Electrophysiological Correlates of Conscious Vision: Evidence from Unilateral Extinction

Carlo A. Marzi and Massimo Girelli
University of Verona, Italy

Carlo Miniussi
Oxford University

Nicola Smania
Rehabilitation Unit Borgo Roma, Italy

Angelo Maravita
University of Verona and University College London

Abstract

■ To study the electrophysiological correlates of conscious vision, we recorded event-related potentials (ERPs) in a patient with partial unilateral visual extinction as a result of right-hemisphere damage. When, following bilateral presentations, contralesional stimuli were not perceived, there was an absence of the early attention-sensitive P1 (80–120 msec) and N1 (140–180 msec) components of the ERP response. In contrast, following unilateral presentations, or in those bilateral presentations in which contralesional stimuli were perceived (about 60%), these ERP components were present. These results provide novel evidence that extinction involves the stage of early focusing of attention and that the P1 and N1 components of visual ERPs are reliable physiological correlates of conscious vision. ■

INTRODUCTION

A major challenge in neuroscience is to characterize the neural processes specific to conscious experience. This is a daunting task when studying normal people but is more feasible in subjects in whom, as a consequence of brain damage, conscious and unconscious mental processes can be dissociated. A particularly interesting form of such dissociation is the phenomenon of unilateral ''visual extinction'' in which a stimulus presented to the visual hemifield contralateral to the damaged cerebral hemisphere can be detected in isolation but is lost when another stimulus is presented simultaneously to the opposite side of the visual field (Driver, Mattingley, Rorden, & Davis, 1997; Bisiach & Vallar, 1988). Interest in this phenomenon, which may concern other sensory modalities as well, stems from two characteristics: First, it is a dynamic condition, such that a given stimulus can access consciousness or not depending upon the presence of another concomitant stimulus. This makes this phenomenon amenable to well-controlled experimental investigations. Second, extinguished stimuli may undergo a considerable extent of unconscious processing in spite of being undetected (Marzi et al., 1996; Baylis, Driver, & Rafal, 1993; Berti et al., 1992; Volpe, LeDoux, & Gazzaniga, 1979), and this makes it possible to study the neural bases of implicit versus explicit processing. The most influential account of extinction posits that it represents an impairment of selective attention, manifesting itself in conditions of competition between the damaged and the intact hemisphere (Driver et al., 1997; Kinsbourne, 1993; Bisiach & Vallar, 1988; Karnath, 1988). A clear prediction follows from this account, specifically that sensory processes preceding the stage at which attentional focusing occurs should be preserved. Psychophysical evidence in accord with this prediction has been provided by experiments showing that in a patient with extinction, pre-attentive vision is preserved (Mattingley, Davis, & Driver, 1997). Once established that extinction spares the basic visual operations, however, it remains to be established at what subsequent stage in visual processing it might occur. A reasonable possibility is that such a stage concerns focusing of spatial attention. A considerable amount of work with event-related potentials (ERPs) (e.g., Hillyard & Anllo-Vento, 1998; Mangun, 1995) has established that there are early components of the ERP,

namely P1, a positive component peaking at 80–120 msec from stimulus onset and N1, a negative component peaking at 140–180 msec from stimulus onset, that can be modulated by spatial attention. Joint electrophysiological and metabolic brain imaging studies have thrown light on the location of the neural generators of these components (Heinze et al., 1994). P1 is thought to be generated in the extrastriate cortex and this is also the case for the most posterior N1 component. There are, in fact, more dorsal and frontal N1 components with parietal and frontal sources, respectively. If early attentional mechanisms are impaired, one might predict that the P1 and the most posterior N1 components should be abnormal during extinction. Alternatively, if extinction affects later processing stages, normal early ERP responses might still be present during this condition. Providing evidence in favor of one of these two alternatives would contribute important clues not only to an understanding of this relatively frequent neuropsychological impairment, but also to casting light on the neural bases of conscious vision. To accomplish this, we compared ERP responses to the same stimulus when perceived or when extinguished in a patient (R.M.) with left-unilateral extinction as a result of right-hemispheric damage. This particular patient was selected because her overall extinction rate was about 40% and this made possible a meaningful comparison between extinction and non-extinction trials.

The predictions derived from our design are as follows: each unilateral stimulus (ipsi- or contralesional) should yield a detectable response in both hemispheres since in extinction, single stimulus processing is supposed to be unaffected. As is well known, lateralized visual stimuli evoke a response in both hemispheres: Input is directly relayed through the visual pathways to the contralateral hemisphere and is indirectly transmitted through the corpus callosum to the ipsilateral hemisphere (e.g., Miniussi, Girelli, & Marzi, 1998). Bilateral stimuli have been shown to yield bihemispheric responses of shorter latency and higher amplitude than unilateral stimuli (Miniussi et al., 1998) in normal subjects. Therefore, we expect a similar effect in those bilateral trials in which the patient does not show extinction. On the contrary, during extinction trials in which the contralesional stimulus is perceptually lost, there are two opposite predictions: If extinction affects *early* visual processing, we ought to find abnormal P1 and N1 responses to the contralesional (left) stimulus in both the damaged right hemisphere and the intact left hemisphere during extinction with bilateral stimuli. This effect should be witnessed by a decrease in amplitude of the early ERP components for extinguished as opposed to non-extinguished bilateral presentations. If, however, extinction affects *late* processing stages only, then P1 and N1 should be normal while only later components, or no components at all, should be affected. In this case, the responses to bilateral stimuli should be similar during extinction and during correctly reported bilateral stimuli.

RESULTS

R.M. is a 57-year-old female with a right-hemisphere lesion involving parietal, temporal, and frontal areas as assessed by reconstructing the lesion with the method of Damasio and Damasio (1989), see Figure 1A. Brain damage was caused by a vascular accident which occurred about 1 year before testing. R.M. had an almost-complete sparing of the visual field (Figure 1B), and did not show any hemineglect, as assessed clinically and by Albert (1973) line cancellation test. Her only neuropsychological impairment was a stable, albeit partial, extinction of the contralesional stimulus during bilateral presentations.

The behavioral task included pressing of a centrally located key as fast as possible following brief presentation of a stimulus either to the right visual hemifield or to the left visual hemifield, or bilaterally. Immediately after the manual response, she was to verbally report the side and number of stimuli just seen. This enabled us to estimate her extinction rate, i.e., the percentage of bilateral presentations in which she reported having seen only the ipsilesional stimulus. We instructed the subject to report any instance of uncertainty as to the number and location of the stimuli.

R.M. detected almost all unilateral stimuli, whether presented to the ipsi- (99%) or to the contralesional (99%) hemifield, but reported only 61% contralesional (i.e., left field) stimuli during bilateral presentations. Errors were represented almost entirely by misses with very few localization errors. Differences in speed of responses among the various experimental conditions were analyzed statistically with a design similar to that used for the ERP data (see below) by means of a one-way ANOVA with Perceptual Report as factor. The analysis yielded a significant effect, $F(3, 16) = 17.37$, $p < .001$. Post hoc tests with Bonferroni correction showed that only the reaction time (RT) to contralesional stimuli (465 msec) was different from all other conditions (ipsilesional stimuli: 368 msec; bilateral-correct stimuli: 362 msec; bilateral with extinction: 390 msec). This slowing down of responses to unilateral-contralesional stimuli has been consistently found before (e.g., Smania et al., 1998; Marzi et al., 1996). Interestingly, the mean RT for correctly reported bilateral stimuli was not reliably different from that of bilateral trials with extinction and this shows that this patient, in contrast to other extinction patients reported before (Marzi et al., 1996), did not show an implicit speeding of RT for double versus single stimuli, the so-called redundant targets effect. One should note, however, that the present patient did not show a reliable explicit redundancy

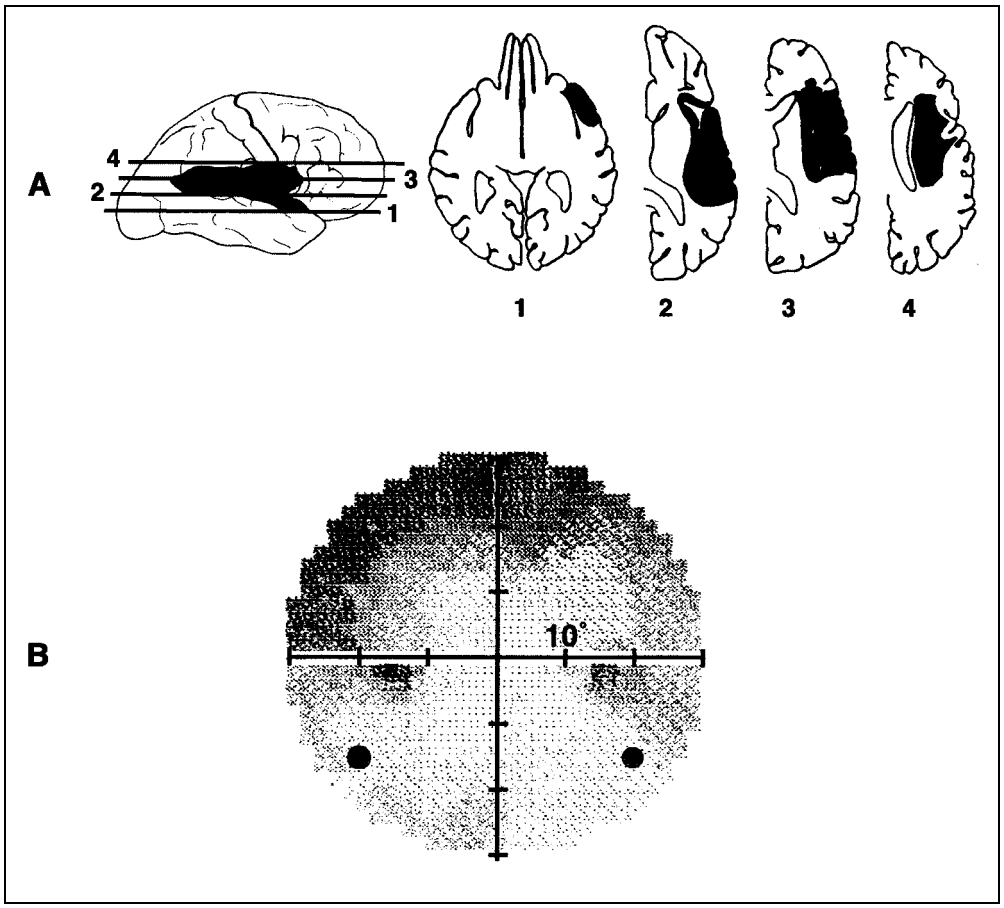

Figure 1. Reconstruction of the magnetic resonance imaging (MRI) of patient R.M.'s brain lesion. (A) At the top-left is shown a lateral view of the right hemisphere with the lesion extent drawn in black. The lines represent the axial sections indicated by progressive numbers from bottom to top. The lesion involved temporal (Brodmann's areas — BA 38, 22, 41, 42), parietal (BA 1, 2, 3), frontal (BA 6 inferior) lobes, and the corona radiata. (B) Composite computerized perimetry obtained by superimposing the field assessments of the left and right eyes. Black circles indicate the visual-field positions of stimulus presentation. (Note: Circle size is not proportional to the actual stimulus size).

gain and therefore, any conclusion as to the presence of an implicit summation between the extinguished contralesional and the preserved ipsilesional stimulus cannot be drawn.

While the patient was performing the task, we recorded ERPs to the visual stimuli. As mentioned above, the crucial comparison was that between responses to bilateral stimuli when the contralesional stimulus was extinguished, and responses to the same stimuli when they were both correctly reported. We focused our attention onto the two attention-sensitive early components of the visual ERP, namely P1 and N1 and on the electrode locations where the signal-to-noise ratio was the highest. Figure 2 shows, for a parietal electrode site in the right (P4) and in the left (P3) hemisphere, the ERP responses in the various conditions of stimulus presentation namely, bilateral-correctly reported stimuli, bilateral presentations with extinction of the contralesional stimulus, unilateral-ipsilesional and -contralesional stimuli.

Right Hemisphere

In the right (damaged) hemisphere (see Figure 2, electrode P4), there was a clear-cut response to bilateral-correctly reported stimuli (thick continuous line) with well-recognizable P1 and N1 components. In contrast, the response to the same stimuli during contralesional extinction (thin continuous line) was absent as far as the P1 and N1 components were concerned. The direct response to the unilateral-contralesional stimulus (thick dotted line) had recognizable, although delayed, P1 and N1 components, while the indirect (commissural) response to the ipsilesional stimulus (thin dotted line) lacked these components altogether.

The important result here was that the responses elicited by the same physical stimuli varied according to the perceptual report of the patient. During correctly reported bilateral stimuli, the P1 and N1 components were clearly detectable while they were absent for bilateral presentations in which the contralesional stimulus was extinguished. This effect was present at all electrode sites in the right hemisphere where there was a detectable response to the visual stimuli used in the task. The absence of reliable P1 and N1 components in the extinction condition made it impossible to statistically evaluate the above effect. It is important to note that despite the absence of P1 and N1, during extinction there was a clear later positive component with a peak latency at about 320 msec, which resembles the well-known P300 component and is presumably related to

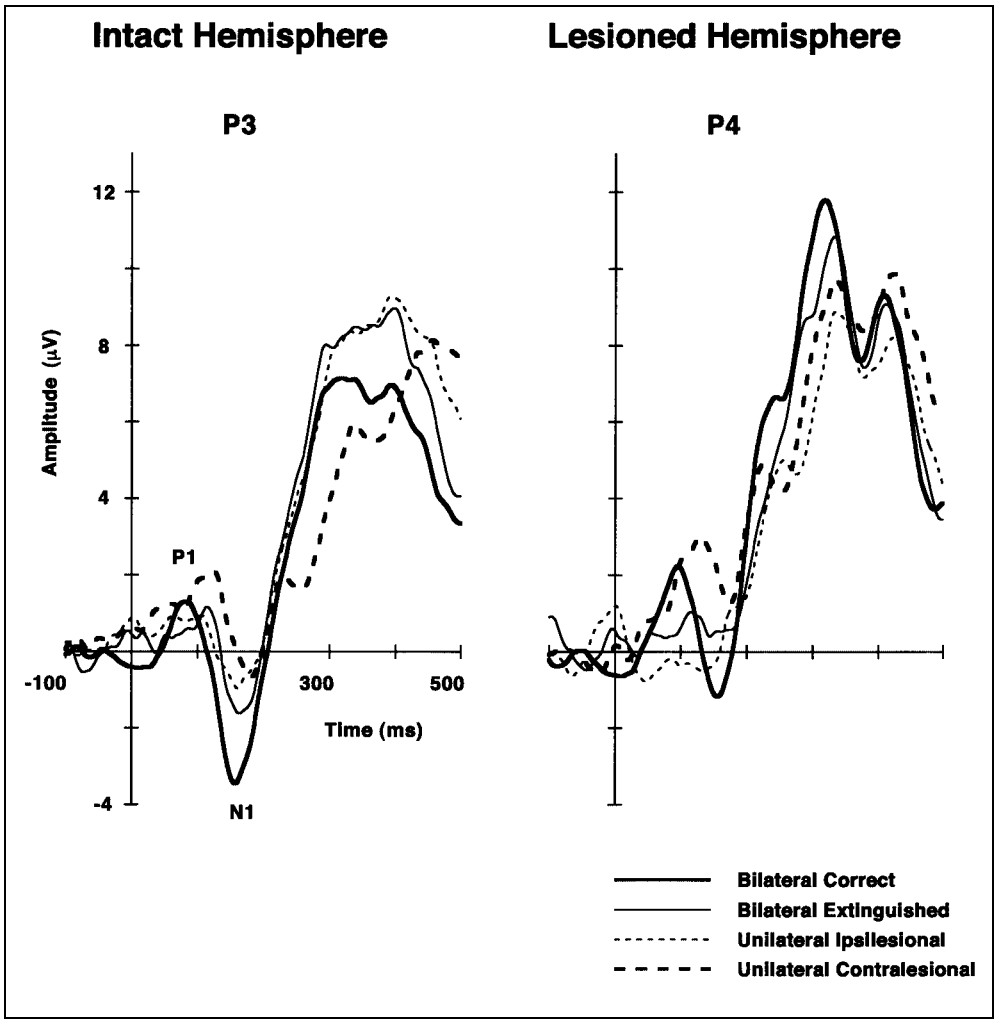

Figure 2. ERP waveforms recorded in parietal sites of the right-damaged hemisphere (P4) and of the left-intact hemisphere (P3) under conditions of bilateral (correctly detected and with contralesional extinction) or unilateral (ipsilesional and contralesional) stimulus presentation. Negative voltages are plotted down.

detection of a task-relevant stimulus (Coles & Rugg, 1995). A similar sparing of a parietal P300 response (P3b) in cortically damaged patients with prefrontal, temporal parietal or lateral parietal cortex lesions has been described recently (Knight, 1997). This sparing of the P300 component indicates that the absence of P1 and N1 cannot be explained by any sort of recording artifact occurring during extinction since this should have affected all ERP components. The presence of a delayed response to contralesional stimuli is in agreement with the RT data showing that in extinction patients, although single stimuli can be perceived, they are nonetheless responded to more slowly than ipsilesional stimuli (Smania et al., 1998; Marzi et al., 1996). This suggests that in the contralesional hemifield, visual processing is not entirely normal even with unilateral stimuli. The abnormal response to ipsilesional stimuli is somewhat puzzling; a reasonable explanation might be that the stimuli presented to the ipsilesional right hemifield must be relayed to the ipsilateral right hemisphere through the corpus callosum and our previous work (Marzi et al., 1997) has shown that in patients with right-parietal lesions, more than in those with left lesions, callosal transmission is severely impaired.

Left Hemisphere

The dramatic difference between ERP responses to extinguished and correctly reported bilateral stimuli was apparently attenuated in the left hemisphere where the response to bilateral stimuli during extinction (thin continuous line) was clearly present albeit of a smaller overall amplitude than that to normally perceived stimuli (thick continuous line) (see Figure 2, electrode P3). The responses to single-ipsilesional (thin dotted line) and contralesional (thick dotted line) stimuli were of roughly similar latency and amplitude as the response to bilateral-extinguished stimuli. The crucial difference between responses to bilateral stimuli following normal detection and following contralesional extinction was statistically evaluated by two separate omnibus ANOVAs using the mean amplitude of the P1 and N1 components, measured in

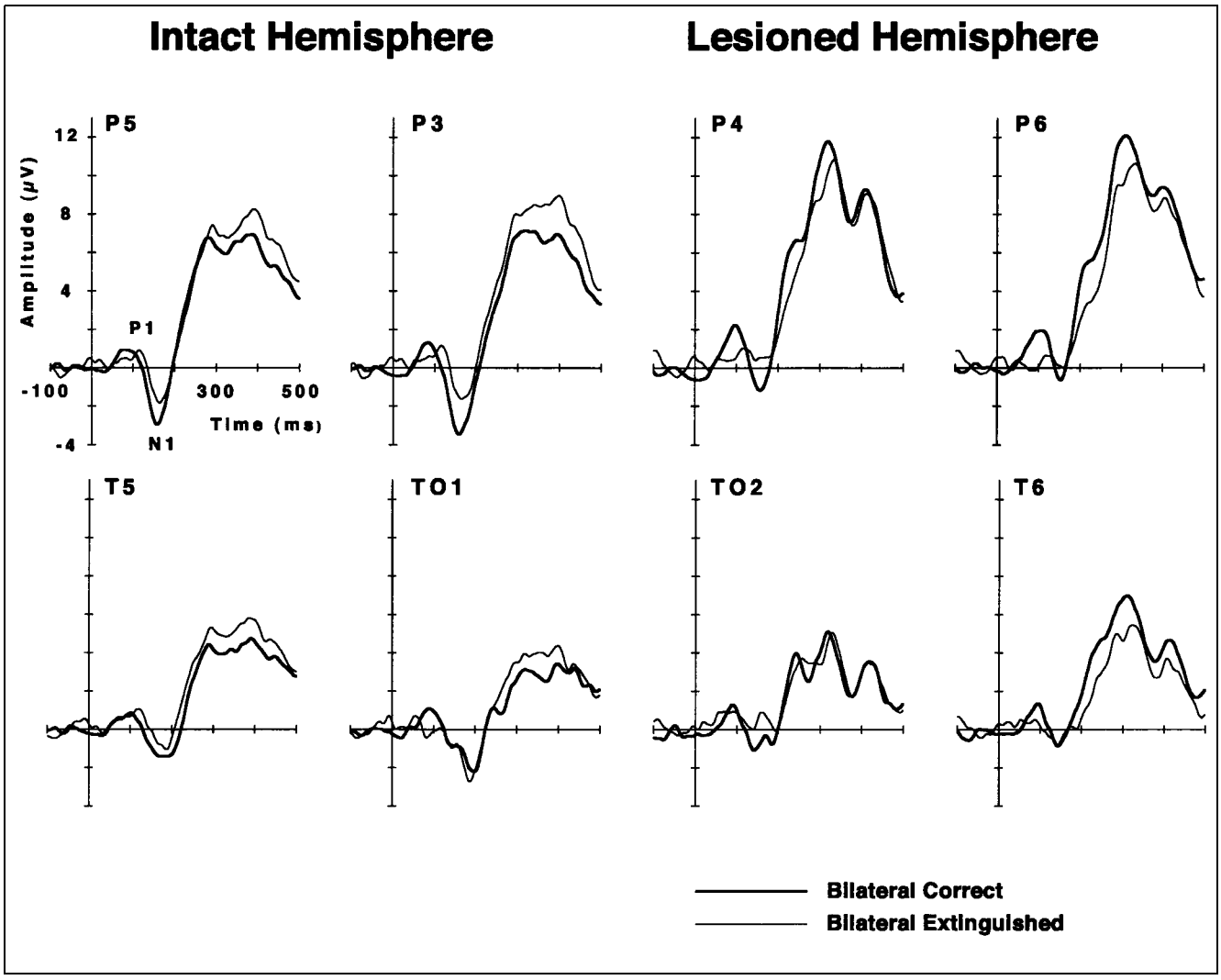

Figure 3. ERP waveforms recorded at the electrode locations in the right (damaged) and in the left (intact) hemisphere which were included in the ANOVA. Bilateral-non-extinguished trials: thick line; Bilateral trials with unilateral extinction: thin line. Negative voltages are plotted down.

five blocks in which the two experimental sessions were divided off-line. Two within-subjects factors were used: perceptual report (bilateral-correctly reported stimuli, bilateral-unilaterally extinguished stimuli, unilateral-ipsilesional, and unilateral-contralesional stimuli), and electrode site (P3, P5, T5, and TO1). This analysis yielded a significant effect of the main factor perceptual report for N1, $F(3, 4) = 4.51, p < .05$ (p value was corrected by the Greenhouse–Geisser epsilon for nonsphericity, Jennings & Wood, 1976), while it was far from significance for the P1 component $F(3, 4) = 0.26$, n.s. Therefore, additional separated ANOVAs were carried out for the N1 component only. These analyses revealed that the response to bilateral-unilaterally extinguished stimuli was significantly smaller than that to bilateral-correctly reported stimuli $F(1, 4) = 12.06, p < .03$, but that the former was not different from the response to unilateral-ipsilesional stimuli $F(1, 4) = 1.38, p > .05$. This means that even in the intact left hemisphere, there was no reliable response to the contralesional stimulus during extinction and that in this condition, the bulk of the response to bilateral stimuli was in fact represented by a response to the ipsilesional stimuli alone. Finally, as expected, there were reasonable, although somewhat delayed, responses to single stimuli, including those to stimuli presented to the contralesional (left) hemifield, which, when recording from the left hemisphere, represents the ipsilateral hemifield indirectly connected through the corpus callosum. This is not in contrast with the lack of commissural responses invoked to explain the absence of ipsilateral responses to single stimuli in the right hemisphere since this effect is bound to be more pronounced in the lesioned than in the intact side (Marzi et al., 1997).

Finally, Figure 3 shows a composite picture of the recordings from the left hemisphere's electrode locations included in the ANOVA, as well as from the corresponding locations for the right hemisphere. One can realize from inspection of this figure that the effects

described in detail for P3 and P4 were present in the nearby locations as well.

DISCUSSION

The main thrust of this study is represented by the evidence that during extinction, the perceptually lost stimulus does not evoke any cortical response either in the damaged or in the intact hemisphere. A crucial control condition was represented by non-extinguished stimuli, which yielded clear cortical responses in both the damaged and the intact hemisphere. In the light of the predictions described in the Introduction, the present results are not in keeping with the idea that in extinction, only access to conscious experience is impaired. Rather, they show that the earliest attention-

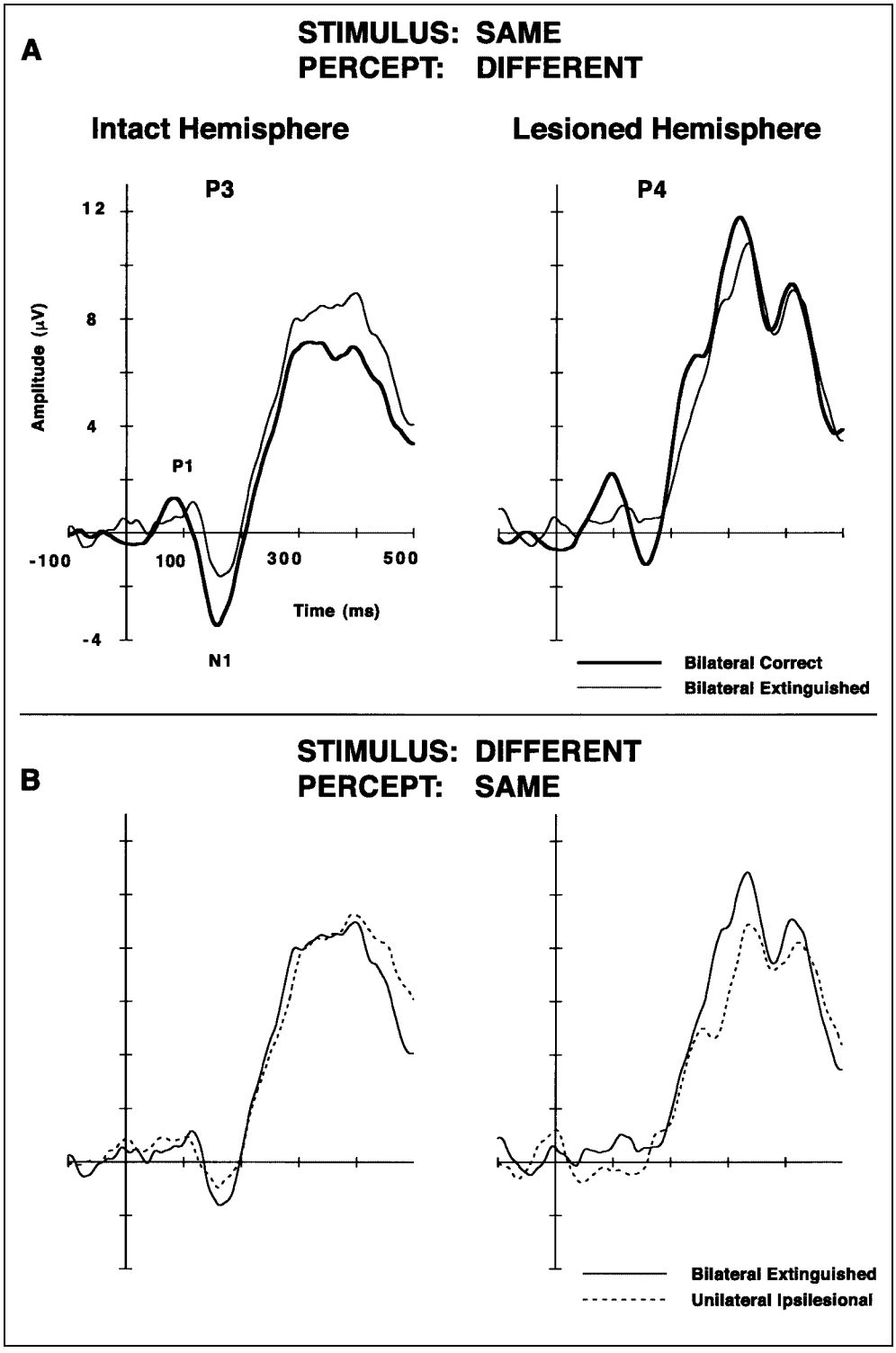

Figure 4. ERP waveforms recorded in the right-damaged hemisphere (P4) and in the left-intact hemisphere (P3) parietal sites. (A) Comparison between physically identical stimuli yielding a different perceptual report, i.e., correctly reported bilateral stimuli versus extinguished stimuli. Clearly, the two responses are widely different in P4 with a lack of early components in the extinction conditions and a good response during correct detection of bilateral stimuli. Such a difference is present, albeit less dramatically, in P3. (B) Comparison between physically different stimuli yielding the same perceptual report, i.e., bilateral-extinguished stimuli versus unilaterally presented ipsilesional stimuli. In the damaged hemisphere (P4), the ERP response is equally poor in either condition. In the intact hemisphere (P3), the responses are very similar although during extinction there is a slightly larger amplitude of P1 and N1 as compared to unilaterally presented stimuli. Negative voltages are plotted down.

sensitive ERP components are missing and therefore, that extinction might occur because of impaired early spatial-attentional mechanisms. Another aspect of the present finding, which has important general implications, is that the absence of these components is incompatible with conscious vision. Hence, our study adds further important evidence to that recently reviewed by Frith, Perry, and Lumer (1999) on patients in various abnormal states. This is clearly illustrated in Figure 4A and B showing that the ERP responses are correlated with the perceptual report of the patient rather than with the actual physical stimulation. Figure 4A shows a comparison between responses to the same stimulus (i.e., a pair of flashes) during different perceptual reports of the patient, while Figure 4B shows a corresponding comparison between responses to different stimuli (i.e., one or two flashes) yielding the same perceptual report.

As to the neural mechanisms leading to the absence of the P1 and N1 components in the damaged hemisphere, and to the absence of N1 in the intact hemisphere, it is reasonable to suppose that the lesion of our patient, while sparing the neural generators of these components, has compromised crucial top-down influences on early attentional mechanisms arising from frontal parietal areas (Knight, 1997; LaBerge, 1997). In normal subjects, these influences are necessary to bias focusing of spatial attention toward features or objects of interest, thus permitting an efficient visual search. In extinction patients, the lack of such influences results, more dramatically, in a loss of perceptual awareness of the stimulus presented to the contralesional hemifield, provided there is competitive stimulation. Such a dramatic effect should not be too surprising given that a complete suppression of the early ERP components has been reported in normal subjects for unattended motion stimuli (Valdes-Sosa, Bobes, Rodriquez, & Pinilla, 1998).

At any rate, it is likely that the generators of P1 and N1 are partially abnormal in the right hemisphere of our patient. When facilitated by the top-down attentional bias exerted by the frontal-parietal areas, the right hemisphere can generate normal waveforms. However, when such influences are partially interrupted, their function is abnormal and this explains the lack of early components during extinction in the damaged right hemisphere. A similar explanation may be invoked for the abnormal responses of the intact left hemisphere where the neural responses to the extinguished ipsilateral left stimulus depend on the commissural input arising from the damaged right hemisphere.

On more general grounds, our findings are in general agreement with the recent functional brain imaging results of Lumer, Friston, and Rees (1998) in normal subjects experiencing binocular rivalry. These authors have shown that the shifts in the perceptual representations related to binocular rivalry were specifically correlated with an increased activity in the frontal parietal cortex. We believe that in our extinction patient, the shifts from correct to incorrect detection of bilateral stimuli may be related to a defective functioning of the frontal parietal cortex with a consequent bias under conditions of stimulus competition toward forming internal representations of the ipsilesional space only.

METHODS

During the recording session, the patient was seated on a chair with the head positioned on a chin rest at 57-cm distance from a perimeter. Stimuli consisted of light flashes produced by small LEDs, subtending 0.3° of visual angle and placed on the perimeter on either side 20° lateral and 15° below a central fixation point. This position was selected in order to stimulate two homologous locations in either hemifield in an area free from field deficits as assessed by standard Humphreys Computerised perimetry (see Figure 1B). The stimuli had a luminance of 400 cd/m^2 and a duration of 20 msec. Mean background luminance was 50 cd/m^2. The patient's fixation was continuously monitored by means of a video camera and by electrooculographic recording. Whenever the patient lost central fixation, the examiner reminded her to maintain the gaze onto the fixation point before the subsequent trial was initiated. The patient was asked to press a button with her right index finger as soon as possible when a stimulus appeared no matter whether it was unilateral left, right, or bilateral. Responses faster than 150 msec and slower than 1,500 msec were considered anticipations and retards, respectively, and discarded. They accounted for only 5.3% of the total. After the manual response, the patient was asked to tell which stimulus she had seen (right, left, or bilateral) with no time constraints. The examiner entered the patient's response on a PC according to a pre-established code and started the following trial. The experiments were carried out in two sessions separated by approximately 2 months. Since both behavioral and ERP results were similar on the two occasions, the data were averaged across sessions. A total of 17 blocks of trials were run, each consisting of 60 unilateral (30 left and 30 right) and 60 bilateral stimuli presented in a random order. A total of 1,020 unilateral (510 left and 510 right) and 1,020 bilateral stimuli were presented.

The electroencephalogram (EEG) was recorded from 28 tin electrodes mounted on an elastic cap, at both standard (10–20 System) and non-standard sites. The right-mastoid electrode served as reference. ERP waveforms were re-referenced to the average of the right and left mastoids off-line. The electrooculogram (EOG) was recorded with two electrodes placed, in a bipolar montage, at about 1 cm from the external canthus of both eyes. Vertical eye movements and eye blinks were detected by an electrode positioned below the right eye and referenced to the right mastoid. The EEG and

the EOG signals were amplified, filtered (DC-100 Hz), and digitized at 500 Hz. Artifacts due to eye movements and blinks, as well as recordings when the patient failed to respond manually or made an anticipatory or retarded response, were not included in the analysis. This led to the rejection of 13.3% of the trials. Average waveforms were computed for each stimulus type: bilateral-correctly reported, bilateral with contralesional extinction, unilateral ipsilesional, and unilateral contralesional, in a time window between 200 msec prestimulus and 824 msec post-stimulus. After the rejection procedure was applied to the data, each waveform resulted from the average of 582, 342, 443, and 402 trials, for bilateral-correctly reported stimuli, bilateral stimuli with contralesional extinction, unilateral-ipsilesional, and -contralesional stimuli, respectively. In order to gather several repeated measures in the same subject in different conditions, the two experimental sessions were divided off-line into five blocks of 408 trials each, and the averages for the four conditions of perceptual report, namely bilateral-correct, bilateral-extinguished, unilateral-ipsilesional, and unilateral-contralesional, were calculated. Mean amplitude of the P1 and N1 components was then measured, in the unfiltered averages, as the mean voltage between 70 and 90 msec post-stimulus for P1 in the bilateral-correct trials; between 105 and 125 msec post-stimulus for P1 in the bilateral-extinguished, unilateral-ipsilesional, and unilateral-contralesional trials; and between 150 and 170 msec post-stimulus for the N1 in all trials. The mean amplitudes were calculated relative to the 200-msec prestimulus baseline interval. Four posterior electrode sites in the intact (left) hemisphere were considered for this analysis: P3, P5, T5, and TO1. The most posterior and medial site, namely, O1, did not show a reliable response and therefore, was not considered in the analysis. This apparent lack of response was probably due to the particular shape of the patient's skull in the occipital pole, that did not perfectly fit the cap, and to the massive muscular artifact, visible in this electrode site, as a consequence of the paresis. The ethical committee of the University of Verona approved this study.

Acknowledgments

We thank L. Chelazzi, J. Driver, M. Eimer, H.-O. Karnath, R. Knight, and A. Nobre for their comments on a preliminary version of the paper. This study was supported by a Grant of the Italian MURST.

Reprint requests should be sent to: C. A. Marzi, Dipartimento di Scienze Neurologiche e della Visione, Università di Verona, 8 Strada Le Grazie, 37134 Verona, Italy.

REFERENCES

Albert, M. L. (1973). A simple test of visual neglect. *Neurology, 23,* 658–664.

Baylis, G. C., Driver, J., & Rafal, R. D. (1993). Visual extinction and stimulus repetition. *Journal of Cognitive Neuroscience, 5,* 453–466.

Berti, A., Allport, A., Driver, J., Dienes, Z., Oxbury, J., & Oxbury, S. (1992). Levels of processing for visual stimuli in an extinguished field. *Neuropsychologia, 30,* 403–415.

Bisiach, E., & Vallar, G. (1988). Hemineglect in humans. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology*, (Vol. 1, pp. 195–222). Amsterdam: Elsevier.

Coles, M. G. H., & Rugg, M. D. (1995). In M. D. Rugg & M. G. H. Coles (Eds.), *Electrophysiology of mind. event-related brain potentials and cognition*, (Vol. 25, pp. 12–23). Oxford: Oxford University Press.

Damasio, H., & Damasio, A. R. (1989). *Lesion analysis in neuropsychology*. New York: Oxford University Press.

Driver, J., Mattingley, J. B., Rorden, C., & Davis, G. (1997). Extinction as a paradigm measure of attentional bias and restricted capacity following brain injury. In P. Thier & H.-O. Karnath (Eds.), *Parietal lobe contributions to orientation in 3D space* (pp. 401–429). Heidelberg: Springer-Verlag.

Frith, C., Perry, R., & Lumer, E. (1999). The neural correlates of conscious experience: An experimental framework. *Trends in Cognitive Sciences, 3,* 105–114.

Heinze, H. J., Mangun, G., Burchert, W., Hinrichs, H., Scholz, M., Munte, T., Gos, A., Scherg, M., Johannes, S., Hundeshagen, H., Gazzaniga, M. S., & Hillyard, S. A. (1994). Combined spatial and temporal imaging of brain activity during visual selective attention in humans. *Nature, 372,* 543–546.

Hillyard, S. A., & Anllo-Vento, L. (1998). Event-related potentials in the study of visual selective attention. *Proceedings of the National Academy of Science, U.S.A., 95,* 781–787.

Jennings, J. R., & Wood, C. C. (1976). The ϵ-adjustment procedure for repeated-measures analyses of variance. *Psychophysiology, 13,* 277–278.

Karnath, H. O. (1988). Deficit of attention in acute and recovered visual hemi-neglect. *Neuropsychologia, 1,* 27–43.

Kinsbourne, M. (1993). Orientational bias model of unilateral neglect: Evidence from attentional gradients within hemispace. In I. H. Robertson & J. C. Marshall (Eds.), *Unilateral neglect: clinical and experimental studies* (pp. 63–86). Hove, UK: Lawrence Erlbaum Associates.

Knight, R. T. (1997). Distributed cortical network for visual attention. *Journal of Cognitive Neuroscience, 9,* 75–91.

LaBerge, D. (1997). Attention, awareness and the triangular circuit. *Consciousness and cognition, 6,* 149–181.

Lumer, E. D., Friston, K. J., & Rees, G. (1998). Neural correlates of perceptual rivalry in the human brain. *Science, 280,* 1930–1934.

Mangun, G. R. (1995). Neural mechanism of visual selective attention. *Psychophysiology, 34,* 4–18.

Marzi, C. A., Fanini, A., Girelli, M., Ipata, A. E., Miniussi, C., Prior, M., & Smania, N. (1997). Is extinction following parietal damage an interhemispheric disconnection phenomenon? In P. Thier & H.-O. Karnath (Eds.), *Parietal lobe contribution to orientation in 3d space* (pp. 431–445). Heidelberg: Springer-Verlag.

Marzi, C. A. Smania, N., Martini, M. C., Gambina, G., Tomelleri, G., Palamara, A., Alessandrini, F., & Prior, M. (1996). Implicit redundant-targets effect in visual extinction. *Neuropsychologia, 34,* 9–22.

Mattingley, J. B., Davis, G., & Driver, J. (1997). Preattentive filling-in of visual surfaces in parietal extinction. *Science, 275,* 671–674.

Miniussi, C., Girelli, M., & Marzi, C. A. (1998). Neural site of the redundant target effect: Electrophysiological evidence. *Journal of Cognitive Neuroscience, 10,* 216–230.

Smania, N., Martini, M. C., Gambina, G., Tomelleri, G., Palamara, G., Natale, A., & Marzi, C. A. (1998). The spatial distribution of visual attention in hemineglect and extinction patients. *Brain, 121,* 759–1770.

Valdes-Sosa, M., Bobes, M. A., Rodriguez, V., & Pinilla, T. (1998). Switching attention without shifting the spotlight: Object-based attentional modulation of brain potentials. *Journal of Cognitive Neuroscience, 10,* 137–151.

Volpe, B. T., Ledoux, J. E., & Gazzaniga, M. S. (1979). Information processing of visual stimuli in an 'extinguished' field. *Nature, 282,* 722–724.