HEAD INJURIES

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Pathophysiology Local effects on brain tissue Systemic effects of TBI • Management protocols Patient triage and classification • mechanism Types of head injuries

Triage: the following criteria suggest severe injury and urgency in care

Revised Trauma Score ¹	Injury Pattern	Injury Mechanism
Glasgow Coma Score <13	Penetrating injury to head, neck chest, abdomen or groin	Fall >6 metres (c. 20 feet)
Systolic Blood Pressure (sBP) <90 mmHg	Two or more proximal long bone fractures	Ejection of patient from a vehicle
Respiratory Rate (RR) <12 or >29 bpm	Flail chest	Roll-over of vehicle
	Burn >15% TBSA* or facial, airway or chest involvement	Death of occupant in same car
	Victim = pedestrian or motorcyclist	Rearward displacement of front axle
	History of ingestion of alcohol or drugs	Passenger compartment intrusion

- The Primary Survey and Resuscitation
- proceed in parallel
- A Clear the Airway. Use chin-lift or jaw-thrust. Immobilize the cervical spine with collars, bags and tape until cleared.
- B Check Ventilation. Administer oxygen at 15 litres per minute with tight-fitting mask with reservoir or use bag, valve and mask.
- C Check for pulses, skin perfusion and consciousness. Identify obvious sources of blood loss;
- D Assess the level of consciousness with A.V.P.U.:
- A (alert); V (responds to verbal communication);
- P (responds only to pain); U (unconscious);
- E Expose and examine the patient thoroughly.

Search for Life-Threatening Injuries and Treat Them Urgently

Airway	Breathing	Circulation	Disability	Exposure
Is the airway compromised? Consider oro-pharyngeal or naso-pharyngeal airway. If ineffective, intubate with endotracheal or naso-tracheal technique.	Ventilate by bag and mask or tube, with oxygen. Perform a needle thoracostomy (2nd L. ICS- MCL)* for a tension pneumothorax. Seal open pneumothorax. Identify flail chest. Insert chest drain	Arrest obvious bleeding. Pre- empt shock with 2 large bore i.v. cannulae. Infuse 2000 mls (20ml/kg in children) of crystalloid before transfusion of warmed blood (O-neg., type- specific or fully	bleeding. Pre- empt shock with 2 large bore i.v. cannulae.consciousness with AVPU (see D. above)Infuse 2000 mls (20ml/kg in children) of crystalloid before transfusion of warmed blood (O-neg., type-Look at the pupils.Can the patient move all 4 limbs?	Undress completely. Avoid hypothermia. Attach ECG monitor. Insert NG tube and urinary catheter. Beware of contra- indications!
If unable, consider crico- thyroidotomy.	thorax.	cross-matched). Drain any significant haemo- pericardium. Use cutdown or intraosseous lines if necessary.	nerve disruption or upper motor neurone/intra- cranial injury (e.g. asymmetry of movement or up going plantar – Babinski or thumb – Hoffman -reflexes).	Blood specimens: plasma chemistry, arterial blood gases, FBC, and cross-match. Get a urine sample (for urinalysis and toxicology etc).

The Secondary Survey

- Recheck A, B, C and D. Proceed to next stage if the patient is stable and analgesia has been effective. The secondary survey is a headto-toe examination of the patient's body.
- A.M.P.L.E. a simple mnemonic for key
- information
- A: allergies (e.g. penicillin or aspirin)
- M: medication (e.g. a beta-blocker or warfarin)
- P: previous medical history (e.g. previous surgery or anaesthetic mishap)
- L: last mealtime (i.e. drink versus major meal)
- E: events surrounding the incident (e.g. fell 5 metres with immediate loss of consciousness)

Examine each body region meticulously

Head	Face		Neck		Chest
Assess for wounds and fractures. Examine the eyes, and check for basal skull fracture.	mandib and ear Exclude	e mid-facial e or airway	Assume initially that a fracture – dislocation exists. Use collar – tape – sandbag neck immobilisation and palpate the spine. Wounds deeper than the platysma need formal surgical exploration.		Look, listen and feel: inspect the chest X-ray. Exclude lung and cardiac contusion, aortic rupture, diaphragmatic hernia, tracheal, bronchial or oesophageal injury. Look for haemo- pneumothorax.
Neurological		Abdomen		Orthopaedic	
Estimate GCS* repeatedly (e.g. every 15 minutes). Perform a full CNS examination, in the co- operative patient.		Peritoneal and retro-peritoneal injuries may present with normal signs.		Check for limb or spinal malalignment, crepitus, and tenderness. Examine the back.	
Evidence of paralysis or sensory deficit requires full spinal immobilisation. Falling GCS levels require urgent CT scan and neurosurgical consultation.		Rectal and perineal examination is essential. CT, ultrasonsography or occasionally diagnostic peritoneal lavage may help in excluding abdominal injury.		Assess neuro-vascular and tendon function. Logroll the patient to assess thoraco-lumbar injury. Search for minor fractures, dislocations, glass or other foreign bodies.	

INITIAL MANAGEMENT OF SEVERE TRAUMA

• Most widely accepted system is that recommended in the American College of Surgeons' Advanced Trauma Life Support (ATLS) course. The key elements of this approach are the primary survey/resuscitation phase, the secondary survey and the rapid implementation of definitive treatment, which may involve early surgery. The objective is to detect life-threatening conditions as quickly as possible, to stabilise the patient and to start definitive treatment as early as possible, in a prioritised fashion.

Intensive management of traumatic brain injury (roles and objectives)

• The primary focus of intensive management is directed towards patients suffering severe head injury (GCS of 8)following initial resuscitation and to prevent those suffering moderate injuries from deteriorating into unresponsive coma

It is universally agreed that a population of neurons is irreversibly damaged at initial impact but neuronal demise continues unchecked until brain resuscitation is initiated

It is also agreed that primary brain injury initiates a cascade of metabolic derangement affecting the cerebral vascular system, glial supporting cells, and neurons, which leaves brain vulnerable to secondary injury



.Brain stem dysf..ventilation prob.. O2 delivery prob.



schemia

.Raised ICP .Vasoconstriction .SOL

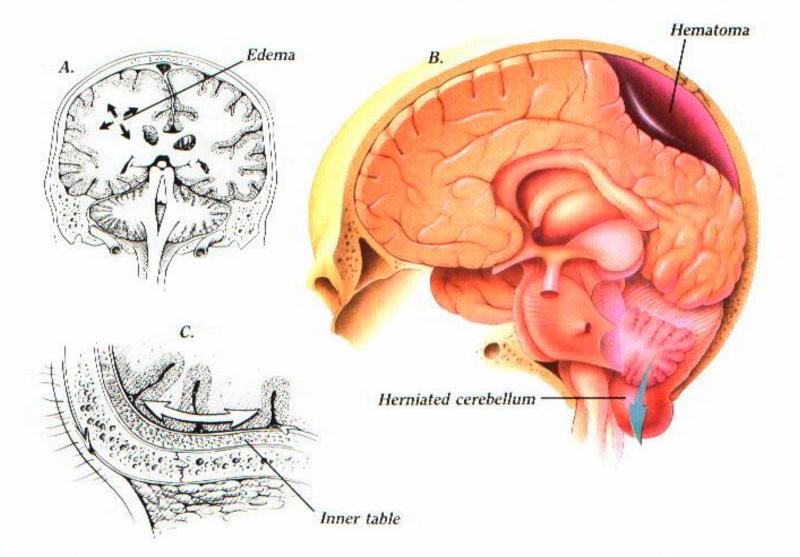
acidosis

acidosis

Traumatic brain (Secondary injury) . Ion pumps dysfunction .deranged autoregulation .oedema(vsogenic,c.toxic)

Herniation syndromes





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Systemic effect of T.B.I **Hypothalamic Cholinergic Adrenal Brain stem** dysfunction hyperactivity dysfunction dysfunction .bradyrrhythmia. .MI .DI .apnoea, hypotension .hypoventilation .neurogenic .SIADH pulmonary .Bradycardia .CSWS .hypersecretion .hypotention edema .hypertention, of ACTH,GH prolactin .decrease COP .arrhythmias

increased secretion of catecholamines and enkephaline, cytokines leading to hypermetabolism, hyperglycemia hypercatabolism and organ dysfunction (lung, liver, heart, gut) through lactic acidosis

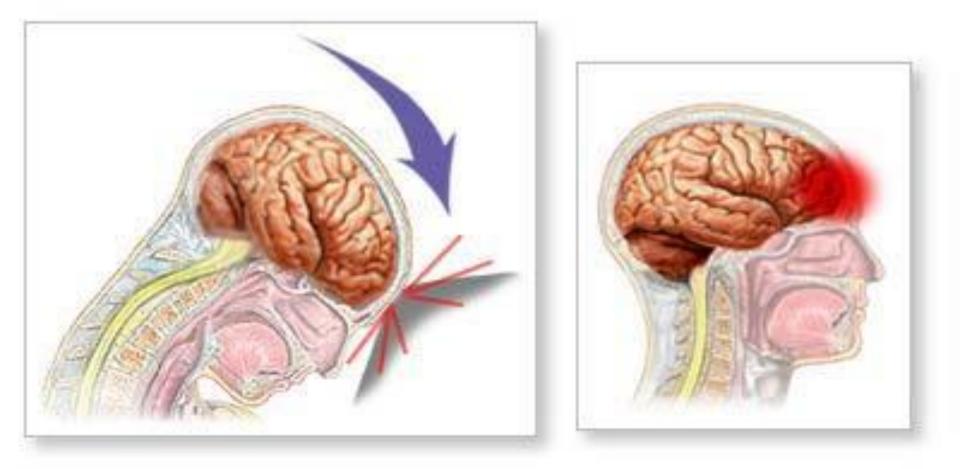
MECHANISMS OF INJURY

inertial or contact mechanisms

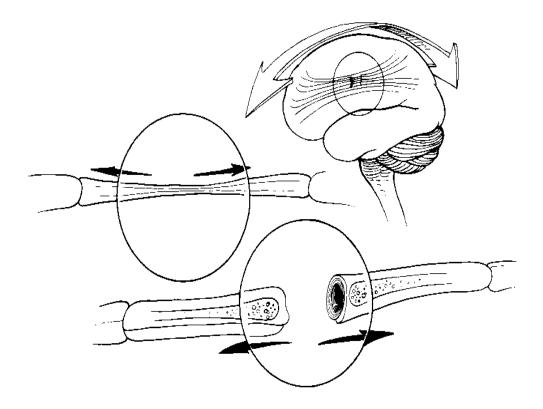
 Inertial injuries are commonly called "acceleration" or "deceleration" injuries

 Contact injuries are commonly called coup & contercoup injuries.

A concussion is a violent jarring or shaking that results in a disturbance of brain function

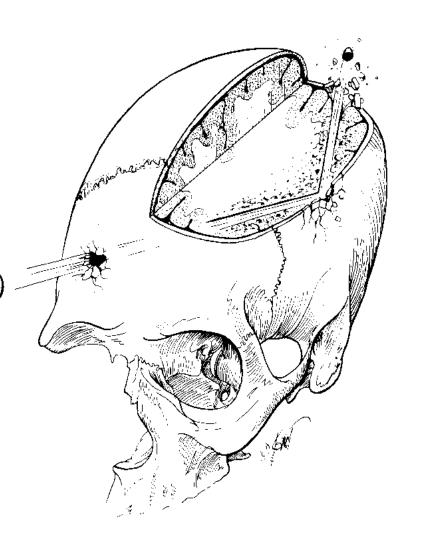




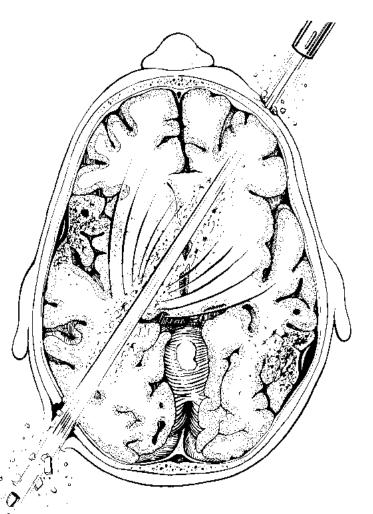


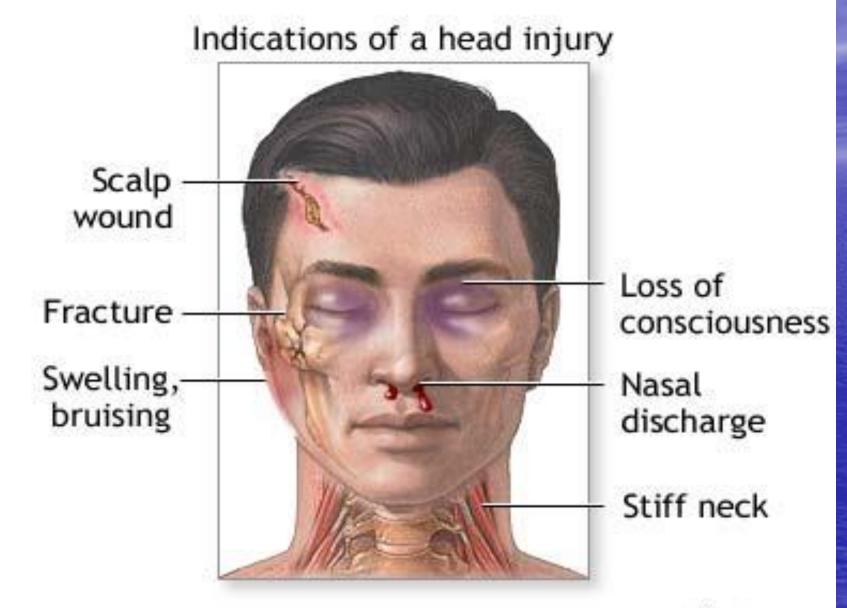
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Figure 1-16. Axonal shearing may occur in acceleration as well as deceleration injuries. The nerve fiber may be stretched or completely severed, producing the manifestations of diffuse head injury.



Traumatic Damage and Mechanisms of Repair 3













Initial management of acute closed head injury

at accident site

emergency room

in the ICU

Triage, resuscitation of ABCD (35% incidence of hypoxia (Apnea > 10 min carries high mortality)

Controlled ventilation to maintain paco2 30-35mmhg GCS less than 8 needs intubation and MVA

Crystaloid solution infusion and treatment of hypotesion (15% incidence of hypotension) presence of one episode of shock > 3 min(mostly hypovolemic) increases mortality by up to 50%

minimum of syst bp of 100mmhg to maintain adequate cerebral perfusion Systemic approach

GCS and B S reflexes

Management protocols

Immediate intubation and CT brain *l*ateral cervical xray, Cxr. us abd, pelvic xr. DPL for all severe injured

Surgery if CT shows a midline shift(Manitol if FND before CT)

Ur cath, NG , laboratory tests

.cardio-respir. Parameters monitoring .Controlled ventilation .PEEP .hyperventilation .Tracheostomy .Chest toilet , physiotherapy .Neuromuscular paralysis .sedation .B gasses ,electrolytes monitoring .Antiepileptics .Raised intracranial pressure treatment

GCS	6	5	4	3	2	1
Best motor response	Obey command	Localize the pain	Withdrawal to pain	Flexion to pain	Extention to pain	none
Verbal response		oriented	confused	Inapprop riate word	Incompre hensible sound	none
Eye opening			Spontaneo us	To verbal	To pain	none

Low risk group

Asymptomatic Head ache Dizziness Scalp hematoma Scalp laceration Absence of moderate or high risk criteria

Moderate risk group

History of change of consciousness History of progressive headache Alcohol or drug intoxication Unreliable or inadequate history Age less than 2 years Post traumatic seizure or vomiting or amnesia Multiple trauma Serious facial injury Signs of basal fracture Possible skull penetration Depressed fracture Child abuse

High risk group

Depressed level of consciousnesss not due to drugs or other causes Focal signs Decreasing LOC Penetrating skull injury Palpable depressed fracture

Observation alone

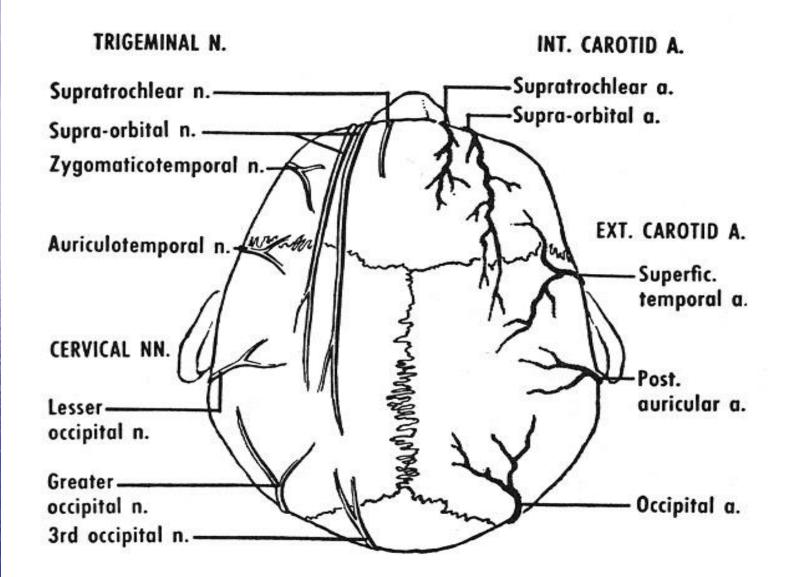
Observation for high risk signs Consider CT Neurosurgical consultation Consider urgent CT & Neurosurgical consultation& examination



1.Injuries severe enough to damage scalp may also damage the underlying skull2.Acomplete examination of the whole patient is mandatory

layers of scalp are

- SSkinCC.TAAponeurotic (Galia)LAreolar tissue
- Periosteum





Lacerations
Avulsions
Physical injuries
Chemical injuries

Scalp Lacerations

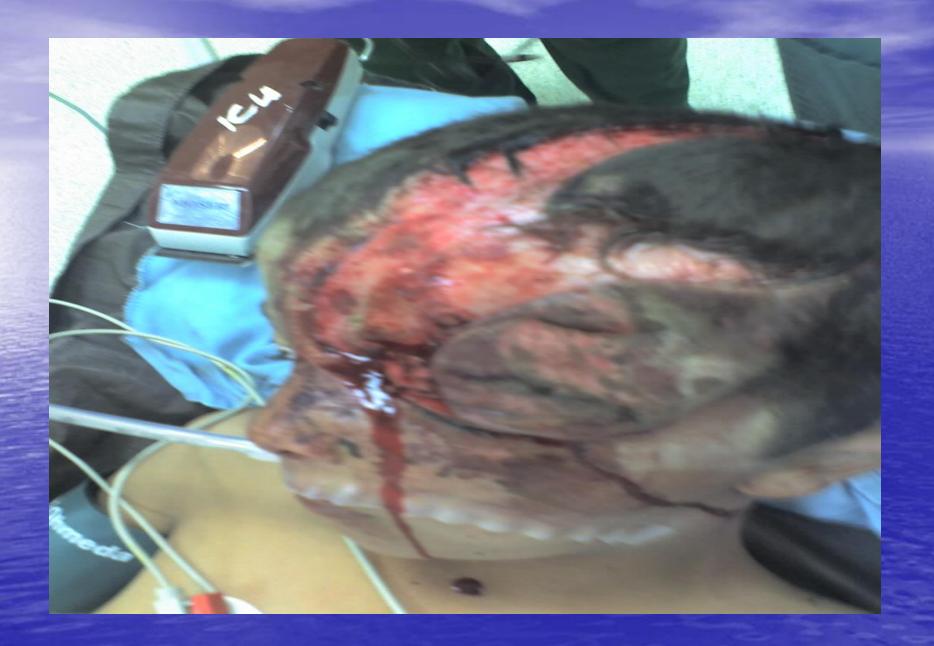
 Small and linear either superficial one (bleeder) or deep one (less beeder) mostly closed under L.A in 2 layers (Galia and skin)

 Stellated and large either superficial one (bleeder) or deep one (less beeder) mostly closed under G.A especially in children, extensive debridement is unnecessary, all devitalised tissue should be derbided.

Reconstruction ladder

- 1. primary suturing in simple lacerations ,sharp, without tissue loss and gross contamination
- 2. secondary intention in wounds with minimum tissue loss
- **3.** debridement and a split thickness graft in wounds with extensive tissue loss and intact pericranium
- 4. local flaps in wounds with extensive tissue loss and stripped pericranium and a split thickness graft for donor area (if a defect is left)
- 5. Pedicular flaps can be used if local flaps are not sufficient especially for occipital areas and small defects
- **6.** Free tissue transplant is used for anterior and large areas





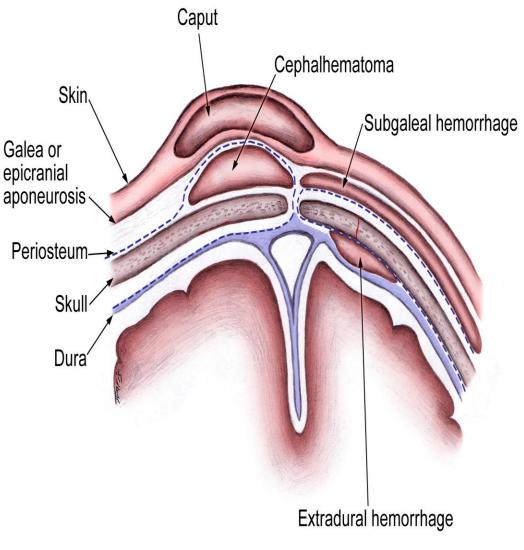


Scalp swelling and hematomas

1.Caput succedaneum

2.Cephal hematoma

3.Subglial hematomas

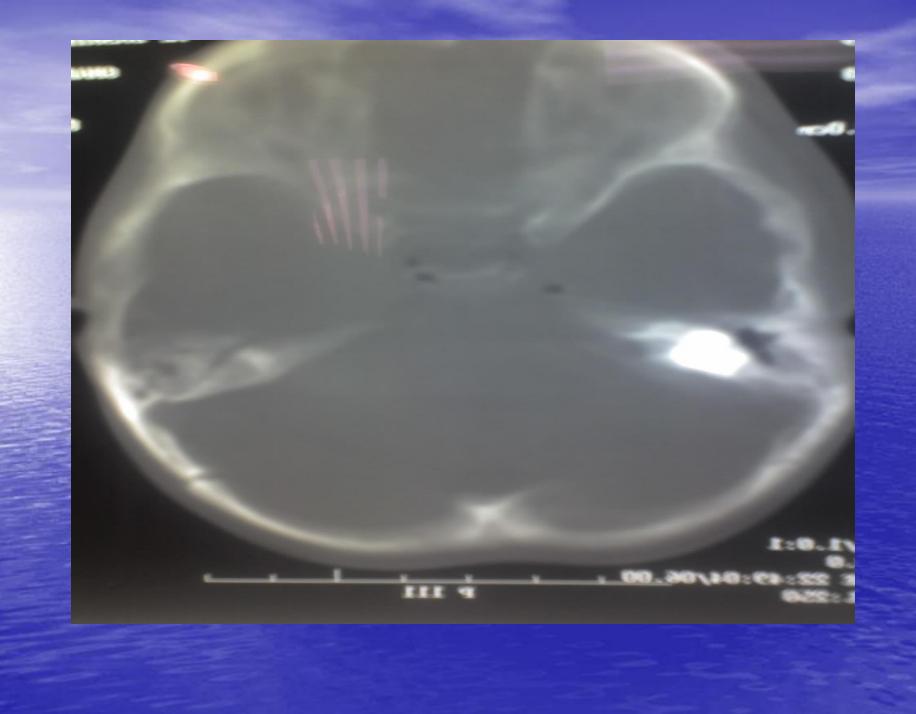


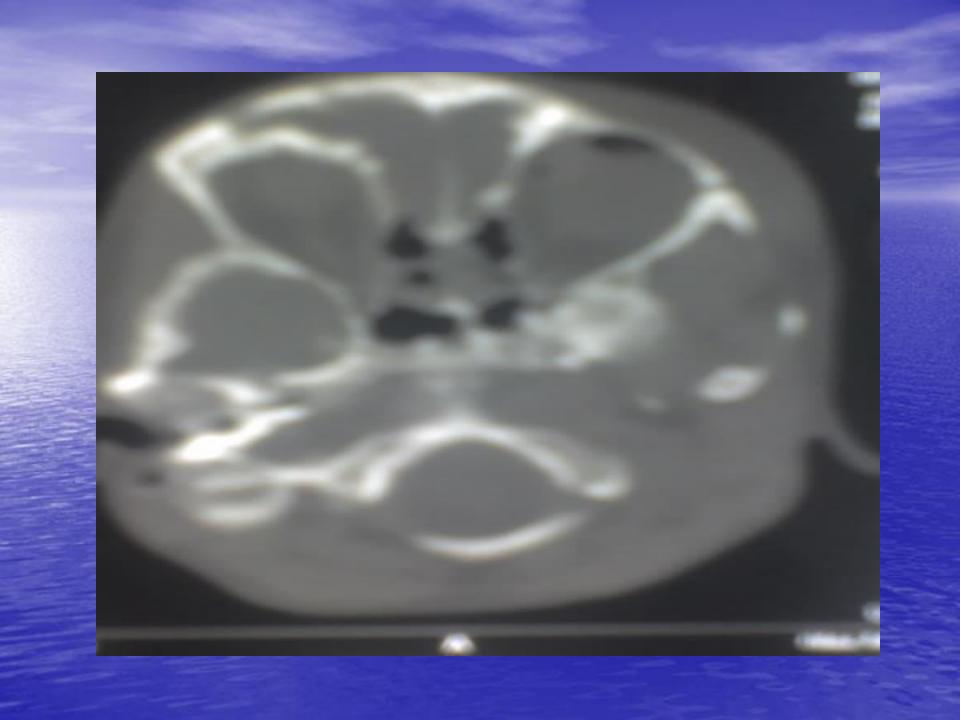
Skull fractures classification

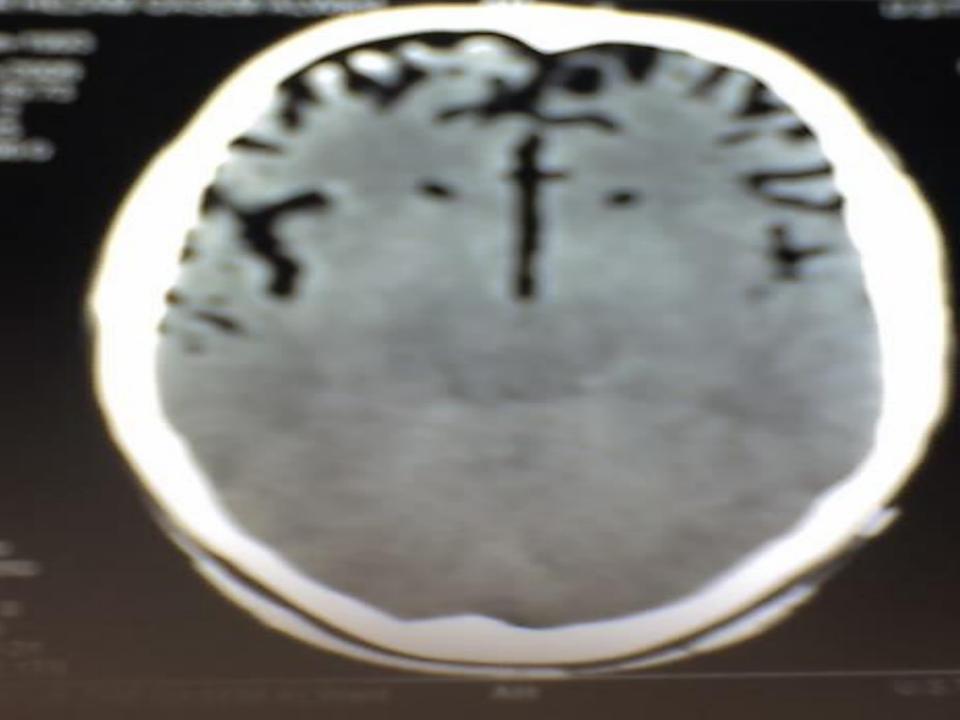
1.by pattern (according to amount of energy, ratio of impact force to surface area)

- Comminuted
- Depressed
- Linear
- Diastatic
- Basal fractures
- Growing
- 2.by type
- OpenedClosed
- 3.by anatomic location
 Basal
 convexity

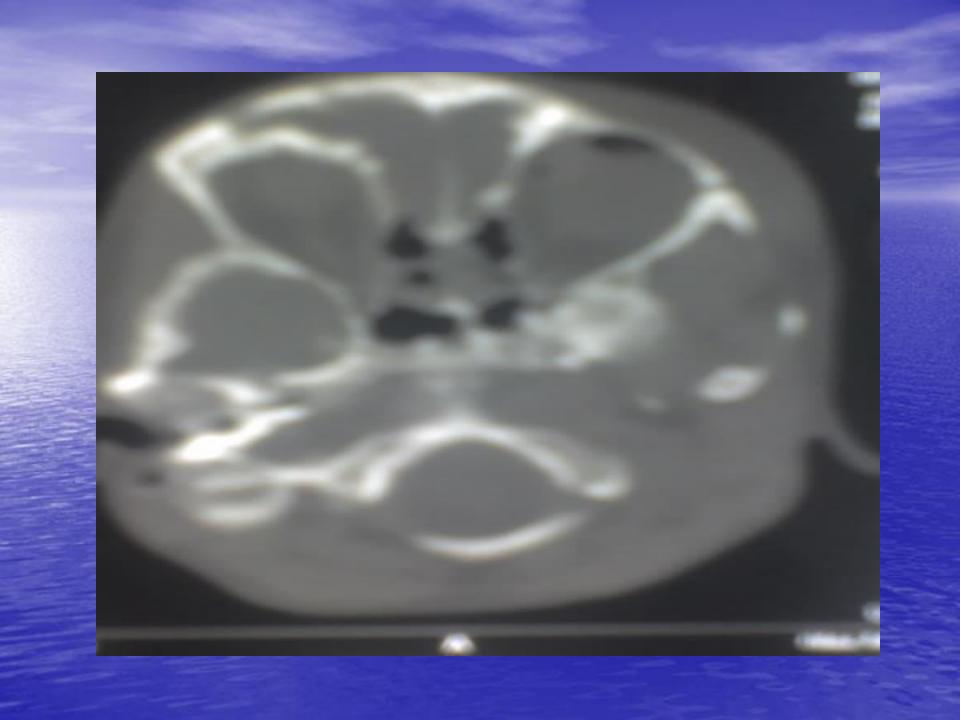


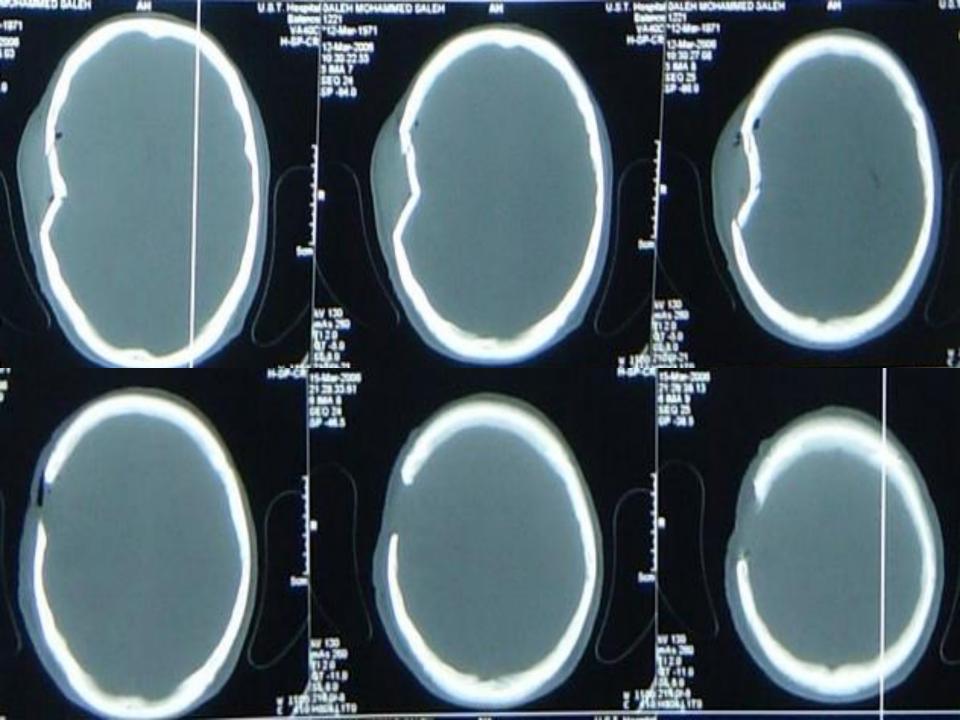


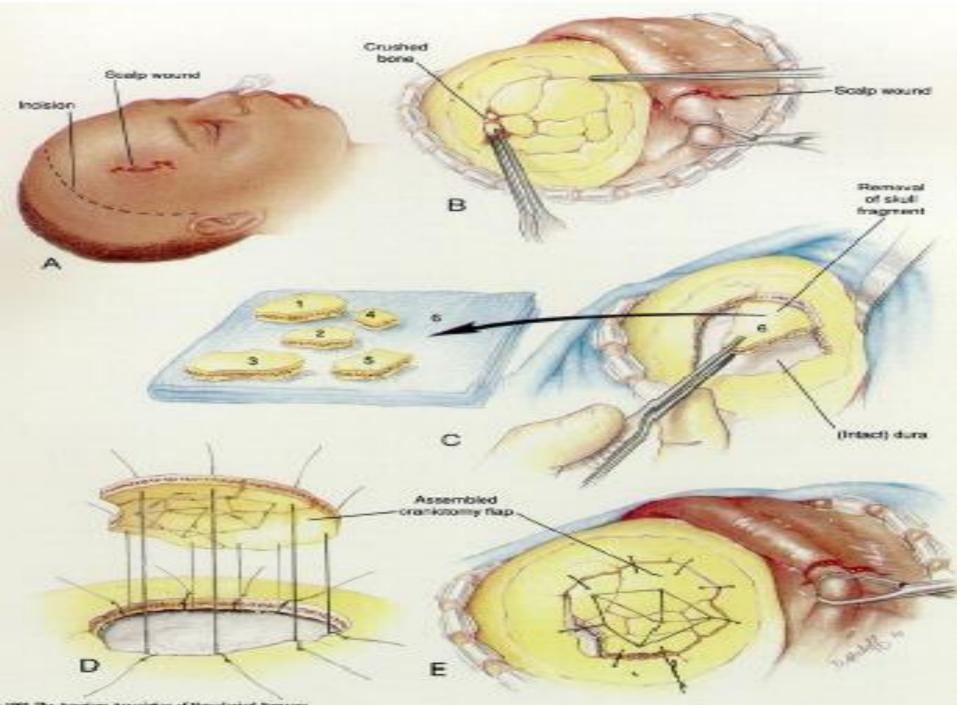




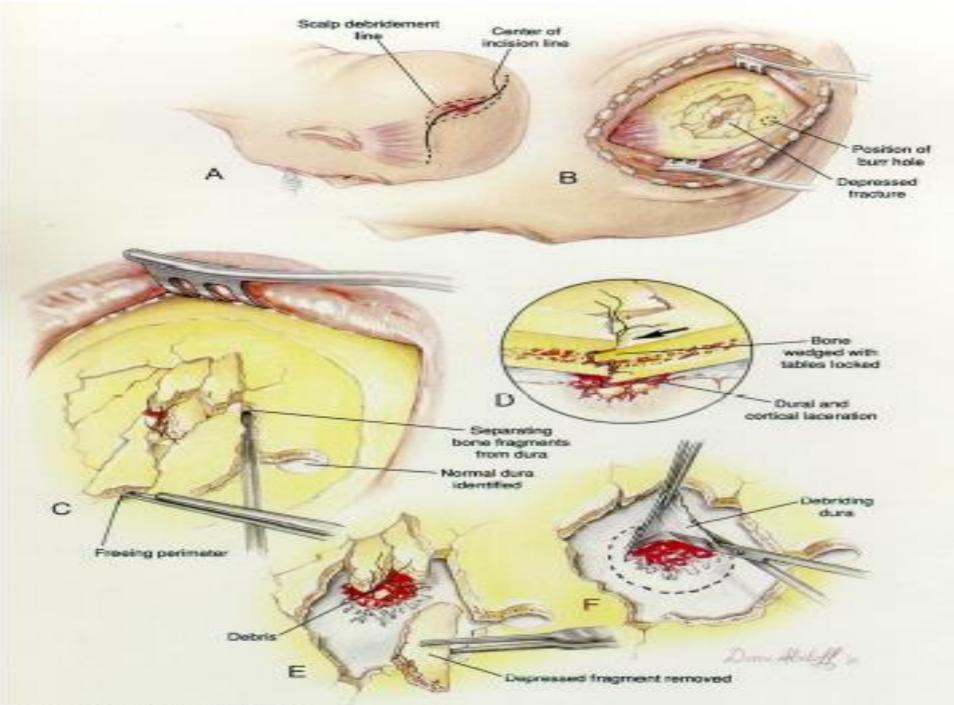




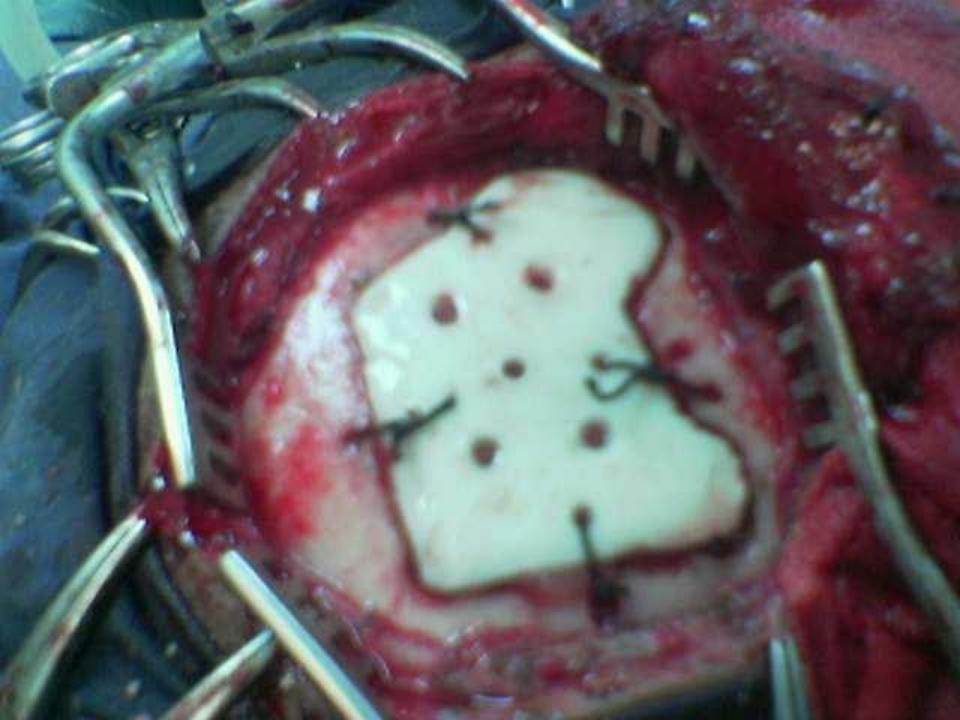














Treatment roles

 Comminuted (replacement as a cranioplasty if not infected with treatment of other associated injuries)

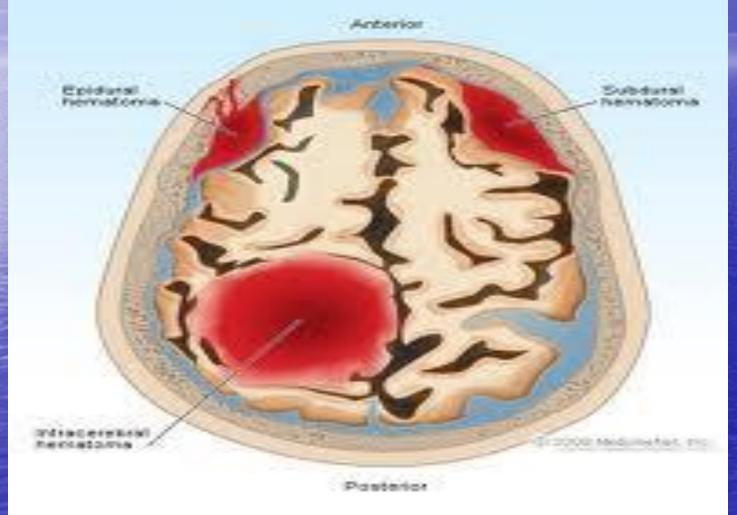
- Linear fractures (no treatment but observation if closed, debridement if infected and compound)
- Depressed(replacement as a cranioplasty if not infected, otherwise good debridement and craniectomy)
- Basilar skull fracture (observation for two days, avoid irrigation of the nose or ear, avoid probing, detailed auditory and vestibular examination is performed at6 weeks interval)
- Frontal sinus fracture (if in the posterior wall cranialization is necessary)

Types of traumatic intracranial leasions

 Focal(epidural hematoma-subdural hematoma-brain contussion-intracerebral hematomas-focal subarachnoid hematoma)

 Diffused(subarachnoid hemorhagediffused axonal injury-concussion)

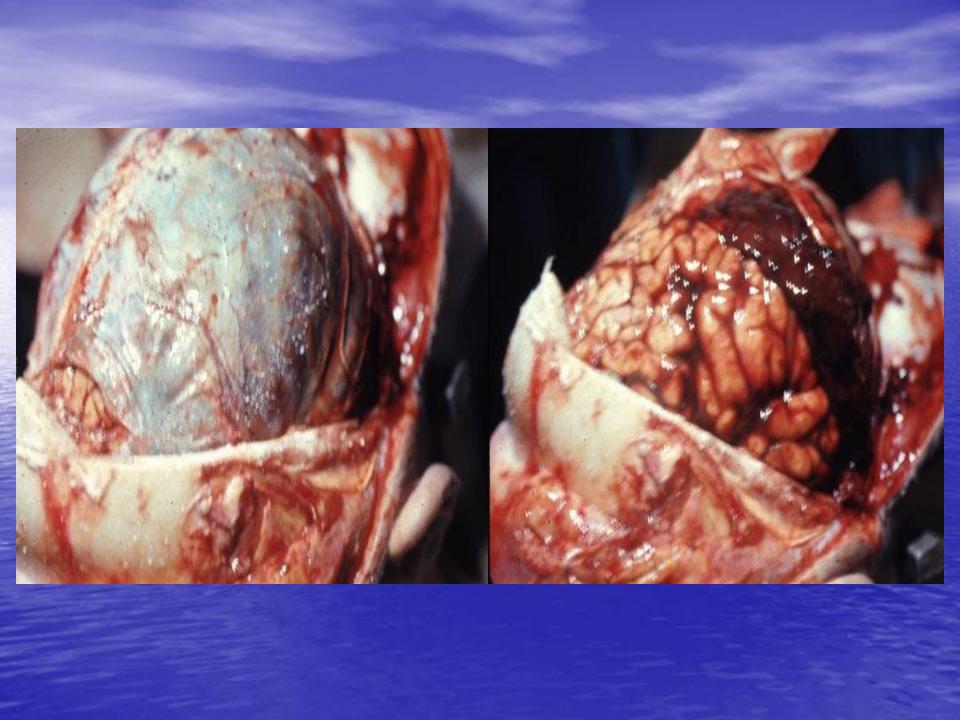




Traumatic intracranial hematomas •**Acute subdural**

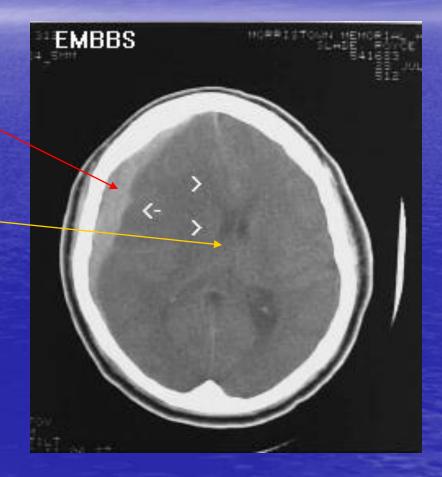
- accelerate high speed impact
- tearing of bridging veins
- bleeding from cortical vessels, venous sinuses
- acute brain trauma may coexist
- altered level of consciousness and focal neurological deficit are common
- CT and rapid evaluation are necessary

 If no signs of rapid deterioration or progressive neurological deficit, no mass effect so observation and control of intracranial p. is necessary, otherwise surgery is the role)



Acute Subdural Hematoma

Another example of acute subdural hematoma with a midline shift (noncontrast CT)



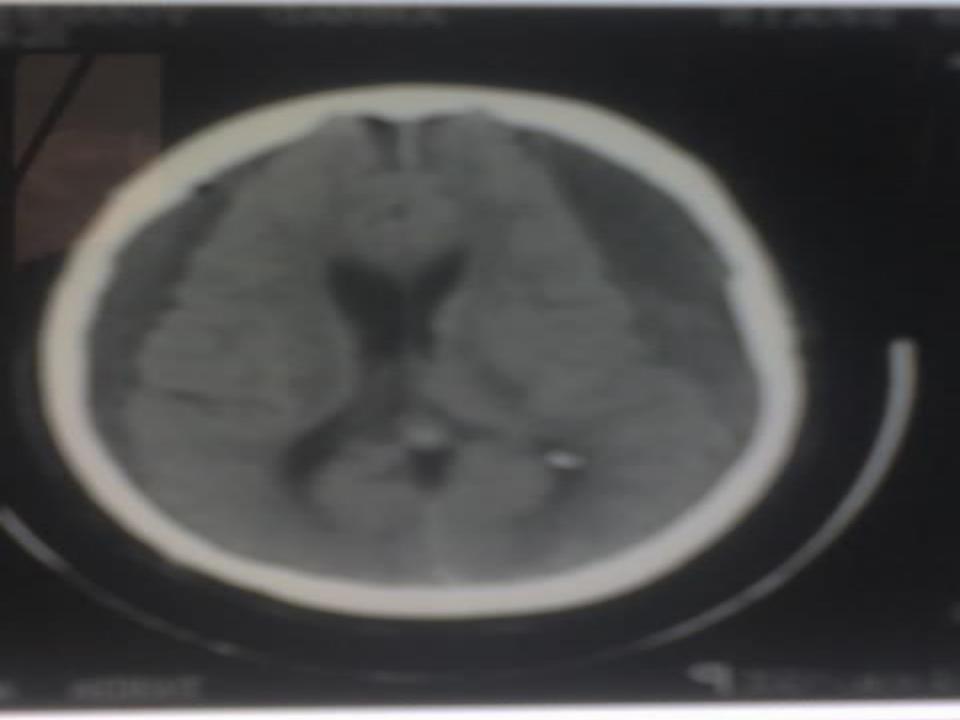
Chronic subdural

Mostly in those over 50 years old • ½ of patients have got no history of trauma If there is any history of trauma , it is trivial Alcoholism, epilepsy, coagulopathy are common Dementia is common presentation In minimal neuological deficit ,medical management is the role If not successful, deterioration of neurological picture so surgery usually by burr hole evacuation.

Subacute Subdural Hematoma

Noncontrast CT note the clot appears less dense in this subacute subdural hematoma.





Subdural Hematoma Copyrig



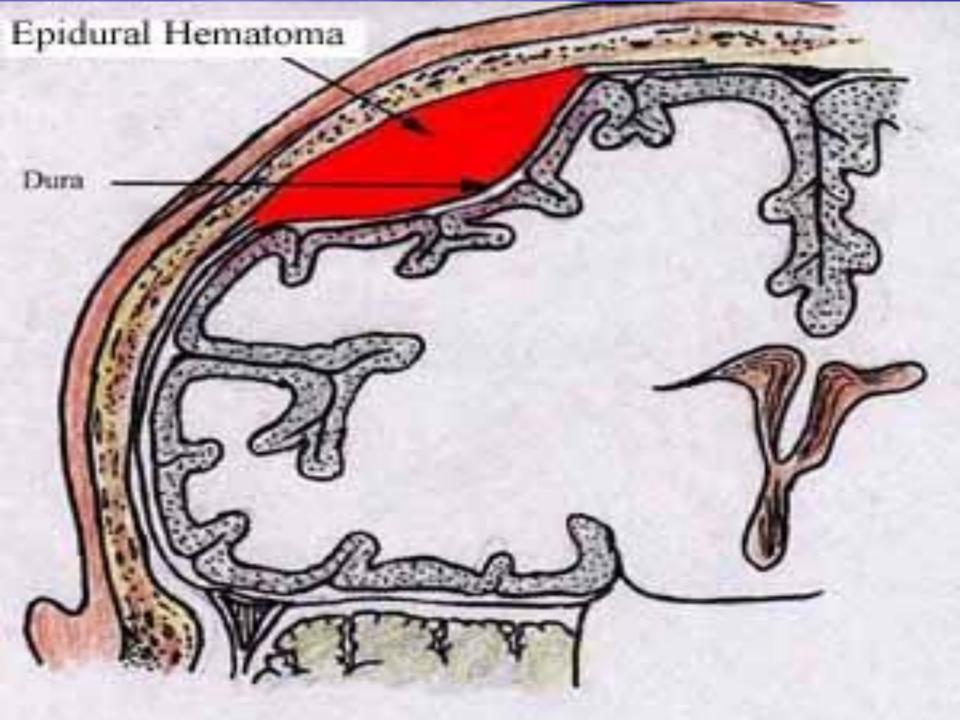
Axial section of skull and brain

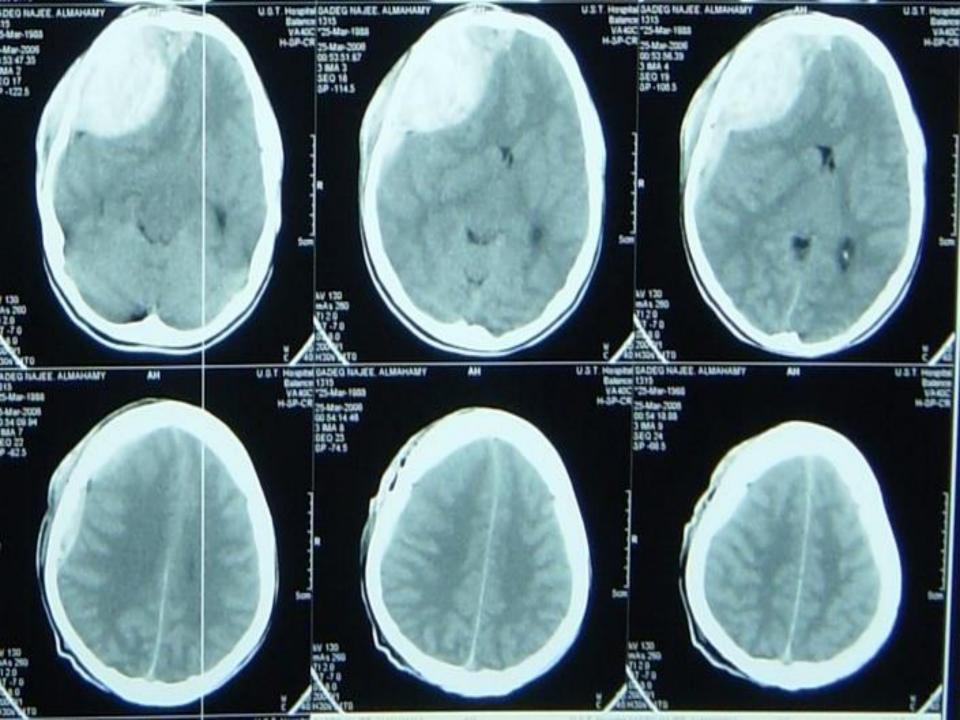
Sample

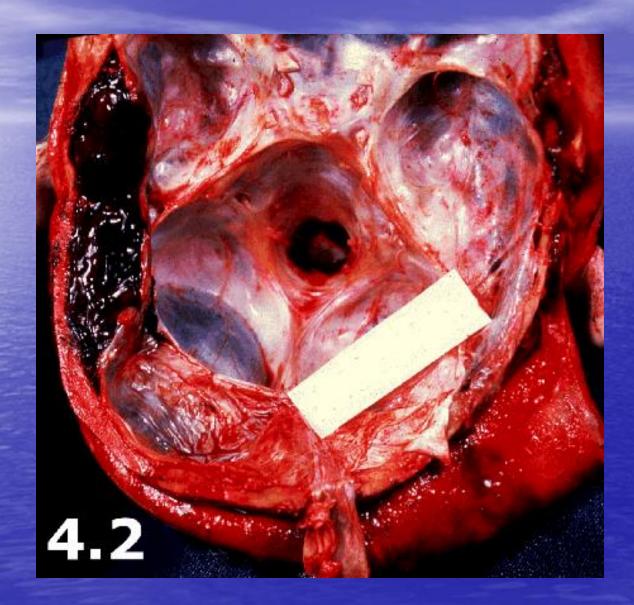


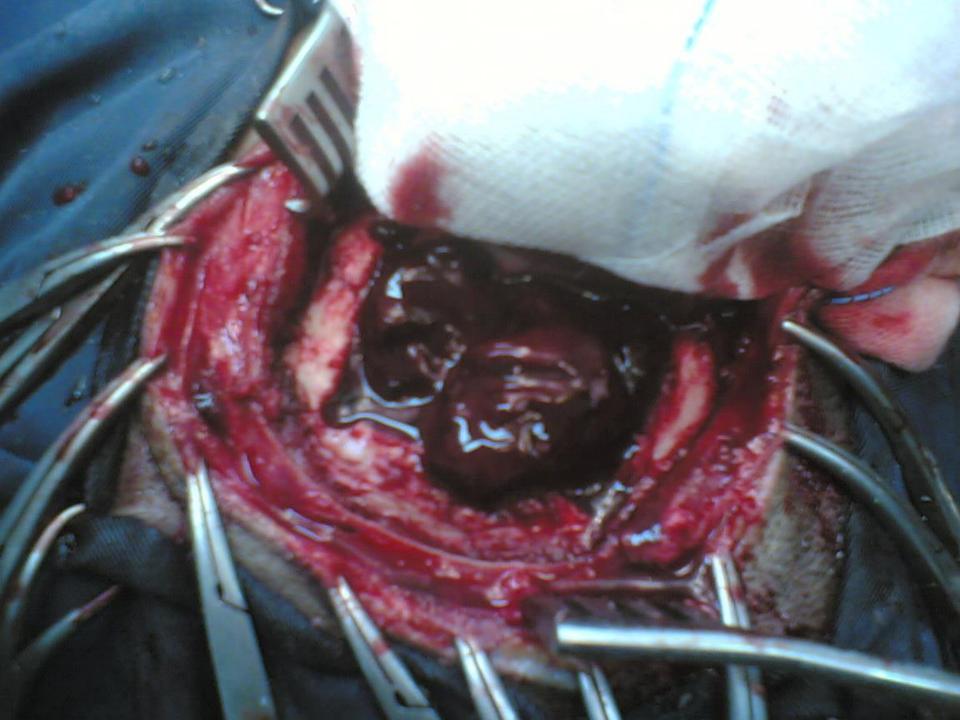
Extradural hematomas

- Mostly resulting from meningial vessel tear (arteries > Veins > sinuses)
- Fractures are common associated injury
- Severe associated brain injury is rare
- Level of consciousness is variable
- If with mass effect must be evacuated within ¹/₂ hour
- if small follow up is recommended
- Prognosis depends on level of consciousness at time of presentation

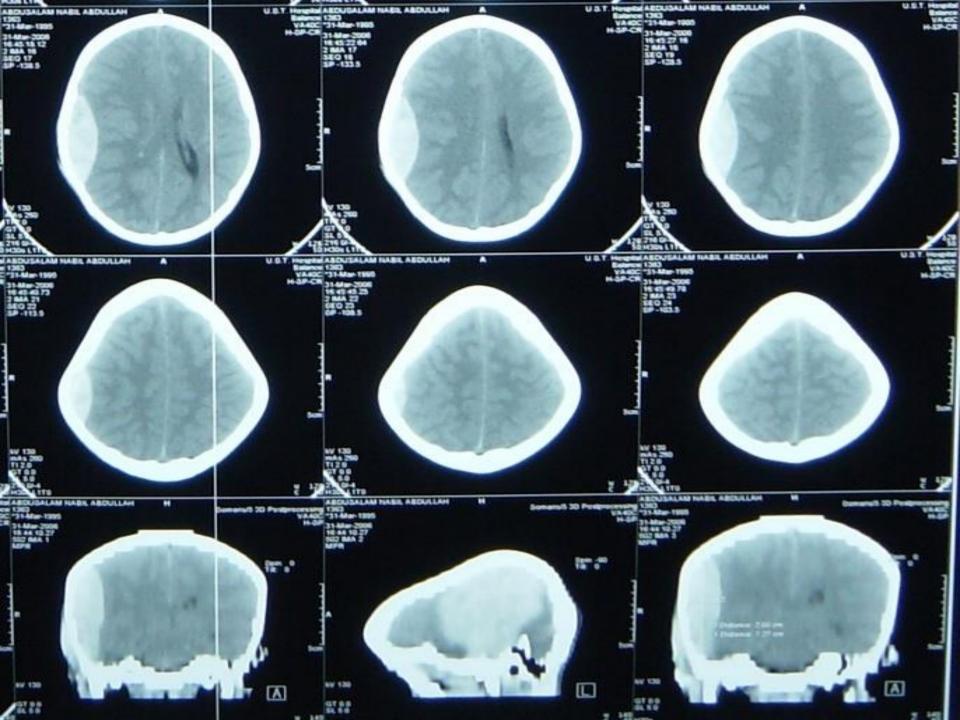








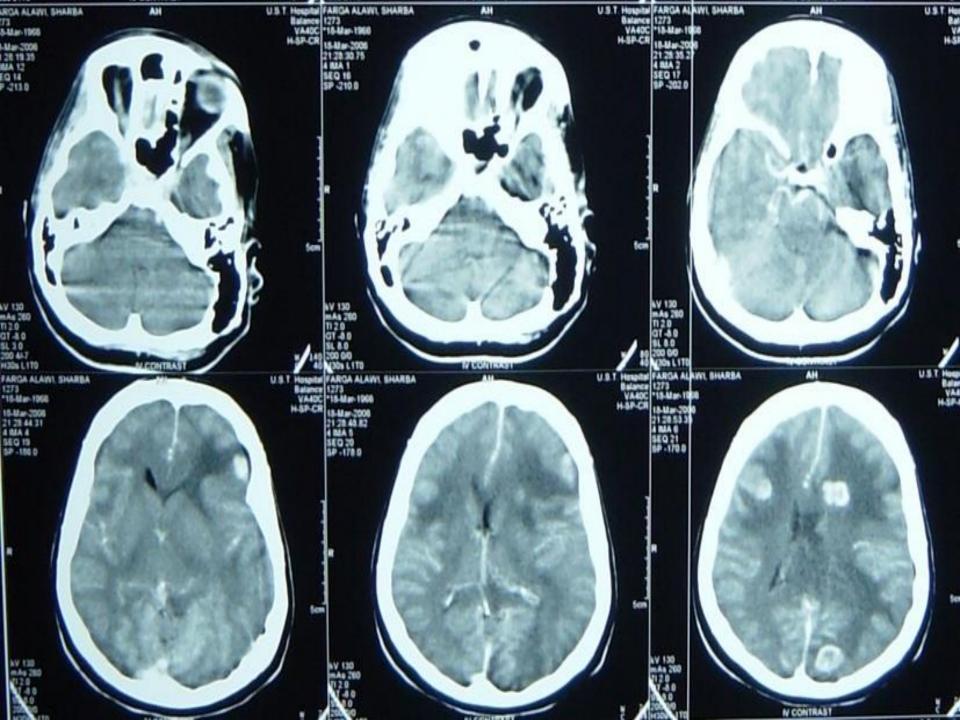


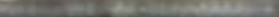




Cerebral contusions

Small and deep one needs follow up Large and with mass effect needs lobectomy Large one may herniate as late as 9 days post trauma Level of consciousness depends on size of contusion and location



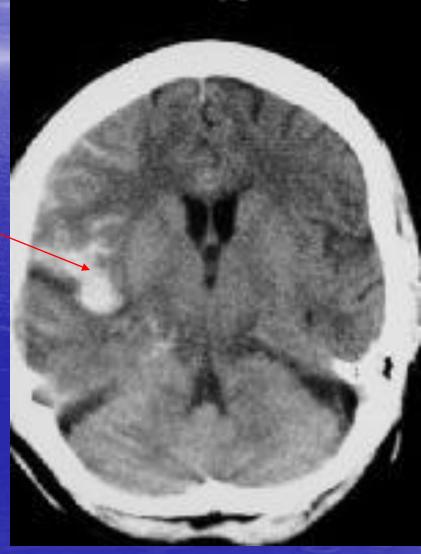


ALL LONG DOM NOT THE OWNER.



SAH—more examples

 Subarachnoid hemorrhage in the right sylvian fissure



SAH—more examples

Blood in the sulci

Edema causing a midline shift

Posterior fossa hematomas

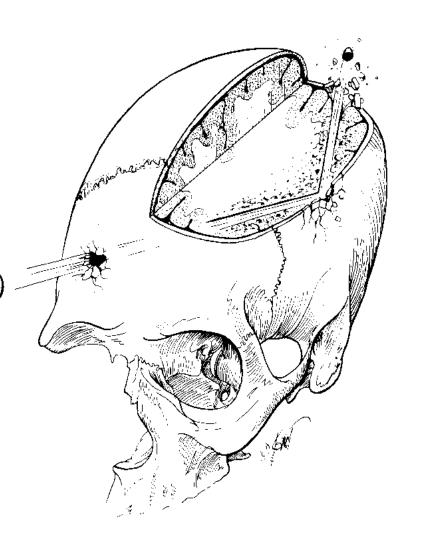
 Usually arise from venous sinuses hemorhage

 Deterioration in level of consciousness is rappid and signs of brain stem and long tract compression are common innitial finding

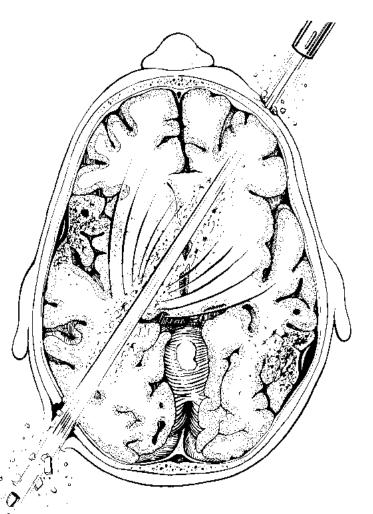
Urgent evacuation is the role

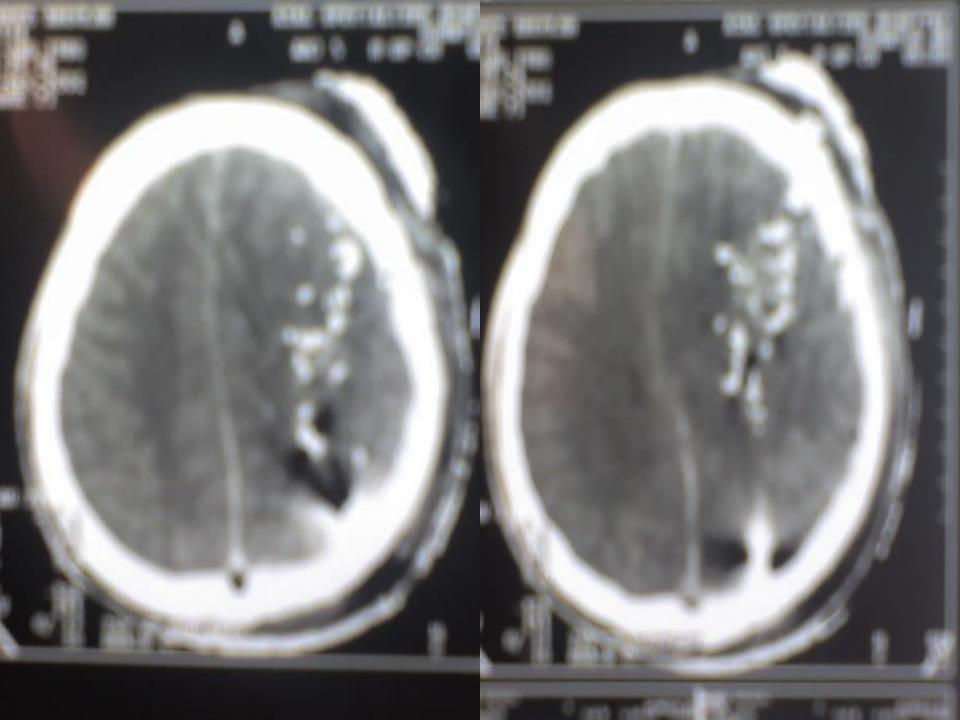
Penetrating head injuries

- Sonic waves and cavitations and decavitation and secondary insults are the injurious mechanisms
- Infection rates are high
- Injury far away from site of entrance and exit is common
- Control of homodynamic state is the initial management
- Surgical interventions are limited to debridement and removal of mass hematomas and for selected cases
- Prognosis is usually bad



Traumatic Damage and Mechanisms of Repair 3





Questions

- What are the types of skull fractures
- What are the parameters of GCS
- Enumerate the types of traumatic scalp swellings
- Enumerate the signs and symptoms of raised intracranisl pressure
- What are the types of traumatic intracranial hemorrhages

Right about types of mechanisms of head injury
What is the pathophysiology of head injury

