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INTERHEMISPHERIC TRANSFER OF VISUAL INFORMATION IN HUMANS: THE ROLE OF DIFFERENT CALLOSAL CHANNELS

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INTRODUCTION

There is a vast body of evidence in the literature showing that the corpus callosum (CC) is a crucial structure for the transfer of visual information from the hemisphere directly receiving the visual input to the other one. Such evidence has been provided by numerous sources including behavioural and electrophysiological assessments of interhemispheric transfer time (IHTT) in either animals or humans. In humans, the now classic studies of Sperry and his collaborators (13) have demonstrated beyond any reasonable doubt that the surgical resection of the CC, carried out as an extreme form of treatment to prevent spreading of otherwise untreatable epileptic seizures, results in the inability of one hemisphere to access information presented only to the other hemisphere. Such inability is complete for complex visual tasks tapping the specialization of one or the other hemisphere (e.g. language and the left hemisphere), while is partial when a simple interhemispheric visuo-motor integration is required. It has been shown repeatedly that in the Poffenberger task (9), in which a subject is supposed to press a key as fast as possible with one hand following visual stimulation of either the ipsilateral or the contralateral visual hemifield, CC-sectioned patients are considerably delayed in comparison to controls when detection of the visual stimulus and manual response are subserved by different hemispheres (8). Such lenghtening can reasonably be interpreted as resulting from the lack of the CC preventing a fast IHTT since these patients do not have particular difficulties integrating the visuo-motor response within a single hemisphere. A similar, albeit less pronounced, IHTT lengthening has been described in patients with a genetic absence of the CC (8). Not only in callosum sectioned patients but also in normal subjects, following the pioneering observations of Poffenberger (9), it can be consistently observed that RT for crossed visuo-motor combinations (visual hemifield of stimulus presentation and responding hand on contralateral sides) is slower than for uncrossed combination (1, 3, 8). Such crossed-uncrossed difference (CUD) is tipically about 3-4 msec and is believed to represent a reliable behavioural estimate of IHTT in normal subjects. An important point is that such a value is much smaller than that obtained by means of electrophysiological methods (Event-Related Potentials = ERPs) of IHTT assessment in human subjects studied with lateralized visual presentations.

That IHTT can be determined with such a technique is witnessed by the observation that the latencies of the ERP components are longer in the ipsilateral than in the contralateral hemisphere (4, 10, 12). Moreover, the absence of physiological, i.e., as opposed to volume conducted, ipsilateral ERP responses to lateralized visual stimuli in patients lacking the corpus callosum for genetic (11) or surgical (6) reasons, confirms that the ipsilateral response is normally due to a transcallosal flow of information.

ERP-IHTT measured as the difference between ipsilateral and contralateral latencies for the N1 component ranges between 8 and 21 msec in the occipital regions and between 2.5 and 3.6 msec in central sites (4). P1 estimates of IHTT have given smaller values than those found for the N1, namely 6.5 vs 9.3 msec for medial sites and 11.1 vs 14 msec for lateral sites (12).

An interesting problem concerns the relationship between behavioural and electrophysiological estimates of IHTT: Rugg *et al.* (10) recorded ERPs to lateralized visual stimuli from lateral, occipital and central scalp sites during a choice RT task, and found that the N1-IHTT measured at central electrode sites was more similar to CUD values than IHTT measured over the occipital sites. In addition, the N1-IHTT at central sites was not significantly affected by visual stimulus intensity, as is the case for behavioural IHTT, while it was affected at occipital sites. This suggests that transfer may occur via different pathways and that the behavioural assessment might rely on a pathway at premotor rather than visual sites.

We believe that such discrepancies between behavioural and ERP estimates of IHTT can cast important light on the callosal mechanisms of IHTT in that they may reveal the presence of different channels employing different interhemispheric pathways. The present study has two aims: Firstly, by using a higher number of electrode recording sites than in previous studies, to attempt a more extensive assessment of the locations yielding a faster IHTT. Secondly, to verify the possibility raised by previous behavioural studies (2, 8) that the callosal mechanisms subserving interhemispheric transfer are asymmetric. In particular, we hypothesized that in a simple visual RT task, IHTT is faster from the right (RH) to the left hemisphere (LH) and this would be in keeping with an overall superiority of the RH for visual-spatial functions. Clear support for this hypothesis has been provided recently by Brown *et al.* (4) in a meta-analysis of 18 ERP studies of IHTT showing a significative predominance of experiments documenting a faster right-to-left rather than left-to-right IHTT. In the present experiment we have expanded Brown *et al.* (4) results by verifying which electrode locations yield clearer evidence of a right-left transfer asymmetry.

METHODS

Subjects.

10 right-handed young normal adults, 3 females and 7 males, of age ranging between 20 and 33 years, with normal or corrected vision took part in the experiment.

Stimuli and task.

The stimuli consisted of 2x2 cm luminous rectangles generated by a VGA monitor with an exposure duration of 56 msec. A rectangle could appear at 8° lateral to the fixation point either in the right (RVF) or in the left visual hemifield (LVF) in a random sequence. The interstimulus interval ranged between 1500 and 2400 msec. The subjects viewed the video monitor from a distance of 57 cm so that the stimuli subtended a visual angle of approximately 2° by 2°. The luminance of the stimuli was 98 cd/m^2 and that of the background was about 0.3 cd/m^2.

The task was a simple manual RT task: The subjects were required to fixate a small cross at the centre of the video monitor and to press a centrally located key with their index finger as fast as possible following stimulus onset. The subjects were to suppress eye movements and blinks. When they could not refrain from moving their eyes toward the side of stimulus presentation, the trial was discarded without replacement. Such, however, was a very rare occurrence.

Each subject performed in 4 blocks; the responding hand was alternated between right and left in successive blocks with the order balanced across subjects. Each block consisted of 50 trials for each hemifield for a total of 200 trials of unilateral presentations for each subject for the whole recording session. The two stimuli were alternated in a random sequence. A third type of stimulation, namely bilateral interfield presentation (50 trials per block, randomly alternated with the unilateral presentations), will not be dealt with in the present account and will be separately described elsewhere. Each block lasted about 5 minutes. Trials with RT faster than 150 msec and lower than 500 msec were considered as anticipations and retards, respectively, and excluded from all the analyses.

ERP recording.

The computerised electroencephalogram (EEG) was recorded with tin electrodes positioned according to the 10-20 International system onto an elastic cap. Four additional pairs of electrodes were placed in the temporo-central-parietal sites (P5 and P6, in the middle of the line

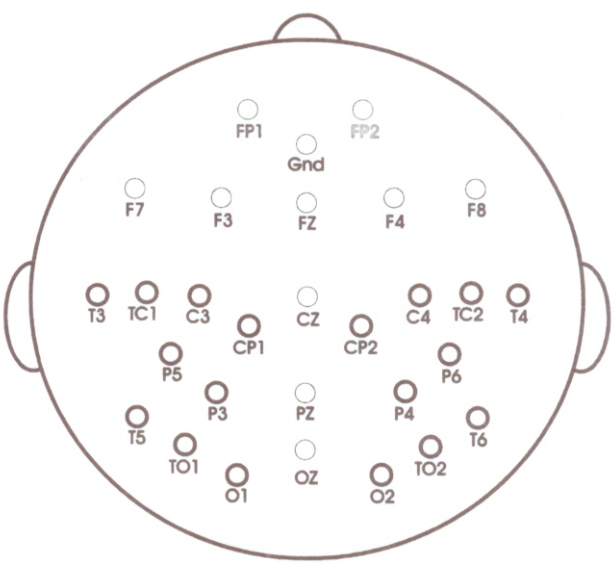

Fig. 1. - *Schematic drawing of the localization of the electrode sites used in the present study (barring EOG and reference electrodes).*

Circles with heavy lines indicate the electrodes used for statistical analysis.

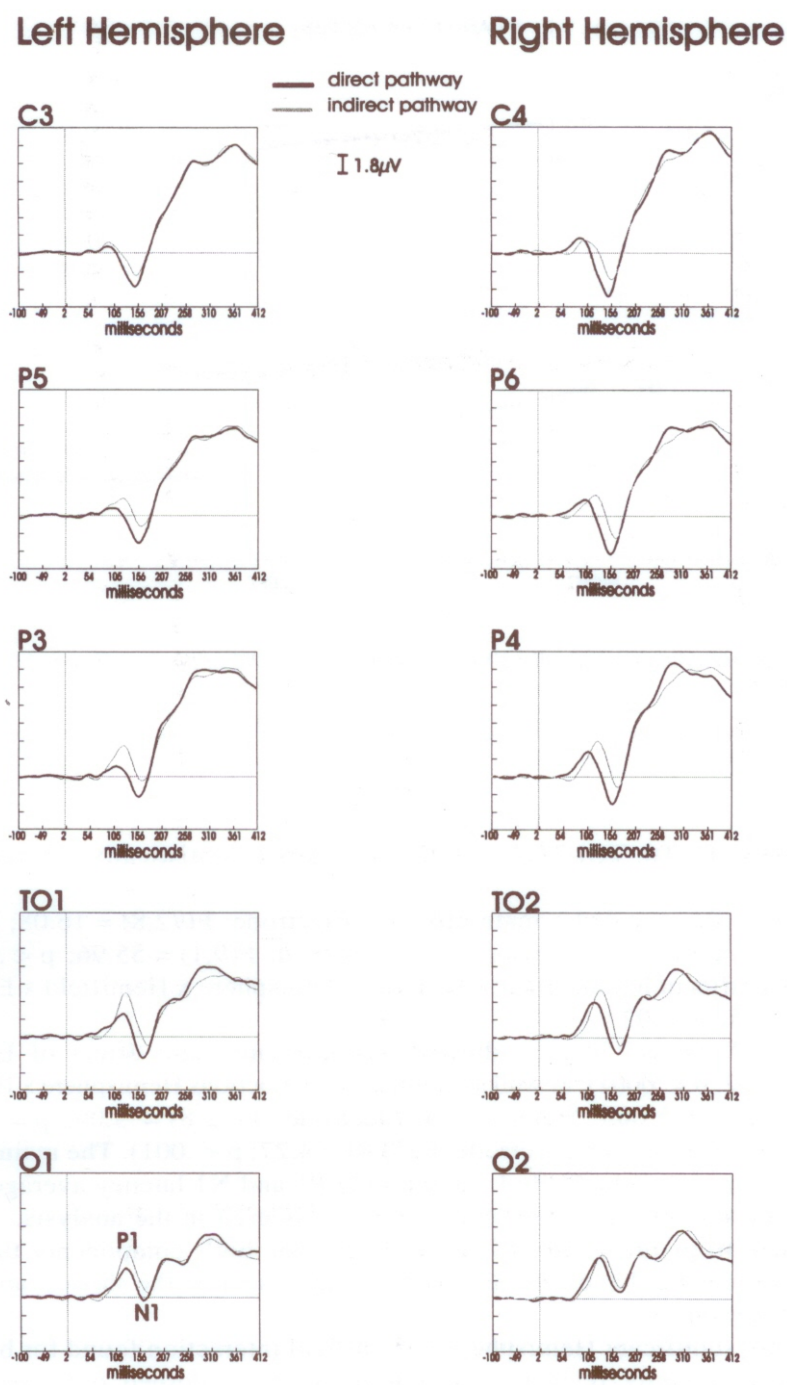

Fig. 2. - *Examples from five recording sites (from anterior to more posterior locations) of grand-average ERP's to stimuli presented to the hemifield contralateral (direct pathway) or ipsilateral (indirect-callosal pathway) to the recording hemisphere.*

between C3 and T5, and between C4 and T6, respectively), in the fronto-temporal-central site (TCl and TC2, in the middle of the line that joins C3 and C4 with T3 and T4, respectively), in the central-parietal site (CP1 and CP2, below the centre of the line between Cz and C3, and Cz and C4, respectively), and in the lateral-occipital sites (TOl and TO2, at the centre of the line that joins T5 and T6 with Ol and O2, respectively) see Figure 1.

The electrodes were referred to linked ears references. Horizontal and vertical ocular movements and ocular blinks were monitored by means of bipolar electrooculogram (EOG).

The signal was digitised at the-rate of 500 Hz (one point every 2 msec). The high-pass filter was at the DC level while the low-pass filter during the acquisition was at 100 Hz. The averaging of the signal, time locked to the onset of the stimuli, was performed off line after the continuous signal had been divided in epochs starting 100 msec before and ending 412 msec after stimulus onset.

Epochs with eye movement artefacts (blinks or saccades) were rejected. The signal was filtered off-line with a low-pass filter set at 70 Hz. The averaging of the epochs time-locked with stimuli appearing in the RVF were performed separately from the averaging of the epochs time locked with LVF stimuli. The latencies of the early components were calculated as the higher positive peak in a temporal window between 80 and 150 msec for the P1 component and as the highest negative peak between 120 and 180 msec for the N1 component. The P1 amplitude values were calculated with reference to the mean value of the 50 msec post-stimulus baseline, while the N1 amplitude was measured with reference to the P1 values as peak to peak difference.

Only the data from 9 couples of electrodes (indicated by a thick line in Figure 1), in addition to those for oculographic recordings, were analyzed since the others did not show visually triggered responses.

RESULTS

Since in a preliminary analysis we found no significant latency or amplitude effect of the responding hand on ERP components, we averaged across the two hand conditions.

Figure 2 shows the ERP waveforms for five electrode sites in the two hemispheres. The P1 and N1 components are clearly visible as the first positive (positive is up) and first negative (down) deflections following stimulus presentation. The direct pathway (RVF-LH and LVF-RH) is represented by the thick black line and the indirect pathway (RVF-RH and LVF-LH) is represented by the thin grey line. Both components have a shorter latency for the direct pathway while overall amplitude is higher for the indirect pathway.

1. *ERP latency data.*

Figure 3 shows the mean values averaged across subjects and across all electrode sites of the P1 and N1 peak latency for both direct and indirect visual stimulation.

A simple inspection of the figure reveals that for both components the ipsilateral hemifield-hemisphere combination yields slower latencies than the contralateral combination.

A repeated measure three-ways ANOVA was carried out with Hemifield (RVF, LVF), Hemisphere (LH,RH) and Electrode (CP1-CP2, C3-C4, TCl-TC2, T3-T4,

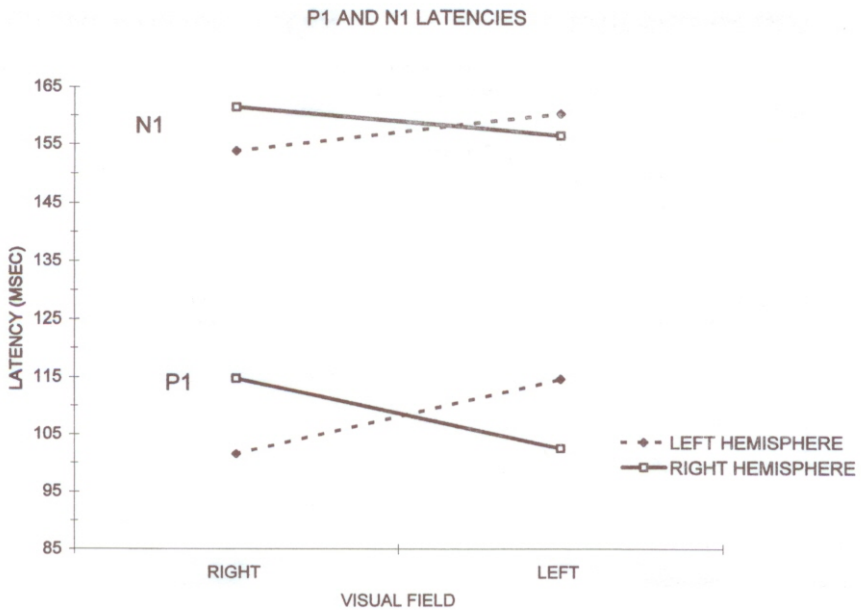

Fig. 3. - *Overall mean latencies for the ERP components Pl and N1 in the four Hemifield-Hemisphere combinations*

For both components the latency is shorter in the hemisphere contralateral to the stimulated visual hemifield.

P5-P6, P3-P4, T5-T6, TOl-TO2, Ol-O2) as factors to statistically substantiate the above data.

Analysis of P1 revealed a main effect of Electrode: $F(72,8) = 16.08$; $p < .001$; a significant interaction Hemisphere x Hemifield: $F(9,1) = 55.96$; $p < .001$; and finally, a significant higher-order interaction Hemisphere x Hemifield x Electrode: $F(72,8) = 2.35$; $p = .02$.

Analysis of the N1 latency showed a significant main effect of Electrode: $F(72,8) = 17.62$; $p < .0001$), as well as significant interactions Hemisphere x Hemifield: $F(9,1) = 13.79$; $p = .005$, Hemifield x Electrode: $F(72,8) = 3.09$; $p = .005$ and Hemisphere x Hemifield x Electrode: $F(72,8) = 4.27$; $p < .001$). The main effect of Electrode is described in Table I showing the P1 and N1 latency averaged across the two hemispheres for the electrode sites considered in the analysis.

It is clear from inspection of Table I that for both components the fastest latencies are in central and central-parietal locations while the slowest are in more posterior locations.

The highly significant Hemisphere x Hemifield interaction found for both components can be easily related to a shorter latency recorded over the directly stimulated hemisphere in comparison to the indirectly stimulated hemisphere and this provides evidence for interhemispheric transfer.

Table I. - *Mean latency values for P1 and N1 across electrode sites ordered from short (C3/C4) to progressively longer latencies.*
Notice that the increase in latency is similar for the two components in corresponding electrodes.

P1			N1		
Electrode site	mean (msec)	sd	Electrode site	mean (msec)	sd
C3/C4	96.65 msec	10.9	C3/C4	150.45 msec	8.9
CP1/CP2	97.3 msec	11.28	CP1/CP2	150.5 msec	12
T3/T4	104.3 msec	11.7	TC1/TC2	152.55 msec	8
TC1/TC2	105.9 msec	11.6	T3/T4	154.1 msec	10.6
P5/P6	107.3 msec	9.5	P5/P6	156.75 msec	8.4
P3/P4	108.95 msec	10.2	P3/P4	159.4 msec	9.9
T5/T6	112.7 msec	7	T5/T6	164.8 msec	9.2
TO1/TO2	119.9 msec	5.7	O1/O2	165.5 msec	8.9
O1/O2	121.95 msec	7.9	TO1/TO2	167 msec	8.6

The most interesting interaction is certainly the Hemisphere x Hemifield x Electrode interaction which is clearly related to differences in IHTT among the various electrode locations.

We analysed this interaction by calculating IHTT for each electrode location (averaged across the two hemispheres); such estimate was obtained by subtracting the latency of the direct ERP response from the latency of the indirect one both for the P1 and N1 components.

Figure 4 shows IHTT for the various locations ordered according to the observed P1 values from small to progressively larger IHTT values. Note that for all locations, P1 estimates are larger than N1 and that all P1-IHTT values are positive, i.e., show a substantial interhemispheric transfer.

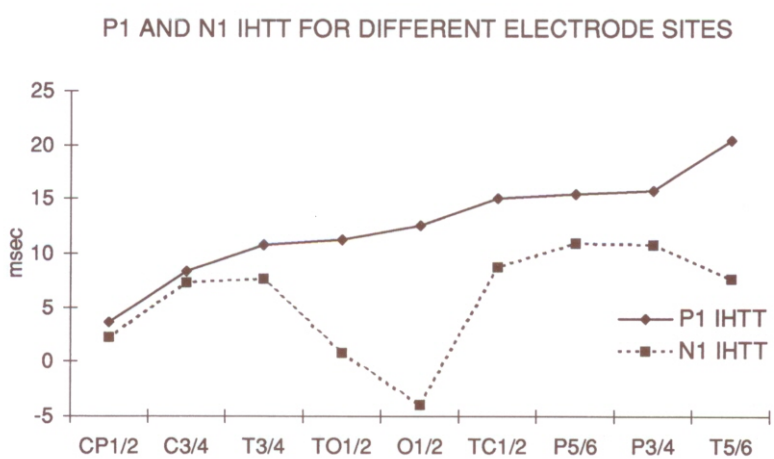

Fig. 4. - *Interhemispheric transfer time (IHTT) for P1 and N1 as function of electrode site.*

The various locations have been ordered according to the observed IHTT for the P1 component. For all sites IHTT is longer for P1 than N1.

A similar consideration applies to N1 with the exception of OI/2 where there was a negative IHTT (i.e., the indirect pathway yielded a slightly shorter IHTT than the direct one perhaps as a result of direct electric spread from the opposite hemisphere). Overall, the P1-IHTT had a mean value of 12.54 msec as opposed to a mean value of 5.77 msec for N1. As shown in Figure 4, for both components more anterior leads show faster values of IHTT than posterior ones and such differences were all highly significant at post-hoc t-test.

In contrast to what was found in the meta-analysis of both behavioural and VEP data (3, 4, 8), there was no clearcut overall evidence of an asymmetric IHTT. However, as one can gather from Table II showing IHTT in the two directions for both P1 and N1 components and the various electrode sites, one can notice that the C3-C4 sites yielding the shortest (positive) account of IHTT for N1 show a markedly faster R-L than L-R IHTT. This is logically the best electrophysiological correlate of behaviourally assessed IHTT and therefore this result is in keeping with the hypothesis of an asymmetric callosal transmission.

Table II. - *Mean values of interhemispheric transfer time (IHTT) for the right-to-left and left-to-right directions for P1 and N1 across the various electrode sites.*
Notice that the shortest estimate of IHTT, namely C3/C4 for N1 yields a faster value for right-to-left than for the opposite direction. This is in agreement with behavioural estimates (8).

P1-IHTT				N1-IHTT			
Electrodes	R-L	L-R	mean	Electrodes	R-L	L-R	mean
C3/C4	8.0	8.6	8.3	C3/C4	5.8	8.8	7.3
CP1/CP2	6.4	.80	3.6	CP1/CP2	-6.0	5.0	2.2
TC1/TC2	14.2	15.8	15.0	TC1/TC2	6.4	11.0	8.7
T3/T4	11.2	10.4	10.8	T3/T4	6.2	9.0	7.6
T5/T6	16.4	24.4	20.4	T5/T6	7.6	7.6	7.6
P5/P6	15.4	15.4	15.4	P5/P6	9.4	12.4	10.9
P3/P4	17.2	14.2	15.7	P3/P4	11.0	10.6	10.8
TO1/TO2	14.4	8.0	11.2	TO1/TO2	5.4	-3.8	0.8
O1/O2	14.2	10.8	12.5	O1/O2	7.0	-15.0	-4

2. *ERP amplitude data.*

Table III shows overall amplitude values for P1 and N1 as a function of Hemisphere. For both components there was a larger amplitude in the RH than in the LH hemisphere while overall difference as a function of hemifield yielded a higher value in the LVF for P1 and negligible differences for N1.

A repeated measures three-ways ANOVA (Hemifield, Hemisphere and Electrode) was carried out on amplitude data.

The analysis of P1 revealed a main effect of Hemifield: $F(9,1)= 5.59$; $p=.042$, with the LVF yielding a higher amplitude than the RVF; a main effect of Hemisphere: $F(9,1) = 20.48$; $p < .001$ with the RH showing a largér amplitude than the LH and of Electrode: $F(72,8) = 20.08$; $p < .001$ with the various electrode sites

Table III. - *Mean overall voltage amplitude for P1 and N1 in the four hemifield-hemisphere combinations.*
Notice that for both components amplitude is larger in the RH than in the LH while the difference between hemifield is minimal.

P1				N1			
Hemisph.	RVF (uV)	LVF (uV)	mean (uV)	Hemisph.	RVF (uV)	LVF (uV)	mean (uV)
RH	3.38 uV	2.82 uV	3.1 uV	RH	-5.99 uV	-6.41 uV	-6.2 uV
LH	1.84 uV	2.92 uV	2.38 uV	LH	-5.03 uV	-4.53 uV	-4.78 uV
mean	2.61 uV	2.87 uV		mean	-5.51 uv	-5.47 uV	

showing different amplitude values, see Figure 5. The interaction Hemisphere x Hemifield was also significant: $F(9,1)= 5.67$; $p=.041$; as well as the Hemifield x Hemisphere x Electrode higher-order interaction: $F(72,8)= 6.41$; $p<.001$. The former can be explained by the amplitude of the indirect response being higher than that of the direct response. The meaning of the higher-order interaction can be grasped more easily by inspection of Figures 6-7 showing amplitude values of the P1 and N1 components for the direct and indirect pathways in the various electrode sites. Inspection of Figure 6 shows that the amplitude of the indirect pathway is higher than that of the direct one at all sites except three and that the amount of such difference was clearly higher for the parietal leads.

The analysis of N1 amplitude showed a significant main effect of Hemisphere: $F (9,1) = 20.26$; $p < .001$, with an overall higher amplitude in the RH, see Table III, of Electrode: $F(72,8) = 5.49$; $p < .001$, see Figure 5, and a significant Hemifield

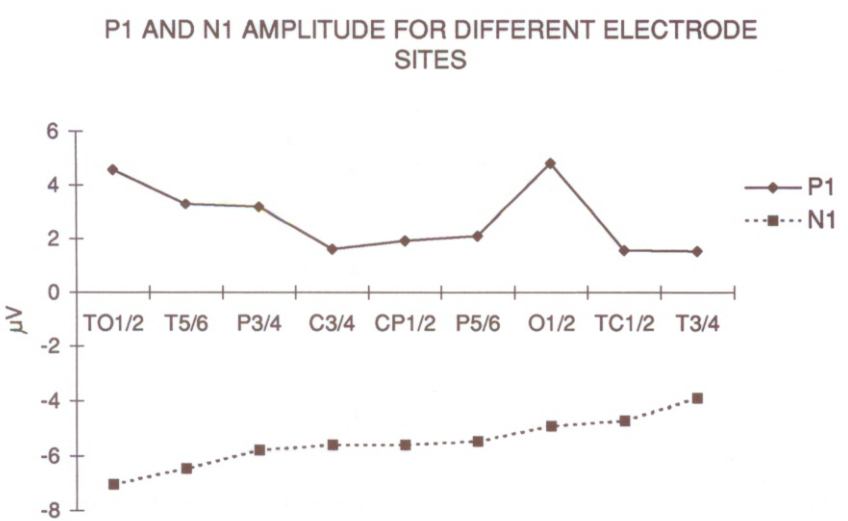

Fig. 5. - *Voltage amplitude of P1 and N1 for various electrode sites (ordered according to amplitude values of N1).*

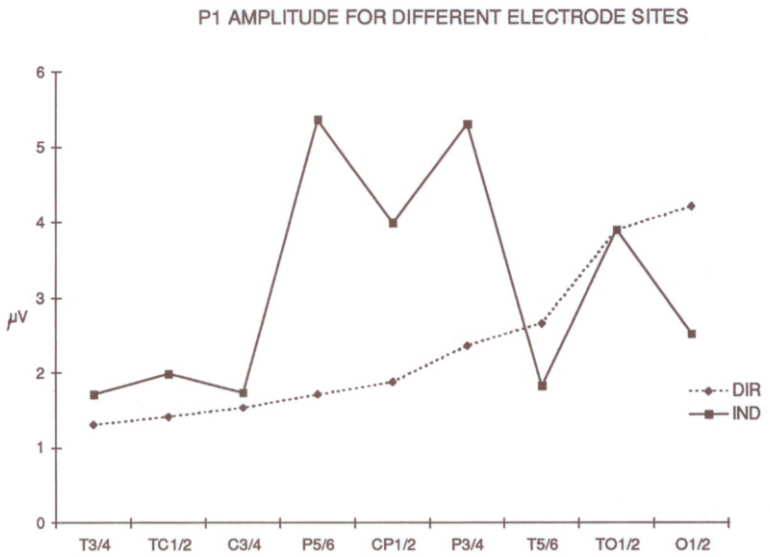

Fig. 6. - *Amplitude of P1 at various electrode sites for the direct (hemifield of stimulus presentation contralateral to recording hemisphere) and indirect (hemifield ipsilateral to recording hemisphere) pathways.*

The locations have been ordered on the abscissae according to the values for the direct pathway. Notice that for all sites but two, the amplitude was larger for the indirect pathway.

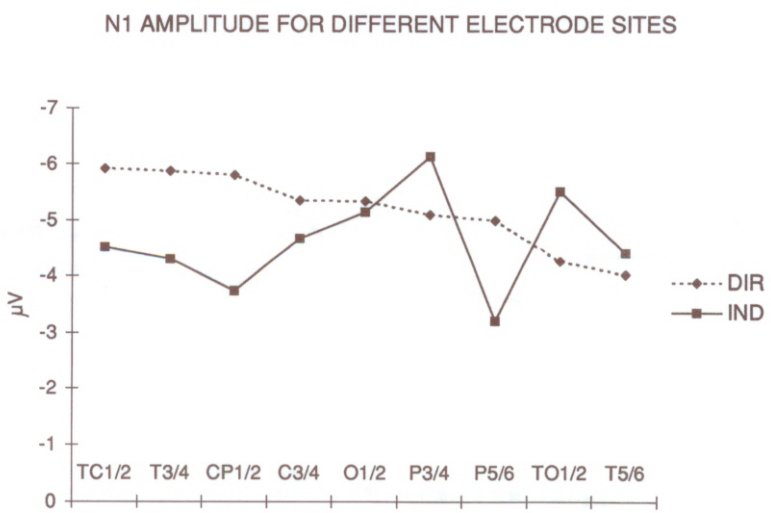

Fig. 7. - *Amplitude of N1 at various electrode sites for direct and indirect pathways.*

The locations have been ordered according to values for the direct pathway. Notice that for all sites but three the direct pathway yields a larger amplitude than the idirect pathway.

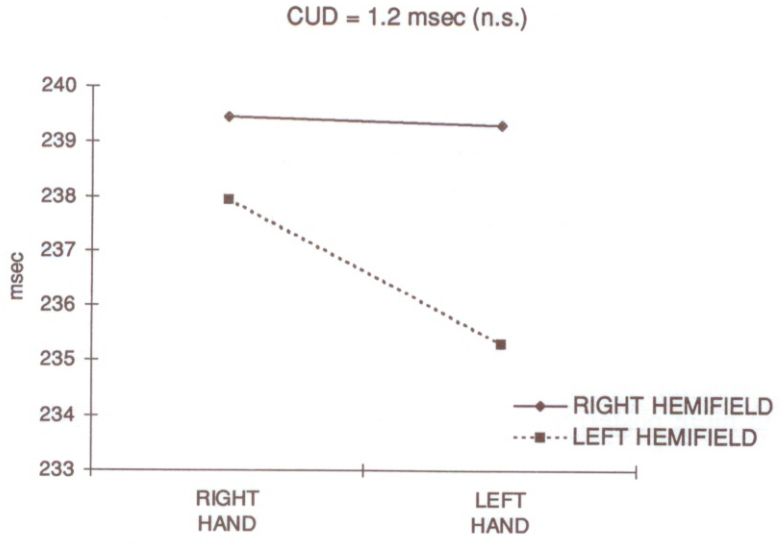

Fig. 8. - *Behavioural assessment of IHTT measured as CUD defined as the difference between crossed (hemifield-contralateral hand) and uncrossed (hemifield-ipsilateral hand) visuo-manual responses.*

The crossed condition requires an interhemispheric transfer while the uncrossed conditions implies an intra-hemispheric pathway. Notice that the CUD is similar for the two hands in the right hemifield but is asymmetric for the left hemifield. Overall, the uncrossed condition is 1.2 msec faster than the crossed one and this is in keeping with previous findings (see Ref. 8).

x Hemisphere x Electrode interaction: $F(72,8) = 4.16$; $p < .001$, see Figure 7. Here, at variance with P1, there was an overall higher amplitude for the direct pathway.

3. *Behavioural results.*

The mean RTs for the four Hemifield-Hand combinations are shown in Figure 8. An ANOVA with Hemifield and Hand as factors showed no statistically significant main effects or interactions; the mean overall CUD was 1.2 msec, a value which is somewhat smaller than that typically found in such studies. In keeping with previous findings, however, the RVF-Left Hand yielded a longer RT than the other crossed combination (8).

DISCUSSION

There are several aspects of this study that deserve discussion: First, the present results provide further evidence that IHTT can be measured by electrophysiological means; the IHTT values found are well in keeping with those of previous assessments (4). By using more numerous electrode locations than in previous studies we were able to better describe the sites yielding various values of IHTT for either the

P1 or the N1 component of the ERP. A second, clearcut finding was that N1 gave overall shorter values of IHTT than P1 (about 7 msec difference) and therefore one is tempted to consider the former component as a more likely electrophysiological correlate of behavioural IHTT than the latter. N1 is believed to reflect different cortical processing stages than P1 and is considered at an immediately successive hierarchical level in visual cortical information processing (6). Our present results confirm previous findings by showing that, unlike P1 which has a more restricted distribution over posterior scalp sites, N1 has a broad distribution encompassing occipital, parietal, temporal and frontal areas, as we could ascertain in our present experiment from an analysis of the distribution of voltage topographic maps to unilateral stimuli at various times following stimulus presentation. The differences in distribution between P1 and N1 are clear with the former component lateralized to more posterior scalp areas in comparison with the latter.

It has been hypothesized (6) that the voltage generator for P1 is in extrastriate visual areas in the fusiform gyrus while the N1 might have either two generators or a more anterior one in the inferotemporal cortex. These generators would be responsible for the more anterior and widespread scalp localization of N1 in comparison to P1. If N1 reflects the activity of visual areas outside the occipital lobe, our finding of a shorter N1-IHTT would be in nice accord with the general idea (7) that the bulk of callosal transfer of visual information occurs at extraoccipital sites. An interesting consideration concerns the functional meaning of the two channels of interhemispheric transfer: a faster and anterior N1- and a slower and posterior P1-channel. A tentative explanation might stem from the consideration that the emphasis on speed typical of RT paradigms requires a fast channel subserving transfer from the hemisphere of stimulus entry to that controlling the manual response. Such a pathway might be represented, on a trial-to-trial basis, by whatever channel happens to be able to transfer information more quickly on a horse-race fashion (5).

Only specific studies trying to correlate the modifications of the latency of P1-IHTT and N1-IHTT with corresponding variations in behavioural IHTT may cast further light on the neural mechanism of interhemispheric transfer. For example, it may be important to assess whether modifications in the visual component of the Poffenberger task may affect both behavioural IHTT and either Pl- or N1-IHTT. By the same reasoning, manipulation of the motor component might in principle affect selectively IHTT as lndexed by one or the other ERP component monitored in the present study.

A final consideration concerns the IHTT asymmetry question: In our present experiment, overall IHTT did not show a convincing asymmetry. However, if one considers only the electrode sites in the two hemispheres yielding the fastest IHTT values, then an asymmetry favouring the RH to LH direction of transfer shows up clearly. Again, only specific studies aimed at manipulating the behavioural asymmetry of IHTT as a function of various task demands and trying to correlate it with corresponding electrophysiological asymmetries can say a conclusive word on the problem.

SUMMARY

We have assessed the interhemispheric transmission time (IHTT) by electrophysiological means in normal subjects performing a visuomotor reaction time task. We subtracted the latency of ERP components (Pl and N1) evoked by lateralized visual stimuli presented to the ipsilateral hemifield (indirect- callosal pathway) from the latency of the same components evoked by stimulation of the contralateral hemifield (direct pathway). Estimates of IHTT ranged between 5.77 msec and 12.54 depending upon the type of component and the location of the electrode sites. More anterior locations yielded shorter values of IHTT, and overall IHTT tended to be 7 msec shorter for the N1 component than for the P1 component. Moreover, there was an asymmetric IHTT for N1 in central sites with shorter latencies in the direction right-to-left hemisphere than in the opposite direction. Taken together, these results are in keeping with the idea that interhemispheric transfer of visuomotor information does not occur at the level of the primary visual areas but at more anterior cortical areas. The shorter IHTT for the N1 component suggests the involvement of the callosal connections between the inferotemporal areas of the two hemispheres.

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