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Contributions of temporal–parietal junction to the human auditory P3

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The P3 component of the event-related potential (ERP) is generated in humans and other mammalian species when attention is drawn to infrequent stimuli. We assessed the role of subregions of human posterior association cortex in auditory P3 generation in groups of patients with focal cortical lesions. Auditory P3s were recorded to target (P3b) and unexpected novel stimuli (P3a) in monaural and dichotic signal detection experiments. Two groups of patients were studied with lesions of: (1) temporal–parietal junction including posterior superior temporal plane and adjacent caudal inferior parietal cortex; and (2) the lateral parietal lobe including the rostral inferior parietal lobe and portions of superior parietal lobe. Extensive lateral parietal cortex lesions had no effect on the P3. In contrast, discrete unilateral lesions centered in the posterior superior temporal plane eliminated both the auditory P3b and P3a at electrodes over the posterior scalp. The results indicate that auditory association cortex in the human temporal–parietal junction is critical for auditory P3 generation.

INTRODUCTION

Electrical fields recorded from the scalp index the timing and sequence of neuronal activity underlying cognitive processes. The P3 is the most widely studied event-related potential (ERP) component. P3s are generated in a number of mammalian species when attention is drawn to significant environmental events[4,12,22,24,25,35]. The P3 has been associated with psychological constructs including orientation, attention, stimulus evaluation and memory[11,21]. Neural sources of the P3 have been proposed in cortical, limbic and diencephalic regions based on scalp topographic analysis[6,33], intracranial recording[8,13,39] and neuromagnetic studies[23]. No consensus has emerged about either the behavioral process or the brain mechanism underlying P3 generation.

Discrete lesions of the temporal–parietal junction can disrupt behavioral processes associated with P3 generation in normals[20]. For example, patients with temporal–parietal lesions show defects in orientation, attention, perception and memory mechanisms[20,26]. Single unit and ablation studies in primates also document an important role of neurons in the temporal–parietal cortex in these functions[2,18]. Intracranial recordings in epileptic patients have found evidence for local phase inverting P3s and N2 potentials in temporal–parietal cortex[29]. Taken together, the data suggest that cortex in the temporal–parietal junction may be an element of the neural circuit engaged during P3 generation. To assess the contribution of temporal–parietal junction to auditory P3 generation, we recorded P3s in subjects with discrete unilateral lesions in subregions of human posterior association cortex.

MATERIALS AND METHODS

Clinical population

Patient groups were matched for pure tone thresholds, lesion etiology and mean lesion volume. Damage in the temporal–parietal junction group was

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centered in posterior Brodmann area 22, including lateral superior temporal gyrus and posterior superior temporal plane and inferior portions of areas 39 and 40 (temporal, Fig. 1). Lateral parietal lesions were centered in superior areas 39 and 40 and inferior portions of areas 5 and 7 (parietal, Fig. 2). None of the lesions from either group involved hippocampus or other mesial limbic structures. A control group, matched in age and sex to the patients, was also studied.

Patients were initially selected based on unilateral posterior association cortex lesions evident on CT scans. The patients were selected on the basis of having no or minimal overlap on axial CT cut 3 (Figs. 1, 2) from a larger group of subjects with posterior association cortex lesions. Patients with medical complications, psychiatric disturbances, substance abuse, multiple neurological events, hearing loss, or dementia were excluded. Three patients were excluded because of interaural threshold differences in excess of 15 dB at 1 and 1.5 kHz, or an absolute hearing loss in excess of 40 dB at these frequencies. ERPs were then recorded from the remaining 14 subjects. The results from 2 subjects could not be reliably evaluated because of excessive eye blink or EMG artifacts and they were excluded from further analysis. The remaining 12 subjects (6 temporal, 6 parietal) performed well behaviorally and had reproducible ERPs in both experiments. The 6 temporal lesions (5 left, 1 right; mean lesion volume 48.3 cm^3) were matched in size to the 6 parietal lesions (3 left, 3 right; mean lesion volume, 40.3 cm^3). All lesions were at least 6 months old and were due to cerebrovascular events (n = 10) or tumor resection (n = 2, one in each group). Control subjects were matched in age and sex with the patients (control 53.3 ± 10, parietal 52.9 ± 11, temporal 56.1 ± 9 years).

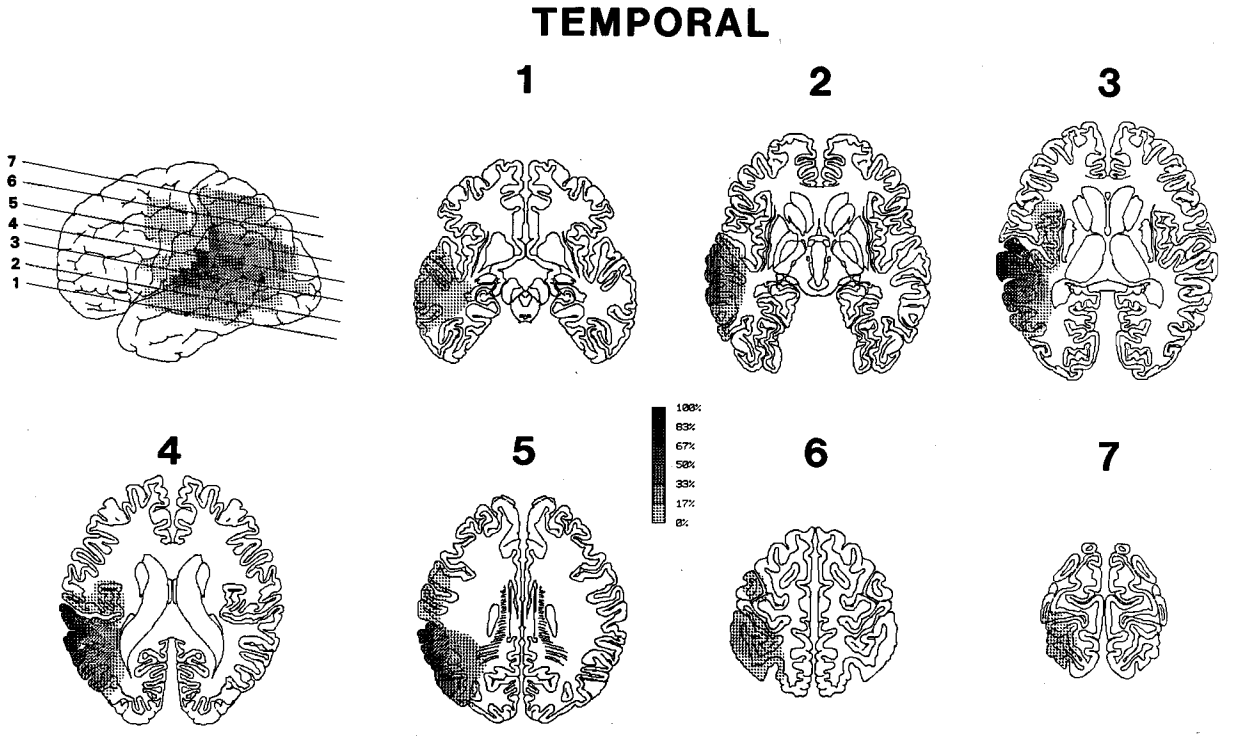

Fig. 1. Lesion extent in patients with focal unilateral damage centered in the temporal–parietal junction (temporal). The lines on the lateral reconstruction indicate the location of the axial sections used in CT transcription. Lesions determined by CAT scan from individual patients were transcribed onto 0 degree to canthomeatal line templates. A lateral view of the lesion extent was then projected from the axial sections by software reconstruction methods. The digitized lesion data from individual subjects was then averaged to generate the group lesion densities for both lateral and axial views. Unilateral right hemisphere lesions have been reflected onto the left hemisphere. Lesions were averaged over 6 patients (5L, 1R, mean lesion volume = 48.3 cm^3). The scale indicates the percentage of patients with damage in the corresponding area.

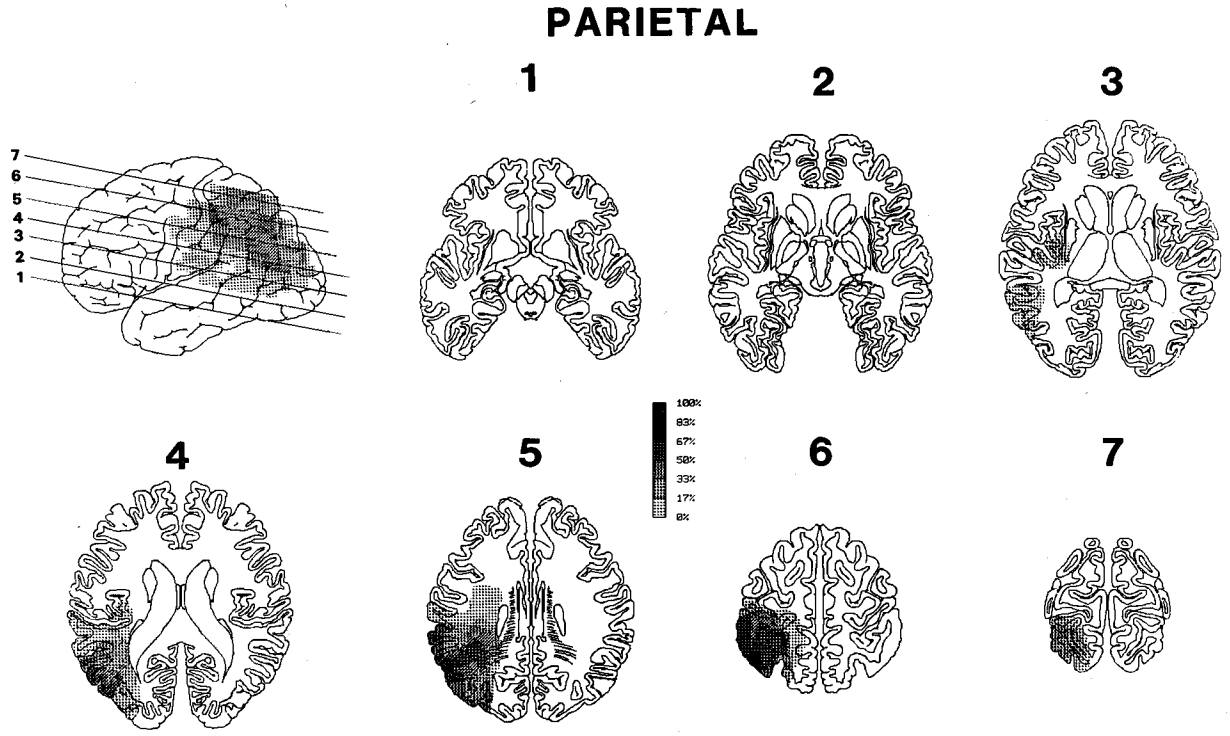

Fig. 2. Lesion extent in patients with focal unilateral damage centered in the lateral parietal cortex (parietal). Lesions were averaged over 6 patients (3L, 3R, mean lesion volume = 40.3 cm^3). See Fig. 1 for further details.

Lesions evident on CT scan were transcribed onto corresponding CT templates by 3 independent raters. Software permitted reconstruction of the lateral perspective, determination of lesion volume and cytoarchitectonic areas affected, and extraction of group-averaged lesions. All temporal patients had damage in area 22. In addition, all temporal subjects had unilateral reduction of the T-components of the auditory evoked potential generated in the lateral superior temporal gyrus[16,27]. Auditory cortex (areas 41 and 42) and inferior portions of angular (area 39) and supramarginal (area 40) gyri were involved in some temporal patients. All parietal patients had damage in rostral areas 39 and 40 and portions of area 7. Inferior portions of areas 39 and 40, somatosensory cortex and area 19 were lesioned in some parietal subjects. Details of individual subject's neurological deficits and location of damage are presented in a separate manuscript[16]. The patients reported here include 4 of the left superior temporal gyrus lesions (numbers 1, 3, 6, 8) and one right superior temporal gyrus lesion (number 4) described in the prior report. A sixth left temporal subject with a clinical history and lesion comparable to case number 1 was also studied. The 6 parietal subjects are identical to those previously described. One parietal patient had slight lesion extent into the posterior temporal plane. However, T-components were preserved in this patient.

Experimental design

Two experiments were conducted. In the first, subjects listened to monaural tone bursts presented at fixed 1.0 s interstimulus intervals. Cross-hearing was masked with 35 dB of white noise in the non-stimulated ear. Frequent standard tones (1.0 kHz, 50 ms duration, 60 dB sound level (SL), 5 ms rise–fall time) occurred on 80% of the trials. Infrequent target tones (1.5 kHz, 50 ms duration, 60 dB SL, 5 ms rise–fall time) occurred randomly on 10% of the trials. The subjects were instructed to press a button to the target tones. Novel sounds (unexpected complex tones and environmental noises) occurred randomly on 10% of the trials.

The second experiment was a more difficult selective attention task. Stimuli were presented

rapidly at intervals ranging from 200 to 400 ms. Tone bursts of 700 Hz in one ear and 1300 Hz in the other were presented dichotically with probabilities of 42% in each ear (standards). The subjects attented to either right or left ear tones and pressed a button to random target tone bursts in that ear (probability 5% in each ear). The targets were identical in frequency to the standards but longer in duration (75 vs 25 ms, 50 dB SL). The ear attended was counterbalanced across recording blocks. Novel stimuli occurred randomly on 3% of the trials in each ear.

The novel sounds consisted of 10 computer-synthesized complex sounds and 10 digitized environmental noises (200 ms duration). To reduce the perceived loudness of the novel sounds, peak sound pressure level (SPL) intensities were attenuated by 6 dB in comparison with the standards and targets. ERPs were recorded in 4 blocks, 8–10 min in duration, with the order of ear stimulated or attended counterbalanced across patients in both experiments. Target and novel stimuli were employed in both experiments to assess lesion effects on the P3 generated to correctly detected targets and on the P3 generated to unexpected novel stimuli[5,30]. For brevity, the P3 generated to correct target detections has been operationally defined as the P3b and the P3 generated to the novel stimuli as the P3a.

Recording techniques

Event-related potentials recorded from electrodes in the international 10-20 system (F3, Fz, F4, C3, Cz, C4, P3, Pz, P4, and below eye) were referenced to a balanced two-vector non-cephalic reference electrode[37]. Continuous EEG was amplified (50K), filtered (0.1–100 Hz), and digitized (256 Hz/channel) on a general purpose computer (PDP 11/73). Averaging was performed off-line after artifact rejection and sorting by stimulus and response type. Mean voltages and peak amplitudes and latency measures referred to a 200 ms prestimulus baseline were obtained by computer for novel and correctly detected target stimuli. Broad measurement windows were used for calculation of peak amplitude and latency (P3a, 280–500 ms; P3b, 300–600 ms). Mean voltages were calculated over restricted 50 ms windows centered on the peak latency for the P3a or P3b in each group in each experiment. Measurement windows were confirmed from inspection of the individual subjects data and from the group super-averages.

Measurements were subjected to repeated measures analysis of variance (subject × group × ear × electrode) with specific comparisons performed when appropriate[14]. Since electrodes do not provide independent amplitude measures of components, amplitudes at single scalp sites were used to evaluate reduction of components (i.e. Pz for the P3).

RESULTS

Behavioral

Both patient groups performed comparably. In Expt. 1, detection accuracies were comparable (percent correct detection: controls = 97.8%, parietal = 95.7%, temporal = 94.5%; P = n.s.). Reaction times (RTs) were prolonged in both patient groups (controls = 445 ± 84 ms, parietal = 487 ± 86 ms, temporal = 535 ± 90 ms); but differences in RTs between parietal and temporal groups were not significant.

In Expt. 2, RTs were increased and response accuracy was reduced in both control and patient groups in comparison to Expt. 1. RTs of parietal and temporal groups were prolonged relative to controls, but again differences between the patient groups were not significant (controls = 774 ± 74 ms, parietal = 867 ± 147 ms, temporal = 853 ± 96 ms). Target detection in Expt. 2 as measured by d' values was not significantly different between control and patient groups (controls = 2.60, parietal = 2.00, temporal = 2.19).

Electrophysiological

Marked intergroup differences were observed in the P3 to correctly detected targets. In Expt. 1, controls generated a parietal P3b to targets (latency = 388 ms) and a central P3a to novel stimuli (latency = 367 ms, solid line in Fig. 3a,b). Similar morphology P3a and P3b potentials were generated by novel and target stimuli in Expt. 2, although the P3b was delayed in latency and more parietal in distribution[9] in comparison with Expt. 1 (Expt. 2: P3b = 448 ms, P3a = 356 ms, solid line in Fig. 4a,b). Parietal lesions had no significant effect on P3 amplitude or latency. In particular, P3 responses to target and

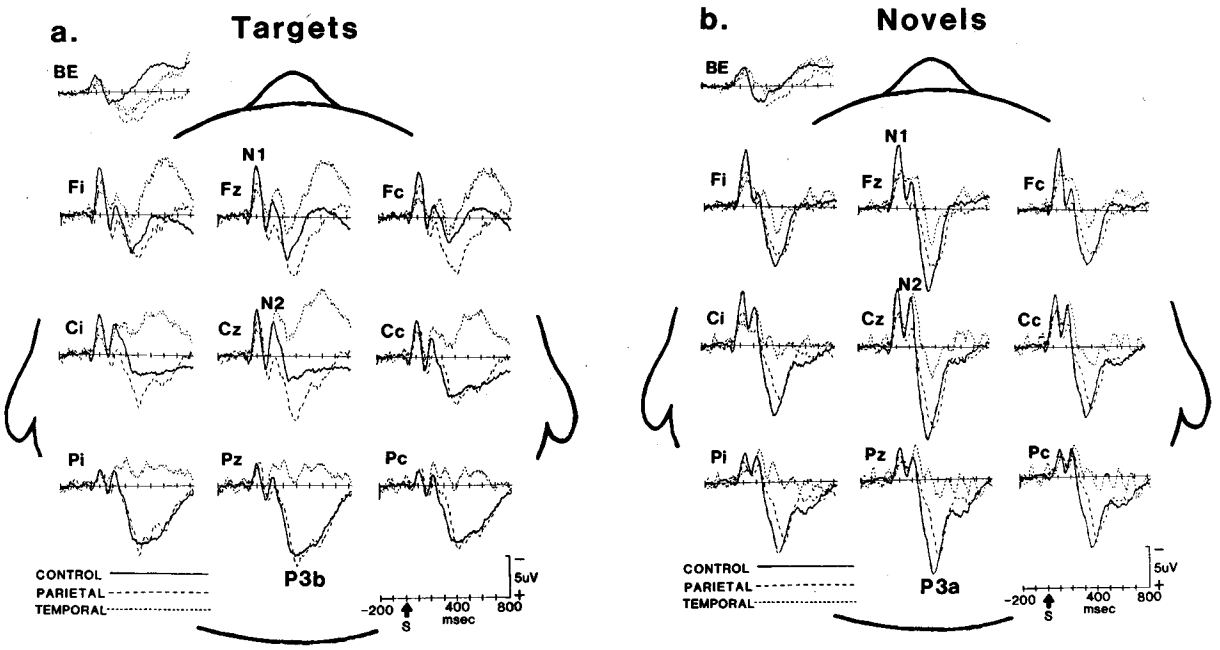

Fig. 3. Group-averaged ERPs recorded to target and novel stimuli in the monaural tone detection task (Expt. 1, 3a and b, respectively). The arrows (S) denote stimulus onset. Solid lines show ERPs from controls, dotted lines from temporal patients, and dashed lines from patients with parietal lesions. Data is shown from the midline and parasagittal scalp sites. Scalp sites are shown ipsilateral (i) and contralateral (c) to lesioned hemisphere for patients, or on the left and right for controls. Lesions in the temporal–parietal junction abolished the P3a and P3b at all posterior scalp sites. ERPs are grand averages over 6 patients in each group.

novel sounds had symmetrical amplitudes over lesioned and non-lesioned hemispheres in Expts. 1 and 2 (dashed lines in Figs. 3a,b, 4a,b). The increase in target P3 amplitude evident in these figures at central sites in the parietal group did not reach significance. P3 latencies in the parietal group were also within the normal range (Expt. 1: P3b = 402 ms, P3a = 381 ms; Expt. 2: P3b = 481 ms, P3a = 401 ms; P = n.s.).

In contrast, temporal lesions abolished the target P3b and the novel P3a over the central and parietal scalp in Expts. 1 and 2 (dotted lines in Figs. 3a,b, 4a,b). In Expt. 1, mean P3b amplitude at Pz averaged 7.95 μV in controls and 8.60 μV in parietal patients, whereas mean voltage measurements were negative for the temporal patients (mean voltage = –0.85 μV; $P < 0.001$ for both peak and mean voltage measurements). P3a voltages at Pz followed a similar pattern: parietal lesions had no effect but temporal lesions reduced P3a amplitudes to noise levels (control = 10.21 μV, parietal = 8.36 μV, temporal = 1.70 μV; $P < 0.001$ for both peak and mean voltage measures). This reduction did not appear to be due to temporal dispersion of the P3, since no P3 was observed to subaverages of correctly detected stimuli with the fastest RTs and smallest RT variance in the temporal group (block 4, Expt. 1 = 489 ± 63 ms).

Similar results were obtained in Expt. 2. P3b and P3a amplitudes at Pz were comparable for control subjects and parietal patients, but both components were abolished by temporal lesions (mean amplitude P3b at Pz: controls = 4.39 μV, parietal = 4.62 μV, temporal = 0.33 μV; $P < 0.02$ for mean and $P < 0.005$ for peak voltage measures; mean amplitude P3a at Pz: controls = 4.81 μV, parietal = 4.26 μV, temporal = 0.09 μV, $P < 0.001$ for both peak and mean voltage measures). In both experiments, the P3a and P3b were abolished over both lesioned and non-lesioned hemispheres. Inspection of individual subject waveforms revealed that the posterior P3a and P3b responses were abolished in every temporal patient in both Expts. 1 and 2, although a shorter latency frontal positivity was partially preserved in both experiments (see Figs. 3a,b 4a,b).

As can be seen in Figs. 3a and 4a, there was an apparent amplitude increase in the late frontal–

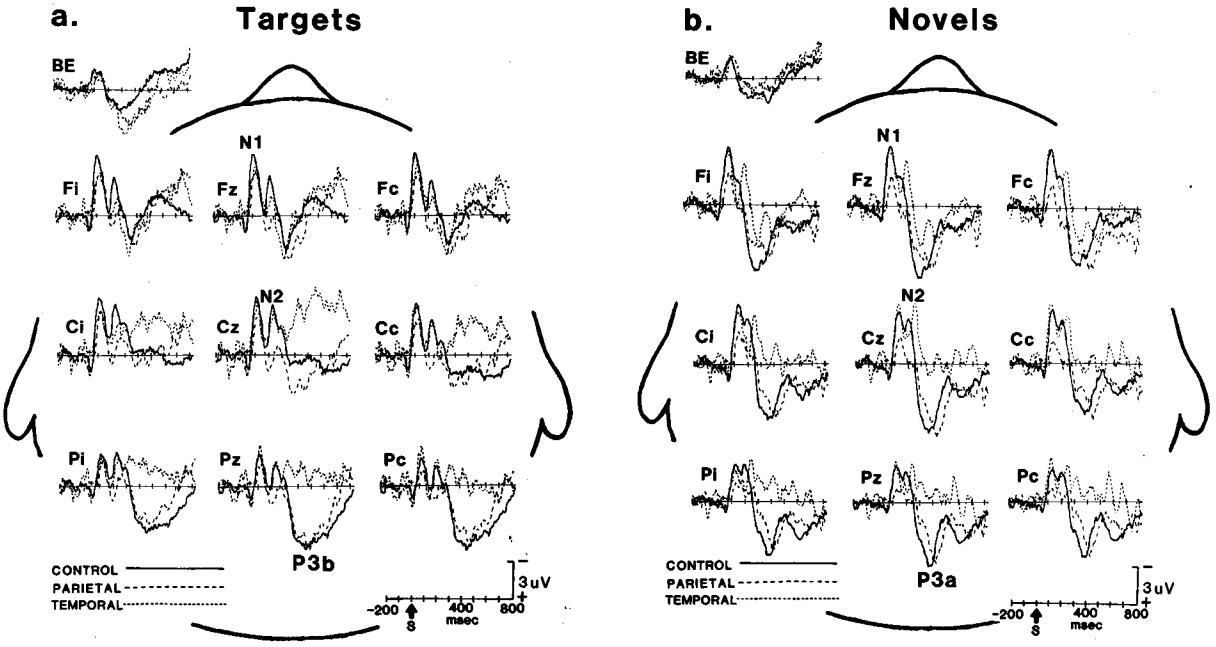

Fig. 4. Group-averaged ERPs recorded to target and novel stimuli in the dichotic attention task (Expt. 2, 4a and b, respectively). Same conventions as in Fig. 3.

central slow wave (at frontocentral sites, $P < 0.025$) to target stimuli in the temporal group. This may reflect the loss of overlapping P3 activity at frontocentral sites. It is unlikely that unmasking or enhancement of this negative slow wave accounts for the P3 decrements observed for two reasons. First, the slow wave was frontocentral in distribution and the P3 was abolished at parietal sites. Second, the P3 was also abolished at posterior sites in response to novel stimuli which did not generate significant frontocentral slow activity.

In the parietal group, the N200 showed evidence of amplitude reduction in both experiments (in Expt. 1, $P < 0.05$ for targets, $P < 0.10$ for novels; in Expt. 2, $P < 0.025$ for target, $P < 0.05$ for novels). The N200 was unaffected by temporal lesions in both experiments. The trend towards N200 reduction in the parietal group was an unexpected post hoc observation and the marginal levels of significance suggest caution in interpretation of the results pending confirmation in a larger patient cohort.

DISCUSSION

Focal lesions in the temporal–parietal junction abolished the auditory P3 at posterior scalp sites in patients who could discriminate the stimuli. Further behavioral studies of these same temporal–parietal patients have shown reduced orienting to distracting stimuli[17]. Other investigators have reported that patients with anterograde memory deficits due to posterior association cortex or limbic pathology have reduced P3s[19,31]. These findings suggest that the auditory P3 is generated by a neural system involved in orientation to and encoding of environmental events.

The electrophysiological results partially clarify the neural structures involved in P3 generation. Extensive lateral parietal lobe lesions produced no decrement in the P3, even from electrodes placed directly over lesioned cortex. Apparently, substantial areas of lateral parietal cortex are not critical for generation of the auditory P3.

In contrast, the P3a and P3b were abolished over both parietal lobes by unilateral lesions of the temporal–parietal junction. These same lesions resulted in partial preservation of P3 activity at frontal scalp sites supporting the notion that multiple generators contribute to the P3a and P3b[15,36].

Five of the six temporal–parietal patients had left-sided lesions. Although the single right temporal–parietal lesion reported in the current study had

an abolished P3, it is possible that the temporal–parietal group P3 reductions are due primarily to dysfunction of a system lateralized to the dominant hemisphere. Examination of a larger series of patients with right temporal–parietal lesions comparable in size to the left lesions reported here is needed to resolve the issue of possible asymmetric hemispheric modulation of the auditory P3 generator.

The bilateral reduction of the P3 by a unilateral lesion is difficult to interpret. The P3 in normal subjects has a symmetrical scalp distribution consistent with cortical or subcortical generators synchronously active in both hemispheres. Bilateral reduction indicates that generators in both hemispheres were compromised by the unilateral lesions. One possibility is that P3 generation is dependent on an interhemispheric comparison of sensory data in the superior temporal planes. Unilateral temporal lesions would destroy one vertically oriented dipole generator in the posterior superior temporal plane[27] and deafferentate and compromise the other, resulting in a bilateral abolition of the P3 at posterior scalp sites. However, a report that a patient with a callosal lesion disconnecting interhemispheric auditory pathways had a normal auditory target P3 mitigates against this interpretation[1,7].

Alternately, generation of an auditory P3 might require efferent hemispheric projections to midline limbic or thalamic generators directly from posterior superior temporal plane or from other association areas whose projections may have been interrupted by these lesions[3,28,34,38]. The lesions which abolish the posterior auditory P3 overly multimodal area cSTP[10] and auditory association area Tpt in the monkey[32], a region with bidirectional connections to area TH in the parahippocampal gyrus. A Tpt-mesial temporal network has been proposed to be critical for acoustic learning and memory in animals and humans[3] and area cSTP has been implicated in the control of global attention[10]. The present results suggest that the auditory P3 is generated by engagement of these regions during encoding of significant environmental events in humans.

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