ANATOMICAL CORRELATION OF VISUAL FIELD DEFECTS

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INTRODUCTION

This chapter includes a review of:

- Pertinent visual pathway anatomy
- Localization of lesions causing VF defect
- Pre-chiasmal VF losses
- Optic chiasm VF losses
- Post-chiasmal VF losses

ANATOMICAL CORRELATION OF VF DEFECTS

VF interpretation, like most clinical procedures, involves the association of findings to the pertinent anatomical structures. The correlation of VF losses to the neural & visual pathways and their **clinico-pathological implication** begins with recognizing & classifying particular characteristics of VF defects. Proper categorization of VF abnormalities provides clues to the **location** of causative lesions. In turn, the location of lesions often points towards the possible **etiology** and facilitate the diagnosis of conditions. One must therefore know and understand the visual pathway very thoroughly and bear the complete anatomical picture in mind when VF defects are analyzed.

PERTINENT VISUAL PATHWAY ANATOMY

The visual pathway is a sensory tract that carries afferent impulses to the brain. The pathway is a 4 neuron pathway that begins at the retina and terminates at the occipital cortex.

1. The **1st order neuron** starts from the end organ photoreceptors (rods & cones) and finishes with a synapse at the bipolar cells within the retina.
2. The **2nd order neuron** is the bipolar cell within the retina which transmits the neural signal from the photoreceptors to the ganglion cells.
3. The **3rd order neuron** involves the long ganglion cells. Each nerve fiber will cross the surface of the retina, enter the ONH, travel through the chiasm, optic tract and terminate with a synapse in the lateral geniculate body (LGB).

The nerve fibers of the retina are arranged in a **characteristically constant topographical pattern** which is carried on relatively **unchanged** throughout the 3rd neuronal pathway. It is <u>crucial</u> to understand this organization to properly correlate VF losses to anatomical lesions involving the 3rd neuron.

The retinal nerve fiber pattern consists of the **papillomacular bundle** that composes **65%** of all nerve fibers, the superior and inferior **arcuate bundle** which arch around the papillomacular bundle from the horizontal raphe to the ON and the superior & inferior **radiating bundles** that pass through the nasal portions of the retina into the optic nerve (Fig. 5.67).

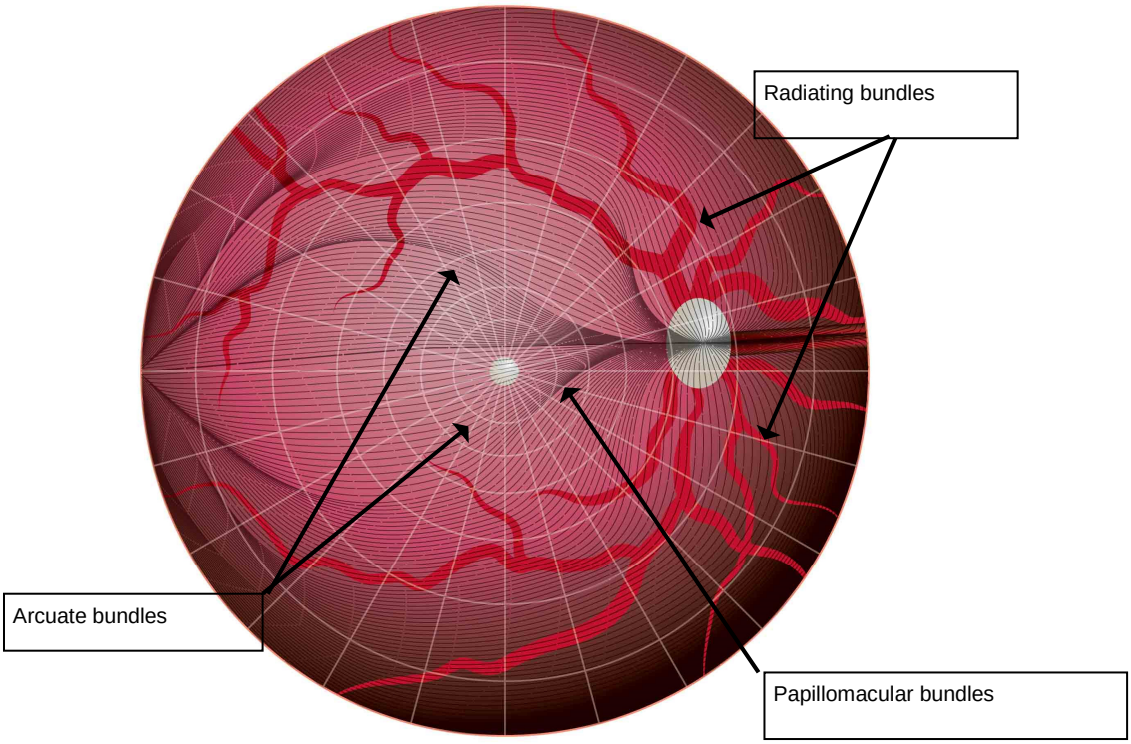


Figure 5.67: *Topographical pattern of retinal nerve fibre layer*

Modified from Harrington DO, Drake MV, The Visual Fields:Text & Atlas of Clinical Perimetry, C.V. Mosby, 1990.

The **papillomacular bundle** will pass directly into the ON on its temporal side adjacent to the central vessels (Fig. 5.68). In the intraorbital ON, these fibers are grouped in the same pattern as in the retina keeping their temporal position. As they approach the chiasm, the papillomacular bundle move from the temporal side to a more central position and lose the bundle arrangement to become **spread out and scattered** mixing freely with all fibers. In the chiasm, the macular fibers are dispersed in almost all areas except for a the anterior & infero-posterior parts. In the optic tract, the macular fibers rise to a more superior aspect.

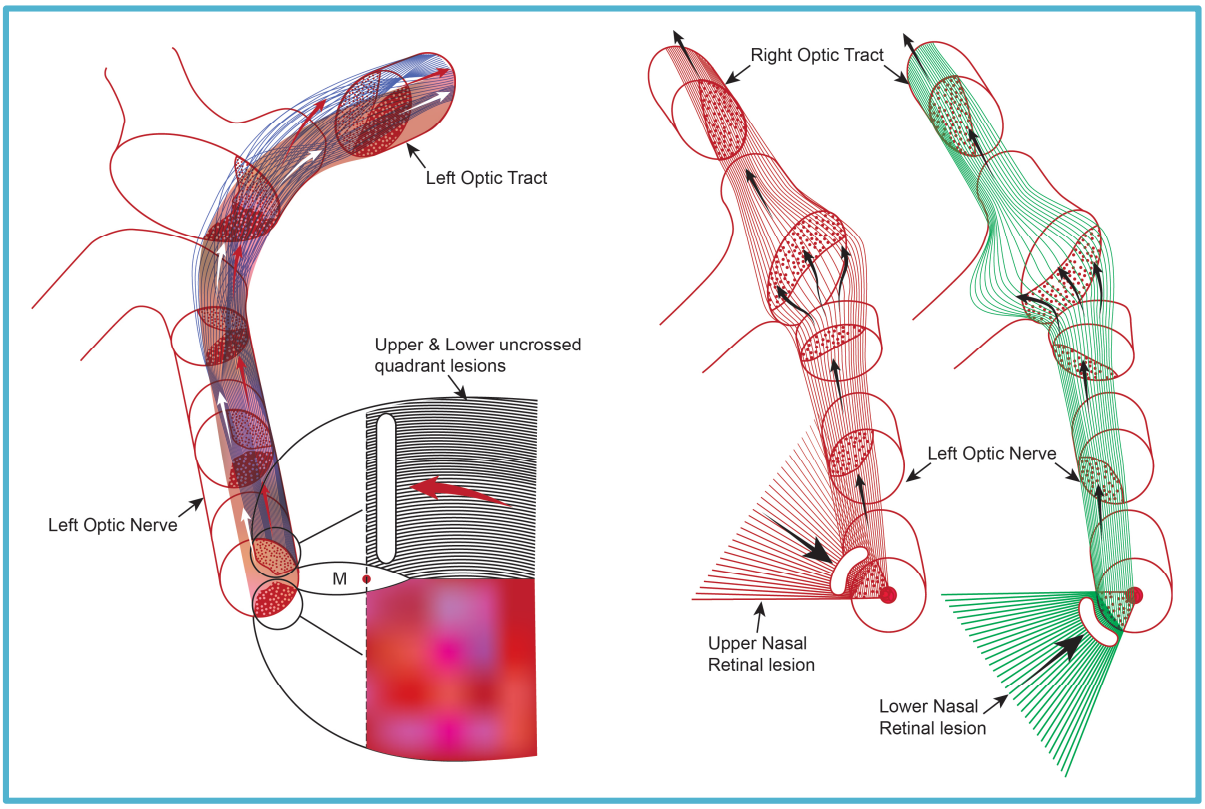


Figure 5.68: *Organisation of the nerve fibers in optic tracts and chiasm*

Drawn from Harrington DO, Drake MV, The Visual Fields: Text & Atlas of Clinical Perimetry, C.V. Mosby, 1990.

The **superior and inferior temporal fibers** of the peripheral retina (nasal VF) will maintain their respective position in the upper and lower outer quadrant of the ONH. Since the papillomacular fiber bundle enter on the temporal side of the optic disc, the fibers from the temporal retina are "squeezed" into the superior and inferior poles of the disc. More posteriorly in the ON, however, the macular fibers take a more central location, and the fibers from the temporal retina assume a more temporal location in the nerve. The temporal fibers remain on the same side and become the **uncrossed fiber bundles** in the optic tract.

The **superior & inferior nasal fibers** (temporal VF) also maintain their position throughout the ON. In the chiasm, these will decussate to become the **crossed fiber bundles. Inferior fibers will decussate immediately while the superior fibers will wait until the end of the chiasm**. Some inferior fibers crossing in the anterior chiasm will loop anteriorly into the contralateral nerve before heading posteriorly forming what is known as "**Knee of Wilbrand**".

In the **optic tract**, all superior fibers (crossed & uncrossed) become medial, all inferior fibers become lateral. The macular fibers rise into a more superior position.

The 3rd order neuron synapses in the **LGB** which is a small 6-layer oval structure. Crossed fibers terminate in layers 1,4,6 while uncrossed fibers terminate in layers 2,3,5. Corresponding points of both retinas represented in all layers of the LGB and are thus somewhat distant from each other.

1. The **4th order neuron** starts from the LGB and forms the **optic radiations** that will end in the cerebral cortex (geniculo-calcarine radiations) (Fig. 5.69). The optic radiations run in a **broad band** towards the cortex. The dorsal fibers (superior fibers) that leave the LGB run backward in a fairly direct route through the **parietal lobe.** The ventral fibers (inferior fibers) initially run forward and downward into the **temporal lobe** to form **Meyer's loop** before running backward to the cortex. The visual fibers lose the "compact" arrangement in the optic radiation and become widespread making them susceptible to a wider range of lesions. However, **throughout the optic radiations, upper and lower fibers will retain their respective upper & lower positions**.

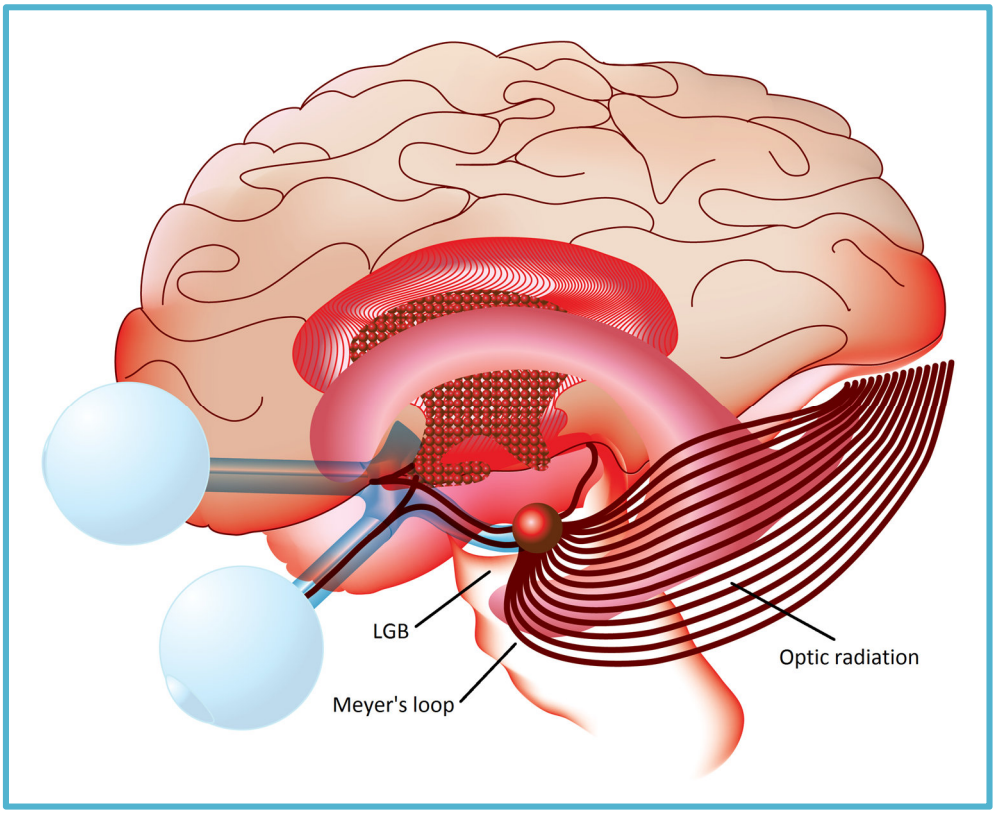


Figure 5.69: *Anatomy and disposition of optic radiations*

Inspired by Harrington DO, Drake MV, The Visual Fields: Text & Atlas of Clinical Perimetry, C.V. Mosby, 1990.

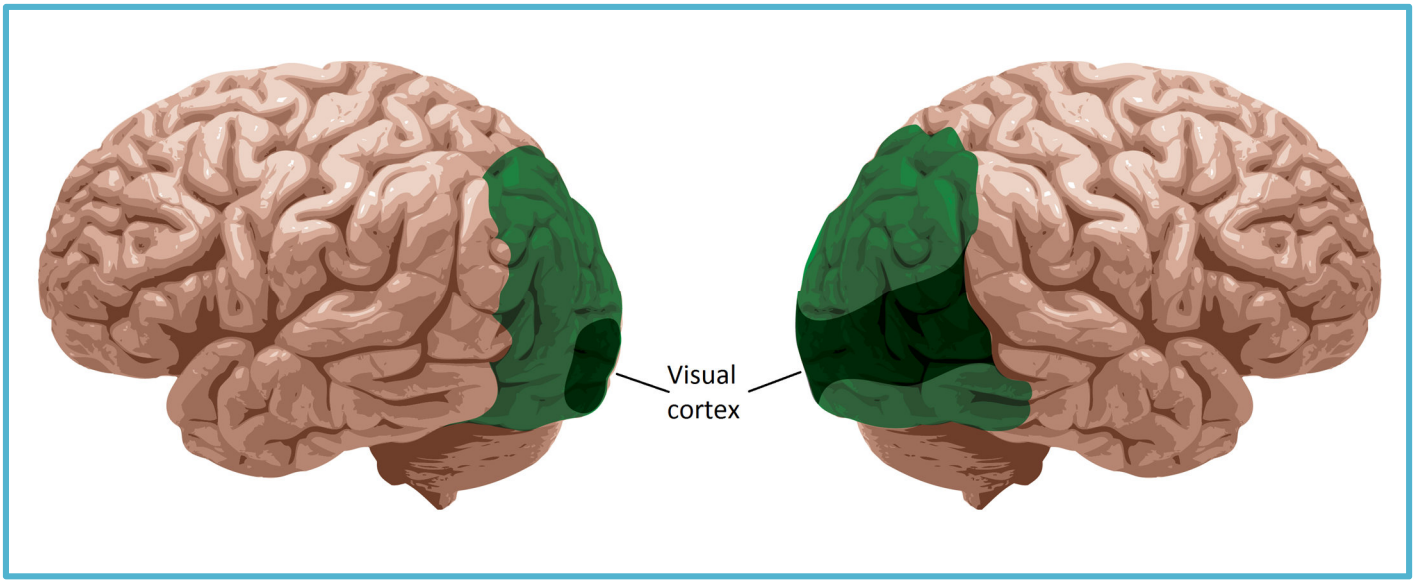


Figure 5.70: *Lateral and medial views of the occipital lobe*

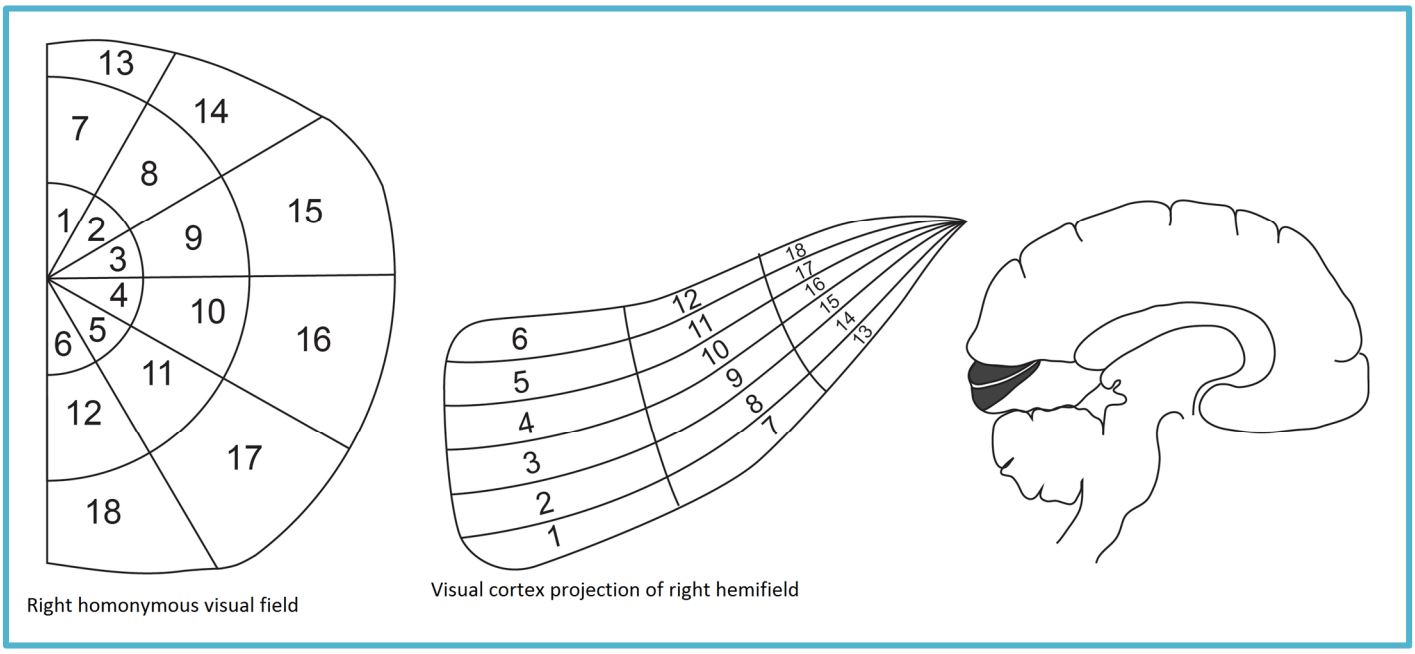
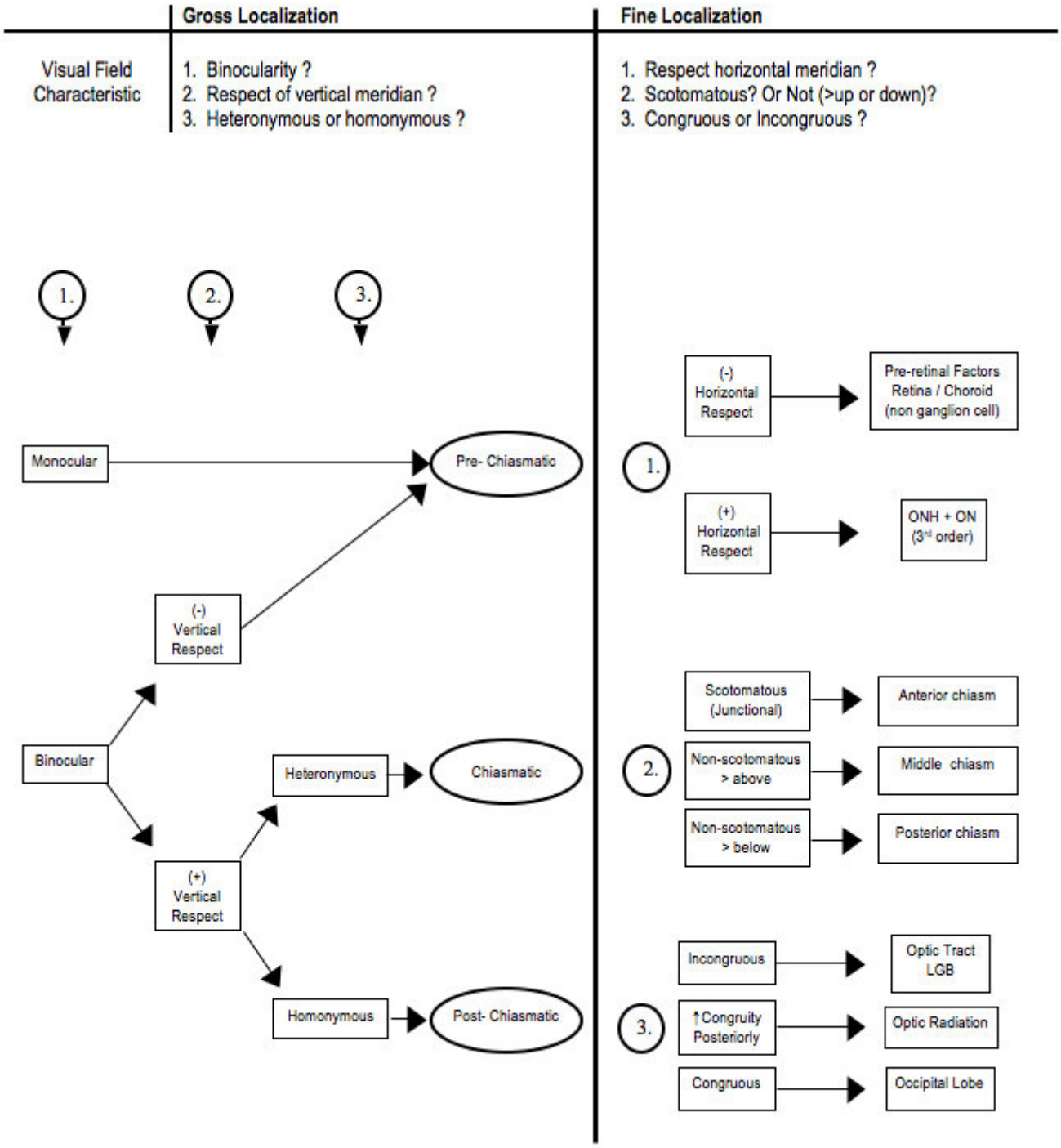


Figure 5.71: *Mapping of the visual cortex*

Diagrams inspired by Harrington DO, Drake MV, The Visual Fields: Text & Atlas of Clinical Perimetry, C.V. Mosby, 1990.

LOCALIZATION OF LESIONS CAUSING VF DEFECT

Given the anatomical organization of the visual pathway, certain characteristics of VF defects will provide clues to the localization of causative lesions. A first set of VF defect descriptors will provide clues to gross location while a second set will help finer localization:



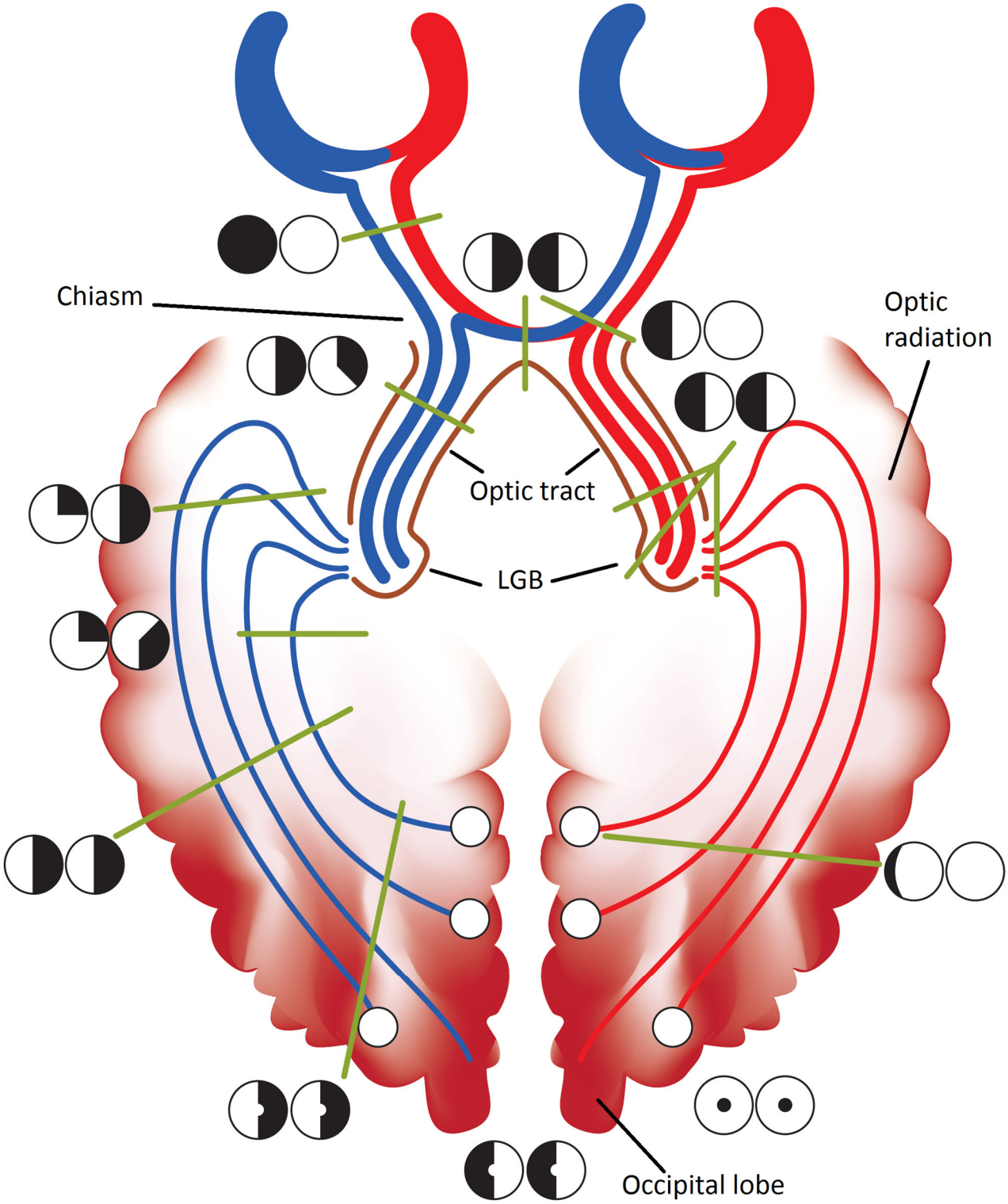


Figure 5.72: *The appearance of visual field loss resulting from lesions at various points throughout the visual pathway*

Diagrams inspired by Harrington DO, Drake MV, The Visual Fields: Text & Atlas of Clinical Perimetry, C.V. Mosby, 1990.

PRE-CHIASMAL VF LOSSES

Pre-chiasmal VF defects are monocular. Although diseases affecting the VF often occur bilaterally, the damage & resulting VF loss in each eye is independent and essentially monocular. Pre-chiasmal defects can take on many shapes. Based on the appearance and the causes, they can grossly be placed in 2 categories:

No respect of the horizontal meridian

- **Preretinal Type**
  - Preretinal factors are abnormalities that produce light blockage, light scatter or image defocus (e.g. cataracts, medial opacities, miosis, etc.). These usually result in a generalized visual depression but they can also result in focal irregular reduction in sensitivity. Pre-retinal factors are often revealed by anterior segment evaluation.
- **Retinal & Choroidal Type**
  - Lesions of the retina and choroid will produce defects that do not follow the anatomical feature of the visual system. The defects correspond directly to the affected areas often as they are perceived ophthalmoscopically (e.g. retinal detachments, chorioretinal scars, ARMD, etc.).

Respect of the horizontal meridian

Optic Nerve Type

Visual field defects produced by ON lesions generally respect the characteristic arrangement of the NFL within the retina and ON.

Several forms of defects can be observed:

- **Paracentral scotoma** occur in the early stages of conditions that affect the ganglion cells (e.g. early glaucoma) in an irregular fashion
- **Arcuate scotoma** involve the curvilinear nerve fiber bundles that arc over and below fixation. Lesions of the optic nerve fibers such as glaucoma (through fusion of paracentral scotoma), optic disc drusen and anterior ischemic optic neuropathy often produce arcuate scotoma that end abruptly along the horizontal midline
- **Nasal steps** occur by uneven damage in the superior & inferior portions of the NFL
- **Central scotoma** occur in conditions that affect primarily the macular fibers. When <u>unilateral</u>, this defect is characteristically found in patients with compressive lesions of the ON (e.g. papilledema, drusen, etc.) or demyelinating optic neuropathies. When <u>bilateral</u>, differential diagnosis includes nutritional deficiencies, toxic optic neuropathies, and hereditary disorders
- **Cecocentral scotoma** are classically found in patients with toxic optic neuropathies but can be seen with any condition that produces a central scotoma
- **Altitudinal defects** are characteristically associated with Anterior Ischemic Optic Neuropathy (AION) which is a vascular disease of the ON
- **Temporal wedge-shaped sector defects** is a rare pie-shape defect that arises from the blind spot and does not respect the horizontal meridian. Associated with glaucoma, it results from damage to the nasal fibers.

OPTIC CHIASM VF LOSSES

Chiasmal syndromes are usually, but not solely, caused by tumors especially pituitary tumors (~50%) & craniopharyngiomas (~25%). Of all brain tumors, 25% are in the chiasmal area. Half of the chiasmal syndromes present initially with **vision loss complaints**. Because chiasmal lesions interrupt the 3rd neuron pathway, chiasmal syndromes will often be accompanied by ON "company" such as VA loss, RAPD, ONH atrophy, loss of colour vision, etc. Because of the wide spread of macular fibers, **VA is usually normal** in chiasmal lesions unless the optic nerve is also involved.

Chiasmal syndromes will cause **variable & mixed VF losses**. The appearance of the defect will depend on the **particular lesion** affecting the chiasm and the **direction** with which it insults the chiasm. The resulting VF defect will also depend on the **anatomical location of the chiasm.** The position of the chiasm with respect to the bony tuberculum sellae which holds the pituitary body is variable among individuals: 80% of chiasms sit on the posterior 2/3 of the sellae, 12% directly above it, 4% are pre-fixed (short ON) and 4% are post-fixed (long ON).

The **distinguishing** characteristic of VF losses arising from chiasmal lesions is the **bitemporal hemianopic nature** of the defect. With the understanding that most chiasmal insults arise from the pituitary body and hence from below, chiasmal VF defects roughly fall within 2 categories: scotomatous and non-scotomatous. Remember that these are only guidelines for the most commonly occurring causes of chiasmal syndrome. VF defects arising from the chiasm can truly take on any shape.

Scotomatous bitemporal hemianopia (a.k.a. Junctional defects)

- **Anterior chiasmal lesions**
  - Anterior chiasm lesions typically produce junctional scotoma which show a central scotoma quadrantic or hemianopic) that respects the vertical midline in one eye and a peripheral superior temporal defect in the other eye. This results from ipsilateral optic nerve damage & contralateral damage of the crossing fibers in the Knee of Wilbrand

Non-scotomatous bitemporal defects that respect the hemianopic vertical midline

- **Middle chiasm lesions: denser above**
  - Lesions affecting the central body of the chiasm present (mid) peripheral bitemporal VF defects. The defect is usually denser above than below because crossed fibers of the chiasm are more sensitive to compression than uncrossed fibers due to their anatomical location.
- **Posterior chiasm lesions: denser below**
  - Posterior chiasm lesions destroy primarily posterior crossing fibers resulting in (mid) peripheral bitemporal VF defects with one eye presenting a denser loss inferiorly.

Binasal hemianopia due to lateral chiasmal compression is possible but extremely rare. It can only be produced by chiasmatic lesions that compress and displace the chiasm laterally against the internal carotid arteries thus producing binasal field loss. Associated homonymous VF defects are also possible when chiasmal lesions extend posteriorly onto the optic tracts.

POST-CHIASMAL VF LOSSES

Post chiasmal VF defects are caused by lesions affecting the Optic Tract (3%), the Lateral Geniculate Body (1%), the Optic Radiations (58%) and the Occipital Lobe (38%).

Because of the widespread distribution of the anatomical pathway retro-chiasmatically, VF defects can be produced by a number of different insults. Lesions that produce VF defects in this region, however, tend to **"keep company"** by producing other neurological signs and symptoms that help localize & identify the responsible lesions. Lesions in the optic tracts or lateral geniculates interrupt nerve fibers from the retinal ganglion cells of both eyes. These lesions can produce characteristic **retrograde ONH and NFL atrophy** as well as an RAPD of the eye with the most affected fibers. Temporal lobe lesions often cause seizures and form visual hallucinations while Parietal lobe lesions may have associated hemiparesis, visual perceptual difficulties, agnosias, apraxias and right-left confusion. Lesions of the primary visual cortex may produce unformed visual hallucinations.

Post-chiasmal lesions are characterized by their essential **homonymous hemianopic** aspect where the same side of the visual space is affected (temporal side of one eye, nasal side of the other). In general, post-chiasmal defects can be classified according to the **law of congruity**:

The more posterior the lesion,
The more congruous the VF defect.

Incongruous type defects

The visual pathway "spreads out" retro-chiasmatically such that corresponding retinal elements lie further away from each other. Proximal post-chiasmal lesions will thus affect corresponding fibers at different degree. Affections of the optic tract, LGB & optic radiations will thus mostly produce VF losses that are **incongruous**.

Increasing Congruity

Nerves carrying corresponding retinal information move gradually closer to each other such that the visual pathway becomes more compact as it progresses towards the occipital lobe. Lesions in the posterior optic radiations affect corresponding fields more equally. VF defects therefore appear progressively more congruous with more posterior insults.

Lesions in the **temporal lobe** (25%) tend to be **incongruous** homonymous hemianopias that are **denser above**. The temporal lobe fibers spread out into Meyer's loop which holds the inferior areas of both retinas. When damaged, **"pie-in-the-sky"** defects are produced.

Lesions in the **parietal lobe** (33%) tend to be **slightly more congruous** homonymous hemianopias that involve the inferior VF to a greater extent. A tighter bundle of fibers representing superior retinal areas courses through the parietal lobe. Damage to these fibers results in **"pie-on-the-floor"** defect.

Congruous

Due to the specific retinotopic arrangement of the striate cortex, fibers relaying input from corresponding retinal elements of the two eyes lie extremely close together by the time they reach the occipital cortex. VF defects, therefore, become **exquisitely congruous** with occipital lesions.

Lesions of the occipital cortex may **spare fixation** (i.e. not split it in hemifields). The anatomical reason for this macular sparing remains controversial. Some explain it on the basis of the dual blood supply of the occipital tip by the middle and posterior cerebral arteries while the rest of the occipital cortex is supplied only by the posterior cerebral artery. Vascular insults affecting the posterior cerebral artery may thus spare the macula. Lesions that affect **strictly the occipital tip** may produce congruous **central** homonymous hemianopias.