patient, or in a patient receiving HTS, check sodium level every 3 hours. Adjust fluids as needed to meet the sodium goals.

● Sodium Management Notes
  ○ Several conditions can develop rapidly in brain-injured patients that can lower serum sodium levels (e.g., cerebral salt wasting, syndrome of inappropriate antidiuretic hormone secretion) or raise serum sodium levels (e.g., diabetes insipidus). Sodium levels, fluid intake, and urine output should be monitored closely.
If laboratory testing for serum sodium level is not available.....

- 250mL of 3% saline expected to raise the serum sodium level of an 80kg patient approximately 2–3mmol/L.
- Assuming a normal serum level of 140mmol/L before starting HTS therapy, it would take six 250mL bags of 3% HTS to raise the serum sodium to concerning levels (i.e. >160mmol/L).
  - This is without factoring in the regulation of serum sodium by the kidneys. If patient is urinating, it will be difficult to raise serum sodium above 160mmol/L with 3% HTS.
  - If patient is not urinating, more caution should be used because sodium levels can build up more quickly.
JTS CPG:
Neurosurgery and Severe Head Trauma In Theatre

Specific cases for surgery:
- **EDH:**
  - OR if > 30cc regardless of GCS
  - < 30ccs, <1.5cm thick, < 5mm midline shift (MLS), GCS >8 without focal deficit → consider observation
- **SDH:**
  - Craniectomy/otomy for evac with thickness > 1cm, or MLS > 5mm
  - Host nationals with GCS < 8 → consider expectant.
- **Traumatic Parenchymal lesions:**
  - Craniectomy/otomy for evacuation of hematoma in patients with GCS 6-8 with frontal and temporal contusions greater than 20cc and MLS of at least 5mm, and cisternal compression.
  - >50cc in volume in a salvageable patient
Background and epidemiology TBI

- TBI definition → “brain damage resulting from external forces, as a consequence of direct impact, rapid acceleration or deceleration, a penetrating object (e.g., gunshot) or blast waves from an explosion. The nature, intensity, direction and duration of these forces determine the pattern and extent of damage.”
- World Health Organization (WHO) forecasts that by 2030, TBI will be a leading cause of disability and death worldwide
  - Estimated that TBI accounts for 9% of deaths around the world
- Gun shot wounds (GSW) to head have >90% mortality rate in civilian cohorts
Pathophysiology of TBI

- **Primary injury** – occurs at the time of injury and results in irreversible damage to brain tissue.
  - Not very modifiable…wear helmets….avoid being Afghanistan…..

- **Secondary injury** – follows the primary injury and results in complex inflammatory cascade → rapid development of brain swelling → rise in intracranial pressure ICP → decrease in cerebral perfusion → can lead to massive swelling, compression of the brainstem, and, ultimately, death
  - Primary focus of TBI management is on limiting the effects of secondary brain injury.

"On a long enough time line, the survival rate for everyone drops to zero."
– Chuck Palahniuk (American author), Flight Club

Ho, Anthony. et. al. (2013). Understanding immortal time bias in observational cohort studies. Anaesthesia. 68. 126-130.