

## TOPICAL DIAGNOSIS OF RETROCHIASMAL VISUAL FIELD DEFECTS

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**Keywords:** relative afferent defect, pupillary defect, hemiakinesia, incongruous homonymous hemianopia, double homonymous hemianopia

**Abstract:** Unilateral lesions of the visual sensory pathway beyond the optic chiasm - the optic tract, lateral geniculate body, optic radiation, or striate cortex - produce homonymous visual field defects without loss of visual acuity. Homonymous defects in the visual fields develop slowly when they are caused by compression and rapidly when they are caused by haemorrhage, ischemia, or inflammation. Compressive lesions generally cause progressive loss of visual field from the periphery of the field to the centre. Upon the decompression of the visual system, improvement typically first occurs in the central region and continues towards the periphery. Defects for coloured objects invariably appear before disturbances either for form or for black and white objects, a reason to use coloured stimuli routinely in the examination of visual fields. When homonymous visual field defects arise from vascular lesions, the onset of the field defects is sudden. Such defects include complete homonymous quadrantanopsias and hemianopias, incomplete homonymous quadrantanopsias and hemianopias with varying degrees of congruity, and homonymous paracentral scotomas. When and if improvement occurs, the central field clears first and may be followed by gradual enlargement of the peripheral fields if they have been affected.

**Cuvinte cheie:** defect pupilar aferent relativ, hemiachinezia pupilară, hemianopie omonimă incongruentă, hemianopsia sectorială, hemianopsia omonimă dublă

**Rezumat:** Leziunile unilaterale ale căii senzoriale vizuale dincolo de chiasma optică-tractul optic, corpul geniculat lateral, radiația optică, sau cortexul striat- produc defecte de câmp vizual omonim fără scăderea acuității vizuale. Defectele omonime în câmpul vizual se dezvoltă încet când ele sunt cauzate de compresiune și rapid când ele sunt cauzate de hemoragie, ischemie, sau inflamație. Leziunile compresive în general cauzează scăderea progresivă a câmpului vizual de la periferia câmpului la centru. La decomprimarea sistemului vizual, îmbunătățirea caracteristică se întâmplă prima dată în regiunea centrală și continuă spre periferie. Defectele de obiecte colorate apar invariabil înaintea oricărei distorbanțe de formă sau de obiecte alb și negru, motiv de folosire a stimulilor colorați în examinarea câmpului vizual. Când defectele de câmp vizual omonim apar de la leziuni vasculare, începutul defectelor câmpului vizual este brusc. Asemenea defecte includ quadranopsia omonimă completă și hemianopsia, quadranopsia omonimă incompletă și hemianopsia cu variate grade de congruitate și scotom paracentral omonim. Când și dacă se întâmplă îmbunătățire, câmpul central se limpezește primul și poate fi urmat de lărgire graduală a câmpurilor periferice dacă ele fuseseră afectate.

Although lesions affecting the optic tracts are infrequent, they are of great importance because they are located in the first region beyond the optic chiasm where lesions produce a homonymous visual field defect. Lesions of the optic tract account for about 3% to 11% of cases of homonymous hemianopia. The causes are varied and include tumours, vascular processes, demyelinating disease, and trauma.

Patients with optic tract lesions often have specific findings that permit the recognition of the location of the lesion on clinical grounds alone. All patients with a complete homonymous hemianopia caused by an isolated optic tract lesion have a relative afferent pupillary defect in the eye contralateral to the side of the lesion (i.e. the eye with the temporal field loss).

Another pupillary phenomenon, that is sometimes associated with lesions of the optic tract that produce a complete or nearly complete homonymous hemianopia is pupillary

hemiakinesia (hemianopic pupillary reaction or Wernicke's pupil). Optic tract lesions do not cause loss of visual acuity nor do they affect colour vision unless they also damage the optic chiasm or the intracranial portions of one or both optic nerves.

Patients with a complete or nearly complete homonymous hemianopia from an optic tract lesion eventually develop a characteristic pattern of optic atrophy. There is a „band” of horizontal pallor of the optic disc in the eye contralateral to the lesion (with temporal field loss). Not all patients with optic tract lesions have a complete homonymous hemianopia. Many patients have a complete or incomplete homonymous quadrantanopia or an incomplete hemianopia. Such field defects are quite incongruous and may also be scotomatous. Neurologic deficits that may occur in patients with lesions of the optic tract include hypothalamic symptoms and signs and contralateral hemiparesis.

Criteria for optic tract syndrome:

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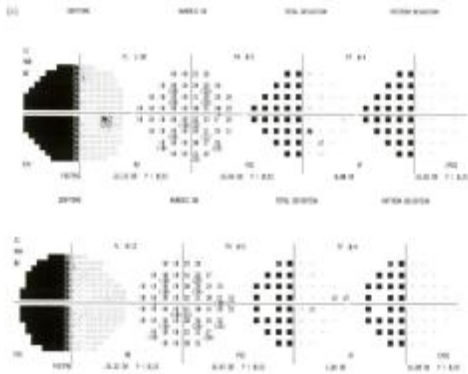
Article received on 14.02.2012 and accepted for publication on 23.05.2012  
ACTA MEDICA TRANSILVANICA September 2012;2(3):199-201

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- a. Incongruous homonymous hemianopia.
- b. Bilateral retinal nerve fibre layer atrophy or optic atrophy.
- c. Pupillary abnormalities:
  - relative afferent pupillary defect: on side opposite the lesion (eye with temporal field loss)
  - Wernicke's pupil: light stimulation of a „blind” retina causes no pupillary reaction, while light projected on an „intact” retina produces normal pupillary constriction.
  - Behr's pupil: anisocoria with larger pupil on the side of hemianopia.

### CONGRUOUS VISUAL FIELD LOSS

**Figure no. 1. Internal carotid artery aneurysm. On visual field assessment, there is a left homonymous hemianopia which is congruous**

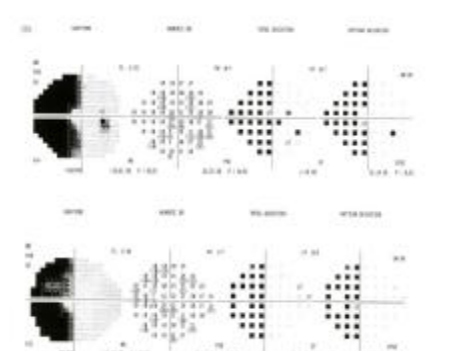


Homonymous hemianopic field defects are said to be congruous when the defect is not complete (i.e. does not occupy the entire half of the field) and the defect extends to the same angular meridian in both eyes. Optic tract lesions tend to produce markedly incongruous field defects. The more congruous a homonymous hemianopia, the nearer the lesion will be to the occipital cortex (ie, more posterior in the visual pathways).

### INCONGRUOUS VISUAL FIELD LOSS

The hemianopia is incongruous when the deficit of those both fields does not exactly correspond. Marked incongruity indicates an optic tract lesion, since lesions of the optic radiations tend to cause only mild incongruity and visual cortex lesions are highly congruous. While visual field defects related to optic radiation or striate visual cortex lesions result from vascular disease and other intracerebral pathology, most optic tract lesions are compressive extrinsic masses.

**Figure no. 2. Craniopharyngioma. On visual field assessment, there is an incongruous left homonymous hemianopia**



### TOPICAL DIAGNOSIS OF LESIONS OF THE LATERAL GENICULATE BODY

Lesions affecting the lateral geniculate body are less

commonly diagnosed than those of the optic tract but they can be caused by a number of different processes, including vascular disease, neoplasms, inflammation, demyelination and trauma.

Lesions of the lateral geniculate body may cause incongruous or congruous homonymous field defects.

Ischemia or other damage in the territory of the lateral choroidal artery typically causes a congruous homonymous horizontal sectoranopia. Ischemia in the region of the lateral geniculate body supplied by the distal portion of the anterior choroidal artery results in loss of the upper and lower homonymous sectors in the visual fields of the two eyes, producing a congruous homonymous quadruple sectoranopia.

The pupillary reactions in patients with lateral geniculate body lesions are normal.

Patients with lesions of the lateral geniculate body frequently have neurologic symptoms and signs consistent with damage to the ipsilateral thalamus or pyramidal tract.

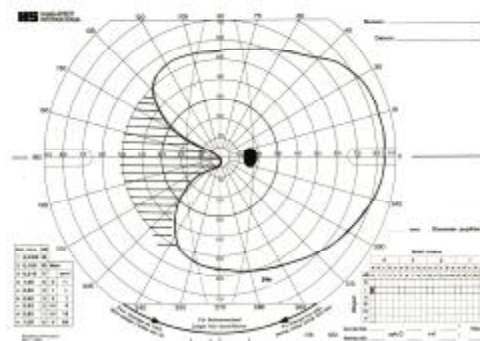
#### Lateral geniculate nucleus field defect:

a. Visual information from ipsilateral eye synapses in layers 2, 3, 5; from contralateral in layers 1, 2, 6. Macular vision is subserved by the hilum and peripheral field by the medial and lateral horns.

b. Types of defects:

- Incongruous homonymous hemianopia.
- Unique sector and sector-sparing defects due to dual blood supply of lateral geniculate nucleus from anterior and posterior choroidal arteries.

**Figure no. 3. Goldmann perimeter visual field assessment: sector defect. There is an incongruous homonymous defect extending to central fixation**



A number of varying types of visual field defects may occur with lesions of the lateral geniculate body, dependent on the extent and nature of the lesion and the area of lateral geniculate body involved.

The hemianopias through lesions of the lateral geniculate body are difficult to be diagnosed, with the exception of those cases where we associate signs of thalamus lesion (thalamus pains, problems of objective sensitivity in half body corresponding hemianopia). The lesions involving the medial part of the right geniculate body will determine an inferior left quadrantanopia, while the lesions of lateral part of right geniculate produce a superior left quadrantanopia.

The superior hemianopias can be determined by symmetrical lesions of external geniculate bodies too.

Sectoral hemianopias may also occur which reflect the vascular supply to the lateral geniculate body (division of anterior and lateral posterior choroidal arteries) (Shacklett et al.1984).

Visual field defects will always involve central fixation due to the representation of the central retinal nerve fibres in all six layers of the lateral geniculate body, whereas monocular and binocular peripheral nerve fibres are distributed

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in varying layers of the lateral geniculate body.

### TOPICAL DIAGNOSIS OF THE LESIONS OF THE OPTIC RADIATION

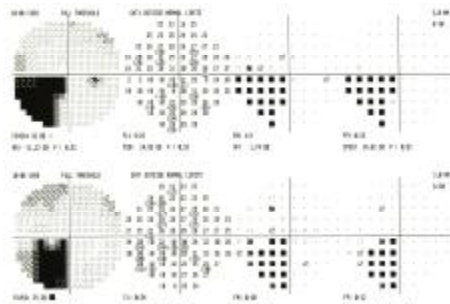
The optic radiation is that part of the postchiasmal visual sensory pathway that begins in the lateral geniculate body and transmits visual information to the striate cortex. It may be damaged by lesions in several different locations, including the internal capsule, temporal lobe and parietal lobe.

#### LESIONS OF THE INTERNAL CAPSULE

Efferent projection fibers from and afferent projection fibers to the cerebral cortex traverse the subcortical white matter where they form a radiating mass of fibers, the corona radiata, which converges toward the brainstem. In the rostral brainstem, the fibers form a broad but compact fiber band, the internal capsule, that is bordered medially by the thalamus and caudate nucleus and laterally by the lenticular nucleus.

Interruption of the optic radiation is characterized by a contralateral, usually complete, homonymous hemianopia that is typically associated with contralateral hemianesthesia from damage to the adjacent thalamocortical fibers in the posterior limb of the internal capsule. Other ocular findings in lesions of the internal capsule include a transient deviation of the eyes to the side of the lesion in many instances and weakness of the frontalis and orbicularis oculi on the contralateral hemiplegic side in a minority of cases. Vascular causes predominate.

**Figure no. 4. Humphrey perimeter visual field assessment: space-occupying lesion. A right sided tumour has produced a left incongruous inferior quadrantanopia which does not align on the horizontal meridia.**



The lesions of the internal capsule affect the optic radiations while they still are compact bundles, determining a hemianopia with variable extension towards horizontal meridia and central vision.

Homonymous hemianopia without the involvement of macula is present in lesions of optic radiation at half way calcarine fissure or anterior to this.

Hemianopia is complete when the interested hemifields are ruined on the whole part involved, including the macula area. It is found in the posterior lesions of the optic radiations (towards occipital pole).

Double homonymous hemianopia, taking into consideration the macular field creating a reduced visual field at 5 degrees around the macula-“in telescope” vision-is found in extensive lesions of both optic radiations.

The homonymous quadrantanopias are well marked lesions of some fascicles. They are found in temporal and parietal tumours. It is generally caused by Meinert’s knee lesion.

The deficit of visual field in superior left quadrant is given by a lesion in the inferior fascicle of the right optic radiations.

The deficit of visual field in crescent appears due to lesions involving the internal and anterior optic radiations. The temporal crescent fascicle is involved.

The hemianopias due to the lesions of optic radiations

are usually incongruous, with the exception of those cases where lesion of radiation is complete. The hemianopic deficit does not involve the macula vision.

The anterior choroidian artery irrigates the optic radiations while they cross through retrolenticular segment of internal capsule. The middle cerebral artery irrigates by means of its posterior branches the most of optic radiations.

#### LESIONS OF THE TEMPORAL LOBE

Temporal lobe lesions may damage the optic radiation. Such lesions account for about 13% to 24% of homonymous visual field defects, with tumours and abscesses causing the majority of cases. Temporal lobe surgery for epilepsy may also cause such defects, which are often asymptomatic.

Superior-inferior separation in the temporal lobe:

1. Inferior fibres (ipsilateral inferotemporal fibres and contralateral inferonazal fibres) course anteriorly from the lateral geniculate body into the temporal lobe, forming Meyer’s loop approximately 2,5cm (range 2,4 to 2,8cm) from the anterior tip of the temporal lobe.
2. Inferior „macular” fibres do not cross as far anteriorly in the temporal lobe.
3. Anterior temporal lobe lesions tend to produce midperipheral and peripheral contralateral homonymous superior quadrantanopia.
4. More extensive temporal lobe lesions may cause field defects that extend to the inferior quadrants, but hemianopia will be „denser” superiorly.

A superior homonymous quadratic defect in the visual fields suggests involvement of either the inferior visual cortex or the inferior optic radiations, and such involvement of the optic radiations may occur in the temporal lobe within Meyer’s loop.

#### LESIONS OF THE PARIETAL LOBE

Lesions of the parietal lobe may produce ocular symptoms that have value in topical diagnosis. Homonymous hemianopia affecting primarily the lower fields is caused by damage to the optic radiation in the superior parietal lobe. Such defects are usually more congruous than those produced by lesions of the temporal lobe. Because the entire optic radiation passes through the parietal lobe, large lesions may produce complete homonymous hemianopia with macular splitting.

Neuro-ophthalmologic features suggesting a lesion in the parietal lobe include an incomplete and relatively congruous (or mildly incongruous) homonymous hemianopia that is denser below than above, conjugate movements of the eyes to the side opposite the lesion on forced lid closure (Cogan’s sign), and an abnormal optokinetic response when the target is moved toward the side of the lesion. A disturbance of fixation reflexes sufficient to interfere with reading ability may develop before the appearance of other symptoms. Other types of visual disturbances caused by lesions in the parietal lobe include visual neglect, visual agnosia, and difficulties with word recognition.

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