Human Intracerebral Potentials Associated with Target, Novel, and Omitted Auditory Stimuli

Claude Alain,* Francois Richer,* André Achim,* and Jean-Marc Saint Hilaire†

Summary: We recorded late auditory potentials from lateral and medial regions in the frontal, temporal and parietal lobes of patients with temporal lobe epilepsy implanted with horizontal depth electrodes. Tone sequences were presented in three tasks: 1) auditory target detection in a tone sequence, 2) target detection with interspersed novel stimuli, and 3) detection of stimulus omissions. At frontal sites, potentials to targets showed a triphasic response with peak latencies around 200, 270 and 350 ms. At temporal sites, potentials consisted of a generally positive 285 ms peak which was sometimes accompanied by a negative peak at 200 ms or at 400 ms. At parietal sites, potentials were generally triphasic with latencies of about 230, 300, and 370 ms. At most sites, potentials evoked by novel stimuli had shorter latencies than those evoked by targets. The frontal and parietal potentials were either absent or strongly attenuated during stimulus omissions. The results lend further support to the multiple generator hypothesis of late potentials and suggest that some of the cerebral sources of the late potentials are stimulus dependent while others are not.

Key words: Depth EEG; event-related potentials; P300; auditory system

Introduction

Infrequent target tones or stimulus omissions interspersed within a sequence of standard stimuli elicit biphasic potentials on the scalp between 200 and 450 ms post-stimulus (N2-P3). Because of the cognitive parameters which affect these late scalp potentials (see Donchin, Ritter, and McCallum, 1978), there is a great deal of interest in finding the location of the cerebral events responsible for them.

Intracerebral evoked potential studies have described activity in the medial temporal lobe which partly overlaps the scalp P3 and which inverts polarity at the level of the hippocampal formation and amygdala (Halgren et al. 1980; McCarthy et al. in press; Stapelton and Halgren, 1987; Wood et al. 1984). These results suggest that one of the sources of N2-P3 may lie in the medial temporal lobe.

Recordings of late potentials in other parts of the brain have been scarce. Wood and McCarthy (1985) showed that targets sometimes evoke a triphasic potential complex in the frontal lobe with peak latencies of 200, 280 and 400 ms respectively. This complex begins earlier than and partly overlaps the potentials recorded in the temporal lobe. Correlates of the scalp N2-P3 were also recorded in thalamic and other subcortical structures (Velasco et al. 1986; Yingling and Hosobuchi, 1983).

Lesion data have also contributed to the investigation of the neural bases of the late potentials. Some studies indicated that unilateral excisions in the temporal lobe did not dramatically affect the topography and amplitude of auditory or visual P3 recorded on the scalp (Johnson, 1989; Stapleton, Halgren and Moreno, 1987), but that they did affect a visual negative potential on the scalp (Smith and Halgren, 1988). Lhermitte et al. (1985) reported a decreased and delayed visual P3 with unilateral parietal lesions when stimulation was in the neglected hemifield but no effect of the same lesions on auditory P3. Knight (1984) reported that unilateral frontal lesions attenuated the P3 evoked by novel stimuli at frontal scalp sites but did not appear to affect the P3 to targets. Lesion data should however be interpreted with caution because damage to one of two symmetric medial generators may produce little change at the scalp and destruction of structures which modulate the activity of primary generators may produce significant changes at the scalp without damaging the generators themselves.

Although very fragmentary, the available data suggest that multiple generators contribute to the scalp late potentials and that some of these generators could be task-specific or modality-specific. Previous intracerebral recording studies have mainly concentrated on a detailed

*Université du Québec à Montréal, Service de Neurologie, Hôpital Notre-Dame.

†Service de Neurologie, Hôpital Notre-Dame, Université de Montréal.

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Correspondence and reprint requests should be addressed to F. Richer, Dept. of Psychology, Université du Québec à Montréal, Box 8888, Montreal, QC, Canada, H3C-3P8.

investigation of potentials recorded in medial temporal regions. This study examined the more general distribution of late potentials within frontal, temporal, and parietal brain regions to try to document more fully the multiple generator hypothesis of P3.

Methods

Subjects

Recordings were obtained from 14 patients with partial complex epilepsy (9 men, 5 women; 16 to 40 years old, mean age 27). Patients had chronically implanted depth electrodes as part of a pre-surgical evaluation to localize the site of seizure onset. Ictal onset as determined by the depth-electrode recordings (SEEG) was in one of the temporal lobes in all patients, with 8 patients showing left temporal onset and 6 patients showing right temporal onset. CT and MRI scans were negative in all patients except for occasional abnormalities in the SEEG-determined epileptogenic temporal lobe. All patients had normal intelligence and showed no major psychiatric problems. Patients had no hearing impairment, were normally alert during testing, and comprehended instructions. Patients were medicated with anticonvulsants that were gradually being removed at the time of testing. Only patients with error rates averaging less than 15% in the tasks were included in the study. This selection criterion eliminated about 12% of the patients.

Recording

All patients were implanted with four to ten multicontact electrodes. The number and location of electrodes were selected on the basis of clinical criteria only, mainly from ictal and interictal EEG and ictal behavior. For example, patients for whom the EEG and ictal behavior were not localizing, would be implanted with either frontal or parietal electrodes in addition to temporal electrodes depending on the ictal symptoms observed and the anterior or posterior distribution of EEG epileptic activity. All patients had one or more electrodes in the temporal lobe, 12 patients had at least one electrode in the frontal lobe, and 4 patients had at least one electrode in the parietal lobe.

Depth electrodes were sterotaxically implanted via a horizontal lateral to medial trajectory. The implantation procedure was adapted from that of Talairach and Bancaud (Bancaud et al. 1965; Bouvier et al. 1987). Electrode placement is guided by the neuroradiological workup (arteriography and ventriculography) done under stereotactic conditions using stereoscopic teleradiography. Brain structures are located in relation to veins, arteries, and ventricles, and by projection of anterior and posterior commissure landmarks in the Talairach atlas (Talairach and Szikla, 1967). Electrodes contained 5 to 10 stainless steel contacts of 1.6 mm diameter and 1.4 mm length. Contacts were separated by 4 mm.

The exact location of electrodes varied from patient to patient. Figure 1 illustrates the regions in which electrodes were implanted in the patients presented here. In the temporal lobe, electrodes were aimed at the anterior (A), middle (B), and posterior (C) portions of the middle temporal gyrus, and reached the amygdala and medial temporal areas in the vicinity of the hippocampus. In the frontal lobe, electrodes were located in the posterior portion of the superior frontal gyrus at the level of the supplementary motor area (M), and at the anterior middle frontal gyrus in the region of Brodmann's area 46 (H), and inferior frontal cortex (O). Medial frontal sites implanted included the anterior cingulate gyrus, the supplementary motor area, and areas just dorsal of medial orbital cortex. In the parietal lobe, the structures implanted were the superior parietal cortex, the angular gyrus, the posterior cingulate gyrus, and the precuneus.

Recordings were all monopolar and referenced to linked ears. Electrode impedance varied between 3 and 8 Kohms. EEG signals were amplified and filtered (bandpass .5 to 100 Hz) using 32 preamplifiers (Grass 7P511) and digitized at 200 Hz over a 800 ms epoch beginning 200 ms before tone onset. Scalp recorded activity was obtained from a midline parietal electrode. Electro-oculographic artifacts were automatically detected from forehead recordings and rejected on-line.

Procedure

Recordings were obtained in three different tasks: 1) Auditory oddball detection (all patients), 2) Oddball detection with rare novel stimuli (8 patients), and 3) Detection of targets and stimulus omissions (7 patients).

In the oddball detection task, stimulation consisted of a sequence of tones containing 80% of 600 Hz tones (standards) and 20% of randomly interspersed 1500 Hz tones (targets).

In oddball detection with novel stimuli the sequence included 80% standards, 10% targets and 10% frequency ramps (single ramp from 2500 to 4500 Hz) used as novel stimuli. In these two tasks, subjects were instructed to count and report the number of target tones that were presented. Tones were delivered with a randomly varying interstimulus interval of 1.5 to 1.8 sec.

In the target and omission detection task, the sequence of stimuli consisted of 80% standard tones and 10% target tones. Also, a random 10% of the tones were omitted. In this task, events were presented at a fixed inter-stimulus interval of 600 ms. Subjects were instructed to count both the targets and the stimulus omissions in a single tally.

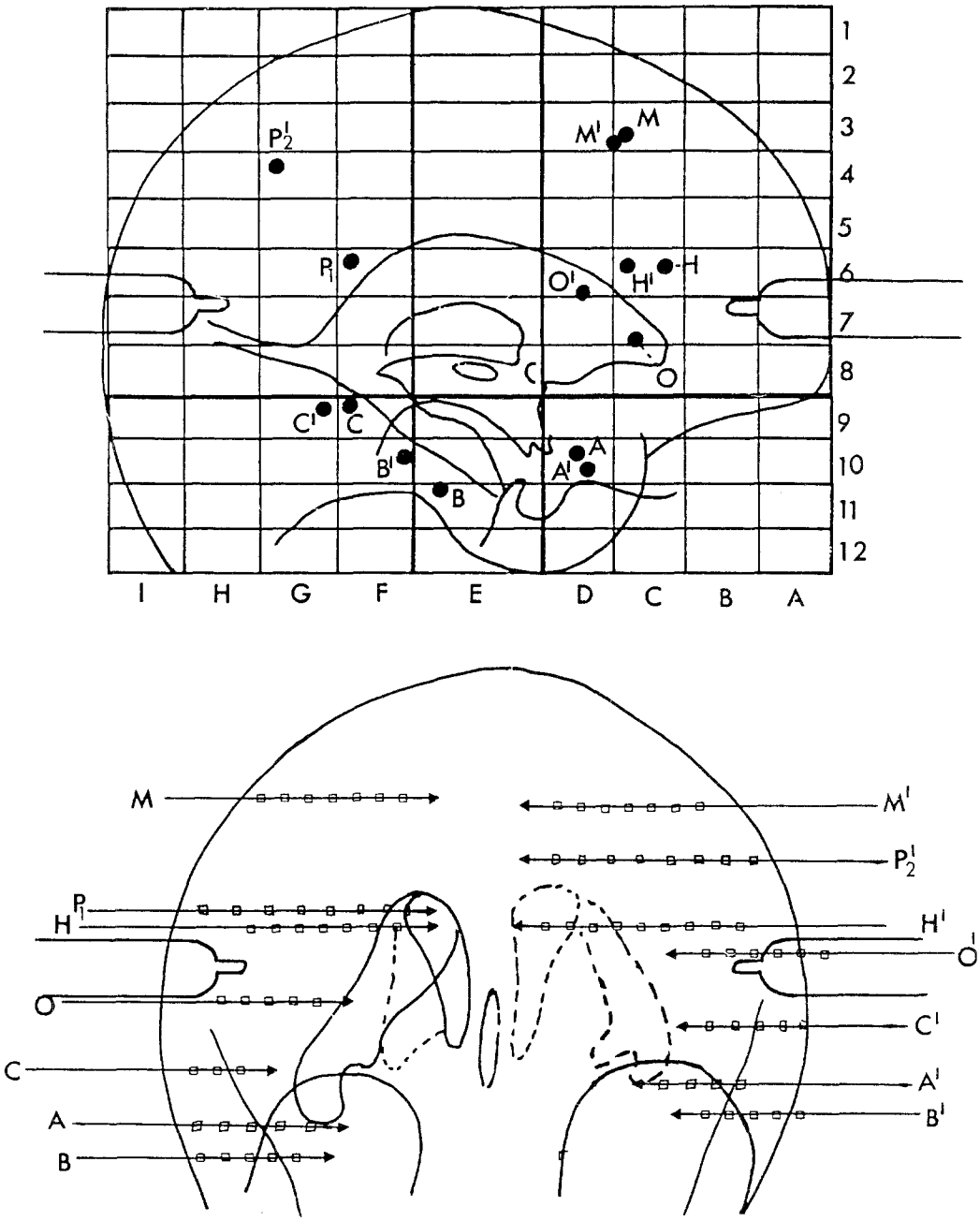

Figure 1. Approximate electrode locations in the patient sample superimposed on drawings from radiographs of an average sized head. The lateral view is projected on the standardized coordinate system of Talairach and Szikla (1967).

All tones were presented binaurally through headphones at an intensity of 70 dB HL and with a 50 ms duration. From three to six different stimulation sequences of 115-180 tones were delivered to each patient in every task. Trials containing EOG artifacts were automatically replaced by adding new stimuli in the sequence to obtain 100 "EOG-free" traces in each sequence. Single trial responses were visually inspected and those containing large amplitude oscillations (epileptic slow waves) or other epileptic activity, were rejected. Average evoked potentials were then computed separately for each type of tone and for stimulus omissions. Peak amplitudes were measured relative to the average amplitude in the pre-stimulus interval.

Results

Because of the variety of intracerebral sites explored, we will first provide a descriptive account of the spatio-temporal patterns of waveforms most consistently ob-

served in individual patients; this will be followed by a group summary of amplitudes and latencies of potentials at the most frequently implanted sites.

Figure 2 illustrates the scalp and intracerebral potentials evoked by standard and target tones in a typical patient. The variability in the potentials from adjacent contacts was very low, and therefore only potentials from the most lateral and the most medial contacts on each electrode are shown.

All tones produced a large and widely distributed biphasic response with latencies similar to that of the scalp-recorded N1 and P2 potentials. As previously reported (Richer et al. 1989), the polarity of these intracerebral potentials was inverted in the posterior (sometimes anterior) temporal lobe relative to that recorded in frontal and superior parietal sites.

Distribution of Late Potentials to Targets

In addition to the N1-P2, target tones evoked a late potential complex at Pz consisting of a negative peak around 220-285 ms (N2) evident in 10 patients and a positive peak at at 290-365 ms (P3) recorded in all patients. In 7 patients, a negative peak also appeared at 385-525 ms (N4).

In frontal sites, target tones generally produced a negative-positive-negative complex with peak latencies of approximately 200, 270, and 350 ms respectively. This triphasic response was present in 8 of the 12 patients with frontal electrodes, the remaining patients showing only the first two peaks. Differences between target-related potentials at various frontal sites were small and generally non-systematic. Potentials at superior frontal sites

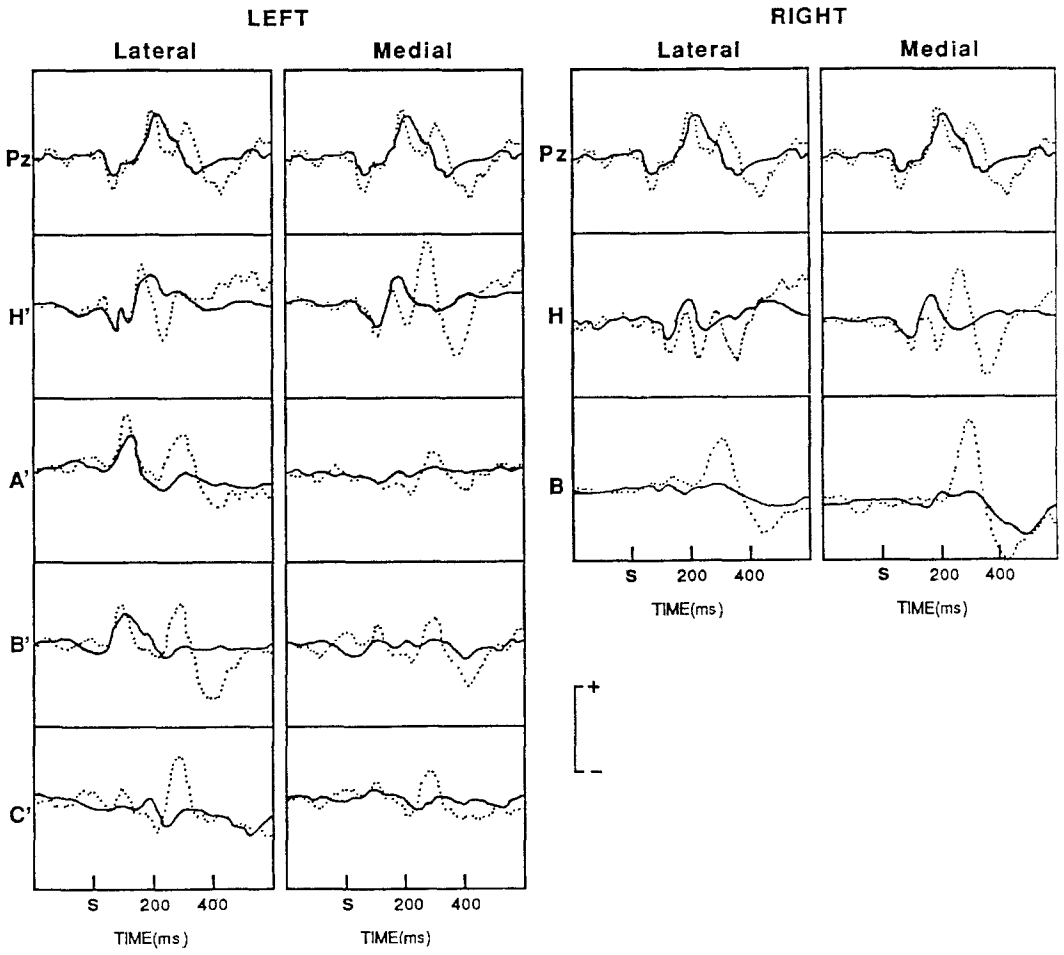

Figure 2. Intracerebral distribution of late evoked potentials to standard and target tones in both hemispheres of a patient with left temporal seizure onset. The waveforms are averages of about 160 individual potentials for standards (solid) and 40 potentials for targets (dotted). The calibration line is 20 μV for scalp traces and 60 μV for intracerebral traces. Electrode locations are as in Figure 1.

(electrode M) had slightly longer latencies than those at middle frontal sites (electrode H) in 3 of the 4 patients implanted with both electrodes. There were no differences between inferior frontal (electrode O) and middle frontal potentials in the single patient with both electrodes.

At temporal sites, the most reliable deflection, which was recorded in the non-epileptogenic lobe of all patients, was a positive peak at about 285 ms. In 4 patients (including the one shown in Figure 2) a negative peak also appeared at about 410 ms. In 3 different patients a negative peak also preceded the positive peak at about 200 ms, but this peak was not always distinguishable in latency from the inverted P2 also present in potentials to standard non-target tones in this region. There were only minor non-systematic differences between potentials recorded from anterior, middle and posterior temporal sites and also between medial and lateral sites in that lobe.

Targets also evoked late potentials in the parietal lobe. These potentials generally had a triphasic waveshape, with negative-positive-negative deflections peaking around 230, 300 and 370 ms.

Figure 3 illustrates the inter-patient variability in the waveshapes and latencies of late potentials to targets at the lateral and medial sites most frequently implanted, that is to say, the middle temporal region (electrode B) and the middle frontal region (electrode H). Intra-patient variability from one replication to another was approximately the same as that observed on the scalp.

We compared the temporal and frontal waveforms in the non-epileptogenic hemisphere in the 9 patients who had both electrodes. In 7 of these 9 patients the latency of the positive temporal peak (285 ms) was 20 to 30 ms longer than that of the frontal positive peak. In the other 2 patients frontal and temporal positive peaks had the same latency.

Comparisons between parietal and frontal waveforms (4 patients) showed that in 3 patients the parietal peaks were generally delayed compared to their frontal counterparts whereas the latencies were similar in the remaining patient. Also, in 3 of these 4 patients, the

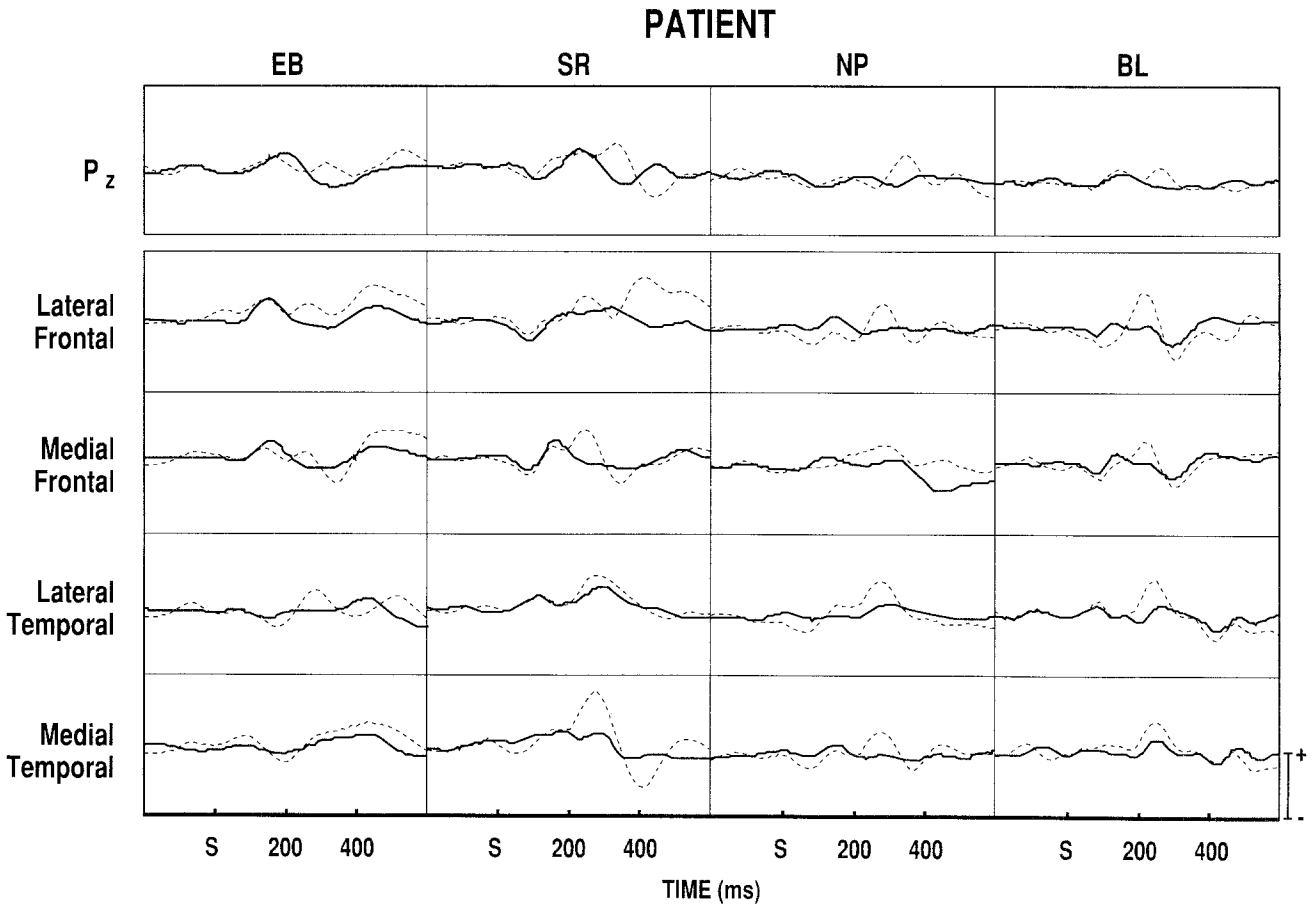

Figure 3. Distribution of late evoked potentials to standard and target tones in four patients at medial and lateral sites in frontal and temporal lobes in the hemisphere contralateral to the epileptic focus. Waveforms are averages of 160-240 individual potentials for standards (solid) and 40-60 potentials for targets (dashed). Calibration is 20 μV for scalp and 60 μV for intracerebral traces.

Table I. Latencies and amplitudes of the late potential peaks evoked by targets at the scalp midline (Pz), and at the most lateral and the most medial contacts of middle frontal (electrode H), middle temporal (electrode B), and parietal (electrode P) electrodes in non-epileptogenic lobes.[a]

	Peak 1		Peak 2		Peak 3	
	Lat.	Amp.	Lat.	Amp.	Lat.	Amp.
Pz	N258	2.3	P325	7.0	N457	7.2
	(18)	(3.6)	(20)	(2.3)	(48)	(2)
Frontal:						
Lateral	N203	16.3	P267	22.1	N346	23.0
	(20)	(11)	(25)	(15)	(28)	(14)
Medial	N201	19.8	P270	28.0	N358	25.4
	(22)	(16)	(24)	(24)	(30)	(15)
Temporal:						
Lateral	N198*	7.6*	P281	27.3	N423*	20.3*
	(25)	(11)	(13)	(12)	(55)	(7.4)
Medial	N214*	9.7*	P289	27.6	N393*	23.5*
	(17)	(4.7)	(16)	(15)	(26)	(11)
Parietal:						
Lateral	N235*	27.2*	P305*	42.7*	N375*	38.0*
	(38)	(12)	(32)	(47)	(21)	(9.9)
Medial	N229*	17.1*	P295*	22.7*	N368*	18.9*
	(27)	(5.8)	(23)	(5.9)	(3.5)	(23)

[a]Latencies in milliseconds post-stimulus are preceded by the polarity of the peak. Amplitudes are in microvolts. Standard deviations are in parentheses. Asterisks indicate values based on 4 patients or less.

positive parietal potential peaked within 10 ms of the temporal positive peak.

None of the intracerebral peaks showed a systematic latency correspondence with the scalp peaks.

Figure 3 also shows that the lateral-to-medial amplitude gradients were small at the intracerebral sites sampled. Amplitude gradients could sometimes be recorded along contacts of frontal and parietal electrodes but these gradients were variable across subjects; amplitude maxima appeared as frequently at lateral sites as at medial sites in the two lobes.

Very few polarity inversions were observed in the intracerebral sites sampled. A polarity inversion occurred in one patient for the first two peaks of the triphasic frontal response between medial and lateral frontal cortex. The negative-positive polarity pattern generally observed was inverted at medial sites. Also, in another patient the positive frontal and parietal peak (270 ms) appeared inverted at anterior temporal sites, although this occurred in the hemisphere showing seizure onset.

Table I summarizes the group amplitude and latency data for the peaks recorded at the lateral and medial contacts of the most frequently implanted electrode in each lobe. Although the averages are derived from electrodes implanted in slightly different sites in each

patient, the variability in the latencies of the most reliable peaks was relatively small. Amplitudes were more variable across patients but averaged about four times those obtained on the scalp.

To correlate the potentials recorded to the epileptic focus of the patients, we compared late potentials recorded from the epileptogenic temporal lobe to that recorded from approximately symmetric sites in the other temporal lobe. Of the nine patients in which a comparison could be made, 2 showed a disappearance of the potentials and 3 showed amplitude attenuation of about 50% on the side of seizure onset. No systematic differences could be observed between potentials at frontal sites ipsilateral and contralateral to the epileptic focus.

Late Potentials to Novel Stimuli

Rare novel stimuli evoked biphasic responses on the scalp that had a similar waveshape to the target-related N2-P3 potentials (see Figure 4). In 5 out of 8 patients this novel-stimulus N2-P3 had latencies that were shorter than the target N2-P3 by 20 to 50 ms.

In the frontal lobe, potentials evoked by novel stimuli were generally similar in waveshape and amplitude to those evoked by targets and had slightly shorter latencies on all peaks in 4 of 6 patients. These patients were the same as those showing earlier latencies for scalp potentials to novel stimuli.

At temporal sites, potentials associated with novel stimuli were also generally similar in waveshape and amplitude to that evoked by targets. Earlier latencies for novel stimulus potentials were found in the temporal lobe waveforms of 8 out of 14 patients.

In the parietal lobe, novel stimuli evoked triphasic potentials with peaks preceding that of target-evoked potentials.

The two polarity inversions reported for targets also occurred in the potentials to novel stimuli.

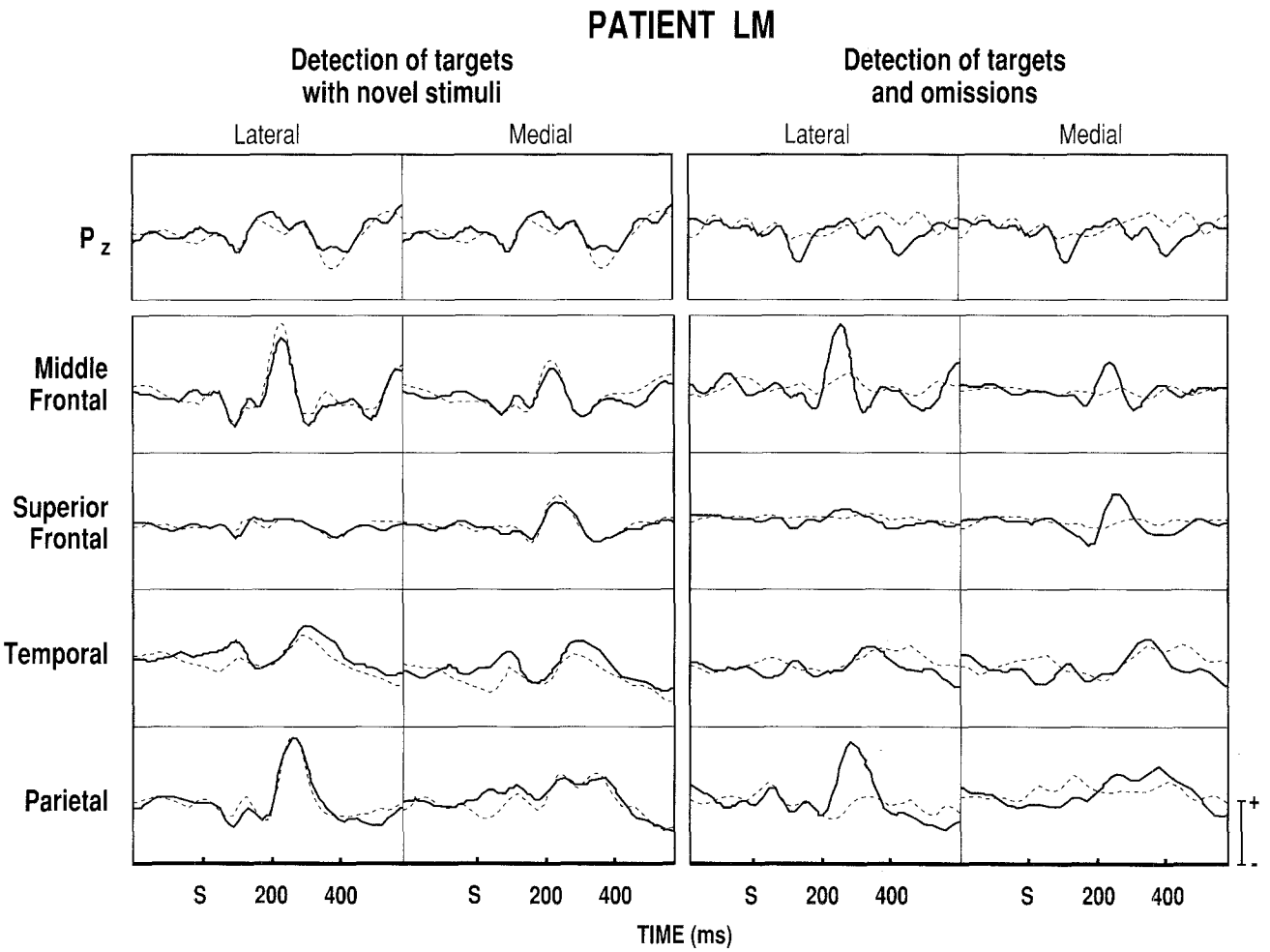

Figure 4. Intracerebral evoked potentials to targets (solid), novel stimuli (dashed), and tone omissions (dashed) in a patient with right temporal seizure onset. Waveforms are averages of 40-46 individual potentials. Calibration is 20 μV for scalp and 70 μV for intracerebral potentials.

Late Potentials to Stimulus Omissions

Four of the seven patients who were given the omission detection task showed a clear scalp response to stimulus omissions consisting of a positive potential peaking at 350-400 ms. In 2 patients, this positivity was preceded by a negative peak at 200-250 ms.

Stimulus omissions generally evoked very little activity in the frontal sites sampled (see Figure 4). In all but one patient, the late potentials produced at frontal sites by targets were not evoked by omissions presented in the same stimulus sequence. In the remaining patient the potentials evoked by omissions were markedly attenuated and did not have the characteristic triphasic waveshape of the frontal potentials to targets.

In the temporal lobe, evoked potentials to omissions were similar to those evoked by targets, (i.e., they generally consisted of a monophasic positive peak averaging 305 ms in latency). However, in all patients potentials evoked by omissions were an average of 20% lower in amplitude than those evoked by targets.

Finally, in the parietal lobe, late potentials to omissions showed the same effect observed in the frontal lobe; no visible potentials (3 patients) or very attenuated potentials (1 patient). The same patient showed an attenuated response in the frontal and parietal lobe.

No polarity inversions were observed in the potentials evoked by stimulus omissions.

Discussion

In the frontal lobe the late potentials to targets and novel stimuli are characterized by a triphasic response between 180 and 400 ms. Also, frontal waveforms show shorter latencies to novel than to target stimuli and disappear in most patients when stimulus omissions are the evoking event. The latencies of the peaks in the triphasic response (200, 268, and 350 ms) are similar to those recorded by Snyder and Hillyard (1976) on the scalp for ignored tone sequences. Rare tones in these unattended sequences evoked a triphasic response with peaks at N196 (N2), P258 (P3a), and N350 (N3) that were of maximal amplitude over the fronto-central midline but did not evoke the later centro-parietal P3 (P3b) evoked by attended rare tones. For tones in attended sequences, the P258 (P3a) is presumed to be embedded in the early portion of the P3b potential peaking at 325-380 ms (Snyder and Hillyard, 1976; Squires, Squires and Hillyard, 1975).

Thus, their similar latencies and frontal distribution suggest that the Snyder and Hillyard (1976) N2-P3a-N3 may correspond to the N-P-N response recorded in intracerebral frontal waveforms. Of course, this hypothesis will need confirmation by more direct evidence. If this correspondence is validated, we would expect that the scalp P3a would be highly attenuated during omissions interspersed in unattended tone sequences. We would also expect that the intracerebral N-P-N response would be evoked by rare tones in unattended sequences.

In the parietal lobe, the typical waveform is triphasic as in the frontal lobe but the latencies of some of the parietal peaks can correlate with either frontal or temporal peaks or differ from both. The latency differences between frontal and parietal waveforms make it unlikely that a single generator is responsible for the two potentials.

Our only reliable temporal potential was generally positive and peaked at about 285 ms. This peak does not appear to correspond to the positive peak in the frontal or parietal triphasic potential because it is not affected by the use of omissions instead of tonal targets.

Our temporal P285 peak may correspond to that recorded by others at temporal sites lateral to the hippocampus (McCarthy et al. in press, figure 9, Stapelton and Halgren, 1987, Figure 5). However, this peak does not appear to correspond to the well-documented wide-area medial temporal lobe potential (the B potential of Stapelton and Halgren, 1987) which has been reported to show anterior-posterior polarity inversions in medial temporal recordings. The B potential peaks after scalp P3 at about 420 ms whereas our P285 systematically preceded scalp P3.

Our failure to record the large B potential previously recorded in medial temporal sites may be due to the fact that our electrodes were generally not implanted as medially as those of other researchers. The negative B potential is reported to have a rather restricted distribution and can thus easily be missed (McCarthy et al. in press).

One of the most intriguing results of these recordings is that stimulus omissions failed to evoke the characteristic frontal and parietal site potentials in most patients while still evoking potentials in temporal sites. This suggests that some of the physiological events contributing to the intracerebral late potentials are stimulus-dependent while others are more generally event-dependent.

Simson et al. (1977) have observed that the scalp distribution of the P3 to omissions showed a decreased frontal extension compared to that of the P3 evoked by targets. This result may correspond to the difference we observed between intracranial potentials evoked by omissions and those evoked by targets. However, other studies (e.g., Hillyard et al. 1976) have not shown the scalp distribution change observed by Simson et al. (1977). Whether this effect can be recorded on the scalp or not, the present intracerebral recordings give evidence against the hypothesis that late potentials evoked by omissions have the same generators as those evoked by targets.

The late potentials recorded in the three lobes showed systematic differences in waveshape and/or latency. This lends support to the multiple generator hypothesis of late potentials. During the time span of the scalp auditory N2-P3, the cortical surface shows several voltage changes including positive and negative deflections in frontal, temporal and parietal sites and these deflections are differentially affected by the use of omissions as targets instead of tones.

The distributions observed in these patients do not suggest any location for the underlying generators of N2-P3 potentials. However, the distribution of the intracerebral late potentials is very different from that of auditory N1-P2 which shows a polarity inversion around the posterior sylvian area (Richer et al. 1989). This suggests that the intracerebral potentials recorded here are not generated in auditory cortex, as seems to be the case for N1-P2. Thus, to the extent that the intracerebral potentials recorded here contribute to the scalp N2 and P3, the generators of these scalp potentials are not likely to be in auditory cortex, unless the multiple generator configuration of late potentials produces a distribution which masks an auditory cortex polarity inversion.

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