CASE REPORT

Invisibility of moving objects: a core symptom of motion blindness

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SUMMARY

Although the higher brain mechanisms of seeing moving objects have been deeply investigated, motion blindness remains a rare and enigmatic symptom. Very few case reports well describe the detailed symptoms and the lesions. We report a case of a patient who presented with invisibility of moving objects, that is, motion blindness resulting from a unilateral right hemispheric lesion. This man, in his 60s, suffered persistent motion blindness from a unilateral right temporoparietal subcortical haemorrhage. He could not see the moving objects, just felt the objects 'disappear' when they began moving. The symptom was observed not only in his daily life but also during investigations in the hospital. To the best of our knowledge, this is the first report of a patient who showed persistent motion blindness with a right unilateral lesion, and only the second one with clinically apparent motion blindness, whose symptoms were similar to those of the first reported case.

BACKGROUND

Motion blindness, which is also called akinetopsia, has only been described in a very few cases^{1–5} in which patients could not see the movement of objects.^{6–7} Most reports have indicated disability of the bilateral V5/medial temporal cortex (MT) complex, which contains direction-selective neurons and is called 'motion center'.⁸ We report of a patient who presented with invisibility of moving objects, a clear symptom of motion blindness resulting from a unilateral right temporoparietal subcortical haemorrhage. To the best of our knowledge, this is the first report of a patient who showed persistent motion blindness with a right unilateral lesion, and only the second one with clinically apparent motion blindness, whose symptoms were similar to those of the first reported case.

CASE PRESENTATION

A right-handed Japanese man in his 60s (left-handed in childhood) suffered left hemiparesis and numbness in the left side of the body while driving a car, and was admitted to the hospital.

The patient was well-oriented. His Edinburgh Inventory score was +1.0, which meant he was ambidextrous.⁹ He showed left homonymous hemianopia, left facial palsy, mild left hemiparesis and left hypoesthesia in touch, pain and temperature. In neuropsychological findings, he had left hemispatial neglect, dressing apraxia and anosognosia (did not recognise his hemiparesis and hemianopia, even after he had a minor car accident). He showed constructional disability when drawing a cube. He also

showed topographical disorientation; he could not describe the route from his house to the nearest railway station, nor show it on a map. Auditory sensation and colour vision were normal. He seemed to have mild simultagnosia in a verbal description task using the Cookie Theft picture, which is a component of the Boston Diagnostic Aphasia Examination: he explained only that there was a man eating a cookie, and a woman being angry. He did not have prosopagnosia or optic ataxia.

Severe hypoperfusion in his right cerebral hemisphere was suspected from the results of the positron emission tomography scan, because of which he underwent superficial temporal artery-middle cerebral artery bypass surgery according to Japanese Extracranial-intracranial bypass Trial (JET)-2 study protocol.¹⁰ Two days after surgery, he had a new subcortical haemorrhage in the right temporoparietal area, juxtaposed with the previous ischaemic lesion. The haematoma was surgically removed. He left the hospital a month after surgery without new neurological symptoms due to the brain haemorrhage.

After leaving the hospital, he discovered various symptoms he had never experienced before the bypass surgery; moving objects were invisible for him. For example, he could not see the landscape going by from the train window but could see the one when the train stopped. He bumped into a person walking towards him because he was invisible. He always failed to pour tea into a cup because the fluid vanished. When he played catch with his son, he could not see the ball which his son threw until it hit on his body. He always got lost in the supermarket because his wife 'disappeared' when he tried to follow her.

On neurological examination, his left hemiparesis, left facial palsy, left hypoesthesia and left hemispatial neglect were slightly improved compared with his preoperative status. The topographical disorientation, simultagnosia in the Cookie Theft picture task and constructional disability were unchanged. He recognised an object and caught it when the object remained still. However, when the object was moved from side to side or back and forth, he reported that he did not see it and he could not catch it.

INVESTIGATIONS**Examinations of visual motion discrimination**

1. Two-dimensional motion assessment: Motion perception was assessed using a coherent motion perception task previously described^{11–12}; in a normally lit room, 20 small randomly scattered

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dots were shown on a flat computer display. Some dots started to move coherently in the same direction while others moved in random directions. The patient was asked to determine the direction of the dot movement. The patient recognised the direction when 75% of the dots moved in the same direction (75% coherence motion: %CM) in parallel motion, which was far inferior to normal subjects' score of 13–14%CM. He also showed very low scores for radial motion (60%CM) and for rotation (80%CM).

2. Three-dimensional motion assessment: The patient could not grab a ball which the examiner threw him. He found the ball by the sound of it falling to the ground. When he was asked to follow the examiner walking in front of him, he was unable to do so.

Neuroradiological examinations

Before bypass surgery

A brain CT and MRI (figure 1A) showed a subcortical ischaemic lesion in the right temporal region. A cerebral MR angiography showed an occlusion of the right internal carotid artery.

After bypass surgery

His brain MRI 19 days after bypass surgery showed that the lesion extended in anterior and posterior directions relative to before surgery. However, the lesion remained limited to the right temporoparietal subcortex (figure 1B).

OUTCOME AND FOLLOW-UP

After about 5 months of his second cerebral haemorrhage, his symptoms remained, but gradually improved. After 7 months, however, his follow-up was lost because of his hospitalisation for the psoas abscess. After 3 years, he died of lung cancer. His relatives have not been contactable.

DISCUSSION

The first reported case by Zihl *et al*¹ showed two striking symptoms; invisibility of moving objects and motionless (like 'frozen') vision of moving objects. The detailed examinations revealed her difficulty in movement perception. It is curious that none of the other reported cases^{3–5} reported invisibility of moving objects; they only reported motion vision difficulty or seeing objects as 'frozen frames.'⁵ The sequential motionless vision of moving objects, so-called 'cinematographic vision' without invisibility of moving objects is also observed in migraine patients as a part of aura.¹³ Whether 'cinematographic vision' is a positive or negative symptom may be explained as a result of the motion perception impairment. Only the first reported case by Ziel *et al* and our patient reported invisibility of moving objects. Besides, only our patient did not show motionless vision. He told that the objects simply 'disappeared' when they began moving. Although understanding neural basis of motion vision has been advanced by extending research including sophisticated functional neuroimaging, the mechanisms of invisibility of moving objects remain unexplained.

Our patient had motion blindness in his bilateral visual field (his hemianopia was incomplete) with unilateral lesions in the right hemisphere. Previously, patients with motion blindness were reported to have bilateral MT/V5 lesion.^{1 6} Cooper *et al*² reported two interesting cases. One patient had transient akinetopsia in her bilateral visual fields caused from unilateral right hemispheric lesions. She realised that smooth movements of people were seen as a series of discontinuous 'freeze frames' (cinematographic vision), but did not disappear. The other patient with the left hemispheric lesion showed motion perception impairment only in his contralateral visual hemifield. Psychophysical,¹⁴ functional MRI,¹⁵ EEG¹⁶ and magnetoencephalogram¹⁷ studies have suggested differences in motion recognition between the left and right hemispheres. Vaina *et al*¹⁴ reported that the patients with unilateral lesions involving the

Figure 1 MRI of the brain: fluid-attenuated inversion recovery images showing (A) cerebral infarction in the right temporoparietal lesion before superficial temporal artery to middle cerebral artery bypass surgery. (B) Nineteen days after bypass surgery, the lesion extended in the anterior and posterior directions, but remained limited to the right temporoparietal subcortex.

anterior temporal or parietal lobes displayed poor motion perception in either visual field. Nakamura *et al* reported that hemispheric laterality was recognised in the visual motion perception task with normal volunteers, although the dominant side varied among participants.¹⁶

Our patient showed motion blindness after he got the second, expanded lesions in the right temporoparietal region, which may explain his impairment in the bilateral visual field.

Learning points

- Persistent motion blindness could occur from unilateral hemispheric lesions.
- The previous reported patients presented with two different types of difficulties in seeing moving objects: (1) not seeing objects when they are moving, (2) seeing objects as 'frozen frames' (cinematographic vision).
- Since our patient experienced only the first symptom but not the second one, these two symptoms might occur from different mechanisms.

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REFERENCES

- 1 Zihl J, von Cramon D, Mai N. Selective disturbance of movement vision after bilateral brain damage. *Brain* 1983;106(Pt 2):313–40.
- 2 Zihl J, von Cramon D, Mai N, *et al*. Disturbance of movement vision after bilateral posterior brain damage. Further evidence and follow up observations. *Brain* 1991;114(Pt 5):2235–52.
- 3 Blanke O, Vachlavik V, Landis T, *et al*. Dog phobia in a motion-blind patient. *Cogn Neuropsychiatry* 2003;8:211–21.
- 4 Tsai PH, Mendez MF. Akinetopsia in the posterior cortical variant of Alzheimer disease. *Neurology* 2009;73:731–2.
- 5 Cooper SA, Joshi AC, Seenan PJ, *et al*. Akinetopsia: acute presentation and evidence for persisting defects in motion vision. *J Neurol Neurosurg Psychiatry* 2012;83:229–30.
- 6 Zeki S. Cerebral akinetopsia (visual motion blindness). A review. *Brain* 1991;114(Pt 2):811–24.
- 7 Watson JD, Myers R, Frackowiak RS, *et al*. Area V5 of the human brain: evidence from a combined study using positron emission tomography and magnetic resonance imaging. *Cereb Cortex* 1993;3:79–94.
- 8 Farah MJ. *The cognitive neuroscience of vision*. Malden, MA: Blackwell Publishing Ltd 2000;45–50.
- 9 Oldfield RC. The assessment of analysis of handedness: the Edinburgh inventory. *Neuropsychologia* 1971;9:97–113.
- 10 Jet-2 Study Group. Interim report of JET-2 study. *Jpn J Neurosurg* 2008;17:823–9 (in Japanese).
- 11 Nawrot M. Disorders of motion and depth. *Neurol Clin* 2003;21:609–29.
- 12 Trick GL, Silverman SE. Visual sensitivity to motion: age-related changes and deficits in senile dementia of the alzheimer type. *Neurology* 1991;41:1437–40.
- 13 Sacks O. *Migraine: revised and expanded*. New York, NY: Random House Inc. 1999: P85.
- 14 Vaina LM, Cowey A, Eskew RT, *et al*. Regional cerebral correlates of global motion perception. Evidence from unilateral cerebral brain damage. *Brain* 2001;124:310–21.
- 15 Barton JJ, Simpson T, Kiriakopoulos E, *et al*. Functional MRI of lateral occipitotemporal cortex during pursuit and motion perception. *Ann Neurol* 1996;40:387–98.
- 16 Nakamura H, Kashii S, Nagamine T, *et al*. Human V5 demonstrated by magnetoencephalography using random dot kinematograms of different coherence levels. *Neurosci Res* 2003;46:423–33.
- 17 Watanabe S, Kakigi R, Miki K, *et al*. Human MT/V5 activity on viewing eye gaze changes in others: a magnetoencephalographic study. *Brain Res* 2006;1092:152–60.

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