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Visual evoked potentials are affected by trunk rotation in neglect patients

Donatella Spinelli[1,2,CA] and Francesco Di Russo[2]

[1]Dipartimento di Psicologia, Universita degli studi di Roma 'La Sapienza', Via dei Marsi 78, 00185 Rome; [2]IRCCS Clinica Santa Lucia, Rome, Italy

[1,CA]Corresponding Author and Address

STEADY-STATE visual evoked potentials (VEPs) were recorded in four patients with unilateral visuo-spatial neglect, stimulating either the left or the right hemifield. In the standard condition (head and body oriented straight ahead towards the stimulus) the left hemifield VEP was delayed. When the body was turned to the left, however, the two hemifield latencies were comparable. These results were confirmed with the transient VEP technique. No effect of trunk rotation was observed in a group of patients with left brain damage and without neglect. The results imply that the sensory afferents from neck muscles might restore the altered occipital activity and suggest that the same conditions which modulate neglect modulate VEPs latencies.

Key Words: Neglect; Visual Evoked Potentials; Latency; Trunk rotation

Introduction

Unilateral neglect syndrome is frequent after right hemisphere damage: patients tend to ignore sensory information on the left side.[1] Experimental data show that some conditions, such as neck torsion and vibration of neck muscles, temporarily reduce the neglect.[2–4] Patients with neglect perform poorly in tasks requiring identification of figures presented tacthistoscopically to the left visual field and they scan the left space poorly, producing very slow and infrequent saccades. Vibration of neck muscles and neck torsion produce temporary improvement in both identification and saccades;[2,3] the number of identifications made rose from 65% to 80% and the saccadic reaction time to left hemifield targets reached values comparable to those of the other hemifield. The recovery effect was specific, being observed only for trunk rotation to the left side (contralateral to the lesion) and for vibration of left posterior neck muscle.[2,3]

The evoked potentials of neglect patients have a longer latency in the left than in the right visual field.[5] These data were acquired in the standard condition, i.e. with patients sitting in front of the screen, looking straight at the visual display. The purpose of the present study was to examine whether the same manipulations which affect the unilateral visuospatial neglect also affect the latencies of evoked potentials. If VEP characteristics were entirely retinotopic, no effect of rotation would be expected, since there is no difference between the retinal representation of the two conditions. On the other hand, an effect of neck torsion would suggest that non-visual information might modulate the VEP.

Patients were tested both in the standard condition, with the head and body oriented straight towards the screen, and with the trunk rotated to the left. In this case, the head was made to face the monitor by turning the neck to the right, stretching the neck muscles. The main experiment was conducted with steady-state VEPs in four patients with right brain damage and neglect, comparing the standard condition with the trunk rotation condition. As a control condition, four patients with left brain damage but without neglect were tested with the trunk rotated to the right. An additional experiment was run with the neglect group, using the transient VEP technique to confirm the data recorded with the steady-state VEP technique.

Materials and Methods

Subjects: Eight brain-damaged patients were tested. Four were had right brain and unilateral neglect and four had left brain damage (LBD) without neglect. The site of the lesion was assessed in seven cases by CT scan and in one case by neurological examination. The presence of neglect was assessed by scores reported in four standard neuropsychological tests.[6] Visual field was measured by kinetic Goldmann perimetry and was intact in all patients. The characteristics of the patients are given in Table 1. Informed consent was obtained, after the nature of the study had been explained.

Stimuli and procedure: The stimuli were vertical sinusoidal gratings generated by framestore (Cambridge Research VSG) and displayed on the monitor

(Barco CDCT 6551). Contrast was 32%, spatial frequency 0.6 cycles per degree, and mean luminance 16.5 cd m^{-2}. The contrast of the stimulus was reversed at 15 temporal frequencies (4–11 Hz, step 0.5 Hz). The grating was presented in the left (LVF) or in the right (RVF) visual field. The stimulus subtended 24° × 20°; the fixation point was 1.5° lateral to the edge of the stimulus. Two conditions were tested: in the baseline condition the trunk and head were facing the monitor straight ahead; in the trunk rotation condition; the trunk was rotated 45° to the left; the head faced the monitor by turning the neck to the right. Patients were instructed to maintain steady fixation of the fixation point, and to sit with the trunk and shoulder sticking to the back of the chair. No restriction was used. In a control experiment, patients with damage to the left hemisphere damage were tested; in this case the trunk rotation was to the right. VEPs were recorded in two sessions: in the first session the baseline and the trunk rotation conditions were tested at seven temporal frequencies (4–7 Hz); in the second session the baseline and the trunk rotation conditions were tested at eight additional temporal frequencies (7.5–11 Hz). Each session lasted about 40 min. Breaks were allowed and instructions were repeated whenever they were felt to be useful. An experimenter monitored eye movement visually, and interrupted data collection if fixation deviated.

Electrophysiological recordings: VEPs were recorded with electrodes placed 3 cm above the inion (Oz) and at the vertex (Cz) with ground half way between. Signals were amplified (50 000-fold), band-pass filtered (1–100 Hz) and averaged (200 sums) by computer after artifact rejection. To minimize contamination by eye blinks or gross eye movements, the computer rejected single sweeps over a threshold voltage. The computer averaged the EEG in synchrony with the stimulus contrast reversal rate[12] and calculated the amplitude and phase of the second harmonics of the averaged signals by discrete Fourier analysis. At each temporal frequency, 200 repetitions of the stimulus were collected. Details of the technique can be found elsewhere.[5]

Data analysis: The latency of the hemifield steady-state VEPs was estimated by measuring phase as a function of temporal frequency, and calculating the slope of the curve (see Refs 5, 7–9). Latencies were submitted to an ANOVA, with group (2 levels) as between-subjects factor and condition (2 levels) and visual hemifield (2 levels) as repeated factors.

Transient VEPS: An additional experiment was performed in three patients with neglect, using transient VEPs. The stimuli were the same as in the steady-state VEP, except that they were square-wave temporally modulated at 1 Hz. Time analysis was 300 ms; responses to 300 repetitions of the stimulus were recorded in each condition. The P100 component of transient VEPs was analysed as its amplitude and latency are related to early stages of visual processing and are well correlated with amplitude and latency of the second harmonic of steady-state VEPs.[10,11] Latencies of the P100 component were submitted to an ANOVA, with condition (2 levels) and visual hemifield (2 levels) as repeated factors.

Results

Results are presented in Figure 1. For the neglect group, the mean latencies of LVF and RVF in the standard condition were different, the LVF response being delayed by 38 ms (top left). In the trunk rotation condition, however, the latencies were similar, the delay being reduced to 7 ms (bottom left). Trunk rotation had no effect in patients with left brain damage (right part of Fig. 1). In these patients the latencies were always similar and the differences between the latencies of the two hemifields very small: 3 and 5 ms. Inspection of individual latencies data Figure 2 confirms the average results.

ANOVA showed that the effects of all main factors

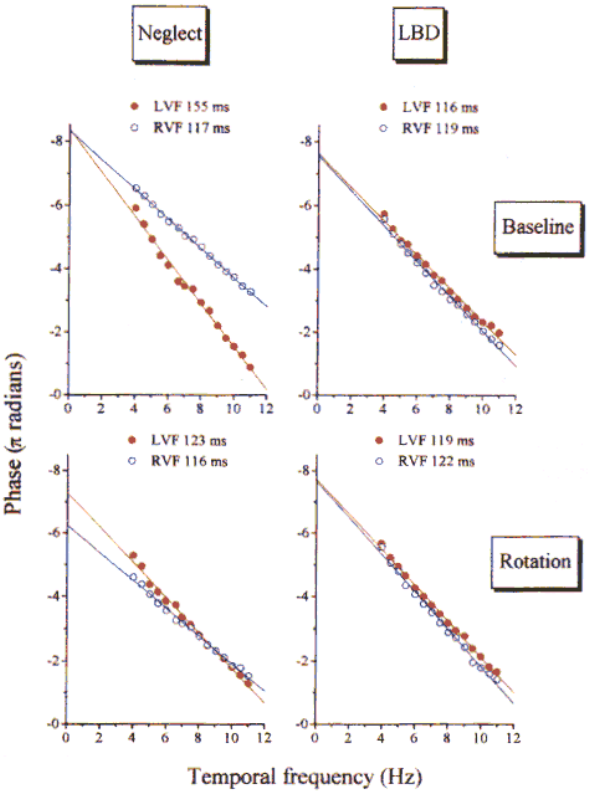

FIG. 1. Mean phase plots for the two groups under the two conditions tested. In all cases, phase varied linearly as a function of temporal frequency ($r > 0.85$). The mean estimated latencies for each hemifield are also reported in the figure in ms.

Table 1. Characteristics of patients

	Case	Sex	Age	Vision	Clinical history
Neglect group	1	F	59	7/10	Tested 100 months after CVA involving right temporal and parietal lobes and part of n. caudatus; left hemiparesis.
	2	F	70	9/10	Tested 9 months after CVA involving right temporal and parietal lobes; left hemiparesis.
	3	M	63	6/10	Tested 12 months after CVA involving right temporal and parietal lobes; left hemiparesis.
	4	F	56	8/10	Tested 36 months after CVA following surgical treatment of a meningeal cyst; left hemiparesis.
LBD group	1	M	74	4/10	Tested 15 months after CVA involving left frontal, temporal and parietal lobes; right hemiparesis, global aphasia.
	2	M	50	8/10	Tested 6 months after CVA involving left temporal lobes; right hemiparesis, non-fluent aphasia.
	3	F	67	6/10	Tested 5 months after CVA involving left temporal and parietal lobes; right hemiparesis, global aphasia, apraxia.
	4	M	45	6/10	Tested 4 months after CVA involving temporal and parietal lobes; right hemiparesis, non-fluent aphasia.

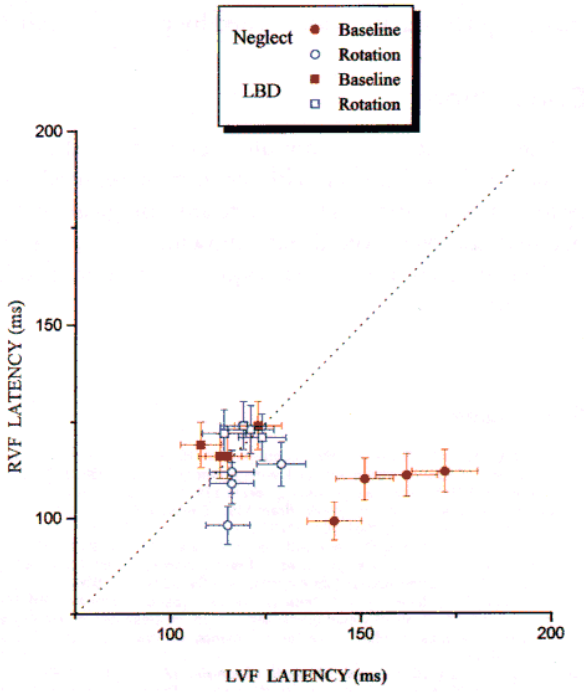

FIG. 2. Scatter plot of individual latencies from stimulation of the right and left hemifields. LVF latencies plotted against the RVF latency. If the latencies of the two hemifields were similar, the experimental points should cluster around the bisecting line, which represent equilatency, distributing roughly equally on either side of it. This occurs for patients with left brain damage in both conditions and for patients with neglect in the trunk–rotation condition. In the baseline condition, the individual data of the neglect patients (●) are to the right of the line, showing longer latencies for the left visual field stimulation. Horizontal bars indicate the individual standard errors of LVF latency, vertical bars indicate the individual standard errors of RVF latency.

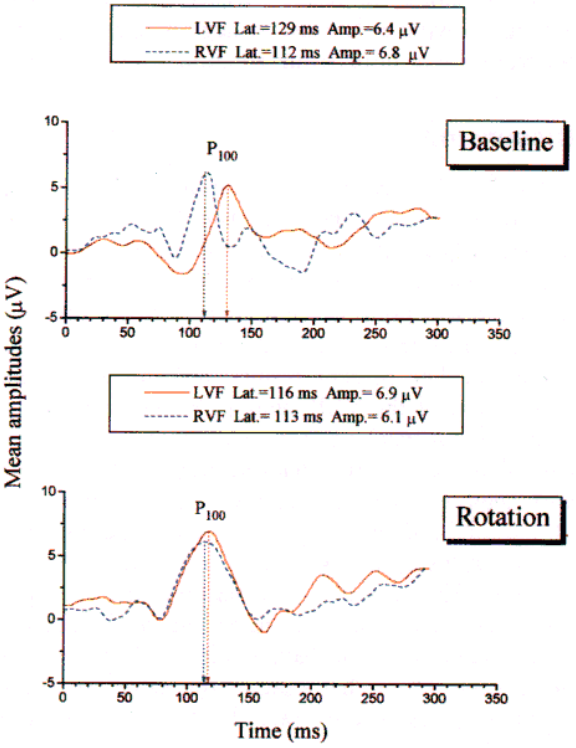

FIG. 3. Mean transient VEPs for three patients with neglect in baseline and trunk rotation conditions. The straight and dashed lines show the LVF and RVF responses, respectively. The vertical lines indicate the P100 latencies.

(group, condition and visual hemifield) were significant ($p < 0.05$) and the interaction among all factors was significant ($F(1,6) = 53.42$; $p < 0.001$). Post-hoc comparison (Tukey HSD test) showed that the mean VEP latency to LVF stimuli in the baseline condition for patients with neglect (155 ms) was longer than all other latencies ($p < 0.0005$). All these other means did not differ significantly from each other.

The VEP delay in patients with neglect was not associated with conspicuous modification of amplitudes to LVF stimuli: there was a tendency to record responses of lower amplitude for the neglected hemifield, particularly at the higher temporal frequency, but differences between hemifields were not statistically significant. Further, the trunk rotation did not affect the VEPs amplitudes.

Three patients with neglect were also tested in one additional session with transient VEPs (one patient could not be recorded for technical reasons). The results (Fig. 3) were consistent with previous results. The interaction between condition and visual field factors was significant ($F(1,2) = 19.75$; $p < 0.05$); and post-hoc comparison confirmed that the P100 latency for the left visual field stimuli in the baseline condition was longer than all other latencies ($p < 0.05$), which did not significantly differ from each other. The value of the P100 latency measured with the transient VEP technique was very close to that estimated by phase regression in the steady-state experiment (10 ms difference between the two means). Moreover, a positive high correlation between the two latency values was present (r-Pearson coefficient = 0.996; dd.f = 2, $p < 0.005$). In summary, the VEP delay for stimuli presented in the left visual field in patients with neglect is reduced by head–trunk rotation. This electrophysiological result parallels the behavioural data in showing a decrease in neglect as a temporary consequence of head–trunk rotation.

Discussion

The result that VEPs are influenced by trunk rotation is at first glance surprising: VEPs are highly dependent on visual parameters (such as contrast, spatial frequency, localization in the visual field etc.) and from this point of view there was no difference between the standard condition and the trunk rotation condition. The present results indicate extra-retinal modulation of the VEP latency. However, it should be noted that trunk rotation was effective only in the particular case of the delayed response of patients with neglect. Indeed no effect was observed in the case of left brain damaged patients and experiments in normal subjects do not show any effect of trunk rotation. This suggests that sensory afference produced by torsion becomes important in the pathological condition of neglect, while it is irrelevant in other cases (note that exactly the same pattern of results was observed for saccadic delay by Karnath *et al*[2]).

Discussion on the neural mechanism underlying the phenomenon of VEP delay is open;[5] in any case, the sensory afference from neck muscles seems to restore the altered occipital activity. The reduction in delay could be mediated by various mechanisms. Head–trunk rotation might produce a general activation of the right hemisphere,[12] including occipital activity. On the other hand, the sensory input by neck muscles might specifically affect the visual cortex response through a direct subcortical pathway. In cats and monkeys, the striate cortex receives important projections from thalamic anterior intralaminar nuclei;[13] the activity of this region is associated with gaze orientation[12] and is possibly involved in mechanisms of attention, arousal and postural orienting.[15,16] These inputs may also be present in the human brain, but their contribution to the VEP would be demasked only in the pathological condition of neglect. Further, head–trunk rotation might specifically affect the activity of polymodal units in the lesioned parietal cortex, which is responsible for building up a representation of body and head position.[12,17,18] This representation would be displaced in neglect patients as a consequence of the lesion and trunk rotation would compensate for such a displacement.[2] The recovery of VEP latency observed in the present experiment would imply that occipital activity is modulated by parietal activity.

Conclusion

The VEP delay for stimuli presented in the left visual field in patients with neglect is reduced by head–trunk rotation. The electrophysiological result parallels the behavioural data showing a decrease in neglect as a temporary consequence of head–trunk rotation.

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