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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

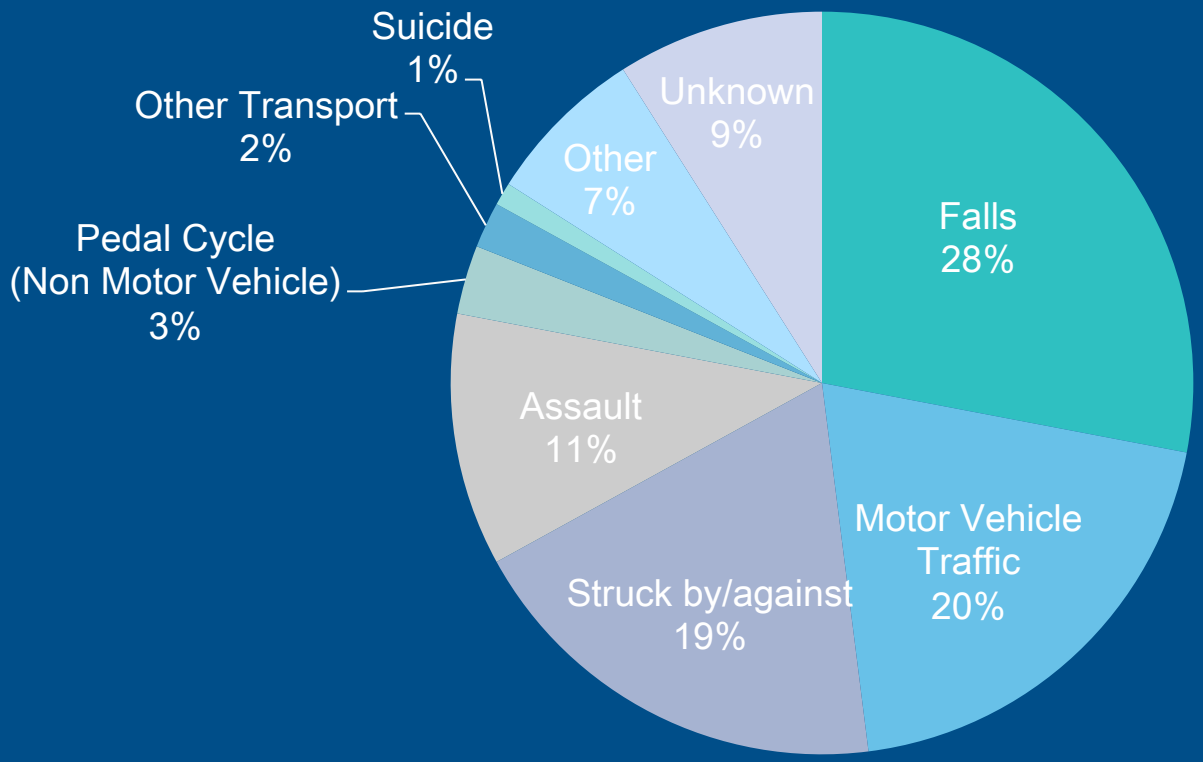
- Epidemiology of Head Injury
- Definition
- Pathophysiology
- Mechanisms of Injury
- Clinical Features
- Evaluation of the Head Injured Patient
- Management of Head Injury
- Sequelae of Head Injury

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

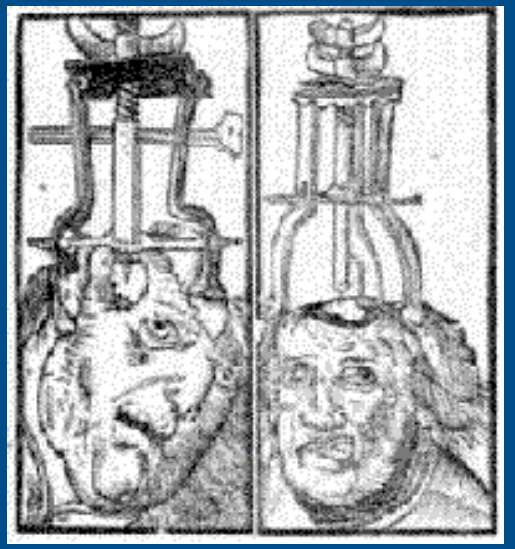
Epidemiology

- Etiology of Head Injury (U.S.)



Definition

- Traumatic Brain Injury has long been recognized as a 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



PD-EXP

http://commons.wikimedia.org/wiki/File:Trepan1b.gif

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

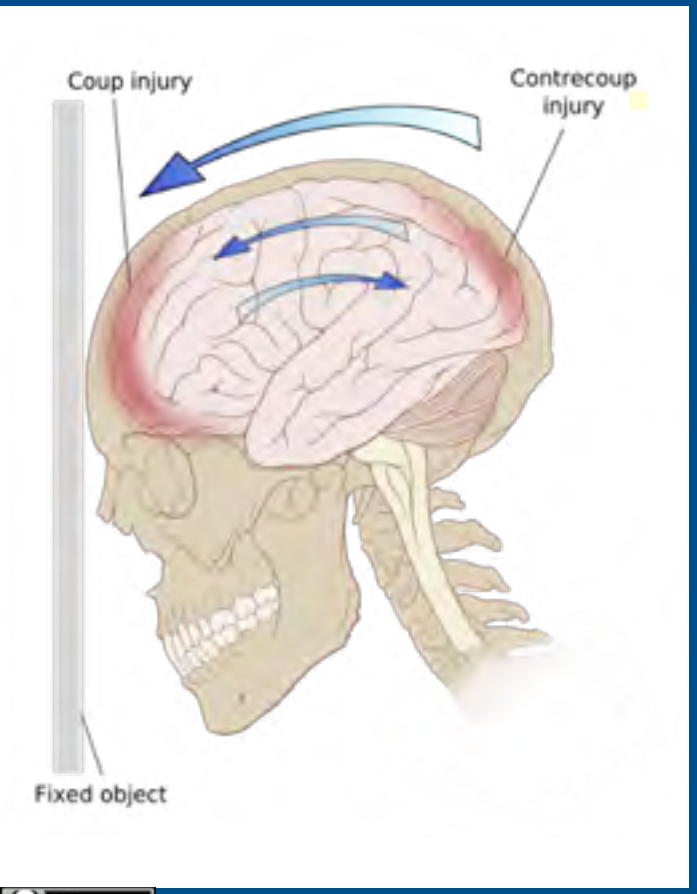
- CPP = CBF = MAP – ICP
- Cerebral Autoregulation
  - Protective mechanism to maintain a tightly controlled environment where fluctuations in systemic arterial pressure or ICP do not have a large impact on cerebral blood flow
  - Maintained by intact blood brain barrier, a specialized set of endothelial 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

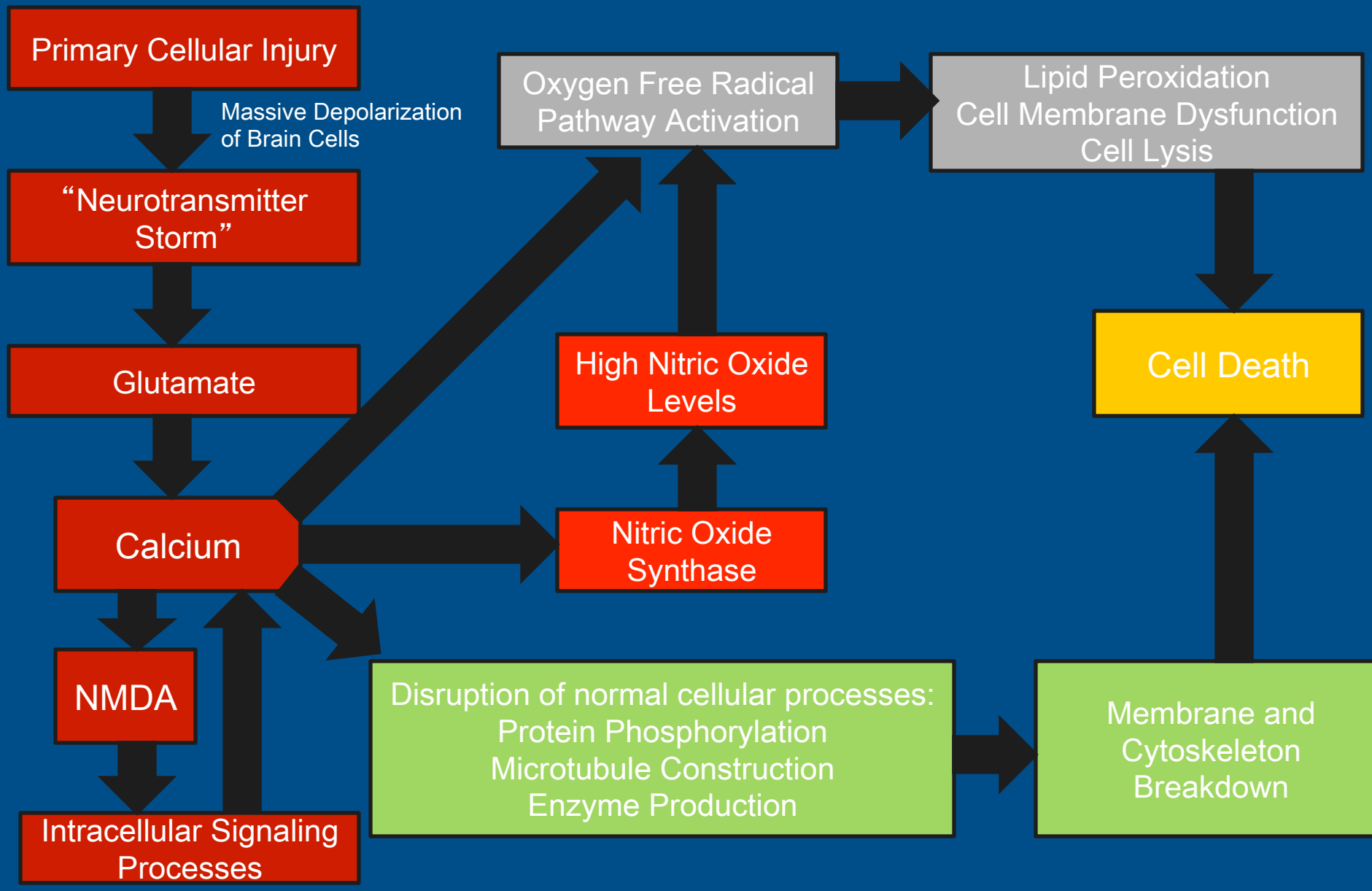


Patrick J. Lynch; illustrator; C. Carl Jaffe; MD; cardiologist (Wikipedia)

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



Secondary Brain Injury

- Secondary Brain Injury
  - Systemic or Intracranial processes that contributes 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 (PaO_2 < 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 → ↓Cerebral Perfusion→↑Cerebral Ischemia →↑Doubles Mortality
  - Anemia 2/2 Blood Loss (↓Oxygen Carrying Capacity)
  - Hypo/Hypercapnia
    - Hyperventilation → ↓pCO_2 Levels → ↑Serum pH → Cerebral Vasoconstriction → ↓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^{o} and 2^{o} 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

PD-INEL

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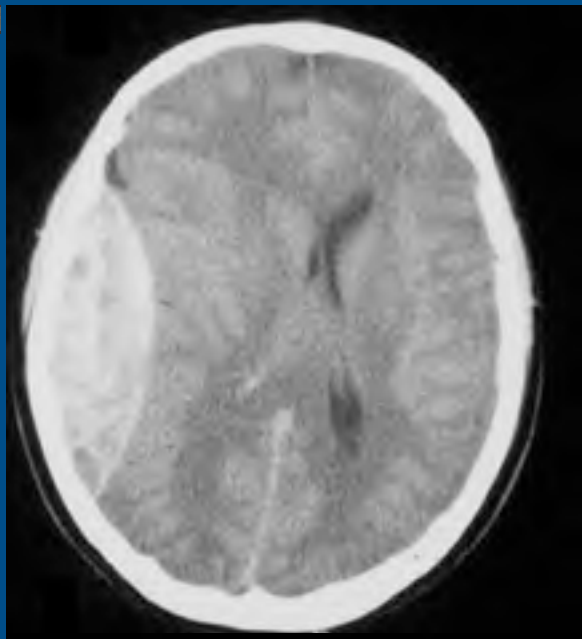
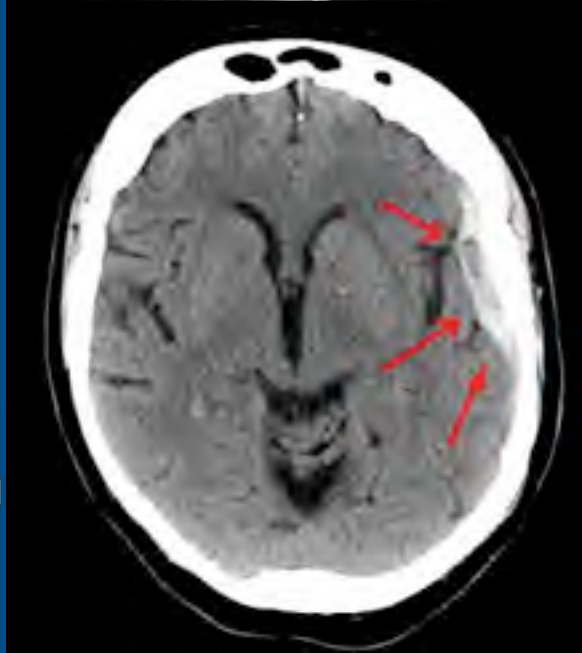


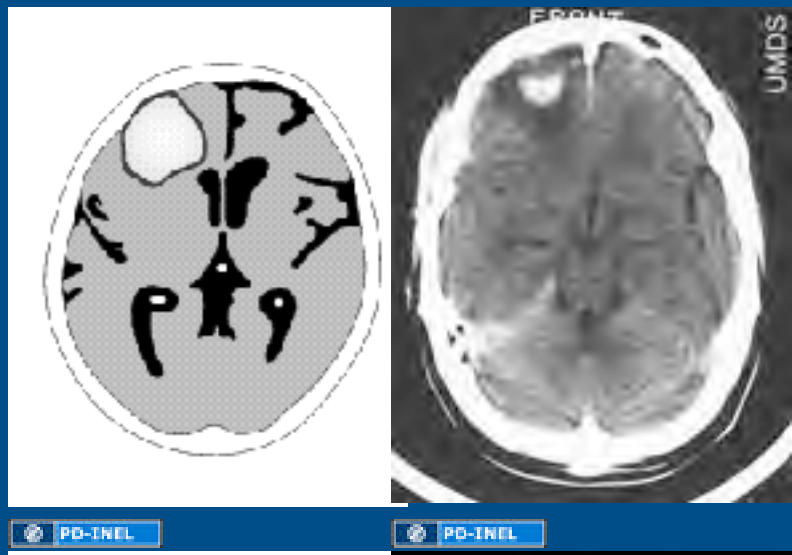


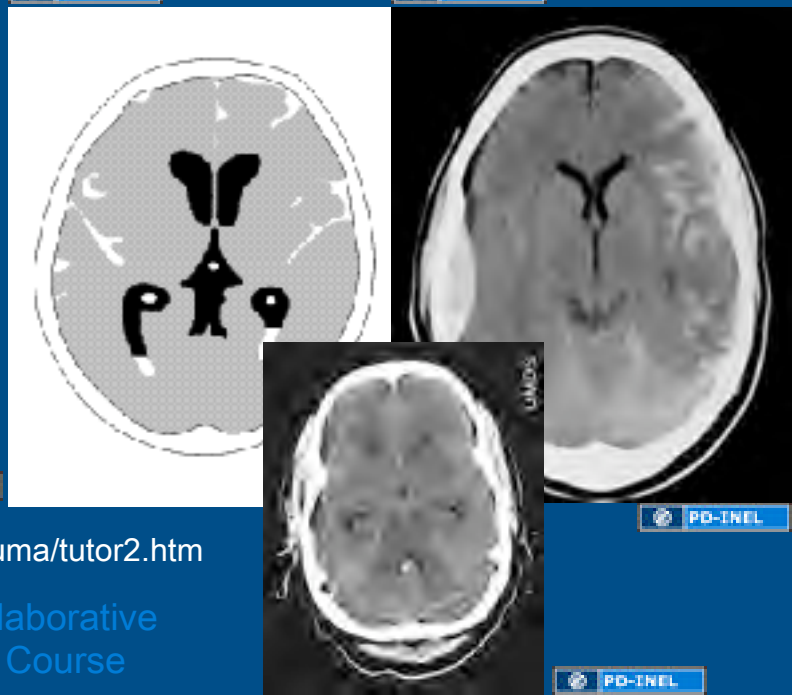
PD-INEL

Wikipedia

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

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

Glasgow Coma Scale (GCS)		
Eye Opening	Opens spontaneously	4
	Responds to verbal command	3
	Responds to pain	2
	No eye opening	1
Verbal	Oriented	5
	Disoriented	4
	Inappropriate words	3
	Incomprehensible speech	2
	No verbal response	1
Motor	Obeys commands	6
	Localizes to pain	5
	Withdraws to pain	4
	Flexion to pain (Decorticate posturing)	3
	Extension to pain (Decerebrate posturing)	2
	No motor response	1

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)

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

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, extra-axial 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. Pedestrican 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

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

- 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 players ability to return to sporting events.

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 tendancy 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

Mod/Severe Head Injury Management

- Hemodynamic Assessment
  - Hypotension (SBP < 90) should be aggressively treated as 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)

Mod/Severe Head Injury Management

- 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

Mod/Severe Head Injury Management

- 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

- 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

- 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 tempopareital 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

Sequelae of Head Injury

- 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?

 Dkscully (flickr)

References

- 1. *Epidemiologic Aspects of Brain Injury.* **Kraus, JF, McArthur, DL.** 2, s.l. : W.B. Sauders Company, May 1996, Neurologic Clinics, Vol. 14, pp. 436 -450.
- 2. *Traumatic Brain Injury.* **Heegaard, W, Biros, M.** s.l. : Elsevier Saunders, 2007, Emergency Medicine Clinics of North America, Vol. 25, pp. 655-678.
- 3. *Trends in Hospitalization Associated with Traumatic Brain Injury.* **Thurman, D, Guerrero, J.** 10, 1999, JAMA, Vol. 282, pp. 954-957.
- 4. *Management of Traumatic Brain Injury in the Intensive Care Unit.* **Ling, GSF, Marshall, SA.** s.l. : Elsevier Saunders, 2008, Neurology Clinics, Vol. 26, pp. 409-426.
- 5. **Kirsch, TD, Lipinski, CA.** *Tintinalli's Emergency Medicine: A Comprehensive Study Guide 6th Edition.* 6th Edition. s.l. : McGraw Hill Professional, 2003. pp. 1557-1569.
- 6. *The Epidemiology and Impact of Traumatic Brain Injury: A Brief Overview.* **Langlois, JA, Rutland-Brown, W, Wald, MM.** 5, 2006, Journal of Head Trauma and Rehabilitation, Vol. 21, pp. 375-378.
- 7. *Incidence, Risk Factors adn Prevention of Mild Traumatic Brain Injury: Results of WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury.* **Cassidy, JD, Carroll, LJ, Peloso, PM et al.** 2004, Journal of Rehabilitation Medicine, Vol. Supplement 43, pp. 28-60.
- 8. *EFNS Guideline on Mild Traumatic Brain Injury: Report of an EFNS Task Force.* **Vos, PE, Battistin, L, Birbamer, G et al.** 2002, European Journal of Neurology, Vol. 9, pp. 207-219.
- 9. *Traumatic Brain Injury and Concussion in Sports.* **Kelly, JP.** 10, September 8, 1999, JAMA, Vol. 282, pp. 989-991.
- 10. *Traumatic Brain Injury.* **Zink, Brian.** 1, s.l. : B. Saunders Company, 1996, Emergency Medical Clinics of North America, Vol. 14, pp. 115 - 148.
- 11. *Sport-related Concussion in the Young Athlete.* **Russo-Buzzini, SR, Guskiewicz, KM.** s.l. : Lippincot, Williams and Wilkins, 2006, Current Opinion in Pediatrics, Vol. 18, pp. 376-382.
- 12. *The Development of Guidelines for the Management of Concussion in Sports.* **Kelly, JP, Rosenberg, JH.** 2, s.l. : Aspen Publishers, 1998, Journal of Head Trauma and Rehabilitation, Vol. 13, pp. 53-65.
- 13. *Diagnosis and Management of Concussion in Sports.* **Kelly, JP, Rosenberg, JH.** 3, March 1997, Neurology, Vol. 48, pp. 575-580.
- 14. *Traumatic Brain Injury Outcome: Concepts for Emergency Care.* **Zink, Brian.** 3, March 2001, Annals of Emergency Medicine, Vol. 37, pp. 318-332.
- 15. *Mild Traumatic Brain Injury: Neuroimaging of Sports Related Concussion.* **Mendez, CV. Hurley, RA et al.** August 2005, Journal of Neuropsychiatry and Clinical Neuroscience, Vol. 17, pp. 297-303.

References

- 15. *Mild Traumatic Brain Injury: Neuroimaging of Sports Related Concussion.* **Mendez, CV. Hurley, RA et al.** August 2005, Journal of Neuropsychiatry and Clinical Neuroscience, Vol. 17, pp. 297-303.
- 16. *Evidence Based Review of Sport-Related Concussion: Clinical Science.* **Johnston, KM, McCrory, PM et al.** 3, s.l. : Lippincott, Williams and Williams, 2001, Clinical Journal of Sports Medicine, Vol. 11, pp. 150-159.
- 17. *Summary and Agreement Statement of the 2nd International Conference on Concussion in Sport, Prague 2004.* **McCrory, P, Johnston, K et al.** 2, March 2005, Clinical Journal of Sports Medicine, Vol. 15, pp. 48-55.
- 18. *Traumatic Brain Injury.* **Parikh, S, Koch, M, Narayan, RK.** 3, 2007, International Anesthesiology Clinics, Vol. 45, pp. 119-135.
- 19. *Defining Acute Mild Head Injury in Adults: A Proposal Based on Prognostic Factors, Diagnosis, and Management.* **Servadei, Franco, Teasdale, Graham et al.** 2001, Journal of Neurotrauma, Vol. 18, pp. 657-664.
- 20. *Surgical Management of Acute Epidural Hematomas.* **Bullock, MR, Chesnut, R et al.** Supplement 2, 2006, Neurosurgery, Vol. 58, pp. 7-15.
- 21. *Traumatic Subarachnoid Hemorrhage: Our Current Understanding and its Evolution Over the Past Half Century.* **Armin SS, Colohan ART, Zhang JH.** June 2006, Neurological Research, Vol. 28, pp. 445-452.
- 22. *Practice Parameter: The management of concussion in sports (Summary Statement) - Report of the Quality Standards Committee.* **Subcommittee, Quality Standards.** 3, 1997, Neurology, Vol. 48, pp. 581-585.
- 23. *Expert Opinion and Controversies in Sports and Musculoskeletal Medicine: Concussion in the Young Athlete.* **Standaert, CJ, Herring, SA, Cantu, RC.** August 2007, Archives Physical Medicine and Rehabilitation, Vol. 88, pp. 1077-1079.
- 24. *Clinicopathological Heterogeneity in Classification of Mild Head Injury.* **Culotta, VP, Sementilli, ME et al.** 2, s.l. : Lippincott Williams and Wilkins, February 1996, Neurosurgery, Vol. 38, pp. 245-250.
- 25. *Computed Tomography and Magnetic Resonance Imaging of Mild Head Injury - Is it appropriate to classify patients with a Glasgow Coma Scale Score of 13 to 15 as "Mild Injury"?* **Uchino, Y, Okimura, Y et al.** s.l. : Springer-Verlag, 2001, Acta Neurochirurgica, Vol. 143, pp. 1031-1037.
- 26. *Concussion.* **Ropper, AH, Gorson, KC.** 2, January 11, 2007, The New England Journal Of Medicine, Vol. 356, pp. 166-172.
- 27. *Seizures and Epilepsy after Traumatic Brain Injury.* **Chadwick, David.** 9201, January 29, 2000, Lancet, Vol. 355, pp. 334-336.
- 28. *Prophylactic Antiepileptic Agents after Head Injury: A Systematic Review.* **Schierhout, G, Roberts, I.** 1, January 1998, J Neurol Neurosurg Psychiatry, Vol. 64, pp. 108-112.
- 29. *Guidelines for the Management of Severe Traumatic Brain Injury.* **Bratton, SL, Chestnut, RM, Ghajar, J et al.** Supplement 1, September 2007, Journal Of Neurotrauma, Vol. 24, pp. 1-94.
- 30. *Patients Who Talk and Deteriorate.* **Rockswold, GL, Pheley, PJ.** 6, June 1993, Annals of Emergency Medicine, Vol. 22, pp. 1004-1007.

References

- 31. *Standardized Assessment of Concussion in Football Players.* **McCrea, M, Kelly, JP et al.** 3, March 1997, Neurology, Vol. 48, pp. 586-589.
- 32. *Sensitivity and Specificity of Standardized Neurocognitive Testing Immediately Following Sports Concussion.* **Barr, WB, McCrea, M.** 2001, Journal of the International Neuropsychological Society, Vol. 7, pp. 693-702.
- 33. *Acute Effects and Recovery Time Following Concussion in Collegiate Football Players: The NCAA Concussion Study.* **McCrea, M, Guskiewicz, KM et al.** 19, November 19, 2003, JAMA, Vol. 290, pp. 2556-2563.
- 34. *External Validation of the Canadian CT Head Rule and the New Orleans Criteria for CT Scanning in Patients With Minor Head Injury.* **Smits, M, Dippel, DWJ et al.** 12, September 28, 2005, JAMA, Vol. 294, pp. 1519-1525.
- 35. *The Canadian CT Head Rule for Patients with Minor Head Injury.* **Stiell, IG, Wells, GA et al.** May 5, 2001, The Lancet, Vol. 357, pp. 1391-1396.
- 36. *Comparison of the Canadian CT Head Rule and the New Orleans Criteria in Patients with Minor Head Injury.* **Stiell, IG, Clement, CM, Rowe, BH et al.** 12, September 28, 2005, JAMA, Vol. 294, pp. 1511-1518.
- 37. *Diagnostic Procedures in Mild Traumatic Brain Injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury.* **Borg, J, Holm, L, Cassidy, JD et al.** 2004, Journal of Rehabilitation Medicine, Vol. Supplement 43, pp. 61-75.
- 38. *Indications for computed tomography in patients with minor head injury.* **Haydel, MJ et al.** July 13, 2000, New England Journal of Medicine, Vol. 343, pp. 100-105.
- 39. *Recent Neuroimaging Techniques in Mild Traumatic Brain Injury.* **Belanger, HG, Vanderploeg, RD, Curtiss, G, Warder, DL.** 2007, The Journal of Neuropsychiatry and Clinical Neurosciences, Vol. 19, pp. 5-20.
- 40. *The use of CT scanning to triage patients requiring admission following minimal head injury.* **Livingston, David, Loder, Patricia et al.** 4, April 1991, The Journal of Trauma, Vol. 31, pp. 483-489.
- 41. *Emergency Department Discharge of Patients with a Negative Cranial Computed Tomography Scan after Minimal Head Injury.* **Livingston, DH, Lavery, RF et al.** 1, 2000, Annals of Surgery, Vol. 232, pp. 126-132.
- 42. *Immediate CT or admission for observation after mild head injury: Cost comparison in randomized control trial.* **Norland, Anders et al.** August 2006, British Medical Journal, Vol. doi:10.1136/bmj.38918.659120.4F, pp. 1-4.
- 43. *Traumatic Brain Injury in Anticoagulated Patients.* **Cohen, DB, Rinker, C, Wilberger, JE.** 2006, The Journal of Trauma, Injury, Infection and Critical Care, Vol. 60, pp. 553-557.
- 44. *Guidelines for evaluation and education of adult patients with mild traumatic brain injuries in an acute care hospital setting.* **Lawler, KA, Terregino, CA.** 6, 1996, Journal of Head Trauma Rehabilitation, Vol. 11, pp. 18-28.
- 45. *A proposal for an evidenced based emergency department discharge form for mild traumatic brain injury.* **Fung, M, Willer, B, Moreland, D, Leddy, JJ.** 9, August 2006 , Brain Injury, Vol. 20, pp. 889-894.

References

- 46. *The Management of Severe Traumatic Brain Injury.* **Chestnut, Robert M.** 3, August 1997, Emergency Medicine Clinics of North America, Vol. 15, pp. 581-604.
- 47. *The Impact of Pre-hospital Endotracheal Intubation on Outcome in Moderate to Severe Traumatic Brain Injury.* **Davis, DP, Peay, J, Sise, MJ et al.** 2005, The Journal of Trauma, Injury, Infection and Critical Care, Vol. 58, pp. 933-939.
- 48. *Out-of-Hospital Endotracheal Intubation and Outcome After Traumatic Brain Injury.* **Wang, HE, Peitzman, AB, Cassidy, LD et al.** 2004, Annals of Emergency Medicine, Vol. 44, pp. 439-450.
- 49. *Paramedic Rapid Sequence Intubation for Severe Traumatic Brain Injury: Perspectives From an Expert Panel.* **Davis, DP, Fakhry, SM, Wang, HE et al.** 2007, Prehospital Emergency Care, Vol. 11, pp. 1-8.
- 50. *The Effect of Paramedic Rapid Sequence Intubation on Outcome in Patients with Severe Traumatic Brain Injury.* **Davis, DP, Hoyt, DB, Ochs, M et al.** 2003, Vol. 54, pp. 444-453.
- 51. *A Follow-Up Analysis of Factors Associated with Head-Injury Mortality After Paramedic Rapid Sequence Intubation.* **Davis, DP, Stern, J et al.** 2005, The Journal of Trauma Injury, Infection and Critical Care, Vol. 59, pp. 484-488.
- 52. *Prehospital Hypertonic Saline Resuscitation of Patients With Hypotension and Severe Traumatic Brain Injury.* **Cooper, DJ, Myles, PS et al.** 11, 2004, JAMA, Vol. 291, pp. 1350-1357.
- 53. *Cranial Burr Hole Decompression in the Emergency Department.* **Springer, MFB, Baker, FJ.** 6, 1988, American Journal of Emergency Medicine, Vol. 6, pp. 640-646.
- 54. *Cranial Burr Holes and Emergency Craniotomy: Review of Indications and Technique.* **Donovan, DJ et al.** 1, January 2006, Military Medicine, Vol. 171, pp. 12-19.
- 55. *Surgical Management of acute subdural hematomas.* **Bullock, MR, Chesnut, R et al.** Supplement, 2006, Neurosurgery, Vol. 58, pp. S16-S24.
- 56. *Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury.* **Sahuquillo, J, Arikan, F.** Issue 1. Art. No.: CD003983. DOI: 10.1002/14651858.CD003983.pub2, 2006, Cochrane Database of Systematic Reviews.
- 57. *Treatment of Traumatic Brain Injury With Moderate Hypothermia.* **Marion, DW, Penrod, LE et al.** 8, 1997, New England Journal Of Medicine, Vol. 336, pp. 540-546.
- 58. *Prolonged Therapeutic Hypothermia After Traumatic Brain Injury In Adults.* **McIntyre, LA, Ferguson DA et al.** 22, June 11, 2003, JAMA, Vol. 289, pp. 2992-2999.
- 59. *Modest cooling therapies (35ºC to 37.5ºC) for traumatic brain injury.* **Saxena M, Andrews PJD, Cheng A.** Art. No.: CD006811. DOI: 10.1002/14651858.CD006811.pub2, 2008, Cochrane Database of Systematic Reviews, Vol. Issue 3.
- 60. *Hypothermia Treatment for Traumatic Brain Injury: A Systematic Review and Meta Analysis.* **Peterson K, Carson, S, Carney N.** January 2008, Journal of Neurotrauma, Vol. 25, pp. 62-71.
- 61. *Hypothermia Therapy after Traumatic Brain Injury in Children.* **Hutchinson JS, Ward RE et al.** 23, 2008, New England Journal of Medicine, Vol. 358, pp. 2447-2456.

References

- 61. *Hypothermia Therapy after Traumatic Brain Injury in Children.* **Hutchinson JS, Ward RE et al.** 23, 2008, New England Journal of Medicine, Vol. 358, pp. 2447-2456.
- 62. *Early Indicators of Prognosis in Severe Traumatic Brain Injury.* **Chestnut, Randall et al.** s.l. : Brain Trauma Foundation (www.braintrauma.org), 2000 - Accessed November 8, 2008, Guidelines for the Management of Severe Traumatic Brain Injury, pp. 157-207.
- 63. *Predicting outcome after traumatic brain injury: practical prognostic models based on large cohort of international patients.* **Collaborators, MRC Crash Trial.** 2008, British Medical Journal, Vol. 336, pp. 425-429.
- 64. *Second Impact Syndrome.* **McCrory, PR, Berkovic, SF.** 3, March 1998, Neurology, Vol. 50, pp. 678-683.
- 65. *Recognition and Characteristics of Concussions in the Emergency Department Population.* **Delaney, JS, Abuzeyad, F et al.** 2, 2005, The Journal of Emergency Medicine, Vol. 29, pp. 189-197.
- 66. *A Population-Based Study of Seizures after Traumatic Brain Injury.* **Annegers, JF et al.** 1998, New England Journal Of Medicine, Vol. 338, pp. 20-24.
- 67. *Medical Aspects of the Persistant Vegetative State.* **PVS, The Multi-Society Task Force on.** 21, May 26, 1994, New England Journal Of Medicine, Vol. 330, pp. 1499-1508.