Neurosurgical Aspects of Closed Head Injury

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Definition of Closed Head Injury

- Insult rendered to the brain following a traumatic event that does not create an opening in the skull aside from a linear skull fracture
- Typical events include:
  - Motor vehicle accident
  - Fall
  - Assault with a blunt object
  - Domestic abuse including “shaken children”
  - Sports injuries

Etiology of CHI

- Acceleration-Deceleration is critical to the development of most CHI (no intracranial seatbelts)
- The skull's interior contour is critical to the development of most CHI (no intracranial air bags)
- The brain's gelatinous consistency is critical to the development of most CHI
- The axonal structure to neurons and the neurologic system's architecture of white matter pathways and tracts is critical to the development of most CHI

Epidemiology of Traumatic Brain Injury

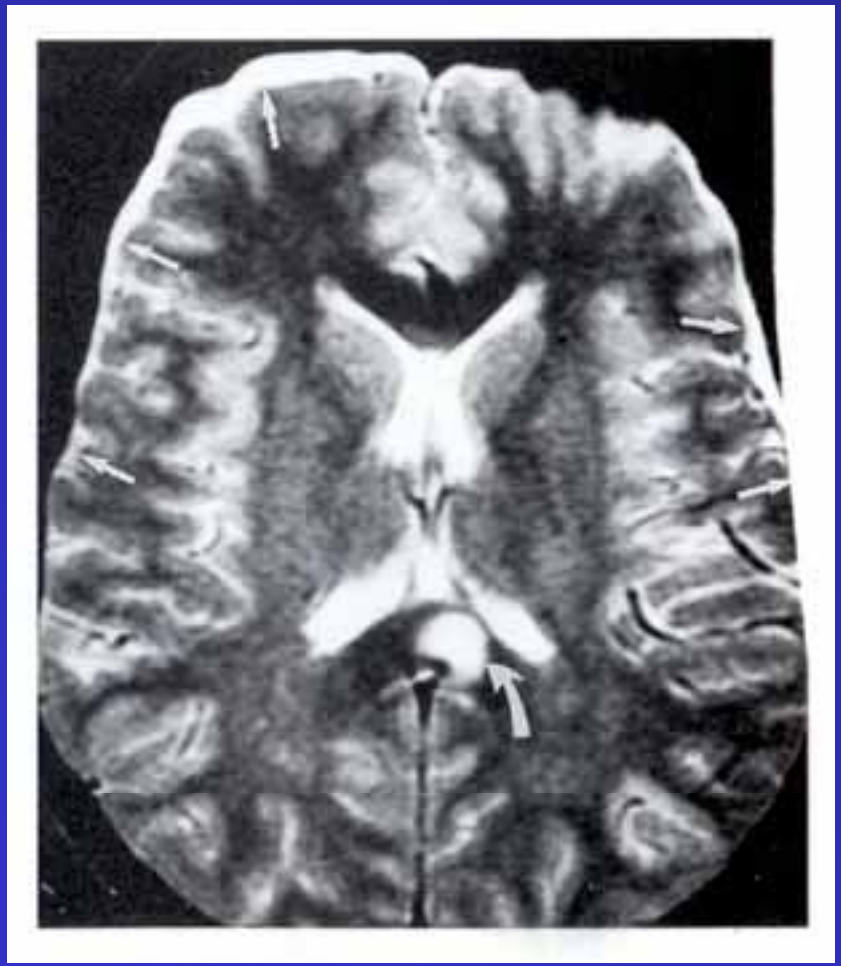
- Incidence between 100-400/100,000/year
- Male:Female 2:1
- Peak incidence ages 15-35
- In England, most common cause of death in children ages 1-15
- Over 50% of trauma related deaths are associated with TBI

Pathophysiology

- Types of CHI
  - Diffuse Axonal Injury (DAI)
    - DAI is caused by angular accelerations of the head and subsequent rotation and torque of the cerebral hemispheres, brainstem, and their deep fiber tracts and nuclei
    - Small hemorrhages are seen in the corpus callosum, septum pellucidum, deep gray matter, midbrain, pons
    - Axons ultimately degenerate

Diffuse Axonal Shearing Injury

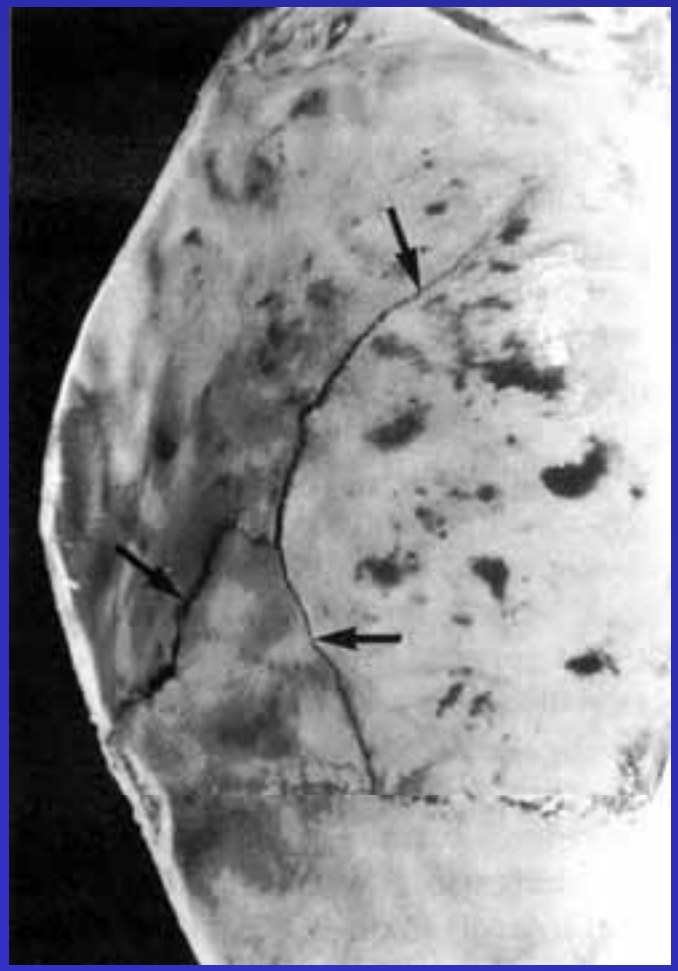
DAI



Pathophysiology

- Types of CHI
  - Focal contusions (bruises) and lacerations (cuts) with subsequent edema
  - *Coup-contra coup* injuries involving frontal lobe and temporal lobe tips, occipital poles
  - Intracranial hematomas
    - Subdural
    - Epidural
    - Intraparenchymal

Linear Skull Fracture

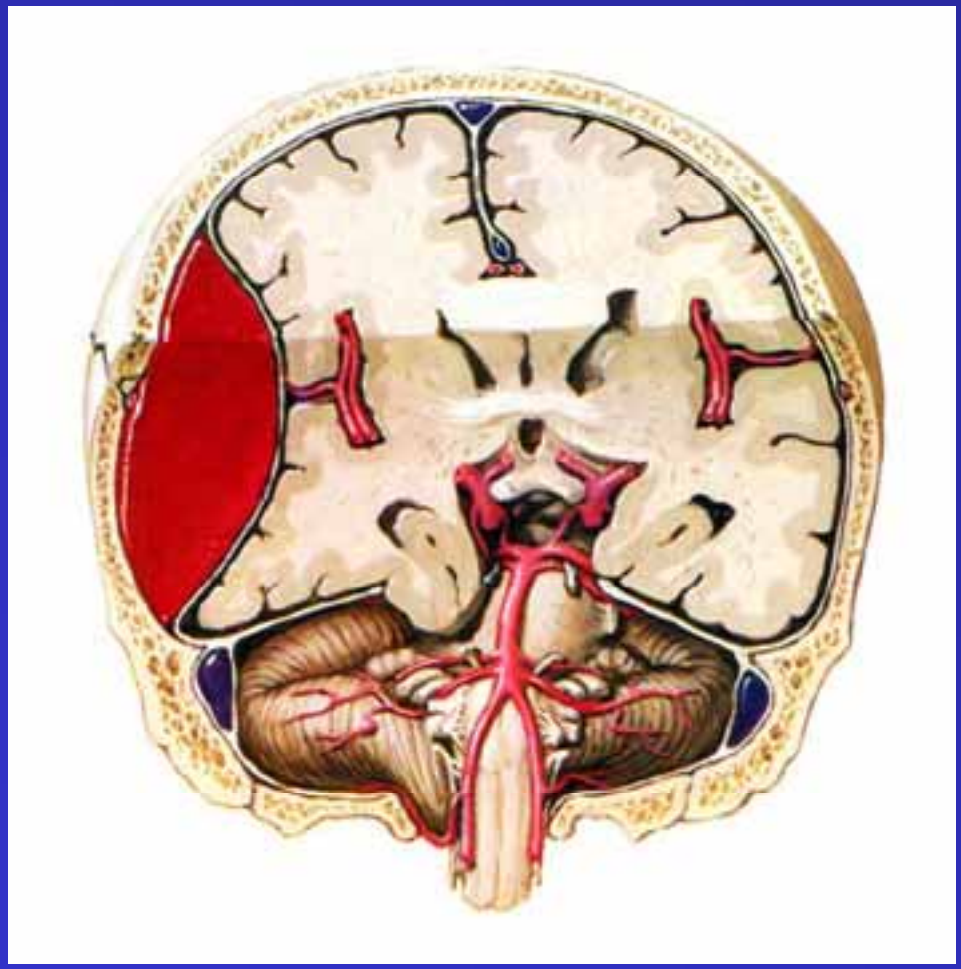


Linear Skull Fracture

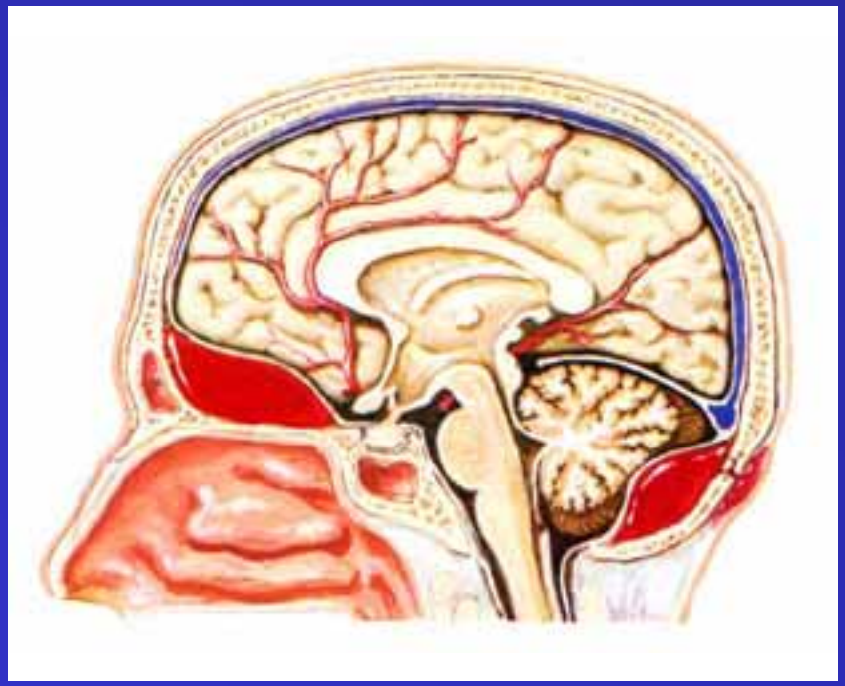
Raccoon's Eyes

Battle's Sign

Epidural Hematoma



Epidural Hematoma



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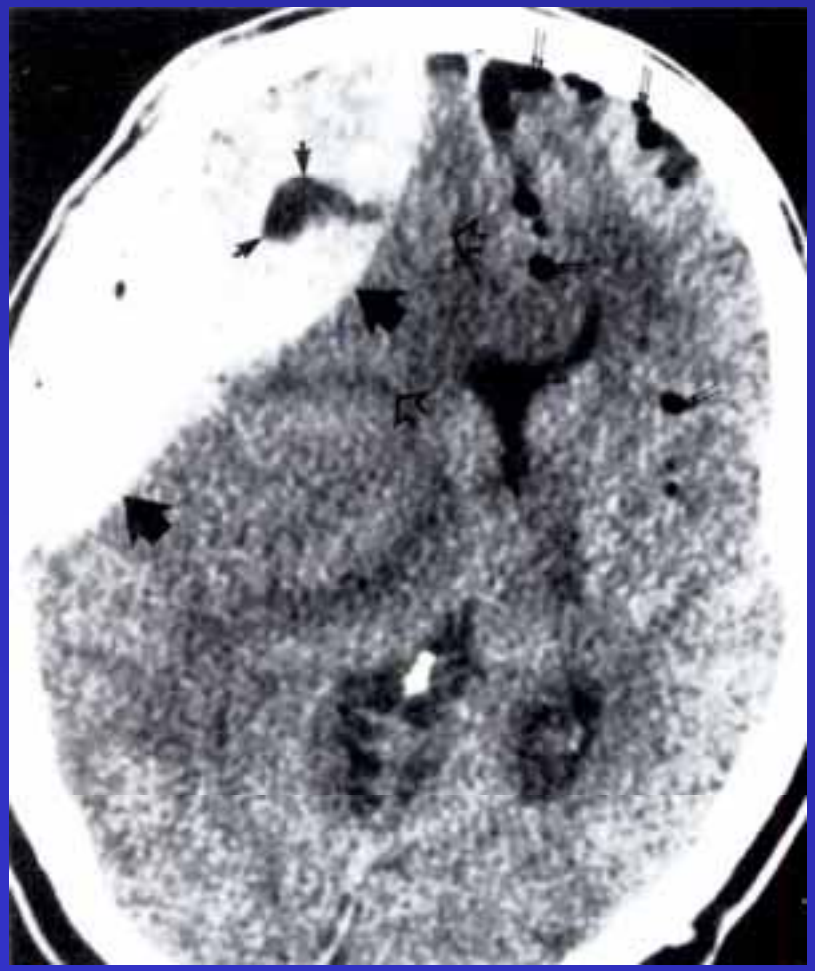
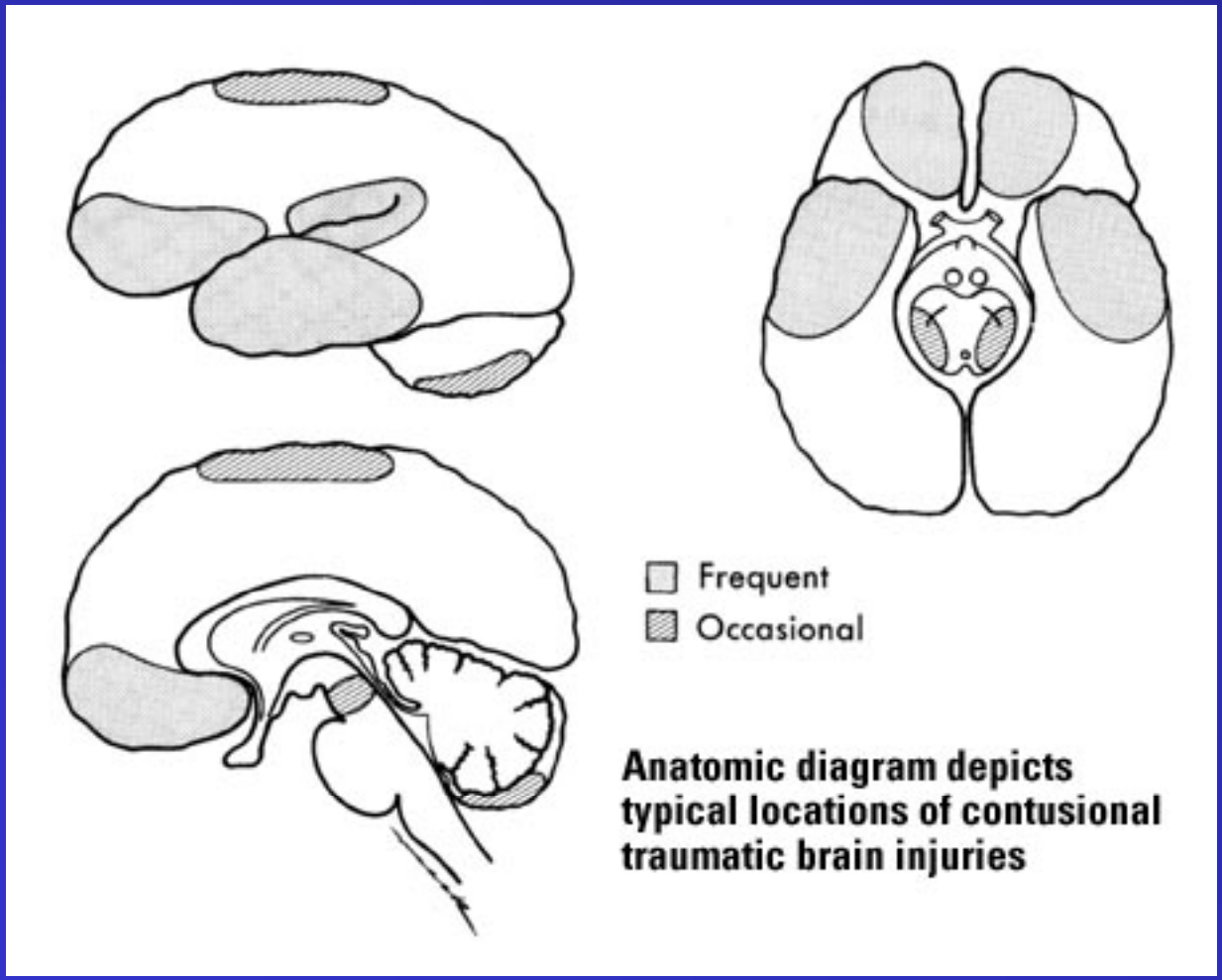
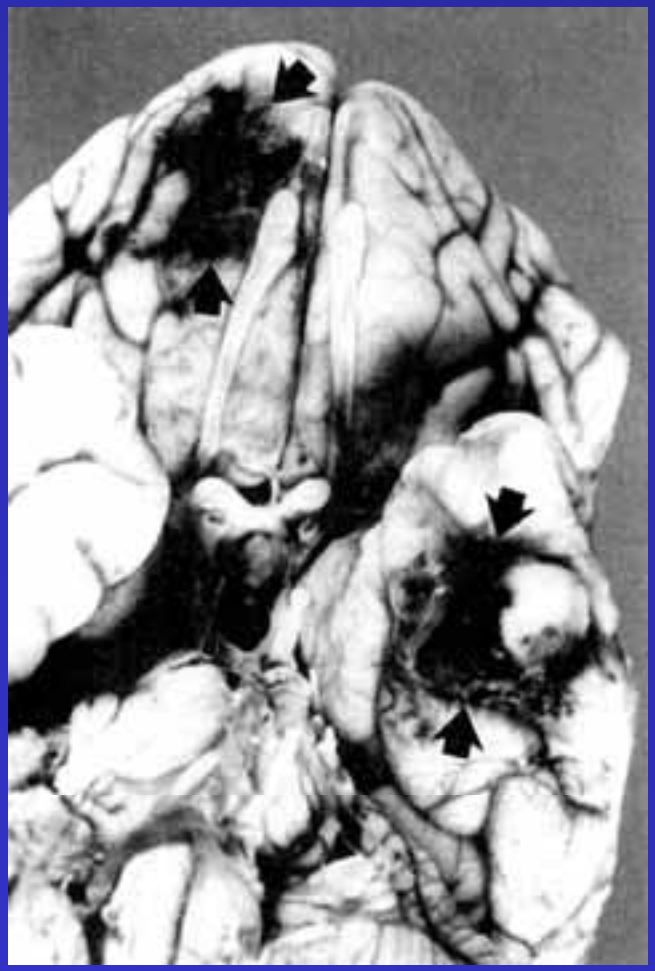


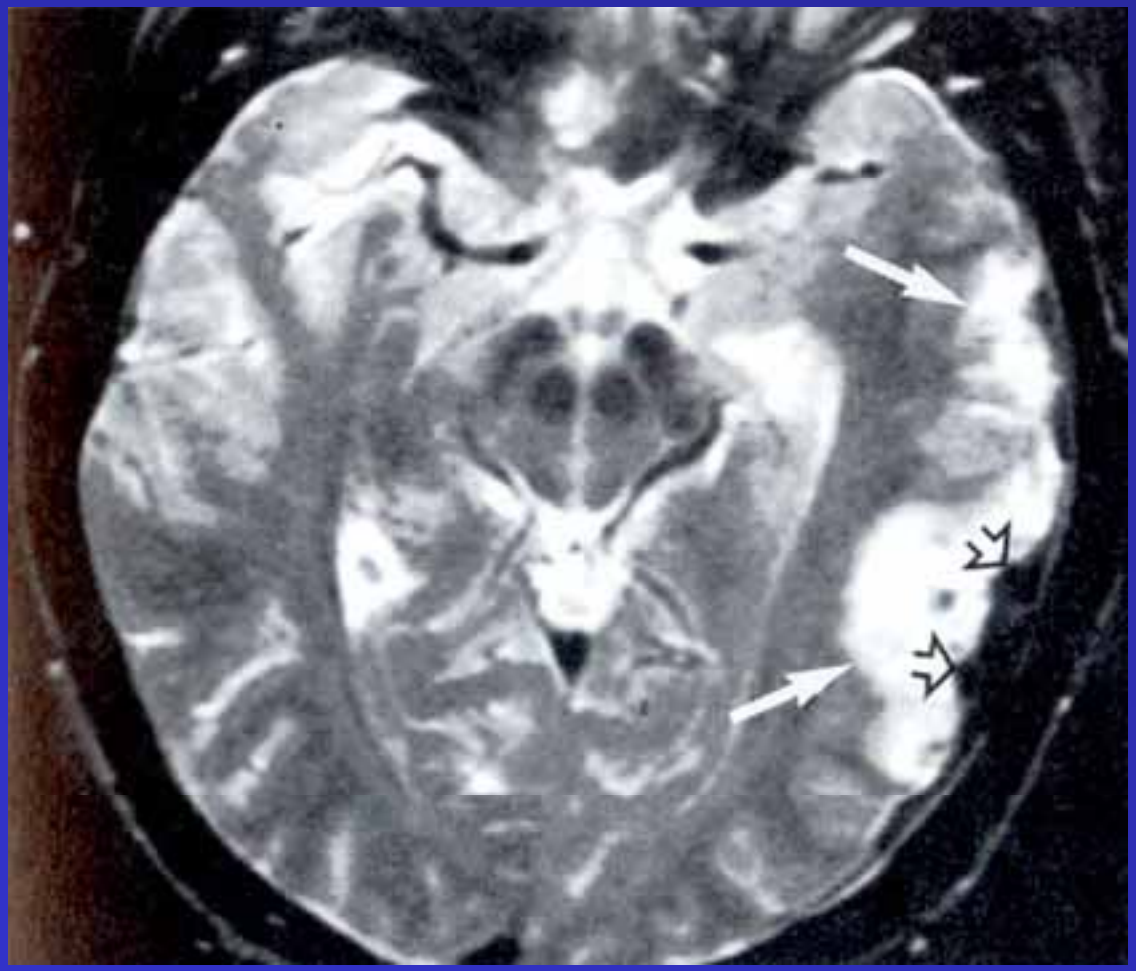


Anatomic diagram depicts typical locations of contusional traumatic brain injuries

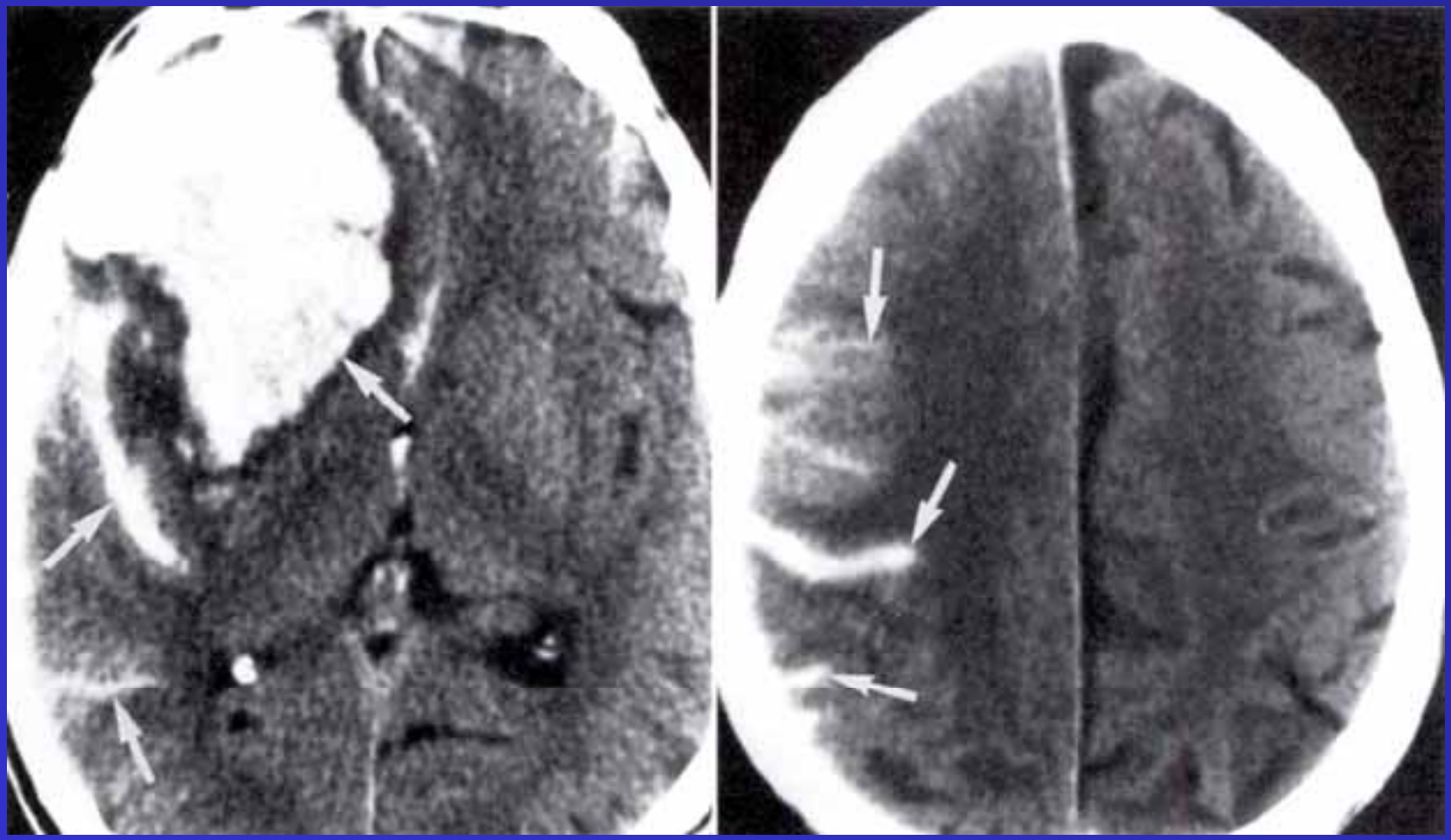
Contusions



Contusions on MRI



Intraparenchymal Hematoma



Pathophysiology

- Primary brain injury at the time of insult leads to a series of other secondary inflammatory biochemical changes in the brain tissue
- Inflammation period lasts 2-3 weeks
- During this inflammatory period the brain is more susceptible to hypotension, hypoxia, pyrexia

Factors in the Post Injury Period

- Excitotoxins
  - Excitatory amino acids (aspartate and glutamate) released in response to reduction in cerebral blood flow
  - Lead to cell death by activation of N-methyl D-aspartate (NMDA) receptor and the associated Ca ion channel
  - Ca influx into the cell leads to cell membrane destruction and cell death
  - Research into NMDA receptor blockers has not yet proved protective

Factors in the Post Injury Period

- Inflammation
  - After injury intracranial cells increase their production of cytokines which activate inflammatory cascades
  - After injury adhesion molecules increase leading to an influx of leukocytes which can in turn damage brain tissue
- Free Radicals
  - Free radical production increases after injury leading to cell membrane damage
- Hyperglycemia
  - Leads to lactic acid production

Secondary Brain Injury

- Secondary brain injury refers to factors that present subsequent to the initial insult and deleteriously affect brain function thus further compromising activity and recovery
- Examples are:
  - Pyrexia
  - Secondary brain swelling and herniation
  - Extra-axial masses and increased intracranial pressure or herniation
  - Intracranial hematomas

Secondary Brain Injury

- Each of these secondary injuries further affects brain function through:
  - Hypoxia
  - Hypoperfusion
  - Reduction in cerebral perfusion pressure
  - Acidosis
- Resulting mismatches between cell metabolism and energy supply along with physical distortion of neural architecture leads to cell and tissue death

What is the clinicians role?

- We cannot prevent the initial injury
- We strive to recognize and treat secondary injuries as well as try to prevent their development
- These goals define a range of community standards for post injury neurosurgical management

Community Standards

- Physicians deal with two communities
  - Geographic community in which we practice (hospital environment and its capabilities)
  - Neurosurgical community
- Often the ideals of one community do not or cannot meet the expectations of the other
- The reasons for these mismatches need to be taken into consideration when reviewing cases or determining liability and when obtaining physician opinions.
- Not everyone practices in an Ivory Tower

Patient Assessment

- Assess in field and try to get patient to a neurosurgical or trauma center ASAP
- Physical Examination
  - Associated trauma- skull, ears, spine, body, heart, lungs
  - In children- retinal examination
- Neurological Examination
  - Glasgow Coma Score
  - Pupillary examination
- History- mode of injury
- Laboratory tests- blood count, coagulation profile

Indications for Neurosurgical Consultation

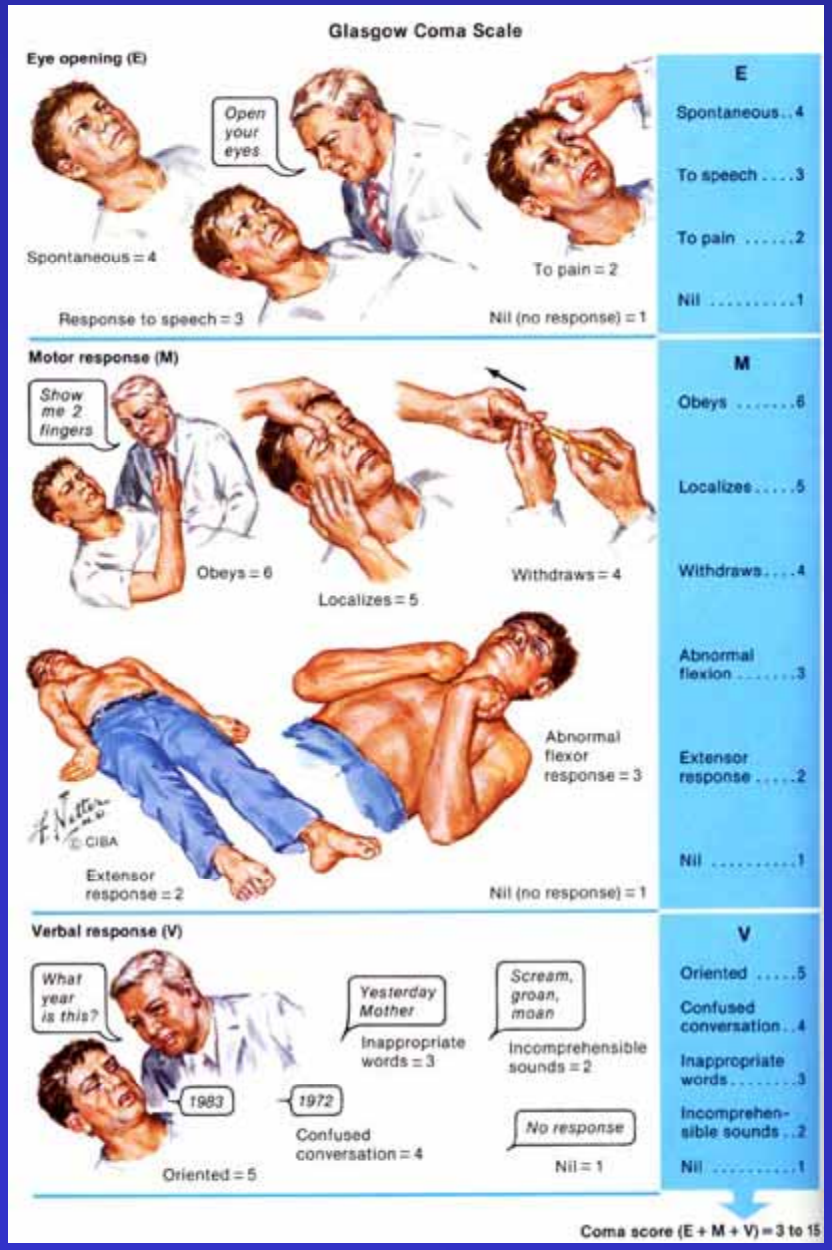
- CT scan demonstrates an intracranial mass lesion
- Intracranial lesion is suspected yet a head CT cannot be obtained because of other life threatening injuries (pneumoventriculography possible)
- GCS < 9; External ventricular drain needed
- Confusion for more than 4 hours
- Deterioration in neurological examination
- Focal neurological findings
- Compound depressed skull fracture
- CSF leak

Patient Assessment

- Glasgow Coma Score
  - **Eye Opening**
    - Spontaneous 4
    - To Speech 3
    - To Pain 2
    - None 1
  - **Verbal Response**
    - Oriented 5
    - Confused 4
    - Inappropriate 3
    - Incomprehensible (sounds) 2
    - None 1
  - **Best Motor**
    - Obeys commands 6
    - Localizes to pain 5
    - Withdraws from pain 4
    - Decorticate 3
    - Decerebrate 2
    - None 1

Total Score 3 - 15

Glasgow Coma Scoring



Patient Assessment

- Why is GCS important?
  - Allows healthcare personnel to quickly discuss patients and relate clinical status
  - Allows for accurate serial assessment of patient progress
  - May predict the likelihood of other injuries
  - May help predict clinical outcomes

Patient Assessment

- Risk of an operable hematoma in CHI patient

GCS	Risk
– 15	1 in 3615
– 14	1 in 51
– 3-8	1 in 7

Patient Assessment

- GCS can help predict patient outcomes
- GCS can help predict likelihood of elevated intracranial pressure (ICP)
  - GCS < 9
    - ICP > 10 in 82%
    - ICP > 20 in 44%
    - ICP > 40 in 10%

Patient Assessment

- Imaging Evaluation
  - CT scanning
    - Indicated for any patient who suffers an injury that leads to a GCS < 15
    - Unclear if it is indicated for normal GCS and head injury such as a linear skull fracture
    - CT looks for blood clots (intra and extraaxial), brain swelling, air leaks, bony fractures
    - Findings dictate need for surgical therapy and types of medical therapy

Patient Management

- CT/Xenon Blood flow studies
  - Look at cerebral blood flow which can help determine subsequent management with ventilator and medications
  - Not available at many centers

Patient Assessment

- MRI scanning
  - Usually useful in the subacute or chronic period when looking for more occult brain injuries especially in deeper areas or in the brainstem
  - Not a typical study done in the acute period
- Angiography
  - Done when there is suspicion of a vascular injury

Patient Assessment

- CT findings may help predict the presence of elevated ICP
- Elevated ICP leads to further brain injury by mechanically disrupting brain tissue and possibly reducing cerebral blood flow

Patient Assessment

- High/low density lesion on CT — 53 – 63% chance increased ICP
- Normal CT — 13% incidence of elevated ICP
  - Factors with a normal CT that increase the incidence of increased ICP
    - Age > 40
    - Systolic BP < 90 mm Hg
    - Abnormal motor posturing
  - 2 or more features — 60% incidence increased ICP
  - 1 or more features — 4% incidence increased ICP

Patient Assessment

- Absent or compressed basal cisterns on first CT scan with GCS < 9
  - Absent 77% mortality
  - Compressed 39% mortality
  - Normal 22% mortality

  - Absent 85% poor outcome
  - Compressed 64% poor outcome
  - Normal 44% poor outcome

  - No survivors when absent cisterns associated with 15 mm shift
  - 74% with absent cisterns had ICP > 30

Basal Cisterns

CT Imaging Diagnostic Categories in CHI and Mortality

• Diffuse Injury I	No visible abnormality	9.6% mort.
• Diffuse Injury II	Cisterns present with 0-5 mm shift; no high density lesion greater than 25cc	13.5% mort.
• Diffuse Injury III	Cisterns compressed or absent; shift 0-5mm; no high density lesion > 25cc (swelling)	34% mort.
• Diffuse Injury IV	Shift > 5 mm; no lesion > 25 cc (shift)	56.2% mort.
• Evac. Mass Lesion	Any lesion surgically evacuated	38.8% mort.
• Nonevac Mass Les	Mass > 25cc not evacuated	52.8% mort.
• Brainstem injury		66.7% mort.

Patient Management

- Remove mass lesion if:
  - Patient has a survivable exam
  - Patient is age appropriate
  - It is associated with brain shift (usually > 1 cm)
  - GCS is abnormal
  - It is in a location that portends imminent danger (ie: low middle fossa)
  - Clot size is considered significant (ie: 60cc rule)

Patient Management

- Acute Subdural Hematoma
  - Very common extra-axial mass lesion in CHI
    - Mortality 42 – 90%
  - Usually a marker for more diffuse underlying brain injury from shear forces
    - GCS 3-5 — Mortality 76-84%, Functional recovery rate 14%
    - GCS 6-8 — Mortality rate 36-48%, Functional recovery rate 25-40%
    - GCS 12-15 — Mortality rate 0%, Functional recovery rate 92%

Patient Management

- Acute Subdural Hematoma
  - Conscious at surgery Mortality rate 6%
  - Unconscious at surgery Mortality rate 77%
  - Pupils abnormal Mortality rate 75%
  - Decerebrate Mortality rate 77%
  - Decerebrate + Unreactive pupils Mortality rate 95%
  - Age >50, time to evacuation > 4 hours, elevated post-op ICP = Poor prognosticators

Patient Management

- Elevated ICP (Usually treat when ICP is greater than 20)
  - ICP monitor placed
  - CSF drained
  - Hyperventilation to reduce CBF ????- Controversial issue
  - Mannitol/Lasix to reduce extracellular fluid
  - Barbiturates to reduce CBF
  - Reduce body temperature (medications, devices)
  - Surgical removal of brain tissue and/or skull

Patient Management

- Try to reduce incidence of secondary insults
  - Hypoxia
  - Infection
  - Hyperglycemia
  - Fever
  - Deep venous thrombosis
  - Decubitus ulcers
  - Brain swelling
  - Seizures

ICU Goals

- Keep serum sodium >135-140 and < 160. A low Na can lead to increased brain swelling
- Avoid hyperglycemia to avoid lactic acidosis
- Nutritional support within 72 hours of CHI. Metabolic expenditure can be 120-250% of a normal patient. Keep some nutrition in gut.
- Mild head elevation ???
- Physical therapy
- DVT prophylaxis

Rehabilitation

- It takes most adults at least one year to recovery maximally from a severe CHI
- Rehabilitation centers are a key part of this recovery phase.

Outcomes Evaluation

- Glasgow Outcome Score
  - 1 Dead
  - 2 Persistent Vegetative State
  - 3 Severe Disability
  - 4 Moderate Disability
  - 5 Good Recovery

Outcomes

- Other outcomes scales exist yet it is unclear which ones are best.