

JOINT TRAUMA SYSTEM CLINICAL PRACTICE GUIDELINE (JTS CPG)



Traumatic Brain Injury Management in Prolonged Field Care

This CPG provide medical professionals who encounter traumatic brain injury (TBI) in austere environments with evidence-based guidance.

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Rapid Update Apr 2024: Levetiracetam dosing (1500 mg loading dose, 1000 mg BID), PaCO₂ (35-45 mmHg), SpO₂ (>93%), and EtCO₂ (35-45 mmHg) parameters updated to match parameters in Traumatic Brain Injury Management and Basic Neurosurgery in the Deployed Environment CPG.

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INTRODUCTION

This Role 1, prolonged field care (PFC) guideline is intended to be used after Tactical Combat Casualty Care (TCCC) Guidelines when evacuation to a higher level of care is not immediately possible. A provider of PFC must first be an expert in TCCC. This clinical practice guideline (CPG) is meant to provide medical professionals who encounter traumatic brain injury (TBI) in austere environments with evidence-based guidance.

Recommendations follow a “best, better, minimum” format that provides alternate or improvised methods when optimal hospital options are unavailable. Refer to the [JTS Traumatic Brain Injury and Neurosurgery in the Deployed Environment, 15 Sep 2023 CPG](#) for more comprehensive guidelines for TBI management

TBI occurs when external mechanical forces impact the head and cause an acceleration/deceleration of the brain within the cranial vault which results in injury to brain tissue. TBI may be closed (blunt or blast trauma) or open (penetrating trauma).¹ Signs and symptoms of TBI are highly variable and depend on the specific areas of the brain affected and the injury severity. Alteration in consciousness and focal neurologic deficits are common. Various forms of intracranial hemorrhage (ICH), such as epidural hematoma, subdural hematoma, subarachnoid hemorrhage, and hemorrhagic contusion can be components of TBI. The vast majority of TBIs are categorized as mild and are not considered life threatening; however, it is important to recognize this injury because if a patient is exposed to a second head injury while still recovering from a mild TBI, they are at risk for increased long-term cognitive effects. Moderate and severe TBIs are life-threatening injuries.

Prompt evaluation and intervention are necessary to reduce disability and mortality. Rapid evacuation and neurosurgical evaluation, while desirable, are not always feasible in austere environments. Nevertheless, recent data from the conflicts in Iraq and Afghanistan have shown improved mortality among military TBI casualties when compared with similar, propensity score–matched civilian TBIs. This is due partly to the aggressive resuscitation that began at the point of injury.² PFC providers, therefore, should be prepared to use resources at hand for aggressive medical management in these patients until additional medical and surgical assets can be made available.

Regardless of mechanism, two categories of injury occur with TBI: primary and secondary. Primary injury occurs at the time of injury and results in irreversible damage to brain tissue.

There are no effective treatments for primary injury. Secondary injury, in contrast, occurs as a result of a complex inflammatory cascade that results in rapid development of brain swelling, rise in intracranial pressure, and subsequent decrease in cerebral perfusion. When severe, this can lead to massive swelling, compression of the brainstem, and, ultimately, death. Thus, the primary focus of TBI management is on limiting the effects of secondary brain injury. The brain possesses minimal cellular oxygen reserve and, therefore, is highly dependent on a continuous supply of oxygenated blood. A systolic blood pressure (SBP) <90mmHg or oxygen saturation via pulse oximetry (SpO₂) <90% more than doubles the risk of death from brain injury.³ The management of hypotension, hypoxia, hypocarbia or hypercarbia, hypoglycemia, and signs of elevated intracranial pressure (ICP) is essential.



Telemedicine – Management of TBI is complex. Establish a telemedicine consultation as soon as possible.

NEUROLOGIC ASSESSMENT

Goal: Rapidly identify the clinical signs and symptoms of TBI and associated traumatic injuries and assess TBI severity.

Track the progression of brain injury over time and be vigilant for the early signs of rising ICP: worsening headache, focal neurologic deficits, and declining neurologic examination.

- **Primary survey:** Perform a rapid trauma survey to assess all injuries. Determine and record the Glasgow Coma Scale (GCS) score (Table 1 below). 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 (Table 2 below), and perform an initial detailed neurologic examination. See [Appendix A](#) for further details on performing a neurologic examination. Annotate findings on the PFC flowsheet.
- **TBI severity classification** using the GCS score⁴:
 - Mild: 13–15
 - Moderate: 9–12
 - Severe: 3–8

Table 1. Glasgow Coma Scale


Eye Opening	Verbal Response	Motor Response
4 – Spontaneous	5 – Oriented	6 – Obeys commands
3 – To verbal command	4 – Confused	5 – Localizes to painful stimuli
2 – To painful stimuli	3 – Inappropriate words	4 – Withdraws from pain
1 – No response	2 – Incomprehensible sounds	3 – Flexion to pain
	1 – No response	2 – Extension to pain
		1 – No response

Table 2. Features Indicative of Moderate to Severe Head Injury

Red Flags
Witnessed loss of consciousness
Two or more blast exposures within 72 hours
Unusual behavior or combative
Unequal pupils
Seizures
Repeated vomiting
Double vision or loss of vision
Worsening headache
Weakness on one side of the body
Cannot recognize people or disoriented to place
Abnormal speech

NEUROLOGIC ASSESSMENT NOTE

An emerging technology that can be considered as an adjunct to neurologic assessment is ultrasound measurement of optic nerve sheath diameter (ONSD). If the patient is unconscious (i.e. does not follow commands or open eyes spontaneously), measure a baseline ONSD. There is no definite diameter that is diagnostic of increased ICP; however, an ONSD >5.2mm, especially if it increases over time, may indicate elevated ICP.⁵ In no circumstance should measurement of ONSD take priority over a neurologic examination, and all results must be considered in the context of the neurologic examination and overall patient status. See [Appendix B](#) for further details on using ultrasound to obtain and interpret ONSDs.

 ONSD should NOT be attempted in any patient who has sustained an open globe injury where any pressure on the globe is contraindicated. As many as 30% of head injuries in combat may also have an eye injury.⁶


MONITORING

Goal: Prevent secondary brain injury by maintaining adequate oxygenation and ventilation, avoiding hypotension, observing for signs and symptoms of elevated ICP, and trending the response to resuscitation. Detect changes in vital signs and neurologic examination as early as possible.

- **Minimum:** Blood pressure cuff, stethoscope, pulse oximeter, method to monitor urine output. If an advanced airway is in place, monitor EtCO₂ with capnometer. Check pupillary response and GCS as often as possible. Document vital signs, GCS score, and urine output on the PFC Casualty Card available at <https://prolongedfieldcare.org>.
- **Best:** Portable monitor providing continuous vital signs display, Foley catheter to monitor urine output. If an advanced airway is in place, monitor end-tidal carbon dioxide (EtCO₂) with capnography. Check pupillary response and GCS score every hour. Document vital signs, GCS, and urine output on the PFC Casualty Card available at <https://prolongedfieldcare.org>.

ASSESSMENT AND MONITORING NOTES

- Perform an initial assessment according to TCCC/MARCH (Massive hemorrhage, Airway, Respirations, Circulation, Head injury/Hypothermia) algorithms.
- Severe head injury is associated with additional trauma in 60% of patients.⁷
- If ONSD is used to evaluate for increased ICP and response of ICP to interventions, repeated ultrasound examinations should be performed if there is any change in neurologic examination and at regular intervals (30 minutes) after performing ICP-lowering interventions.
- When possible, a pocket ophthalmoscope can be used to assess for the presence or absence of spontaneous venous pulsations (SVPs). SVPs are only present when ICP is normal. Visualization of SVPs can reassure the provider that ICP is not critically elevated.⁸ See [Appendix C](#) for additional information on the rapid assessment of SVPs.
- Consider early C-spine immobilization. The incidence of concomitant brain and spinal cord injury in trauma ranges from 25% to 60%, with motor vehicle crashes and falls having the highest incidence of co-occurrence.⁹ Ensure the cervical collar does not compress the jugular veins in the neck, because that could worsen ICP.
- The neurologic examination is essential to identify deterioration in a TBI patient. Treat for elevated ICP for any deterioration in neurologic examination findings.
- Pain medication and sedation are usually required for TBI patients; however, these medications also make it difficult to follow the neurologic examination.
- Obtain a telemedicine consultation if possible. If in doubt, treat for elevated ICP according to ICP management outlined in the next section.
- Close control of EtCO₂ is critical for severe TBI patients. Plan and ensure the capability to monitor EtCO₂ is available whenever advanced airway is placed. Goal EtCO₂ is 35-45mmHg.

 Cushing's triad (i.e., increased SBP and/or widening pulse pressure, bradycardia, and irregular respirations) is a physiologic response that can occur with elevated ICPs, resulting in medullary compression. It is a late finding of severe brain injury with brainstem herniation. Cushing's triad should be viewed as a sign of cerebral herniation and addressed immediately when recognized (see ICP Management).

MANAGEMENT

HEMODYNAMIC CONTROL

Goal: Maintain SBP >110mmHg. In polytrauma patients with ongoing bleeding, aggressively control hemorrhage using all means available and restore circulating blood volume by using blood products.

Note: An SBP >90mmHg has traditionally been targeted in TBI patients, though recent literature has suggested better outcomes may occur when SBP is maintained above 110mmHg in TBI patients.^{10,11}

- **Minimum:** Stop all external bleeding. Manage internal bleeding to the extent possible with available resources. Administer tranexamic acid (TXA) per TCCC guidelines. Avoid medications that may lower the blood pressure (e.g., narcotics).
- **Better:** If there is evidence of bleeding and no blood products are available, administer 1L 0.9% sodium chloride (NaCl). Target is an SBP >110mmHg.
- **Best:** If there is evidence of bleeding, transfuse whole blood or, if not available, transfuse blood products. Greater emphasis on the use of Low Titer O Whole Blood is the optimal strategy. Target is an SBP >110mmHg.

HEMODYNAMIC CONTROL NOTES

- Do not neglect scalp bleeding. This can become a significant source of blood loss. Scalp lacerations should be sutured or stapled as soon as possible.
- Take caution if an underlying skull fracture is present or if there is obvious penetrating trauma. DO NOT tightly pack or irrigate an open head wound. Suture or staple the skin closed, if bleeding. A pressure dressing may be placed if needed to control bleeding.
- Hypotension is usually not caused by TBI except as a late finding due to herniation.¹² Always look for other causes of hypotension, such as ongoing bleeding or tension pneumothorax.
- Urine output (UOP) provides an important assessment of blood supply to the organs. Monitoring continuously by a Foley catheter is ideal. If a Foley catheter is not available, monitor by a graduated cylinder. Goal UOP in a polytrauma patient is 30–50mL/h.¹³
- The role of TXA in TBI patients is currently under investigation in the CRASH-3 trial. Limited data suggest TXA limits ICH expansion and may improve outcomes in TBI patients.¹⁴ Until more definitive data are available, TXA can be used in TBI patients.
- Colloids (e.g., albumin) have demonstrated a trend toward worsening outcomes in brain-injured patients.¹⁵ 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.



Most trauma patients with moderate or severe TBI will have other traumatic injuries. A careful search for bleeding should be performed in any hypotensive trauma patient.



Brain injury with associated hemorrhagic shock is a complicated scenario with a high risk of death. Balancing hemorrhage control (which is easier with lower blood pressure) with maintaining cerebral perfusion pressure (which requires higher blood pressure) should be guided with expert teleconsultation (i.e. critical care, neurocritical care, neurosurgical) whenever possible.

AIRWAY, OXYGENATION/VENTILATION MANAGEMENT

Goal: Manually maintain or secure the patient's airway and avoid hypoxia, hypocapnia, or hypercapnia to reduce the risk of secondary brain injury. If GCS score is ≤ 8 or there is facial trauma with compromised airway, a definitive airway is most likely needed. The provider should place the type of airway (i.e. cricothyroidotomy or endotracheal tube [ETT]) that they have the most confidence in placing, based on their training and practice.

- **Minimum:** Nasopharyngeal airway and bag-valve-mask with PEEP valve as needed. Use supplemental oxygen, if available. Maintain SpO₂ >93%.
- **Better:** Perform a cricothyroidotomy/ETT placement or place a supraglottic airway (e.g., laryngeal mask airway [LMA], King laryngeal tube [LT]) followed by continuous sedation and airway maintenance, supplemental oxygen via oxygen concentrator, and portable ventilator to maintain an SpO₂ >95% and EtCO₂ of 35-45mmHg.
- **Best:** Cricothyroidotomy or ETT followed by continuous sedation and airway maintenance, supplemental oxygen, portable ventilator. Targets: SpO₂ >95% and EtCO₂ 35-45 mmHg. Check arterial blood gas results and correlate with EtCO₂ within 30 minutes of intubation if laboratory capability is available. A positive end-expiratory pressure (PEEP) of 5cmH₂O should be used routinely. PEEP can be safely increased up to 15cmH₂O if needed to improve SpO₂, but be alert for problems caused by increased intrathoracic pressure (e.g., lower blood pressure or increased ICP).¹⁶

In the JTS Traumatic Brain Injury Management and Basic Neurosurgery in the Deployed Environment CPG, a goal of >93% is recommended, However, in order to establish a safety buffer in the prolonged field care setting often characterized by equipment challenges and limited personnel, in addition to anticipated transport challenges, a goal SpO₂ of > 95% for ventilated patients is recommended.

AIRWAY MANAGEMENT NOTES

- Patients with a GCS score ≤ 8 should undergo placement of an advanced airway (i.e. cricothyroidotomy or ETT) unless arrival to a higher level of care will occur in a timely manner or the airway can be manually maintained. The risk versus benefit of advanced airway placement should be carefully considered and discussed by telemedicine consultation whenever possible.
- 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, selecting the most experienced provider available, and using the technique most familiar to the provider.
- Patients typically require less sedation after cricothyroidotomy than after ETT placement. This may help conserve resources if medications are limited.
- Monitor EtCO₂ and adjust ventilations to achieve the target range. Avoid hyperventilation (EtCO₂ <35mmHg) except in extreme cases where imminent herniation is suspected, because hyperventilation worsens cerebral ischemia. Also avoid hypoventilation (EtCO₂ >45 mmHg) that will increase ICP.
- 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.

ICP MANAGEMENT

Goal: Suspect high ICP in any head injury patient with GCS score ≤ 8 OR declining findings on neurologic examination (unless explained by sedation, hypotension, hypoxia, hypercarbia, high fever). Minimize factors that contribute to elevated ICP, such as pain, anxiety, and fever. Rapidly recognize and manage elevated ICP, and maintain an adequate cerebral perfusion pressure.

- **Minimum:** Use general measures to reduce ICP.
 - Elevate head of bed (HOB) 30°–60°.
 - Maintain neck in midline position.
 - Maintain arterial blood oxygen saturation (SpO₂) >93% (or >95% if receiving ventilatory support.)
 - Maintain EtCO₂ between 35mmHg and 45mmHg
 - Maintain core temperature between 96°F and 99.5°F.
 - Maintain SBP >100mmHg, ideally at >110mmHg.
 - Prevent or rapidly manage seizure activity.
 - If concerned for impending herniation (e.g., unresponsive patient with unilateral dilated pupil, presence of Cushing’s triad), ventilate the patient for no more than 20 minutes to an EtCO₂ target of 35mmHg¹⁷ Seek expert consultation immediately.

The optimum duration of hyperventilation and frequency that can be repeated are not known. If performed, assess response (i.e. pupils, GCS score, and so forth). If patient responds, consider performing again if needed, guided by expert teleconsultation, if possible

- **Better:** Even 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 the JTS [PFC Analgesia and Sedation CPG](#).¹⁸
 - 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.

- **Best:** In addition to all minimum and better measures, administer osmotic therapy via peripheral intravenous (IV) or intraosseous (IO) access:
 - Hypertonic saline (HTS): 3% NaCl 250mL bolus over 20 minutes; repeat every 3 hours as needed when concerned for elevated ICP.
 - Mannitol can be used if there is no sign of bleeding and the SBP is >110mmHg. Mannitol 1g/kg IV/IO over 20 minutes. Repeat at 0.5g/kg IV/IO every 3 hours as needed when concerned for elevated ICP.




Seek additional medical direction as soon as possible and evacuate to neurosurgical care at the earliest opportunity.


ICP MANAGEMENT NOTES


- ICP cannot be directly measured without advanced intracranial monitoring devices. Therefore, vigilant clinical observation and the use of noninvasive ICP assessment modalities are critical to monitoring TBI patients until neurosurgical placement of neuromonitoring devices can occur.
- HTS bolus lowers ICP and has a duration of action of approximately 3 hours.^{19,20}
- Mannitol, although effective, has several potentially adverse complications. It is a diuretic and might lower the blood pressure. Also, after repeated use, it can cross a damaged blood–brain barrier and potentially worsen ICP.²⁰ For these reasons, HTS is preferred to mannitol in TBI and polytrauma patients.

- Some institutions have reported ICP-lowering benefits from vertical positioning of patients, particularly when high intraabdominal or intrathoracic pressure is suspected. Lower intraabdominal and intrathoracic pressures may facilitate venous drainage from the intracranial compartment. If safe to do so, this can be attempted when other measures have failed.²¹

 Always treat hypotension before treating elevated ICP. Cerebral blood flow is more affected by a decrease in blood pressure than an increase in ICP.

Cerebral perfusion pressure (CPP) = mean arterial pressure (MAP; mmHg) – ICP (mmHg)

 Hyperventilation reduces CO₂ and rapidly lowers ICP by causing cerebral vasoconstriction and decreasing the overall cerebral blood volume. However, hyperventilation also damages the brain by causing ischemia and should only be performed for brief periods. **Avoid hyperventilation** unless all other interventions have been ineffective.²²

 Although there are invasive interventions to help assess and treat elevated ICP, evacuate hematomas, and so forth, such as decompressive craniectomy, extra ventricular drains, intracranial bolt monitors, and burr holes, such procedures are not recommended unless the PFC provider has training and experience in performing these procedures and is directed by expert teleconsultation.

INFECTION CONTROL

Goal: Dress all wounds to prevent further exposure to environmental pathogens and administer antibiotic prophylaxis to all patients with penetrating TBI.

- Minimum:** 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.
- Better:** Antibiotics should be used for open or penetrating TBI.
 - Ertapenem 1g IV/IO every 24 hours and moxifloxacin 400mg PO every 24 hours for 5 days.

Ertapenem and moxifloxacin may increase the risk of seizure and ertapenem may not penetrate an intact blood-brain barrier. This combination, while commonly available, should only be used in TBI patients when the antibiotics with proven CNS penetration are not available.

- Best:** Administer CNS-penetrating antibiotics for open or penetrating TBI.
 - 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).¹⁷

INFECTION CONTROL NOTES

- Moxifloxacin may be replaced with levofloxacin 750 mg PO daily to provide better coverage of bacteria found in wet terrain/ jungle environment.
- If recommended antibiotics are not available or significant drug allergies are present, obtain teleconsultation to discuss alternative medications. Additional detailed recommendations may be found in Forgiione, et al.²³

SEIZURE PROPHYLAXIS AND MANAGEMENT

Goal: Rapidly identify and manage seizure activity in TBI patients.

- **Minimum:** For witnessed convulsive seizure activity, place the patient on his/her side and clear the area of any 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).
- **Best:** For witnessed or suspected seizures, administer a rapidly acting benzodiazepine (midazolam 5mg IV/IO/intramuscularly [IM]) plus a maintenance antiepilepsy drug. Broad-spectrum IV agents such as levetiracetam (Keppra; UCB Pharma, <http://www.ucb.com/>) are preferred.
 - Maintenance antiepilepsy-drug dosing: Levetiracetam: 1500mg IV/IO loading dose over 15 minutes followed by maintenance dosing of 1000mg 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.

SEIZURE NOTES

- Not all seizures are easy to see. At times, the findings may be obvious with generalized convulsions, or they may be subtle (e.g., persistent twitching of facial muscles, fingers).
- Risk factors for seizures after TBI include: GCS score <10, skull fractures, penetrating injuries, prolonged length of coma (>24 hours).²⁴
- 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. NCSs may persist after convulsive seizures are stopped and may be associated with higher morbidity and mortality.²⁵ The most common signs of patients with NCS are coma, delirium, agitation, aphasia (impairment of language affecting production and/or comprehension of speech, reading, and/or writing) and/or "blank staring."
- Prompt initiation of seizure prophylaxis reduces early seizures after TBI.²⁶ In PFC settings, where possible, an anti-epilepsy drug should be used early after injury to help prevent seizure.
- Midazolam has a high rate of seizure control and works rapidly to terminate seizure activity.²⁷ Midazolam is preferred because it is a short-acting medication (elimination time: 2–4 hours) and will allow for more regular and comprehensive neurologic examinations. It can be given IM in patients who do not yet have IV/IO access.

- If not already placed, strong consideration should be given to placing an advanced airway (cricothyroidotomy or ETT) in any TBI patient who experiences seizures (place airway after seizures are controlled).


FEVER CONTROL

Goal: 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.

- **Minimum:** 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.
- **Better:** Apply cold packs to axillary regions, posterior cervical region, and the groin.
- **Best:** 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.
- Although targeted temperature management (previously referred to as therapeutic hypothermia) is used to reduce ICP in a critical care setting, hypothermia is part of the “lethal triad” in trauma patients, along with coagulopathy and acidosis. Targeted temperature management strategies beyond what is outlined in TCCC should NOT be attempted in the field or Role 1 setting.
- Hypothermia prevention and management kits should continue to be used in all trauma patients. In TBI patients, however, warming measures should be avoided when the core body temperature is above the target range.

 Avoid non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen, naproxen, ketorolac. Although these agents can effectively lower temperature, their antiplatelet effect may increase bleeding in TBI if intracranial hemorrhage is present (e.g., epidural hematomas, subdural hematomas).

SODIUM MANAGEMENT


Goal: 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.

- **Minimum:** Avoid the administration of any free water or hypotonic fluids that will lower serum sodium levels.
- **Best:** 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, then, as a reference, 250mL of 3% saline can be 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.

-  Obtain telemedicine consultation, preferably from a critical care or neurocritical care expert, before giving more than two 250mL boluses of 3% NaCl HTS.

BLOOD GLUCOSE CONTROL

Goal: Avoid both hypoglycemia and hyperglycemia. Target a blood glucose level of 180mg/dL via handheld glucometer.

- **Minimum:** Monitor for clinical signs and symptoms of hypoglycemia (e.g., sweating, confusion, tremor, generalized weakness, generalized lethargy). If patient is hungry and able to safely swallow, allow the patient to eat to avoid hypoglycemia. Avoid the administration of any substances that are excessively high in sugar or carbohydrate content to prevent hyperglycemia.
- **Best:** Check blood glucose level every 6 hours. If glucose level is <100mg/dL, give 20g of oral glucose (5 teaspoons of sugar or 4 teaspoons of honey) PO or by NG tube. Or administer 25g (50mL) dextrose 50% in water (D50) solution IV/IO. Recheck blood glucose in 1 hour, then continue to check every 6 hours.

BLOOD GLUCOSE CONTROL NOTES

- Hypoglycemia is more harmful to the brain than hyperglycemia.
- Hyperglycemia may occur in TBI as an acute stress response or as a result of brain-induced catecholamine release. Early hyperglycemia (i.e., >180mg/dL) has been associated with poor neurologic outcome in severe TBI.²⁸
- The treatment of hyperglycemia requires insulin. If the appropriate medications, laboratory capability, and expertise are available, hyperglycemia may be treated; however, the treatment of hyperglycemia is not covered in this CPG.

TRANSPORTATION CONSIDERATIONS

Goal: Safely prepare TBI patients for ground and/or air transportation to higher levels of care.

- **Minimum:** Dose sedative/analgesic medications, osmotic agent, and/or benzodiazepine before any significant ground or air transportation, as resources permit.

Neuromuscular blocking agents will mask seizures and clinical examination changes; therefore, the risk versus benefit of use during transport must be considered.

- **Best:** Ensure transport assets are aware of the patient's brain injury in advance so appropriate accommodations and/or alterations to the travel plan can be made.
 - Most patients are transported in a supine position. Every effort should be made to transport TBI patients with the HOB elevated to at least 30°.
 - For fixed-wing transport, TBI patients should be loaded with their head toward the front of the aircraft to minimize G-forces transmitted to the brain.

- Air transport of TBI patients requires additional considerations. For air movements that involve altitudes >8,000 ft, TBI patients are at risk for additional complications that may worsen brain injury, such as hypobaric hypoxia and high-altitude cerebral edema.²⁹ Preplanning with air assets is highly recommended.
- Recommended Packing List. See [Appendix D](#).
- Management of Traumatic Brain Injury Summary Table. See [Appendix E](#).

PERFORMANCE IMPROVEMENT (PI) MONITORING

POPULATION OF INTEREST

1. All patients with a diagnosis of traumatic brain injury and an initial GCS of 3-8.
2. All patients who receive a cranial procedure (ICP monitor, craniectomy, craniotomy).

INTENT

1. All patients in population of interest avoid hypotension and hypoxia: SBP never < 100 mmHg, MAP never < 60, SaO₂ never < 93%.
2. All patients in population of interest have PaCO₂ monitored at every role of care.
3. All patients in population of interest have a head CT performed within 4 hours of injury.
4. All patients with a ventriculostomy have hourly documentation of ICP/ CPP and ventriculostomy output.
5. Patients in population of interest unable to be monitored clinically (e.g., unable to hold sedation for Q1 hour neuro exam) have an ICP monitor or ventriculostomy placed prior to transport out of theater.

METRICS

1. Number and percentage of patients in the population of interest with lowest SBP<100 within first 3 days after injury.
2. Number and percentage of patients in the population of interest with MAP<60 within first 3 days after injury.
3. Number and percentage of patients in the population of interest with SaO₂<93% within first 3 days after injury.
4. Number and percentage of patients in population of interest who have PaCO₂ documented at every role of care (POI, POI MEDEVAC, ROLE 2-4, interfacility MEDEVAC).
5. Number and percentage of patients in the population of interest who maintain PaCO₂=35-45mmHg.
6. Number and percentage of patients who had a head CT performed within 4 hours of injury.
7. Number and percentage of patients with a ventriculostomy who had hourly documentation of ICP/ CPP and ventriculostomy output.
8. Number and percentage of patients in the population of interest unable to be monitored clinically (e.g., unable to hold sedation for Q1 hour neuro exam) who have an ICP monitor or ventriculostomy placed prior to transport out of theater.

DATA SOURCE

- Patient Record and the ASIA or Combat Neuro Exam worksheet

- Department of Defense Trauma Registry

SYSTEM REPORTING & FREQUENCY

The above constitutes the minimum criteria for PI monitoring of this CPG. System reporting will be performed annually; additional PI monitoring and system reporting may be performed as needed.

The system review and data analysis will be performed by the JTS Chief and the PI team.

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APPENDIX A: NEUROLOGICAL EXAMINATION

I. MENTAL STATUS

Level of Consciousness: Note whether the patient is:

- Alert/responsive
- Not alert but arouses to verbal stimulation
- Not alert but responds to painful stimulation
- Unresponsive

Orientation: Assess the patient's ability to provide:

- Name
- Current location
- Current date
- Current situation (e.g., ask the patient what happened to him/her)

Language: Note the fluency and appropriateness of the patient's response to questions. Note patient's ability to follow commands when assessing other functions (e.g., smiling, grip strength, wiggling toes). Ask the patient to name a simple object (e.g., thumb, glove, watch).

Speech: Observe for evidence of slurred speech.

II. CRANIAL NERVES

All patients:

- Assess the pupillary response to light.
- Assess position of the eyes and note any movements (e.g., midline, gaze deviated left or right, nystagmus, eyes move together versus uncoupled movements).

Non-comatose patient:

- Test sensation to light touch on both sides of the face.
- Ask patient to smile and raise eyebrows and observe for symmetry.
- Ask the patient to say "Ahhh" and directly observe for symmetric palatal elevation.

Comatose patient:

- Check corneal reflexes; stimulation should trigger eyelid closure.
- Observe for facial grimacing with painful stimuli.
- Note symmetry and strength.
- Directly stimulate the back of the throat and look for a gag, tearing, and/or cough.

III. MOTOR

Tone: Note whether resting tone is increased (i.e. spastic or rigid), normal, or decreased (flaccid).

Strength: Observe for spontaneous movement of extremities and note any asymmetry of movement (i.e. patient moves left side more than right side). Lift arms and legs, and note whether the limbs fall immediately, drift, or can be maintained against gravity. Push and pull against the upper and lower extremities and note any resistance given. Note any differences in resistance provided between the left and right sides.

(NOTE: it is often difficult to perform formal strength testing in TBI patients. Unless the patient is awake and cooperative, reliable strength testing is difficult.)

Involuntary movements: Note any involuntary movements (e.g., twitching, tremor, myoclonus) involving the face, arms, legs, or trunk.

IV. SENSORY

If patient is not responsive to voice, test central pain and peripheral pain.

Central pain: Apply a sternal rub or supraorbital pressure, and note the response (e.g., extensor posturing, flexor posturing, localization).

Peripheral pain: Apply nail bed pressure or take muscle between the fingers, compress, and rotate the wrist (do not pinch the skin). Muscle in the axillary region and inner thigh is recommended. Apply similar stimulus to all four limbs and note the response (e.g., extensor posturing, flexor posturing, withdrawal, localization). *NOTE: In an awake and cooperative patient, testing light touch is recommended. It is unnecessary to apply painful stimuli to an awake and cooperative patient.*

V. GAIT

If the patient is able to walk, observe his/her casual gait and note any instability, drift, sway, and so forth.

APPENDIX B: ULTRASONIC ASSESSMENT OF ONSD

If a patient is unconscious (i.e. does not follow commands or open eyes spontaneously), they may have elevated ICP. There is no reliable test for elevated ICP available outside of a hospital; however, optic nerve sheath diameter (ONSD) measurement is a rapid, safe, and easy-to-perform ultrasonographic assessment that may help identify elevated ICP when more definitive monitoring devices are not available.

- The optic nerve sheath directly communicates with the intracranial subarachnoid space. Increased ICP, therefore, displaces cerebrospinal fluid along this pathway. Normal ONSD is 4.1–5.9mm.³⁰
- A 10–5-MHz linear ultrasound probe can be used to obtain ONSDs. ONSD is measured from one side of the optic nerve sheath to the other at a distance of 3mm behind the eye immediately below the sclera.³¹
- In general, ONSDs >5.2mm should raise concern for clinically significant elevations in ICP in unconscious TBI patients.^{5,32} The ONSD can vary significantly in normal individuals, so one single measurement may not be helpful; however, repeated measurements that detect gradual increases in ONSD over time may be more useful than a single measurement.
- ONSD changes rapidly when the ICP changes, so it can be measured frequently.³³ If ONSD is used, it is best to check hourly along with the neurologic examination.

TECHNIQUE

1. Check to make sure there is no eye injury. A penetrating injury to the eyeball is an absolute contraindication to ultrasound because it puts pressure on the eye.
2. Ensure the head and neck are in a midline position. Gentle sedation and/or analgesia may be necessary to obtain accurate measurements.
3. Ensure the eyelids are closed.
4. If available, place a thin, transparent film (e.g., Tegaderm; 3M, <http://www.3m.com>) over the closed eyelids.
5. Apply a small amount of ultrasound gel to closed eyelid.
6. Place the 10(–5)MHz linear probe over the eyelid. The probe should be applied in a horizontal orientation (Figure 1) with as little pressure as possible applied to the globe.
7. Manipulate the probe until the nerve and nerve sheath are visible at the bottom of screen. An example of a proper ultrasonographic image of the optic nerve sheath can be seen in Figure 2.
8. Once the optic nerve sheath is visualized, freeze the image on the screen.
9. Using the device’s measuring tool, measure 3mm back from the optic disc and then obtain a second measurement perpendicular to the first. The second measurement should cover the horizontal width of the optic nerve sheath (Figure 2). An abnormal ONSD is shown in Figure 3.
10. Repeat the previous sequence in the opposite eye. Annotate both ONSDs on the PFC Casualty Card.
11. ONSDs should be obtained, when possible, at regular intervals to help assess changes in ICP, particularly when the neurologic examination is poor and/or unreliable (i.e. with sedation). Serial measurements with progressive diameter enlargement and/or asymmetry in ONSDs should be considered indicative of worsening intracranial hypertension.

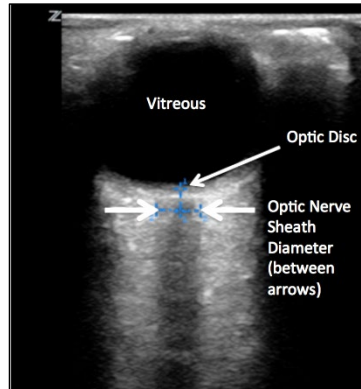
CAUTION: ONSD measurements are contraindicated in eye injuries. NEVER apply pressure to an injured eye.

Figure 1. Appropriate placement of the linear probe.



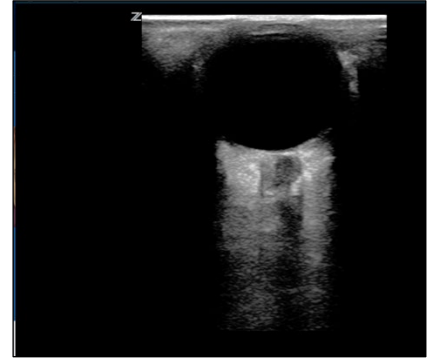
Ultrasound gel is placed over a closed eyelid and the probe placed horizontally over the eyelid, applying as little pressure to the globe as possible. If available, Tegaderm or other thin covering (e.g., Latex glove) should be placed over a closed eyelid for further protection.

Figure 2 An ultrasonographic view of a normal eye and optic nerve sheath.



To measure ONSD, apply the ultrasound measuring device to the optic disc and measure back 3mm along the length of the optic nerve. A second, perpendicular measurement is obtained at the previously measured point that spans the horizontal width of the optic nerve sheath. In this image, ONSD was determined to be 5.1mm, a normal value.

Figure 3. Ultrasound image of the right optic nerve sheath of a 61-year-old man with a traumatic subdural hematoma.



The optic nerve sheath measured 6.8mm in diameter. Elevated ICP was subsequently confirmed (26mmHg) after the placement of an ICP bolt monitor.

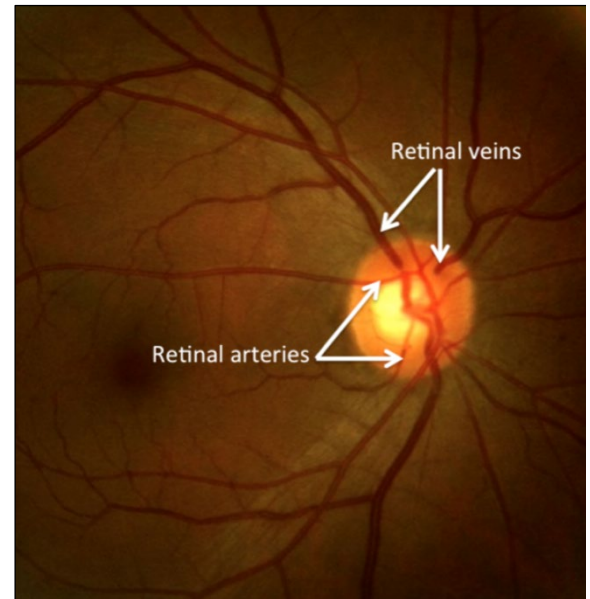
APPENDIX C: SPONTANEOUS VENOUS PULSATATIONS

- Spontaneous venous pulsations (SVPs) are subtle, rhythmic variations in retinal vein caliber on the optic disc and have an association with ICP.
- It is difficult to see SVPs without advanced equipment; however, if a handheld ophthalmoscope is available, it is worth an attempt to visualize the retinal veins.
- Don't worry if you cannot see SVPs; this may actually be normal. However, if you do see them, it is very reassuring that ICP is normal.¹⁰
- If SVPs are initially present and can no longer be seen on subsequent examinations, the provider should be concerned for increasing ICP.

TECHNIQUE

1. Gently lift the eyelid until the pupil is in view.
2. Using a handheld ophthalmoscope, the provider should maneuver himself or herself to a position where the optic disc can be visualized.
3. Identify the retinal veins as they emerge from the optic disc. Retinal veins are typically slightly larger and darker than retinal arteries. Figure 1 demonstrates the typical appearance of the retina.
4. Observe the retinal veins for pulsations. Note the presence or absence of spontaneous venous pulsations
5. Repeat the step 1–4 sequence in the contralateral eye.

Figure 1. Typical appearance of a healthy retina.



The retinal vessels can be seen emerging from the optic disc. Retinal veins can be identified by their slightly larger, thicker size and darker color. Retinal arteries are small, thin, and lighter in color than retinal veins.

APPENDIX D: RECOMMENDED PACKING LIST

Assumptions: One patient with a moderate to severe traumatic brain injury. To calculate the amount of fluid or medication you would need for a single TBI patient, use your worst case, longest possible evacuation extrapolated from your mission planning. For example, if you think you might have a 36-hour evacuation, you might need 4,500mg levetiracetam (a 1,500mg loading dose and 1000mg every 12 hours)

MINIMUM

- **Equipment:** vital-sign trending chart, BP cuff, stethoscope, wristwatch, pulse oximeter, capnometer, cricothyroidotomy kit, bag-valve-mask with PEEP valve, nasopharyngeal airway, disposable thermometer, Nalgene bottle to measure urine
- Medications/Fluids: ketamine, midazolam, lorazepam, acetaminophen, ceftriaxone, 3% hypertonic saline

BETTER

- **Equipment:** portable vital sign monitor, capnometer, cricothyroidotomy kit and/or ETT plus laryngoscope/glidescope and/or laryngeal mask airway, cold/ice packs, graduated cylinder to measure urine, oxygen concentrator
- Medications/Fluids: ketamine, IV hydromorphone, IV fentanyl, midazolam

BEST

- **Equipment:** portable monitor providing continuous vital-signs display with capnography, cricothyroidotomy kit, and/or ETT plus laryngoscope/glidescope, portable point-of care-testing device such as an iStat (Abbott Point of Care; <https://www.pointofcare.abbott>) or Epoch (Alere, <http://www.alere.com>) for arterial blood gas samples, and electrolyte monitoring, blood glucose monitor, Foley catheter kit, and supplemental oxygen or oxygen concentrator
- Medications/Fluids: Fresh whole blood drawing supplies or stored blood products, 3% hypertonic saline, mannitol, ceftriaxone, metronidazole, levetiracetam or phenytoin, acetaminophen, dextrose 50% in water

OTHER PACKING LIST CONSIDERATIONS

- **Equipment:** Portable ultrasound, nasogastric tube, red- (or red/yellow-speckled) top test tubes to test for electrolytes if a host-nation laboratory is available
- Medications/Fluids: TXA

APPENDIX E: MANAGEMENT OF TBI SUMMARY TABLE

Management of Traumatic Brain Injury Summary			
GOAL	MINIMUM	BETTER	BEST
Neurological Assessment			
Goal: Rapidly identify TBI and associated injuries; assess TBI severity.	Primary survey: Perform rapid trauma survey to assess all injuries. Determine and record Glasgow Coma Score. Assess pupils and motor function in all four extremities. Recognize Cushing's triad.	Secondary survey: Evaluate for TBI red flags. Perform detailed neurologic examination.	Consider: If patient is unconscious, measure a baseline optic nerve sheath diameter with ultrasound using a 10(–5)MHz linear probe.
ONSD should not be attempted on any patient with an open globe injury to the eye. Cushing's triad: increased SBP/widened pulse pressure, bradycardia, irregular breathing (typically rapid/shallow)			
Monitoring			
Prevent secondary brain injury, maintain adequate oxygenation and ventilation, avoid hypotension, detect elevated ICP.	Blood pressure cuff, stethoscope, pulse oximeter, method to monitor urine output. If advanced airway is in place, monitor EtCO ₂ with capnometer. Check pupils and GCS hourly or as often as possible.		Portable monitor with continuous vital-signs display, Foley catheter to monitor urine output. If advanced airway in place, monitor end-tidal CO ₂ (EtCO ₂) with capnography. Check pupils and GCS hourly.
Neurologic examination and vital-sign trends are essential to identifying a deteriorating patient with TBI. Monitoring EtCO ₂ is critical for severe TBI patients. Bring the correct equipment whenever possible.			
Management Hemodynamic Control			
Maintain systolic pressure >110mmHg	Stop all external bleeding. Manage internal bleeding to the extent possible with available resources. Administer TXA per TCCC guidelines. Avoid medications that may lower the blood pressure.	If there is evidence of bleeding and no blood products are available, 0.9% sodium chloride 1L. Target SBP: >110mmHg	If evidence of bleeding, transfuse whole blood or, if not available, transfuse blood products per TCCC guidelines. Target SBP: >110mmHg
Brain injury in the presence of hemorrhagic shock requires balancing hemorrhage control with cerebral perfusion. Telemedicine consultation is strongly encouraged. Do not neglect scalp bleeding. Take care to inspect for skull fractures. DO NOT tightly pack an open head wound. Hypotonic fluids (to include lactated Ringer's) should be avoided whenever possible because they can worsen cerebral edema.			
Management: Airway, Oxygenation/Ventilation			
Maintain or secure airway. If GCS score ≤8 or there is facial trauma or other airway compromise, consider definitive airway placement.	Nasopharyngeal airway and bag-valve-mask with PEEP. Supplemental oxygen, if available. Maintain SpO ₂ >93%.	Perform cricothyroidotomy/ETT or place supraglottic airway followed by continuous sedation, supplemental O ₂ , portable ventilator. Target SpO ₂ : >95%, EtCO ₂ : 35-45mmHg	Perform cricothyroidotomy/ETT followed by continuous sedation, supplemental O ₂ , portable ventilator. Target SpO ₂ >95%, EtCO ₂ 35-45mmHg. Check arterial blood gas values. PEEP: 5cmH ₂ O (increase up to 15cmH ₂ O if needed).
Avoid hyperventilation except in extreme cases where imminent cerebral herniation is suspected.			
Management ICP			
Suspect high ICP in any head injury patient with GCS score ≤8 or declining findings on neurologic examination. Minimize factors that could contribute to elevated ICP, such as pain, anxiety, and fever.	<ul style="list-style-type: none"> Elevate HOB 30–45° Neck midline, loosen collar SBP >110mmHg (or at least > 100mmHg) SpO₂ >93% or 95% on ventilator EtCO₂ 35-45mmHg Core temp 96–99.5°F Prevent/treat seizure Last choice if sign of herniation: hyperventilate to EtCO₂ 35mmHg-x 20 minutes. 	In addition to minimum steps, ensure adequate sedation and analgesia. If SBP >160mmHg or agitated: <ul style="list-style-type: none"> Ketamine 20mg IV/IO Hydromorphone 0.5–2mg IV/IO Fentanyl 25–50µg IV/IO Midazolam 1–2mg IV/IO 	In addition to minimum and better steps, give osmotic therapy IV/IO: <ul style="list-style-type: none"> HTS 3% 250mL over 20 minutes. Repeat every 3 hours if needed. Mannitol (if no bleeding and SBP >110mmHg) 1g/kg IV/IO over 20 minutes, repeat 0.5g/kg every 3 hours, if needed.
Telemedicine consultation early and often in the patient with elevated ICP.			
Management: Infection Control			
Dress all wounds and administer antibiotic prophylaxis for penetrating brain injuries and open wounds.	Dress all wounds to prevent further introduction of infection. Optimize wound care and patient hygiene to extent possible.	Ertapenem 1g IV/IO every 24 hours and moxifloxacin 400mg PO every 24 hours for 5 days.	Use an antibiotic with strong CNS penetration. <ul style="list-style-type: none"> 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 if wounds contaminated with organic debris.
Moxifloxacin may be replaced with levofloxacin 750 mg PO daily to provide better coverage of bacteria found in wet terrain/ jungle environment. Ertapenem and moxifloxacin may increase the risk of seizure and ertapenem may not penetrate an intact blood-brain barrier. If recommended antibiotics are not available or significant drug allergies are present, obtain teleconsultation to discuss alternative medications.			

Management of Traumatic Brain Injury Summary			
GOAL	MINIMUM	BETTER	BEST
Management: Seizures			
Rapidly identify and manage seizures.	For a witnessed or suspected seizure, ensure safety and airway is clear. Treat with rapid-acting benzodiazepine: • Midazolam 5mg IV/IO/IM every 5 minutes until seizure stops		Levetiracetam 1,500mg IV/IO loading dose over 15 minutes + 1000mg every 12 hours. Alternate therapy: • Phenytoin 1.5g IV/IO load + 100mg IV/IO every 8 hours • Phenobarbital 1.5g IV/IO load + 100mg IV/IO daily.
Consider nonconvulsive seizures in any TBI patient with GCS score ≤ 8 who is not improving with appropriate treatments. If not already placed, consider a definitive airway in any patient who experiences seizure. Perform after seizures are controlled.			
Management: Fever Control			
Maintain core temperature between 96°F and 99.5°F. Treat fever aggressively.	Remove patient from heat or sun. Remove clothing. Use surface cooling measures with misting and fan cooling	Apply cold packs to axillary, posterior cervical, and groin regions.	Acetaminophen 650mg every 4 hours PO or rectally for rectal temperature $>99.5^\circ\text{F}$. Cold saline IV bolus if available.
Avoid NSAIDs, such as ibuprofen, naproxen, and ketorolac, because these agents may increase intracranial hemorrhage, if present.			
Management: Sodium Control			
Avoid hyponatremia. Mild hypernatremia optimal. Target sodium level: 145–160mmol/L.	Avoid administration of free water or hypotonic fluids.		Monitor serum sodium via laboratory blood samples. If patient is stable, check levels every 6 hours. In an unstable patient or one receiving HTS, check sodium level every 3 hours and adjust fluids as needed.
Numerous conditions can rapidly affect sodium levels in TBI patients. Monitor sodium and urine output whenever possible.			
Management: Blood Glucose Control			
Avoid both hypo- and hyperglycemia. Target blood glucose 100–180mg/dL.	Monitor for signs and symptoms of hypoglycemia. Allow patient to eat as long as they are able.		Check blood glucose every 6 hours with handheld glucometer. If $<100\text{mg/dL}$, give 25g (50mL) dextrose 50% (D50) IV/IO or 5tsp sugar/4tsp honey PO/NG.
Transportation			
TBI patients for ground and/or air transport to higher levels of care.	Dose sedative/analgesic medications, osmotic therapies, and/or benzodiazepines before any significant ground or air transport if possible.		Provide a detailed brief to the transport assets specifically highlighting any neurologic deficits and treatments and/or accommodations required during transport.
Neuromuscular blockers should only be used when the benefit outweighs the risks. Most patients should be transported in the supine position with the HOB elevated at least 30° . **For fixed-wing aircrafts: load patient with head to the front of the aircraft to minimize G-forces transmitted to the brain. **For altitudes >8000 ft, TBI patients are at risk of hypobaric hypoxia and high-altitude cerebral edema (HACE), which can worsen brain injuries.			

APPENDIX F: TELECONSULTATION / TELEMEDICINE

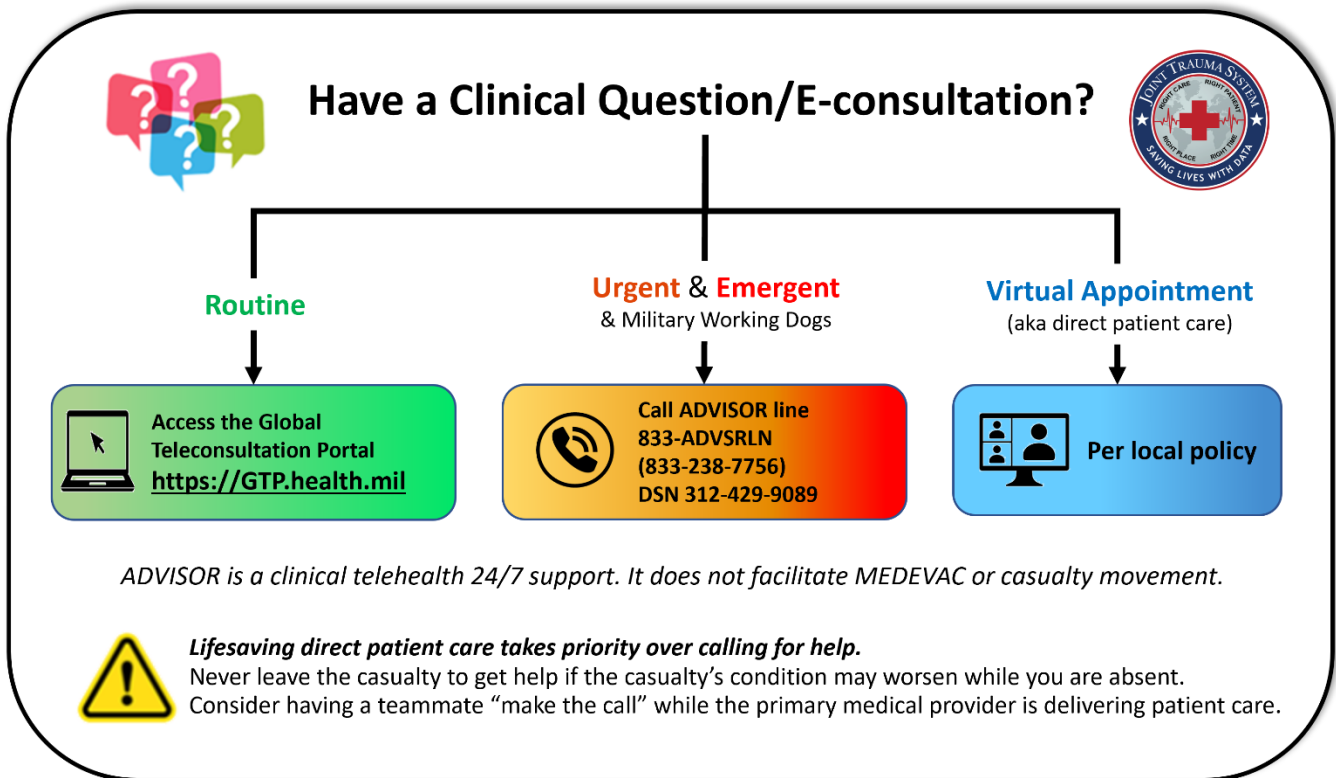


Illustration by Raymond Samonte

GTP: <https://GTP.health.mil>

APPENDIX G: INFORMATION REGARDING OFF-LABEL USES IN CPGS

PURPOSE

The purpose of this Appendix is to ensure an understanding of DoD policy and practice regarding inclusion in CPGs of “off-label” uses of U.S. Food and Drug Administration (FDA)–approved products. This applies to off-label uses with patients who are armed forces members.

BACKGROUND

Unapproved (i.e., “off-label”) uses of FDA-approved products are extremely common in American medicine and are usually not subject to any special regulations. However, under Federal law, in some circumstances, unapproved uses of approved drugs are subject to FDA regulations governing “investigational new drugs.” These circumstances include such uses as part of clinical trials, and in the military context, command required, unapproved uses. Some command requested unapproved uses may also be subject to special regulations.

ADDITIONAL INFORMATION REGARDING OFF-LABEL USES IN CPGS

The inclusion in CPGs of off-label uses is not a clinical trial, nor is it a command request or requirement. Further, it does not imply that the Military Health System requires that use by DoD health care practitioners or considers it to be the “standard of care.” Rather, the inclusion in CPGs of off-label uses is to inform the clinical judgment of the responsible health care practitioner by providing information regarding potential risks and benefits of treatment alternatives. The decision is for the clinical judgment of the responsible health care practitioner within the practitioner-patient relationship.

ADDITIONAL PROCEDURES

Balanced Discussion

Consistent with this purpose, CPG discussions of off-label uses specifically state that they are uses not approved by the FDA. Further, such discussions are balanced in the presentation of appropriate clinical study data, including any such data that suggest caution in the use of the product and specifically including any FDA-issued warnings.

Quality Assurance Monitoring

With respect to such off-label uses, DoD procedure is to maintain a regular system of quality assurance monitoring of outcomes and known potential adverse events. For this reason, the importance of accurate clinical records is underscored.

Information to Patients

Good clinical practice includes the provision of appropriate information to patients. Each CPG discussing an unusual off-label use will address the issue of information to patients. When practicable, consideration will be given to including in an appendix an appropriate information sheet for distribution to patients, whether before or after use of the product. Information to patients should address in plain language: a) that the use is not approved by the FDA; b) the reasons why a DoD health care practitioner would decide to use the product for this purpose; and c) the potential risks associated with such use.