* Left Optic Tract
  - Uncrossed temporal Ipsilateral
  - Crossed nasal fibers contralateral

* Right Optic Tract
  - Uncrossed temporal Ipsilateral
  - crossed nasal C/L

* Left optic Tract → Represent Right side of field of vision
  Right optic Tract → Represent Left side of field of vision

- Nasal field of vision corresponds to Temporal Retina
  Temporal field of vision corresponds to nasal Retina

- Optic Chiasm → decussation of nasal fibers of ~~Retina~~ Optic nerve
  ↳ Temporal fibers go uncrossed

Optic Nerve
↓
optic Chiasma
↓
Optic Tract
↓
Lateral Geniculate Body
↓
Optic Radiation
↓
visual cortex

* Von Willebrand Knee → Fibres from inferior nasal quadrant of retina loop into opposite optic nerve, before decussation and are called Von Willebrand Knee
- Defect will lead to contralateral upper temporal ~~de~~ defect in field of vision (ar lower nasal part of optic nerve is defected)

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HYPERMETROPIA

LONG SIGHTEDNESS
HYPEROPIA

·Light Rays focus behind the retina

COMPONENTS OF HYPERMETROPIA

1- Latent Hypermetropia
- The amount corrected by inherent tone of ciliary muscle
- +1 D

2. Manifest Hypermetropia
(i) Facultative Hypermetropia
- corrected by accomodative effect

(ii) Absolute Hypermetropia
- residual part of manifest

3. Total Hypermetropia

CAUSES OF HYPERMETROPIA

1- Axial Hypermetropia

1mm Axial Length Decrease = 3D of hypermetropia

2. Curvatural Hypermetropia

1mm change in radius of curvature = 6 D of hypermetropia

3. Index Hypermetropia
- related with diabetes

4. Positional Hypermetropia
- lens position changes → backward

5. Absence of crystalline lens

COMPLICATIONS

1- Recurrent styes, blepharitis, chalazions

2. Esodeviation → Accomodative Convergent Squint

3. Amblyopia
- Anisometropic
- Strabismic
- Ametropic Amblyopia

4. Primary narrow angle glaucoma

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Tx of HYPERMETROPIA

Convex lens → Converging lens

How To IDENTIFY CONVEX LENS

1- Thick at center, thin at periphery
2. Magnifies the object
3- Movement of image in opposite direction to movement of lens

PRINCIPLES OF HYPERMETROPIA CORRECTION

1- Importance of complete cycloplegic (→ paralyze ciliary muscle) examination
↳ (cyclopentolate, Atropine)
↳ to find total hypermetropic correction

2- For error < +1 D,
prescribe error only if patient is symptomatic

3. Children < 4yrs of age → will accept full correction
> 4 yrs of age → Reduce correction to 1/3rd

4. In Exophoria
↳ under correct about 1-2 D

5. In Accomodative Convergent squint (Esotropia)
- Full correction

6. In Amblyopia
- Full correction

7. Follow up every six months

As the child grows, eyeball increase in size and hypermetropic error might decrease with age

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PUPILLARY LIGHT REFLEXES

ARGYL ROBERTSON PUPIL (ARP)

Cause:

- Damage to rostral midbrain (tectum)
- Specifically lesions in dorsal aspect of Edinger westfal nucleus interrupts the pretectal oculomotor light reflex fibers
- The ventral fibers of Edinger Westpal nuclei are spared

Important Features

- small → miotic pupil that do not dilate in dim light
- irregular
- Little or no constriction to light (Absent Light Reflex)
- Brisk near reflex (present near reflex) Light Near Dissociation
- Iris abnormality and trans illumination defect

Why ARP pupils are miotic

- Disruption of supranuclear adrenergic fibers that inhibit the Edinger Westpal nucleus
- Uninhibited parasympathetic neurons of EW nucleus leads to miosis (spinal miosis)

ADIE TONIC PUPIL

- Parasympathetic denervation of pupil
- Poor or absent light reflex
- Near reflex is present (Tonic Reaction)

Cause:

- Damage to parasympathetic ciliary ganglion
- Upregulation of post synaptic receptors to allow re innervation
- Abberent innervation / regeneration → Fibers re-innervation to sphincter pupillae instead of ciliary muscle (Normally
  - ~90% innervation to ciliary muscle
  - ~10% innervation to sphincter pupillae)
- Tonic miosis on near reflex along with accomodation (Light Near Dissociation)

How To TEST ADIE TONIC PUPIL

- Anisocoria → Adie Tonic pupil larger (helps differentiate from ARP)
- Worsening of anisocoria in light
- sectoral palsy or vermiform movements of pupillary margin on slit lamp (due to abberent regeneration)
- Light near dissociation

Testing For Super Sensitivity

- Commercial available 1% pilocarpine diluted to 0.125% with saline
- Check pupil after 30-60 min
  - Abnormal pupil constricts more than normal pupil

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RAPD / MARCUS GUNN

- Rapid onset pupillary defect
- Lesion in afferent pathway of pupillary pathway
- Afferent : From Retina to pretectal nucleus

Conditions causing RAPD

- Lesions of Retina / Posterior Segment
  - Large retinal detachments
  - Ischemia (CRVO, CRAO)
  - Dense macular lesion (chorioretinal scar)
- Lesions in anterior optic pathway (anterior to lateral geniculate body i.e upto optic tract)
  - Lesions of optic nerve (optic neuropathy, glaucoma)
  - Lesions of optic chiasm
  - Lesions of optic tract
  - Lesions of pretectum

SWINGING FLASHLIGHT TEST

- Both eyes get dilated (instead of constriction) when light is shone on defected eye

GRADING OF RAPD

Grade I : A weak pupillary constriction followed by greater dilatation

Grad II : Pupillary stall followed by dilatation

III : Immediate pupillary dilatation

IV : Following 6sec illumination, mild constriction or stall with dilatation

V : Amaurotic pupil, immediate pupillary dilation

WERNICKE'S PUPIL

- Best source of illumination : Focal beam of a slit lamp reduced to a spot size
- In optic Tract Lesion → Wernickes hemianopic pupil
  - When light is thrown in temporal half of same side and nasal half of other side → No pupillary Reflex
  - When light is thrown in nasal half of same side and temporal half of other side → pupillary Reflex present

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VISUAL PATHWAY LESIONS

- Visual Fields and Retina have inverted and reversed relationship
  - The superior part of visual field is represented in inferior part of retina and vice versa
  - The temporal part of visual field represented in nasal part of retina
  - The nasal " " " " temporal "

- Homonymous: both eyes are involved with same laterality
  e.g right visual field affected in both eyes → homonymous
- Congrous Field Defect: If the field effect is alike in both eyes
- Incongrous Field Defect: " " " different " "

Lesion In Optic Nerve

- Optic nerve might be affected due to:
  - Optic Atrophy
  - Traumatic avulsion of optic nerve
  - Transection of optic nerve
- Complete blindness of same side
- ipsilateral direct light reflex Absent
- contralaterel consensual light reflex absent

Lesion In Optic Nerve Near The Chiasm

- Total blindness in ipsilateral eye → due to defect in optic nerve
- Upper temporal defect in field of vision ~~of due to defs~~ in contralakral eye → due to defect in Von Willebrand Knee

* Von Willebrand Knee → Fibers from inferior nasal quadrant of retina loop into opposite (contralateral) optic nerve, before decussating and are called Von Willebrand knee
  - As Lower nasal part of optic nerve is defected → Defect will be in contralateral upper temporal part of field of vision
  - Such defect is called Junctional Scotoma

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CENTRAL CHIASMAL LESION

Optic Chiasm → decussation of nasal fibers of retina.

- Bitemporal hemianopia
  (Nasal Fibers decussate and nasal fibers correspond to temporal field of vision)
- Bitemporal hemianopic paralysis of pupillary reflexes
  (pupillary response on temporal part of pupil is absent)
- Optic Atrophy

[Defective pupillary reflexes and optic atrophy seen only in lesion upto the lateral geniculate body]

LATERAL CHIASMAL LESION

- Binasal hemianopia
- Binasal hemianopic paralysis of pupillary reflex
- Optic Atrophy

OPTIC TRACT LESION

- ~~Left~~ Optic Tract Fiber consist of
  - uncrossed temporal fibers from ipsilateral side
  - crossed nasal fibers from contralateral side
- Left optic tract → Represent right side of field of vision
- Right optic tract → Represent left side of field of vision

Optic Tract Lesion:

- Incongrous homonymous hemianopia
  (Incongrous bcz nasal part of field are smaller compared to temporal part of field)
- Hemianopic pupillary response (Wernicke pupil)
  [No pupillary response in affected part of pupil
  Pupillary response is present in normal part of pupil]
- Optic Atrophy
- contralateral 3rd nerve palsy and ipsilateral hemiplegia (sometimes)

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LATERAL GENICULATE BODY LESIONS

- homonymous incongrous hemianopia
- sparing of the pupillary reflex
- May have partial optic atrophy

OPTIC RADIATION LESIONS

- Total optic Radiation Lesion → Complete homonymous hemianopia
- Involvement of superior fibers of radiations in parietal lobe:
  ↳ Inferior quadrantic hemianopia (pie in the floor)

→ Left optic Radiation Defect cause defect in Right visual field

- Involvement of inferior fibers of radiations in temporal lobe:
  ↳ Superior quadrantic hemianopia (pie in the sky)

- No effect on pupillary reflex

VISUAL CORTEX LESION

- Blood Supply of Visual Cortex: Posterior cerbral Artery
  Middle cerebral Artery
  - Both carotid system and vertebral system contribute to blood supply of visual cortex
  - Max number of fibers in visual cortex represent macula
  - Mostly posterior cerebral artery contribute to the blood supply of visual cortex (most of the anterior part of visual cortex). So whenever there is a stroke involving posterior cerebral, almost entire visual cortex will be affected except macular part.
- contralateral congruous homonymous hemianopia (usually sparing the macula)
- Normal pupillary Reflex
- No Optic Atrophy

Macular sparing due to its dual blood supply

- Macula is spared bcz macula gets its blood supply from middle cerebral artery even in case of posterior cerbral artery stroke
- If

Defect In Tip of Occipitial Lobe

Tip of occipital lobe may be affected by:

- Head injury
- Gunshot Injury

Here macular fibers get affected and the lesion is

- Contralateral congrous homonymous macular defects

macular Defect

* Tests For visual standard
  - Visual Acuity
  - Color vision
  - Visual Field
  - Binocular Function

* Normal Vision → 20/20
  Low Vision → < 6/18
  Blindness → < 3/60

* Visual Acuity: Sharpness of vision, measured as maximum distance a person can see a certain object, divided by the maximum distance at which a person with normal sight can see the same object

* Global causes of Blindness
  - Cataract
  - Glaucoma
  - DM
  - Vascular Disease
  - Accidents and degeneration of ocular tissue
  - Leading causes of childhood blindness
    - Xerophthalmia
    - Congenital cataract
    - Congenital glaucoma
    - Optic Atrophy

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* For Myopia → Concave (-) lens
  For Hypermetropia → Convex (+) lens

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GRADUAL VISUAL LOSS

- Refractive errors
- Cataract
- Primary open angle Glaucoma
- Age Related Macular Degeneration
- Diabetic Retinopathy
- Cornea (Ectasia, Dystrophy)
- Optic Neuropathies (Compression, Toxic, Drugs, Nutritional Deficiency)
- Choroid and Retina (Inflammations, Tumors, Dystrophies)
- Papilledema

SUDDEN VISUAL LOSS

- Retinal vascular diseases (CRVO, BRVO, CRAO, BRVO)
- Retinal Detachment
- Vitreous hemorrhage
- Acute Angle Closure
- Optic Neuropathy (Optic Neuritis, AION)
- Endophthalmitis
- Trauma

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PAINFUL VISUAL LOSS

- Acute Angle Closure Glaucoma
- Neovascular Glaucoma
- Lens induced Glaucoma
- Endophthalmitis / panophthalmitis
- Optic Neuritis
- Anterior uveitis
- Temporal arteritis
- Trauma

PAINLESS VISUAL LOSS

- Cataract (except LIG)
- Primary open Angle Glaucoma / NTG
- CRVO
- CRAO
- Retinal Detachment
- Vitreous hemorrhage
- Macular Degeneration

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