

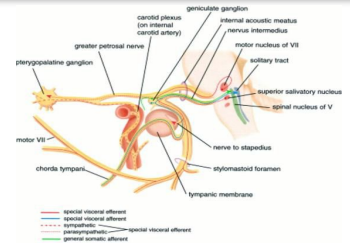
# Facial Nerve

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## Lecture Objectives:

- ★ Anatomy : course and branches
- ★ Causes of facial palsy : including bell's palsy, middle ear complication, traumatic, and ramsay hunt syndrome

# Facial nerve anatomy



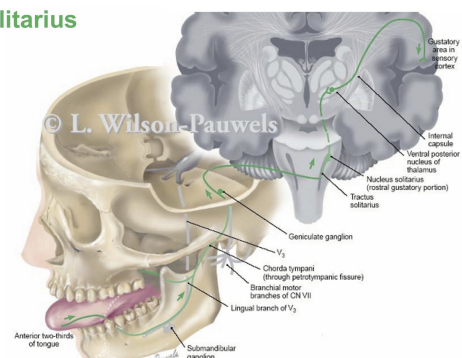
## ● Introduction:

- The facial nerve is the 7th paired cranial nerve
- It originates in the 7th nerve nucleus in the pons, and has fibers from several other nuclei
- It supplies muscles originating from the 2nd pharyngeal arch
- Provides motor fibers to the muscles of facial expression
- Consists of 10,000 neurons, 7000 of which have myelinated motor fibers (facial expression)
- Mixed nerve but mainly motor
- 70% motor and go to muscles of expression, 30% sensory (10% for taste, 10% for general sensation and 10% secretomotor)
- Why is it related to ENT? Passes through the ear with vestibulocochlear nerve (VIII)

## ● Nuclei (PONTS) 4 Ss:

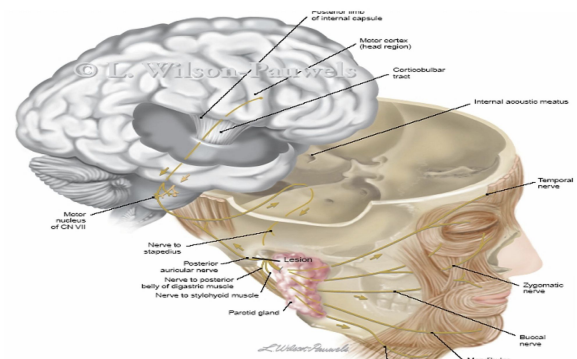
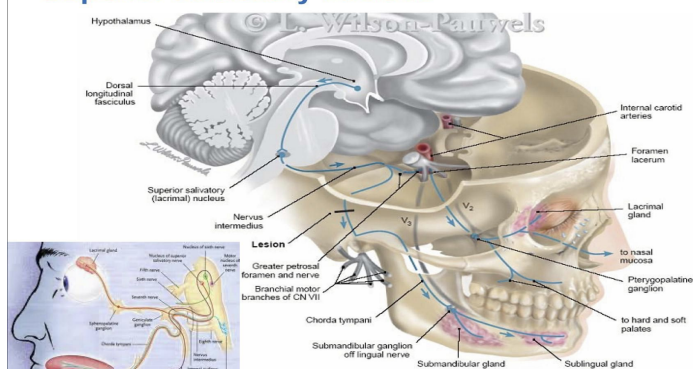
- **Seventh (motor) nucleus:** (in the pons) (70% of facial fibers are from this nucleus forming facial nerve proper)
  - Motor supply to the muscles:
    - Muscles of facial expression
    - Posterior belly of digastric (anterior -> trigeminal)
    - Stylohyoid muscle
    - **Stapedius muscle** (middle ear muscle)
- **Nucleus solitarius:** (in the medulla oblongata)
  - Taste sensation of the anterior 2/3 of the tongue
- **Spinal nucleus of the trigeminal nerve:**
  - General sensation (send certain fibers to facial nerve) (external auditory canal)
- **Superior salivatory (lacrimal) nucleus:** (in the pons)
  - Parasympathetic secretory fibers to the following glands:
    - Submandibular glands → salivary
    - Sublingual glands → salivary
    - Lacrimal glands
    - Nasal mucous glands
    - Palatine mucous glands
    - Pharyngeal mucous glands

Solitarius



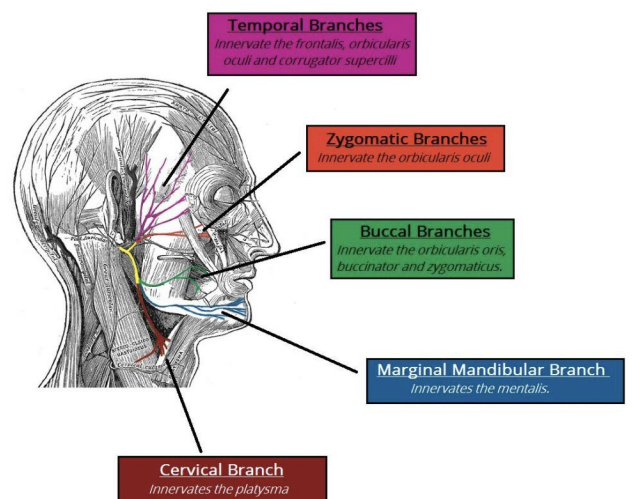
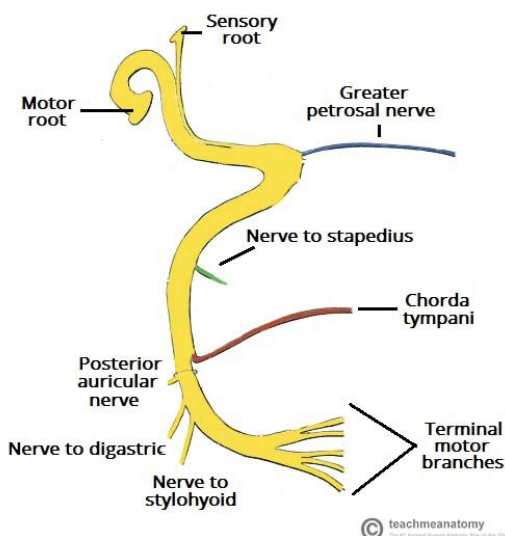
Imp → a Q can come from this part, the facial nerve that is responsible for general or special sensation comes from where?

## Superior salivatory nucleus



# Facial nerve anatomy (cont.)

- **Anatomical course of the facial nerve:** (will be discussed later in more detail)
  - **Intracranial part :**
    - The nerve arises in the pons (cerebellopontine angle)
    - Begins as 2 roots; a large motor root and a small sensory root (called nervus intermedius<sup>1</sup>)
    - The 2 roots travel through the internal acoustic meatus then they leave it and enter the facial canal<sup>2</sup>; a 'Z' shaped structure
    - **In the facial canal 3 events happen:**
      - The **2 roots fuse to form the facial nerve** (sometimes called the facial nerve proper)
      - The nerve **forms the geniculate ganglion**
      - **The nerve gives rise to:**
        - Greater superficial petrosal nerve (arises immediately distal to geniculate ganglion)
        - Nerve to stapedius
        - Chorda tympani
    - The facial nerve then exits the facial canal via the stylomastoid foramen (this exit is located just posterior to the styloid process of the temporal bone)
  - **Extracranial part:**
    - After exiting the skull; the facial nerve gives rise to the posterior auricular nerve
    - Then motor branches are sent to the posterior belly of digastric muscle & stylohyoid muscle
    - The main trunk of the nerve now termed the motor root of the facial nerve continues into the parotid gland
    - Within the parotid gland the nerve terminates by **splitting into 5 branches:**
      - Temporal branch
      - Zygomatic branch
      - Buccal branch
      - Marginal mandibular branch
      - Cervical branch



1. The fibers of solitarius, superior salivatory and spinal nucleus form nerve of wrisberg also called nervus intermedius then they join the fiber of the 7th nucleus & form 100% of the facial nerve. It is called facial nerve proper.
2. Also called fallopian canal

# Facial nerve anatomy (cont.)

## Branches of the facial nerve:

- **Greater petrosal nerve :**
  - Parasympathetic innervation to:
    - mucous glands of the oral cavity, nose & pharynx
    - Lacrimal glands → thus if this nerve is injured it will lead to dryness

## Chorda tympani:

- Innervates the anterior  $\frac{2}{3}$  of the tongue with the special sense of taste
- Parasympathetic fibers of chorda tympani combine with the lingual nerve (branch of trigeminal nerve) to innervate the submandibular & sublingual salivary glands
- Pain, temp and touch sensation for external auditory canal
- It is easily injured because it passes in the inner ear
- If injured → minor defect in taste (because it affects the anterior  $\frac{2}{3}$  of only one side of the tongue & there will be no dryness since the parotid gland is unaffected<sup>1</sup>)

## Nerve to stapedius:

- Supplies the stapedius muscle of the middle ear (responsible for dampening loud sounds as it stabilizes the foot of stapes → any damage to this nerve → hyperacusis & phonophobia)

## Posterior auricular nerve:

- Innervates the occipital part of the occipitofrontalis muscle
- Innervates the intrinsic and extrinsic muscles of the outer ear

## Nerve to posterior belly of digastric:

- This muscle is responsible for raising the hyoid bone

## Muscular branch to stylohyoid muscle:

- This muscle is also responsible for raising the hyoid bone

## Terminal branches: (in the parotid gland)

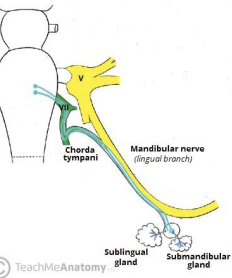
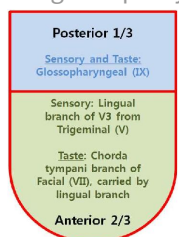
- **Temporal branch:**
  - Innervates frontalis
  - Orbicularis oculi
  - Corrugator supercilii
- **Zygomatic:** (important branch to protect the eye)
  - Innervates orbicularis oculi
- **Buccal:**
  - Innervates orbicularis oris
  - Buccinator
  - Zygomaticus muscles
  - If it gets injured → food will accumulate on cheek + weak chewing
- **Mandibular:** (important branch → paralysis causes cosmetically bad deformity)
  - Innervates the mentalis muscle + muscles of the angle of the mouth
- **Cervical:** (least important branch)
  - Innervates the platysma

### Tongue sensory supply:

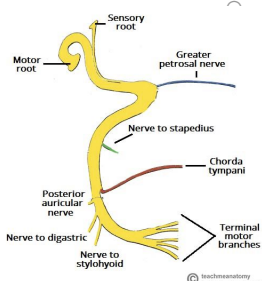
- the anterior  $\frac{2}{3}$  of the tongue sensory supply:**
- 1- taste → chorda tympani
  - 2-general sensation → trigeminal nerve (V3)

### Posterior $\frac{1}{3}$ sensory supply:

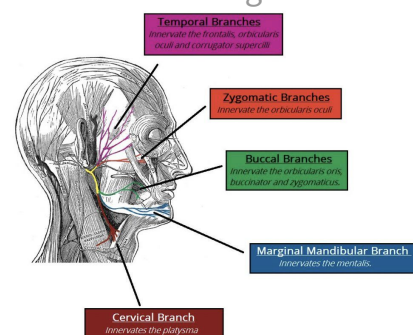
- 1-general sensation → glossopharyngeal
- Taste → glossopharyngeal



**IMP FOR EXAM!!!**



1. The parotid gland is supplied by the glossopharyngeal nerve (CN IX)



# Facial nerve anatomy (cont.)

## Types of facial nerve fibers:

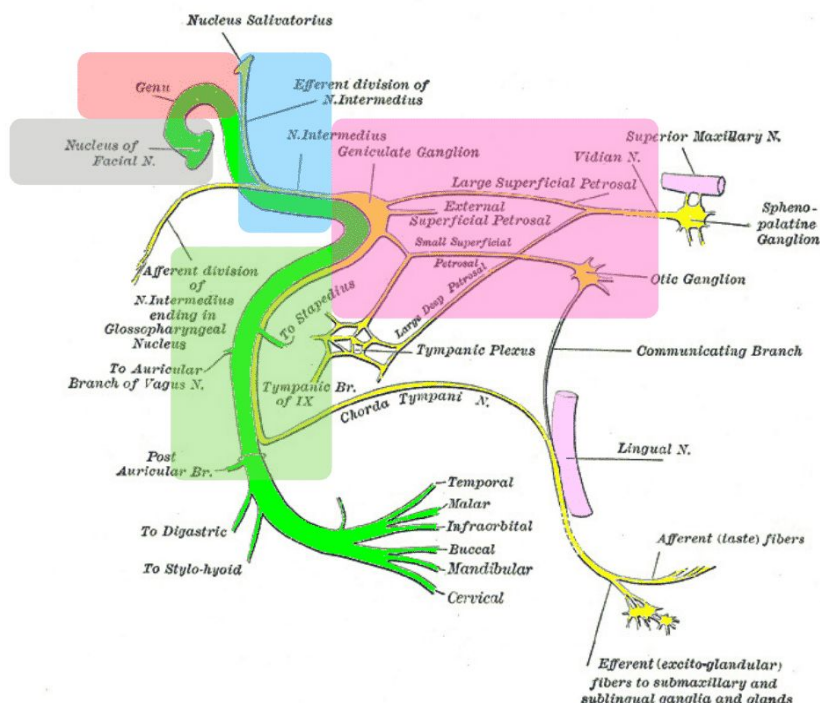
- **Motor fibers:**
  - To the stapedius (muscle of the middle ear) → nerve to stapedius branch
  - Facial muscles
- **Secretomotor fibers (parasympathetic):**
  - To the lacrimal gland & palatine/nasal mucous glands → greater petrosal nerve
  - To submandibular & sublingual salivary glands → chorda tympanii branch
  - DOES NOT SUPPLY THE PAROTID GLAND (PAROTID IS SUPPLIED BY CN IX)
- **Taste fibers:**
  - From the anterior 2/3 of the tongue & palate → chorda tympani nerve (posterior part of the tongue is innervated by glossopharyngeal CN IX)
- **Sensory fibers:**
  - General sensation → pain, temperature and touch from the external auditory canal (small part of the EAC)

## Facial nerve segments: (7 segments) → will be discussed in the next page

- Pons
- Cerebellopontine angle
- Internal auditory canal
- Labyrinthine segment → in fallopian canal (facial canal)
- Tympanic segment → in fallopian canal
- Mastoid segment → in fallopian canal
- External segment

## INTRA-CRANIAL COURSE OF THE FACIAL NERVE

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### Legend:

Cisternal Segment

Intracanalicular segment

Labyrinthine segment

Tympanic segment

Mastoid segment

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# Facial nerve anatomy (cont.)

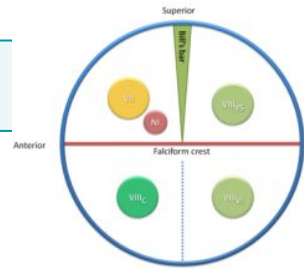
## Facial nerve segments:

### Intracranial (cisternal) segment

- Nuclei & cerebellopontine
- Zero branches
- The nerve emerges from the pons and travel through the cerebellopontine angle to the internal acoustic meatus

### Meatal (canalicular) segment

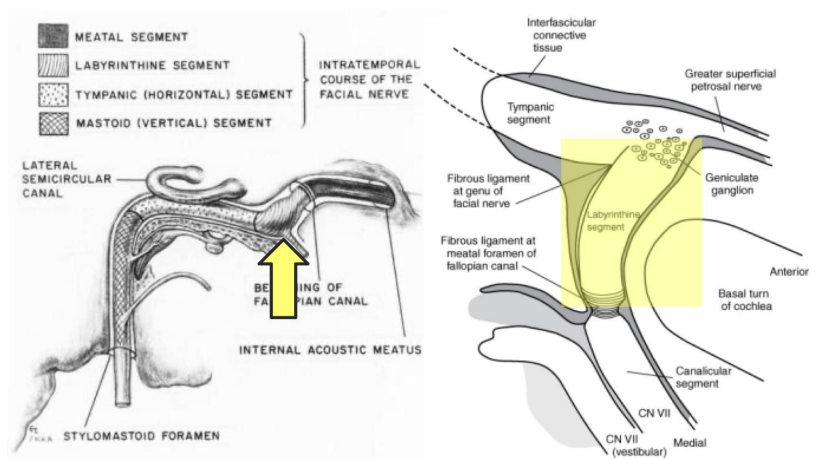
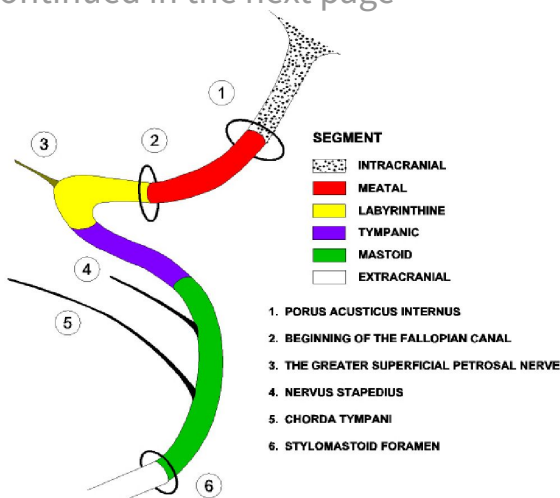
- Zero branches
- Internal auditory canal
- 8mm long
- The first entrance to the internal auditory canal
- The facial nerve enters with the vestibulocochlear nerve (7UP → 7th CN is in the anterior superior portion of the IAC and behind it is the vestibulo (sup and inferior) cochlear



### Labyrinthine segment

- 3 branches
- Inner ear segment
- Shortest & narrowest segment (3-4 mm long)
- In the fallopian canal (facial canal)
- Starts from the IAC to the geniculate ganglion (one of the most imp landmarks)
- The geniculate ganglion before entering the middle ear gives the 1st branch (1st genu) → greater superficial petrosal nerve (1st branch) → fibers for lacrimation & salivation
- It is the only segments that lacks arterial anastomosis → imp because if there was edema and compression of the nerve, ischemia results (risk of injury)
- Any problem here would cause lacrimation deficit; if the patient isn't able to lacrimate (dry eyes) → we would know the problem was before the greater superficial petrosal nerve
- If there was a lesion in the middle ear (e.g. otitis media), after the first branch → normal lacrimation and salivation (this process is called topography)

Continued in the next page



# Facial nerve anatomy (cont.)

- **Facial nerve segments:**

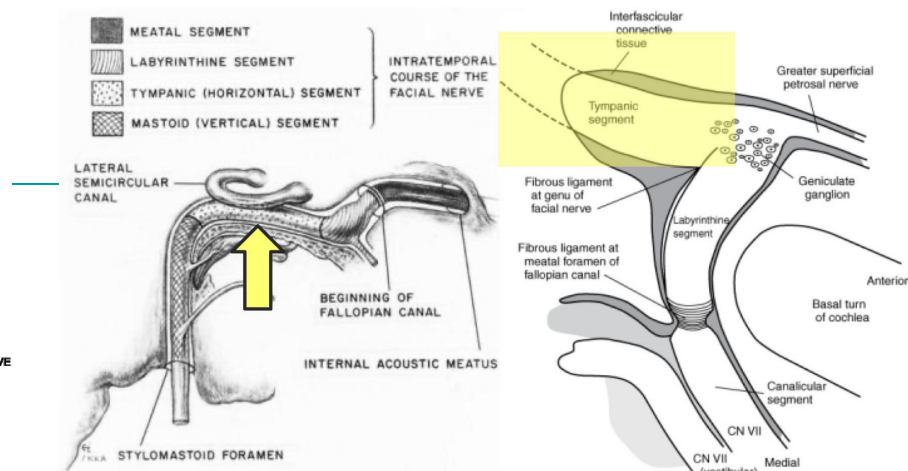
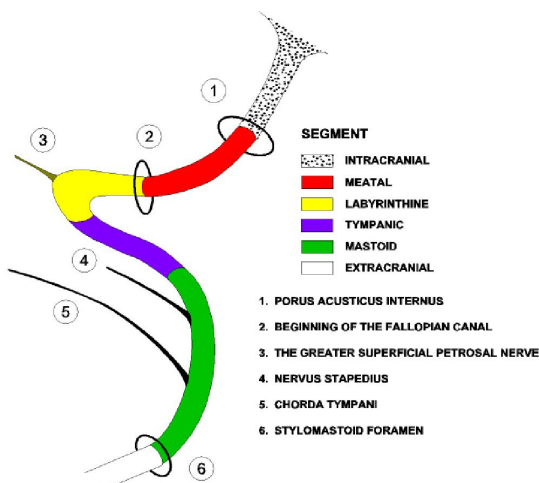
## Tympanic (horizontal) segment

- Zero branches
- Middle ear segment
- 8-11 mm long
- 2nd genu
- In the fallopian canal (facial canal)
- Starts **from the geniculate ganglion to the pyramidal eminence** (if you remove the drum you can see the facial nerve passing → any infection or cholesteatoma can easily put pressure on the facial nerve)
- 50% of people are dehiscent (dont have bone covering the nerve → that's why children might get acute otitis media which can lead to facial nerve palsy)

## Mastoid (vertical) segment

- External ear segment
- 8-14 mm long (longest part of the intratemporal course)
- In the fallopian canal
- Starts **from the pyramidal eminence to the stylomastoid foramen** (exits the skull)
- High risk of injury during surgery (since it is the longest)
- **3 branches:**
  - \* nerve to stapedius muscle
  - \* chorda tympani
  - \* and a sensory branch which joins the auricular branch of the vagus nerve
- If a lesion is affecting the stapedius nerve or chorda tympani, it might be at high mastoid or tympanic segments
- Patients with normal lacrimation but absent stapedial reflex would have a lesion here
- If a patient has absent lacrimation, absent stapedial reflex and absent taste in the anterior 2/3 of the tongue → the lesion was before all the branching

## Extratemporal (extracranial) segment → next page

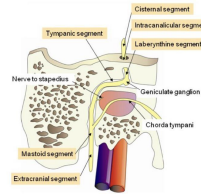


# Facial nerve anatomy (cont.)

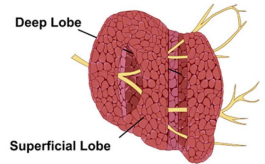
## ● Facial nerve segments:

### Extratemporal (extracranial) segment

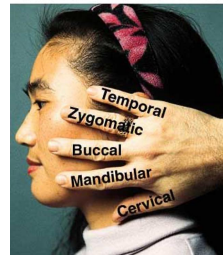
- 15-20mm long
- **Starts from the stylomastoid foramen to the parotid** → pes anserinus (latin for duck's foot) is the branching point of the extratemporal segment in the parotid gland, which **separates the gland into deep and superficial lobes**
- **Branches:**
  - \* posterior auricular nerve
  - \* nerve to posterior belly of digastric
  - \* nerve to stylohyoid muscle
- In the parotid it gives **5 terminal branches:**
  - \* temporal (this branch is at risk during surgery in this region) → most superior
  - \* zygomatic
  - \* buccal
  - \* mandibular (risk of injury in surgeries in the submandibular region<sup>1</sup>)
  - \* cervical



The Parotid Gland and Facial Nerve



Branches of the Facial Nerve



## Notes:

- Labyrinthine segment is the shortest and narrowest part of the facial nerve segments → that's why most patients who present with facial nerve palsy have facial nerve compression in this segment
- Mastoid segment is the longest intracranial segment → at risk of injury in trauma & mastoidectomy
- Parotid surgeries can cause facial nerve paralysis

## Embryology of the facial nerve

### ● Embryology:

- The facial nerve is developmentally derived from the 2nd pharyngeal (branchial) arch<sup>2</sup>
- The facial nerve supplies motor and sensory innervation to the muscles formed by the 2nd pharyngeal arch<sup>3</sup>
- **Divisions:**
  - motor division of the facial nerve → derived from the basal plate of the embryonic pons
  - The sensory division → originates from the cranial neural crest

### ● Variations anomalies:

- **Dehiscence** → a defect in the fallopian canal<sup>4</sup> where the nerve is exposed inside the middle ear
- It is mainly congenital where there is a deficiency of bone, thus the nerve will not be covered by a bone & lies immediately in the middle ear
- The nerve becomes more subjected to trauma and infection leading to facial nerve paralysis (when they have otitis media they get facial nerve paralysis)
- 50% of people are dehiscent
- **The facial nerve doesn't necessarily pass through the typical pathway in all patients, especially in those who have syndromes; surgeons have to be careful in surgeries.**

1. Injury causes paralysis of mouth depressors (paralysis of the angle of the mouth)  
2. The 2nd branchial arch is also called the hyoid arch  
3. Muscles of 2nd pharyngeal arch → muscles of facial expression + post. Belly of digastric + stylohyoid + stapedius  
4. Also known as → facial canal



# Facial nerve paralysis

## ● Introduction :

- 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)
- Results in asymmetric movement of some or all the muscles of facial expression

## ● Clinical presentation:

- Difficulty with smiling, frowning and expressing emotions
- Facial asymmetry is accentuated by attempting to close the eyes tightly or attempting to show the teeth or to whistle

### ○ paralysis of the facial muscles :

- Asymmetry of the face
- Inability to close the eyes → **orbicularis oculi**
- Accumulation of food in the cheeks → **paralysis of buccinator & orbicularis oris**

### ○ In LMNL: (upper pic)

- No wrinkles in the forehead when looking up → due to frontalis weakness
- Inability to close the eye completely → most accurate sign
- Flattening of the nasolabial fold
- Angulation of the mouth when showing the teeth (angle goes to the other side)

### ○ In UMNL: (lower pic)

- Looks normal at rest
- Orbicularis oculi and frontalis will not be affected

### ○ Phonophobia & hyperacusis: (lesion is above nerve to stapedius)

- Due to failure of stapedius attenuation reflex (because of loss of innervation to stapedius muscle) → uncomfortable feeling and unable to tolerate when exposed 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: (lesion is above geniculate ganglion)

- Inability to close the eye, loss of lacrimation
- Some people present with lacrimation and others with dryness of the eyes, why?
- Lacrimation is due to paralysis of orbicularis oculi as this muscle helps in draining the tears
- Dryness is due to affecting the greater superficial petrosal nerve which arises from the geniculate ganglion
- So if the paralysis is above the level of geniculate ganglion → dryness
- If below the level of the geniculate ganglion → no dryness

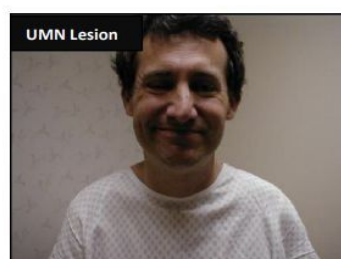
### ○ Loss of taste: (lesion is above origin of chorda tympani)

- Very minor because the chorda tympani supplies only the anterior  $\frac{2}{3}$  of one side of the tongue

## ● Examples:



Whole left side of the face is affected  
Grade 4-5



Only the lower part of the left side is affected





Fig. 16.1 Post-traumatic right facial palsy. Shown at rest (left) and on attempted eye closure.

# Facial nerve paralysis (cont.)

## ● UMN vs LMN facial nerve injury:

### Explanation: IMP!

- **The facial nucleus is divided into 2 parts:**
  - The part responsible for the muscles of the upper half of the face → receives innervation from both cerebral cortices
  - The part responsible for muscles of the lower half of the face → receives innervation from the contralateral cerebral hemisphere only
- UMN lesion → means a lesion above the nucleus
- LMN lesion → a lesion at the level of the nucleus or below, so any lesion from the internal auditory canal to the parotid gland is considered LMN lesion
- LMN lesions affect ALL the ipsilateral facial muscles
- UMN lesions spare the upper facial muscles and affect the contralateral lower face, because the forehead is innervated bilaterally
- Example → cerebrovascular accidents might cause UMN lesion
- In LMN lesions 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 they are spared

|                           |  UMN lesion                                      |  LMN lesion   |
|---------------------------|--|---|
| <b>Lesion location</b>    | <ul style="list-style-type: none"> <li>● Motor cortex</li> <li>● Connection from motor cortex to facial nucleus in pons</li> </ul> | <ul style="list-style-type: none"> <li>● Facial nucleus</li> <li>● Anywhere along CN VII</li> </ul>   |
| <b>Affected side</b>      | contralateral  | ipsilateral   |
| <b>Muscles involved</b>   | LOWER muscles of facial expression   | Upper and lower muscles of facial expression  |
| <b>Forehead involved?</b> | Spared, due to bilateral UMN innervation   | Affected  |
| <b>Other symptoms</b>     | None   | Incomplete eye closure (dry eyes, corneal ulceration), hyperacusis, loss of taste sensation in the anterior tongue (depends on where the lesion is) |

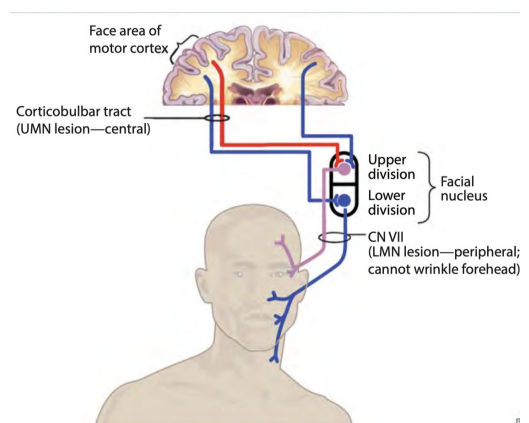
### ● Examples:



Whole left side of the face is affected



Only the lower part of the left side is affected



# Causes of facial nerve paralysis

## CAUSES OF FACIAL NERVE PARALYSIS

### Supranuclear and nuclear

Cerebral vascular lesions  
Poliomyelitis  
Cerebral tumours

### Infranuclear

Bell's palsy  
Trauma (birth injury, fractured temporal bone, surgical)  
Tumours (acoustic neurofibroma, parotid tumours, malignant disease of the middle ear)  
Suppuration (acute or chronic otitis media)  
Ramsay Hunt syndrome  
Multiple sclerosis  
Guillain-Barré syndrome  
Sarcoidosis

Table 15.1 Common causes of facial nerve paralysis

### Supranuclear and nuclear (upper motor neurone)

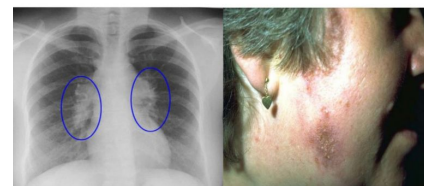
- Vascular lesions, e.g. stroke
- Intracranial tumours
- Multiple sclerosis

### 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

Table 16.1 Causes of facial nerve paralysis.

## Sarcoidosis (Heerfordt's)



## 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: Forceps delivery, Basal skull/temporal bone fractures, Facial injury, Penetrating to middle ear, Barotrauma, Lightning, Head and neck injuries & surgery parotid, mastoid and intracranial surgeries.
- Infection: Malignant otitis externa, Otitis media, Mastoiditis, Ramsey Hunt (Herpes zoster), Encephalitis, Polio, Syphilis
- Toxic - Neurologic
- 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**, **Guillain Barre**, MS, Mysethenia gravis, Sarcoidosis(Heerfordt's).
- Iatrogenic: Parotid surgery, Mastoid surgery, Local anesthesia, Acoustic neuroma
- **Bilateral: Neurofibromatosis**

## ● Evaluation of facial nerve:

- Careful history - physical exam - audiometry - CT/MRI/other - topography - electrophysiology
- Since the facial nerve is in close relation to the 8th CN, if a patient has facial paralysis, the auditory function should be evaluated
- Sometimes the lesion is originating from the 8th CN leading to signs & symptoms

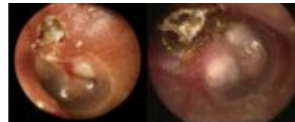
# Diagnosics:

## ● History & Physical Examination

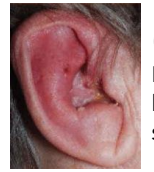
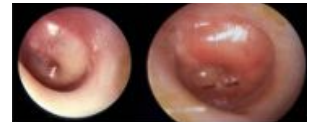
### ● History:

- Hearing loss or vertigo, SNHL, **by audiometry & acoustic reflex**
- Timing:
  - Sudden onset ? **aggressive or slow?**
  - Evolution over 2 - 3 weeks
- Presence of ear disease (1+2):
  - acute/ chronic otitis media
  - Cholesteatoma
- Vesicular eruption (3)
  - Ramsay- hunt syndrome
- Bilateral (4+5) **diseases that cause bilateral nerve damage**
  - Guillain-barre syndrome
  - Lyme disease
  - Intracranial neoplasm
  - **Neurofibromatosis**
- Recurrence(6):
  - Melkersson-Rosenthal syndrome: 4Fs
    - **Recurrent Facial nerve palsy**
    - **Furrowed tongue**
    - Faciolabial edema
    - Family history
- Severe pain, trauma, associated symptoms, past medical history

(1) Chronic otitis media

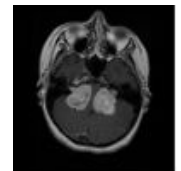


(2) Cholesteatoma



(3) Ramsay-hunt syndrome

(5)



(4)



(6)

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

### ● Examination: complete head and neck exam

- Ask the patient to:
  - Look up to test frontalis.
  - Close eyes to test orbicularis oculi.
  - Blow the cheek to test buccinators.
  - Whistling
  - Show the teeth for angulation (wide smile).
- LMNL:
  - Forehead wrinkling
  - Eye closure
  - Bell's phenomenon



# Diagnostics:

## ● House brackmann scale syndrome:

- **The most useful classification in the facial nerve palsy** (just know the name/ partial or complete/ that it is used because we can't just say nerve is paralyzed)
- Grade 1: Normal.
- Grade 2: Slight weakness. **Looks normal without movement.**
- Grade 3: Patient Can Close the Eye.
- Grade 4: Patient Can't Close the Eye.
- Grade 5 and 6: Masked Face, Asymmetric.

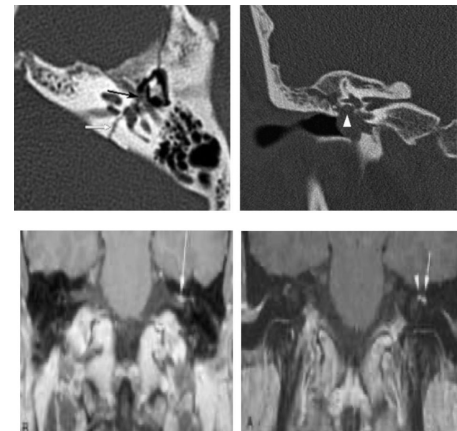
| Grade | Appearance                                       | Forehead                    | Eye                                | Mouth                             |
|-------|--|-----------------------------|------------------------------------|-----------------------------------|
| I     | Normal   | Normal                      | Normal                             | Normal                            |
| II    | Slight weakness<br>Normal resting tone           | Moderate to good movement   | Complete closure<br>Minimal effort | Slight asymmetry                  |
| III   | Non- disfiguring weakness<br>Normal resting tone | Slight to moderate movement | Complete closure<br>Maximal effort | Slight weakness<br>Maximal effort |
| IV    | Disfiguring weakness<br>Normal resting tone      | None                        | Incomplete closure                 | Asymmetric with maximal effort    |
| V     | Minimal movement<br>Asymmetric resting tone      | None                        | Incomplete closure                 | Slight movement                   |
| VI    | Asymmetric                                       | None                        | None                               | None                              |

## ● Radiology:

- **Localized lesion:**
  - **Computed tomography (CT) (inratemporal):**
    - Trauma
    - Mastoiditis
    - Cholesteatoma
  - **Magnetic resonance imaging (MRI) (intracranial):**
    - used in case of infection or inflammation
    - Nerve enhancement due to infection
    - Exclude neoplasm (vestibular schwannoma or neurofibromatosis)
    - Usually MRI enhancement in labyrinthine segment.

Fracture

cholesteatoma



# Diagnostics:

## ● Topography: Important, MCQs

- Indicated in some cases to locate the site of the injury
- Not used now but it was used before to localize the lesion
- 1. Schirmer test → greater superficial petrosal (test lacrimation function)
  - 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. 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)
- 2. Stapedial reflex → stapedial branch with tympanometry if normal we know problem after the branch, routinely done with audiogram these days
- 3. Electrogustometry “taste sensation” → chorda tympani
- 4. Salivary flow → chorda tympani “comparing salivation on both sides”
- 5. Imaging



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

## ● Audiology:

- Evaluate pathology of eighth cranial nerve
- Bell's palsy most common facial paralysis
  - Symmetric audiological function normal hearing
  - Absent ipsilateral acoustic (stapedial) reflex; problem in labyrinthine segment or after the branch
- Retrocochlear pathology:
  - Asymmetrical thresholds asymmetric hearing (VIII cranial nerve palsy)

## ● Electrophysiological Tests

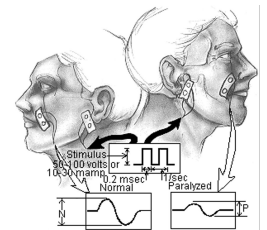
- Provides prognostic information.
- Differentiate between conduction block (neuropraxia) and degeneration of nerve fibers (neurotmeses) in clinically complete facial paralysis
- Principle: stimulate the nerve and look for response -
- Tests:
  - Electroneuronography (ENoG)
  - Electromyography (EMG) tests the strength of the muscle, important for prognosis
  - Nerve Excitability Test (NET)
  - Maximum stimulation test (MST):  
ACUTE=Acute+Complete+Unilateral+Three Days+Evaluate
    - used for prognostic follow up to see how the nerve is healing

# Diagnostics:

## ● Electrophysiology cont;

| Nerve Excitability Test (NET)   | Electroneurography (ENoG)  |
|---|--|
| <p>Stimulate the nerve in the stylomastoid foramen and compare both sides.</p> <p>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>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</p> |

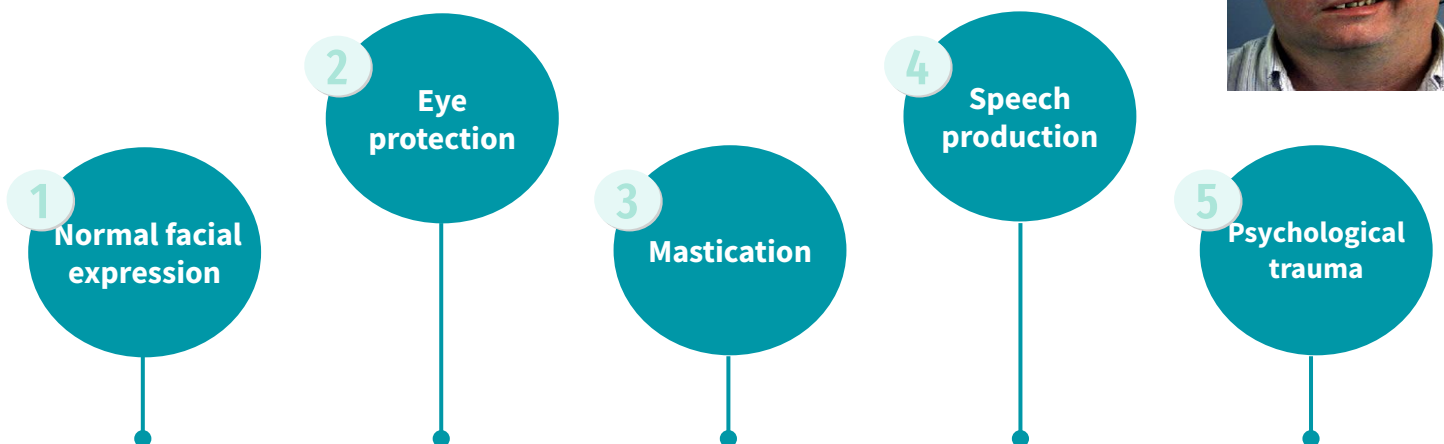
- Not useful in the first 3 days after onset of complete paralysis (the findings always lag several days behind the biologic events themselves)
- Most applicable in the evaluation of acute paralysis (while the nerve is in the degenerative phase)
- It detects degeneration of the nerve fibers
- Useful only 48-72 hours following the onset of the paralysis.
- Useful for cases where there is no known cause such as trauma.
- 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.
- **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



## Complication of facial paralysis



Facial Paralysis will severely affect:



since the facial nerve supplies facial muscles of expression

because patient can't close mouth

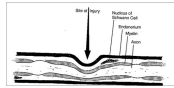
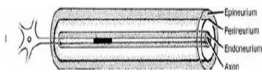
The most significant complication is the social isolation these patients

- 1, 2, 3 and 4 are severely affected by facial paralysis

# Pathophysiology of Nerve Injury:

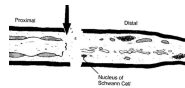
## 1. Neuropraxia (conductive loss)

- 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.



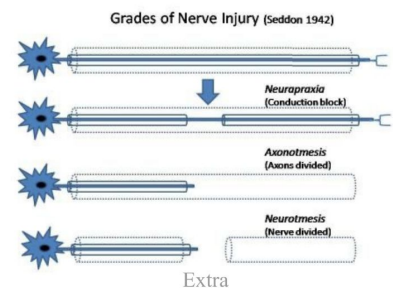
## 2. Neurotmesis (Degeneration)

- Wallerian degeneration (distal to lesion), wallerian degeneration of the distal part of the nerve, takes 2-3 days to occur.
- Axon disrupted, loss of tubules, support cells destroyed
- Anatomical block due to complete transection of the facial nerve.
- 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)”



## 3. Axonotmesis

- Wallerian degeneration (distal to lesion)
- Endoneural sheaths intact
- 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.





# Principle of Management of Facial Nerve injury

- Take care of the eye, the patient is unable to close his eye so the cornea will be exposed to trauma, protect it by:
  - a. Artificial tears if the patient has dryness.
  - b. Protect them from dust by wearing sunglasses
  - c. See ophthalmologist in case of any irritation
  - d. Cover the eye while sleeping
- Treatment of the cause if applicable.
- Treatment of the nerve varies according to the degree of the paralysis.

## Clinically partial Paralysis

- Being partial means that some of the nerve fibers are in **continuity** and the rest are neuropraxic and/or degenerated. Recovery is expected by **conservative treatment** (e.g. removal of pressure, steroid etc.). No need for surgical intervention.

## Clinically complete Paralysis

- may be either due to neuropraxia or due to degeneration or due to a mix of both.
- 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.

### Notes:

- If we plan on doing a surgery for a nerve after exposure to trauma, we **wait 1 year** before doing it to make sure that permanent damage has occurred; as the nerve could **regenerate** by itself. Unless you could prove with imaging that the nerve is cut, then go for surgery.
- Regeneration occurs after 48-72 hrs; that's why we wait 48 hrs before doing electrophysiological tests.

## Bell's palsy

- Most common diagnosis of acute facial paralysis, if **slowly progressive** it is NOT Bell's palsy.
- **Diagnosis is by exclusion**
- The **acoustic reflex will be absent (ipsilaterally)** because the Lesion is above the stapedial branch
- Unknown cause, LMNL, limited duration, minimal symptoms, spontaneous recovery, no sensory loss
- 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). 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.

# Bell's palsy

|                                 |  |
|---------------------------------|--|
| <p><b>Pathophysiology</b></p>   | <ul style="list-style-type: none"> <li>● Edema of the facial nerve sheath along its entire intratemporal course, “fallopian canal <b>mainly labirithinate segment or inner ear</b>” → Swelling of the nerve → Compression and ischemia → Complete paralysis, if mild edema → neuropraxia, if severe → degeneration.</li> <li>● <b>Most common cause of facial paralysis, diagnosed by exclusion.</b></li> </ul>  |
| <p><b>Etiology</b></p>          | <ul style="list-style-type: none"> <li>● <b>Edema due to vascular</b> (ischemia of the nerve) vs. viral <b>measles in particular, cold weather but the exact etiology is still unknown</b> (not proven)</li> </ul>   |
| <p><b>Clinical Features</b></p> | <ul style="list-style-type: none"> <li>● Can't close mouth and can't speak well</li> <li>● <b>Sudden unilateral facial paralysis</b>, occurs after exposure to cold weather could be vascular spasm, <b>Patient may have in the beginning pain behind the ear → few hours later facial paralysis</b></li> <li>● Partial or complete</li> <li>● No other manifestations apart from occasional mild pain <b>behind the ear</b>. No discharge, no parotid swelling, not following trauma.</li> <li>● May recur in 10% (6 – 12%), previous history of paralysis in the same side 12%, other side 6%.</li> <li>● 80% complete recovery</li> <li>● 10% satisfactory recovery</li> <li>● 10% no recovery</li> </ul>   |
| <p><b>Risk factors</b></p>      | <ul style="list-style-type: none"> <li>● Family history</li> <li>● pregnancy</li> </ul>  |
| <p><b>diagnosis</b></p>         | <ul style="list-style-type: none"> <li>● Weakness of the entire half of the face –</li> <li>● In doubt → CT and MRI scans</li> <li>● MRI may show contrast enhancement of the facial nerve</li> </ul>  |
| <p><b>Treatment</b></p>         | <ul style="list-style-type: none"> <li>● <b>Reassurance.</b> Prevent corneal drying</li> <li>● <b>Physiotherapy within first few days only.</b> <ul style="list-style-type: none"> <li>● Natural tears</li> </ul> </li> <li>● Medication (steroids, antiviral, vasodilators)           <ul style="list-style-type: none"> <li>● Isotonic saline</li> </ul> </li> <li>● Surgical decompression in selected cases           <ul style="list-style-type: none"> <li>● Strips of skin tape to close the eye</li> </ul> </li> <li>● <b>Remove bony compression to release the nerve, Usually not unless it is recurrent; then there is an anatomical problem.</b></li> <li>● Eye protection.(Drops to prevent eye dryness and exposure keratitis)</li> <li>● <b>Prednisolone oral (treatment of choice, but only if started in the 1st 24 hrs)</b></li> <li>● <b>Depends on the grade, if 2,3 → conservative and hydration drops</b></li> <li>● <b>If worse → eye closure and aggressive steroids.</b></li> </ul> |
| <p><b>Medication</b></p>        | <ul style="list-style-type: none"> <li>● <b>Steroids</b> to decrease edema, <b>antivirals, vasodilators.</b> Antiviral and vasodilators only given in combination with steroids, not effective alone.</li> <li>● Corticosteroids (80 mg/day po), within 24 to 48 h of onset for 1 wk, decreased gradually over the 2nd wk</li> <li>● Antivirals (Acyclovir ), less degrees of facial weakness</li> </ul>   |

# Bell's Palsy cont;

## Surgical decompression "in selected cases"

if patient is getting worse

- Debate over years
- Patients with 90% degeneration.
- Within 14 days of onset.
- Done by removing part of the bone around the nerve to decrease the compression
- Surgery is not usually done because most of patients recover with conservative treatment

## prognosis

- 80% Complete recovery
- 10% Satisfactory recovery
- 10% No recovery
- Partial usually recovers within 4-6 weeks while complete may take up to 6 months

## Inflammatory Causes of Facial Paralysis

### 1. Facial paralysis in Acute Otitis Media (AOM) MCQ

- Mostly due to pressure on a dehiscence nerve by inflammatory products and fluid (pus) accumulation.
- Usually is partial and sudden in onset.
- Treatment: is by antibiotics and myringotomy; open the drum and drain the fluid to the relieve the pressure (incision & drainage)
- Mastoiditis can cause pressure on the nerve.
- 3 days history of fever, earache and facial palsy = AOM
- left picture: patient has right facial paralysis and a red congested membrane.
- In mastoiditis similar SXS to AOM but for treatment we put tube to release pus if fistula appears then mastoidectomy is indicated.

#### Otitis Media



#### Mastoiditis



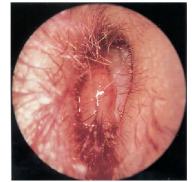
### 2. Facial paralysis in Chronic Suppurative Otitis Media (CSOM):

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

# Inflammatory Causes of Facial Paralysis

## 3. Malignant otitis externa (4Ds)

- **Osteomyelitis of the temporal bone**, osteomyelitis of the external auditory canal bone
- Granulation (at the junction of bony and cartilage) obscured Tympanic membrane, drum and Tympanic membrane can't be seen
- 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
- Scenario of old diabetic pt (immunocompromised) that has ear discharge and multiple Cranial Nerves get affected and they show the picture (we can't see the drum) say MOE or osteomyelitis of temporal bone
- **4Ds:**
- Diabetes mellitus (DM) or **anything considered immunocompromising**
- Discharge (purulent)
- Discomfort
- Dysfunction of cranial nerves (can be more than one)



Granulation obscured TM  
drum and tm can't be seen

## 4. Herpes Zoster Oticus (Ramsay Hunt Syndrome)

- This is due to herpes zoster (varicella zoster virus) infection of the geniculate ganglion
- Herpes zoster affection of cranial nerves VII, VIII, and cervical nerves, vestibulocochlear dysfunction (multi-nerves involvement).
- Elderly patients
- **Symptoms:**
  - facial palsy, pain, **skin rash vesicles (the hallmark, help to differentiate it from Bell's palsy), SNHL (sensorineural hearing loss) usually irreversible, vertigo (due to 8th cranial nerve involvement), which improves due to compensation from the other side "takes few weeks"**
  - Severe pain precedes the facial palsy & the herpetic eruption in the ear **within concha, where the branches of the facial nerve innervate (sometimes on the tongue and palate)**
  - **Facial nerve recovers in about 60%.** Recovery of facial nerve function is much less likely than in Bell's palsy
- Treatment by: **Acyclovir**, steroid to relieve edema and symptomatic
- Similar symptoms to bell's palsy but it is more severe with more symptoms, for example here there is a skin rash
- Treatment is equivalent Bell palsy acyclovir and steroids
- Poor outcome
- 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).
- you will be asked about it in exam, patient has facial palsy and skin rash and Ear symptoms it's Ramsay hunt syndrome



# Traumatic Facial Injury

## 1. Birth trauma (congenital facial palsy)

- 80-90% are associated with birth trauma forceps use
- At birth the facial nerve is about 1 mm behind the bone and each year the distance increase by 1 mm, which puts the newborn at risk of injury. Until it reaches 1 cm and stops at this level.
- 10-20% are associated with developmental lesions, **ex neurofibromatosis**
- Most of them are partial and need only conservative management

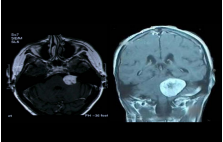


# Traumatic facial injury

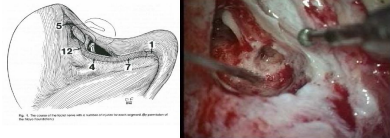
## 2. Iatrogenic

- Operations at the CP “cerebellopontine” angle, ear and the parotid glands.
- **Acoustic neuroma** resection or mastoidectomy.
- Local anesthesia.

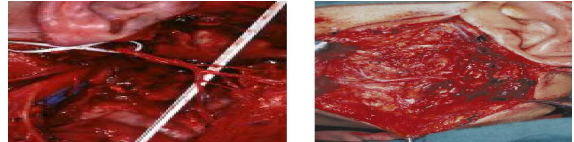
Acoustic neuroma (schwannoma)



Iatrogenic facial nerve palsy Mastoidectomy



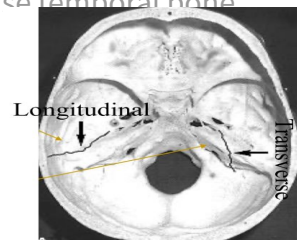
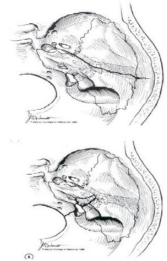
Iatrogenic facial nerve palsy Parotid gland (total parotidectomy)



## 3. Temporal bone fracture MCQ

### 2 classification:

- Longitudinal: **بمىسى مع العظم**
  - 80% of Temporal Bone Fractures.
  - 15-20% Facial Nerve involvement.
- Transverse: **بوص العظم من النصف**
  - 20% of Temporal Bone Fractures.
  - 50% Facial Nerve Involvement (more likely to cause paralysis)
  - Complete transection
- 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 (skull base fractures in general):**
  - Raccoon eyes sign
  - Battle's sign
  - also called Skull base fracture signs
  - Ossicles injury
  - CSF or blood leak from ear



### • Pathology of traumatic facial nerve injury:

- edema
- transection of the nerve



Battle's sign



Raccoon eyes sign



### • Management of traumatic facial nerve injury:

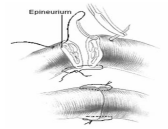
- If it is delayed in onset, it is usually incomplete and is due to edema → **Conservative** “steroids and relieve the pressure” directly after the trauma there is no problem because edema takes time
- If of immediate onset, it is usually complete and due to transection of the nerve: → immediate **Surgical repair** (re → If borderline; conservative)



### SAQ

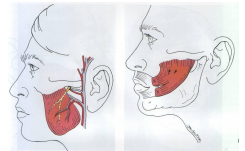
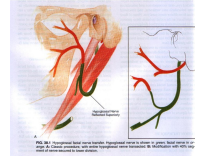
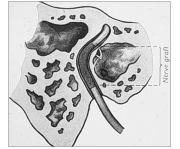
- What is the name of this sign?
- What does it indicate?
- What are the possible associated features?
  - Hearing loss.
  - Facial nerve paralysis.
  - CSF leak.

# Traumatic Facial Injury

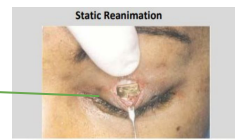


## Surgical Repair: -

- **Direct Anastomosis:** If the proximal and distal parts are identified and no distance between them. **Repair the external sheath, we don't touch the inner part.**
- **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, or 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.
- **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. **We compromise hypoglossal nerve for facial nerve; because facial nerve loss is disfiguring.**
- **Muscle flap:**
  - If the distal and proximal parts can't be identified.
  - For cosmetics only "temporalis or masseter muscles are used"



Gold plate insertion to put pressure on eyelid for complete closure and avoidance of eye dryness.



## Botulinum toxin

- Synkinesis and hypertonia
- Advantages
  - Ease of use
  - Selective
- Disadvantages
  - Temporary
  - Repeated every 3 months.
- Synkinesis is a neurological symptom in which a voluntary muscle movement causes the simultaneous involuntary contraction of other muscles. An example might be smiling inducing an involuntary contraction of the eye muscles, causing a person to squint when smiling

## Neoplastic

- Malignant parotid lesion, Cholesteatoma, Acoustic neuroma, CN VII tumor, Meningioma.
- SPORT > Neoplasm
  - Slowly progressive
  - Persistent >4 months
  - Other C.N. Ex SNHL
  - Recurrent
  - Tumor History

## Conclusion

- Facial paralysis sequelae (significant):
  - Functional
  - Cosmetic
  - Psychological
- The primary goals of facial reanimation:
  - Corneal protection
  - Symmetry at rest
  - Smile restoration

# Questions from the doctor's slides

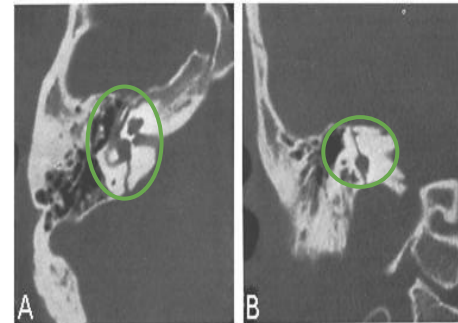
## ● Case:

- **What is the most likely diagnosis?**
  - Left LMN facial paralysis (most likely bell's palsy), be no wrinkles.
- **Mention 2 common causes?**
  - All diseases of this lecture causes LMN facial paralysis
  - 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?**
  - LMN facial nerve paralysis
  - CSF leak
  - Ossicles injury and other signs of skull base fracture (raccoon eye, battle's sign)



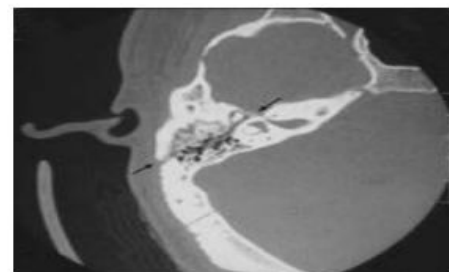
## ● 34 years old with LMN facial paralysis:

- **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?**
  - LMN facial nerve paralysis
  - CSF leak
  - Ossicles injury and other signs of skull base fracture



# Doctor's notes 437A

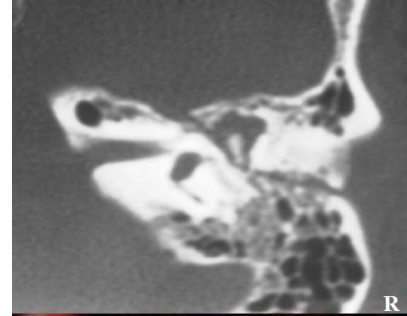
- **What is the embryological origin of facial nerve?**
  - 2nd branchial arch
- **Where is the position of the facial nerve during its course of the auditory canal?**
  - The upper part (7UP)
- **Facial nerve segments:**
  - **What is the narrowest segment of the facial nerve, that is easily compressed?**
    - Labyrinthine segment
  - **Which segment of the facial nerve is highly exposed and more prone to injury after otitis media?**
    - Tympanic segment
  - **What is the largest segment of the facial nerve that is easily injured after trauma?**
    - Mastoid segment
- **Name one of the branches that supplies the lacrimal gland & what will happen if it gets injured?**
  - Greater superficial petrosal nerve, shedding of tears while eating (crocodile tears)
- **Name one of the branches that gives taste sensation and what will happen if it gets injured?**
  - Chorda tympani, metallic taste
- **Name the 5 major terminal branches of facial nerve :**
  - Temporal
  - Zygomatic
  - Buccal
  - Mandibular
  - Cervical
- **What is the name of the classification used to grade facial nerve palsy:**
  - Brackmann classification
  - 1=almost normal
  - 2=partial degeneration
  - 3<=can close their eyes
  - 4>=can't close their eyes
  - 6=almost all muscles are affected (masked face)
- **What is the management of facial nerve palsy:**
  - Eyes → artificial tears, might need to put a special stripes to close the eyes while sleeping
  - Synkinesis → botox
- **What is the management of a patient who had a complete nerve cut?**
  - Surgery (like surgical repair)
- **What is bell's phenomenon?**
  - Upward and outward movement of the eye, when an attempt is made to close the eyes
- **What's the prognosis of facial nerve palsy in general?**
  - 90% completely recover



## SAQ

- 20 years old female brought to the ER after a car accident, her face was covered red because of the blood and it was clearly asymmetric, she lost one of the nasolabial folds, and she can't close her eyes at that side. An urgent CT was done, which showed the following pic.

- What is the most likely diagnosis that explains the patient presentation?
  - Facial nerve palsy
- After seeing the CT what's the most likely etiology?
  - Temporal bone fracture (longitudinal)



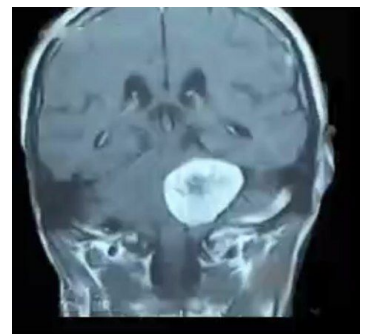
- Patient presented to the clinic complaining of ear pain, vomiting and spinning sensation. On face inspection the doctor noted an abnormality (pic.).

- What's the abnormality seen on this picture?
  - Asymmetric face Facial nerve palsy
- Which cranial nerves are most likely to be affected?
  - CN VII, VIII (facial and vestibulocochlear)
- Which syndrome does this patient most likely have?
  - Ramsay hunt syndrome



- A patient presented with asymmetric face, metallic taste, drooling, hearing loss, an MRI was done which showed the following picture.

- What is the most likely lesion seen on this MRI?
  - Acoustic neuroma (schwannoma)



# THANK YOU!

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