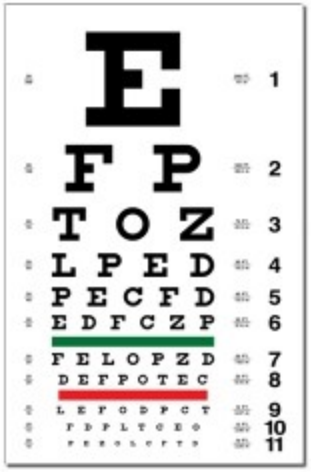
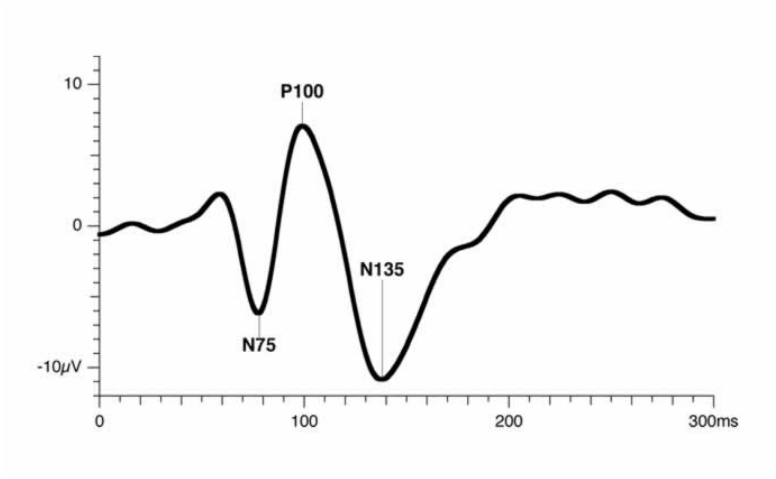
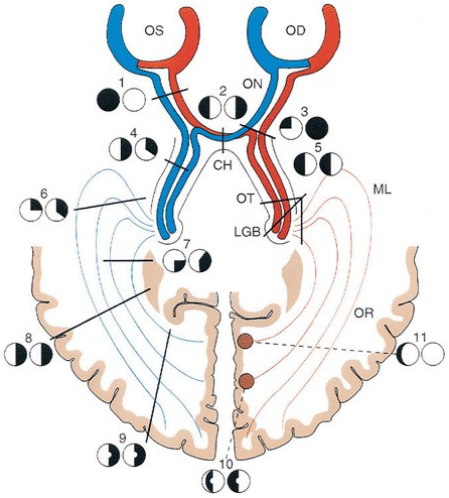
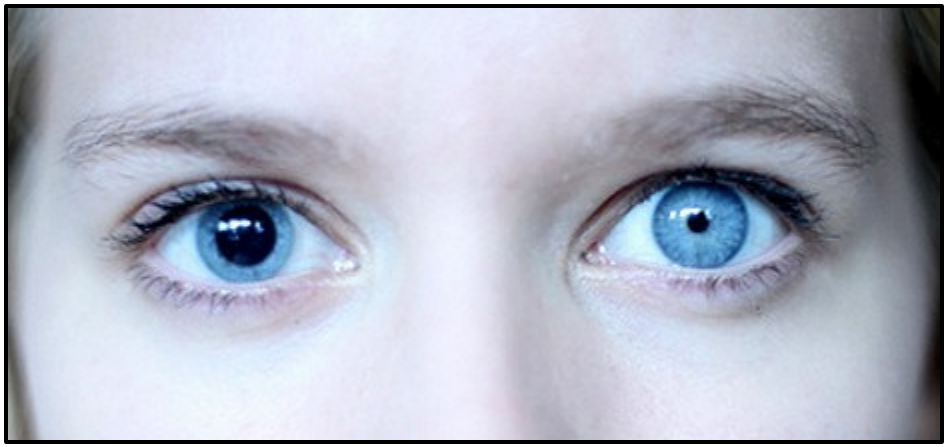






Neuro-ophthalmology



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

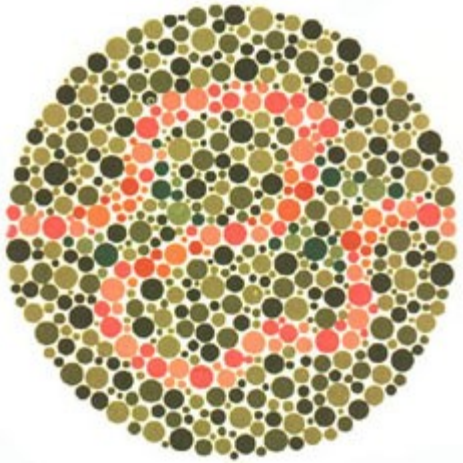
Neuro-ophthalmologic Examination

Examination

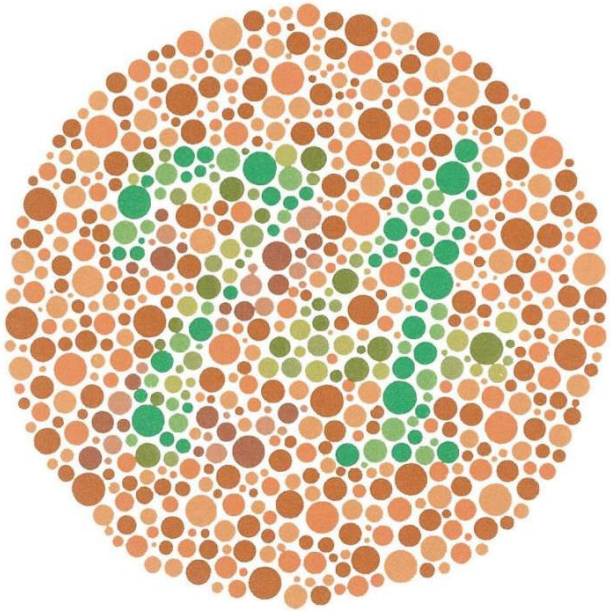
- History
- Eye examination (visual acuity, tonometry, anterior segment examination, funduscopic examination)
- Perimetry
- Color vision, contrast sensitivity, electrophysiology (ERG, VEP)
- MRI of brain,
- Neurologic examination

Visual acuity

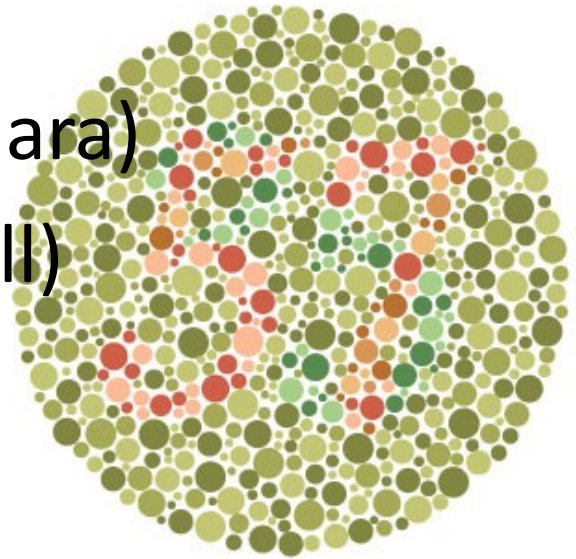
- Each eye separately
- Distance and near vision
- Using of corrective lenses, pinhole
- Using Snellen chart (20 feet) – normal 20/20
- Count fingers, hand motion, light perception, no light perception



Color vision

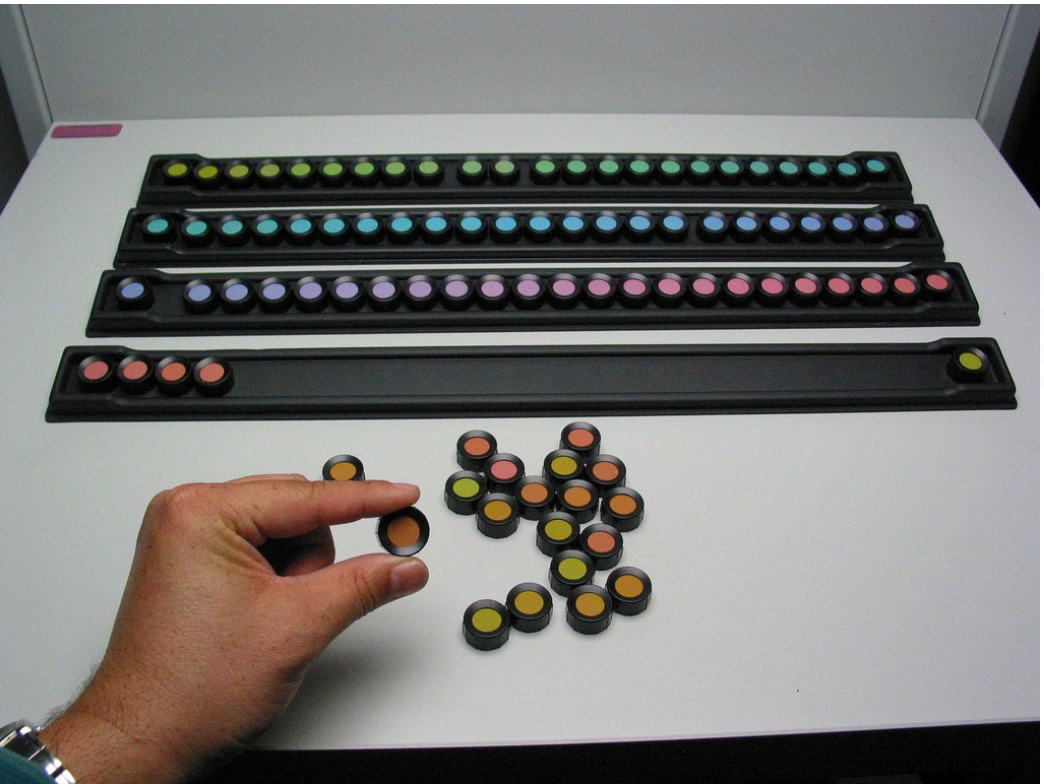


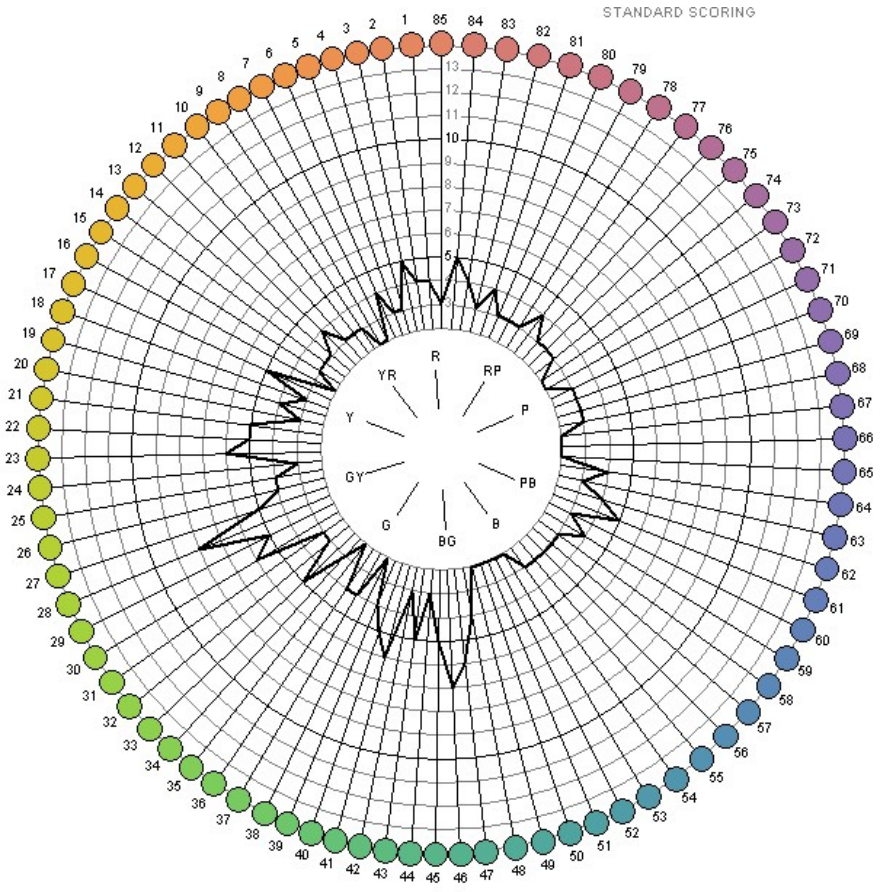
- Each eye separately
- Comparison between eyes
- Examination:
- **pseudoisochromatic plates** (Ishihara)
- **100 Hue test** (Farnsworth-Munsell)



Farnsworth-Munsell 100 Hue test

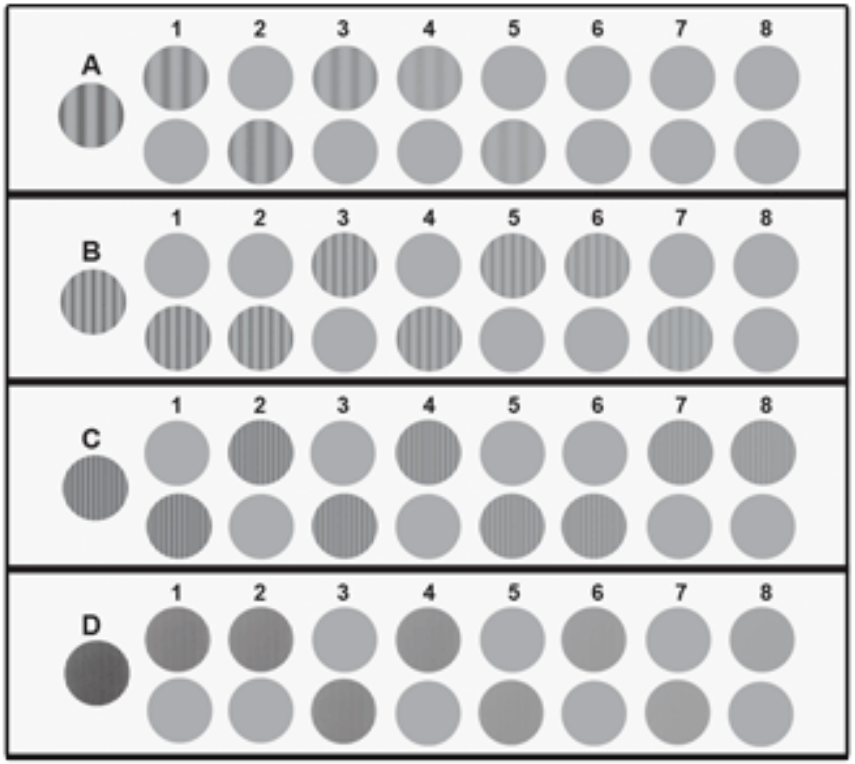
- Ordering the color tiles as patient sees it

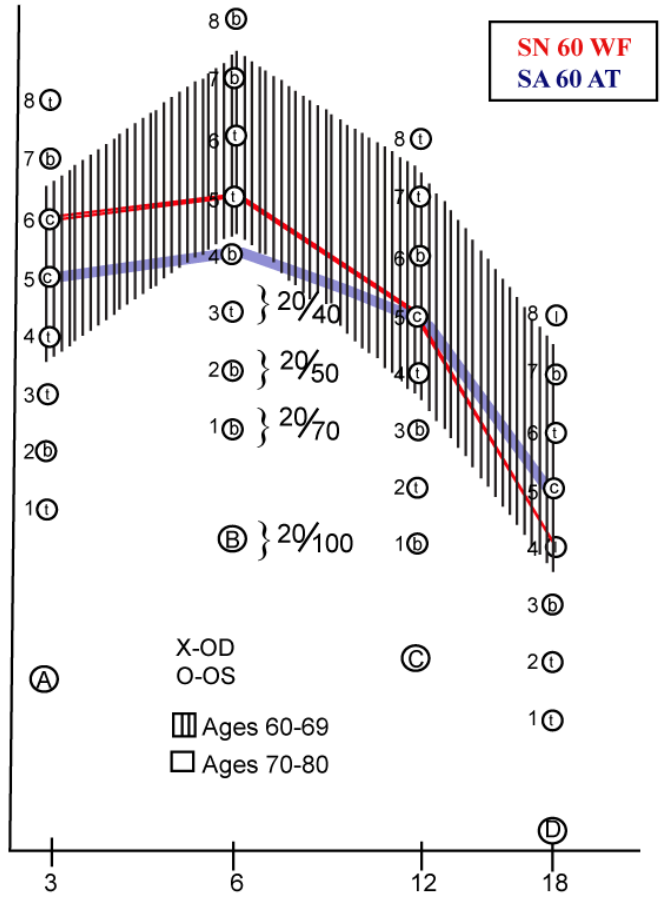




Contrast sensitivity

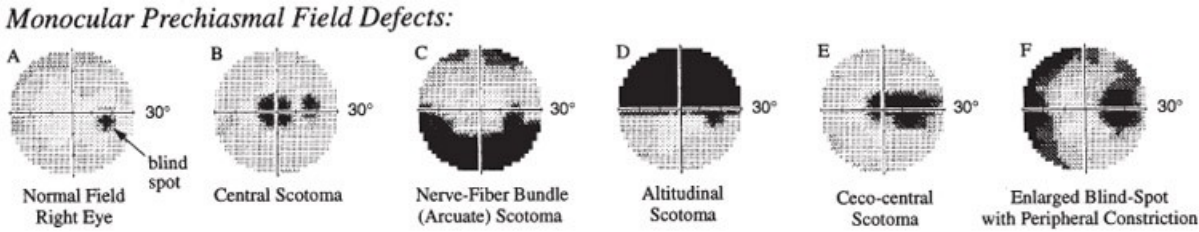
- Examining spatial frequency
- Decreased in some optic nerve disorders (typically optic neuritis)

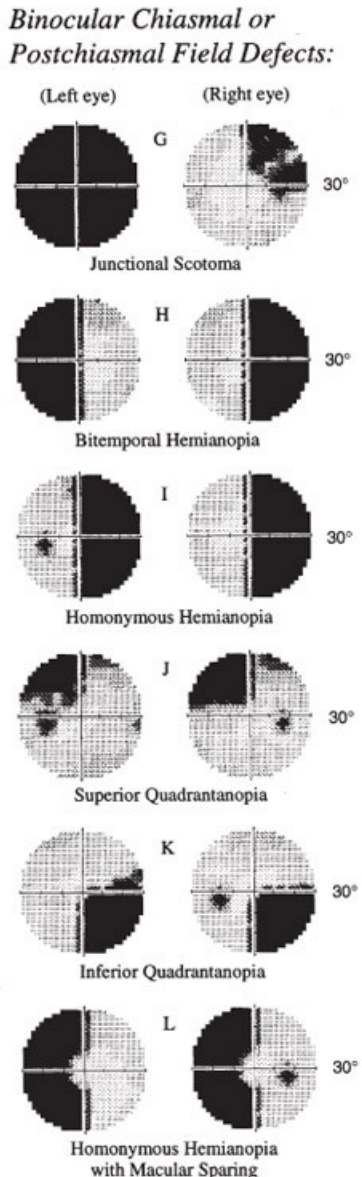


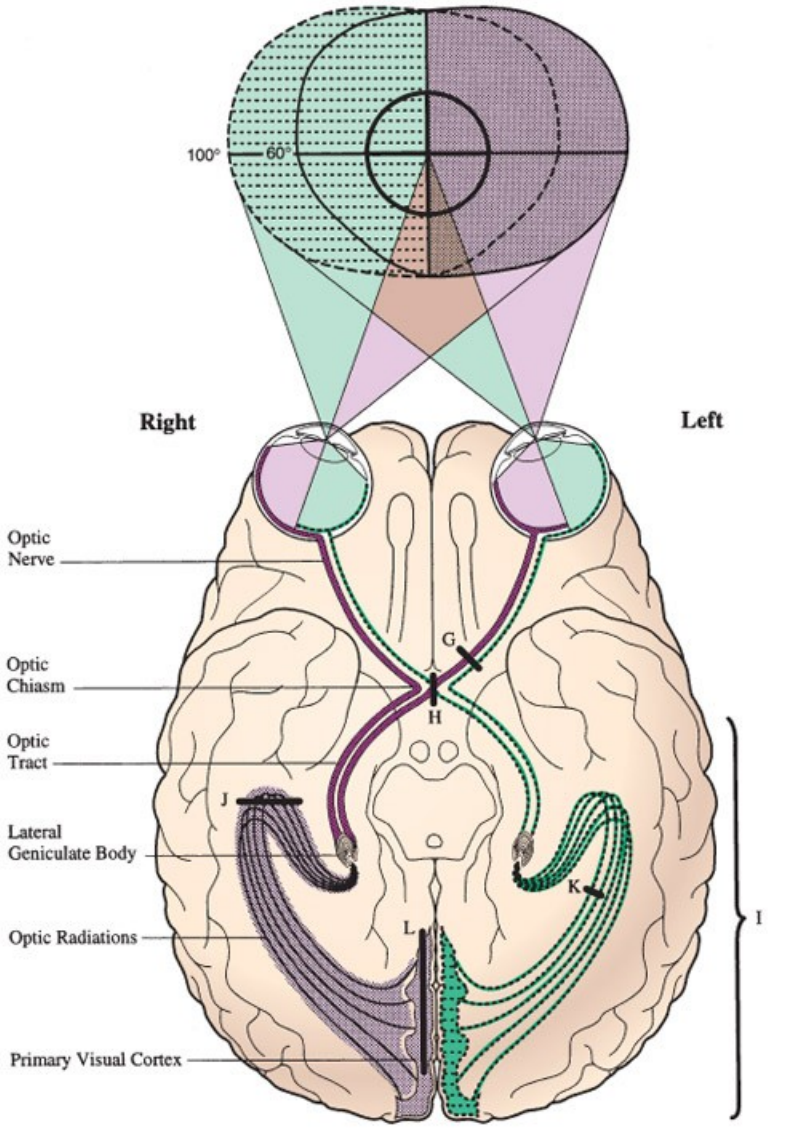


Perimetry

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

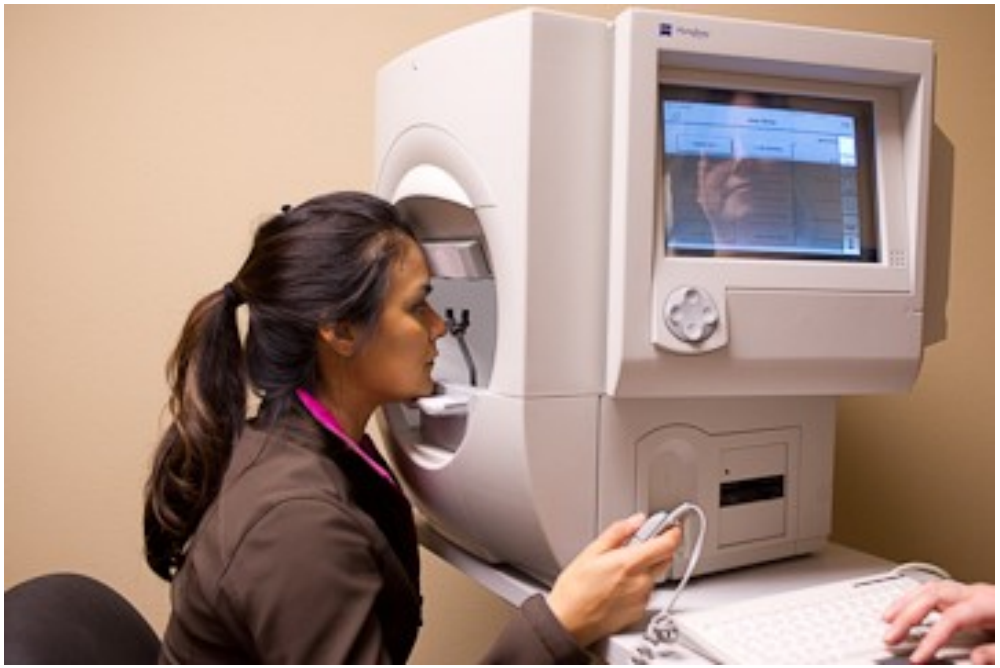


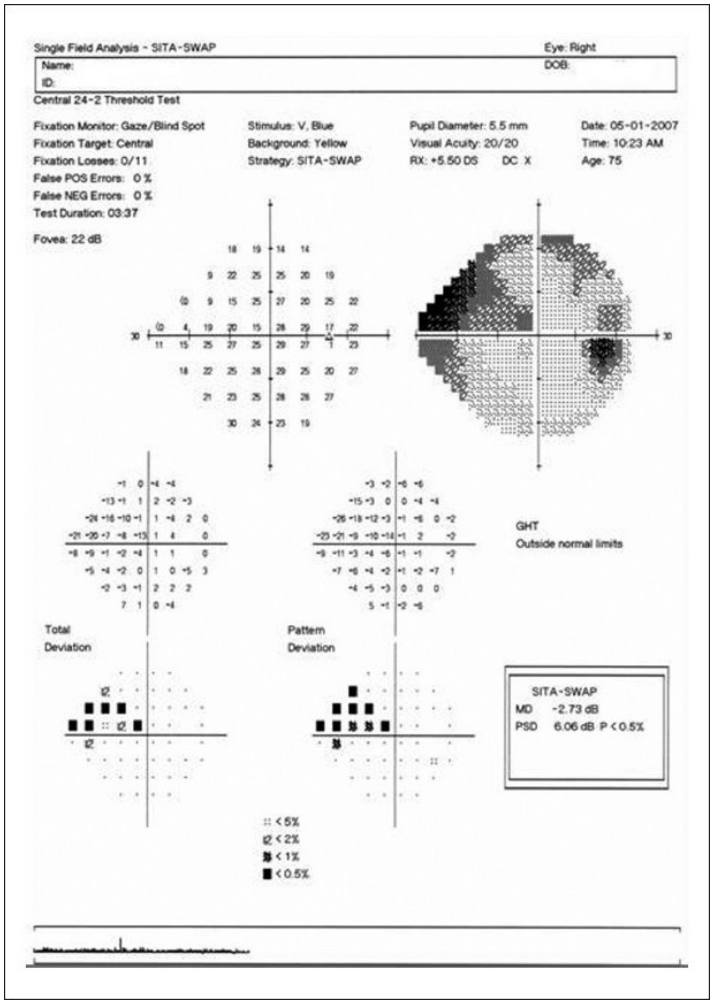




Perimetry

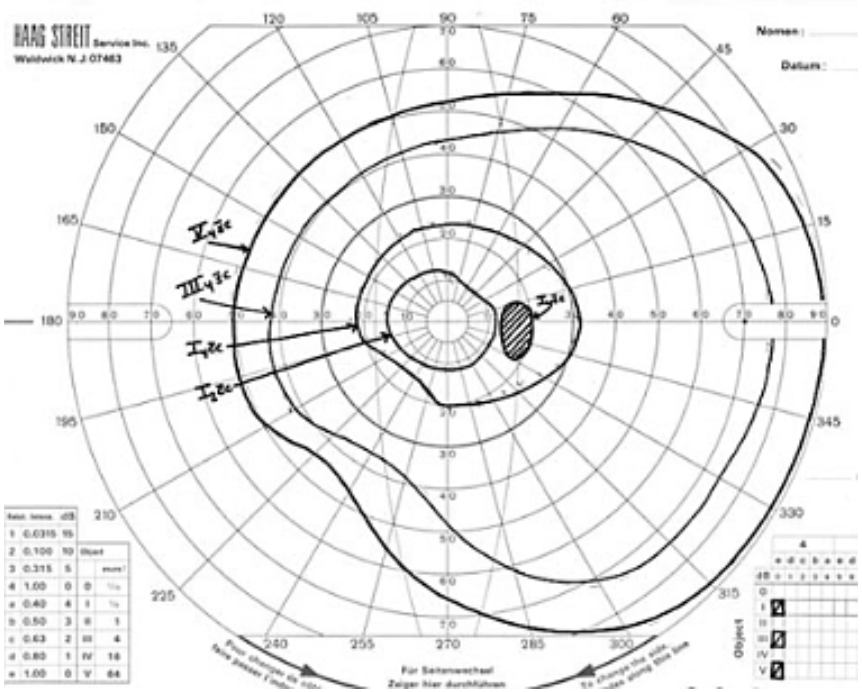
- Automated static perimetry





Perimetry

- Goldmann kinetic perimetry



Electrophysiologic examination

ERG = Electroretinography

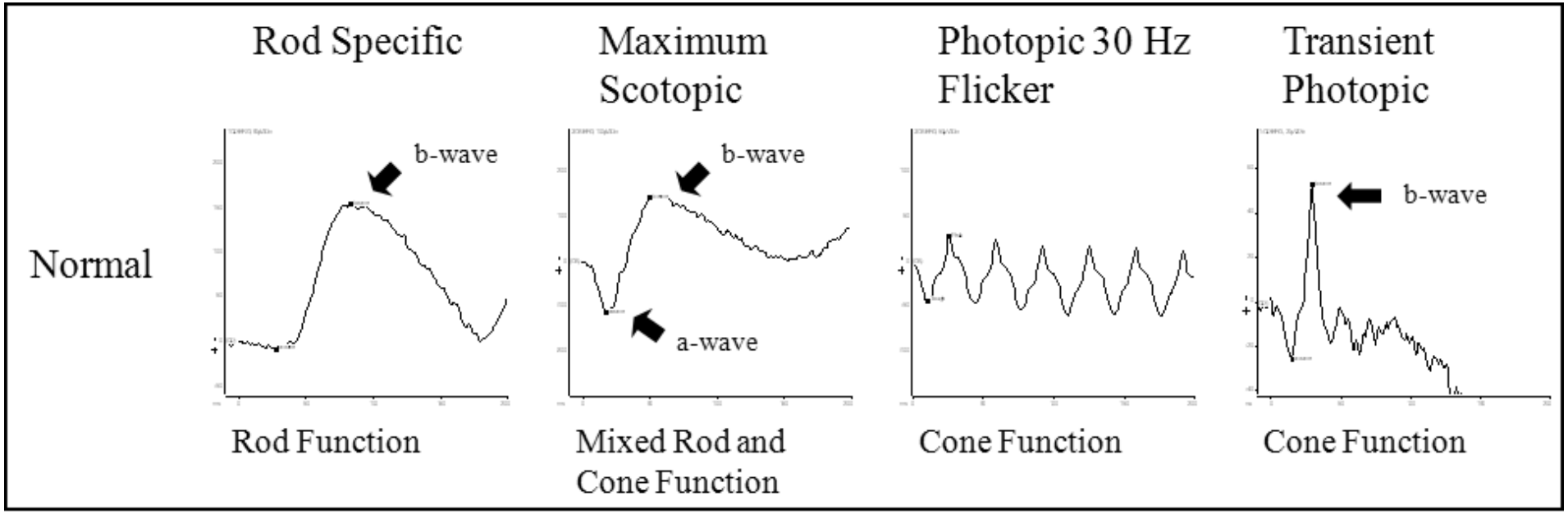
- Access possible functional pathology of retina (scotopic, photopic and central part)
- **Flash ERG** (activity of bipolar cells as an answer to stimation of photosensitive cells – rods, cones)
- **Pattern ERG** (activity of gaglionar cell as a response to stimulation of cones in macula)

VEP = Visual evoked potentials (responses)

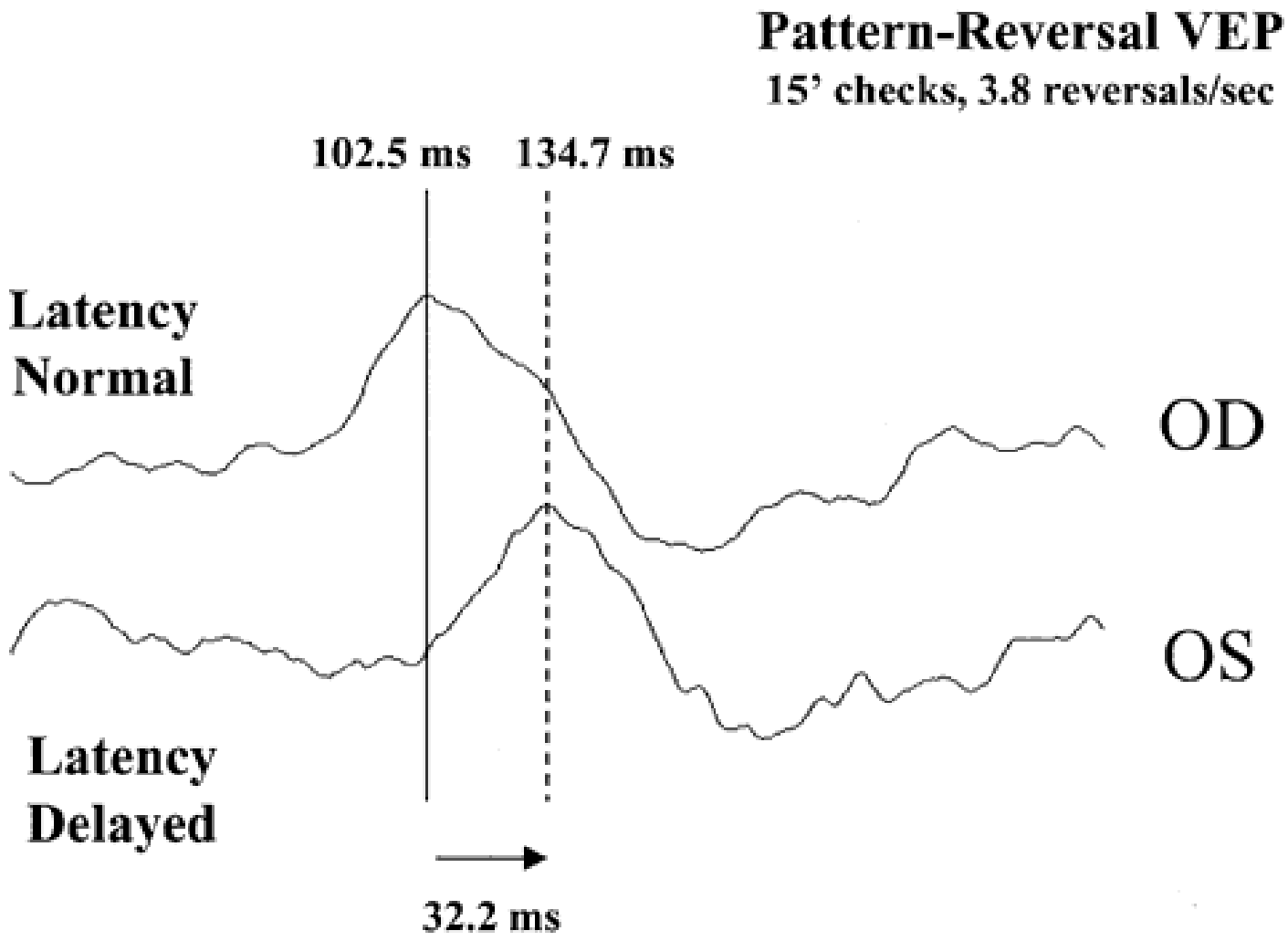
- Access the capability of anterior visual pathways – optic nerve
- Major use: diagnosis/confirm of optic neuritis

Electrophysiologic examination

Electroretinography

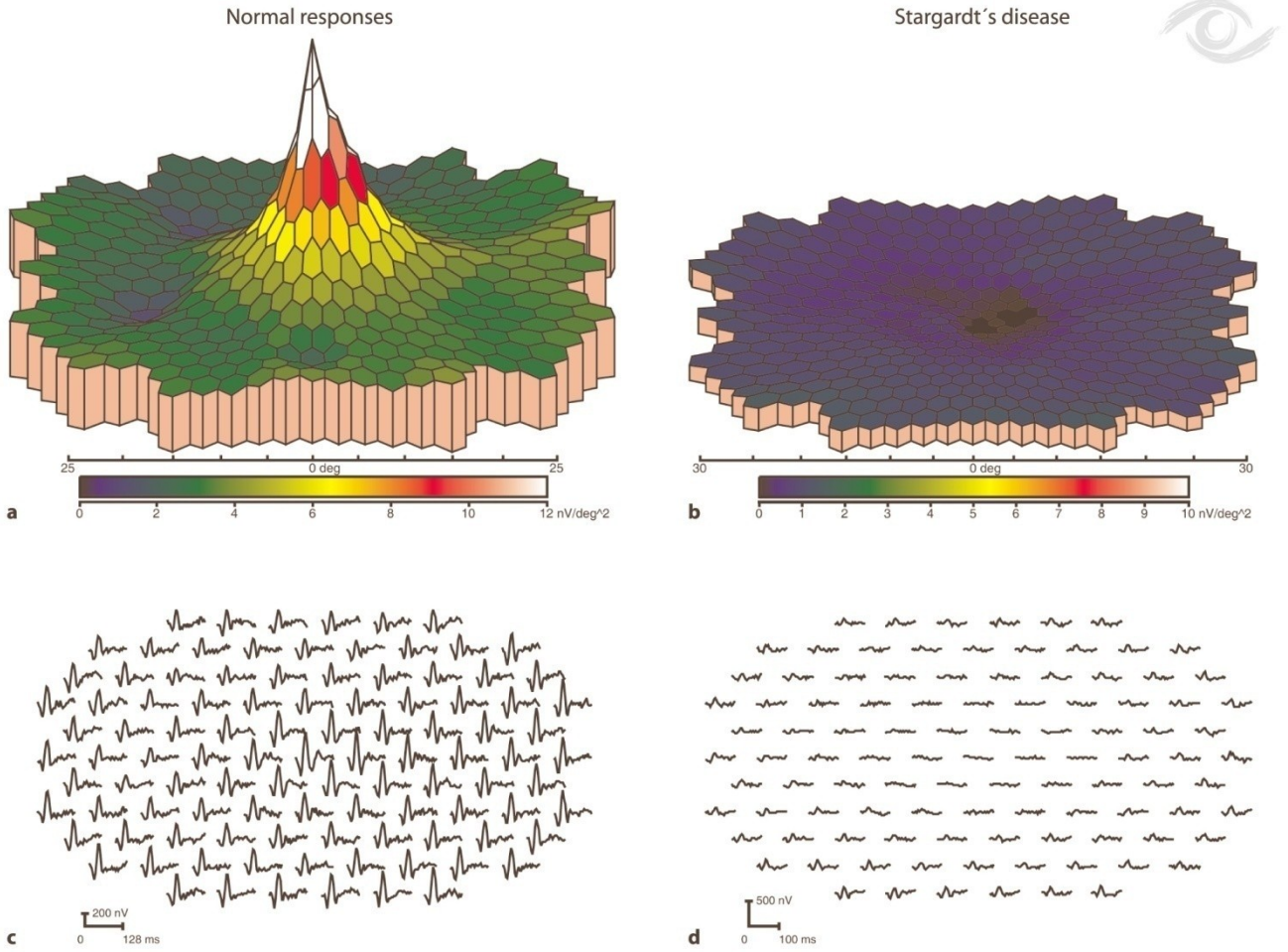


Visual evoked potentials



Multifocal ERG, Multifocal VEP

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

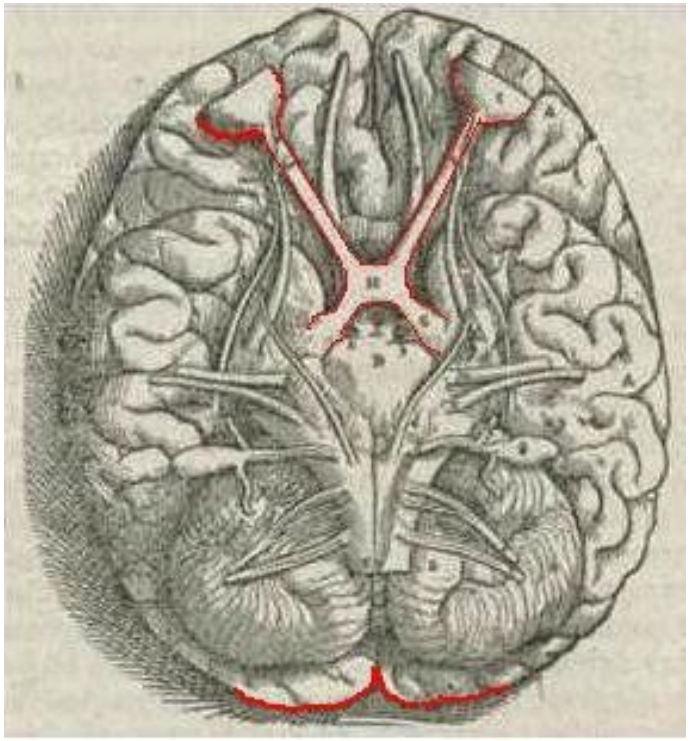


Part II

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 - sing 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

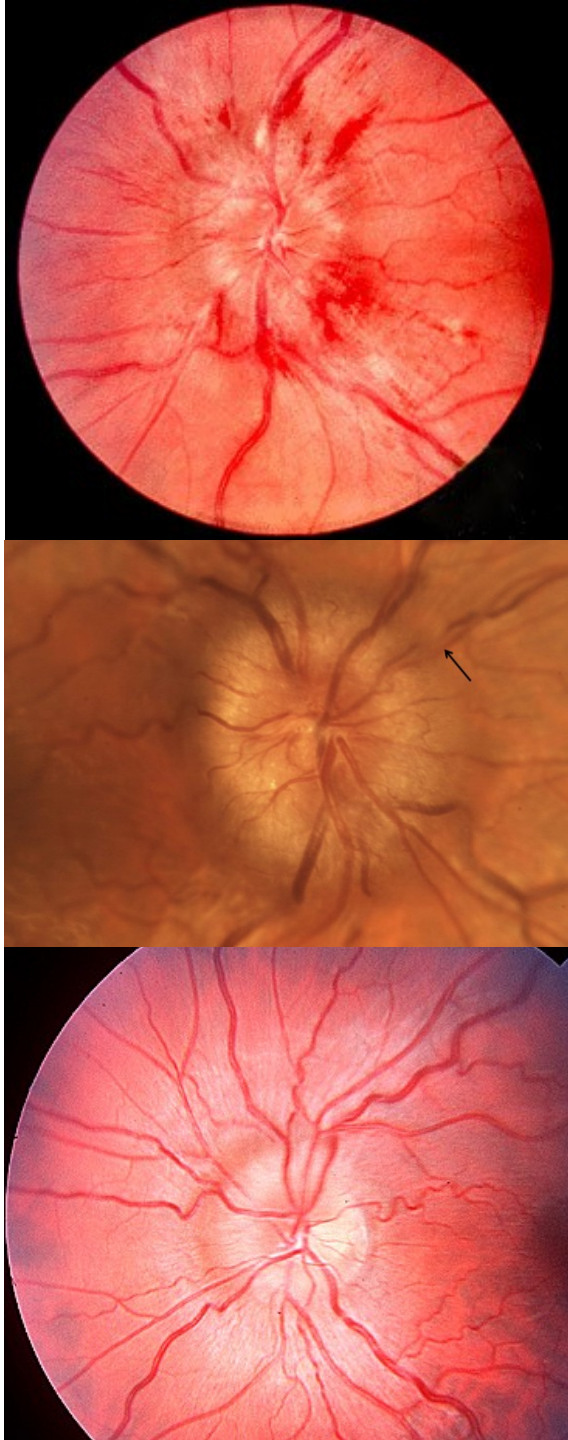
- Margins are obscured
- Optic cup initially preserved
- Hyperemic disc

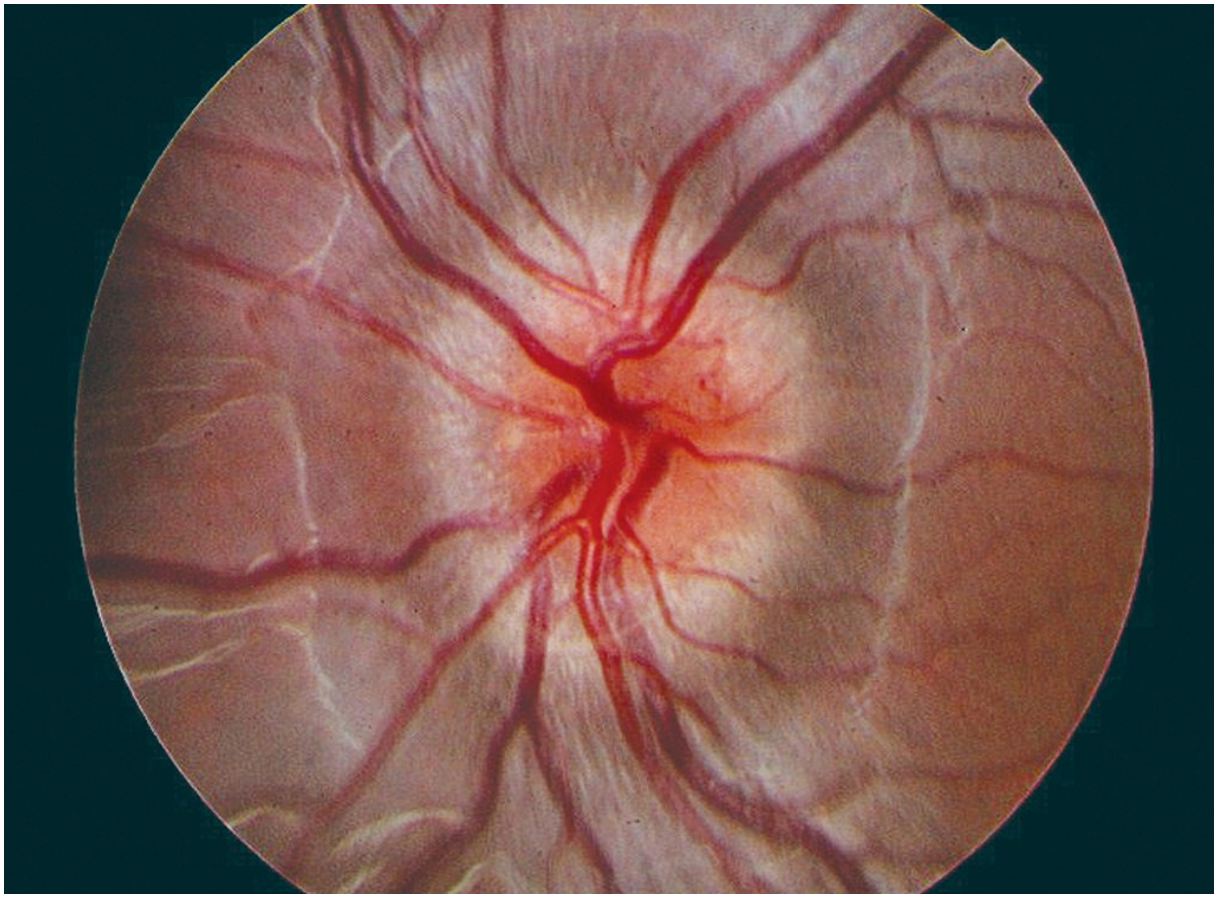
Acute

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

Chronic

- Disc edema
- Obiterated optic cup





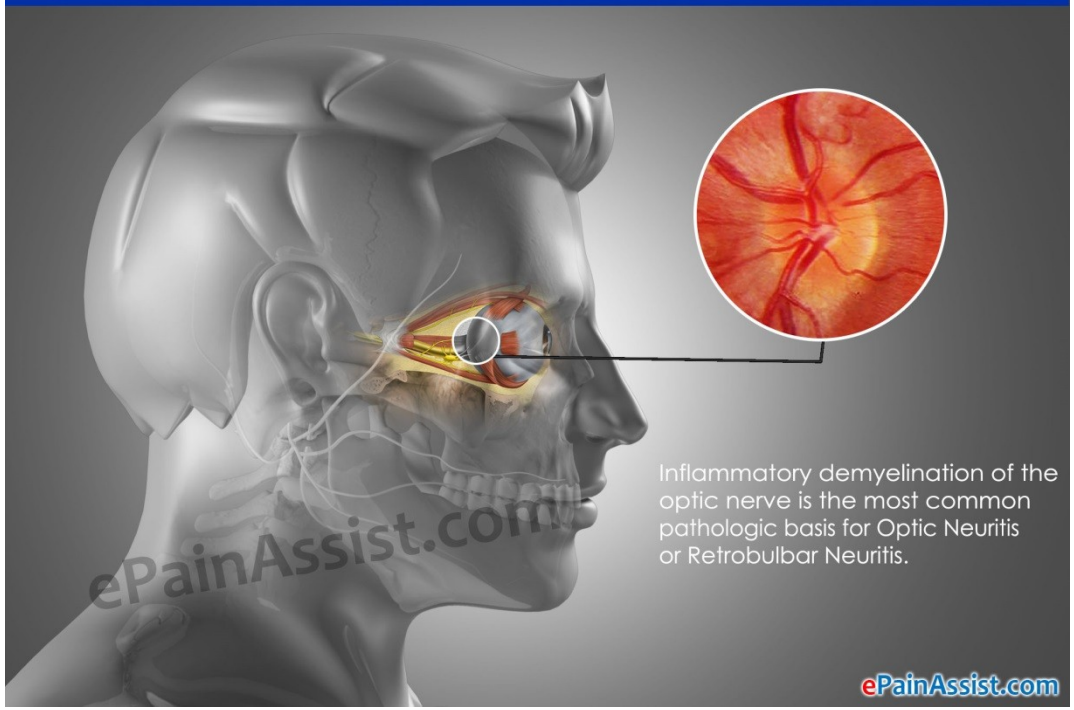
Optic neuritis

- Inflammation of the optic nerve
- **Intraocular** – within the globe
- **Retrobulbar** – posteriot 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 or Retrobulbar Neuritis



Optic neuritis

Symptoms

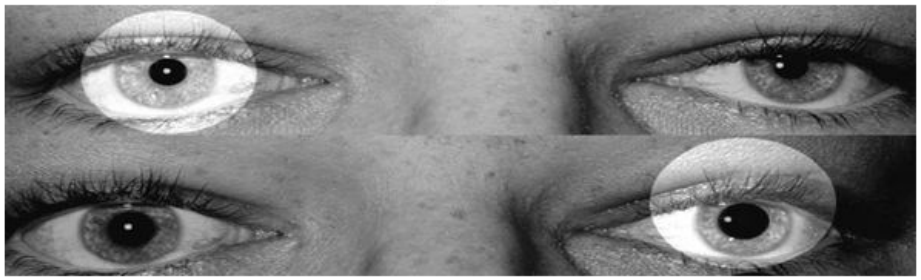
- Sudden vision loss within several hours (mild blurring/light perception)
- Central, paracentral scotoma
- Retrobulbar/parabulbar pain
- Present afferent pupillary defect

Prognosis

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

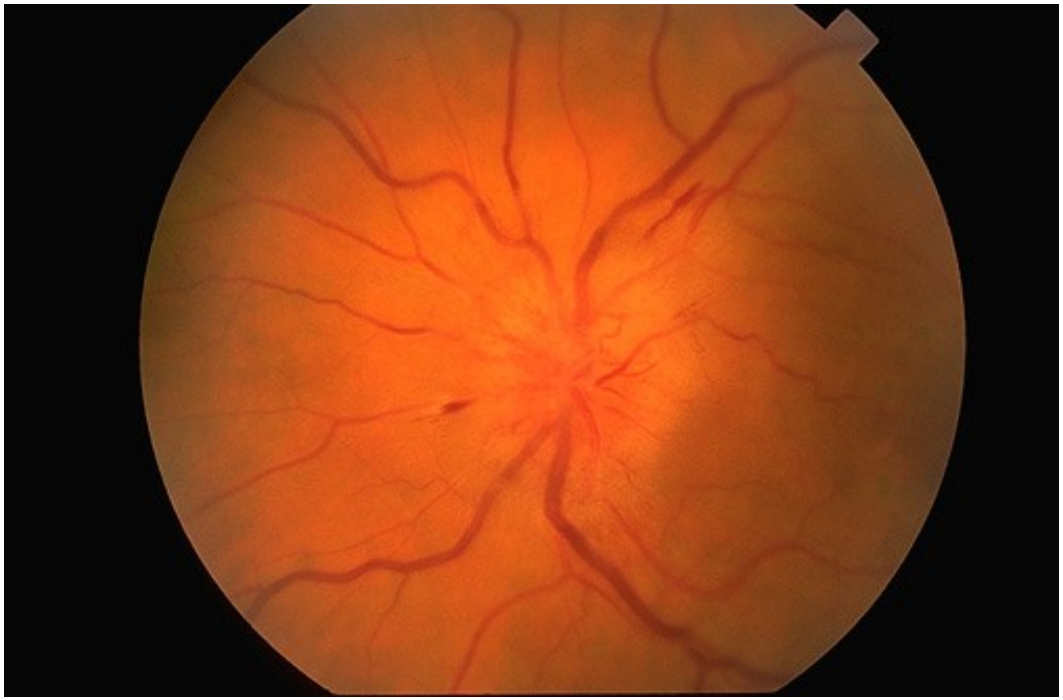
Pupil Testing

- **Relative Afferent Pupillary Defect**



- **Adie's Tonic Pupil-slow response to light**
- **Argyll Robertson-no reaction to light; reaction to accommodation**

How long should the light be held in front on the eye during pupil testing?



Anterior Ischemic Optic Neuropathy

Etiology

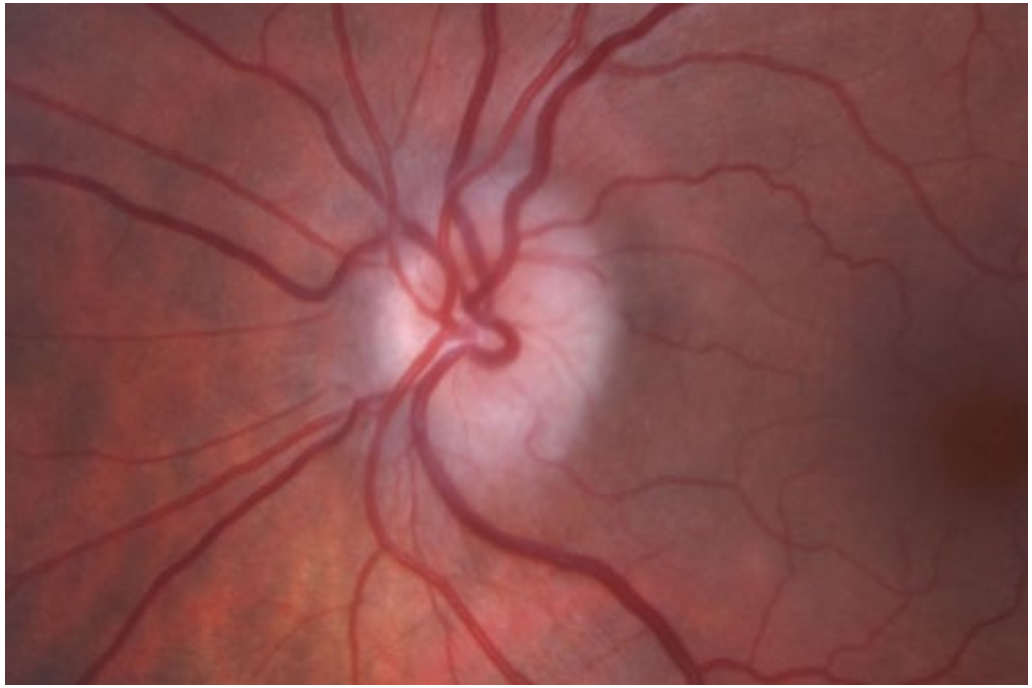
- Acute disruption of blood supply (due to vascular changes, infarction)

Symptoms

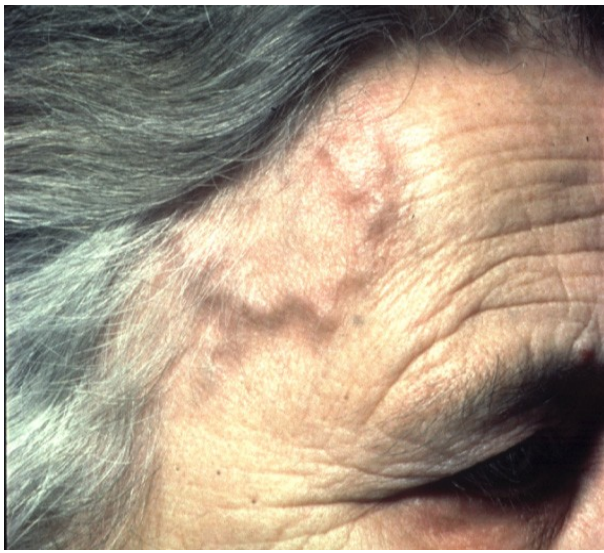
- Sudden unilateral loss of vision
- Altitudinal or wedge-shaped visual field defect
- Present afferent pupillary defect

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

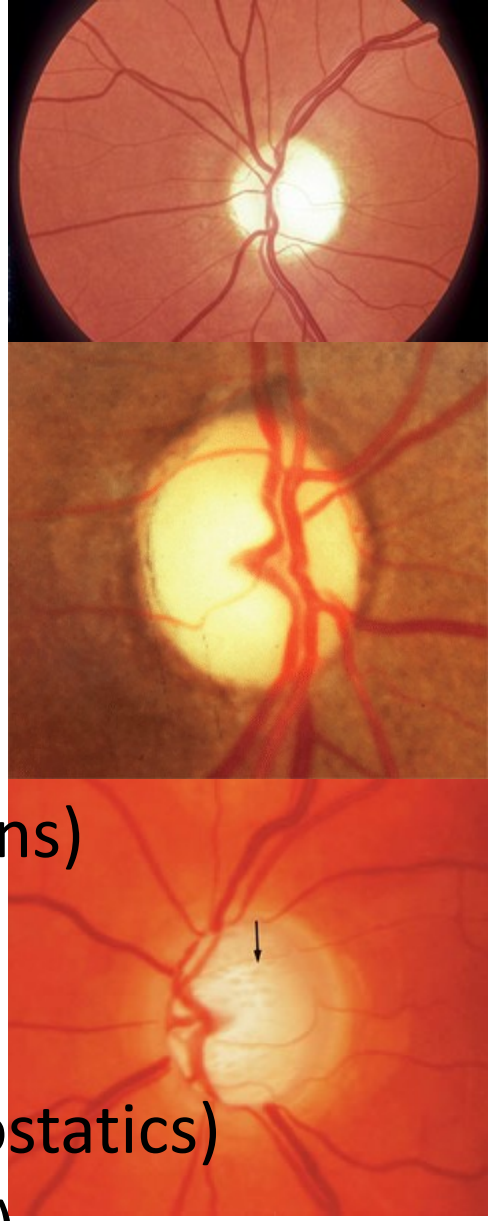
Etiology

- **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 posteriot 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

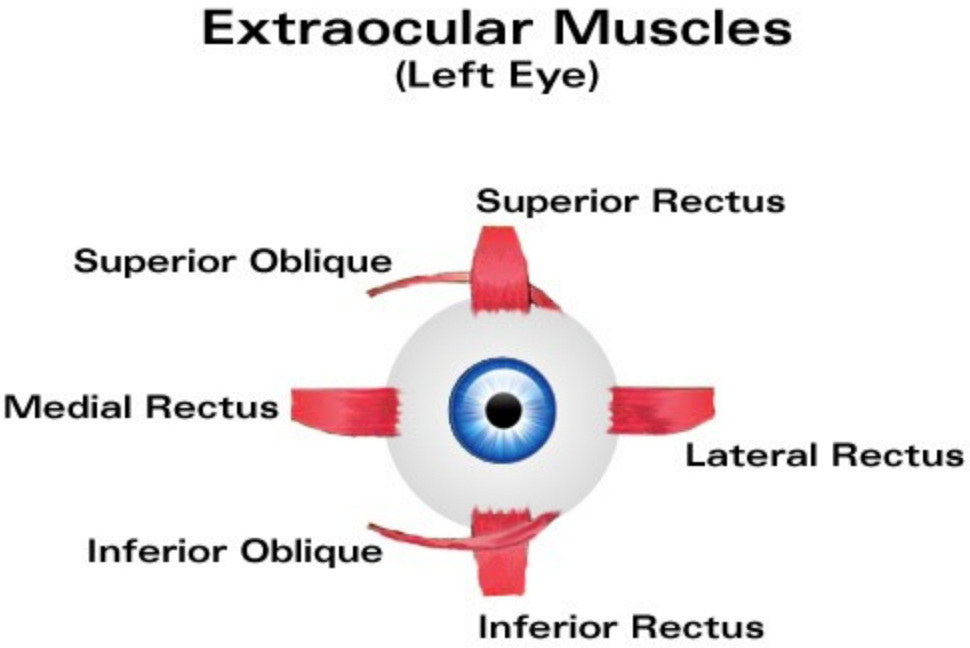
Pathology of Efferent system

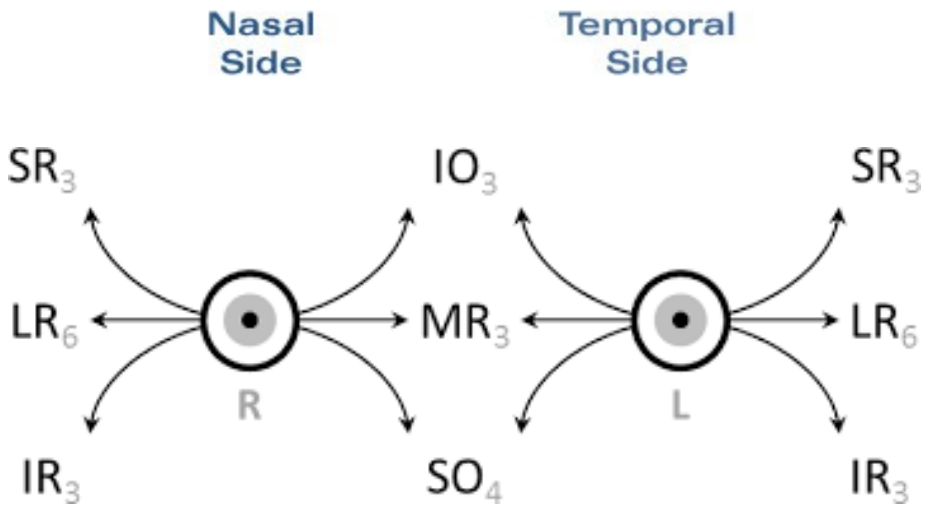
Efferent system

- 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)





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

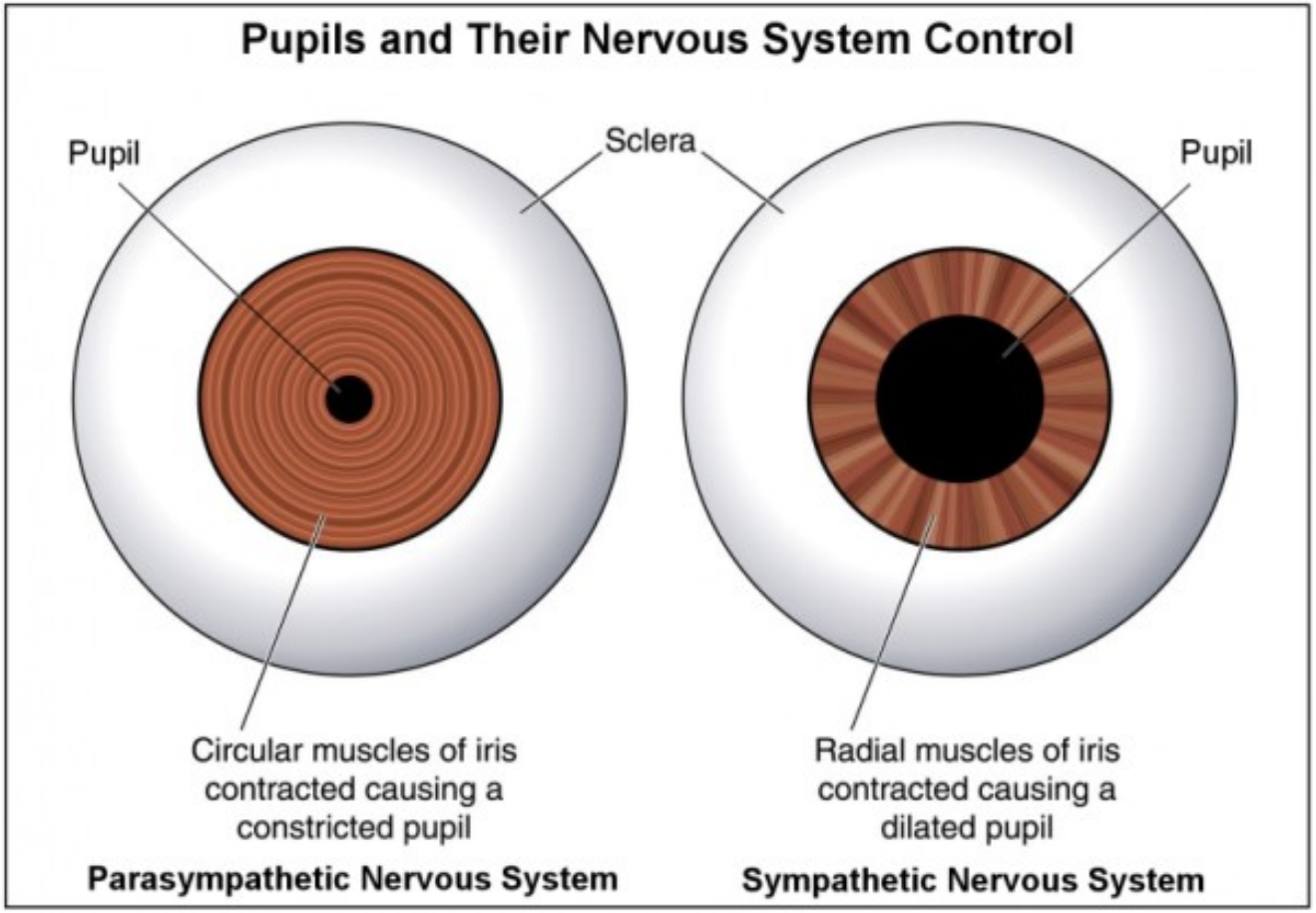
Etiology

- Ischemic (diabetes, hypertension, hyperlipidemia)
- Demyelinating disease (MS)
- Compressive (tumor, aneurysm)
- Elevated ICP

- Multiple cranial neuropathies = suspect lesion in the posterior orbit or cavenrous sinus region

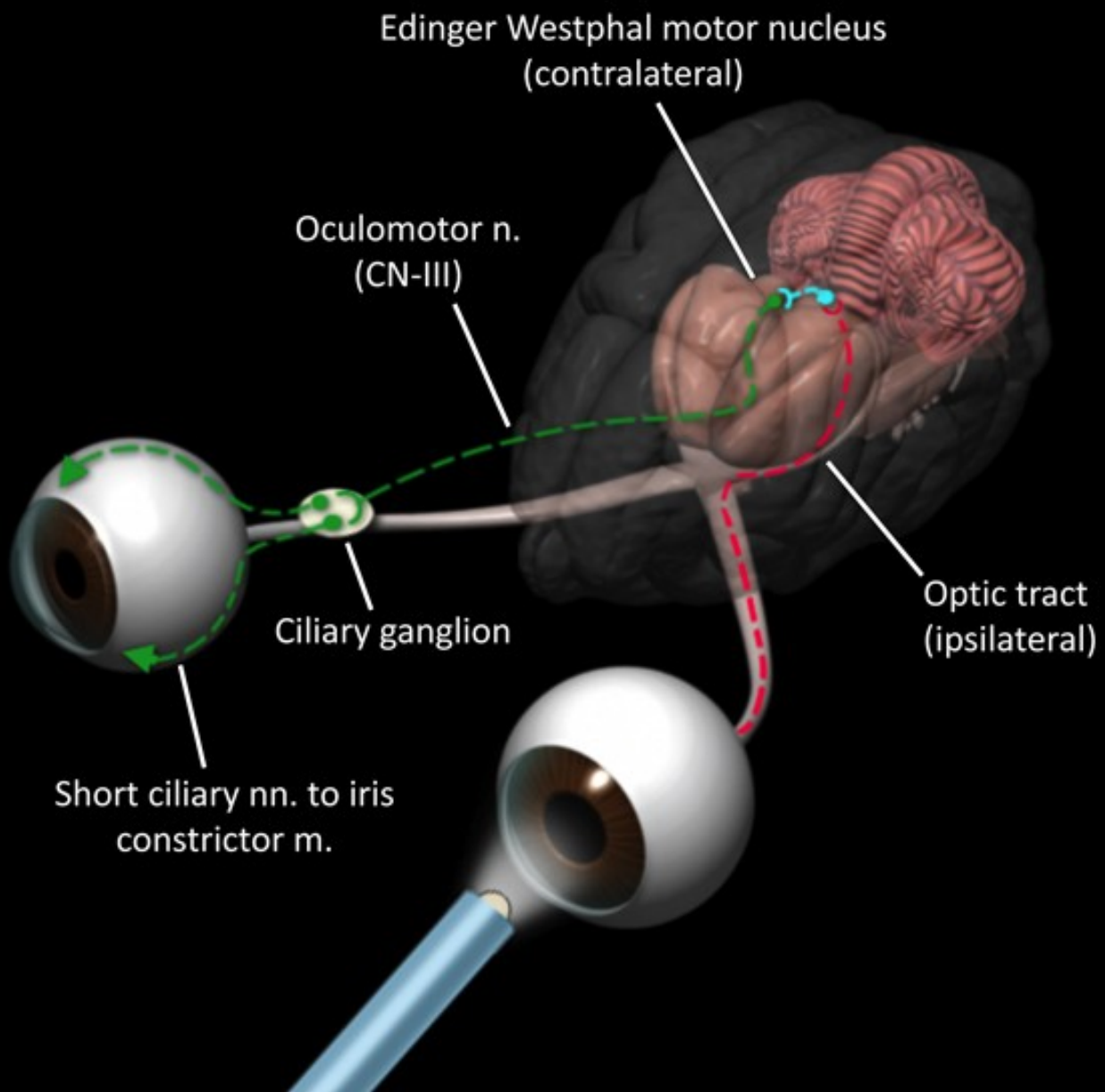
Pupil

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

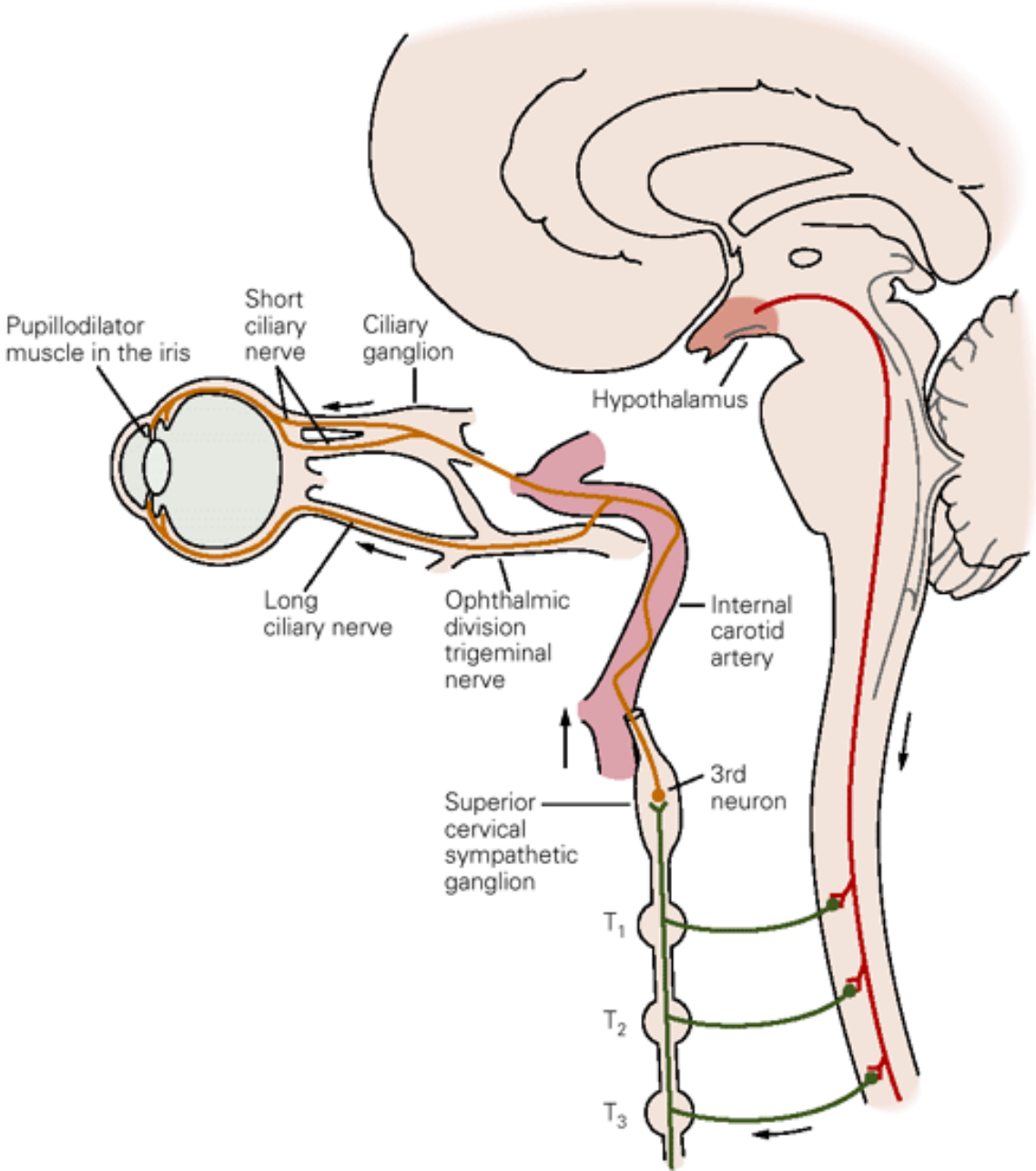


PARASYMPATHETIC CONSENSUAL PATHWAY

Afferent: CN-II | Interneuron | Efferent: CN-III



Sympathetic pathway



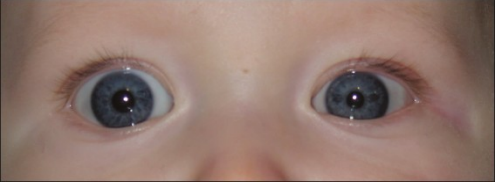
Pupillary abnormalities

Anisocoria

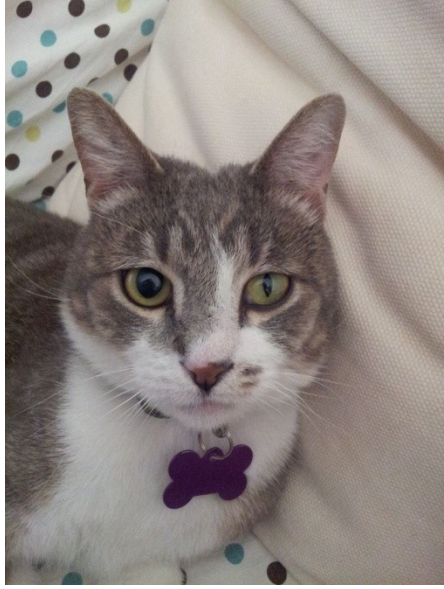
- inequality of pupil size
- May be physiologic
- Possible accidental discovery
- May be isolated / associated with eyelid or ocular motility abnormalities

Diagnosis

- Direct shine at pupil
- Test near response (miosis with accomodation)
- 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 accomodation
- Pupil is larger with light/near dissociation

Etiology

- Inflammation (viral or bacterial infection)

Therapy

- Pilocarpine drops, thoracic sympathectomy

Thank you for your attention!