Crossed non-dominant hemisphere syndrome in a right-hander

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A right-handed patient with a large left temporo-parietal infarction manifested various non-dominant hemisphere signs. He had two left-handed children. On neurobehavioural examinations, he did not show aphasia or ideomotor apraxia, but did show hemispatial neglect, spatial agragraphia, constructional apraxia, auditory and tactile extinction, anosodiaphoria and affective changes, all of which are usually observed after right hemispheric damage. We conclude that he has a reversed cerebral laterality of cognitive functions and showed crossed non-dominant hemisphere syndrome.

Keywords: Cerebral laterality – Handedness – Right hemisphere syndrome

INTRODUCTION

Injury of the right hemisphere may cause left hemispatial neglect, constructional apraxia, extinction on double-simultaneous stimulation, motor impersistence, anosognosia or prosopagnosia (Hier *et al.*, 1983). Other studies have reported that spatial agragraphia (Hécaen and Marcie, 1974), acute confusional state (Mesulam *et al.*, 1976; Mori and Yamadori, 1987) or affective deficits (Gainotti, 1989) occur after right hemispheric damage. These neurobehavioural abnormalities (non-dominant hemisphere syndrome) may be seen in left-sided injuries if cerebral laterality of cognitive function is reversed.

In several case reports, lesion in the left hemisphere was associated with normal language functions and visuospatial deficits (Taylor and Solomon, 1979; Kellar and Levick, 1985; Junqué *et al.*, 1986; Caramazza and Hills, 1990; Maeshima *et al.*, 1992), but the authors did not mention most of the other signs. We here report a patient with a large left temporo-parietal infarction who manifested various non-dominant hemisphere signs.

CASE REPORT

A 53-year-old male university graduate was referred to our neuropsychology section for evaluation 2 months after an ischaemic stroke. He had been the

director of a driving school but retired after the stroke. He reported himself as a definite right-hander, that is, before the stroke he had always used the right hand for writing, drawing, throwing, brushing his teeth, cutting with scissors and eating with chopsticks. He had two right-handed and two left-handed children. A neurological examination revealed a right hemiparesis and a right homonymous hemianopsia. A cranial computed tomogram (CT) showed a left temporo-parietal infarction. In brief neuropsychological assessment, there were no signs of aphasia but he showed spatial distortion in writing and right-sided hemineglect in copying a transparent cube.

He was admitted to our hospital 5 years later, because of an episode of falling, and we had a chance to re-examine him. His wife reported that his inattention to the right side had been unchanged for 5 years, and he repeatedly hit his right limbs. His neurological state was unchanged, and routine laboratory findings were normal. Magnetic resonance imaging (MRI) of the head delineated the old infarction but revealed no new lesions (Fig. 1).

He was awake and cooperative during the assessment. His digit span was five forward, two backward, and he scored 23 in the Mini-Mental State Examination (MMSE). The Wechsler Adult Intelligent Scale—Revised (WAIS-R) showed verbal IQ of 76.

FIG. 1. Cranial magnetic resonance T1-weighted images 5 years after onset demonstrated a left temporo-parietal infarction.

TABLE I. The patient's scores in the subtests in the Western Aphasia Battery

Spontaneous speech	19/20
Auditory comprehension	9.5/10
Repetition	10/10
Naming	8.4/10
Reading	7.4/10
Writing	1.2/10

His running speech was fluent without paraphasia, articulatory disturbance or dysprosody. Repetition, confrontation naming and auditory comprehension were all normal. Comprehension of written words was preserved, but he tended to neglect right-sided words in reading sentences. His writing, performed with the left hand, consisted of distorted letters, occasional stroke omissions and slanted lines, with no signs of aphasic dysgraphia. Thus, he had spatial agraphia (Hécaen and Marcie, 1974). Table I shows the scores of subtests in the Western Aphasia Battery (WAB). There was no buccofacial or ideomotor apraxia.

Many non-dominant hemisphere signs were present. We observed right hemineglect in line bisection, line cancellation, copying figures and imitating finger postures, as well as reading sentences (Fig. 2). On Raven's Colored Progressive Matrices, he tended to point to left-sided pictures, and never pointed to right-sided ones. His neglect did not improve when

FIG 2. Line bisection and copying figures showed distinct right hemineglect.

the stimuli were presented in the left visual field. We believe that his neglect cannot be attributed to the visual field deficit although we did not assess any representational form of neglect. Constructional disturbance was apparent in the Block Design subtest of WAIS-R. Auditory and tactile extinction of a right-

TABLE II. Reported patients with left-sided lesions who showed non-dominant hemisphere signs and no aphasia

	Taylor <i>et al.</i> (1979)	Kellar <i>et al.</i> (1985)	Junqué <i>et al.</i> (1986)	Caramazza <i>et al.</i> (1990)	Maeshima <i>et al.</i> (1992)	Present patient
Handedness	Mixed	Right	Right	Left	Right	Right
Familial sinistrality	+	—	—	N.D.	+	+
Extent of lesion	T-P	F-T-P	F-T-P	P (subcortical)	F-T-P	T-P
Neurobehavioural findings						
Language assessment	Token: 21/22	Token: 29/36 BNT: 69/85	Token: 35/36 BNT: 55/60	Token: 33.5/36 42/60	Normal SLTA subtests in speech and hearing	WAB AQ: 93.8
Agraphia	Spatial	N.D.	Apraxic	Spatial	Aphasic	Spatial
Ideomotor apraxia	N.D.	N.D.	+	N.D.	—	—
Right hemineglect	+	+	+	+	+	+
Constructional apraxia	+	+	+	—	+	+
Sensory extinction	N.D.	N.D.	+	+	N.D.	+
Motor impersistence	N.D.	N.D.	N.D.	N.D.	N.D.	—
Prosopagnosia	+	N.D.	N.D.	N.D.	N.D.	—
Anosognosia or anosodiaphoria	N.D.	N.D.	N.D.	N.D.	+	+
Affective deficit	+	+	+	N.D.	N.D.	+

+, positive; —, negative; N.D., not described; T-P, temporo-parietal; F-T-P, fronto-temporo-parietal; P, parietal; Token, Token Test; BNT, Boston Naming Test; SLTA, Standard Language Test for Aphasia (Takeda, 1977); WAB, Western Aphasia Battery; AQ, Aphasia Quotient.

sided stimulus was present on double-simultaneous stimulation. Motor impersistence was not demonstrated in the eye closure and tongue protrusion procedure.

Anosodiaphoria for the right hemiparesis was apparent in his daily activities. For example, he often stood up from a wheelchair with no assistance, as if he had no motor weakness, and almost fell to the right side. When asked, however, he did not deny the presence of the hemiparesis verbally. Nursing staff recorded his affective state as “flat” and “indifferent”. He and his family reported no disturbance in his face recognition ability, though we did not assess this formally.

His neurological and neurobehavioural states did not change during the 2 months of his hospital stay.

DISCUSSION

This right-handed patient with a large left temporo-parietal lesion showed persistent right hemispatial neglect, spatial agraphia, constructional apraxia, auditory and tactile extinction, anosodiaphoria and affective change over 5 years after the stroke onset. On the other hand, aphasia and ideomotor apraxia were never observed.

Right hemispatial neglect, if present, is usually slight in degree and rarely persists for months in left-brain-damaged patients with normal laterality of cognitive functions (Weintraub and Mesulam, 1989). This

patient's hemineglect, however, was distinct in many tasks and persisted for 5 years. We also observed spatial agraphia (Hécaen and Marcie, 1974), anosodiaphoria for hemiparesis (Babinski, 1914) and affective change with indifference reactions (Gainotti, 1972), which usually occur after right hemispheric damage. These findings suggest the affected left hemisphere of this patient functioned as non-dominant. Constructional apraxia and persistent sensory extinction may occur after damage to either hemisphere, but are more frequent in right-sided injury (Benton, 1989; De Renzi *et al.*, 1984). We interpreted his low scores in digit span and MMSE as a sign of confusional state, which may be associated with non-dominant brain lesion (Mesulam *et al.*, 1976; Mori and Yamadori, 1987) and persist for years (Mullaley *et al.*, 1982). Thus, these signs in this patient are consistent with non-dominant hemispheric injury.

Right brain damage may also cause motor impersistence or prosopagnosia, both of which were absent in the patient. A right anterior lesion is said to be responsible for motor impersistence (Kertesz *et al.*, 1985) and right occipital damage for prosopagnosia (Landis *et al.*, 1988). We believe that the patient's temporo-parietal lesion spared these areas.

Thus, we conclude that this patient had a reversed laterality of cognitive functions and showed a crossed non-dominant hemisphere syndrome. Table II summarizes reported patients with left-sided lesions who

showed non-dominant hemisphere signs and were described as having no aphasia. All of them had a diffuse cortical infarction in the territory of the left middle cerebral artery except the patient of Caramazza and Hillis (1990). Taylor and Solomon (1979) and Kellar and Levick (1985) did not mention ideomotor apraxia, which was observed in the patient of Junqué *et al.* (1986). Most reports contain no mention of some non-dominant hemisphere signs, such as sensory extinction, motor impersistence, anosognosia or anosodiaphoria. On the other hand, we found most of the non-dominant hemisphere signs in our patient, but no ideomotor apraxia or aphasia.

The patient had two left-handed children, though he himself was right-handed. Most patients with left handedness or family history of sinistrality have normal or bilateral presentation of cognitive functions (Hécaen *et al.*, 1981; Bryden, 1988). On the other hand, this patient showed an almost complete non-dominant hemisphere syndrome, and absence of language dysfunction. These facts suggest that he had a discrete and reversed lateralization of cognitive function.

REFERENCES

- Babinski MJ (1914) Contribution à l'étude des troubles mentaux dans l'hémiplégie organique cérébrale (anosognosie). *Revue Neurologique*, **27**, 845-848.
- Benton A (1989) Constructional apraxia. In: *Handbook of Neuropsychology*, Vol. 2 (Eds F. Boller and J. Grafman), pp. 387-394. Elsevier, Amsterdam.
- Bryden MP (1988) Cerebral specialization: clinical and experimental assessment. In: *Handbook of Neuropsychology*, Vol. 1 (Eds F. Boller and J. Grafman), pp. 143-159. Elsevier, Amsterdam.
- Caramazza A and Hillis AE (1990) Levels of representation, co-ordinate frames and unilateral neglect. *Cognitive Neuropsychology*, **7**, 391-445.
- De Renzi E, Gentilini M and Pattacini F (1984) Auditory extinction following hemisphere damage. *Neuropsychologia*, **22**, 733-744.
- Gainotti G (1972) Emotional behavior and hemispheric side of lesion. *Cortex*, **8**, 41-55.
- Gainotti G (1989) Disorders of emotions and affect in patients with unilateral brain damage. In: *Handbook of Neuropsychology*, Vol. 3 (Eds F. Boller and J. Grafman), pp. 345-361. Elsevier, Amsterdam.
- Hécaen H and Marcie P (1974) Disorders of written language following right hemisphere lesions: Spatial dysgraphia. In: *Hemisphere Function in the Human Brain* (Eds J. Beaumont and S. Dimond), pp. 345-366. Paul Elek, London.
- Hécaen H, De Agostini M. and Monzon-Montes A (1981) Cerebral organization in left-handers. *Brain and Language*, **12**, 261-284.
- Hier DB, Mondlock J and Caplan LR (1983) Behavioral abnormalities after right hemisphere stroke. *Neurology*, **33**, 337-344.
- Junqué C, Litvan I and Vendrell P (1986) Does reversed laterality really exist in dextrals? A case study. *Neuropsychologia*, **24**, 241-254.
- Kellar LA and Levick SE (1985) Reversed hemispheric lateralization of cerebral function: A case study. *Cortex*, **21**, 469-476.
- Kertesz A, Nicholson I, Cancelliere A, Kassa K and Black SE (1985) Motor impersistence: A right-hemisphere syndrome. *Neurology*, **35**, 662-666.
- Landis T, Regard M, Bliestle A and Kleihues P (1988) Prosopagnosia and agnosia for noncanonical views. An autopsied case. *Brain*, **111**, 1287-1297.
- Maeshima S, Kajiwaru T, Dohi N, Yoshimura M, Nakagawa T and Shigeno K (1992) "Left unilateral agraphia with right hemiparesis" after occlusion of the left middle cerebral artery. *Brain and Nerve*, **44**, 661-666 (in Japanese).
- Mesulam M-M, Waxman SG, Geschwind N and Sabin TD (1976) Acute confusional states with right middle cerebral artery infarctions. *Journal of Neurology, Neurosurgery and Psychiatry*, **39**, 84-89.
- Mori E and Yamadori A (1987) Acute confusional state and acute agitated delirium. Occurrence after infarction in the right middle cerebral artery territory. *Archives of Neurology*, **44**, 1139-1143.
- Mullaley W, Huff K, Ronthal M and Geschwind N (1982) Chronic confusional state with right middle cerebral artery occlusion. *Neurology*, **32**, 496.
- Takeda K (1977) Standard language test of aphasia: detailed description of construction of aphasia test in Japanese. *Shinkei Kenkyu no Shinpo*, **3**, 450-458 (in Japanese).
- Taylor HG and Solomon JR (1979) Reversed laterality: A case study. *Journal of Clinical Neuropsychology*, **1**, 311-322.
- Weintraub S and Mesulam M-M (1989) Neglect: hemispheric specialization, behavioral components and anatomical correlates. In: *Handbook of Neuropsychology*, Vol. 2 (Eds F. Boller and J. Grafman), pp. 357-374. Elsevier, Amsterdam.

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