Author(s): Patrick Carter, Daniel Wachter, Rockefeller Oteng, Carl Seger, 2009-2010.

License: Unless otherwise noted, this material is made available under the terms of the Creative Commons Attribution 3.0 License:
http://creativecommons.org/licenses/by/3.0/

We have reviewed this material in accordance with U.S. Copyright Law and have tried to maximize your ability to use, share, and adapt it. The citation key on the following slide provides information about how you may share and adapt this material.

Copyright holders of content included in this material should contact open.michigan@umich.edu with any questions, corrections, or clarification regarding the use of content.

For more information about how to cite these materials visit http://open.umich.edu/education/about/terms-of-use.

Any medical information in this material is intended to inform and educate and is not a tool for self-diagnosis or a replacement for medical evaluation, advice, diagnosis or treatment by a healthcare professional. Please speak to your physician if you have questions about your medical condition.

Viewer discretion is advised: Some medical content is graphic and may not be suitable for all viewers.
Citation Key
for more information see: http://open.umich.edu/wiki/CitationPolicy

Use + Share + Adapt

{ Content the copyright holder, author, or law permits you to use, share and adapt. }

- **Public Domain – Government**: Works that are produced by the U.S. Government. (USC 17 § 105)
- **Public Domain – Expired**: Works that are no longer protected due to an expired copyright term.
- **Public Domain – Self Dedicated**: Works that a copyright holder has dedicated to the public domain.
- **Creative Commons – Zero Waiver**
- **Creative Commons – Attribution License**
- **Creative Commons – Attribution Share Alike License**
- **Creative Commons – Attribution Noncommercial License**
- **Creative Commons – Attribution Noncommercial Share Alike License**
- **GNU – Free Documentation License**

Make Your Own Assessment

{ Content Open.Michigan believes can be used, shared, and adapted because it is ineligible for copyright. }

- **Public Domain – Ineligible**: Works that are ineligible for copyright protection in the U.S. (USC 17 § 102(b)) *laws in your jurisdiction may differ

{ Content Open.Michigan has used under a Fair Use determination. }

- **Fair Use**: Use of works that is determined to be Fair consistent with the U.S. Copyright Act. (USC 17 § 107) *laws in your jurisdiction may differ

  Our determination **DOES NOT** mean that all uses of this 3rd-party content are Fair Uses and we **DO NOT** guarantee that your use of the content is Fair.

  To use this content you should **do your own independent analysis** to determine whether or not your use will be Fair.
Advanced Emergency Trauma Course

Head Injury

Presenter: Patrick Carter, MD

Ghana Emergency Medicine Collaborative
Patrick Carter, MD • Daniel Wachter, MD • Rockefeller Oteng, MD • Carl Seger, MD
Lecture Objectives

- Epidemiology of Head Injury
- Definition
- Pathophysiology
- Mechanisms of Injury
- Clinical Features
- Evaluation of the Head Injured Patient
- Management of Head Injury
- Sequelae of Head Injury
Epidemiology

United States

- 1.4 million annual incidents of TBI
  - 50,000 die from TBI
  - 235,000 are hospitalized
  - 1.1 million are treated and released from ED
- Peak Incidence = 15-24 years old (50%)
  - Smaller peaks in elderly and children
  - Children – typically result of child abuse
- High cost to society in terms of lost productivity and money required to care for patients in long term facilities

Ghana

- Epidemiology of Head Injury Unknown
- RTA are significant problem and cause of mortality
Epidemiology

- **Etiology of Head Injury (U.S.)**

  - Falls: 28%
  - Motor Vehicle Traffic: 20%
  - Struck by/against: 19%
  - Assault: 11%
  - Other: 9%
  - Pedal Cycle (Non Motor Vehicle): 3%
  - Other Transport: 2%
  - Suicide: 1%
  - Unknown: 9%
  - Other: 7%

Ghana Emergency Medicine Collaborative
Advanced Emergency Trauma Course
Definition

- Traumatic Brain Injury has long been recognized as an important medical entity
  - Hippocrates first commented on mechanisms of head injury and first described trephination as modality to treat head injury
  - 16th century – French military surgeon Ambrose Pare introduced term *commotio cerebri* to describe mild head injury to brain
  - Traumatic Brain Injury first came into use in 1996 after U.S. based *Traumatic Brain Injury Act* which established federal funding for study of brain injury
Definition

• Traumatic Brain Injury
  • Spectrum of Intracranial Injury
  • Results from:
    ■ Direct Forces = Object Striking or penetrating cranium
    ■ Indirect Forces = Acceleration/Deceleration or Rotational Mechanism

• Traumatic Brain Injury Scale
  ■ Glasgow Coma Scale = 30 minutes after head injury
    • Mild = 14-15, Moderate = 9-13, Severe < 9
    • Dynamic Scale
    • Over 25 different scales available
Definition

- Concussion
  - Concussion represents a subset of mild traumatic brain injury
  - Derived from Latin term “Concutere” or to shake violently
  - Historically, defined by the loss of consciousness
  - 1965 Consensus Definition = Congress of Neurological Surgeons
    - “A clinical syndrome characterized by the immediate transient post-traumatic impairment of neural function such as alteration of consciousness, disturbance of vision or equilibrium due to brainstem involvement”
    - Definition recognized as has many limitations
Concussion Definition

- American Academy of Neurology and the International Conference on Concussion in Sport held in 2004 created a modified consensus definition of concussion as “a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces that typically includes:
  - Concussion may be caused by either a direct blow to the head, face, neck or elsewhere on the body with an “impulsive” force transmitted to the head.
  - Concussion typically results in the rapid onset of short lived impairment of neurologic function that resolves spontaneously.
  - Concussion may result in neuropathologic changes but the acute clinical symptoms largely represent a functional disturbance rather than structural injury.
  - Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
  - Concussion is typically associated with grossly normal structural imaging studies.”
Normal Cerebral Autoregulation

- Brain is a semisolid organ that occupies 80% if 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)
Normal Cerebral Autoregulation

- CPP = CBF = MAP – ICP
- Cerebral Autoregulation
  - Protective mechanism to maintain a tightly controlled environment where fluctuations in systemic arterial pressure or ICP do not have a large impact on cerebral blood flow
  - Maintained by intact blood brain barrier, a specialized set of endothelial cells with tight junctions
  - Disruption of the blood brain barrier by traumatic injury may impair normal cerebral autoregulation
Pathophysiology

Traumatic Brain Injury

- Primary Brain Injury
  - Types of Primary Brain Tissue Injury
  - Cellular Injury Mechanisms
- Secondary Brain Injury
  - Systemic Insults
  - Intracranial Insults
- Mechanisms of Traumatic Brain Injury
  - Skull Fractures
  - Extra-axial Fluid Collections
  - Intraparenchymal Hemorrhage
  - Subarachnoid Hemorrhage
Primary Brain Injury

- Primary Brain Injury
  - Direct or indirect force to brain tissue, resulting in cellular injury

Types of Primary Brain Injury

- Cortical Brain Contusion
  - Shear Stress 2/2 Coup/Contracoup injury
  - Focal injury at gray matter closest to the brain surface generates localized brain edema and disruption of normal neurological function
  - Size of contusion defines extent of injury

- Diffuse Axonal Injury
  - Rotational Mechanism –
    - Widespread shearing strain at deep cerebral white matter that disrupts normal axonal organization resulting in disruption of axonal fibers and myelin sheaths
  - Non-lateralizing neurological deficits
  - Generalized edema occurs after injury, typically within 6 hours without any focal lesion on CT imaging
Primary Brain Injury – Cellular Level

Primary Cellular Injury

- Massive Depolarization of Brain Cells
- “Neurotransmitter Storm”

Glutamate

- Calcium
- NMDA

Intracellular Signaling Processes

- Disruption of normal cellular processes:
  - Protein Phosphorylation
  - Microtubule Construction
  - Enzyme Production

Oxygen Free Radical Pathway Activation

- High Nitric Oxide Levels
- Nitric Oxide Synthase

Lipid Peroxidation

- Cell Membrane Dysfunction
- Cell Lysis

Cell Death

- Membrane and Cytoskeleton Breakdown
Secondary Brain Injury

- Secondary Brain Injury
  - Systemic or Intracranial processes that contribute to the primary brain injury cycle and results in greater tissue injury
  - Categorized into:
    - Systemic Insults
    - Intracranial Insults

- Emergency Department Treatment
  - Focused on limiting the extent of secondary brain injury
Secondary Brain Injury

- **Systemic Insults**
  - Hypoxia (PaO2 < 60 mmHg)
    - Mortality of TBI pts with hypoxia = doubled
    - 40% of TBI ED patients exhibit hypoxia during course
  - Hypotension (SBP < 90 mmHg)
    - Present in 33-35% of TBI patients
    - Results from hemorrhagic shock, cardiac contusion, tension pneumothorax, etc
    - Hypotension $\rightarrow$ ↓Cerebral Perfusion $\rightarrow$ ↑Cerebral Ischemia $\rightarrow$ ↑Doubles Mortality
  - Anemia 2/2 Blood Loss (↓ Oxygen Carrying Capacity)
  - Hypo/Hypercapnia
    - Hyperventilation $\rightarrow$ ↓pCO$_2$ Levels $\rightarrow$ ↑Serum pH $\rightarrow$ Cerebral Vasoconstriction $\rightarrow$ ↓Cerebral Blood Flow
    - Previously was a mainstay of treatment and will help to buffer an expanding hematoma in short-term, but will ultimately decrease cerebral perfusion to penumbra region and increase tissue death
Secondary Brain Injury

- **Other Systemic Insults**
  - Seizures
  - Electrolyte Abnormalities
  - Coagulopathy
  - Infection
  - Hyperthermia
  - Iatrogenic (Under-resuscitation)

- **Intracranial Insults**
  - Intracranial Hypertension
  - Extra-axial Lesions
  - Cerebral Edema (Peaks at 24-48 hrs post injury)
Mechanisms of Injury

- Mechanisms of Injury
  - Mediators of 1\(^{\circ}\) and 2\(^{\circ}\) Injury

- Skull Fractures

- Extra-axial Lesions
  - Epidural Hematoma
  - Subdural Hematoma

- Intraparenchymal Hemorrhage

- Subarachnoid Hemorrhage
Skull Fractures

- Skull Fracture = High Degree of Energy

Classification:
- Location
- Pattern of fracture
- Open vs. Closed

Location may indicate underlying injury:
- Depressed skull fracture = Often tear underlying dural tissue
- Fracture over pteryion = Middle Meningeal Artery = EDH
- Fracture over dural sinus = Subdural Hematoma

Presence of skull fracture increases risk of intracranial bleeding 174 times compared to patients without a skull fracture
Extra-axial Fluid Collections

- **Epidural Hematoma**
  - Middle Meningeal Artery (36%)
  - Head Injury w/ LOC + Lucid Interval followed by deterioration
    - Classic presentation = 47% of cases
  - Lenticular Shape on CT

- **Subdural Hematoma**
  - Injury to Bridging Veins
  - Blood accumulation between dura mater and pia arachinoid mater
  - Increased risk in elderly and alcoholics due to decreased brain volume
  - Hyperdense crescent shaped lesion
Intracerebral Hemorrhage

- Intraparenchymal Hemorrhage
- Subarachnoid Hemorrhage
  - Disruption of subarachnoid vessels
  - Common in moderate to severe brain injury
  - Worse prognosis
    - Twice as likely as other head injured patients to suffer from death, persistent vegetative state or severe disability

Images from http://www.radiology.co.uk/srs-x/tutors/cttrauma/tutor2.htm

Ghana Emergency Medicine Collaborative
Advanced Emergency Trauma Course
Clinical Features

- Traumatic Brain Injury
  - Spectrum of clinical presentations
  - Hallmark Symptoms
    - Confusion and amnesia w/ or w/o LOC
  - Severe brain injury often characterized by decreased mental status and presence of neurological deficits
  - Patients may also deteriorate from mild to severe head injury during course of evaluation
Clinical Features

- **Confusion**
  - Characterized by three cardinal features
    - Disturbance of vigilance and heightened distractibility
    - Inability to maintain a coherent train of thought
    - Inability to carry out a sequence of goal directed movements

- **Amnesia**
  - May be anterograde or retrograde
  - Often characterized by repetitive questioning, inability to follow commands, inability to retain information during medical evaluation
  - Amnesia will decrease slowly over time and small amount of memory deficit remains
  - No loss of biographical data
    - i.e. Name, etc. – typically the result of hysterical rxn or malingering
  - Duration does correlate with severity and outcome of head injury

- **Loss of Consciousness**
  - Results from rotational forces at the junction of the upper midbrain and thalamus that results in disruption of reticular neuron function and inability to maintain alertness
  - Presence of LOC is not a predictor of long term neuropsychiatric sequelae of head injury
Clinical Features

- **Glasgow Coma Scale**
  - Developed by Teasdale and Jennett in 1974
  - Originally designed for measure 6 hours after injury to provide long term prognostic information about mortality and disability
  - Now, standardized to measure 30 min after injury and repetitive measurements throughout patient’s stay
  - Should be performed after adequate resuscitation b/c scale is sensitive to hypotension, hypoxia, intoxication and pharmacologic interventions
  - Current Classification
    - GCS = 14-15 = Mild Head Injury
    - GCS = 9 – 13 = Moderate Head Injury
    - GCS < 9 = Severe Head Injury
  - Best prognostic indicator of outcome = CT Scan
Clinical Features

- **Glasgow Coma Scale**

<table>
<thead>
<tr>
<th>Glasgow Coma Scale (GCS)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye Opening</strong></td>
<td></td>
</tr>
<tr>
<td>Opens spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>Responds to verbal command</td>
<td>3</td>
</tr>
<tr>
<td>Responds to pain</td>
<td>2</td>
</tr>
<tr>
<td>No eye opening</td>
<td>1</td>
</tr>
<tr>
<td><strong>Verbal</strong></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Disoriented</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible speech</td>
<td>2</td>
</tr>
<tr>
<td>No verbal response</td>
<td>1</td>
</tr>
<tr>
<td><strong>Motor</strong></td>
<td></td>
</tr>
<tr>
<td>Obey commands</td>
<td>6</td>
</tr>
<tr>
<td>Localizes to pain</td>
<td>5</td>
</tr>
<tr>
<td>Withdraws to pain</td>
<td>4</td>
</tr>
<tr>
<td>Flexion to pain (Decorticate posturing)</td>
<td>3</td>
</tr>
<tr>
<td>Extension to pain (Decerebrate posturing)</td>
<td>2</td>
</tr>
<tr>
<td>No motor response</td>
<td>1</td>
</tr>
</tbody>
</table>
Clinical Features

- **Neurologic Exam**
  - 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
  - Neurological Posturing
    - Decorticate Posturing = Upper extremity flexion with lower extremity extension
      - Cortical Injury above the midbrain
    - Decerebrate Posturing = Arm extension and internal rotation with wrist flexion
      - Indicative of brainstem injury
      - Very Poor predictor of outcome
  - Full, Complete Neurological Exam
    - Examine for subtle neurological deficits
    - Look for specific injury patterns:
      - Battle’s sign, CXF Otorrhea, CSF Rhinorrhea, Hemotympanum, peri-orbital Ecchymosis is indicative of skull fracture and is concerning for underlying brain injury
Clinical Features

- **Mild Head Injury**
  - Signs and symptoms (early/late)

<table>
<thead>
<tr>
<th>Signs and Symptoms of Head Injury</th>
<th>Cognitive</th>
<th>Somatic</th>
<th>Affective</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confusion</td>
<td>Headache</td>
<td>Emotional Lability</td>
<td></td>
</tr>
<tr>
<td>Anterograde amnesia</td>
<td>Fatigue</td>
<td>Irritability</td>
<td>Sadness</td>
</tr>
<tr>
<td>Retrograde amnesia</td>
<td>Disequilibrium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>Dizziness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disorientation</td>
<td>Nausea/vomiting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feeling “zoned out”</td>
<td>Visual disturbances</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feeling “foggy”</td>
<td>Photophobia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vacant stare</td>
<td>Phonophobia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inability to focus</td>
<td>Difficulty sleeping</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delayed verbal/motor response</td>
<td>Ringing of the ears</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slurred or incoherent speech</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excessive Drowsiness</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Clinical Features

- Grading scale for mild head injury with GCS between 14-15 and concussion syndrome
  - Developed by Colorado Medical Society and American Academy of Neurology
  - Composite Grading System
    - Grade 1 – Any head injury with transient confusion, no LOC and symptoms that last less than 15 minutes
    - Grade 2 – Transient confusion, no LOC, symptoms that last longer than 15 minutes
    - Grade 3 – All head injury with LOC
Clinical Features

- Moderate
  - GCS = 9-13
  - Clinical presentation varies widely
  - 10% of patients
  - Specialized Subset = “Talk and Die Syndrome”
    - Initially, talkative and without significant signs of external injury
    - Within 48 hours of injury, rapidly deteriorate
    - Epidural Hematoma is cause in 78-80% of cases
    - Patients with “talk and die syndrome” who present with a GCS > 9 but who deteriorate have been shown to have a worse outcome than patients who present with severe TBI at outset
      - ? Delayed Diagnosis
Severe Head Injury

- GCS < 9
- 10% of patients with TBI
- Early aggressive treatment is required with airway control, resuscitation, admission to ICU setting
- 25% of this patient population will require neurosurgical intervention
- Outcome is poor with mortality as high as 60%
- Exam typically with abnormal exam, often evidence of external trauma, abnormal pupillary exam and neurological deficits
- Cushing’s Triad = Acute entity seen in severely head injured patients with significant increased intracranial pressure and impending herniation
  - Results from ischemia to hypothalamus with poor perfusion to the brain, resulting in sympathetic stimulation of the heart to correct poor perfusion. The sympathetic stimulation results in hypertension, but carotid baroreceptors respond with parasympathetic stimulation resulting in bradycardia
  - Characterized by:
    - Progressive Hypertension
    - Bradycardia
    - Irregular or impaired respiratory pattern
Evaluation of the Head Injured Patient

■ Sideline Evaluation
  • Head Injury may not be recognized by an injured player or non-medical personnel
  • Multiple Standardized tools for evaluation of head injured sports player
    - Standardized Assessment of Concussion (SAC)
    - Sport Concussion Assessment Tool (SCAT)

■ E.D. Evaluation
  • Neurological Exam
  • Imaging
Neuroimaging

- Skull Radiography
- CT Scan (Gold Standard)
- Magnetic Resonance Imaging (MRI)
- Experimental Modalities for Neuroimaging
  - Functional MRI (fMRI)
  - PET Scanning
  - SPECT Scanning
  - Magnetic Source Imaging (MSI)
Neuroimaging

Skull Radiography

- Prior to CT, Skull radiography used as triage tool
- Can evaluate for
  - Skull fractures
  - Pneumocephalus
  - Blood in sinus
  - Penetrating foreign body
- Patients with abnormal findings are at increased risk of intracranial findings
- However, still misses a large number of patients with normal skull films but extensive injury
- Limited utility at very rural sites without access to CT imaging
Neuroimaging

- Computed Tomography (CT Scan)
  - Imaging modality of choice
  - Especially good at identifying skull fracture, extraxial fluid collection and hemorrhagic contusion
  - CT imaging has increased in United States 120% from 1990 to 2000
  - High utilization has led to clinical decision rules to identify appropriate patients requiring evaluation
    - New Orleans Criteria
    - Canadian Head CT Rule
### New Orleans Criteria
CT imaging is required for patients with minor head injury with any one of the following findings. The Criteria only apply to patients who have a GCS of 15.

1. Headache
2. Vomiting
3. Age > 60 years
4. Drug or Alcohol Intoxication
5. Persistent anterograde amnesia
6. Visible trauma above the clavicle
7. Seizure

### Canadian CT Head Rule
CT Imaging is only required for patients with minor head injury with any one of the following findings. The criteria apply to patients with minor head injury who present with GCS of 13-15 after witnessed LOC, amnesia or confusion.

#### High Risk for Neurosurgical Intervention
1. GCS < 15 at two hours after injury
2. Suspected open or depressed skull fracture
3. Any sign of basilar skull fracture (Hemotympanum, Peri-orbital Eccymosis, Otorrhea or Rhinorrhea, Battle sign)
4. Two or more episodes of vomiting
5. Age > 65 years

#### Medium risk for Brain Injury Detection by CT Imaging
1. Amnesia before impact of 30 or more minutes
2. Dangerous mechanism (E.g. Pedestrian vs. Motor vehicle, Ejection from motor vehicle or fall from an elevation of 3 or more feet or 5 stairs)
Neuroimaging

- Head CT Clinical Rules
  - New Orleans Criteria
    - Sensitivity and Specificity of detecting a clinically significant CT finding
    - Sensitivity = 100%
    - Specificity = 24.5%
    - Estimated to decrease CT imaging by 23%
  - Canadian Head CT Rule
    - Sensitivity and Specificity for need for neurosurgical intervention and clinically significant finding on CT imaging
    - Sensitivity = 100%
    - Specificity = 68%
    - Proposed to reduce CT scanning by 46%
  - Both decision rules have subsequently been validated
Management

- Mild Head Injury
  - Admission Criteria
  - Discharge Criteria
  - Discharge Instructions
  - Return to Play Guidelines

- Moderate and Severe Head Injury
  - General Principles
  - Airway Management
  - Hemodynamic Assessment
  - Seizure Prophylaxis
  - Operative Management
  - Intracranial Monitoring
Mild Head Injury Management

Management

• Symptomatic treatment and prevention of secondary injury
• Appropriate management depends on assessment of risk of neurological decompensation and risk factors for intracranial hematoma
• Risk factors for intracranial hematoma
  ■ Coagulopathy, Drug/Alcohol Intoxication, Previous neurosurgical procedures, Pre-trauma epilepsy or older age (> 60 y/o)
• Low risk features
• All patients with mild traumatic brain injury should be observed for 24 hours after that injury (either inpatient or outpatient).
Mild Head Injury Management

- Admission Criteria
  - Hospital Admission is required for all patients at higher risk for complications including:
    - GCS < 15
    - Abnormal CT Scan
    - Seizure Activity
    - Abnormal Bleeding Parameters (Anticoagulation or bleeding diathesis)
    - Unable to be observed at home

- Discharge Criteria
  - Low risk patients can be discharged home with oral and written discharge instructions
  - Patients can be discharged if:
    - GCS = 15
    - Normal neurological exam
    - Normal Head CT
    - No predisposition for bleeding
Mild Head Injury Management

- **Discharge Instructions**
  - Appropriate follow-up instructions should be provided both verbally and written instructions.
  - No need to awaken patient q 2 hours.
  - Patients who return to ED due to persistent symptoms should undergo careful repeat neurological evaluation but little data supports repeat CT Scanning.

- **Return to Play Guidelines**
  - Patients should return to sporting activities in a step-wise fashion that emphasizes physical and cognitive rest.
  - Patients should not return to sporting events if they are still symptomatic.
  - There are many commonly used tools for assessing a player's ability to return to sporting events.

---

**Warning Signs after Discharge**

- Inability to awaken the patient
- Decreased/Altered mental status
- Severe or worsening headache
- Somnolence or confusion
- Restlessness, Unsteadiness
- Seizure activity
- Visual difficulties
- Change in behavior
- Vomiting, fever, neck stiffness
- Urinary or bowel incontinence
- Weakness or numbness
Mod/Severe Head Injury Management

- **General Principles**
  - All moderate and severe head injured patients should undergo CT imaging
  - Stabilization and prevention of secondary insults is mainstay of treatment

- **Airway Management**
  - Prevention of hypoxia and hypoventilation key to preventing secondary insults
  - Patients with GCS < 9, should have endotracheal airway placed
  - Rapid Sequence Intubation is preferred method of intubation
  - Nasotracheal Intubation contraindicated due to tendency for ICP to increase 2/2 cough/gag
  - Lidocaine for prevention of increased ICP has not been shown to have a benefit
  - Special attention should be paid to maintaining cervical spinal immobilization
Hemodynamic Assessment

- Hypotension (SBP < 90) should be aggressively treated as a significant cause of worse outcome.
- Rarely, hypotension is due to head injury itself and other traumatic injuries should be investigated.
- Treatment of hypotension is directed at maintenance of cerebral perfusion.
  - Hypotonic fluids are contraindicated.
  - Typically isotonic fluids are used (NS).
Sedatives, Analgesia and Neuromuscular Blockade

- Agitation is common finding and may result from pain, delirium or difficulties with oxygenation and ventilation
- Minimizing agitation should be goal to limit increases in ICP or inabilities to oxygenate and ventilate
- Typically, short acting opiates and benzodiazepines are utilized to decrease agitation
- Long term sedation should be accompanied by sedation holidays to evaluate neurological exam (Diprovan, Midazolam)
- Barbiturates are not typically used in the emergency department but do have a limited role in long term management of patients with increased ICP who require sedation and have failed other medical and surgical treatments for increased ICP.
- Neuromuscular blockade is indicated for airway control with RSI but long acting blocking agents should be avoided because they limit serial examinations
Mod/Severe Head Injury Management

Seizure Prophylaxis

• Post-traumatic seizures = Seizures occurring in less than 7 days post-injury
• Risk factors = GCS < 10, Cerebral Contusion, Depressed Skull Fracture, EDH, SDH, Intracerebral bleeding, Penetrating head injury or seizure activity within 24 hours of injury
• Brain Injury Foundation recommends anti-epileptic medications be administered to high risk patients for first 7 days post-injury
• Acute management of seizure activity is managed with benzodiazepines and other typical anti-epileptic agents
• No proven benefit to administration of anti-epileptic meds after 7 days to decreasing post-traumatic epilepsy
Operative Management

- Indications
  - Penetrating injuries or blunt injuries with breach of the calvarium/skull
  - Presence of expanding intracranial hematoma
    - Epidural Hematoma
      - If volume > 30 cm$^3$ or if comatose (GCS < 9)
    - Subdural Hematoma
      - If size > 10 mm on CT or if 5 mm shift regardless of GCS score
      - Decompression if GCS decreases by 2 points from time of injury to hospital arrival
      - ICP > 20 mmHg or if pt with fixed, dilated pupils

- Malignant cerebral edema
Mod/Severe Head Injury Management

- Operative Management
  - Decompressive Craniotomy
    - Salvage operation used to manage increasing ICP
    - Removal of part of skull and underlying dura
    - Decreases ICP, improves cerebral perfusion, prevents ischemia
    - Serves to limit secondary insults
    - Literature divided on true benefit
Intracranial Monitoring

- Developed in 1960’s for close monitoring of ICP in intubated patients
- Indications
  - Severe TBI with GCS < 9
  - Intubated patients with moderate or severe head injury with significant intracranial findings on CT

Methods of Monitoring

- External Ventricular Drain
  - Blind placement of catheter through brain parenchyma into lateral ventricle with transducer to measure pressure
  - Can also drain excess CSF in high ICP patients
- Subdural Bolt Catheter
  - Technically easier than ventriculostomy
  - Monitoring bolt placed beneath the dura into the subarachnoid space
  - Doesn’t allow for CSF drainage
- Fiber optic Catheter
  - Similar to a Subdural bolt but provides more accurate readings
Increased Intracranial Pressure

- Most frequent cause of death and disability after severe head injury
- Identified in any patient with clinical signs of impending herniation, Cushing triad or rising ICP as identified by intracranial monitoring techniques
- Recommended ICP < 20 mmHg with CPP > 60 mmHg
- Initial First line treatment of increased ICP
  - HOB – 30 degrees
- Subsequent first line treatment measures
  - Short term hyperventilation
  - Osmotic diuretic administration (i.e. Mannitol, Hypertonic saline)
- Second Line Treatments
  - High dose barbiturates
  - Severe hyperventilation
  - Mild/moderate hypothermia
  - Decompressive craniotomy
Emergency Burr Hole Trephination

- **Indication**
  - Patient w/ TBI with evidence of rapid deterioration and signs of impending transtentorial herniation with expected delay in neurosurgical management
  - Performed after medical management of increased ICP has failed
  - Ideally, hematoma is localized by CT imaging but can be performed as blind procedure
    - 85% of the time will be on the same side as the dilated pupil

- **Procedure**
  - Landmark = 6 cm anterior and superior to the tragus of the ear over the tempoparietal region
  - Vertical incision is made through the scalp, subcutaneous tissue and temporalis muscle until galea aponeurotica is reached
  - A rotary or twist drill is then used to breach the inner table of the cranium and the site is examined
  - EDH will present as immediate clot and bleeding before the dura is reached
  - SDH will present as dark bluish mass of blood beneath the bulging dura
  - In the case of a SDH, the dura will need to be incised with a scalpel and subdural blood will need to be suctioned from the site
  - If no blood is identified, additional holes are made superior to the initial site and if blood is still not identified, then the opposite side is attempted
  - All sites should be covered with a sterile non-occlusive dressing
Second Impact Syndrome

- Rare, Controversial entity
- Athlete who has sustained a mild concussion who subsequently suffers a second head injury before the symptoms from the first have resolved
- Patients subsequently develop rapid diffuse cerebral edema (within 2 min), increased ICP and eventual herniation, coma and death
- The first head injury is postulated to cause a disruption of the normal cerebral vascular autoregulation that causes increased cerebral blood flow, making the brain vulnerable to the second impact, when the rapid malignant swelling occurs
- Return to play guidelines have been developed to prevent this type of secondary injury
Sequelae of Head Injury

- **Post Concussive Syndrome**
  - Constellation of symptoms that develops within 4 weeks of the injury and may persist for months (90% at 1 month, 25% at 1 year)
  - Treatment is with analgesia, anti-depressents and anti-emetics

- **Post-traumatic Epilepsy**
  - Seizure activity > 7 days from traumatic injury
  - Head trauma is cause of long term epilepsy in 3% of patients with epilepsy
  - Incidence is highest in patients with compound skull fracture, intracranial hemorrhage or presence of early acute symptomatic seizure (presence of all 3 factors increases risk by 50-80%)
  - Cannot be prevented with prophylactic use of antiepileptics

- **Persistent Vegetative State**
  - Rare complication of severe head injury, first described in 1972 by Jennett and Plum
  - Disruption of cerebral cognitive function with sparing of brainstem function
  - No awareness of themselves or environment and cannot interact with others but will maintain normal sleep-wake cycle
  - Recovery is rare if symptoms persist for > 3 months, no recovery documented after 12 months of symptoms
Questions?
References


References


References


