



433 Teams
ENT

Facial Nerve

Color index:

432 Team – **Important** – 433 Notes – Not important

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Objectives:

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

Facial Nerve Fibers

- 1. Motor fibers:**
To the stapedius and facial muscles.
- 2. Secreto-motor fibers (parasympathetic):**
To the lacrimal gland and the submandibular and sublingual salivary glands.
- 3. Taste fibers:**
From the anterior two third of the tongue and palate.
- 4. Sensory fibers:**
From the external auditory meatus "very few".

*10,000 neurons.
7,000 are myelinated (70% of facial nerve is motor).

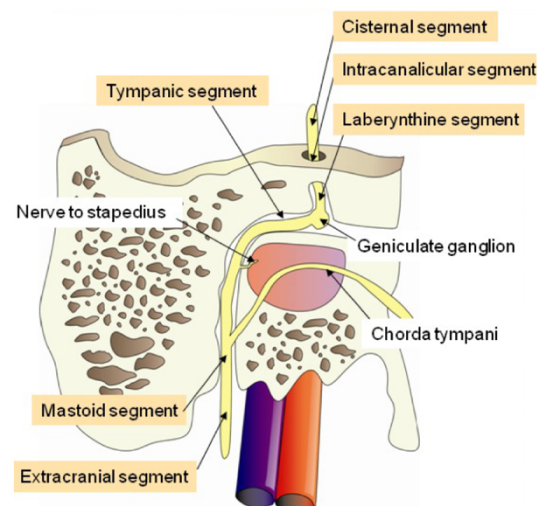
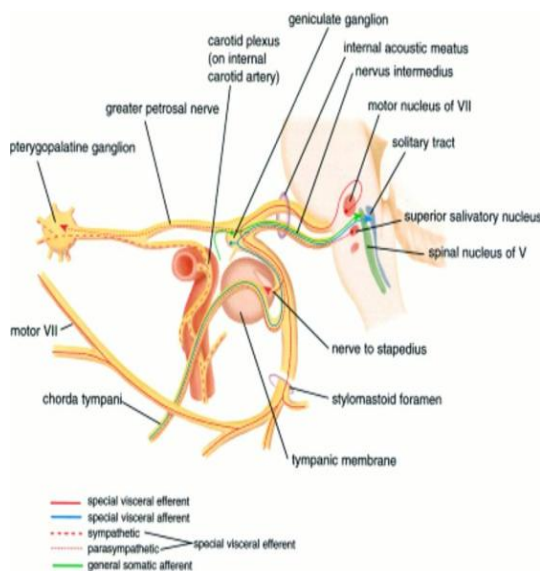
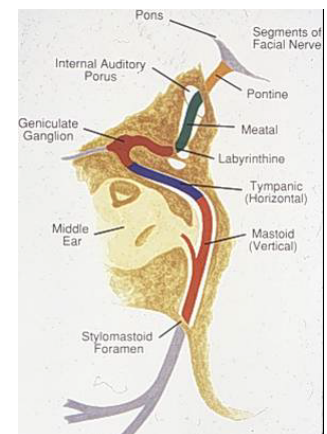
*Parotid gland supplied by the 9th CN.

Anatomical Divisions

The course of motor fibers:

Facial Nerve Segments:

- 1. Intracranial (cisternal) segment.**
Meatal segment (internal auditory canal): zero branches.
- 2. Crania (intratemporal):**
 - Labyrinthine segment: in the inner ear, **shortest segment**.
 - Tympanic segment: in the middle ear, zero branches.
 - Mastoid segment: in the external ear, 3 branches.
- 3. Extracranial (extratemporal): 9 branches.**

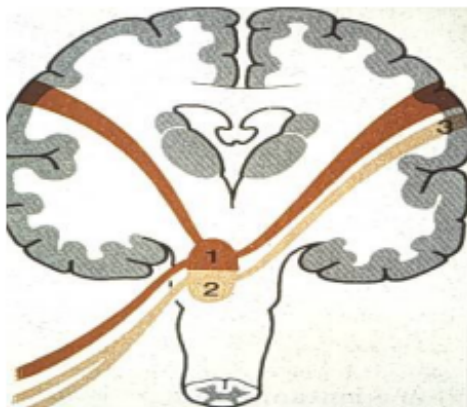
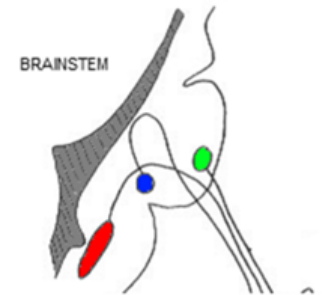


1. The intracranial part:

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

Facial Nerve Nuclei (in the pons):

- **Nucleus Solitarius:** receives taste fibers.
- **Facial nucleus:** main nucleus which gives motor fibers.
- **Superior salivatory nucleus:** gives parasympathetic fibers.



The facial nucleus is divided into 2 parts:

1. The upper half that receives innervation from both cerebral cortices.
2. The lower half that receives innervation only from the contralateral cerebral cortex.

Lower motor lesions affect **all the ipsilateral** facial muscles “Lower motor neuron lesion is from the nucleus downward”.

Upper motor lesions spare the upper facial muscles and affect **the contralateral lower face** “The upper half of the face still receives innervation from the other side, so this will cause lower face paralysis of the other side”.

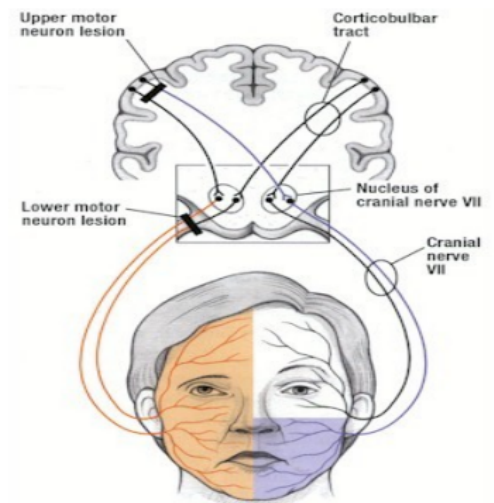


Figure 2a: The color lines show the distribution of facial muscles paralyzed after a supranuclear lesion of the corticobulbar tract and after a lower motor neuron lesion of the facial nerve.



Lower Motor

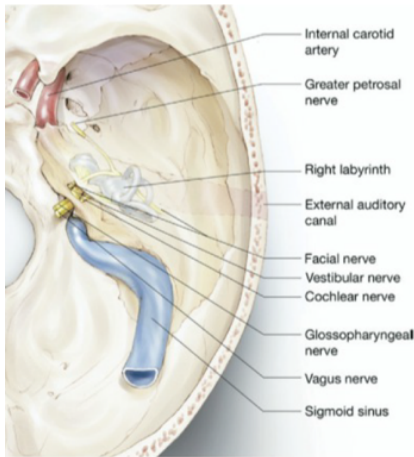
Whole left side of the face is affected



Upper Motor

Only the **lower part** of the left side is affected

- *UMN spare the upper face because the forehead is innervated bilaterally.
- *Acoustic neuroma can cause facial nerve paralysis.



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 vestibulo-cochlear nerve (8th).

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

2. The Cranial Part (intratemporal):

*What segment is most likely affected?
Labyrinthine segment.

*First branch is from the geniculate ganglion (greater superficial petrosal nerve).

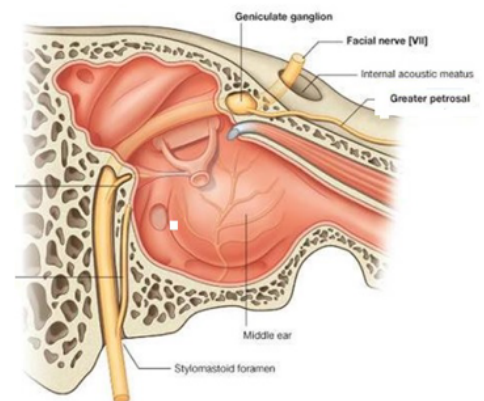
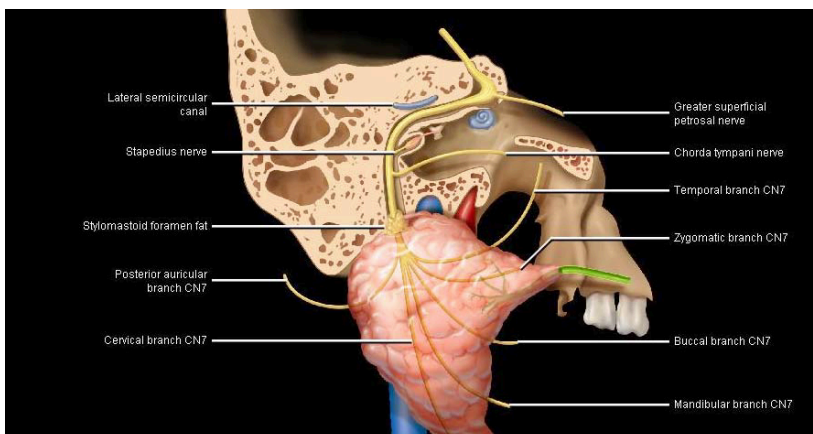
*Nerve to stapedius and chorda tympani are branches from mastoid segment.

From the internal auditory meatus, 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, high risk of ischemia (**possible MCQ**).
2. **Tympanic** (from geniculate ganglion to pyramidal eminence) “in the middle ear”
3. **Mastoid or vertical** (from pyramidal eminence to stylomastoid foramen) “in the external ear”

It finally leaves the skull through stylomastoid foramen.



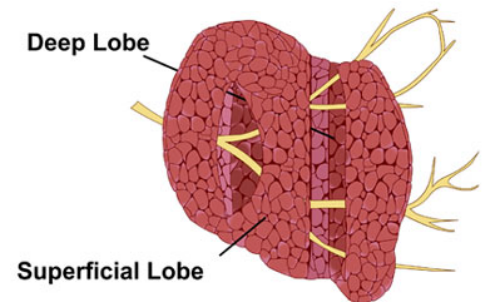
3. The Extracranial Part (extratemporal):

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.

Then, it branches within the anterior border of the parotid into five terminal branches:

The Parotid Gland and Facial Nerve



*Parotid surgeries can cause facial nerve paralysis.

1. **Temporal:** most superior > supplies the frontalis muscle.
2. **Zygomatic:** supplies orbicularis oculi muscle.
3. **Buccal:** supplies buccinators muscle.
4. **Mandibular:** supplies the muscles of the angle of the mouth.
5. **Cervical:** “long but thin branch”: supplies platysma muscle.

Most important 2 branches:

Zygomatic “to protect the eye”

Mandibular “its paralysis causes cosmetically bad deformity”.

Branches of the Facial Nerve



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

Least important branch is **cervical**.

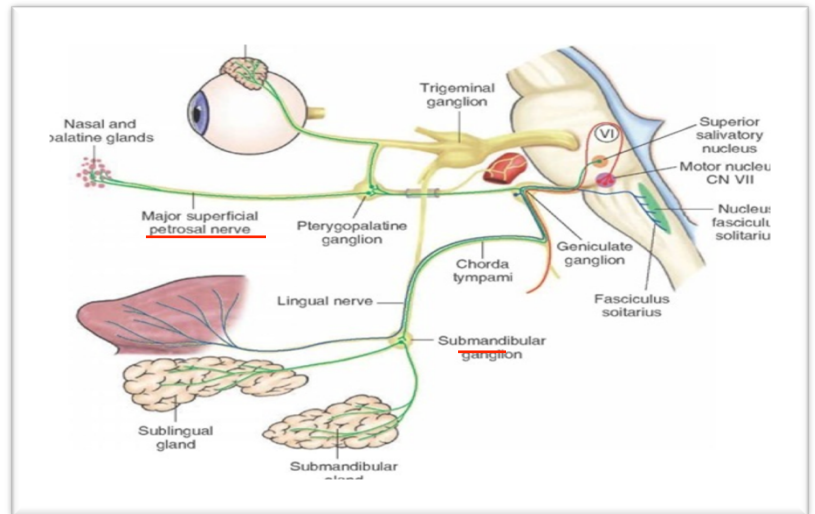
There are usually some variations in different branches. Some branches may get divided into two and each branch divides into another two etc.

The course of secreto-motor and taste fibers:

The **secreto-motor fibers** leave the superior salivatory 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**.

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 2/3rd of the tongue go through the chorda tympani to the facial nerve and finally to nucleus solitarii.

What happens if there is an injury of the chorda tympani?

It easily gets injured because it passes in the inner ear

1. Minor defect in the taste “because it affects the anterior 2/3 of only one side of the tongue”
2. There will be no dryness “because the parotid is supplied by the glossopharyngeal nerve (9th)”

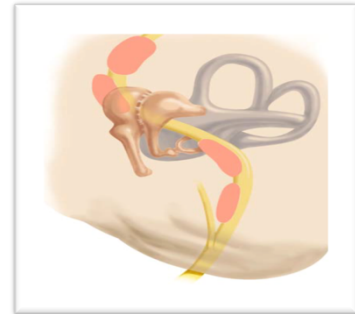
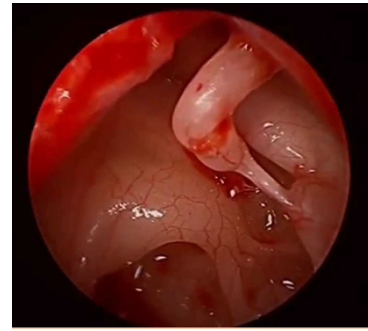
Variations and Anomalies

Dehiscence: a defect in the fallopian canal.

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). (possible **MCQ**)



Facial Nerve Paralysis

Clinical manifestations:

Paralysis of facial muscles:

- Asymmetry of the face.
- Inability to close the eye.
- Accumulation of food in the cheeks. “Paralysis of buccinators”

Lower motor neuron lesion of the left side:

- 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

The orbicularis oculi and frontalis muscles will not be affected.



Other manifestations of facial nerve paralysis:

- Phonophobia “due to failure of contractions of trapedius muscle, uncomfortable feeling in exposure to loud sounds”
- 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 ganglia
 - *So if the paralysis is above the level of geniculate ganglia > dryness
 - *If below it > no dryness
- Loss of taste “very little”

*Acoustic reflex (stapedial reflex). If there is a facial nerve lesion in the parotid, the stapedius will not be affected, but if the lesion is before the branch to stapedius (mastoid segment of the facial nerve) it will be affected.

*Dryness of the eye can cause exposure keratitis. The eye must be protected.

*Loss of taste (they feel a metallic taste).

Clinical examination: ask the patient to:

1. Look up to test frontalis.
2. Close eyes to test orbicularis oculi.
3. Blow the cheek to test buccinators.
4. Show the teeth for angulation.

Bilateral:

- Guillain-Barre syndrome
- Lyme disease
- Intracranial neoplasm

Recurrent:

Melkersson-Rosenthal syndrome



House-Brackmann Scale

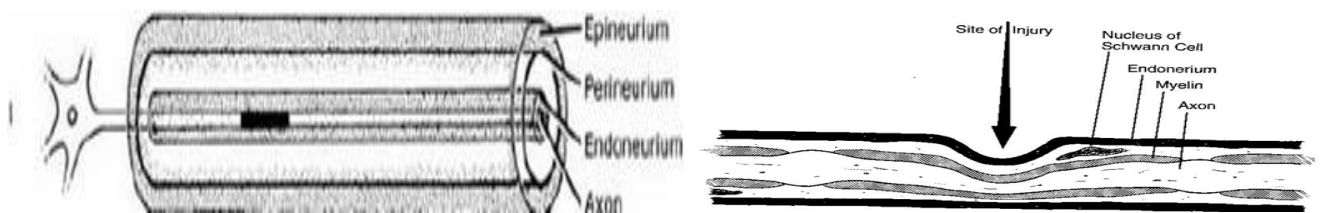
Grade	Appearance	Forehead	Eye	Mouth
I	normal	normal	normal	normal
II	slight weakness normal resting tone	moderate to good movement	complete closure minimal effort	slight asymmetry
III	non-disfiguring weakness normal resting tone	slight to moderate movement	complete closure maximal effort	slight weakness 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

Pathophysiology of Nerve Injury

Neuropraxia (conductive block):

In cases of mild trauma causing only functional block of the facial nerve, the fibers still keep their integrity.

In Regeneration: there will be restoration of full function if the cause is treated.



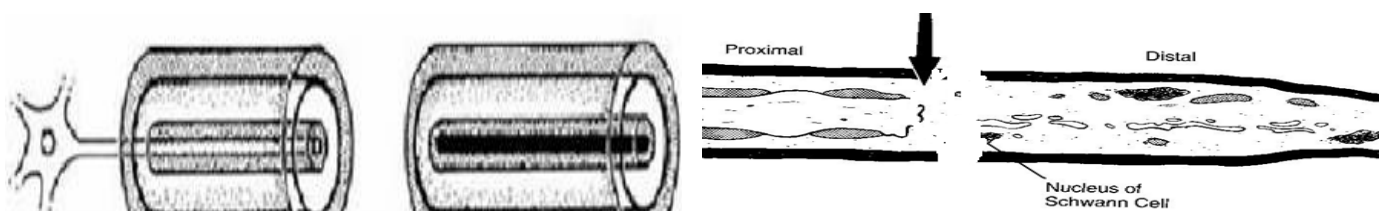
Neurotmesis (degeneration):

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.

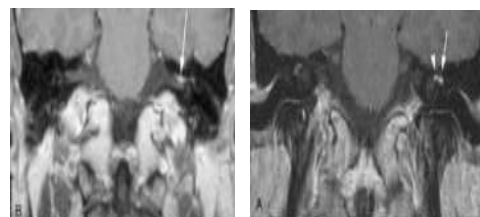
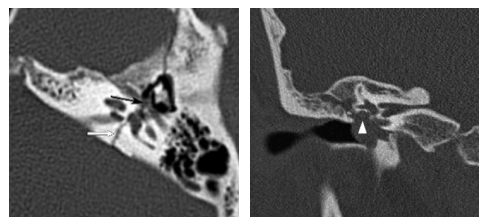
To differentiate between degeneration and neuropraxia electrophysiological tests are required.

Diagnostics

Radiology:

- Localize lesion
- Computed tomography
 - Trauma
 - Mastoiditis
 - Cholesteatoma
- Magnetic resonance imaging (MRI)
 - Nerve enhancement
 - Exclude neoplasm

Usually MRI enhancement in labyrinthine segment.



Topognostic tests:

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

- Schirmer's test: test lacrimation function. > greater superficial petrosal
- Stapedial reflex > stapedial branch
- Taste sensation (Electrogustometry) > chorda tympani
- Salivary flow > chorda tympani

*If lacrimation is involved then most likely the lesion is 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.
 - Absent **ipsilateral** acoustic reflex.
- Retrocochlear pathology
 - Asymmetrical thresholds.

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

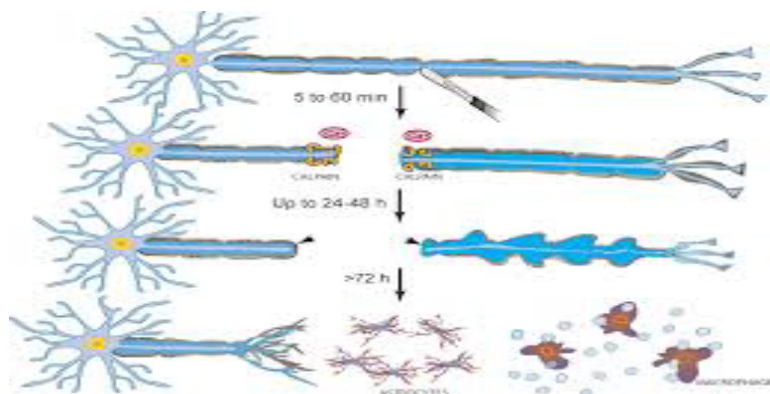
Electrophysiology:

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: **Principle: stimulate the nerve and look for response:**

- Nerve Excitability Test (NET)
- Electroneurography (ENoG)
- Electromyography (EMG)
- Maximum stimulation test (MST)

<p>Nerve Excitability Test (NET): 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.</p>	<p>Electroneurography (ENoG): 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.</p> <p style="text-align: center;">More objective</p>
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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.**

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 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

Most of them are partial and need only conservative treatment.



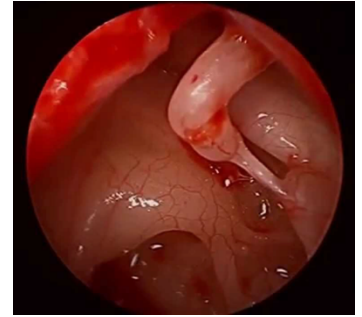
Left side

Inflammatory Causes of Facial Paralysis

Facial paralysis in Acute Otitis Media (AOM):

- 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.



Facial paralysis in Chronic Suppurative Otitis Media (CSOM):

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



Herpes Zoster Oticus (**Ramsay Hunt Syndrome**):

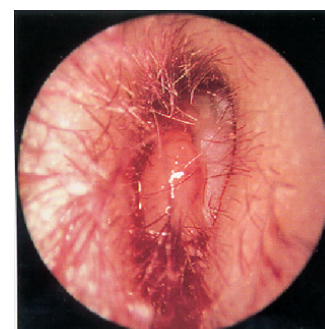
- Herpes zoster affection of cranial nerves VII, VIII, and cervical nerves.
- Symptoms: Facial palsy, severe pain, **skin rash**, SNHL and vertigo.
- **Vertigo improves** due to compensation from the other side “takes few weeks”.
- **SNHL is usually irreversible**.
- **Facial nerve recovers in about 60%**.
- Treatment by: Acyclovir, steroid and symptomatic.



Vesicles

Malignant Otitis Externa:

- Osteomyelitis of the temporal bone (osteomyelitis of the external auditory canal bone).
- It could affect multiple nerves other than 7th if it reaches the jugular foramen (9th 10th 11th)
- In immunocompromised.
- 4Ds:
 - Diabetes mellitus
 - Discharge (purulent)
 - Discomfort
 - Dysfunction cranial nerve



Granulation obscured TM

Traumatic Facial Injury

Iatrogenic:

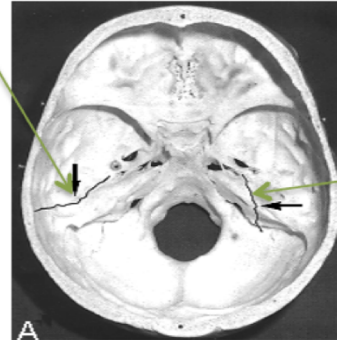
Operations at the CP angle, ear and the parotid glands.



Temporal bone fracture (possible MCQ):

- Longitudinal:
 - 80% of Temporal Bone Fractures.
 - 15-20% Facial Nerve involvement.
- Transverse:
 - 20% of Temporal Bone Fractures.
 - 50% Facial Nerve Involvement (more likely to cause paralysis).

Longitudinal



Transverse

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.

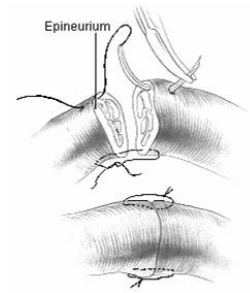
Management of traumatic facial nerve injury:

- 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 have to stitch it.



3. Nerve Transfer (anastomosis)

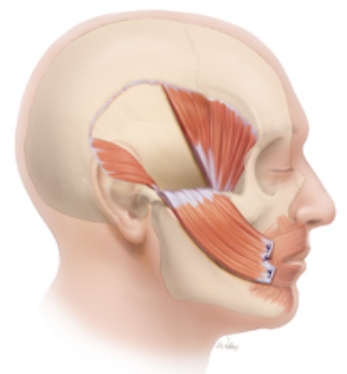
If the proximal part can't be identified, get a nerve and connect it to the distal part.

- Most common nerve used is hypoglossal nerve **IF** the other one is healthy and functioning well, because bilateral hypoglossal nerve damage is catastrophic.



4. Muscle Flap

If the distal and proximal parts can't be identified.
For cosmetics only "temporalis or masseter muscles are used".



Bell's Palsy

- Most common diagnosis of **acute** facial paralysis “if slowly progressive it is **NOT Bell's palsy**”.
- Diagnosis is by **exclusion**.

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 “**HSV**”.

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 “**No** discharge, **no** parotid swelling, **not** following trauma”.
- May recur in 6 – 12% “previous history of paralysis in the same side “12%” other side “6%”.

Family history and pregnancy. “risk factors”

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.

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.

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

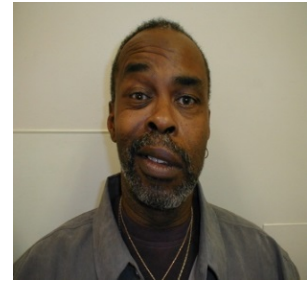
Questions from the doctor's slides:

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



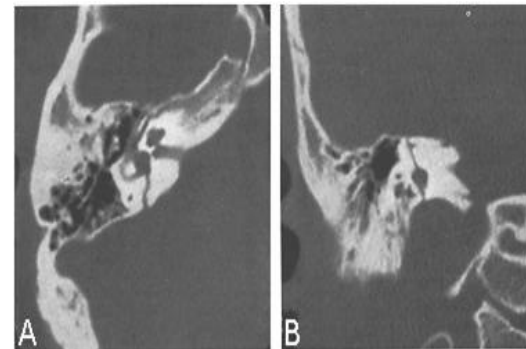
36 years old man with RTA:

What is your diagnosis?

Transverse fracture of the temporal bone.

Mention 2 clinical findings?

- Facial nerve paralysis
- CSF leak
- Ossicles injury



34 years old with LMN facial paralysis:

What is your diagnosis?

Herpes Zoster Oticus (Ramsay Hunt syndrome).

What is your management?

- Acyclovir
- Steroids
- Physiotherapy



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



Summary

Table 9. Differential Diagnosis of Peripheral Facial Paralysis (PFP)

Etiology	Incidence	Findings	Investigations	Treatment, Follow-up, and Prognosis (Px)
Bell's Palsy Idiopathic, (HSV) infection of the facial nerve Diagnosis of exclusion	80 to 90% of PFP Risk Factors: Diabetes Pregnancy Viral prodrome (50%)	Hx: Acute onset Numbness of ear Schirmer's test Recurrence (12%) + FHx (14%) Hyperacusis (30%) P/E: Paralysis or paresis of all muscle groups on one side of the face Absence of signs of CNS disease Absence of signs of ear or CPA diseases	Stapedial reflex absent Audiology normal (or baseline) EMG – best measure for prognosis Topographic testing MRI with gadolinium – enhancement of CN VII and VIII High resolution CT	Rx: Protect the eye to prevent exposure keratitis with patching or tarsorrhaphy Systemic steroids may lessen degeneration and hasten recovery Consider antiviral (acyclovir) F/U: Spontaneous remission should begin within 3 wk of onset Delayed (3 to 6 mo) recovery portends at least some functional loss Px: 90% recover spontaneously and completely overall; >90% recovery if paralysis was incomplete Poorer if hyperacusis, >60 yr, diabetes, HTN, severe pain
Ramsay-Hunt Syndrome (Herpes Zoster Oticus) Varicella zoster infection of CN VII/VIII	4.5 to 9% of PFP Risk Factors: >60 yr Impaired immunity Cancer Radiotherapy Chemotherapy	Hx: Hyperacusis SNHL Severe pain of pinna, mouth, or face P/E: Vesicles on pinna, ext. canal (erupt 3-7 d after onset of pain) Associated herpes zoster ophthalmicus (uveitis, keratoconjunctivitis, optic neuritis, or glaucoma)	Stapedial reflex absent Audiology – SNHL Viral ELISA studies to confirm MRI with gadolinium (86% of facial nerves enhance)	Rx: Pt. should avoid touching lesions to prevent spread of infection Systemic steroids can relieve pain, vertigo, avoid postherpetic neuralgia Acyclovir may lessen pain, aid healing of vesicles F/U: 2 to 4 wk Px: Poorer prognosis than Bell's palsy; 22% recover completely, 66% incomplete paralysis, 10% complete paralysis
TEMPORAL BONE FRACTURE				
Longitudinal (90%)	20% have PFP	Hx: Blow to side of head P/E: Trauma to side of head Neuro findings consistent with epidural/subdural bleed	Skull X-rays CT head	Px: Injury usually due to stretch or impingement; may recover with time
Transverse (10%)	40% have PFP	Hx: Blow to frontal or occipital area P/E: Trauma to front or back of head	Skull X-rays CT head	Px: Nerve transection more likely
Iatrogenic		Variable (depending on level of injury)	Wait for lidocaine to wear off EMG	Rx: Exploration if complete nerve paralysis No exploration if any movement present

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