

The Visual System: Central Visual Pathways

Visual field representation

Each eye sees a part of the visual space that defines its visual field. The visual fields of both eyes overlap extensively to create a binocular visual field. The total visual field is the sum of the right and left *hemifields* and consists of a binocular zone and two monocular zones.

Just like the visual field is divided into two hemifields, the retina is divided in half, relative to the fovea, into a *nasal* and a *temporal hemiretina*. Each hemifield is projected onto the nasal hemiretina of the ipsilateral¹ eye and the temporal hemiretina of the contralateral² eye.

The axons of ganglion cells exit the eyes via the *optic nerve*, partially cross at the *optic chiasm*, and form two *optic tracts*, so that the right and left hemifields reach the left and right hemispheres. Each optic tract looks at the opposite hemifield, combining inputs from the ipsilateral temporal hemiretina and the contralateral nasal hemiretina.

Central Projections of the retina

The retina projects to *four subcortical regions* in the brain: 1) the *lateral geniculate nucleus*, the major subcortical center relaying visual information to the primary visual cortex; 2) the *superior colliculus*, which control orienting eye movements; 3) the *hypothalamus*, which regulate the circadian rhythms; and 4) the *pretectum*, which control the pupillary light reflex.

Pretectum and pupillary light reflex

Shining a light into someone's eye causes the pupil to narrow reflexively. The pretectum controls the action of the iris' pupillary constrictor muscle via its projection to both *Edinger-Westphal* nuclei, whose neurons send their axons to the ciliary ganglion via the third cranial nerve. The pretectum bilateral projections to the Edinger-Westphal nuclei ensure that both eyes react to light: shining a light into the left eye elicits a *direct* pupillary reflex in the left eye and a *consensual* pupillary reflex in the right eye. This light reflex informs us about someone's visual pathways status.

Lateral geniculate nucleus

The Lateral Geniculate nucleus (LGN) in the thalamus is the major target of the retinal ganglion cells. It receives inputs from both eyes and relays these messages to the primary visual cortex via the optic radiation.

The retinal Magno and Parvo ganglion cells respectively project to 2 ventral *magnocellular* layers and 4 dorsal *parvocellular* layers of the Lateral Geniculate Nucleus. Each of the six LGN layers receives inputs from either the ipsilateral or contralateral eye, i.e., the ganglion

¹ "on the same side"

² "on the opposite side"

cells of the left eye project to *layer 1, 4 & 6* of the right LGN, and the right eye ganglion cells project to its *layer 2, 3 & 5*.

Selective lesions of the parvocellular and magnocellular LGN layers alter specific visual functions. Lesions restricted to the parvocellular layers severely disrupt the processing of *color* and *fine detail*, whereas lesions of the magnocellular layers severely disrupt the detection of *fast moving stimuli*, leaving color and fine detail vision unaffected.

Optic radiation

The LGN projections reach the primary visual cortex through the *optic radiation*. Axons carrying information about the superior visual field sweep around the lateral horn of the ventricle in the temporal lobe (*Meyer's loop*). Those carrying information about the inferior visual field travel under the cortex of the parietal lobe.

Primary visual cortex

The primary visual cortex (V1) has a representation of the contralateral visual hemifield. The foveal region is mapped in its most posterior part, whereas the more peripheral regions are mapped in progressively more anterior parts. The upper visual field is mapped on the lower bank of the calcarine sulcus, the lower visual field on the upper bank.

Because of the high density of ganglion cells in the fovea, the visual cortex has an expanded representation of the fovea.

Visual field deficits

The highway of visual information (retina-LGN-V1) can be vulnerable to strokes and tumors. Because of the orderly organization of this central visual pathway, such lesions produce characteristic gaps in the visual field.

A lesion of the *right optic nerve* causes a total loss of vision in the right eye; it also produces a right afferent pupil deficit.

A lesion of the *optic chiasm* causes a loss of vision in the temporal half of both visual fields: *bitemporal hemianopsia*³. This kind of damage is most commonly caused by a tumor of the pituitary gland that compresses the chiasm. This is the only lesion in the visual system that causes a “*nonhomonymous*” deficit in vision, i.e., a deficit in two different parts of the visual field resulting from a single lesion.

A lesion of the *right optic tract* causes a complete loss of vision in the left hemifield: *contralateral “homonymous” hemianopsia*.

A lesion of the *right optic radiation* just after the LGN also causes a loss of vision in the left hemifield: *contralateral “homonymous” hemianopsia*.

³ “Relatively large visual field deficits are called *anopsias*, while smaller ones are called *scotomas*.”

A lesion of the *right optic radiation* specific to *Meyer's loop* causes a loss of vision in the upper quadrant of the left hemifield. The same is true for lesions to the *lower bank of the calcarine sulcus*.

A lesion of the *parietal* portion of the *right optic radiation* causes a loss of vision in the lower quadrant of the left hemifield. The same is true for lesions to the *upper bank of the calcarine sulcus*.

A lesion of the right visual cortex can cause a complete loss of vision in the left hemifield but they usually *spare foveal vision*, probably because the cortical foveal representation is so extensive that a single lesion is unlikely to destroy it all.





