NOTE

RIGHT-SIDED NEGLECT IN A LEFT-HANDED: EVIDENCE FOR REVERSED HEMISPHERIC SPECIALIZATION OF ATTENTION CAPACITY

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Abstract—Severe hemi-spatial neglect, anosognosia, contralateral hypokinesia, aprosodia, and visual-spatial constructive difficulties—typically seen in right-handers with right hemisphere lesions—were observed in a left-handed patient with an acute left frontal cortical and subcortical infarct. There was no evidence of accompanying aphasia and the neglect syndrome gradually resolved over a 2-week period. The assumption by the left hemisphere of a classic right hemisphere attention, visuo-spatial and prosodic superiority may represent a case of reversed hemispheric specialization.

INTRODUCTION

Neglect of the contralateral hemi-space with accompanying anosognosia and visual-spatial abnormalities is common after right hemisphere lesions in right-handers [4, 8, 15]. Although contralateral neglect is reported after left hemisphere lesions [9, 15], right hemisphere lesions cause a neglect syndrome which is more intense and of longer duration [1, 16, 23, 24]. HEILMAN et al. [18] have defined major components of the neglect syndrome to include decreased orientation and attention to the contralateral hemifield, hemiakinesia, and unilateral extinction on double simultaneous stimulation. Associated signs may include anosognosia, anosodiaphoria, allesthesias, flattened affect, decreased vigilance, motor impersistence, constructional apraxia, dressing apraxia, spatial dyslexia, spatial dyscalculia and loss of topographic orientation [15, 18]. Patients with the neglect syndrome may demonstrate some or all of these symptoms, though the differential severity of these symptoms is rarely reported.

Lesions in several different structures including inferior parietal [18] and dorsolateral frontal neocortex [9, 17], cingulate gyrus [17], thalamus [36], striatum [9, 19] and subcortical connections [12, 14] can result in neglect in humans. Studies correlating CT evidence with clinical findings of neglect in large series of patients with single right hemisphere lesions report a predominance of right inferior parietal [35] and occasionally dorsolateral frontal [22] lesions in relation to visual neglect. It has been suggested that a lesion in a cortico-limbic-reticular activating system controlling arousal and orientation underlies the neglect syndrome [16]. It has, however, been shown that animals with large reticular lesions and arousal deficits are still able to orient attention [31], suggesting that specific deficits in attention also contribute to the neglect syndrome [28].

The finding that right hemisphere lesions produce a more severe and long-lasting neglect syndrome supports an hemispheric asymmetry in either an arousal or attention system. It has been proposed that the right hemisphere may control both attention and arousal ability in both the contra- and ipsilateral hemispace, whereas the left hemisphere mediates these functions only in the contralateral hemispace [23]. According to this theory, left hemisphere lesions produce minor neglect symptoms since the right hemisphere can assume control of the right hemispace. Marked left-field neglect is seen after right-sided lesions since the left hemisphere is unable to mediate left hemispace attention and orientation ability. However, PLOURDE and SPERRY [26] have demonstrated that the disconnected left hemisphere in patients with complete forebrain commissurotomy reveals no evidence of left hemispatial neglect or neglect of the left hemibody. These authors suggest that right hemisphere pathology alone cannot explain the neglect syndrome.

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Other studies suggest that a deficit in the ability to access an internal representation of the external world is critical to the neglect syndrome. Patients with the neglect syndrome are unable to describe previously recollected images on the neglected side [2]. Furthermore, both right- and left-hemisphere damaged patients make more errors comparing stimulus pairs differing on the contralesional side of space even when the stimuli are presented by slowly passing them behind a central vertical slit [3, 25]. This method of presentation allows the patient only a partial view of the stimulus at a given time, and elicits the patient's inability to conceptualize the contralesional side of mental images. Recent evidence also indicates that visual neglect is both body- and environment-centered [6]. Neglect patients report stimuli more often to the right of the body midline and to the right of the environmental vertical axis even under conditions in which the midline and the environmental vertical axis do not correspond (e.g., by having patients recline on their sides). These findings suggest that attention is directed relative to both an environmental axis as well as a body frame of reference.

Hemispheric specialization in sinistrals

The pattern of cerebral hemispheric organization in left-handers is less well defined than that of right-handers. Various studies have shown that while between 86 to 99% of right-handers show strong left hemisphere lateralization for language, only 61 to 70% of left-handers demonstrate this pattern, with the remaining cases dividing equally between bilateral and right hemisphere representation for speech [5, 27, 30]. Familial sinistrality appears to have an effect on cerebral lateralization of function, as bilateral representation for language is more likely in left-handers with a family history of left-handedness.

Little information is available regarding attention disorders in left-handers. The paucity of reports of contralateral neglect in left-handers may indicate that, as with language, attention capacity has a more bilateral distribution across the hemispheres in left-handers. Since a subset of left-handers can have well organized right hemisphere language dominance, it seems likely that the right hemisphere attention dominance in right-handers might on occasion be strongly lateralized to the left hemisphere of left-handers. We had the opportunity to observe such a patient whose case forms the basis of this report.

CASE REPORT

A 49-year-old left-handed diabetic woman controlled on oral hypoglycemics was admitted for the acute onset of right-sided weakness. General medical evaluation was unremarkable except for mild obesity and early diabetic retinopathy. There was no history of familial sinistrality. The patient reported that she had always used her left hand for writing and throwing a ball and her left foot for kicking. Neurological exam at 24 hr after onset revealed a woman sitting with her right hand positioned behind her back with her head, eyes and trunk turned to the left. She would not acknowledge her illness. When asked why she was in the hospital she replied, “I feel constipated”. Although she was not lethargic, she had flattened affect, was confused and poorly attentive and had motor impersistence. She did not orient to right-sided auditory, visual or somatosensory stimuli, and evaluation of right-sided primary and parietal sensory functions was impossible due to the severe neglect. She had a right central seventh and no distal motion in her right arm or leg although with repeated prompting she could shrug her right shoulder. When her parietic right hand was placed in front of her and she was asked why it wouldn’t move she replied, “Because I’m left-handed”. She was able to move her normal left side quickly into either hemi-space and could perform sequential motor sequences with her left hand. Visual fields on day 1 were uninterpretable. There was a left gaze preference and a marked saccadic gaze deficit to the right. Although she would not perform voluntary saccades to the right, there was a full range of eye movements with the Doll’s eye maneuver.

CT scanning obtained at this time revealed an infarct in the dorsolateral left prefrontal cortex extending superiorly into the inferior portions of the frontal eye fields and posteriorly into the centrum semiovale (see Fig. 1). There was additional subcortical extension into the anterior limb of the internal capsule and the lateral surface of the caudate nucleus with some compression of the frontal horn.

At 3 days post-onset the anosognosia and motor paresis persisted. Visual fields were full, and primary and parietal sensory testing including graphesthesia and stereognosis were markedly improved on the right side. There was no reproducible visual, auditory or somatosensory extinction. However, the patient still manifested right-sided neglect, turning behavior to the left and hypometric saccades on right gaze.

Neuropsychological testing at 9 days post onset revealed normal speech and language except for a mild dysarthria and aprosodic, monotonic speech typically associated with right hemisphere disease [21, 29]. No syntactic or paraphasic errors were noted. Left-right discrimination and two-digit addition and multiplication were also intact. Right-sided neglect was still evident, though anosognosia had decreased. When asked about the reason for her hospitalization she replied, “It seems I can’t move my arm”. On Albert’s line cancellation test [1], the patient began working on the left side of the page and claimed to have finished the task after only the lines on the left had been crossed out. Moving the page to the patient’s right did not alter her perception that the task was completed. Similar performance was seen on a letter cancellation task in which the patient was asked to pick out all the “A’s” in a page of randomly scattered letters [33]. Her solution to this task was to circle only the “A’s” on the left-most side of the page.
Testing for constructional apraxia produced distorted drawings with neglect of the right-side of space apparent in her omission of half the petals on the daisy and the right side of the house and window (see Fig. 2). The patient also exhibited topographic disorientation as reflected in her distorted labeling of major locations on a United States map (see Fig. 3).
DISCUSSION

This left-handed patient with an acute left frontal lesion developed symptoms including contralateral neglect, anosognosia, constructional difficulties and motor impersistence classically associated with acute right hemisphere injury in right-handers. Right hemisphere symptomatology in left-handers has not received the same attention as linguistic disorders in left-handers. He
cahn [15] reported right-sided asomatognosia in three left-handers although it is not clear from the description whether these patients manifested the complete neglect syndrome or simply showed abnormalities in double simultaneous stimulation. The paucity of further reports of severe neglect syndrome in left-handers may indicate that, as for language, attention is organized bilaterally in a larger percentage of this group, as opposed to the right hemisphere superiority found in right-handers. The severity and persistence of the neglect syndrome from a left hemisphere lesion in this patient, however, indicates that attention capacity may on occasion be inversely represented in left-handers just as language capacity is lateralized to the right hemisphere in approx. 15–20% of left-handers. Whether attention capacity also undergoes inverse representation to this degree awaits study of larger numbers of cases.

That this classic non-dominant syndrome was produced by a frontal lesion is not surprising. Neuroradiological studies indicate that patients with severe neglect often have extensive lesions involving both frontal and parietal association cortex [20, 22]. Other studies have reported marked neglect from isolated right and on occasion left frontal lesions in right-handers [9, 17, 32]. One series reported that right brain-injured patients show a higher incidence of neglect following posterior lesions while neglect after left hemisphere lesions in right-handers tends to occur more often following frontal lesions [24].

The classic right hemisphere neglect syndrome may in fact be separable into several different components. Prominent neglect, as in this case, may be seen with minimal sensory abnormalities after frontal lesions. Conversely, marked abnormalities in double simultaneous stimulation may be seen in parietal patients without marked neglect or anosognosia. In the majority of cases of severe neglect, however, damage due to middle cerebral or internal carotid artery occlusion extends to both frontal and parietal regions producing a mixture of contralateral neglect and high order sensory dysfunction [23].

Cases of reversed hemispheric specialization which have been reported typically describe "crossed aphasia" in which language skills become disturbed following right hemisphere lesions in right-handers [7, 11]. Few persistent left-handed aphasis patients with right hemisphere lesions have been described [e.g. 10], perhaps indicative of the higher percentage of bilateral language representation in this group. Taylor and Solomon [34] reported a case of left hemisphere damage in a right-hander resulting in visuo-spatial dysfunction but without accompanying neglect or aphasia. In the present case, a left-hander with a left frontal lesion developed neglect, visuo-spatial disturbance and aprosodic speech, all typical findings in right-handers with right hemisphere lesions [23, 29]. In addition, the patient showed no indication of aphasia. This syndrome can be best explained as a reversal in hemispheric organization wherein attention and visuo-spatial skills are organized in the left hemisphere of this left-hander, similar to the right hemisphere organization of these functions in the right-handed population.

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