بسم الله الرحمن الرحيم

CRANIAL NERVES DISORDERS

Cranial Nerves

I- OLFACTORY
II- OPTIC
III- OCULOMOTOR
IV- TROCHLEAR
V- TRIGEMINAL
VI- ABDUCENS
VII- FACIAL
VIII- VESTIBULOCOCHLEAR
IX- GLOSSOPHARYNGEAL
X- VAGUS
XI- ACCESSORY
XII- HYPOGLOSSAL

OLFACTORY nerve

-fibers enter the cranium through the cribriform plate to form the olfactory tract.

Cortical olfactory area is in the temporal lobe.

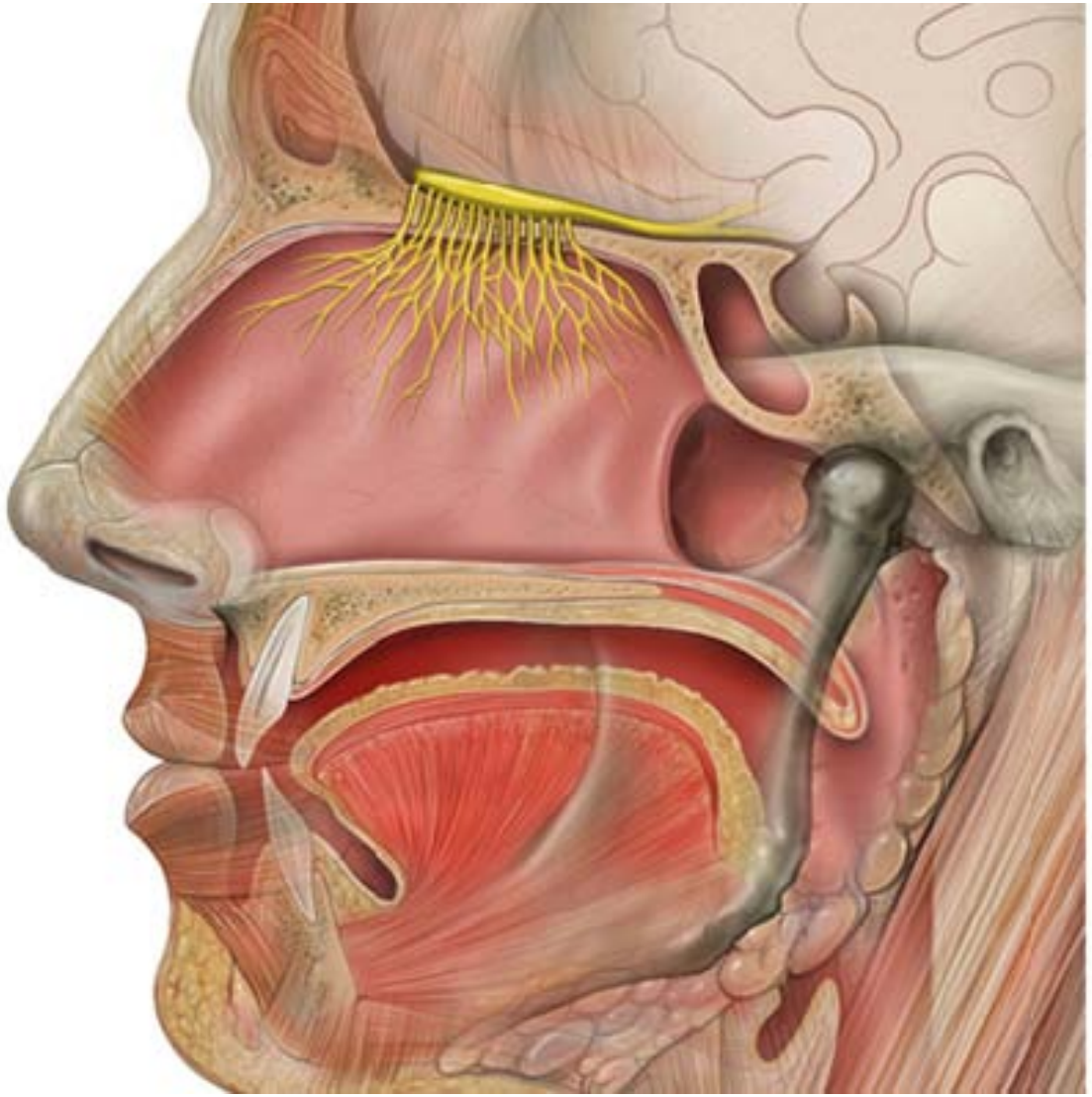
causes of anosmia:

A-nasal obstruction by infective or allergic oedema of the nasal mucosa.

B-degenerative including aging, Parkinson's and Huntington's diseases.

C-head injury

D- anterior fossa tumor



Optic

Discussed in the introduction

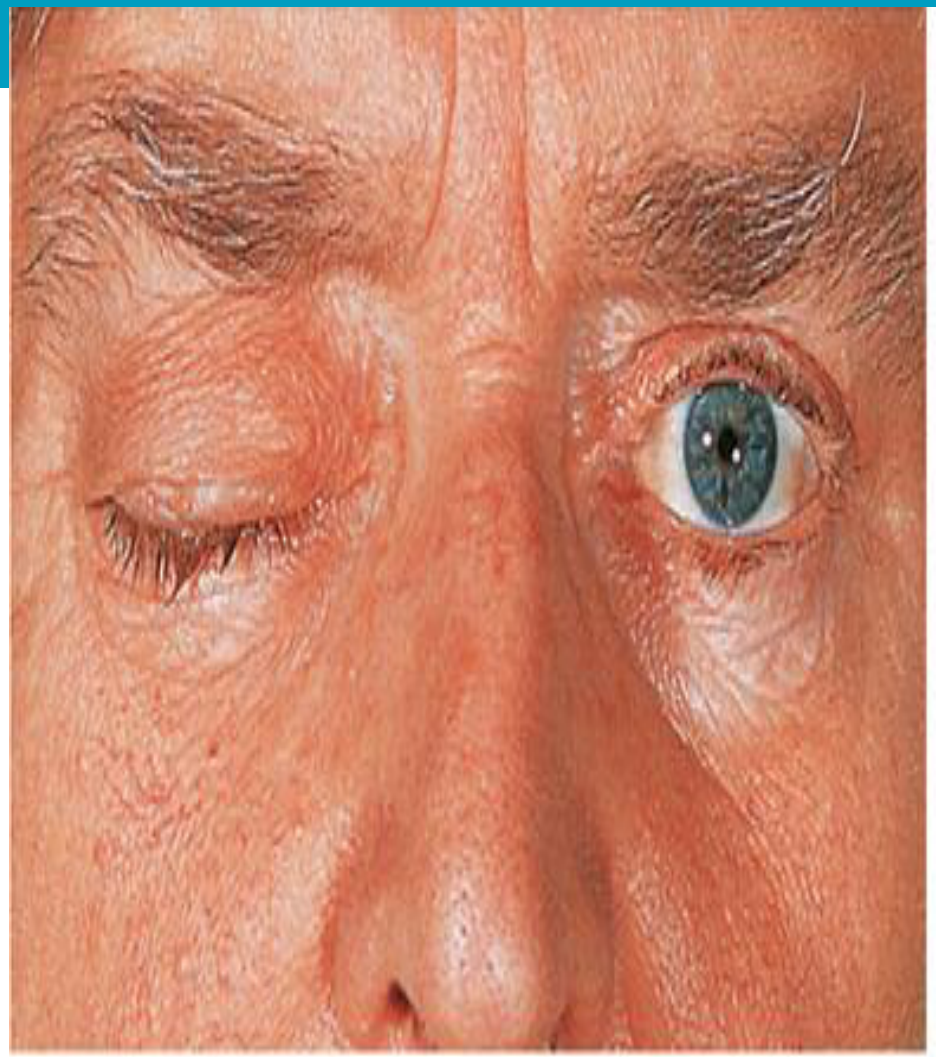
The oculomotor (III)

-The nerve innervates the superior, medial and inferior recti, the inferior oblique and levator palpebrae superioris muscles.

-parasympathetic fibres arising from the Edinger-Westphal nucleus, the nerve indirectly supplies the sphincter muscles of the iris, causing constriction of the pupil.

-it passes in relation to the posterior communicating artery and enters the dura surrounding the cavernous sinus.

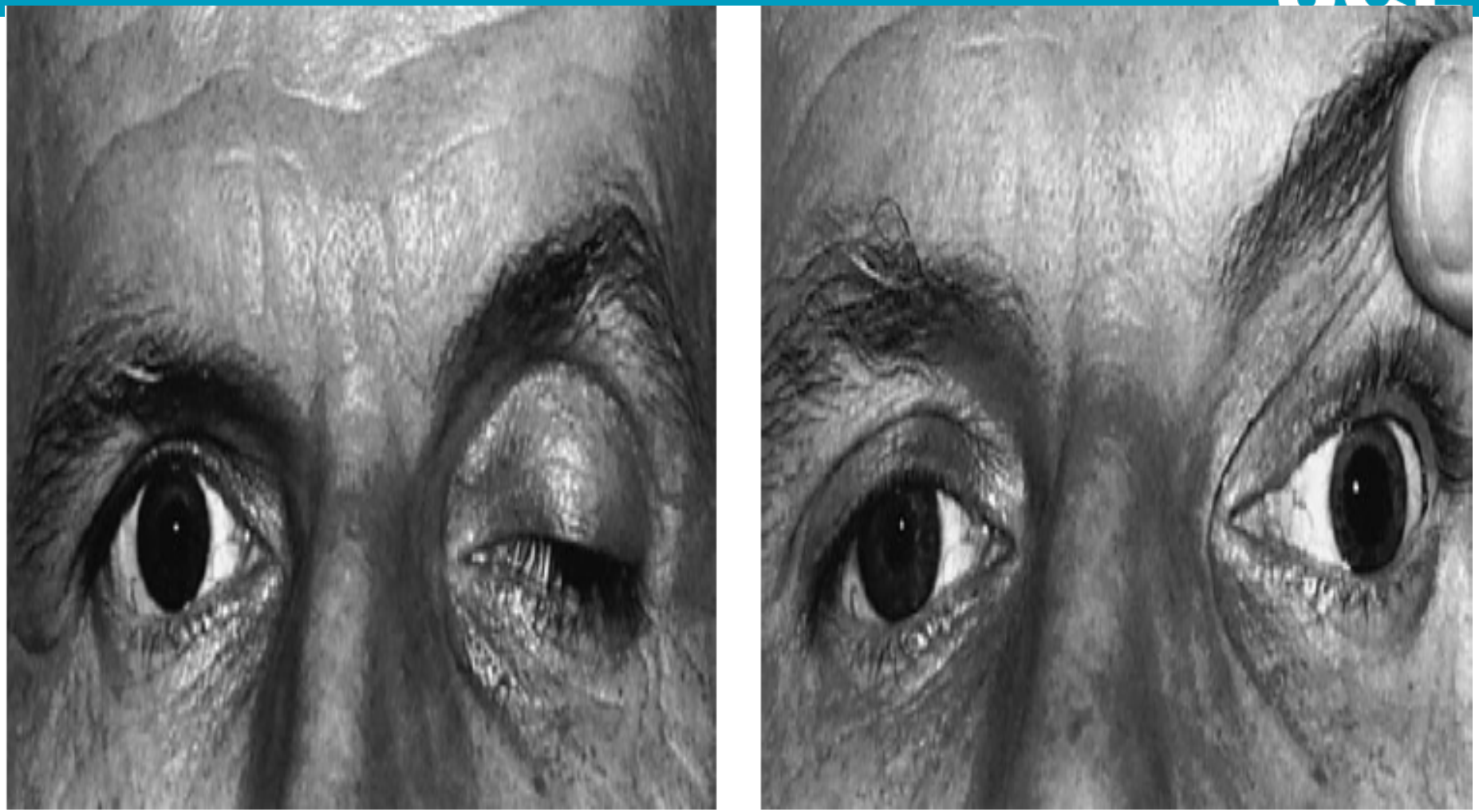
- III palsy= squint + complete ptosis + dilated pupil



A

B

Medical 3rd nerve palsy



Surgical 3rd nerve palsy

Trochlear (IV)

-innervate sup. Oblique muscle. Causes are

(1) Trauma

(2) Idiopathic

(3) Ischemic

(4) Congenital

(5) Tumor

-Vertical diplopia is most clear in down gaze.

ABDUCENS (VI)

Innervates the lateral rectus muscle.

-causes are

(1) Idiopathic

(2) Tumor

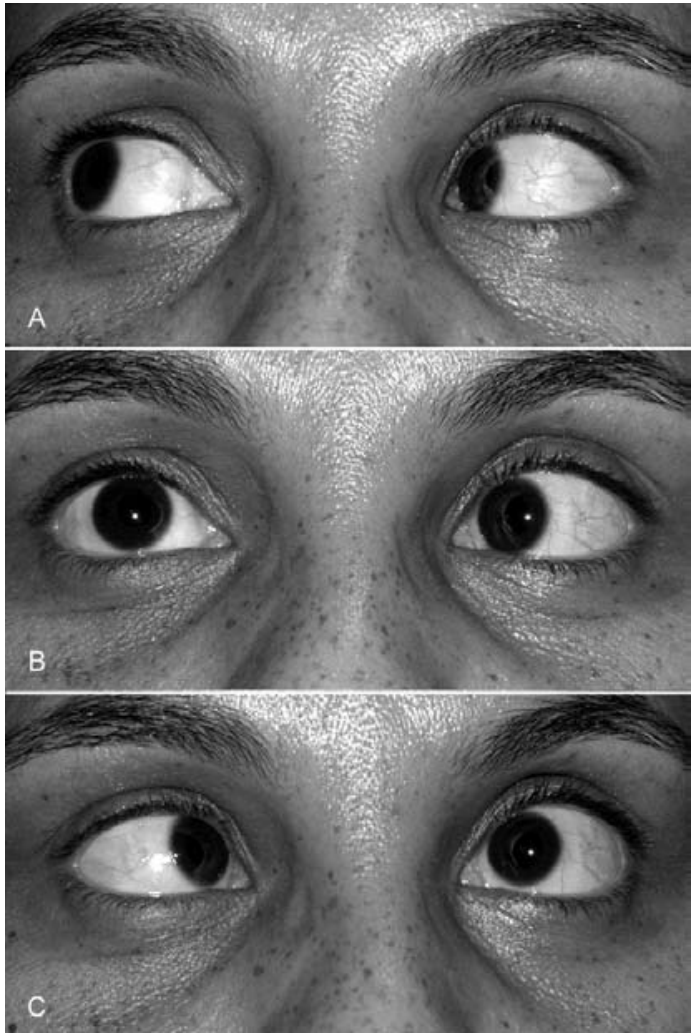
(3) Trauma

(4) Ischemia

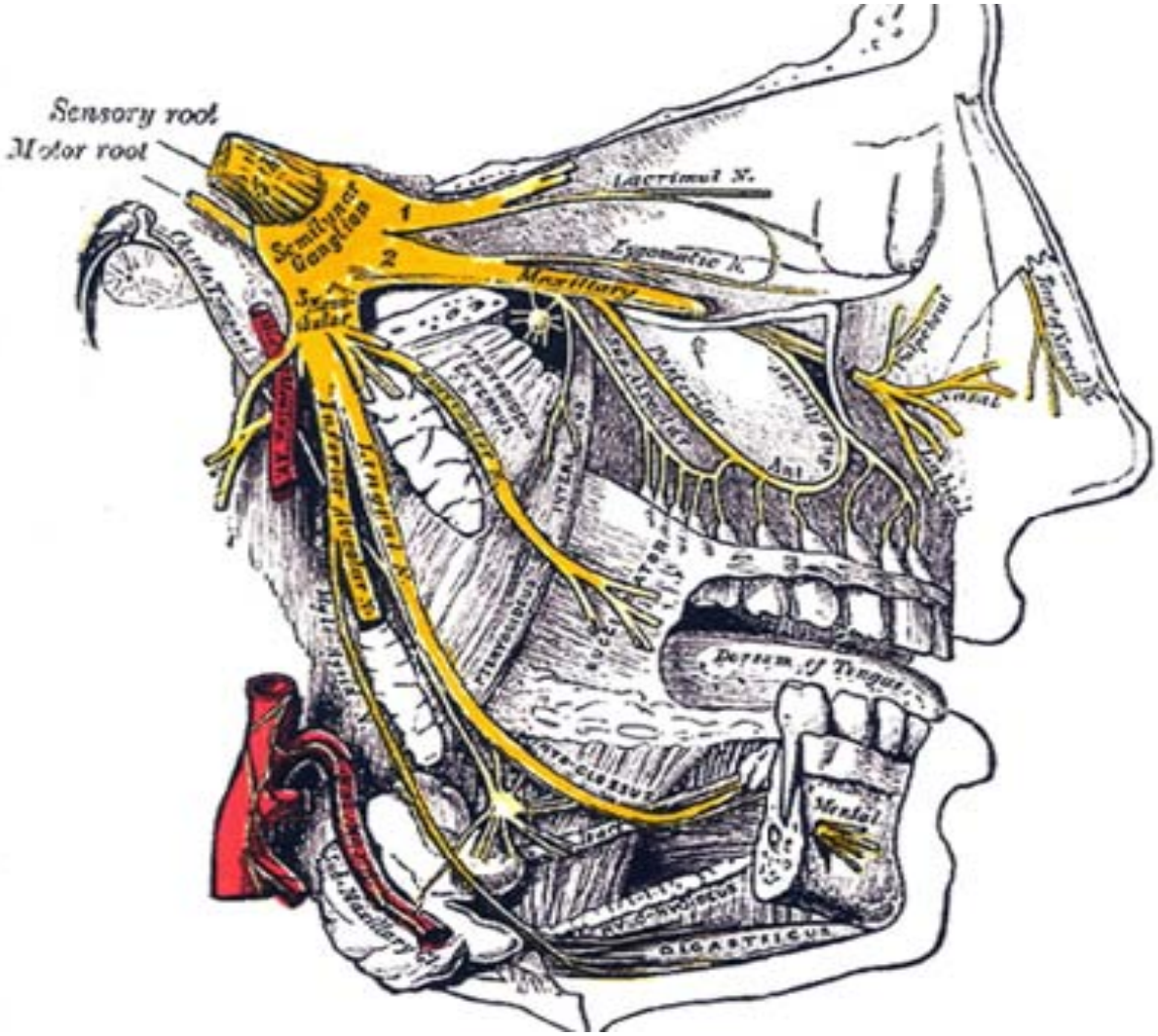
(5) Raised ICP as a false localizing sign

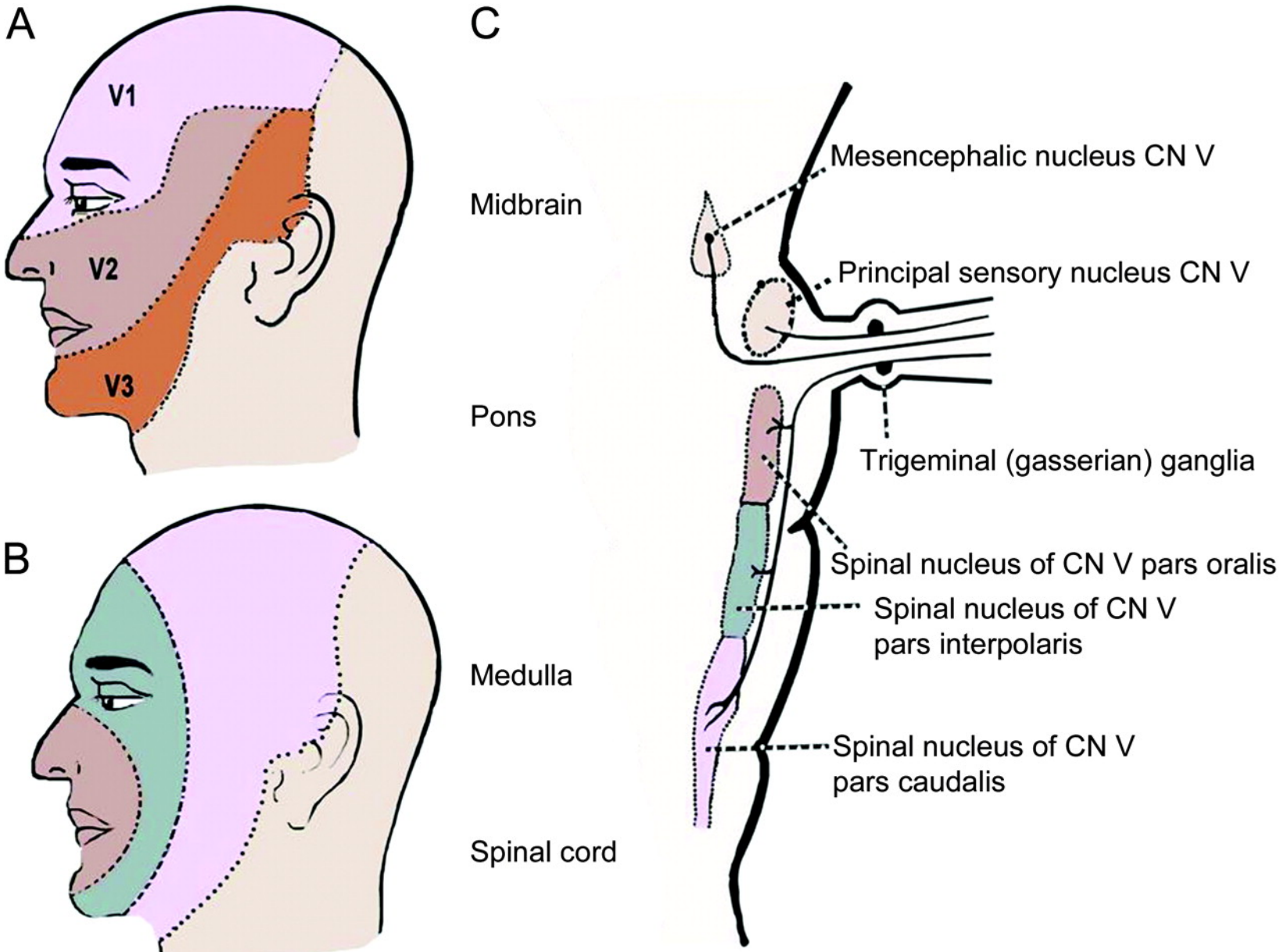
- VI palsy produces horizontal diplopia maximal to the direction of weakness.

CN VI palsy



Trigeminal nerve palsy


Sensory root
Motor root
Lacrimal N.
Zygomatic N.
Meckel's
Semilunar Ganglion
Auriculo-Temporal
Inferior Alveolar N.
Inferior Alveolar
Buccal
Mental
Digastricus


A
V1
V2
V3
B
C
Midbrain
Pons
Medulla
Spinal cord
Mesencephalic nucleus CN V
Principal sensory nucleus CN V
Trigeminal (gasserian) ganglia
Spinal nucleus of CN V pars oralis
Spinal nucleus of CN V
pars interpolaris
Spinal nucleus of CN V
pars caudalis

CN V disorders

Lesions lead to loss of sensation in the face and weakness in muscles of mastication

causes:

1-tumors(acoustic neuroma)

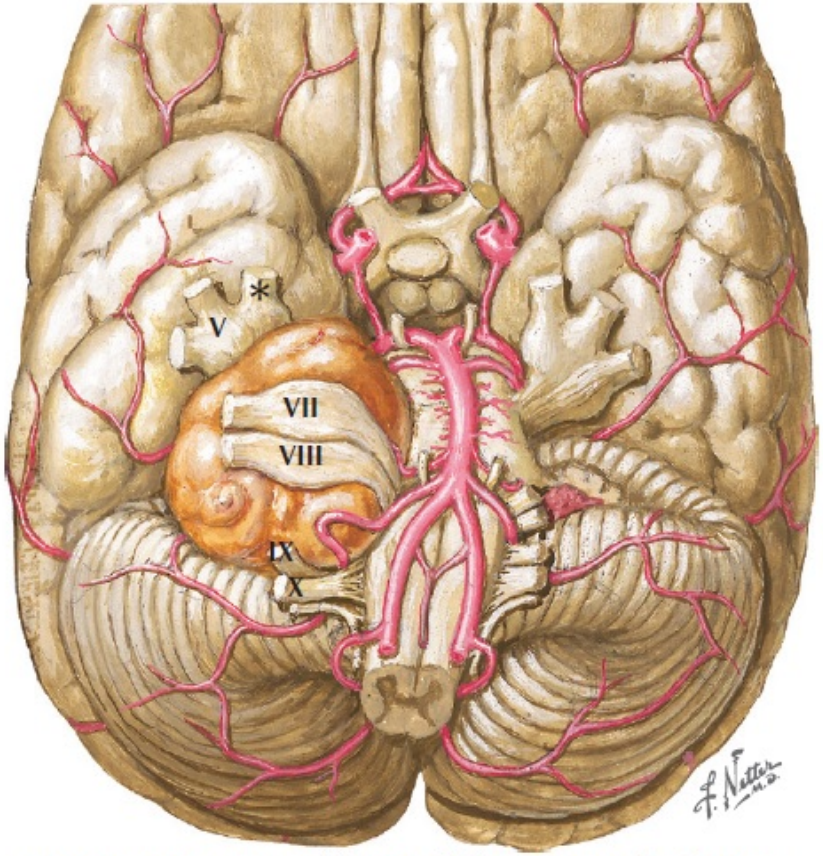
2-Sjogren”s disease

3-trauma

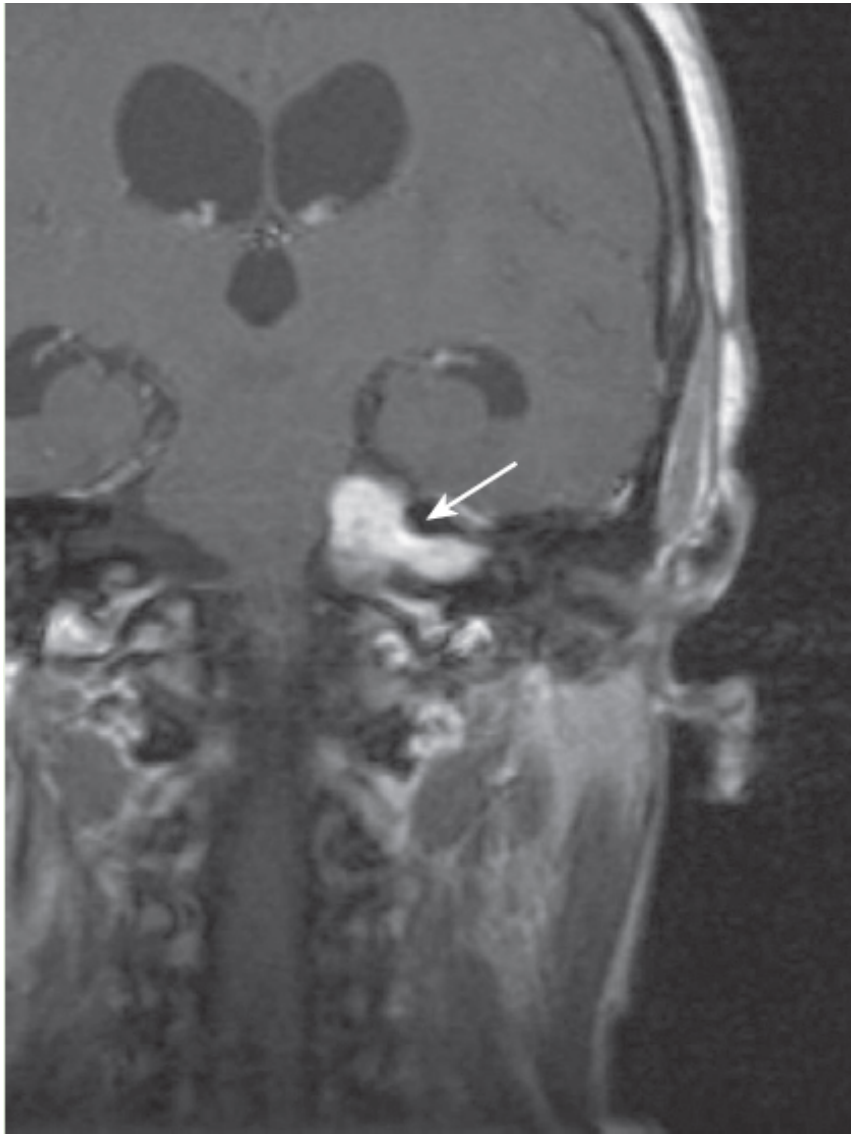
4-idiopathic

5-herpes zoster (usually ophthalmic division)

Acoustic Neur(in)oma



Large acoustic neurinoma filling cerebellopontine angle, distorting brainstem and cranial nerves V, VII, VIII, IX, X



Left V palsy

Facial (VII) nerve

Mediates

Sensory function: somatic sensation from external auditory meatus; taste (anterior 2/3 of tongue,)

Motor function to muscles of facial expression

parasympathetic function(GVE) to the lacrimal, submandibular and sublingual salivary glands (via nervus intermedius).

-it is emerging from the lateral pontomedullary junction in close association with the VIII nerve; together they enter the internal acoustic meatus.

-exiting the skull via the stylomastoid foramen.

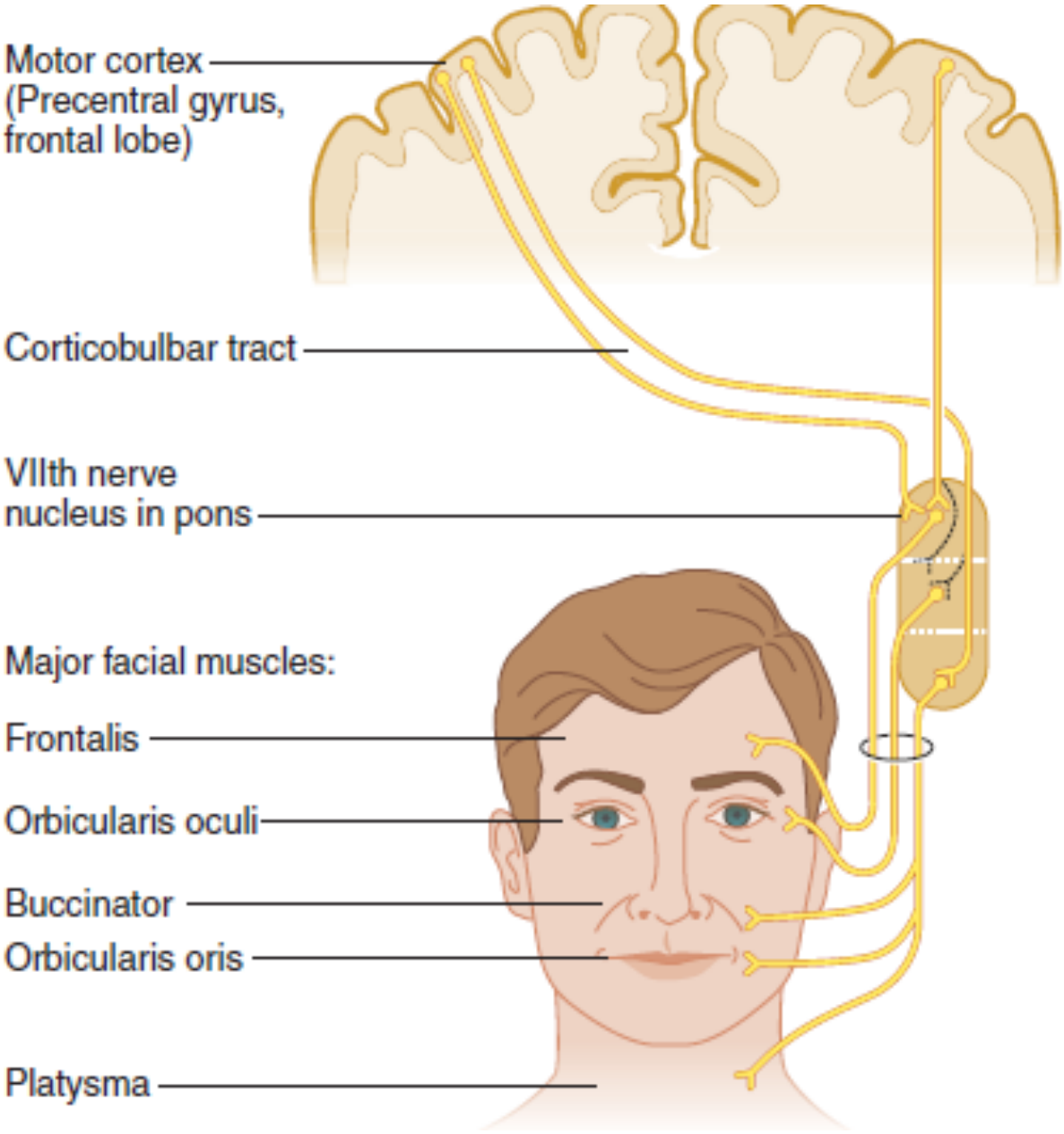
-Passing through the parotid gland

CN VII disorders

-In a unilateral **lower motor neurone** VII nerve lesion, there is weakness of both upper and lower facial muscles.

-**Bell's phenomenon** occurs when the patient is unable to close the eye. As he or she tries, the eyeball rolls upwards, exposing the conjunctiva below the cornea.

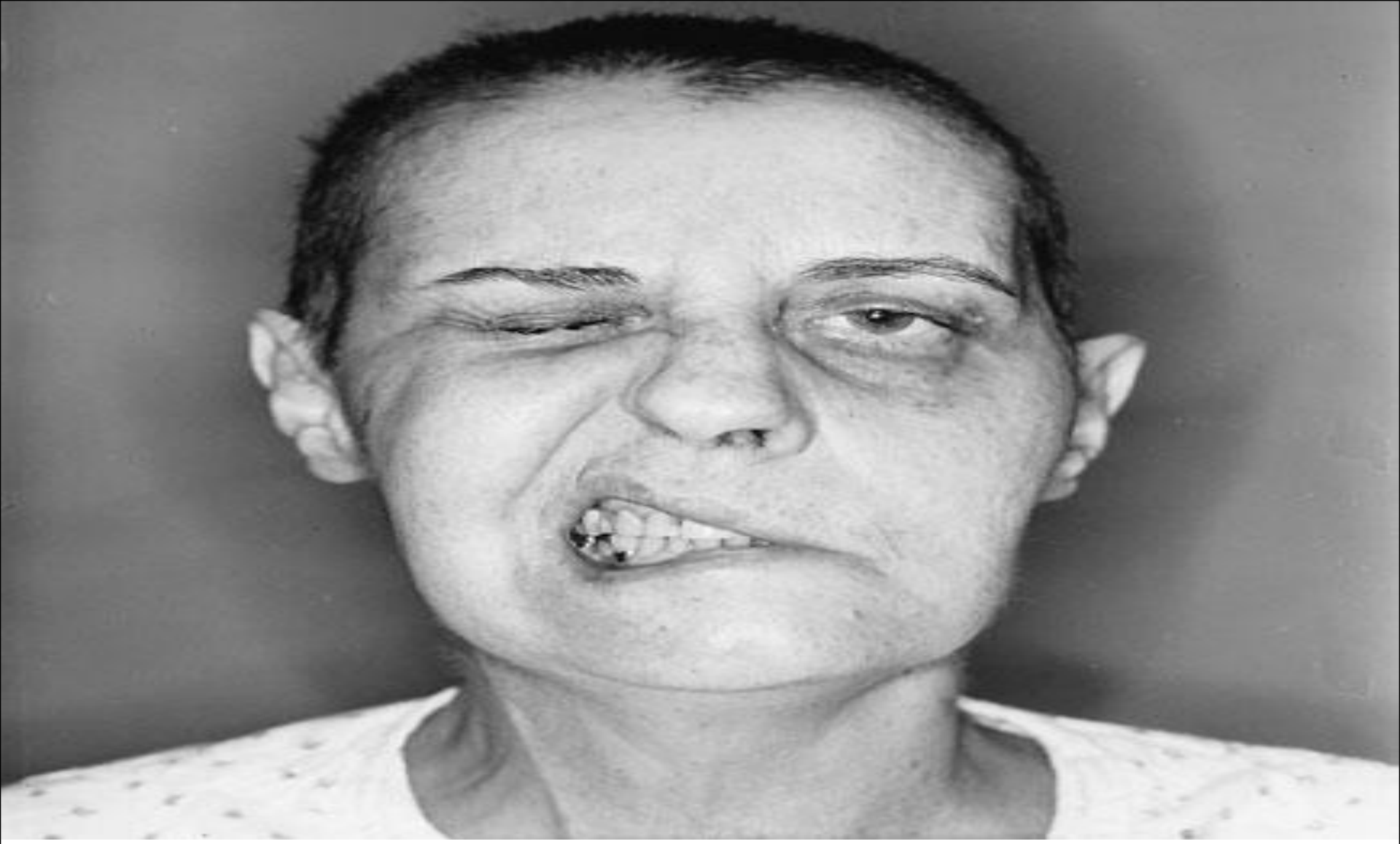
-In unilateral VII nerve **upper motor neurone** lesions, weakness (facial paresis) is marked in the lower facial muscles with relative sparing of the upper face. This is because there is bilateral cortical innervation of the upper facial muscles. While the nasolabial fold may be flattened and the corner of the mouth drooping, eye closure is usually well preserved.


Motor cortex
(Precentral gyrus,
frontal lobe)
Corticobulbar tract
VIIth nerve
nucleus in pons
Major facial muscles:
Frontalis
Orbicularis oculi
Buccinator
Orbicularis oris
Platysma

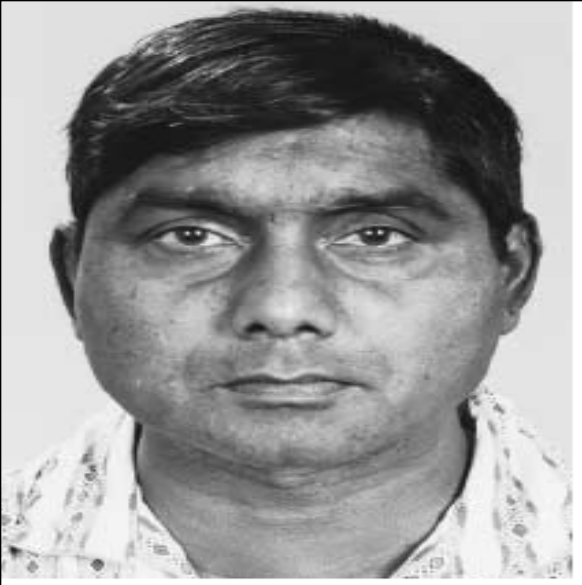
Bell's palsy

- The commonest cause of LMN palsy.
- The cause of this condition is not certain, although there is some evidence to suggest inflammation due to reactivation of herpes simplex virus within the nerve ganglion in many cases.
- The lesion is usually proximal enough to have effects on taste and hearing.
- After some aching around the ear, the facial weakness develops quite quickly within 24 hours.
- The cornea may be vulnerable to infection because of impaired eye closure.

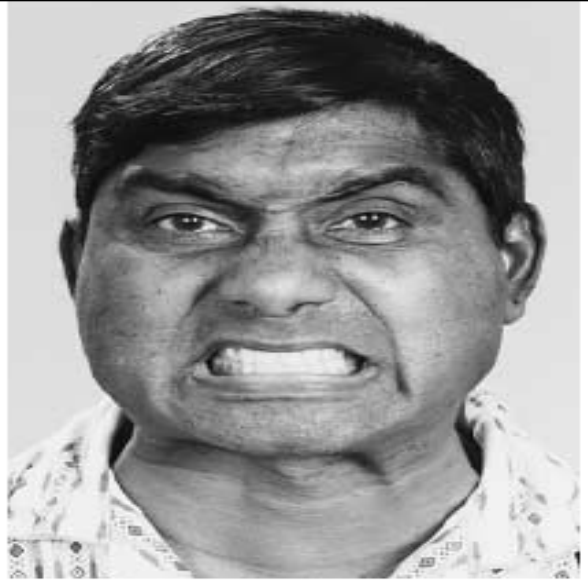
- Mx: steroid+ antiviral + eye protection



LMN V Palsy



A



B

C

D

E

F

Emotional and voluntary UMN facial weakness

Bilateral V palsy + Bell”s phenomenon

Causes of CN VII palsy

LMN palsy

1-Bell's palsy

2-diabetes

3- herpes zoster (Ramsay – Hunt syndrome)

4- cerebello-pontine angle tumors (acoustic neuroma)

5-parotid tumor or injury.

UMN palsy

1-stroke

2-multiple sclerosis

3-cerebral tumor

4-trauma

5-encephalitis

Vestibulocochlear nerve

consists of two functional divisions:

- Auditory nerve (cochlear)
- Vestibular nerve

Pure special visceral afferent nerve

Exits the brainstem at cerebellopontine (CP) angle

Disorders of VCN-A

Destructive (negative symptoms): sensori-neural hearing loss secondary to:

- Vascular
- inflammatory
- neoplastic aetiologies
- Meniere disease

Irritative (positive symptoms): tinnitus

Disorders of vestibular nerve

Lesions result in : vertigo, nystagmus and ataxia

Causes:

1-Vestibular neuropathy (diabetes, meningitis, hypothyroidism)

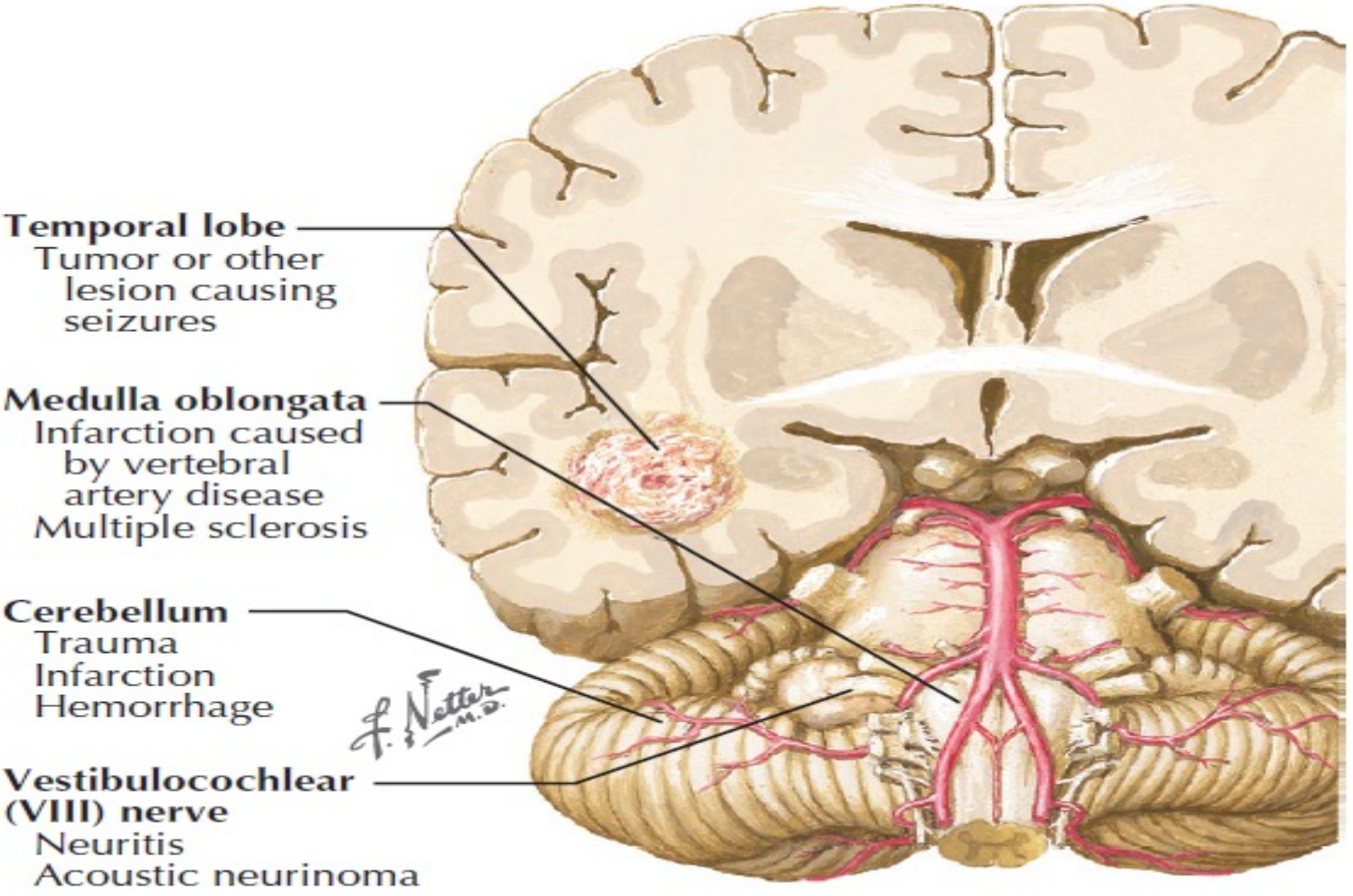
2-Acute peripheral vestibulopathy (vestibular neuritis)

3-Benign positional vertigo

4-Toxic vestibulopathy (drugs, alcohol)

5-Meniere disease (severity decrease with time, SNHL)

6-Otosclerosis


Temporal lobe
Tumor or other lesion causing seizures
Medulla oblongata
Infarction caused by vertebral artery disease
Multiple sclerosis
Cerebellum
Trauma
Infarction
Hemorrhage
Vestibulocochlear (VIII) nerve
Neuritis
Acoustic neurinoma
F. Netter M.D.

Glossopharyngeal nerve

Mediates:

Sensory function (somatic: part of external auditory , taste: from posterior 1/3 of tongue), taste sensation from posterior 1/3 of tongue

Motor function (: stylopharyngeus muscle)

Visceral efferent (parasympathetic) (GVE: Otic ganglia to parotid gland)

Nuclei: medulla

Exit foramen: jugular foramen

Vagus nerve

Mediates:

Sensory function (GSA: infratentorial dura, posterior surface of EAM, tympanic membrane; SVA: taste from epiglottis)

Motor function (SVE: , palate, muscles of swallowing, laryngeal muscles)

Visceral efferent (parasympathetic) to viscera of the neck, thocoabdominal viscera down to left colic flexure.

Nuclei: medulla

Exit foramen: jugular foramen

Disorders of GPN and VN

Bulbar and Pseudo bulbar palsy- similarities and differences, causes

Vascular

Inflammation

Tumors

Motor neuron disease

Myasthenia gravis

Accessory Nerve

Two parts: **cranial** (with CN X supplying the larynx; exit from jugular foramen), and **spinal** (from C1-C6, enter the skull via the foramen magnem to exit again via jugular foramen)

Mediates

Pure Motor function to the sternomastoid and trapezius muscles

Most common cause is iatrogenic injury(neck surgery)

Injury results in weakness or paralysis of respective muscles

Hypoglassal nerve

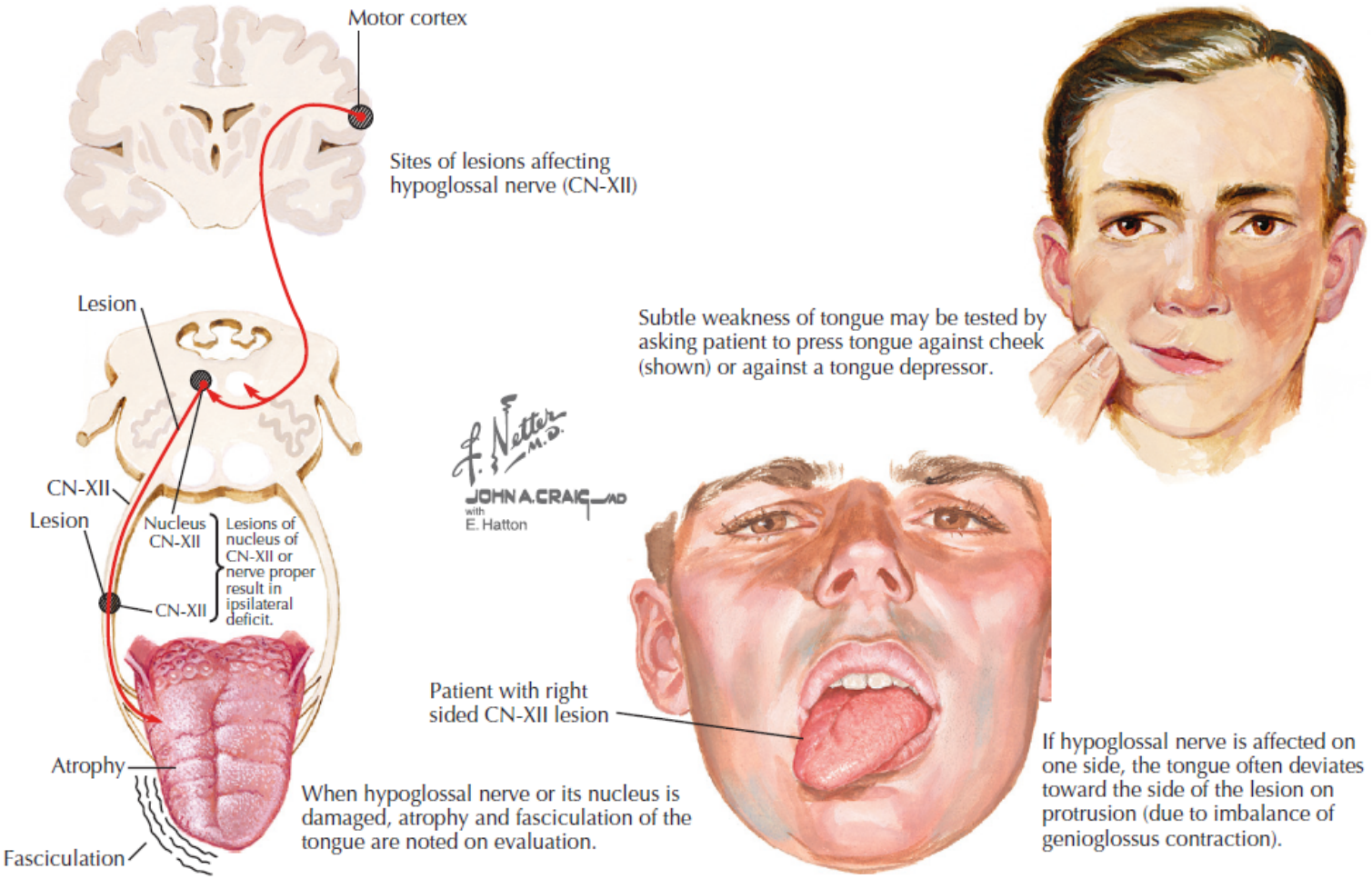
Pure motor nerve

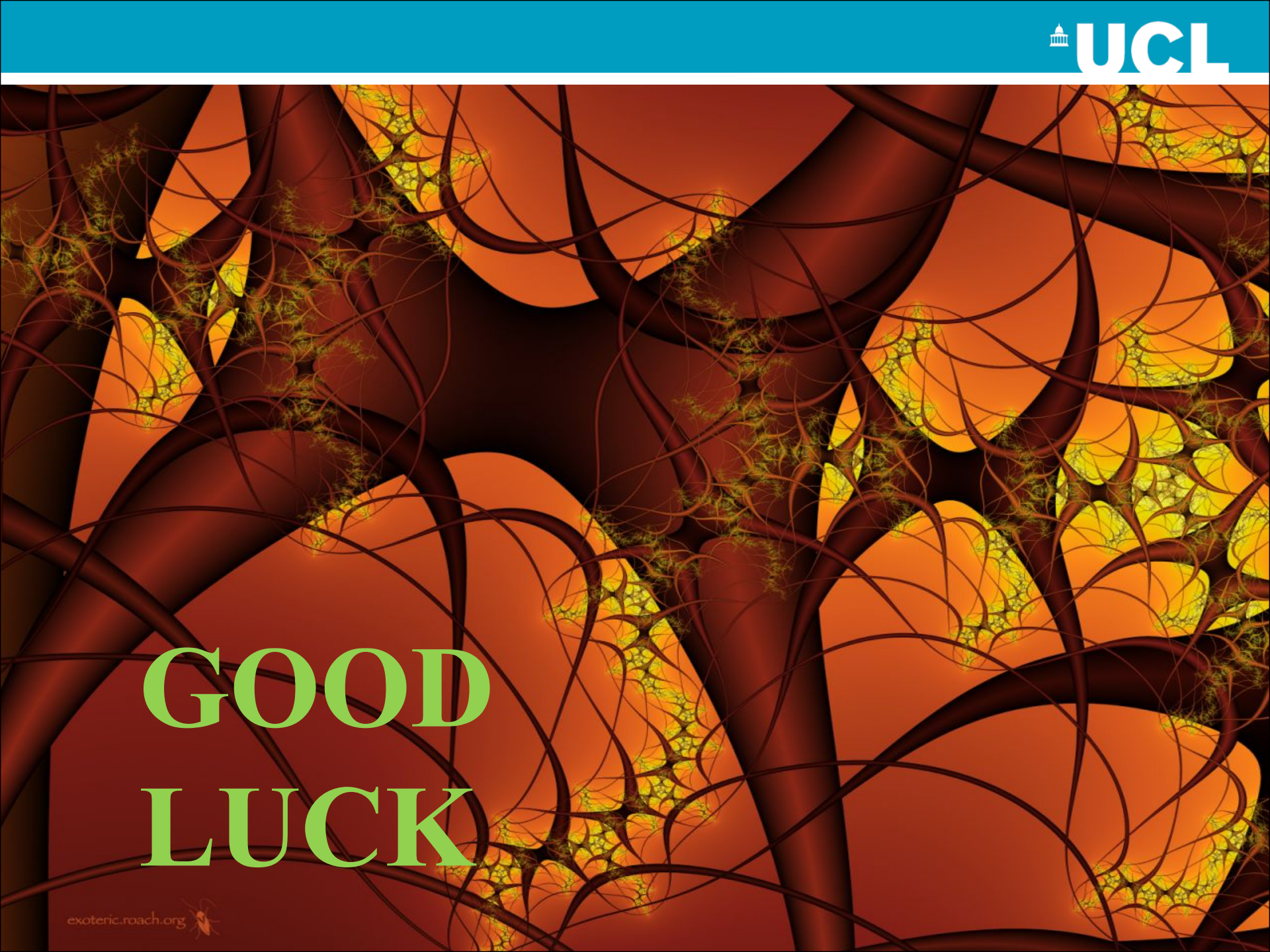
GSE to the extrinsic and intrinsic muscles of tongue

Nucleus: medulla

Exit foramen: hypoglossal canal

•Deviation of tongue will be to the side of affected hypoglossal nerve.


Motor cortex
Sites of lesions affecting hypoglossal nerve (CN-XII)
Lesion
CN-XII
Lesion
Nucleus CN-XII
Lesions of nucleus of CN-XII or nerve proper result in ipsilateral deficit.
CN-XII
Atrophy
Fasciculation
When hypoglossal nerve or its nucleus is damaged, atrophy and fasciculation of the tongue are noted on evaluation.
F. Netter M.D.
JOHN A. CRAIG MD
with
E. Hatton
Subtle weakness of tongue may be tested by asking patient to press tongue against cheek (shown) or against a tongue depressor.
Patient with right sided CN-XII lesion
If hypoglossal nerve is affected on one side, the tongue often deviates toward the side of the lesion on protrusion (due to imbalance of genioglossus contraction).


GOOD
LUCK
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