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CONTRIBUTIONS OF TEMPORAL-PARIETAL JUNCTION LESIONS TO THE HUMAN AUDITORY P300

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P300 event-related brain potentials (ERPs) were recorded using auditory stimuli from 8 patients with confirmed left (4) or right (4) unilateral damage to the temporo-temporal junction. 6 normal control subjects were matched to each patient with respect to age and sex. P300 latency, but not amplitude, shows significant differences between control and pathologic groups. This kind of results does not permit to establish critical relations between temporo-parietal junction and P300 generators.

Contribuciones de las lesiones de la unión temporo-parietal al componente P300 auditivo. Regitramos el componente P300 generado mediante estímulos auditivos en 8 pacientes con lesiones de la unión temporo-parietal izquierda (4) y derecha (4). A estos sujetos añadimos otros 6 sujetos control igualados con los pacientes en edad y sexo. Los resultados en la latencia del componente P300 muestran diferencias significativas entre el grupo control y los grupos patológicos. Sin embargo, este tipo de resultados no permite establecer una relación causal entre la unión temporo-parietal y el generador o generadores del componente P300.

Research in the field of the anatomic source or origin of P300 component is where most of the work is taking place in the field of the endogenous event-related potentials (ERPs).

Usually, two different methods have been used in these studies. One of the approaches used is with intracranial recordings (McCarthy et al., 1982; Wood et al., 1984) for the diagnosis of brain disorders, especially with epileptic cases. These studies have found that P300-like wave forms which exhibit polarity reversal within the medial temporal lobe (MTL) or near the hippocampal formation are present in tasks that also elicit a P300 recorded from the scalp. This phase reversal initially would have suggested that the MTL might be the major neural generator of the P300 scalp.

However, other authors, using similar methods, have proposed the following different P300 neural generators: diencephalic (Velasco et al., 1986; Kropotov and Ponomarev, 1991) and neocortical (Desmedt and Debecker, 1979; Wood and McCarthy, 1985) regions.

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On the other hand, another approach has involved an assessment of patients with pathological changes in the left or right MTL originating from epilepsy. Although the results have often been variable, many of these studies have found differences and asymmetries in the scalp P300 between the affected and unaffected hemispheres (McCarthy et al., 1987; Puce et al., 1989; Nelson et al., 1991). These findings would suggest that the MTL makes a significant contribution to the scalp P300. In contrast, studies of patients who have sustained temporal lobe resections have detected little or no hemispheric asymmetry and differences between patients and control subjects (Scheffers et al., 1991; Rugg et al, 1991).

Facing these apparent contradictions, some authors propose to shift the focal point of interest to other cortical regions such as the temporo-parietal junction (Rugg et al., 1991), taking into account the important role of this region in functions that are associated with the P300 including attention, stimulus evaluation and memory (Neville et al., 1986). After various publications (Knight, 1990; Yamaguchi and Knight, 1991) unilateral damage of this area (with no change of the MTL) eliminates the scalp P300 from auditory and somatosensory stimuli. Likewise, to assess the contribution of temporal-parietal junction to auditory P300 generation, we recorded P300s in subjects with discrete unilateral lesions in the posterior association cortex.

Materials and method

Subjects

Three groups of subjects have been tested. The control group consisted of 6 right-handed subjects (mean age, 36,5 _ 5), matched in age and sex to the patients. They were recruited among the relatives accompanying the patients and were healthy and without history of neurological or psychiatric diseases. Patients were selected on the basis of a unilateral focal lesion in the posterior association cortex, determined through CT scan. Lesions primarly involved lateral superior temporal gyrus, posterior superior temporal plane, and inferior portions of the supramarginal gyrus. Four patients had damaged the right hemisphere (mean age, 37.5 _ 6), and four others the left hemisphere (mean age, 36.25 _ 4), provoked by a tumour. They were assessed after surgery.

Recording conditions and Procedure

P300 was elicited with auditory stimuli that consited of a pseudorandom sequence of 400 binaural tones (65 dB, 50 ms duration, 1.5/s ratio) with a frequency of either 1000 Hz (non-target, p = 0.85) or 2000 Hz (target, p = 0.15). The subject indicated the occurance of a target stimulus by moving the index finger of the preferred hand.

P300 was recorded with bandwiths of 0.3-70 Hz from three midline scalp sites, Fz, Cz, and Pz, and from lateral electrodes, T5, T6, C3, C4, P3, and P4, according to the 10/20 international system, referring to both ear lobes and rejection of artefacts. These responses were recorded and averaged with the ATI-NAUTILUS system.

Data analysis

Latency and amplitude of grand averages of ERPs were analyzed by measuring and averaging positive and negative peaks. Measures of latency and amplitude were carried out manually by means of the cursor in the screen. (Figure 1).

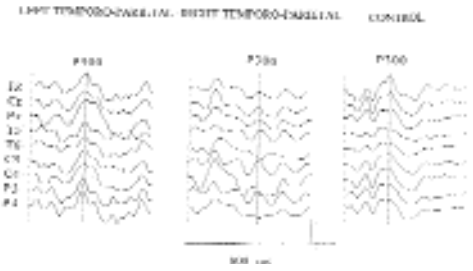

Figure 1. Grand average of cerebral evoked-potentials for the rare tone of one patient. The cursor is represented by vertical line.

Statistical Analysis

Recordings were analysed in P300 amplitude and latency measurements. Two ANOVAs 3 x 3 (Kind of Lesion x Zone) with repeated measures were applied for the midline electrodes and two ANOVAs 3 x 3 x 2 (Kind of Lesion x Zone x Hemisphere) with repeated measures for the lateral electrodes. These analysis were carried out with the BMDP 2V subprogram (Dixon, 1983). Due to differences in the sizes of the groups, probability values were obtained after the application of Geisser-Greenhouse corrections available in the BMDP2V. In case of significant statistical results the Tuckey test was applied later in order to compare relevant pairs of means.

Results

P300 latency and amplitude at the Fz, Cz, and Pz midline electrode sites

No significant differences were obtained in P300 amplitude and latency measures from the midline electrodes. (Table 1).

P300 latency and amplitude at the T5, T6, C3, C4, P3, P4 lateral electrode sites

The factor Kind of Lesion ($F_{2,11} = 4.02$; $p < 0.05$) shows a reliable effect in the P300 latency from the lateral electrodes. (Figure 2). This result indicates that P300 latency is significantly shorter for the control group compared to the patient group. The P300 latency in the right temporo-parietal group is longer than in the left temporo-parietal group. (Table 2).

At first, a reliable effect in the amplitude for the variable Zone ($F_{2,22} = 11.24$;

Table 1
Means and standart deviations of P300 latency over mid-line electrodes (Fz, Cz, Pz) in the left temporo-parietal, right temporo-parietal and control groups

	Left		Right		Control	
	Mean	Sd	Mean	Sd	Mean	Sd
Fz	348.2	68.52	339.2	75.57	290.16	17.71
Cz	347.25	68.68	355.75	68.17	287.00	22.71
Pz	360.50	74.23	366.0	61.00	286.66	33.99

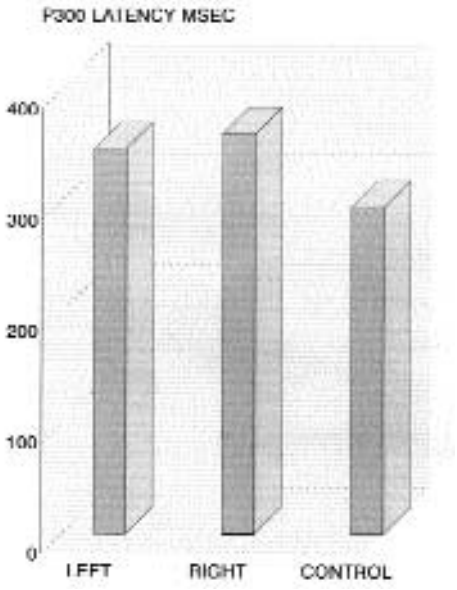

Figure2. Effect of Type of Lesion factor over P300 latency, showing reduced measures in control group.

$p < 0.01$) was found. (Figure 3). This shows that there was a singnificant reduction of the amplitude in the temporal areas, independently of any other variables, even the kind of lesion.

Table 2
Means and standart deviations of P300 amplitude over mid-line electrodes (Fz, Cz, Pz) in the left temporo-parietal, right temporo-parietal and control groups

	Left		Right		Control	
	Mean	Sd	Mean	Sd	Mean	Sd
Fz	5.25	3.55	6.49	4.24	5.22	2.23
Cz	5.19	2.93	6.81	5.11	4.98	2.39
Pz	4.85	3.56	6.74	5.12	4.38	2.83

Finally, the interaction of all the variables of this study has been significant: Kind of Lesion x Zone X Hemisphere ($F_{4,22} = 3.96$; $p < 0.05$). As a result, the amplitude in the left hemisphere is superior to the right hemisphere ($q = 5.17$; $p < 0.01$) in the central zone (C3, C4). (Table 2).

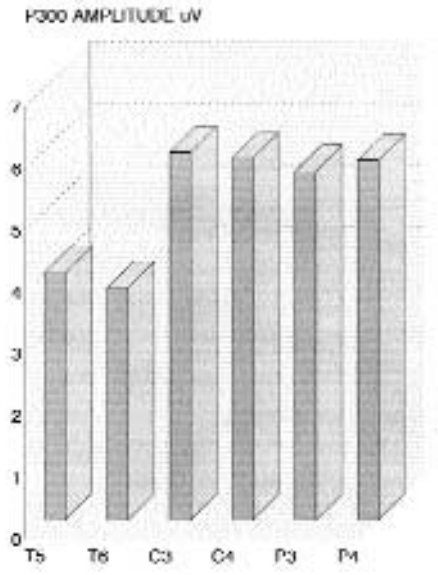

Figure 3. Effect of Zone factor over P300 amplitude, showing reduced punctuations over temporal electrodes.

Discussion

Table 3
Means and standart deviations of P300 latency over lateral electrodes (T5, T6, C3, C4, P3, P4) in the left temporo-parietal, right temporo-parietal and control groups

	Left		Right		Control	
	Mean	Sd	Mean	Sd	Mean	Sd
T5	348.0	70.97	369.5	58.48	304.3	22.33
T6	343.2	17.61	336.0	59.93	320.0	17.93
C3	348.7	70.40	372.2	56.99	289.6	16.09
C4	339.5	26.26	347.0	66.94	318.5	22.69
P3	362.2	79.94	390.2	50.38	298.6	22.23
P4	338.0	33.00	343.2	60.25	292.8	20.70

Table 4
Means and standart deviations of P300 amplitude over lateral electrodes (T5, T6, C3, C4, P3, P4) in the left temporo-parietal, right temporo-parietal and control groups

	Left		Right		Control	
	Mean	Sd	Mean	Sd	Mean	Sd
T5	3.77	1.47	5.67	3.18	3.07	2.01
T6	2.71	2.45	6.42	5.30	2.67	1.94
C3	6.71	2.41	7.05	3.00	4.72	3.57
C4	4.38	4.09	7.85	4.02	5.85	2.67
P3	4.99	3.49	6.89	2.84	5.25	2.80
P4	6.11	1.95	7.64	3.77	4.42	2.44

The findings of the present study do not suggest any relation between the temporal-parietal junction lesions and a significant reduction or abolition of the scalp P300. This result is not consistent with the findings of other authors (Yamaguchi and Knight, 1991), in studies with patients affected with temporal-parietal lesions.

Nevertheless, the results referred to the latency show an evident alteration of the wave associated with the lesion; both groups of patients present a significant raise of their latencies in the lateral electrodes. The P300 latency increase associated to the brain lesions is well known (Polich and Squire, 1993). As a matter of fact, the main interest to establish its origin appears after the correlation of this wave with important aspects of the cognitive functions such as short-term memory or sequencial processing of the information (Donchin, 1981). On the other hand, P300 is altered in certain brain pathologies that directly alter theses cognitive aspects such as dementia (Goodin, 1978; Brown et al., 1982).

Discrete lesions of the temporal-parietal junction can disrupt behavioural processes associated with P300 generation. For example, patients with temporal-parietal lesions show defects in orientation, attention, perception and memory mechanisms (Posner et al., 1984).

As such, in the studies of the temporal-parietal junction, a reduction is often found in the P300 amplitude in the temporal electrodes.

In the present study, a reduction of the amplitude independent of the lesion in all the groups has been identified. The interaction in the left temporo-parietal group between the affected hemisphere and the recorded amplitude is more complex, because the result obtained was inexpected. This means that in the central electrodes (C3 and C4), the amplitude is superior in the damaged hemisphere. A possible interpretation (according to Polich and Squire, 1993) woud be that ERPs are affected by the openings in the skull made at the time of surgery. A unilateral skull defect would produce less electrical resistance on the side of the lesion.

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