BrienHolden Vision Institute

REVIEW OF PUPILS

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1. DEFINITIONS

- Miosis - Constriction of the pupil of the eye, resulting from a normal response to an increase in light or caused by certain drugs or pathological conditions.
- Mydriasis - prolonged abnormal dilation of the pupil of the eye induced by a drug or caused by disease.
- Direct pupillary reflex - constriction of the illuminated pupil
- Consensual pupillary reflex - constriction of the opposite pupil to that being illuminated
- Near pupillary reflex - constriction of the pupil when viewing a close object
- Anisocoria - asymmetric sizes of pupils
- Sphincter pupillae- a broad flat band of smooth muscle in the iris that surrounds the pupil of the eye and is responsible for pupillary constriction. This muscle is controlled by the parasympathetic nervous system
- Dilator pupillae- an outer band of radial fibres, this smooth muscle in the iris is responsible for dilation of the pupil. This muscle is controlled by the sympathetic nervous system.

2. INTERESTING PUPIL FACTS

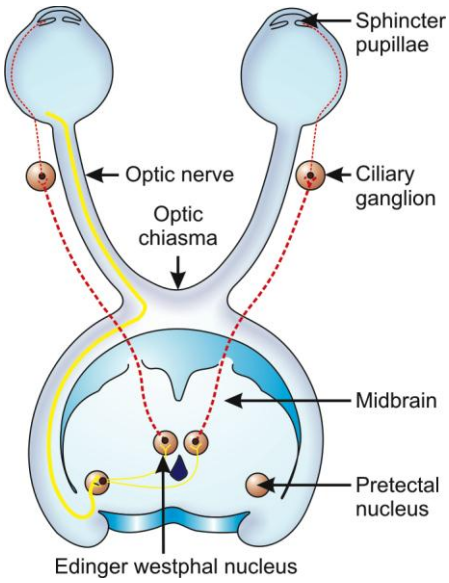
- Function of the pupil:
  - Modifies amount of light entering the eye; increases sensitivity of the eye
  - Increases depth of focus
  - Minimizes chromatic and spherical aberrations
- Newborns and the elderly are miotic, with the elderly having 1/3rd size of that of a 20 year old
- Pupils are miotic during sleep, with blinking, and with forced closure
- Retinal stimulation produces a pupil response, however, a larger pupil response is noted when there is foveal stimulation
- Accommodation and convergence can change pupillary size
- Physiological and emotional states can change pupillary size:

Dilation	**Constriction**
Sensory nerve stimulation	Eye irritation (Trigeminal)
Vestibular stimulation	Sleepiness
Emotional stimulus	
Systemic pain	

- Various drugs can affect the pupils:
  - Parasympathomimetics imitate the effects of the parasympathetic nervous system
  - Sympathomimetics – imitate the effects of the active sympathetic nervous system
  - Parasympatholytics – inhibit the action of the parasympathetic nervous system
  - Sympatholytics – inhibit the action of the sympathetic nervous system

3. INNERVATION PATHWAYS

A. PARA-SYMPATHETIC



Afferent pathway - yellow line
Efferent pathway – dotted red line

***Figure 1.1** Light reflex pathway*

LIGHT REFLEX (FIG. 1.1)

- Originates from any point on the retina as light stimulates it.
- **Afferent pathway:**
  - Begins in the ganglion cell layer, through the optic nerve, then fibres decussate at the chiasm.
  - Nasal fibres of each retina cross but temporal ones do not cross.
  - Posterior to the chiasm, afferent fibres pass into the optic tract, then separate from the tract in its posterior third, just anterior to the Lateral Geniculate Nucleus (LGN).
  - They enter the midbrain, and then go to the pretectal nucleus.
  - Synapses occur in the pretectal nuclei and the fibres hemidecussate through the posterior commissure and terminate in the Edinger-Westphal nuclei.
- **Efferent pathway**
  - Begins at the Edinger-Westphal nuclei.
  - Parasympathetic fibres of CN III course through the inferior division of the nerve when it bifurcates in the cavernous sinus.
  - In the sinus, CN III is closely related to the first and second divisions of CN V.
  - CN III enters the orbit through the superior orbital fissure and synapses at the ciliary ganglion.
  - Postganglionic fibres pass to the smooth muscle fibres of the iris sphincter through the short ciliary nerve. These nerves travel forward in the suprachoroidal space and release acetylcholine at the neuromuscular junction.

CLINICAL NOTE:
If one of the afferent pathways is affected, you will see an afferent pupillary defect (APD) during your pupillary testing. However, be sure to rule out other factors that can simulate a false APD, for example: amblyopia, a previously patched eye, a tilted light source directed off the macula. Efferent pupillary defects can be part of a CN III nerve palsy or internal ophthalmoplegia.

REVIEW QUESTION:
What is an afferent pupillary defect (APD)?

NEAR REFLEX	• **NEAR REFLEX** – Initiated by the attempt to fixate a near object. – A triad of responses occurs: convergence, accommodation, and miosis, called the NEAR TRIAD. – The afferent path is similar to that of the light reflex back to the posterior third of the optic tract. Fibres then pass to the occipital cortex, through the prestriate area to the premotor area of the frontal lobe. – From here, the fibres pass through the corona radiata and internal capsule to the oculomotor nucleus (CN III). – The efferent path from the oculomotor nucleus is likely the same as that of the light reflex; although it is possible that a separate path for near fibres exists and that these fibres do not form a synapse in the ciliary ganglion. The final path is through the third nerve directly or through the ciliary ganglion to the ciliary and sphincter pupillae muscles.

CLINICAL NOTE:
Argyll-Robertson Pupil (Light-near dissociation) occurs along this pathway because the near reflex fibres are more ventrally located than the light fibres. This means that a lesion can impact the afferent light reflex fibres but not the near reflex fibres. Argyll-Robertson pupils are small and irregular and do not react to light but have a brisk near-point response. The cause is usually syphilis.

CLINICAL NOTE:
Parinaud's syndrome involves light/near dissociation (Argyll-Robertson pupil), paralysis of upward gaze and lid retraction (which may look like a ptosis on the opposite eye). It is caused by a dorsal midbrain problem, possibly a pinealoma. Refer for a CT or MRI to rule this out.

B. SYMPATHETIC:

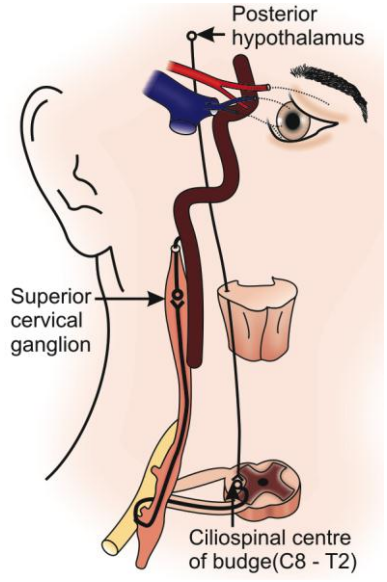


Figure 1.2 *Pupillo-dilator pathway*

PUPILLO-DILATOR PATHWAY (FIG. 1.2)

- Begins with afferent impulses from the cortex that terminate in the hypothalamus.
- Sympathetic outflow then begins in the posteriolateral area of the hypothalamus and the preganglionic fibres pass uncrossed through the tegmentum of the midbrain and pons.
- These preganglionic fibres then pass through the lateral portion of the medulla and terminate in the dilator centre of the spinal cord, lying in the lateral column of the cord, at the junction of the thoracic and cervical regions (at the C8 to T2 levels of the ciliospinal centre of Budge).
- Fibres exit the cord in this area, passing through the white rami communicans of the uppermost thoracic nerves and up the cervical sympathetic trunk.
- Fibres then reach the superior cervical ganglion at the base of the skull, where the first synapse takes place.
  - Postganglionic fibres run upwards around the internal carotid artery and eventually join the trigeminal ganglion; by passing into the orbit through the nasociliary nerve they enter through the long ciliary nerves, terminating in the iris dilator.

CLINICAL NOTE:
Horner's Syndrome results from a lesion within the sympathetic pathway and has a clinical triad of ptosis, miosis and anhydrosis. The site of the lesion can be life threatening so be sure to do your differential pharmacological testing!

B. SYMPATHETIC:	• Signs of Horner's syndrome: anisocoria greater in dim illumination (because a small pupil does not dilate as well as a larger, normal pupil). • **Causes of Horner's syndrome:** – **First-order neuron:** - Stroke (vertebrobasilar artery problem or infarct) - Tumour - Severe osteoarthritis of the neck - Multiple Sclerosis (MS) - Trauma ****NOTE: Patient has sudden onset vertigo and sensory defects** – **Second-order neuron:** - Lung tumour or breast metastasis - Trauma - Thyroid adenoma - Neurofibroma ****NOTE: If patient also has arm pain suspect pancoast tumour** – **Third order neuron:** - Headache/migraine (especially if ipsilateral (IPSI)) - Cavernous sinus tumour - Skull fracture - Herpes zoster - Otitis media - Congenital Horner's Syndrome: o Trauma during delivery (may also have iris heterochromia)
OTHER PUPIL ANOMALIES	• **Adie's Tonic Pupil** – Occurs usually in young women 20-40 years old – Blurry unilateral VA at near – Anisocoria (with the Adie's pupil being the larger pupil) – Recent upper respiratory tract infection (URI) – Pupil responses: - Vermiform segmental pupil - Slow/tonic near response, greater than light response - Smaller with time - Deep tendon reflexes diminished - Decreased corneal sensation - Test with diluted Pilocarpine. Eye is supersensitive to 0.125% solution – Causes: - Idiopathic - Orbital trauma or infection - Herpes Zoster - Diabetes - Autonomic Neuropathies

CLINICAL NOTE:
The best place to observe an Adie's pupil is under the slit lamp. Focus your light on the pupillary margin and you will see it "ratchet" or squiggle as it tries to constrict.

OTHER PUPIL ANOMALIES	– Physiologic Anisocoria - Pupil size disparity is same in dark and light, usually 1 mm - Normal pupillary reactions - No APD - Often there since birth