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Hemispatial neglect

To the Editor: Vallar et al¹ report three right-brain-damaged patients with left homonymous hemianopia or hemianesthesia who manifested left spatial hemineglect and had normal somatosensory or visual evoked potentials recorded over the right hemisphere. Three left-brain-damaged patients without neglect showed no evoked response to contralateral visual or somatosensory stimuli. All three right-brain-damaged patients with preserved evoked potentials denied their defects, whereas the left-brain-damaged patients with no evoked potentials were aware of their defects. Vallar et al draw a conclusion that “evoked potentials may be used for distinguishing primarily sensory hemianopia and hemianesthesia from visual and somatosensory deficits due to neglect,”¹ ie, that in neglect the evoked potentials are present over the involved (right) hemisphere. This generalization based on six patients markedly differs from our report in this journal² that somatosensory evoked potentials were absent in nine patients with anosognosia for left hemiplegia and in one patient with anosognosia for right hemiplegia. This paper was not cited by Vallar et al. The absence of neglect in their three patients with left-brain damage may not be related to “an absence of sensory processing” since “sensory processing” has also been absent in right-brain-damaged patients with neglect.² The reason anosognosia and neglect are more common in right-hemisphere lesions remains elusive.

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Reply from the Authors: We thank Dr. Green for his interest in our article¹ and for the opportunity he gives us to clarify our views about neglect and denial of illness. The following summarizes our response to the main points of his letter.

(1) According to Dr. Green, our conclusion is that “in neglect the evoked potentials are present over the involved (right) hemisphere.” This is an overstatement and an undue generalization drawn from our discussion. We have shown that right-brain-damaged patients with visuospatial neglect, hemianopia, and hemianesthesia may have normal somatosensory or visual evoked potentials (SEPs, VEPs) to stimuli they fail to perceive consciously. We suggest that in these patients, hemianopia and hemianesthesia are likely to be manifestations of neglect, since primary sensory processing, as assessed by SEPs or VEPs, is preserved. These modality-specific perceptual components of the neglect syndrome (somatosensory and visual hemi-inattention) may be distinguished from spatial neglect, as assessed by exploratory tasks.³ Our results do not imply, however, that in *all* patients with visuospatial neglect hemianopia and hemianesthesia are entirely produced by hemi-inattention, primary sensory processing being spared. In some neglect patients, somatosensory and visual half-field deficits may be due, wholly or in part, to a primary sensory disorder. For instance, in neglect patients with lesions involving also the right sensory occipital regions, left hemianopia is likely to have an important sensory component and VEPs should be abnormal. By contrast, in our patients 1 and 3, who had left hemianopia and normal VEPs, the primary visual cortex was largely spared.

(2) The article by Green and Hamilton² concerned SEPs in patients with denial of hemiplegia. This was not the topic of our study,¹ which involved a different, although related, issue: VEPs and SEPs in patients with visuospatial neglect, hemianopia, and hemianesthesia. In 10 hemisphere-damaged patients with denial of hemiplegia and a severe contralateral sensory loss, Green and Hamilton² found absent SEPs on stimulation of the contralateral median nerve. In our series,¹ patients 2 and 3, who denied their left motor deficits, had a profound left somatosensory loss but normal SEPs. In the series of 97 right-brain-damaged patients of Bisiach et al,⁴ denial of left hemiplegia was frequently associated with somatosensory deficits, but instances of double dissociation (ie, patients without somatosensory deficits who denied their hemiplegia, and patients with hemianesthesia without denial of hemiplegia) were also found. Taken together, these observations^{1,2,4} suggest that denial of hemiplegia cannot be explained, as argued by Green and Hamilton,² in terms of defective processing of somatosensory stimuli delivered to the affected side. Denial of hemiplegia, as well as of hemianopia and hemianesthesia, reflects instead the more central failure of the monitoring function performed by areas such as the inferior parietal lobule.¹

(3) Green writes that the lack of neglect in our three left-brain-damaged patients (nos. 4, 5, and 6) may not be related to an absence of sensory processing. We entirely agree with Green. Visuospatial neglect may occur without hemianopia or hemianesthesia⁵ and, therefore, cannot be explained in terms of primary sensory deficits. Disorders such as hemianopia and hemianesthesia, which are typically regarded as primary sensory defects, may however be *in some patients* a manifestation of neglect. This was, in a nutshell, the message from our study.

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Correction

The name of Mark A. Ross, MD, Iowa City, Iowa, should have accompanied that of A. Arturo Leis, MD, as one of the signatories of the “To the Editor” portion of the “Psychogenic seizures” correspondence published in the May issue (*Neurology* 1992;42:1128-1129). The editors apologize for the error.

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Psychogenic seizures

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