

Functional Anatomy of the Human Visual Pathway

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Nearly a half of all cortical neurons are devoted to the processing of visual information. The afferent visual pathway from the retina to the primary visual cortex has four neuronal elements (■ Fig. 3.1).

- First neuron: photoreceptors
- Second neuron: bipolar cells
- Third neuron: retinal ganglion cells (and their axonal processes, including the chiasm and optic tracts)
- Fourth neuron: geniculocalcarine neurons

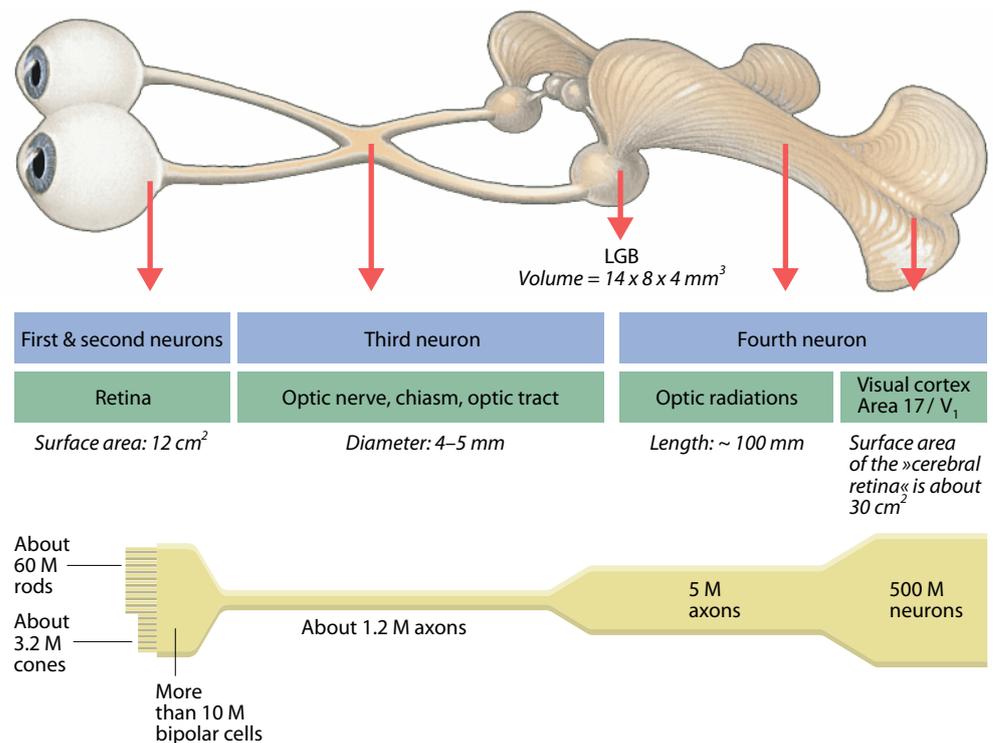


Fig. 3.1. Schematic diagram of the human visual pathways and their neuronal components. *LGB* Lateral geniculate body (modified after Krey et al. 1986; see “Further Reading”)

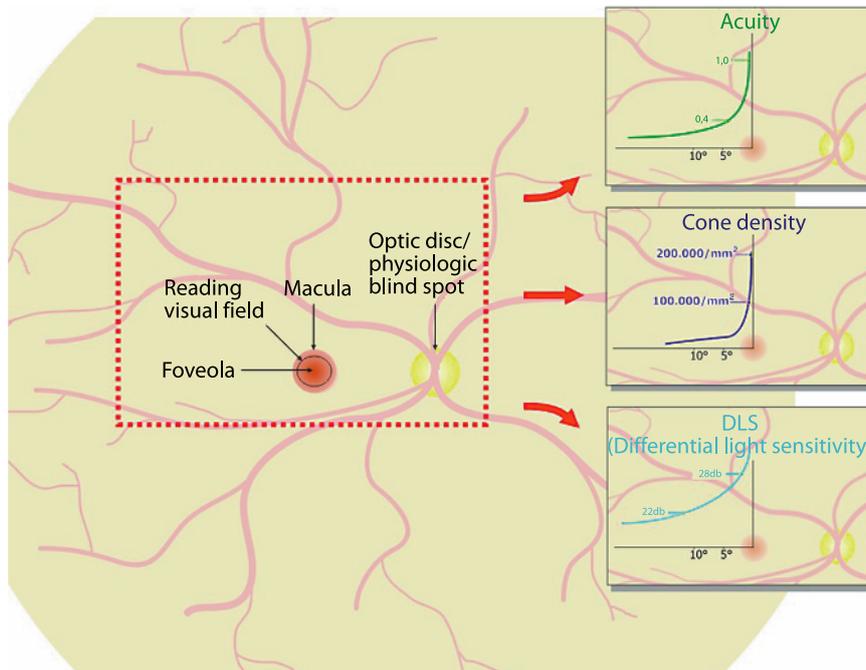


Fig. 3.2. Visual acuity, cone density and differential light sensitivity (*DLS*) as a function of visual field eccentricity (modified from Coren and Ward 2003, and Trauzettel-Klosinski et al. 1994; see “Further Reading”)

First Neuron: Photoreceptors

The retina contains across its outer surface (about 12 cm²) nearly 65 million photoreceptors per eye (see also Chap. 7): about 3.2 million cones and 60 million rods.

The areal density of photoreceptors falls rapidly from the fovea into the retinal periphery (■ Fig. 3.2). Efficient perimetric stimulus presentation considers these factors, using more closely spaced stimuli at the visual field center, with a rapidly decreasing density of stimuli for more peripherally located visual field areas. But even in the most peripheral parts of the retina, there are sufficient numbers of cones to dominate vision under photopic levels of illumination when the rods are completely bleached.

● Pearl

Most of today’s perimeters operate with an adapting background luminance of 3 to 10 cd/m² in the lower photopic range, and consequently, test the function of cone-initiated vision only.

Second Neuron: Bipolar Cells

In the human retina, “only” 10 million bipolar cells (see also Chap. 7) process the signals arriving from the approximately 65 million photoreceptors. The neural convergence found at this level of retinal circuitry is not homogenous: While the peripheral retinal regions operate with a comparatively sparse population of bipolar cells, the central

portions of the retina (foveal and perifoveal macula) process the photoreceptor signals in a 1:1 or cell-for-cell arrangement. In other words, while there is high neural convergence in the retinal periphery, there is a parallel processing of the signals from the densely clustered receptors at the fovea and perifoveal macula.

Third Neuron: Retinal Ganglion Cells

The retinal ganglion cells give rise to axons that are about 75 mm in length (see Chaps. 8 and 12). They join one another at the optic disc to form the optic nerve, being myelinated only in their extraocular course. They pass through the optic chiasm with decussation of more than one half of the fibers to the contralateral side, and pass through the optic tracts to the lateral geniculate body, where they terminate.

Retinal Ganglion Cells and the Optic Nerve

The neuronal signals are concentrated into “merely” 1.2 million ganglion cells (per eye). Their axons form the retinal nerve fiber layer, just deep to the internal limiting membrane. They are characterized by a widely fanned-out shape that skirts the macula, and they then converge at the margin of the optic disc. Their spatial arrangement gives rise to a typical pattern when disease damages associated groups of fibers at the disc margins: The fibers arriving at the temporal sectors of the disc margin arise from cell bodies located either above or below the temporal horizon-

tal raphe, which they “respect,” or do not cross, but form superior and inferior arcuate shapes that converge as they approach the disc.

Pearl

Damage to the retinal ganglion cells in the vicinity of the optic disc produces typical arcuate defects in the visual field that do not cross the nasal horizontal meridian, hence the so-called nasal step. The shape of an arcuate scotoma is similar to that of a scimitar; its hilt located at the physiologic blind spot and its tip at the nasal horizontal meridian. The fibers arriving at the disc from the temporal hemiretina (both superior and inferior arcuate groups) cross the borders of the disc at the superior and inferior poles. Bundles of fibers arriving from the nasal hemiretina form a more wedge-like shape with straight sides and the apex located at the nasal disc border.

The ganglion cell axons join one another to exit the eye through the optic disc, which is about 1.5 mm in diameter. In doing so, the fibers from the retinal locations that are closest to the disc rise to the retinal surface and enter the disc at its most central core. Fibers that originate in the retinal periphery, by contrast, course through the innermost portions of the nerve fiber layer, closest to the vitreous body. They exit the eye through the outermost portions of the optic disc, closest to its border (■ Fig. 3.3). Upon exiting the ocular wall through the lamina cribrosa, the axons acquire a myelin sheath, and the diameter of the nerve increases to 4 mm. The fibers are separated into about 300 to 1,000 bundles by connective tissue septae. The optic nerve has a floppy, sinusoidal course within the orbit, which allows the globe to rotate at high speeds and with minimal inertia. The optic nerve has an intraorbital length of 20 to 30 mm, an intracanalicular length of 3 to 8 mm, and an intracranial length of 3 to 16 mm. At the posterior extreme of the optic nerve, the afferent visual pathway acquires a new name, the chiasm, as the optic nerves merge with one another.

Pearl

The optic nerve is particularly susceptible to damage by space-occupying lesions within the optic canal. Masses of any kind arising in the canal will compress the nerve, and the rigid walls prevent any escape or decompression of the neural tissues.

Within the retina, the axons arising within the temporal hemiretina follow an arcuate path that skirts the macula above and below the horizontal meridian with the axons respecting (not crossing) the horizontal meridian. As they

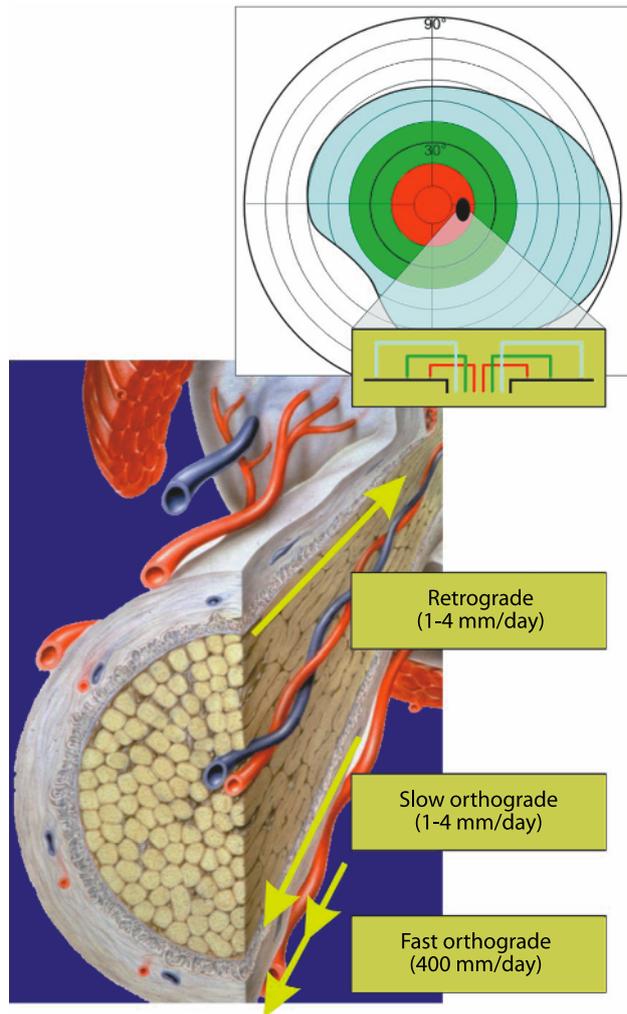


Fig. 3.3. Schematic diagram of the pattern created by the layering of the ganglion cell axonal nerve fibers in the nerve fiber layer of the retina and the corresponding visual field regions (upper insert) as well as the structures within the optic nerve and the axoplasmic flow parameters (modified from Krey et al. 1986; see “Further Reading”)

pass into the optic nerve, the ganglion cell axons acquire a completely different organization, as they separate from one another according to their origins with respect to the vertical meridian. Axons from cells located to the nasal side of the vertical meridian (remember: the vertical meridian through the fovea, not the optic disc) decussate at the chiasm, while axons from cells located in the temporal hemiretina remain ipsilateral. The tight, septate axon bundles so highly organized in the optic nerve undergo reorganization at this point in the afferent pathway. They intermix in what seems to be a relative disorganized fashion, giving rise to a loss of the topographic organization of the visual field found with diseases of the retina and optic disc. Consequently, the perimetric spatial localization of disease is very poor for lesions of the intracanalicular optic nerve.

Nonetheless, perimetry remains a very important diagnostic tool for following the course of disease in this region.

The very thin fibers of the papillomacular bundle are clustered together in the core of the optic nerve, and they represent the largest number of fibers in the so-called parvocellular system. These axons project into the layers of the lateral geniculate body that contain neuronal somas, which are also quite small (see ■ Fig. 3.6). Parvocellular neurons transmit the encoded image information from the central-most portions of the visual field, which is characterized by high spatial resolution, good color perception, and stereopsis. This subsystem of the afferent pathway is particularly susceptible to demyelinating or toxic damage, resulting in defects of the central visual field – central scotomas.

Pearl

This central group of fibers from the papillomacular bundle occupies approximately 70% of the cross-sectional area of the afferent pathway in the optic nerve, chiasm, and optic tract. These fibers are also more sensitive to damage caused by space-occupying disorders, and their axonal transport mechanisms suffer,

whether by direct mechanical compression of the fibers or impairment of their capillary blood supply (cf., ■ Fig. 3.3).

For this reason, it is generally the case that examination of the central 30° of the visual field is adequate for the detection of visual loss caused by the overwhelming majority of neuro-ophthalmic disorders that damage the anterior portions of the afferent pathway.

Optic Chiasm

The fibers that arise in the nasal hemiretinas of both eyes (nasal to the fovea-bisecting vertical meridian) decussate in the chiasm to the contralateral side, while the fibers arising in the temporal hemiretinas remain ipsilateral as they enter the optic tracts (■ Fig. 3.4). During embryonic development, this process is thought to be controlled chemotactically, so that there are no sharply defined or straight lines at the interfaces between adjacent groups of fibers. The neural bundles originating in the nasal hemiretinas – and which decussate to the contralateral side within the chiasm – form short loops that protrude into the proximal contralateral optic nerve (by the inferior half of the decussating

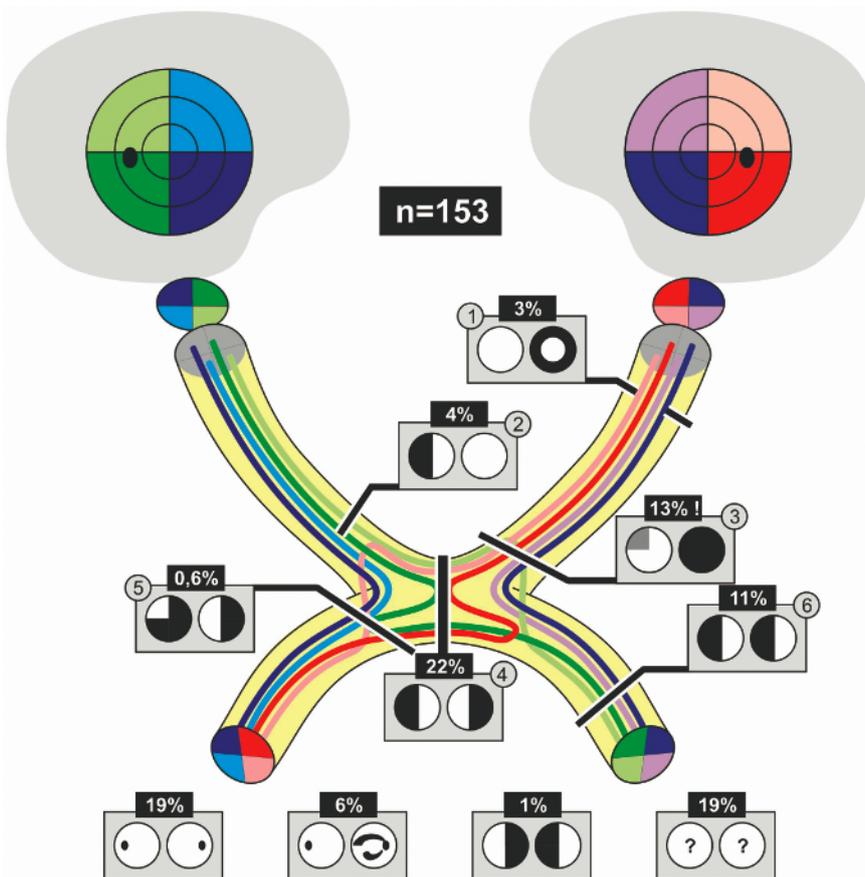


Fig. 3.4. Schematic diagram of the course of the ganglion cell axons in the region of the chiasm and the corresponding visual field defects with their frequency of occurrence. The effect of the more common lesions within the various visual pathway segments in the chiasmal region on the central 30° of the visual field are diagrammed as examples. 1 Compressive optic neuropathies. 2 Partial prechiasmal lesion. 3 Anterior junctional syndrome with subtotal damage to anterior Wilbrand's knee, which carries the afferent signals from the contralateral inferior nasal retinal fibers, which correspond to superior temporal visual field quadrant. This pattern is often found with advanced levels of damage or even complete loss of function in the ipsilateral optic nerve. 4 Disease of the central chiasmal region. 5 Posterior junctional syndrome with damage to the posterior knee of Wilbrand, which carries signals from the ipsilateral superior nasal retinal quadrant and represents the inferior temporal quadrant of the visual field. This ipsilateral defect is associated with a homonymous hemianopia to the contralateral side. 6 Lesion of the optic tract (from Schiefer et al. 2004; see "Further Reading")

Animation 3.2

axons) and into the ipsilateral optic tract (by the superior half of decussating fibers) before completing the transition to the contralateral optic tract, where they are joined by the fibers originating in the corresponding contralateral temporal hemiretina (for further details, please see the “Anterior Junction Syndrome” section below). The chiasm measures 8 mm from the anterior to the posterior commissure, and is about 12- to 18-mm wide and 4-mm thick.

Pearl

The chiasm is the locus of the greatest risk for catastrophic loss of vision. The sum total of visual information within the afferent pathway is concentrated into the small volume of about 1 cm³, where it is maximally susceptible to damage by focal disease processes. Consequently, comparatively small foci of disease in this region can produce rapid and complete, bilateral blindness. The space-occupying diseases of the sellar region are numerous (■ Fig. 3.5). Most common is the pituitary adenoma, which arises from just beneath the chiasm, and which is the most common (and mostly benign) intracranial tumor (see Chap. 12). A point to keep in mind is that a tumor arising from the pituitary gland must rise a full 10 mm above the diaphragma sellae before contacting the chiasm. After the advent of MRI scanning, it has become quite common for small adenomas to be detected long before they do visual damage, and their frequency is far greater than had been suspected.

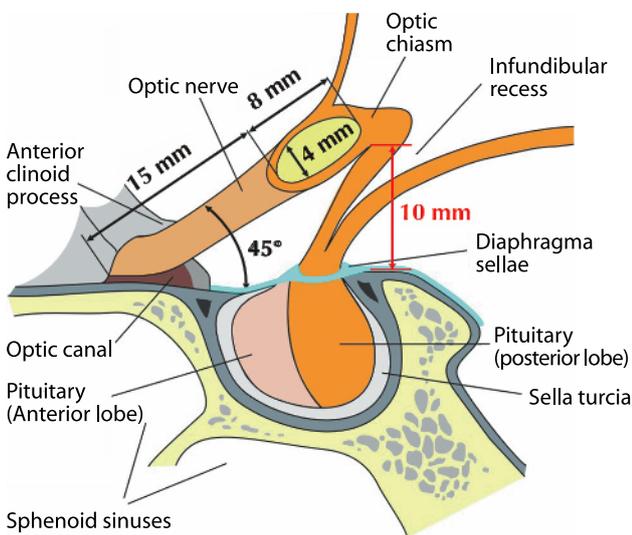


Fig. 3.5. Structures of neuro-ophthalmic relevance and their relative positions in the perichiasm region (modified from von Lanz et al. 2003; see “Further Reading”)

The relative positions of chiasm and hypophysis means, on the one hand, that microadenomas (by definition as intrasellar lesions) never cause perimetrically detectable visual field defects. On the other hand, when an adenoma has grown so large that it is detectable by the visual field defect it causes, it is already at an advanced stage of the disease (■ Fig. 3.5). Since the risk of pituitary apoplexy (intralesional hemorrhage with rapid expansion of the mass) increases as the tumor enlarges, visual field loss attributable to a pituitary adenoma always requires that an imaging procedure (preferably an MRI) be done quickly (see Chaps. 12 and 20).

There are other mass lesions in the sellar region that must be included in the differential diagnosis: meningiomas (tuberculum sellae, sphenoid, clivus), and craniopharyngiomas (see Chap. 12). Anteriorly located are the paranasal sinuses that are at risk for bacterial infections and trauma, especially the neighboring ethmoid sinuses.

The carotid arteries and cavernous sinuses lie on each side of the chiasm, along with the oculomotor cranial nerves, the first and second divisions of the trigeminal nerve, and fibers of the sympathetic supply to the eye and orbit. Pituitary tumors can spread laterally to cause primarily motor pareses of the eye without any sign of damage to the afferent visual pathway (see Chaps. 10 and 12).

Superior to the chiasm is the floor of the third ventricle. Invading mass lesions are limited in their movement by the relatively immobile tissues above; in addition certain blood vessels in the chiasmal region (e.g., the anterior cerebral and the anterior communicating arteries) can produce deep, pressure-induced furrows in the chiasm along with corresponding deficits in visual function.

Pearl

Chiasmal lesions produce characteristic visual field deficits that show respect for the vertical meridian (in at least one eye) and can be monocular or binocular with widely varying asymmetries (■ Fig. 3.4). This typical asymmetry also explains why patients with chiasmal disease are commonly found to have relative afferent pupillary defects (see Chaps. 2, 5, and 12).

Anterior Junction Syndrome

Definition

Anterior junction syndrome (syndrome of the antero-lateral chiasm) presents as a combination of severe central visual loss in one eye and asymptomatic or subtle defects in the superior temporal visual field of the contralateral eye.

This unique and unmistakable pattern is attributable to an anatomic feature in the chiasm called Wilbrand's knee, which is a temporary detour taken by the inferior nasal crossing fibers into the proximal end of the contralateral optic nerve (■ Fig. 3.4, no. 3). The existence of this redundant loop has been called into question by several authors, believing that the loop seen histologically is an artifact of tissue preparation. If the knee is not a true morphological feature of the chiasm, one can plausibly conclude that axons are being displaced by local forces attributable to axonal loss or by the mass lesions compression of the chiasm and/or proximal optic nerve. This would at least make the common clinical findings of anterior junction syndrome comprehensible.

An asymmetric disorder affecting the anterior chiasm can produce complete amaurosis of the ipsilateral eye. It is important when finding that vision has recently been lost in one eye to examine carefully its contralateral partner. If one finds a partial loss of the superior portions of the temporal half of the contralateral field, this should be regarded as manifest evidence of a chiasmal syndrome, and if it has not yet been done, imaging of the chiasm is mandatory (■ Fig. 3.4).

Pearl

Every unexplained (even unilateral) loss of vision requires careful perimetric testing of the visual fields of *both* eyes.

Optic Tract

The decussation of the axonal bundles that originated in the nasal half of the retina causes them to associate with fibers arising from the temporal hemiretina of the contralateral eye, and identical visual field loci for both eyes are processed in the cerebral hemisphere that is contralateral to its homonymous hemifield. All postchiasmal lesions of the visual pathway (with one small exception) are characterized by homonymous visual field defects.

The optic tract, about 20 to 30 mm in length, is comparatively well vascularized. It is not commonly affected by traumatic or space-occupying processes. It is very unusual for visual field defects to be caused by disease in this location: About 4% of all homonymous visual field defects are due to tract disease.

Because of relative disorder among axon bundles in the immediately postchiasmal tract, disease in this locale produces rather incongruent homonymous (contralateral) hemianopic defects. A few weeks after their onset, these defects are accompanied by a characteristic, manifest, bilat-

eral, asymmetric optic atrophy (see Chap. 8, ■ Fig. 8.23 and Chap. 19, ■ Fig. 19.6).

Tract lesions are usually accompanied by a relative afferent pupillary defect (see Chaps. 4, 5, and 12). The relative afferent pupillary defect (RAPD) is found in the eye contralateral to the lesion. This probably reflects the greater size of the temporal hemifield (processed by the nasal hemiretina of the contralateral eye), as compared with the nasal hemifield.

The proximity of the optic tract to the structures of the internal capsule explains the frequent association of somatic hemisensory loss ipsilateral to the homonymous hemifield defect.

Pearl

Tract lesions are characterized by:

- A contralateral relative afferent pupillary defect
- A contralateral, incongruous, bilateral, homonymous visual field defect
- A bilateral, asymmetric optic atrophy that appears with a latency of several weeks or months after the visual loss

Fourth Neuron

Lateral Geniculate Body: Synaptic Relay

The lateral geniculate body (LGB), or lateral geniculate nucleus (LGN), is the last synaptic component of the afferent pathway prior to arrival at the primary visual cortex. About 1.2 million tract fibers on each side project onto about 5 million neurons of the optic radiations, which constitute the last segment of the afferent pathway to the cerebral cortex. The lateral geniculate body has a unique onion peel structure, which receives the input signals from both eyes and processes the data in its various layers with a strongly retinotopic organization (■ Figs. 3.1, 3.6, and 3.7a).

The projecting axons of the retinal ganglion cells that carry the data for high spatial resolution, color perception, and stereopsis terminate in layers of the lateral geniculate body, having comparatively small cell somas (see the parvocellular system, ■ Fig. 3.6). The encoded data from receptors that respond primarily to moving stimuli are carried by fibers that have poor spatial resolution and are processed in layers of the lateral geniculate body having cell bodies that are large (see the magnocellular system, ■ Fig. 3.6).

Lesions of the lateral geniculate body are relative rare and are principally caused by vascular disease. The structural organization of the geniculate underlies the features

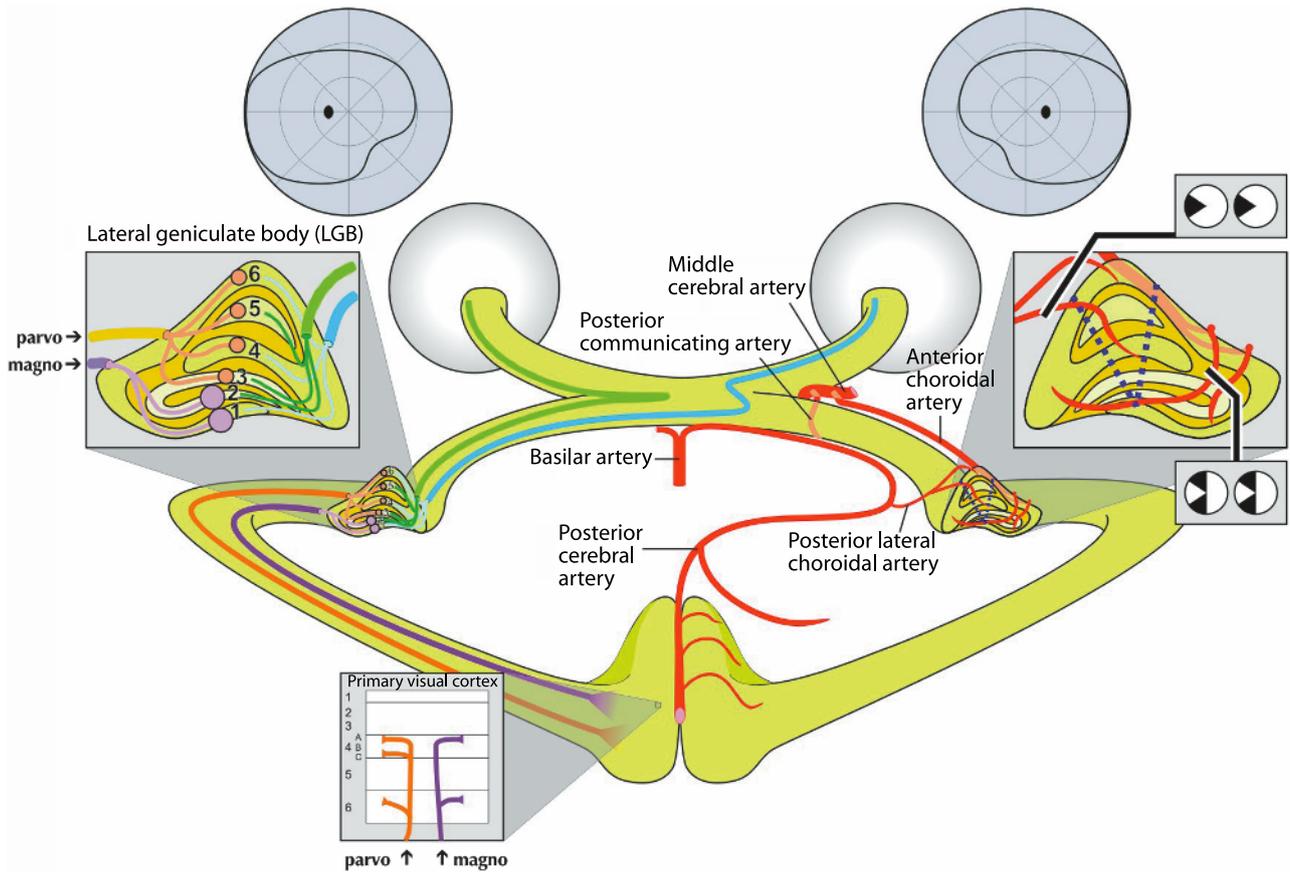


Fig. 3.6. Arrival of the third-order neuron axonal fibers innervating the layers of the lateral geniculate body (LGB) and their segregation into the magno- and parvocellular systems. *Left inset* Post-synaptic projection of the fourth-order neurons to layers (1–6/A–C)

of the primary visual cortex. *Right inset* Blood supply of the lateral geniculate body and the corresponding visual field defects caused by subtotal infarctions of the geniculate nucleus

of its related visual field defects, which affect large portions of the central visual field with wedge-shaped, homonymous defects with relatively poor congruence (■ Fig. 3.6). Usually, these defects are associated with a contralateral RAPD when the lesions are located in the geniculate body or in a surrounding region of up to 16 mm away. When lesions of the postgeniculate fibers are located more than 16 mm away from the lateral geniculate nucleus (LGN), an RAPD is not usually present.

Optic Radiations

Shortly after leaving the geniculate body, the axons of the optic radiations fan out broadly to wrap around the temporal horn of the lateral ventricle. The fibers of corresponding retinal (and therefore of corresponding visual field) loci are located together in spatial proximity with one another and

with a high degree of precision. This means that even small structural lesions will produce circumscribed, sharply marginated, absolute, congruent homonymous contralateral visual field defects. These characteristics are demonstrable with clinical perimetry, but only with instruments that control stimulus presentation with a high degree of spatial resolution.

Projection of this last neuron to the visual cortex appears to obey the all-or-none principle, since visual field defects of postgeniculate disease are almost always absolute.

Postgeniculate disease, i.e., damage to the optic radiations or visual cortex, is characterized by contralateral homonymous hemianopic visual field loss, which in contrast to lesions of the optic tract, are not accompanied by optic atrophy. This is because (at least among adults) there is no trans-synaptic degeneration at the lateral geniculate body.

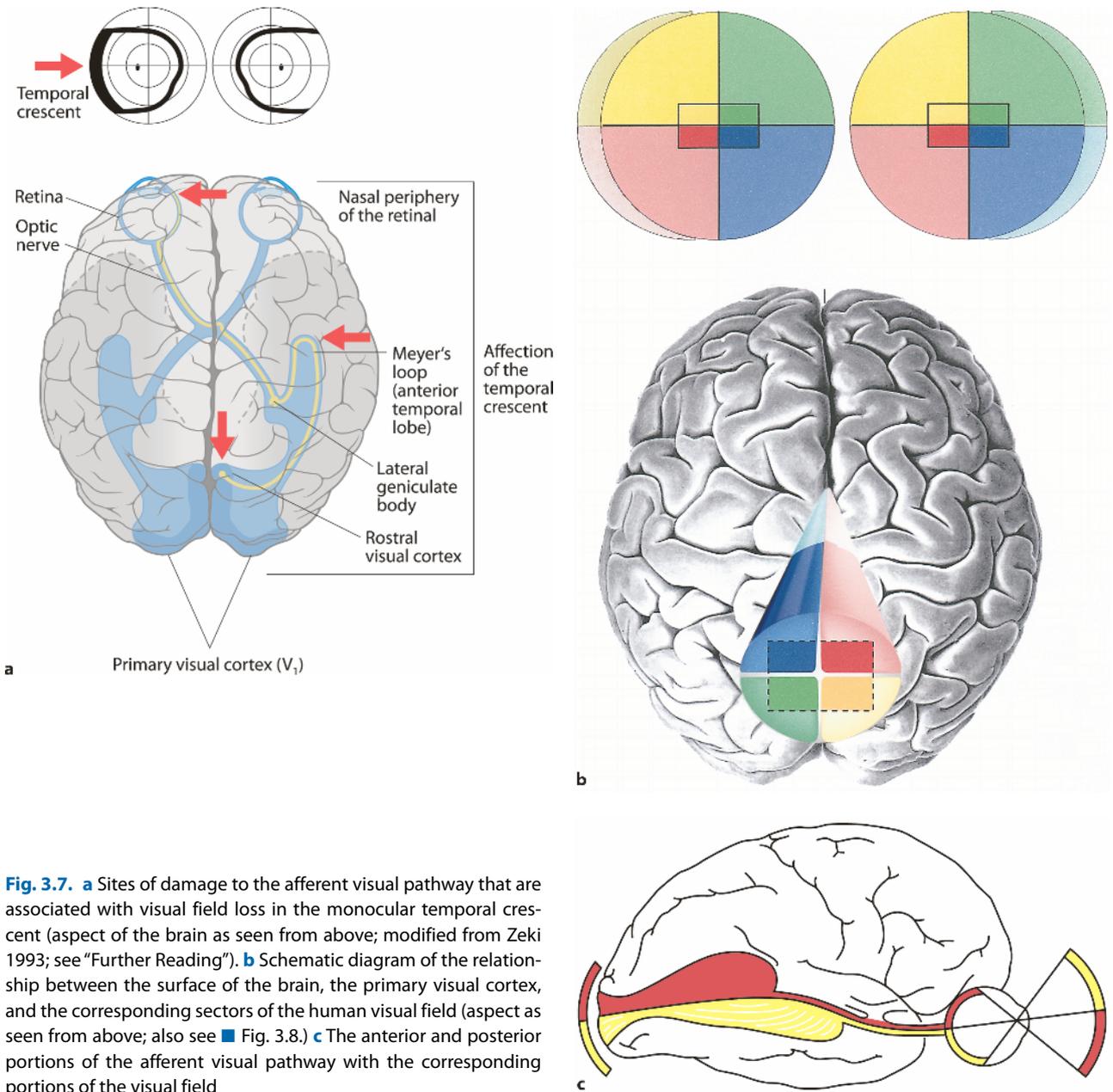


Fig. 3.7. **a** Sites of damage to the afferent visual pathway that are associated with visual field loss in the monocular temporal crescent (aspect of the brain as seen from above; modified from Zeki 1993; see “Further Reading”). **b** Schematic diagram of the relationship between the surface of the brain, the primary visual cortex, and the corresponding sectors of the human visual field (aspect as seen from above; also see ■ Fig. 3.8.) **c** The anterior and posterior portions of the afferent visual pathway with the corresponding portions of the visual field

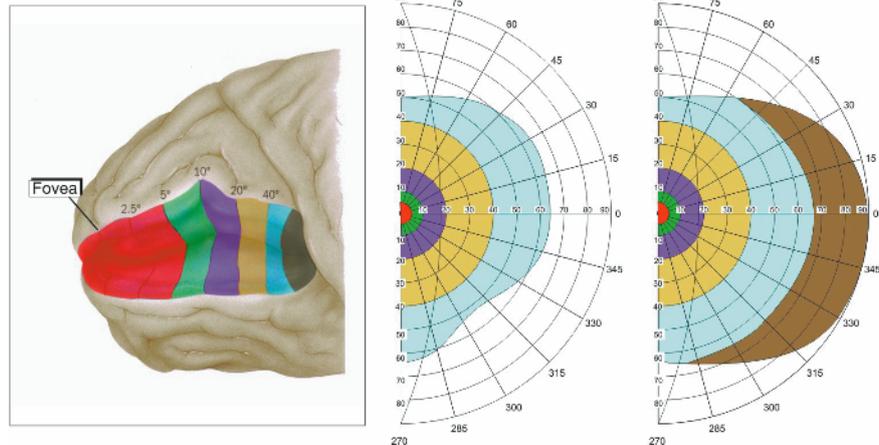
Pearl

The comparatively uncommon homonymous scotomas in the inferior quadrants are associated with damage to the superior (parietal) portions of the afferent pathway. Conversely, homonymous defects in the superior quadrants are associated with disease in the inferior (temporal) fibers of the retrogeniculate pathway (■ Fig. 3.7 c).

Postgeniculate fibers of the afferent visual pathway that carry signals originating in the far periphery of the nasal retinal quadrants, i.e., the part of the visual field that is always monocular (the temporal crescent), participate in forming the so-called Meyer’s loop. These fibers are found in the most rostral part of the loop, located near the apex of the inferior horn of the lateral ventricle (■ Fig. 3.7 a). These structures can be lost when the anterior portion of the temporal lobe is resected, e.g., when removing drug-resistant epileptogenic tissue, resulting in a homonymous defect in the superior quadrant of the contralateral visual field or a defect in the contralateral temporal crescent.

Animation 3.3

Fig. 3.8. The location and extent of (homonymous) visual field defects and their corresponding lesions within the visual cortex (modified from Walsh 1997; see “Further Reading”)



! Note

Disease in the region of the anterior optic radiations (in Meyer’s loop) indicates the need for careful examination of the peripheral visual field, by looking for a defect in the contralateral (monocular) temporal crescent (■ Fig. 3.7a).

Visual Cortex

The primary visual cortex (synonyms include calcarine cortex, area V_1 , or area 17) with its complement of about 500 million neuronal cell bodies is the primary destination of the afferent visual pathways.

Having an area of about 30 cm^2 of cortical surface, the cerebral map of the visual field is considerably larger than that of the retina (about 12 cm^2). The visual cortex is the largest contiguous sector of the brain’s surface that is devoted to a single sensory function.

Crudely simplified, the visual cortex forms the shape of a four-part cone with its basis at the occipital pole of the brain (■ Figs. 3.7b and 3.8). Lines of separation between the four parts include the interhemispheric fissure, separating right and left occipital lobes (and the right and left visual hemifields), and the calcarine fissures, which extend into the mesial aspects of the two occipital lobes, nearly orthogonal to the interhemispheric fissure (and separating the superior and inferior quadrants of each hemifield).

The primary visual cortex contains a retinotopic map of the visual field. About 50% of the cortical visual area is devoted to the central 5° of the visual field, corresponding to no more than 3% of the total visual field area. This high degree of cortical magnification in humans is illustrated by comparison of the red sectors in ■ Fig. 3.8.

The far periphery of the visual field, in the form of the two monocular temporal crescents, i.e., the entirety of

the monocular portions of the visual field, is represented in the deeply situated, most anterior portions of the primary visual cortex, just posterior to the splenium of the corpus callosum and on the mesial surfaces of the occipital lobes. Each side, left and right, represents the monocular crescent of the contralateral eye (■ Figs. 3.7 and 3.8; also see Chap. 4).

! Note

When evaluating lesions in the region of the rostral visual cortex, perimetric study of the temporal monocular crescent is indicated (■ Figs. 3.7 and 3.8).

The occipital pole contains a region of collateral blood supply (a “watershed zone”) along the boundary between the areas perfused by the posterior and the middle cerebral arteries. Homonymous hemianopias caused by cortical lesions are attributable to vascular disease in the vast majority of cases. For all lesions of the visual cortex, vascular disease accounts for 75% of all cases. Tumors are the next most common disease category in this region, amounting to 15% of cases.

Bilateral hemispheric lesions are not uncommon and produce homonymous defects in both sides of the visual field. If lesions in such cases are located above or below the calcarine fissure, the result can be bilateral altitudinal defects in the visual field that also appear to respect the horizontal meridian. Therefore, for example, a complete bilateral inferior hemianopsia (or bilateral, homonymous inferior quadrantanopsia) can be the result of bilateral hemispheric damage to the superior half of the postgeniculate visual pathway (■ Fig. 3.7b; see also Chap. 4). If on one side, the lesion occupies the superior half of the occipital cortex, above the calcarine fissure, while the contralateral lesion lies below the fissure, the result is called a checkerboard visual field (see Chap. 4).

Homonymous hemianopias can be divided into those that split fixation (total loss of one macular hemifield) and those that “spare” the macular region. It is thought that the tendency to spare the macular region (particularly for infarctions of the tissue supplied by the posterior cerebral artery) is related to the watershed zone phenomenon. When the macular cortical representation located at the posterior tip of the occipital lobe lies within a region of collateral supply from the middle cerebral artery, a significant portion of the macular hemifield can be spared. By convention, the term of macular sparing means that an area of at least 3° of radius from fixation has been spared. The pathogenesis and the topographical significance of macular sparing as a perimetric phenomenon have not been uniformly agreed upon. However, the functional significance is very meaningful: With macular splitting there is a profound impairment of reading fluency, while macular sparing usually preserves good reading fluency. Normal reading ability requires at least intact visual function in a small, horizontally oval area surrounding fixation: 2° to either side (right and left) and at least 1° above and below (see Chap. 24 for an extensive explanation).

Pearl

Postgeniculate lesions of the afferent visual pathway:

- Are not associated with optic atrophy when they are not congenital or acquired during infancy
- Are characterized by homonymous, mostly absolute, contralateral, binocular visual field loss
- Have an increasingly more pronounced congruence with locations approaching the primary visual cortex
- Are associated with reading disturbances when there has been loss of the central 2° of the affected visual hemifield
- Are caused by vascular disease in a majority of cases (particularly in the occipital region)
- Are associated (in as many as 30% of cases) with a relative afferent pupillary defect contralateral to the damaged hemisphere

Higher Visual Centers

Further processing of the neurally encoded visual data takes place in circumscribed and specialized cortical regions for specific modalities of vision (e.g., motion, color, etc.). Further, there are centers in the dorsal cortical regions close to the corpus callosum that have strong interhemispheric connections to corresponding structures in the contralateral hemisphere/visual hemifield. To be mentioned in this context also are connections to cortical re-

gions with associative and mnemonic functions, to those with motor and sensory language function, and to efferent visual brain centers (e.g., oculomotor accommodation).

A more complete discussion of the processing of visual sensation in higher centers can be found in Chap. 13).

Conclusion

Knowledge of the course, the neuronal organization, and the association with neighboring structures of the afferent visual pathways at their various neuronal levels (first through fourth neurons) is a fundamental prerequisite for understanding the topographic and pathogenic features of neuro-ophthalmically relevant visual disturbances.

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