

Luc Crevits
Karel Deblaere
Isabelle Aers

Visual extinction for motion

Received: 26 June 2003
Revised: 25 November 2003
Accepted: 26 November 2003
Published online: 14 January 2004
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L. Crevits (✉)
Oto-neuro-ophthalmology Unit,
Department of Neurology,
Ghent University Hospital,
De Pintelaan 185, 9000 Ghent, Belgium
e-mail: luc.crevits@ugent.be
Tel.: +32-9-2404539
Fax: +32-9-2404971

K. Deblaere
fMRI Workgroup,
Department of Radiology,
Ghent University Hospital,
Ghent, Belgium

I. Aers
Department of Neurology,
General Hospital
Maria Middelaers–Sint Jozef,
Ghent, Belgium

Abstract *Purpose:* To alert clinicians to the heretofore undescribed visual behavioral phenomenon of visual extinction limited to motion. *Methods:* Neuro-ophthalmological, neuropsychological and neuroimaging assessment of a 57-year-old man with vague visual complaints. *Results:* Extinction limited to visual motion perception in the left hemifield was demonstrated. The visual defect was attributed to a lesion in-

volving the right occipito-temporo-parietal region in the presence of a left posterior infarction. *Conclusion:* The importance of clinical examination for detection of this specific higher-order visual defect is stressed. The present observation may be helpful in understanding the effects of attention on visual perception and may have important implications for rehabilitation of patients with visual neglect.

Introduction

Hemispatial neglect can involve multiple sensory modalities, visual extinction often being a prominent feature. Affected patients perceive visual stimuli presented separately in either their right or left hemifield, but ignore stimuli in the left hemifield when both hemifields are stimulated simultaneously [7]. This phenomenon is classically attributed to a lesion in the right parietal lobe. Inferior regions of the parietal lobe are an important component of the dorsal occipitofugal visual pathway [13].

A defect in the visual perception of motion with preservation of perception of other modalities of vision is known as akinetopsia (for review see [16]). A deficit

in motion processing in the contralateral hemifield in patients with unilateral occipitoparietal lesions has also been described (hemi-akinetopsia) [14]. The middle temporal area (MT) in the monkey is particularly important for motion perception. The human homologue of MT (V5) is thought to be situated at the temporo-occipital confluence and the junction of Brodmann areas (BA) 19 and 37 inferiorly [15].

We report the unique case of a patient with visual extinction for motion in the left hemifield. So far, visual extinction limited to motion has not been described. This clinical observation may be helpful to understand the effects of attention on visual perception and may have important implications for rehabilitation of patients with spatial neglect.

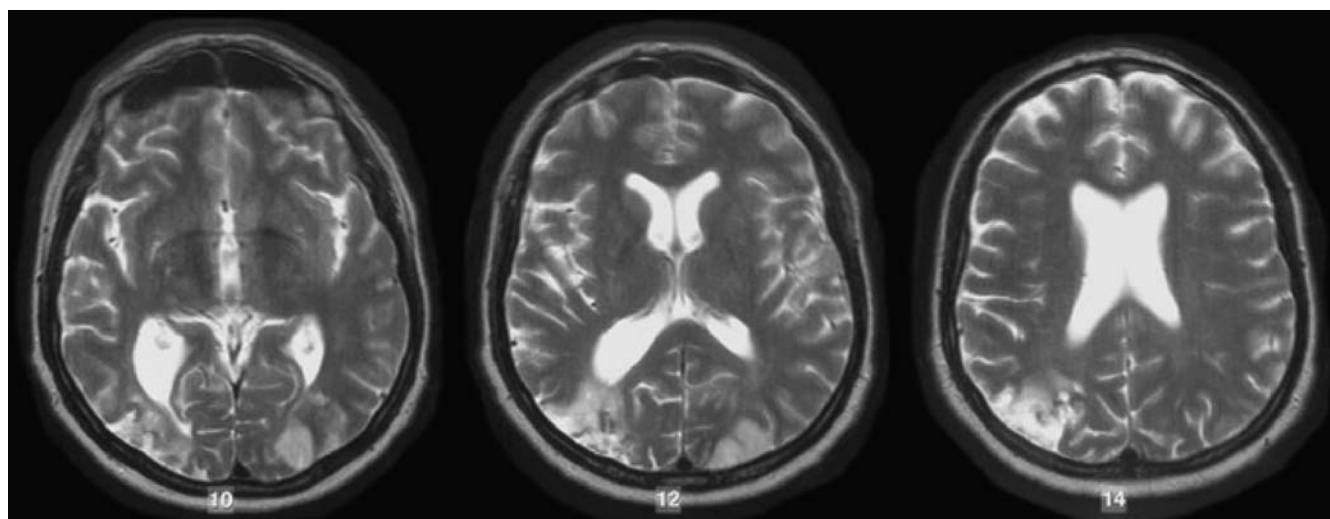


Fig 1 T₂-weighted axial MRI showing increased signal in the left occipital and right temporo-occipital regions (left and right according to radiological convention)

Case report

Clinical presentation

A 57-year-old right-handed man was admitted because of acute onset of right sided weakness. Apart from a mild right hemiparesis, a right homonymous hemianopia was found on visual confrontation testing. Cranial CT scan showed a recent infarction in the left occipital region and an old parieto-occipital watershed infarction on the right.

The patient also reported an episode from 1 year earlier during which he had experienced a temporary visual problem “on the left”. This had apparently returned rapidly to normal and he remained functionally normal until the present admission.

Apart from nicotine abuse, no specific vascular risk factors were detected on general and cardiovascular investigations.

During the days following admission, the right hemisindrome, including hemianopia, resolved. The patient, however, had persistent vague visual complaints, especially in a moving environment. He could not further specify his problems, despite his profession as a medical doctor.

Neuro-ophthalmology

Visual acuity was 1.0 in both eyes. Eye pressure was normal. Automated perimetry could not detect any hemianopic defect. Fundoscopy showed atherosclerotic signs. Stereopsis, as checked with the Lang test, was normal. Eye movements were normal. Pupils were isocoric and normally reactive.

Visual fields were examined monocularly by a test in which the clinician's outstretched finger is shown in different areas of the visual field while the patient fixates the heteronymous eye of the clinician. The results of this so-called confrontation test were normal. To test for visual neglect, the right index finger of the clinician was shown in the patient's left upper quadrant, simultaneously with the left finger in the patient's right upper quadrant. Thereafter, the same procedure was done for the lower quadrants. The patient could readily detect and count the stationary fingers in all four quadrants. On trials for motion detection, we wiggled the

right and left index fingers simultaneously, four times, first in both upper and then in both lower lateral visual quadrants. The patient perceived the right index finger as wiggling, but did not perceive any movement of the left index finger. This extinction phenomenon was present in the left upper and in the left lower quadrant of either eye. It was tested repeatedly and could be reproduced each time. However, when each index finger was wiggled separately, the patient could see the moving finger in the left as well as in the right hemifield.

Tactile extinction was tested in a standard bedside evaluation with a light touch simultaneously on both upper and then on both lower limbs. No deficit could be detected.

Neurophysiology

Eye movements were registered by electro-oculography. Saccades on command were of normal metricity and velocity. Horizontal pursuit was elicited by a swinging pendulum. The initial excursion and peak velocity were 40° (20° about zero) and 50°/s respectively. The oscillations progressively damped to zero. Pursuit was smooth in both horizontal directions throughout the swinging cycles.

Neuropsychology

Neuropsychological examination with classical tests for frontal executive functions, language and memory was normal except for some difficulties for visual memory (Rey visual design learning test). Copying of a complex three-dimensional figure revealed mild constructive apraxia. Several commonly used tests for visuospatial functions including line bisection, line cancellation, identifying bells among simple figures, reading individual words and sentences, drawing of a clock, copying of a scene, visuospatial judgment (VSJ), the Hooper visual organization test and the Money road map test (MRMT) showed normal results.

Neuroimaging

Magnetic resonance imaging (MRI) of the head showed ischemic lesions in both posterior regions, more extensive on the right (Figs. 1 and 2). According to the classical atlas of Talairach and Tournoux [12], the right occipito-temporo-parietal lesion involved BA 18–19, 39–40, the gyrus temporalis superior, the gyrus supramarginalis and the inferior parietal lobule. On the left, only BA 18 was infarcted.

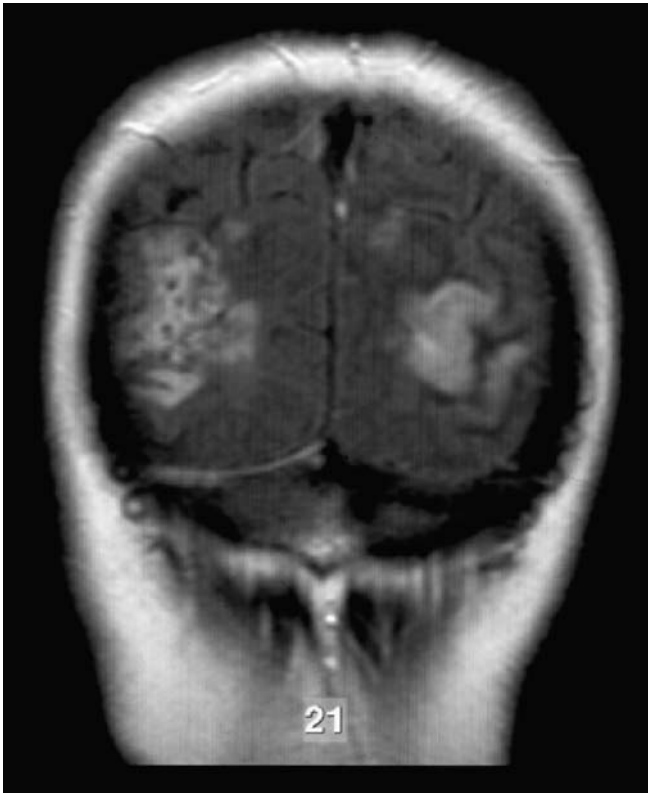


Fig 2 Fluid-attenuated inversion recovery (FLAIR) coronal MRI showing the extension of the lesion to the right inferior parietal lobule (left and right according to radiological convention)

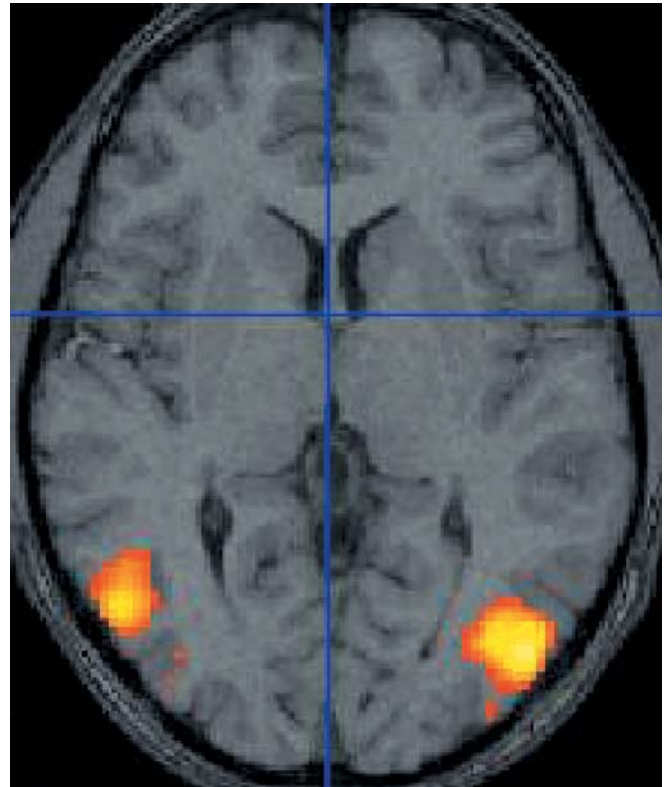


Fig 3 Results of a functional MRI experiment. The image shows activation in MT/V5+ during an experiment in which the healthy subject looks at his or her own fingers while performing a finger opposition task compared with a baseline task of looking at the same fingers during rest

Discussion

The patient was admitted because of a left posterior artery cerebral infarction. After recovery of the right homonymous hemianopia, his residual visual complaints could be attributed to visual extinction for motion in the left hemifield. It is to be noted that this kind of visual disturbance can not be detected by perimetry, stressing the importance of clinical testing. Moreover, the classical confrontation test should be expanded by double stimulation (i.e., simultaneous in the nasal and temporal hemifields) for stationary as well as for moving stimuli.

Figure 3 shows the location of area V5 as revealed with functional MRI in an experiment in which the healthy subject looks at his or her own fingers performing a finger opposition task, compared with a baseline task of looking at the same fingers at rest. It seems that the right area V5 was involved in our patient. However, hemi-akinetopsia could not be demonstrated. This can be explained by the fact that the analysis of motion involves a complex system of several interrelated cortical areas that are involved in processing various components of motion perception [2] and that may adapt to the loss of area

V5. This would explain the preservation of some aspects of visual motion perception in patients with akinetopsia and the rarity of the syndrome. Moreover, it seems likely that hemi-akinetopsia is often obscured by coexistent incomplete field defects. The rather crude clinical methods used to detect akinetopsia may also account for non-detection.

Parietal lesions often result in spatial neglect of the contralesional hemifield. The prevalence of left neglect implies a dominant role for the right hemisphere in spatial attentional processing. Only left hemispatial neglect is seen as a persistent phenomenon. Neglect is a multifaceted disorder, manifesting itself within different processing domains and reference frames [8]. Stimuli presented in isolation to the left are often still normally perceived. However, when two objects are presented simultaneously to the left and the right, the object on the left may be 'ignored' or 'extinguished'.

Using a rather sophisticated procedure, Rorden et al. [11] explained visual extinction after unilateral parietal damage as impaired temporal order with intact motion perception. Although we used simple clinical methods, our results rule out (hemi-)akinetopsia, as preserved per-

formance for motion was demonstrated in both hemifields. Given that the patient could detect unilateral movement, there is no indication of a sensory dysfunction in motion perception. On double stimulation, however, impaired motion perception in the left hemifield was clearly shown. The present case with dissociated visual neglect (present for moving stimuli and not for the same stationary stimuli) suggests that extinction is not a sensory problem but rather a 'higher-order' disorder. It also provides evidence that visual extinction can be limited to specific modalities. It can not be excluded, however, that visual extinction for stationary objects might well have been demonstrated in our case if less salient stimuli had been used.

The present observation illustrates that patients can have normal sinusoidal smooth pursuit eye movements but visual extinction for motion perception as is known in the case of impaired motion perception as such [1]. The preserved smooth pursuit may be an argument suggesting that the posterior parietal cortex is not the typical site in patients with visual extinction. This observation corresponds with recent studies of spatial awareness in humans that implicate the superior temporal gyrus [3, 4].

The patient was able to attentionally process visual motion information, as he could describe movement when it was presented unilaterally in the left or right visual hemifield. However, movement was ignored or neglected on the left when presented bilaterally. This observation is in line with actual localization of spatial awareness at the transition zone between the two major pathways of cortical visual processing [3] and with the view of hemispatial neglect bridging the dorsal and ventral visual streams [9].

The patient had experienced an earlier episode of visual problems "on the left", probably as minor symptoms of an otherwise subclinical right hemispheric stroke. However, his persistent complaints followed left posterior

or infarction. This raises the question of the relation between symptoms and brain lesions. The phenomenon of ipsilesional neglect has been reported only rarely. Therefore, the anatomical features are not fully elucidated, although the right frontal-subcortical regions have been implicated [5]. These locations were not affected in our case. We assume that the older right occipito-temporo-parietal lesion was responsible for the visual extinction. Thus, the stroke in the left occipital region unmasked a visual higher-order defect that could be attributed to an earlier right occipito-temporo-parietal lesion.

The question of why the patient remained free of complaints in the time between the two strokes can not be answered with certainty. Karnath et al. [4] demonstrated an overlap between the neural network for stimulus-driven attention and that for visual extinction at the right temporo-parietal junction. Thus, our patient might have adopted a compensatory strategy for extinction. Further, it has been shown that extinguished left visual field stimulation still activates the right visual cortex, including striate and early extrastriate areas [10]. We postulate that the synchrony of neural firing in these intact areas bilaterally interacting with higher visual cortices, masked the extinction phenomenon. If that mechanism failed after left occipital infarction, the patient eventually became aware of the problem.

The present findings may be important in light of rehabilitation of patients with unilateral spatial neglect. Visual background motion has been suggested to reduce spatial neglect [6]. However, as extinction for motion may easily be overlooked, we strongly advise the testing of patients with a simple confrontation method extended for extinction for motion.

Finally, the present report reminds clinicians that vague and subjective visual complaints may be important for the assessment of unusual visual defects. Extinction is not evaluated by classical perimetry but can easily be assessed clinically.

References

1. Barton JJ, Sharpe JA, Raymond JE (1996) Directional defects in pursuit and motion perception in humans with unilateral cerebral lesions. *Brain* 119:1535–1550
2. Dupont P, Orban GA, De Bruyn B, Verbruggen A, Mortelmans L (1994) Many areas in the human brain respond to visual motion. *J Neurophysiol* 72:1420–1424
3. Karnath HO, Ferber S, Himmelbach M (2001) Spatial awareness is a function of the temporal not the posterior parietal lobe. *Nature* 411:950–953
4. Karnath HO, Himmelbach M, Küker W (2003) The cortical substrate of visual extinction. *NeuroReport* 14:437–442
5. Kim M, Na DL, Kim GM, Adair JC, Lee KH, Heilman KM. Ipsilesional neglect: behavioural and anatomical features. (1999) *J Neurol Neurosurg Psychiatry* 67:35–38
6. Mattingley JB, Bradshaw JL, Bradshaw JA (1994) Horizontal visual motion modulates focal attention in left unilateral spatial neglect. *J Neurol Neurosurg Psychiatry* 57:1228–1235
7. Mesulam MM (1990) Large-scale neurocognitive networks and distributed processing for attention, language, and memory. *Ann Neurol* 28:597–613
8. Mesulam MM (1999) Spatial attention and neglect: parietal, frontal and cingulate contributions to the mental representation and attentional targeting of salient extrapersonal events. *Philos Trans R Soc Lond B* 354:1325–1346
9. Milner AD (1998) Neuropsychological studies of perception and visuo-motor control. *Philos Trans R Soc Lond B* 353:1375–1384
10. Rees G, Wojciulik E, Clarke K, Husain M, Frith C, Driver J (2000) Unconscious activation of visual cortex in the damaged right hemisphere of a parietal patient with extinction. *Brain* 123:1624–1633

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11. Rorden C, Mattingley JB, Karnath HO, Driver J (1997) Visual extinction and prior entry: impaired perception of temporal order with intact motion perception after unilateral parietal damage. *Neuropsychologia* 35:421–433
 12. Talairach J, Tournoux P (1988) *Coplanar stereotaxic atlas of the human brain*. Thieme, Stuttgart
 13. Ungerleider LG, Mishkin M (1982) Two cortical visual systems. In: Ingle DJ, Mansfield RJW, Goodale MS (eds) *The analysis of visual behavior*. MIT Press, Cambridge, pp 549–586
 14. Vaina LM (1989) Selective impairment of visual motion interpretation following lesions of the right occipito-parietal area in humans. *Biol Cybern* 61:347–359
 15. Vaina LM, Makris N, Kennedy D, Cowey A (1998) The selective impairment of the perception of first-order motion by unilateral cortical brain damage. *Vis Neurosci* 15:333–48
 16. Zeki S (1991) Cerebral akinetopsia (visual motion blindness). A review. *Brain* 114:811–824