MANAGEMENT OF TEMPORAL BONE TRAUMA
SURGICAL ANATOMY OF TEMPORAL BONE

Composite bone

- tympanic bone
- mastoid bone
- squama
- petrosa

Styloid process – not!
Figure A–1  Lateral surface anatomy: note the zygomatic process, tympanic annulus, temporal line, and mastoid tip.

Figure A–2  Superior surface anatomy: important landmarks for the middle fossa surgeon are the temporosquamous suture line, facial hiatus (greater superficial petrosal nerve), tympanic canaliculus (lesser petrosal nerve), arcuate eminence (relative position of superior semicircular canal), and foramen lacerum (carotid artery).

Figure A–3  Posterior surface anatomy: the sigmoid sulcus forms a prominent depression on this surface. Anterior to the midpoint of the sigmoid sinus is a lip of bone (operculum). Beneath the operculum is the opening for the vestibular aqueduct. Further anteriorly lies the internal auditory canal (IAC).

Figure A–5  Inferior surface anatomy: crucial relationships here for the skull base surgeon include the jugular fossa, stylomastoid foramen, and carotid canal.
EPIDEMIOLOGY
When head trauma is sufficient to fracture the skull, 14 to 22% of injured patients sustain a temporal bone fracture. 

- Occurs across all age groups
  - > 70% # in second, third, fourth decade

- Male: female = 3:1

- 8 to 29 % of # occur bilaterally
PATHOPHYSIOLOGY

- greater **force required**, 1875 pounds

- # takes path of least resistance

- injury to various imp structures in temporal bone
  - Facial nerve
  - Cranial nerves 9,10,11
  - Cochlea
  - Labyrinth
  - Middle ear ossicles, TM
  - Carotid art
  - Jugular vein
CLASSIFICATION

• Traditionally
  on the basis of # line in relation to axis of temporal bone
    1. transverse – 10 to 30%
    2. longitudinal – 70 to 90%
    3. mixed

• Recently modified
  on the basis of inner ear damage
    1. otic capsule disrupting
    2. otic capsule sparing
<table>
<thead>
<tr>
<th></th>
<th>Longitudinal</th>
<th>Transverse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>80 %</td>
<td>20 %</td>
</tr>
<tr>
<td>Type of inj.</td>
<td>Parietal blow</td>
<td>Occipital blow</td>
</tr>
<tr>
<td># line</td>
<td>Parallel to long axis, squamous part to end at foramen lacerum</td>
<td>Across petrous bone, from foramen magnum towards foramen spinosum</td>
</tr>
<tr>
<td>Bleeding from ear</td>
<td>common</td>
<td>Absent , TM intact</td>
</tr>
<tr>
<td>CSF otorrhea</td>
<td>+ , mixed with blood</td>
<td>- , unmanifested</td>
</tr>
<tr>
<td>Structures inj.</td>
<td>Tegmen, ossicles, TM</td>
<td>Labyrinth , CN VIII</td>
</tr>
<tr>
<td>Hearing loss</td>
<td>Conductive</td>
<td>Sensorineural</td>
</tr>
<tr>
<td>Vertigo</td>
<td>Less often</td>
<td>severe</td>
</tr>
<tr>
<td>Facial paralysis</td>
<td>Less (20%), delayed onset. Inj in tympanic seg distal to gen.ganglion</td>
<td>More common (50%), imm.onset. Inj in meatal or labyrynthine seg.proximal to gen.ganglion</td>
</tr>
</tbody>
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Otic capsule sparing #

- Typically involve squamous portion of temporal bone & posterosuperior wall of EAC

- Passes through the mastoid air cells, middle ear, tegmen mastoideum, tegmen tympani

- Proceeds anterolateral to otic capsule.

- Fracturing the tegmen in the region of facial hiatus.

- Results from temporoparietal blow
Otic capsule disrupting #

- Pass through otic capsule
- Proceeding from the foramen magnum across petrous pyramid & otic capsule.
- Often passes through jugular foramen, IAC, foramen lacerum
- Do not affect ossicular chain, EAC
- Results from occipital blow
Temporal bone # in pediatric

- higher incidence of intracranial complications (58%)

- Lower incidence of facial nerve paralysis (3%)
EVALUATION

- Uncommon for temporal bone # to occur in isolation.
- Initial evaluation & management
  - securing airway with stabilizing spine.
  - manage breathing
  - controlling hemorrhage,
  - evaluating neurological status,

- The neurootologic examination assess facial nerve function in emergency dept. as soon as possible, before administrating muscle relaxants.
Ear examination

- External lacerations and hematoma

- The ear canal is not packed unless req to control significant bleeding.

- Hemotympanum

- Raccoon sign

- Battle’s sign

# along the scutum, & roof of EAC
**TM**

- Integrity of TM should be checked
- TM perforations heals spontaneously
- Req.no acute intervention
• Nasal exam for rhinorrhea
• Facial nerve exam
• Ocular movement exam for nystagmus or diplopia
• Tuning fork test
• Audiometry
• CSF analysis
• Imaging
Imaging

- NCCT head
to assess intracranial hemorrhage
- HRCT - gold standard
  - in presence of facial paralysis,
  - CSF fistula,
  - disruption of superior wall of EAC
  - suspected vascular injury

- MRI for cranial nerve injury
- MRA or angiogram for vascular injury
COMPLICATIONS

- Facial nerve injury
- CSF fistulas & meningitis
- Hearing loss
- Carotid artery injury
- Cholesteatoma, EAC stenosis
INDICATIONS FOR SURGICAL INTERVENTION

- Facial nerve injury
- CSF fistulas & meningitis
- Hearing loss

For vast majority of temporal bone fractures, we do nothing!!
FACIAL NERVE INJURY

- Severly disfiguring complication
- 7% result in facial paralysis,
- 25% inv. complete paralysis
- 27% of facial nerve injuries present with immediate onset
- 73% will have facial motion in the initial examination & subsequently deteriorate.
- The latency of delay – ranges from 1-16 days.
- It is crucial to differentiate between
  ‘delayed onset’ from ‘delayed diagnosis’
• **Delayed onset** – as documented facial function in the emergency dept that subsequently deteriorates.

• **Delayed diagnosis** – occurs when pt is given a paralytic agent & is intubated before the examination of facial function, in such situation assessment is delayed until extubation.

• These pt should be categorized as ‘**unestablished onset**’ & treated in a manner that is similar to the immediate onset.
- Most imp. predictive factor – delay in onset

- Most of the studies – against surgical exploration & decompression of delayed post traumatic facial paralysis.

- Consequently complete paralysis of immediate onset or unknown onset or nerve is suspected of being severed, crushed, impaled with bone fragment– considered for surgical exploration
Figure 125-11 Management of traumatic facial paralysis.

Immediate onset: paralysis
  Yes
  Unknown
  No
  Observation and systemic steroids

Complete paralysis
  Yes
  Loss of stimulability or 95% degeneration on EnoG within 14 days
  No
  Observation and systemic steroids

Facial nerve exploration
  Yes
  Otic sparing fracture
    Yes
    Well aerated mastoid air cells or ossicular discontinuity
      No
      Facial nerve severed
        Yes
        Great auricular nerve cable graft or direct anastomosis
        No
        Total facial nerve decompression achieved
          Yes
          Severed facial nerve encountered
            Great auricular nerve cable graft or direct anastomosis
            Adequate exposure
              Yes
              Procedure completed
              No
              Combined transmastoid middle cranial fossa total facial nerve decompression
CSF fistula & Meningitis

- Most serious complications
- Occuring in 17% of cases, incidence 2 to 88%
- In otic capsule sparing # - through floor of MCF (tegmen tympani, mastoideum)
- In otic capsule disrupting # - from posterior cranial fossa
- TM disrupted – CSF otorrhea
- Delay in CSF leakage –
  1. herniation of dura or brain fungus into bony defect
  2. haematoma obstructing the outlet
• The CSF fistula continue to leak until fibroblastic proliferation creates a fibrous barrier to close the defect.

• During early stage of healing fibrous barrier is weak & mucosal barrier remains fragile.

• If CSF pressure gradient > tensile strength of barrier – leak will continue
Diagnosis

- Differentiated from watery rhinitis, lacrimal secretions, serosanguineous discharge on the basis of composition.
- CSF – elevated glucose,
  - less protein
  - less potassium
- Protein electrophoresis - beta 2 transferrin
- Beta 2 transferrin – specific to CSF & small amount of CSF (50 microL) required for test
- HRCT and CT cisternography.
Management

• The most common infecting organisms = streptococcus pneumoniae & haemophilus influenzae

• 57 to 85 % post traumatic fistulas – treated conservatively cease leaking within 1 wk

• T/t includes – head end elevation
  stool softener
  avoid nose blowing, sneezing, straining
  repeat LP or drain if leak persists
Figure 125-13 Management of traumatic cerebrospinal fluid fistula.

CSF fistula

Suspected leak
- Inadequate fluid sample obtainable
  - \( \exists_2 \) transferrin (Positive)
    - Omnopaque CT of temporal bone/paranasal sinuses
      - Negative and low suspicion
        - Observe
          - Negative
            - Intrathecal fluorescein (Positive)
              - Leak resolves
            - Positive
              - Lumbar drain
                - Leak persists for 7–10 days
                  - Fistula through cribiform plate or fora ethmoidalis
                    - Intranasal mucoperichondrial flap
                      - Surgical intervention
                        - Otic = capsule – sparing temporal bone fracture
        - Positive
          - Elevate head of bed
            - Bedrest
            - Stool softeners
            - Prophylactic antibiotics
    - Positive
      - Leak resolves

Obvious leak

Persistent leak

Lumbar drain

Surgical intervention

Otic = capsule – disrupting temporal bone fracture

Mastoidectomy with mini middle cranial fossa craniotomy

Resect EAC, TM and obliterate middle ear and eustachian tube

**Note:** This diagram outlines the management of traumatic cerebrospinal fluid fistula, detailing the steps from suspected to obvious leaks, including diagnostic tests, interventions, and outcomes.
Hearing loss

- Can be conductive, sensorineural, mixed
- Otic capsule sparing # - tearing of TM, ossicular chain disruption
- Most common inj. –
  - incudostapedial joint sep. (82%)
  - incus dislocation (57%)
  - # of stapes crura (30%)
  - fixation of ossicles (25%)
  - # of malleus (11%)
THANK YOU