

## 2 Functional Visual Anatomy

A basic knowledge of how the visual field is represented at different levels of the neuraxis is fundamental to the performance of perimetry. Without this knowledge, it is impossible to intelligently select a perimetric program, decide among perimetric techniques, or appropriately guide the exploration of the field during manual perimetry.

A couple of general rules deserve statement up front. The first is that the optics of the eye, like those of any camera, create an inverted retinal image, such that the superior visual field is projected onto the inferior retina and the nasal field onto the temporal retina. The second is that the topographic arrangement of the retina tends to be preserved throughout much of the visual pathway, with the superior retina placed in the superior or dorsal aspect, and the left portions of the retina on the left side of various structures. The exceptions are the distal optic tract and lateral geniculate nucleus (LGN), where the map tilts 90°, and in its horizontal arrangement at the striate cortex, where the periphery-to-center topography turns to assume an anterior–posterior dimension, with the central field posterior and the peripheral field anterior.

### 1. RETINA

There are two classes of photoreceptors: rods and cones. The rhodopsin protein of the rods is highly sensitive to light, with each rod able to respond to a single photon, and a mere five to eight photon detections needed to reach the threshold for perception of light in darkness (scotopic conditions) (1). However, in bright light (photopic conditions) the rods lose visual sensitivity, and perception in this setting depends on cones. Normal subjects have three cone types that differ in their opsin pigments. These differences cause different peaks of sensitivity along the spectrum of light, with designations of short (S, sometimes colloquially referred to as “blue”), medium (M, or green), and long (L, or red) wavelength cones. The retinal ganglion cells compare the activity of the different cones to determine what wavelengths of light are impinging on the retina. Color perception also requires participation of extrastriate cortex, in part to adjust for the type of lighting in a scene, to achieve constancy of colors despite variations in such lighting (2,3).

Cones are more numerous than rods in the fovea, while rods are more numerous than cones outside the central 5°. Around

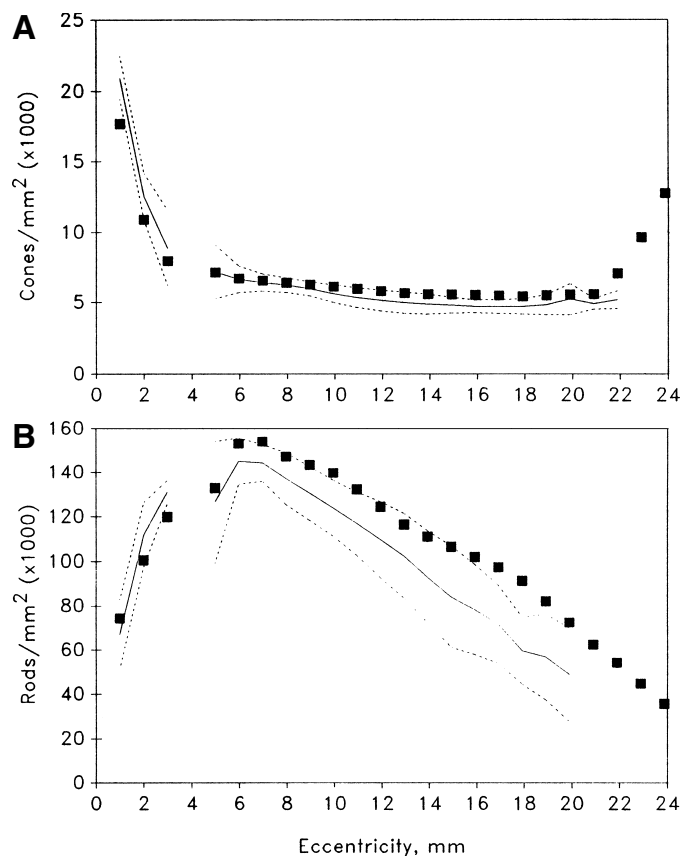


Fig. 1. Distribution of (A) cones and (B) rods. Photoreceptor density is plotted on the y-axis against retinal eccentricity on the x-axis. (From ref. 49 with permission.)

50% of cones are concentrated in the central 30°, with a steep decline in density from center to 3°, followed by a shallower, fairly linear rate of decrease with increasing eccentricity (Fig. 1). This decline in density with eccentricity is true of almost all retinal elements with the exception of rods, which are not found at the fovea, but rather are maximally dense at an eccentricity of about 6–8°, followed again by a gradual decline with increasing eccentricity (Fig. 1).

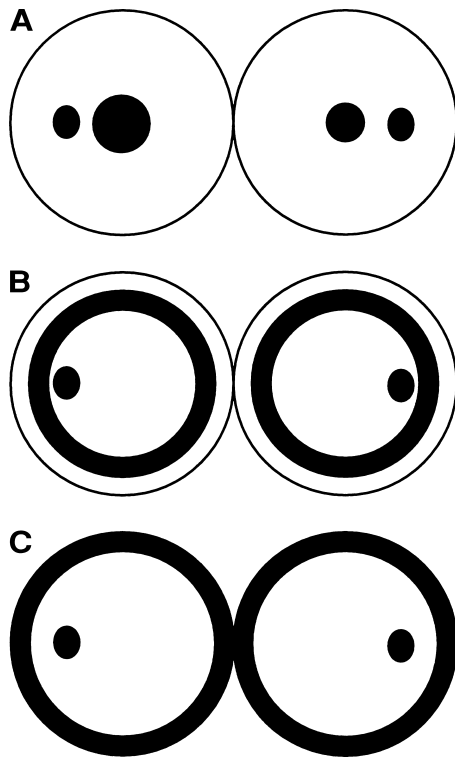


Fig 2. Illustrations of retinal patterns of visual loss: (A) macular or cone disease, causing central scotomata; (B) rod disease, such as retinitis pigmentosa, causing ring scotomata; (C) generalized constriction. The temporal ovals are the physiologic blind spots, and the right eye is on the right, with fields plotted from the patient's view, with right hemifield on the right.

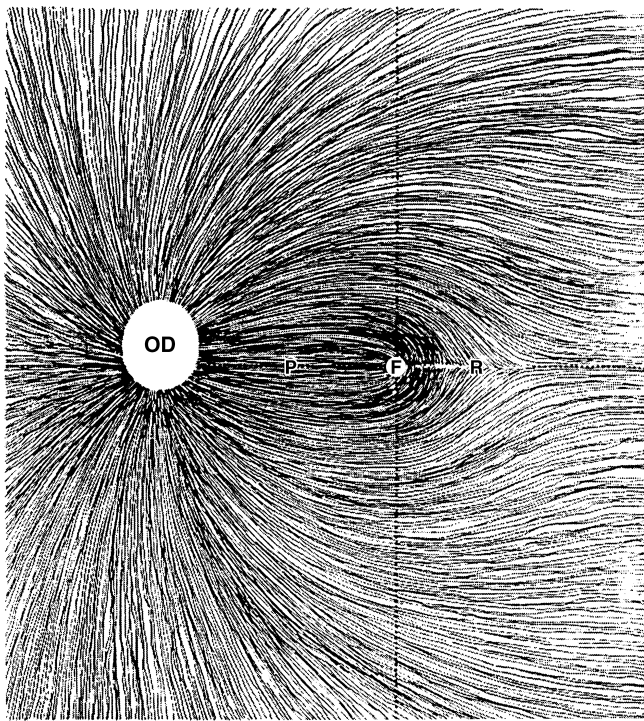


Fig. 3. Retinal nerve axons in the retina. The optic disc (OD) is the white disc, which is left of the fovea (F), in this view of a left eye. The course of the nerve fibers toward the optic disc is shown. The temporal retina lies to the right of the vertical dotted line. P = papillomacular bundle, R = raphe. (From ref. 49 with permission.)

The consequence of the distribution pattern of cones and rods is that retinal disorders that preferentially affect cones (e.g., cone dystrophies) will tend to affect central vision first (Fig. 2). Disorders that are specific to rods (e.g., retinitis pigmentosa) will tend to affect the midperiphery of vision more, sparing both central vision and color vision. Otherwise, there are no specific anatomic issues regarding the distribution of retinal elements that are reflected in the topography of the field defects of retinal disease. Some conditions present simply with generalized constriction. Examples include the toxic effects of vigabatrin and mild background diabetic retinopathy (4). Others present with defects that correspond to the location of retinal damage and, hence, are related more to issues of preferential pathology than anatomy. Examples include macular degeneration, retinal detachments, and congenital defects such as staphylomata. Most retinal lesions that produce focal defects in the visual field are visible on fundoscopy, and perimetry does not add much to the diagnostic process.

### 1.1. VASCULAR SUPPLY

The main supply of the outer retina, where the photoreceptor layer lies, is the posterior ciliary arteries, which, like the central retinal artery, are terminal branches of the ophthalmic artery, the first major branch of the internal carotid artery within the cavernous sinus.

## 2. RETINAL NERVE FIBER LAYER

The axons of the retinal ganglion cells project to the optic disc, where they form the optic nerve. Because the optic disc is situated in the nasal retina, rather than in the center of the field, there are asymmetries in the paths followed by the axons to reach it. The organization of these axons is key not only to understanding disturbances of the inner retinal layer, as with retinal arterial disease, but also to understanding the field defects in optic neuropathy, as this topography is maintained within the optic nerve.

The most important feature of nerve fiber layer topography is the papillomacular bundle (Fig. 3). The large concentration of retinal ganglion cells at the fovea gives rise to a sheaf of axons that projects directly to the optic disc. Those from the temporal side of the fovea must arch around the large group of fibers from the nasal fovea and thus divide into superior and inferior groups divided by a raphe along the horizontal meridian. This division continues into the peripheral temporal retina; all of these fibers must arch around the massive papillomacular bundle to reach the optic disc, which they enter supero- and inferotemporally. By contrast, fibers from the superior, inferior, and nasal retina are not obstructed by the papillomacular bundle and project in direct radial lines toward the optic disc.

Consequently, there are three classic field defects found with disorders of the optic nerve (Fig. 4):

1. A lesion of the *papillomacular bundle* causes a central scotoma or, if more extensive, a cecocentral scotoma, in which the central defect is continuous with the physiologic blind spot.
2. A lesion of the *temporal retinal fibers* arching around the papillomacular bundle will cause a nasal arcuate defect in either the superior or inferior field. This will come to an abrupt halt at the horizontal meridian in the nasal field. If more extensive, it will follow a curved path around the central macular region and point toward the

blind spot marking the location of the optic disc. A more subtle arcuate defect may have a paracentral scotoma along this path.

3. A lesion of the *nasal retinal fibers* will cause a temporal wedge defect. This will rarely have a border that is aligned along the temporal meridian, as there is no anatomic divide between the upper and lower fibers in the nasal retina.

Combinations of these exist. A superior altitudinal defect, for example, combines damage to the inferior temporal arcuate fibers and the inferior nasal radial fibers. The result is loss of the upper half of vision with a sharp horizontal border nasally and a variable border temporally, which can spare some of the upper temporal field or involve part of the lower temporal field. The macular region can be spared or involved with extension into the lower field, depending on the degree of involvement of the papillomacular bundle. Altitudinal defects are not uncommon with ischemic damage to the optic nerve.

The 1.25 million retinal ganglion cells in each human eye are not a homogeneous group, but divisible into at least 22 different subtypes. Three major types are the parvocellular, magnocellular, and koniocellular groups, which constitute 70%, 8–10%, and 1–10% of the total population, respectively. The parvocellular (P, midget) neurons have small dendritic fields, somas, and axonal diameters and physiologically respond with sustained bursts to light, have color opponency, and conduct information at moderate speeds. Because of these characteristics, they are said to be specialized for stimuli with fine spatial detail (high spatial frequencies) and color (5). Magnocellular (M, parasol) cells have large dendritic fields, somas, and axonal diameters. They respond transiently to the onset and offset of lights, lack color opponency, and have rapid conduction. They are specialized for rapidly changing stimuli (high temporal frequencies) and are poor at fine spatial detail (5). Koniocellular cells receive input from blue-cone bipolar cells and have blue-yellow opponency. The clinical relevance of these subtypes is still being determined (6), but these subtypes are guiding much of the development of newer perimetric strategies (see Chapter 1).

### 2.1. VASCULAR SUPPLY

The inner retina, which contains the retinal ganglion cells and their axons in the nerve fiber layer, is supplied by the central retinal artery, an end branch of the ophthalmic artery.

## 3. OPTIC NERVE

At the optic disc the arrangement of the axons of the retinal ganglion cells is much as expected from the above discussion about the nerve fiber layer. The papillomacular bundle occupies about the central third of the temporal half of the optic disc (7). Beside it the superior and inferior arcuate fibers from the nasal field enter the superotemporal and inferotemporal disc. The rest of the disc is straightforward, with nasal retina (temporal field) flowing into the nasal optic disc, superior retina (inferior field) into the superior aspect, and inferior retina into the inferior disc. For each of these latter regions, the more peripheral fibers occupy the periphery of the optic disc (8–10).

As the optic nerve progresses through the orbit and enters the cranium through the optic canal, just medial to the superior orbital fissure, the retinotopy gradually shifts so that the macular fibers occupy the center of the optic nerve. The approximate arrangement mirrors the origin in the retina, with superior retinal axons

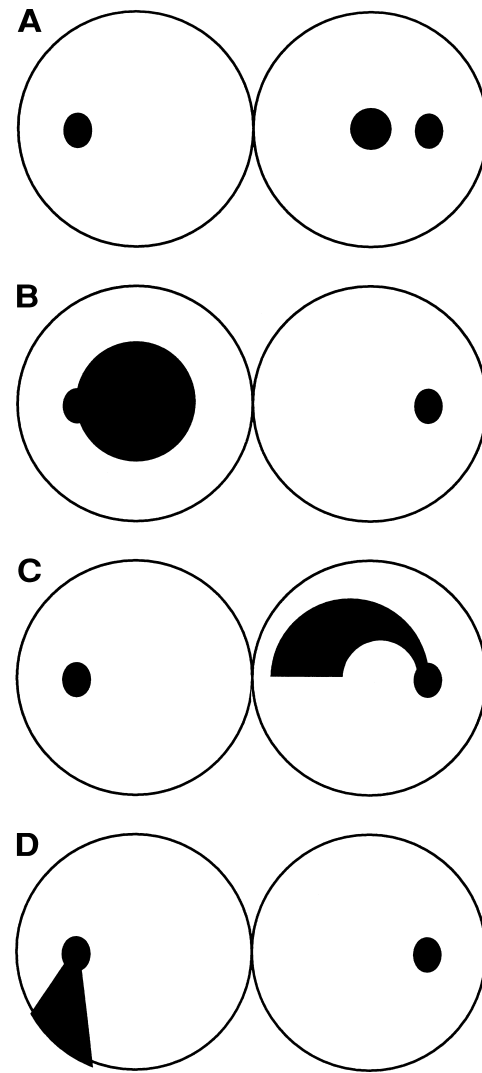


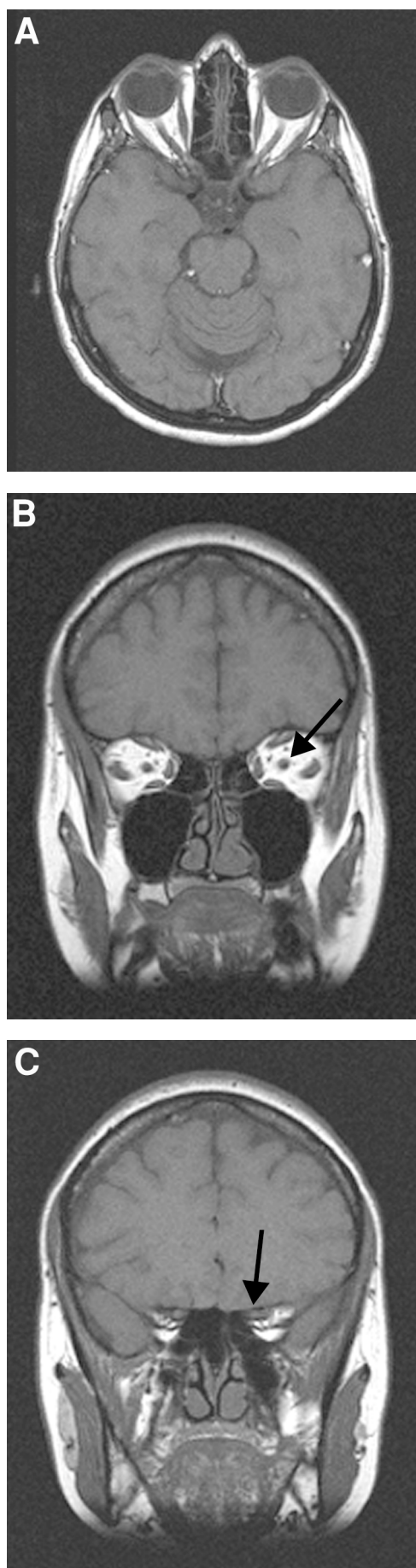
Fig. 4. Illustrations of retinal nerve fiber bundle and optic neuropathic patterns of visual loss: (A) central scotoma; (B) cecocentral scotoma; (C) nasal arcuate defect; (D) temporal wedge defect.

located superiorly, nasal retinal axons nasally, and peripheral axons peripherally.

### 3.1. NEIGHBORHOOD AND VASCULAR SUPPLY

Within the orbit the optic nerve lies within a cone of extraocular muscles that proceeds from the apex of the orbit to insert on the circumference of the globe (Fig. 5). Large mass lesions within the cone cause proptosis but tend not to displace the eye in any particular direction. The optic canal through which the nerve passes is bordered medially by the ethmoid sinuses, and pathology here such as aspergillus infection or Wegener's disease may affect the optic nerve. Laterally lies the superior orbital fissure, which contains cranial nerves III, IV, the first division of V, and VI. A lesion here may cause visual loss with ophthalmoparesis and numbness of the forehead.

The intraorbital optic nerve is supplied by branches of the ophthalmic artery. The optic disc is supplied by the posterior ciliary artery.



#### 4. OPTIC CHIASM

The fibers of the nasal retina decussate in the chiasm, while the temporal retinal fibers do not. Amazingly, this anatomic fact may have been first proposed by Isaac Newton in 1704 (11). The result is that the axons of the temporal field of the contralateral eye join the axons of the nasal field of the ipsilateral eye to form the optic tract, which leaves the chiasm. The hallmark of all visual field defects at or beyond the chiasm is the hard anatomic divide between the left and right visual fields at the vertical meridian.

One clinical point of note about the junction of the optic nerve and the optic chiasm is Wilbrand's knee (12). This is a hypothesized loop of decussating axons from the superior temporal field, in the inferior aspect of the chiasm, which is said to project slightly into the contralateral optic nerve. A lesion here causes a junctional scotoma, which is the combination of an optic neuropathy in the ipsilateral eye with a superotemporal field defect in the other eye that respects the vertical meridian (Fig. 6C). However, more recent studies have suggested that Wilbrand's knee is a myth, an artifact of fixation (13). Rather, junctional scotomata may result from compression of both the intracranial optic nerve and the adjacent optic chiasm by an inferior mass, a not uncommon type of pathology in this region. Regardless of the explanation, the localizing value of a superotemporal field defect in the eye opposite to one with optic neuropathy remains unchallenged. Its clinical importance is that it shifts the etiologic differential diagnosis from the large and varied list associated with optic neuropathy to that of perichiasmal pathology, which implies a mass lesion until proven otherwise.

Because the nasal retina is larger than the temporal retina, slightly more optic nerve fibers (about 53%) decussate than remain uncrossed (14). The macular crossing fibers are diffusely scattered throughout the chiasm, with a slight concentration toward its central and posterior aspects (15). Again, axons from the inferior and superior retina tend to retain the same inferior and superior relations in the chiasm. Beyond this, though, there is much uncertainty on the topography of the chiasm, particularly in humans.

Lesions of the decussating fibers in the optic chiasm cause bitemporal field defects (Fig. 6A). Because the majority of the mass lesions in this region compress the chiasm from below, the superior and central fields are particularly vulnerable. Compression of the lateral aspect of the chiasm can, on rare occasions (16), produce an ipsilateral nasal hemifield defect respecting the vertical meridian (unlike the nasal arcuate defects of optic neuropathy) (Fig. 6B). Lateral compression is more likely from masses in the region of the cavernous sinus, such as giant intracavernous aneurysm, than from pituitary tumors. It is even claimed that bilateral lateral compression might cause binasal field defects. However, nasal field defects in both eyes are far more likely to represent bilateral optic neuropathies than chiasmal lesions, and one would have to make certain that any vertical meridian effect in such a case was not an artifact of perimetry, before embarking on neuroimaging of the sella.

Fig. 5. Magnetic resonance imaging (MRI) of optic nerve, showing orbital T1-weighted images: (A) axial view showing optic nerves from globe to chiasm; two coronal views, one (B) anterior through orbit, showing optic nerves (arrow) within cone of extraocular muscles, and (C) one at the level of optic canal (arrow).

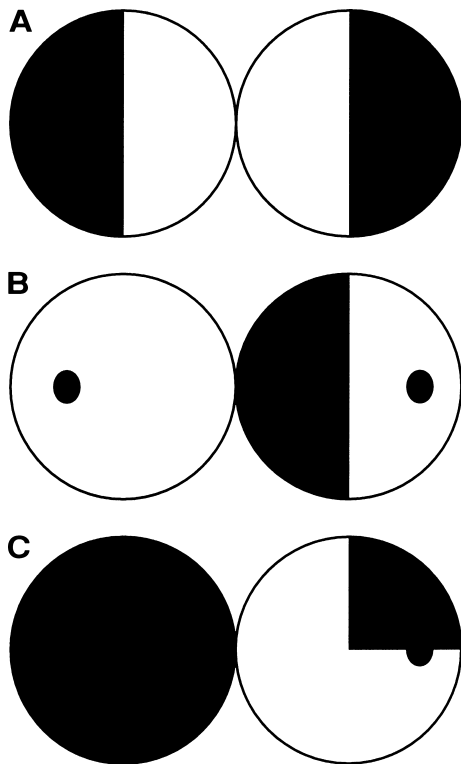


Fig. 6. Illustrations of optic chiasmal patterns of visual loss: (A) bitemporal hemianopia; (B) unilateral nasal hemianopia; (C) junctional scotoma.

Long-standing severe lesions of the optic chiasm will be associated with optic atrophy, as the axons degenerate in retrograde fashion. Loss of the fibers from the nasal retina leads to a characteristic pattern of atrophy. The nasal optic disc will be affected because of loss of fibers from the peripheral nasal retina, as will the temporal optic disc, because this contains axons from the central nasal retina, which lies between the blind spot and the fovea. The superior and inferior aspects of the optic disc, which are occupied by the arcuate fibers coming from the temporal retina, are spared, however. The result is a pattern called “bowtie” or “band” optic atrophy (17) (see Case 65).

#### 4.1. NEIGHBORHOOD AND VASCULAR SUPPLY

The optic chiasm lies superior to the pituitary gland, and inferior to the hypothalamus (Figs. 7 and 8). It is supplied by perforating branches originating from the anterior communicating artery and the A1 segments of both anterior cerebral arteries (18).

### 5. OPTIC TRACT

The visual pathway leaving the optic chiasm is no longer organized as separate structures for each eye (the optic nerves) but as separate structures for each homonymous hemifield. There are two key features about the retinotopy within the optic tract. One is that the correspondence of the retinal map of one eye with that of

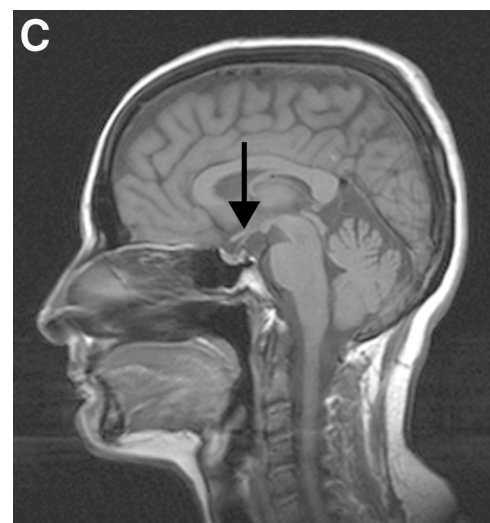
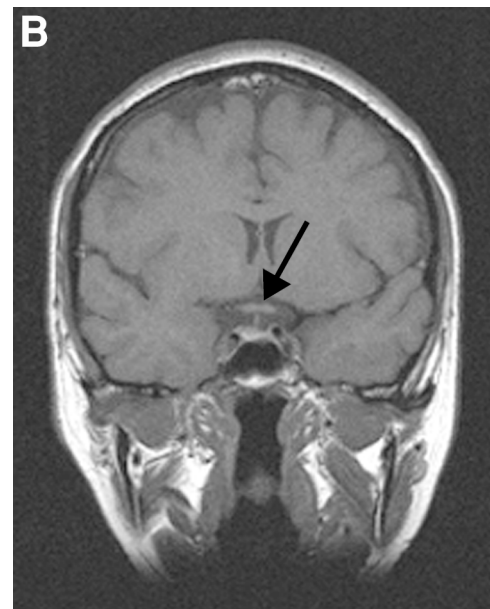


Fig. 7. MRI of optic chiasm showing sella T1-weighted images: (A) axial view showing chiasm (arrow) as X-shaped structure just anterior to infundibulum of pituitary gland; (B) coronal view showing the flat chiasm (arrow) in suprasellar cistern, just above infundibu-

lum, together forming a “T”; (C) midline sagittal view showing chiasm (arrow) just above sella and infundibulum, which slopes down to the pituitary gland.



Fig. 8. Pathology specimen, ventral surface of brain, with temporal lobe removed on right side of image. N = optic nerve, C = optic chiasm, T = optic tract.

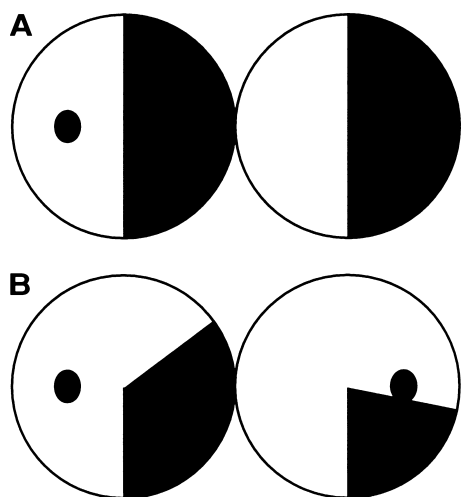


Fig. 9. Illustrations of optic tract patterns of visual loss: (A) complete hemianopia; (B) incongruous partial hemianopia.

the other is only approximate. Hence, partial tract lesions will cause incomplete hemianopias that are quite different in one eye compared with the other (Fig. 9). Although homonymous, in that the defects of the two eyes are in the same hemifield, they are thus incongruous. In general, congruity increases gradually as one proceeds from the chiasm to striate cortex, with a milder degree of incongruity occurring with optic radiation lesions and high congruity typical of striate lesions.

The second feature is a gradual rotation of the retinal map as the tract approaches its termination in the lateral geniculate

nucleus. The superior retina (inferior field) ends up in the dorsomedial tract, the inferior retina in the ventrolateral aspect, and the central field in a dorsolateral position. In addition to this retinotopy, recent data suggest a segregation of magnocellular and parvocellular axons in the tract also, with the magnocellular axons located more ventrally (19).

Because the fibers of the optic tracts are still the axons of the retinal ganglion cells, there will be optic atrophy with long-standing lesions (see Case 69). The eye with temporal field loss may have a bowtie or band optic atrophy (17), as described for chiasmal lesions. The eye with nasal field loss will have more diffuse atrophy, affecting the superior, inferior, and temporal disc, with relative sparing of the nasal disc.

Fibers for the pupillary light reflex also travel in the optic tract, leaving it just prior to the tract's termination in the lateral geniculate nucleus to project to the pretectal nucleus. Asymmetries in field loss from partial tract lesions will thus be associated with a relative afferent pupillary defect in the eye with more profound visual loss. Even with complete tract hemianopia, there will be a relative afferent pupillary defect in the eye with temporal field loss (20–22). Because the temporal field is larger than the nasal field, and the uncrossed nasal field fibers represent 47% of the optic nerve whereas the decussating temporal field fibers constitute 53%, there is more loss of visual input from the eye with temporal hemianopia than from the eye with nasal hemianopia. A relative afferent pupillary defect in the absence of optic atrophy may be the only clue that a homonymous hemianopia stems from optic tract dysfunction (21).

As is true for all homonymous hemifield defects, from the optic tract to striate cortex, visual acuity is not affected unless there is either bilateral damage or additional involvement of the optic chiasm or optic nerves (22,23). One surviving hemifovea is sufficient to support good central spatial resolution.

### 5.1. NEIGHBORHOOD AND VASCULAR SUPPLY

The optic tract travels medial to the anterior temporal lobe and inferolateral to the hypothalamus (Fig. 10). The main arterial supply of the optic tract is the anterior choroidal artery.

## 6. PARASELLAR LESIONS

A word about the impact of mass lesions in the vicinity of the optic chiasm is important. Most practitioners are aware, from the early days of their training, of how lesions such as pituitary macroadenomas compress the optic chiasm and produce bitemporal hemianopia. However, the anatomic position of the optic chiasm with relation to the pituitary fossa is variable (24,25). In some cases, the chiasm is situated anterior to the fossa. A pituitary mass in a patient with such a “prefixed” chiasm may present with homonymous hemianopia rather than bitemporal hemianopia, because the compression may affect one of the optic tracts more than the optic chiasm. Other patients may have a “post-fixed” chiasm situated posterior to the pituitary fossa. In this situation, a mass may present with compressive intracranial optic neuropathy, with or without a junctional scotoma.

## 7. LATERAL GENICULATE NUCLEUS

The LGN, a hat-shaped structure, is located in the ventro-posterolateral thalamus. It is the terminus of the axons of the retinal ganglion cells and contains the cell bodies of the next (and last) neurons in the relay of visual information to striate cortex. In

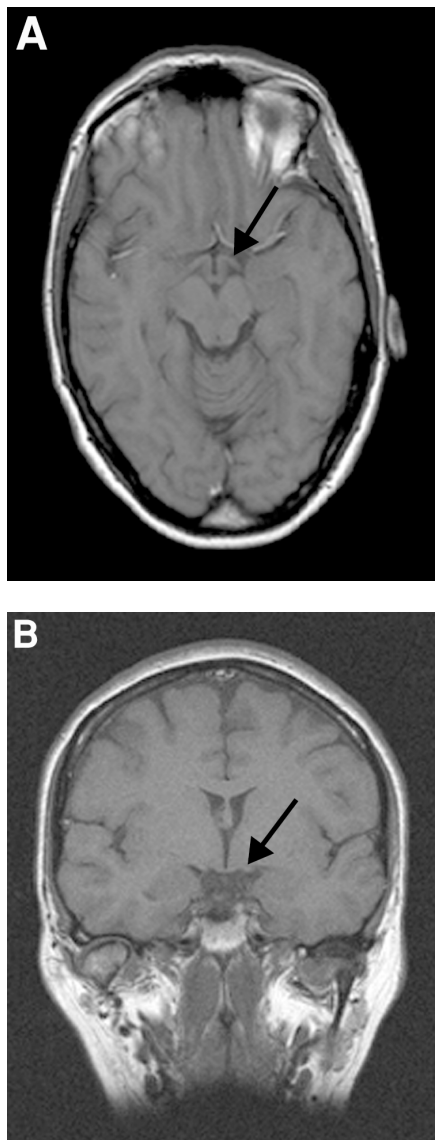


Fig. 10. MRI of optic tract showing T1-weighted images: (A) axial view showing tracts projecting posterolaterally (arrow), anterior to cerebral peduncles and paired medially positioned mamillary bodies; (B) coronal view showing tracts on undersurface of thalami, just superior to hippocampi (arrow).

addition to being a relay station, there is substantial modulation of visual responses in the LGN (26), which involves feed-back and feed-forward projections from extraretinal sources, including superior colliculus, striate cortex, and midbrain nuclei such as the locus ceruleus and dorsal raphe nucleus.

The LGN contains six main horizontal layers (Fig. 11), with each eye providing a segregated innervation to three, in an approximately alternating order (the contralateral eye projects to layers 1, 4, and 6). The ventral two are the magnocellular layers, with the dorsal four being the parvocellular layers. These names derive from the histology of the neuronal cell bodies in these layers. Functionally, the retinal ganglion cells that project to these two different types of layers differ (5). The magnocellular layer receives input from cells with large receptive fields and

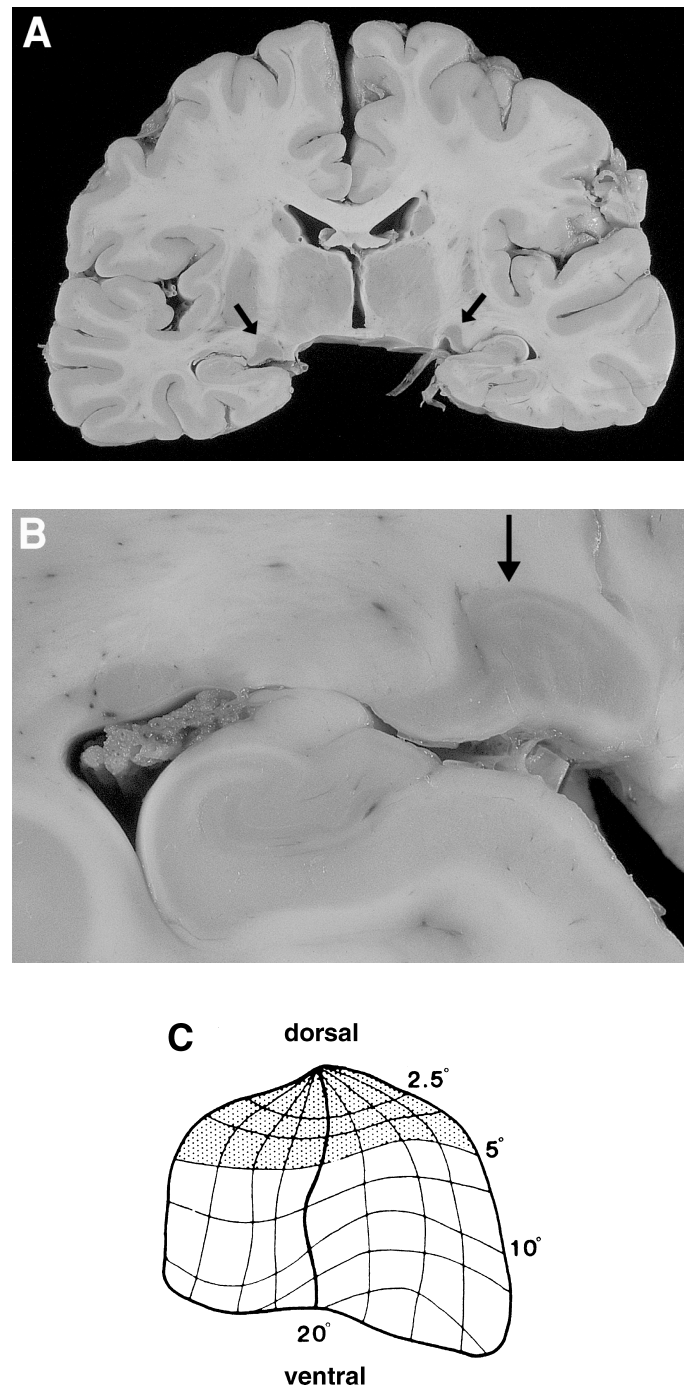


Fig. 11. LGN: (A) pathologic specimen of LGN, coronal section; (B) close-up shows layering; (C) diagram of retinotopy of LGN. Here horizontal layering is shown for retinal eccentricity, not by cell type. (Modified from ref. 49 with permission.)

transient responses to either the onset or offset of light stimuli, whose axons are larger and conduct at a fast rate. The parvocellular layer receives input from neurons with smaller receptive fields, color opponent organization, sustained responses to light and slower conduction along its axons (see Retinal Nerve Fiber Layer, p. 8).

In coronal section the representation of the visual field is an approximate continuation of that found in the terminal optic tract.



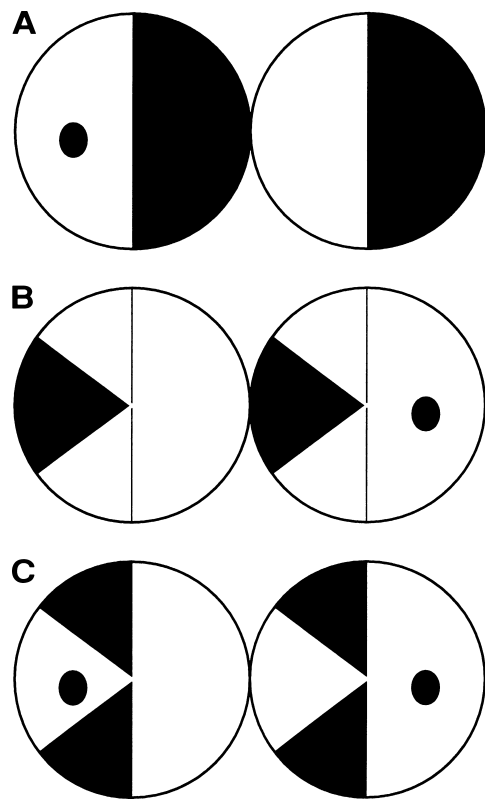


Fig. 12. Illustrations of LGN patterns of visual loss: (A) complete hemianopia; (B) horizontal sectoranopia, from damage to midzone; (C) vertical sectoranopias from lesion sparing midzone.

That is, the macular region occupies a large portion of the dorsal aspect of the nucleus (27), with the periphery located in the broader ventral surface, proceeding from the inferior visual field medially to the superior field laterally (28,29) (Fig. 11C).

The clinical importance of the retinotopy of the LGN derives from lesions that can preferentially affect parts of this structure and spare others. The classic example is an ischemic insult to the LGN. The midzone of the LGN is supplied by the posterior (lateral) choroidal artery, whereas the lateral and medial zones are supplied by the anterior choroidal artery. Infarcts in one or the other zone cause sectoranopias (Fig. 12). In the case of the posterior choroidal artery, the result is a homonymous sector of visual loss straddling the horizontal meridian from the center to the periphery (30). In the case of the anterior choroidal artery, the field defect is the reverse: a hemianopia sparing a wedge straddling the horizontal meridian (31). Incongruity of these hemifield patterns is also the rule with LGN lesions.

Optic atrophy often accompanies LGN lesions. Complete LGN destruction will lead to the same combination of contralateral bowtie atrophy and ipsilateral diffuse atrophy seen with optic tract lesions. The partial damage with sectorial hemianopias causes more subtle optic atrophy restricted to the relevant disc sectors (30,31). Because the afferent fibers subserving the pupillary light reflex have already left the optic tract, there is no relative afferent pupillary defect (RAPD). With incongruous hemianopia and optic atrophy, this is the only feature that distinguishes optic tract from LGN lesions.

### 7.1. NEIGHBORHOOD AND VASCULAR SUPPLY

Nearby thalamic subnuclei include the medial geniculate nucleus ventromedially, the ventral posterior nucleus dorso-medially, and the pulvinar superiorly and dorsally. The medial geniculate nucleus, a relay nucleus in the auditory pathway, gives rise to the acoustic radiations, which pass by the dorsomedial aspect of the LGN on their way to the auditory cortex in the temporal lobe. The optic radiations arise from the dorsolateral surface of the LGN. Ventrally, the hippocampus and parahippocampal gyrus face the LGN across the ambient cistern and the inferior horn of the lateral ventricle. The dual blood supply to the LGN has been discussed already.

## 8. OPTIC RADIATIONS

Optic radiations contain the axons from the LGN to the ipsilateral striate cortex. There may also be direct projections to extrastriate cortex, which may support residual covert or unconscious perception ("blindsight") within the homonymous field defects of striate lesions.

The radiations leave the LGN as a compact bundle. These quickly fan out and pass as a wedge-shaped stream of axons coursing through the white matter of the temporal and parietal lobes to their destination in striate cortex. This fan preserves the topography, with the superior (dorsal, or parietal) radiations representing the superior retina and the inferior (ventral, or temporal) radiations representing the inferior retina. The central field is spread over the lateral surface of the radiations.

One important anatomic feature is the displacement of the temporal radiations anteriorly by the growth of the lateral ventricle during embryogenesis. Thus, this half of the radiations, representing the superior visual field and known as Meyer's loop, projects anterolaterally from the LGN to pass superior to the temporal ventricular horn, deep in the anterior temporal lobe (Fig. 13). Although there is some individual variability, the most forward extent of the radiations is to within about 5 cm of the anterior tip of the temporal lobe. Temporal lobectomies for complex partial seizures do not cause visual loss if they are limited to the most anterior 4 cm of the lobe. The first portion of the field to be affected with lobectomies that proceed a little farther posteriorly is the region adjacent to the vertical meridian (32). With more daring resections, the field defects expand down toward the horizontal meridian, becoming larger pie-shaped wedges. Lesions extending more than 8 cm posterior to the temporal lobe tip start to affect the inferior visual field also. Parietal white matter lesions are most likely to affect the superior optic radiations in isolation. Lesions may also affect the central portion, causing a sectoranopia (33) (Fig. 14).

With damage to the visual pathway distal to the LGN, there is rarely optic atrophy or relative afferent pupillary defects. The only exceptions are long-standing, generally congenital lesions that presumably have been followed by transsynaptic retrograde degeneration.

### 8.1. NEIGHBORHOOD AND VASCULAR SUPPLY

Nearby relations are essentially the cerebral lobes through which the radiations pass. Meyer's loop is close to the hippocampus, and the superior and inferior parietal lobules are lateral to the parietal optic radiations. Thus, associated signs of cerebral damage are frequent with lesions of the optic radiations. Superior quadrantic defects may be associated with complex partial seizures, memory disturbances, or a fluent aphasia if the domi-



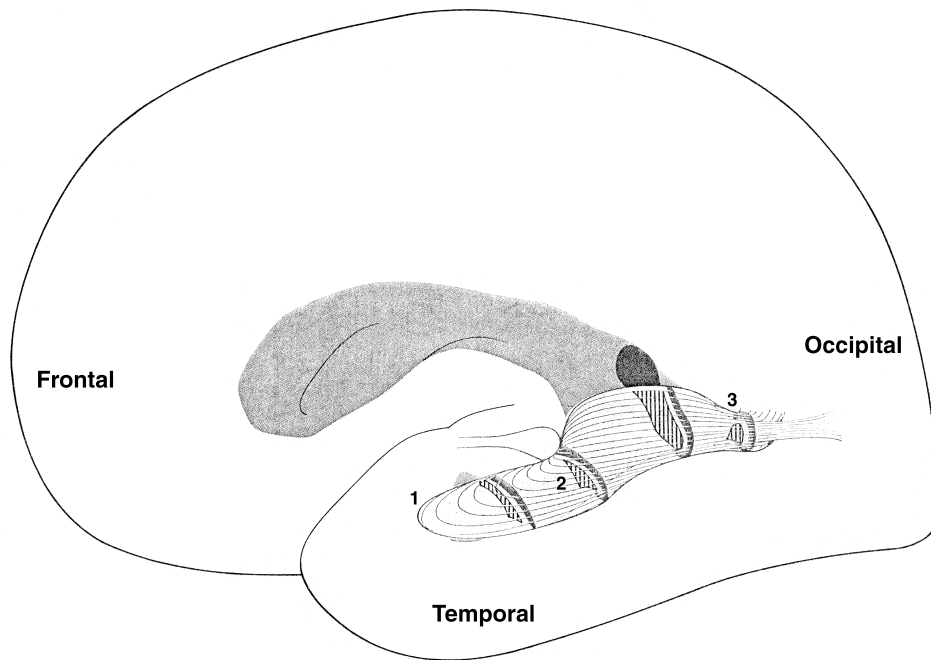


Fig. 13. Diagram of optic radiations showing how radiations loop around anterior temporal horn of lateral ventricle. 1 = temporal optic radiations (Meyer's loop); 2 = central bundle; 3 = upper (parietal) bundle, representing inferior visual field. (From ref. 50 with permission.)

nant (usually left) temporal lobe is involved. Inferior quadrantic defects may be associated with somatosensory disturbances in the contralateral hand, or impaired smooth pursuit eye movements for targets moving toward the side of the lesion. Dominant hemisphere lesions may have Gerstmann syndrome (acalculia, finger anomia, right-left disorientation, and agraphia), fluent or global aphasia, or alexia with or without agraphia.

The blood supply to the optic radiations is primarily the middle cerebral artery. The terminal portion enters into the territory of the posterior cerebral artery, and the portion just exiting from the LGN is supplied by the anterior choroidal artery.

## 9. STRIATE CORTEX

Striate cortex is the primary visual cortical area ("visual area 1" or V1, also known as calcarine cortex or Brodmann area 17) and the termination of the optic radiation and the retino-geniculocalcarine relay. It occupies the depths and upper and lower banks of the calcarine fissure, running anteroposteriorly along the medial surface of the occipital lobe, approximately parallel to the cerebellar tentorium (Figs. 15 and 16). The parietooccipital fissure forms a reasonably reliable marker of the anterior extent of striate cortex. The posterior limit is more variable, extending from the medial occipital surface over the first 1 or 2 cm of the superficial posterior surface of the occipital pole.

The retinotopic map proceeds from the fovea posteriorly at the occipital pole to the far periphery anteriorly at the parieto-occipital fissure (34,35). The superior bank of the calcarine fissure corresponds to the superior retina, and hence the inferior visual field,

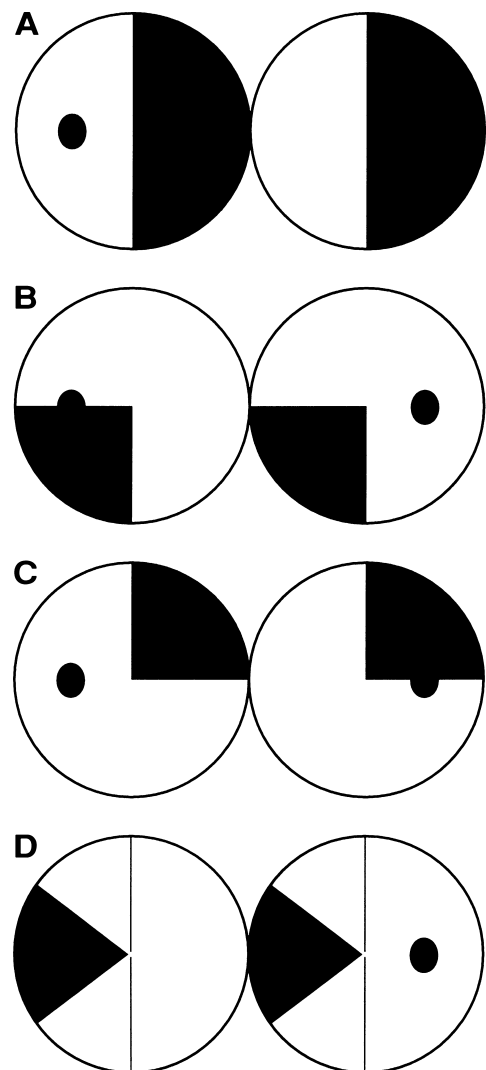
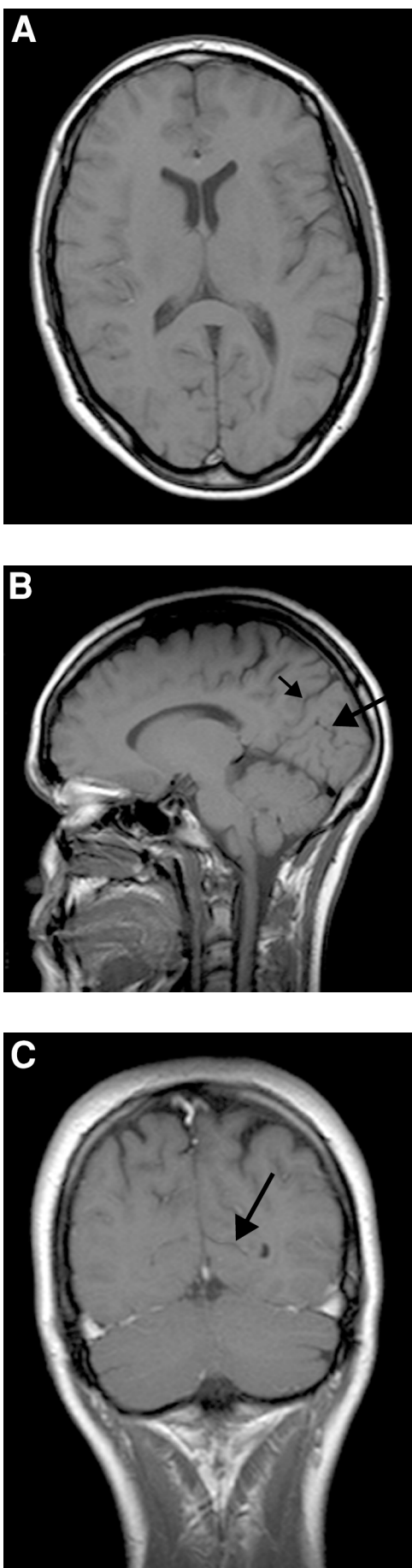


Fig. 14. Illustrations of optic radiation patterns of visual loss: (A) complete hemianopia; (B) lower quadrantanopia, from parietal optic radiation damage; (C) upper quadrantanopia, from temporal optic radiation damage; (D) sectoranopia, from damage to midzone of combined parietal and temporal optic radiations.



while the inferior bank represents the inferior retina and superior visual field (Fig. 16). The most anterior part of striate cortex corresponds to the monocular temporal crescent, the temporal region in the contralateral eye that lies beyond the nasal limits ( $60^\circ$ ) of the ipsilateral eye. As in most of the visual system, there is a gradient of decreasing neuronal resources as one proceeds more peripherally in the field. The “cortical magnification factor” is a value in an equation that captures this relation (36,37). Over half of striate cortex is devoted to the central  $10^\circ$  (38,39). The striate cortex contains a mix of monocular and binocular cells in ocular dominance columns, and the retinal maps of the two eyes are closely registered with each other, resulting in high congruity of the various field defects from lesions there (Fig. 17).

### 9.1. NEIGHBORHOOD AND VASCULAR SUPPLY

The striate cortex is supplied by branches of the posterior cerebral artery (40). A parieto-occipital branch supplies the superior calcarine bank, a posterior temporal branch supplies the inferior bank, and a calcarine branch supplies the central region posteriorly. The most important variation among individuals is the location of the watershed between the posterior and middle cerebral arteries at the occipital pole, with respect to where the foveal representation lies. In some individuals, a good portion of the fovea may be supplied by the middle cerebral artery, whereas in others the posterior cerebral artery supplies all striate cortex (40). The result is that some individuals with posterior cerebral arterial infarcts will have hemianopia with sparing of the fovea, while others will have complete hemianopia.

Structures anterior to striate cortex in the medial occipital lobe include the lingual and fusiform gyri, and farther afield the hippocampus; all of these are supplied by the posterior cerebral artery and not uncommonly are damaged along with striate cortex during infarction. Variable degree of memory impairment, dyschromatopsia, and rarely visual agnosia may result.

## 10. EXTRASTRIATE CORTEX

Beyond the striate cortex the stream of visual information changes drastically. Instead of a serial relay with information modulation at each stage, perceptual data now fan out into a large array of cortical regions, each specialized for a particular type of visual function. This array is organized in a loose hierarchy, with feed-forward inputs, back projections, and interconnections among many regions (41). The retinal topography of these areas is much coarser than that in striate cortex and the preceding elements of the visual pathway, and it is gradually lost farther up the hierarchy, as the receptive fields of neurons become larger and larger, with some eventually spanning the entire ipsilateral and contralateral visual field. Instead, visual processing becomes more and more specialized, with regions selective for faces, colors, and motion, for example. This selectivity can be grouped approximately into a dorsal stream through occipitoparietal cortical regions that is dedicated to visuospatial analysis (the “Where” path) and a ventral stream through medial occipitotemporal

Fig. 15. MRI of striate cortex. (A) The axial view shows the more convoluted sulci and gyri of striate cortex on the medial surface of the occipital lobe. Locating striate cortex is easier on (B) the sagittal view, where the calcarine fissure (long arrow) runs parallel to the tentorium, and (C) the coronal view, where this fissure is easily seen on the medial occipital lobe (arrow). Short arrow shows parieto-occipital fissure in (B).

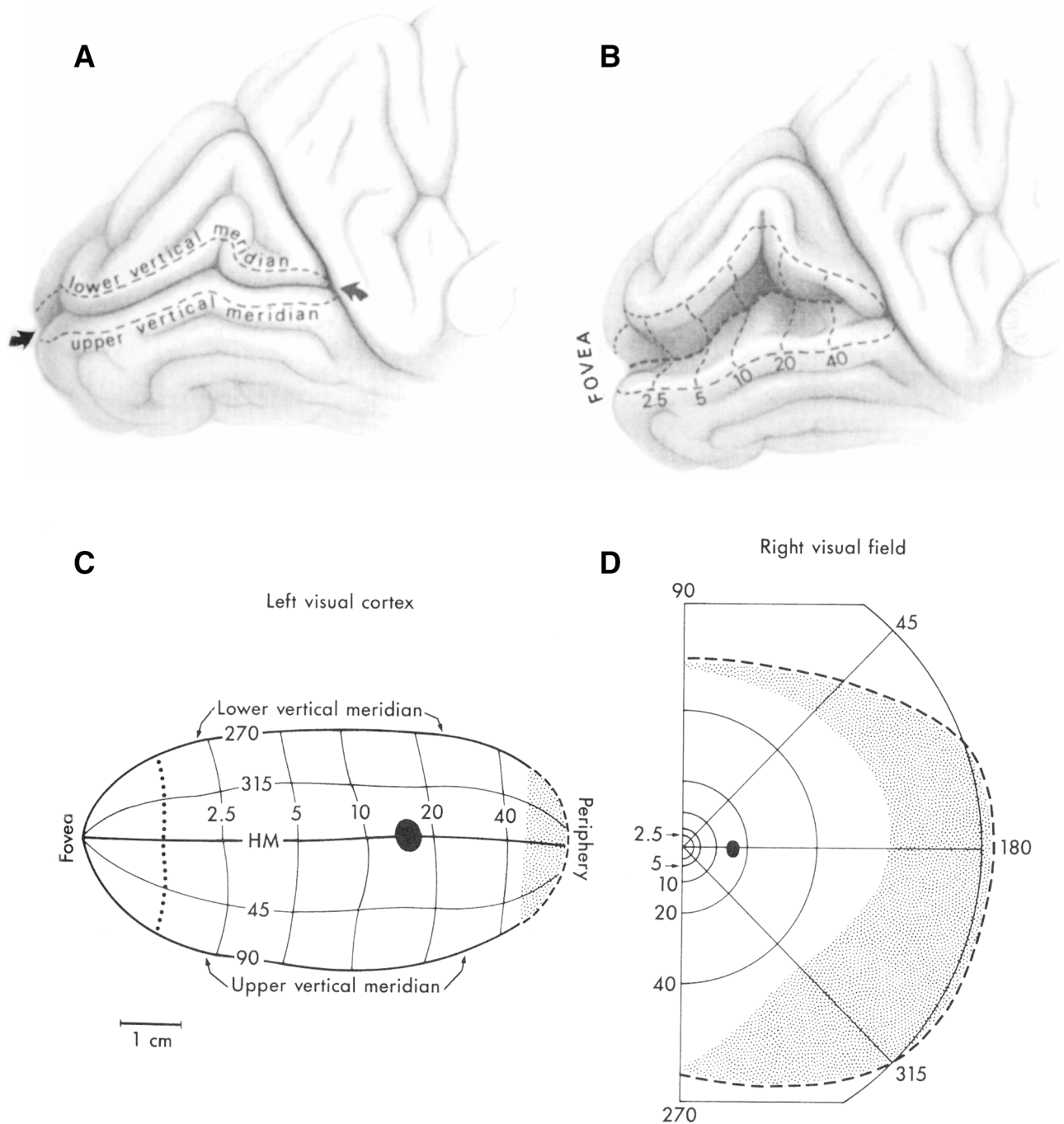


Fig. 16. Representation of vision in striate cortex showing views of striate cortex with occipital pole, or posterior aspect, at left side (A,B) and flat maps of topography of visual cortex (C) and corresponding visual field (D). HM = horizontal meridian ( $180^\circ$ ). Grey stipple is the monocular temporal crescent. (From ref. 38 with permission.)

regions that focuses on object recognition (the “What” path) (42). Lesions of these regions are typified not so much by field defects as by interesting highly selective defects, such as achromatopsia, the loss of color discrimination, and prosopagnosia, the inability to recognize familiar faces.

Do lesions of extrastriate cortex ever lead to visual field defects? It has been proposed that quadrantic defects may arise from V2 lesions (43); however, this remains a point of contention, and is not supported by data from nonhuman primate studies. Areas V4 and V5 do have a coarser retinotopic map, and lesions in these

regions may generate visual dysfunction limited to one hemifield or even one contralateral quadrant. However, these hemifield defects are selective, in that they affect some types of vision but spare others. Lesions of the fusiform gyri, which contain a human region specialized for color perception, cause hemiachromatopsia, the impaired discrimination of hue in the contralateral hemifield (44,45). Lesions of the lateral occipitotemporal cortex, which likely contains a human homolog of area V5, can cause a hemiakinetopsia, in which the perception of complex motion patterns is degraded in the contralateral field (46,47).

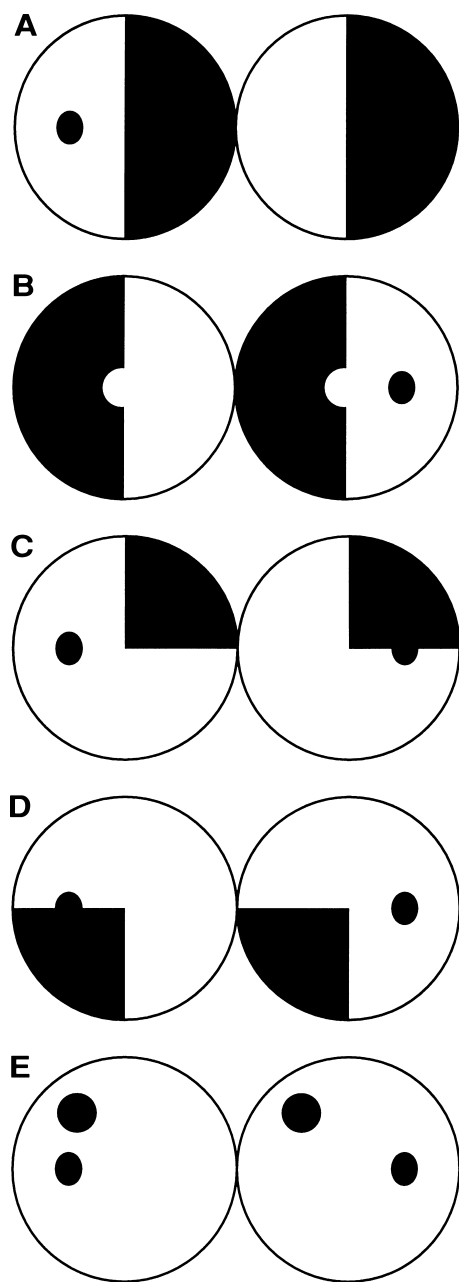


Fig. 17. Illustrations of striate patterns of visual loss. (A) complete macula-splitting hemianopia; (B) macula-sparing hemianopia, from sparing of occipital pole; (C) upper quadrantanopia, from lesion of inferior calcarine bank; (D) lower quadrantanopia, from lesion of superior calcarine bank; (E) congruous hemifield scotomata, from lesion of midzone of inferior calcarine bank.

Current perimetric devices are not designed to pick up these selective hemifield deficits. Adequate testing for such problems requires specialized software or equipment, not commonly found in most clinics or standardized for clinic use.

## REFERENCES

1. Hecht S, Shlaer S, Pirenne M. Energy, quanta and vision. *J Gen Physiol* 1942;224:665–699.
2. Land E. Recent advances in retinex theory. *Vision Res* 1986; 26:7–21.
3. Zeki SM. A century of cerebral achromatopsia. *Brain* 1990;113: 1721–1777.
4. Trick G, Trick L, Kilo C. Visual field defects in patients with insulin-dependent and noninsulin-dependent diabetes. *Ophthalmology* 1990;97:475–482.
5. Schiller P, Logothetis N. The color-opponent and broad-based channels of the primate visual system. *Trends Neurosci* 1990;11:392.
6. Sample PA. What does functional testing tell us about optic nerve damage? *Surv Ophthalmol* 2001; 45(Suppl. 3):S319–S324.
7. Radius R, Anderson D. The course of axons through the retina and optic nerve head. *Arch Ophthalmol* 1979;97:1154–1158.
8. Wolff E, Penman G. The position occupied by the peripheral retinal fibers in the nerve fiber layer and at the nerve head. *Trans Ophthalmol Soc UK* 1950;70:35.
9. Hoyt W, Luis O. Visual fiber anatomy in the infrageniculate pathway of the primate: uncrossed and crossed retinal quadrant fiber projections studied with Nauta silver stain. *Arch Ophthalmol* 1962; 68:94–106.
10. Minckler D. The organization of nerve fiber bundles in the primate optic nerve head. *Arch Ophthalmol* 1980;98:1630–1636.
11. Brewster D. *Memoirs of the Life, Writings and Discoveries of Sir Isaac Newton*. Vol. 1. Edinburgh: Thomas Constable and Co., 1855:426.
12. Wilbrand H. Schema des Verlaufs der Sehnervenfaser durch das Chiasma. *Z Augenheilkd* 1926;59:135–144.
13. Horton J. Wilbrand's knee of the primate optic chiasm is an artefact of monocular enucleation. *Trans Am Ophthalmol Soc* 1997; 95: 579–609.
14. Kupfer C, Chumbley L, Downer JDC. Quantitative histology of optic nerve, optic tract, and lateral geniculate nucleus of man. *J Anat* 1967;101:393–401.
15. Hoyt W, Luis O. The primate chiasm: details of visual fiber organization studied by silver impregnation techniques. *Arch Ophthalmol* 1963;70:69–85.
16. Cox T, Corbett J, Thompson H, Kassell N. Unilateral nasal hemianopia as a sign of intracranial optic nerve compression. *Am J Ophthalmol* 1981;92:230–232.
17. Unsöld R, Hoyt WF. Band atrophy of the optic nerve: the histology of temporal hemianopia. *Arch Ophthalmol* 1980;98:1637–1638.
18. Perlmutter D, Rhoton A. Microsurgical anterior cerebral-anterior communicating-recurrent artery complex. *J Neurosurg* 1976;45:259–272.
19. Tassinari G, Campara D, Balercia G, Chilosi M, Martignoni G. Magno- and parvocellular pathways are segregated in the human optic tract. *Neuroreport* 1994;5:1425–1428.
20. Bell R, Thompson H. Relative afferent pupillary defect in optic tract hemianopias. *Am J Ophthalmol* 1978;85:538–540.
21. O'Connor P, Mein C, Hughes J, Dorwart RH, Shacklett DE. The Marcus Gunn pupil in incomplete optic tract hemianopias. *J Clin Neuroophthalmol* 1982;2:227–234.
22. Newman S, Miller N. Optic tract syndrome: neuro-ophthalmologic considerations. *Arch Ophthalmol* 1983;101:1241–1250.
23. Frisén L. The neurology of visual acuity. *Brain* 1980;103:639–670.
24. Renn W, Rhoton AJ. Microsurgical anatomy of the sellar region. *J Neurosurg* 1975;43:288–298.
25. Rhoton A, Harris F, Renn W. Microsurgical anatomy of the sellar region and cavernous sinus. *Clin Neurosurg* 1977;24:54–85.
26. Sillito A, Murphy P. The modulation of the retinal relay to the cortex in the dorsal lateral geniculate nucleus. *Eye* 1988;2(Suppl.): S221–S232.
27. Kupfer C. The projection of the macula in the lateral geniculate nucleus of man. *Am J Ophthalmol* 1962;54:597–609.
28. Shacklett DE, O'Connor PS, Dorwart RH, Linn D, Carter JE. Congruous and incongruous sectoral visual field defects with lesions of the lateral geniculate nucleus. *Am J Ophthalmol* 1984;98:283–290.
29. Connolly M, van Essen D. The representation of the visual field in parvocellular and magnocellular layers of the lateral geniculate nucleus in the monkey. *J Comp Neurol* 1984;226:544–564.
30. Frisén L, Holmegaard L, Rosenkrantz M. Sectoral optic atrophy and homonymous horizontal sectoranopia: a lateral choroidal artery syndrome? *J Neurol Neurosurg Psychiatry* 1978;41:374–380.

31. Fris  n L. Quadruple sectoranopia and sectorial optic atrophy: a syndrome of the distal anterior choroidal artery. *J Neurol Neurosurg Psychiatry* 1979;42:590–594.
32. Jacobson D. The localizing value of a quadrantanopia. *Arch Neurol* 1997;54:401–404.
33. Carter J, O’Connor P, Shacklett D, Rosenberg M. Lesions of the optic radiations mimicking lateral geniculate nucleus visual field defects. *J Neurol Neurosurg Psychiatry* 1985;48:982–988.
34. Inouye T. *Die Sehstorungen bei Schussverletzungen der kortikalen Sesphare*. Leipzig: Engelmann, 1909.
35. Holmes G, Lister W. Disturbances of vision from cerebral lesions with special reference to the cortical representation of the macula. *Brain* 1916;39:34–73.
36. Rovamo J, Virsu V. An estimation and application of the human cortical magnification factor. *Exp Brain Res* 1979;37:495–510.
37. Tolhurst D, Ling L. Magnification factors and the organization of the human striate cortex. *Hum Neurobiol* 1988;6:247–254.
38. Horton J, Hoyt W. The representation of the visual field in human striate cortex: a revision of the classic Holmes map. *Arch Ophthalmol* 1991;109:816.
39. McFadzean R, Brosnahan D, Hadley D, Mutlukan E. Representation of the visual field in the occipital striate cortex. *Br J Ophthalmol* 1994;78:185–190.
40. Smith C, Richardson W. The course and distribution of the arteries supplying the visual (striate) cortex. *Am J Ophthalmol* 1966; 61:1391–1396.
41. Felleman D, Van Essen D. Distributed hierarchical processing in the primate cerebral cortex. *Cereb Cortex* 1991;1:1–47.
42. Ungerleider L, Mishkin M. Two cortical visual systems. In: Ingle DJ, Mansfield RJW, Goodale MS, eds. *The Analysis of Visual Behaviour*. Cambridge, MA: MIT Press, 1982:549–586.
43. Horton JC, Hoyt WF. Quadrantic visual field defects: a hallmark of lesions in extrastriate (V2/V3) cortex. *Brain* 1991;114:1703–1718.
44. K  lme   HW. Pure homonymous hemiachromatopsia: findings with neuroophthalmologic examination and imaging procedures. *Eur Arch Psychiatr Neurol Sci* 1988;237:237.
45. Paulson HL, Galetta SL, Grossman M, Alavi A. Hemichromatopsia of unilateral occipitotemporal infarcts. *Am J Ophthalmol* 1994; 118:518.
46. Greenlee M, Lang H, Mergner T, Seeger W. Visual short-term memory of stimulus velocity in patients with unilateral posterior brain damage. *J Neurosci* 1995;15:2287–2300.
47. Plant G, Laxer K, Barbaro N, Schiffman J, Nakayama K. Impaired visual motion perception in the contralateral hemifield following unilateral posterior cerebral lesions in humans. *Brain* 1993;116: 1303–1335.
48. Curcio C, Sloan K, Kalina R. Human photoreceptor topography. *J Comp Neurol* 1990;292:497–523.
49. Miller N, Newman N. Walsh and Hoyt’s *Clinical Neuro-ophthalmology*. Baltimore: Williams & Wilkins, 1998.
50. Ebeling U, Reulen H-J. Neurosurgical topography of the optic radiation in the temporal lobe. *Acta Neurochir (Wien)* 1988;92:29–36.



Field of Vision

A Manual and Atlas of Perimetry

Barton, J.J.S.; Benatar, M.

2003, XI, 335 p. 232 illus., Hardcover

ISBN: 978-1-58829-175-2

A product of Humana Press