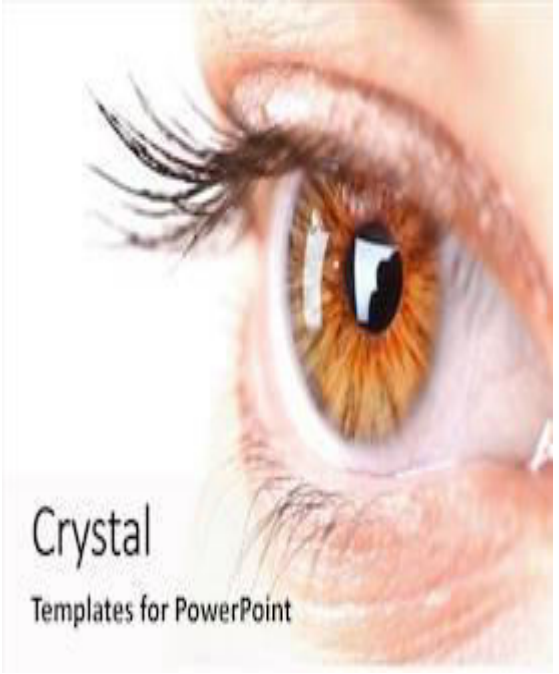


PUPILLARY REFLEXES

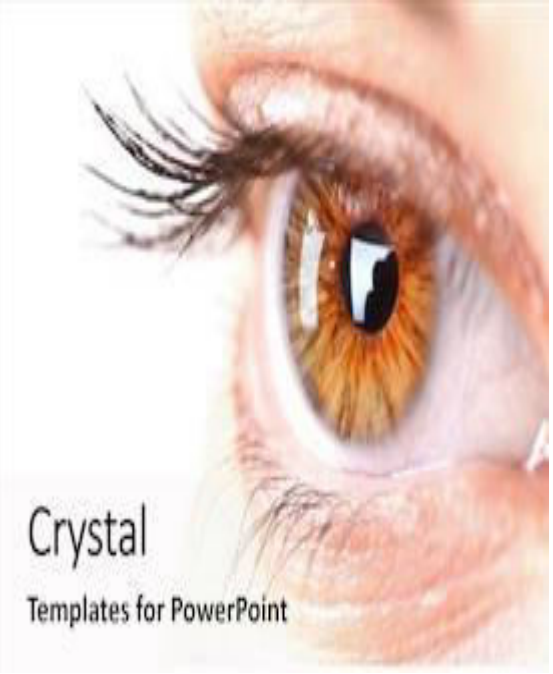
DR SAMINA

AP OHTHALMOLOGY

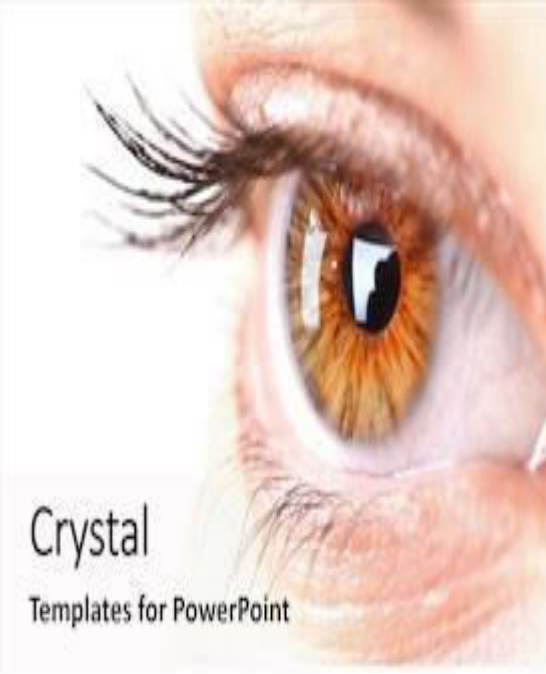
Objectives

1. what are normal pupillary reflexes

2. what are the abnormal pupillary reflexes



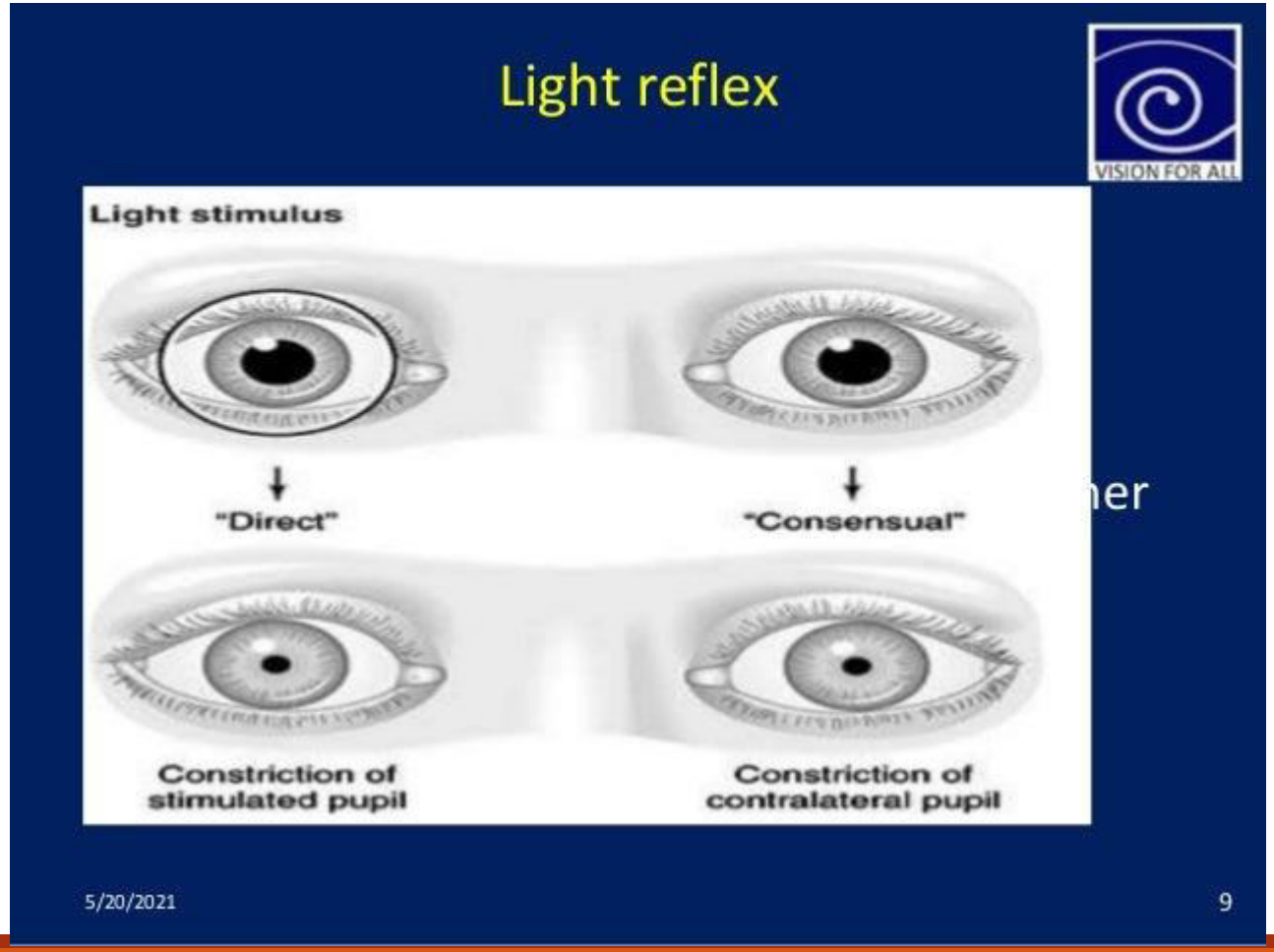
NORMAL PUPILLARY REFLEXES

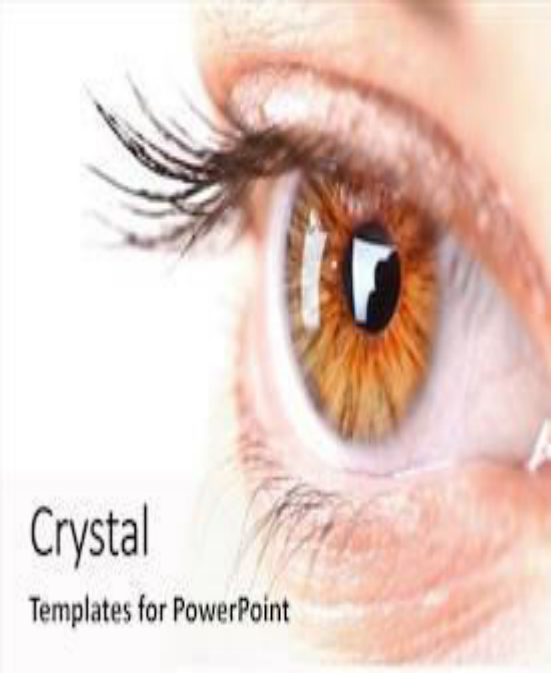


- LIGHT REFLEX
- NEAR REFLEX
- OTHER REFLEXES
  - DARKNESS REFLEX
  - PSYCHOSENSORY REFLEX
  - LID CLOSURE REFLEX

LIGHT REFLEX

- DIRECT
- CONSENSUAL

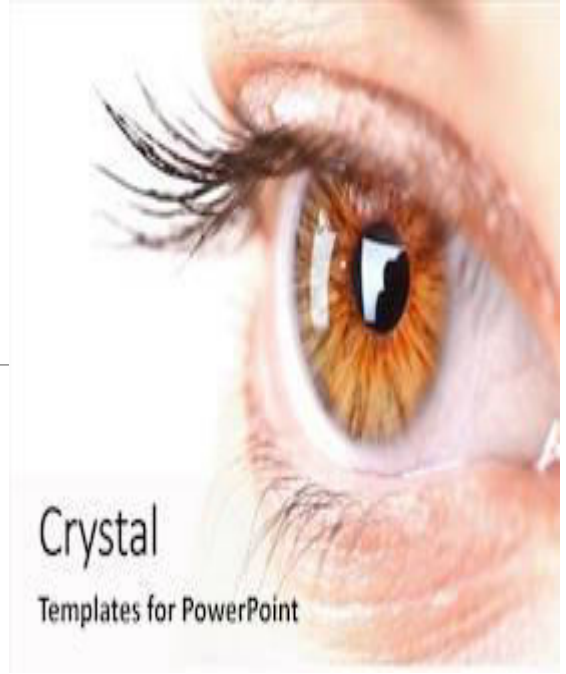




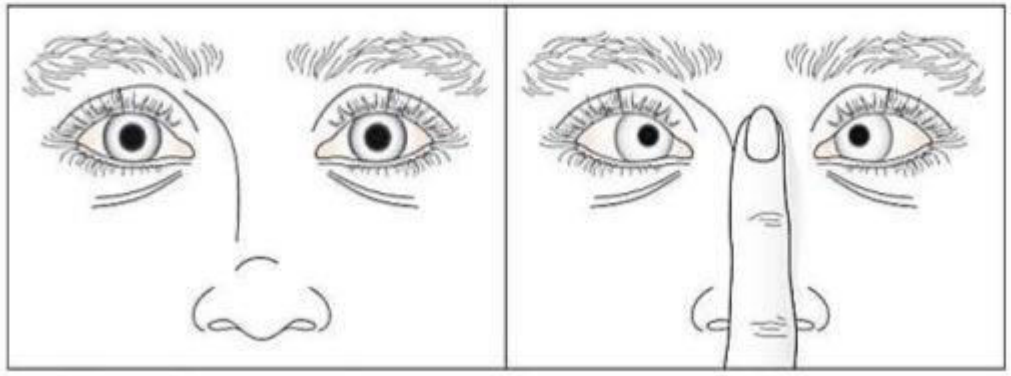
NEAR REFLEX

Near reflex is a Triad of:

- Inc. accommodation
- Convergence of visual axes
- Constriction of the pupils



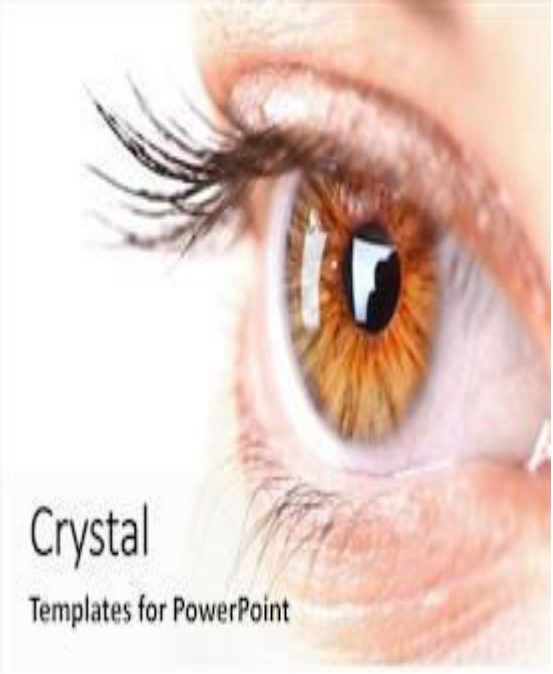
- **Accommodation reflex:**
  - **The patient is asked to look at a distant object and then at an object close to his face.**
  - **Both pupils should constrict and dilate again when distant gaze is resumed**

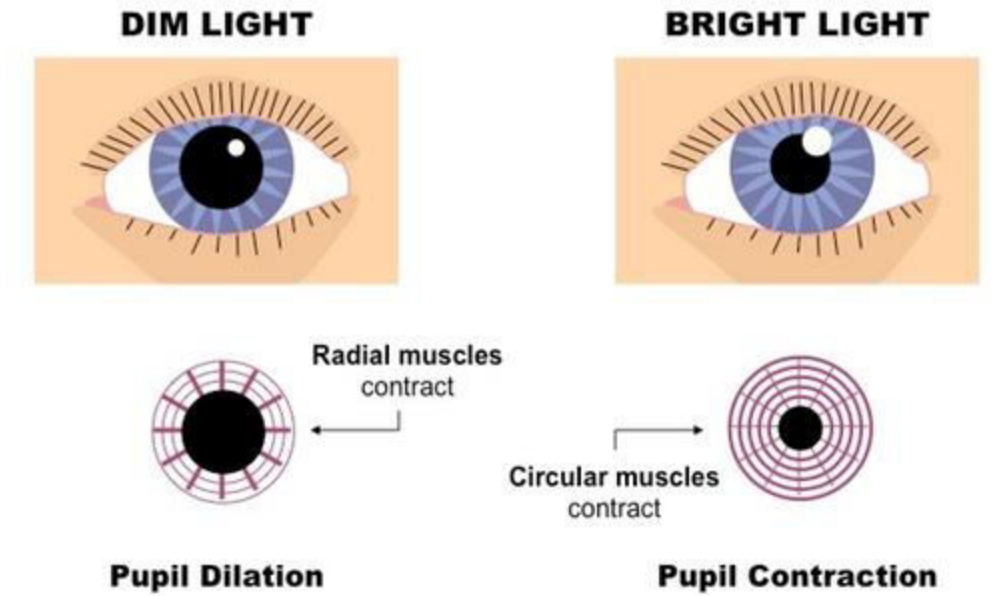


DARKNESS REFLEX

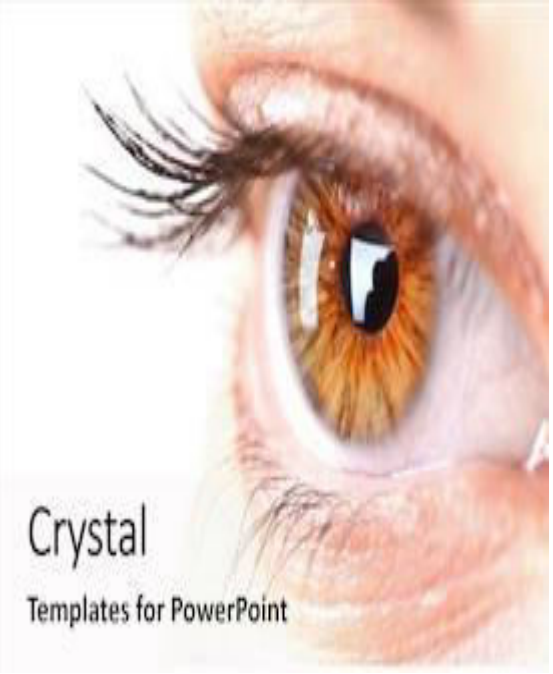
Abolition of light reflex – relaxation of sphincter pupillae and

Contraction of dilator pupillae – dilatation of the pupil -----supplied by sympathetic nervous system





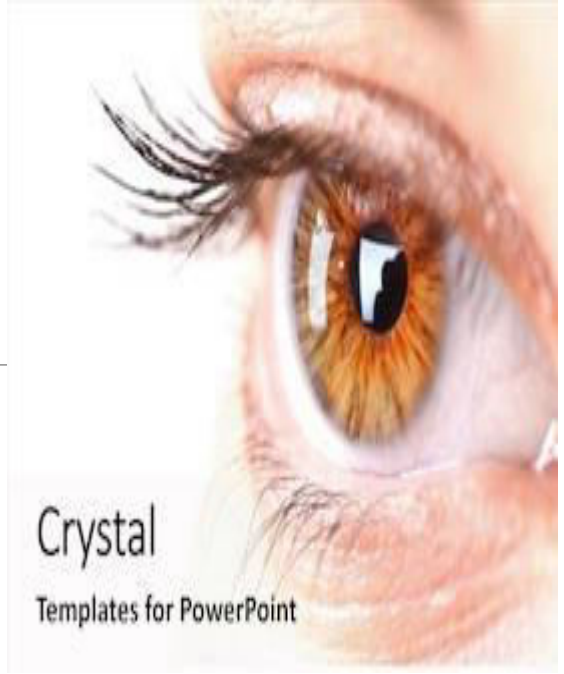
PSYCHOSENSORY REFLEX



- Dilatation of pupil in response to sensory & psychic stimuli
- Fully developed by 6 months of age
- Pathways – unknown
- Two components –
  - Sympathetic discharge to dilator pupillae muscle
  - Inhibition of parasympathetic discharge to sphincter pupillae muscle PSYCHOSENSORY REFLEX

LID CLOSURE REFLEX

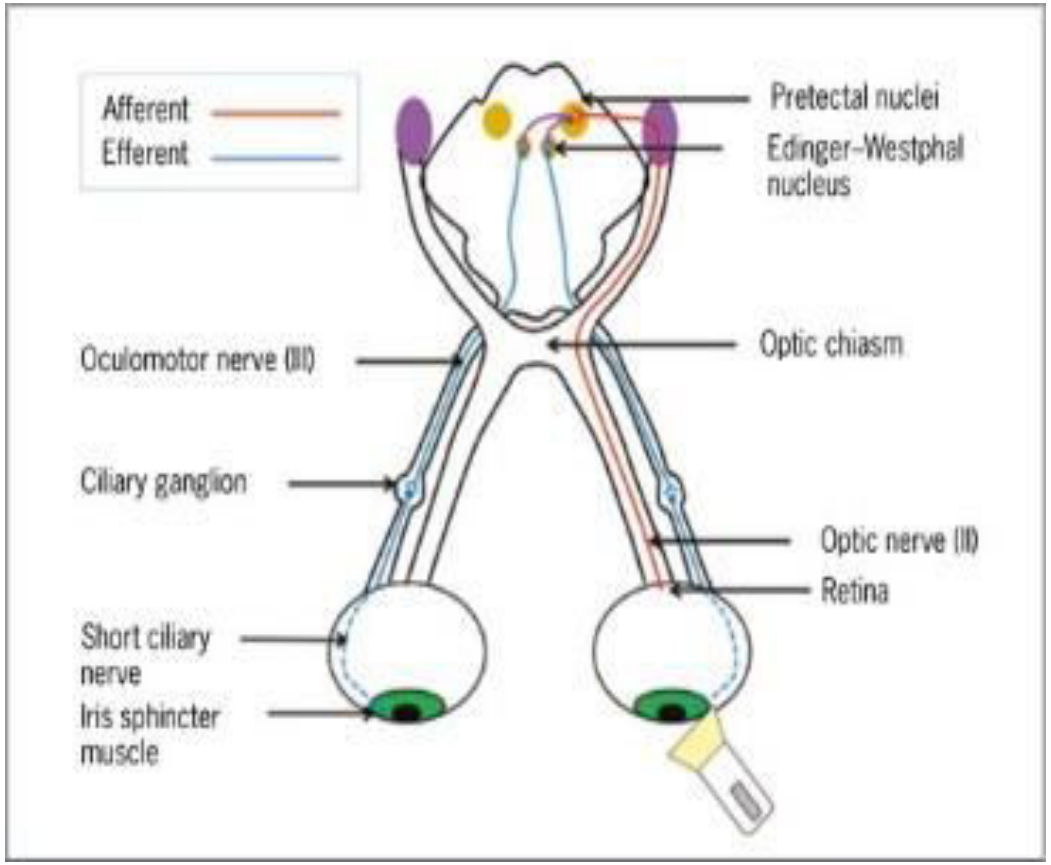
Constriction of pupil associated with blinking –



Normal visual pathway to the iris muscle

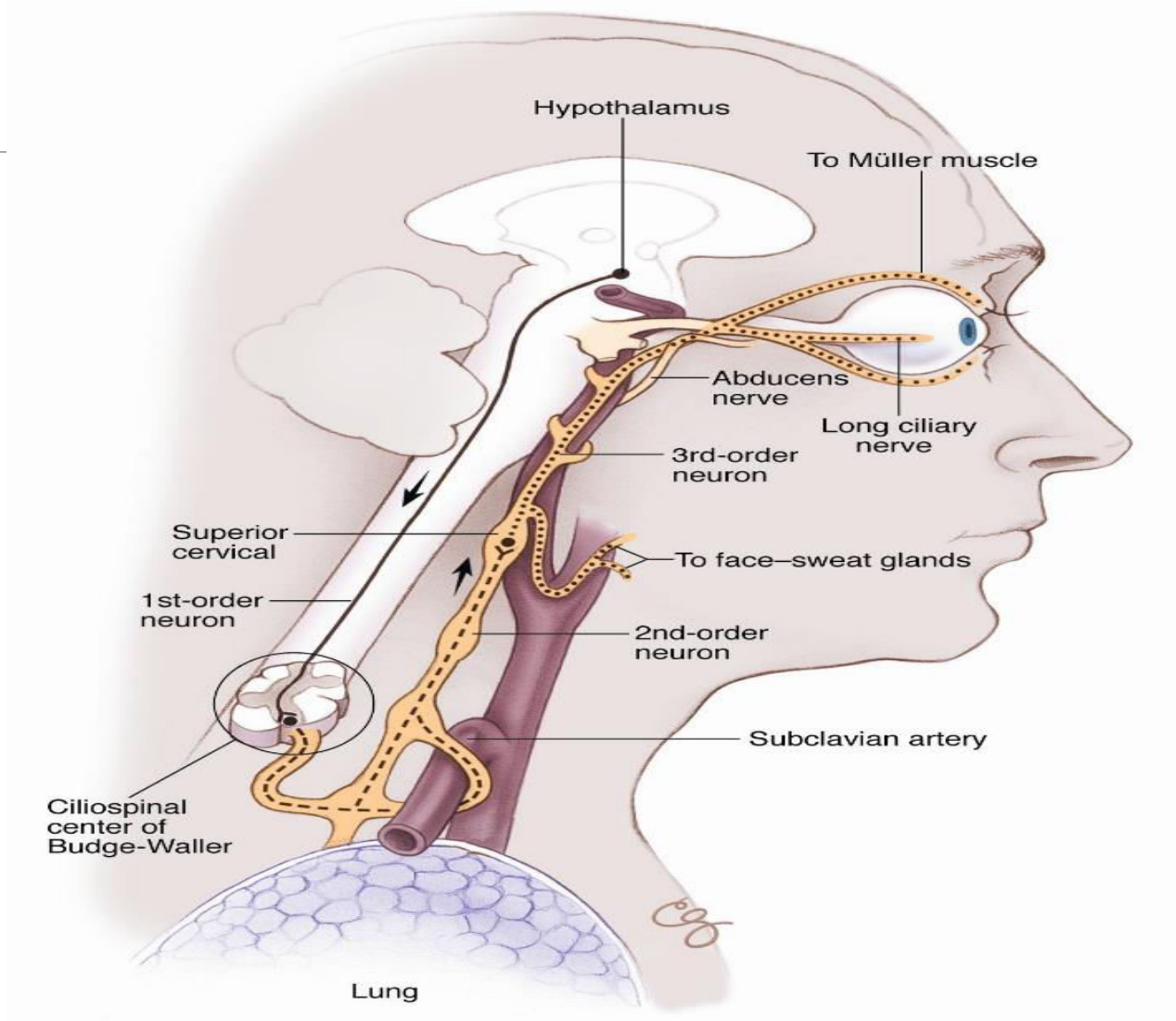
Parasympathetic pathway

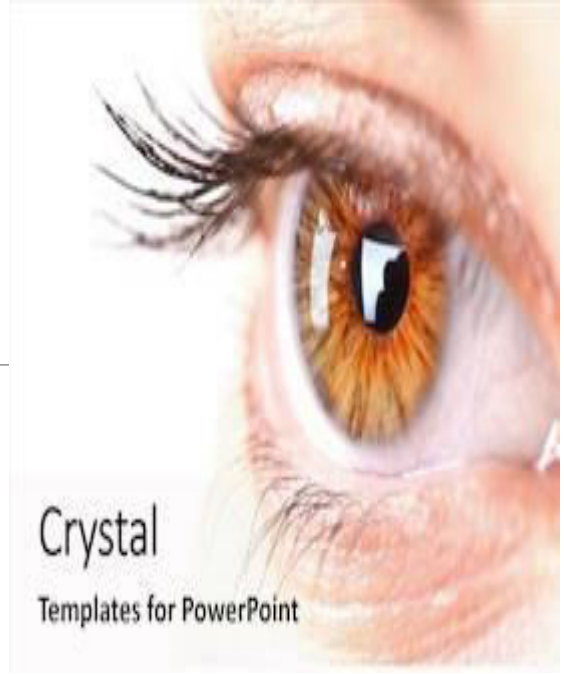
- Supply to sphincter muscle of the iris



Sympathetic pathway

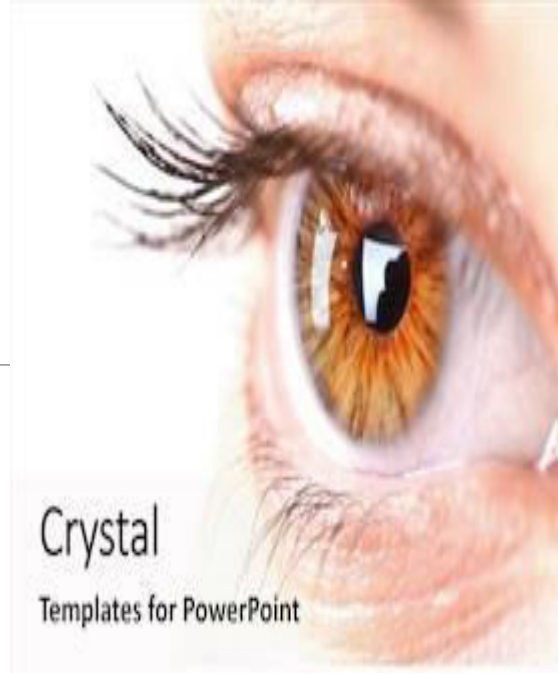
- Supply to Dilator muscle of the iris





ABNORMALITIES OF PUPILLARY REFLEXES

Abnormalities of Pupillary Reflexes



PARASYMPATHETIC PARESIS

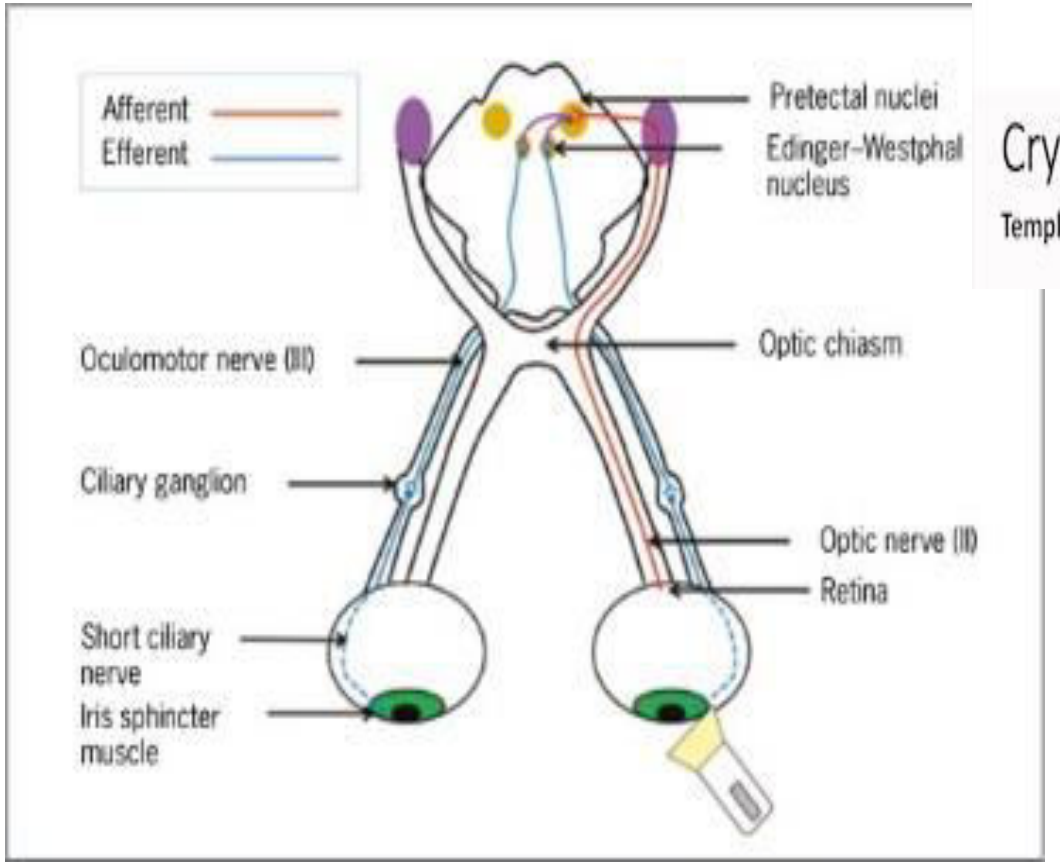
- AFFERENT PATHWAY DEFECTS
  - Total afferent pathway defect
  - Relative afferent pathway defect
  - Wernicke's hemianopic pupil
- EFFERENT PATHWAY DEFECTS
  - Tonic pupil
  - Oculomotor nerve palsy
  - Pharmacologic mydriasis
- PUPILLARY LIGHT-NEAR DISSOCIATION
  - Argyll Robertson pupil

SYMPATHETIC PARESIS

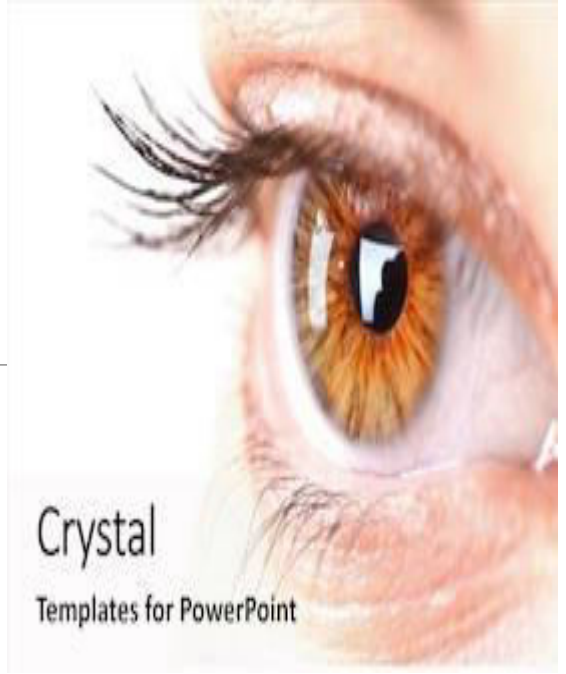
- Horner's syndrome

AFFERENT PATHWAY DEFECTS

- Total afferent pathway defect
- Relative afferent pathway defect
- Wernicke's hemianopic pupil



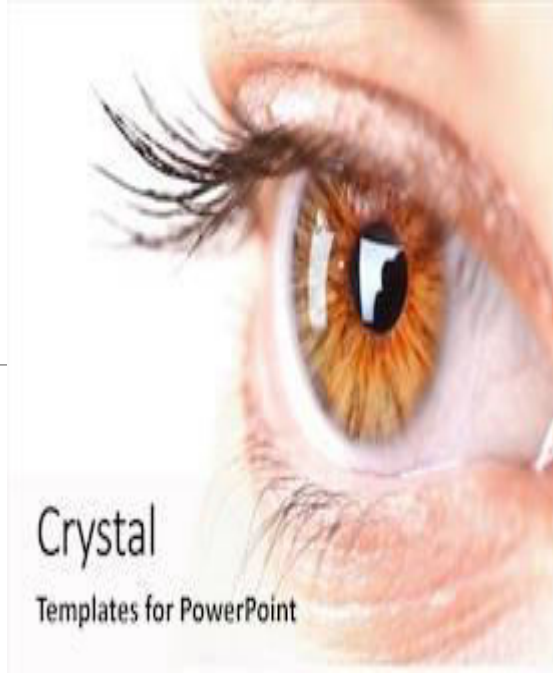
TOTAL/ABSOLUTE AFFERENT PATHWAY DEFECT



An absolute afferent pupillary defect (amaurotic pupil) is caused by a complete optic nerve lesion and is characterized by the following:

- The involved eye is completely blind (i.e. no light perception).
- Both pupils are equal in size.
- When the affected eye is stimulated by light neither pupil reacts.
- When the normal eye is stimulated both pupils react normally.
- The near reflex is normal in both eyes

Relative afferent pupillary defect



- A relative pupillary defect (Marcus Gunn pupil) is caused by an incomplete optic nerve lesion or severe retinal disease, but never by a dense cataract.
- The clinical features are those of an amaurotic pupil but more subtle.
- Thus the pupils respond weakly to stimulation of the diseased eye and briskly to that of the normal eye.
- The difference between the pupillary reactions of the two eyes is highlighted by the ‘swinging flashlight test’
- in which a light source is alternatively switched from one eye to the other and back, thus stimulating each eye in rapid succession.

A left relative defect is characterized by the following

Relative pupillary defect
(Marcus Gunn pupil)

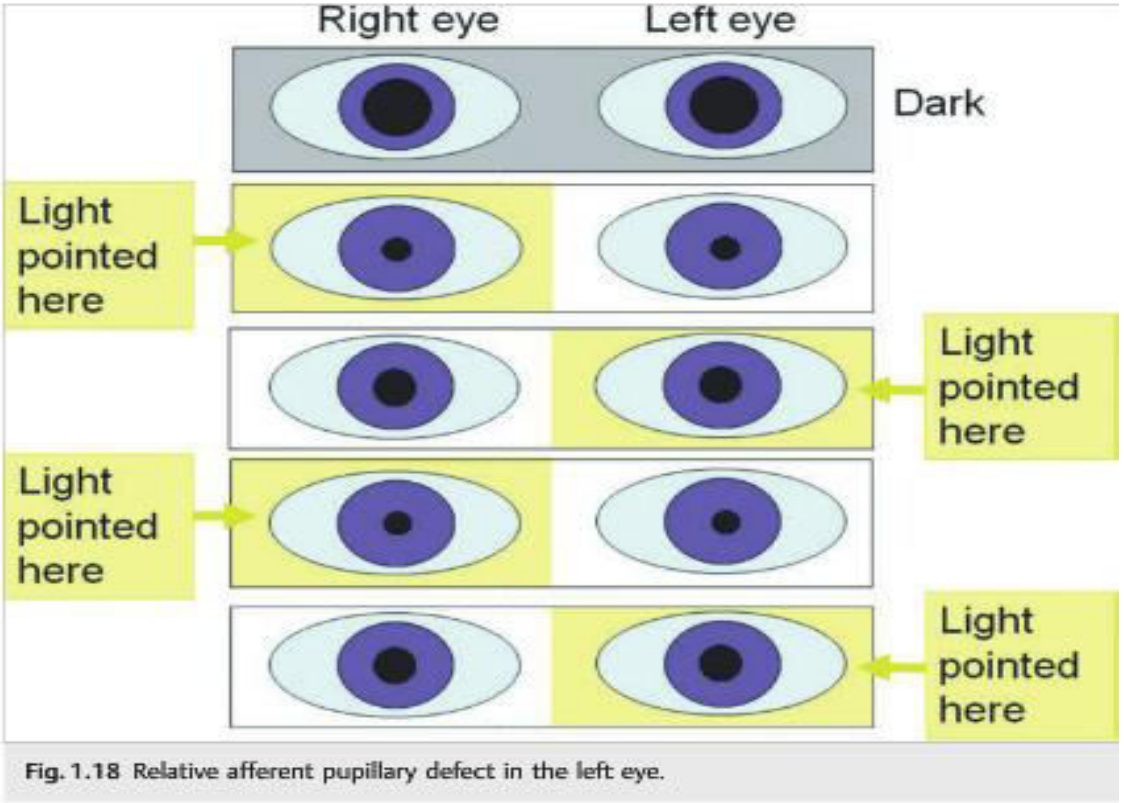
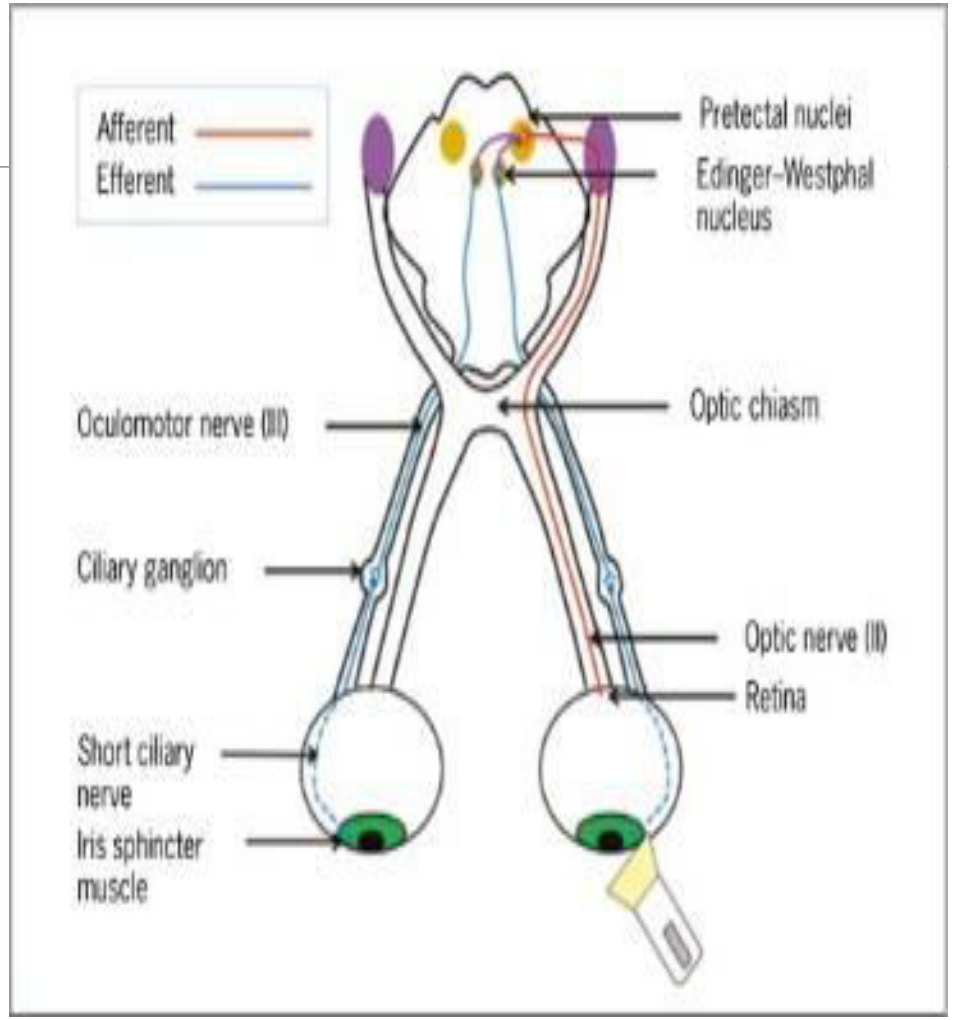


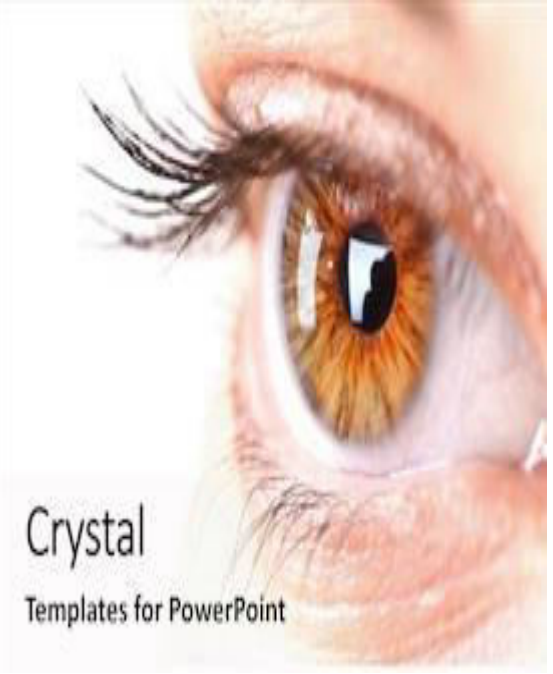
Fig. 1.18 Relative afferent pupillary defect in the left eye.

EFFERENT PATHWAY DEFECTS

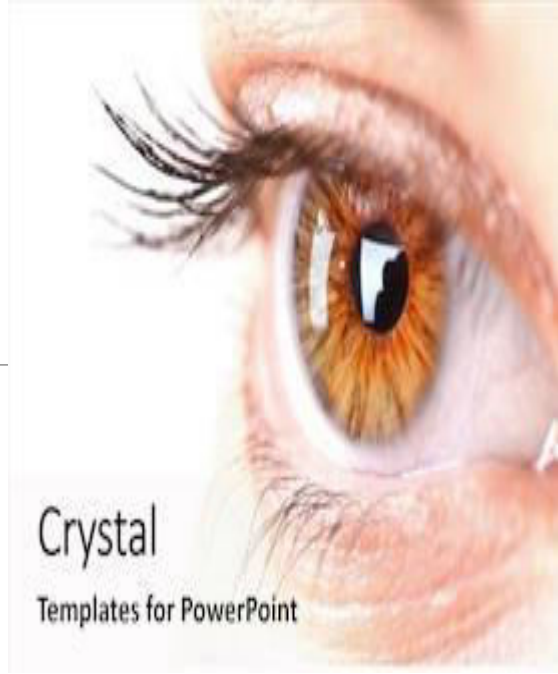
- Tonic pupil
- Oculomotor nerve palsy
- Pharmacologic mydriasis



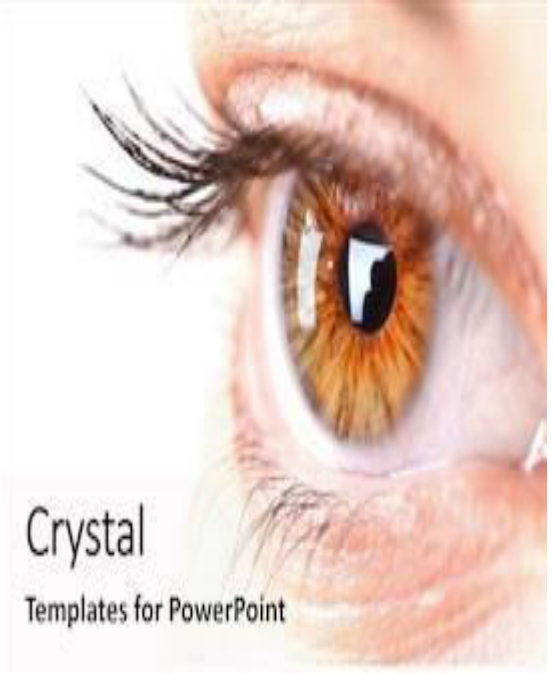
Adie /Tonic pupil



- An Adie pupil (tonic pupil, Adie syndrome) is caused by denervation of the postganglionic parasympathetic supply to the sphincter pupillae and the ciliary muscle,
- **Causes**
  - viral illness.
  - inherited in an AD pattern.
- Sites of dysfunction are presumed to be the ciliary ganglion.
- Affect young women



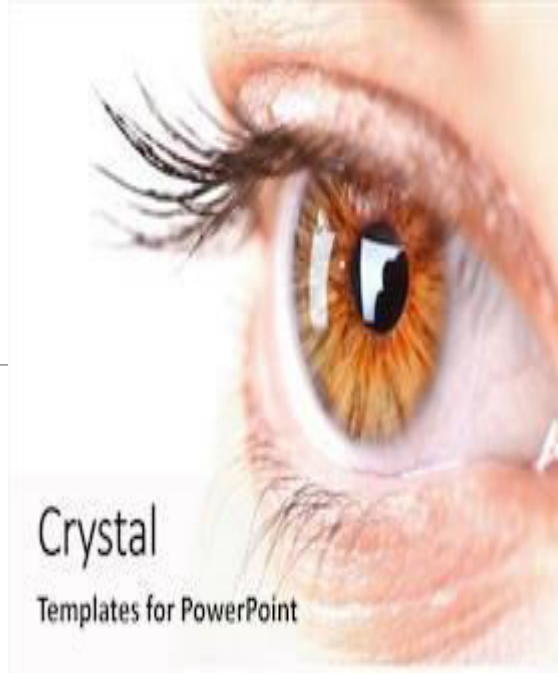
- **Symptoms**.
- Patients may notice anisocoria, or may have blurring for near due to impaired accommodation.
- **Signs**
- Large, regular pupil
- The direct light reflex is absent or sluggish
- On slit lamp examination, vermiform movements of the pupillary border are typically seen.
- Constriction is also absent or sluggish in response to light stimulation of the fellow eye (consensual light reflex)
- The pupil responds slowly to near, following which re-dilatation is also slow.



Pharmacological testing.

Instillation of 0.1–0.125% pilocarpine into both eyes leads to constriction of the abnormal pupil due to denervation hypersensitivity, with the normal pupil unaffected.

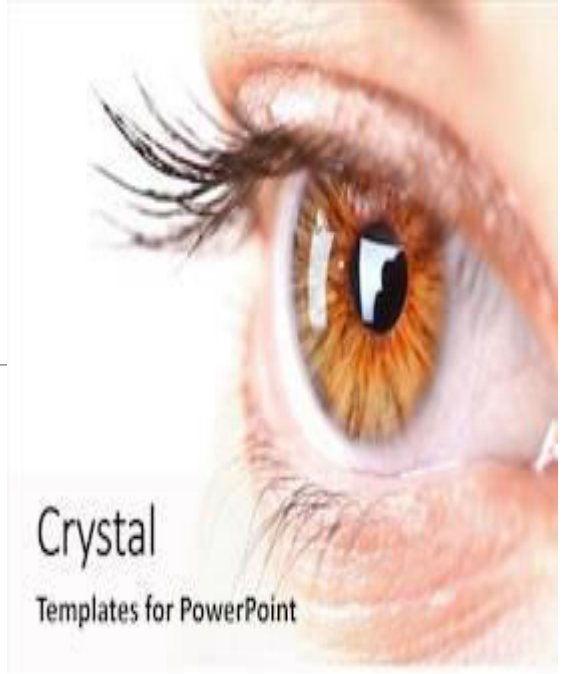
Pharmacological mydriasis



- Dilatation of one or both pupils due to instillation of a mydriatic agent can be inadvertent

- The pupil does not constrict in bright light or on accommodation and there is no response to any concentration of pilocarpine.

- There are no other neurological features.



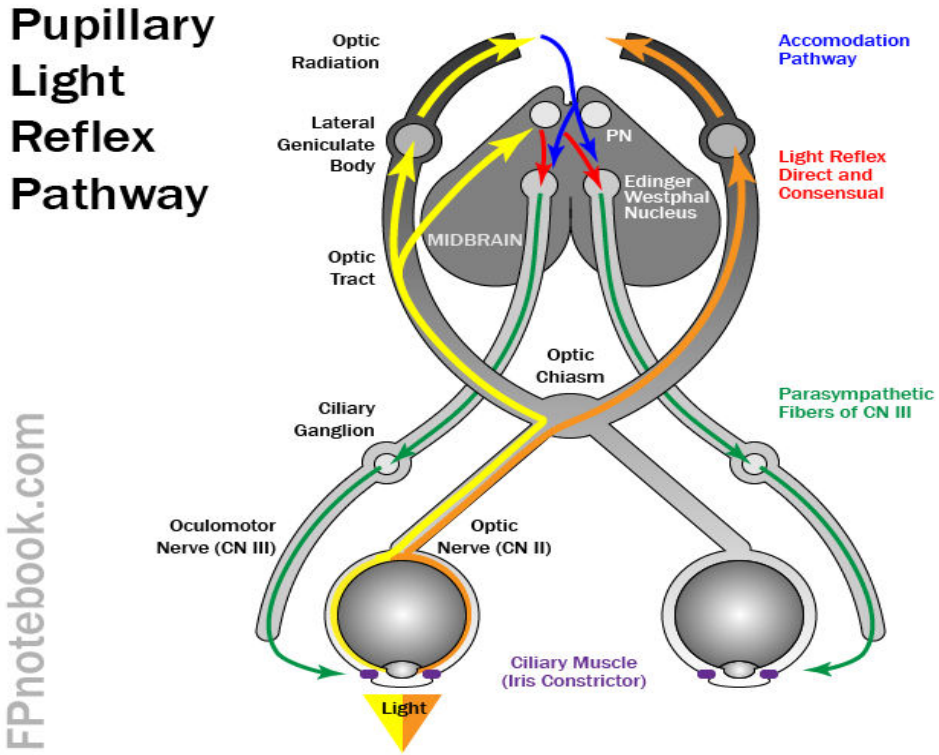
PUPILLARY LIGHT-NEAR DISSOCIATION

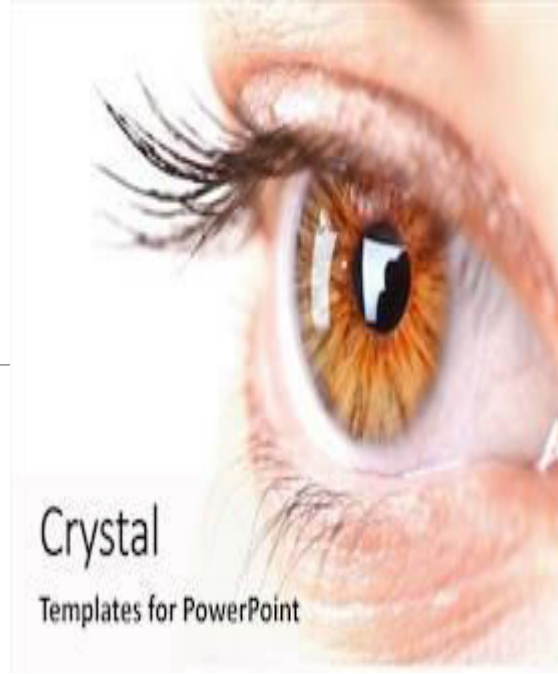
ARGYLL ROBERTSON PUPIL

Argyll Robertson pupils

- caused by neurosyphilis,

and have been attributed to a dorsal midbrain lesion that interrupts the pupillary light reflex pathway but spares the more ventral pupillary near reflex pathway – light–near dissociation intact.

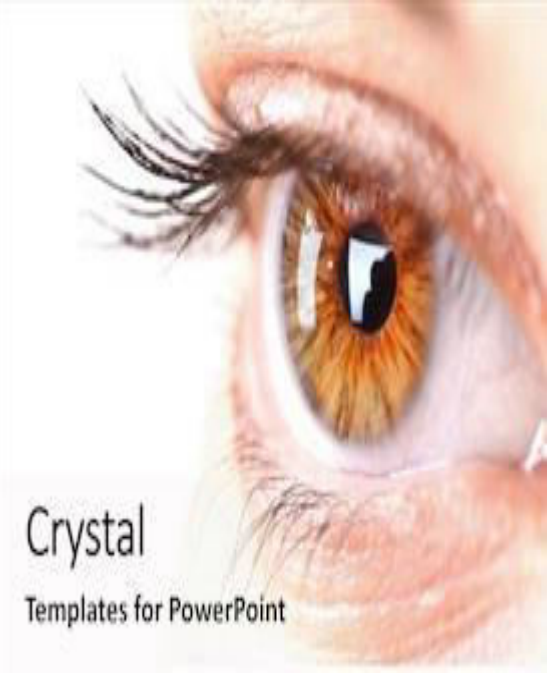




Light-near dissociation (LND) is **a pupillary sign that occurs**

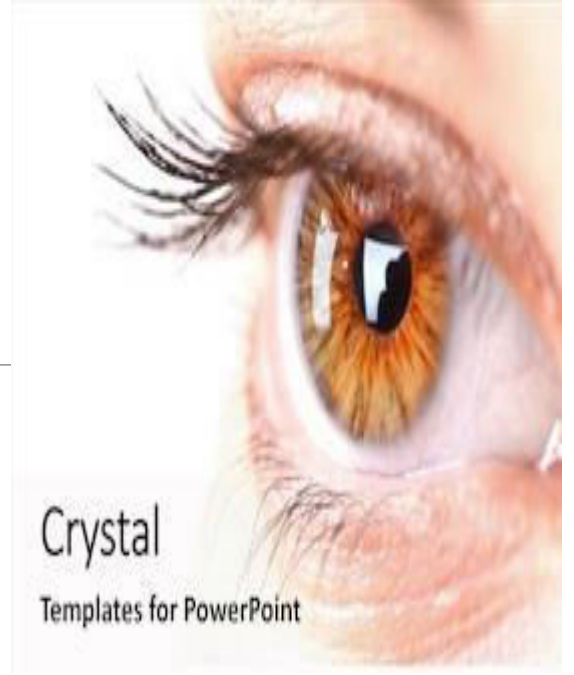
when the pupillary light reaction is impaired

while the near reaction (accommodative response) remains normal.



- **Sign ;**
- In dim light both pupils are small and may be irregular.
- In bright light neither pupil constricts,
- but on accommodation (near target) both constrict.
- The pupils do not dilate well in the dark,
- but cocaine induces mydriasis unless marked iris atrophy is present.

Causes of light–near dissociation

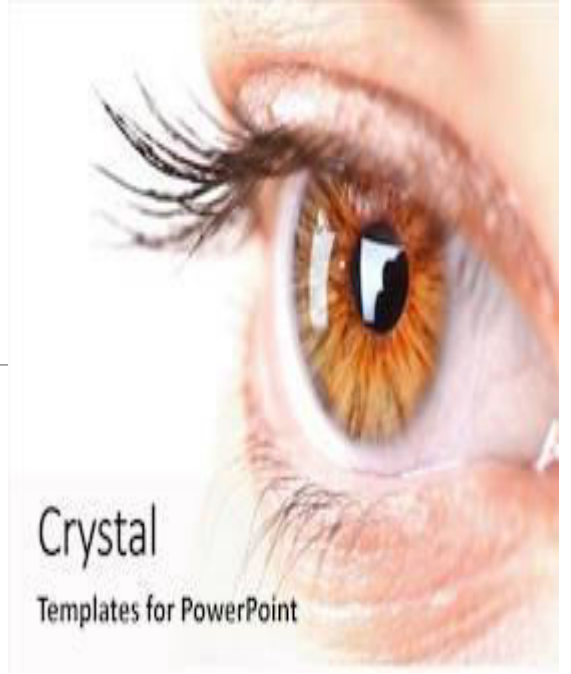


CAUSES

- Unilateral
  - Afferent conduction defect
  - Adie pupil
  - Herpes zoster ophthalmicus
  - Aberrant regeneration of the third cranial nerve

CAUSES

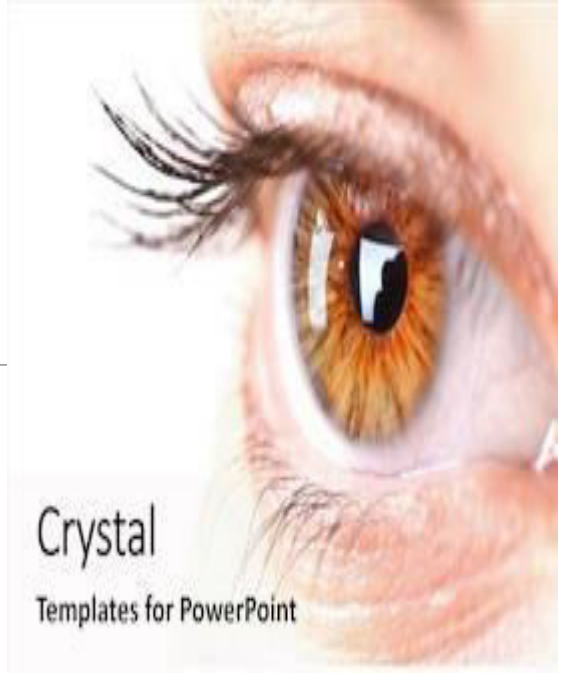
- Bilateral
  - Neurosyphilis
  - Type 1 diabetes mellitus
  - Myotonic dystrophy
  - Parinaud (dorsal midbrain) syndrome
  - Familial amyloidosis
  - Encephalitis
  - Chronic alcoholis



SYMPATHETIC PARESIS

HORNER'S SYNDROME

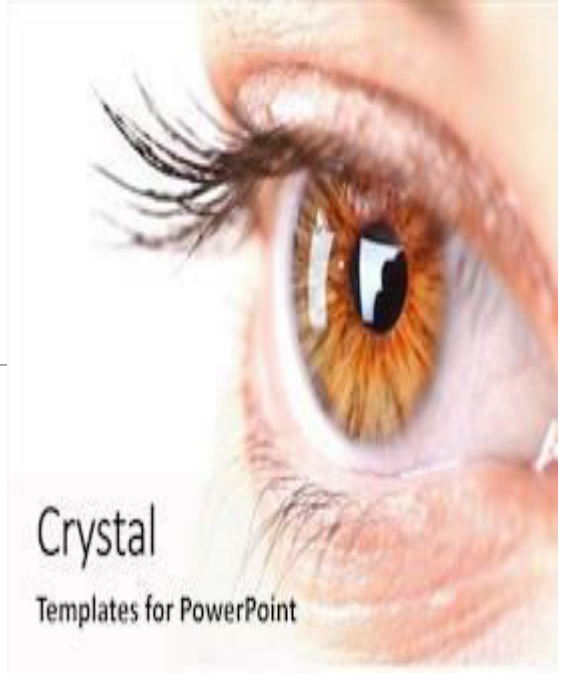
Horner syndrome (oculosympathetic palsy)



- Sympathetic supply involves three neurones

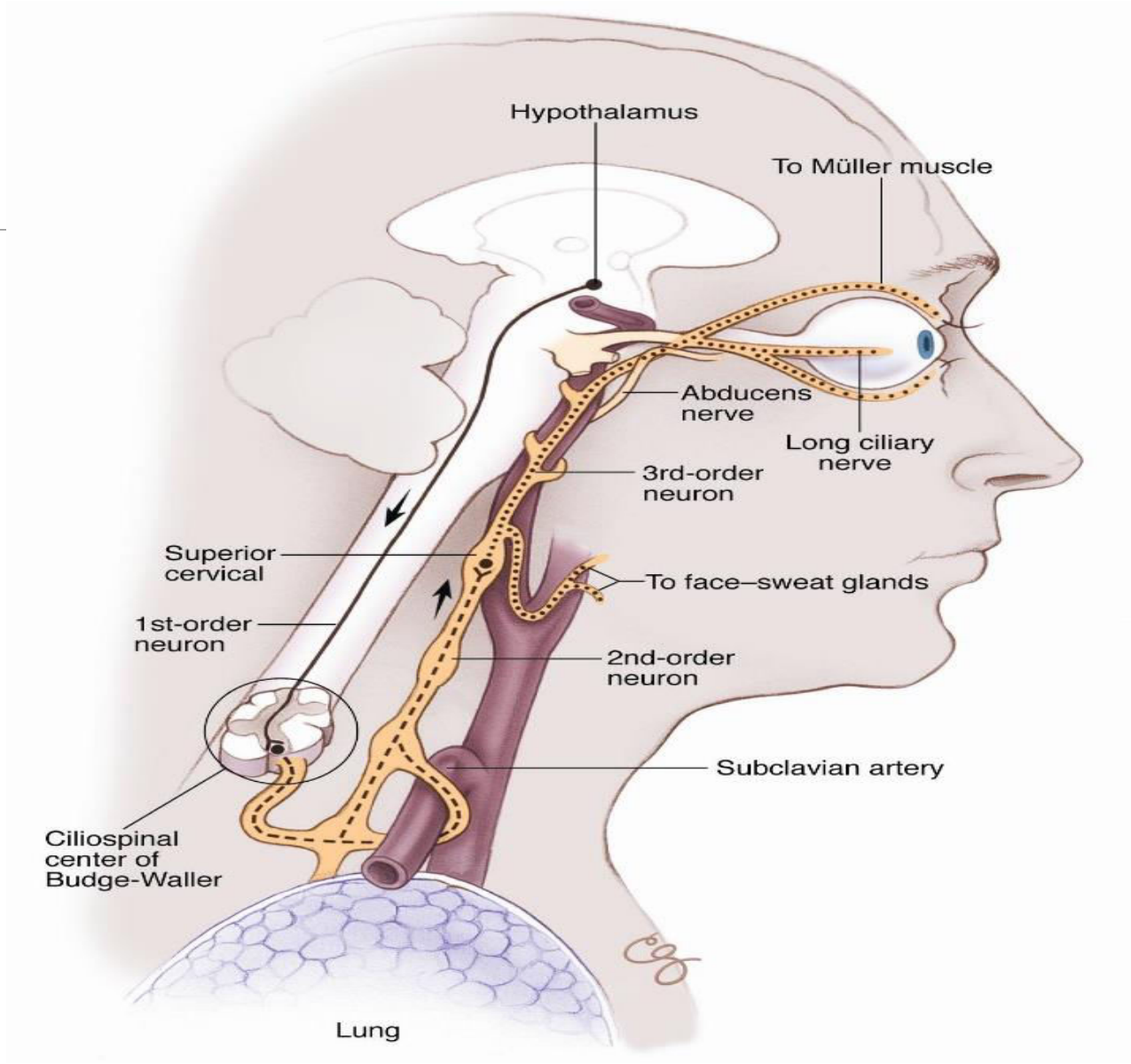
- First (central) starts
  - in the posterior hypothalamus and descends, uncrossed, down the brainstem to terminate in the ciliospinal centre of Budge, in the intermediolateral horn of the spinal cord, located between C8 and T2.

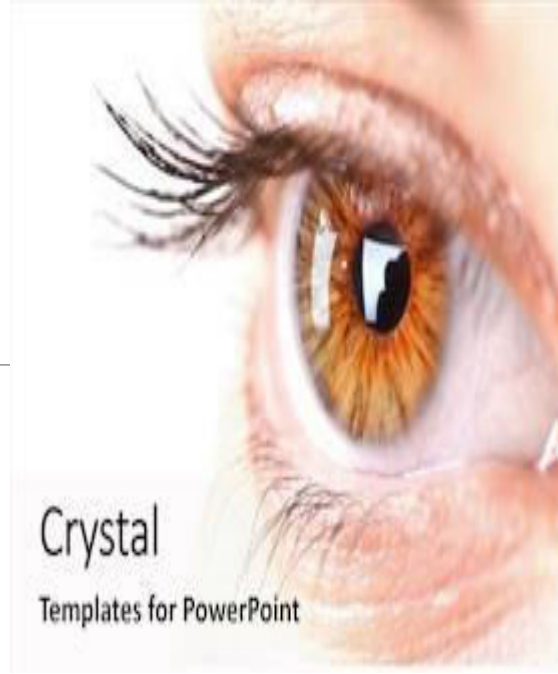
- Second (preganglionic)
  - passes from the ciliospinal centre to the superior cervical ganglion in the neck. During its long course, it is closely related to the apical pleura where it may be damaged by bronchogenic carcinoma (Pancoast tumour) or during surgery on the neck.



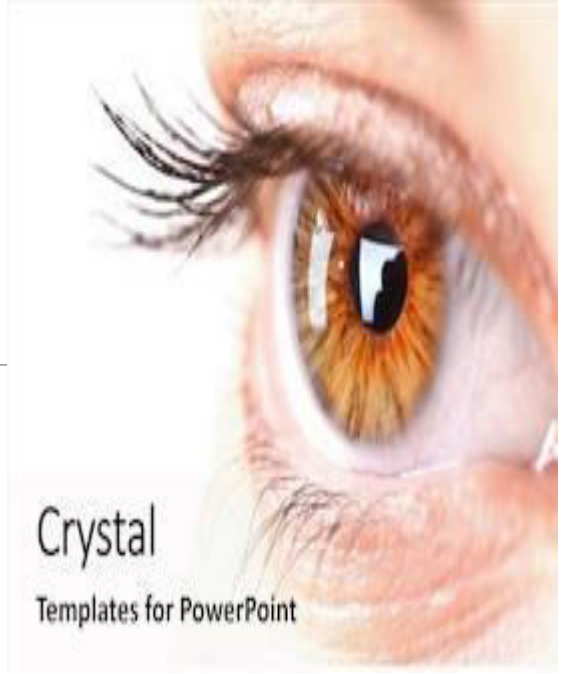
- Third (postganglionic)
  - ascends along the internal carotid artery to enter the cavernous sinus where it joins the ophthalmic division of the trigeminal nerve.
  - The sympathetic fibres reach the ciliary body and the dilator pupillae muscle via the nasociliary nerve and the long ciliary nerves.

Sympathetic supply



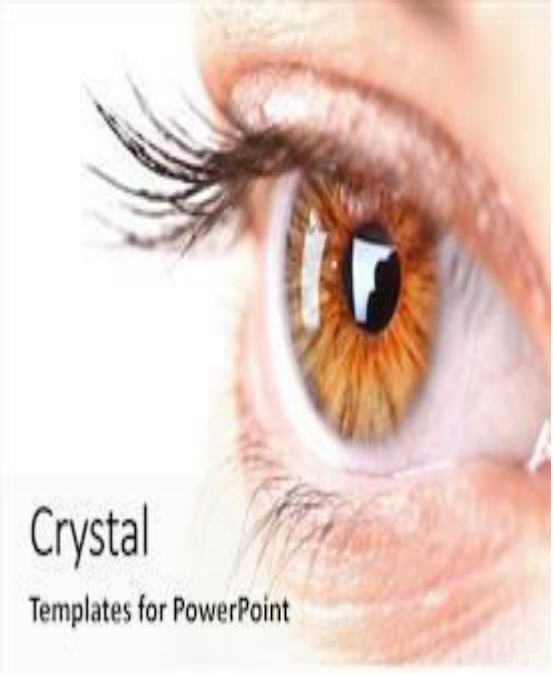


Causes of Horner syndrome

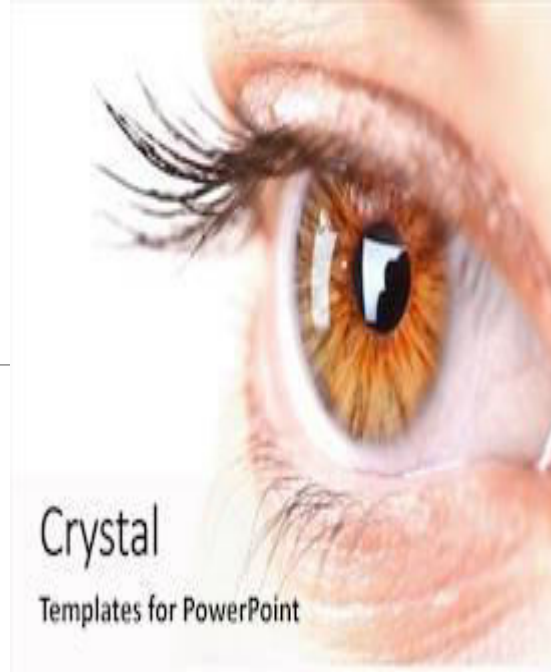


- Central (first-order neuron)
  - Brainstem disease – commonly stroke (e.g. lateral medullary infarction), but also tumor, demyelination
  - Syringomyelia
  - Lateral medullary (Wallenberg) syndrome
  - Cervical spinal cord lesion
  - Diabetic autonomic neuropathy

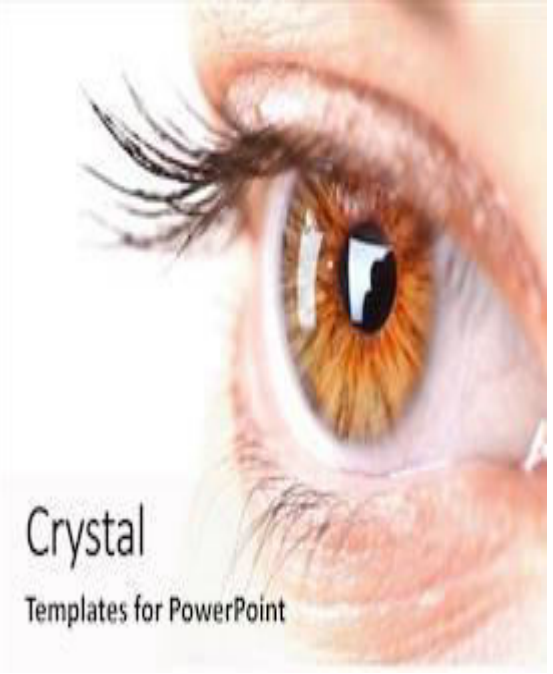
- Preganglionic (second-order neuron)
  - Pancoast tumor
  - Carotid and aortic aneurysm and dissection
  - Thoracic spinal cord lesion
  - Miscellaneous neck lesions (thyroid tumor, enlarged lymph nodes, trauma, postsurgical)



- Postganglionic (third-order neuron)
  - Internal carotid artery dissection
  - Nasopharyngeal tumor
  - Cavernous sinus mass
  - Otitis media
  - Cluster headache (migrainous neuralgia)



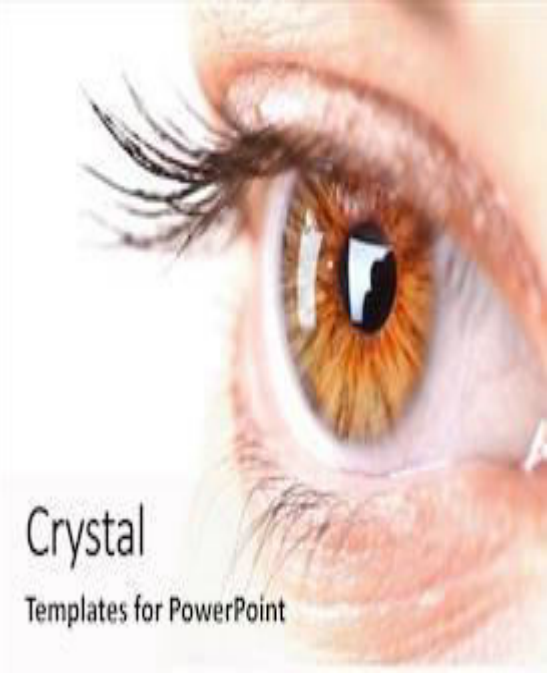
- **Presentation**
- The majority of cases are unilateral.
- Bilateral involvement occur in cervical spine injuries and autonomic diabetic neuropathy.
- Mild ptosis (usually 1–2 mm) as a result of weakness of Müller muscle,
- Miosis due to the unopposed action of the sphincter pupillae with resultant anisocoria.
- A key examination finding is that anisocoria is accentuated in dim light, since in contrast to a normal fellow pupil the Horner pupil will dilate only very slowly; the dark-induced anisocoria diminishes with time spent in the dark environment



- Pupillary constriction to light and near stimuli is normal.
- Hypochromic heterochromia (irides of different colour,

  the Horner being lighter) may be seen if congenital or longstanding .
- Slight elevation of the inferior eyelid (inferior ptosis)

  as a result of weakness of the inferior tarsal muscle.

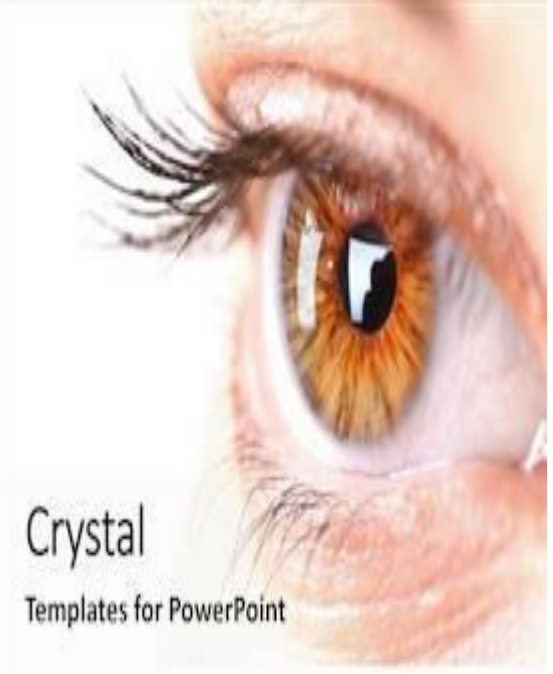


- inferior Reduced ipsilateral sweating,

- Because the psudomotor fibres supplying the skin of the face run along the external carotid artery this occurs only if the lesion is below the superior cervical ganglion;

- patients may mistakenly interpret the normal side to be sweating excessively.

Pharmacological tests



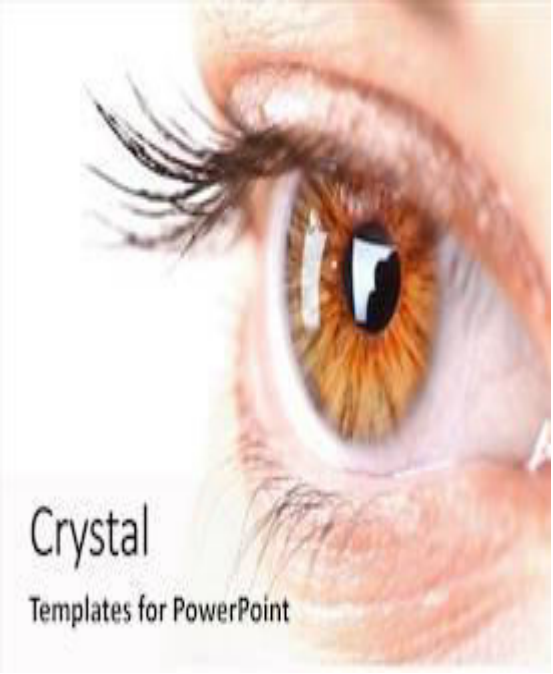
- **Apraclonidine or cocaine test**:

  used to confirm the diagnosis

- **Hydroxyamphetamine and adrenaline**

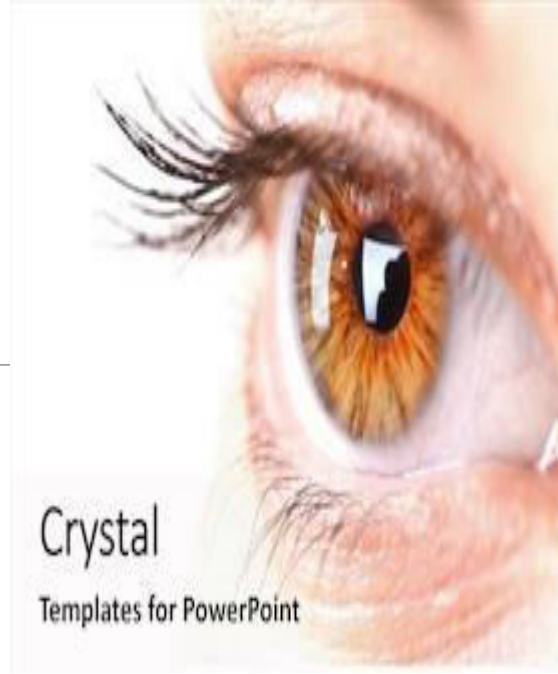
  may be used to differentiate a preganglionic (abnormal first- or second-order neurone) from a postganglionic lesion (abnormal third-order neurone)

Pharmacological tests

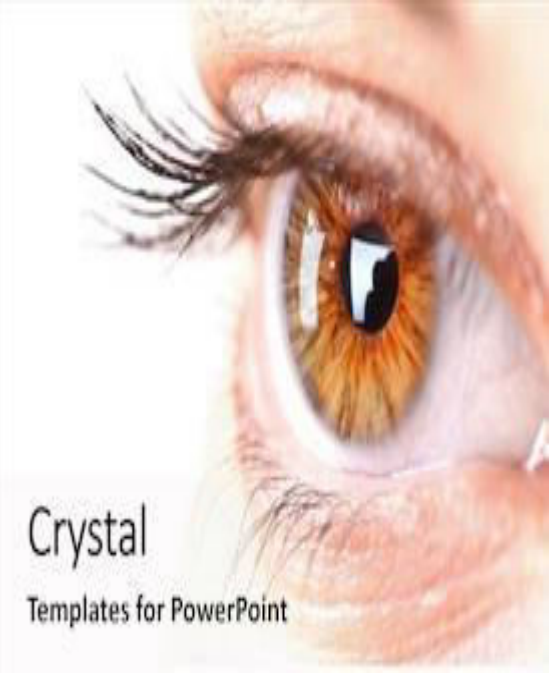


Apraclonidine or cocaine test

- Apraclonidine 0.5% or 1.0%.
- One drop is instilled into both eyes to confirm or refute the presence of Horner syndrome.
- The pupils should be checked at 30 minutes and, if negative, rechecked at 45 minutes.
- Apraclonidine penetrates the blood–brain barrier, so should be used only with great caution in infants under one year of age.



- **Result:**
- A Horner pupil will dilate but a normal pupil is essentially unaffected.
- The ptosis commonly also improves.
- Sensitivity is around 90% and
- specificity close to 100%
- **Explanation:** Alpha-1 receptors are upregulated in the denervated dilator pupillae.



➢Cocaine test

➢4% is instilled into both eyes;

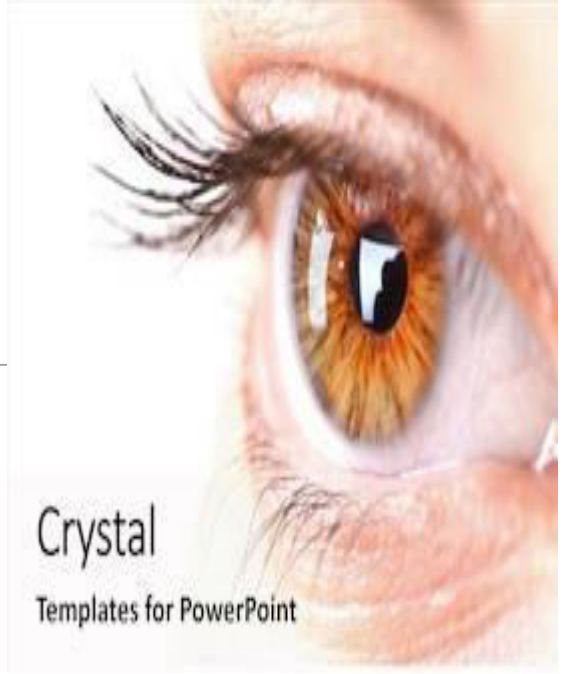
➢**Result:**

- ➢ The normal pupil will dilate but the Horner pupil will not;
- ➢Anisocoria of as little as 0.8 mm in a dimly lit room is significant.

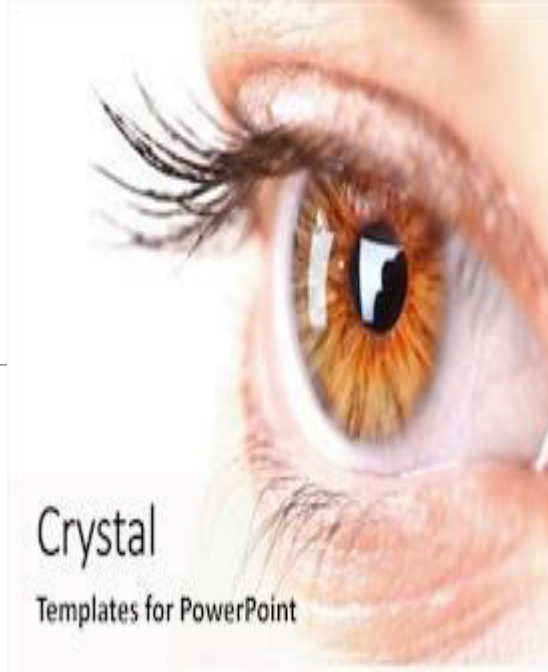
➢**Explanation:**

- ➢ Cocaine blocks the re-uptake of noradrenaline secreted at the postganglionic nerve ending, which accumulates and causes dilatation of a normal pupil.
- ➢In Horner syndrome, there is no noradrenaline being secreted, so cocaine has no effect.

Phenylephrine 1%



- More readily available than hydroxyamphetamine and adrenaline
- It distinguish pre- and postganglionic lesions.
- Prepared by dilution of commonly available 2.5% or 10% solution.
- Result:
  - In an established (10 days) postganglionic lesion, the **Horner pupil will dilate** and ptosis may be temporarily relieved.
  - A central or preganglionic Horner pupil and a normal pupil will not dilate or will dilate minimally.
- Explanation:
  - In postganglionic Horner syndrome the dilator pupillae muscle develops denervation hypersensitivity to adrenergic neurotransmitters due to its dysfunctional local motor nerve.



- **Hydroxyamphetamine 1%.**
- Two drops are instilled into each eye.
- It may be slightly more sensitive than phenylephrine testing.
- **Result:**
  - A normal or preganglionic Horner pupil will dilate but a postganglionic Horner will not.
- **Explanation:**
  - Hydroxyamphetamine potentiates the release of noradrenaline from functioning postganglionic nerve endings.
  - In a lesion of the third-order neurone (postganglionic) there is no release of noradrenaline from the dysfunctional nerve
- Adrenaline 0.1% has an action similar to that of phenylephrine

Thanks