Head Trauma  
PFN: SOMEML35  
Hours: 3.0  
Instructor:

Terminal Learning Objective

- Action: Communicate knowledge of head trauma
- Condition: Given a lecture in a classroom environment
- Standard: Received a minimum score of 75% on the written exam IAW course standards

References

- USASOC Neurocognitive Testing and Post-Injury Evaluation and Treatment Clinical Practice Guideline (CPG)
- USASOC Guidelines For Post-Concussion Rehabilitation
- USASOC mTBI Symptom Cluster Treatment Recommendations
- USASOC Concussion Patient Information Sheet
References

- Tintinalli’s Emergency Medicine, 7th edition, 2010
- Tactical Trauma Protocols
- PHTLS, 7th edition, 2010
- USASOC Policy 10-10 Neurocognitive Testing Program

Reason

Agenda

- Identify key definitions and epidemiology of traumatic brain injury
- Identify the pathophysiology of traumatic brain injury
- Identify types of head injuries
- Identify the clinical presentation and management of scalp injuries
- Identify the clinical presentation and management of open head injuries
Agenda

- Identify the clinical presentation and management of penetrating injuries
- Identify the clinical presentation and management of closed skull injuries
- Identify the clinical presentation and management of closed head injuries
- Identify the clinical presentation of blast head injuries

Identify Key Definitions and Epidemiology of Traumatic Brain Injury

- Identify the clinical presentation and management of brain injuries
- Identify the Clinical Presentation and management of moderate to severe TBI
- Identify the Clinical Presentation and management of mild TBI
- Participate in a head trauma case review
- Mace Demonstration
TBI in the Department of Defense

- Over 80% of TBIs occur in a non-deployed setting
- Common causes of TBI include MVA, falls, sports and recreation activities, and military training

TBI Epidemiology

- Leading cause of death and disability in children and adults from ages 1 to 44
- Twice as common in men
- Falls, MVCs, and concussive weapons
- Trimodal distribution
  - > 0 to 4 years old
  - > 15 to 24 years old
  - > 75 years old
- Mortality rises continuously with age
- > 20% of combat personnel sustain TBI

Traumatic Brain Injury (TBI)

- Impairment in brain function as a result of mechanical force
- Can be temporary or permanent
- May or may not result in underlying structural changes in the brain
- Clinical severity ranges from very mild to profoundly impaired
Identify the Pathophysiology of Traumatic Brain Injury

Intracranial Pressure

- Brain is a semisolid organ that occupies 80% of the cranial vault
  - 20% of the body’s oxygen supply
  - 15% of cardiac output
- Cranial Vault = Fixed in size by outer rigid skull
  - Contains brain tissue, blood vessels and CSF
- Monroe-Kelli Doctrine
  - Defines the relationship between the volumes of the three compartments
  - The expansion of one compartment MUST be accompanied by a compensatory reduction in the volumes of the other compartments to maintain a stable intracranial pressure (ICP)

Primary Injury

- "Impact Injury"
  - Tissue destruction that occurs as a direct result of a physiologic trauma
  - Very little can be done by providers to influence primary injury
  - Severity and location of the primary brain injury will dictate the patients immediate level of consciousness, metal status, and focal neurologic signs
Secondary Brain Injury

- Focus of TBI treatment, to minimize secondary brain injury - can be more damaging than primary injury
- HYPOTENSION AND HYPOXIA are the two most acute and easily treatable mechanism of secondary injury
- Continues for an indefinite period of time

Secondary Brain Injury

- Systemic Insults
  - Hypoxia (PaO2 < 90)
    - Mortality of TBI pts with hypoxia = doubled
    - Increases brain cell death and edema
  - Hypotension (SBP < 90 mmHg)
    - Hypotension → ↓Cerebral Perfusion→↑Cerebral Ischemia →↑Doubles Mortality
  - Anemia 2/2 Blood Loss (↓Oxygen Carrying Capacity)
  - Hypocapnia (↓pCO2) /Hypercapnia (↑pCO2 retention)
    - Hyperventilation → ↓pCO2 Levels → ↑Serum pH → Cerebral Vasoconstriction → ↓Cerebral Blood Flow

Other Systemic Cause
- Anemia (impair oxygen delivery to the brain)
- Hypotension (impair oxygen delivery to the brain)
- Increased or decreased blood glucose (Brain cells cannot function without it)
- Seizures (seen in 30%–40% of patients with penetrating brain injuries)

Intracranial Cause
- Cerebral edema
- Hematomas
- Increased intracranial hypertension (ICP)
Increased Intracranial Pressure

- Most frequent cause of death and disability after severe head injury
- Delayed cerebral swelling is the major cause of raised ICP and death
- When ICP rises:
  - CPP decreases → PO2 decreases → PCO2 levels increase → cerebral vasodilation → cerebral blood volume increases → further increased ICP

Pathophysiology

Identify Types of Head Injuries
Head Injuries

- Scalp Injuries
  - Open (puncture, laceration, avulsion)
  - Closed (contusion)

- Skull Fractures

- Open Head Injuries
  - Penetrating

- Closed Head Injuries
  - Blunt
  - Diffuse Axonal Injury (DAI)
  - Intracranial hemorrhages
  - Cerebral contusions

- Primary Blast
  - Overpressure central nervous system injuries

Identify the Clinical Presentation and Management of Scalp Injuries

Scalp Injury

Injury to the overlying skin of the scalp, which may be in combination with injury to the skull, brain and/or face

- Causes
  - Penetrating trauma (rifle, impaled objects, missile wounds)
  - Blunt trauma (MVA, blast)

- Closed (contusion)

- Open (puncture, laceration, or avulsion)
  - Can lead to massive blood loss and hypovolemic shock
Scalp Injury

- Hemorrhage control
  - Direct pressure or pressure dressings
  - Donut ring
  - Lidocaine with epinephrine infiltration
  - Clamp or ligate vessels
- Assess for fracture
- Prevent contamination
- Repair galeal defects

Identify the Clinical Presentation and Management of Open Head Injuries

Open Head Injuries

- Causes
  - Penetrating trauma
  - Blunt trauma
- Signs and Symptoms
  - Profuse bleeding no matter how minor the injury
  - Crepitus
  - Edema
  - Depressions
  - Deformities
  - Visualize skull or bony fragments
Identify the Clinical Presentation and Management of Penetrating Injuries

Penetrating Injury

- Missiles and stab wounds
- Associated injuries
  - Skull fracture
  - Damage to cerebral vasculature
  - Intracranial hemorrhage
- Complications include infection and post-traumatic epilepsy

Penetrating Injury

A. Lateral skull film of a 19-year-old male who sustained multiple stab wounds to the head, demonstrating extensive pneumocephalus.

B. A CT scan of the head of the same patient demonstrates numerous pockets of air in the suli with marked subarachnoid pneumocephalus.
Identify the Clinical Presentation and Management of Closed Skull Injuries

Skull Fractures

Classification of Skull Fractures

- Open versus closed
- Location
  - Specific skull bone (i.e. temporal)
  - Basilar
- Pattern
  - Linear
  - Depressed
  - Comminuted
Linear Skull Fracture
- Accounts for about 80 percent of all open head injuries
- Injury does not penetrate brain tissue, most are minor and require little treatment

Depressed Skull Fracture
- Typically occurs when significant force is applied over a small area
- Scalp lacerations should undergo sterile exploration

Basilar Skull Fracture
- May occur anywhere along the skull base
- Significant risk factor for intracranial injury
- Fractures to the basilar area typically do not have localizing symptoms
- Indirect signs often develop
Battle’s Sign
- Retro-auricular Ecchymosis
- Discoloration of the soft tissue behind the ear
- Associated with fracture of auditory canal and lower areas of skull
- This is a late sign and may not be readily seen.

Raccoon Eyes
- Bilateral Periorbital Ecchymosis
- Fracture in the anterior portion of the skull base

Hemotympanum
- Blood behind tympanic membrane
- Fracture line communicates with the auditory canal
- May have associated vertigo, hearing loss, and CN VII palsy
Classify this Fracture

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CSF Leak

- CSF can leak from the nose (rhinorrhea) or ears (otorrhea)
- Dextrose or halo test can assist in confirmation, but is not reliable
- Patients can develop meningitis
- Requires surgical repair
- Head elevation and antibiotics are field treatment

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Identify the Clinical Presentation and Management of Closed Head Injuries
Closed Head Injury

May or may not be lacerations of the scalp, but the skull is intact, and there is no opening to the brain. Injury to the brain itself may be far more extensive in a closed head injury because more of the injuring force is transmitted deeper into the brain due to pressure build-up.

- **Causes**
  - Coup-Contrecoup
  - Blunt Trauma

- **Signs and Symptoms**
  - Signs around injury site
  - Headaches
  - Neurological symptoms:
    - Altered LOC
    - Restlessness
    - Unequal pupils
  - Bruising
  - Drainage - drainage of blood or CSF fluid from the ears, nose, or eyes.
  - Bradycardia
  - Increased systolic blood pressure
  - Nausea/vomiting
  - Decreased respiratory/Cheyne Stokes breathing pattern
  - Deformity of the skull

Coup/Contrecoup

**Focal Brain Injury**

- **Coup**
  - The brain is injured directly under the area of impact
- **Contrecoup**
  - Brain is injured on the side opposite the impact

Diffuse Axonal Injury (DAI)

**Diffuse Brain Injury**

- Stretching and shearing of white matter and axons
- Generated by sudden deceleration or rotational forces
- Edema develops rapidly
- Often produces devastating and irreversible deficits
- Blunt trauma, MVA, or Shaken Baby
Diffuse Axonal Injury (DAI)  
Diffuse Brain Injury

Identify the Clinical Presentation of Blast Head Injuries

Primary Blast
A direct injury to the brain or via a force transmitted by the great vessels of the chest to the brain

- Associated with: unconsciousness, confusion, headache, tinnitus, dizziness, tremors, increased startle response, and increasing ICP
- Bleeding may occur from multiple orifices
- May have no external signs of injury and only subtle signs of cognitive dysfunction in attention, concentration, reaction time, and balance
Identify the Clinical Presentation and Management of Brain Injuries

Direct Brain Injury
- Focal
  - Occur at a specific location in brain
  - Differentials
    - Cerebral Contusion
    - Intracranial Hemorrhage
    - Epidural hematoma
    - Subdural hematoma
    - Intracerebral Hemorrhage
- Diffuse
  - Concussion
  - Diffuse Axonal Injury (DAI)

Cerebral Contusion
Focal Brain Injury
- Cerebral Contusion
  - Blunt trauma to local brain tissue
  - Capillary bleeding into brain tissue
  - Common with blunt head trauma
    - Confusion
    - Neurologic deficit
      - Personality changes
      - Vision changes
      - Speech changes
    - Results from
      - Coup-contrecoup injury
Cerebral Contusion

- The head CT scan of a young male who after becoming intoxicated fell to the ground, striking the left side of his head. Note the large left-sided scalp contusion. Also note the right-sided cerebral contusion (contrecoup injury).

Types of Brain Hemorrhage

- Classified according to location
  - Epidural
  - Subdural
  - Subarachnoid
  - Intracerebral (intraparenchymal)

Epidural Hematoma

- Blood between the skull and dura mater
- Blunt trauma to the temporal or temporoparietal area with an associated skull fracture and middle meningeal arterial disruption is the primary mechanism of injury
- Can lead to herniation within hours after an injury because of high pressure arterial bleeding
Epidural Hematoma

- Classic Symptoms
  - Transient loss of consciousness, followed by a lucid interval then rapid neurological decline
- Not common

- Lucid interval usually last between 6-18 hours
- As ICP rises symptoms present, level of consciousness decreases
- Patient may only complain of HA and drowsiness in early stages

Subdural Hematoma

- Blood between the dura mater and the surface of brain
  - Usually bleeding from veins that bridge subdural space due to sudden acceleration and deceleration
  - Associated contusion or laceration of the brain is frequently present
  - Often the result of blunt head trauma
  - Commonly associated with skull fracture
Subdural Hematoma

Subarachnoid Hemorrhage

- Intracranial bleeding into CSF resulting in bloody CSF and meningeal irritation
- Bleeding typically results from trauma or rupture of an aneurysm
- Classic presentation = “worst headache of life” - Thunderclap
- Neck stiffness (meningeal irritation)

Intracerebral Hemorrhage

- > 5 ml blood somewhere within brain
- Causes
  - Multiple lacerations produced by penetrating head trauma
  - High velocity deceleration injury
  - Compression and distortion from increased ICP
- Often associated with subdural hemorrhage and skull fracture
Identify the Clinical Presentation and Management of Moderate to Severe TBI

Goals of TBI Management

- Three primary goals
  - Identify other life-threatening injuries
  - Prevent further secondary brain injury
  - Identify treatable mass lesions
- Correct or prevent hypoxia, hypotension, anemia, and hyperthermia
- Evacuate intracranial masses
  - Performed at surgical facility

TBI Assessment

- History
  - Reconstruct the MOI
  - SAMPLE history
  - Nausea, vomiting, headache, memory impairment, visual/auditory symptoms, seizures, and loss of consciousness
  - Drug or alcohol intoxication
TBI Assessment
Physical Examination

- Head Exam
  - Scalp trauma
  - Skull fractures
  - Signs of basilar skull fracture
- Assess for other injuries
  - C-spine and extremities
  - May be difficult to detect
  - All head trauma patients are assumed to have a cervical spine injury until proven otherwise

Airway
Respiration/Breathing

- Early establishment of a definitive airway is imperative
  - Suctioning
  - Patient Positioning
  - OPA & NPA Use
  - Endotracheal Intubation
  - Cricothyrotomy
- Avoid hypoxia by maintaining the oxygen saturation >90% by initiating pulse oximetry monitoring
- Avoid vasoconstriction or vasodilation by maintaining the PaCO2 between 35 and 40 mm Hg.
- Given supplemental oxygen when available to maintain an oxygen saturation

Circulation

- Hemorrhage Control
  - Cover open head wounds securely enough to aid in the clotting process without pressing skull fragments or impaled objects inward by using donut o-ring.
- Blood Pressure Maintenance
  - SBP must be maintained above 90
- Initiate fluid bolus as indicated - DO NOT LET THE SBP GO BELOW 90 (can DOUBLE mortality rate)
Fluid Resuscitation

Goal - Systolic Blood Pressure above 90

- Traumatic brain injury and in shock (has a weak or absent radial pulse)
  - Reduces cerebral perfusion & hypoxia
- Initiate IV/IO Hextend, LR, or Plasma-Lyte A
- Continue until restoration of palpable radial pulse, improved mental status, or SBP > 90mmHg

Neurological Examination

- Level of Consciousness (LOC)
- LOC is best indicator of perfusion
  - AVPU
  - Glasgow Coma Score (GCS)
  - Reevaluate several times during encounter
  - Deterioration in LOC = best indicator of increasing ICP

TBI Classification

- Classified based patient’s LOC, not actual underlying injury
- Patients with same TBI severity classification may have dramatically different pathophysiology
- Mild TBI - GCS 14 to 15
- Moderate TBI - GCS 9 to 13
- Severe TBI - GCS 3 to 8
Neurological Examination

- Pupillary Size + Reactivity
  - Fixed Dilated Pupil = Ipsilateral intracranial hematoma resulting in uncal herniation
  - Bilateral Fixed + Dilated = Poor brain perfusion, bilateral uncal herniation or severe hypoxia
    - Indicative of very poor neurological outcome
  - Alcohol and other drugs can cause abnormal pupillary reactions

- Full, Complete Neurological Exam
  - Examine for subtle neurological deficits
  - Look for specific injury patterns:
    - Battle's sign, Otorrhea, Rhinorrhea, Hemotympanum, peri-orbital
    - Ecchymosis is indicative of skull fracture and is concerning for underlying brain injury

Increasing ICP Clinical Presentation

- Early warning signs
  - Headache
  - Nausea and vomiting
  - Altered level of consciousness
    - The earliest sign of increasing ICP is a change in LOC
    - Decline in GCS score of 2 points or more

Cranial Nerves

- CN III
  - Oculomotor Nerve
    - Controls pupil size
    - Pressure paralyzes nerve; pupil dilates and becomes unreactive

- CN X
  - Vagus Nerve
    - Supplies SA and AV nodes
    - Pressure on nerve stimulates bradycardia
Herniation Syndromes

- Eventually some part of the brain will push through an opening
- Various syndromes and presentations
- Uncal transtentorial is the most common

Increasing ICP
Clinical Presentation

- Rapidly unresponsive to verbal and painful stimuli
- Neurological Posturing
  - Decorticate Posturing
  - Decerebrate Posturing
- Ipsilateral dilated and unreactive pupil

Increasing ICP
Clinical Presentation
Cushing's Reflex (triad)

- Acute entity seen in severely head injured patients with significant increased intracranial pressure and impending herniation
  - Hypertension
  - Bradycardia
  - Respiratory Irregularity (e.g., Cheyne-Stokes)

Papilledema

- Unilateral presentation is extremely rare - PROBABLY NOT GOING TO BE SEEN, it is a late finding in ICP and thus is not seen in acute head injury.
Increasing ICP Management

Head Elevation

- If cerebrospinal fluid (CSF) is identified leaking from the ears and/or nose, elevate the head **30 to 60 degrees** if the casualty's other injuries permit and the casualty is hemodynamically stable.

- If the casualty exhibits signs of increased ICP and is hemodynamically stable, consider elevating the head **30 degrees** to improve venous outflow from the brain and decrease ICP.

Increasing ICP Management

Controlled Hyperventilation

- Temporizing measure for evidence of increasing ICP and herniation - **NOT ROUTINE**

  - With Capnography
    - Ventilate to achieve pCO₂ of 30-35 mm Hg

  - Without Capnography
    - Ventilate at 20 BPM, tidal volume of approximately 500ml

    - Use the highest oxygen concentration possible

Increasing ICP Management

Hypertonic Saline (3%)

- Osmotic agent used to increase the osmotic pressure gradient between blood and tissues in order to draw water out of the brain and reduce intracranial pressure

  - **Isolated TBI** (Hemodynamically stable)
    - Administer 3% HS 250 ml IV/IO Bolus

  - **TBI with controlled external hemorrhage**
    - Administer 3% HS 250 ml IV/IO Bolus plus Hextend/other fluids as per the fluid resuscitation protocol if required
Increasing ICP Management
Mannitol (Osmotrol)

- Large glucose molecule, Does not leave blood stream, Osmotic Diuretic, Effective in drawing fluid from brain
- Dose
  - 1gm/kg
- CAUTION
  - Forms crystals at low temperatures
  - Reconstitute with rewarming & gentle agitation
  - USE IN-LINE filter & PREFLUSH line

Fosphenytoin (Cerebyx®)
Seizure Prophylaxis

- Indications
  - Prevention and treatment of seizures
  - TMEP: Seizure Protocol
  - TTP: Head Injury Management
- Dose
  - 18 mg/kg IV/IO at 100 to 150mg/min (over 15 mins)
  - Repeat 100mg IV/IO Q 8 h for maintenance

Fosphenytoin (Cerebyx®)
Seizure Prophylaxis

- Special Considerations
  - WARNING - Do not administer faster than 150 mg/min since this may result in hypotension
- Other Notes
  - Store under refrigeration at 2° C to 8° C (36° F to 46° F).
  - The product should not be stored at room temperature for more than 48 hours
Active Seizure Management

- Diazepam (Valium) 5 to 10 mg IV/IO q 5 min max dose of 20 mg
- Midazolam (Versed) 5 mg IV/IO q 5 min (no maximum dose)
  - Monitor casualty closely for apnea when administering benzodiazepines
- Fosphenytoin (Cerebyx) 18 mg/kg IV/IO at 100 to 150 mg/min (slow IVP) for seizures refractory to benzodiazepines
  - WARNING - Do not administer faster than 150 mg/min since this may result in hypotension

Antibiotics

- Given for penetrating head trauma or open skull fracture
  - Ertapenem (Invanz) 1gm IV/IO
  - Ceftriaxone (Rocephin) 1gm IV/IO

Sedation

- Agitation is common finding and may result from pain, delirium or difficulties with oxygenation and ventilation
- Painful stimuli and stress increase metabolic demands and increase blood pressure and ICP
- Minimizing agitation should be goal to limit increases in ICP or inabilities to oxygenate and ventilate
  - Midazolam (Versed) 1 to 2 mg IV/IO if no evidence of shock or hypotension exists
  - Diazepam (Valium) 5-10mg IV/IO if no evidence of shock or hypotension exists
Analgesia

- **Mild to Moderate Pain**
  - (still able to fight)
  - Combat Wound Medication Pack
    - Tylenol 650mg bilayer caplet, 2 PO every 8 hours
    - Meloxicam 15mg PO once a day

- **Moderate to Severe Pain**
  - (Patient IS NOT in shock/respiratory distress AND IS NOT at risk of developing either condition)
    - Oral transmucosal fentanyl citrate (OTFC) 800µg
      (IV Morphine is an alternative if IV is in)

  - **Moderate to Severe Pain**
    - (Patient IS in hemorrhagic shock/respiratory distress OR IS at risk of developing either condition)
      - Ketamine 50mg IM or IN
      - Ketamine 20mg slow IV or IO

(Ketamine and OTFC

- **TCCC Guidelines for Medical Personnel**

  - Both ketamine and OTFC have the potential to worsen severe TBI. The combat medic, corpsman, or PJ must consider this fact in his or her analgesic decision, but if the casualty is able to complain of pain, then the TBI is likely not severe enough to preclude the use of ketamine or OTFC

Evacuation

- If possible, evacuate casualty to a facility with a neurosurgeon available
Identify the Clinical Presentation and Management of Mild TBI

mTBI

- Consider mTBI (concussion) in anyone who is dazed, confused, “saw stars”, lost consciousness (even if just momentarily) or has memory loss that resulted from a fall, explosion, MVC, or any other event involving abrupt head movement, a direct blow to the head or other head injury.

- Triage and treat other injuries as required, and evaluate for mTBI as soon as tactically feasible.

Mild Traumatic Brain Injury (mTBI) "Concussion"

- Physiologic changes in brain functioning resulting from trauma to the head without radiographic evidence of structural damage.

- Does not require loss of consciousness.

- Clinical symptoms and cognitive deficits linked to brain-related changes in physiology.

- Normal structural neuroimaging.
mTBI

- Concerns:
  - Period of vulnerability
  - Less biomechanical force results in more serious injury
  - Physical and cognitive exertion protracts and complicates recovery

Cellular Level Changes

mTBI Assessment

- History
- Physical exam
- Neurocognitive testing
History

- Loss of consciousness
- Drowsiness, restlessness, confusion, anxiety
- Amnesia
  - Retrograde - unable to recall events before the injury
  - Antegrade - unable to recall events after the injury
- Abnormal speech; repetitive questioning
- Vomiting

Physical Exam

- Ruptured tympanic membranes
- Trauma to the head and/or neck
  - Penetrating and non-penetrating
- Cranial nerve deficits
- Sensory deficits
- Motor deficits

Physical Exam

- Inability to do rapid alternating movements
- Visual field deficits
- Abnormal mini-mental status evaluation
- Abnormal vestibular screening exam
  - Inability to maintain balance
  - Persistent nystagmus
  - Tracking and convergence problems with extraocular movements
**Red Flags**

- **Neurological**
  1. Witnessed loss of consciousness
  2. Amnesia and memory problems
  3. Unusual behavior or combative
  4. Seizures
  5. Worsening headache
  6. Cannot recognize people
  7. Abnormal speech

- **Eyes**
  1. Double vision

- **General**
  1. Two or more blast exposures within 72 hours
  2. Repeated vomiting
  3. Weakness
  4. Unsteady on feet

**Neurocognitive Testing**

- **Immediate Post-concussion Assessment and Cognitive Testing (ImPACT)**

- **Military Acute Concussion Evaluation (MACE)**

**ImPACT**

- **Immediate Post-concussion Assessment and Cognitive Testing**
- **Computerized neurocognitive assessment**
- **Assist in determining patients ability to return to duty after suffering a concussion**
- **All SOF personnel should have a baseline**
- **Retest after injury**
When to Test

Clinical Protocol: Neurocognitive Testing

MACE
Military Acute Concussion Evaluation
- Can be easily used by medics to confirm a suspected diagnosis of concussion
- Can be administered in 5 minutes
- Assesses four cognitive domains
  - Orientation
  - Immediate memory
  - Concentration
  - Delayed recall

MACE
Mandatory events requiring MACE
- Personnel in a vehicle associated with a blast, collision or rollover
- Personnel within 150 meters of a blast
- Personnel with a direct blow to the head
- Command directed evaluation
MACE

- Total possible score is 30
- Mean total score for non-concussed patients is 28
- Scores below 25 may represent clinically relevant neurocognitive impairment
  - Requires further evaluation for the possibility of a more serious brain injury
- Serial assessments are important and used to document either a decline or improvement in cognitive functioning

Protracted Recovery Risk Factors

- Age
- History of migraine headache
- Exertion
- Gender
- History of previous concussion?

mTBI Management

- Symptomatic treatment and prevention of secondary injury
- All patients with mild traumatic brain injury should be observed for 24 hours after that injury
Protect from Further Injury

- Controlled environment
  - Come out of the fight
  - No contact activities
- Limit exertion
  - Physical and mental
  - “Sweet Spot”

Protect from Further Injury

- Do not allow a patient with a mTBI to return to duty prior to a full recovery
- mTBI patients are at risk for greater injury if they sustain another head injury or exceed the “sweet spot of exertion” while still recovering
  - Prolonged recovery
  - Second impact syndrome
  - Post-Concussive syndrome
  - PTSD; depression

Protect from Further Injury

Second Impact Syndrome

- Additional injury during period of vulnerability
- With the first head injury a disruption of the normal cerebral vascular auto regulation occurred, causing increased cerebral blood flow, making the brain vulnerable to the second impact, when the rapid malignant swelling occurs
- Less biomechanical force leading to injury in susceptible individuals
Treat the Headache

- Tylenol is the best choice
  - If possible, avoid the use of COX 1 NSAIDs (Motrin/ibuprofen, Aleve/naproxen) due to effects on platelets and a potential increased risk of bleeding
  - If COX 1 NSAIDs are the only medication available and the patient has no red flags they may be used
- Avoid Ultram (tramadol), Benadryl, and narcotics due to their ability to alter level of consciousness

Evacuate if Indicated

- Red Flags present
  - Consult with medical provider for possible urgent evacuation
- If MACE < 25 or symptoms persist despite rest and appropriate treatment, consult with medical provider for possible priority evacuation

Educate, Reevaluate, and Rehabilitate

- Educate your patient
- If MACE is normal, recommend 24 hour rest and re-evaluation
- Perform ImPACT testing during the 24 to 72 hour post-injury window
- Graded return of cognitive and physical exertion
**Rehabilitation Protocol**

<table>
<thead>
<tr>
<th>Stage 1</th>
<th>Target Heart Rate: 30‐40% of maximum exertion</th>
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<tbody>
<tr>
<td>Recommendations:</td>
<td>- Exercise in quiet area; no impact activities; balance and vestibular exercises as needed; limit head movement/position changes; limit concentration activities; 10‐15 minutes light cardio - Very light aerobic conditioning - Sub‐max isometric strengthening - ROM/Stretching - Low‐level balance activities</td>
</tr>
<tr>
<td>Examples:</td>
<td>- Stationary Bike; seated elliptical; treadmill walking (10‐15 minutes) - Quad sets; Ham sets; light hand weights; resistive band rowing; SLR's; resistive bands ankle strengthening - Cervical ROM exercise; trap/LS stretching, pec stretching; hamstring stretching, quad stretching, calf stretching - Romberg exercises, single leg balance</td>
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<tr>
<th>Stage 2</th>
<th>Target Heart Rate: 40‐60% of maximum exertion</th>
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<tbody>
<tr>
<td>Recommendations:</td>
<td>- Exercise in gym areas; use various exercise equipment; allow some positional changes in head movement; low level concentration activities; 20‐30 minutes of cardio - Light to moderate aerobic conditioning - Light weight - Active stretching - Moderate balance activities; initiate activities with head position changes</td>
</tr>
<tr>
<td>Examples:</td>
<td>- Treadmill; stationary bike; elliptical (20‐30 minutes) - Light weight strength exercises, resistive band exercises; wall squats; lunges; step up/downs - Any stage 1 stretching; active stretching as tolerated - Romberg exercises, VOR exercise (walking with eyes focused and head turns); Swiss ball exercises; single leg balance exercises</td>
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<tr>
<th>Stage 3</th>
<th>Target Heart Rate: 60‐80% of maximum exertion</th>
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<tr>
<td>Recommendations:</td>
<td>- Any environment ok; integrate strength, conditioning, and balance/proprioceptive exercises; can incorporate concentration challenges; 25‐30 minutes of cardio - Moderately aggressive aerobic exercise - All forms of strength exercise - Active stretching exercise - Impact activities (running, plyometrics) - Challenging proprioceptive and dynamic balance; challenging positional changes</td>
</tr>
<tr>
<td>Examples:</td>
<td>- Treadmill (jogging); stationary bike; elliptical (25‐30 min) - Resistive weight training including free weights; functional squat; dynamic strength activities - Active stretching (lunge walks, side to side groin stretching, walking hamstring stretch) - Initiate agility drills (zig‐zag runs, side shuffle), jumping on blocks - Higher level balance activities: ball toss on plyo floor, balance discs, squats and lunges on BOSU ball</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Stage 4</th>
<th>Target Heart Rate: 80% of maximum exertion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recommendations:</td>
<td>- Resume aggressive training in all environments - Non‐contact physical training - Aggressive strength training - Impact activities/plyometrics - Job‐specific physical training</td>
</tr>
<tr>
<td>Examples:</td>
<td>- Program to be designed by unit physical therapist - Interval training - Job‐specific drills/training</td>
</tr>
</tbody>
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<table>
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<tr>
<th>Stage 5</th>
<th>Target Heart Rate: Full exertion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recommendations:</td>
<td>- Resume full physical training with contact - Continue aggressive strength and conditioning exercises - Job‐specific activities (shooting, CQB training, fast‐roping)</td>
</tr>
<tr>
<td>Examples:</td>
<td>- Program to be designed by unit physical therapist - Train at full combat intensity</td>
</tr>
</tbody>
</table>

**Return to Duty**

- Completed stage 5 rehabilitation protocol
- Symptom free
- Consider additional ImPACT exam
- If you do not feel that a patient is back to their baseline, do not allow them to RTD and consult a medical provider

**Post‐Concussion Syndrome**

- A set of symptoms develops within 4 weeks of the injury and may persist for months (90% at 1 month, 25% at 1 year)
  - Chronic migraine type headache
  - Photosensitivity
  - Nausea
  - Neurobehavioral changes
  - Sleep deficits
  - Cognitive deficits
  - Academic difficulties
mTBI and PTSD

- Warrior 2 Warrior
  - www.warrior2warrior.org/
- Veterans Crisis Line
  - www.veteranscrisisline.net
- 22Kill
  - www.22kill.com

Case Review

Case Review Part 1/3

A 28 year old 18-series Soldier was the driver in a vehicle which struck an improvised explosive device (IED). The vehicle was destroyed and the other occupant in the vehicle was killed instantly. The Soldier recalled hitting the roof of the vehicle at least once; however, he suffered no life threatening injuries and was not initially evacuated to higher medical care. Immediately following the event the Soldier noticed a headache, mild dizziness, nausea, and short term memory loss. Upon return to the forward operating base (FOB), he was evaluated by the forward surgical team physician who performed a military acute concussion exam (MACE). His score was 24 out of 30 and he was diagnosed with a mild concussion, his symptoms were treated with acetaminophen, and he was released.
Case Review Part 2/3
On day three, he participated in another combat patrol. During the operation he suffered from dizziness and headaches. He self medicated with acetaminophen and meclizine transdermal for his symptoms. On this patrol, he was exposed to overpressure from explosions on two separate events. Each explosion was the equivalent of approximately 27 pounds of TNT. Following the mission, he returned to his FOB and noticed increased dizziness, nausea and memory loss. No other members of his team who were exposed to those two explosions reported any symptoms. He continued to self medicate with acetaminophen and meclizine transdermal. He did not seek follow-up medical care. Later that same day, he participated in a third mission as part of a quick reaction force which included a high altitude helicopter assault. He was not exposed to any additional blast or injuries.

Case Review Part 3/3
After returning from the third mission, the patient experienced significant fatigue. He went to sleep and later the same night experienced loss of consciousness after quickly standing from a lying position. Upon recovering consciousness, he experienced increased dizziness, nausea, and emesis. He was evaluated by the unit medical provider and evacuated to a Level III theater Hospital. Over the next three days, his nausea and dizziness improved; however, he continued to have significant issues with short term memory loss, difficulty concentrating, short term memory recall, and headaches. His MACE scores slowly improved to 27 out of 30 over several days. He was evacuated through Landstuhl regional Medical Center to the U.S. for additional evaluation and treatment.

Test Your Knowledge
• What diagnosis would you give this patient after his initial blast injury?
  - Concussion without loss of consciousness/mTBI
• What red flag did this patient display after the initial blast injury?
  - Amnesia
• What is the significance of this finding?
  - Amnesia is 10Xs more predictive than loss of consciousness in predicting deficits on ImPACT
Test Your Knowledge

- How many blasts was this patient exposed to over a three day period?
  - Three
- No other teammates suffered injury from blast 2 or 3. So what happened to this patient?
  - Second Impact Syndrome

Test Your Knowledge

- Was evacuation indicated?
  - "If Red Flags present, consult with medical provider for possible urgent evacuation. If MACE < 25 or symptoms persist despite rest and appropriate treatment consult with medical provider for possible priority evacuation."
- Was the headache treated appropriately?
  - Tylenol was administered
  - Unsure of effectiveness

Test Your Knowledge

- How would you manage this patient on initial presentation with a headache, mild dizziness, nausea, short term memory loss, and a MACE score of 24?
  - Evacuate if indicated
  - Treat the headache
  - Protect from further injury
  - Educate, reevaluate, and rehabilitate
  - RTD
Test Your Knowledge

- Was the patient protected from further injury?
  - No

- Did the patient receive appropriate education, reevaluation, and rehabilitation prior to being returned to duty (RTD)?
  - No

MACE Demo

Questions?
Agenda

- Identify key definitions and epidemiology of traumatic brain injury
- Identify the pathophysiology of traumatic brain injury
- Identify types of head injuries
- Identify the clinical presentation and management of scalp injuries
- Identify the clinical presentation and management of open head injuries

Agenda

- Identify the clinical presentation and management of penetrating injuries
- Identify the clinical presentation and management of closed skull injuries
- Identify the clinical presentation and management of closed head injuries
- Identify the clinical presentation of blast head injuries

Agenda

- Identify the clinical presentation and management of brain injuries
- Identify the Clinical Presentation and management of moderate to severe TBI
- Identify the Clinical Presentation and management of mild TBI
- Participate in a head trauma case review
- Mace Demonstration
Reason

References
- USASOC Neurocognitive Testing and Post-Injury Evaluation and Treatment Clinical Practice Guideline (CPG)
- USASOC Guidelines For Post-Concussion Rehabilitation
- USASOC mTBI Symptom Cluster Treatment Recommendations
- USASOC Concussion Patient Information Sheet

References
- Tintinalli’s Emergency Medicine, 7th edition, 2010
- Tactical Trauma Protocols
- Tactical Medical Emergency Protocols
- PHTLS, 7th edition, 2010
- USASOC Policy 10-10 Neurocognitive Testing Program
Terminal Learning Objective

- **Action:** Communicate knowledge of head trauma
- **Condition:** Given a lecture in a classroom environment
- **Standard:** Received a minimum score of 75% on the written exam IAW course standards

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Head Trauma
PFN: SOMEML35

- **Hours:** 3.0
- **Instructor:**