ETIOPATHOPHYSIOLOGY

- direct impact to skull → inbending or outbending of skull beyond its elastic tolerance.
  - skull fracture - indicator of severe blow to head (increased chance of intracranial abnormality).
  - skull fracture does not necessarily mean that brain is injured (but in many cases it is).
- skull fractures are detected in 5% mild head injuries.
- skull fractures are detected in ~ 30% of all head injuries.

With increasing severity of head injury, likelihood of skull fracture increases, presence of skull fracture increases likelihood of brain injury 4-fold.

N.B. prognosis depends more on brain damage than on skull injury!

Generated forces

- walking into fixed obstruction (73 N force – enough to fracture skull!).
- 4.5 kg adult head falling 1 m on hard surface (510 N).
- falling from standing position (873 N).
- running into obstruction (1020 N).
- 10 times more required to fracture skull with overlying scalp than to fracture one without scalp cover.
- skull thickness is not uniform – force required to cause fracture depends on site of impact: skull vault* is thinner than skull base (weakest parts of skull base → see below);
- skull vault diploë does not form (skull vault is thinner than skull base covered with muscles* (esp. squamous-temporal and parietal bones);
- skull is thick - glabella, external occipital protuberance, mastoid processes, external angular process.

*prone to fracture.

CLASSIFICATION, CLINICAL FEATURES

Fracture type depends on impact force and ratio of impact force to impact area.

Communication with outside:

A. CLOSED
B. OPEN (COMPONDO) - torn pericranial tissues; patient is likely to have severe brain damage.
- fracture that disrupts paranasal sinuses or middle ear is also considered open.

Location:

A. Vault
B. Basilar

Fracture form:

A. Linear (incl. suture diastasis)
B. Depressed
C. Comminuted

Underlying cerebral substance damage:

A. No injury (UNCOMPLICATED FRACTURE)
B. Compression (by depressed fractures)
C. Contusion
D. Laceration (by comminuted fractures)

1. Linear fracture (80%)

- single fracture line goes through entire skull thickness: no displacement.
  - etiology, low-energy blunt trauma over wide surface area of skull.
  - starts at point of maximum impact → extends toward skull base.
  - with multiple points of impact or repeated blows, fracture lines of subsequent injuries do not extend across prior fracture lines.
  - when individual falls while awake → occipital impact; fall that follows loss of consciousness → frontal impact.
  - clinically - just tender bump on head; skin may or may be breached* (most patients are asymptomatic; without loss of consciousness - it is often difficult to predict presence of skull fracture by clinical examination).
  - *scalp is mobile → possible nonalignment of fracture with scalp laceration

- little significance (except when runs over arterial groove, venous sinus groove, or suture → epidural hematoma, venous sinus thrombosis, sutureal diastasis).

2. Basilar fractures (19-21%)

- linear fractures at skull base (often are extensions of adjacent convexity fractures).
  - basilar bones are thick – much more force required to fracture them!
  - most basilar fractures occur at specific locations:
    1) most commonly (75%) - temporal bone.
    2) occipital condylar region (fissure of Stenvers; see below).
    3) along inner surface of sphenoid wings, sphenoid sinus, toward sella turcica and cribiform plate, roof of orbits.
    4) areas between mastoid and dural sinuses in posterior cranial fossa.

- N.B. middle-cranial fossa is weakest part (thinnest + multiple foramina)
  - etiology - impact to side of head (rather than blow to vertex).
  - difficult to detect at postmortem examination (require careful removal of tightly adherent dura).
  - often associated with dural tears.
  - clinically - ecchymoses (periorbital / retroauricular) distant from point of impact, cranial nerve palsy, CSF leaks, pneumocephalus, cavernous-carotid fistula (see below).

ANTERIOR FOSSA

- periorbital ecchymosis limited at edge of orbit ("raccoon eyes") - blood dissecting from disrupted skull cortex into periorbital soft tissue.

Skull Fractures

Last updated: January 18, 2020
NO TABLE OF CONTENTS ENTRIES FOUND FRONTAL SINUS FRACTURES – see p. THH27 >>

1. Closed depressions:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

2. Open depressions:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

3. Composite depressions:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

4. Noncontiguous depressions:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

5. Open sinus:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

6. Noncontiguous sinus:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

7. Contiguous sinus:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

8. Composite sinus:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

9. Noncontiguous open sinus:
   - thin areas (i.e. nasoethmoidal complex)
   - thick areas (i.e. lacrimal bones)

10. Contiguous open sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

11. Composite open sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

12. Noncontiguous closed sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

13. Contiguous closed sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

14. Composite closed sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

15. Noncontiguous open sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

16. Contiguous open sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

17. Composite open sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

18. Noncontiguous closed sinus:
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19. Contiguous closed sinus:
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20. Composite closed sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

21. Noncontiguous open sinus:
    - thin areas (i.e. nasoethmoidal complex)
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22. Contiguous open sinus:
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23. Composite open sinus:
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24. Noncontiguous closed sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

25. Contiguous closed sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

26. Composite closed sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

27. Noncontiguous open sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

28. Contiguous open sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)

29. Composite open sinus:
    - thin areas (i.e. nasoethmoidal complex)
    - thick areas (i.e. lacrimal bones)
2) **CSF rhinorrhea** - CSF leak through cribiform plate or adjacent sinus.

3) **CN 1-2 damage.**

### Middle fossa

**Periosteal portion of temporal bone:**
1. **retroauricular ecchymosis** - delayed ecchymosis over mastoid process (Battle sign) - blood dissecting from disrupted skull cortex.
2. **CSF otorrhea.**
3. **blood in ear canal** (more commonly due to local laceration of external canal)
4. **hemotympanum** (Blood & CSF behind tympanic membrane)
5. **CN7 palsy, hearing loss, vertigo. see below (temporal bone fractures)**

### Sphenoid, Sellar fractures:
1. **air-fluid level in sphenoid sinus**
2. **CN2, 6, 7 palsies**
3. **neuroendocrine dysfunction.**
4. **ICA pseudoaneurysms, carotico-cavernous fistulas.**

### Posterior fossa:

**Clinoid fracture** – **CN6 palsy, ganglion trigeminale lesion.**
**Occipital condylar fractures** – **CN9-12 palsy.**

**see below (occipital condylar fractures)**

### 3. Depressed fracture (is, impressed fracture) - hony piece is driven by direct traumatic impact* below plane of skull:

*usually small blunt objects (such as hammer or baseball bat)

- 75% frontoparietal (may involve frontal sinuses and orbits), > 10% temporal, 5% occipital.
- most (75-90%) depressed fractures are open fractures.
- edges of depressed portion may become locked underneath adjacent intact bone and fail to rebound into previous position.
- in gunshot cases, bullet exit causes **expressed fracture.**
- clinically – depression under generalized swelling (avoid driving bone fragment deeper!), depressed area may be several centimeters away (due to scalp mobility); focal seizures (from confusion underlying fracture).

### Ping-Pong fractures (akin to greenstick fracture of long bones)

- occur in first few months of life.
- etiology:
  1. fall when skull hits edge of hard blunt object.
  2. birth trauma (newborn head was impinged against mother's sacral promontory during uterine contractions).
  3. birth trauma with forceps (rare).
  4. clinicalia - skull appears deformed, with shallow trench on skull surface.

### 4. Comminuted fracture - multiple linear fractures that radiate from impact site (= 2 bone fragments)

- suggests more severe blow (than in single linear fracture).
- portion of bone may be depressed.

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**DIAGNOSIS**

Indications for **skull X-ray** → see p. TiH1 >>

- plain radiographs may miss basal skull (esp. temporal bone) fractures – only clues may be fluid levels (bleeding or CSF leakage) in sphenoid, frontal sinus or petromastoid air cells.
- **am** within cranium:
  a) extradural - sharply defined, superficial, adjacent to midline or fractured sinus;
  b) subdural - very extensive;
  c) subarachnoid - diffuse air, in bubbles, or outlining brain;
  d) within damaged brain;
  e) intraventricular (can cause acute hydrocephalus).

**X-ray differences between linear fractures, normal sutures, and normal vascular markings:**

<table>
<thead>
<tr>
<th>Fractures</th>
<th>Sutures</th>
<th>Vascular Markings</th>
</tr>
</thead>
<tbody>
<tr>
<td>width &lt; 2 mm</td>
<td>same width throughout. lighter on X-rays</td>
<td>engrave inner table only. less transparent than fractures. ill-defined margins. meningeal grooves taper as they run peripherally. symmetrical branching pattern. diploic venous markings are wide.</td>
</tr>
</tbody>
</table>
Skull fracture is indication for **CT**!

- **CT with wide windows (1000-4000 HU)** are needed to evaluate skull injuries.
- **degree of skull depression is easily measured on CT.**
- **CT may miss:**
  1. *small vertex fractures* (often, small streak artifact caused by misaligned fracture may be clue).

**MRI** easily misses skull fractures (low sensitivity and specificity)!  

**Persistent metopic suture** that has not yet fused; this is not fracture:

**Importance of straight patient position for lateral imaging.** Because patient is slightly malpositioned, both coronal sutures are seen as separate entities (also applies to lambdoid sutures); because they are separated - could be mistaken for fracture:

**Bilateral vault fracture** - fluid level in sphenoid sinus (open arrow); two fracture lines - more anterior (upper on this film) is better defined and is therefore on side nearer film; bone islands (small arrows) are typical:

**Depressed skull fracture** (CT ‘brain and bone windows’):
**SKULL FRACTURES**

**TrH5 (4)**

Skull depressed fracture:
A. Lateral projection: typical appearance of dense flake deep to skull vault.
B. Half axial projection.

---

**TREATMENT**

Prehospital management → see p. TrH1 >>

N.B. 15% patients with skull fractures have concomitant cervical spine injury?

**Linear fractures** – no special therapy.
- in children, skull fractures heal within 3-6 months; in adults, complete healing may take up to 3 years.

**Basilar fractures** – treat only complications (CSF leak, etc.)
- no benefit of prophylactic antibiotics for basilar skull fractures.
  
  **Open (compound) fractures** – treated conservatively:
1. tetanus toxoid vaccination
2. irrigation and scalp debridement & repair
3. antibiotic prophylaxis for 5-10 days (guidelines recommend for all open fractures; some experts use only for obviously contaminated cases), e.g.
   - "TRIPLE ANTIBIOTIC" x 5 days: 1) CEPHTRIAXONE 2 g q12h or cefepime 2 g q8h plus 2) NAFCILLIN 2 g q4h plus 3) METRONIDAZOLE 500 mg q8h
4. CT few times over next 2-3 months (to check for abscess formation).

Criteria for operative management of open fracture:
- dural penetration / pneumocephalus
- gross contamination / wound infection
- frontal sinus involvement
- depressed greater than the thickness of the cranium

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.
- open fractures have 10.6% incidence of infection (→ persistent neurological deficit, late epilepsy, and death); operative debridement reduces the incidence of infection to 4.6%, however, operative delay greater than 48 hours from injury dramatically increases the incidence to 36.5%.
  
  **Depressed fractures**

1. Prophylactic anticonvulsants.
2. Most depressed fractures heal well and smooth out with time, without elevation; indications for surgical elevation:
   - depressed greater than the thickness of the cranium (i.e. > 10 mm inward displacement)
   - 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.
   - cosmetic deformity (FRONTAL BONE is most important esthetically + it forms roof and portions of medial and lateral walls of orbit).

- no proof that elevation of depressed fragments decreases epilepsy risk.
  
  **COMPLICATIONS**

Skull fracture per se does not indicate trauma severity.

Skull fracture importance – risk of intracranial infection and bleeding!

Clinically significant skull fractures (prone to complications):
1. extend into air sinuses → infection.
2. basal → CSF leaks (→ infection), cranial nerve / vascular injuries.
3) open → infection.
4) depressed below level of inner table → underlying brain injury → posttraumatic epilepsy.
5) overlie major dural venous sinus/middle meningeal artery → bleeding.
6) linear fractures associated with dural tear in young children → leptomeningeal cyst.

N.B: basilar fractures are most serious - deserve closer monitoring than linear vault fractures!

Bleeding
- infants may bleed significantly intracranially from skull fractures (skull is very vascular – any fracture may cause venous epidural hematoma); check hematocrit q 12-24 h.
- EPIDURAL HEMATOMA is associated with skull fracture in ≈ 50% cases.
- SUBDURAL HEMATOMA is associated with skull fracture in ≈ 33% cases.

Pneumocephalus
See p. TdH1 >>

CSF leaks - otorhea and rhinorrhea (after basilar skull fracture), see p. S64 >>

Meningitis
(via wound or CSF fistula); may extend into brain abscess.

Cranial nerve palsy (after basilar skull fracture), see p. TdH1 >>

Posttraumatic epilepsy (after depressed skull fractures) – risk factors: loss of consciousness for > 2 hours, associated dural tear, early seizures (within first week).

Carotid-cavernous fistula (after sphenoid bone fracture) see p. TdH9 >>

Traumatic aneurysms (e.g. after sphenoid bone fracture) see p. TdH1 >>

Superior longitudinal sinus compression (by depressed vertex fractures) → thrombosis.

Leptomeningeal cyst (s. growing fracture) → extrusion (in form of cyst) of leptomeninges and brain tissue through dural defect:
- encephalopathy: skull fracture with separation of fracture edges [depressed or diastatic skull fracture] and dural 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!!!
- most are located in calvarium (rare sites are basiocciput and orbital roof).
- underlying brain may herniate through skull defect.
- prominent pachymeningeal cyst or focal dilation of lateral ventricle usually underlies fracture.
- 
  - Type I - leptomeningeal cyst herniating through skull defect into subgaleal space.
  - Type II - damaged or gliotic brain.
  - Type III - leptomeningeal cyst is seen.
  - clinically (manifests several weeks + months after fracture): 1) growing subgaleal mass (slowly expanding pulsatile nototender swelling in area of previous skull fracture); 2) convulsive seizures or focal neurologic deficit; 3) mental retardation; 4) mass effect with increased ICP.
  - diagnosis: serial X-ray (sufficient for diagnosis) – enlarging oval area of skull erosion (progressive separation of long edges of seemingly benign linear skull fracture)

- *but CT better defines exact pathology, intracranial hypoplasinating area may be encephalomalacia, arachnoid loculation, or cortical atrophy.
- tools for early diagnosis (→ early simpler surgical intervention – prolonged untreated neurologic sequela).
- 1) MRI - depicts dural tears early.
- 2) ultrasound (tool for assessing state of dura).
- treatment: cyst excision → dural closure → cranioplasty. See p. Op320 >> occasionally, shunt surgery is performed to decompress cyst and treat localized dilatation of ventricles.

LATERAL SKULL RADIOGRAPH OF LEPTOMENINGEAL CYST

AXIAL CT OF LEPTOMENINGEAL CYST: WIDENED DURAL SINUS AND FLUID COLLECTION EXTENDING FROM INTRACRANIAL CAVITY INTO AND THROUGH DURAL SINUS.

Dislocation of bones of auricular chain (after temporal bone fracture), see below.

### SPECIAL SITUATIONS

#### TEMPORAL BONE FRACTURES
- fractured in 15-48% of all skull fractures (75% of all skull base fractures).
- clinical features: Battle's sign, bleeding from ear (hemotympanum or from fracture line in ear canal), CN7 & 8 damage, ossicular chain & tympanic disruption, CSF otorrhea.

Subtypes (by Ulrich, 1926)

1. LONGITUDINAL (70-90%) - PARALLEL TO PETROITIDY PYRAMID:
   - FARE SQUAMOUS, POSTERIOR SUPERIOR WALL OF EXTERNAL AUDITORY CANAL, TEIMEN TYPANUM → RUN EITHER ANTERIOR OR POSTERIOR TO COCHLEA AND LARYNHOTYNE CAPSULE → END IN MIDDLE CANAL FORA NEAR FORAMEN SPINO SUM OR ON MASTOID AIR CELLS, RESPECTIVELY.
   - CAUSED BY DIRECT LATERAL FORCE OVER MASTOID OR SQUAMOUS BONE OR BLOW TO MASTOID.

2. TRANSVERSE (30%) - RIGHT ANGLE TO PETROITIDY PYRAMID:
   - ENGAGE IN TRANSVERSE PROCESS OF SQUAMA XIII, EMERGE FROM MASTOID AIR CELLS, OR OPEN INTO MASTOID AIR CELLS.
3. MIXED - components of both LONGITUDINAL and TRANSVERSE fractures.

Complications:
1) facial nerve paralysis (twice more common with transverse fracture):
   a) delayed incomplete – due to neuropaenia (10-20% longitudinal fractures; injury site is usually horizontal segment distal to geniculate ganglion; H: steroids).
   b) 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 electronencephalography [EENOG] in decision making).
2) hearing loss (hemotympanum and mucosal edema in middle ear may cause temporary deafness - resolves within 3 weeks):
   a) conductive hearing loss due to hemotympanum, ossicular dislocation / fracture or tympanic rapture (≈ 50% longitudinal fractures);
      – incus (relatively loose ligamentous attachments) is most frequently dislocated ossicle.
      – 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 (≈ 30% transverse fractures); H: cochlear implants.
3) vertigo due to:
   a) fracture extending into vestibular apparatus (e.g. with transverse fractures).
   b) labyrinth concussion (e.g. with longitudinal fractures).
   c) development of perilymphatic fistula with fluctuating or progressive hearing loss; H: exploratory tympanotomy.
   d) posttraumatic benign paroxysmal positional vertigo.
4) CSF otorrhea (in any subtype of fracture).
5) unusual complications:
   • carotid injury.
   • CN8 paralysis (recovery within 6 months is usual).
   • CN9 damage.
   • sigmoid sinus thrombosis.
   • posttraumatic cholesteatoma (can grow undetected for years).
   • EASLE syndrome (classic follows nonaccident fracture of ossified styloid and stylohyoid ligament can cause pressure on ECA or ICA → atypical pain referred to cheek or eye; treatment is surgical).
   • sympathetic cochleolabyrinthitis (autoimmune inner ear damage - autoimmune antibodies against inner ear proteins [as in polyarteritis nodosa]; H: immunosuppression).

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

Diagnosis – high-resolution CT (axial and coronal images) with 1-mm slices and magnified views; both windows alone are necessary.

Longitudinal fracture of right temporal bone (axial CT) - fracture line follows long axis of temporal bone (medium arrowhead); manubial air cells opacified with blood (large arrowhead).

Transverse fracture of temporal bone (axial CT) - fracture line (arrowheads) crosses petrous pyramid at level of posterior semicircular canal and posterior genu of CNT canal (arrow).
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, AO ligamentous injury - due to forced rotation and lateral bending.
**SKULL FRACTURES**

**CONSIDER**
- 30% patients present comatose, 30% - neuro intact, 40% - with neuro deficits.
- occipitocervical tenderness, reduced craniocervical motion, lower cranial nerve abnormality, retropharyngeal soft tissue swelling.

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

**RADIOPHGRAPHIC**
- difficult to delineate (XR has sensitivity only 1.4%); CT is recommended (sensitivity 100%; Level II recommendation); MRI is recommended to assess ligaments (Level III recommendation).

**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.

**BIBLIOGRAPHY** for ch. “Head Trauma” ➔ follow this LINK ➔

Please visit website at www.NeurosurgeryResident.net