Journal Review: Temporal bone trauma

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Introduction

- Injuries to the temporal bone occur in 30-70% of cases involving blunt head trauma.
- Commonly suffer from multiple other body injuries.
- Motor vehicle accidents are the most common cause, with falls and gunshot wounds contributing to a lesser extent.
Complications

- Intracranial hemorrhage, cerebral contusion, meningitis, hearing loss, facial paralysis, cerebrospinal fluid fistula, cholesteatoma and external auditory canal stenosis.

- May result in death or permanent deficits.
Anatomy and function

- The temporal bones form parts of the **middle and posterior cranial fossa** and contribute to the neurocranium or skull base.

- Protecting the brain, middle and internal ear apparatus including the cochlea, vestibule and the vestibulocochlear nerve (cranial nerve VIII), the facial nerve (cranial nerve VII), the internal carotid artery, and the jugular vein.
Anatomy

Each temporal bone is divided into five components: squamous, tympanic, styloid, mastoid, and petrous.
Skull
Lateral View

- Styloid process
- Squamous part of temporal bone
- Mastoid process of temporal
Symptom

- **Hearing loss**: immediately apparent to conscious patients, the most common (40% of patients with head injury).

- ± **Tinnitus**: no prognostic significance

- **Dizziness and dysequilibrium** often noticed unless severe labyrinthine injury has occurred.
  - Many patients notice imbalance only after becoming ambulatory.
PHYSICAL EXAMINATION

- Nystagmus: vestibular injury, the direction of nystagmus is usually away from the affected ear.

- Extensive physical assessment of vestibular complaints immediately after temporal bone trauma is unnecessary, as complete recovery from imbalance and nystagmus is to be expected.
Sign

With history of head trauma are sufficient for the diagnosis of temporal bone fracture, even in the absence of radiographic evidence.

- Hemotympanum
- Postauricular ecchymosis or Battle’s sign: fracture defect usually involves the mastoid cortex or squamous portion.
- Periorbital ecchymosis or raccoon sign
PHYSICAL EXAMINATION

- Facial paresis (weakness)
  - Easily unnoticed due to facial swelling, lacerations, and abrasions.
  - Immediate: the first few hours after injury
    Hilger nerve stimulator, day 3~7 after injury.
    No loss of stimulability → observed.
    Loses stimulability → facial nerve exploration
  - Late-onset: delayed for days or weeks, common.
    2-week systemic corticosteroids and observed
    Generally has an excellent recovery of function.
ELECTRONEURONOGRAPHY (ENoG)

Stimulating and Recording Electrode Sites

TIP: test this side first and use ENoG values as a reference

Electroneuronography (ENoG) Analysis:

Analysis: "Significant degeneration in facial nerve response"

Amplitude in microvolts (µV)

<table>
<thead>
<tr>
<th>Time in milliseconds (ms)</th>
<th>Uninvolved (normal) side</th>
<th>Involved (paralyzed) side</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>400 µV</td>
<td>750 µV</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
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</tbody>
</table>

Percent degeneration = 100 - \frac{\text{amplitude on involved side of face (µV)}}{\text{amplitude on uninvolved side of face (µV)}} \times 100

Example:

\[
\text{Percent degeneration} = 100 - \frac{40 \, \mu V}{750 \, \mu V} \times 100 = 95\%
\]
PHYSICAL EXAMINATION

- CN V injury: facial hypesthesia (decreased or absent touch sensation)
- CN VI injury: diplopia
- Usually not noticed immediately after injury → Edema is responsible for the damage (not direct trauma)
- Spontaneous recovery of both facial hypesthesia and diplopia is the general rule.
RADIOGRAPHIC EVALUATION

- **High-resolution CT** scans with bone algorithms are the standard.
  - >1/3 fractures detected by CT are missed by clinical diagnosis.
  - Invaluable in assessing the location of facial nerve injury, as well as for planning surgical approaches.
- **MRI**: useful in corroborating cranial nerve injury.
- **Gunshot wounds or other penetrating trauma** → **angiography or MRA**: greater possibility of injury to the internal carotid artery.
Fig. 3  Longitudinal fractures (red lines) are roughly parallel to the long axis of the petrous bone, whereas transverse fractures (yellow lines) are perpendicular. Majority of temporal bone fractures do not fit to this classification system. Same anatomic structures may be injured by both types of fractures limiting the clinical value of this classification system in predicting patients’ symptoms and outcome.
<table>
<thead>
<tr>
<th>Hearing loss</th>
<th>Injury site</th>
<th>Fracture type</th>
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</thead>
<tbody>
<tr>
<td>Conductive</td>
<td>conducting system distal to the cochlea</td>
<td>Longitudinal fractures of the temporal bone or injuries with no identifiable fracture</td>
</tr>
<tr>
<td>repaired at any time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>Both</td>
<td></td>
</tr>
<tr>
<td>Sensorineural</td>
<td>Internal ear (cochlea and CN VIII)</td>
<td>Transverse fracture</td>
</tr>
<tr>
<td>Poor prognosis, not influenced by treatment</td>
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Hearing loss type: Prognostically important but does not influence the timing of surgery.
Otic capsule-based classification

- Otic capsule: the bone that houses the cochlea and the semicircular canals.
- More clinically relevant.
<table>
<thead>
<tr>
<th></th>
<th>Otic sparing fracture</th>
<th>Otic capsule–disrupting</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incidence</strong></td>
<td>94.2-97.5%</td>
<td>2.5-5.8%</td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>blow to temporo-parietal region.</td>
<td>blows to the occipital region</td>
</tr>
<tr>
<td><strong>Pathway</strong></td>
<td>Mastoid air cell, middle ear → tegmen mastoideum → tegmen tympani → tegmen in the region of the facial hiatus</td>
<td>Foramen magnum → petrous pyramid and otic capsule → jugular foramen, IAC, foramen lacerum</td>
</tr>
<tr>
<td><strong>Involvement</strong></td>
<td>Squamosal portion of temporal bone, posterosuperior wall of EAC</td>
<td>Not typically affect the ossicular chain or EAC</td>
</tr>
<tr>
<td><strong>Hearing loss</strong></td>
<td>Conductive/mixed HL</td>
<td>SNHL (7 times)</td>
</tr>
<tr>
<td><strong>Facial nerve paralysis</strong></td>
<td>6-14%</td>
<td>30-50%</td>
</tr>
<tr>
<td><strong>CSF fistula</strong></td>
<td>1X</td>
<td>2-4X</td>
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Evaluation

- Electroneuronography (ENOG): the most effective method for testing facial nerve function, by comparing the summed action potential of the affected side with that of the uninjured side.
- Any observation of facial paralysis should be followed with ENOG.
- ENOG is generally performed 2 to 3 days after facial nerve injury but within 2 to 3 weeks.
Emergent intervention

- **Obvious brain herniation (encephalocele)** into the middle ear, mastoid, or external acoustic meatus.

- **Massive bleeding** from intratemporal carotid artery laceration.
  - Balloon occlusion of the vessel is generally faster than surgical ligation and repair.
Early surgical intervention for facial nerve paresis

- Immediate paralysis and no evidence of return of function after 1 week.
- Immediate paralysis + significant temporal bone disruption (CT): severe nerve laceration or sectioning.
- Immediate paralysis and progressive decline in ENOG functioning to less than 10% of the normal side.
Transmastoid approach

- Only for lesions determined to be distal to the geniculate ganglion.

Postauricular ecchymosis (Battle’s sign): the fracture defect usually involves the mastoid cortex or squamous portion. The fracture line can be followed medially to the point of facial nerve injury.
Middle cranial fossa approach

Facial nerve injury **proximal to the geniculate ganglion** and no sensorineural hearing deficits.

*Figure 9* Diagram of the facial nerve as exposed in a middle cranial fossa approach. Careful dissection is essential to avoid the superior semicircular canal and the basal turn of the cochlea.
Transmastoid-translabyrinthine approach

- For sensorineural hearing loss that is unlikely to improve.
- Less associated morbidity than the middle cranial fossa approach

Figure 10  Diagram of the facial nerve as exposed in a translabyrinthine approach. When combined with a transmastoid approach, the entire intratemporal course of the facial nerve can be visualized.
Facial nerve decompression

- Identify location of facial nerve injury, removed any bone chips.
- Examined for stretching, compression, laceration, or transection.
- Largely intact nerve → decompression of the epineural sheath in proximal to distal.
- Partial transection → repaired with suture.
- Separation >50% of the axons → interpositional nerve graft (greater auricular nerve)
Management of traumatic facial paralysis.
CSF leakage

- Usually **resolves spontaneously within 2 weeks without intervention**.
- **Antibiotics are not routinely prescribed**, for fear of masking early infection.
  - No statistically significant effect on the incidence of meningitis.
- **Questioned frequently** about meningeal symptoms (headaches with nuchal rigidity, photophobia)
- **Lumbar puncture** if meningitis is suspected, before beginning antibiotic therapy.
CSF leakage- surgical intervention

- Surgery is indicated for continuous CSF otorrhea or rhinorrhea persisting longer than 14 days.
- If lumbar drainage for 72 hours fails, surgical exploration is recommended for closure of the dural tear and prevention of meningitis.
- Dehiscent brain tissue extending into the temporal bone is nonfunctional and can be removed by electrocautery.
Figure 145-13. Management of traumatic cerebrospinal fluid (CSF) fistula. EAC, external auditory canal; TM, tympanic membrane.
Management- hearing

- Conductive hearing loss secondary to hemotympanum resolves without intervention.
- Ossicular disruption can be repaired electively.
- Surgery is not recommended earlier than 3 months after trauma because of postinjury edema, bleeding, and friability of healing tissues.
- Sensorineural hearing loss may show improvement over time but tends to persist and is refractory to treatment.
Management

- **Intravenous corticosteroids**: reduce edema in and around the nerve, for sensorineural hearing loss and facial nerve injury.
  - Little data examining the efficacy
  - Relatively inexpensive, and a short course of steroids presents minimal risk of complications.
Management

- Dysequilibrium usually responds to activity and should resolve without additional intervention.
- Benign paroxysmal positional vertigo can follow head injury after days to weeks, resolves spontaneously.
- Vestibular suppressants: used briefly, tapered rapidly to allow for CNS compensation.
- Early ambulation also stimulates CNS compensation.
Reference

- **Management of Temporal Bone Trauma**
  *Craniomaxillofacial trauma & reconstruction/volume 3, number 2, 2010*

- **Temporal bone fractures**

- **Management of Temporal Bone Trauma**
  *Cummings Otolaryngology: Head & Neck Surgery, 5th ed. Chap. 145*

- **A comparison of temporal bone fracture classification systems**
  *Clinical Otolaryngology, 31, 287–291*
Thanks for your attention!