HEAD TRAUMA

Traumatic brain injury (TBI) - *nondegenerative*, *noncongenital* brain insult from acute external mechanical force, with associated altered state of consciousness, and temporary or permanent impairments of ***cognitive***, ***physical***, ***psychosocial*** functions.

Degree - determined by initial* postresuscitation** **GCS score**. *within 6-48 hours of TBI
**no hypoxia, hypotension, hypothermia, intoxication, sedation, paralytics

TBI degree	GCS score	Duration of loss of consciousness	Duration of antegrade amnesia
MILD (s. concussion)	13-15	< 30 min	minutes
MODERATE	9-12	30 min ÷ 6 h	hours
SEVERE	≤ 8 (coma)	> 6 h	days

Seizures:
< 7 days – early posttraumatic seizures
> 7 days – posttraumatic epilepsy

systolic BP < 90 mmHg doubles mortality!
PaO_2 < 60 mmHg doubles mortality!

N.B. SAH is most common type of traumatic intracranial hemorrhage!

N.B. despite disrupted autoregulation, vasoreactivity to P_{CO2} remains (enables therapeutic hyperventilation)

BBB

- mechanical forces and ischemia *disrupt BBB* for several hours (demonstrated by contrast MRI) → **vasogenic brain edema** → ICP↑.
- ischemia (produced by any mechanism) causes **cytotoxic brain edema**.

In TBI, both **vasogenic** and **cytotoxic** brain edema occur!

- brain edema reaches maximum at 48-72 hours.

CONCUSSION

- immediate brief (< 6 hours) **loss of consciousness**
  - having ApoE-4 gene increases risk for chronic problems following repeat concussion.
  - normal CT/MRI.
  - DTI is abnormal!

Second Impact Syndrome
- malignant (fatal) cerebral edema.

- cause – one minor HI followed in short order by **second minor HI** in athletes who are **still symptomatic from first injury**.
- mechanism - impaired cerebral autoregulation → vascular congestion → brain edema → herniation → sudden death.

Postconcussion (s. posttraumatic) syndrome - frequent (30-50%) sequela of mild head injury: headaches, dizziness, nonspecific psychological symptoms, blurred vision, hyposmia.

- on objective examination - minimal or no neurologic abnormalities (fixed neurologic deficits are not part of PCS).

Chronic traumatic encephalopathy (s. dementia pugilistica) - **parkinsonism** and other extrapyramidal features + progressive **dementia**, **behavioral** abnormalities

HIGH RISK MILD INJURY	**LOW RISK MILD INJURY**
A. External signs of trauma B. Skull fracture C. Initial GCS 13 D. Loss of consciousness (> 2-5 min) E. Posttraumatic confusion/amnesia (> 20 min) F. Focal neurologic findings	A. Currently asymptomatic (incl. fully awake, GCS 15, no focal neurologic findings, normal pupils) B. No other injuries (incl. no evidence of skull fracture) C. No loss of consciousness

G. Asymmetric pupils H. Posttraumatic seizure I. Repeated vomiting or vomiting for > 8 hours after injury J. Persistent severe or worsening headache K. Second ED visit because of persistent symptoms L. Multiple trauma M. Serious painful distracting injuries N. Bleeding disorder/anticoagulation O. Cerebrovascular malformation P. Intoxication (→ unreliable examination) Q. Mechanism: high-speed motor vehicle accident, fall > 8 ft R. Unreliable / unknown history of injury S. Suspected child abuse T. Age > 60 or < 2 yrs	D. Intact orientation/memory E. Not intoxicated F. Accurate history G. Trivial mechanism H. Injury > 24 hr ago* I. Reliable home observers

*may miss chronic subdural hematoma

DIFFUSE AXONAL INJURY

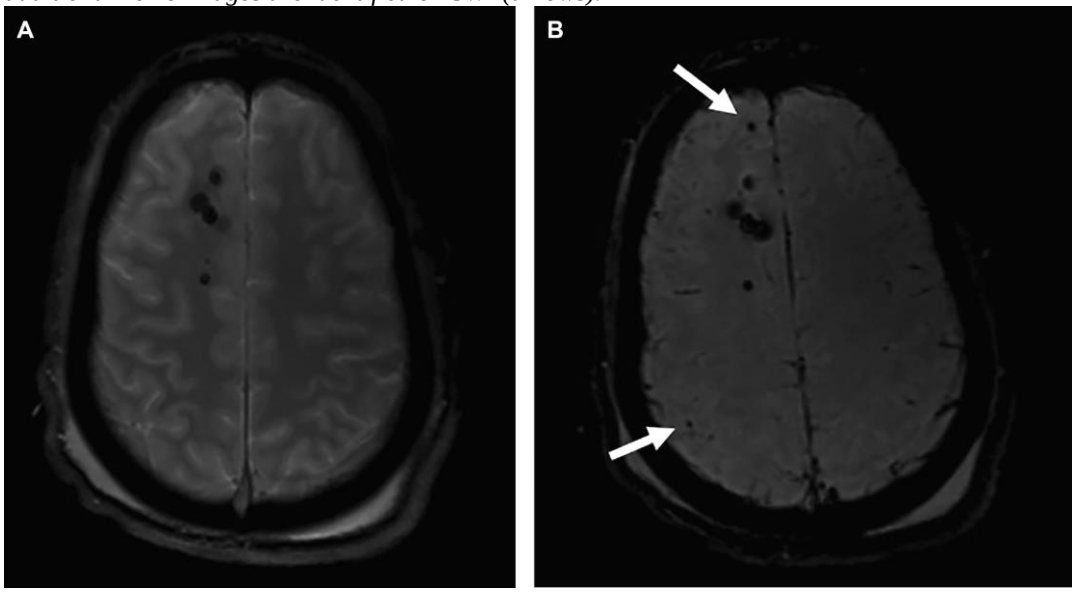
- immediate loss of consciousness lasting > 6 hours (absence of any *intracranial mass lesion* or history of *hypoxia*)
- little cerebral swelling - no ICP↑ (? children may develop diffuse cerebral edema).

All patients with DAI present identically in coma - no early clinical predictor differentiates mild, moderate, or severe DAI!

Centripetal theory of A.K. OMMAYA and T.A. GENNARELLI (1982) - increases in rotation / acceleration / deceleration force involve progressively deeper (medial) areas of brain:

1) **mild DAI (grade 1)** – lesions only in *subcortical axons* (mainly in parasagittal white matter of cerebral hemispheres).
2) **moderate DAI (grade 2)** – plus lesions in *corpus callosum*.
3) **severe DAI (grade 3)** – plus lesions in dorsolateral quadrants of rostral brain stem (*cerebral peduncle*).

SWI is more sensitive for IPH than traditional GRE sequences. **A**, *GRE shows several small ICH.* **B**, *Two additional hemorrhages are identified on SWI (arrows).*



CONTUSION AND LACERATION

- foci of hemorrhagic necrosis (*hemorrhage mixed into tissue**) on brain surface - result of CONTACT (IMPACT) injury.

*vs. ***HEMATOMA*** - focal collection of blood

CONTUSION - pia-arachnoid is intact (e.g. in blunt injuries).
LACERATION - pia-arachnoid is torn (e.g. in penetrating injuries).

N.B. contusions *progress with time* in size, number, and amount of hemorrhage within contusions – most evident *over first 24-48 hours ("blossom")* – routinely repeat CT!!!

PENETRATING INJURY

penetrating injury (esp. due to impalement) – absolute indication for **angiography*** + **seizure prophylaxis**!

*pseudoaneurysm – very vulnerable to delayed ruptures

A. Penetrating injury - projectile breaches cranium but does not exit;
B. Perforating injury (worst prognosis!) - projectile passes entirely through head, leaving both entrance and exit wounds.

Angiography / CTA – when vascular injury is suspected:

a) any patient that goes to OR
b) wound's trajectory passes near Sylvian fissure, supraclinoid carotid, major venous sinus.
c) unexplained SAH or delayed hematoma.

- pseudoaneurysms may form in ***delayed fashion*** (repeat CTA in several weeks, esp. before cranioplasty).

CAROTID-CAVERNOUS FISTULA

DEXAMETHASONE → Endovascular obliteration

HEMATOMAS

Rule of thumb: blood remains ***denser*** than brain for 1 week, and is ***less dense*** after 3 weeks

- causes of acute ***hematoma density***↓: *severe anemia*, *hyperacute hematoma* (no clots at all).

EVALUATION, TREATMENT

ABC

Always start: hypoxia, hypotension, hypothermia, anemia, and multiple injuries!
+ Seizures!

"**GOLDEN HOUR**" – *first hour is very important prognostically* – treat hypoxia & hypotension in the field and en route to the hospital

- early **endotracheal intubation**:
    1) **COMA** (GCS < 8)! ← i.e. even if localizing, making sounds, eyes swollen shut (GCS 8)
    2) **PENETRATING INJURY** (if physician waits for coma before intubating patient, mortality approaches 100%).
    3) extensive **FACIAL INJURIES**
    4) **COMBATIVE** patient
- isotonic (or hypertonic*) saline to aggressively restore SBP to > 100-110 mmHg.
  *there are studies showing that 250 mL of hypertonic (7.5%) saline bolus in the field improves survival

  N.B. in all neurotrauma cases maintain SBP > 110 mmHg (DAMAGE CONTROL RESUSCITATION)

1. Systolic BP goal 100-160 mmHg (IV fluids ± vasopressor agents)
   Level III recommendation: Maintain SBP ≥ 100 mmHg (for 50-70 years old) or ≥ 110 mmHg (for < 50 or > 70 years old) to decrease mortality and improve outcomes.
   - do not treat hypertension < 160* mmHg until intracranial hypertension is excluded (Cushing reflex is for brain perfusion)

     *< 140 mmHg if ongoing risk of intracranial bleeding

     N.B. *albumin worsens outcomes* (albumin extravasates and worsens cerebral edema)

     Level II evidence – avoid SBP < 90 mm Hg
     single episode of SBP < 90 mm Hg is associated with doubling of mortality in severe TBI (≥ 2 hypotension episodes increase mortality 8-fold)
2. CVP 5-15 cmH_2O
3. $PaCO_2$ goal 35-45 mm Hg
4. Avoid PaO2 < 60 mmHg or SaO2 < 94%.
5. Hb goal > 7
6. No PEEP (increases ICP, lowers CPP), tidal volume 8 mL/kg (modern approach – OK to use PEEP and tidal volume 6-7 mL/kg)

CERVICAL SPINE INSTABILITY

Spinal cord injury is present in as many as 10% patients!

Every patient with significant TBI has cervical spine injury until proved otherwise!!! → C-spine stabilization

BRIEF NEUROLOGIC STATUS

1. **Level of consciousness** (ideally GCS)
2. **Pupil size and light reactivity** (asymmetry is most important) → **brainstem reflexes**.
3. **Extremity motorics** (asymmetry is most important) - [1]spontaneous movements, [2]following commands, [3]reaction to painful stimuli.

N.B. decrease of even 1-2 points in GCS score indicates significant change in neurologic status → prompt reevaluation!

Pupils - only indicator of neurological function in chemically paralyzed patient:

Asymmetry - measurement difference of ≥ 1mm.
Dilated - pupillary size of > 4 mm.
Fixed – pupillary response < 1 mm to bright light.

pupillary asymmetry is due to intracranial injury unless proved otherwise.
unilateral dilated pupil in unconscious patient – CN3 compression (uncal herniation).
pinpoint pupils - pontine lesions.
nonreactive pupils in mid position - midbrain tectum lesions.

IMAGING

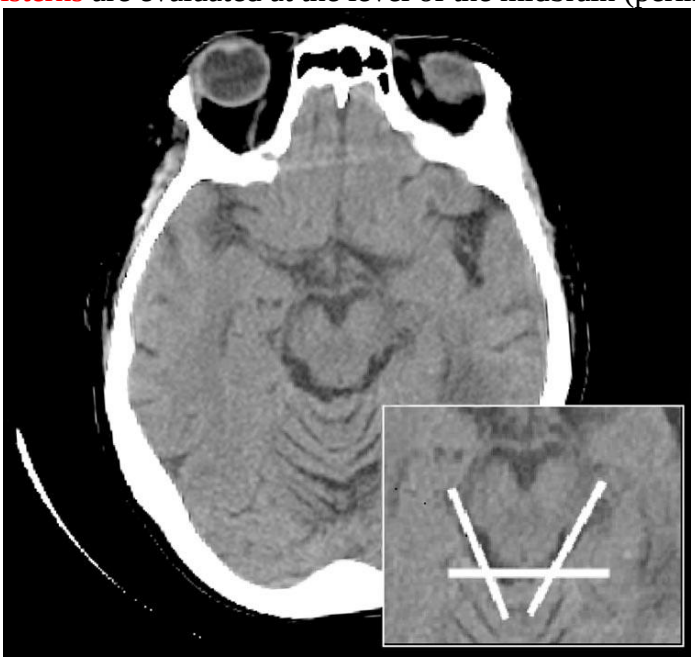
1. CT – head, panspinal (as chest-abdomen-pelvis)
CTA – if fracture patterns indicate vascular injury (skull base fracture), penetrating injury
Always look for all images (incl. panspine CT)

DSA indications – if CTA shows:

a) impaled objects / trajectories close to major vessels
b) symptomatic carotid injury
c) symptomatic skull base fracture

5 important things to look in CT: mass lesions (hematoma), state of basal cisterns (incl. blood inside), midline shift, ventricular size, fractures (depressed, frontal sinus)

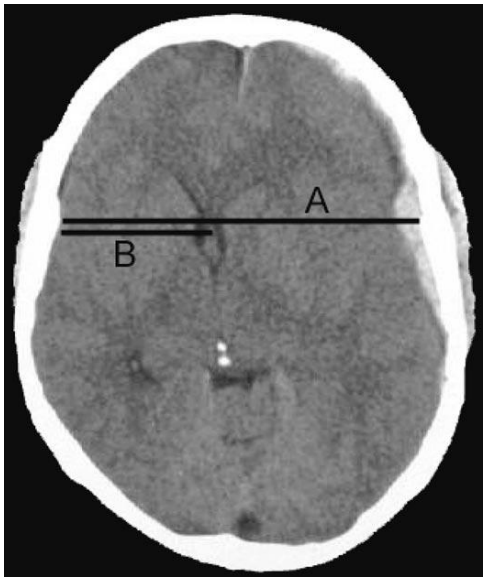
Basal cisterns are evaluated at the level of the midbrain (perimesencephalic cisterns):



Basal cisterns can be: **open** (all limbs open), **partially closed, s. "crowded"** (one or two limbs obliterated), or **completely closed s. "effaced"** (all limbs obliterated).

Midline shift is calculated at the level of the foramen of Monro to the septum pellucidum:

Midline shift = (A/2) - B



Marshall Classification of Diffuse Brain Injury

Grade 1 = **normal** CT scan (9.6% mortality)
Grade 2 = cisterns present, **shift < 5 mm** (13.5% mortality)
Grade 3 = **cisterns** compressed / absent, shift < 5 mm (34% mortality)
Grade 4 = **shift > 5 mm** (56.2% mortality)

Marshall Scoring of TBI

	MLS	Cisterns	High or mixed-density lesion	Notes
I	None	Present	None	No visible pathology on CT scan
II	0-5mm	Present	None	
III	0-5mm	Compressed or absent	None	Swelling
IV	>5mm		None	
V	Any	Any	Any	Any lesion surgically evacuated
VI			>25cm3	Not surgically evacuated

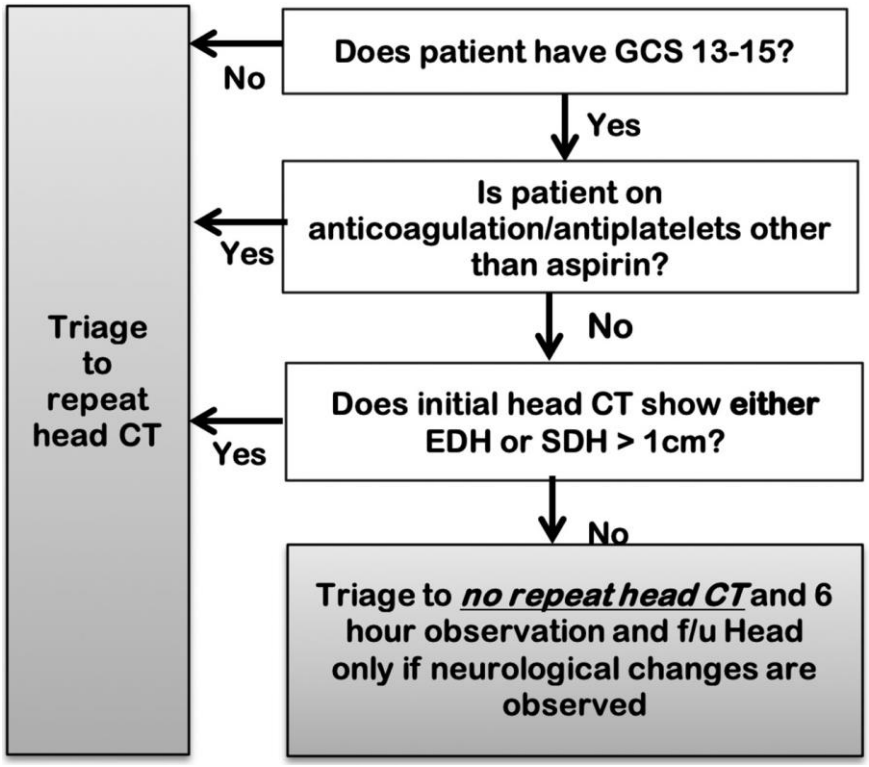
*MLS-midline shift

(Heustein Sy)

Harvard protocol

Complicated mild TBI (head CT positive for an intracranial traumatic finding + nonfocal neurological examination + GCS 13-15) – if initial presentation is not operative, then ***does not need repeat CT*** (unless patient is on "blood thinners"* or there is EDH or SDH > 1 cm) – can be discharged after clinically stable 6-hr observation in ED:

*except Aspirin



- probability of needing operative intervention is extremely low (< 1%) if the initial CT scan was none operative.
- stable routine follow-up head CT does not protect the patient's condition from deteriorating later!
- traumatic intracranial hemorrhages are associated with 17-20% progression rate but this ***radiographic progression rate does not result in need for neurosurgical intervention***.
- 1 year of implementation - protocol allowed to avoid 71% unnecessary CT scans
  - there were no missed injuries or delays in neurosurgical intervention.
  - none of the patients in no-repeat-CT group underwent delayed surgery.

MRI prognostic use during subsequent care

T2* (either GRE [gradient-recalled echo] or SWI) is most sensitive sequence to detect small *hemorrhages, blood breakdown products*.
DWI - useful for *infarctions*;
FLAIR - useful for *SAH*.

N.B. gadolinium enhancement gives no notable advantage!

EEG - not emergency test!

Most useful role of EEG - diagnosis of **nonconvulsive status epilepticus**!*

*may be detected in ≈ 8% comatose patients when imaging and normal ICP do not explain poor exam

Transcranial Doppler

- normal average linear blood velocity (LBV) in MCA ≈ 60 cm/s.
- LBV > 100-120 cm/s:
  a) ***hyperemia*** (parallel LBV increase in MCA and ICA) – esp. days 2-4 after TBI
  b) ***vasospasm*** (LBV in MCA exceeds ICA ≥ 3 times) – usually on days ≥ 5 post TBI

GOSLING (PULSATILITY) index = (systolic LBV - diastolic LBV) / mean LBV.

if > 1 – sign of intracranial hypertension

HEMATOMAS

- traumatic SAH (most common type of bleed in TBI) is typically located in ***interhemispheric or sylvian fissure***, ***cerebral sulci*** (vs. SAH from ruptured cerebral aneurysm – primarily in basal cisterns).
- *DELAYED HEMORRHAGE (DTICH – DELAYED TRAUMATIC ICH) S.* ***BOLLINGER'S SPÄT-APOPLEXIE*** (occurs in 1-7% TBI patients) – ICH that develops in the part of the brain that on the initial CT looked "normal".

SYSTEMIC ICU MEASURES

SBP > 100 (110), pO2 > 60, pCO2 35-45

NUTRITIONAL SUPPORT

- **early nutritional support** can directly affect TBI outcome – 1-1.5 g/kg/d protein!

  Level II recs: Transgastric **jejunal** feeding (to reduce the incidence of ventilator-associated pneumonia) at least by the 5th day (earlier for GSW)

DVT

- early (< 24 h) initiation of VTE chemoprophylaxis even in traumatic intracranial hemorrhage appears to be safe.

HEMOGLOBIN

Maintain Hb > 7 g/dL

SEIZURE PROPHYLAXIS

- routinely in severe TBI and/or cortical irritation but only for 7 days
N.B. norepinephrine is seizure suppressant.
if patient has developed seizures after first 24 hours, antiepileptic is continued for 6 months ÷ 1 year
N.B. seizures within first 24 hours is not indication to extend AED beyond 7 days.
N.B. no evidence that deeply situated foreign body predisposes to development of seizures.

STRESS ULCER PROPHYLAXIS

PPI

ABX

For open skull fractures (incl. extending to paranasal sinuses, middle ear):

"TRIPLE ANTIBIOTIC" x 5 days???:

1) CEFTRIAXONE 2 g q12h or cefepime 2 g q8h **plus**
2) NAFCILLIN 2 g q4h **plus**
3) METRONIDAZOLE 500 mg q8h

TXA

CRASH-3 trial

- randomised, placebo-controlled trial, 175 hospitals in 29 countries – 9202 patients.
- inclusion: within 3 h of TBI, GCS 4-12 or any intracranial bleeding on CT
  + no major extracranial bleeding, no fixed dilated pupils.
- TXA protocol: loading dose 1 g over 10 min then infusion of 1 g over 8 h.

- risk of vascular occlusive events, seizures was similar in the tranexamic acid and placebo groups.
- primary outcome was head injury-related death in hospital within 28 days of injury: 12.5% in TXA group vs 14.0% in placebo group (RR 0·89 [95% CI 0·80–1·00]).
  - in mild-to-moderate TBI (RR 0·78 [95% CI 0·64–0·95]) but not in severe TBI (0·99 [95% CI 0·91–1·07])
  - early treatment was more effective than was later treatment in mild and moderate TBI (p=0·005) but time to treatment had no obvious effect in severe TBI (p=0·73).
  - ***no difference in disability*** among survivors.

[CRASH-2 trial - in patients with trauma with **major extracranial bleeding** (isolated TBI was specifically excluded), early TXA (within 3 h of injury) reduces bleeding deaths by a third]

ICP

Indications for ICP monitor:

A. ***GCS*** > ***8*** + significant mass lesions on CT scan (but often such patients need to go to OR)
B. ***Salvageable patient with GCS*** 3-***8*** (after resuscitation) plus:
   a) abnormal CT scan - ***mass lesions*** (contusions, hematomas) when patient is not taken to operating room for evacuation OR ***diffuse cerebral edema*** (esp. with obliteration of perimesencephalic cisterns – limited residual compliance).
   b) normal CT scan + any two of the following (on admission): SBP < 90 mmHg, age > 40 yrs, unilateral or bilateral motor posturing.

- even patients taken to operating room - ICP monitor is placed at end of operation - later brain swelling may still be a problem even after successful mass evacuation (and even after craniectomy).
- ICP monitor is ***zeroed at midbrain*** level.
- MAP by convention is calibrated to the level of the right atrium of the heart.
- Central venous pressure (CVP) is also calibrated to the level of the right atrium of the heart; but in TBI ICP is higher than CVP.
- when ICP remains < 20 mmHg for 24-72 hours without treatment, ICP monitoring is discontinued (exceptions exist).
- ICP > 22* (formerly 25) mmHg for > 15 mins (cumulative) within 1 hour must be treated ASAP (to keep CPP 60-70 mmHg).
  *> 15 mmHg after decompressive craniectomy

- CPP goal 60-70 mmHg

  Level III recommendation: avoid aggressive attempts to maintain CPP > 70 mm Hg with fluids and pressors because of the risk of ARDS.

  Level III recommendation: CPP < 50 should be avoided.

  - attention to pressure autoregulatory status: patients with intact autoregulation (PRx < 0.05) are best served by higher CPP values while pressure-passive patients with dysfunctional pressure autoregulation do better with lower CPP values.

Growing evidence that **hypertonic saline** is better for underresuscitated patient than mannitol!

N.B. mannitol is a diuretic and HTS is not.

HTS bolus therapy appears to be superior to mannitol in reduction of the combined burden of intracranial hypertension and associated hypoperfusion in severe TBI.

Comment (Dr. Hawryluk): it is important to replace fluid renal losses after mannitol administration; sometimes ICP responds better to mannitol than to HTS (mannitol is not naturally found – may have better osmotic gradient).

- pooled data from 3 trials showed continuous HTS therapy was associated with improved survival over bolus HTS therapy (?, need to check)

SEDATION

- PROPOFOL is recommended for ICP control. Alt - PRECEDEX!!!
- PENTOBARBITAL is recommended to control refractory ICP - *hypotensive effect* may offset any ICP lowering effect on cerebral perfusion pressure!!!!
- for agitation **antipsychotics** are useful when used sparingly.

ANALGESIA – PARACETAMOL, FIORICET, IBUPROFEN, FENTANYL.

HYPOTHERMIA should be used as a last resort!!! - *may increase mortality* (coagulopathy, immunosuppression, cardiac dysrhythmia)

NABIS: H II trial (National Acute Brain Injury Study Hypothermia II)

Each 1° C drop → cerebral metabolic rate of oxygen ($CMRO_2$) drops by 7%

ADVANCED BRAIN OXYGENATION / METABOLIC STATUS MONITORING

brain tissue oxygenation (pbO2) goal > 20 mmHg
jugular venous oxygen saturation (SjO_2) goal > 50%

normal brain tissue O_2 partial pressure Pti_{O2} = 25-45 mmHg

BOOST-2 trial – ICP monitor alone* vs. ICP monitor plus $BtpO_2$
*brain oxygenation monitor was still placed but the monitor and numbers were hidden from treating staff

- having brain oxygenation data available reduced the duration of brain hypoxia
- condition at discharge was better in ICP + $BtpO_2$ group

ORTHO SURGERY

Long bone fractures – operate when safe (earlier if OK from TBI standpoint).
Open fractures – débridement within 24 hrs.

SURGERY

1. Coags, Hb & type-and-cross
2. AED
3. abx + tetanus
4. mannitol & hyperventilate
5. check for fractures (skull & C-spine)
6. check for vascular issues - CTA

- if suspect vascular lesion (e.g. young person with deep bleed, penetrating injury):
  1) order **CTA** preop
  2) **exposure of ICA in neck** if injury to intracranial portion of artery is suspected.
  3) plan craniotomy so will have *proximal control* (e.g. by dissecting Sylvian fissure).
  4) start evacuating blood clot ***farthest from suspicious area*** and may leave small clots.

N.B. once intracranial hematoma begins to be removed blood pressure may fall precipitously (esp. if multiple injury has produced hypovolemia, masked by effects of raised ICP).

- **ICP monitor** (ideally EVD) usually is placed intraoperatively in patients with preop GCS ≤ 8 (even if removed bone flap)

N.B. early surgery (by CT criteria) has better morbidity* and mortality outcomes vs. delayed surgery (by clinical deterioration criteria)!

*e.g. ischemic effect of prolonged cortical compression by hematoma

- surgical intervention is *not beneficial* in most patients with GCS 3-5 (unless pupils are reactive and CT shows no bihemispheric/multilobar dominant hemispheric injuries).

SCALP LAC

- if scalp avulsion is *completely detached* from scalp → reimplant ASAP.
- *small scalp deficit* is repaired by rotating portion of scalp.
- *large scalp deficit* requires skin graft or vascularized free flap.

Tetanus!
Bleeding - prispaudžiama prie kaulo *a. temporalis superficialis* (virš arcus zygomaticus).

Traumatic ICH

Surgical Trial In Traumatic intraCerebral Haemorrhage (STITCH)

- randomized trial: Early Surgery (within 12 hrs) vs. Initial Conservative Treatment (delayed evacuation if it became clinically appropriate); tICH > 10 mL.
- clinical equipoise - only patients for whom the responsible neurosurgeon was uncertain about the benefits of either treatment were eligible.

Early Surgery may be a valuable if **GCS is 9-12**; those with GCS of 13–15 can be watched carefully; if GCS has dropped < 8, surgical intervention appears to be less effective.

At 6 months:

- Early Surgery patients were 10.5% more likely to have a favorable outcome and mortality was *significantly* lower (15% vs. 33%).

DECOMPRESSIVE CRANIECTOMIES

1. **Primary decompression – prophylactic measure** during emergency evacuation of traumatic mass lesion.
2. **Secondary decompression – therapeutic to ↑ICP refractory** to medical treatment.

Guidelines for the Management of Severe Traumatic Brain Injury (4th ed): 2020 Update:

- incorporated RESCUEicp data + DECRA 12-month data.

- both RESCUEicp and DECRA are class 1 studies.

  N.B. these two ***studies cannot be compared*** as they used different outcome cut-points to define good vs unfavorable outcomes!

- both RESCUEicp and DECRA studied secondary craniectomy:

	DECRA	RESCUEicp
Timing of surgery	**Early** - ICP > 20 mmHg for 15 min over a 1-h period despite optimization of tier 1 treatments within the first 72 h	**Late (more established intracranial hypertension)** - ICP > 25 mmHg for 1-12 h refractory to 2 tiers of treatment within 10 d
Type of surgery	Exclusively **bifrontal DC**	Mostly **bifrontal DC**
Outcomes	Mortality the same but more worse outcomes and less good outcomes with early DC	Improved mortality and favorable outcomes with late DC

	Mortality DC vs medical management 6 mo. 19% vs 18% 12 mo 21% vs19%	Mortality: DC vs medical management 6 mo 16.9% vs 48.9% 12 mo 30.4% vs 52.0%

DECRA allowed fixed pupils (RESCUEicp – did not) = "DECRA operated unsurvivable and also too mild cases"

My thought: Surgery is good but not for everyone, only for the sickest but still rescuable!
DC has risk of complications (up to 50%)

New Level IIA recommendations
1. Secondary DC performed for LATE refractory ICP elevation is recommended to improve ***mortality and favorable outcomes***.
2. Secondary DC performed for EARLY refractory ICP elevation is not recommended to improve mortality and favorable outcomes.
N.B. recommendation #2 should not be extrapolated to primary DC.
3. A **large frontotemporoparietal DC (12 × 15 cm or 15 cm in diameter)** is recommended over a small frontotemporoparietal DC for reduced mortality and improved neurological outcomes.
4. Secondary DC (either EARLY or LATE) is suggested to ***reduce ICP and duration of ICU care***, though relationship between these effects and favorable outcome is uncertain.
Removed old recommendation: "***Bifrontal DC is not recommended***"

- diffuse brain edema with no mass lesion or midline shift:
  a) nondominant hemisphere unilateral DC
  b) bifrontal DC.

In rapidly deteriorating patient with acute SDH/EDH, immediate temporal decompression with temporal burr hole

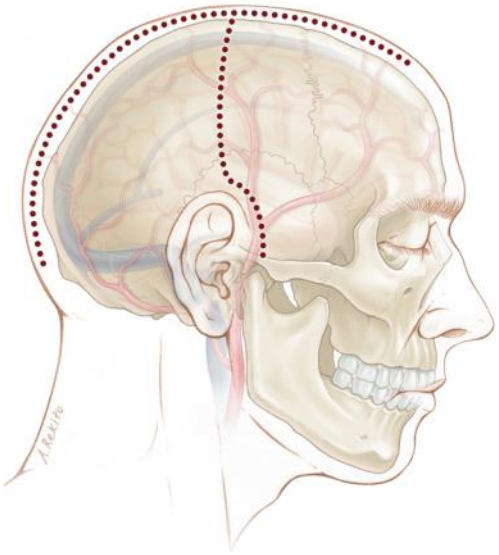
dural onlay substitutes (e.g. DuraGuard) to cover entire craniectomy defect (not just dural gaps) – prevents dura scarring to scalp flap; some recommend to close dura well!!! (to prevent CSF leak once edema subsides)
at least two Jackson-Pratt drains (without tamponading effect provided by bone flap, risk of EDH is high)
bone flap into freezer / abdomen - *in left lower quadrant* to avoid contamination by PEG tube placement and to decrease confusion with appendectomy scar

- after DC, new ICP treatment threshold is > 15 mmHg.
- protective **helmet** if out of bed and a **sign at the bedside** "No bone flap".
- CSF is normally absorbed passively due to pressure gradient, at least 5 mmHg, across arachnoid granulations, which is lost after craniectomy.

Boards: huge bleed with nonreactive pupils – either no surgery or osmotic load (if improves → DC)

HEMI DC

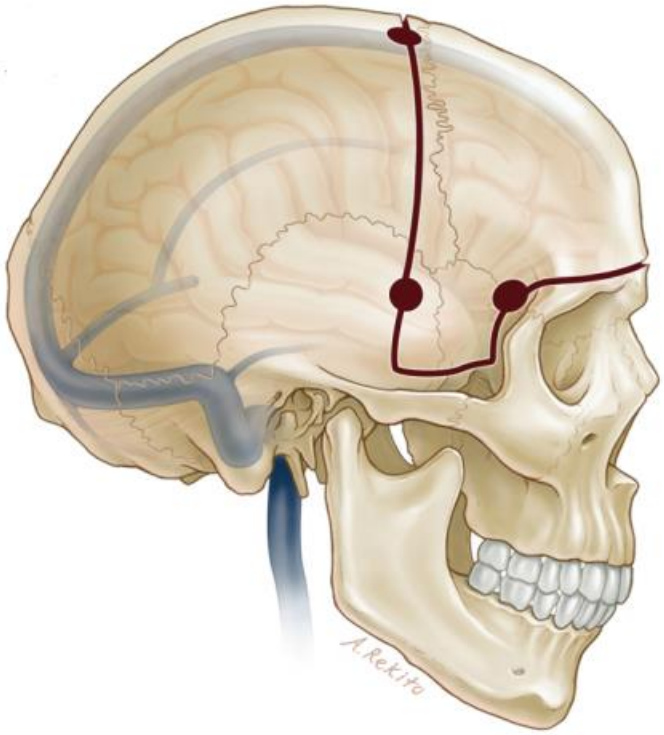
Ludwig G. Kempe hemispherectomy incision (midline sagittal incision with "T-bar" extension)
- spares STA, posterior auricular and occipital arteries, unlike large reverse question mark

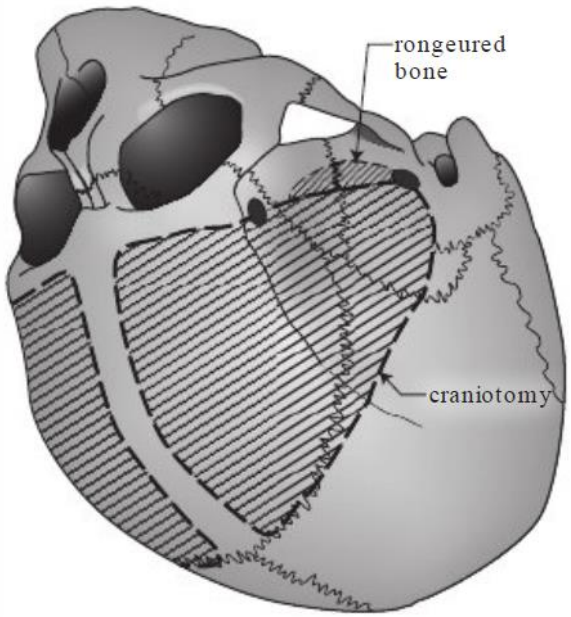


- bur holes placed at:
    1) **pterion or Dandy keyhole** (exposing frontal and temporal dura) – absolutely necessary
    2) **just above posterior root of zygoma** – absolutely necessary
    3) All other burr holes are optional!
- create bone flap 12 cm × 15 cm - use ruler to measure back from keyhole
- flap should extend 1.5 cm from midline, 1-2 cm above transverse sinus – to decompress vein of Labbé, parasagittal bridging veins.

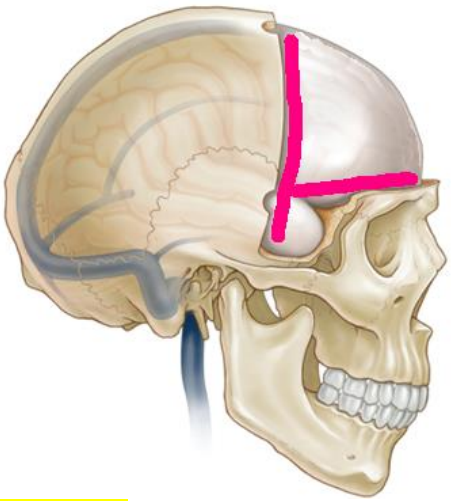
BIFRONTAL DC (KJELLBERG)

- bicoronal (Souttar) incision; Dr. Villanueva uses wavy (vs. straight) incision – scar does not interfere with chewing.
- bur holes are placed in:
    1) pterion (keyhole)
    2) root of zygoma / 2-3 cm below temporalis insertion
    3) over superior sagittal sinus (last bur hole to make) – if a large single bone flap is planned*; cross the superior sagittal sinus with the footplate last
        *posteriorly – 2 cm behind coronal suture!!! (Dr. Villanueva extends even further to parietal bossings – the adequate decompression of sagittal sinus); alternatively, strip of bone can be left over sagittal sinus for protection (N.B. if midline bone strip is too wide, it can damage brain)

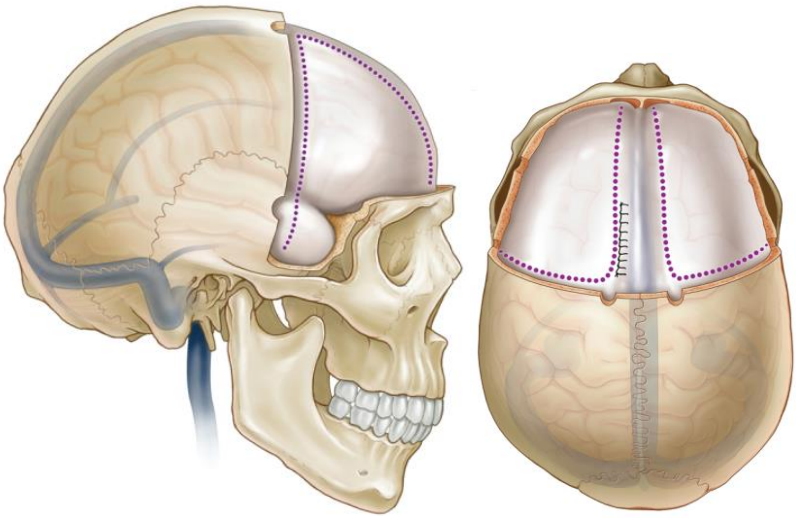




- dissect dura off with Penfield #3 (esp. if making cut over SSS).
- immediately after removing bone put lap pad with H_2O_2 over entire dura – air embolism prophylaxis (in case SSS was damaged) plus hemostatic.
- no need to go low to enter frontal sinus (if frontal sinus is entered, it must be cranialized - dissect pericranium to cover it).
- division of the anterior superior sagittal sinus and falx is crucial!!!
- dural cuts:
  a) standard Kjellberg open fish-mouth cuts made along floor of anterior fossa with release of inferior aspect of interhemispheric falx and then curved up (along posterior bone edge) towards SSS:



  b) mitral valve–type dural incisions - parallel to SSS and parallel to posterior bone edge (some recommend connecting bilateral incisions along floor of anterior fossa with release of inferior aspect of falx):



MALIGNANT CEREBRAL EDEMA

- ominous sign! → external herniation
- after period of hypotension / hypoxia – maximally dilated paralyzed brain vessels.

Management - see 00. Condensed (last minute) read >>

GSW

- antibiotics + tetanus + seizure prophylaxis!!!
- rapid gentle local debridement of devitalized brain.

N.B. retained fragments have not been associated strongly with infection, most authors *remove fragments only if they are accessible*!

watertight closure

N.B. **in the ABSENCE OF SIGNIFICANT MASS EFFECT**, surgical debridement of missile track in the brain is not recommended; routine surgical removal of fragments lodged distant from entry site and reoperation solely to remove retained bone or missile fragments are not recommended.

- first surgery - "damage control" - quick removal of hematomas (leave small amount along SSS), parenchymal debridement without overly aggressive pursuit of deep and small fragments, place EVD, and quick decision for decompressive craniectomy (GSW tend to swell!!! – absolute majority need early decompression).
- ***scalp incision*** (during primary craniotomy) is better to locate away from penetration wound so that least possible scarring will overlie site of any future cranioplasty; **Kempe incision** is the first choice.
- **DSA/CTA** ASAP, latest at 1-2 weeks (pseudoaneurysm? – small can be observed).
- second or third operations are sometimes necessary for further debridement of necrotic brain tissue; deep imploded bone fragments and foreign bodies would often deliver themselves to surface at this time (ultrasound is helpful in finding).
- cranioplasty is delayed for ≥ 1 year (when risk of infection is low).

See T3 case >>

CRANIOPLASTY

a) ***previous infection*** – 6 months should pass after all infection has cleared.
   - ***military penetrating injury*** – 12 months

b) ***no previous infection*** – after brain edema subsided (usually > 6 weeks – incision must be healed; skull edges ossify – less risk of autologous bone flap resorption);

- check if CTA was ever done (for pseudoaneurysms after GSW).
- timing of cranioplasty:
  within 10 weeks – infection↑
  > 10 weeks – communicating hydrocephalus, ↑risk of EDH postop

SEATTLE INTERNATIONAL SEVERE TBI CONSENSUS CONFERENCE (SIBICC) – SEVERE TBI MANAGEMENT ALGORITHM (2019)

Adapted thresholds: **ICP** - 22 mmHg, **CPP** - 60 mmHg

TIER-ZERO

Tier Zero (Basic Severe TBI Care - Not ICP Dependent)

Expected Interventions:

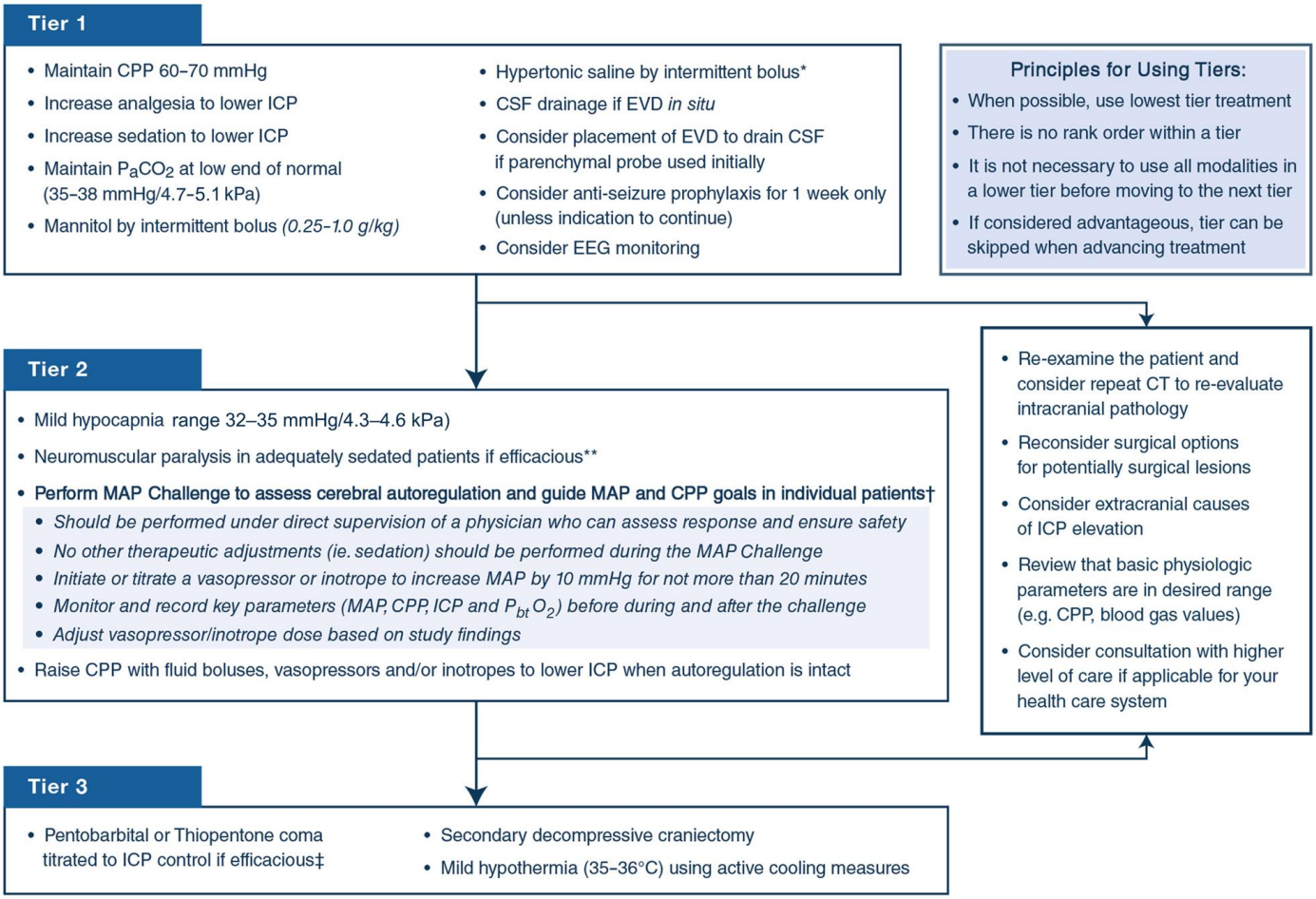
- Admission to ICU
- Endotracheal intubation and mechanical ventilation
- Serial evaluations of neurological status and pupillary reactivity
- Elevate HOB 30-45°
- Analgesia to manage signs of pain (not ICP directed)
- Sedation to prevent agitation, ventilator asynchrony, etc. (not ICP directed)
- Temperature management to prevent fever
  Measure core temperature
  Treat core temperature above 38°C
- Consider anti-seizure medications for 1w only (in the absence of an indication to continue)
- Maintain CPP initially ≥ 60 mmHg
- Maintain Hb > 7g/dL
- Avoid hyponatremia
- Optimize venous return from head (eg. keeping head midline, ensure cervical collars are no too tight)
- Arterial line continuous blood pressure monitoring
- Maintain SpO2 ≥ 94%

Recommended Interventions:

- Insertion of a central line
- End-tidal CO_2 monitoring

- goal of Tier-zero is to establish a stable, neuroprotective physiologic baseline regardless of eventual ICP readings.
- sedatives and analgesics target comfort and ventilator tolerance rather than ICP.
- temperature management targets avoiding fever (> 38 °C).

TIERS 1-3 WHEN ONLY ICP MONITOR IS PRESENT



* We recommend using sodium and osmolality limits of 155 mEq/L and of 320 mEq/L respectively as administration limits for both mannitol and hypertonic saline.
** We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion when efficacy is demonstrated.
† Rosenthal G. et al 2011
‡ Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated.
Titrate barbiturate to achieve ICP control but do not exceed the dose which achieves burst suppression.
Hypotension must be avoided when barbiturates are administered.

Tier 1:

2) ↑analgesia & sedation
3) CSF drainage
4) normocarbia (35-38)
5) osmotherapy
   - no recommendation for one hypertonic solution over the other.
   - recommendation for using same upper limits for serum sodium and osmolality for both agents

Tier 2:

1) **neuromuscular blockade**
2) **mild hypocarbia** (32-35) - caution with even mild hyperventilation when brain oxygenation monitoring is not employed!
3) **MAP challenge** – test if static pressure autoregulation (sPAR) is intact* vs disrupted:
   *then use pharmacological MAP augmentation
   - no other active changes in care during challenge.
   - use vasopressor to increase MAP by 10 mmHg for up to 20 min.
   - observe CPP, ICP (and PtbO2).
   - disrupted sPAR will present as a sustained increase in ICP with MAP elevation.
   - adjust the target MAP back to baseline (disrupted sPAR) or to new, elevated target (intact sPAR).

Tier 3: Greatest associated risks!

1) **mild hypothermia** (35–36 °C)
2) **high-dose barbiturate** - based on ICP and EEG response to a test dose.
   – do not increase dose if burst suppression occurs, as further reduction in ICP is not anticipated and toxicity increases (esp. hypotension);
   – endpoint of barbiturate treatment is ICP control not serum levels or EEG response.
3) **decompressive craniectomy**

TIERS 1-3 WITH BOTH ICP AND BRAIN OXYGEN MONITORS

	ICP < 22 mmHg	ICP > 22 mmHg
$P_{bt}O_2$ > 20 mmHg	Type A	Type B
$P_{bt}O_2$ < 20 mmHg	Type C	Type D

Type B (↑ICP) - hyperventilation to hypocarbia 30-32

Type C (↓brain oxygen) – increase 4 parameters: oxygen (PaO2, Hb) + delivery (PaCO2, CPP)

Type D – as Type C, except two things: keep normocarbia, CPP↑ needs MAP challenge

More elaborate

Type B (elevated ICP, normal PbtO2)
Tiers 1-3 same as with ICP monitor only, except Tier 3 allows hyperventilation to hypocarbia 30-32

Type C (normal ICP, low PbtO2) – increasing 4 parameters: CPP, PaO2, PaCO2, Hb + consider ICP target < 22

Tier 1:
1) increase CPP to 70
2) keep normocarbia > 35
3) increase FiO2 to 0.6

Tier 2:
1) increase CPP > 70
2) increase PaO2 to 150

Tier 3:
1) induce hypercarbia 45-50
2) increase PaO2 > 150 (normobaric hyperoxia)
3) if Hb < 9, transfuse 1 unit of pRBC

Type D (high ICP, low PbtO2) – combination of Type B and C, except keep normocarbia (avoid hypocarbia – induces ischemia; avoid hypercarbia – worsens ICP)

TREATMENT NOT RECOMMENDED FOR SEVERE TBI

1. Mannitol by continuous (non-bolus) IV infusion
2. Scheduled infusion of hyperosmolar therapy (e.g. every 4–6 h)
3. Lumbar CSF drainage
4. Furosemide
5. Routine use of steroids
6. Routine use of therapeutic hypothermia < 35 °C (due to systemic complications)
7. Routinely decreasing PaCO2 < 30 mmHg
8. Routinely raising CPP > 90 mmHg
9. High-dose propofol to attempt burst suppression
10. Barbiturates as treatment for low PbtO2 (unless barbiturates are otherwise indicated)
11. Hypothermia as treatment for low PbtO2 (unless hypothermia is otherwise indicated)
12. Hypercarbia in "type D" patients (high ICP + low PbtO2)

SAFETY OF SEDATION HOLIDAY

Condition: ICP "acceptable" for ≥ 24 hours with ongoing treatment.
Factors that matter:

1) duration of "acceptable" ICP
2) amount of treatment that is needed (tiers used)
3) pupils - normal (NP) vs abnormal (AP)
4) GCS motor score
5) Marshall Score on the most recent CT

Goal of sedation holiday – to determine if sedation can be tapered (favorable exam) vs new deficits requiring investigation

SAFETY OF ICP MONITOR REMOVAL

Condition: patient is no longer receiving ICP treatment.
Factors that matter:

1) duration of "acceptable" ICP
2) amount of treatment that was needed (tiers used)
3) pupils - normal (NP) vs abnormal (AP)
4) GCS motor score
5) Marshall CT Head Score

Most experts agree that **72 h of acceptable ICP** is safest; whereas removal at 24 h is recommended only if fairly benign CTs and favorable exams.

PROGNOSIS

Poor prognosis risk factors

1. TBI degree↑
2. Age↑
3. Fixed dilated pupil
4. Hypotension (at any time)
5. Abnormal CT (esp. obliterated basal cisterns, midline shift, tSAH).
6. Penetrating TBI (vs. closed TBI – mortality 2.5 times lower), esp. gunshot TBI.
7. Multiple organ injuries - primarily via hypotension.
8. Anticoagulants / antiplatelets (esp. Aspirin).
9. Presence of even one of *APOE4* alleles - 14-fold greater likelihood of poor outcome.

Specific for **penetrating injuries**

1. **Suicide** correlates with a higher rate of mortality.

N.B. patients who recover from attempted suicide frequently report relief over survival and express gratitude to their caregivers!

2. **Perforating** injuries correlate with a poorer outcome when compared with either penetrating or tangential brain injuries (Class III).
3. **Intraventricular hemorrhage** is strongly correlated to increased mortality (Class I) - odds ratio 2.83-96.9; **Subarachnoid hemorrhage** is also correlated to increased mortality (Class I) - odds ratio 1.44-10.6
4. Trajectory:
   Bihemispheric injuries relate to increased mortality (Class II) - odds ratio 1.18-20.05; possible exception may be bifrontal injuries
   Multilobar (> 1 lobe) injuries are strongly associated with mortality (Class III) - odds ratio 3.27-84.4, negative predictive value for mortality 77-98%.
   Injuries with **ventricular** involvement have an increased mortality (Class III) - odds ratio 3.35-27.5

GLASGOW OUTCOME SCALE (GOS)

Jennett B , Bond M . Assessment of outcome after severe brain damage. Lancet 1975 ; 1 : 480 – 484

5 – **GOOD RECOVERY** - normal life despite minor deficits

4 - **MODERATE DISABILITY** - disabled but independent; can work in sheltered setting

3 - **SEVERE DISABILITY** - conscious but disabled; dependent on others for daily support

2 - **VEGETATIVE** - no evidence of meaningful response

1 - **DEAD**

GOS can be divided further into:
good outcomes (good, moderate disability, **independent**): 5 and 4
poor outcomes (severe disability, vegetative, dead): 1-3

See T1 case >>
See T2 case >>
See T5 case >>

SKULL FRACTURES

By convention, **open depressed** cranial fractures are treated **surgically** (early debridement and elevation), primarily to decrease the incidence of infection.

However, at least a select group of patients with compound depressed cranial fractures will do well without surgery.

Depressed fractures

1. Prophylactic **anticonvulsants**.
2. Most depressed fractures heal well and smooth out with time, without elevation; indications for **surgical elevation**:
   a) ***depressed greater than cranium thickness*** (i.e. > 10 mm inward displacement)
   b) ***focal neurologic deficit*** (but focal deficits are caused by brain parenchyma damage more than by continuing compression by bone fragments; i.e. compression relief does not guarantee deficit disappearance).
      depressed fracture over venous sinus: neurologically stable patient → observe (or primary wound debridement without elevation); neurologically unstable patient → urgent elevation (prepare for sinus repair).
   c) ***cosmetic deformity*** (FRONTAL BONE is most important esthetically + it forms roof and portions of medial and lateral walls of orbit).

- ***burr hole*** near fracture (and over intact dura) → bony fragments are elevated → soaked in antibiotic
- ***dural tears are repaired***.
- ***bony fragments are reassembled*** - no difference in infection rate *(vs titanium mesh)*

Joker periosteal elevator for ping pong fracture* (if that fails – extend incision across entire fracture; make circular craniotomy along fracture line; flip bone flap).
*may place temporary cranial screw through the middle of fracture to assist with grasp for elevation

Open fractures – just standard periop abx prophylaxis (even if enters sinus)! Other say for 7-10 days!

See T6 case >>

LEPTOMENINGEAL CYST (S. GROWING FRACTURE)

– extrusion (in form of cyst) of leptomeninges and brain tissue through dural defect.

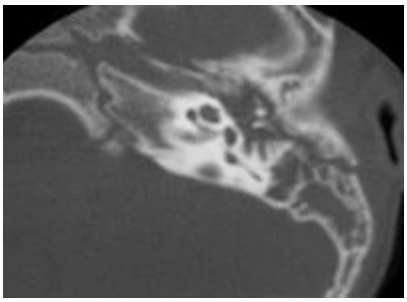
- etiopathology: *skull fracture with separation of fracture edges* [depressed or diastatic skull fracture] *and dura laceration* → arachnoid and brain are caught between edges of fracture → brain pulsation forces CSF into cyst → skull erosion.
  N.B. seen almost exclusively in children < 1-3 yrs with fracture accompanied by dural tear – such children must be followed up closely for several months!!!
- treatment: **cyst excision** +/- resect seizure foci → **dural closure** → **cranioplasty**.

Avoid shunt!

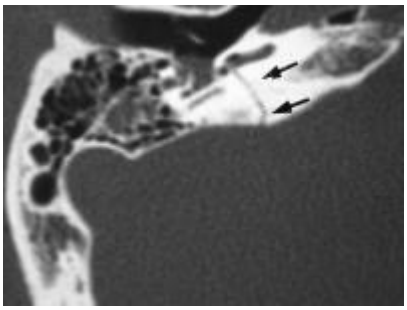
TEMPORAL BONE FRACTURES

- high-resolution **CT**

1. ***LONGITUDINAL*** *(70-90%)* - parallel to petrous pyramid:
   - pars squamosa, posterosuperior wall of external auditory canal, tegmen tympani → run either anterior or posterior to cochlea and labyrinthine capsule → end in middle cranial fossa near foramen spinosum or in mastoid air cells, respectively.
   - caused by direct lateral force over mastoid or squamous bone or blow to mandible.



2. ***TRANSVERSE*** *(5-30%)* - perpendicular to petrous pyramid:
   - originate at foramen magnum → extend through cochlea and labyrinth (pneumolabyrinth) → end in middle cranial fossa.
   - caused by frontal or parietal blow but may result from occipital blow.



TRANSVERSE FRACTURES nearly always produce *facial paralysis*, permanent *hearing loss*, severe ablative *vertigo*.

CN7; ***facial palsy*** may be delayed 5-7 days (progressive edema within nerve - good prognosis).

- ***delayed-incomplete*** – due to neurapraxia (10-20% longitudinal fractures); injury site is usually horizontal segment distal to geniculate ganglion; H: **steroids** - spontaneous recovery is usual.
- ***immediate-complete*** – due to nerve transection (50% transverse fractures); injury site is anywhere from internal auditory canal to horizontal segment distal to geniculate

ganglion; **decompression surgery** is not always indicated (use electroneuronography [ENOG] in decision making).

CN8 (nerve laceration in 80% *transverse* temporal fractures) → ***sensorineural hearing loss***, ***vertigo***, positional ***nystagmus*** immediately after injury.

- patients with low- **or** high-frequency hearing loss may have some recovery but those with low- **and** high-frequency loss usually do not recover.
- vertigo due to labyrinth concussion usually resolves within year.

a) ***conductive*** **hearing loss** due to hemotympanum, ossicular dislocation / fracture or tympanic rupture (≈ 50% longitudinal fractures);
   - hemotympanum and mucosal edema in middle ear may cause temporary deafness - resolves within ≈ 3 weeks.
   - most tympanic membrane perforations and hemotympanum usually resolve in 3-4 weeks.
   - if conductive hearing loss is present at > 30 dB after 3 months → **tympanoplasty with ossicular chain repair**.

b) ***sensory*** **hearing loss** (≈ 80% transverse fractures); H: **cochlear implants**.

OCCIPITAL CONDYLAR FRACTURES

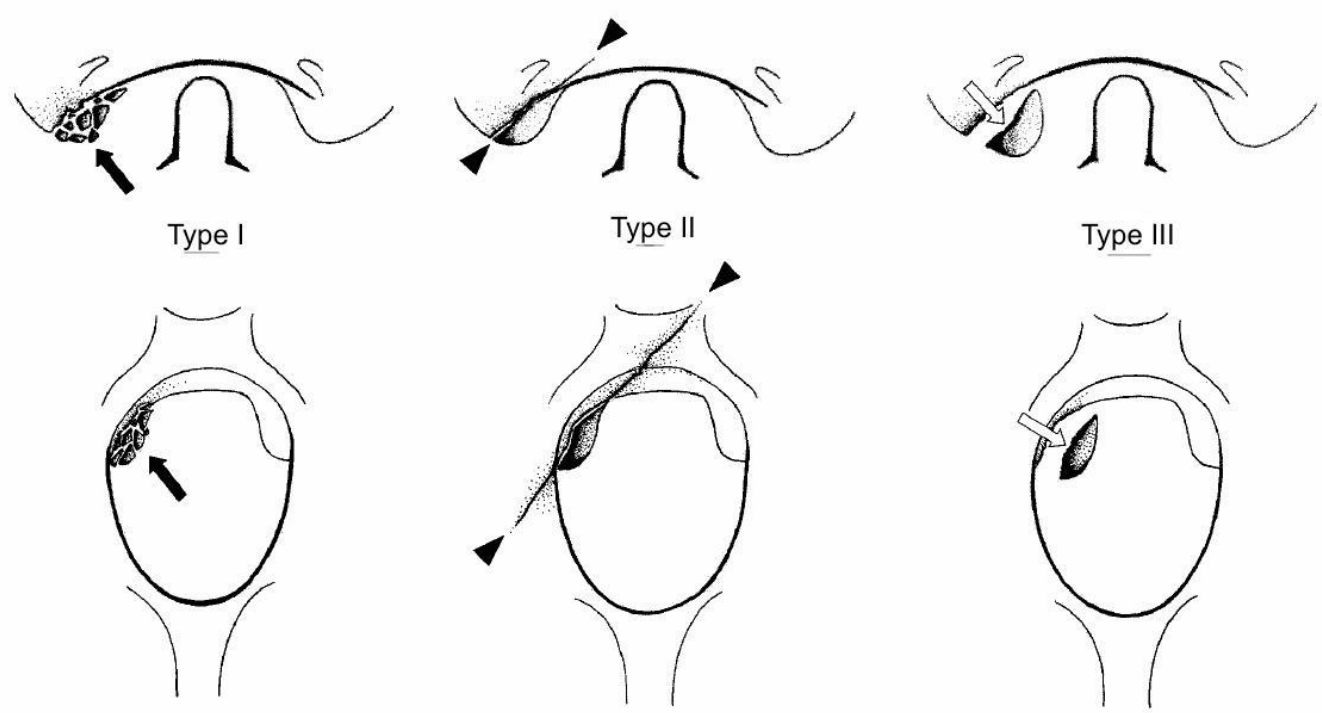
ANDERSON AND MONTESANO TYPES

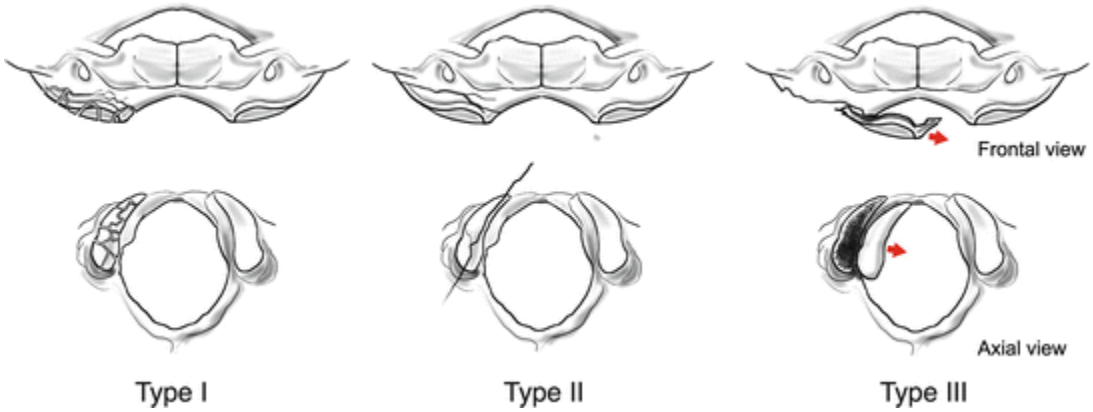
*preserved **alar ligament** and tectorial membrane

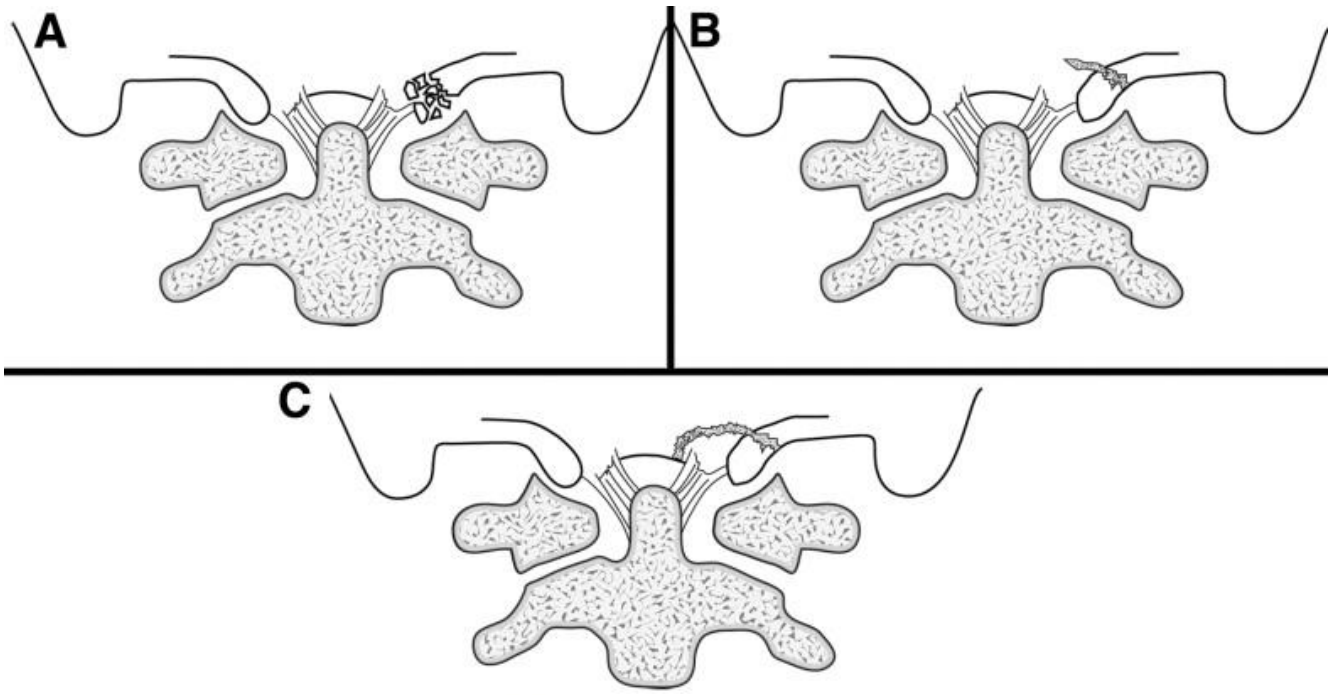
Type I fracture – stable* **comminuted** (**impacted**) fracture of occipital condyle - due to axial compression injury.

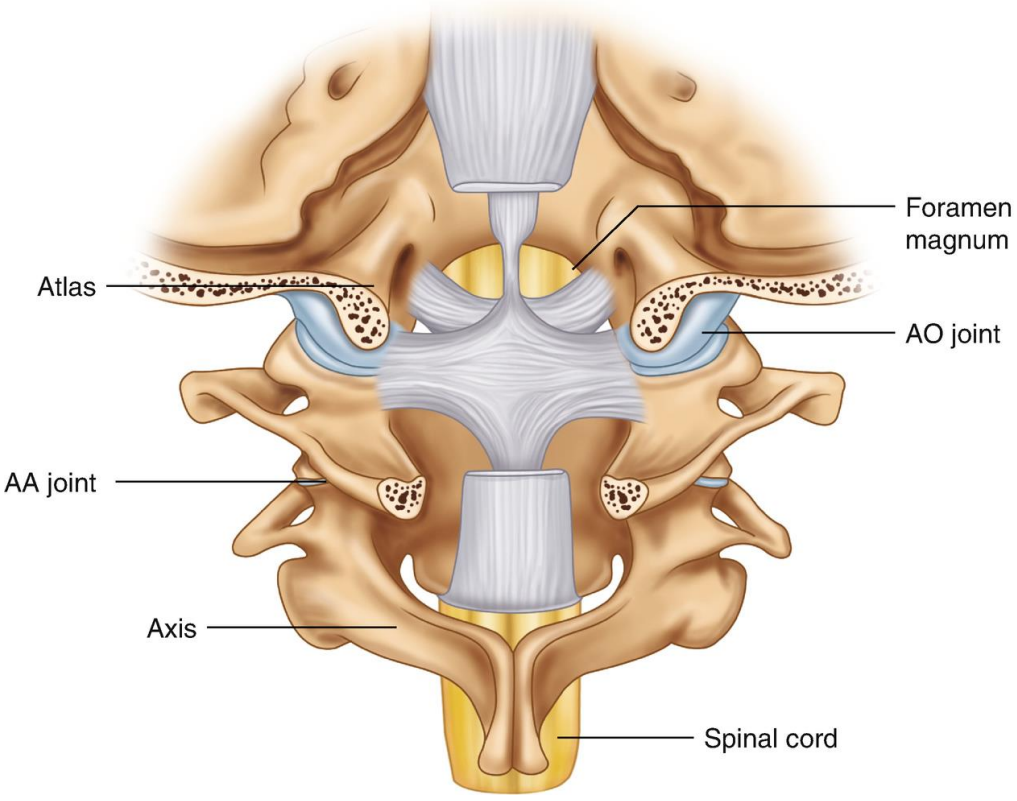
Type II fracture – stable* **extension of fracture of basioccipital region** - caused by direct blow.

Type III fracture – unstable **avulsion** injury (alar ligament insertion avulsed from occipital bone), AO ligamentous injury - due to forced rotation and lateral bending.




Frontal view
Axial view
Type I
Type II
Type III


A
B
C

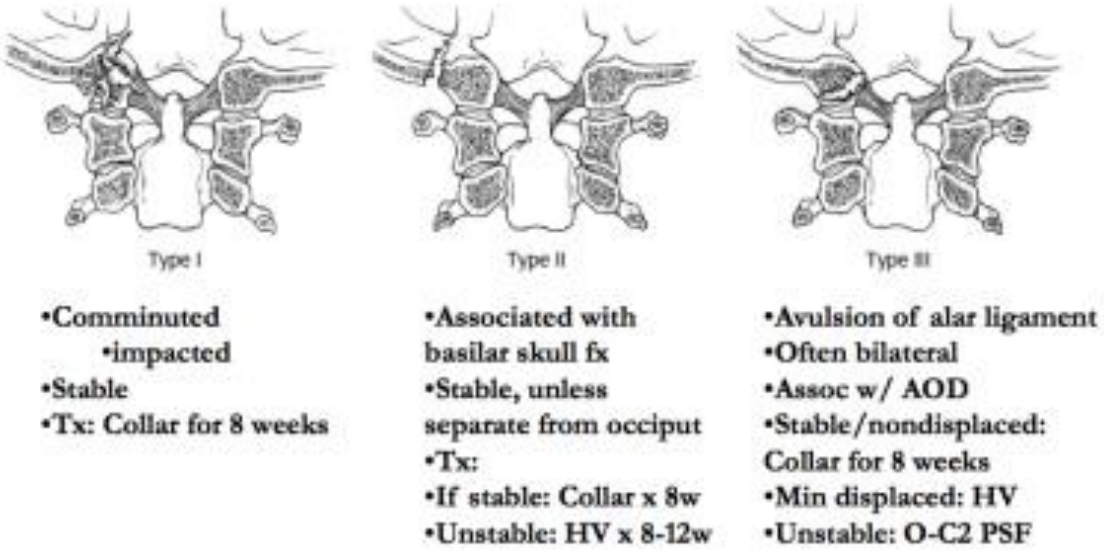

Foramen magnum
Atlas
AO joint
AA joint
Axis
Spinal cord

CLINICALLY

Complications: **CN9-12 palsy** (***Collet-Sicard syndrome***), **CN9-11 palsy** (***Vernet syndrome***).

TREATMENT

(CNS/AANS Guidelines):
Types I-II - neck stabilization with hard collar or halo (for ***bilateral*** OCF).
Type III - halo or occipitocervical fusion; anecdotally, patients (esp. young ones) heal in hard collar.



HEMATOMAS

Rule of thumb: blood remains ***denser*** than brain for 1 week, and is ***less dense*** after 3 weeks.

- causes of ***hematoma density***↓: *severe anemia*, *hyperacute hematoma* (no clots at all).

Do not state "SDH" or "EDH" – better say "Extra-axial hematoma"

If CT is unavailable – side of surgery is chosen:

Feature	Hematoma Side
First (or only) dilated pupil	Ipsilateral 94%
Most abnormal motor response	Contralateral 82%
Skull fracture	Ipsilateral 66%

First burr hole – at root of zygoma

- tack-up sutures 4-0 silk around craniotomy perimeter + in the center of bone flap ← postop EDH prophylaxis.

EPIDURAL HEMATOMA

– 60% patients are < 20 yrs (but rare in children < 2 yrs*).
– only < 10% patients are > 50 yrs; rare at age > 60 yrs** (vs. SDH!)
*very elastic immature skull rarely fractures

**as person ages, dura becomes more adherent to skull

a) 36-85% cases - high-pressure **arterial** bleeding from lacerated **meningeal artery** (most commonly middle meningeal artery)

b) 15-32% cases - bleeding is **venous** (torn **dural sinuses**, **diploic veins**, **meningeal veins**) - only with *depressed fractures*

EDH needs direct blow to the head!
Delayed (subacute, chronic) EDH may develop as result of temporary *INTRACRANIAL HYPOTENSION* (but, more commonly, SDH)

- body has no mechanism for absorption of extradural hemorrhage - clotted blood remains in epidural space as tumor.

≈ 47% (10-50%) demonstrate classic **LUCID INTERVAL**

N.B. posterior fossa EDH may have dramatic rapid delayed deterioration - patient can be conscious and talking and minute later apneic, comatose, and minutes from death.

Indications for surgery

- anisocoria, GCS < 9 → immediate surgery
- EDH > 30 mL → surgery regardless of GCS score.
  75 ml is critical EDH volume – any volume above → loss of consciousness.
- EDH < 30 mL and < 15 mm thickness and < 5-mm midline shift and GCS score > 8 and no focal deficit - can be managed nonoperatively with serial CT* and close neurological observation in a neurosurgical ICU. *first routine repeat CT within 6 hrs after TBI

Most dangerous EDH (likely will need surgery):
1) location – middle fossa (temporal location), posterior fossa
2) volume > 20 cm^3
3) hyperacute (on CT)
4) associated fracture

N.B. criteria for SDH are slightly different: in SDH, parenchymal injury plays bigger role, so it is ***managed by ICP criteria*** – even smaller SDH may contribute a lot to ICP: evacuate > 10 mm SDH or > 5 mm midline shift or refractory ICP (also, of course, anisocoria, GCS < 9 or drop ≥ 2 points)

See T4 case >>

SUBDURAL HEMATOMA

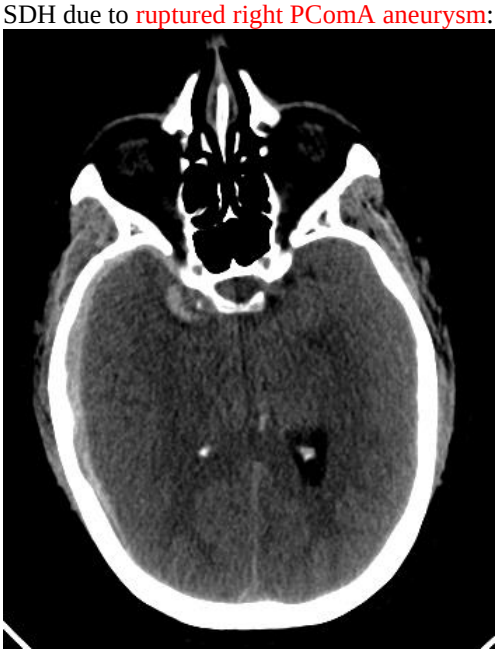
- most patients > 70 yrs.
- more common than EDH.
- commonly (> 50%) associated with *extensive primary brain injury*!!! (vs. EDH) - play major role in outcome!!!
- Suspect child abuse! (esp. if posterior interhemispheric & tentorial SDHs)

Nonepileptic, Stereotypical, and Intermittent Symptoms (NESIS)

- NESIS clinically manifests (vs. seizures) as negative symptoms lasting > 5 minutes, dysphasia, preserved awareness, lack of positive symptomatology (such as clonic movements), lack of response to AEDs.
- possible pathophysiology – ***cortical spreading depression or depolarization (CSD)***

Absence of clear history of trauma → **angiography** (search for ruptured aneurysm or dural AV fistula).

SDH due to ruptured right PComA aneurysm:



In all cases, hematoma *complete resolution* should be documented (conservatively treated acute SDH can evolve into chronic SDH).

ACUTE SDH

After evacuation of traumatic SDH, place EVD if preop GCS was ≤ 8

Guidelines "Surgical management of acute subdural hematomas" in Neurosurgery. 2006 Mar;58(3 Suppl):S16-24

- indications for ASAP* surgery: also see p. TrH1 >>
    a) acute SDH with a thickness > 10 mm, regardless of the GCS score
    b) midline shift > 5 mm, regardless of the GCS score.
    c) GCS < 9 and GCS decreased between the time of injury and hospital admission by ≥ 2 points
    d) GCS < 9 and asymmetric or fixed and dilated pupils
    e) GCS < 9 and refractory ICP > 20 mm Hg

N.B. criteria for EDH are slightly different: in EDH, parenchymal injury is absent, so it is ***managed by focal mass effect criteria***: evacuate > 15 mm EDH or > 5 mm midline shift or > 30 mL (also, of course, anisocoria, GCS < 9)

CHRONIC SDH

If patient is minimally symptomatic (e.g. headaches) – wait until hematoma liquefies (10-14 days)
After surgical evacuation, subdural hematoma recurs in 10-20% patients.

Close VPS valve (if present)

- postoperative seizures are reported in 3-10% patients (many surgeons use prophylactic AED).

N.B. there is no difference in recurrence (6.4-8.4%) or outcomes with drainage for 1 or 2 days

N.B. there is no difference in recurrence rates (7.7-9.1%) and outcomes with **subdural** vs **subgaleal** drains but **subgaleal** drains have reduced risk of infection, intracranial hemorrhage, brain injury, empyema, and epilepsy.
Drain suction creates negative intracranial pressure → bradycardia, lightheadedness

1. Chronic SDH can be treated with TRANEXAMIC ACID (TXA) 750 mg/d PO for 30 days
2. Dr. Okonkwo (UPMC) gives MEDROL DOSEPAK; Dr. Day (UAMS) gives DEXAMETHASONE 2 mg q8hrs for 2 weeks, then tapers based on repeat CT findings.

Dex-CSDH Trial

Outcome	Dexamethasone	Placebo
Favorable outcome	83.9%	90.3%
Favorable outcome in never operated patients	82%	100%
Adverse events	10.9%	3.2%
Any infection	6.4%	1.1%
Repeat surgery	1.7%	7.1%

3. Recurrent (or new in high surgical risk patients – elderly on anticoagulation) symptomatic SDH - **middle meningeal artery (MMA) embolization** with PVA particles - eliminates arterial supply to vascularized membrane.

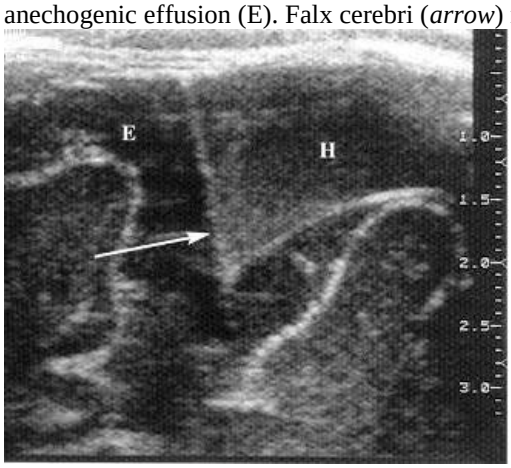
Recurrent SDH workup:
1) MRI + DWI, CTA – for ***vascular abnormalities***
2) MRI of spine – for ***occult CSF leak***

PEDIATRIC

- SDH is most common intracranial lesion in children < 2 years:
  a) ***shaken baby syndrome***!!! (chronic SDH in infants ***who do not yet walk***)
  b) complication of shunt procedures
  c) bleeding disorders.

- diagnosis in newborns – **ultrasound**, **CT**, **funduscopy** (50% show retinal or subhyaloid hemorrhages).

Ultrasound - left echogenic acute subdural hematoma (H), associated with right subarachnoid anechogenic effusion (E). Falx cerebri (*arrow*) remains straight:



Shaken baby syndrome
Retinal hemorrhages (bilateral) in absence of coagulopathy - most specific sign of shaken baby syndrome!!!;
for children < 2 years, **skeletal survey** is recommended (look for old fractures); **isotopic bone scans** may be useful.

- treatment – repeated daily **subdural taps** monitored by CT, head circumference measurements.
  - spinal needle with stylet (20 G, 1½ or 2½ inch)
  - **insert needle** through skin at extreme lateral limit of anterior fontanelle where it meets coronal suture (i.e. at least 2-3 cm from midline - to prevent sagittal sinus injury).
  - use **ZIGZAG PUNCTURE** to prevent later fluid leakage (puncture dislocated skin at right angle, then aim needle laterally).
  - cerebral cortex is ≈ 1.5 cm from skin surface (***attachment of hemostat*** 5-7 mm from beveled end of needle should provide adequate safeguard).
  - remove stylet - subdural fluid is *allowed to drain spontaneously* (fluid is never aspirated).
  - **only 10-20 mL of subdural fluid** should be removed at one time (removing larger amounts may precipitate rebleeding or shock).
  - if pial vessel is punctured, bleeding will usually cease spontaneously.
  - resorption of hematoma must occur over weeks ÷ months.

if > 10 taps are done (symptoms persist after 2 wk of daily drainage) → surgical treatment (e.g. ***subdural-peritoneal shunting*** or ***subdural-subgaleal shunting*** for 6 months)

SUBDURAL HYGROMA

- excessive CSF collection in subdural space. [Greek *hygros* – wet]

1. MOST COMMON CAUSE - **cranial trauma** with arachnoid tearing and arachnoid-dura separation (→ CSF escape into subdural space) - **TRAUMATIC SUBDURAL HYGROMA**.
2. **Complication of ventricular shunting** (esp. if overdrainage occurs)

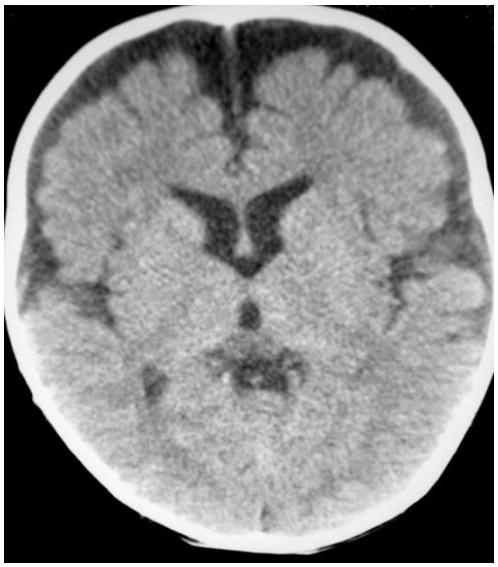
A. Spontaneous RESOLUTION.
B. Hygroma PROGRESSION: transudation / further CSF accumulation (flap-valve mechanism) → rupture of bridging veins → bleeding (well documented *transformation to subdural hematoma*) → neomembrane (capsule) formation (chronic subdural hematoma).

DIFKE

"Cortical vein sign" on gadolinium MRI - in **cerebral atrophy** (vs. hygroma) - cortical veins seen traversing widened CSF spaces over cerebral convexities

*hygroma displaces cortex and cortical veins → cortical veins do not traverse hygroma!

Benign enlargement of subarachnoid space (BESS) in child – no mass effect; resolves within first 2 yrs of life:



CAROTID-CAVERNOUS FISTULA

- A-V shunting within cavernous sinus → ophthalmic venous hypertension, cerebral venous infarction may occur.

a) **head trauma** (75-80%)
b) **spontaneous** (≈ 20%) - associated with (1)***ruptured intracavernous aneurysm***, (2)***fibromuscular dysplasia***, (3)***Ehlers-Danlos syndrome*** and other collagen vascular diseases

CLASSIFICATION

Direct type (70-90%):

Type A fistula - direct connection between **intracavernous ICA** and cavernous sinus.

- high-flow and high-pressure fistulas → fast progression of clinical features!!!
- most commonly traumatic in *young males*.

Dural types:

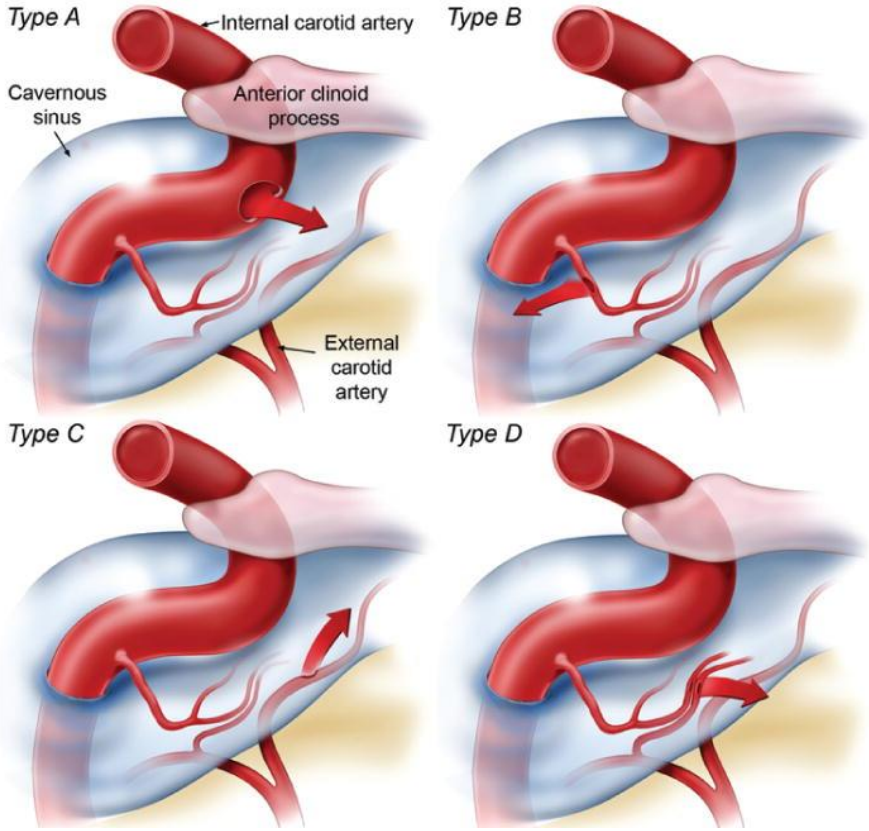
- low-flow.
- most commonly spontaneous in *women > 50 years* (7:1 female-to-male ratio).

Type B fistula - dural shunt between intracavernous branches of **ICA** and cavernous sinus.

Type C fistula - dural shunt between meningeal branches of **ECA** and cavernous sinus.

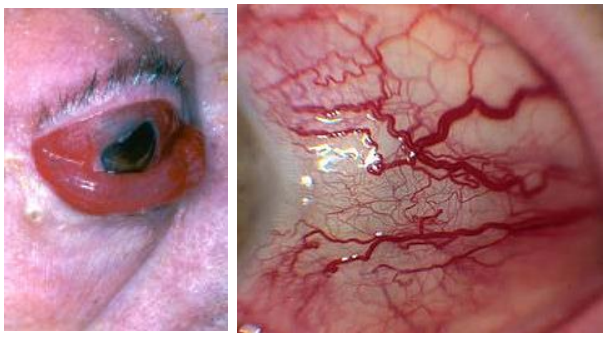
Type D fistula - **combination** of types B and C.

Diagrammatic representation of 4 types of fistulas:



CLINICAL FEATURES

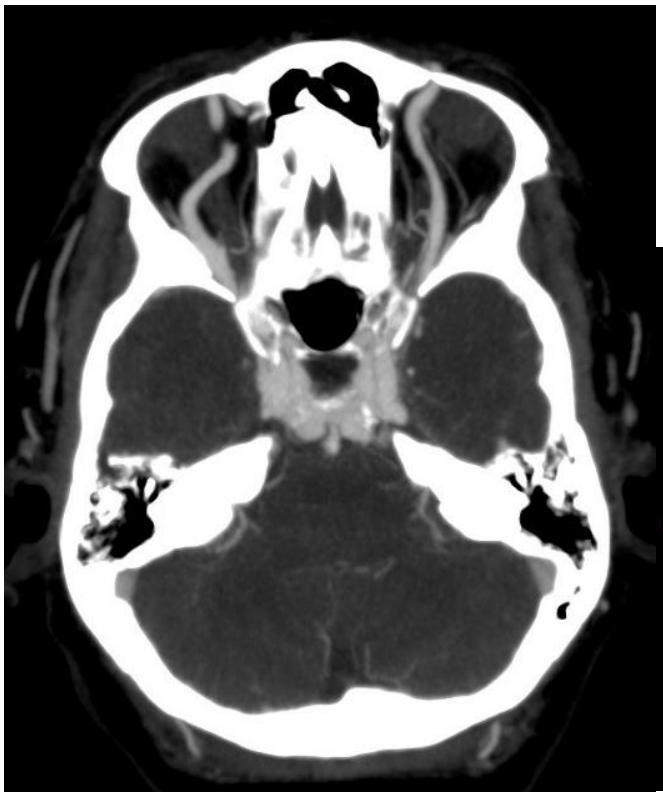
1. Ipsilateral **ocular manifestations**: pulsatile proptosis, chemosis, edema + cranial nerve palsy (III-VI), monocular **visual loss**, secondary open-angle glaucoma (ask ophthalmologist to evaluate and measure IOP!!!)



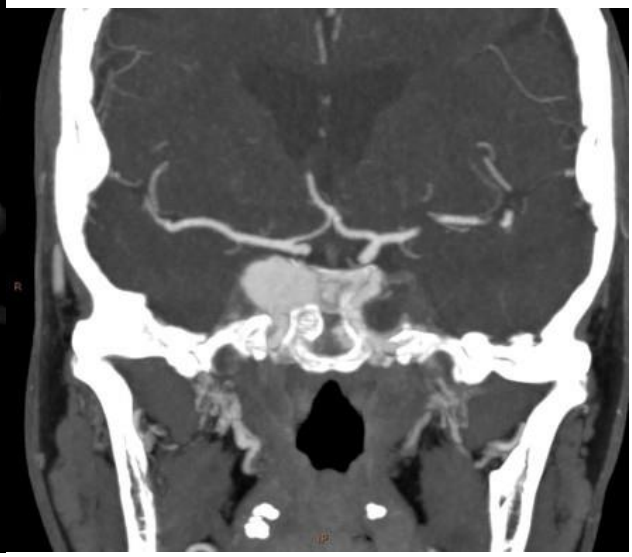
2. Audible **bruit** + **pulsatile tinnitus** (reduced by manual occlusion of carotid artery)
3. **Headache** (± other signs of ICP↑)
4. Exsanguinating **epistaxis** (H: place Foley into nose + hold manual carotid compression)

DIAGNOSIS

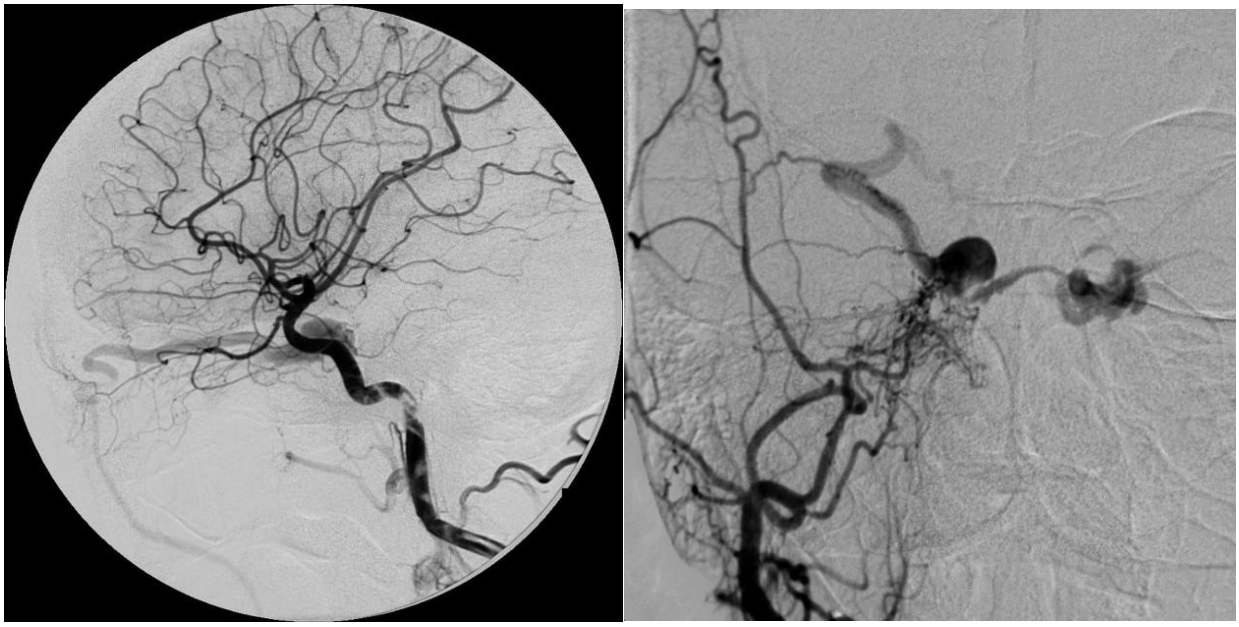
1. **CTA** - dilated ophthalmic veins*, bulging cavernous sinus, asymmetric enhancement of cavernous sinus with attenuation similar to that of internal carotid artery and higher than that of transverse sinus, contrast extravasation, intracranial hemorrhage from a ruptured cortical vein.
   *esp. superior ophthalmic vein (SOV)!
2. **ANGIOGRAPHY** - bilateral ICA and ECA* - diagnostic test of choice: early filling of cavernous sinus and its enlarged draining tributaries (esp. ophthalmic veins).
   *only for spontaneous fistulas



ICA injection (note dilated SOV):



ECA injection (note dilated SOV):



TREATMENT

Measure IOP – if > 20 → emergent treatment!
In acute setting of vision loss / CN paralysis – DEXAMETHASONE, DIAMOX
Cortical venous drainage → treat!

Type-A fistulas rarely resolve spontaneously because of high flow → **endovascular obliteration of fistulous connection** - through arterial approach (N.B. ICA hole may be big – use balloon-assisted technique!)

- severely refractory fistulas → surgical or endovascular **sacrifice of ICA** (+ clipping of supracavernous segment proximal to PComA to prevent fistula from stealing blood from cerebral vasculature).

Type B, C, D fistulas have higher incidence of *spontaneous resolution*.

- **carotid self-compression** for 20-30 seconds 4 times per hour using contralateral hand.
- venous approach **endovascular obliteration** (posterior approach via *inferior petrosal sinus* or transocular via *SOV*)

Routine ***follow-up angiogram***

PEDIATRIC TBI

More **diffuse injuries**, less **focal injuries**

- pediatric brain is more prone to *acceleration-deceleration injury* (i.e. *diffuse axonal injury* is more frequent, but *intracranial hematomas* are less frequent).

*unmyelinated brain is more susceptible to shear injuries

- children often develop **vasodilation*** in minutes ÷ hours following relatively mild head injury → rapid neurologic deterioration (mimics enlarging intracranial mass); good prognosis with control of intracranial hypertension.
- *infants can lose significant blood amount into cranial cavity*
- treatment of ICP > 20 mmHg* may be considered.

*esp. for > 5 mins; optimal ICP treatment threshold may be physiologically age-dependent (e.g. lower ICP therapeutic target for infants and young children than older children or adults)

- intracranial hypertension may be present in children with open fontanelles and sutures - ICP monitoring is of significant use in these patients.
- minimum CPP 40 mmHg may be considered in children; CPP threshold 40–50 mmHg may be considered; there may be age-specific thresholds.
- ***Level II recommendations***: HYPERTONIC SALINE 3% 6.5-10 mL/kg should be considered for severe pediatric TBI with intracranial hypertension;

As stated by FDA, continuous infusion of PROPOFOL is not recommended.
Therapeutic hypothermia - *not recommended*.
Much lower morbidity and mortality (than in adults)! – children demand aggressive approach!
N.B. *infants < 2 yrs* with severe TBI have uniformly poor prognosis

- physicians are legally mandated to report suspected child abuse.

FACIAL TRAUMA

Facial trauma frequently **compromises upper airway** (accumulation of blood, vomitus, avulsed parts, foreign substances + certain maxillofacial and mandibular fractures):

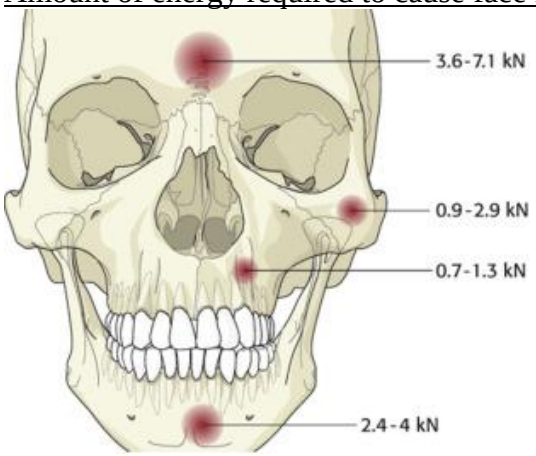
Most important aspect of (prehospital) care is maintenance of airway!

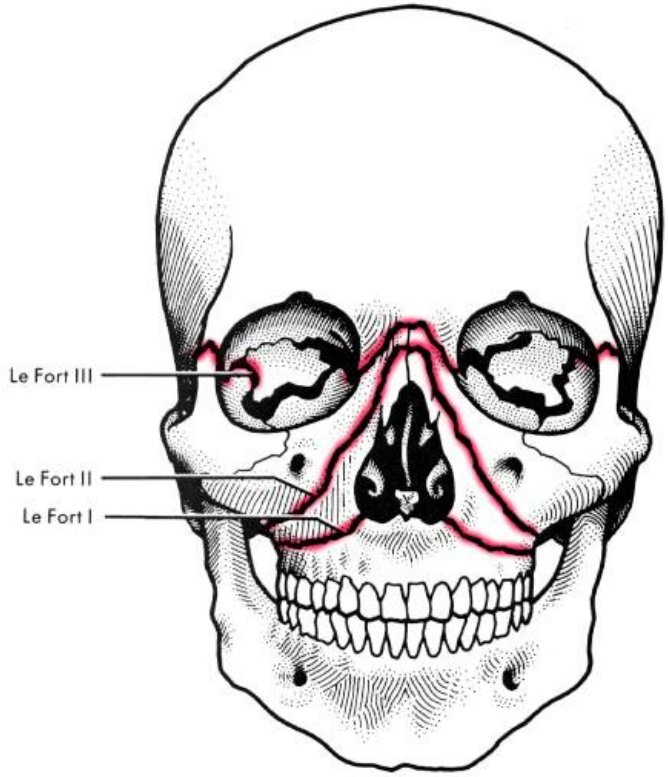
Thin-section high-resolution CT – preferred initial diagnostic study!

Weakest parts of orbit: inferior wall, then, medial wall

Entrapment of extra-ocular muscles – test with forced duction

Amount of energy required to cause face fractures:





N.B. airway compromise is possible with any of these fractures (esp. Le Fort II and III).

TIMING of operative repair

a) **EARLY** (within 24 hours of injury) - readily accomplished (because of minimal edema); indications:
   1) *large, open lacerations* (aid in exposure of fractures).
   2) *needed early neurosurgical repair* – attempt synchronous* neurosurgical maxillofacial repair (asynchronous later repair invariably disrupts neurosurgical dural repair!); intraoperative ICP monitoring is required!

*e.g. via same bicoronal flap

Mandible fracture – treatment as precise and expeditious as possible (malocclusion is major long-term complication!!! + risk↑ of osteomyelitis and nonunion by extended period without reduction and fixation)

b) **STAGED** - to wait for facial edema resolution, using time for preoperative planning, detailed radiographic analysis, production of operative aids (such as acrylic interocclusal splints); ideal window for staged repair - between 3 and 14 days; frequently performed for *cosmetic indications* or when *severe associated injuries* take priority; appropriate use of antibiotics is necessary.

PRINCIPLES of repair

N.B. in ear, nose, tarsal plate of eyelid, epinephrine is contraindicated!!!

- ***facial bones heal by fibrous union*** (not callus formation) - suture material must remain in place for long period of time (perhaps months) - until fibrous tissue is laid down and remodeled - monofilament surgical steel is ideal for its lack of elasticity.
- some severe complex injuries (panfacial fractures, gunshot wounds to upper and midface) require **postrepair tracheostomy**.

FRONTAL SINUS FRACTURES

- treated as "open fractures" (because of communication with paranasal sinuses).

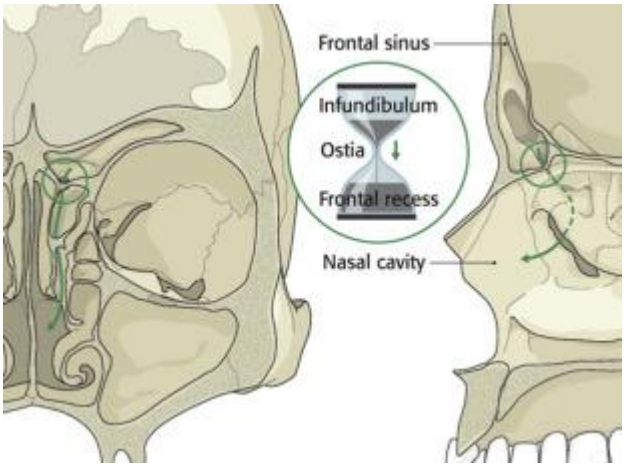
- considerable force is required to cause these fractures, and thus patients usually have other associated injuries (esp. eye injury – ophtho consult!)
- prophylactic **antibiotics**!
- close lifelong F/U - frontal sinus outflow obstruction can lead to ENT problems (chronic sinusitis, chronic pain, chronic osteomyelitis, Pott puffy tumor, mucoceles, and mucopyoceles) many years after the initial injury.

> More conservative regimen with close follow-up is becoming more mainstream for ***all types of frontal sinus fractures***

The optimal management of frontal sinus fractures remains controversial:

A. **Close life-long follow-up** (for symptoms/signs of infection or mucocele) – recent trend; counsel about the importance of follow-up* and the need to seek medical care if any concerning signs or symptoms.
   *check CT scans for restoration of sinus ventilation; if obstruction persists, endoscopic frontal sinusotomy or endoscopic Lothrop procedure.
B. More morbid and **invasive techniques** that have been the mainstay for years.

Frontal sinus drainage pathway has an hourglass configuration: infundibulum → ostium (3–4 mm) → frontal recess → small outflow tract into the ethmoid sinus/nasal cavity:



The most important goal of frontal sinus fracture repair is to create a safe sinus:

1. Reestablish **frontal bone contour** – cosmesis.
2. **Drainage system**:
   A) Restore patency (if feasible)
   B) Obliterate sinus cavity if a patent drainage system cannot be reestablished.
3. Create a **watertight barrier** between the intracranial system and nose:
   a) observation
   b) endoscopic repair
   c) open reduction and internal fixation, sinus obliteration, or sinus cranialization.

TREATMENT - ANTERIOR TABLE FRACTURES

Anterior table – stronger; only cosmetic problem! → plastic surgery

Isolated, nondisplaced (< 2 mm) anterior table fractures

- usually managed nonoperatively: nasal decongestants and sinonasal toilet.

Comminuted, depressed (2-6 mm) fractures

- either acute reduction or delayed camouflage is recommended:

a) repaired acutely within 1-10 days
b) repaired at 2 - 4 months after the injury - complete resolution of forehead edema, allowing for a more accurate evaluation of deformity.

Severely impacted fractures

- during the course of trauma to the frontal region, the frontal bone goes through a compression phase before becoming concave; the fragments need to be pulled back through the compression phase before reduction can be achieved – if fragments are unable to be elevated adequately, ***postage-stamp perforations*** can be drilled along the edges of bone, releasing the tension and reducing the interfragmentary resistance → bone hook can then be placed between the fragments to help with elevation.

Delayed presentation

- fully healed fractures are not amenable to traditional open reduction.
- if aesthetic deformity remains obvious, the defect can be camouflaged - through an endoscopic or coronal incision - ***biocompatible materials*** (titanium mesh, hydroxyapatite cement, methyl methacrylate, and polyether ether ketone implants).

TREATMENT - POSTERIOR TABLE FRACTURES

- thickness of posterior table (approx. 2 mm).
  - others believe that all posterior table fractures warrant surgical exploration to rule out dural tears and frontal sinus recess injury.
  - many investigators believe that the amount of displacement of the posterior table is not the main factor in determining whether surgical intervention is required; rather, the presence or absence of a CSF leak (intradural pneumocephalus) and the presence of absence of frontal outflow tract* injury (affected drainage → mucocele → subdural abscess) are key determinants in making treatment decisions.

*direct ostium / nasofrontal duct into middle meatus

CFS leak:

- may be observed: stool softener, head of bed elevation, and sneezing through open mouth for 1 week.
  - lumbar drain can be considered to lessen the pressure at the level of the dural tear.
  - if there is no spontaneous resolution within 1-2 weeks (posttraumatic meningitis risk), exploration with possible dural repair and/or sinus obliteration is recommended.

Frontal outflow tract injury

- surgical treatment (**frontal sinus obliteration, exenteration and cranialization**):
  - bicoronal incision → develop pericranial flap.
  - frontal craniotomy
  - frontal sinus infundibulum mucosa is elevated and inverted inferiorly to occlude the ostium → plug of temporalis muscle is placed atop this, and the sinus cavity is obliterated with one of many autologous materials (abdominal fat, pericranium, cancellous bone, and/or muscle or Gelfoam soaked in antibiotic solution).
  - complete removal of the sinus mucosa (sinus **exenteration**) is imperative for successful outcomes, because residual mucosa can lead to chronic sinusitis, mucoceles, and pain.
  - inner bony cortex is burred, to remove mucosal lining invaginations along **the channels of Bréchet**.
  - lacerated dura (thin in this region!) is closed (running silk suture) → reinforced with pericranial flap; graft may be performed on outer surface of dura, but it is frequently easier to perform it from inner surface after dura has been opened and frontal lobe retracted.
  - if posterior wall is comminuted, it may be removed (sinus **cranialization**).
  - close sinus opening by pericranial flap + fibrin glue.