



DISORDERS OF PUPILLARY REACTIVITY: LIGHT-NEAR DISSOCIATION



Eye Learn

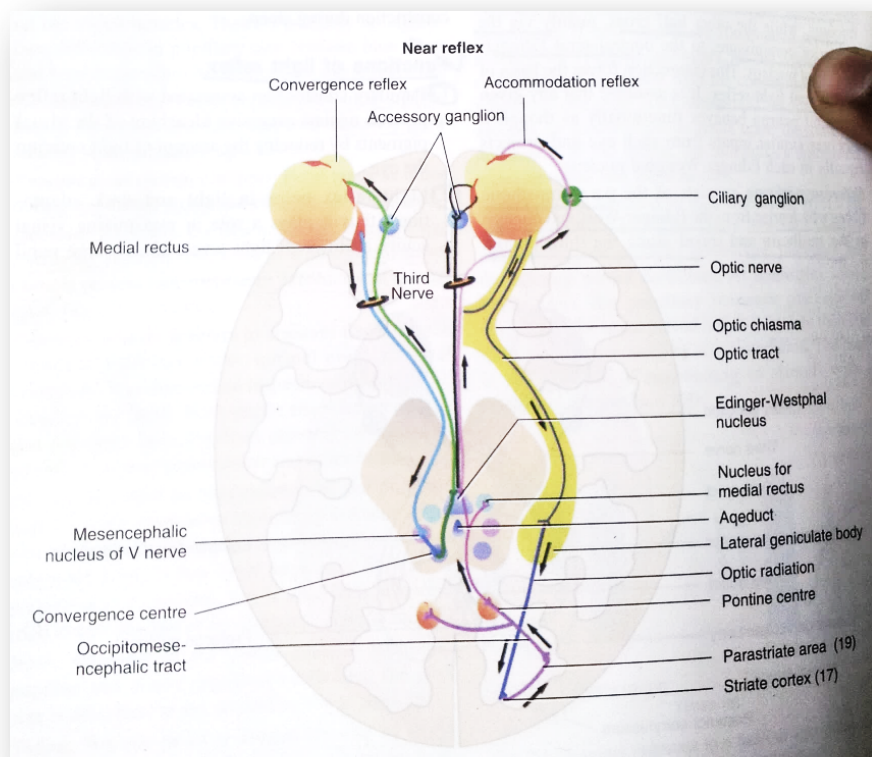
All about the Eye

Dr. Krati Gupta

Dr. Saurabh Deshmukh

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	Convergence reflex	Accommodation reflex
Afferent fibers	Still not elucidated. Assumed that the afferents from the medial rectus travel via the III N to the mesencephalic nucleus of V n to a presumptive convergence center in the tectal or pretectal region	Extend from retina to parastriate cortex via optic nerve, optic chiasma, optic tract, LGB, optic radiation, Striate cortex.
Internuncinal fibers	From so called convergence center to EWN	Relay impulses from parastriate cortex to EWN of both sides via occipitomesencephalic tract and pontine center.
Efferent fibers	From EWN Via IIN relay in accessory ganglion to reach sphincter pupillae.	From EWN Via IIN relay in ciliary & accessory ganglion to reach ciliary muscle & sphincter pupillae.

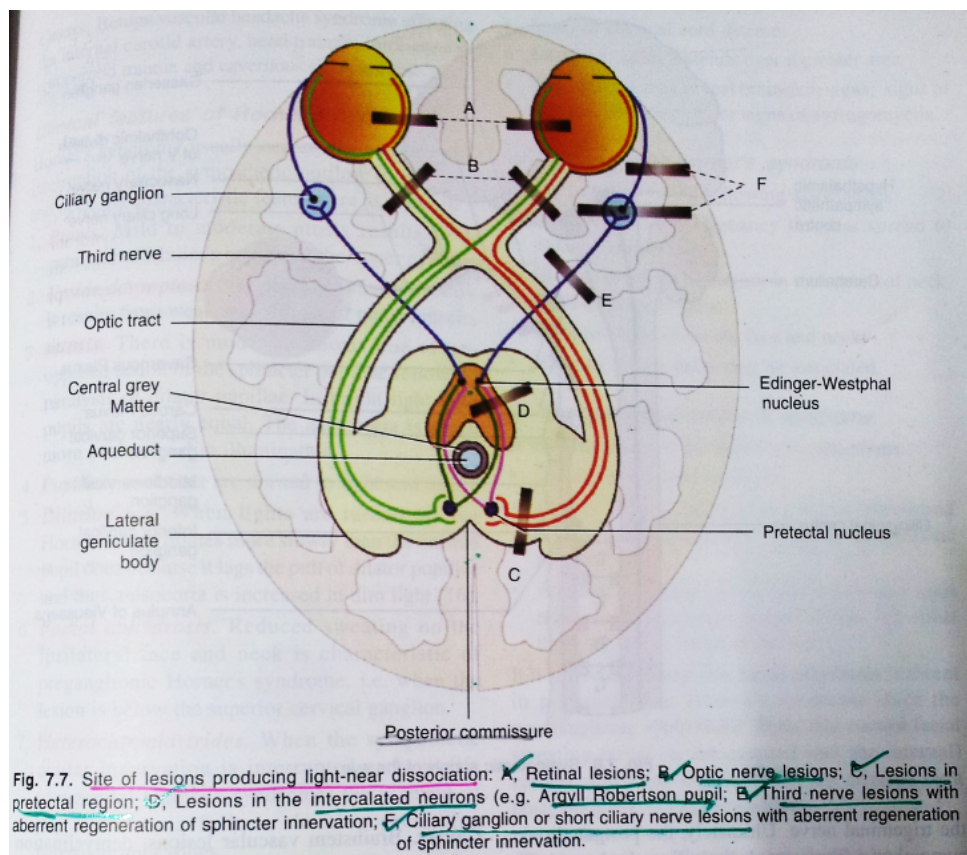
1. Give physiological basis and causes of light-near dissociation (4+2) J2018

Light-near dissociation

1. Light-near dissociation occurs when a near response exceeds the best pupillary constriction that bright light can produce.
2. The near reflex, a synkinesis rather than a true reflex, is activated when gaze is changed from a distant to a near target.
3. It comprises accommodation, convergence and miosis.
4. The pupillary constriction in such cases is however not dependent on the associated convergence, accommodation or the amount of ambient light present in the environment
5. Vision is not a prerequisite and there is no clinical condition in which the light reflex is present but the near response absent. Although the final pathways for the near and light reflexes are identical (i.e. third nerve, ciliary ganglion, short ciliary nerves), the centre for the near reflex is ill-defined.
6. There are probably two supranuclear influences: the frontal and occipital lobes.
7. The midbrain centre for the near reflex is probably located more ventrally than the pretectal nucleus, the afferent limb of the light reflex and comprises a supranuclear connection between the neurons subserving the ciliary body muscles (accommodation), the pupillary sphincters (miosis) and the medial rectus muscle (convergence).
8. This forms the anatomical basis of some instances of light near dissociation (dorsal midbrain syndrome where the pupillary light reflexes are affected but the near reflex is not because of lesions at the level of the dorsal midbrain).

9. It may explain why compressive lesions such as pinealomas, preferentially involving the dorsal internuncial neurones involved in the light reflex, spare the near reflex fibres until later.
10. Though the pathways are not as well defined as the pathways for the light reflex, the fibers subserving the near reflex, reach the striate cortex and further relay of information is provided to the frontal eye fields (FEF) and the EWN, bypassing the pretectal olivary nucleus, in the dorsal midbrain.
11. From the EWN, these fibers follow the efferent course through the different components of the oculomotor nerve, the motor part serving the medial rectus muscle and the parasympathetic part serving the sphincter pupillae through the ciliary ganglion and the short posterior ciliary nerves.

Causes of light–near dissociation



1. Bilateral complete afferent pathway defect	Old RD B/L Optic atrophy	A B
2. Lesions in midbrain Pretectal area lesion(without damage to ventrally located near reflex input)	Pinealoma manifesting as Parinaud syndrome/sylvian aqueduct syndrome Vascular lesion Encephalitis Demyelination Neurosyphilis ARG (damage to intercalated neuron in pretectum)	C D
3. IINP with aberrant regeneration to sphincter pupillae	Pseudo ARG	E
4. Ciliary ganglion/ short ciliary nerve with aberrant regeneration of accommodation impulse into sphincter pupillae	Tonic pupil	F
5. Peripheral neuropathies leading to aberrant regeneration	Diabetes ,Alcoholism, Amyloidosis	
Unilateral	Bilateral	
1. Afferent conduction defect	1. Neurosyphilis	
2. Adie pupil Herpes zoster ophthalmicus	2. Type 1 diabetes mellitus	
3. Aberrant regeneration of the third cranial nerve	3. Myotonic dystrophy	
4. (pseudo-ARP)	4. Parinaud (dorsal midbrain) syndrome	
	5. Familial amyloidosis	
	6. Encephalitis	
	7. Chronic alcoholism	
	8. Charcot-Marie-Tooth disease	
	9. Dejerine-Sottas disease	



- What differentiates true Argyll Robertson pupil (light near dissociation) from pseudo-Argyll Robertson pupil is the fact that true ARP occurs due to lesions in the pupillary pathway.
- The pathogenesis of pseudo-ARP in case of third nerve palsy is due to aberrant regeneration of the nerve, such that the pupillary fibers find their way into the nerve sheath, innervating the medial rectus muscle. Due to this, any action of the medial rectus muscle, including convergence, results in miosis, thus simulating a strong near reflex in the absence of normal constriction to light. A true 'light near dissociation' is present only if the near response, tested in mesopic background illumination exceeds the best constriction that bright light can produce.

Cause	Location	Mechanism
Severe loss of afferent light input to both eyes	Anterior visual pathway (retina, optic nerves, chiasm)	Damage to the retina or optic nerve pathways
Panretinal photocoagulation, retinal cryotherapy, orbital surgery	Short, posterior ciliary nerves	Aberrant reinnervation following damage to short, posterior ciliary nerves
Peripheral neuropathy	Short, posterior ciliary nerves	Axonal loss
Adie syndrome	Ciliary ganglion	Aberrant reinnervation of iris sphincter by accommodative neurons
Third nerve aberrant reinnervation	Course of CN III	Aberrant reinnervation of iris sphincter by accommodative neurons or extraocular muscle neurons
Loss of pretectal light input to Edinger-Westphal nucleus	Tectum of the midbrain	Infection (Argyll Robertson pupils) or compression

Afferent Visual Pathway

- Optic neuropathy is the most common cause of light–near dissociation (unilateral or bilateral).
- In optic neuropathy, light–near dissociation is the result of damage to only the afferent limb of the pupillary light reflex (optic nerve); the central near impulses remain unaffected.

Midbrain

- Dorsal midbrain damage can result in midsize pupils with poor light response and preserved near response.
- Such responses occur when the lesion spares the more ventrally located fibers of the near reflex pathway.
- Associated findings include bilateral eyelid retraction (Collier sign), vertical gaze palsy, accommodative paresis, and convergence-retraction nystagmus (Parinaud syndrome)
- The Argyll Robertson pupil occurs in patients with tertiary syphilis involving the central nervous system.
- Affected patients have small pupils (<2 mm) that are often irregular.
- The pupils do not react to light, but the near response and subsequent redilation is normal and brisk.
- This feature distinguishes Argyll Robertson pupils from bilateral chronic tonic pupils, which may also result from neurosyphilis.
- In addition, iris atrophy frequently occurs, portions of the iris transilluminate, and dilation is poor after instillation of mydriatic eye drops.
- Argyll Robertson–like pupils are observed in widespread autonomic neuropathies such as bilateral tonic pupils (chronic), diabetes mellitus, and chronic alcoholism, as well as in encephalitis and after panretinal photocoagulation.
- Serologic tests for syphilis, such as the serum fluorescent treponemal antibody-absorption (FTA-ABS) test and the Treponema pallidum hemagglutination assay (TPHA), should be considered in the evaluation of patients with bilateral pupillary light–near dissociation with miosis.

Aberrant Regeneration

- Light–near dissociation can also result from aberrant regeneration of damaged nerves that restores the near reflex but not the light reflex.



- In tonic pupil syndrome, the injured short ciliary nerves resprout and accommodative fibers mistakenly reinnervate the iris sphincter.
- Similar misdirected growth can occur after traumatic injury or chronic compression of the oculomotor nerve.
- Sometimes aberrant regeneration involves the medial rectus fibers and pupillary contraction occurs during attempted adduction. This synkinetic pupil movement can resemble light–near dissociation.

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