15-Facial Nerve

Objectives:

- Anatomy (course and branches).
- Causes of facial palsy (including Bell's palsy, middle ear complication, traumatic and Ramsey Hunt syndrome).

Dcotor's notes are very important in this lecture, if you don't have enough time make sure you read the golden and red notes at least.

Resources: Team 435, slides, team 436 group A

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[Color index: Important | Notes | Extra] Editing File

Introduction:

- The facial nerve (CN VII) provides motor fibers to the muscles of **facial expression.**
- It originates in the seventh nerve nucleus in the brain stem (**pons**), enters the **middle ear** and mastoid and exits the skull at the **stylomastoid foramen** just in front of the mastoid process.
- From here it enters the **parotid** gland where it divides into its branches (Figure below).
- Paralysis can be caused by pathology anywhere along the nerve course or in the cortical nerves which control the nucleus (supranuclear or upper motor neuron fibers) resulting in **asymmetric movement** of some or all the muscles of facial expression.
- Facial nerve palsy causes difficulty with smiling, frowning and expressing emotions, it is a devastating condition for the patient.
- The causes are numerous and are listed in (Table 15.1). 'Supranuclear' or upper motor neuron causes will often spare the forehead as these muscles receive fibers from both facial nerve nuclei.

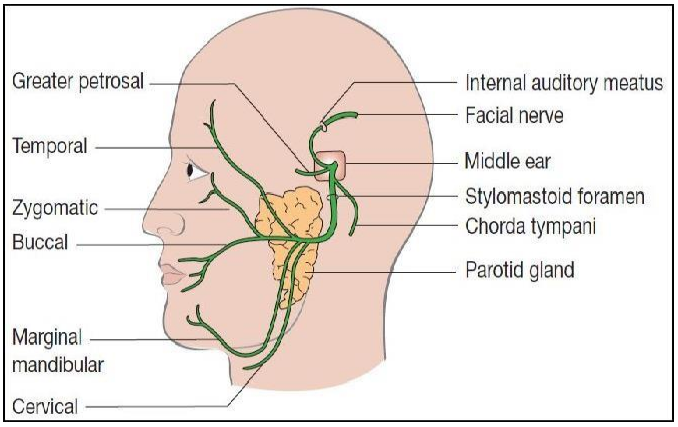


Table 15.1 Common causes of facial nerve paralysis

Supranuclear and nuclear (upper motor neurone)
- Vascular lesions, e.g. stroke
- Intracranial tumours
- Multiple sclerosis

Infranuclear (lower motor neurone)
- 'Bell's palsy'
- Trauma (birth injury, fractured temporal bone, surgical)
- Tumours (parotid tumours, acoustic neuroma, malignant disease of the middle ear)
- Middle ear suppuration (acute or chronic otitis media)
- 'Ramsay Hunt' syndrome
- Guillain–Barré syndrome
- Sarcoidosis

Facial Nerve Fibers

Consists of 10k (10,000) neurons, 7k (7,000) of which have myelinated motor fibers (facial expression). 70% motor and go to the muscles of expressions, 30% sensory (10: Taste, 10: General sensation, 10: secretomotor)

- **Motor fibers:**
  - To the stapedius and facial muscles.
  - What does the stapedius do? A dampening action as it stabilizes the foot of stapes, so any damage to the stapedial nerve ⟶ hyperacusis and phonophobia

- **Secretomotor fibers (parasympathetic):**
  - To the lacrimal gland and the submandibular and sublingual salivary glands.
  - Also supplies palatine and nasal glands.
  - Note that the parotid gland is supplied by CN IX

- **Taste fibers:**
  - From the anterior two third of the tongue and palate.

- **Sensory fibers:**
  - pain, temperature, and touch from the external auditory canal (external auditory meatus) very few.

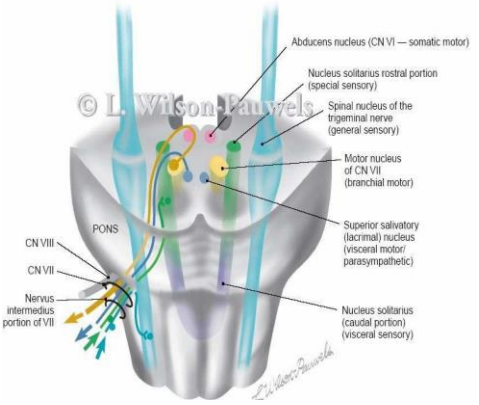
They carryout pain, temperature and touch sensation

Nuclei (PONS) 4Ss' (The intracranial part)

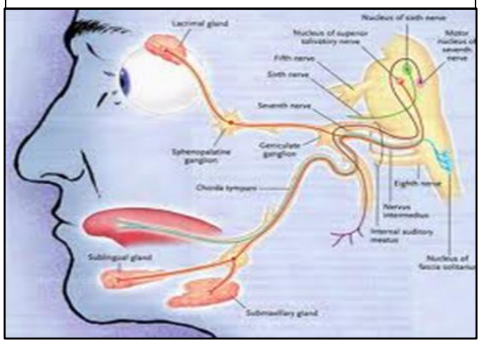
- This part includes the nuclei of facial nerve and the cerebellopontine (CP) angle segments.

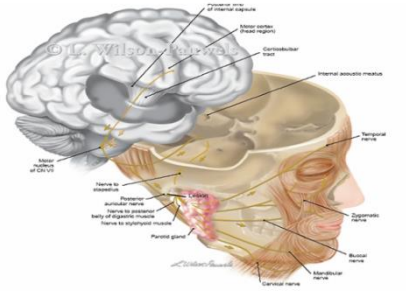
- Facial Nerve Nuclei (in the pons):

↪ **Solitarius (Taste)** receives taste fibers.
↪ **Seventh (motor)** Facial nucleus: main nucleus which gives motor fibers
↪ **Superior Salivatory (Lacrimal) Nucleus** gives parasympathetic fibers.
↪ **Spinal Nucleus of The Trigeminal Nerve**. General sensation

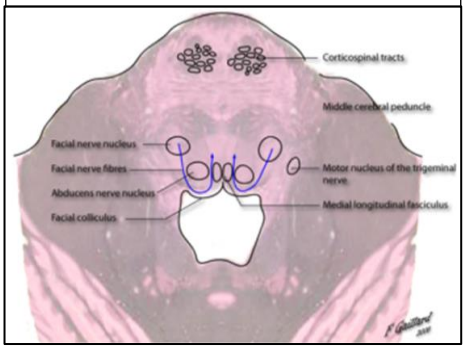


Superior Salivatory Nucleus

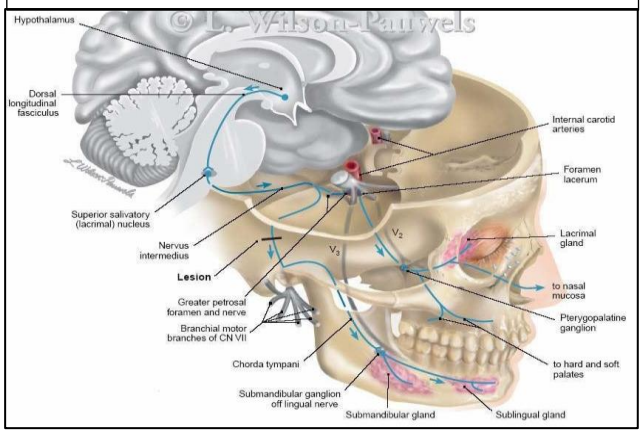




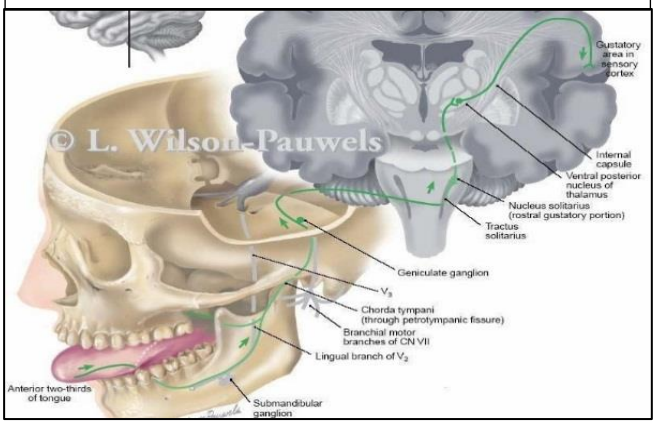
Intracranial

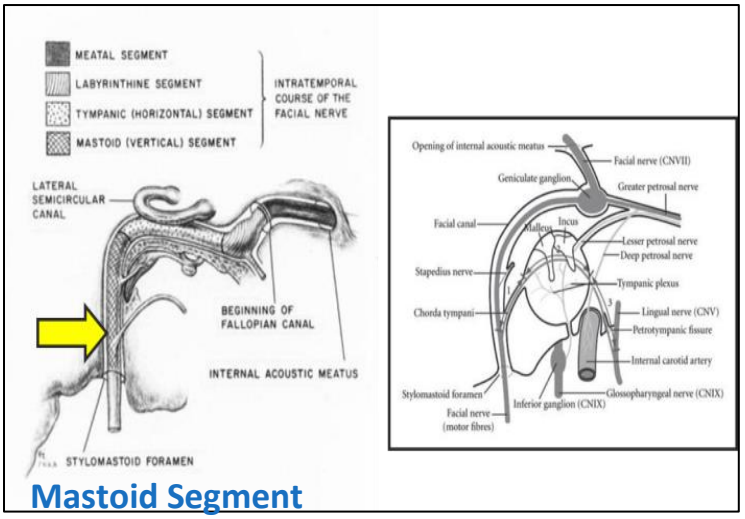


Superior Salivatory Nucleus

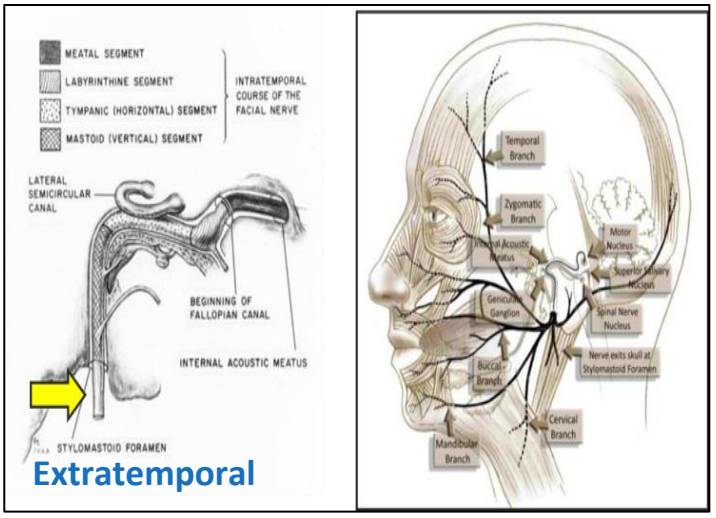


Solitarius

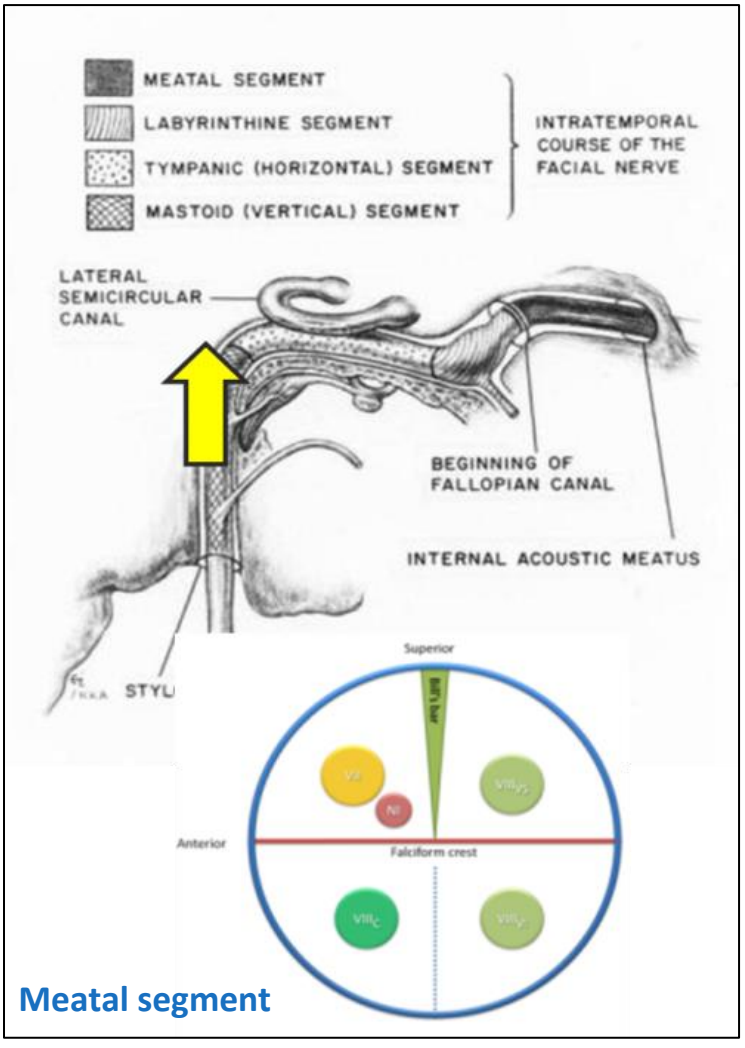




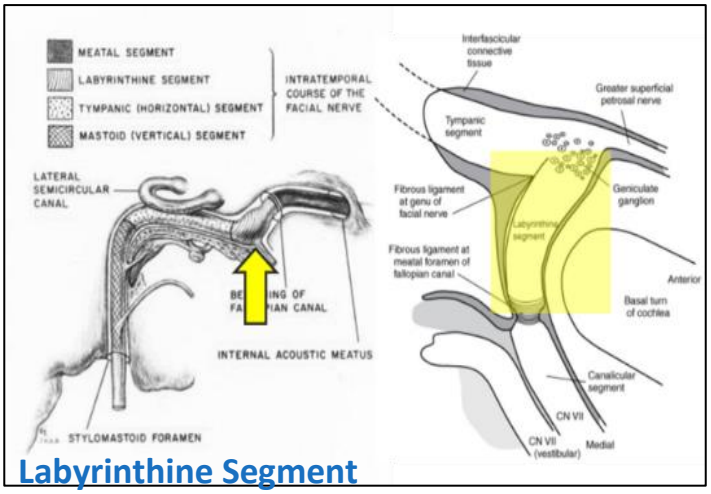
Mastoid Segment



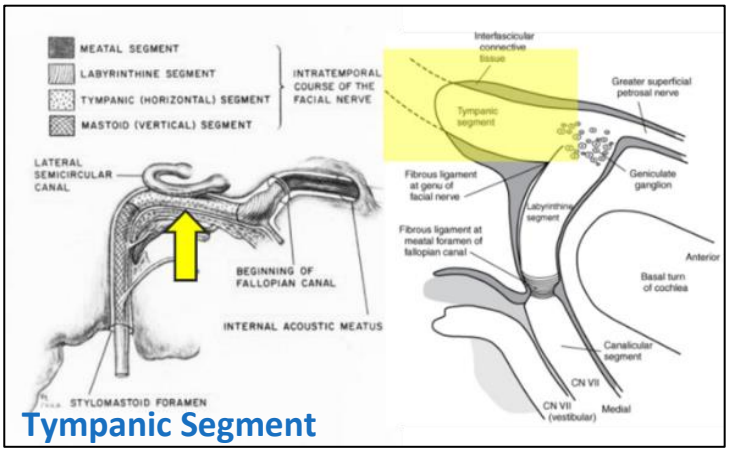
Extratemporal



Meatal segment



Labyrinthine Segment



Tympanic Segment

The **Facial nucleus** is divided into 2 parts:

A. The upper half that receives innervation from <u>both *cerebral cortices*</u>.

B. The lower half that receives innervation <u>only</u> from the contralateral cerebral cortex.

- Lower motor lesions affect all the ipsilateral facial muscles "Lower motor neuron lesion is form the nucleus downward".
- Upper motor lesions spare the upper facial muscles and affect the contralateral lower face because the forehead is innervated bilaterally.
- LMN vs UMN lesions is a possible MCQ

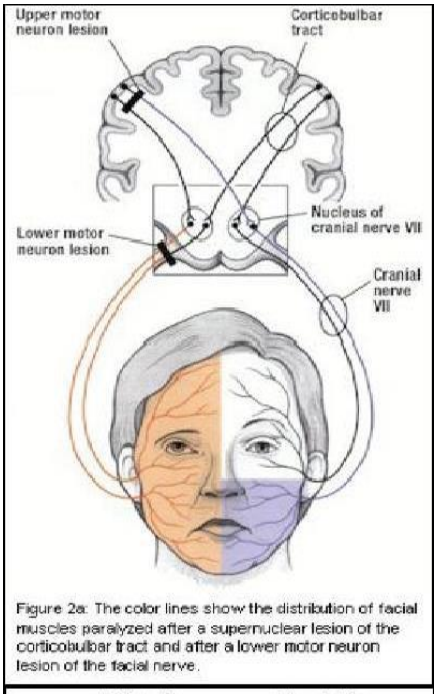
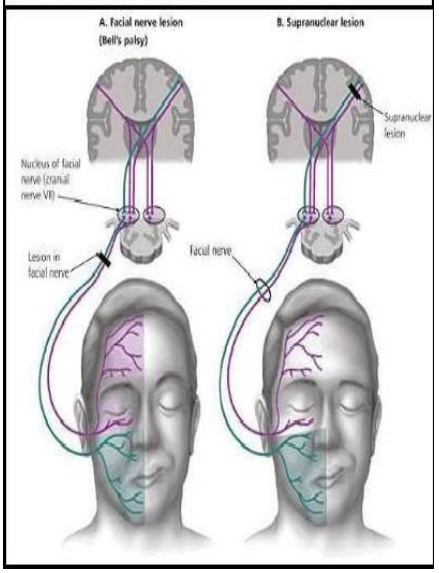
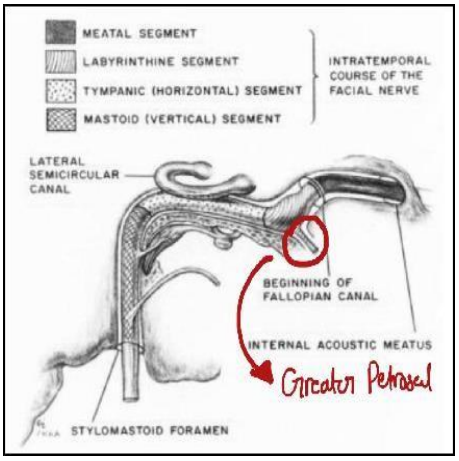


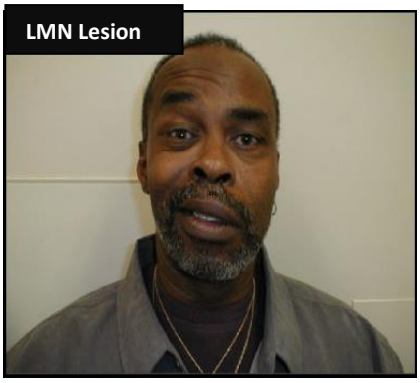
Figure 2a: The color lines show the distribution of facial muscles paralyzed after a supernuclear lesion of the corticobulbar tract and after a lower motor neuron lesion of the facial nerve.



- Facial nerve segments:
  1. Pons.
  2. Cerebulo-pontineangle (CPA).
  3. InternalAuditoryCanal (IAC).
  4. Labyrinthine.
  5. Tympanic. → Fallopian Canal.
  6. Mastoid
  7. External



- Examples:



Whole left side of the face is affected

Only the lower part of the left side is affected

In LMNL the Idea is basically the involvement of frontalis (wrinkles of the forehead) and orbicularis oculi (give you the ability to close the eye), while in UMNL Frontalis and orbicularis occuli are spared.

Anatomical Divisions:

- **Embryology**
  - The facial nerve is developmentally derived from the hyoid arch, which is the second branchial arch
  - It arises as 2 main divisions-motor and sensory
  - The motor division of facial nerve is derived from the basal plate of the embryonic pons.
  - The sensory division originates from the cranial neural crest.

- **Anatomy**

Facial Nerve Segments:

1. Intracranial (cisternal) segment

2. Meatal segment (internal auditory canal): 8mm, zero branches, 7UP

7UP: it is in the anterior superior portion of the IAC and behind it is the vestibulo (sup and inferior) and cochlear

3. Labyrinthine segment: 3-4mm, 3 branches[1] (IAC geniculate ganglion).

Shortest segment. (only segment that lacks arterial anastomosis) → vulnerable to injury → the geniculate ganglion before entering the middle ear gives the first branch (1st genu) → greater superficial petrosal nerve → fibers for lacrimation and salivation

↳**What is the first branch of CN VII? greater superficial petrosal nerve**

↳If there is a lesion in the middle ear after the first branch there will be normal lacrimation and salivation this process is called "Topography"

4. Tympanic segment: 8-11mm, zero branches, 50% dehiscent, (Geniculate ganglion to pyramidal eminence) if you remove the drum you can see the facial nerve passing → importance; any infection or cholesteatoma can easily put pressure on the facial nerve

5. Mastoid segment: 8-14mm, 3 branches, Pyramidal eminence to stylomastoid foramen If a lesion is effecting the stapedius nerve or the chorda tympani it maybe high mastoid or tympanic

6.Extratemporal (extracranial) segment: 15- 20mm, 9 branches, Stylomastoid foramen to the parotid and gets separated into superficial and deep and it ends in 5 major branches.

[1] Stapedial (to stapedius), greater petrosal (to lacrimal glands) and chorda tympani (taste fibers from ant. ⅔ of tongue as well as parasympathetic fibers to the sublingual and submandibular salivary glands)

- The CP angle segments:

↪Facial nerve is in relation with the last 4 cranial nerves.

↪The facial fibers cross the CP angle and pass through the internal auditory canal (**meatal segment**) with vestibulocochlear nerve (8th).

↪7th CN occupies the anterior superior part of the internal auditory canal "7up" (possible **MCQ**).

1. The intratemporal part:

- From the internal auditory meatus or canal it crosses the temporal bone through **fallopian canal** and it is related directly to the inner, middle and external ear.
- It is divided into 3 segments:

1. **Labyrinthine** (IAC to geniculate ganglion) "in the inner ear": Only segment that lacks arterial anastomosis, (embolic phenomena, vascular Compression) **high risk of ischemia** (possible **MCQ**).
2. **Tympanic** (from geniculate ganglion to pyramidal eminence) "in the middle ear"

(50% of the Tympanic part is open in children, that's why they might get Acute otitis media which can lead to facial nerve palsy) (imp for OSCE)

3. **Mastoid or vertical** (from pyramidal eminence to stylomastoid foramen) in the external ear it finally leaves the skull through stylomastoid foramen. (Mastoid has a high risk to get injured during surgery)

Branches are:

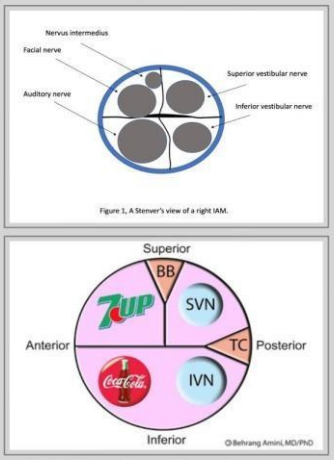
- Stapedius muscle: if get injured patient won't be able to tolerate high sound
- Chorda tympani nerve, which give: 1-Submaxillary, 2-sublingual and Taste anterior2/3tongue, 3- pain, temperature, and touch EAC.
- Auricular nerve

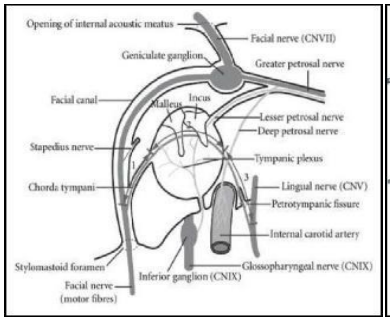
(very imp for MCQ):

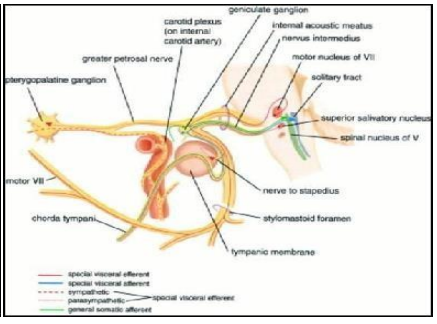
- Labyrinthine segment is the shortest and narrowest part of the facial nerve segments, that's why most of the patients presenting with facial palsy due to compression of this segment.
- Whereas the mastoid segment is the longest → At risk of injury in trauma and mastoidectomy

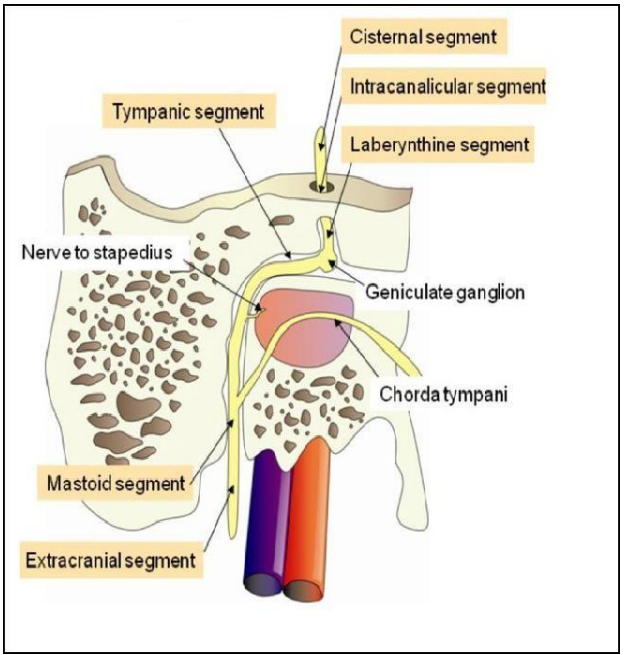
EXTRA

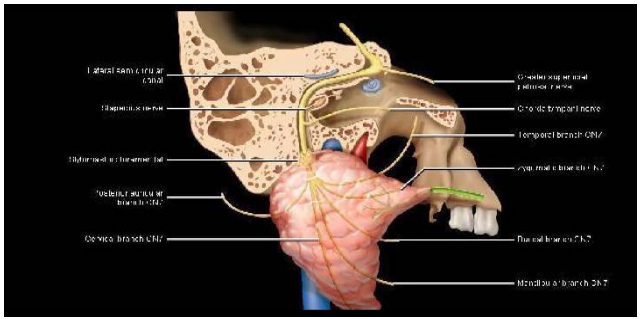
Anatomy of the internal auditory canal:
The internal auditory canal is the bony channel in the posterior fossa, that houses the facial (VII), cochlear and vestibular (VIII) nerves. At the fundus it is split horizontally by the falciform or transverse crest. The superior portion of the canal above the transverse crest is again split into anterior and posterior parts by vertical crest or Bill's bar.



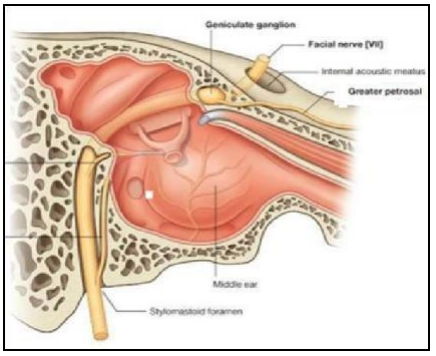




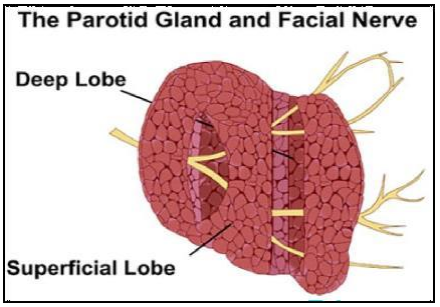




2. The extratemporal (extracranial) part:



- From stylomastoid foramen to division into major branches.
- As soon as the nerve leaves the stylomastoid foramen, it goes within the parotid gland and separates it into superficial and deep lobes (check the figure), Parotid surgeries can cause facial nerve paralysis.
- Then, it branches within the anterior border of the parotid into five terminal branches: (Always in Exam either MCQ or SAQ, know the nerve + the muscles + the functions):



1. **Temporal:** most superior > supplies the frontalis muscle.
2. **Zygomatic:** supplies orbicularis oculi muscle.
3. **Buccal:** supplies buccinators muscle. (if get injured food will accumulate on cheek + weak chewing) remember that the muscles of mastication are supplied by CN V
4. **Mandibular:** supplies the muscles of the angle of the mouth.
5. **Cervical** "long but thin branch": supplies platysma muscle.

- Least important branch is **cervical**.
- There are usually some variations in different branches; some branches may get divided into two and each branch divides into another two etc.

★ Most important 2 branches:

1. **Zygomatic** "to protect the eye" **(imp)**
2. **Mandibular** "its paralysis causes cosmetically bad deformity" **(imp)**

- Commonest surgical procedure that affects the mandibular branch → **Submandibular** salivary gland surgeries, leading to paralysis of the **angle** of the mouth.

Pes anserinus(goose'sfoot)

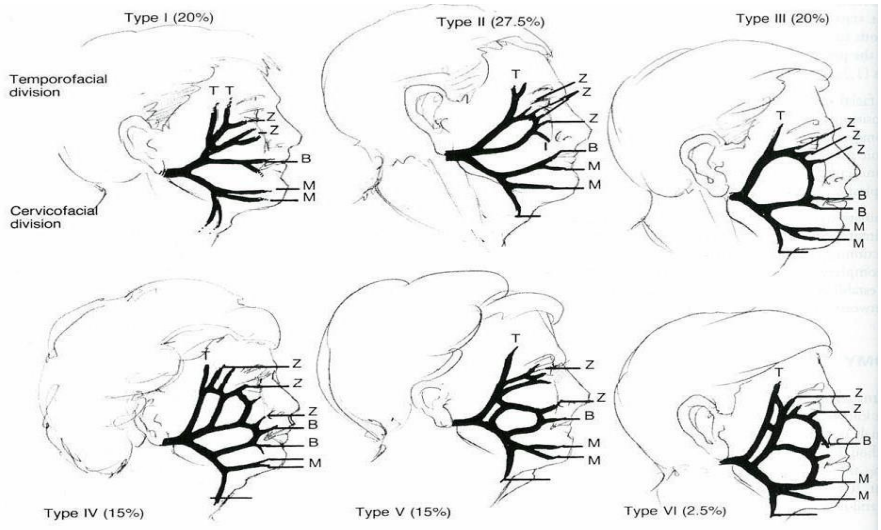


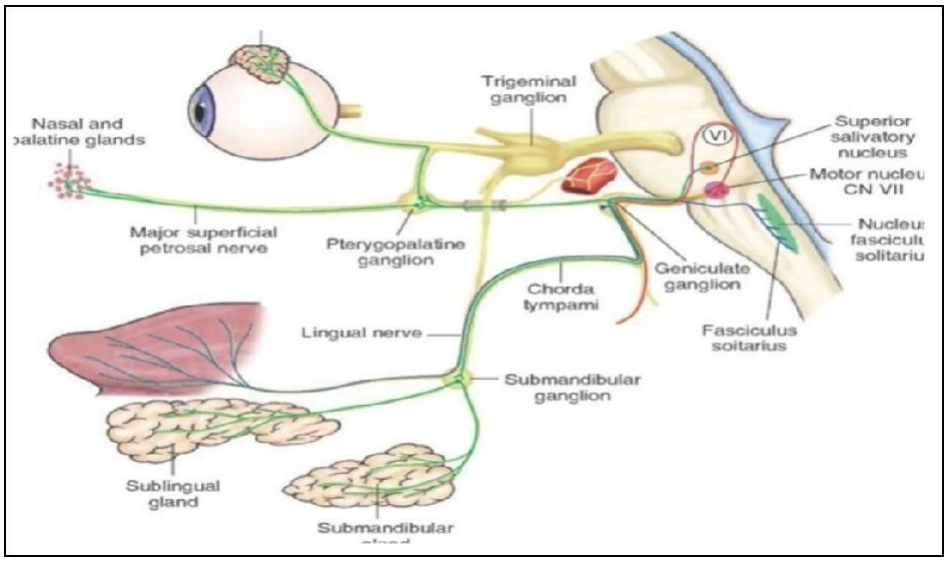
FIGURE 169.2. Terminal branches of the facial nerve, demonstrating its variability. *B*, buccal; *M*, mandibular; *T*, temporal; *Z*, zygomatic.

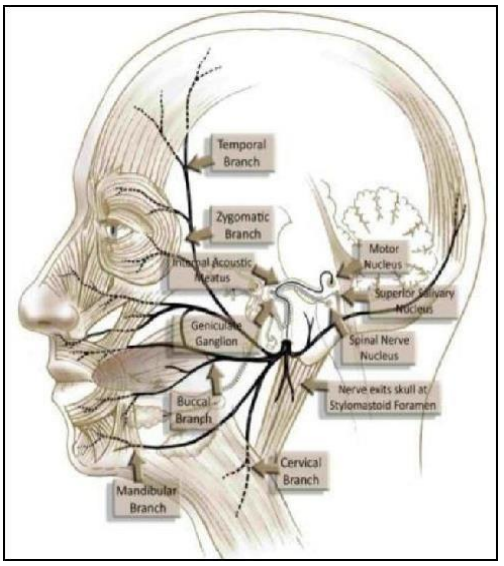
The course of secretomotor and taste

- The secreto-motor fibers leave the superior salivary nucleus with the facial nerve. Some fibers leave the facial nerve in the geniculate ganglion as great petrosal nerve and this supplies the **lacrimal glands**. (if greater petrosal nerve get injured patient will have Dryness) (imp of MCQ)
- The other fibers leave the facial nerve in the chorda tympani and supply the **submandibular** and **sublingual** salivary glands.
- Taste fibers follow the same course but in the other way. Taste fibers from anterior 2/3 of the tongue go through the chorda tympani to the facial nerve and finally to nucleus solitares.
- What happens if there is an injury of the chorda tympani? It easily gets injured because it passes in the inner ear
- Minor defect in the taste "because it affects the anterior 2/3 of only one side of the tongue"
  - ↳ There will be no dryness "because the parotid is supplied by the glossopharyngeal nerve (9th)"
- Function of Facial nerve:
  - •Lacrimation
  - •Salivation
  - •Expression
  - •Speech
  - •Mastication
  - •Hearing

Nerve Fiber Modality	*Nucleus*	*Function*
General sensory (afferent)	Spinal of the trigeminal nerve	To carry sensation from the skin of the concha of the auricle, a small area of skin behind the ear, and possibly to supplement V_3, which carries sensation from the wall of the external auditory meatus and the external surface of the tympanic membrane
Special sensory (afferent)	Solitarius (rostral gustatory portion)	For taste sensation from the anterior two-thirds of the tongue
Branchial motor (efferent)	Motor of cranial nerve VII	To supply the muscles of facial expression (ie, frontalis, occipitalis, orbicularis oculi, corrugator supercilii, procerus, nasalis, levator labii superiorus, levator labii superioris alaeque nasi, zygomaticus major and minor, levator anguli oris, mentalis, depressor labii inferioris, depressor anguli oris, buccinator, orbicularis oris, risorius, and platysma). In addition, the branchial motor fibers supply the stapedius, stylohyoid, and posterior belly of digastric muscles
Visceral motor (parasympathetic efferent)	Superior salivatory (lacrimal)	For stimulation of the lacrimal, submandibular, and sublingual glands as well as the mucous membrane of the nose and hard and soft palates

Named Branches	*Muscles Supplied*
Nerve to stapedius	Stapedius
Nerve to posterior belly of digastric	Posterior belly of digastric
Nerve to stylohyoid	Stylohyoid
Temporal	Frontalis, occipitalis, orbicularis oculi, corrugator supercilii, procerus
Zygomatic	Orbicularis oculi
Buccal	Buccinator, orbicularis oris, nasalis, levator labii superioris, levator labii superioris alaeque nasi, zygomaticus major and minor, levator anguli oris
Mandibular	Orbicularis oris, mentalis, depressor anguli oris, depressor labii inferioris, risorius
Cervical	Platysma
Posterior auricular	Occipitalis

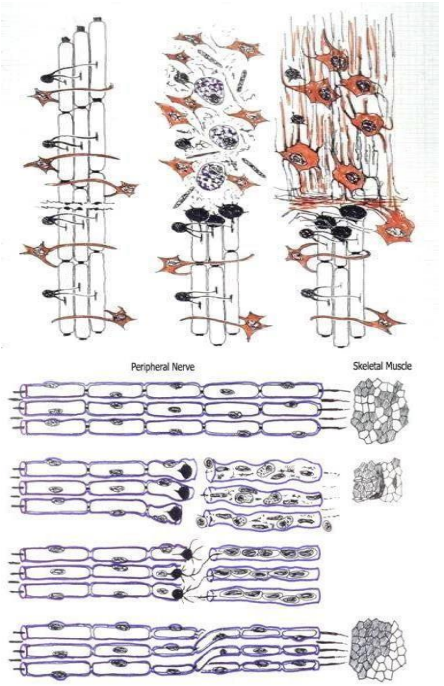




Variations Anomalies:

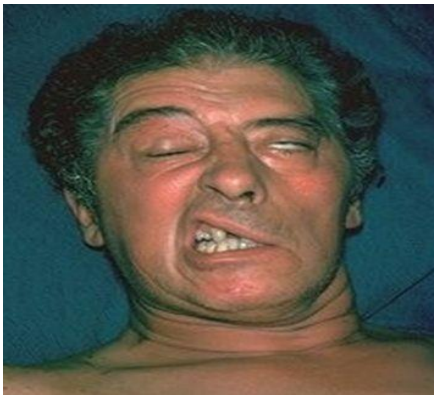
- Dehiscence: a defect in the fallopian canal. (The Nerve is Exposed inside the middle ear).
- Fallopian canal is a bony canal through which the facial nerve passes inside the temporal bone.
- Mainly congenital, when there is a deficiency of the bone, thus the nerve will not be covered by a bone and lies immediately in the middle ear. 50% of people are dehiscent.
- Becomes more subjected to trauma and infection leading to facial nerve paralysis. (when they have otitis media they get facial paralysis).

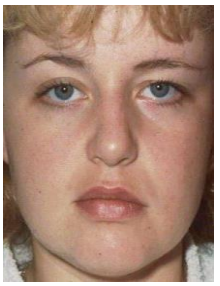
Facial nerve Palsy:



- Degeneration.
- Metabolic source (cell body).
- Wallerian degeneration:
  - Begins within 24 hours
  - Degeneration distal axon & myelin sheath
  - Distal to the site of an injury.
  - Without local Inflammation.
- Macrophages degrade myelin and axons
- Regeneration
- Axonal stumps swell and proliferating neuro-filaments
- Misdirected regrowth of nerve fibers
- Facial muscle contractures >> Synkinesia
- Salivation>>crocodile tears.

Facial Nerve Paralysis:





Clinical manifestations

- Paralysis of facial muscles:
  - Asymmetry of the face.
  - Inability to close the eye. Orbicularis oculi
  - Accumulation of food in the cheeks. Paralysis of buccinators.
- **Lower motor neuron lesion** of the left side: (upper picture)
  - No wrinkles in the forehead when looking up due to failure of contractions of frontalis muscle
  - Inability to close the eye completely "most accurate sign"
  - Flattening of the nasolabial fold
  - Angulation of the mouth when showing the teeth "the angle goes to the other side"
- **Upper motor neuron lesion** of left side: Looks normal at rest (lower 2)
  - The orbicularis oculi and frontalis muscles will not be affected.

- **Other manifestations of facial nerve paralysis:**
  - Phonophobia due to failure of strapedius atteniuation reflex, uncomfortable feeling in exposure to loud sounds.
  - Acoustic reflex (stapedial reflex) is a useful tool to localize the lesion; if intact the problem is distal to it and vice versa.
  - Dryness of the eye; Some people present with lacrimation and others present with dryness. Why?
    - Lacrimation is due to paralysis of **orbicularis oculi** as this muscle help in draining the tears.
    - Dryness is due to affection of **greater petrosal nerve** which arise from geniculate ganglion.
    - So if the paralysis is above the level of geniculate ganglion > dryness
    - If below it > no dryness.
  - Loss of taste; very little just in the ant. ⅔ of one side. They feel a metallic taste.

House-Brackmann is the most useful classification in facial nerve palsy: (just know the name)

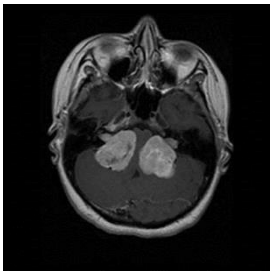
House-Brackmann	
Grade 1	Grade 2-3
Neurapraxia Spontaneous recovery	Axonotmesis Flow interruption. Wallerian anterograde degeneration. Incomplete degeneration
Grade 4	Grade 5-6
Neurotmesis (permanent loss of axons) Demyelinization Moderate to severe facial musculature dysfunction Regenerative>> synkinetic movements	Partial or complete transection of the nerve Minimal/complete loss of function

Clinical examination and history

- **Hearing loss or vertigo**
- **Timing**
  - Sudden onset, aggressive? Or slow?
  - Evolution over 2-3 weeks
- **Presence of ear disease** (Pics 3+5)
  - Chronic otitis media
  - Cholesteatoma
- **Vesicular eruption** (pic 2)
  - Ramsay-Hunt syndrome
- **Ask the patient to:**
  - Look up to test frontalis.
  - Close eyes to test orbicularis oculi.
  - Blow the cheek to test buccinators.
  - Show the teeth for angulation.
- **Bilateral:** (Pics 4+6)
  - Guillain-Barre syndrome.
  - Lyme disease.
  - Intracranial neoplasm.
  - Neurofibromatosis
- **Recurrent:** (Pic 1)
  - Melkersson-Rosenthal syndrome[2]

(2)

(1)



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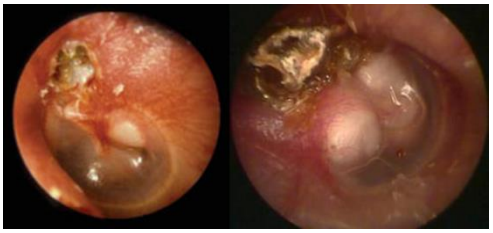


(3) Chronic otitis media

(6)

(5) Cholesteatoma

House-Brackmann Scale

(just know the name)

Grade 1: Normal.

Grade 2: Slight weakness.

Grade 3: Patient Can Close the Eye.

Grade 4: Patient Can't Close the Eye.

Grade 5 and 6: Masked Face, Asymmetric.

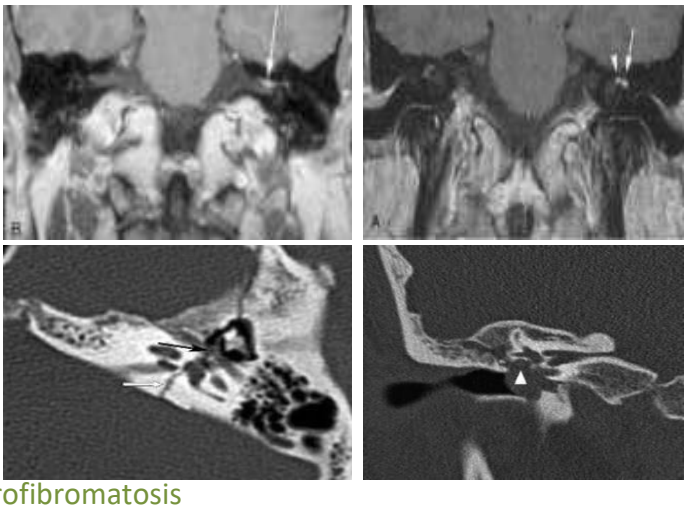
Grade	Appearance	Forehead	Eye	Mouth
I	normal	normal	normal	normal
II	slight weakness normal resting tone	moderate to good movement	complete closure minimal effort	slight asymmetry
III	non-disfiguring weakness normal resting tone	slight to moderate movement	complete closure maximal effort	slight weakness maximal effort
IV	disfiguring weakness normal resting tone	none	incomplete closure	asymmetric with maximal effort
V	minimal movement asymmetric resting tone	none	incomplete closure	slight movement
VI	asymmetric	none	none	none

In History of Facial nerve palsy the most important question is WHEN DID IT START?

Because if it starts from weeks and it's gradual you have to think of Malignancy

[2] A rare neurological **disorder** characterized by recurring facial paralysis, swelling of the face and lips (usually the upper lip cheilitis granulomatosis) and the development of folds and furrows in the tongue (fissured tongue)

Investigations



Radiology: To localize lesion

- **Computed tomography** (intratemporal)
  - Trauma
  - Mastoiditis
  - Cholesteatoma
- **Magnetic resonance imaging (MRI)** (intracranial)
  - Nerve enhancement
  - Exclude neoplasm: vestibular schwannoma or neurofibromatosis

Usually MRI enhancement in labyrinthine segment.

Topography (Topognostic tests): could beMCQs

- Indicated in some cases to determine the level of the lesion:

1. Schirmer's test; test lacrimation function →greater superficial petrosal.

   Put two tapes in the eyes and check if they're equal on both sides, if one eye is tearing and the other isn't; lesion proximal to this branch.

2. Stapedial reflex →stapedial branch with tympanometry
3. Taste sensation (Electrogustometry) →chorda tympani.
4. Salivary flow →chorda tympani.

   Collect from both sides and compare the amount; If lacrimation is involved then the lesion is most likely proximal to the geniculate ganglion (before the greater superficial petrosal nerve branch)

Audiology:

- Evaluate for pathology of eighth cranial nerve
  - **Bell's palsy:**
    - Symmetric audiological function (normal).
    - Absent ipsilateral acoustic (stapedial) reflex.
  - Retrocochlear pathology
    - Asymmetrical thresholds.

Evaluate for retro cochlear pathology (e.g. neoplasm) with either ABR or MRI. The facial nerve might be affected secondary to a lesion

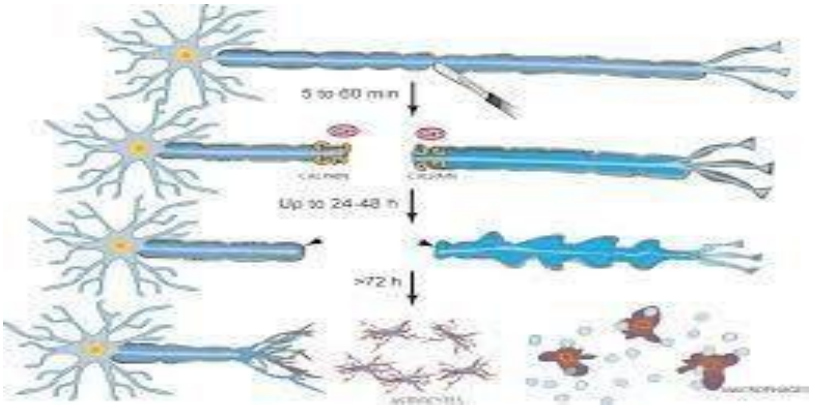
Electrophysiology:

- It detects degeneration of the nerve fibers
- Useful only 48-72 hours following the onset of the paralysis. Provides prognostic information.
- If the nerve is stimulated distal to the injury in the first 2-3 days > there will be a response in all cases.
- After 3 days > there will be no response in case of degeneration.
- Electrophysiological tests: Provides prognostic information

  Principle: stimulate the nerve and look for response:

  - Nerve Excitability Test (NET)
  - Electroneurography (ENoG)
  - Electromyography (EMG) tests the strength of the muscle, important for prognosis
  - Maximum stimulation test (MST)

    ACUTE=Acute+Complete+Unilateral+Threedays+Evaluate



Nerve Excitability Test (NET)	Electroneurography (ENoG)
• Stimulate the nerve in the stylomastoid foramen and compare both sides. • The current's thresholds required to elicit just-visible muscle contraction on the normal side of the face are compared with those values required over corresponding sites on the side of the paralysis.	• The amplitude of action potentials in the muscles induced by the maximum current is compared with the normal side; and used to calculate the percentage of intact axons. More objective

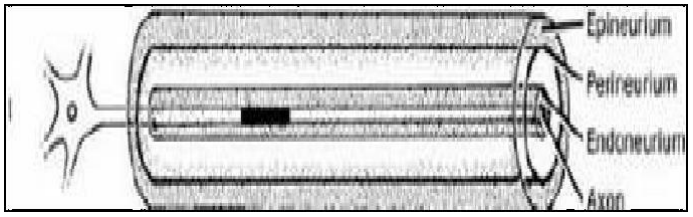
Interpretation of the tests:

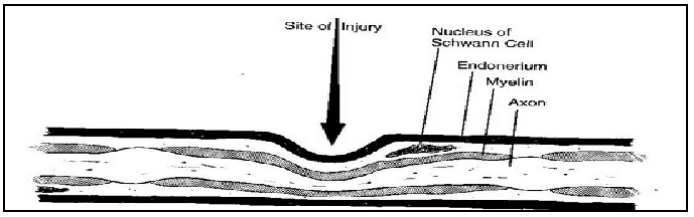
- Not useful in the first 48-27 hours.
- After 48-72 hours (the time required for degeneration to take place):
- Normal results > no degeneration (neuropraxia)
- Abnormal result > degeneration.

Pathophysiology of Nerve Injury:

Neuropraxia (conductive block):

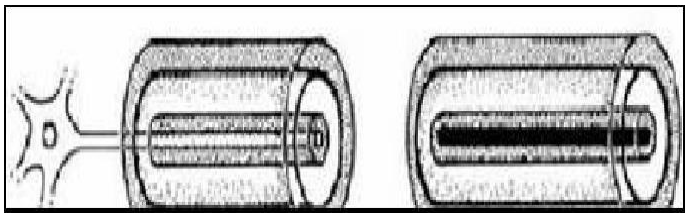
- In cases of mild trauma causing only functional block of the facial nerve, the fibers keep their integrity.
- In Regeneration: there will be restoration of full function if the cause is treated.

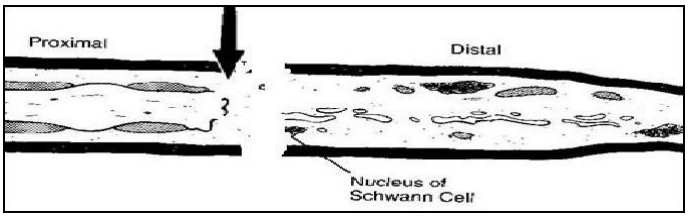




Neurotmesis (degeneration):

- Anatomical block due to complete transection of the facial nerve. Wallerian degeneration of the distal part of the nerve, and this takes 2-3 days to occur.
- In Regeneration: no recovery unless the distal and proximal parts are approximated.
- The recovery here is delayed and usually incomplete "why?" The rate of growing is 1mm/day or 1 inch/month > it will be delayed (It takes the fibers approximately 2-3 months to reach the angle of the mouth if the injury is in the temporal bone). Not all the fibers of the proximal part will reach the distal > it will be incomplete.





- After regeneration, sometimes **misdirection** of the fibers occurs: the fibers that go to the salivary gland deviate to sweat gland "the patient sweats when he eats" Or to lacrimal gland "patient tears when he eats (crocodile tears)"

Principles of Management of facial nerve injury:

- Care of the eye the patient is unable to close his eye so the cornea will be exposed to trauma

  Protect it by:

  1. Artificial tears if the patient has dryness.
  2. Protect them from dust by wearing sunglasses
  3. See ophthalmologist in case of any irritation
  4. Cover the eye while sleeping
- Treatment of the cause if applicable.
- Treatment of the nerve varies according to the degree of the paralysis.
- Partial facial paralysis:
  - Being partial means that some of the nerve fibers are in continuity. Recovery is expected by conservative treatment (e.g. removal of pressure, steroid etc.). No need for surgical intervention.
- Complete facial paralysis:
  - Complete paralysis may be a result of neuropraxia or/and degeneration.
  - If it is due to neuropraxia, recovery is expected by conservative treatment.

  If it is due to degeneration, surgical treatment is required.
  - To differentiate between degeneration and neuropraxia electrophysiological tests are required.

Complications of Facial Paralysis

The most significant complication is the social isolation these patients.
Facial paralysis severely affect:

- Normal facial expressions
- Mastication
- Speech production
- Eye protection

Causes of facial paralysis:

According to the anatomy:

- Intracranial causes "brain tumors and neurosurgical trauma".
- Cranial (intratemporal) causes "middle ear infection or trauma".
- Extracranial causes "parotid tumors".

According to the cause itself:

- Congenital: Birth trauma.
- Traumatic: Head and neck injuries & surgery parotid, mastoid and intracranial surgeries.
- Inflammatory: O.M, Necrotizing O.E., Herpes.
- Neoplastic: Meningioma, malignancy of the ear or parotid.
- Neurological: Guillain-Barre syndrome, multiple sclerosis.
- Idiopathic: Bell's palsy "most common", Melkersson Rosenthal, Gullian Barre, MS, Mysethenia gravis, Sarcoidosis(Heerfordt's).
- Iatrogenic:
  - Parotid surgery.
  - Mastoid surgery.
  - Local anesthesia.
  - Acoustic neuroma.

A- Inflammatory Causes of Facial Paralysis

1. Facial paralysis in Acute Otitis Media (AOM) (imp for MCQ):

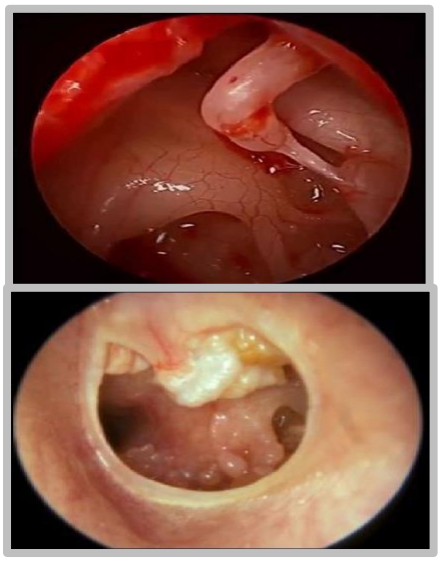
- Mostly due to pressure on a **dehiscent** nerve by inflammatory products and fluid accumulation.
- Usually is partial and sudden in onset.
- Treatment: is by antibiotics and myringotomy; open the drum and drain the fluid.
- Mastoiditis can cause pressure on the nerve.
- 3 days history of fever, earache and facial palsy = AOM

Otitis media

The right side is paralyzed, the left has facial expressions; the mouth is pulled upwards

Mastoiditis

2. Facial paralysis in Chronic Suppurative Otitis Media (CSOM):



- Usually is due to pressure by cholesteatoma or granulation tissue causing bony erosion.
- Insidious in onset -slow onset- long history of deafness and discharge.
- May be partial "if detected early" or complete.
- Treatment is by immediate surgical exploration and "proceed" Mastoidectomy; remove the cholesteatoma and repair the nerve.

3. Herpes Zoster Oticus (Ramsay Hunt Syndrome):

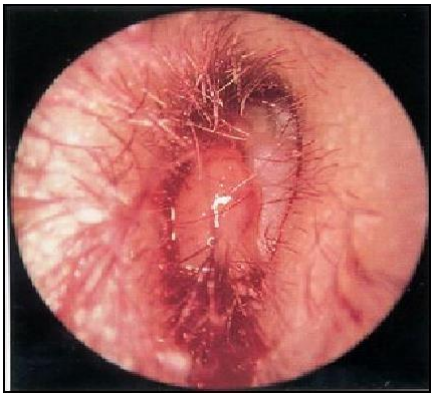
- Herpes zoster affection of cranial nerves VII, VIII, and cervical nerves.
- Symptoms: Facial palsy, severe pain, skin rash, SNHL (sensory neural hearing loss) and vertigo.
- The patient is usually elderly, and severe pain precedes the facial palsy.
- The characteristic clinical feature is a vesicular eruption in the ear (sometimes on the tongue and palate).
- Treatment by: Acyclovir, steroid and symptomatic.

  - Vertigo improves due to compensation from the other side "takes few weeks".
  - SNHL is usually irreversible.
  - Facial nerve recovers in about 60%. Recovery of facial nerve function is much less likely than in Bell's palsy.

(you will be asked about it in exam, patient has facial palsy and skin rash and Ear symptoms it's Ramsay hunt syndrome)

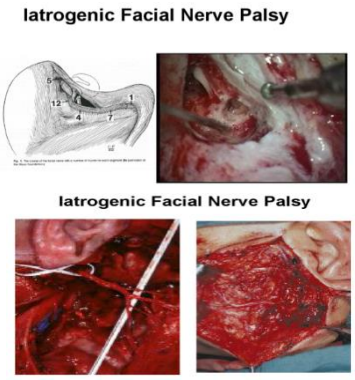
4. Malignant Otitis Externa:

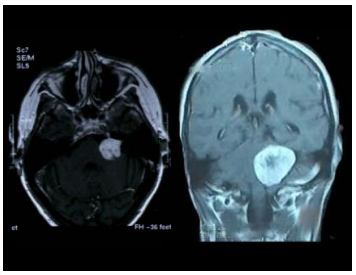
- Osteomyelitis of the temporal bone (osteomyelitis of the external auditory canal bone).
- Granulation tissueobscured TM
- It could affect multiple nerves other than 7th if it reaches the jugular foramen (9th 10th 11th)
- Very aggressive, which is why it was thought to be malignant



  4Ds:
  - Diabetes mellitus or anything considered immunocompromising
  - Discharge (purulent)
  - Discomfort
  - Dysfunction cranial nerve

B- Traumatic Facial Injury:



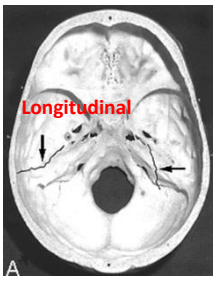


Vestibular schwannoma

1. **Iatrogenic:** Operations at the CP (cerebellopontine) angle, ear and the parotid glands. acoustic neuroma resection or mastoidectomy.

2. **Birth trauma (congenital facial palsy):**
   - 80-90% are associated with birth trauma
   - 10 -20 % are associated with developmental lesions
   - Most of them are partial and need only conservative management

3. **Temporal bone fracture** (possible **MCQ**):

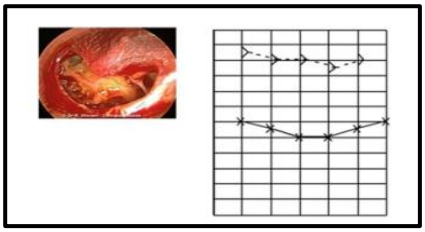


A- Longitudinal:
   - 80% of Temporal Bone Fractures.
   - 15-20% Facial Nerve involvement.

B- Transverse:
   - 20% of Temporal Bone Fractures.
   - 50% Facial Nerve Involvement (more likely to cause paralysis)

Most common cause of facial nerve palsy in temporal bone trauma is transverse temporal bone fracture

It is important to differentiate between the two.



- **Signs for temporal bone fracture:**
  - CSF or blood leak from ear.
  - Raccoon eyes sign.
  - Battle's sign.
  - Ossicles injury.

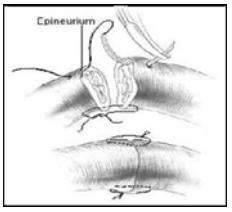
- **Pathology: Edema or transection of the nerve.**

- **Management** of traumatic facial nerve injury:
  - If it is delayed in onset, it is usually incomplete and is due to edema → Conservative "steroids and relieve the pressure"

  - If of immediate onset, it is usually complete and due to transection of the nerve
    → Surgical repair
    → If borderline; conservative.

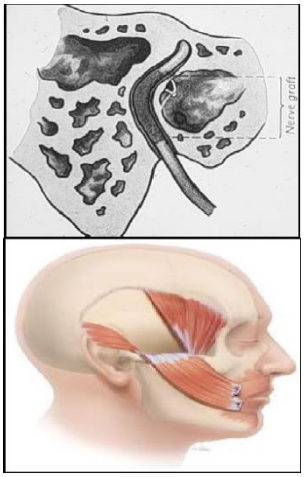
- **Surgical Repair:**

1. **Direct Anastomosis**:
   - If the proximal and distal parts are identified and no distance between them.



2. **Nerve Graft:**
   - If there is a distance between them
   - Most common nerve used is great auricular nerve; it can give up to 10 cm and has the same thickness of facial nerve.
   - Sural nerve.
   - If the injury is in the temporal bone, sometimes we graft the nerve in the fallopian canal without stitching
   - But if outside > we must stitch it.

1. **Nerve Transfer** (anastomosis):
   - If the proximal part can't be identified, get a nerve and connect it to the distal part.
   - Most common nerve used is hypoglossal nerve[3] IF the other one is healthy and functioning well, because bilateral hypoglossal nerve damage is catastrophic.
2. **Muscle flap:**
   - If the distal and proximal parts can't be identified.
   - For cosmetics only "temporalis or masseter muscles are used".



C- Bell's Palsy

- Bell's palsy is a lower motor neuron facial palsy of unknown cause, but thought to be viral. (Will come in the exam)
- Bell's palsy may be complete or incomplete; the more severe the palsy, the worse the prognosis. In practice, full recovery may be expected in over 90% of cases.
- The remainder may develop persistent paralysis and other complications including ectropion (weakness of the muscles of the lower eyelid causing persistent overflow of tears) or an aberrant sequence of movements of the face (synkinesis[4]).
- CT or MRI scanning may be needed if the symptoms persist or a specific cause (i.e. other than Bell's palsy) is suspected.
- Electrodiagnosis is used in the assessment of the degree of involvement of the nerve and includes nerve conduction tests and electromyography. These tests are done in a specialist center and be invaluable in predicting prognosis.
- Most common diagnosis of acute facial paralysis, if slowly progressive it is NOT Bell's palsy.
- Diagnosis is by exclusion.

[3] Interestingly, patients have to learn how to control facial motility through the use of tongue voluntary movements

- **Pathology:** Edema of the facial nerve sheath along its entire intratemporal course (Fallopian canal)
  if mild edema → neuropraxia, if severe → degeneration.

- **Etiology**: Vascular or viral measles, cold weather but the exact etiology is still unknown (not proven)

- **Clinical features:**
  - Sudden onset unilateral LM FP. Occurs after exposure to cold weather could be vascular spasm. Pain behind the ear → few hours later facial paralysis.
  - Partial or complete.
  - No other manifestations apart from occasional mild pain behind the ear. No discharge, no parotid swelling, not following trauma.
  - May recur in 10% (6 – 12%), previous history of paralysis in the same side 12%, other side 6%.
  - Risk factors: family history and pregnancy.

- **Prognosis:** if left untreated
  - 80% complete recovery.
  - 10% satisfactory recovery.
  - 10% no recovery.

  Partial usually recovers within 4-6 weeks while complete may take up to 6 months.

  Surgery is not usually done because most of patients recover with conservative treatment

- **Treatment:**
  - Reassurance.
  - Eye protection.
  - Physiotherapy.
  - Medications (steroids; to decrease edema, antivirals, vasodilators)
    Antiviral and vasodilators only given in combination with steroids, not effective alone.
  - Surgical decompression in selected cases:
    ↳ Patients with 90% degeneration. ↳ Within 14 days of onset.

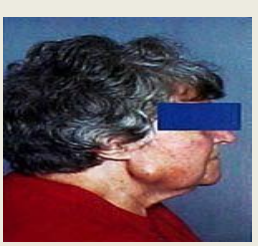
[4] E.g. squinting when smiling.

These are extra slides taken from doctor Badi Aldosari slides with notes:

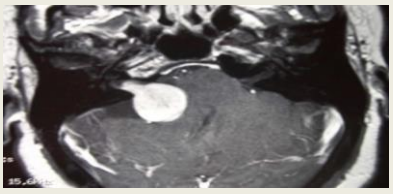
Neoplastic Cause of facial nerve Palsy
- Malignant parotidlesion
- Cholesteatoma
- Acoustic neuroma
- CN VII tumor
- Meningioma.

❖ **SPORT** Neoplasm:
- **S**lowly progressive
- **P**ersistent>4months
- **O**ther C.N. Ex SNHL
- **R**ecurrent.
- **T**umor History.

❖ Functional deficit of facial nerve palsy:
- Lagopthalmos and ectropion
- Oral incompetence
- Nasal obstruction
- Mastication difficulties
- Articulation difficulties
- Often severe psychological distress.



Acoustic neuroma



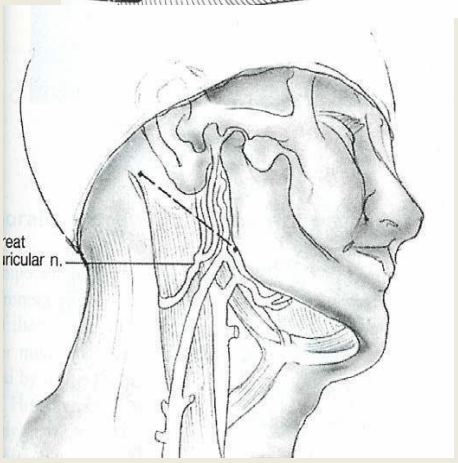
❖ Treatment:

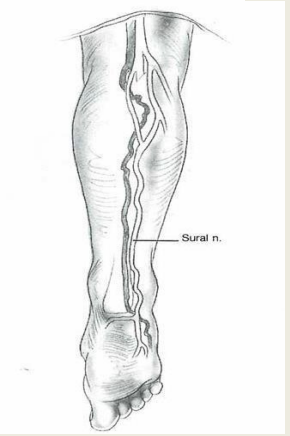
■ **Dynamic Reanimation**

1. Primary repair
2. Interposition nerve grafts
3. Crossover reinnervation procedures (Ansahypoglossi, Hypoglossal & Cross-facial)
4. Regional musclet ransfer (Temporalis, Masseter & Digastric)
5. Microneuro vascularfree-flap (Gracilis, Latissimus dorsi& rectus abdominis)

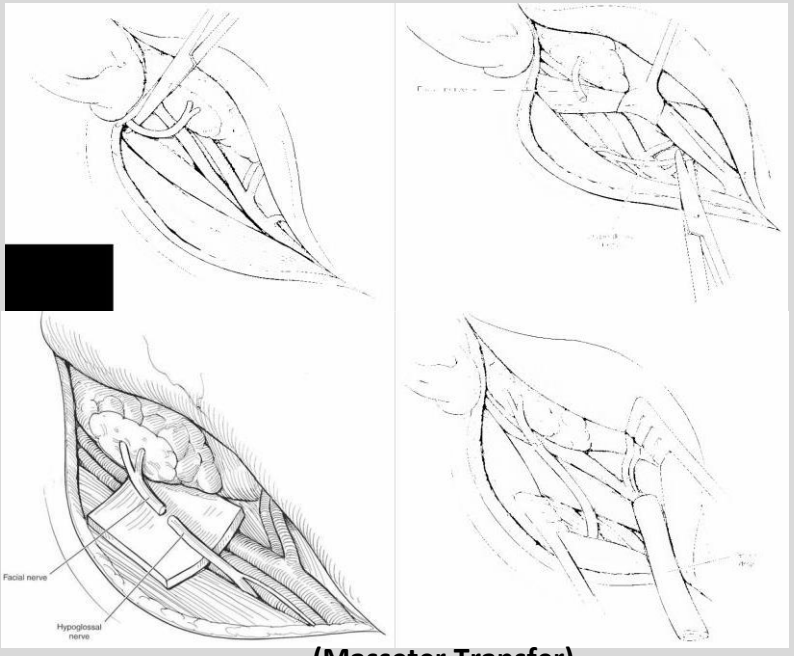
■ **Restore neural input:**
- Primary nerve repair
- Preformed immediately
- Small gap(<17mm)
- Epineural or perineural
- Magnification
- No tension
- Best outcome Expect HB III

■ **Static Reanimation:**
1. Brow and fore head lift Eyelid procedures (Gold weight, Spring & Lower lid tightening)
2. Correction of mid facial deformity (Fascialata, Alloplastic sheets & Face lift)
3. Lower lip wedge resection
4. Botulinum toxin



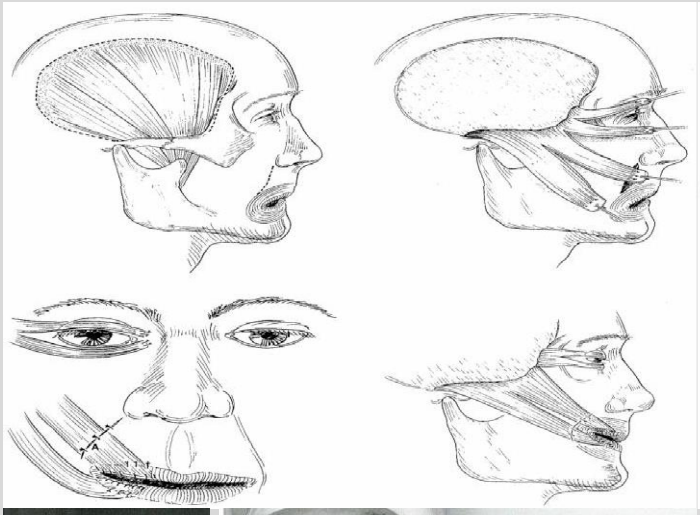


❖ **Hypoglossal-facial crossover:**

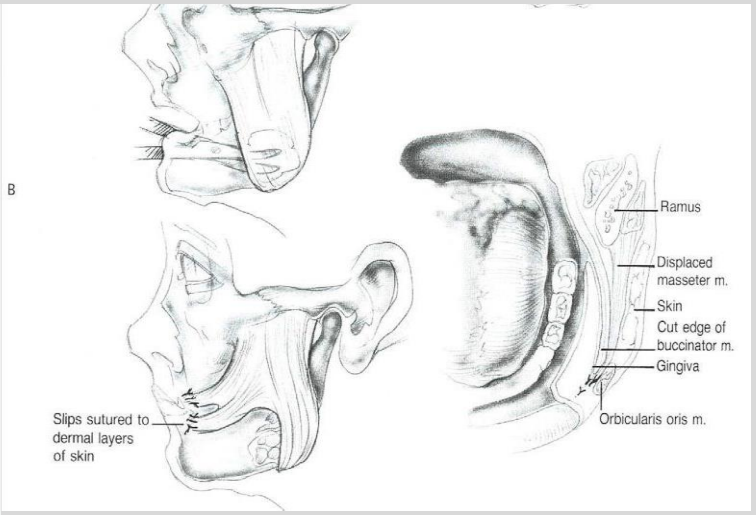


❖ **Muscle transposition:**

(Temporalis Transfer)



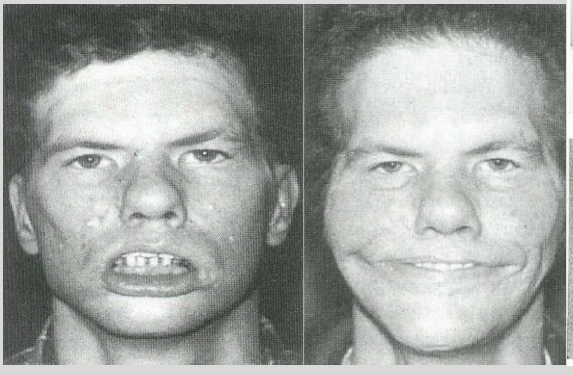
(Masseter Transfer)

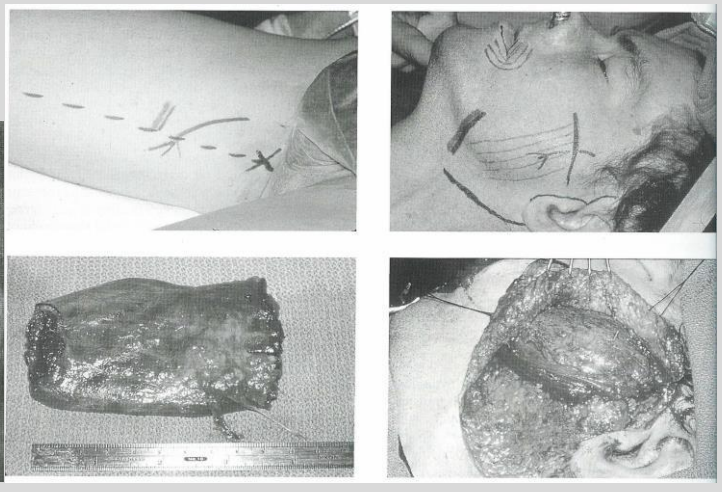


Free Gracilis

Nonfunctional facial muscles:

Free gracilis trigeminal innervation





❖ BotulinumToxin

•Synkinesis and hypertonia

•Advantages

- Ease of use
- Selective

•Disadvantages

- Temporary
- Repeated every 3 months.

Conclusion:

Facial paralysis sequlae (significant)

•Functional

•Cosmetic

•Psychological

The primary goals of facial reanimation

•Corneal protection

•Symmetry at rest

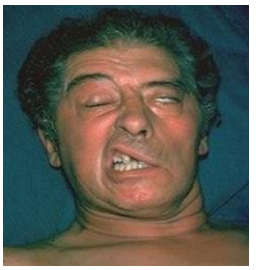
•Smile restoration

Static Reanimation

Questions from the doctor's slides:

What is the most likely diagnosis?

Left lower motor neuron facial paralysis (most likely bell's palsy).



Mention 2 common causes?

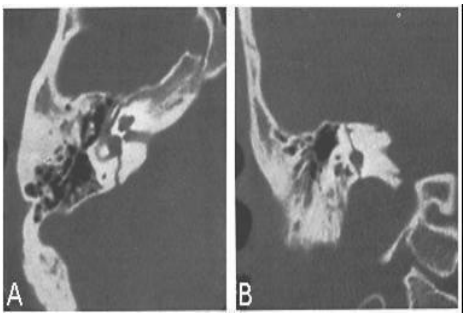
- Bell's palsy (most common)
- Temporal bone fracture
- Acute otitis media

---

36 years old man with RTA:

What is your diagnosis?

Transverse fracture of the temporal bone.



Mention 2 clinical findings?

- Facial nerve paralysis
- CSF leak
- Ossicles injury

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34 years old with LMN facial paralysis:

What is your diagnosis?

Herpes Zoster Oticus (Ramsay Hunt syndrome).

What is your management?

- Acyclovir
- Steroids
- Physiotherapy

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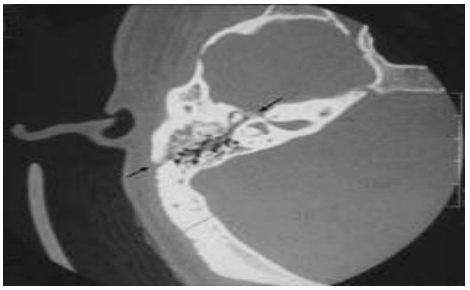
24 years old man involved in RTA: What is your diagnosis?

Longitudinal fracture of the temporal bone.



Mention 2 other clinical findings?

- Facial nerve paralysis
- CSF leak
- Ossicles injury