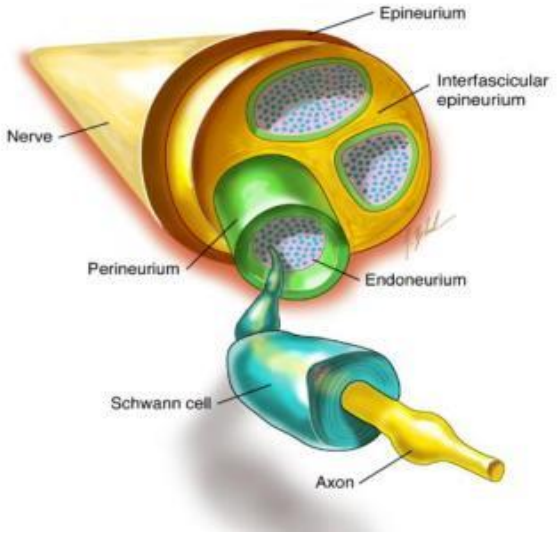
FACIAL NERVE VII



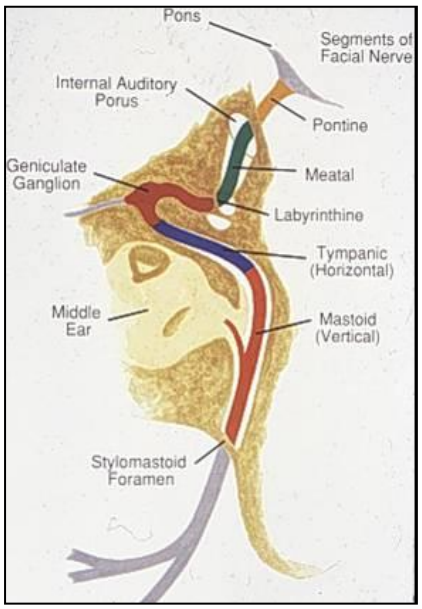
Objectives: *Anatomy (course + branches) + *Causes of facial palsy (congenital, traumatic, middle ear complication , Ramsy Hunt's syndrome and bell's palsy)

Sources: the same as 429 lecture notes with the addition of some pictures from the doctor's slides.

GOOD LUCK..

ASMA ALHEDAITHY

The Facial Nerve VII



FACIAL NERVE FIBERS:

- **Motor** > to the stapedius and facial muscles.
- **Secreto-motor** > to the submandibular, sublingual salivary glands and to the lacrimal glands.
- **Taste** > from the anterior two thirds of tongue and palate (Chorda Tympani).
- **Sensory** > from the external auditory meatus.

ANATOMICAL DIVISIONS:

- **Intracranial :** includes Nuclei+ Cerebellopontine angle
- **Cranial** (intratemporal)
- **Extracranial** (extratemporal) : after leaving stylomastoid foramen.

(Course of Motor fibers)

1. Intracrainal part:

It has the nuclei WHICH ARE:

- Superior salivary nucleus (**Parasympathetic)**
- Motor Nucleus (most part of the facial nerve)
- Nucleus tractus solitarii (Solitary Nucleus) > (**taste**)

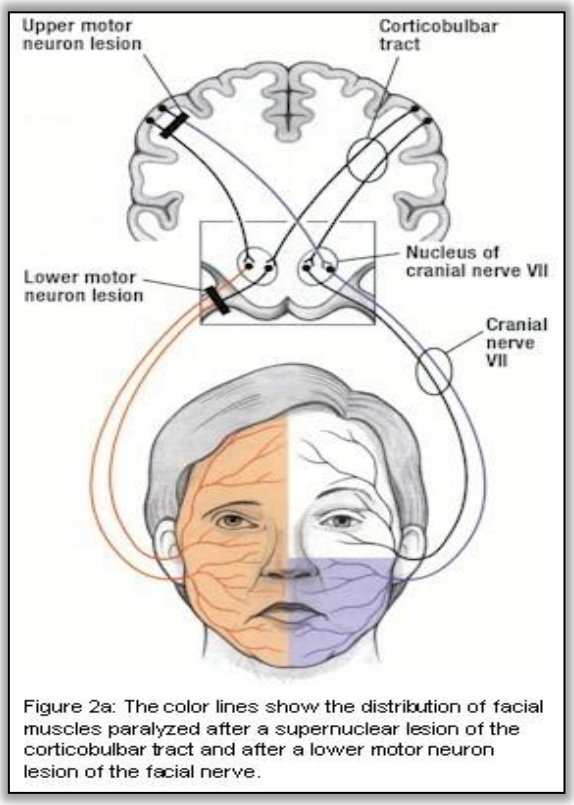
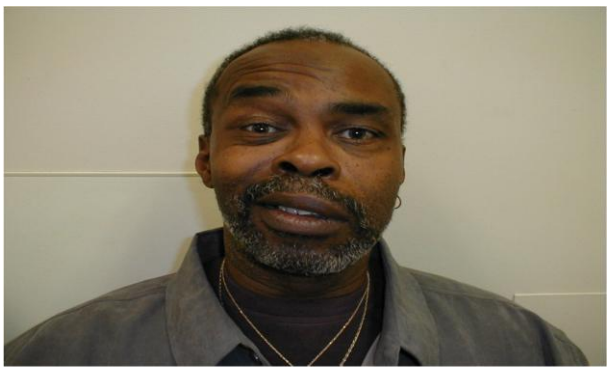


Figure 2a: The color lines show the distribution of facial muscles paralyzed after a supernuclear lesion of the corticobulbar tract and after a lower motor neuron lesion of the facial nerve.

---Upper part of the motor nucleus receives fibers from both cerebral cortexes.
--- Lower part of the motor nucleus receives fiber from the contralateral cerebral cortex.

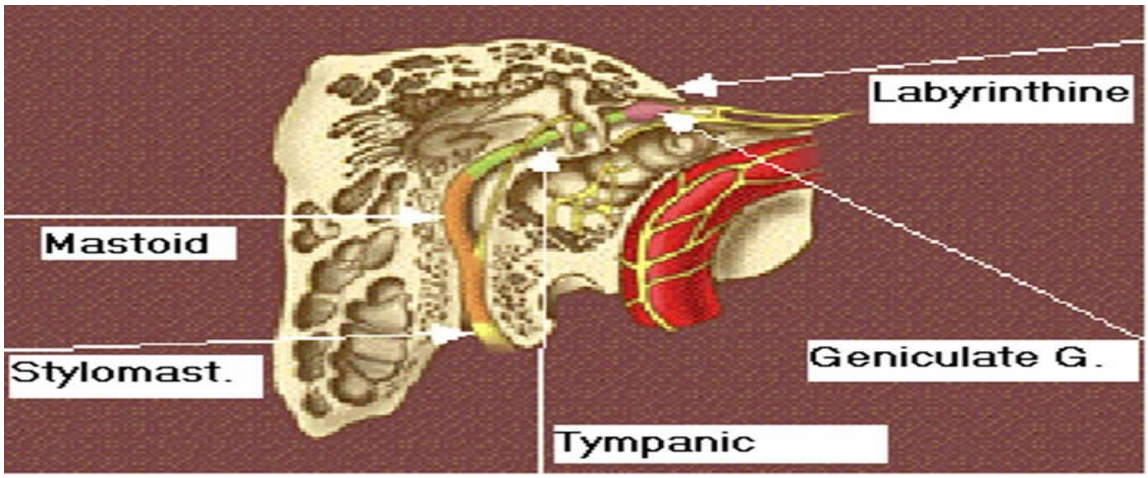
- **Upper motor lesions** spare the upper facial muscles (frontalis M. and Orbicularis Occuli M.) and affect the contralateral lower face.
- **Lower motor lesions** affect all the ipsilateral (Whole half) facial muscles.

UPPER MOTOR

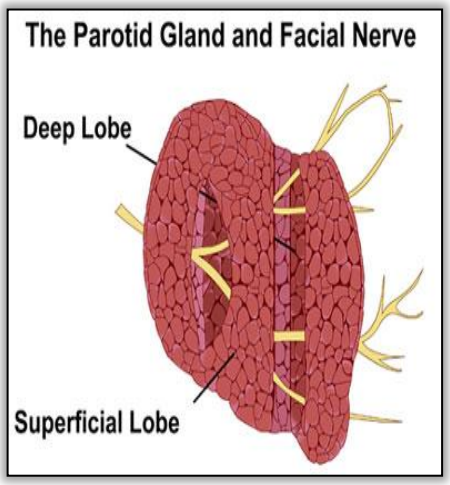


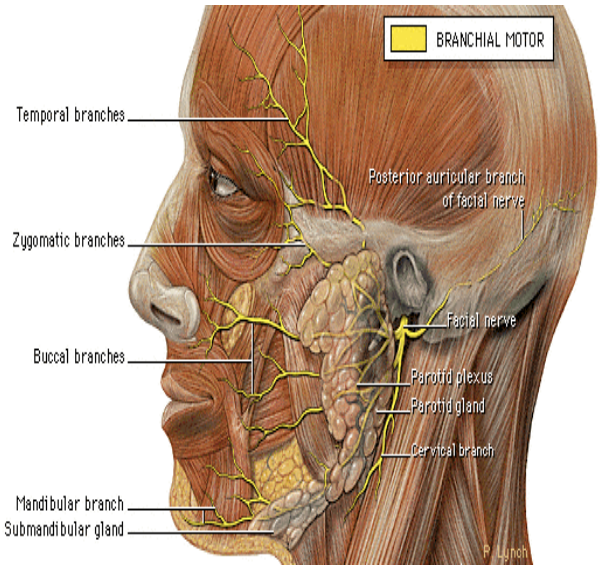
LOWER MOTOR

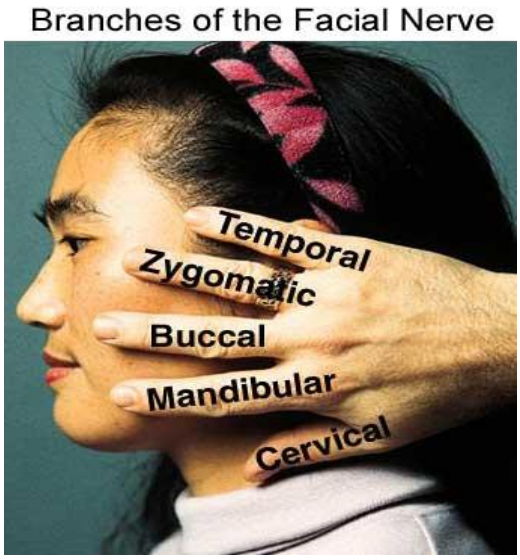
2. Intratemporal part:
From the Internal Auditory Meatus (association with **vestibulocochlear nerve**). Then goes to Fallopian canal.
Fallopian Canal has a horizontal part and a vertical part. AND its divided to:
- **Labyrinthine:** Related to the inner ear.
- **Tympanic:** Related to the middle ear.
- **Vertical part (mastoid):** Related to the external ear (Branches: 1-Supplies Stapedius Muscle 2-Chorda Tympani).



3. Extracranial part:
After it leaves the temporal bone through the **Stylomastiod foramen**, it goes through the parotid gland.
* In the parotid gland it divides into its **FIVE** branches which are:
- **Temporal:** Supplies frontalis
- **Zygomatic:** orbicularis oculi (most important branch) responsible for closure of the eye.
- **Buccal:** buccinator
- **Mandibular:** orbicularis oris
- **Cervical:** Platysma







(Course of Secreto-motor [parasympathetic] + taste)

Parasympathetic fibers: (Superior Salivary Nucleus) Some fibers go with motor fibers and some fibers leave the facial nerve at the Geniculate ganglion and goes to the lacrimal gland and some other glands.

The other secreto-motor fibers leave the vertical part of the facial nerve through the Chorda Tympani, which crosses the middle ear.

The Chorda Tympani then joins the **Lingual** nerve and innervates the two salivary glands: the submandibular gland and sublingual gland.

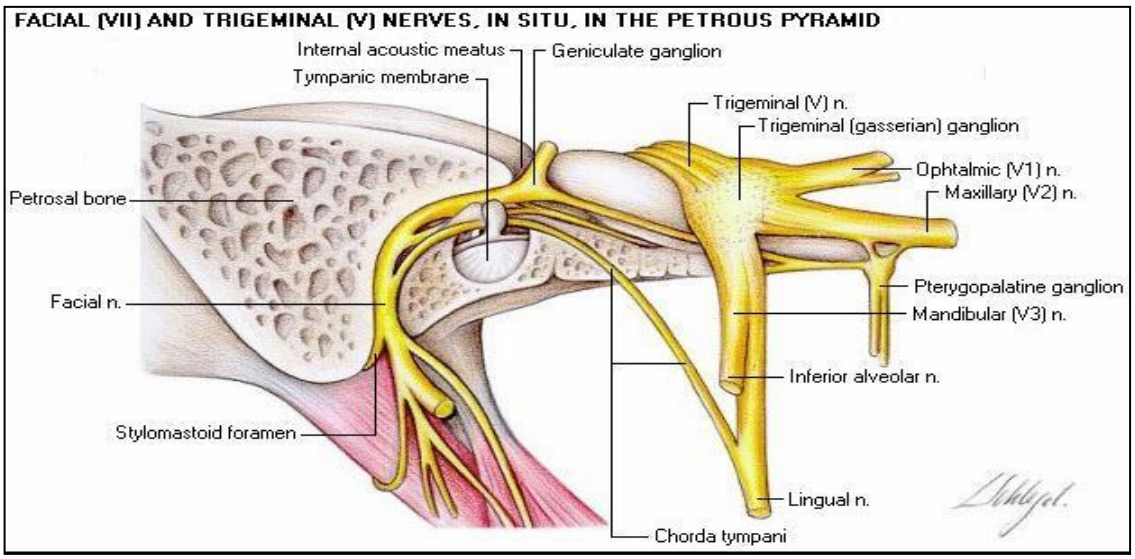
Taste: takes the same course of the Parasympathetic but in the opposite way (from peripheral to central).

* Fibers from the palate goes to the Geniculate ganglion> nucleus solitarii.
* Fibers from the anterior 2/3 of the tongue> chorda tympani> nucleus solitarii.

NOTE: This is important to know the level of the lesion of the facial nerve.

For Example:

- If the lesion is above the level of the chorda tympani> patient will loose the function of chorda Tympani he will have dryness and loose of taste sensation.
- If the lesion is below the level of chorda tympani> patient will have only facial paralysis with normal taste sensation.



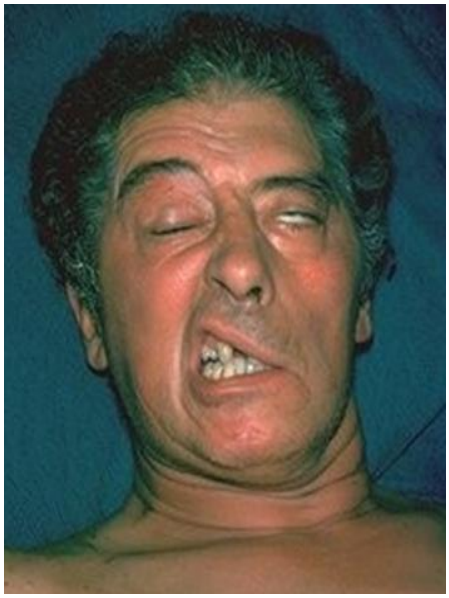
VARIATIONS AND ANOMALIES :

Only one normal variation the doctor talked about, which is:

- At the junction of the Tympanic vertical part there is: (dehiscent) no bony coverage of the nerve (no protection). It's a common anomaly.

This subjects the facial nerve to infection and trauma in the middle ear.

CLINICAL MANIFESTATIONS



- **Paralysis of facial muscles**
  - Asymmetry of the face
  - Inability to close the eye
  - Accumulation of food in the cheek
- **Phonophobia>** Stapedius muscle (LMNL).
- **Dryness of the eyes>** impaired drainage.
- **Loss of taste >** anterior 2/3 of the tongue.

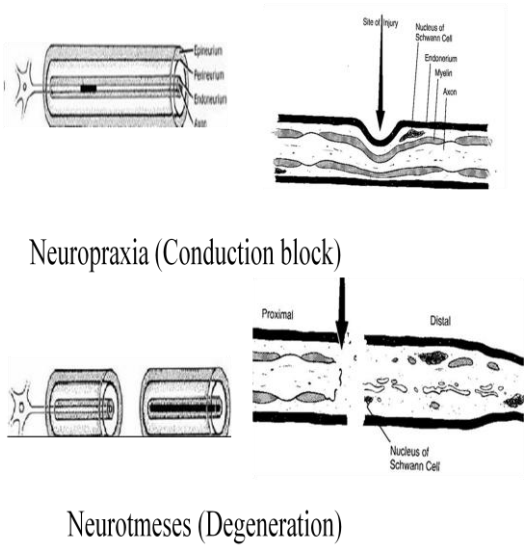
Pathologies of facial nerve injury:

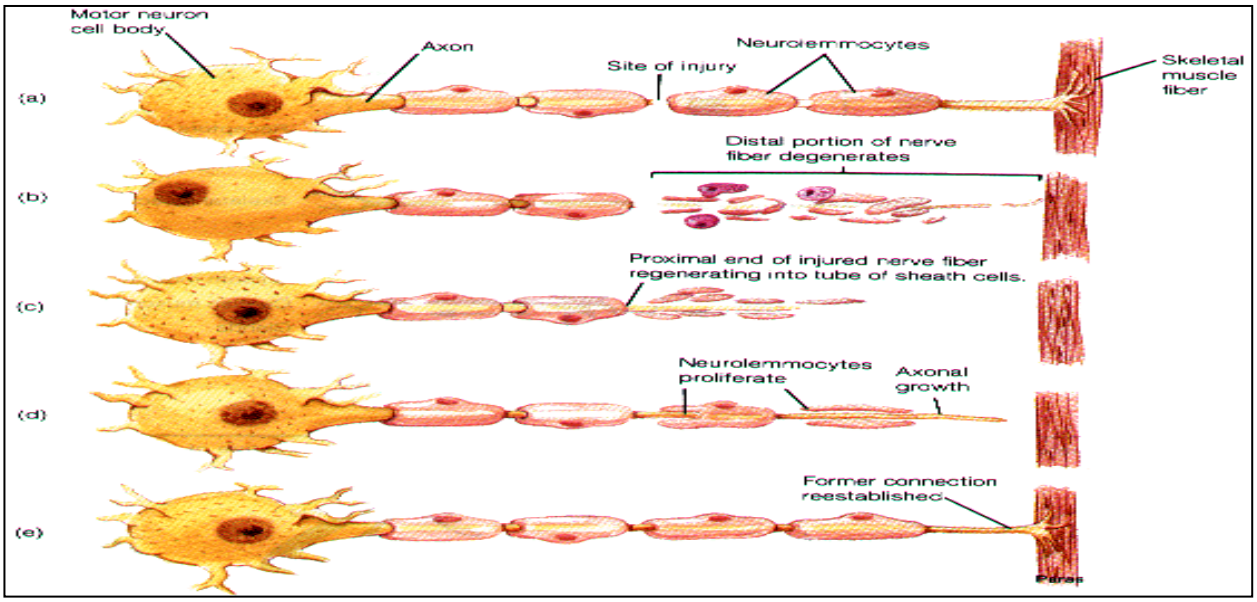
Facial nerve paralysis could be:

1- Conduction block (Neuropraxia) : due to mild trauma, nerve is affected functionally only and there is no degeneration anatomically. this one you expect complete recovery if the cause was removed.

2- Degenerative (Neurotomesis) : you have to bring the nerve together (bring distal part to proximal part), recovery is much slower. The rate of regeneration is 1mm/per day. (Distal part will have degeneration)

3- Mixed





Principles of management of facial nerve paralysis:

• Care of the eye.
• Treatment of the cause if applicable.
• Treatment of the nerve varies according if the paralysis is partial or complete.

A) Partial facial paralysis:

Being partial means that the nerve fibers are in continuity and recovery is expected by **conservative** treatment (e.g. removal of pressure, steroid etc) Remove the cause only, NO SURGERY.

B) Complete facial paralysis:

- If it is due to **neuropraxia**, recovery is expected by conservative treatment.
- If it is due to **degeneration (Neurotmeses)** , surgical treatment is required.
- To differentiate between degeneration and neuropraxia **electrophysiological tests** are required

Electrophysiological Tests:

- **Nerve Excitability Test (NET)**
- **Electroneurography (ENoG)**

- It detect degeneration of the nerve fibres .
- Useful only 48-72 hours following the onset of the paralysis.

Nerve excitability test (NET): 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.

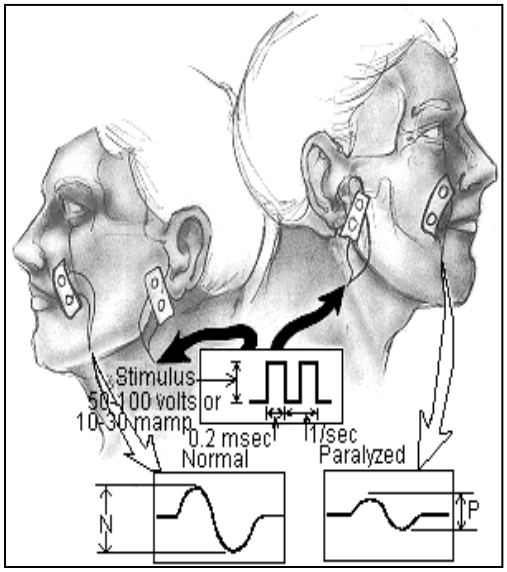
- **Advantage:** Simple, available everywhere
- **Disadvantage:** Subjective only (rough estimation either you see it move or not)

Electroneurography (ENoG):

***Compares compound action potential of both sides**
-Stimulate nerve at stylomatoid foramen.
-Measure muscular response near nasolabial groove

*Less intact motor axons with Wallerian degeneration.
*Worse prognosis with rapid degeneration.
*Inaccurate within first 3 days of Bell's palsy onset.
*Quantitative analysis, observer independent

Advantage: More accurate measurements, Objective
Disadvantage: Not available as common as NET



Interpretation of the tests:

*Not useful in the first 48 – 72 hours
*After 48-72 hours (the time required for degeneration to take place)
-**Normal results** means that there is no degeneration **(Neuropraxia)**
-**Abnormal** results means **degeneration** .

*So, If the patient is presenting already with partial paralysis this test will be waste of time because you already know that there is some fibers acting and some are not!

For partial you do:

Topognostic tests : Indicated in some cases to locate the site of injury:

- **Schirmer's test >** Test the lacrimation function
- **Stapedial reflex**
- **Taste sensation**

Causes of Facial nerve paralysis:

we can distribute it as Anatomical or Pathological

Anatomical:

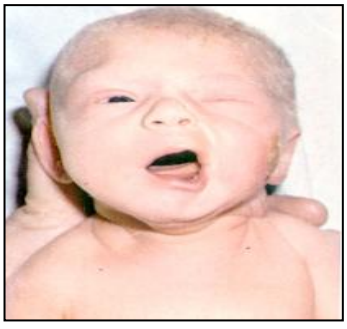
-Intracranial causes
-Cranial (intratemporal) causes
-Extracranial causes

Pathological:

- Congenital: Birth trauma
- Traumatic: Head and neck injuries & surgery
- Inflammatory: O.M, Necrotizing O.E., Herpes
- Neoplastic: Meningioma, malignancy ear or parotid
- Neurological: Guillain-Barre syndrome, multiple sclerosis
- Idiopathic: **Bell's palsy** (most common)

Congenital Facial Palsy:

- 80-90% are associated with birth trauma
- 10 -20 % are associated with developmental lesions
- at the level of Stylomastoid Foramen; Because it is not well developed yet and the nerve is superficial there.



Inflammatory causes of facial paralysis:

A- Facial Paralysis in AOM (Acute Otitis Media)

- Mostly due to pressure on a dehiscent nerve by inflammatory products
- Usually is **partial** and **sudden** in onset
- Treatment is by antibiotics (treat otitis media) and myringotomy (drainage/relieve pressure)

B- Facial Paralysis in CSOM (Chronic supportive Otitis Media)

- Usually is due to pressure by cholesteatoma or granulation tissue
- Insidious in onset
- May be partial or complete
- Treatment is by immediate surgical exploration and "proceed"

C- Herpes zoster oticus (RAMSAY HUNT SYNDROME)

- Herpes zoster affection of cranial nerves VII, VIII, and other nerves
- Facial palsy, pain, skin rash, SNHL and vertigo
- Vertigo improves due to compensation
- SNHL is usually irreversible
- Facial nerve recovers in about 60% (Means that 60% of patients is having Neuropraxia and the other 40% is having Neurotomesis)
- Treatment by: Acyclovir, steroid and symptomatic

Traumatic facial injury:

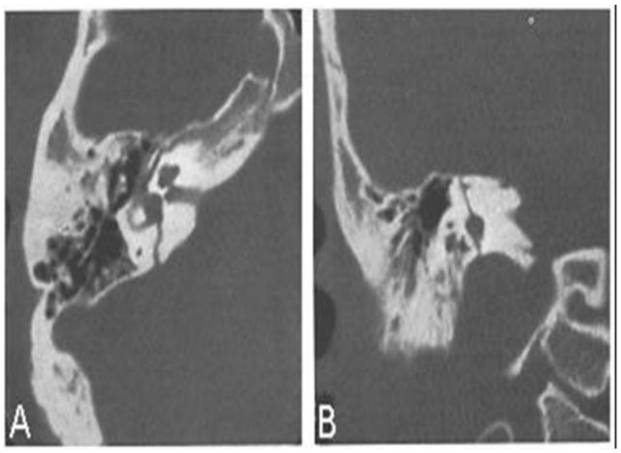
A-Iatrogenic (surgical) :
Operations at the CP angle, ear and the parotid Glands.

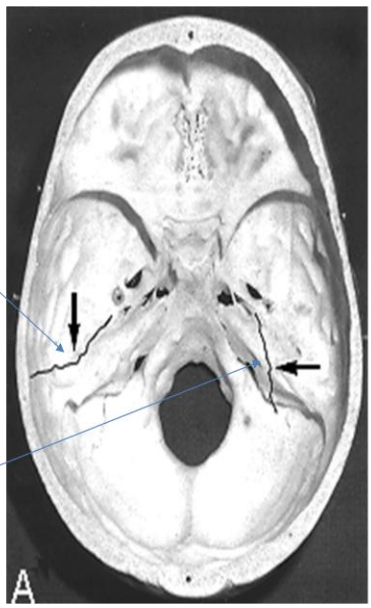
B-Temporal bone fracture:

1-Longitudinal: More common, Less severe, might cause conductive hearing loss, usually does not affect facial nerve.
2-Transverse: Less common, More severe, More likely to affect facial nerve.

*Pathology:
(Why the Facial Nerve get effected)?
Edema: will lead to partial paralysis
Transaction of the nerve: will lead to complete paralysis.

Transverse Fracture





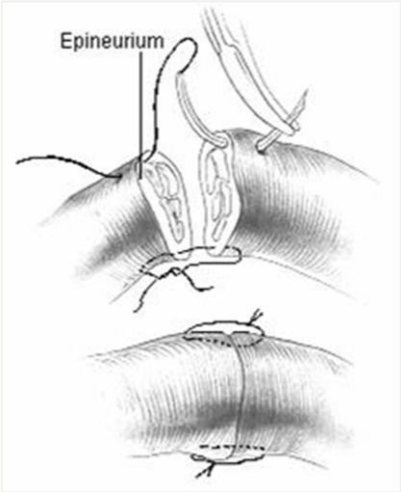
Management of Traumatic Facial Nerve Injury:

- If it is *delayed* in onset, it is usually <u>incomplete</u> and is due to edema
  - **Conservative**
- If of *immediate* onset, it is usually <u>complete</u> and due to transection of the nerve
  - **Surgical repair**

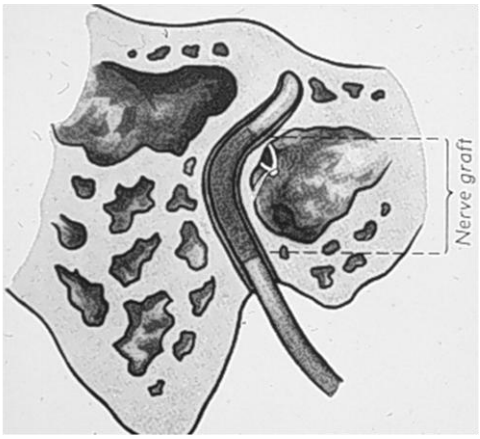
SURGICAL REPAIR:

1) **Direct Anastomosis** > No gap in nerve
2) **Nerve Graft** > Gap (From the great ocular Nerve)
3) **Nerve Transfer** > Distal part is there, but NO proximal (From the Hypoglossal Nerve; it is supplying the tong Motor but paralysis to ipsilateral to the tong is not going to effect it function that is why we use it)
4) **Muscle Flap** > NO distal and NO proximal (either Buccinators or temporalis)

1) Direct Anastomosis



2) Nerve Graft



3) Nerve transfer (anastomosis)

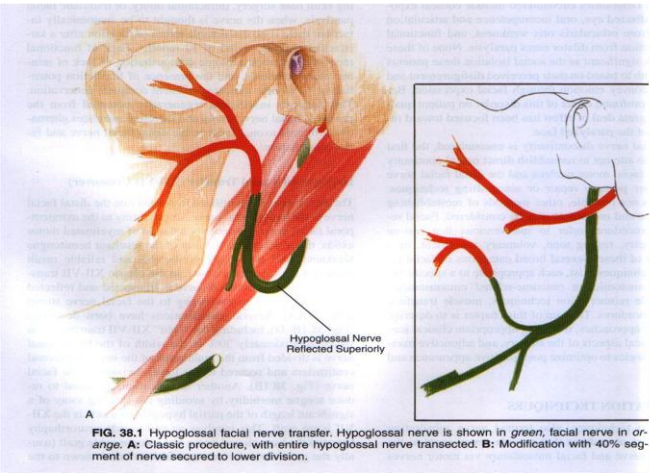
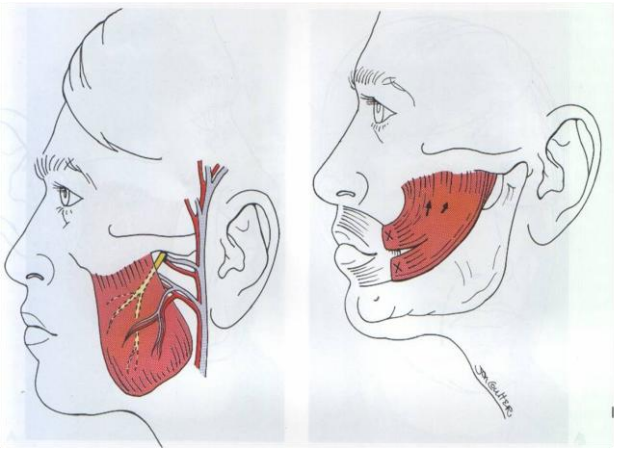


FIG. 38.1 Hypoglossal facial nerve transfer. Hypoglossal nerve is shown in *green*, facial nerve in *orange*. **A:** Classic procedure, with entire hypoglossal nerve transected. **B:** Modification with 40% segment of nerve secured to lower division.

4) Muscle flap



BELL'S PALSY

• Most common diagnosis of acute facial paralysis
• Diagnosis is **by exclusion** (exclude all other causes of face paralysis)

***Pathology:** Edema of the facial nerve sheath along its entire intratemporal course (Fallopian canal)

***Etiology:** Two theories: Viral vs Vascular

***Clinical features:** (Don't diagnose it at least it's typical)
• Sudden onset unilateral FP
• Partial or complete
• No other manifestations apart from occasional mild pain
• May recur in 6 – 12%

***Prognosis:**
• 80% complete recovery
• 70%satisfactory recovery
• 10% no recovery

***Treatment:**
• Reassurance
• Eye protection
• Physiotherapy
• Medications (steroids, antivirals,vasodilators)
• Surgical decompression in selected cases.

***Surgical Management:**
• Debate over years
• Patients with 90% degeneration
• Within 14 days of onset