New insights from a not-so-neglected field: Hemispatial neglect

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Hemispatial neglect remains one of the most remarkable syndromes investigated by behavioral neurologists. It occurs only after relatively focal (or at least asymmetric) brain damage, most commonly stroke, but is occasionally observed in asymmetric atrophy syndromes such as corticobasal syndrome or Posterior Cortical Atrophy due to Alzheimer’s Disease pathology. It is also sometimes associated with other focal lesions such as tumor, abscess, head injury, or following resection. It is mild and transient in primates, even after large lesions, which reveals that only humans, who may have developed hemispheric asymmetry due to language, have hemispheric specialization for spatial attention or some aspect of attention such that a unilateral lesion causes marked hemispatial neglect. For example, Corbetta and Schulman [1] have proposed a model in which the dorsal spatial attention network is bilateral and symmetric, but the ventral nonspatial frontoparietal attention network is right-hemisphere dominant. Ventral lesions also cause physiological changes and disrupt connections to and within the spatial attention. Only a lesion to the ventral (right) network, which also affects the dorsal network, not the left dorsal network alone, causes marked hemispatial neglect. This model would account for more frequent and severe hemispatial neglect reported in many studies of neglect. However, Kleinman and colleagues [2] reported that hemispatial neglect is equally common after left and right hemisphere stroke, although the type of neglect observed is different after a lesion to the left versus the right hemisphere. Individuals with left hemisphere stroke were found to have stimulus-centered or object-centered neglect more often than viewer-centered neglect; while individuals with right hemisphere stroke were found to have the opposite: more frequent viewer-centered neglect. Viewer-centered neglect is easier to detect, as the person will neglect everything on the contralateral (usually left) side of his or her view, bump into things on the contralateral side of the body, fail to eat food on the contralateral side of the tray, and so on; while the person with stimulus-centered neglect will make more subtle errors in processing the contralateral side of individual stimuli on both sides of space (sometimes not recognized as hemispatial neglect). Therefore, the detection rate for viewer-centered neglect may be higher, accounting for the higher rate of neglect after right hemisphere stroke reported in some studies of neglect.

Severity of hemispatial neglect also increases with age, even after controlling for lesion volume [3], possibly due to worse ability to compensate with the intact hemisphere [4]. In fact, hemispatial neglect in children is rare, even after hemicraniotomy, including removal of the entire right cortex [5]. Severity of neglect also increases with the degree of leukoaraiosis, even after controlling for infarct size, consistent with the findings of the important role of lesions to white matter tracts in causing hemispatial neglect [6–9]. Thus, even though hemispatial neglect has been well-described and investigated for centuries, many new insights are being gained about the neural substrates underlying spatial attention and representation from investigating the various types of spatial neglect and related disorders and the factors that influence their occurrence and severity.

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Most of the literature on hemispatial neglect has focused on left visual neglect in near space (e.g. as tested on bedside examinations). However, visual neglect in far space, as well as tactile, auditory, motor, and olfactory neglect have been described [10,11]. In this issue, Aimiola and colleagues provide a novel investigation of near and far space. Heidler-Gary et al. report a double dissociation between left spatial motor extinction and left body motor extinction in individuals with acute stroke with somewhat distinct sites of lesions. Pitteri and colleagues demonstrate that tactile neglect can be distinguished from hemianesthesia with careful assessment.

Neglect frequently co-occurs with a variety of other cognitive deficits (such as anosognosia, impaired empathy, and aprosody), likely because the neural networks that support spatial attention or the cognitive processes that underlie spatial attention tasks used to test for hemispatial neglect overlap with those that support task switching, recognition of one’s own deficits, recognition of facial expression and vocal intonation, theory of mind, and so on. Kleinman and colleagues report an investigation of perseveration versus hemispatial neglect, and the lesion sites associated with each in acute stroke. Invernizzi et al. describe a study of somatoparaphrenia and anosognosia for hemiplegia. Finally, in a Clinical Note, Carota et al. report a patient with anosognosia, attention blindsight, and left neglect. The special issue also includes an important paper by Ishizaki and co-workers of impaired visual-spatial attention in Alzheimer’s Disease, which shows how a symmetric neurodegenerative disease results in impaired shifting of visual spatial attention, but not hemispatial neglect. Together, these novel studies of neglect and related disorders provide new insights into brain-behavior relationships on the basis of detailed analysis of patient performance (and in many cases, their lesion sites).

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References
