2.4.2.3 Pupillary Response

Pupillary response or pupil light reflex is a physiological response that leads to a change in the pupil diameter (size). While the pupil diameter (PD) is about 3.5 mm, the pupil constricts under a bright light to 1.5 or 2 mm in diameter and expands under dim light up to 8 mm.

Pupil constriction and dilation can be brought about by the contraction of the sphincter and the dilator pupillae, two antagonistic autonomic (reflex) muscles. The sphincter muscle (σφιγκτήρας), innervated by the parasympathetic nervous system, is a circumferential muscle that forms a ring around the iris edge; its contraction leads to pupil constriction. The dilator muscle (διαστολέας), innervated by the sympathetic nerve system, forms radially from the iris edge into the ciliary body; its contraction leads to excitation of the radial fibers of the iris, which leads to an increased pupillary aperture.

![Figure 2-42: Pupil muscles driving pupil response.](image)

Figure 2-42: Pupil muscles driving pupil response.

Pupil size may be pharmacologically induced: It may constrict (miosis) in response to agents such as opiates/opioids or anti-hypertension medication. The pupil may dilate (mydriasis) by anticholinergic agents and amphetamines that block the responses of the ciliary muscle during accommodation (cycloplegia, § 7.1) and also act on the sphincter muscle, producing mydriasis. A pharmacologically mydriated pupil remains dilated even in bright light. Anticholinergic and alpha-1 adrenergic agonists are used as mydriatic agents.

![Figure 2-43: Scheimpflug images taken before (left) and after (right) pharmacologic mydriasis.](image)

Figure 2-43: Scheimpflug images taken before (left) and after (right) pharmacologic mydriasis.\(^{158}\)

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The clinical presentations of photokeratitis include ocular pain, tearing, conjunctival chemosis, blepharospasm, and deterioration of vision typically several hours after exposure. The presentation can be transient (recessing as the epithelium regenerates) but can also be long term. Chronic UV-B exposure is associated with abnormal cornea conjunctiva growth such as pterygium. UV exposure may lead to the irrevocable loss of corneal endothelial cells, since these non-regenerating cells are very susceptible to UV radiation.

Figure 2-68: Absorption of UV bands by various components of the human eye.

The crystalline lens of the eye strongly absorbs UV, mainly due to its longer optical path (being much thicker than the epithelium). UV absorption by the lens is associated with cataract development: Studies suggest that doubling the lifetime of UV-B exposure increases the risk of cortical and posterior subcapsular cataract by 60%; other studies conclude that individuals with a high, long-term UV-B exposure have over 3× increased chance of developing a cortical cataract.

While the UV radiation is strongly absorbed (1% remaining) before reaching the retina, even this small fraction, if phototoxic, is of concern. Lens removal by cataract surgery leads to an increase in the UV that reaches the retina if the IOL does not effectively block it.

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