Neural Site of the Redundant Target Effect: Electrophysiological Evidence

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Abstract

■ The present study represents an attempt to find an electrophysiological correlate of the redundant targets effect, or RTE (i.e., the speeding up of reaction time, or RT, for redundant vs. single targets). Subjects made a speeded response either to one small checkerboard presented to the left or right of fixation or to a pair of identical checkerboards presented simultaneously to both hemifields. Both single and double targets could appear either in the upper or lower visual hemifield. The task required detection but not discrimination of the stimuli. During task performance, we recorded the event-related potentials (ERPs) elicited by the checkerboard targets. As in previous studies, we found that manual RTs to bilateral stimuli were faster than those to unilateral stimuli. This effect was more marked for lower- than for upper-field stimuli and could not be ascribed to probability summation. In addition, we found that the P1 and N1 components of the visual ERP had a shorter latency for bilateral than for summed unilateral stimuli presented to the two hemifields. In parallel with the behavioral findings, the latency values for the above components showed a larger RTE for lower-field stimuli. These findings indicate that the RTE occurs at the level of early visual processing, probably in the extrastriate visual cortex, rather than at late decisional or premotor stages. ■

INTRODUCTION

It is well established that button-press responses are faster for redundant signals than for similar single signals. Evidence for this phenomenon, known as the RTE, comes from experiments using both bimodal (visual-acoustic) and unimodal (visual) stimuli. Following Hershenson's (1962) and Raab's (1962) original demonstrations, a number of other studies confirmed the existence of the phenomenon and attempted to distinguish between a probabilistic explanation (Raab, 1962) and a "neural" explanation. Probabilistic interpretations, also known as "race models," postulate stochastically independent afferent channels each carrying information from one of the redundant signals. Whichever channel reaches a criterion level of activation first (i.e., "wins the race") activates a decision center mediating the motor response. The rate of processing within a given channel is assumed to vary randomly from trial to trial, independently of the rate of processing in the other channels. As a result, increasing the number of channels (e.g., by presenting multiple redundant stimuli) increases the probability that the fastest channel will be substantially quicker than an average channel. Therefore, RT will be faster when two stimuli are presented than when a single stimulus is presented, for purely statistical reasons, even if the two stimuli are processed completely independently.

This interpretation does not require any neural mechanism to account for the summation effect but simply relies on the fact that the probability of a fast detection increases with the number of targets. A neural alternative to the race model has been proposed by Miller (1982, 1986), who presented a "coactivation model" in which signals from the various channels are summed in an activational pool before reaching threshold for initiating the motor response.

Other models incorporating parts of the above models have been proposed, such as Meijers and Eijkman's (1977) and, more recently, the "superposition" model of Schwarz (1989, 1994) and Schwarz and Ischebeck (1994).

The present study represents an attempt to find a neural correlate of the RTE and thereby obtain some clues as to its mechanism. Evidence in the literature for a cerebral locus of the RTE is scanty and indirect. Some of it derives from the very fact that the RTE occurs with stimuli of different modalities. This obviously suggests that it is not likely to be mediated at the early stages of sensory processing where intersensory convergence is very limited if it exists at all. Furthermore, in the visual modality, the effect is not retinotopic in the sense that it occurs independently of the absolute and relative position of the redundant signals in the visual field. The RTE can also be demonstrated when stimuli are presented in different hemifields (i.e., to different cerebral hemispheres) and far from the vertical meridian (Marzi

et al., 1986; Marzi et al., 1997; Reuter-Lorenz, Nozawa, Gazzaniga, & Hughes, 1995). The primary visual areas are known to be interconnected by means of the corpus callosum only for visual-field representations that are close to the vertical meridian (see Marzi, 1986, for a review). Therefore, the presence of an RTE with stimuli presented at peripheral visual locations in different hemispheres provides further evidence that its neural basis must be sought in extrastriate visual areas that have interhemispheric connections between cortical areas with peripheral visual field representations. This assumes a neural mechanism as opposed to a statistical one.

Evidence from brain-damaged patients is also consistent with an extrastriate locus for the RTE: Marzi, Tassinari, Aglioti, and Lutzemberger (1986) and Corbetta, Marzi, Tassinari, and Aglioti (1990) have demonstrated that some hemianopic patients with a lesion in the striate cortex show an RTE even when one of the stimuli in a pair is presented to the blind hemifield and goes undetected by the patients. Furthermore, Tomaiuolo, Ptito, Marzi, Paus, and Ptito (1997) have found evidence of an RTE with pairs of stimuli presented across the vertical meridian even in some hemispherectomized subjects. Marzi et al. (1996) have found that patients showing extinction of contralesional visual stimuli as a consequence of a unilateral lesion of the right parietal-temporal cortex show an RTE despite their reporting only one stimulus of a pair. Finally, Reuter-Lorenz et al. (1995) and Marzi et al. (1997) have documented that subjects who underwent a complete section of the corpus callosum show a large RTE when stimuli are presented to different hemispheres even though they show extinction of stimuli presented to the left visual field (LVF) during bilateral stimulation. Taken together, these findings suggest that the RTE is likely to be subserved by visual areas other than the primary visual cortex. Moreover, on the basis of the results with hemispherectomized patients, one might argue that it is subcortically mediated by visual centers such as the superior colliculus (SC) that have large receptive fields and send projections via the pulvinar to extrastriate cortical visual areas. This hypothesis also fits in well with single-cell evidence (Stein, Meredith, & Wallace, 1993) of polymodal convergence in the SC, although it has been shown that such a convergence depends upon corticotectal input (Wallace & Stein, 1994).

On the basis of these considerations, we conducted a study of the RTE using ERPs elicited by the same stimuli used for the behavioral response. This approach has the advantage of allowing a direct comparison between ERP and RT data from same task and subjects. The predictions were straightforward. First, if the cognitive stage at which the RTE occurs is perceptual, evidence of it should be apparent in the early visual ERP components as an increase in amplitude and/or a decrease in latency. In contrast, early ERP effects should not be observed if the RTE is mediated at the decisional or motor level, as

has been previously hypothesized (Mordkoff, Miller, & Roch, 1996). The C1 is the earliest component in the visual ERP; it has a latency range of 40 to 80 msec and is thought to be generated in the primary visual cortex. Evidence that C1 is generated by the striate cortex and in particular by neurons in the calcarine area has been provided quite convincingly by its polarity reversal for upper vs. lower visual-field stimulation (Clark, Fan, & Hillyard, 1995; Mangun, 1995). It is known that the visuotopic organization of the striate cortex is such that the upper and lower hemifields are mapped in the lower and upper banks of the calcarine fissure, respectively. Therefore, stimulation of opposite sides of the horizontal meridian of the visual field would activate neuronal pools with geometrically opposite orientations. As a consequence, the ERP response mediated by neurons located within the depths of the calcarine fissure should show a polarity reversal for upper vs. lower visual-field stimuli. Such is not the case for other visual areas that do not have the peculiar retinotopic organization (so called cruciform model) of the calcarine cortex, although it cannot be excluded that some up-down polarity inversion might be present for V2 and V3 as well (Schroeder et al., 1994; Simpson, Foxe, Vaughan, Mehta, & Schroeder, 1994; Simpson et al., 1995). The P1 component is the second component in the visual ERP waveform with an onset at 60 to 80 msec and a peak around 100 to 130 msec. There is evidence that this component, which does not show a polarity reversal for upper vs. lower visual-field stimulations, is mainly generated in the extrastriate visual areas. The N1 component is the third component, with a latency range of 150 to 220 msec poststimulus. It is thought to be made up of multiple subcomponents and generated in the extrastriate visual areas, with some subcomponents originating from frontal areas. Consistent with its likely anatomical source, it does not show a polarity inversion between upper and lower visual-field stimulations (Mangun, 1995; Clark et al., 1995).

In the present experiment, the stimuli consisted of small, rectangular, high-contrast checkerboards presented in the left and right periphery of either the upper or lower visual field (see "Methods" section for details). In each trial there could be one stimulus in one of the four visual-field quadrants or one pair of stimuli in either the upper or the lower visual field, as illustrated in Figure 1. The different types of stimuli (bilateral upper or lower and unilateral left or right and upper or lower) were randomly intermixed in each trial block. The subjects signaled the detection of the stimuli by pressing a button regardless of the position of the stimulus or number of the stimuli in the display.

The kind of approach we have followed in this study to compare ERP responses to bilateral and unilateral stimuli in a meaningful way involves a comparison between responses to bilateral stimuli and summed responses to unilateral stimuli presented to one or the

Figure 1. Illustration of the conditions of stimulus presentation; see text for details.
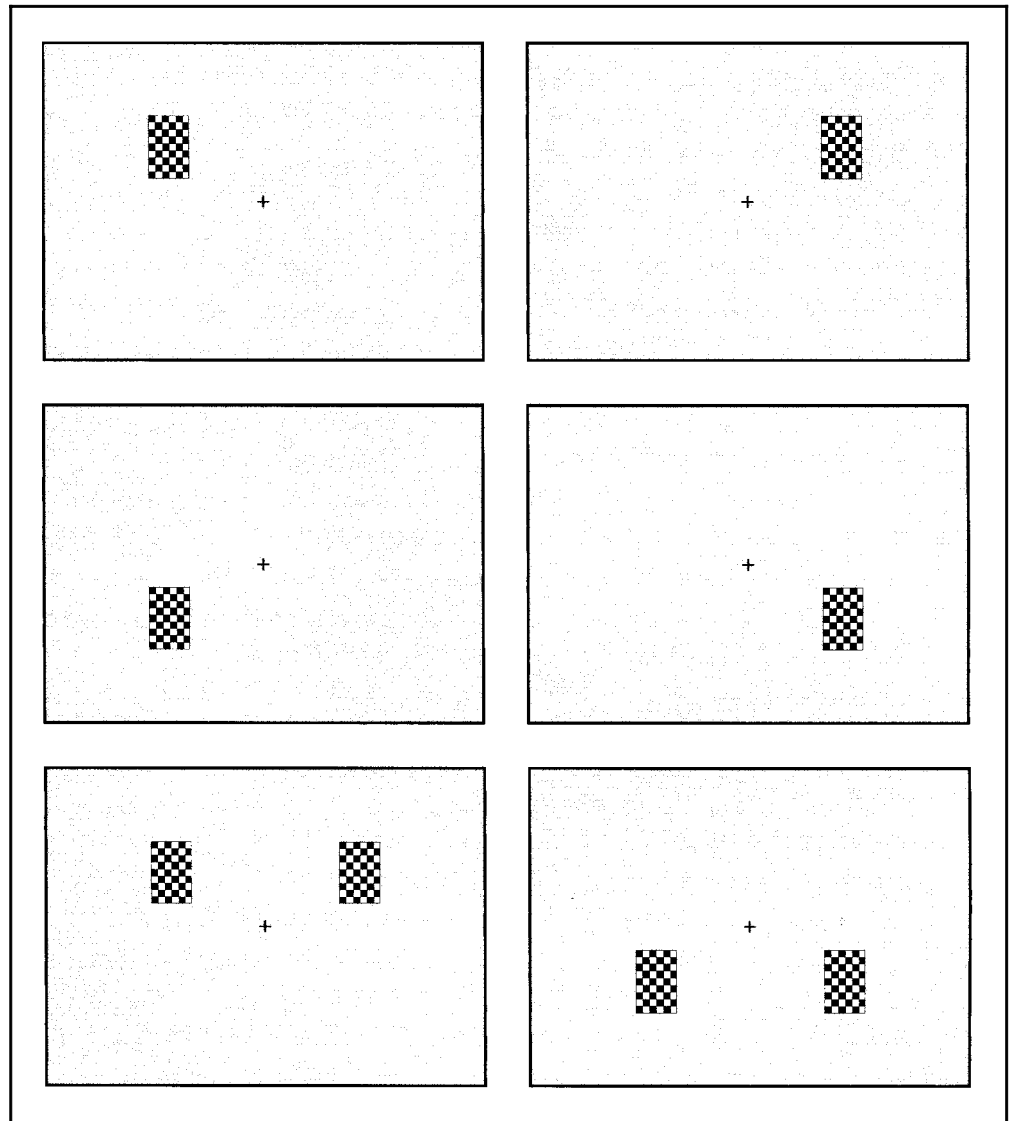
other hemifield. In other words, for each electrode site of interest we have calculated the algebraic sum of the response to contralateral and ipsilateral stimuli and compared it to the response to bilateral stimuli presented simultaneously. Such a comparison is more appropriate than just a comparison between responses to bilateral stimuli and those to the "direct" (i.e., contralateral) unilateral stimuli. Furthermore, to minimize contamination by volume conduction effects, we based our conclusions mainly on latency rather than amplitude effects.

In sum, the main goal of the present study was to ascertain whether a possible ERP correlate of RTE can be found in the early components of the visual ERP, which would suggest an origin in visual sensory processing rather than in decision and motor stages. Moreover, one would predict that P1 and N1 rather than C1 should show an RTE given that the former is an index of extrastriate cortical activity, whereas the latter is likely to represent the activity of the primary visual cortex.

RESULTS

Behavioral Results

Overall, subjects responded faster to bilateral (248 msec) than to unilateral stimuli (257 msec), and this is evidence of an RTE. Inspection of Figure 2 shows that this effect is more marked for lower- than upper-field stimuli. An analysis of variance (ANOVA) was conducted on RT values with two within-subjects factors: Hemifield (upper vs. lower) and Stimulus Type (unilateral left, unilateral right, and bilateral). The effect of Stimulus Type was significant ($F(2, 22) = 35.13$, $p < 0.0001$) with bilateral stimuli (248 msec) faster than left (258 msec) and right (256 msec) unilateral stimuli. Post-hoc t tests with Bonferroni criteria correction showed that the contrasts between the bilateral and the unilateral left and right conditions were both significant, Bilateral vs. Unilateral Right $t(11) = 6.37$, $p < 0.0001$; Bilateral vs. Unilateral Left $t(11) = 8.45$, $p < 0.0001$. The difference between the two
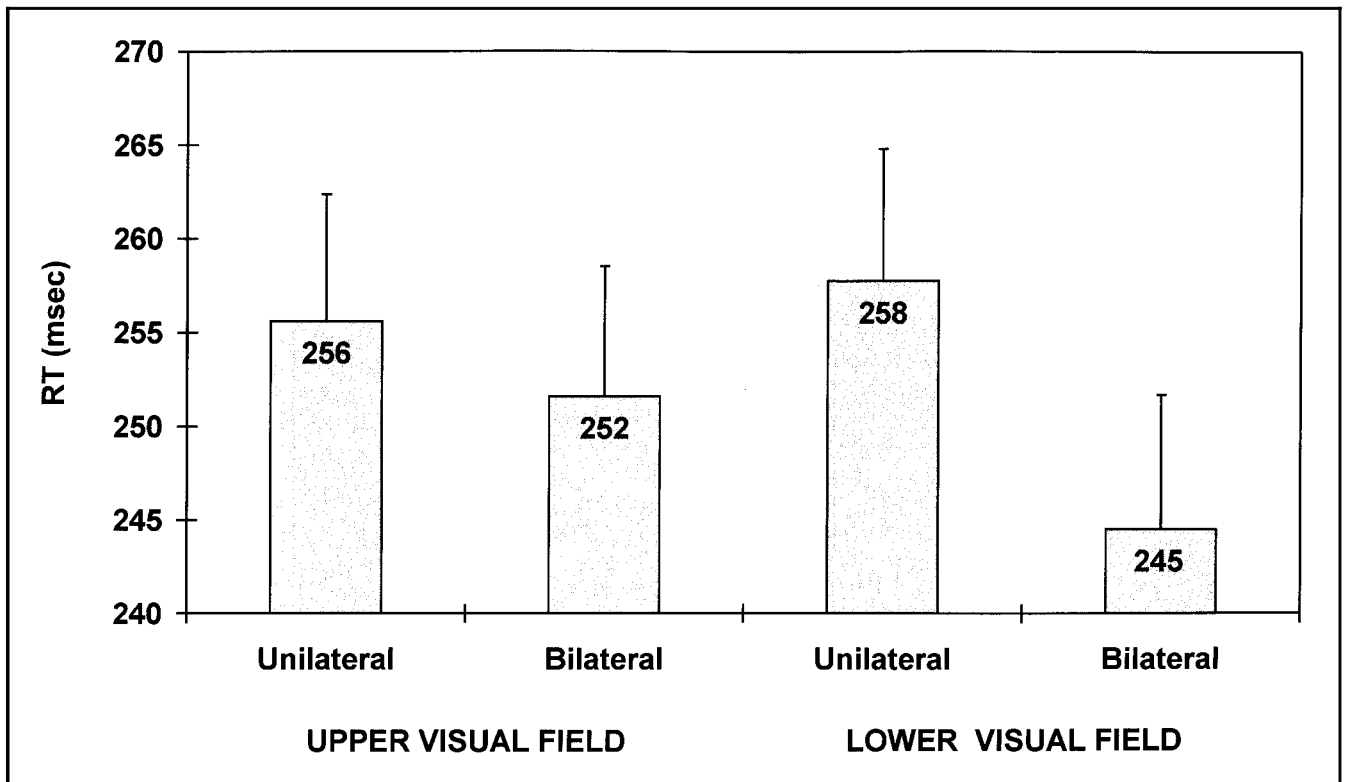
Figure 2. Mean RT for bilateral and unilateral stimulus presentations. The unilateral data have been averaged across left-right hemifields. The bars indicate standard errors of the mean.

unilateral conditions did not reach significance, $t(11)$ = 1.68, p = 0.12. The effect of Hemifield (Upper vs. Lower) did not approach significance ($F(1, 11)$ = 0.76, p = 0.4), but the interaction between Hemifield and Stimulus Type was highly significant ($F(2, 22)$ = 21.85, p < 0.0001). This shows that the RTE was different for the upper and the lower hemifields. Post-hoc t tests carried out directly on the difference between bilateral and unilateral presentations confirmed that the RTE was reliably greater in the lower than in the upper hemifield, $t(11)$ = 7.8, p < 0.0001. Importantly, as discussed below, this effect is also present in the ERP results.

In order to assess the nature of the RTE, we followed Miller's (1982, 1986) procedure to determine whether a probabilistic explanation could account for the results. Such a procedure relies on the mean cumulative frequency distribution of RTs in the bilateral and the two unilateral conditions of stimulus presentation (see Figure 3). Such a detailed graphical description of the RT distribution is clearly more suitable than summary statistics for examining the present data. It is evident from simple inspection of Figure 3 that the RTs in the bilateral condition are faster than those of the unilateral conditions throughout the whole distribution with very little overlap, which provides convincing evidence for the reliability of the RTE. To distinguish between probabilistic and "neural" explanations of the RTE, we carried out an

analysis of the violation of Miller's inequality (see Miller, 1982, 1986; Marzi et al., 1996). The race-model inequality test of Miller sets an upper limit for the cumulative probability of a response by any time t given redundant targets:

$$P(RT \leq tT^L \text{ and } T^R) \leq P(RT \leq tT^L) + P(RT \leq tT^R),$$

where $P(RT \leq tT^L \text{ and } T^R)$ is the cumulative probability of a correct detection with double targets, $P(RT \leq tT^L)$ is the cumulative probability of a response given a target in the left visual hemifield and nothing in the right hemifield, and $P(RT \leq tT^R)$ is the cumulative probability of a response given a target in the right hemifield and nothing in the left hemifield. When the upper bound is violated, a neural summation is likely to occur; if not, a probabilistic explanation is sufficient to explain the RTE.

This analysis showed that the RTE effect in the present study is probably attributable to coactivation rather than probability summation, in that there was a violation of the race inequality (see Figure 4, where the amount of violation is represented by the negative values). This violation suggests that the summation effect found cannot be explained simply by a probabilistic model and leaves open the possibility of a neural effect.
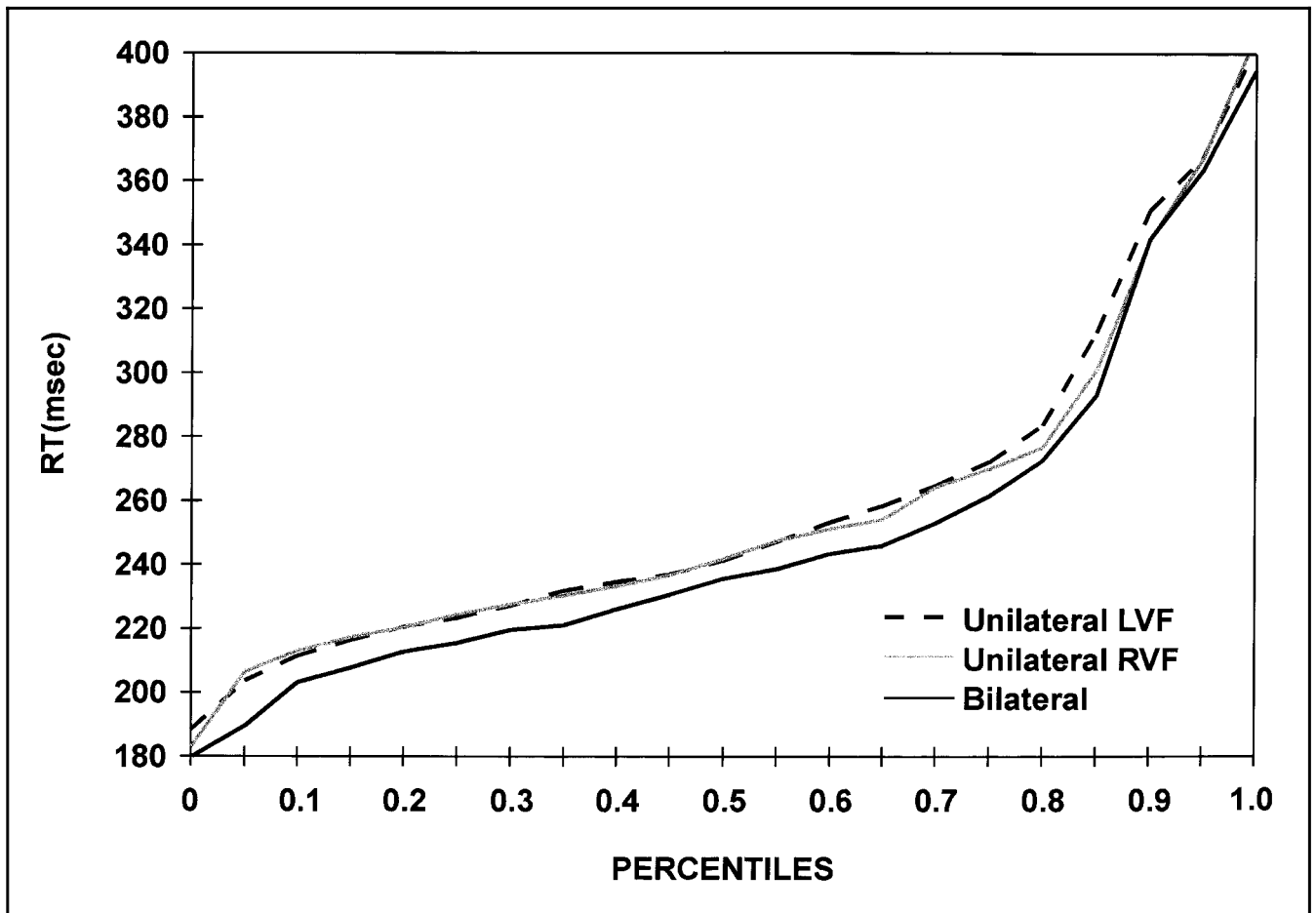
Figure 3. Cumulative frequency distribution of the mean RTs for unilateral left and right visual fields (LVF and RVF) and bilateral presentations. Note that although the distributions of unilateral RTs overlap considerably, there is an ample segregation between unilateral and bilateral RTs. This provides clear evidence that RTs to bilateral stimuli are consistently faster than those to unilateral stimuli throughout the whole range of RTs.

ERP Results

Basic Components

Figure 5 shows the grand average of responses to bilateral stimuli in the upper and lower hemifield at various electrode sites. As shown in the upper left panel of the figure, the ERP waveforms include three clearly identifiable components: C1, P1, and N1. Note that the polarity of the C1 component for upper vs. lower hemifield stimulations reverses from negative to positive, respectively. This peculiarity can be related, as mentioned in the "Introduction," to the organization of the primary visual cortex that is thought to be the main generator of this component. The stimuli presented in the upper visual field stimulate neurons in the lower bank of the calcarine fissure that constitute an upright electric dipole, whereas the stimuli presented in the lower visual field stimulate neurons in the upper bank of the calcarine fissure, and consequently the electric dipole is oriented downward. It should be noted that the sign of the polarity of the ERPs components is, at the moment, of uncertain physiological meaning. The polarity reversal of the C1 component is only a sign of the stimulation of neural

structures that are one in front to the other, and therefore it might also be potentially related to other areas such as V2 and V3 in which such spatial arrangement is likely to occur.

Figure 6 shows isocontour voltage maps of the above three components for upper visual-field stimulation. C1 can be seen as an occipital-medial negative component beginning at about 55 to 60 msec from stimulus onset, whereas P1 can be seen as a positive component beginning at approximately 80 msec and lateralized to both sides, posteriorly. This distribution is in keeping with the possibility (Mangun, 1995; Clark et al., 1995) of a striate cortex generator for C1 and an extrastriate generator for P1. Finally, the N1 component appears as a negative deflection starting at around 130 msec, more anteriorly and centrally located in comparison to P1; this distribution is also in agreement with previous findings (Mangun, 1995; Clark et al., 1995).

Redundant Target Effect

Figure 7 shows examples of ERP recordings for two electrode sites in the left (P3, O1 on the left-hand side

Figure 4. Violation of the race inequality for the behavioral data. The negative values indicate the amount of violation; notice that violation occurs for the fast portion of the RTs distribution.
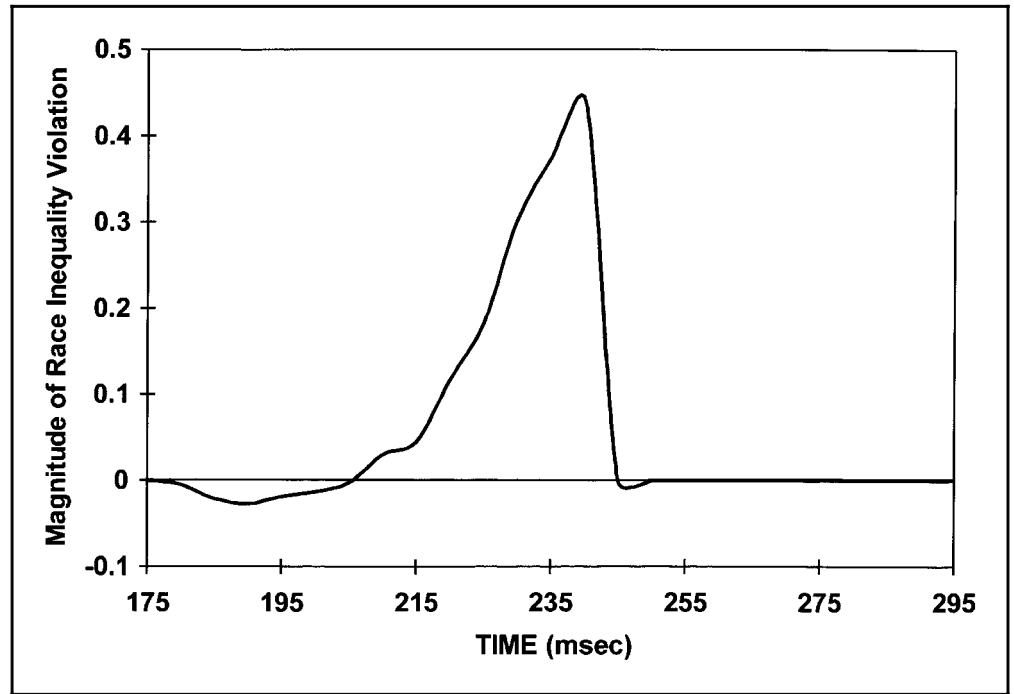
of the figure) and right (P4, O2 on the right-hand side of the figure) hemisphere for unilateral and bilateral presentations in the upper and lower hemifield. Clearly, for both the upper and lower hemifields, bilateral targets evoke larger amplitude and faster responses than unilateral targets presented contralaterally. As explained in the "Introduction," we thought that only latency measures were relatively free from contamination of volume conduction effects related to the bilateral stimulation, and therefore the description of the results will focus on such a measure. It will suffice to say that usually the amplitudes of the various components were larger for responses to bilateral than to the contralateral "direct" unilateral stimuli.

Figure 8 shows a composite diagram of the latency differences between responses to bilateral and unilateral targets for various electrode sites of interest and for the three components considered in the study. The figure reports only lower hemifield values because the RTE was significantly larger in this hemifield than in the upper one. Clearly, a latency advantage for the redundant vs. the single target is present practically at all electrode sites and for all three components.

C1. An ANOVA carried out on peak latency measurements of the C1 component showed a significant main effect of Stimulus Type ($F(1, 11) = 12.90, p = 0.004$), with bilateral presentations (67.14 msec) yielding faster values than unilateral (direct) presentations (70.48 msec). The main effect of Hemifield (Lower vs. Upper) did not approach significance. Importantly, the interaction between Hemifield and Stimulus Type was significant ($F(1, 11) =$

$14.05, p = 0.003$), with the lower hemifield yielding a larger RTE than the upper hemifield.

P1. The latency of the P1 component showed a significant effect of Hemifield ($F(1, 11) = 12.06, p = 0.005$), with the lower hemifield yielding shorter latency values (101.1 msec) than the upper hemifield (106.86 msec). Stimulus Type ($F(1, 11) = 5.15, p = 0.044$) showed a significant advantage of bilateral vs. unilateral presentations (102.18 vs. 105.78 msec). Finally, there was a significant effect of Electrode ($F(7, 77) = 7.49, p = 0.002$), but no interactions reached statistical significance.

N1. The analysis of the N1 component yielded the following significant main effects: Hemifield ($F(1, 11) = 13.26, p = 0.004$), with the lower hemifield yielding a shorter latency than the upper hemifield (141.66 vs. 149.57 msec); Stimulus Type ($F(1, 11) = 11.47, p = 0.006$), with shorter latencies for bilateral than unilateral stimuli (143.49 vs. 147.73 msec); and Electrode ($F(7, 77) = 5.03, p = 0.015$). Finally, the interaction between Hemifield and Stimulus Type was significant ($F(1, 11) = 8.87; p = 0.013$), with the lower hemifield yielding a larger RTE than the upper.

Comparison between "Physiological" Responses to Bilateral Stimuli and the Summed Responses to Unilateral Stimuli

One possible problem for interpreting our ERP results is that the observed RTE in amplitude and latency might be just a result of volume conduction. In principle, it is reasonable to assume that during bilateral stimulation, a
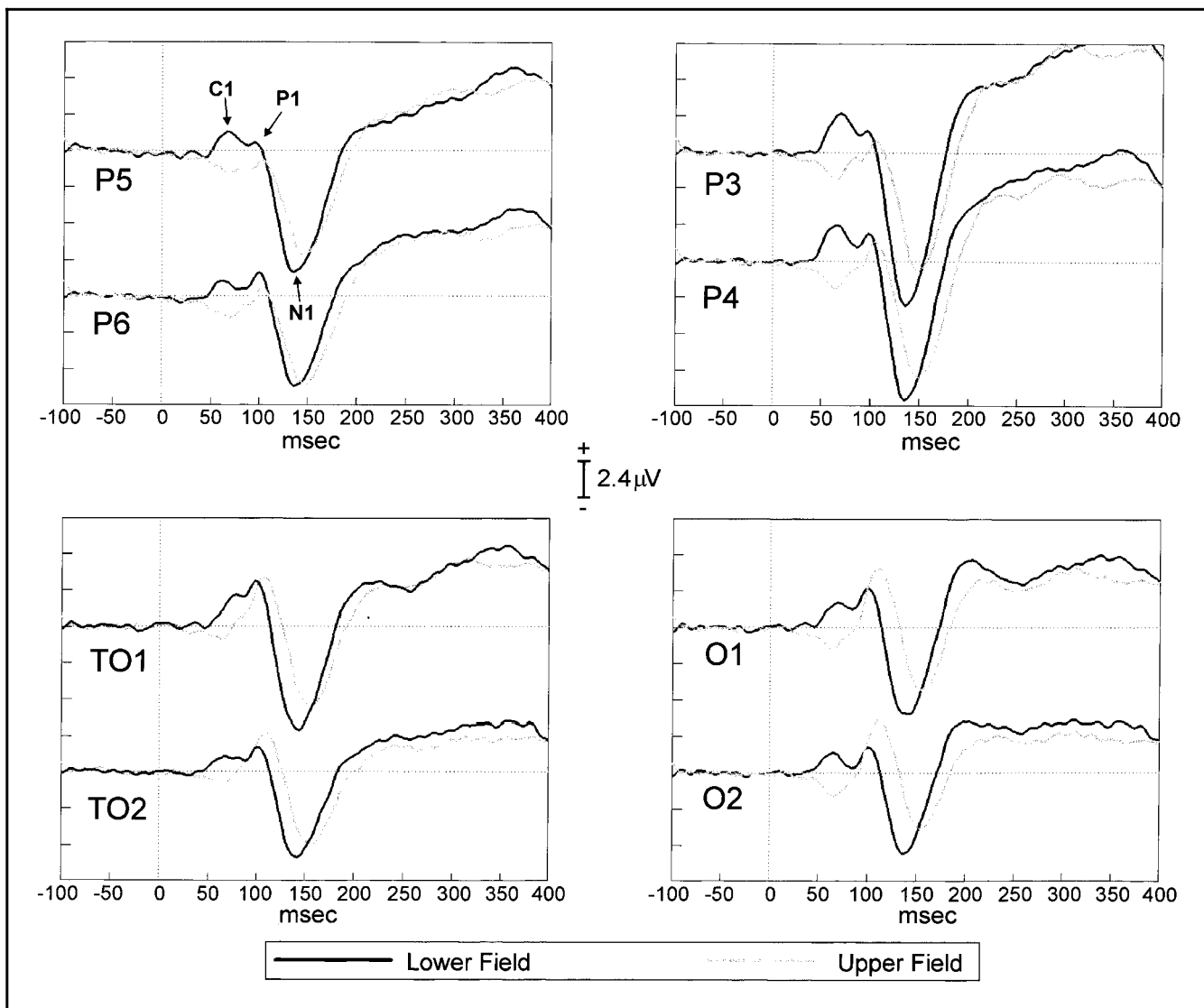
Figure 5. Samples of grand average ERP waveforms elicited by bilateral stimuli at eight electrode locations in the left (P3, P5, O1, TO1) and right (P4, P6, O2, TO2) hemispheres. Among the three components considered in the present study (C1, P1, N1; see upper left tracing), only the shortest latency C1 component shows a clear polarity reversal for upper versus lower field stimulations.

given electrode might record activity not only from a generator located in the same hemisphere but also from, possibly, the homologous one in the opposite hemisphere. If such were the case, the RTE found in our ERP recordings would be an artifact with no neurophysiological meaning.

We can provide clear-cut evidence that such is not the case, at least for P1 and N1, on the basis of a comparison between the actual recording from a given electrode during bilateral stimulation and the algebraic sum of the direct and the indirect unilateral responses recorded at the same electrode location. Figure 9 shows examples of recordings from the same sites as in Figure 7; clearly the amplitudes of the P1 and N1 components were larger for the summed values than for responses to bilateral stimuli, while the latencies were shorter for the re-

sponses to bilateral stimuli. This difference was not visible or was less clear for the C1 component.

The fact that the physiological response to bilateral stimuli was of smaller amplitude than the algebraic sum of the responses to the two unilateral stimuli speaks in favor of a minor role of volume conduction in explaining the electrophysiological RTE because pure volume conduction should lead to identical amplitudes. One might speculate here that the responses to bilateral stimuli entail an inhibitory component whereby their amplitude does not simply represent the sum of the two unilateral responses but a smaller value. More decisive are the results of the above comparisons for latency values: One would predict no latency differences between real and summed values if the RTE was uniquely related to volume conduction.
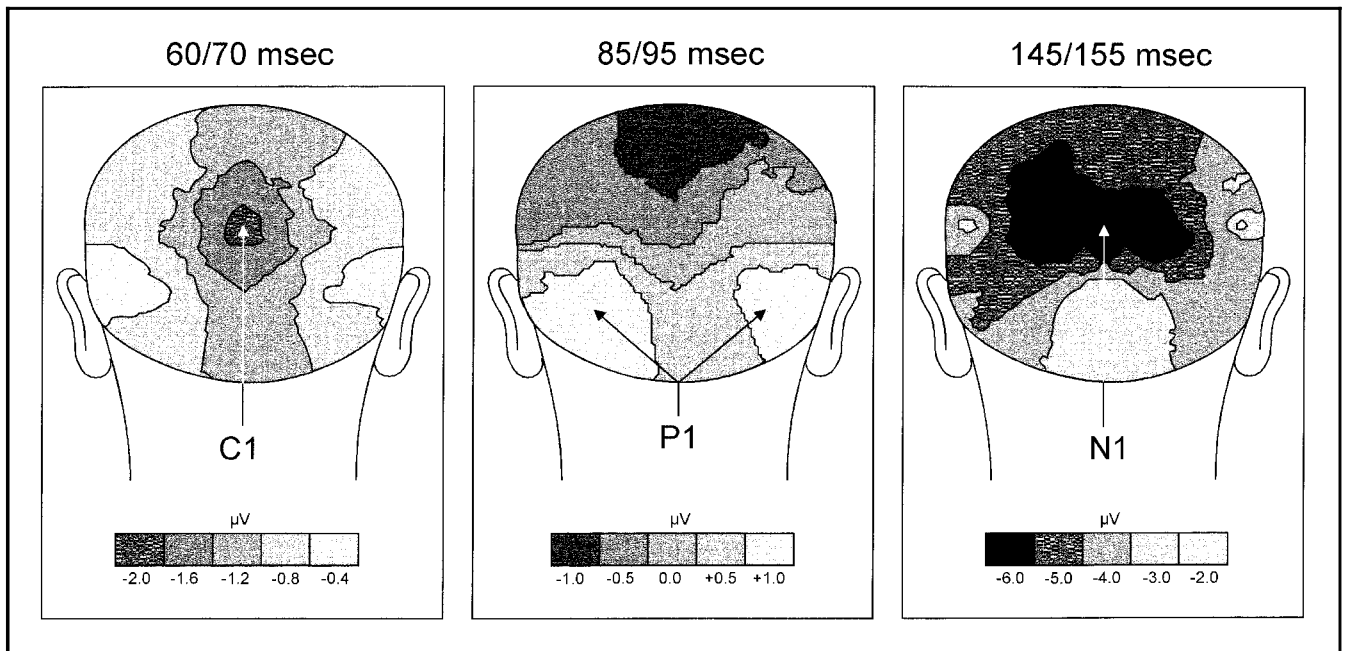
Figure 6. Isocontour voltage maps for the C1, P1, and N1 components of the ERP elicited by bilateral upper-field stimuli. The voltage was measured in the following time windows: 60 to 70 msec for the C1 component; 85 to 95 msec for the P1 component; 145-155 msec for the N1 component. Each level of shading represents a different voltage range in the three maps: Dark shading corresponds to negative values and light shading corresponds to positive values.

By means of separate ANOVA's we compared the responses to bilateral stimuli with the algebraic sum of the two unilateral responses. In these analyses we also included the three central electrodes Cz, Pz, and Oz that were left out in the preceding ANOVAs; see Figure 10 for a summary diagram. For latency values of the C1 component, the difference bilateral vs. algebraic sum was significant: $F(1, 11) = 6.44, p = 0.038$, with the summed values yielding slower latencies (70.23 msec) than "real" bilateral values (67.01 msec). Importantly, the interaction relating lower-upper fields with the summed-bilateral comparison was also significant: $F(1, 11) = 9.05, p = 0.012$, with the latency advantage of bilateral values larger in the lower hemifield.

For the P1 component there was also a significant advantage of "real" vs. summed values, with the latter yielding slower latencies (108.87 msec) than the former (102.29 msec) $(F(1, 11) = 8.56, p = 0.014)$, and the same was true for N1 (sum = 151.30 msec; response to bilateral stimuli = 143.30 msec) $(F(1, 11) = 36.39, p = 0.0001)$.

In conclusion, the latency differences between actual bilateral responses and summed unilateral responses are clearly not in keeping with a volume conduction explanation of our results. It is difficult to envisage how a volume conduction mechanism could account for a shorter latency during bilateral stimulation.

DISCUSSION

The main thrust of the above ERP results is that bilateral stimuli generally yielded shorter latencies than the sum of the two unilateral stimuli (i.e., of targets presented to the contralateral or ipsilateral hemifield only). Such an effect is common to all three components considered in the present study, although the effect on C1 may be contaminated by volume conduction. This is suggested by the similarity between the amplitude of the C1 component for responses to bilateral stimuli and for responses to summed unilateral stimuli. Moreover, the absolute mean values of the above difference tend to increase as one goes from C1 (3.2 msec) to P1 (6.6 msec) and N1 (8.0 msec). Together with the behavioral results showing that the RTE found in our study cannot be explained by a probabilistic hypothesis, the present electrophysiological results strongly argue for a neural coactivation mechanism that begins to operate at a perceptual rather than decisional or motor processing stage. The neural site of such operations is likely to be located in extrastriate visual areas given that P1 rather than C1 is the first component showing a clear RTE. In previous studies such a possibility has been ruled out on the basis of the observation that the RTE is present (and, in fact, larger) for bimodal than unimodal stimulus presentations (Miller, 1982). Given that the convergence of polimodal input is likely to occur beyond the initial information processing stages, the site of coactivation has been located at the decision (or motor) level. However, it should
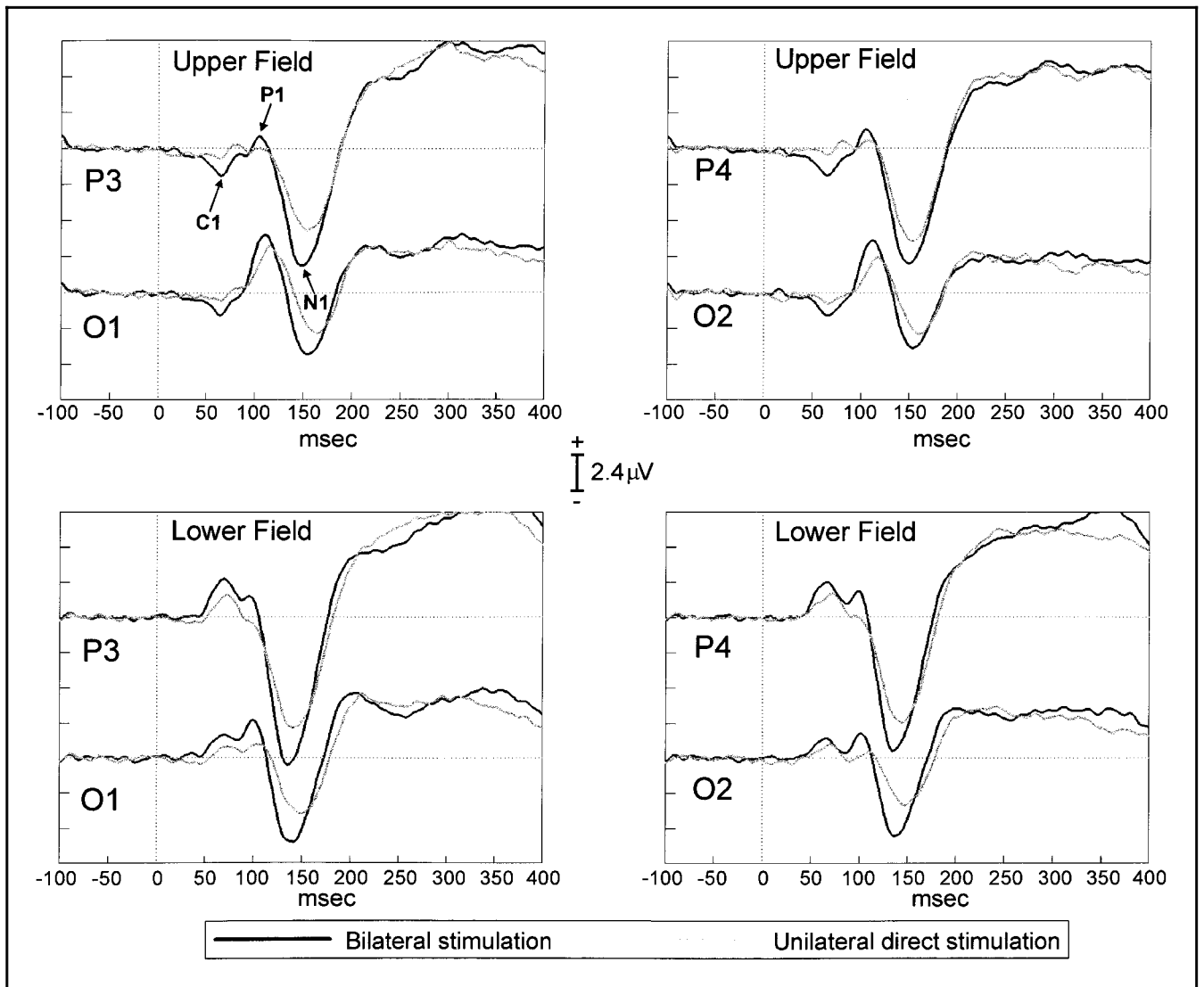
Figure 7. Samples of grand average ERP waveforms from four electrode locations in the left (P3, O1) and right (P4, O2) hemisphere for upper- and lower-field stimulations and bilateral and unilateral direct (contralateral hemifield) presentations. It can be seen that all three components (see upper left tracing) show a larger amplitude and a shorter latency for bilateral than unilateral presentations.

be noted that usually the above studies have used paradigms such as go/no-go that require a full identification of the stimuli rather than simple stimulus detection as in the present study. Therefore, we believe that the cognitive stage at which the RTE occurs may be not generalizable to all paradigms but depends on the cognitive stage required by the task. In our experimental conditions, namely, with unimodal stimuli and the requirement of simple stimulus detection, it is reasonable to predict an early visual stage for the effect, and this fits in very well with the present electrophysiological findings.

An intriguing observation in our study, namely, the smaller amplitude found for bilateral responses in comparison to the sum of the two unilateral responses, shows that the two hemispheres interact in a sort of inhibitory way rather than simply performing a linear summation of the unilateral inputs. A broadly similar

conclusion has been drawn previously by Kutas, Hillyard, Volpe, and Gazzaniga (1990) for the P300 in a study of the lateral distribution of late positive ERP components in normals and in commissurotomized patients.

Our main finding of a faster latency of responses to bilateral as opposed to summed unilateral stimuli is not consistent with a volume conduction explanation. A further, albeit indirect, evidence against a volume conduction explanation of our findings can be found in two patients that we have studied in a similar paradigm. They have different cerebral lesions but share a similar interruption of interhemispheric transfer. The first patient CZ (male, 50 years old, see Smania, Martini, Prior, & Marzi, 1996 and Marzi et al., 1997, for clinical details), 2 years before ERP testing, suffered from a vascular accident in the territory of the middle cerebral artery. Magnetic resonance imaging (MRI) revealed a large parietal-tempo-
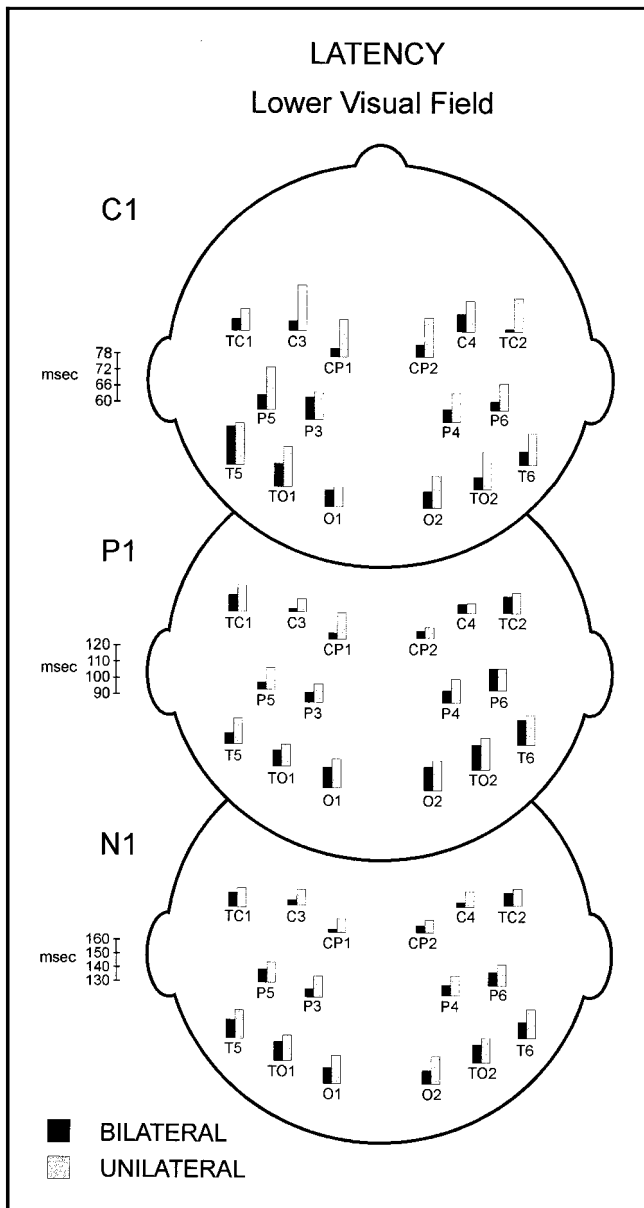
Figure 8. Mean latency of the C1, P1, and N1 components as recorded from 16 electrode sites (8 in each hemisphere) for bilateral and unilateral (contralateral hemifield) presentations in the lower hemifield. Note that the latency of the responses to bilateral stimuli is faster than that to unilateral stimuli for practically all electrodes and components.

ral-frontal lesion of the right hemisphere. CZ had a transient left hemineglect and a long-lasting left unilateral visual extinction. The important point here is that, following presentations in the left visual field of stimuli similar to those used in the present study, the responses of intact areas of the directly stimulated right hemisphere were as good as in the intact left hemisphere. On the contrary, the indirect (ipsilateral) responses of either hemisphere were small or lacking, and this can be interpreted as an impairment of callosal functioning (see Marzi et al., 1997). For our present purposes, this provides evidence for a minor role of volume conduction

in our results. The second patient, SM (male, 35 years old), as a result of a car accident that occurred about 6 months before ERP testing, suffered from a concussive lesion of the middle posterior third of the corpus callosum, as ascertained by MRI. As in the previous patient, stimulation of the hemifield ipsilateral to the recording hemisphere yielded only a very insubstantial response, whereas the response of the directly stimulated contralateral hemisphere was relatively good. In addition to providing evidence against a significant contribution of volume conduction to ipsilateral responses in a task and recording condition similar to that of the present study, the above data broadly confirm and extend previous ERP evidence of an impairment of interhemispheric transmission gathered in genetically acallosal subjects (Rugg, Milner, & Lines, 1985) and split-brain (Mangun, Luck, Gazzaniga, & Hillyard, 1991) patients.

Once a volume conduction explanation of the observed RTE effect on the latency of ERP responses is ruled out, we can offer some speculations on the underlying neurophysiological mechanisms. When two targets are presented simultaneously, there must be a functional interaction between the two hemispheres, with the generators of the early components reaching the peak of activity earlier than when only a single target is presented. For stimuli in corresponding positions across the vertical meridian such a functional interaction could occur via the homotopic callosal connections between visual cortices in the two hemispheres (Clarke & Miklossy, 1990). However, as pointed out in the "Introduction," RTE occurs even with stimuli presented to noncorresponding locations in opposite hemifields and with stimuli presented to distant locations in the same hemifield. In the latter conditions the RTE could be ensured via intrahemispheric long-range horizontal connections (McGuire, Gilbert, Rivlin, & Wiesel, 1991) while in the former the RTE might be subserved by heterotopic callosal connections (Clarke & Miklossy, 1990). It is well known (for a review see Marzi, 1986) that area 17 in many mammalian species including humans (Clarke & Miklossy, 1990) is free from callosal connections with the exception of the representation of a vertical strip of visual-field representation near the vertical meridian. Our stimuli were presented within 5° of eccentricity, and therefore it is reasonable to assume that the interhemispheric interaction necessary for the RTE may have included the callosal connections of the primary visual areas. A problem arises when trying to explain the RTE with stimuli presented at larger eccentricities; in such a case the callosal connections involved are probably those interconnecting areas further up in the visual processing hierarchy such as V5 that in humans has been found to have widespread callosal connections that apparently violate the vertical meridian rule (Clarke & Miklossy, 1990).

As to the cortical site showing an RTE effect in the ERP, it is clear that this is to be found among the early
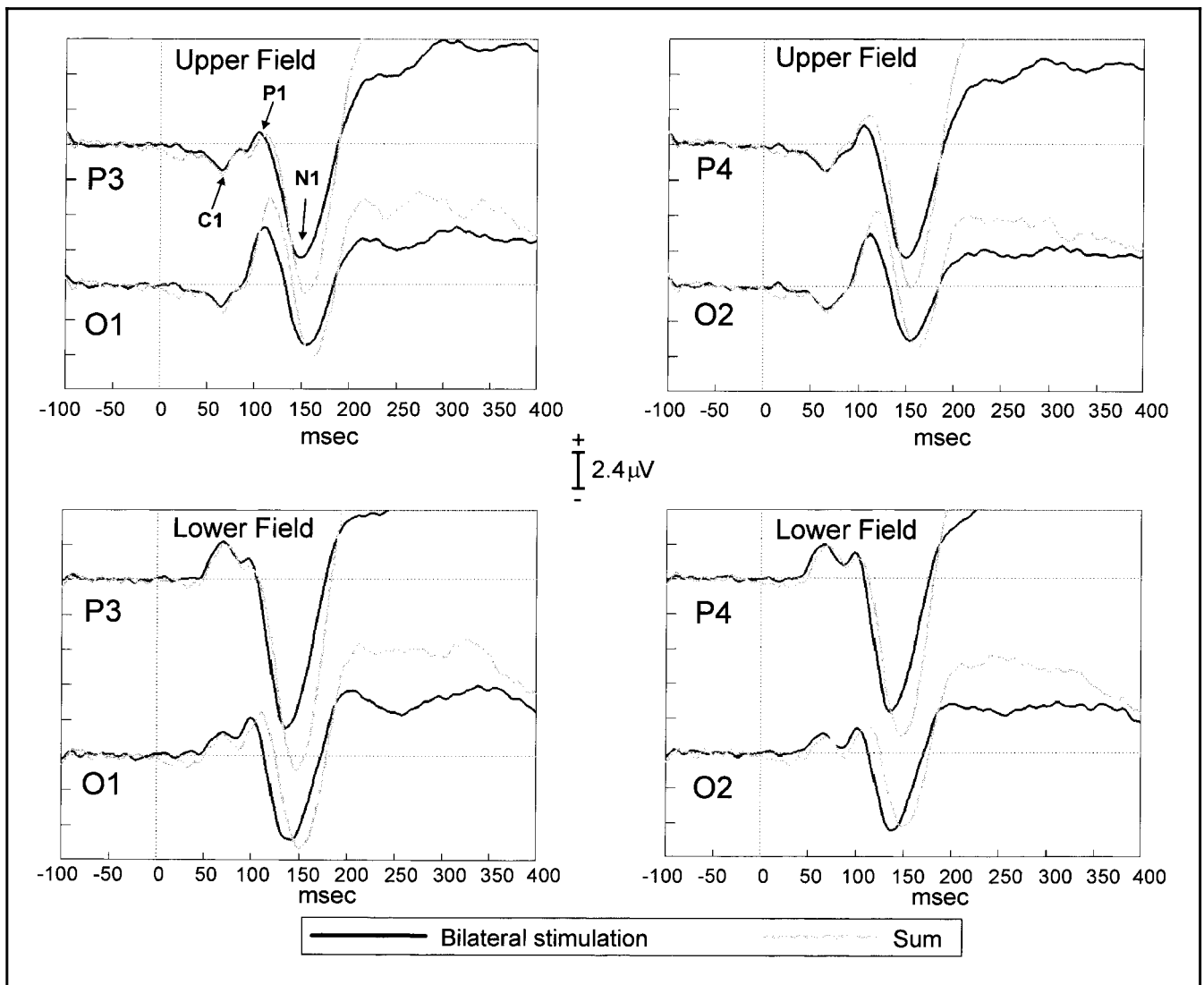
Figure 9. Samples of grand average ERP waveforms from the same electrode locations as in Figure 7. Thick lines show responses to bilateral stimulation and thin lines show responses to algebraically summed left and right unilateral stimuli. Note the lack of amplitude differences for the C1 component, whereas for the P1 and N1 components the responses to bilateral stimuli yield larger amplitudes and shorter latencies than those to the summed responses to unilateral stimuli.

visual components of the ERP, more probably P1 and N1 than C1, which is more affected by volume conduction effects with bilateral stimuli than are the other two components. Interestingly, the ERP responses show a similar interaction between hemifield of stimulus presentation (upper vs. lower) and stimulus type (unilateral vs. bilateral) as the behavioral results. This brings further weight to the possibility of a mediation of the RTE at the level of extrastriate cortical visual areas. However, one should consider that, in principle, the earliest stage of summation might be located even at subcortical visual sites, such as the superior colliculus. The presence of an RTE in patients who underwent callosotomy (Reuter-Lorenz et al., 1995; Marzi et al., 1997) or hemispherectomy (Tomaiuolo et al., 1997) suggests a subcortical locus of coactivation. It is notoriously difficult to record

ERP activity generated by subcortical structures (Coles & Rugg, 1995), and it is unlikely that coactivation effect caused by the RTE occurring at the level of the superior colliculus could be detected by our ERP recordings. The possibility of a collicular mediation of the RTE is consistent with the above-mentioned evidence of spared RTE in patients with lesions of the primary visual cortex.

The larger RTE found for lower- than for upper-field presentations was an unexpected finding. Such an advantage is independent of vertical asymmetries with unilateral stimuli, which turned out to be absent. It is difficult to speculate on its mechanism and functional significance, but certainly it represents an important correlation between behavioral and electrophysiological data. We found up-down asymmetries favoring the lower hemifield not only as far as the RTE is concerned but

LATENCY
Lower Visual Field

C1

msec 78 72 66 60

TC1 C3 CZ C4 TC2
CP1 CP2
P5 P3 PZ P4 P6
T5
TO1 OZ TO2 T6
O1 O2

P1

msec 120 110 100 90

TC1 C3 CZ C4 TC2
CP1 CP2
P5 P3 PZ P4 P6
T5
TO1 OZ TO2 T6
O1 O2

N1

msec 160 150 140 130

TC1 C3 CZ C4 TC2
CP1 CP2
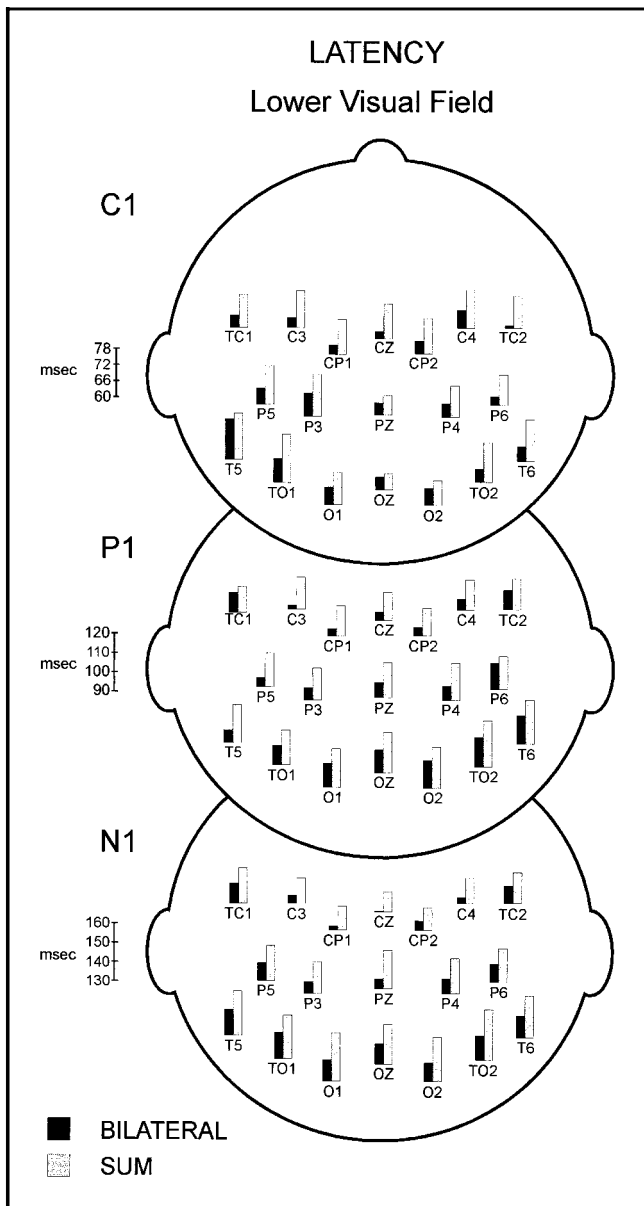P5 P3 PZ P4 P6
T5
TO1 OZ TO2 T6
O1 O2

■ BILATERAL
▨ SUM

Figure 10. Comparison between responses to bilateral stimuli and the sum of the responses to the two unilateral stimuli: Mean latency of the C1, P1, and N1 components (same electrodes and same field presentation as in Figure 8). Note that for all electrodes and components the responses to bilateral stimuli yield shorter latencies than those to the summed responses to unilateral stimuli.

also in the amplitude (N1 component) and latency (P1 and N1) of response to unilateral stimuli. These data clearly outline an overall advantage of the lower hemifield in ERP responses, and this fits well with single-cell evidence provided by Van Essen, Newsome, and Maunsell (1984) of a greater representation of the lower hemifield in the visual cortex of macaques and with multisource evidence of an overall greater sensitivity of the lower hemifield (upper retina) in comparison with the upper hemifield (lower retina) as reviewed by Skrandies (1987).

An important general consideration about the present findings is that the latency advantage of the early visual ERP components for redundant signals in our study does not in principle rule out the possibility of a second stage of coactivation at a decisional or motor level. However, in a series of recent experiments requiring target discrimination Mordkoff et al., (1996) measured behavioral response, response-force, and premotor ERPs' latency and provided evidence of a lack of coactivation at the motor level in a go/no-go task. The only thing that seemed to matter for an effect at the motor level was the number of stimuli. Such a result clearly reinforces the present evidence of a perceptual locus for the RTE. Given that the RTE occurs at a visual stage, an important point is whether it is entirely stimulus driven or can be affected by top-down influences. We have recent evidence (Prior & Marzi, submitted) showing that the RTE is susceptible to the allocation of spatial attention. Normal subjects performing in an RTE task broadly similar to that described above were asked to tonically allocate their attention either to the left or to the right visual hemifield. This resulted in a speeding up of RTs to single stimuli presented to the attended hemifield (valid trials) and in a slowing down of RTs to stimuli presented to the unattended side (invalid trials). The main thrust of the experiment was that bilateral presentations yielded a speed advantage over single presentations when attention was allocated to both fields simultaneously, whereas when attention was allocated to one side only, RTs to valid single stimuli were not significantly different from those to bilateral stimuli. In other words, single stimuli presented to the unattended side did not speed up RTs to attended single stimuli. This shows that under conditions of bilateral presentations and unilateral allocation of attention the RTE is not automatic and stimulus bound but is influenced by spatial attention. That is in keeping with our present ERP results showing that the RTE occurs at neural sites indexed by the P1 (i.e., a component that has been shown to be clearly influenced by spatial attention) (Hillyard, Mangun, Woldorff, & Luck, 1995; Mangun, 1995).

METHODS

Subjects

Twelve healthy right-handed adults (age range 23 to 33 years), 6 females and 6 males, took part in the experiment. Their visual acuity was normal or corrected to normal.

Stimuli

As shown in Figure 1, the stimuli consisted of rectangular black-and-white checkerboards presented on a video monitor. The spatial frequency was 2.15 cycles/degree of

visual angle, and the exposure duration was 57 msec. Each rectangle subtended 2.5° horizontally and 4° vertically and was presented 3.6° left or right of the fixation point (measured from the inner side of the rectangle) and 1.7° above or below the horizontal meridian (measured from the near edge). A single rectangle was presented on unilateral trials and a pair of rectangles was presented on bilateral trials. When a pair was presented, both rectangles were in either the upper or the lower field. These different stimulus types were randomly intermixed within each trial block. The interstimulus interval was randomized and ranged between 1500 and 2400 msec (in steps of approximately 100 msec). The mean luminance of the checkerboard was 20 cd/m^2 against a dark gray background with a luminance of 0.2 cd/m^2.

Procedure

Subjects were instructed to make a simple manual RT to the onset of the visual stimuli. They were required to steadily fixate on a small cross that was continuously present at the center of the video screen and to press a key with the index finger of the right (dominant) hand as soon as possible following onset of either a unilateral or a bilateral stimulus. To minimize spatial stimulus-response compatibility effects, which are in any case minimal in a simple RT paradigm (Berlucchi, Crea, Di Stefano, & Tassinari, 1977), the response key was centrally located. The subjects were seated in front of the computer screen with their eyes at 57 cm from the center of the screen.

Each subject was given 10 trial blocks, each consisting of 20 trials for each of the six conditions of stimulus presentations: unilateral left visual field (LVF), lower and upper; unilateral right visual field (RVF), lower and upper; and bilateral stimuli, lower and upper. The whole testing session lasted about 2 hours, including a short rest break after each trial block.

The subjects were instructed to suppress saccades toward the site of stimulus presentation and to avoid blinking. Trials with RTs faster than 150 msec or slower than 500 msec were excluded from statistical analysis of both behavioral and electrophysiological data.

ERP Recording and Analysis

The electroencephalogram (EEG) was recorded with nonpolarizable tin electrodes mounted in an elastic cap and positioned according to the 10–20 International system. Four additional pairs of electrodes were located in nonstandard sites: P5 and P6, in the middle of the line between C3 and T5 and the line between C4 and T6, respectively; TC1 and TC2, in the middle of the lines that join C3 and C4 with T3 and T4, respectively; CP1 and CP2, 1 cm toward the inion from the midpoint between Cz and C3 and between Cz and C4, respectively; and TO1 and TO2, at the center of the lines that join T5 and T6 with O1 and O2, respectively.

The right mastoid served as the reference for all electrodes. Recordings obtained from a left mastoid electrode were used off-line to re-reference all the scalp recordings to the average of the left and right mastoids. Horizontal eye movements were detected by recording the electrooculogram (EOG) as the voltage difference between two electrodes located 1 cm lateral to the left and right external canthi. An electrode located beneath the left eye and referenced to the right mastoid served to detect vertical eye movements and blinks.

The signal was digitized at a sampling rate of 500 Hz and amplified by a Neuroscan SynAmps amplifier. The data were recorded with a bandpass from dc to 100 Hz and filtered again off-line using a high-pass filter at 0.01 Hz and a low-pass filter at 60 Hz (12 dB per octave). The averaging of the signal, time-locked to the onset of the stimuli, was performed off-line using epochs starting 200 msec before and ending 824 msec after stimulus onset. Epochs with eye movement artifacts (blinks or saccades) and incorrect behavioral responses were rejected (this artifact-rejection procedure led to the rejection of about 10% of trials). The averaging was performed separately for each of the six conditions of stimulus presentation and subject; see above.

The latency of the C1 component was calculated as the time point of the largest positive voltage deflection for lower-hemifield presentation and the largest negative voltage deflection for upper-hemifield presentation, using a window of 45 to 90 msec poststimulus. The latency of the P1 component was calculated as the time point of the largest positive peak between 80 and 150 msec, and the latency of the N1 component was calculated as the time point of the largest negative peak between 110 and 180 msec. The amplitude values were calculated with reference to the prestimulus baseline for all components at the same peaks used for the latency measurements.

Only the data from eight pairs of electrodes (C3-4, P3-4, O1-2, T5-6, CP1-2, TC1-2, P5-6, and TO1-2) were statistically analyzed. Both amplitude and latency data were analyzed by means of a four-way within-subjects ANOVA using the Greenhouse-Geisser epsilon correction for nonsphericity (Jennings & Wood, 1976). The ANOVA factors were: Visual Hemifield (lower vs. upper), Stimulus Type (bilateral vs. unilateral), Hemisphere (left vs. right), and Electrode (position of the eight pairs of electrodes, as indicated above).

The latency as well as the amplitude data were analyzed by comparing the bilateral presentation values with the better unilateral (direct) response and with the algebraic sum of the two unilateral inputs.

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