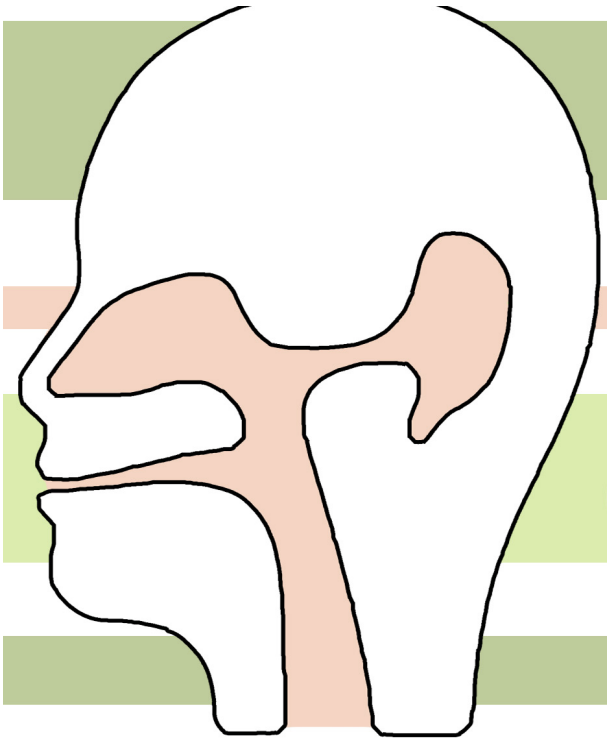


EAR, NOSE AND THROAT

(23) Facial Nerve

Leader: Maha Allhaidan

Done by: Reem Al-Hefdhi

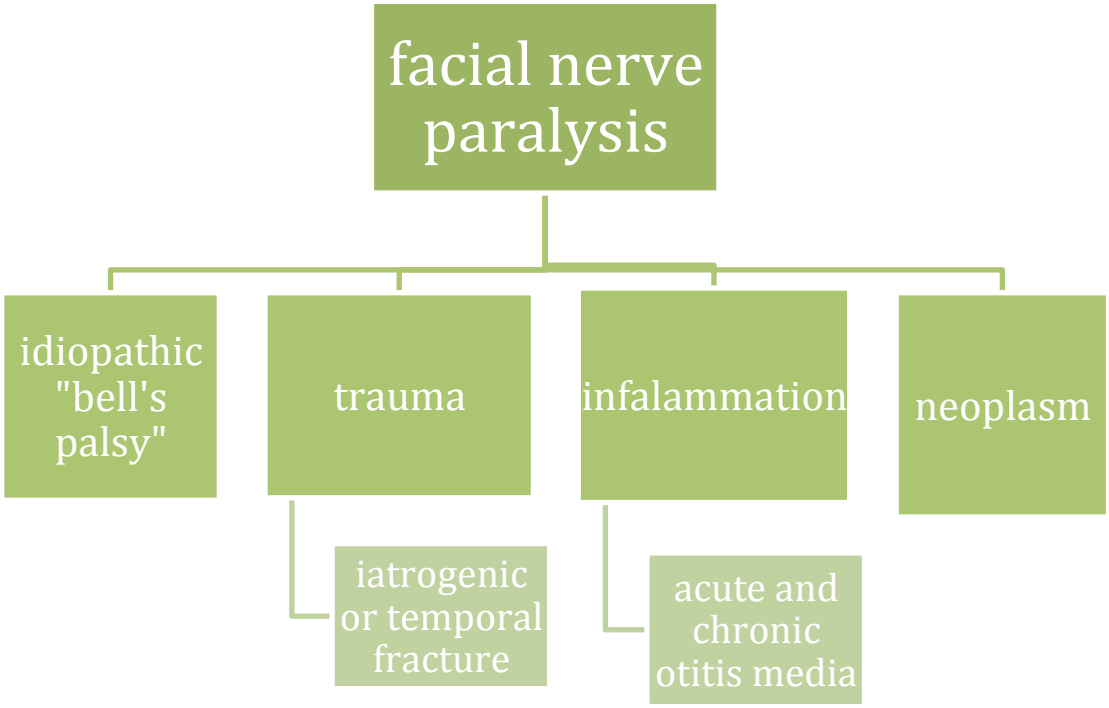
Revised by: Maha Allhaidan

Doctor's note Team's note Not important Important 431 teamwork

(431 teamwork do not highlight it in yellow, but put it in a yellow "box")

Objectives:

- Gross applied anatomy
- Pathology of nerve injury and regeneration
- Principles of the electrophysiological tests
- Causes of facial nerve paralysis
- Principles of treatment of facial paralysis
- Bell's palsy

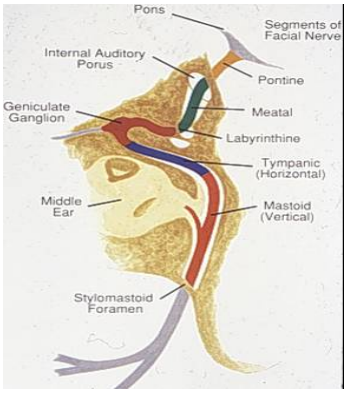


Facial Nerve Fibers

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"

Anatomical Divisions

The course of motor fibers



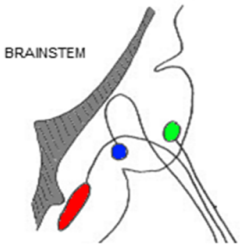
1. Intracranial
2. Cranial (intratemporal)
3. Extracranial (extratemporal)

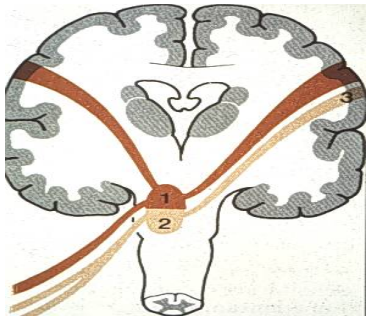
(1) The intracranial part

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

Facial Nerve Nuclei

- 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 form 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"

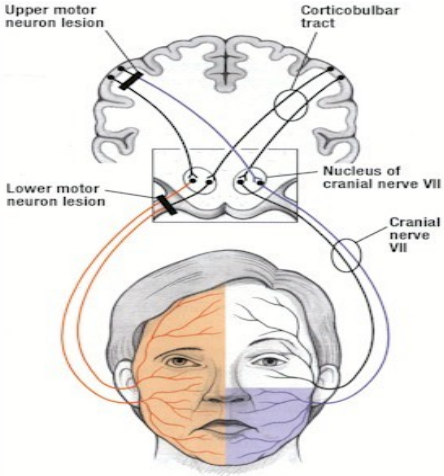


Figure 2a: The color lines show the distribution of facial muscles paralyzed after a supernuclear lesion of the corticobulbar tract and after a lower motor neuron lesion of the facial nerve.

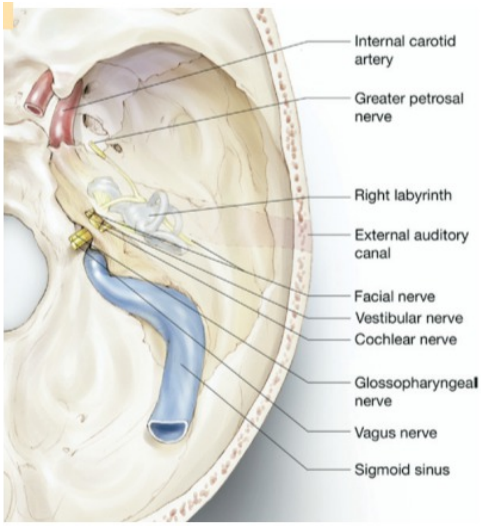
Upper motor
Only the **lower part** of the left side is affected

Lower motor
Whole left side of the face is affected

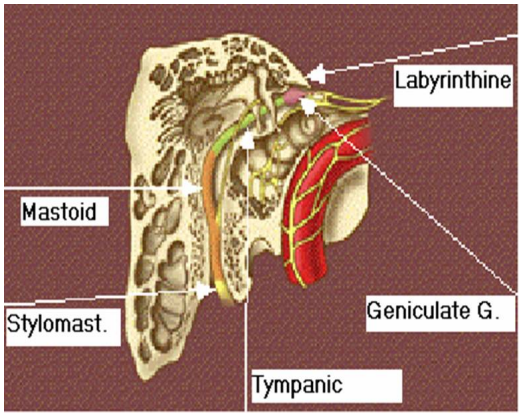
The CP angle segments:

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 with vestibulo-cochlear nerve.



(2) The intratemporal part (cranial):

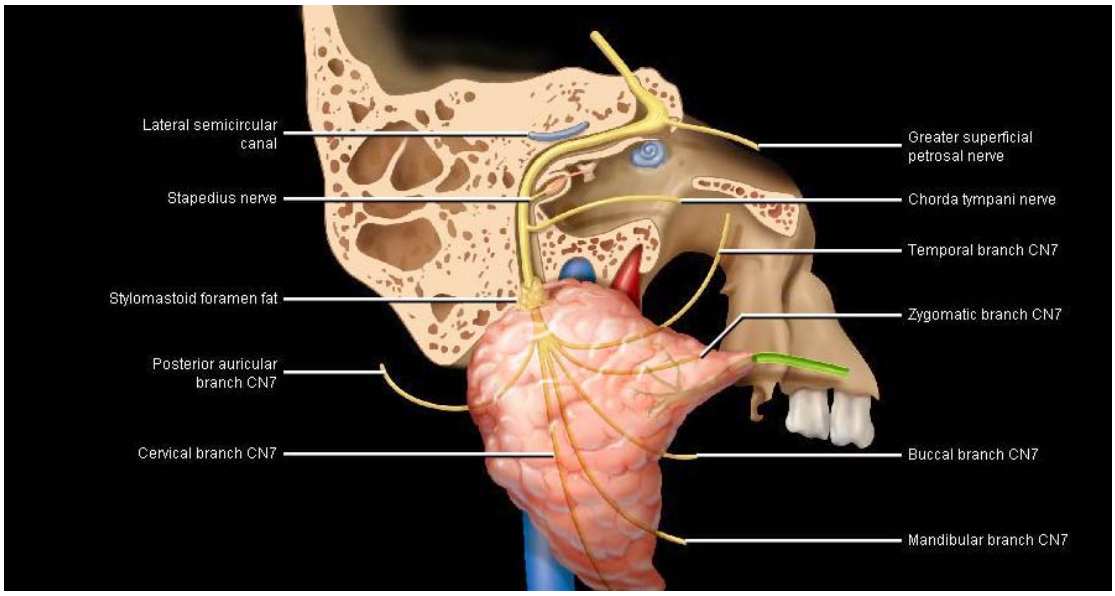


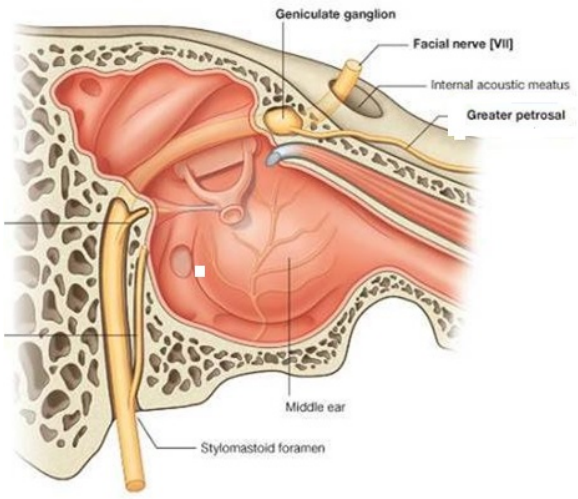
From the internal auditory meatus it crosses the temporal bone through fallopian canal and it is related directly to the inner, middle and external ear.

It is divided into 3 parts:

- Labyrinthine "in the inner ear"
- Tympanic "in the middle ear"
- Mastoid or vertical "in the external ear"

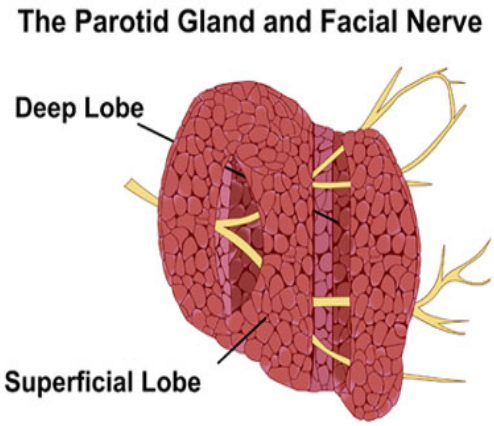
It finally leaves the skull through stylomastoid foramen.





(3) The extracranial part:

- 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.



Temporal: most superior → supplies the frontalis muscle.
Zygomatic → supplies orbicularis oculi muscle.
Buccal → supplies buccinator muscle.
Mandibular → supplies the muscles of the angle of the mouth.
Cervical "long but thin branch" → supplies platysma muscle.

Most important 2 branches:
Zygomatic "to protect the eye"
Mandibular "its paralysis causes cosmetically bad deformity"

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

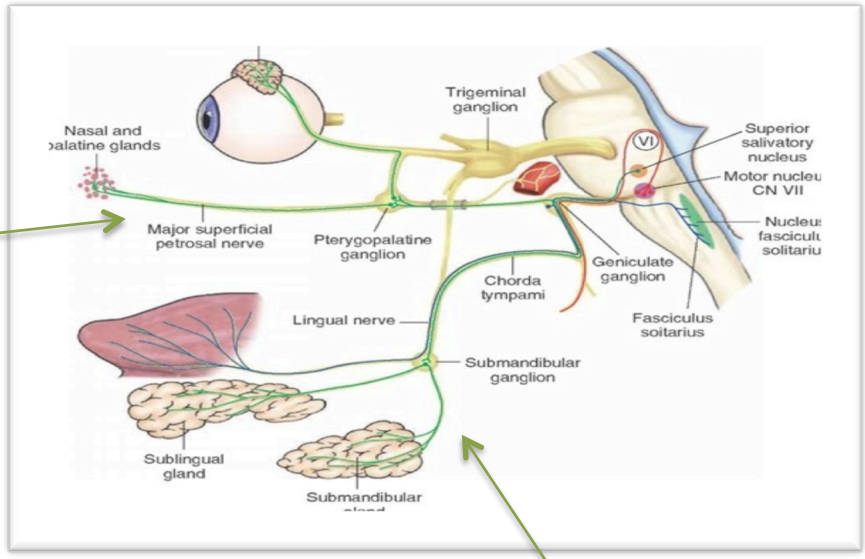
Least important branch is cervical.

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 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.



The other fibers leave the facial nerve in the chorda tympani and supply the submandibular and sublingual salivary glands

Taste fibers follow the same coarse 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 solitarius

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"

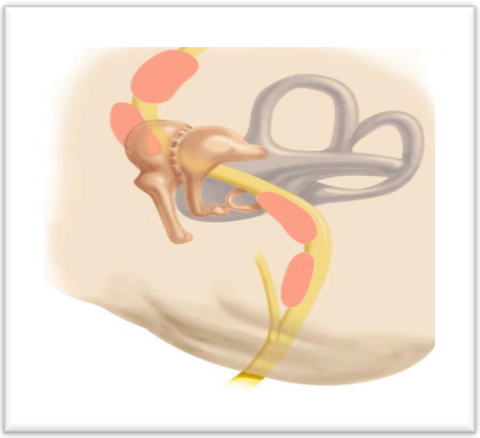
Variations and Anomalies

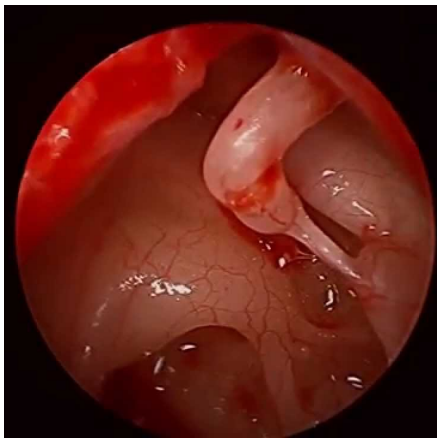
Dehiscence: a defect in the fallopian canal.

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

→ Becomes more subjected to trauma and infection leading to facial nerve paralysis.

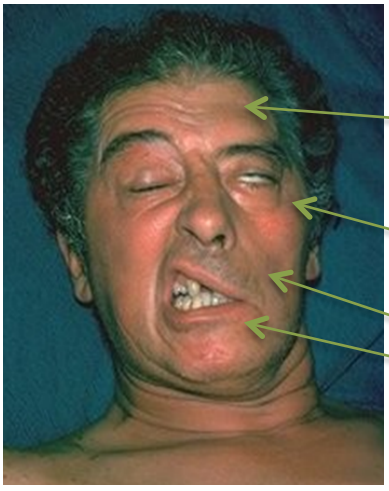




Facial Nerve Paralysis

Clinical manifestations:

- Paralysis of facial muscles.
  - Asymmetry of the face.
  - Inability to close the eye.
  - Accumulation of food in the cheeks. "Paralysis of buccinators"

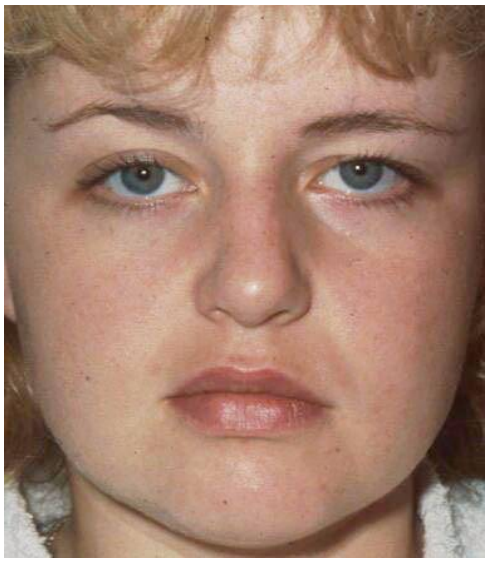


Lower motor neuron lesion of the left side

- 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
"The orbicularis oculi and frontalis muscles will not be affected"

Looks normal at rest



Clinical examination: ask the patient to
1. Look up to test frontalis
2. Close eyes to test orbicularis oculi
3. Blow the cheeck to test buccinator
4. Show the teeth for angulation

Other manifestations of facial nerve paralysis:

- Phonophobia "due to failure of contractions of strapedius 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

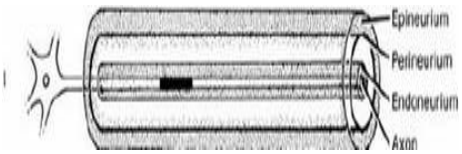
- Loss of taste "very little"

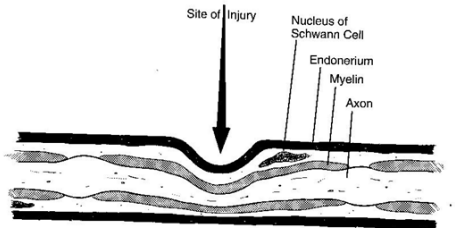
Pathophysiology of Nerve Injury

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.





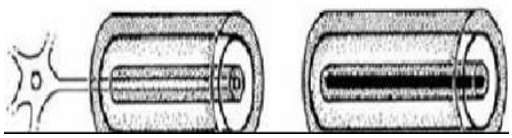
Neurotmesis (degeneration)

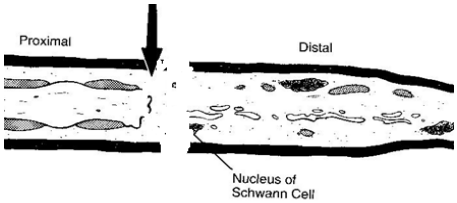
Anatomical block due to complete transection of the facial nerve. Wallerian degeneration of the distal part of the nerve, and this takes 2-3 days to occur.

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 1 mm/day or 1 inch/month → it will be delayed.
E.g. 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.





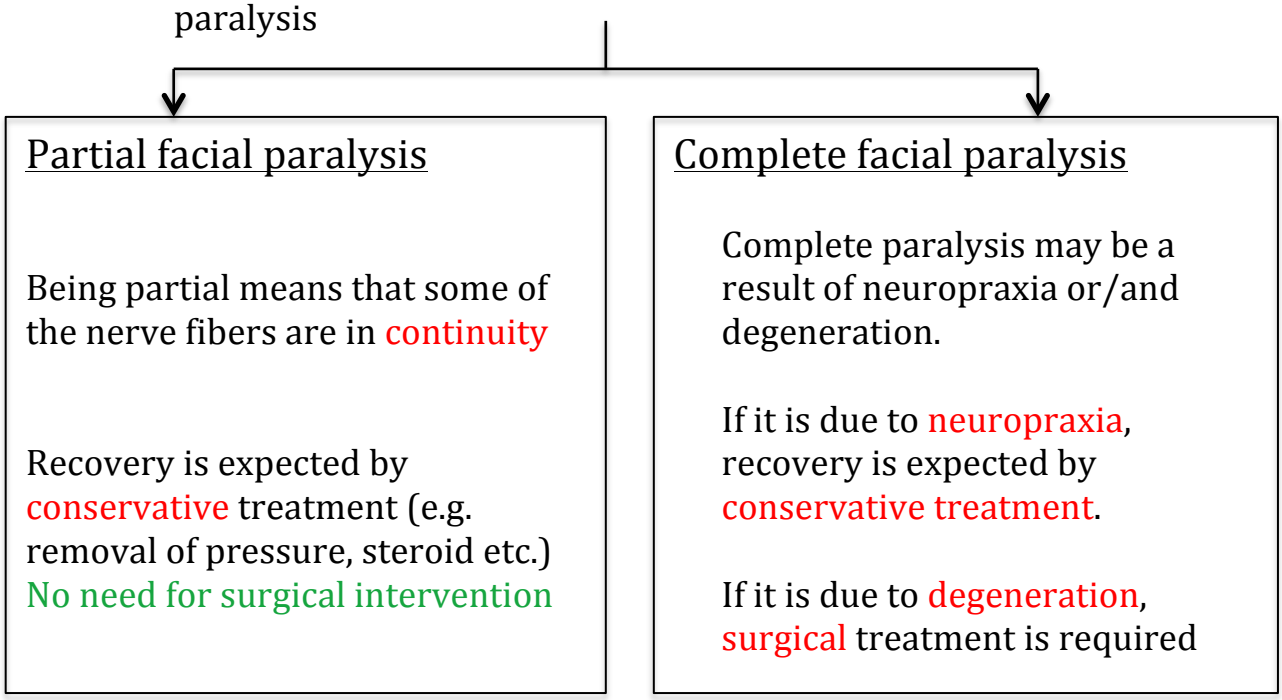
After regeneration Sometimes misdirection of the fibers occurs:
E.g. the fibers that go to the salivary gland deviate to sweat gland "the patient sweats when he eats"
Or to lacrimal gland "patient tears when he eats (crocodile tears)"

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

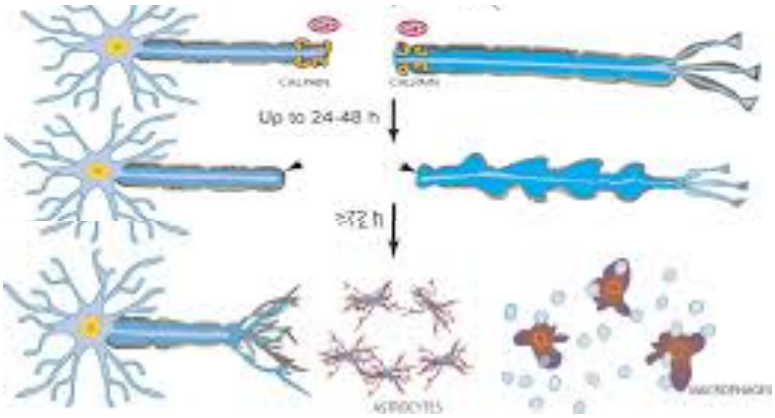


To differentiate between degeneration and neuropraxia electrophysiological tests are required

Electrophysiological tests

- It detects degeneration of the nerve fibers
- Useful only 48-72 hours following the onset of the paralysis

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)

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 results → degeneration.

Topognostic tests:

- Indicated in some cases to locate the site of the injury
  - Schirmer's test: test lacrimation function.
  - Stapedial reflex
  - Taste sensation

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"

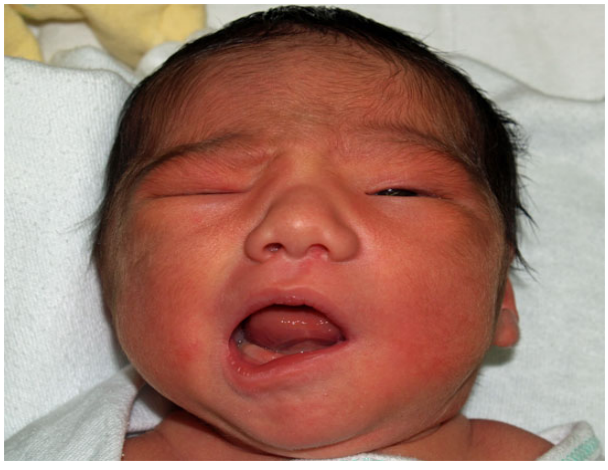
According to the cause itself:

- 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

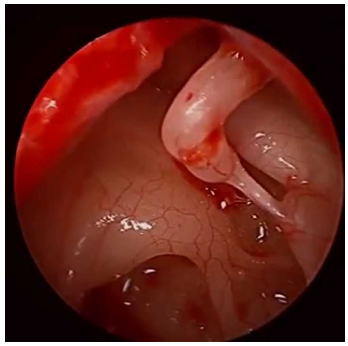
Most of them are partial and needs only conservative treatment.



Left side

Inflammatory causes of facial paralysis

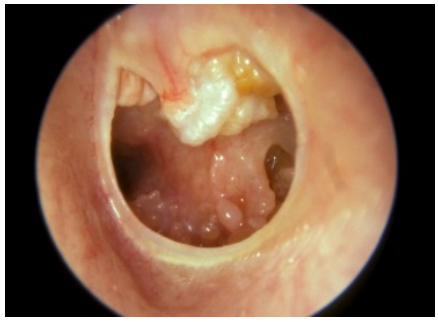
Facial paralysis in Acute Otitis Media (AOM)



- 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"

Facial paralysis in Chronic Suppurative Otitis Media (CSOM)

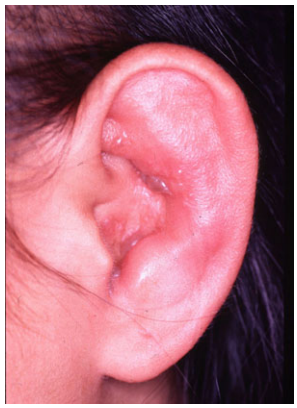
- Usually is due to pressure by cholesteatoma or granulation tissue causing bony erosion



- 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

Traumatic Facial Injury

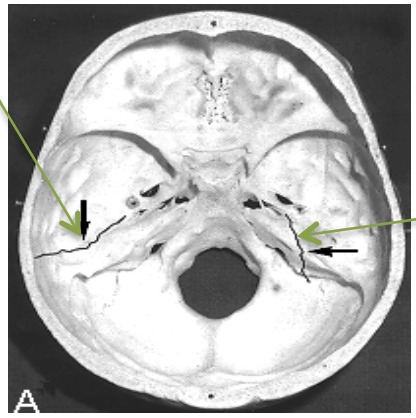
Causes:

Iatrogenic

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

Temporal bone fracture

Longitudinal



Transverse
"More likely to cause paralysis"

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"

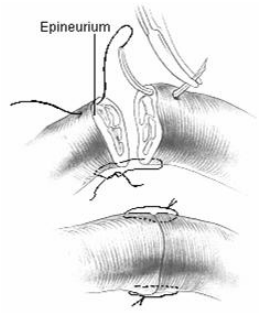
- If of immediate onset, it is usually complete and due to transection of the nerve
  - Surgical repair

- If borderline → conservative.

Surgical Repair

1. Direct Anastomosis

If the proximal and distal parts are identified and no distance between them

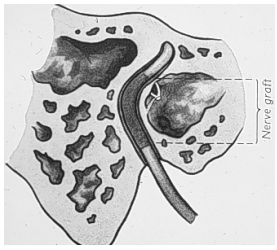


2. Nerve Graft

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



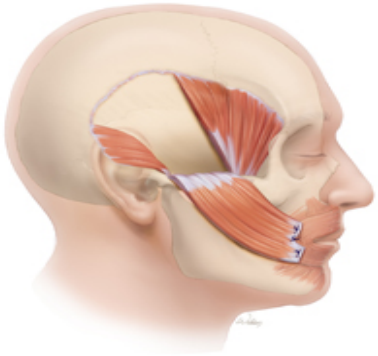
3. Nerve Transfer (anastomosis)

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 → vascular spasm"
- 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 cause most of patients recover with conservative treatment

Summary

Facial nerve has four main fibers: motor, secreto-motor, taste and sensory, and it has 3 main anatomical divisions: intracranial, cranial (intratemporal) and extracranial.

In case of facial nerve paralysis, the first thing to do is to decide wether it is lower or upper motor neuron lesion
If upper motor neuron lesion → Lower face paralysis of the other side.
Or lower motor neuron lesion → Whole face paralysis of the same side.

Facial nerve paralysis is manifested by: paralysis of facial muscles, phonophobia, dryness of eye and loss of taste.

Nerve damage can be partial or complete.
Partial: full recovery is expected
Complete: if neuropraxia → restoration of full recovery if cause is treated
If degeneration → delayed, incomplete or no recovery at all.

Electrophysiological tests differentiate between neuropraxia and degeneration, only useful after 48-72 hours.

Facial paralysis can be due to trauma, inflammation, neoplasm, congenital or idiopathic "bell's palsy"

Surgical repair can be done by direct anastomosis, nerve graft, nerve transfer or muscle flap.

Bell's palsy is the most common cause of acute facial paralysis
It is diagnosed by excluding all other causes.
Treatment is reassurance and steroids.

From Toronto Notes:

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 Topognostic testing MRI with gadolinium – enhancement of CN VII and VIII High resolution CT	**Rx:** Protect the eye to prevent exposure keratitis with patching or tarsorraphy 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 (errupt 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

MCQ's:

Q1: After a facial nerve injury a patient started to accumulate more food on his cheek and had weak chewing, which muscle was affected, and is responsible for such presentation?

A. Orbicularis oculi.
B. Orbicularis oris
C. Buccinators
D. Mandibular

Q2: In upper motor neuron facial nerve palsy:

A. Paralysis of the upper face
B. Paralysis of the lower face
C. Paralysis of the whole face
D. None of the above.

Q3: A 37-year-old intravenous drug user is referred by his GP for a suspected Bells Palsy. On examining his inner ear there are a number of vesicles visible on his eardrum. His cranial nerve examination reveals a weakness of the whole of the left side of his face. The most likely diagnosis is:

A. Steven Johnson Syndrome
B. HIV
C. Ramsay Haunt Syndrome
D. Bell's palsy

For mistakes or feedback

ENTteam432@gmail.com

Answers:
1: C
2: B
3: C