

## Hemispatial visual inattention masquerading as hemianopia

**Article abstract**—The defect occurring when a patient fails to report a visual stimulus presented in a visual half-field may be attributed to hemianopia (deafferentation) caused by a geniculocalcarine lesion. However, failure to report a stimulus presented in a visual field may also be caused by hemispatial visual inattention. We report a patient with right thalamic and temporo-occipital lesions who had a left visual field defect when her eyes were directed either straight ahead (midsagittal plane) or toward left hemispace. However, this visual field defect abated when her eyes were directed to right hemispace, suggesting that the patient had hemispatial visual inattention rather than hemianopia.

NEUROLOGY 1989;39:1125-1127

C.A. Kooistra, MD, and K.M. Heilman, MD

When visual fields are tested by confrontation, the subject usually fixates in the midsagittal plane (straight ahead). Failure to report stimuli contralateral to a cerebral lesion is usually attributed to a hemianopia (deafferentation) caused by either a lesion in the visual pathways or in the calcarine cortex. However, stimuli that are not attended to also may not be reported.<sup>1</sup> Thus, the standard confrontation technique cannot differentiate hemianopia and attentional disorders.

Heilman and Valenstein<sup>2</sup> proposed that attentional fields may be different from sensory fields. Whereas the visual half-field is hemiretinal, the attentional half-field may be hemispatial. The term "hemispatial field" refers to the corporeal and extracorporeal space to the right or left of a midsagittal plane through the head and body. When the confrontation technique is used with the eyes fixed at the midsagittal plane, the visual half-fields and the hemispatial fields are aligned. However, if a subject deviates the eyes fully toward one hemispace, then both visual half-fields may fall entirely in one hemispace.

Bowers et al<sup>3</sup> used a reaction time paradigm in normal subjects to demonstrate a hemispace-visual half-field compatibility, providing evidence for a relationship between each cerebral hemisphere and the contralateral hemispace (independent of visual half-field). Although they posited that this relationship was attentional, its nature could not be ascertained.

We report a patient who, on confrontation testing, appeared to have a left hemianopia. However, when the patient moved her eyes toward right hemispace the visual defect improved, suggesting the defect was caused by hemispatial inattention rather than deafferentation.

**Case report.** An 81-year-old woman had the abrupt onset of left hemiparesis while hospitalized for pneumonia, and was seen in neurologic consultation. She appeared awake, alert, and attentive. She had left hemispatial neglect by line cancellation and line bisection tasks that failed to significantly improve with placement of the paper in right hemispace. She additionally demonstrated astereognosis and agraphesthesia in her left hand. She appeared to have a left visual field deficit and hypometric saccades to the left. Strength was normal throughout. Sensation to touch and pinprick were normal on the left with no extinction on double simultaneous tactile

stimulation. Proprioception in the left hand and leg was impaired. Deep tendon reflexes were increased on the left with an extensor plantar response. Her EEG revealed right posterior slowing. CT demonstrated a right lateral thalamic infarct with minimal involvement of the medial posterior limb of the internal capsule and an infarct of the right medial temporo-occipital region (figure).

**Additional visual testing.** Additional confrontational visual field testing was performed at the time of initial consultation. The patient's eyes were placed in primary position, 30° right gaze, or 30° left gaze while maintaining head position aligned with the body. Eccentric eye position was achieved by having the patient gaze horizontally such that the medial border of the adducting iris was aligned with the punctum of the lacrimal sac. Fixation was then maintained on the examiner's nose with the examiner sitting directly in the line of gaze at a distance of 3 feet. A visual stimulus was presented to either the patient's right or left visual field at eye level and consisted of the examiner raising 1 forefinger for 1 second. Stimuli were placed 12 inches lateral to the examiner's midline such that left visual field testing on right gaze fell into right hemispace and right visual field testing on left gaze fell into left hemispace. The patient was allowed 5 seconds to acknowledge the stimulus by saying "one."

Practice trials prior to testing included 10 stimuli (5 per visual field) in each of the 3 gaze positions. Testing then consisted of 15 blocks of 10 randomized visual stimuli (5 per visual field). The 15 blocks were randomly and equally distributed across the 3 gaze positions. Responses were scored as

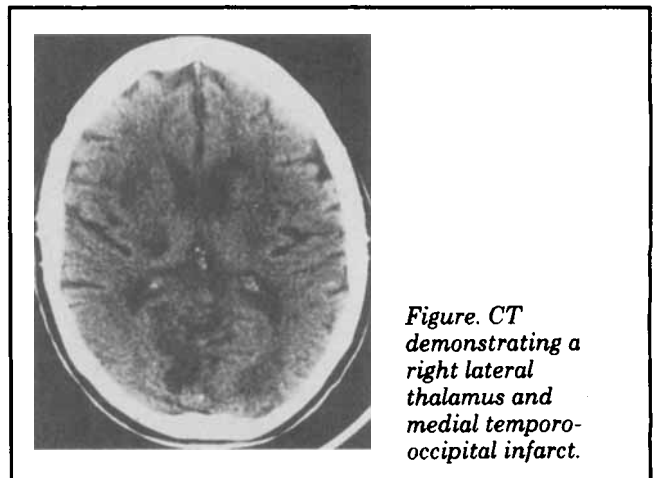


Figure. CT demonstrating a right lateral thalamus and medial temporo-occipital infarct.

**Table. Error rate**

<u>Left gaze</u>		<u>Primary position</u>		<u>Right gaze</u>	
LVF	RVF	LVF	RVF	LVF	RVF
5/5	0/5	4/5	0/5	2/5	0/5
2/5	0/5	4/5	0/5	1/5	0/5
5/5	1/5	4/5	0/5	2/5	0/5
4/5	0/5	4/5	0/5	0/5	0/5
2/5	0/5	3/5	0/5	1/5	0/5
Total	18/25	1/25	19/25	0/25	6/25

LVF Left visual field.  
RVF Right visual field.

correct/incorrect and an error rate was calculated for both visual fields for each block. Student's *t*-test was then used to compare error rates across conditions.

**Results.** The error rates (table) demonstrated a large number of errors when the left visual field was aligned with left hemisphere (left gaze or primary position). However, the error rate for left visual field in right hemisphere (right gaze) was significantly improved when compared to either left ( $p < 0.01$ ) or primary ( $p < 0.01$ ) positions. There was no significant change in the error rate for right visual field when moved from right hemisphere (primary and right gaze) to left hemisphere (left gaze).

Follow-up examination 2 months later showed resolution of the hemispatial neglect, visual field defect, agraphesthesia, and astereognosis. There was no evidence of extinction on double simultaneous stimulation in any modality. Moderate proprioception deficits persisted in the left lower extremity. The patient's chief complaint concerned her inability to perceive writing in her left half-field unless she stared at a printed page for several seconds, after which the letters would gradually appear.

**Discussion.** Because the same stimuli presented to the same visual half-field were better detected when the eyes were directed to right hemisphere than when directed to left hemisphere, the defective performance in the midsagittal plane cannot be attributed entirely to a hemianopia and must, at least in part, be due to hemispatial inattention.

These results have both pragmatic and theoretical implications. In regard to the former, testing visual fields in both right and left hemisphere may help differentiate visual field defects from attentional defects. In regard to the latter, these results may help us understand theoretical aspects of neglect. When a patient with a hemisphere lesion who is not hemianopic fails to cancel stimuli on one side of a page or to bisect a line (spatial neglect), the defect may be attributed to hemispatial inattention.<sup>4</sup> Because spatial neglect may be demonstrated using even nonvisual tests,<sup>4</sup> hemispatial neglect is not synonymous with hemispatial visual inattention. Other neuropsychological mechanisms, such as directional akinesia and exploratory, representational, and memory defects, may also account for defective

performance on these spatial tasks.<sup>4</sup> Although directional akinesia and exploratory, representational, or memory defects can explain hemispatial neglect, they cannot explain the results reported here. The results reported in this patient are best explained by hemispatial inattention. The presence of hemispatial inattention provides support for the postulate that each hemisphere is responsible for attending to contralateral hemisphere. Whereas this patient's inattention appeared to be hemispatial, it was also partially hemiretinal. If it were entirely hemispatial, one would also expect a partial right visual field defect with left gaze, and even though the patient made an error in her right visual field on left gaze, this difference was trivial.

Although the patient showed spatial neglect on the line bisection task, she failed to improve when the line was placed in the right hemisphere (unlike stimulus detection, which improved on right gaze). The discrepancy between performance on line bisection and detection tasks provides evidence that this patient's hemispatial inattention could not entirely account for her spatial neglect and that the spatial neglect was probably related to either a directional akinesia or an exploratory or memory defect.

Posner et al<sup>5</sup> demonstrated, in normal subjects, that cues as to a target's ultimate appearance in space improved the speed at which a target was recognized. He proposed that these spatial cues enabled subjects to covertly orient their attention to the target. When Posner et al<sup>5</sup> tested patients with unilateral parietal lesions, they were able to covertly direct their attention to correct cues both ipsilaterally and contralaterally. However, if the patients received an incorrect cue to direct their attention ipsilaterally (the target being contralateral), they had prolonged reaction times. Posner et al<sup>5</sup> postulated 3 stages of directed attention: disengagement from the current focus of attention, movement of attention to a new target, and engaging the target. Posner et al<sup>5</sup> proposed that their subjects had prolonged reaction times only after being miscued because they had difficulty disengaging their attention from the ipsilateral stimuli. In a subsequent study, Rafel and Posner<sup>6</sup> demonstrated that thalamic lesions not only affected the disengage operation, but also slowed engagement of stimuli in the contralateral visual field. Attention in these studies<sup>5,6</sup> was assessed by facilitation of a motor response. However, unlike the subjects of Rafel and Posner,<sup>6</sup> rather than having slow engagement, our subject did not engage stimuli in the contralateral visual field when they were in contralateral hemisphere. Attentional deficits therefore have at least 2 independent manifestations. There can be slowing of engagement, as reported by Rafel and Posner,<sup>6</sup> or a failure of recognition or report, as demonstrated by this case.

It is unknown whether this patient's failure to report left hemifield stimuli when looking to the left but not to the right was due to her temporo-occipital or thalamic lesion. Further studies with patients who have isolated thalamic, parietal, and temporo-occipital lesions will have to be performed to learn which area or areas, when damaged, can be associated with the spatially specific recognition defect we report.

## Acknowledgments

Our thanks to Robert Mitchell, MD, Neil Parnes, MD, and the Spartanburg Regional Medical Center for their assistance in obtaining the CT scan.

---

From the Veterans Administration Medical Center and the Department of Neurology, University of Florida College of Medicine Gainesville, FL.

Supported by the Medical Research Service of the Veterans Administration.

Received January 5, 1989. Accepted for publication in final form February 13, 1989.

Address correspondence to Dr. Heilman, Box J-236, Department of Neurology, College of Medicine, University of Florida, Gainesville, FL 32610.

---

## Lack of cerebrospinal fluid myelin basic protein in HIV-infected asymptomatic individuals with intrathecal synthesis of IgG

**Article abstract**—We evaluated 130 consecutive HIV-infected neurologically asymptomatic individuals for intrathecal IgG production and myelin basic protein (MBP) levels. Although 56.7% of immunologically normal and 68.8% of immunocompromised patients had some CSF abnormality, no patient had an abnormal MBP level. The lack of MBP elevation in the CSF of these patients suggests that the production of intrathecal IgG is not related to active demyelination.

NEUROLOGY 1989;39:1127-1129

Douglas W. Marshall, MD; Robin L. Brey, MD; and Clifford A. Butzin, PhD

**Immunologic abnormalities** in CSF as measured by IgG synthesis rate, IgG index, or oligoclonal bands occur in various disorders including human immunodeficiency virus (HIV) infection.<sup>1,2</sup> CSF myelin basic protein (MBP) is another study often included in laboratory batteries assessing demyelinating disorders and thought to be an indicator of active myelin injury.<sup>3-5</sup> The neuropathology of AIDS dementia complex (ADC) consists of white matter abnormalities with ill-defined large areas of pallor not due solely to selective demyelination.<sup>6</sup> Since patients in all stages of HIV infection commonly have abnormal CSF immunologic findings such as oligoclonal bands and elevated IgG synthesis rate or IgG index,<sup>1</sup> we determined whether these indicators of intrathecal IgG production correlated with acute demyelination as indicated by elevated CSF MBP.

**Patients and methods.** *Patient population.* All United States Air Force (USAF) personnel are screened for serologic evidence of antibody against HIV. Those who test positive are referred to Wilford Hall USAF Medical Center (WHMC) at Lackland Air Force Base, Texas, for an initial evaluation, annual reevaluation, and reevaluation upon the occurrence of HIV-related symptoms.<sup>1</sup> All patients are staged according to the Walter Reed (WR) system.<sup>7</sup> Patients classified as WR stages 1 and 2 are those who have T-helper (CD4) cell counts greater than 400, do not have skin anergy, and may or may not have lymphadenopathy. Patients classified as WR stages 3, 4, 5, and 6 all have CD4 counts of less than 400. Stages 5 and 6 correspond to AIDS-related complex (ARC) and AIDS

## References

1. Norman DA. Memory and attention: an introduction to human information processing. New York: John Wiley, 1969.
2. Heilman KM, Valenstein E. Mechanisms underlying hemispatial neglect. *Ann Neurol* 1979;5:166-170.
3. Bowers D, Heilman KM, Van Den Abell T. Hemispace-visual half field compatibility. *Neuropsychologia* 1981;19:757-765.
4. Heilman KM, Watson RT, Valenstein E. Neglect and related disorders. In: Heilman KM, Valenstein E, eds. *Clinical neuropsychology*. New York: Oxford, 1985:243-293.
5. Posner MI, Walker JA, Friedrich FJ, Patel R. Effects of parietal injury on covert orienting of attention. *J Neurosci* 1984;4:1863-1874.
6. Rafel RD, Posner MI. Deficits in human visual spatial attention following thalamic lesions. *Proc Natl Acad Sci USA* 1987;84:7349-7353.

groups, respectively. A cohort of 130 consecutive HIV-infected individuals who were evaluated at WHMC had CSF MBP assayed regardless of clinical stage. These patients are a subset of those reported previously.<sup>1</sup> The initial WR stages of these individuals were as follows: 107 WR1, 7 WR2, 6 WR3, 3 WR4, 7 WR5, and 0 WR6.

*Methods.* CSF studies, as described elsewhere,<sup>1</sup> included protein, nucleated cells, CSF IgG, IgG index, IgG synthesis rate, oligoclonal bands, albumin index, VDRL, cryptococcal antigen, and fungal and mycobacterial cultures. Abnormal study findings were defined as protein greater than 45 mg/dl, nucleated cells greater than 5 per  $\mu$ l, IgG greater than 6.0 mg/dl, IgG index greater than 0.66, IgG synthesis rate greater than 3.3 mg/day, and 2 or more oligoclonal bands present in CSF but not serum (by agarose gel electrophoresis). All patients had a standardized neurologic examination in addition to the CSF studies, and 65% (84/130) were similarly reevaluated after 1 year. All patients had CSF from their initial evaluations sent to Smith-Kline Bioscience Reference Laboratory for MBP determination. This laboratory uses a double-antibody radioimmuno assay<sup>3</sup> in which MBP of 4 to 8 ng/ml indicates a slow rate of myelin degradation, and greater than 8 ng/ml suggests rapid myelin breakdown. Repeat CSF MBP studies are planned on all those who develop CNS complications.

**Results.** No patient had CNS abnormalities on the first clinical evaluation. The initial CSF findings in this cohort is similar to those of the aggregate data previously reported<sup>1</sup> on a larger group of HIV-infected patients, of which they are a subset. Of the 114 patients classified as WR1 or 2, 56.7% had at least 1 abnormal CSF study and 21.1% had 3 or more abnormal. Of the 16

# Neurology<sup>®</sup>

## Hemispatial visual inattention masquerading as hemianopia

C. A. Kooistra and K. M. Heilman

*Neurology* 1989;39;1125

DOI 10.1212/WNL.39.8.1125

**This information is current as of August 1, 1989**

<b>Updated Information &amp; Services</b>	including high resolution figures, can be found at: <a href="http://n.neurology.org/content/39/8/1125.full.html">http://n.neurology.org/content/39/8/1125.full.html</a>
<b>Citations</b>	This article has been cited by 5 HighWire-hosted articles: <a href="http://n.neurology.org/content/39/8/1125.full.html##otherarticles">http://n.neurology.org/content/39/8/1125.full.html##otherarticles</a>
<b>Permissions &amp; Licensing</b>	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: <a href="http://n.neurology.org/misc/about.xhtml#permissions">http://n.neurology.org/misc/about.xhtml#permissions</a>
<b>Reprints</b>	Information about ordering reprints can be found online: <a href="http://n.neurology.org/misc/addir.xhtml#reprintsus">http://n.neurology.org/misc/addir.xhtml#reprintsus</a>

*Neurology*® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright © 1989 by AAN Enterprises, Inc.. All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.

