بسم الله الرحمن الرحيم

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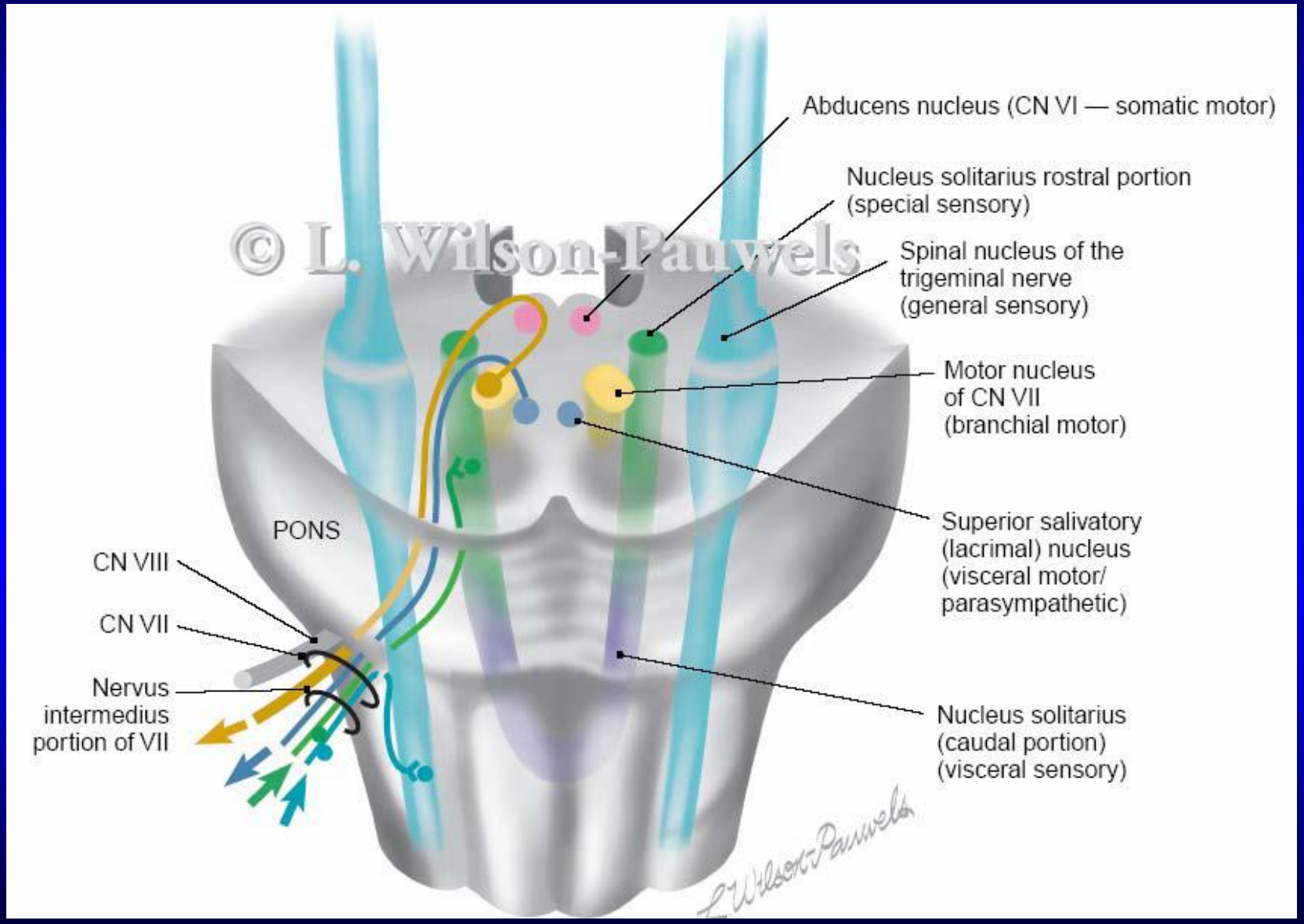
THE FACIAL NERVE

FACIAL NERVE FIBERS

- Motor
  - to the stapedius and facial muscles
- Secreto-motor
  - to the submandibular, sublingual salivary glands and to the lacrimal glands
- Taste
  - from the anterior two thirds of tongue and palate
- Sensory
  - from the external auditory meatus


Abducens nucleus (CN VI — somatic motor)
Nucleus solitarius rostral portion (special sensory)
Spinal nucleus of the trigeminal nerve (general sensory)
Motor nucleus of CN VII (branchial motor)
Superior salivatory (lacrimal) nucleus (visceral motor/ parasympathetic)
Nucleus solitarius (caudal portion) (visceral sensory)
PONS
CN VIII
CN VII
Nervus intermedius portion of VII
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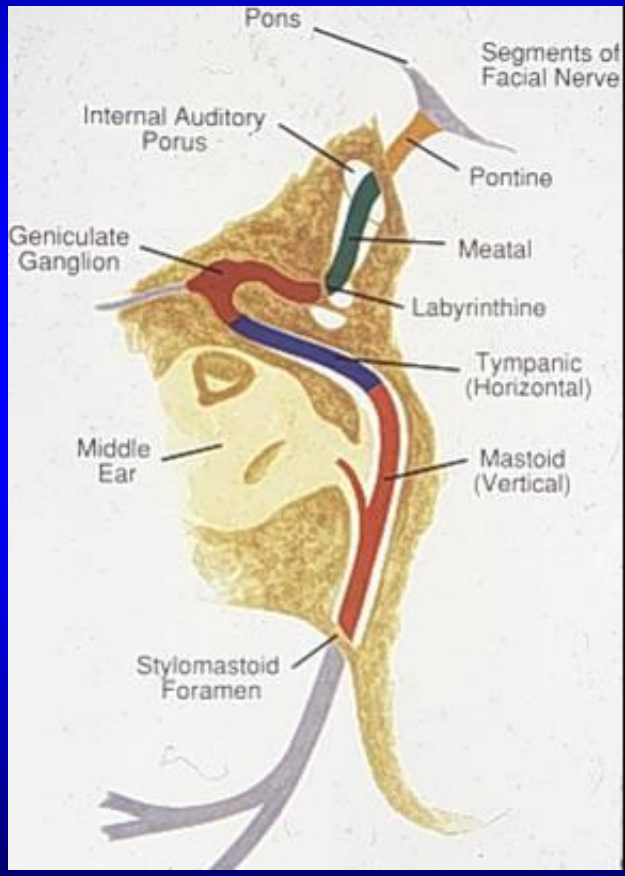
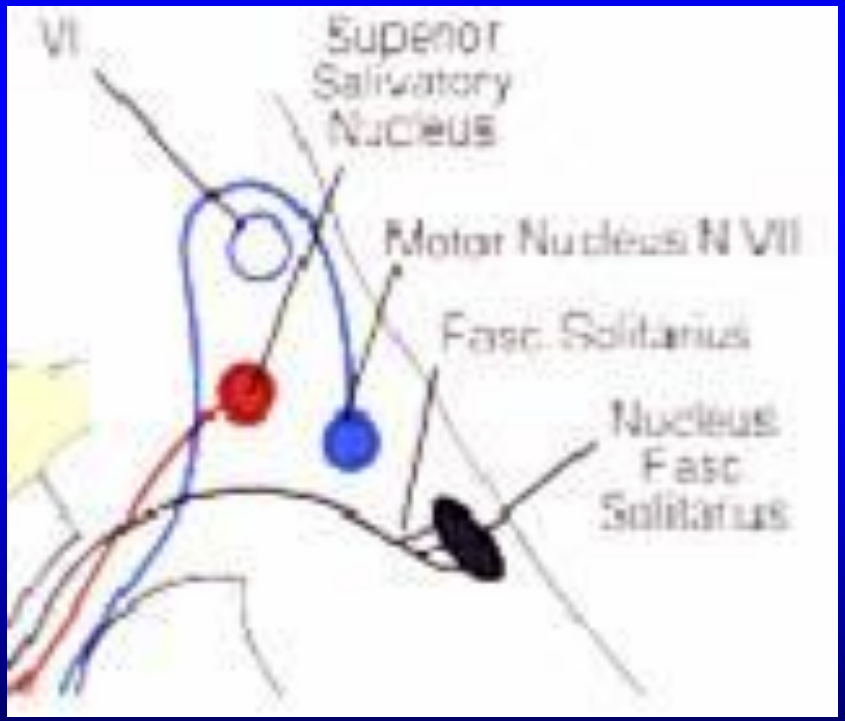
ANATOMICAL DIVISIONS

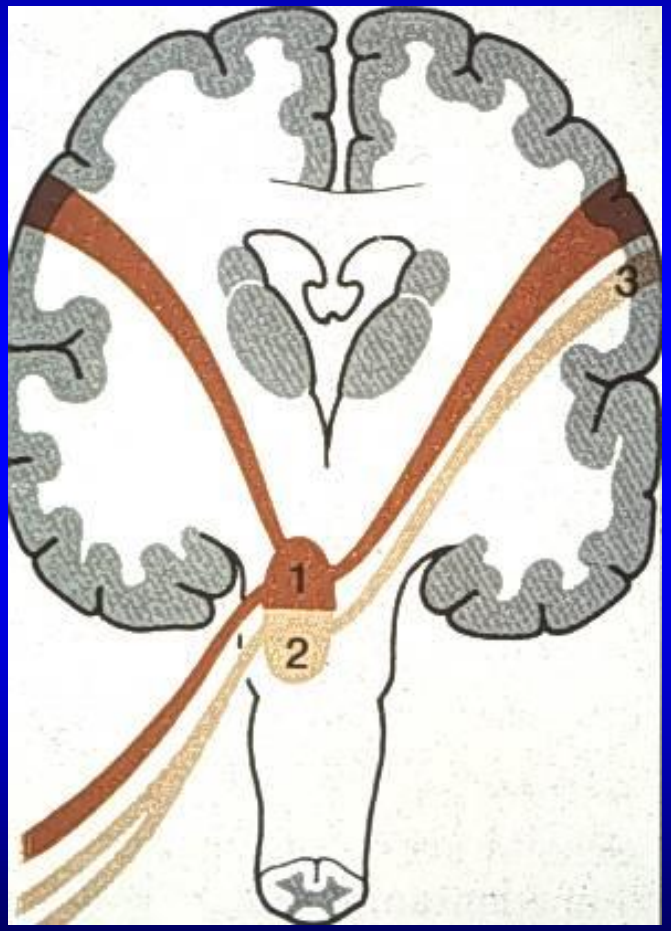
- Intracranial
  - Nuclei & cerebellopontine
- Cranial (intratemporal)
  - Meatal
  - Fallopian canal (labyrinthine, tympanic and mastoid)
- Extracranial (extratemporal)



THE INTRACRANIAL PART

1. The nucleui





- Upper motor lesions spare the upper facial muscles and affect the contralateral lower face

- Lower motor lesions affect all the ipsilateral facial muscles

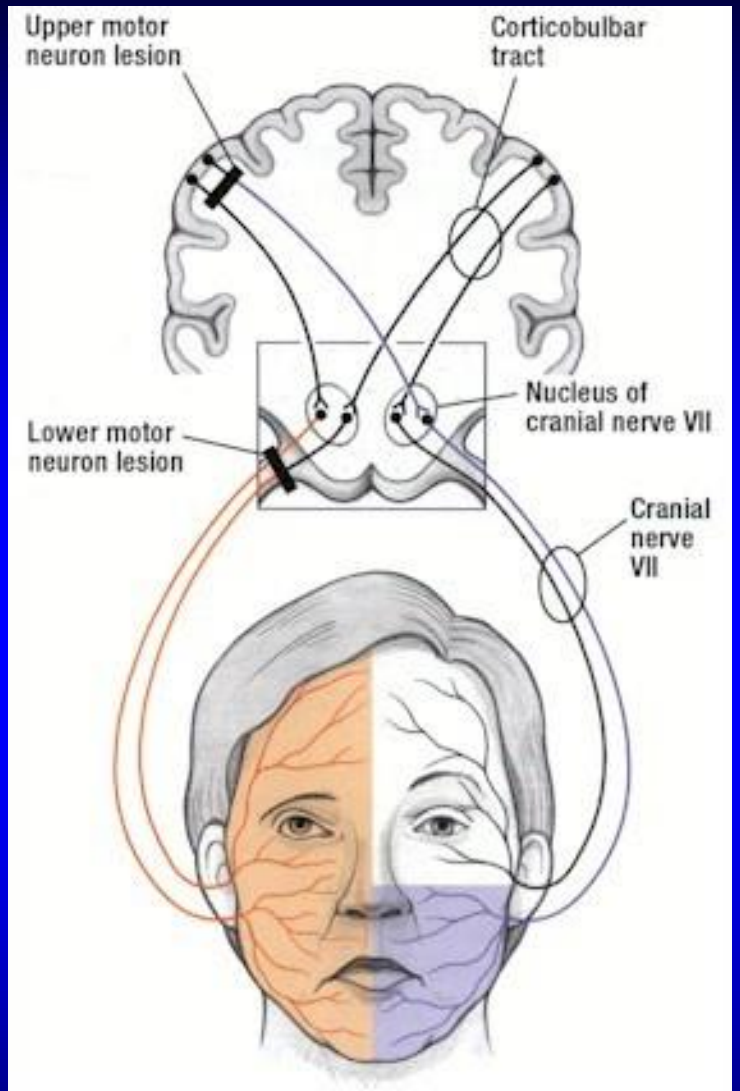
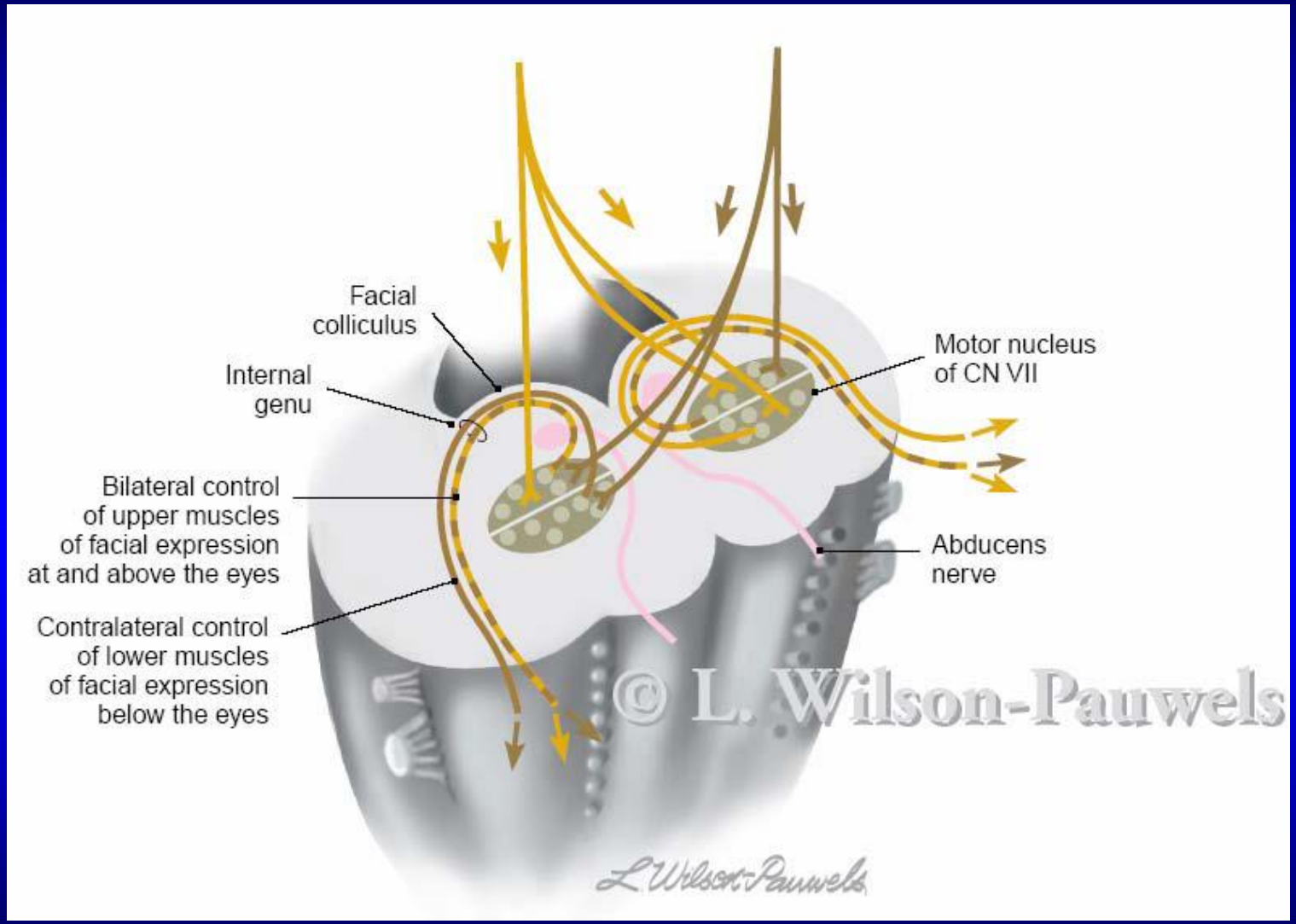
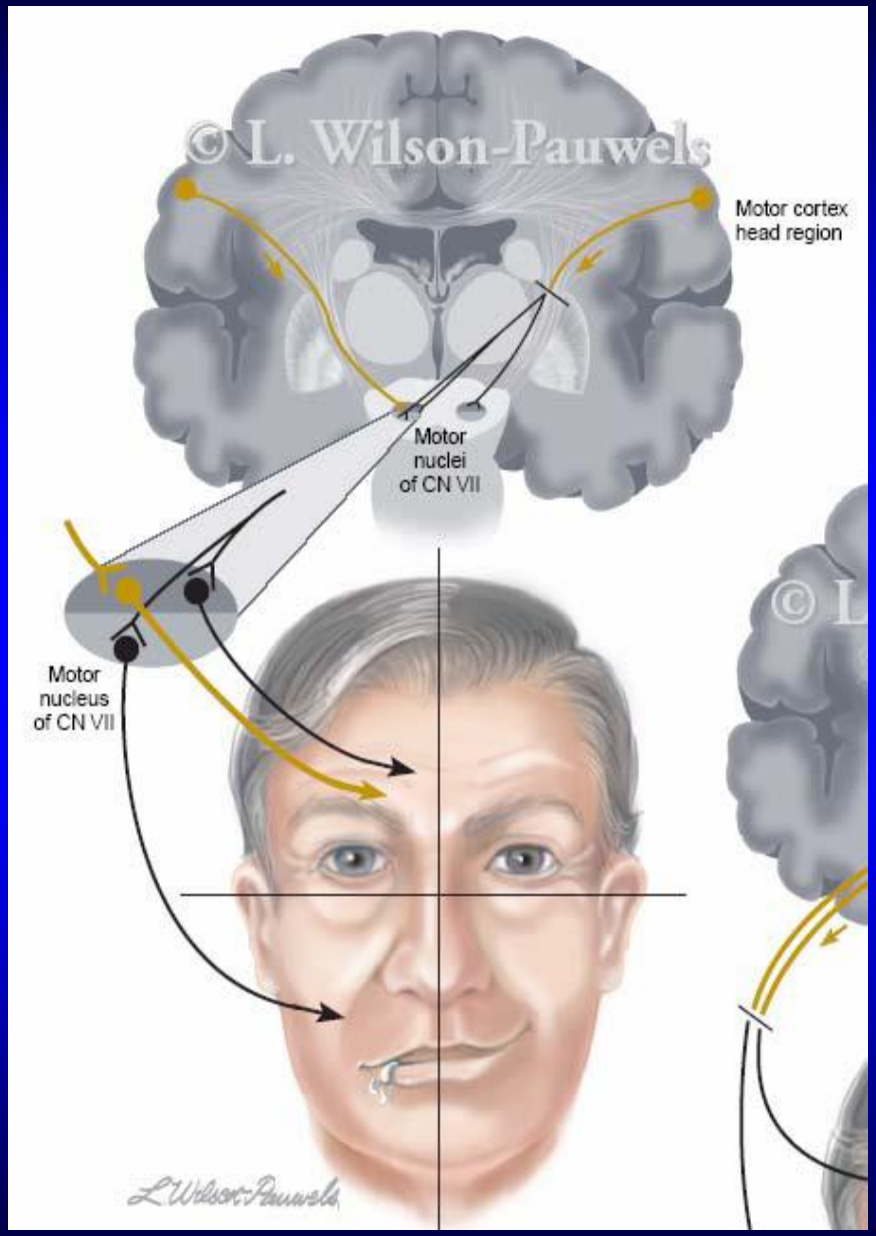
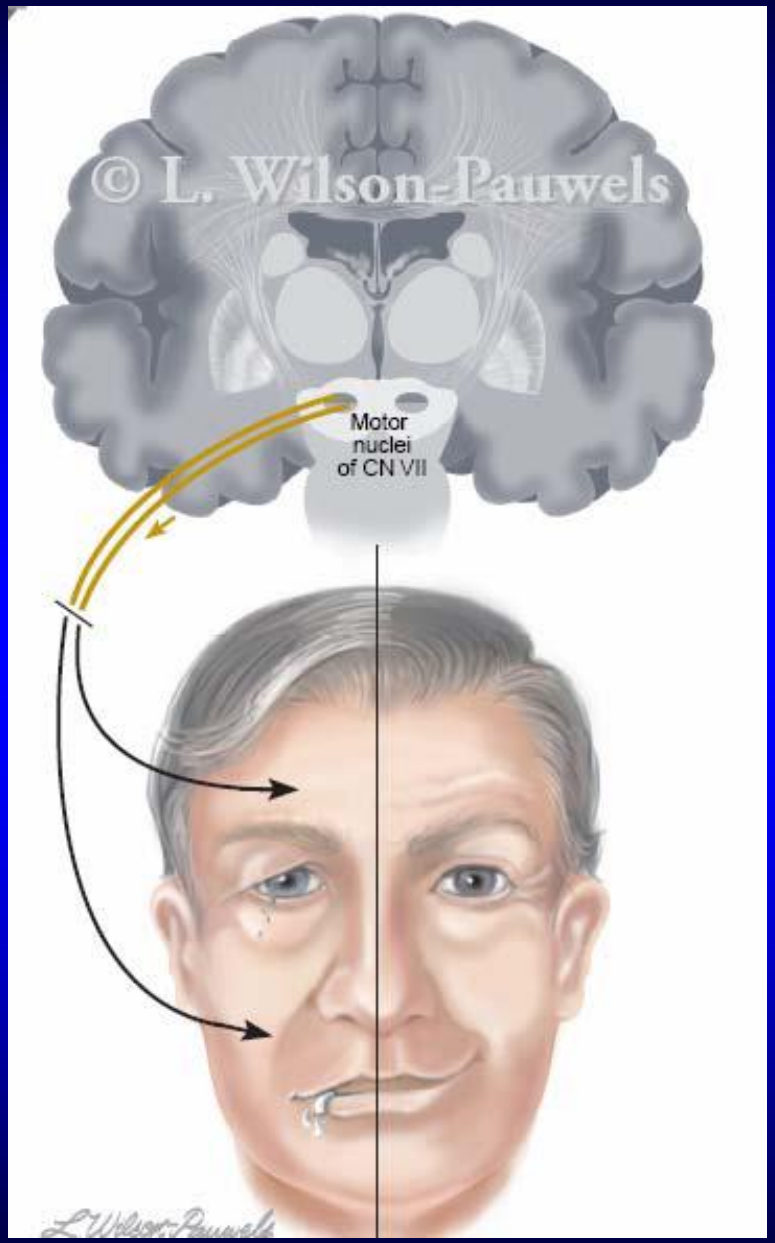


Figure 2a: The color lines show the distribution of facial muscles paralyzed after a supernuclear lesion of the corticobulbar tract and after a lower motor neuron lesion of the facial nerve.


Facial colliculus
Internal genu
Bilateral control of upper muscles of facial expression at and above the eyes
Contralateral control of lower muscles of facial expression below the eyes
Motor nucleus of CN VII
Abducens nerve
© L. Wilson-Pauwels
L Wilson-Pauwels


© L. Wilson-Pauwels
Motor cortex head region
Motor nuclei of CN VII
Motor nucleus of CN VII
L Wilson-Pauwels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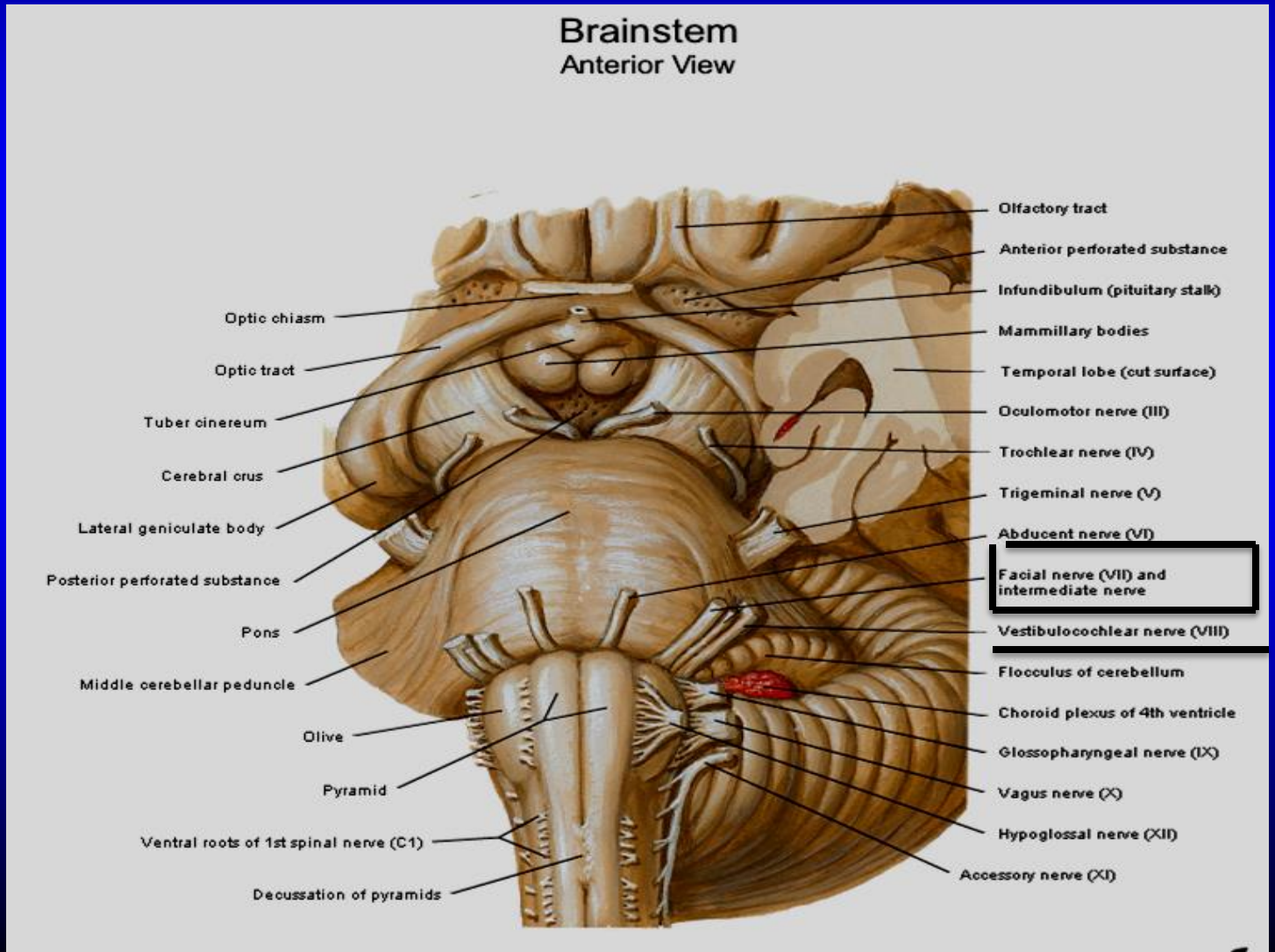
Motor nuclei of CN VII

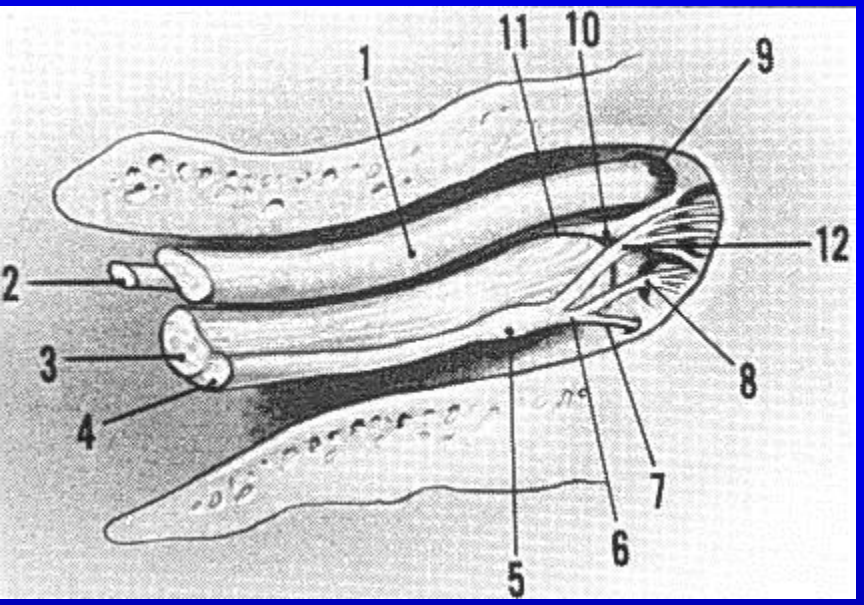
UPPER MOTOR

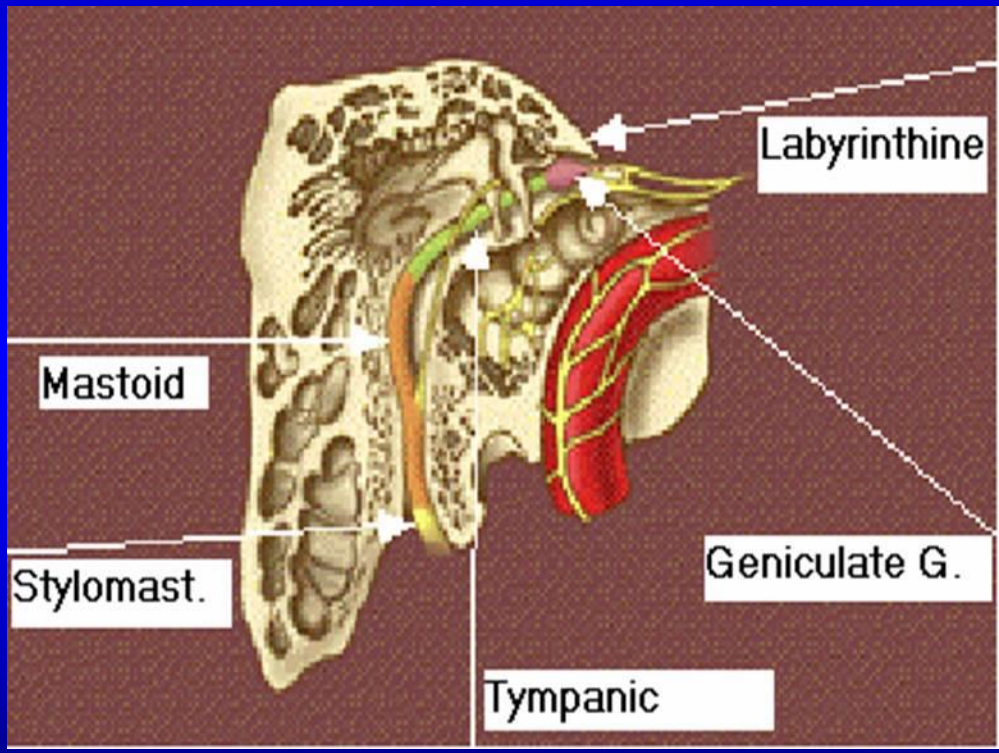
LOWER MOTOR

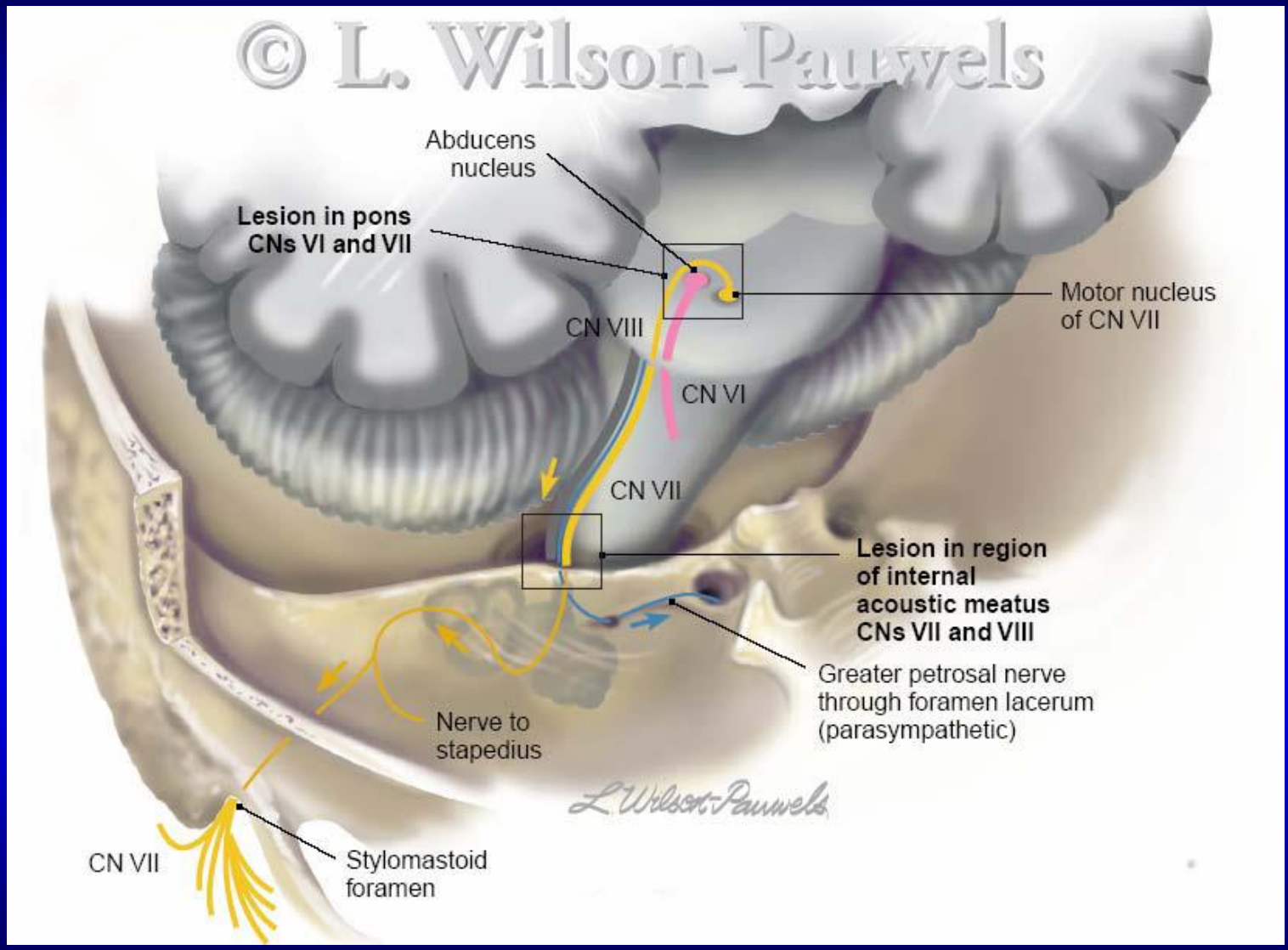
Intracranial part (CP angle)

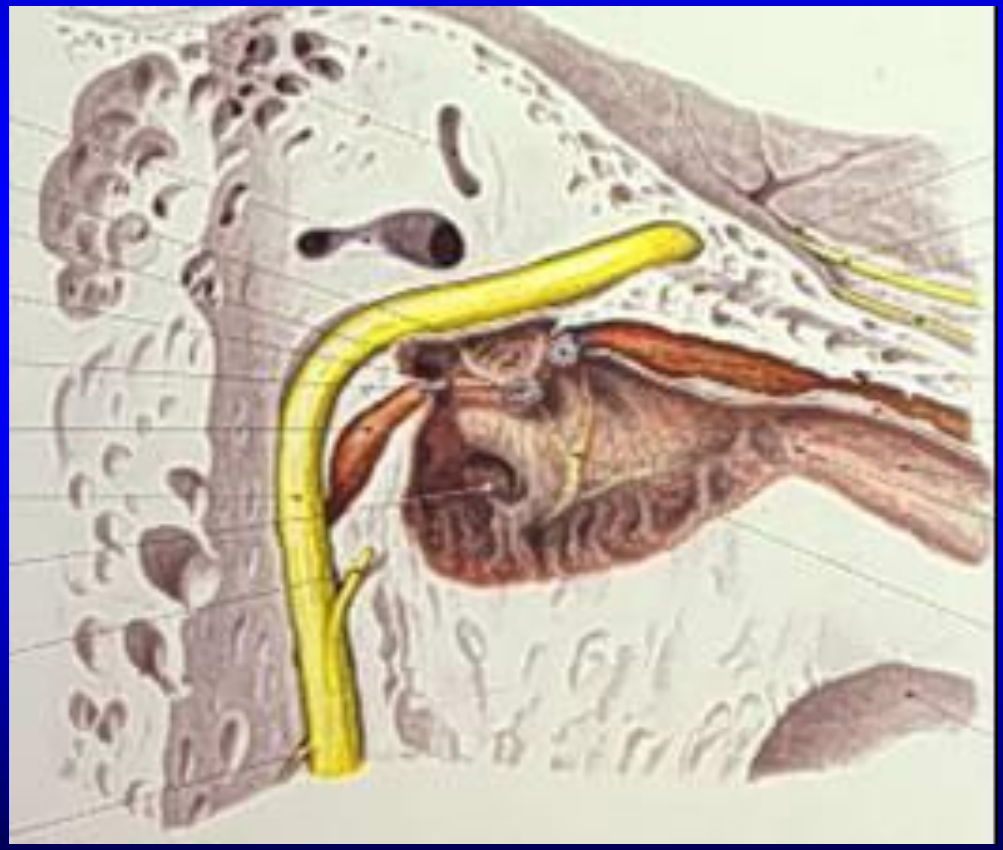


THE INTRA-TEMPORAL (CRANIAL)

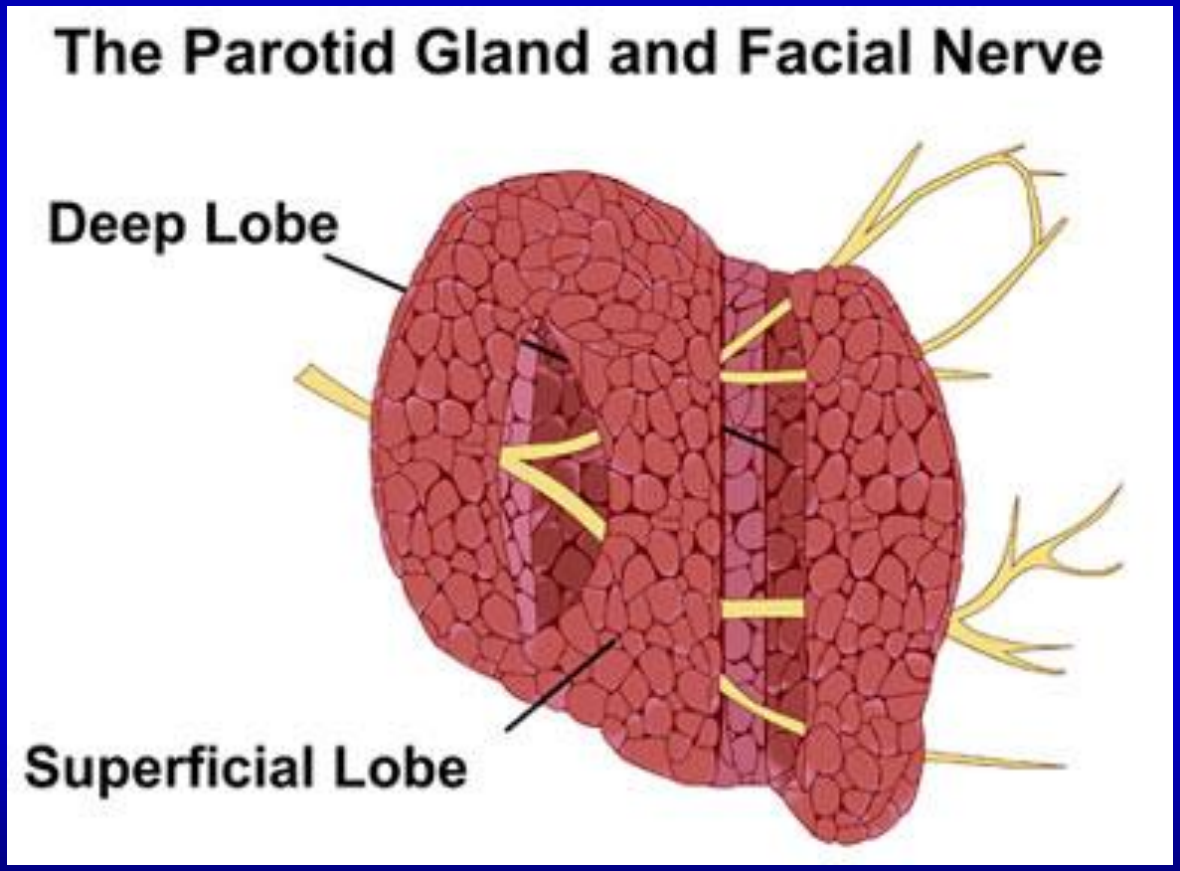





© L. Wilson-Pauwels
Abducens nucleus
Lesion in pons CNs VI and VII
Motor nucleus of CN VII
CN VIII
CN VI
CN VII
Lesion in region of internal acoustic meatus CNs VII and VIII
Greater petrosal nerve through foramen lacerum (parasympathetic)
Nerve to stapedius
L Wilson-Pauwels
CN VII
Stylomastoid foramen

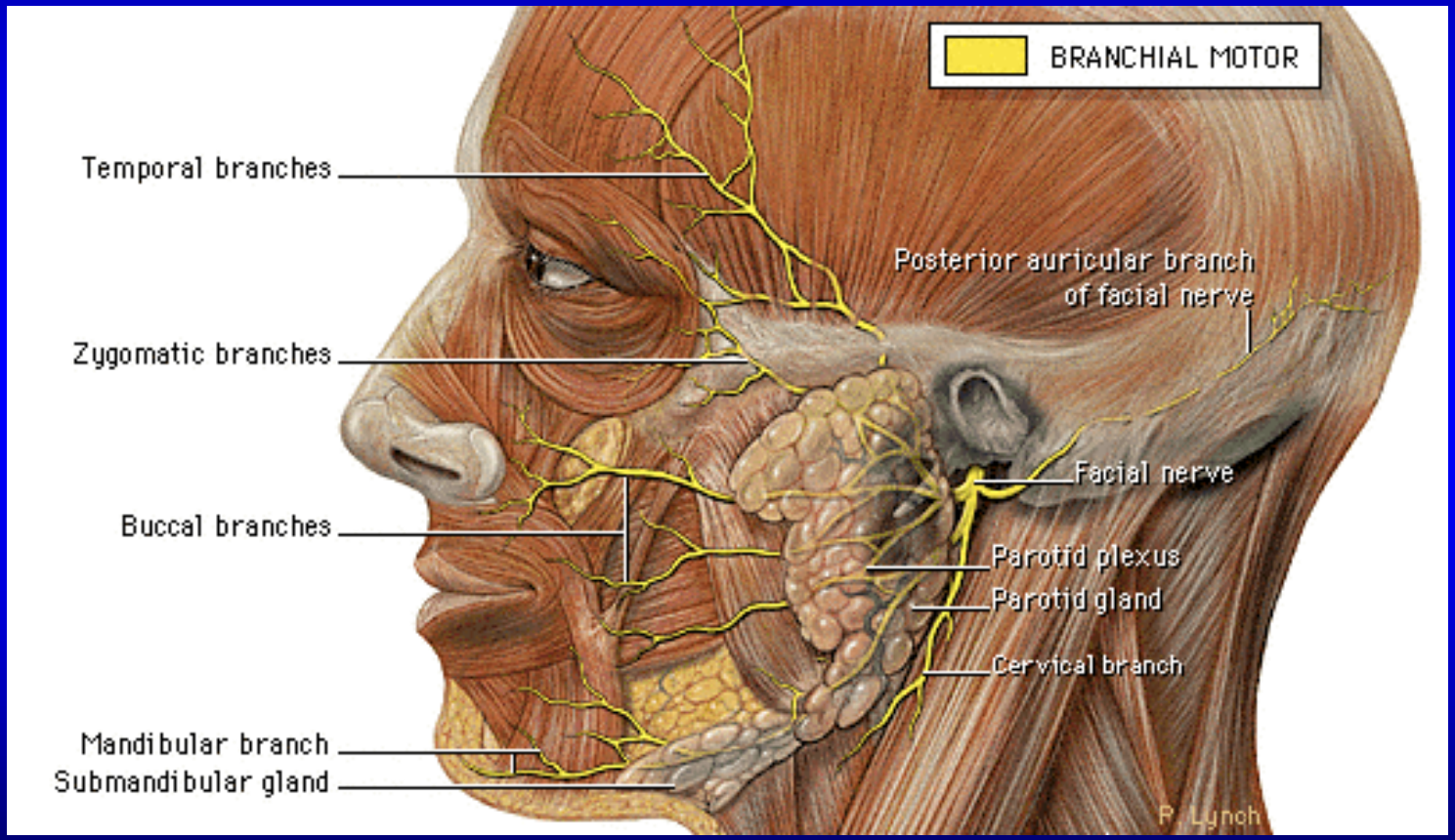


THE EXTRACRANIAL PART



Branches of the Facial Nerve

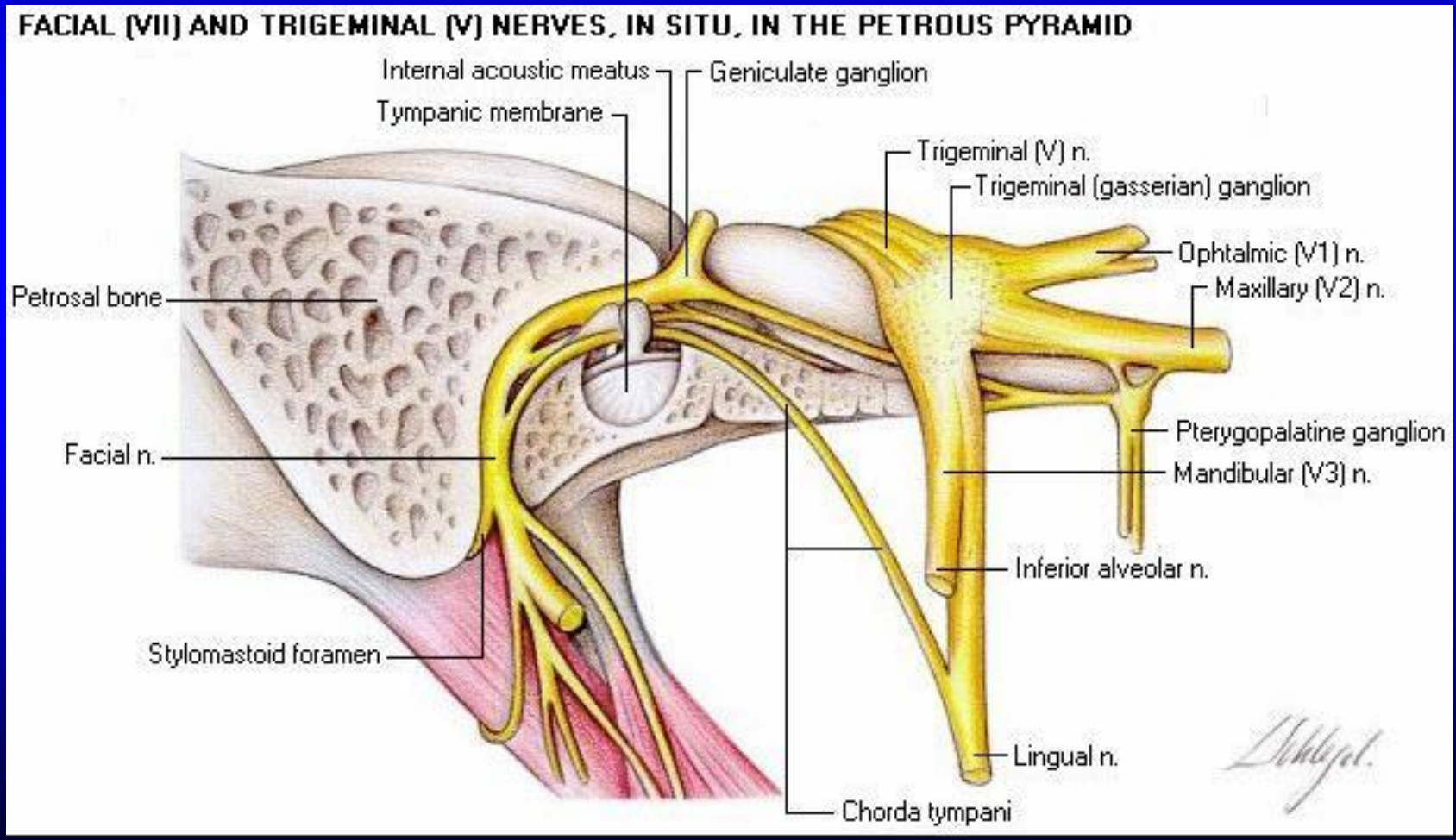
THE EXTRACRANIAL PART

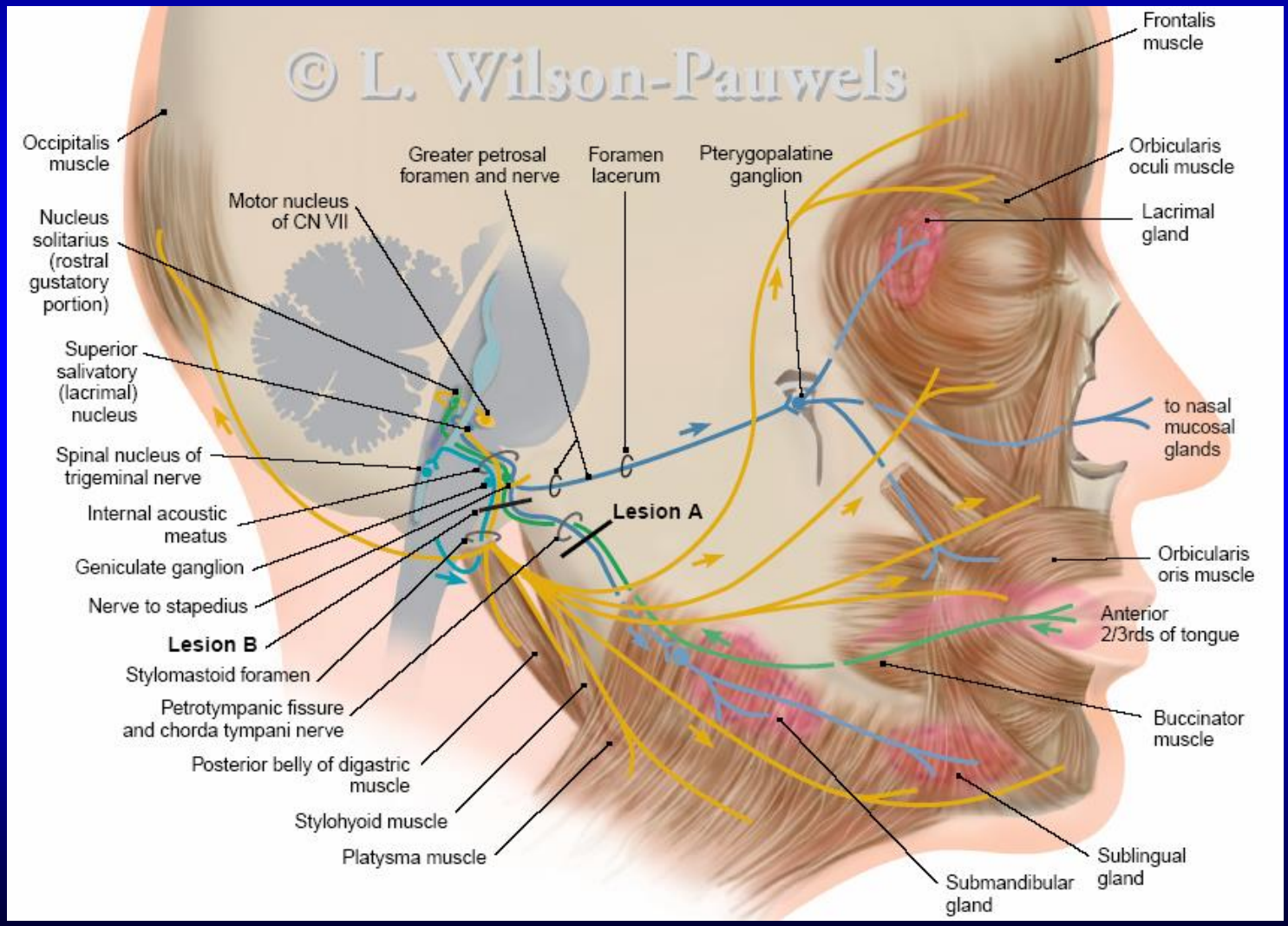


FACIAL NERVE FIBERS

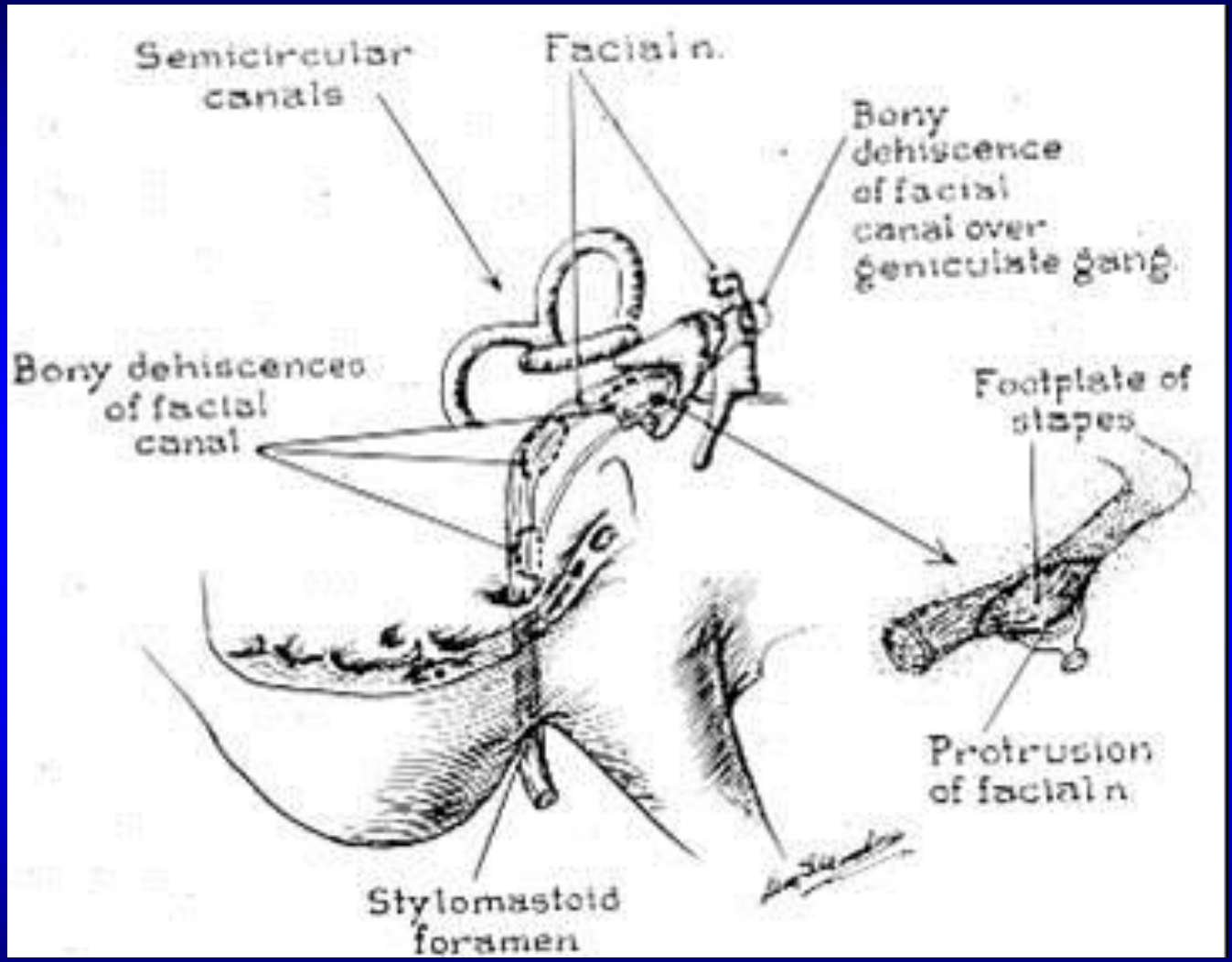
- Motor
  - to stapedius, and facial muscles
- Secreto-motor
  - to the submandibular, sublingual, and lacrimal glands
- Taste
  - from the anterior two thirds of tongue and palate
- Sensory
  - from the external auditory meatus

The secreto-motor and the taste fibres

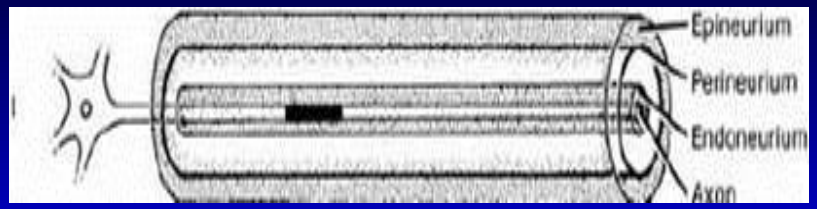


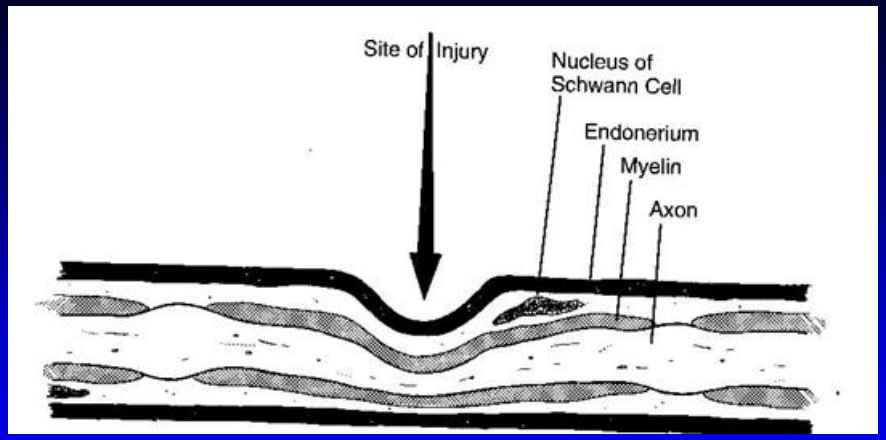

© L. Wilson-Pauwels
Frontalis muscle
Occipitalis muscle
Greater petrosal foramen and nerve
Foramen lacerum
Pterygopalatine ganglion
Orbicularis oculi muscle
Motor nucleus of CN VII
Lacrimal gland
Nucleus solitarius (rostral gustatory portion)
Superior salivatory (lacrimal) nucleus
to nasal mucosal glands
Spinal nucleus of trigeminal nerve
Lesion A
Internal acoustic meatus
Orbicularis oris muscle
Geniculate ganglion
Anterior 2/3rds of tongue
Nerve to stapedius
Lesion B
Buccinator muscle
Stylomastoid foramen
Petrotympanic fissure and chorda tympani nerve
Posterior belly of digastric muscle
Stylohyoid muscle
Platysma muscle
Sublingual gland
Submandibular gland

VARIATIONS AND ANOMALIES

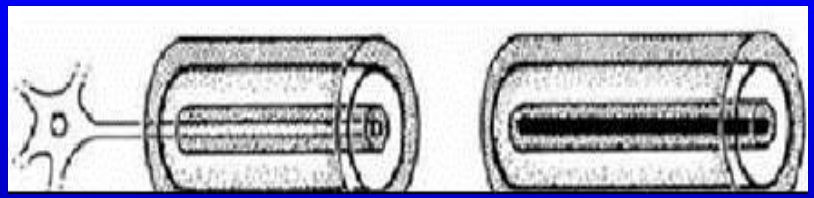

Semicircular canals
Facial n.
Bony dehiscence of facial canal over geniculate gang.
Bony dehiscences of facial canal
Footplate of stapes
Protrusion of facial n.
Stylomastoid foramen

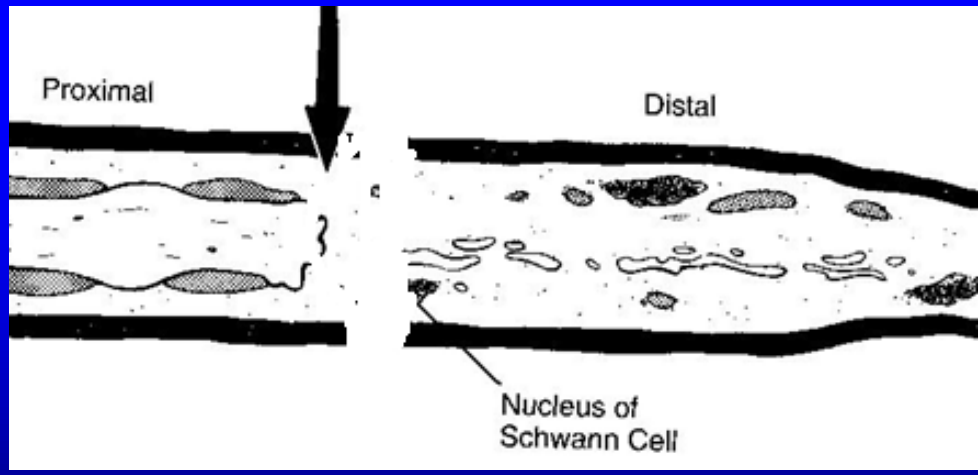
PATHOPHYSIOLOGY OF FACIAL NERVE INJURY





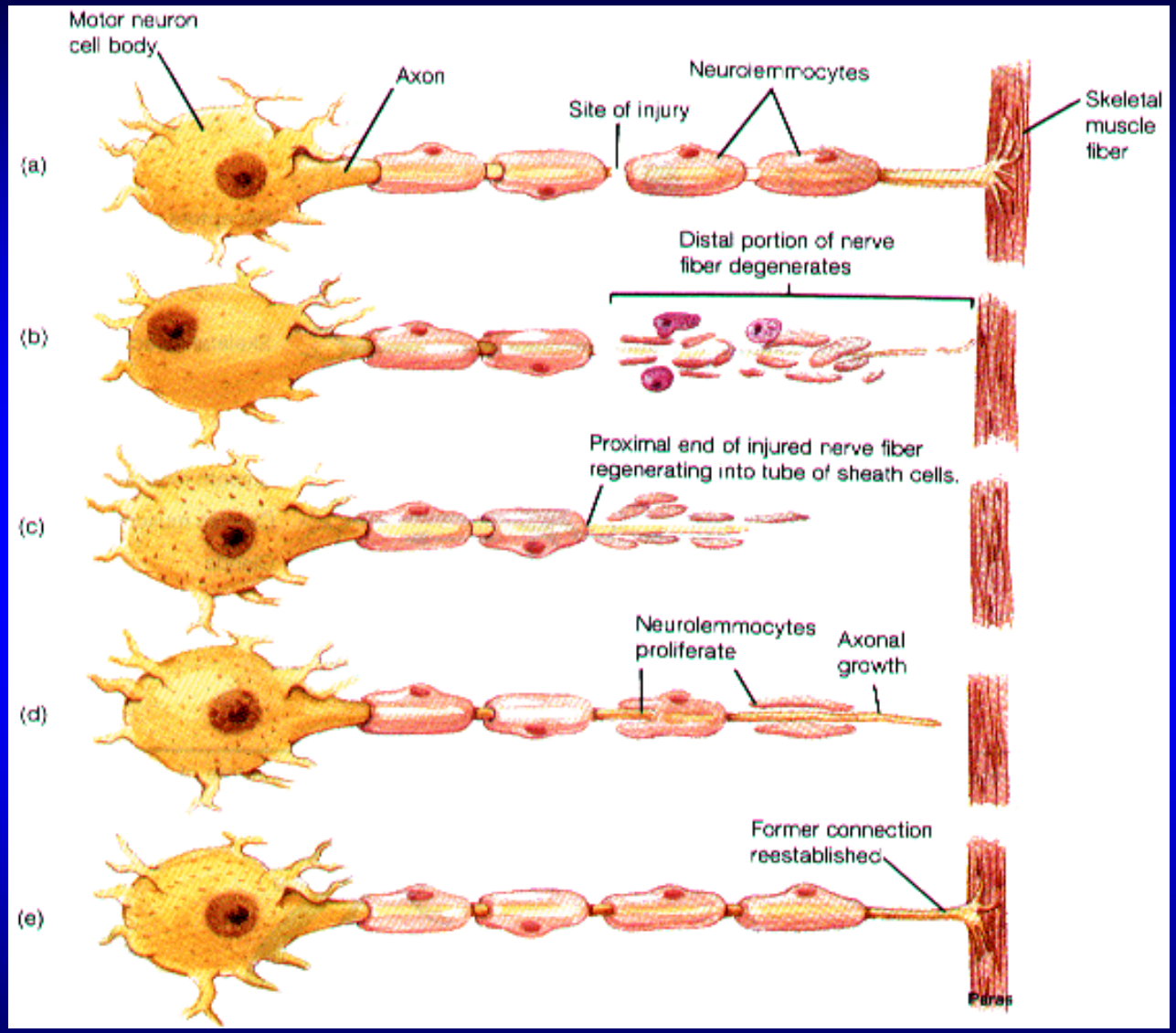
Neuropraxia (Conduction block)





Neurotmeses (Degeneration)

REGENERATION


Motor neuron cell body
Axon
Site of injury
Neurolemmocytes
Skeletal muscle fiber
(a)
Distal portion of nerve fiber degenerates
(b)
Proximal end of injured nerve fiber regenerating into tube of sheath cells.
(c)
Neurolemmocytes proliferate
Axonal growth
(d)
Former connection reestablished
(e)

Clinical Application

- ‘Clinically partial paralysis” means that some fibres are in continuity and the rest are neuropraxic and/or degenerated
  - Treatment is conservative
- “Clinically complete paralysis ”may be either due to neuropraxia or due to degeneration or due to a mix of both.
  - Neuropraxia responds to conservative treatment while degeneration usually needs surgical treatment

Electrophysiological Tests

- Differentiate between conduction block (neuropraxia) and degeneration of nerve fibers (neurotmeses) in clinically complete facial paralysis

Electrophysiological Tests

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

When NET & ENoG are normal?

- If the stimulated nerve fibres are normal or neuropraxic (not degenerated)

When NET & ENoG are abnormal?

- If the nerve fibres have degenerated

When the nerve fibres become degenerated?

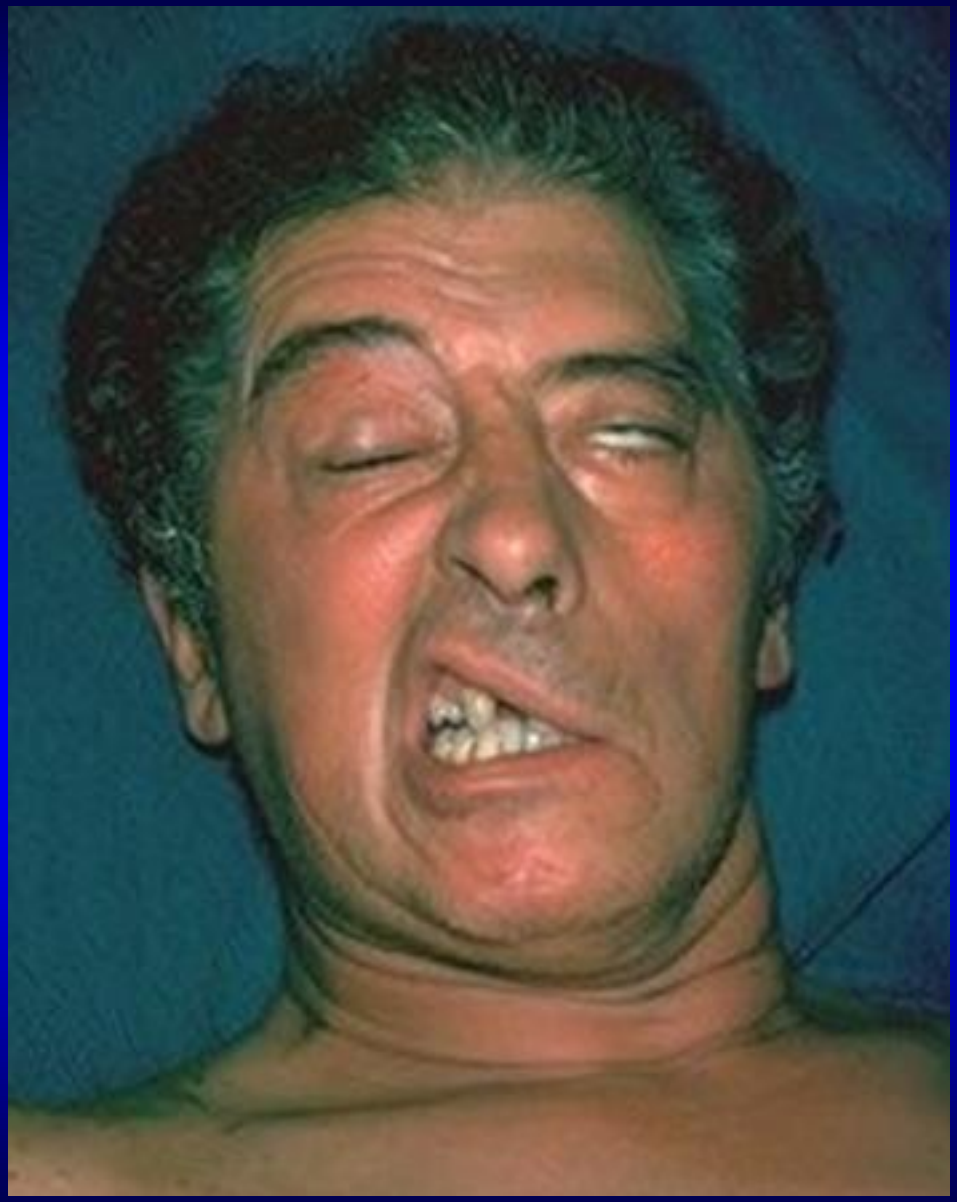
- 48 – 72 hours post injuiry

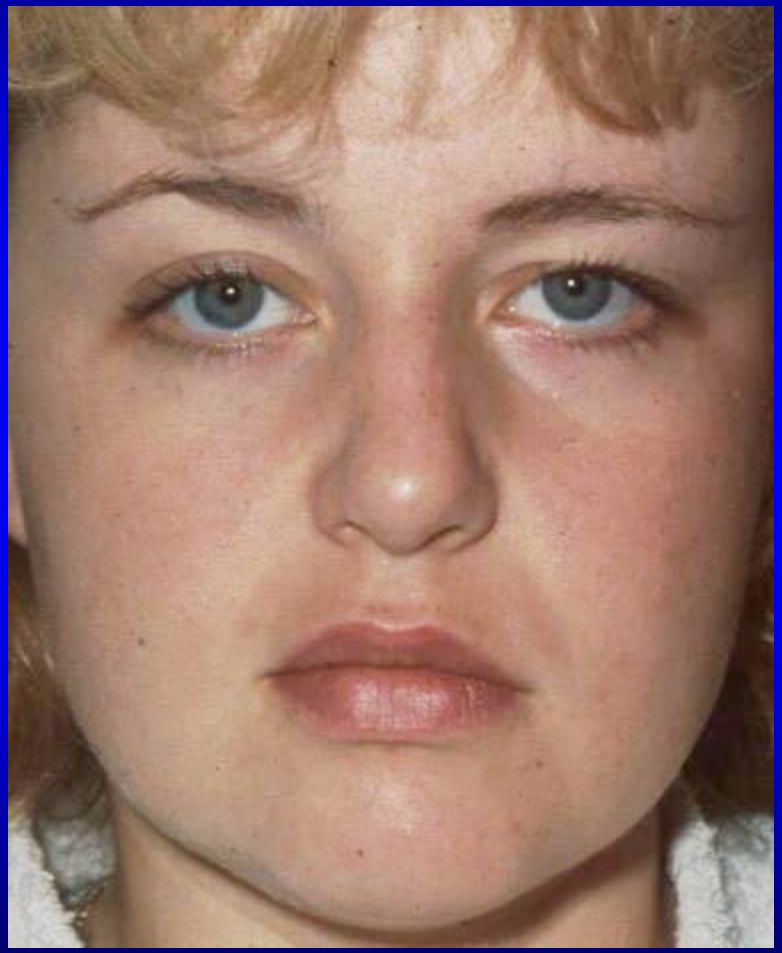
NET & ENoG

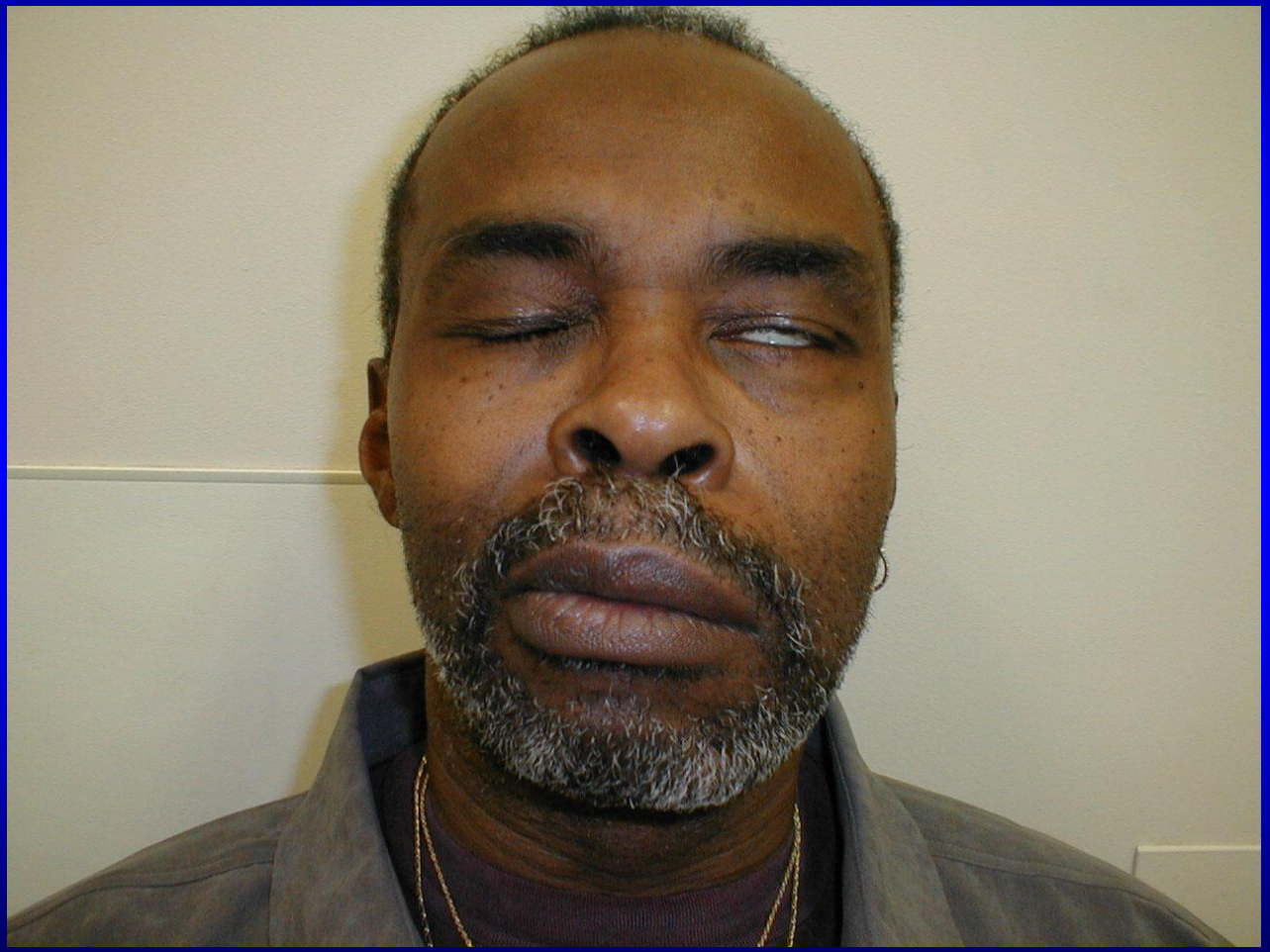
- Not useful in the first 3 days after onset of complete paralysis (the findings always lag several days behind the biologic events themselves)
- Most applicable in the evaluation of acute paralysis (while the nerve is in the degenerative phase)

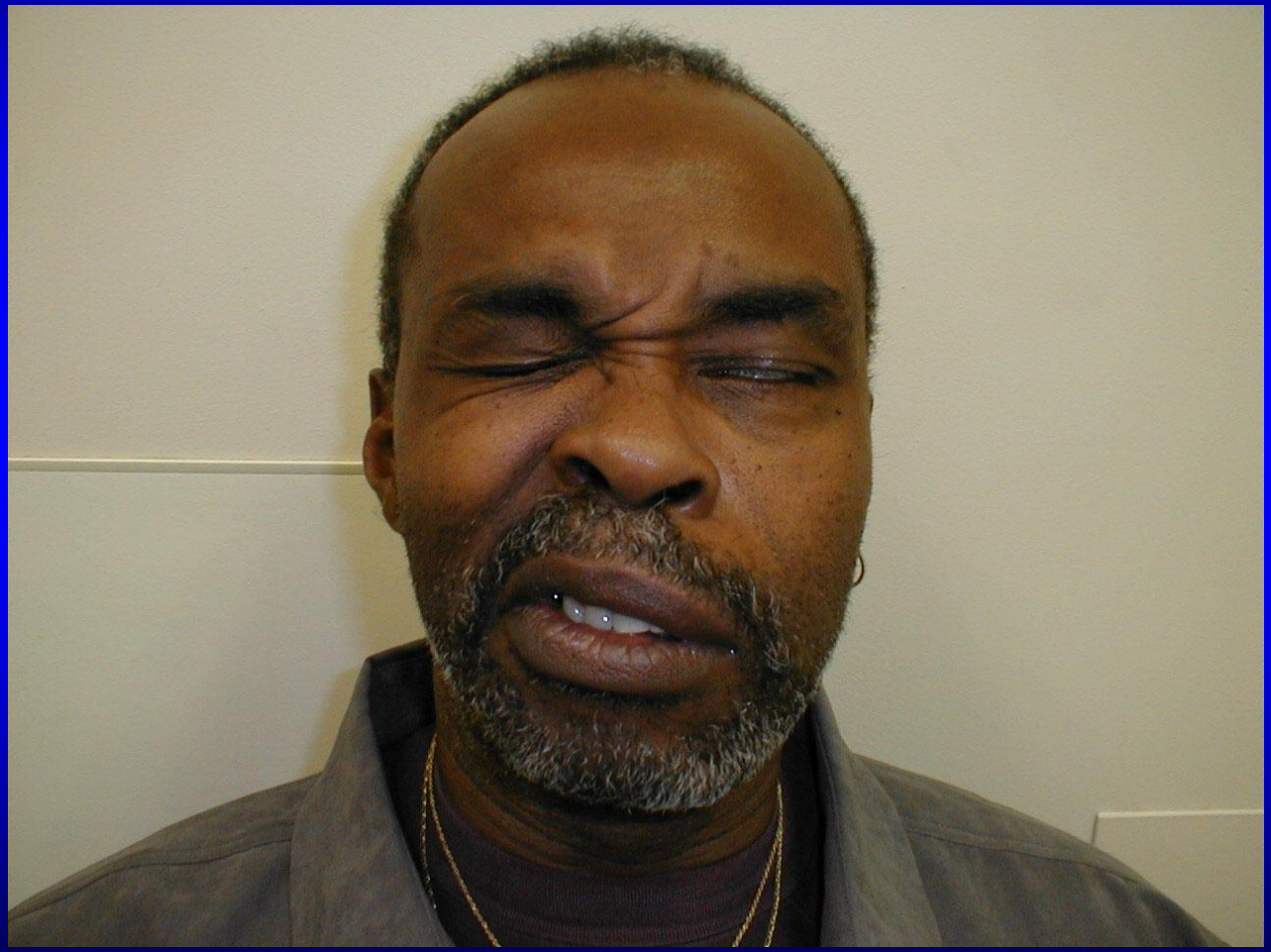
CLINICAL MANIFESTATIONS

- Paralysis of facial muscles
  - Asymmetry of the face









CLINICAL MANIFESTATIONS

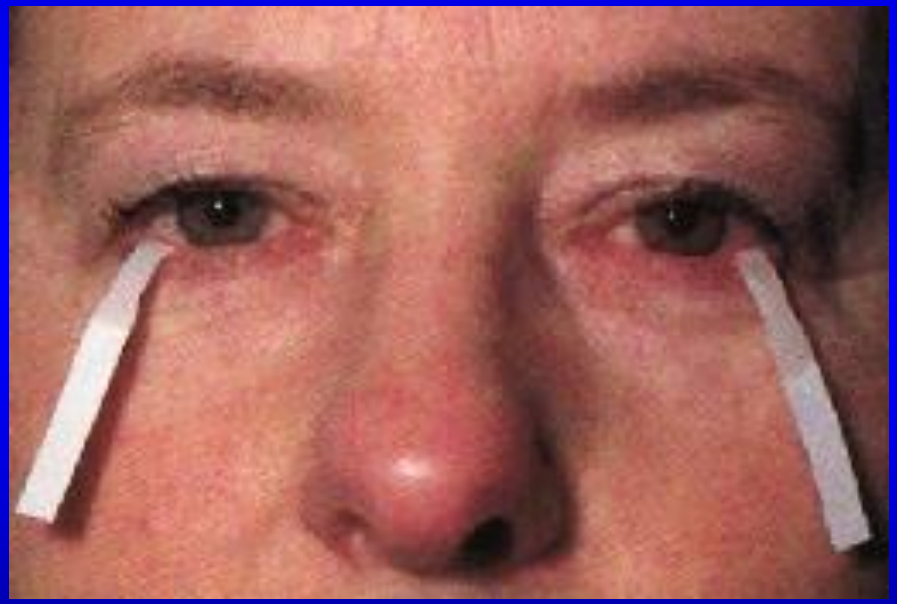
- Paralysis of facial muscles
  - Asymmetry of the face
  - Inability to close the eye
  - Accumulation of food in the cheek
- Phonophobia
- Dryness of the eyes
- Loss of taste

TOPOGNOSTIC TESTS

- Indicated in some cases to locate the site of the injury

TOPOGNOSTIC TESTS

- Schirmer's test
  - Test the lacrimation function



TOPOGNOSTIC TESTS

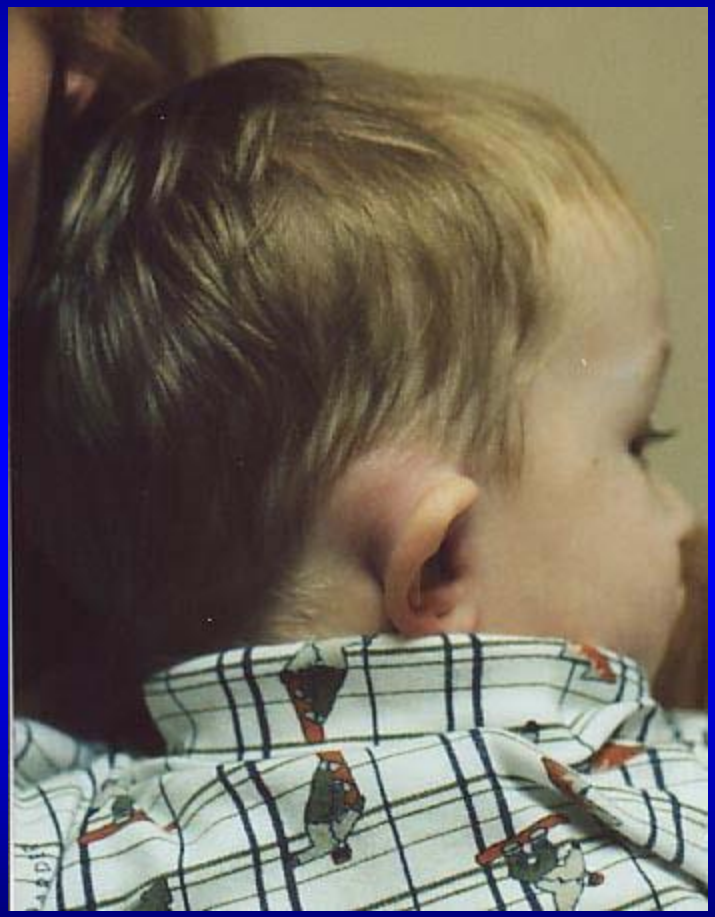
- Schirmer's test
- Stapedial reflex
- Taste sensation

TOPOGNOSTIC TESTS

- Schirmer's test
- Stapedial reflex
- Taste sensation
- Salivary flow

CAUSES OF FACIAL PARALYSIS

- 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

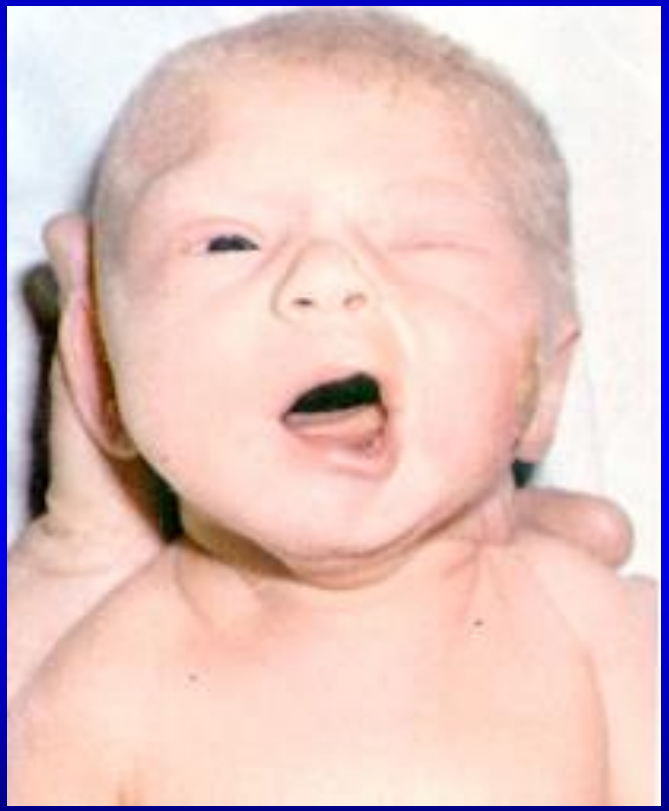


CAUSES OF FACIAL PARALYSIS

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

Congenital Facial Palsy

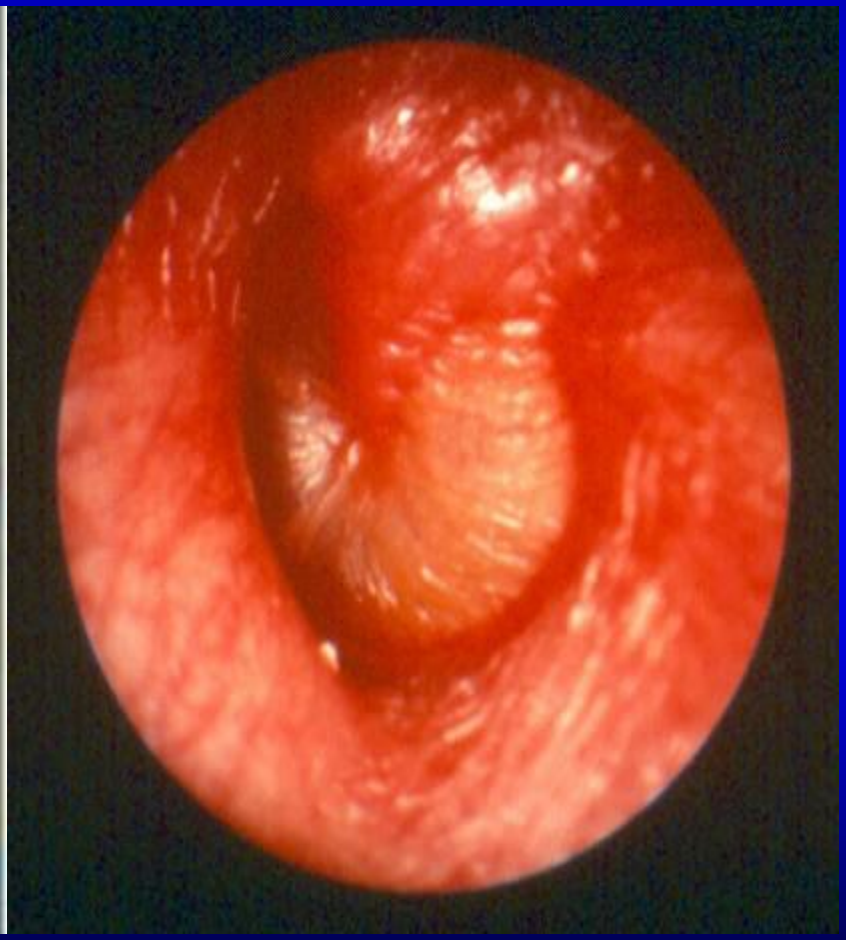
- 80-90% are associated with birth trauma
- 10 -20 % are associated with developmental lesions



INFLAMMATORY CAUSES OF FACIAL PARALYSIS

Facial Paralysis in AOM

- Mostly due to pressure on a dehiscent nerve by inflammatory products
- Usually is partial and sudden in onset
- Treatment is by antibiotics and myringotomy

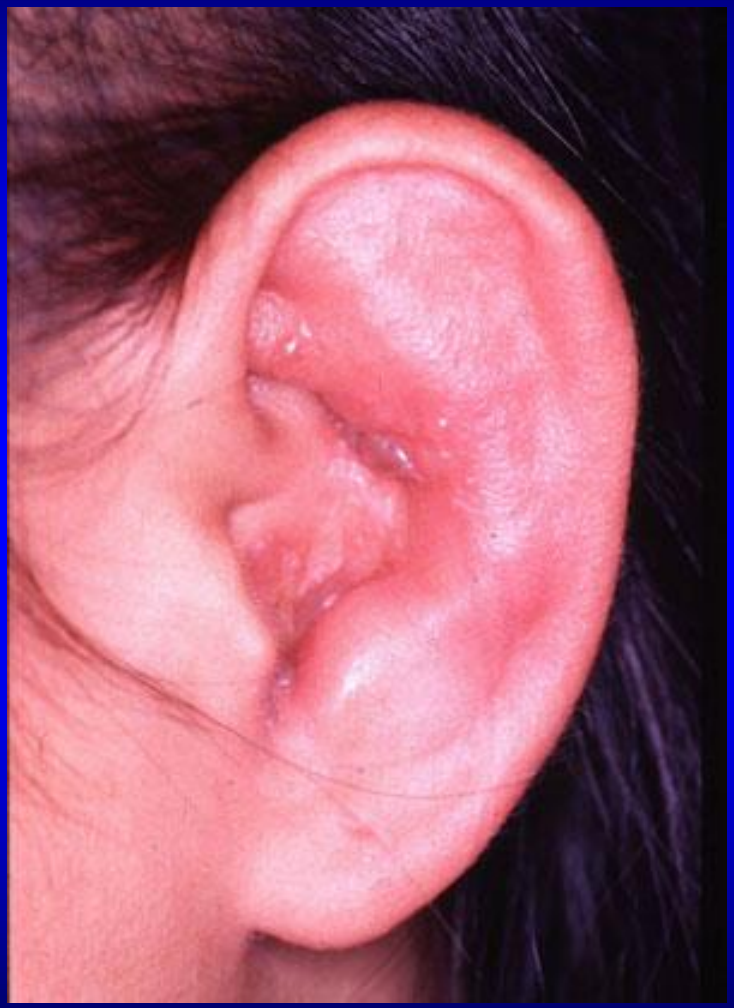


Facial Paralysis in CSOM

- 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"

HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME)

- Herpes zoster affection of cranial nerves VII, VIII, and cervical nerves
- Facial palsy, pain, skin rash, SNHL and vertigo



HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME)

- Herpes zoster affection of cranial nerves VII, VIII, and other nerves
- Facial palsy, pain, skin rash, SNHL and vertigo
- Vertigo improves due to compensation
- SNHL is usually irreversible
- Facial nerve recovers in about 60%
- Treatment by: Acyclovir, steroid and symptomatic

Traumatic Facial Injury

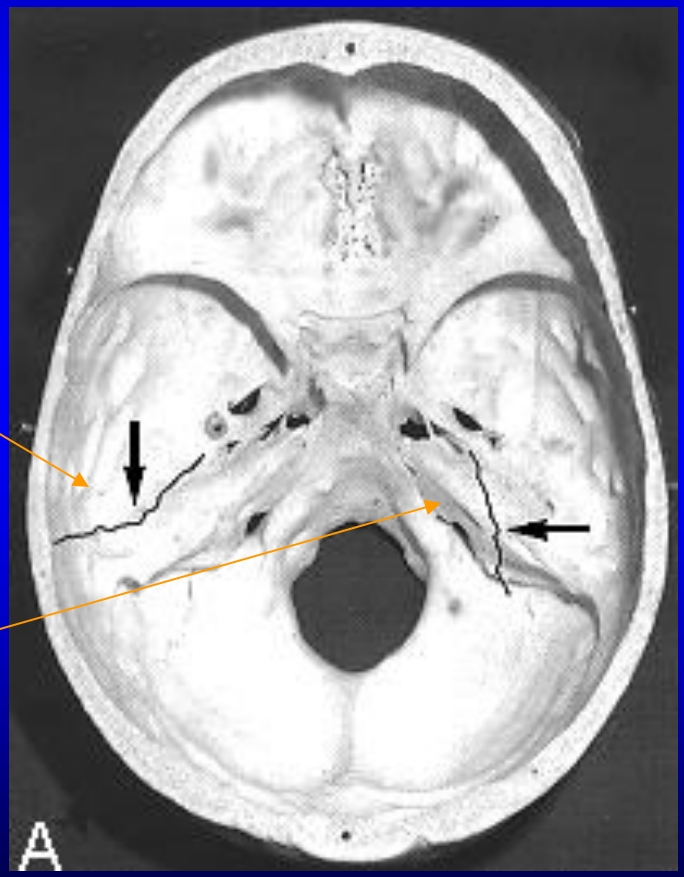
- Birth trauma
- Iatrogenic
- Temporal bone fracture

Iatrogenic Facial Nerve Injury

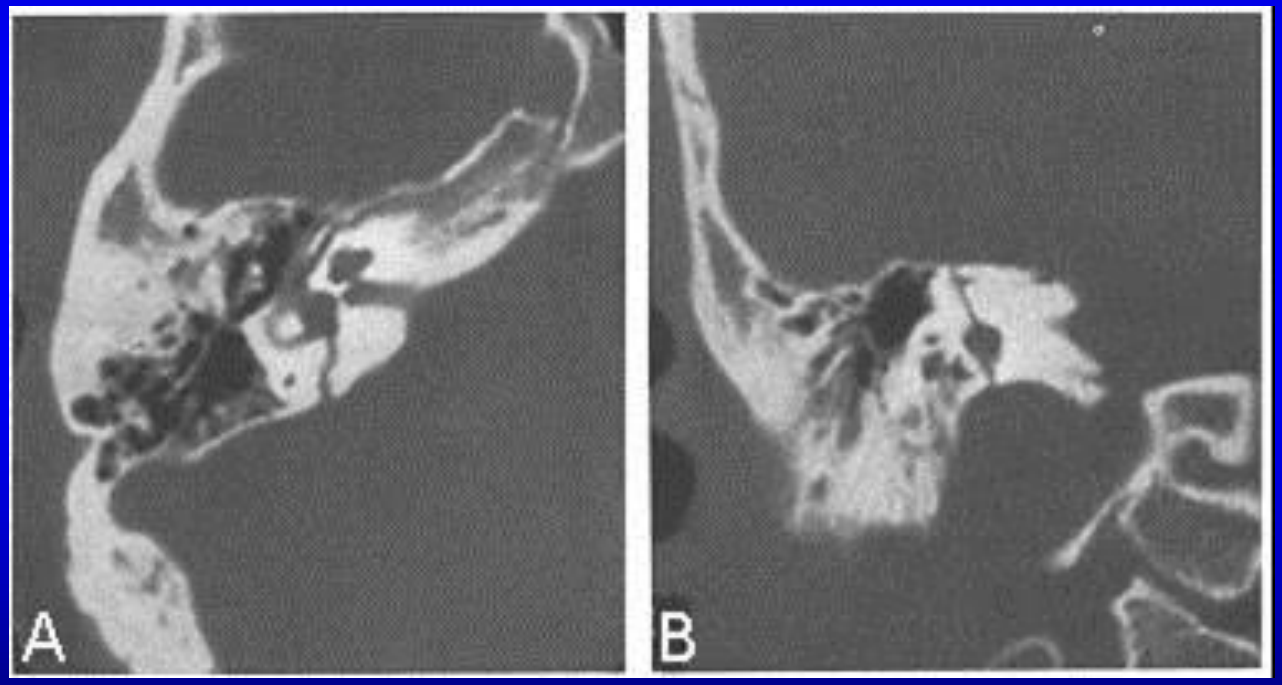
- Operations at the CP angle, ear and the parotid glands

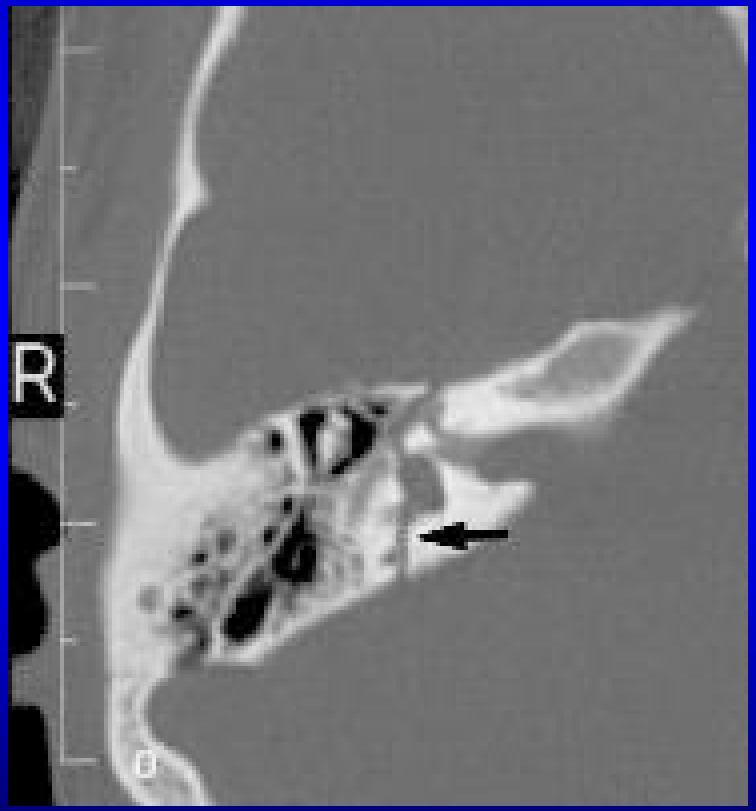
Temporal Bone Fracture

- Longitudinal
- Transverse



Transverse Fracture

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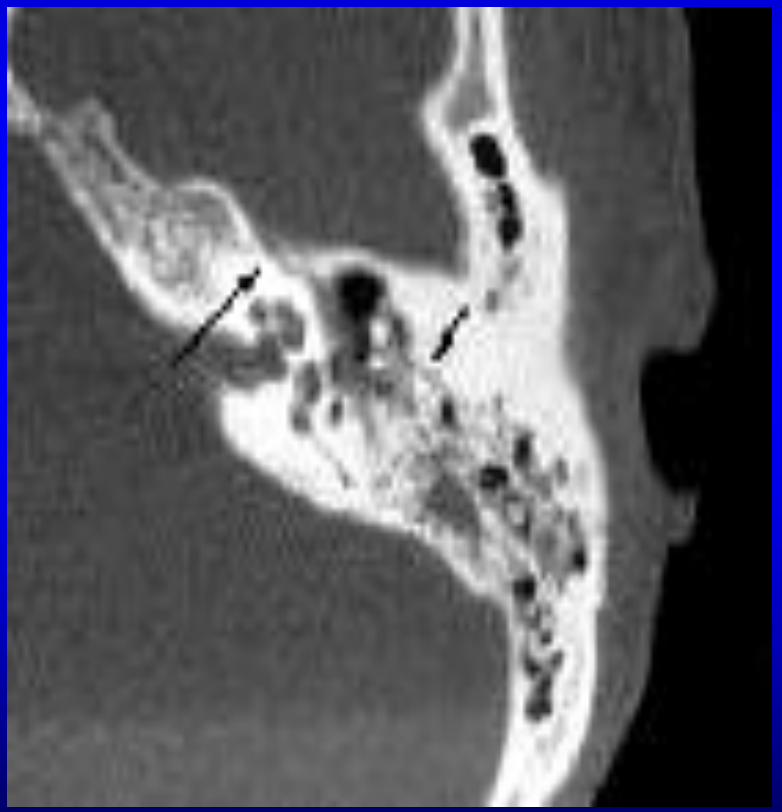


Pathology

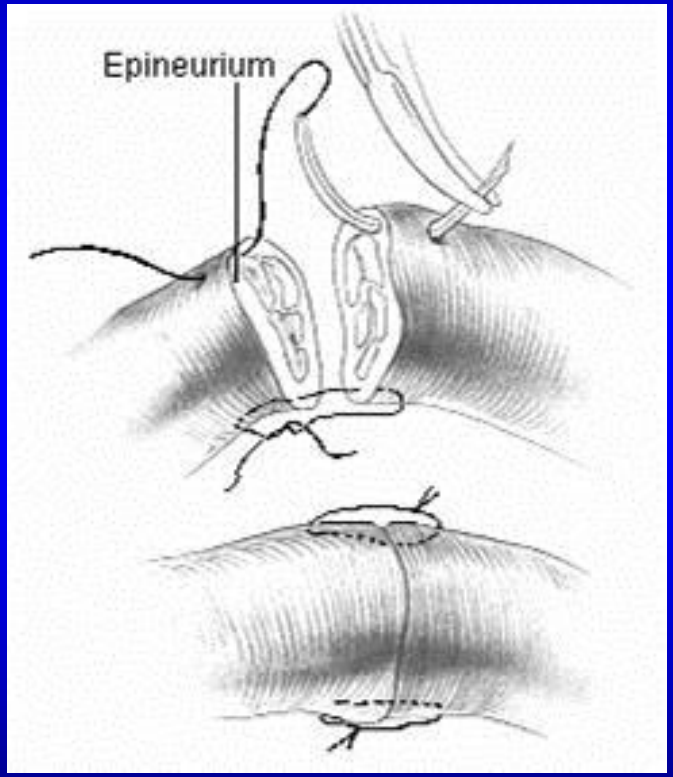
- Edema
- Transection of the nerve

Management of Traumatic Facial Nerve Injury

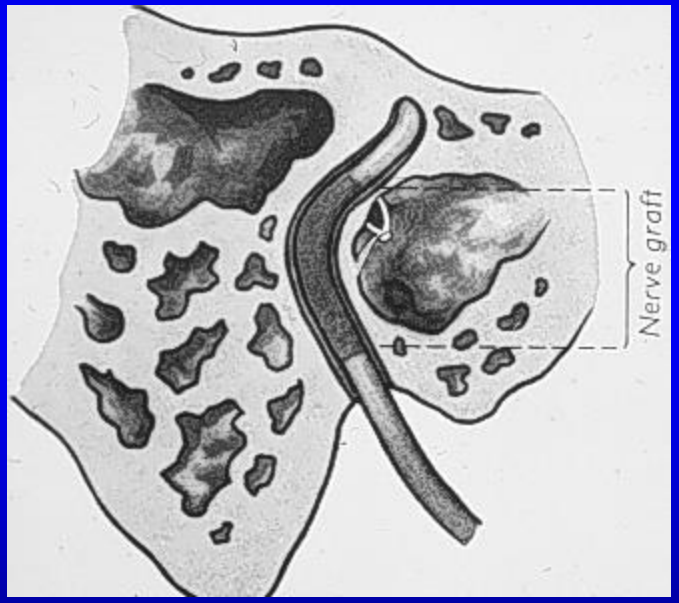
- If it is delayed in onset, it is usually incomplete and is due to edema
  - Conservative
- If of immediate onset, it is usually complete and due to transection of the nerve
  - Surgical repair

SURGICAL REPAIR

DIRECT ANASTOMOSIS



NERVE GRAFT



NERVE TRANSFER (ANASTOMOSIS)

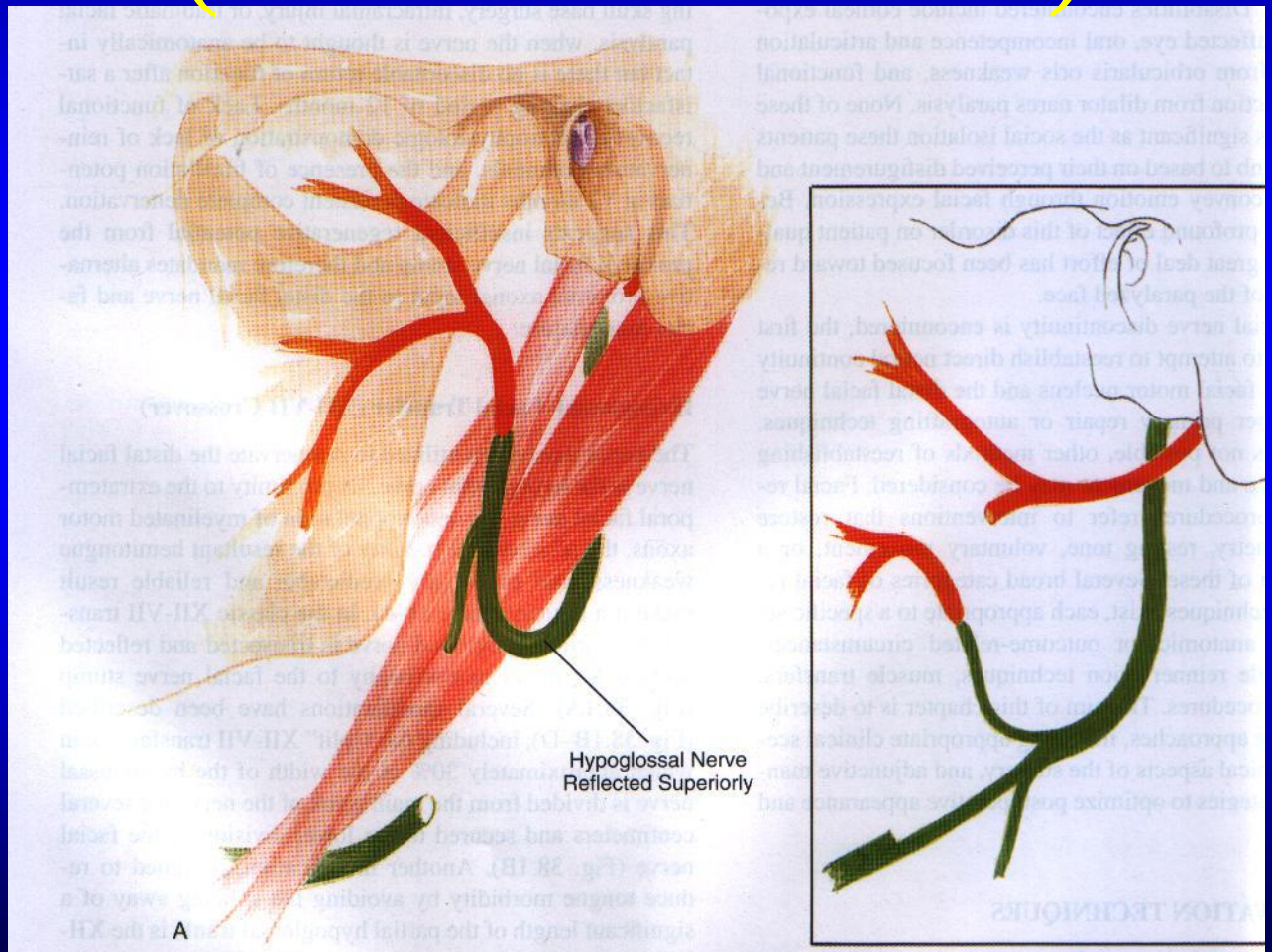
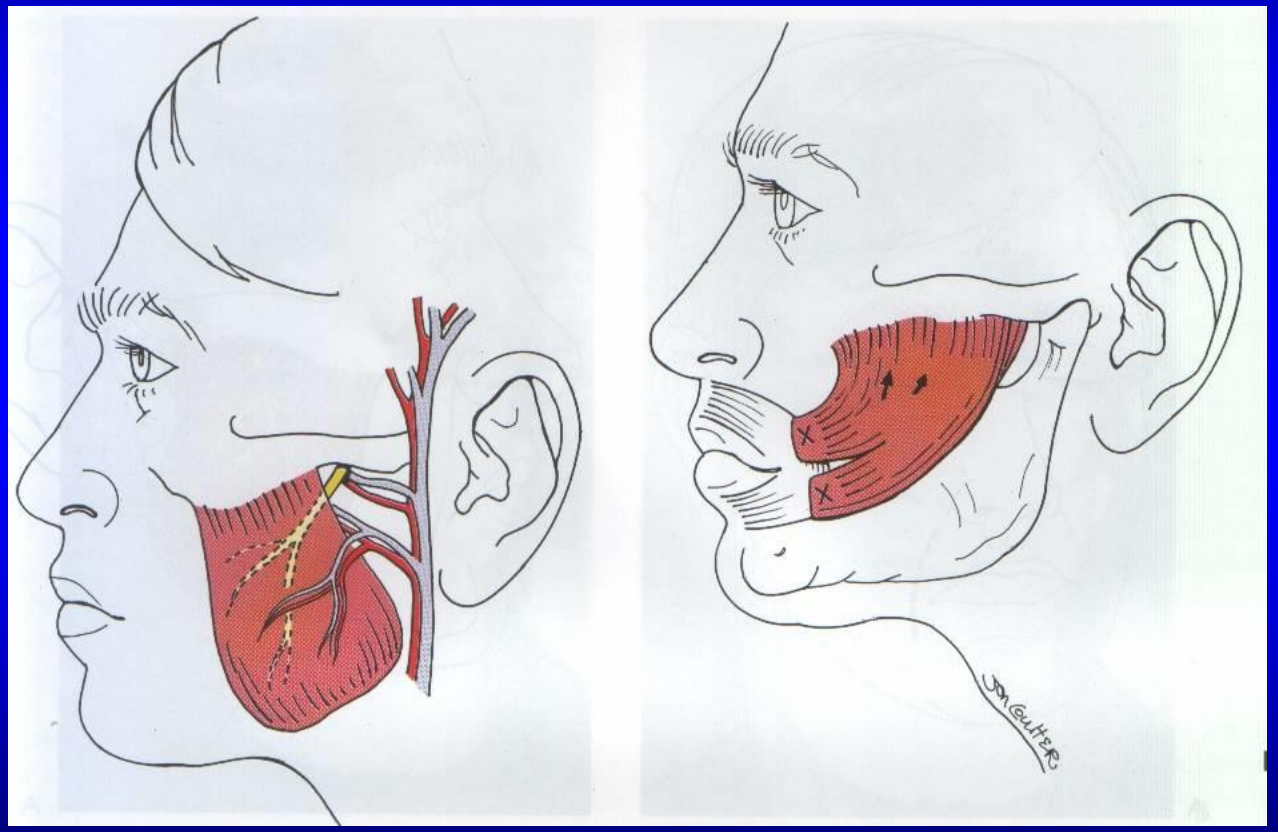
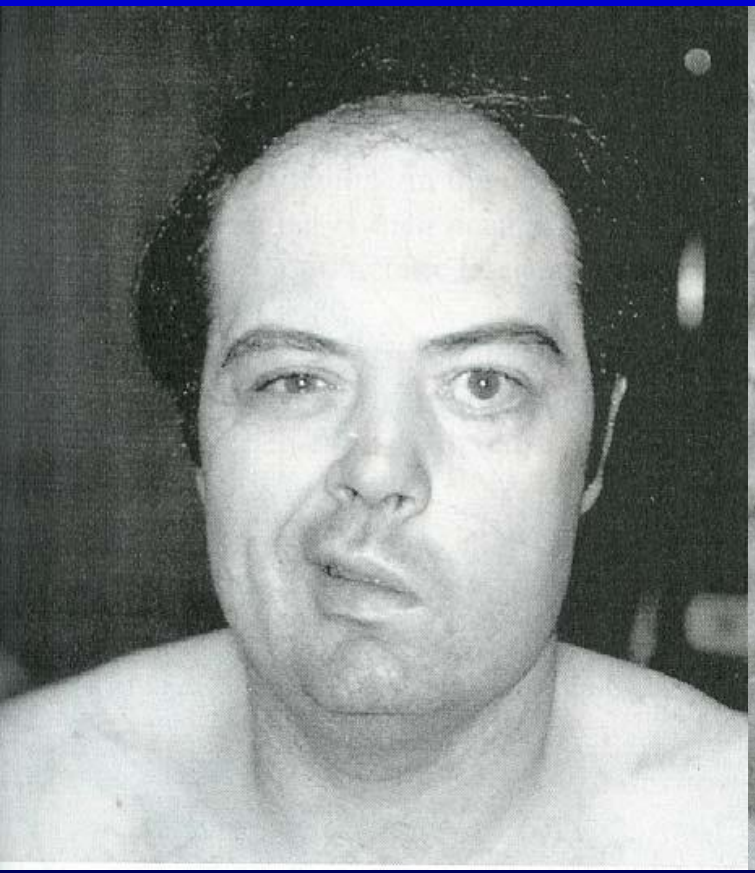
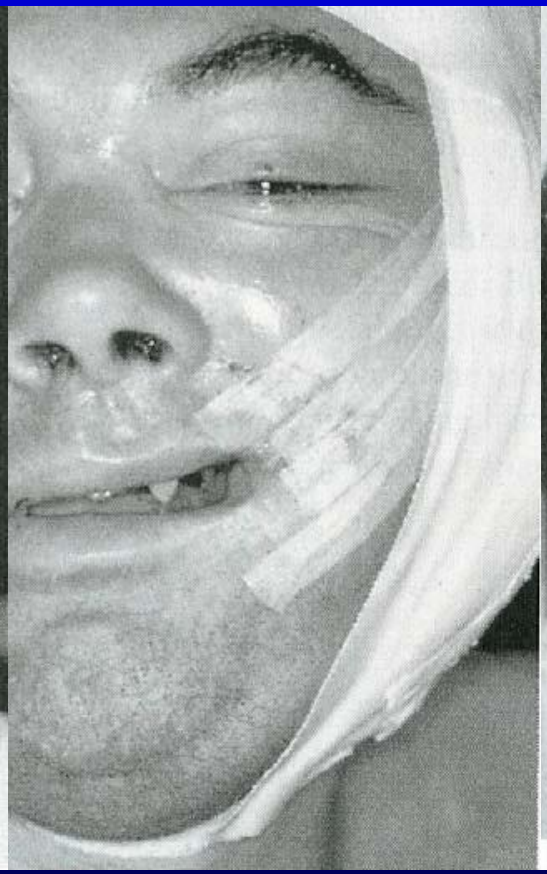
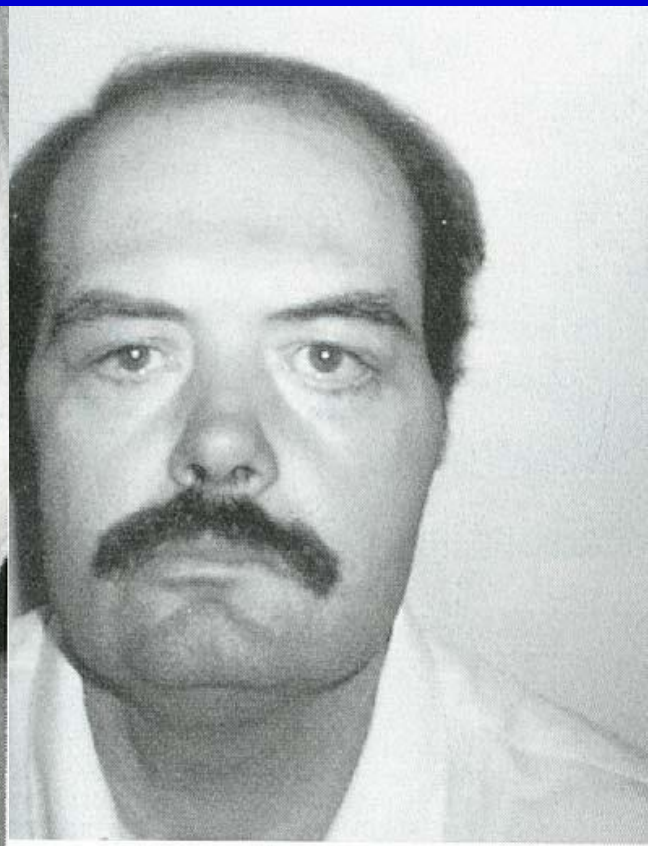


FIG. 38.1 Hypoglossal facial nerve transfer. Hypoglossal nerve is shown in *green,* facial nerve in *orange.* **A:** Classic procedure, with entire hypoglossal nerve transected. **B:** Modification with 40% segment of nerve secured to lower division.

MUSCLE FLAP



BELL'S PALSY

- Most common diagnosis of acute facial paralysis
- Diagnosis is by exclusion

PATHOLOGY

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

ETIOLOGY

- Vascular vs. viral

CLINICAL FEATURES

- Sudden onset unilateral FP
- Partial or complete
- No other manifestations apart from occasional mild pain
- 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

THANK YOU