LOCALISATION OF VISUAL FIELD DEFECTS

AUTHORS

Anand Moodley: Department of Neurology, University of KwaZulu Natal

PEER REVIEWER

Leonard Messner: Illinois College of Optometry

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INTRODUCTION

The field of view is everything that causes light to fall onto the retina at any given time. This input is processed by the visual system, which computes the visual field as the output.

The normal human visual field extends:

- 60 degrees nasally
- 100 degrees temporally
- 60 degrees superiorly
- 75 degrees inferiorly

VISUAL FIELD TESTING BY CONFRONTATION

FACIAL AMSLER

- It assesses approximately 10 degrees of the visual field
- The macular region is tested
- It involves asking the patient to count or add fingers

Hand comparison

- Detects subtle altitudinal or hemifield defects (e.g. patient can't find place on reading)
- Both hands may be seen but one may be vague or appears as a shadow
- Darker on one side compared to the other (on affected side)

PERIPHERAL FIELD

- Tests 30-60 degrees of field
- Useful for detecting constriction
- Use red target for subtle central field defects
- Blue targets for peripheral field defects
- Document the way the patient sees it.

The **kinetic confrontation** allows for the screening of visual field defects. However, the problem with this technique is the Riddoch's phenomenon where the patient with field defects is able to perceive movement but not static objects. A stationary target is therefore preferred.

VISUAL PATHWAY AND LESIONS

VISUAL PATHWAY

The visual pathway (Figure 2.1) consists of the following:

- Optic nerve
- Optic chiasma
- Optic tract
- Lateral geniculate nucleus
- Optic radiations

A lesion in any of the above-mentioned structures will result in a visual field defect.

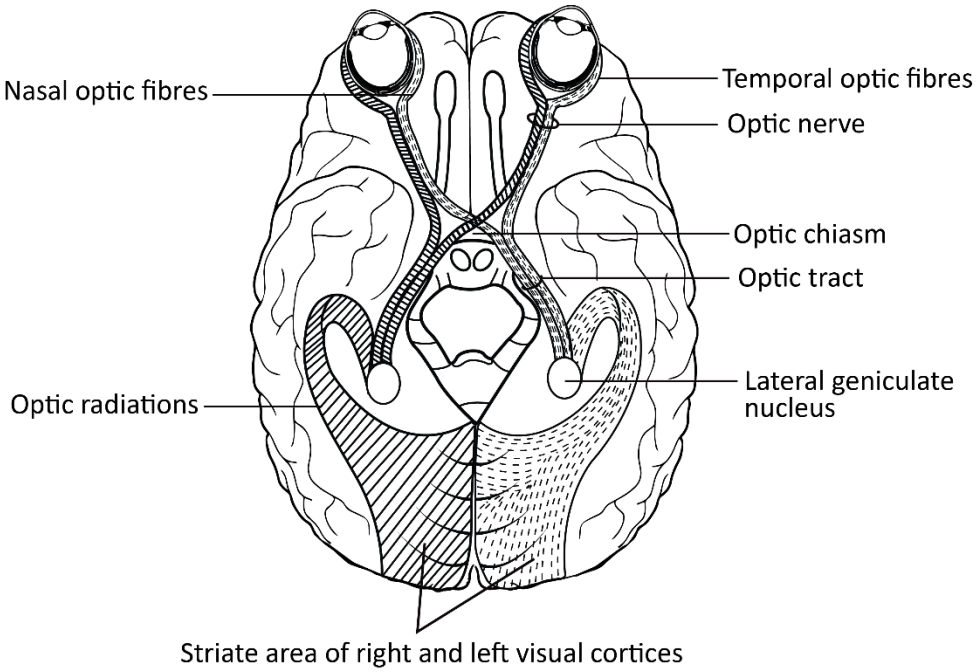

***Figure 2.1**: The visual pathway*

INTRACRANIAL LOCALISATION OF VISUAL FIELD DEFECTS

- Optic Nerve Lesions
- Optic Chiasm Lesions
- Optic Tract lesions
- Lateral Geniculate Body lesions
- Optic radiation lesions
- Anterior Visual cortex
- Macular cortex
- Cortical Blindness
- Occipital lobe lesions have macula sparing. E.g. post cerebral artery stroke will produce a homonymous hemianopia with macula sparing because the middle cerebral artery still supplies the macula area.

The Visual field defects associated with the various possible locations of a pathological lesion

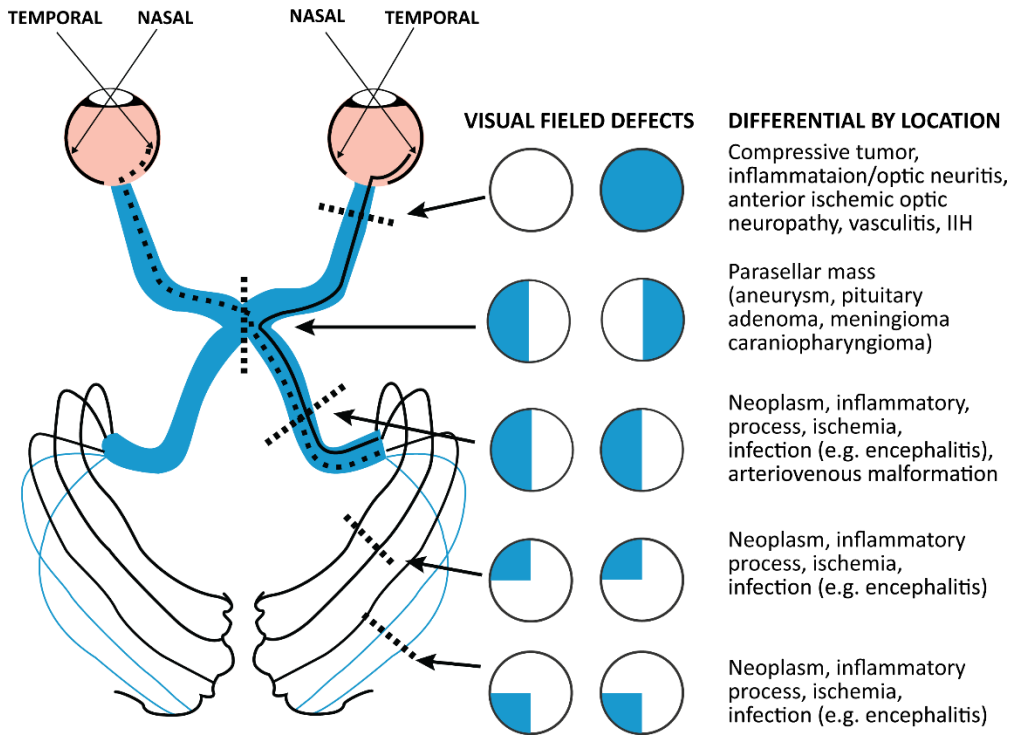

***Figure 2.2**: Visual field defects*

LESIONS ALONG THE OPTIC PATHWAY

OPTIC DISC LESIONS

Lesions at the optic disc result in an enlarged blind spot or constricted visual field. This visual field defect can be due to:

- Coloboma of the disc
- Drusen
- Glaucoma
- Optic disc swelling, such as papilloedema

OPTIC NERVE LESIONS

Optic nerve lesions result in a central scotoma or monocular blindness. The colour vision is impaired early. This defect can result due to:

- Optic neuritis
- Retrobulbar neuritis
- Toxic optic neuropathy
- Leber's hereditary optic neuropathy
- Primary optic atrophy
- Optic nerve glioma

OPTIC CHIASM LESIONS

Optic chiasm lesions can be divided into:

- Anterior chiasmal lesions
- Posterior chiasmal lesions
- Lateral chiasmal lesions.

Anterior chiasmal lesions cause a peripheral field defect in the opposite eye referred to as a junctional scotoma. This can occur due to any inferior compression of the junction of the optic nerve and optic chiasm. Lesions giving a junctional scotoma lie inferior to the optic chiasm. At the chiasm, 53% of the axons from the nasal retina cross the midline to join the uncrossed temporal fibers.

Central Chiasmal lesions produce a bitemporal hemianopia of the macula region only because macula fibers are found here. This could be due to:

- Pituitary Tumours
- Carotid siphon aneurysms
- Chordomas
- Craniopharyngiomas

Lateral Chiasmal lesions: If it is unilateral, it produces an ipsilateral nasal field defect. If it is bilateral, it produces a binasal hemianopia. This could be due to: arteriosclerotic carotid artery, dilated 3rd ventricle.

OPTIC TRACT LESIONS

These lesions cause incongruous homonymous hemianopias. Incongruous defects are those in which the shape of the defect is different in the two half fields.
Causes of optic tract lesions include:

- Pituitary adenoma
- Craniopharyngioma
- Chordoma
- Meningioma
- Multiple sclerosis is an uncommon cause.

LATERAL GENICULATE BODY LESIONS

Lesions of the lateral geniculate body cause incongruous homonymous hemianopias. These lesions are rare and occur due to ischaemia or tumours.

OPTIC RADIATION LESIONS

The optic radiations consist of two parts:

- The temporal lobe
- The parietal lobe.

Temporal lobe lesions cause incongruous contralateral superior quadrantanopia aka “Pie in the sky”.
This could be due to:

- Strokes

- Tumours
- Encephalitis
- Abscess

Parietal lobe lesions result in incongruous contralateral inferior quadrantanopia aka “Pie on the floor”
This could be due to:

- Strokes
- Tumours
- Abscess
- Encephalitis

PEARL:

PITS: Parietal = Inferior defect, Temporal = Superior defect

VISUAL CORTEX LESIONS

Anterior visual cortex

- This results in macular sparing hemianopia.
- The posterior cerebral artery supplies it.
- Lesions could be due to strokes, tentorial meningioma or migraine

Macular cortex

- This results in small congruous homonymous hemianopia of the macula area only.
- The middle cerebral artery supplies it.
- Causes include strokes, tentorial meningioma, migraine.

Intermediate calcarine cortex

- Rare
- Half ring of visual loss surrounding the intact macula, and preserved peripheral vision.
- Causes include strokes, tentorial meningioma, migraine

CORTICAL BLINDNESS

Total destruction of the occipital cortex by trauma or stroke will result in cortical blindness. There is preservation of the light reflexes. Anton’s Syndrome, which is due to a lesion of the visual association cortex, results in denial of blindness.

Bilateral blindness with intact pupil reflexes in a patient without primary ocular disease suggests cortical blindness and requires brain imaging.