

Comment on “Electrophysiological Correlates of Conscious Vision: Evidence from Unilateral Extinction” by Marzi, Girelli, Miniussi, Smania, and Maravita, in *JOCN* 12:5

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In their paper “Electrophysiological correlates of conscious vision: Evidence from unilateral extinction,” published in the September 2000 issue of this journal, Marzi et al. report a single-case study of a patient suffering from marked unilateral extinction after right-hemispheric lesion. P1 and N1 components of the potentials evoked by peripheral light stimuli (left, right, bilateral) were measured. These components were missing with left-side stimuli of bilateral pairs when these left-side stimuli were extinguished (i.e., when gone unnoticed by the patient). The authors conclude that visual extinction is a process that involves relatively early stages of visual perception.

This is certainly an interesting paper but is, on the other hand, a single-case study, and no data are provided from healthy controls. The results should, therefore, be evaluated in the light of other, previously published, event-related potential studies on visual extinction. From Marzi et al.’s treatment, one might get the impression that there have been no relevant studies so far. In fact, there are at least three such studies, one of which is directly pertinent to the present report. These three papers are:

(1) An early study by Lhermitte et al. (1985). Admittedly, these authors used only unilateral stimulation and measured the P300 component only, so the paper is not perfectly related to the present paper.

(2) A very recent study by Deouell et al. (2000). Admittedly, this paper is too recent to be included in Marzi et al.’s report, and furthermore, auditory stimuli were used. Nevertheless it is of much interest: Measuring the mismatch negativity evoked by deviant tones presented on the left or right side, the authors found smaller amplitudes with left-side deviance (compared to right-side deviance and to healthy controls) and concluded that the patients’ preattentive processing of left-side stimuli is disturbed. Of interest, these abnormal MMN amplitudes were obtained in the presence of normal N1 amplitudes to sounds on the left, which might be seen to be different from Marzi et al.’s result but, of course, the difference in stimulus modality precludes any detailed comparison of results.

(3) A study by Verleger et al. (1996). In that paper we measured potentials evoked by cues and targets in

Posner et al.’s (1984) visual cueing task from 10 patients with lesions of the right parietal cortex and from age-matched healthy subjects. In essence, this task is a computerized version of the visual extinction test as used by Marzi et al. (2000), with the sequences left cue–right target, left cue–left target, and right cue–right target being not extinguished and therefore comparable to those bilateral stimuli in Marzi et al., which were not extinguished, the sequence right cue–left target leading to response delays and therefore comparable to those bilateral stimuli in Marzi et al., which were extinguished. The patients’ N1 component evoked by left-side cues was reduced at the right parietal recording site, suggesting a general impairment in processing left-side visual input. Of more importance, the patients’ EEG potentials evoked by the critical combination of right cue/left target differed in two features from the other sequences: Their mean amplitude 160–280 msec after target onset (“Nd”) was less negative than with other combinations of cue and target, and the following frontal P300 was enhanced. We concluded that the Nd reduction might be an on-line measure of patients’ momentary decrease of attention for the left hemifield, while the frontal P300 might reflect the patients’ attempts of reorienting. Unfortunately, these findings cannot be directly compared to the data published by Marzi et al. because we measured these components at midline sites, due to the affection of lateral sites by artificial potentials generated by stimulus-induced horizontal saccades, whereas Marzi et al. displayed and analyzed lateral sites only.

Somewhat like Marzi et al., we obtained an N1 reduction to “extinguished” left targets (i.e., those preceded by right cues) compared to “not extinguished” left targets (i.e., those preceded by left cues) but this result was not significant, unlike in Marzi et al. This different result might be due to worse signal/noise ratios in our data: 60 trials per condition were presented in our study, about 500 trials in Marzi et al. On the other hand, the one patient studied by Marzi et al. might be less representative than the sample of 10 patients studied by us.

Of further interest is that there was no contralateral enhancement of N1 at the lesioned side in our data, i.e., N1 amplitudes evoked by left targets were not larger than N1 amplitudes evoked by right targets at the right occipital site O2. This might appear to be pathological but in fact the same was true for the control group's right occipital site, and indeed this right–left asymmetry (marked N1 enhancement by contralateral stimuli at the left hemisphere, no such enhancement at the right hemisphere) is not uncommon in the literature (e.g., Yamaguchi et al., 1994). This result underlines the need for data from healthy participants as a control.

To conclude, it would have been nice, and indeed appropriate, if an evaluation of Marzi et al.'s results in comparison to previous results, like the one attempted here, would have been provided by Marzi et al. themselves, especially in view of the fact that the literature on event-related potentials in visual extinction is so scarce that it can be easily overviewed.

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