Right Angular Lesion and Selective Impairment of Motion Vision in Left Visual Field

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Saito, H., Kanayama, S. and Takahashi, T. Right Angular Lesion and Selective Impairment of Motion Vision in Left Visual Field. Tohoku J. Exp. Med. 1992, 166 (2), 229-238 — A 45-year-old right-handed man had, for more than 20 years, a depressed fracture of the right parietal bone and selective impairment of movement vision in the left visual field; an illusory overestimation of speed of moving objects and extreme sensitivity to moving stimuli. Visuo-spatial perceptions of immobile objects, and other visual functions were normal apart from slightly decreased stereoacuity. The extent of depressed fracture corresponded approximately to the right angular gyrus. Carbamazepine or phenytoin did not alter the symptoms. After the operation, his symptoms were restricted to peripheral parts of the left visual field, with transient overestimation of speed of moving objects in the right hemifield. It was suggested that the human angular gyri may be involved in the central mechanisms of movement perception, and shift of visual attention or gaze to moving stimuli in contralateral visual field, especially in its peripheral part. — angular lesion; motion vision; illusory perception

Lesions in the primate posterior parietal cortex cause various impairments of visuo-spatial perceptions and visuo-motor integrations (Newcombe and Russell 1969; Ratcliff and Davis-Jones 1972; Ungerleider and Mishkin 1982; Hyvärinen 1982; Pierrot-Deseilligny et al. 1986; Andersen 1988). The central mechanism of movement vision has been extensively studied neurophysiologically in monkeys (Mountcastle et al. 1984; Maunsell and Newsome 1987) and psychophysically in normal human subjects (Brandit et al. 1973; Nakayama 1985). However, clinical reports on impaired movement vision with discussions on anatomical and pathophysiological mechanisms are scarce (Pötzl and Redlich 1911; Goldstein

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and Gelb 1918; Holmes 1919; Zihl et al. 1983). A patient of Pötzl and Redlich (1911) and that of Goldstein and Gelb (1918) showed impaired visual attention and spatial vision including motion vision. Their visual fields and auditory or language functions were also involved to various extents, suggesting bilateral lesions beyond the parietal lobes. Holmes (1919) studied eight patients with severe visuo-spatial dysfunction, and attributed those deficits to lesions in supramarginal and angular gyri of both sides. This opinion was supported by Riddoch (1935). Zihl et al. (1983) made detail investigations on a woman showing selective loss of the motion-related visual functions, and concluded the disorder was due to bilateral cerebral lesions affecting the lateral temporo-occipital cortex and the underlying white matter.

Most of these reports deal with loss of motion perception associated with bilateral destructive lesions of the parieto-occipital region. The impaired movement vision of the opposite mode, overestimation of motion speed, has only been briefly described (Brown 1985). Here we report a patient with a depressed fracture of the right parietal bone who had illusory overestimation of speed of moving objects and excessive sensitivity to moving stimuli in the left visual field.

**Case Report**

A 45-year-old right-handed man working in a construction company, was referred to the Department of Neurology, Hachinohe City Hospital on June 18, 1987, because he felt abnormally rapid movements of moving objects in his left visual field.

At the age of 17, he accidentally hit the right side of his head against a concrete wall, and had severe focal headaches for several days without other neurological symptoms. Later he felt as if his “left eye” was covered with a thin transparent film, and had slight difficulty in judgement of the distance and speed of rapidly moving objects on his left side. The following year, he obtained a driver’s license and started working as a dump-truck driver in a construction company. While driving his truck, he noted that, when he looked to the left side, he felt the velocity at 40 km an hour to be more than 70 km an hour. When he was stopping at a cross road, the speed of cars seen in the left visual field appeared him very fast, and he also felt as if they were going to collide with his truck. When he stared directly at those cars, the illusory sensations disappeared. Thus, the velocity of cars was recognized faster in the peripheral than in the central hemifield. Cars running in his right side never gave him such sensation. Because of these symptoms he changed his job to a rock-driller in the same company. Still, his visual troubles persisted. He had fears on crossing heavily congested streets. Otherwise, he had no gross troubles in his daily life, though he was extremely nervous about and annoyed by subtle movements of objects in the left visual field, especially in its periphery. He had no troubles with immobile or slowly moving objects.

He believed that the trouble was in his left eye because of slight alleviation of the abnormal visual sensation by covering it with an eye patch. He visited several ophthalmological clinics, where no apparent deficits were detected. Some ophthalmologists regarded his symptoms psychogenic, and others prescribed glasses for astigmatism. He changed the glasses more than 20 times without beneficial effects.

Routine physical and neurological examinations revealed no abnormalities, except for a depressed fracture of the right parietal bone. CT and MRI of the brain indicated that the fractured bone has been compressing the underlying cerebral structures, but without appar-
ent abnormal images in the parenchyma (Fig. 1). The depressed part of the skull on horizontal and semi-coronal CTs was serially reconstructed and projected on the brain schema. The extent of the fracture was shown to correspond approximately to the right angular gyrus (Fig. 2).

Cerebral angiograms were normal. The EEG was normal apart from occasional suppression of sleep-spindles in the right hemisphere (Fig. 3). There was no apparent hemispheric difference in photic-driving responses elicited by 5 Hz stroboscopic white light stimulation,
or in power spectrum analysis in responses to 5 Hz flickering polka-dots stimulation. Lateral hemifield stimulation of either right or left eye (Takahashi 1989) gave similar results. For pattern reversal visual evoked potentials, unipolar derivation was used with ipsilateral earlobe reference. The right and left eyes were tested separately or simultaneously. The latency of P-100 of VEPs thus obtained showed no hemispheric difference, but its peak-to-peak amplitude was higher on the right side than on the left (Fig. 4).

**INVESTIGATIONS AND RESULTS**

Neuropsychological evaluation demonstrated normal speech and auditory comprehension of language as well as reading and writing. Two point-discrimination and stereognosia were also intact. His visual acuity and visual fields examined by Goldmann’s perimeter and by static automated perimeter (Hunfry’s field analyzer with 81 points over 0-60°) were normal. Optokinetic nystagmus (OKN) was tested by an optokinetic tape; a strip of white cloth with 8 red squares (4 × 4 cm) attached uniformly. The tape was moved about 40 cm apart from the patient’s eyes. OKN was equally elicited either when the tape was moved rightward or leftward, and the features of OKN did not differ from those observed in normal subjects. Perception of colors, forms, and various objects and faces were prompt and correct. He could accurately thread a needle, pick up coins on the table, bisect a horizontal line, and copy designs without omitting any parts on the left side. Judgment of line orientation (Iowa University: Form-V)
was superior with only one misjudged out of 30 test items. He showed no constructional or dressing apraxia. He could draw well a self-portrait, a ground plan of his house, and a map of Japan.

Visual functions for moving objects were evaluated in the following manners, because a tachistoscope or other specific apparatus for that purpose were not available. Two pendulums consisting of a fishing line and Japanese 5-yen brass coin with an outer diameter of about 21 mm and a hole in its center were used to examine the perception of distance, depth or movements. Each pendulum was shown from the back of the patient in different parts of his visual field, and sometimes in the same quadrant. Next, each pendulum was swung separately or simultaneously on his right and left sides parallel to the sagittal or coronal planes, or in a circular form. The relative angular speed or frequency was roughly altered by changing the length of strings. The patient was asked to keep watching the nose of a examiner, who sat about 1.5 m from the patient and checked the shift of patient’s gaze. The patient was further asked to indicate,
verbally or by using his index fingers, the approximate distance of two coins, directions and phase synchrony of their movements as well as their relative velocity.

He correctly distinguished the relative distance of immobile coins as well as the direction, frequency and the phase differences of their movements. But he more frequently blinked when the left coin approached him than when the right did. He stated that movements of the coin appeared accelerated in the peripheral part of the left hemifield, and felt as if the left coin would pierce his eye. These tendencies were more apparent under binocular vision than under monocular vision. In the latter condition, the abnormal sensation was stronger in the left eye than in the right eye.

**Treatments and course**

Based on these findings, it was suspected, at first, that his visual symptoms might be due to the irritative lesions in the right posterior parietal cortex resulting from the compression and/or possible leptomeningeal adhesion to the cortical surface. Carbamazepine 400 mg or phenytoin 200 mg per day given for several weeks were noneffective. An operation for the depressed fracture was performed on January 23, 1988. The parietal bone, leptomeninges or the parietal cortex showed no adhesion. The blood vessels and color of the cortical surface appeared normal. The fractured bone was replaced with an artificial bone. Operative procedures were eventless. During the next several weeks after operation, he said that the speed of moving objects in the right side appeared to be more rapid. This symptom in the right side regressed gradually and reappeared in the peripheral part of the left side. He was no longer frightened by overestimation of speed of moving things, but was still nervous about subtle movements on his left side. Fifteen months after the operation the patient was tested again with the pendulums. Though he stated that the movement appeared faster in the periphery of his left visual field, his blinking tendency decreased. His stereoacuity tested with STEREOTEST-CIRCLES (Stereo Optical CO., Inc. IL, USA) showed that the patient was able to resolve a visual disparity of 60 seconds or larger, but not that of 50- and 40-seconds.

**DISCUSSION**

Our patient had, for more than 20 years, a depressed fracture of the right parietal bone and selective impairment of movement vision; the illusory overestimation of speed of moving objects and an extreme sensitivity to moving stimuli in the periphery of the left hemifield. Stereoacuity tested after the operation was slightly low (60 s of arc). Visuo-spatial perceptions of immobile objects and other visual functions; visual acuity, visual fields, oculomotor movements, perception of objects, color, form, size or geographical orientations were intact. Radiologically, the depressed bone appeared to have compressed the angular
region, but no apparent destructive lesions were detected during the operation.

With respect to alterations of motion perception in patients with the parieto-occipital lesions, Brown (1985) described briefly that "In rare cases, there may be a loss of the continuity of perception. The patient sees a series of more or less stationary images." This may correspond to symptoms in patients reported by Pötzl and Redlich (1911) Goldstein and Gelb (1918) and by Zihl et al. (1983). Brown (1985) further commented that "more common is the illusion that events are speeded up or, less often, slowed down. There may be a slowing in central vision and acceleration in the periphery. A stimulus may seem to move excessively fast in defective areas of the visual field." This illusion was one of our patient's chief complaints. Though Brown described this illusory motion perception to be rather common, we could not find detail case reports on the disorder or discussions on its pathophysiological mechanisms in available literature.

In monkey, a major corticocortical pathway involved in motion analysis originates in the area V1, progresses through the middle temporal area (MT) to the medial superior temporal area (MST) and to the area 7a (PG) (Maunsell and Newsome 1987). Areas MT and MST containing neurons with high selectivity for direction and speed of movement, rotation or size change, are supposed to be involved in motion analysis (Newsome et al. 1985; Saito et al. 1986; Tanaka et al. 1986; Maunsell and Newsome 1987). The area 7a also contains light sensitive neurons, in addition to those related to gaze-fixation, oculomotor movements and arm projection-manipulation. These light sensitive neurons are markedly activated by behavioral attention (Mountcastle et al. 1981, 1984; Sakata et al. 1985). Furthermore, Motter and Mountcastle (1981) found, in the area 7a, movement- and direction-sensitive neurons with large or even bilateral receptive fields preferentially distributed in the peripheries of the visual field with the radial arrangement. They concluded that the light sensitive neurons of area 7 possess properties well suited for signaling motion in the immediate behavioral surround and of the apparent motion that accompanies head movements and forward locomotion, as well as for the attraction of gaze and attention toward events in peripheral visual fields. McKee and Nakayama (1984) described that, in looking straight ahead and walking, the binocular disparity of the foveal visual image supplies informations about depth, and the peripheral visual field plays a role in registration of velocity information.

We initially suspected some irritative mechanism in our patient, but carbamazepine or phenytoin did not alter his complaints. Electrical stimulation of the human inferior parietal cortex has been reported to induce no sensory experiences (Penfield and Rasmussen 1968). The non-paroxysmal nature of his symptoms also contradicted our initial assumption. Because of the long history of his depressed fracture, we could not assess the precise nature of functional alterations in the underlying cerebral structures. However, an increased visual attention and sensitivity to the moving stimuli might be best explained by supposing that
certain neuronal groups similar to above-described opponent vector neurons do exist in human parietal cortex, and that the depressed fracture might have resulted in their constant activation possibly by a disinhibitory mechanism. Though the exact homologies between the areas of the posterior parietal cortex in monkeys and humans are unclear, the angular gyrus is regarded cytoarchitecturally homologous to the monkey area PG (Andersen 1988).

Human psychophysical studies suggest that the central and peripheral moving stimuli have different influences on movement perception. According to Brandit et al. (1973), with moving stimuli restricted to the periphery of the visual field, the observer refers to the motion to his own body and experiences the objects in the outer world as being stationary, and on gazing at moving stimuli in the center of the visual field, the subject experiences himself as being stationary and the stimulus as moving. Mountcastle et al. (1984) discussed the functional relation of opponent vector neurons to physiological illusions of self motion in human induced by the moving visual stimuli. This visual illusion and other visual functions such as critical flicker fusion were not examined in our patient. The test using the moving pendulums could not detect any differences of his motion perception in either hemifields, except for more frequent blinking to the moving stimuli in his left side than those in the right side. Furthermore, while OKN was normally elicited by an optokinetic tape in our patient, this method stimulates the central and pericentral rather than peripheral parts of the visual field. Several studies indicated that there may be some differences in features of OKN elicited by moving stimuli to different parts of retina (Hood 1975; Cheng and Outerbridge 1975; Dubois and Collewijn 1979), and that OKN is frequently impaired by the posterior parietal lesions (Smith and Cogan 1959; Hyvärinen 1982; Lynch and McLaren 1983). Therefore, some abnormalities of OKN would have been detected in our patient, if the peripheral and central parts of each hemifields were stimulated separately under electro-oculographic recording. Thus, more quantitative and systematic tests are required to clarify the mechanism of illusory overestimation of motion speed in our patient. Still, his selectively impaired motion vision may provide an evidence that the human angular gyrus may be involved in the central mechanisms of motion perception in the opposite hemifield, as suggested by Holmes (1919). This assumption does not exclude the probable involvement of other structures such as the posterior part of the middle temporal sulcus (Zihl et al. 1983).

Postoperative transient appearance of the illusory motion vision in the right hemifield is also interesting. The striate and extrastriate visual cortex has abundant connections with the homo- and heterotopical areas in the opposite hemisphere, and transfers visual informations for binocular fusion, stereopsis, and perceptual continuity of the visual space (Pandya and Vignolo 1969; Doty and Negrao 1973; Berardi et al. 1988). The interhemispheric interactions between the homotopical areas may involve inhibitory action (Doty and Negrao 1973).
Though we did not examine the patient sufficiently during this period, we suppose that the surgical removal of the compression on the right parietal lobe might have caused a transient rebound in the balance or in the rivalry of visuo-spatial information flow in both hemispheres.

References


