Letter to the Editor

Ipsilaterial neglect for non-verbal stimuli following left brain damage

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Unilateral spatial neglect (USN) is usually defined as the failure of unilateral brain damaged patients to attend or respond to stimuli in the hemispace contralateral to the site of lesion. This definition encompasses either a complete lateralisation of omissions or their asymmetric distribution in space, whereby only a few are committed ipsilaterally (Gainotti et al., 1990).

A few exceptions to this rule have been reported. Right-brain damaged patients showing right USN in drawing, line bisection and cancellation tasks have been described (e.g., Robertson et al., 1994). Similarly, cases of patients with left brain damage presenting with left neglect dyslexia, but no other signs of left neglect, have been reported (e.g., Patterson and Wilson, 1990; Haywood and Coltheart, 2001). We report on the novel observation of a left-brain damaged patient showing left USN in non-verbal tasks.

P36, a 42-year-old skilled workman, right-handed, with 8 years of formal education, after having suffered from hypertension for years, had an ischaemic stroke in the territory of the left middle cerebral artery encroaching upon the temporal and the parietal lobes, the posterior aspect of the frontal lobe, the insula and part of the Putamen (see Fig. 1). No other hypodensity or abnormality was noted on a series of CT and MRI scans.

At the time of testing, 40 days post onset he still suffered from severe right-sided paresis both in the upper and lower limb, while no neurological signs were detected on the left side. He was not aware of his motor deficits, as evinced by his performance on the VATA-M (Della Sala et al., 2009), suggesting a moderate anosognosia (score 13/36, zero best score, cut-off = 6.3). He showed no clear evidence of visual field deficits, clinically assessed, as he correctly identified 17 stimuli on the right side and 16 on the left; however he showed signs of extinction missing a total of 5/20 stimuli on the left under simultaneous presentation. P36 presented also with language deficits, labelled as global aphasia, as testified by his poor performance on the Token Test (10.75/36, cut-off = 26.25, Spinnler and Tognoni, 1987) and by the profile he achieved on the Aachen Aphasia Test (AAT) (Huber et al., 1984). He was not aware of his language problems, as demonstrated by his performance (score 13.9/36, zero best score, cut-off = 11.9) on the VATA-L (Cocchini et al., 2010).

As part of a large study he was also assessed with a large battery of tests aimed at investigating different aspects of USN (Della Sala et al., 2004). P36 performed normally on tests assessing representational neglect, including drawing from memory (no qualitative or quantitative difference between the left and the right side of the picture) and drawing a clock-face from memory (100% both for the left and the right side). He also had no problems with tests assessing personal neglect, including the Comb-and-Razor test (Beschin and Robertson, 1997) whereby he scored 27 on the left side and 24 on the right, the Fluff test (Cocchini et al., 2001, score 9/9 on the left and 15/15 on the right side) and the One-item test (Bisiach et al., 1986), whereby he promptly reached his right hand with his left. However, his performance on several tests assessing extrapersonal neglect was grossly asymmetrical, revealing a consistent tendency to ignore left-sided stimuli (see Table 1). He showed a mild impairment in reading single words (correct responses 23/35; 66%). Even if only three errors (25% of all errors)
were classified as left neglect paralexias (e.g., scostare [to push aside] → tostare [to toast]; autonomamente [autonomously] → nimamente [non-word]) according to the criteria proposed by Hillis and Caramazza (1990), almost all incorrect responses involved the left beginning letters of the stimulus words (e.g., transatlantico [transatlantic] → ratantico [non-word]).

As for his paresis and his aphasia, he was unaware of his neglect as shown by his performance on the CBS (Catherine Bergego Scale) compared to that of his professional carer (Azouvi et al., 2003).

No other cognitive deficit has been detected; in particular, he did not show signs of a dysexecutive syndrome, in particular he scored within the normal range in the Raven Progressive Matrices (29/48, cut-off = 17.5, Spinnler and Tognoni, 1987) and in the Brixton test, Burgess and Shallice, 1997 (4/10, cut-off = 3), or of callosal disconnection.

His neuropsychological profile is indicative of a left ipsilesional USN in both verbal and non-verbal tasks. This is the first report of a left ipsilesional USN involving tasks other than reading, even if limited to extrapersonal space. As in all ipsilesional USN cases reported in the literature, the presence of a subtle lesion in the other hemisphere cannot be ruled out completely. However, no neurological signs have been detected indicating damage to the right hemisphere and a series of morphological scans failed to find other lesions, including lesions of the corpus callosum. The presence of global aphasia is consistent with a cerebral lesion located in the left cerebral hemisphere of a right-handed individual. Therefore, unless the existence of two separate lesions (one on the left side causing aphasia and one, undetected, on the right side producing contralateral neglect) is assumed, the most parsimonious conclusion is that left neglect also followed the damage in the left hemisphere.

The ipsilesional cases pose a challenge to the theories proposed to explain USN (Karnath et al., 2002), as they only account for defective performance in the contralesional space. Each cerebral hemisphere is assumed to control perception and action in the contralateral space, as it does for sensory and motor processes. Therefore, each hemisphere is thought to subserve the cognitive mechanisms responsible for orienting attention towards the contralateral space (Heilman et al., 1993) or computing the contralateral half of mental representations (Bisiach et al., 1979). A lesion located in only one hemisphere is expected to cause patients to neglect information on the contralesional space and to limit their action within the ipsilesional space. The asymmetric behaviour following a brain lesion is not predicted to result in a preference for the space ipsilateral to the damaged hemisphere.

Ipsilesional cases described in the literature, showing left dyslexia following left-sided lesions and right neglect in exploration and drawing tasks following right-sided lesions, could be reconciled by postulating that the deficit manifests in tasks mainly processed by the damaged hemisphere. Indeed, several patients presenting with ipsilateral neglect dyslexia have lesions encroaching upon the corpus callosum (e.g., Cubelli et al., 1993; Binder et al., 1992). This interpretation does not pertain to the pattern presented by case P36, whose neglect encompasses cancellation tasks. Cases like P36 therefore should be considered when discussing possible theories of USN.

According to Robertson et al. (1994), the paradoxical ipsilesional, right omissions could be due to a combination of two different and independent factors: a compensatory leftward, contralesional scanning and a non-lateralised attentional loss. However, it is worth noting that the patients described by Robertson et al. (1994) omitted stimuli in the cancellation tasks bilaterally, even if ipsilesional omissions were more frequent. For instance, their right-brain damaged patient e3 omitted stimuli on both sides: on star cancellation his omissions were 11% on the left and 15% on the right, on line cancellation they were 15% on the left and 40% on the right. In contrast, P36’s performance was always flawless in the contralesional half of the stimulus array.

Ellis et al. (2006) described six right-brain damaged patients showing a severe left neglect coupled with signs of neglect for extreme right space. The authors infer that neglect is bilateral but asymmetric and that ipsilesional extreme neglect could due to a transhemispheric diaschisis, i.e., to a reduced activation of the intact hemisphere. However, the patient whose case is reported here did not show evidence of contralateral neglect and, importantly, was tested with the stimulus array placed just in front of him, having the most lateral stimuli not very far from the body midline.
In conclusion, the behavioural pattern presented by P36 has never been reported before. To attempt an explanation, one could infer that in some individuals each cerebral hemisphere is involved in processing the ipsilateral space. As in the cases of “crossed aphasia” or “crossed apraxia” (Marchetti and Della Sala, 1997), some ipsilesional neglect cases could be accounted for as evidence of “crossed neglect”.

REFERENCES


