

Neuro-ophthalmology

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Neuro-ophthalmology

- Study integrating ophthalmology and neurology
- Disorders affecting parts of CNS devoted to vision or eye:
- Afferent system (visual pathway, incl. optic nerve)
- Efferent system (ocular motor control, pupillary function)

Part I

Neuro-ophthalmologic Examination

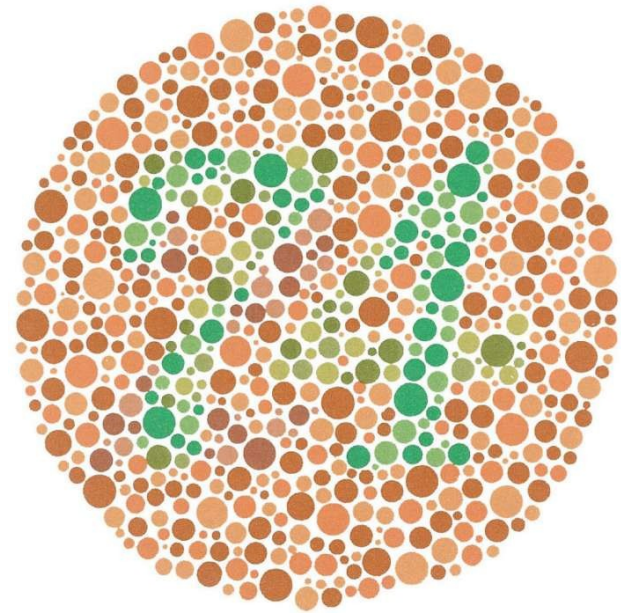
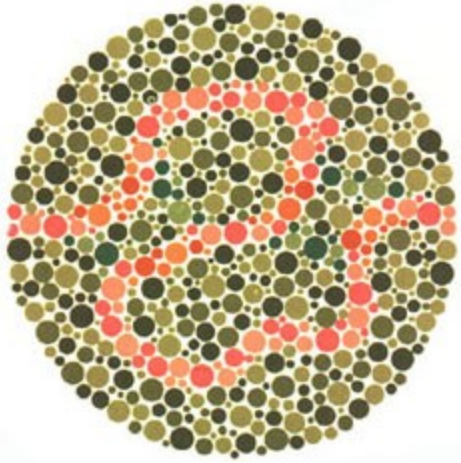
Examination

- History
- Eye examination (visual acuity, tonometry, anterior segment examination, funduscopic examination)
- Perimetry
- Color vision, contrast sensitivity, electrophysiology (ERG, VEP)
- MRI of brain,
- Neurologic examination

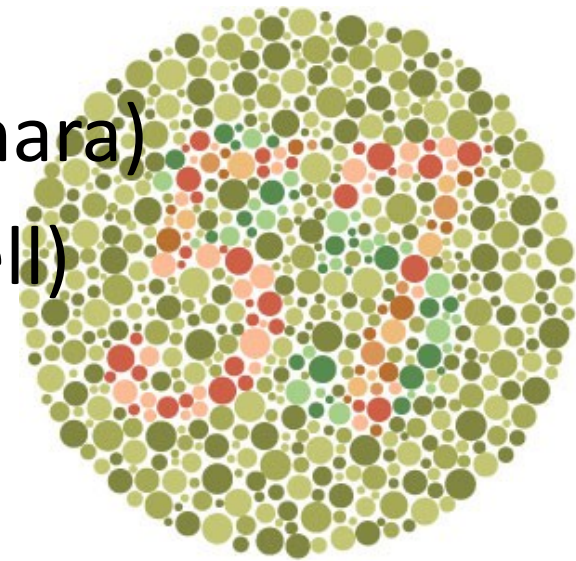
Visual acuity

- Each eye separately
- Distance and near vision
- Using of corrective lenses, pinhole
- Using Snellen chart (20 feet) – normal 20/20
- Count fingers, hand motion, light perception, no light perception

Color vision

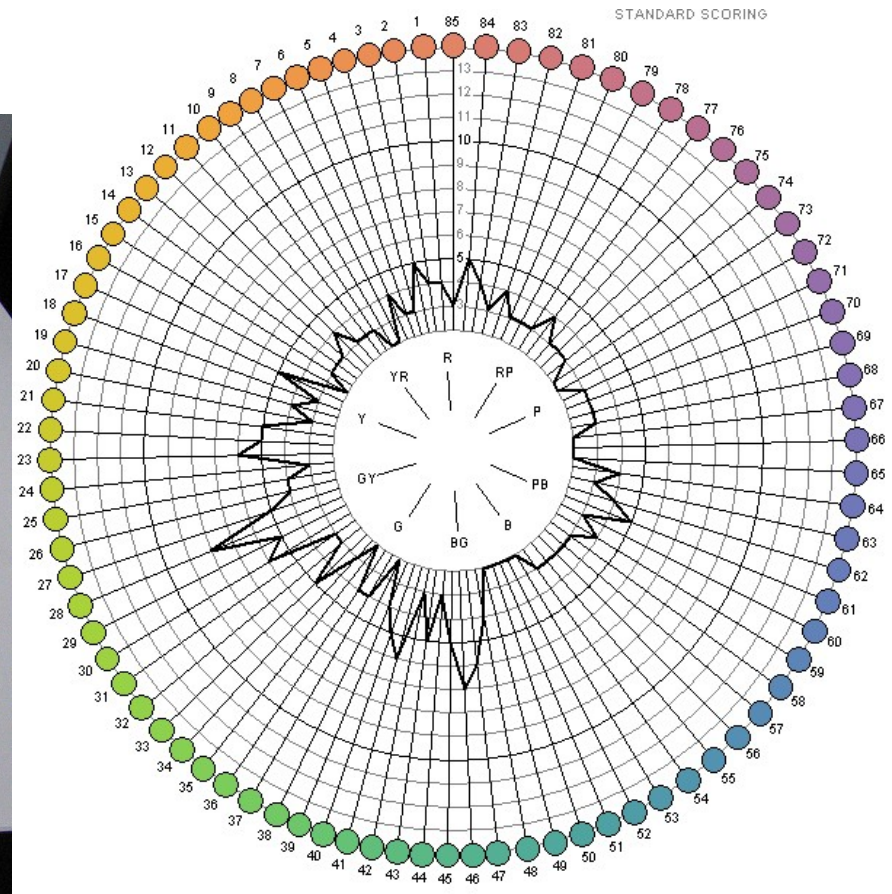
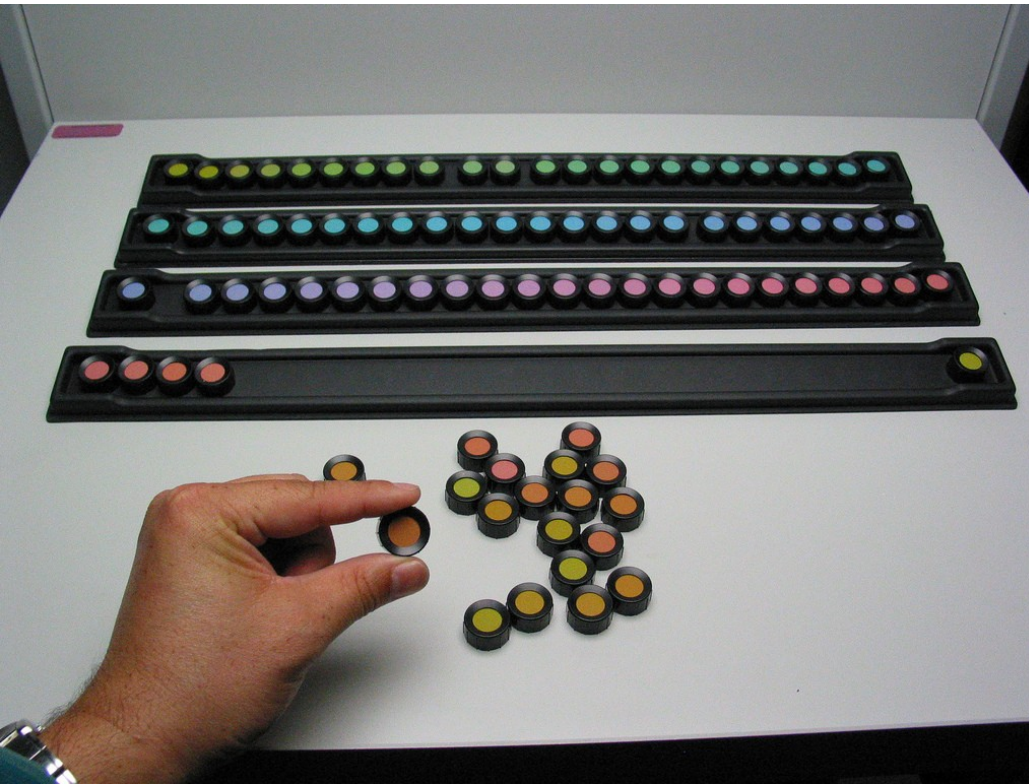


- Each eye separately
- Comparison between eyes
- Examination:
- **pseudoisochromatic plates (Ishihara)**
- **100 Hue test (Farnsworth-Munsell)**



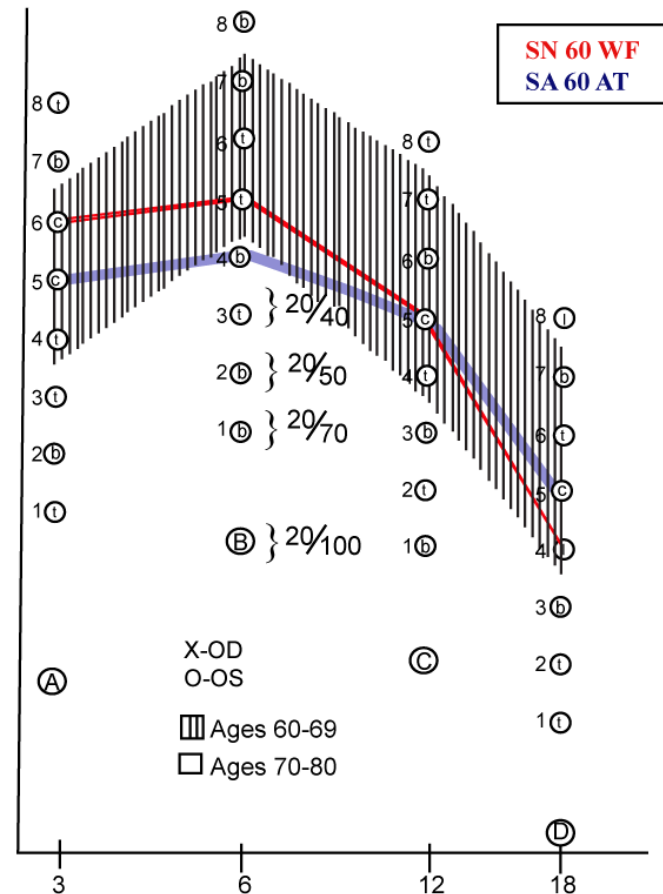
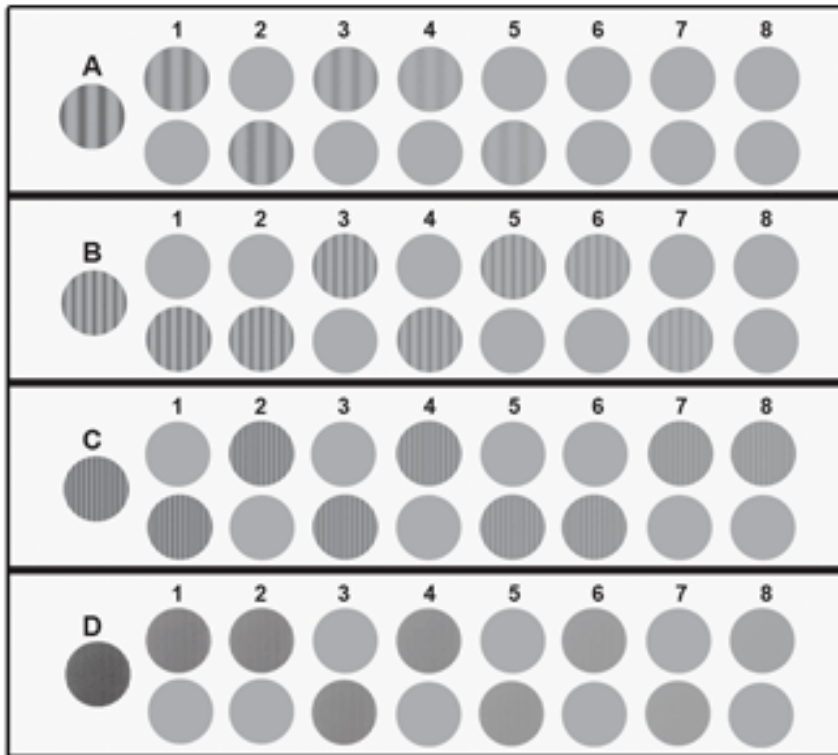
Farnsworth-Munsell 100 Hue test

- Ordering the color tiles as patient sees it



Contrast sensitivity

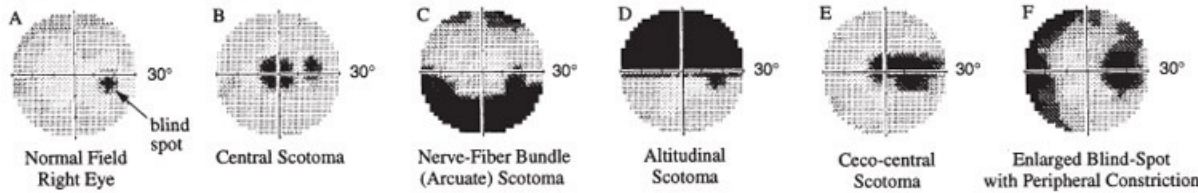
- Examining spatial frequency
- Decreased in some optic nerve disorders (typically optic neuritis)



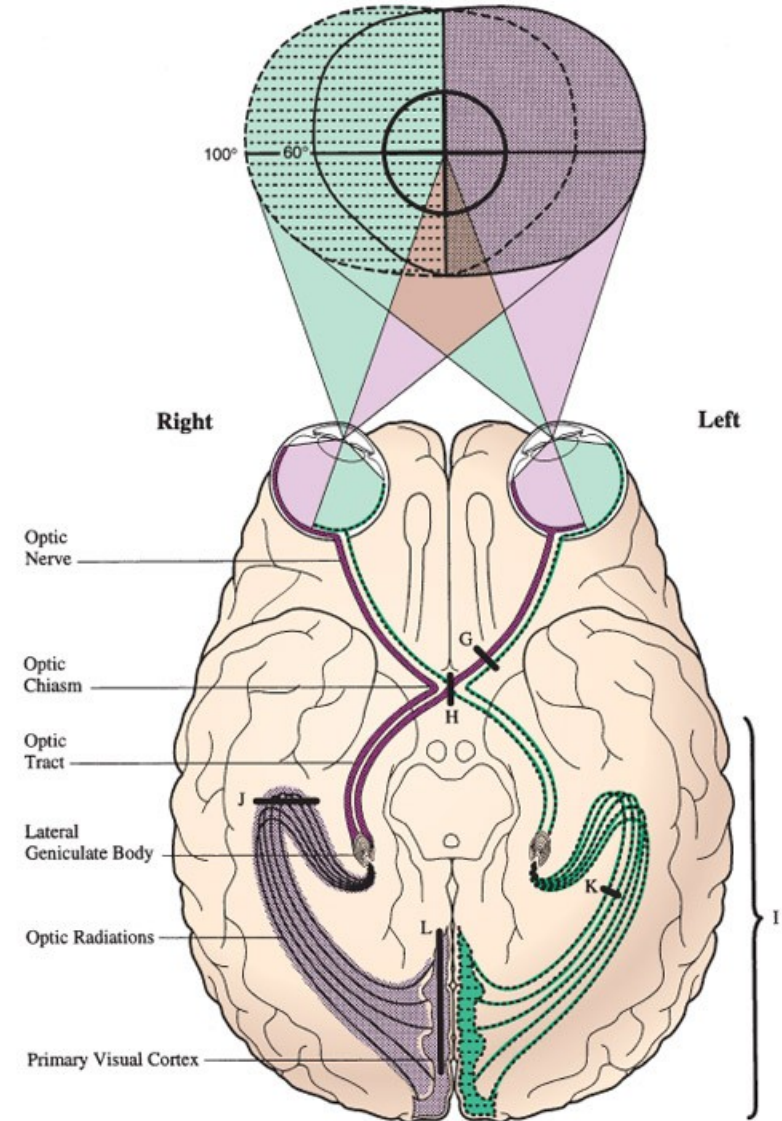
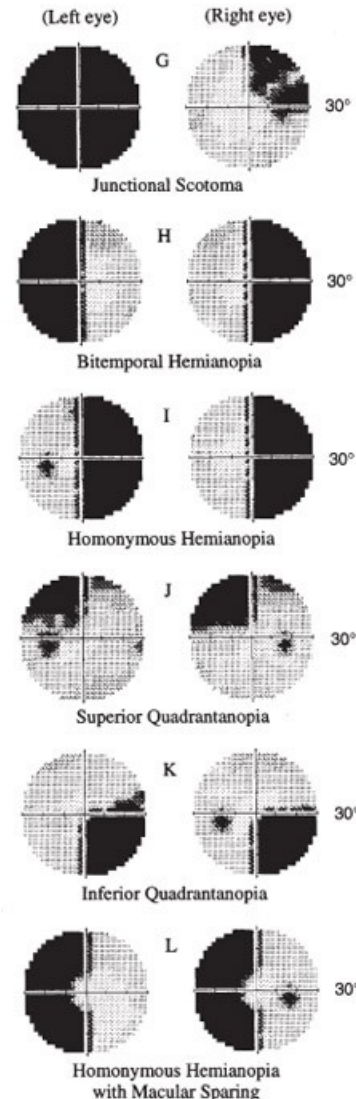
Perimetry

- To assess the quality of visual field
- Characteristic visual field defect = location of possible intracranial lesions

Monocular Prechiasmal Field Defects:

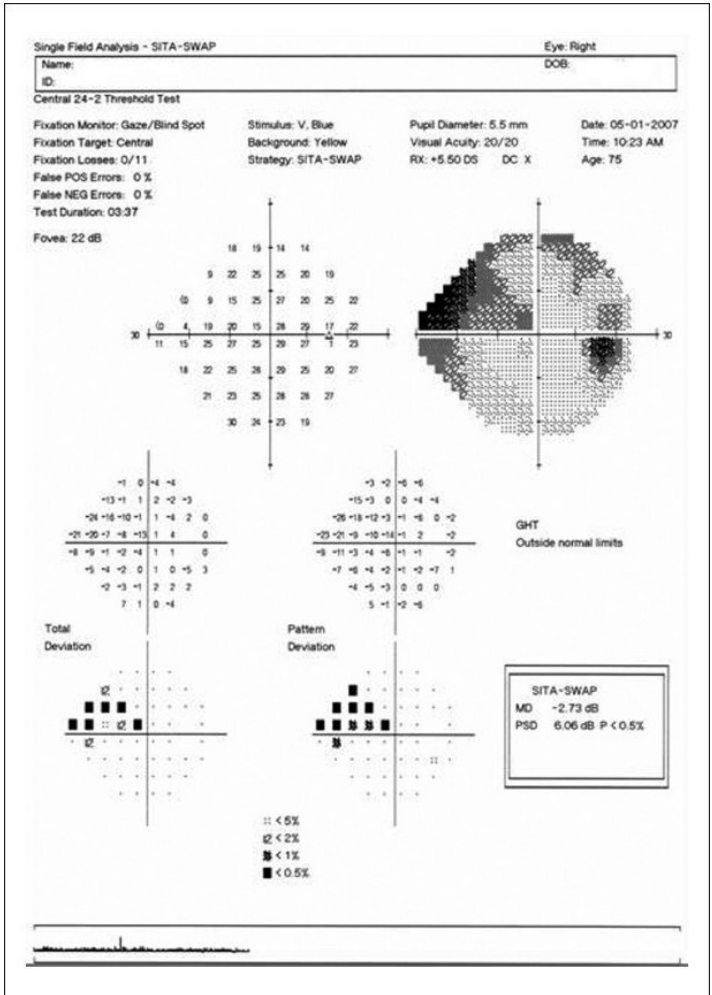
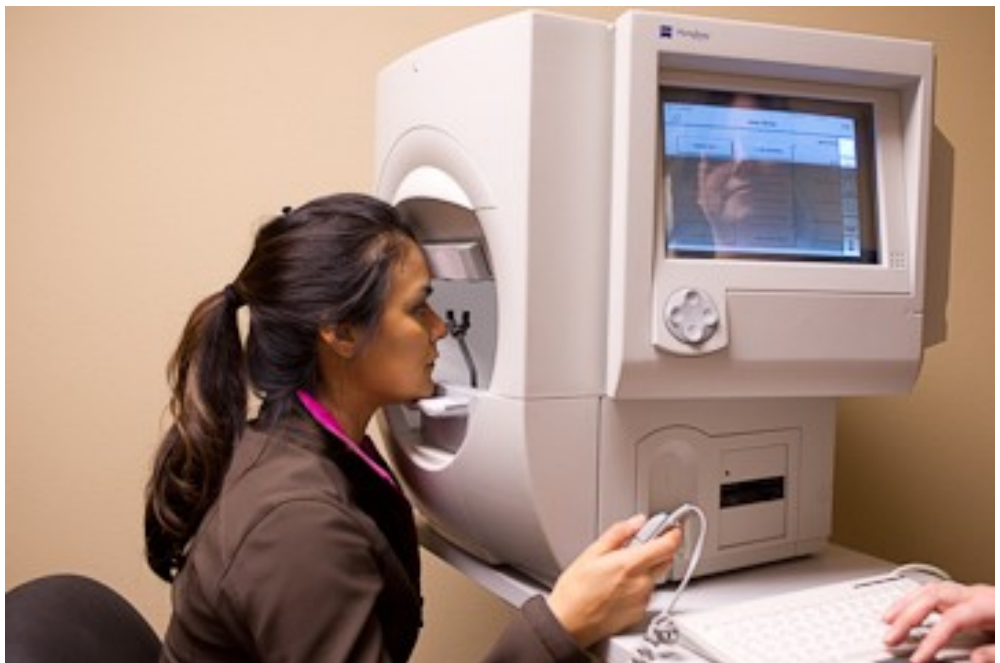


Binocular Chiasmal or Postchiasmal Field Defects:



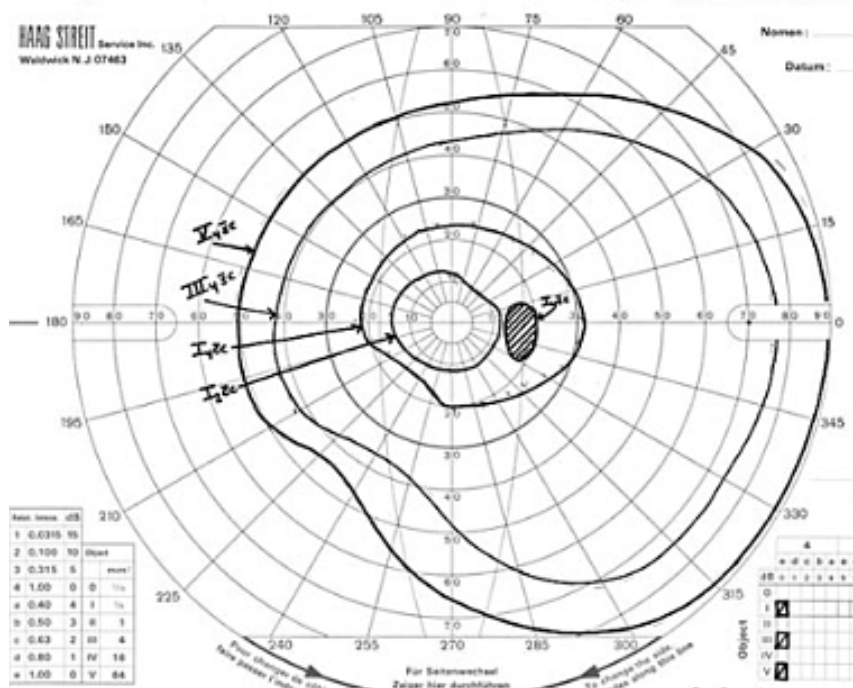
Perimetry

- Automated static perimetry



Perimetry

- Goldmann kinetic perimetry



Electrophysiologic examination

ERG = Electroretinography

- Access possible functional pathology of retina (scotopic, photopic and central part)
- **Flash ERG** (activity of bipolar cells as an answer to stimulation of photosensitive cells – rods, cones)
- **Pattern ERG** (activity of ganglionar cell as a response to stimulation of cones in macula)

VEP = Visual evoked potentials (responses)

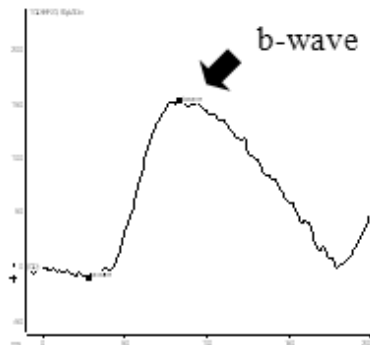
- Access the capability of anterior visual pathways – optic nerve
- Major use: diagnosis/confirm of optic neuritis

Electrophysiologic examination



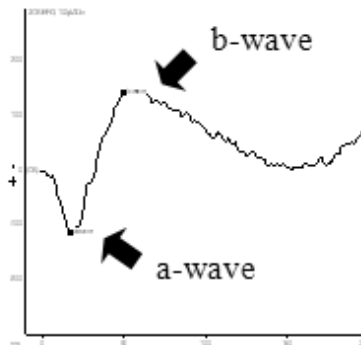
Electroretinography

Rod Specific



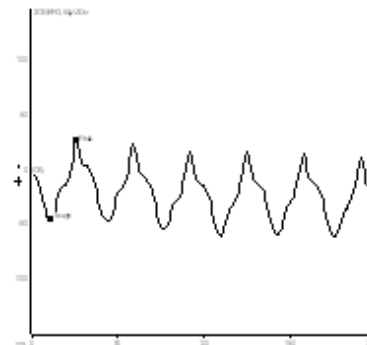
Rod Function

Maximum Scotopic



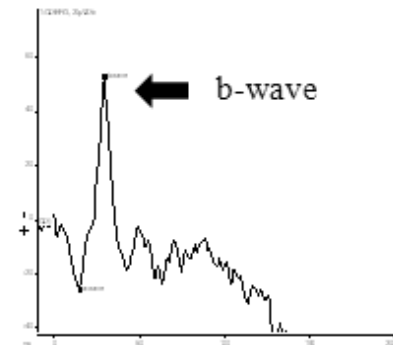
Mixed Rod and Cone Function

Photopic 30 Hz Flicker



Cone Function

Transient Photopic

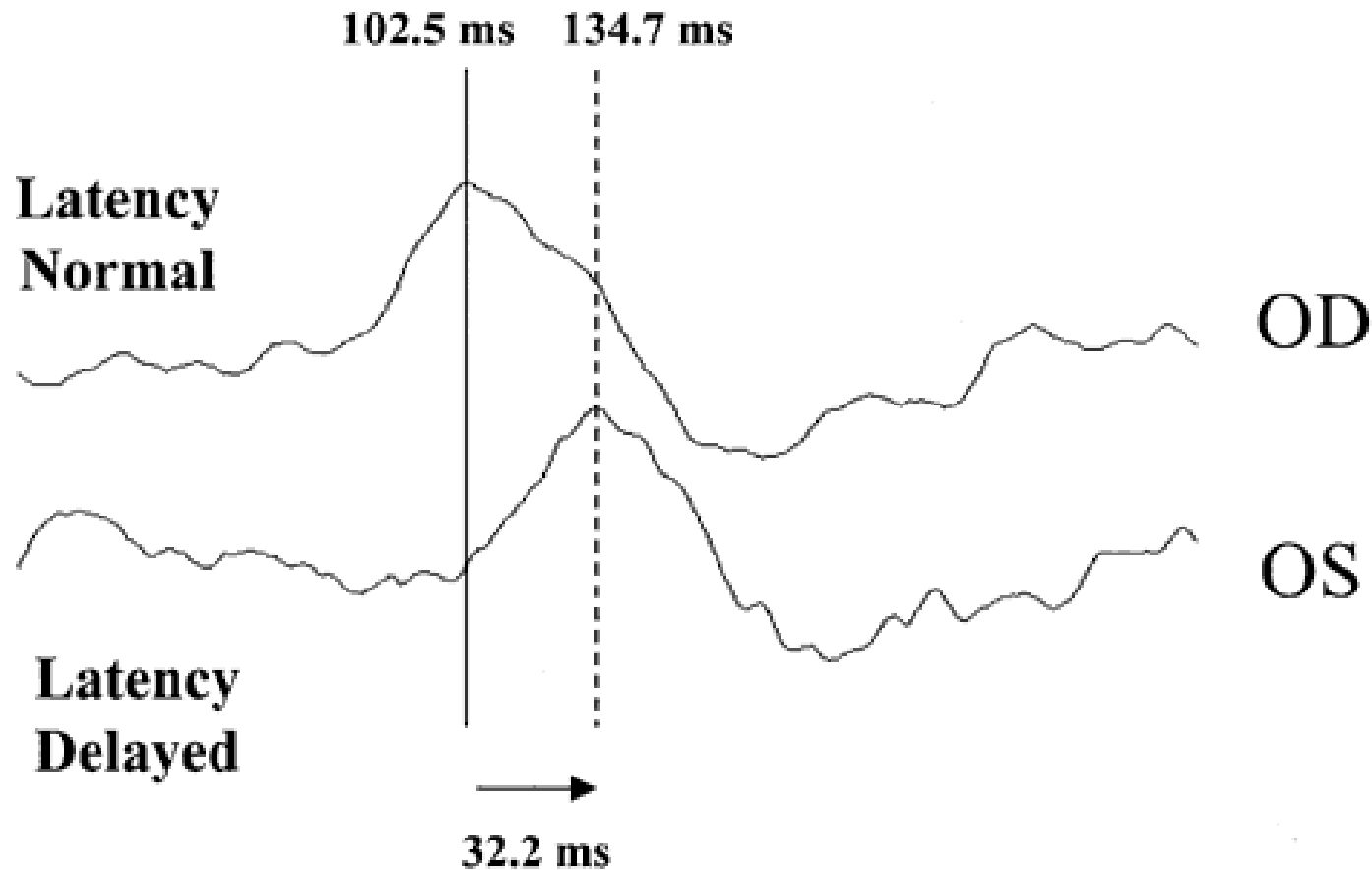


Cone Function

Normal

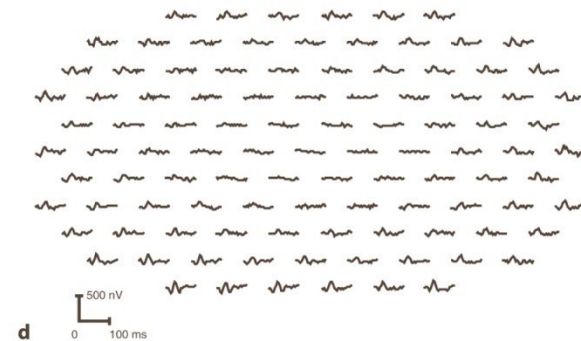
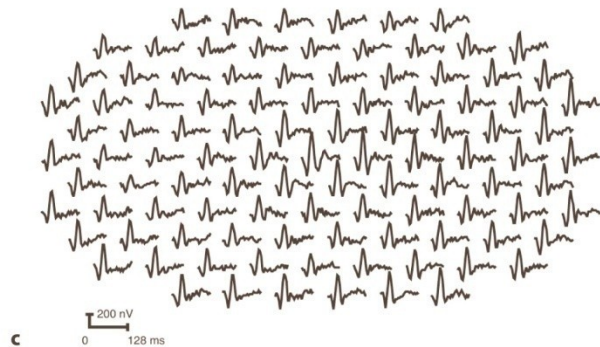
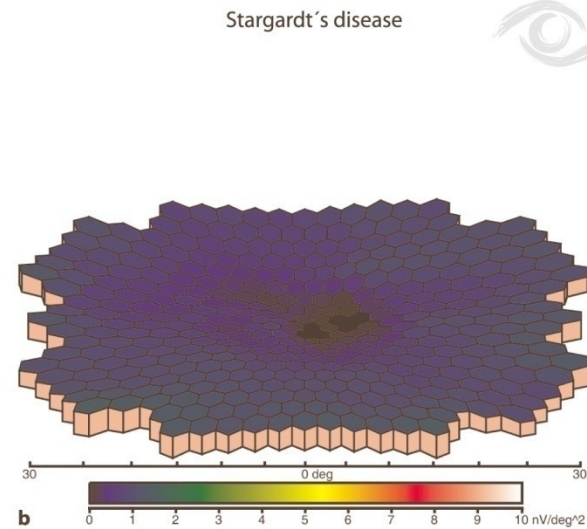
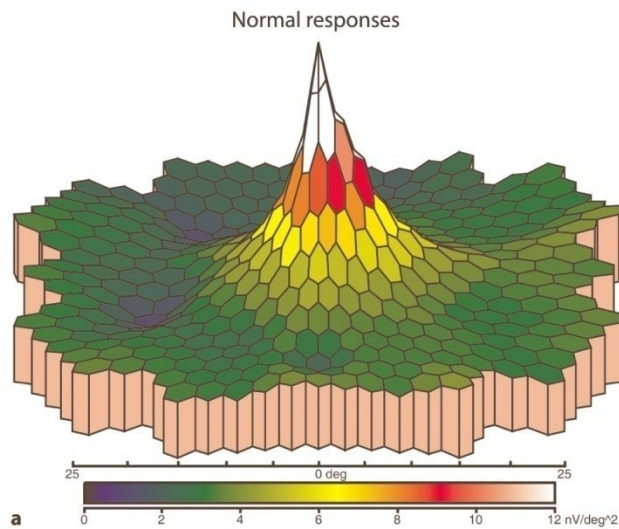
Visual evoked potentials

Pattern-Reversal VEP
15' checks, 3.8 reversals/sec



Multifocal ERG, Multifocal VEP

- Mostly experimental use, not standard in clinical medical practice here

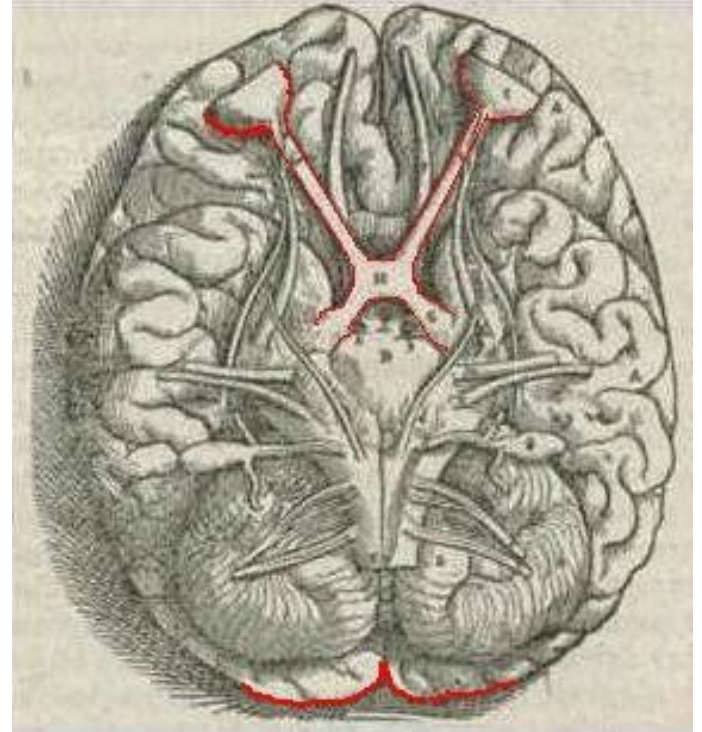


Part II

Pathology of Afferent system

Afferent system

- **Retina** (cones, rods, bipolar and ganglion cells)
- **Optic nerve**
- **Optic chiasm**
- **Optic tract**
- **Lateral geniculate body**
- **Optic radiation**
- **Visual cortex** (V1 = Brodmann area 17)



Pathologies of Afferent Visual System

- Papilledema
- Optic Neuritis
- Optic Neuropathy
- Optic Atrophy

Papilledema

- Not a disease - sign secondary due to elevated intracranial pressure (ICP)
- Unspecific sign
- Require immediate diagnosis = increased ICP is a life-threatening situation!!!
- 60% of cases = increased ICP caused by intracranial tumor!!!
- Other possible causes: hydrocephalus, meningitis, encephalitis, brain abscess...

Papilledema

Clinical picture

Early

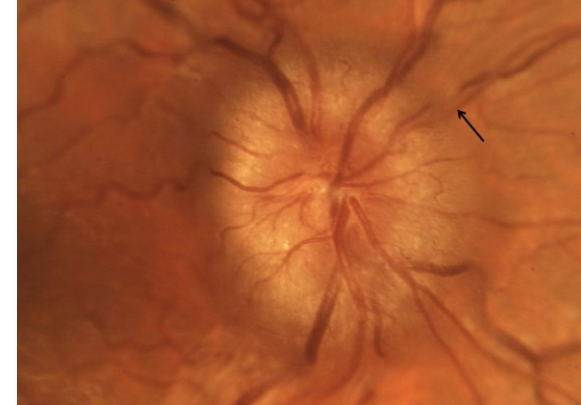
- Margins are obscured
- Optic cup initially preserved
- Hyperemic disc

Acute

- Elevation of disc
- Radial hemorrhages
- Grayish-white exudates

Chronic

- Disc edema
- Obliterated optic cup



Optic neuritis

- Inflammation of the optic nerve
- **Intraocular** – within the globe
- **Retrobulbar** – posterior to the globe
- Usually unilateral
- Tendency to repeat

Etiology

- Often associated with multiple sclerosis (MS) = demyelinating optic neuritis (20% = first sign of MS)
- Other possible inflammatory causes: Lyme disease, syphilis, inflammation from orbit, paranasal sinuses...

Optic neuritis

Symptoms

- Sudden vision loss within several hours (mild blurring/light perception)
- Central, paracentral scotoma
- Retrobulbar/parabulbar pain
- Present afferent pupillary defect

Prognosis

- depends on underlying disorders
- MS = usually good – significant spontaneous improvement (several weeks)
- Some permanent disturbances of vision are possible (color vision decreasing, scotoma)

Anterior Ischemic Optic Neuropathy

Etiology

- Acute disruption of blood supply (due to vascular changes, infarction)

Symptoms

- Sudden unilateral loss of vision
- Altitudinal or wedge-shaped visual field defect
- Present afferent pupillary defect

Clinical picture

- Edema of optic disc
- Segmental obscuration of margins (correlation with visual field defect)

Anterior ischemic optic neuropathy

- **2 forms**
- Benign: **Nonarteritic AION**
- Malign: **Arteritic AION**



Arteritic AION

- Association with systemic vasculitis (giant cell arteritis)
- Diagnosis: sedimentation rate, biopsy of temporal artery
- High risk of affection of contralateral (fellow) eye within days/ weeks!!!
- Need for immediate therapy with high dose intravenous corticoids!!!

AION forms

	Arteritic form	Non-arteritic form
% of cases AION	10 %	90%
age	70 years	60 years
Sex	Female > male	Female = male
Systemic disease association	Giant cell arteritis (Horton disease)	idiopathic
Prognosis	Very rare	mild
Fellow eye affection	often (50-90%)	rare (10-20%)
Diagnostics: Sedimentation (FW)	Very high	normal
treatment	High dosage of systemic corticoids	Not available

Optic Atrophy

- Irreversible loss of axons as a result to damage of optic nerve

Etiology

- **Primary** due to trauma, direct pressure by tumor
- **Secondary** due to affection of optic nerve (optic neuritis...)
- **Glaucomatous** due to glaucomatic damage

Pathogenesis

- **Ascending** - lesion located anterior to the lamina cribrosa
- **Descending** – lesion located posterior to the lamina cribrosa

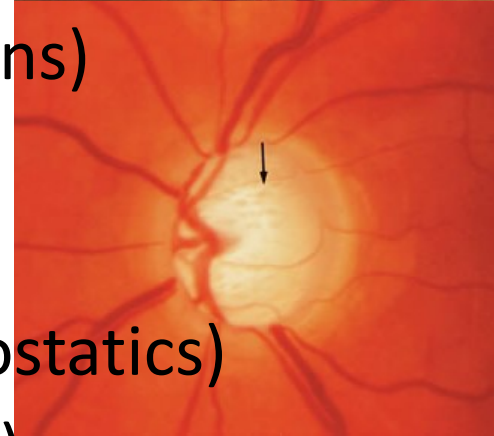
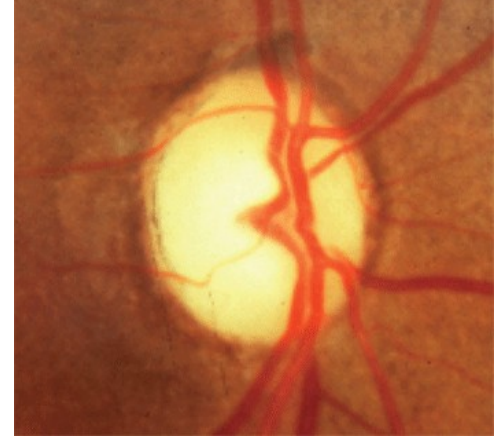
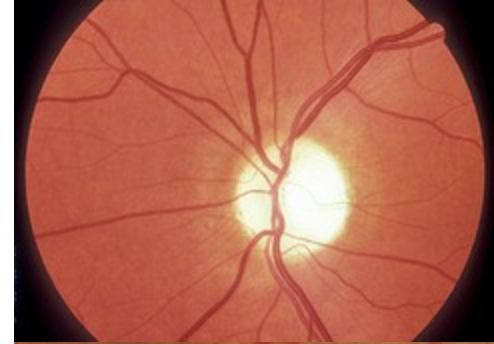
Optic Atrophy

Clinical picture

- Total/partial pale optic disc
- Well defined / blurred margins
- Constricted / reduced retinal vessels

Etiology

- Vascular (AION, RAO)
- Inflammation (optic neuritis, neuroinfections)
- Compressive (orbital/intracranial mass)
- Traumatic (avulsion, bone fracture)
- Toxic (methyl alcohol, various poisons, cytostatics)
- Congenital/hereditary (LHON, Kjer atrophy)
- Systemic (hematooncological diseases)



Part III

Pathology of Efferent system

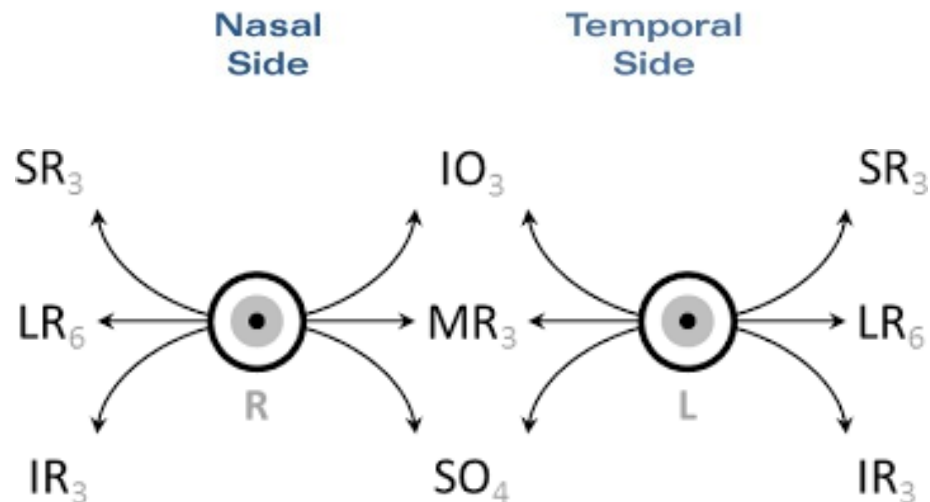
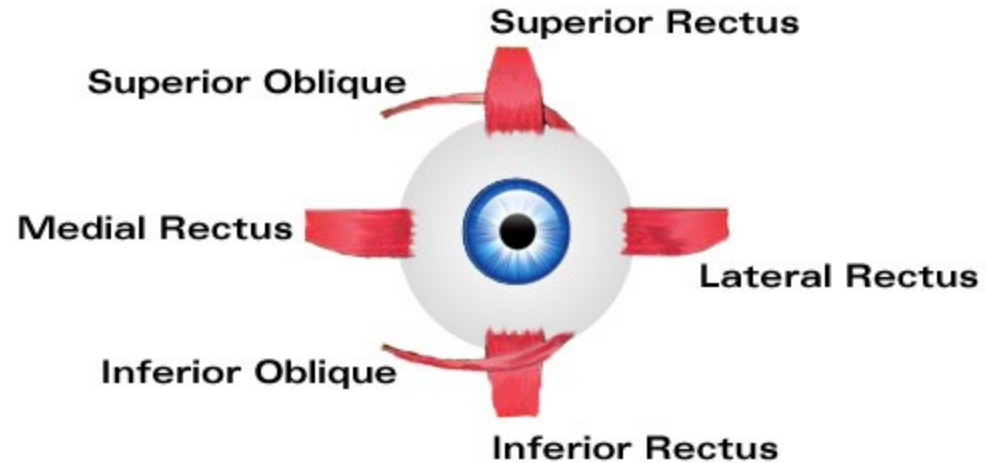
Efferent system

- 1) Cranial neuropathies (III, IV, VI)
- 2) Pupillary abnormalities

Eye movement

- Ocular motility – produced by extraocular muscles
- 4 rectus muscles (lateral, medial, superior, inferior)
- 2 oblique muscles (superior, inferior)

**Extraocular Muscles
(Left Eye)**



Cranial neuropathies

Signs

Oculomotor nerve palsy

- Diplopia
- Multiple muscle paralysis
- Ptosis
- Anisocoria

Trochlear nerve palsy

- Vertical diplopia
- Abnormal head tilt

Abducens nerve palsy

- Horizontal diplopia in the gaze palsy

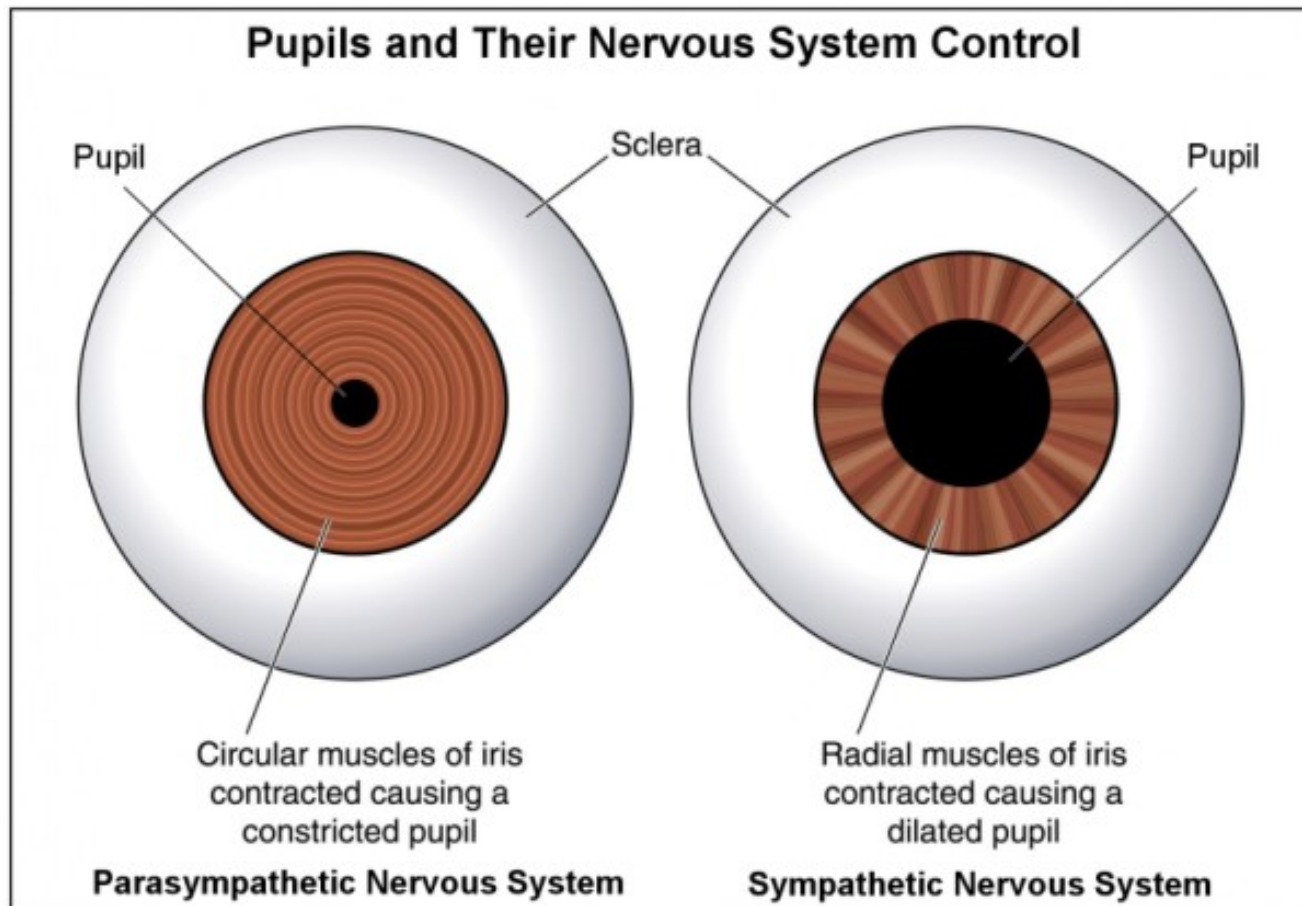
Cranial neuropathies

Etiology

- Ischemic (diabetes, hypertension, hyperlipidemia)
 - Demyelinating disease (MS)
 - Compressive (tumor, aneurysm)
 - Elevated ICP
-
- Multiple cranial neuropathies = suspect lesion in the posterior orbit or cavernous sinus region

Pupil

- **Miosis** – parasympathetic nervous system
- **Mydriasis** – sympathetic nervous system

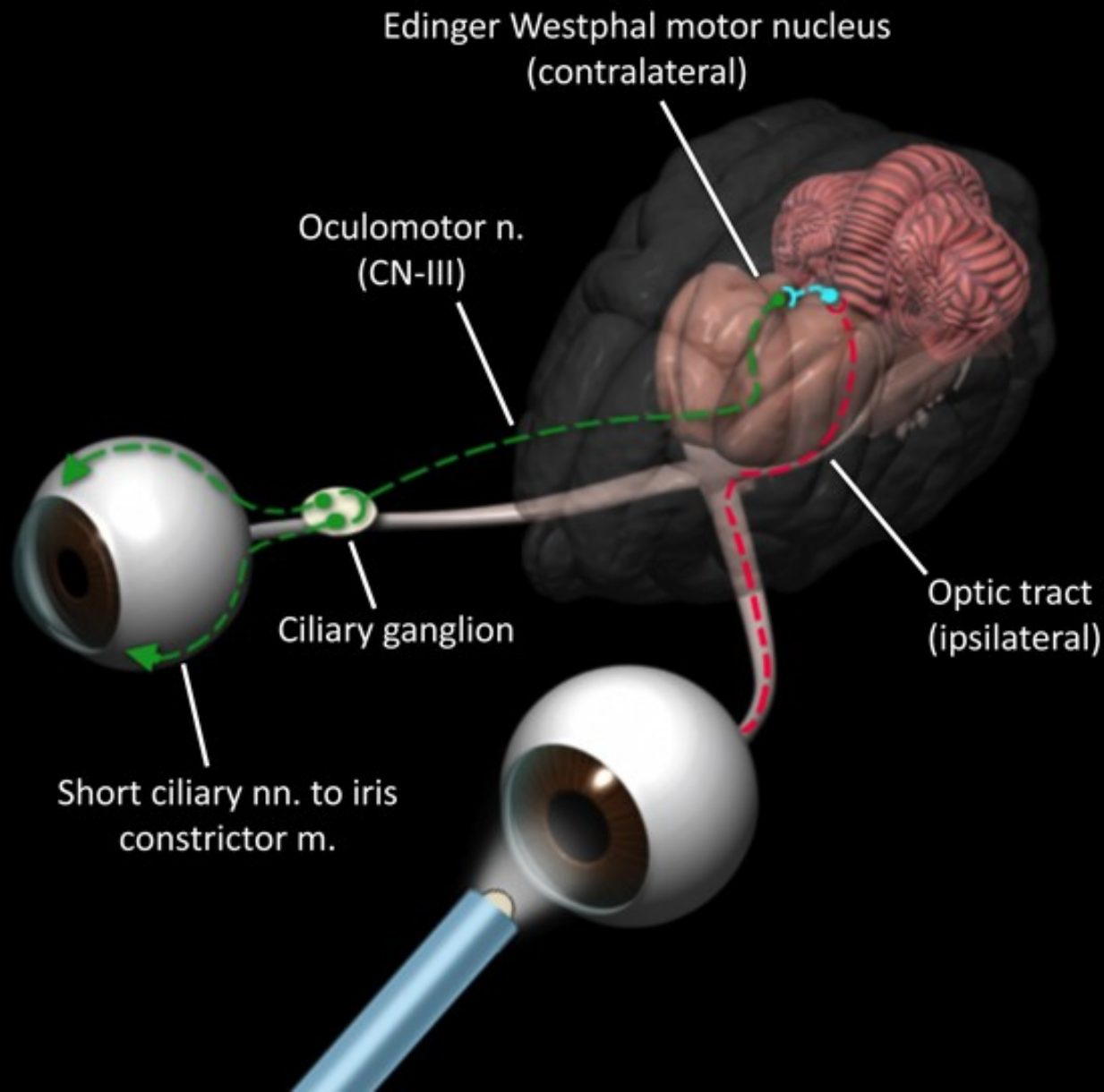


PARASYMPATHETIC CONSENSUAL PATHWAY

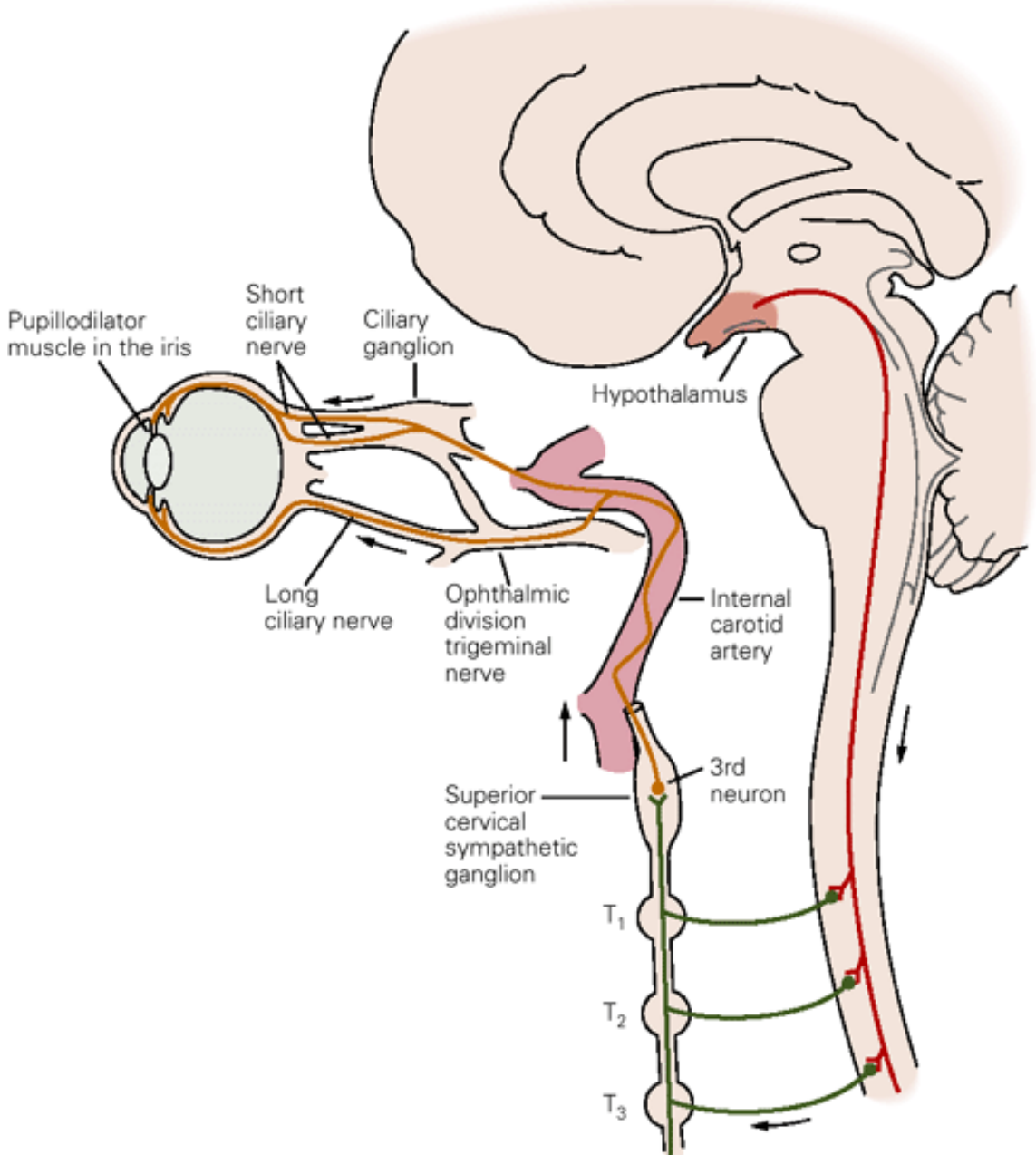
Afferent: CN-II

Interneuron

Efferent: CN-III



Sympathetic pathway



Pupillary abnormalities

Anisocoria

- inequality of pupil size
- May be physiologic
- Possible accidental discovery
- May be isolated / associated with eyelid or ocular motility abnormalities

Diagnosis

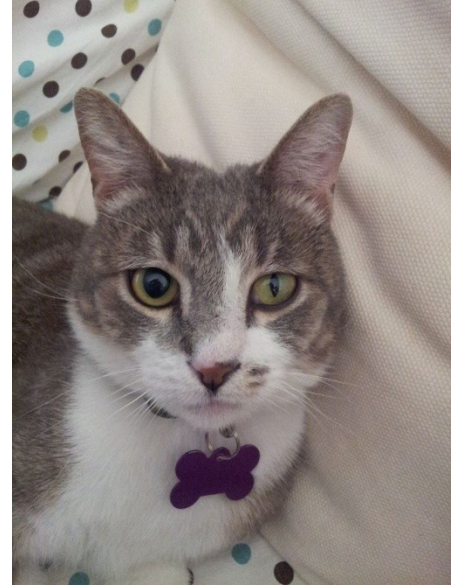
- Direct shine at pupil
- Test near response (miosis with accommodation)
- Pupil sizes in light and dark



Horner's Syndrome

Signs

- Miosis (pupil does not dilate in dark)
- Ptosis
- Pseudo-enophthalmus
- Anhidrosis (diminished sweating)
- Heterochromia (if congenital)



Etiology

- Trauma, internal carotid artery dissection, brain stem strokes, MS, brain tumor, syringomyelia, apical lung tumor, goiter, thyroid carcinoma...



Adie's Pupil

Signs

- No present / slow miosis to light
- Present miosis to accommodation
- Pupil is larger with light/near dissociation

Etiology

- Inflammation (viral or bacterial infection)

Therapy

- Pilocarpine drops, thoracic sympathectomy

Thank you for your attention!