Underlying cerebral substance damage
Fracture form
Location
Generated forces
- fracture indicator of severe blow to head (increased chance of intracranial abnormality).
- presence of fracture is not consise with history of minor head injury!
- skull fracture does not necessarily mean that brain is injured (but in many cases it is).

With increasing severity of head injury, likelihood of skull fracture increases;
- skull fractures are detected in 5% mild head injuries.
- skull fractures are detected in ~30% of all head injuries.

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 force is 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 fracture does not necessarily mean that brain is injured (but in many cases it is).

Impact to skull → inbending or outbending of skull beyond its elastic tolerance.

Presence of fracture is not consistent with history of minor head injury!

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

Communication with outside:
A. CLOSED
B. OPEN (COMPOUND) – torn pericranial tissues; patient is likely to have severe brain damage.

- fracture that disrupts parasagittal sinuses or middle ear is also considered open.

Location:
A. Vault
B. Basilar

Fracture form:
A. Linear (incl. sutureal diastasis)
B. Depressed
C. Commminuted

Underlying cerebral substance damage:
A. No injury (UNCOMPLICATED FRACTURE)
B. Compression (by depressed fractures)
C. Contusion
D. Laceration (by depressed fractures)

1. Linear Fracture (80%): single fracture line goes through entire skull thickness; no displacement

- edema, 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.
- clinical signs: just tender bump on head; skin may or may not be breached* (most patients are asymptomatic; without loss of consciousness - it is often difficult to predict presence of skull fracture by clinical examination).
- skull vault is mobile → possible notchalignment 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 fracture (19-21%): linear fractures at skull base (often are extensions of adjacent convexity fractures).

- basilar botens are thick – much more force required to fracture them!
- most basilar fractures occur at specific locations.

1) most commonly (75%) – temporal bone. see below.
2) occipital condyl vegetation (foramen magnum). see below.
3) along inner parts of sphenoid wings, sphenoid sinus, toward sella turcica and cristaiform plate, roof of orbits.
4) areas between mastoid and dural sinuses in posterior cranial fossa.

N.B. middle cranial fossa is weakest point (thinnest + multiple foramina)

- edema, impact to occipital or sides 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 palsies, CSF leaks, pneumocephalus, cavernous-carotid fistula. see below (complications).
**SKULL FRACTURES**

**TIH5 (2)**

**Anterior fossa:**
1. *periorbital ecchymosis* limited at edge of orbit (“raccoon eyes”) - blood dissecting from disrupted skull cortex into periorbital soft tissue;

2. CSF otorrhea - CSF leak through cribiform plate or adjacent sinus.

3. CN 1-2 damage.

**Middle fossa:**

**Petrous portion of temporal bone:**

1. *retroauricular ecchymosis* - delayed ecchymosis over mastoid process (Battle sign) - blood dissecting from disrupted skull cortex;

2. CSF rhinorrhea - CSF leak through cribriform plate or adjacent sinus.

3. *blood in ear canal* (more commonly due to local laceration of external canal)

4. *hemotympanum* (Blood in 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:**

**Clivus fracture – CN6 palsy, ganglion trigeminale lesion.

**Occipital condylar fractures – CN9-12 palsy.**

see below (*Occipital Condylar Fractures*)

---

**Depressed fracture** (*impressed fracture*): bony 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 rebond 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 contusion underlying fracture).

**Pempo-pong fractures** (akin to greenstick fracture of long bones)

- occur in first few months of life.

1. fall when skull hits edge of hard blunt object.

2. birth trauma (newborn head was impinged against mother’s sacral promonto.

3. traumatic impact behind 

- *clinical signs* - 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.

---

**DIAGNOSIS**

Indications for **skull X-ray**: see p. TIH5 "">

- 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 cranial:

  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>
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<th><strong>Sutures</strong></th>
<th><strong>Vascular markings</strong></th>
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Skull fractures are indications 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:

的重要性 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:

**Lateral CT scanogram and axial bone-window CT** - *ping-pong ball temporal fracture* - slight inward bulging of bone, but inner and outer tables are intact:

**Depressed skull fracture** with parenchymal contusion (CT ‘brain and bone windows’):
Skull fractures

**Skelto 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 fracture** → 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).

- Cochrane review found no benefit of prophylactic antibiotics for basilar skull fracture
  

**Open fractures**:
1) **tetanus** toxoid vaccination
2) **irrigation and debridement**
3) **antibiotic** prophylaxis for 5-10 days (only for obviously contaminated cases, e.g.
   "**TRIPLE ANTIBIOTIC**" x 5 days:
   1) **CEFTRIAXONE** 2 g q12h or cefepime 2 g q8h plus
   2) **NAFULIN** 2 g q4h plus
   3) **METRONIDAZOLE** 500 mg q6h
4) **CT** few times over next 2-3 months (to check for abscess formation).

**Depressed fractures**

1. **Prophylactic anticonvulsants**.
2. Most depressed fractures heal well and smooth out with time, without elevation; **surgical elevation** indicated:
   a) **depth of depression thicker than calvaria** [i.e. > 3-10 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.
   - **cosmetic defect** (FRONTAL BONE is most important esthetically + it forms roof and portions of medial and lateral walls of orbit).
   - open contaminated fracture.
   - no proof that elevation of depressed fragments decreases epilepsy risk.
   - elevation of small depressed fractures need not be performed immediately (but before discharge).
   - indications for immediate elevation: gross contamination, dural tear with pneumocephalus, underlying hematoma.
   - **surgery details**: see p. Op 320 >>

**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) **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).
3) **depressed below level of inner table** → underlying brain injury → posttraumatic epilepsy.
4) **overlie major dural venous sinus / middle meningeal artery** → bleeding.
5) **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.

**CSF leaks** - otorrhea and rhinorrhea (after basilar skull fractures), see p. S64 >>

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

**Pneumocephalus** see p. TrH1 >>

*See text for further details.*
Cranial nerve palsy (after basilar skull fractures), see p. ThHII >>

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. ThHII >>

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

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

Leptomeningeal cyst (c. growing fracture) – exursion (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 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 poremephalic cyst or focal dilatation 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: prominence of cyst is seen.
- clinically (manifests several weeks + months after fracture): 1) growing subgaleal mass (slowly expanding pulsatile nontender 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 hypoattenuating area may be encephalomalacia, arachnoid laceration, or cortical atrophy.
- tools for early diagnosis (→ early simpler surgical intervention) → prevented long-term neurologic sequelae:
  1) MRI – depicts dural tears early.
  2) ultrasonography (tool for assessing state of dura).
- occasionally, shunt surgery is performed to decompress cyst and treat localized dilatation of ventricles.

LATERAL SKULL RADIOGRAPHS OF LEPTOMENINGEAL CYST

ANTERIOR CT OF LEPTOMENINGEAL CYST WITH SPHERICAL 10-20% VACUITY INTO AND THROUGH FRACTURE SITE

Dislocation of bones of malaric 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), CNT & R damage, ossicular chain & tympanic disruption, CSF rhinorrhea.

Subtypes (by Ulric, 1926)

1. LONGITUDINAL (70-90%) - PARALLEL TO PETROUS PYRAMID:
   - PART SIQUAERA (POSTERIOR WALL OF EXTERNAL AUDITORY CANAL, TESSEIM tympanum) → RUN基金 ANTHER OR POSTERIOR TO COCHLEA AND Labyrinth CIVPS. === END IN MIDDLE CRANIAL FOSSA NEAR FORAMEN SPINOSUM OR IN MAXFARD AIR CELLS, RESPECTIVELY.
   - CAUSED BY DIRECT LATERAL FORCE OR MAXFARD OR SQUAERA'S BONE OR BLOW TO MAN.)

2. TRANSVERSE (3-35%) - PERPENDICULAR TO PETROUS PYRAMID:
   - ORIGINATE AT FORAMEN MAGNUM → EXTEND THROUGH COCHLEA AND Labyrinth → END IN MIDDLE CRANIAL FOSSA.
   - CAUSED BY FRONTAL OR PARTRAL BLOW BUT MAY RESULT FROM OCCIPITAL BLOW.
   - PNEUMOLABYRINTH MAY BE SIGN.

3. MIXED – components of both LONGITUDINAL and TRANSVERSE fractures.

Complications:
1) facial nerve palsy (twice more common with transverse fracture):
   a) delayed-incomplete – due to neurapaxia (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 electromyoneurography [ENOG] in decision making).

2) bearing loss (hemotympanum and mucosal edema in middle ear may cause temporary deafness - resolves within < 3 weeks):
   a) conduction hearing loss due to hemotympanum, ossicular dislocation / fracture or tympanic rupture (< 50% longitudinal fractures),
SKULL FRACTURES

TH5 (6)

SKULL 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 (= 80% 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 (parasymptomatic vertigo 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.
   - CN6 paralysis (recovery within 6 months is usual).
   - CN5 damage.
   - sigmoid sinus thrombosis (can grow undetected for years).
   - EAGLE syndrome (classically follows tonsillectomy; 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 cocchleolabyrinthitis (autoimmune inner ear damage - autoantibodies against inner ear proteins (as in polyarteritis nodosa); H: immunosuppression).

   - carotid injury.
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SKULL FRACTURES

Type III fracture - unstable avulsion injury. AO ligamentous injury - due to forced rotation and lateral bending.

CLINICALLY
- 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).

RADIOGRAPHIC
- 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.

FRONTAL FRACTURES that extend into paranasal sinuses - treated as “open fractures” (because of communication with paranasal sinuses).
- look for pneumocephalus, fluid in frontal sinuses.
- if posterior wall of frontal sinus is fractured (esp. if sinus duct is violated – affected drainage → mucocele → subdural abscess) → surgical treatment (frontal sinus exenteration and cranialization):
  - open adequate scalp flap (bicoronal incision) → develop pericranial flap (alternatively – make full thickness scalp flap and dissect pericranial flap immediately before using it) → frontal craniotomy.
  - take cultures.
  - sinus is exenterated (mucosa removed and superficial bone layer drilled with heat-generating diamond drill) and occluded with muscle, fat, or Gelfoam soaked in antibiotic solution.
  - 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.
  - it may be necessary to ligate anterior extent of sagittal sinus if it has been injured.
  - close sinus opening by pericranial flap.
  - replace bone flap.

BIBLIOGRAPHY for ch. “Head Trauma” – follow this LINK >>

Viktor's Notes℠ for the Neurosurgery Resident
Please visit website at www.NeurosurgeryResident.net