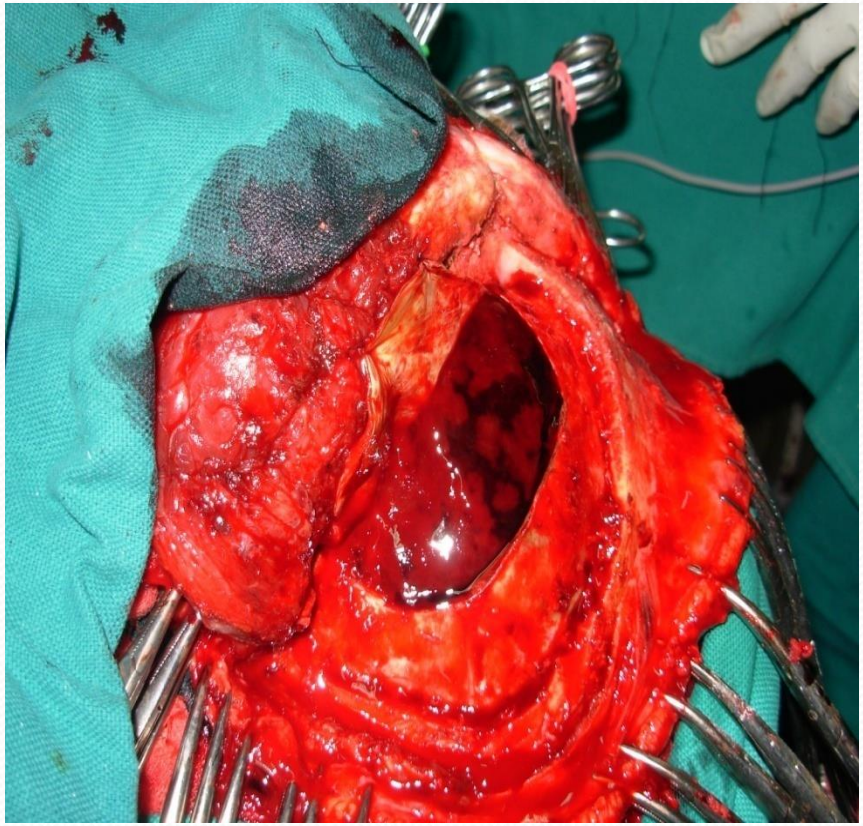
Seth GSMC lecture series : Neurosurgery

T B I and HEAD TRAUMA MANGEMENT



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Introduction - Prof. Chagla

Finding solutions to medical inadequacies in rural areas | Aadil Chagla | TEDxXIE

TEDx Talks 2.4K views • 2 years ago

A neurosurgery professor, Dr. Aadil Chagla, shares his ideas setting up trauma centers and strengthening the infrastructure on ...

Over the Rainbow - Concert (A) for Shaukat C Chagla Memorial Trust directed by Prof. Aadil S Chagla

aadilchagla • 1.7K views • 3 years ago

Shaukat C. Chagla Memorial Trust presents " OVER THE RAINBOW" Concert held at Sophia Bhabha Auditorium on the 8th Aug.

Disclosures

- I have not financial interests what so ever
- Some slides or matter may have been borrowed from text or other presentations and are used solely for teaching purposes with no financial interest
- This dedicated lecture was for the final year medical students of the Seth G S Medical College Mumbai

Introduction

- Trauma related deaths will be third leading cause of death for all age groups.
- For India road traffic accidents would be a major killer accounting for 5, 46,000 deaths 1, 53, 14,000 disability adjusted life years with an estimated economic loss of around 2% of GDP.
- Most common cause of trauma is road traffic accident.

Traumatic Brain Injury : How big of a problem is it?

	AIDS/HIV	Lung Cancer	TBI
Incidence per year	38,000	1.3 million	1.5 million
Die / Year	15, 000	1.1 million	52, 000
Disabled (cumulative)	449,000	351,000	5.3 million

CDC 2005

Road Traffic Accidents (RTA)

- Lives of 1.2 million men, women and children around the world are lost each year, 20-50 million are injured or disabled per year, 12,000 disabled throughout life.
- India has just 1% of total vehicle in the world but India's share of global car accidents is nearly 10%. [Daily Telegraph Sep. 06]

R T A in INDIA

- The accident rate of 35 per 1000 vehicles in India is the highest in the world. The mortality has increased from 30 per lakh (1970) to 50 per lakh (2002)
- On India's roads, 95,000 persons died in 2005. On an average one person dies every 6 minutes; 10 are injured in the same time frame [BBC; Sep 2005]
- 70% the are due to head and spinal injury

A point to note !

- Parents of teenagers worries about lots of things – drugs, sex, poor choice of friends.

- But the activity that causes the most harm to the teenagers is none of the above.

DID YOU KNOW THAT….

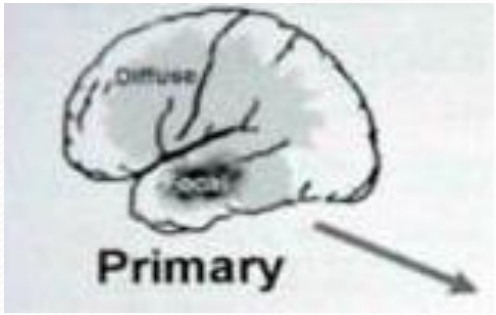
- Motor vehicle crashes are the leading cause of death for 16 to 20 years olds.

Primary injury

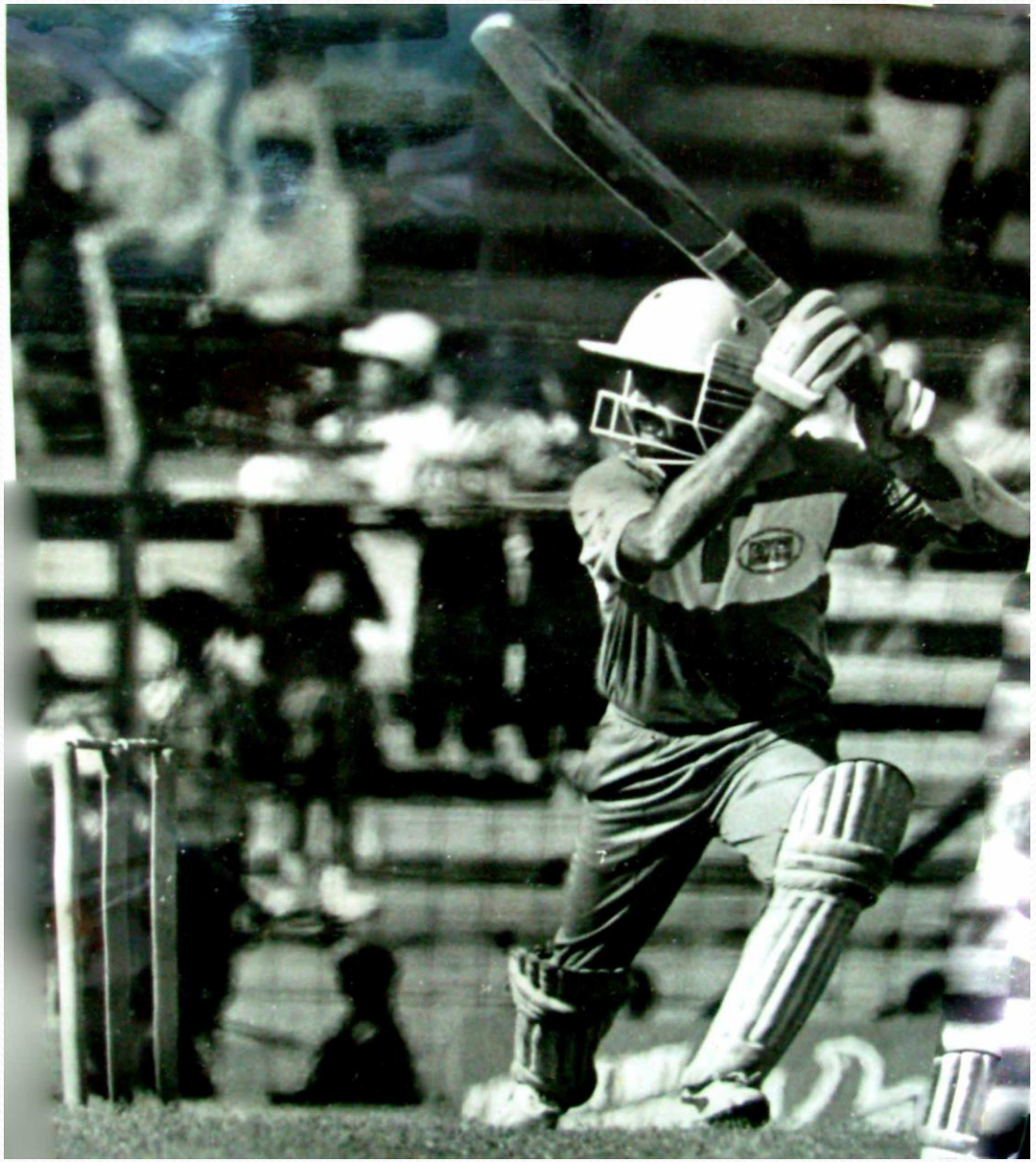
Result of immediate mechanical disruption of brain tissue.
Primary injury can be prevented or reduced (education, air bags) but not eliminated

- Diffuse Axonal Injury
- Contusion
- Intraparenchymal hematoma
- Subarachnoid hemorrhage
- Subdural hematomata
  - Acute
  - Chronic

- Epidural hematoma
- Skull fractures
- Secondary brain swelling
- Vascular dissections and other vascular injuries
- Associated cervical spine injury



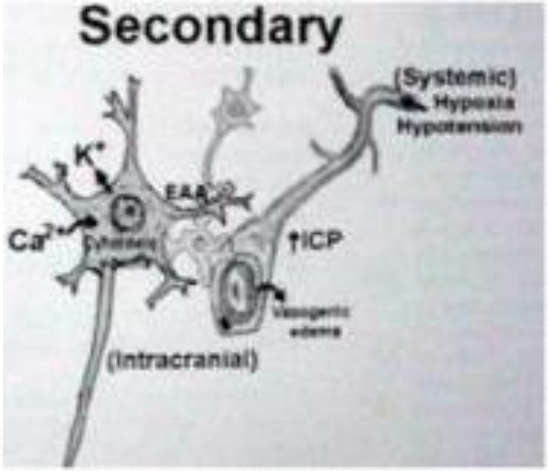
Use of Helmet in sports to prevent head injury



Secondary Brain Injury

Neuronal damage from systemic physiologic responses to primary injury.

- Ischemia
- Hours to days after the primary brain injury.
- Progressive, not singular
- Hypoxia and hypotension are the two major causes of secondary CNS injury following head trauma
  - Preventing hypoxia and hypotension could have the greatest effect of any currently available treatment for head trauma
    - SaO_2 > 90%, PaO_2 > 60mmHg
    - SBP > 90mmHg
- High Intracranial Pressuresdue to diffuse swelling
  - Edema and Herniation causes tissue compression and vascular compromise
- Hyperthermia
- Brain Edema
- Hemorrhage



Mechanisms of injury

Mechanistic Types and Causes of Head Injuries

- Contact Injuries
  - Local Contact Effects
  - Remote Contact Effects

Contact Injuries

Skull Deformation Injuries
Local Skull Bending
Skull fracture (linear, depressed)
Epidural hematoma
Coup contusion (bruise)
Laceration

Skull Volume Changes
Vault, basilar fracture
Contrecoup contusion

Shock wave Propagation
Intracerebral hemorrhage

Head Motion Injuries

Skull Brain Relative Motion

Subdural hematoma

Contrecoup Contusion

Intermediate Coup contusion

Brain Deformation

Concussion syndromes

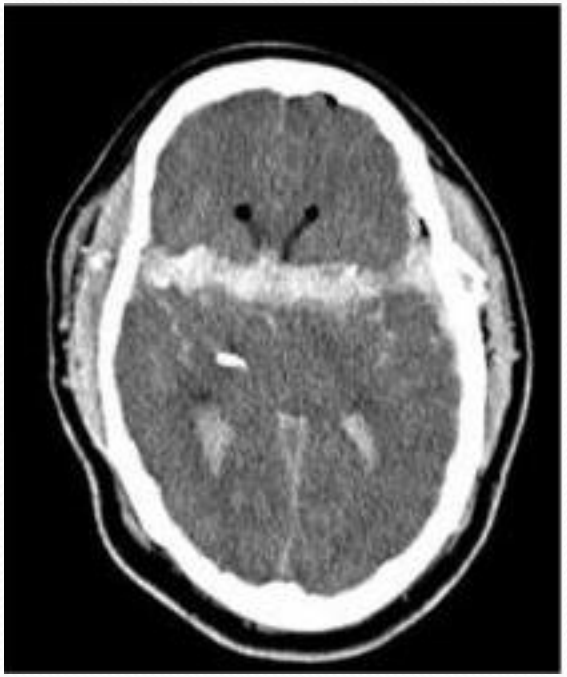
Diffuse Axonal injury

Intracerebral hemorrhage

Tissue tear hemorrhage

Gunshot Wounds

- Primary injury
  - Injury to tissue by shock waves and cavitation effect
  - Comminuted fracture of bone, secondary projectiles into brain
  - Cerebral injuries from missile
    - Direct injury to brain tissue in path of bullet
- Secondary Injury
  - Caused by rapid increase in ICP
- Late Complications
  - Cerebral abscess ~ 10%
  - Traumatic aneurysm ~18%
    - Consider angiogram
  - Seizures
  - Migration of fragments



Primary Head Injury

Clinical Classification of Head Injuries

- Skull Fractures
- Focal Brain Injuries
- Diffuse Brain Injuries

Classification (Contd.)

- **Skull Fractures**

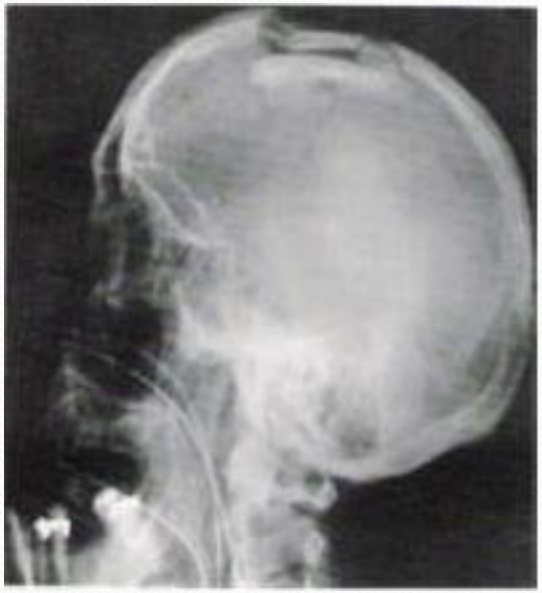
Vault

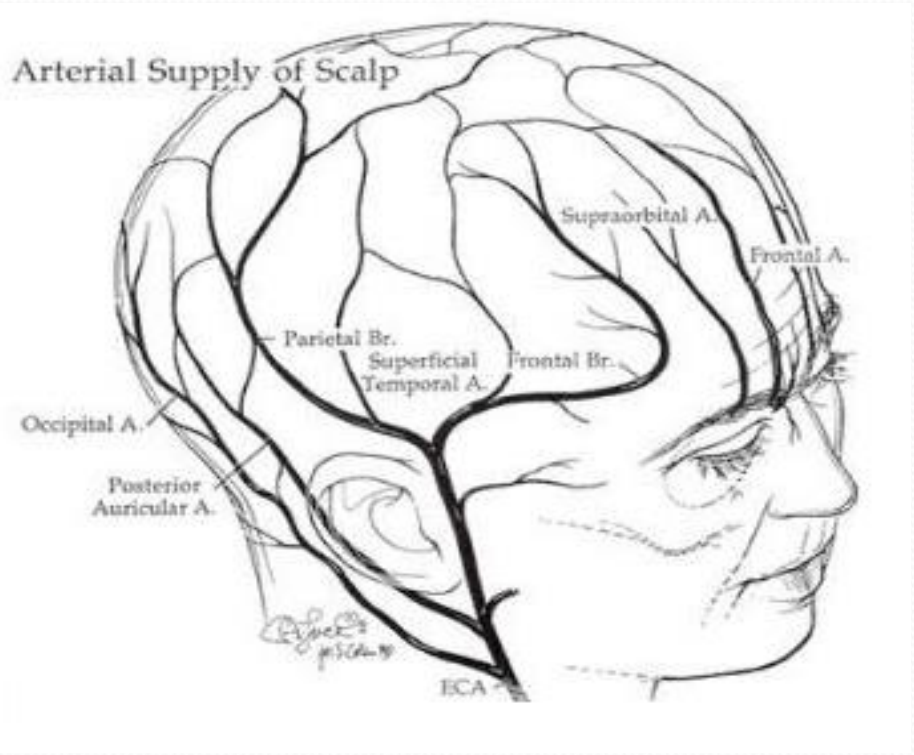
- Linear
- Depressed

Basilar

Compound Depressed Skull Fractures

- Laceration communicates with cranial cavity
- Bleeding (Superficial Temporal, Occipital Artery)
- Depressed skull fractures
- Violation of venous sinus





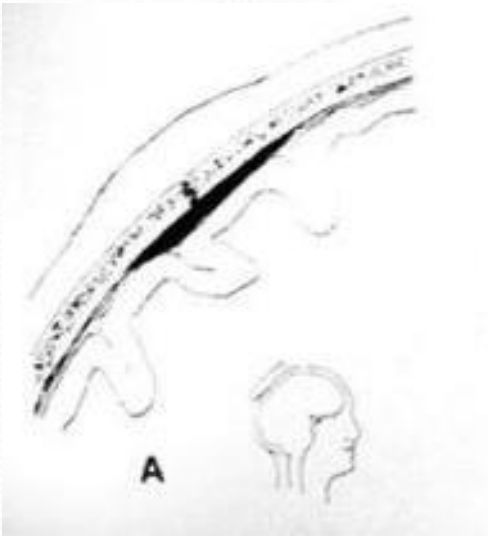
Depressed Skull Fractures

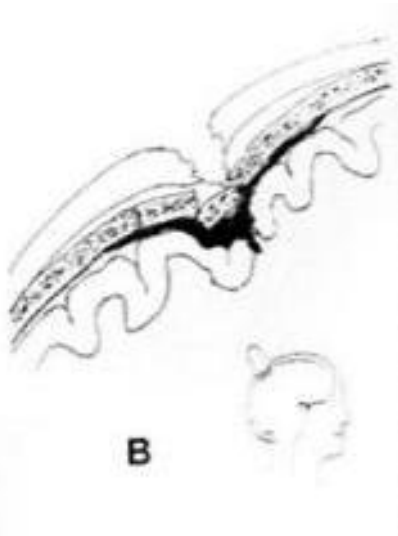
Criteria for surgical elevation

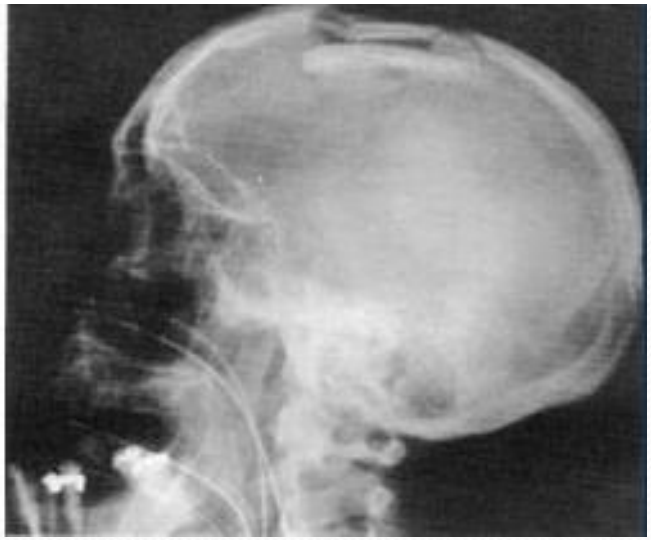
- >8-10 mm depression (greater than the thickness of the skull)
- Depression of bone compressing underlying brain
- CSF leak
- Open depressed skull fracture

– No evidence to suggest that elevation of skull fracture reduces posttraumatic seizure

– Entertain surgically debrided if fracture is through posterior wall of frontal sinus



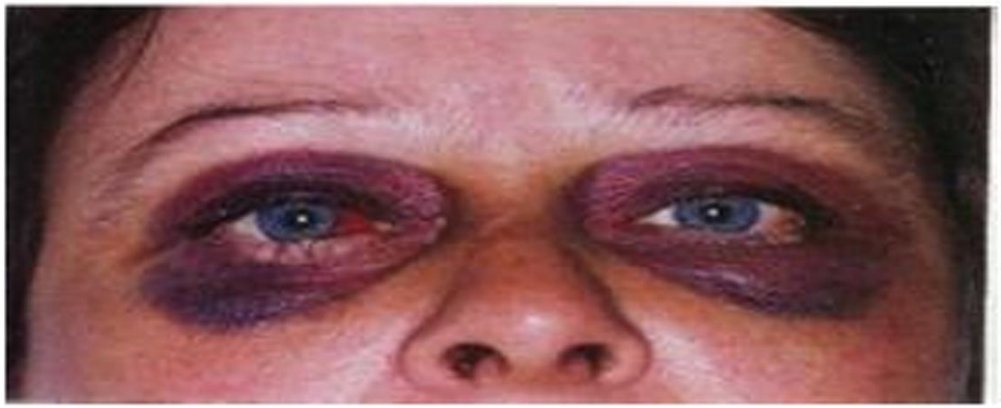


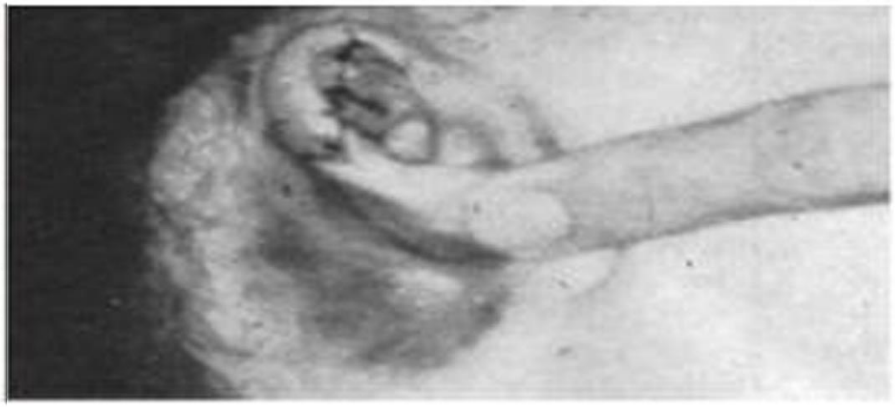


Basal Skull Fractures

Gross inspection

- Periorbital ecchymosis- Raccoon/Panda Bear eye
  - frontal bone fracture
  - Rhinorrhea- fluid leaking from nose, high incidence of meningitis
- Periauricular ecchymosis- Battle sign
  - suspect temporal bone fracture
  - Otorrhea- fluid leaking from ear, risk of meningitis, is less than for rhinorrhea





Classification (Contd.)

- **Focal Brain Injuries**

Contusion

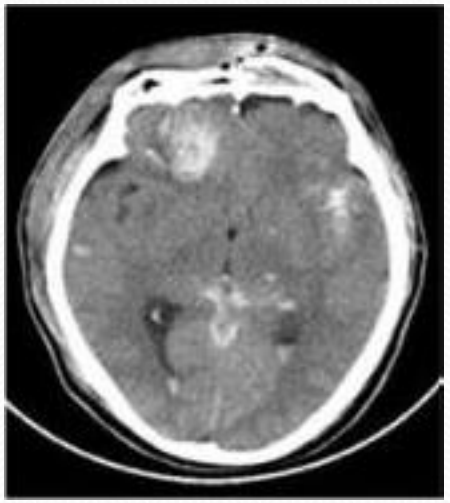
- Coup
- Contrecoup
- Intermediate

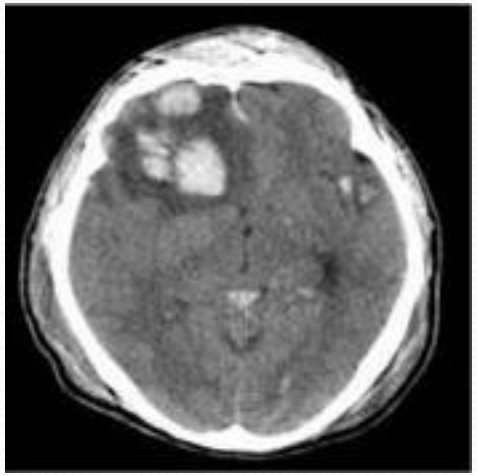
Hemorrhage / Hematoma

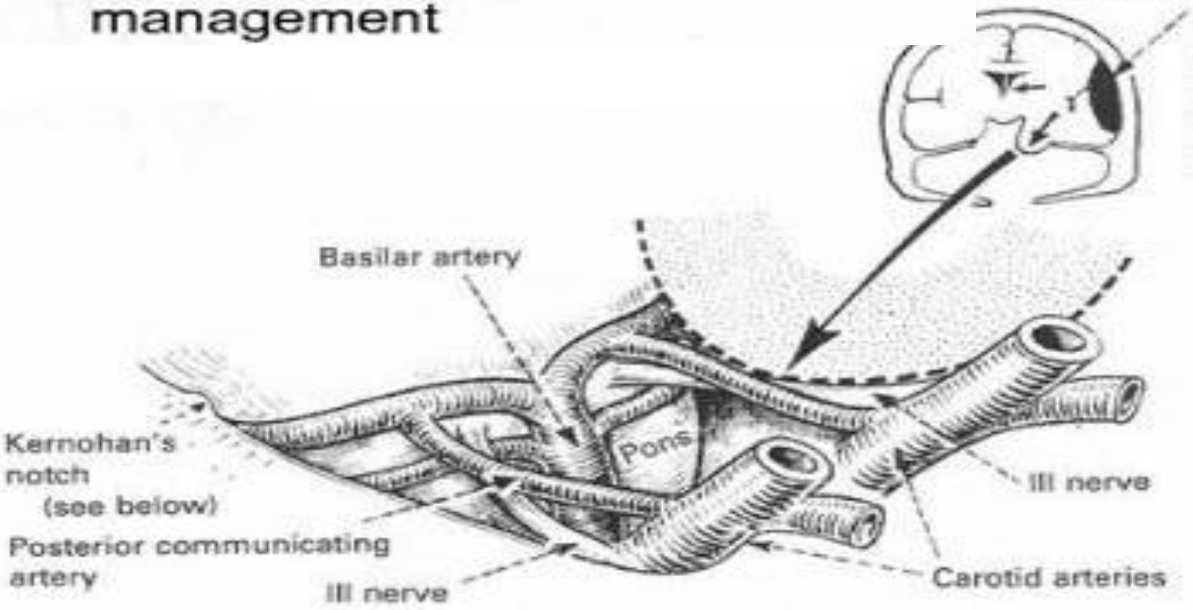
- Epidural
- Subdural
- Intracerebral
- Petechial

Cerebral Contusions

- ¼ enlarge over time (1st four hours)
  - Need serial CT scans
  - ICP monitor
    - Less useful in temporal lesions, may herniate without increase in ICP's only clinical signs of uncal herniation
  - Treatment
    - Surgical if mass effect on CT
    - ICP>20 with maximized medical management

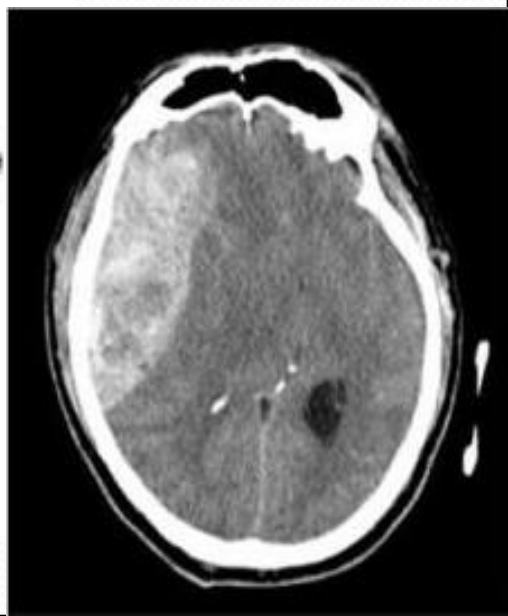






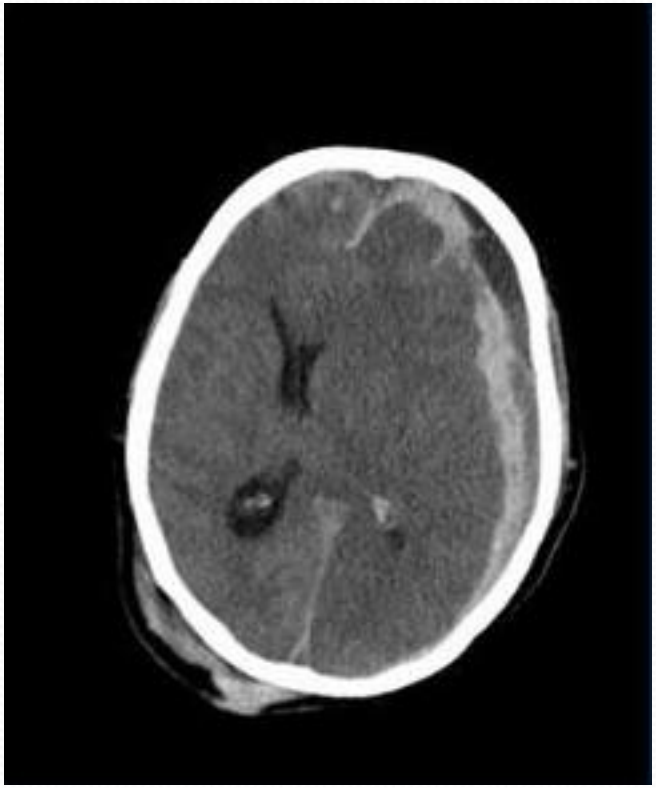
Epidural Hematoma

- LOC→ Lucid Interval → Deterioration (obtundation, hemiparesis, dilation of pupil)
- Most M&M is secondary to delayed diagnosis
- 1 % of all head trauma admissions
- M:F = 4:1
- 85 % arterial with fracture
  - —Middle Meningeal Artery
- 15 % venous
  - — Middle Meningeal Vein
  - —Sinus
  - —Oozing from fracture (Diploe)
- Biconvex shape (Lenticular shape)
  - —at convexity (frontal, occipital, posterior fossa)
- Usually younger than 50
- Usually associated with skull fracture
- If untreated will result in death
- Mortality Rates



Subdural Hematoma

- From parenchymal laceration
  - associated with severe underlying injury to brain
- Tearing of bridging veins
  - Secondary to cerebral acceleration-deceleration forces
  - Brain damage may be less severe
- Damage due to impact
  - Higher magnitude of impact than that of EDH
  - more lethal
  - More brain injury and edema
- Crescent shaped Usually
  - Tearing of surface or bridging veins
- Patients older than EDH
- Treatment
  - Craniotomy if > 1 cm thick at its biggest point
  - Smaller subdual's in GCS 14-15 may be observed with serial CT scans
- Mortality
  - Higher Mortality rate
- High mortality rate
  - Much greater > 4 hrs
  - Age >60
  - Neurological condition on admission
  - Low post resuscitation Glasgow
  - Highest if on anticoagulants
  - Higher peak ICP after surgery



Classification (Contd.)

- **Diffuse Brain Injuries**

Concussion

- Mild
- Classic

Diffuse Axonal Injury

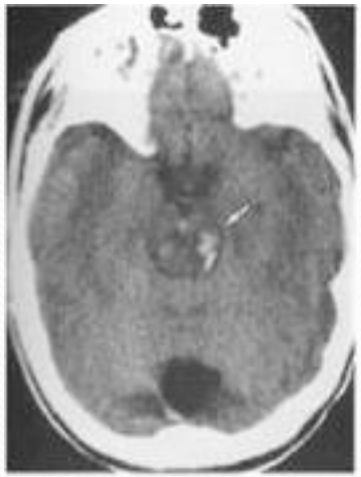
Other

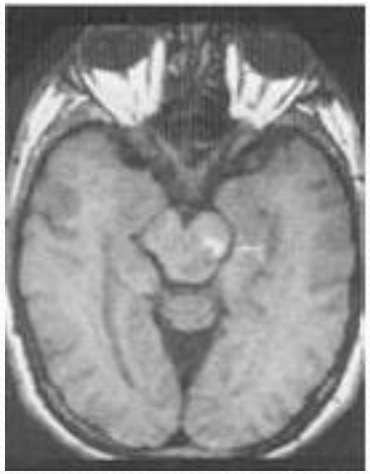
Concussion

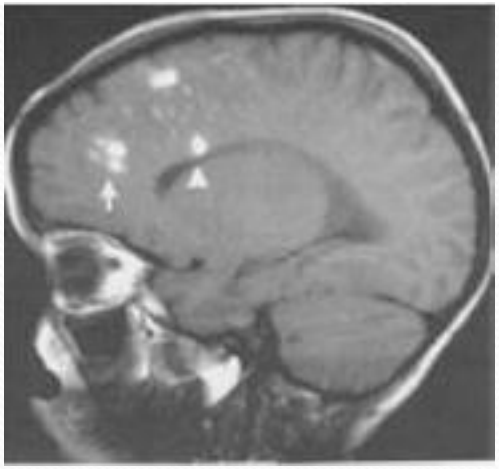
alteration of consciousness without structural damage as a result of non penetrating traumatic brain injury

Diffuse Axonal Injury

- A result of rotational acceleration/deceleration
- Severity of coma is out of proportion to imaging studies
- Often comatose immediately
- CT Scan, in severest form, may show hemorrhagic foci in corpus callosum and dorsolateral rostral brainstem (10-20%)
- Microscopic evidence of diffuse injury to axons
  - Axonal retraction balls
  - Microglial scars
  - Degeneration of white matter fiber tracts

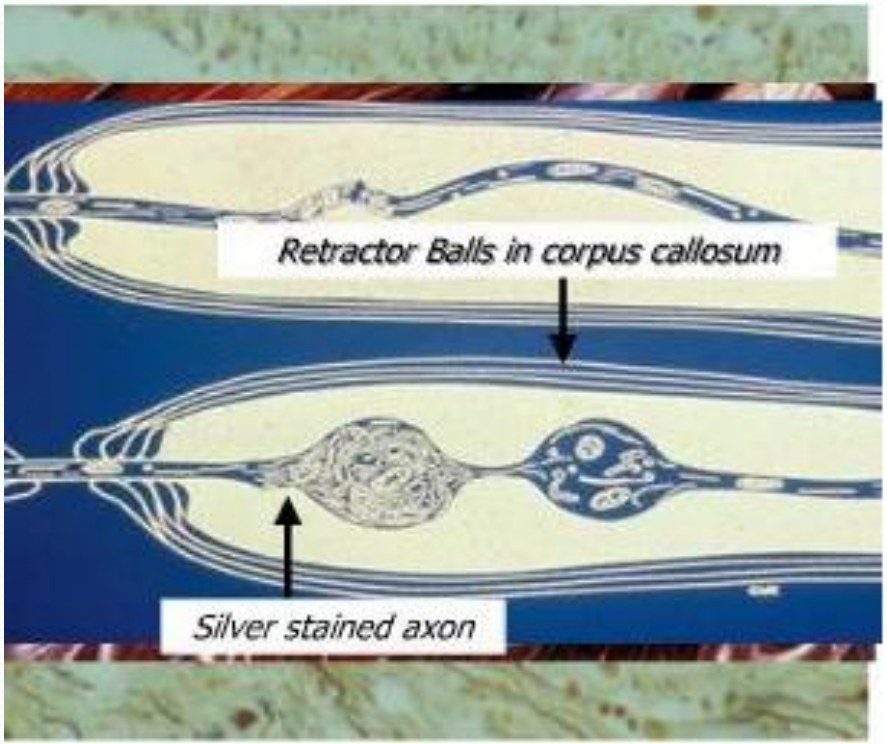






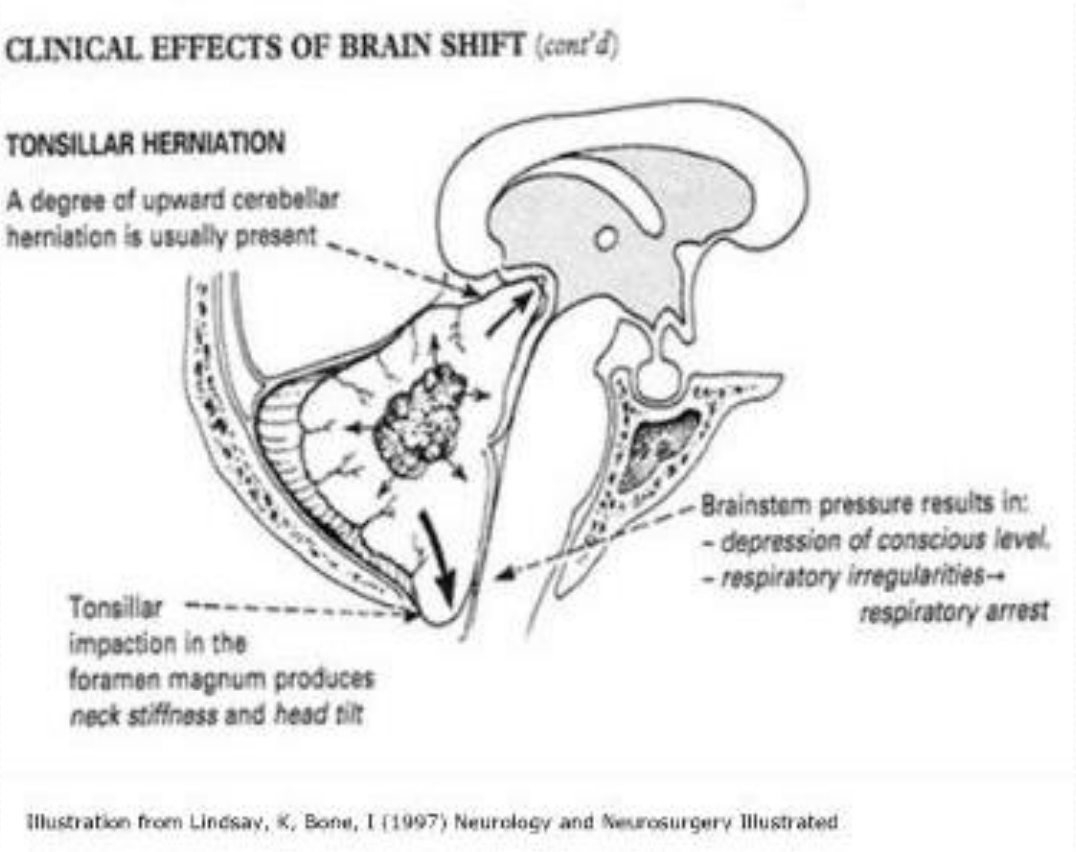
Diffuse Axonal injury - Neuropathology

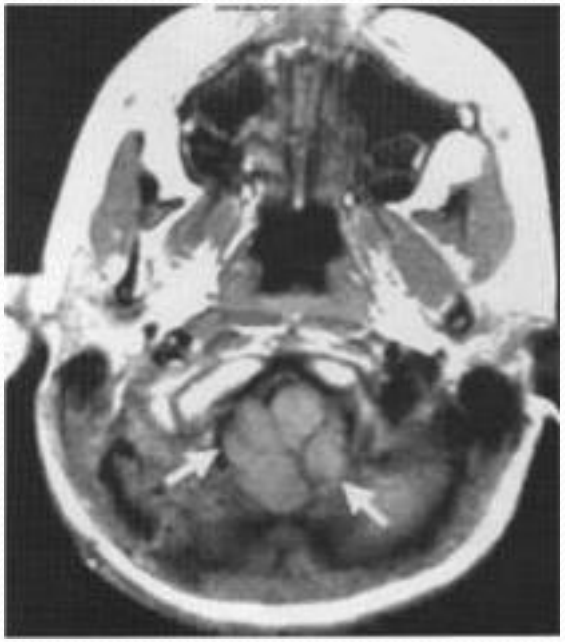
Ionic flux, Ca entry at nodes of Ranvier



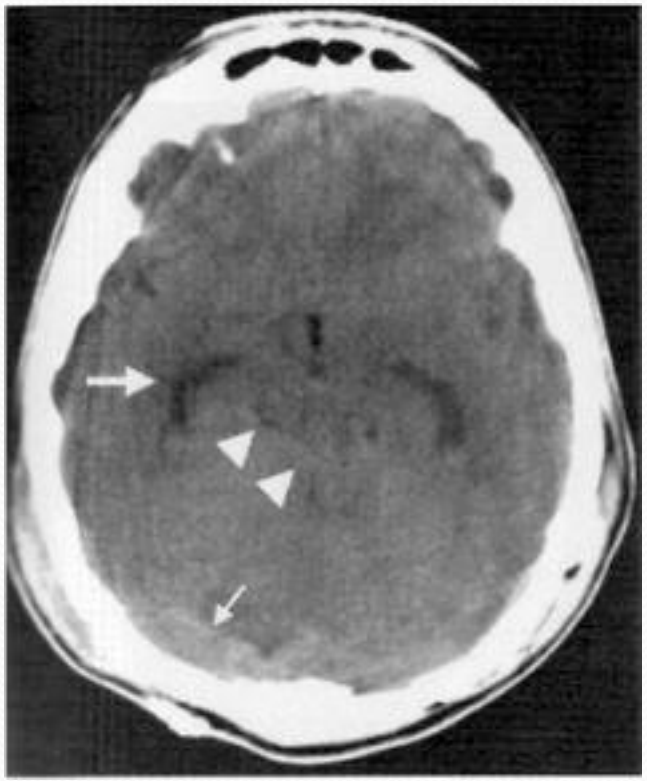
Tonsillar Herniation

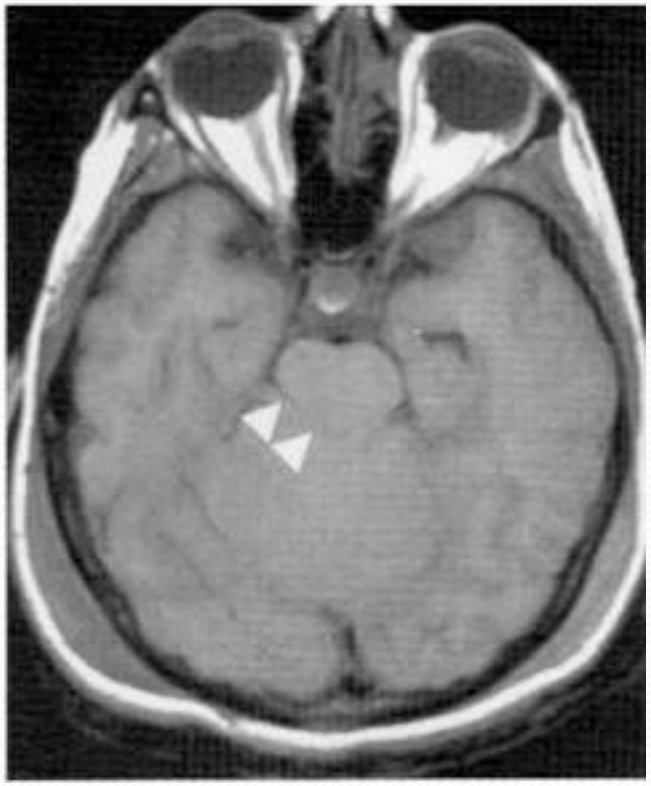
- Tonsils herniating through foramen magnum
- Supra or infratentorial masses
- Direct compression of medulla
- May show no signs of herniation





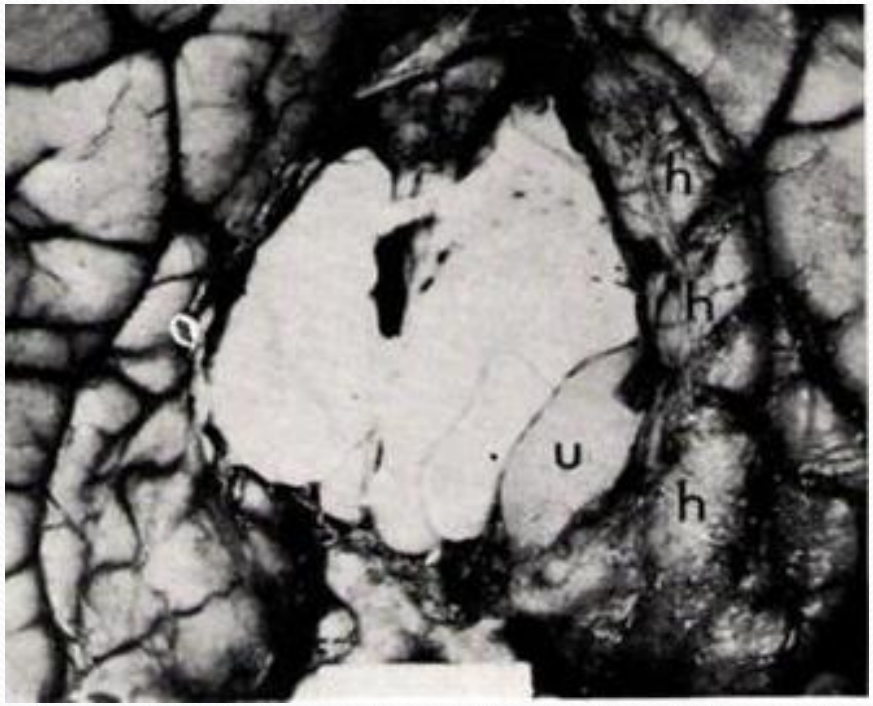
Tentorial Herniation

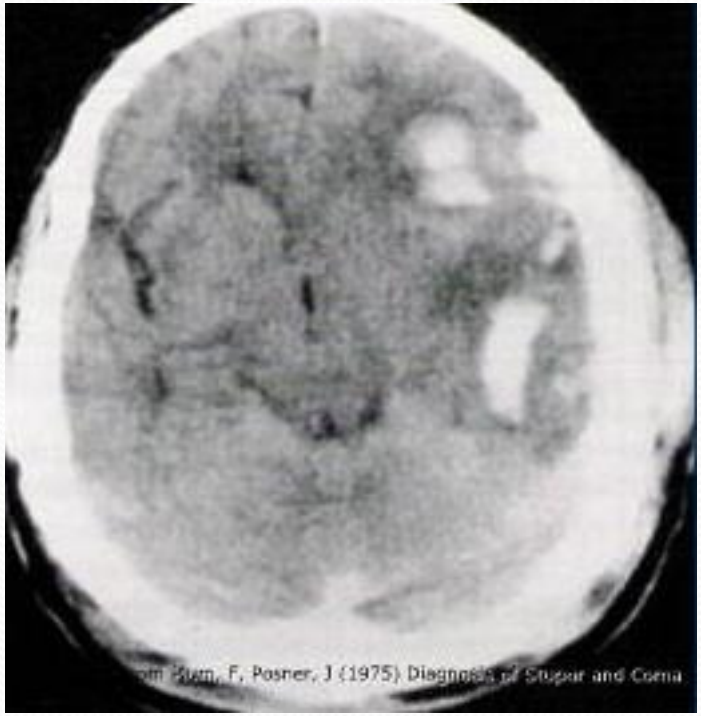




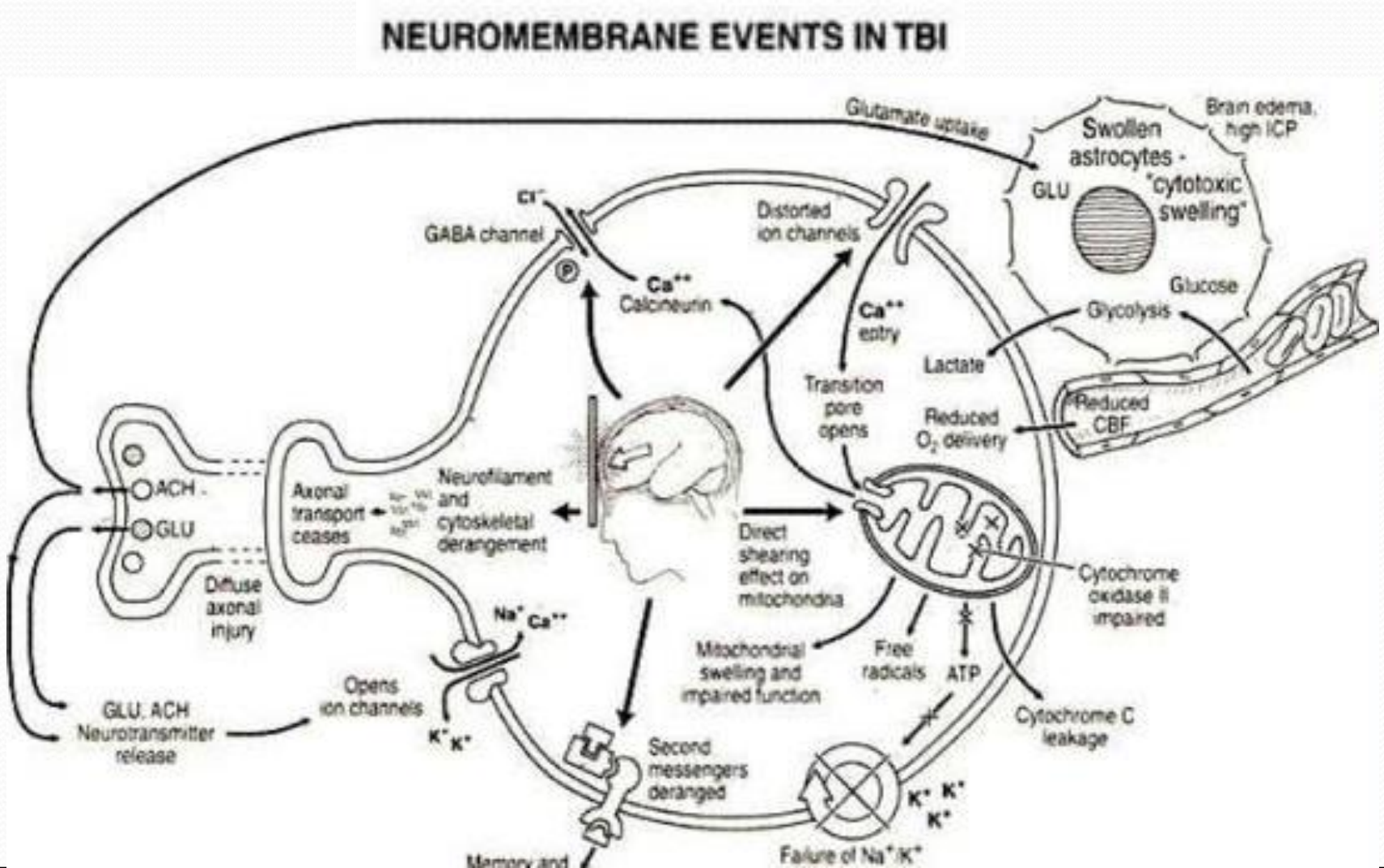
Uncal Herniation

- Middle fossa involving temporal lobe herniating uncus and hippocampus over tentorial edge with compression of midbrain
- Compression of oculomotor nerve, peduncle and PCA
- Contralateral hemiparesis
- Coma due to midbrain reticular activation system compression/ ischemia
- Ipsilateral pupil dilation
- Effects reversible, within 1-1½ hours





What happens at the subcellular level?

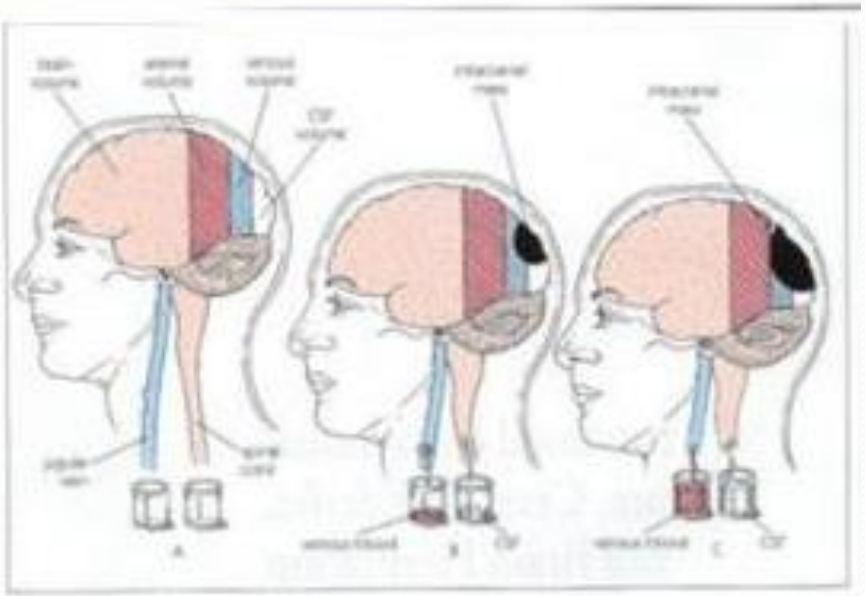


Monroe-Kellie Doctrine: Unique to the brain

- Monroe-Kellie Doctrine: Brain resides in closed compartment, the skull
  - Intracranial Contents (Incompressible)
    - Brain 80%
    - CSF 10%
    - Intracranial Blood 10%
  - Space/Mass Occupying Lesion
    - As it expands one of the three Intracranial Contents must compensate, to prevent herniation

Figure 9.1 Intracranial Contents and Their Respective Volumes

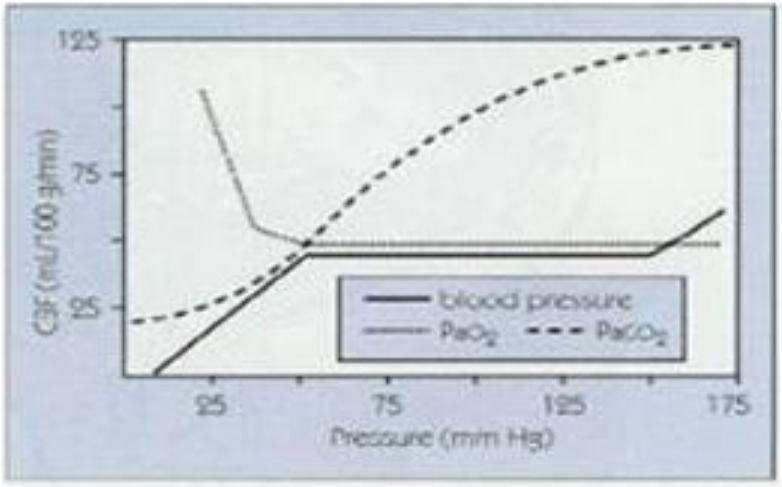
Content	Volume	Percentage of Total Volume
Brain (70%) and interstitial fluid (10%)	1400 mL	80%
Blood	150 mL	10%
CSF	150 mL	10%
Total	1700 mL	100%

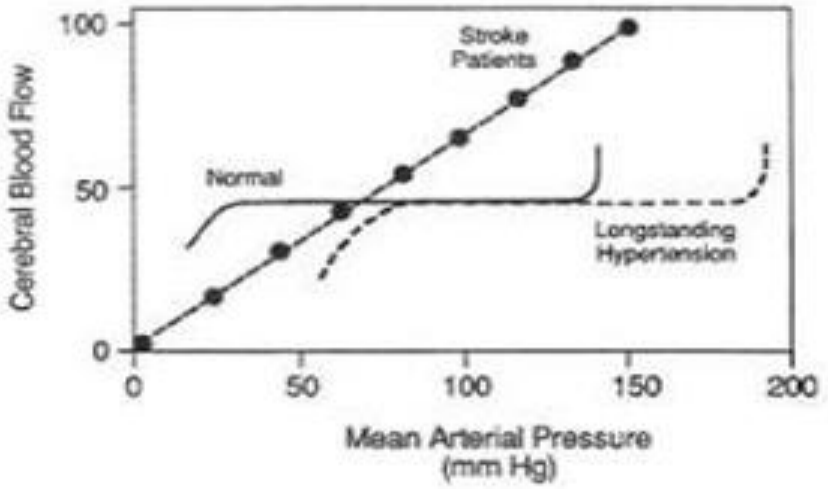


Autoregulation : Unique to cerebral vasculature

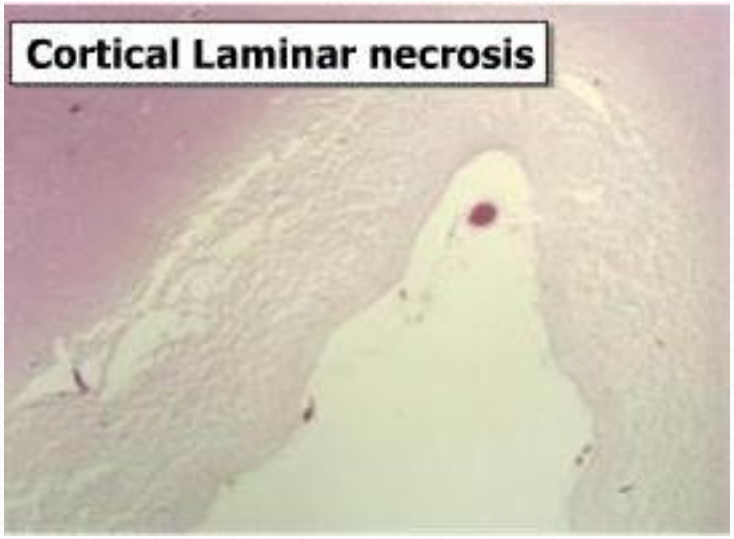
Ability to maintain CBF constant over wide range of Mean Arterial Pressure (50-150 mm Hg)

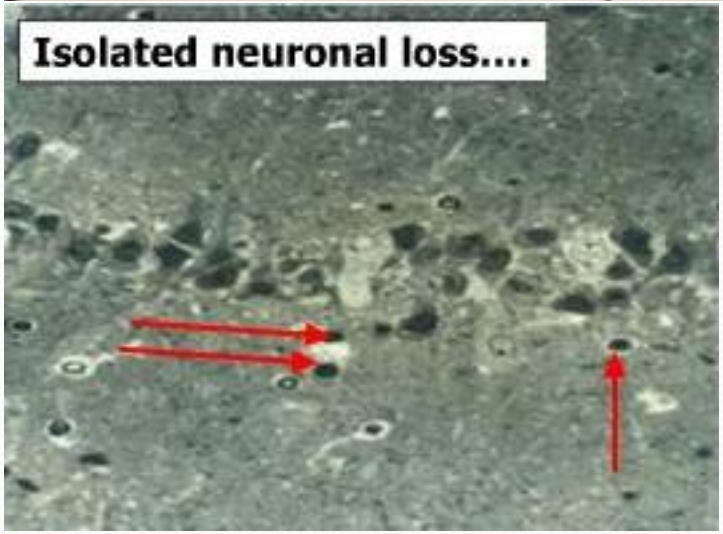
- Mechanism: Vasodilatation and vasoconstriction of intracerebral vessels
- P_aCO_2 and P_aO_2 also effect caliber of vessels CBF is maintained over a wide range of CPP
- Autoregulation of cerebral vasculature
- Malfunction of autoregulation in brain injury (CBF becomes linearly dependent on MAP)





What happens when the brain becomes ischemic?



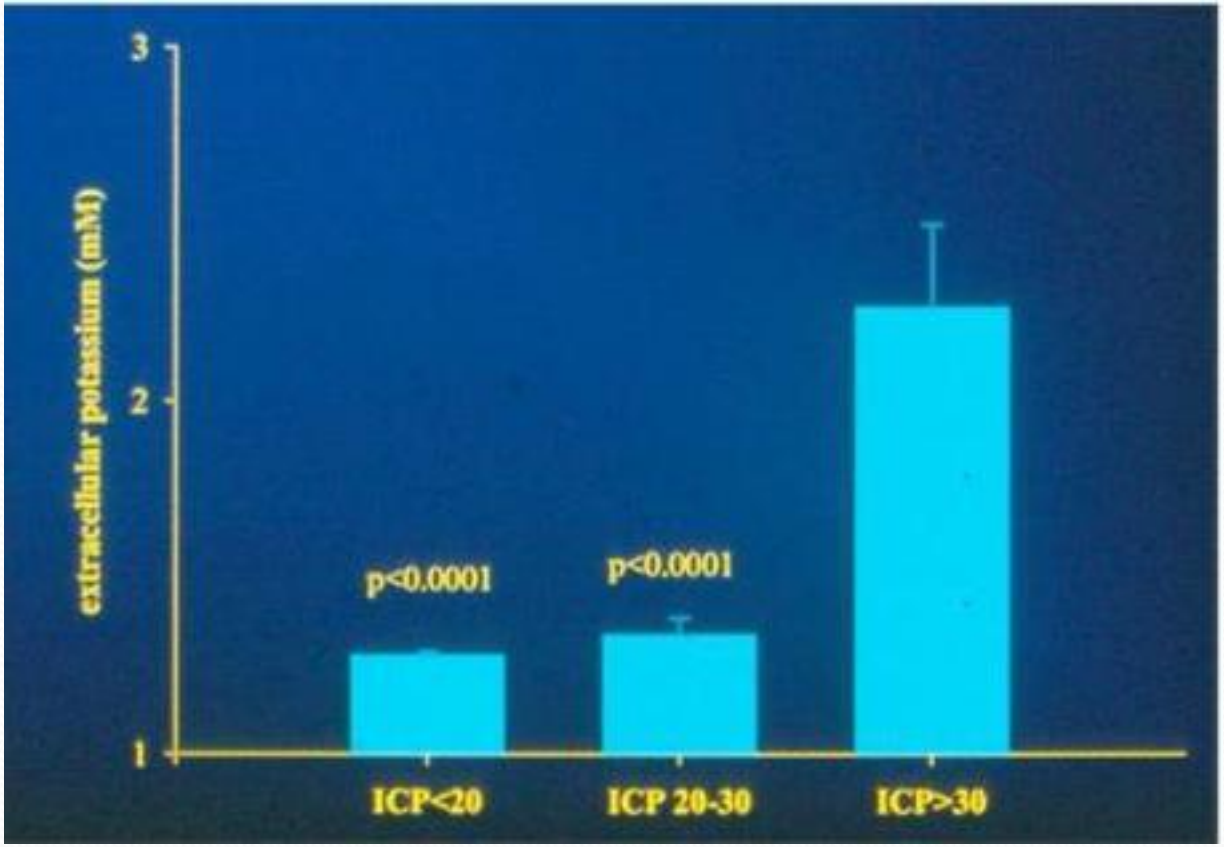


Secondary deterioration after head injury…

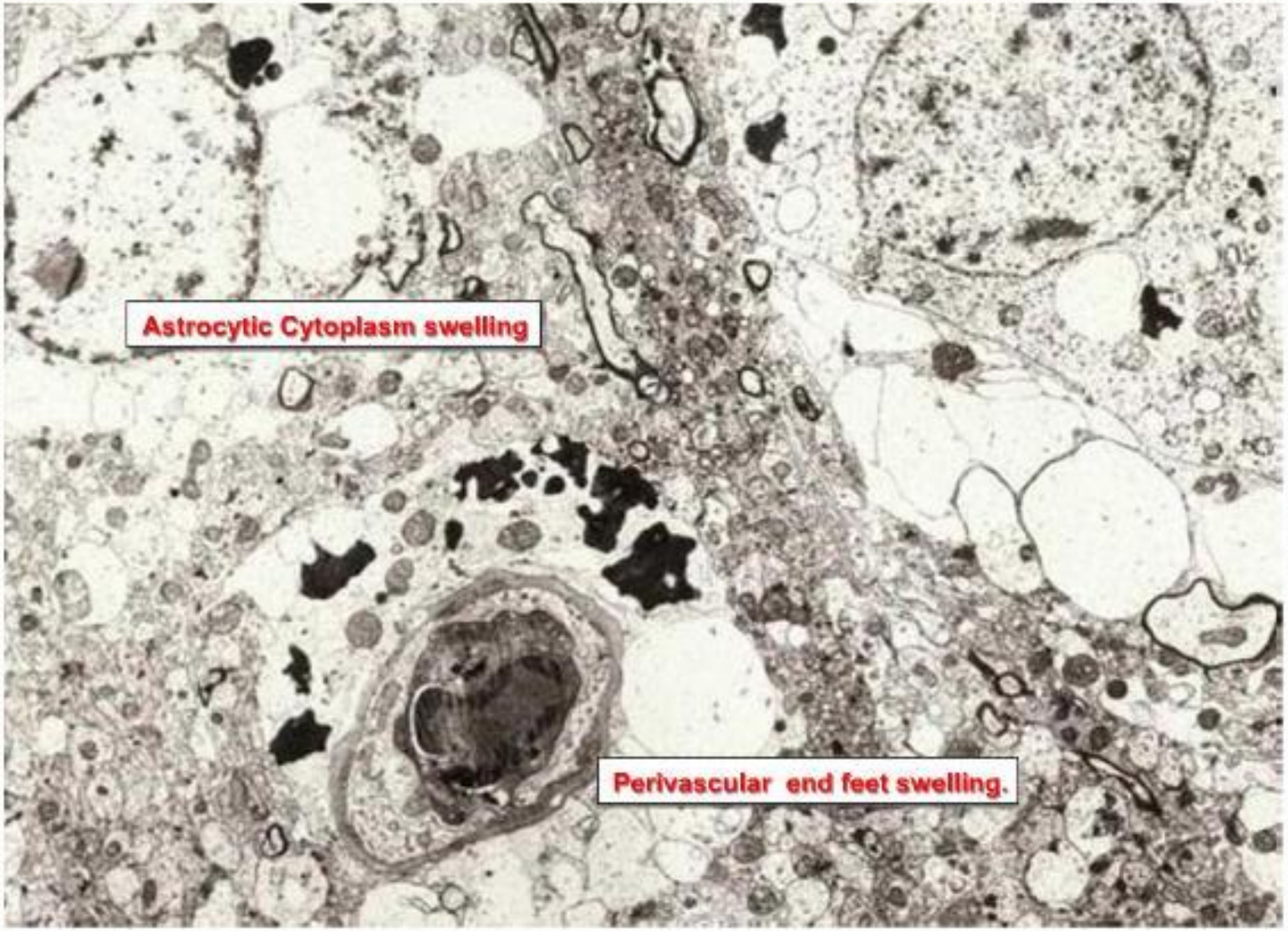
Occult hematoma	75%
Fever	~60%
Systemic hypoxia	15%
Meningitis	5%
Seizures	3%
Diffuse brain swelling	2%

Causes of Diffuse Brain Swelling – "Leaky" ion channels

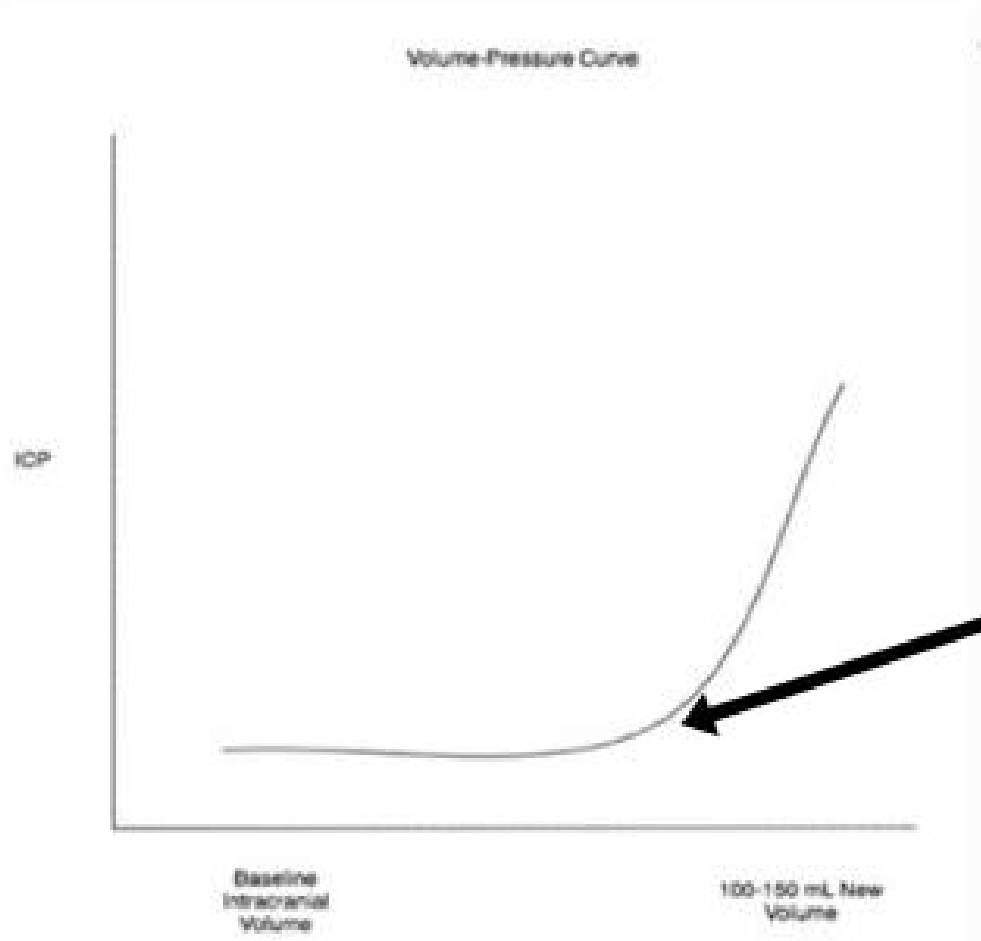
- Relationship between ICP and extracellular Potassium



Ultrastructure of Human cortex, with massive swelling



Volume Pressure Curve and ICP's



- Volume Pressure Curve
  - X axis Intracranial Volume
  - Y axis Intracranial Pressure
    - As the space/mass occupying lesion expands the ICP will not rise, because of compensatory mechanism (Monroe Kellie Doctrine.)
    - Once compensatory mechanisms exhausted small increases in volume will result in an exponential increase in ICP's

Systemic Manifestations of Head Injury

- Abnormalities of Water and Electrolyte Homeostasis
- Other Hormonal Abnormalities
- Neurogenic Pulmonary Edema
- Cushing's Ulcers
- Myocardial injury – e c g changes, vagus mediated Brady arrythmias

Levels of Consciousness

- Consciousness- awareness of self and environment
- Clouding of consciousness- mild depression in awareness, slow thinking
- Lethargic- impaired alertness, lacks spontaneous speech/alertness, may preserve intellect
- Obtunded- aroused with light touch/loud sounds
- Stupor- arousable with tactile stimulation
- Comatose- incapable of following commands, no eye opening overall inability to interact cognitively with environment
  - Recovery
  - Persistent Vegetative state
  - Permanent Vegetative state

Glasgow Coma Scale

- Eye opening
- Spontaneous
- To speech
- To pain only
- No eye opening
-
- Best Motor Response
- Obeys commands appropriately
- Localizes to stimulation
- Withdraws from stimulation
- Abnormal flexion response
- Abnormal extension response
- No motor response
-
- Best Verbal Response
- Oriented and appropriate
- Confused conversation
- Inappropriate words
- Incomprehensible sounds
- No verbal response

Objectives

- Recognize Head Injury
- Assess Injury Severity (GCS)
- Unique factors attributed to anatomy/physiology
- Common Types of head injury
- Imaging Characteristics (Herniation Syndromes)
- Pathophysiology
- Management
- Severe Head Injury Guidelines

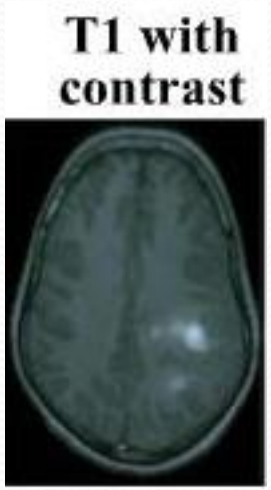
HEAD INJURY SEVERITY CLASSIFIED BY POST-RESUSCITATION GLASGOW COMA SCORE

13-15 = MILD INJURY (80%)

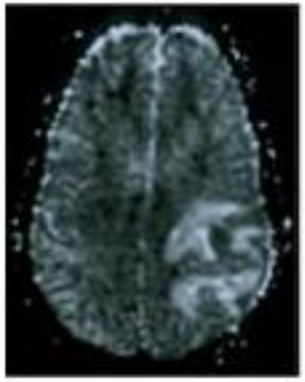
9-12 = MODERATE INJURY (10%)

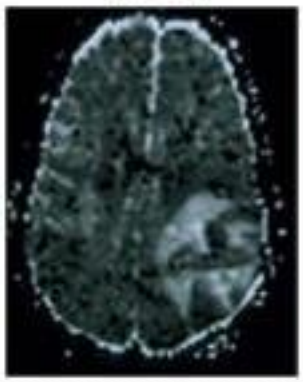
3-8 = SEVERE INJURY (COMA) [10%]

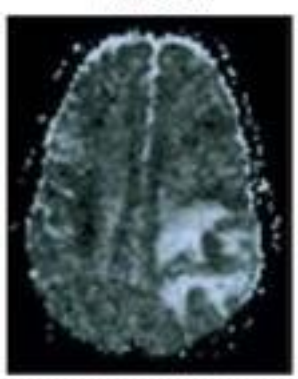
Diffusion Weighted Images in contusions

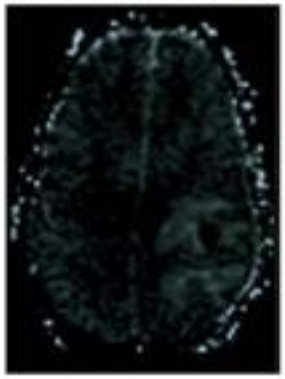


- *ADC is REDUCED early, in Peri contusional brain..indicates Water protons move into cells..*
- *So its mostly early **CYTOTOXIC Edema**...(Marmarou, et al..)*
- **Later BBB breakdown...**
- *Cytokine and Leukcocyte induced...*









Blood pressure and oxygenatior

- GUIDELINES : BP should be monitored and hypotension avoided
- (Keep systolic blood pressure < 90 mm Hg) avoided, or corrected immediately if possible.
- Oxygenation - HYPOxia
- OPTION : O2 should be monitored and hypoxia avoided
- (PaO2 > 100 mm Hg and/or SaO2 >90%) corrected immediately if possible.

KEEP THE BLOOD PRESSURE UP

-monitor arterial BP and CVP

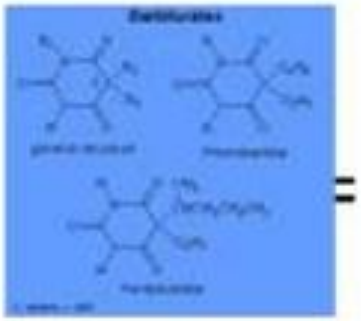
-keep euvolemia

-raise BP if CPP<60 (CPP=MABP-ICP)

Anesthetics Analgesics and Sedatives

GUIDELINE: ***Prophylactic*** administration of high dose barbiturates is contraindicated for intracranial pressure (ICP) control in patients with diffuse injuries, and is associated with significant hypotension.

OPTION: High dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability essential before and during barbiturate therapy.



= fall in

and may

Hyperosmolar Therapies –(1/2 New

- GUIDELINE: HI-ICP's Mannitol is effective for control of raised ICP's at .25gm/kg to 1gm/kg. Arterial hypotension should be avoided
  - Restrict mannitol use prior to ICP monitoring to patients with signs of transtentorial herniation and / or progressive neurological deterioratio not attributable to extracranial causes

Hypertonic saline use is an option to control intracranial hypertension

Fluid	Osmolarity
Ringer's Lactate	277 mmol/l
0.9 % NaCl	309 mmol/l
1.7 % NaC	598 mmol/l
3 % NaCl	1030 mmol/l
7.5 % NaCl	2575 mmol/l

Steriods

STANDARD:

The use of steroids is not recommended for improving outcome or reduc intracranial pressure (ICP).

In patients with moderate or severe traumatic brain injury (TBI), high dos methylprednisolone is associated with increased mortality and is contraindicated.

Antiseizure Prophylaxis

GUIDELINE:

Prophylactic use of phenytoin or valproate is not recommended for preventing late posttraumatic seizures (PTS).

> 7 days

Anticonvulsants are indicated to decrease the incidence of early PTS. However, early PTS is not associated with worse outcomes.

< 7 days OK

Immediate – within 24 hours of injury

Intracranial Pressure Monitoring Technology

COST RS /- or $$

- Can be recalibrated in situ.
- ICP transduction via fiberoptic or micro strain gauge devices placed in ventricular catheters provide similar benefits, but at a higher cost.

- Ventricular catheter connected to an ex accurate low cost reliable method of mo (ICP)

- Parenchymal ICP monitors cannot be recalibrated during monitoring. Comparison to zero drift after removal for current parenchymal micro strain gauge transduced ICP monitors is negligible at levels > +/-5 mmHg. The measurement drift is independent of duration of monitoring.

Subarachnoi d, subdural, epidural

- Subarachnoid, subdural, and epidural monitors (fluid coupled or pneumatic) are less accurate.

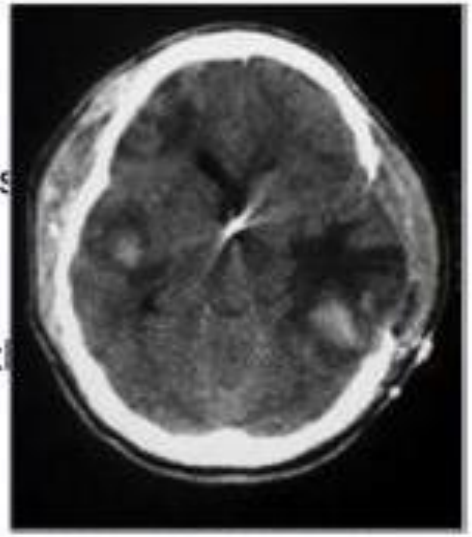
Indications for Intracranial Pressure Monitorir

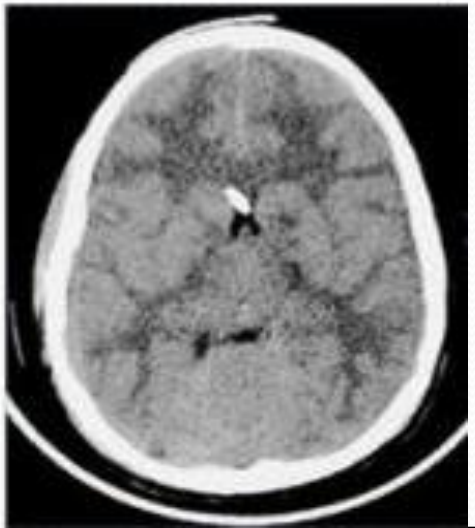
GUIDELINE: Intracranial pressure (ICP) should be monitored in all *salvageable* patients with a severe traumatic brain injury (TBI) (GCS 3 – 8 after resuscitation) and an abnormal CT scan. An abnormal CT scan of the head is one that reveals hematomas contusions, swelling, herniation, or compressed basal cisterns..

OPTION:

Indicated if GCS ≥ 8, with normal CT scan if two or more of t following features are noted at admission:

- **age over 40 years**
- **unilateral or bilateral motor posturing**
- **SBP < 90 mm Hg**





INTRACRANIAL PRESSURE

TABLE 9

*Outcome related to intracranial pressure**

ICP Status	No. of Cases	GR/MD (%)	SD/Veg (%)	Dead (%)
normal (0–20 mm Hg)	91	74	9	18
raised but reducible	74	55	19	26
not reducible	31	3	3	92
total	196	56	12	33

*ICP = intracranial pressure; GR = good recovery; MD = moderate disability; SD = severe disability; veg = vegetative state.

Miller and Becker, 1981

ICP Control Algorithm

Levels of Therapy

7 — Sedation
- Barbiturate or etomidate
- To burst supression on EEG

6 — Surgical decompression

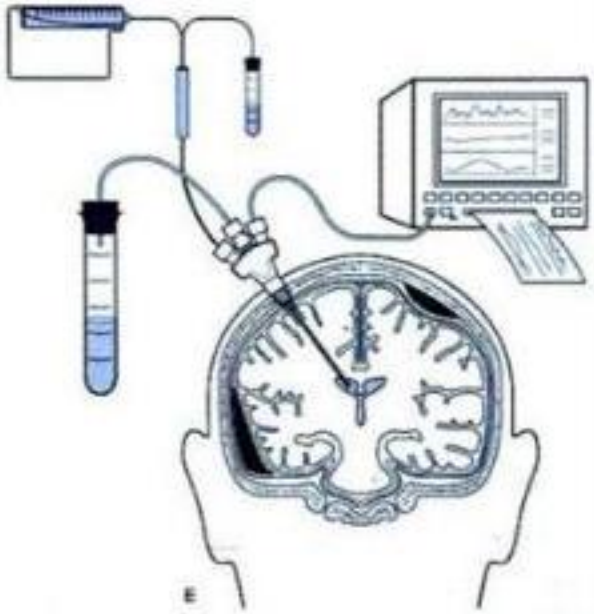
5 — Hyperventilation ($Pa\,CO_2$ 28 ± 5mmHg) + $AVDO_2$ monitoring or tissue PO_2 monitor

4 — Mild hypothermia (34 ± 2 C)

3 — Mannitol (0.25 – 0.5gm/kg)

2 — Ventriculostomy + ventricular drainage

1 — Intubation, normocarbic ventilation ($Pa\,CO_2$ 35 ± 2mmHg)



Hyperventilation

GUIDELINE:

Prophylactic hyperventilation ($PaCO_2$ of 25 mm Hg or less) is not recommended.

OPTION:

Hyperventilation is recommended as a temporizing measure for the reduction of elevated intracranial pressure (ICP).

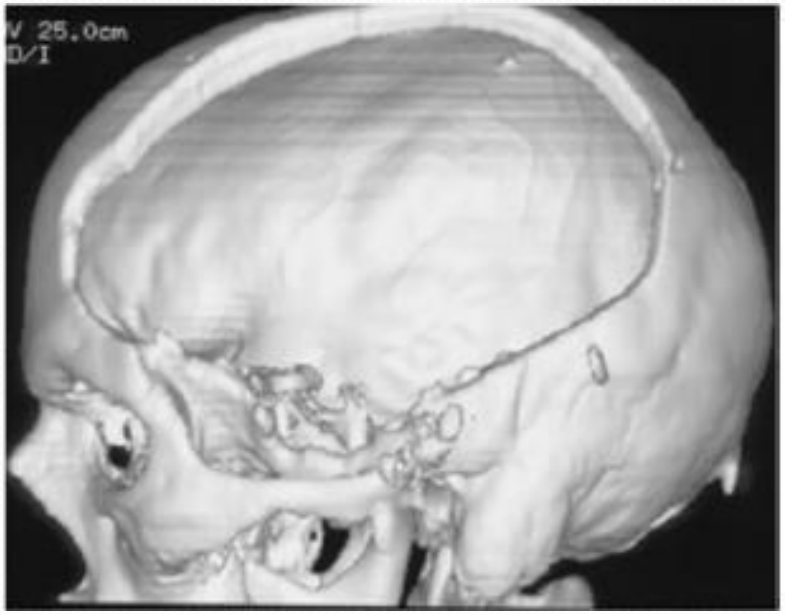
<25

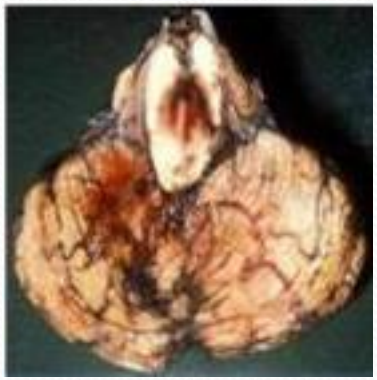
Hyperventilation to a $PaCO_2$ below 29 mm Hg should be avoided during the first 24 hours after injury, when cerebral blood flow (CBF) is often critically reduced.

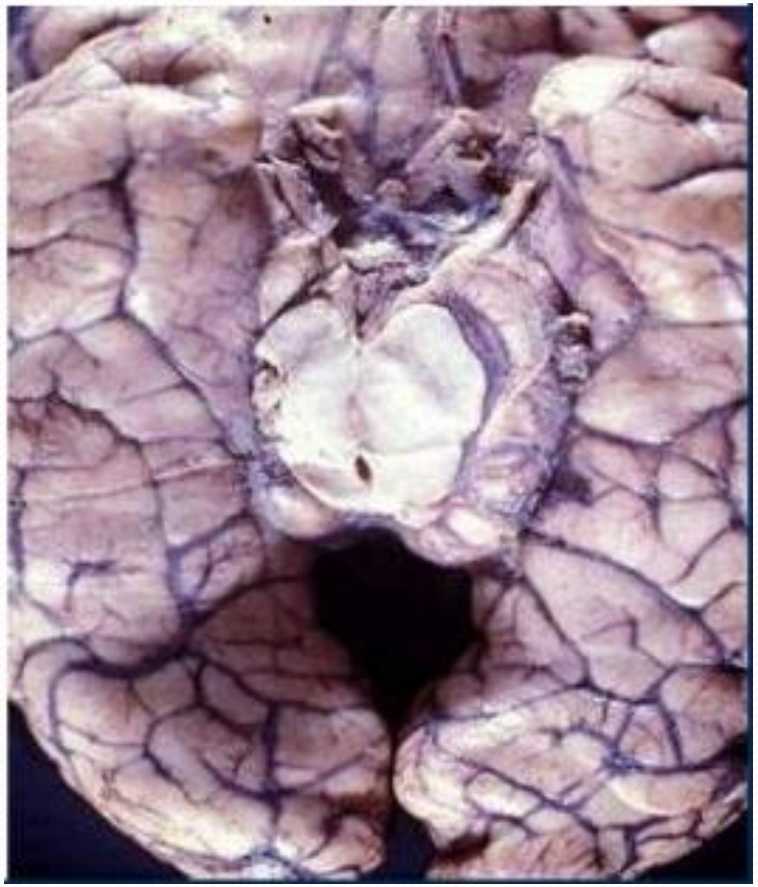
<29 in 1st 24

If hyperventilation is used, jugular venous oxygen saturation (SjO_2) or brain tissue O2 partial pressure ($PbtO_2$) measurements are recommended to help determine if hyperventilation is causing ischemia.

Massive decompressive craniectomy and duroplasty-
The most effective current therapy, for high ICP……??







Gun Shot Injuries

- Surgical Treatment
  - Patients with poor neurological condition including fixed and dilated pupils, decorticate posturing, intractable ICP, decerebrate posturing have about 95% mortality
- Goals of Surgery—Always consider ICP monitor…
  - Debridement of devitalized tissue
  - Evacuation of hematomas
  - Removal of accessible bullet or bone fragments
  - Water tight dural closure
  - Obtaining hemostasis
  - Separation of intracranial compartments from air sinuses
- Prognostic Factors
  - Level of consciousness
    - 94% of patients GCS 3-5 admission die
    - 3% of survivors are severely disabled
  - Path of bullet
    - Crosses midline—almost always fatal..
    - Pass through center of brain
    - Enter or traverse the ventricles
    - More lobes traversed, worse off
  - Hematomas are poor prognostic factor
  - Suicide attempts are more frequently fatal