

**CORRELATIVE  
NEUROANATOMY**

# **CORRELATIVE NEUROANATOMY**

*by*

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## SECTION I

### THE PERIPHERAL NERVES

#### ANATOMY

The peripheral nerves constitute an intricate conduction system which serves as the mediator of neural impulses travelling in both directions between the central nervous system and other tissues of the body, and through which many important bodily functions are regulated. For descriptive purposes the peripheral nerves may be classified according to their function and site of origin from the central nervous system.

1. The cranial nerves - emerging from the base of the brain.
2. The spinal nerves - originating from the spinal cord.
3. The autonomic system - being intricately associated with the cranio-spinal nerves, but differing in function and details of structure and distribution.

A peripheral nerve trunk is composed of many nerve fibers bound together by supporting connective tissue. Each fiber represents the greatly elongated process of a nerve cell, the cell body of which lies within the central nervous system or one of the out-lying ganglia. The nerve cell or neuron, consisting of a cell body and all of its processes, constitutes the structural and functional unit of the nervous system (the neuron doctrine). The cell body, which contains the nucleus, is the vital center controlling the metabolic activity of the cell, and any injury which severs a nerve fiber will result in degeneration of the distal segment. Functionally three main groups of fibers occur in peripheral nerves.

1. Motor (or efferent) fibers which deliver impulses from the central nervous system to skeletal muscles for the control of voluntary muscular activity. Their cell bodies are located in the gray matter of the spinal cord and brain stem.
2. Sensory (or afferent) fibers carry impulses arising from various receptors in the skin, muscles, special sense organs, etc. to the central nervous system where they are interpreted as sensations. The cell bodies lie in special ganglia located along the roots of origin of the sensory nerves.
3. Autonomic fibers are efferent in function, concerned with the control of smooth muscle, glandular activities, and probably certain trophic functions of the body. Anatomical details are described in the section on the autonomic system.

LESIONS OF THE PERIPHERAL NERVES include the various types of pathological disturbances which affect other tissues of the body:

- |                         |                         |
|-------------------------|-------------------------|
| 1. Congenital defects   | 5. Vascular lesions     |
| 2. Inflammatory lesions | 6. Toxic lesions        |
| 3. Neoplasms            | 7. Degenerative lesions |
| 4. Trauma               | 8. Functional disorders |

#### SYMPTOMS AND SIGNS

The abolition of conductivity in a nerve results in an impairment of neurological function in the motor, sensory, and trophic spheres. (These are more fully described in the section on Neurodiagnosis).

1. Motor loss is manifested by paralysis or weakness of muscles.
2. Sensory involvement may be subjective or objective.
  - a. Subjective sensory findings include pain and paresthesias (feeling of numbness, tingling, crawling sensations, etc.). These usually indicate partial or irritative lesions.
  - b. Objective findings include the loss of various sensibilities, as analgesia (loss of pain), anesthesia, etc.
3. Trophic disturbances are related to impaired nutritional and metabolic activities in the tissues, which are partially under neurogenic control. The signs are most marked in the cutaneous tissues as dryness, cyanosis, loss of hair, brittle nails, ulcerations, slow healing of wounds, etc.

## DISEASES OF THE PERIPHERAL NERVES

NEURITIS - a local or widespread inflammation or degeneration of the peripheral nerves.

MONONEURITIS (or localized neuritis) affects a small group of nerves or a single nerve trunk. Etiological factors include:

1. Trauma - as contusion, tearing, compression, or stretching of the nerve.
2. Chronic intoxications by alcohol or metallic poisonings.
3. Infections - local or generalized, or by extension from adjacent infected parts.

Pathologically the inflammatory reaction may be of three types:

1. Perineuritis - limited to the perineurium ) nerves swollen and red.
2. Interstitial neuritis - affecting the interstices )
3. Parenchymatous neuritis - affecting the nerve fibers themselves (myelin sheath, axis cylinders, and neurilemma) causing a shrunken, pale, translucent appearance.

Symptoms include - 1. Irritative phenomena, as pain, tenderness, paresthesias.

2. Motor loss - flaccid paralysis with muscle atrophy and reaction of degeneration.
3. Sensory loss, and at times trophic and vasomotor changes.

Prognosis depends upon the extent and character of the injury, and treatment is directed toward removal of the cause, relief of pain, and prevention of contractures.

MULTIPLE NEURITIS (polyneuritis or peripheral neuritis) is an acute or subacute disseminated inflammation or degeneration of symmetrically distributed nerves, affecting principally their distal portions and occurring most frequently in the second, third, and fourth decades of life. The known causes include:

1. Exogenous toxins - as alcohol, lead, CO, nitrobenzol, zinc, coal tar products.
2. Endogenous toxins associated with diabetes, tuberculosis and syphilis, etc.
3. Infectious diseases - diphtheria, influenza, typhoid fever, and others.
4. Deficiency diseases - beri-beri.

Pathologically the nerve lesion is only part of a generalized intoxication which also affects the brain and spinal cord to a variable degree.

The symptoms are those of the causative disease which include the neurological disturbances; the onset is often gradual beginning with numbness, slight fever, and tingling in the extremities; convulsions may occur in children. The resulting paralysis is of the flaccid, lower motor neuron type, with muscle atrophy, loss of reflexes, reaction of degeneration and is associated with sensory loss. Foot drop and wrist drop are common and due to involvement of the extensor muscles. Differentiation must be made from tabes dorsalis and poliomyelitis.

NEUROMATA or tumors growing from nerves are of two types:

- A. True neuromata (rare) arising from actual nerve tissue, usually in connection with the sympathetic system; distributed beneath the skin, they are often multiple.
- B. False neuromata - arise from the connective tissue of the nerve trunk, usually on the spinal nerves and often in large numbers. They appear in the first half of life, as a rule, and are often hereditary. Various types include plexiform neuromata, pachydermatocoele, and multiple neurofibromatosis (von Recklinghausen's disease). The most common symptom of nerve tumors is pain, usually intermittent, and radiating to the periphery of the nerve. Paresthesias may occur. Treatment is by excision.

NEURALGIA is a syndrome affecting various sensory nerves and is characterized by sudden paroxysmal attacks of pain, usually of short duration, occurring in the distribution of the nerve fibers and not associated with pathological changes in the nerve. An attack may be brought on by various causes, as local pressure, cold, movement, pressure on the nerve trunk or stimulation of a "trigger zone". Vasomotor symptoms may accompany an attack as reddening of the skin, sweating, edema, tearing, and sialorrhea. The various types of neuralgia include:

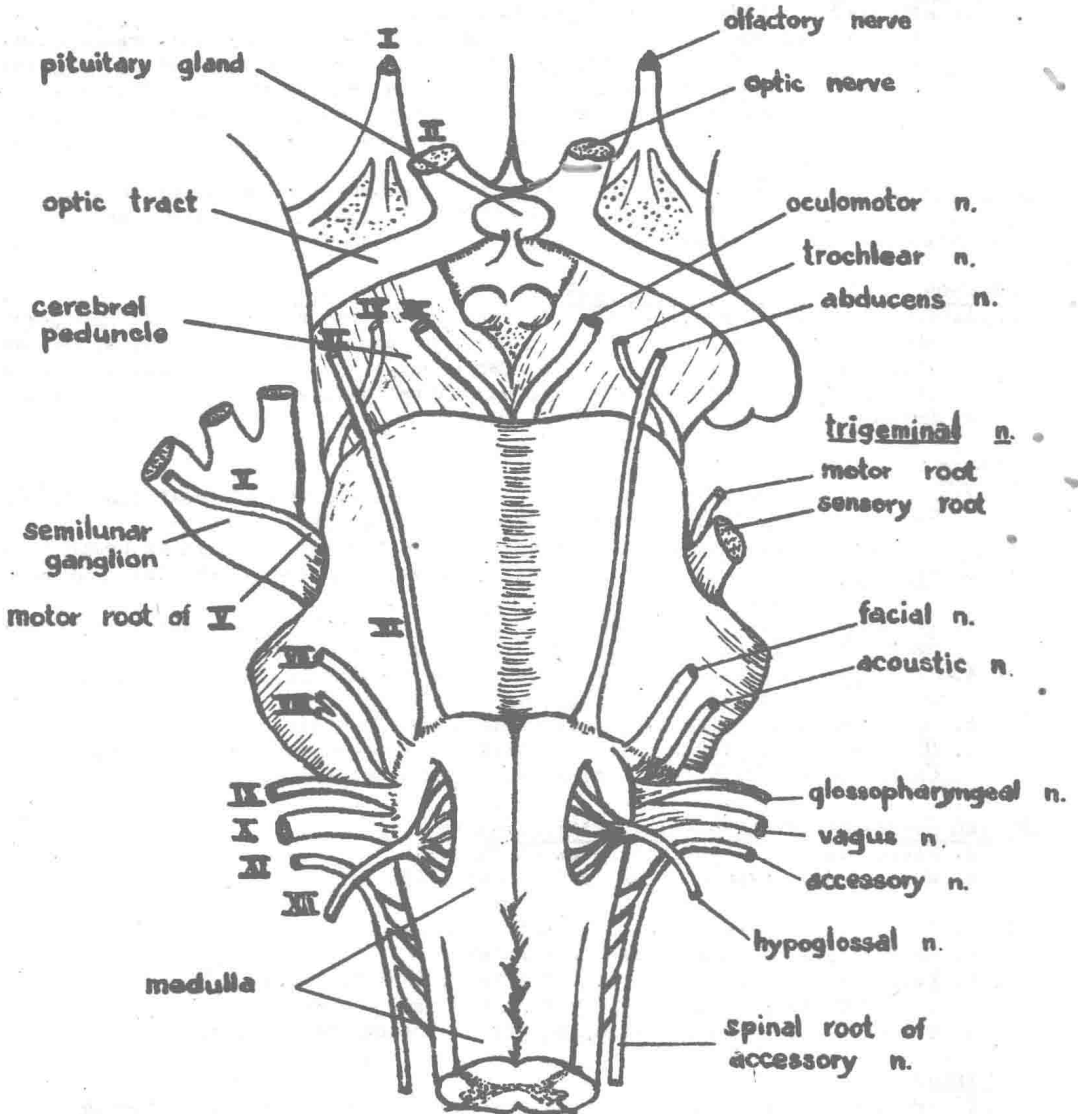
- |                     |                       |                |               |
|---------------------|-----------------------|----------------|---------------|
| 1. Trigeminal       | 4. Superior laryngeal | 7. Intercostal | 10. Lumbar    |
| 2. Sphenopalatine   | 5. Cervico-occipital  | 8. Phrenic     | 11. Sciatic   |
| 3. Glossopharyngeal | 6. Brachial           | 9. Visceral    | 12. Coccygeal |

## THE CRANIAL NERVES

3

The cranial nerves are customarily described as comprising 12 pairs which are referred to by number. The first two (olfactory and optic) are not true nerves but fiber tracts of the brain. The caudal ten pairs emerge from the brain stem in which lie their nuclei of origin, although a part of the 11th (accessory) nerve is derived from the upper cervical segments of the spinal cord. Anatomical and clinical aspects are outlined on the following pages.

Fig. 1 - THE CRANIAL NERVES



EMERGENCE OF CRANIAL NERVES FROM THE BRAIN (I-1.5)



4

**CRANIAL NERVE I - OLFACTORY NERVE AND TRACT**  
(A sensory nerve)

**ANATOMY -**

1. Primary neurons - Unmyelinated processes of the ciliated receptors in the upper part of the nasal mucosa are gathered into about 20 branches which pass through the cribriform plate of the ethmoid bone to the olfactory bulb.
2. Secondary neurons - Myelinated processes of the bipolar cells of the bulb form the olfactory tract and terminate in the subcallosal and hippocampal gyri where
3. Tertiary neurons - pass via the longitudinal striae and uncinate gyrus to the hippocampus which is the cortical representation of smell.

**Central Connections - are complex.**

1. Association fibers to the tegmentum and pons:
  - a. Pass directly as 3rd order neurons from the anterior perforated substance and
  - b. Indirectly from the hippocampus via the fornix and olfactory projection tracts through the mammillary bodies and anterior nuclei of the thalamus.
2. Reflex connections are thus established with nuclei of the other cranial and spinal nerves and are functionally significant in swallowing and digestion.

**Special Consideration** - The olfactory nerves may serve as a portal of entry for cryptogenic infection of the brain and meninges, e. g., poliomyelitis, epidemic meningitis and encephalitis.

**CONDITIONS AFFECTING THE SENSE OF SMELL -**

- |  |   |                                 |
|--|---|---------------------------------|
| 1. Tumors of the frontal lobe and pituitary region.    | 4. Fracture of the anterior fossa of the skull. | 8. Hydrocephalus.               |
| 2. Inflammatory and other lesions of the nasal cavity. | 5. Hysterias.                                   | 9. Arteriosclerosis.            |
| 3. Meningitis.   | 6. Congenital defects.                          | 10. Certain drug intoxications. |
|  | 7. Insanities.                                  |                                 |

**SYMPTOMS -**

1. Anosmia - (loss of sense of smell) - in general, not of great significance.
  - a. Bilateral - commonly with colds, rhinitis, etc.
  - b. Unilateral - may be of diagnostic significance in locating brain lesions.
2. Hyperosmia - (acute sense of smell) - present in some hysterias, and some cocaine addicts.
3. Parosmia - (perverted sense of smell) - a subjective disorder seen in some cases of schizophrenia, uncinate gyrus lesions and hysterias.
4. Cacosmia - unpleasant odors, usually due to decomposition of tissues and noticed by patient on expiration.
5. Hallucinations of smell - present in some insanities.
6. Uncinate gyrus fits - caused by lesions of the uncus and hippocampus, and characterized by hallucinating disturbances of smell.

**SPECIAL SYNDROMES INVOLVING THE OLFACTORY NERVE -**

1. Foster Kennedy - see syndromes on page 145
2. Aura of epilepsy - see epilepsy on page 136

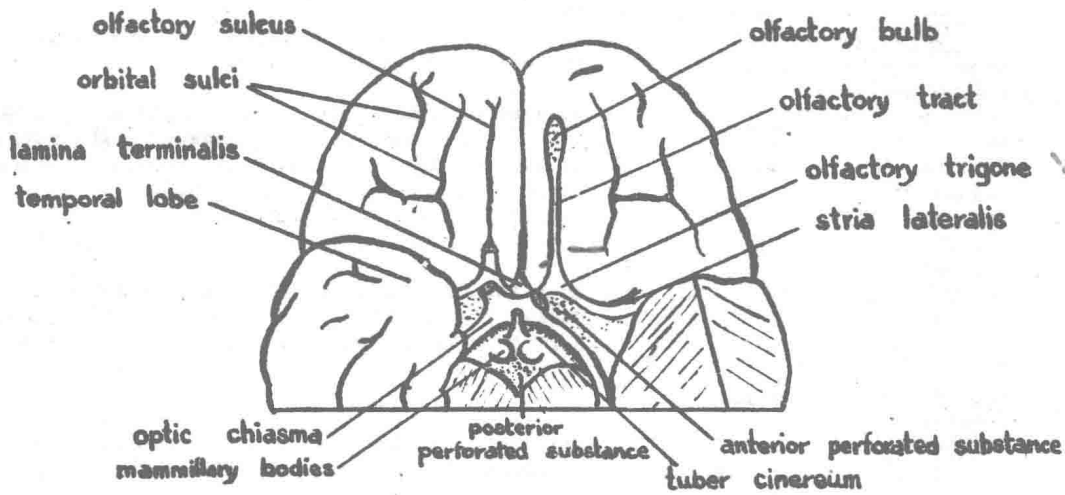
**TESTS -** for presence or absence of olfactory sense.

1. Each nostril must be tested separately.
2. Familiar non-irritating odors are best. (volatile oils)  
Oil of cloves, turpentine, citron, etc. may be used.
3. Elsberg test - records time required to recognize an odor.

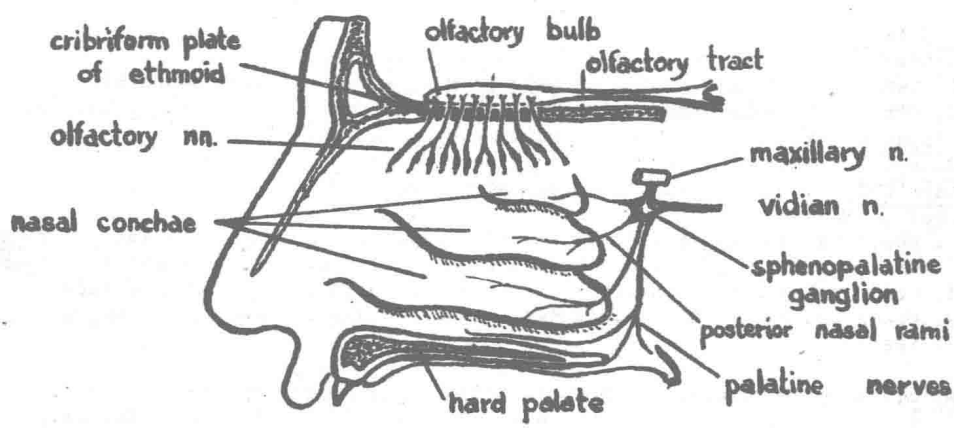
**REFERENCES -**

1. Wechsler - Textbook of Clinical Neurology (5th ed.) - pages 254-256
2. Grinker - Neurology (3rd ed.) - pages 337-339
3. Jelliffe and White - Diseases of the Nervous System (6th ed.) - pages 357-364
4. Brain - Diseases of the Nervous System (2nd ed.) - pages 134-135

Fig. 2 - THE OLFACTORY NERVE



a. Inferior view



b. Lateral view

## CRANIAL NERVE II - OPTIC NERVE (A sensory nerve)

ANATOMY - (structurally a fiber tract of the brain):

- A. Rods and cones of the retina are the first-order neurons which connect with
- B. Bipolar cells of the retina which in turn synapse with the
- C. Ganglion cells or third-order neurons, myelinated axons of which
  1. Form the optic nerve: fibers from the nasal half of each retina
  2. Cross at the optic chiasma - thus fibers from homolateral halves of the retinae
  3. Form the optic tracts - passing to the lateral geniculate bodies, superior colliculi and pretectal region.
- D. The geniculocalcarine tract contains the fourth order neurons from the lateral geniculate bodies passing to the occipital (calcarine) cortex.

Central connections -

- A. From the pretectal region to the Edinger-Westphal nuclei via the posterior commissures (these fibers being responsible for the simple and consensual light reflexes).
- B. From the superior colliculi via the tectobulbar and tectospinal tracts to other cranial and spinal nuclei for involuntary oculo-skeletal reflexes.
- C. From the occipital cortex - association and reflex fibers pass to
  1. Other cortical centers - (related to higher functions, e.g., reading, speech, etc.).
  2. The superior colliculi - and thus through the tectobulbar and tectospinal tracts to cranial and spinal nuclei for voluntary reflexes, e.g., accommodation.
  3. The pontile nuclei - via the corticopontile tract - for postural reflexes.

LESIONS OF THE VISUAL APPARATUS -

- A. Neuritis -
  1. Retrolbulbar - (involves the optic nerve or tract) - axial, peripheral and diffuse types. The most common cause is multiple sclerosis.
  2. Optic or Bulbar - includes various forms of retinitis, e.g., simple, albuminuric, syphilitic, diabetic, hemorrhagic, and hereditary.
- B. Papilledema - (choked disc) - usually a symptom of increased intracranial pressure - from brain tumors, abscesses, hemorrhage, and many other causes.
- C. Optic atrophy - atrophy of the optic nerve.
  1. Primary - (or simple) - from tabes, multiple sclerosis, or heredity.
  2. Secondary - to neuritis, glaucoma, or increased intracranial pressure.
- D. Opacities of the lens, corneal scars and arteriosclerotic changes in retina.
- E. Tumors and other lesions interrupting the optic pathways - (see below).

VISUAL DEFECTS -

- A. Scotomata - abnormal blind spots in the visual fields.
  1. Central scotomata - loss of macular vision due to axial neuritis.
  2. Other scotomata - due to patchy lesions as in hemorrhage and glaucoma.
- B. Amblyopia - a defect in visual acuity - weak vision.
- C. Amaurosis - complete blindness - may be congenital or acquired.
- D. Field defects - (See diagram on opposite page).
- E. Other disorders -
  1. Hemeralopia - day blindness - vision best in dim light - (a fatigue syndrome).
  2. Nyctalopia - night blindness - a symptom of retinitis pigmentosa (congenital).
  3. Color blindness - acquired (or total) and dichromatism or inherited.
  4. Optic agnosia - (word blindness) - cannot name objects seen - angular gyrus lesion.

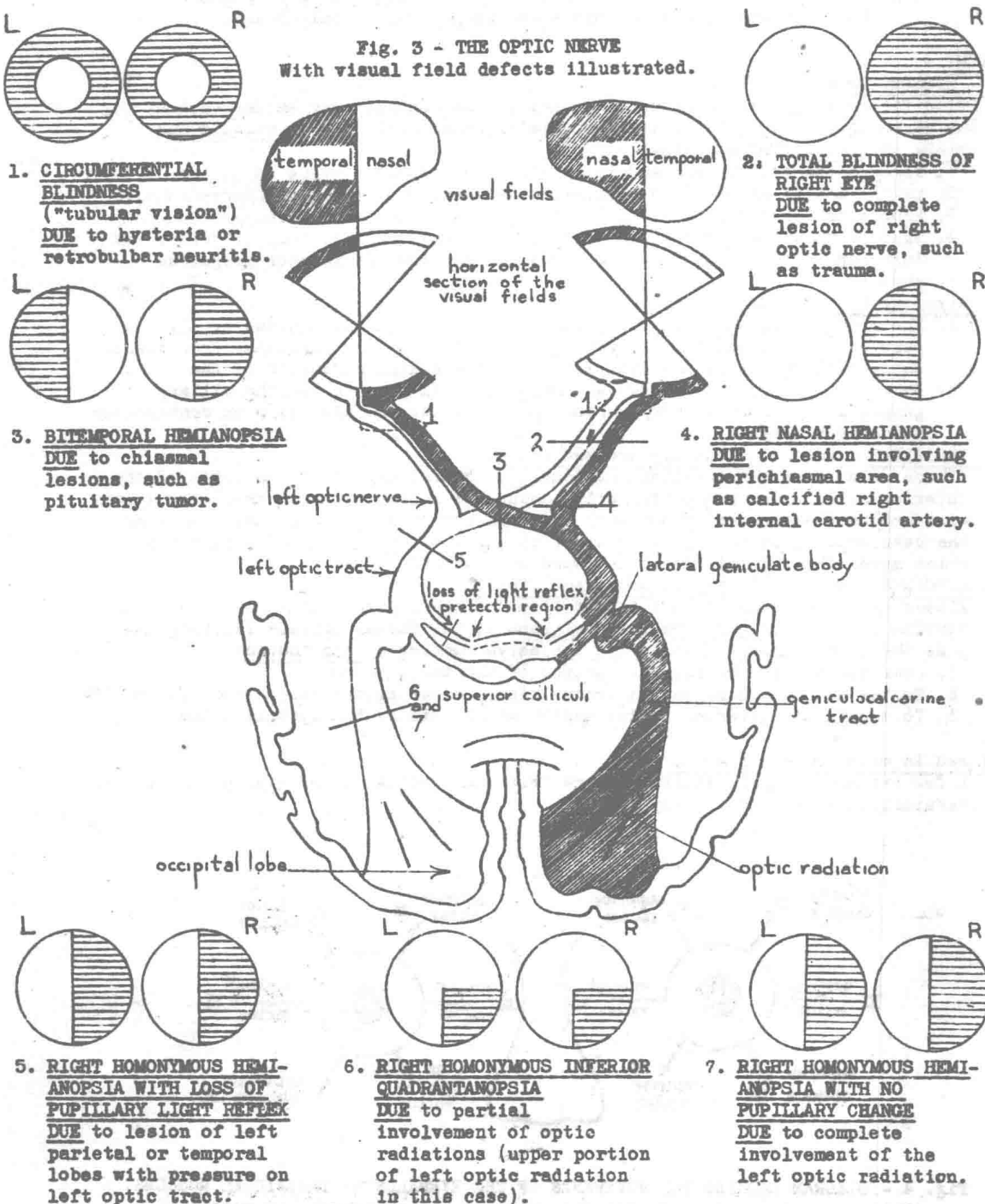
SPECIAL SYNDROMES - involving the optic nerve. See pages 141-145

1. Foster Kennedy.
2. Amaurotic family idiocy.
3. Argyll Robertson pupil.

TESTS -

1. Visual acuity - Snellen card test for those with fairly normal vision. Finger counting and finger movement tests for subnormal cases. Light perception and projection for markedly subnormal cases. (Cataracts are never removed if light perception is gone).
2. Perimetry - Plotting fields of vision - for determining presence of scotomata. May be done with colors.
3. Color blindness tests - using colored wools or special cards.
4. Fundus examination - by ophthalmoscopy.

**Fig. 3 - THE OPTIC NERVE**  
With visual field defects illustrated.



**REFERENCES -**

1. Purves-Stewart - (9th ed.) pages 80-86
2. Wechsler - Textbook of Clinical Neurology - (5th ed.) pages 255-259
3. Jelliffe and White - Diseases of the Nervous System - (6th ed.) pages 365-384
4. Grinker - Neurology - (3rd ed.) pages 339-345
5. Gifford - Textbook of Ophthalmology - pages 312-330

CRANIAL NERVES III, IV, and VI - OCULOMOTOR, TROCHLEAR, and ABDUCENS.  
(Motor to muscles of the eye - including levator palpebrae).

**ANATOMY -**

**III - OCULOMOTOR -**

A. Motor fibers arise from a group of nuclei in the central gray matter ventral to the cerebral aqueduct at the level of the superior colliculus. Crossed and mainly uncrossed fibers course:

1. Through the red nucleus and inner side of substantia nigra to emerge
2. On the mesial side of the cerebral peduncles, from where the nerve runs
3. Alongside the sella turcica, in the outer wall of the cavernous sinus and
4. Through the superior orbital fissure to supply the internal, superior, and inferior recti muscles, and the inferior oblique and levator palpebrae muscles.

B. Parasympathetics - arise from:

1. The Edinger-Westphal nucleus just rostral to the motor nucleus of III and pass via the nasociliary branch of III to the ciliary ganglion, from where short ciliary nerves are distributed to the sphincter muscle of the iris.
2. The upper portion of the medial nucleus of III passing via the ciliary ganglion and short ciliary nerves to the ciliary muscle which on contraction thickens the lens.

**IV - TROCHLEAR - Motor - (entirely crossed fibers)**

Fibers arise from the trochlear nucleus just caudal to III at the level of the inferior colliculus, run posteriorly, decussate in the anterior medullary velum, wind around the cerebral peduncles, and from here the nerve follows III along the cavernous sinus to the orbit where it supplies the superior oblique muscle which moves the axis of vision downward and outward.

**VI - ABDUCENS - Motor - (entirely uncrossed fibers)**

Fibers arise from the nucleus in the floor of the fourth ventricle in the lower portion of the pons near the internal genu of the facial nerve. Piercing the pons the fibers emerge anteriorly, the nerve running a long course:

1. Over the tip of the petrous portion of the temporal bone.
2. To the outer wall of the cavernous sinus, entering the orbit with III and IV.
3. To supply the external rectus muscle which rotates the eyeball outward.

Included in each of the above nerves -

A few sensory (proprioceptive) fibers from the muscles of the eye, whose central termination is not definitely known.

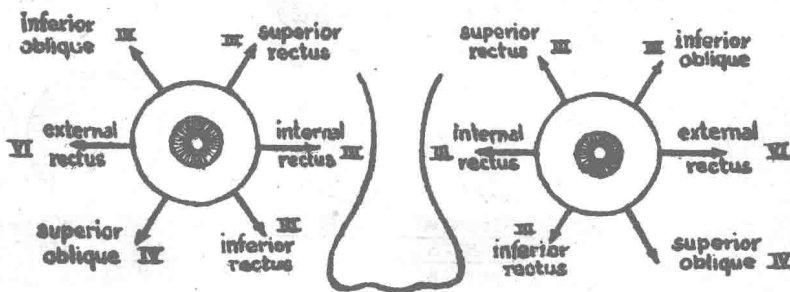
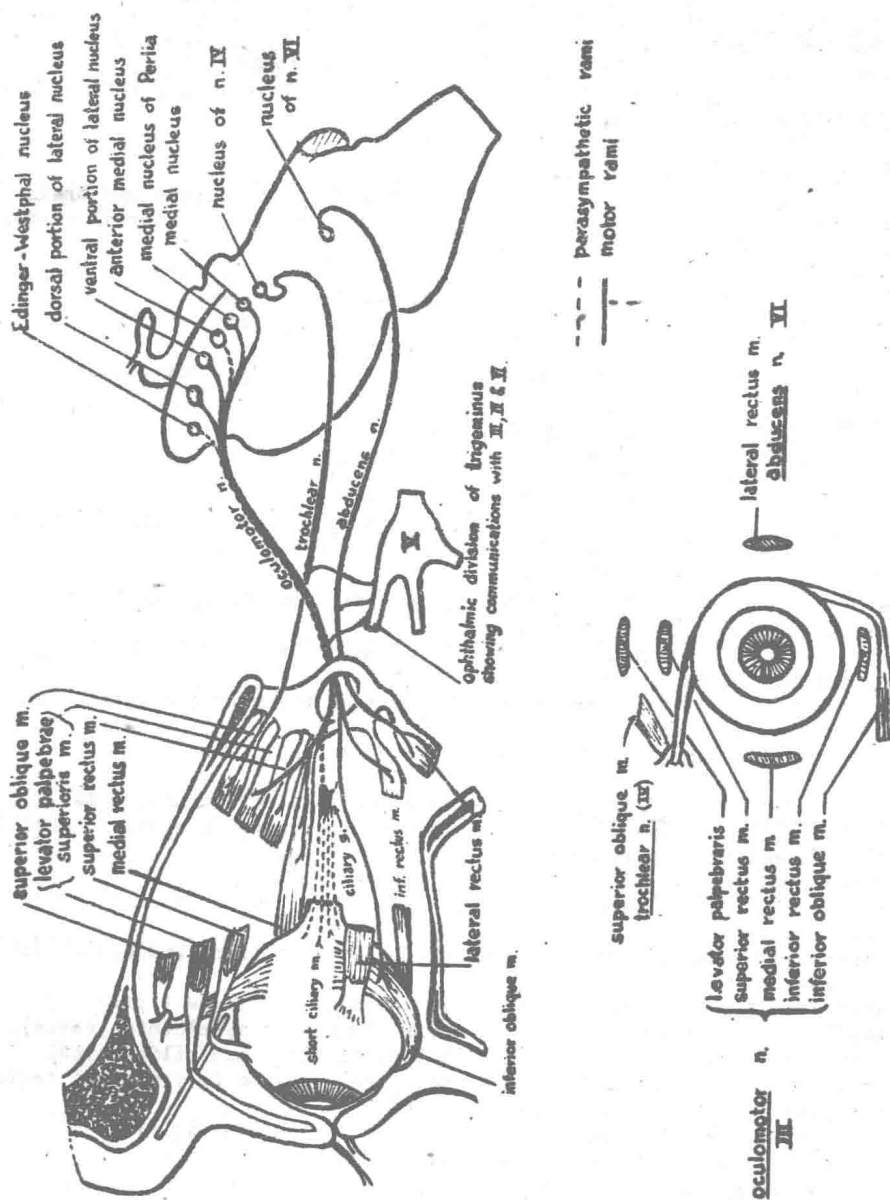


Fig. 4 - DIAGRAM SHOWING THE MOVEMENTS OF THE EYEBALLS BY INDIVIDUAL MUSCLES.  
(This shows direction of movement only and not attachment of muscles)

Fig. 5. THE OCULOMOTOR, TROCHLEAR, AND ABDUCENS NERVES



Central reflex connections -

1. From the pretectal region via the posterior commissure to the Edinger-Westphal nuclei for mediation of ipsilateral and consensual light reflexes. Interruption of this pathway causes the Argyll Robertson pupil.
2. From the superior colliculi via the tectobulbar tract to the nuclei of III, IV, and VI for the modulation of accommodation reflex and others.
3. From the inferior colliculi via the tectobulbar tract to the eye muscle nuclei for reflexes correlated with hearing. From the vestibular nuclei via the medial longitudinal fasciculus for reflex correlation with balance.
4. From the cortex through the corticobulbar tract for mediation of voluntary and conditioned movements of the eyes.

CONDITIONS WHICH MAY INVOLVE THESE NERVES -

- |   |   |
|---|---|
| 1. Syphilis                                   | 9. Tumors of the orbit and brain            |
| 2. Meningitides                               | 10. Aneurysm of the internal carotid artery |
| 3. Encephalitides                             | 11. Cerebral hemorrhage                     |
| 4. Diphtheria toxin                           | 12. Multiple sclerosis                      |
| 5. Botulism                                   | 13. Skull fracture                          |
| 6. Cavernous sinus thrombosis                 | 14. Hysterias                               |
| 7. Polioencephalitis hemorrhagica superior    | 15. Certain drug intoxications              |
| 8. Suppuration of the accessory nasal sinuses |   |

SIGNS - - -to look for in examining these nerves -

1. Squint or strabismus - deviation of either eye or both.
  - a. Internal - visual axes cross each other.
  - b. External - visual axes diverge from each other.
2. Diplopia or double vision - a subjective phenomenon present only when looking with both eyes (monocular diplopia is generally hysterical in nature).
3. Tilting of head to compensate for diplopia.
4. Conjugate deviation - both eyes turned to same side - may be spasmodic or paralytic. Usually from central lesions.
5. Nystagmus - rhythmic or undulating movements of the eyes. May be physiologic or due to lesions, either central or labyrinthian.
6. Ptosis or lid drop - due to weakness or paralysis of the superior levator muscle.
7. Dizziness - often associated with diplopia.
8. Limitations of movement, loss of reflexes, etc. (see TESTS - below).

CLASSIFICATION OF DISORDERS OF THESE NERVES - (See references for further details).A. Ophthalmoplegias - (or paralyses) - lesions causing these may be acute, chronic, progressive, and central or peripheral.1. Oculomotor paralysis (III) -

- a. External ophthalmoplegia - divergent strabismus, diplopia, and ptosis of lid.
- b. Internal ophthalmoplegia - dilated pupil, loss of light and accommodation reflexes.
- c. Paralysis of individual muscles - see chart at top of next page.
- d. Paralysis of levator palpebrae - ptosis (common in myasthenia gravis).
- e. Argyll Robertson pupil - miosis with loss of light and ilio-spinal reflexes. Accommodation reflex is retained - lesion in pretectal region.
- f. Paralysis of convergence - (central lesion).
  - (1) Internal recti normal except they cannot converge the eyes.
  - (2) Also a loss of the associated pupil contraction.

2. Trochlear paralysis (IV) - (rare)

- a. Slight convergent strabismus and diplopia on looking downward.
- b. Cannot look down and out, hence have difficulty in descending stairs.
- c. The head is tilted as a compensatory adjustment and may be the first indication of a trochlear lesion.

3. Abducens paralysis (VI) - (most common of eye palsies - due to long course of nerve)

- a. Convergent strabismus and diplopia.
- b. Especially common in late syphilis, basilar diseases, and trauma.

4. Chronic progressive ophthalmoplegia - (Graefe's disease) - rare. Usually involves all 3 nerves together - caused by nuclear lesion, e.g., bulbar paralysis, late tabes, or progressive muscular atrophy.



### 5. Chart of paralyses of individual eye muscles:

Muscle	Nerve	Deviation of eyeball	Diplopia present when looking*	Direction of image
1. Internal rectus	III	Outward (external squint)	Nasalward	Vertical
2. Superior rectus	III	Downward and outward	Up and in	Oblique
3. Inferior rectus	III	Upward and outward	Down and in	Oblique
4. Inferior oblique	III	Downward and inward	Up and out	Oblique
5. Superior oblique	IV	Upward and inward	Down (and out)	Oblique
6. External rectus	VI	Inward (internal squint)	Temporalward	Vertical

\*Diplopia is noted only when the affected eye attempts these movements.

### B. Myasthenic states -

1. Show difficulty in keeping visual axes parallel without effort.
2. Present with functional, congenital, and neurasthenic weaknesses.
3. Muscular asthenopia - causes visual disturbances, vertigo, migraine, cerebral paresthesia, and pains in the head, especially in the occipital and cervical regions.
4. Spasmodic ocular disorders - produced by supranuclear lesions
  1. Conjugate deviation spasm. (See Dana or Jelliffe and White).
  2. Lateral association spasm (or palsy).
  3. Ventral association spasm (or palsy).
  4. Loss of convergence (q.v. - under 3rd nerve palsies)
  5. Central nystagmus - a. Rhythmic (usually of vestibular origin).  
b. Undulating - usually cerebral or cerebellar.

### SYNDROMES INVOLVING THESE NERVES - (see page 143)

- |                            |                |   |
|----------------------------|----------------|---|
| 1. Benedikt's              | 5. Foville's   | 9. Argyll Robertson pupil (q.v.)        |
| 2. Bulbar palsy            | 6. Korsakoff's | 10. Millard-Gubler                      |
| 3. Gradenigo's             | 7. Nothnagel's | 11. Syndrome of the superior colliculus |
| 4. Graefe's disease (q.v.) | 8. Weber's     | 12. Wernicke's syndrome                 |

### DRUGS AFFECTING THE PUPILS AND CILIARY MUSCLES -

Mydriatics (dilate pupil)	Cycloplegics (relax ciliary muscle)	Miotics (constrict pupil)
1. Atropine	1. Atropine	1. Morphine
2. Homatropine	2. Homatropine	2. Opiates
3. Scopolamine	3. Scopolamine	3. Pilocarpine
4. Cocaine	4. Other atropine derivatives	4. Physostigmine (eserine)
5. Epinephrine		5. Picrotoxin

Mydriatics generally increase intra-ocular tension in glaucoma, miotics relieve it.

### TESTS -

1. Finger following tests for ocular movements.
2. Accommodation - by noting convergence and pupillary change when patient follows objects brought from a distance up close to the eyes.
3. Light reflex - by shining light into eye from side (also depends on optic nerves).
4. Consensual light reflex - by shining light into one eye and noting change in opposite pupil.
5. Prism tests - for ability of internal and external recti to coalesce images.
  - a. Normally externi should overcome a prism of 8° or more.
  - b. Normally interni should overcome a prism of 23° to 25° or more.

### REFERENCES -

1. Dana - Textbook of Nervous Diseases - pages 108-115
2. Jelliffe and White - Diseases of the Nervous System (6th ed.) - pages 384-397
3. Wechsler - Textbook of Clinical Neurology (5th ed.) - pages 259-265
4. Grinker - Neurology (3rd ed.) - pages 345-352
5. Oppenheim - Textbook of Nervous Diseases (5th ed.) - pages 465-472
6. Brain - Diseases of the Nervous System (2nd ed.) - pages 145-150
7. Gifford - A Textbook of Ophthalmology - pages 361-401



ANATOMY -

A. Motor - fibers from the motor nucleus of V at the mid-level of the pons pass as the motor root (portio minor) from the ventral surface of the pons through the foramen ovale to supply:

1. The muscles of mastication - (the masseter, temporal, internal and external pterygoids), and via the otic ganglion to supply:
2. The tensor tympani and tensor veli palatini, and via the mylohyoid nerve to
3. The mylohyoid muscle and anterior belly of the digastric muscle.

B. Sensory - fibers arise from unipolar cells in the semilunar (Gasserian) ganglion, Peripherally they supply sensation via the:

1. Ophthalmic division - to the forehead, eyes, nose, temples, meninges, paranasal sinuses, and part of the nasal mucosa.
2. Maxillary division - to the upper jaw, teeth, lip, cheeks, hard palate, maxillary sinuses, and nasal mucosa.
3. Mandibular division - to the lower jaw, teeth, lip, buccal mucosa, tongue and part of the external ear, meatus, and meninges.

Centrally - the fibers pass as the portio major and split into

1. Short ascending rami ending in the main sensory nucleus of V (just lateral to the motor nucleus) and subserving mainly touch.
2. Long descending rami giving off collaterals to the spinal nucleus of V which extends through the medulla to overlap with Lissauer's tract; these fibers carry sensation of touch, pain, and temperature.

C. Sensory - proprioceptive fibers arise from unipolar cells within the mesencephalic nucleus of V; the peripheral processes pass via the motor root to nerve spindles in the muscles of mastication, and possibly also to the extra-ocular muscles.

Central connections -

- A. The motor nucleus receives bilateral (mainly crossed) cerebral connections from the corticobulbar tracts, reflex connections from the spinal tract of V and extrapyramidal tracts.
- B. From the main sensory nucleus of V - touch pathways pass to the thalamus and higher centers via the dorsal secondary tract of V.
- C. From the spinal nucleus of V - touch, pain, and temperature pathways pass:
  1. To the thalamus via the ventral secondary tract of V and
  2. Reflex connections pass to the motor nuclei of cranial nerves V, VII, and IX.
- D. Central connections of the mesencephalic nucleus are obscure.

CONDITIONS AFFECTING THE TRIGEMINAL NERVE -

- |                            |                        |                                |
|----------------------------|------------------------|--------------------------------|
| 1. Neuralgias and neuritis | 5. Tumors of the brain | 9. Carotid aneurysm            |
| 2. Syphilis                | 6. Basilar meningitis  | 10. Hysteria                   |
| 3. Tuberculosis            | 7. Pentile diseases    | 11. Cavernous sinus thrombosis |
| 4. Syringobulbia           | 8. Skull fracture      |                                |

SYMPTOMS OF TRIGEMINAL INVOLVEMENT -

1. Pain - marked if Gasserian ganglion or peripheral branches are involved.
2. Loss of sensation - over sensory distribution - corneal anesthesia early.
3. Dissociate anesthesia - loss of pain but not touch may be noted when the spinal tract of V is involved (e.g., in syringobulbia).
4. Paresthesia - occasionally seen in anemia and in nervous and hysterical patients.
5. Paralysis of muscles of mastication - with deviation of jaw to affected side.
6. Loss of jaw jerk, sneeze, lid, conjunctival and corneal reflexes.
7. Impaired hearing - from paralysis of tensor tympani.
8. Trismus (lock jaw) - tonic spasm of muscles of mastication - in rabies, tetany, tetanus, epilepsy, and hysteria.
9. Trophic and secretory disturbances - herpes, neurokeratitis, dryness of nose (this causes anosmia as moisture is necessary to smell), ulcerations of face, and the teeth may fall out.