Facial Nerve

See the slide for the Pictures only, Study from the Team!
427 is not a good source to study this lecture from

Notes:

- Facial Nerve Fibers:

- **Motor** → Supplies Stapedius muscle and facial muscles
- **Secreto-motor** → Submandibular gland, sublingual gland and lacrimal gland
- **Taste** → Anterior 2/3 of the tongue (Corda Tympani)
- **Sensory** → External auditory meatus

-Anatomical division:

1. Intracranial: Include the Facial Nerve Nuclei and the cerebellopontine angle. Then passes the Temporal bone.
2. Cranial (Intratemporal)
3. Extracranial: When it leaves the skull.

Details about divisions: (Course of Motor fibers)

1. **Intracrainal part:** It has the nuclei WHICH ARE:
    - Superior salivary nucleus→ **Parasympathetic**
    - Motor Nucleus (most part of the facial nerve)
    - Nucleus tractus solitarii (Solitary Nucleus) → **taste**

NOTE: the doctor mentioned something about the **cerebellopontine angle. (Just know where it is located)**

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

Therefore, **Upper motor lesions** spare the upper facial muscles (frontalis M. and Orbicularis Occulai M.) and affect the contralateral lower face. **Lower motor lesions** affect all the ipsilateral (whole half) facial muscles.

2. **Intratemporal part:** From the Internal Auditory Meatus (association with **vestibulocochlear nerve**). Then goes to Fallopian canal.

Fallopian Canal has a horizontal part and a vertical part. AND its divided to:

- Labyrinthine → Related to the inner ear
- Tympanic → Related to the middle ear
- Vertical part (mastoid) → Related to the external ear (Branches: 1- Supplies Stapedius Muscle 2- Corda Tumpani)

3. **Extracranial part:** After it leaves the temporal bone through the **Stylomastiod foramen** then it goes through the parotid gland. In the parotid gland it divides into its FIVE branches which are:
    - Temporal → Supplies frontalis
    - Zygomatic → orbicularis oculi (doctor said this is the most important branch)
    - Buccal → buccinator
    - Mandibular → orbicularis oris
    - Cervical → Platysma

    The Most Important Divisions are: Zygomatic (Closure of the eye) and Mandibular (it will cause a very bad cosmetic deformity)

    The Least Important is the Cervical

Course of Secreto-motor and taste

Secreto-motor (parasympathetic):

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

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

The Corda Tympani then joins the Lingual then goes to glands (Submandibular and Sublingual)

- Taste:

takes the same course of the Parasympathetic but in the opposite way (from peripheral to central). Fibers from the palate goes to the Geniculate ganglion → nucleus solitarii. The fibers from the anterior 2/3 of the tongue → Corda tympani → nucleus solitarii.

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

For Example:

If the lesion is above the level of the corda tympani → patient will loose the function of corda Tympani he will have dryness and loose of taste sensation

If the lesion is below the level of corda tympani → patient will have only facial paralysis with normal taste sensation.

Variations and Anomalies:

Only one normal variation the doctor talked about, which is: At the junction of the Tympanic vertical part there is: (dehiscent) there is no bony coverage of the nerve (no protection). It's a common anomaly. This subjects to the facial nerve infection and trauma in the middle ear.

Clinical Manifestations:

- **Paralysis of facial muscles:**
    - Asymmetry of the face
    - Inability to close the eye
    - Accumulation of food in the cheek

- **Photophobia** → Stapedius muscle (LMNL)
- **Dryness of the eye** → impaired drainage
- **Loss of taste** → anterior 2/3 of the tongue

Note: in the pictures: the doctor explains the difference between upper and lower motor lesions. AND in mild cases sometimes you can't judge if the patient has paralysis of facial muscles at rest.

Pathologies of facial nerve injury:

Facial nerve paralysis could be:

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

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

3- **Mixed**

Principles of management of facial nerve paralysis:

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

A) Partial facial paralysis:

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

B) Complete facial paralysis:

- If it is due to neuropraxia, recovery is expected by conservative treatment
- If it is due to degeneration, surgical treatment is required

  - To differentiate between degeneration and neuropraxia **electrophysiological tests** are required

- Electrophysiological tests:

- It detect degeneration of the nerve fibres
- Useful only 48-72 hours following the onset of the paralysis (Why) because degeneration will take 2-3 days to occur So, if you stimulate the nerve immediately after an injury it will get stimulated.

Electrophysiological tests are:

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

Nerve Excitability Test (NET): The current's thresholds required to elicit just-visible muscle contraction on the normal side of the face are compared with those values required over corresponding sites on the side of the paralysis.

- Advantage: Simple, available everywhere

- Disadvantage: Subjective only (rough estimation either you see it move or not)

Electroneurography (ENoG): 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.

- Advantage: More accurate measurements, Objective

- Disadvantage: Not available as common as NET

Interpretation of he tests:

- Not useful in the first 48 – 72 hours
- After 48-72 hours (the time required for degeneration to take place)
    - Normal results means that there is no degeneration (Neuropraxia)
    - Abnormal results means degeneration
    - So, If the patient is presenting already with partial paralysis this test will be waste of time because you already know that there is some fibers acting and some are not !

For partial you do:

Topognostic tests → Indicated in some cases to locate the site of injury (Knowing the level of the lesion) not very useful cause we can know the level by other methods like X-ray.. etc

Tests:

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

Causes of Facial nerve paralysis:

we can distribute it as Anatomical or Pathological

Anatomical:

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

Pathological:

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

1) Congenital Facial Palsy (LMNL)

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

2) Inflammatory causes of facial paralysis:

A- Facial paralysis n AOM (Acute Otitis Media)

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

B- Facial paralysis on CSOM (Chronic supportive Otitis Media)

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

3) HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME) (Intra Temporal Causes)

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

4) Traumatic facial injury:

A- Iatrogenic (surgical) → Operations at the CP angle, ear and the parotid glands

B- Temporal bone fracture:

1- Longitudinal→ More common. Less severe, might cause conductive hearing loss, usually does not affect facial nerve.

2-Transverse → Less common, More severe. More likely to affect facial nerve.

- Pathology: (Why the Facial Nerve get effected) ?

  Edema: will lead to partial paralysis

  Transaction of the nerve: will lead to complete paralysis.

Management of traumatic facial nerve injury:

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

Surgical Repair:

- Direct Anastomosis → No gap in nerve
- Nerve Graft → Gap (From the great ocular Nerve)
- Nerve Transfer → Distal part is there, but NO proximal

(From the Hypoglossal Nerve; it is supplying the tong Motor but paralysis to ipsilateral to the tong is not going to effect it function that is why we use it)

- Muscle Flap → NO distal and NO proximal

(either -Buccinators or – temporalis)

5) BELL'S PALSY

Most common diagnosis of acute facial paralysis

Diagnosis is by exclusion (exclude all other causes of face paralysis)

Pathalogy:

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

Etiology: Two theories: Viral vs Vascular

Clinical Features: (Don't diagnose it at least its typical)

- Sudden onset unilateral FP
- Partial or complete
- No other manifestations apart from occasional mild pain (such as deafness or any other thing)
- May recur in 6 – 12%

Prognosis:

- 80% complete recovery
- 10% satisfactory recovery
- 10% no recovery

Treatment:

- Reassurance
- Eye protection
- Physiotherapy
- Medications (steroids, antivirals, vasodilators)
- Surgical decompression in selected cases

Surgical Management:

- Debate over years
- Patients with 90% degeneration
- Within 14 days of onset