THE FACIAL NERVE

Course / Nerve Fibers

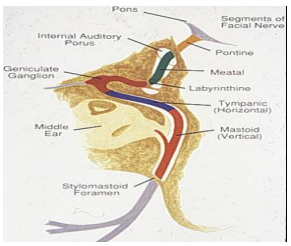
- ***Motor:*** to the stapedius and facial muscles of expression.
- ***Secreto-Motor:*** parasympathetic fibers to the submandibular, sublingual salivary glands (via the chorda tympnai) and to the lacrimal glands (via the greater superficial petrosal nerve). {Parotid supplied by CNIX-glossopharyngeal}
- ***Taste:*** from the anterior two thirds of tongue and palate. (Afferent fibers – lingual nerve – chorda tympani – goes intracranially)
- ***Sensory:*** from the external auditory meatus (external canal) - very few fibers

Anatomical Divisions

I. ***Intracranial:*** Nuclei & cerebellopontine

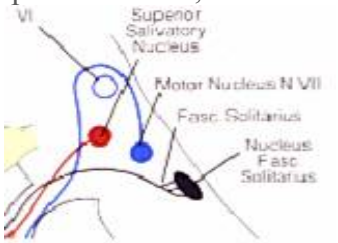
II. ***Cranial (intratemporal):***
1. Meatal: the facial nerve leaves the cranium thru the internal auditory meatus along with the vestibulocochlear nerve. So in the meatal part we have the Facial – Vestibular-Cochlear Nerves. Lesions will affect the vestibulochoclear nerve.
2. Fallopian canal {Labyrinthine (inner ear), Tympanic (middle ear) and Mastoid (external ear)} – At the Labyrinthine, the facial nerve forms geniculate ganglion prior to entering the facial canal, which carries the taste fibers at the junction of labyrinthine with the tympanic part.

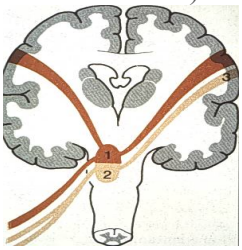
III. ***Extracranial (extratemporal)*** Here the facial nerve leaves the stylomastoid prominence and passes thru the superficial surface of the parotid and branches within the parotid gland.
1. Temporal
2. Zygomatic
3. Buccal – supplies the Buccinator
4. Mandibular – Oribicularis oris and submandibular gland (maybe injured during facial surgery and leads to angulation of the mouth)
5. Cervical – supplies the Platysma



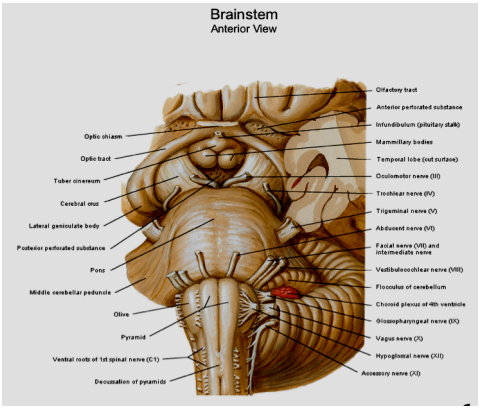
THE INTRACRANIAL PART

1. The Nuclei (here: the motor fibers, the superior salivary nucleus for the parasympathetic fibers, and the nucleus solitaris for the taste fibers.)





2. Cerebellopontine angle

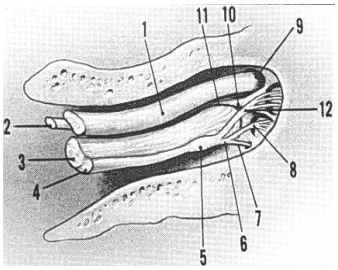


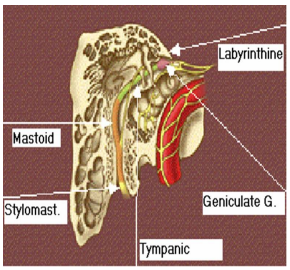
Notice:

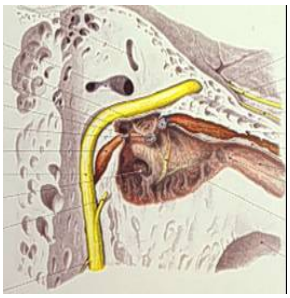
Facial nerve (VII) and intermediate nerve

Vestibulocochlear nerve (VIII)

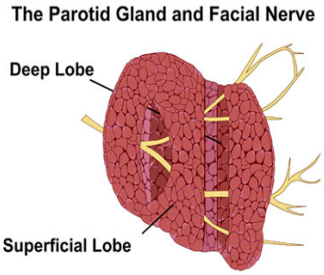
THE INTRA-TEMPORAL (CRANIAL) PART

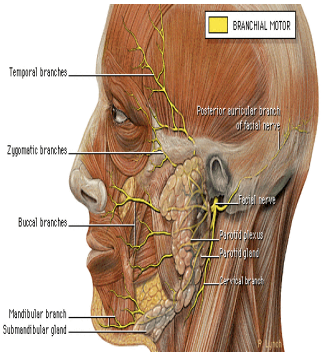






THE EXTRACRANIAL PART





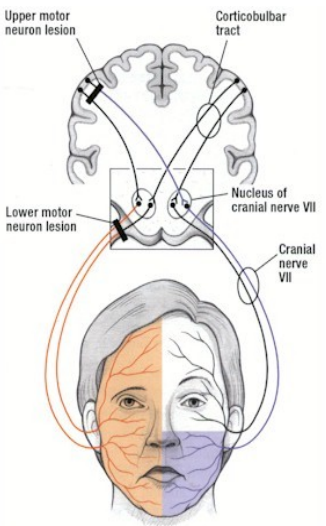
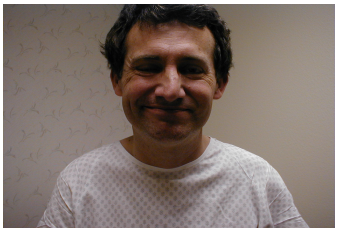


Figure 2a: The color lines show the distribution of facial muscles paralyzed after a supernuclear lesion of the corticobulbar tract and after a lower motor neuron lesion of the facial nerve.

- Upper motor lesions spare the upper facial muscles and affect the contralateral lower face.
- Lower motor lesions affect all the ipsilateral facial muscles.
- If one side is affected then UMNL of the opposite side, if LMNL then the entire face is affected.

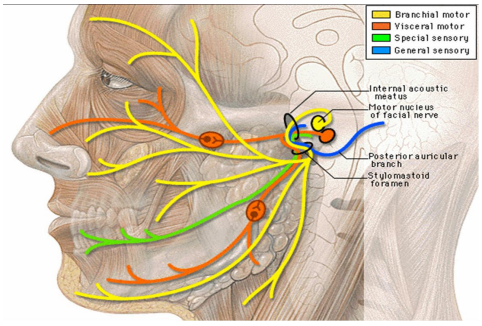
UMNL	LMNL
Intact upper ½ due to bilateral representation	Upper and lower halves of the face are paralyzed
Preservation of the emotional movements of the face (extrapyramidal)	Loss of emotional face movements
Spastic paralysis No wasting of the muscles No reaction of degeneration Electrical-Diagnostic tests – Normal	Flaccid paralysis Wasting of the muscles Reaction of degeneration Electrical tests – Abnormal



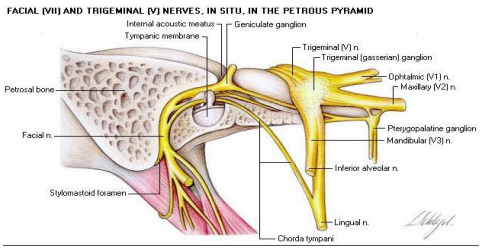
Upper Motor Neuron lesion

Lower Motor Neuron Lesion

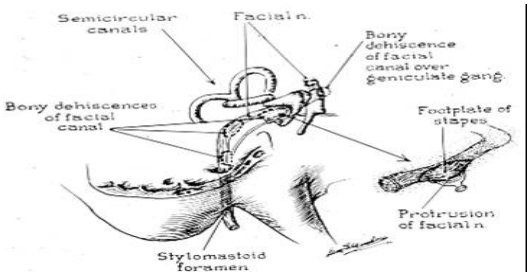
Distribution of the facial nerve fibers



The secreto-motor and the taste fibers



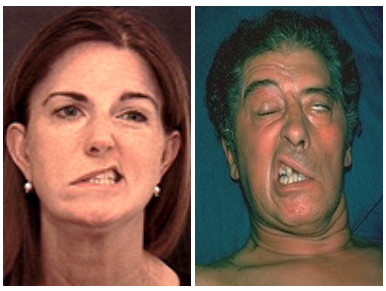
Variations and Anomalies

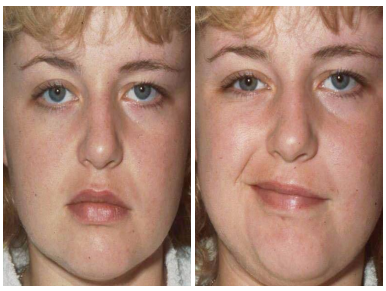


The tympanic portion of the facial nerve is important clinically because it is dehiscent in 50% of normal patients. The nerve may lie in the middle ear and can be injured during surgery; therefore, we have to know before surgery if there are any variations in the nerve because it is more prone to trauma.

Clinical Manifestations of Facial Nerve Injury:

- Paralysis of facial muscles
  - Asymmetry of the face (paralysis of Frontalis)
  - Inability to close the eye (paralysis of orbicularis oculi)
  - Accumulation of food in the cheek (due to paralysis of buccinator)
  - Wrinkling of the forehead
  - Angulation of the mouth on the affected side.
- Phonophobia (paralysis of the stapes, only if the lesion is above the level of the geniculate ganglion. Ex. If you have a lesion in the middle ear, it wont affect the ear)
- Dryness of the eyes
- Loss of taste in the anterior 2/3 of the tongue on the affected side
  - Sometimes there maybe only partial paralysis only when the patient tries to move. So to test the Facial nerve we ask the patient to:
    1. Close the eyes
    2. Close the mouth
    3. Blow the cheeks
    4. Show the teeth.

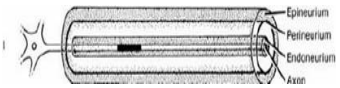


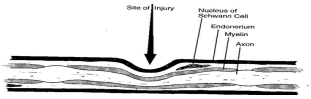


Pathophysiology of Facial Nerve Injury

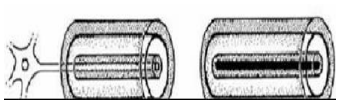
Sunderland Nerve Injury Classification:

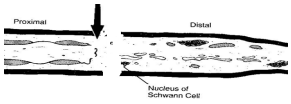
- ***Neuropraxia*** (Conduction Block): compression leads to damage of the axoplasm and physiologic conduction block (mechanical block). Partial loss of the nerve (weakness), the nerve is still intact and Recovery is complete within 1-4 weeks once the cause is treated, no need for surgical intervention. The nerve will not be functional that is paralyzed but it is still intact (no degeneration).





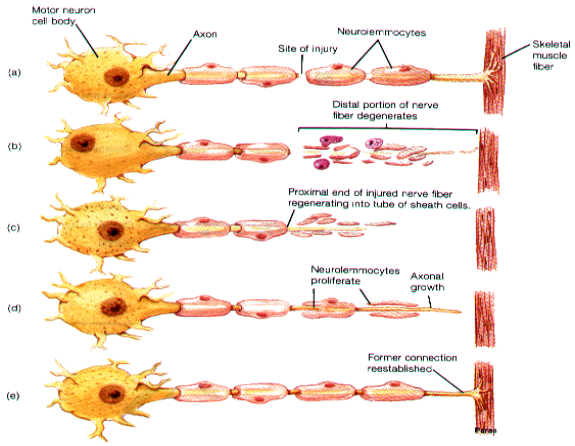
- ***Neurotmeses*** (Degeneration): loss of the myelin tubes with loss of neuron continuity. Loss of myelin tubes, new axons have an opportunity to get up and split causing associated mouth and eye movement (synkinesis). Also traumatic neuroma may form made up of the enlarged axons that cannot cross the cut area. Surgery is a must to approximate the nerve endings. Recovery takes about 6 – 12 months after surgery to reanastomose the cut ends.





- ***Axontmesis:*** loss and degeneration of the axons but the endoneural tubes persist. The axons grow into the intact empty myelin tubes at a rate of 1mm/day. Recovery occurs within 2-3 months. Surgical interference is required if 90% degenration occurs.
- Initially all present the same but it is important to differentiate to know the proper management and prognosis.

Regeneration



When a nerve is transected, we bring the cut ends to close proximity for regeneration to occur. Regeneration occurs at a rate of 1mm/day, so it takes about 6 months for the nerve to regenerate.

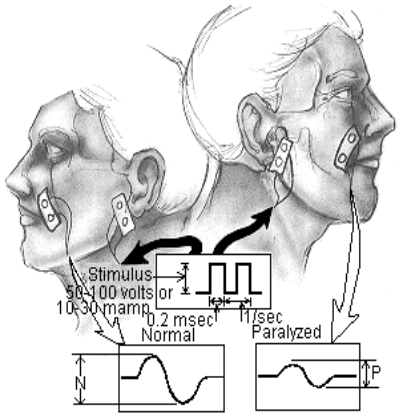
Investigations:

I. To diagnose the cause of the lesion
- CT-Scan
- MRI
- X-Ray
- Angiography

II. To diagnose the level of the lesion
- Audiological Evaluation
- Acoustic Reflex
- Shirmer's test for Lacrimation
- Salivary flow and PH

III. To diagnose the status of the nerve and muscles after injury

- **Electrophysiologic Tests**
  - Detect degeneration of the nerve fibers, to differentiate between neuropraxia and neurotmesis for proper management and prognosis.
  - Useful only 48-72 hours following the onset of the paralysis. (not reliable in the first 24 – 48 hours following trauma because nerve degeneration takes time to show and may occur 3 days following trauma.)
  - ***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. Stimulate the nerve at the stylomastoid foramen and compare the threshold of the *electric currents* which cause contraction and compare both sides. – subjective and not very reliable.
  - ***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. (more reliable, apply a stimulus then record the contractions and compare the *contraction percentage* on both sides. But it has to be done by a professional)



Indications:

- In clinically **complete** facial paralysis to differentiate between conduction block (neuropraxia) and degeneration of nerve fibers (neurotmeses) {in partial paralysis some fibers are still intact and there will be some activity}

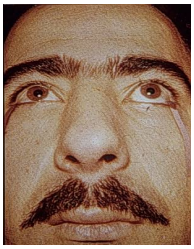
Interpretation of the test:

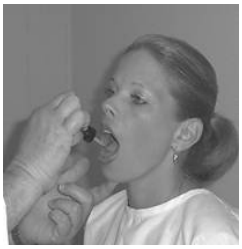
- Not useful in the first 48 – 72 hours. After 48-72 hours (the time required for degeneration to take place)
- Normal results means that there is no degeneration (Neuropraxia)
- Abnormal results means degeneration

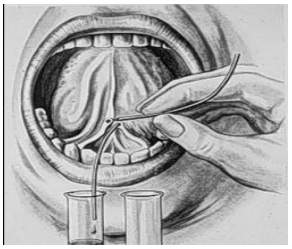
Topognostic Tests:

- Indicated in some cases to locate the site of facial nerve injury (cranial-intracranial-extracranial). - Not useful or commonly used
  1. Schirmer's test: Test the lacrimation function. {compare the lacrimation on both sides, if equal (below geniculate ganglion) if one side is dry (above the geniculate ganglion)}.
  2. Stapedial reflex – very practical, to know if the lesion is above or below the stapedius.
  3. Taste sensation – if it is affected then the lesion is above the level of the chorda tympani.
  4. Salivary flow – invasive and not informative

NOTE: {The **House-Brackmann score** is a score to grade the degree of nerve damage in a facial nerve palsy. The measurement is determined by measuring the upwards (superior) movement of the mid-portion of the top of the eyebrow, and the outwards (lateral) movement of the angle of the mouth. Each reference point scores 1 point for each 0.25cm movement, up to a maximum of 1cm. The scores are then added together, to give a number out of 8.} – not imp





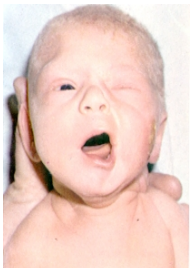


Causes of Facial Paralysis

- Congenital: Birth trauma
- Traumatic: Head and neck injuries & surgery
- Inflammatory: Otitis Media, Necrotizing Otitis Externa, Herpes Zoster
- Neoplastic: Meningioma (intracranial), malignancy of the ear (cranial) or parotid (extracranial part)
- Neurological: Guillain-Barre syndrome, multiple sclerosis
- Idiopathic: Bell's palsy

Another classifiction:

- Intracranial causes
  - Vascular Lesions of the pons (hemorrhage, thrombosis, embolism)
  - Acoustic neuroma – in the cerbellopontine angle.
  - Meningioma, Surgery, Trauma, etc.
- Cranial (intratemporal) causes (above) – Otitis Media, Trauma, etc.
- Extracranial causes
  - Parotid tumors
  - Surgical or cut wounds in the face.

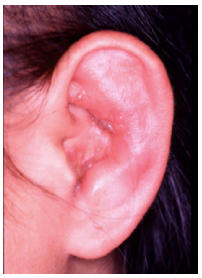


I. Congenital Facial Palsy

- 80-90% are associated with birth trauma and forceps delivery. Mostly partial paralysis.
- 10 -20 % are associated with developmental lesions.

II. Inflammatory causes of Facial Palsy

1. Facial Paralysis in *AOM {Acute Otitis Media}* – rare
   - Mostly due to pressure on a dehiscent nerve by inflammatory products (pus) on the head of the facial nerve
   - Usually is partial and sudden in onset
   - Treatment is by antibiotics and myringotomy (incision in the eardrum to relieve pressure on the dehiscent facial nerve)



2. Facial Paralysis in *CSOM {Chronic Suppurative Otitis Media}*
   - Usually is due to pressure by cholesteatoma or granulation tissue
   - Insidious in onset – destruction of the bony wall of the facial nerve due to pressure of the cholosteatoma.
   - May start partial and progress to complete (depends when the patient presents)
   - Treatment is by immediate surgical exploration and excision of the cholesteatoma (even if it is mild) and "proceed" (nerve management depends on the extent of the injury, either we resect it or just relieve the pressure)
3. *Ramsay Hunt Syndrome {Herpes Zoster Oticus}*
   - Herpes zoster affection of cranial nerves VII, VIII, and other cervical nerves due to infection of the geniculate ganglion.
   - Sudden onset, maybe partial or complete (most cases)
   - Facial palsy, pain, skin rash, SNHL and vertigo
   - Vertigo improves due to compensation, but complete recovery less likely compared to Bell's palsy.
   - SNHL is usually irreversible
   - Facial nerve recovers in about 60%
   - Treatment by: Acyclovir (antiviral), steroid and symptomatic treatment

III. Traumatic Facial Nerve Injury

1. Iatrogenic: Operations at the CP angle, ear and the parotid glands
   Surgical trauma of the facial nerve:
   - If evident intraoperatively then repair the facial nerve.
   - If evident postoperatively
     - If immediate and complete – reexplore
     - If delayed onset and incomplete (conservative Tx) it is usually due to edema – wait and observe So after any surgery that involves the facial nerve, examine the patient as soon as he recovers from GA to make sure there is no facial injury to treat if there is any immediate paralysis to the nerve.
2. Temporal bone fracture

Longitudinal	***Transverse***
Most common	Less common – more likely causes facial nerve paralysis
Causes conductive hearing loss	Patients may lose vestibular function
Less intense	Requires a more intense blow to fracture the skull
Best prognosis	Worse prognosis
Facial paralysis affect 20%	Facial paralysis affected 50% of cases
Delayed and incomplete facial paralysis	Immediate and complete facial paralysis

IV. Bell's Palsy (IMP)

- The most common diagnosis of acute idiopathic facial paralysis
- Sudden onset, very imp to exclude other possible causes.
- Male = Female, risks increase in DM and pregnancy
- Cause:
  1. Viral {HSV, EBV}
  2. Vascular ischemia: maybe Primary – to cold and emotional stress, or Secondary – to edema.
  3. Hereditary: narrowing of the fallopian canal, 6-8% of patients have a positive family history.
  4. Autoimmune
- Clinical Features: Classical facial nerve injury signs (sudden onset unilateral facial nerve defect):
  1. Inability to close lid.
  2. Asymmetry of the face
  3. Epiphoria "excessive tears"
  4. Noise intolerance
  5. Loss of taste

  - To diagnose Bell's palsy the above manifestations (facial nerve defect) and mild pain, if any other symptoms (swelling, severe pain, etc) are present then exclude facial palsy, it's most likely NOT Bell's palsy.
- Pathophysiology: due to edema of the facial nerve (due to viral or ischemia)
- Diagnosis: by exclusion of other causes, Hx, PE, Investigations, Nerve conduction studies.

- Prognosis:
  - Excellent in most patients, spontaneous recovery without intervention.
  - 80% - 90% will recover completely
  - Good prognostic factors are:
    1. incomplete paralysis – not all the face
    2. young age
    3. slow progression – early improvement
    4. normal salivation
    5. normal taste
    6. electro-diagnostic tests – normal

Treatment:

A. Bell's Palsy

- Reassurance (imp)
- Care of the Eye-Eye Protection, patch, artificial tears, etc. (cornea will be exposed may lead to keratitis and corneal ulceration)
- Care of the muscles – physiotherapy
- Medical Treatment – corticosteroids (to reduce edema of the nerve within the bony canal - controversial) + Antivirals + Vasodilators {ALL may reduce degeneration and synkinesis, or may hasten recovery} – if the patient presents early
- Surgical Treatment:
  1. Nerve Decompression of labyrinthine – within 14 days in patients with 90% degenerations (controversial)
  2. Surgical anastomosis:
     - If the nerves are cut and near each other, regeneration will occur without any intervention at a rate of 1mm/day.
     - For proximal injuries, nerve anastomosis by nerve graft > Great Auricular nerve
     - For Distal injuries, nerve anastomosis using the Hypoglossal nerve.
     - If neither distal nor proximal, then, muscle flap using the Temporalis, or Masster.

B. Traumatic Paralysis

3. Immediate paralysis after surgery: do an immediate or next morning surgical exploration – nerve decompression and nerve suture. Or Nerve rerouting (shorten the nerve and reanastomose the cut ends), Or Nerve grafting using the great auricular or sural nerve grafts.
4. Delayed onset of paralysis after surgical trauma: general care and medical managments and ENoG daily starting from the third day. If degeneration reaches 90% within 6 days from the onset of paralysis > surgical decompression of the nerve.