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HEAD INJURY

Head Injury ..... the silent epidemic!

It occurs every 15 seconds throughout the world It is the commonest cause of morbidity and mortality world wide Two thirds of victims are under 35years of age Males are twice as affected as compared to females

Each year one million people are treated in casualty for head injury, out of which 50,000 die and 80,000 become permanently disabled About 50 billion dollars spent each year in their management worldwide Increasing road accidents in India are attributed to unique combination of high and low speed traffic

Modes of Injury

- Various modes viz. vehicular accidents, homicidal, industrial, domestic, armed hostilities, and natural calamities, and even sports injuries
- Vehicular accidents are overwhelmingly higher than any other mode throughout the world
- Vehicular accidents comprise motor vehicle, bike, and Airplane crash and even sinking of boats

Strict implementation of traffic rules, easy & cheap availability of radio imaging, and

improved ICU facilities have reduced mortality to significant extent, but conversely morbidity has been persistently rising

'Loss of consciousness is not mandatory to label as Head injury'

"No head injury is so trivial as to be ignored, nor so severe as to be despaired of"

Types of Head injuries

Primary :

- Immediate & direct result of the trauma
- Occurs within seconds after impact, can be of variable intensity
- May be anatomically classified as either Focal or Diffuse
- Focal injuries : contusions, fractures, coup & contrecoup, hematomas(epidural, subdural), and CSF leak
- Diffuse injuries : concussion & diffuse axonal injury

Secondary :

- Cellular damage set into motion by the cascade of events following initial injury, that develops over hours to days after initial traumatic insult
- It includes cerebral edema, brain herniations

Secondary insult :

- Refers to factors aggravating primary or secondary injuries, such as hypoxia, hypotension, raised Intracranial tension (ICT), cerebral ischemia
- Commonly follows severe head injury .

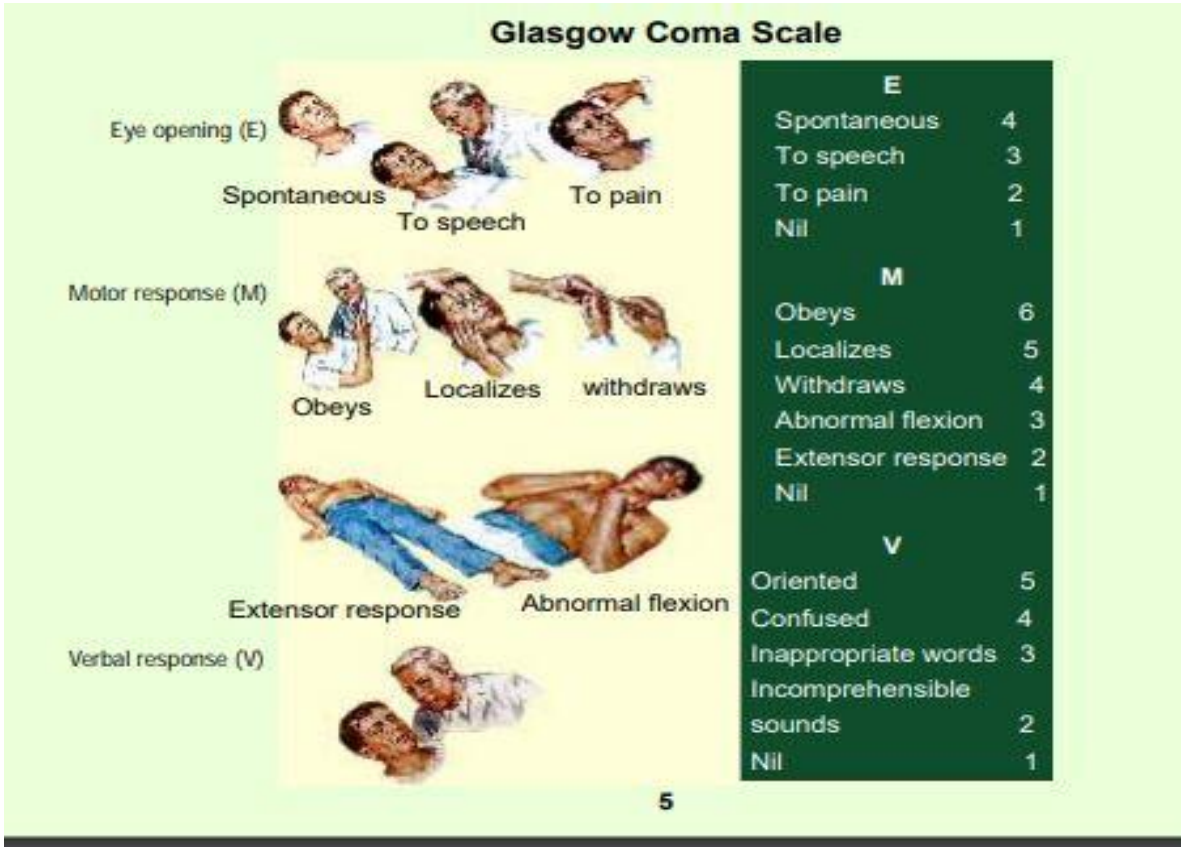
Head injury & Shock make a deadly combination!

Classification of Head injuries : :based on Glasgow Coma Scale (GCS)

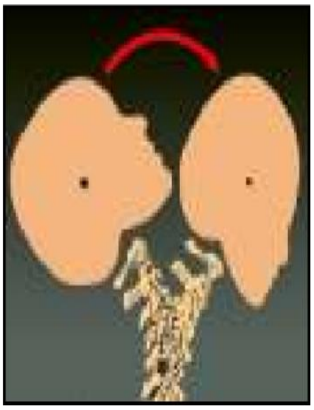
Glasgow coma scale :

- Introduced in 1974 & revised in 1977 by the addition of motor response level
- Has been adopted unanimously by neurosurgical units world wide


PERFORM ABCs OF RESUSCITATION
WHAT IS PATIENT'S GLASGOW COMA SCALE (GCS) ? *
GCS 13-15
MINOR HEAD INJURY
CT urgently needed if all 3 of the following:
1. History of blunt head trauma within the last 24 hours
2. History of loss of consciousness, amnesia or disorientation
3. One or more of the following:
• GCS < 15 at 2 h after injury
• Suspected open or depressed skull fracture
• Sign of basal skull fracture (hemotympanum, "raccoon eyes", cerebrospinal fluid oto/rhinorrhea, Battle's sign)
• Vomiting x 2 or more times
• Age > 65
Minor Head Injury Management
Abnormal CT
• Call Air Medical Critical Care Transport and Trauma Hotline at
1-800-743-1334
Normal CT or no CT indicated
• Observe until well and discharge home
• Follow up with primary care physician
If GCS deteriorating, or evidence of penetrating head injury, treat as per "Major Head Injury"
GCS 3-12
MAJOR HEAD INJURY
Intubate (c-spine in neutral position) for GCS ≤ 8 or deteriorating GCS; oxygen by mask for all others
Spine immobilization (c-spine collar and back board)
2 minute neurological assessment:
• GCS
• pupil size and reaction to light
• biceps and knee jerk reflexes
• Babinski responses
• Gross motor function (equal movement in all 4 limbs?)
Call Air Medical Critical Care Transport and Trauma Hotline at 1-800-743-1334
PREPARATION FOR TRANSPORT AS PER PROVINCIAL TRAUMA GUIDELINES
PEDIATRIC HEAD INJURY (AGE < 16 YEARS)
GCS 3-12 (Major Head Injury)
Treat as per adult guideline
GCS 13-15 (Minor Head Injury)
AGE 2-15 years:
GCS 13 or 14: CT Head
GCS 15:
History of LOC or PTA: CT Head or observe overnight
No LOC or PTA: CT Head if one or more of the following:
• Change in GCS
• Focal neurologic deficit
• Intoxication
• Clinical evidence of skull fracture
• History of coagulopathy
AGE <2 years: (Consider child abuse as cause)
Symptomatic and/or neurologically abnormal: CT Head
Asymptomatic and normal neuro exam: No further imaging studies except if <1 year and scalp hematoma, then do skull x-rays
• If x-rays normal: No further investigations
• If fracture: CT Head & contact neurosurgery
Symptomatic: LOC, vomiting, amnesia, irritability
LOC: Loss of consciousness
PTA: Post traumatic amnesia
CT: Computerized tomography
* CALCULATE GLASGOW COMA SCALE (E + V + M; RANGE 3-15)
GCS for age ≥ 5 years
EYE OPENING
VERBAL RESPONSE
BEST MOTOR RESPONSE
BEST GCS WORST
E = 1-4
V = 1-5
M = 1-6
GCS for age < 5 years
BEST MOTOR RESPONSE
VERBALIZATION
EYE OPENING
Mr 1-6
V = 1-5
E = 1-4

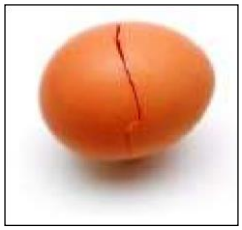

Glasgow Coma Scale
Eye opening (E)
Spontaneous
To speech
To pain
Motor response (M)
Obeys
Localizes
withdraws
Extensor response
Abnormal flexion
Verbal response (V)
E
Spontaneous 4
To speech 3
To pain 2
Nil 1
M
Obeys 6
Localizes 5
Withdraws 4
Abnormal flexion 3
Extensor response 2
Nil 1
V
Oriented 5
Confused 4
Inappropriate words 3
Incomprehensible sounds 2
Nil 1
5

Type	GCS SCORE
Minimal	15, No loss of consciousness, amnesia
Mild	14, or 15 and amnesia or brief loss of consciousness (LOC) or impaired alertness
Moderate	9-13 or LOC ≥ 5min or focal neurodeficit
Severe	5-8
Critical	3-4



Biomechanics of head injury

According to Goldsmith theory, collision of the head with a solid object at an appreciable velocity causes impulsive load, producing sudden motion of the head without significant physical contact, and results in straining of tissues



Static load compresses the head with a gradual force over greater period of time

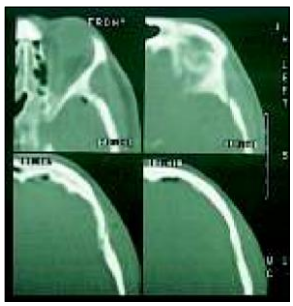
Types of acceleration :

Translational acceleration : Brain moves in a straight line

Rotational acceleration : Brain moves around its a center of gravity
Pressure cracks the skull like an Egg Angular acceleration : Movement of Brain in an angular manner akin to combination of above to

Fractures :

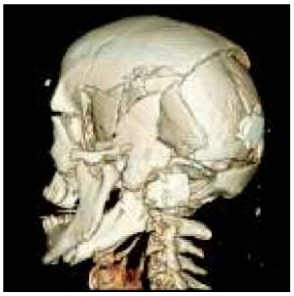
- Skull fractures are stigmata of significant skull and head injury
- Found in 8% of fetal head injuries



A) Linear Fractures : Simple fracture line, tends to radiate away from site of impact, along the line of least resistance

A result of broad based forces such as traffic accidents and fall

B) Basilar Skull Fractures :



May be linear, depressed or comminuted

Usually limited to base, though can spread to the calvarium

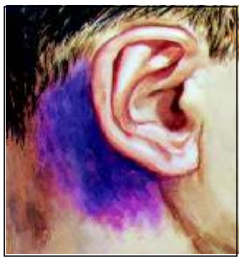
Multiple interlacing fracture lines suggest multiple blows

CSF leak can be evident in the form of rhinorrhoea, and or otorrhoea, which are the cause of concern

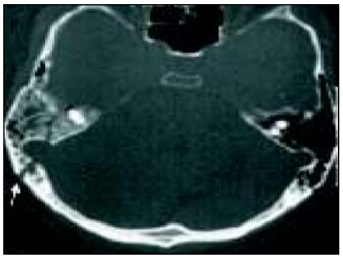
Cranial nerve palsies are associated injuries, not to be missed!

Panda

Panda Sign



Battle Sign



Posterior fossa fracture

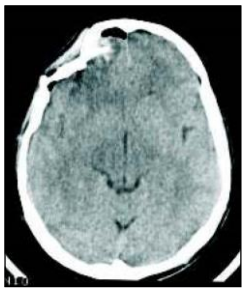
c) Depressed Fractures:

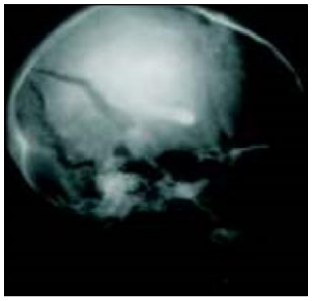
Produced by forceful & heavy impact, striking the skull over a small area leading to impingement of portion of fractured bone on the brain

If the fracture shows bending outward distal to the impact site, it's known as "Bursting Fracture"

When fracture separates the suture line, it's called "Diastatic Fracture"

Fractures may be closed or open depending upon nature of overlying soft tissue injuries





D)Comminuted Fractures:

Results from forceful / heavy impact striking on wider area of skull breaking it into multiple

E) Compound Fracture:

If fracture line is communicating to exterior through lacerated wound over scalp

F) Childhood Fractures:

Most of them are due to abusive trauma, traffic accidents, fall from height or sports injuries

G) Growing Fracture of Skull:

Term used originally by Pia and Tonnis, in 1953

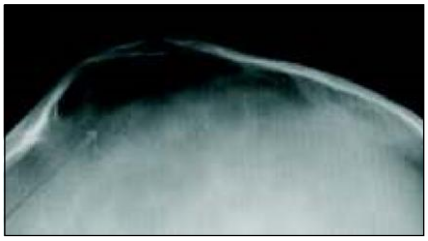
Synonyms – Leptomeningeal cyst, craniocerebral erosion, cerebrocranial erosion, Meningocele Spuria (Bilroth, 1862),

Cephalhydrocele (Godlee, 1885), Traumatic ventricular cyst,

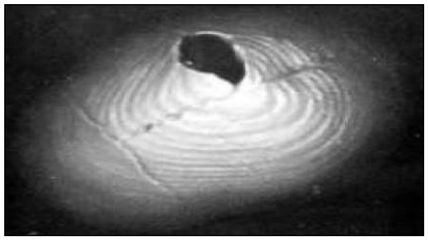
Fibrosing osteitis, Craniomalacia, Expanding skull fracture

Frequently found in infants & children

Though average is variable, the usual time interval between injury & evolution is 4 to 6 wks



Plain X-rays

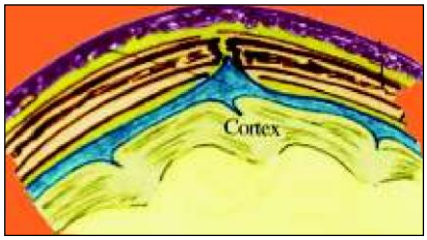


CT scan 3D reconstruction

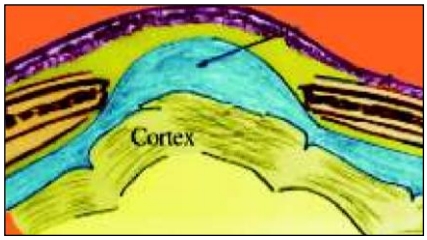
Pathogenesis :

Hypothesised as out-pouching of arachnoid through breached dura by pulsations of brain with each heart beat

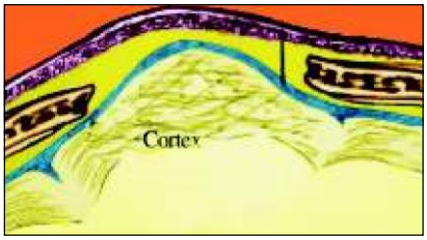
Fluid enters the pouch and the neck acts as unidirectional valve



Stage I



Stage III



Stage II

With each pulsation, cyst enlarges, erodes the bone & separates the dural edge more and more

Underlying brain becomes gliotic and encephalomalacic

Frequent features are progressive, cystic, scalp swelling, focal/generalized seizures, cranial defect, hemi / monoplegia and even a proptosis

X-ray skull shows characteristic irregular / elliptical skull defect with tapering ends

Margins are sclerotic, scalloped & everted

Outer table is less affected than inner

Rarely even expansion of diploic space & enlarged orbits have been demonstrated

CT scan : Apart from bony destruction, it reveals gliotic brain, porencephalic cyst, hydrocephalus, pulling of ventricles, or ventriculomegaly

MRI provides detail information about underlying brain but is poor in demonstrating bony changes

Treatment:

No role for conservative line of treatment

Anticonvulsants to control seizures

Indications
Scalp swelling
Rapid progressive hemiparesis
intractable epilepsy

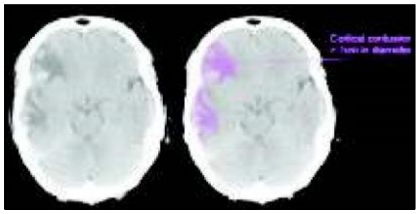
Surgical Treatment :

Aimed at repair of bony & dural defect

Surgical treatment does not prevent epilepsy; however excision of gliotic brain might reduce frequency of epileptic attacks

Cranial defect is repaired by simultaneous cranioplasty

Contusion



Bruise of cortical surface of the brain

Results from hemorrhages around cerebral vessels, brain necrosis and infarction

Pia mater is always intact which differentiates

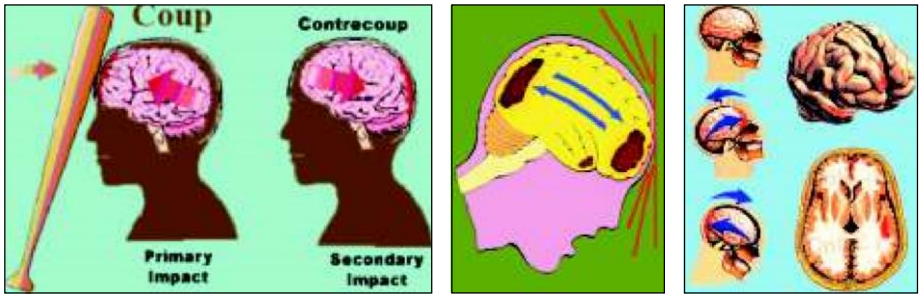
them from laceration CT scan : contusion

Are wedge shaped with base toward surface and apex directed towards ventricle

Extent of damage depends on forcefulness of impact

Classically divided into nonhemorrhegic or hemorrhagic

Hypertensive, alcoholic or patients having bleeding diathesis will have large & hemorrhagic contusion



Coup contrecoup Mechanism

Coup Contusion : Appears at the site of cranial impact

Countercoup Contusion : Found at diagonally opposite the site of impact

Herniation Contusion : at the margins of brain herniations, frequently located along margin at falx cerebri, tentorium or foramen magnum

Facture Contusions : Bruises along the edges of skull fractures

Gliding Contusions : Deep parenchymal injuries which may/ may not be comparable to surface contusion

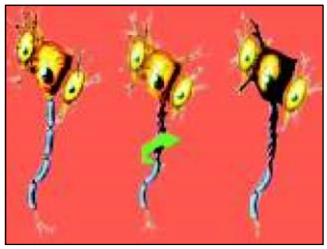
Mechanism :

Rotational movements of brain generated by impact, leads to shearing forces which produce countercoup injury

As contusion ages, necrotic cortical tissue is phagocitized, leaving behind

& shrunken brown scar

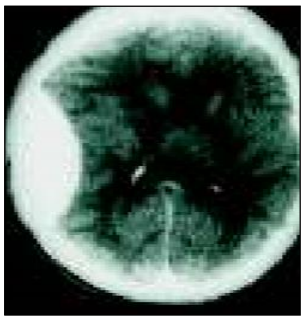
Mechanism



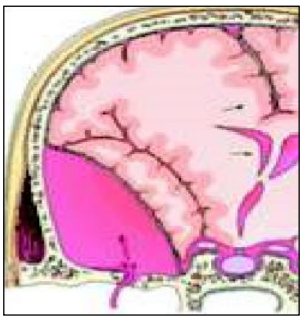
Neuronal Injury

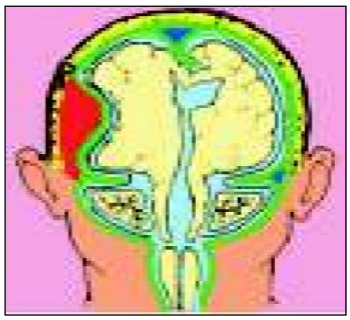
Hematomas:

Epidural Hematomas:



CT scan





Epidural Hematomas

- Is a collection of blood between the dura and the skull bone
- Incidence is 1 to 3% in head injury patients
- There is steep rise in its incidence in patients with GCS < 8
- Usual sites of occurrence are temporal (60-80%) & frontal (7-20%)
- They are demonstrated even at other sites
- Fall, vehicular accidents & assaults give rise to epidural hematoma

Pathogenesis:

Following impact → skull bends → stripping of dura from inner table

↓

Extradural hematoma ← Bleeding ← Vascular Trauma

Middle meningeal artery is the commonest source, rarely dural venous sinuses and diploic veins lead to epidural hematoma

Clinical Manifestations : No definite symptoms but classically five patterns have been described

- Conscious throughout
- Unconscious throughout
- Initially unconscious & subsequently recovered
- Initially conscious followed by deterioration
- Classical description of unconsciousness, recovery and followed by second loss of consciousness (Interval is Lucid interval)

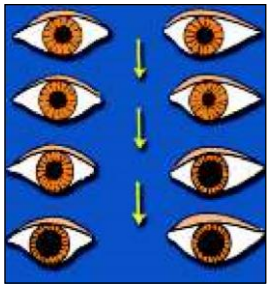
Triad of lucid interval, ipsilateral mydriasis and contra/ipsilateral hemiparesis (Kernohan's notch) and transient followed by permanent loss of vision

Hutchinson's Pupil : Initial ipsilateral papillary constriction (IIIrd nerve irritation)

↓

↓ Ipsilateral dilatation (IIIrd N. Palsy)

Bilateral dilatation (Bil IIIrd N. Palsy)



Normal

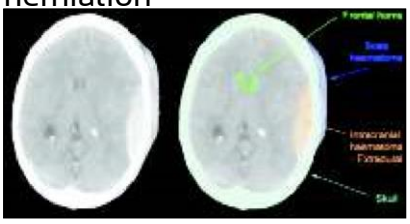
Ipsilateral constriction

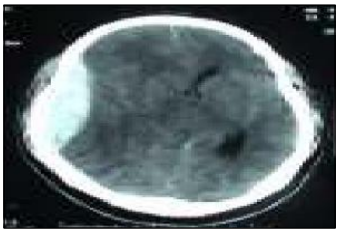
Ipsilateral dilatation

Bilateral dilatation

"CT Scan of brain quickly rules it out!"

Biconvex, hyperdense SOL, beneath skull vault causing subfalcine/ transtentorial herniation



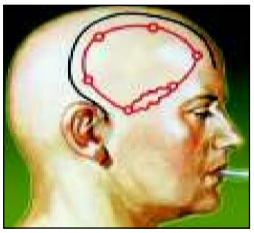


CT scan : epidural hematoma

Treatment :

Small epidural hematomas with good GCS score can be treated conservatively

Surgical treatment is indicated in progressively deteriorating patients (GCS) or large hematoma


Skin incision


Craniotomy


Epidural hematoma

Posterior fossa epidural hematoma:

- Rare lesion
- Associated with fracture of occipital bone & transverse sinus injury
- Headache, nausea, vomiting & neck stiffness are presenting symptoms
- Prompt surgical evacuation is essential to save life

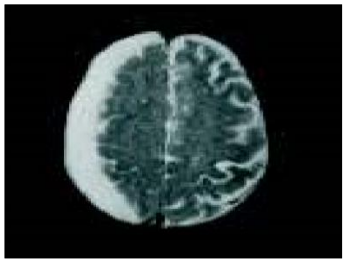
"Clinical signs of Neurological deterioration without pupillary changes are a clue to diagnosis"

Subdural Hematomas :

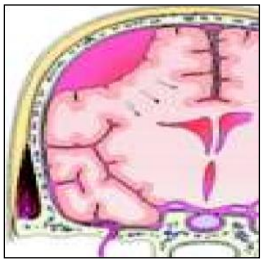
Collection of blood between dura and arachnoid mater

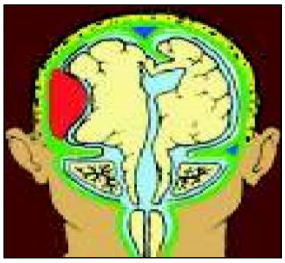
Types	Duration
Acute	Within 48 to 72 hrs of injury
Sub acute	between 3 & 20 days
Chronic	between 3 wks & several months

- Found in 10 to 30% of severe head injury
- Males are affected thrice as common as females
- Outcome of patient is decided by extent of underlying brain injury



MRI





Subdural hematoma

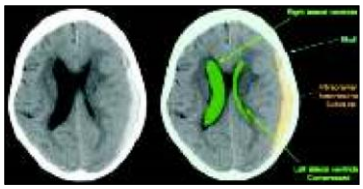
Pathogenesis :

- Rupture of parasagittal and sylvian bridging veins or cortical arteries following angular acceleration
- May affect perisylvian, or medial surface of cerebral hemispheres
- Associate injuries may be countercoup injury, contusions, lacerations and
- diffuse axonal injury
- **Burst Lobe :** Acute subdural hematoma, extensive lobar contusions and intracerebral hemorrhage together are referred to as burst lobe
- **Clinical Findings :**
- Headache, nausea, vomiting, seizures, aphasia, and contra/ ipsilateral weakness
- Lucid interval
- Cushing's triad - bradycardia, hypertension & bradypnea

(Signs of transtentorial herniation)... & impending death!

True Localizing signs	False Localizing signs
Ipsilateral pupillary dilatation with no reaction Contralateral motor weakness	Contralateral pupillary dilatation (optic/oculomotor nerve injury) Ipsilateral motor weakness (Kernohan's notch)

Investigations :



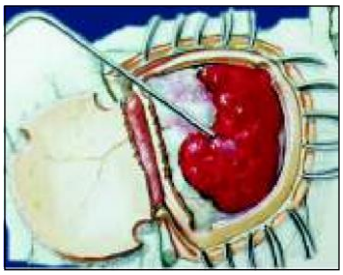
CT scan : Subdural hematoma

Investigation of choice – CT scan!

Appears as hyperdense, extra-axial, crescentic mass lesion, concavity of which follows brain surface while convexity outwards

MRI though not the fist modality of imaging, is ideal for detection of underlying brain injury

Treatment :



Subdural hematoma

Surgical evacuation is indicated in hematoma >5mm thickness, midline shift & progressive deterioration of neurological status

Careful study of CT scan to detect injury of underlying brain is essential.....

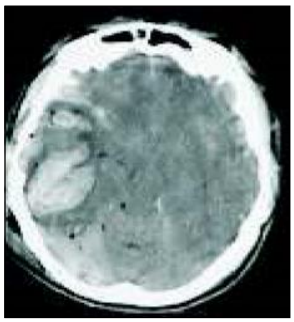
Intra-operative brain swelling: A matter of concern for Neurosurgeon as it poses difficulty in managing the patients intra & or postoperatively

"Failure of cerebral autoregulation"

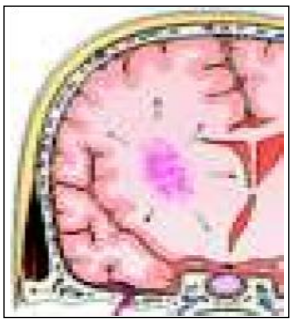
Causes may be cerebral vascular engorgement, epidural hematoma on opposite side, intracerebral contusion hematoma, or even a residual hematoma

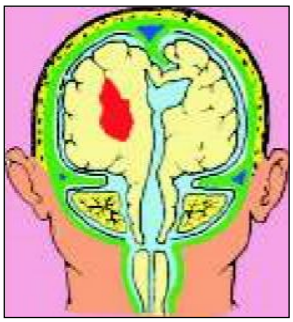
Intracerebral Hematoma :

- May result from coalescence of hemorrhages within the contusion
- Loss of cerebral autoregulation & post traumatic coagulopathy leading to delayed parenchymal hematomas
- When extends to ventricles known as intraventricular hemorrhage
- Mechanism of development is similar to cerebral contusions
- Development of focal neurological deficit, failure to improve, and neurological deterioration warrant emergency CT Scan brain and even surgical evacuation



CT Scan



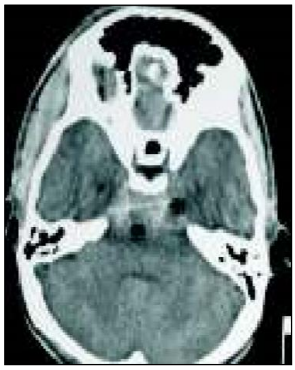


Intracerebral hematoma

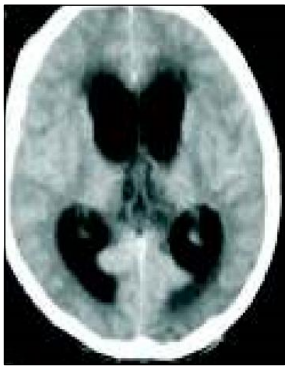
Treatment :

- Small lesion in stable patient can be conserved
- Mass effect & herniation warrant emergency evacuation of hematoma

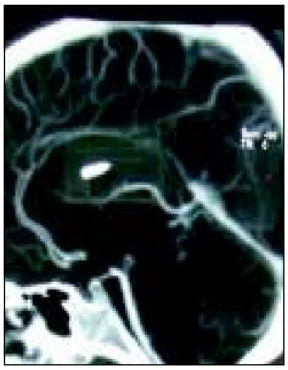
- Traumatic Subarachnoid Hemorrhage (SAH)
- Rupture of superficial vessels into subarachnoid space
- Results from relatively severe head injury due to prolonged and high angular acceleration
- Unlike aneurysmal SAH, Posttraumatic SAH is not limited to basal cisterns
- Though to a lesser extent, vasospasm has also been documented in post traumatic SAH



Suprasellar Clot



Hydrocephalus



Angiogram : normal

Penetrating Head Injuries :

Worldwide less common than blunt trauma except USA where it is reverse Either by bullet/ shrapnel or by knife / nails/ screwdrivers

Missiles:

high velocity	low velocity
> 320m/s	< 320m/s

(speed of sound in air is the dividing line)

Mechanism:

Direct crushing and laceration along the tract of missile- both low/high missiles

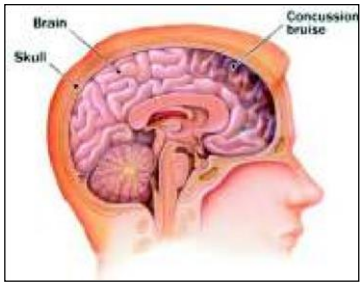
Cavitation – high velocity displaces tissue several centimeters from wound track

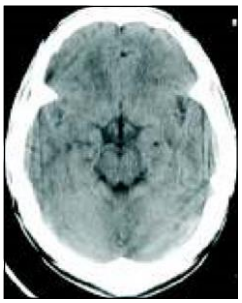
Shock waves causes structural and functional disruption well beyond course of missile

Concussion :

Mildest form of diffuse injury and is due to rotational acceleration of head in absence of significant contact injury

Transient loss of consciousness & rapid return to normal life



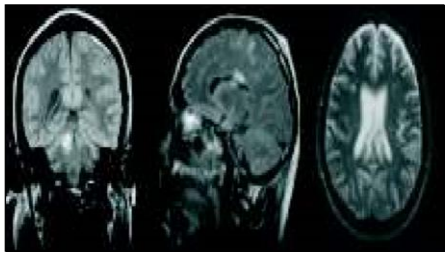


CT scan : normal

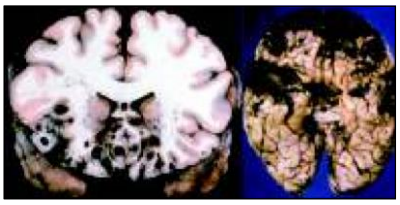
Diffuse Axonal Injury (DAI) : Common but less recognized type of injury

"Diffuse white matter degeneration"

- Results from severe angular or rotational Acceleration
- Shear and tensile forces acting together cause this injury
- Responsible for severe neurodeficit in the absence of gross hematoma, parenchymal contusion or laceration
- Histological findings include axonal swelling, disruption "retraction balls" punctate hemorrhages in the pons, midbrain & corpus callosum • Typically CT Scan shows normal brain!

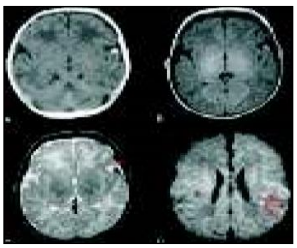


MRI : DAI

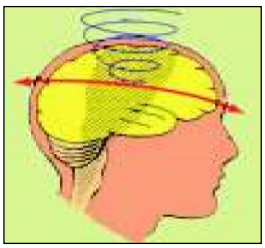


Pathological specimen of DAI

- Extent & location of DAI determines functional recovery
  - Higher grade of DAI is related to deeper coma & prolonged hospitalization



MRI



Mechanism

Spinning Top

Grade	Lesion
I	Axonal injury of parasagittal white matter
II	G-I with focal lesions in corpus callosum
III	G-II with focal lesions in cerebral peduncle

Traumatic brain edema : Mechanism

Focal contusion/ laceration → Raised ICP → Impaired cerebral blood flow → Neuronal hypoxia → Disturbed blood brain barrier → Vasogenic edema

axonal flow fails → glutamate accumulation → release of free radicals → Failure of ion pumps → Cytotoxic edema

Brain Edema

Herniation :

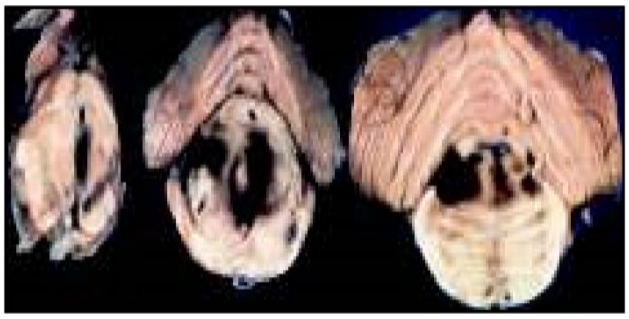
Any discrete, expanding intracranial mass lesion can lead to secondary displacement of brain through existing rigid openings in the dura & skull

Subfalcine

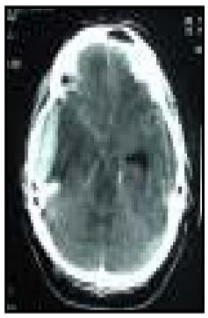
Uncal

Tonsillar

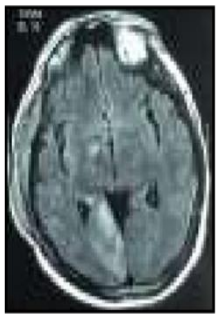
Site of Herniation	Structures involved	Clinical Symptoms / Signs
Subfalcine	Pericallosal arteries, cingulated gyrus	Leg Weakness
Lateral (Uncal, medial temporal)	Oculomotor Nerve, Posterior cerebral artery (PCA), Cerebral peduncle	Ptosis, ipsilateral mydriasis, contra lateral hemiparesis, and altered consciousness, Homonymous hemianopia
Posterior (tectal)	Superior colliculi	Bilateral ptosis, upward gaze paresis (Perinaud's syndrome)
Central (axial)	Perforators of Basilar Artery, midbrain, pons and medulla	Depressed consciousness, impaired eye movements, irregular breathing, hypertension, Bradycardia
Tonsillar (Cerebellar)	Medulla	Apnea



Durette's Hemorrhages



Uncal Herniation



Right PCA infarct

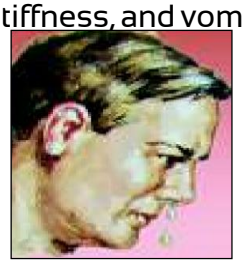
Subfalcine herniation : is usually asymptomatic • However extreme compression of pericallosal arteries can lead to contralateral leg weakness

- **Uncal / lateral / central herniation** : should be detected early and must be tackled urgently to prevent mortality
- **Tonsillar herniation** : is the phenomenon found in the posterior fossa • Early medullary compression resulting in apnea & death without any evidence of pupillary or oculomotor abnormalities are hallmark of it

CSF Leak

- Post traumatic CSF leak occurs in 2% of closed and 9% of penetrating head injures
- CSF leak appears soon or later in the clinical progress of patient
- Two types :
- CSF rhinorrhea: Leakage of CSF from nose
- CSF otorrhea: Leakage of CSF from ear
- CSF Rhinorrhea occurs through fractures at cribriform plate, roof of ethmoidal and/ or sphenoidal sinus
- Both conditions are cause of concern for Neurosurgeon
- Most of the leaks stops on its own or conservative treatment by the end of 1st wk
- Presenting complaints may be just watery discharge from nose/ear or critical meningitis(fever, neck stiffness, and vomiting)


CSF rhinorrhea


Rhinorrhea


Otorrhea


Temporal

Investigations :

bone fracture

Detecting CSF in the discharge -

- Profuse, persistent, clear nasal/ ear discharge
- Halo sign/Target sign: Clear fluid circles around a central blood stain when CSF mixed with blood is dropped on a filter paper
- Reservoir Sign: When patient is given correct position, collected CSF in the ethmoid & sphenoid sinus will drain out

- Glucose content: >30 mg/ ml is probably CSF as nasal secretions have low glucose content
- Immunoelectrophoresis: Detection of ß-2 transferrin, is characteristic of
- CSF
- Isotope cisternography: Radioactive tracers, using In-DTPA

Localizing site of CSF leak :

1) Plain X rays -

Though not a preferred investigation for detection of leak, presence of air fluid level in frontal, ethmoidal or sphenoidal sinuses indirectly indicates fracture site

2) CT Scan -

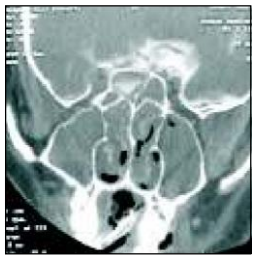
Investigation of choice

Thin, 1 to 1.5mm slices in coronal place can detect fracture line

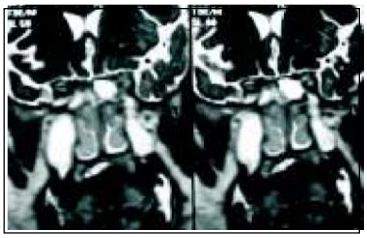
3) Contrast CT cisternography-

After intrathecal injection of water soluble contrast via lumbar puncture

Detects site of leak : presence of dye in sphenoid sinus



4) **MR Cisternography -**



Diagnostic sequences : T2 drive/ CISS

Detects exact site of CSF leak

Conservative treatment :

1) Positioning : Head elevation by 30° to 45°

Improves venous drainage & reduces ICP

2) Lumbar Drainage : Reduces ICP by diverting flow of CSF away from site of leak
3) Drugs- Acetazolamide reduces production of CSF
4) Antibiotics – No role in prophylaxis of Meningitis

Operative Treatment :

Indication 1)Persistent, profuse CSF leak beyond 1st wk

2) CSF leak due to penetrating injuries

3) Presence of meningitis

4) Presence of Intracranial aerocele

Extra cranial approach :

- Done endoscopically through the nose
- Preferred for sphenoidal & ethmoidal CSF leaks

Intracranial approach :

- May be extradural or intradural
- Preferred for cribriform, frontal sinus, or ethmoidal leaks occuring through dural tears not amenable to endoscopic repair
- Involves direct repair of dural breach hence much effective
- Bony defect can be tackled using methyl methyl acrylate or cyanoacrylate
- Otorrhoea usually responds to conservative treatment but may need surgical repair through mastoidectomy or subtemporal extradural approach
- **Post traumatic cranial neuropathy :**
- Coma may obscure all but III, VI & VII CNs damage
- Injury to orbital muscles may be difficult to differentiate from CNs injuries

Nerves most frequently injured :

- Olfactory facial & Vestibulocochlear CNs are most often damaged due to close head injury
- Optic, Oculomotor, Trochlear and Abducent are affected next
- Trigeminal and lower cranial nerves are rarely injured
- A suicidal injury usually affects optic nerves as path of gunshot will be directed anteriorly • The victim may survive
- Homicidal injuries involve lower cranial nerves, internal carotid artery and sympathetic trunk, and are usually fatal
- Blunt head injuries result in basilar fracture, affecting cranial nerves while sparing the brain

- Hyperextension neck injuries selectively damage VI & XII th CNs

Recovery :

Facial nerve usually recovers, followed by III, IV & VI th CNs

Olfactory & Optic show significant improvement in less than 30% of patients

VIII th nerve lesion is usually permanent

Olfactory Nerve :

- Usually neglected by medical students/house officers
- 10% anosmeas are trauma related

Optic nerve :

- ⅓ th of civilian optic nerve injuries are due to penetrating gunshot injuries
- Indirect optic nerve injury results from trauma to the ipsilateral outer eyebrow
- Associated with severe head injury & unconsciousness
- Restoration of vision is unlikely unless diagnosed early & treated promptly

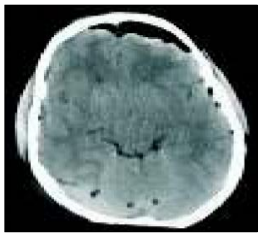
Symptoms :

Complete blindness of one eye or loss of direct (in same eye) & consensual pupillary reflex (in the opposite eye)

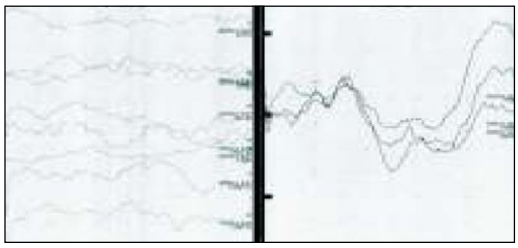
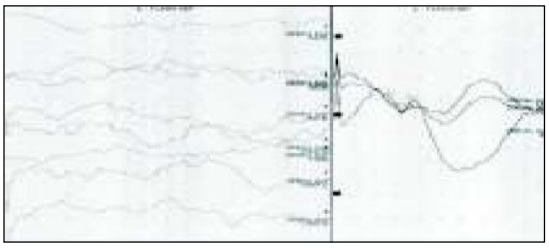
- Visual field defects have also been demonstrated

- Usual site of affection is optic canal






CT scan : soon after injury CT scan : 4 days after injury

VEP : Pre op VEP : Post op(after nasal endoscopic excision of bony strut compressing the optic nerve)

Investigation :

- CT Scan – detects the fracture at optic canal causing optic nerve compression
- VEP- electrophysiological test to detect anatomical integrity of optic pathway

• Treatment :

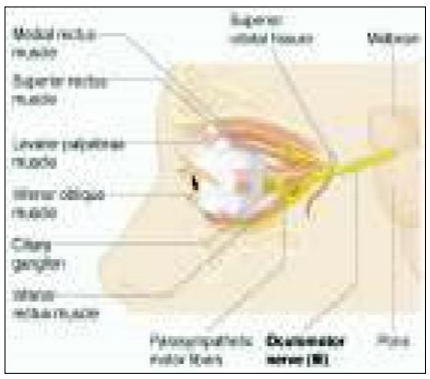
- High dose steroids have been found effective
- Surgical treatment :

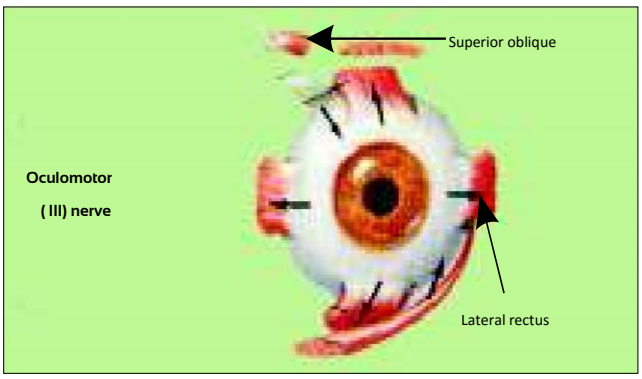
  Endoscopic medial optic nerve decompression minimal invasive procedure but very effective and can restore the vision

Oculomotor nerves – (III, IV & VI)

III rd Nerve : Trauma is one of the most common causes

Raised ICT causes uncal herniation





Dilated pupil & outward turned eye is indicative of third CN palsy

Differential diagnoses are orbital blow out fracture causing limited extraocular muscle contraction

Usually recovery of oculomotor nerve function occurs 6 wks after trauma

th Nerve : Rarely diagnosed on acute neurosurgical Services

A comatose patient who is awakening may closes one eye inadvertently to focus indicates IV th N palsy

Vertical diplopia is the main symptom

Examination shows affected eye to be slightly higher

VI th Nerve : Lateral rectus weakness is hallmark

Most of the time it is "false localizing sign"

Trigeminal nerve

Investigations : Diplopia Charting

CT scan / MRI scan to detect site of lesion, tentorial herniation, and orbital fracture

Trigeminal Nerve :

Branches of TN are frequently injured by facial laceration and fractures

Usual presentation is infraorbital numbness due to maxillary nerve injury (V2)

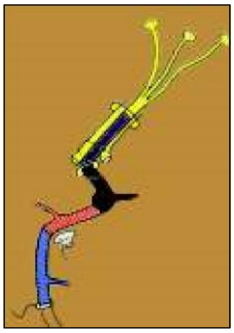
Gasserian ganglion/ Trigeminal trunk are rarely involved in closed head injuries

Concomitant VI & VII th weakness is found with basilar fractures

Facial Nerve :

- Due to long, tortuous course though the temporal bone, facial nerve is vulnerable to penetrating / blunt trauma to head
- Penetrating object affects horizontal part of VIIth N
- Self instrumentation of external auditory canal is commonest cause, second being accidental entry of foreign bodies
- A common industrial injury by penetration of middle ear is by welding torch
- Blunt trauma causes temporal bone fracture (transverse or longitudinal)
- Facial nerve is injured in Internal acoustic canal in 50% of cases of transverse fractures
- Longitudinal fractures are more common but do not cross internal acoustic canal thus sparing seventh CN
- Tearing of facial nerve from its root entry zone presents as early facial palsy while delayed palsy is usually secondary to edema, and carries a better prognosis

A - Longitudinal Fracture B - Transverse Fracture



Facial Nerve

Treatment :

- Paresis needs conservative treatment
- Simultaneous loss of VIIth & VIIIth function is indication for mastoidectomy & decompression of VIIth nerve
- As it is affected at multiple places, it is advisable to decompress it throughout its course, and particular attention should be given in the region of Geniculate ganglion

Physiotherapy :

- Important aspect of treatment of post traumatic facial palsy
- Electrical stimulation and massage to prevent facial muscular atrophy & contractures
- Facial exercises are best performed looking in the mirror

Vestibulocochlear Nerve :

- Temporal bone fractures result from direct, lateral blunt trauma to the skull in the parietal region
- Otic capsule is much thicker hence fractureline courses around it to involve all neighbouring formina, most commonly carotid canal and jugular fossa
- Longitudinal fractures constitute 70 to 90% of temporal bone fractures, which run from external auditory canal to foramen lacerum, and VIIIth CN injury is rare
- Transverse fractures (20%) damage both VII & VIIIth CNs, usually laceration of both

Post traumatic positional Vertigo : (also called Benign positional Vertigo)

- Brief attacks of Vertigo/ Nystagmus
- Precipitated by changing head position
- Attacks are of brief duration
- Dislodged calcium carbonate crystals from Macula of Utricle, get attached to Cupula of Posterior semicircular canal, causing vertigo
- Spontaneous remission by 3 months • Occurs usually within 2 yrs of head injury

Investigations :

- Audiometry - quantifies extent of neurological involvement
- CT Scan (HRCT) - detects site of fracture • BERA - confirms integrity of auditory pathways

Treatment :

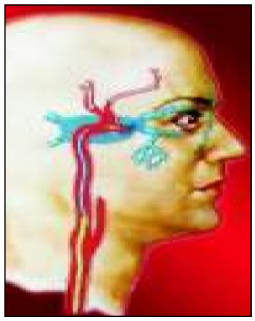
- No specific treatment
- The condition is self limiting
- Occasionally respond to steroids, and positional exercises

Traumatic Carotid Cavernous fistula(CCF) :

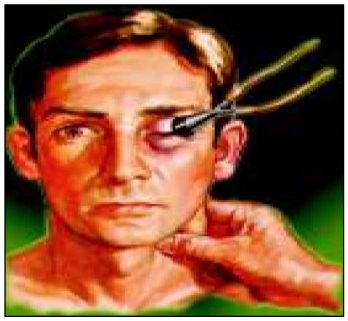
- Tear / perforation in internal carotid artery (ICA) within the cavernous sinus
- Rare consequence of Head injury
- 75 to 80% of CCFs are traumatic
- Penetrating as well as blunt trauma can cause CCF
- Cavernous part of ICA is immobile due to dural attachments at both ends hence prone to injury

Pathophysiology :

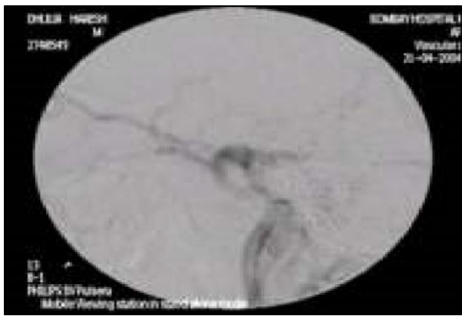
Eyes	Cranial nerves	Cerebral circulation
Ipsilateral & even	III, IV, and VI CN	Steal Phenomenon
Contralateral ptosis	Palsy	Cerebral ischemia
Orbital congestion		Cortical venous
Chemosis		hypertension
Corneal ulceration		Subarachnoid
Retinal ischemia		hemorrhage
		Vitreous hemorrhage
		Presenile cataract



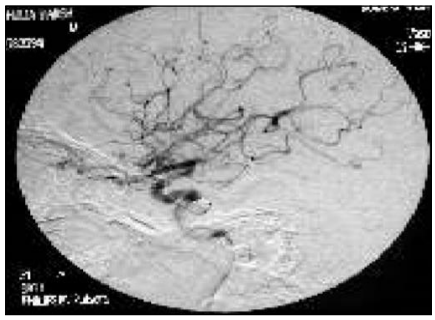
CCF



Bruit



Angiogram : Pre embolization



2 years post embolization

Classification

A	Direct
B	Internal Carotid Artery to Cavernous Sinus
C	External Carotid Artery to Cavernous Sinus
D	Internal & External Carotid Artery to Cavernous Sinus

Investigations :

- ✓ CT Scan :- Shows distended, dilated, tortuous superior ophthalmic vein
- ✓ MRI:- shows detail information about enlargement of cavernous sinus
- ✓ MRA & MRV:- Shows CCF (anatomical position)
- ✓ DSA:- Detects exact site of fistula

  Steal Phenomenon

  Dilated tortuous superior & inferior ophthalmic veins

Treatment : Indication : Progressive visual failure

Intolerable bruit

Progressive proptosis

Early filling of cortical veins

Subarachnoid hemorrhage

Repeated Transient ischemic attacks

Conservative : Intermittent manual compression- effective in type B, C & D

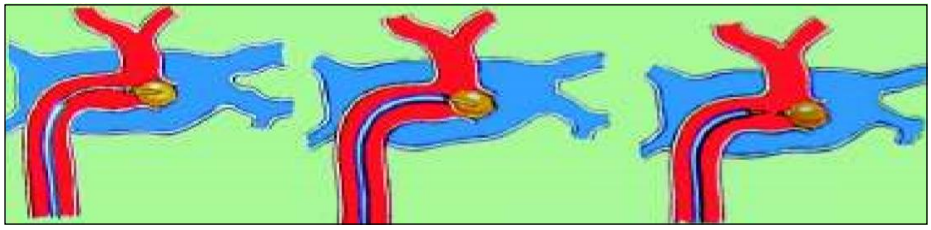
Surgical : Transvenous packing of cavernous sinus

Direct exposure & Ligation of fistula

Carotid artery ligation

Endovascular : Transarterial - Balloon occlusion - Universally accepted modality which has stood test of time

Transvenous - Coil embolization



Balloon occlusion of Type A CCF

Traumatic Arterial Dissection :

Dissection is a condition in which blood penetrates forcefully through a tear in the intima, separating and dissecting it from the media

Etiology : Blunt injury } both can cause Penetrating injury traumatic dissection

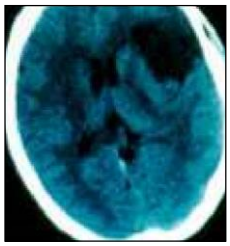
Mechanism of Injury :

Stretch / Traction / Rotation force

Direct penetrating injury

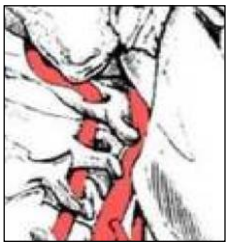
Internal Carotid Artery (ICA) Vertebral Artery (VA)

Intimal rupture Intimal rupture Occurs Occurs in the region maximally at C_1-2 of transverse process of C_2-3

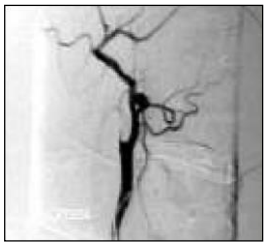


Left MCA infarct

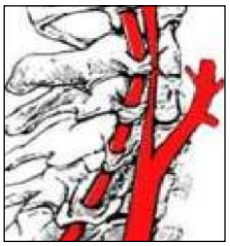
Flexion injury – Direct compression of ICA between angle of mandible and upper cervical vertebrae causes intimal rupture and / or transmural crush



Internal Carotid Artery

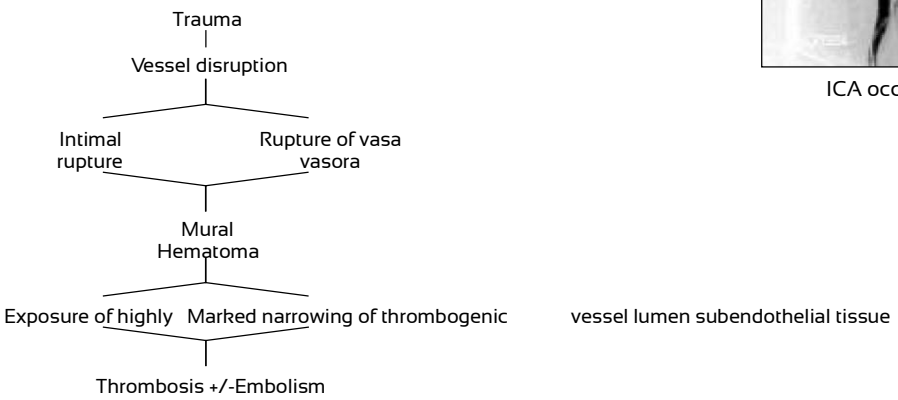


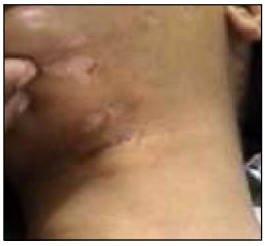
ICA occlusion



Vertebral Artery

Pathogenesis :





Site of ICA injury

Consequences :Complete occlusion / Double barrel lumen

Dissecting aneurysm (Subadventitial) leads to subarachnoid hemorrhage (SAH)

Symptoms : Asymptomatic neck pain
Ischemic stroke
SAH

CT scan : shows – infarct

MRI / MRA : Detects site of dissection, infarct (umbra), and penumbra

DSA : Dynamic investigation

Detects site of dissection, Occluded vessel

Treatment :

Anticoagulation :- Injection Heparin (100 unit / hrs for a wk) & later on switch over to Oral anticoagulants (maintain INR 2-2.5)

Surgical ligation:- Indication – progressive stroke despite anticoagulation

Balloon / coil occlusions – to prevent further embolization

- Stenting :- for short length dissection

Antiplatelet agents (Aspirin, Plavix etc.) are needed life long for stented patients

Post traumatic concussion syndrome : In 1835, Gama wrote, "fibres as delicate as those of which the organ of mind is composed are liable to break as a result of violence of head"

Usually follows mild head injury

Symptoms

Headache : Muscle contraction, migraine, cluster, occipital neuralgia, scalp laceration

Cranial nerve symptoms :

Dizziness, vertigo, tinnitus, blurred vision, diplopia, diminished taste/ smell

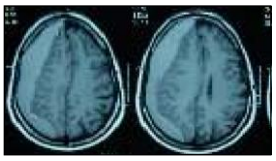
Psychological / Somatic :

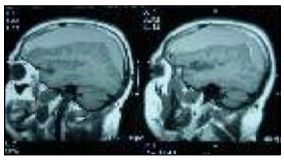
Irritability, Anxiety, Depression, Fatigue, Sleep disturbance, decreased libido / appetite

Cognitive impairment :

Memory dysfunction, impaired concentration / attention, slowing of reaction time

Rare sequelae :





Epidural / Subdural hematomas

Seizures

Transient amnesia

Tremors

Treatment :

Headache	Migraine
NSAID	Sibelium (Flunarazine) / Sumatriptan / Ergotamine
Antidepressants	Beta blockers
TENS	Ca channel blockers

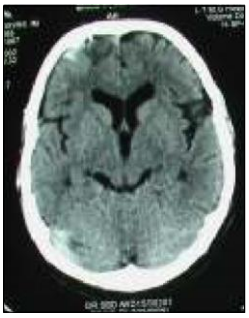
Valproate Carbamazepines

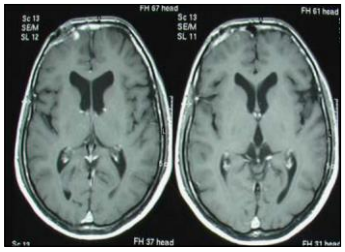
Post traumatic epilepsy :

- Within first year after head injury the incidence of seizures exceeds 12 times the population risk

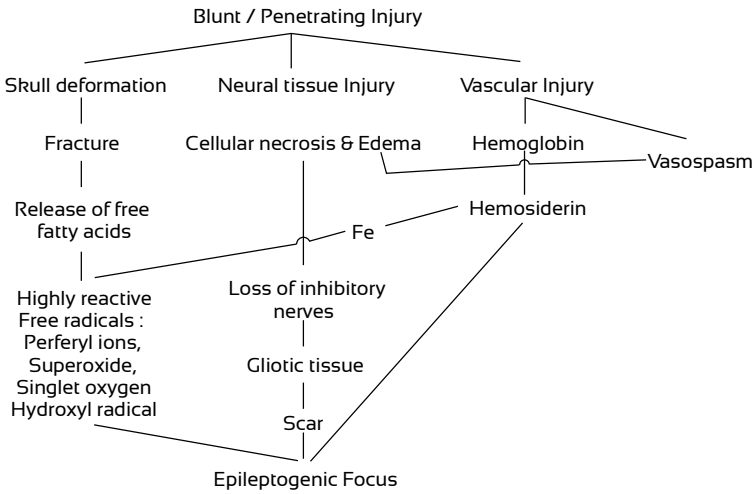
Injury	Incidence
Brain injury with intact dura	7 to 39%
Brain injury with breached dura	20 to 57%

- High risk of seizures is associated with missile wound penetrating the dura, central parietal area, early occurrence of seizures, intracerebral hematoma, depressed fracture, hemiplegia, cerebral infarction, and cortical laceration
- Though a seizure soon after trauma may be a nonspecific reaction, above causes should be ruled out
- But seizures occurring after 1wk increase chances of delayed seizures





Epileptogenesis :



Chronic subdural hematoma (CSDH) :

Develops after 3 weeks to several month of head injury

Either history of trivial trauma, no history of trauma

Elderly patients are at greater risk owing to cerebral atrophy causing widening of subdural space

Bridging veins are exposed and stretched by shrunken brain, and are liable to get snapped after jerky brain movements after trivial injury

Oozing of blood over surface of brain over a period of time

Neomembrane : these hematomas evoke peculiar reaction derived from dura

Two membranes :

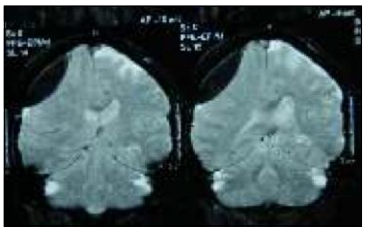
Outer : facing dura and outer surface of hematoma, is thick and hypervascular

Inner : facing brain and inner surface of hematoma, is and thin relatively avascular

Repeated microhemorrhages between the membranes increase hematoma size

Osmotic process between the membranes draws fluid and increase size of hematoma

- Factors responsible : Low ICP
  - Cerebral atrophy
  - Coagulopathies
  - Anticoagulant therapy
  - Chronic alcoholism
  - Liver cirrhosis



Gradient Echo

Symptoms :

Mimic brain stroke, dementia or low pressure hydrocephalus
Headache, altered sensorium, vomiting, weakness, visual symptoms, seizures, and dementia
Signs: hemiparesis, altered sensorium and papilloedema Investigations: CT Scan -
1st wk- hyperdense
2nd wk- isodense

(hence liable to be missed especially bilateral CSDH, which do not produce shift of midline structures)
3rd wk onwards-hypodense MRI : More authentic, especially Gradient echo sequences

(hence liable to be missed especially bilateral CSDH, which do not produce shift of midline structures)

MRI : More authentic, especially Gradient echo sequences

Treatment :

Medical : Rest, Osmotic diuretics

Surgical : Burr hole and evacuation

Twist drill and controlled drainage

Electrolyte disturbances:

Diabetes insipidus (DI) : Evacuation of CSDH

Common phenomenon after head injury and craniofacial injuries and basilar skull fractures

May be transient or permanent

Occurs due to injury to hypothalamopituitary axis → to reduction in ADH secretion

↓

Diuresis Main features is fluid loss from kidneys

Clinical manifestations :

Polyuria, polydypsia, hypernatremia (high serum sodium), high serum osmolality (>300 mosm/ kg) and low urinary specific gravity (<1004) May be triphasic response : DI → followed by SIADH → DI

Treatment:

Increase oral intake of water and hypotonic fluids

Desmoprressin nasal spray / injections or even tablets can be prescribed

Syndrome of inappropriate (excess) secretion of ADH (SIADH) :

Patients usually suffer in first or second week after injury

Continuous secretion of ADH despite hyponatremia (low serum sodium), hypoosmolar serum, hyperemia → fluid retention hyponatremia

Symptoms :

Anorexia, vomiting , nausea, irritability, and personality changes

High CVP

Treatment :

Water restriction to less than a liter per day

Cerebral salt wasting syndrome (CSWS):

Much common than SIADH, less frequently appreciated

Increase in ANP secretion from atria → natriuresis → fluid loss

Increase in natriuresis

Patient is usually much dehydrated, low CVP, low sodium

Treatment :

Fluid infusion

Sodium supplementation

Rapid correction can produce central Pontine myelinolysis

Factor	**SIADH**	**CSWS**
Plasma Volume	Increase	Decrease
Salt Balance	Variable	Negative
Dehydration	Absent	Present
Weight	Increase/No difference	Reduction
CVP	High / Normal	Low
Hematocrit	Decrease / Normal	Increase
Serum osmolality	Low	High / Normal
Urinary Sodium	High	Very high
Serum uric acid	Low	Normal

Head injury in pediatric patients:

" Child is not a small adult"

Mechanism of injury, types and mode of management are entirely different

Neurological assessment

Level of consciousness

Papillary size

Respiratory status

Systolic BP

CNS status

Open wounds

Fractures

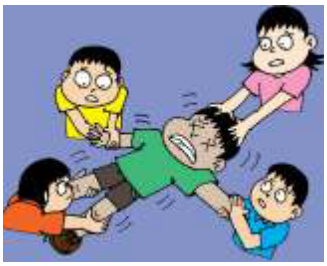
A Alert or conscious

B Responds to verbal commands

P Responds to pain

U Unresponsive

Evaluation of Head Injury Patient :



History:- Questions to be asked When did it happen? Precise time..... How did it happen? Mechanism of injury How long was the patient unconscious? Was there any recovery?

Any episode of seizures? Has he thrown out?

Any past medical disease?

Has he/she consumed alcohol / any medication

Initial examination :

Vital Signs :

Pulse rate : Tachycardia > 100/min

Bradycardia < 60/min

Blood pressure :

Hypertension- associated Bradycardia	Indicates raised ICT- increasing (expanding) blood clot
Unexplained Hypotension with Bradycardia	Suggest - cervical spine injury
Hypotension & Tachycardia	Abdominal or thoracic injury

Breathing :

Cheyne - Stokes breathing -

Alternate shallow & deep

Suggestive of diffuse by bilateral cortical dysfunction

Can increase PCO_2 causing cerebral vasodilatation and raised ICT

Biot breathing :

Irregularly irregular

In medullary or pontomedullary injuries

General examination :

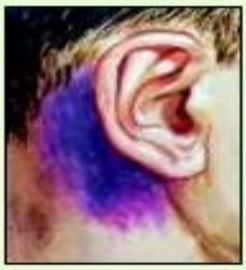
Scalp wounds : laceration, Bruises, Grazes, contusion, and avulsion

Skull fractures :

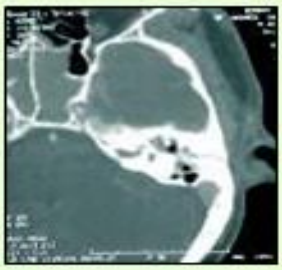
Ear/ Nose/ Throat : bleeding / Watery discharge (CSF rhinorrhea / otorrhea)

Skullbase fractures :

Panda sign
(Raccoon eyes)



Battles sign



Facial nerve injury

Level of consciousness : single most important

parameter Glasgow coma Scale

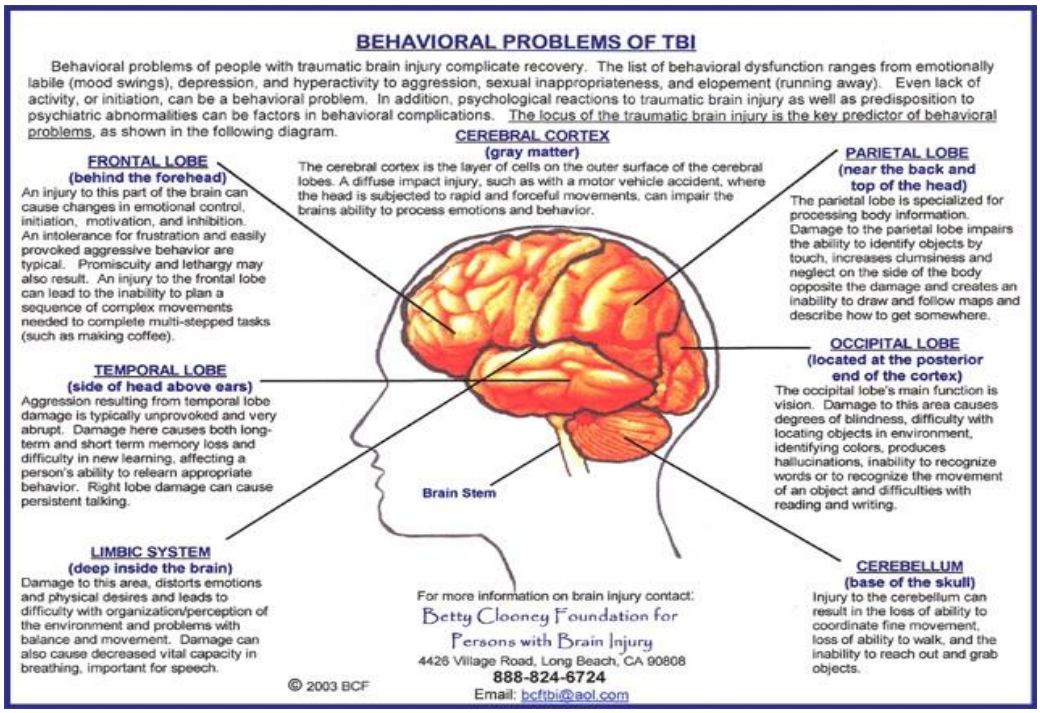
Sum of E+M+V - maximum 15 / 15 & minimum 3 / 15

Sum of E+M+V - maximum 15 / 15 & minimum 3 / 15

Condition	Self Awareness	Motor function	Experience of sufferings	Respiratory function	Prognosis
Persistent vegetative state	Absent	No purposeful movements, no visual tracking	No	Normal	1/3rd die,1/3rd disabled, few good recovery
Locked in syndrome	present	Quadriplegia, Pseudobulbar palsy, preserved eye movements	Yes	Normal	Quadriplegia, prolonged survival possible
Akinetic mutism	Present	Paucity of movement	Yes	Normal	Recovery unlikely
Brain death	Absent	Absent	No	Absent	None

Memory :

mmed ate	or seconds to m nutes	mmed ate reca , Te ephone No. for short per od			
Recent	Hours to days	our objects to remember			
Remote	Over years	( ) d	marr	age ann	versary



Eyes :

Pupils: - Hutchinson's pupil : unilateral, non reactive pupillary dilatation

Direct: - constriction of the pupil on which light is thrown

Consensual:- constriction of opposite side, even when no light is projected on that eye

Marcus Gunn Pupil - (efferent pupillary defect) Paradoxical pupillary dilatation observed as the light is moved from the normal to the abnormal eye

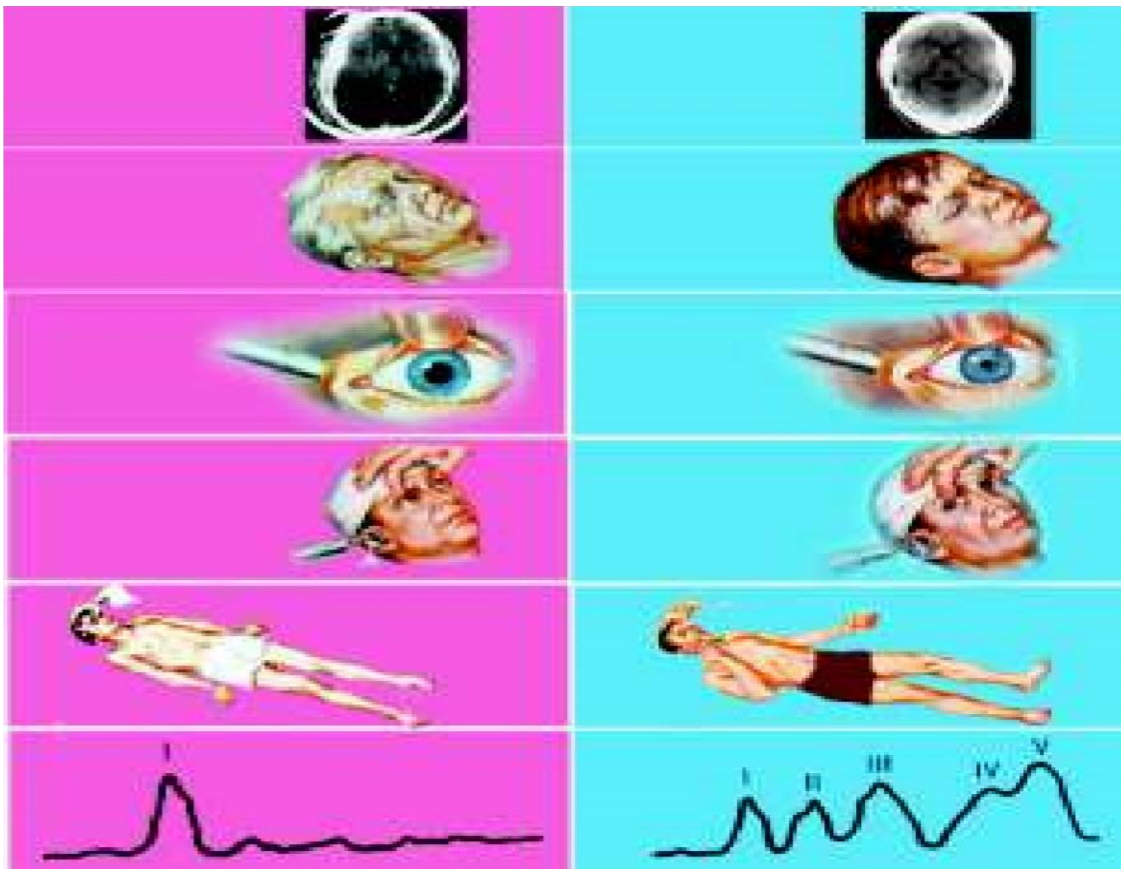
Horner's syndrome:- interruption of sympathetic supply

Miosis:- Pupillary constriction } Associated with :

Ptosis:- Drooping of lid Brachial plexus injury

Anhidrosis:- over face Spinal cord injury

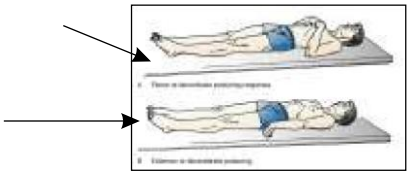
Enophthalmos:



Decorticate Posture : Flexion response in upper limbs, extension in lower limbs

Suggests severe Supratentorial damage (above midbrain)

Decerebrate posture : Extension response in all four limbs



Deep tendon reflexes : Exaggerated after recovery from initial Neurogenic shock

Babinski response : Uni/bilateral upgoing plantars

Investigations :

Plain X-rays : Primary & cheapest mode of imaging, but unnecessary with the advent of CT (hence useful only if CT is not available)

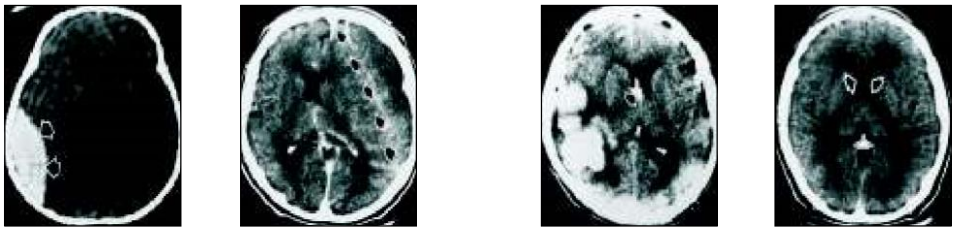
- Detects gross & hair line fractures
- Air fluid level in sinuses indicates probable site of CSF leak
- Associated facial, spinal injuries can be detected

CT Scan : Investigations of choice for screening of red alerts

- Reliable demonstration of midline shift, acute hemorrhage, intracranial mass lesion and fractures
- Rapid imaging
- Can be done even on intubated & ventilator supported patients
- Diffuse axonal injuries cannot be detected
- Not preferred for posterior fossa imaging owing to bony artifacts

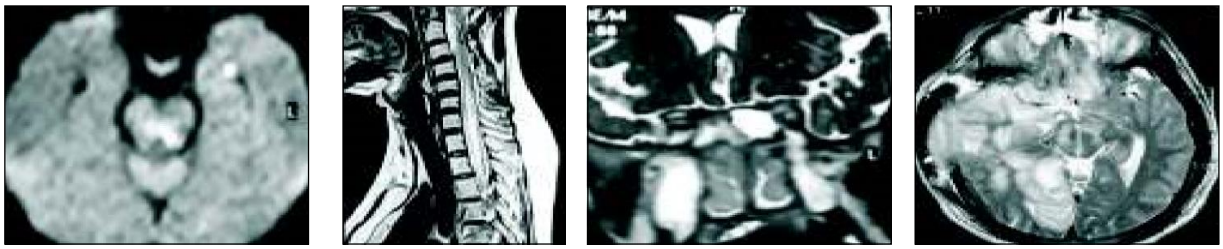
Indications in Head injured patients :

- GCS 13,14 or 15 and Abnormal mental status
- GCS 15 but has nausea, vomiting, headache, and skull fracture
- GCS 12,14,15 with LOC
- GCS < 13, all patients
- Focal deficit / basilar fracture / scalp injury / older than 60yrs
- Clinical deterioration



MRI : Though superior modality, it is rarely used in acute brain injuries

- Due to long imaging time & inability to do it on ventilator supported patients
- Vital for Nonhemorrhagic contusion, diffuse axonal injury, posterior fossa mass, and brainstem lesions



Brainstem Contusion | Posttraumatic syrinx | Posttraumatic CSF leak | Brain Swelling

For diagnosis needs mind and eye not just MRI!

Hematoma	T1w	T2w
Hyperacute (w th n few hours)	Hypo	Hyper (Oxy Hb)
Acute	s › to hype	Hypo (Deoxy Hb)
Sub acute	Hyper	Hypo(ntrace
Chron c	Hyper	H yper (Re ? 3C ys s)

T2 drive / CISS sequences for CSF rhinorrhea, for reliable detection of site of CSF leak

Angiography : Not a routine test in trauma patients

- Gold standard to detect vascular lesions
- Detects :- Traumatic arterial dissection, carotid cavernous fistula (CCF),

arterial spasm, arterial thrombosis, venous sinus thrombosis and False or peudoaneurysms

Management of head injuries : Head injury is often associated with multisystemic injuries hence needs interaction with multiple specialities particularly Orthopaedic, ENT, Ophthalmological, Faciomaxillary, Plastic and even General Surgeons apart from Intensivists, Nutritionists, and Physiotherapists

- ✓ Prehospital care (At the spot)
- ✓ Transfer
- ✓ Hospital care

Management within first minute :



- Airway
- Beware broken neck- Cervical collar
- Stop obvious bleeding
- Occlude sucking wounds in chest
- Patient retrieval in the "Golden Hour"
- Equipped Ambulance
- First aid by specialized Paramedics
- Resuscitation at accident site

A- Airway
B- Breathing
C- Circulation
D- Drugs
E- Equipped ambulance
F- Fluids
G - Gas analysis
H - Hospital transfer
I - Injury assessment

Head injury needs not only intensive but also comprehensive care units (ICCU)

Criteria for admission :

- ✓ Confusion, Depression of
- ✓ Consciousness
- ✓ Skull fracture, clinical or
- ✓ radiological
- ✓ Signs of neurological dysfunction
- ✓ Severe headache or vomiting


- ✓ Alcoholic
- ✓ Convulsions
- ✓ Bleeding from Ear, Nose and Throat
- ✓ Associated medical Conditions :-
- ✓ Diabetes mellitus
- ✓ Hemophilia
- ✓ Anticoagulant therapy
- ✓ No scope of observation at home
- ✓ Pediatric head injury / elderly

Air Ambulance

Management in First Hour :

- Apart from care within first min, and
- Detect multiple injuries
- Internal bleeding
- Baseline imaging
- Fluids : correct hypotension ; hypoxia
- Baseline blood investigations :
- Hemogram, Blood group / Cross match.
- Coagulation profile, concise biochemical profile
- SMA 12 + 2 & Serum Electrolytes
- Urgent evacuation of intracranial space occupying hematoma
- Scalp laceration:- Cleaning
- Inspection of fracture
- Suturing

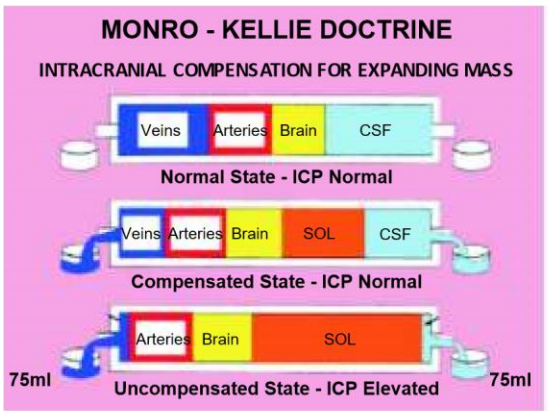
- Anticonvulsants: - Given in severe head injury & unconscious patient as seizures aggravate hypoxia, hypotension & brain injury, and poses problems in management of such patients
- ICP monitoring when available or indicated
- Brain protectives:

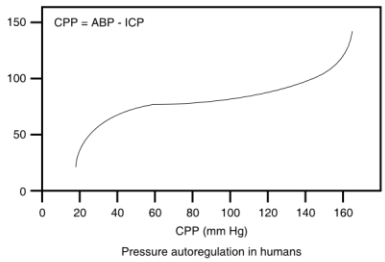
Piracetam, Citicholine, Mannitol, Antioxidants (Vitamin A, C, & E)

Intracranial pressure (ICP) : Intracranial cavity primarily contains brain, blood and CSF

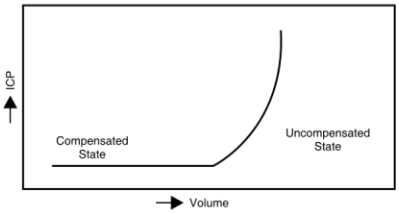
Monro-Kellie doctrine -

The doctrine states that total intracranial volume will remain essentially constant, and if any one of the intracranial components i.e. brain parenchyma, blood or CSF is increased or if another process occupies space, one of the primary components





Pressure autoregulation in humans



Indication of ICP Monitoring :

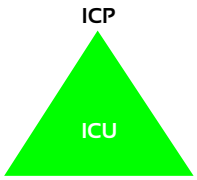
- Severe head injury patients

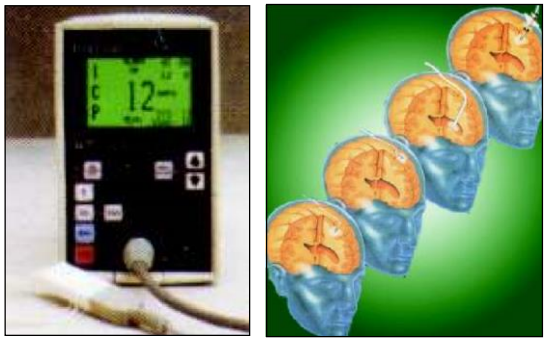
- Patients with good coma scale, with CT scan showing multiple small hematoma not meriting surgery
- Patients with good coma scale with single large intracranial hematoma
- Diffuse brain swelling
- Post operative ICP monitoring following evacuation of hematoma

Which patients are at high risk of raised ICP?

- Elderly patients > 40 years
- Systolic BP < 90mmHg
- Unilateral / Bilateral extensor posturing (decerebrate)

Cerebral Perfusion pressure (CPP)





ICP monitor

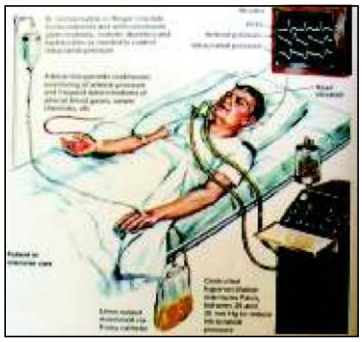
Various Instruments used for ICP measurement

- ✓ Richmond subarachnoid screw
- ✓ Leed's bolt
- ✓ Intra ventricular catheter

Duration: Usually 48 to 72 hrs, may be retained longer but beware of risk of infection

Goal of therapy : Keep ICP < 20mmHg Keep CPP > 60mm Hg

ICP management : Keep neck straight



- ✓ Avoid hypotension (Systolic BP < 90 mmHg)
- ✓ Use vasopressors if needed
- ✓ Control hypertension
- ✓ Avoid hypoxia (Po2 < 60mm Hg)
- ✓ Ventilate to normocarbia (Pco2 35-40)

- ✓ Light sedation

Specific measures :

Heavy sedation / paralysis

Drain 3-5 ml CSF per hour

Mannitol / Hyper tonic saline : increases serum osmolality and draws fluid from interstitium, hence reduces cerebral edema

Furosemide : causes diuresis and reduces extra cellular fluid and decreases edema

Hyperventilate to reduce PCO_2 30-35 mmHg

CT scan : To rule out expanding hematoma

Second Tier :

High dose barbiturates : Depress neuronal activity associated with reduction in cerebral metabolic rate • they alter cerebrovascular tone and inhibit free radicalmediated lipid peroxidation

Hyperventilate to reduce Pco2 to 25- 30mmHg

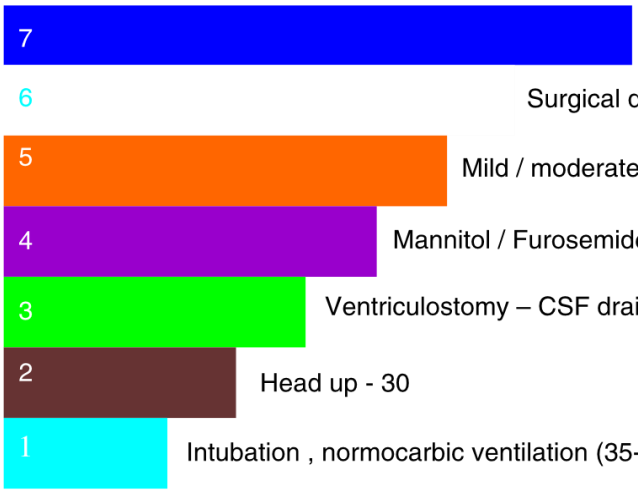
Hypothermia reduces Blood brain barrier dysfuction, cerebral edema and inflammation

Decompressive Craniectomy (removal of skull bone)

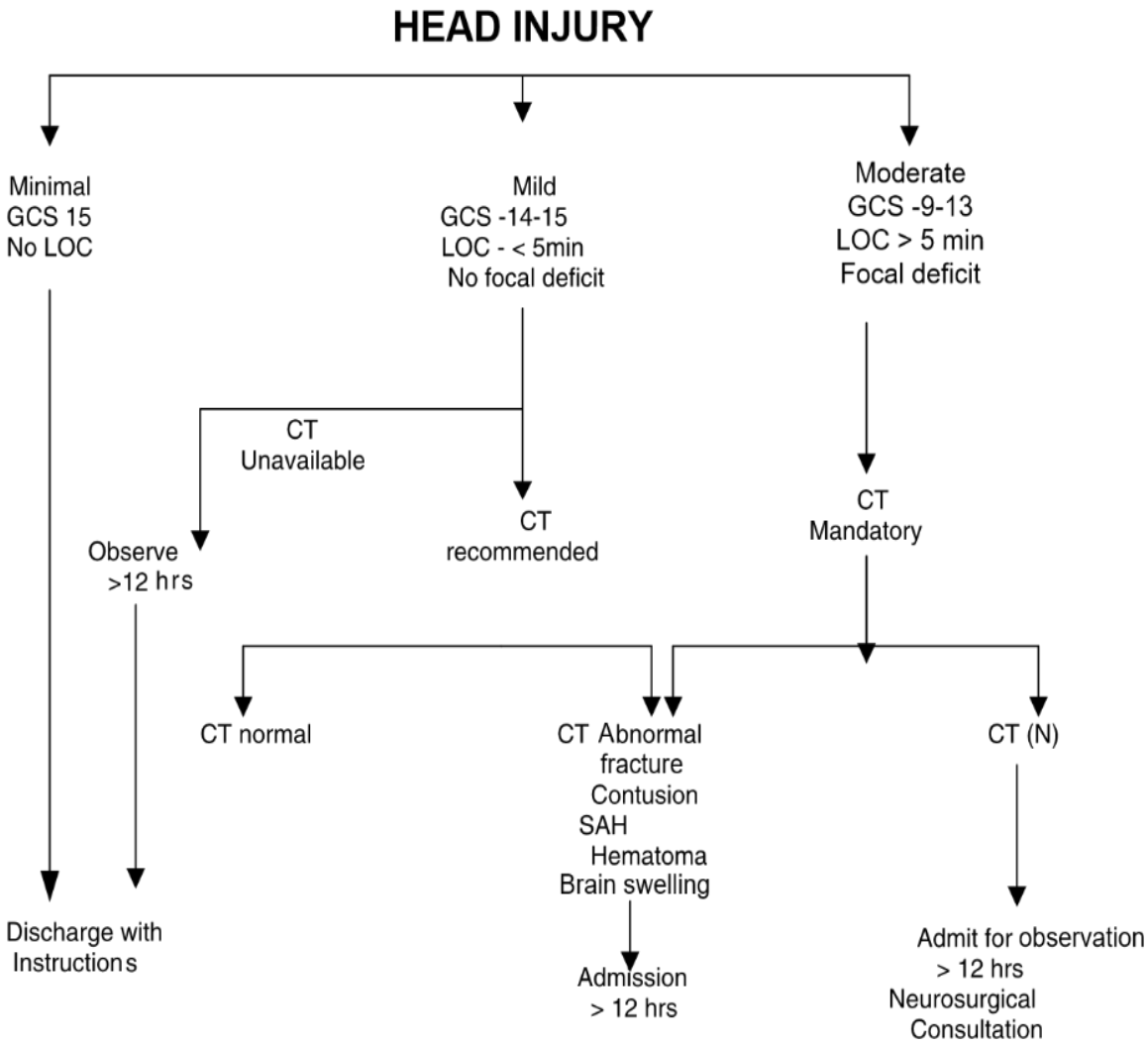
Hypertensive therapy

Leed's Bolt

Staircase ICP Control :



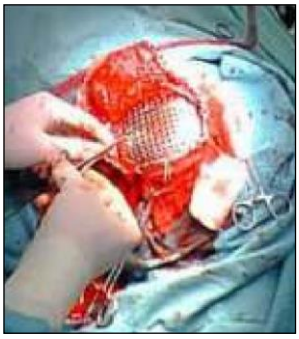
Whenever there is sudden increase in ICP during the course of



Prevent chest complications

Prevent bed sores

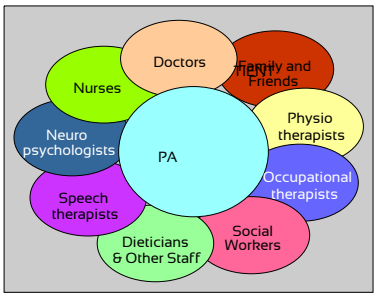
- ✓ Infection control
- ✓ Subacute epi / subdural hematoma
- ✓ Attend to limb fractures
- ✓ Physiotherapy and rehabilitation



Cranioplasty

Management in first month :

- ✓ Nursing Care
- ✓ Chronic Hematoma
- ✓ Hydrocephalus : Tend to be overlooked by after acute phase is over
- ✓ Physiotherapy and rehabilitation
- ✓ Rehabilitation
- ✓ Cranioplasty



Rehabilitation :

Management of Traumatic Brain Injury (TBI) patient is a multidisciplinary team approach

All the team members must have a forward looking approach during the immediate acute stage management to prevent subsequent

secondary complications and disabilities

Though rehabilitation starts from the time of primary head injury, active rehabilitation starts

once patient becomes free of early critical period and other related problems

Role of physiotherapy :

Prevent respiratory complications : Avoid further hypoxic brain damage

Correct positioning and handling of the patient in bed : To prevent deep vein thrombosis (DVT) and maintain neuromusculoskeletal integrity

Maintain the joint ranges and neuromuscular soft tissue proper lengths : hypertonia, spasticity, dystonia, decerebrate responses and ataxia should be tackled

Environmental and coma stimulation techniques are beneficial for arousal of the comatose patient which is achieved by administering different tactokinesthetic inputs from the body and the environment, encouraging visual scanning of the family members, team members, supporting surface and the environment around him **Splints :** Are advocated to prevent musculoskeletal deformities



Speech therapy :

Vital aspect of rehabilitation soon after improvement from initial critical phase

Dysarthric patients need lessons for proper pronounciation

Aphasic patients should be thoroughly introduced to the language and surrounding

Occupational rehabilitation:

Last part of the treatment which may take years together

Morbid patients with good mental status can be halped by sitting jobs like telephone booth or call centres, and computer centres **Prevention :**

The best treatment for head injury is prevention!

Prevention is always better than cure

Use of seat belts & encouraging others to do so

- Supporting legislation requiring use of seat belt & protective headgear
- Legislation aimed at eliminating drunk drivers
- Speed limit - 55km ph
- Protective equipment for sports participants
- Use of Helmet
- Arrangement of air bags inside the cars and driver cabin
- Construct broad multilane highways
- Warning signs and Sign boards at potentially hazardous sites viz. acute turn, blind curves, 'T' juctions, and slopes

Sit belt

Helmet

- Strong Police control and low enforcement for issuing driving licence and vehicle registration
- Vehicles must have proper head
- lamps, tail lights and even fog lamps

Multilane Highway

- Strong Police control and low enforcement for issuing driving licence and vehicle registration
- Vehicles must have proper head lamps, tail lights and even fog lamps

Air Bags

Sign Board

Conclusions :

Head injury is one of the commonest cause of morbidity

It is preventable cause of death

Proper pre hospital care saves golden hours and improves out come of trauma victims

Recent advents in Neuroradioimaging has dramatically improved out come

ICU care should be not just critical but also comprehensive

Physiotherapy and rehabilitation are vital part of management of trauma patients

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